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CASE REPORT

Dysphagia due to necrotizing otitis externa

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Abstract

An 88-year-old man presented with delirium, and subsequently developed hoarseness and oropharyngeal dysphagia. This was due to skull-based osteomyelitis from necrotizing otitis externa (NOE), causing lower cranial nerve (X, XII) palsies and venous sinus thrombosis. Diagnosis was delayed as the patient reported no otalgia, had an almost normal looking external auditory canal and was not diabetic. He deteriorated and died despite intravenous antibiotics. We need a high index of suspicion for NOE and its complications in patients presenting with otolaryngeal symptoms.

Keywords: delirium, dysphagia , necrotizing otitis externa, skull-based osteomyelitis, lower motor neuron palsy, older people

Key Points

- Older people with diabetes, or who are immunosuppressed, are most commonly affected by necrotizing otitis externa (NOE).
- Severe, unrelenting otalgia and persistent otorrhea are the symptomatic hallmarks of NOE.
- This case highlights an atypical presentation, with delirium, of NOE in an older person.
- Dysphagia caused by Skull Based Osteomyelitis secondary to a NOE is very rarely seen on our geriatric wards.
- A high index of suspicion for NOE and its complications is needed in patients presenting otolaryngeal symptoms.

Introduction

Dysphagia in older people is commonly due to decompensation of neurogenic causes such as dementia, vascular or Parkinson's disease [1], but other causes are possible. We report here a case of an older person presenting with dysphagia due to NOE despite no history of diabetes or immunosuppression.

Case

An 88-year-old man presented to the geriatric medical admission unit with confusion, unsteadiness and cough. He was normally mobile with a stick, and had no relevant past medical history. His wife reported that 2 weeks previously he was treated with antibiotics for chest and ear infections, which resolved, but he became progressively more drowsy, sometimes falling asleep whilst talking.

On admission, his temperature was 36.8°C, blood pressure 127/70 mmHg and oxygen saturations 97% on room air. He appeared alert and abbreviated mental test score was 9/10. He had crepitations at the left lung base. There was wax in his right ear, but no discharge. Blood tests showed haemoglobin 115 g/L and white cell count 7.2 × 10^9/L. He had hyponatremia (sodium 119 mmol/L) with normal cortisol, low serum osmolality, high urinary osmolality and sodium consistent with syndrome of inappropriate antidiuretic hormone secretion. Chest X-ray showed no consolidation. Computed tomography (CT) head scan showed bilateral chronic otitis media.

The initial diagnosis was delirium secondary to hyponatremia and lower respiratory tract infection. He became unwell the following day with cough, breathlessness, raised white cell count and C-reactive protein and was treated with 5 days of piperacillin and tazobactam to which he responded well clinically. Audiology review described pus in his right auditory canal. Hyponatremia responded to fluid restriction. He then developed severe hoarseness and dysphagia. The purulent discharge was micro-suctioned by Ear, Nose and Throat (ENT) Team. A fibreoptic nasendoscopy found no

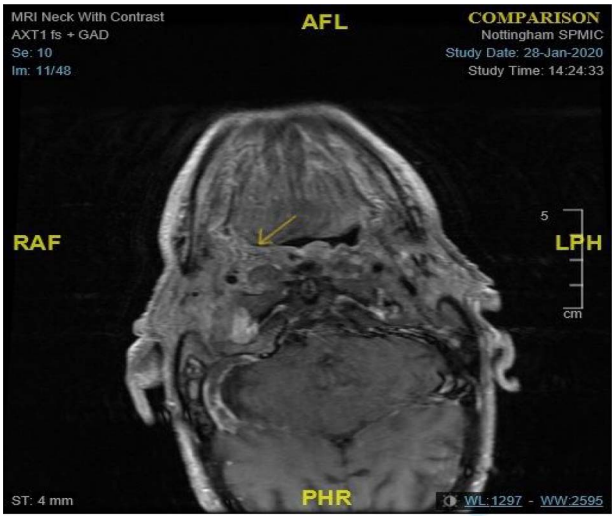

Figure 1. MRI neck post contrast axial T1 image showing an enhancing inflammatory mass in the right infratemporal fossa denoted by arrow.

masses, but there was bilateral vocal cord palsy. On further examination, he had severe dysphonia and bilateral equal palatal movements, although the uvula was deviated to the right and his tongue was deviated to the right on protrusion, representing X and XII cranial nerve palsies. Autoimmune, vasculitic, human immunodeficiency virus, syphilis, Lyme and paraneoplastic serology, as well as CT chest, abdomen and pelvis were normal.

His ear swab grew *Pseudomonas aeruginosa* for which he was given ciprofloxacin ear drops. Nasogastric tube feeding was commenced. Magnetic resonance imaging (MRI) revealed an extensive mass lesion from a nasopharyngeal source to the right skull base region in keeping with a right necrotising otitis externa (NOE), with skull base osteomyelitis as a complication (Figure 1). There was right venous sinus thrombosis. A posterior fossa CT demonstrated erosive changes involving the right skull base and the inferior aspect of the petrous apex but with relatively normal mucosal lining of the right external auditory canal.

Despite being treated with broad spectrum antibiotics, anticoagulation, otic antibiotics and regular micro-suction of his otorrhea, he died from aspiration pneumonia.

Discussion

NOE is an invasive infection of the external auditory canal and skull base that can cause cranial nerve palsies, meningitis, brain abscess and dural sinus thrombosis [2]. This case highlights an atypical presentation, with delirium, of NOE in an older person.

Severe, unrelenting otalgia and persistent otorrhea are the symptomatic hallmarks of NOE [4]. Older people with diabetes, or who are immunosuppressed, are most commonly affected [2]. If untreated, cranial neuropathies can develop due to subtemporal extension of the infection [2], which, in this case, resulted in dysphagia from involvement of the lower cranial nerves. Prompt diagnosis and aggressive treatment with systemic antibiotic therapy should reduce disease extension and the need for surgery [3]. Mortality remains at 20% despite antibiotic treatment, with intracranial complications being the major cause of death [4]. Prognosis is worse in those with cranial nerve palsies or venous sinus thrombosis [2,5].

Declaration of Consent: Written informed consent for publication of their clinical details and clinical images was obtained from the proxy.

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Declaration of Conflicts of Interest: None.

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