A GCT of Scapula – A Rare Site of a Common Tumor

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Abstract:
We report a case of GCT of scapula with a 4 Years of follow up. GCT of scapula is very rare and diagnosis may be missed both clinically and radiologically. In this case we report the the course through which we reached the diagnosis, the treatment given and the functional outcome at the end of 4 years

Key words: giant cell tumor, scapula

Introduction

We have reported this case with a 4 years of follow up and functional results at the end of 4 years, with the purpose of emphasizing that GCT of flat bones, especially of scapula is very rare and the diagnosis may be missed, both clinically and radiologically, unless there is a high index of suspicion. On the other hand, the diagnosis of other conditions e.g. hyperthyroidism must be ruled out satisfactorily before making the diagnosis of GCT by serum chemistry and parathormone levels.

Case Report
30 years old female was admitted to department of orthopaedics for pain in left shoulder. She was suffering from intermittent pain in left shoulder which was mild in nature. She had recently noted a slowly growing, bulging mass on the posterior aspect of left shoulder since past 6 months. Physical examination revealed diffuse, firm and non tender swelling in left scapular region measuring 7 by 8 cm. Which extended from axillary fold to inferior angle of scapula posteriorly. Surface was smooth and skin over it was restricted due to mechanical block. Liver function tests were unremarkable. Serum calcium and phosphorous were within normal limit. Serum parathormone was within reference range. A simple shoulder view and CT were taken. X ray of left shoulder showed expansile lytic lesion with soap bubble appearance (Fig 1). CT of scapula revealed destructive lesion involving scapula with soft tissue extension (Fig 2). Under general anaesthesia, patient in prone position tumor approached through posterior approach. A greyish, bony hard mass of 7 by 8 cms size was found to be present arising below the spine of scapula and the tumour had invaded from posterior to anterior aspect of scapula. The portion of whole of the scapula along with the tumour with surrounding soft tissues was resected and sent for histopathological examination. Acromion process and angle of scapula were saved (Fig 3). Histopathological report showed cellular tumour involving bone and soft tissue. It is composed of oval to sphenoid cells. Eosinophilic cytoplasm and round to oval tissue, occasional mitotic figures seen. Amidst these cells are seen giant cells with multiple nuclei resembling above described cells. Some areas showed increased vascularity and dilated cystic spaces (Fig 4). Immunohistochemistry marker study of tumour mass done. CD34, CD68 IHC marker were positive suggesting giant cells and stromal tissue positive. These study were in favour of Giant cell tumour. Post operatively early exercises were started for elbow and hand passive and active movements of left shoulder encouraged subsequently. Patient was able to use her left upper extremity for most of her needs.

Discussion
GCT of the, previously known as myeloid sarcoma, tumor of myeloplasax, osteoblastoclastoma and osteoclastoma, has a distinctive microscopic appearance, and its diagnosis is usually not difficult despite the fact that the gross appearance of a GCT is less characteristic than its microscopic appearance.

The tumor is usually seen as a soft, brown mass within which areas of hemorrhage, which appears dark red, and areas of collagen, which appear gray, may be observed. Although Cooper first reported GCT in the 18th century. It was in 1940 that Jaffe and Lichtenstein described GCT.
in detail to distinguish it from other tumours. Although many different types of bony neoplasms may contain giant cells, GCT has giant cells as its most prominent component.

The lesion almost always involves the epiphysis or the ends of long bones. In long bones, GCT is considered an epiphyseal lesion. A diagnosis other than GCT such as an osteosarcoma rich in giant cells or an aneurismal bone cyst, must be considered if lesion containing giant cells is seen in the metaphysis or the diaphysis of a long bone.

GCT usually occurs after completion of maturation of the skeleton. More than 80% of the patients with GCT are between 20-40 years of age. About 75-90% of GCTs occurs in long tubular bone. More than 50% of GCTs arise in distal femur and proximal tibia. Other common sites of involvement include the distal radius and sacrum and other less common sites includes flat bones like ribs, skull, patella sternum and clavicle. The rarity of occurrence of GCT at these sites may often lead to misdiagnosis, both clinically and radiologically; however, if a giant cell lesion is seen in the flat bone, it is important to rule out hyperparathyroidism and giant cell rich osteosarcoma before making diagnosis of GCT.

On roentgenogram, a characteristically, purely lytic lesion with destruction is seen extending to the end of the bone. Mineralization with in lesion is lacking. The center is most radiolucent with increasing density towards the periphery. These tumours present with cortical thinning and may expand into the soft tissues surrounding the bone, or they may expand the bone extensively, remaining within the eggshell thin rim of periosteal new bone.

Histopathologically, there is proliferation of two cell populations. There are numerous multinucleate giant cells with an average more than 50 nuclei. In mixed with them are mononuclear cells. Which are of uniform size and round to oval in shape, the nuclei of the giant cells resemble those of the mononuclear cells. There is no cytologic atypia and atypical mitotic figures are absent in a typical GCT. GCTs may show secondary aneurismal bone cyst like changes.

We have reported this case with the purpose of emphasizing that GCT of flat bones, especially of scapula is very rare and the diagnosis may be missed, both clinically and radiologically, unless there is a high index of
suspicion. On the other hand, the diagnosis of other conditions e.g. hyperparathyroidism must be ruled out satisfactorily before making the diagnosis of GCT by serum chemistry and parathormone levels.

### References


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