Reminder of important clinical lesson

Recurrent syncope and chronic ear pain

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Summary

An elderly gentleman presented to hospital with recurrent blackout episodes consistent with syncope and a 3-month history of right ear pain. Significant postural hypotension was recorded. White cell count and C reactive protein were elevated. MRI of the head and neck revealed a soft tissue abnormality in the right nasopharynx and base of skull. Tissue biopsies were obtained and microbiology specimens revealed a mixed growth of *Pseudomonas* and diphtheroids. There was no histological evidence of malignancy.

A diagnosis of skull base infection was made. Infective involvement of the carotid sinus was considered to be the cause of the recurrent syncope and postural hypotension.

The patient responded well to a 12-week course of intravenous meropenem. Inflammatory markers returned to normal and a repeat MRI after 3 months of treatment showed significant resolution of infection. The syncopal episodes and orthostatic hypotension resolved in parallel with treatment of infection.

BACKGROUND

We describe a case of a pseudomonal skull base infection involving the region of the carotid sinus causing recurrent syncope in an elderly gentleman. The cause of syncope was unusual yet eminently treatable. A review of the literature has revealed no previous reports of a skull base infection in the region of the carotid sinus presenting with recurrent syncope.

CASE PRESENTATION

An 83-year-old Asian man was admitted to hospital with dizziness, blackouts and increasing confusion. A history was obtained from his daughter that was consistent with syncope. Direct questioning revealed a 3-month history of recurrent episodes of right ear pain and discharge requiring courses of antibiotics. Significant medical history included diabetes and ischaemic heart disease. He was taking ramipril and bisoprolol for hypertension.

INVESTIGATIONS

The patient was afebrile at presentation. Supine and standing blood pressures were 101/57 mm Hg and 68/37 mm Hg, respectively. Marked diurnal variability in resting blood pressure was noted. Cranial nerve and ear examinations at presentation were normal. Initial investigations revealed an elevated C reactive protein of 86 mg/l (normal <10 mg/l), a moderate leukocytosis (white cell count $15.5 \times 10^9/l$; normal $4-11 \times 10^9/l$) and mild normocytic anaemia (haemoglobin 10 g/dl; normal 13.5–18 g/dl). A resting ECG was normal. Limited spoken English precluded formal cognitive testing.

The antihypertensives were stopped. Persistence of symptomatic orthostatic hypotension led to consideration of a diabetic autonomic neuropathy and treatment with fludrocortisone was initiated but with little effect.

Figure 1 Axial fat saturated T2-weighted image showing a large right temporomandibular joint effusion (white arrow) with associated oedema in the lateral pterygoid and around the internal carotid artery, skull base and nasopharynx. There is opacification of the right mastoid (red arrow).

The history of recurrent blackouts and increasing confusion led to a CT scan of the head. The cranial images showed evidence of small vessel ischaemia, but the history of intermittent ear pain prompted a more detailed review of the neck images and this raised the possibility of a soft tissue mass in the region of the right nasopharynx with skull

Figure 2 T2-weighted image (non-fat saturated; at the same level as figure 1) 3 months later following treatment shows no temporomandibular joint effusion and reduced oedema in the lateral pterygoid. There is less high signal around the internal carotid artery but residual right mastoid opacification.

base bone erosion. Following ear, nose and throat assessment, a MRI scan of the head and neck was performed. The scan demonstrated a diffuse soft tissue abnormality in the right nasopharynx (figure 1) and skull base. There was evidence of involvement of the temporomandibular joint and the right internal carotid artery.

DIFFERENTIAL DIAGNOSIS

Nasopharyngeal carcinoma or skull base infection were considered as differential diagnoses and tissue biopsies were obtained. Microbiology specimens revealed a mixed growth of pseudomonas and diphtheroids species. Histological examination demonstrated inflammatory changes with no evidence of malignancy.

A diagnosis of skull base infection was made. Involvement of the internal carotid artery in the region of the carotid sinus was considered to be the cause of the recurrent syncope, orthostatic hypotension and labile resting blood pressure.

TREATMENT

The patient responded well to a 12-week course of intravenous meropenem. Inflammatory markers returned to normal and a repeat MRI after 3 months of antibiotic treatment showed significant resolution of the infection (figure 2).

OUTCOME AND FOLLOW-UP

The syncopal episodes, orthostatic hypotension, labile blood pressure and confusion all resolved in parallel with treatment of infection. Our patient remains well following a prolonged period of rehabilitation.

DISCUSSION

Skull base infection is an uncommon complication of otitis externa that can be difficult to diagnose and treat.¹ A history of persistent otalgia in combination with elevated acute phase proteins and subsequent development of cranial nerve palsies can be clinically suggestive of skull base infection. Involvement of the facial nerve and, less commonly, lower cranial nerves IX to XI can occur during disease progression. Unilateral headache, purulent otorrhoea and leucocytosis are recognised as associated clinical features.² Older patients and patients with diabetes mellitus are at increased risk of developing skull base infection and, as in this patient, *Pseudomonas aeruginosa* is the usual bacterial pathogen.² A minimum 4–8-week course of antibiotics with activity against pseudomonal species is required for treatment.

A review of the literature has revealed no previous cases of a skull base infection in the region of the carotid sinus presenting with recurrent syncope and orthostatic hypotension.

Learning points

- Skull base infection is an uncommon complication of otitis externa that can be difficult to diagnose and treat.
- A history of persistent otalgia in combination with elevated acute phase proteins and subsequent development of cranial nerve palsies can be clinically suggestive of skull base infection.
- *Pseudomonas aeruginosa* is the usual bacterial pathogen.
- Skull base infection is an unusual, yet treatable, cause of recurrent syncope.

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Competing interests None.

Patient consent Obtained.

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