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Craniofacial necrotizing fasciitis secondary to sinusitis

EDUARDO RABOSO, M.D., MARIA T. LLAVERO, M.D., ANTONIO ROSELL, M.D.,
ANTONIO MARTINEZ-VIDAL, M.D., PH.D.

Abstract
Necrotizing fasciitis is a rare condition which usually affects the trunk, perineum and limbs. Head and neck involvement is very uncommon and in most cases it is secondary to orbital or dental infection. We report a case of craniofacial necrotizing fasciitis (CNF) secondary to a maxillary sinusitis. The patient was treated intensively with antibiotics, surgical procedures and life-support measures. Despite all efforts, the patient died one week after admission. This case highlights early diagnosis and aggressive management as decisive factors for the outcome of the patient.

Key words: Necrosis; Fasciitis; Sinusitis

Introduction

Necrotizing fasciitis is a potentially lethal infection which is even more dangerous when it affects the craniofacial territory. Craniofacial involvement is very rare and is usually secondary to orbitary or oral infections (Banerjee *et al.*, 1996). This condition requires early diagnosis and aggressive treatment. We present what seems to be the first case of necrotizing fasciititis secondary to a sinus infection. This paper illustrates the difficulties found in the diagnosis and management of these patients.

Case report

A 79-year-old non-insulin dependent diabetic male arrived at our Emergency Department complaining of rapidly progressive facial swelling after the ingestion of two doses of 200 mg of acetylcistein. Physical examination revealed a widespread non-crepitant facial oedema in a patient with a deteriorated cardiovascular condition. The initial diagnosis was anaphylactic reaction and the patient was treated accordingly. The lack of response to the treatment led to a re-evaluation of the patient and the suspicion of cellulitis. Dental infection was excluded because the patient had been edentulous for many years. A sinus X-ray film showed an opacification of the left maxillary sinus which was confirmed with a computed tomography (CT) scan (Figure 1) and a course of intravenous imipenem and vancomycin was commenced. One day after admission the patient had worsened even further with heart and renal failure requiring ventilation, and the swelling had developed to the oral mucosa and the neck. Necrotic patches and blisters began to appear at the eyelids and other facial areas (Figure 2). The left maxillary sinus was punctured through the nasal fossa and 5 ml of pus was drained. Gram staining of the pus demonstrated Gram positive cocci and Gram negative bacilli which were later cultured as β-haemolytic *Streptococcus* group A and *Haemophilus influenzae*, both sensitive to the antibiotics

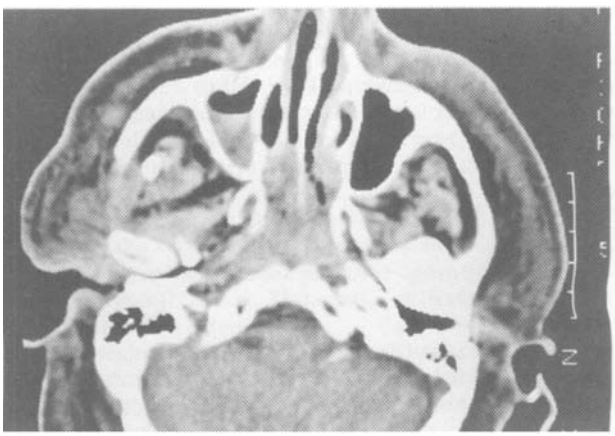

FIG. 1
Axial section of the CT scan showing a left maxillary sinusitis. Note the facial soft tissue oedema.

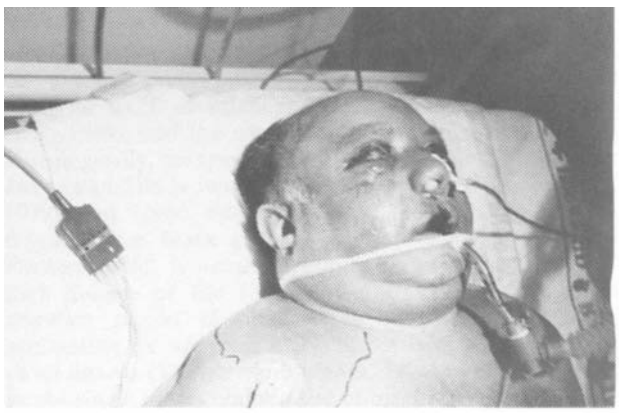

FIG. 2
Photograph of the patient after the sinus punction. Notice the facial oedema with eyelid and lip necrosis.

From the ENT Department, Hospital Ramon y Cajal, Madrid, Spain.
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used. Cultures for anaerobes were negative. A surgical debridement of the necrotic areas was performed and the histopathology examination of the tissues excised indicated numerous Gram positive cocci in the subcutaneous tissues and in the muscles. Cultures of the necrotic tissues grew β-haemolytic *Streptococcus* group A, *Haemophilus influenzae*, *Streptococcus pyogenes* and *Staphylococcus aureus*. Despite debridement and intensive antibiotic therapy the patient died one week after the onset of the symptoms.

Discussion

To the best of our knowledge this is the first description of CNF as a complication of a sinusitis. CNF is a rare entity which has been related mainly to dental infection but also to orbital disease, trauma and peritonsillar abscess (Banerjee *et al.*, 1996). Alcoholism and diabetes are considered risk factors and poor prognosis indicators (Umbert and Winkelman, 1989; Asfar *et al.*, 1991; Overholt *et al.*, 1991).

The diagnosis of CNF is clinical and, as in our case, it is frequently mistaken for facial cellulitis or oedema (Klabacha *et al.*, 1982), but as the disease progresses, the skin shows bluish patches and blisters due to the thrombosis of its vessels leading to skin necrosis. Crepitance is also common. Lethargy, hyperpyrexia, tachypnoea, tachycardia and, eventually, septic shock are also associated features often seen in CNF (Spankus *et al.*, 1984).

If the diagnosis is not clear or if some assessment is needed for the surgical debridement a CT scan is the imaging modality of choice. It provides information about the extent of the disease, the presence of gas, the initial site of infection and anatomical data to guide the debridement (Henrich *et al.*, 1996).

CNF are usually polymicrobial infections, the most common pathogens are *Streptococcus* spp., but *Staphylococcus aureus* and anaerobes are also present in a large proportion of cases and a synergistic activity of aerobic and anaerobic organisms has been suggested (Chow *et al.*, 1978). *Bacteroides melaninogenicus* seems to be the most frequent anaerobe in CNF (Dormer and Babett, 1972).

The management of CNF requires early diagnosis and immediate endovenous therapy with broad-spectrum antibiotics combined with aggressive surgical debridement with extensive excision of the necrotic tissues and leaving, if necessary, the skin flaps open to allow inspection and drainage of the fascial planes. In our experience, the extension of the debridement required can be extremely mutilating and could eventually affect the quality of life of the patient in case of survival. Constant monitoring of the haemodynamic status and haematological and renal function parameters should be considered essential (Nallathambi *et al.*, 1987).

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Address for correspondence:
E. Raboso,
Departamento de ORL,
Hospital Ramon y Cajal,
Carretera de Colmenar Viejo Km 9.1,
28034 Madrid,
Spain.

Fax: 34-1-3368733
e-mail: pantoja@anit.es

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