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Horner's syndrome as a complication of acute otitis media

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Abstract. *Horner's syndrome as a complication of acute otitis media.* A seventeen-year-old girl presented with acute otitis media, unilateral miosis and ptosis (partial Horner's syndrome) and tenderness of the ipsilateral jugular vein. A culture of the otorrhoea showed *Staphylococcus aureus* and *Streptococcus pyogenes*. A CT scan revealed an infiltrate near the ipsilateral carotid artery and jugular vein. The patient was hospitalised and treated with antibiotics. Clinical signs disappeared within 6 days.
This report discusses the first case with a partial Horner's syndrome as an unusual complication of acute otitis media (AOM). Imaging studies suggest extracranial lymphatic spread of the infection along the adjacent jugular vein causing pressure on the postsynaptic sympathetic fibres. Recognition of the Horner's syndrome is of importance because it may be an early sign of an extracranial complication of AOM.

Introduction

Acute otitis media (AOM) is a common disease in childhood and is characterised by a pulsating earache, fever and a hyperaemic bulging eardrum on otoscopy. A spontaneous perforation usually occurs due to rising pressure within the middle ear and subsequent local necrosis of the tympanic membrane. In normal circumstances the fever and earache then disappear rapidly and otorrhoea will occur. AOM may be complicated by a mastoiditis.[1] Swelling of the inflamed middle ear mucosa at the aditus ad antrum blocks the mastoid cells, and a cascade of local inflammation, osteitis and necrosis results. AOM may also be complicated by a facial nerve paresis or paralysis, caused by local swelling and inflammation of the nerve within its bony canal. A congenital defect in the bony wall of the facial canal may serve as a *porte d'entrée*.[2] Occasionally, an AOM may be complicated by a labyrinthitis or an intracranial complication such as an epidural abscess, a sigmoid sinus thrombophlebitic infection and a meningitis.[3,4] Extracranial complications of AOM are mostly abscess formation and vascular disorders. The wide spread use of antibiotics has made complications of AOM rare.[5] We report a patient with an unusual complication of AOM.

Case report

A seventeen-year-old girl with a negative medical history presented with left sided otorrhoea and an ipsilateral small pupil and ptosis of one day duration. She had been complaining of a sore throat and earache the previous week. A slight fever had waned and after otorrhoea appeared, she felt better. Her father had noticed a temporary unilateral facial weakness the day before the consultation. This was not observed at the initial presentation. Her eyesight was normal. She had been taking a prostaglandin synthesis inhibitor (Ibuprofen®) and combination ear drops (Sofradex®), without a positive effect.

At physical examination we saw an alert woman with normal temperature, blood pressure and heart rate. Otoscopy showed purulent left sided otorrhoea. Palpation of the neck revealed tenderness of the superior part of the jugular vein. The left pupil was smaller than the right and a ptosis was present on the left. Anhydrosis (i.e. a dry skin because of reduced sweat secretion) could not be detected. The remaining ENT and the neurological examination of the other cranial nerves did not reveal any abnormalities.

Laboratory findings showed an elevated sedimentation rate of 60 mm/hour (normal <12 mm/hour) and a normal white blood count. The C-reactive protein was slightly elevated at 57 mg/l (normal <5 mg/l) as were the thrombocytes at 360×10^9/l (normal 130-150 $\times 10^9$/l).

Streptococcus pyogenes and a *Staphylococcus aureus* were cultured from the otorrhoea.

A CT scan showed partial obliteration of the left mesotympanum and epitympanum and of the

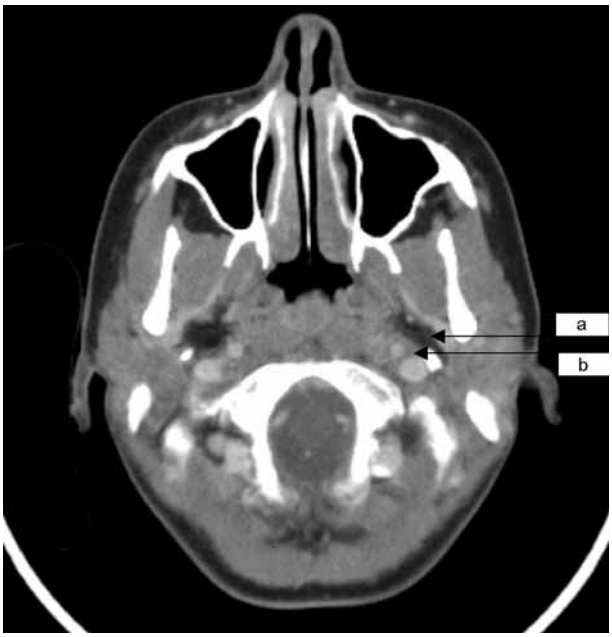

Figure 1
Axial CT scan. The left parapharyngeal fat-pad is smaller (a). There is an inflammatory mass around the left internal carotid artery (b).

aditus ad antrum and the mastoid. There were no signs of bone destruction or abscess formation or of involvement of the facial nerve. However, there was an infectious mass alongside the internal carotid artery on the left side without signs of an abscess (Figure 1). This $0.7 \times 1 \times 3$ cm infectious mass was situated ventro-medial to the left internal carotid artery. No thrombosis of the jugular vein was seen.

The patient was diagnosed as having an AOM with otorrhoea, complicated by a partial Horner's syndrome, which had been caused by an infectious mass alongside the internal carotid artery. Based on the antibiogram of the cultures, intravenous amoxicillin/clavulanic acid 1200 mg four times daily and metronidazol 500 mg three times daily were started.

Within the next six days, her clinical condition improved. At physical examination her pupils were symmetric, there was no ptosis, and the neck pain had diminished. The antibiotics were continued for two weeks orally. On follow up, the symptoms had resolved completely. The tympanic membrane had healed well and hearing had returned to normal.

Discussion

Partial Horner's syndrome as a complication of AOM or mastoiditis has not previously been reported. It is known to result from a nearby intracranial lesion, or secondary to a lesion of the internal carotid artery and is caused by pressure on the postganglionic sympathetic fibres along the internal carotid artery. It has also been called Raeder syndrome[6] (Figure 2). In 1918 and 1924, Raeder described five patients with a Horner's syndrome and ipsilateral head pain.[7] Autopsy in those patients revealed a tumour in the medial cranial fossa. The clinical course was characterized by incomplete oculosympathetic paresis (partial Horner's syndrome), ipsilateral head pain and facial pain. It is differentiated from Horner's syndrome by the absence of anhydrosis, however different classifications have been proposed.[8] The term Raeder syndrome is now frequently replaced by the term "paratrigeminal syndrome".

In the current case report, we presume that the infection near the internal carotid artery was spread from the middle ear by the contiguity of the adjacent jugular vein, and probably due to the large number of adhering lymph nodes. The subsequently formed inflammatory mass along the extracranial part of the internal carotid artery caused pressure on the postganglionic sympathetic fibres with secondary loss of function. This resulted in symptoms comparable to the Horner's or paratrigeminal syndrome.

A six-year-old girl with pharyngitis has been reported with a pseudo-aneurysm of the cervical internal carotid artery due to a deep neck space infection and had presented with a Horner's syndrome.[9]

More recent case reports of patients with a paratrigeminal syndrome involve pathology near the intracranial part of the internal carotid artery and cavernous sinus, e.g. dissection, aneurysm, or stenosis of the carotid artery.[6,10] One patient had a weakening of the arterial wall caused by extension of the inflammatory disease.[11]

One report discusses an 18-month-old boy with an acute left otomastoiditis without a Horner's

syndrome.[12] A CT scan showed thrombosis of the left lateral and sigmoid sinuses and a decreased flow in the left internal carotid artery.

Among the extracranial complications of AOM, vascular complications and abscesses are most common, especially the subperiosteal abscesses (mastoid and Bezold's abscesses). In the "classic" Bezold's abscess, pus escapes from the mastoid through the incisura digastrica and tracks along the digastric and sternocleidomastoid muscles into the upper neck.[13,14] Different other routes have been described, all forming abscesses in the neck deep to the sternocleidomastoid muscle.[15] Vascular complications of AOM include Lemierre syndrome. This usually has an oropharyngeal origin but it can also be otogenic.[16] The anaerobe rod *Fusobacterium necrophorum* is the causative micro organism and is able to form infectious thrombi. The infection leads to thrombophlebitis of the internal jugular vein, and often metastasizes to the lungs, spleen, liver and other organs. Typically, Lemierre syndrome occurs postanginal, with a tender, unilateral swollen neck and pulmonary infiltrates ("postanginal septicaemia").[17]

One must consider arterial complications of AOM in a prolonged clinical course, in the presence of recurrent haemorrhages from ear, nose and throat, or a Horner's syndrome.[12] The diagnosis is often made at an advanced stage when haemorrhages, pseudoaneurysms or acute hemiplegia have developed. The early features of carotid spasms or arteritis can be shown particularly when MR imaging is combined with MR angiography.

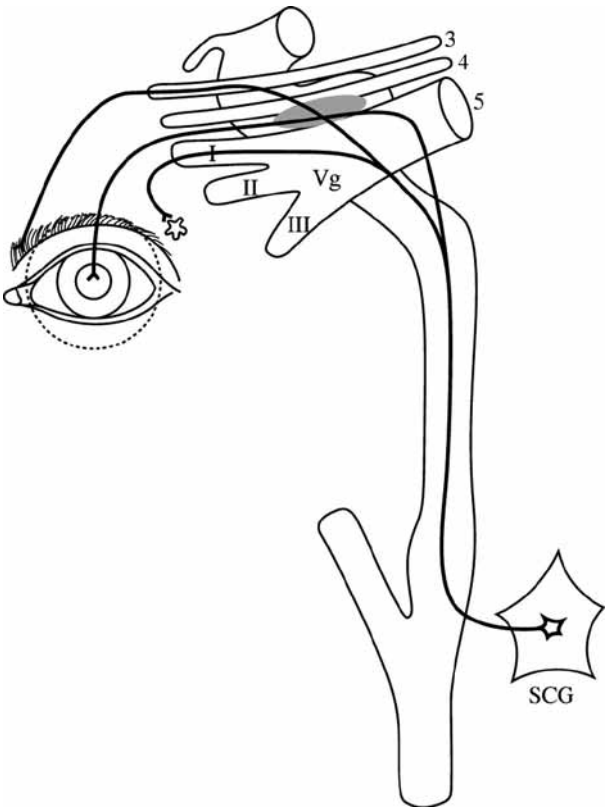

Figure 2
Drawing of the anatomy of the paratrigeminal oculosympathetic syndrome. Sympathetic fibres from the superior cervical ganglion (SCG) travel with the internal carotid artery in the neck, branching to innervate the sweat glands, levator palpebrae superioris (specifically Müller's muscle), and the pupilodilator fibres. The pupilodilator fibres travel near the ophthalmic portion of the trigeminal nerve, so that in this paratrigeminal region (grey shade) lesions produce the classic syndrome. 3: oculomotor nerve; 4: trochlear nerve; 5: trigeminal nerve; Vg: trigeminal ganglion, with first (I), second (II), and third (III) branches. (Printed, with permission, from Goadsby PJ.[18]).

Conclusion

This case report presents a patient with an AOM complicated by a Horner's syndrome. Pressure of an extracranial inflammatory mass on postganglionic sympathetic fibres along the internal carotid artery caused secondary loss of function. We presume the infection was spread by the contiguity of the adjacent jugular vein and its adhering lymph nodes. Due to the early detection of Horner's syndrome, a more protracted clinical course with possible severe vascular complications was prevented.

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