JOURNAL OF CASE REPORTS 2016;6(2):233-236

Medical Management of Facial Nerve Palsy Secondary to Acute Otitis Media

Hickson CJ, Cho WS, Juratli ARS
Leicester Royal Infirmary, Infrimary Square, Leicester, Leicestershire, United Kingdom, LE1 5WW.

Abstract:

A 40-year-old female presented with unilateral, lower motor neuron, facial nerve palsy preceded by a four day history of ipsilateral otalgia and pyrexia. CT scan revealed middle ear opacification without bony erosion or intracranial complication consistent with acute facial nerve palsy secondary to acute otitis media. Usual practice previously described in the literature involves intravenous antibiotics, steroids and urgent surgical intervention in the form of myringotomy and grommet insertion. Following initial treatment with intravenous antibiotics and steroids a rapid improvement of both otalgia and facial nerve palsy was observed rendering surgical intervention unnecessary. The patient was discharged 48 hours later without surgical intervention and made a full recovery.

Key words: Bell Palsy, Earache, Facial Paralysis, Motor Neurons, Otitis Media.

Introduction

The incidence of facial nerve palsy (FNP) is reported as between 17-35 cases per 100,000 [1,2]. Of these just 4% of cases are attributable to infection. Infective causes include chronic suppurative otitis media (CSOM), acute suppurative otitis media (ASOM) and malignant otitis externa. The incidence of FNP complicating ASOM is just 0.2% and is thought to result from a congenital dehiscence of the horizontal portion of the facial nerve present in some 6%-8% of the population [3,4].

While FNP is a well-documented sequelae of chronic suppurative middle ear disease, it is infrequently described in the literature as a consequence of ASOM. Consequently there is no widely recognised consensus on its management. The few case reports described to date advocate the use of corticosteroids, intravenous antibiotics and myringotomy with ventilation tube insertion in the absence of spontaneous tympanic membrane perforation [5,6]. This case adds to the literature by demonstrating successful medical management of a FNP secondary to AOM without tympanic membrane perforation - either spontaneous or surgical.

Case Report

A 40-year-old female was seen in the ear nose and throat (ENT) emergency clinic following a referral by the local urgent care centre. She presented with

Corresponding Author: Dr. Craig Hickson
Email: craighickson@doctors.org.uk
Received: February 15, 2016 | **Accepted:** April 18, 2016 | **Published Online:** May 30, 2016

Conflict of interest: None declared | **Source of funding:** Nil | **DOI:** http://dx.doi.org/10.17659/01.2016.0058

a one-day history of left sided facial numbness and paresis, following a diagnosis of ASOM by her GP 3 days earlier. Her GP had prescribed a course of oral amoxycillin-clavulanic acid upon diagnosis. Two weeks previously she had suffered a lower respiratory tract infection. She had not suffered with previous ear infections, was mildly asthmatic and did not smoke. On presentation at the ENT clinic she had a House-Brackmann (HB) V FNP [7]. Otoscopy revealed a bulging left tympanic membrane without perforation or the presence of vesicles within the external auditory canal. She reported left sided otalgia, hearing loss and tinnitus of one day's duration. Cranial nerve examination was otherwise unremarkable and there was no evidence of corneal abrasions.

On admission the patient was given intravenous amoxycillin-clavulanic acid 1.2 grams thrice daily and a single dose of intravenous hydrocortisone 100 mg followed by oral prednisolone 60 mg once a day. Meticulous eye care was administered. CT scan revealed middle ear opacification in keeping with infection but without bony erosion or intracranial complication [Fig.1].

Twelve hours later her signs and symptoms were much improved. FNP was now HB III and her otalgia was significantly improved without discharge from the left ear. Otoscopy revealed an intact tympanic membrane with less of a bulge. With significant improvement in her symptoms and completion of 24 hours of intravenous antibiotics, she was discharged home with a one week course of oral amoxycillin-clavulanic acid 625 mg thrice daily and a one week course of prednisolone 60 mg once daily. Blood tests, history and examination all pointed to a diagnosis of facial nerve palsy secondary to AOM.

The patient was seen 3 weeks later in an outpatient setting. Her FNP improved with only a very mild weakness remaining; a HB grade II. Tympanogram was type A with normal middle ear volume while an audiogram revealed mild to moderate conductive hearing loss. Further follow-up at 2 months showed complete resolution of FNP – now HB I. Tympanogram was type A, and audiogram revealed mild conductive hearing loss in the higher frequencies only. The patient reported that her hearing was back to pre-infection levels.

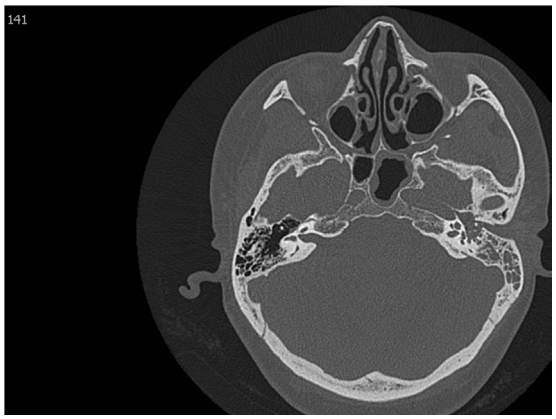

Fig.1: CT temporal bone showing no evidence of bony destruction or intracranial complications.

Discussion

Whilst it is important to try to localise the exact site of the lesion by taking a detailed history and carrying out a thorough examination, this case highlights the findings of previous clinically studies in patients with Bell's palsy that have shown that clinical history and examination is not helpful in determining the exact site of a lesion [8]. In our case the absence of alteration to the sensation of taste would suggest an infrachordal lesion. However this is contrary to our other findings of left sided facial numbness, which suggests trigeminal neuronitis and a more proximal lesion in the region of the genicular ganglion. Trigeminal neuronitis with involvement of the trigeminal ganglion or maxillary and mandibular branches of the trigeminal nerve would result from their close proximity to the genicular ganglion.

This case demonstrates the successful management of FNP secondary to AOM without surgical intervention or spontaneous perforation of the tympanic membrane. The literature to date advocates conservative medical management for FNP secondary to AOM with a discharging ear. In the presence of FNP with an intact tympanic membrane the literature stipulates myringotomy +/- grommet.

Literature searches revealed just three small case series detailing FNP secondary to AOM and its management. The first included 11 patients aged 21-71 years with HB III-V. All patients except one were treated with intravenous antibiotics and oral or intravenous steroids; the exception received antibiotics alone. Eight patients underwent surgery, either myringotomy alone or with ventilation tube insertion. One further patient underwent simple mastoidectomy without facial nerve decompression. The authors did not give details of individual cases or outcomes [5]. The second study of note included 40 patients with FNP secondary to AOM, of whom 38 experienced sudden FNP. Ages ranged from 4 months to 67 years (mean age 15). HB grade varied from I-V with the majority of patients (95%) having grade III-V FNP. This case series reported that 80% of patients developed total improvement from paralysis with 'clinical treatment' alone. Whilst 8 patients were reported to have undergone mastoidectomy the authors failed to report any cases undergoing myringotomy or ventilation tube insertion. Presumable this constituted part of their clinical treatment [6].

The final and smallest series details three cases aged 15 months to 10 years with HB IV-V FNP. All patients received intravenous antibiotics and steroids. Two patients underwent mastoidectomy whilst the third underwent myringotomy. All three patients recovered fully [9].

Conclusion

FNP associated with AOM can be managed successfully with intravenous antibiotics and steroids alone in the absence of a perforated tympanic membrane but may take longer for full resolution. A period of medical management without myringotomy or insertion of ventilation tubes should be considered in all cases. As with any surgical procedure myringotomy and grommet insertion is associated with risks. These include bleeding, perforation, chronic suppurative otitis media, infected grommet and potential need for further procedures.

References

1. Rahman I, Sadiq SA. Ophthalmic management of facial nerve palsy: a review. Surv Ophthalmol. 2007;52(2):121-144.
2. Colbert S, Coombes D, Godden D, Cascarini L, Kerawala C, Brennan PA. How do I manage an acute injury to the facial nerve? Br J Oral Maxillofac Surg. 2014;52(1):67-71.
3. Roland N, McRae R, McCombe A (2001) Key topics in Otolaryngology and head and neck surgery. Bios Scientific Publishers Ltd, Oxford.
4. Joseph E, Sperling N. Facial nerve paralysis in acute otitis media: Cause and management revisited. Otolaryngology - Head and Neck Surgery 1998;118(5):694-696.
5. Redaelli de Zinis LO, Gamba P, Balzanelli C. Acute otitis media and facial nerve paralysis in adults. Otol Neurotol. 2003;24(1):113-117.
6. Yonamine F, Tuma J, Silva R, Soares M, Testa J. Facial paralysis associated with acute otitis media. Braz J Otorhinolaryngol. 2009;75(2):228-230.
7. House JW, Brackmann DE. Facial nerve grading system. Otolaryngol. Head Neck Surg. 1985;193:146-147.
8. Seok JI, Lee DK, Kim KJ. The usefulness of clinical findings in localising lesions in Bell's

palsy: comparison with MRI. J Neurol Neurosurg Psychiatry. 2008;79(4):418-420.
9. Gaio E, Mariono G, Filippis C, Tregnaghi A, Caltran S, Staffieri A. Facial nerve paralysis secondary to acute otitis media in infants and children. J. Paediatr Child Health. 2004;40:483-486.