

CASE REPORT

Facial Nerve Paralysis: A Rare Complication of Parotid Abscess

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Abstract

Benign parotid neoplasm and inflammatory processes of the parotid resulting in facial paralysis are extremely rare. We report a 72-year-old Malay female with poorly-controlled *diabetes mellitus* who presented with a painful right parotid swelling associated with right facial nerve palsy. The paralysis (Grade VI, House and Brackmann classification) remained after six months.

Keywords: facial nerve injuries, parotid neoplasms, parotitis, neurosciences

Introduction

Facial paralysis associated with benign inflammation in and around the parotid gland is unusual in the absence of a malignant process. The association appears to be rare with less than 15 cases being reported in the literature (15).

Case Report

A 72-year-old Malay female with poorly-controlled *diabetes mellitus* presented with a five-day history of painful swelling of the right parotid, trismus and fever. Three days later, she developed a Grade III right facial nerve palsy that later progressed to Grade VI (House and Brackmann classification). She denied any history of parotid swelling. Examination revealed a diffuse, inflamed and tender swelling of the right parotid gland measuring approximately 5 x 7 cm with purulent discharge noted from the right Stensen's duct. There were no palpable stones within the parotid duct. There was a complete right lower motor neuron facial nerve palsy. The remainder of the examination was unremarkable. A parotid abscess with facial nerve palsy was the final impression.

Complete blood count revealed a total white cell count of $29.6 \times 10^9/L$ and a blood glucose of 18.6 mmol/L. She was started on intravenous amoxicillin and subcutaneous

insulin. A CT scan was not performed, as there was no evidence of parapharyngeal extension.

She underwent incision and drainage of the abscess via a modified Blair incision. Intra-operatively, there was massive necrosis involving the deep lobe of the parotid gland, which mandates aggressive surgical debridement. Identifying the facial nerve was difficult because necrotic tissue had replaced most of the normal parotid tissue. One week after the first operation, she underwent further surgical debridement to remove necrotic parotid tissue. Subsequently, the large wound created by aggressive debridement was closed with a V-Y advancement flap. The tissue biopsy obtained during the first operation showed necrotic tissue with no evidence of malignancy or tuberculosis. Culture of the pus grew *Klebsiella* spp. that was sensitive to amoxicillin. Grade VI paralysis remained at six months.

Discussion

Parotid abscess is a condition seen mainly in elderly, diabetic, and immunocompromised patients, as presented in this case. Ascending bacterial migration from the oral cavity to the salivary duct is the likely route of bacterial entry. However, this patient did not have any predisposing factors for parotitis (e.g. poor oral hygiene, Stensen's duct obstruction, dehydration).

The incidence and the pathophysiology of facial palsy in conjunction with parotid abscess or parotitis are unknown. It may range from a partial 40to total paralysis. Multiple and poorly-understood factors may account for facial dysfunction in the setting of parotid gland inflammation. Various mechanisms have been proposed to account for the nerve involvement in a non-malignant lesion. These include direct pressure (6), inflammation and necrosis (7) and haemorrhage into a cyst or a tumour (8). In this case and other suppurative diseases, the virulence of the organisms, the extent of perineuritis, and acute nerve compression are likely to be the causes. Nerve damage may be induced by increased pressure over an abbreviated time in addition to the toxic effects of surrounding inflammation (9).

Management of parotitis complicated by facial paralysis should initially be conservative with aggressive broad spectrum antibiotics, rehydration, sialogogues (e.g. lemon juice) and good oral hygiene (9). In most reported cases of parotitis with facial palsy, causative organisms were not identified. Therefore, it is important to use a broad-spectrum antibiotic until a specific organism can be isolated. Most cases report recovery of the facial nerve after resolution of the acute inflammatory process (9). Surgery does not play a major role in the treatment of isolated inflammatory disorders of the parotid gland unless there is abscess formation and facial nerve palsy. The surgical technique involves elevating a standard parotidectomy flap with careful dissection of the facial nerve fibres. After elevation of a skin flap superficial to the parotid fascia, a haemostat is introduced to make multiple openings into the parotid gland and is spread in the direction of the facial nerve branches. An attempt should be made to identify the facial nerve by standard methods or by using facial nerve stimulator. However, with a fulminant necrotizing process, debridement of all areas of necrosis may compromise full return of facial nerve function (4,9) as occurred in this case.

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