*CASE REPORT*

# Giant-cell reparative granuloma of the tibia

Mehmet SUBASI, Ahmet KAPUKAYA, Huseyin BUYUKBAYRAM, Yasar BUKTE

**Giant-cell reparative granuloma (GCRG) occurs in the jaw, temporal bone, and short tubular bones of the hands and feet. Although GCRG can affect long bones, only small numbers of such cases have been sporadically reported. This report describes a giant-cell reparative granuloma in the proximal tibia in a 60-year-old woman, describes features of GCRG in long bones and reviews the literature.**

**A 60-year-old female patient was referred to us with complaints of moderately tender swelling of the right leg. Whole-body scintigraphic scanning was performed, which incidentally also disclosed a distal femoral lesion. The patient was admitted for surgery and incisional biopsies were performed on both lesions. Pathology analysis of the specimen from the tibia showed new bone lamellæ encircled by osteoblasts and multinucleated giant cells which were more numerous in the hæmorrhagic regions of the stroma ; the latter displayed fibroblasts, histiocytes and inflammatory cells. The specimen from the femoral lesion showed typical features of a benign enchondroma. The patient was readmitted for surgery. The femoral enchondroma was curetted and the cavity was packed with bone graft. The tibial GCRG was treated with marginal resection, autogenous and allogenous bone grafting and intramedullary nailing. Follow-up examination after two years showed no clinical or radiological evidence of a recurrence.**

**Although GCRG is uncommon, it should be considered whenever a lucent, expansile, and possibly destructive lesion of a long bone is encountered. It should be distinguished from true giant cell tumours occurring in the same locations because they have different biologic behaviours.**

## INTRODUCTION

Giant-cell reparative granuloma (GCRG) was first described as a non-neoplastic fibrous lesion with scattered multinucleated giant cells of the jaw bones by Jaffe in 1953 (6, 14). Two lesions involving small bones were termed "giant cell reactions" in 1962 (14). Since then, other authors have reported similar lesions occurring in the small bones of the hands and feet (1, 9, 10, 11, 13). Although GCRG can affect long bones, only small numbers of these cases have been sporadically reported (3, 5, 7, 8, 12, 14). We report one additional case that presented in the tibia.

## CASE REPORT

A 60-year-old female was referred to our clinic with a 2-month history of pain and swelling in her right leg. She gave no history of previous trauma.

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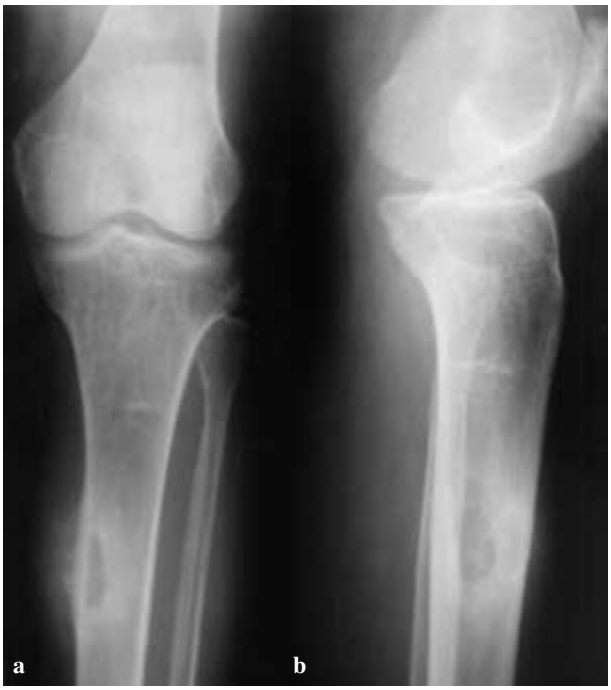


***Fig. 1a, b.*** — Anteroposterior and lateral plain radiographs showing a large lytic expanding lesion of the proximal right tibia with scattered calcification.

Examination revealed a bony mass moderately tender on palpation of the medial aspect of the proximal tibia. Range of motion, ambulation, muscle strength and sensitivity were normal. Laboratory findings including serum calcium, phosphorus, and alkaline phosphatase values were normal. Plain radiographs demonstrated a large expanding lytic lesion of the proximal right tibia with scattered calcification (fig 1). Whole-body scintigraphic scanning was performed ; it also incidentally disclosed a distal femoral lesion. Computed tomography showed cortical destruction with an intra and extra-osseous mass on the anterior aspect of the tibia. (fig 2). Magnetic resonance imaging (T1 and T2 weighted images) clearly showed that the tumour had broken through the cortex to form a soft-tissue mass. T1 and T2-weighted MR images showed low signal intensity. On the T2-weighted image, small bright signal areas were sometimes seen in the lesion, which corresponded to cysts filled with blood. After contrast administration, the hypointense solid mass lesion on the T1-weighted image showed enhancement. Most areas of the lesions

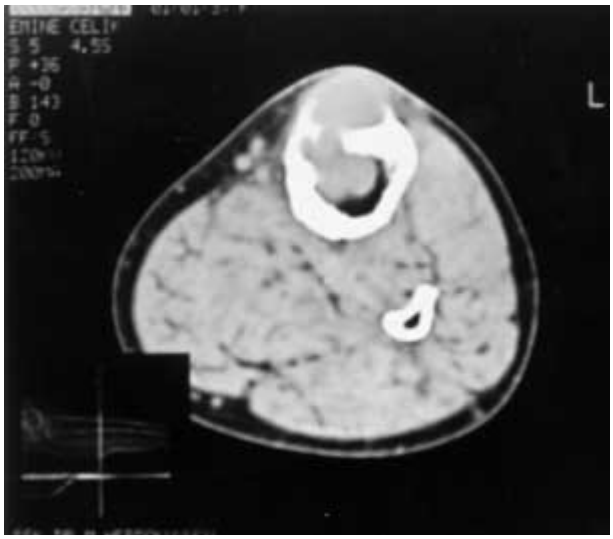


***Fig. 2.*** — Computed tomography showing cortical destruction with the intra and extra-osseous mass on the anterior aspect of the tibia.

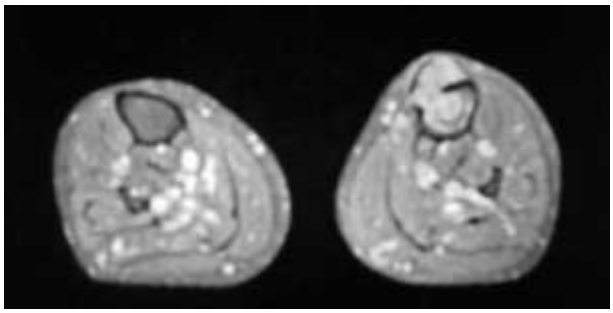

***Fig. 3.*** — On the T2-weighted image, small bright-signal areas were sometimes seen in the lesion, which corresponded to cysts filled with blood. After contrast administration, the hypointense solid mass lesion on T1-weighted image showed enhancement. Most areas of lesions became brighter focally with unstained dark areas on enhanced fat suppressed T1-weighted images.

became brighter focally with unstained dark areas on enhanced fat suppressed T1-weighted images (fig 3). The right femoral metaphyseal lesion was shown as a centrally-localised radiolucent and scalloped one. There was no sclerotic rim and no cortical destruction.

Surgical biopsy of the femoral and tibial lesions was performed and tissue specimens were obtained. Microscopic examination of the specimens revealed mononucleated chondrocytes displaying local hyalinisation with a lobular appearance in a single

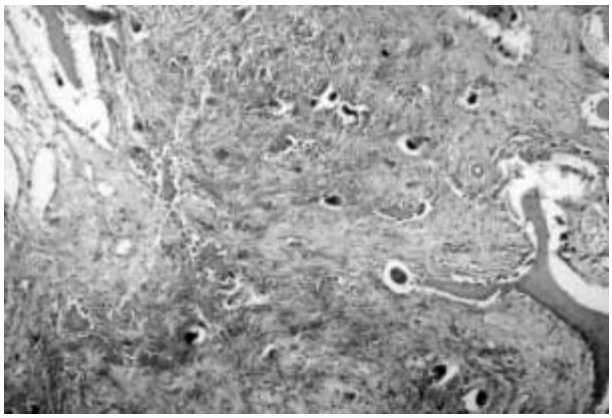

*Fig. 4.* — The microscopic characteristics of the tibial mass are lamellae of new bone encircled by osteoblasts, multinucleated giant cells denser in the hæmorrhagic regions of the stroma ; the latter consists of fibroblasts, histiocytes and inflammatory cells (H & E stain, × 200).

lacuna of the femoral mass. The specimen from the tibia featured new bone lamellæ encircled by osteoblasts and multinucleated giant cells which were more numerous in the hæmorrhagic regions of the stroma ; the latter consisted of fibroblasts, histiocytes and inflammatory cells (fig 4).

Upon receipt of the pathology report, the patient was readmitted for surgery during which the femoral enchondroma was curetted and the cavity packed with bone graft material. The GCRG was treated with marginal resection, autogenous and allogenous bone grafting and intramedullary nailing of the tibia.

Follow-up examination two years later showed no clinical or radiographic evidence of a recurrent lesion. Knee function was normal and laboratory studies were within normal limits. Roentgenograms showed consolidation of the bone grafts and a decrease in the dimensions of the tibial lesion as compared to the preoperative roentgenograms.

## DISCUSSION

The pathogenesis of GCRG, whether it occurs in the jaw, hands, feet, or long bones, remains unknown (1). The lesion has been thought to be related with trauma, repair, or faulty development. There is also some indication that CGCGs may be related pathogenetically to aneurysmal bone cysts

Table I. — Distribution of Giant Cell Reparative Granulomas in Long Bones*

| Localisation | Numbers |
|---|---|
| Femur | 5 |
| Tibia | 5 |
| Fibula | 2 |
| Humerus | 3 |
| Radius | 1 |

* Authors' case plus cases from the literature.

or to simple bone cysts, which are also known as traumatic bone cysts (2). There was no history of trauma in our patient.

Most lesions occur in the jaw, the temporal bone, or in short tubular bones of the hands and feet (4, 9, 10, 13). Although GCRG can affect long bones, only small numbers of these cases have been sporadically reported (3, 5, 7, 8, 12, 14). To our knowledge, 15 cases termed GCRG and arising in the long bones have been reported in the literature, including the femur in five cases, the tibia in four, the humerus in three, the fibula in two, and the radius in one (table I). Multiple involvements were reported in only two cases in the literature, one in the hand and the other in the foot (1, 11). While its preferred location is the metaphysis, diaphyseal involvement has been noted to a lesser degree (14). In the literature localisation of the four tibial cases was proximal metaphysis to epiphysis in two, proximal metaphysis in one, and diaphysis in one case (7, 8, 14), while diaphyseal involvement was also found in our case.

Though the second decade is suggested as the age of occurrence, the ages reported in the literature range from 3 to 76 years (1, 4, 10, 13) ; our patient was 60 years old. The gender distribution was reported by some authors as symmetrical although some others suggest a female predominance (1, 7, 10, 14).

Although GCRG is an expansile, lytic lesion, its cortical margins usually remain intact (10, 14). It is very unusual for the lesion to disrupt the cortex and extend into the soft tissues (10, 13). The lesions produce an eccentrically located expansile area of osteolysis in the metaphyseal or diaphyseal region, occasionally extending into the epiphysis in the

skeletally mature patients (14). In our patient computed tomography identified cortical destruction with an intra and extra-osseous mass on the anterior aspect of the tibia. In magnetic resonance images (T1 and T2 weighting images), the tumour had clearly broken through the cortex to form a soft-tissue mass. T1 and T2-weighted MR images showed low-signal intensity. On the T2-weighted image, small bright signal areas were sometimes seen in the lesion, which corresponded to cysts filled with blood. After contrast administration, the hypointense solid mass lesion on the T1-weighted image showed enhancement. Most areas of lesions became brighter focally with unstained dark areas on enhanced fat suppressed T1-weighted images.

It is necessary to distinguish GCRG from giant-cell tumours, aneurysmal bone cysts, and brown tumours of hyperparathyroidism (10). The characteristic histology of the GCRG includes a cellular fibrous stroma with irregularly distributed multinucleated giant cells, many of which occur in clusters associated with foci of haemorrhage. Occasionally, mononuclear inflammatory cell infiltration is present and osteoid formation is frequently found (1, 4, 10, 13). In cases such as ours, where two lesions are diagnosed in different locations, GCRG should be differentiated from the multicentric giant cell and brown tumour. A giant cell tumour usually occurs in the third or fourth decade of life. This tumour typically affects the epiphyseal region but may extend into the metaphysis. It is an eccentric, lytic and expanding lesion. Histologically, a giant cell tumour consists of a homogeneous stroma with giant cells and mononuclear cells dispersed evenly throughout the tumour. It rarely contains osteoid or new bone. This contrasts with the GCRG in which the giant and mononuclear cells predominate in the areas of haemorrhage (10). GCT has a high tendency to recurrence and, therefore, requires more aggressive treatment (10). The brown tumour of hyperparathyroidism may be radiologically and histologically indistinguishable from GCRG (9). It can be excluded by normal blood and renal profile. The overlapping clinical/histologic features and the similar biologic behaviour of GCRG and aneurysmal bone cysts represent related responses to an intraosseous haemorrhage (7). Both show a highly reparative process and spindle cell proliferation, which are associated with immature bone production. Aneurysmal bone cysts are typically composed of large, blood-filled vascular spaces. These large vascular channels are not a feature of GCRG (7, 9, 10).

Most authors agree that GCRG is a benign tumour-like condition. Though surgical curettage is usually sufficiently curative for this lesion, local recurrence rates ranging from 23 to 75% have been reported for lesions in the short tubular bones of the hands and feet (9, 13). It has been suggested that GCRG in the axial skeleton and long bones has a better biologic behaviour compared with GCRG in the short tubular bones of the hands and feet (7). For this reason GCRG can be adequately treated with curettage and bone grafting for both primary lesions as well as recurrences (10).

GCRG has a wide range of morphologic presentation. Radiologically it showed aggressive features in our patient, with bony permeation, breaking of the cortex, and soft tissue extension. These features may suggest a malignant lesion. Awareness of this lesion is important to avoid diagnostic errors and potential mismanagement.

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