Case Descriptions

Case 1
An 8-year-old boy had rapid development of exophthalmos, pain, and diplopia, with normal visual acuity. There was no history of ocular surgery or injury. CT and MR imaging revealed an expansile and lytic bone lesion with solid and cystic components (Figs 1 and 2). The solid portion of the mass showed strong enhancement with contrast material. The mass extended into the orbit and middle cranial fossa. Open biopsy suggested a GCT. Thereafter, the patient underwent surgery with complete resection of the mass and a final histologic diagnosis of GCRG. One year later, MR imaging was repeated with no evidence of recurrence.

Case 2
A 53-year-old man with a history of a recent dental procedure and a suggestion of dental abscess underwent CT examination of the neck. A CT-guided tru-cut (wide) needle biopsy of the mass showed a giant cell containing bony lesion (Fig 6). Thereafter, the patient underwent surgery involving complete resection of the mass, with the final histologic diagnosis of GCRG (Fig 7).

Discussion

GCRG is a reactive inflammatory process secondary to trauma and intraosseous hemorrhage and predominantly affects children and young women.1,8 GCRG occurs in many bones,5 with the mandible, maxilla, and the small bones of the hand and feet being the most common sites.1,6 Extragnathic GCRG has another peak of occurrence in the 50- to 80-year range, with the most common site in the craniofacial region being the temporal bone.1,6 In the English-language literature, to the best of our knowledge, GCRG of the pterygoid plate has not been reported. GCRG is characterized by a non-neoplastic fibrous stroma, with unevenly distributed multinucleated giant cells, particularly appearing around foci of hemorrhage. Reactive osteoid formation is almost always present. GCRG is frequently misdiagnosed as GCT,4,6 which is a neoplastic process.4 In GCRG, giant cells contain fewer nuclei than those of GCT.5,6
Radiologically, GCT and GCRG are indistinguishable. On CT, both usually appear as nonspecific lytic lesions. On MR imaging, most lesions show areas of low signal intensity on T1- and T2-weighted imaging, corresponding to the areas of fibrosis and/or hemosiderin. Both tumors enhance, with the degree of enhancement ranging from slight to strong. The primary difference between the 2 is the prognosis. GCT has a higher incidence of recurrence than GCRG and may undergo malignant transformation and metastasize. GCRG does not undergo malignant transformation, and metastasis has not been reported.

GCT is a histologically benign but locally aggressive bone lesion and mostly observed between the ages of 20 and 40 years and rarely seen before puberty. GCT most commonly involves the epiphyses of major long bones, occasionally the bones of the hands, feet, sacrum, and, rarely, the vertebrae above the sacrum. Less than 2% of GCTs present in the craniofacial region. Within the skull, the sphenoid and temporal bones are the most commonly affected. The infrequency of GCT in the other bones of the skull may be related to their intramembranous bone formation. Histologic cell types are osteoclast-like multinucleated giant cells, round mononuclear cells resembling monocytes, and a spindle-shaped fibroblast-like stromal cell. Osteoid or bone formation may occur.

Giant cells are found in many bone tumors, such as osteosarcoma, chondroblastoma, osteoblastoma, aneurysmal bone cyst, malignant fibrous histiocytoma, chondromyxoid fibroma, fibrous dysplasia, GCRG, eosinophilic granuloma, and the brown tumor of hyperparathyroidism. Evenly distributed abundant and uniform multinucleated giant cells, round mononuclear cells resembling monocytes, and a spindle-shaped fibroblast-like stromal cell. Osteoid or bone formation may occur.

GCT can have a clinically malignant behavior due to its aggressive growth and potential to metastasize and recur. Radiologically, GCT is seen as a radiolucent lesion without sclerotic borders. CT demonstrates cortical expansion or penetration, trabeculations, absence of matrix mineralization, and pathologic fracture. MR imaging shows cystic and solid parts and better delineates the soft-tissue extension, which occurs in more than one third of cases. GCT is usually well defined and margined by a rim of low signal intensity, either because of reactive osteosclerosis or a fibrous pseudocapsule. The signal intensity characteristics of solid tissue can be non-

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Fig 2. Axial and coronal T2-weighted (A, D), T1-weighted (B, E), and gadolinium-enhanced T1-weighted (C, F) images show a heterogeneously enhancing lesion with extension into the orbit, masticator space, and middle cranial fossa and proptosis of the right eye. Enhancement and edema are also noted within the masticator muscles and surrounding soft tissue. Nonenhancing parts of the lesion with low T2 signal intensity probably represent dense fibrosis identified on the histologic examination.

Fig 3. Noncontrast axial (A) and coronal (B) CT images show an expansile bone lesion arising from the lateral plate of the right pterygoid process.

Fig 4. Contrast-enhanced axial CT scan obtained 3 months after Fig 3 shows interval rapid enlargement of the lesion with enhancing soft-tissue component and fluid-fluid levels containing cystic parts.
specific, but most GCTs reveal moderate-to-marked hypointensity on T1- and T2-weighted images, which is attributed to hemosiderin deposits and/or high collagen content. This feature is useful to exclude chondroid tumors, which have high signal intensity on T2-weighted images. Cystic parts are the result of hemorrhage or formation of a secondary aneurysmal bone cyst.7

GCRG must be also differentiated from the brown tumor, aneurysmal bone cyst, chondroblastoma, fibrous dysplasia, synovial sarcoma, and osteosarcoma.5

Rapid growth, fluid-fluid levels, and an enhancing solid part are suggestive of GCT, GCRG, secondary aneurysmal bone cyst, or telangiectatic osteosarcoma.11 Fluid-fluid levels can be seen in other bony lesions, such as fibrous dysplasia, solitary bone cyst, chondroblastoma, osteosarcoma, osteomyelitis, osteoblastoma, malignant fibrous histiocytoma, synovial sarcomas, and bone metastases, which can be differentiated from GCT and GCRG with relative ease.12,13 Aneurysmal bone cyst may mimic GCT and GCRG more closely than the others mentioned previously, but it is rarely seen at the skull base.7,11,14 Aneurysmal bone cyst can be a primary lesion or associated with or preceded by other bone lesions, such as GCT, fibrous dysplasia, osteoblastoma, chondroblastoma, unicameral bone cyst, nonossifying fibroma, and fibromyxoma. Presence of an enhancing soft-tissue component without matrix calcification makes the diagnosis of GCT and GCRG more likely.5,7,14

GCRG is more likely than GCT when benign giant cell lesions occur in locations other than the ends of long tubular bones and in skeletally immature patients.4 Accordingly, the most plausible diagnoses in the first case, even though there was no history of injury, were GCRG and secondary aneurysmal bone cyst, and in the second case, GCRG and GCT with or without aneurysmal bone cyst. Of all the lesions of the sphenoid bone, metastases are the most common.11

References