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Clinical Record

Traumatic facial nerve neuroma following mastoid surgery: a case report and literature review

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Abstract

Traumatic facial nerve neuroma is rare. There are only 10 reported cases in the literature, caused either by physical trauma or chronic inflammation. Traumatic facial neuromata differ from true facial nerve neoplasms in radiological, macroscopic and microscopic appearance, but clinical presentation is less reliable in differentiating the two. Management depends on the pre-operative grade of facial palsy, as this is a benign condition and surgical management carries a risk of further affecting facial nerve function. We present a further case of traumatic facial nerve neuroma following surgery for cholesteatoma. We also review the literature and discuss the management of this condition.

Key words: Cranial Nerve Neoplasms; Facial Nerve; Neuroma

Introduction

Tumours of the facial nerve are uncommon. A recent review found that, since the first reported case in 1931, there have been a total of 467 published cases.[1,2] One cadaveric temporal bone study found that the incidence of asymptomatic intratemporal facial nerve neuromas was as high as 0.8 per cent,[3] although in other post-mortem studies the figure was less than 0.002 per cent.[4,5] The majority of these are true neoplasms, and may either be benign tumours of the facial nerve itself (such as schwannomas, neurofibromas or haemangiomas), or primary or secondary malignant tumours of the central nervous system or meninges. Most tumours of the facial nerve are benign schwannomas. To our knowledge, only five cases of malignant intratemporal facial nerve neuromas have been previously reported.[6–10]

The development of a traumatic neuroma represents an exuberant, reparative response of the nerve to injury, and identical histological appearances are seen regardless of aetiology. There have been 10 previously reported cases of traumatic neuroma of the facial nerve (Table I), of which four were associated with trauma and six with chronic inflammation of the middle ear.[11–16]

We present a further case of traumatic neuroma of the facial nerve associated with chronic inflammation of the middle ear, and we review the literature on this rare condition.

Case report

A 43-year-old woman presented with a two-year history of vertigo that had developed following a mastoid cavity infection. Her initial mastoid surgery had been at the age of eight years and had resulted in a House–Brackmann grade three facial palsy.

Her vestibular symptoms resolved completely with intensive aural care in our out-patient clinic. However, in the ensuing weeks, her facial recess continued to accumulate keratin. Therefore, she underwent revision mastoid surgery.

A pre-operative audiogram showed normal hearing on the left and a pure tone average (PTA) air conduction threshold of 50 dB with bone conduction PTA of 15 dB. Pre-operative computerised tomography (CT) scanning revealed a normal left ear, while on the right there was evidence of previous limited cortical mastoidectomy. The cavity was sclerotic and fully opaque. A soft tissue density mass was seen intimately related to the second genu of the facial nerve, with extension along the tegmen to the sinodural angle.

At operation, cholesteatoma was found in the sinus tympani and separately over the lateral semicircular canal extending to the sinodural angle. The mastoid cavity was extremely small and sclerotic. In addition, there was a 1 cm diameter mass present in the mastoid cavity and adherent to the dura of the middle fossa, which had the appearance of fibrotic granulation tissue. This was surrounded by and adherent to general fibrosis secondary to chronic inflammation. As the fibrosis was mobilised, the mass was seen to be in continuity with the facial nerve and arising from the second genu. At no stage did the facial nerve monitor alarm, even during gentle mobilisation of the mass. The densely adherent mass was suspected to be a facial nerve neuroma, so was left in situ and the cholesteatoma was removed.

Fourteen months post-operatively, the patient's ear was dry, her hearing and facial palsy unchanged, and she remained free of vertigo.

The position and nature of the patient's facial nerve lesion, in conjunction with the history of previous surgery complicated by complete facial palsy, suggests that the nerve was injured during the first procedure. The injury would have partially transected the nerve, which then recovered incompletely, with development of the neuroma.

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TABLE I

REPORTED CASES OF TRAUMATIC NEUROMA OF THE FACIAL NERVE

Reference	Presentation	Location & diagnostic method	Cause	Management	Outcome
Present case	Chronic middle-ear infection Pre-existing House–Brackmann grade III palsy Transient vertigo Conductive hearing loss	Horizontal segment, 2nd genu (CT)	Chronic middle-ear inflammation	Revision mastoidectomy	House–Brackmann grade III function (unchanged)
Telischi *et al.*[16] (1995)	Chronic middle-ear infection & ipsilateral House–Brackmann grade V paralysis for 3 months Moderate mixed left hearing loss	Tympanic segment (intra-operative finding: mass obscured on CT by middle-ear effusion)	Chronic middle-ear inflammation	1 Oral ciprofloxacin, antibiotic & steroid ear drops 2 Mastoidectomy with tympanoplasty	House–Brackmann grade I function (improved)
Snyderman *et al.*[14] (1988)	Right facial paresis since birth (some movement in all areas of face except forehead) Involuntary facial hyperkinesis (twitching) Normal auditory function Decreased vestibular function (on electronystagmography)	Geniculate, horizontal & vertical segments (tomogram)	Trauma (forceps delivery)	1 Exploration of CNVII (combined transmastoid & middle cranial fossa approach) 2 Brow lift, upper eyelid gold weight implant, palmaris longus tendon transfer to mouth	Nerve function returned to pre-op level, 2 years post-op
Snyderman *et al.*[14] (1988)	Progressive left CNVII palsy over 4 years Slight movement of corner of mouth Weak eye closure Eye/chin synkinesis Decreased tearing & taste Atrophied tongue papillae Normal auditory & vestibular function	Geniculate & labyrinthine segments (CT)	Trauma (hit with baseball bat, no fracture)	1 Transmastoid extralabyrinthine excision Proximal CNVII accidentally avulsed from brainstem 2 Hypoglossal-facial anastomosis	CSF leak post-op day 1, re-explored & repaired Movement at corner of mouth retained Improved facial movement at 1 year

TABLE I
CONTINUED

Snyderman *et al.*[14] (1988)	Progressive left CNVII paralysis over 7 months, greatest in buccal area Decreased tearing left eye Absence of left acoustic reflexes Normal vestibular function	Posterior tympanic & superior vertical segments (CT)	Trauma (mild concussion following road accident)	1 Transmastoid CNVII exploration 2 Upper eyelid gold weight implant for corneal protection during recovery	Partial recovery with slight movement of midface (Further recovery expected at time of writing)
Babin *et al.*[12] (1981)	Normal CNVII function Longstanding decreased hearing right ear Normal vestibular function	Horizontal segment (post-mortem: dehiscent fallopian canal)	Chronic middle-ear inflammation	Not applicable	Not applicable
Babin *et al.*[12] (1981)	Normal CNVII function 1 year history of bilateral chronic serous otitis media 1 episode of vertigo 15 years previously	Horizontal segment (post-mortem: dehiscent fallopian canal)	Chronic middle-ear inflammation	Not applicable	Not applicable
Babin *et al.*[12] (1981)	Hearing loss secondary to mucopolysaccharidosis V Normal CNVII & vestibular function	Chorda tympani (post-mortem)	Chronic middle-ear inflammation	Not applicable	Not applicable
Eby *et al.*[15] (1988)	Incomplete right CNVII palsy Sensorineural hearing loss on left Mixed hearing loss on right	Tympanic segment (post-mortem)	Trauma (repeated longitudinal temporal bone fractures following seizures)	Attempted decompression of CNVII (transmastoid approach)	Worse
Greifenstein[11] (1936)	–	Horizontal segment	Chronic middle-ear inflammation	Unknown	Unknown
Babin *et al.*[13] (1984)	Fullness & conductive hearing loss in right ear Retraction pocket in pars flaccida Normal CNVII & vestibular function	Horizontal segment (incidental finding at operation: dehiscent fallopian canal)	Chronic middle-ear inflammation	Anterior-posterior tympanotomy	Not applicable

CT = computed tomography; CN = cranial nerve; pre-op = pre-operative; post-op = post-operative

Discussion

Traumatic neuromas are not true neoplasms but represent a hyperplastic response of the nerve to injury, either direct or indirect trauma or chronic inflammation. Macroscopically, it may be difficult to differentiate between the types of lesion. However, in general, traumatic neuromas are firm, grey-white, unencapsulated and diffusely involving the nerve, and they are always solitary. In contrast, schwannomas are encapsulated, generally solitary (although they may be multiple) and are attached to the nerve peripherally. Neurofibromas are unencapsulated, often multiple and diffusely involve the nerve.[17]

True nerve sheath tumours can usually be differentiated from traumatic neuromas on the basis of their histological characteristics. Traumatic neuromas show disrupted axons with distal demyelination and Wallerian degeneration, and a tangled mass of Schwann cells and fibroblasts, all in a dense, collagenous matrix. Schwannomas consist of highly ordered, cellular areas (Antoni A) together with loose, myxoid, relatively acellular areas (Antoni B). Axons are generally absent, small areas surrounded by pallisading nuclei (Verocay bodies) may be seen, and blood vessels may be thick and hyalinised. Neurofibromas contain a varying amount of interlacing spindle cells, dense collagen and mucoid matrix; axons may be seen throughout the tumour.[17,18]

Traumatic neuromas have been reported to arise in numerous sites in the head and neck, in addition to the facial nerve, namely, the: maxillary division of the trigeminal nerve, inferior alveolar nerve, auriculotemporal nerve, glossopharyngeal nerve, greater auricular nerve, vestibular nerve and cervical nerve roots.[19–21] However, they do seem to be exclusively an affliction of nerves carrying sensory fibres, as there are no reported cases of traumatic neuroma in purely motor nerves of this region (e.g. the oculomotor, trochlear, accessory or hypoglossal nerves).

Snyderman *et al*. assessed the differences in presentation, imaging and management of true facial nerve neoplasms and traumatic neuromas.[14] In their study, the presentation of traumatic neuromas of the facial nerve appeared to vary depending on the cause. The persistence of facial nerve palsy for longer than three weeks tended to suggest neoplasm, although it was not possible to exclude traumatic neuroma on the basis of presence or absence of progressive palsy. Six out of seven traumatic neuromas associated with chronic mastoiditis had clinically normal nerve function. In the seventh case, the palsy resolved once the mastoiditis had been treated surgically. In the four cases caused by trauma, there was some degree of facial nerve palsy. Two of the cases were static and two were steadily progressive to incomplete palsy in seven months and four years, respectively. In two of the four trauma cases, the nerve palsy remained unchanged following decompression; in one of the cases, it improved slightly from its pre-operative level; and in the fourth case, it was worse post-operatively. Unfortunately, the degree of facial palsy was only assigned a House–Brackmann grade in one paper, so it is not possible to compare cases in this respect. From this small series, traumatic neuromas arising as a direct result of trauma appeared to be associated with facial palsy, whereas those caused by inflammation did not.

The patients with chronic mastoiditis usually had normal or unchanged facial nerve function, and their traumatic neuroma was diagnosed either as an incidental finding at operation (two out of seven), at post-mortem (four out of seven) or on CT (one out of seven). The patient investigated with CT presented after 1994, and the fact that they underwent radiological investigation was probably due to the greater availability of CT at this time, compared with earlier cases. In the trauma group, three out of four patients were investigated with CT or tomogram, and the remaining one was diagnosed at autopsy.

In Snyderman and colleagues' comparison of traumatic neuroma (all of which were traumatic) and facial nerve neoplasms, both showed smooth, concentric enlargement of the facial canal.[14] In the case reported by Teilischi *et al*., which was associated with chronic mastoiditis, the patient underwent CT but a middle-ear effusion obscured the mass.[16] In our patient with chronic mastoiditis, the CT was reported as showing diffuse opacification of the residual sclerotic mastoid, consistent with recurrent cholesteatoma. No mention was made of the possible diagnosis of facial nerve neuroma pre-operatively, but a retrospective review of the films confirmed the operative findings (see Figures 1 and 2).

There is no established management of traumatic neuroma, but it should probably be similar to that of a benign nerve sheath tumour.[14] Benign lesions can usually be differentiated from malignant ones on the basis of history, examination and imaging, and they can generally be observed if facial paresis is stable and function is acceptable. The surgeon should be guided by: the degree, rate of progression and duration of facial palsy; the need for a precise tissue diagnosis; and the expected success of

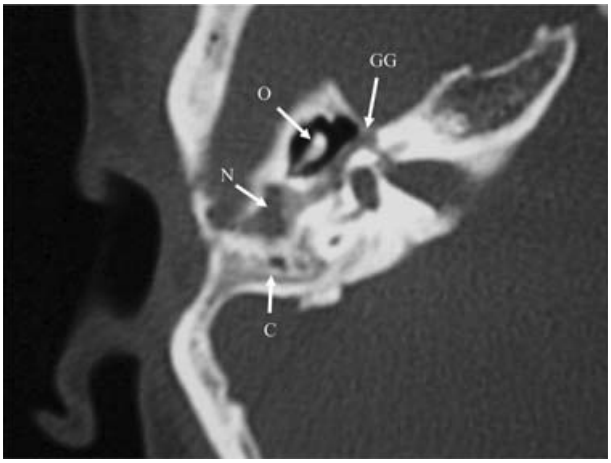

FIG. 1

Axial computed tomography of right temporal bone showing neuroma (N) arising from the second genu of the facial nerve. At operation, the mastoid cavity (C) was small and sclerotic. O = ossicle; GG = geniculate ganglion

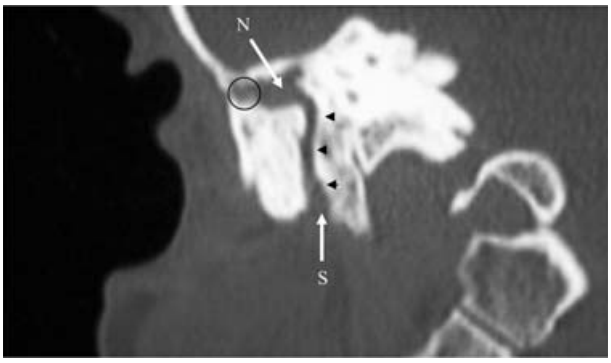

FIG. 2

Coronal computed tomography of right temporal bone at the level of the stylomastoid foramen (S) showing neuroma (N), facial nerve (arrowheads) and the region containing cholesteatoma (black circle).

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nerve grafting. If the facial palsy is severe (i.e. House–Brackmann grades four to six), the lesion should be resected and the continuity of the nerve reinstated via either primary anastomosis or cable grafting. With a less severe palsy (i.e. House–Brackmann grades one to three), the risk is that resection of a neuroma will result in worsening or loss of facial nerve function, so it may be more prudent to adopt a 'watch and wait' strategy and then proceed to resection and nerve grafting if the palsy progresses to completion. There are a number of plastic surgical techniques for improving cosmesis and providing corneal protection if adequate facial function does not return following nerve grafting, including hypoglossal-facial crossover, palmaris longus tendon transfers, brow lifts and implantation of gold weights in the upper eyelid.

- **This paper describes a case of traumatic facial nerve neuroma following surgery for cholesteatoma**
- **Traumatic facial neuromata differ from true facial nerve neoplasms in radiological, macroscopic and microscopic appearance**
- **Management depends on the pre-operative grade of facial palsy, as this is a benign condition and surgical management carries a risk of further affecting facial nerve function**

Conclusion

Traumatic neuroma of the facial nerve is an uncommon entity, but it should be kept in mind when re-exploring a mastoid cavity in a patient with pre-existing, longstanding facial palsy. Computed tomography scanning should be performed pre-operatively but may require careful interpretation. The rarity of traumatic neuroma in the presence of a diseased cavity may make accurate assessment of the scan difficult.

In our case, the mass was enveloped in fibrotic tissue, making satisfactory clearance of cholesteatoma difficult. The surgeon should always ensure adequate exposure of the cavity, particularly if disease clearance is uncertain. If the origin of any mass found is uncertain, the nerve should be traced from its most normal, accessible section towards the mass to determine its relations safely. A nerve monitor may be helpful for this, but it is unwise to rely on it completely, as demonstrated in our case, in which the monitor did not alarm even on manipulating the neuroma.

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