ISSN: 2634 - 680X

Journal of Clinical Case Studies Reviews & Reports

SCIENTIFIC Research and Community

Review Article

Open Access

Necrotizing Fasciitis: Obscure Nature and Challenging Diagnosis (Review)

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ABSTRACT

Necrotizing fasciitis is a rapidly progressive synergistic aerobical-anaerobical deadly infection. It spreads quickly along the fascial planes leading to widespread soft tissue gangrene and systemic sepsis. Early diagnosis of the disease is critical to ensure timely and effective treatment, as delays significantly increase morbidity and mortality rates. Keeping high index of suspicion at initial presentation and raising clinical awereness are of paramount importance. Despite better knowledge and advancements in laboratory diagnostics, clinical judgment is still challenging and remains the cornerstone of early detection. This review discusses the key elements involved in the diagnostic evaluation of necrotizing fasciitis.

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Received: May 12, 2025; **Accepted:** May 19, 2025; **Published:** May 26, 2025

Keywords: Necrotizing Soft Tissue Infections, Necrotizing Fasciitis, Sepsis, Clinical Presentation, Diagnosis

History and Terminology

The description of necrotizing soft-tissue infections by Hippocrates in the fifth century BC, and that of Confederate Army surgeon Dr Joseph Jones during the American civil war, who documented a 46 % mortality rate (1871), do not substantially differ from the definitions of today [1-4]. In 1883, French physician J.A. Fournier reported necrotizing infections of the perineum and external genitalia in five male patients - a condition that is now described in both sexes [1,2]. A confusing plethora of terms used in the ensuing years had been referring to similar entities : necrotizing erysipelas, synergistic necrotizing cellulitis, acute non-clostridial crepitant cellulitis, (haemolytic) streptococcal gangrene, and suppurative fasciitis [1,2]. The term "necrotizing fasciitis" (NF) was introduced by Dr Wilson in 1951, who stated that fascial necrosis is the "sine qua non" of certain infectious process [1]. More recently, the term "necrotizing soft-tissue infection" (NSTI) has been adopted to encompass all such necrotizing infectious conditions.

Introduction

NF is a rare but rapidly spreading and life-threatening NSTI, which typically involves the fascia, subcutaneous tissues and skin, and occasionally muscle layers, associated with systemic sepsis [2-10]. The aggressive nature and devastating evolution of the disease is attributed to the synergistic activity of the implicated pathogens, some of which produce gas in the soft tissues ("gas gangrene") [2,7]. Commonly affected sites include the abdomen, perineum-external genitalia, and the extremities [4,7,9].

Clinical onset and outcome of NF has been correlated with underlying comorbidities, the most prevalent being diabetes mellitus (40 %-60 % of cases) [7]. Prognostic factors for the clinical course and outcome of NF remain obscured and are currently under research [10-12]. Reported mortality rates for NF range from 8 % to 76 %, approaching 100 % without treatment, and underscoring the need for timely diagnosis and prompt medical and surgical intervention [4, 7-9, 12-17].

Detecting NF at an early stage remains challenging, due to the non-specific initial symptoms, which are often indistinguishable from cellulitis or abscesses [8-10,12,13]. Studies emphasize that, all NF patients should receive emergency aggressive surgical debridement (necrectomy) within 12 to 15 hours of hospital admission ; delayed initial surgical management, particularly in the fulminant form of NF, is often being fatal [5,15,16]. It has been reported that, about 85 % of the patients are not initially recognized as NF cases, and the condition is frequently omitted in the differential diagnosis [15,16,18]. In light of these challenges, Fais et al reviewed the relative literature on the main ante- mortem and post-mortem diagnostic dilemmas of both clinical and forensic interests [15].

Several critical issues are discussed in this review, which outlines the natural features of NF, highlights the most reliable laboratory indicators, explores current diagnostic scoring systems, and examines the integration of imaging techniques

with other diagnostic means. The review stresses the importance of clinicians' skills and their vigilance in facilitating early diagnosis and initiating timely effective treatments. It does not focus on medical or surgical treatment strategies. Accordingly, a search of the MEDLINE database via PubMed was conducted for recent English- language publications using the search items "necrotizing fasciitis", "necrotizing soft-tissue infection", and "diagnosis". Select unpublished images from our own cases are included, with informed consent obtained from patients involved.

Incidence

The present time is characterized by the dramatic increase in the frequency and severity of NSTIs, alongside increasing resistance to many of the antimicrobial agents traditionally used to treat them.

The overall incidence of NF is low, estimated as less than 0.4 cases per 100 000 population annually, although this rate may vary by region, population and season [1,2,4,7,19,20]. The apparent increase in NF cases is largely attributed to better data recording [1,2]. A predilection for male patients has been reported, with a male-to-female ratio of 3 : 1 or higher, mainly correlated with the increased incidence of Fournier's gangrene in men [7,21]. The disease affects all age groups, although elderly and middle-aged patients are more likely to be infected [7].

On the contrary, acute cellulitis is fairly common, non-necrotizing tissue infection involving localized inflammation that remains above the fascial layer in the soft tissue. It may infrequently complicated by erysipelas, lymphadenitis / lymphangitis, or, rarely, NF. Most cases are managed in outpatient settings [8].

Etiology and Risk Factors

Two distinct mechanisms in the evolution of the infectious invasion of subcutaneous (or areolar) tissue and skin have been described:

1. Direct invasion – through an identifiable portal of bacterial entry, such as external trauma, scrape, or surgical wound. This is the most common route, with pathogens entering the soft tissue initiating a localized infection [1,5-7].
2. Haematogenous spread – less common route, with bacteria spreading through the bloodstream from a perforated viscus, such as the gastrointestinal or genitourinary tracts [1,5,17,22].

Fournier's gangrene, a relatively common form of NSTI, is often triggered by surgical wounds in the external genitalia or groin region, inadequately drained abscesses (e.g., Bartholin's abscess in women), skin pressure sores, anorectal infections, or catheter - related urethral trauma [6,7]. Specific injuries, such as chest wall traumas involving bone cross section (sternum, ribs), or neglected extremity wounds with underlying bone damage (vulnerable to osteomyelitis), can lead to NF, which substantially should increase mortality [7]. Mao et al [23] analysed the craniocervical NF cases with and without thoracic extension and observed worst survival outcomes of patients with thoracic involvement.

Deep infections extend below the dermis and may involve the subcutaneous tissues, fascial planes,and even muscular compartments presenting as complex abscess, necrotizing fasciitis or myonecrosis [6]. The exceptional necrotizing deep neck infections (DNIs) - such as the descending cervicomediastinal or the cervicofascial - are life-threatening and originate from dental infections (primarily) or oropharyngeal /esophageal injury (occasionally). These polymicrobial infections involve both aerobic and anaerobic species [24-29]. Approximately 2.6 % of all dental infections may progress to NF [27]. However, many NF -focused studies do not include these rare but lethal cases.

The second, less frequent route of bacterial invasion leading to NF involves complicated intraabdominal infections, such as the sealed perforated diverticulitis or appendicitis, gastroduodenal or small / large bowel perforations, and necrotic cholecystitis [17,22].

With either way of invasion, bacteria spread along subcutaneous / areolal tissue planes, producing toxins that cause tissue ischaemia, liquefactive necrosis, and systemic illness [1].NF disrupts tissue blood flow by inducing occlusive intravascular cell aggregation, which is associated with toxic production [20].

Several comorbidities are strongly linked to the clinical onset and outcome of NF. The most frequent is diabetes mellitus, present in 30 %-60 % of patients of any NF type, which predisposes patients to sepsis [2-4,7,9,25,28]. Other commonly associated comorbidities encompass conditions causing immunosupression or immune dysfunction, such as chronic kidney disease, congestive heart failure, liver cirrhosis /alcohol abuse, corticosteroid therapy, Addison disease, systemic lupus erythematosus, chronic hypertension, peripheral vascular disease, and advanced age [2-4,7,9]. Progression to severe sepsis in these patients is more likely [6-9,11,14,30].

There is no consensus for specific predictors of mortality between numeral studies dealing with this issue. Over the decades, reported risk factors for NF-related mortality include advanced age (> 60 years), extensive infection, high serum creatinine or blood lactate levels, presence of multiple organ dysfunction syndrome (MODS), delays in first surgical debridement, and female gender [7,10-12,14,30-33]. Most studies highlight advanced age as a strong independent predictor of mortality [7]. Nonetheless, the factors leading to the fulminant form of NF with fatal outcomes within 24 hours still remain unidentified.

Pathophysiology

NF typically begins after bacterial invasion, followed by secondary infection from the host's indigenous aerobic or anaerobic microflora, usually resulting in a polymicrobial infection [11,34]. Necrotic infection originates in the superficial fascia or the hypodermis, sparing the more superficial layers (epidermis and dermis) in the early stages [1,5,7,33]. The pathogenesis of NF involves a synergistic interaction between virulent factors of bacteria and specific responses of the host [2,7].

Pathogenic organisms proliferate in the soft tissues and release destructive endotoxins and exotoxins into the systemic circulation, triggering acute inflammatory reactions (systemic inflammatory response syndrome, SIRS). This leads to infiltration of leucocytes and cytokine production by the leucocytes [1,2,5,8]. The ensuing cytokine cascade leads to endothelial damage, increasing permeability of the micrvasculature and results in prolonged vasoconstriction in the dermal capillary network (capillary leak syndrome), along with microthromboses of the feeding vessels of the fascia [1,2,5]. The coagulation cascade of thromboplastin is activated, resulting in disseminated microthrombosis of the fascial vessels. Tumour Necrosis Factor -alpha (TNF-a) further exacerbates injury to the vascular endothelium by stimulating neutrophil degranulation [1]. Additionally, the complement and bradykinin-kallikrein systems are activated [1].

As the process progresses, hypoxia and ischaemic oxidative destruction of the subsutaneous tisuues and the deep fascias result in extensive necrosis and liquefaction of the fascias and the surrounding tissues [5]. Increased vascular permeability allows high-protein fluid exudates to accumulate along the deep fascias [1,5].

The central histopathological feature in the evolution of these necrotizing infections is thrombosis of vessels perforating the skin and subcutis [5]. NF, as a soft tissue infection "per se", typically does not cause myonecrosis [2,5].

The underlying fascial and hypodermic necrotic spread presents in discrepancy with the lesser extent of the overlying skin changes, which are happening later [2,5,7]. Besides, severe pain "out of proportion to the physical findings " or, conversely, reduced or even absent subjective sensation of pain, are explained by the degree of tissue destruction, namely, the vascular occlusion (commonly) or the nerve damage (later) [2,5,7]. SIRS phenomena can rapidly progress to septic shock, MODS, multiple organ failure (MOF), and ultimately death [2,5].

Among the DNIs, those of odontogenic (submandibular) or oropharyngeal (e.g., peri-tonsillar) neglected infectious foci can insidiously but rapidly progress to destructive suppurative necrotic processes and occupying swellings [28]. These infections spread along the fascial planes of the neck, and, due to absence of barriers,may involve the lateropharyngeal, retropharyngeal, pretracheal, prevertebral and intracranial spaces or the mediastinum. Negative intrathoracic pressure facilitates downward spread to the mediastinum [24-29]. Septicaemia and the serious compressing effects on vital structures (e.g., airway obstruction, jugular vein thrombosis, disseminated intravascular coagulation) explain the deadly nature of the disease [25,28,29].

Epidemiology and Classification

NSTIs and NF are typically classified into four types:

Type I: Polymicrobial infections involving synergistic mixture of aerobic (Gram-positive cocci, Gram-negative rods) and anaerobic bacteria such as *Streptococci*, *Pseudomonas* spp, *E.Coli*, *Bacteroides* spp, and *Clostridia* spp (commonly, 55 %-80 % of cases).

Type II: Usually monomicrobial, caused by Gram-positive bacteria, such as group A b-haemolytic *Streptococcus* (GAS), either alone or in combination with *Staphylococcus aureus*. Can be associated with toxic shock syndrome (< 20 % of cases).

Type III: Monomicrobial infections from Gram negative bacteria,such as *Vibrio* spp (typically, marine-related in Asia), which have fulminant course and can cause MOF).

Type IV: Rare fungal infections, most commonly affecting the immunocompromised individuals (e.g., *Candida* spp, *Aspergillus*, Zygomycetes) [1-5, 9-11,24,26,29,30,34].

The anatomic sites of infection can be classified as abdominal/ groin, perineum /sacral, extremities (upper and lower limbs), chest /breast, and neck/ fascial areas [14,28]. NF can involve any body part [4,5,9,19,21,30]. For example, in a 2016 review of 58 NF patients, the body parts affected were the central part in 48.3 % of cases, extremities in 36.2 %, and combination of central part with one of extremities in 13.8 % [4]. Perineum was most frequently affected in the 198-patients old cohort of Elliott et al, with Fournier's gangrene comprising 36 % of cases. Similarly, Vayvada et al supported these findings. Anaya et al reported that, the lower extremities were mostly affected (57.8 %), followed by the abdomen and the perineum. A recent review [9] indicates that, the extremities,particularly the lower limps, are affected in 36 % - 55 % of cases [9,21,30,31].

In 2014, the Infectious Diseases Society of America (IDSA) updated practice guidelines for the diagnosis and management of the skin and soft-tissue infections (SSTI), classifying them as purulent vs. non purulent, necrotizing vs. non necrotizing, and by severity (mild, moderate, severe) [35]. In 2018, the World Society of Emergency Surgery (WSES) and Surgical Infection Society- Europe (SIS-E) issued their own recommendations for management, dividing SSTIs into surgical site infections (SSIs), non-necrotizing SSTIs, and necrotizing SSTIs [6].

Diagnosis

Diagnosis of necrotizing infections/ NFs relies on evaluation of clinical symptoms and signs, laboratory measures and imaging studies [5,7,9,14,16]. Their multifaceted nature has led to strict collaboration among general and emergency surgeons, thoracic surgeons, intensivists, anaesthesiologists, and infectious disease specialists [6]. Patients should be promptly admitted into Emergency Departments or Intensive Care Units prior to any evidence of haemodynamic instability [5,7,19,28,36].

1. Clinical Signs and Symptoms

In the early stage, the spread and extent of necrotizing infection within the subcutaneous tissues may not correlate with the lesser, or even absent, overlying skin changes. This discrepancy often leads to uncertainty in diagnosis or underestimation of severity of the disease in progress, delaying accurate recognition and appropriate treatment [1,6]. Furthermore, preadmission treatment with non- steroidal anti-inflammatory drugs (NSAIDs) or antibiotics can mask the true nature of the underlying disease. Repeated clinical assessment along with multiparametric (clinical and laboratory) and multidisciplinary approach, is essential for effective management [2].

In daily practice, in the absence of distinctive early clinical features, differentiation of NSTIs from the more common non-NSTIs (e.g., severe cellulitis, abscess, or lymphedema) remains challenging and critical. NSTIs require urgent and aggressive surgical intervention, unlike non-NSTIs typically resolved with anti-microbial therapy [1,6-8,18,19,35].

According to the 2018 WSES/ SIS-E conference guidelines, any rapidly progressive soft -tissue infection, especially in a deteriorating patient, should raise suspicion for necrotizing infection (recommendation 1C). Clinical suspicion still remains superior to laboratory testing or the LRINEC score (see below) in these cases [1,3,4,6].

Common manifestations associated with the early stages of NF, when the disease may not be apparent, often include minimal erythema or skin discoloration, along with tender local swelling or edema extending beyond the visible erythematosous area. This may be accompanied by local warmth induration, numbness, or local pain disproportionate to the physical skin findings, and occasionally fever [1,3,6,7,14,29,35]. However, pain and fever may be absent in some cases [7,16].

As the infection progresses, advanced symptoms and "hard signs" may appear, including local intense pain or anaesthesia, blistering, purulent discharge, foul odor, crepitus, skin sloughing or necrosis (at later stage), and compartment syndrome in limbs [1,6,7,35]. The presence of crepitus suggests infection from gas- forming anaerobic bacteria (e.g., *C. perfringens*), and usually appears after five days or more, affecting only 13 % to 31 % of patients [1,6,7].

In advanced stages, patients may present with symptoms and signs of systemic toxicity, including fever, tachycardia (> 100 beats / min), tachypnea (> 20 breaths/ min), hypotension (SAP < 100 mm Hg), dehydration, confusion, nausea, general malaise or weakness [5,7]. Patients who meet the SIRS criteria can develop sepsis,

and patients with hypotension resistive to fluid resuscitation may develop septic shock and MOF (late phase) [7]. If the patient is undiagnosed or untreated, the clinical course deteriorates rapidly, sometimes during a few hours [6]. In fulminant cases, MOF may develop within the first 24 hours of infection [5].

Fournier's gangrene in males often begins with severe pain and itching in the scrotal skin and perineum. In the common genitourologic type, the infection may spread through fascial planes and affect fascias such as Buck's, Dartos, Colles' and Scarpa's. Necrosis of the superficial fascia and fat produces a thin watery malodorous fluid and crepitus ; this appearance resembles the late-stage NF [5].

The necrotizing cervicomediastinal DNIs,such as descending suppurative cervicomediastinitis and descending necrotizing mediastinitis, can rapidly compromise the airway and other neighboring vital structures (as enlarging/ compressing swellings), causing pain, fever, dysphagia, dysphonia/ hoarseness, dyspnea, acute respiratory distress syndrome (ARDS), airway obstruction (suffocation), and potentially death [24-29]. Necrotizing cervical fasciitis with necrotizing descending mediastinitis has a mortality rate as high as 50 %, despite intensive treatments [24-28]. The complex multidisciplinary approach (into Intensive Care Units), namely intensive medical support with establishment of the airway security and aggressive surgery, is the only key to save the lives of these patients [24,25,28,29]. Unfortunately, the recent literature does not provide much more optimistic results, and medicolegal implications for the caring physicians still arise from daily practice and decisions [28].

Summarizing, in cases in which the patient's overlying inflammation findings may resemble cellulitis, which actually should respond to antimicrobial treatment alone, features that indicate deeper tissue involvement suggestive of obscuring necrotizing infection /NF, which requires prompt surgical intervention, include :

1. Pain disproportionate to the clinical findings
2. Hard (''wooden'') feel of subcutaneous tissue beyond the area of skin involvement, and edema / tenderness beyond the cutaneous erythema
3. Skin necrosis or ecchymoses, possibly accompanied with crepitus
4. Systemic toxicity signs
5. Lack of improvement with antibiotic treatment.

2. Tests and Scoring Systems

The use of diagnostic and prognostic tools in the daily practice can support clinicians in proper, on-time management. Common and highly suggestive of NF laboratory results include: leucocytosis (WBC > 20000/ L, with left shift-not always present in the immunocompromised patients), elevated C-reactive protein (CRP), increased creatinine (cr) and blood urea nitrogen (BUN) (indicative of uremia/ dehydration), marked hypoglycemia (particularly in diabetics), hypocalcemia (due to calcium depositions in necrotic subcutaneous fat), and hyponatremia (serum sodium <135 mmol / L) [1,6-9,14,19,30,31,33,37].

Risk stratification can be enhanced using laboratory index measures based on serum parameters, aspects of clinical presentation in conjuction with variables such as bacteremia, presence of *clostridia /aeromonas /vibrio* infection, radiographic evidence of tissue gas, degree of MODS or presence of shock at admission, extent of necrosis, and the associated comorbidities (e.g., liver cirrhosis, cancer, advanced age) [7,9,30,31,33]. For critically ill patients, the Sequential Organ Failure Assessment (SOFA) and Acute Physiology and Chronic Health Evaluation (APACHE) II scores help assess the severity of sepsis and guide treatment choices [5,14,36].

The laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) scoring system, developed by Wong et al, remains the most widely used lab-based diagnosting tool for the disease and to rule out cellulitis [3,6-9,13,20,38,39]. This system incorporates six routine serum parameters (WBC, CRP, cr, hemoglobin, sodium, glucose) and it is used in order to enable early recognition of the condition, discriminate between non-necrotizing and necrotizing disease, and choose treatment [6,13,14]. A score of > 6 suggests necrotizing infection, with further stratification of possibility of the infection into low (score < 6), moderate (< 8), or severe (≥9) [7,13]. However, recent studies question the LRINEC score's sensitivity, cautioning against sole reliance on it for diagnosis [6,40]. Newer models have been introduced to improve diagnostic accuracy, such as the modified LRINEC (MLRINEC), which includes additional variables like serum lactate levels and liver disease and redefines the cut-off values for WBC, CRP and hemoglobin, and the J-LRINEC score for japanese patients [8,9,37,40].

Other scoring systems and indices which are in use in order to facilitate diagnosis of NF and, consequently, to determine the need for surgical intervention include:

- Laboratory and Anamnestic Risk Indicator for NF (LARINF), which combines three serum markers with three comorbidities [9,10].
- Site other than lower limb, Immunosuppression, Age < 60 y, Renal Impairment, Inflammatory Indicators (SIARI) score, which uses four comorbidities and three serum markers [9,38,40].
- Fournier Gangrene Severity Index (FGSI), which uses three vital signs and six serum markers [9].

All above, and additional systems such as imaging-based tools, the Physiology and Operative Severity Score for the enUmeration of Mortality and morbidity (POSSUM), and the ANF amputation risk scoring system, are under ongoing validation [10,39,40].
Near-infrared spectroscopy (NIRS) is a non-invasive method to measure tissue oxygenation (nSO2) for distinguishing NF from cellulitis, using comparisons with the values of affected and healthy contralateral tissues [20]. Its utility is limited in many cases, including those with lesions at the midline of the trunk or those with bilateral involvement. Diagnostic accuracy may also be reduced in rare patients with underlying vascular condition causing blood flow disorder [20].

Bedside interventional tests - as well as imaging- are carried out in patients with equivocal clinical findings to aid surgical decision and scheduling [3,6,7]. Firstly, the "finger test" at the suspected area involves a 2-cm incision to the deep fascia. The ease of subcutaneous tissue dissection with the index finger, and the findings of grey necrotic tissue, fascial edema, thrombosed vessels, '' dishwater '' pus, and non-contracting muscles are consistent with NF [3,6,7]. Secondly, the "triple diagnostics" include an incisional biopsy over the suspected area, a fresh frozen section and Gram staining. They may help in early stages and be an important adjunct (recommendation 1C) [6]. These tests have been argued to be subject to sampling error, and they have been implicated in treatment delays [6,8].

Recent advancements in Artificial Intelligence (AI) contributed to its application within medical science, particularly using sophisticated Machine Learning (ML) Algorithms. The use of

ML Algorithms, such as the ''Random Forests'' (RF), k-nearest neighbors (KNN), Support Vector Machines (SVM), ''Light Gradient Boosting Machine'' (Light GBM), and the ''Logistic Regression'', has shown promise in facilitating scheduling of accurate, unbiased and efficient models for the early disease detection [37,41,42]. In a 2024 study, Chang et al postulated that, AI models (particularly the RF) analyzing clinical and laboratory data, including ultrasound-assisted aspiration fluid parameters, could significantly improve diagnostic speed and accuracy, being cost-effective as well [19]. However, these novel diagnostic adjuncts require further validation in larger clinical trials before widespread adoption.

3. Imaging

Unless the disease is advanced, imaging at the site of the infection in the earlier stages, subject to certain conditions, can aid in confirming the diagnosis. However, all imaging modalities evaluated so far have notable limitations : either low sensitivity in detecting necrotizing infections early, or low specificity to accurate diagnosis [1]. Importandly, imaging should never delay surgical consultation and intervention (Recommendation 1A) [6].

Plain x-rays may appear normal in the early disease but can sometimes show increased soft tissue thickness and opacity, as well as, subcutaneous gas ; typically, gas in deeper fascial layers is not detected [1,6].

Ultrasonography (US) is a fast, bedside modality, especially useful in gas gangrene cases [6]. The presence of diffuse subcutaneous thickening accompanied with fluid accumulation along the deep fascia is considered suggestive of NF [6,7,16,25].

Computed tomography (CT) scan, which is widely accessible, offers greater sensitivity. It can detect inflammatory changes, such as fascial edema and thickening or abscess or tissue gas, as well as the extent of infection [1,6,7,16,27-29,43]. US- or CT- guided aspiration of infectious fluid prior to administering empiric antibiotics can support diagnosis through Gram staining and culture, alongside blood cultures [8,25]. Arteriography can be necessary in certain DNI cases [29].

Magnetic Resonance Imaging (MRI) provides greater accuracy and more detailed information than CT scan (e.g., in DNIs), but is infrequently used [6,15,27]. In the case of vascular complications (thrombosis of the internal jugular vein, rupture of the carotid artery, or aneurysm), MRI angiography is recommended [28,29]. MRI poses challenges in critically-ill or unstable patients, due to its time-consuming nature resulting in delays in treatment, and its higher cost [1,6,7].

Future Research

Current research on diagnosing NF faces several limitations, including that, most available data is gathered from single center cohorts, the number of participants is limited, the critically-ill condition of many patients often precludes their collaboration, and heterogeneity of comparison material and methods introduces bias. To date, there remains a shortage of well-diagnosed trials directly comparing diagnostic modalities. Future research should focus on large-scale, prospective, multi-center studies. Such trials could prove advantageous in earlier recognition, improve diagnostic evaluation, and ultimately lead to improved patient outcomes.

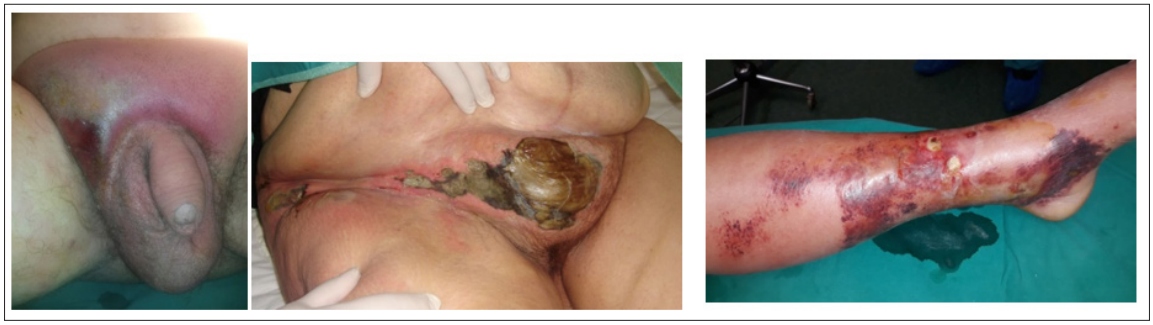

Figure 1a **Figure 1b** **Figure 1c**

Figure 1: NF of External Genitalia (Fournier's Gangrene) and Lower Abdominal Wall in a) 60 -Year-Old Man and b) 70 -Year-Old Woman, and NF of Lower Extremity in c) 45-Year-Old Man

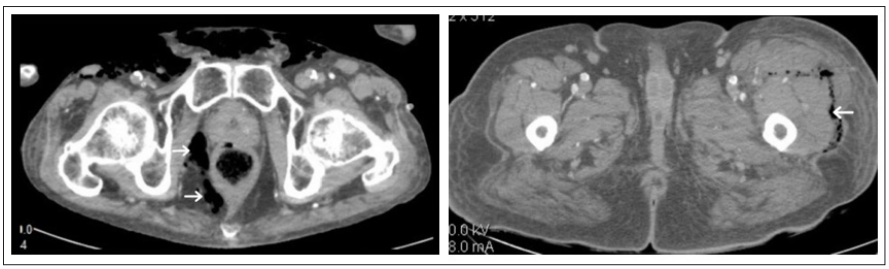

Figure: 2a **Figure: 2b**

Figure 2: Emergency Pelvic-Thigh CT: a) Neglected NF of External Genitalia-Perineum in a 90-Year-Old Patient on Sepsis/ MODS; Arrows Show Emphysema in Rectosigmoid; Patient underwent index Aggressive Surgery and Two Additional Limited Debridements, but Died on Day 7.b) Thigh NF with Compartment Syndrome -Arrow Shows Pathognomonic Emphysema; Patient needed only a Wide Necrectomy, Recovered, Hospitalized for 28 Days.

Conclusions

Necrotizing infections, with necrotizing fasciitis the worst of all, are distinguished from the milder superficial soft tissue infections by their clinical features, the systemic toxic manifestations potentially resulting in lethal outcome, and the need for aggressive treatment including intense medical support and prompt surgical intervention. Clinical awareness of NF remains pivotal, as earlier diagnosis and prompt initiation of treatment are key to interrupting the lethal course of the disease. In this context, challenging clinical judgment remains the cornerstone of effective diagnostic decision-making.

Consent

Written informed consent was obtained from patients included in the study (images) for publication in this review.

Competing Interest

Authors declare that no competing interests exist.

Ethical approval

It is not applicable.

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