Craniocervical necrotizing fasciitis of odontogenic origin with mediastinal extension

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Abstract
We review an interesting case of craniocervical necrotizing fasciitis with thoracic extension in an immunocompetent 44-year-old man. The patient underwent aggressive medical and surgical management during a long hospitalization. Multiple surgical debridements, including transcervical mediastinal debridement, and eventually a thoracotomy for mediastinal abscess were required. The patient eventually recovered, and 3 months later he showed no sign of complications or recurrence. Craniocervical necrotizing fasciitis is a fulminant soft-tissue infection, usually of odontogenic origin, that requires prompt identification and treatment to ensure survival. Broad-spectrum intravenous antibiotics, aggressive surgical debridement and wound care, hyperbaric oxygen, and good intensive care are the mainstays of treatment.

Introduction
Necrotizing fasciitis is a fulminant soft-tissue infection that occurs most frequently in the extremities, abdomen, and perineum.1,2 When it occurs in the head and neck, it is called craniocervical necrotizing fasciitis (CCNF), and it is very aggressive and life-threatening. Instead of walling off to form an abscess, this polymicrobial infection spreads along the superficial fascial planes of the neck and tracks down into the mediastinum. It spares the overlying skin initially, but eventually the skin becomes erythematous and it can become necrotic when feeding microvessels become thrombosed.3 Historically, necrotizing fasciitis has occurred following surgery or minor trauma,1 but most cases of CCNF are odontogenic in origin.4 Predisposing factors include diabetes mellitus, peripheral vascular disease, cirrhosis, and alcoholism.2,5-7 Early mortality rates from CCNF ranged from 50 to 73%,2 but these rates had decreased to 22% in the early 1980s and to 0 to 10% in more recent studies.3,5,6 Complications of CCNF include airway obstruction, vascular occlusion or thrombosis, and mediastinal extension. The infection classically involves the mediastinum via two routes: (1) by fascial spread along the carotid sheath inferiorly into the mediastinum or (2) by spread through the retropharyngeal space, into the prevertebral space (danger space), and inferiorly into the mediastinum.5 Once CCNF reaches the mediastinum, it can cause mediastinitis, pericarditis, pleural or pericardial effusion, empyema, pneumonitis, cardiac tamponade, and esophageal bleeding.3,7 Early identification of the disease process, broad-spectrum intravenous antibiotics, aggressive surgical debridement and wound care, and supportive measures such as hyperbaric oxygen are the keys to a successful outcome in CCNF.

In this article, we describe an interesting case of CCNF with thoracic extension.

Case report
A 44-year-old black man presented to an outside emergency room with a 2-day history of facial pain and swelling and a 1-day history of chest pain. Five days earlier, he had been started on oral penicillin to treat an abscess of the right lower first and second molars. His medical history was significant for hypertension and alcoholism. On presentation to the ER, he was noted to have a firm and edematous anterior neck with a protruding tongue. Other major complaints included dysphagia and odynophagia. His white blood cell count was 5,800 cells/mm³ and he was febrile at 38.5°C. Computed tomography (CT) detected air in the anterior neck spaces.

The patient was admitted and started on clindamycin and ceftriaxone. On hospital day 2, he was intubated in
response to increasing respiratory distress. Examination demonstrated a decrease in his anterior neck edema and tongue protrusion. He was then transferred to the intensive care unit at the George Washington University Hospital, and the otolaryngology service was consulted. On examination, he was noted to have extremely poor dentition and a necrotic, fibrinous floor of the mouth with multiple holes in the oral mucosa, which were communicating. On digital examination, the otolaryngologist was able to palpate through the necrotic areas down to the hyoid bone. The tongue was mobile, the tongue base was viable, and there was no involvement of the oropharynx. Minimal edema of the submental and submandibular areas of the neck was noted. There was no crepitus, but erythema extended from the mandible down over the anterior chest.

A culture specimen was obtained from the floor of the mouth. Repeat CT of the neck and chest demonstrated (1) subcutaneous air in the neck bilaterally that extended from the floor of the mouth to the superior and anterior mediastinum, tracking along the area of the superficial cervical fascia, and (2) bilateral pleural effusions (figure 1). At this point, the patient was hemodynamically stable and showed no signs of overt sepsis. The antibiotic regimen was changed to clindamycin, ciprofloxacin, and penicillin G.

The patient was taken to the operating room on hospital day 3 for surgical debridement of the neck. Four wide incisions were made in the neck—an upper and lower incision on each side. Necrotic “dishwater-type” fluid was encountered once the platysma was incised bilaterally. Purulent material was encountered in the submandibular space, tracking down from the floor of the mouth. Necrotic material was discovered in bilateral parapharyngeal spaces, with necrosis of the fascia along the carotid sheath extending inferiorly into the mediastinum (figure 2). No myonecrosis was seen.

Cardiothoracic surgery was consulted at this point for a transcervical debridement of the anterior mediastinum. Exploration of the mediastinum revealed necrotic material, similar to the findings in the neck. The anterior mediastinum and neck were packed open with sterile gauze. Pathology specimens confirmed necrotizing fasciitis. Cultures grew *Streptococcus anginosus*.

Twice-daily changes of sterile dressings were initiated. The patient remained stable, but repeat CT of the chest on hospital day 5 demonstrated continued pneumomediastinum with increased fluid collection. The patient was taken to the operating room the following day for a right thoracotomy and mediastinal debridement. Intraoperatively, purulent necrotic material was present in the anterior mediastinum. Three thoracostomy tubes were placed, along with two Blake drains to allow for irrigation. Continuous mediastinal irrigation with 0.5% povidone iodine was initiated through the Blake drains.

The patient required two further surgical debridements of the neck, with continued twice-daily wet-to-dry dressing changes during his hospital stay. The wound slowly began to granulate, and the incision had almost closed by secondary intention at the time of discharge. An open jejunostomy feeding tube was placed for nutrition on hospital day 18. On day 25, persistent, loculated pleural effusions—which were thought to be a source of a continually elevated white blood cell count and which could not be drained by multiple thoracostomy incisions—required left video-assisted thoracoscopic surgery with decortication. The decision was made not to perform a tracheotomy in an infected open field; instead, the patient was kept orally intubated. He failed several attempts at extubation before he was successfully extubated on hospital day 36. The patient was then transferred to the floor, and he was discharged to a rehabilitation facility on hospital day 51.

The patient was not seen for follow-up until 3 months later, when he was admitted to the hospital for delirium.
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Secondary to alcohol withdrawal. At that time, his neck incisions were well healed, and he was having no difficulty swallowing.

Discussion
Joseph Jones, a Confederate army surgeon, was the first to note what later came to be known as necrotizing fasciitis when he described “hospital gangrene,” which he had observed in wounded soldiers during the Civil War.

In 1924, Meleney described 20 cases of superficial fascial necrosis in China, and he isolated beta-hemolytic streptococcus as the causative organism. It was Wilson who first coined the term necrotizing fasciitis in 1952.

As noted earlier, surgery and minor trauma have most often been cited as the cause of necrotizing fasciitis. In most of the case series on CCNF, the primary etiology has been an odontogenic infection, although infections secondary to surgery and peritonsillar abscess have also been reported. Because the roots of the second and third molars lie below the mylohyoid line, odontogenic infection from a periaxial abscess can descend into the submandibular space.

This area is contiguous with the parapharyngeal space. From here, the infection has two routes of spread. All layers of the deep cervical fascia join to form the carotid sheath, or what Mosher in 1929 called “the Lincoln Highway” (the nation’s first transcontinental highway). Extension along the carotid sheath allows infection to spread throughout the neck and into the mediastinum. The disease process can also spread to the retropharyngeal space and then down the prevertebral space and into the mediastinum.

Mediastinal extension of CCNF is a very serious complication that can lead to mediastinitis, pericarditis, pleural or pericardial effusion, empyema, pneumonitis, cardiac tamponade, and esophageal bleeding.

If there is any suspicion of mediastinal extension, cardiothoracic surgery should be considered for transcervical mediastinal debridement or, if the infection extends below the 4th thoracic vertebra, possible thoracotomy.

Airway management is of paramount importance in patients with CCNF. In a review of cases of CCNF with thoracic extension published in 2001, Bahu et al reported an overall mortality rate of 35%. The overall mortality rate for CCNF without mediastinal extension has been reported to range from 0 to 10%. In 1994, Maisel and Karlen reported no deaths in their series of 9 patients with cervical necrotizing fasciitis. This illustrates the improvement in diagnosis and management, because in 1984, Spankus et al reported that mortality from CCNF was 22%. Many patients with CCNF already had an underlying disorder (e.g., diabetes or alcoholism), and they were therefore more likely to develop a necrotizing infection as opposed to an abscess. Common comorbidities in patients who develop CCNF are diabetes mellitus, peripheral vascular

Figure 2. A: In the submandibular area of the neck, necrosis is seen in the deep cervical fascia. B: In the left lower neck, evidence of fascial necrosis is seen along the carotid sheath (C) and the sternocleidomastoid muscle (S). C: Necrosis of the deep cervical fascia is also seen in the right lower neck. (Arrows point toward the head.)
disease, cirrhosis and, as was the case with our patient, alcoholism.2,3,5,7

On initial presentation, infection in CCNF patients must be differentiated from cellulitis, which is sometimes accompanied by erythema. Cellulitis will respond to medical intervention alone, but necrotizing fasciitis requires surgical debridement. Erythema can be present in both diseases, but crepitus occurs only in CCNF. Even so, many patients with CCNF do not manifest crepitus even when there is subcutaneous air along the fascial planes, as was the case with our patient.

CT is a good tool in the early diagnosis of necrotizing fasciitis. Becker et al reported that the most common CT findings in CCNF were the thickening and infiltration of subcutaneous tissues, fluid collection in multiple neck compartments, and diffuse enhancement and thickening of the cervical fascia, platysma, and sternocleidomastoid and strap muscles.14 Only 60% of their patients demonstrated subcutaneous air on CT. Follow-up CT is useful for tracking the resolution of disease and for demonstrating any residual fluid collection. The timing of surgical debridements in our patient was dictated by findings on CT as well as on clinical examination.

The bacteriology of these infections usually involves a combination of anaerobic and facultative aerobic bacteria. Streptococci or staphylococci in combination with anaerobic oral flora such as Peptostreptococcus or Bacteroides species are the most prevalent pathogens in necrotizing fasciitis.15 The synergy between the two pathogens accounts for the aggressive nature of this disease; the removal of oxygen by the facultative aerobes allows the obligate anaerobes to flourish. Streptococcal mucoproteins and bacterial enzymes such as lipase and hyaluronidase can cause tissue necrosis, leading to the easy separation of fascial planes and dissection into other areas of the neck.2 Initial antibiotic coverage should entail broad-spectrum antibiotics until cultures can be obtained.

Aggressive surgical debridement with wide exposure of all fascial planes and safe removal of all necrotic fascia is necessary to ensure survival. Repeat surgical exploration and debridement is necessary until there is no further evidence of necrosis. Wound care requires changing dressings and packing at least twice a day to allow granulation tissue to form and to prevent fluid sequestration and collection. CT is useful for determining if fluid has collected following the initial debridement.

Hyperbaric oxygen therapy has been used in necrotizing fasciitis to minimize mortality and to reduce the number of surgical debridements required to control infection.16 Hyperbaric oxygen increases tissue oxygenation and promotes angiogenesis, collagen deposition, and capillary budding, thereby improving wound healing and making the cellular environment unfavorable for anaerobic bacteria.17

References

ENT, Ear, Nose & Throat Journal • August 2004