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MULTIPLE CRANIAL NERVE PALSIES COMPLICATING TYMPANOMASTOIDITIS: CASE REPORT
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# MULTIPLE CRANIAL NERVE PALSIES COMPLICATING TYMPANOMASTOIDITIS: CASE REPORT

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# **SUMMARY**

Otitis media either acute or chronic, is not uncommon in childhood. Multiple cranial nerve palsies occuring as a complication of either form of otitis media is unusual. A case of a nine year old boy with chronic suppurative otitis media with associated mastoiditis complicated with ipsilateral multiple cranial nerve palsies is presented. A skull X-ray and MRI scan showed sclerotic mastoids. The outcome on antibiotic treatment was good.

#### INTRODUCTION

Mastoiditis is a well known complication of both acute and chronic otitis media, often referred to as tympanomastoiditis. Tympanomastoiditis on the other hand may have intratemporal or intracranial complications(1-3). Intratemporal complications, due to anatomical proximity, may involve the cranial nerves, sixth and seventh (and occasionally the fifth) or the labyrinthine system manifesting as vertigo and hearing impairment. Vertigo is often difficulty to discern as a symptom in childhood. The abducens nerve is also commonly involved in petrositis due to its course close to the petrous apex though it may be a late sign(4). Other than cranial nerve palsies, intracranial complications may include meningitis(3,5), intracranial abscesses (epidural, subdural and brain abscesses) (1-3,6-8) and sinus thrombophlebitis(1,2) with their associated clinical features.

Diagnosis of otitis media is clinical whereas in mastoiditis, radiological investigation may be required to confirm the clinical findings especially in latent or masked mastoiditis(9). MRI scan is quite sensitive. A case of otitis media with intratemporal and intracranial complications is presented.

### CASE REPORT

P.N.W., a nine year old boy was seen having been referred for neurological evaluation with a history of ear discharge and a double vision for one month; and regurgitation of fluids into the mouth and nostrils on swallowing for two weeks. One month earlier, the patient had first presented with ear ache, ear discharge, fever, headache and oral sores. He was found then to have suppurative otitis media and stomatitis and treated with crystallin penicillin injection stat followed with oral amoxycillin, later changed to cephalexin, with some good clinical improvement. Ten days later on review, he was noted to have neck pain and nasal speech and oral-nasal regurgitation of fluids. Tonsillopharyngeal and tongue examination was reportedly normal at this time. The oral antibiotics were continued together with analgesics.

Nineteen days from the first day of presentation he was found to have prominent nasal speech and clinically had left sixth and twelfth cranial nerve palsies. The gag reflex was noted to be normal. An X-ray done revealed sclerotic mastoids on the left side.

At the neurological clinical evaluation visit, one month into the illness the patient had oro-nasal regurgitation of fluids on swallowing, sparing swallowing of solid foods: the discharge from the left ear had resolved: had double vision worsened by looking to the left; and mild intermittent headaches were noted without neck stiffness. On examination he was afebrile with palpable tender discrete, mobile left cervical nodes. On neurological examination, no features of meningism were elicited: speech was non fluent with stammer; pupils were normal and so was fundoscopy. He had cranial nerve palsies involving left 5th, 6th, 8th, 9th, 10th and 12th nerves. The tongue was mildly wasted on the left side but fasciculations were not observed and the fifth nerve involvement was mild manifested with absence of corneal reflex only, whereas the features of 8th nerve involvement were impairment of hearing on the left side from an audiogram. The other systems were normal.

The patient was started on empirical treatment for intracranial abscess (of crystalline penicillin, chroramphenicol and metronidazole) while waiting for an MRI scan. MRI scan (done four days into the treatment) showed: moderate left mastoiditis and a normal brain parenchyma; no evidence of edema, sclerosis or mass effect. Hearing assessment confirmed left side hearing impairment Lumbar puncture was not done. The patient showed dramatic improvement and on review two weeks after completing fourteen days of treatment, regurgitation of fluids had resolved, speech had normalised, double vision had cleared and had normal function of left 5th, 6th, 9th and 10th cranial nerves. He still showed mild hearing impairment and 12th nerve palsy (associated with mild wasting of muscles on the left side of the tongue).

# DISCUSSION

The findings of diplopia, a feature of sixth nerve palsy was not unusual in this patient due to its anatomical relationship with mastoids. The features of the 5th nerve palsy were subtle, manifested only as absence of corneal reflex. This is one of the earliest

and most sensitive sign of fifth nerve palsy. The occurrence of diplopia, ear ache (and clinical 5th nerve palsy features) with otorrhoea, define Gradenigo syndrome in this patient.

The sparing of the facial nerve in the disease process is also unusual in this patient since it courses through the mastoids and very close to the middle ear, which were both affected.

Hearing impairment was however, not surprising. Labyrinthitis, one of the causes of this defect, with or without vertigo may arise from direct spread of the infection from the middle ear and /or mastoid, via the round window (the more usual route) or the oval window. Labyrinthitis may also occur secondary to spread of infection from the mastoid antrum, the meninges, the petrous bone or due to bacteremia(6). Conversely, suppurative labyrinthitis may disseminate bacteria into the subarachnoid space via the inner ear fluid and the cochlear aqueduct resulting in menengitis. Suppurative labyrinthitis is a serious disease with severe effects on hearing(6). Due to the moderate hearing impairment, and with fast resolution on antibiotic treatment, this could only have been serious labyrinthitis if at all it was the cause of the hearing deficit. Basal meningitis could also affect the eighth cranial nerve among others as seen here and cause hearing deficit and/or vestibular dysfunction. Clinical features of vestibular system dysfunction were not elicited. Audiogram showed mixed sensorineural hearing defect and conductive deafness too.

Intracranial complications of tympanomastoiditis often occur in the presence of local features of ear discharge or ache, post auricular swelling and tenderness which augment the index of suspicion. Our patient had otorrhoea and earlier features of meningism which together with ipsilateral adjacent multiple cranial nerve palsies (5th, 6th, 8th, 9th, 10th and 12th) suggested more of either intracranial abscess in the cerebellopontine angle and beyond or basal meningitis of contiguous spread. The large number of cranial nerves involved was striking in this patient. Cerebellar signs however, were absent. MRI scan did not show an intracranial space-occuping lesion like abscess. The MRI scan was however, done four days into the empirical treatment for abscess with crystalline penicillin, chrolamphenical and metronidazole. Possibly, the features of the lesion had resolved by this time especially if it was still at the inflammatory stage(10). The earlier use of mild antibiotics definitely contributed to the subacute nature of this infection.

Lumbar puncture was not done due to the aforementioned reasons and fear of increased intracranial pressure. Following the MRI scan findings, and the

already observed clinical improvement it was not considered useful then.

Intracranial complication of tympanomastoiditis are reported to be on the decline with the advent of antibiotics and improved health care but when appropriate antibiotics are delayed, cases are still observed and this is a case to note(11). And if the complications are not considered or suspected delay in treatment is likely to be associated with severe residual effects including mortality. Gower and McGuirt reported a mortality rate of 12% among 76 patients who had meningitis following acute and chronic otitis media(12).

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