Necrotizing fasciitis of the scalp stemming from odontogenic infection

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ABSTRACT

Necrotizing fasciitis is a severe, rapidly progressive infectious process characterized by rapid spread and extensive tissue destruction. This condition stems from a variety of sources, may affect any area of the body, and requires rapid diagnosis and aggressive intervention. We present a rare case of odontogenic infection progressing to acute cervicofacial necrotizing fasciitis of the scalp and neck.

KEYWORDS Necrotizing fasciitis; necrotizing soft tissue infection; odontogenic infection; scalp infection

Necrotizing fasciitis (NF) was first described by the Confederate surgeon Jones in 1871, followed by Pfanner in 1918 and Meleney in 1924.¹ NF is caused by a variety of organisms^{2,3} and most commonly affects the abdomen, perineum, and extremities; cervicofacial NF is uncommon.⁴ Signs and symptoms include severe pain, fever, subcutaneous gas, crepitus, edema, erythema, tachycardia, confusion, and shock. Exploratory incisions will reveal dissection and necrosis of subcutaneous and fascial tissue planes without resistance, as well as thin, brownish “dishwater fluid.”^{4,5} Even with rapid recognition and aggressive intervention, mortality rates range from 25% to 40%.^{5–7}

CASE DESCRIPTION

A 52-year-old man with no reported past medical or surgical history presented to the Baylor University Medical Center emergency department with a progressive painful right facial swelling of 4 weeks’ duration. The process spread to the right postauricular skin and scalp a week prior to admission and progressed to involve most of his scalp and superior periorbital tissues bilaterally. The patient was unable to properly feed himself for several days. Examination revealed profuse edema of the upper eyelids, forehead, and scalp bilaterally extending posteriorly and inferiorly to the occipital scalp. On the right postauricular skin, an area of necrotic tissue was noted with the thin “dishwater” fluid

expressed on palpation. Necrotic areas of the bilateral eyelids were also noted. The entire scalp and upper face were tender to palpation and exhibited substantial crepitus. His oral examination revealed inflammation and edema around the right posterior mandible and several carious teeth. A computed tomography scan with intravenous contrast revealed subcutaneous gas throughout the scalp, as well as bony erosion around an impacted right mandibular molar consistent with osteomyelitis (*Figure 1*). Lab values were significant for an elevated serum carbon dioxide of 41 mEq/L and white blood cell count of 23,000 K/uL.

The patient was taken to the operating room less than 4 hours after arrival. An open tracheotomy was performed. The right mandible was explored, multiple teeth were removed, and necrotic bone was debrided. A tract extending superiorly from the lateral surface of the temporalis toward the scalp was noted. The right postauricular tissue was then explored and the full thickness of the necrotic area was excised. The surrounding tissues were then examined, and the deeper layers dissected readily, revealing extension of the infection into those planes. A coronal flap was raised and reflected anteriorly and posteriorly (*Figure 2*). Extensive gross necrosis was noted encompassing the periosteum, areolar connective tissue, and galea across most of the scalp, with copious output of dishwater fluid throughout the scalp, forehead, and upper eyelids. All questionable tissue was removed, including galea,

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Figure 1. Computed tomography scan demonstrating significant subcutaneous gas formation within the scalp bilaterally.

Figure 2. Coronal flap reflected showing the extent of the necrotic connective tissues and pericranium.

connective tissue, and pericranium across most of the scalp. Multiple drains were placed.

Due to septic shock and hemodynamic instability, the patient was kept in the surgical intensive care unit for 8 days. His hemoglobin A1C was 8.4%, indicating previously undiagnosed diabetes. Cultures grew *Prevotella*, coagulase-negative *Staphylococcus*, *Candida*, *Bacteroides*, and diphtheroids. An aggressive antibiotic regimen of vancomycin, piperacillin-tazobactam, and clindamycin was implemented. The patient was subsequently taken three additional times

Figure 3. (a) Patient following the second debridement, showing the extent of excised necrotic tissue. (b) Final result following tissue expansion and rotational flap coverage of the affected area.

for washout and debridement (Figure 3a). Skin grafting was performed to his right eyelid, and a matrix wound dressing was sutured to the edges of the postauricular defect. The patient was discharged on postoperative day 14.

Subsequently, he was taken for surgery 2 weeks following discharge for washout and replacement of the wound matrix. At 3 months, a split-thickness skin graft was placed over the newly formed tissue bed. Subsequent follow-up revealed a healthy postauricular tissue bed but with significant scarring, irregular tissue, absent hair, and poor cosmesis. At 6 months, a tissue expander was placed in the postauricular wound site. Expansion was performed over 6 weeks allowing for rotation of the expanded local tissues over the wound at 7.5 months. The patient has since recovered uneventfully (Figure 3b).

DISCUSSION

NF is an uncommon disease characterized by a fulminant, aggressive bacterial infection that rapidly spreads, causes extensive destruction, and may affect any part of the body.^{3,5} Cervicofacial NF is even more uncommon, with a reported incidence of 0.048%⁸ to 2.6%⁹ of all odontogenic infections. Cases involving the face and scalp are rare; approximately 50 total cases have been reported in the literature since the 1960s.^{10,11}

There are four classifications for NF. Type I is polymicrobial, incorporating gram-positive cocci, gram-negative rods, and anaerobes. Type II is caused by group A streptococci with or without *Staphylococcus aureus*. It is rising in incidence and can affect young, healthy, immunocompetent hosts.^{2,3} The rare type III is caused by *Vibrio* spp. Type IV describes fungal infection in compromised patients.³ NF is associated with diabetes mellitus, chronic systemic disease, immunocompromised states, peripheral vascular disease, obesity, and alcohol or drug abuse.^{3,12} Both minor and major trauma are precipitating causes of NF.³ Odontogenic infection is a common source of cervicofacial NF¹³; other sources include peritonsillar abscess,¹⁴ otitis media,¹⁵ and sialadenitis.¹⁶

Early recognition of NF is often difficult on clinical examination. Inflammation and necrosis typically underlie initially normal-appearing skin. Disproportionate tenderness, pain, and

warmth are often noted. The skin becomes progressively necrotic due to underlying thrombosis.¹⁷ Exploration reveals tissue planes that separate easily, as well as minimal bleeding and the pathognomonic finding of dishwater fluid. Although not always present, subcutaneous gas and associated crepitus are highly sensitive for NF.^{18,19} Surgical intervention should not be delayed if imaging is not available.¹⁸ The Laboratory Risk Indicator for Necrotizing Fasciitis scoring system combines C-reactive protein, white cell count, hemoglobin, sodium, creatinine, and glucose values into a score from 0 to 13. Scores of ≥ 6 raise suspicion for NF, and scores of ≥ 8 are strong indicators.²⁰ A meta-analysis revealed score sensitivity of 68.2% and 40.8%, respectively, with high specificity of 84.8% and 94.9%.²⁰

Delay in surgical intervention >14 hours is associated with significantly higher mortality, septic shock, and more repeat debridements.^{6,21} Initial surgical intervention is aimed at determining the extent of the infection and removing all grossly necrotic tissues. Debridement is repeated every 1 to 2 days until all necrotic tissue is removed and the remaining tissue bed appears healthy. Antibiotic treatment alone is ineffective due to necrosis and poor vascularity.⁸ Broad-spectrum antibiotics should be used until cultures are available.⁵ Studies estimate the mortality of NF at 23.5% to 29%.^{5,22} Hospital-acquired infections, age >75 years, peripheral vascular disease, sepsis, and septic shock are predictors of increased mortality.²³ Though cervicofacial NF is unquestionably a deadly disease, rapid and repeated surgical intervention, a secure airway, and control of underlying disease can result in acceptable outcomes.

DISCLAIMER

The views expressed in this article reflect the results of research conducted by the author and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the US Government. Lieutenant Commander Lee and Lieutenant Votto are military servicemembers. This work was prepared as part of their official duties. Title 17, USC, §105 provides that "copyright protection under this title is not available for any work of the U.S. Government." Title 17, USC, §101 defines a US Government work as a work prepared by a military service-member or employee of the US Government as part of that person's official duties.

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