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Central nervous system complications of acute tonsillitis

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Abstract
Acute tonsillitis is a common infection of early childhood which may even run a self-limiting course without antibiotic therapy in some cases. Complications are encountered infrequently and rarely assume a life-threatening propensity. Central nervous system involvement has not to our knowledge been described in association with tonsillitis without local abscess formation. We describe three cases in which acute tonsillitis/peritonsillitis was complicated by major central neurological sequelae.

The neurological complications encountered in young previously healthy adults were: facial palsy and hemiplegia; superior sagittal sinus thrombosis with communicating hydrocephalus and papilloedema; Guillain-Barré syndrome and facial palsy. The pathogenesis and management is discussed. All patients made satisfactory recoveries, though with minor residual neurological disabilities.

Key words: Tonsillitis; Central nervous system, complications

Introduction

Tonsillitis is a common infection and may be due to viral or bacterial organisms. Most cases resolve spontaneously or with the aid of suitable antibiotic treatment. Serious complications such as acute rheumatic fever or glomerulonephritis are rare and usually due to streptococcal infection which is readily treatable with broad-spectrum antibiotics. Major central neurological sequelae are extremely rare in the absence of peritonsillar abscess formation. We describe three cases in which acute tonsillitis or peritonsillitis was complicated by major central neurological sequlae.

Case reports

Case 1

A 42-year-old man presented to his general practitioner with a five-day history of acute tonsillitis. There was associated pyrexia, left-sided otalgia and left-sided dental pain and he was prescribed oral penicillin. Three days later he woke with a left-sided facial palsy and ipsilateral loss of taste. Dexamethasone was commenced but there was no early recovery. About five to six weeks later weakness and paraesthesiae of the left upper limb developed and he was admitted for expert neurological assessment. On examination, a dense left facial palsy was present with left hypoageusia and an absent gag reflex on the same side. The remaining cranial nerves were functioning normally. The left upper limb was weak, being worse distally. Reflexes were generally brisker on the left side and both plantar responses were down-going. Co-ordination and sensation were found to be normal, as was the general medical examination.

A computed tomography (CT) scan was performed and showed a small area of reduced density lateral to the body of the right lateral ventricle, with a similar area of possible reduced density within the deep white matter of the posterior part of the right parietal lobe. Electroencephalogram (EEG) studies showed isolated focal slow wave abnormalities in the right parietal area and runs of irregular theta activity. Left facial nerve stimulation gave no response and electromyogram (EMG) monitoring showed no remaining units of left frontalis, orbicularis oculi or mentalis to be under voluntary control. It was thought that inflammation around the right internal carotid artery may have caused emboli and so digital subtraction angiography was performed. This failed to show an abnormality. Two years later, the patient is left with only a slight weakness of the left upper limb and an odd sensation in his mouth, the remaining neurological deficit having resolved.

Case 2

A 15-year-old boy presented with a short history of sore throat, dysphagia, otalgia, headache and vomiting. A diagnosis of tonsillitis was made and penicillin therapy commenced. Three weeks later he rapidly became weak in the left arm and leg with associated headache, nausea and mild neck stiffness. On examination he was found to be systemically unwell with a pyrexial of 39°C. He had mild peri-orbital swelling, particularly on the right side together with meningism and mild papilloedema. He displayed weakness of both the left facial muscles and left trapezius and a profound left hemiparesis and hemianaesthesia.

Cranial CT scanning was performed, showing a haemorrhagic infarct in the right frontal region. There was prominent enhancement along the falx and its junction with the tentorium. The appearances were consistent with sagittal sinus thrombosis with a degree of secondary haemorrhagic infarction. The fever and neurological signs resolved following commencement of phenytoin, benzylpenicillin and chloramphenicol. The patient was left with visual blurring owing to persistent mild intra-cranial hypertension secondary to the sagittal sinus thrombosis.

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Case 3

A 30-year-old woman presented to her general practitioner complaining of sticky, inflamed sore eyes for which she was prescribed topical drops. She attended one week later with a sore throat, mouth ulcers, cervical lymphadenpathy and pyrexia. A diagnosis of tonsillitis was made and she was commenced on erythromycin. Five days later, she woke with a feeling of numbness of her fingers and toes, the sensation gradually ascending to her abdomen. This was followed by weakness of her legs and episodes of faecal and urinary incontinence, urgency and reduced rectal and urethral sensation. On examination, she was found to have mild facial weakness, together with weakness of both upper and lower limbs bilaterally, the proximal musculature being affected more than the distal.There was a glove and stocking sensory loss to pinprick up to the level of the wrists and mid-calves on both sides. She displayed no reflexes other than downgoing plantars bilaterally. A diagnosis of Guillain-Barré syndrome was made. The neurological signs improved after four to five days and showed a significant improvement after eight weeks. The sore throat became chronic and after acute exacerbations requiring systemic antibiotics, tonsillectomy was performed. The neurological improvement was then able to proceed in a slow but consistent manner.

Discussion

Tonsillitis commonly has a viral aetiology, often occurring as part of a cold or flu-like illness and as such is usually self-limiting. Bacterial tonsillitis which is frequently due to β-haemolytic streptococci, such as *Strep. pyogenes*, is seen less frequently. Its true incidence is difficult to determine, however, since the clinical picture is very similar to viral tonsillitis and throat swabs tend to be unhelpful in the diagnosis.

A number of serious complications are associated with bacterial tonsillitis, in particular. Peritonsillar and parapharyngeal abscess, suppurative cervical adenitis, acute rheumatic fever and glomerulonephritis are well recognized, although less common since the advent of antibiotics (Danforth, 1963). More rarely, myocarditis has been desribed as a sequela to tonsillitis (Dickson *et al.*, 1983), as have life-threatening complications including adult respiratory distress syndrome, disseminated intra-vascular coagulopathy and pyothorax (Davis *et al.*, 1986). Reports of intracranial and neurological complications are, however, scant in the literature. Cases of cerebral abscess and cavernous sinus thrombosis secondary to peritonsillar abscess formation have been described (Buchheit *et al* 1970; Nguyen *et al.*, 1984), some reviews placing the incidence of cerebral abscesses due to otolaryngological causes at up to 30 per cent (Garfield, 1969). The majority of these are, however, likely to be due to suppuration in the sinuses or mastoid (Samson and Clark, 1973).

The spread of infection from the tonsillar region occurs, presumably, due to the embolization of foci of infected material by either retrograde venous spread, or from an internal carotid artery damaged by the effect of local infection in the parapharyngeal space. It is well-known that the internal carotid artery may be affected by local pus, causing septic necrosis in the most extreme cases (Danforth, 1963; Blum and McCaffrey, 1983), or pseudoaneurysm (Watson *et al*, 1991), which may be discovered in a catastrophic fashion at subsequent tonsillectomy. This explains the observation that intracerebral complications are seen more frequently in cases where the parapharyngeal space is involved in the infective process, since the vascular structures, together with the accompanying cranial nerves IX-XII and the sympathetic chain are found in the post-styloid compartment, 2 to 3 cm deep to the tonsil. Neither of our cases affected by central neurological compromise were clinically shown to suffer more than focal tonsillitis, however.

It is possible that local spread of infection via lymphatics was responsible for the complications. Similarly, palsies of cranial nerves IX-XII and more rarely Horner's syndrome (Blum and McCaffrey, 1983) due to involvement of the cervical sympathetics have been recorded in cases of peritonsillar or parapharyngeal infection and in one case, a XII nerve palsy occurred in relation to influenza immunization (Felix *et al.*, 1976). We have found one record of a unilateral hypoglossal palsy in a nine-year-old girl suffering from 'chronic tonsillitis' although there was no acute episode apparent (Chamdawalla and Lubec, 1985). This was deemed by the author to be due to a para-infectious neuropathy.

More difficult to explain, however, is the lower motor neurone palsy of the facial nerve seen in *Case 1*. Palsies of cranial nerves IX-XII may be explained by virtue of their anatomical position but the facial nerve does not enter the parapharyngeal space during its course. The pathological process responsible for the palsy must have occurred at a site distant to the cranial nerve nucleus, however, since the weakness was lower motor neurone in nature. It is possible that the aetiology was an inflammatory process independent of the hemiplegia which occurred subsequently. Certainly, there was a significant delay of several weeks before the onset of the CNS features.

Similarly, our third case in which a diagnosis of Guillain-Barré was made is difficult to explain in terms of either local suppuration or embolism-infected material. We assume that the Guillain-Barré syndrome and the facial nerve palsies are part of a polyneuropathy of likely, though unproven, viral aetiology.

Conclusion

The association of such severe neurological impairment with tonsillitis in the absence of abscess formation in the parapharyngeal space is unusual. These cases serve as a timely reminder that tonsillitis may not always be a uniformly benign condition. Peritonsillitis itself, with or without subsequent abscess formation, merits aggressive treatment with high-dose systemic antibiotics and when severe, hospitalization is indicated.

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