



Outcomes of Facial Nerve Decompression in Local versus Systemic Disease: A Case Series of Four Patients

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Abstract

Background: Facial nerve dysfunction / palsy can have numerous causes, the commonest being Bell's palsy. However, local and systemic causes which can also result in facial weakness to various degrees. Facial palsy is very debilitating if not addressed promptly. Surgical decompression is a treatment option when conservative measures fail, but its benefit is debated. This case series presents four examples of facial nerve dysfunction/ palsy treated with decompression and compares outcomes between local and systemic causes.

Cases: We present four cases with various aetiologies. Three patients had a local cause. Case 1 had generalised osteitis of the facial canal within the mastoid cavity, causing complete House–Brackmann grade VI palsy; total facial nerve decompression

resolved the palsy completely. In case 2, cholesteatoma involved the facial nerve just above the oval window. To avoid weakness from postoperative oedema, the horizontal segment of the nerve was completely decompressed, and she had no postoperative weakness. Case 3 had a metastatic lesion at the mastoid tip, also causing grade VI palsy; excision of the metastasis with local segmental decompression resolved the palsy completely. Case 4 had a systemic cause, presenting with grade VI facial palsy and multiple other cranial nerve palsies, along with sudden hearing loss in both ears at an interval. Initial CT suggested an oedematous facial nerve within the canal, with no other obvious cause. He underwent full-length facial nerve decompression, without improvement. Subsequent MRI showed inflammatory changes in the brainstem affecting the

facial nuclei, which were refractory to medical treatment. He awaits dynamic facial reanimation.

Conclusion: Patients with a local aetiology recovered fully or experienced no resultant weakness after full or partial facial nerve decompression. In absence of well-defined local cause, the facial nerve decompression should be the last resort to restore functional weakness.

Introduction

The facial nerve, cranial nerve VII, is composed of branchial motor, visceral motor, general sensory and special sensory fibres along an intracranial, intratemporal and extratemporal course. It controls muscles of facial expression, but also taste at the anterior two thirds of the tongue, sensory innervation to the part of the external ear and parasympathetic innervation to the lacrimal, submandibular and sublingual glands [1]. Facial nerve dysfunction may arise from local or systemic causes and most commonly presents as facial weakness. Bell's palsy is the commonest cause [2]. Within the temporal bone, the facial nerve runs in a narrow bony canal (the Fallopian canal) and, in disease states, the nerve can become compressed with minor swelling in this location. This can be caused by oedema of the nerve itself in inflammatory conditions; but the nerve can also be compressed from outside by local disease such as cholesteatoma, osteitis or tumour. Compression impairs the nerve's blood supply, initially causing a conduction block, neurapraxia, which is reversible [3]. Surgical decompression aims to relieve this pressure before irreversible damage occurs [4]. Included in this case series are different

examples of facial nerve palsy managed surgically, arising from local and systemic causes, and discussion of how the underlying aetiology may influence outcome.

Case Series

Case 1

A 70-year-old man presented to the emergency department with a left-sided House–Brackmann grade VI facial palsy (Figure 1) [5]. He had undergone mastoid surgery on the same side 25 years earlier. On examination, there was no evidence of active infection, and a small dry perforation was noted within the mastoid cavity. He was treated with a 10-day course of oral steroids. At an ENT review two months later, his facial weakness had not improved. A CT scan was requested and reviewed in the otology clinic. It showed multiple areas of osteitis affecting the facial canal within the mastoid cavity (Figure 2), with no evidence of active infection. By the time surgery was considered, more than six months had passed since the onset of the palsy, and it was uncertain whether decompression would still be of benefit. The treatment options were discussed with the patient, who opted for surgery. In view of the multiple sites of osteitis, full-length facial nerve decompression with nerve mobilisation was performed. No further treatment was required apart from regular home physiotherapy. He made a full recovery to House–Brackmann grade I within six months of surgery, and the mastoid cavity remained dry. At 12 months postoperatively (Figure 3), his facial function remained normal and there was no evidence of infection in the mastoid cavity.



Figure 1: Case 1: 70-year-old man with a left-sided House–Brackmann grade VI facial palsy secondary to osteitis.

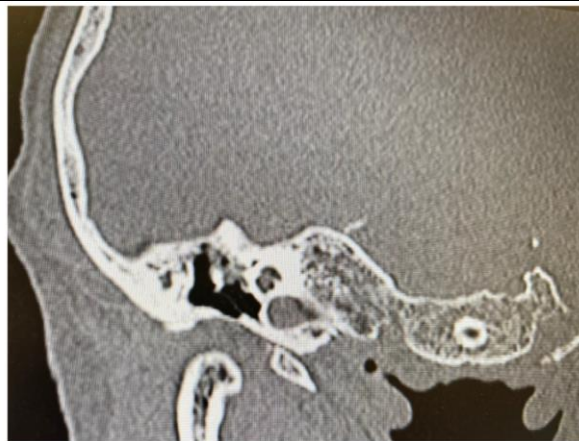


Figure 2: Case 1: The axial computed tomography image showing multiple areas of osteitis on the horizontal segment of the facial nerve within the mastoid cavity with no evidence of active infection.



Figure 3: Case 1: a 70-year-old man 12 months after facial nerve decompression for a left-sided House–Brackmann grade VI facial palsy secondary to osteitis, showing recovery to House–Brackmann grade I.

Case 2

A 56-year-old woman presented with a few months history of discharge from her right ear. She had a history of right-sided conductive hearing loss, treated with grommet insertion. On examination, an attic defect filled with cholesteatoma was identified, and she was listed for surgery. The cholesteatoma was removed by combined-approach tympanoplasty. During the operation, the facial nerve monitor was

triggered while the retraction pocket was being cleared. Once the disease had been removed, the facial nerve was exposed around the second genu and over the oval window, appearing inflamed and oedematous (**Figure 4**). To prevent postoperative weakness from further oedema, the nerve was decompressed anteriorly until normal-appearing nerve was reached. Postoperatively, she had no facial weakness and did not require steroids.

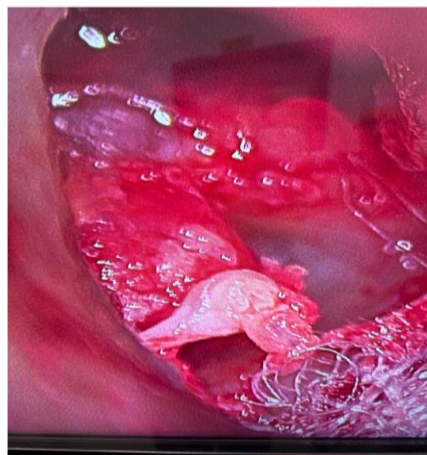


Figure 4: Case 2: Intraoperative view of the exposed facial nerve around the second genu and over the oval window, showing inflammation and oedema.

Case 3

A 76-year-old woman presented with a three-month history of recurrent right-sided otalgia. Her past medical history included breast carcinoma, and at presentation she was receiving palliative care. In primary care, she had been treated with antibiotics and topical ear drops, but the pain continued to worsen. On referral to an ENT consultant, examination showed bilateral age-related hearing loss, with a normal ear canal and tympanic membrane. As her symptoms persisted, CT imaging was requested. It showed a small area of mucosal thickening or haziness with the mastoid cells without bony destruction. Over this period, she developed

intermittent right-sided facial weakness, which progressed to House–Brackmann grade VI (**Figure 5**). In view of the worsening symptoms and her history of breast cancer, the mastoid was surgically explored. Intraoperatively, a fleshy lesion was found at the mastoid tip (**Figure 6**), involving the underlying facial nerve. The facial nerve was exposed. Histological analysis of the lesion confirmed metastatic adenocarcinoma of breast origin. Postoperatively, her facial nerve function was fully restored and her pain reduced. She later developed multiple brain metastases and died approximately one year after surgery.



Figure 5: Case 3: 76-year-old woman presented with right-sided House–Brackmann grade VI facial palsy secondary to breast cancer metastasis at the mastoid tip.



Figure 6: Case 3: Intraoperative view of a fleshy lesion at the mastoid tip involving the underlying facial nerve, later confirmed as metastatic adenocarcinoma of breast origin.

Case 4

A 64-year-old man presented with left-sided facial weakness. Over the preceding 24 months he had developed sudden hearing loss in both ears, 14 months apart, followed by left vocal cord palsy and then left facial weakness. As he had no hearing, recovery of facial function was his main priority. He underwent full-length facial nerve decompression. The nerve was oedematous in multiple segments, and histology of the oedematous tissue showed only inflammatory changes. His facial weakness did not improve after surgery. Subsequent MRI showed similar inflammatory changes in the brain stem affecting the facial nuclei. He did not respond to medical treatment. A tarsorrhaphy was performed to protect his eye, and he is currently awaiting a dynamic facial reanimation procedure.

Discussion

In this case series, patients with a local aetiology recovered fully or did not experience post operative weakness after full or partial facial nerve decompression. Whereas in those with a systemic aetiology, the benefit was temporary or absent. Surgical removal of local disease with facial nerve decompression is associated with good recovery in

most patients [6,7]. In cholesteatoma and chronic ear infections, some authors report early intervention is favourable for the best outcomes [6,7]. However, recovery has also been reported despite delayed surgery [8], similar to case 1, in which decompression more than six months after onset still resulted in full recovery. One possible explanation is that the compression from osteitis caused neurapraxia, rather than complete degeneration. In these injuries, the nerve's internal structure was preserved and can recover fully once the cause is removed [3]. This suggests that delayed presentation should not by itself exclude surgery. Partial decompression, in which the canal is opened only at a localised segment, is the preferred approach by some surgeons [7], as was performed in case 2. In case 3, the patient presented with facial palsy secondary to metastatic breast cancer. In this case, surgical exploration established the diagnosis, and decompression of the facial nerve improved facial function and pain; however, the underlying disease progressed. Temporal bone metastases are uncommon, but when symptoms such as facial palsy and hearing loss occur in patients with advanced malignancy, temporal bone metastasis could be investigated [9]. Surgery may only offer diagnosis and symptomatic relief rather than cure in neoplastic

diseases, and the decision to operate should be made jointly by the patient and surgeon.

In case 4 the patient presented with facial palsy secondary to systemic cause. Full-length decompression revealed oedema in multiple segments of the facial nerve and the subsequent MRI showed inflammation of the brainstem involving the facial nucleus. Decompression could have relieved pressure on the nerve where accessible by the surgeon, but it cannot address disease proximal to it, which may explain the absence of recovery. Despite extensive investigation, no underlying cause of the symptoms were identified, and the patient did not respond to steroids or immunosuppressants. Surgery was a reasonable option for this patient, who prioritised recovery of facial function.

In contrast to the previous cases, where the cause is systemic, the role of decompression is less clear. In systemic inflammatory disease, primary management is medical with corticosteroids and immunosuppressants. Treatment should be tailored to the underlying cause. There is little evidence to support decompression as being beneficial where facial palsy has a systemic cause.

Conclusion

Facial nerve palsy has a wide range of causes, and the underlying aetiology should guide treatment decisions. In this series, surgical decompression was successful where the cause was local, including in a patient who recovered fully despite surgery more than six months after onset. If a local cause, such as osteitis or cholesteatoma is identified, we recommend that it be addressed surgically, even when presentation is delayed. In contrast, where the cause is systemic, decompression will not treat the underlying disease; therefore, its benefit remains

debatable. Surgery may still offer diagnosis or symptomatic relief, and the choice to operate should be individualised through shared decision-making with the patient.

Bibliography

1. Myckatyn TM, Mackinnon SE. A review of facial nerve anatomy. *Semin Plast Surg.* 2004;18(1):5–12.
2. Hohman MH, Hadlock TA. Etiology, diagnosis, and management of facial palsy: 2000 patients at a facial nerve center. *The Laryngoscope.* 2014;124(7):E283-293.
3. Seddon HJ. A Classification of Nerve Injuries. *Br Med J.* 1942;2(4260):237–9.
4. Gantz BJ, Rubinstein JT, Gidley P, Woodworth GG. Surgical management of Bell's palsy. *The Laryngoscope.* 1999;109(8):1177–88.
5. House JW, Brackmann DE. Facial Nerve Grading System. *Otolaryngol Head Neck Surg.* 1985;93:146-147.
6. Choi JW, Park YH. Facial Nerve Paralysis in Patients With Chronic Ear Infections: Surgical Outcomes and Radiologic Analysis. *Clin Exp Otorhinolaryngol.* 2015;8(3):218–23.
7. Psillas G, Constantinidis J. Facial Palsy Secondary to Cholesteatoma: A Case-Series of 14 Patients. *Audiol Res.* 2023;13(1):86–93.
8. Siddiq MA, Hanu-Cernat LM, Irving RM. Facial palsy secondary to cholesteatoma: analysis of outcome following surgery. *J Laryngol Otol.* 2007;121(2):114–7.
9. Song K, Park KW, Heo JH, Song IC, Park YH, Choi JW. Clinical Characteristics of

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