# Synergistic necrotizing cellulitis resulting from peri-tonsillar abscess

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## Abstract

This case demonstrates the rare but potentially fatal condition of synergistic necrotizing cellulitis of the head and neck. Although similar to necrotizing fasciitis, this is more extreme as it also destroys muscle. Peri-tonsillar abscess is a recognized cause, but has not previously been described in this country, however it is a condition commonly referred to otolaryngologists and awareness of the complication of synergistic necrotizing cellulitis is necessary to allow early recognition and prompt treatment. This should be by broad-spectrum intravenous antibiotics with extensive surgical debridement and drainage procedures repeated as necessary. Although this case had a successful outcome, many patients do not survive, particularly if treatment is delayed or inadequate.

Key words: Peritonsillar abscess; Cellulitis, synergistic necrotizing; Fasciitis, necrotizing

### Introduction

Peri-tonsillar abscess is a common condition, and deep tissue spread of infection should be considered if the patient fails to respond to conventional treatment, or develops rapidly enlarging cervico-facial swelling. A diagnosis of synergistic necrotizing cellulitis can be confirmed by the operative appearance of blackened non-viable muscle, this differentiates it from necrotizing fasciitis. There have been no previous reports of cervicofacial synergistic necrotizing cellulitis from this country. Of the few cases described abroad only two resulted from a peritonsillar abscess (Wenig et al., 1984; Greinwald et al., 1995). The complication of synergistic necrotizing cellulitis is more common in immunocompromised, diabetic and malnourished patients, however this case occurred in a previously fit and healthy individual. Early recognition, appropriate intravenous broad-spectrum antibiotic therapy, extensive surgical debridement and supportive measures are essential to avoid the otherwise high incidence of mortality.

# **Clinical history**

A fifty-three-year-old English man working in Russia presented with a sore throat. He was flown back to England by Emergency Air Ambulance with a provisional diagnosis of diphtheria. On admission to the Infectious Disease Unit he was found to have a left peri-tonsillar abscess. Several attempts had apparently been made to drain this in St Petersburg. Despite further incision of the quinsy under local anaesthesia and intravenous antibiotic therapy with cefotaxime and metronidazole, there was no symptomatic improvement and the patient developed a large left-sided neck swelling with trismus. The cervical swelling enlarged to involve the left cheek and both sides of the neck, the trismus increased and the patient became pyrexial with a neutrophil leucocytosis. The cervicofacial swelling was tense and tender with no definite margins and the overlying skin was erythematous. Ultrasound scanning identified a homogeneous mass, but suggested that it was solid.

The swelling was assumed clinically to be a parapharyngeal abscess secondary to a peritonsillar abscess, and exploration under anaesthetic was performed. The left tonsil was necrotic and pus exuded from the parapharyngeal space into the tonsillar bed. The affected tonsil was excised and the neck explored via an external cervical incision, revealing multiple pockets of foul-smelling pus. These were opened with blunt finger-dissection and irrigated, several drains being inserted. Microbiological swabs grew mixed Gram-positive and Gram-negative aerobic and anaerobic flora including *Streptococcus milleri* (Lancefield group G antigens) and two species of *Bacteroides*. On the advice of the microbiology department the antibiotic regime was changed to imipenem, benzyl penicillin, metronidazole and gentamicin.

On this regime the patient became apyrexial and the neutrophil leucocytosis resolved. There were no features suggesting that the patient was immunocompromised, and although an HIV test was performed this was negative.

The cervicofacial swelling persisted and copious amounts of offensive-smelling pus continued to drain from the neck (Figures 1 and 2). Four further general anaesthetics were needed for debridement of necrotic muscle and deep tissues, and excision of the overlying skin of the neck and left facial area to allow open packing of the wound. The excision eventually extended from skull base on one side across to the other, leaving the laryngeal cartilages, right parotid and submandibular glands completely exposed. Drainage tracts extended medial and lateral to the mandible, and posterolateral to the pharynx (Figures 3 and 4).

The patient was fed by nasogastric tube and a polythene

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Fig. 2

Persistent cervico-facial swelling after initial exploration and drainage.

'oxygen tent' was used around the surgically exposed wound to ensure a high oxygen delivery to the tissues.

FIG. 1

Each anaesthetic induction was complicated by trismus, and fibre-optic awake intubation was initially necessary; eventually the patient was maintained on nasotracheal intubation and ventilation with sedation for six days.

Two weeks after the initial surgical debridement with resolution of the infection the exposed neck was covered by skin grafting. The patient continued to make a good recovery and has since been discharged home. He is currently fit and healthy.

## Discussion

Infection of deep soft tissues may spread along fascial planes involving subcutaneous tissue and overlying skin. Synergistic necrotizing cellulitis can be distinguished from the original description of necrotizing fasciitis (Wilson, 1952) by the additional involvement of skeletal muscle which appears blackened and non-viable at surgery (Rippe, 1991). The chance of survival is reduced if more than one muscle compartment is involved (Stone and Martin, 1972).

Most cervical deep tissue infections are due to dental (63 per cent) (Spankus *et al.*, 1984), pharyngeal (Gaukroger, 1992) or traumatic causes (Herr *et al.*, 1991; Morrow and Gianoli, 1993; Palchum *et al.*, 1993; Maisel and Karlen, 1994). In this case the focus of infection was a peritonsillar abscess. Although this may be the commonest deep abscess of the head and neck (Smith and Hemingway, 1978), it rarely progresses to deep soft tissue involvement.

Deep soft tissue infections of the head and neck have received little attention in the literature although these

sites are associated with significant morbidity. Mortality of up to 73 per cent has been reported with necrotizing fasciitis (Rouse et al., 1982). Over the past 30 years there have only been 62 reported cases of cervicofacial necrotizing fasciitis (Wilson, 1952; Wills and Vernon, 1981; Spankus et al., 1984; Wenig et al., 1984; Balcerak et al., 1988; Kronish and McLeish, 1991; Lalwani and Kaplan, 1991; Williams et al., 1992; Greinwald et al., 1995; Kaplan et al., 1995) seven of which were caused by peritonsillar abscess (Wills and Vernon, 1981; Spankus et al., 1984; Wenig et al., 1984; Lalwani and Kaplan, 1991; Tovi et al., 1991; Greinwald et al., 1995). Only two of these had documented muscle involvement (Wenig et al., 1984; Greinwald et al., 1995) thereby fulfilling the criteria of synergistic necrotizing cellulitis. There have been no previous reports of cervicofacial synergistic necrotizing cellulitis from the UK, and only four of cervicofacial necrotizing fasciitis, (Gaukroger, 1992; Williams et al, 1992) none of which occurred as a result of peritonsillar abscess.

The clinical presentation is usually of a toxic patient with a high fever and severe pain (Ward and Walsh, 1991), rapidly progressive non-fluctuant swelling of the face and neck, purplish skin discolouration, vesicle formation (Balcerak *et al.*, 1988), surgical emphysema and a scanty brown offensive discharge which drains from any wounds. Underlying systemic disorders include diabetes, ischaemic heart disease, immunosuppression, poor nutrition and alcoholism (Kronish and McLeish, 1991; Ward and Walsh, 1991). However no risk factors need be present (Burge, 1995) as in the case described here where the patient was otherwise healthy prior to development of tonsillitis.

#### CLINICAL RECORDS



Fig. 3

FIG. 4

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Eventual extent of resection of necrotic tissue and overlying skin.

The microbiological flora in this case were similar to the mixed growth described in other published series from around the world (Zakharov and Orlova, 1990; Williams et al., 1992; Morrow and Gianoli, 1993; Maisel and Karlen, 1994). It appears that cases of synergistic necrotizing cellulitis are not caused by a single pathogenic organism, but by the synergistic effect of several bacteria, which are usually relatively innocuous. The bacteria responsible for deep cervico-facial infections often include a haemolytic streptococcus and an anaerobe (Morrow and Gianoli, 1993). A neutrophil leucocytosis is typical and was present in this case, occasionally hypocalcaemia occurs as a result of liquefactive necrosis of fat (Wilson, 1952). Subcutaneous gas may be present even in the absence of clostridial organisms. This may be detected by plain radiographs or ultrasound scan, although CT scan is probably the best modality for differentiating gas bubbles in the deep cervico-facial spaces in addition to cellulitis, oedema and abscesses (Yamaoka et al., 1994; Kaplan et al., 1995). In this case ultrasound scan was used, but misleadingly suggested the neck abscess was a solid mass.

As this condition is rare, and deep space infection can be occult (Herr et al., 1991), a high index of clinical suspicion is necessary. Early diagnosis and treatment greatly reduces the morbidity and mortality (Peled et al., 1994). Microbiological specimens must be taken and, until Gram stain cultures and sensitivities are known, intravenous antibiotic therapy should cover Gram-negative, aerobic and anaerobic flora. Examination under anaesthetic is needed for adequate incision and drainage of abscesses with debridement of necrotic tissue. Histological changes are seen in apparently healthy tissue specimens taken from beyond the macroscopically affected areas. Even if frozen tissue sections are taken the area resected is often insufficient. Excision and debridement may have to be repeated several times and the area left exposed until the infection has resolved, when skin grafts or flaps will be needed for reconstruction (Herr et al., 1991; Palchun et al., 1993; Yamaoka et al., 1994).

Wound care is necessary at least twice daily, this also enables detection of new foci of infection (Tovi *et al.*, 1991). Hyperbaric oxygen therapy is thought to be beneficial (Kronish and McLeish, 1991; Kurakin *et al.*, 1993; Peled *et al.*, 1994), in this case oxygen was supplied into a small polythene 'tent' around the area of surgical exposure. Enteral feeding support is vital due to catabolic changes as a result of septicaemia. Mortality is usually due to multisystem organ failure and overwhelming sepsis, particularly if mediastinitis occurs, or treatment is delayed (Spankus *et al.*, 1984; Tovi *et al.*, 1991).

# Conclusion

Peritonsillar abscess is a common condition, and although synergistic necrotizing cellulitis is a rare complication, it is vital to recognize it early. It may be suspected if the patient fails to respond to conventional therapy, pain is disproportionately severe, the general condition deteriorates rapidly, or subcutaneous emphysema is present. Therapy depends on early intravenous broad-spectrum antibiotics (before culture results available), extensive surgical debridement and supportive measures. The outcome is poor, and devastating morbidity or mortality can only be avoided if early recognition and prompt treatment are provided.

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