To the Editor: Giant cell reparative granuloma (GCRG) is a nonneoplastic, benign lesion derived from bone tissue. The morbidity of GCRG is relatively low, comprising approximately 7% of benign bone lesions. GCRGs are mostly found in adolescents, and no apparent gender inclination has been identified. In addition, GCRGs are predominantly found in the mandible and maxilla; a few cases have occurred in the short bones as the hands and feet. Occasionally, GCRGs are detected in long bones and vertebral; temporal GCRGs are rarely reported. GCRGs associated with intracranial invasion are rare.\(^{(1)}\) Although some cases of GCRG have been previously reported, characteristic and elaborate descriptions have not yet been clearly established for imaging diagnosis and differential diagnosis. This article reported one case of left temporal GCRG associated with cholesteatoma caused by otitis media mastoiditis and another case of temporal giant cell tumor (GCT).

Case 1: A 39-year-old man was admitted due to an intermittent headache with left-side hearing impairment for 5 months and an intracranial space-occupying lesion that was found 4 months prior. Results of neurology examinations were as follows: the patient was fully conscious and capable of answering questions; he exhibited a positive light reflex, normal eye movement, and no blurry vision or visual defect; he showed no facial drooping or numbness; bilateral frontal lines and nasolabial folds were symmetrical; his tongue was centrally positioned and he showed no stiffness around the neck; he demonstrated Level V muscle strength of the limbs and normal muscle tension; and he reported no abnormal feeling of tenderness on the limbs and no previous history of traumatic surgery.

A left external auditory canal mass, bulging front wall of external auditory canal, and invisible tympanic membrane were demonstrated by ear-nose-throat endoscopy. Pure-tone hearing tests detected a conductive hearing loss in the left ear. For radiological examinations, computed tomography (CT) scans showed a suborbicular lesion with heterogeneously hyperdense signal at the top of the zygomatic arch; some bony invasion was found in the squamous part of temporal bone. The size of the bone defect was approximately 2 cm × 2 cm. The bone margin was ill defined and serration shaped. The lesion was superficially located outside the dura mater, as observed by endoscopy. After satisfactory separation of the lesion and dura mater under endoscopy, complete resection of the lesion was performed. The dura remained intact. GCT was detected outside the epidural space with well-defined borders. Stripe-like cerebrospinal fluid signals were found around the edges of the lesion. The lesion was isointense and slightly hypointense on T1-weighted imaging (T1WI); an apparent hypointense area was found in the lesion, due to neogenesis ossification. On T2WI, the signal was heterogeneously hypointense [Figure 1c]. On enhanced scan, the lesion was heterogeneously enhanced, while the internal ossification component was not. However, adjacent dura mater was enhanced [Figure 1d and 1e]. It was suspected as meningioma on preoperative radiological diagnosis.

Surgical treatment was performed through the inferior part of the left temporal bone. Pathological tissue was detected at the top of the zygomatic arch; some bony invasion was found in the squamous part of temporal bone. The size of the bone defect was 2 cm × 2 cm. The bone margin was ill defined and serration shaped. The lesion was superficially located outside the dura mater, as observed by endoscopy. After satisfactory separation of the lesion and dura mater under endoscopy, complete resection of the lesion was performed. The dura remained intact. GCT was detected outside the epidural space with well-defined borders. Stripe-like cerebrospinal fluid signals were found around the edges of the lesion. The lesion was isointense and slightly hypointense on T1-weighted imaging (T1WI); an apparent hypointense area was found in the lesion, due to neogenesis ossification. On T2WI, the signal was heterogeneously hypointense [Figure 1c]. On enhanced scan, the lesion was heterogeneously enhanced, while the internal ossification component was not. However, adjacent dura mater was enhanced [Figure 1d and 1e]. It was suspected as meningioma on preoperative radiological diagnosis.

According to the pathological reports, lesions were purple-red and exhibited a soft texture with a gritty sense. The internal part of the lesion presented as grayish-yellow with poor blood supply; it exhibited a solid texture associated with calcification. A large amount of osteoclast-like multinucleated giant cells could be observed under the microscope. Immune spindle-shaped fibroblast cells, apparent fibrosis, distinct and nonuniformly distributed less nucleated giant cells, and potential inflammatory mononuclear cells with osteoid or bone formation were also identified by microscopy. Immunohistochemistry reported no detection of p63 protein; the skull, associating with soft-tissue swelling of the left temporal scalp [Figure 1b]. On magnetic resonance imaging, the lesion was detected outside the epidural space with well-defined borders. Stripe-like cerebrospinal fluid signals were found around the edges of the lesion. The lesion was isointense and slightly hypointense on T1-weighted imaging (T1WI); an apparent hypointense area was found in the lesion, due to neogenesis ossification. On T2WI, the signal was heterogeneously hypointense [Figure 1c]. On enhanced scan, the lesion was heterogeneously enhanced, while the internal ossification component was not. However, adjacent dura mater was enhanced [Figure 1d and 1e]. It was suspected as meningioma on preoperative radiological diagnosis.

Surgical treatment was performed through the inferior part of the left temporal bone. Pathological tissue was detected at the top of the zygomatic arch; some bony invasion was found in the squamous part of temporal bone. The size of the bone defect was approximately 2 cm × 2 cm. The bone margin was ill defined and serration shaped. The lesion was superficially located outside the dura mater, as observed by endoscopy. After satisfactory separation of the lesion and dura mater under endoscopy, complete resection of the lesion was performed. The dura remained intact. GCT was detected outside the epidural space with well-defined borders. Stripe-like cerebrospinal fluid signals were found around the edges of the lesion. The lesion was isointense and slightly hypointense on T1-weighted imaging (T1WI); an apparent hypointense area was found in the lesion, due to neogenesis ossification. On T2WI, the signal was heterogeneously hypointense [Figure 1c]. On enhanced scan, the lesion was heterogeneously enhanced, while the internal ossification component was not. However, adjacent dura mater was enhanced [Figure 1d and 1e]. It was suspected as meningioma on preoperative radiological diagnosis.

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heterogeneously distributed internal density. In CT, striped‑like bone was recognized as an epidural hyperdense mass in the brain with lesion was parallel to the anterior part of the temporal mastoid. It closely related to the left temporal mastoid; the radial line of the From analysis of the patient’s imaging appearance, the lesion was likely cause of intracranial GCRG in this case, particularly because temporal bone was involved; local destructive growth of bone, skull base, temporal bone, craniofacial bone, short bones of the hand and feet, and long bones of the extremities. The etiology of GCRG has remained unclear; intraosseous bleeding, which is generally secondary to trauma or inflammation, is suspected to initiate GCRG development.[2]

Clinical symptoms of temporal GCRG include hearing loss, tinnitus, apparent mass, pain, dizziness, and facial weakness. Conductive hearing loss is the most common associated symptom. The first symptom of the patient in this case was hearing loss; thus, the patient presented to the otolaryngology department. Endoscopic examination confirmed mass formation and inflammation in the external auditory canal. Imaging examination revealed an intracranial space‑occupying lesion and further evidence of otitis media. To the best of our knowledge, cholesteatoma is the likely cause of intracranial GCRG in this case, particularly because temporal bone was involved; local destructive growth of cholesteatoma may have led to reactive GCRG growth.[3]

From analysis of the patient’s imaging appearance, the lesion was closely related to the left temporal mastoid; the radial line of the lesion was parallel to the anterior part of the temporal mastoid. It was recognized as an epidural hyperdense mass in the brain with heterogeneously distributed internal density. In CT, striped‑like bone density and grit‑like dispersal bone density shadow were observed in the lesion; in addition, the lesion was hypointense in T2WI. Combined with pathological findings, considerations should be new bone, rather than hemorrhagic foci or hemosiderin deposition, both of which were reported by previous literature. Lesion features mainly comprised expansive and osteolytic destruction. Dura mater was intact at the medial margin of the lesion; thickening of the dura mater could be observed with calcification, which indicated extensive progress. In addition, there were signs of repair related to inflammatory infiltration. At the lateral margin of the lesion, local temporal bone was completely destroyed. The outer cortical plate of temporal bone was compressed and tilted externally, indicating that the lesion grew in an intracranial to extracranial direction. Moreover, the anterior part of the left temporal mastoid bone was also destroyed, while soft‑tissue density shadow appeared at the left tympanic cavity, mastoid, and external auditory canal. These findings suggested the existence of otitis media, accompanied by the formation of mastoiditis cholesteatoma.

Treatments for GCRG include complete surgical resection, corticosteroid injection, calcitonin therapy, and radiation therapy. Complete radical resection is the preferred method for treatment of GCRG. The recurrence rate after simple curettage was 50%; however, the recurrence rate after complete radical resection was 10%.[4] Close observation and regular follow‑up after surgery are necessary. For patients in whom radical resection cannot be applied because of the involvement of complex structures in the base of the skull, postoperative radiotherapy should be supplemented.

Differential diagnosis between GCRG and GCT is quite challenging. In previously reported cases, the diagnosis of GCRG was not based on a specific imaging feature. Therefore, diagnosis
was generally established on the basis of the age of onset, clinical symptoms, histological analysis, and response to treatment. In this article, we have introduced another case of GCT with identical pathological and radiographic appearance to the GCRG in the previous case. We focused on the analysis and comparison of the imaging appearance between these two cases, in order to determine features that could be potentially used for differential diagnosis.

Case 2: A 55-year-old female patient was admitted to the hospital due to intermittent dizziness that had occurred for more than 1 year without nausea or vomiting. The symptom of dizziness appeared irregularly with varied duration and resolved spontaneously. In the operation, the lesion was purple-red, temporal bone was destroyed with the erosion of the inner cortical plate, and the outer cortical plate was focally destroyed. The diploic vein was significantly dilated with a rich blood supply. Massive bleeding appeared during the operation. The texture of the lesion was relatively hard with the invasion of the petrous part of temporal bone but without invasion of dura mater. Pathological results indicated a low-grade malignant bone GCT.

Based on the imaging appearance, GCRG and GCT are difficult to distinguish; however, they have several differences. GCT originates from the connective tissue of the bone marrow. The growth center of the tumor was located on the median axis of the diploe bone, and it exhibited distensibility and osteolytic changes [Figure 1f]. Both inner and outer cortical plates were involved, with partially marginal sclerosis and eggshell-like changes; cortical bone had begun to invade adjacent soft tissues [Figure 1g]. GCRG originates from the connective tissue of periosteum. The lesion was connected with the corresponding inner cortical plate on a wide basement. The inner cortical plate was extensively destroyed, while the outer cortical plate was compressed; bone structure was partially or completely destroyed. Approximately 75% of GCRGs show osteoid or new bone formation, but the density of lesions is nonuniform. On T2WI, GCRG demonstrated a typical characteristic hypodensity, which was rarely seen in GCT [Figure 1h]. In GCRG, septations could be seen within the lesion, as a large amount of hyperplastic collagen fibers separated the granulomas. Thus, on enhanced scan, separations were identified on GCRG, while GCT was more homogenously enhanced than GCRG [Figure 1i and 1j].

Other points for distinguishing between GCRG and GCT include: 1) onset age: GCRG generally occurs between 10 and 20 years of age, while GCT occurs frequently in patients between 20 and 40 years of age and rarely below the age of 20 years; 2) occurrence site: mandible and maxilla are common sites for GCRG, which is rarely observed on other bone. The end of the long bone is a common site for GCT, but GCT of the skull can be found occasionally; and 3) the crucial point of differentiation between GCRG and GCT is the response to treatment, which drives overall prognosis. After radical resection of GCRG, the recurrence rate was relatively low without malignant metastasis. Conversely, the recurrence rate of GCT was 30–50%, with a malignancy rate of 5–10%. Malignant transformation and metastatic spread could also occur. In addition, based on immunohistochemical detection, p63 was identified as an effective method of diagnosing GCT of bone and has gradually been recognized by scholars worldwide. As the positive result from immunohistochemical detection in p63 for GCT, while GCRG remained negative.

In summary, GCRG is rarely found in temporal bone. Differential diagnosis with GCT can be problematic. However, these lesions can be identified through their sites of origination, their signaling characteristics, postenhancement appearance, and involvement of the mastoid, all of which enable distinction for the differential diagnosis.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identities, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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