Case report

Insidious onset of headache, diplopia and Horner's syndrome: a rare case of petrous bone osteomyelitis

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SUMMARY

We present an unusual case of skull base osteomyelitis in an 88-year-old woman. She presented with gradual onset unilateral headache and diplopia. On examination, there was evidence of a left-sided Horner's and ipsilateral sixth nerve palsy. In addition to persistent raised inflammatory markers, an MRI neck identified signal change in the petrous bone confirming a diagnosis of skull base osteomyelitis. Skull base osteomyelitis should be considered in presentations of subacute raised inflammatory markers in the context of ipsilateral cranial nerve signs.

BACKGROUND

We present a case of skull base osteomyelitis presenting with headache, a left-sided Horner's syndrome, and a partial sixth nerve palsy. This case is of interest due to its rarity and the diagnostic challenge it represented to the medical team.

CASE PRESENTATION

An 88-year-old woman presented with a 4-week history of worsening left-sided headache and diplopia on leftwards gaze.

The prior medical history was of atrial fibrillation, chronic kidney disease, an old left-sided ischaemic stroke and a spontaneous, minor left-sided parietal intracerebral haemorrhage while on anti-coagulation. Her regular medications were furosemide, apixaban, paracetamol and oxycodone for back pain.

Of note in the previous 6 months, she had been treated for suspected left-sided giant cell arteritis and had seen her general practitioner (GP) and an

ear nose and throat (ENT) specialist for a slowly resolving pseudomonal left middle ear infection.

On examination, the patient was systemically well. She had a left-sided ptosis and miosis in addition to diplopia provoked with leftwards gaze. Important negative findings included a lack of nystagmus, intact visual acuity and colour appreciation and an absence of scalp tenderness.

Initial investigation revealed a persistently raised white cell count (WCC), C reactive protein (CRP) and erythrocyte sedimentation rate (ESR) (present since treatment for suspected giant cell arteritis (GCA)) and a normal CT brain. Temporal artery Doppler demonstrated intimal thickening but no arteritis.

After review by rheumatology (given a history of presumed GCA and a left-sided headache), and neurology—the differentials were felt to include cavernous sinus or skull base pathology given the involvement of ipsilateral sympathetic chain and cranial nerves and the ongoing unilateral headache.

An initial MRI brain was reported as nil acute—with background microangiopathic white matter disease and bilateral internal carotid atheromatous changes. However, due to our suspicions, a second opinion was sought from a neuroradiology specialist, who identified an area of abnormal signal in the left skull base.

A repeat MRI neck with contrast identified bone marrow changes of the petrous apex in the base of the skull, suggestive of an osteomyelitis (figure 1).

The patient was diagnosed with osteomyelitis of the left petrous bone, and was discharged from hospital with ongoing intravenous antibiotics via the outpatient antibiotic therapy (OPAT) service.

OUTCOME AND FOLLOW-UP

The patient was treated with a once daily carbapenem for a total of 11 weeks as an outpatient. After this long course of treatment, the patient's headache and diplopia have resolved, a repeat MRI has shown a significant improvement in appearances of the petrous bone.

DISCUSSION

Skull base osteomyelitis is a rare condition,¹ that usually develops after a local sinus infection or a significant otitis.² In addition to this contiguous spread, haematological spread is also reported. The condition has variable presentations, dependent on the direction of spread of infection. It has been reported in association with Villaret's syndrome³—a

Figure 1 MRI neck: axial fat saturated T2 sequences through level of temporal apex, petrous bone and basiocciput. Pretreatment: images show asymmetry with high signal on the left.

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unilateral lower cranial nerve palsy with Horner's syndrome, as well as in Gradenigo syndrome—featuring trigeminal pain and an ipsilateral sixth nerve palsy.⁴ The condition can be mimicked by primary or secondary malignancies causing local compression.

Skull base osteomyelitis can be challenging to diagnose due to its subtle signs and insidious onset. It is most commonly seen in patients with diabetes or in those who are immunosuppressed and is more common in developing countries. A history of chronic ear problems suggestive of a suppurative process should raise concern for contiguous spread of infection—especially when resistant organisms such as *Pseudomonas* are isolated.⁵

Learning points

- From documentation at the time, the team treating the patient for GCA questioned the lack of inflammatory marker resolution on treatment and felt the patients symptoms did not respond as expected to steroid therapy—the failure of a presumed inflammatory condition to respond to steroids should provoke a reconsideration of the diagnosis.
- The patient had recently been treated for otitis externa with *Pseudomonas* identified as the causative organism. *Pseudomonas* is the most common pathogen⁵ responsible for skull base osteomyelitis and ongoing symptoms should always raise suspicion of potential deep-seated infection.
- The skull base contains many important neurological and vascular structures; ipsilateral cranial nerve neurology, especially with involvement of the sympathetic chain is suggestive of a pathology causing localised damage to these structures.

Although CT would commonly be used to identify osseous destruction, radiological evidence may be lacking until significant bone loss has occurred. As such, focused MRI enables a better evaluation of marrow signal, enhancement and surrounding soft tissue changes.⁶

Multiple cranial nerves transverse the skull base in close proximity to the carotid plexus and associated sympathetic chain. An appreciation of this anatomy enables a physician to isolate a lesion clinically and allow targeted investigation. Clearly, the speed of onset is key in this case—wherein a more abrupt onset would be more suggestive of a vascular aetiology.

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