

## NECROTIZING FASCIITIS OF THE HEAD AND NECK: REPORT OF THREE CASES

Mustafa H. Ali, MD; Mohamed E. Zayed, MD

Necrotizing fasciitis (NF) is a rare but life-threatening multimicrobial soft tissue infection characterized by progressive, usually rapid, necrotizing process of the subcutaneous tissues and fascial planes, with resulting skin gangrene and systemic toxicity.<sup>1-6</sup> The condition, commonly described in the extremities, abdominal wall and perineum, is rarely seen in the head and neck.<sup>1,7</sup> Because of the fulminant course of NF, early diagnosis is imperative. Broad spectrum antibiotics, aggressive surgical treatment and supportive therapy are the cornerstones of successful treatment.<sup>5-7</sup> The diagnosis of NF depends mainly on clinical features which are not always obvious, while a high index of suspicion is needed. Unfortunately, the disease is often diagnosed late in its course, resulting in high mortality.<sup>6,8</sup> We describe three cases of necrotizing fasciitis of the head and neck, arising from different origins with different courses and outcomes. To the best of our knowledge, there are no previous reported cases from Saudi Arabia.

### Case 1

A 34-year-old insulin-dependent diabetic female patient presented to the emergency room complaining of right mandibular dental pain, associated with a tender right-sided neck swelling. The past medical history was significant for a positive serologic test for hepatitis B and a long-standing right heel ulcer. She was oriented and alert. The head and neck examination showed a diffuse, tender, nonfluctuant swelling of the right submandibular area. Oral examination revealed a painful carious right second mandibular molar. The rest of the examination was essentially normal. The vital signs were as follows: temperature 37.5°C, pulse rate 86 beats/min, blood pressure 110/70 mm Hg and respiratory rate 20/min. The abnormal blood tests were leucocytosis of 18,100/ $\mu$ L and glucose of 15.1 mm/L (271.8 mg/100 mL). Panoramic orthopantomogram revealed a radiolucency at the root of

the right mandibular second molar. The patient was admitted to the ENT service with diagnosis of a right submandibular space abscess secondary to dental infection. However, needle aspiration and ultrasonography of the involved area were negative. The patient was started on intravenous antibiotics (metronidazol 500 mg tid, gentamycin 80 mg tid and Velosef 1 g qid). Blood glucose was controlled on subcutaneous insulin according to a sliding scale. On the second day of admission, the carious molar was extracted under local anesthesia. The patient's condition deteriorated over the next two days. The temperature and the leucocytic count increased to 40.2°C and 64,700/ $\mu$ L respectively. Subsequently, the patient developed breathing difficulty. The swelling was spreading to involve the neck regions bilaterally with severe tenderness and crepitation. On the fifth day of admission, the right submandibular area was explored through a transverse incision under general anesthesia. A foul-smelling brownish fluid was found. A sample was sent for culture and sensitivity. The patient was admitted postoperatively to the Intensive Care Unit (ICU), intubated and artificially ventilated. Her fever persisted and the swelling progressed superiorly to the right face and inferiorly to the upper thoracic wall. The skin over the drained area and around the surgical incision became dusky, then black, and showed necrotic changes. The diagnosis of odontogenic cervicofacial necrotizing fasciitis was made. The patient was taken directly to the operating room (OR), where a wide midcervical collar incision was performed. Brown gray-colored and offensive copious fluid with bubbles was obtained. The fascial planes were gray and necrotic. Blunt dissection by finger and hemostat was done for all sites of the lesion which were not confined to one anatomic space. The muscles appeared healthy. The necrotic skin and fascia were excised. Wound swabs were sent for culture and sensitivity. The patient was taken back to the ICU. Further debridement was done on the following day. Extensive excision of the skin, subcutaneous tissue, platysma, investing layer of the deep fascia and part of the right sternocleidomastoid and masseter muscles was undertaken (Figure 1). Over the ensuing two days, the patient's general condition improved. The original wound swab grew alpha hemolytic *streptococcus*, while the

---

From the Department of Surgery, King Fahad Hospital, Al Baha, Saudi Arabia.

Address reprint requests and correspondence to Dr. Ali: Department of Surgery, King Fahad Hospital, P.O. Box 204, Al Baha, Saudi Arabia.

Accepted for publication 9 September 1997. Received 29 April 1997.

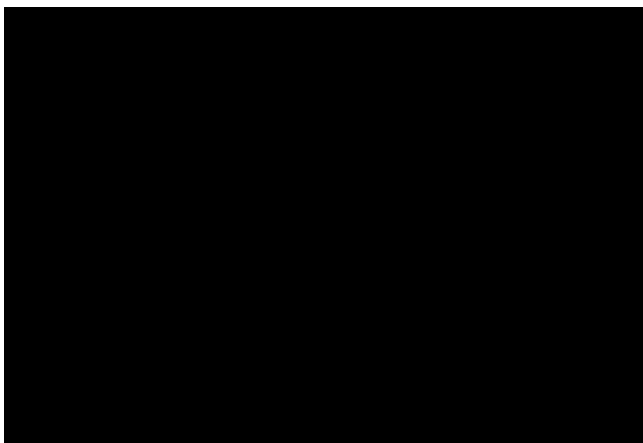


FIGURE 1. Case 1, following the second debridement. The right mandible is exposed.

subsequent wound cultures yielded alpha hemolytic *streptococcus* and *Pseudomonas aeruginosa*. Betadine-soaked wound dressings were changed four times daily. The postoperative course was stormy. The patient developed septic shock, which was confirmed by growth of *Pseudomonas aeruginosa* from the blood. Blood biochemistry revealed abnormal renal and liver function tests. The antibiotic regimen was adjusted according to the sensitivity. She had one session of hemodialysis. The renal functions improved, but the liver function tests remained abnormal. The patient suffered a sudden cardiopulmonary arrest on the 35th day of admission, and obviously incurred hypoxic brain insult. She was resuscitated, kept intubated and artificially ventilated. At this stage, the patient was beginning to develop generalized edema and ascites. Brain CT scan and chest x-ray were unremarkable. Nutritional support via nasogastric tube and later intravenous hyperalimentation were maintained. Blood transfusions, human albumin 20%, and adjustment of fluids and electrolytes were provided as needed. The patient's condition deteriorated progressively despite all supportive measures. The patient expired on the 62nd day of admission, most likely due to multiple organ failure.

### Case 2

An 84-year-old male patient presented to the emergency department with an eight-day history of sore throat, a three-day history of odynophagia, dysphagia to all but water, and a right submandibular swelling. He was seen by a general practitioner one day before presentation. Treatment with ampicillin was initiated and the patient was referred to the King Fahad Hospital, Al Baha, for further evaluation. On examination, vital signs were as follows: blood pressure 140/80 mm Hg, pulse rate 110 beats/min, respiration 24/min and temperature 38.2°C orally. The patient was oriented and alert but in distress.

The head and neck examination revealed a tender, erythematous, crepitant and nonfluctuant swelling extending superiorly from the right submandibular region, inferiorly to the right supraclavicular fossa and medially to the manubrium sterni. Oral examination showed right peritonsillar swelling and mild trismus. The chest was clear. Aspiration of the neck swelling gave about 1 mL of offensive-smelling liquid. Lateral neck x-ray disclosed gas in the soft tissue. Ultrasound scan of the right neck was unremarkable. Hematologic tests revealed a WBC of 18,300/ $\mu$ L. All other variables, including electrolyte levels and liver function tests, were normal. Chest x-ray was normal. The diagnosis of quinsy with parapharyngeal abscess was made. The patient was admitted to the ENT service and started on intravenous antibiotics (ceftazidime 1 g tid, gentamycin 80 mg tid, and metronidazol 500 mg tid). Under general anesthesia that followed difficult intubation, quinsy tonsillectomy and incision of the neck swelling at three sites were carried out. The tonsillar abscess drained offensive whitish pus. In the neck, a foul-smelling brownish fluid containing bubbles beneath the platysma and grey necrotic deep cervical fascia were found. Samples were sent for culture and sensitivity. The patient was transferred postoperatively to the ICU and kept intubated and artificially ventilated. On the following day, his temperature rose to 39.5°C and the WBC increased to 22,800/ $\mu$ L. The lesion extended inferiorly to the anterior upper thoracic wall. These features were highly suggestive of necrotizing fasciitis. The patient was returned to the OR where further debridement of the fascia and skin was done. Two days later the patient became normothermic. Hydrogen peroxide-soaked wound pack dressings were changed four times daily. The nutrition support was maintained via nasogastric tube. The patient was extubated seven days later. On the fourteenth day of admission the granulating area was covered by split skin graft. Eight days later the patient was discharged home. The final culture results showed growth of *Klebsiella pneumonia* and alpha hemolytic *streptococcus* in the swabs obtained from the tonsillar abscess and neck swelling. *Enterobacter cloacae* was also isolated from the neck discharge. At follow-up two months later, the patient was in good health.

### Case 3

A 50-year-old female presented with a four-day history of right-sided painful facial swelling and a seven-day history of sore throat. The patient stated that the swelling developed after insect bite a few days prior to presentation. Clinical examination revealed an obese patient in fair general condition. Vital signs were: temperature 36.8°C, pulse rate 82/min, respiration 20/min and blood pressure 120/80 mm Hg. The head and neck examination showed an erythematous, nonfluctuant, tender swelling of the right

cheek and chin with two small ulcerations. The one over the chin was draining thin pus. Examination of the nose demonstrated thick secretions filling both nasal cavities. Hematologic and biochemical tests were within normal limits, apart from WBC of 12,600/ $\mu$ L. Chest x-ray was normal. X-ray of paranasal sinuses yielded signs of chronic sinusitis. The patient was admitted to the ENT service with diagnosis of facial cellulitis. Wound swab was sent for culture and sensitivity. Intravenous antibiotics (gentamycin and velosef) were initiated. Over the next two days the edema decreased slightly, but the discharge persisted. The small opening of the chin was probed with blunt hemostat, which could be passed easily and painlessly to the cheek and upwards to the zygoma. Diagnosis of NF was suspected. On the third day of admission, the patient was taken to the OR, where the involved area was explored under general anesthesia. Odorless, brownish thin fluids and necrotic fascial plane were found, consistent with the diagnosis of NF (Figure 2). The necrotic tissue was excised extensively and sent for histopathology. A wound swab was sent for culture and sensitivity. Intraoperative laryngoscopy revealed a subglottic granulating lesion from which a biopsy was taken. Hydrogen peroxide-soaked wound dressings were changed several times daily. The first wound swab grew beta-hemolytic *streptococcus* group A, whereas the subsequent culture demonstrated alpha hemolytic *streptococcus*. Antibiotic regimen was changed according to the sensitivity. The necrotizing process progressed deeply and peripherally, therefore, the patient was returned to the OR for further debridement on the 11th day of admission. Despite the extensive debridement, the skin remained viable. The microscopic study revealed widespread necrosis with dense polymorphonuclear infiltration. The larynx biopsy yielded necrotic tissue with nonspecific inflammation. The wound showed good healing and the patient was discharged home in good health.

### Discussion

Necrotizing fasciitis has been described under various synonyms, including hospital gangrene, hemolytic or acute streptococcal gangrene, gangrenous or necrotizing erysipelas, suppurative fasciitis, Meleney's gangrene and Fournier's gangrene.<sup>3-6,9-12</sup> The term necrotizing fasciitis was first coined by Wilson in 1952.<sup>3,6</sup> NF usually occurs in the perineum, lower limbs or abdominal wall following surgery or trauma, particularly in individuals with underlying systemic diseases such as diabetes mellitus, arteriosclerosis, chronic renal failure or malnutrition.<sup>1,3,4,13</sup> NF may affect patients of all ages,<sup>5,13-15</sup> without sex or race predilection.<sup>3,12</sup> The condition has probably been underdiagnosed in the past, and is being increasingly recognized nowadays. NF is rarely seen in the head and neck,<sup>1,6,12,16</sup>

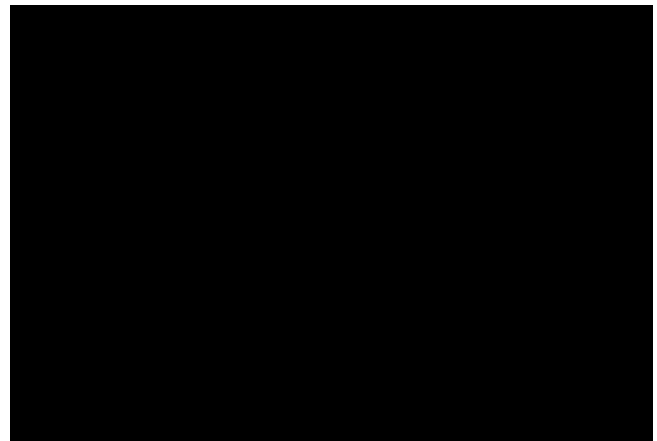


FIGURE 2. Case 3, four days after first debridement, showing thin exudate and necrotic subcutaneous plane.

however, its occurrence is probably more than the reported cases would suggest. In their review of the literature, Balcerak et al.<sup>3</sup> found 21 cases of NF in the head and neck, and reported three more cases. Of these, 10 cases of neck involvement were reported, while the remaining involved other head and neck areas. In 1989, Legreid and Hendrix found 35 cases of cervical necrotizing fasciitis and reported two more cases.<sup>17</sup> A total of 21 additional cases of neck involvement have been published since then.<sup>1,2,4,12,18-25</sup> NF involvement of the scalp and face is less common than the involvement of the cervical region. Kronish and McLeish in 1991 found 16 cases of NF reported involving periorbital and face,<sup>26</sup> and several other reports followed this.<sup>14,15,27,28</sup> In the majority of NF cases involving the neck, the disease follows dental or oropharyngeal infection,<sup>1-3,12,18,20,22,23,25</sup> whereas in the cases of scalp or face involvement, there is mostly a previous trauma that might be minor.<sup>2,3,14,15,26-30</sup> The first two cases described here had obvious cause, while the cause of the third one was unclear. The disease presents with a deceptively benign superficial appearance. Our cases demonstrate the fact that the fascial plane destruction is more extensive than the external evidence of the infection.<sup>3,5,7,11</sup> The clinical picture is dependent on the stage of the disease and its primary origin. Several authors have reviewed the presentation and the management of the disease.<sup>2-5,7,11,12,18,26,27,29</sup> The clinical signs and symptoms are nonspecific. The skin is red-hot, smooth, tense and tender without sharp demarcation between the involved and normal skin.<sup>1-4,11,12</sup> Crepitation, which was present in two of our cases, may be elicited. As the disease progresses, the skin becomes dusky and small blisters appear. Ultimately, frank gangrene takes place. The skin necrosis is secondary to thrombosis of the nutrient vessels passing through necrotic fascia.<sup>1-4,11,18,31</sup> The patient is acutely ill, with low to medium grade fever associated with tachycardia out of

proportion to the temperature elevation.<sup>3,4,11,26</sup> An extreme leucocytosis, as in our first case, can be seen. Lymphadenopathy and lymphangitis are usually not found.<sup>3,26,29</sup> Until now, there has been no specific diagnostic method for NF. Several adjuvant studies have been used as an aid in detecting the condition, e.g., radiographic study, CT scanning, isotope scanning and frozen section biopsy.<sup>1,7,10,20</sup> Gas in soft tissue, which was disclosed in one of our cases, is highly suggestive but not pathognomonic.<sup>3,6,8,11</sup> Diagnosis of our first case was delayed because of initial unawareness of the disease, and misinterpretation of the obvious clinical picture. The experience acquired from the first case aided in the early diagnosis and management of the subsequent two cases and consequently helped avoid a fatal course. All fascial planes of the head and neck can be involved in NF. Avascularity of the fascial planes is probably the cause of their involvement and initially sparing the muscles and skin.<sup>3,4</sup>

Bacterial necrotoxins might play a role in tissue damage.<sup>1,3,26,29</sup> The hallmarks of NF are widespread fascial necrosis with extensive undermining and serosanguinous, often foul-smelling exudate.<sup>1-7,12,18,26,31</sup> Passing a blunt instrument easily along the fascial plane is a specific clinical sign.<sup>3,4,26</sup> A spectrum of microbes have been reported to be the primary pathogens.<sup>1-6,9,11,15,26,29-31</sup> The micro-organisms isolated in our cases are consistent with the view that NF is a synergetic bacterial infection usually produced by a combination of gram-positive cocci and gram-negative rods.<sup>3,6,7,11,12</sup> However, some isolated organisms could represent superinfection in necrotic tissues which offer a favorable medium for bacterial colonization.<sup>19,25</sup> Co-existing anaerobic pathogens in the first two cases was considered as a possibility. Although the repeated cultures did not yield any, the patients were given antibiotics covering anaerobic organisms.

Prompt and successful management of NF is universally agreed to be aggressive surgical intervention, intravenous wide-spectrum antibiotics and supportive therapy.<sup>2-31</sup> It is of the utmost importance to explore and drain all affected areas. Wide extensive fasciotomies with exposure of all involved fascia and excision of necrotic tissues should be carried out. Early surgical intervention would minimize loss of the covering skin and avoid cosmetic disfiguring.<sup>4-6,8</sup> The healthy skin should not be excised initially, even in extensive debridement of the underlying fascia. Skin necrosis occurring late can be dealt with later. Skin loss was extensive in the first case due to delayed diagnosis and treatment, less in the second case and very minimal in the third case. We did not use any irrigation solution after debridement. We used wound dressings soaked in H<sub>2</sub>O<sub>2</sub>, normal saline or Betadine as topical treatment after debridement. However, we believe that the frequency of dressing is far more important than

the solution used. Broad-spectrum antibiotics covering both aerobic and anaerobic organisms should be initiated and changed according to the sensitivity patterns. We would like to stress the point that antibiotic therapy alone is considered insufficient for the management of NF without surgical intervention.<sup>1-7,26,29</sup> The nutritional support, whether enteral or parenteral, is also of great importance. The local and systemic complications of NF are numerous and include extension to the cervical viscera and mediastinum, vessel erosion and functional and cosmetic disfiguring.<sup>1,3,20</sup> Death from necrotizing fasciitis is usually due to overwhelming sepsis, respiratory failure, renal failure or multiorgan failure.<sup>3,6,7,11,12,28</sup> The mortality rate ranges between 8% and 73%.<sup>1,3,6</sup> The principal factors contributing to the high mortality are delayed diagnosis and treatment, extent of the disease, old age and associated systemic illnesses.<sup>1-5,11-13,26,29</sup> Necrotizing fasciitis involving the scalp and upper face has better prognosis than that involving the neck.<sup>3,26</sup>

These presented cases emphasize the need for early diagnosis, proper aggressive surgical intervention and intravenous antibiotic treatment of this serious life-threatening infection. The condition must be recognized clinically as early as possible to prevent unnecessary morbidity and mortality.

#### Acknowledgements

The authors thankfully acknowledge the helpful review and correction of the manuscript by Dr. Ahmed Ghali, Consultant Cardiologist and Chief of ICU, KFH, Al Baha. Thanks also to Ms. Carole T. Caramat for providing secretarial assistance in the preparation of this manuscript.

#### References

1. Reed, JM, Anand, VK. Odontogenic cervical necrotizing fasciitis with intrathoracic extension. *Otolaryngol Head Neck Surg* 1992;107:596-600.
2. Scott PMJ, Dhillon, RS, McDonald, PJ. Cervical necrotizing fasciitis and tonsillitis. *J Laryngol Otol* 1994;108:435-437.
3. Balcerak RJ, Sisto JM, Bosack RC. Cervicofacial necrotizing fasciitis: report of three cases and literature review. *J Oral Maxillofac Surg* 1988;46:450-9
4. Valko PC, Barrett SM, Campbell JP. Odontogenic cervical necrotizing fasciitis. *Ann Emerg Med* 1990;15:568-71.
5. Freischlag JA, Ajalat G, Busuttill RW. Treatment of necrotizing soft tissue infections: the need for a new approach. *Am J Surg* 1985;149:751-5.
6. Miller JD. The importance of early diagnosis and surgical treatment of necrotizing fasciitis. *Surg Gynecol Obstet* 1983;157:197-200.
7. Majeski JA, Alexander JW. Early diagnosis, nutritional support and immediate extensive debridement improve survival in necrotizing fasciitis. *Am J Surg* 1983;145:784-7.
8. Stamenkovic I, Lew PD. Early recognition of potentially fatal necrotizing fasciitis: the use of frozen section biopsy. *N Eng J Med* 1984;310:1689-93.
9. Cohn I, Bornside GH. Infections. In: Schwartz SI, Shires GT, Spencer FC, editors. *Principles of surgery*. Vol. I. Singapore: McGraw-Hill Information Services Company, 1989:181-215.

10. Joo P, Peters WJ. Fournier's gangrene. *Can J Surg* 1985;28:180-2.
11. Baxter CR. Surgical management of soft tissue infections. *Surg Clin North Am* 1972;52:1483-99.
12. Kaddour HS, Smelt GJC. Necrotizing fasciitis of the neck. *J Laryngol Otol* 1992;106:1008-10.
13. Frick T, Simmen HP, Kach K, Duff C, Hoffmann R, Zellweger G, et al. Schwere nekrotisierende Fasziiitis. *Helv Chir Acta* 1992;59:341-4.
14. Rose GE, Howard DJ, Watts MR. Periorbital necrotizing fasciitis. *Eye* 1991;5:736-40.
15. Pannier M, Bouchot-Hemouet M, Lavergne-Hepner D, David A, Stalder JF. Fasciite necrosante pri-orbitaire a streptocoque beta-hemolytique chez l'enfant. *Ann Chir Plast Esthet* 1991;36:75-8.
16. Rea WJ, Wyrick WJ. Necrotizing fasciitis. *Ann Surg* 1970;172:957-64.
17. Legreid RJ, Hendrix RA. Cervical necrotizing fasciitis: two case reports and review of the literature. *Trans Pa Acad Ophthalmol Otolaryngol* 1989;41:864-71.
18. Maqbool M, Ahmad R, Ahmed A, Qazi S. Necrotizing fasciitis in the head and neck. *Brit J Plast Surg* 1992;45:481-3.
19. Gillis AR, Gillis TM. Necrotizing cervical fasciitis of unknown origin. *J Otolaryngol* 1992;21:171-3.
20. Lalwani AK, Kaplan MJ. Mediastinal and thoracic complications of necrotizing fasciitis of the head and neck. *Head Neck* 1991;13:531-9.
21. Gaukroger MC. Cervicofacial necrotizing fasciitis. *Brit J Oral Maxillofac Surg* 1992;30:111-4.
22. Rapoport Y, Himelfarb MZ, Zikk D, Bloom, J. Cervical necrotizing fasciitis of odontogenic origin. *Oral Surg Oral Med Oral Pathol* 1991;72:15-8.
23. Shupak A. Cervical necrotizing fasciitis: an uncommon sequela to dental infection (letter). *Ann Otol Rhinol Laryngol* 1991;100:432.
24. Yamaoka M, Furusawa K, Kiga, M, Iguchi K, Hirose I. Necrotizing buccal and cervical fasciitis. *J Cranio Maxillofac Surg* 1990;18:223-4.
25. Moss RM, Kunpittaya S, Sorasuchart A. Cervical necrotizing fasciitis: an uncommon sequel to dental infection. *Ann Otol Rhinol Laryngol* 1991;99:643-6.
26. Kronish JW, McLeish WM. Eyelid necrosis and periorbital necrotizing fasciitis. Report of a case and review of the literature. *Ophthalmology* 1991;98:92-8.
27. Williams SR, Carruth JA, Brightwell AP. Necrotizing fasciitis of the face without significant trauma. *Clin Otolaryngol* 1992;17:344-50.
28. Kiely PD, Gilvary A. Periorbital necrotizing fasciitis: trivial facial injury resulting in cardiac arrest. *Br J Clin Pract* 1993;47:169-70.
29. Walters R. A fatal case of necrotizing fasciitis of the eyelid. *Br J Ophthalmol* 1988;72: 428-31.
30. Skef Z, Harding R, Graham WP. Disseminated necrotizing fasciitis of the scalp. *Ann Plast Surg* 1981;6:322-6.
31. Drake-Lee AB, Broughton SJ, Rampling A, Lancer, JM, Moffat DA. Necrotizing fasciitis. *J Laryngol Otol* 1983;97:193-6.