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A case of a temporal bone meningioma presenting as a serous otitis media

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Abstract

We report the imaging features of a case of a temporal bone meningioma extending into the middle ear cavity and clinically presenting as a serous otitis media. Temporal bone meningioma extending in the mastoid or the middle ear cavity, however, is very rare. In case of unexplained or therapy-resistant serous otitis media and a nasopharyngeal tumor being ruled out, a temporal bone computed tomography (CT) should be performed. If CT findings are suggestive of a temporal bone meningioma, a magnetic resonance imaging (MRI) examination with gadolinium will confirm diagnosis and show the exact extension of the lesion.

Keywords

Head/neck, computed tomography (CT), magnetic resonance imaging (MRI), ear, meninges, primary neoplasms

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Introduction

Meningiomas are frequently found tumors, accounting for 13–26 % of all primary intracranial tumors (1). However, they also tend to occur in extracranial locations. The orbit is most frequently involved and to a lesser extent the nasal cavity, the paranasal sinuses, and the neck. Meningiomas spreading to the temporal bone are very uncommon (2). In most cases they are part of an intracranial meningioma extending into the temporal bone. Meningiomas originating in the walls of the mastoid or the middle ear cavity are much more rare and can present as chronic otitis media, and more specifically as a serous otitis media. In case of unexplained or therapy-resistant serous otitis media and a nasopharyngeal tumor being ruled out, temporal meningioma should be included in the differential diagnosis. Computed tomography (CT) or cone beam CT (CBCT) will show particular changes in the involved bony structures that are very suggestive of temporal bone meningioma. Magnetic resonance imaging (MRI) is the modality of choice to confidently establish the diagnosis and determine the exact extent of the temporal bone meningioma.

Case report

An otherwise healthy middle-aged woman consulted the ear-nose-throat (ENT) department of our hospital because of decreased hearing on the left side. Audiometry confirmed a mild left-sided conductive hearing loss. Otoscopy revealed fluid behind an intact left tympanic membrane. A serous otitis media with secondary conductive hearing loss was diagnosed. Since fiberscopy could not find any space-occupying lesion in the nasopharynx, obstructing the Eustachian tube, an inflammatory cause was assumed to be responsible. Initial conservative treatment with decongestants was started. As no improvement was noted after a few weeks, a ventilation tube was inserted in the left tympanic membrane. As again after a few months no clinical improvement was noted and as chronic ear discharge persisted, a CBCT was performed to evaluate the local status of middle ear and mastoid. This investigation showed a pathological appearance of the bone of the anterior wall of the middle ear cavity as well as

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the tegmen tympani. There was marked thickening and hyperostosis with preserved trabecular architecture (Fig. 1). Also an associated soft tissue opacification was noted in the middle ear cavity without any signs of ossicular erosion (Fig. 1). Based upon these findings an intra-osseous temporal bone meningioma was suggested. An additional MRI was performed to confirm diagnosis. On T2-weighted (T2W) images, a fluid-filled left middle ear was found, with a marked thickened and hypo-intense aspect of the anterior wall of the middle ear cavity (Fig. 2). Gadolinium-enhanced T1-weighted (T1W) images demonstrated enhancement of the thickened anterior middle ear wall and the tegmen tympani (Fig. 2). An intra-osseous meningioma or a so-called *en plaque* meningioma of the bony wall of the middle ear and middle cranial fossa extending through the tegmen tympani to the middle ear cavity was diagnosed. Diagnosis was confirmed at surgery. Surgery was performed via a combined middle ear and middle fossa approach.

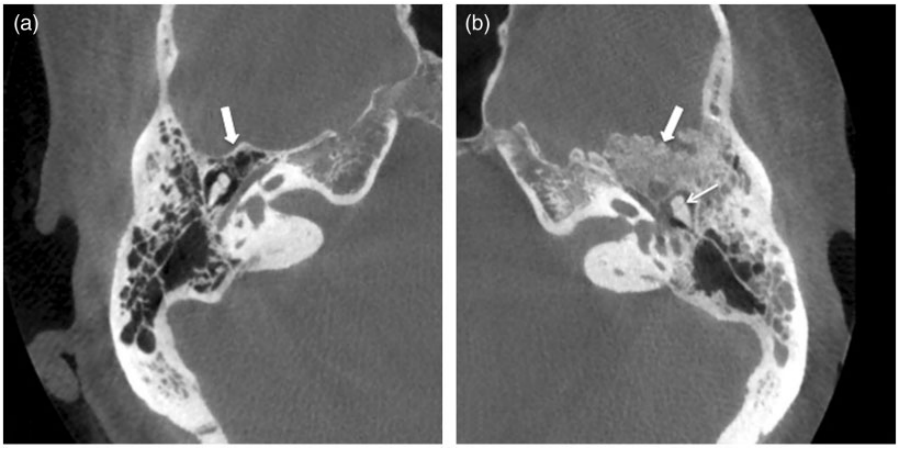

Fig. 1. (a) Axial CBCT image of the right temporal bone at the level of the internal auditory canal (same level as (b)). Note the normal aspect of the anterior wall of the middle ear (large arrow) and the clear aerated aspect of the middle ear. (b) Axial CBCT of the left temporal bone at the level of the internal auditory canal. There is clear thickening and hyperostosis of the anterior wall of the middle ear (large arrow). Note the associated soft tissue opacification of the middle ear cavity. There are no signs of ossicular erosion (small arrow).

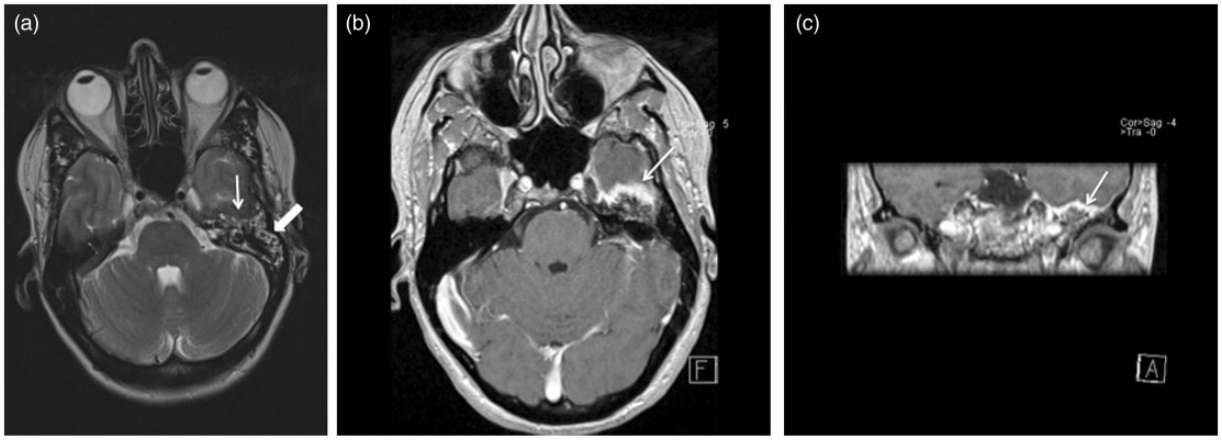

Fig. 2. (a) Axial T2W MR image of the brain at the level of the temporal bone pyramid shows a hyperintense signal in the tympanic cavity and partially in the mastoid cells, compatible with serous otitis media (large arrow). Note the hypointense and thickened aspect of the anterior wall of the left middle ear cavity (small arrow). (b) Axial gadolinium-enhanced T1W MR image at the level of the temporal bone pyramid shows an intensely enhancing anterior wall of the middle ear cavity (arrow). (c) Coronal gadolinium-enhanced T1W image through the temporal bone demonstrates a clear enhancement of the roof of the tegmen. Compare to the normal non-enhancing anterior wall and tegmen on the right side.

Discussion

Meningiomas are very common, slowly growing, benign intracranial tumors arising from arachnoidal cap cells. They account for 13–26% of all primary intracranial neoplasms (1). In rare cases they can be found extracranially. The orbit is most frequently involved and to a lesser extent the nasal cavity, the paranasal sinuses, and the neck. Temporal bone involvement is very rare. Three main access routes into the temporal bone have been described by Hamilton et al. (2). Most frequently intracranial meningiomas spread into the internal acoustic canal, often invading the inner ear, causing sensorineural hearing loss, tinnitus, vertigo, or facial palsy. The next route is direct transosseous spread through the tegmen tympani into the mastoid or the middle ear cavity of an intra-osseous meningioma leading to symptoms of conductive hearing loss and chronic otitis media. The latter is thought to be the result of obstruction of the Eustachian tube due to tumor extension into the middle ear cavity. In some cases a secretory type of meningioma (3,4) or a cerebrospinal fluid (CSF) leakage into the tympanic space (5) is found to be responsible for the fluid accumulation. Chronic discharge will be seen in case of ventilation tube insertion in these patients. The third route is growth through the jugular foramen into the mastoid or the middle ear cavity. A (CB) CT of the temporal bone is recommended in case of atypical serous otitis media persisting despite optimal medical therapy and ventilation tube insertion, and in case a nasopharyngeal tumor has been ruled out. CBCT illustrates the changes in temporal bone architecture suggestive for meningioma. These include the presence of hyperostosis of the invaded bone but with preserved trabecular architecture. In *en plaque* meningioma the free edge of the affected bone can have a typical hairy, irregular aspect. Next to the bony changes a soft-tissue opacification in the middle ear cavity is seen, often encasing but not destructing the ossicles. T2W images will not only demonstrate the hyperintense fluid accumulation in the middle ear but will also demonstrate the global thickening and hypointense sclerosis of the walls of the middle ear. Gadolinium enhanced T1W MRI images will reveal an enhancing dural-based globular mass and/or an *en plaque* enhancing thickened middle ear wall due to the invasion by the meningioma, with possible extension into the temporal bone (6). The preserved bony architecture can be used to distinguish temporal bone meningioma from the typical ground-glass changes seen in fibrous dysplasia. Cholesteatomas and paragangliomas will typically destruct the ossicles and the involved temporal bone. Furthermore cholesteatomas will not enhance after gadolinium administration and show a clear hyperintensity on diffusion-weighted sequences. The above-mentioned imaging characteristics should also be sufficient to exclude granulation tissue, facial nerve hemangioma, and facial Schwannoma. Surgery is the treatment of choice (7). However the decision to do so should be made on a case-to-case basis since damage of important neural and vascular structures may possibly lead to functional impairment of hearing and balance. In the absence of symptoms or if potential morbidity is too high a conservation approach with imaging follow-up is justified (8,9).

In conclusion, temporal bone meningiomas are rare tumors. Extension of these tumors into the mastoid or the middle ear cavity can present as serous otitis media. In patients with unexplained or therapy-resistant serous otitis media a (CB) CT of the temporal bone should be performed. When CT features are suggestive of temporal meningioma, gadolinium-enhanced MRI will confirm diagnosis and give the exact extension of the lesion.

Conflict of interest

None declared.

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