CASE REPORT

# Cervical Necrotising Fasciitis Consequent to Mastoid Infection

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**Summary**

We present a case of cervical necrotising fasciitis in a 56 year old man, secondary to a rare mastoid infection. The patient had coexisting diabetes mellitus and hypertension. He was treated with early surgical debridement followed by neck and chest reconstruction and radical mastoidectomy. Aggressive antibiotic therapy and supportive care was given. He recovered well with minimal residual functional deficit.

*Key Words:* Necrotising fasciitis, Neck, Mastoiditis

## Introduction

Necrotising fasciitis is a severe soft tissue infection characterised by extensive necrosis of superficial fascia, widespread undermining of surrounding tissues and extreme systemic toxicity. The disease usually progresses rapidly to diffusely involve adjacent fascial spaces with resultant non-viable skin[1]. Most cases are caused by synergistic infections with both aerobic and anaerobic organisms.

In this report we highlight a case of cervical necrotising fasciitis which developed from acute on chronic suppurative otitis media in a diabetic patient.

## Case Report

In December 2000, a 56 year old police inspector was referred from Medan, Indonesia, to another private hospital in Penang and was subsequently referred to the first author and admitted for further management. He complained of fever of one months' duration and right neck swelling extending from the mastoid region down to the upper chest, with associated purulent ear discharge. In November 2000, he had been admitted under the care of an ENT Specialist in Medan and had been treated conservatively for a right mastoid abscess with ceftriaxone, clindamycin and metronidazole. He is a chronic diabetic and hypertensive. On examination, he looked toxic but was alert with a temperature of 38°C. There was diffuse erythema and non-fluctuant swelling of the right neck extending down to the upper chest with a huge retro-mastoid abscess cavity/fistula containing black offensive non-viable tissue (Fig. 1).

Laboratory investigations revealed haemoglobin of 12.1g/dl and total white count 11.5 x $10^3/\mu l$ with a shift to the left. Random blood glucose was 15.4mmol/l, and renal profile, liver function tests,

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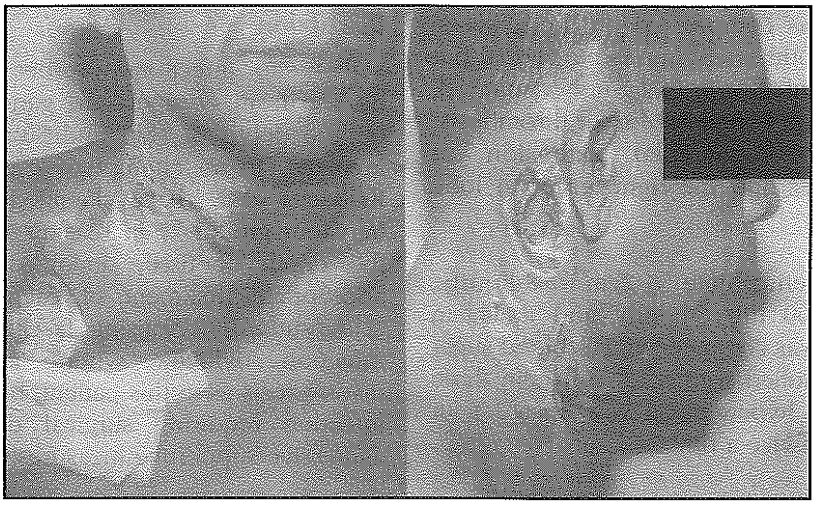

**Fig. 1: Cervical necrotising fasciitis extending from the right mastoid region to the neck and upper chest wall.**

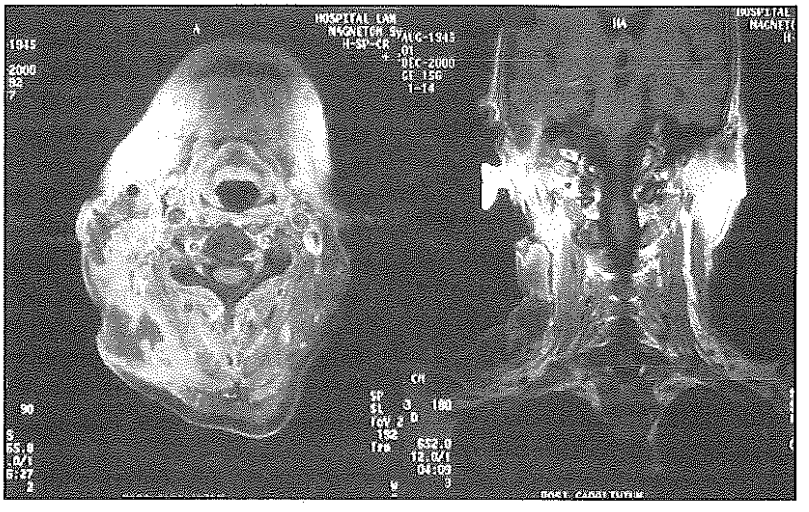

**Fig. 2: MRI scan showing right mastoiditis and deep abscess extending down to the neck.**

urinalysis, electrocardiogram and chest x-ray were normal. Right ear swab cultured a heavy growth of *Actinetobacter anitratus* resistant to ampicillin, cotrimoxazole, gentamicin, ofloxacin and cefprozil but sensitive to ampicillin/sulbactam and cefepime.

A magnetic resonance imaging scan (Fig. 2) revealed right mastoiditis and deep ulcerative abscess extending from the right retro-mastoid region with extension into the sternocleidomastoid muscle and tracking down to the root of the neck and chest wall. The right

carotid space was compressed by the abscess; the common carotid artery appeared normal but the internal jugular vein was not visualised, probably because of compression or thrombosis. There was no intracranial extension or extradural abscess and the right temporal lobe looked normal.

Intravenous ceftazidime 2gm 8-hourly, metronidazole 500 mg 8-hourly and amikacin 250mg 8-hourly was commenced. His blood sugar levels were controlled with soluble insulin three times a day and hypertension was treated with nifedipine 20mg twice a day.

The day following admission, he underwent neck exploration under general anaesthesia with wide excision of necrotic skin, fat, fascia and neck muscle. Post-operatively he made a good recovery and had daily debridement of necrotic tissues under sedation and analgesia, with hydrogen peroxide dressing and eusol packing three times a day. He was given good nutritional support with a high protein, high calorie diet and required blood transfusions. On this treatment regimen, the wound granulated well.

Two weeks later, he underwent left cervical transpositional flap, right axilla-pectoral transpositional flap and split skin graft under general anaesthesia to cover the huge neck/chest defect. Post-operatively recovery was good with more than 90% closure of the defect. A week later, he had right radical mastoidectomy and facial transpositional flap in order to reduce the retro-mastoid defect. The post-operative recovery was good and patient was discharged looking well one month post-admission.

On subsequent review two weeks later, the patient had well healed mastoid cavity and neck wound with a minimal functional deficit; there was a residual 50 decibel conductive hearing loss.

## Discussion

Soft tissue infections present a spectrum of disease from erysipelas to gangrene depending on the depth of skin and subcutaneous tissue involved. In an attempt to clarify the situation Kalbacha *et al*[2] have classified these infections into four groups of increasing severity based on the depth of tissue involved and suggested appropriate treatment of each group. In this classification, necrotising fasciitis is a Type III infection which, when associated with myonecrosis as in this patient, becomes a type IV infection requiring surgical debridement and aggressive supportive measures.

Necrotising fasciitis most commonly affects the trunk, perineum and limbs, but head and neck involvement can occur[3]. Dental infection is the commonest aetiology in cervical necrotising fasciitis; tonsillar and skin infection, and facial trauma are other causes. The infection may rapidly extend to the cervical viscera, mediastinum, and anterior chest wall. Chronic suppurative otitis media with mastoiditis is a very rare cause of this condition and no previous reports have been found. In a recent review, the overall mortality for 48 cases of cervical necrotising fasciitis was 30%[1]. Thirty percent of these cases had diabetes mellitus and this group had a higher mortality rate of 43%. Another 10% had ischaemic heart disease, carcinoma or were drug abusers. The remaining 60% of cases occurred in those with no associated disease or evidence of immune incompetence.

Our patient presented with a subacute form of this disease probably because he had prior antibiotic treatment. Although the fulminant form is the best recognised entity, it has been reported that necrotising fasciitis represents a spectrum of disease which can range from fulminant through acute to the subacute varieties[4]. Furthermore, the organism isolated, *Acinetobacter anitratus*, was most likely not the primary causative pathogen but the result of nosocomial infection.

Radical surgical debridement is the cornerstone of treatment and must be complemented with intensive antimicrobial administration, supportive care and treatment of comorbid conditions to achieve a good outcome.

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