

Frequency of Orofacial Cleft in an Oral Diagnosis Clinic in A Tertiary Hospital a 5 Year Retrospective Study

MADUKWE IKECHUKWU UDO

Department Of Oral Surgery & Pathology, Faculty Of Dentistry, College Of Medical Sciences
University Of Benin City, Nigeria.

ABSTRACT:- Background: Orofacial clefts including cleft lip, cleft lip and palate and cleft palate alone, as well as median, lateral and oblique facial clefts are among the most common congenital anomalies. This study therefore aims at ascertaining the frequency of clefts in oral diagnosis clinic and proffers some biological explanations

Materials and Methods: A hospital –based study in oral diagnosis clinic using a non-confidential register for patients was used to ascertain the frequency of orofacial cleft.

Results: The study revealed a frequency of 1.35% and one in every seventy four persons had orofacial cleft. It is therefore recommended that there is the need for birth defect surveillance system in line with WHO 2003 recommendation starting from diagnostic clinics to regional population based-system.

Conclusion: We therefore suggest increased practitioners awareness, early identification as a *sin qua non* for early intervention. This will no doubt save children and parents from grave psychological morbidity.

Keywords:- cleft, frequency, orofacial,

I. Introduction

Orofacial clefts, including cleft lip, cleft lip and palate and cleft palate alone, as well as medial, lateral, (transversal) and oblique facial clefts are among the most common congenital anomalies¹ in which these children and their families experience serious psychological problems²In facial morphogenesis, neural crest cells migrate into the facial region, where they form the skeletal and connective tissue and all dental tissues except the enamel³. Generally the development of upper lip is from medial nasal and maxillary processes. Failure of merging result in cleft lip or it may extend to the primary palate resulting in cleft lip and primary palate. Involvement of palate entails, defective growth of palatal shelves or failure to attain a horizontal position, or contact failure or breakdown after fusion of shelves.² Golalipour et al.⁴ explored maternal risk factors to include multifactorial causes as genetic and environmental factors, gender, geographic location, nationality, nutritional and periconceptional consumption of folic acid⁽⁵⁻¹⁰⁾, tobacco, antiepileptic drugs and alcohol¹¹ contribute to clefts. Racial, ethnicity⁽¹²⁾, and consanguinity^{13,14} have also been implicated. This study therefore aims at ascertaining the frequency of clefts in oral diagnosis clinic and proffer some biological explanations

II. Materials And Methods

This is a hospital – based retrospective study in oral diagnosis and radiology department, University of Benin Teaching Hospital. This is one of the cleft referral and orofacial defects Centre. This was a retrospective study from January- December2010 to January-December 2014 from a non-confidential register for patients. Monthly records for patients with orofacial cleft were checked and confirmed with their hospital case notes with no reference to demographic details, as this bears no relevance to frequency details. The number of patients with orofacial cleft was documented excluding trauma induced.

III. Results

Out of 14,865 patients seen in the oral diagnosis clinic during the 5 year duration 201 had cleft of different forms representing 1.35% or one in every seventy four persons (1:74). These varied findings are cleft lip (fig 1), bilateral cleft lip (fig 2), cleft palate (fig 3), cleft lip and palate (fig 4) and oblique facial cleft (fig 5)



Fig 1: A clinical picture of a four month old male unilateral cleft lip patients.



Fig 2: A clinical picture of a four month old female bilateral cleft lip patients.



Fig 3: Clinical picture of 3 year old female cleft palate patients.



Fig 4: A clinical picture of a 7-month old male cleft lip and palate patient



Fig 5: Clinical picture of a 23 year old female patient with oblique facial cleft which was repaired surgically

IV. Discussion

The structures of the craniofacial region are largely derived from the neural crest cells, which undergo extensive migrations and interactions, in the facial region and they give rise to almost all the skeletal structures including the teeth (except enamel) and connective tissues. Interaction of neural crest cells and other cells with matrix and growth factors at various locations along the migratory path or at their destinations determines the differentiation of the cells. Failure of these neural crest cells to migrate, inadequate migration, failure to proliferate during migration and premature cell death or apoptosis serve as basis for the many syndromes, collectively known as neurocristopathies of which orofacial cleft is one. Our findings showed 1.35% of orofacial cleft in the study population representing one in every seventy four persons (1:74).

This is understandably so, because oral diagnosis clinic is a specialized centre where most referrals are made from paediatric, surgical and private clinics both within the state and outside. A normal birth is always regarded as a logical natural event, but when for any reason a deviation occurs, the result is very often looked upon with a sense of fear, horror and taboo. Craniofacial anomaly in the visible part of the human body, is unsightly making learning to live with this appearance a difficult task and most challenging. This is complicated by the fact that large population of births occur at home¹⁶ making infanticide in children with orofacial cleft likely.¹⁷ common among those facial deformities are cleft lip (fig 1) unilateral cleft lip is due to failure of the maxillary process on the affected side to join with the merged medial nasal processes. Bilateral cleft lip (fig 2). This is due to failure of the mesenchyme masses of the maxillary process to merge with the merged median nasal process on both sides. Cleft palate (fig 3) this is due to failure of the mesenchyme masses of the lateral palatine processes to meet and fuse with each other with the nasal septum and with the posterior border of the median palatal process (primary palate) Cleft lip and palate (fig 4) this is due to disturbances at any stage during palate development example, defective palatal shelf growth, failed or delayed elevation, and blocked fusion with or without lip. In general the etiology of orofacial clefts is theorized to be combination of factors associated with genes and environment.

The advent of gene targeting technology and basic conventional techniques using animal models has led to the identification of genes associated with known and unknown etiological factors. Characterization of the genomic sequences will greatly impact regulation of gene networks and pin point any variations in the different stages of craniofacial morphogenesis.²⁰

Orofacial cleft are common congenital deformities that often affect speech, hearing and esthetics and may at times lead to airway compromise. Maxillofacial surgeons and otolaryngologist are key members orofacial cleft team as currently advocated.

We therefore believe that further documentation of the frequency of this facial anomaly is apt and will definitely serve as a guide for future further preventing strategy and proper intervention.

V. Conclusion

This frequency is a clear indication that there is a compelling need for birth defect surveillance system as recommended by the WHO in 2003⁽²¹⁾ beginning with hospital-based birth defects surveillance system and then progressing to a regional population-based system, and in addition establishing Intercentre collaborative studies of the etiology of orofacial clefts which will serve as a template for required prevention strategy.⁽²²⁾ We therefore suggest increased practitioners awareness, early identification as a *sin qua non* for early intervention. This will no doubt save children and parents from grave psychological morbidity.

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Table 1 PERCENTAGE ORAL CLEFT 2010-2014

S/N	January– December each year	Number of patients	Number of orofacial cleft	Percentage
1	2010	2,865	44	0.30
2	2011	2,950	46	0.31
3	2012	2,864	33	0.22
4	2013	3021	53	0.36
5	2014	3165	25	0.17
Total	5 years	14,865	201	1.35

(1.35%;1:74)