Introduction:
The modern interpretation of GCT is predominantly osteoclastogenic stromal cell tumors of mesenchymal origin.[1] According to Dahlin's bone tumours; GCT is a distinctive neoplasm of undifferentiated cells. The multinucleated giant cells apparently result from fusion of the proliferating mononuclear cells, and although they are a constant and prominent part of these tumors.[2] Seventy percent of patients are between 20 and 40 years of age at the time of presentation. It is very unusual for GCT to occur in patients younger than 20 years or older than 55 years.[3,4] GCT is seen with a slightly increased frequency in females. The typical giant cell tumor (GCT) is a solitary neoplasm that occurs in the epiphysis or metaphysis of long bones. The most common sites include the distal femur, proximal tibia and distal radius. The sacrum, distal tibia, proximal humerus, proximal femur, pelvis and proximal fibula are not infrequent sites. Rarely, bones of the hand and feet, vertebral bodies and ribs may be involved.[5]

Case Report:
A 75 years old male was admitted in our hospital with a complaint of swelling over his left forearm since 3 years. He had history of trauma 1 year back. Physical examination revealed swelling over the distal outer aspect of his left forearm. On palpation, the swelling was tender, compressible, nonreducible and pulsatile at distal end of radius. Movement at wrist, fingers and elbow was full in range and was painless with no neurovascular deficit. Radial pulse was 74 per minutes. Systemic examination was within normal limits. All biochemical investigations were within normal limits. Radiological picture of the swelling showed expansile lytic lesion with no surrounding sclerosis. (Figure 1)

The patient underwent fine needle aspiration cytology. Cytomorphology revealed highly cellular smears comprising of singly scattered and cohesive clusters of numerous, large osteoclastic giant cells which are often attached to the periphery of the cluster of spindle cells (Figure 2,3). Occasional mitotic activity were noted. Nuclear atypia was absent in background stromal cells. Again the clinical and radiological findings were very important as there are many lesions which contain numerous osteoclast type giant cells. In view of clinical presentation with history of trauma, gradually enlarging mass & radiological finding of expansile lytic lesions, cytomorphological diagnosis of GCT was offered in this case. The patient was surgically treated with curettage and autogenous bone grafting. Tumour tissue was sent for histopathological examination for further confirmation. Cytological features were in conjunction with...
histological features of giant cell tumor. Postoperatively, the patient made a full recovery following the surgery with no neurological deficit. At subsequent follow-up no recurrence was found till one year.

**Discussion:**

GCT of bone is an uncommon neoplasm accounting for 4-5% of all primary bone neoplasms. GCTs are more common in female and 20-40 years of age group at the time of presentation. It is very unusual for GCT to occur in patients younger than 20 years or older than 55 years. Edward et al studied behavior of GCT in elderly patients, which is not different from lesions occurring in young patients. The location, radiographic features of GCT are similar in all age group. In fact in elderly these lesions are less aggressive than those in younger patients. The distal end radius is a site commonly affected by various lytic lesions and their clinical and radiological findings often overlap posing difficulty in diagnosing these lesions. GCT in the present case, involving distal end radius in 70 years of male patient, is rare for its age of occurrence. GCT needs to be differentiated from other lesions such as aneurysmal bone cyst (ABC), giant cell rich osteosarcoma, brown tumour of hyperparathyroidism, giant cell reparative granuloma (GCRG), metastatic carcinoma, pigmented villonodular synovitis or GCT occurring with Paget’s disease.

ABC may mimic GCT and presents as slowly enlarging mass in young patients and adolescents. Aspirates from ABC hemorrhagic with sparse cellular yield comprised of, scattered osteoclastic giant cell fibroblast, hemosiderin laden macrophages. Radiological evidence of extremely rapid disappearance of bone structure in less than 3 weeks, the apparent “blow-out” expansion of bone and the appearance of a fluid-fluid level on the computed tomography (CT) scan and magnetic resonance imaging (MRI) all suggest a diagnosis of ABC formation.

However, if a giant cell lesion is seen in the long bone is important to rule out giant cell-rich osteosarcoma and hyperparathyroidism before making the diagnosis of GCT. The major challenge is to differentiate giant cell rich osteosarcoma from malignant giant cell tumor. Both the lesions will have anaplastic, spindle shaped, stromal cells with a heavy population of osteoclast like giant cells. The radiographs of both may poorly defined lytic lesions as was seen in our case. Meticulous examination of slides is required to detect direct formation of osteoid by malignant spindle cells, a feature that is in favor of the giant cell-rich osteosarcoma as the management or giant cell tumor is curettage while that for giant cell-rich osteosarcoma is amputation. Additional helpful criterion is young age of the patient, which favors giant cell-rich osteosarcoma as chances of developing giant cell tumor are remote in a patient who has not reached skeletal maturity. Brown tumour typically involves diaphysis of long bone. Radiological features of GCT, giant cell reparative granuloma, and brown tumour of hyperparathyroidism are often indistinguishable. Distinction from hyperparathyroidism can be easily made on the basis of serum calcium, phosphate, alkaline phosphatase, and parathormone levels.

Giant cell reparative granuloma is an uncommon benign intraosseous reactive lesion. It needs to be differentiated from GCT bone as its radiological and histological findings may mimic GCT. Some author have regarded as GCT & giant cell reparative granuloma as a continuum of same disease process modified by age of the patient, location & possibly by other factors.

Fifteen to 50 percent of cases of pigmented villonodular synovitis involve bone and its clinicopathologic and radiologic features can mimic giant cell tumor. Because the process arises in joints, the bone involvement is almost always epiphysseal and metaphysseal, identical to the bone destruction pattern seen in giant cell tumor. A pre-operative MRI showing extensive synovial membrane involvement and secondary bone invasion can guide the diagnosis. Some carcinomas will metastasize to the ends of long bones. Moreover, some carcinoma, including carcinoma of the breast, kidney, lung and pancreas, contain heavy populations of osteoclast-like giant cells. This histologic pattern may be mistaken for giant cell tumor of bone.
A GCT rarely occurs with Paget's disease; however, it is most commonly associated with the polyostotic form Paget's disease.[14] Therefore, any elderly patient with a giant cell tumor should be studied for the possibility of Paget’s disease. Currently various surgical treatments are available like curettage, curettage and bone grafting, radiation, amputation, resection and reconstruction. Curettage and bone grafting provides better results as compared to enblock excision in terms of functionality.[15]

To conclude, for an early diagnosis of giant cell rich lesions on cytology FNAC can be used as a diagnostic tool when it is supplemented with sufficient clinical and radiological data.

References: