**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 giant lesions and giant cell lesions elsewhere in the body.

**KEYWORDS:**

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 reparative 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 of 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.

**References**


