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Review Article

Necrotizing Fasciitis of the Head and Neck in Adults: Clinical Features, Diagnosis, and Therapeutic Strategies

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ABSTRACT

Necrotizing fasciitis (NF) is a critical, life-endangering infection of the soft tissues that targets the deep fascia and subcutaneous layers. Its hallmark is fulminant progression and a high fatality rate. NF in the head and neck region is extremely rare, and dental sources account for the majority of cases. This paper aims to provide an exhaustive review of the principal characteristics of cervical necrotizing fasciitis (CNF) in adults, supplementing the literature with our own clinical insights into its treatment. The most frequently harvested pathogens are *Streptococcus* spp. and *Staphylococcus* spp. Patient outcomes become exceedingly bleak if the infection descends and establishes mediastinitis. Given that early signs can mimic a standard, non-necrotizing deep cervical infection, maintaining keen clinical alertness is vital for prompt detection. Computed tomography imaging is indispensable for confirming the diagnosis, mapping the spread of the disease/eliminating the possibility of descending mediastinitis. Immediate and extensive surgical removal of all nonviable tissue, alongside intravenous antibiotics and aggressive fluid replacement, are non-negotiable pillars of care that must commence without delay for culture reports. Even with swift and optimal treatment, the associated mortality of CNF is known to reach rates as steep as 35%.

Keywords: Necrotizing fasciitis, Soft-tissue infections, Mediastinitis, Cervical necrotizing fasciitis, Descending necrotizing mediastinitis

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Introduction

Necrotizing fasciitis (NF) is an infection with the potential to be lethal, defined by the swift, unrelenting destruction of the superficial fascia and the overlying subcutaneous fat. Septic shock and failure of one or more organ systems are frequent companion conditions. The label “necrotizing fasciitis” was coined by Wilson [1]; nevertheless, documentation by Hippocrates circa 500 BC depicts an ailment where there was a “great falling off of the flesh, tendons, and bones; and the defluxion which seated in the parts was not like pus, but a sort of putrefaction” [2, 3]. The zones most regularly implicated are the torso’s lower limbs, abdominal wall, or perineum. Estimates of occurrence range from 1.8 cases per 100,000 population per year in New Zealand [4] to 19.7 per 100,000 per year in Fiji [5]. Based on figures from the Centers for Disease Control, since the year 2010, anywhere from 700 to 1150 incidents of NF driven exclusively by group A *Streptococcus* are recorded on an annual basis in the United States [6], and the United Kingdom registers an NF caseload of roughly 500 per year [7]. A correlation with socioeconomic disadvantage has been theorized, pointing to factors such as residential overcrowding and barriers to obtaining medical attention [4]. That said, necrotizing fasciitis confined to the head and neck is a rare finding, accounting for only 1–10% of cases

[2], a phenomenon likely attributed to the territory's rich vascular network. The core objective of this review is to present a detailed synthesis of the most pivotal aspects of cervical necrotizing fasciitis (CNF) in the adult population, emphasizing the absolute necessity of early diagnosis and therapeutic intervention. Our motivation is to aid the physician with limited experience in distinguishing this singular form of infection or harboring doubt about it in clinical practice.

Pathogenesis and clinical features

Pathogenesis

NF is a deep-seated, invasive process within the subcutaneous compartment that migrates rapidly along fascial sheaths, owing to their inherently limited vascular supply. This leads to clotting in the local vasculature, skin starvation, and death of the membranous tissue. During the initial phase, the exterior skin can appear deceptively innocuous, while the destruction of the underlying fascia is far more widespread [2, 8]. With a few days passing, it becomes a heated, flushed surface sensitive to touch. Infectious involvement of the subcutis leads to venous blockages in the small dermal vessels and a buildup of inflammatory cells, producing pockets of pus, which are subsequently followed by arterial compromise due to obliterating endarteritis. This cascade leads to a critical deficit in blood supply to the skin, manifesting as vesicles or larger, fluid-filled bullae. Death of the skin tissue and a form of wet, colliquative gangrene then takes hold. At this advanced stage, the patient's pain sensation lessens, a result of damage to the superficial sensory nerves [9-11].

The most common initial presentations of CNF are dental and throat infections **(Table 1)** [12, 13].

Table 1. Causes of CNF.

Causes of CNF	Frequency
Odontogenic	47%
Pharyngeal	28%
Tonsillar / Peritonsillar	6%
Major salivary glands	2.5%
Skin disruption	1.7%
└ Surgical wounds	—
└ Animal bites	—
└ Lacerations and abrasions	—
└ Injection (e.g., intravenous drug use)	—
No identifiable source	10%
Other causes	4.8%
└ Otitis media and mastoiditis	—
└ Blunt trauma without skin laceration	—
└ Radiotherapy	—

Pulling data from a systematic review undertaken by Gunaratne *et al*. [14], the microbial species most commonly encountered in CNF turned out to be *Streptococcus* spp. (found in 61% of the subjects) and *Staphylococcus* spp. (18%). *Prevotella* spp., *Peptostreptococcus* spp., *Bacteroides* spp., *Fusobacterium* spp., *Enterobacter* spp., *Klebsiella* spp., *Escherichia coli*, *Pseudomonas* spp., and *Candida* spp. made up the rest of the findings. A mean tally of

2 ± 0.98 distinct organisms were isolated from each patient in the study by Gunaratne *et al*. [14].

The relationship between nonsteroidal anti-inflammatory drugs (NSAIDs) and necrotizing soft-tissue infections, most notably those instigated by group A *streptococcus*, is a matter of ongoing dispute. NSAIDs were singled out as an independent predisposing element for limb-based NF within one case-control investigation [15], and studies on mouse models have demonstrated that both ketorolac and ibuprofen can hasten illness evolution and lead to poorer results [16, 17].

Classification

Table 2 outlines the microbiological taxonomy of NF. The reported prevalence of each subtype differs significantly across published series [18]:

NF type I is marked by a mixed-growth infection harboring both oxygen-dependent and oxygen-intolerant bacterial strains. As a rule, no fewer than one anaerobic isolate (with *Bacteroides* spp., *Clostridium* spp., or *Peptostreptococcus* spp. being the most frequently encountered) is recovered alongside members of the Enterobacteriaceae family (*E. coli*, *Enterobacter* spp., *Klebsiella* spp., and *Proteus* spp.)

and a contingent of one or more facultative anaerobic streptococci (distinct from group A *Streptococcus*) [19]. When the process localizes to the head and neck, organisms indigenous to the oral cavity (*Fusobacterium* spp., anaerobic Streptococci, *Bacteroides* spp., and *Spirochetes* spp.) are typically cultured. *Pseudomonas aeruginosa* and *Candida* spp. surface only sporadically in these combined infections. This variant predominates in patients with impaired host defenses or those burdened by underlying illnesses such as diabetes mellitus. The incubation period for these multibacterial infections tends to be more protracted than that observed in monomicrobial (type II) infections [20].

NF type II denotes a single-organism infection, most often arising from group A *Streptococcus*, other beta-hemolytic Streptococcal species, or *Staphylococcus aureus* [21]. It shows no predilection for a specific age range and can strike individuals lacking any preexisting medical conditions; in half of such cases, no obvious point of microbial entry can be traced. Its onset commonly follows the bloodstream dissemination of group A *Streptococcus* from the oropharynx (pharyngitis, whether clinically overt or silent) to a site subjected to blunt impact or muscular overuse [13, 18]. Necrotizing infections caused by group A Streptococcus strains expressing M protein serotypes 1 and 3 are associated with toxic shock syndrome in roughly half of affected patients [22, 23]. Fever-inducing exotoxins secreted by these strains provoke a massive cytokine release, which in turn drives hypotension, tissue disintegration, and end-organ dysfunction [24].

Table 2. Microbiologic classification of necrotizing infections (modified from "Approach to microbiologic diagnosis of necrotizing infections", Up to date 2024 [25]).

Presence of gas in soft tissue (radiographic imaging)			
Microbial category	**Clinical form/presentation**	**Diagnosis**	**Typical organisms/Notes**
Polymicrobial (1)	—	Necrotizing fasciitis type I (polymicrobial)	—
—	—	Necrotizing cellulitis (non-clostridial anaerobic/crepitant cellulitis)	—
Gram-positive rods	Acute presentation	Clostridial myonecrosis (gas gangrene)	*Clostridium perfringens* (traumatic cases); *Clostridium septicum* (spontaneous cases)
Gram-positive rods	Indolent presentation	Clostridial (anaerobic) cellulitis	*C. perfringens* (more frequent); *C. septicum* (less frequent)
Absence of gas in soft tissue (radiographic imaging)			
Microbial category	**Clinical entity**	**Causative organisms/Notes**	
Gram-positive cocci (increasing [26])	Necrotizing fasciitis type II (monomicrobial)	Group A *Streptococcus* or other beta-hemolytic streptococci (Groups C–G; increasing incidence)	
—	—	*Staphylococcus aureus* (MSSA or MRSA; less common but rising, up to 16% [2])	
—	Necrotizing myositis	Group A *Streptococcus* or other beta-hemolytic streptococci	
—	—	*Enterococcus* species	
Gram-negative rods	—	*Aeromonas* species (freshwater exposure)	
—	—	*Vibrio* species (saltwater exposure; associated with chronic liver disease and diabetes mellitus)	
—	—	Enterobacteriaceae and non-fermenting bacteria (immunocompromised patients [27-29])	
Rare etiologies	—	*Mycobacterium tuberculosis* [30]	
—	—	Fungal infections	

(1) Gram-positive cocci, gram-positive rods, gram-negative cocci, and gram-negative rods. *S. pneumoniae* is exceptionally prevalent in patients with extreme age and comorbidities [31]; (2) Risk factors for MRSA: local endemicity, long-stay care facility, chronic dialysis, permanent transcutaneous medical device, previous MRSA infection or colonization, children under 2, athletes, parenteral drug users, military, veterinarians, and institutionalized patients [28, 32].

Clinical features

The foremost element of the clinical picture is a necrotizing infection that advances at a breakneck pace. Those afflicted often provide a narrative of a

dental infection that was either dismissed or failed to respond to antimicrobial measures. In some of these instances, the use of nonsteroidal anti-inflammatory agents and orally administered antibiotics may mask the symptomatology, delaying recognition. Pyrexia does not manifest universally, and early on, patients—above all those whose immune response is blunted, as seen in diabetes—can appear deceptively well from a systemic standpoint. As the individual's constitutional state declines, tumefaction, rubor, and fluid engorgement of the cervical/submandibular territory generally supervene. The demarcations between involved and uninvolved tissues are commonly indistinct. A hallmark is pain and exquisite tenderness whose severity far exceeds what the visible extent of disease would suggest. Diminished cutaneous perception or outright sensory loss has also been noted at a more evolved stage, consequent to full-thickness skin necrosis [28]. The finding of palpable crackling on physical examination is another telltale local clue, indicating the activity of gas-generating bacteria. Wang *et al*. [29] cataloged the successive cutaneous stigmata as the pathological process unfolds: (1) soreness, erythema, increased warmth, and induration; (2) formation of vesicles and larger bullous lesions; and (3) subcutaneous emphysema on palpation, tissue devitalization, and regional numbness. As the disease progresses, a rising heart rate or falling blood pressure becomes a characteristic systemic manifestation [2].

Diagnosis

The diagnosis of CNF hinges on integrating findings from the bedside examination, radiographic studies, and serum biomarkers **(Table 3)**. Direct operative inspection is the most sensitive and specific modality for verifying or refuting NF [20] and should be pursued whenever the diagnosis is entertained. Histopathological examination routinely demonstrates necrosis involving fascial planes and muscle bundles, together with a dense infiltrate of polymorphonuclear leukocytes. Abundant gram-positive organisms can be identified abutting muscle fibers in infections of group A streptococcal origin, whereas a clostridial source generally yields more accentuated tissue edema and gaseous distention. Angiothrombotic occlusion of medium and small-caliber vascular channels is also a pathological signature [13]. The roster of alternative diagnoses to consider in CNF is condensed in **Table 4**.

Table 3. Diagnosis of CNF.

Clinical features (local and systemic signs with rapid progression)	**Laboratory findings (non-specific but supportive)**	**Radiological findings**
Local manifestations	Leukocytosis with left shift	Presence of gas within soft tissues
Facial or cervical swelling, edema, erythema, and increased warmth	Elevated C-reactive protein (CRP) and/or erythrocyte sedimentation rate (ESR)	Absent or heterogeneous enhancement following IV contrast administration
Crepitus with or without overlying skin necrosis	Coagulation abnormalities (coagulopathy)	Fluid collections
Severe localized pain	Hyponatremia	Inflammatory changes extending beneath the fascial planes
Blister formation and bullae	Metabolic acidosis	—
Systemic manifestations	Elevated serum creatinine, lactate, creatine kinase (CK), and aspartate aminotransferase (AST)	—
Fever	—	—
Hemodynamic instability	—	—

Abbreviations: CK = creatine kinase; AST = aspartate aminotransferase.

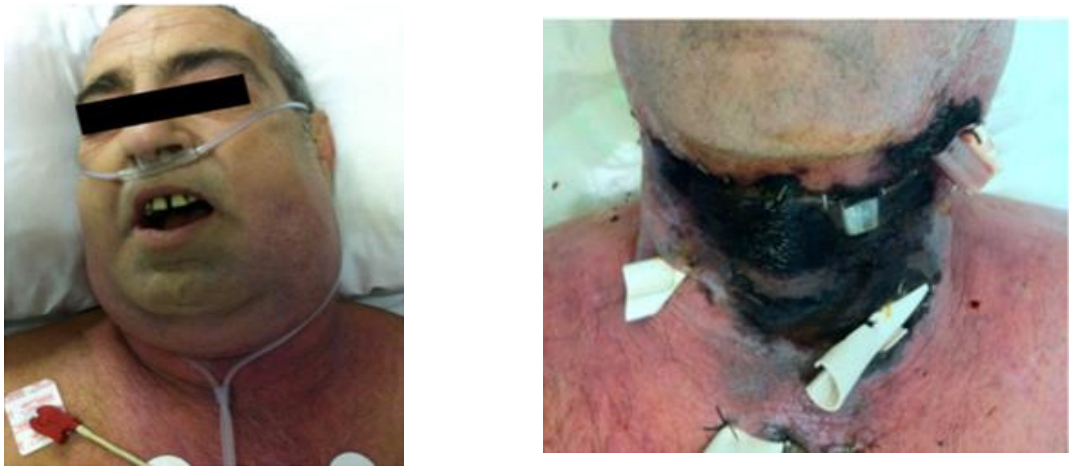
Table 4. Differential Diagnosis of CNF.

Condition	**Key clinical features/Distinguishing characteristics**
Cellulitis	Usually, it does not present with hemodynamic instability; fever may be present, but systemic compromise is uncommon.
Pyoderma gangrenosum	Characterized by slow progression with low likelihood of sepsis; strong association with inflammatory bowel disease. Does not clinically mimic cellulitis. Typical findings include a violaceous ulcer border. Fascial planes demonstrate normal resistance to surgical dissection. Clinical worsening occurs after surgical intervention, with no response to antibiotic therapy. Blood and tissue cultures are typically negative.
Gas gangrene (clostridial myonecrosis)	May occur spontaneously or following traumatic injury. Caused by Gram-positive rod bacteria. In severe cases, limb amputation may be required rather than simple debridement.

Pyomyositis	Infection characterized by abscess formation within skeletal muscle, most commonly caused by *Staphylococcus aureus* and generally associated with lower systemic toxicity compared to necrotizing infections.
Deep venous thrombosis (cervical involvement context)	Often associated with prior neck interventions such as surgery, puncture procedures, drug use, or trauma. May present with sore throat sensation, perceived neck swelling, restricted neck movement, and compensatory head tilting.

Clinical diagnosis

CNF warrants strong consideration in any patient who develops facial or cervical/submandibular puffiness, tissue edema, cutaneous rubor **(Figure 1)**, and systemic signs such as temperature elevation and cardiovascular instability. The concomitant finding of crackling on palpation, a rapidly evolving clinical picture, and agonizing pain wildly out of step with the apparent degree of involvement should trigger immediate alarm. Because CNF is rare, many surgeons are unfamiliar with its presentation. Thus, securing a diagnosis in the early window is inherently difficult. Moreover, distinguishing CNF from non-necrotizing deep neck space infections is especially problematic at the outset, given the near-identical initial clinical presentations of these two pathologies [9, 10]. A correct diagnosis at the first point of contact is achieved in fewer than 40% of cases [11]. The development of vesicles and large, fluid-filled bullae has been flagged as a pivotal clue that helps distinguish it from other non-necrotizing entities, including erysipelas and uncomplicated cellulitis. Palpable subcutaneous gas accompanied by full-thickness skin loss is a definitive diagnostic sign, yet it emerges only late in the disease course [29].



a) b)

Figure 1. Patient A: (a) Necrotizing fasciitis arising from a dental source in a 51-year-old man harboring undiagnosed diabetes mellitus and a smoking burden of 30 cigarettes each day. He self-medicated at home using amoxicillin/clavulanic acid and ibuprofen and presented to medical care only once the illness had progressed to an advanced stage. He was in a compromised general condition, displaying submandibular tumescence and erythema that descended to the clavicular level, together with palpable crepitus across the anterior neck and supraclavicular zone. His temperature was 37°, and he was tachycardic and hypotensive. Investigations showed a leukocyte count of 14.8 per mm³, creatinine 2.29 mg/dL, urea 170 mg/dL, sodium 121 mmol/L, and glucose > 800 mg/dL. The patient was taken to the operating room within 5 hours of arrival. No nonviable skin was visible either on admission or after the index operation. Accordingly, a tissue-sparing approach was selected at the outset, limited to surgical drainage and copious irrigation. He was subsequently transferred to the intensive care unit. His trajectory deteriorated into septic shock complicated by acute renal shutdown and diabetic ketoacidosis. (b) At 12 hours following the initial surgical drainage, patches of frankly necrotic skin materialized, necessitating a return to the operating theatre. Three further procedures, including wide excision of all devitalized tissue, were required. The diagnosis of necrotizing fasciitis was ultimately confirmed histopathologically, and microbiological cultures yielded *Streptococcus* sp. and *Candida albicans*. This illustrative case was previously reported by Leyva *et al*. [30].

When the constellation of clinical findings points toward necrotizing fasciitis but a degree of uncertainty lingers, proceeding straight to surgical exploration must be contemplated without procrastination. Some practitioners endorse the bedside finger test, which lays bare a lack of bleeding, disintegrating tissue

consistency, and a thin, murky fluid resembling dishwater [33, 34]. Direct intraoperative visualization of the wound uncovers a dull, grayish, nonviable fascial layer and surrounding soft tissue, an absence of hemorrhage, dissolution of normal subcutaneous fat architecture, and negligible tissue resistance when probed [2, 35]. A pungent, deeply offensive stench is another hallmark, generally signaling the proliferation of anaerobic flora. Tissue specimens for histology must be taken from zones that have not yet become frankly necrotic; samples must likewise be forwarded for microbiological analysis, including Gram staining, antimicrobial susceptibility profiles, and culture.

Laboratory tests

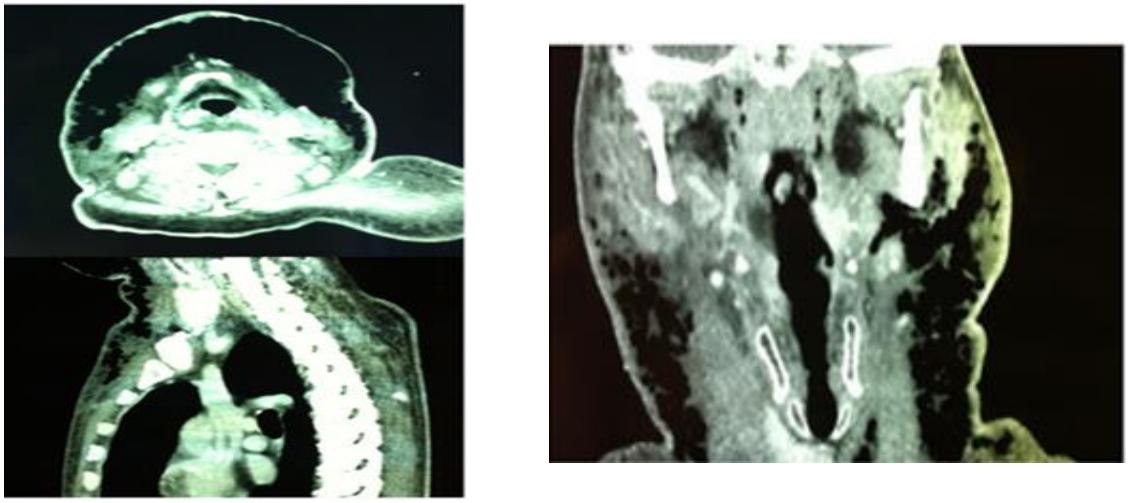
Blood test abnormalities tend to be broadly non-specific. A pronounced neutrophilic leukocytosis with a leftward shift is common; however, roughly 20% of individuals exhibit a leukocyte count within the reference range. Metabolic acidosis, deranged coagulation indices, hyponatremia, elevated inflammatory biomarkers such as C-reactive protein, and rising serum creatinine, along with lactate concentrations, may all be observed. Raised serum creatine kinase or aspartate aminotransferase levels intimate a deeper infectious process that has breached muscle or fascial planes (as opposed to a superficial cellulitis) [18]. Some investigative work has also explored the potential contribution of serum procalcitonin measurement as a diagnostic supplement [36, 37], proposing that a low value may effectively rule out NF during the nascent stages, when the clinical picture and physical examination are similarly reassuring.

The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) is a point-based stratification tool originally constructed by Wong *et al*. [38] with the intent of facilitating the differentiation of NF from a spectrum of other soft-tissue infections using six routine biochemical parameters (leukocyte count, hemoglobin, serum sodium, glucose, creatinine, and C-reactive protein levels). The cumulative tally ranges from 0 to 13, with scores of 6–7 corresponding to a predicted NF probability of 50%–75%, and scores of 8–13 corresponding to a probability exceeding 75%. A revised variant of the LRINEC has subsequently been put forward [39], which folds in co-existing diabetes mellitus and renal impairment and swaps conventional C-reactive protein for the high-sensitivity assay. Among the individual LRINEC constituents, glucose and C-reactive protein are both recognized for their value in predicting mortality in critically ill populations, particularly in the context of sepsis, uncontrolled hyperglycemia, and decompensated acute kidney injury [20]. In addition, Kim *et al*. [9], through their systematic review and meta-analysis centered on NF of the head and neck, calculated a sensitivity and specificity of 75% and 85%, respectively, when deploying an LRINEC threshold of six, versus 17% and 96%, respectively, with a threshold of eight. On this basis, they endorse using the score as a complementary decision aid rather than a standalone diagnostic instrument and argue that combining CT imaging with an LRINEC cutoff of 6 could facilitate earlier recognition of CNF. Certain authors maintain that LRINEC should never be relied on to rule out NF, citing the tool's erratic sensitivity [40, 41]. Fernando *et al*. [41] established, through their own systematic review and meta-analysis, that an LRINEC score of 6 (categorized as "moderate" risk) exhibited disappointingly low sensitivity for detecting NF, paired with only modest specificity—a performance profile notably inferior to that documented in the tool's validation cohort. They further noted that raising the threshold to an LRINEC score of 8 ("high" probability of NSTI) improved specificity but simultaneously led to a steep decline in sensitivity. These authors stress the critical importance of acknowledging the sensitivity-related shortcomings inherent to the LRINEC score, particularly because the score is derived from laboratory data, which may unintentionally delay the timeline to definitive surgical intervention and thereby contribute to inferior clinical outcomes.

Imaging studies

Computed tomography (CT) examination may reveal diffuse expansion of the subcutaneous fat and the cervical fascial layers, loculated fluid accumulations occupying the compartments of the neck, and pockets of gas. The mere demonstration of gas does not constitute a pathognomonic finding for necrotizing fasciitis, since it can accompany other musculoskeletal infectious processes [42]; by the same token, its absence should never be used to overrule the diagnosis when clinical suspicion persists. Gas entrapped within fluid collections that dissect along subfascial planes serves as the radiographic hallmark of NF [43]. Becker *et al*. [44] enumerated the invariant diagnostic elements observable on CT studies obtained from subjects whose NF was ultimately verified by histology: (1) cellulitis (diffuse expansion of the dermal and subcutaneous planes together with reticular pattern enhancement of the subcutaneous adipose layer); (2) fasciitis (thickening and/or contrast enhancement of the cervical fasciae); (3) myositis (asymmetric expansion or hyperenhancement of the cervical musculature); and

(4) fluid pooling across multiple neck compartments. Gas inclusions and mediastinal extension were found to be inconsistent observations. The superficial cervical fascia was invariably implicated, and thickening and/or enhancement of the sternocleidomastoid muscle was a universal finding. The CT study of our first patient showed copious, uninterrupted gas collections cascading from the mandibular region down to the uppermost segment of the anterior thoracic wall **(Figure 2)**. This stands in sharp contrast to the observations of Becker *et al*. [44], in which gas was confined within fluid pockets in every instance.



a) b)

Figure 2. Patient A: Preoperative CT scan showing abundant gas extending subcutaneously from the upper anterior chest wall and the anterolateral neck bilaterally up to the paramandibular region.

Because the infective process can track downward and invade the mediastinal space, this anatomical region demands routine scrutiny on a thoracic CT. CT imaging further enables reassessment of newly developed or persistent fluid collections or regions of advancing disease when the postoperative clinical trajectory deviates from the expected course after debridement.

The sensitivity ascribed to CT has been placed at 80%, coupled with poor specificity [9, 10]; however, a systematic review dating from 2019 [41] arrived at a sensitivity of 94.3% for the combined criteria of fascial enhancement, fascial edema, or fascial gas, alongside a specificity of 93.3% for the isolated presence of fascial gas. Whenever uncertainty prevails, bedside clinical suspicion should be accorded greater weight. Martinez *et al*. [45] reported a sensitivity of 100%, specificity of 98%, positive predictive value of 76%, and negative predictive value of 100% after a retrospective analysis of CT scans and clinical files from 184 individuals referred with suspected necrotizing soft-tissue infection. Building upon their results, they concluded that an unremarkable intravenous contrast-enhanced CT study can be relied upon to safely obviate the need for operative exploration in patients whose presentation raises concern for necrotizing soft-tissue infection.

Magnetic resonance imaging (MRI) outperforms CT [42] in characterizing soft-tissue detail. It can discriminate this entity from non-necrotizing cellulitis [46] (a condition amenable to pharmacological treatment alone), yet it is seldom readily available in the emergency care environment. MRI features that lend weight to a necrotizing fasciitis diagnosis comprise extensive encroachment upon the deep intermuscular fascial planes (highly sensitive but poorly specific), a caliber exceeding 3 mm in thickness, and a partially or fully absent signal uptick within the thickened fascial bands on sequences acquired following gadolinium administration (yielding relatively robust sensitivity and specificity) [47]. Nevertheless, given MRI's exceedingly high sensitivity for flagging deep fascial irregularities, the generated images warrant a cautious, nuanced interpretation [47].

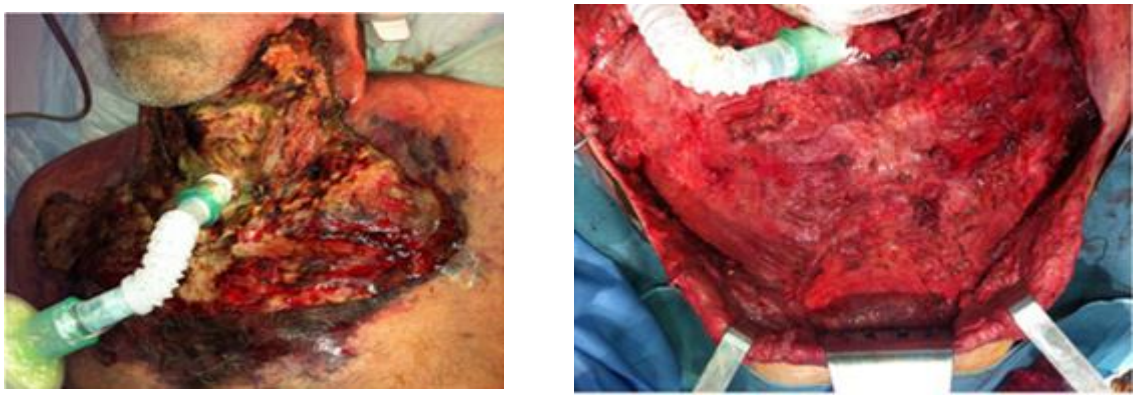
Kwee and Kwee [48] conducted a systematic review evaluating the diagnostic performance of MRI relative to CT in suspected NF. They ultimately concluded that MRI's edge lies in its superior soft-tissue discrimination, while CT offers faster acquisition and greater sensitivity for soft-tissue gas detection. Still, they were unable to determine which cross-sectional modality offers the highest diagnostic yield.

According to our own institutional practice, the ready accessibility and rapid throughput of CT scanning render this technique more clinically advantageous than MRI when formulating management strategies for CNF.

Management

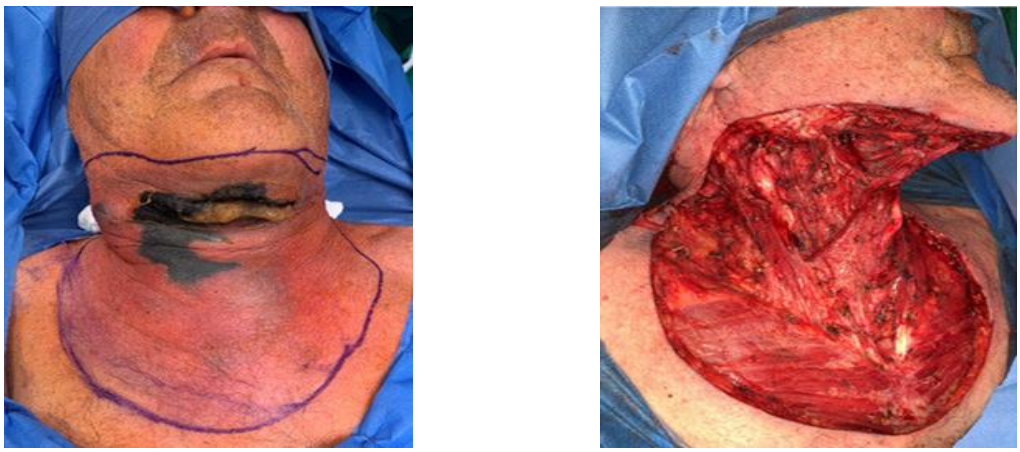
Early, wide-ranging surgical excision of all nonviable tissue represents the bedrock of effective therapy. Repeat operative interventions are typically mandated as the infectious process marches forward. Some groups have even advocated a protocolized return to the operating room for a second-look procedure [49]. When mapping out the resection margins, the surgeon should remain cognizant that immediately neighboring, apparently uninvolved tissues are apt to harbor early, diffuse microvascular thrombosis and vasculitic infiltration [50]. Tissue samples must be forwarded to both the histopathology and microbiology services for tissue-based confirmation, Gram staining, cultivation, and susceptibility assessment.

In the case of our first subject, a more restrained, tissue-preserving initial strategy was deliberately pursued. Since neither the cutaneous envelope nor the underlying fascia showed macroscopic evidence of necrosis at the time of the inaugural surgical exploration, the procedure was limited to skin incisions and fasciotomies to decompress and drain the purulent exudate, followed by copious irrigation. The incisions and the surrounding integument were monitored with vigilant attention, and the patient was expeditiously brought back to the operating theatre at the very first appearance of necrotic changes **(Figure 1b)**. **Figure 3** captures the advancing wave of tissue necrosis following the second operative takeback. By contrast, our second patient arrived at the emergency department with a well-demarcated patch of frank skin necrosis and blistering bullae; in this instance, an immediate, unsparing resection of all compromised skin, investing fascia, and cervical adipose tissue was performed in a single, decisive procedure **(Figure 4)**.



a) b)

Figure 3. Patient A: (a) Progression of skin and muscle necrosis after the second surgery, and (b) Final extension of debridement before skin grafting.



a) b)

Figure 4. Patient B: (a) A 62-year-old male with a background of chronic ischaemic heart disease and suboptimally regulated diabetes mellitus despite a four-drug regimen. Active smoker. A dental infection

originating from a left upper tooth had been managed with amoxicillin/clavulanic acid over the preceding 14 days. He arrived at the emergency room exhibiting subcutaneous crackling, tumefaction, and erythema across the anterolateral neck and upper chest, along with a devitalized cutaneous patch, bullae, and purulent drainage. The laboratory panel revealed the following: glucose 125 mg/dL, white blood cell count 27 per mm^3, hemoglobin within normal limits, creatinine 1.81 mg/dL, C-reactive protein 385 mg/L, sodium 126 mmol/L; urea exceeded the upper limit of normal by a factor of three, and hepatic function indices were similarly elevated. Parenteral broad-spectrum antimicrobials were initiated, and the patient was transported to the operating theatre within 3 hours of arrival. (b) Radical debridement was performed as illustrated in the image. A tracheotomy was undertaken to protect the airway definitively. The patient became acidotic and septic during his intensive care stay and required progressively escalating doses of vasopressor agents. Intravenous immunoglobulin was infused. The patient succumbed 16 hours after presentation. Histopathological examination substantiated the diagnosis of NF. The microbiology analyses identified *Stenotrophomonas maltophilia* and *Enterobacter cloacae*; *Staphylococcus epidermidis* and *Streptococcus constellatus* were recovered from aerobic culture media, while *Eggerthia catenaformis* and *Prevotella nigrescens* were isolated under anaerobic conditions.

A temporary tracheotomy is performed on a routine basis to secure a dependable airway in patients dependent on mechanical ventilation or in those for whom multiple surgical procedures under general anesthesia are anticipated. In our hands, tracheotomy can be strategically postponed when there is dense swelling or frankly purulent loculations occupying the anterior compartment of the neck; the procedure can then be executed, if it remains necessary, during a subsequent return to the operating room, after those loculations have been drained and the anterior cervical field has been cleared of nonviable tissue. By adopting this staged strategy, contamination or downward seepage of infected fluid into the tracheal lumen may be circumvented.

The initial empirical antibiotic backbone must deliver activity against Gram-positive, Gram-negative, and anaerobic pathogens [28, 49, 51]. It is vital to bear in mind that the most appropriate antimicrobial strategy is determined by the local antibiogram and region-specific therapeutic guidelines, which should be consulted before launching any empiric protocol. The following may be regarded as a broad template:

- A carbapenem: Imipenem 1 g dosed every 6 to 8 hours or Meropenem 2 g intravenously every 8 hours (administered as an extended infusion) or Piperacillin-tazobactam 4.5 g every 6 hours PLUS an agent possessing reliable efficacy against methicillin-resistant *Staphylococcus aureus*: Vancomycin (a 20 mg/kg loading dose with subsequent therapeutic drug monitoring) or Daptomycin (10 mg/kg every 24 hours) PLUS Clindamycin 600 to 900 mg intravenously every 8 hours or Linezolid 600 mg intravenously every 8 hours as initial therapy—accompanied by serum level monitoring—in the setting of clindamycin resistance (to harness its antitoxin properties directed against toxin-elaborating strains of beta-haemolytic streptococci and *S. aureus*).

As soon as Gram stain morphology, culture speciation, and antimicrobial susceptibility profiles are returned, the antibiotic regimen should be refined accordingly.

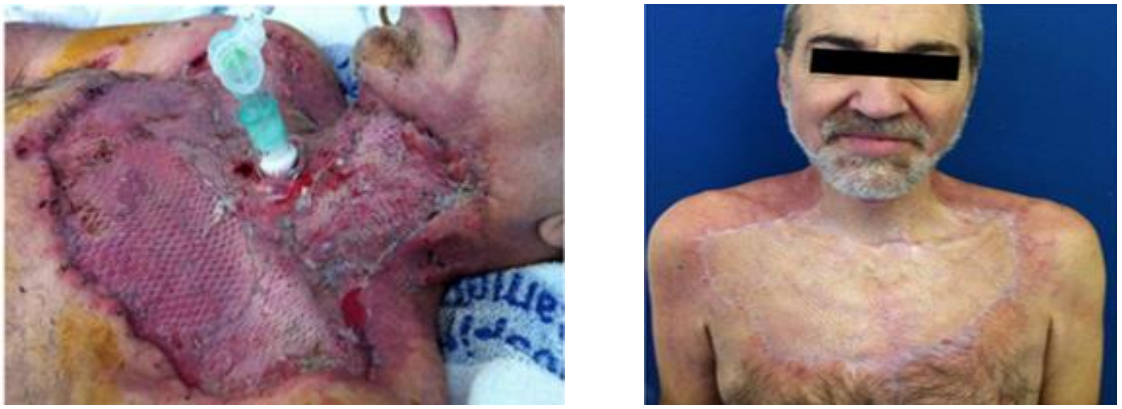
Circulatory support delivered in the intensive care unit should target hemodynamic collapse through vigorous volume resuscitation and the administration of vasoactive infusions. Such patients may exhibit exceptionally high fluid requirements and severe albumin depletion, rendering albumin-based colloid replacement a potential necessity. Serial hematocrit measurements should track the need for packed red cell transfusion (a parameter that outperforms hemoglobin concentration in this clinical context [18]). Concurrently, any acid-base derangements and elevations in blood glucose must be closely monitored and managed.

The place of hyperbaric oxygen therapy remains contested [18, 52]. Furthermore, access to this modality may be restricted in many institutions, and the practical complexities of relocating a systemically unstable patient for hyperbaric treatment appear formidable; it should never, under any circumstances, be permitted to postpone definitive surgical intervention. The adjunctive administration of intravenous immune globulin likewise occupies a position of considerable complexity and ongoing dispute [53, 54].

Lastly, given the scarcity of these life-threatening infections, channeling NF management to referral centers with a high annual caseload of such pathologies substantially improves the odds of a favorable outcome [52].

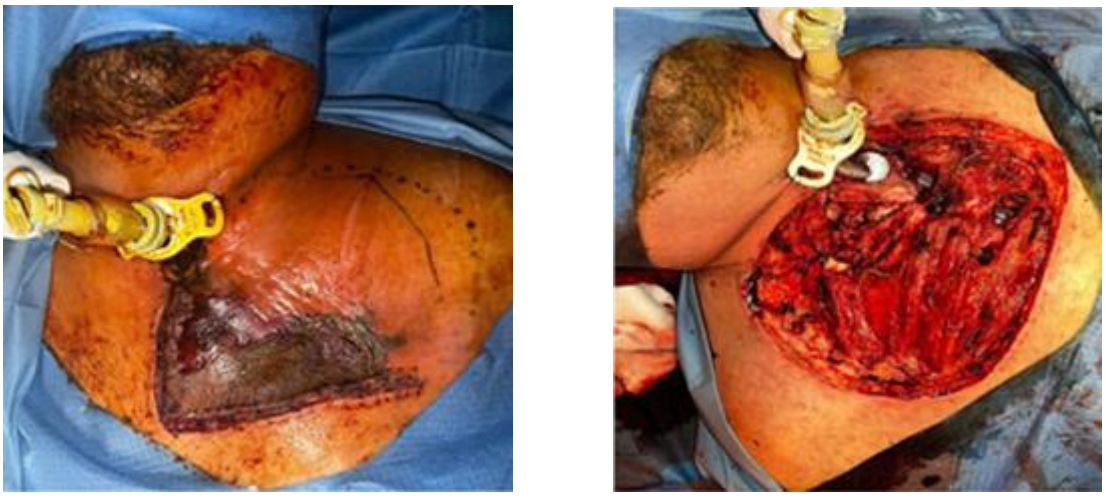
Wound dressings ought to be renewed at 8-hour intervals to enable ongoing surveillance for the appearance of fresh necrotic tissue and to sustain a moist, non-contaminated wound environment. Saline

lavage and gauze coated with topical antimicrobial agents are applied. Once the infectious process has been suppressed, the patient reaches a hemodynamically stable plateau, and the wound surface is clean and populated with granulation tissue, the reconstructive phase can be timetabled. According to the dimensions of the preceding surgical defect and the extent to which critical structures—exemplified by the trachea or the great vessels of the neck—have become exposed, the surgeon may elect to reconstruct the tissue gap using either skin grafts or composite tissue flaps. Split-thickness or full-thickness skin grafts are the reconstructive modality of choice when debridement is limited to the skin and underlying fascia. The thigh region is ordinarily the preferred donor territory. Such procedures are minimally invasive, well accepted by patients, and carry a low complication burden. In the setting of larger defects, a pedicled or microvascular free flap may be required to provide adequate tissue bulk. Under these circumstances, a pedicled latissimus dorsi musculocutaneous flap or an anterolateral thigh free flap frequently represents the optimal solution. In the case of our first patient, split-thickness skin grafts procured from the thigh were selected, as the debridement had occasioned little loss of muscle mass and the resultant defect was superficial and of consistent thickness **(Figure 5)**. For our third patient, the operative sequence entailed excision of the sternoclavicular joint and a portion of the pectoralis major muscle, along with the affected skin and fascial layers. A pedicled latissimus dorsi flap was employed to provide coverage for the resultant defect **(Figure 6)**. To achieve direct closure at the harvest site, a diminutive skin island was elevated, and the exposed remnant of the latissimus dorsi muscle was simultaneously covered with a split-thickness skin graft in a single operative session.

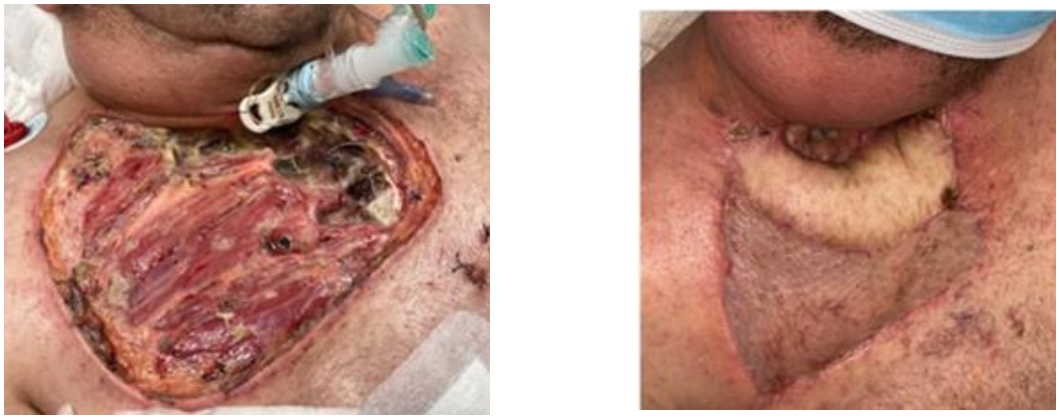


a) b)

Figure 5. Patient A: (a) Split-thickness skin grafts were harvested to reconstruct the defect 39 days after the first surgery, and (b) Outcome 6 months after reconstruction. This clinical case was previously reported by Leyva *et al*. [30].



a) b)



c) d)

Figure 6. Patient C: (a) was a 54-year-old male transferred from another hospital with cellulitis and abscessification of the thoracic wall and mediastinitis needing thoracic surgery. The patient already had a tracheotomy due to a supraglottic cancer, treated with chemo-radiotherapy. *S.* maltophilia, *C. albicans*, *P. aeruginosa*, *S. constellatus*, *Serratia liquefaciens*, *Prevotella* spp., and *Chryseobacterium indologenes* were isolated. Necrotizing fasciitis and mediastinitis were confirmed by histopathology; (b) The pectoralis major muscle was partially resected together with the medial portion of the right clavicle (white arrow), the superolateral portion of the sternum, and the sternocostal joints of the first and second right ribs, in 3 different surgeries; (c) Appearance before reconstruction; (d) Outcome: a latissimus dorsi pedicled flap + skin graft was harvested for reconstruction 2 weeks after admission.

Synthesizing the available published evidence with our own accumulated clinical experience, we submit the following algorithmic framework for the orchestrated management of CNF:

Management summary **(Figure 7)**:

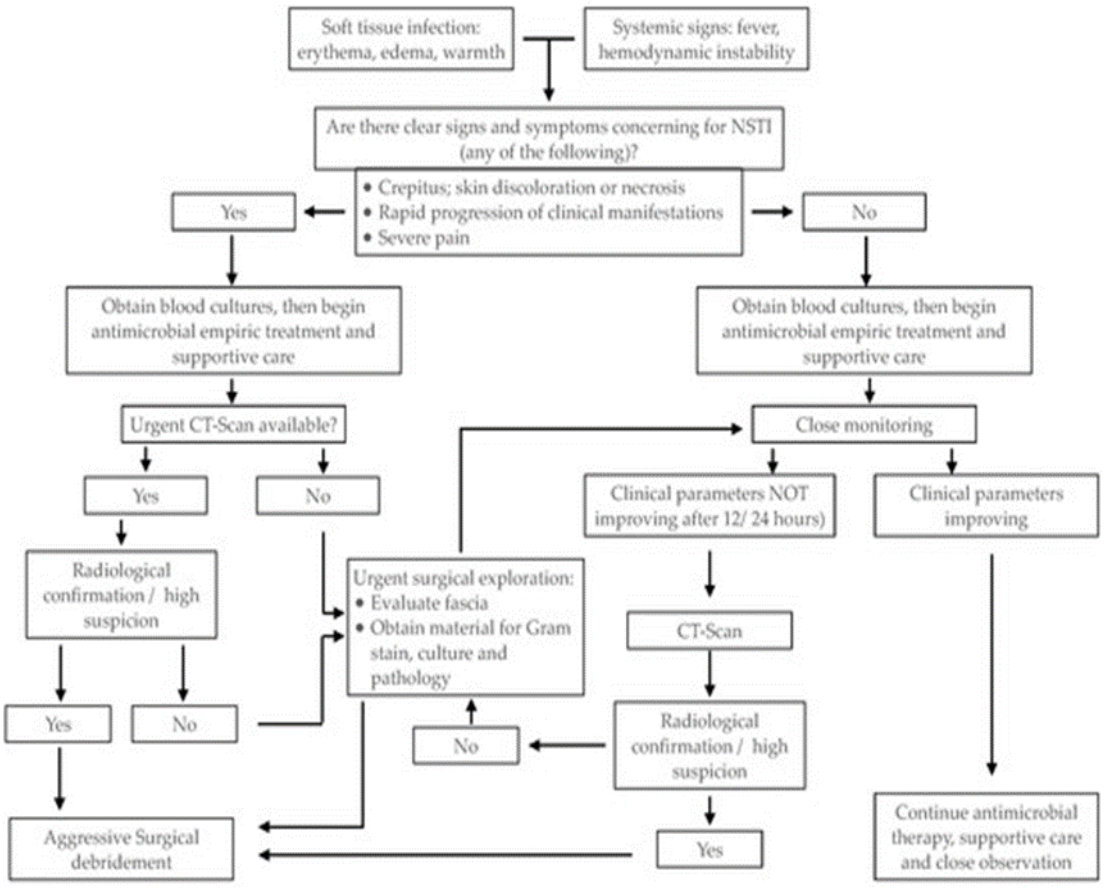


Figure 7. Management of CNF.

- The diagnosis ought to be established without hesitation. When the available clinical evidence adequately points toward NF, proceeding directly to surgical exploration takes precedence over supplementary laboratory analyses or imaging investigations that risk postponing operative intervention;
- Surgical debridement must be undertaken without any postponement (and without awaiting microbiology reports), entailing excision of all devitalized tissues (skin, fascia, muscle, and adipose tissue);
- Tracheotomy is carried out as a routine precaution to safeguard the airway and in situations where an extended stay in the intensive care setting is foreseen.
- Vigorous resuscitative efforts delivered by critical care specialists constitute a further cornerstone of NF management, alongside broad-spectrum antimicrobial agents targeting the most commonly implicated pathogens until culture results and Gram staining are returned.
- Return visits to the operating theatre are commonly required. The wounds must be closely monitored for signs of advancing disease, while laboratory parameters and vital signs are continuously reassessed. When uncertainty arises, a repeat CT examination should be obtained to evaluate for newly developed fluid collections or progression to descending mediastinitis.

Prognosis

Even with timely and appropriate therapeutic measures, the case fatality rate for CNF remains elevated. The death rate climbs among individuals who go on to develop streptococcal toxic shock syndrome or septic shock (38% and 45%, respectively) [18]. As the infectious process extends, tissue necrosis readily tracks downward along fascial routes into the mediastinum. The superimposition of descending necrotizing mediastinitis pushes the incidence of sepsis upward, from 7% to 22%, and drives mortality from 31% to 41%. When both descending necrotizing mediastinitis and sepsis become established, the mortality attributable to CNF can soar to 64% [55].

Patients with compromised immune defenses and those with long-standing medical conditions are at heightened risk of acquiring CNF. Diabetes mellitus ranks as the most prevalent comorbid condition [14, 56-58]. Such individuals also experience complications more frequently and endure more protracted inpatient stays [51], as exemplified by the clinical course of our first patient. Alcohol dependence or substance misuse, the existence of malignant disease, treatment with corticosteroids or HIV seropositivity [14], renal dysfunction, hepatic pathology, and obesity [32] have likewise been linked.

Additional variables have been correlated with a greater likelihood of death, among them a leukocyte count surpassing 30,000 per microliter, serum creatinine exceeding 2 mg/dL, age older than 60 years, streptococcal toxic shock syndrome, clostridial etiology, a delay in reaching the operating room of greater than 24 hours, and infection extending to the head, neck, thorax, or abdominal territory [56, 59]. An analysis of 89 patients identified a surgical delay exceeding 24 hours from hospital presentation as the sole independent predictor of death in a multivariate logistic regression model [56]. Progressive age and the coexistence of two or more associated comorbid illnesses were also found to impact survival in univariate analysis negatively.

Complications

Descending necrotizing mediastinitis (DNM) was documented in 255 out of 808 cases (31.56%) within the systematic review published by Gunaratne *et al*. [14] and stemmed from an odontogenic source in exactly half of those instances. This figure contrasts with the findings of the systematic review by Prado-Calleros *et al*. [60], which reported the predominant origin as pharyngeal (45%), followed by odontogenic sources (36%). Once firmly entrenched, DNM can in turn precipitate septic shock with multiorgan dysfunction (if not already manifest), pneumonia, airway compromise, catastrophic hemorrhage, cranial nerve palsies, empyema, or bronchocavitary fistulae [60, 61].

Serious vascular sequelae encompass thrombosis of the internal jugular vein, septic thrombophlebitis of the jugular vein (Lemierre's syndrome), necrotic breakdown of the carotid sheath with consequent rupture, and aneurysmal dilatation of the carotid artery [51].

Conclusion

Cervical necrotizing fasciitis represents an infrequent yet critical surgical emergency. Although the clinical diagnosis becomes self-evident at a late phase, distinguishing it from other, more innocuous, deep neck infections at the time of first presentation can be extremely taxing. Cutaneous signs are tardy manifestations and do not faithfully mirror the true extent or gravity of the underlying infection. Hence, even though palpable crepitus and full-thickness skin loss are specific diagnostic indicators, these features—along with other systemic cues—may be altogether absent. A CT scan can help secure an early diagnosis

and is indispensable for tracking the progression of the infectious process. The mediastinum should be methodically evaluated on every occasion. Prompt and radical surgical debridement, broad-spectrum antibiotic coverage, and intensive organ support measures constitute the bedrock of CNF treatment. Even with optimal management, mortality remains substantial.

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Conflict of Interest: None

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Ethics Statement: The study was conducted in accordance with the Declaration of Helsinki; the study did not require ethical approval.
Written informed consent has been obtained from the patients to publish this paper.

References

1. Wilson B. Necrotizing fasciitis. Am Surg. 1952;18:416-31.
2. McGurk M. Diagnosis and treatment of necrotizing fasciitis in the head and neck region. Oral Maxillofac Surg Clin North Am. 2003;15:59-67.
3. Adams F. The genuine works of Hippocrates. 1771st ed. London: Sydenham Society; 1849.
4. Das DK, Baker MG, Venugopal K. Increasing incidence of necrotizing fasciitis in New Zealand: a nationwide study over the period 1990 to 2006. J Infect. 2011;63:429-33. doi:10.1016/j.jinf.2011.08.002
5. Thean LJ, Jenney A, Engelman D, Romani L, Wand H, Mudaliar J, et al. Hospital admissions for skin and soft tissue infections in a population with endemic scabies: a prospective study in Fiji, 2018-2019. PLoS Negl Trop Dis. 2020;14:e0008887.
6. Centers for Disease Control and Prevention. Necrotizing fasciitis: information for clinicians [Internet]. [cited 2023 May 15]. Available from: https://www.cdc.gov/groupastrep/diseases-hcp/necrotizing-fasciitis.html
7. Hasham S, Matteucci P, Stanley PRW, Hart NB. Necrotising fasciitis. BMJ. 2005;330:830-3.
8. Sepúlveda A, Sastre N. Necrotizing fasciitis of the face and neck. Plast Reconstr Surg. 1998;102:814-7.
9. Kim DH, Kim SW, Hwang SH. Application of the laboratory risk indicator for necrotizing fasciitis score to the head and neck: a systematic review and meta-analysis. ANZ J Surg. 2022;92:1631-7.
10. Sideris G, Sapountzi M, Malamas V, Papadimitriou N, Maragkoudakis P, Delides A. Early detecting cervical necrotizing fasciitis from deep neck infections: a study of 550 patients. Eur Arch Otorhinolaryngol. 2021;278:4587-92.
11. Salati SA. Necrotizing fasciitis – a review. Pol Przegl Chir. 2022;95:1-8.
12. Nuwayhid ZB, Aronoff DM, Mulla ZD. Blunt trauma as a risk factor for group A streptococcal necrotizing fasciitis. Ann Epidemiol. 2007;17:878-81.
13. Stevens DL, Bryant AE, Goldstein EJ. Necrotizing soft tissue infections. Infect Dis Clin North Am. 2021;35:135-55.
14. Gunaratne DA, Tseros EA, Hasan Z, Kudpaje AS, Suruliraj A, Smith MC, et al. Cervical necrotizing fasciitis: systematic review and analysis of 1235 reported cases from the literature. Head Neck. 2018;40:2094-102.
15. Pitché P, Diata AB, Faye O, Tounkara TM, Niamba P, Mouhari-Toure A, et al. Risk factors associated with necrotizing fasciitis of the lower limbs: a multicenter case-control study. Ann Dermatol Venereol. 2021;148:161-4.
16. Hamilton SM, Bayer CR, Stevens DL, Bryant AE. Effects of selective and nonselective nonsteroidal anti-inflammatory drugs on antibiotic efficacy of experimental group A streptococcal myonecrosis. J Infect Dis. 2014;209:1429-35.
17. Weng TC, Chen CC, Toh HS, Tang HJ. Ibuprofen worsens Streptococcus pyogenes soft tissue infections in mice. J Microbiol Immunol Infect. 2011;44:418-23.
18. Stevens DL, Bryant AE. Necrotizing soft-tissue infections. N Engl J Med. 2017;377:2253-65.
19. Anaya DA, Dellinger EP. Necrotizing soft-tissue infection: diagnosis and management. Clin Infect Dis. 2007;44:705-10.
20. Lancerotto L, Tocco I, Salmaso R, Vindigni V, Bassetto F. Necrotizing fasciitis: classification, diagnosis, and management. J Trauma Acute Care Surg. 2012;72:560-6.
21. Miller LG, Perdreau-Remington F, Rieg G, Mehdi S, Perlroth J, Bayer AS, et al. Necrotizing fasciitis caused by community-associated methicillin-resistant Staphylococcus aureus in Los Angeles. N Engl J Med. 2005;352:1445-53.
22. Kaul R, McGeer A, Low DE, Green K, Schwartz B, Simor AE. Population-based surveillance for

group A streptococcal necrotizing fasciitis: clinical features, prognostic indicators, and microbiologic analysis of seventy-seven cases. Ontario Group A Streptococcal Study. Am J Med. 1997;103:18-24.
23. Darenberg J, Luca-Harari B, Jasir A, Sandgren A, Pettersson H, Schalén C, et al. Molecular and clinical characteristics of invasive group A streptococcal infection in Sweden. Clin Infect Dis. 2007;45:450-8.
24. Stevens DL, Bryant AE, Hackett SP, Chang A, Peer G, Kosanke S, et al. Group A streptococcal bacteremia: the role of tumor necrosis factor in shock and organ failure. J Infect Dis. 1996;173:619-26.
25. Stevens DL, Baddour L. Necrotizing soft tissue infections. UpToDate [Internet]. 2024 [cited 2024 Apr 4]. Available from: https://www.uptodate.com/contents/image?csi=3a7bd772-c906-476d-bf1c-80c663b6ddab&source=contentShare&imageKey=ID%2F116305
26. Hawkes M, Barton M, Conly J, Nicolle L, Barry C, Ford-Jones EL. Community-associated MRSA: superbug at our doorstep. CMAJ. 2007;176:54-6.
27. Borschitz T, Schlicht S, Siegel E, Hanke E, von Stebut E. Improvement of a clinical score for necrotizing fasciitis: 'pain out of proportion' and high CRP levels aid the diagnosis. PLoS One. 2015;10:e0132775.
28. Leiblein M, Marzi I, Sander AL, Barker JH, Ebert F, Frank J. Necrotizing fasciitis: treatment concepts and clinical results. Eur J Trauma Emerg Surg. 2018;44:279-90.
29. Wang YS, Wong CH, Tay YK. Staging of necrotizing fasciitis based on the evolving cutaneous features. Int J Dermatol. 2007;46:1036-41.
30. Leyva P, Herrero M, Eslava JM, Acero J. Cervical necrotizing fasciitis and diabetic ketoacidosis: literature review and case report. Int J Oral Maxillofac Surg. 2013;42:1592-5.
31. Parra Caballero P, Pérez Esteban S, Patiño Ruiz ME, Sanz SC, Vadillo JAG. Actualización en fascitis necrotizante. Semin Fund Esp Reumatol. 2012;13:41-8.
32. Hua C, Urbina T, Bosc R, Parks T, Sriskandan S, de Prost N, et al. Necrotising soft-tissue infections. Lancet Infect Dis. 2023;23:e81-94.
33. Livshits D, Sokup B, Farrell R, Jeong J. Finger test for the diagnosis of a critically ill patient with necrotizing fasciitis. J Emerg Med. 2022;63:102-5.
34. Lau JK, Kwok K, Hung Y, Fan C. Validation of finger test for necrotising soft tissue infection. J Orthop Trauma Rehabil. 2020. doi:10.1177/2210491720950932
35. Hua J, Friedlander P. Cervical necrotizing fasciitis, diagnosis and treatment of a rare life-threatening infection. Ear Nose Throat J. 2023;102:NP109-13.
36. Kishino T, Asai N, Ohashi W, Sakanashi D, Kato H, Shiota A, et al. Usefulness of serum procalcitonin for necrotizing fasciitis as an early diagnostic tool. J Infect Chemother. 2021;27:787-93.
37. Novoa-Parra CD, Wadhwani J, Puig-Conca MA, Lizaur-Utrilla A, Montaner-Alonso D, Rodrigo-Pérez JL, et al. Usefulness of a risk scale based on procalcitonin for early discrimination between necrotising fasciitis and cellulitis of the extremities. Med Clin (Barc). 2019;153:347-50.
38. Wong CH, Khin LW, Heng KS, Tan KC, Low CO. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med. 2004;32:1535-41.
39. Wu H, Liu S, Li C, Song Z. Modified laboratory risk indicator for necrotizing fasciitis (m-LRINEC) score system in diagnosing necrotizing fasciitis: a nested case-control study. Infect Drug Resist. 2021;14:2105-12.
40. Wilson MP, Schneir AB. A case of necrotizing fasciitis with a LRINEC score of zero: clinical suspicion should trump scoring systems. J Emerg Med. 2013;44:928-31.
41. Fernando SM, Tran A, Cheng W, Rochwerg B, Kyeremanteng K, Seely AJE, et al. Necrotizing soft tissue infection: diagnostic accuracy of physical examination, imaging, and LRINEC score: a systematic review and meta-analysis. Ann Surg. 2019;269:58-65.
42. Carbonetti F, Cremona A, Carusi V, Guidi M, Iannicelli E, Di Girolamo M, et al. The role of contrast enhanced computed tomography in the diagnosis of necrotizing fasciitis and comparison with the laboratory risk indicator for necrotizing fasciitis (LRINEC). Radiol Med. 2016;121:106-21.
43. Tso DK, Singh AK. Necrotizing fasciitis of the lower extremity: imaging pearls and pitfalls. Br J Radiol. 2018;91:20180093.
44. Becker M, Zbären P, Hermans R, Becker CD, Marchal F, Kurt AM, et al. Necrotizing fasciitis of

the head and neck: role of CT in diagnosis and management. Radiology. 1997;202:471-6.
45. Martinez M, Peponis T, Hage A, Yeh DD, Kaafarani HMA, Fagenholz PJ, et al. The role of computed tomography in the diagnosis of necrotizing soft tissue infections. World J Surg. 2018;42:82-7.
46. Kim MC, Kim S, Cho EB, Lee GY, Choi SH, Kim SO, et al. Utility of magnetic resonance imaging for differentiating necrotizing fasciitis from severe cellulitis: a magnetic resonance indicator for necrotizing fasciitis (MRINEC) algorithm. J Clin Med. 2020;9:3040.
47. Malghem J, Lecouvet FE, Omoumi P, Maldague BE, Berg BCV. Necrotizing fasciitis: contribution and limitations of diagnostic imaging. Joint Bone Spine. 2013;80:146-54.
48. Kwee RM, Kwee TC. Diagnostic performance of MRI and CT in diagnosing necrotizing soft tissue infection: a systematic review. Skeletal Radiol. 2022;51:727-36.
49. Stevens DL, Bisno AL, Chambers HF, Dellinger EP, Goldstein EJC, Gorbach SL, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis. 2014;59:147-59.
50. Andreasen TJ, Green SD, Childers BJ. Massive infectious soft-tissue injury: diagnosis and management of necrotizing fasciitis and purpura fulminans. Plast Reconstr Surg. 2001;107:1025-35.
51. Vieira F, Allen SM, Stocks RMS, Thompson JW. Deep neck infection. Otolaryngol Clin North Am. 2008;41:459-83.
52. Mladenov A, Diehl K, Müller O, von Heymann C, Kopp S, Peitsch WK. Outcome of necrotizing fasciitis and Fournier's gangrene with and without hyperbaric oxygen therapy: a retrospective analysis over 10 years. World J Emerg Surg. 2022;17:43.
53. Parks T, Wilson C, Curtis N, Norrby-Teglund A, Sriskandan S. Polyspecific intravenous immunoglobulin in clindamycin-treated patients with streptococcal toxic shock syndrome: a systematic review and meta-analysis. Clin Infect Dis. 2018;67:1434-6.
54. Kadri SS, Swihart BJ, Bonne SL, Hohmann SF, Hennessy LV, Louras P, et al. Impact of intravenous immunoglobulin on survival in necrotizing fasciitis with vasopressor-dependent shock: a propensity score-matched analysis from 130 US hospitals. Clin Infect Dis. 2017;64:877-85.
55. Sarna T, Sengupta T, Miloro M, Kolokythas A. Cervical necrotizing fasciitis with descending mediastinitis: literature review and case report. J Oral Maxillofac Surg. 2012;70:1342-50.
56. Wong CH, Chang HC, Pasupathy S, Khin LW, Tan JL, Low CO. Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality. J Bone Joint Surg Am. 2003;85:1454-60.
57. Arif N, Yousfi S, Vinnard C. Deaths from necrotizing fasciitis in the United States, 2003-2013. Epidemiol Infect. 2016;144:1338-44.
58. Pelletier J, Gottlieb M, Long B, Perkins JC. Necrotizing soft tissue infections (NSTI): pearls and pitfalls for the emergency clinician. J Emerg Med. 2022;62:480-91.
59. Huang KF, Hung MH, Lin YS, Lu CL, Liu C, Chen CC, et al. Independent predictors of mortality for necrotizing fasciitis: a retrospective analysis in a single institution. J Trauma. 2011;71:467-73; discussion 473.
60. Prado-Calleros HM, Jiménez-Fuentes E, Jiménez-Escobar I. Descending necrotizing mediastinitis: systematic review on its treatment in the last 6 years, 75 years after its description. Head Neck. 2016;38 Suppl 1:E2275-83.
61. Sumi Y. Descending necrotizing mediastinitis: 5 years of published data in Japan. Acute Med Surg. 2015;2:1-12.