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Mandibular Plexiform Ameloblastoma in Adolescent: A Case Report

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Abstract

Ameloblastoma is an aggressive local benign odontogenic tumour. They can be seen at any age, but around 50% of all tumours are between 20 and 40 years of age. Although it is the most prevalent neoplasm of the jaws, it just reports for 1% of all maxilla and mandible and 11 percent of all odontogenic tumours. Ameloblastoma typically affects the mandibular molar & ramus sites. After inadequate treatment, recurrence is common. They normally have a benign development pattern, although they commonly invade and metastasize locally. The clinical, radiological, histological, and therapeutic methods of a case of a large plexiform ameloblastoma were discussed in this research.

Keywords

Ameloblastoma, Plexiform, Mandible, Odontogenic Tumours.

Introduction

The most frequent odontogenic neoplasm is ameloblastoma. Cusack described Ameloblastoma initially in 1827[1].The first usage of the word ameloblastoma in 1934 has been attributed to Churchill[2].Ameloblastoma is an odontogenic tumour which is benign yet aggressive. The tumour cells, on the other hand, do not produce enamel or hard tissue. Ameloblastomas are recognized for its aggressive development and predilection for recurrence. They can strike at any age, however almost half of all tumours strike people during the ages of 20 and 40. Although it is the most prominent neoplasm of the jaws, it only accounts for 1% of all maxilla and mandible tumours and 11% among all odontogenic tumours[3]. As per World Health Organisation, ameloblastoma, which generally has a follicular or plexiform pattern, is a benign but highly aggressive polymorphic tumour, comprised of the reproducing odontogenic epithelium.[4]. Ameloblastoma is a radiolucent tumour exhibiting a soap bubble or honeycomb pattern that can be unilocular or multilocular. Ameloblastoma is divided into five types based on histopathology: follicular, acanthomatous, granular cell, basal cell, and plexiform.[5]. With prevalence rates of 27.7% and 21.1 percent, respectively, follicular and plexiform ameloblastomas are the most frequent, followed by acanthomatous and granular forms[6].The possibility of a final treatment, the ability of managing the disease with a subsequent operation if a recurrence is detected, the patient's age, the degree of function and growth disturbance, and the likelihood of follow-up examinations are all factors that individuals consider when deciding on the best treatment for ameloblastoma[7].Since the jaws have not fully developed in children, determining the best effective therapy for ameloblastoma can be complex. It should be treated with surgical excision of the whole tumour followed by suitable rehabilitation. We described a case of plexiform ameloblastoma in a 17-year-old male patient, clarifying its clinical characteristics, differential diagnosis, and treatment.

Case Report

A 17 years old male patient reported to the Department of Oral & Maxillofacial Surgery with a chief complaint of swelling in left lower back tooth region since 3 months. The patient had been asymptomatic for 3 months when he developed swelling in the mandibular left posterior area. Initially, the swelling was modest, but it progressively became larger until it reached its current size, which has been consistent for the last month. There was no pain, damage, or discharge in that area when the swelling appeared. Medical and family history have no effect on the matter. All vital signs were in normal ranges after a general assessment.Upon clinical examination, a diffuse, ill-defined swelling was detected on the left lower part of the face, superoinferiorly 1 cm below the ear lobule to inferior border of mandible measuring approx. 2-2.5cm and from the corner of mouth to the angle of mandible anteroposteriorly measuring approx. 3-3.5cm (Figure 1). The swelling was non-tender, hard, and the skin above it seemed normal. Over the swelling, no increase in temperature was anticipated. The lymph node in the left submandibular region was palpable but not painful. Swelling was found in the mandibular posterior area on intraoral examination, extending from the mesial surface of 37 to the pterygomandibular raphae. In the same area, a large oval-shaped opening of

roughly 3 cm x 2.5 cmin size was observed (Figure 2). Upon inquiry, the patient revealed that a previous doctor had attempted to enucleate a cyst but failed. On palpation, the adjacent teeth did not seem to be movable. A preliminary diagnosis of ameloblastoma in the left mandibular area was obtained based on the patient's history and clinical examination.Odontogenic myxoma, central giant cell granuloma, and odontogenic keratocyst were all offered as differential diagnoses. The radiographic examinations revealed a unilateral, well-defined, multilocular radiolucency in the mandibular left angle region, measuring about 4 cm x 3 cm in diameter, extending from the distal surface of the 36 to 38 region anteroposteriorly, superoinferiorly it extended till theinferior border of mandiblewith unerupted partially developed 38 (Figure 3). Under local anaesthetic, an incisional biopsy was performed and submitted for histological analysis. The diagnosis of plexiform ameloblastoma was confirmed by the pathology report, and the patient was planned for surgical excision of the affected mandible.An extraoral submandibular approach (Figure 4)was used to remove the tumour under general anaesthesia, with nasoendotracheal intubation and aseptic measures. The tumour mass was uncovered buccally and lingually. A buccal and lingual osteotomy cut was made after removing the lower second premolars. The patient then underwent a left mandibular segmental resection without exarticulation (Figure 5). Tumor mass along with 1.5 cm of bone margin were removed, and a 2.7 mm titanium reconstruction plate was used to reconstruct the area (Figure 6).Superiorly the plate was secured along with the condyle and inferiorly it was secured along with remaining mandible using screws. As soon as hemostasis was established, a vacuum drain was placed, and layer wise closure was performed. In the post-operative period antibiotics, analgesics and anti-inflammatory medications were administered. Sutures were removed seven days after surgery since the wounds healed uneventfully. Since then, the patient has been monitored on a regular basis (Figure 7, Figure 8). There hasn't been a recurrence recorded yet.

Discussion

Ameloblastoma is an odontogenic tumour that is benign yet aggressive. The tumour cells, on the other hand, do not produce enamel or hard tissue. It accounts for 1% of all radiolucent jaw lesions[3].Ameloblastomas are derived from neoplastics of odontological cyst transformation or residual epithelial rest that has survived from tooth formation; for example, residue from the enamel organ (reduced enamel epithelium) found above the crown of an unerupted tooth, residue in a periodontal ligament of the Hertz epithelial root sheath (Rests of Malassez), or residue of the dental lamina (rests of Serres)[3].

In children, ameloblastoma is a rare condition. The most frequently referenced article is a study of 1036 incidents, with a mean age of 38.9 years, only 2.2 percent (19 of 858) below the age of ten, and 8.7 percent (75 of 858) between the ages of ten and nineteen (Small & Waldron)[8]. This article, although, was written in 1955, when adenoameloblastoma as well as ameloblastic fibroma were both classified as ameloblastomas. In 1962, Young and Robinson[9] documented the first case of ameloblastoma in children, reporting seven instances in children under the age of nine, two of which were ameloblastic fibromas and one of which was odontoameloblastoma. Ord et al.[10] compared Western versus African cases of ameloblastomas among children documented between 1970 to 2001. The average age of the children in this study was 14.3 years (Western) and 14.7 years

(African), confirming that less than 10% of cases occur in children under the age of ten. The gender ratio in adults is 1:1. Male to female ratio in Western children is 1:1.2. In African children, males outnumber females by 1.4 to 1.

The most commonly involved site is posterior mandible, 70% are in the molar or ascending ramus area, 20% in the premolar region, and 10% in the anterior region[11] . Our patient, too, reported with the lesion in ascending ramus area, corroborating the above findings. Ameloblastomas in 10-15% cases are linked with a non-erupted tooth. A large plexiform ameloblastoma was discovered in the ascending ramus and molar area of the jaw in this case, linked to a non-erupted tooth. There are two primary histological patterns: follicular and plexiform, with the former actually being the more frequent. The stroma in both patterns is made up of mature fibrous connective tissue, but the follicular form has islands of epithelial components within it[3]. In contradiction to a follicular form, plexiform refers to the presence of anastomosing areas of odontogenic epithelium. Because it did not meet the histologic criteria set out by Vickers and Gorlin, several pathologists first misdiagnosed it as a hyperplastic epithelial growth of the cystic lining rather than definitive ameloblastoma[12]. Gardner established this by examining histologic sections of 19 cystic lesions of the jaws that showed this plexiform pattern of epithelial growth in 1981, concluding that this pattern should always be characterized as ameloblastoma[13]. Ameloblastoma affects both men and women equally, however greater rates in males have been observed on occasion as per Alok et al[14].

Ameloblastoma in children is challenging to diagnose since most tumours seem radiographically to be dentigerous cysts. Ameloblastoma is linked to an unerupted tooth in 70 to 83 percent of cases, and in our instance, it was linked to the mandibular third molar, similar to the findings of Robinson et al and Shteyer et al.[2][15]Because of the radiological resemblance to a dentigerous cyst, early management would include marsupialization, curettage, and enucleation. Usually when the specimen has been thoroughly examined can a clear diagnosis be made, and at that time it should be assessed whether additional therapy is required[16]. In our study, a similar circumstance occurred, where it was previously identified as a cyst and enucleation was performed.

Depending on the size of the lesion and its clinical characteristics, an incisional or excisional biopsy may be performed.[17]. If a relevant specimen can be obtained, an incisional biopsy is beneficial. This will provide the doctor a definitive diagnosis and allow for a thorough workup before establishing a treatment plan. An excisional biopsy is generally conducted on a patient with a tiny, unilocular lesion in which the clinical impression is an odontogenic cyst or fibro-osseous lesion[17]. Histologically, ameloblastoma is marked by the growth of local structures, such the enamel organ, by epithelial cells organised by collagenous fibrous connective tissue stroma in conjunctive vascular tissue[18]. Differential diagnosis of ameloblastoma involving mandible includes odontogenic keratocyst, central giant cell granuloma, odontogenic myxoma, giant cell lesions of hyperparathyroidism, central haemangioma[14][19].

The treatment of mandibular ameloblastoma is still up for debate. Surgical excision is the recommended therapy. However, when it comes to the scope and kind of operation, there is no universal agreement. While the main goal is to obtain a full resection to avoid tumour recurrence, many studies

have focused on how to do so without conducting a disproportionate operation, which necessitates determining the location, size, and kind of ameloblastoma, as well as the individual's age.Some authors recommended enucleation or relatively limited treatment for children[20][21], whereas Fung et al. (1978)[22] recommended that because young patients have more cancellous bone, the lesion will progress more aggressively with more destruction, causing the surgical procedure complicated and challenging. Conservative local therapy seems to be appropriate in young, growing children to reduce the psychological burden of an aggressive resection and eventual functional or growth difficulties, as well as in older patients to prevent significant surgical complications.It is indeed appropriate in unicystic luminal ameloblastomas if the tumour has not yet progressed beyond the cyst's basement membrane, and in lesions that haven't been diagnosed accurately before[23]. Large or severe ameloblastomas (multicystic) with signs of cortical bone infiltration and soft tissue invasion should undergo extensive surgical therapy[24]. According to Ord et al.[10], solid/ multicystic ameloblastoma or recurring tumours in children should be treated similarly to adults with mandibular resection. If there is cortical breach, patients should be treated with mandibular resection with a minimum margin of 1 cm of cancellous bone and soft tissue resection.Because of his age (growing mandible), a total resection of the tumour (sparing condyle) with at least 1cm of healthy tissue proved to offer a good result in our case.

Other treatment options range from simple surgery to more invasive treatments. Radiotherapy, curettage, and enucleation are examples of conservative treatments. The rates of recurrence fluctuate depending on the technique utilised to treat the initial lesion.

According to several studies, all ameloblastomas addressed conservatively had a recurrence incidence of 55 to 90 percent (enucleation and curettage)[12].Ameloblastoma is a kind of epithelial tumour that is histologically comparable to basal cell carcinoma. As a result, some studies argue that their radiosensitivities must be identical [25]. Radiation therapy, on the contrary, is rarely utilised as a first-line treatment. Gardner considers that radiation should only be used in instances when surgery is not a solution. [26]. Other researchers argue that the treatment of selected individuals with recurrence may include radiation in conjunction with surgery. Over the years, Pinsolle et al. considered that (50 Gy postoperatively) surgery and radiation therapy should be done for mandibular recurrences, soft tissues involvement and positive surgical margins are present after resection.[25].

After tumour excision, mandibular reconstruction is required due to significant abnormalities in mandibular arch integrity and tooth loss. Non-vascularized bone grafts are used in basic reconstruction, as well as dental implants and implant-supported prostheses to replace missing teeth[24][27][28]. A non-vascularized iliac crest grafts could be used to repair patients with a mandibular segmental resection of less than 5 cm. In situations when bone excision leads in a significant continuity defect, a microvascularized free flap repair is necessary[29][30].According to Andrade et al[31] children undergoing resection of mandible can be reconstructed using reconstruction plate or free fibula flap. In their study 17 children who underwent mandibular resection were reconstructed using recon plate. Similarly with the insertion of titanium reconstruction plates, our described case was handled after resection. These plates are a feasible option for recreating the anatomical shape of a patient.

The infiltrative development of the ameloblastoma through the surrounding bone, which causes localised cancellous bone invasion beyond the radiographically apparent boundaries, induces recurrence following first surgical therapy. To a significant part, recurrence is the outcome of an insufficient primary treatment. The recurrence rate varies depending on the location, tumour histology, and surgical resection radicality, and should be studied in a large sample over a significant period of time[32]. Average recurrence rates of 21.1 percent and 45 percent, respectively, were reported by Kim and Jang[33] and Escande et al[34]. Pedro et al[32] found that total recurrence was 29 percent in a study that comprised 31 patients over a decade. In a retrospective study of 239 patients of ameloblastomas, Hong et al.[35] found a 4.5 percent recurrence rate following segmental resection or maxillectomy, 11.6 percent after marginal resection, and 29.3 percent after conservative treatment (enucleation, curettage, and marsupialization), with a statistically important correlation among therapeutic approach and recurrence rate.Experiments have been undertaken to employ several markers to distinguish the kinds of ameloblastoma and minimize recurrences, but no positive findings have yet been obtained[36]. Currently, the probability of recurrence seems to be linked to surgical planning prior to histological subtype assessment.

Conclusion

Ameloblastomas are among the pediatric patients is most frequent between the ages of 12 and 18, with a strong male preference. The posterior mandible is the most prevalent location of occurrence. A preoperative incisional biopsy must be performed to look for a link between the radiographic appearance and the histology type, since a unilocular radiolucent-appearing lesion may be solid, modifying the management. Enucleation and peripheral ostectomy can be used to treat unilocular, unicystic ameloblastomas conservatively, saving a more aggressive treatment for recurrence. Children with solid/ multicystic ameloblastoma or recurring lesions should have mandibular resection, just as adults do with a 1 cm clear border. To eliminate any recurrence, however, long-term and regular follow-up is required. Primary reconstruction with a titanium reconstruction plate is a feasible choice since it provides function and shape as early as possible.

References

1. Amzerin M, Fadoukhair Z, Belbaraka R, Iraqui M, Boutayeb S, M'rabti H, et al. Metastatic ameloblastoma responding to combination chemotherapy: case report and review of the literature. J Med Case Rep 2011;5(1):1–5.
2. Robinson L, Martinez MG. Unicystig ameloblastoma. A prognostically distinct entity. Cancer 1977;40(5):2278–85.
3. Celur S, Babu KS. Plexiform ameloblastoma. Int J Clin Pediatr Dent 2012;5(1):78.
4. Sharma S, Goyal D, Ray A, Gupta N. Ameloblastoma in children: Should we be radical? J Indian Soc Pedod Prev Dent 2011;29(6):74.
5. Reichart PA, Philipsen HP, Sonner S. Ameloblastoma: biological profile of 3677 cases. Eur J Cancer Part B Oral Oncol 1995;31(2):86–99.
6. Eversole LR, Leider AS, Hansen LS. Ameloblastomas with pronounced desmoplasia. J oral Maxillofac Surg 1984;42(11):735–40.
7. Vedtofte P, Hjørting-Hansen E, Jensen BN, Roed-Petersen B. Conservative surgical treatment of mandibular ameloblastomas. Int J Oral Surg

1978;7(3):156–61.

8. Small IA, Waldron CA. Ameloblastomas of the jaws. Oral Surgery, Oral Med Oral Pathol 1955;8(3):281–97.
9. Young DR, Robinson M. Ameloblastomas in children: Report of a case. Oral Surgery, Oral Med Oral Pathol [Internet] 1962;15(10):1155–62. Available from: https://www.sciencedirect.com/science/article/pii/0030422062901500
10. Ord RA, Blanchaert Jr RH, Nikitakis NG, Sauk JJ. Ameloblastoma in children. J Oral Maxillofac Surg [Internet] 2002;60(7):762–70. Available from: https://doi.org/10.1053/joms.2002.33242
11. Adebiyi KE, Ugboko VI, Omoniyi-Esan GO, Ndukwe KC, Oginni FO. Clinicopathological analysis of histological variants of ameloblastoma in a suburban Nigerian population. Head Face Med 2006;2(1):1–8.
12. Gardner DG, Corio RL. The relationship of plexiform unicystic ameloblastoma to conventional ameloblastoma. Oral surgery, oral Med oral Pathol 1983;56(1):54–60.
13. Gardner DG. Plexiform unicystic ameloblastoma: a diagnostic problem in dentigerous cysts. Cancer 1981;47(6):1358–63.
14. Alok A, Panat SR, Singh ID, Singh S. Non-syndromic multiple keratocyst odontogenic tumor: A rare case report. J Indian Acad Oral Med Radiol 2015;27(2):264.
15. Shteyer A, Lustmann J, Lewin-Epstein J. The mural ameloblastoma: a review of the literature. J Oral Surg (American Dent Assoc 1965) 1978;36(11):866–72.Chapelle KAOM, Stoelinga PJW, de Wilde PCM, Brouns JJA, Voorsmit RACA. Rational approach to diagnosis and treatment of ameloblastomas and odontogenic keratocysts. Br J Oral Maxillofac Surg 2004;42(5):381–90.
16. Williams TP. Management of ameloblastoma: a changing perspective. J oral Maxillofac Surg 1993;51(10):1064–70.
17. Iordanidis S, Makos CH, Dimitrakopoulos J, Kariki H. Ameloblastoma of the maxilla. Case report. Aust Dent J 1999;44(1):51–5.
18. Alok A, Singh ID, Singh S, Kishore M. Central giant cell granuloma: A report of 2 cases and review of literature. J Indian Acad Oral Med Radiol 2015;27(1):156.
19. Rapidis AD, Angelopoulos AP, Skouteris CA, Papanicolaou S. Mural (intracystic) ameloblastoma. Int J Oral Surg 1982;11(3):166–74.
20. Isacsson G, Andersson L, Forsslund H, Bodin I, Thomsson M. Diagnosis and treatment of the unicystic ameloblastoma. Int J Oral Maxillofac Surg 1986;15(6):759–64.
21. Fung EH. Ameloblastomas. Int J Oral Surg 1978;7(4):305–10.
22. Black CC, Addante RR, Mohila CA. Intraosseous ameloblastoma. Oral Surgery, Oral Med Oral Pathol Oral Radiol Endodontology 2010;110(5):585–92.
23. Mareque Bueno J, Mareque Bueno S, Pamias Romero J, Bescos Atín M, Huguet Redecilla P, Raspall Martin G. Mandibular ameloblastoma: Reconstruction with iliac crest graft and implants. Med Oral, Patol Oral y Cirugía Bucal 2007;12(1):73–5.
24. Pinsolle J, Michelet V, Coustal B, Siberchicot F, Michelet F-X. Treatment of ameloblastoma of the jaws. Arch Otolaryngol Neck Surg 1995;121(9):994–6.

25. Miyamoto CT, Brady LW, Markoe A, Salinger D. Ameloblastoma of the jaw. Treatment with radiation therapy and a case report. Am J Clin Oncol 1991;14(3):225–30.
26. Belli E, Rendine G, Mazzone N. Ameloblastoma relapse after 50 years from resection treatment. J Craniofac Surg 2009;20(4):1146–9.
27. Chiapasco M, Colletti G, Romeo E, Zaniboni M, Brusati R. Long-term results of mandibular reconstruction with autogenous bone grafts and oral implants after tumor resection. Clin Oral Implants Res 2008;19(10):1074–80.
28. Chana JS, Chang Y-M, Wei F-C, Shen Y-F, Chan C-P, Lin H-N, et al. Segmental mandibulectomy and immediate free fibula osteoseptocutaneous flap reconstruction with endosteal implants: an ideal treatment method for mandibular ameloblastoma. Plast Reconstr Surg 2004;113(1):80–7.
29. Zemann W, Feichtinger M, Kowatsch E, Kärcher H. Extensive ameloblastoma of the jaws: surgical management and immediate reconstruction using microvascular flaps. Oral Surgery, Oral Med Oral Pathol Oral Radiol Endodontology 2007;103(2):190–6.
30. Andrade NN, Shetye SP, Mhatre TS. Trends in pediatric ameloblastoma and its management: a 15 year Indian experience. J Maxillofac Oral Surg 2013;12(1):60–7.
31. Infante-Cossio P, Prats-Golczer V, Gonzalez-Perez L-M, Belmonte-Caro R, Martinez-De-Fuentes R, Torres-Carranza E, et al. Treatment of recurrent mandibular ameloblastoma. Exp Ther Med 2013;6(2):579–83.
32. Kim S-G, Jang H-S. Ameloblastoma: a clinical, radiographic, and histopathologic analysis of 71 cases. Oral Surgery, Oral Med Oral Pathol Oral Radiol Endodontology 2001;91(6):649–53.
33. Escande C, Chaine A, Menard P, Ernenwein D, Ghoul S, Bouattour A, et al. A treatment algorythmn for adult ameloblastomas according to the Pitié-Salpêtrière Hospital experience. J Cranio-Maxillofacial Surg 2009;37(7):363–9.
34. Hong J, Yun P-Y, Chung I-H, Myoung H, Suh J-D, Seo B-M, et al. Long-term follow up on recurrence of 305 ameloblastoma cases. Int J Oral Maxillofac Surg 2007;36(4):283–8.
35. Coleman H, Altini M, Ali H, Doglioni C, Favia G, Maiorano E. Use of calretinin in the differential diagnosis of unicystic ameloblastomas. Histopathology 2001;38(4):312–7.

Figures

Figure 1. Diffuse, ill-defined swelling seen on left lower face

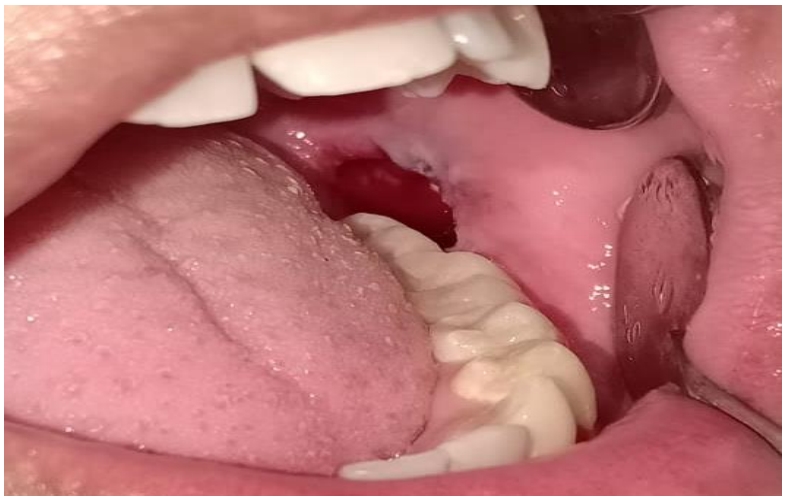

Figure 2. Large oval-shaped opening of roughly 3 cm x 2.5 cmin size

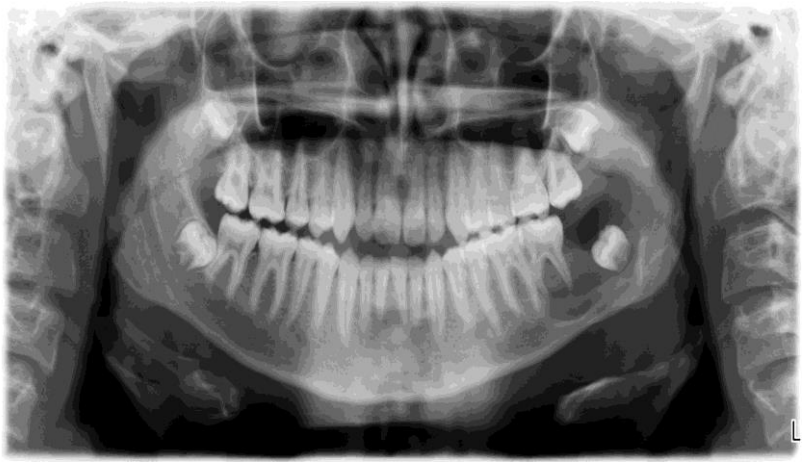

Figure 3. Pre-op OPG showing multilocular radiolucency in the mandibular left angle region

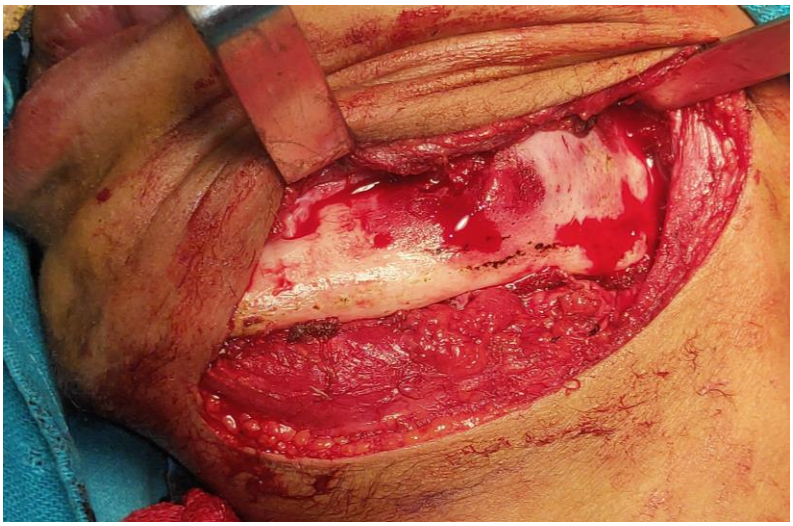

Figure 4. Submandibular Incision

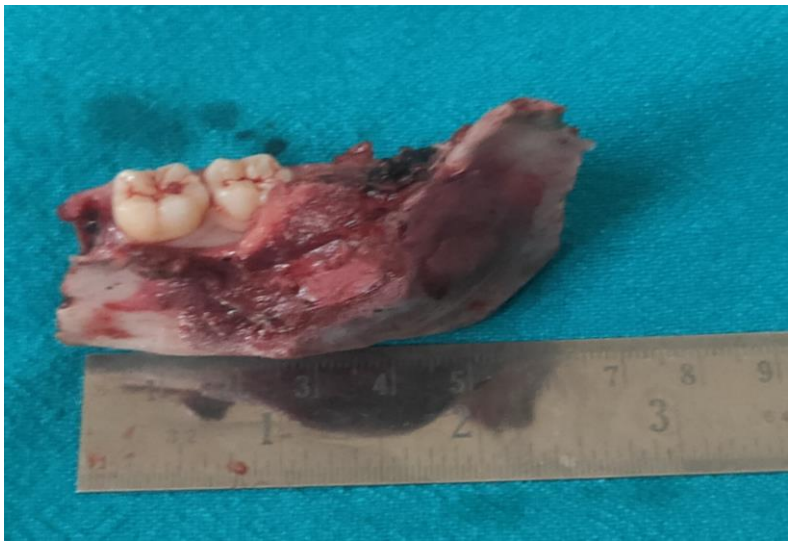

Figure 5. Mandibular segmental resection without exarticulation

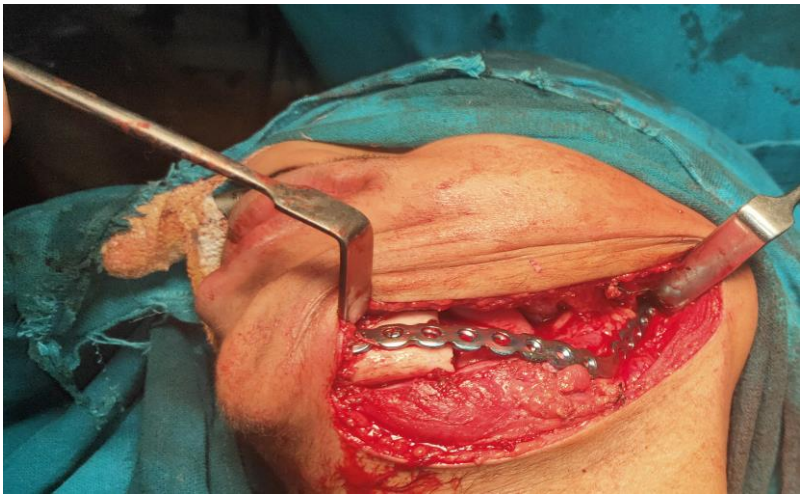

Figure 6. Area reconstructed using Titanium reconstruction plate

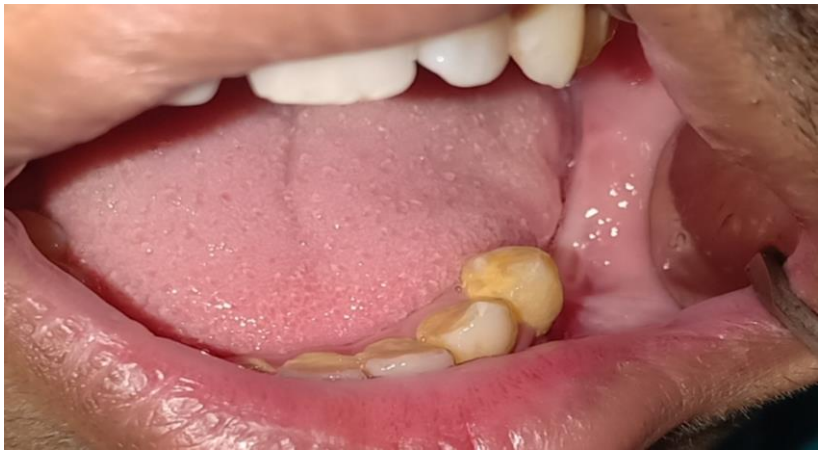

Figure 7. Post op follow up 6 months

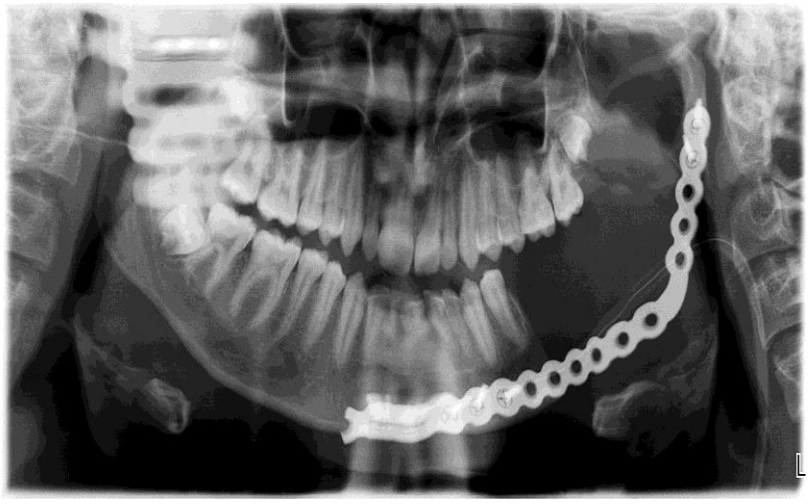

Figure 8. Post op OPG