Acute coronary syndrome
Cerebellopontine angle tumours
Non-traumatic headache
Implications of palatal height
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Clinical characteristics of Acute coronary syndrome patients under 10 years follow up-A retrospective observational study from a tertiary care centre, Kerala


ABSTRACT

Introduction: Currently, cardiovascular diseases (CVD) are the leading cause of mortality and morbidity globally. The current CVD epidemic needs prompt attention and effectual preventive measures. Among CVD, Acute coronary syndrome (ACS) contributes the maximum towards morbidity and mortality worldwide. There is paucity of data related to clinical characteristics of ACS patients from resource poor settings.

Methodology: This is a retrospective longitudinal observational study conducted in a tertiary care centre. We selected ACS patients who had three to ten years follow up in the same institution. All coronary artery disease (CAD) patients who took treatment from 2005-2015 in the study institution were enlisted with the help of Hospital information system (HIS). From this list, patients with confirmed diagnosis of ACS in the age range of 30-80 years were identified. We used a structured proforma to collect data regarding the clinical characteristics of ACS patients. The study duration was six months. Data were analysed using SPSS version 21.

Results: We enrolled 373 patients in the study. The mean age of the study population was 61.75 (±9.19) years. Majority of the patients (83.9%) were males. Most of the ACS patients were in the age group 60-69 years (39.1%). Among male ACS patients 16.3% were smokers. The common modifiable risk factors identified were dyslipidemia (63.5%), hypertension (62.73%) and diabetes (55.2%). Data for type of ACS was available for 191 patients and among them 48.2% had ST-elevation myocardial infarction (STEMI), 33.5% had Non-ST-elevation myocardial infarction (NSTEMI) and 18.3% had unstable angina. Majority of the patients had single vessel disease (32.4%) followed by double vessel (27.6%) and triple vessel (10.7%) disease. The most common vessel involved was Left anterior descending artery (68.1%). The most frequent management strategy seen was PTCA with medical management (63.8%). Among patients who underwent PTCA, majority (93.6%) were treated with a drug eluting stent.

In the final follow up data 45 patients had secondary ACS. Among those with a recurrence, 51.11% had recurrence within 1 to 5 years.

Conclusion: The most common risk factors identified for ACS were dyslipidemia, hypertension and diabetes. STEMI constituted a considerable proportion of ACS. PTCA with DES was the commonly used treatment strategy. Secondary cardiovascular events were seen in 12% of patients.

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INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death all over the world. It is responsible for 17.3 million deaths which are about 30% of all deaths per year; this figure is expected to grow to 23.6 million deaths per year by 2030 according to the World Health Organization reports1,2.

ACS is a group of conditions including unstable angina and myocardial infarction (MI) with or without an observed ST elevation3. The non-modifiable risk factors for ACS are age, sex, family history, genetic factors and modifiable risk factors are alcohol consumption, smoking, physical inactivity, obesity, hypertension, hyperlipidemia, diabetes mellitus and chronic kidney disease. The recently recognized factors are insulin resistance, impaired fibrinolytic and enhanced procoagulatory activity and an increased inflammatory activity as reflected by an elevated C-reactive protein4. The INTERHEART study revealed that as much as 90% of the risk for an acute MI can be attributed to modifiable risk factors, with the greatest impact of smoking and lipoprotein profile abnormalities followed by abdominal obesity, hypertension, diabetes, psychosocial factors, lack of regular physical activity and dietary habits5.

CVD in India gives a similar picture, where it has led to premature death, disability, and financial crisis because of high expenditure for acute cardiovascular care. The global average of age standardized CVD death rate is 235 per 100 000 population and the same for India is 272 per 100 000 population6,7. Coronary artery disease (CAD) is the foremost cause of disability and death across the world and is one of the top five causes of death in Indian population5,7.

Kerala, with a population of over 33 million, is the most advanced state in epidemiological transition and has the highest prevalence of CAD risk factors in India8,9. Urbanization has resulted in marked increase in the intake of high calorie foods, a sedentary lifestyle, and an increased level of psychosocial stress. This has increased the number of dyslipidemic, hypertensive and dysglycemic individuals which predisposes them to CVD. Approximately 1.5 lakhs people develop myocardial infarction in Kerala every year9,10. Even though
the incidence of ACS is increasing, data regarding clinical profile of ACS patients is limited from our setting. The aim of the current study was to delineate the clinical characteristics of a cohort of ACS patients followed up in a tertiary health centre for a period ranging from three to ten years.

**METHODOLOGY**

This is a hospital based retrospective longitudinal observational study carried out in a tertiary care center (Amrita Institute of Medical Science, Kochi). The list of ACS patients, who took treatment from the study center during the period of January 2012- March 2018, was prepared from the hospital health information system (HIS). Patients in the age range of 30-80 years having confirmed diagnosis of ACS attending the study institution with maximum follow up period of ten years and a minimum follow up period of three years were enrolled for the study. Patients with a concurrent diagnosis of malignancy and those on incomplete follow up were excluded from the study. Patients having confirmed diagnosis of CAD and under follow up during the time period January 2012- March 2018 were consecutively enrolled for the study. The list of patients visiting Cardiology department of the study institution during the period of January 2012- March 2018 from HIS was collected. From this list, the patients having Coronary Artery Disease (CAD) were filtered out. Subsequently patients with confirmed diagnosis of ACS were filtered from the enlisted CAD list. A structured proforma was used for collecting data in the study. The proforma contained demographic details, health related variables, anthropometric measurements, information regarding events of ACS, relevant investigations, treatment and reoccurrence of cardiovascular events. The study duration was for a period of six months (1/3/2018-30/9/2018).

**Statistical analysis**

The data collected was compiled using Microsoft Excel. All statistical analyses were carried out using IBM Statistical Package for Social Science (SPSS version 21). We reported summary statistics for categorical variables using frequency and percentage and continuous variables using mean (SD). We reported the association of the outcome variable with predictor variables such as age (categories), gender and place of residence using Chi-square tests.

**RESULTS**

We recruited a total of 373 patients with documented ACS who satisfied the inclusion criteria. The mean age of the study population was 61.75 (±9.19) years. Among patients, 313 (83.9%) were males. All the women included in the study were post-menopausal. Majority of ACS patients (39.1%) were in the age group of 60-69 years. (Table 1) Among male ACS patients, 51 were smokers (16.3%) and 46 reported consumption of alcohol (14.7%). The other significant risk factors prevalent in the group were dyslipidemia (63.5%), hypertension (62.75%) and diabetes (55.2%).

In the enlisted ACS patients, specific details of the primary events were available only for 191 (39.1%) patients. Among those with this information, 92 (48.2%) had STEMI, 64 (33.5%) had NSTEMI and 35 (18.3%) had unstable angina.

Among ACS patients, 172 (46.1%) patients did ECG and Troponin I/T level investigations, 167 (44.8%) had undergone ECG, Troponin levels, CPK or CK-MB and 167 (44.8%) had done ECG, Troponin levels, CPK/CK-MB and ECHO.

A total of 121 (32.4%) patients had single vessel disease, 109 (29.2%) had double vessel disease, 106 (27.6%) had triple vessel disease and 40 (10.7%) of them had mild vessel disease.

The most common vessel involved was Left anterior descending artery (254 patients, 68.1%) followed by Right coronary artery, (161 patients, 43.2%) and left circumflex artery, (102 patients, 27.3%).

<table>
<thead>
<tr>
<th>Age Categories</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-39</td>
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<td>0.5</td>
</tr>
<tr>
<td>40-49</td>
<td>38</td>
<td>10.2</td>
</tr>
<tr>
<td>50-59</td>
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<td>29.2</td>
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<tr>
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<td>39.1</td>
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<td>70-79</td>
<td>72</td>
<td>19.3</td>
</tr>
<tr>
<td>80-89</td>
<td>6</td>
<td>1.6</td>
</tr>
</tbody>
</table>

Table 1: Age distribution of ACS patients (n=373)

Fig 1: Distribution of coronary arteries involved in ACS (n=373)
The most common management strategy seen was PTCA with medical management (233 patients, 63.8%). CABG was done in 46 (12.3%) patients. A total of 10 patients (2.7%) underwent both PTCA and CABG. Among patients, 79 (21.2%) were under medical management alone. Among patients who had undergone PTCA, majority (218 patients, 93.6%) had received drug eluting stents. Among patients with PTCA, 10 (4.3%) had received bare metal and 3 (1.3%) had bioresorbable stents. Both DES and bare metal were received by 2 (0.9%) patients.

According to the data available secondary ACS occurred in 45 patients (12.06%). Among patients with secondary ACS events, 8 (17.78%) had reoccurrence within the same year, 23 (51.11%) patients had within 1 to 5 years and 14 (31.11%) patients within 6 to 10 years. In this group, four patients (12.1%) had STEMI, 16 patients (48.5%) had NSTEMI and 13 (39.4%) patients had unstable angina.

<table>
<thead>
<tr>
<th>Treatment strategies</th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical alone</td>
<td>79</td>
<td>21.2</td>
</tr>
<tr>
<td>PTCA</td>
<td>238</td>
<td>63.8</td>
</tr>
<tr>
<td>CABG</td>
<td>46</td>
<td>12.3</td>
</tr>
<tr>
<td>PTCA+CABG</td>
<td>10</td>
<td>2.7</td>
</tr>
</tbody>
</table>

Table 2: Treatment taken (n=373)

DISCUSSION

India is having high burden of risk factors for CAD such as hypertension, dyslipidemia, diabetes, obesity, smoking and alcoholism. Mortality due to cardiovascular disorders constitutes nearly 53% of all deaths in the country. The age of presentation of CAD in India is much less compared to other developed countries.

The mean age of the study population was 61.75 (±9.19) years. Among patients, 313 (83.9%) were males. All the women included in the study were post-menopausal. Majority of ACS patients (39.1%) were in the age group of 60-69 years. Among male ACS patients, 51 were smokers (16.3%) and 46 reported consumption of alcohol (14.7%). The other significant risk factors prevalent in the group were dyslipidemia (63.5%), hypertension (62.75%) and diabetes (55.2%). Most of the patients had STEMI, n= 92 (48.2%) and single vessel disease, n=121 (62.7%). The most common vessel involved was Left anterior descending artery, n=254 (68.1%). The most common management strategy seen was PTCA with medical management, n=233 patients (63.8%). Secondary ACS occurred in 45 patients (12.06%) in the study population over a period of 3-10 years.

The current study had 83.9% male patients. According to the study done by Abu-Assi et al 28.7% were women. The age stratified occurrence of ACS in our study were 10.7% of patients belonged to <50 years, 68.3% in 50-70 years and 20.9% in >70 years category. Other studies have shown similar results.

The risk factors prevalent in the group were dyslipidemia (63.5%), hypertension (62.75%) and diabetes (55.2%). The CREATE registry data had 40.2% hypertensives and 30.4% diabetics. According to the study by Mohanan et al, 48.4% of patients had hypertension and 37.6% patients had Diabetes mellitus. The study by Abu-Assi et al had 56% patients with Hypertension, 26.8% patients with Diabetes. Compared to the previous studies our study population had higher prevalence of risk factors such as hypertension, diabetes and dyslipidemia.

About 50% of the patients that we assessed had STEMI, less than 35% had NSTEMI and the least primary event recorded was unstable angina. The study conducted by Mohanan et al had patients with 37% STEMI, 31% patients with NSTEMI and 32% patients with unstable angina. While in Abu-Assi et al study showed 31.4% patients with STEMI, 49.7% patients with NSTEMI and 18.9% patients with unstable angina. In developed nations less than 40% had STEMI.

Almost half of the patients in our study did ECG and estimated Troponin I/T levels. Less than half the patients, i.e., 44.7% did all investigations needed to diagnose ACS such as ECG, Troponin levels, CPK or CK-MB and ECHO. Previous studies have shown that 57.7% - 73.5% of patients had positive cardiac enzyme investigations and 19.5% - 23.2% did coronary angiography.

In the present study, 10.7% of the ACS patients had mild disease, 32.4% suffered from single vessel disease, 29.2% of the patients had double vessel disease and 27.6% had triple vessel disease. The most common vessel involved was Left anterior descending artery (68.1%). Sharma et al, Tewari et al and Kumar et al had similar results.

PTCA is the most common treatment received by patients. Almost two-third of them (66.5%) had received PTCA followed by medical management alone (21.2%). Patients undergone CABG were 12.3% and that of both PTCA and CABG were 2.7%. According to Mohanan et al, 11.9% of patients performed PTCA and 1.4% did CABG. According to Abu-Assi and et al, CABG was done by 4.5%. Majority of the patients received Drug eluting stents (93.6%) followed by bare metal stents (4.3%) and bioresorbable stents (1.3%).

In the study population of 373 ACS patients, 45 (12.06%) had secondary events during the follow up period of 10 years. In that group, 8 (17.78%) had reoccurrence within one year, 23 (51.11%) patients had within 1 to 5 years and 14 (31.11%) patients within 6 to 10 years. In this group, four patients (12.1%) had STEMI, 16 patients (48.5%) had unstable angina.
NSTEMI and 13(39.4%) patients had unstable angina. In Abu-Assi et al study\textsuperscript{19}, 45% ACS patients had composite outcome of secondary events such as acute myocardial infarction, strokes or cardiovascular deaths\textsuperscript{10}.

**CONCLUSION**

The occurrence of ACS in most of our patients was seen in the sixth decade of life. The common risk factors identified for ACS were dyslipidemia, hypertension and diabetes. STEMI constituted the major proportion of ACS in our study population. PTCA with DES was the commonly used treatment strategy. Lifestyle modifications and cardiac rehabilitation are needed to decrease the morbidity and mortality associated with ACS.

**Conflicts of Interest**

None declared

**REFERENCES**


Distortion Product Oto Acoustic Emission (DPOAE) findings in patients with Cerebellopontine angle tumors - Effect of tumor type, size and extent

Prem Govindan Nair*, Nagarajrao ShivaShankar**, Bhagavatula Indira Devi**, S.G. Srikanth***, V.Shanmugham****, Aparna Prasanna*

ABSTRACT

Objectives: Current study focusses on the pathophysiological basis of the CPA tumors by analysing Distortion Product OAE (DPOAE) and correlated it with the type, size, and extent of tumors.

Method: The study was undertaken at National Institute of Mental Health and Neuro Sciences (NIMHANS). The clinical group comprised of 98 patients with CPA tumors aged between 15-55 years who were grouped based on tumor type, size and extent. Matched to the age range of the clinical group, the control group comprised of 100 normal hearing subjects. OAEs and corresponding noise floor were measured and their difference in amplitude was estimated. Its correlation with the type, size, and extent of tumors was analysed statistically.

Results: Results suggested that DPOAE in the tumor as well as the nontumor ears were abnormal which implicated the vulnerability of peripheral pathway (both tumor and nontumor side) to the pathological effects of the tumor. Greater deficits were noted in the tumor ears rather than the nontumor ears. DPOAE were significantly poorer in the acoustic tumor ears than the nonacoustic tumor ears which indicated the preponderance of acoustic tumors to cause cochlear dysfunction in the tumor ears. Poor DPOAE in the acoustic nontumor ears when compared with the nonacoustic nontumor ears implied that acoustic tumor type causes a higher outer hair cell (OHC) dysfunction in the nontumor ears compared to nonacoustic type.

Conclusion: Thus, the predominance of acoustic type to cause cochlear dysfunction in the nontumor ears is higher. DPOAE in the tumor ears and non tumor ears did not demonstrate any significant difference with respect to tumor size and presence/absence of the intracanalicular component.

KEYWORDS: DPOAE, Cerebellopontine angle tumor, tumor size, tumor type, tumor extent.

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INTRODUCTION

The Cerebellopontine Angle (CPA) can be considered as a space bounded by the cerebellum, pons and petrous part of the temporal bone. The CPA cistern houses some of the vascular structures that are vital for the auditory system. Some of them are the Anterior Inferior Cerebellar Artery (AICA) and its branches and the Posterior Inferior Cerebellar Artery (PICA)1. The Internal Auditory Artery (IAA), a branch of the AICA, branches within the IAM and they supply blood to the cochlea and vestibular end organs. Given this neuroanatomy of the CPA region, it can be expected that lesions of the CPA is potent to cause significant auditory deficits.

The CPA is one of the most common sites of intracranial tumors and approximately 10% of them originate in the CPA2. The CPA tumors are persuasive to cause direct and/or indirect pathological effects on the auditory system. These effects generally are in the form of vascular compression or occlusion of the blood supply to the eighth nerve or to the cochlea, biochemical changes within the inner ear and toxicity of inner ear3. Pressure on adjacent structures including compression or displacement of brainstem may also contribute to auditory dysfunction.

The cochlear dysfunction has been theorized to arise from any one or a combination of the following pathophysiological mechanisms: vascular compromise, hypdrops, degradation of inner ear fluids/tumor toxicity, deafferentation and loss of efferent nerve tuning4. With the introduction of Otoacoustic Emission (OAE) testing, it is now possible to assess the cochlear status, in particular the Outer Hair Cell (OHC) dysfunction.

The pathophysiology of hearing in CPA tumors may depend upon tumor characteristics such as type, size and extent and these aspects could influence the audiological test results as well. The preponderance of acoustic tumors to cause cochlear dysfunction have been reported by studies of Telischi, Roth, Stagner et al4, Prasher, Tun, Brookes et al5, Oeken6, Ferber-Viart, Colleaux, Laoust et al7, Telischi7 and Odabasi, Telischi, Gomez-Marin, et al8. Further, Telischi et al9 and Ferber-Viart, Colleaux, Laoust et al9 reported no clear relationship of tumor size to OAE levels. Mobley, Odabasi, Ahsan et al9 have suggested that the extent of IAM involvement by VS was not significantly related to the negative effects of the tumor on cochlear function. Hence the current study was designed to focus on the pathophysiological basis of the CPA tumors by analysing Distortion Product OAE (DPOAE). The present study had two objectives:

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1) To characterize the DPOAE profiles in CPA tumors,
2) To correlate DPOAE test results with the type, size, and extent of tumors.

METHODS

The study was undertaken at National Institute of Mental Health and Neuro Sciences (NIMHANS), an apex tertiary public sector hospital in the city of Bangalore, India. The NIMHANS ethics committee had approved the protocol for the study. Department of neurosurgery was the referral source for the study population.

Clinical group

This group comprised of 98 patients with CPA tumors. The inclusion criteria are:
 a) Confirmed CPA tumor patients as evidenced by neuroradiological findings on Magnetic Resonance Imaging or Computed Tomography.
b) Patients aged between 15-55 years.
c) Patients giving signed informed consent. Patients with the following history or coexisting conditions were excluded from the study:- Continuous noise exposure for prolonged periods, drug ototoxicity, chronic middle ear infections, Diabetes Mellitus, Meniere’s disease, Multiple Sclerosis, Myasthenia gravis, head or ear injury, Auditory Neuropathy, syphilis, any viral infections affecting the auditory system and hereditary familial sensorineural hearing loss.

Tumors were classified into Group I having tumor volume less than or equal to 30 cc, Group II having tumor volume 31-60 cc, Group III having volume greater than 60 cc and Group IV where tumor volume could not be ascertained. Based on the extent of tumor the patients were classified into Group A having internal auditory meatus (IAM) and brainstem involvement, Group B having only brainstem involvement and Group C-a mixed group having patients with the lesion extension to IAM only, patients with no involvement of IAM and brainstem and patients in whom the tumor extent could not be estimated (Table I). Patients of Group IV (volume) and Group C (lesion extent) were not included for statistical analysis due to its limited number.

Control group

This group comprised of 100 normal hearing subjects. The control group comprised of patients’ relatives as well as staff and students of the hospital and was selected based on the following criteria:- Subjects aged between 15-55 years, Having normal hearing, i.e., puretone thresholds 25 decibel (dB) or below at octave frequencies from 250-8000 Hertz (Hz) and including 6 kHz, and subjects giving signed informed consent. The exclusion criteria being set was impaired hearing, i.e., puretone threshold more than 25 dB at any of the octave frequencies from 250-8000 Hz and including 6 kHz, History of occupational noise exposure, ear infections or any other neurological/psychiatric conditions.

DPOAE

The DPOAE evaluations were carried out using an Oto-acoustic Emission Analyser DP Echoport plus TE+DPOAE Screener/Analyser (Otodynamics Ltd. ILO292, V2.0). The testing was carried out in a quiet room. Non-essential equipments, computers, fans and air conditioner were turned off during evaluation. The subjects were given clear instructions to remain still and quiet during testing. A comfortable, secure probe fit was assured before evaluation. The time consumed for carrying out OAE testing for each subject ranged between 15-30 minutes.

The DPOAEs were measured by presenting two puretone stimuli (f1 and f2) simultaneously to the ear. The f2/f1 ratio was maintained at 1.2. The intensity of both stimuli was kept at moderate levels, i.e., stimulus settings of 65 dB Sound Pressure Level (SPL) (L1) and 60 dB SPL (L2) for f1 and f2 respectively. Two points per octave DPOAE resolution was utilized to obtain the Distortion Product (DP)-gram. Two thousand stimulus repetitions were recorded. Even though due to intermodulation distortion responses occur from different frequency regions, emissions from 2f1-f2 are believed to be the largest and are commonly utilized in OAE measurements. In the present study, OAEs and corresponding noise floor were measured at 1 kilo Hertz (kHz), 1.5 kHz, 2 kHz, 3 kHz, 4 kHz and 6 kHz. At each of these frequencies, the difference in amplitude between OAEs and noise floor
were estimated.

Statistical treatment of the data

Statistical comparison of the audiological data were made with respect to patient versus control group, tumor versus nontumor ear, acoustic versus nonacoustic tumor types, Group I versus Group II versus Group III tumor volumes and Group A versus Group B classified based on lesion extent. Statistical tests such as independent samples t-test, analysis of covariance (ANCOVA), analysis of variance (ANOVA) were employed. Statistical comparison of mean values of clinical group as to evaluate differences between tumor types, tumor volumes and tumor extent. Independent samples t-test was primarily employed to assess the differences between tumor and nontumor ears.

RESULTS

Demographic data

The clinical group consisted of 98 patients of radiologically confirmed CPA tumors. There were 39 males and 59 females with a combined mean age of 37.4 years. Matched to the age range of the clinical group, the control group (normal hearing) was comprised of 100 subjects, 59 males and 41 females, with a combined mean age of 33.6 years.

<table>
<thead>
<tr>
<th>Variables</th>
<th>No: of ears</th>
<th>Mean (SD) of DPOAE/noise in dB at 1 kHz</th>
<th>Mean(SD) of DPOAE/noise in dB at 1.5 kHz</th>
<th>Mean (SD) of DPOAE/noise in dB at 2 kHz</th>
<th>Mean (SD) of DPOAE/noise in dB at 3 kHz</th>
<th>Mean (SD) of DPOAE/noise in dB at 4 kHz</th>
<th>Mean (SD) of DPOAE/noise in dB at 6 kHz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group</td>
<td>200</td>
<td>5.49 (6.02)</td>
<td>10.24 (7.38)</td>
<td>10.53 (6.9)</td>
<td>10.4 (7.79)</td>
<td>9.9 (7.7)</td>
<td>4.87 (8.8)</td>
</tr>
<tr>
<td>Tumor ear</td>
<td>98</td>
<td>-2 (7.62)</td>
<td>0.01 (11.43)</td>
<td>-1.15 (10.61)</td>
<td>-0.87 (10.8)</td>
<td>0.54 (11.24)</td>
<td>-3.71 (9.7)</td>
</tr>
<tr>
<td>Nontumor ear</td>
<td>98</td>
<td>2.71 (7.22)</td>
<td>9.12 (11.13)</td>
<td>8.75 (10.62)</td>
<td>8.1 (12.03)</td>
<td>7.06 (11.35)</td>
<td>3.26 (10.45)</td>
</tr>
<tr>
<td>Acoustic tumor</td>
<td>61</td>
<td>-3.34 (7.19)</td>
<td>-3.46 (9.83)</td>
<td>-4.86 (8.71)</td>
<td>-4.68 (9.33)</td>
<td>-3.13 (9.44)</td>
<td>-6.15 (8.22)</td>
</tr>
<tr>
<td>Nonacoustic tumor ear</td>
<td>37</td>
<td>0.21 (7.89)</td>
<td>5.73 (11.71)</td>
<td>4.96 (10.72)</td>
<td>5.41 (10.19)</td>
<td>6.58 (11.48)</td>
<td>0.33 (10.67)</td>
</tr>
<tr>
<td>Acoustic nontumor ear</td>
<td>61</td>
<td>2.31 (7.67)</td>
<td>7.62 (12.19)</td>
<td>6.98 (11.67)</td>
<td>5.96 (11.92)</td>
<td>4.9 (11.43)</td>
<td>2.37 (10.58)</td>
</tr>
<tr>
<td>Non acoustic nontumor ear</td>
<td>37</td>
<td>3.39 (6.47)</td>
<td>11.61 (8.71)</td>
<td>11.67 (7.91)</td>
<td>11.64 (11.52)</td>
<td>10.63 (10.4)</td>
<td>4.74 (10.19)</td>
</tr>
<tr>
<td>Group I tumor ear</td>
<td>23</td>
<td>-3.1 (7.08)</td>
<td>-1.92 (9.86)</td>
<td>-1.27 (6.71)</td>
<td>-3.48 (11.26)</td>
<td>-0.63 (10.52)</td>
<td>-2.87 (7.78)</td>
</tr>
<tr>
<td>Group II tumor ear</td>
<td>40</td>
<td>-2.75 (7.09)</td>
<td>-2.29 (9.7)</td>
<td>-4.7 (10.18)</td>
<td>-3.91 (9.54)</td>
<td>-2.51 (9.15)</td>
<td>-6.24 (8.14)</td>
</tr>
<tr>
<td>Group III tumor ear</td>
<td>32</td>
<td>0.14 (8.57)</td>
<td>4.54 (13.2)</td>
<td>3.38 (11.76)</td>
<td>4.16 (10.36)</td>
<td>4.21 (12.44)</td>
<td>-1.45 (12.18)</td>
</tr>
<tr>
<td>Group I nontumor ear</td>
<td>23</td>
<td>3.57 (7.16)</td>
<td>10.52 (9.5)</td>
<td>9.15 (9.16)</td>
<td>6.53 (11.77)</td>
<td>4.34 (9.74)</td>
<td>1.82 (8.75)</td>
</tr>
<tr>
<td>Group II nontumor ear</td>
<td>40</td>
<td>0.49 (7.05)</td>
<td>5.91 (10.63)</td>
<td>5.51 (10.97)</td>
<td>6.98 (11.98)</td>
<td>6.22 (12.51)</td>
<td>1.96 (11.33)</td>
</tr>
<tr>
<td>Group III nontumor ear</td>
<td>32</td>
<td>4.87 (6.9)</td>
<td>12.53 (12.08)</td>
<td>12.76 (9.97)</td>
<td>10.57 (12.43)</td>
<td>9.81 (10.6)</td>
<td>6.17 (10.4)</td>
</tr>
<tr>
<td>Group A tumor ear</td>
<td>57</td>
<td>-2.41 (7.6)</td>
<td>-0.34 (10.12)</td>
<td>-2.39 (10.22)</td>
<td>-1 (10.83)</td>
<td>1.16 (10.78)</td>
<td>-4.89 (9.5)</td>
</tr>
<tr>
<td>Group B tumor ear</td>
<td>28</td>
<td>-0.48 (8.3)</td>
<td>2.25 (14.8)</td>
<td>2.55 (11.38)</td>
<td>1.28 (11.44)</td>
<td>2.32 (12.61)</td>
<td>-0.78 (10.85)</td>
</tr>
</tbody>
</table>

Table II: Mean DPOAE/noise values in the control group and each of the clinical group

and the control group was carried out using ANCOVA by considering age and gender as covariates. ANCOVA followed by post hoc (Tukey) test was employed to compare the control group with discrete clinical group classified based on tumor type, size and extent as well as 59 males and 41 females, with a combined mean age of 33.6 years.

DPOAE

The mean difference in the control group between the right and left ears for DPOAE/noise difference was not
statistically significant on independent samples t-test and hence a combined mean of DPOAE/noise values were computed. They were 5.49 dB (SD=6.02), 10.24 dB (SD=7.38), 10.53 dB (SD=6.9), 10.4 dB (SD=7.79), 9.9 dB (SD=8.8) at 1, 1.5, 2, 3, 4 and 6 kHz respectively.

In the clinical group the test could be administered in all. Table II depicts the mean DPOAE/noise values in both control and the patient group. To analyse the

<table>
<thead>
<tr>
<th>Variables</th>
<th>p value for DPOAE/noise at 1 kHz</th>
<th>p value for DPOAE/noise at 1.5 kHz</th>
<th>p value for DPOAE/noise at 2 kHz</th>
<th>p value for DPOAE/noise at 3 kHz</th>
<th>p value for DPOAE/noise at 4 kHz</th>
<th>p value for DPOAE/noise at 6 kHz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group versus tumor ear</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
</tr>
<tr>
<td>Control group versus nontumor ear</td>
<td>0.023*</td>
<td>0.985</td>
<td>0.634</td>
<td>0.614</td>
<td>0.234</td>
<td>0.914</td>
</tr>
<tr>
<td>Control group versus acoustic tumor ear</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
</tr>
<tr>
<td>Control group versus nonacoustic tumor ear</td>
<td>0.001*</td>
<td>0.029*</td>
<td>0.002*</td>
<td>0.01*</td>
<td>0.139</td>
<td>0.026*</td>
</tr>
<tr>
<td>Control group versus acoustic nontumor ear</td>
<td>0.01*</td>
<td>0.198</td>
<td>0.037*</td>
<td>0.018*</td>
<td>0.004*</td>
<td>0.248</td>
</tr>
<tr>
<td>Control group versus nonacoustic nontumor ear</td>
<td>0.23</td>
<td>0.727</td>
<td>0.78</td>
<td>0.796</td>
<td>0.916</td>
<td>0.997</td>
</tr>
<tr>
<td>Control group versus Group I tumor ear</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.002*</td>
</tr>
<tr>
<td>Control group versus Group II tumor ear</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
</tr>
<tr>
<td>Control group versus Group III tumor ear</td>
<td>0.001*</td>
<td>0.016*</td>
<td>0.001*</td>
<td>0.005*</td>
<td>0.015*</td>
<td>0.005*</td>
</tr>
<tr>
<td>Control group versus Group I nontumor ear</td>
<td>0.584</td>
<td>0.999</td>
<td>0.901</td>
<td>0.348</td>
<td>0.061</td>
<td>0.519</td>
</tr>
<tr>
<td>Control group versus Group II nontumor ear</td>
<td>0.001*</td>
<td>0.063</td>
<td>0.012*</td>
<td>0.269</td>
<td>0.172</td>
<td>0.372</td>
</tr>
<tr>
<td>Control group versus Group III nontumor ear</td>
<td>0.966</td>
<td>0.614</td>
<td>0.586</td>
<td>1.000</td>
<td>1.000</td>
<td>0.911</td>
</tr>
<tr>
<td>Control group versus Group A tumor ear</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
</tr>
<tr>
<td>Control group versus Group B tumor ear</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.014*</td>
</tr>
</tbody>
</table>

Table III Statistical significance on comparison of mean DPOAE/noise values between control group and each of the clinical group

difference in mean DPOAE/noise values between the control group and the patient group (T-tumor ear and NT-nontumor ear) ANCOVA was employed and significant difference at p<0.05 for DPOAE/noise values were observed for tumor ears at all frequencies. In the nontumor ears, statistically significant difference (p<0.05) with the control group for DPOAE/noise values was observed only at 1 kHz. To analyse the difference in mean DPOAE/noise values between the control group and discrete clinical groups classified based on tumor type, size and extent ANOVA was used and found sig-
values between the control group and discrete clinical
groups classified based on tumor type, size and extent
and in the tumor side, significant difference at p<0.05
for DPOAE/noise values were observed for acoustic tu-
mor ears, Group I, II and III tumor ears as well as Group A
and B tumor ears at all frequencies. Nonacoustic tumor
ears also revealed significant difference at all frequen-
cies except 4 kHz (p=0.14). In the nontumor side, statisti-
cally significant difference at p<0.05 with the control
group for DPOAE/noise values was observed at 1, 2, 3
and 4 kHz for acoustic nontumor ears and at 1 and 2
kHz for Group II nontumor ears. Nonacoustic nontumor
ears did not show any statistically significant difference
when compared with the control group for DPOAE/
noise values (Table III).

**Comparison of DPOAE/noise values between tumor and nontumor ears**

Tumor and nontumor ear comparison revealed highly
significant difference (p<0.001) at all frequencies, with
poor scores in tumor ears. Similar findings were noted
when acoustic tumor ears and Group A tumor ears were
compared with their corresponding nontumor ears. In
the nonacoustic group, tumor ear demonstrated poor
DPOAE/noise values than nontumor ears at 1.5, 2 and
3 kHz at p<0.05. With respect to tumor size, statistically
significant difference at p<0.05 was noted between
tumor and nontumor ears (poor scores in tumor ears),
except at 4 and 6 kHz (p>0.05) in Group I and 4 kHz
(p=0.06) in Group III. On comparing tumor and nontu-
mor ears in Group B, DPOAE/noise values were not sta-

tistically significant (p>0.05) except at 2 and 3 kHz (Table

<table>
<thead>
<tr>
<th>Variables</th>
<th>p value for DPOAE/noise at 1 kHz</th>
<th>p value for DPOAE/noise at 1.5 kHz</th>
<th>p value for DPOAE/noise at 2 kHz</th>
<th>p value for DPOAE/noise at 3 kHz</th>
<th>p value for DPOAE/noise at 4 kHz</th>
<th>p value for DPOAE/noise at 6 kHz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tumor ear versus Nontumor ear</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
</tr>
</tbody>
</table>
| Acoustic tumor ear versus Acoustic nontu-
  mor ear                                 | 0.001*                           | 0.001*                            | 0.001*                           | 0.001*                           | 0.001*                           | 0.001*                           |
| Nonacoustic tumor ear versus Nonacoustic
  nontumor ear                            | 0.063                            | 0.017*                            | 0.003*                           | 0.016*                           | 0.116                            | 0.074                            |
| Group I tumor ear versus Group I nontu-
  mor ear                                 | 0.003*                           | 0.001*                            | 0.001*                           | 0.005*                           | 0.104                            | 0.061                            |
| Group II tumor ear versus Group II nontu-
  mor ear                                 | 0.043*                           | 0.001*                            | 0.001*                           | 0.001*                           | 0.001*                           | 0.001*                           |
| Group III tumor ear versus Group III nontu-
  mor ear                                 | 0.018*                           | 0.014*                            | 0.001*                           | 0.029*                           | 0.057                            | 0.009*                           |
| Group A tumor ear versus Group A nontu-
  mor ear                                 | 0.001*                           | 0.001*                            | 0.001*                           | 0.001*                           | 0.001*                           | 0.001*                           |
| Group B tumor ear versus Group B nontu-
  mor ear                                 | 0.062                            | 0.055                             | 0.045*                           | 0.029*                           | 0.066                            | 0.094                            |

Table IV: Statistical significance on comparison of mean DPOAE/noise values between tumor and nontumor ears

Note: The mean (SD) of clinical groups are shown in Table VI. *statistical significance at p<0.05.
crete clinical groups classified based on tumor type, size and extent were compared pair wise using post hoc (Tukey) test. Between acoustic and nonacoustic tumor ears, the DPOAE/noise value difference was statistically significant (p<0.05) with acoustic group demonstrating poorer scores at all frequencies. Similarly, acoustic nontumor ears demonstrated poorer DPOAE/noise values than nonacoustic nontumor ears.

Similar findings were noted when Group I and III tumor ears were compared, except for significantly poorer (p=0.012) DPOAE/noise value for Group I at 3 kHz. Group II tumor ears demonstrated significantly poorer (p<0.05) DPOAE/noise values than Group III tumor ears at 1.5, 2, 3 and 4 kHz. Similarly, Group II nontumor ears demonstrated significantly poor (p<0.05) DPOAE/noise values than Group III nontumor ears at 1, 1.5 and 2 kHz.

<table>
<thead>
<tr>
<th>Variables</th>
<th>p value for DPOAE/noise at 1 kHz</th>
<th>p value for DPOAE/noise at 1.5 kHz</th>
<th>p value for DPOAE/noise at 2 kHz</th>
<th>p value for DPOAE/noise at 3 kHz</th>
<th>p value for DPOAE/noise at 4 kHz</th>
<th>p value for DPOAE/noise at 6 kHz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acoustic tumor ear versus Nonacoustic tumor ear</td>
<td>0.034*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.002*</td>
</tr>
<tr>
<td>Acoustic nontumor ear versus Nonacoustic nontumor ear</td>
<td>0.716</td>
<td>0.103</td>
<td>0.031*</td>
<td>0.018*</td>
<td>0.012*</td>
<td>0.467</td>
</tr>
<tr>
<td>Group I tumor ear versus Group II tumor ear</td>
<td>0.998</td>
<td>0.999</td>
<td>0.423</td>
<td>0.998</td>
<td>0.866</td>
<td>0.503</td>
</tr>
<tr>
<td>Group I tumor ear versus Group III tumor ear</td>
<td>0.311</td>
<td>0.058</td>
<td>0.198</td>
<td>0.012*</td>
<td>0.226</td>
<td>0.942</td>
</tr>
<tr>
<td>Group II tumor ear versus Group III tumor ear</td>
<td>0.284</td>
<td>0.012*</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.013*</td>
<td>0.129</td>
</tr>
<tr>
<td>Group I nontumor ear versus Group II nontumor ear</td>
<td>0.274</td>
<td>0.228</td>
<td>0.38</td>
<td>0.998</td>
<td>0.876</td>
<td>1.000</td>
</tr>
<tr>
<td>Group I nontumor ear versus Group III nontumor ear</td>
<td>0.886</td>
<td>0.857</td>
<td>0.425</td>
<td>0.461</td>
<td>0.16</td>
<td>0.352</td>
</tr>
<tr>
<td>Group II nontumor ear versus Group III nontumor ear</td>
<td>0.026*</td>
<td>0.015*</td>
<td>0.003*</td>
<td>0.438</td>
<td>0.392</td>
<td>0.257</td>
</tr>
<tr>
<td>Group A tumor ear versus Group B tumor ear</td>
<td>0.448</td>
<td>0.472</td>
<td>0.042*</td>
<td>0.546</td>
<td>0.861</td>
<td>0.139</td>
</tr>
</tbody>
</table>

Table V: Statistical significance on comparison of mean DPOAE/noise values between clinical groups classified based on tumor type, size and extent

Note: The mean (SD) of clinical groups are shown in Table VII. *statistical significance at p<0.05.

Comparison between Group A and B tumor ears did not reveal any significant difference for DPOAE/noise values except at 2 kHz (p=0.042), where Group A had poor scores (Table V).
DISCUSSION

In the present study, insult to the cochlear apparatus was clearly evident as the tumor ear DPOAE values at all the test frequencies (1, 1.5, 2, 3, 4 and 6 kHz) were significantly poorer than the control group. The acoustic tumor ears displayed similar results. The nonacoustic tumor ears demonstrated significantly poor DPOAE values than the control group at all frequencies except 4 kHz. Patient population subcategorized based on tumor size and extent also demonstrated the tumor ear DPOAE abnormality at all frequencies. The pathophysiology of cochlear dysfunction in CPA tumors could be attributed mainly to disruption of blood flow, degradation of inner ear fluids/tumor toxicity, endolymphatic hydrops, deafferentation and loss of efferent nerve tuning. The preponderance of cochlear dysfunction in CPA tumor ears, particularly acoustic type, have been documented by Telischi et al, Oeken, Telischi and Odabasi et al. The proportion of the tumor ears with cochlear disruption as reported by these authors was 59%, 54%, 57% and 60.5% respectively. The nontumor ears did not demonstrate statistically significant difference with the control group at majority of the test frequencies. Similar results were noted when the nontumor ears were subcategorized based on the tumor size. Thus, in general, the nontumor ear cochlea seems to be unaffected by the pathological effects of the CPA tumor.

The pathophysiology associated with the tumor ear cochlea was greater than those of the nontumor ear, as DPOAE values were significantly poorer in the tumor ears at all frequencies. When patient population was subcategorized based on tumor size, similar findings were noted at majority of the test frequencies. Telischi et al also reported similar findings. In their study, for the cochlear group, DPOAEs for the tumor ears were considerably lower in amplitude than were those recorded in the corresponding nontumor ears. DPOAE levels for the noncochlear group tended to be similar for the tumor and the nontumor ears but there was a tendency for emissions in the tumor ear to be at a slightly lower level.

When tumor type was taken into account, some interesting findings emerged. The acoustic nontumor ears demonstrated abnormal DPOAE values at 1, 2, 3 and 4 kHz but the nonacoustic nontumor ears did not differ with the control group. Thus nonacoustic tumors seem to exert less pathological influence on the nontumor ear cochlea. For the tumor and the nontumor ear comparison, the acoustic tumor ears demonstrated significantly poor DPOAE values at all frequencies. However, the nonacoustic tumor ears differed with the nontumor ears only at 1.5, 2 and 3 kHz. The predominant cochlear dysfunction associated with the acoustic tumors was evident when comparison was made with the nonacoustic tumors. In the tumor ears, acoustic group demonstrated poorer DPOAE values than the nonacoustic group at all frequencies. Similarly in the nontumor side, poorer DPOAE values in the acoustic nontumor ears than the nonacoustic nontumor ears were evident at 2, 3 and 4 kHz. Mobley et al compared the frequencies of cochlear and noncochlear patterns of DPOAE in patients with nonacoustic tumors of the CPA with those in patients with VS. In their study, in the nonacoustic group, 42% patients had a cochlear pattern and 58% had a noncochlear pattern. In the acoustic group, 58% patients had a cochlear pattern and 42% had a noncochlear pattern. The differences between the nonacoustic and the acoustic groups were statistically significant. They concluded that the acoustic tumors appeared to differ from nonacoustic tumors of the CPA in their propensity to cause sensory versus neural hearing loss.

Analysis to unravel the effect of tumor size on DPOAE revealed interesting findings in the current study. Group III tumor ears had significantly better DPOAE values than Group II at 1.5, 2, 3 and 4 kHz. Similarly, Group III nontumor ears demonstrated better DPOAE values than Group II at 1, 1.5 and 2 kHz. These findings could probably be attributed to the confounding effects of tumor type as Group II had greater proportion of acoustic tumors (73%) when compared to Group III (47%) and the prevalence of cochlear dysfunction in acoustic tumors is already evident in the present study. Similarly, Telischi et al have also reported about ambiguous relationship between tumor size and DPOAE findings. In their study group, the average tumor size in the mixed grouping (2.6 cm) was significantly larger than the average tumor size in either the cochlear subjects (1.76 cm) or the noncochlear subjects (1.48 cm) and no clear relation emerged between tumor size and DPOAE. However, possible role of tumor size in influencing DPOAEs of nonacoustic CPA tumors has been documented by Mobley et al. They noted that in the nonacoustic group, patients with a cochlear pattern DPOAE had significantly smaller tumors compared with the ears with the noncochlear pattern. However, the authors could not find any statistically significant difference between the cochlear and the noncochlear types with respect to tumor size in the acoustic group.

Intracanalicular component of tumor did not seem to influence cochlear function since Group A and B tumor ears did not differ in terms of DPOAE values. Similar findings were reported by Odabasi et al. In their study group, 67% of tumors had full extension into the IAM and 33% had partial IAM involvement. They noted cochlear patterns of DPOAEs in 55.2% of the tumors in the full IAM group and in 71.4% of those in the partial IAM group (not statistically different). They concluded that the patterns of DPOAE (cochlear and noncochlear) are not affected by the degree of IAM involvement in the Vestibular Schwannoma (VS).
CONCLUSIONS

**CPA tumor versus control group**

OAE test results in the tumor as well as the nontumor ears were abnormal. These findings implicate the vulnerability of peripheral pathway (both tumor and nontumor side) to the pathological effects of the tumor. The pathophysiological basis for the audiological deficits found in the present study may be because of the direct effect of the tumor on the VIIIth nerve on the tumor ear side. This direct lesion effect has been attributed to compression, stretching or infiltration of the auditory nerve trunk, vascular compromise, biochemical changes in the inner ear fluids, hydrops, inner ear toxicity, deafferentation or loss of efferent nerve tuning. The nontumor ear deficits seem to evolve out of brainstem compression. In the current study, 87% of the patients had brainstem compression.

**Tumor versus nontumor ears**

In the current study, the pathophysiology induced by the tumor per se on the ipsilateral side of the lesion seemed to be more perilous than the nontumor ear deficits brought about by brainstem compression. Greater deficits were noted in the tumor ears rather than the nontumor ears DPOAE test findings.

**Tumor type versus DPOAE**

DPOAE test findings were significantly poorer in the acoustic tumor ears than the nonacoustic tumor ears. The findings obtained clearly indicate the preponderance of acoustic tumors to cause cochlear dysfunction in the tumor ears.

DPOAE were significantly poorer in the acoustic nontumor ears when compared with the nonacoustic nontumor ears. This implicates that acoustic tumor type causes a higher outer hair cell (OHC) dysfunction in the nontumor ears compared to nonacoustic type. Thus, the predominance of acoustic type to cause cochlear dysfunction in the nontumor ears is higher.

**Tumor size versus DPOAE**

DPOAE findings in the tumor ears did not demonstrate any significant difference with respect to tumor size.

OAE did not demonstrate any difference with respect to tumor size in the nontumor ears.

**Tumor extent versus DPOAE**

DPOAE did not demonstrate any significant difference in the tumor ears with respect to presence/absence of the intracanalicular component.

**ACKNOWLEDGEMENTS**

The present study is not funded by any authority and there is no conflict of interest.

**REFERENCES**

ABSTRACT

Background: Headache is one of the most common neurological symptoms in ED patients, majority being due to non-traumatic causes. The types of headache are too many as also their presentations, incidences, etiologies and demographic profiles. Though some types of headaches are life threatening, most are harmless. The primary role of physicians, amidst their busy schedules in the atypical setting of EDs, is not only to differentiate and diagnose the potentially life-threatening secondary headaches from the most common primary benign headaches but also to appropriately manage the former at the earliest to avert any untoward serious consequences including mortality.

Objectives: To study and analyze the demographic and clinical profiles of the South Indian ED patients presenting with non-traumatic headache.

Materials and methods: This retrospective study was conducted in the ED of Amrita Institute of Medical Sciences, Kochi. It included 80 consecutive patients of both the genders presenting to the ED with the primary complaint of non-traumatic (no cranial trauma in the last three months) headache. The third edition of the International Classification of Headache Disorders (ICHD-3) published in January 2018 was adhered to for the classification of the headaches. After thorough clinical evaluation, demographic and clinical profiles of these patients were studied and analyzed. Patients with insufficient clinical data and pregnant women were excluded.

Results: In the present study, most of the headaches were primary, chronic, benign, non-life threatening and non-diffuse type. Tension type headaches score over migraine, both being common in females, mostly occurring in the age group below 30 years. There is a close correlation between the headache and menstruation. Amongst males, the incidence of acute headaches was more, occurring commonly in the age group between 41 and 50 years. Two thirds of secondary headaches were life threatening; their causes being intracranial bleeds, mass lesions, vascular malformations and meningitis in that order. All our patients with intracranial bleed were found to have hypertension with IHD on medications for the same.

The most common symptoms associated with the headaches in order were: nausea, vomiting, fever (mostly low grade), giddiness, photophobia and syncope. Some of the patients had hypertension on arrival. Most of the patients’ pain score was below 3. About one third of the patients underwent imaging studies—CT and/or MRI, half of them with positive reports. More than half of the patients were disposed as out patients; males, mostly with chronic headache, of the age groups of below 20 years and between 41 and 50 years occupied most of the admissions.

Conclusion: With the small sample size, the study throws light on the demographic and clinical profiles of the South Indian ED patients with non-traumatic headache. This includes the population based variations in their presentation also.

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INTRODUCTION

Headache or cephalalgia is the symptom of pain anywhere in the region of the head and/or neck.1 “Headache is one of the commonest neurological symptoms and a complaint for which patients often seek emergency department (ED) care.”2 Headache accounted for 2.1 million ED visits per year (2.2% of visits).3 Studies have shown that of all patients referring yearly to an ED, 0.5%1 to 4.5%4-6 report non-traumatic headache as a major medical problem.4 Frequent headaches can affect relationships and employment.1 A There is also an increased risk of depression in those with severe headaches.1 Headache has a major impact on public health; worldwide, it has been ranked among the ten most disabling conditions by the WHO.2 The types of headache are varied, as also their etiology.1 Some are harmless and some are life-threatening.1 Patients who consult doctors for headache have different clinical features. The distribution of headache types and severity are also observed to differ in various population based studies.4 Studies from Greece7, Hungary8, Asia9, Chile10 and Denmark11.

There is also evidence that most of these subjects will be finally diagnosed with a benign primary headache, while a lower but noteworthy percentage (up to 19%)1 are diagnosed with a secondary headache, including life-threatening conditions such as subarachnoid hemorrhage (SAH), central nervous system infections and tumors.5 The primary role of ED physicians is to discriminate between the most common primary benign headaches and potentially life-threatening secondary headaches.12 Failure to recognize a serious headache can have serious consequences, including permanent neurologic deficits, loss of vision, and death.1 However, in the atypical setting of the ED, where time is limited and emergencies are unpredictable and drive the complex organization of the staff, the diagnosis and management of non-traumatic headache is often challenging.12

1Dept.of Emergency Medicine,AIMS,Amrita Vishwa Vidyapeetham,Kochi, India.
Objectives
To study and analyze the demographic and clinical profiles of the South Indian ED patients presenting with non-traumatic headache.

Materials and methods
This retrospective study was conducted in the Department of Emergency Medicine, Amrita Institute of Medical Sciences, Kochi- a multi-specialty, tertiary care, Postgraduate teaching Institute in South India.

Inclusion Criteria
Consecutive patients of both the genders who presented to our ED during the period between July and December 2015 with the primary complaint of non-traumatic headache.

Exclusion criteria
1. Patients in whom headache is not the primary presenting complaint.
2. Post-traumatic headache: history of cranial trauma during the last three months.
3. Pregnant women.
4. Insufficient clinical data.

Methods
Having obtained appropriate consents, all patients were exhaustively evaluated with face to face interviews by the EM physicians to obtain valid and detailed history including the type of pain, its distribution, duration, number of episodes, recurrences, frequency, aggravating and alleviating factors, associated symptoms and medical and medication history. They were assessed for pain scoring with Visual Analog Scale, neurological examination including fundus examination and all essential investigations including relevant imaging. The demographic and clinical profiles of these patients were studied and analyzed.

RESULTS
i) Total number of patients studied (Sample size)=80

<table>
<thead>
<tr>
<th>Gender</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>39</td>
<td>41</td>
</tr>
</tbody>
</table>

Table 1: Gender Distribution

<table>
<thead>
<tr>
<th>Age (yrs.)</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Min. age</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>Max. age</td>
<td>83</td>
<td>75</td>
</tr>
<tr>
<td>Below 10</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>11 to 20</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>21 to 30</td>
<td>7</td>
<td>13</td>
</tr>
<tr>
<td>31 to 40</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>41 to 50</td>
<td>10</td>
<td>4</td>
</tr>
</tbody>
</table>

Table 2: Age versus Gender distribution

<table>
<thead>
<tr>
<th>Headache</th>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>48</td>
<td>32</td>
</tr>
</tbody>
</table>

Table 3: Headache-Acute versus Chronic

<table>
<thead>
<tr>
<th>Age Group (years)</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below 20</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>21 to 30</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>31 to 40</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>41 to 50</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>51 to 60</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Above 60</td>
<td>5</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 4: Acute - Age versus Gender Distribution

<table>
<thead>
<tr>
<th>Age Group (years)</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Below 20</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>21 to 30</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>31 to 40</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>41 to 50</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>51 to 60</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Above 60</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 5: Chronic - Age versus Gender Distribution

<table>
<thead>
<tr>
<th>Headache associated with menstruation</th>
<th>Total number of female patients</th>
<th>Number menstruating females</th>
<th>Patients with headache during menses</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>41</td>
<td>38</td>
<td>27</td>
</tr>
</tbody>
</table>

Table 6: Headache-Pain distribution
<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Symptom</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Nausea</td>
<td>36</td>
</tr>
<tr>
<td>2</td>
<td>Vomiting</td>
<td>36</td>
</tr>
<tr>
<td>3</td>
<td>Giddiness</td>
<td>11</td>
</tr>
<tr>
<td>4</td>
<td>Photophobia</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>Fever</td>
<td>13</td>
</tr>
<tr>
<td>6</td>
<td>Syncope</td>
<td>5</td>
</tr>
<tr>
<td>7</td>
<td>Light headedness</td>
<td>4</td>
</tr>
<tr>
<td>8</td>
<td>Limb Weakness</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>Blurred vision</td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td>Abdominal Pain</td>
<td>2</td>
</tr>
<tr>
<td>11</td>
<td>Orbital swelling</td>
<td>1</td>
</tr>
<tr>
<td>12</td>
<td>Nasal discharge</td>
<td>1</td>
</tr>
<tr>
<td>13</td>
<td>Ear pain</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 7: Associated symptoms

Total number of patients having hypertension on arrival to ED = 10
Pulse oximetry- SpO2
SPO2 was normal on room air for all the patients on arrival.

Body temperature in °F
a) Number of patients having fever - 13

<table>
<thead>
<tr>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>99.9 °F</td>
<td>102.1 °F</td>
</tr>
</tbody>
</table>

Table 8: Fever: Minimum and Maximum temperatures

<table>
<thead>
<tr>
<th>Pain score</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
</tr>
<tr>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 9: Assessment of Pain severity
Pain scoring on arrival with Visual analog scale

<table>
<thead>
<tr>
<th>Group</th>
<th>Analgesics</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>a</td>
<td>Inj. Ketorolac 30 mg IM</td>
<td>17</td>
</tr>
<tr>
<td>b</td>
<td>Inj. Paracetamol 1 gram</td>
<td>25</td>
</tr>
<tr>
<td>c</td>
<td>Inj. Fentanyl 30 micro gram IV</td>
<td>8</td>
</tr>
<tr>
<td>d</td>
<td>Combination of (a) and (b)</td>
<td>7</td>
</tr>
<tr>
<td>e</td>
<td>Combination of (a) and (c)</td>
<td>8</td>
</tr>
<tr>
<td>f</td>
<td>Combination of (b) and (c)</td>
<td>5</td>
</tr>
<tr>
<td>g</td>
<td>Combination of (a), (b) &amp; (c)</td>
<td>6</td>
</tr>
</tbody>
</table>

Table 10: Pain relief for headache:
76 patients received the parenteral analgesics initially as follows

Efficacy of pain relief between groups (a) and (b) - comparison
Group (a) patients had better pain relief than group (b) patients.

<table>
<thead>
<tr>
<th>Imaging</th>
<th>Total</th>
<th>Positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT Brain</td>
<td>17</td>
<td>10</td>
</tr>
<tr>
<td>MRI Brain</td>
<td>14</td>
<td>10</td>
</tr>
</tbody>
</table>

Table 11: Imaging-CT & MRI: Total = 31

Final diagnosis
a) Category (1)-Primary headaches = 50
b) Category (2)-Secondary headaches = 17
c) Category (3)- Miscellaneous = 13

Tension headache-Total = 28
Males - 12
Females - 16

Migraine – Total = 22
Male - 9
Females - 13

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No</th>
<th>Ages (Years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass lesions</td>
<td>2</td>
<td>25 &amp; 48</td>
</tr>
<tr>
<td>SAH</td>
<td>3</td>
<td>46,48 &amp; 48</td>
</tr>
<tr>
<td>Intracerebral</td>
<td>4</td>
<td>42,50,51 &amp; 53</td>
</tr>
<tr>
<td>A-V mal-formation</td>
<td>2</td>
<td>58 &amp; 74</td>
</tr>
</tbody>
</table>
DISCUSSION

The third edition of the International Classification of Headache Disorders (ICHD-3), published in January 2018, is adhered to for the classification of headaches. The findings of the present study are:

The incidence of primary headaches was 62.5%, comparable with the results of three other similar studies: 50.1%5, 58%2 and 59.4%4. In contradiction are three other study reports: 81.2%2, 90%13 and >90%14,2. Among the primary headaches, 35% are tension headache and 27.5% are migraine in the present study. In three other studies also, tension headache ranks first over migraine-28.7%15, 23.7%6 and 16%6. Our study report of the incidence of migraine (27.5%) also correlates well with the reports of other studies- 23%15 and 23.8%5; but is in contrast with the reports of two other studies: 55%6 and 56%6(nearly more than double the incidences of our study). Migraine has a high chance to generate disability during attacks, leading patients to the emergency service for symptomatic control6. Both tension headache and migraine are more common in females-their incidences in females being 57% and 59% respectively; these findings correlate well with the other study reports13,16,17. Again 42.85% of the incidence of tension headache and 54.5% of that of migraine occur in the age group below 30 years-both the genders being put together.

The incidence of secondary headaches in this study is 21.25%- a comparable result with that of other studies:16%13,27%5 and 32%4. Among the secondary headaches 15% are because of life threateningcauses; this result is also comparable with another study report-18%6. The incidences of individual life threatening causes in the present study and other comparable studies-mentioned within brackets-are as follows: intracerebral mass lesions-2.5% (2%13),SAH-3.75%(1%14), intracerebral bleeds-5% (3%16), pyogenic meningitis-1.25% (3%16) and A-V malformations-2.5%. All our patients with intracranial bleeds are known hypertensive with IHD on medications for the same.

Remaining 16.25% of the headaches in our study population fall under the Category 3 of ICHD-3 classification; their causes and incidences are: hypertension-12.5%, post ictal- 2.5% and Cervical spondylitis-1.25%.

Sixty percent of the headaches are acute in nature,52% of them occurring in males, most of them belonging to the age groups below 20 years and between 41 and 50 years-each group with 12.5% incidence. In females, the incidence of acute headache is maximum in the age group between 21 and 30 years-12.5%. Chronic headaches are more common in females-56.25%, mostly occurring in their agegroup between 21 and 30 years-18.5%. In the males the incidence of chronic headache is maximum in the age group between 41 and 50 years-15.6%.

Overall, there is female preponderance in the incidence of headache-59%, most of them occurring in their age groups between 21 and 30 years-31.7% and below the age of twenty years-19.5%; the results correlate with similar study reports: 6219 and 77.84. In contrast, in the West, the incidence of headaches in females is more common in their middle age4. 92.7% of the females of our study were in the menstruating age group. Among them 71% had headache associated with menstruation, most of them belonging to the age group between 21 and 30 years-76.9%.

In the males, the incidence of headache was more common in the agegroup between 41 and 50 years (25.6%). After the age of 50 years, in both the sexes the incidence falls drastically, the results being comparable with other studies.15

In 56.25% of the patients the headache was unilateral in nature. All the patients in our study had one or more associated symptoms each with its own with incidences.[vide Table (xii) under results]. The most common associated symptoms are nausea and vomiting- the incidence of each being 45% - a comparable data with similar study report19. The other common symptoms and
their incidences in the order are: fever-16.25%, giddiness-13.75%, photophobia-12.5% and syncpe-6.25%. The other associated symptoms with least incidences are light-headedness, limb weakness, blurred vision, abdominal pain, orbital swelling, nasal discharge and ear pain.

In those who had fever, the body temperature was between 1010°F and 1020°F only in 7.7% of the patients; in all the rest, it was only less than 1010°F. The pain score was 3 and below in majority of the patients-62.5%; it was 5, 6 and 7 in 7.5%, 2.55% and 2.55% of the patients respectively. For pain relief, Inj. Ketorolac 30 mg IM was found to be more efficacious than 1 gram Paracetamol intravenous infusion over 10 minutes. 38.75% of our patients had imaging, either CT and/or MRI, 54.8% of them with positive reports-the results being comparable with other study report18. More than half of the patients were disposed as outpatients; very few had short stayobservation-2.5%-being comparable with other study report18. Only 40% of the patients were admitted. In other two studies, the admission rates were 20%19 and 82%18, the short stay for observation rate was 23%19. Most of the admissions included male patients (59.3%) with chronic headaches (59.3%), majority of them belonging to the age groups below 20 years (31.6%) and between 41 and 50 years (31.6%). In the admitted females, 23.1% were under the age of 20 years, another 23.1% were between 51 and 60 years of age and 7.7% were aged above 60 years.

CONCLUSION

With a small sample size, the study conducted to evaluate the demographic and clinical profiles of our ED patients presenting with non-traumatic headache as the primary symptom, throws light on the following facts which mostly correlate with the reports of many other similar studies conducted across the globe.

In South Indian ED population also, non-traumatic headache is one of the common presentations, most of them being primary, non serious, acute, unilateral, tension types; migraine being the next common one. Both tension headache and migraine were found to have a female preponderance, occurring commonly in the age group below thirty years irrespective of the gender. In majority of the female patients, headache was associated with menstruation.

One fifth of the headaches in our study were secondary causes, of which one sixth are because of life threatening etiologies. All the patients in our study who had intracranial bleeds were found to be on medications for hypertension and IHD. All our patients had one or more associated symptoms with varying incidences, along with headache. Pain score in the majority were only below 3. Inj. Ketorolac 30 mg IM was found to be more efficacious than Inj. Paracetamol 1 gram infusion in 10 minutes for pain relief (this needs more elaborate study with larger sample size for a firm conclusion). Less than half of the study population had imaging CT brain plain and/or MRI, more than half of them with positive findings. Most of our patients were managed as outpatients. Of the admitted patients, majority were males with chronic headaches. Ultimately, the fundamentals which form the cornerstones of ED management are, to quote: “

1) to determine the correct headache diagnosis,
2) to exclude secondary causes of headache, such as infection, mass-lesion, or hemorrhage,
3) initiate headache abortive therapy in appropriate cases,
4) provide the patient with an appropriate discharge plan that includes a diagnosis, patient education, prescriptions, and
5) prompt referral to an appropriate health care provider for definitive management.”20.

REFERENCES

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Demographic profile and distribution pattern of patients treated with radiosurgery at Amrita Institute, Kochi

Debnarayan Dutta*, Ajinkya Gupte*

ABSTRACT

Introduction: Present study is to assess the radiosurgery patient population distribution parameters in a tertiary cancer centre with dedicated robotic radiosurgery facility.

Materials and methods: During Feb 2016 till Nov 2018, all consecutive patients treated with radiosurgery accrued in the study. Demographic parameters, treatment details and geographical information were documented in a prospective protocol. Distributions of patients accordingly to the geographic locations, site of disease, intent of treatment and other parameters were documented. Analysis of patient distribution was done according to distance from the facility and disease specific distribution.

Results: Among 306 patients’ accrued (172, 56% male, mean age 51.7 years) 272 (89%) were from India and only 33 (11%) from abroad. Only 5 (2%) and 32 (11%) patients are from neighbouring states and other parts of India respectively. Among 272 Indian patients, 236 (86%) patients are from Kerala. 146 (48%), 62 (20%) and 27 (9%) patients were within 100 Km, 200 Km and 300 Km respectively. Meningioma (44, 14%) is the most common intracranial indication, followed by acoustic schwannoma (33, 11%) and AVM (28, 9%). Brain metastases were only 34 (11%). HCC was most common (37, 13%) extracranial indication followed by oligo-metastasis (45, 15%). 5% patients were below 12 years and 27% above 70 years age. 66% patients were ‘curative-intent’ treatment, 16% had fiducial placement. Curative intent treatment among Indian, within state (Kerala) and abroad patients were 89%, 78% and 67% respectively.

Conclusions: Majority of the patients are within 100 Km of radiosurgery facility. 70% patients were ‘radical intent’. Lack of expertises and dedicated academic teaching facilities are bottleneck in the expansion of radiosurgery program in SAARC countries.

Keywords: radiosurgery, demography, Indian subcontinent, distribution

INTRODUCTION

Cancer cases are increasing over the past few years.1 Apart from the nihilistic approach and huge expense for treatment, the issues that matter most to the caregivers are the time required for treatment and repeated hospital visits. Total loss of ‘work hours’ for accompanying the patient is a huge loss not only as individual, but also a loss for the society and nation. Usually, the most active and earning person of the family leads the discussions, patient care and hospital visits during treatment of the patient. It affects the family financially. Shorter treatment durations, treatments close to home and home care for palliative patients have a huge potential to reduce cost burden to the patient.2-3 Among the cancer treatments, radiation therapy requires long and continuous stay, usually for six to eight weeks. Radiosurgery has the potential to reduce radiation therapy treatment duration and have an impact on patients’ quality of life, especially in palliative setting.3 Radiosurgery is an integral part of cancer treatment.2 In recent years with invent of dedicated radiosurgery facilities for cranial and extra-cranial sites, there is both an increase in high-level evidence and acceptance as a treatment modality.4-7 There is also surge in treatment facilities. Radiosurgery is a high precision, high dose and short-course (usually up to 5 fractions) radiation therapy delivery facility to the target.5 Impact of radiosurgery is equivalent to surgery to some extent but the toxicity is almost similar to radiation therapy, which is quite low.6, 7 It is an out-patient, short course, painless procedure and hence attractive to the patients. In many indications, radiosurgery is an alternative option to open surgery, especially in benign brain tumours, prostate cancer, liver tumour and lung cancer.8-10 Though radiosurgery is a standard and integral part of cancer treatment, dedicated facilities are not yet established in the Indian subcontinent.8-10 The present study is to assess the radiosurgery patient population distribution parameters in a tertiary cancer centre located at Kochi, India with dedicated robotic radiosurgery facility.

MATERIALS AND METHODS

During the period of Feb 2016 till Dec 2018, all consecutive patients treated with radiosurgery were accrued in the study. All the demographic parameters, treatment details and geographical information were documented in a prospective protocol. Distributions of patients accordingly to the geographic locations, site of disease, intent of treatment and other parameters were documented. Analysis of patient distribution was done according to distance from the facility and disease specific distribution. All the data was collected and analyzed with SPSS 24.

RESULTS

306 consecutive patients treated with robotic radiosurgery were accrued in the present analysis. The details of patient distribution are in Table 1 & 2. Among 306 patients accrued, 172 (56%) were male and 134 (44%) female. 272 (89%) patients treated were from India and only 33 (11%) patients were from abroad. Most

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of the patients from abroad are from Oman, Bangladesh and Maldives. Only 5 (2%) patients are from neighbouring states. 32 (11%) patients are from farther states (West Bengal, Maharashtra, Delhi). Among 272 Indian patients, 236 (86%) patients hail from Kerala. 146 (48%) patients are within 100 Km of the facility. Whereas, 62 (20%) are with in 200 Km and 27 (9%) are within 300 Km of the facility. Majority of patients 62 (20%) are adjacent to the facility (within 20 Km).

Among intracranial indications, meningioma (44, 14%) is the most common, followed by acoustic schwannoma (33, 11%) and arteriovenous malformation (28, 9%). Brain metastasis patients accounted only 34 (11%). Other (54, 16%) intracranial tumours are glomus, pituitary, cavernomas and gliomas. In extracranial indications, hepatocellular carcinoma (HCC) was most common (37, 13%), followed by lung (10, 3%) and prostate (7, 2%). Oligo-metastasis (apart from brain metastasis) was 45 (15%) patients.

Mean age of presentation was 51.7 years. Only 16 (5%) patients were less than 12 years and 82 (27%) were above 70 years age. Sixty-six percentage of patients (215) were treated curative intent. Palliative intent patients were brain metastasis, oligo-metastasis (34%). 49 (16%) patients had fiducial placement (liver, pancreas).

The age distribution among Indian, abroad and within state (Kerala) patients were 52.6, 44.5 and 53.6 years respectively. Curative intent treatment in the similar cohort was 89%, 67% and 78% respectively.

### Table 1: Distribution of patients treated with robotic radiosurgery at Amrita Institute, Kochi

<table>
<thead>
<tr>
<th>Gender</th>
<th>Male</th>
<th>321 (12)</th>
<th>95 (31)</th>
<th>126 (44)</th>
<th>31 (10)</th>
<th>1 (1)</th>
<th>3 (1)</th>
<th>3 (1)</th>
<th>3 (1)</th>
<th>2 (1)</th>
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<tr>
<td>Female</td>
<td>172 (56)</td>
<td>51 (34)</td>
<td>44 (31)</td>
<td>5 (4)</td>
<td>0 (0)</td>
<td>0 (0)</td>
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<td>0 (0)</td>
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</table>

<table>
<thead>
<tr>
<th>Distance km</th>
<th>Within state (Kerala)</th>
<th>70</th>
<th>34 (21)</th>
<th>43 (27)</th>
<th>9 (5)</th>
<th>0 (0)</th>
<th>0 (0)</th>
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<tbody>
<tr>
<td>&lt;1000</td>
<td>75</td>
<td>19 (25)</td>
<td>11 (38)</td>
<td>8 (11)</td>
<td>4 (5)</td>
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<td>0 (0)</td>
</tr>
<tr>
<td>&lt;1000</td>
<td>100</td>
<td>21 (6)</td>
<td>7 (23)</td>
<td>15 (52)</td>
<td>4 (13)</td>
<td>2 (6)</td>
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<td>0 (0)</td>
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<tr>
<td>&gt;1000</td>
<td>75</td>
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<td>11 (38)</td>
<td>8 (11)</td>
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</table>
DISCUSSION

Radiosurgery is an evolving treatment option with expanding the indications and modifications in the treatment approach are common as well. Common intracranial indications are

1. Brain metastasis (less than 4 lesions with good performance status),
2. Acoustic schwannoma (Koos 2 or less, <2.5 cm),
3. Meningioma - post surgery residual / recurrent disease or small (<3 cm) deep seated,
4. AVM (<3 cm, deep seated),
5. Trigeminal neuralgia (not responding to medications or no vascular loop visible),
6. Glomus jugulare (at present SRS is standard indication),
7. Pituitary tumour (recurrent / residual).

Common extra-cranial indications are
- Lung cancer (stage I, NSCLC),
- Prostate cancer (low / intermediate risk elderly, comorbid),
- Liver cancer (inoperable HCC),
- Inoperable pancreas cancer.

Common metastatic diseases treated with radiosurgery:
- Lung metastasis (<3 in number),
- Oligo-liver metastasis, bone metastasis.

There are level I evidence for brain metastasis, lung cancer, liver tumour4-7. High level evidence for acoustic schwannoma, meningioma, AVMs4. There are exciting evidences in metastatic diseases. Indications for radiosurgery are expanding with new evidences and rationale. ‘Ascopal effect’ of radiosurgery in metastatic disease is well accepted. Immunotherapy with radiosurgery is gaining momentum.

Amrita Institute at Kochi is the only university hospital in SAARC country to have a dedicated radiosurgery facility. The dedicated robotic radiosurgery facility is functional only for last 2 years. The ‘Kochi’ model need to be analyzed to understand the potential of radiosurgery in SAARC countries. In first 18 months after the facility is functional, 306 patients received radiosurgery treatment. Among these 300 odd patients only 34 patients (10%) were brain metastasis, 52 (12%) were metastatic (stage IV) patients. 86 (22%) patients of benign brain tumours (acoustic schwannoma, meningioma, glomus) were treated with radiosurgery. Benign brain tumour patients have excellent long-term control and have minimal acute toxicity. Fiducial-based radiosurgery for liver tumours are one of the most difficult radiosurgery procedure both in terms of technicality and execution. Thirty-seven primary liver tumours (10%) were treated with radiosurgery in a protocol. As large proportion of patients are treated with curative intent and expected to have long-term control. These patients have potential for ‘cure’ and will establish the radiosurgery program. Brain metastasis is the most common indication for radiosurgery in western countries. However, there is only cognitive function benefit and no long-term survival benefit, hence cautions were taken to accrue brain metastasis patients9,10. Intracranial and extra-cranial indications are almost equal (54% intracranial and 46% extracranial).

More than 90% of the cases are from the state (Kerala) itself. More than 70% of patients are within 150 km radius of our facility. 85% of patients are with 500 km radius. Only 10% of patients are from outside the state (but from India) and only 3% of patients are from International destinations. This shows that local patients form the major proportion of radiosurgery patients. Population of the Kochi (in Kerala) is similar to any of the other metro cities in SAARC countries [Table 3]. The prevalence of cancer and the distribution of different cancers are also similar to any other state. Per capita income and ‘purchasing power’ is also similar among SAARC countries [Table 3]. Hence, all other major cities with more than 1 million population may able to sustain a dedicated radiosurgery program with full capacity (20-30 patients per month). More brain metastasis and metastatic disease patient recruitment after 2 years will improve the utilization and make the facility more sustainable. Prostate cancer patient recruitment will also increase extra-cranial patient proportion.

Radiosurgery facilities in SAARC countries

Unfortunately, there are not many ‘dedicated’ ra-
diosurgery specialists or facility in SAARC countries to pursue the tremendous opportunity. Radiosurgery tools are
1. Gammaknife,
2. LA based SRS systems (Varian Edge, Novalis Tx, Tomotherapy),
3. CyberKnife.

In SAARC countries, at present five (5) Gammaknife facilities functional and three (3) are non-functional. There are one (1) Varian Edge, six (6) Novalis Tx (with iPlan + Exactrac) facilities functional. There are at present eight (8) CyberKnife facility functional and one closed. Apart from the above, there are ongoing CyberKnife installation one each in Pakistan and Bangladesh and three in Indian centres. However, there is a need for more dedicated radiosurgery facility in all the major cities in SAARC countries [Table 4].

**Radiosurgery potential in SAARC countries**

Radiation therapy facilities are lagging far behind in SAARC countries when compared with Western countries. Majority of the radiation therapy facilities are clustered in metro cities and capitals of the country. In India, most of the dedicated radiosurgery facilities are in Delhi (4 out of 7), in Pakistan only facility is in Karachi, in Srilanka and Bangladesh also the facilities are planned only in the respective capitals. Patients from other part of the country need to travel to the capital to avail the treatment. Short course (one to three days) treatment with radiosurgery will be very cost effective and suitable for these patients. There is huge potential for radiosurgery patients in majority of the cities in SAARC countries with more than 1 million population.

**‘Bottle-neck’ for radiosurgery facility**

Major ‘bottle-neck’ for radiosurgery treatment is trained ‘man-power’. Radiosurgery was dominated by neurosurgeons, but in last decade there is a paradigm shift. Half of the patients for radiosurgery are now extra-cranial and trained radiation oncologists need to take the lead. Hence, there is a need for trained and dedicated radiation oncologists who can treat both intra and extra-cranial indications. Radiosurgery treatment needs skills like surgeon. Case selection and planning need to be like a surgery approach, not as planned treatment like adjuvant radiation therapy. One dedicated radiosurgery unit at Delhi stopped functioning due to lack of trained personal. Centres struggling for patients are centres without dedicated radiosurgery specialists. There is a need for more fellowship programs for radiosurgery, ‘hand holding’ by established centres to improve workflow of newer facilities and regular audit of the patients to improve treatment delivery and outcome.

<table>
<thead>
<tr>
<th>Countries</th>
<th>Population</th>
<th>Area (Km2)</th>
<th>Density (/km2)</th>
<th>GDP PCI (USD)</th>
<th>GDP per capita PPP ($)</th>
<th>GDP growth rate (%)</th>
<th>GDP (USD Bn)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bangladesh</td>
<td>167,114,654</td>
<td>147,570</td>
<td>1127.4</td>
<td>1093</td>
<td>3524</td>
<td>7.30%</td>
<td>250</td>
</tr>
<tr>
<td>Bhutan</td>
<td>821,347</td>
<td>38,394</td>
<td>21.28</td>
<td>2956</td>
<td>8709</td>
<td>8%</td>
<td>2.51</td>
</tr>
<tr>
<td>India</td>
<td>1360,507,890</td>
<td>3,287,590</td>
<td>411.87</td>
<td>1963.5</td>
<td>6427</td>
<td>7.40%</td>
<td>2597.4</td>
</tr>
<tr>
<td>Nepal</td>
<td>29,763,637</td>
<td>147,181</td>
<td>201.28</td>
<td>728</td>
<td>2443</td>
<td>6.30%</td>
<td>24.4</td>
</tr>
<tr>
<td>Pakistan</td>
<td>202,466,623</td>
<td>881,912</td>
<td>227.7</td>
<td>1223</td>
<td>5035</td>
<td>5.79%</td>
<td>305</td>
</tr>
<tr>
<td>Sri Lanka</td>
<td>20,950,041</td>
<td>62,705</td>
<td>334</td>
<td>3842</td>
<td>11669</td>
<td>3.70%</td>
<td>87.1</td>
</tr>
<tr>
<td>Afghanistan</td>
<td>36,373,176</td>
<td>652,230</td>
<td>55.3</td>
<td>618.3</td>
<td>1804</td>
<td>7.20%</td>
<td>20.82</td>
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<tr>
<td>Maldives</td>
<td>386,715</td>
<td>300</td>
<td>1289</td>
<td>8980</td>
<td>15184</td>
<td>6.90%</td>
<td>4.6</td>
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</table>

Table 3: Demographic and economic profile of SAARC countries

<table>
<thead>
<tr>
<th>No</th>
<th>Hospital name</th>
<th>City</th>
<th>State</th>
<th>Country</th>
<th>Model</th>
<th>No of pt treated*</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>Bangalore Institute of Oncology</td>
<td>Bangalore</td>
<td>Karnataka</td>
<td>India</td>
<td>CyberKnife</td>
<td>450/ year</td>
</tr>
<tr>
<td>2</td>
<td>Apollo Speciality Hospital</td>
<td>Chennai</td>
<td>Tamil Nadu</td>
<td>India</td>
<td>CyberKnife</td>
<td>200/ year</td>
</tr>
<tr>
<td>3</td>
<td>Artemis Hospitals</td>
<td>Gurgaon</td>
<td>Haryana</td>
<td>India</td>
<td>CyberKnife</td>
<td>280/ year</td>
</tr>
<tr>
<td>4</td>
<td>Amrita Institute (AIMS)</td>
<td>Kochi</td>
<td>Kerala</td>
<td>India</td>
<td>CyberKnife</td>
<td>180/ year</td>
</tr>
<tr>
<td>5</td>
<td>Omega Hospital</td>
<td>Hyderabad</td>
<td>Andhra Pradesh</td>
<td>India</td>
<td>CyberKnife</td>
<td>220/ year</td>
</tr>
<tr>
<td>6</td>
<td>Dr. B.L.Kapur Hospital</td>
<td>Delhi</td>
<td>Delhi</td>
<td>India</td>
<td>CyberKnife</td>
<td>CLOSED</td>
</tr>
<tr>
<td>7</td>
<td>Medanta Medcity</td>
<td>Gurgaon</td>
<td>Haryana</td>
<td>India</td>
<td>CyberKnife</td>
<td>350/ year</td>
</tr>
</tbody>
</table>
Table 4: Radiosurgery facilities in SAARC countries

<table>
<thead>
<tr>
<th>No.</th>
<th>Institution</th>
<th>City</th>
<th>State/Region</th>
<th>Country</th>
<th>Facility Type</th>
<th>Equipment</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>Indian Cancer Society</td>
<td>Mumbai</td>
<td>Maharashtra</td>
<td>India</td>
<td>CyberKnife</td>
<td>Installation</td>
</tr>
<tr>
<td>9</td>
<td>Jinah Medical Center</td>
<td>Karachi</td>
<td>Karachi</td>
<td>Pakistan</td>
<td>CyberKnife</td>
<td>800/year</td>
</tr>
<tr>
<td>10</td>
<td>ACTREC</td>
<td>Mumbai</td>
<td>Maharashtra</td>
<td>India</td>
<td>TomoTherapy</td>
<td>36/day</td>
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<tr>
<td>11</td>
<td>Tata Memorial Hospital</td>
<td>Mumbai</td>
<td>Maharashtra</td>
<td>India</td>
<td>TomoTherapy</td>
<td>35/day</td>
</tr>
<tr>
<td>12</td>
<td>TATA Medical Center</td>
<td>Kolkata</td>
<td>West Bengal</td>
<td>India</td>
<td>TomoTherapy</td>
<td>40/day</td>
</tr>
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<td>13</td>
<td>Apollo Hospital</td>
<td>Hyderabad</td>
<td>Telangana</td>
<td>India</td>
<td>TomoTherapy</td>
<td>42/day</td>
</tr>
<tr>
<td>14</td>
<td>Medanta Medcity</td>
<td>Gurgaon</td>
<td>Haryana</td>
<td>India</td>
<td>TomoTherapy</td>
<td>45/day</td>
</tr>
<tr>
<td>15</td>
<td>HCG Cancer Care</td>
<td>Bangalore</td>
<td>Karnataka</td>
<td>India</td>
<td>TomoTherapy</td>
<td>70/day</td>
</tr>
<tr>
<td>16</td>
<td>Amrita Institute</td>
<td>Kochi</td>
<td>Kerala</td>
<td>India</td>
<td>TomoTherapy</td>
<td>62/day</td>
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<tr>
<td>17</td>
<td>Indo-American</td>
<td>Hyderabad</td>
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<td>India</td>
<td>TomoTherapy</td>
<td>45/day</td>
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<tr>
<td>18</td>
<td>HCG Manavta</td>
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<td>Maharashtra</td>
<td>India</td>
<td>TomoTherapy</td>
<td>55/day</td>
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<tr>
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<td>HCG Cancer Center</td>
<td>Ahmedabad</td>
<td>Gujarat</td>
<td>India</td>
<td>TomoTherapy</td>
<td>48/day</td>
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<td>20</td>
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<td>Colombo</td>
<td>Colombo</td>
<td>Sri Lanka</td>
<td>TomoTherapy</td>
<td>35/day</td>
</tr>
<tr>
<td>21</td>
<td>Indian Cancer Society</td>
<td>Mumbai</td>
<td>Maharashtra</td>
<td>India</td>
<td>Radixact</td>
<td>Installation</td>
</tr>
<tr>
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<td>Bir Hospital</td>
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<td>Installation</td>
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<td>23</td>
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<td>Radixact</td>
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<td>25</td>
<td>Amrita Institute (AIMS)</td>
<td>Kochi</td>
<td>Kerala</td>
<td>India</td>
<td>Radixact</td>
<td>Started</td>
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<tr>
<td>26</td>
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<td>India</td>
<td>Radixact</td>
<td>Started</td>
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<td>27</td>
<td>Indraprastha Apollo Hospital</td>
<td>New Delhi</td>
<td>Delhi</td>
<td>India</td>
<td>Radixact</td>
<td>Started</td>
</tr>
<tr>
<td>28</td>
<td>TATA Medical Center</td>
<td>Kolkata</td>
<td>West Bengal</td>
<td>India</td>
<td>Radixact</td>
<td>Started</td>
</tr>
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<td>HCG EKO</td>
<td>Kolkata</td>
<td>West Bengal</td>
<td>India</td>
<td>Radixact</td>
<td>Started</td>
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</tbody>
</table>

Table 4: Radiosurgery facilities in SAARC countries

CONCLUSION

Radiosurgery is an expanding new treatment modality in both cranial and extra-cranial indications. Majority of the patients are within 100 Km of the radiosurgery facility. Usually 70% of patients treated with radiosurgery are radical intent treatment. Lack of expertise and dedicated academic teaching facilities are bottleneck in the expansion of radiosurgery program in SAARC countries. There is a need for more dedicated radiosurgery facilities in SAARC countries. Established facilities need to take the lead to ‘handhold’ the newer facilities to establish the facilities and improve expertise.

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Validation and implications of palatal height index at a tertiary care centre

Asha CL*, Rahul D Prabha*, Sapna Varma NK*, Ajith VV*

ABSTRACT

Purpose: Palatal dimensions are critical elements in orthodontic treatment planning. Outcome of arch expansion treatment either dental or skeletal are closely dependent on the initial arch dimensions. Dimensional relationship of palate has been reported as index by Korkhaus. The aim of our study is to evaluate and identify the prevalent Korkhaus palatal height index in adult patients at a tertiary care Centre.

Materials and methods: Sample consisted of study models of patients that matched the inclusion and criteria from a tertiary care centre. Fifty-seven study models (27 males and 30 females) were selected for the study. The palatal measurements were recorded with a digital Vernier caliper. Palatal height index was calculated with the Korkhaus formula.

Results: The palatal height index for males in the molar area and canine area were 58.60 ± 8.84 and 21.48 ± 4.53 respectively. The palatal height index for females in the molar area and canine region were 59.23 ± 6.57 and 24.60 ± 5.47 respectively. However, at the canine region the palatal height index was found to be increased in females than in males.

Conclusion: Proper understanding of native palatal dimensions would aid in treatment planning. The knowledge of the prevalent palatal height index for males and females would form a clinical guideline for expansion of maxillary arches in growing children.

Keywords: Palatal height index, Korkhaus, Palatal height index, Rapid maxillary expansion.

INTRODUCTION

The human dental arch dimensions are subject to continuous dimensional changes until adulthood. The maxillary arch development is dependent on dimensional changes in the inter-canine width, inter-molar width and palatal height. Major dimensional changes in the maxillary arch were reported to occur during the transition of primary to mixed dentition. Further, an increase in palatal width and height is also observed at time of the permanent canine’s eruption and the associated alveolar processes divergence. A gradual steady increase in the palatal height were reported between 5-16 yrs of age. Deviations in palatal dimension may occur due to genetic predisposition, local factors or developmental anomalies.

Abnormal palatal growth may lead to failure in attaining adequate transverse dimension. Failure to achieve the requisite palatal morphology may often lead to requirement of orthopedic corrections later. Thus, an index of palatal dimension would give prediction of adult palatal dimension.

The palatal height to depth variation were quantified and reported by Korkhaus as the palatal height index (PHI) in 1939. Palatal height index was calculated as percentage of palatal height to width. The palatal dimensional changes have considerable implications in orthodontic diagnosis, treatment planning and post retention stability.

The knowledge of the prevalent palatal height index could be used as a guide to determine the required expansion for a maintainable result. Therefore, the aim of this retrospective observational study is to evaluate the Korkhaus palatal height index percentage in adult patients in a tertiary care Centre.

MATERIALS & METHODS

Study models satisfying the inclusion and exclusion criteria were selected from the Department of Amrita School of dentistry, Kochi. Based on the results from a previous publication (8), the palatal height index for permanent dentition at canine area the palatal height index was 19.32 ± 4.64 (mean ± SD) for males and 17.27 ± 4.65 (mean ± SD) for females. Based on the mean and standard deviation, the required sample size was calculated with relative allowable error 10% and 95% confidence. The sample size obtained was 22 and 28 for males and females, respectively. Thus, we selected fifty-seven study models (27 males and 30 females) for the study (age 17-35yrs).

The inclusion criteria were
1. Study models with full complement of teeth
2. Cases with absence of any transverse discrepancies
3. Cases with minimal rotations, crowding or spacing

The exclusion criteria were
1. Teeth with carious lesions or fracture
2. Previous history of orthodontic treatment
3. Cross bite and scissor bite.
4. Habits like thumb sucking and mouth breathing.

The palatal height index was calculated by the
formula given by Korkhaus as

\[
\text{Palatal height index} = \frac{\text{Palatal height}}{\text{Palatal width}} \times 100
\]

The palatal dimensions were recorded as described by Eslami Amirabadi et al. Palatal inter-molar width was measured linearly of interdental papillae between second maxillary bicuspid and maxillary permanent first molar with digital Vernier calliper (Fig. 1). Palatal inter-canine width is measured linearly between permanent cuspid tips.

Palatal height at molar region was measured as the perpendicular distance from the distal margin of permanent first molars to the palatal vault in the midline as depicted in (Fig.2). Palatal height at canine area is measured as the perpendicular distance from a line drawn from the permanent cuspid tip to the palatal vault in the midline.

An intra-class correlation coefficient (ICC) test was employed to identify Intra-observer reliability. Randomly selected study models (n= 40) were repeatedly evaluated by one investigator with an interval of one day.

Statistical analysis was performed using IBM SPSS software. All the measurable variables such as palatal height at molar area and canine area, palatal width at the molar area and canine area, values were presented in Mean ±
SD and all categorical variables like palatal height index at canine and molar area were presented in percentage. Independent sample t test was used to compare the mean parameters between two groups, palatal height index at canine area (males and females) and palatal height index at molar area (males and females).

RESULT
From the fifty-seven study models (27 males and 30 females), the palatal height index for males in the molar area is 58.60 ± 8.84% and for the canine area is 21.48 ± 4.53% (Table 1). The palatal height index for females in the molar area is 59.23 ± 6.57% and for the canine area is 24.60 ± 5.47% (Table 2). Intra-observer reliability was found to be excellent with ICC value of 0.984.

DISCUSSION
The dental arches develop by continuous slow growth from birth to adulthood. Dimensional changes in dental arches between adolescence and adulthood is a slow continuous change. The natural palatal growth changes also influence orthodontic diagnosis, treatment planning, and retention protocols. The Korkhaus palatal height index was found to be 42% in European study population, whereas, many authors from multiple regions reported a diverse percentage. Therefore, there is a significant role played by ethnicity in determining the palatal height indices of a particular population. Hence, the determination of palatal height index pertaining to a particular population is essential in transverse arch discrepancy treatment.

Our study showed there is no difference in the palatal height index at the molar region for both sexes studied. Previous studies reported on inter molar palatal width with an increase in males when compared to females. However, Louly et al and Tsai et al suggested no statistically significant sexual dimorphism in inter-molar width. On the contrary, Al-Zubair and Thilander showed that palatal height in molar site was larger in females than males. However, the canine area palatal height index was significantly higher in females than in males.

The diverse morphological variations reported in literature suggested plausible need to establish an index for palatal variation pertaining to the local population. The results from our study indicated the prevalent palatal index of patients reported to our clinic were higher than the original Korkhaus’s index.

The treatment of transverse discrepancies depends on the skeletal age of the patient. Rapid palatal expansion is one of the commonly employed treatment modality to correct the transverse discrepancies at an early age. The Rapid Maxillary skeletal expansion should not merely be limited to correction of crossbite; as the mandibular arch development follows maxillary arch dimensions.

Based on the adult palatal height index obtained in our study, we would like to recommend the early correction of constricted maxillary arches in the selected population. The rate of mandibular arch development also needs to be monitored periodically to avoid any iatrogenic scissors bite development.

<table>
<thead>
<tr>
<th>Number of samples</th>
<th>Region</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
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<tbody>
<tr>
<td>27</td>
<td>Molar</td>
<td>58.60</td>
<td>8.84</td>
</tr>
<tr>
<td>27</td>
<td>Canine</td>
<td>21.48</td>
<td>4.53</td>
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</table>

Table 1. Palatal height index for males at molar and canine area

<table>
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<th>Number of samples</th>
<th>Region</th>
<th>Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>Molar</td>
<td>59.23</td>
<td>6.57</td>
</tr>
<tr>
<td>30</td>
<td>Canine</td>
<td>24.60</td>
<td>5.47</td>
</tr>
</tbody>
</table>

Table 2. Palatal height index for females at molar and canine area
CONCLUSION

The palatal height index at the molar area in the study population was found to be higher than the original Korkhaus palatal height index. The knowledge of prevalent adult palatal height index for males and females would form a clinical guideline for expansion of maxillary arches in growing children. Further prospective studies are planned to identify an arch expansion protocol to obtain maintainable arch dimensions.

REFERENCES

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Table 3. Literature review of palatal height index

<table>
<thead>
<tr>
<th>Author</th>
<th>Study population</th>
<th>Morphological variations in palate PH(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cleaton Jones¹⁵</td>
<td>Bantu speaking Africans</td>
<td>Males-54.3±7.5, Females-56.1±7.9</td>
</tr>
<tr>
<td>Younus et al¹⁶</td>
<td>Saudi and Egyptian</td>
<td>Saudi - 49.81±8.33, Egyptian - 48.78±8.13</td>
</tr>
<tr>
<td>Zaaba and Ashish¹⁷</td>
<td>Malaysian and Indian</td>
<td>Malaysian population –</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. 67% - low type palate (PHI ≤27.9%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. 20% - medium type of palate (PHI=28.0 to 39.9%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. 13% - high palate (PHI ≥40.0%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Indian Population –</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1. 33% - medium palate (PHI=28.0 to 39.9%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. 20% - high palate (PHI ≥40.0)</td>
</tr>
<tr>
<td>EslamiAmirabadi et al⁸</td>
<td>Iranian males and females</td>
<td>Males - 40.90 ± 6.76, Females-42.00 ± 6.17</td>
</tr>
<tr>
<td>Asha et al</td>
<td>Tertiary care centre at Kochi</td>
<td>Males - 58.60 ± 8.84 % (PHI- molar area)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Females - 59.23 ± 6.57 % (PHI- molar area)</td>
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<td></td>
<td>Males - 21.48 ± 4.53 % (PHI - canine area)</td>
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<tr>
<td></td>
<td></td>
<td>Females - 24.60 ± 5.47 % (PHI - canine area)</td>
</tr>
</tbody>
</table>


16. Younes, MF El Angbawi, AM L Dosarl. A comparative study of palatal height in a Saudi and Egyptian popula-


Cannabinoid smoking induced corpus callosum stroke in a young adult complicated with post dengue guillain-BARRE; A Rare Case report


ABSTRACT
Infarcts of corpus callosum are rare. This is a case report about the corpus callosum infarct the probable cause being cannabinoid smoking along with hypercoagulable state due to dengue fever. Though the patient managed conservatively, later he went for post dengue guillain–barre syndrome.

Keywords: corpus callosum stroke, dengue, cannabinoids, magic mushroom, GBS.

INTRODUCTION
The corpus callosum is the largest white matter tract in the human brain, interconnecting both cerebral hemispheres. The corpus callosum receives abundant blood supply from both anterior and posterior cerebral circulation. Subcallosal and the medial callosal arteries are branches of anterior communicating artery they supply rostrum and genu respectively. The anterior cerebral artery supplies most of body of corpus callosum through pericallosal artery and its branches. Posterior pericallosal artery, a branch of posterior cerebral artery supplies splenium. Infarcts of corpus callosum are not common because of its rich blood supply.

Corpus callosal lesion produces disturbance of higher brain function. Clinical features of corpus callosal infarcts are (a) interhemispheric disconnection syndrome manifested as apraxia, agraphia, tactile anomia of left hand (b) alien hand syndrome and (c) frontal type gait disorder including a wide base, shuffling gait with short steps and loss of concomitant arm swing.

Case Report
20 yr old male B.com student with no known co morbidities who was apparently normal A Day back, suddenly developed high grade continuous fever followed by gradual onset of decreased sensorium. He had associated chills and rigor, myalgia and headache. Few days later he developed paraparesis, gait imbalance and dysarthria. No history suggestive of urinary tract infection, Gastroenteritis and lower respiratory tract infection. Further probing into history revealed that he had a travel to Kodaikanal(a hill station in south India) and consumed magic mushroom (Psilocybin) few hours prior to onset of fever. He was symptomatically managed in a local hospital and found to have Dengue NS1 positive, so he was referred for further evaluation and management.

A detailed history revealed that he had the habit of regular alcohol consumption and drug abuse (cannabinoids and nicotine) for last 3 years. On examination the patient was found to be disoriented confused with Mini mental score of 24. Vital parameters were normal. Neurological examination revealed bilateral equal and reactive pupils, paraparesis of power 3/5, hypotonia and areflexia. No signs of meningeal irritation were observed.

Initial work up done for encephalitis and further work up for dengue fever. All blood parameters were normal except low platelet (1.3 lakh/ul) hemoconcentration (Hb-18 gm/dl) and dengue NS1, IgM positivity. Peripheral smear and blood C&S within normal limits. MRI BRAIN contrast study showed area of T2&FLAIR hyper intensity showing diffusion restriction noted involving the splenium of corpus callosum-likely to represent acute infarcts. He was started on conservative management considering late presentation. Work up for young stroke were initiated. All parameters like Echo, cardiac, biomarkers, fasting lipid, profile, ESR,ANA, RA-factor, Homocystine, Antiphospholipid antibody profile, lumbar puncture and CSF analysis, VDRL, HIV titre, concentration of Protein C & S, antithrombin 3 7-12 were within normal limits.

MRA and MRV showed normal study. Toxicological screening for substance abuse found to be positive for alcohol and tetrahydro-cannabinoids. Possibilities of stroke due to cannabinoids and smoking were considered. After 2 weeks of conservative management his lower limb weakness progressed with involvement of upper limb, sparing the respiratory muscles and facial nerve. His deep tendon reflex shows areflexia. Lumbar puncture shows albumino cytological dissociation and motor nerve conduction study shows absent CMAP amplitude from left peroneal nerve with normal sensory conduction. Antiganglioside antibody test shows negative test and diagnosis of post dengue associated Guili-
DISCUSSION
Cerebrovascular events in young and middle-aged persons can become a diagnostic problem since numerous disorders may lead to a stroke. In some patients, the cause remains unclear. Stroke in adult over 65 years constitute majority of strokes. However approximately 25% of stroke cases occur in patients under 65 years of age. Common causes of these stroke include haematological disorder, cardioembolic disease, trauma and substance abuse. About 30% of young adult stroke have cryptogenic aetiology.

Magic mushrooms are mushroom containing psilocybin & psilocin chemically related to lyseric acid diethylamide (LSD). That produces hallucination mostly visual, feeling of euphoria, sensory distortion within 30 minutes to 2 hours after ingestion of mushroom. Duration of symptom is typically 4-12 hours after consumption. Patient can present with mild tachycardia, hypertension, dilated pupils and hyper-reflexia. Treatment is supportive, can give benzodiazepine in case of anxiety and panic attacks. Toxicoological samples are negative for psilocybin. Stroke due to psilocybin toxicity is not reported till now14,15,16.

Marijuana remains most commonly used recreational drug. It is commonly used in cigarette form, but can be consumed orally. It mainly produces psychotropic effects, also produces hypotension, tachycardia, increase in concentration of carboxyhemoglobin, nausea, hunger, conjunctival congestion, and dryness of mouth & throat17,18. Heavy smoking associated with chronic bronchitis, airway obstruction and squamous metaplasia of respiratory tract.

Stroke symptoms have been described after marijuana smoking in few cases17-21. Here patients smoked marijuana cigarette daily for last 3 years and toxico logical sample for substance abuse was positive for Tetrahydrocannabinoids (THC). He was a light smoker, smokes less than 10 cigarette per day, so the risk of stroke from this factor is may not be significant according to the Framingham study22. MRA shows no evidence of atherosclerosis also. Intravenous administration of THC produces systemic and ocular hypotension23-24,17. It can produce inconstant changes in pulse rate and blood pressure25,26.

A review of 64 published case of acute ischemic stroke associated with cannabis found that majority had a temporal causal relationship with cannabis after excluding other possible cause of stroke and recurrent stroke after reuse of cannabis. Most of patient developed stroke immediately following cannabinoid smoking. A cross sectional national survey of hospitalised patient with acute ischemic stroke found that as compared to non-users cannabinoid users had a 17% increased chance of ischemic stroke27,28.

Stroke after dengue is uncommon, that may be hemorrhagic or ischemic. Intracerebral haemorrhage due to thrombocytopenia have been reported, but ischemic stroke is a rare complication. Probable mechanism for occurrence of ischemic stroke in dengue fever is usually meningo vasculitis. Hypercoagulable state due to dengue infection is also another possibility. Here patient doesn't show any features of vasculitis like skin lesion, raised CRP&ESR, and leukocytosis29-30.

Here stroke is multifactorial – most probably due to cannabinoids in the background of hypercoagulable state produced by dengue infection. After 2 weeks of conservative management patient developed post dengue Guillain-Barre.

Dengue fever is a mosquito borne infection caused by arbovirus & transmitted by Aedes mosquitoes. Most common neurological complication of dengue is encephalopathy and encephalitis. The other manifestation include myositis, GBS, polyneuropathy and myelitis-31. Dengue has 4 different serotype-DEN-1, DEN-2, DEN-3 and DEN 4. Out of these DEN-2 & DEN-3 are mostly associated with severe neurological disease31. Most common antecedent infection in GBS is campylobacter jejuni and cytomegalovirus infection. Few cases of GBS after dengue fever also reported. It is characterised by areflexia, ascending paralysis & sensory changes32. It is the most common peripheral nervous system complication of dengue fever in GBS and it occurs during recovery phase of illness33. Essential investigation in GBS include lumbar puncture and nerve conduction studies. Lumbar puncture shows increased protein concentration without pleocytosis (albuminocytological dissociation), and nerve conduction studies will show features of de myelinating polyneuropathy34. Mainstay of treatment include plasmapheresis and IV Ig. Corticosteroid have been proven ineffective as reported in several studies.

CONCLUSION
Cannabinoid smoking induced corpus callosal stroke in young adult in the background of hypercoagulable state due to dengue fever is a rare case. GBS is a rare neurological complication of dengue infection. It should always be considered if a patient develops progressive weakness of limbs and treatment should be initiated as early as possible.

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A case of Recurrent Fractures

Anuj Singhal*, Sonal Singh*

Corresponding Author: Anuj Singhal, Associate Professor, Dept. of Internal Medicine, AFMC Pune, India.

INTRODUCTION

Unlike western world, Primary Hyperparathyroidism (PHPT) still remains a symptomatic disease in India. Routine biochemical screening for serum calcium accounts for identification of majority of cases of asymptomatic hypercalcemia in western world1. However the disease profile has definitely changed in the past few years in India. In a retrospective study conducted in South India the clinical, biochemical and pathological profile of PHPT was studied in 110 patients. It was found that the disease is being diagnosed now earlier and pathological fractures have become less frequent2. These geographical differences in the manifestations of PHPT can be partly explained on the basis of Vit D deficiency. It has been seen that individuals with co existent Vit D deficiency have more severe disease and more frequent fractures.

Here we present a case of Primary Hyperparathyroidism who presented to us with recurrent fractures.

Case

A 42 year old male presented with complaints of bony pains and constipation of 6 months duration. He also gave history of fracture of left forearm (figure 1) which he sustained 02 months back while lifting a 3 kg weight and another fracture of left arm (figure 2) 2 years back. However there was no history of abdominal pain, nausea, vomiting, weight loss or anorexia. There was no history of abnormal behavior. He also gave history of renal calculi 2 years back which was managed conservatively. On examination patient was afebrile, his vitals were stable. There was no neck swelling and his general and systemic examination was unremarkable. His investigations were as follows-

- Hb-13.6 g/dl, TLC-7900/cmm, Platelet count-2.53 lakhs/ cmm, Total Bilirubin-1.0mg/dl, Total Protein-6.8 g/L, Albumin/Globulin-4.2/2.6 g/l, AST/ALT-22/19 IU/L, ALP-1077 IU/L, Serum Urea/Creatinine-10/0.6 mg/dl, Na/K-142/4.5 meq/l, Serum Calcium(corrected)-12.6 mg/dl, Serum Phosphorus-4.3 mg/dl, ECG-WNL, Chest X Ray-NAD. Ultrasound abdomen revealed bilateral non obstructing renal calculi.

On further evaluation his serum PTH levels were found to be elevated (536.8 pg/ml) and Vit D3 levels was 18.9 ng/ml (30.0-100) which was deficient. Subsequently Ultrasound imaging of the thyroid gland revealed an oval hypoechoic lesion measuring 1.9 x 2.5 x 4.7 cms noted on the posterior aspect of Right lobe of Thyroid gland (Fig 3). CECT neck confirmed the findings of ultrasound (Fig 4). Dual Energy X ray Absorptiometry (DEXA) scan for bone mineral density was suggestive of osteoporosis (Table 1). On further evaluation, Bone scan revealed abnormally increased activity in iliac crests. Tc 99 Sestamibi Scan for Parathyroids revealed abnormal focus of tracer concentration in Right inferior Parathyroid gland. Based on these findings a diagnosis of Primary Hyperparathyroidism secondary to Right Inferior Parathyroid Adenoma was made.

Patient was initially managed with IV and oral hydration, Inj Zolendronic acid and Vit D replacement following which he underwent surgical removal of inferior Parathyroid Gland (fig 5).
Fig 3- USG Thyroid depicting an oval hypoechoic lesion on the posterior aspect of Right lobe of Thyroid gland

Fig 4- CECT neck showing Right Inferior Parathyroid Gland Adenoma

<table>
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<tr>
<th>SITE</th>
<th>T SCORE</th>
<th>Z SCORE</th>
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<td>Spine</td>
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<td>-3.7</td>
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<tr>
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<td>-3.0</td>
</tr>
<tr>
<td>Radius</td>
<td>-9.0</td>
<td>-8.7</td>
</tr>
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</table>

Table 1- Dual Energy X ray Absorptiometry (DEXA) scan for bone mineral density
DISCUSSION

The classical symptoms of PHPT described as “bones, stones, abdominal moans and psychic groans” are due to combined effects of increased PTH secretion and hypercalcemia. The bone and stone disease are due to prolonged PTH excess whereas hypercalcemia results in anorexia, nausea, constipation, polydipsia, and polyuria. The classical manifestation of parathyroid bone disease, osteitis fibrosa cystic characterized by bone pain, subperiosteal bone resorption on radiograph, bone cysts, and brown tumors of the long bones is rare and is seen in patients with Parathyroid carcinoma. Patients have reduced bone mineral density (forearm and hip) and are at increased risk of vertebral fracture. The renal manifestations of PHPT are nephrolithiasis, hypercalciuria, nephrocalcinosis and chronic renal insufficiency. Nephrolithiasis is seen in 15-20% of cases. Neuropsychiatric manifestations of PHPT include lethargy, depressed mood, psychosis, decreased social interaction and cognitive dysfunction. Cardiovascular abnormalities are the major cause of mortality in patients with severe PHPT and Hypertension is the most common cardiovascular manifestation.

Diagnosis of hyperparathyroidism is suspected on the basis of clinical manifestations and elevated serum calcium. Measurement on intact PTH helps to distinguish it from other causes of PTH independent hypercalcemia such as malignancy. An elevated or high normal intact PTH in the setting of hypercalcemia is suggestive of PHPT. A 24 hr urinary calcium excretion is required for distinguishing PHPT from familial hypocalciuric hypercalcemia (FHH). Vitamin D level can be elevated in PHPT as these patients convert more 25OHD (calcidiol) to 1,25 dihydroxyvitamin D (calcitriol) than normal individuals. However there is a significant prevalence of concomitant Vit D insufficiency in patients with PHPT. BMD measurement and renal imaging are not required for the diagnosis of PHPT. However, BMD measurement at hips, forearm and spine forms an essential part of management of the disease. Routine imaging for renal stones is no longer recommended, but if there is a history of renal stones or if they are suspected, an ultrasound can be done. Localization studies with USG or technetium-99m sestamibi, CT, or MRI scanning is not required for the diagnosis. However, they may be required intraoperatively for minimally invasive surgery.

Parathyroidectomy forms the mainstay of treatment for patients with symptomatic primary hyperparathyroidism. Traditionally bilateral neck exploration is the standard surgical approach. However, with the availability of improved imaging modalities and intraoperative PTH monitoring, minimally invasive Parathyroidectomy is emerging as the procedure of choice. Patients who cannot undergo surgery should be managed with medical therapy. For patients in whom the primary indication for surgery is low bone mass and high risk of fracture and are unable to undergo surgery Bisphosphonates should be used. For patients in whom the primary indication for surgery is severe symptomatic hypercalcemia and are unable to undergo surgery Cinacalcet should be used. Patients with concomitant Vitamin D deficiency should be given Vitamin D supplements.

CONCLUSION

Asymptomatic Primary Hyperparathyroidism is a common entity in western world. However, in India patients still present with symptomatic disease. Concomitant Vitamin D deficiency further worsens and complicates the disease. An increasing awareness of Primary Hyperparathyroidism among general physicians and routine biochemical screening for serum calcium may change the scenario. Although symptomatic PHPT is discussed here asymptomatic PHPT also requires close observation and follow and follow up.

REFERENCES

Case report on Intra hepatic Portal vein aneurysm - A rare vascular anomaly

Vijay Anand Viswanathen*, Remya Sudevan**

ABSTRACT
Aneurysms of the portal vein, superior mesenteric vein and splenic vein at spleno – portal junction constitute the portal venous aneurysm (PVA). The condition is less reported in literature since the prevalence is low. PVA can be congenital or acquired. There are intra hepatic and extra hepatic PVA. Recent advancements in the imaging technology have increased the detection rate but the exact aetiology, clinical manifestations and management plans are in the evolving phase. We report a case of a PVA detected incidentally while evaluating for abdominal pain in a 36 year old male. A literature review and various management plans are also discussed for this distinctive condition.

Corresponding Author: Remya Sudevan, Clinical Epidemiologist, Dept. of Health Sciences Research, AIMS, Kochi, India.

Introduction with literature review
The portal venous system or hepatic portal system in humans is composed of superior mesenteric, inferior mesenteric and splenic veins draining blood to the liver through portal vein. The hepatic portal vein eventually drains into the inferior vena cava (Fig 1). The area covered by the system extends from lower part of oesophagus to the upper part of anal canal, spleen and pancreas. Among vascular disorders venous aneurysms are unusual in occurrence. Portal venous aneurysm (PVA) represents the focal dilatation of portal venous system. This is an extremely rare congenital/acquired vascular anomaly constituting <3% of all venous aneurysms. PVA was foremost described by Barzilai and Kleckner in 1956. Less than 200 cases have been reported under PVA across the globe till date. The reported prevalence of PVA is 0.43%. The usual parts affected with PVA are main portal trunk and the confluence of splenic and superior mesenteric veins. Liver cirrhosis and portal hypertension are the primary causes for acquired PVA. Pancreatitis, trauma and invasive malignancies are the secondary causes for acquired PVA. The diagnostic standard diameters for extra hepatic and intra hepatic PVA are >20 mm and >9 mm respectively.

Due to the rarity of the condition PVA was observed only in 0.067% of patients by ultrasonography. Newer imaging modalities, especially Multidetector computed tomography (MDCT) has expanded the detection rate of PVA. This is detected incidentally with asymptomatic findings. The documented complications are thrombosis, spontaneous/progressive rupture, portal hypertension and compression of adjoining vessels/visceral structures. PVA has multimodality appearance imitating solid, cystic and hyper vascular abdominal masses. Even though the surveillance of the condition has increased the exact pathogenesis and management remains ambiguous. The clinical characteristics of PVA patients are shown in Table 1.

Our report is an attempt to characterize the etiology, clinical presentation, diagnostic and treatment strategies of PVA on the basis of literature review and current practice pattern.

<table>
<thead>
<tr>
<th>Patient characteristics</th>
<th>Values</th>
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<tbody>
<tr>
<td>Total number of patients</td>
<td>190</td>
</tr>
<tr>
<td>Median age of diagnosis (years)</td>
<td>52 (0–89)</td>
</tr>
<tr>
<td>Male:Female ratio</td>
<td>1:1</td>
</tr>
<tr>
<td>Portal hypertension</td>
<td>62 (32%)</td>
</tr>
<tr>
<td>Liver cirrhosis</td>
<td>50 (26%)</td>
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<td>Aneurysm localization</td>
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<tr>
<td>Main portal trunk</td>
<td>73 (38.4%)</td>
</tr>
<tr>
<td>Spleno-mesenteric confluence</td>
<td>45 (23.6%)</td>
</tr>
<tr>
<td>Portal bifurcation/Intra-hepatic</td>
<td>72 (38%)</td>
</tr>
<tr>
<td>Surgical management</td>
<td>40 (21%)</td>
</tr>
<tr>
<td>Postoperative mortality</td>
<td>7 (17.5%)</td>
</tr>
</tbody>
</table>

Table 1: Characteristics of PVA patients from various published reports

*Dept. of Gastroenterology, Aster medical centre Dubai,
**Dept. of Health Sciences Research, AIMS, Amrita Vishwa Vidyapeetham, Kochi, India.
Case description
A 36 year old gentleman presented with recurrent abdominal pain exacerbating in the evening and needed emergency care for duration of one week was evaluated. He was a known smoker, non alcoholic and known hypertensive and was under regular treatment for hypertension. He had normal bladder habits. Bowel habits were altered with constipation. There was no history of diabetes, hepatitis, any other chronic diseases and surgery. Family history was not relevant. On physical examination the vitals were stable. On palpation abdomen appeared soft, nontender with normal bowel sounds. There was no palpable mass detected. The liver felt enlarged with 2 cms. No abnormalities were detected in other organ systems. Complete blood count, Liver function test and Serum amylase were within normal limits. Ultrasonography was done and revealed tortuous portal vein, portal vein aneurysm and hepatomegaly (Fig2&3). MDCT abdomen was done to confirm PVA and the conclusion was Portal vein aneurysm with portal vein diameter 26 mm(Fig 4&5).Upper gastro intestinal video endoscopy was done during follow up to rule out varices and the impression was Hiatus hernia with LA grade B reflux oesophagitis.

The provisional diagnosis was dyspepsia with generalised abdominal pain and hepatomegaly. The condition was initially managed with Proton pump inhibitors and health education on lifestyle modifications. The final diagnosis was Hiatus hernia with LA grade B reflux oesophagitis and PVA. The management was with Proton pump inhibitor, antispasmodic agent and antihypertensive with regular follow up. Plan to manage PVA was to do regular follow up without any treatment.
Fig 3: Ultrasonography of abdomen showing dilated and tortuous main portal vein

Fig 4: MDCT abdomen showing PVA

Fig 5: MDCT abdomen showing PVA
DISCUSSION

PVA being a vascular anomaly coming under visceral aneurysms has been classified as congenital and acquired. The etiology of the congenital condition is defined as; Portal vein develops from vitelline and umbilical veins during embryological development. Absence of regression of right primitive vitelline vein cause congenital portal vein aneurysm. A diverticulum will be developing from the vitelline vein remnant which enlarges by time, increasing the portal vein pressure and eventually leading to saccular portal vein aneurysm. For acquired PVA portal hypertension, cirrhosis, pancreatitis, trauma and invasive malignancies are given as the causative factors. According to location PVA are of two types- extrahepatic (63%) and intrahepatic. PVA has no gender predisposition in occurrence and the mean age of diagnosis is 53 years. A retrospective study of 4186 randomly assigned patients had shown that 72% of the total was asymptomatic. The clinical symptoms are proportionally related to size. The presenting symptoms are nonspecific epigastric pain and gastrointestinal bleeding, abdominal swelling and jaundice. Mass effect occurs due to compression of adjacent structures resulting in jaundice and duodenal obstruction. PVA rupture is rare(2.2%) due to low portal venous pressure. Cavernous transformation of the main portal vein can cause spontaneous regression of PVA.

Ultrasound is a useful modality to understand the character of portal vein aneurysms and to distinguish from hyper vascular mass. Color Doppler ultrasonography shows color flow in the lesion and duplex Doppler ultrasonography displays the hepatopetal flow along the aneurysmal wall. Contrast-enhanced Computed tomography (CT) and Magnetic resonance imaging (MRI) are needed to confirm the findings and to plan for management. In CT/MRI scan, PVA appears as a well-defined contrast enhanced mass communicating with the portal vein in portal phase. Management lines for PVA are constant monitoring, medical, surgical and endovascular treatment. When PVA are stable with no complications serial imaging is advised. Small asymptomatic aneurysm without cirrhosis/portal hypertension are managed conservatively by regular monitoring of size and symptoms. In complicated PVA such as with rupture, thrombosis, symptomatic aneurysm and non thrombotic PVA of size >3cm, surgical management is the better option. Aneurysmorrhaphy or aneurysmectomy can be performed in saccular or fusiform PVA. This procedure helps to restore the laminar flow of portal vein, maintains normal hepatic flow, decreases stasis and prevents formation of thrombosis. Shunt procedures like spleno-renal shunt/ porto-caval shunt that helps to decompress the portal venous system thereby preventing progressive dilatation of the aneurysm is effective in treating the aneurysm. When anticoagulation therapy is unsuccessful, Percutaneous thrombolysis or thrombectomy can be done in patients with splenic and superior mesenteric vein thrombus. PVA leading to liver transplantation is also documented. The procedures required are technical innovations like arterialisation of portal vein, interposition graft and cavoportal hemi transposition.

CONCLUSION

Our case was an asymptomatic PVA incidentally detected during the evaluation of abdominal pain. Finally the diagnosis was Hiatus hernia with LA grade B reflux esophagitis. The PVA detected was planned to manage conservatively without any treatment. PVA is a rare vascular abnormality which can mimic solid, cystic and hyper vascular abdominal mass tumors. Clear evidence based recommendations for the management of the condition are unavailable in current literature. Various treatment modalities exist for the management of PVA. Conservative management is indicated for majority of patients who are asymptomatic and without any complications. For patients at risk of mortality and with complications surgical intervention is performed. Post operative mortality is high as per the literatures. To conclude, for a PVA patient, a multidisciplinary approach with regular follow up and clinical care from a tertiary care centre is needed for the optimal outcomes.

REFERENCE


Case Report: Osteopoikilosis - Incidental Finding

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**INTRODUCTION**

Osteopoikilosis is a rare disease with an estimated incidence of 1 in 50,000 and an unknown etiology. It is an asymptomatic, autosomal dominant characterized by multiple, discrete round or ovoid radio densities in cancellous bone. Initially described by Albers-Schonberg in 1915, these are symptomatic and incidental findings. They histologically represent compact bone islands. They are occasionally associated with cutaneous lesions as well as other osteoclerotic skeletal disorders.

**Case Summary**

A 28 years old male, chronic smoker was admitted with low to moderate grade fever, intermittently associated with chills and rigors of 6 days duration and associated cough with expectoration of 6 months duration. There was no h/o bodyache, malaise or bone pain.

Clinical examination was unremarkable. His laboratory values including erythrocyte sedimentation rate, rheumatoid factor, kidney and liver function tests, serum calcium, phosphorous, magnesium, alkaline phosphatase, uric acid level and parathyroid hormone levels were normal.

Investigations: Hb - 12.6 gm/dl, TLC - 8,300/cu mm, PCV - 36.8%, MCV - 65.9 cu micron, DLC - N 70%, L-24%, E-04%, M-02%, Platelet- 1,77,000/cu mm, MP- Negative. Sr Urea - 16 mg%, Sr Creatinine - 0.9 mg%, LFT- Bilirubin - T/D- 0.6/0.4 mg%, SGPT- 16 IU/L, SGOT- 19 IU/L, Widal - Negative, ESR - 06 mm 1st Hr. (Win Trobe Method), Sr Alkaline Phosphatase - 63 U/L, Sr Uric Acid - 6.1 mg/dl, Sr Calcium - 8.6 mg%, Sr PO4 - 4.2 mg%, Sr PTH - 37.9 pg/ml, Vitamin D3 - 17.4 ng/ml, Sr-TSH - 1.76 mIU/L.

The radiograph of hip joint showed juxta-articular osteopenia and multiple small discrete, round, oval, dense lesion in symmetric distribution. These lesions were distributed more in the epiphysis and metaphysis of both the femoral bones at the proximal end extending down to intertrochanteric region (Figure 3). Pelvic bones showed dense small discrete lesions along the left sacroiliac joints, pubic symphysis and bilateral inferior pubic rami. All findings were suggestive of osteopoikilosis.

He was treated symptomatically, and he recovered from his acute febrile illness. However, the incidental finding of osteopoikilosis was made in this patient who never complained of any bony pain.

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**DISCUSSION**

Osteopoikilosis (osteopathia condensans disseminata, or spotted bone disease) is an asymptomatic disorder characterized by an abnormality in the enchondral bone maturation process. It may either be inherited in an autosomal dominant fashion or occur sporadically. Familial clustering suggests a dominant inheritance. Men and women are equally affected.4,1

According to an epidemiologic study on 53 patients with osteopoikilosis, members of four families, Benli et al.1, noticed that most frequent sites for OP appearance were the phalanges (100%), carpal bones (97.4%), metacarpals (92.3%), phalanges of the foot (87.2%), metatarsals (84.4%), tarsal (84.6%), pelvis (74.4%), femur (74.4%), radius (66.7%), ulna (66.7%), sacrum (58.9%), humerus (28.2%), tibia (20.5%), and fibula (12.8%). It is generally accepted in the literature that it is more frequently located in long bones and pelvis and male to female ratio is 3:2.2,3

Although osteopoikilosis is generally considered an incidental finding, several developmental dysplasias coexisting with this disorder have been reported8. Izge Gunal et al. reported a family in whom various members had osteopoikilosis with 5 different associated lesions and suggest that osteopoikilosis is bone manifestation of a generalized fibroproliferative or stenosing disease.9

**Differential diagnosis**

Enostosis, histopathologically and radiographically, most closely resembles osteopoikilosis and the difference is that bone islands can be isolated and small. If multiple, they are usually scattered and do not display a characteristic periarticular distribution. Histopathologically, the lesions consist of compact, markedly hypertrophied trabeculae composed of lamellar osseous tissue.10 Osteons within a bone island are not regularly oriented and contain well-vascularized canals surrounding narrow rings of lamellae, which are empty of osteoclasts and osteoblasts.10 Both osteopetrosis and pycnodysostosis have a very different radiographic appearance from osteopoikilosis; the lesions appear in a more diffuse pattern of sclerosis, rather than spotted or rounded.11 These patients also have severe systemic manifestations.

Osteopathia striata also has a different appearance. The sclerotic areas within the bone are neither round nor oval. Instead, they are linearly striated and periarticular in distribution.10 Clinical manifestations of this disorder are subtle or nonexistent. Histopathologic and biochemical studies are able to differentiate osteopetrosis and pycnodysostosis from osteopoikilosis, but little pathologic data are available on osteopathia striata. Fortunately, all of these disorders can be diagnosed and differentiated readily by their radiologic features.12

**CONCLUSION**

When uniform multiple radio-dense round or oval sclerotic lesions in a periarticular distribution lesions are found on radiographic examination, Osteopoikilosis must be in the differential diagnosis before invasive diagnostic procedures and unnecessary treatment is planned.

Early recognition is essential to prevent unnecessary emotional distress and invasive testing. No routine follow-up or studies are necessary. The recognition of this asymptomatic condition by plain radiograph makes other expensive investigations unnecessary.4

**REFERENCES**

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