Craniofacial anomalies and the role of the dental therapist: the link between cleft lip, palate and alveolus, and periodontal disease. A literature review

Chloe Smart shares a concise version of the literature review she conducted in the final year of her degree in Oral Health Science at the University of the Highlands and Islands (UHI).

Question

Are patients born with a cleft classification, compared to those without the anomaly, predisposed to periodontal disease?

Introduction

An orofacial cleft presents as one of the most prevalent congenital anomalies, occurring in approximately one in every 700 births.[1] The presence of varying classifications of cleft at birth can result in other anomalies such as hypodontia, hyperdontia, misshapen or malformed teeth. This in turn can lead to rotation, tilting and crowding of the dental hard tissues.[2]

According to de Almeida *et al.*[3] cleft lip and palate (CLAP) patients, particularly those who have undergone reconstructive surgeries, can also present with mucogingival alterations and can have clinical attachment loss in areas surrounding the cleft due to presence of scar tissue after reconstructive surgery. It is believed that the occurrence of clefts is due to a combination of syndromic and non-syndromic genetics, maternal diseases, stress as well as the intake of alcohol, drug use and smoking during the foetal development period.[4]

The treatment of CLAP involves reconstructive surgery of the associated tissues to gain optimum aesthetics and functionality from the oral cavity. Multidisciplinary teams are utilised greatly within the long-term care of CLAP patients. These can include dental and medical specialities such as: oral and plastic

surgery, orthodontics, paediatric dentistry, prosthodontics and psychiatry. Associated healthcare fields such as audiology, psychology and speech therapy provide optimal long-term care for these patients also.[4]

Periodontal disease is the sixth most common disease present within the global population.[5] The combined published guidance produced by the British Society of Periodontology and the British Society of Paediatric Dentistry aims to outline a method to screen patients under 18 years of age for periodontal issues at the earliest opportunity.[6] It aims to utilise specialist services and pathways if required for these patients to diagnose as early as possible and treat accordingly.

As well as undertaking a complete periodontal screening for all patients, dental therapists (DTs) play a significant role in paediatric dentistry which is implemented through the *Scope of practice* set out by the General Dental Council (GDC).[7] Dental therapists are in a prime position to monitor and maintain a cleft patient's oral status. The subsequent dental anomalies that may have occurred due to the presence of a cleft may limit manual dexterity for oral care. For this reason, the role of a dental therapist is vital as the child progresses into adolescence to ensure anomalies such as malpositioned teeth don't lead to any form of oral disease.[8]

Methodology

An extensive literature search was carried out on the relation between cleft and the prevalence of a periodontal disease diagnosis due to the orofacial anomaly. To obtain the most relevant literature to review, I utilised databases that comprised of PubMed, Wiley Online Library and *The Cleft Palate-Craniofacial Journal*. Using Boolean logic as well as truncators allowed for further scope. Within these databases I initiated inclusion criteria for relevant findings. These included:

- Works published between 1999 and 2021
- Published in English language
- Relevant studies: systematic reviews/ meta-analysis; randomised control trials; cohort studies; case control studies; cross-sectional surveys; case reports.

Papers obtained through the database search were critically appraised with use of the Critical Appraisal Skills Programme[9] and AXIS[10] tools. After the selected papers had been critically appraised, the 25 papers were further narrowed down to nine. These published works formed the understanding and basis of this literature review. Relevant obtained literature includes: one systematic review, three case control studies, one cohort study and four cross-sectional studies. These all sit on varying levels of the hierarchy of evidence.

Results

The results were comprised following the review of nine relevant literatures and displayed the following themes:

- The presence of a variation of cleft highlighted the prevalence of plaque stagnation in seven studies. When the classification was of cleft lip and palate (CLAP), and cleft lip and alveolus (CLPA), the plaque accumulation was more pronounced compared to cleft lip (CL) or cleft palate (CP) alone
- Bleeding on probing (BOP) was seen more in cleft patients than in those without the anomaly
- A common theme was derived from the periodontal pocket depth (PPD) of eight studies which showed that there was an increased prevalence in PPD with the presence of a cleft. This was further identified when the classification involved a cleft lip compared to a cleft palate
- In studies where mobility was clinically recorded, 100% of the literature showed presence of mobility in teeth surrounding or neighbouring the cleft
- Bacteria pools were recorded as a parameter in two studies. Both stated there was an increase in sub and supragingival bacteria in cases of cleft compared to existing oral flora.

Discussion

Reviewed were three case control studies in which the cases were patients with cleft lip and palate (CLP) who were matched and compared to the general population. Across all three studies, the methodology used included plaque (PL) and bleeding (BL) indices as well as detection and record of PPD. A study conducted by Perdikogianni *et al.* in 2009 found that the cases had significantly higher plaque accumulation than those in the control group with a p-value of $p < 0.005$.[11] This was detected more in the maxillary anterior sextant of both groups but even as both groups' oral health (OH) improved, the highest prevalence of plaque stagnation was detected within the case group. This was of a similar finding in a study by Al-Wahadni, Alhaija and Al-Omari in 2005 where there was also a significant difference between the two groups and the highest mean of PL detected was in cases with a p-value of $p = 0.16$.[12] Plaque stagnation was also apparent in CLP patients in the study conducted by Passinato Gheller *et al.* in 2021 where cases were compared to their matched general population.[13]

PPD measurements are used to detect PD and to what extent the disease presents. In all three case control studies, PPD was clinically recorded, and it was found that there was a significant difference between cases and controls in all studies conducted. Perdikogianni *et al.*[11] detected that there was a vast difference in PPD between teeth neighbouring the cleft (NC) and teeth in the cleft (IC) in comparison to the corresponding teeth in the control group ($p < 0.007$). Passinato Gheller *et al.*[13] had a p-value of $p < 0.001$ when the PPD of CLP patients were

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compared to the control group.

Tooth mobility can be caused by PD due to the loss of surrounding alveolar bone. Within the case control studies reviewed, only one used the clinical parameter of mobility. Perdikogianni *et al.*[11] concluded that teeth NC or teeth IC presented with grade 1–3 mobility in up to 65% of teeth examined.

A cross-sectional survey conducted by de Almeida *et al.* in 2009 compared cleft lip and alveolus (CLA) with CLP in unilateral left, right and bilateral classes among 400 participants.[14] It was found that the mean PPD was <3 mm indicating no periodontal involvement. Out of all teeth examined for PPD only 0.18% were >6 mm, demonstrating periodontal destruction. Furthermore, only 0.07% were found within the cleft region. With regards to PL accumulation, the sextants in the region of the cleft did not present with more PL stagnation than other non-cleft sextants. According to the results of this study, it would indicate that the presence of a cleft, or its classification, does not dictate the periodontal status of a patient. de Almeida *et al.*[14] detected no other clinical measurement to be of significant difference between CLA and CLP. When detecting BOP, moderate inflammation was identified in the cleft sextants but not more so than any other sextants examined. This study discussed that the results were not as expected due to patients with orofacial anomalies usually having scar tissue and lack of keratinised mucosa; this in-turn would accumulate to a higher plaque accumulation.

Schultes, Gaggl and Kärcher *et al.*[15] compared 60 patients, 30 CP and 30 unilateral cleft lip, palate and alveolus (UCLPA), and assessed the periodontal status by use of a CPITN probe. Fifty percent of patients with UCLPA presented with PPD >5.5 mm with a p-value of $p < 0.05$. It was found that the highest prevalence of periodontal destruction was detected in 80% of teeth next to the cleft, where a CPITN code 4 was identified. This was similar in patients with CP also, with the highest incidence of periodontal destruction in the maxillary anterior sextant. Within this study, it is discussed that a possible risk factor for periodontal destruction is orthodontic interference due to a reduced height of the alveolar presence and corresponding clinical attachment loss (CAL).

Boloor and Thomas conducted a cross-sectional survey with 30 CP, 30 CL and 30 CLPA patients.[16] Results from this study indicated alveolus involvement influenced higher PL, BL and PPD compared to the other subjects where the p-value was $p = 0.001$. This in particular was identified in the maxillary anterior sextant.

CP and UCLPA patients were compared to evaluate their periodontal status in a study by Mutthineni, Nutalspati and Kasagani.[17] In all clinical parameters recorded, PL, BL, CAL, PPD and mobility were identified with a higher prevalence in UCLPA patients compared to CP. This study was the only one reviewed in which radiographs were used as a clinical parameter. These detailed that patients with UCLPA had lower bone height and therefore increased CAL of involved teeth. The authors discussed the reduced presence of attached gingiva correlates to sensitivity of the mucosal tissue in patients with cleft. This is a risk factor for high PL and BL scores as well as possible periodontal destruction.

The study by Salvi, Brägger and Lang[18] followed patients over a 14-year period and were examined on three separate occasions (1979, 1987 and 1993). The participants consisted of two groups: 40 CLAP and 40 CL/CP patients in 1979, but due to unavailability of subjects, only 13 CLAP and 13 CL/CP patients were re-examined at the end of the 14-year period. The clinical parameters used were PL, BOP, PPD and probing attachment level (PAL) which is similar to previous studies discussed within this review. Between 1979 and 1993, the PPD of the cleft site in CLAP patients had increased by significant amount (1.72±1.08 mm) when compared to other control sites where the p-value was set at $p < 0.05$. Comparing the CLAP group with CL/CP group, both showed a mean PPD of 0.57±0.21 mm which is of significant difference. The methodology used within this study detailed that the participants had no supportive phase therapy (SPT) intervention during the 14-year period. The conclusions of this study yielded that without regular SPT, patients with cleft of any classification were unable to maintain the periodontal conditions first recorded in 1979. As this study had no comparison group, it could be a possible factor that the PPD increase could be attributed to participant age.

Marzouk *et al.* conducted a systematic review in which 23 studies were used to assess the periodontal clinical measures of the participants.[19] The meta-analysis carried out concluded that patients with CL/P, compared to those without the anomaly, presented with higher PL stagnation and BOP. This was in the maxillary anterior region, which correlates to various other studies within this literature review. This is in conjunction with the PPD recorded within the studies analysed, where the CL/P group presented with higher PPD compared to the unaffected group, measured also by radiographic report.

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Two papers within this review conducted analysis of bacteria pools, Passinato Ghellar *et al.*[13] and Perdikogianni *et al.*,[11] which displayed similar findings. Perdikogianni *et al.*[11] discussed the presence of harbouring bacteria, spirochaetes, which were more prevalent in teeth IC in comparison to NC and the control group. Passinato Gheller *et al.*[13] compared pathogens in oral biofilm. The presence of *Aggregatibacter actinomycetemcomitans* ($p=0.02$) and *Porphyromanas gingivalis* ($p < 0.001$) was significantly higher in CLP patients than their controls.

Within the reviewed literature, ethical approval and consent was not gained in every study. The same clinician was only utilised in three studies and within the systematic review, only 13 out of 23 employed use of the same clinician for clinical readings. This presents possible bias and questions the confidence

intervals within the studies reviewed.

The varied clinical parameters used within the methodology of these studies provided limitation with regards to obtaining full periodontal status of participants. As four types of study design were reviewed and various classifications of cleft were compared, it was difficult to obtain clear and concise results from the review of literature. Furthermore, the studies reviewed indicate OH status to be the predominant risk factor found within cleft patients but in conjunction to the association of developing periodontal disease. The inclusion criteria of my search, as well as focused PICO question and word count also provided limitation upon undertaking this review.

Conclusion

Comparing the classifications of cleft, it was concluded that the presence of a cleft lip involving alveolar destruction contributed greatly to the periodontal status of affected teeth in this region. It is apparent that PL stagnation and BOP are prevalent in patients with a cleft. Both parameters as well as PPD could be attributed to surgical and orthodontic interference, presence of residual scar tissue, hard tissue rotation and misalignment in the cleft site. Fear of brushing the cleft site also hinders the oral health status of the patient. It is paramount that OHI and patient education is delivered at every visit to ensure biofilm disruption occurs daily.

As a student DT, paediatric dentistry is vast within our scope of practice. It has been demonstrated through review of relevant research that identification of a patient's oral status is vital in preventing progression of disease. Assessing children early allows for tailored prevention and strategic and routine recall to prevent possible lapse.

Recommendations

As a student DT, I am constantly learning and developing my knowledge with various patient bases. I have never treated a patient with a cleft, but patients with a cleft classification could present within my care at any age and I would like to be aware if this could be a risk factor for periodontal disease. This knowledge would be beneficial to have to communicate to my peers and colleagues within the workplace.

For a future review of the literature, I would obtain studies that use the same clinical parameters and methodology, so that results are tailored, and clear comparison can be made. To diagnose patients with PD, I would put forth that the 2017 BSP periodontal classifications[20] are used for relevant studies, again, for uniform and comparable results.

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