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S.U.M.S.

**Shiraz E-Medical Journal**

**Vol. 9, No.2, April 2008**

**http://semj.sums.ac.ir/vol8/apr2007/86058.htm**

# Necrotizing Fasciitis - the Flesh Eating Bacterial Disease - of the Neck.

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Received for Publication: January 12, 2008, Accepted for Publication: February 15, 2008.

**Abstract:**

Necrotizing fasciitis is uncommon and can be difficult to diagnose. It causes progressive morbidity until the infectious process is diagnosed and properly treated. A delay in diagnosis is associated with a grave prognosis and increased mortality. The main goal of the clinician must be to establish the diagnosis and initially treat the patient within the standard of care. We would like to present a case of 70-year-old gentleman with poorly controlled diabetes, who was diagnosed with type 1 primary necrotizing fasciitis of the neck and present the imaging findings which led to the diagnosis.

Key Words: Necrotizing fasciitis, necrotizing cellulitis, Flesh eating disease, fasciitis necrotans, synergistic necrotizing cellulitis.

**Introduction:**

Necrotizing fasciitis is a rare and life threatening condition with extensive fasciitis and gas in the superficial fascia [1]. It was recorded in France in 1783 and first described in 1848. In 1918, the cause of the disease was identified as bacterial and in 1952, the disease was named "necrotizing fasciitis" by Wilson [3]. It is also known as Flesh-eating bacterial disease, killer bug disease, synergistic necrotizing cellulitis and fasciitis necrotans [3]. It causes extensive undermining of surrounding tissues and subcutaneous layers and necrosis is typically more advanced than the appearance suggests. By far, it is the fastest-spreading disease known to man, with progression rate of up to 3 cm per hour. Common locations are lower extremity, arm, neck, back and scrotum (Fournier gangrene) [2]. Since 1883, about 500 cases have been reported in the literature. The diagnosis is confirmed by either blood culture or aspiration of pus from tissue, but early medical treatment is crucial and often presumptive. Radiological investigations, mainly CT scan can help with the diagnosis and show the extent of the disease which is useful in directing surgical debridement. Antibiotics should be started as soon as this condition is suspected. Initial treatment often includes a combination of intravenous antibiotics including penicillin, vancomycin and clidamycin. If necrotizing fasciitis is suspected, surgical exploration is always necessary, often resulting in aggressive debridement.

**Case Presentation:**

A 70 years old man was brought to the emergency department with a painful left-sided neck swelling over 2 weeks duration. The swelling has been rapidly increasing in size and associated with fever and pain on chewing. There was no history of trauma, hypodermic injection or bite. There was no dysphagia or breathlessness. He is a known case of diabetes mellitus, hpertension, hypercholesterolaemia and ischaemic heart disease. On examination, patient is febrile, temperature = 38°C, BP = 130/90 mmHg and HR = 88/min. Physical examination revealed left, level 2, infra-auricular mass, pushing ear lobe outwards, measuring 10 x 12 cm in size, it is tender, hard and fixed. Overlying skin is erythematous, hot and friable. Skin induration extends to mastoid and occipital regions. Cranial nerves VII are intact bilaterally. Intra-oral examination revealed erythematous tonsils. There is no medialization of the left pharyngeal wall and no fungating mass. Otoscope examination shows bulging left tympanic membrane which is covered by slough. External auditory canal is coated by pus.

Blood count result shows raised white blood cells (30.0 x 103 / ml), hemoglobin is normal at 14.3 mg/dl, platelet is 809 x 103 /ml and Neutrophil count is signifi-

cantly high at 26.5 x 103 /ml consisting 87.7% of total WBC count. Chest radiograph (Figure 1) showed presence of gas in the soft tissue of the left side of the neck. Contrast enhanced CT Scan of the neck (Figures 2a, 2b ,3a, 3b) revealed gas within the soft tissue plains of the left side of the neck associated with deep seated abscess formation and necrosis.

Ear pus culture and sensitivity showed Klebsiella sp. and Enterobacter sp. growth, sensitive to gentamicin, cefuroxime (zinacef), cefotaxime (claforan), ceftriaxone (rocephine), ciprofloxacin and amoxicillin clavulanate (augmentin).

Diagnosis of type 1 primary necrotizing fasciitis of the neck was made and intravenous antibiotics were started for the patient. Metronidazone 500 mg 8-hourly and ceftriaxone 1 g 12-hourly. Patient also underwent surgery. Intra-operative findings revealed extensive necrosis and multiple abscess formation involving the soft tissue and muscular tissue of the left side of the neck. Left cortical mastoidectomy, neck fasciotomy and debridement were done. Patient had a slow but steady recovery after the operation and was discharged after three weeks.

**Discussion:**

Necrotizing fascitis is caused by multiple bacteria however the bacteria do not actually eat the tissue. So "Flesh-eating bacteria" is a misnomer.

The bacteria produce pyogenic exotoxins and other virulence factors capable of activating T cells nonspecifically which cause over-production of cytokines that over stimulate macrophages. The macrophages cause the actual tissue damage by releasing oxygen free radicals that are normally intended to destroy bacteria but are capable of damaging nearly any macromolecule they contact in the body[3].

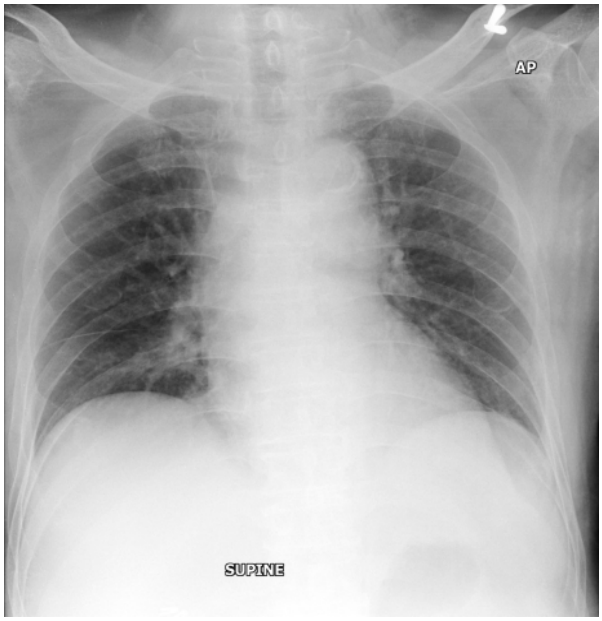
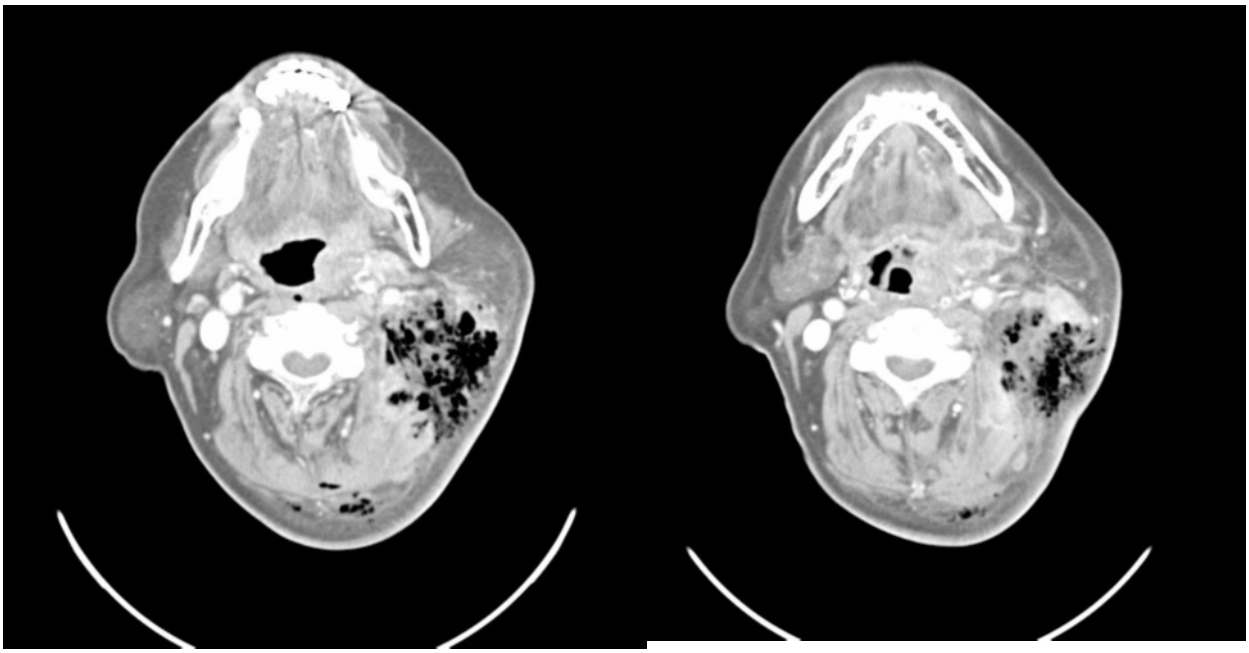
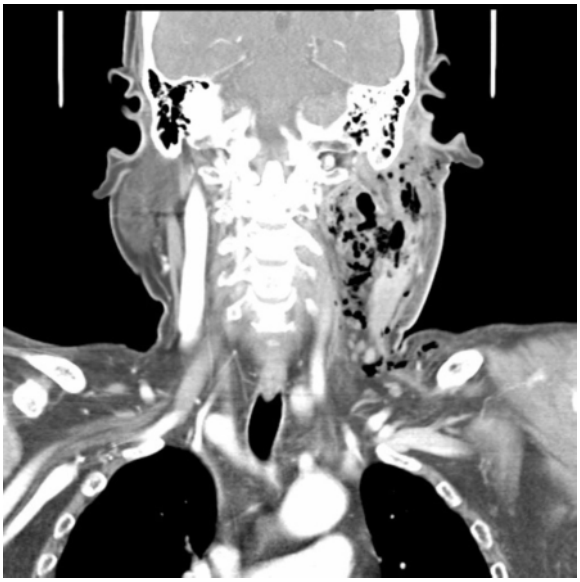
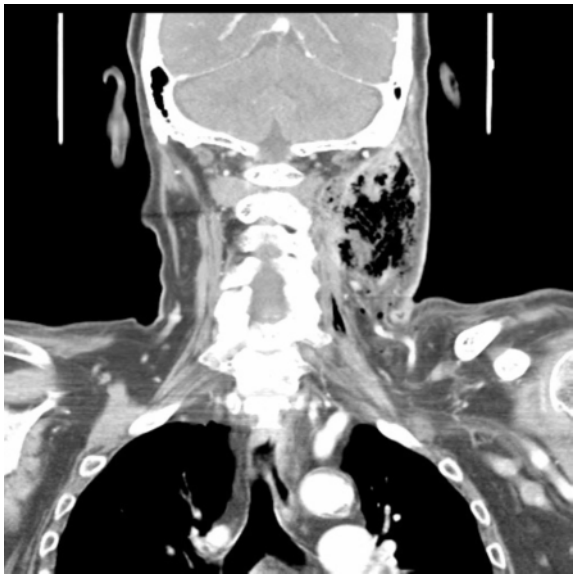


Figure 1. Chest radiograph demonstrates presence of gas in the soft tissue of the left side of the neck.



Figures 2a, 2b. Contrast enhanced CT scan of the neck clearly demonstrates presence of gas within the soft tissue plains of the left side of the neck associated with deep seated abscess formation and necrosis.





Figures 3a, 3b. Coronal reconstruction of the CT images clearly demonstrates the extent of the disease.

Bacterial factors including surface proteins M-1 and M-3 increase the adherence of bacteria to the tissues, and further protect the bacteria against phagocytosis by neutrophils.

Deep infection causes vascular occlusion, ischemia, and tissue necrosis. Nerve damage produces characteristic localized anesthesia. Septicemia and systemic toxicity ensues.

The disease commonly occurs in patients in 4th to 7th decade and is more common in men. M:F ratio is 2 to 3:1. If untreated, it is invariably fatal[3] with mortality rate of 25 – 83%[4]. Predisposing factors are diabetes mellitus, malignancy, alcohol or drug abuse and poor nutrition[2]. There are 3 types of necrotizing fascitis based on causing organism:

Type I: polymicrobial infection, Type II: Group A streptococcal infection and Type III: Gas gangrene or clostridial myonecrosis [3].

Infective organisms are Aerobes: Group A hemolytic streptococci, Staphylococcus aureus (including MRSA) and Anaerobes: Bacteroides fragilis, Clostridium, Peptostreptococcus and Escherichia coli. Early symptoms are minor skin trauma, pain in the general area of injury which is usually disproportionate to the injury and becomes more and more painful associated with flu-like symptoms, diarrhea, nausea, fever, confusion, dizziness, weakness and general malaise and dehydration. Advanced symptoms include swelling with purplish rash, dark marks that become blisters filled with blackish fluid. Wound appears necrotic with a bluish, white or dark, mottled, flaky appearance. Critical symptoms are severe hypotension, septic shock and loss of consciousness. Complications are renal failure, metastatic cutaneous plaques, severe disfigurement and death.

Clinically necrotizing fasciitis is divided into two subgroups. Primary necrotizing fasciitis: there is absence of known cause or portal of entry for bacteria [6,7]. The underlying pathogenesis is still unknown [3] and diagnosis may be challenging.

Secondary necrotizing fasciitis: it is seen in most cases. Known aetiologies are laceration, abrasion, contusion, burn, bite, subcutaneous injection, operative incision, perforated viscus, peri-rectal abscess or infected Bartholin cyst [5]. In necrotizing fasciitis, plain radiography, ultrasound and MRI can show subcutaneous gas but CT scan is the best technique. CT scan can demonstrate subcutaneous air, and can depict the deep extents of the disease, which is useful in directing surgical debridement.

Necrotizing fasciitis of the head and neck is not a common entity. A recent review of the literature revealed only 64 reported clinical cases of necrotizing fasciitis of the head and neck area, with a cumulative

mortality rate of 25%. Among the most common causes are odontogenic, tonsillar, and pharyngeal infections; less common causes included surgery and radiation therapy [8]. It is also important to remember that any anterior neck mass has the potential to produce airway compromise.

On CT scans, cellulitis is characterized by thickening of the cutis and subcutis and increased attenuation of fatty tissue with streaky, irregular enhancement and without fluid collections. Fasciitis appears as thickening or enhancement of fasciae. While the superficial cervical fascia can routinely be visualized on CT scan, the three layers of deep cervical fascia, as well as the fasciae that surround individual muscle groups, are usually too thin to be visualized on CT scan images and show no marked enhancement after intravenous administration of contrast material. Myositis appears as thickening of cervical muscles or muscle groups with or without inhomogeneous enhancement, and myonecrosis usually becomes visible as a hypoattenuating area within enhancing portions of a muscle or as frank muscle disruption. Any marked enhancement of portions of muscles must be considered a pathologic finding at CT because the enhancement of normal muscular tissue is not seen on CT scan after administration of contrast material. An abscess appears as a single or multiloculated area of fluid attenuation with or without gas collections, usually expands the compartment that is involved, and typically demonstrates a peripheral rim enhancement [9].

Overall, features on CT scan include asymmetric fascial thickening with fat stranding (80%), gas in soft tissue dissecting fascial planes from gas-forming organisms (55%), associated deep abscesses (35%) with or without secondary muscle involvement [2].

Plain X-ray can detect presence of gas within the soft tissue as streaks of lucencies along the soft tissue planes. B-mode ultrasonography can demonstrate liquefied fractions of tissue and lymphadenopathy. Presence of air can also be detected as it causes sonographic reflections. Color Doppler ultrasonography is not widely used although it can show areas of reduced vascularity where dead tissue is present.

T2 weighted Spin Echo MRI shows affected tissue as well-defined regions of high signal intensity in the deep tissue. MRI sensitivity exceeds its specificity [1]. Absence of gadolinium contrast enhancement in T1 weighted images reliably detects fascial necrosis in those requiring operative debridement [1].

Other diagnostic tests include skin biopsy, culture and sensitivity and Gram staining [3].

The treatment for necrotizing fasciitis is a combination of surgical debridement, appropriate antibiotics and optimal oxygenation of the infected tissues. Necrotizing fasciitis a challenging and potentially debilitating or lethal disease. Early diagnosis is of principal importance and aggressive multidisciplinary treatment is mandatory. Early recognition and treatment by extensive debridement and antibiotics can prevent its

fulminant course with a grave outcome [6, 7, 10]. The priority in every case is to proceed to radical surgical debridement.

Once necrotizing fasciitis is diagnosed, all patients must be treated with immediate surgical debridement, and broad spectrum antibiotic combinations against the anaerobs, gram-negative and gram-positive bacilli. This combination can be changed to other antibiotic combinations as determined according to the culture sensitivity of the microbial isolates and clinical course of the patients. The microbiological isolates are polibacterial in the majority of the patients with either primary or secondary necrotizing fasciitis in concordance with some recent studies [3]. Therefore, administration of broad-spectrum antibiotics appears to be important in the management of these patients. Penicillin-clindamycin-gentamicin or ampicillin-sulbactam or nafcillin or cefazolin plus metronidazol combinations could be considered in the initial antibiotherapy of necrotizing fasciitis according to the physician's preference [11]. Hyperbaric oxygen therapy (HBO) treatment increases tissue oxygenation in both healthy tissue and in the vicinity of infected tissue. Hyperbaric oxygen-therapy is administered at 2.5 to 3.0 atmospheres for 90 min twice daily, following surgical debridement until no ongoing necrosis is evident in patients with clostridial infections [12]. Critical care support should be provided for patients with hemodynamic and ventilatory instability.

Elderly patients with underlying diabetes mellitus that have suspicious clinical findings of necrotizing fasciitis without any causative factors (trauma or operation) should be carefully examined for the presence of primary necrotizing fasciitis. Imaging studies, especially CT scan can be very helpful in early detection of the disease in such patients.

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