AETIOLOGY AND PREVALENCE OF GINGIVAL ENLARGEMENT AMONG TEENAGE STUDENTS IN TWO SECONDARY SCHOOLS.

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DECLARATION

I KAMOLO EDWARD do hereby declare that this is my original work and has not been submitted in any other institution for the award of degree or otherwise.

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ABBREVIATIONS

BDS; Bachelor of Dental Surgery.

UON; University of Nairobi.

KNH; Kenyatta National Hospital.

DIGE; Drug Induced Gingival Enlargement.

OH; Oral Hygiene.

OHI; Oral Hygiene Instructions.

GE; Gingival Enlargement.

SPSS; Statistical Package For Social Science

MRes; Master of Research
ABSTRACT

Several studies have demonstrated that gingival hyperplasia affect different populations and associated with different causes. Causes range from gingival irritation by plaque, atmospheric air and suppurative infections and are classified as inflammatory. Some medications like anticonvulsants, antihypertensives and immunosuppresants have been associated with gingival overgrowth. Hormonal variations during puberty and pregnancy induces enlargements that manifest varying severities. Each of the above causes require identification and appropriate management. This study therefore aims to determine the etiology and prevalence of gingival enlargement among teenagers. It will involve examining the gingiva of teenage students in secondary school. Data obtained will be analysed using manual methods and computer. The perceived benefits of the study include creating of awareness and reinforcement of oral hygiene practices that may help to alleviate the condition.
INTRODUCTION

Gingival enlargement is the increase in size of the gingiva. It is also referred to as gingival overgrowth. Teenage, also referred to as puberty is the time young boys and girls undergo maturity transition into adulthood, between 11 and 19 years. According to location and distribution, gingival overgrowth can be designed as localized, when limited to the adjacent single tooth or group of teeth; generalized, involving the gingiva throughout the mouth; marginal, confined to the marginal gingiva; papillary, confined to the interdental papilla; diffuse, involving the marginal, attached and papilla and discrete, consisting as an isolated sessile or pendunculated tumor-like enlargement. The degree of gingival enlargement can be scored as done by Bokenkamp (1994) as grade 0, with no signs overgrowth; grade 1, enlargement of the interdental papilla; grade 2, involving both the papilla and marginal gingiva and grade 3 where the enlarged gingiva covers three quarters or more of the tooth crown.

Generally, gingival enlargement is classified according to the etiologic factor and pathologic changes. Inflammatory gingival enlargement can either be chronic or acute. Chronic inflammatory is characterized by a bulge around the tooth as the early stage clinical feature. The enlargement increases to cover the crown either on one tooth (localized) or the whole mouth (generalized). It is slow in progression and painless, unless complicated by bacterial infection or trauma. Prolonged exposure to dental plaque, favored by poor oral hygiene, irritation by anatomic abnormalities, improper restorations and orthodontic appliances, are the etiology of chronic inflammatory enlargement. However, mouth breathers and those with incompetent lips suffer from gingivitis and chronic inflammatory enlargement due to irritation from surface dehydration. In acute inflammatory enlargement, gingival abscesses, which are localized, painful, rapidly expanding and of sudden adjacent teeth become sensitive to percussion. If the abscess progress, it/they rupture spontaneously, postulated to result from bacterial infection.

Drug induced gingival enlargement is observed in patients under some medication for epilepsy, immunosuppression and cardiovascular conditions. Anticonvulsants such as hidatoins (phenytoin, ethotoin, mephenytoin), succimides (ethsuximide, methsuximide and valproic acid) cause gingival enlargement in about 50% of the users. Immunosuppressive agents such as
cyclosporine for treatment of organ treatment rejection and diseases of autoimmune origin have been reported to cause gingival overgrowth in about 30% of patients\textsuperscript{5}. The comparative effects of azathioprine and cyclosporine suggest that patients taking >500mg/day are more predisposed to gingival overgrowth\textsuperscript{6}. Calcium channel blockers for the management of hypertension, angina pectoris, coronary artery spasm and cardiac arrhythmias are known to cause gingival overgrowth in about 20% of users\textsuperscript{7}.

Gingival enlargement is also caused by systemic conditions such as hormonal (puberty and pregnancy), nutritional deficiencies (vitamin C deficiency) and allergies\textsuperscript{8,9}. During these periods, sexual hormones level in blood is very high. They act as growth hormones on the gingival tissue receptors hence gingival hyperplasia. Systemic diseases such as leukemia, granulomatous diseases (Wegener's granulomatosis and sarcoidosis) also cause gingival enlargement\textsuperscript{10}. Neoplastic gingival enlargement, benign or malignant account for a small proportion of gingival enlargement cases\textsuperscript{11,12}. Idiopathic fibromatosis, presenting with gingival overgrowth is a rare condition of undetermined cause but some cases are associated with heredity whose mechanism is unknown\textsuperscript{13,14,15}.

The fact that gingival enlargement is a clinical entity of different distribution, degree and localization in dental and other patients, it may rarely be diagnosed. This study is therefore aimed at determining the etiology and prevalence of gingival enlargement in puberty among students from two secondary schools.
LITERATURE REVIEW

Several causes of gingival hyperplasia have been established. The most common and recognized is drug induced gingival enlargement. Furthermore, congenital GE due to hereditary and metabolic disorders has also been observed. Gingival overgrowth secondary to drugs was first reported in dental literature in early 1960’s in institutionalized epileptic children who were receiving phenytoin (Dilantin) for the treatment of seizures. Cyclosporine, a potent immunosuppressant widely used since 1980’s in organ transplant patients and for psoriasis, and numerous calcium channel blocker agents including nifedipine and amlodipine have also been associated with gingival overgrowth. Nifedipine appears to have an additive effect when used together with cyclosporine in transplant recipients with hypertension. Nevertheless, phenobarbital-induced gingival overgrowth has been reported but is rare and therefore needs further evaluation. A study to find out the pathophysiology of DIGE gingival enlargement concludes that these drugs interact with fibroblasts, altering their metabolic functions, inducing increased collagen synthesis than breakdown.

Surprisingly, not all patients on phenytoin, cyclosporine and/or calcium antagonist develop gingival overgrowth. Thus patients at risk should be identified in order to take all the necessary measures to minimize the onset and severity of this condition. Some of the risk factors known to contribute to gingival overgrowth include the presence of gingivitis resulting from poor oral hygiene. The presence of dental plaque may provide a reservoir for the accumulation of phenytoin or cyclosporine. In orthodontic patients, gingival overgrowth has been suggested to be due to nickel allergy causing epithelial cell proliferation. Other intrinsic factors include the susceptibility of some subpopulations of fibroblasts and keratinocytes to phenytoin, cyclosporine and/or nifedipine, and the number of langerhan cells present in oral epithelium.

In the US, incidence rates are reported from case series studies. The prevalence of phenytoin-induced GE is estimated at 15-50% in patients on the medication; whereas that of cyclosporine transplant recipient patients is 27%. The incidence of GE has been reported as 10-20% in patients treated with calcium antagonist in the general population. In India, 57% of epileptic children...
aged 8-13 years who were undergoing phenytoin monotherapy developed GE within 6 months of treatment.

A study by Hassan AA and Ciancios (2004) to determine the relationship between amphetamine ingestion and GE reports a statistically significant increased prevalence (P<05) of GE in a group of patients taking the drug. No sexual or racial predilection has been reported for drug-induced GE. However, men are more likely to develop gingival overgrowth with calcium antagonist. Phenytoin-induced GE appears to be more prevalent in young-aged epileptic patients. This is most likely to be related to the age population, the nature of the disease and poor OH.

Some systemic diseases have been observed to develop oral manifestations of GE. Among them are those which magnify an existing inflammation initiated by dental plaque. Such situations have been described as "conditioned enlargements". Marginal enlargement during pregnancy results from aggravation of previous inflammation and its incidence has been reported as 10% and 70%. The GE does not occur without the presence of bacterial plaque. Tumor-like gingival enlargement—so called pregnancy tumor (not a neoplasm) occurs after the third month of pregnancy or earlier. The reported incidence is 1.8-5%. Enlargement of gingiva is widely seen in puberty. It occurs both in male and female adolescents and appears be favored areas of plaque accumulation. Pubertal and pregnancy GE are induced by hormonal instability. During these periods, the level of sex steroid hormones is high. These hormones act as growth factors with the gingiva being target. They also amplify the effect of inflammatory mediators in response to plaque accumulation. Hormonal changes during puberty result in gingival enlargement for which other local factors may play a significant role.

Puberty begins between ages 11-14 years in most females and 13 years in males, and terminates at 19 years. Thus, this period is referred to as teenage. Two major sex hormones involved during puberty are testosterone in males and estrogen in females. During puberty, periodontal tissue may have an exaggerated response to local factors such as plaque and calculus, resulting in enlargement. The size of the gingival enlargement may reduce after puberty but does not disappear until adequate plaque removal is achieved.
It has been found that puberty brings a new set of oral health challenges especially for girls. The surge of progesterone and estrogen in blood cause a shift in the microbial flora in the oral environment from a "healthy" to a more destructive or pathogenic one. The gram negative bacteria are associated with virulent factors (exotoxins and endotoxins). The body immune system respond by induction of inflammation of the gingival tissue. Negligence of proper oral hygiene practices exacerbate the inflammatory response to an overgrowth. (Julio Herdenez, 2011)

A longitudinal study of 127 children, 11-17 years of age showed a high initial prevalence of GE that tended to decline with age. When the mean number of inflammed gingival sites was observed and the OH index at that time, it could be clearly seen that a pubertal peak of gingival inflammation that was unrelated to OH factors occurred.

Acute vitamin C deficiency does not on itself cause gingival inflammation, but it does cause haemorrhage, collagen degeneration and oedema of the connective tissue. These changes modify the response of the gingiva to plaque to the extend that the normal defensive delimiting reaction is inhibited, and the extend of inflammation is exaggerated. The combined effect of acute vitamin C deficiency and inflammation produces massive GE in scurvy.

Gingival enlargements due to neoplasms have also been recorded. In a survey of 257 oral tumors, approximately 80% occurred on the gingiva. In another study of 868 growths of the gingiva and palate, of which 57% were neoplastic and the remainder inflammatory, the following incidence of tumors was noted: carcinoma, 11.0%; fibroma, 9.3%; giant cell tumor, 8.4%; papilloma, 7.3%; leukoplakia, 4.9%; mixed tumor (salivary gland type), 2.5%; angioma, 1.5%; osteofibroma, 1.3%; sarcoma, 0.5%; melanoma, 0.5%; myxoma, 0.45%; fibropapilloma, 0.4%; adenoma, 0.4% and lipoma, 0.3%.

A slowly progressive and fibrous enlargement of the maxillary and mandibular feature has been reported as a feature of idiopathic fibrous hyperplasia of the gingiva. Characteristically, this massive GE appears to cover the tooth surfaces and displace the teeth. Whilst the cause of the disease is unknown, there appears to be a genetic predisposition. It has not been proven on which gene this genetic disorder is located.
No mortality is associated with GE. Morbidity has been observed to be severe in some cases because of gross overgrowth of gingival tissue, which can lead to gingival bleeding, pain, teeth displacement and periodontal disease.

CHAPTER 2
MATERIALS AND METHODS

STATEMENT OF THE PROBLEM
Gingival enlargement has been reported to have different etiologies. It is not a spontaneously occurring pathology but progresses from mild to severe forms. Hormonal changes have been implicated as a predisposing factor in GE. While in the initial stages, it may not be of much concern to the patients. Complications in the advanced states include pain, speech, feeding and aesthetic problems. Self esteem and productivity are likely to deteriorate as patients get concerned about their appearances. Maintenance of proper oral hygiene by plaque removal using toothbrushes may be very difficult which aggravates the condition by increasing inflammatory response. It is therefore imperative that the aetiological factors associated with gingival enlargement among teenagers be identified.

JUSTIFICATION OF STUDY
Many people may not be aware of the health status of their gingival tissue unless it is symptomatic with bleeding. As a result, they may have gingival enlargements which can only be clinically diagnosed by the physician including the dentist. It is therefore important to examine the whole extent of the gingiva since it may hypertrophy marginally, interdentally or diffusely. Once patients are thoroughly examined and awareness created, they are able together with the clinician’s support, to eliminate etiological factors if possible and also those which aggravate the condition. Their attitude towards the gingival and entire oral health is likely to be provoked hence motivated to attend dental and medical check-ups. Hormonal changes fluctuations...
amplified in puberty predispose individuals to GE. In Kenya, this study has not been conducted among teenagers.

**OBJECTIVES**

**Main objective**

- To determine the etiology and prevalence of gingival enlargement among secondary school students aged 11 to 19 years in Makueni county, from Kitonguni and Kiketi high schools.

**Specific objectives**

1. To determine the factors associated with gingival enlargements and the awareness of the condition among the students aged 11-19 years
2. To determine the prevalence of gingival enlargement among the students
3. To determine factors aggravating gingival enlargement among the students
4. To determine oral hygiene practices in relationship to the gingival health status

**HYPOTHESIS**

- 50% of gingival enlargement in teenagers is the inflammatory type
- The severity of gingival enlargement depends on oral hygiene practices
VARIABLES

Social demographic variables

- Age
- Gender
- Education level
- Residence

Independent variables

- Oral hygiene practices
- Medication
- Self reported immune status
- Other medical conditions, e.g., cardiovascular disorders
- Oral health status

Dependent variables

- Gingival swelling

MATERIALS AND METHODS

Study area
The study is to be conducted in two secondary schools in Makueni county, situated along Nairobi-Mombasa road.
**Study population**
The study will involve secondary school students in the two schools, Kitonguni and Kiketi high schools, aged 11-19 years and Kenyans in nationality.

**Study design**
The study will be a cross-sectional study.

**Data collection, instruments and technique**
Bio-data will be obtained by questioning the participants. They will then be examined under visible natural light. The examiner’s clean hands will be gloved and wooden tongue depressors used to retract and expose the oral tissues. An assistant will be available to record findings. Gloves and tongue depressors will be changed before examining the next student to prevent cross infection. The gingiva will be scored as grade 0 to 3. Location and distribution of enlargements will be designed as localized, generalized, marginal, papillary, diffuse or discreet. The degree of enlargement will be scored using the gingival overgrowth index by Bokenkamp 1994 as:

Grade 0, if no signs of enlargement
Grade 1, GE confined to the interdental papilla
Grade 2, GE involving the papilla and attached marginal gingiva
Grade 3, enlargement covers three quarters or more of the crown.

Their OH practices, medical conditions, dietary vitamin deficiencies will also be queried. Plaque and calculus will scored as either present or absent. All information will be recorded on a designed form.

**Sample determination**
The sample size will be computed from the following formula

\[ N = \frac{Z^2 P (1-P)}{C^2} \]
Whereby  
N=Sample size  
P=Prevalence  
Z=Z value  
C=1-confidence level

Taking a confidence level of 95%, Z value of 1.96 and prevalence of 50%,

\[
N = \frac{1.96^2 \times 0.5(1-0.5)}{0.05}
\]

\[
= \frac{1.96^2 \times 0.5 \times 0.5}{0.05^2}
\]

=384.

Since the study population is less than 10,000, the sample can be moderated using the formula

\[
f_n = \frac{n}{1 + n/N}
\]

Where \(f_n\) = desired sample for a population less than 10,000

\(n\) = sample size derived for a population greater than 10,000

\(N\) = estimated size of population with the characteristic of interest

Taking \(N\) to be 500,

\[
f_n = \frac{384}{1 + 384/500}
\]

=384/1.768

=217

So, a minimum of 217 participants will be examined.
Sampling methods.

The sampling unit will be a student. Simple random sampling of the willing students will be applied appropriately to achieve the desired minimum sample size.

Inclusion criteria

All willing students between the agegroup 11-19 years.

Exclusion criteria

All students below 11 or above 19 years of age. All unwilling students will not be involved.

Logistics

The study is selectively chosen to be in the schools for convenience availability of the study population. It will therefore be time saving and cost effective.

Ethical considerations

- Ethical approval by the KNH/UON ethics, research and standard committee
- Permission will be sought from the schools’ board of governors through the principals
- Consent will be sought from the students
- Confidentiality of each participant’s information will be upheld
- Aseptic handling of participants will be maintained to prevent disease transmission