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Clinical Records

Cholesteatoma causing facial nerve transection

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Abstract
Cholesteatoma is a well recognized cause of a facial nerve palsy. The usual mechanism for this complication is direct pressure on the nerve. We present a case in which the facial nerve has been transected by cholesteatoma and discuss the possible causes.

Key words: Cholesteatoma; Facial Nerve

Case report

A 69-year-old female patient presented to the otolaryngology clinic in 1997 with a chronic discharge from her left ear. Her past medical history included grand mal epilepsy, Parkinson's disease and manic depression. A cholesteatoma was observed and she was noted to have normal facial nerve function.

Three months later she returned to the clinic with an acute onset partial weakness of all divisions of her left facial nerve. Five days later she had a modified radical mastoidectomy at which it was noted that cholesteatoma was surrounding the descending portion of an intact nerve. Her facial nerve was surgically skeletonized from the second genu to near the stylomastoid foramen but the nerve function was unchanged in the immediate post-operative period.

For two years her facial nerve function was seen to decline gradually until in late 1999 it was noted to have no function (House Brackman grade 6). The ear was further explored under general anaesthesia and a transected end of the descending portion of her left facial nerve was found embedded in recurrent cholesteatoma near the mastoid tip (Figures 1 and 2). No distal stump was identified. A biopsy of the nerve was felt not to be relevant at the time of surgery.

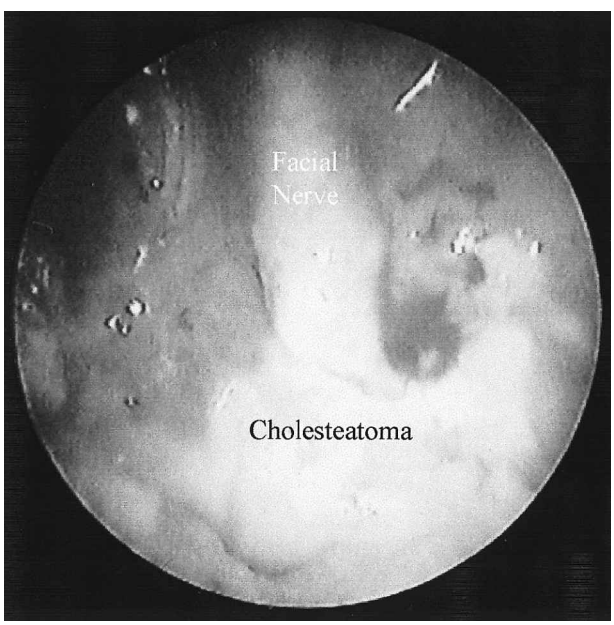

Fig. 1
Descending portion of the facial nerve in the left ear.

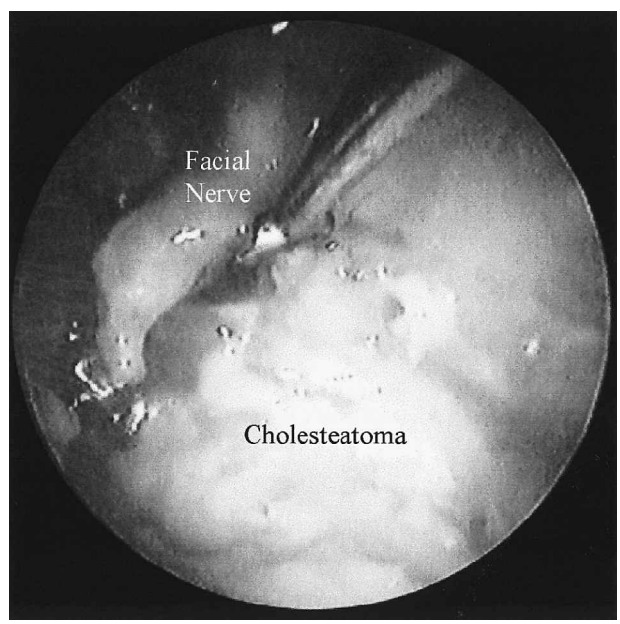

Fig. 2
Deflected descending portion of the facial nerve in the left ear.

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Discussion

Facial nerve palsy in association with cholesteatoma is well recognized but, to our knowledge, there have been no previously reported cases in which the facial nerve has been transected by the cholesteatoma. There are several possible aetiologies for this finding: interruption of the blood supply to the facial nerve, surgical trauma, compression by cholesteatoma or collagenase activity by the cholesteatoma.

The arterial blood supply to the descending portion of the facial nerve is predominantly via the stylomastoid branch of the posterior auricular artery. Near the second genu of the nerve these vessels anastamose with those of the subarcuate artery, a branch of the basilar artery via the labyrinthine artery. In our case the facial nerve was diseased at the stylomastoid foramen, with an otherwise healthy nerve in a more proximal (less well supplied) position. However, in this patient the combination of surgery and the continuing presence of cholesteatoma caused occlusion of the vessels passing through the stylomastoid foramen resulting in localized ischaemia.

Surgical trauma in itself is a recognized cause of facial nerve transection but in this case the patient was noted to have some facial nerve function for around 18 months post-operatively.

Direct compression by the cholesteatoma would seem unlikely as both the nerve and the cholesteatoma were lying in an open mastoid cavity with no resistance to growth in almost any direction.

Studies by Abramson[1,2] on the collagenase activity of cholesteatoma suggested that this was the probable mode of bone reabsorption. Although this might possibly be the cause of the degeneration in this case it would have been expected to have been reported before, especially where the nerve is completely encased in cholesteatoma. Furthermore, Ylikoski[3] studied light and electron microscopic changes in the facial nerves of four patients with cholesteatoma. He found the nerve components to be largely replaced by fibrosis although there was some preservation of myelinated nerve fibres and Schwann cell tubes. There were similar findings in histological specimens from two nerves studied one year after temporal bone fractures.

References

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