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Acute suppurative parotitis and facial paralysis

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Abstract
A case of facial nerve paralysis secondary to acute suppurative parotitis is described. This is a rare complication in the absence of malignant processes in the parotid.

Key words: Facial paralysis; Parotitis, acute

Introduction

We describe a case of an unusual presentation of lower motor neuron facial palsy related to acute parotitis.

Case report

An 88-year-old lady was referred to our unit with a short history of a painful swelling of the right side of the face. Over this period, she rapidly developed a total right facial paralysis. This was not associated with any constitutional symptoms. There was no recent past medical history.

Initial examination showed a diffuse tender swelling over the right parotid area with no palpable stones in the parotid duct. Pus was expressed from the duct and the culture grew *Staphylococcus aureus*. There was a complete right lower motor neuron facial palsy. The remainder of the ENT examination and neurological assessment was normal. The clinical impression was that of acute parotitis with a facial nerve palsy.

The blood film showed leucocytosis with neutrophilia. Erythrocyte sedimentation rate was 110 mm/h and blood biochemistry was within normal range. An auto-antibody screen was negative. C-reactive protein was raised at 282 mg/h.

Serial viral titres against adenovirus, mycoplasma, mumps and cytomegalo virus were not raised. IgG antibody titres to Epstein-Barr virus (EBV) nuclear antigen were present but as Epstein-Barr nuclear antigen appears late, a recent EBV infection was considered unlikely. X-ray of the parotid did not reveal any radio-opaque stone in the parotid gland. On the advice of the radiologist, a computer tomography of the parotid was performed. It showed an enlarged gland with a diffuse patchy enhancement after contrast administration indicating diffuse parotitis (Figure 1). No localized mass was identified. Electromyographic studies were consistent with a lower motor neuron paralysis.

The possibility of Sjögren's disease was not investigated further as the patient did not have any symptoms suggestive of the disease.

The patient was treated with analgesia and intravaneous Augmentin 1.2 g three times daily. Over the next few days, the swelling subsided and pain decreased. The facial palsy did not significantly improve. At three months follow-up, there was no facial nerve recovery, and the patient was referred for facial nerve stimulation. On review at six months, the facial nerve function had fully recovered.

Discussion

Acute suppurative parotitis is a condition seen mainly in the elderly, the diabetic and the immunocompromised. The incidence is equal in both sexes. Decreased salivary flow brought about by dehydration or drug administration such as diuretics and anticholinergic drugs predispose the patient to parotitis. Poor oral hygiene and Stensen's duct obstruction are the other predisposing factors. Before the advent of antibiotics, this condition was seen in post-surgical patients giving rise to the term post-surgical

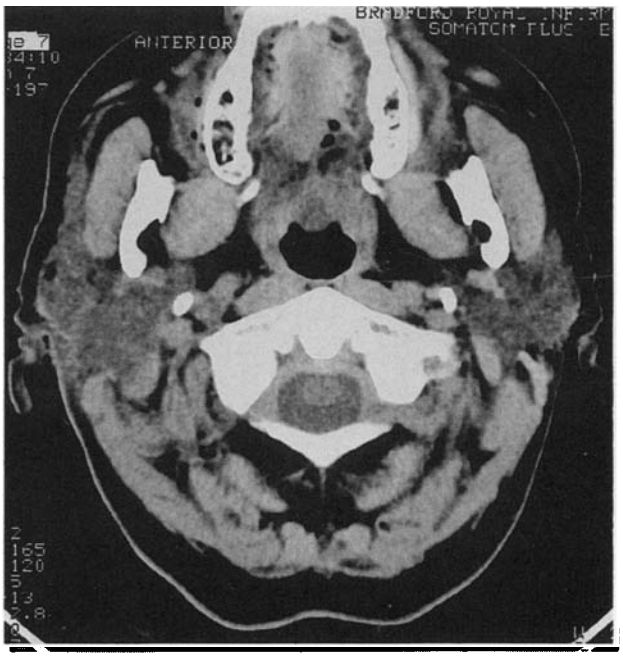

FIG. 1

Axial CT scan showing a diffuse enlargement with patchy enhancement throughout the right parotid gland. There is no mass lesion nor lymphadenopathy identified in the parotid.

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parotitis. Retrograde bacterial migration from the oral cavity up the salivary duct is the most likely route of bacterial entry. These infections are seen in all salivary glands, but the parotid gland is most frequently involved because of inferior bacteriostatic activity in its secretion (Spratt, 1961). The most commonly incriminated bacteria are *Staphylococcus aureus* and the streptococcal species (Lundgren *et al.*, 1976; Mandel, 1976).

Complications in acute parotitis are rare. An early complication is abscess formation. This would require surgical drainage if the patient does not improve with conservative treatment. Long term complications include recurrent infections due to sialectasis and there may be decreased salivation and chronic facial pain.

Facial nerve involvement is very uncommon in non-malignant conditions of the parotid. It may range from a partial to total paralysis. Only six cases of facial nerve dysfunction associated with acute parotitis (Kapadia *et al.*, 1967; Duff, 1972; Shone and Stewart, 1985; Andrews *et al.*, 1989) have been reported in the literature. The extent of facial nerve dysfunction depends on the virulence of the organism, the extent of perineuritis and nerve compression.

Management of these cases is initially conservative with aggressive broad spectrum antibiotics, rehydration, sialogogues and good oral hygiene. In cases that do not respond clinically, surgical debridement may be necessary. Parotidectomy should only be considered in cases where fine needle biopsy suggests malignancy, or where a mass persists after the resolution of the acute inflammatory process, or where there is a failure of recovery of the facial nerve.

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