

Oral mucosal lesions in children with and without cleft palate: a case control study

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ABSTRACT

Oral mucosal lesions are conditions affecting the oral cavity; they include candidiasis, recurrent herpetic lesions, recurrent aphthous stomatitis, hairy tongue and lichen planus. Recording and diagnosis of oral mucosal lesion requires a thorough familial history to be taken before proceeding to conduct a complete oral examination. The aim of the study was to determine the prevalence of oral mucosal lesions in children with cleft palate. A study was carried out by collecting data by reviewing patients data and analysing the data of 86000 patients between June 2019 and March 2020 at the private dental institute. The present study consists of 66 children divided into 2 groups: children with cleft palate and children without cleft palate. In both groups, presence of oral mucosal lesions were noted. Absence of oral mucosal lesions in both groups (children with cleft palate and children without cleft palate). The prevalence of oral mucosal lesions was compared for both case and control group were compared by Mann- Whitney U Test which gave a result of $p=1.000$. Within the limitations of the study, there is no evidence regarding oral mucosal lesions in children with and without cleft palate.

KEY WORDS: Cleft Palate, Oral Mucosal Lesion, Prevalence

INTRODUCTION

Cleft lip /cleft palate is known as a congenital craniofacial anomaly with cleft lip being recorded in 1556 by Pierse Franco and cleft lip and palate was discovered in 1691 by Hendrik Van Roonhuyzez. Although the earliest evidence . Although the earliest evidence of it in antiquity was discovered in Egyptian mummy.[1] There are roughly 7000 infants born with orofacial clefts in the U.S. annually.[2] Beyond the physical effects of the patient and family including disruptions of psychological functioning and decreased quality of life.[3]

Cleft lip and palate or cleft palate alone can be categorised as orofacial clefts. Cleft lip and palate is the second most common congenital birth defect in the world with only Down syndrome higher than them. The goals in surgical correction of orofacial clefts are to optimize feeding, facial growth, and speech and language development.[4] Successful repair of the cleft lip is simultaneously rewarding and challenging by itself.

Orofacial clefts (OFC) is the most common craniofacial birth defect and it is the most congenital malformation which occurs in humans. Orofacial clefts exhibit both ethnic and geographic variations. Incidence of cleft lip and palate (CLP) ranges from 0.25 to 2.29 per 1000 births in India.[5] Orofacial clefts are caused by various factors such as infection, toxicity, poor diet, hormonal imbalance and genetic interference. Among these factors, genes play an important role in the formation of cleft lip and palate.[6] The development of craniofacial structure is a coordinated process involving the growth of multiple independently derived prominence called primordial. Incomplete fusion of these prominence during 4th to 12th week during intra- uterine life leads to cleft lip, cleft palate or combination of both .[5]

Based on the embryological characteristics, varieties of cleft lip and cleft palate may be categorised as:

- 1) Those involving the lip and alveolus
- 2) Those involving the lip and palate
- 3) Those in which palate alone is affected
- 4) Congenital insufficiency of the palate. [6]

Dental complications of orofacial clefts includes congenitally missing teeth, ectopic eruption, supernumerary teeth, anomalies in size and shape of the tooth such as microdontia and macrodontia, peg laterals, developmental enamel defects such as enamel hypoplasia, fused tooth, malocclusions such as deep bite, anterior or posterior crossbite, crowding and spacing of teeth.[7] In addition to dental complications, dental lesions such as dental caries and dental caries with pulpitis are more prevalent as the bacteria growth is more colonised and the oral mucosal lesion acts as a breeding ground for further infections to take place [8] [9] [10]

Oral mucosal lesions are defined as any abnormal alteration in colour, surface aspect, loss of integrity or swelling on the oral mucosal surfaces. Oral mucosal lesions are benign and they require no active treatment and symptomatic relief is only required. It may interfere with physiologic functions such as mastication, swallowing and speech. It also causes burning, irritation and pain during food consumption.[11] Oral mucosal lesion is caused by various etiological factors such as vital, bacterial or fungal infections, local trauma or irritation, systemic disease, excessive consumption of tobacco, betel quid and alcohol.[12]. A study conducted by Alessandra Majorana et al [13] in 2010 evaluated the prevalence of oral mucosal lesions among 10128 children aged between 0-12 years in Italy and concluded that 28.9% of children had some kind of oral mucosal lesion. According to this study, the most common oral mucosal lesion were oral candidiasis (28.4%) children, followed by traumatic lesions (17.8%), erythema multiforme that affected (0.9%) of children were the least common form of oral mucosal lesion.

Traumatic oral soft tissue lesions due to habits that tend to cause severe injuries such as lip and cheek biting, fractured endodontic instruments, orthodontic appliances, food burns, sharp edges of wires in space maintainers.[14] [15] [16]. Previously our team has a rich experience in working on various research projects across multiple disciplines The [17–19][20–31]

There is surprisingly little information on the prevalence of oral mucosal lesions and extraoral lesions among cleft palate and/or lip patients in India. This initiated our present study to determine the oral mucosal lesions in children with and without cleft palate.

MATERIALS AND METHODS

The retrospective study was conducted under a hospital based university setting. Ethical approval for this study was obtained from the institutional ethical committee (ethical approval number:SDC/SIHEC/2020/DIASDATA/0619-0320). Consent to use treatment records for research purposes were obtained from patient/ guardian at the time of patient entry into the university for dental needs. The retrospective data were collected by obtaining and analysing the 89000 dental case records of the university from June 2019 to March 2020. The inclusion criteria for the current study were children with cleft palate only, children between the age of 6 months to 17 years age, complete photographic and written records regarding the complete intra-oral examination of the patient. Age and gender matched controls i.e.

children without cleft palate, were taken according to the relevant cases obtained from the inclusion criteria. The exclusion criteria were incomplete and censored dental records and children below the age of 6 months were excluded. The total case acquired for the study was 66, with 33 patients from the case group and 33 patients from the control group. The selected case and control group were examined by three people; one reviewer, one guide and one researcher. The patients' case sheets were reviewed thoroughly. Cross checking of data including digital entry and intra oral photographs was done by an additional reviewer and as a measure to minimise sampling bias, samples for the group were picked by simple random sampling. Digital entry of clinical examinations and intra oral photographs of selected subjects were assessed and this included the assessment of oral mucosal lesion as mentioned before by the examiner based on intraoral photographs and clinical examination data for each tooth. The examiner was trained to add data of oral mucosal lesion as present or absent for both case and control group by tabulation using excel software. Data analysis was done using SPSS PC Version 23.0 (IBM;2016) software for statistics. The incidence of oral mucosal lesions for both case and control group were compared by Mann-Whitney U Test which gave a result of $p=1.000$ significance.

RESULTS

The final study population included 66 patients with 33 patients in the case group (children with cleft palate) and 33 children in the control group (children without cleft palate). [Figure 1]. In the case group, out of the 33 patients, 17 (25.8%) were males and 16 (24.2%) were females. Similar distribution was done while selecting the control group. [Figure 2]. There is an absence of oral mucosal lesions in all the cases in both groups (children with and without cleft palate) [Figure 3]. On comparison of data by Mann-Whitney U Test. statistically significant differences were obtained. (p -value=1.000).

DISCUSSION

Oral mucosa serves as a protective barrier against trauma, pathogens and carcinogenic agents. Oral cavities can be considered as a gateway into the digestive system. Sometimes, oral lesions are the first indication of a systemic disease.[32] Andres Pinto et al, in 2014 divided paediatric soft tissue oral lesions into several categories: 1. Developmental lesions: geographic tongue, fissured tongue, retrocuspid papilla, gingival overgrowth.2. Mucosal changes (colour): white lesions- leukoderma, pseudomembranous candidiasis, white spongy nevus. Red and/ or white lesions: petechiae, purpura, ecchymosis, erythematous candidiasis. 3. Brown-black lesions such as physiologic pigmentation, amalgam tattoo, melanotic nevus. 4. Soft tissue nodules- inflammatory/ reactive lesions: mucocele, peripheral ossifying fibroma, pyogenic granuloma. 5. Cysts such as eruption cyst, 6. Ulceration- traumatic ulcers, aphthous ulcers. 7. Benign tumors- hemangioma, lymphatic malformation, fibroma, benign neoplasm and for infections, herpes simplex virus. [33]

Dental neglect by the caretaker of the children can lead to the formation of oral mucosal lesions as bacterial growth are more prominent in these environments, the most commonly seen oral mucosal lesion from abuse and neglect cases was ranula formation due to contact between the tooth and the lips through a hard force. [34] [35] [36] Treatments to prevent further spread of oral mucosal lesion includes the fluoride application, proper oral hygiene and prevention and early management of Early Childhood Caries to prevent the spread and growth of more complex bacterial cultures in the oral cavity. [37] [38] The usage of fluoridated toothpaste would increase the fluoride content in the enamel layer, preventing the spread of streptococcus mutans causing a prevalence of dental caries.[19] [39]

Bazerra et al [35] found that childhood oral mucosal lesions among 104 patients ranging from 0 to 5 years children to be 2.3% by observing their dental records.[40] According to this study, the most common oral mucosal lesions were Bohn nodules (37%) followed by candidiasis (25%) and benign migratory glossitis were the least common with 21%. Bessa et al found that incidence of childhood mucosal lesions among 0 to 4 year olds to be 24.9% and the most common lesions were geographic tongue (9.8%) followed by bite injuries (6.1%).[41] The most common lesion was caused by *Candida albicans*.

According to Maxill, bottle use was a significant predictor of maternal and infant oral colonization. The simplest theory behind this is that a bottle acts as a vector for the candida in the environment 40-60% of the infants carry candida organism in their mouth and within the first 18 months of life, 24% of the infants developed oral thrush.[42] [43]

Traumatic ulcers develop from various injuries such as physical, thermal or chemical. Accidental biting during mastication or consumption of hot food may cause traumatic ulcerations. Iatrogenic damages caused by dental

treatments also cause traumatic ulceration.[44]. Frictional keratosis also called mastico buccasum are white patches caused due to traumatic tooth brushing (toothbrush keratosis) due to constant rubbing of mucosa, frictional keratosis can occur.[45] [46] [30]

Leukodema is a white lesion which is found on the buccal and/or the labial mucosa. It can be unilateral or bilateral and is associated with smoking and local irritation. Prevalence differs from 0.96% to 58% and highest prevalence noted in African populations.[47]

In our study, we found that children with palate as well as children without cleft palate both had absence of oral mucosal lesions with the p value >0.05 is not significant. There were a few contradicting studies by Amandeep Chopra et al, in 2014, which found that children with clefts have a higher prevalence of anterior open-bite, increased overjet and presence of oral mucosal lesions.[48] Meral Umur et al, in 2015 carried out a study in Turkey stated that among all oral mucosal lesions in patients with cleft palate, fissured tongue (3.4%) was the most frequent lesion followed by traumatic ulcer (3.2%) and presence of cheek biting in (2.5%) of the study population.[49] The consensus of our study disagreed due to a smaller sample size as well as the geographic restrictions present while carrying out this study.

The advantages of the present study were that this was a case- control study with age and gender matched control to provide better results and high internal validity. The limitations found in the study are geographic restrictions as the patients are from around the same region. Besides, there was only a single ethnicity as the group of people are from the same ethnicity group. Unicentric study, small sample sizes and indirect clinical observation are also some of the challenges faced while conducting our study. The future scope of this study could involve studies with a larger sample size for the case group, that is not confined to a particular geographic region.

CONCLUSION:

Within the limitations of the present study, there is no evidence of oral mucosal lesions in children with and without cleft palate. However careful clinical examination needs to be performed for proper diagnosis of oral mucosal lesion despite the presence of absence of cleft palate.

AUTHOR CONTRIBUTIONS

- Design - Sivesh S, Vignesh Ravindran
- Intellectual content - Vignesh Ravindran
- Data collection - Sivesh S
- Data analysis - Vignesh Ravindran, Visalakshi Ramanathan
- Manuscript writing - Sivesh S
- Manuscript editing - Vignesh Ravindran, Visalakshi Ramanathan

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CONFLICT OF INTEREST

The authors declare that there are no conflict of interest

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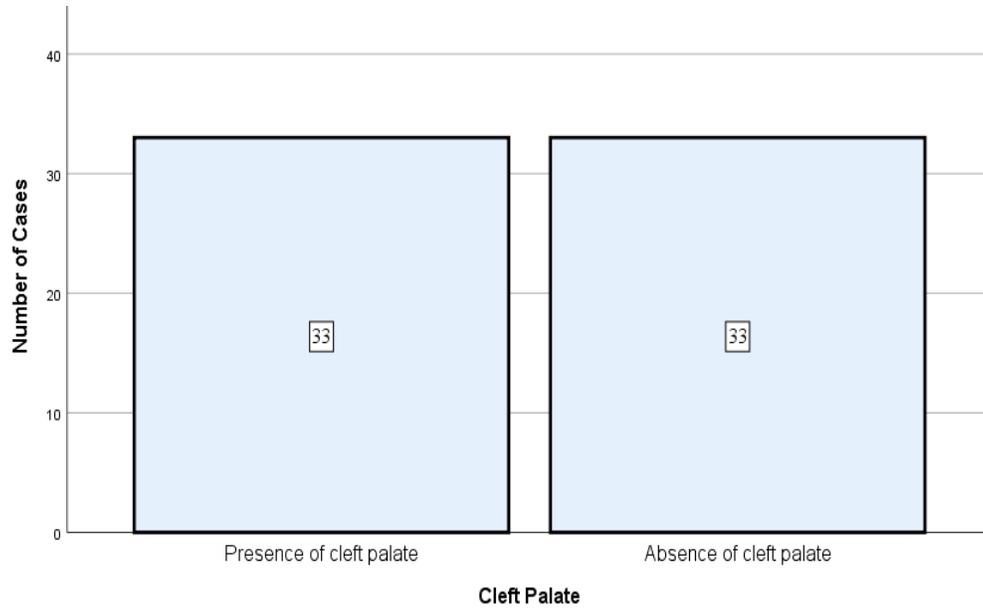


Figure 1: The bar graph shows the frequency distribution of cases in the case group (children with cleft palate) and the control group (children without cleft palate). X-axis represents the presence or absence of cleft palate and Y-axis represents the number of cases. Notice the equal distribution of cases for both the groups.

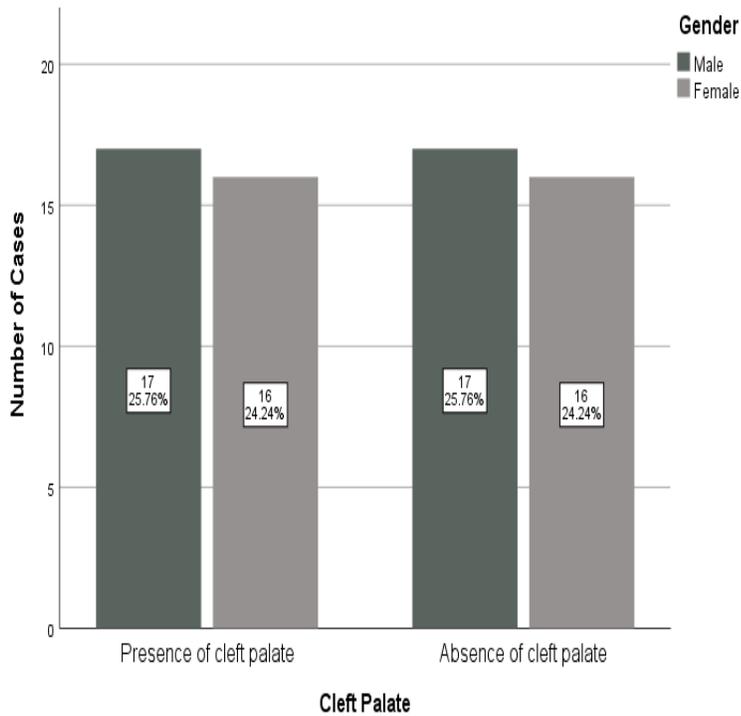


Figure 2: The bar graph represents the gender distribution of cases in the case group (children with cleft palate) and the control group (children without cleft palate). X-axis represents the presence or absence of cleft palate and Y-axis represents the number of cases; darker grey represents males and lighter grey represents females. Notice the equal distribution of gender for both the groups.

Oral mucosal lesions in children with and without cleft palate: a case control study

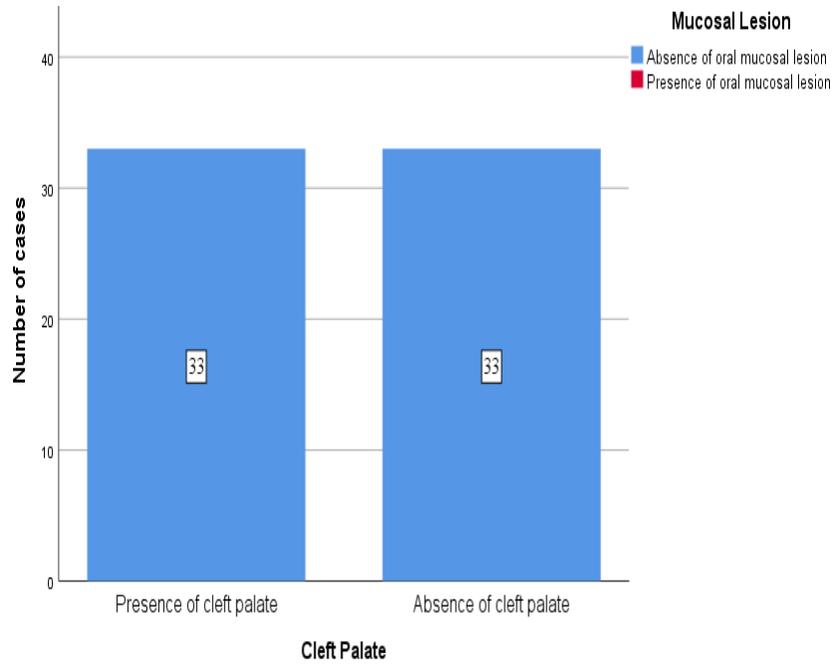


Figure 3: The bar graph depicts the association of presence or absence of oral mucosal lesion in children with cleft palate and children without cleft palate. X-axis represents the presence or absence of cleft palate and Y-axis represents the number of cases. None of the cases in both the groups had oral mucosal lesions (blue), which was highly significant. (Mann-Whitney test, p-value=1.000 - highly significant)