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AMELOBLASTOMA - A LITERATURE REVIEW

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ABSTRACT

Ameloblastoma is the most common clinically significant odontogenic tumor. It is relative frequency equals the combined frequency of all other odontogenic tumors. Ameloblastoma are tumors of odontogenic epithelial origin. These are slow growing, locally invasive tumors that run a benign course in most cases. Ameloblastoma is a true neoplasm of odontogenic epithelial origin. It is the second most common odontogenic neoplasm, and only odontoma outnumbers it in reported frequency of occurrence. Its incidence, combined with its clinical behavior, makes ameloblastoma the most significant odontogenic neoplasm. The present article presents a review on Ameloblastoma and its clinical manifestations.

INTRODUCTION

Unicystic ameloblastoma (UA) refers to those cystic lesions that show clinical, radiographic, or gross features of a mandibular cyst, but on histologic examination show a typical ameloblastomatous epithelium lining part of the cyst cavity, with or without luminal and/or mural tumor growth. It accounts for 5-15% of all intraosseous ameloblastomas [1]. Ameloblastoma is classically described as a unicentric, non functional anatomically benign and clinically persistent tumor of jaw. According to Robinson, Ameloblastoma is a locally aggressive neoplasm of odontogenic epithelium that has a wide spectrum of histologic patterns resembling early odontogenesis. It is also known as Admantinoma, Admantoblastoma or as a Multilocular cyst.

Literature review: This tumor probably was recognized first by Cusack in 1827 and described in detail by Falksson in 1879. In 1885 Malassez introduced the term adamantinoma while in 1930; Ivey and Churchill used the name ameloblastoma [2]. The histologically benign ameloblastoma can be divided into three clinic-pathologically distinct types [3]: (1) classic ameloblasroma *(2)* "malignant" ameloblastoma and (3) mural ameloblastoma. There is a histologically malignant ameloblastoma referred to as ameloblastic carcinoma. In reviewing 706 odontogenic tumors Regezi and colleagues (1978) found that ameloblastoma accounted for 11% of sample.

Etiology: They may arise from rests of dental lamina, from a developing enamel organ, from the epithelial lining of an odontogenic cyst or from basal cells of oral mucosa. Ameloblastoma is benign, locally aggressive, infiltrative, odontogenic lesion. It is a true neoplasm of enamel organ like tissue but does not differentiate sufficiently to form enamel [4].

Clinical features : Age: Occurs in any age including children. Adekeys (1980) in their study on a series of 109 Nigerian pts found 68% were in the third and fourth decades of life and 80% were younger than 40 years. It is slightly more in men. Sirichitra (1984) reported male to female ratio of 1.1:1. Ajagbe and Daramola (1987) reported a ration of 4:3. Adekeye reported (1980) reported a ration of 1.7:1. The site of occurrence is mandibular molar region and size is less than 1cm upto 16cm with

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mean size 4.2cm. It is characterised by slow growth and painless swelling. Facial swelling is seen in posterior mandible. Other signs and symptoms include tooth mobility, paresthesia, purulent discharge, trismus and ill fitting dentures [5].

Classification: Clinical classification of Ameloblastoma is as follows;

- Intraosseous ameloblastoma
- Extraosseous ameloblastoma
- Pituitary ameloblastoma
- Adamantinoma of long bones
- Solid/Multicystic ameloblastoma
- Unicystic ameloblastoma
- Peripheral ameloblastoma
- Desmoplastic ameloblastoma [6]

Classification of Unicystic Ameloblastoma: In a clinicopathologic study of 57 cases of unicystic ameloblastoma, Ackermann classified this entity into the following three histologic groups:

Group I: Luminal UA (tumor confined to the luminal surface of the cyst).

Group II: Intraluminal/plexiform UA (nodular proliferation into the lumen without infiltration of tumor cells into the connective tissue wall).

Group III: Mural UA (invasive islands of ameloblastomatous epithelium in the connective tissue wall not involving the entire epithelium).

Another histologic subgrouping by Philipsen and Reichart has also been described: Subgroup 1: Luminal Unicystic ameloblasoma, Subgroup 1.2: Luminal and intraluminal, Subgroup 1.2.3: Luminal, intraluminal and intramural & Subgroup 1.3: Luminal and intramural.

Investigations:

- Radiographs
- Advanced imaging
- Histopathology
- Immunohistochemistry

Radiological features: Classically ameloblastoma described as multilocular, expansile radiolucency that occurs most frequently in the mandibular molar/ ramus area. 85% lesions occur in mandible. Ueno and colleagues 1986 found 93% occuring in the mandible and 97% of these involved the molar region. Extension into ramus occurred in 62%. Mile and coauthors 1991 stated it begins as unilocular lesion and involve into multilocular lesion.

Age: Mean age of patients with unilocular lesions is 26 yrs and 38 yrs with multilocular.

Periphery: Well defined cortical border. Border is often curved. Periphery is ill defined.

Internal structure: It varies from totally radioluscent to mixed appearance due to presence of bony septa creating internal compartments. Septa are coarse and curved. Locules less than 1cm in diameter tend to be numerous resembling a 'honey comb appearence'. Larger locules are few in number and because expansion is invariably present have 'soap bubble appearance' Adekey 1980 found that 10% were unilocular and 90% had honey comb or soap bubble appear.

Effect on surrounding structure: Buccal and lingual expansion of the cortex present. This is especially notable on axial CT and helps to distinguish amelobastoma from dentigerous cyst. The expanded cortex of amelobalstoma may be significantly thinned and intact, with an eggshell-like appearance, or in some instances perforations may be seen.

Relationship to Teeth: Ueno and colleagues (1986) observed that an impacted tooth was involved in 38%of cases; of these, 82% were third molar. Five cases affected a lower second molar, and in two cases the tooth was a premolar. Root resorption is essentially present. Sirichitra (1984) found root resorption in 39% of the cases. Root resorption has knife edge pattern because all of the adjacent roots are cut off along a single linear plane, corresponding to margin of lesion.

Worth's Classic Description (1963) of Ameloblastoma: Worth's (1963) descriptions are especially applicable to mandibular lesions. He divided ameloblastoma into 4 possible radiologic manifestations:

Firstly; it resembles a dentigerous cyst without septa within the lesion, seen most frequently in the ramus, and patient older than 30 yrs.

Another sign: extension of lesion in the body of mandible into the ramus. The presence of some septa, especially if partial loculation can also be seen. If a portion of ramus wall is lost, especially the anterior wall or less frequently the superior wall the lesion is ameloblastoma [7].

Secondly (most common); It consists of cystic appearing cavity with distinctive septa. The trabeculae vary widely in their shapes and arrangements but one frequently sees strands radiating from a common center. Gross caricature of a spider seen in some cases- pathognomic of ameloblastoma. Trabecular arrangement disordered, some are curved suggesting that they may be embracing cystic areas. They also may be thin or coarse, some more than 2mm wide. When there is also a defect in wall of cyst like lesion it is almost certainly an ameloblastoma. Characteristically the angle of mandible is preserved. The inferior aspect may be ballooned out with a significant smooth downward convexity that may be egg shell thin and intact.

Thirdly; less common than second but more than first. It has a multilocular cystic appearance and is seen most commonly in posterior portion of mandible and ramus. Two or three or more cavities appear in continuity, with thin septa separating them. Features include, patients' age especially if older than 30 yrs and loss of continuity of one of the free walls. There is a significant downward enlargement of the inferior border of the jaw which maintains a convex lower border. In the maxilla this multilocular pattern is highly suggestive of ameloblastoma.

Fourthly; solid variety of ameloblastoma. In this pattern the normal bone is replaced with a honey comb appearance in which a cavity is relatively small and fairly uniform in size. The cavity walls are coarse, and the margins of the lesion are lobulated in conformity with the adjacent cavities. Margins separating normal bone from tumor are denser and may be interpreted as a cortex; it is wider and less attached than most cortices of cyst or benign tumors. It is unusual for any unerrupted tooth to be associated with this presentation of the tumor. A combination of cystic and solid type often is found.

Radiologic Features of Maxillary Ameloblastoma: Maxillary ameloblastoma are important as they often extend to adjacent facial structures, have an increase potential for recurrence and may result in significant disfigurement after treatment. Imai & colleagues (1980) reviewed 77 cases and found only 6% occur in maxilla. Male to female ratio is 1.5:1 Imai & colleagues (1980); 2.4:1 Tsaknis & Nelson (1980). Average patient age is 46 years. Imai & colleagues (1980) stated 75-90% occurred in premolar, molar region. Tsaknis & Nelson (1980) observed that maxillary antrum is involved in 12 of 24 cases and 6 of 8 recurrences shows sinus involvement. Radiologically when the antrum was involved there was destruction of antral wall, antral cloudiness and thickening of lining membrane.

Radiologic Features Of Malignant Ameloblastoma: Primary lesion occurs in the mandible 80% (Buff & colleagues 1980), 70% primary lesions occur in third molar region. Slootweg and Muller (1984) found 90% of 20 cases of malignant ameloblastomas in mandible. The features were similar to radiologic features of ordinary ameloblastoma. A dense fibrous stroma may be found in CT.

Advanced imaging of ameloblastoma: Cohen & colleagues (1985) discussed utility of CT in evaluating four extensive cases involving the mandible and one in the maxilla. In maxilla extension into infratemporal fossa or soft tissue extent can be seen in CT which is not seen on conventional plain radiography. Axial CT scans help to determine buccal and lingual extension. Heffez & colleagues (1988) discussed role of MRI in its diagnosis. It is useful when decreased CT attenuation resulting from fibrosis and edema make it difficult to delineate the interface of tumor and normal tissue, especially after radiation therapy or previous surgery. It also helps the clinician to distinguish clinician between solid structures and fluid thus useful in planning surgical margins.

Histologic features: Conventional solid/ multicystic intraosseous ameloblastomas show a remarkable tendency to undergo cystic change; grossly most tumors have varying combinations of cystic and solid features. Several subtypes of conventional ameloblastomas are recognized. These are: follicular, plexiform, acanthomatous, granular cell, desmoplastic and basal cell types

Treatment/ prognosis: The Unicystic ameloblastomas diagnosed as subgroups 1 and 1.2 can be treated conservatively (careful enucleation), whereas subgroups 1.2.3 and 1.3 showing intramural growths require treated radical resection, as for a solid or multicystic ameloblastoma. Following enucleation, vigorous curettage of the bone should be avoided as it may implant foci of ameloblastoma more deeply into bone. Chemical cauterization with Carnoy's solution is also advocated for subgroups 1 and 1.2. Subgroups 1.2.3 and 1.3 have a high risk for recurrence, requiring more aggressive surgical procedures. This is because the cystic wall in these cases has islands of ameloblastoma tumor cells and there may be penetration into the surrounding cancellous bone [8]. Late recurrence following treatment is commonly seen, the average interval for recurrence being 7 years. Recurrence is also related to histologic subtypes of Unicystic ameloblastoma, with those invading the fibrous wall having a rate of 35.7%, but others only 6.7%. Robinson and Martinez (1977) found an overall 67% recurrence rate. Gardner and colleagues (1987) reported 71% recurrence rate. Tsaknis and Nelson (1980) found 75% recurrence involved the maxillary sinus. Recurrent lesions were managed by marginal resection, segmental resection or hemisection. Maxillary lesions are more difficult to manage especially when antrum is involved [9].

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