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Arachnoid granulations and spontaneous cerebrospinal fluid otorrhea: Role of imaging

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Arachnoid granulations (AGs) are villous structures that are formed during the development of the cerebrospinal fluid (CSF) system. Their function is to drain the CSF into the dural venous sinuses. A variable number of AGs do not reach a dural venous lumen and are aberrantly embedded in the skull bones, primarily in the tegmen tympani, tegmen mastoidea, petromastoid plate, occipital squama, cribriform plate, and greater wing of the sphenoid bone. It has been shown that when these AGs are adjacent to pneumatized parts of the skull, they are lesions responsible for spontaneous otorrhea or rhinorrhea in adults. This article reviews the role of computed tomography and magnetic resonance imaging in the evaluation of patients with CSF otorrhea caused by AGs.

Introduction

Cerebrospinal fluid (CSF) otorrhea is most often related to direct or indirect (postsurgical) trauma to the temporal bone.[1-8] Spontaneous CSF otorrhea is far less common than traumatic CSF leak and may be seen in both adult and pediatric age groups. The clinical evidence of traumatic CSF otorrhea is usually obvious and the precise location can often be identified on computed tomography (CT) and magnetic resonance imaging (MRI).[5-7] However, the clinical evidence of spontaneous CSF leak and its exact location is frequently obscure, resulting in a delay in diagnosis and treatment, and at times results in repeated episodes of meningitis.[1-4] In the pediatric age group, spontaneous CSF otorrhea may be a manifestation of congenital inner ear malformation[6,7] (Figure 1) (see article by A. Sepahdari in this issue of the journal) or a congenital defect of the bone and dura, allowing brain herniation or the CSF to flow in the mastoid air cells and middle ear cleft (Figures 2 and 3).[5-7] Adult-onset spontaneous CSF otorrhea may be because of focal or diffuse dural dysplasia and undetected dural and bone defect of the tegmen tympani or tegmen mastoidea, associated with meningocele or meningoencephalocele (Figure 3). At times, dural dysplasia or ectasia may be extensive, resulting in CSF space following the facial canal along its labyrinthine segment, tympanic segment and into the anterior epitympanic recess. (Figure 4).

However, spontaneous CSF otorrhea in adults is most commonly attributed to arachnoid granulation (AG) as the primary responsible lesion.[1-4] These patients usually present clinically with a history of blockage of the ear, hearing loss (conductive) with a serous otitis media, or meningitis following acute otitis media.[8]

At times, otologic examination of middle ear effusion is treated with decongestants and antibiotics, followed by myringotomy. When the ventilating tube is expelled, and the larger-gauge ventilating tube is inserted, a gush of clear watery fluid prompts the possibility of CSF otorrhea,[1-4] confirmed by the analysis of beta-2 transferrin protein. A diagnostic imaging study, which should start with high-resolution axial and coronal CT study, would frequently identify the potential site of CSF leak, which is often related to aggressive AG (Figures 5 and 6).[1-4]

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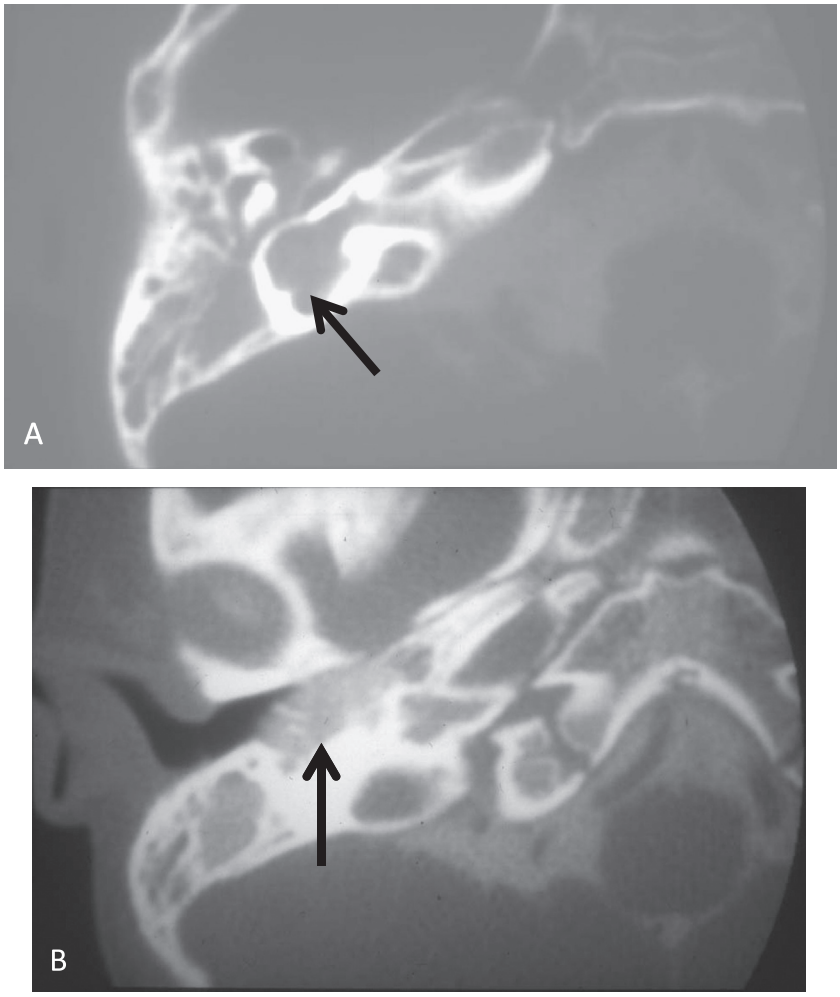

Figure 1 Mondini anomaly. CT positive contrast cisternography showing contrast in the dilated vestibule (arrow in A) and in the middle ear (arrow in B).

Arachnoid villi are formed during the development of the subarachnoid space as the primary method of CSF filtration into the dural sinuses.[1-4] Arachnoid villi are minute projections of the pia and arachnoid mater. When they become larger, forming a spongelike arrangement of channels lined by arachnoid cell processes, they are referred to as AGs. AGs normally protrude through a dural opening into the venous sinuses or venous lacunae.[1-4] Although most AGs are found at or near dural venous sinuses, a variable number of AGs in their development do not reach the dural venous sinuses. After penetrating the dura mater these aberrant "modified" AGs will come to be against the bony surface of the skull, where they may erode bone over a long period.[1-4] When these AGs are embedded adjacent to pneumatized parts of the skull, they are often responsible for spontaneous CSF otorrhea or CSF rhinorrhea.[1,2]

Imaging techniques for CSF leak

High-resolution thin-collimation (0.5, 0.625-1 mm) CT is extremely helpful in the evaluation of patients with a suspected CSF leak. The entire base of the skull should be carefully examined for bone defect and focal or diffuse bony dehiscences (Figure 5). Giant AGs can be appreciated on nonenhanced CT scan as an indentation in the dural sinuses or as lytic area with a lobulated appearance or irregular border on the temporal bone or other skull bones, with attenuation values resembling that of the CSF or brain parenchyma (Figure 7). In some instances, AGs can have associated calcifications, which can be easily resolved on CT.[9] On enhanced CT scans, AGs appear as a smooth, round filling defect within the lumen of venous sinuses (Figure 8A). AGs in venous sinuses and those embedded within the bone do not show significant contrast enhancement. We prefer not to use contrast for CT scan and instead recommend obtaining MRI including contrast enhancement (Figure 8B). Enhancement on MRI, if significant, is not in keeping with AGs. Enhancement similar to the normal brain should be used in favor of meningoencephalocele (Figure 8).

MRI combined with unenhanced high resolution CT is the most valuable imaging modality in the management of patients with CSF leak and symptomatic giant AGs.[1-14] MRI with and without contrast, in combination with magnetic resonance (MR) venography if necessary, can characterize AGs and differentiate other simulating pathologies, such as meningoencephalocele, small endolymphatic sac tumor, and