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From COVID-19 to "JAPA"

In Nigeria, "JAPA" refers to emigration for "greener pastures" especially in reference to young individuals who in search of better opportunities have decided to relocate abroad to improve their lots. Although the internet has ascribed an acronym status to the term as representing "Just Another Person Abroad" or "Just Absconding Probably to Abroad", the original word comes from the Nigerian language meaning to escape! There are multiple perspectives on the push-pull factors for the emigration of professionals, but in the Dentistry/oral health context, the implications are rather serious.

As young dental professionals flee the country, the immediate implication is brain drain in the dental/oral health sector. With the already poor outlook of oral health indices in Nigeria, the effect of this further shortage is rather frightening. The mass exodus of dental professionals limits the availability and accessibility of dental services in Nigeria especially in the rural and undeserved communities. The obvious result is reduced access and increased unmet dental/oral health needs among our teeming populations.

It is projected that the remaining workforce will suffer decreased morale and be "tempted" to emigrate at the earliest opportunity. Even those who decide to stay back are likely to suffer burnout from increased workload. Emigration creates a skills gap within the local dental workforce which is difficult to fill in the short term. As with decreased moral, taking on unhealthy workloads is a likely natural consequence with the attendant problems.

Emigrating dentists do so with their skills and expertise. The impact on the development of dentistry in the areas of capacity building, research and future planning in colossal! Nigeria must improve the working conditions of its dentists through governmental and non-governmental interventions and collaboration. Private-public partnership needs to be intensified and job opportunities must be increased to give young dentists hope in Nigeria.

Above all, there must be a national reorientation to teach Nigerians to believe in Nigeria and express confidence in its future. Leadership at all levels must show a commitment to an egalitarian unified, corruption-free, merit-based country. Then and only then will we stem the tide of the current deluge of harmful emigration!

S. Olusegun Nwhator

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NIGERIAN DENTAL ASSOCIATION

Teeth Avulsion Secondary to Oro-Tracheal Intubation in a Tertiary Healthcare Facility: A Case Series

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Abstract

General anaesthesia using endo-tracheal intubation, carried out to provide pain-free, surgical and medical procedures, sometimes results in Traumatic Dental Injuries [TDIs]. Among the rarely reported TDI complication is tooth avulsion. We present two cases of such complications, factors which can be responsible, management of such cases, and the need to educate medical and paramedical personnel on what to do if such complications arise before definitive management can be carried out by the dentist. Such awareness will go a long way in preventing unnecessary litigations which might likely occur from such routine procedures.

Key words: *Traumatic Dental Injuries, oro-tracheal intubation, Tooth Avulsion*

Introduction

The advancement in anaesthetic practices has revolutionised the practice of modern-day medicine as it provides pain-free surgical and medical procedures. The primary goal of general anaesthesia is rendering a patient unconscious and unable to feel painful stimuli, while controlling autonomic reflexes.[1]. This procedure is carried out with endotracheal intubation using endotracheal tubes and laryngoscopes to access the airway via the oro-trachea for administering anaesthetic medications and mechanical ventilation during surgeries.

Among the complications which can occur during this procedure are Traumatic Dental Injuries [TDIs].[2] Previous retrospective studies have shown a prevalence range of between 0.02-0.05%[3,4] for TDIs while a prospective study reported a higher prevalence of 25.0%[4]. These peri-anaesthetic dental injuries have also been reported as a leading cause of medico-legal claims following anaesthesia, accounting for about one third of medico-legal cases according to a report.[4] Such injuries include enamel/tooth fractures, tooth avulsion, concussion injuries, subluxation, intrusion, severe form of crown

fracture, anterior dislocation of mandibular condyle, and a variety of pressured-induced lesions of the oral cavity, and soft tissue[5,6]. The effects of such TDIs include low self-esteem, decreased quality of life, speech impediment and some degree of functional disability.

The aim of these two case reports is to show avulsion of maxillary central incisor teeth, an uncommon Traumatic Dental Injury (TDI) following oro- tracheal intubation, its management, and the importance of educating medical personnel on what to do whenever such complication arises.

Case presentations

Case 1: A forty-eight (48) year old woman presented to the dental clinic on account of avulsed upper right and left central incisors (11 and 21) following traumatic intubation (Fig1) during a general anaesthetic procedure one month previously. Patient claimed she was not given the avulsed teeth which could possibly have been re-implanted and splinted. However, she claimed there was a midline space between the 11 and 21.

Intra-oral examination revealed missing 11 and 21, grade 1 mobility of 12 using Miller's grading system, missing 14 and 15 which she claimed has been longstanding, anterior open bite, and Class 11 Angle's malocclusion incisal relationship. An assessment of Ellis Class V injury secondary to traumatic intubation was made on missing 11 and 21.

Periapical Radiograph of teeth number 12, 22 revealed empty sockets of 11 and 21 with no widening of their periodontal ligament (PDL) spaces, nor apical radiolucency of 12 and 22. Due to lack of facility for fabrication of an implant-supported restoration coupled with lack of financial capability for a fixed denture fabrication [fixed PFM bridge], upper and lower alginate impressions were taken for the fabrication of acrylic upper replaceable partial denture (URPD) to replace the missing teeth. The denture was delivered at the next appointment.

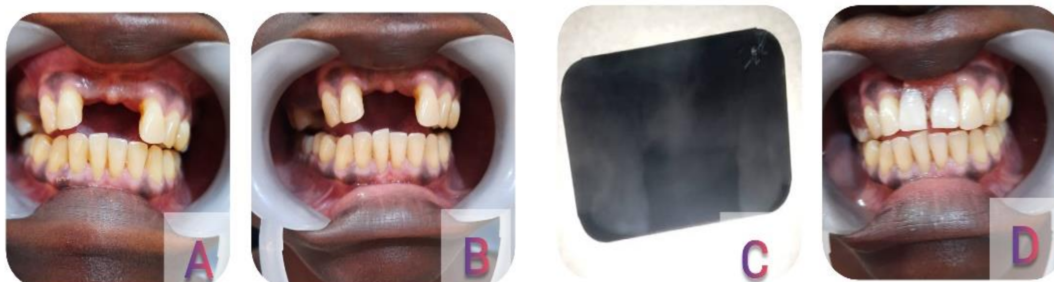

Fig1: A shows missing (11,12), anterior open bite; B shows the edentulous spaces of the missing upper central incisors edentulous space; C shows periapical radiograph (PAR) revealing the empty

sockets of 11 and 21 with no widening of the PDL spaces nor apical radiolucency of 12 and 22; D shows upper removable partial denture replacing 11 and 21.

Case 2: A forty-six (46) year old woman who presented at the dental clinic on referral from the Obstetrics & Gynaecology department, following avulsion of the upper right central incisor secondary to traumatic intubation from an elective surgery she underwent two days prior to presentation. She came along with the avulsed tooth already sealed inside a nylon envelope.

She was asked to spit her saliva inside the nylon envelope, while temporarily agitating the tooth in the envelope in order to remove loose debris and visible contamination. Comprehensive history was thereafter taken and thorough examination was done. The examination revealed presence of granulation tissue in the socket of tooth number 11 with incomplete re-epithelialization. extraction socket with There was presence of midline diastema measuring about 1cm with presence of high frenal attachment. The teeth numbers 12 and 22 are pegged shape and have about 1cm of spacing distal to each of them. An assessment of Ellis class V injury resulting from traumatic intubation was made.

Local anaesthesia was administered and the socket was irrigated with normal saline. The avulsed tooth was replanted carefully with slight digital pressure. Its correct position was verified clinically and radiographically, and splinted with flexible 0.5mm^2 stainless steel wire. Composite splinting of the labial surface of 11 and 12 was done to further reinforce the stability of the splinted tooth within the socket. Pre-operative root canal therapy (RCT) could not be carried out due to the patient's poor medical condition as she was not fully ambulatory. She was placed on antibiotics, analgesic, and chlorhexidine mouthwash rinse for a week. RCT was subsequently commenced on the tooth after re-implantation of 11 and non-setting calcium hydroxide intracanal dressing was placed.

The second visit of RCT was done 4 weeks after the first visit due to the patient defaulting on her appointment. Since there was no fresh history of pain or any associated complaints, the flexible stainless-steel wire and composite splinting was removed. The canal was obturated with gutter percha and AH26 sealant while the access cavity was restored with Glass Ionomer Cement (GIC); post-operative radiographs were also taken. The patient was asked to come for post-op review one week later. However, she failed to show up for review and all efforts to reach her on phone or trace her proved abortive.

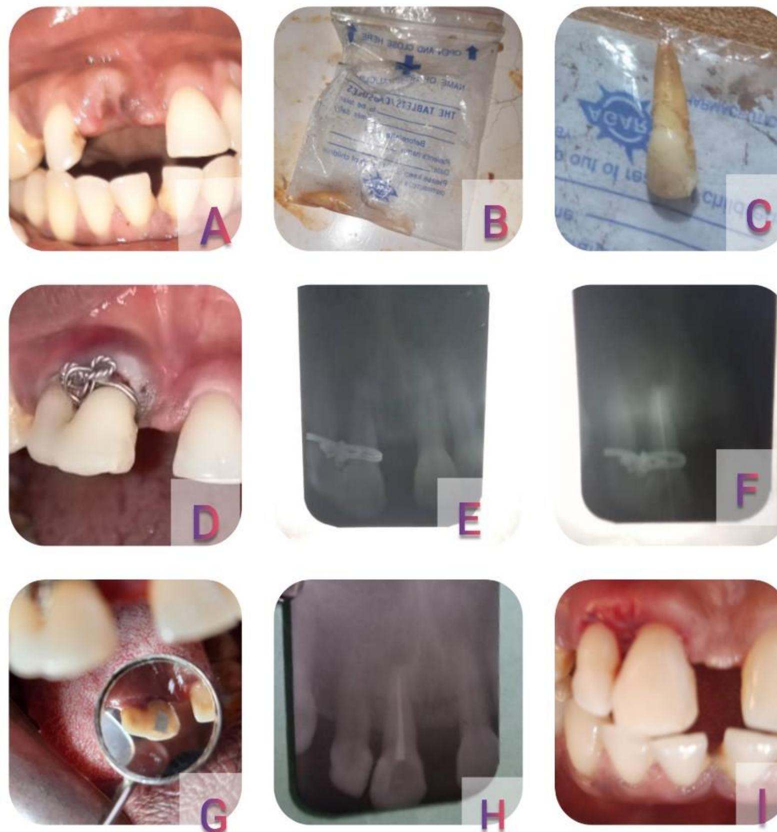

Fig 2: **A**. Edentulous space from avulsed 11 with low labial frenal attachment ; **B.** Avulsed upper tooth in saliva as medium storage; **C.** Avulsed tooth out for reimplantation; **D.** Avulsed 11 to be re-implanted in the refreshed socket, re-implanted 11, 12 splinted with eyelet wire and reinforced with composite, midline diastema, and low labial frenal attachment; **E.** PAR shows splinted 11, 12 periapical radiolucency on the apex of 11; **F.** PAR showing initial working length of 11 file size 15 with splint insitu; **G.** Access cavity opening for RCT with ZnOE; **H.** Periapical radiographs showing obturated 11; **I.** Obturated tooth and removal of stainless steel wiring and composite splinting.

Discussion

Two cases, all females, in the 4th to 5th decade of life, were presented to the clinic between July and August 2022. The affected teeth are maxillary central incisors (11, 12).

During laryngoscopy, the blades of the laryngoscope might hit against the upper centrals, or the anesthetist might use the upper centrals as fulcrum to depress the tongue thereby damaging the teeth, resulting in their avulsion. It can also occur during extubation when a patient involuntarily bites on the oropharyngeal airway using the anterior teeth as a fulcrum[5]. Previous study shows that upper centrals are the commonest teeth involved in traumatic avulsion of the anterior teeth following oro-tracheal

intubation[2]. Hence, there is a possibility of inadvertent dental injury (microscopic) to the upper or the lower incisors which might not result in injuries that are obvious (macroscopic) but may later result in pulpal necrosis with time. A detailed examination of the patient's mouth pre-operatively by the dental surgeon can help in identifying the teeth at risk of damage. And since maxillary and mandibular anterior teeth are more prone to injuries, these teeth should be properly inspected for any mobility or crown fractures before intubation commences.

The two index patients had anterior open bite, midline diastema, generalised poor oral hygiene, and proclined upper central incisors. These findings agree with previous studies which identify these as predisposing factors to TDIs following oro-tracheal intubation.[5,7]

The management followed the 2020 International Association of Dental Traumatology Guidelines for the management of TDIs. The guidelines recommended two weeks of stabilization with short-term passive, flexible splints for replanted tooth, and an additional one week if the avulsed tooth is unable to remain in the correct position[8]. The flexible splint encourages periodontal and pulp healing if the replanted tooth is subjected to slight mobility and function. An animal study revealed that 60% of the mechanical properties of the injured ligament return two weeks following injury.[9]

The patient returned seven weeks post-splinting, claiming inability to transport herself from her city of residence to the clinic due to financial reasons, delay in ambulation due to slow recovery, and gradual stability of the replanted tooth resulting in less urgency to come for a review. The long duration between the day of the splinting and the presentation has no effect on the likelihood of successful periodontal healing. In this case, due to delayed presentation, the expected outcome is ankylosis-related (replacement) root resorption not periodontal healing.

There are evidence that replantation restores esthetics and function while maintaining alveolar bone contour, width and height[9]. (Fig 2A and 2I).

Throwing away an avulsed tooth following intubation without the patient's or relative's consent as shown in case 1 has medico-legal implication and could result in unnecessary litigation[4]. This might be due to lack of education on the treatment modalities for avulsed anterior teeth by medical and paramedical staff who might never be aware that such a tooth can be replanted if the extra-oral dry time is minimized. This further highlights the importance of improving dental awareness among medical and paramedical staff. In addition, some degree of dental curriculum should be included in the training of medical and paramedical personnel. This will go a long way towards managing our patients holistically and preventing unnecessary and avoidable litigations.

Conclusion

A detailed examination of the patient's mouth pre-operatively by the dental surgeon can help in identifying the teeth at risk of tooth avulsion, following orotracheal intubation. This will help in the holistic management of such patients and prevent unnecessary litigation that might result from traumatic loss of anterior teeth following oro-tracheal intubation. Further studies will be necessary to identify microscopic TDI (mTDIs) which might likely occur following endotracheal intubation in the future, so as to predict tooth that might undergo progressive pulp necrosis over time.

References

1. Smith G, D'Cruz JR, Rondeau B, Goldman J. General Anaesthesia for Surgeons. 2022 May 24. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan– . PMID: 29630251.
2. Holzapfel L. Nasal vs. oral intubation. Minerva Anestesiol. (2003) 69:348– 52.
3. Mourão J, Neto J, Luís C, Moreno C, Barbosa J, Carvalho J, Tavares J. Dental injury after conventional direct laryngoscopy: a prospective observational study. Anaesthesia. 2013; 68:1059– 65.
4. Givol N, Gerhtansky Y, Halamish-Shani T, Taicher S, Perel A, Segal E. Perianaesthetic dental injuries: analysis of incident reports. J Clin Anesth. 2001;16:173– 6.
5. Dattatray A.D., Alok D, Rajashree G, Swapnil D, Sonali B.D, Ashish D.D; Assessment of the risk factors for oro-dental injuries to occur during general anaesthesia and measurement taken by anaesthetisiologist to prevent them J Int oral health. 2015 Jul; 7(7): 77-79.
6. Warner ME, Benenfeld SM, Warner MA, Schroeder DR, Maxson PM. Perianaesthetic dental injuries: frequency, outcomes, and risk factors. Anesthesiology. 1999; 90:1302– 5.
7. Newland MC, Ellis SJ, Peters KR, Simonson JA, Durham TM, Ullrich FA, Tinker JH. Dental injury associated with anaesthesia: a report of 161,687 anaesthetics given over 14 years. J Clin Anesth. 2007;19:339– 45.
8. Bourguignon C, Cohenca N, Lauridsen E, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries. 1. Fractures and luxations. Dent Traumatol. 2020;36:314-330.
9. Fouad AF, Abbott PV, Tsilingaridis G, et al. International Association of Dental Traumatology guidelines for the management of traumatic dental injuries. 2. Avulsion of permanent teeth. Dent Traumatol. 2020;36(4):331-342

Malignant Transformation of Oral Leukoplakia to Squamous Cell Carcinoma in a Patient with HIV: A Case Report

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Abstract

Oral leukoplakia is a potentially malignant lesion found more in the middle-aged and elderly, with an estimated global prevalence of 2.60%. Most oral squamous cell carcinomas develop on the background of oral leukoplakia. The risk of malignant transformation increases with the clinical type of leukoplakia, affected sites, immunosuppressive states of affected patients, alcohol and tobacco consumption, human papilloma virus infection, and chewing betel leaf and areca nut. Regular monitoring of patients with oral leukoplakia is very important for early detection of any mucosal and dysplastic change. This will aid early intervention and improve patient's survival.

Keywords*: Oral leukoplakia, potentially malignant, malignant transformation.*

Introduction

Oral leukoplakia (OL) refers to white plaques on the oral mucosa that cannot be characterized as any other specified disease, clinically or histologically[1], having excluded other known diseases or disorders that carry no increased risk for cancer[2]. Common intra-oral sites include the gums, buccal mucosa, lower lip, tongue, and floor of the mouth. The white lesions of OL in the affected sites cannot be scraped off, and it may contain speckles of reddish discoloration (erythroleukoplakia)[3,4]. OL is usually asymptomatic, but some areas of the OL may be sensitive to heat, touch, or spicy food[5]**.** The pathogenesis of OL is not well known; however, some factors have been found to play some roles in the development and progression of leukoplakia. They include *candida albicans* infection, *human papilloma* virus infection, immunosuppressive state like Human immunodeficiency virus (HIV) infection, poor oral hygiene, nutritional deficiency (vitamin A,B complex, C, Beta-carotene) repeated cheek or tongue biting, chewing betel leaf and areca nut, smoking, and alcohol consumption [3,6,7].

OL is considered the most common potentially malignant lesion with a global prevalence estimated at 2.60%[8]. Most oral squamous cell carcinomas (OSCC) occur on the background of oral leukoplakias,[9] with a malignancy conversion rate of 0.1%-17.5%[10] occurring within 15 years[11]. OSCC accounts for more than 90% of head and neck tumors.[12] Oral leukoplakia can occur years before a diagnosis of cancer.[13] Clinical evidence exists for the role of the immune system in malignant transformation in immunosuppressed patients.[9] Other factors that can also be considered risk factors for malignant transformation of oral leukoplakia include: female sex, advanced age, long duration of leukoplakia, alcohol, tobacco consumption, site of lesion (tongue and/or floor of the mouth), clinical types of the lesion (verrucous leukoplakia, leukoplakia exceeding 200mm, non-homogenous type), and presence of epithelial dysplasia—especially high grade dysplasia [8,14,15].

Routine monitoring of patients with OL is very important for early detection of any mucosal change, with strict instructions on avoidance of major risk factors for oral epithelial dysplasia like alcohol and tobacco[7]. OL rarely undergoes spontaneous regression[16], although cessation of most habits like tobacco may result in regression of leukoplakia. In the presence of persisting leukoplakia, treatment may be instituted to prevent malignant transformation.[17] Treatment of OL is usually non-specific as the predisposing factors, patient's clinical presentation and medical history, are important factors that are of utmost consideration.

This is a report of a malignant transformation of oral leukoplakia in a patient with HIV infection, who had the lesion for two years prior to the diagnosis of cancer. This report also reviews the likely risk factors for malignant transformation of oral leukoplakia, and outcome of regular monitoring.

Case report

A 56-year-old widow reported at the Oral medicine clinic, University of Benin Teaching Hospital, on account of white plaques on the lateral borders of her tongue of two years duration. There was no history of tobacco use and alcohol consumption. The patient is a known retroviral disease (RVD) patient and has been on Highly Active Antiretroviral Therapy (HAART) for about 10 years. The white plaques had been asymptomatic but persistent, with the recent appearance of a small swelling on the right lateral border the tongue which was first noticed about four months before presentation. This was of concern to her, hence her presentation at the clinic.

Upon physical examination, patient was apparently healthy looking, no evidence of pallor, not cyanosed and anicteric. The submandibular and cervical lymph nodes were not tender and palpable and there was no evidence of any associated cutaneous lesions. Intra-oral examination revealed fair oral hygiene status, with presence of homogenous white plaques on the lateral borders of the tongue

measuring 3cm by 2cm, and could not be scraped off. (fig. A). And the cusps of the posterior teeth were devoid of sharp edges. However, a small swelling with a smooth surface and which was associated with mild pain was seen on the lateral border of the tongue. Tongue swab was taken for mycology and patient placed on warm saline oral rinse 8 hourly/day for a week, with a plan for tissue biopsy of the swelling.

A week later, the result of the mycology showed presence of *candida albican spp* and the patient commenced nystatin (1:150,000 IU) oral rinse 3 times daily and fluconazole 100mg daily for 2 weeks. Exfoliative cytology of the lesion was done which revealed a reactive lesion showing hyperkeratosis-hyperplastic squamous epithelial lesion (fig. B). Patient was reassured, counselled on good nutrition and good oral hygiene, and encouraged to keep up with the HAART. Patient was also placed on chlorhexidine oral rinse 8 hourly/day for 1 week to optimize her oral hygiene. Following this, patient was placed on monthly review. Patient, however, did not keep her appointment and presented about 8 weeks later. At this point, the swelling was observed to have increased in size, presenting as a nodular lesion with marked pain (fig. C). Patient was then referred to the Oral Surgery clinic for an incisional biopsy of the lesion. An incisional biopsy of the nodular lesion was taken, and the result revealed the presence of dysplastic changes (fig. D) with a definitive diagnosis of squamous cell carcinoma made. Patient was promptly referred to the oncology team of the same hospital for treatment, and placed on monthly reviews at the oral medicine clinic.

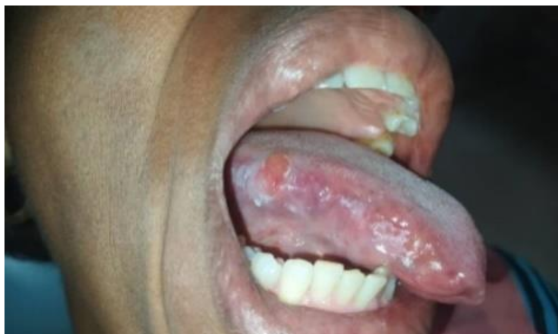

Figure A: Intraoral picture showing lesion at initial presentation

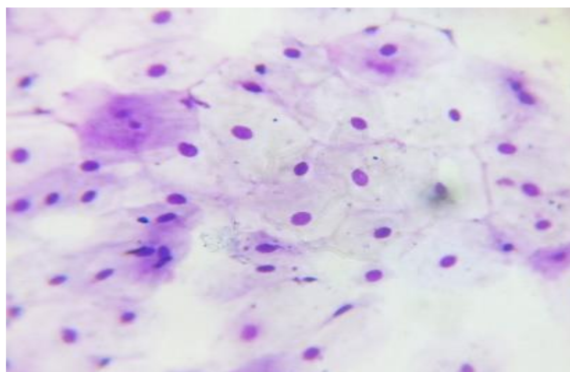

Figure B: Cytology showing clusters and disco-hesive squamous cells with neutrally placed small nuclei, with uniform nuclear chromatin, and abundant cytoplasm with dark staining granules. The background is loose with infiltrates of lymphocytes and necrotic debris (H&E stainx400)

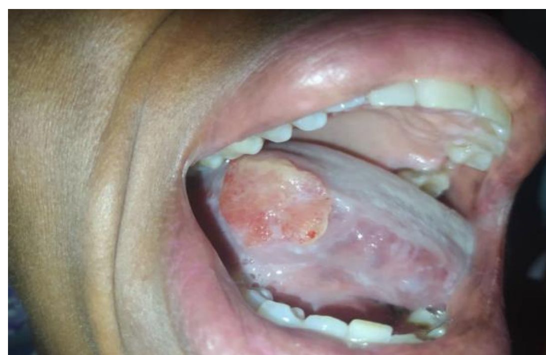

Figure C: Intraoral picture showing an increased size of the lesion on a background of leukoplakia

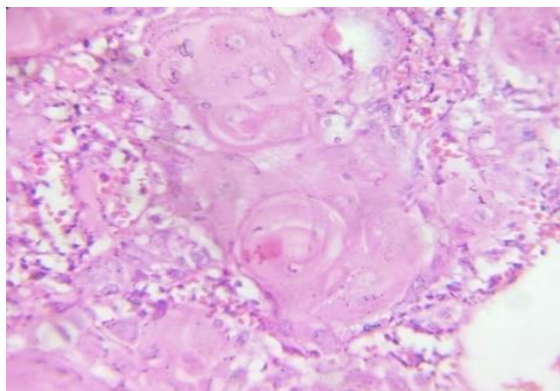

Figure D: Histological section showing numerous dysplastic squamous cells arranged in nests with keratin pearls, individual cell keratinization with presence of mixed chronic inflammatory cells infiltrates (H&E stainx400)

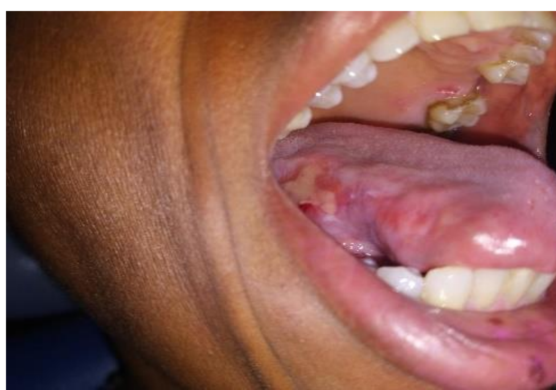

Figure E

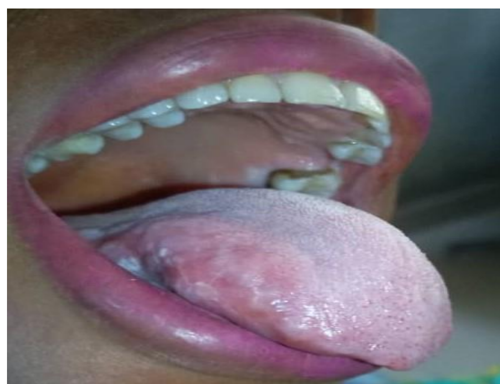

Figure F

Intraoral pictures showing marked regression of the lesion after commencement of chemotherapy. (Fig E) after 1st session of chemotherapy; (Fig F) after 4th session of chemotherapy.

Discussion

Oral leukoplakia (OL) is considered the most common potentially malignant lesion[8] which requires close monitoring to detect any clinical and histological changes. Most cases of OL may be asymptomatic for years. This was the case of our patient who had the white plaques for two years prior to presentation, without pain or discomfort. The cause of the swelling in the region of the OL on the right border of the tongue could not be ascertained. However, some strong indicators of leukoplakia transforming to cancer include appearance of ulceration, nodules, and bleeding[11]. In line with this, the appearance of a swelling in the region of the leukoplakia called for a closer monitoring of the patient.

An exfoliative cytology was initially done because the lesion was small, and the result showed a reactive lesion devoid of dysplastic cells. An increase in the size of the initial swelling to a nodular lesion weeks later was the reason an incisional biopsy was done. Studies have reported epithelial dysplasia to be a strong

indicator of malignant transformation,[8,11] hence, the dysplastic changes seen on the histology of this patient confirmed a diagnosis of OSCC. The likely factors that may have contributed to the malignant change have been considered thus. The first is the patient's immunosuppressive state.[3,9] The patient has been a known RVD patient, on HAART for about 10 years. Oral lesions are often the first signs and symptoms of HIV infection[18], and are considered high predictive markers of immunosuppression [19]. Some oral lesions associated with HIV infection include oral candidiasis, linear gingival erythema, periodontitis, oral hairy leukoplakia, oral warts, and Kaposi's sarcoma[20], and they can have a negative impact on the patients' quality of life.[20] The advent of HAART has however been associated with a decrease in the incidence of some these oral diseases[21]. While the immunosuppressive state of HIV can cause the development of opportunistic infections,[21] it has also been reported to be associated with an increased risk of oral cancers caused by human herpes viruses and human papilloma virus.[22]

The HIV-induced immunosuppression can hinder the control of cancer- associated viruses.[23] In immunocompetent people, these viruses are often carried asymptomatically. However, in immunocompromised states caused by illness, age, or HIV infection, the viruses can manifest to produce diseases. A study by Speicher on the role of HIV in the pathogenesis of oral cancer has reported possible mechanisms to include the following: (1) increasing the immune-suppression, and (2) immune activation with resultant chronic inflammation and subsequent carcinogenic effects. This can also result in an altered microbiome and loss of local immune surveillance.[22] The immunosuppressive state caused by HIV can be further complicated by the presence of co-factors for head and neck cancers (e.g. smoking and alcohol).[24] A study by Chen et al[25] also reported that HIV-infected patients were at a significant risk for oral cancer. The use of HAART, while improving the survival of HIV-infected persons, also resulted in long-term morbidities like cancers.[25,26] This reckons with our patient who has been on HAART for about 10 years.

OL that develop as a result of conditions like HIV infection may clear upon institution of antiviral therapy[3]. The OL in this patient, however, persisted despite the HAART. Patient was counseled on improved nutrition and oral hygiene, as poor oral hygiene and nutritional deficiency have been implicated in the transformation of OL to oral cancer.[3,27] Studies by Warnakulasuriya[8,15] observed factors that stand out as significant determinants contributing to the malignant potential of cancer, to include advanced age, the female gender, and leukoplakia exceeding 2cm. Barfi et al[28] reported that in female patients over 50 years of age, malignant transformations were associated with lesions located on the tongue as opposed to the males where the tongue and buccal mucosa were common sites for malignant change. These reports align with our patient who is a 56-year-old female, with lesion on the tongue.

The site of the lesion in this patient is considered a high risk for malignant change, as a study by Castagnola et al [29] reported that lesions on the ventrolateral surface of the tongue show a greater risk of aneuploidy and loss of heterozygosity which are features associated with a higher risk of malignant transformation. Barfi et al [28] also reported that most female patients with malignant transformation were non-smokers compared to male patients with malignant transformation who were mostly smokers. Our patient neither smokes nor takes alcohol, which are risk factors for malignant transformation. Non-homogenous leukoplakia (erythroleukoplakia) has been reported to be more associated with malignant transformation[8,13]. Our patient, however, had homogenous leukoplakia for two years before the appearance of the swelling in which the dysplastic changes were found. Monthly review of this patient aided the early diagnosis of OSCC. Early detection of OSCC and its preceding lesions is therefore very vital in improving patients' survival,[30] as OSCC is curable with reduced morbidity and disfigurement if detected at an early stage.[31]

Various studies have reported the treatment modalities for oral cancers to include chemotherapy, radiotherapy, and surgery. These can be employed singly or in combination.[32–34] The stage of the disease and the histologic cell type determine the choice of treatment.[35] Treatment upon late diagnosis is, however, associated with considerable morbidity as well as functional impairment and poor prognosis[9]. With the diagnosis of OSCC made in this case, the patient was immediately referred to the oncology team for treatment. Chemotherapy as the choice of treatment was promptly instituted, and the lesion was seen to have regressed remarkably (fig E) and (fig F). This was comparable to a report by Remco de Bree et al,[36] which observed frequent and significant regressions in head and neck squamous cell carcinoma after chemotherapy alone. The patient is still on routine follow-up at the oral medicine clinic.

Conclusion

This study reported a case of malignant transformation of oral leukoplakia in a known RVD patient on HARRT. Regular patient monitoring is very key in the early detection of mucosal changes, institution of appropriate therapy in the presence of dysplasia, and improved survival rate of patients.

References

1. Hong WK, Hittelman WN, Mao L, et al. Predicting cancer development in oral leukoplakia: ten years of translational research. Clinical Cancer Research. Published 2000. Accessed February 8, 2023. http://ovidsp.ovid.com?T=JS&CSC=Y&NEWS=N&PAGE=fulltext&D=med4&AN=10815888%5Cnhttp://library.newcastle.edu.au:4550/resserv?sid=OVID:medline&id=pmid:10815888&id=&issn=1078-0432&isbn=&volume=6&issue=5&spag

e=1702&pages=1702-10&date=2000&title=Clinical+Cancer

2. Warnakulasuriya S, Johnson NW, Van Der Waal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. *J Oral Pathol Med*. 2007;36(10):575-580. doi:10.1111/J.1600-0714.2007.00582.X
3. Jennifer H. Leukoplakia: Symptoms, causes, and prevention. Published 2019. Accessed February 10, 2023. https://www.medicalnewstoday.com/articles/317689
4. Vlad R, Panainte I, Stoica A, Monea M. The Prevalence of Oral Leukoplakia: Results From a Romanian Medical Center. *Eur Sci Journal, ESJ*. 2016;12(27):12.
5. Leukoplakia - Cancer Care of Western New York. Accessed May 15, 2023.
6. Shiu MN, Chen THH, Chang SH, Hahn LJ. Risk factors for leukoplakia and malignant transformation to oral carcinoma: a leukoplakia cohort in Taiwan. *Br J Cancer*. 2000;82(11):1871-1874.
7. Erugula S, Umar Farooq M, Jahagirdar D, Srija C, Meruva S, Venkata G. Oral Leukoplakia Etiology , Risk Factors , Molecular Pathogenesis , Prevention and Treatment : A Review. *Int J Contemp Med Res*. 2020;7(11):5-9.
8. Warnakulasuriya S, Ariyawardana A. Malignant transformation of oral leukoplakia: a systematic review of observational studies | Request PDF. *J oral Pathol Med*. 2019;45(155). Accessed January 31, 2023. https://www.researchgate.net/publication/337656720_Malignant_transformation_of_oral_leukoplakia_a_systematic_review_of_observational_studies
9. Weber M, Wehrhan F, Baran C, et al. Malignant transformation of oral leukoplakia is associated with macrophage polarization. *J Transl Med*. 2020;18(1):1-18. doi:10.1186/S12967-019-02191-0/TABLES/4
10. Srivastava VK. To Study the Prevalence of Premalignancies in Teenagers having Betel, Gutkha, Khaini, Tobacco Chewing, Beedi and Ganja Smoking Habit and Their Association with Social Class and Education Status. *Int J Clin Pediatr Dent*. 2014;7(2):86-92. doi:10.5005/JP-JOURNALS-10005-1243
11. Clevelandclinic. Leukoplakia; Causes, Symptoms, Management & Treatment. Published 2017. Accessed May 11, 2023.
12. Attar E, Dey S, Hablas A, et al. Head and Neck Cancer in a Developing Country: A Population-Based Perspective Across 8 Years. *Oral Oncol*. 2010;46(8):591. doi:10.1016/J.ORALONCOLOGY.2010.05.002
13. Sudbø J, Reith A. Which putatively pre-malignant oral lesions become oral cancers? Clinical relevance of early

targeting of high-risk individuals. *J Oral Pathol Med*. 2003;32(2):63-70.

14. Sun Z, Gong Y, Huang J. Analysis of relationship between risk factors of malignant transformation of oral leukoplakia and the LSCP system. Zhonghua kou qiang yi xue za zhi = Zhonghua kouqiang yixue zazhi = Chinese journal of stomatology. Published 2001. Accessed January 31, 2023.
15. Aguirre-Urizar JM, Lafuente-Ibáñez de Mendoza I, Warnakulasuriya S. Malignant transformation of oral leukoplakia: Systematic review and meta-analysis of the last 5 years. *Oral Dis*. 2021;27(8):1881-1895.
16. Waal I. Oral Leukoplakia : Present Views on Diagnosis , Management , Communication with Patients , and Research. Published online 2019:9-13.
17. Amagasa T, Yamashiro M, Ishikawa H. Oral Leukoplakia Related to Malignant Transformation. *Oral Sci Int*. 2006;3(2):45-55.
18. Ottria L, Lauritano D, Oberti L, et al. Prevalence of HIV-related oral manifestations and their association with haart and CD4+ T cell count: A review. *J Biol Regul Homeost Agents*. 2018;32(2):51-59.
19. Sharma G, Pai KM, Setty S, Ramapuram JT, Nagpal A. Oral manifestations as predictors of immune suppression in a HIV-/ AIDS-infected population in south India. *Clin Oral Investig*. 2009;13(2):141-148.
20. Lomelí-Martínez SM, González-Hernández LA, Ruiz-Anaya A de J, et al. Oral Manifestations Associated with HIV/AIDS Patients. *Med*. 2022;58(9).
21. El Howati A, Tappuni A. Systematic review of the changing pattern of the oral manifestations of HIV. *J Investig Clin Dent*. 2018;9(4):e12351.
22. Speicher DJ, Ramirez-Amador V, Dittmer DP, Webster-Cyriaque J, Goodman MT, Moscicki AB. Viral infections associated with oral cancers and diseases in the context of HIV: A workshop report. *Oral Dis*. 2016;22:181-192.
23. Bouvard V, Baan R, Straif K, et al. A review of human carcinogens--Part B: biological agents. *Lancet Oncol*. 2009;10(4):321-322.
24. Gupta B, Johnson NW. Systematic review and meta-analysis of association of smokeless tobacco and of betel quid without tobacco with incidence of oral cancer in south asia and the pacific. *PLoS One*. 2014;9(11).
25. Chen CH, Chung CY, Wang LH, Lin C, Lin HL, Lin HC. Risk of cancer among HIV-infected patients from a population-based nested case-control study: Implications for cancer prevention. *BMC Cancer*. 2015;15(1):1-9.
26. Detels R, Muñoz A, McFarlane G, et al. Effectiveness of potent antiretroviral

therapy on time to AIDS and death in men with known HIV infection duration. Multicenter AIDS Cohort Study Investigators. *JAMA*. 1998;280(17):1497-1503.

27. Zain RB. Cultural and dietary risk factors of oral cancer and precancer - A brief overview. *Oral Oncol*. 2001;37(3):205-210.
28. Barfi Qasrdashti A, Habashi MS, Arasteh P, Ardakani MT, Abdoli Z, Eghbali S. Malignant Transformation in Leukoplakia and Its Associated Factors in Southern Iran: A Hospital Based Experience. *Iran J Public Heal*. 2017;46(8):1110-1117. Accessed January 31, 2023.
29. Castagnola P, Malacarne D, Scaruffi P, Maffei M, Donadini A. Chromosomal aberrations and aneuploidy in oral potentially malignant lesions : distinctive features for tongue. *BMC Cancer*. 2011;11(1):445.
30. Bettendorf O, Piffkò J, Bànkfalvi A. Prognostic and predictive factors in oral squamous cell cancer: important tools for planning individual therapy? *Oral Oncol*. 2004;40(2):110-119.
31. Baykul T, Yilmaz HH, Aydin Ü, Aydin MA, Aksoy MÇ, Yildirim D. Early diagnosis of oral cancer. *J Int Med Res*. 2010;38(3):737-749.
32. Haddad R, Annino D, Tishler RB. Multidisciplinary approach to cancer treatment: focus on head and neck cancer. *Dent Clin North Am*. 2008;52(1):1-17.
33. Aa O, Ao A, To A, et al. Orofacial Cancers : Pattern and Management in. 2018;45(4):179-188.
34. Awodutire PO, Ilori OR, Uwandu C, Akadiri OA. Pilot study of new statistical models for prognostic factors in short term survival of oral cancer. *Afr Health Sci*. 2022;22(2):310-317.
35. da Lilly-Tariah OB, Somefun AO, Adeyemo WL. Current evidence on the burden of head and neck cancers in Nigeria. *Head Neck Oncol*. 2009;1:14. doi:10.1186/1758-3284-1-14
36. de Bree R, Wolf GT, de Keizer B, et al. Response assessment after induction chemotherapy for head and neck squamous cell carcinoma: From physical examination to modern imaging techniques and beyond. *Head Neck*. 2017;39(11):2329-2349.

Ameloblastoma with Infratemporal Extension: A Review of the Literature

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Abstract

Ameloblastomas are benign tumors of odontogenic epithelium. They are locally aggressive with the tendency to recur, and sometimes with metastatic behavior. Recurrences often happen due to incomplete treatment and they can occur at difficult sites such as temporal and infratemporal fossa. Recurrences in the temporal area are very rare and are related to the type of primary treatment.

***Aim**: This literature review aims to answer the question on how common recurrent ameloblastoma extends to the infratemporal fossa and how this is related to the site of the primary lesion.*

***Materials and methods**: Web search for case reports, and case series of ameloblatoma with temporal, infratemporal extension, published in the English literature were carried out. Search results were further scrutinised for age, sex, location of lesion, histology, treatment modalities, and recurrence, following the adopted treatment modalities and treatment outcome.*

***Result:** A total 15 full length articles were included in this study. Twelve were case reports and three were case series. Of 28 patients with ameloblastoma in the articles, only 22 were recorded to have presented with ameloblastoma with infratemporal or temporal fossa involvement. All the cases of ameloblastoma involving the infratemporal/temporal fossa were recurrent tumors and the average time from first surgical intervention to recurrence was 11.36 years. Most of the primary cases were seen in the mandible (73%) with the body/ramus region being the commonest location. Only five cases were reported to be primarily maxillary ameloblastoma.*

***Conclusion**; This review has shown that temporal/infratemporal extension of ameloblastoma occurs commonly with recurrent lesions, although the overall reported incidence is relatively low. Aggressive primary tumor resection, especially for extensive mandibular lesions, may be key to preventing this tumor extension.*

***Keywords**: Ameloblastoma, temporal, infratemporal extension*

Introduction

Ameloblastomas are odontogenic lesions characterised by local invasiveness and the potential for direct involvement of vital structures with high tendency for recurrence, leading to extensive local morbidity and mortality. Ameloblastoma is the second most common odontogenic tumor of the jaws. It commonly occurs in the mandible and in the third to fifth decades of life.[1–9] Eighty percentage of ameloblastomas arise in the mandible, it infrequently involves the maxilla[2,7]. Only about 5 to 20% occur in the maxillary bone, with majority of these occuring in the molar region.[10,11] Some authors reported no gender predilection,[4,12,13] some others reported male predilection,[1,2] while others documented female predilection.[3]

Due to its tendency to cause extensive destruction of jaw bones, various treatment modalities of "conservative" and "radical" surgery have been described.[14,15] Conservative surgical approaches may be favored due to benign histology, however, these treatment modalities have very high recurrence rates (90% for mandibular tumors, 100% for maxillary tumors).[9,15] Recurrences often occur due to incomplete treatment and they can occur at difficult sites such as temporal and infratemporal fossa, orbit, anterior cranial base, paranasal sinuses, etc.[7,16-19] Recurrences in the temporal area are very rare and are related to the type of primary treatment. Most of the studies done on the temporal and/or intra cranial extension of ameloblastoma are mostly case reports and case series with little or no reviews of cases so far published. This literature review therefore aims to answer the question on how common recurrent ameloblastoma extends to the infratemporal fossa, and how this is related to the site of the primary lesion.

Materials and methods

We conducted systematic searches for published articles in PubMed (NLM), Cochrane, Ovid Medline, and OpenGrey databases up till December 2021 using the keywords: "ameloblastoma," "temporal," and "infratemporal extension." Additional searches for relevant studies were done via the following methods: hand-search of the reference section of eligible studies and purposeful Google Scholar searches. Only articles written in English or with English language translations were considered for the review. Both authors independently screened the titles and abstracts (when available) of all reports identified through electronic searches. The search was designed to be sensitive to include all available studies. For studies appearing to meet the inclusion criteria, or for which there was insufficient data in the title and abstract to make a clear decision, we obtained the full report. The full reports were also independently assessed by the two authors to establish whether the publication met the inclusion criteria or not. Disagreements were resolved through discussion between the two authors.

This search returned 37 articles in PubMed and 207 articles in PubMed Central. The initial

screening process resulted in 29 articles and these articles were retrieved and reviewed for relevance of content by the two authors (OAO and AAA). A total of 16 full articles were included in the final list for review (Table 1).

Data retrieved from search results included number of patients, age, gender, location of lesion and histology, treatment modalities carried out, any recurrence following the adopted treatment modalities, and treatment outcomes. Furthermore, other odontogenic tumours such as KCOT, ameloblastic fibroma, adenomatoid odontogenic tumour, etc. were excluded. Articles with cases more than two were adopted as case series.

Result

Out of the 15 papers found in the literature, 12 were case reports and 3 were case series. The total number of patients with ameloblastoma in the review was 28. However, only 22 were recorded to have presented with ameloblastoma with infratemporal or temporal fossa involvement and reviewed for this study. Sixty four percent (n = 14/22) were females, and their ages ranged between 18-73 years (mean = 43.10, SD ±17.39). All the cases of ameloblastoma involving the infratemporal/temporal fossa were recurrent tumors and the average time from first surgical intervention to recurrent lesion/involving the infratemporal/temporal fossa was 11.36 years. Seventy-three percent of the cases with infratemporal/temporal extension were found in the mandible (n = 16/22), with body/ramus region being the commonest location. Only 5 cases were reported to be primarily maxillary ameloblastoma.

Table 1. Case Series and Case Reports of Ameloblastoma and Temporal/Infratemporal Extension

Author	Title	Type of report	No of patients	Primary location	Secondary location	No of patients with infratemporal/temporal extension	Initial treatment	Time to temporal involvement	Treatment outcome	Age	Sex	Remarks
Zwahlen et al., 2002[11]	Maxillary ameloblastomas: a review of the literature and of a 15-year database	CS	5	Maxilla	maxilla	1	resection	NA	6yrs follow up	26	F	Ameloblastoma?
				Maxilla	ethmoid Sphenoid			NA		33	F	
				Maxilla	temporal	1		0.17		73	F	
				Maxilla				NA		42	M	
				Maxilla				NA		44	M	
Weiss et al., 1985[20]	Maxillary Ameloblastoma with Orbital Invasion A	CR	1	Maxilla	infratempo	1	resection	5	6 yrs and died same yr	72	M	follicular

	Clinicopathologic Study				ral and sphenoidal							
To et al., 2002[18]	Recurrent Ameloblastoma a Presenting in the Temporal Fossa	CR	1	Mandible	temporal	1	curettage, resection	25	2.5 yrs follow--up	18	F	Ameloblastoma?
Al-Bayaty et al., 2002[17]	Soft Tissue Recurrence of a Mandibular Ameloblastoma a Causing Facial Deformity in the Temporal Region: Case Report	CR	1	Mandible	temporal	1	resection	4	tumour free 2yrs follow-up	32	F	follicular
Faras et al., 2016[21]	Multi-recurrent invasive ameloblastoma: A surgical challenge	CR	1	Mandible	infratemporal	1	repeated resection	23	NA	56	F	follicular

Sharma et al., 2009[22]	Recurrent Unicystic Ameloblastoma of the Infratemporal and Temporal Fossa	CR	1	Mandible	temporal	1	enucleation and later resection	2.6	NA	20	F	follicular
Auluck et al., 2007[16]	Recurrent ameloblastoma of the infratemporal fossa: diagnostic implications and a review of the literature	CR	1	Mandible	infratemporal	1	resection	6	NA	44	F	follicular
Ferretti et al., 2000[23]	Recurrent Ameloblastoma a Report of 2 Cases	CR	1	Mandible	temporal	1	resection	1.5	2 yrs tumour free	50	M	ameloblastoma
			1	Mandible	temporal	1	resection	25	3 yrs tumour free	42	M	ameloblastoma

Scaccia et al., 1991[24]	Maxillary Ameloblastoma a Case Report	CS	1	Maxilla	ethmoidal , sphenoidal, infra temporal and intracranial	1	resection	NA	2 yrs follow-up	16	F	Ameloblastoma
			1	Maxilla	ethmoidal, sphenoidal,		resection	NA	2 yrs. follow-up	66	M	
			1	Maxilla	infra temporal and intracranial			17	Recurrence after 2 yrs	53	M	
			1	Maxilla	infra temporal and intracranial , ethmoidal, sphenoidal,	1	resection	2	2 yrs tumour free	36	F	Ameloblastoma
Luc et al., 1988[25]	Late Ioco-regional recurrences after radical resection for mandibular	CS	5	mandible	Maxilla	1	Resections	NA	Recurrence	51	F	Ameloblastoma

	ameloblastoma											
				mandible	Infratemporal	1	Resection	29	18 months	49	F	Ameloblastoma
				mandible	maxilla	1	Resections	NA	Recurrence after 2 yrs	46	M	Ameloblastoma
				Mandibular angle	infratemporal	1	hemimandibulectomy including the condyle	8	Recurrences and resections	67	M	Ameloblastoma
				Mandibular angle	temporal	1	hemimandibulectomy including the condyle and coronoid process	29	Recurrences and resections ,1 yr tumour free	50	F	Ameloblastoma
Aramanadka et al., 2018[26]	Recurrent Ameloblastoma: A Surgical Challenge	CR	1	Mandible		1	Resection	NA	2 yrs follow-up	56	M	Follicular Amelolastoma

			1	Mandible	Infratemporal		Hemimandibulectomy	6	NA	45	M	Follocular Ameloblastoma
Vaishampayan et al., 2014[4]	Recurrent ameloblastoma in temporal fossa: A diagnostic dilemma	CR	1	Mandible	temporal	1	resection, hemimandibulectomy	5	tumour free in 1.5 yrs	32	F	Ameloblastoma
Phillips et al., 1992[27]	Ameloblastoma of the Mandible With Intracranial Metastasis A Case Study	CR	1	mandible	temporal and intracranial	1	resections	13	NA	65	M	Ameloblastoma
Oka et al., 1986[7]	Mandibular ameloblastoma with intracranial extension and distant metastasis	CR	1	mandible	temporal and intracranial and femur	1	resections	19	femur mets chemo recurrence and died 43 yrs later	25	M	ameloblastoma

Rauso et al., 2010[12]	Recurrence of Ameloblastoma in Temporal Area: Primary Treatment Influences Recurrence Rate	CR	1	mandible	temporal fossa	1	enucleation and currettage	3	5 yrs follow up tumor free	29	F	Acanthomatous ameloblastoma

CR- case report; CS – case series

Discussion

In this review, 22 out of 28 patients were recorded to have presented with ameloblastoma with infratemporal or temporal fossa involvement. The initial site of involvement for majority was the mandible (73 percent) with the mandibular body and ramus being the most affected. The increased prevalence of infratemporal and temporal involvement of mandibular lesions compared to maxillary lesions could be attributed to higher prevalence of ameloblastoma in the mandible than in maxilla as reported in the literature.

Furthermore, 12 of the articles selected for this study were case studies while 3 articles accounted for case series with no cohort study. This could be due to rarity of this ameloblastoma with temporal and intra cranial extension. All reported cases involving the infratemporal/ temporal fossa were recurrent tumors and the average time from first surgical intervention to recurrent lesion involving the infratemporal/ temporal fossa was 11.36 years. Recurrence may be attributed to factors such as inadequate tumor removal, "seeding", aggressive histology, and spread along the muscle attachment.[17,23] Treatment of recurrence often mandates extensive ablative and reconstructive surgery with inherent morbidity, even in expert hands.[4,16] Recurrences of ameloblastoma often occur at difficult sites, and has been documented to recur in sites such as temporal and infratemporal fossa, orbit, anterior cranial base, paranasal sinuses etc.[16-19,11, 28, 29]

Due to the tendency of ameloblastoma to cause extensive destruction of jaw bones, various treatment modalities of conservative" and "radical" surgery have been described.[28] Other treatments described in literature include electrocautery, cryosurgery, chemotherapy, and radiotherapy.[3,7,13] Conservative surgical approach has been reported to have very high recurrence rates (90% for mandibular tumors, 100% for maxillary tumors).[4] The gold standard of care for ameloblastoma is complete surgical excision; aggressive surgical resection is advocated in patients with maxillary ameloblastoma to ensure recurrence-free outcome.[13] Although some authors have reported successful results with radiotherapy,[30,31] its use is however considered more in inoperable cases, primarily in the posterior maxilla.[31] Furthermore, chemotherapy as treatment modality has also been employed for inoperable lesions.[3] It is important to know that spread of the lesion from the infratemporal fossa and temporal region to adjacent structures to involve the pterygopalatine fossa or maxillary sinus, the skull base, and into the intracranial cavity or orbit makes radical surgical treatment more difficult.[16] Nastri et al[3] reported preoperative radiographic evidence of tumour in all of the cases in which surgical treatment failed to control the tumour, suggesting residual lesion. Therefore, early and aggressive surgical treatment is key in the management of ameloblastoma.

Treatment of maxillary ameloblastoma is inherently more difficult compared to its mandibular counterpart.[13] This is reported to be

due to the insidious nature of the lesion within the thin bones and hollow spaces of the midfacial bones, as the tumor easily spreads to the skull base, and, occasionally, may extend into orbit and/or the intracranial cavity by destroying the bones.[4] Numerous surgical approaches have been employed to access the infratemporal region, some of them being the coronal,[23] trans-oral, trans nasal, trans palatine, trans zygomatic, trans cervical, and extended maxillectomy approach.[16,26] Others include subtemporal epidural approach, and combined transcranial and transcervical approach.[32] The surgical approach to the lesion is often determined by clinical presentation, extent and location, as well as histopathological findings.[16] In addition, involvement of adjacent tissues requires collaborative surgical care[13] that would be provided by the oral and maxillofacial surgeons, otolaryngologists, plastic and reconstructive surgeons, ophthalmologists, and neurosurgeons.

This review has shown that temporal/infra-temporal extension of ameloblastoma occurs commonly with recurrent lesions, although the overall reported incidence is relatively low. Aggressive primary tumor resection, especially for extensive mandibular lesions may be key to preventing this tumor extension.

References

1. Olaitan AA, Adeola DS, Adekeye EO. Ameloblastoma: clinical features and management of 315 cases from Kaduna, Nigeria. *J Cranio-Maxillofacial Surg*. 1993;21(8):351-355. doi:10.1016/S1010-5182(05)80497-4
2. Ladeinde AL, Ogunlewe MO, Bamgbose BO, Adeyemo WL, Akinwande JA. Ameloblastoma : Analysis of 207 cases in a Nigerian teaching hospital. *Quintessence Int*. 2006;37(1):69-74.
3. Nastri AL, Wiesenfeld D, Radden BG, Eveson J, Scully C. Maxillary ameloblastoma: a retrospective study of 13 cases. *Br J Oral Maxillofac Surg*. 1995;33(1):28-32. doi:10.1016/0266-4356(95)90082-9
4. Vaishampayan SS, Nair D, Patil A, Chaturvedi P. Recurrent ameloblastoma in temporal fossa : A diagnostic dilemma. *Contemp Clin Dent*. 2013;4(2):220-222. doi:10.4103/0976-237X.114852
5. Kyriazis AP, Karkazis GC, Kyriazis AA. Maxillary ameloblastoma with intracerebral extension. Report of a case. *Oral Surgery, Oral Med Oral Pathol*. 1971;32(4):582-587. doi:10.1016/0030-4220(71)90323-9
6. Komisar A. Plexiform Ameloblastoma of the Maxilla with Extension to the Skull Base. *Head Neck Surg*. 1984;7:172-175.
7. Oka K, Fukui M, Yamashita M, et al. Mandibular ameloblastoma with intracranial extension and distant metastasis. *Clin Neurol Neurosurg*. 1986;88(4):303-309.
8. Daramola J, Abioye A, Ajagbe H, Aghadiuno P. Maxillary malignant ameloblastoma with intraorbital extension: report of case. *J Oral Surg*

(Chic). 1980;38:203–6.

9. Reichart PA, Philipsen HP, Sonner S. Ameloblastoma: Biological profile of 3677 cases. *Eur J Cancer Part B Oral Oncol*. 1995;31(2):86-99. doi:10.1016/0964-1955(94)00037-5
10. Luo Q, Diao W, Luo L, Zhang Y. Comparisons of the Computed Tomographic Scan and Panoramic Radiography Before Mandibular Third Molar Extraction Surgery. *Med Sci Monit*. 2018;24:3340-3347. doi:10.12659/MSM.907913
11. Zwahlen RA, Gratz KW. Maxillary ameloblastomas : a review of the literature and of a 15-year database. *J Cranio-Maxillofacial Surg*. 2002;20:273-279. doi:10.1054/jcms.2002.0317
12. Rauso R, Tartaro G, Gherardini G, Puglia F, Santagata M, Colella G. Recurrence of ameloblastoma in temporal area: primary treatment influences recurrence rate. *Journal of Craniofacial Surgery*. 2010;21(3):887-891. doi:10.1097/SCS.0b013e3181d80a1a = ruso
13. Maia EC, Sandrini FAL. Management techniques of ameloblastoma: a literature review. *RGO - Rev Gaúcha Odontol*. 2017;65(1):62-69. doi:10.1590/1981-863720170001000093070
14. Adeyemo WL, Bamgbose BO, Ladeinde AL, Ogunlewe MO. Surgical management of ameloblastomas: Conservative or radical approach? A critical reviewof the literature. *Oral Surg*. 2008;1(1):22-27. doi:10.1111/j.1752-248X.2007.00007.x
15. Carlson ER, Marx RE. The ameloblastoma: Primary, curative surgical management. *J Oral Maxillofac Surg*. 2006;64(3):484-494. doi:10.1016/j.joms.2005.11.032
16. Auluck A, Shetty S, Desai R, Mupparapu M. Recurrent ameloblastoma of the infratemporal fossa : diagnostic implications and a review of the literature. *Dentomaxillofacial Radiol*. 2007;36:416-419. doi:10.1259/dmfr/45988074
17. Al-Bayaty HF, Murti PR, Thomson ERE, Niamat J. Soft tissue recurrence of a mandibular ameloblastoma causing facial deformity in the temporal region: Case report. *J Oral Maxillofac Surg*. 2002;60(2):204-207. doi:10.1053/joms.2002.29826
18. To EWH, Tsang WM, Pang PCW. Recurrent ameloblastoma presenting in the temporal fossa. *Am J Otolaryngol*. 2002;23(2):105-107. doi:10.1053/ajot.2002.30629
19. Todd R, Gallagher GT, Kaban LB. Mass in the infratemporal fossa. In: *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics*. Vol 84. Mosby Inc.; 1997:116-118. doi:10.1016/S1079-2104(97)90054-8
20. Weiss JS, Bressler SB, Jacobs EF, Shapiro J, Weber A, Albert DM. Maxillary Ameloblastoma with Orbital

Invasion: A Clinicopathologic Study. *Ophthalmology*. 1985;92(5):710-713.
21. Faras F, Abo-Alhassan F, Israël Y, Hersant B, Meningaud JP. Multi-recurrent invasive ameloblastoma: A surgical challenge. *International Journal of Surgery Case Reports*. 2017;30:43-45.
22. Sharma S, Kumar D, Vashistha A, Bihani U, Trehan M. Recurrent unicystic ameloblastoma of the infratemporal and temporal fossa. *International Journal of Clinical Pediatric Dentistry.* 2009;2(1):33.
23. Ferretti C, Polakow R, Coleman H, Dent M. Recurrent ameloblastoma: Report of 2 cases. *J Oral Maxillofac Surg*. 2000;58(7):800-804. doi:10.1053/joms.2000.7271
24. Scaccia FJ, Strauss M, Arnold J, Maniglia AJ. Maxillary ameloblastoma: case report. *American Journal of Otolaryngology.* 1991;12(1):20-25.
25. Luc JM, Mommaerts MY, Fossion E, Bossuyt M. Late loco-regional recurrences after radical resection for mandibular ameloblastoma. *International journal of oral and maxillofacial surgery.* 1988;17(5):310-315.
26. Aramanadka C, Kamath AT, Kudva A. Recurrent ameloblastoma: a surgical challenge. *Case Reports in Dentistry*. 2018;2018:1-7
27. Phillips SD, Corio RL, Brem H, Mattox D. Ameloblastoma of the mandible with intracranial metastasis: A case study. *Archives of Otolaryngology–Head & Neck Surgery*. 1992;118(8):861-863.
28. Pogrel MA, Montes D. Is there a role for enucleation in the management of ameloblastoma? *Int J Oral Maxillofac Surg*. 2009;38(8):807-812.
29. Miloro M. Microneurosurgery. In: Miloro M, Ghali GE, Larsen PE, Waite P, eds. *Peterson's Principles of Oral and Maxillofacial Surgery* . 2nd ed. BC Decker; 2004:819-827.
30. Mbarki I, Randriamarosona N, Touim SH, et al. Radiotherapy for large recurrent ameloblastoma of the mandible previously treated by surgery: A case report. *Int J Clin Case Reports Rev*. 2021;6(2):01-05. doi:10.31579/2690-4861/089
31. Gardner DG. Radiotherapy in the treatment of ameloblastoma. *Int J Oral Maxillofac Surg*. 1988;17(3):201-205.
32. Yoshida K, Kawase T, Tomita T, et al. Surgical Strategy for Tumors Located in or Extending From the Intracranial Space to the Infratemporal Fossa — Advantages of the Transcranial Approach (Zygomatic Infratemporal Fossa Approach) and the Indications for a Combined Transcranial and Transcervi. *Neurol Med Chir*. 2009;49:580-586

Presentation and Management of Dental Fluorosis in a Resource-Limited Facility in North-Central, Nigeria

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Abstract

Background: *Dental fluorosis is a developmental disturbance characterized by excess fluoride in hard tissues of the teeth. The appearance of teeth affected by dental fluorosis may negatively affect individuals' self-esteem and overall quality of life. Hence, the need for treatment, although there is still debate on the best treatment modalities. The objective of this study was to document the presentation and management of dental fluorosis in a resource limited facility in North-Central Nigeria.*

Materials and methods: *A cross-sectional hospital-based study conducted among out-patients attending a secondary oral health care facility in Jos, North-Central Nigeria between June 2020 and June 2022. Sociodemographic characteristics were recorded. All patients were examined for presence of dental fluorosis. Dean's Index was used to classify dental fluorosis. Patients with dental fluorosis who consented to the study were treated. Treatment modalities included oral prophylactic treatment, micro-abrasion, and resin infiltration. Data analysis was by the use of Statistical Package for Social Sciences (SPSS) version 23.0. A p-value of < 0.5 was taken as statistically significant.*

Results: *During the study period, 1201 patients presented with different oral health challenges. Among these, 200 (16.7%) were diagnosed of dental fluorosis. The mean of the patients with dental fluorosis was 14.15 ± 1.91 years. Nearly half, 98 (49.0%), of the patients who presented with dental fluorosis were children, 65 (32.5%) were teenagers, and 37(18.5%) were young adults (p=0.037). Among the patients, 123 (61.5%) were females. The moderate type of dental fluorosis 75(37.5%) was the commonest type of dental fluorosis seen. Out of the 200 patients with dental fluorosis, dental caries was*

present among 55(37.5%) patients. 185 (92.5%) requested for treatment. 231 intervention procedures were undertaken on different classes of dental fluorosis.

Conclusion: *Dental fluorosis was prevalent among the dental out-patients seen at the study center during the study period. More patients presented with moderate class of dental fluorosis. Presentation was more among children and teenagers below 20 years. More females presented and requested for intervention than males. Resin infiltration was found to be cost effective at this facility.*

Keywords: *Dental fluorosis, management, presentation, Nigeria*

Introduction

Dental fluorosis is a developmental disturbance characterized by excess fluoride in hard tissues of the teeth.[1] It is a common presentation in many oral health care facilities in Nigeria, among all age groups and sexs.[1, 2] Tooth affected by dental fluorosis may present with mottling of enamel, unpleasant appearance, esthetic embarrassment, and may negatively affect individual self-esteem and overall quality of life.[1,2,3] Other clinical features of dental fluorosis apart from enamel mottling include: white striation, brown discoloration, pitting of enamel, and in severe cases, corroded appearance of teeth.[1,2,4] While fluoride is needed for healthy teeth development, remineralisation, and dental caries prevention, excess fluoride can become toxic to the body and lead to dental and skeletal fluorosis.[3,5] Bone and teeth are therefore major markers for fluoride in the body apart from saliva, milk, nails and urine.[5,6] The pathogenesis of dental fluorosis is due to disturbances in the activities of a specialized cell called ameloblast during enamel formation and mineralization of organic matrix in tooth development.[3,7] To prevent dental fluorosis, 0.05 to 0.07mg F/Kg body weight/day intake is recommended.[4]

While prevalence of dental fluorosis varies from country to country, there is still debate on the best treatment modalities. Dental fluorosis has been mostly reported among people in less developed than developed countries,[3] due to relative scarcity of treated water consumed by the public in the former. Surface water contains less fluoride concentration (<1.5mg/L) than underground water.[5,8] Underground water from boreholes and wells, without chemical analysis and treatments, is a major source of drinking water in many communities in Nigeria. Fluoride concentration in drinking water within 0.5-1mg/l benefits the body, but a concentration higher than 1.5 mg/L constitute a risk of dental and skeletal fluorosis.[5,9] Although the leading cause of fluorosis is a higher concentration of fluoride in drinking water, [9-12] fluorosis has also been reported in areas where fluoride concentration in drinking water is lower or even within the safety level.[5] Fluorosis in this situation was

ascribed to high environmental temperature that induced a higher water consumption rate or fluoride from other sources. Other documented sources of fluoride include vegetables, milk, beverages, salt, food, tea, tobacco, and tooth paste.[1,4,5,11,12,] According to United States Environmental Protection Agency, a concentration of 0.7-1.2mg/l fluoride in drinking water is adequate for protection against dental caries and cannot lead to fluorosis.[5] It has equally been recommended that 0.9-1 part per million of fluoride in public water is adequate.[3,9]

Dental fluorosis affects all ages; however, higher prevalence has been reported among 15-year-olds and below.[3,5] Nevertheless, the relationship between dental fluorosis and sex, environmental condition and dental caries is inconclusive.[3,5] Among teenagers, a prevalence of 72% was reported in india,[5] 91.9% in Mexico,[5,13] 91% in Ethopia,[5,14] 11.3% [15] and 11.4% [16] in Nigeria. A Nigerian study also reported 47% as proportion of patients attending a tertiary hospital in northeastern part of the country who presented with dental fluorosis.[2] Fluoride levels are low in most parts of Nigeria, being 0.3 ppm or less in 62% of the LGAs. However, fluoride concentrations were generally higher in the north-central geopolitical zone from all drinking water sources, than the other zones in the country.[17] Nonetheless, the magnitude of dental fluorosis and its management among patients is yet to be fully documented in North-Central Nigeria, hence this study. The objective of this study was to document the presentation and management of dental fluorosis in a resource limited facility in North-Central Nigeria.

Materials and Methods

This cross-sectional hospital-based study was conducted among out-patients attending a secondary oral health care facility (Our Lady of Apostle Hospital) in Jos, North-Central Nigeria. Our Lady of Apostle Hospital was established in 1943 by the Catholic Mission. It was to serve Plateau and neighboring states. The fluoride level (ppm) of the different water sources in this zone are 0.70±0.62 for water works, 0.41±0.31 for rivers and streams, 0.96±0.81 for shallow wells, 0.67±1.28 for deep wells, 0.68±0.75 for boreholes, and 0.44±0.24 for ponds.[17] The hospital attends to all religious faiths. The Dental clinic component was established in 2016. This clinic is run by a maxillo-facial surgeon and a family dentist. There is one dental health technician, no therapist, no technologist and no laboratory facility.

This study took place between June 2020 and June 2022. In 2021, there were about 720 patients seen in the clinic. Most of them (80.4%) were on social health insurance scheme (National Health Insurance Scheme). The others were out of pocket treatment. Ethical approval was obtained from the Institutional Review Board of Our Lady of Apostle Hospital, the study center. Inclusion criteria for the study were presence of dental fluorosis in oral cavity of patients and their willingness to participate in the study. The detail of the study, especially the treatment modality, was thoroughly explained to

them after which those who were interested in participating signed a written, informed consent form. For pediatric patients, their parents or legal guardians signed the consent form.

Information on sociodemographic characteristics such as age and sex as well as the date of first visit, principal complaint, history of presenting complaint, and initial treatment were recorded. All patients were dentally examined for presence of dental fluorosis by a trained and calibrated examiner. Investigations which included pulp vitality, radiographic and mobility tests, were undertaken. Dean's Index was used to classify dental fluorosis,[18] while DMFT index was used to report the presence of dental caries. All patients who had dental fluorosis and signed the written, informed consent form were treated by only one dentist (E.A) while those who had dental caries were referred to the restorative dentist for treatment.

Materials used for treatment of dental fluorosis included: pumice, polishing rubber cups, Microhybrid Composite Resin and its etchant (Henry Schein, Langen, Germany-2020), gauze, Glass Ionomer cement- (Prevestdent PRO Ltd, Jammu India–2020), Prophylaxis Paste (Bestdent, Milan, Italy-2020), composite polishing burs, oral examination set, caries detection probes, and ultrasonic scaler (Woodpecker, Guangxi, China- 2019).

Routine scaling using ultrasonic scaler (Woodpecker made by Guilin woodpecker, Guangxi, China 2019) was carried out as first line of treatment, and prophylaxis paste without fluoride content was used for polishing the teeth. Patients that were still not satisfied with the appearance of their teeth after scaling and polishing were recalled 72 hours after for micro abrasion and or resin infiltration. Due to high cost of resin infiltration, attention was paid to teeth that were within the esthetic zone i.e., the anterior teeth and premolars. Consequently, the labial and buccal surfaces of lower/upper anterior teeth and first premolars with moderate and severe dental fluorosis were selected for resin infiltration. Applying the resin infiltration, Manufacturer's instructions (Henry Schein INC. USA) were followed and the labial or buccal surfaces of the selected teeth were acid etched with Natural Elegance Etching gel (Henry Schein INC. USA, 2020) containing phosphoric acid for 2 minutes after isolation with rubber dam. The etched surfaces were rinsed with sterile water for 30 seconds and then dried with air jet. Bonding agent (Henry Schein INC. USA 2020) containing Natural Elegance Universal Bond was applied on etched surfaces and was light cured. Appropriate resin shade (A1 or B3) chosen by each patient was applied on etched surfaces and light cured in layers until there was improvement in the appearance. Excess materials were removed from the interdental surfaces and polishing of the restoration was later done after 24-48 hours. After treatments, patients were followed-up for 3, 6, 12 and 18 months after initial treatments. A digital camera was used by the dentist to document the presentation of dental fluorosis on the teeth before and after treatment.

Data obtained were analyzed using Statistical Package for Social Sciences (IBM SPSS) version 23.0 (SPSS Inc., Chicago, 11, USA). The patients' ages were grouped into 0-9 years (children), 10-20 years (preteen ages and teenagers) and above 20years (adults). Frequencies, percentages, means, and standard deviations were generated. Association between categorical variables were undertaken using chi-square test at *p*-value of < 0.5 test of statistically significant.

Results

During the study period, 1201 patients presented with oral health challenges. Among these patients, 255 complained of teeth discoloration, of which 200 (16.7%) were diagnosed of dental fluorosis using Dean's Index. Statistical power analyses using G*Power 3.1.9.7[19] at an α of o.o5, sample size of 200, medium effect size of 0.3 and a degree of freedom of 3 for χ^2 tests gave the Power of the study (1 – β) as 0.959. The mean ± SD age of the patients with dental fluorosis was 14.15 ± 1.91 years. Table 1 shows that the majority, 98 (49.0%), of the patients who presented with dental fluorosis were children, 65(32.5%) were preteen ages and teenagers and 37(18.5%) were young adults (p=0.037).

Among the patients, 123 (61.5%) were females while 77 (38.5%) were males (p=0.003). The majority, 13 (65.0%), of patients who presented with questionable dental fluorosis were children while the majority, 20 (50.0%), that presented with severe dental fluorosis were young adults. Except in moderate dental fluorosis, females presented more with questionable 12 (60.0%), mild 40 (61.5%, and severe 34(85.0%) dental fluorosis than males with corresponding values of 4 (40.0%), 25 (38.5%) and 6 (15.0%) respectively.

Table 1: Dental fluorosis presentation among patients by age and sex

Biodata	**Classes of dental fluorosis**					**χ^2**	***p*-value**
	Question able Freq. (%)	Mild Freq. (%)	Moderate Freq. (%)	Severe Freq. (%)	Total Freq. (%)		
Age(years)							
Children (0-9)	13(65.0)	35(53.8)	40(53.3)	10(25.0)	98(49.0)	13.433	0.037
Preteen ages and	2(10.0)	10(15.4)	15(20.0)	10(25.0)	37(18.5)		

teenagers (10-19)							
Adults (≥20)	5(25.0)	20(30.8)	20(26.7)	20(50.0)	65(32.5)		
Sex							
Male	8(40.0)	25(38.5)	38(50.7)	6(15.0)	77(38.5)	14.037	0.003
Female	12(60.0)	40(61.5)	37(49.3)	34(85.0)	123(61.5)		

Mean ± SD age = 14.15±1.91 years Freq. = Frequency

Figure 1 shows that the differences in the presentation of various types of dental fluorosis was statistically significant (p<0.05), with most, 75 (37.5%), patients presenting with moderate types while the least, 20 (10.0%), presented with severe types. There was no patient with very mild type of dental fluorosis.

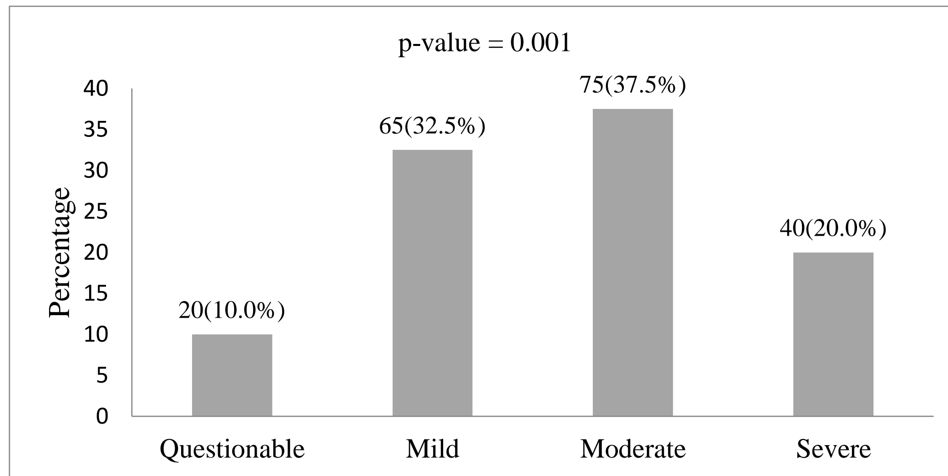


Figure 1: Distribution Dean's Index of Dental fluorosis among the patients.

Table 2 shows there was no statistically significant relationship between occurrence of dental caries and classes of dental fluorosis among patients (*p*=0.245). Among the 200 patients with different degree of dental fluorosis, dental caries was present among 55 (37.5%) patients.

Table 2: Relationship between dental caries and different classes of dental fluorosis among patients

	Dental Caries Present Frequency (%)	Dental caries Absent Frequency (%)	Total Frequency (%)	χ^2	p-value
Questionable	8(14.5)	12(8.3)	20(10.0)	4.157	0.245
Mild	20(36.4)	45(31.0)	65(32.5)		
Moderate	15(27.3)	60(41.4)	75(37.5)		
Severe	12(21.8)	28(19.3)	40(20.0)		

Out of the 200 patients with dental fluorosis, 185 requested for treatment and 231 intervention procedures were undertaken on different classes of dental fluorosis. There was a statistically significant relationship between the intervention procedures and classes of dental fluorosis (p=0.001). The different interventions administered to the patients are shown in Table 3.

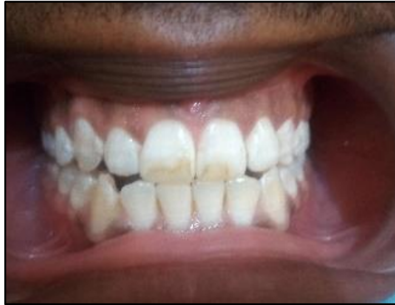
Table 3: Intervention modalities given to patients with classes of dental fluorosis

	S&P Freq. (%)	Micro abrasion Freq. (%)	Resin Infiltration Freq. (%)	Total Freq. (%)	χ^2	p-value
Questionable	15(8.2)	0(0.0)	0(0.0)	15(6.5)	46.092	0.001
Mild	60(32.4)	2(16.7)	1(2.9)	63(27.3)		
Moderate	70(37.8)	10(83.3)	10(29.4)	90(39.0)		
Severe	40(21.6)	0(0.0)	23(67.7)	63(27.2)		

Freq. = Frequency

Among those who received resin infiltration, 23 (67.7%) presented with severe dental fluorosis while among those that had micro abrasion, 10 (83.3%) had moderate dental fluorosis.

Figures 2a and 2b show clinical pictures taken before and after treatment of a 29-year-old man who presented with discolorations of 11 and 12 teeth. He was diagnosed with mild fluorosis and resin infiltration was done on teeth 11 and 12 after scaling and polishing of all the teeth.



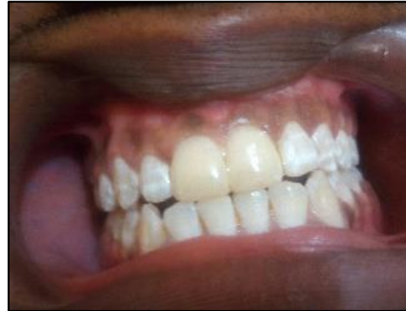


Figure 2a **Figure 2b**

Figures 3a and 3b show clinical pictures taken before and after treatment of a 36-year-old woman who presented with discolored teeth. The complaint was dissatisfaction in the appearances of her teeth, psychological embarrassment, and loss of confidence while speaking in the public. Diagnosis of moderate dental fluorosis was made and resin infiltration was undertaken on the upper right and left anteriors and first premolars — 11, 12, 13, 14, 21, 22, 23 and 24 after routine scaling and polishing

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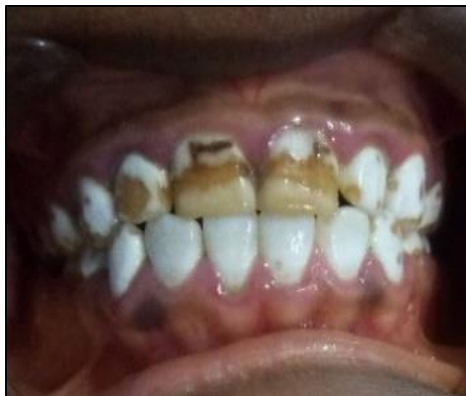

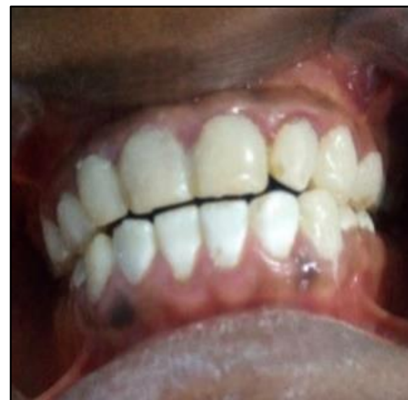

Figure 3a **Figure 3b**

Figures 4a and 4b show clinical pictures taken before and after intervention procedures (resin infiltration after scaling and polishing) were undertaken on upper and lower anteriors and first premolars (11,12, 13, 14, 21, 22, 23, 24, 31, 32, 33, 34, 41, 42, 43 and 44) of severely fluorosed teeth of a 22-year-old young lady.

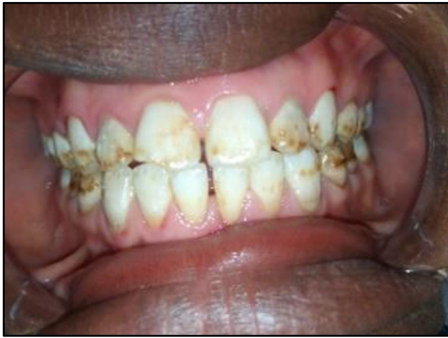

Figure 4a

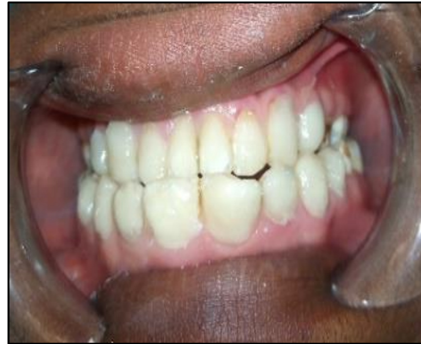

Figure 4b

Figures 5a and 5b show a 17-year-old young man's clinical pictures taken before and after receiving resin infiltration procedures on the following teeth — 11,12,13,21,22,23,31,32, 33,41,42 and 43, which were severely fluorosed

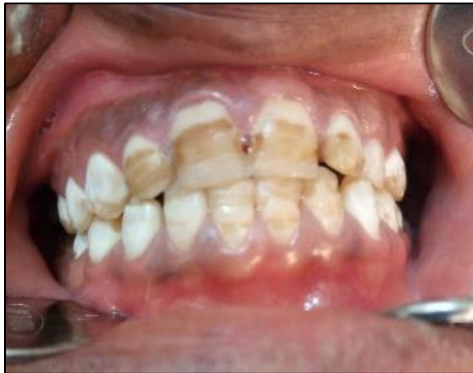

Figure 5a

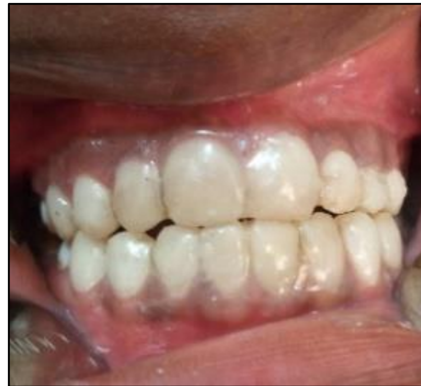

Figure 5b

Figures 6a and 6b show the clinical pictures of a 27-year-old lady who presented with severe dental fluorosis on 11,12, 13, 14, 21, 22, 23, 24, 31, 32, 33, 34, 41, 42, 43 and 44. After scaling and polishing, resin infiltration was done on teeth and glass ionomer cement was applied to cover the exposed dentine where there was gross loss of enamel structure. The restorations on many of the teeth were found to be intact for 18 months except on teeth 31 and 41 of the 27-year-old lady that were re-treated

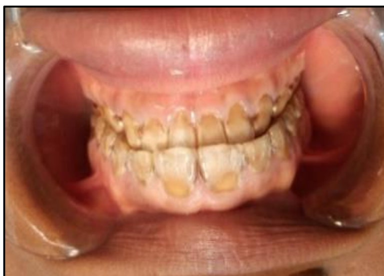

Figure 6a

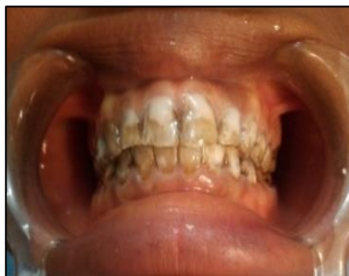

Figure 6b

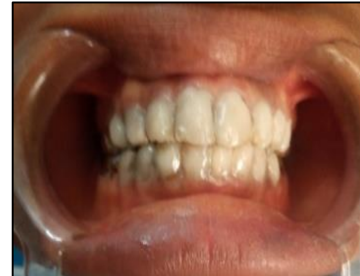

Figure 6c

Discussion

In this present study, the majority of the patients who presented with dental fluorosis were children and adolescents as shown by their mean age of 14.15 years which could be the reason why nearly all studies on dental fluorosis have been conducted with children and adolescents.[18,20-22] Presentation of dental fluorosis was more among the children and adolescents who were below 20 years compared to adults who were above 20 years. Results from this study on age groups of patients in relationship with dental fluorosis presentation in the clinic conformed to previous reports of dental fluorosis which showed that the condition is more common among children than adults.[15, 23] The quest for better appearance of teeth among the children and adolescents compared to adults may be the reason why there was a higher proportion of patients who were in the younger age group than those who were in the older age group.

Females presented with more dental fluorosis than males as reported in other previous studies.[24-27] However, these results were at variance with findings from other studies that reported that dental fluorosis was higher among males than females[23,28] and no sex preponderance.[29] The higher proportion of female patients than male patients presenting with dental fluorosis was also observed in the classes of fluorosis presentation where more females presented all classes of dental Fluorosis, except in the moderate class where males were slightly more. Also, patients with severe dental fluorosis were more among females than males. This is in agreement with a previous report where there were more patients with severe dental fluorosis compared to any other class of dental fluorosis presented in the clinic.[2] The unpleasant appearance of teeth in severe dental fluorosis may be the major reason for these findings. More females presenting with severe dental fluorosis in this study may be due to females' consciousness of better aesthetic appearance than males.

The preponderance of female patients presenting with dental fluorosis in this study may be attributed to the fact that more females sought dental care than males as reported previously in other clinically based studies.[30, 31] It may also be as a result of females having more concern and seeking for better appearance of their teeth and that of their children compared to male patients. The prevalence of dental fluorosis was 11.4% and 11.3% among secondary school adolescents in South West and South East Nigeria respectively.[15,16] This prevalence was lower than 16.7%, the proportion of patients who presented with dental fluorosis in this present study. The observed differences may be due to differences in study design. Results of classification of dental fluorosis using Deans Index among the patients showed that more patients presented with moderate dental fluorosis as compared to other classes of dental fluorosis. This is in agreement with results from a previous study,[23] and contrary to another study where mild fluorosis was reported to be preponderant among school aged children.[15]

Assessment of dental caries on teeth affected by dental fluorosis among patients showed no statistically significant relationship in agreement with a previous study where there was no relationship between the severity of dental fluorosis and dental caries, [32] but contrary to results from other previous studies where the severity of dental fluorosis was reported to have a significant reduction on caries development. [33, 34] Although the initiation and progression of dental caries was strongly linked to fluoride content of daily water consumed, [15,20] the lack of significant relationship between the classes of dental fluorosis and dental caries in this study may be due to multifactorial etiology of dental caries. Other factors such as oral hygiene practices, refined sugar diet, and presence of cariogenic bacteria play important roles.

Among the patients who were seen at the oral health care facility during the study period, a proportion of 200 (16.7%) were diagnosed of dental fluorosis. Out of the patients diagnosed of dental fluorosis, 185 (92.5%) requested for treatment due to unpleasant appearances of their teeth. This proportion was higher than the result from a similar study where 9.3% of patients with dental fluorosis requested for intervention.[2]
Interventions were carried out among 185 out of 200 patients diagnosed with dental fluorosis and the aim was to improve appearances and enhance the patients' self-esteem. All the intervention procedures and the cost implications were explained to each patient to allow them make an informed choice. While all the patients received scaling and polishing, others however received multiple treatments among which were scaling and polishing, micro abrasion, and resin infiltration. The treatment modalities of dental fluorosis in this study conformed with previous studies.[1,34] The majority of patients who had micro abrasion as a treatment modality were in the classes of mild and moderate dental fluorosis. This also conforms to findings from a previous report of treatment options among patients with dental fluorosis.[35] Results from this present study shows that only one patient with mild fluorosis had resin infiltration while majority of the patients with severe dental fluorosis received resin infiltration. This was probably due to the degree of the dental fluorosis as it affects appearances of the teeth, cost of treatment, and a better appearance after treatment. The outcome of resin infiltration significantly improved the recipient's appearances and this is in agreement with other previous studies.[1, 34, 35] The patients were monitored for/between 1-2 years with the majority having their resin restorations intact.

Conclusion

Dental fluorosis causes low self-esteem and negatively affects quality of life among dental patients. It was a reason for dental visit as a significant proportion of the dental out-patients sought for intervention at the study center. Presentation was more among children and teenagers below 20 years. More females presented and requested for intervention than the males. Resin infiltration was found to be cost effective at this facility. More patients presented with moderate class of dental fluorosis, and

dental caries infection was found not to be significantly related to any specific class of dental fluorosis in this study.

References

10. Shahroom NSB, Mani G, Ramakrishnan M. Interventions in management of dental fluorosis, an endemic disease: a systematic review. J of Family Med Prim Care. 2019;8(10):3108-3113.
11. Idon, PI, Enabulele J. Prevalence, severity and request for treatment of dental fluorosis among adult in an endemic region of Northern Nigeria. Eur J Dent 2018;12(2):184
12. Akuno MH, Nocella G, Milia EP, Gutierrez L. Factors influencing the relationship between fluoride in drinking water and dental fluorosis: a ten-year systematic review and meta-analysis. J Water Health 2019;17(6): 845-862. https://doi.org/10.2166/wh.2019.300
13. Albanto AJ, Rezende KMP, Salazar MSM, Bucholdz TAF, Celibrerti P, Ciamponi AL. Dental fluorosis: exposure, prevention and management. Med Oral Oatol Oral Cir Bucal. 2009;14:E103-107.
14. Haritash AK, Aggarwal A, Soni J, Sharma K, Sapra M, Singh B. Assessment of fuoride in groundwater and urine, and prevalence of fuorosis among school children in Haryana, India. Appl Water Sci 2018;8:52.
15. World Health Organization (WHO). Basic methods for assessment of renal fluoride excretion in community prevention programmes for oral health. 2014, WHO. Geneva.
16. Aoba T, Fejerskov O. Dental fluorosis: chemistry and biology. Crit Rev Oral Biol Med. 2002;13:155-70.
17. Sananda D. Biplab G. Fluoride fact on human health and health problems: a review. Med Cli Rev 2016;2(1:2):1–6.
18. World Health Organization (WHO). Guidelines for Drinking Water Quality, 2014, 4th ed. WHO, Geneva.
19. Mohanta A, Mohanty PK. Dental fluorosis-–revisited. Biomed J Sci Technol Res 2018;2:2243-2247. doi:10.26717/BJSTR.2018.02.000667.
20. Rango T, Kravchenko J, Atlaw B, McCornick PG, Jeuland M, Merola B. et al. Groundwater quality and its health impact: an assessment of dental fluorosis in rural inhabitants of the main Ethiopian Rift. Environ Int 2012;43:37–47.
21. O'Mullane DM, Baez RJ, Jones S, Lennon MA, Petersen PE, Rugg-Gunn AJ, et al. Fluoride and oral health. Community Dent. 2016;33:69–99.
22. Del Carmen AF, Javier FH, Aline CC. Dental fuorosis, fuoride in urine, and nutritional status in adolescent students living in rural areas of Guanajuato, Mexico. J Int Soc Prev Community Dent. 2016;6(6):517–522.

23. Rango T, Vengosh A, Jeuland M, Tekle-Haimanot R, Weinthal E, Kravchenko J, et al. Fluoride exposure from groundwater as reflected by urinary fluoride and children's dental fluorosis in the Main Ethiopian Rift Valley. Sci Total Environ. 2014;496:188–19.
24. Okoye LO, Ekkwueme OC, Sote EO, Amaechi BT. Prevalence of dental fluorosis among12-15-year-old students in Enugu Metropolis, Nigeria. Indian J Dent Res 2019;30(3):462-467.
25. Ajayi D, Arigbede A, Dosumu OO, Ufomata D. The prevalence and severity of Dental Fluorosis among secondary school children in Ibadan, Nigeria. Postgrad Med J 2012;19(2):102-6.
26. Akpata ES, Danfillo IS, Otoh EC, Mafeni JO. (2009). Geographical mapping of fluoride levels in drinking water sources in Nigeria. Afri Health Sci, *9*(4), 227–233.
27. Dean HT. The Investigation of physiological effects by the epidemiological method. In: Moulton FR (ed). Fluorine and dental health. Washington, DC: American Association for the Advancement of Science, Publication No. 1942;19:23–31.
28. Faul F, Erdfelder E, Buchner A, Lang A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behav Res Methods*, *41*, 1149-1160
29. Whelton HP, Ketley CE, McSweeney F, O'Mullane DM. A review of fluorosis in the European Union: Prevalence, risk factors and aesthetic issues. Community Dent Oral Epidemiol 2004;32(1):9-18.
30. Clark DC, Hann HJ, Williamson MF, Berkowitz J. Aesthetic concern of children and parents in relation to different classifications of the Tooth Surface Index of fluorosis. Community Dent Oral Epidemiol 1993;21(6):360-364.
31. Curtis AM, Levy SM, Cavanaugh JE, Warren JJ, Kolker JL, Weber-Gasparoni K. Decline in dental fluorosis severity during adolescence: a cohort study. J Dent Res. 2020;99(4):388-394.
21 Verma A, Shetty BK, Guddattu V, Chourasia MK, Pundir P. High prevalence of dental fluorosis among adolescents is a growing concern: a school based cross-sectional study from Southern India. Environ H Prev Med 2017;22(1):1-7.
22 Gopalakrishnan P, Vasan RS, Sharma PS, Nair KS, Hankappan KR. Prevalence of dental fluorosis and associated risk factors in Alappuzha district Kerala. Natl Med J India 1999;12(2):99-103.
23 Demirci M, Tuncer S, Yuceokur A. Prevalence of caries on individual tooth surfaces and its distribution by age and sex in university clinic patients. Eur J Dent 2010;4:270-277.

24 Arvind BA, Isaac A, Murthy NS, Shivaraj NS, Suryanarayana SP, Pruthvish S. Prevalence and severity of dental fluorosis and genu valgum among school children in rural field practice area of a medical college. Asian Pac J Trop Dis Asian Pac Trop Med Press 2012;2:465-469.

25 Sukhabogi JR, Parthasarathi P, Anjum S, Shekar BRC, Padma CM, Rani AS. Dental fluorosis and dental caries prevalence among 12- and 15-year-old school children in Nalgonda district, Andhra Pradesh, India Annals of Medical and Health Sciences Research. 2014;4(SUPPL 3): S245-S252.

26 Mane AB, Revathi S, Sarale DG, Paul CN, Hiremath SG. Studies of dental fluorosis among primary school children residing in rural area of Raschar district, Karnataka. Int J Biol Med Res 2011;2(3):716-720.

27 Singh M, Saini A, Saimbi CS, Bajpai AK. Prevalence of dental diseases in 5- to 14-year-old school children in rural areas of the Barabanki district, Uttar Pradesh, India. Indian J Dent Res. 2011;22:396–399.

28 Idowu EA, Afolabi AO, Fakuade BO, Akintububo OB, Ibiyemi O. Self-medication
profile of Dental Patients Attending a North Eastern Tertiary Hospital in Nigeria. Ann Ibd. Pg. Med 2019;17(2):173-180.

29 Ogbebor OG, Azodo CC. Reasons for seeking dental healthcare services in a Nigerian missionary hospital. Sahara medical journal 2016;19(1):38-43.

30 Taghipour N, Amini H, Mosaferi M, Yunesian M, Pourakbar M, Taghipour H. National and sub-national drinking water fluoride concentrations and prevalence of fluorosis and of decayed, missed, and filled teeth in Iran from 1990 to 2015: a systematic review. Environ. Sci. Pollut. Res 2016;23:5077–5098.

31 Sebastian ST, Soman RR, Sunitha S. Prevalence of dental fluorosis among primary school children in association with different water fluoride levels in Mysore district, Karnataka. Indian J Dent Res 2016;27(2):151–154.

32 Fan Z, Gao Y, Wang W, Gong H, Guo M, Zhao S, et al. Prevalence of brick tea-type fluorosis in the Tibet autonomous region of China. J Epid 2016;26(2):57–63.

33 Garg I, Kumar A. Resin Infiltration for esthetic improvement of mild to moderate non-pitted fluorosis stains in 6-12-year-old Children: a randomized 6-month intervention study. J South Asian Assoc Pediatr Dent 2021;4(2):96-103.

34 Loguercio A, Correia L, Zago C, Tagliari D, Neumann E, Gomes O, et al. Clinical effectiveness of two microabrasion material for the removal of enamel fluorosis stains. Oper Dent. 2007;32:531-538.

35 Gugnani N, Pandit I, Goyal V, Gugnani S, Sharma J, Dogra S. Esthetic improvement of white spot lesions and non-pitted fluorosis using resin infiltration technique: series of four clinical cases, J Indian Sov Pedod Prev Dent. 2014;32:176.