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GIANT CELL LESIONS OF THE JAWS

Dental Science

Dr. Kanupriya Gupta MDS, Oral And Maxillofacial Pathology ,Senior Research Fellow, Faculty of Dental Sciences, IMS, BHU, Varanasi (U.P.) INDIA-221005. - Corresponding contributor

ABSTRACT

This article will review current thoughts with regard to the etiology, histopathology, diagnosis, and management of giant cell lesions of the jaws. It will attempt to point out the differences between these lesions and giant cell lesions elsewhere in the body.

KEYWORDS:

Introduction-

Giant cell lesions of the jaws were separated out from other jaw lesions by Jaffe in 1953[1] when they were termed "giant cell reparative granulomas." The concept at that time was that these lesions only seemed to occur in the jaws, they were found in the first two decades of life, more frequently in females (approximately 2:1), and were believed to be related to the teeth in some way, though they were not truly thought to be an odontogenic lesion. One idea at that time was that the giant cells involved in the lesion may have been derived from the odontoclasts, which led to resorption of the deciduous teeth. This might explain why they were found in a certain age group and generally found in areas of the jaws that had previously held deciduous teeth.

It still remains unclear whether this lesion occurs anywhere else in the body. A very similar lesion does occur in the hands and feet [2] but its exact relationship is unknown. Although other lesions containing giant cells do occur in other bones of the body, they are much less frequent and are generally felt to be variants of other tumors, most often a low-grade osteosarcoma. Whether these other types of giant cell lesions also occur in the jaws has been hotly debated for many years, and is still undecided.

Initially it was believed that the jaw lesions were indeed reparative lesions and would resolve spontaneously,[3,4] which is why they were not found in older patients. The origin for this idea is difficult to confirm, and there are few reports in the literature of these lesions actually resolving. Worth in the last edition of his radiology textbook in 1981[5] does describe a group of central giant cell granulomas, which were treated by diagnostic biopsy only and then followed radiographically, and the majority of them did indeed appear to resolve over a period of time. Occasional articles over the years do appear to have confirmed this, particularly after biopsy.[6] At the present time, however, the overwhelming opinion is that these are not in fact self-healing lesions and will continue to increase in size without definitive treatment. It is even believed that a subgroup may be more aggressive than most and require more aggressive treatment[7] extending as far as "en bloc" resection[8] However, all attempts to diagnose the more aggressive type preoperatively have been unsuccessful.[9-11] A soft tissue variant of this lesion known as a peripheral giant cell granuloma, or giant cell epulis, is seen on the gingiva and is felt to represent a similar lesion, but is normally considered less aggressive and responds well to local excision.[12]

Histologic features

Diagnosis of central giant cell granuloma is normally made histologically from an incisional biopsy. The appearance is generally distinctive with multinucleated giant cells spread throughout the lesion but often focal in distribution around areas of possible hemorrhage. This is in contrast to the giant cell tumor of long bones, where the giant cells are more evenly distributed. There is generally a spindle cell matrix with possible areas of hemorrhage. The giant cells can have up to 30 nuclei fairly evenly distributed, unlike the giant cells of tuberculosis, which are arranged in a horseshoe shape. Interestingly enough, studies have shown that it may be the spindle cell, which is the active cell in this lesion,[13] and the giant cells are osteoclasts.[14] Similar histological appearances are seen in the following lesions, which must often be differentiated:

1. The brown tumor of hyperparathyroidism. If there is any doubt (with an aggressive lesion, a recurrent lesion, an atypical lesion or multiple lesions), hyperparathyroidism should be excluded with serum calcium and phosphate determination and in many cases also a parathormone assay.
2. The aneurysmal bone cyst. This lesion has more hemorrhage in it and also cystic areas, but many authorities agree that it actually represents a cystic variant of the central giant cell granuloma.
3. Cherubism. Although the histological appearance in cherubism is similar, the clinical history is different with multiple lesions, and a male:female ratio of 2:1. It is an autosomal dominant with higher penetrance in the male. Histologically there are also subtle differences from the central giant cell granuloma with fewer giant cells in cherubism and perivascular cuffing. Additionally, the genetic basis of cherubism has now been identified with a gene defect on chromosome 4p 16.3, which encodes the binding protein SH3 BP2,[15-19] and a test for this is now becoming available.

Conclusion-

Conventional management is surgical and consists of enucleation and curettage. Despite this, a recurrence rate of 15-20% is often quoted, and in these cases treatment may need to be more aggressive and may need to consist of an "en bloc" resection. However, because of the confusion regarding the etiology of this condition (whether it is a benign tumor, a reactive lesion, an inflammatory lesion or even a self-healing lesion), alternative medical treatments have been introduced over the last 15 years and are felt to have promise in some cases.

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