CASE STUDY

Arachnoid Granulations as Cause of Tegmen Tympani Defects

Granulaciones aracnoideas como causa de perforación del tegmen timpani

Javier Pereda-Rodríguez,1 Javier González-Llorente, Laia Pérez-Tapia, David Vicente-Mérida

Departamento de Radiodiagnóstico, Complejo Hospitalario de Segovia, Segovia, Spain

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Clinical Case

A woman aged 73 presented with a four-week history of hypoacusis and low-density clear discharge from her right ear, with no history of traumatic or surgical events. Physical examination showed right middle ear invasion and discharge of clear liquid, which was suggestive of CSF caused by tympanic defects. Left middle ear invasion presented with no other condition.

Computerised tomography (CT) was requested of the cranium, with multiplanar reconstruction to detect hearing disturbances and defects in the petrous bone. The CT scan study (Fig. 1) showed partial invasion of both middle ears and mastoid cells with no ossicular chain or scutum defects. There was no observation of post-traumatic lesions or pathological contrast enhancement. Multiple erosive bony lesions (arrows) existed throughout the internal table at the base of the cranium, predominantly in the middle anterior cranial fossa (Fig. 1B) and temporal bones, including the bilateral tegmen tympani. We decided to complete the study with magnetic resonance (MR) imaging.

Fig. 1C and D (coronal CT) confirmed marked thinning with focal interruption of the bilateral tegmen tympani (arrows). The MR (Fig. 2) showed that, coinciding with the before-mentioned bony lesions, there were small areas of hypointensity in T1 and hyperintensity in T2 (similar to CSF), with well-defined lobed contours. There was no observation of bony expansion or associated soft tissue mass. These findings suggested arachnoid granulations. The remaining sequences showed no evidence of defects except signs of inflammation in both petrous bones, with no bony ossicular chain destruction.

Discussion

Otoliquorrhoea is cause for great clinical alarm and implies communication between the ear and subarachnoid space with a high risk of CNS infection. For this reason its origin must be determined and promptly treated. The most frequent cause of cerebrospinal fluid (CSF) leak through the external auditory canal is post-traumatic and postsurgical events, when a defect is produced in the dura mater enabling CSF to pass beyond the temporal bone. Infectious and neoplastic aetiologies, in addition to congenital aetiologies, are other causes for consideration. One condition considered as a variant of normality is, however, arachnoid granulations which may exceptionally produce erosion of the tegmen tympani and subsequent CSF discharge.1–4

Due to the infrequency of this condition and the possibility of overlooking it, we consider at least a basic knowledge of its radiological characteristics to be necessary. Inclusion in the differential diagnosis would be the next logical step. Arachnoid or Pacchoni granulations were described for the first time by the anatomist Antonio Pacchioni in 1705.4 The arachnoid villi are not visible on a macroscopic scale, but when grouped together they may be detected by the
eye and are called arachnoid granulations. These granulations are therefore simply accumulations of arachnoid villi protruding into the subarachnoid space. According to Grosman they may be observed in 1% of patients. They project towards the cranial vault, specifically within the venous sinuses of the dura mater or towards the calotte and erode the bone forming depressions called granular pits, which look like minor lytic lesions. Any adjacent granulation to the tegmen tympani may cause erosion and establish an escape route for CSF towards the ear.

The main reabsorption and drainage sites of CSF to the venous system are the arachnoid villi. Reabsorption is one directional and occurs when the CSF pressure exceeds that of the venous sinus.

On analysis of CSF fistulas from temporal bone alteration, Savva determined that 90% of cases were related to previous trauma and surgery. In 9%–10% of remaining cases no aetiology could be determined and fistulas were classified as spontaneous. Arachnoid granulations are included within this group.

One theory regarding the aetiopathogeny of the granulations, put forward by Gacek, is that during their development a variable number of arachnoid villi do not end up in the venous sinus but cross the dura mater and come into contact with the bony surface. The pulsating pressure of CSF through them rises, resulting in erosion of the bone and it is possible for them to subsequently create lesions of continuity in the adjacent bone.

Patients with spontaneous otoliquorrhoea generally suffer from impaired hearing or a sensation of fullness. These non-specific symptoms coincide with defects in the tegmen mastoideum and/or tegmen tympani with no abnormality of

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**Figure 1** CT imaging with volumetric and coronal reconstruction in bone cavity window. Image A shows the presence of multiple lytic focal lesions at the base of the cranium, ovoid and lobular in appearance, which vary in size; many of which are connected to one another (included in the image circles, one of them with arrows). No affected soft tissue parts are identified. The volumetric reconstruction of the base of the cranium (Image B) shows that the erosive lesions predominate in the middle (yellow) and anterior (red) cranial cavity, compared with the posterior (green) cavity. Planar coronal reconstruction (Image C) showed lytic lesions affecting the internal table and extending through the dipole thickness to the external table (arrows). We can observe sclerosis of said bony lesion edges which probably translates as benign long-standing aetiology. Thin-cut reconstruction of the temporal bone area (Image D) shows how the bone lesions perforate both tegmen tympani, more severely on the left side (arrow) and there is invasion of both middle ears. This is compatible with arachnoid granulations.
inner ear anatomy. Diagnosis is performed with CT, correlating findings with MR.

Arachnoid granulations are a common and incidental finding. They are mainly detected because they project towards the venous sinuses or calotte. MR imaging shows oval-shaped images which in T1 sequences are hypointense or isointense in cerebral parenchyma and hyperintense in T2 sequences with similar signal intensity to CSF.

The granulations which protrude towards the venous sinuses may simulate venous thrombosis, as they appear as repletion defects in flow sequences (angiographic venous phase) or after contrast.

If the granulations protrude towards the calotte, they are more easily detected in CT and are identified as erosions in the internal table. Although it is true that secondary erosions to arachnoid granulations may affect the tegmen tympani, few cases have been described in literature.

Conclusions

Perforation of the tegmen tympani due to the presence of arachnoid granulations is a condition which, albeit infrequent, should be considered in the differential diagnosis of spontaneous adult otoliquorrhoea. The risk of meningi-tis necessitates an early diagnosis (with MR to corroborate clinical suspicion) and appropriate early ENT treatment.

Conflict of Interest

The authors have no conflict of interests to declare.

References