Indexed in
Index Copernicus
Indian Science Abstracts
Google Scholar
Asian Education Index

Volume 7 Number 1 July - December 2015 **ISSN 0976 - 402X**

PUSHPAGIRI MEDICAL JOURNAL

An International Journal

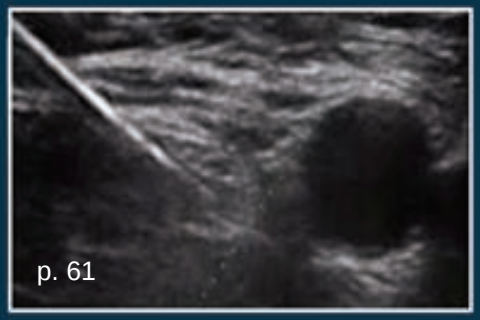
Official publication of

PUSHPAGIRI INSTITUTE OF MEDICAL SCIENCES AND RESEARCH CENTRE, TIRUVALLA - 689 101

Technique of immersion of hind paw for recording volume

The three-dimensional CT

Multiple unerupted teeth on OPG



Ultrasound guided nerve block

Also available at
journals.pushpagiri.in

PUSHPAGIRI MEDICAL SOCIETY

Pushpagiri Medical Society is a Charitable Society registered under Travancore Cochin Literary Scientific & Charitable Societies Registration Act, 1955 bearing the registration number P.73/92.

OUR MISSION

To have life for everybody and to have it in abundance through Science and Technology for a Knowledge Society and for improving the Health of our immediate community, the State, the Country and the World at large.

OUR VISION

We care God cures

INSTITUTIONS UNDER THE SOCIETY

1. Pushpagiri Institute of Medical Sciences & Research Centre
1.1 Pushpagiri Medical College Hospital
1.2 Pushpagiri Medical College
1.3 Pushpagiri Institute of Medical Sciences & Research Centre Cardiology, Cardiovascular and Thoracic Surgery
2. Pushpagiri College of Dental Sciences
3. Pushpagiri College of Pharmacy
4. Pushpagiri College of Nursing
5. Pushpagiri College of Allied Health Sciences
6. Pushpagiri School of Nursing
7. Pushpagiri Centre for CGFNS & IELTS Training
8. Pushpagiri Research Centre
9. Pushpagiri Centre for Virology

AIMS AND SCOPE

'Pushpagiri Medical Journal', an International Journal, is the official publication of Pushpagiri Institute of Medical Sciences and Research Centre, Tiruvalla. It is a peer reviewed multi-disciplinary journal providing health professionals with a forum to discuss current challenges in healthcare, sharing innovative evaluation and treatment techniques, learning about and assimilating advanced methodologies being developed in various disciplines in modern medicine as well as related professions, and communicating information regarding newer developments and research programmes. The journal serves as a valuable tool for helping therapists deal effectively with the emerging problems, stumbling blocks and challenges in the field, and emphasizing articles and reports that are directly relevant to medical practice and public health. It will publish original research articles, concise evidence based review articles, unusual and interesting case reports and technical reports. We offer an online submission facility, and a fast reviewing process and editorial decisions so as to avoid delay and inconvenience to the authors. The journal is being included and indexed with many international databases, and it will be published half-yearly, in March (January-June) and September (July-December).

ETHICAL CONSIDERATIONS

Manuscripts submitted for publication should comply with the following:

Informed consent

Before patients are considered for participation in any study their informed consent should be obtained. Patient identifying information, such as names, initials, hospital number and photographs should not be included in the manuscript.

Protection of human subjects and animals in research

In case of original research articles on clinical trials, appropriate approval must have been obtained from the relevant Ethical Committee (both Institutional and National) on human experimentation and the Helsinki Declaration of 1964 (as revised in 2008). While reporting animal experiments, authors must follow the institutional and national guidelines for the care and use of laboratory animals.

COPYRIGHT

The articles must represent original research or clinical study material, should not have been published before and should not be under consideration for publication elsewhere. It is the duty of the author to obtain the necessary permission for extensive quotations, tables, illustrations or any other copyrighted material they are using in the article. Once the copyright statement has been signed by the corresponding author, no change in authorship or in the order of authors listed on the article would be accepted. Copyright of the articles gets transferred to the publisher, once they have been accepted for publication.

While all possible care shall be taken to provide accurate and genuine information in accordance with the date of publication, neither the authors and editors nor the publisher take legal responsibility for any unintentional omission or error. The publisher makes no expressed or implied warranty with respect to the information contained herein. The published material cannot be photocopied for general distribution, promotion, new works or resale. Exclusive rights to reproduce and distribute the articles in this journal, as well as translational rights have been protected by copyright. No form of material published in this journal can be reproduced in digital format or stored in electronic databases, videodisks, etc.

Authors should give due acknowledgement to the assistance received from any other source while conducting research studies, and also disclose the funding sources.

SUBSCRIPTION INFORMATION AND ADVERTISEMENT

For Subscription rates, orders, back volumes and advertisement queries please contact:

Dr Santosh Pillai
Associate Editor, Pushpagiri Medical Journal
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla - 689 101, Kerala, India
Phone: 0469 2700755 Ext. 550
Mob. 9447596426
tusukumaran@gmail.com
drsantosh74@gmail.com

ELECTRONIC MEDIA

An electronic edition of this journal is available at journals.pimsrc.in

Manuscripts can be submitted online at

drsantosh74@gmail.com

PMJ

Pushpagiri Medical Journal

An International Journal

Volume 07, Number 01 July - December 2015

Official Publication of
Pushpagiri Institute of Medical Sciences and Research Centre

Editorial Office:
Office of the Principal
PIMS & RC, Tiruvalla
Pathanamthitta

Phone: 0469-2733761, 2700755
(Ext. 555, 556)
Fax: 0469-2600020
E-mail: pcm@pushpagiri.in
Website: www.pimsrc.in

Printed, Published and Owned by:
Rev Fr Dr.Shaji Mathews Vazhayil
in his official capacity as Chairman and
CEO of Pushpagiri Group of Institutions
Phone: 0469-2603833, 2700755 (Ext. 401)
E-mail: ceopushpagiri@gmail.com

Printed at:
Furore Digital Printing, Pathanamthitta

Published at:
Tiruvalla - 689 101
Pathanamthitta, Kerala, India by the
Pushpagiri Institute of Medical Sciences
& Research Centre

ISSN 0976-402X

PUSHPAGIRI MEDICAL JOURNAL

Volume 7, No. 1

CONTENTS

July-December 2015

CASE REPORTS

✍ EDITORIAL

Publishing for survival - MCI norms

Rajeev A

From:
Pushpagiri Institute of
Medical Sciences
& Research Centre
Tiruvalla, India - 689 101

Rajeev A
Professor
Department of Paediatrics

Correspondence to:
Dr. Rajeev A
E-mail: rajeevtka@gmail.com

Medical Council of India as per the Minimum Qualifications for Teachers in Medical Institutions Regulations, 1998 has been dictating that a minimum of four research publications indexed in Index Medicus/national journals "as desirable" for considering a medical teacher for promotion as Associate Professor or Professor, in addition to the requisite years of experience. The fundamental idea behind this was that the young faculty in the department must be well-versed with research as an endeavor to discover facts by study or investigation by themselves so much so they are able to inculcate the same curiosity in the undergraduates they are teaching as well as the post-graduates they develop with[1]. Medicine never ceases to change and to apply ever-evolving knowledge one must be open to experimentation with data relating to the diagnosis, aetiology, prognosis, as well as therapeutic aspects of diseases.

Besides, the teachers are expected to be role-models and must be able to infuse the new entrants to the field to ethical facets of research and this requires daily practice of research methodology. However, all this was fine until the medical council made publications mandatory for promotion beyond assistant professor post as per the amendments in 2009. The requirements meant that it was compulsory to publish two papers during the assistant professor ship and two more during the associate professorship in a graded fashion. This led to discussion on open fora regarding the importance of research in teaching of medicine. The thinking in India has been always that research is secondary to clinical work and must not be made compulsory for busy clinicians and "good teachers". The remaining faculty had to swallow the bitter pill and become proficient writers to make up for their short-comings as faculty. However, this led to the new argument that the quality of such "re-search" will be poor where resources are lacking. Quantity would beat quality as defined by the doyens of publishing industry.

The world-wide trend, however, was of the line "publish or perish". Though, India was far behind in the race to publish, we also felt the requirement growing in importance. The market of publishing industry grew in terms of new publications which threatened to de-throne many of the top journals in the medical field. Some of the online open-access journals beat the popularity of standard journals. The impact factor game was thus borne to keep the supreme journals from succumbing to the market pressure. The standard of publication was thus defined by the number of references which the articles made in the very same journals which promoted the importance through cyclical referencing. The natural English speakers had an upper hand in promoting this idea to retain the rejection-ridden field of indexed publications. Indexing was in fact the criteria for the initial desirable requirement of MCI for promotions.

However, as the publications were made mandatory at each step of promotion, getting indexed publication in high impact journals became a herculean task for medical teachers of the country. The time-bound nature of promotions was threatened by the time it took to publish even two articles. This was circumvented by the faculty through multiple authorships. Though, multi-disciplinary teams are ideal for medical research, the multiple authored articles in the country were characterized by gift authorship and "salami" publications etcetera. Medical Council of India did one up on this by restricting the authorship to first author limiting the importance of the remaining authors including the senior author into oblivion. As per the criteria in 2009 these research publications needed to be published/accepted for publication in the Journals by the National Associations/ Societies of the respective specialities as the First Author. The backlash was that the guiding and overseeing faculty stopped supporting the publications. This resulted in an amendment which expanded authorship to first two authors but curtailing to indexed and/or national journal.

Since the amendments had a window period for coming into enforcement, a strict mandate was not followed by institutions especially in the government sector where the promotion rules were existent in the seniority ladder format for years together. The latter was neither time-bound nor research dependent. The divarication of the public and private teaching careers ensured that the independently run institutions could dictate terms and public run institutions could procrastinate by the unwritten rule that the promotions are on the basis of availability of posts. The stalemate continued till 2015 following the publication of Minimum Qualifications for Teachers in Medical Institutions Regulations, 1998 (amended upto May, 2015)[3] and the additional clarifications were given as under.

a) Indexing agencies: Scopus, PubMed, Medline, Embase/ExcerptaMedica, Index Medicus and Index Copernicus

b) Types of articles to be considered: Original research articles and original research papers.

c) Criteria for National/International journal: Published by a National/International Specialty Journal/ Journal of a National/International Society provided it included in one of the indexes mentioned above.

d) Authorship: First author, second author.

e) E-journals: e-journals not included

This has created additional confusions with regard to the original ideology behind promoting publications. The documentation of individual investigations was to share valid practice-changing evidence and that too in peer-reviewed publications of repute. The reputation of journals had been dependent on the monopolistic few which required not only proficiency in English language as well as the unwritten rule of lead 'expert' authors with a track record. Online e-journals thwarted as well as disrupted the dissemination equation of evidence. However, since the internet is full of questionable evidence, separating chaff from the wheat has been solved by removing e-journals from being considered at all[3]. The circular in question solved the relatively lower hierarchy of the so-called national publications from the richer cousins by giving similar treatment to both regardless of geographical boundaries to Medical Council's credit.

Indexing is another contentious issue. Just as there are qualifying and competitive examinations, promotional criteria are to be liberal and qualifying only; as authorship is currently restrictive in nature with respect to the number of authors per article. Rigorous indexing of older style (Medline/Index Medicus) would have curtailed career advancement of a lot of teachers. Many popular journals of pubmed fame have even started charging heavily for publication as most path-breaking publications are funded and permits use of the fund for such dissemination. The guild of Indian medical editors condemns the use of inferior indexing even when they are supporting inclusion of home-grown IndMed into the regulations. The ever popular Google is trying to gain upper hand by the Scholar H-index as also similar initiatives such as Researchgate. Definition of original articles needs to be expansive enough to include data synthesis while not ignoring the contribution of case-series with data analysis. Senior authorship may be incentivized and even the status of corresponding author may be accepted as valid authorship for appropriate accreditation of institutes or departments. Going overboard one might even bring in the UGC system of including research publications (books and book chapters), research projects, research guidance, and training courses and conferences/seminars[4].Rather than eulogizing the inconsequential animal research studies of herbal medications as original research, accepting the medical error report for the sake of preventing such occurrences or changing practice in future could even be noble to say the least[5].

References

1. Sukhlecha A. Research publications: Should they be mandatory for promotions of medical teachers?. J Pharmacol Pharmacother. 2011 Oct-Dec; 2(4): 221–224.
2. Medical Council of India. Minimum Qualifications for Teachers in Medical Institutions Regulations, 1998 (amended upto May, 2015) [Last accessed on 2016 May 06]. Available from: http://www.mciindia.org/Rules-and-Regulation/TEQ-REGULATIONS-16.05.15.pdf and http://www.mciindia.org/circulars/Circular-03.09.2015-TEQ-Promotion-Publication.pdf
3. Aggarwal R, Gogtay N, Kumar R, Sahni P, for the Indian Association of Medical Journal Editors. The revised guidelines of the Medical Council of India for the academic promotions: Need for a rethink. J Conserv Dent 2016;19:1-4.
4. Bandewar SVS, Pai SA. Regressive trend: MCI's approach to assessment of medical teachers' Performance. Indian Journal of Medical Ethics Vol XII No 4 October-December 2015
5. Jain DK, Sharma H, Arya RK. Questioning definition of research in India. World Journal of Pharmaceutical Research. Volume 3, Issue 7, 568-579.

✪ SPECIAL REPORT

Report on National Conference on Frontiers in Diabetes Research and Treatment held at Pushpagiri Institute of Medical Sciences and Research Centre, Tiruvalla, Kerala

Shilpa Joy

C.K.K. Nair

From:
Pushpagiri Institute of
Medical Sciences
& Research Centre
Tiruvalla, India - 689 101

Shilpa Joy
Scientist

C K K Nair
Dean

Pushpagiri Research Centre
PIMS & RC, Tiruvalla

Correspondence to:
Dr Shilpa Joy
E-mail: shilpajoy09@gmail.com

Diabetes is the most serious and the fastest growing disease of all worldwide. It is reported to be the fifth leading cause of death in the U.S. Diabetes has an endemic status in India with more than 62 million individuals diagnosed with it. Several recent biochemical, genetic, and clinical studies have revealed that multiple gene defects and polymorphisms, combined with physiological consequences of modern life styles (food habits, nutrition, consumption of food items contaminated with toxic chemicals, consumption of alcohol and beverages, lack of physical exertion and inadequate exposure to sun light etc) contribute to the risk of diabetes manifested as reduced insulin secretion, reduced insulin sensitivity (insulin resistance), and increased hepatic glucose output – all in turn culminate in increased fasting blood glucose levels, inadequate plasma insulin levels, impaired glucose tolerance, and misregulated lipid metabolism. The continuance of the diabetic condition over long period leads to the development of several complications - neuropathy, retinopathy, nephropathy, micro- and macroangiopathies, and cardiovascular diseases and mortality. More than 36 genes have been found to be involved for the risk of type 2 diabetes. Apart from these, diabetes also arise due to an abnormality in a single gene (known as monogenic forms of diabetes); which include maturity onset diabetes of the young (MODY), Donohue syndrome, and Rabson-Mendenhall syndrome. More than 20 genes have been identified to cause monogenic diabetes. Neonatal diabetes is mainly caused by mutations in the kcnj11, abcc8 or ins genes.

This year's Bioradiance Conference of Pushpagiri Research Centre was focussed on Diabetes with research and treatment options in modern medicine, ayurveda and herbal/phyto medicine. The one day National Conference on Frontiers in Diabetes Research and Treatment was held on 24th July, 2015 at Senate Hall, Pushpagiri Institute of Medical Sciences and Research Centre. Dr. PG Latha, Director, JawaharLal Nehru Tropical Botanical Garden Research Institute, Trivandrum, inaugurated the function in the presence of Rev. Dr. Shaji Mathwes Vazhayil, CEO, Pushpagiri Group of Institutions, Rev. Dr. Mathew Mazhavancheril, Director-Academics and Research, Dr. C.K.K. Nair (Dean of Research) and Dr. Anand Manoharan (Head, Research and Development). The conference highlighted various aspects of the disease Diabetes, importance of aggressive research and identifying feasible therapeutic modalities, both by modern medicine and by exploring natural plant resources. The chief guest, Dr.Latha gave an exhaustive list of plants and herbals, traditionally used and scientifically tested, with therapeutic activity against diabetes. Dr. KV Swapna, Head, Department of Panchakarma, Arya Vaidya Pharmacy, Coimbatore highlighted the importance and modern developments in Ayurvedic system for treatment of Diabetes. Dr. Manish, faculty of Pushpagiri Research Centre, gave the molecular aspects of signalling partners involved in Diabetes and certain current and future research goals related to the treatment. The Endocrinologist, Dr. Rajeev Philip, Pushpagiri Medical College, emphasised the newer therapeutic targets involved in modern medicine to control Diabetes. A total of 22 papers were presented and 141 participants including students, scientists and faculties from medical colleges, dental college, Ayurvedic Institutions, Science colleges and Research Centres attended the Conference. The best 2 posters among them

were selected for Dr D.M. Vasudevan Awards which were sponsored by Origin Diagnostics and Research. The valedictory function of the conference was presided by Dr. Tomy Phillip, Vice-Principal, and the Chief Guest for the function was Dr. T.P. Thankappan, Principal, Pushpagiri Medical College. Dr. Shilpa Joy, Scientist, proposed the vote of thanks at the function. The following are the abstracts of the papers presented in the Conference.

ENHANCED BONE REGENERATION BY GREEN TEA EXTRACT CONJUGATED CHITOSAN SCAFFOLD

Vishnu P, Thushara P, Nebu G T, Shilpa Joy, Regenerative Medicine and Nanotechnology lab, Pushpagiri Institute of Medical Sciences and Research Centre, Tiruvalla, Email- shilpajoy09@gmail.com

Bone tissue engineering is a complex and dynamic technology that facilitates osteoblast proliferation, differentiation, matrix formation and finally complete bone formation. The most promising strategy used in tissue engineering is based on three dimensional scaffolds. Chitosan is one such material. Microbial invasion at the site of regeneration slows down the cell proliferation. This study addresses the influence of antimicrobial green-tea aqueous extract loaded chitosan scaffold on bone regeneration. Sprague Dawley rats of 250-300 g body weight were used for the experiment. They were divided as sham operated control (SC), animal with bone defect (BD) and animal with bone defect which was implanted with chitosan scaffold on the defect (BDMB). In BDMB, 8 mm diameterdefects in calvarial bone were made with a trephine bur and then green-tea aqueous extract loaded chitosan membrane was interposed between the osseous defect and overlying flap before suturing. In BD, the defect was only covered by the soft tissue flap and sutured the injury. Blood samples were collected on 5th, 10th, 15th, 20th and 25th day after surgery for analyzing Alkaline Phosphatase (ALP) activity and after 25th day bone section collected for histological examination. The biocompatibility of the membrane was confirmed by studying the occurrence of inflammatory responses. ALP activity was increased gradually in BDMB that denoted active osteoblast multiplication and subsequent bone regeneration. This has immense therapeutic significance in bone tissue engineering.

PREVALENCE AND RISK FACTORS OF ANAEMIA AMONG ADOLESCENT SCHOOL GIRLS: A CROSS SECTIONAL STUDY IN A TEHSIL OF SOUTHERN INDIA

Lucy George*, B Sreedevi** Philip Mathew** , * Department of Biosciences, Mar Athanasious College for Advanced Studies, Tiruvalla and Department of Nutrition, Dr NGP Arts and Science College, Coimbatore, Tamil Nadu; ** Department Of Community Medicine, Pushpagiri Institute of Medical Sciences and Reseach Centre ,Thiruvalla.

Prevalence of anaemia in India is the highest in comparison with the rest of the world and it is estimated that 50 percent of global maternal death due to anaemia is contributed by the country. Low dietary intake, poor consumption of iron and folic acid, low bioavailability absorption of nutrients, diet rich in phytate, fibre and chronic blood loss due to infections are the major reasons attributed to anaemia prevalence in India. A cross sectional study using multi-stage random sampling, was conducted in KonniTaluk of Pathanamthitta district, Kerala. A total of 200 school-going adolescent girls in the age group of 15-18 years were enrolled into the study. A pre-tested questionnaire was administered to the participants to find out about their socio-demographic correlates, dietary habits and awareness regarding anemia. Venous blood was collected for Hemoglobin estimation and nutritional anthropometry was done. The prevalence of anemia in the study group was 98%, with 37% having mild anemia and 61% having moderate anemia. Anaemia among the study group was significantly associated with Body Mass Index (p-0.024), Father's occupation (p- 0.005) and dietary consumption of fruits (p-0.001). The prevalence of anaemia was much higher in the study group when compared to previous studies done on the same subject, possibly as a result of the study being done in a predominantly rural and backward taluk. This study shows that anaemia among adolescents is a significant public health problem even in relatively well-developed states like Kerala, despite various governmental efforts.

DIETARY SUPPLEMENT FORMULATION DESIGNED FOR PATIENTS WITH DIABETES

R. Rajakumari a, NandakumarKalarikkal a, b, Sabu Thomas a, c*
*a International and Inter-University Centre for Nanoscience and Nanotechnology, Mahatma Gandhi University, Kerala, 686560, India; *b School of Pure and Applied Physics, Mahatma Gandhi University, Kerala, 686560, India. E-mail: nkkalarikkal@mgu.ac.in; Fax: +91-481-2731669; Tel: +91-481- 2731669; *c School of Chemical Sciences, Mahatma Gandhi University, Kerala, 686560, India. E-mail: sabuchathukulam@yahoo.co.uk; Fax: +91-481-2731002; Tel: +91-481-2731002

It is rather impossible to find one consuming a well-balanced diet regularly. Somehow, one can expect the deficiency of certain nutrients in their regular diet. In order to overcome such drawback in the normal diet, the combination of vitamins, minerals and grape seed extract as a dietary supplement in the form of bilayer tablet is proposed to see the various nutrients can find a place in one tablet when normal man consumes the same. The main objective of the present work is to design a film coated bilayer tablet in which one layer contains minerals while the other one encompasses the minerals. The uniquely formed bilayer tablet is assessed for the physiochemical properties, microbial load and stability studies. The optimized bilayer tablet results found to be within the limits. The sub-chronic toxicity study of 28 days has been carried out using wistar rats and swiss albino mice. The observation has shown a normal body weight gain throughout the period of studies bearing the age, weight and sex of the animal. The histopathological examination of organs like adrenal, heart, kidney, liver, lung and stomach have not brought out any abnormality and lesion. Hence, it is concluded that the bilayer tablets are observed to be a toxic free nutrient supplement.

POLYSACCHARIDES ISOLATED FROM THE ALGAE, ENTEROMORPHA INTESTINALIS SHOWS ANTI-PROLIFERATIVE ACTIVITY AGAINST HUMANHEPATOMA CELL LINES, HEP3B

Akhil GC, Reshma S, Nevin KG, Prakashkumar B, *School of Biosciences, Mahatma Gandhi University, PD Hills PO, Kottayam-686560, Email: akhil.gc@gmail.com; nevinkg@gmail.com

Hepatocellular carcinoma is the most frequent primary malignancy of the liver. Statistics shows that, by 2015 about 35,660 new cases of hepatocellular carcinoma will be diagnosed. Due to the side effects of the available synthetic drugs in the market, there is a constant demand to develop low toxic, effective, and affordable anticancer drugs. Marine algae, a rich source of bioactive compounds are important ingredient of many health products and drugs for treating cancer and other diseases. The algal cell wall is composed of different type of polysaccharides with some sulphated. The sulphated polysaccharides have effective anticancer activities by attacking the cancer cell directly or enhancing the host's immune function. Enteromorphaintestinalis is green algae widely distributed in the coastal regions of south India, which are rich in polysaccharides. The present study evaluates the antiproliferative activity of polysaccharide isolated from the marine algae E. intestinalis. The algae E. intestinalis was collected from the coastal regions of Trivandrum district. The algal material was air dried and crude polysaccharide was prepared with hot water and precipitated with ethanol. Polysaccharides thus obtained were deprotienized using Sevag method. Total carbohydrate content and reducing sugars were estimated using Phenol- sulfuric acid and Dinitrosalisylic acid (DNSA) method respectively. Total proteins were estimated by Bradford method, while total sulfate content was estimated by barium chloride method. Cytotoxicity of polysaccharides was evaluated against Hep3B cells. MTT assay was employed to study the dose dependent cytotoxicity. Acridine orange/Ethidium bromide (AO/EB) staining was used to differentiate the live and apoptotic cells. DAPI staining was used to determine the nuclear changes of treated cells. The ability of the polysaccharide to induce intracellular ROS formation was determined using DCHFA and mitochondrial membrane potential of treated cells by Rhodamine 123 staining Results: The amount of total carbohydrate present was 27.92µg/100mg while the amount of reducing sugar present was 3.62µg/100mg. The amount of total protein and sulfate present in 100 mg polysaccharide was found to be 14.36 and 14.21 µg respectively. MTT assay showed that the polysaccharides induced dose dependent cytotoxicity towards Hep3B cells. Studies with different stains showed that the polysaccharides induced nuclear changes corresponding to apoptosis and also reduced the mitochondrial membrane potential by increasing the intracellular ROS production.The polysaccharide isolated from the algae E. intestinalis showed potent anticancer activity on Hep3b cancer cell line which may be mediated by mitochondrial ROS production.

COGNITIVE IMPAIRMENT IN SLE

Byju P*, David**, Robert Mathew*, Padmakumar**, *Department of of Neurology, Pushpagiri institute of medical sciences; ** Department of General Medicine, Alappuzha medical college

The aim of this work is to study the pattern of cognitive dysfunction in SLE. Here we used the Prospective cross sectional study. 30 consecutive SLE patients attending a tertiary care teaching hospital during the period 2010 to 2012 were studied. Cognition was assessed using ACE-R. Trail A and B digit span and CBI. All the patients were females. Mean age was 33.4(SD 9.53) years and education 2.07 (SD0.83 Duration of SLE at the time of presentation was 50.93(SD 43.27) and duration of neurologic manifestation from onset of SLE 12.77(SD 17.045).Sixty percent of the patients had neurologic manifestations. Mean ACE score was 86.17(SD1.05) which was higher than the existing cut off score of 67.The ACE subscores were converted to percentage of maximum score for purpose of comparison and showed high values ranging from 63.57(SD9.06) for executive function to 90(SD 17.16) for visuospatial function. When one sample T test was used keeping 90 as test value significant difference in performance was seen between visuospatial function and executive function($p<.001$) and language function($p<.001$) and not other domains-memory($p=0.328$) and orientation($p=0.856$).Significant impairment in ACE score was seen in the group with neurolupus ($p=0.053$) when compared to others. When cognitive subscores were compared no difference was seen between the groups. In this cohort of SLE patients tests of cognitive functions appeared normal. However the performance in visuospatial function appeared poor when compared to performance in other cognitive domains.When compared with patients without neurolupus patients with neurolupus had significantly low ACE score.

MUSCARINIC M1 RECEPTOR SUBTYPE FUNCTIONAL REGULATION IN THE CEREBELLUM OF GABA AND 5-HT CHITOSAN NANOPARTICLE TREATED PARTIALLY HEPATECTOMISED RATS

Akhilraj P R, Anju T R, Ajayan M S, Paulose C S.*, *Molecular Neurobiology and Cell Biology Unit, Centre for Neuroscience, Department of Biotechnology, Cochin University of Science and Technology, Cochin-682022, Kerala, India. E MAIL-biomncb@cuasat.ac.in

Hepatic injury and cell loss makes structural changes and neurological dysfunctions in central nervous system by affecting the metabolic homeostasis. Metabolic alterations during partial hepatectomy can affect the functional regulation of neurotransmitters. Cerebellum plays a central role in many complex brain functions including voluntary movements such as posture, balance, coordination, and speech, resulting in smooth and balanced muscular activity. It is also important for learning and cognition. Liver dysfunctions can cause damages in cerebellum seriously affecting its functions. The present study investigated the role of Gamma aminobutryric acid (GABA) and 5-hydroxytryptamine (5-HT) chitosan nanoparticles in the functional regulation of Muscarinic M1 receptor subtype in cerebellum during enhanced liver cell proliferation. Liver injury was achieved by partial hepatectomy of male wistar rats and GABA and 5-HT chitosan nanoparticle treatment was given intraperitoneally. The experimental groups were sham operated control (C), partially heptectomised rats with no treatment (PH) and GABA and 5-HT chitosan nanoparticle treatment (PHT). Receptor binding of muscarinic M1 receptor subtype was studied by scatchard analysis using [3H] QNB binding against piranzepine in the cerebellum. Partial hepatectomy resulted in an increased muscarinic M1 receptor subtype density, which was reversed to near control by treatment. Histological analysis showed that there was no significant cell loss in cerebellum due to partial hepatectomy. Partial hepatectomy also resulted in an elevated antioxidant level with a reduction in calcium level. The Real Time PCR analysis revealed a decreased gene expression of transcription factors - Akt-1, CREB and NF?B in the cerebellum of the partially hepatectomised rats. These alterations in antioxidant and calcium levels and gene expression of transcription factors were reversed in the treatment group implicating the possibility that the GABA-5-HT chitosan nanoparticle treatment can effectively regulate the cerebellar alterations at a very early stage itself thereby controlling cerebellar damage. Thus it was concluded that GABA and 5-HT chitosan nanoparticle treatment in partially hepatectomised rats render liver proliferation with reduced damage in the cerebellum which has therapeutic significance in liver disease management.

Acknowledgements: This work was supported by research grants from DBT, DST, ICMR, Govt. of India and KSCSTE, Govt. of Kerala to Dr. C. S. Paulose.

PREPARATION AND CHARACTERIZATION OF CALCIUM ALGINATE NANOPARTICLES FOR THE CONTROLLED DELIVERY OF A POORLY WATER SOLUBLE DRUG

Deepa Thomas1, Anju Anand1, M.S Latha*, 1Research and Post Graduate Department of Chenistry, Bishop Moore College Mavelikara, Kerala ,India. *Department of Chemistry, SreeNarayana College, Chengannur, Kerala, India, Email - deepasmammen@gmail.com,latams@yahoo.com

This work presents the development of pH-sensitive and biocompatible nanoparticles based on calcium alginate, prepared for controlled oral delivery of poorly water-soluble drug. The nanoparticles prepared were characterized by Fourier Transform Infrared spectroscopy (FTIR) and Scanning Electron Microscopy (SEM). A model drug, rifampicin, was entrapped into these nanoparticles and release studies were carried out. Drug content and encapsulation efficiency were determined. The designed nanoparticles have particle size in the range 80 to 160 nm. Nanoparticles revealed a sustained release of rifampicin. The simplicity of preparation, high drug encapsulation efficiency and sustained release demonstrate the potential of the formulation for the controlled release of poorly water soluble drug.

PARKINSONISM IN pNPH

Prasanna, Sreenivas, Robert Mathew, Sunitha Robert, Shahul Hameed*, Pushpagiri Instituite of Medical Sciences and Research Centre, Tiruvalla, Kerala

To study the Parkinsonism features in patients with Possible Normal Pressure Hydrocephalus. The method used is Prospective cross sectional study. Patients availing dementia care services from a tertiary care teaching centre were studied. Fourteen patients with pNPH were included in the study. They underwent imaging, Addenbrookes Cognitive Examination(ACE), assessment of Parkinsonism using UPDRS and Cambridge Behavior Inventory(CBI). 50% were males, mean age was 76.06(SD6.39)years, mean duration of illness 26.14(SD18.73). Mean ACE score was 43.25(SD22.19). The mean UPDRS score was 22(SD2.71), range 0-94 thereby indicating mild impairment. 12(86%) patients had at least one UPDRS item impaired. Memory, behavior and mood impairment was seen in 7(43%), ADL in 9(64%) and motor impairment in 11(79%). Of the motor scores maximum number had impairment in leg agility 10(71%) and minimum had impairment in action tremor 2(14%). The maximum number impaired in ADL score was in cutting food 7(50%) and least impaired in 'turning in bed' 2(14%). When the individual UPDRS was analysed minimum impairment was for sensory symptoms (mean score 0.14) and maximum leg agility (1.5). Minimum motor score was for action tremor (mean 0.21) and maximum for leg agility. No correlation was seen between ACE composite score and UPDRS ($p= 0.172$). Negative correlation was seen between ACE subscores and UPDRS subscores(pearson's correlation $p<0.05$) which was not seen when controlled for age, sex and duration of illness. Parkinsonism appeared very common in NPH; motor scores being more impaired. Impairment appeared mild even when the cognitive impairment was significant. Parkinsonism and cognitive impairment did not correlate.

COMPUTATIONAL ANALYSIS OF LEUCOCYANIDIN WITH INSULIN RECEPTOR TYROSINE KINASE

Koshy Jesvin Joy, Suparna.S, Asha Mathew , Betsy M Baby *, Hareesh R and Stephen James*, *School of Biosciences, Mar Athanasios College for Advanced Studies,Tiruvalla-689101, *Email: betsy@macfast.org

Diabetes mellitus is a metabolic disorder caused due to insulin deficiency.Aim of our study is to find the effect of leucocyanidin with insulin receptor tyrosine kinase. Insulin receptor is a tetramer that belong to a family of receptor tyrosine kinase. It contains two alpha subunit that form the extracellular domain and beta subunit that constitute the extracellular region of the receptor and cause conformational changes that lead to the activation of tyrosine kinase. This leads to autophosphorylation hence the compound that increase insulin receptor tyrosine kinase activity would be useful in the treatment of diabetes. The 3D structure of insulin receptor tyrosine kinase and structure of flavanoid were obtained from PDB database and NCBI PubChem respectively. The result of the study is that the flavanoidluecocyanidin shows a binding affinity with tyrosine kinase receptor.Softwares used is Hex 8.0, AutoDock 4.0, Swiss PDB Viewer, RasMol.

SPECTRUM OF HORMONE RESISTANCE SYNDROMES IN PUSHPAGIRI MEDICAL COLLEGE

Nithin Thomas, Terence Rajeev Philip , C.S.Kesavan , P. Viswanathan, Pushpagiri Instituite of Medical Sciences and Research Centre, Tiruvalla, Kerala

Hormone resistance syndromes are fascinating aberrations in normal hormone action. We present three different hormone resistance syndromes which presented to us. In case one, 26 yr old female, nursing student who had history of ADHD, sub normal intelligence,hearing loss was detected to have hyperthyroidism elsewhere 5 years back and was on started on treatment with anti thyroid drugs (high doses) which was further hiked based on TFT.On review of history & blood investigations,it was found that patient was clinically euthyroid, Had increased S.T4, T3, free T4 and free T3, with an inappropriately normal TSH, MRI of the pituitary and sex hormone binding globulin were within normal limits., giving a diagnosis of Thyroid hormone resistance . In case two, 20 year old female was evaluated for primary amenorrhoea, found to have high LH, high FSH, a lowish normal estradiol and USG abdomen and pelvis done outside showed hypoplastic uterus and ovaries. In view of primary amenorrhoea, well developed breasts, minimal pubic hair and axillary hair, LH>FSH ,in a phenotypical female, the diagnosis of complete androgen insensitivity was made, with MRI confirming male gonads.In case three, a 12 year boy was evaluated for recurrent episodes of carpopedal spasms, and on examination found to have chubby face, short stature, and short 4rth and 5 th metatarsals. His calcium was low, Phosphate high and alkaline phosphatase normal with elevated PTH and normal vitamin D, leading us to the diagnosis of pseudohypoparathyroidism. Hormone resistance syndromes are fascinating aberrations in normal hormone action, giving us valuable insights into the hormone physiology and mechanism of action. They are made more intriguing by the fact that each hormone resistance is different, and for a particular hormone resistance, the resistance can occur only in certain tissues only, as seen in our cases.

BIOPOLYMERIC MATRICES LOADED WITH 'HEALING' PROTEIN NANOCONSTRUCTS FOR THE TREATMENT OF DIABETIC WOUNDS

Praveen G * 1, Nandakumar Kalarikkal 1, 2, Sabu Thomas 1,3, 1 International and Interuniversity Centre for Nanoscience and Nanotechnology,2 School of Pure and Applied Physics, 3 School of Chemical Sciences, Mahatma Gandhi University, Priyadarshini Hills P O, Kottayam 686 560, Kerala, India, E-mail address (praveengovindh@gmail.com)

Wound healing is a complex biological process, which involves the restoration of cellular structures and tissue layers by the coordination of a variety of cellular activities, such as inflammation, migration of cells to the wound area, proliferation, angiogenesis, and remodelling of the extracellular matrix (ECM). In humans, self regeneration and repair potential of damaged skin tissues are restricted in chronic diabetic patients. This research work describes the strategy to develop biomimetic tissue engineered matrices from bio-polymeres and proteins. Fibrin is one such secreted biomolecule actively elicited during coagulation cascade in the immediate vicinity of a wound. Fibrin, apart from protecting the wound from bacterial invasion, is critical in enhancing angiogenesis or revascularization needed for the healing process.The choice of fibrin and its use as a control delivery agent for desired drug/ growth factor was done based on the preliminary studies reported earlier.1 Biopolymeric matrices of bare agarose and Agarose-Fibrin (AnF) composite were fabricated through controlled freeze drying technique. Physico- chemical characterization of material was performed using SEM, FTIR, XRD and UTM. Cytocompatibility of the fabricated material was performed in vitro. Incorporation of desired growth factors within the biopolymeric matrices was performed to provide an added efficacy for the angiogenic potential of such matrices.2 Research focus was also given for developing anti- microbial drug loaded matrices for treating microbial infested wounds.3

ANTIBACTERIAL AND ANTIOXIDANT EFFECT OF BIOGENIC CRYSTALLINE SILVER NANOPARTICLES SYNTHESIZED USING ZIZIPHUS OENOPLIA (L.) AQUEOUS LEAF EXTRACT

SoumyaSoman, J G Ray, School of Biosciences, Mahatma Gandhi University, Kottayam; EmailID:soumya860@gmail.com, jgray@mgu.ac.in

An important aspect of current nanotechnology research is the biological approach to synthesize metal nanoparticles using plant extract. Phytosynthesized silver nanoparticles have better inhibitory and antimicrobial effects compared to aqueous plant extract and silver nitrate. In the present investigation crystalline silver nanoparticles (AgNPs) with size of 10nm have been successfully synthesized using aqueous leaf extract (ALE) of Ziziphusoenoplia (L.), which act as both reducing as well as capping agent. The particles were characterized using UV Visible spectroscopy, HRTEM-EDAX & XRD. The synthesized AgNPs were evaluated for their free radical

scavenging ability using DPPH assay. An evaluation of the anti bacterial activity was carried out using Agar well diffusion method against four bacterial strains, Klebsiellapneumoniae, Pseudomonas aeruginosa, Escherichia coli & Salmonella typhi. The results demonstrated the effectiveness of synthesized AgNPs as antibacterial agents. This study brings out a green method of silver nanoparticle synthesis which is simple, rapid, eco friendly and reliable method which may have potential use in biomedical field.

COMPUTATIONAL STUDIES ON THE EFFECT OF 4-HYDROXY ISOLEUCINE IN TRIGONELLA FOENUM-GRAECUM FOR TYPE II DIABETES.

Steffimol Rose C.K, Rinimol Sebastian, Jeseena Mathew, Athira S, Anu.T.Vijayan, Betsy M.Baby, Stephen James and Harish R
Mar Athanasios College For Advanced Studies School of Biosciences, Thiruvalla, Email: betsy@macfast.org

The seeds of Trigonellafoenum- graecum (Fenugreek) is traditionally known for its medicinal properties and are used as ethnic food supplement to treat Type II diabetes.The presence of the phytochemical 4-hydroxy isoleucine has been previously reported to enhance insulin secretion under hyperglycemic conditions and also increase the insulin sensitivity of target cell receptors. Although, the antidiabetic properties of this unusual aminoacid has been well documented, their exact mode of action and target molecules are poorly understood. Hence, the molecular interaction of the compound with specific target muscle cells need to be elucidated. In the present study, molecular docking experiments of 4-hydroxy isoleucine with target cells were analyzed using tools like Pubchem, Corina, Hex and Swiss PDB Viewer.The number of Hydrogen bonds formed, binding energy etc were predicted using the tools Pubchem, Corina, Hex and Swiss PDB Viewer.

CLINICAL CHARACTERISTICS OF ADVANCED DEMENTIA

Sheetal S, Robert Mathew, Sauda, Department of of Neurology, Pushpagiri Instituite of Medical Sciences and Research Centre, Tiruvalla, Kerala

Clinical features of dementia change as the disease advances. Not only that, the existing deficits worsens and new deficits appear as well .The aim of our study is to compare the clinical features of advanced dementia with early stage of dementia. The method used is Cross sectional study. Data from patients availing dementia care services from 3 different tertiary care centres were analyzed. Data from 900 patients were analyzed. Neuropsychologic tests could be done in 283 patients and definite diagnosis was available in 279(29.7%) . Based on ACE R score, patients were divided into 3 groups. There were 140 patients with early dementia, 40 with advanced dementia and 103 in the intermediate group. The mean ACE R score in the groups were 66.66 SD9.7, 37.62SD7.2, 13.86SD7.3 respectively. The mean age was 69.88 (SD11.3).Mean age of early dementia group (67.27SD12.84) was significantly less than the advanced dementia group(73.42SD8.1),p <0.001.Females were less than males . Mean duration of illness was 2.84 years (SD 1.98) and was comparable across the groups (2.83,2.87,2.8 respectively). Mean education was 8.97SD 4.5.Education was significantly more in early dementia group (11.12SD3.53) than in advanced dementia(5.55SD4),p<.001. Significant correlation was seen between dementia stage and age as well as education even after controlling for each other (P<.001).No correlation with duration of illness(p - 0.94). Less educated patients tend to present at an advanced stage of cognitive impairment. Duration of illness at the time of presentation did not influence the degree of cognitive impairment probably because of the variability in progression of disease across various dementia syndromes.

MOLECULAR EPIDEMIOLOGY OF GROUP A ROTAVIRUS STRAINS FROM HOSPITALIZED CHILDREN IN CENTRAL TRAVANCORE.

Sincy George1, Jagan O.A1, Atul Philip Varghese1, Seema Oommen1, Sara Chandy1, Dr.Yogesh Dalvi2, Dr.Sushma Bai3
1Center for Virology, 2Pushpagiri Research Center, 3Department of Paediatrics, Pushpagiri Institute for Medical Sciences (PIMS), Thiruvalla.

Group A rotaviruses are the major cause of severe dehydrating gastroenteritis in young children globally. The purpose of this study was to estimate the burden of rotavirus disease in children and prevalence of circulating genogroups in Central Travancore region. Stool samples from children (n=50) less than 10 years with symptoms of acute watery diarrhoea were collected from hospitalized cases in Pushpagiri Medical College Hospital, Kerala. Among these, 34% (n=17) cases were positive for rotavirus A by antigen detection ELISA. Positive samples were subjected to reverse transcription PCR using VP6 gene specific primers and amplicons were sequenced. Phylogenetic analysis revealed that rotavirus A strains clustered as two distinct VP6 genogroups, I and II. While 94% of strains were clustered in genogroup II showing 98.89% bootstrap support and one case distinctly clustered

as genogroup I, with (97.50% bootstrap value).This study highlights the circulation of rotavirus infection in children in Travancore region of Kerala and emphasis the need for the implementation of vaccines.

PREVALENCE OF ENTERICALLY TRANSMITTED HEPATITIS VIRUSES IN CENTRAL TRAVANCORE: A PILOT STUDY

Soumya Jose1, Ritto Varghese2, Jagan O.A1, Reeshma R Nair1, Sincy George1, Seema Oommen11Center for Virology, 2 Pushpagiri College of Allied Health Science, Pushpagiri Institute for Medical Sciences (PIMS), Thiruvalla

Hepatitis-A virus (HAV) and Hepatitis E virus (HEV) infections are enterically transmitted diseases of public health importance in India. The aim of the study is the prevalence of HAV and HEV related hepatitis in Central Travancore, Kerala. Blood samples (n=168) from patients with suspected HAV/HEV infection (Jan 2014 to Dec 2014) were screened for anti-HAV IgM (Enzyme linked Fluorescent Assay (ELFA) VIDAS, BioMerieux, France) and anti-HEV IgM (ELISA Dia.Pro Italy). Seropositivity for HAV and HEV was 28.5%(47/165) and 5.7% (3/52) respectively. HAV seropositivity was highest in the age range 16- 30 years. HAV cases were higher during the latter part of the year (September and October). This study indicates the activity of HAV and HEV in Central Travancore. It also provides evidence for epidemiological shift of HAV infections from children (< 10 years) to young adults.

NEURO BEHAVIORAL ABNORMALITIES IN ADVANCED DEMENTIA

Abraham Verghese, Pushpagiri Medical College, Tiruvalla.

Neurobehavioral abnormalities vary at various stages of dementia. The aim of this study is to compare the neurobehavioral abnormalities of advanced dementia with that of early stage dementia. And the method used is Prospective cross sectional study. Data from patients availing dementia care services from 3 different teretiary care centres were analyzed. All the patients were administered a neuropsychology test battery comprising of ACER,MMSE,ADL,HADS,IQCODE,CBI,Trail A and B. 184 patients were selected during the period 2010 - 2014.Based on ACER score they were classified as stage 1(ACER ≥50 N 68),stage 2(ACER 25-49 N 49) and stage 3(ACER<25 N25). Patients in stage 3 were analyzed. Mean age was 72.41SD 9.8, 62% males. Mean education 6.47SD4.65.Mean duration of illness at the time of presentation 2.42(SD1.8) years. Cambridge behavioral inventory (CBI) was used to assess neurobehavioral abnormality. For purpose of comparison the total CBI score for each domain was converted into percentage of maximum score (PMS).The impairment at early stage as well as advanced stage was maximum for memory (PMS 46.91SD24.79vs48.07SD28.79). The least impaired domain was eating behavior at initial stages as well as in advanced stage (PMS21.61SD23.64vs21.87SD22.49). As cognition worsens worsening was seen in all the behavioral domains except in stereotypy and motor behavior .The amplitude of change was maximum for selfcare (14.43)and least for stereotypy and motor behavior(-0.90). No correlation was seen between ACER total score and CBI total score (Pearson correlation p0.45). In this cohort of hospital based patients with advanced dementia neurobehavioral abnormalities appear more or less similar to that seen in early stages.

INHIBITION OF INDOLEAMINE 2, 3-DIOXYGENASE (IDO) AMELIORATES IFN-Γ INDUCED TUMOR INVASION IN MCF-7 CELLS

Reshma C K*, Jayalekshmi H#, AthiraOmanakuttan#, Aditya Menon*, Bipin Nair#, ManeeshMailankot*@*Pushpagiri Research Centre, @Pushpagiri Institute of Medical Sciences, Tiruvalla, Pathanamthitta, #Amrita School of Biotechnology, Vallickavu, Kollam

The present study was designed to dissect out possible metabolic basis of cytokine induced tumor invasion in MCF-7 cells. MCF-7 cells were treated with IFN-ã &kynurenine (Kyn), +/- methyl tryptophan (MT) for 24 hrs in DMEM. To study the cell migration, cells were treated in 12 well plates followed by crystal violet staining. PCR was performed with gene specific primers for indoleamine 2,3 dioxygenase-1 (IDO1), IDO2 & tryptophan 2,3 dioxygenase (TDO). Western blot was performed for IDO1 using monoclonal antibody. Kynurenine was detected by reverse phase-HPLC. IDO1, IDO2 gene expressions were detected in MCF-7 cells, but not TDO. HPLC could detect peaks corresponding to Kyn. IDO1 protein was also detected. IFN-ã treatment induced IDO1 gene expression but not IDO2. There was increased cell migration in IFN-ã treated cells which was blocked by the MT treatment. Exogenous Kyn could also induce cell migration.The present results elucidate the role of kynurneine pathway (KP) in tumor invasion in MCF-7 cells. Blockade of KP could be a potential therapeutic target.

SCREENING OF BREAST CANCER RISK IN KUTTANADU: IDENTIFICATION OF MUTATIONS IN BRCA-2 GENE

Tiju Chacko, AdityaMenon, Sreevidhya C.P, Teejumajeed, C.K.K Nair, Pushpagiri Institute of Medical College and Research Centre, Tiruvalla 689101, Kerala, India

Breast and ovarian cancers are the most prevalent cancers in Indian women population. Mutations in BRCA-1 and 2 are implicated in most of the cases. BRCA-2 is a tumour suppressor gene which helps repair damaged DNA. It is involved in repair of chromosomal damage particularly error-free repair of DNA double strand breaks. Mutations in this gene have been related with an increased risk of developing cancers; breast and ovarian specifically. The present study is focused on screening population of Kuttanad for BRCA-2 mutations and identifying the type of prevalent mutations. DNA from blood samples were isolated and the amplification of several of the exons of BRCA-2 gene was carried out. Conformation Sensitive Gel Electrophoresis (CSGE) was performed on these samples revealed that 4 samples collected from the same family showed a similar type of mutation. Sequence analysis was performed to identify the exact location and type of mutation.

DIFFERENTIAL EXPRESSION OF HYPOXIA INDUCIBLE FACTORS IN RESPONSE TO SANAZOLE UNDER IN VITRO AND IN VIVO CONDITIONS

Sreeja.S a, SnehaVijayan b and C. K. Krishnan Naira a*Pushpagiri Institute of Medical Sciences and Research centre & b St. Berchman's College, Changanasserry

Hypoxia is one of the major physiological conditions responsible for the resistance in tumors to chemotherapy and radiotherapy. Sanazole (SAN), a hypoxia seeking compound, is found to sensitize hypoxic cells and get accumulated in tumors. In the present study the influence of SAN in hypoxia inducible factors (HIFs) under both in vitro and in vivo conditions were examined. The in vitro and in vivo experiments were performed in Dalton's Lymphoma Ascitic (DLA) cells and tumor- bearing Swiss albino mice, respectively. The expressions of genes were studied by real time PCR. During hypoxia, the expression of the genes- hif-1á, vegf and egfr-were up- regulated in DLA cells and tumor tissues. SAN up- regulates the expression of these genes under normoxia, while down-regulates under hypoxia. Radio- and chemotherapy sensitizing property of SAN could be due to the differential expression of HIFs in response to oxygen. As SAN get accumulated in hypoxic regions of tumor this compound could be used for specific targeting of drugs to tumor.

NEPHROPROTECTIVE ACTIVITY OF NARDOSTACHYS JADAMANSI

Lakshmi S1, Anila Rani2, Jinumol Joseph3,Sreelakshmi Harikrishnan3, Tiju Chacko1and C K K Nair1, 1Pushpagiri Research Centre; 2St.Mary's College for women, Thiruvalla,3 S .B College Changanassery

Cisplatin is one of the most widely used antineoplastic drug . The clinical use of cisplatin is limited by its nephrotoxicity and neurotoxicity. This study was to investigate the protective effects of Nardostachys jatamansi, against the nephrotoxicity induced by cisplatin in an animal model . Cisplatin caused severe nephrotoxicity in mice as evidenced from increased levels of Serum urea and creatinine and depletion in tissue antioxidant levels. Also lipid peroxides was higher in tissues. The oral administration of the extract of jatamansi (100 mg/kg, 200 mg/kg, 500mg/kg) to mice following cisplatin treatment resulted in restoring the elevated levels of urea and creatinine to normal levels and also prevented the depletion of tissue antioxidant levels. Also reduced the levels lipid peroxides in tissues. The action of cisplatin is mediated through oxygen radicals and jatamansi extract could be mitigating the toxicity by scavenging these free radicals .

MYELOPEROXIDASE, PARAOXANASE-1 AND LIPID PROFILE IN ATORVASTATIN VERSUS ROSUVASTATIN TREATED CHRONIC CORONARY ARTERY DISEASE PATIENTS.

Sowmya Varadhan1, Neethu M K2, Supriya Simon A3, 1Lecturer, Department of Biochemistry, Pushpagiri Institute of Medical Science and Research .Tiruvalla, 2Project Student, Department of Biochemistry, Pushpagiri Institute of Medical Science and Research .Tiruvalla, 3Asst. Professor, Department of Biochemistry, Pushpagiri Institute of Medical Sciences and Research Centre, Tiruvalla.

Myeloperoxidase (MPO) is a powerful pro-oxidative and proinflammatoryenzyme that plays an important role in the oxidative modification of LDL in the arterial wall. Oxidation of LDL which is a major event in development of Coronary Artery Disease can involve enzymatic and non enzymatic mechanisms. Human paraoxonase (PON1) is a calcium dependent HDL associated ester hydrolase responsible for the antioxidant and anti inflammatory properties of HDL which retards the oxidation of LDL by preventing the generation of lipid peroxides. The aim of this study is to evaluate and compare the lipid profile parameters in atorvastatin versus rosuvastatin treated chronic CAD patients. And compare the pleiotropic benefits of different types of statins by comparing the pro oxidant and pro inflammatory enzyme MPO and anti oxidant and anti inflammatory enzyme PON-1 in atorvastatin versus rosuvastatin treated chronic CAD patients. This study was conducted in Pushpagiri Institute of Medical Sciences and Research Center, Tiruvalla, Kerala for a period of 3 months.49 clinically proven chronic CAD patients on statin treatment (for >than 1 year)were categorized as GROUP 1 Chronic CAD patients with atorvastatin treatment (n = 30)GROUP 2 Chronic CAD patients with rosuvastatin treatment (n = 19)5 ml of peripheral blood was collected from each subjects for analysis of total cholesterol (TC),Triglycerides (TG), HDL Cholesterol (HDL-C), LDL cholesterol (LDL-C) ,Serum MPO and Serum PON .And the study shows atrovastatin treated patients have lower total cholesterol and LDL cholesterol levels than rosuvastatin treated patients whereas rosuvastatintreated patients have significantly higher levels of HDL-C .And the MPO value lower in rosuvastatin treated chronic CAD patients. And the PON 1 value high in rosuvastatin treated chronic CAD patients.TC and LDL/HDL ratio were significantly lower in chronic CAD patients on atorvastatin treatment compared to those on rosuvastatin treatment. LDL-C was also lower in Group 1.HDL-C was significantly high and VLDL was significantly lowered in chronic CAD patients on rosuvastatin .The present study suggests better antioxidant properties of rosuvastatin compared to atorvastatin .

✪ ORIGINAL ARTICLE

Role of prism III (paediatric risk of mortality III)score in predicting the outcome of children admitted in pediatric intensive care unit(PICU)

Nishad Ali K
Carol Sara Cherian
S Sushmabai
Rajeev A

From:
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla, India - 689 101

Nishad Ali K
Junior Resident

Carol Sara Cherian
Associate Professor

S Sushmabai
Professor & HOD

Department of Paediatrics
PIMS & RC

Rajeev A
Professor & HOD

Department of Community Medicine
PIMS & RC

Correspondence to:
Dr Carol Sara Cherian
E-mail: benjicarol@gmail.com

Abstract

This study aimed to assess the validity of PRISM III score in predicting the quality of clinical outcomes in critically ill children admitted in the PICU and to find the correlation of PRISM III scores with duration of PICU stay. Design: Prospective study. Setting: Pediatric Intensive Care Unit of a Tertiary level medical College. **Methods:** 315 children aged 1month to 15 years admitted in PICU from December 2012 to December 2013 were enrolled in the study. History, physical examination, provisional clinical diagnosis and PRISM III scoring at admission were performed and recorded . PRISM III scoring scale was applied for all patients in PICU in first two hours of PICU admission. Patient followed up and assessed at 6, 12, 24 hours by using clinical parameters of PRISM III. Relevant lab investigations were repeated in indicated patients . Statistical tests like chi square test, Paired and unpaired T tests through SPSS 17 software from the data on Microsoft office excel 2007 were applied. **Results:** Total 315 children satisfied the study criteria were enrolled in the study. Out of 315 children 193 (61%) were boys . Age and sex(demographic profile) did not influence the outcome significantly. Observed mortality rate was 3.2%. The area under the ROC curve for PRISM III scores at admission was 0.951, at 6 hours it was 0.965, at 12 hours it was 0.998 and at 24 hours it was 1.000 i.e. the maximum area under the ROC curve. At 24 hours for a score with a cut off of 9, the false positivity was zero with 100% sensitivity. According to the results of Hosmer Lemeshow goodness of fit test, the model of PRISM III designed in study has showed to be well fit. In our study the survival rate for a score between 0 to 9 was 100%, between 10 to 19 it was 0% and between 20 to 29 it was 0%, showing that if PRISM III score increases mortality chance increases. Pearson correlation showed a positive correlation of PRISM III score with PICU stay . PRISM III exhibited good discrimination and calibration in our study. **Conclusion:** PRISM III predicted the outcome in the PICU of our hospital and had good discrimination and calibration. According to Pearsons correlation there was definitely a positive correlation of length of stay with PRISM III scoring.

Key Words

PRISM III, PICU , Children, Length of PICU stay, ROC curve,Mortality, Discrimination; Calibration.

Introduction

PICU has a unique environment , because a wide spectrum of critically ill patients with various ages and diagnoses are admitted, where they are undergone different therapeutic interventions including administration of great variety of medications and doses.It makes PICU a potentially high risk system[1].

Clinical scoring systems are widely used in daily medical practice. Most of the time the physician uses clinical assessment scores to reflect current clinical status as well as probability of a clinical outcome.. Lack of consistency, reliability and accuracy in physician's subjective assessment necessitates formulation of quantitative clinical scores. This has led to the development of predictive scores to aid prognostication.Prognostic scores were developed to maximize prediction of the overall outcome among groups of

critically ill patients, given the severity of the patients.

Pediatric risk of mortality (PRISM) score allows for mortality risk assessment in the paediatric ICU. PRISM was developed from PSI to reduce the variables from 34 to 14 and number of ranges from 75 to 23 without losing the predictive power [2] It is institution independent and can be used within limits to compare different intensive care units. 151996 physiological variables and their ranges as well as diagnostic and other risk variables reflective of mortality risk were re evaluated by Pollack MM etal to update and improve the performance of second generation PRISM score. Thus PRISM III was developed to update and improve the performance of second generation PRISM score[3]. PRISM III is a risk adjustment tool for evaluation of intensive care which provides a comparative report to participating units . PRISM III is one of the best known scoring system for assessment of PICU performance[4]

This study was undertaken to use PRISM III as an extensive system to evaluate our health care service and provide a background for further advancement in health care quality control .

Aims

1. To assess the validity of PRISM III score in predicting the quality of clinical outcome in critically ill children in South central Kerala
2. To find the correlation of PRISM III scores with the duration of PICU stay.

Methodology

This study was a prospective observational study conducted in Pediatric Intensive Care Unit of Department of Pediatrics, Pushpagiri Institute of Medical Sciences and Research Centre, a tertiary care medical college hospital in Tiruvalla, Kerala, India. Enrollment of subjects was done from December 2012 to December 2013 (13 months)

All children more than 1 month and less than 15 years of age with medical problems admitted to PICU were included in the study.Re-admissions to the PICU during the same hospitalization was analyzed as separate subjects, as there was possibility of a different outcome at each admission.

Those patients who stayed less than 2 hours in the PICU, newborn infants less than 1 month and children above 15 years of age and all surgical cases were excluded in the study

Ethical and scientific committee clearance were obtained prior to commencement of the study

History, physical examination, provisional clinical diagnosis and PRISM III scoring at admission was performed on all the patients in PICU. Pre-structured proforma was used to record the relevant information (personal data, clinical findings, laboratory findings etc.) of the study population.. PRISM III scoring scale was applied for all patients in PICU within his/her first two hours of PICU admission. Appropriate treatment was implemented and modifed as per initial PRISM III . Initial PRISM III scoring done within 2 hours(as early as possible) and again at 6 hours ,12 hours and 24 hours. Progress of the disease was followed up clinically and by noninvasive monitoring parameters like Systolic Blood pressure, Heart rate ,Temperature,and lab parameters.

PRISM III parameters[10]

Parameters relevant to PRISM III are:

1. Cardiovascular vital signs:
a. Heart rate,
b. Systolic Blood pressure
2. Body Temperature
3. Neurologic vital signs:
a. Pupillary reflexes,
b. Mental status (Glasgow Coma Scale)
4. Acid-Base/Blood gases:
a. Acidosis,
b. Total CO2,
c. PH,
d. PaO2,
e. PCO2.
5. Chemistry tests:
a. Glucose,
b. Potassium,
c. Creatinine,
d. Blood Urea Nitrogen.
6. Hematology tests:
a. White blood cell count,
b. ProthrombinTime or Partial thromboplastin time,
c. Platelet count.

Associated conditions which also predict the outcome, like the use of ionotropes,mechanical ventilation and co morbid conditions like carcinoma were also recorded.

Patients were followed up and were assessed at 6, 12, 24 hours by using clinical parameters of PRISM III. Relevant lab investigations of PRISM III were repeated in indicated patients.

Patients fulfilling the study criteria were incuded in the study and PRISM III scoring were done at 2,6,12,and 24 hours and length of PICU stay and the outcome was recorded.

Statistical analysis

Cut off values for these outcomes were worked out by comparing the score and plotting it on ROC curve. The validity of these cut off points was assessed using Sensitivity, False positivity and area under Receiver Operator Characteristic (ROC) curve. The capacity of PRISM III to discriminate between survived and expired

was assessed by using ROC curve. The score was correlated with the duration of stay in the PICU using Pearson correlation and was also assessed using scatter plot. The correlation of PICU stay with subscores were done using Spearman's rho. Various other relevant statistical tests including Chi Square test, Paired and unpaired T tests through SPSS 17 software from the data on Microsoft office excel 2007 were applied to know the statistical significance. A p-value of 0.05 was taken as cut-off for statistical significance.

Results

Total PICU admissions during the study period was 913 subjects. 315 children in total who satisfied the study criteria were enrolled in the study.

Out of 315 children enrolled in the study , 193 (61%) were boys and 122 (39%) were girls.

Infants (less than 1 year of age) were 92 (29%), children 1 year to 12 year were 200 (64%) and 12 year to 15 year were 23 (7%). Among these first admissions were 308 (98%) and readmissions were 7 (2%).

Among the 315 children , 305 improved with treatment and 10 expired The observed mortality rate was 3.17%.

PRISM III Scores according to time intervals

At admission minimum score recorded was 0 and maximum score was 17 with a mean of 2 and standard deviation of 2.9.

At six hours of admission maintained same minimum and maximum scores with a mean of 1.90 and standard deviation of 2.86 and at 12 hours the maximum score became 21 with a mean of 1.54 and standard deviation of 2.95. At 24 hours score the maximum score was 24 with a mean score of 1.30 and standard deviation of 3.19.

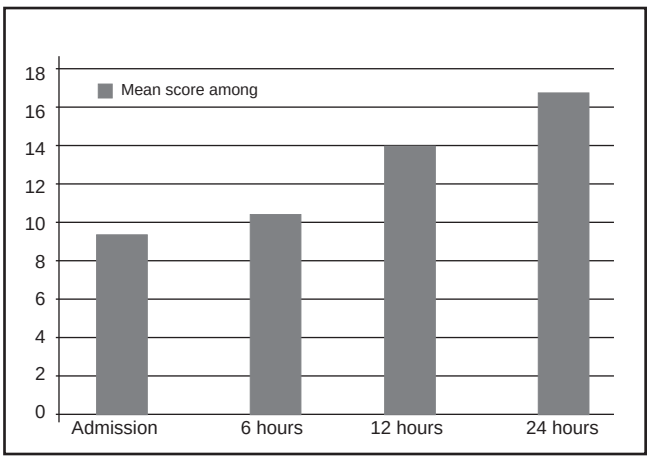
At admission the mean score among survived was 1.83 (SD 2.52) and among expired the mean score was of 9.40 (SD 4.32). At 6 hours the mean value among expired became 10.40 (SD 4.78) and at 12 hours among the expired the score became more worse with a mean score of 14 (SD 3.85). At 24 hours mean score among expired showed a worsening trend i.e,17.10 (SD 3.31). All the mean scores among expired were statistically significant. (p value:0.000). Refer table 1.1.

Table 1.1: PRISM III score in fixed time intervals in total subjects, in survivors and in expired subjects

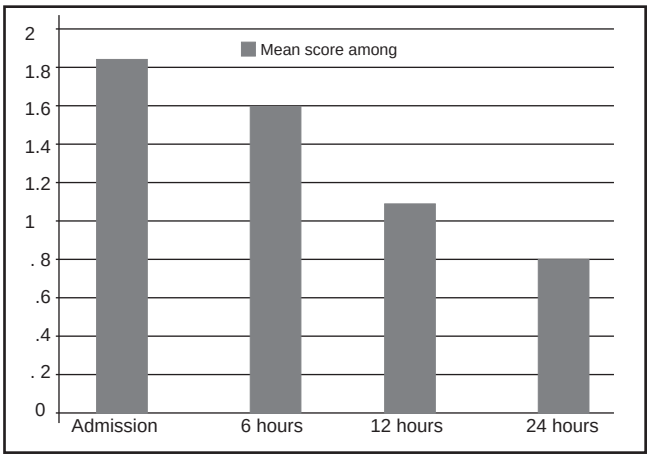
PRISM III score in fixed time intervals in total subjects					
	N	Minimum	Maximum	Mean	Std. Deviation
AD_Score	315	0	17	2.07	2.911
@6h score	315	0	17	1.90	2.866
@12_Score	315	0	21	1.54	2.955
@24_Score	315	0	24	1.30	3.196

PRISM III score in fixed time intervals in survivors and expired children					
	Outcome	N	Mean	Std. Deviation	Std. Error Mean
AD_Score	Survived	305	1.83	2.525	.145
	Expired	10	9.40*	4.326	1.368
@6h score	Survived	305	1.62	2.213	.132
	Expired	10	10.40*	4.789	1.514
@12_Score	Survived	305	1.13	1.817	.104
	Expired	10	14*	3.859	1.220
@24_Score	Survived	305	.78	1.320	.076
	Expired	10	17.10	3.315	1.048

*Student's T-test p = 0.000



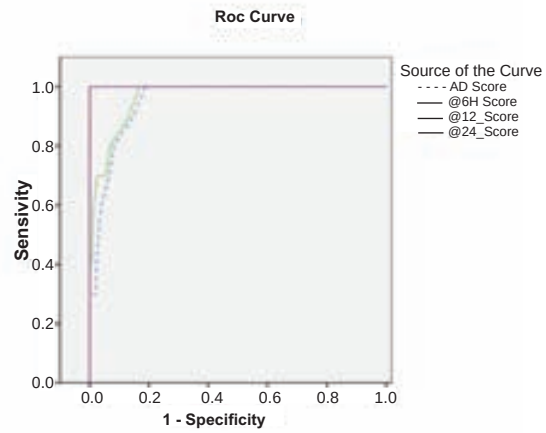
Graph 1.1: Mean PRISM III score among expired



Graph 1.2: Mean PRISM III score among survived subjects

According to the results of HosmerLemeshow goodness of fit test, the model of PRISM III designed in study has shown to be well fit for mortality rate in our PICU as goodness of fit value (P) 1.000 for infants,1.000 for children and 1.000 for total population. Using ROC curve , the capacity of PRISM III scoring for discrimination between survived and expired patients was analyzed and it was found that the PRISM III scoring was well suitable for the same .

Roc curve 1



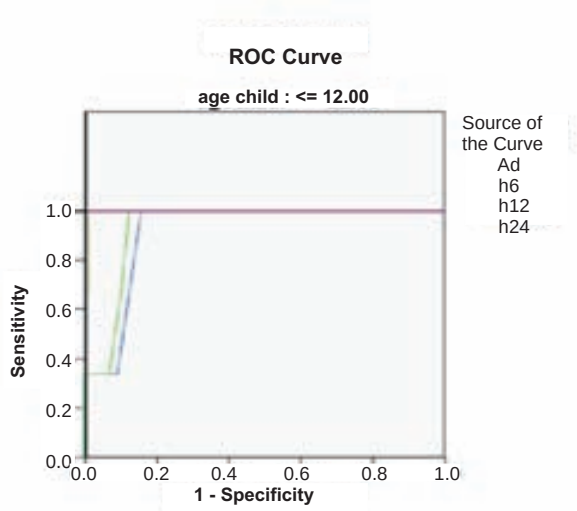
The ROC analysis showed a strong predictive power for PRISM III as depicted below:

- For total subjects area under the curve for PRISM III at admission was 0.951, at 6 hours is 0.965, at 12 hours is 0.998 and at 24 hours is 1.000, i.e. maximum area under the ROC curve was for 24 hours score.

Infants less than one year

For infants (1 to 12 months) area under the curve for PRISM III was 0.919,0.935,0.996,1.000 at admission, 6hours, 12hours,24hoursrespectively

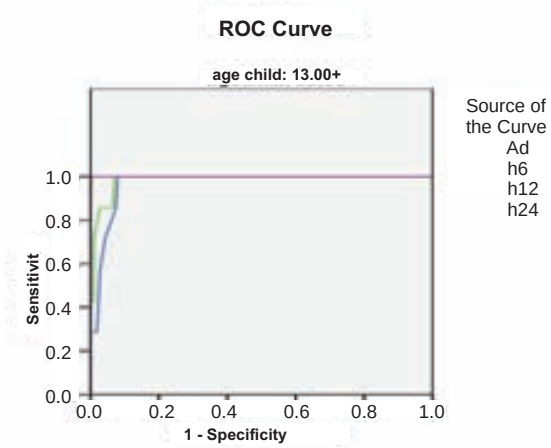
ROC curve 2 :For Infants 1-12 months



More than one year

For children (>1 year of age) area under the curve for PRISM III was 0.969,0.985.,1.000,1.000 at admission, 6hours, 12hours, 24 hours respectively.

ROC curve 3:For >12 months



Diagonal segments are produced by ties.

Table 1.2:Area under ROC curve

Area Under the Curve		
Test Result Variable(s)	Area	
Ad score	Cut off 3.5 Sens 100% FP 18.7%	.951
@6HSCORE	Cut off 3.5 Sens 100% FP 16.7%	.965
@12_SCORE	Cut off 3.5 Sens 100% FP 8.2%	.998
@24_SCORE	Cut off 3.5 Sens 100% FP 4.3% Cut off 9 Sens 100% FP 0%!!	1.000

Table 1.2 shows that discrimination at admission score was for a score of 3.5. It has got a sensitivity of 100% and False Positivity (FP) of only 18.7%. At 6 hours, the cut off score was same with 100% sensitivity and 16.7% FP. At 12 hours, cut off was same with a sensitivity of 100% and FP of 8.2%.

At 24 hours with a cut off of 3.5, sensitivity was 100% and FP was 3.5% and with a cut off of 9 the false positivity became 0 with 100% sensitivity. The same score at 24 hours has got a less false positivity compared to the other scores. It means that if the same score was persisting even at 24 hours, there is more chance of worst outcome compared to others

Length of stay in PICU

The average duration of PICU stay was 32.71 hours with a range of 139 hours (SD 21.74) for infants who survived and 154.67 hours, 170 hours (SD 85.19) respectively for those who expired.

The average duration of PICU stay was 31.94 hours with a range of 142 days and a standard deviation of 20.58 for children who survived and 71.29 hours,106, 40.41 respectively for those who expired.

Scatter plot 1:Admission score with length of stay in PICU

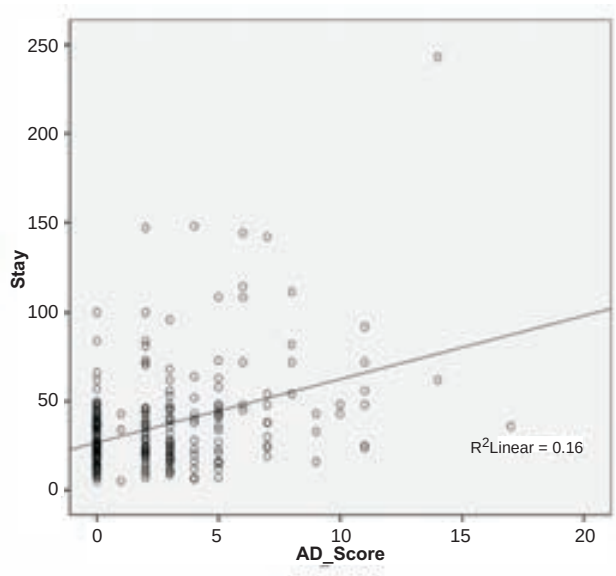


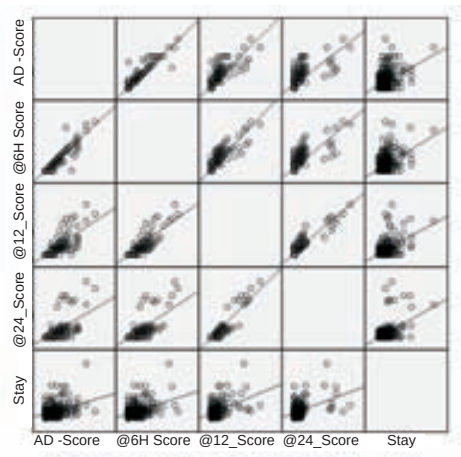
Table 1.3:Pearson correlation for length of stay in PICU:

Correlations						
		AD_SCORE	@6HSCORE	@12_SCORE	@24_SCORE	Stay
Stay	Pearson Correlation	.400**	.442**	.475**	.462**	1
	Sig. (2-tailed)	.000	.000	.000	.000	
	N	315	315	315	315	315
**. Correlation is significant at the 0.01 level (2-tailed).						

The above scatter plot and Pearson correlations showed positive correlations of PRISM III score with PICU stay and showed more positive correlation with 6 hour, 12 hour and 24 hours scores compared to score at admission and this positive correlations were statistically significant.

For a PRISM III score of 0 to 9 the mean PICU stay was 23 hours with a standard deviation of 24 and for a score of 10-19 mean stay was 43 hours with a standard deviation of 28. So as the PRISM III score increases the duration of PICU stay also is seen to be increasing

Scatter plot 2: Relation of admission score, 6 hours score, 12 hours score, 24 hours score and length of stay: .



The spearman's rho showed a significant association of PICU stay with neurological, ABG, chemistry and haematological subscores while with the cardiovascular subscore the correlation was not significant.(table 1.4)

Table 1.4: Spearman's rho for relation of subscores with length of PICU stay:

Spearman's rho		Stay
CV_SCORE	Correlation Coefficient	.096
	Sig. (2-tailed)	.90
	N	315
NEUR_SCORE	Correlation Coefficient	.134*
	Sig. (2-tailed)	.017
	N	315
ABG_SCORE	Correlation Coefficient	.118*
	Sig. (2-tailed)	.037
	N	315
CHE_SCORE	Correlation Coefficient	.146**
	Sig. (2-tailed)	.010
	N	315
HAEM_SCORE	Correlation Coefficient	.210**
	Sig. (2-tailed)	.000
	N	315

P * < 0.05 ** < 0.01

Discussion

In this study 193 (61.26%) were boys and 122 (38.74%) were girls with a M:F ratio of 1.6:1, which was comparable with other studies[6,7]. In the present study, age and sex (demographic profile) didn't influence the outcome significantly.Similar observations were noted in other Indian studies[11,12]and in the study done by S M Tibby et al in U.K[17].

The present study mortality rate was comparable to that of developed countries.Refer table 2.1

Table 2.1: Mortality rate

Studies	Pollack MM etal study(%) in USA 1997[20]	G H Tan et al Study(%) in Singapore 1997[8]	KMS Choi et al in Hong kong (%)2001-03[5]	Present study (%)2012-13
Mortality rate	4.9	4.5	2.6	3.1

Distribution of PRISM III score

Table 2.2: Score wise distribution of subjects

PRISM III score (24hrs)	Prashanth MR et al study (%) 2011 in Karnataka, India[9]	Present study (%) 2012-13
0-9	66	96
10-19	14	2
20-29	13	.6

Table 6.3 showed that the majority of subjects had a score of <9 and it was of same trend as that of Prashanth MR et al study[9].

In our study the survival rate for a score between 0 to 9 was 100%, between 10 to 19 was 0% and between 20 to 29 was 0%, Showed that if PRISM III score increases mortality chance increases. This was comparable with other studies[9].

Mechanical ventilation, ionotrope use and central venous cannulation had an influence in the outcome as evidenced by increased mortality in all the three[22,23,24]interventions. Similar observation was also observed in Hwang HS et al study[13] and Bellad R et al study[16].

For total subjects under curve surface area at admission is 0.951, at 6 hours it was 0.965, at 12 hours it was 0.998 and at 24 hours it was1.000 . Maximum area under the ROC curve was for 24 hours score.

For infants (1 to 12 months) under curve surface area was 0.919, 0.935, 0.996, 1.000 at admission, 6 hours, 12 hours, 24 hours respectively.

For children (>1 years), under curve surface area was 0.969,0.985.,1.000,1.000 at admission, 6 hours, 12 hours, 24 hours respectively.

All this AUCs are significant, because all values are >0.7.Refer table 2.3

Table 2.3: AUC by ROC distribution in total subjects, Infants and Children

Patients	AUC by ROC in CN Bilan etal[6] 2009 in Iran	AUC by ROC in present study 2012-13
Total patients	0.898	0.998
Infants	0.939	0.996
Children	0.890	1.000

In our study, the observed mortality rate was 3.17%. (10 died out of 315 subjects). PRISM III predicted a total death of 10 and observed mortality was 10.The predicted and observed deaths were similar in number (O/E ratio=1).The study done by G.R.Karambelkar et al[26] got an O/E ratio of 1.090 which is comparable to our study as revealed in Table2.4 and showed that PRISM III score is a good predictor of mortality.

Table 2.4: Observed and expected mortality comparison

PRISM III score	G.R. Karambelkar et al 2012[26] in Pune, India		Present study 2012-2013	
	Observed mortality	Expected mortality	Observed mortality	Expected mortality
0-5	1	4	2	2
6-10	3	4	5	5
11-15	3	2	2	2
16-20	3	1	1	1

The discriminatory power evaluated using ROC curve showed area under curve of 1 at 24 hours was in contrast with the observation by Qureshi AU et al study (0.78)[14]. Shann et al observed that the area under the curve of ROC equal to one is a perfect model, which was proven by our study also[15].

The PRISM III score with a cut off of 3.5 at admission, at 6 hours, at 12 hours and with a cut off of 9 at 24 hours in the ROC curve had good sensitivity and specificity in predicting the outcome.

The cut off of 9 at 24 hours scoring was comparable to GH Tan et al [8]study and was less than that of Ana Lilia Ponce et al study (cut off 13)[21]. Refer table 2.5.

Table 2.5: PRISM III cut off score

Studies	Ana Lilia Ponce et al study[21] 2003 in Mexico	G H Tan et al study 1997[8] in Singapore	Present study 2012-13
PRISM III cut off with maximum sensitivity and specificity	13	8	9

The reason for lower cut off and outcome related to it when compared to developed countries may be due to different illness profile in our study and other Indian studies[19].

The accuracy of present study with cut off of 15 was 99% which was better than G.R. Karambelkaret al[26]study (67%).

The PRISM III score at admission, 6 hours, 12 hours and 24 hours have well predicted the outcome and this was similar to GH Tan etal study[8].

The score helps to improve the quality of information given to parents about the possible outcome of the patient at the time of admission and possible length of PICU stay. This underlines the importance of scoring soon after admission (before 2 hours) and then at 6, 12 and 24 hours. Here the maximum area under the curve was for score at 24 hours and then for 12 hours as compared to 6 hours and at admission, eventhough all the scores have got good discrimination and calibration power. The study done by StephaneLeteurtre et al in France showed that at 4 hours PRISM III had good discrimination but calibration was poor[25].

Time intervals for scoring described in original PRISM III (i.e, at 12 hours and 24 hours) were best for predicting the outcome in the present study also,eventhough scores at admission and at 6 hours predicted the outcome reasonably well.

The mean length of PICU stay was 32.16 hours among survivors with a standard deviation of 20.89 and among non survivors the mean stay was 96 hours with a standard deviation of 65.76 which significantly affected the outcome. The length of stay in our study was less compared to other studies[18]and it may be due to early active interventions in view of early PRISM III scoring practiced in our study.Refer table 2.6.

Table 2.6: Comparison between length of stay in various studies

Studies	C N Bilan et al in Iran 2009[6]	Wang JN et al in Taiwan (2000)[18]	KMS Choi et al in Hong kong 2001-03[5].	Present study (2012-2013)
Length of stay in PICU	5 days(range 2-34 days)	4.52 days (range 1 day to 81 days)	3 days (range 0-186 days)	34.2 hours (range 5 hours to 10.1 days)

Pearson correlation showed a positive correlation of PRISM III score with PICU stay and showed more positive correlation with 6 hour, 12 hour and 24 hours scoring compared to admission scoring and this was statistically significant. For a PRISM III score of 0-9 the mean PICU stay was 23 hours with a standard deviation of 24 and for a score of 10-19 mean stay was 43 hours with a standard deviation of 28. So as the PRISM III score increases the duration of PICU stay was also increased.

The various sub scores mainly affecting the duration of stay in present study were Acid-basesubscore(p value 0.037), Neurological subscore (p value 0.017), chemistry subscore (p value 0.010) and Haematologysubscore(p value 0.000). All these were statistically significant.

In our study PRISM III score predicted the outcome of patients significantly and also showed positive correlation with length of PICU stay.

Conclusion

PRISM III predicted the outcome in the PICU of our hospital and had good discrimination and calibration. PRISM III score exhibited a good capacity to discriminate between survivors and nonsurvivors. It can also be used as a measure for prognosis of pediatric patients in PICU.

Even though the standard 12 hours and 24 hours PRISM III scoring showed superiority over admission and 6 hours scoring, the latter two also showed good discrimination and calibration. Present study also compared the PRISM III score with the length of stay in the PICU and it showed if PRISM III score was more the length of PICU stay was also more.

The various sub scores mainly affecting the duration of stay in present study were Acid-basesubscore(p value 0.037), Neurological subscore (p value 0.017), chemistry subscore (p value 0.010) and Haematologysubscore(p value 0.000

Limitations

- small sample size compared with original study
- shorter period of study
- single PICU study

Acknowledgement

Nisha Kurian –Assistant Professor Department of Biostatistics PIMS and RC

Bibliography

1. Scanlon MC, Mistry KP, Jeffries HE. Determining pediatric intensive care unit quality indicators for measuring pediatric intensive care unit safety. PediatrCrit Care Med. 2007 Mar;8(2 Suppl):S3-10.
2. Pollack MM, Ruttiman UE, Getson P R. The Pediatric Risk of mortality (PRISM) SCORE. Crit Care Med 1988; 16: 1110-1116.
3. Pollack MM, Patel KM, Ruttimann UE. PRISM III: an updated Pediatric Risk of Mortality score. Crit Care Med. 1996 May;24(5):743-52.
4. Bhadoria P, Bhagwat AG. Severity scoring system in paediatric intensive care units. Indian J Anaesth 2008;52suppl(5):663-75.
5. KMS Choi, DKK Ng, SF Wong, KL Kwok, PY Chow, CH Chan, JCS Ho Assessment of the Pediatric Index of Mortality (PIM) and the Pediatric Risk of Mortality (PRISM) III score for prediction of mortality in a paediatric intensive care unit in Hong Kong. *Hong Kong Med J 2005;11:97-103.*

6. Bilan N, Galehgolab BA, Emadaddin A, Shiva S. Risk of mortality in pediatric intensive care unit, Assessed by PRISM-III. Pakistan Journal of Biological Sciences, 2009, 12, 480.

7. De León AL, Romero-Gutiérrez G, Valenzuela CA, González-Bravo FE. Simplified PRISM III score and outcome in the pediatric intensive care unit.Pediatr Int. 2005 Feb;47(1):80-3.

8. Tan GH, Tan TH, Goh DY, Yap HK. Risk factors for predicting mortality in apaediatric intensive care unit.Ann Acad Med Singapore. 1998 Nov;27(6):813-8.

9. Prashanth M.R., Basavaraj G.V., Bharath Kumar Reddy K.R , Govinraj and Shivananda .prism iii score - an effective tool for the prediction of outcome ina pediatric inten-sive care unit.KarnatakaPaediatric Journal.2012April – June;Vol. 26, No. 2:5-7.

10. Kliegan, Stanton, St.Geme, Schor, Behrman, Chapter66,Nelson Text Book Of Pediatrics,18thedition.Vol1,Saunders,1996,387-388.

11. Thukral A, Lodha R, Irshad M, Arora NK. Performance of Pediatric Risk of Mortality (PRISM), Pediatric Index of Mortality (PIM), and PIM2 in a pediatric intensive care unit in a developing country. PediatrCrit Care Med. 2006 Jul;7(4):356-61.

12.. Khilnani P, Sarma D, Singh R, Uttam R, Rajdev S, Makkar A, Kaur J. Demographic profile and outcome analysis of a tertiary level pediatric intensive care unit. Indian J Pediatr. 2004 Jul;71(7):587-91.

13. Hwang HS, Lee NY, Han SB, Kwak GY, Lee SY, Chung SY, etal. Performance effectiveness of pediatric index of mortality 2 (PIM2) and pediatricrisk of mortality III (PRISM III) in pediatric patients with intensive care in single institution: Retrospective study.Korean J Pediatr 2008; 51(11): 1158-64.

14. Qureshi AU, Ali AS, Ahmad TM. Comparison of three prognostic scores (PRISM, PELOD AND PIM 2) at pediatric intensive care unit under Pakistani circumstances. *J Ayub Med Coll Abbottabad* 2007; 19(2):49-53.

15. Shann F. Are we doing a good job: PRISM, PIM and all that.*Intensive Care Med* 2002; 28(2):105-7.

16. Bellad R, Rao S, Patil VO, et al. Outcome of intensive care unit patients using pediatric risk of mortality (PRISM) score. *Indian Pediatr*2009; 46(12):1091-2.

17. Tibby SM, Taylor D, Festa M, Hanna S, Hatherill M, Jones G, *et al*. A comparison of three scoring systems for mortality risk among retrieved intensive care patients. Arch Dis Child 2002, 87, 421.

18. Wang JN, Wu JM, Chen YJ. Validity of the updated pediatric risk of mortality score (PRISM III) in predicting the probability of mortality in a pediatric intensive care unit.ActaPaediatr Taiwan. 2001 Nov-Dec;42(6):333-7.

19.. Thukral A, Lodha R, Irshad M, Arora NK. Performance of pediatric riskof mortality (PRISM), pediatric index of mortality (PIM) and PIM2 in apediatric intensive care unit in a developing country.PediatrCrit CareMed 2006, 7, 356.

20. Pollack MM, Patel KM, Ruttimann E. Pediatric critical care training programs have a positive effect on pediatric intensive care mortality. *Crit. Care Med.* 1997; 25 : 1637–42.

21. Ana lilia ponce-ponce de león, Gustavo romero-gutiérrez, Carlos aldanavalenzuela and Francisco ernestogonzález-bravo .Simplified PRISM III score and outcome in the pediatric intensive care Unit. *Pediatrics International* (2005)47, 80–83.

22.. Maulen-Radovan I, Gutierrez-Castrellon P, Zaldo Rodriguez R, Martinez Natero O. PRISM score evaluation to predict outcome in pediatric patients on admission at an emergency department. Arch Med Res 2006; 27(4):553-8.

23. El Nawawi A. Evaluation of the outcome of patients admitted to the pediatric insentive care unit in Alexandria using the Pediatric Risk of Mortality (PRISM) score. J. Trop Pediatr 2003; 49(2): 109-14.

24. Peters M, Dobson S, Novelli V, Balfour J, Macnab A. Sepsis and fever. In:MacnabAJ,Macrae DJ and Henning, RJ. editors. Care of the critically ill child. London: Churchill Livingstone 1999: 103-19.

25. Stephane Leteurtre, Francis Leclerc, Jessica Wirth, OdileNoizet, Eric Magnenant, Ahmed Sadik, Catherine Fourier and Robin Cremer. Can generic paediatric mortality scores calculated 4 hours afteradmission be used as inclusion criteria for clinical trials?, *Critical Care* 2004, 8:R185-R193.

26.G.R.Karambelkar*,S.V.Mane,S.R.Agarkhedkar,R.P.Karambelkar, S.S.Singhania, S.R. Kadam. The relevance of 24 hour PRISM III score in predicting mortality in pediatric intensive care unit.*Int J Pharm Biomed Sci*2012, 3(4), 214-219.

✪ ORIGINAL ARTICLE

Prevalance of childhood psychiatric disorders and substance use among adolescents.

Geethu Parvathy O
Joice Geo
Fazal Mohammed A M
Roy Abraham Kallivayalil

From:
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla, India - 689 101

Geethu Parvathy O
Intern

Joice Geo
Asst. Professor

Fazal Mohamed A M
Associate Professor

Roy Abraham Kallivayalil
Professor & HoD

Department of Psychiatry
PIMS & RC

Correspondence to:
Dr joice Geo
E-mail:

Abstract

Adolescence is the period when most of the psychological problems begins to set in. In this study, a single-informant type of Strengths and Difficulties Questionnaire (SDQ) with an impact supplement was used as a screening tool to identify the prevalence of overall psychiatric disorders and substance use among adolescents in a school from rural area of Pathanamthitta District of Kerala. The Multi-informant Type of SDQ identified individuals with a psychiatric diagnosis with a specificity of 94.6% and a sensitivity of 63.3%, while single informant type has substantially poorer sensitivity and specificity. Method:this was a cross sectional study in which 245 plus two students aged between 15-18 years, from a rural area of Kerala were assessed using the single informant type of Strengths and Difficulties Questionnaire and prevalence of substance abuse was also assessed. Results: 5.31% adolescents were found to have abnormal total difficulties score and 9.39% had borderline problems. A total of 14.7% were suffering from some abnormalities. Hyperactivity was most prevalent [10.2 %] among the studied sample, 6.1% had serious emotional problems, 7.3% subjects had abnormal conduct disorders and peer relationship problems in abnormal range were exhibited by only 3.7%. In case of prosocial behaviour, about 4.9% were in abnormal range. Hyperactivity had significant correlation [at 0.01 level] with emotional [0.261] and conduct problems [0.206] and emotional problems had significant correlation [at 0.05 level] with peer relationship problems [0.128]. There was significant conduct problems in males compared to females [p=0.028].The prevalence of substance use among the studied sample was, alcohol=2.4%; cigarettes=0.8%; pan masala=0.8% and others [including ganja, hashish etc.]=0.4%. However we could not establish any significant relationship between substance use and childhood psychiatric abnormalities.

Key Words

Strength and Difficulties questionnaire [SDQ]; Hyperactivity; Emotional Problems; Conduct disorders; Peer-relationship problems.

Introduction

Childhood is considered to be a happy idyllic period of life. Children are not thought to suffer from mental or emotional problems since they are not supposed to face the stress adults' encounter on a daily basis[1]. But it has been found that adolescence is the period when most of the psychological problems begin to set in[2, 3]. Major disorders seen among children and adolescents are:anxiety disorders, disruptive behaviour disorders, pervasive development disorders, eating disorders, elimination disorders, affective [mood] disorders, schizophrenia, tic disorders etc[4, 5, 6]. Adolescents with untreated mental disorders are highly prone to develop frank psychiatric illness as well as substance abuse in adulthood[7, 8].

Most of the researchers give importance to studies on mental illness in adults. However more studies are now coming forth regarding the psyche of adolescents[9]. Various researches from abroad as well as from India have shown that children do suffer from depression, manic-depressive disorders as well as anxiety disorders, which were once thought to be reserved for adults[10, 11]. But studies on incidence of

childhood mental disorders are extremely rare globally and there are only few from India[12, 13, 14]. Studies from India have shown that the annual incidence rate of psychiatric disorders during childhood and adolescents is 18/1000/yr[15].

It has been shown that 13.3% of children on average, has a diagnosis of psychiatric abnormality at any measurement point[16]. But the rates are higher when longitudinal studies are done[18]. By mid-adolescence although some disorders of childhood will disappear, impairing adult disorders such as depression, panic disorder, and SUD[substance use disorder] becomes more prevalent[19, 20].

Prevalence of mental disorders among children has been reported to be 14-20% in various studies[18]. According to World Health Report (2000), 20% of children and adolescents suffer from a disabling mental illness worldwide and suicide is the third leading cause of death among adolescents[21].

Incidence of substance abuse among adolescents has increased now. Various studies from foreign countries has revealed that, among the adolescents, 18.1% reported having eversmoked cigarettes, 47.8%reported having ever drunk any alcoholic beverages,5.0% reported having everused marijuana, and only 0.5% having ever used cocaine.Overall, 5.6% had ever used any illicit drug, 5.1% in the past year[22].

Childhood psychiatric disorders are common and treatable, but often go undetected and therefore remain untreated[23]. In this study, the Strength and Difficulties Questionnaire [SDQ] is used as a potential mean for detection of child psychiatric disorders in the community and thus to assess the prevalence of such disorders in a community sample[24]. In psychiatric clinical samples, diagnostic predictions based on the Strengths and Difficulties Questionnaire [SDQ] agree well with clinical diagnoses[25]. The Multi-informant Type of SDQ identified individuals with a psychiatric diagnosis with a specificity of 94.6% and a sensitivity of 63.3%.Sensitivity was substantially poorer with single informant rather than multi-informant SDQs[26, 27].

In this study, a single-informant type of SDQ with an impact supplement was used as a screening tool to identify the prevalence of overall psychic disorders among adolescents. Along with this, prevalence of substance use among adolescents was also assessed. This study helped to identify the likely cases with childhood psychiatric disorders to a certain extent even by a trained person, though a complete diagnosis is possible only with the help of a psychiatrist, so that such cases were identified at school and are referred early for further assessment and management.

Aims & Objectives

1. To assess the prevalence of conduct disorders, hyperactivity disorders and emotional disorders.
2. To assess prevalence of overall risk for any disorder among adolescents.
3. To assess the prevalence of substance use and its association with any of these childhood psychiatric disorders

Material and Methods

A cross sectional[prevalence] study was done. Subjects were adolescent students with an average age of 16 [15-18] (n=245) selected from a government aided school, which was randomly selected from a rural area of Kerala.

Instruments used include a structured questionnaire containing 1. Socio-demographic details: including ID, age, gender, occupation and education of parents and family income, 2. Details of substance use [alcohol, cigarettes, pan masala and others]. For analytical purposes they were grouped as follows: group 1- never used, group 2- once or twice [experimentally], group 3- occasionally, group 4- frequently, and single informant type Strengths and Difficulties Questionnaire [SDQ], with impact supplement.

Subjects were explained about the nature and purpose of study and informed consent was taken. The students were asked to fill up the structured questionnaire. SDQ asked about 25 attributes, some are positive and others negative. These 25 items have been divided between 5 scales [5 items each]:

1. Emotional symptoms- refers to emotional reactions
2. Conduct problems- refers to behavioral issues
3. Hyperactivity/inattention-
4. Peer relationship problems
5. Prosocial behavior- a measure of the child's ability to act prosocially, independent of the difficulties measured by the other subscales.

Scales 1 to 4 were added together to generate a total difficulties score [based on 20 items].The 'total difficulties score' will be generated by summing the scores from all scales except the prosocial scale. The scores were classified into normal borderline and abnormal. An abnormal score on total difficulties score was used to identify likely 'cases' with mental health disorders.

The data analysis was conducted using SPSS windows software [Version 17]. The categorical variables were tallied and presented as tables and percentages were worked out wherever necessary. The prevalence of individual problems and total difficulties

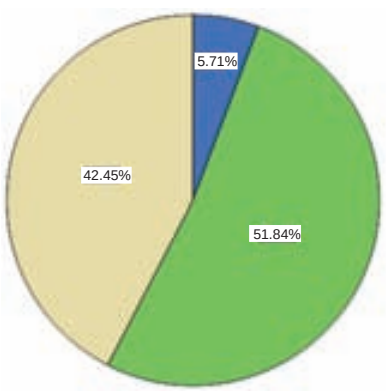
and prevalence of substance use were found out. Comparison between prevalence of such disorders and socio-demographic variables and substance use were carried out by chi-square tests and individual components are correlated by bivariate correlation. The p-value for ascertaining statistical significance was set at 0.05.

Observations & Results

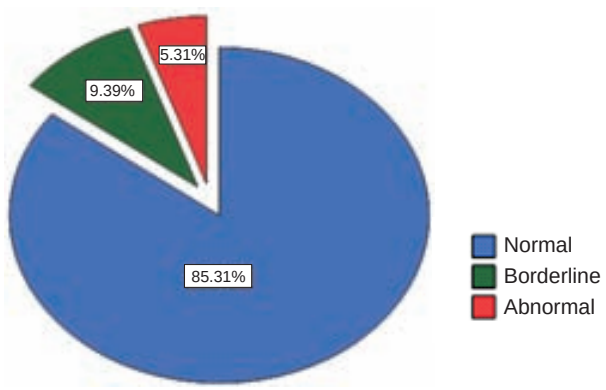
Study comprised of 245 students out of which 80[32.7%] only were males. A majority [161: 65.7%] of students in study were 16 years old as shown in table 1.

		Gender		Total
		Male	Female	
Age	15	8	10	18
	16	50	111	161
	17	20	43	63
	18	2	1	3
Total		80	165	245

Majority [127; 51.8%] belonged to middle class families as shown in fig 1.



Among the 245 subjects screened, SDQ identified that a psychiatric disorder is “unlikely” in 85.3% of the subjects. Only 5.31% [13 out of 245] were found to have abnormal total difficulties or “any psychiatric disorder” as shown in fig.2.



The following were the prevalence of individual problems

Table.2: Prevalence of emotional symptoms.

	Frequency	Percent
Normal	222	90.6
Borderline	8	3.3
Abnormal	15	6.1
Total	245	100.0

Table.3: Prevalence of hyperactivity disorders

	Frequency	Percent
Normal	197	80.4
Borderline	23	9.4
Abnormal	25	10.2
Total	245	100.0

Table.4: Prevalence of conduct problems

	Frequency	Percent
Normal	210	85.7
Borderline	17	6.9
Abnormal	18	7.3
Total	245	100.0

Table.5: Prevalence of peer relationship problems.

	Frequency	Percent
Normal	201	82.0
Borderline	35	14.3
Abnormal	9	3.7
Total	245	100.0

The prosocial scale was used to assess the socialization of children and the result were as follows.

Table.6: Prosocial scale.

	Frequency	Percent
Normal	213	86.9
Borderline	20	8.2
Abnormal	12	4.9
Total	245	100.0

The individual variables are correlated with bivariate correlation.

Table.7: Correlation between individual variables

		Emotional	Hyperactivity	Conduct	Peerl
Emotional	Pearson Correlation	1	.261**	.069	. 128*
	Sig. (2 tailed)		.000	.283	.045
	N	245	245	245	245
Hyperactivity	Pearson Correlation	.261**	1	.206**	.041
	Sig. (2 tailed)	.000		.001	.520
	N	245	245	245	245
Conduct	Pearson Correlation	.069	.206**	1	.052
	Sig. (2 tailed)	.283	.001		.418
	N	245	245	245	245
Peerl	Pearson Correlation	.128*	.048	.052	1
	Sig. (2 tailed)	.045	.520	.418	
	N	245	245	245	245
** Correlation is significant at the 0.01 level (2-tailed).					
* Correlation is significant at the 0.05 level (2-tailed).					

The prevalence of substance use among studied sample is shown in Table 8.

Table 8: Prevalence of substance use.

	Alcohol	Cigerette	Pan Masala	Others
Frequency	6	2	2	1
Percent	2.4%	0.8%	0.8%	0.4%

Gender-individual variable crosstabulations were done. The following findings were noted.[Fig 3. And Fig.4]

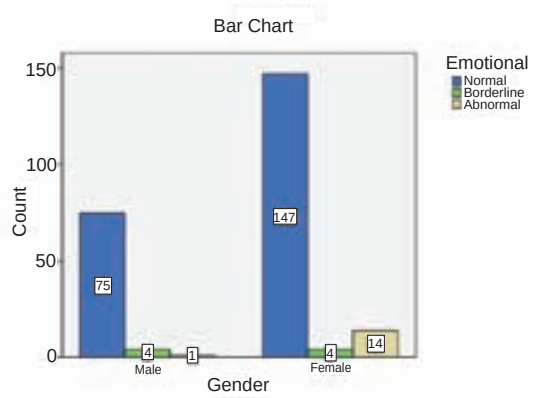


Fig 3. Gender-emotion crosstabulation [p=0.054]

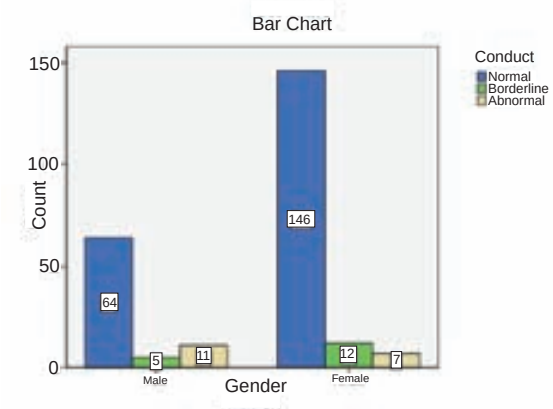


Fig.4: Gender-Conduct disorder cross tabulation.[p=0.028]

When substance abuse was cross tabulated with total difficulties score, alcohol and cigarette use does not show any significant association with presence of any disorder. But of the subjects who reported use of pan and other drugs, they all had some total abnormalities.

Table.9: Use of pan and relation to total difficulties[p=0.000]

		Total			Total
		Normal	Borderline	Abnormal	
Pan	Never	209	23	11	243
	Once or Twice	0	0	2	2
Total		209	23	13	245

Table10. Use of other illicit drugs and association with total difficulties [p=0.000]

		Total			Total
		Normal	Borderline	Abnormal	
Others	Never	209	23	12	244
	Once or Twice	0	0	1	1
Total		209	23	13	245

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .05.

Discussion

The aim of the study was to assess the prevalence of any conduct disorders, hyperactivity disorders and emotional disorders and overall prediction for 'any disorder' among adolescents and too assess the prevalence of substance use and its association with childhood psychiatric disorders. In the present study, out of 245 samples studied, about 5.31% were found to have abnormal total difficulties score and 9.39% comes under borderline category. That is, a total of 14.7% were suffering from some abnormalities. This percent is comparable with a study done byE.Jane Costelo in which it has been shown that 13.3% of children on average, had a diagnosis at any measurement point[16].

Another study by Brandenburg et al also found out that the prevalance ranges from 14-20%[18] which also correlates well with the finding of present study.

When individual symptoms were considered SDQ has identified that, among the sample studied, 6.1%[15] of the sample studied had serious emotional problems, while 3.3%[8] had borderline emotional symptoms. In case of hyperactivity disorders, about 10.2%[25] showed abnormal hyperactivity, while 9.4%[23] had borderline hyperactivity problems. 7.3%[18] subjects had abnormal conduct disorders and 6.9%[17] had borderline conduct problems. Peer relationship problems in abnormal range were exhibited by only 3.7%[9] subjects while 14.7% [35]actually had borderline peer problems.

The present study also shows that the most prevalent disorder among the individual items is hyperactivity[10.2%] and it is equally prevalent in both genders

In case of prosocial behavior, about 4.9%[12] were in abnormal range and about 8.2% [20] were borderline.

When these individual variables are correlated, it has been found that hyperactivity had significant correlation[at 0.01 level] with emotional[0.261] and conduct problems [0.206] and emotional problems had significant correlation [at 0.05 level] with peer relationship problems [0.128].

When gender emotional cross tabulation was done, it has been found out that 14 out of 165 females were having abnormal emotional disorders. [p=0.054]. In gender-conduct disorders cross tabulations, it has been found out that there is significant conduct problems in males compared to females. [p=0.028]

There was no significant relation between presence of “any disorder” with socio-economic status of the subjects [p>0.05].

The prevalence of substance use among the studied sample was, alcohol=2.4%; cigarettes=0.8%; pan masala=0.8% and others [including ganja,hasish etc] =0.4%

When substance use – total difficulties cross tabulations were done, it has been found that there was no significant relationship between alcohol and cigarette use reported and total difficulties, while out of 2 people who reveled to use Pan masala both of them had abnormal difficulties. [p=0.000] and one person who reveled to use other drugs [viz. ganja, cocaine etc] also had abnormal total difficulties[p=0.000].

As the sample size was too less [n=245] and reported cases of substance use were few, association between substance use and childhood psychiatric disorders cannot be assessed.

Conclusion

In the present study, 245 samples of plus two students aging between 15-18 years, from a rural area of Kerala were assessed using the Strengths and Difficulties Questionnaire[single informant; self-answering type] and prevalence of substance use was also assessed. It has been found that 14.7% of the sample had some psychiatric abnormalities of which 5.31% had significant total difficulties and 9.39% had borderline problems.Most prevalent disorder was hyperactivity irrespective of gender, which had significant correlation [at 0.01 level] with emotional [0.261] and conduct problems [0.206]. Emotional problems had significant correlation [at 0.05 level] with peer relationship problems [0.128]. There was significant conduct problems in males compared to females. [p=0.028]

As the sample size was too less [n=245] with unequal gender distribution and reported cases of substance use were few, association between substance use and childhood psychiatric disorders cannot be assessed.

Clinical Implications

Although childhood psychiatric disorders can be cured when treated appropriately and early while only a few develops such disorders in adulthood, most people are not aware of the need of detecting and treating such disorders. It is very important to seek treatment for a child who exhibits any symptoms of mental illness or else many such disorders can continue into adulthood and lead to problems in all areas in the person's adult life. Early uses of substances are also one of the major problems in the society. This study would be helpful to identify the likely cases of childhood psychiatric disorders, even by trained personal, by screening community samples and thus support and early treatment can be given so that complete cure can be ensured in most cases.

Also further extensive studies are required on this topic. Ideally this community study should be done in a huge sample and it requires future follow up, especially of those subjects who had abnormal scores, because these children are having a high possibility of developing adult psychiatric disorders and it can be prevented by early interventions, so that burden of such disorders in community can be reduced.

Limitations

Sample size was small with unequal gender distribution. Single informant self-answering type SDQ was used which had a low sensitivity compared to multi-informant type SDQ, hence some psychiatric illness which lack insight may be missed. Also reported cases of substance use were also less. Hence association between substance use and psychiatric illnesses could not be established.

References

1. Costello EJ, Angoid A. Developmental epidemiology. Developmental Psychopathology. Vol 1, NY: John Wiley & Sons; 1995:23-56.
2. Cohen P, Cohen J, Kasen S, Velez C N, Hartmark C, Johnson J, Rojas M, Brook J Streuning EL. An epidemiological study of disorders in late childhood and adolescence- age and gender specific prevalence. Journal of Child Psychology and Psychiatry. 1993; 34. 851-867.
3. Anderson J C, Williams S, Partridge F, Silva PA, Kelly J. DSM III disorders in preadolescent children: prevalence in a large sample from the general population. Arch Gen psychiatry. 1987;44: 69-77.
4. Brook, J. S., Whiteman, M. M., & Finch, S. Childhood aggression, adolescent delinquency, and drug use: A longitudinal study. Journal of Genetic Psychology, 1992;153. 369-383.
5. Block, J. H., Gjerde, P. E., & Block, J. H.Personality antecedents of depressive tendencies in 18-year-olds: A prospective study. Journal of Personality and Social Psychology, 1991;60. 726-738.
6. Brandenburg NSA, Friedman RM, Silver SE. The epidemiology of childhood psychiatric disorders: Prevalence findings from recent studies. J Am Acad Child Adolesc Psychiatry. 1990;29:76–83. [PubMed]
7. Boyle, M. H., & Offord, D. R. Psychiatric disorder and substance use in adolescence. Canadian Journal of Psychiatry, 1991;36. 699-705.
8. Robins LN: The natural history of adolescent drug use. AmJ Public Health. 1984;74. 656-657.
9. Steinhausen HC, Metzke CW, Meier M, Kannenberg R. Prevalence of child and adolescent disorders: The Zurich Epidemiological Study. Acta Psychiatr Scand. 1998;98. 262–71. [PubMed]
10. Garrison CZ, Waller JL, Cuffe SP, McKeown RE, Addy CL, Jackson KL. Incidence of major depressive disorder and dysthymia in young adolescents. J Am Acad Child Adolescent Psychiatry. 1997;36. 458–65

11. Burke KC, Burke JD, Regier DA, Rae DS. Age at onset of selected mental disorders in five community populations. Arch Gen Psychiatry. 1990;4. 511–8. [PubMed]

12. Lal N, Sethi BB. Estimate of mental ill health in children in an urban community. Indian J Paediatrics 1977; 4: 55-64.

13. Srinath S, Girimaji SC. Epidemiology of child and adolescent mental health problems and mental retardation. NIMHANS J 1999; 17: 355-66.

14. Hackett R, Hackett. L, Bakta P, Gowers S. The prevalence and association of psychiatric disorder in children in Kerala, South India. J Child Psychology Psychiatry 1999; 40: 801-7.

15. Savitha Malhotra, Adarsh Kohli, Mehak Kapoor, and Basant Pradhan. Department of Psychiatry, Postgraduate Institute of Medical Education and Research, Chandigarh - 160 012, India. Incidence of childhood psychiatric disorders in India Indian J Psychiatry. 2009 Apr-Jun; 51(2): 101–107.

16. E.Jane Costelo, Sarah Mustillo, Alaattin Erkanli, Gordon Keeler, Adriam Angold. Prevalance and development of Psychiatric disorders in Childhood and Adolescence. Arch gen Psychiatry/vol 60, Aug 2003; page 837-844

17. Fergusson DM, Horwood LJ, Lynskey MT. Prevalance and comorbidity of DSM III-R diagnoses in a birth cohort of 15 year olds. J Am Acad Child Adolescent Psychiatry. 1993;32. 1127-1134.

18. Brandenburg NSA, Friedman RM, Silver SE. The epidemiology of childhood psychiatric disorders: Prevalence findings from recent studies.J Am Academy of Child Adolescent Psychiatry. 1990;29:76–83. [PubMed]

19. Christie, K. A., Burke, J. D., Regier, D. A., Rae, D. S., Boyd, J. H, & Locke, B. Z. Epidemiological evidence for early onset of mental disorders and higher risk of drug abuse in young adults. American Journal of Psychiatry, 1988;145, 971-975

20. Canino G, Shrout PE, Rubio-Stipec M, Bird HR, Bravo M, Ramirez R, et al. The DSM-IV rates of child and adolescent disorders in Puerto Rico: Prevalence, correlates, service use, and the effects of impairment. Arch Gen Psychiatry. 2004;61:85–93. [PubMed]

21. WHO. The World Health Report 2000- Health Systems: Improving performance. Geneva: World Health Organization; 2000

22. Denise B. Kandel, Jeffrey G. Johnson, Hector R. Bird, Glorisa Canino, Sherryl H.Goodman,Benjamin B. Lahey, Darrel A. Regier,and Mary Schwab-Stone. Psychiatric Disorders Associated with Substance Use Among Children and Adolescents: Findings from the Methods for the Epidemiology of Child and Adolescent Mental Disorders (MECA) Study -Journal of Abnormal Child Psychology, Vol. 25, No. 2, 1997, pp. 121-132

23. World Health Organisation. The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research. 1993; Geneva: World Health Organisation.

24. Robert Goodman, Tamsin Ford, Helen Simmons, Rebecca Gatward, Howart Meltzer. Using the Strengths and Difficulties Questionnaire to screen for child psychiatric disorders in a community sample. British Journal of Psychiatry (2000), page 534-539.

25. Goodman R. Psychometric properties of the strengths and difficulties questionnaire. J Am Acad Child Adoelsc Psychiatry. 2001;40. 1337–1345.

26. Robert Goodman. The Strengths and Difficulties Questionnaire: A Research Note.Journal of Child Psychology and Psychiatry. 1997, July;38-5. 581–586

27. R. Goodman, H. Meltzer, V. Bailey. The strengths and difficulties questionnaire: A pilot study on the validity of the self-report version

✪ ORIGINAL ARTICLE

Effectiveness of Styloidectomy in Eagle's Syndrome - A Prospective analysis.

Gopinathan Pillai

Binu Babu

From:
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla, India - 689 101

Abstract

Aim: The purpose of this prospective analysis is to find out the effectiveness of styloidectomy for the treatment of Eagle's syndrome or stylohyoid syndrome. Methods: We conducted this study from August 2011 to October 2014. 15 patients who underwent surgery for Eagle syndrome were included in the study and followed up them for 6 months to 1 year. Strong clinical suspicion is essential for the diagnosis of this syndrome. When suspected radiological imaging helps to identify it. Pre operative X rays , orthopantamogram and Computed Tomography especially 3D computed tomogram , scan are valubable for the conformation . Pre operatively we found abnormality of elongated styloid process or calcified stylohyoid ligament in all 15 cases .Post operatively all the patients were followed up 6 months to 1 year, for their primary complaints and the quality of life changes. **Results:** Most common complaints preoperatively were foreign body sensation throat either right or left followed by throat pain , dysphagia and pain in the ear. Few patients had pain on the neck or facial pain on rotation of the neck. All the patients were relieved of their symptoms by the end of 6 months after the surgery and their quality of life were improved **Conclusion:**Styloidectomy is the treatment of choice for Eagle syndrome. Our approach were transoral transtonsillar styloidectomy under general anaesthesia . We did'nt come across any complications neither immediate nor late in the present study.

Gopinathan Pillai
Associate Professor

Binu Babu
Assistant Professor

Department of Otorhinolaryngology
PIMS & RC

Correspondence to:
Dr Gopinathan Pillai
E-mail: gopidrent@gmail.com

Key Words

Styloid process, Eagle syndrome, ossifications, stylohyoid ligament, styloidectomy Roland's Bouquet , Surgical outcome.

Introduction

The styloid process projects downwards , forwards and medially from the inferior surface of the temporal bone . It is a cylindrical bony outgrowth with a tapering tip. It emerges between the tympanic plate and the petrous part of the temporal bone of the skull bone. So the jugular foramen is just medial to it and stylomastoid foramen immediately behind it. The styloid process develops from the dorsal end of the Reicherts cartilage (the 2nd brachial arch cartilage).

The part of the cartilage between the styloid process and hyoid horn regress normally. The retained perichondrium forms the stylohyoid ligament extends from the tip of the styloid process to hyoid bone. Superior part of the body of the hyoid bone and the lesser horn also develops from the ventral part of the Reicherts cartilage[1] . The bony process of the styloid process is termed stylohyal and the embedded portion is tympanohyal , lesser horn and stylohyoid ligament is hypophayal [2] Reicherts cartilage also gives rise to stapes. The length of styloid process is variable ranging from a few millimetres to an average of 25mm. The styloid process elongation can be assumed either the styloid process or the adjacent stylohyoid ligament ossification show an over all length of more than 30 mm. 4-7% general population have elongated styloid process . 4% population with elongated styloid process or normal styloid process with calcification of stylohyoid ligament are symptomatic[3,4]. Studies in India have shown that 19.4 – 52.1% general population have

elonagted styloid process[5]. Styloid process gives origin to stylohyoid ligament , stylomandibular ligament and 3 muscles – stylohyoid , stylopharyngeus and stylogossus forming a bouquet. "RIOLAND'S BOUQUET". The muscles have variable sites of origin on the styloid process and nerve innervations . Stylopharyngeus arise from the base ,stylohyoid muscles from the middle and styloglosses from the tip of the styloid process . These 3 muscles are innervated by glossopharyngeal nerve, facial nerve and hypoglossal nerve respectively[6].

Ossification of the stylohyoid ligament.

The stylohyoid ligament extent from the tip of styloid process to the lesser horn of the hyoid bone , medial to the ligament , in close association , are the internal carotid artery , internal jugular vein and last four cranial nerves. The external carotid artery lies lateral to the ligament[8] . The ligament lies posterior to the tonsillar fossa and lateral to the pharyngeal wall. Ossification of this ligament and elongation of the styloid process produces the symptoms of dysphagia(80%), pharyngeal foreign body sensation (55%) and a constantly aching throat(40%)[9] Other symptoms include otalgia , headache, pain on neck rotation and facial pain[10]

Ossification of stylohyoid ligament is reported in only 1.4% of the population with no sex predilection but very few individuals are symptomatic . The syndrome is rare in persons under 30years of age and is most frequent in the 40-80 years of old age range; women are symptomatic more often than men[11,12] . The radiographic identification of stylohoid ossification is readily apparent on plain radiographs , coronal CT images , Reformatted CT is visually impressive.

Stylohyoid syndromes:

Three dominant syndromes are related to stylohyoid structures : Eagles' syndrome , styloid carotodynia , and hyoid fascitis[20] .

Eagle's syndrome: In 1937 Eagle described the elongated styloid process syndrome which consist of pharyngeal pain , referred otalgia and foreign body sensation in the throat occurring after tonsillectomy . It is believed that this syndrome develops because of the fibrosis tonsillar fossa and elongated styolid process; hence the symptoms are greatest during phonation , deglutition and deep inspiration . Since the time of this discovery the term Eagle's syndroms has come to be applied to any patient who has these symptoms and with an abnormally elongated styloid process whether or not there has been prior tonsillectomy surgery. Complicating the picture even more is that some patient also have ossified stylohyoid ligament which can themselves fracture and become symptomatic[14] . The treatment , if any , is surgical removal of the elongated styloid process (es)[2,3,4,5,6] . Because there are many asymptomatic patients , some clinicians question the validity of Eagle's syndrome . Nonthless , the symptomatic patients , shortening of styloid process usually relives their complaints . The presence of an elongated syloid process also makes this structure more prone to injury during flexion- type , trauma[1]. The symptoms related to Eagle's syndrome can also be confused with those of facial neuralgia , oral , dental and tempromandibular diseases

Styloid Carotodynia .

In which the elongated styloid process tip is deviated so that it causes pressure on either internal carotid artery which lies medially or external carotid artery lies laterally . This pressure causes stimulation of the pain –sensitive receptors found in the adventitia of these vessels and results in pain along the distribution of the affected vessels. Styloidectomy relieves the pressure effect on the vessels and disappearance of the symptoms .

Hyoid Fascitis

The head turning stretches, the muscles between the hyoid bone and the styloid process which produces pain . it is due to tendinitis or facilities of the muscles attached to the styloid process viz , styohyoid, stylopharyngeus and styloglossus . Styloidectomy reduce the stretching on turning the head . So the symptoms are relieved.

Materials and Methods.

One hundred and twelve patient presented to the OPD department of ENT ,Pushpagiri Institute of Medical Sciences & Research Centre , Thiruvalla between 2011August to October 2014 with complaints of foreign body sensation of throat , throat pain , dysphagia , Vague pain in the ear and facial pain. All these patients were clinically, endoscopicaly and radiographically examined. Of these one hundred and twelve patients 97 patients were diagnosed to have Gastroesophagel reflex disease chronic tonsillitis , early malignancies, spontaneous tonsillolith , paraphargeal benign tumours and tempromandibular joint diseases . A total of 15 patients were found to have elongated styloid process

or calcified stylohyoid ligament.8 cases have bilateral and 7 cases have unilateral elongated styloid process either left or right there by causing clinical signs and symptoms. Eleven were females and four were males. The diagnosis of elongated styloid process were confirmed by palpating the tonsillar fossa . All these patients underwent a course of medical treatment but without any relief symptoms on stoppage of drugs. A radiological correlation were done with X –ray , Orthopantamogram , computerised tomogram with three dimensional reconstruction . Those cases were taken up for styloidectomy under general anaesthesia. All patients underwent transoral transtonsillar approach. The steps in the surgery included conventional

tonsillectomy by dissection and snare method. The styloid process was identified deep to the superior constructor muscles and skeletonised. The muscular attachments to the styloid process were identified and separated from the styloid process and the styloid process excised using bone nibbler ,. The separated superior constrictor muscle was approximated with sutures and good hemostasis was achieved. Post operatively patients were treated with broad-spectrum intravenous antibiotic, antinflamtory and analgesic agents and betadine gargle .The intra-operative problems were

1. Difficulty in skeletonising the styloid process
2. Difficulty in lateralized styloid process
3. Bleeding from the tonsillar fossa

Twelve patients had styloid process measuring between 30-35mm. Three patients had 25-30 mm but they had stylohyoid ligament calcification .In all the bilateral process the length of the styloid process were unequal on either side. All the patients were followed up at monthly intervals for a period of six months to one year. At the end of six months all the patients in the present study were the relieved of their symptoms.

Prospective analysis of all the 15 patients who have undergone styloidectomy from 2011August to 2014 October. Details are the patients given below

Sl.No	Age	Sex	Presentation	Bilateral/ Unilateral	Follow up
1	58	Male	Foreign body sensation throat &ear pain dysphagia	Bilateral	1 year
2	55	Female	Foreign body sensation and dysphagia	Right	1 year
3	58	Female	Foreign body sensation throat and dysphagia	Bilateral	1 year
4	40	Female	Foreign body sensation and dysphagia	Bilateral	6 months
5	57	Female	Foreign body sensation and throat pain	Left	9 months
6	49	Female	Dysphagia, pain in the ear	Bilateral	6 months
7	30	Female	Foreign body sensation throat	Right	6 months
8	67	Female	Foreign body sensation dysphagia	Bilateral	9 months
9	66	Female	Foreign body sensation pain, throat	Right	1 year
10	51	Male	Foreign body sensation throat	Bilateral	1 year
11	49	Male	Dysphagia, Foreign body sensation	Bilateral	6 months
12	59	Female	Dysphagia, pain in the ear	Bilateral	9 months
13	41	Female	Dysphagia	Right	10 months
14	50	Female	Foreign body sensation throat	Right	6 months
15	28	Male	Throat pain, Foreign body sensation	Right	6 months

Sex	Numbers
Male	4
Female	11
Total	15

Age	Numbers
20-40 years	3
40-50 years	3
50-60 years	7
60-70 years	2
Total	15

Sides	Numbers
Bilateral	8
Right	6
Left	1
Total	15

Discussion

The styloid process is a thin cylindrical bony projection tapering towards the apex(styloid: stick like)orginates from the under surface of the temporal bone averages about 2to 3 cm length in adults and projects downwards , forwards and medialy. Styloid foramen is situated between the mastoid processs and the styloid process. This foramen constitutes the terminates of bony facial nerve canal . Embryologically styloid process develops from the Reichter's cartilage , the second branchial arch. It gives origin to the stylohyoid ligament and 3 muscles – stylopharygeus , stylohyoid and styloglossus forming a bouquet , “RIOLAND's BOUQUET” . The stylohyoid ligament extents from the tip of styloid process to the lesser cornu of the hyoid bone. The apex of the styloid process is located between the internal and external carotid arteries ,just lateral to the tonsillar fossa within the lateral pharyngeal wall .These muscles are innervated by Glossopharyngeal nerve,the facial nerve and the hypoglossal nerve respectively [IJO 264] .The stylohyoid ligament may contain cartilage remnant , so it may be ossified partially or completely and can cause pain and other symptoms in symptomatic cases[IJL 3,7,264] . Then it is known as either Eagle syndrome or elongated styloid process syndrome or styloid process carotid artery syndrome or stylohyoid syndrome or styloid process neuralgia[5] .

Eagle reported several cases of styloid process syndrome occurring in the post tonsillectomy cases[8] . It may be due to inflammatory changes occurring in the adjacent nerves or artery . Eagle syndrome diagnosis is applied only when ossification develops after the trauma with accompanying symptoms. Eagle syndrome may be caused by tonsillectomy or traumatic fracture of the mineralized stylohyoid ligament [(10)] . Diagnosis can be made by digital palpation of the styloid process in the tonsillar fossa, which exacerbates pain ,with X –ray and CT work- up.2 to 4 percent of general population have radiological evidence[13] .The estimated radiographic length of styloid process is 22 to30 mm. Spatial position of tip also is the cause of symptoms[4,8] .

Majority of the patients have no history of tonsillectomy or severe pharyngeal trauma[13-15]. It may be misdiagnosed as salivary gland disease , otitis media, temperomandibular myofacial dysfunction glossopharyngeal neuralgia or tempromandibular joint problems[7,16]. The treatment options are surgical removal of the styloid process or ossified stylohyoid ligament [7,17] through transoral trans tonsillar approach or external lateral neck approach or combined approach in difficult cases as in trismus [15] . Thromosis of internal carotid artery is a dreaded complication.

Conclusion

Elongated styloid process is an anatomical variant . Glossopharyngeal neuralgia is a part of the Eagle syndrome. Psychological status of the patients should be assed to rule out malingering Eagle syndrome is a rare cause for dysphagea . Diagnosis could be missed unless the tonsillar fossa is palpated . Radiological co- relation is mandatory like X ray neck lateral& AP view , modified Town's view , Orthopandamogram , CT scan of neck , 3D CT helps in planning of surgery. The disappearance of the symptoms after styloidectomy is the final confirmation.

References

1. Blomgren K , Qvarnberg Y, Valtonen H spontaneous & Fracture of an ossified sytlohyoid ligament . J Larngol Otol 1999;113:854-855
2. Miller DB. Eagle's syndrome and the trauma patient . Significance of an elongated styloid process and /or ossified stylohyoid ligament [in process citation]Funcet Orthod 1997 ,14:30
3. Yetiser S Gerek M , Ozkaptan Y . Elongated styloid process .Dignostic problems related to symptomatology. Cranio 1997 ;15:236-241
4. Dupont JS Jr Panoramic imaging of the styloid complex in patients with suspected Ernest or Eagle's syndrome .Carnio 1998;16:60-63.
5. Lugmayr H.Krennmair G, Lenglinger F. Ossification of the stylohyoid chain in computerized tomography –Eagle syndrome . Aktuelle Radio 1997 :7:331:332
6. Miyar V .Morais D Santos J (Surgery of the elongated styloid syndrome .An otorhinolaryngology lbero Am 1997 :24:303-307
7. Baugh RF, stoeks RM .Eagle's syndrome a reappraisal .Ear nose throat J 1993 :72:341-344
8. Lorman JG ,Biggs JR. The eagle syndrome .AJR 1983:140:881-882
9. Lavine MH , stoopack JC , Jerrold TL calcification of stylohyoid ligament . Oral surg oral Med oral pathol 1968 .25 55- 58
10. Gossman JR , Taristano JJ . The styloid –stylo hyoid syndrome . J oral surgery 1977 :35 :555:560
11. Correll RW , Jensen JL , Taylor JB , Rhyne RR Minerlization of the stylohyoid ligament a radiographic incidence study . Oral surgery 1979:48 : 286-291
12. Keur JJ , Campbell JP , Mec Carthy JJ , Ralph WJ . The clinical significance of the elongated styloid process : Oral surg oral med oral patho of 1986- 61 . 399-404
13. Ahmat S , Lokman U ,Three dimensional CT of Eagle's syndrome . Diagm interv Radiol 2005;11 206 – 209
14. Deepika Raina , Rajesh Gothi , Sriram Rajan . Head & Neck radiology . Eagle syndrome Indian J Radiology imaging 2009 19:2
15. Godder Dr. Adam S Wrodwards RT Eagle syndrome .BR Dent J 1999 ; 186 – 489 -90
16. Oluseisi AD .Traumatic Eagle syndrome : Does neck trauma result in complete ossification in partially ossified stylohyoid ligament .int.J.Otorhinolaryngology 2006 .4:2.
17. Ryan MD CT findings associate with Eagle syndrome .AJNR .AMJ Neuroradiol 2001 :22:1401-2
18. Camarada AJ , Deschampsc, Forest D11 1989 stylohyoid chain ossification : A discussion of etiology . Oral surg oral med Oral pathol 67: 515 – 20
19. Balbuena L Jr , Hayes D , Ramirez SG Johnson R (1997) Eagle's syndrome (elongated styloid process) south med J 90:331-14.
20. Peter M Som , Hugh D Curtin . Head & neck imaging 4th edition vol II 1984: 2199- 2202.

✪ ORIGINAL ARTICLE

Characteristicsof children and adolescents attending child and adoloscent clinic in a tertiary care teaching hospital

Fazal Mohamed A M
Joice Geo
Ashitha M L
Abraham Verghese
SanuSudhakar
Roy Abraham Kallivayalil

From:
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla, India - 689 101

Fazal Mohamed A M
Associate Professor

Joice Geo
Asst. Professor

Ashitha M L
Jr. Resident

Abraham Vergheses
Sr. Resident

Sanu Sudhakar
Sr. Resident

Roy Abraham Kallivayalil
Professor & HOD

Department of Psychiatry
PIMS & RC

Correspondence to:
Dr Fazal Mohamed A M
E-mail: fazalam2006@yahoo.com

Abstract

Background: Child psychiatry is a specialized branch of psychiatry in which more research need to be focussed to provide in depth care. This gains more significance considering the fact that many of the psychiatricdisorders identified in the adults first appears in childhood and adolescence. **Method:** Consecutive new patients attending the child and adolescentclinic (CAC) at the Department of Psychiatry, Pushpagiri Institute of Medical Sciences were assessed for their psychiatric diagnosis, socio demographic details and pathway to care. **Results:** Attention Deficit Hyperactivity Disorder (ADHD) and Conduct disorder were the 2 most common diagnoses. Parents were the most common source of referral to the CAC.

Introduction

Child and adolescent psychiatry is a branch of psychiatry which specializes in the study, diagnosis, treatment and prevention ofpsychopathological disorder of children, adoloscents and their relatives. An important antecedent to the specialty of child (paediatric) psychiatry was the social recognition of childhood as a special phase of life with its own developmental stages, starting with the neonate and eventually extending through adolescence[1]. Kraepelin's psychiatric taxonomy published in 1883, ignored disorders in children[2].Johannes Trüper, TheodorZiehenand Wilhelm Strohmayer were the earliest pioneers in the field of child psychiatry. In 1936, Kanner established the first formal elective course in child psychiatry at the Johns Hopkins Hospital[2].Michael Rutter carried out the first comprehensive survey of 9 -11 year old children in 1970[3]. It addressed questions important to child psychiatry.

Psychiatric assessment in a child psychiatry clinic starts with a detailed history, taken from important persons in the child's life which may range from the parents to the caregivers including neighboursand teachers. Collateral information is usually obtained from the child's school with regards to academic performance, peer relationships, and behaviour in the school environment[4].The median prevalence estimate of functionally impairing child and adolescent psychiatric disorders is 12%[5].Many of the life time psychiatric disorders may first make its appearance in childhood or adoloscence[5].

Aim

1. Identify the psychiatric disorders of patients attending the child and adolescent clinic (CAC) of Department of Psychiatry, Pushpagiri Institute of Medical Sciences and Research Centre (PIMS& RC).
2. To identify the characteristics of the psychiatric disorders identified

Materials and Methods

Setting

The study was conducted at the Child and Adolescent clinic (CAC), Department of Psychiatry, Pushpagiri Institute of Medical Sciences and Research Centre (PIMS & RC). PIMS & RC is a tertiary care post graduate teaching and research centre. The department of psychiatry is a post graduate teaching department. The child and adolescent clinic is a speciality clinic run by the department. CAC caters to children and adolescents upto the age of 16 years.

Study Design

Cross sectional

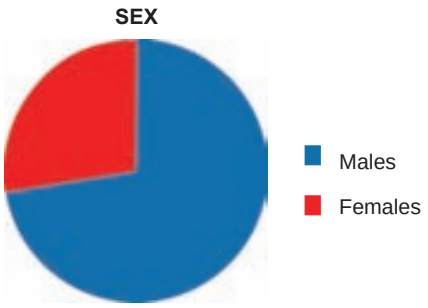
Methods

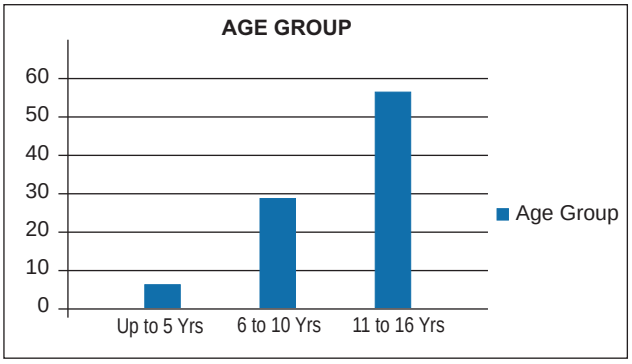
Consecutive new patients attending the CAC from 01/01/2015 to 31/12/2015

were included in the study. An intake proforma prepared for the CAC is filled for all the patients. The intake proformaincludessociodemographic status, illness details, family history, medical comorbidities and pathways to care. 'Teachers report' in a prescribed format for the CAC is also collected from each patient. Diagnosis of the patient's condition is made using ICD10.

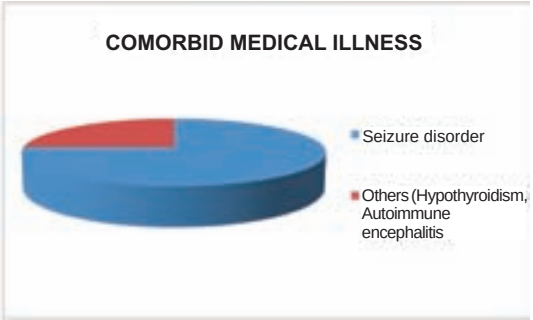
Results

[Children belonging to the age group of 0- 16 years are included in the study.] A total of 93 patients were included in the analysis which included68(73.1%) malepatients and 25(26.9%) female patients. The mean age of children attending CAC is 11.5 years with 7(7.5%) patientsbelong to agegroup 0- 5 years, 29(31.2%) patients belong to the age group of 6-10 yearsand 57(61.3%)patients belong to age group 11-16years

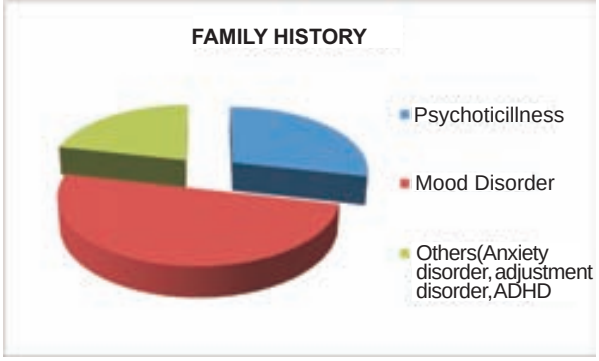




28(30%) children had positive family history of psychiatric illness with psychotic illness in 28.5%, mood disorder in 50% and others (Anxiety disorder, Adjustment Disorder, ADHD)21.5%.

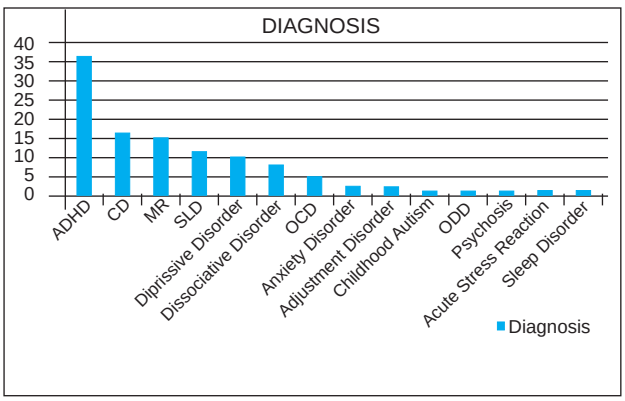


Only 8 children have comorbid medical illness of which 6 had seizure disorderand1patient each had hypothyroidism and autoimmune encephalitis

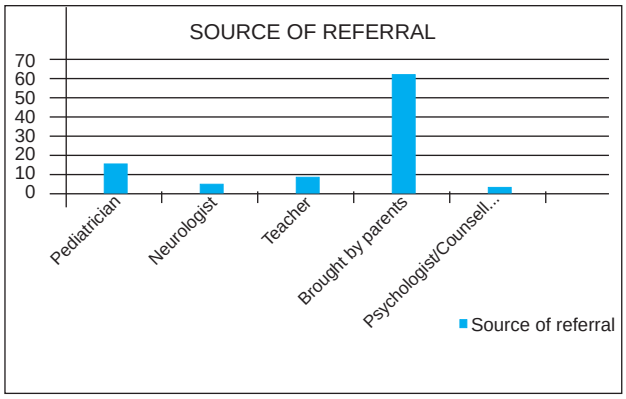


Teachers report was available for 40 out of the 93 patients.Ofthe 40 available reports, 22(55%) of the teachers were able to identify the problem correctly whereas12 (30%) could identify to some extent and 6(15 %) failed to identify any problem with the child.

Among 93 children, 36(38.7%) received the diagnosis of ADHD, 17(18.3%) had Conduct disorder, 15(16.1%) had Mental Retardation, 12 (12.9%) SLD, 10(10.8%) hadDepressive disorder, 7(7.5%) Conversion disorder, 5(5.4%) OCD, 2(2.2%) had Anxiety Disorder, 2(2.2%) Adjustment disorder, 1(1.1%) was diagnosed with Psychosis, ASD, ODD, sleep disorder andAcute stress reaction each.Many children with ADHD had comorbid Conduct disorder and SLD.



Regarding the source of referral, 63(67.8%) were brought by the parents,15(16.1%) were referred by the paediatrician, 4(4.3%) by the Neurologist, 8 (8.6%)by the Teachers and 3(3.2%) by psychologists/counsellors.



Discussion

The study gains importance in the background that the forerunner amongst the global burden of diseases are the illnesses that first makes its appearance in childhood. The median prevalence rate of childhood occurring psychiatric disorders is 12%[5].This study revealed that more boys seek the helpof CAC than girls. This is in concordance with earlier studies that psychiatric disorders weremoreprevalent amongst boys[6]. ADHD and Conduct disorder were the 2 commonest psychiatric disorders in the sample studied. Earlier studies also revealed that conduct disorder to be common among school children with a high comorbidity of ADHD[7, 8]. Almost 3/4th of the patients attending the CAC in the study were brought by their parents. 50% of the teachers were able to identify the problem in their students but they failed to be the main source of referral. This reinforces the findings of earlier studies that parents can be considered as the main gatekeepers for access to specialist care in children[9].

Conclusion

Early identification of psychiatric morbidities in children is needed as the help seeking behaviour is only in late childhood which may be late for adequate remedial measures. Teachers should be imparted training for early identification of behavioural problems in their students and accessing specialist care.

References

1. Kuczaj, Stan A. (1991). Developmental psychology: childhood and adolescence. New York: Macmillan Pub. Co. ISBN 0-02-377010-4.
2. Kanner, Leo (July 1960), "Child Psychiatry: Retrospect and Prospect", American Journal of Psychiatry **117** (1): 15–22
3. Rutter, Michael and Taylor, Eric. Chapter 2, Clinical assessment and diagnostic formulation. In Rutter and Taylor (2002) .
4. Green, Jonathan; Yule, William (2001),"The Significance of Genetic Variation for Abnormal Behavioural Development", in Jonathan Green and William Yule,Research and Innovation on the Road to Modern Child Psychiatry: Festschrift for Professor Sir Michael Rutter, London, p. 20
5. Costello JE, Egger H, AngoldA J American Acad Child and Adol Psych vol 44, 10 2009 972-86
6. E Fombonne Chartres study 1– prevalence of psychiatric disorders among French school age children,The British Journal of Psychiatry Jan 1994, 164 (1) 69-79.
7. SujitSarkhel, Vinod Kumar Sinha, Manu Arora, PushpalDeSarkar,Indian Journal of Psychiatry, Year 2006, Volume 48, Issue 3 [p. 159-164]
8. JyothsnaAkamVenkata, Anuja S Panicker,Indian Journal of Psychiatry, Year 2013, Volume 55, Issue 4 [p. 338-342]
9. Kapil sayal, Eric taylor, Jennifer beecham, Patrick byrne,The British Journal of Psychiatry Jul 2002, 181 (1) 43-48.

✪ ORIGINAL ARTICLE

Anti-inflammatory activity of aqueous extract of aloe vera leaves

Sri Harsha H
Dennis Varghese Thomas
Vikram Gowda N R
Jiyo Chacko

From:
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla, India - 689 101

Sri Harsha H
Assistant Professor

Department of Pharmacology
Navodaya Mical College
Raichur

Dennis Varghese Thomas
Assistant Professor
Department of General Medicine
PIMS & RC

Vikram Gowda N R
Assistant Professor
Department of Physiology
PIMS & RC

Jiyo Chacko
Assistant Professor
Department of Pharmacology
PIMS & RC

Correspondence to:
Dr Vikram Gowda
E-mail: vikramgowda2@gmail.com

Abstract

Objectives: To study the effect of aqueous extract of Aloe vera leaves and its combination with conventional oral anti-inflammatory drugs on oedema induced by sub plantar injection of carrageenan, in the hind paw of albino rats. **Material & Methods:** The authenticated leaves of Aloe vera were grinded to form fresh aqueous extract and tested for anti-inflammatory activity in albino rats, where in inflammation was induced by sub plantar injection of carrageenan (1%) in normal saline. Aloe vera extract (300 mg/kg and 600mg/kg p.o.) anti-inflammatory drug Indomethacin (25mg/kg p.o.) were administered alone or in combination. The hind paw volume was determined using Plethysmometer and recorded in all group of animals. **Results:** Aloe vera leaves extract significantly reduced the paw edema by 29% to 32% in two different doses and in combination with conventional anti-inflammatory drug it reduced the edema by 35%. **Conclusion:** The results indicate that freshly prepared aqueous extract of AV leaves possesses significant anti-inflammatory activity and also enhances the anti-inflammatory activity of conventional oral anti-inflammatory drugs.

Key Words

Aloe vera leaves, Carrageenan induced edema, Indomethacin, Plethysmometer

Introduction

Aloe vera (L.) Burm. fil. (synonym A. Barbadensis Miller) is a cactus-like plant with green, dagger-shaped leaves that are fleshy, tapering, spiny, marginated and filled with a clear viscous gel. The name was derived from the Arabic 'alloeh' meaning 'bitter', because of the bitter liquid found in the leaves. It is also known as 'lily of the desert', the 'plant of immortality', and the 'medicine plant' with qualities to serve as alternate medicine.

There are more than 360 types of aloe vera species identified and more often gel form is preferred as medicinal preparation[1]. *Aloe barbadensis is* one of the common Aloe vera type used for commercial and also therapeutic purposes in Asia and all over the world[2]. The common active ingredient identified in aloe vera leaves extract is anthraquinone, polysaccharide and carbohydrates[3].

Aloe vera plays an important role in many medical properties as a traditional medicine in treatment of many physiological disease and conditions such as Insomnia, Burns, Ulcerative colitis, Psoriasis, wound healing[4]. Antioxidant properties of Aloe vera are shown in various studies and it has been reported that it stimulates the antiviral activity of macrophages[5].

Inflammation is a protective phenomenon observed on application of noxious stimuli (physical, chemical or thermal) to tissue. It involves release of various mediators like prostaglandins, leukotrines, platelet activating factor, bradykinin and others[6]. The present management of inflammation with anti-inflammatory drugs for long duration is always associated with adverse effects, hence alternative medications mainly plant origin are emerging as an alternative.

Aloe has been a traditional folk medicine for more than 3000 years, three types of commercial preparations are available dried exudates – excreted from the aloin cells in the zone of vascular bundle, gel

form – present in the centre of leaf and oil – extracted from organic solvents[7]. Studies have shown that gel has anti-inflammatory activity but very limited numbers of studies have demonstrated fresh aloe vera aqueous leaves extract as an anti-inflammatory action hence this study was conducted in view to observe the anti-inflammatory action of aqueous aloe vera leaves extract.

Materials & Methods

It is a prospective, randomized analytical, interventional, placebo-controlled single blind study

Animals – Albino rats of either sex weighing around 150-300g were selected for the study. They were housed under standard conditions of temperature, humidity and dark-light cycle (12h – 12h). They had free access of water. They were under supervision in the Animal house; the experiments were carried after obtaining approval from institutional animal ethical committee.

Preparation of extract - The whole leaves of Aloe vera was collected after identification from a plant of at least 4-5years. The leaves were then washed, and manually grinded in mortar. The extract was mixed subsequently with water for proper homogenization and appropriate amount (i.e. 300mg/kg and 600mg/kg) were immediately delivered to rats which needed aloe vera, fresh prepared extract was used on each day of experiment.

Carrageenan-induced edema in rat hind paw - Carrageenan is a mixture of polysaccharides composed of sulfated galactose units and is obtained from Irish Sea moss. It was dissolved in normal saline and used in strength of 1%. This method is based on the plethysmometeric measurement of edema produced by sub plantar injection of Carrageenan, in the hind paw of rat.

Procedure - The drugs were administered orally with the help of a sterile, nontoxic tube made up of polyvinyl chloride which was introduced into the stomach of rat.

36 rats were selected and divided into 6 groups of 6 each.
Group I: Control rats- receive vehicle (distilled water) only
Group II: Standard rats – receive Indomethacin 25 mg/kg
Group III: Test rats – receive Aloe vera *300* mg/kg
Group IV: Test rats – receive Aloe vera *6*00 mg/kg
Group V: Test rats – receives Indomethacin 25mg/kg + Aloe vera 300 mg/kg
Group VI: Test rats – receives Indomethacin 25mg/kg + Aloe vera 600 mg/kg

The IITC 520 Plethysmometer was used to measure the paw volume. It consists of a water cell which is fitted on to a plexi glass stand. This water cell has an inlet in which the paw is dipped and an outlet with a stopcock. The water cell is connected to an electronic display which shows the volume of displacement of water in millilitre (ml) from the water cell when paw is dipped into water cell which is taken as paw volume. The paw is inserted into water, contained in a special water cell of which the resistance is changed due to the immersion of the animal's paw. This resistance change is calibrated in ml and displayed on the read out in ml with a resolution of 0.1 ml.

A- IITC 520 Plethysmometer

B- Technique of immersion of hind paw for recording volume

Acute inflammation was produced by sub plantar injection of 0.1 ml of freshly prepared 1% suspension of Carrageenan in normal saline in the right hind paw of the rats and paw volume was measured plethysmometrically at 1h, 2h, 3h and 4h after Carrageenan injection. A line was marked at the level of the malleolus to facilitate the dipping of the foot up to the same mark every time. The hind foot of the animal was dipped up to the line marked on the foot into the water cell and the reading was recorded

The hind paw volume obtained at 1 h, 2 h, 3 h and 4 h after Carrageenan injection both in control and test animals. By comparing the edema produced in control rats and in those treated with drugs the percentage inhibition of edema was calculated as follows.

$$\text{Percentage inhibition} = \frac{Vc - Vt}{Vc} \times 100$$

Vc = Volume of paw edema in control rats; Vt = Volume of paw edema in test rats

Statistical analysis - The mean of the individual readings was taken to represent the group. Mean increase in paw volume and percent inhibition were calculated and Analysis of variance (ANOVA) to find the significant difference in the group ($P<0.05$) was considered statistically significant, the percentage inhibition of edema in drug treated rats (standard and test drugs) was calculated and tabulated in percentage

Results & Analysis

The experimental protocol described in methodology was carried out for three times spacing two weeks apart and the mean value of the recording was considered for the experiment, all the values were expressed as mean and standard deviation of volume as displayed in Table No.1 below

Table no.1 Mean values of hind paw volume

Hind paw volume (ml) expressed in Mean ± SD				
	1hr	2hr	3hr	4hr
Group I Control rats	1.2±0.14	1.27±0.19	1.33±0.08	1.39±0.06
Group II Indomethacin 25mg/kg	1.2±0.07	1.15±0.09*	1.05±0.12**	1.01±0.11**
Group III Aloe vera 300 mg/kg	1.18±0.16	1.11±0.10*	1.04±0.10**	0.98±0.07**
Group IV Aloe vera 600 mg/kg	1.14±0.09	1.07±0.05*	1.00±0.10**	0.95±0.09**
Group V Indomethacin 25mg/kg + Aloe vera 300 mg/kg	1.19±0.12	1.13±0.09*	1.08±0.08**	1.00±0.08**
Group V Indomethacin 25mg/kg + Aloe vera 600 mg/kg	1.16±0.09	1.1±0.10*	0.99±0.12**	0.92±0.09**
	ONE WAY ANOVA			
F value	0.257	3.76	10.57	25.1
P value	0.933	0.009	0.0001	0.0001

(N = 6) df = (5, 30)
Statistical analysis by One-way ANOVA followed by Dunnett's multiple comparisons.
P value * < 0.05 is significant; **< 0.001 is highly significant.

In control group there was a progressive increase in mean paw volume on comparison to test group rats where progressive decrease in mean paw volume was observed from first to fourth hour of recording after the induction of edema by carrageenan injection.

At the end of the first hour on multiple comparisons by one way analysis of variance between the groups we observed difference between the control and test group mean volume but F value (0.257) suggesting p value (> 0.05) and statistically not significant

In contrast at the end of second, third and fourth hour we found the decrease in hind volume was statistically significant where F value (3.76, 10.57 and 25.1) which had very high statistical significance (P < 0.001) suggesting that both standard anti-inflammatory drug (Indomethacin 25mg/kg) and the plant origin Aloe vera leaves extract 300 mg/kg and 600mg/kg also had significant action of anti-inflammatory by reducing the paw edema, as shown in table the combination of standard drug indomethacin with aloe vera leaves also had similar action of reducing the paw edema in the hind legs

Post – hoc Analysis of the mean values with Dunnett's Test was applied to analyze the difference in paw volume levels in each group with control group

At the end of first hour on comparison of paw volume in the all the groups we found no difference but in second hour recording we noted the reduction of paw edema was statistically significant in all test group rats (p <0.05)

At the end of third and fourth hour the reduction in test group animals was highly statistically significant (p<0.001) stating that indomethacin (25mg/kg) and aloe vera (300mg/kg) and (600mg) and in combination with each other also had anti-inflammatory activity.

Percentage inhibition of edema was calculated as mentioned earlier and observed the percentage of decrease in edema on compared to control group as expressed in Table No.2

Table no.2 Percentage inhibition of paw edema

Inhibition % of paw edema				
	1hr	2hr	3hr	4hr
Group II Indomethacin 25mg/kg	0	10.26	21.19	27.59
Group III Aloe vera 300 mg/kg	8.45	13.44	22.26	29.44
Group IV Aloe vera 600 mg/kg	8.65	16.12	25.62	32.61
Group V Indomethacin 25mg/kg + Aloe vera 300 mg/kg	5.24	11.19	19.58	28.50
Group V Indomethacin 25mg/kg + Aloe vera 600 mg/kg	3.36	13.35	26.33	34.85

The percentage of inhibition was not very high as observed in the first hour of induction of edema but progressively we observed that percentage of inhibition increased and reached around 30 to 35% in all the test group suggesting the anti-inflammatory action of indomethacin 25mg/kg and aloe vera leaves extract in dosage of 300mg/kg and 600mg/kg.

Discussion

This study showed that the aqueous extract of the *Aloe vera* contain compounds with a potential to reduce carrageenan induced-edema. We found that at the doses used, *Aloe vera* inhibited edema formation, by a percentage close to the inhibition produced by the well established anti-inflammatory agent indomethacin like most of the non-steroidal anti-inflammatory compounds, inhibits the biosynthesis of prostaglandins and this effect might explain its anti-inflammatory activity[8].

The inhibition of carrageenan induced inflammation in rats is an established model to screen compounds for potential anti-inflammatory activity. The inflammatory phenomenon is biphasic where the first hour of carrageenan induction leads to the release of histamine and serotonin in to cytoplasm by mast cells. The second phase starts after one hour of induction and is mediated by release of prostaglandins and the bradykinins serve the mediators between first and second phase of inflammatory process [9].

Our results suggests that the aqueous extract of aloe vera leaves inhibited in-vitro conversion of arachidonic acid to PGE2 suggesting the idea that the extract has cyclooxygenase inhibitory properties. As suggested in presence of exogenous arachidonic acid leads to bypassing the effect of lipooxygenase enzymatic activity and in presence of microsomes which have high cyclooxygenase activity and negligible lipooxygenase activity lads to an hypothesis that the anti-inflammatory effect of leaves of *Aloe vera* is related to cyclooxygenase inhibition, rather than an effect on lipooxygenase activity.

A number of Japanese workers have found antiinflammatory compounds in *Aloe* species other than *Aloe vera.* Fujita et al.[10] described in vitro bradykininase and carboxypeptidase activities in *Aloe arborescens* while Yagi et al.[11] reported in vitro antibradykinin activity in *Aloe saponaria;* the faster breakdown of bradykinin might reduce pain and inflammation.

In summary, we have demonstrated that the aqueous leaves extracts of *Aloe vera* gel has inhibitory effect on carrageenan-induced edema mainly by inhibiting the production of PGE2 and due to inhibitory action on the arachidonic acid pathway via cyclooxygenase. The results of this study conclude that the freshly prepared *Aloe vera leaves extract* has potential antiinflammatory activity, and thus provides a scientific basis for the utilization of this plant in folk medicine for the treatment of inflammatory processes.

References

1. Paulsen E, Korsholm L, Brandrup F. A double-blind, placebo-controlled study of a commercial Aloe vera gel in the treatment of slight to moderate psoriasis vulgaris. JEADV. 2005; 19: 326–331.
2. Hu Y, Xu J, Hu Q. Evaluation of antioxidant potential of aloe vera (aloe barbadensis miller) extracts. J. Agric. Food Chem. 2003; 51: 7788 –7791.
3. Vinson JA, Kharrat HA, Andreoli L. Effect of Aloe vera preparations on the human bioavailability of vitamins C and E. Phytomedicine. 2005; 12: 760–765.
4. Atherton P."*Aloe vera* revisited: Review of Aloe gel". Brit J Phytother. 1984; (4):176-183.
5. Zhang L, Tizard IR. Activation of a mouse macrophage cell line by acemannan: the major carbohydrate fraction from Aloe vera gel. Immunopharmacology. 1996; 35: 119–128.
6. Grglewski RJ. Molecular mechanisms of inflammation. Eu J Rheumatol inflamm. 1981:4:153-9
7. Saccu D, Bagoni P,Procida G. Aloe Exudate: Characterization by reverse phase HPLC and head space GC-MS. J Agric Food Chem. 2001:49;4526-30
8. Higgs, A.G., Flower, J.R. and Vane, R.J. A new approach to anti-inflammatory drugs. *Biochemical Pharmacolagy. 1979;* 28, 1959 1961
9. Vinegar R, Traux JF, Selph JH, Johnston PR, Venable AL, Mekenzie KK. Pathway to carrageenan induced inflammation in the hind limb of the rat. Fed Proc. 1987:46;118-26
10. Fujita, K., lto, S., Teradaira, R. and Beppec, H. Properties of a carboxypeptidase from aloe. *Biochemical Pharmacology. 1979;* 28, 1261 1262
11. Yagi, A., Harada, N., Yamada, H., Iwadare. S. and Nishioka, I. Antibradykinin active material in *Aloe~aponaria. Journal Pharmaceutical Sciences. 1982;* 71, 1172-1174

✪ ORIGINAL ARTICLE

Rotaviral diarrhoeal diseases in hospitalised children under five years with special reference to the strain of virus

Carol Sara Cherian
Joseph J

From:
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla, India - 689 101

Carol Sara Cherian
Associate Professor

Joseph J
Senior Resident

Department of Paedictrics
PIMS & RC

Correspondence to:
Dr Carol Sara Cherian
E-mail: benjicarol@gmail.com

Abstract

Diarrhoeal diseases are a leading cause of childhood morbidity and mortality in developing countries, and an important cause of malnutrition. **Aims :** This study aimed to assess the proportion of diarrhoea cases/ deaths, if any, attributable to Rotavirus among children less than five years of age admitted in a tertiary level hospital in Central Travancore and to find the type of rotavirus causing the disease in the region **Methods:** .The study was done for one year in the pediatric department of a tertiary care teaching hospital of south central Kerala All children between 2 month and 5 years of age, presenting with diarrhoea with parental consent for Rotaviral testing were included. The Enzyme Linked Immune Sorbent Assay (ELISA) was done for rotaviral antigen detection in samples negative for parasites and bacteria. All such positive specimens, were subjected to RT-PCR, for VP4 and VP7 for strain identification. Stool samples were processed for rotavirus VP6 antigen using Rotaclone ELISA kit. Viral nucleic acid was extracted from the stool samples and the RT-PCR serotyping was carried out with the help of primers specific to genes encoding VP7 (G-type) and VP4 (P-Type). Severity of Diarrhoea was assessed using the Vesikari scoring system. **Results:** The prevalence rate of rotaviral disease in children presenting with diarrhoea was 37%. Of the children with diarrhoeal diseases ,Vomiting(p=0.009), degree of dehydration(p=0.019), were observed more in the rotaviral antigen positive group. Duration of rehydration therapy(ORS p=0.047 and need for intravenous fluid therapy p=0.022) and need for ICU care(p=0.005) was found to be higher in the Rotavirus positive group comparing to negative group. PCR sequencing has revealed all samples were Type A strain with 96% of samples G3P8 serotype. **Conclusions:** The prevalence of rotaviral diarrhoeal disease is 37%. Vomiting and degree of dehydration if associated with diarrhoea were found to be significant predictors of Rotaviral disease PCR sequencing has revealed all samples were Type A strain with 96% of samples G3P8 serotype.

Key Words

Rotavirus, Diarrhoea, Children, hospitalised ,strain

Introduction

Diarrhoeal diseases are a leading cause of childhood morbidity and mortality in developing countries, and an important cause of malnutrition[1]. It accounts for 16% of deaths in children under 5 years of age(WHO 2008) almost 800000 fatalities worldwide[2]

On average, children below 3 years of age in developing countries experience three episodes of diarrhoea each year. In many countries diarrhoea, including cholera, is also an important cause of morbidity among older children and adults. Diarrhoea as a result of bacterial infections like shigella are reducing in incidence while virus causes esp Rotavirus most easily identifiable cause is increasing[3]. In India 113000 younger than 5 years died from Rotaviral infection in 2005, mortality rate of 4.14 deaths per 1000 live births [4]. Diarrhea in children can result in long term problems such as malnutrition and anaemia, repeated infections or co infections and long term negative psychomotor effect.

This is study from Kerala which aims to identify the circulating strains of

Rotavirus in a hospital environment, aiming to identify differences in the genotype in respect to population and location

Aims

1. To assess the proportion of diarrhoea cases/ deaths, if any, attributable to Rotavirus among children less than five years of age admitted in hospital

2.The identify the circulating strains of Rotavirus using RT-PCR[5]

Methodology

This study was conducted in the Paediatric Department of Tertiary care teaching hospital of south central Kerala (Pushpagiri Institute of Medical Sciences and Research Centre Tiruvalla)

The study period was for one year from January 2014 to December 2014

All children between 2 month and 5 years of age, presenting with diarrhoea with parental consent for Rotaviral testing were included.

Children below 2 month and above 5 years of age, those with duration of illness more than 14 days, antibiotic induced diarrhea, diarrhoea developing after admission in hospital for unrelated illness were excluded from the study.

Stool samples were collected from each child enrolled in the study on the day of presentation. All stool samples were collected and transported to the study laboratory (Diagnostic Virology, Pushpagiri Institute of Medical Sciences and Research Centre, Tiruvalla).All samples were tested microscopically to rule out the presence of parasites, bacterial pathogens causing diarrhoea. Enzyme Linked Immune Sorbent Assay (ELISA) was done for rotaviral antigen detection in samples negative for parasites and bacteria. All such positive specimens, for Rotaviral antigen were subjected to RT-PCR, for VP4 and VP7 for strain identification[6]

Stool samples were processed for rotavirus VP6 antigen using Rotaclone ELISA kit. Viral nucleic acid was extracted from the stool samples and the RT-PCR serotyping was carried out with the help of primers specific to genes encoding VP7 (G-type) and VP4 (P-Type). Severity of Diarrhoea was assessed using the Vesikari scoring system[7]

An analysis of the age, sex distribution, seasonal variation, severity of symptoms, treatment required including time for recovery and complications if any were assessed and entered in proforma

Statistics

The mean and median were calculated for age, duration of hospitalisation, treatment time and complications in the two groups and was evaluated using a Student T test to assess statistical significance.Sex distribution and Seasonal variation was plotted as a frequency table and assessed using a Chi^2 test.An episode was considered *mild* for Vesikari Score 0-5, *moderate* for score 6-10, *severe* for score 11-15, *very severe* for score 16-20 [7]

Results

Total of 47 children with consent participated in the study, out of which 17 were positive for Rotavirus Ag with a prevalence rate of 37%. The median age of admission was 13.5 months and the disease was more in boys.

Maximum number of rotaviral antigen positive cases was noted during the months of February to May. Children in the age group of 7-12 months were most affected with rotaviral infection

Of the children with diarrhoeal diseases ,Vomiting (p=0.009), degree of dehydration(p=0.019), were observed more in the rotaviral antigen positive group which was statistically significant

Duration of rehydration therapy(ORS p=0.047 and need for intravenous fluid therapy p=0.022) and need for ICU care(p=0.005) was found to be higher in the Rotavirus positive group comparing to negative group which was statistically significant (table 1.1)

There was no deaths in the study population. PCR sequencing has revealed all samples were Type A strain with 96% of samples G3P8 serotype.

Table 1.1 Clinical features of the children

	Rotavirus Positive	Rotavirus Negative
Age		
Mean(months)	25	22.2
Median(months)	13.5	16.5
Sex		
Male	11	22
Female	6	7
Season	Feb-May (14 Cases)	Nov - Jan 11 April-June 12
Median Duration (Days)		
Hospitalization	4.82	4.5
Vomiting	3.94	2.37
Dierrhia	5.11	4.4
Symptoms (Days)		
Vomiting	3.94	2.37
Fever	2.52	2.55
Treatment received (Days)		
IV Fluids	1.4	0.72
Rehydration Therapy	3.35	2.58
Intensive care unit	1.17	0.24
Complications		
Hypoglycemia(<50mg%)	Nill	Nill
Hypokalemia(2.5mEq/L)	Nill	Nill
Hyponatremia(<135mEq/L)	4/17	4/29

Outcome at discharge		
Recovered	14	29
Ongoing Gastroenteritis	3	-
Transfered	-	-
Died	-	-
Dehydration		
No	5	22
Some	9	6
Severe	3	1

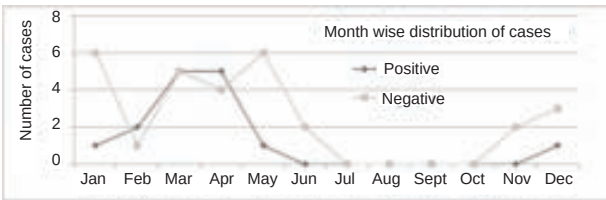


Figure1.1Month wise distribution of cases

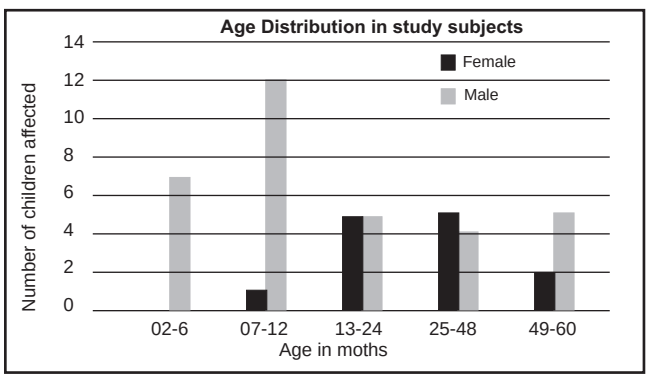


Figure 1.2 Age Distribution

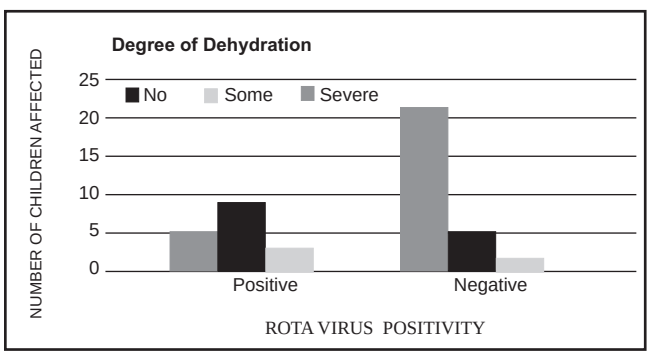


Figure 1.3 Degree of dehydration

Discussion

This study aimed to find the burden of Rotaviral diarrhoea in children under 5 years admitted to our hospital and to identify the most prevalent circulating serotypes of Rotavirus causing diarrhoea.

A prevalence of 37 % was noted in the study. This is slightly higher compared to other studies published from within India[11,12,13,14,15]. Clustering of cases was seen during the months of February to May as shown in similar studies [8,9,15]

Vomiting and degree of dehydration if associated with diarrhoea were found to be significant predictors of Rotaviral disease as found in studies by Rodriguez WJ and Kovacs A [20,21]. All the children because of the severity of the disease required increased time for rehydration therapy including increased ICU care. There were no significant electrolyte abnormalities or complications noted. There were no deaths observed in the study group. This could be due to early recognition and prompt management of the condition with close observation.

Rotavirus is a member of the Reoviridae family and contains a double stranded RNA (dsRNA) enclosed in a triple layered capsid. The outer layer of rotaviruses is composed of two proteins, VP7 and VP4, encoded by RNA segments 9 and 4, respectively. Those proteins elicit neutralizing antibody responses and form the basis of the current classification of group A rotaviruses into G (VP7) and P (VP4) types, where G stands for glycoprotein and P for protease sensitive protein. Currently, 27 G genotypes and 35 P genotypes have been reported in humans and animals [10.]

PCR sequencing in our study revealed 96%(17) cases to be Type A strain G3P8. None of the other published studies from India[13,14,15,16] recognise G3P8 as a cause for diarrhoea differing from international studies[17,18,19] which identifies it as one of the leading causes.

This part of Kerala has a significant expatriate population which could in part explain the variation observed in sequencing as compared to other centres.

At present we have two vaccines for Rotaviral diarrhoea-the monovalent vaccine and the pentavalent vaccine. While both offer similar levels of protection G3P8 as a serotype is covered exclusively by the pentavalent vaccine. While our study has a very small sample size, it recognises that even within the same state there[15] may be a wide variety of circulating strains of Rotavirus depending on the geographical location, and effective prevention can be made only if thorough knowledge of the strain types is made. Further studies are required in this regard.

What this study adds

Rotavirus is a significant cause of diarrhoeal disease in children

G3P8 serotype is a significant strain causing diarrhoea

Acknowledgement

Dr Sara Chandy – Head of Virology PIMS and RC

Bibliography

1. World Health Organization.The Treatment of diarrhoea : a manual for physicians and other senior health workers. -- 4th rev.
2. Liu L, Johnson HL, et al.: Global, regional, and national causes of child mortality: an updated systematic analysis for 2010 with time trends since 2000.Lancet 2012, 379(9832):2151-2161

3. Parashar UD, Bresee JS, et al. Rotavirus. Emerg Infect Dis. 1998;4:561–70. DOI

4. Shaun K Morris, ShallyAwasthi, Ajay Khera Bull World Health Organ 2012;90:720–727 | doi:10.2471/BLT.12.101873

5. World Health Organization: Manual of rotavirus detection and characterization methods, www.who.int/vaccines-documents/; October 2009

6. Leen-Jan van Dorn, Bernhard Kleter, Evert Hoefnagel, Wim Quint et al: Detection and Genotyping of human rotavirus VP4 and VP7 genes by Reverse transcriptase PCR and Reverse hybridisation. Journal of Clinical Microbiology, Sept. 2009; p. 2704–2712.

7. Ruuska T, Vesikari T. A Prospective study of acute diarrhea in Finnish children from birth to 2 ½ years of age. Act PaediatrScand 1991;80:500-07.

8. Ansari SA, SpringthorpeVS, Sattar SA, et al.Survival and vehicular spread of human rotaviruses:possible relation of seasonality of outbreaks. Rev Infect Dis. 1991;13:448-461

9. Levy K, Hubbard AE, Eisenberg JNS. Seasonality of rotavirus disease in the tropics:a systemic review and meta-analysis.Int J Epidemiol. 2008;Epub ahead of print

10. Matthijnssens J, Ciarlet M, et al: Uniformity of rotavirus strain nomenclature proposed by the Rotavirus Classification Working Group (RCWG); 2011. Arch Virol 2011

11. Indrani Banerjee1, Sasirekha Ramani1 et al:Comparative Study of the Epidemiology of Rotavirus in Children from a Community-Based Birth Cohort and a Hospital in South India;J. Clin. Microbiol. July 2006 vol. 44 no. 7 2468-2474

12.Ramani, Sasirekha ; Kang, Gagandeep (2007) Burden of disease & molecular epidemiology of group A rotavirus infections in India Indian Journal of Medical Research, 125 (5). pp. 619-632.

13.Rajiv Bahl, Pratima RayIncidence of Severe Rotavirus Diarrhea in New Delhi, India, and G and P Types of the Infecting Rotavirus Strains J Infect Dis. (2005) 192(Supplement 1): S114-S119.

14. Gagandeep Kang, RashmiArora et al, Multicenter, Hospital-Based Surveillance of Rotavirus Disease and Strains among Indian Children Aged <5 Years: J Infect Dis. (2009) 200 (Supplement1): S147-S153

15.Mathew MA et al; Prevalence of Rotavirus Diarrhea among Hospitalized Under-five ChildrenIndian PediatricsVol51-Jan15,2014

16.Gagandeep Kang, Rishi Desai, et al Diversity of circulating rotavirus strains in children hospitalizedwith diarrhoea in India, 2005–2009:VaccineVolume 31, Issue 27, 12 June 2013, Pages 2879–2883

17. Tort, L. F. L., et al Molecular epidemiology of group a rotavirus among children admitted to hospital in Salto, Uruguay, 2011-2012: First detection of the emerging genotype G12. J. Med. Virol., 87: 754–763.

18. Kirkwood CD, Boniface K, et al; Distribution of rotavirus genotypes after introduction of rotavirus vaccines, Rotarix® and RotaTeq®, into the National Immunization Program of Australia.Pediatr Infect Dis J. 2011 Jan;30(1 Suppl):S48-53.

19.Hull JJ, Teel EN, et al; National Rotavirus Strain Surveillance System United States rotavirus strain surveillance from 2005 to 2008: genotype prevalence before and after vaccine introduction. Pediatr Infect Dis J. 2011 Jan;30(1 Suppl):S42-7.

20.Rodriguez WJ, Kim HW, Arrobio JO, et al.Clinical features of acute gastroenteritis associated with Human Reovirus –like agent in infants and young children. J Pediatr.1977:91:188-193

21.Kovacs A, Chan L, Hotrakitya C, et al.Rotavirus gastroenteritis: clinical and laboratory features and use of Rotazymetest.Am J Dis Child.1987;141:161-166

✪ CASE REPORT

Diagnostic tests; is it a real alternative to clinical judgement ?

Biju Sebastian

Anuna Laila Mathew

Abstract

There is a great degree of reliance on investigatory procedures rather than clinical evaluation to arrive at a diagnosis among the present day medical practioners .This can cause disastrous consequences in cases where investigatory reports are erroneous. To illustrate this, a case report regarding multiple impacted teeth which was diagnosed as hypothyroidism is presented in this article.

Keywords: Parathyroid hormone, calcium, phosphorous, impacted teeth, vitamin D

From:
Pushpagiri College of Dental Sciences
Tiruvalla, India - 689 101

Biju Sebastian
Professor & HoD

Department Of Orthodontics

Anuna Laila Mathew
Reader

Department Of Anaesthesia

Pushpagiri College of Dental Sciences, Thiruvalla

Correspondence to:
Dr Biju Sebastian
E-mail: drbijuseb00@gmail.com

Introduction

Modern day medical field has become so advanced that clinicians have started depending on investigations as an alternative to thorough clinical examination in arriving at a diagnosis and treatment protocol. This starts right from the undergraduate level itself leading to disastrous consequences. An experienced practitioner of yester years who didn't have the help of sophisticated and highly advanced equipments for diagnostic tests were able to diagnose cases to a high degree of accuracy. This ability is lost nowadays. To highlight this aspect we would like to present a case report of a patient who came to our department.

Case Report

A 24 year old female patient (Fig: 1, 2 & 3) reported to Department of Orthodontics, Pushpagiri College of Dental Sciences for correction of her mal-aligned teeth. On clinical examination we found multiple unerupted teeth (13, 14,18,28,33,38,47 and 48) which is a rare occurrence in a patient with normal medical condition (Fig: 4, 5 & 6). An OPG was taken, which showed multiple impacted teeth (Fig 7).

Fig. 1

Fig. 2

Fig. 3

Fig. 4

Fig. 5

Fig. 6

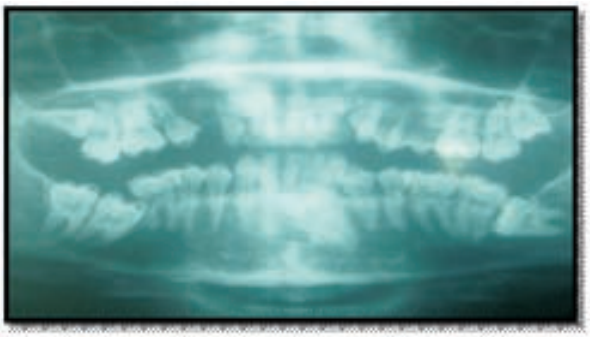
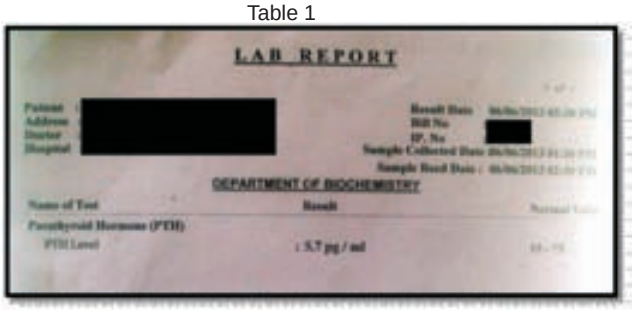


Fig. 7

Suspecting an endocrinal abnormality we referred the patient to an endocrinologist. Endocrinal evaluation of the case showed a decrease in PTH level indicating hypoparathyroidism (Table: 1).

Table 1



LAB REPORT

[redacted]

DEPARTMENT OF BIOCHEMISTRY

Name of Test | Result

Parathyroid Hormone (PTH)

PTH Level : 5.7 pg / ml

The patient was then advised to undergo treatment for the same. The patient went for a second opinion regarding her condition and the diagnostic tests were repeated by the second endocrinologist at another diagnostic centre. The results showed PTH level was within normal range and a decreased Ca serum level (Table: 2 & 3).

Table 1

[redacted]

DEPARTMENT OF HORMONES

DHEA SO4

LH

PARA THYROID HORMONE (PTH)

TESTOSTERONE

17-HYDROXY PROGESTERONE

Table 3

[redacted]

DEPARTMENT OF CLINICAL BIOCHEMISTRY

CALCIUM SERUM

PHOSPHOROUS, SERUM

As per the advice of the second endocrinologist, she underwent Vitamin D supplementation and her Ca level became normal (Table: 4) following which she was declared fit to undergo orthodontic correction of her malaligned teeth.

Table 4

LAB REPORT

OP NO : [redacted] NAME [redacted]

BILLNO: [redacted] AGE/SEX: 24 /FEMALE REPORT NO : [redacted]

TEST | RESULT | NORMAL RANGE

BIOCHEMISTRY

LIVER FUNCTION TEST

Serum Albumin 4.5 gm/dl 3.97 - 4.94 gm/dl

Serum Calcium 9.2 mg/dl 8 - 10 mg/dl

Discussion

There are many reasons like local factors, injuries, genetic disorders, altered hormone levels behind multiple impacted teeth[1,2]. Hypoparathyroidism is a disorder of parathyroid gland with PTH deficiency. Reduced PTH level can result in intra-oral features like multiple impacted teeth, hypoplastic teeth with increased risk of caries, shortened roots and sometimes mucocutaneous candidiasis[3].

Hypoparathyroidism can be either due to (1) autoimmune destruction of parathyroid gland 2) due to extensive neck surgery for thyroid cancer[4]. Other than these two causes it can also occur as a congenital disorder in which the parathyroids and other derivatives of the third and fourth pharyngeal pouches do not develop (DiGeorge syndrome) [4,5,6,7]. The other general clinical features of hypoparathyroidism include circumoral numbness, parasthesias, hypertension, weakness, acute pancreatitis, carpel and pedal muscle spasms, greater trabecular bone volume, tetany and/ or seizures. These symptoms are also associated with classical biochemical constellation of hypocalcemia and low levels of PTH[3,4,5,6,8]

But in some cases, patients with hypoparathyroidism can be asymptomatic. In such cases, the combination of low serum calcium concentration and low or undetectable PTH levels and elevated levels of phosphorous essentially rules out all other causes of hypocalcemia[4,7,8]. In case of any doubt, it is always better to go for a retest. The retest done in this particular case helped in avoiding disastrous consequences.

Conclusion

In this case, there were no other clinical features other than multiple impacted teeth from which a diagnosis of hypoparathyroidism could be inferred. Still the decision to start the treatment for hypoparathyroidism was taken on the basis of the first laboratory report. Fortunately the patient decided to go in for a second opinion and retest done ruled out hypoparathyroidism. This is not a one of incidence now a day. So as far as possible we should not rely only on the investigations in our treatment planning and a good clinical assessment is mandatory to avoid unwarranted treatment for the patients. Critical assessment is a continuing process which would help us to improve our clinical standard and we hope that this report would be taken in a positive light.

References

1. Peedikayil FC. Delayed tooth eruption. e-Journal of Dentistry. 2011; Oct – Dec. Vol 1: Issue 4.
2. Suri L, Gagari E, Vastardis . Delayed tooth eruption: Pathogenesis, diagnosis, and treatment. A literature review .Am J Orthod Dentofacial Orthop 2004;126:432-445.
3. Crispian Scully. Medical Problems in dentistry. 5th Edition .Elsevier; 2013: 99-100.
4. Rubin MR, Bilezikian JP. Hypoparathyroidism: clinical features, skeletal microstructure and parathyroid hormone replacement. Arq Bras Endocrinol Metab. 2010; 54(2):220-226.
5. John T Potts, Jr. Harrison's principles of internal medicine. 17th Edition. McGraw- Hill; 2008: 2391-2396.
6. M.W.J.Strachan, J.Newll-Prince. Davidson's principle and practice of medicine. 22nd Edition .Churchill Livingstone ;2014: 770-771.
7. Greenberg, Glick, and Ship. Burket's Text Book of Oral Medicine. 11th Edition .Elsevier; 2008: 533-534.
8. Neville, Damm, Allen, Bouquot. Oral and Maxillofacial Pathology. 2nd Edition . Elsevier; 2004: 722-723.

✪ CASE REPORT

Acute Psychosis in a patient with Duchenne's Muscular Dystrophy

Fazal Mohamed A. M.

Joice Geo

Roy Abraham Kallivayalil

Abstract

Studies on Duchenne's muscular dystrophy (DMD) have focussed mainly on the genetic and the protein aspect of the disease. Neuropsychiatric disorders like Attention deficit hyperactivity disorder (ADHD), Autism spectrum disorder (ASD), Obsessive compulsive disorder (OCD) and learning disabilities (LD) have been found to be associated with DMD patients. This is a case report of a male DMD patient presenting with acute psychosis.

Keywords: Duchenne's muscular dystrophy, Acute psychosis

From:
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla, India - 689 101

Fazal Mohammed A M
Associate Professor

Joice Geo
Professor

John Shalu Mariam
Asistant Professor

Roy Abraham Kallivayalil
Professor & HOD

Department Of Psychiatry
PIMS & RC

Correspondence to:
Dr Fazal Mohammed A M
E-mail: fszalam@yahoo.com

Introduction

Duchenne muscular dystrophy (DMD) is a recessive X-linked form of muscular dystrophy, affecting around 1 in 3,600 boys, which results in muscle degeneration and premature death. It is the most common form of muscle dystrophy. Females are rarely affected. Dystrophin is a muscle protein which helps keep the muscles intact. In DMD there is absence of dystrophin. This is due to the mutation in the gene in the human X chromosome which codes for dystrophin.

The symptoms manifest by two to three years with an average life expectancy of 25 years. The usual symptoms include pseudo hypertrophy of muscles of the tongue and calf, frequent falls, difficulty walking, scoliosis, lumbar lordosis and fatigue. DMD patients are usually wheelchair bound by the end of first decade. By their teens their respiratory and cardiac muscles are affected.

Research in DMD has mainly focussed on muscle degeneration[1]. Evidence of CNS involvement has led to focus on neuropsychiatric disorders. Fitzpatrick et al[2] found that Dysthymia and depressive disorder was more common among boys with DMD. Neuropsychiatric disorders like ADHD, ASD and OCD are reported to be higher in DMD patients than in normal population[3].

Clinical Presentation

Mr. X is a 20 year old bedridden male diagnosed earlier to have DMD. He presented with reduced sleep and appetite with difficulty to breath for 10 days. He had fear, suspiciousness, refusing feeds, along with crying spells for the past three days. He was irritable and demanded to see a neighbouring girl whom he claimed was killed by his parents. He was very restless and was writhing in the bed. He felt that snakes kept by his father were crawling all over his body and tried to pull them away from his body. He accused his mother of having injected virus into his body. He became agitated and tried to assault his mother leading to admission to the psychiatry ward.

Mr. X was earlier diagnosed to have dilated cardiomyopathy as a complication of his diagnosis of DMD. His general examination showed hypertrophied tongue, knee and ankle deformities and scoliosis. Neurological examination showed muscle weakness of the limb muscles. His MSE revealed increased psychomotor activity and poor rapport. Delusion of persecution and tactile hallucination were present. Intelligence was average, His insight was grade 1. Cardiology consultation was done for breathlessness and he was continued on Digoxin, losartan, furosemide and spironolactone. Mr. X was diagnosed to have Acute psychotic disorder and

started on risperidone 2mg/day. He showed complete remission of his symptoms and was discharged after one week. Further follow up of Mr. X in the outpatient showed him to be symptom free.

Discussion

DMD is a hereditary condition with early onset of morbidity. Comorbid association of psychiatric problems have focussed mainly on the cognitive, learning and intelligence domains. Neuropsychiatric disorders like ADHD, ASD and OCD have also been reported. A pilot study of 10 patients showed a preponderance of ADHD4. Hendriksen et al[3] reported that among 351 males with DMD 11.7% had ADHD, 3.1% ASD and 4.8% OCD. Studies have also postulated the association of ASD with DMD in the genetic or the protein level[5]. Chronic stress and adjustment problems have also been reported among parents with DMD6. Mayana zatz et al[7] has reported a family of 4 out of 5 members with Becker muscular dystrophy having schizophrenia. Although association between schizophrenia and DMD have been suggested by other authors[8] there have been no reports on psychotic disorders among DMD patients. This case report proves the above postulation. Also Indian studies on DMD have not focussed on psychiatric problems.

Conclusion

The diagnosis of DMD underlines early onset of morbidity and mortality. Earlier studies have pointed to association of ADHD, OCD, ASD and Depressive disorder with DMD. The above case report points to the possibility of occurrence of psychotic disorder with DMD which has only been hypothesized till now.

References:

1. Eric P Hoffman, Kenneth H Fishbeck, Robert H Brown Characterization of dystrophin in muiscle biopsy specimen of patients with duchenne's and becker's muscular dystrophy. N Eng J Med 1988; 318, 1363 - 1368
2. Fitzpatrick.C, Barry.C, Garvey.C. Psychiatric disorders among boys with DMD. Dev Med & Child neurol;1986,28(5): 589-595
3. Joseph G M, Hendricksen, Johan S H Vlas, Neuropsychiatric disorders in males with DMD J.child neurology M
4. Margaret steele, Elizabeth taylor, Catherine young, Mental health of children and adoloscents with DMD; Dev med and child neurol 2008: 50, 8
5. Joyce y wu, karl C K kuban Association of DMD and ASD J child neurol 2005; 20:790 – 795
6. James poysky behaviour pattern in DMD – report on the parent project muscular dystrophy
7. Mayana zatz, Homero vallada,marco S melo, consaggregation of schizophrenia with becker muscular dystrophy, J med genetics 1983 30; 131- 134
8. Delisi le, Crow T J Sex chromosome anomalies and schizophrenia, Psychiatr gen 1991; 1; 101

✪ CASE REPORT

Osteochondroma of angle of the mandible : a case report

Akhilesh Prathap
Eapen Thomas
Ravirajan Areekal
Vinesh Udayakumar

Abstract

Osteochondroma / osteocartilagenous exostosis is a cartilage-capped exophytic lesion that arises from the cortex of bone. It constitutes 20 – 50% of all benign tumours and 10 – 15% of all bone tumours, but it is actually a developmental lesion rather than a true neoplasm. However, this process only rarely affects the craniofacial bones. In the craniofacial region, condyle and coronoid process of the mandible are the most commonly affected areas. This lesion may present in a solitary fashion or as multiple osteochondromas as part of an inherited syndrome that results in disturbances of skeletal growth and development. The treatment of osteochondroma is primarily surgical resection of the tumours. The present article reports the extremely rare case of OC arising from the angle of the mandible causing facial asymmetry.

Keywords: Osteochondroma (OC), osteoma ,benign bone tumors

From:
Pushpagiri College of Dental Sciences
Tiruvalla, India - 689 101

Akhilesh Prathap
Reader

Eapen Thomas
Professor & HoD

Ravirajan Areekal
IInd Year Post Graduate Student

Vinesh Udayakumar
Associate Professor

Dept. of OMFS
Pushpagiri College of Dental Sciences, Thiruvalla

Correspondence to:
Dr Akhilesh Prathap
E-mail: akhileshomfs@gmail.com

Introduction

Osteochondroma (OC) is also known as osteocartilaginous exostosis[1-4]. It is an exophytic lesion that arises from the cortex of bone and is capped with cartilage. In 1899, Jacob first described osteochondroma of the mandible which occurred on the coronoid process, forming a pseudoarthrosis joint between the coronoid process and the zygomatic arch[2].It is frequently observed on the metaphyses of long and flat bones, with no transition to the underlying normal bone. It is rather infrequent on the skull. The coronoid process and mandibular condyle are the most commonly affected areas, especially the medial aspect in the latter [2]The other reported sites are mandibular symphysis, body of mandible, in the soft tissues at the angle of the mandible, maxillary sinus and posterior maxilla[3-5] Different etiologies have been proposed. The most recent theory is based on the presence of nests of chondrocytes in the periosteum [3]. Mechanical stress may lead to hyperplasia of these cells, because the lesion is usually located in areas such as tendon insertions. On the mandible, especially the condyle, Meckel cartilage remains may play a role. OC is usually seen in younger individuals with male predominance[6]. OC may present in a solitary fashion or as multiple OCs seen in autosomal dominant syndrome known as osteochondromatosis[3,6]. OC occurring in the angle of the mandible is extremely rare and according to literature search, this is perhaps the only case at the angle of mandible.

Case Report

A 15-year-old male presented to a private hospital with a chief complaint of painless slow-growing swelling since 2 years in the left side of the lower jaw. There was no significant medical history. On examination, face was asymmetric due to swelling at the angle of mandible. It was a solitary swelling, well-localized, measuring 4 × 3 cm at the angle of mandible. On palpation it was nontender and hard. Orthopan-tomogram showed mixed radiolucent and radiopaque lesion in the angle of the mandible [Figure 1]. Computed tomography (CT) showed an irregular bony outgrowth at angle of mandible, measuring 12 × 14 × 6 mm in size, with a small irregular area of sclerosis. There were no cystic or destructive changes identified and no adjacent extraosseous soft tissue

swelling or fluid collection seen. The three-dimensional CT showed margin of the lesion to be continuous with the margins of the mandible [Figure 2]. Radiological diagnosis of benign OC was made. Under general anesthesia, through extraoral approach, submandibular incision, the lesion at the angle of mandible was exposed [Figure 3]. The lesion had a broad base and an irregular surface, excision of the lesion was done along with a margin of normal bone all around. Bony margin were smoothened and closed in layers[Figure 4& 6]. Pressure dressing was placed. Postoperative wound healing was uneventful. Histology revealed features of OC. Six month postoperative radiograph was obtained to rule out dry signs of recurrence.

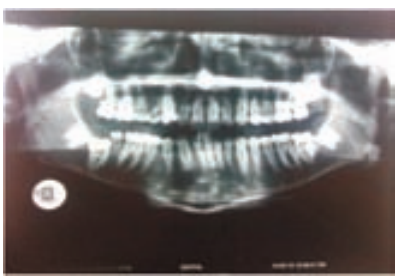


Fig. 1

Fig. 2

Fig. 3

Fig. 4

Fig. 5 Resected tumor

Fig. 6

Discussion

OC is a benign tumor exhibiting features of chondroma and osteoma together. OC is the most common osteogenictumor of the axial skeleton constituting up to 50%. OC accounts for 35.8% of benign bony tumors and 8.5% of bony tumors. This is most frequently found on the metphyseal region of long bone (femur, tibia etc) and unusual on the skull.

Osteochondroma most commonly seen metaphyseal region of long bones is also seen in ribs, scapulae, clavicles, and vertebrae, as well as the mandible rarely affecting coronoid process, symphysis, posterior maxilla, maxillary sinus, and zygomatic arch, with slowly changing occlusion, progressive facial asymmetry, and limited and painful mandibular movements.The growth of tumor is slow and is in coordination with the growth of the skeleton[2]. The tumor may be sessile or pedunculated and is usually asymptomatic. Solitary OC are exophytic lesions of bone arising from the cortex and covered by periosteum that is continuous with that of the adjacent bone and this accounts for 75% of the OCs[1]

Multiple OCs are associated with a syndrome known as osteochondromatosis.

Osteochondroma associated with langergiedion

Syndromeis accompanied by learning difficulties, redundant skin, multiple exostosis, characteristic facial features and cone shaped phalanges.

The pathogenesis of osteochondroma has been the subject of much debate. The most commonly accepted view is mataplastic change of the periosteum and or the osteochondral layer, leading to production of cartilage, which subsequently ossifies. Several theories have been proposed to explain the pathogenesis of osteochondroma. In 1891 Virchow[12]postulated the physeal theory: a portion of the cartilage becomes separated from the parent tissue then rotates 90 degrees and grows in a direction transverse to the long axis of the bone.In 1920 Keith[13]proposed a defect in the perichondral ring surrounding the physis is the cause of osteochondroma. Müller's periosteal theory states exostoses are produced by small nests of cartilage derived from the cambium layer of the periosteum.[14]Lichtenstein[15]theorized the periosteum has the potential to develop osteoblasts and chondroblasts. The osteochondroma could develop by spontaneous or induced metaplasia of the periosteum to form cartilage that subsequently undergoes endochondral ossification.

The lesion is usually discovered incidentally on radiographic examination or on palpation of a protruding mass in the affected area[4,9].The radiographic image is one of a globular, radiolucent, lobulated mass which distorts the normal morphology of the mandible. The radiographic appearance of osteochondroma can be pathognomonic, especially in long bones. The lesions usually point away from the joint space and can have a pedunculated stalk or sessile base.[14]On a conventional radiograph the osteochondroma may exhibit density with a sclerotic appearance. Computer assisted tomography (CT) scanning may help delineate the anatomy of the lesion and that of surrounding structures.[15]

Several authors have reported the usefulness of CT imaging [5-8]In diagnosing OC. CT visualizes abnormalities of tissues and the size of the lesion and clarifies its shape and composition, location, and relationships with the neighbouring structures.James et al. reported that although panoramic radiographic examination is useful for screening, superimposition oftumors can cause complications in diagnosis [(5).] In the present case, we ensured that the diagnosis was not based on panoramic radiography alone, and in fact CT images of the lesion were much clearer.3D reconstruction—also often referred to as 3DCT—is essential for confirming the diagnosis, for determining the size and relationship of the tumor to surrounding structures, and for surgical planning. Emekli et al. reported that 3D reconstruction was essential for revealing the dimensions of the mass and the relationships with the surrounding structures [6]. Etöz et al. reported that preoperative CT with 3D reconstruction was essential for establishing the exact extent of the lesion, and for deciding whether an intraoral or extraoral approach should be used [8]. Tavassol et al. reported that CT with 3D reconstruction was essential for determining the correct diagnosis, and specifically for surgical planning [8]. 3D reconstruction is essential for confirming the diagnosis, determining the size and relationship of the tumor to surrounding structures, and for surgical planning. We used CT imaging to confirm the presence of a mass with the same morphological characteristics as those described in previous reports.

Histologically, OC can be diagnosed by the presence of bony trabeculae covered with a cartilaginous cap. OC should be differentiated histologically from osteoma which consists of hard dense compact lamellar bone; benign osteoblastoma containing well-vascularized connective tissue stroma and widely dilated capillaries with active production of osteoid and woven bone; chondroma consisting of lobules of hyaline cartilage with chondrocytes within well-formed lacunae and chondroblastoma consisting of broad areas of chondrocytes.[8]

Complete resection with surrounding periosteum is curative. Recurrences of OC are rarely reported[9].Malignant transformation of OC is about 2% in solitary OC and 11% in osteochondromatosis.[10]

Conclusion

Though osteochondroma is commonly seen in long bones it has a significant presence in oral and maxillofacial region most commonly affecting mandibular condyle, where the cause could be the trauma to the temperomandibular joint.Many anatomical variations of the lesion in condyle are seen through different radiological methods of intrepretations, diagnosis can be through the clincal and radiological assessment, conclusive would be histological where the striking feature is presence of cartilaginous cap. This paper describes the details of a very rare case of OC arising in the angle of the mandible. CT along with histopathological examination confirmed the diagnosis. OC are benign slow growing tumors. They have a low risk of recurrence and malignant transformation. Regular clinical and radiological follow-up is required.

Reference

1. Ortakoglu K, Akeam T, Sencimen M, Karakoc O, Ozyigit HA, Bengi O. Osteochondroma of the mandible causing severe facial asymmetry: A case report. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 2007;103:e21-8.
2. Navaneetham A, Rao KA, Kumaran S, Baweja HH. A unique case of multiple osteochondroma: Mandibular symphysis and femur. Ann MaxillofacSurg 2012;2:182-4.
3. Ongle R, Pillai RS, Ahsan AK, Pai KM. Osteochondroma of the mandibular condyle. Saudi Med J 2003;24:213-6.
4. Ashok KK, Omprakash TL, Goudar GN, Ravikumar R. Osteochondroma of mandibular condyle. J Dent Sci Res 2010;1:57-66.

5. Totsuka Y, Fukuda H, Iizuka T, Shindoh M, Amemiya A (1990) Osteochondroma of the coronoid process of the mandible. Report of a case showing histological evidence of neoplasia. J CraniomaxillofacSurg 18, 27-32.

6. Kerscher A, Piette E, Tideman H, Wu PC (1993) Osteochondroma of the coronoid process of the mandible. Report of a case and review of the literature. Oral Surg Oral Med Oral Pathol 75, 559-564.

7. Etöz OA, Alkan A, Yikilmaz A (2009) Osteochondroma of the mandibular coronoid process: a rare cause of limited mouth opening. Br J Oral MaxillofacSurg 47, 409-411.

8. Tavassol F, Spalthoff S, Essig H, Bredt M, Gellrich NC, Kokemüller H (2012) Elongated coronoid process: CT-based quantitative analysis of the coronoid process and review of literature. Int J Oral MaxillofacSurg 41, 331-338.

9. Nanda Kishore D, Shiva Kumar HR, Umashankar KV, Rai KK. Osteochondroma of the mandible: A rare case report. Case Rep Pathol 2013;2013:167862.

10. González-Otero S, Navarro-Cuéllar C, Escrig-de gTeigeiro M, Fernández-Alba-Luengo J, Navarro-Vila C. Osteochondroma of the mandibular condyle: Resection and reconstruction using vertical sliding osteotomy of the mandibular ramus. Med Oral Patol Oral Cir Bucal 2009;14:E194-7.

11. Koga K, Toyama M, Kurita K. Osteochondroma of the mandibular angle: Report of a case. J Oral MaxillofacSurg 1996;54:510-3.

12. Keith A. Studies on the anatomical changes which accompany certain growth disorders of the human body. J Anat 1920; 54:101-10.

13. Müller E. Über hereditare multiple cartilaginäreExostosen und Ecchondrosen. BeitrPatholAnat 1913; 57:232-313.

14 Azaz B, Zelter R, Nitzan DW. Pathoses of coronoid process as a cause of mouth-openingrestrictions. Oral Surg Oral Med Oral Pathol 1994; 77(6):579-84.

15. Gaines JR RE, Lee MB. Osteochondroma of the mandibular condyle: case report an review of the literature. J Oral MaxillofacSurg 1992; 50:899-903.

16. Karras SC, Wolford LM, Cottrell DA. Concurrent osteochondroma of the mandibular condyle report of a case and review of the literature. J OralMaxillofacSurg 1996; 54:640.

✪ CASE REPORT

Static lung volumes and D_{LCO} in patients cured of pulmonary tuberculosis

Doye george
Sukumaran P
Mathew Ninan
Sethu Lekshmi A

From:
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla, India - 689 101

Doye George
Associate Professor

Sukumaran P
Professor

Mathew Ninan
Associate Professor

Sethu Lekshmi A
Respiratory therapist

Department Of Pulmonary Medicine
PIMS & RC

Correspondence to:
Dr Doye George
E-mail: drdoye@yahoo.com

Abstract

Static lung volumes- Vital capacity, Functional residual capacity, Total lung capacity, and Residual volume- and Diffusing capacity of the lung for Carbon monoxide were measured in cured cases of Pulmonary tuberculosis two to four years after successful completion of treatment. The patients were divided into four groups depending on the extent of residual radiological lesions—complete clearing, minimal, moderately advanced and far advanced. The extent of changes in static lung volumes and D_{LCO} in the different groups were compared. Only in smokers with far advanced residual lesions there were significant abnormalities. They showed significantly higher FRC, RV, and RV/TLC ratio and also reduced diffusing capacity. This shows that smoking and larger extent of residual lesion contribute to development of obstructive airways disease and reduction in D_{LCO} values.

Keywords

Tuberculosis, pulmonary, cured, vital capacity, functional residual capacity, total lung capacity, residual volume, diffusing capacity, lungs

Introduction

Pulmonary tuberculosis is a disease which can be completely cured by chemotherapy. India is the country with the highest number of Tuberculosis patients in the world. India's Revised National Tuberculosis Control Programme has now achieved an overall cure rate of more than 90 per cent. In 2014 more than 11lakh pulmonary tuberculosis cases were notified in India[17.] So there are a large number of patients in India who are cured of tuberculosis and are leading a normal life.

Tuberculosis heals by fibrosis and calcification, which are likely to cause impairment in lung function. There are a large number of studies which have shown that pulmonary tuberculosis impairs lung function in its active phase[1 2 3]. But very few workers have studied the state of lung function in cured cases of pulmonary tuberculosis (PTB)[4 16].

A previous study from our department[5] has shown that about 10 percent of cured cases of PTB showed evidence of restrictive ventilatory defect and that about 37 per cent showed airway obstruction. It was also observed that the greater the extent of residual lesion the severer the extent of airway obstruction. In that study only spirometry was done in 54 patients cured of PTB.

Some studies on the effect of drug treatment on pulmonary function have been reported[6 7]. Obstructive, restrictive and obstructive-cum-restrictive defects have been reported[8 9 10]. Some workers have also demonstrated reduction in diffusing capacity of the lungs in patients with pulmonary tuberculosis[15 16].

The present study is an attempt to find out the static lung volumes in these patients. The lung volumes studied are

1. Vital capacity
2. Functional residual capacity
3. Residual volume
4. Total lung capacity

Pulmonary gas exchange was assessed by measuring diffusing capacity of the lungs for carbon monoxide in these patients.

Materials and methods

All patients selected for the study were cured PTB patients. They were included in the study 2 to 4 years after successful completion of treatment. An X-ray chest PA view was done in all

patients to assess the extent of residual lesion. Sputum AFB (2 samples) was done in patients who complained of cough. They were included in the study only if sputum AFB was negative. ECG was done in all patients and patients with cardiac problems were excluded from the study.

Vital capacity was measured in 54 patients. Due to inconvenience of some patients, other static lung volumes were measured only in 49 patients and D_{LCO} in 39 patients.

in 39 patients.

The patients were divided into four groups depending on the extent of residual lesions on the chest X-ray PA view – Complete clearing, minimal, moderately advanced and far advanced. This was based on the Tuberculosis Association of India Classification of Tuberculosis[11].

1.*Complete clearing- no residual lesions*

2. M*inimal- lesions* involving one or both lungs, total extent not exceeding the volume of one lung on one side upto the level of the lowest point of the second costochondral junction.

3. *Moderately advanced* – lesions involving one lung or both lungs, total extent not exceeding the volume of one lung upto the level of the lowest point of the fourth costochondral junction.

4. *Far advanced*- lesions more extensive than moderately advanced.

In each of the above groups patients were further subdivided into smokers and nonsmokers.

The extent of changes in static lung volumes and D_{LCO} in the different groups were compared.

Geratherm Respiratory Body plethismograph was used for the study. Predicted values were

Results

The observations in 54 patients have been analyzed. The mean values +/- standard deviation of age, height, weight and body surface area of these patients were 37.6+/_ 11.3 years, 161.6+/_5.8 cms., 50.2+/_8.4 kg, and 1.51+/_0.13 m^2 respectively.

Distribution of patients according to age groups, smoking habits, extent of residual lesions and presence or absence of respiratory symptoms is shown in Table 1. It can be seen that 40(74%) of the 54 patients were smokers. On the basis of residual extent of radiological lesions 14.8% showed complete clearing, 63% showed minimal, 18.5% moderately advanced and 3.7% far advanced lesions. Forty one (75.9%) were symptom free, while 4(7.4%) had cough with expectoration, 1(1.9%) had dyspnoea on exertion, and 8(14.8%) had cough and dyspnoea at the time of inclusion in the study. All the 12 patients who complained of cough with expectoration had negative sputum- AFB (2samples) results. 12 lead ECG was taken in all patients. None of them showed any evidence of cardiac disease.

Table -1: Distribution of patients according to age, Smoking habits, extent of disease and symptoms.

		Age Groups				
		20-29	30-39	40-49	50-59	Total
Smoking habits	Smokers	10	12	7	11	40(74.1%)
	Non-Smokers	9	3	2	0	14(25.9%)
Residual extent of disease (RED)	Complete clearing	4	2	2	0	8(14.8%)
	Minimal	11	11	6	6	34(63.0%)
	Mod. advanced	3	2	1	4	10(18.5%)
	Far advanced	1	0	0	1	2(3.7%)
Symptoms	**Asymptomatic**	17	13	6	5	41(75.9%)
	Symptomatic					
	Cough With expectoration	1	2	0	1	4(7.4%)
	Dyspnoea on exertion	0	0	1	0	1(1.9%)
	Cough with expectoration and dyspnoea on exertion	1	0	2	5	8(14.8%)
	Total	19	15	9	11	54

Table -2 :Mean value ± SD of VC in patients according to the extent of disease

Extent of disease	Smoking habit	No.of patients	VC % predicted
Residual lesion			
Complete clearing	Smokers	6	87.73±10.33
	Non-Smokers	2	91.4±5.09
	Total	8	88.65±9.10
Minimal	Smokers	27	89.59±12.36
	Non-Smokers	7	93.79±9.23
	Total	34	90.46±11.79
Mod. advanced	Smokers	5	72.14±4.69
	Non-Smokers	5	85.16±18.81
	Total	10	78.65±14.63
Far Advanced	Smokers	2	70.70±14.71
	Non-Smokers	0	
	Total	2	70.70±14.71
Total		54	87.27±13.00

Table 3: Mean value ±SD of functional residual capacity (FRC),residual volume,Total Lung Capacity (TLC) in patients according to the extent of disease

Extent of disease	Smoking habit	No.of patients	FRV % predicted	RV % predicted	TLC predicted	RV/TLC %
1	2	3	4	5	6	7
Residual lesion						
Complete clearing	Smokers	5	80.1±19.92	82.36±37.66	88.34±10.79	25.06±8.63
	Non-Smokers	2	96.05±39.39	85.25±29.63	90.2±4.81	25.05±8.27
	Total	7	84.66±24.16	83.19±33.07	88.87±9.07	25.06±7.81
Minimal	Smokers	25	104.17±34.69	106.96±57.93	96.24±18.28	32.3±10.58
	Non-Smokers	6	106.08±47.65	116.42±84.27	101.38±23.44	29.47±13.67
	Total	31	104.54±36.63	108.79±62.31	97.25±19.06	31.75±11.04
Mod. advanced	Smokers	4	95.88±11.36	104.95±18.45	83.28±6.2	42.75±8.04
	Non-Smokers	5	106.08±32.84	138.94±66.0	100.62±23.44	37.52±8.37
	Total	9	101.54±24.83	123.83±51.25	92.91±19.3	39.84±8.18
Far Advanced	Smokers	2	127.65±33.59	167.9±46.95	99.65±19.45	49.9±4.1
	Non-Smokers	0	0	0	0	0
	Total	2	127.65±33.59	167.9±46.95	99.65±19.45	49.9±4.1
Total		49	102.09±33.37	110.31±57.69	95.35±17.82	33.02±11.23

Table -4 :Distribution of TLC % predicted in patients

Test	Age Group	80% n	70-79% n	60-69% n	50-59% n	50% n
	20-29	18	0	0	0	0
	30-39	13	1	1	0	0
TLC% Predicted	40-49	6	1	1	0	0
	50-59	8	0	0	0	0
	Total	45	2	2	0	0
TLC was estimated in only 49 patients						

Table-5:Mean values ± SD of diffusing capacity of the lungs (D_{LCO}) in patients according to extent of disease

Extent of disease	Smoking habit	No.of patients	D_{LCO} -ml/min mm Hg
Residual lesion			
Complete clearing	Smokers	5	10.03±3.27
	Non-Smokers	1	18.10
	Total	5	11.37±4.41
Minimal	Smokers	18	9.60±4.35
	Non-Smokers	5	8.94±1.61
	Total	23	9.46±3.89
Mod. advanced	Smokers	4	6.51±1.98
	Non-Smokers	4	11.38±2.15
	Total	8	8.94±3.23
Far Advanced	Smokers	2	6.17±3.04
	Non-Smokers	0	0
Total	Total	2	6.17±3.04
		39	9.48±3.83

Vital Capacity

Table 2 shows that the greater the extent of residual lesions, the greater the reduction in vital capacity. This reduction in VC was statistically significant in smokers ($p<0.05$).

Mean value of ± SD of FRC, RV, TLC, and RV/TLC ratio according to extent of disease - (Table3)

FRC and RV did not show any significant deviation from their mean predicted values in patients with less than far advanced lesions. In far advanced groups the mean values of FRC and RV were much higher than their predicted normal values. This increase should be viewed the light of the fact that in this group there was a reduction of vital capacity. TLC showed no definite trend with the extent of residual lesions. RV/TLC% showed a systematic rise with increase in extent of residual lesions. With greater extent of residual lesions the rise RV/TLC was statistically significant in smokers. ($p<0.05$).

Pulmonary gas exchange

Diffusing capacity of the lungs was measured in 39 patients and the results are shown in table 4. In those with minimal and moderately advanced extent of residual lesions the mean values of D_{LCO} were close to normal. But in patients with far advanced lesions the diffusing capacity was slightly below normal. Smokers on the whole show lower values than nonsmokers. Smokers with far advanced lesions show very much reduced D_{LCO} compared to nonsmokers.

Discussion

One may expect a restrictive defect in lung function with increase in extent of residual radiological lesions. As the pathology of PTB mainly involves the lung parenchyma and pleura it is not surprising that the lung volumes in the active phase of the disease are reduced according to the extent of the disease[1 2 3]. Our previous study (Spirometry in treated cases of pulmonary tuberculosis)[5] showed that with more extensive residual lesions there was actually more severe airway obstruction. Fibrosis causing narrowing of the airways, peribronchial fibrosis, post-tubercular bronchiectasis and development of emphysema might have contributed to this.

In the present study the mean values of static lung volumes are very near their predicted normal values. About 90% of the patients showed TLC greater than 80% of predicted values. Only the remaining 10% showed TLC values which were reduced. The values of Vital Capacity also showed a similar trend. It seems that successful treatment with chemotherapeutic drugs successfully resolved most of the lesions to restore distensibility of the alveoli or else the healthier alveoli have undergone over distention to compensate for the diseased alveoli. Improvement in lung volumes after treatment has been noticed[4 6].

Several workers have noticed airway obstruction in 22 to 80 % of treated cases of PTB[3 13 14 15]. Our previous study[5] published in 2015 showed airway obstruction in 37% of treated PTB cases. Most of the patients were smokers and it was noticed that in smokers, increased extent of residual disease led to increased airway obstruction. So in addition to smoking, extent of residual disease also contributed to airway obstruction. The systematic rise in RV/TLC %, with increase of residual extent of disease is an indication of the obstructive airways disease in such patients.

This study of static lung volumes also corroborates the finding of our previous study[5] that greater extent of residual lesions leads to greater airway obstruction. Smokers with far advanced residual lesions show greater extent airway obstruction and also reduction in DL_{CO} values. Emphysematous changes and ventilation-perfusion mismatching in such patients might have contributed to the reduced D_{LCO} values.

The gas-transfer in the lungs in the present series shows slight to moderately severe impairment; the latter is more marked in smokers compared to non-smokers. In smokers presumably the presence of emphysema might have caused the decreased gas transfer in the lungs. Other workers have also demonstrated reduced diffusing capacity of the lungs which was related to the extent of the disease[15 16]. It has been observed that that reduction in diffusing capacity of the lungs was related to the presence of airway obstruction[12].

Conclusions

Successful treatment with anti-tuberculosis drugs restore lung function to near normal levels in most of the patients with less than far advanced residual lesions. However smokers with far advanced residual lesions mostly show FRC and RV which are significantly higher than their predicted normal values. RV/TLC ratio showed a statistically significant rise with increase in extent of residual lesions. Smokers with far advanced residual lesion showed reduced DL_{CO} values.

This study corroborates the findings of our previous study[5] that in patients with healed PTB, smoking as well as greater extent of residual lesions contribute to development of obstructive airways disease.

How can one prevent this? Two measures--- (1) early diagnosis of PTB and correct complete treatment, so that residual lesions will be minimal and (2) cessation of smoking in all patients who are on treatment for PTB.

References

1. Birth G, Caro J, Mamberg R,and Simson B G Airways obstruction in pulmonary tuberculosis .Scand J Respir Dis 1966,47,27
2. Hnizdo E,Singh T,Churchyard G,Chronic Pulmonary function impairment caused by initial and recurrent recurrent pulmonary tuberculosis following treatment ,Thorax 2000,55:32-38
3. Pasipanodya J G,Miller T L,Munguia G,Pulmonary impairment after tuberculosis Chest 2007131(6) 1817-1824
4. Malik S K, Martin C J,Tuberculosia,corticosteroid treatment and pulmonary function, Am Rev Respir Dis 1969,100,13
5. Doye George,Sethu Babu,P.Sukumaran,Anu Sam Mathew,Salini M R,Spirometry in Treated Case of Pummonary tuberculosis Jounal of Medical Science And Clinical Research,Volume 03,Issue 03.March 2015
6. Plit M L,Anderson R,Van Rosenburg C E,Influence of antimicrobial therapy on spirometric parameters and proinflammatory indices in severe pulmonary tuberculosis European Respir J,August 1998, vol 12,no.2 ,351-356
7. Long R,Maycher B,Dhar A,Manfreda J,Hershfield E,Anthonisen N,pulmonary tuberculosis treated with directly observed therapy serial changes in lung structure and function,Chest April 1998,113(4)933-943
8. Maguire G P,Anstey N M,Adrian M,Pulmonary tuberculosis,impaired lung function,disability and quality of life in a high burden setting,international J TB and Chest Dis 2009,13.12,pp 1500-1506
9. De Valliere S,Barker RD.Impaired lung function after treatment for MDR-TB Int J TB and LD 2004 June N.6 pp 767-771
10. Menzes MB,Hallal PC,Perez-Padilla R,Tuberculosis and air flow obstruction evidence from PLATINO study in Latin America,European Res J,vol 30 No. 6 1180-1185
11. Tuberculosis association of India (1969):Classification of tuberculosis
12. Nefedov VB, Popora LA,SherquiraE A,Function in patients with chronic fibro cavernous tuberculosis(Russian) Probl Tuberk Bolezn Leqk 2008 (4) 24 -8
13. M Chuslain,S yartsev,Dzhutikov, The impairment of respiratory function in patients with treated pulmonary tuberculosis Chest Oct-2011,vol 140 no.4
14. Aggarwal D, Vivek K U,Asok K,Janmija Lung function defects in treated pulmonary tuberculosis patients European Resp.J. Jan 2016
15. Singh B,Chaudhary O trends of Pulmonary impairment in persons with treated pulmonary tuberculosis 2015;1 (1) ;8-11
16. Nefedov VB,Izamalova Z F,Dzhenzhera EN Lung diffusion capacity of Pulmonary tuberculosis patients (Russian) Ter Arkh 1987 59(7); 65-9
17. www.tbcindia.nic.in ,TB India 2015-RNTCP Annual Status Report

✪ CASE REPORT

Ultrasound guided nerve block for emergency knee arthrotomy in a cirrhotic patient in septic shock

Litha Mary Mathew
Rajiv Alex
Rosely Thomas

From:
Pushpagiri Institute of Medical Sciences & Research Centre
Tiruvalla, India - 689 101

Litha Mary Mathew
Senior Resident

Rajiv Alex
P G Student

Rosely Thomas
Profesor & HoD

Department Of Anaesthesia
PIMS & RC

Correspondence to:
Dr Litha Mary Mathew
E-mail: lithchery@gmail.com

Abstract

A critically ill patient posted for emergency surgery poses many challenges to an anaesthetist. As he is not an ideal candidate for general anaesthesia, central neuraxial blocks and blind regional techniques are usually done. We report such a case which was managed successfully with ultrasound guided nerve block.

Keywords: Chronic liver disease, septic shock, coagulopathy, emergency kneearthrotomy, ultrasound guided femoral nerve block.

Introduction

The use of ultrasound to guide peripheral nerve blocks is becoming increasingly frequent.[1,2,3] The advantages include a more complete block, local anaesthetic volume sparing and fewer vascular puncture.[1]

Case Report

A 54year old male patient, a known case of alcoholic liver disease presented with fever, swelling and pain in right knee of 4 days duration. General examination showed pallor, icterus, bilateral pitting pedal edema. Pulse rate was 110/min, blood pressure was 76/50mm of Hg and SpO2 was 92% on room air. Systemic examination showed tenderness in the right hypogastrium. Local examination of right knee showed edema, local rise in temperature and restricted movements. Laboratory workup revealed deranged renal and liver parameters, coagulopathy, anemia and raised WBC count(Hb: 8.4g%, TC: 31,100/mm3, PT: T/C-26.9/14.6sec, aPTT: T/C-55.7/32sec, INR: 2.08, urea: 90mg%, creatinine: 0.89mg%, bilirubin: total/direct- 4.5/2.7mg%, A/G: 0.5). Ultrasound abdomen showed features of chronic liver disease. ECG and Chest X-ray were normal. A diagnosis of chronic liver disease with septic arthritis knee and septic shock was made. The patient was posted for emergency knee arthrotomy to control the source of infection. Patient was optimised with fluid resuscitation, ionotropic support with noradrenaline, blood products, antibiotics, vitamin K and thiamine. He had a fasting period of 6 hours.

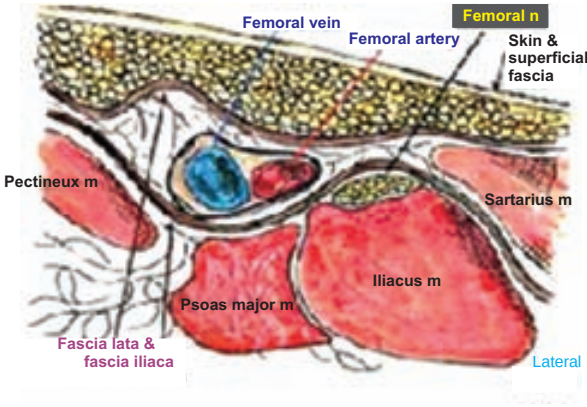
Patient arrived in the theatre with two patent IV cannulas and Noradrenaline infusion on flow. Oxygen was given via a face mask at 5l/min. Preinduction monitors were ECG, SpO2 and NIBP. He was premedicated with injection Ranitidine 50mg, injection Ondansetron 4mg and injection Midazolam 1mg intravenously. Plan of anaesthesia was ultrasound guided femoral nerve block. Under aseptic precautions, the linear transducer (GE Venue 40) was placed over the right inguinal region to visualise the femoral artery, vein and the nerve. Nerve block was given successfully with a 25 guage spinal needle using the in-plane technique with 10ml of 2% lignocaine with adrenaline and 10ml of 0.5% Bupivacaine. The procedure lasted for 1hour, during which the patient had adequate analgesia.

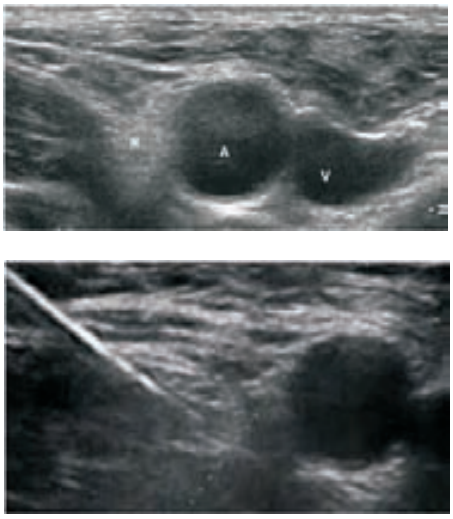
Discussion

A chronic liver disease patient in septic shock coming for emergency surgery poses several challenges to the anaesthetist. Patients with liver disease have a limited hepatic reserve and are vulnerable to surgical and anaesthetic stress.[4] General anaesthesia in such a patient carries the risk of haemodynamic instability, aspiration and hepatic encephalopathy. Central neuraxial blocks are contraindicated because ofhaemodynamic instability and coagulopathy. In a patient with coagulopathy, blind nerve blocks carry the risk of accidental vascular puncture and haematoma formation.[3,5] In this patient ultrasound guided femoral nerve block proved to be an efficient and safe alternative.

The use of ultrasound to guide puncture in peripheral nerve blocks has become increasingly frequent.[1,2,3] The advantages of using ultrasound include a more complete block, local anaesthetic volume sparing, fewer vascular puncture, lower failure rates, faster onset of action and longer duration of action.[1,2,3]

The femoral nerve usually lies lateral to the femoral artery in the groove formed by the iliacus and psoas muscles.[1,2] For a femoral nerve block, a broad(35-50mm foot print) linear transducer is used.[1,2] Both in-plane and out-of-plane approaches can be used.[1,2] The advantage of the in-plane approach is visualisation of the approaching needle.[1] The out-of-plane approach is used for catheter placement.[1] For either approach needle tip is positioned between the fascia iliaca and iliopsoas muscle near the lateral corner of the femoral nerve to avoid the femoral vessels.[1,2] The desired distribution is local anaesthetic layering under or completely around the femoral nerve.[1,2] For patients who are obese, femoral nerve imaging is challenging. Ultrasound can be combined with nerve stimulation for successful block in these patients.[1,2]





Lignocaine with adrenaline and Bupivacaine were used for the block.Addition of adrenaline prolongs the duration of action of lignocaine. Bupivacaine has a duration of action of 4-8hours.Thus the patient had adequate analgesia during the intraoperative period as well as in the postoperative period.

Conclusion

Ultrasound is a guidance tool that many people are choosing for regional analgesia blocks. It has become an effective and safe alternative to general anaesthesia, central neuraxial blocks and blind nerve blocks in critically ill patients.

References:

1. Miller`s anaesthesia, Eight Edition; Volume 1 ; Chapter 58 :page 1752
2. Morgan and Mikhail's Clinical Anaesthesiology, Fifth Edition; Chapter 46; page 1003
3. Ultrasound guided femoral and sciatic nerve blocks in an anticoagulated patient: case repots.Rev BrasAnaesthesiology 2010; 60:4:422-428
4. Ultrasound guided ipsilateral transverse abdominus plane and ilioinguinal-hypogastric nerve block foringuinal hernia repair in patients with liver cirrhosis. Ains shams journal of anaesthesiology 2014, may; 7(1): 51-58
5. A case report of ultrasound guided peripheral nerve block for lower extremity amputation of a patient with anti-phospholipid syndrome. Masni 2013, june; 62(6): 718-720.

✪ CASE REPORT AND LITERATURE REVIEW

Pheochromocytoma - Anaesthetic Management for Laproscopic Excision

Dr Mary Mammen DNB
Dr Rajamma Cherian MD
Dr Rosily Thomas MD

From:
Pushpagiri Institute of Medical Sciences
& Research Centre
Tiruvalla, India - 689 101

Dr Mary Mammen DNB
Assistant Professor

Dr Rajamma Cherian MD
Professor

Dr Rosily Thomas MD
Professor & HOD

Department Of Anaesthesiology
PIMS & RC

Correspondence to:
Dr Mary Mammen
E-mail: drnebuim@yahoo.co.in

Introduction

Pheochromocytoma is a tumour of the adrenal medulla. The tumour produces, stores and secretes catecholamines – both epinephrine and nor epinephrine.Incidence of this tumour in hypertensive patients is less than 0.2%. 85 to 90% of tumours are solitary. It is also called the 10% tumour. Clinical presentation can be at any age. Tumours are not innervated, So there is no neurogenic control for catecholamine release. Attacks of hypertension may be paroxysmal. Patients may be normal in between (1)

Case Report

A 58year old male patient weighing 47 kg presented with complains of chest discomfort and breathlessness. He was a known case of hypertension, Diabetes Mellitus and recently detected Coronary artery disease. ECG showed ST-T wave changes, ECHO showed concentric left ventricular hypertrophy, regional wall motion abnormality and ejection fraction of 59%. His troponin level was raised (0.28). BP was high and fluctuating around 250 - 280/120-130. CT showed a mass lesion in left adrenal gland 5 -6 cm in sze. Biochemical evaluation could not confirm pheochromocytoma. (Metanephrine 0.53ng/ml.). He was posted for laproscopic left adrenelectomy. He was on Atorvostatin 20mg, Amlodepine 5mgm, Aldactone12.5mgm, Frusemide 20mgm, Clonidine 100microgm/day. His blood sugars were controlled with Insulin. Hemoglobin was 15.4 on admission. Other investigations were within normal limits. He was admitted 2 days before surgery, for control of fluctuating BP and early hydration. NTG infusion was started with haemodynamic monitoring, oxygen by mask and intravenous fluids. After premedication patient was shifted to the operating room on NTG infusion and oxygen by mask. Arterial and cvp lines were inserted . His BP varied between 250/120 -280/130.The heart rate was normal. Patient was induced with 100microgm of Fentanyl, Midazolam 2mg, .Morphine 9mg, Xylocard 60mg, and Propofol 100mg. Precurarisation was done with Vecuronium, and he was intubated on Succinylcholine 100mgm. NTG infusion, Sevoflurane 1-3%, additional dose of propofol were used to control blood pressure. CVP was maintained at 8-10 cms of H2O. Beta blocker-Esmolol was also used to controll intra op BP. The fall in BP after adrenal vein ligation was supported with Noradrenaline infusion and crystalloids. Duration of surgery was 3 hrs .The patient was ventilated for a day electively on SIMV mode with pressure support. He was extubated the next morning. His BP was120/80, PR-92/mt, regular, CVP-7cms of H2O, FBS-111mgm/dl, Na 139, K 3.8, S.creatine 0.92. No new ECG changes were noted. ABG was normal. His Hb was10.1gms. He was discharged after a week and the biopsy was reported as pheochromocytoma.

Discussion

Pheochromocytoma is a catecholamine secreting tumour arising from the chromaffin cells of the sympatho adrenal systems. It accounts for 0.1 % of all cases of hypertension. 80%of Pheochromocytomas are located in the adrenal medulla [2]. Adult Pheochromocytomas are solid highly vascular tumours 3 to 5cms in diameter with an average weight of 100gms. Most Pheochromocytomas

secrete Norepinephrine in high quantities. The release of catecholamines is mostly sustained. It can be paroxysmal also. Sustained hypertension is the commonest presentation in more than 80% of adults. The head ache which follows alerts physicians only when it becomes intermittent, regular and severe, associated with nausea and vomiting. Prolonged exposure to circulating Norepinephrine results in both arteriolar and venous segment constriction and marked decrease in circulating blood volume[3]. Induction of anaesthesia can produce profound arteriolar hypotension. Persistent untreated hypertension results in left ventricular failure, systemic arterial shutdown, severe metabolic acidosis and death. Malignant hypertension, cerebrovascular accidents, myocardial infarction, left ventricular outflow obstruction are also observed. Catecholamine induced cardiomyopathy - both dilated and hypertrophic - can occur due to Norepinephrine secreting tumours. This cardiomyopathy is treatable if it is treated before fibrosis occurs [4]. Glycogenolysis and impaired insulin release by islet cells of pancreas lead to increased blood sugar.Epinephrine secreting tumours produce paroxysmal tachycardia presenting as palpitation, sweating, fainting, blanching associated with a feeling of panic and doom. Dopamine causes vasodilatation of the GI tract and produces nausea and vomiting. Dopamine also produces bladder outlet obstruction [2]. The most sensitive test in the diagnosis of Phechromocytoma is plasma free Normetanephrine and metanephrine level estimation. Normetanephrine level more than 400pg/ml, metephrine more than 200pg/ml and plasma total catecholamine level more than 200pg/ml are highly suggestive of Pheochromocytoma. The single best urinary finding is elevated metanephrine in 24hour urine study. High performance liquid chromatography with electrochemical detection will give accurate levels of Epinephrine. CAT scan and MRI scan can provide accurate consistent identification of majority of Pheochromocytomas. CT can detect tumours as small as 1 cm. Cystic Pheochromocytomas are best identified and characterisedwith MRI. Recurrent tumours, metastaic tumours and tumours in unusual situations are identified with MIBG scans. PET scan and selective venous catheterization and sampling of catecholamines from the adrenal vein are other useful tests. Germline mutation in genes has been identified to be responsible for familial Pheochromocytoma(5). Micro RNA profiling is a novel diagnostic method. Main objective in medical management of Phechromocytoma is control of hypertension. Use of Alpha 1 adrenoreceptor antagonist decreases the mortality from 60% in 1951 to 0 to 6% in 1967. Alpha blockers lower blood pressure, increase intravascular volume, prevent paroxysmal hypertensive response, improve myocardial function and allow resensitisation of adrenergic receptors. In emergency situations rapid intravenous alpha blockade can be tried with Phenoxybenzamine infusion in gradually increasing doses over a period of three days. Beta blockers are added only after ensuring adequate arteriolar dilatation. Calcium channel blockers and ACE inhibitors are also used. Calcium channel blockers prevent catecholamine mediated coronary spasm and does not produce hypotension[6]. Alphamethyl Tyrosine inhibits Tyrosine hydroxyllate and decreases catecholamine production by 50 to 80 %. It is useful in malignant Pheochromocytoma.

ALPHA ANTAGONISTS	Dose
Phenoxybenzamine	10-20mg
Phentolamine	BID 1-2 mg
Prazocin	IV1-4mg
Doxazocin	BID2-6 mg OD
BETABLOCKERS	
Atenolol	50-100mg/day
Propranolol	80-120mg/day
Metprolol	50-200mg/day
Celiprolol	200-400mg/day
Carvidilol	12.5-50mg/day
Esmolol	200-500microgm/kg/mt.
Labetolol	50-100mg/day
CALCIUM CHANNEL BLOCKERS	
Nicardipine	1-2microgm/day
Nifidepine	30-90mg/day
Diltiazem	100-200mg/day
Verapamil	200-400mg/day
ARTERIOVENOUS DILATORS	
Sodium nitroprusside	0.5 to1.5 microgm/kg/mt
Nitroglycerine	0.5 to 10 microgm/kg/mt
Magnesium sulphate	2-4gm intravenous
OTHERS	
Prostaglandins	
Adenosine	0.05-0.3mg/kg

(Table 1)

Laparoscopic adrenalectomy allows early mobilization, less hospital stay and less post operative pain[7]. Insufflation of carbon dioxide during laparoscopy and manipulation of highly vascular tumour can release more catecholamines and lead to hypertensive crisis..

Catecholamines cause contraction of spleen thereby increasing red cell production. Mesentric vasoconstriction causes nausea and vomiting. Renal vasoconstriction slows renal function. Exercise, postural changes, emotions, hypoglycemia and palpation of abdomen can stimulate catecholamine release [2]

Preoperative evaluation should conform to Roizen's criteria [8].

1.In hospital recording of BP should be less than 160/95 for 24 hours prior to surgery.

2.Presence of orthostatic hypotension.

3.No ST-T wave changes on ECG for one week.

4.Not more than one arrhythmia in 5 minutes.

5.Serial hematocrit checking.

6.Improvement of psychological aspects.

The goal of preparation is control of blood pressure, resolution of symptoms, elimination of ST-T wave changes and arrhythmias on ECG. After tumour excision, withdrawal of catecholamines can cause hypovolemia, persistent fatigue, somnolescence, hypoglycemia and a fall in hematocrit. In catecholamine resistant hypotension, vasopressin can be used (9).

Anaesthetic management - The goal of anaesthetic management is control of hemodynamic status, maintenance of intravascular volume, good analgesia and to provide adequate depth of Anaesthesia. Maintenance of adequate ventilation by avoiding hypoxia, hypercarbia and hypocarbia is mandatory. Various combinations of intravenous antihypertensive drugs are available which can be used to provide hemodynamic stability.

Magnesium sulphate infusion was used successfully to control blood pressure in pregnancy with phechromocytoma [10]. Clevidpine is a new drug used before tumour resection . Though Morphine releases histamine, it is better than all other analgesics. Among inhalational anaesthetics Isoflurane and Sevoflurane are preferred. Epidural anaesthesia at T1 level also has been used [1].

Conclusion

Early admission and proper management can avoid per operative problems. Biochemical assessment can be misleading. CT scan can identify most of the adrenal tumours. Better antihypertensive drugs and advanced monitoring have improved safety and surgical outcome in the surgery of pheochromocytma

References:

1. Paramita Trivedi, Parthosarathi Roy, Shuvro Roy Choudhari, Srinivas Narayan: Anaesthetic management of previously non diagnosed pheochromocytoma clinical vigilance- ultimate savior of anaesthesiologist. Indian Journal of Anaesthesiology vol 57: May -June2013:295-297.
2. Stoelting;s Anesthesia and co existing disease: 5th Edition : 427-432
3. Prys Roberts Pheochromoctoma recent progress in its management; Brit.Journ.Anaesth.: 2000: 85 , 44- 57.
4. Clinical Anaesthesiology: Paul G Barash, Bruce F Gullen, Robert K Stoelting. 5th Edition, 2006, 1142-44.
5. A Tisrin, R Sharma, A Kansara, A Giwa, World Journal of Surgery, 2014, 38.: 724-732.
6. Michael S Lord, John GT, Augoustides MD:Journal of Cardiothoracic and vascular anaesthesia: June 2012; Vol. 26:3: 526-531.
7. Staffan Grondal;L.Bendslev, A.Sollevi; B.Hamberger: Adenosine a new Antihypertensive Agent During Pheochromocytoma surgery: World Journal of Surgery. 12,1988:581-585
8. Ronald Miller: Millers Anaesthesia:8th Edition: Vol.1: 1170-1171.
9. Anjum Nas,Sharmila Ghosh, Debdas Saha, Karabai Baral: Catecholamine resistant post resection hypotension in a case of pheochromocytoma, J. Anaesthesia clinical pharmacology, 2010: 26(3): 391-391.
10. Jyostna Krishna HM, Gurudaskini. Case report Magnesium Sulphate as rescue haemodynamic stabilizer during pheochromocytoma excision. Journal of Anaesthesiology Clinical pharmacology: Oct. 2009 Vol 25: 491-492.

Copyright Transfer/ Financial Disclosure/ Conflict of Interest

CONDITIONS FOR SUBMISSION

The articles must represent original research material, should not have been published before, and should not be under consideration of publication elsewhere. This however does not include previous publication in the form of an abstract or as a part of published literature (review or thesis). It is the duty of the author to obtain the necessary permissions for extensive quotations, tables, illustrations, or any other copyrighted material they are using in the paper before a paper can be considered for publication.

COPYRIGHT TRANSFER FORM

Copyright of the article gets transferred to the publishers, once the article has been accepted for publication. The author has to sign the "Copyright Transfer Form" before his/her article is considered for publication. Once the Copyright Transfer statement has been signed by the corresponding author, no change in authorship or in the order of the authors listed in the article would be accepted by the publishers. Also by signing the above mentioned form, the author reassigns the right of co-publishing, or translation, if considered necessary in future, to the publisher. In the advent of occurrence of any dispute, the matter would be resolved within the jurisdiction of Tiruvalla court.

LEGAL OBLIGATIONS

While all care has been taken to provide accurate and correct information in accordance with the date of publication, neither the authors, editors or publisher takes any legal responsibility for any unintentional omission or error. The publisher makes no expressed or implied warranty with respect to the information contained herein. The published material cannot be photocopied for the following purposes: general distribution, promotion, new works or resale. If this is required, specific written permission requires to be obtained from the publisher. Exclusive rights to reproduce and distribute the articles in this journal have been protected by copyright. This also covers the rights to reproduce or distribute the article as well as the translation rights. No material published in this journal can be reproduced in digital format or stored in the form of electronic databases, video disks etc.

1. COPYRIGHT TRANSFER AGREEMENT

I have read the above mentioned details related to copyright of the manuscript submitted and I ....., the author of
.....
.....
.....certify that I willingly assign the copyright of my work/ title.....
to the publisher of Pushpagiri Medical Journal, who will have the exclusive right of producing (in print or digital format) the complete work or any portion of it.

I hereby certify that the work which I am submitting to the publisher is my own and does not contain any matter which no way is infringement of the copyright law.

Name:

Signature: Date signed:

2. FINANCIAL DISCLOSURE

This is to certify that I ....., the author of.....
.....
..... do not have any commercial association or financial interest in the publication of this manuscript.

Name:

Signature:

Date signed:

3. CONFLICT OF INTEREST

This is to certify that I ....., the author of
.....
.....
..... have no conflict of interest regarding the publishing of the manuscript.

Name:

Signature: Date signed:

SUBSCRIPTION INFORMATION

Annual subscription:

Individual: Rs. 1000.00/ 20 USD

Institutional: Rs. 2500.00/ 50 USD

Subscription can be sent to
Dr Santosh Pillai
Associate Editor, Pushpagiri Medical Journal
Pushpagiri Institute of Medical Sciences and Research Centre
Tiruvalla, Kerala, India 689 101
Phone: 0469 2700755 Ext. 550
Mob. 9447596426
pmj@pushpagiri.in

This journal is published twice in a year, i.e. March and September, every year. Dollar rates apply to subscribers in all the countries except India where INR price is applicable. All subscriptions are payable in advance and all the rates include postage. Journals are sent by air to all the countries except Indian subcontinent. Subscriptions are on an annual basis, i.e. from January to December.

For further queries please contact at

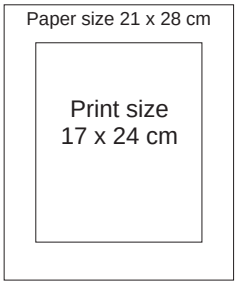
pmj@pushpagiri.in

ADVERTISEMENT RATES

(For the Print Issues)

Page	Single issue
Back cover - colour	Rs. 15,000
Inside back cover - colour	Rs. 10,000
Inside full page - colour	Rs. 7,500

Special position: **Price on request**



Technical Details

Paper size	21 x 28 cm
Print size	17 x 24 cm
Digital file format	Jpg / Psd on Cd (at 300 dpi resolution)

Printed on white paper.

Schedule

Issues are published in the months of January, May and September.
Advertisement material along with purchase order and payments should reach us at least four weeks prior to the scheduled print date.

Payment Details

- Payments should favour Pushpagiri Medical Journal, payable at Tiruvalla.
- Payment to be done at the time of submitting the advertisement material/booking the advertisement. Please send your advertisement request, payment and material to the address given above. Editorial board reserves the right to accept or decline the advertisement.
- For further queries please contact the tusukumaran@gmail.com or drsanthosh74@gmail.com

Title Verification No: KERENG02271/1/1/1/2010 - TC

Pushpagiri Medical Journal

Volume 7, No.1, July - December 2015

ISSN 0976-402X

9 770976 402009

Printed at Furore Designs & Printing Pathanamthitta. Published by Rev Fr Dr Shaji Mathews Vazhayil, CEO, Pushpagiri Group of Institutions, Tiruvalla 689 101 Phone:0469 2603833 / 2700755 (Ext: 401); Email:ceopushpagiri@gmail.com. Chief Editor: Dr T P Thankappan, Principal Pushpagiri Institute of Medical Sciences & Research Centre, Tiruvalla, Pathanamthitta 689 101, Kerala, India. Phone: 0469 2733761 / 2700755(Ext:555) Fax:0469 2600020 Email:pcm@pushpagiri.in, Website: www.pushpagiri.in