Ameloblastic Fibrodentinoma - A Case Report

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Abstract: A case of ameloblastic fibrodentinoma of the mandible in a 27-year-old man is presented. Under general anesthesia the mass was excised. Histologically, the lesion was characterized by neoplastic proliferation of odontogenic epithelial, mesenchymal tissues and dentin-like hard tissue. He showed no signs or symptoms of recurrence during the follow-up period.

Key words: Ameloblastic fibrodentinoma, Mandible, Surgical treatment

INTRODUCTION

Ameloblastic fibrodentinoma (AFD) was formerly called dentinoma. AFD is histologically similar to the ameloblastic fibroma, and is composed of odontogenic mesenchyme, odontogenic epithelium, dentin matrix in the form of amorphous hyaline materials and dysplastic dentin which can be seen in the lesion⁰. The WHO classification⁰ defines this lesion as follows: “A neoplasm similar to ameloblastic fibroma, but also showing inductive changes that lead to the formation of dentine”.

AFD is a very rare odontogenic tumor. Since the first case was reported by Straith⁳ in 1936, only 35 cases have been reported worldwide.

The purpose of this article is to report a case of AFD occurring in the mandible.

CASE REPORT

On November 17th, 1997, a 27-year-old man was admitted to our clinic complaining of a painless swelling in the premolar region of the left mandible. Seventeen months before, a radiolucent lesion below the left mandibular first premolar was noted during a routine dental examination at the department of preventive dentistry in our hospital, but the mass had not been treated. The expanding mass was noticed 1 month before consulting our department. Oral examination revealed a firm painless swelling in the left gingivo-buccal region from the canine to the molar teeth. The first premolar and molar teeth were vital, but the second premolar tooth was not.

Preoperative panoramic radiography revealed a round-shape radiolucent area of the left mandibular region, and the second premolar tooth showed resorption of the root (Fig. 1). A clinical diagnosis of the mandibular tumor was made.

Under general anesthesia the mass was excised, and the second molar tooth was extracted. Some of the cortical bone overlying the mass was resorbed and the mass was en-capsulated by the connective tissue. The root of the second premolar tooth was sharply resorbed. No recurrence was observed in the postoperative period of 21 months.

HISTOLOGICAL FINDINGS

Histologically, the lesion was characterized by neoplastic proliferation of odontogenic epithelial and mesenchymal tissues (Fig. 2). In the mesenchymal tissues, some odontogenic cells mimicking dental papilla cells were identified. Some eosinophilic hard tissue was seen adjacent to epithelial and mesenchymal tissues. The hard tissue appeared immature bone embedding odontogenic
like cells in the matrix (Fig. 3). However, the matrix had neither lamellar structure like mature bone, nor dentinal tubles. The hard tissue was regard as osteoid dentin, because odontogenic cells were associated with it. Immunohistochemistry of the rabbit anti-human keratin (DAKO) revealed positive results of these cells (Fig. 4). No enamel formation was found.

**DISCUSSION**

In 1993, ULMANSKY et al.\textsuperscript{13} reviewed the literature and evaluated 33 AFD cases reported since 1936. Then, Akal et al.\textsuperscript{9} reported 2 additional cases of AFD. Therefore, Only 36 cases, including our case have been reported in the world. ULMANSKY
et al. reported that AFD is more common in males than in females (ratio 2:1), and is more common in the mandible than in the maxilla (ratio 3:1). Although AFD is seen in every generation, it affects persons below 30 to 35 years of age.

Pathologically, AFD is similar to the ameloblastic fibroma as it is composed of strands, cords and islands of odontogenic epithelium embedded in cell-rich, primitive ectomesenchyme resembling the dental papilla. In addition, abortive dentin, is deposited, often preceded by the appearance of a hyalinization zone. The odontogenic epithelium is often found adjacent to the dentine. Poorly mineralized or abortive dentin usually contains entrapped odontogenic epithelium and mesenchymal cells.
Takeda suggested that dysplastic dentin could be induced not only in ameloblastic fibromas but also in odontogenic fibroma-like lesion. The former is classified as ameloblastic fibrodentinoma, which may be develop into ameloblastic fibro-odontoma. The latter is a hypothetical entity, which might be called odontogenic fibroma-like lesion with marked induction of dentin. In our case, immature dentin adjacent ameloblastic epithelial like cells were admitted and these cells revealed positive results of the keratin staining. Furthermore these cells were found in the dentin. This may suggest that these ameloblastic epithelial cells induce immature dentin. Calcification of this immature dentin in our case may be develop into an ameloblastic fibro-odontoma.

The biological nature of this tumor remains controversial. It has been suggested that it is not a true neoplasm but a hamartoma due to the limited growth potential. AFD may be a stage in the development from ameloblastic fibroma to odontoma.

Although recurrence was uncommon in a previous study, malignant transformation of the ectomesenchyme component of this tumor likely occurs, resulting in ameloblastic fibro-dentinosarcoma. Continuous observation is needed due to the malignant transformation.

REFERENCES


