PACIFIC JOURNAL OF MEDICAL SCIENCES

VOLUME 9, No. 1, September 2011

PACIFIC JOURNAL OF MEDICAL SCIENCES

(Formerly Medical Sciences Bulletin)

ISSN: 2072 - 1625

Volume 9, No. 1, Sept 2011

A multidisciplinary journal for publication of medical and biomedical research findings on issues pertinent to improving family health and related issues of public health

INFORMATION

SUBSCRIPTIONS: Correspondences concerning subscriptions, purchase of single copies and back issues, lost copies and related business should be addressed to the Secretary, Basic Medical Sciences, School of Medicine and Health Sciences, University of Papua New Guinea, P. O. Box 5623 Boroko, N.C.D., PNG. Official website is www.pacjmedsci.com; Instructions to Authors are indicated at the back of the journal.

Pacific Journal of Medical Sciences

(Formerly Medical Sciences Bulletin)

A multidisciplinary journal for publication of medical and biomedical research findings on issues pertinent to improving family health and related issues of public health

September 2011: ISSN: 2072 – 1625 VOLUME 9, No. 1

TABLE OF CONTENTS:

ADENOMATOID ODONTOGENIC TUMOR WITH RARE CLINICAL AND RADIOLOGICAL PRESENTATION- A CASE REPORT

T.G Shrihari,

Department of Oral Medicine and Radiology, Krishnadevaraya Dental College and Hospital, Yelahanka, Bengaluru, Karnataka, India

(Email: drshrihari.harry@yahoo.co.in; drshriharіomr@gmail.com.)

ABSTRACT:

The Adenomatoid odontogenic tumor (AOT) is a benign (hamartomatous) noninvasive lesion but progressive growth, constituting only 3% of all odontogenic tumors. Most common site of AOT is maxillary anterior region especially canine region. Most common variety of AOT is Follicular variety (73%) which is associated with impacted tooth (maxillary canine). Females are most commonly affected than males. We report on a rare case of follicular AOT in the mandibular anterior region seen in an 18 years old male patient. Diagnosis of adenomatoid odontogenic tumor should be considered when the clinician is presented with a corticated radiolucency in the anterior lower jaw, especially in teens and young adults.

Key words: Adenomatoid odontogenic tumor; Maxilla; Root resorption;

Received: May 2011: Accepted June 2011

INTRODUCTION:

The odontogenic tumors are a diverse group of lesions that represent the deviation from normal odontogenesis. The AOT is an epithelial tumor with an inductive effect on odontogenic ectomesenchyme. AOT can be clearly distinguishable from the classic intraosseous, infiltrative ameloblastoma. It was suggested to abandon the previously used term adeno-ameloblastoma; Philipsen and Brin [1] introduced the above term (AOT), which was adopted by the WHO classification in 1971 [2]. The benign (hamartomatous) noninvasive AOT appears in 3 clinic topographic variants; 1) Follicular, 2) Extra follicular and 3) peripheral. Follicular and extra follicular variants are both intra-bony or central tumors and account for 97% of all AOTs of which 73% are of the follicular type [3]. The extra follicular variant is not associated with a unerrupted tooth like the follicular variant, and the well defined, unilocular radiolucency is found between, above, or superimposed on the roots of erupted teeth. It is characteristic that the rare sub variant mimicking a periapical lesion is in fact located palatally (or lingually) to the tooth

involved [4]. Sixty – nine percent of AOTs are diagnosed in the second decade of life, and more than half of the cases (53%) occur during the teenage years (13 to 19 years of age). The females to male ratio for all age groups and AOT variants together is very close to 2:1 and predominantly seen in the maxilla (maxilla : mandible = 2.6:1) [5].

CASE REPORT:

An 18 year old male reported with a swelling of the right lower jaw region since 2 months. The swelling gradually progressed to attain its present size in two months duration. Extraorally (Figure 1) swelling measured 4x5cm and extended mesio-distally from left parasymphisis to right parasymphysis region. Supero-inferiorly extended from 1.0cm below the vermilion border of the lower lip to the sub mental region; it covered the whole chin region with diffuse margins. Intraorally (Figures 2 & 3) the swelling extended labially from distal aspect of region 32 to mesial aspect of region 44, with vestibular obliteration. Lingually a diffuse swelling was noted extended from distal aspect of region 41 to mesial aspect of region 44. The consistency was firm. Grade 3 mobility i.r.t over retained 83 (lower right deciduous canine). 31, 41, 42 showed grade 2 mobility.

We came to the Provisional diagnosis of dentigerous cyst and differential diagnosis of calcifying epithelial odontogenic cyst, central giant cell granuloma, adenomatoid odontogenic tumor was considered.

INVESTIGATIONS:

Radiographically Intra oral periapical radiograph (IOPA) shows well defined unilocular radiolucency measuring about 4x3 cm extending mesiodistally from distal aspect of 45 region to other side of the radiolucency can't be make out with an impacted dilacerated 43, radiolucency extending at the apex of the root. supero- inferiorly extending from alveolar crest region i.r.t 41,42, over retained deciduous right canine (83) to lower border of the mandible causing thinning of the lower border of the mandible. External root resorption i.r.t 83 (right lower deciduous canine) with displacement of teeth roots i.r.t 41, 42, 44 & 45 (Figure 4);

Occlusal radiograph shows well defined buccolingual cortical expansion (Hydraulic expansion), extending from distal aspect of 33 region to mesial aspect of 46 (Figure 5).

Orthopantomogram (OPG) shows well defined radiolucency measuring about 3x4cm extending mesio- distally from region 33 to 45, supero–inferiorly from alveolar crest region from 42, 44 region to lower border of the mandible causing thinning and expansion, which is surrounded by well defined corticated radiolucency. Dilacerated apically displaced impacted tooth is embedded within the radiolucency which is, extending till apex of the 43. Loss of lamina dura i.r.t apical 1/3rd of 31, 32, 41 to 45 regions (Figure 6); Pulpal vitality test was done, except 83 all the teeth showed

positive response.

FNAC (Fine needle aspiration cytology) was performed showed straw – color aspiration, subjected for protein investigation (3.9 mg %), other microscopic examinations like cholesterol crystal examination, which was negative (Figure 7).

Surgical enucleation of the tumor for histopathological examination was performed under local anesthesia.

Microscopically tissue section of the specimen showed odontogenic epithelium proliferating in the form of whorls with rosset like pattern, showing globular calcifications, duct like areas are seen with hyperchromatic epithelial cells suggestive of adenomatoid odontogenic tumor (Figures 8 A & B)

DISCUSSION:

AOT is an uncommon tumor or benign, hamartomatous growth derived from odontogenic epithelium. These tumors tend to develop in younger people and are more common among women, but in our case it was found in adult male patient [3].

Three clinico - pathological variants are well recognized; follicular, extra follicular, and peripheral. The follicular and extra follicular variants are intra-osseous and account for about 96%. The maxilla, often together with an unerupted canine, is most commonly affected than the mandible. In our case it was found in mandible with an unerupted canine seen in only 35.7% of cases [3, 5].

Our case we found separate follicular AOT associated with a right mandibular canine, in a 18 year old boy with radiographic findings mimicking dentigerous cyst. Radiographically unilocular radiolucency with no internal calcification has been seen in only 27% of cases. To differentiate from dentigerous cyst, CEOC, Radicular cyst, and CGCG, mandibular premolar- molar region with an impacted tooth is the common site for dentigerous cyst; maxillary canine region is the most common site for AOT.

Dentigerous cyst radio graphically appears as the radiolucency attached to the cementoenamel junction; in case of AOT the radiolucency extends more apically beyond Cemento enamel junction as seen in our case.

CEOC (Calcifying epithelial odontogenic cyst) is most commonly seen in either maxilla or mandibular region anterior to first molar of young adults, not associated with an impacted tooth, radiographically unilocular radiolucency with internal calcification are seen. Radicular cyst also can be considered in the differential diagnosis because, pulpal vitality test shows no response i.r.t 83, and the periodontal ligament and lamina dura were not found to be intact around involved teeth as seen in our case. Radiographically radicular cyst presents as a well defined unilocular radiolucent area, thin rim of cortical bone, larger than 1.5cm, with displacement of adjacent teeth. In our case shows buccolingual cortical expansion with well defined unilocular radiolucency approximately

about 4cm with displacement of adjacent teeth. Most common site for radicular cyst is maxillary anterior region but in our case it was found in mandibular anterior region and on aspiration it yields yellow color pus discharge, but in our case it yielded straw color fluid.

Central giant cell granuloma (CGCG) is an aggressive lesion commonly seen in females younger than 30years; mandibular anterior region is the common site, crossing midline. Radiographically it shows unilocular or multilocular radiolucency with wispy septae, displacement of teeth and root resorption are evident [6]. AOT is usually well – encapsulated, so conservative enucleation and currettage is the most common treatment modality for this tumor, recurrence is extremely rare. Care full follow -up examinations should be conducted in the case [3, 8, 10].

CONCLUSION:

It should be emphasized that careful diagnostic procedures and adequate interpretation of clinical and radiographic findings and differential diagnosis can be listed out to arrive at a correct diagnosis as in our case the AOT is mimicking dentigerous cyst. The final diagnosis of an AOT was arrived by histologic examination.

Unusual findings seen in our case related to typical features of an adenomatoid odontogenic tumor (AOT).

Typical features of most AOT	**Unusual findings in our case**
Most commonly seen in females, maxillary canine being the most common site [5].	Seen in male patient with manbibular (35%) Canine region.
Cortical plate penetration rare size usually does not exceed 1-3 cm [3,7].	Cortical expansion is seen with AOT Size exceeded more than 4 cms.
Unilocular radiolucent area with radiopaque specks Associated with impacted teeth [4].	Unilocular radiolucent area without radiopaque specks (seen in only 27% of cases).
Root resorption extremely rare; only four cases Reported to date to our knowledge [7 – 10].	Root resorption of the over-retained deciduous canine, with displacement of adjacent teeth (premolars).

Figures and figure legends:

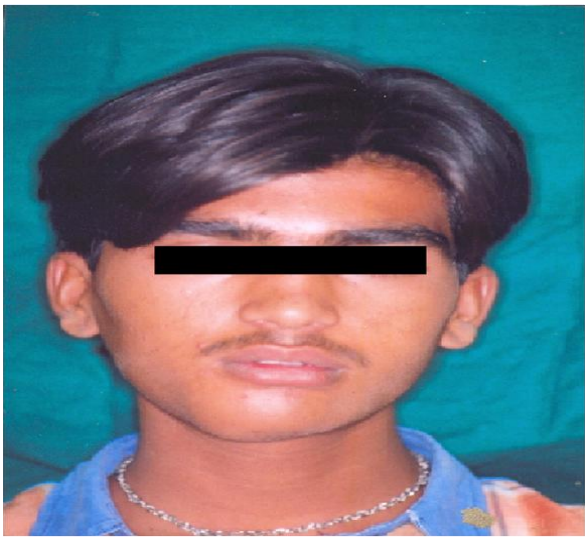

Figure 1:

Extra oral photograph of the patient

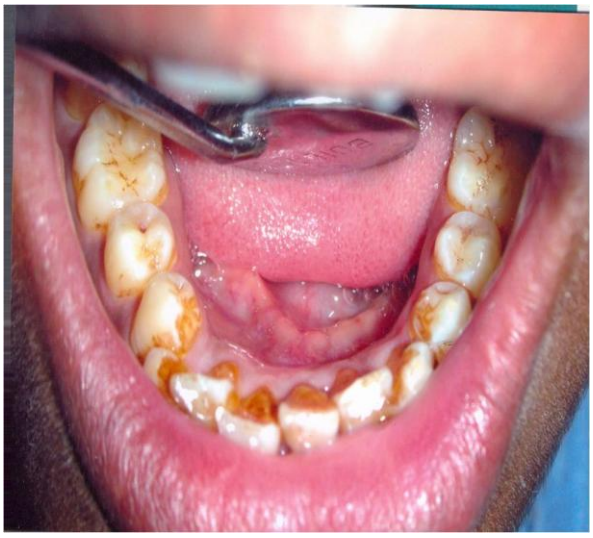

Figure 2:
Intra-oral photograph showing lingual swelling in the anterior region of the mandible

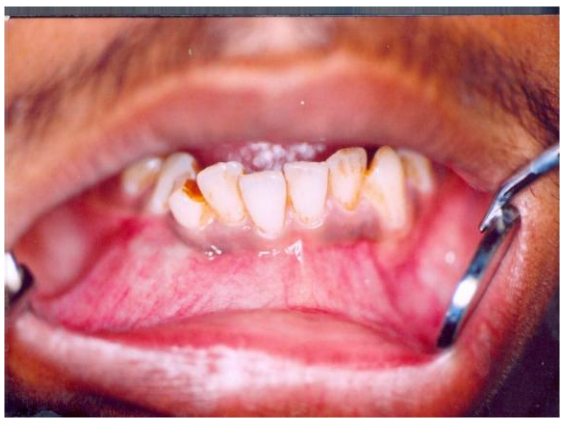

Figure 3:
Intra-oral photograph showing lower labial vestibular obliteration

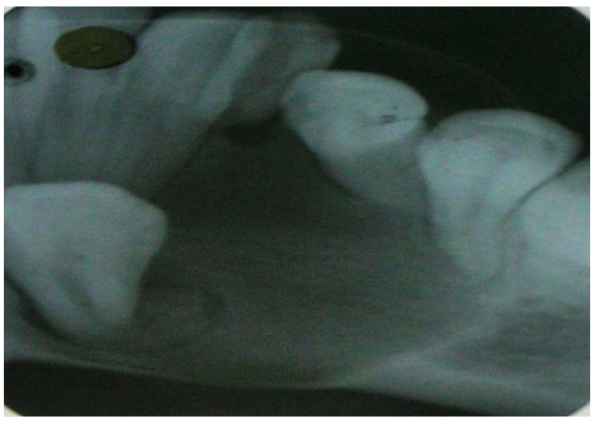

Figure 4:
Patient Intra-oral Periapical radiograph showing well defined radiolucency with impacted dilacerated canine. Also notice displaced premolars

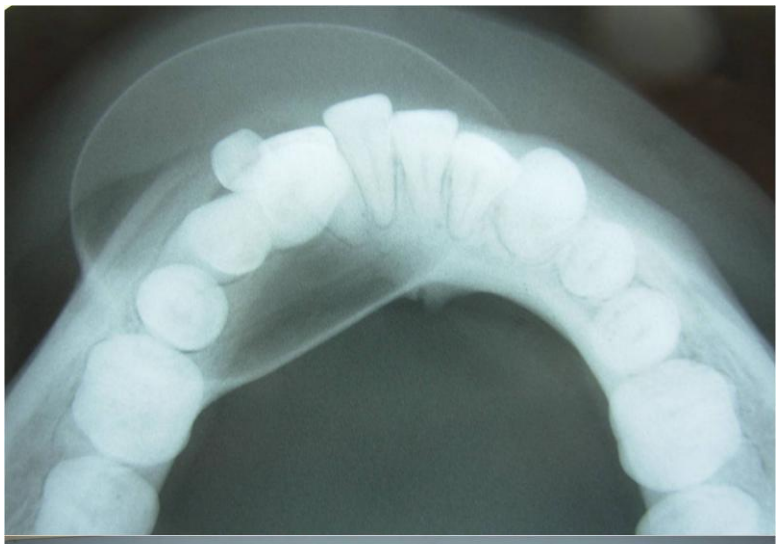

Figure 5:
Mandibular occlusal radiograph showing buccal and lingual cortical expansion with no cortical perforation

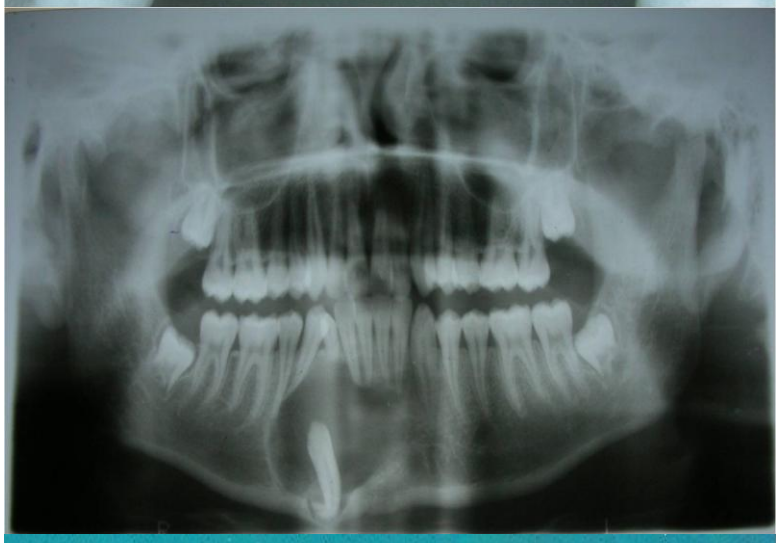

Figure 6:
Orthopantomogram showing well defined radiolucency with impacted canine and displaced premolars, overretained deciduos canine

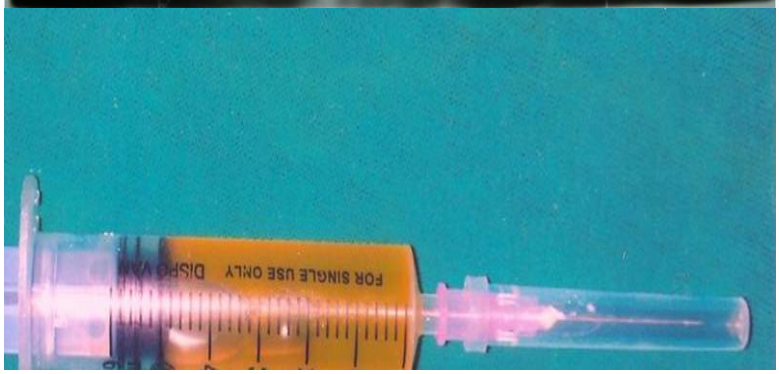

Figure 7: Fine needle aspiration cytology showing straw coloured aspirate

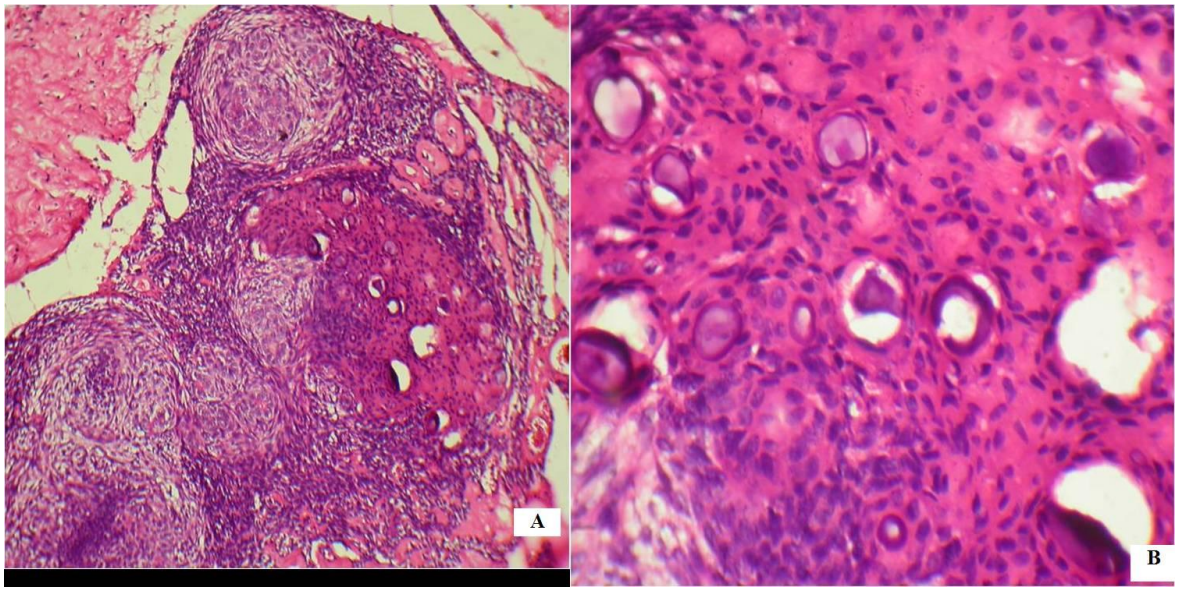

Figure 8: Pictomicrograph 40X magnification (A) and 100X magnification (B) (H & E). Showing odontogenic epithelium proliferating in the form of whorls with rosset like pattern, showing globular calcifications, duct like areas are seen with hyperchromatic epithelial cells suggestive of adenomatoid odontogenic tumor

REFERENCES:

1. Philipsen HP, Birn H. The adenomatoid odontogenic tumor; Ameloblastic adenomatoid tumor or Adeno-Ameloblastoma . Acta Pathol Microbial Scand 1969; 75; 375-98.
2. Pindborg J J, Kramer IRH. WHO international histological classifications of tumors.no.5 Histological typing of odontogenic tumors, jaw cysts and allied lesions.1st ed. 1971, Berlin; springer-verlag.
3. Philipsen HP, Reichart PA, Zhang KH,Nikai H, Yu Qx. Adenomatoid odontogenic tumor; biological profile based on 499 cases. J Oral Pathol Med 1991; 20; 149-58.
4. Philipsen HP, Reichart PA. The adenomatoid odontogenic tumor(AOT); Facts and figures. Oral Oncol 1998;35;1-7.
5. Philipsen HP, Reichart PA, Nikai H. The adenomatoid odontogenic tumor (AOT); An update. Oral med pathol 1997;2;55-60.
6. Stuart C. White, Michael J. Pharoah; Oral radiology; 6th edition; Benign Tumors of the jaws; Chapter 22; 383-385.
7. Dayi E,Gurbuz G,Bilge OM,Ciftcioglu MA. Adenomatoid Odontogenic tumor (adenoameloblastoma). Case report and review of the literature. Aust Dent J 1997; 42; 315-8.
8. Nomura M, Tanimoto K, Takata T, Shimasato T. Mandibular adenomatoid odontogenic tumor with unusual clinicopathologic features. J Oral Maxillofacial Surg 1992; 50; 282-5.
9. Nigam S, Gupta SK, Chaturvedi KU. Adenomatoid odontogenic tumor- A rare cause of jaw swelling. Braz dent J 2005; 16; 251-3.
10. Chuan-Xiang Z, Yan G. Adenomatoid odontogenic tumor; A report of a rare case with recurrence. J Oral Pathol Med 2007; 36; 440-3.

SECOND TO FOURTH DIGIT RATIO (2D:4D) IN MEN ATTENDING INFERTILITY CLINICS IN AKURE METROPOLIS NIGERIA: A PREDICTIVE INDEX?

Richard Bamidele Obe* and Oshiozokhai Eboetse Yama**

***State specialist Hospital, Akure, Owoyemi Specialist Hospital Akure, Nigeria**

****Department of Anatomy, Faculty of Basic Medical Sciences, University of Lagos, Idi-Araba, Lagos, Nigeria**

****Correspondence Author:** E-mail: dro_yama@yahoo.com.

Running title: Digit ratio a predictive index: case study of infertile men in Akure, Nigeria

ABSTRACT:

The ratio of index finger length to ring finger length is called the "2D:4D digit ratio," or more simply, the "digit ratio". This study was to investigate if there are significant differences in the digit ratio (2D:4D) of infertile men attending an infertility clinic and men drawn from the general population in Akure Nigeria; to generate data locally to serve as a source for future referencing in anthropometry as it relates to male fertility assessment. A total of 84 participants were involved in this study. They include men attending an infertility clinic (n=42), and those drawn randomly from the general population (n=42) with regards to their fertility. Information on 2D:4D and the seminal fluid data from two samples were obtained. Direct digit estimates was done using digital calipers and indirectly by taking measurements from a digital image of the hand. The digit ratios were obtained by dividing the lengths, of the index finger by the ring finger. Semen was collected from each participant by masturbation and examined for count and motility to ascertain their fertility status. There was a statistically significant ($p < 0.05$) increase in the length of the fourth digit compared to the second digit in fertile men. The 2D:4D ratio in fertile men was significantly lower ($p < 0.05$) compared to infertile men. This study demonstrates an association between 2D:4D ratio and the fertility status in adult men in Akure metropolis Nigeria.

Key words: Digit ratio, Infertility, Index finger, Ring finger, Seminal fluid

Received: May 2011: Accepted August 2011.

INTRODUCTION:

The associations between the second and fourth digits of the hand (the 2D:4D ratio) and fertility-associated traits probably arise from early organizational effects of testosterone rather than from activational effects of current testosterone [1].

The ratio of the lengths of 2D:4D has received considerable attention as a possible marker of the prenatal effects of androgen on the developing fetus [2]. Men average lower on this measure than women. In studies done, it was reported that, for males, the index finger is generally of about 96 % the length of the ring finger. This gives an average digit ratio of 0.96 for males [2]. The digit ratio is equal to 1.00 if length of 2D equal to 4D and greater than 1.00 if 2D were longer than 4D. Males generally have a digit ratio below 1.00 hence they have what is termed a "low digit ratio" that is 4D longer than 2D ring.

The digit ratio is a normal sexual dimorphic anatomic trait [3] determined at the 14th gestational week, and relatively stable throughout development [4 - 6]. The 2D:4D is assumed to be an 'indicator' of circulating prenatal gonadal hormones; smaller ratio reflects higher fetal testosterone and lower fetal estrogen. The suggestion that 2D:4D is a correlate of prenatal testosterone and estrogen was first made in 1998 by Manning, *et al.* [2]. In their study evidence was shown, that higher levels of testosterone during this critical developmental stage facilitates the growth of the ring finger, while higher levels of estrogen facilitates the growth of the index finger. It also appears that the right hand is affected more by these hormonal levels than the left hand such that length differences are more pronounced on the right hand. Averaged across samples from various populations, female values were found to be about 0.25 standard deviations higher than male values [7].

Several investigative methods are being routinely deployed in evaluating male patients with infertility. Notable amongst these are seminal fluid analysis, hormonal profile, and testicular biopsy [8]. These methods are usually met with diverse constraints. For example, the collecting seminal fluids samples require masturbating which has serious cultural/religious biases. The hormonal profile on the other hand is very expensive, testicular biopsy is an invasive procedure and requires expertise. If low digit ratios in men are associated with high sperm counts and testosterone levels and vice versa [2], it therefore follows that its use as a means of assessing infertility is invaluable.

Apart from the fact that the use of anthropometry offers a cheap, simple and easily repeatable means of evaluating fertility in men, there are scanty literature on this study and almost no data for males in the African continent. This present study was thus designed to determine the association of digit

ratio and infertility in Akure men as a possible predictive guide for male fertility.

SUBJECTS AND METHODS:

A total of 84 subjects were recruited for this study. They subject included 42 men attending infertility clinics in Akure metropolis in Nigeria. The sampling sites were randomly selected infertility clinics in Akure. They include the State Specialist Hospital, Hope Specialist Hospital and Owoyemi Specialist Hospital. All the participants attending these clinics did semen analysis to confirm their infertility. Patients were then diagnosed as either azoospermic (absence of sperm cells) or oligospermic (< 20 $x10^6ml^{-1}$) following two separate semen analyses [9]. The normative participants comprised 42 healthy fertile, married men that had no problems with ejaculation and sex life and has been able to father a child. They were contacted via their family doctor. The seminal fluid analysis was done in each of them to confirm the normospermic ranges (> 20-120 $x10^6ml^{-1}$) [9].

Informed consent was obtained from the authorities of the above named hospitals. The participants were duly informed of the purpose of the study and a signed consent was obtained from each of them. The protocol was approved by the local ethical committee.

We excluded inappropriate candidates through history taking and physical examination. The 'disqualified entrants' were those who had a history of cryptochidisms, varicoceles, or testicular injury which can have an influence on semen analysis as well as those who had burns or trauma on hands that can have an influence on finger length [10]. We also excluded the following short comings: males with females co-twin [11].

Since 2D:4D ratios vary greatly between different ethnic groups [12], we excluded other tribes (concentrated on the Yoruba's Western Nigeria where the hospitals were located).

After due counseling was done, informed permission was obtained. The participants were asked to sit comfortably and positioned the dorsum on a flat, smooth surface the right hand was used because relations with right-hand ratio are typically stronger than left hand [2]. The vernier caliper was the instrument used for direct measurement of digital lengths.

First we measured the digit lengths from the ventral crease proximal to the palm to the tip of the finger, using vernier calipers recording to 0.01 mm [13]. A second measurement was taken in order that repeatability of the 2D:4D ratio [12] could be calculated. All the measurements were made by one observer with right and left hands measured first and this procedure repeated after a period of at least 5 minutes blind to first measurements. The 2D:4D ratio was calculated by dividing the

length of the index finger by the length of the ring finger.

The length of the ring and the index finger was also alternatively estimated indirectly by taking measurements from a digital image of the hand [14] and data compared.

Results were expressed as mean ± standard deviation. Analysis was carried out using analysis of variance (ANOVA) with Scheffe's post hoc test. The level of significance was considered at $p < 0.05$.

RESULTS:

The result in Table 1 shows that there was a statistically significant ($p < 0.05$) increase in the length of the fourth digit as compared to the second digit in fertile men in Akure.

The digit ratio is less than one, since it is expressed as the ratio of the value of the length of the index finger in centimeters to the value of the length of the ring finger in centimeters. This means that most fertile men randomly sampled in Akure had their fourth fingers longer than the second finger.

Table 2 shows a significant ($p < 0.05$) increase in the length of the ring finger (fourth digit) as compared to index finger (second digit) in infertile men in Akure. In both cases the digit ratio was less than one. The values of 2D, 4D and digit ratio in fertile men were slightly but significantly ($p < 0.05$) lower compared to those of infertile men.

Data in Table 2 also shows that 2D:4D ratio in fertile men was significantly lower compared to infertile men, (unpaired *t*-tests, 2D:4D for infertile men, x = 0.954, 2D:4D for fertile men, x =0.946, *t*-test =2.61, $P r > t = 0.011$).

There was a significant difference for the digit ratio summary score, with greater digit ratio in infertile men compared to fertile men.

Table 3 shows the correlations between age, index finger, ring finger, and digit ratio of both the fertile and infertile men. The age of the participants were found to be positively correlated with 2D, 4D, and the digit ratio i.e. as the age increases, the values of 2D, 4D, and the relative digit ratio increases.

Table 1: Mean values for fertile (MF) and infertile (MIF) men in Akure

Variables	MIF	MF	MIF	MF	MIF	MF
	Minimum		Maximum		Mean ± SD	
Age	25.00	25.00	53.00	42.00	33.14±0.99	30.83±0.56
2D	6.43	6.13	8.26	8.16	7.63±0.05	7.31±0.07
4D	6.70	6.32	8.79	8.68	8.01±0.06	7.73±0.08
Digit ratio	0.93	0.90	0.98	0.98	0.95±0.002	0.94±0.002

Table 2: Status of 2D, 4D, and digit-ratio in fertile and infertile men in Akure

Status	2 D	4 D	Digit ratio
Infertile	7.63	8.01	0.954
fertile	7.31	7.73	0.946
T-test	3.61	2.71	2.61
P r > t	0.0005	0.0082	0.011
	$p < 0.01$	$p < 0.01$	$p < 0.05$

Table 3: Status of 2D, 4D, and digit-ratio in fertile and infertile men using t-test

	Age	2 D	4 D	Digit ratio
Age	1.00	0.18	0.14	0.13
		0.11	0.21	0.26
2 D	0.18	1.00	0.97	- 0.07
	0.11		< 0.0001	0.52
4 D	0.14	0.97	1.00	- 0.31
	0.21	< 0.0001		0.0042
Digit ratio	0.13	- 0.07	- 0.31	1.00
	0.26	0.52	0.0042	

DISCUSSION:

The assessment of fertility in men using relative digit lengths (2D:4D) ratio is not popular in African settings with scanty documentations if any. The efficacy in terms of percentage reliability in evaluating male fertility has been verified to be statistical significant [15]. Findings from our initial unpublished pilot study (before commencement of this current research) were interesting and the data was worthwhile.

The digit ratio is arguably ubiquitous in appraising many anthropometric limits in relation to other clinical conditions. For instance some authors have reported significant correlations between 2D:4D and such diverse traits as psychological, fertility, sexual attitudes and orientation, status, and cognitive abilities [16, 17].

Our study provided further evidence that 2D:4D is a sexually dimorphic trait with the normative 'control' participants having a significantly lower digit ratio compared to their infertile counterparts.

The data were less than one as in the fertile men which were in tandem with those described previously in other studies [2, 15].

The overall sample data showed a weak but significant positive correlation between 2D:4D and age, although more data are required to clarify this situation. However, at present it appears that the relationships were either weak or non-existent. This finding provides some support to our prediction concerning a positive association between 2D:4D and male fertility.

However a study has shown an inverse relationship between digit ratio and semen quality [2]. This means that the lower the digit ratio, the higher the sperm count and motility and the more fertile the individual. Our study correctly identified with the variations in the finger ratio in fertile and infertile men and vertical association to their sexual status in the population. Manning *et al.* have presented the negative relationship between the 2D:4D ratio and sperm number, motility and testosterone concentrations [2, 12]. In their study, they had a subset of oligospermic males, which reduced the overall mean sperm numbers accounting for the significant relationship between sperm number and 2D:4D ratio [2]. In the same vein, our study showed meaningful findings where oligospermic males had higher digit ratio. Thus, the 2D:4D ratio is a likely predictive value for men's semen quality in the Yoruba populace of Akure Nigeria.

This study shows that the influence of finger ratio is significant in the evaluation of fertility in men. This means that in addition to semen analysis, hormonal profile and environmental/sex life of an individual [8] a more influencing factor would be the digit ratio.

CONCLUSION:

This study demonstrates an association between 2D:4D ratio and the fertility status in adult men in Akure. Our data have provided a fertility predictive information/data on the relative digit lengths (2D:4D) ratio in men in Akure capital city of Ondo state Nigeria. This could serve as a future template for comparative studies on male fertility. However, there still exist controversies over this subject of relation between finger ratio and male fertility.

Therefore the need for a larger scale study that requires a greater comparative study design is inevitable; and also to demonstrate further correlations between the finger ratio and male fertility.

ACKNOWLEDGEMENTS:

We wish to thank the medical directors of State specialist Hospital, Akure, Owoyemi Specialist Hospital Akure, and Hope Specialist Hospital Akure for availing us the opportunity to use their patients. Also for their direction, questions, and suggestions, which have helped to develop and shape this project.

REFERENCES:

1. Manning JT, Stewart A, Bundred PE, Trivers RL (2004). Sex and ethnic differences in 2nd to 4th digit ratio of children. *Early Human Development* 80 (2): 161-168.
2. Manning JT, Scutt D, Wilson J, Lewis-Jones DI (1998). The ratio of 2nd to 4th digit length: a predictor of sperm numbers and concentrations of testosterone, luteinizing hormone and oestrogen. *Human Reproduction,* 13(11), 3000-3004.
3. George R. (1930). Human finger types. Anat. Rec. 46, 199-204.
4. Brown WM, Finn CJ, Breedlove SM. (2002). Sexual dimorphism in digit-length ratios of laboratory mice. *The Anatomical Record,* 267(3): 231-234.
5. McIntyre MH (2006). The use of digit ratios as markers for perinatal androgen action. *Reproductive Biology and Endocrinology* 4: 10.
6. Trivers R, Manning J, Jacobson A (2006). A longitudinal study of digit ratio (2D:4D) and other finger ratios in Jamaican children. *Horm. Behav.* 49 (2), 150-156.
7. Manning JT, Barley L, Walton J, Lewis-Jones DI, TriversSingh D. (2000). The 2nd:4th digit ratio, sexual dimorphism, population differences, and reproductive success evidence for sexually antagonistic genes *Evolution and Human Behavior* 21 (3): 163-183.
8. Baker HWG, 2008. Endocrinology of the Male Reproductive System Chapter 7 - Clinical Management of Male Infertility; Nature and causes of male infertility definitions, Updated, December ;2008,www.endotext.org/male/male7/maleframe7.htm
9. WHO (1999). World Health Organization Laboratory Manual for the Examination of Human Semen and Sperm-Cervical mucus Interaction. 4 Ed. 1999, Cambridge: Cambridge Uni Press.
10. Haepyoung S, Kyeon YK, Joon R. (2010). Is the Index Finger and Ring Finger Ratio (2D:4D) Reliable Predictor of Semen Quality? *Korean J Urol.* 51(3): 208-211.
11. Van Anders, SM, Vernon, PA, Wilbur CJ. (2006). Finger-length ratios show evidence of prenatal hormone-transfer between opposite-sex twins. *Horm. Behav.* 49, 315-319

12. Manning JT, Wood S, Vang E, Walton J, Bundred PE, van Heyningen C, Lewis-Jones DI (2004). Second to fourth digit ratio (2D:4D) and testosterone in men. *Asian J Androl.* 6:211-215.
13. Manning JT, Trivers RL, Thornhill R, singh D (2000). The 2nd:4th digit ratio and asymmetry of hand performance in Jamaica children. *Laterality* 5 (2): 121-132.
14. Gonzalez RC, Woods RE. (2002). Digital Image Processing, Prentice-Hall.
15. Manning JT, (2002b). Digit ratio: A pointer to fertility, behavior and health. New Brunswick, NJ: Rutgers University Press.
16. Wilson GD. (1983). Finger-length as an index of assertiveness in women. *Personality and Individual Differences* 4 (1): 111 - 112.
17. David AP, Steven JCG, Robert JS Donald HM (2004). Sex hormones and finger length what does 2D:4D indicate? *Evolution and Human Behavior,* 25; 1882-199.

PREVALENCE OF SELF-MEDICATION AMONG STUDENTS IN UNIVERSITY OF PAPUA NEW GUINEA

Gillian Meauri, Victor J. Temple* and Jackson AK Lauwo

School of Medicine and Health Sciences, University of Papua New Guinea, National Capital District, Port Moresby, Papua New Guinea

*** Correspondence Author: templevictor@gmail.com, templevj@upng.ac.pg.**

ABSTRACT:

The inappropriate use of over-the-counter (OTC) and prescription medicines to self-medicate can cause significant medical problems. This cross-sectional descriptive study assesses prevalence and factors associated with self-medication among students in the University of Papua New Guinea during the 2005 academic year. Data from randomly selected consented students on the Taurama and Waigani campuses of the University of Papua New Guinea were obtained by self-administered, structured, pre-tested questionnaires. The sample size for the two campuses was calculated using the "proportionate to population size" (PPS) cluster sampling technique. A total of 583 questionnaires were distributed as follows, 124 (21.3%) among students on Taurama campus and 459 (78.7%) among students on Waigani campus. However, only 309 (53%) of the questionnaires received from all the students were suitable for analysis. Data for all the 309 students indicate that OTC and Prescription medicines were

used in 710 instances (63.8%) and 402 instances (36.2%) respectively. Paracetamol was the most frequently (59.6%) used OTC medicine. Antibiotics (54.5%) and Antimalarials (45.5%) were the prescription medicines used for self medication. For antibiotics, Amoxicillin (89.5%) was the most frequently used. For antimalarial medicines, Chloroquine (47.5%) and Artemether (38.3%) were the most frequently used for self medication. Headache and malaria were the most common symptoms for self-medication, whereas the most common reasons were previous experience of treatment in relation to symptoms and mild illness. Sources of medicines for self-medication were friends (53.8%), pharmacy (52.6%) and supermarkets (43.1%). There was no significant difference in the inappropriate use of medicines by students on Taurama campus compared to those on Waigani campus. Self-medication practices were similar among students on both campuses. There is a need for intensive education and comprehensive awareness campaign to advocate for reduction in the prevalence of self-medication practices among students on both campuses in the University of Papua New Guinea.

Key words: Self-medication, Students, Over-the-counter, Prescription, Medicines

Received: April 2011; Accepted: July 2011.

(Special consideration: Partly published in proceedings of UPNG National Science Conference, 2009)

INTRODUCTION:

The use of medicines to treat self-diagnosed disorders or symptoms, or the intermittent or continued use of a prescribed medicines, without consultation with qualified medical practitioners, for chronic or recurrent disease or symptoms is considered as self-medication [1,2]. In most developed countries self-medication is a major component of the primary health care system. Rational use of over the counter (OTC) medicines can be achieved by appropriate labelling, information leaflets and also by ensuring that Pharmacists or Physicians give all necessary additional information or professional advice to consumers [1 – 3].

The use of prescription medicines for self-medication is common practice in some developing countries [2 – 6]. Some of the reasons for such common practice include non-licensed providers of medicines, availability of prescription medicines in open markets, actions of unregistered practitioners, use of leftovers, medicines obtained from family members or friends with previous similar symptoms [4 – 6]. There are also reports of increased and sometimes irrational use of OTC medicines in some developing countries [3 – 6]. This practice is on the increase among the poor socio-economic status groups, because of several reasons which include lack of modern healthcare facilities in rural areas, difficulties of

accessing these facilities in urban areas, non-availability of doctors, high cost of private medical care and deviation from daily schedule caused by waiting in long queues [4 – 6].

In developing countries, such as Papua New Guinea (PNG), self-medication may pose a threat to public health unless the population is adequately educated on responsible self-medications, so as to avoid problems associated with irrational medicine use. PNG has a fair share of Heath Service problems, with reports indicating that anti-malarial medicine resistance and resistance to some antibiotics are on the increase [7]. This indicates the possibility of existing irrational use of these medicines, including their indiscriminate non-prescription use.

The Medicines and Cosmetic Act 1999 of PNG as amended in the Medicine and Cosmetics Regulations 2001; lists 1070 Prescription Only Medicines, 334 Pharmacy Only Medicines and 493 OTC medicines [8]. There are no published data indicating awareness of the OCT and Pharmacy only medicines that are available for self-medication in PNG. In addition, there are no data to indicate the prevalence and factors associated with self-medication among the various communities in PNG. More specifically, there are not data on the pattern of self-medication among students in the various universities in PNG. This calls for appropriate research to establish the extent of self-medication in communities in PNG, so as to develop appropriate strategy to address the problems.

The University of Papua New Guinea (UPNG) is the premier university in PNG. There are five Schools, offering various degree programs in UPNG. The School of Medicine and Health Sciences (SMHS), located in the Taurama campus of UPNG, offers degree programs in Medicine, Pharmacy, Dentistry and other related medical courses. The other four Schools are located in the Waigani campus of UPNG.

The aim of this study was to assess the prevalence and factors associated with self-medication among students in the UPNG. The objective was to ascertain the types of medicines used; the sources of medicines and medicine information, and also the common symptoms for which the medicines were used for self-medication.

METHODS:

Collection of data for this descriptive cross-sectional study was conducted during the 2006 academic session, in the UPNG, which is one of the four major universities in PNG. The study population included all the 2160 full time undergraduate students, excluding all foundation year students. The duration of the UPNG academic session is 30 weeks.

A calculated total sample size of 550 students was obtained based on a design effect of two, a relative precision of 10%, confidence level (CL) of 95% and predicted non-response rate of 20%. As there was no available information on likely prevalence rate of self-medication in PNG, an assumed prevalence rate of 25% was used. The sample size for the two campuses was calculated using the "proportionate to population size" (PPS) cluster sampling technique.

The identification number (ID) for each of the 2160 full time students registered in the Taurama and Waigani campuses of the UPNG were obtained from the Executive Officers in each of the five schools. These numbers were further confirmed in the academic office in Waigani. The computer-generated random numbers were obtained and each number was randomly allocated to individual student using the ID numbers. The computer generated random numbers were used for selection the students to participate in the study. This was carried out using the standard procedures (9) as follows; the sample interval (k) was calculated and used as the starting point in the random number table. Each number selected randomly was then allocated to a questionnaire which also contains the ID number of a student. The final list obtained was then separated according to the ID numbers of the students, Taurama and Waigani campuses. Thus, a total of 620 full-time students, 124 (20%) on Taurama campus and 496 (80%) on Waigani campus were selected. This amounted to about 10% over sampling of the students on both campuses. The questionnaires were given to those students whose ID numbers were selected using the sample interval. The purpose of the study was explained to each student before giving them a written consent form to read and sign. Questionnaire was given only to students that signed the consent form. To guarantee confidentiality, names were omitted from both the consent form and the questionnaire.

Self-administered, structured, pre-tested questionnaire was used to collect information on age, sex, type and reasons for medication, self-use of medication during the 2005 academic year, names and doses of self-prescribed medicines, duration of use, sources of the medicines, knowledge of OTC and prescription medicines, and reasons for not consulting a doctor. The questionnaire was pre-tested for content and designed using a different cohort of 50 students, selected randomly from both campuses.

Ethical clearance for the study was obtained from the School of Medicine and Health Sciences, UPNG Ethical and Research Grant Committee. Data analysis was by Statistical Package for Social Sciences (SPSS-PC Software, Version 11). Chi square test was used to assess significance amongst variables.

A p-value of < 0.05 was considered as significant. The data are presented for all the students as a group and for students on the Taurama campus and Waigani campus.

RESULTS:

A total of 620 students from UPNG were recruited for the study, of which 583 (94%) consented to participate by signing the consent form. The 37 (6%) students that did not sign were on the Waigani campus. Thus, a total of 583 questionnaires were distributed as follows, 124 (21.3%) among students on Taurama campus and 459 (78.7%) among students on Waigani campus. However, only 309 (53%) of the questionnaires received from all the students were suitable for analysis. Thus, the total consent rate, 309 of the 620 students recruited, was 49.8%. The age range of all the consented students was 19 – 49 years. Gender distribution of these students was 148 (47.9%) males mean age 23.6 ± 4 years, and 161 (52.1%) females, mean age 21.9 ± 2.1 years.

Further analysis of questionnaires from the 309 students indicated that 85 (27.5%), mean age 22.4 ± 2.4, years were from Taurama campus and 224 (72.5%), mean age 22.8 ± 3.6 years, were from Waigani campus. Gender distribution of the students on Taurama campus was, 36 (42.4%) males with mean age 22.9 ± 3.2 years and 49 (57.6%) females with mean age 22.1 ± 1.5 years; on Waigani campus was 112 (50%) males, with mean age of 23.8 ± 4.2 years and 112 (50%) females, with mean age of 21.9 ± 2.3 years.

Of the 309 students, 253 (82%), mean age 22.7 ± 3.4 years, indicated that they had used self-medication during the 2005 academic year. Of these students, 90% (228) indicated regular use of self-medication, while 10% (25) occasionally self-medicated. Of the 253 students that self-medicated, the highest prevalence (84%) of self-medication was among the students in the 20 – 24 years age group, followed by students (9.5%) in the 25 – 29 years age group. Gender distribution of all the students that self-medicated was 117 (46%) males, mean age 23.6 ± 4.1 years, and 136 (54%) females, mean age 22.0 ± 2.2 years. This indicates that 79.1% of the male and 84.5% of the female students self-medicated. Thus, there was no statistically significant difference ($p > 0.05$) between the male and female students that self-medicated.

When the 253 students that self-medicated, were distributed according to campuses 30% (76) were on Taurama campus and 70% (177) on Waigani campus. This indicated that 89.4% and 79.0% of students on Taurama and Waigani campuses respectively self-medicated during the academic year. This difference was not statistically significant ($p > 0.05$).

The 253 students used various medicines for self-medication. Medicines were consumed in a

total of 1112 instances, which gives an average medicine consumption rate of 4.4 instances per student during the 30 weeks duration of the 2005 academic Session.

The OTC and Prescription medicines were used in 63.8% (710) and 36.2% (402) respectively of the 1112 instances (Table 1). Paracetamol used in 423 (59.6%) instances was the most frequently used OTC medicines, followed by other non-steroidal antiinflammatory medicines (NSAIDs) used in 122 (17.2%) instances. Cough and cold remedies were used in 96 (15.5%) instances. The use of other OTC medicines, such as, antacids, heat-rub, lozenges and worm tablets was relatively low (69 instances, 9.7%). Gender distribution (Table 1) of the frequency of OTC medicines consumption pattern indicates that the female students consumed more (402 instances, 56.6%) than the male students (308 instances, 43.4%). This difference was however, not statistically significant ($p > 0.05$).

Further distribution (Table 1) of the 710 instances indicated that the students on Taurama and Waigani campuses consumed OTC medicines in 193 (27.2%) and 517 (72.8%) instances respectively. This gives an average medicine consumption rate of 2.5 and 2.9 instances per student in Taurama and Waigani campuses respectively. This difference was not statistically significant ($p > 0.05$). Paracetamol was the most frequently used OTC medicine among students on both campuses.

Of the 402 (36.2%) instances that all the students used prescription medicines, antibiotics were the most frequently used, 219 instances (54.5%), followed by Antimalarials used in 183 instances (45.5%). In the antibiotic group, Amoxicillin (196 instances, 89.5%) was the most frequently used, followed by Septrin (17 instances, 7.8%), Chloramphenicol (four instances, 1.8%) and Penicillin (two instances, 0.9%). In the antimalarial group, Chloroquine (87 instances, 47.5%) was the most frequently used, followed by Artemether (70 instances, 38.3%), Fansidar (22 instances, 12.0%), Quinine (two instances, 1.1%) and Primaquine (two instances, 1.1%). There was not significant difference ($p > 0.05$) in the frequency of usage of prescription medicines among the female (204 instances, 50.7%) students, compared to male (198 instances, 49.3%) students.

Analysis of the data indicated that of the total 1112 instances, the medicines were used appropriately in 931 (83.7%) instances compared to their inappropriate use in 181 (16.3%) instances by all the students. Of the 181 inappropriate instances, the OTC medicines were used inappropriately in 21 instances (11.6%) compared to 160 inappropriate instances (88.4%) for prescription medicines. Antibiotics and antimalarial medicines were used in 121 (75.6%) and 33

(20.6%) instances respectively of the 160 inappropriate instances of medicines used by all the students. Amoxicillin (101 instances, 83.5%) was the most frequently abused antibiotic, while Chloroquine (33 instances, 51.5%) was the most frequently abused antimalarial medicine. Inappropriate use of the medicines was higher among the male students (53.6%) compared to the female students (46.4%). The difference was however, not significant statistically.

Further analysis of the distribution of the 402 instances that prescription medicines were used indicates usage of 36.2% (132 instances) by students on Taurama campus compared to 270 instances (67.2%) by the students on Waigani campus, (Table 1). This is equivalent to an average drug consumption rate of 1.7 instances per student on Taurama campus compared to 1.5 instances per student on Waigani campus. This difference was not statistically significant ($p > 0.05$). In both campuses, Amoxicillin was the most frequently used antibiotic, whereas Chloroquine was the most frequently used antimalarial medicines. There was not significant difference in the inappropriate use of medicines by students on Taurama campus compared to those on Waigani campus.

Table 1: Medicines / Medicine groups used by students for self-medication

Medicines/Medicine groups	**Instances (%) of usage in University of PNG**			**Instances (%) of usage in University PNG Campuses**	
Over the counter Medicines (OTC)	**Total students**	**Females**	**Males**	**Taurama**	**Waigani**
Paracetamol	**423 (59.6%)**	**249 (61.9%)**	**174 (56.5%)**	**126 (65.3%)**	**297 (57.4%)**
Other NSAIDs	**122 (17.2%)**	**64 (15.9%)**	**58 (18.8%)**	**22 (11.4%)**	**100 (19.3%)**
Cough & Cold remedies	**96 (15.5%)**	**57 (14.2%)**	**39 (12.7%)**	**20 (10.4%)**	**76 (14.7%)**
Others	**69 (9.7%)**	**32 (8.0%)**	**37 (12.0%)**	**25 (13.0%)**	**44 (8.5%)**
Total Instances	**710.0**	**402.0**	**308.0**	**193**	**517**
Prescription Medicines	**Total**	**Females**	**Males**	**Taurama**	**Waigani**
Antibiotics	**219 (54.5%)**	**117 (57.4%)**	**102 (51.5%)**	**74 (56.1%)**	**145 (53.7%)**
Antimalarials	**183 (45.5%)**	**87 (42.6%)**	**96 (48.5%)**	**58 (43.9%)**	**125 (66.3%)**
Total Instances	**402.0**	**204.0**	**198.0**	**132.0**	**270.0**

Table 2 shows the prevailing conditions for which the students self-medicated. Headache and malaria were the two major reasons for self-medication among all the students in UPNG and on both campuses. In most cases, self-medication for headache was commenced as soon as the pain started. Dizziness was the prevailing symptom for the commencement of self-medication for malaria. Although, according to most of the students, malaria was usually associated with fever, the onset of fever associated with body ache or joint pain was not related to malaria. Self-medication for cough and skin infections was higher among students on Taurama campus, compared to those on Waigani campus.

Table 2: Conditions for which students self-medicated *

	University of PNG			University of PNG Campuses	
Conditions	**Males (n = 117**	**Females (n = 136)**	**Total (n = 253)**	**Taurama (n = 76)**	**Waigani (n = 177)**
Headache	**92.3% (108)**	**92% (125)**	**92.1% (233)**	**92.1% (70)**	**92.1% (163)**
Malaria	**71% (83)**	**76.5% (104)**	**74% (187)**	**72.4% (55)**	**74.6% (132)**
Cough	**41% (48)**	**49.3% (67)**	**45.5% (115)**	**61.8% (47)**	**38.4% (68)**
Fever	**43.6% (51)**	**36.8% (50)**	**40% (101)**	**46.1% (35)**	**37.3% (66)**
Flu	**34.2% (40)**	**39.7% (54)**	**37.2% (94)**	**28.9% (22)**	**40.7% (72)**
Running nose	**22.2% (26)**	**28.7% (39)**	**25.7% (65)**	**18.4% (14)**	**28.8% (51)**
Back ache	**31.6% (37)**	**19.1% (26)**	**24.9% (63)**	**11.8% (9)**	**30.5% (54)**
Stomach ache	**17.9% (21)**	**27.2% (37)**	**23% (58)**	**18.4% (14)**	**24.9% (44)**
Sore throat	**23.1% (27)**	**22.1% (30)**	**22.5% (57)**	**22.4% (17)**	**24.9% (44)**
Ear ache	**8.5% (10)**	**14.7% (20)**	**11.9% (30)**	**6.6% (5)**	**14.1% (25)**
Skin infections	**15.4% (18)**	**7.4% (10)**	**11.1% (28)**	**15.8% (12)**	**9% (16)**
Diarrhoea	**4.3% (5)**	**4.4% (6)**	**4.3% (11)**	**6.6% (5)**	**3.4% (6)**
Vomiting	**2.6% (3)**	**2.2% (3)**	**2.4% (6)**	**2.6% (2)**	**2.3% (4)**
Others	**24.8% (29)**	**25.7% (35)**	**25.3% (64)**	**23.7% (18)**	**26% (46)**

* Figures indicate multiple responses; Total does not add up to 100%

The students were asked to indicate all the sources from which they obtained the various medicines they had used to self-medicate. Friends (53.8%), pharmacy shops (52.6%) and supermarkets (43.1%) were the major sources of medicines used for self-medication by all the students. The students indicted that they had visited pharmacy shops 133 times during the academic session.

Pharmacists were present during 47% of the times they visited pharmacy shops. Pharmacists were not present in any of the supermarkets during the time of their visits. Although this trend was similar for students on both campuses, the use of leftover medicines from previous visits to clinics was more prevalent among students (41.2%) on the Waigani campus, compared to 17.1% of students on the Taurama campus.

The students obtained information about the medicines used for self-medication from multiple sources. Most (65%) of the students indicated that the labels on packets and leaflets inside packets were the major sources of information about the medicines used to self-medicate. Previous prescriptions (54.5%), friends (37.2%), relatives (28.1%) and News media (13%) were the other sources of information. This trend was similar for students on both campuses.

When asked about the knowledge of side effects of some of the medicines that they used to self-medicate, 55.7% of the students had some knowledge of the side effects of some of the medicines, compared to 44.3% that had no knowledge of any side effects of any of the medicines. When distributed according to gender, 56.4% and 55.9% of male and female students respectively had some knowledge of the side effects of some of the medicines.

A total of 75% and 47.5% of students on Taurama and Waigani campuses respectively, indicated that they had some knowledge of the side effects of some of the medicines that they used to self-medicate. Thus, 25% of the students in Taurama campus were not aware of any side effects of the medicines used to self-medicate compared to 52.5% of the students on the Waigani campus.

The reasons for self-medication among all the students and students on each campus are presented in Table 3. Previous experience of treatment in relation to symptoms (73.5%) and mild illness (66.0%) were two of the major reasons for self-medication among all the students. The trend was similar among both male and female students.

Mild illness was the major prevailing reason for self-medication among 84.2% of students on Taurama campus, compared to 58.2% of

students on Waigani campus. On the Waigani campus 30.5% of students self-medicated because of long queues to doctors compared to 17.1% of students on the Taurama campus. It was more expensive for students on Waigani campus to see the doctors compared to students on Taurama campus.

Table 3: Reasons for self-medication among the students *

	University of PNG			University of PNG Campuses	
	Males (n = 117)	**Females (n = 136)**	**Total (n = 253)**	**Taurama (n = 76)**	**Waigani (n = 177)**
Previous experience of treatment in relation to symptoms	**79.5% (93)**	**68.4% (93)**	**73.5% (186)**	**81.6% (62)**	**70% (124)**
Mild illness	**67.5% (79)**	**64.7% (88)**	**66% (167)**	**84.2% (64)**	**58.2% (103)**
Long queues to doctors	**27.4% (32)**	**25.7% (35)**	**26.5% (67)**	**17.1% (13)**	**30.5% (54)**
Expensive seeing the doctors	**18.8% (22)**	**8.8% (12)**	**13.4% (34)**	**5.3% (4)**	**17% (30)**
Number of visits to clinics because of lectures	**18.8% (22)**	**14.7% (20)**	**16.6% (42)**	**15.8% (12)**	**17% (30)**

* Figures indicate multiple responses; Total does not add up to 100%

DISCUSSION:

The high (50.2%) total non-response rate obtained in this study highlights the already reported common problems in conducting surveys by questionnaires [2,3,4]. The non-response rate of 54.8% (272 students) was higher on Waigani campus compared to 31.5% (39 students) on Taurama campus. Reasons for the high non-response rate include failure to complete and return the questionnaire despite several attempts made to contact some of the students, incomplete questionnaires and difficulty in remembering the names of medicines, dosage and duration of use. This supports the observation [2,3, 4] that problems in recalling retrospective information, such as

types of medicines used and understanding the meaning of self-medication are major contributing factors to the low response rate from some consented participants. Although the total number of female students was slightly higher than that of male students, the difference was not statistically significant. There was no significant difference in the mean age of students that self-medicated on both campuses.

The 82% prevalence of self-medication amongst the students in UPNG was lower that the 94% and 88% reported among university students in Hong Kong [3] and Croatia [11] respectively, but higher than the 45% and 76% reported amongst university students in Turkey [10] and Karachi [6] respectively.

No statistically significant differences were observed in the mean ages and self-medication practices among the male and female students in UPNG. This finding was similar to that reported by some authors [3,6,12], but different from that of others [3,4,5,13], who reported significant differences in self-medication practices between male and female respondents.

The relatively higher but non-significant prevalence of self-medication practice among the students (89.4%) on Taurama campus compared to students (79.0%) on Waigani campus was similar to the data reported for medical and non-medical students in Karachi [6] and Sudan [13]. Our data do not strongly favour the idea that students in the medical sciences tend to self-medicate more than other students [6]. However, the set up and location of the two campuses in UPNG makes it difficult to explain the reason for the high prevalence of self-medication practices among students in the Taurama campus. This is because there is only one clinic for staff and student on the Waigani campus, compared to the Taurama campus, which, apart from having a clinic for students, is located in close proximity to the PMGH. In addition, students on Taurama campus have easy access to their teachers; some of them are qualified medical doctors, pharmacists, dentists and nurses. Most of these staff members offer free consultations to students without prior appointment. Despite this easy access the prevalence of self-medication was 89.4% among the students on Taurama campus. It seems plausible that the students in Taurama and Waigani campuses may not be fully aware of some of the implications of indulging in self-medication. There is therefore an urgent need to carry out intensive education, information and awareness campaign to advocate for reduction in the prevalence of self-medication among students on both campuses in UPNG.

Paracetamol and other NSAIDs were the most frequently used OTC medicines for self-

medication among the students in UPNG and on both campuses. This corresponds to the findings that headache was the commonest prevailing illness for self-medication among the students. This is similar to reports on university students by other authors [2 – 6]. Panadol, the most frequently used OTC medicine for self-medication, is a Paracetamol-based analgesic that is known to provide fast, effective temporary relief of pain and discomfort associated with headache, tension headache, migraine headache, muscle pain, backache, toothache, period pain, cold and flu symptoms [9]. Panadol is a much safer medicine to use than Aspirin [9]. It is the analgesic of choice for individuals with a sensitive stomach, and stomach ulcers [9]. The various brands of Panadol available in Port Moresby are sold as OTC medicines [9]. There was no statistically significant difference between the frequency of usage of Panadol by students in Waigani and Taurama campuses.

The calculated frequency (545 instances by all the 253 students) of use of paracetamol and other NSAIDs is equivalent to 2.2 instances per student over the duration of the academic year. This frequency is higher than the values reported by other authors [3,4]. This should be of concern because the abuse of OTC medications may produce unwarranted medical complications or interact with prescription medications. Thus, the need to educate the students on the appropriate use of the OTC medications cannot be overemphasized. The lack of awareness of the "hidden" ingredients in some OTC medicines may exacerbate existing medical problems in susceptible individuals [9].

The high frequency with which antibiotics and anti-malarial medicines were used for self-medication is a controversial issue that must be addressed because of the possibility of developing drug resistance, if the medicines are not properly used. Inappropriate use of antibacterial and antimalarial medicines, such as inadequate dosing, incomplete courses and indiscriminate usage, are the major contributing factors to the development and spread of drug resistance, particularly in developing countries [2,6].

Port Moresby is not an endemic zone for malaria, thus the indiscriminate use of anti-malaria medicines must be discouraged. It is therefore necessary to conduct regular advocacy on the potential benefits and risks associated with the use of Artemether and Artesunate, which are the currently available anti-malarial medicines in PNG and other developing countries including.

Prevalence of self-medication for diarrhoea and vomiting was very low, because most of the students consider these to indicate serious illness, which calls for a visit to the doctor. Those that self-medicated indicated that they

use commercially available oral rehydration solution, coconut water or rice water.

Friends, relatives and medicine retail shops are responsible for promoting self-medication among the students. The high percentage of students from Waigani campus that used leftover medicines from previous visits to the clinic indicates that either they had not taken the prescribed dose of the medication or that too much medication had been prescribed. The presence of pharmacists in all medicine retail outlets can play a significant role in controlling some of the factors that promote self-medication. The non-availability of pharmacists in most medicine retail outlets to give professional advice on the use of OTC medicines and to restrict the sale of prescription only medicines is a major issue that requires immediate attention by the health authorities in Port Moresby. Consumers of OTC medicines require proper access to accurate and clear information on the uses of these medicines. It is therefore important to ensure that all medicine labels and leaflets inside packets are written in simple English and in the local Tok Pisin language commonly spoken by people in PNG.

Comprehensive information on the side effects of OTC medicines should be included on information leaflets in the packets of medicines. In addition, intervention, such as distribution of information about side effects of OTC medicines should be carried out via media, health education sessions, posters and education councillors on the UPNG campuses. The current UNPG foundation year course on Drug Abuse and Misuse should include the effective use of self-medication.

There was no significant difference in the prevailing reasons for self-medication among all the students in UPNG. However, self-medication in the treatment of mild illness was higher among students on Taurama campus compared to students in Waigani campus. This indicates that students on Taurama campus are more aware of the concept of self-medication, as visiting the doctor is unnecessary for mild illnesses. On the other hand, using self-medication because of previous symptoms indicates insufficient knowledge of the concept of self-medication. In order to correct and consolidate the prevailing self-medication practices among the students on Taurama campus, it is important that students get a clear understanding of the concept of mild illness and learns about the appropriate medicines that should be used in these cases. Experts in the field of drug education and self-care practices should conduct intensive advocacy and education of proper self-medication practices among the general student population in UPNG. Increasing the number of doctors in the student clinic on Waigani campus can shorten the

queues to doctors, thus encouraging more students to refrain from self-medication.

CONCLUSION:

The prevalence of self-medication is high (82%) among students in the University of Papua New Guinea, with no significant difference between male and female students. There was no significant difference observed in the self-medication practices among the students on the Taurama and Waigani campuses.

Paracetamol and other NSAIDs were the most frequently used OTC medicines for self-medication. Amoxicillin was the most frequently used antibiotic, while Chloroquine was the most frequently used antimalarial medicine. Inappropriate use of medicines was higher among the male students (53.6%) compared to the female students (46.4%) in UPNG. Headache and malaria were the two major reasons for self-medication. Friends, relatives and medicine retail shops were responsible for promoting self-medication among the students. The presence of pharmacists in all medicine retail outlets can play a significant role in controlling some of the factors that promote self-medication among the students.

There is need to carry out intensive education and comprehensive awareness campaign to advocate for reduction in the prevalence of self-medication among students on the Taurama and Waigani campuses in the University of Papua New Guinea.

ACKNOWLEDGEMENT:

We gratefully acknowledge all the students especially those on the Waigani campus in the University of Papua New Guinea that participated in this project. We thank Samson Grant, Roxanne Komeng, Henry Jeremiah, Dinah Tetaga and Christie Tande for their various contributions towards the success of this project.

REFERENCES:

1. Responsible self-medication: Joint statement by The International Pharmaceutical Federation and The World Self-Medication Industry (WSMI). International Pharmaceutical Federation (FIP); June 1999; 1–3.
2. Abdelmoniem A, Eltayeb I, Matowe L, Thalib L. (2005) Self-medication with Antibiotics and Antimalarials in the community of Khartoum State, Sudan. J. Pharmaceut Sci. 8 (2): 326 – 331.
3. Lau GSN, Lee KKC, Luk CT. (1995) Self-Medication among University Students in Hong Kong. Asia Pac J Public Health; 8 (3): 153 – 157.
4. Shankar PR, Partha P and Shenoy N. (2002) Self-medication and non-doctor prescription practices in Pokhara valley, Western Nepal: questionnaire-based study. Bio Med Central Family Practice; 3: 17–23.
5. Buck ML. (2007) Self-medication by adolescents. Pediatr Pharm. 13 (5): 1 – 4.
6. Zafar SN, Syed R, Waqar S, Zubairi AJ, Vaqar T, Shaikh M, Yousaf W, Shahid S, Saleem S. (2008) Self-medication amongst university students in Karachi: Prevalence,

Knowledge and Attitudes. J. Pak Med Assoc. 58 (4): 214 – 217.
7. Joint Malaria Meeting 2004, Report. Edited by: ICRMC UPNG and Tokyo Women's Medical University. 7th to 9th June 2004, Port Moresby, NCD, PNG.
8. Hulley SB, Cummings SR, Browner WS, Grady D, Hearst N, Newman TB (2001): Choosing the study subjects: Specification, Sampling and Recruitment. In: Designing Clinical Research: An Epidemiologic Approach, 2nd Edition, Lippincott Williams & Wilkins, NY, pp 25 – 36
9. Medicines and Cosmetics Regulation, Papua New Guinea, National Department of Health. 2001: Sect 8; 57 – 98.
10. Buke C, Limoncu M, Ermevtcan S, Cicekliogu M, Tuncel M, Kose T. (2005) Irrational use of antibiotics among uni students. J. Infect; 51: 135 – 139.
11. Vucic VA, Trkulja V, Lackovic Z. (2005) Content of home pharmacies and self-medication practices in households of pharmacy and medical students in Zagreb, Croatia: findings in 2001 with a reference to 1977. Croat Med J. 46: 74 – 80
12. James H, Handu SS, Al Khaja KAJ, Otoom S, Sequeira RP. (2006) Evaluation of knowledge, attitude and practice of self-medication among first-year medical students. Med Princ Pract. 15: 270 – 275
13. Awad AI, Eltayeb IB. (2007) Self-medication practices with antibiotics and antimalarials among Sudanese undergraduate university students. Annals of Pharmacotherapy. Vol. 41, No. 7, 1249 – 1255.

NEONATAL HYPOGLYCAEMIA, RELATIVE PLACENTAL WEIGHT AND MATERNAL PRE-ECLAMPSIA: ANY RELATIONSHIP?

Alphonsus N. Onyiriuka* and Eugene M. Ikeanyi**

***Department of Child Health, University of Benin Teaching Hospital, Benin City, Nigeria;**

****Department of Obstetrics and Gynaecology, St Philomena Catholic Hospital, Benin City, Nigeria;**

***Correspondence author: E-mail:** alpndiony@yahoo.com, and didiruka@gmail.com

ABSTRACT:

Pre-eclampsia is known to be associated with various placental morphologic changes as well as fetal growth restriction. Growth restricted neonates are at increased risk of hypoglycaemia in the first three days of life. The aim of the study was to examine the relationship between occurrence of neonatal hypoglycaemia and the relative placental weight in mothers with pre-eclampsia. The blood glucose concentrations of 69 neonates born to mothers with pre-eclampsia were determined three times daily during the first three days of life. The birthweight of each of the neonates as well as the corresponding

weight of the placenta were determined and recorded. The relative placental weight was calculated using the formula: Weight of placenta x 100/Birthweight of the infant. Overall prevalence of neonatal hypoglycaemia was 47.8%. Of the 69 neonates, severe neonatal hypoglycaemia (blood glucose < 1.6 mmol/L) was prevalent in 10 (14.5%) and 15(21.7%) had blood glucose level between 1.6 and 2.5 mmol/L. The relative placental weight did not differ with the severity of maternal pre-eclampsia. No statistically significant correlation was obtained between the relative placental weight and neonatal hypoglycaemia.

Key Words: Hypoglycaemia, neonates, placental weight, pre-eclampsia;
Received: July 2011: Accepted September 2011

INTRODUCTION:

Neonatal hypoglycaemia (blood glucose concentration below 2.6mmol/L) is one of the common clinical care problems encountered in some neonatal units [1]. The frequency is influenced by the cut-off point used for blood glucose concentration [1, 2]. In a study in Nepal, when the cut-off point for blood concentration was below 2.6mmol/L the incidence was 41.0%; the incidence dropped when a cut-off point below 2.0mmol/L was applied [2].

Pre-eclampsia is an established pregnancy-associated clinical condition that leads to fetal growth restriction [3-5]. It has been suggested that fetal growth restriction in pre-eclampsia might depend on abnormal placental development [3, 6]. For instance, shallow trophoblastic invasion of decidual arteries can precipitate pre-eclampsia with the attendant reduction in placental perfusion and insufficient transport of nutrients to the developing fetus which in turn leads to fetal growth restriction [4, 7-9]. Vascular spasm in the placental bed is believed to cause placental infarction, resulting in deterioration in the metabolism between the mother and her fetus with fetal growth restriction as a consequence [8, 10]. Some investigators [11, 12] have reported occurrence of placental growth retardation in pre-eclampsia. Some of the principal pathological changes of the placenta in pre-eclampsia include decidual arteriopathy, infarcts, abruptio placentae and Tenney-Parker changes [11, 13]. All these pathological features are not always present [13]. Khong et al [14] in their study of 39 patients with pre-eclampsia reported that the placental lesions were unrelated to maternal parity, degree of proteinuria, severity and duration of hypertension, or its therapy. Considering the various placental morphologic changes in pre-eclampsia and the resultant fetal growth restriction, it may be surmised that the relative

placental weight could predict the likelihood of neonatal hypoglycaemia among infants born to mothers with pre-eclampsia.

The present study, therefore, sought to examine the relationship between occurrence of neonatal hypoglycaemia and relative placental weight in maternal pre-eclampsia.

PATIENTS AND METHODS:

All neonates delivered at St Philomena Catholic Hospital (SPCH), Benin City between January and December 2010 to Nigerian women with pre-eclampsia were recruited into the study. Ethical clearance and permission for the study were approved by the hospital authority. The mothers were given a detailed verbal explanation of the study and their permission sought before enrolment.

The criteria for diagnosis of pre-eclampsia and enrolment of the mother-infant pair into the study were:

An increase in either systolic or diastolic blood pressure greater than 30 mmHg or 15 mmHg respectively above the booking blood pressure (BP) plus proteinuria (using albustix) of one plus (1+) and above in the absence of urinary tract infection (UTI).

An intrapartum BP = 140/90 mmHg obtained on at least two occasions not less than 6 hours apart during delivery plus presence of proteinuria as indicated above. A single reading of 110 mmHg diastolic BP or more with proteinuria as indicated above was accepted as pre-eclampsia.

The Korotkoff sound phase 5 (disappearance phase) which is more reproducible, correlated better with intra-arterial measurements of diastolic BP and is more closely related to outcome was used [10].

Nigerian women who did not smoke or drink alcohol and had not been diagnosed with diabetes mellitus or sickle cell anaemia and were not on medication, such as propanolol or drugs such as narcotics were recruited into the study.

Gestational diabetes mellitus was excluded by routine determination of blood glucose concentration in all pregnant women attending antenatal care clinic in our hospital.

Pregnant women with random or fasting blood glucose concentrations below 8.0mmol/L or 6.0mmol/L respectively, were deemed to be free from gestational diabetes mellitus [15].

Those excluded from the study were mothers with eclampsia; Infants with rhesus isoimmunisation, polycythaemia and major congenital abnormalities; Infants of diabetic mothers and twins.

Pre-eclampsia was categorized into mild, moderate and severe according to the criteria suggested by Redman with some modifications [16]. In this Classification System, mild pre-eclampsia was defined as a diastolic BP increase of at least 30mmHg and proteinuria of 1+ (using albustix); moderate pre-eclampsia as

an increase in diastolic BP of at least 30mmHg and proteinuria of 2+; and severe pre-eclampsia as a diastolic BP increase of at least 30mmHg or a single diastolic BP equal or greater than 110mmHg and proteinuria of 3+.

In our hospital, the approved routine treatment of pre-eclampsia consisted of bed rest, anti-hypertensive drugs (Hydralazine, Methyl dopa) and sedatives (Diazepam) as determined by the patient's clinical condition.

Following delivery of the neonate, the umbilical cord was clamped and this was followed by delivery of the placenta at the appropriate time. The placenta with all its membranes was weighed and recorded.

The corresponding birthweight of each baby was measured to the nearest 50g using a Waymaster Weighing Scale and the value obtained was recorded. From the weights obtained, the relative placental weight was calculated using the formula:

$$\frac{\text{Placental weight} \times 100}{\text{Birthweight of baby}}$$

The relative placental weight was categorized into two groups; those equal or less than 15% and those greater than 15%. Blood glucose measurement was performed for each neonate three times daily for the first three days of life, using a Glucometer (Acutrend meter product 128485 with glucose test strips) which display results in mmol/L. Neonates whose blood glucose concentration was less than 3.0mmol/L had their blood glucose concentration confirmed in the central laboratory of the hospital, using the standard glucose-oxidase-perioxidase method [17]. If there was a single blood glucose concentration with value less than 2.0mmol/L, the tests were continued for at least one day after the blood glucose concentration has returned to normal or after the therapy had been discontinued. At the time of this study, breast feeding in the neonatal unit/newborn nursery of the hospital was routinely started about 1-2 hours after birth. Some of the babies were given pre-lacteal 5% glucose orally. When the blood glucose concentration of a neonate was less than 1.6mmol/L or a neonate was symptomatic (irrespective of blood glucose concentration), intravenous administration of 10% dextrose in water was started immediately. To ensure reliability of the results, discoloured strips were not used and care was taken to avoid contamination with alcohol skin-cleansers. It was also ensured that the drop of blood covered the whole surface of the test-pad. The packed cell volume of each of the study

neonates was also determined. The Chi-square test and Z-score test were used in ascertaining the significance of differences in proportions with the p-value set at <0.05.

RESULTS:

Seventy one (5.2%) of the 1,360 pregnancies delivered in the hospital during the one-year study period were complicated by pre-eclampsia. Clinical data of the mothers with pre-eclampsia are shown in Table 1. The distribution of cases into subgroups of mild, moderate and severe did not differ in relation to maternal parity. Two (2.8%) of the 71 neonates delivered by pre-eclamptic mothers were stillborn, leaving 69 live-born babies whose data were further analysed. The frequency of delivery of neonates with birthweight less than the 10th percentile increased with the severity of maternal pre-eclampsia (Table 2); X^2 =5.26 p>0.05. As shown in Table 3, the relative placental weight was greater than 15% in majority of cases. The severity of maternal pre-eclampsia did not influence the relative placental weight (Table 3).

Table 1: Clinical data of mothers according to severity of pre-eclampsia

Severity of pre-eclampsia	Percent (n)	Mean age in Years	Maternal Parity in percent (n)		
			Para zero	Para 1 – 4	Para ≥5
Mild	42.3 (30)	24.7	44.7 (17)	48.1 (13)	0
Moderate	23.9 (17)	26.3	23.7 (9)	22.2 (6)	33.3 (2)
Severe	33.8 (24)	26.8	31.6 (12)	29.7 (8)	66.7 (4)
Total	100 (71)	25.4	100 (38)	100 (27)	100 (6)

Table 2: Distribution of birthweight percentile of neonates according to severity of maternal pre-eclampsia.

Severity of Pre-eclampsia	Number of neonates	Birthweight of infants by percentile {% (n) of neonates}			Total
		<10th	10 – 90th	>90th	
Mild	29	13.8 (4)	75.9 (22)	10.3 (3)	100 (29)
Moderate	17	17.6 (3)	82.4 (14)	0	100 (17)
Severe	23	39.1 (9)	60.9 (14)	0	100 (23)
Total	69	23.2 (16)	72.5 (50)	4.3 (3)	100 (69)

Table 3: Distribution of relative placental weight according to severity of maternal pre-eclampsia.

Severity of pre-eclampsia	Number (n) of mothers	Percent (n) of Relative placental weight	
		≤15%	>15%
Mild	30	13.3 (4)	86.7 (26)
Moderate	17	23.5 (4)	76.5 (13)
Severe	24	20.8 (5)	79.2 (19)
Total	71	18.3 (13)	81.7 (58)

Table 4: Frequency of neonatal hypoglycaemia according to relative placental weight.

Relative placental weight	Blood glucose concentration (mmol/L)		
	<1.6	1.6 – 2.5	>2.5
	% (n) of neonates		
≤15% (n = 13)	30.8 (4)[a]	38.4 (5)[c]	30.8 (4)
>15% (n = 56)	10.7 (6)[b]	32.1 (18)[d]	57.2 (32)
Total (n = 69)	14.5 (10)	33.3 (23)	52.2 (36)
p-values:	a vs b >0.05	c vs d >0.05	

Table 5: Distribution of relative placental weigh among normoglycaemic and hypoglycaemic neonates of mothers with pre-eclampsia.

Relative placental weight	Category of neonates		
	Normoglycemic	Hypoglycaemic	
	% (n) of neonates		P-value
≤15% (n = 13)	30.8 (4)	69.2 (9)	
>15% (n = 56)	57.1 (32)	42.9 (24)	>0.05
Total (n = 69)	52.2 (36)	47.8 (33)	

The mean age of the neonates at the time of determination of the first blood glucose concentration was 2.3±0.6 hours (95% Confidence Interval, CI = 2.1-2.5). A total of 33 (47.8%) neonates born to mothers with pre-eclampsia had at least one blood glucose concentration less than 2.6mmol/L (neonatal hypoglycaemia). Of the 69 neonates, 10 (14.5%) had at least one blood glucose concentration less than 1.6 mmol/L (severe neonatal hypoglycaemia). In addition, 8 (11.6%) neonates had 2 or more blood glucose concentration between 1.6 and 2.5 mmol/L and 15 (21.7%) had only one blood glucose concentration between 1.6 and 2.5 mmol/L.

Of the 33 hypoglycaemic neonates, 18(54.5%) were diagnosed in the first 12 hours of life and 25 (75.8%) were diagnosed during the first 24 hours of life. Although the prevalence of neonatal hypoglycaemia was higher among infants associated with relative placental weight equal or less than 15%, the difference was not statistically significant Z-statistic=1.83 p>0.05(Table 5). Similarly, the severity of hypoglycaemia did not correlate with relative placental weight Z-statistic: a versus b=1.49 p>0.05, c versus d= 0.22 p>0.05 (Table 4). The glucose values obtained using the Acutrend glucometer correlated well with values obtained from the central laboratory. Only 5(15.2%) of neonates with hypoglycaemia were symptomatic. The symptoms observed were poor feeding (3 cases), lethargy (3 cases), jitteriness (2 cases) and circumoral pallor (1 case).

DISCUSSION:

The overall prevalence (47.8%) of neonatal hypoglycaemia found in this study was lower than the 64.9% reported from Oulu, Finland [18], but higher than 38.0% reported from Nepal [2]. Although the same methodology and definition were used in the present study and in the Nepalese study, the study population in the later was not at risk of hypoglycaemia which may explain the lower prevalence reported in that study [2]. With regard to the higher prevalence rate reported in the Finnish study compared to the present study, one possible explanation might be the differences in feeding practices in the immediate postnatal period.

Breast feeding of the newborn infant routinely commenced within 1 to 2 hours after birth in our hospital compared to 24 hours after birth in the Finnish hospital [18]. The pattern of breast-feeding practice has been variously shown to influence the prevalence of neonatal hypoglycaemia, (19, 20] a finding attributed to the ketogenesis-promoting property of breast milk [21]. In addition, skin-to-skin contact with the mother during breast feeding facilitates stable temperature and blood glucose for the neonate [22, 23].

Data from the present study showed that majority of mothers with pre-eclampsia, had relative placental weight greater than 15%, suggesting minimal placental growth retardation. The relative placental weight did not differ with the severity of the maternal pre-eclampsia. Similar finding was reported in the Finnish study [18].

In the present study, although the prevalence of neonatal hypoglycaemia was higher among neonates associated with relative placental weight equal or less than 15%, it was not statistically significant. Similar finding has been reported from Oulu, Finland [18]. Among the hypoglycaemic neonates born to mothers with pre-eclampsia, the severity of the hypoglycaemia did not differ with the relative placental weight. This may be explained, as documented by Benirchke et al [13] that the placental pathological features are not all invariably present. Although the number of neonates with symptomatic hypoglycaemia was small, the two leading symptoms of were poor feeding and lethargy. A study from India has reported a similar finding [24].

In conclusion, newborn infants of mothers with pre-eclampsia were at increased risk of hypoglycaemia, particularly in the first 24 hours of life but the prevalence of hypoglycaemia did not differ significantly with the relative placental weight.

REFERENCES:

1. Fox G, Hoque N, Watts T. Oxford Handbook of Neonatology, Oxford University Press, 2010: 354-356.
2. Pal DK, Manandhar DS, Rajbhandari S, Land JM, Patel N, de Castello AM. Neonatal hypoglycaemia in Nepal I: prevalence and risk factors. Arch Dis Child Fetal Neonatal Ed 2000; 82: F46-F51.
3. Ness RB, Roberts JM. Heterogenous causes constituting the single syndrome of pre-eclampsia: A hypothesis and its implications. Am J Obstet Gynecol 1996; 175: 1365-1370.
4. Odegard RA, Vatten LJ, Nilsen ST, Salvensen KA, Austgulen R. Pre-eclampsia and fetal growth. Obstet Gynecol 2000; 96(6): 950-955.
5. Onyiriuka AN, Okolo AA. Perinatal outcome in patients with pre-eclampsia in Benin City, Nigeria. Trop J Obstet Gynaecol 2004; 21(2): 148-152.
6. Ghidini A, Salafia CM, Pezzullo JC. Placental vascular lesions and likelihood of diagnosis of pre-eclampsia. Obstet Gynecol 1997; 90: 542-545.
7. Roberts JM, Redman CW. Pre-eclampsia: more than pregnancy-induced hypertension. Lancet 1993; 341: 1447-1451.
8. Barker PN. Obstetrics by Ten Teachers, 18th edition, London, Hodder Arnold publishers 2006: 156-164.
9. Cunningham FG, Leveno KJ, BloomSL, Hauth JC, Gilstrap LC, Wenstram KD. Williams Obstetrics, 22nd edition, New York, McGraw Hill Medical Publishing Division, 2005: 761-808.
10. Agboola A. The placenta, umbilical cord and membranes. In: Agboola A(ed), Textbook of Obstetrics and Gynaecology for medical Students, 2nd edition, Ibadan, Heinenmann Educational Books(Nigeria) Plc, 20006: 265-273.
11. Soma H, Yoshida K, Mukaida T, Tabuchi Y. Morphologic changes in the

hypertensive placenta. Contrib Gynecol Obstet 1982; 9: 58-75.
12. Naeye RL, Friedman EA. Causes of perinatal deaths associated with gestational hypertension and proteinuria. Am J Obstet Gynecol 1979; 133(1): 8-10.
13. Benirschke K, Kaufmann P. Pathology of the Human Placenta, 2nd edition, New York, Springer-Verlag 1990: 499-529.
14. Khong TY, Pearce JM, Robertson WB. Acute atherosis in pre-eclampsia: maternal determinants and fetal outcome in the presence of the lesion. Am J Obstet Gynecol 1987; 157: 360-363.
15. Abudu OO, Afolabi BB. Diabetes mellitus. In: Agboola A (ed). Textbook of Obstetrics and Gynaecology for Medical Students. 2nd edition. Ibadan, Heineman Educational Books (Nigeria) plc, 2006: 365-370.
16. Redman CWE. Hypertension in Pregnancy. New York, Perinatology Press, 1987.
17. Cheesbrough M. District Laboratory Practice in Tropical Countries (Part I), Cambridge, Cambridge University Press, 2006: 340-348.
18. Koivisto M, Jouppila P. Neonatal hypoglycaemia and maternal toxaemia. Acta Paediatr Scand 1974; 63: 743-749.
19. Huffman SL, Zehner ER, Victora C. Can improvements in breast-feeding practices reduce neonatal mortality in developing countries? Midwifery 2001; 17(2): 80-92.
20. Hoseth E, Joergensen A, Moeller M. Blood glucose levels in a population of healthy, breast fed term infants of appropriate size for gestational age. Arch Dis Child Fetal Neonatal Ed 2000; 83: F117-F119.
21. Hawdon JM, Ward Platt MP, Aynsely Green A. Patterns of metabolic adaptation for preterm and term infants in the first neonatal week. Arch Dis Child 1992; 67: 357-365.
22. Fransson AL, Karisson H, Nilsson K. Temperature variation in newborn babies: importance of physical contact with the mother. Arch Dis Child Fetal Neonatal Ed 2005; 90: F500-F504.
23. Chiu SH, Anderson GC, Burkhammer MD. Newborn temperature during skin-to-skin breast feeding in couples having breast feeding difficulties. Birth 2005; 32(2): 115-121.
24. Singhal PK, Singh M, Paul VK, Deorari AK, Ghorpade MG, Malhotra A. Neonatal hypoglycaemia – clinical profile and glucose requirements. Indian Pediatrics 1992; 29: 167-171.

CASE REPORTS

A RARE CASE OF GINGIVAL CYST OF INFANT OCCURRING IN A BABY AGE FOUR MONTHS

KM Veena*^, H Jagadishchandra, Sham S Bhat***, and Prasanna Kumar Rao***

Departments of Oral Medicine and Radiology*, Oral and Maxillofacial Surgery, Pedodontics and Preventive Dentistry*** Yenepoya Dental College, Yenepoya University, Mangalore, Karnataka, India;**

^Corresponding author: Email: veenaomr@rediffmail.com.

[Running title: Gingival cyst of infants]

ABSTRACT:

Gingival cyst of infant is an odontogenic cyst. It is developmental in nature. It arises from the epithelial remnant of dental lamina called cell rests of Serres. The Gingival cyst may appear within three months of age. Clinically it appears on the maxillary and mandibular ridges, and appears creamish white in color. The cyst usually does not need treatment because it tends to undergo involution and disappears. We present a case report of a solitary gingival cyst observed in a baby age four months.

Key words: Gingival cyst, dental lamina, alveolar cyst.

Received: June 2011; Accepted August 2011

INTRODUCTION:

Gingival cyst of an infant is derived from the remnant of the dental lamina. The cyst may be solitary or many in numbers. The Gingival cysts are seen in the anterior part of the alveolar ridge. The cysts are usually present during the time of birth and rarely seen after three months of age [1]. They regress spontaneously, so no treatment is usually required [2].

Case report:

A four months old male infant reported to the private clinic with a small nodule in the upper gum pad on the right lateral incisor region. Mother said she noticed it one day before when

the child was crying. It was asymptomatic and did not interfere with feeding. On examination, a solitary whitish papule measuring about 0.5 cm in diameter was present on the maxillary alveolar ridge on the right lateral incisor region. It was firm in consistency, non tender and mucosa over the lesion was smooth. Based on the history and clinical examination, a provisional diagnosis of gingival cyst of infant was made. Biopsy of the lesion could not be carried out as parents were not keen on it. The infant was kept under observation. The lesion regressed by itself after three months without any treatment.

The ethical clearance for the publication of the case report was obtained from the concerned authority.

DISCUSSION:

Gingival cysts of infant are small, superficial, keratin filled cysts that are found on the alveolar ridge. They originate from the dental lamina. The cyst is lined by thin epithelium and the lumen is filled with desquamated keratin, occasionally containing inflammatory cells [3]. Since they regress on their own by rupture, the lesion may go unnoticed or not sampled for biopsy. They are small, whitish papules on the alveolar ridges or on the palate. Based on the location they are classified either as 'palatal' or as 'alveolar' cysts [2]. Usually multiple cysts are present with each measuring not more than 3.0mm in diameter. Involvement of maxillary ridge is more common than mandibular ridge. Occasionally these cysts appear on the mandibular anterior ridge of newborn and misdiagnosed as natal teeth [2]. In the present case report the gingival cyst was a whitish papule measuring about 3.0mm on the maxillary ridge in the lateral incisor region. Majority of these type of cysts degenerate and involutes or rupture in to oral cavity within two weeks to five months of age [4,5]. But in our case the cyst was noticed only at four months of age and disappeared when the child was seven months old. Even though, the gingival cyst of infant is of little pathologic significance, it has to be diagnosed to avoid unnecessary therapeutic procedures and parents should be reassured.

REFERANCES:

1. Neville, Damm, Allen, Bouquot. Oral & Maxillofacial Pathology. 2nd edition, 2004; p601
2. Kumar A, Grewal H, Verma M. Dental lamina cyst of newborn: A case Report. J Indian Soc Pedod Prevent Dent. Dec 2008, 26(4): 175-6.
3. Shafer WG. Cysts and tumors of odontogenic origin. In: Hine MK, Levy BM, Tomrich CE, editors. Textbook of oral pathology. 4th Dent 1994; 4:67-73. ed. India: W.B. Sounders Co, Prism(Reprint); 1993; p 268-9.
4. Paula JD, Dezan CC, Frossard WT, Walter LR, Pinto LM. Oral and Facial inclusion cysts in newborns. J Clin Pediatr Dent 2006; 31: 127-9.
5. Flinck A, Paludan A, Matsson L, Holm TL, Axelsson I. Oral findings in group of newborn Swedish children. Int J Clin Pediatr Dent 1994; 4:67-73.

LICHENOID REACTION ASSOCIATED TO AMALGAM RESTORATION: A CASE REPORT

Pradeep K*, Gary Ignatius, Vidaydhar Shetty and Harish Kumar Shetty
Department of Conservative Dentistry & Endodontics, Yenepoya Dental College, Yenepoya University, Deralakatte, Mangalore, Karnataka, India
***Corresponding Author: Email:** endopradeep@gmail.com.

ABSTRACT

Lichenoid amalgam restoration also known as amalgam associated oral lichenoid reaction, is an uncommon allergic reaction following long-term exposure to dental amalgam restorations. This is a case of oral lichenoid reaction associated to amalgam restorations in a 34 year-old male patient. He presented with a whitish discoloration on his left lower buccal mucosa seven months after a non-contributory medical and dental history. On examination, the presence of class I (buccal pit) silver amalgam restorations in relation to left Mandibular first and second molars was observed. Management included removing the amalgam restoration and using composite resin as a substitute. After 40 days, complete healing was observed. This case was reported from Yenepoya dental college, Mangalore, India.

Key words: Oral lichenoid reaction, Amalgam restorations, composite resins.
Received: June 2011; Accepted August 2011

INTRODUCTION:

Silver amalgam has been used as a dental restorative material for more than 150 years. Even today, with the advent of new synthetic non-metallic materials and novel time-saving procedures, silver amalgam is the most widely used and cost-effective dental material in restorative dentistry. Its superior compressive strength and minimal technique sensitivity makes it an ideal material for posterior restorations and core build ups [1]. In addition to corrosion and metallic colour, amalgam has got a major disadvantage [1]. Amalgam fillings are in direct contact with the oral mucosa and may directly alter the antigenicity of basal keratinocytes by the release of mercury and other metal salts as corrosion products [2,3,4]. In susceptible individuals, therefore, amalgam fillings nay induce amalgam-contact hypersensitivity lesions (ACHL) with features similar to oral lichen planus (OLC). Such lesions are likely to occur on mucosal surfaces

in intimate contact with amalgam fillings and could be expected to improve following removal of the fillings [5]. Pinkus [6] in 1973 coined the term Lichenoid lesion. Koch et al 1999 [7] proposed "Dental restoration metal intolerance syndrome". Skoglund [4] showed that removal of amalgam usually affects the lesions favorably and that epicutaneous patch tests are of little prognostic value in patients with oral mucosal lesions of lichenoid character [7,8]. Bratel et al [9] proposed that vast majority of contact lesions (CL) can be resolved by selective replacement of restorations of dental amalgam, provided that correct clinical diagnosis had been established [9].

The ethical clearance for the publication of the case report was obtained from the Yenepoya University Ethics Committee.

Case report:

A 35 year-old male patient reported to the Department of Conservative Dentistry and Endodontics, Yenepoya Dental College and Hospital, Mangalore with a chief complaint of whitish discoloration on his left buccal mucosa since seven months with a non-contributory medical and dental history. On detailed hard tissue examination, the presence of class I (buccal pit) silver amalgam restorations in relation to left Mandibular first and second molars was observed. On further detailed soft tissue examination of the entire oral cavity, an unilateral Whitish discoloration of the buccal mucosa was also observed in relation to the left mandibular teeth, extending from mandibular second molar to first premolar [Figure 1].

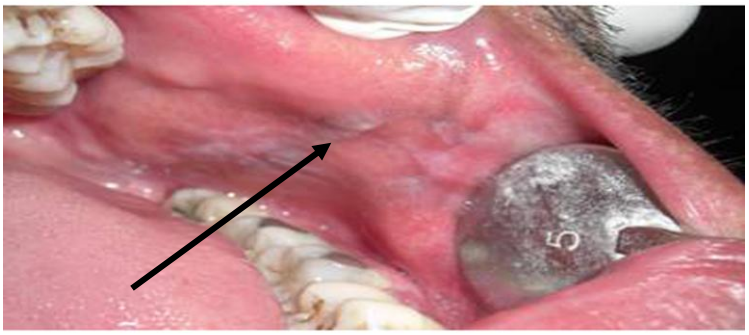

Figure 1: Buccal pits of left lower mandibular first and second molar teeth and white keratotic patch seen on the buccal mucosa

The Patient gave a dental history of undergoing silver amalgam restorations of his decayed teeth in relation left mandibular first and second molars a year back with no presence of any other decayed or restored teeth in the oral cavity.

The whitish discoloration was diagnosed as a reaction of the oral mucosa to silver amalgam restorations – Lichenoid reaction.

The management was for the replacement of silver amalgam restorations with composite resin. Amalgam restorations were removed using a high speed rotary handpiece with a round bur following the occupational and safety health administration (OSHA) regulations. After removal of the entire amalgam restorations, Glass ionomer cement (Fuji II) was placed as base on the pulpal floor and was temporarily restored with Zinc Oxide eugenol cement. The whole procedure was done in a dental clinic as outpatient procedure. On the following day the temporary restoration was removed leaving behind the Glass ionomer cement. The tooth was etched using 37.0% phosphoric acid for 15 sec followed by application of dentin bonding agent and curing. Composite restoration was done by incremental layer technique and curing for 30sec. Finishing and polishing (silicon carbide stone & alpine stone, sofelex disc) of the restoration was done after a week. The patient was recalled once in every 10 days for a period of one month. After a time period of 30 to 40 days there was a total disappearance of the lichenoid reaction and the patient was followed up for a period of six months with no recurrence seen [Figure 2].

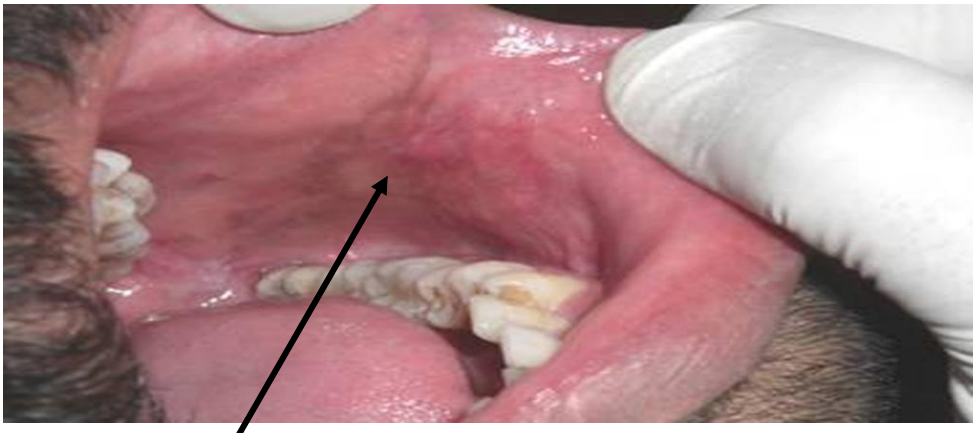

Figure 2: After 40 days – Total disappearance of the Lichenoid reaction

DISCUSSION:

Oral mucosal lesions related to dental restorative materials may be caused by delayed cell-mediated hypersensitivity reactions [10]. In the present case histopathological examination was not done, because it is an invasive procedure; the intention was to use a more conservative approach for the management of the patient.

The most common contact lesions of the oral mucosa due to metal hypersensitivity are caused by nickel or chromium in orthodontic appliances or frame-work for partial dentures [11]. A review of cases reported as Mercurial hypersensitivity from mercury exposure in dentistry has been given by Bauer and First [12]. Accumulations of mercury have been found in lysosomes of macrophages and fibroblasts of submucous connective tissue of contact lesions, and also in normal mucosa [1]. There seems to be a great discrepancy in the manifestation of the incidence of hypersensitivity reactions inherent with the use of amalgam restorations as treatment of choice for the restoration of carious teeth [10].

Wong and Freeman [13] in their study confirm the mercury allergy is a factor in the pathogenesis of oral lichenoid reaction and healing of oral lichenoid reaction after replacement of amalgam restorations with Glass ionomer or composite resin.

It has been proposed that hypersensitivity to mercury from corroding amalgam fillings plays an important part in the etiology of oral lichen planus [14]. Some studies have demonstrated hypersensitivity to mercury among 16.0-62.0% of patients with oral lichen planus, whereas mercury hypersensitivity has been found in 1.0-4.0%in the general population of Sweden [14]. Only in 10.0% of the patients the mucosal affections disappeared after replacement of type II glass ionomer cements or composite resins [14]. Further, the presence of lichen planus on the oral mucosa may well render the patients more susceptible to mercury hypersensitivity because of the increased penetration of the affected oral mucosa by mercury [14].

A recent study demonstrated a different response of lichenoid mucosal lesions to replacement of amalgam fillings depending on the extensions of the lesions: those lesions, denoted contact lesions, which were confined to the area of contact with amalgam showed a total or almost disappearance without recurrence after replacement, whereas lesions exceeding the contact zone showed minor changes only [15].

In Conditions like lichenoid reactions secondary to silver amalgam restorations, using composite restoration having added advantages like good aesthetic and wear resistance properties compare to other restorative materials.

CONCLUSION:

Silver amalgam has been used as a dental restorative material for more than 150 years. Even today, with the advent of new synthetic tooth coloured materials, silver amalgam is the most widely used and cost-effective dental material in restorative dentistry. Local allergic reactions are rare, and when they occur, they can be eliminated by substitution with glass ionomer or composite resin. In the present case, the tissue becomes normal within 40 days after the buccal amalgam restorations were removed. The present article gives information about allergic reactions related to silver amalgam restorations and its managements.

REFERENCES

1. Sunit M. Kumar R. Amalgam associated oral lichenoid reaction. Journal of conservative dentistry .2006;9: 148-51.
2. Camisa C, Taylor JS, Bernat JR, Helm TN. Contact hypersensitivity to mercury in amalgam restorations mimic oral lichen planus. Cutis1999;63: 189-92.
3. Pang BK, Freeman S. Oral lichenoid lesions caused by allergy to mercury in amalgam fillings. Contact Dermatitis.1995; 33: 423-7.
4. Skoglund A. Value of epicutaneous patch testing in patients with oral, mucosal lesions of lichenoid character. Scand J Dent Res.1994;102:216-22.
5. Thornhill MH, Pemberton MN, Simmons RK, Theaker ED. Amalgam - contact hyper sensitivity lesions and oral lichen planus. Oral Surg Oral Med Oral Pathol Oral Radiol Endod.2003; 95:291-9.
6. Do Prado RF, Marochio LS, Felipini RC. Oral lichen planus versus oral lichenoid reactions: Difficulties in diagnosis. Indian J Dent Res. 2009;20:361-64.
7. Koch P, Bahmer FA, Oral lesions and symptoms related to metals used in dental restorations : a clinical, allergological, and histologic study, J Am Acad Dermatol 1999;41::422-30.
8. Smart ER, Macleod RI, Lawrence CM. Resolution of lichen planus following removal of amalgam restorations in patients with proven allergy to mercury salts: a pilot study. Br Dent J.1995; 178: 108-12.
9. Bratel J, Hakeberg M, Jontell M; Effect of replacement of dental amalgam on oral lichenoid reactions, J Dent,1996; 24 : 41-5.
10. Bolewska J, Hansen HJ, Holmstrup P, Pindborg JJ, Stangerup M. Oral mucosal lesions related to silver amalgam restorations. Oral Surg Oral Med Oral Pathol. 1990; 70:55-8
11. Bergman M, Bergman B, Soremark R. Tissue accumulation of nickel released due to electrochemical corrosion of non-precious dental casting alloys. J Oral Rehabil 1980; 7: 325-30
12. Bauer JG, First HO. The toxicity of mercury in dental amalgam. Calif Dent Assoc J 1982; 10: 47-61
13. Wong. L, Free mans .S .Oral lichenoid reaction and mercury in amalgam fillings. Contact dermatitis. 2003 Feb; 48: 74-79
14. Lundstrom JM. Allergy and corrosion of dental materials in patients with oral lichen planus. Int J Oral Surg 1984; 13: 16-24
15. Finne K, Goransson K, Winckler L. Oral lichen planus and contact allergy to mercury. Int J Oral Surg 1982; 11: 236-9.

DIVERSE FORMS OF GINGIVAL ENLARGEMENT – REPORT OF TWO CASES

Saba Khan*, Sreeja P. Kumar, Laxmikanth Chatra, Prashanth Shenai and Prasanna Kumar Rao

Department of Oral Medicine and Radiology, Yenepoya Dental College, Yenepoya University, Deralakatte, Mangalore, Karnataka, India;

***Corresponding Author: Email - dr.sabakhan23@gmail.com.**

ABSTRACT:

Gingival hyperplasia (GH) is an increase in the gingival height or mass due to proliferation and thickening of gingiva. Gingival hyperplasia represents an over-exuberant response to certain inflammatory and genetic factors, drugs, systemic diseases, neoplasms. Hereditary gingival fibromatosis (HGF) is a rare oral disease, affecting only one in 750,000 people. It is characterized by a slow and progressive enlargement of both maxilla and mandibular gingiva. It usually develops as an isolated disorder, but can be one feature of a syndrome. Drug induced gingival enlargement is frequently observed as a side effect with the use of several medications in the susceptible patients. These reports address the diagnosis, treatment and follow up of two separate cases of gingival enlargement.

Keywords: gingival hyperplasia, hereditary gingival fibromatosis, drug induced gingival enlargement.

Received: June 2011; Accepted: August 2011.

INTRODUCTION:

Gingival hyperplasia is the increase in size of the gingiva resulting from proliferation of its cellular elements [1]. The clinical features of gingival enlargement depend on the etiologic factors and the pathological processes associated with it. Gingival enlargements are broadly classified into inflammatory, fibrotic and combination of these two. The main etiologic factors are inflammation, genetic factors, drugs, systemic diseases and neoplasms [2,3]. Inflammatory gingival enlargement can be caused by prolonged exposure to dental plaque, which may occur due to poor oral hygiene. In puberty and pregnancy, hyperplasia of the gingival tissues may be due to poor oral hygiene, inadequate nutrition, or systemic hormonal stimulation [4]. Fibrotic gingival enlargement is a non-inflammatory type of enlargement seen in hereditary and drug induced forms. It is neither hypertrophy nor hyperplasia. Increase in the intercellular matrix

is responsible for the enlargement. Hereditary gingival enlargement is rather rare but the drug induced enlargement is much more common [5].

Gingival enlargement is the preferred term for all medication-related gingival lesions previously termed “gingival hyperplasia” or “gingival hypertrophy” [6]. Drug induced gingival enlargement is frequently observed as a side effect with the use of several medications in the susceptible patients. Drugs associated with gingival enlargement can be broadly divided into three categories: anticonvulsants, calcium channel blockers, immuno-suppressants [6].

Hereditary gingival fibromatosis (HGF) is a rare condition characterized by a proliferative fibrous overgrowth of the gingival tissues. It usually develops as an isolated disorder but can be one of the features of several multisystem syndromes [7].The syndromic characteristics most commonly seen in association with HGF are hypertrichosis, mental retardation, epilepsy [7]. HGF is an autosomal dominant disorder with a high degree of penetrance, although recessive forms are also described in literature [8]. Males & females are equally affected as a phenotype frequency of 1:175,000 and a gene frequency of 1: 350,000 [8]. Here we are presenting two case reports of gingival enlargement, one with hereditary cause and another induced by the drug amlodipine.

CASE REPORT 1

A 21year old female presented with a complaint of excessive swelling of the gums and bleeding while brushing. The swelling caused difficulties in mastication and phonation and significant esthetic problem. Besides these no other complains of pain, inflammation, discharge or halitosis were present. Patient remembered having the enlargement since childhood. She did not give a history of taking any drugs known to cause gingival enlargement. The patient gave a history of her brother (35 years) having a similar gingival enlargement, which was later confirmed on his examination. Intra oral examination of the patient revealed uniform, generalized and severe gingival overgrowth involving buccal and lingual tissues of both mandibular and maxillary arches with morphologically normal teeth. The tissue covered the crowns of the teeth till middle 1/3 rd (**Figure. 1**). The gingival surface was pink, firm ,granular and pebbled with abundant stippling. No acute inflammatory signs were present. Moderate local deposits were present. Routine blood investigations showed normal values. Panoramic radiograph revealed bone resorption, more severe in respect to the mandibular teeth’s indicating periodontitis (**Figure. 2**). A incisional biopsy was performed and it was confirmed as gingival fibromatosis. Thus a diagnosis of hereditary gingival

fibromatosis was made based on absence of drug history, positive familial history, clinical and histopathological features. Gingivectomy procedure with periodontal pack placement was done over duration of two weeks. Instructions were given to the patient to strictly maintain the oral hygiene. After the last gingivectomy procedure the patient returned for post surgical follow up after one month (**Figure. 3**). Patient was advised for routine scaling and oral prophylaxis procedure once in every six months.

The ethical clearance for the publication of the case report was obtained from the concerned authority.

CASE REPORT 2:

A 40 year old female patient reported to our department with a chief complaint of enlarged gums in the upper and lower front and back teeth region noticed since 1 year. Initially there was small bead-like nodular enlargement of the gums that gradually progressed to the present size covering almost the entire front teeth. Enlargement was associated with intermittent pus discharge, bleeding and difficulty in chewing food. Her past medical history revealed that the patient was on Amlodipine 5 mg taking once daily since 2 years. On intraoral examination, marginal, attached and interdental gingival enlargement was well appreciated covering almost coronal one-third of maxillary and mandibular teeth and is extending to the lingual and palatal mucosa. Gingiva was pink in colour with erythematous area in relation to maxillary left lateral incisor and has lobulated surface. Margins of the gingiva were rolled out with normal gingival scalloping. On palpation, gingiva was firm and resilient in consistency. Hypertrophied areas were painless and did not bleed on touch. Poor oral hygiene status of the patient was assessed from the presence of local irritating factors contributing to the mild inflammatory component of the gingival enlargement (**Figure. 4**). Patient was subjected to complete hemogram and all the parameters were found to be within normal range. Orthopantomograph revealed complete set of dentition with generalized horizontal bone loss (**Figure. 5**). On the basis of the patient's history and clinical features, a clinical diagnosis of amlodipine induced gingival overgrowth (AIGO) was made. Patient was subjected to gingivectomy procedure and was recalled for follow up after a month (**Figure. 6**). Patient's physician was consulted regarding drug substitution or withdrawal of the drug. Patient was instructed to maintain good oral hygiene with the use of chlorhexidine oral rinses.

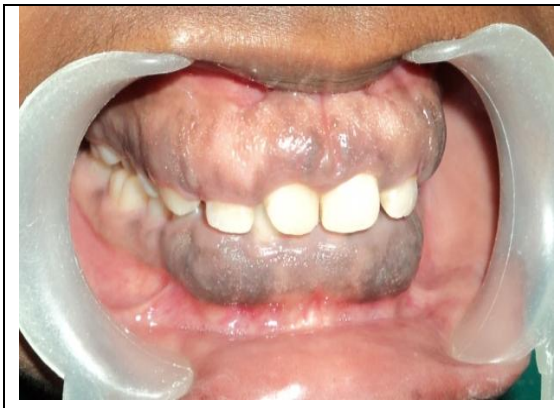

Figure 1: Hereditary gingival enlargement in relation to maxillary and mandibular gingiva.

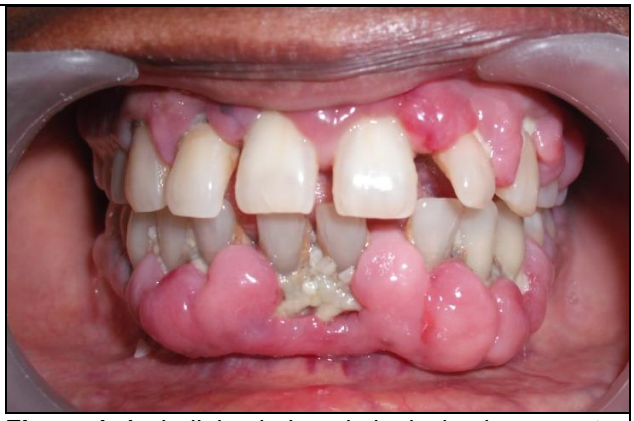

Figure 4: Amlodipine induced gingival enlargement of marginal,attached gingival,interdental papilla.

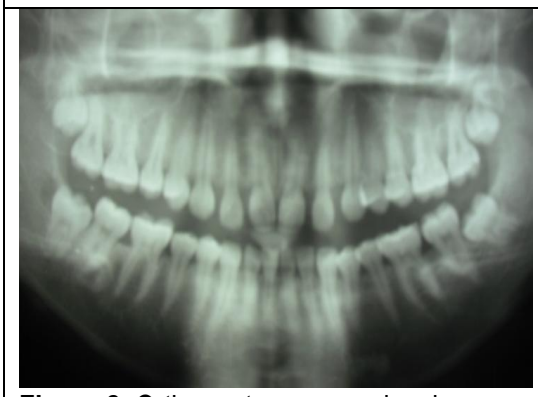

Figure 2: Orthopantomogram showing generalized horizontal bone loss in hereditary gingival enlargement.

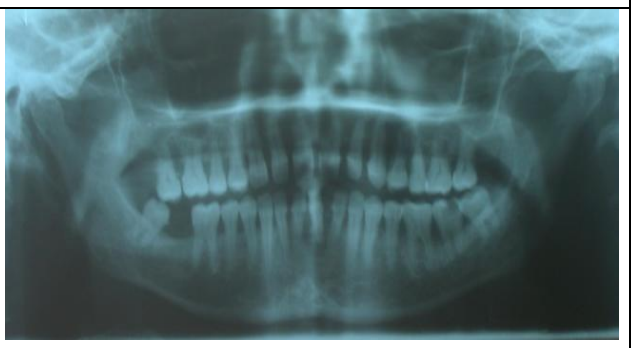

Figure 5: Orthopantomogram showing generalised horizontal bone loss in Amlodipine induced gingival enlargement.

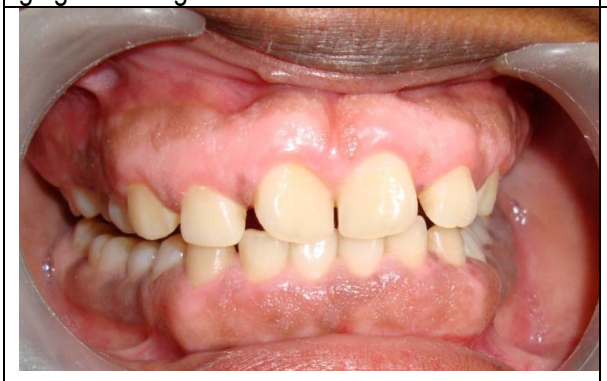

Figure 3: Post operative - after gingivectomy procedure in hereditary gingival enlargement

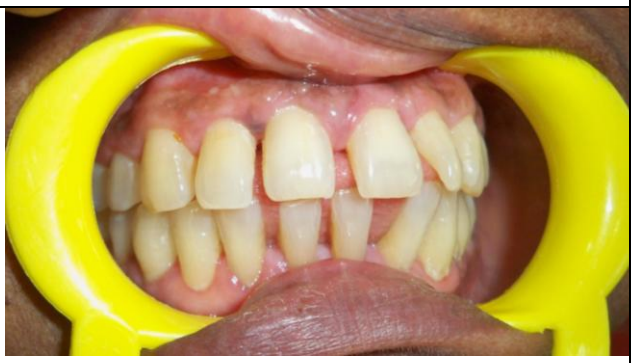

Figure 6: Post operative -after gingivectomy procedure in Amlodipine induced gingival enlargement.

DISCUSSION:

This report documents two cases of gingival enlargements due to two different etiologies. Gingival enlargements have hereditary and acquired forms causes of which are inflammation, leukemia, use of medication such as phenytoin, cyclosporine & calcium channel blockers [9].

HGF can be inherited as an autosomal dominant or recessive condition. Autosomal dominance in a four generation pedigree with 50 of 105 at risk of developing gingival fibromatosis was reported by Bozzo et al [10]. According to Bitten court et al [11], this anomaly is classified in two types according to its form. The nodular form is localized and characterized by presence of multiple enlargements of gingival. The symmetric form is most common type and results in uniform gingival enlargement as was seen in the present case. The enlargement usually begins at time of eruption of permanent dentition and rarely develops with eruption of deciduous dentition. Fletcher reported that the enlargement progresses rapidly during "active" eruption and decreases with end of this stage [12]. He also stated that presence of teeth appears to be necessary for HGF to occur as the condition is not seen before eruption of teeth and disappears with loss of teeth. It is accepted that HGF is a disease of genetic origin. Some authors report increase in proliferation of fibroblasts, collagen synthesis and elevated matrix metalloproteinase's while others suggest a decrease in collagenase activity [13]. A gene locus for hereditary gingival fibromatosis has been localized to the 37CM genetic interval on chromosome 2p 21-p22 flanked by D2 S1788 and D2S441 [13].

Calcium channel blockers are considered potential etiologic agents for drug-induced gingival hyperplasia. Although the incidence of nifedipine-induced gingival hyperplasia is about 10%, very few reports of amlodipine-related gingival hyperplasia have been reported in the literature [6]. The prevalence rate of gingival enlargement in patients taking amlodipine is found to be 3.3% [14]. Because only a subset of patients treated with this medication will develop gingival overgrowth, it has been hypothesized that these individuals have subsets of fibroblasts with an abnormal susceptibility to the drug. It has been showed that fibroblast from overgrown gingiva in these patients are characterized by elevated levels of protein synthesis, most of which is collagen [15]. Most types of pharmacological agents implicated in gingival enlargement have negative effects on calcium ion influx across cell membranes, thus it has been postulated that such agents may interfere with the synthesis and function of collagenases, thereby inhibiting collagen degradation [16]. Several factors such as age, genetic predisposition, pharmacokinetic variables, and alteration in gingival connective tissue homeostasis, histopathology, ultrastructural factors, and inflammatory changes may influence the

relationship between the drugs and gingival tissues.

CONCLUSION:

The above case reports outline the two forms of gingival enlargements, their identification and diagnosis. Gingival hyperplasias have potential cosmetic implications and also provide new niches for the growth of microorganisms, which is a serious concern for both the patients and oral diagnostician

REFERENCES:

1. Nayar RB, Bai M, Anil S. Symmetrical gingival fibromatosis-report of a case JIDA1993;64:233-35.
2. Shivaswamy S, Siddiqui N, Jain SA, Koshy A, Tambwekar, Shankar A. A rare case of generalized pyogenic granuloma: A case report. Quintessence International 2011; 42:493-99.
3. Dannewitz B. Proliferation of the gingiva:aetiology, risk factors and treatment modalities for gingival enlargement. Periodontal practice today 2007; 4:83-91.
4. Cekmez F, Pirgon O, Tanju IA. Idiopathic Gingival Hyperplasia. International journal of biomedical science 2009;5(2):198-200.
5. Varma BRR, Nayak RP. Current concepts in periodontics , Arya publishing house,New Delhi, 1st edition 2002:p 136-145.
6. Seymour RA, Ellis JS, Thomason JM, Monkman S, and Idle JR. Amlodipine-induced gingival overgrowth. J Clin Periodontol 1994; 21: 281-83.
7. Ramer M, Marrone J, Stahl B, Burakoff R. Hereditary gingival fibromatosis: identification, treatment, control. Journal of American Dental Association 1996; 127:493-495.
8. Santosham K , Suresh R, Malathi N. A Case report of Idiopathic gingival fibromatosis: Diagnosis & treatment. Journal of International Academy of Periodontology 2009; 11: 258-63.
9. Sharma A, Nagpal S. Treatment of Recurrent Gingival Enlargement associated with Zimmermann-Laband Syndrome: A Case Report. Int. Journal of Contemporary Dentistry 2011;2:77-80.
10. Baptista IP. Heriditary gingival fibromatosis : A case report . J. Clinical Periodontology 2002;29: 871-74.
11. Bittencourt LP , Campos V , Moliterno LF , Ribeiro DP, Sampaio RK. Hereditary gingival fibromatosis: Review of literature &a case report. Quintessence Int 2000 ;31:415-418.
12. Fletcher JP. Gingival abnormalities of gingival origin: A preliminary communication with special reference to hereditary generalized gingival fibromatosis. Journal of Dental Research 1966; 45: 597- 612.
13. Hart TC, Zhang Y , Gorry MC. A mutation in the SOS1 gene causes hereditary gingival fibromatosis type1. American Journal of Human Genetics 2002; 70: 943-54.
14. Taib H, Ali TBT, Kamin S. Amlodipine-induced gingival overgrowth: a case report. Archives of Orofacial Sciences 2007; 2: 61-64.
15. Hassell TM, Page RC, Narayanan AS, Cooper CG. Diphenylhydantoin (Dilantin) gingival hyperplasia: Drug-induced abnormality of connective tissue. Proc Natl Acad Sci (USA) 1976; 73:2909-12.
16. Grover V, Kapoor A, Marya CM. Amlodipine Induced gingival hyperplasia-case report. J Oral Health Comm Dent 2007; 1:19-22.

TOBACCO INDUCED LICHENOID REACTION

Prasanna Kumar Rao*, Veena KM, Laxmikanth Chatra and Prashanth Shenai

Department of Oral Medicine and Radiology, Yenepoya Dental College, Yenepoya University, Mangalore, Karnataka, India

***Corresponding Author: Email: drjpkrao@gmail.com.**

ABSTRACT:

The oral mucosa may present clinical features of a certain conditions similar to those observed in lichen planus called lichenoid reaction. The pathological feature resembles that of lichen planus. The pathologist requires the indication of a cause – effect relationship by the clinician in order to provide a diagnosis of lichenoid reaction. This condition is treated by removal of the causal factor. Here we report a case of lichenoid reaction due to tobacco chewing habit in an 30 year old male patient. This case was reported from Yenepoya dental college, Yenepoya University, Mangalore, India.

Key Words: Lichenoid reaction, tobacco, habits.

Received: June 2011; Accepted August 2011

INTRODUCTION:

The oral mucosa may present different types of clinical and microscopic alterations similar to Lichen planus. One such condition is called as lichenoid reaction. These conditions are triggered by various systemic or topical causative agents. The etiology of Lichenoid reaction is related to the contact with specific agents, such as restorative materials, drugs, and tobacco habits [1]. One such sensitivity reaction is known as lichenoid reaction. The first microscopic features are discussed in 1973 and term lichenoid reaction was introduced in 1986 [2,3]. He described the features as destruction of basal cell layer due to hydropic degeneration causing consequent interruption in the basal membrane Pathologists use the terms “lichenoid mucositis” or “chronic mucositis with lichenoid features’. The reason for this is because there are not enough distinctive features that make the lichenoid reaction a definitive diagnosis for true lichen planus. Therefore, the diagnosis may be lichen planus or a lichenoid reaction depending upon how clearly consistent the features may be in a tissue sample [4].

The ethical clearance for the publication of the case report was obtained from the Yenepoya University Ethics Committee.

Case report:

A 30-year-old male patient presents with a complaint of burning sensation of left buccal mucosa of 1 month duration. Personal history indicates that he has habit of chewing areca nut since last two years. Frequency of the chewing habit was four to five quids per day. He usually keeps the betel quid in the left buccal sulcus approximately two to three hours after chewing.

On examination an erythematous area interspersed with white striac and blackish pigmentation was observed on the left buccal mucosa [Figure 1]. The lesion was non scrapable and tested negative for Candida. A provisional diagnosis of Tobacco induced Oral Lichenoid Reaction was made. Habit councelling was done and the patient was advised to stop the quid chewing habit. The patient was asked to report back two weeks later. He reported with a relief of symptoms. Another review conducted after 3 months showed complete clinical healing of the lesion.

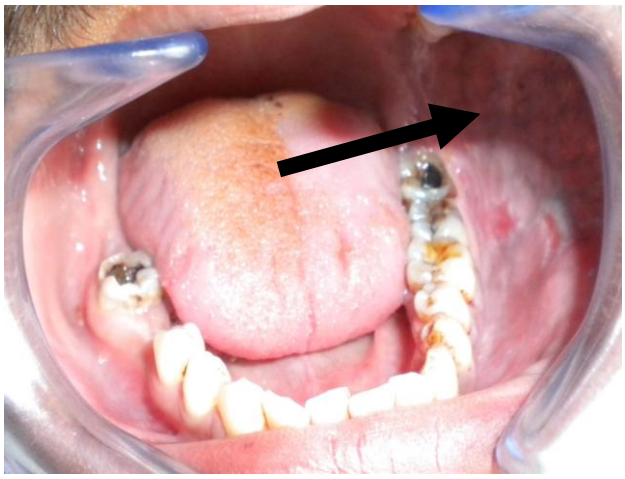

Figure 1 – White striations and pigmentation with eroded areas on the left buccal mucosa

DISCUSSION:

Few lichenoid reaction cases are reported due to the contact of cobalt, nickel, gold, palladium, due to corrosion of the amalgam restorations and after placement of orthodontic arch-wires [5,6]. Few studies also suggested lichenoid reactions with a characteristic microscopic aspect associated with the habit of chewing gum or eating candies with cinnamon flavor, with disappearance of symptoms when the habit was discontinued [1].

Tobacco induced lichenoid lesions are mostly involving buccal mucosa or the tongue and they are unilateral in nature because these are the

sites of betel quid retention. These lesions usually resolve after cessation of the habits. Lichenoid reaction is a mucocutaneous condition with multiple etiologies ranging from silver amalgam contact to quid chewing habit. Accurate identification of the etiologic agent helps in arriving at the appropriate diagnosis and hence is considered to be most important factor in treatment planning.

REFERENCES:

1. Dunlap CL, Vincent SK, Barker BF. Allergic reaction to orthodontic wire: Report a case. J Am Dent Assoc 1989; 118:449-450.
2. Pinkus H. Lichenoid tissue reactions. A speculative review of the clinical spectrum of epidermal basal cell damage with special reference to erythema dyschromicum persistans. Arch Dermatol 1973;107:840-846.
3. Lind PO, Hurlen B, Lyberg T, Aas E. Amalgam-related oral lichenoid reaction. Scand J Dent Res 1986; 94:448-451.
4. Thornhill MH, Pemberton MN, Simmons RK, Theaker ED. Amalgam - contact hyper sensitivity lesions and oral lichen planus Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2003; 95(3):291-299.
5. Stenman E, Bergman M. Hypersensitivity reactions to dental materials in a referred group of patients. Scand J Dent Res 1989; 97:76-83.
6. Nancy W. Burkhart. Lichenoid Reactions. www.Dentistry iQ. Oct 2009

Letter to the Editor:

ORAL FINDINGS IN ISOLATED GLOSSOPHARYNGEAL PALSY

Reshma Suvarna*, Shishir R. Shetty and Subhas G. Babu

Department of Oral Medicine and Radiology, AB Shetty Memorial Institute of Dental Sciences, Nitte University

Corresponding author: Email: itsreshma_11@yahoo.co.in.

Dear Editor, Glossopharyngeal nerve is the IX cranial nerve, which helps in palatal movement, as it innervates the stylopharyngeus muscles (has role in elevation of the pharynx), whose damage can result in a complication knows as glossopharyngeal nerve palsy [1].

Here we report a case of isolated glossopharyngeal nerve palsy, following tonsillectomy.

A 24-year-old man reported to the out patient department of a dental college in Mangalore, with the chief complaint of difficulty in swallowing. He denied any history of trauma but gave us the history of tonsillectomy three years back.

Clinical examination showed incomplete elevation of the soft palate on the right side. There was absence of gag reflex and uvular deviation was seen to the right [Figure 1].

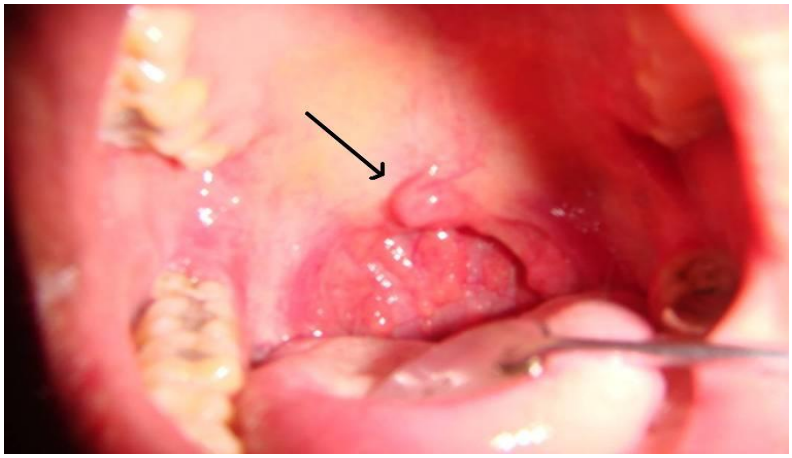

Figure 1: Uvular deviation seen towards the right side

No facial palsy, diplopia, nor any evidence of motor weakness or sensory deficit. Deep tendon reflex of jaw, limbs was normoflexive and symmetric.

We present a case report and proposing an anatomic explanation for a rare complication of dysphagia following tonsillectomy, caused by paralysis of glossopharyngeal nerve.

The mean distance between postero-superior tonsillar fossa and the main trunk of glossopharyngeal nerve is 10.7mm and the mean distance from the postero-inferior tonsillar fossa and the closest lingual branch of the nerve is 6.5mm [2].

Direct nerve injury seems to be the most plausible explanation for this rare complication. The proximity of the nerve to the tonsillar fossa emphasizes the importance of maintaining the correct surgical plane during surgery [2].

Occipital condyle fracture, bulbar palsy, traumatic dissection of internal maxillary artery and compression of the nerve by rheumatoid pannus can be associated with isolated glossopharyngeal palsy [3, 4, 5].

Due to widespread practice of tonsillectomy, students of health sciences and practitioners should be taught on this entity and should be trained to maintain a proper surgical plane during tonsillectomy so as to prevent any damage to glossopharyngeal nerve.

Patient consent and the ethical clearance from the concerned institution were obtained for the above report.

REFERENCES

1. Greenberg M S, Glick M. Burket's Oral Medicine Diagnosis and Treatment Planning. 10th ed, Philadelphia: BC Decker; 2002: 21.
2. Ford LC, Cruz RM. Bilateral glossopharyngeal nerve paralysis after tonsillectomy: case report and anatomic study. Laryngoscope. 2004 D; 114: 2196-9.
3. Urculo E, Arrazola M, Arrazola M Jr, Riu I, Moyua A. Delayed glossopharyngeal and vagus nerve paralysis following occipital condyle fracture: Case report. Journal of Neurosurgery 1996; 84: 522-5.
4. Park JK, Oh HG. Isolated glossopharyngeal nerve palsy presenting with bulbar paralysis. Journal of Soonchunhyang Medical Science 2009; 14: 671-2.
5. Ahn JY, Chung YS, Chung SS, Yoon PH. Traumatic dissection of the internal maxillary artery associated with isolated glossopharyngeal nerve palsy: case report: Neurosurgery 2004; 55: 710.

INSTRUCTIONS FOR AUTHORS

AIMS AND SCOPE:

Pacific Journal of Medical Sciences is a peer-reviewed, multidisciplinary journal published by the School of Medicine and Health Sciences (SMHS), University of Papua New Guinea (UPNG).

The aim of The Pacific Journal of Medical Sciences is to provide the forum for researchers, medical practitioners and other professionals to record, publish, and share ideas and research findings that serve to enhance the understanding of the aetiology, symptoms, diagnosis, prevention, control and management of human disease conditions world wide.

The Pacific Journal of Medical Sciences publishes original scientific research reports, case reports, short communications, letters to the editor and reviews, representing new and significant findings in all areas of medical, biomedical and health sciences (including epidemiology, public and environmental health). Book reviews, scientific news and conference proceedings are published on special request.

EDITORIAL POLICIES:

The Pacific Journal of Medical Sciences (Pac. J. Med. Sci.) editorial policies require that: All manuscripts accepted for publication must reflect novelty and originality of quality research and be of interest to a multidisciplinary audience. All papers submitted for publication are peer-reviewed by two anonymous reviewers and the editor-in-chief or a designated member of the editorial board.

The editorial board may request for review articles, commentaries or short reviews on contemporary medical or biomedical issues that the board considered important to the advancement of the aims of the journal.

Original research papers should be both complete and concise; they should essentially offer conclusive results, but they should not exceed 7,500 words, including abstract, tables, figures and references.

Short communications and reports should not exceed 3500 words, including abstract, tables, figures and references. Review articles should not exceed 6,000 words, including tables, figures and references. Letter to the Editor should be brief and to the point.

On preliminary editing, all manuscripts that fail to meet the basic requirements indicated above and those that contain significant and obvious

typographical errors are returned without further processing.

Manuscripts submitted will be reviewed and considered for publication only if they have not been published, simultaneously submitted or already accepted for publication in another journal. The author responsible for correspondence must show evidence of approval of all co-authors when submitting a paper for publication.

All relevant ethical approval for research involving human and animal subjects must conform to the provisions of the Declaration of Helsinki in 1995 (as revised in Edinburgh 2000).

Only research methods that comply with internationally accepted principles of humane animal experimentation are accepted for publication in The Pacific Journal of Medical Sciences. The decision to accept, revise or reject any manuscript for publication in The Pacific Journal of Medical Sciences is the responsibility of the editor-in-chief; this is done after reviewing the reports and comments from the reviewers, in consultation with members of the editorial board.

Disclaimer:

All statements and opinions expressed in any of the manuscripts published in The Pacific Journal of Medical Sciences are of the authors and co-authors, and not necessarily of the editors or members of the editorial board.

The editor-in-chief and members of the editorial board of The Pacific Journal of Medical Sciences disclaim any responsibility or liability for such material and do not guarantee or endorse any products or services mentioned in the articles, nor guarantee any claims made by the authors of the articles.

SUBMISSION OF MANUSCRIPT:

Manuscript should be written in clear and concise English and be intelligible to those that are not specialists in the particular scientific area. Manuscript that does not satisfy these requirements but is acceptable for publication in the Pacific Journal of Medical Sciences because of its essential scientific content will be returned to the authors for extensive and appropriate revision, as recommended by the reviewers and editors.

A covering letter to clarify the following should accompany any manuscript submitted for publication in the Pacific Journal of Medical Sciences: (a) That the scientific data contained in the manuscript has not been published or submitted for publication in any other journal; (b) That ethical clearance and permission for the research had been obtained from the appropriate committee(s) in the institution(s) where the work was carried out; (c) That all the authors have read and approved the content of

the manuscript; (d) The name, address and email contact of the author responsible for correspondence and for communicating with others about revisions and final approval of proof of manuscript; (e) Statements quantifying the contribution of each author to the manuscript (*this is a requirement in the latest guidelines of the International Committee of Medical Journal Editors*).

Only electronic copy of the manuscript sent as e-mail attachment should be submitted using the approved format indicated in the appropriate sections of this document.

Manuscript should be sent by email to any of the following: pacjmedsci@gmail.com.; templevictor@gmail.com;

PREPARATION OF MANUSCRIPT:

Manuscripts should be prepared on one side of A4 paper, using double-spacing. Pages are to be numbered consecutively in the bottom right-hand corner. Manuscript should include the following sections: Title page, abstract and keywords, text, acknowledgements, references, tables and figures.

Style:

The Pacific Journal of Medical Sciences uses both UK and US spelling. Only one or the other should be used throughout a manuscript. SI units should be used for all measurements. Use abbreviations to avoid repetition of long technical terms: Indicate the abbreviation in parentheses when the word is used in full for the first time. Use the approved generic names of chemical substances and drugs. Do not use trade names or brand names of chemicals and drugs.

Title page:

The following should be on the title page: (a) Title of the manuscript – it should be concise and informative; (b) Short running title of not more than 40 characters (optional); (c) Name of each author (first name, middle initial and last name), including highest academic degree; (d) Name and address of institution(s) in which the work was carried out; (e) Name, postal address and email contact of the author responsible for correspondence; Source(s) of research or other types of support for the research project, if any,

Abstract and key words:

The abstract should not be more than 300 words. The following should be clearly stated in the abstract: the purpose of the study, basic procedures, main findings (specific results and statistical significance, if any), and principal conclusions. Abbreviations and references should not be included in the abstract. Not more than 8 key words should be put below the abstract.

Key words are used to assist indexers in cross-indexing published articles and may be published with the abstract. Medical Subject Headings (MeSH) list of the Index Medicus

should be used for selecting key words (www.nlm.nih.gov/mesh/meshhome.html)

Text:

Text of an original manuscript should be separated into the standard IMRAD format as follows: Introduction, Materials and Methods, Results, Discussion. Sections on Acknowledgements and References should be included.

Introduction:

This section should: (a) summarize relevant previous work, using appropriate references, without any extensive review of the subject; (b) clearly state the purpose of the study and summarize the rational for the study or observation; (c) avoid given any data on the work being reported.

Materials and Methods:

This section should: (a) clearly indicate either the sampling procedure or observational subjects; (b) give appropriate references for established techniques and procedures; (c) new techniques and procedures and extensive modifications of existing ones should be presented in sufficient details so that other researchers can easily reproduce and evaluate them; (d) indicate appropriate quality control procedures used for laboratory methods and techniques; (e) indicate ethical procedures if either human subjects were involved [if informed consent was obtained from each subject] or if appropriate guide line for using laboratory animals were followed [see editorial policies above]; (f) indicate statistical methods used, if any.

Results:

Data obtained should be presented in logical sequence in the text, tables and figures should be adequately explained to facilitate their interpretation. Avoid duplicating the results by repeating in the text all the data presented in the tables and figures. The text in the results section should only emphasize or summarize the important data.

Discussion: Major findings should be highlighted before the minor findings. All findings should be related to other relevant studies, if any, using appropriate references. Indicate the implications of the findings and their significance or limitations, including implications for future research. When warranted, propose new hypotheses with appropriate data, but be sure to clearly label them as such. The conclusions should be linked with the goals of the study and be clearly supported by evidence / data. Include recommendations, if applicable.

Acknowledgements:

The following should be acknowledged: Research or other financial grants; Material support, Contributions of Institutions, Colleagues, and other relevant participants.

References:

The Pacific Journal of Medical Sciences uses the Vancouver system of referencing.

The references should be numbered, using Arabic numerals in square brackets, in the order in which they are first used in the text, tables, figures, and legends.

In the reference section, list the references in the order of appearance in the text, tables, figures and legends. Abstracts, unpublished data, oral communications, and personal communications should not be included in the reference section.

All references should be verified against the original documents.

In the reference section, the names of all authors should be included. Avoid using "et al." in the reference section.

Names of journals should be abbreviated, using the approved style indicated in Index Medicus/PubMed.

References should be listed according to the examples given below:

Journal articles:

1. Brander LC, Buess H, Haldimann F, Harder M, Hanggi W, Herrmann U, Lauber K, Niederer U, Zurcher T, Burgi U, Gerber H. Urinary iodine concentration during pregnancy in an area of unstable dietary iodine intake in Switzerland. J Endocrinology Invest. 2003, 26 5: 389 – 396.

***Book*:**

2. Gillett JE. The health of women in Papua New Guinea. PNGIMR: Kristen Press, 1991

Chapter in a Book:

3. Chaney SG. Principles of nutrition II: Micronutrients. In: Delvin TM, editor. Textbook of Biochemistry with Clinical Correlations, 4th ed. Brisbane: Wiley-Less, 1997: 1107– 36.

Published proceedings paper:

4. Kruse-Jarres JD. Basic principles of zinc metabolism. *In*: Kruse-Jarres JD, Scholmerich J, editors. Zinc and diseases of the digestive tract. Proceedings of the International Falk workshop, Germany, 1996: 3 – 15.

Tables:

Tables should be numbered sequentially in Arabic numerals, typed double-space on separate A4 paper for each table; vertical lines should not be used to separate columns. Each table should be self-contained with a comprehensive but concise legend/heading; column headings should be brief, with units in parenthesis.

All non-standard abbreviations used in tables should be explained in footnotes, using the following symbols in this sequence: *, §, ¶, #, $.

Illustrations:

Graphs, line drawings, bar charts, maps, etc., should be labelled as 'figures' and numbered consecutively, using Arabic numerals. All figures should be drawn using computer graphics. Legends should be brief but understandable without referring to the text. Photographs should be unmounted sharp, glossy black and white prints. Photographs should contain scale bars, not magnifications. Colour photographs are not acceptable. Figures, reproduced from another source, should be clearly indicated and appropriate references and written permission from the copyright holder must be submitted with the manuscript.

Electronic copy of manuscripts (e-mail):

When a manuscript is accepted for publication, the corresponding author will be required to send an electronic copy of the corrected or modified manuscript by email.

All email attachments should be scanned with anti-virus software before sending. Automatic software for referencing, footnotes, headers, footers, etc., should not be used during formatting.

All manuscripts should be formatted using MS WORD.

GALLEY PROOFS:

Galley proof will be sent by email to the correspondent author. Only minor corrections should be made, no major alterations to the manuscript will be accepted. Galley proof should be returned within maximum 5 working days from the date of receipt. If any major modification is made, the manuscript will be rejected at this stage.

Correspondent author should correct all printing errors and ensure that the typesetting is accurate in the galley proof. Note that the correspondent author, not the publisher will be responsible for any such errors, should they occur in the published paper.

REPRINTS:

Reprints will not be sent to authors. One copy of the journal will be sent to the correspondent author on request only, subject to availability of funds to cover postage.

COPYRIGHT:

Manuscripts accepted for publication in The Pacific Journal of Medical Sciences become the property of the journal. Therefore, all authors will be requested to sign a transfer of copyright form before the accepted manuscript is published.

Manuscript will not be published, if all the authors (or the correspondent author, on behalf of the others) do not sign the transfer of copyright form. The copyright gives the journal the exclusive rights to reproduce, translate and distribute the article for academic purposes.

CHECKLIST FOR AUTHORS:

- Running title included (Can be omitted if not applicable);
- Abstract prepared according to instructions and include key words;
- Manuscript typed double-space on one side of A4 paper;
- References cited in square brackets in text and listed according to approved
- style for this journal; Uniform spelling throughout the text;
- Tables and Figures on separate A4 pages;
- Covering letter written as required by this journal;
- E-mail address of corresponding author indicated.