

# Ameloblastoma: demographic data and treatment outcomes from Melbourne, Australia

T Singh,\*† D Wiesenfeld,\*† J Clement,† A Chandu,\*† A Nastri\*†

\*Oral and Maxillofacial Surgery, The Royal Melbourne Hospital, Parkville, Victoria.

†Melbourne Dental School, Faculty of Medicine, Dentistry and Health Sciences, The University of Melbourne, Victoria.

## ABSTRACT

**Background:** There is a lack of published data on the demographics and treatment outcomes of ameloblastomas treated in Australia. Our objective was to collect this data and compare the findings to other international studies.

**Methods:** A retrospective study of 42 patients with ameloblastoma was conducted at The Royal Melbourne Hospital, Australia. Data on the demographic features, management techniques (ablative and reconstructive), and outcomes were collected and analysed.

**Results:** The majority of tumours were solid/multicystic (81%) and occurred most commonly in the mandible (80.5%). Unicystic ameloblastomas affected a younger age group, with Type 3 being the most common subtype. Overall, the recurrence rate for solid/multicystic ameloblastomas was 14.7%; however, radical surgery was found to have a significantly lower recurrence rate when compared to conservative management ( $p = 0.015$ ), with a mean of 51 months follow-up. Results indicated that vascularized free-flaps had fewer postoperative complications than non-vascularized bone grafts; however, the differences did not reach statistical significance.

**Conclusions:** This is the largest clinicopathological study regarding ameloblastoma management from Australia, and our results support the current literature in recommending radical surgery for the treatment of solid/multicystic and Type 3 unicystic tumours.

**Keywords:** Ameloblastoma, outcomes, reconstruction, recurrence, surgery.

**Abbreviations and acronyms:** AC = ameloblastic carcinoma; PA = peripheral ameloblastoma; SMA = solid/multicystic ameloblastoma; UA = unicystic ameloblastoma.

(Accepted for publication 19 March 2014.)

## INTRODUCTION

Ameloblastoma is an odontogenic tumour that arises within the jaws. Although the majority of ameloblastomas are classified as benign tumours, they are locally invasive and can cause significant morbidity if left untreated. The most up-to-date classification by the World Health Organization (WHO) describes four types of benign ameloblastoma:<sup>1</sup> solid/multicystic (SMA), unicystic (UA), peripheral (PA) and desmoplastic ameloblastoma. The SMA is the most common subtype of ameloblastoma (approximately 80% of cases), and has a predilection for the posterior aspect of the jaws, particularly the body, angle and ramus of the mandible.<sup>2–7</sup>

Treatment of the ameloblastoma is surgical, and can be divided into ‘radical’ and ‘conservative’ options.<sup>8–10</sup> Current evidence supports radical surgery as the mainstay of treatment for all ameloblastomas, with the exception of Type 1 (luminal) and Type 2

(intraluminal) UA, which has been shown to respond adequately to more conservative techniques.<sup>11</sup>

There is geographic variation in the distribution of this tumour;<sup>3,5</sup> however, a review of the literature reveals only a limited number of articles from the Australian population. Several case reports and small case series have recently been published,<sup>12,13</sup> with the last research based study regarding the management of this tumour published in 1995.<sup>7</sup>

Given this paucity of data we conducted a comprehensive clinicopathological review of ameloblastoma recently managed in Melbourne, Australia. In particular, demographics, tumour subtypes, methods of treatment (ablative and reconstructive), and outcomes of treatment were evaluated and compared to the current literature.

## MATERIALS AND METHODS

The oral and maxillofacial surgery and pathology databases from a single institution were searched for

reported cases of ameloblastoma from 2001–2012. Forty-nine cases were identified, and their patient files and original histological slides were reviewed. A specialist oral pathologist and a senior oral and maxillofacial surgical registrar examined the slides using an Olympus® BX51 microscope. The diagnosis of ameloblastoma was confirmed in each case, and its tumour subtype was identified using the current WHO classification system. Where the original slides contained insufficient information for diagnosis or subtype classification, the original formalin-fixed paraffin-embedded tumour blocks were located and reprocessed for additional histopathological examination.

Patient files of the confirmed cases were reviewed, and a database constructed with the following variables: age, gender, preoperative signs and symptoms (pain, swelling, sensory disturbance, mobile teeth, discharge or none), history of tobacco or alcohol use, and method of treatment. Treatment was divided into radical and conservative methods. Radical treatment followed a standard protocol, and involved either a mandibulectomy (marginal or segmental), or maxillectomy (subtotal or total), with a margin of uninvolved soft tissue and bone that was confirmed on specimen radiograph and final histopathological examination. In contrast, conservative treatment consisted of enucleation and/or peripheral ostectomy. Reconstructive techniques and complications, follow-up time periods and outcomes were also recorded. Outcomes fell into two groups: (1) no signs of recurrence; or (2) recurrence – tumour recurrence or death due to disease.

Seven cases were excluded due to insufficient pathological and/or clinical information. Of the remaining 42 cases, one ameloblastic carcinoma (AC) was identified and excluded from the statistical analysis as this malignancy is an inherently different subtype from its benign counterparts. The remaining 41 cases underwent analysis using Minitab® software, and a Fisher's Exact test was conducted on the above variables in relation to patient outcomes. Statistical significance was determined by  $p < 0.05$ .

The Melbourne Health Human Research Ethics Committee granted ethical approval for the study (Reference no. 2011.127).

## RESULTS

### Demographic data

Males (63%) were affected more than females, the mean age of patients at the time of diagnosis was 43 years. Information regarding smoking history and alcohol consumption were poorly recorded in patient files, and thus they were excluded from further analysis.

The majority of tumours were located in the mandible (80.5%) compared to the maxilla (19.5%), with the posterior aspect of the jaws being the most common subsite affected (85%). Nearly all tumours were unilateral (95.1%), with the left and right sides of the jaws affected approximately equally ( $n = 22$  left,  $n = 19$  right). Only two tumours crossed the midline to affect the mandible bilaterally.

### Tumour subtypes

Forty-one (41) cases of benign ameloblastoma were identified and 1 case of ameloblastic carcinoma. The breakdown of the ameloblastoma subtypes can be seen in Fig. 1. SMA was the most common subtype (34 cases), followed by the UA (6 cases). A single case of PA was confirmed; however, there were no established cases of desmoplastic ameloblastoma or malignant ameloblastoma.

### Signs and symptoms

The majority of patients were found to have signs and/or symptoms present prior to their diagnosis. The most common findings were swelling (36.6%), pain (17.1%) and discharge (7.3%), although 36% of the tumours were asymptomatic and discovered incidentally on routine dental examination or radiographs.

### Methods of treatment

The majority of SMA tumours (85.3%) were treated with radical treatment (surgical resection), with the remaining 14.7% treated conservatively. All resective patients had clear surgical margins except for one patient who returned to theatre for a further marginal resection. The reasons for conservative management were essentially patient driven; they either declined

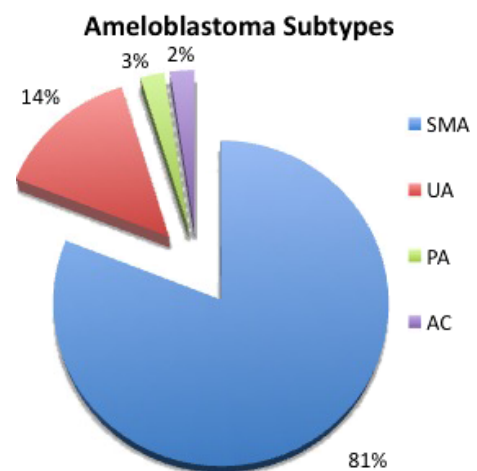


Fig. 1 Ameloblastoma subtypes.

surgical resection, or had complex medical issues that inhibited a radical ablative and reconstructive procedure. Although the numbers of UA were limited (6 cases), most were treated conservatively (66.7%) rather than with surgical resection (33.3%).

## Outcomes

Patients were followed up for an average of 51 months postoperatively. There was an overall recurrence rate for SMA tumours of 14.7%, and with recurrences occurring after a mean of 8.5 years following initial treatment. Further analysis showed that only one of the SMA tumours treated with surgical resection recurred (1/29), and one case died of disease due to direct intracranial extension (1/29), resulting in a combined recurrence rate of 6.9%. In contrast, 60% (3/5) of the SMA tumours treated with conservative management showed evidence of recurrence (Fig. 2). There was a statistically significant difference between these two methods of treatment ( $p = 0.015$ ), with radical treatment having a significantly lower rate of poor outcomes (recurrence or death) compared to conservative management. Of the 6 UA cases, 2 were treated with surgical resection, and 4 with conservative management. The only recurrences occurred in 2 Type 3 UA tumours that were managed conservatively 2 and 5 years previously (33.3% recurrence rate) (Table 1).

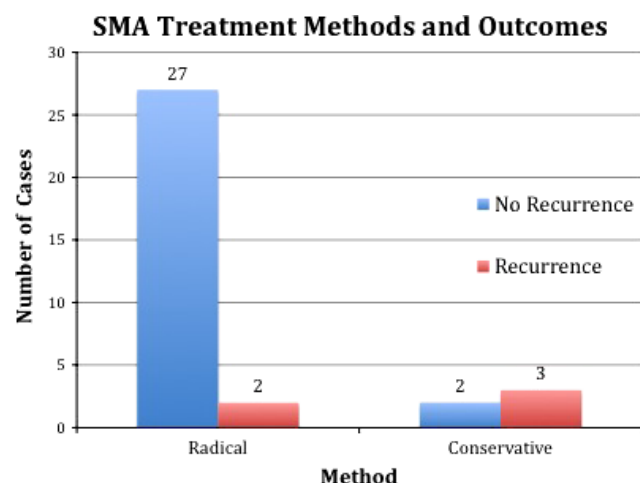
## Methods of reconstruction

Most patients who were managed by surgical resection also underwent a reconstructive procedure (27/31) either immediately following the ablative surgery (92.6%), or in a delayed procedure (7.4%). Two

**Table 1. Summary of ameloblastoma subtypes, location, methods of management, outcomes, and reconstruction**

Subtype	Number	Percent (%)
SMA	34	81.0%
UA	6	14.3%
- Type 1 = 1		
- Type 2 = 1		
- Type 3 = 4		
PA	1	2.4%
AC	1	2.4%
<b>Location</b>		
Mandible	33/41	80.5%
Maxilla	8/41	19.5%
Anterior	6/41	15.0%
Posterior	35/41	85.0%
<b>Methods of treatment</b>		
<b>SMA</b>		
- Radical	29/34	85.3%
- Recurrence 1/29		- 3.4%
- Death 1/29		- 3.4%
- Conservative	5/34	14.7%
- Recurrence 3/5		- 60.0%
<b>UA</b>		
- Radical	2/6	33.3%
- Type 1: 1/2		
- Type 3: 1/2		
- Recurrence: Nil		
- Conservative	4/6	66.7%
- Type 2: 1/6		
- Type 3: 3/6		
- Recurrence: 2/3		- 66.7%
<b>Timing of reconstruction</b>		
Non-reconstructive cases	14/41	34.1%
Reconstructive cases	27/41	65.9%
- Primary	- 25/27	- 92.6%
- Secondary	- 2/27	- 7.4%
<b>Type of reconstruction</b>		
Plate only	2/27	7.4%
Soft tissue only	2/27	7.4%
Bone grafts	9/27	33.3%
- Complications	- 4/9	- 44.4%
- Primary recon 3/4		
- Secondary recon 1/4		
Osseous free flaps	14/27	51.9%
- Complications	- 3/14	- 21.4%

SMA = solid/multicystic ameloblastoma; UA = unicystic ameloblastoma; PA = peripheral ameloblastoma; AC = ameloblastic carcinoma.



**Fig. 2** Solid/multicystic ameloblastoma (SMA) treatment methods and outcomes. Radical treatment resulted in a statistically significant reduced recurrence rate when compared to conservative treatment ( $p = 0.015$ ).

maxillary defects were reconstructed with soft tissue flaps only, and 2 mandibular defects with titanium reconstructive plates only without an osseous component. The remaining 23 cases were reconstructed with hard tissue – 9 with (non-vascularized) iliac crest bone grafts and 14 osseous vascularized free-flaps (iliac or fibula). Unfortunately, 7 patients suffered reconstructive complications including postoperative infection and complete failure of the flap (Table 2).

## DISCUSSION

This is the largest clinicopathological study of ameloblastoma from Australia. It not only contributes to the international literature on this rare tumour, but

**Table 2. Complications of reconstruction**

<b>Bone graft</b>	
- Total loss	2
- Partial loss requiring debridement	1
- Graft infection	1
Total	4/9
<b>Osseous free flap</b>	
- Total loss	1
- Partial loss requiring debridement	1
- Plate infection	1
Total	3/14

allows comparison of the data with other centres. The last study on the management of ameloblastomas from Australia was published in 1995 by Nastri *et al.*<sup>7</sup> which described a combined total of 13 maxillary ameloblastomas from both Melbourne, Australia and Bristol, United Kingdom. More recently in 2013 a study in Queensland, Australia evaluated odontogenic and non-odontogenic cysts and tumours submitted to a histopathology laboratory.<sup>14</sup> Over a 12-month period, 93 odontogenic tumours were collected, with ameloblastoma being the second most common tumour in this population (11 cases) after the keratocystic odontogenic tumour (69 cases).

In our group of patients, the mean age at the time of diagnosis was 43 years with the UA affecting a younger age group (26.8 years) compared to the SMA (46.3 years), a finding consistent with other studies.<sup>3,5,15</sup> Ameloblastoma affects the mandible more commonly than the maxilla<sup>2-6,16</sup> at a ratio of approximately 80–92%:8–20%, with a propensity for the posterior aspect of the jaws.<sup>2</sup> Our results are consistent with these findings as the majority of tumours involved the mandible, particularly the posterior region. Despite these results, multicentre studies have shown geographical variation in ameloblastoma location. In a Korean population the mandible was affected 13 times more than the maxilla, and in a North American group the anterior mandible was the most commonly involved subsite.<sup>3</sup>

Approximately one-third of our cases were found coincidentally, which is in keeping with other studies<sup>5,6</sup> including Becelli *et al.*<sup>2</sup> who found 35% of their cases were asymptomatic at the time of diagnosis. Swelling (36.6%) and pain (17.1%) were the two most common presenting features, and can be easily confused with other pathological entities such as dental disease or odontogenic infection.<sup>17</sup> The maxillary sinus may allow an ameloblastoma to grow to considerable size prior to the development of signs and symptoms such as nasal obstruction, facial pain, globe position changes, or sensory changes via the maxillary division of the trigeminal nerve (V2). Furthermore, the close proximity of important structures in the infratemporal fossa and the central nervous system

means that this tumour may cause significant morbidity and even death.<sup>7,18</sup>

The majority of the tumours in this study were benign (41/42), with only 1 malignant tumour identified (AC). SMA made up the majority of ameloblastomas (81%), followed by the UA (14.3%) and the PA (2.4%). These ratios are consistent with the literature where the proportion of SMA ranges from 70% to 85%,<sup>3,15,19</sup> and the UA 10% to 26%.<sup>1,3,15,19,20</sup>

The SMA behaves in an expansive and locally invasive manner, following the path of least resistance and infiltrating cancellous bone 2–8 mm from the plain radiographic margin of the lesion.<sup>2,8,21,22</sup> There are two surgical approaches to managing the SMA:<sup>20</sup> ‘radical’ or ‘conservative’. Radical treatment involves tumour resection with a margin of uninvolved bone and soft tissue, usually resulting in a segmental mandibulectomy or maxillectomy. Conservative treatment involves enucleation, and curettage with peripheral ostectomy, cryosurgery or chemical cautery (e.g. Carnoy’s solution) employed as adjuncts to surgery. Carlson and Marx in 2006<sup>8</sup> advocated radical treatment of the SMA with 1–1.5 cm margin of uninvolved bone, confirmed on specimen radiograph and histological examination. Their results revealed no recurrences in 82 patients treated with this method after 5 years of follow-up. Over the years research has confirmed a significantly higher recurrence rate with conservative treatment (33–83%) compared with radical treatment (0–19%).<sup>4,18,23–28</sup> Our results support these findings with a statistically significant difference between these two treatment methods ( $p = 0.015$ ). Although most of the recurrences occurred in the mandible, only one recurrence occurred in the maxilla, which resulted in death due to direct intracranial spread of the disease. Thus, the results of this study are consistent with the literature in supporting the radical management of SMA tumours.

The UA also has several histological variants and several classification systems have been described in the literature.<sup>1,11,29</sup> Ackermann *et al.* proposed a three-group classification system (Type 1–3) that relates to the management of the UA.<sup>29,30</sup> Importantly, a UA may have more than one grouping, and thus the entire lesion should be examined by the pathologist to check for mural invasion (Type 3). The majority of UA lesions in our study were Type 3 (67%), which is slightly higher than other groups where approximately 50% are of this subtype.<sup>11,31</sup> Overall, the UA has a lower recurrence rate after conservative treatment compared to the SMA (10–25% vs 33–93%).<sup>8,22,32,33</sup> However, Type 3 (mural invasion) lesions can invade the cancellous bone and thus should be treated in the same method as an SMA.<sup>11,29,30,34</sup> Our results revealed that two out of the three (66.7%) Type 3 UAs that were treated



conservatively recurred. In contrast the Type 3 UA that was treated with radical surgery did not recur, nor did the Type 2 UA (intraluminal) managed conservatively. Despite the small number of UA cases, these results support the recommendation of radical surgery in the management of the Type 3 UAs.

The PA is an uncommon variant of the ameloblastoma, forming 1-10% of all subtypes,<sup>1,3,15,19</sup> and is thought to occur more commonly in an older population.<sup>1</sup> It does not show the invasive characteristics of the SMA, which is reflected in its low recurrence rate following conservative treatment.<sup>1</sup> The single case of PA in our population occurred in a 54 year-old male, and was treated conservatively with no signs of recurrence after 36 months of follow up.

In contrast to the PA, our single case of AC suffered multiple recurrences despite receiving radical surgical treatment. The patient moved overseas at the time of publication, but ongoing correspondence indicates that she has undergone radiotherapy in her new country of residence. The AC is one of the two types of malignant ameloblastoma in the WHO Classification.<sup>1</sup> The malignant ameloblastoma has the same histological features of the benign ameloblastoma, but it has been shown to metastasize and spread to regional lymph nodes or distant sites such as the lung, brain, bone and kidney.<sup>32,35,36</sup> Conversely, AC is a true malignancy with typical features of a carcinoma including cytological atypia, high rate of tumour recurrence and metastasis.<sup>19,37</sup>

Reconstruction of the surgical site can be complex, and completing it at the same time as resection (primary reconstruction) can prove to be challenging.<sup>16</sup> Unrestored defects can result in hypernasal speech, fluid leakage, facial deformity, impaired mastication and phonation, malnutrition, malocclusion and oral incompetence. The majority of our cases that underwent radical surgery were also reconstructed (87.1%) to aid oral rehabilitation (Fig. 3a and 3b). Complications occurred in 44.4% of the cases that received non-vascularized bone grafts compared to only 21.4% in those with free-flaps. Although these differences were not statistically significant, other studies have found free-flaps to have a reduced complication rate, particularly when the surgical defect was longer than 6 cm.<sup>10,24,28,38,39</sup>

In conclusion, we have completed the largest clinicopathological review of ameloblastomas treated in Australia to date (42 cases). The demographic parameters are similar to that found in other international studies. After a follow-up period of approximately 4½ years, a statistically significant higher recurrence rate was found in those SMA lesions treated conservatively compared to those that received radical management. Type 3 UA lesions also had a tendency to recur following conservative management, and this supports

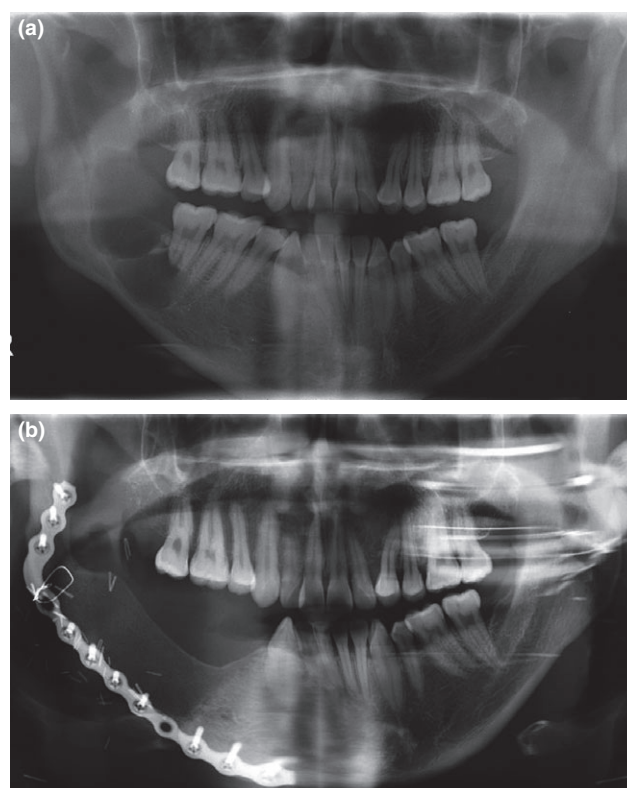


Fig. 3 (a) A solid/multicystic ameloblastoma (SMA) affecting the (right) posterior mandible. (b) Deep circumflex iliac artery (DCIA) free flap with titanium reconstruction plate used to reconstruct the mandible. Note the adequate bone height available for possible dental rehabilitation using osseointegrated implants.

current theories that these lesions should be managed similarly to the SMA. Multiple recurrences were seen in the isolated case of AC, and one death occurred in a maxillary SMA tumour due to intracranial spread. These results highlight the importance of radical surgery in the management of these tumours.

## ACKNOWLEDGEMENTS

Special thanks to the Anatomical Pathology staff at the Royal Melbourne Hospital for their assistance with this study. There was no financial support for this study, and there are no competing interests.

## REFERENCES

- Gardner D, Heikinheimo K, Shear M, Philipsen H, Coleman H. Ameloblastoma. In: World Health Organization Classification of Tumours: Pathology and Genetics of Head and Neck Tumours. Lyon, France: International Agency for Research on Cancer, 2005.
- Becelli R, Carboni A, Cerulli G, Perugini M, Iannetti G. Mandibular ameloblastoma: analysis of surgical treatment carried out in 60 patients between 1977 and 1998. *J Craniofac Surg* 2002;13:395-400.
- Dhanuthai K, Chantarangsu S, Rojanawatsirivej S, *et al.* Ameloblastoma: a multicentric study. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2012;113:782-788.

4. França LJ, Curioni OA, Paiva DL, Vianna DM, Dedivitis RA, Rapoport A. Ameloblastoma demographic, clinical and treatment study: analysis of 40 cases. *Braz J Otorhinolaryngol* 2012;78:38.
5. Reichart PA, Philipsen HP, Sonner S. Ameloblastoma: biological profile of 3677 cases. *Oral Oncol* 1995;31B:86–99.
6. Servato JP, de Souza PE, Horta MC, *et al.* Odontogenic tumours in children and adolescents: a collaborative study of 431 cases. *Int J Oral Maxillofac Surg* 2012;41:768–773.
7. Nastri AL, Wiesenfeld D, Radden BG, Eveson J, Scully C. Maxillary ameloblastoma: a retrospective study of 13 cases. *Br J Oral Maxillofac Surg* 1995;33:28–32.
8. Carlson ER, Marx RE. The ameloblastoma: primary, curative surgical management. *J Oral Maxillofac Surg* 2006;64:484–494.
9. Sachs SA. Surgical excision with peripheral osteotomy: a definitive, yet conservative, approach to the surgical management of ameloblastoma. *J Oral Maxillofac Surg* 2006;64:476–483.
10. Pogrel MA, Montes DM. Is there a role for enucleation in the management of ameloblastoma? *Int J Oral Maxillofac Surg* 2009;38:807–812.
11. Li T, Wu Y, Yu S, Yu G. Unicystic ameloblastoma: a clinicopathologic study of 33 Chinese patients. *Am J Surg Pathol* 2000;24:1385–1392.
12. Sham E, Leong J, Maher R, Schenberg M, Leung M, Mansour AK. Mandibular ameloblastoma: clinical experience and literature review. *ANZ J Surg* 2009;79:739–744.
13. De Silva I, Rozen WM, Ramakrishnan A, *et al.* Achieving adequate margins in ameloblastoma resection: the role for intra-operative specimen imaging. Clinical report and systematic review. *PLoS One* 2012;7:e47897.
14. Johnson NR, Savage NW, Kazoullis S, Batstone MD. A prospective epidemiological study for odontogenic and non-odontogenic lesions of the maxilla and mandible in Queensland. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2013;115:515–522.
15. Scheper MA, Duarte EC, Intapa C, *et al.* Expression of midkine in ameloblastomas and its correlation with clinicopathologic parameters. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2012;114:497–502.
16. Dandriyal R, Gupta A, Pant S, Baweja HH. Surgical management of ameloblastoma: conservative or radical approach. *Natl J Maxillofac Surg* 2011;2:22–27.
17. Singh T, Schenberg M. Delayed diagnosis of oral squamous cell carcinoma following dental treatment. *Ann R Coll Surg Engl* 2013;95:369–373.
18. Sehdev MK, Huvos AG, Strong EW, Gerold FP, Willis GW. Proceedings: Ameloblastoma of maxilla and mandible. *Cancer* 1974;33:324–333.
19. Golubovic M, Petrovic M, Jelovac D, Nenezic D, Antunovic M. Malignant ameloblastoma metastasis to the neck: radiological and pathohistological dilemma. *Vojnosanit Pregl* 2012;69:444–448.
20. Singh TSC. Ameloblastoma: report on two cases and review of the literature. *N Z Dent J* 2009;105:13–17.
21. Gortzak RA, Latief BS, Lekkas C, Slootweg PJ. Growth characteristics of large mandibular ameloblastomas: report of 5 cases with implications for the approach to surgery. *Int J Oral Maxillofac Surg* 2006;35:691–695.
22. Ghandhi D, Ayoub AF, Pogrel MA, MacDonald G, Brocklebank LM, Moos KF. Ameloblastoma: a surgeon's dilemma. *J Oral Maxillofac Surg* 2006;64:1010–1014.
23. Siar CH, Lau SH, Ng KH. Ameloblastoma of the jaws: a retrospective analysis of 340 cases in a Malaysian population. *J Oral Maxillofac Surg* 2012;70:608–615.
24. Bianchi B, Ferri A, Ferrari S, *et al.* Mandibular resection and reconstruction in the management of extensive ameloblastoma. *J Oral Maxillofac Surg* 2013;71:528–537.
25. Eckardt AM, Kokemuller H, Flemming P, Schultze A. Recurrent ameloblastoma following osseous reconstruction—a review of twenty years. *J Craniomaxillofac Surg* 2009;37:36–41.
26. Li Y, Han B, Li LJ. Prognostic and proliferative evaluation of ameloblastoma based on radiographic boundary. *Int J Oral Sci* 2012;4:30–33.
27. Hertog D, Bloemena E, Aartman IHA, van der Waal I. Histopathology of ameloblastoma of the jaws; some critical observations based on a 40 years single institution experience. *Medicina Oral Patologia Oral y Cirugia Bucal* 2012:e76–e82.
28. Chaine A, Pitak-Arnnp P, Dhanuthai K, Ruhin-Poncet B, Bertrand JC, Bertolus C. A treatment algorithm for managing giant mandibular ameloblastoma: 5-year experiences in a Paris university hospital. *Eur J Surg Oncol* 2009;35:999–1005.
29. Philipsen HPRP. Unicystic ameloblastoma. A review of 193 cases from the literature. *Oral Oncol* 1998;34:317–325.
30. Ackermann GLAM, Shear M. The unicystic ameloblastoma—a clinicopathological study of 57 cases. *J Oral Pathol* 1988;17:541–546.
31. Rosenstein T, Pogrel M, Smith R, Regezi J. Cystic ameloblastoma behaviour and treatment of 21 cases. *J Oral Maxillofac Surg* 2001;59:1311–1316.
32. Gardner D. Some current concepts on the pathology of ameloblastomas. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996;82:660–669.
33. Lau SL, Samman N. Recurrence related to treatment modalities of unicystic ameloblastoma: a systematic review. *Int J Oral Maxillofac Surg* 2006;35:681–690.
34. Hertog D, Schulten EA, Leemans CR, Winters HA, Van der Waal I. Management of recurrent ameloblastoma of the jaws: a 40-year single institution experience. *Oral Oncol* 2011;47:145–146.
35. Berger AJ, Son J, Desai NK. Malignant ameloblastoma: concurrent presentation of primary and distant disease and review of the literature. *J Oral Maxillofac Surg* 2012;70:2316–2326.
36. Chaisuparat R, Sawangarun W, Scheper MA. A clinicopathological study of malignant odontogenic tumours. *Histopathology* 2012;61:107–112.
37. Huang CM, Chen JY, Chen CH, Huang CJ. Radiotherapy for a repeatedly recurrent ameloblastoma with malignant transformation. *Head Neck* 2014;36:E1–E3.
38. Pogrel MA, Podlesh S, Anthony JP, Alexander J. A comparison of vascularized and nonvascularized bone grafts for reconstruction of mandibular continuity defects. *J Oral Maxillofac Surg* 1997;55:1200–1206.
39. Foster RD, Anthony JP, Sharma A, Pogrel MA. Vascularized bone flaps versus nonvascularized bone grafts for mandibular reconstruction: an outcome analysis of primary bony union and endosseous implant success. *Head Neck* 1999;21:66–71.

*Address for correspondence:*

*Dr Thasvir Singh*

*Oral and Maxillofacial Surgery Office*

*C/- 2 North*

*The Royal Melbourne Hospital*

*Parkville VIC 3050*

*Email: thasvirsingh@hotmail.com*