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Cervicofacial necrotizing fasciitis: an unusual complication of chronic suppurative otitis media

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Abstract

Necrotizing fasciitis is a rare microbial soft tissue infection characterized by rapidly spreading areas of necrosis and a high mortality rate. It may be of odontogenic or traumatic origin or may arise from insect bites, burns or surgical infections. We present a clinical case of an eight-year-old child with facial and cervical necrotizing fasciitis as a complication of chronic suppurative otitis media. The causes, diagnosis and management of necrotizing fasciitis are reviewed.

Key words: Fasciitis, Necrotizing; Otitis Media; Cholesteatoma; Middle Ear

Introduction

Necrotizing fasciitis is an invasive infection usually caused by multiple pathogens. It is characterized by infectious thrombosis of the blood vessels between the skin and the deep circulation, producing skin necrosis resembling ischaemic vascular or clostridial damage, although sometimes the bacterial flora is mixed.[1] The areas most frequently involved are the thorax, limbs, perineum, abdomen and groin.[2–4] Involvement of the head and neck is less common; in this region, the usual nidus of infection is the teeth.[5] Other reported causes include peritonsillar abscess, cutaneous infections, insect bites and trauma to the eyelids.[6,7] In the literature, there have only been two reports of necrotizing fasciitis that originated in the ear.[8,9] The first patient had osteoradionecrosis of the temporal bone after receiving external beam radiation for squamous cell carcinoma. The second patient developed cervical necrotizing fasciitis secondary to perichondritis of the pinna. No case of cervicofacial necrotizing fasciitis as a complication of otitis media has been reported to date.

Necrotizing fasciitis is very rare in the paediatric age group,[10] with a high mortality, reported at between 9 and 31 per cent.[11] We report a child who survived such an infection and discuss the case, emphasizing the role of early, aggressive surgical debridement of necrotic tissue, combined with appropriate parenteral antibiotic therapy and maintenance of nutrition.

Case report

An eight-year-old boy was brought to the emergency department in August 2002 with a three-month history of bilateral ear discharge. The discharge was purulent and foul-smelling and had been associated with otalgia. Over the previous three days, the patient had developed rapidly progressive swelling of the head, face and neck, which had originated in the region of the left pinna. He had been given but had been semiconscious, with intermittent, high-grade fever, for the previous 24 hours. There was no history suggestive of any dental infection, throat infection or trauma.

On examination, the child was semiconscious and responding to painful stimuli only. He had cellulitis involving the anterior half of the scalp, left temporal region, both eyelids, cheeks and upper neck. A profuse, foul-smelling, dirty brown coloured, watery discharge was seen issuing from the left ear, with bogginess of the skin of the left external auditory canal and sagging of part of the posterosuperior canal wall. The tympanic membranes could not be visualized initially, although a later examination showed posterior epitympanic cholesteatomas in both ears. The child was put on intravenous antibiotics in the form of crystalline penicillin, gentamicin and metronidazole and was also given intravenous fluids. A tracheotomy was undertaken in view of the extensive head and upper neck swelling, and drainage of fluid collections in the left supra-aural and submandibular regions was performed. The ear discharge was sampled and sent for culture, and a computed tomography (CT) examination of the head and neck was performed, which showed no intracranial lesion and subcutaneous cellulitis with no involvement of the deep neck spaces.

Within 48 hours, localized areas of necrosis of skin, subcutaneous tissue and muscle had developed over the left temporal and submental regions (Figure 1). These areas progressed to involve two-thirds of the scalp and neck over the next four days. The general condition of the child remained poor but stable.

The condition of the child worsened, with increasing areas of necrosis, and culture of the ear discharge showed mixed organisms, so the antibiotics were changed on the third day to co-amoxyclav, amikacin, metronidazole and ofloxacin, in consultation with the microbiologists. There was no positive culture at this time. Meticulous care was given to the wound, with debridement and dressings two to three times a day. A high-protein, high-calorie diet was started via nasogastric tube on day two.

The child's condition slowly improved and by three weeks he was non-toxic but had wounds on the scalp and

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FIG. 1
Extent of necrotizing fasciitis on presentation.

FIG. 2
Patient's condition after 12 weeks of treatment.

neck. A large area of the skull was exposed without any soft tissue cover and a large area of the neck had skin loss with the underlying muscles exposed. Over the next four weeks, the wounds started to granulate and over the next three to four months the neck wounds closed but the scalp wound did not close completely (Figure 2). The child developed progressive dyspnoea which was found to be due to a tracheal stenosis, and this was managed by stenting the trachea over a flexometallic endotracheal tube cut to size. The child was referred to a cardio-thoracic surgeon for management of the tracheal stenosis.

Discussion

Necrotizing fasciitis was first described in 1871 by Jones[12] during the American civil war and later by Meleny in 1924.[13] It is a severe soft tissue infection that is disseminated through the fascial planes causing thrombosis in the affected blood vessels. The skin is devascularized, with external discoloration and haemorrhagic bullae. The microbiology influences the severity of the infection, as does the host's underlying condition. Generally, the condition is caused by a synergistic bacterial infection, usually Streptococcus and some combination of other gram-positive cocci, gram-negative rods and anaerobic organisms.[14] The incidence of positive blood cultures ranges from 25 per cent to 100 per cent.[11] Since the infection is usually synergistic, with a broad range of bacteria being possibly involved, no specific chemotherapeutic regime or protocol has evolved.

The risk factors that contribute to the establishment of necrotizing fasciitis in adults include diabetes mellitus, atherosclerosis, peripheral vascular disease and age greater than 50 years.[15] These risk factors are not commonly encountered in children, in whom the predisposing conditions differ; they include immunodeficiency states, particularly neutropenic states following chemotherapy.[16,17] The only predisposing condition in our patient was a subperiosteal abscess as a result of unsafe suppurative otitis media.

We have not found in the literature any previous report of necrotizing fasciitis as a complication of unsafe otitis media. Our patient had a bulge in the posterosuperior canal wall along with bogginess of the skin of the external auditory canal, representing a subperiosteal abscess with associated soft tissue cellulitis of the external auditory canal. The nidus of infection in this case seems to have been the subperiosteal abscess, with spread of the infectious process to the surrounding soft tissues and resultant cellulitis and rapidly progressive necrotizing fasciitis. The three classical signs of necrotizing fasciitis – high-grade fever, wound crepitus and subcutaneous gas – were absent in our patient and are rarely reported in the paediatric literature.[18]

Necrotizing fasciitis cannot be successfully treated with antibiotics alone, and aggressive, prompt surgical management is essential.[5,6] Fascial involvement frequently extends beyond the area of skin involvement, and it may be necessary for the patient to have repeated debridement if the infection continues to spread. Our patient was managed with drainage of fluid collections and debridement; however, the difficulties faced included a severe, rapidly spreading head and neck infection; a toxic, paediatric patient; no positive culture reports; and extensive skin necrosis, with large areas of exposed skull and neck soft tissue. These problems presented significant management challenges.

In conclusion, it would seem that the ear is an unusual source for cervicofacial necrotizing fasciitis; however, although such infections are rare in children they should

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be borne in mind, particularly in the immunocompromized and in patients with marked toxicity and progressive, subcutaneous oedema. A high index of suspicion, with early recognition, antibiotics and supportive therapy, are essential if such patients are to survive.

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