CASE REPORT

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NECROTIZING FASCIITIS OF THE HEAD AND NECK:
A REPORT OF TWO PATIENTS AND REVIEW

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Abstract: Background. Necrotizing fasciitis is a disfiguring condition that can be fatal. The head and neck region is rarely affected. However, when involved, the functional and cosmetic sequelae can be considerable.

Materials and Methods. We present two case histories, discuss salient diagnostic points, treatment, and review published data on this topic.

Results. With a timely diagnosis we were able to diagnose and appropriately treat these patients.

Conclusions. Necrotizing fasciitis is a disfiguring condition that can be fatal if not diagnosed in a timely fashion. Diagnosis and treatment require a high index of suspicion, immediate operative intervention, broad-spectrum antibiotics, and appropriate supportive care. © 2002 Wiley Periodicals, Inc. Head Neck 24: 497–501, 2002

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Necrotizing fasciitis was first described during the American Civil War. It is a rare and rapidly spreading superficial infection involving the superficial fat and fascial layers with necrosis of the overlying skin. Muscle involvement, however, is unusual or minimal. Regions of the body frequently involved include the extremities, genitalia, and trunk. Cases affecting the head and neck region are unusual. Diabetic and alcoholic patients, in particular, and other immunocompromised patients are at risk for this condition.

On presentation patients may report a history of minor trauma or a recent surgical procedure 2 to 4 days preceding the full-blown condition. Initially, the area involved is usually edematous and erythematous, which may lead the clinician to misdiagnose the condition as a skin infection. However, skin necrosis usually develops late in the course. Without aggressive medical and surgical intervention, the patient usually becomes toxic, frequently requiring critical care support, with the potential for severe cosmetic deformity and death if not treated appropriately in a timely manner.

We report two cases of necrotizing fasciitis of the craniocervical region with survival and review the clinical presentation, diagnosis, and treatment of this condition.
CASE 1
A 36-year-old man with medical history significant only for polysubstance abuse was brought to our trauma center after being found in the face-down position. It was believed that the patient had been physically assaulted and had been lying on his face for several hours. On presentation the patient was confused, disoriented, and unable to recall any events preceding his arrival at the hospital. His vital signs were a temperature of 36.9°C; HR, 135; BP, 104/45; and RR, 34. The physical examination was only significant for massive right-sided facial, neck, and bilateral periorbital edema, erythema, and sloughing of the skin in these areas. The right orbit was noted to contain a corneal abrasion. His laboratory tests demonstrated a WBC of 2.4, and a BUN/Cr of 44/3.8. Radiographic studies of the head and neck only demonstrated soft tissue swelling. A diagnosis of craniocervical necrotizing faciitis was made, and penicillin G, cefotaxime, and clindamycin were begun immediately. The patients respiratory and ventilatory status decompensated and he required intubation in the trauma bay; furthermore, the patient went into renal failure. Thus, the patient was taken to the intensive care unit for appropriate resuscitation.

Once stabilized, the patient was brought to the operating room, where extensive wide surgical debridement of the craniocervical region and a tracheostomy were performed. Intraoperative cultures grew group A beta-hemolytic streptococcus, with subsequent adjustments in his antibiotics. The patient subsequently underwent a total of six operative debridements and split-thickness skin grafts, one full-thickness skin graft to his right periorbital region, a scalp advancement flap, and enucleation of the right eye.

He had a prolonged course of mechanical ventilation requiring a tracheostomy. The patient was able to be withdrawn from pulmonary support, and his tracheostomy was eventually removed. The patient requested that he be transferred to another hospital that was more convenient for his family. He was lost to follow-up.

CASE 2
A 49-year-old woman with a medical history significant for a partial gastrectomy secondary to bleeding and an idiopathic tachyarrhythmia noticed a small erythematous indurated area under her left eye 4 days before admission. Seventy-two hours before admission the erythematous area spread to the other eye. She went to her primary care physician who made a presumptive diagnosis of lupus and placed the patient on topical steroids. The erythema did not improve, and 2 days before admission the patient went to the physician again, who placed the patient on oral steroids. The redness worsened on the day before admission, and she developed a temperature of 38.9°C; on the day of admission the patient woke up with increased edema, pustules, and black patches on the right side of her face, as well as nausea, vomiting, and anorexia. The patient was not able to recall any recent trauma or oral infections.

On presentation her vital signs were 36.9°C, BP, 105/76; HR, 72; with a respiratory rate of 24. The right side of her face was swollen with areas of patchy necrosis from the eye down to the neck just above the supraventricular area (Figure 1). The left eye was also swollen. There were no fluctuant areas appreciated. She began to have respiratory distress, necessitating pulmonary support and renal insufficiency.

Laboratory examinations demonstrated a WBC of 3.9, Bun/Cr of 83/4.6, and PT/PTT of 17.0/
36.9. A diagnosis of necrotizing fasciitis was made, and she was begun on ampicillin sulbactam vancomycin, and ceftriaxone and admitted to the intensive care unit for resuscitation. After resuscitation, she was taken to the operating room for extensive debridement, a tracheostomy, and cultures were taken. Intraoperative findings demonstrated dermal vessel thrombosis and extensive necrosis of the subcutaneous fat and fascia. Cultures demonstrated group A streptococcus, and she was thus placed on penicillin G. The patient eventually underwent a radial arm free flap, forehead myocutaneous advancement flap, and two revisions of these flaps. In all, she underwent nine debridement procedures, four full-thickness skin grafts, and a tracheostomy. She was able to wean off of the ventilator, and more than a year and half later she is doing well (Figure 2).

**DISCUSSION**

Necrotizing craniocervical fasciitis of the head and neck is a rapidly spreading and potentially fatal infection initially involving the superficial musculoaponeurotic system and superficial fascial planes of the head and neck. This fascial plane envelopes the muscles of facial expression in a sheath from the posterior aspect of the frontalis muscle to the platysma. When these structures are destroyed by necrotizing infections, it is easy to see why the deformity that results can be cosmetically and functionally devastating. As the infection spreads, the deep cervical fascial layers may become involved with the infection extending into the mediastinum and subsequently leading to respiratory and neurologic complications. When the cervicofacial region is involved, the origin of the infection will be oral.7-8

Group A and non-A streptococcus and staphylococcus are normal flora found in the human oropharynx and skin. These organisms are frequently identified in necrotizing fasciitis of the face, and they may be found synergistically with each other and with other organisms in infected wounds. It has been reported that streptokinase and staphylokinase activate proteolytic enzymes (collagenases and or hyaluronidase) in the subcutaneous tissue leading to collagen necrosis.9 In addition, the mucoprotein fraction of streptococcus group A’s cell wall has been shown to combine with dermal collagen.10 Cromartie et al injected mucoproteins into dermal cells and were able to demonstrate that dermal necrosis occurred.11 As the ground substance of the papillary dermis is broken down, the infection and necrotic process spreads to the epidermis and subcutaneous fat. The infection initially affects the subcutaneous tissue just superficial to the deep fascia, leading to fascial necrosis and rapid extensive subcutaneous destruction, which is an ideal culture medium for bacterial growth. Skin, usually spared initially, becomes gangrenous with time secondary to thrombosis of the perforating vessels that supply it. If not treated promptly, the area of necrosis will quickly enlarge.

Early diagnosis is imperative for successful therapy and minimization of cosmetic deformity. The diagnosis of necrotizing fasciitis is a clinical one, and a high index of suspicion is required. During the early stages of the illness (first 36 h) physical findings may be minimal, which may make it difficult to distinguish necrotizing fasciitis from any other wound infection. Patients may complain of pain, whereas the physical examination may only demonstrate some edema and erythema. However, this infection spreads with alarming rapidity, and an entire region may

**FIGURE 2.** Patient in case 2, 1 year after reconstruction.
become involved within 36 hours. During the second to fourth days bluish black patches may appear on the skin. Dark brown–filled bullae may be seen on the skin. Once these have developed, frank gangrene frequently follows. With cell death and the release of inflammatory mediators, patients frequently become septic. Clinically, patients may have mental status changes, multisystem organ failure, and hemodynamic lability. The elevated white count or leukopenia as demonstrated by our patients has been seen by others. The leukopenia may be secondary to sequestration of white blood cells within the spleen and lymphatic system, as well as marrow inhibition. Additional laboratory findings include hypocalcemia secondary to deposition in necrotic tissues. Blood cultures and wound cultures are of utmost importance, because they may influence the therapeutic antibiotic regimen. The usefulness of radiographic studies in helping with the diagnosis is unclear. Fisher et al.12 and Yamoka et al.13 have used plain films to aid with the diagnosis by detecting gas. However, Shindo et al believe there is no value in plain films.14 Several groups have used CT to assist with the diagnosis when plain films were not helpful.13–15 CT is believed to be beneficial when the diagnosis is not straightforward; it can provide information on the extent of the disease and possibly localize the initial site of infection, while providing anatomic information that can aid with the surgical intervention. Others have used frozen section to aid with the diagnosis.16 However, this may be difficult for certain parts of the face (i.e. eyelids).

Once the diagnosis has been made, it is important that broad-spectrum intravenous antibiotics begin. The regimen should include a penicillinase-resistant penicillin for streptococcal and staphylococcal bacteria and an aminoglycoside for gram-negative coverage. Anaerobic coverage can be provided with clindamycin or metronidazole. The cornerstone of therapy, however, is surgery. Surgical incisions are made through the discolored skin down to the fascia parallel to the cutaneous nerves and blood vessels. The amount of debridement can be estimated by cutting to tissue that bleeds and passing a gloved finger above the superficial musculoaponeurotic system of the face; when inability to pass the finger is encountered, further debridement is not necessary. Failure to achieve satisfactory drainage and debridement leads to further spread of the necrotizing process to adjoining areas, requiring additional surgical intervention. Foul, thin murky fluid will frequently be encountered. The wounds are left open, packed, and changed frequently with wet-to-dry dressings. Arehnholz has recommended packing with povidone-iodine–moistened kerlex.17 Either way it is important that pooling of secretions in the wound not occur, because this may predispose the patient to growth of opportunistic infections. Once the patient is mobile, he or she may be allowed to shower frequently. In areas of the body with large defects, split-thickness skin grafts will be necessary. On occasion, free flaps or advancement flaps will be necessary to achieve cosmetic and soft tissue coverage, as we performed in our patients.

A controversial area in the treatment of necrotizing fasciitis is the use of hyperbaric oxygen. Gozal et al, and Riseman et al have suggested that it can decrease the mortality and morbidity associated with this condition.18,19 However, Shupak et al found that the mortality and number of surgical debridements required for treatment was actually higher in patients who received hyperbaric oxygen therapy.20

Bilton and colleagues have found that the keys to appropriate therapy are early wide surgical debridement with broad-spectrum antibiotics, and, if need be, return to the operating room for additional debridement.5 As with any critical condition, appropriate supportive measures are indicated.

**CONCLUSION**

Necrotizing fasciitis of the face is a rare condition that has a high morbidity and mortality. Its diagnosis requires a high index of suspicion on the part of the clinician. A history of trauma or surgical procedure is frequent. The cornerstones of therapy are extensive surgical debridement and appropriate critical care management.

**REFERENCES**

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