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ORIGINAL RESEARCH

Prevalence and Appearance of the Posterior Wall Defects of the Temporal Bone Caused by Presumed Arachnoid Granulations and Their Clinical Significance: CT Findings

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BACKGROUND AND PURPOSE: Arachnoid granulations (AGs) of the posterior wall of the temporal bone are a rare cause of spontaneous CSF leakage. The purpose of this study was to investigate the prevalence and appearance of presumed AGs, evidenced by the posterior wall defects of the temporal bone with lobulated or scalloped margins on CT scans.

MATERIALS AND METHODS: We retrospectively reviewed CT scans of the temporal bone obtained in 1255 patients (573 men and 682 women; mean age, 42 years). We evaluated the prevalence, multiplicity, size, and location of presumed AGs in the posterior wall of the temporal bone. Preservation of the posterior wall of the mastoid air cells was also investigated and correlated with clinical features.

RESULTS: Thirty patients (2.4%), including 13 men and 17 women, aged 27–88 years (mean, 55 years), showed a total of 40 presumed AGs on CT scans. Their prevalence tended to increase with age. They were most commonly located at the lateral third of the temporal bone at a level of or above the common crus. Although 15 lesions in 11 patients also caused focal loss of the posterior wall of the mastoid air cells, symptomatic CSF leakage was found in only 2 patients, in both of whom the adjacent mastoid air cells were also opacified on CT scans.

CONCLUSION: Although rare, radiologists need to be familiar with the posterior wall defects of the temporal bone caused by presumed AGs on CT scans, because they might cause CSF leakage.

Arachnoid granulations (AGs), also known as Pacchionian bodies, are pseudopodial projections of the pia-arachnoid, which normally extend through the dura into the venous sinuses or venous lacunae.[1] They are reported to be located most commonly beneath the superior sagittal sinus and the adjacent lateral lacunae, followed by the transverse sinus, cavernous sinus, superior petrosal sinus, and straight sinus.[2] For unknown reasons, however, AGs can occur at unusual locations where they do not communicate with the venous sinuses. At these sites, AGs can cause spontaneous CSF leakage by cortical bone erosion, resulting from the pressure of the CSF.[1,3-8]

In adults, spontaneous CSF otorrhea can occur when there is communication of the subarachnoid space with the mastoid air cells, which results most commonly from erosion of the tegmen tympani by AGs situated in the middle cranial base[4,5,9] and less frequently from erosion of the posterior wall of the temporal bone by AGs in the posterior cranial fossa. This can cause serous otitis media or septic meningitis secondary to acute otitis media and is best displayed on CT scans. Although there are no clinical signs of CSF leakage or meningitis, it would be important for the radiologist to report to the otolaryngologist the presence of the temporal bone defects on CT scans, because these bone defects may enlarge, as AGs are known to enlarge with age.[10,11]

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Although the tegmen tympani is known to be the most common site of spontaneous CSF otorrhea in adults, it is hard to evaluate the tegmen defects on CT scans because they are frequently too small to be seen on CT scans and also because many of them actually represent natural bony dehiscences unrelated to AGs. Unlike the thin bony plate of the tegmen tympani, the posterior wall of the temporal bone is thick and devoid of natural dehiscences. Even small AGs can create recognizable pitholes,[5] which can easily be demonstrated on CT scans. A considerable number of studies have been reported on the CT and MR imaging findings of AGs located in the cerebral venous sinuses.[2,12-16] To our knowledge, however, there have been only a few case reports on the CT features of the AGs of the posterior wall of the temporal bone.[5-9] The purposes of this study were to investigate the prevalence and appearance of presumed AGs, evidenced by the posterior wall defects of the temporal bone with lobulated or scalloped margins on CT scans, and also to correlate these CT findings with the presence of clinical features, such as CSF otorrhea or meningitis.

Materials and Methods

Patients

This study was approved by the institutional review board of our hospital. Between July 2004 and June 2005, a total of 1411 consecutive patients underwent temporal bone CT scans at our institution for evaluation of various otologic or neurologic problems. Of these 1411 patients, 151 patients with a previous operation on the temporal bone and 5 patients with severe bone destruction caused by cholesteatoma or malignant tumor were excluded from the study. Consequently, we reviewed 1255 temporal bone CT scans obtained in 1255 patients who formed the basis of this study. They included 573 men and 682

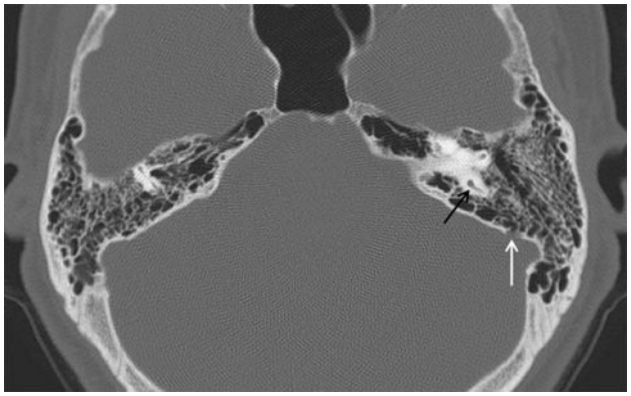

Fig 1. Subject 24. Axial CT scan at the level of the common crus (*black arrow*) shows a small bone defect caused by presumed AG (*white arrow*), located at the lateral third of the posterior wall of left temporal bone. Although a focal loss of the posterior wall of the mastoid air cells is also present, there is no evidence of tympanomastoid opacification. This 66-year-old woman presented with hemifacial spasm, and there was no clinical feature suggesting CSF leakage.

women ranging in age from 11 days to 89 years with the mean age of 42 years.

CT Examination

CT studies of the temporal bone performed in the axial plane with various models of single or multidetector helical CT scanners (GE Healthcare, Milwaukee, Wis) were available for review. In most of the patients, CT scans were obtained with a conventional, nonhelical mode with the parameters of 120 kVp, 280–400 mAs, 1.00–1.25-mm section thickness and table increment, 18-cm FOV, and a high-resolution algorithm. In some cases performed on multidetector helical CT, the axial images were obtained with 0.625-mm beam collimation and displayed with 1.25-mm section thickness. All of the images were viewed with the window width of 4000 HU and window level of 400 HU.

Image Analysis

All of the CT scans were independently reviewed by a dedicated head and neck neuroradiologist and a general neuroradiologist, who have been practicing in the field for 18 years and 11 years, respectively. Disagreements were solved by consensus. AGs were determined to be presumably present if axial CT scans demonstrated cortical defects of the posterior wall of the temporal bone with lobulated or scalloped margins (Fig 1). We evaluated the prevalence, multiplicity and bilaterality, size, and location of defects in the posterior wall of the temporal bone. In addition to the overall prevalence, we determined the prevalence of bone defects according to the age of the patients grouped into every decade. Statistical analysis for estimating the trend of prevalence of presumed AGs according to increasing ages of the patients was performed with the linear-by-linear association test. We also compared the prevalences of presumed AGs between men and women by using the χ^2 test. All of the statistical analyses were performed with SPSS (Version 12; SPSS, Chicago, Ill) with a P value of $<.05$ considered statistically significant. The size of bone defects was measured at both the greatest horizontal diameter (width) of the area of cortical bone defect and the greatest diameter perpendicular to it (depth). As for the transverse location, we evenly divided the posterior wall of the temporal bone from the petrous apex to the squamous temporal bone into 3 parts: medial, middle, and lateral. We also determined the longitudinal location in reference to the common crus and divided them into high AGs, which were located at the level of or above the common crus, and low AGs, which were located below it. Preservation of the posterior wall of the mastoid air cells contiguous to presumed AGs was also investigated and correlated with clinical symptoms and signs.

Prevalence of presumed AGs according to age of subjects

Age	Total No. of Subjects (n = 1255)	No. of Subjects Showing Presumed AGs on CT Scans (n = 30), n (%)
<10 y	138	0 (0)
<20 y	82	0 (0)
<30 y	104	1 (0.1)
<40 y	167	2 (1.2)
<50 y	283	6 (2.1)
<60 y	271	13 (4.8)
<70 y	159	4 (2.5)
<80 y	45	3 (6.7)
<90 y	6	1 (16.7)

Note:—AG indicates arachnoid granulation.

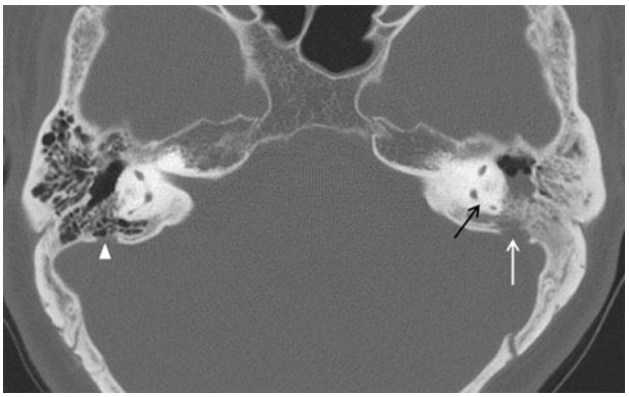

Fig 2. Subject 4. Axial CT scan at the level of the common crus (*black arrow*) shows a large bone defect caused by presumed AG (*white arrow*), located at the lateral third of the posterior wall of the left temporal bone. There also is a loss of the posterior wall of the mastoid air cells, which are opacified with fluid. This 41-year-old man presented with CSF otorrhea and recurrent septic meningitis. He underwent dural repair via intact canal wall mastoidectomy. Also noted is a small bone defect caused by presumed AG (*arrowhead*), located at the lateral third of the posterior wall of right temporal bone.

Results

The prevalences of presumed AGs demonstrated on CT scans according to age of the patients are summarized in the Table. Overall, CT scans showed presumed AGs in 30 (2.4%) of 1255 patients, including 13 men and 17 women, with their ages ranging from 27 to 88 years (mean, 55 years). There was a tendency of bone defects to be more prevalent as the patients' ages increased ($P < .001$ by linear-by-linear association test). There was no significant statistical difference in the prevalence between men and women ($P = .465$).

The on-line Table summarizes the CT characteristics of each of the presumed AGs seen in 30 patients. Twenty-two (73.3%) had a single defect of the posterior wall of the temporal bone, and 8 (26.7%) had 2 ($n = 6$) or 3 ($n = 2$) defects, resulting in a total of 40 bone defects (16 on the right and 24 on the left) demonstrated on CT scans (Figs 1 and 2). Unilateral presentation was found in 26 patients (86.7%; 11 on the right and 15 on the left) and bilateral presentation in 4 patients (13.3%). The size of the 40 bone defects ranged from 1.9 to 13.5 mm in width (mean, 6.6 mm) and from 1.1 to 5.0 mm in

depth (mean, 2.3 mm). The bone defects were most commonly located at the lateral third of the posterior wall of the temporal bone: 33 (82.5%) were located at the lateral third; 7 (17.5%) at the middle third; and none at the medial third. In reference to the common crus, 31 (77.5%) of 40 bone defects were located high, 7 (17.5%) were located low, and the remaining 2 (5%) spanned both high and low positions. All of the presumed AGs but 1 were located at the pneumatized portion of the mastoid air cells. In 15 presumed AGs (37.5%) seen in 11 patients, the posterior wall of the mastoid air cells contiguous to presumed AGs was found to be lost. Review of the medical records revealed that 2 of these 11 patients had clinical signs of CSF otorrhea with or without recurrent meningitis. In both patients, CT scans demonstrated opacification of the mastoid air cells contiguous to presumed AGs (Fig 2). In contrast, the mastoid air cells were clear in the remaining 9 patients without clinical signs of CSF leakage.

Discussion

Although CSF otorrhea is usually secondary to surgery, trauma, tumor, or infections, it can also occur spontaneously without an obvious antecedent pathology and is frequently overlooked because it may be subtle and intermittent.[5,9] Early diagnosis and surgical intervention are very important to prevent the serious complications associated with bacterial meningitis. Spontaneous CSF otorrhea is more common in infants and children and may result from various congenital anomalies in which CSF leaks into the middle ear via a preformed bony pathway through or around the bony labyrinth, as seen in Mondini anomaly, patent cochlear aqueduct, patent Hyrtl fissure, patent petromastoid canal, and patulous facial canal.[5,9,17] Occasionally, spontaneous CSF otorrhea may affect adults and is associated with a defect in the bone separating the middle ear and the intracranial cavity, where AGs have frequently been implicated as a causative factor for creating the craniodural defects.[3-5]

AGs are herniations of arachnoid tissue through a dural defect into the lumen of a dural venous sinus. The central core of an AG is composed of stellate arachnoid cells with intervening tubular spaces and is filled with CSF and covered by a herniated layer of arachnoid cells loosely attached to a fibrous capsule derived from dura and connective tissue.[4,18] Because CSF pressure is higher than venous pressure, CSF from the subarachnoid space drains into the intracranial venous system through gaps between endothelial cells covering the AG and by pinocytosis through this cell layer.[19]

Although most AGs are found at or near the cerebral venous sinuses or cortical veins, a variable number of AGs do not find a venous termination in development. Instead, after penetrating dura mater, they come to lie against the bony surface of the skull, where they may erode bone over a long period of time.[5,20] Erosion of bone is not clinically significant unless it is located near pneumatized parts of the skull, such as the paranasal sinuses or the middle ear and mastoid air cells, where it can lead to CSF leakage.[5] The common locations for these unusual AGs include the roof of the sphenoid and ethmoid sinus cells lateral to the cribriform plate in the anterior cranial fossa and along the floor of the middle cranial fossa from the tegmen tympani to the lateral surface of the sella turcica. Less frequently, AGs may be found in the posterior cranial fossa near the posterior wall of the temporal bone between the sigmoid sinus and the bony labyrinth and in the region of the jugular foramen.[4,5] Unlike the thin bony plate of the tegmen tympani where bone defects have been reported to be found in 21%–22%,[9,21] the posterior wall of the temporal bone is thick and devoid of natural dehiscences. Even small AGs can create recognizable pitholes.[5]

In our study, 30 (2.4%) of 1255 patients showed a total of 40 presumed AGs (single in 22 patients; multiple in 8 patients), evidenced by defects of the posterior wall of the temporal bone on CT scans. There has been only 1 previous study on the prevalence and appearances of AGs in the posterior fossa mastoid plate. In the study using human cadavers, Gacek[4] reported an 8.5% prevalence (16 of 188) of AGs found in that area, the rate of which is much higher than ours. Apart from the inherent difference in the study design (ex vivo versus in vivo), it could be stated regarding the discrepancy of the 2 studies that AGs were differently defined based on the ability of techniques used. Although CT examinations, as in our study, could demonstrate only presumed AGs with bone erosion, pathologic examinations, as in Gacek's study,[4] could reveal the small AGs without bone erosion. Gacek's series[4] included 7 specimens that were not associated with bone erosion. If these 7 specimens are excluded, the prevalence of AGs falls to 4.8% (9 of 188), which is double the prevalence of our study. Different age distribution might also have exerted influence on the different results between the 2 studies, because AGs are known to enlarge with age. The age of the patients showing AGs was somewhat older in Gacek's series[4] (mean, 74.6 years; range, 52–92 years) than in ours (mean, 55 years; range, 27–88 years). As might be expected, our study showed that bone defects caused by presumed AGs tended to be more prevalent as the patients became older. In regard to sex, Gacek reported an 11:5 female preponderance.[4] Although our results also showed a 17:13 female predilection, the difference between the sexes was not statistically significant. In our study, presumed AGs were more commonly encountered on the left. This was contrary to the general premise that AGs would be more frequent on the right side, where the cerebral venous sinus flow dominates.

The predilection sites of AGs in the posterior wall of the temporal bone have not been reported before. In this study, bone defects caused by presumed AGs were most commonly found at the lateral third of the posterior wall of the temporal bone at the level of or above the common crus. Variations of the location of AGs in the posterior wall of the temporal bone may represent varied aborted attempts of AGs to reach and invade the sigmoid sinus to resorb CSF. Early encapsulation by ossifying mesenchyme could be a factor in the isolation of AGs within bone defects.[4,5]

The presence of the clinical signs of CSF otorrhea has been reported to be related to the size of AGs. Gacek[4] divided AGs into 3 groups according to size and reported that, while small, AGs were not associated with bone erosion and of no clinical significance, and most intermediate and large AGs were associated with bone erosion and clinical signs that indicated communication with the mastoid air cells. In our study, 11 patients had 15 (37.5%) presumed AGs that caused loss of the posterior wall of the mastoid air cells, allowing the presumed AGs to apparently communicate with the mastoid air cells on CT

scans. However, clinical signs related to CSF leakage were present in only 2 patients. We speculate that, in addition to the size of AGs, the degree of mastoid pneumatization is an important factor that may provoke clinical problems, because even a very small AG can easily break the posterior wall of the mastoid air cells in cases where well-developed mastoid pneumatization results in a very thin posterior wall of the temporal bone. We also think that coexistent opacification of the mastoid air cells contiguous to presumed AGs may be a reliable CT finding suggestive of symptomatic CSF leakage, as seen in both of those 2 patients in our series. Symptomatic CSF leakage caused by AGs in the posterior wall of the temporal bone is best treated by an intact canal wall mastoidectomy, followed by the repair of the dural defect with a free adipose tissue or other tissue graft.[5]

There is a serious drawback to our study. Except for high-resolution CT scans of the temporal bone, we provided neither pathologic proof that what we claim to be AG is indeed that pathology nor additional support for these claims by using other imaging studies, such as low attenuation on CT scans or fluid signal intensity on MR images. Aside from AGs, various factors have been postulated as the pathogenesis of creation of osteodural defects in various locations related to spontaneous CSF leaks, such as elevated intracranial pressure, empty sella, and congenital cause.[22] This is why we selected the term "presumed AGs" instead of "AGs." To validate our results, further comparative study by using high-resolution CT and MR imaging of the temporal bone is highly warranted.

Conclusions

The prevalence of bone defects of the posterior wall of the temporal bone caused by presumed AGs on CT scans was 2.4% in our study, and there was a tendency of these defects to be more prevalent with age. Although rare, radiologists need to be familiar with this CT feature, especially when there is opacification of the adjacent mastoid air cells confluent with presumed AGs, because it might indicate underlying CSF leak.

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