The role of Ultrasound managing Necrotizing Fasciitis, helps to improve patients outcome.

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Learning objectives

The purpose of our educational exhibit is to:

1. Describe ultrasound (US) features of necrotizing soft tissue infection (NSTI).
2. Describe the main differential diagnosis of NSTI.

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Background

In a strict sense, NECROTIZING FASCIITIS is a rare, rapidly spreading deep-seated infection located in the fascia. Necrotizing soft tissue infections have been recognized and reported for centuries, the earliest dating back to Hipocrates. The original description of "hospital gangrene" was made by Joseph Jones in 1871 following his experience during the U.S. Civil War. Later the French veneorologist Fournier describe a type of necrotizing fasciitis affecting the perineum which he called "Fournier gangrene" caused by group A beta hemolytic streptococci, also known as "flesh eating bacterium". The term "Necrotizing fasciitis" was popularized by Wilson in 1952. Additional terms used in the past include "gas gangrene" (Clostridial myonecrosis), when the disease affected the muscle tissue.

Now, the term "NECROTIZING SOFT TISSUE INFECTIONS" is use to encompass all of these necrotizing infections, defined as infection of any layer within the soft tissue compartment (dermis, subcutaneous tissue, superficial fascia, deep fascia or muscle) that are associated with necrotizing changes. The term NSTI advocate an approach to all of them that uses the same principles for diagnostic and treatment strategies.

ANATOMY

Care should be taken to avoid ambiguous term. The term "Fascia" is vague. A fascia is a structure of connective tissue that is large enough to be visible to the naked eye. In the fascia superficialis, fat cells accumulate within the meshes of the fibroareolar tissue, forming a fat layer of variable thickness. The dermis is continuous with the fascia superficialis, which contains nerves and blood vessels. The term "Fascia superficialis" is uses to designate either the entire hypodermis or a layer of connective and vascular tissue located immediately deep to the dermis or deep to the fat layer. Anatomist define the underlying "Deep fascia" as a structure composed of more tightly packed collagen fibers and often indistinguishable from the aponeurosis, which is the superficial insertion site of the muscle fibers. The deep fascia is continuous with similar structures that separate the muscle groups into compartments, known as the "intermuscular septa".

Many recent articles, specially in orthopedic literature, use the term "Superficial fascia" to designate the deep fascia as defined by the anatomist and the term "Deep fascia" for the intermuscular septa. (Fig 1). It is very important to note this to avoid confusion. We prefer to use the terminology of orthopedic literature to avoid confusion.

EPIDEMIOLOGY AND RISK FACTOR

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NSTI is a rare disease. The incidence has increased since 1980. The exact reason remains speculative. Possible explanation includes increase microbial virulence and resistance due to the excessive use of antibiotics, population aging, increased of immunosuppression and better disease reporting.

The majority of patients with NSTI have pre-existing conditions that render them susceptible to infections, such as obesity, diabetes mellitus or alcohol abuse. NSIT however can also occur in otherwise healthy adults. In almost all cases, there is a precipitating event such as a history of surgery or a penetrating injury that can be as trivial as an insect bite or a scratch.

**MICROBIOLOGY:**

Four basic causes of microbial subtypes are described.

**Type I** is the most common one with an average of four pathogens, usually a mixture of aerobic and anaerobic organism.

**Type II** is a monomicrobial infection that tends to occur in healthy young. Group A (Streptococcus pyogenes) is the typical pathogen.

The infections caused by Vibrio species are classified as **type III** with a fulminant systemic course. It can be acquired through a skin lesion an exposure to warm sea water.

Finally, some authors have described **type IV** or fungal infection. (Table 1).

**DIAGNOSIS:**

Establishing the diagnosis of NSTI is challenging. The diagnosis is based on clinical findings, laboratory findings, pathology and surgical exploration and diagnosis imaging.

**CLINICAL FINDINGS:**

- Involved areas in NSTI tend to be **extremely painful in early stages** and painless in more advanced stages.

- Kim and colleagues divided NSTI into three stages. In stage I (early stage) the overlying skin is warm, **erythematous** and indurated, producing "wooden skin". In stage II (intermediate stage) **blisters and bulla** form, and in stage III (late stage) the bulla become hemorrhagic, crepitus can be noted and skin necrosis (Fig 2).
The progression of the clinical findings is usually relatively fast. One characteristic of the disease is the quick progression of the erythema margins (>1 cm hours) despite antibiotic treatment. For this reason, it is useful to paint the margins of the erythema.

However, in select cases, NSTI can progress in a more insidious manner which makes the diagnosis even more difficult to establish.

LABORATORY FINDINGS.

Systemic findings in NSTI include fever, tachycardia, hypotension, and shock.

Even though laboratory findings are not specific, a combination of certain laboratory test may enable to discrimination between NSTI and non-necrotizing infections. In 2004 Wong et al proposed a scoring system (laboratory risk indicator for necrotizing fasciitis: LRINEC) based on six different variables that give a specific number of points (Table 2). The total score ranges from 0 to 14 and classifies patients into three categories: Low, intermediate and high risk of having NSTI. This score, however, needs to be prospectively validated.

MACRO AND MICROSCOPICS FINDINGS.

The gold standard modality for the diagnosis of NSTI remains the direct visualization of the necrotic tissue.

A diagnosis possibility is to perform a "frozen biopsy". Histological criteria for diagnosing NSTI are necrosis of the soft tissue, polymorphonuclear infiltration of the dermis and fascia, fibrin thrombi with fibrinoid necrosis of arterial and venous walls of arteries and veins coursing through the fascia and the presence of microorganisms within the destroyed fascia and dermis (Fig 3).

Other test is the so called "finger test". This involves infiltrating the suspect area with local anesthetic and making a 2 cm incision down to the deep fascia. If the index finger dissects the subcutaneous tissue off the deep fascia easily along the tissue plane, the test is positive (Fig 4. A).

The positive macroscopic findings are: grey necrotic tissue, fascial edema, thrombosed vessels, thin, watery, foul-smelling fluid, describe as dishwater pus and non-contracting muscles (Fig 4. B).
Fig. 1. Fascias. The superficial fascia described by anatomist is usually not mentioned in current literature. The "deep fascia" and "intermuscular septa" by anatomist are often called "superficial fascia" and "deep fascia" by the orthopedic surgery literature respectively.

Table 1. Classification of NSTI according to etiology.
Fig. 2

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<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>LRINEC score (points)</th>
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<tbody>
<tr>
<td>C-Reactive protein ≥150 mg/l</td>
<td>4</td>
</tr>
<tr>
<td>White blood cell count (cells/mm³)</td>
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<tr>
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<td>0</td>
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<tr>
<td>15-25</td>
<td>1</td>
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<tr>
<td>&gt;25</td>
<td>2</td>
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<tr>
<td>Hemoglobin level (g/dl)</td>
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<td>11.13.5</td>
<td>1</td>
</tr>
<tr>
<td>&gt;11</td>
<td>2</td>
</tr>
<tr>
<td>Sodium level &lt;135 mmol/l</td>
<td>2</td>
</tr>
<tr>
<td>Creatinine level &gt;1.6 mg/dl</td>
<td>2</td>
</tr>
<tr>
<td>Glucose level &gt;180 mg/dl</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 2. Laboratory risk indicator for NSTI (LRINEC). A score ≤ 5 indicates a low risk (<50% probability) of NSTI. 6-7 points indicate an intermediate risk (50-70% probability) of NSTI; ≥ 8 indicates a high risk (>75% of probability) (Wong et al. Crit Care Med 2004; 32: 1535-41)
Fig. 3

Histological findings. Fatty tissue with dead adipocytes and a fascia with necrosis and polymorphonuclear cells.

Fig. 4

“Finger test”.

Surgical findings: grey necrotic tissue, fascial edema, thrombosed vessels and “dishwater pus”.

Fig. 4. Macroscopic Findings. A: “Finger test”. B: Surgical findings.
Findings and procedure details

Although some authors have affirmed that the image test are not indicated, since they could delay the urgent treatment of these patients, imaging test are an invaluable diagnostic adjunct because:

1. They may confirm the diagnosis in cases where signs are unclear.
2. The image test delineates the extent of the disease. This is very useful to map disease extent to aid in planning the surgical approach and margins.
3. They help in the identification of the source and complications of the infection.
4. The image test allows exclude other processes.

Each of the imaging techniques has its advantages and disadvantages:

**Plain film** can only help to identify subcutaneous gas. This is a very specific finding, but it is not very sensitive in patients with NSTI. However, plain film can be normal until the infection and necrosis are advanced and manifest as soft tissue emphysema tracking along fascial planes.

**CT** findings include dermal thickening, increased soft tissue attenuation, inflammatory fat stranding and possible superficial or deep crescent fluid or air in the subfascial planes. CT is the most sensitive modality for soft tissue gas detection.

**MR** is a very sensitive technique, however is often not performed for NSTI evaluation because its acquisition is time-consuming and will delay treatment. In our experience, we don't perform MRI on any of our patients.

**US** has the following advantages:

**US** is rapid, very sensitive for fluid collections and joint effusion, can be performed at the bed side and the images are not degraded by metallic or motion artefacts. No sedation is required; no ionizing radiation is used. Moreover, symptomatic and contralateral side can be compared in different planes and the effect of compression on lesions can be examined. US has been shown a 100% sensitivity for detection of subcutaneous and intramuscular air and a specificity of 85%.

However, the diagnosis performance of US depends heavily on the creativity, experience and patience of the examiner.
US is not indicated for the evaluation of deeper structures, such as pelvis, paravertebral and mediastinal lesions, due to its limited penetration depth, and lack of penetration through bone and air.

In assessing the patient with suspected NSTI, the check list is as follow:

1. **Subcutaneous tissue.**
2. **Superficial and deep fascia.**
3. **Skeletal muscle.**
4. **Other: nerves, arteries, veins and bone contour.**

To perform ultrasound of the soft tissues it is necessary to use transducers of high resolution, with a frequency range between 5-18 mHz according to the area of the body to be explored. The US findings in the NSTI are:

1. **Cellulitis.**
2. **Soft tissue gas.**
3. **Thickening and distortion of the deep fascia (>4 mm; fasciitis).**
4. **Turbid fluid collections along the deep fascia.**
5. **Myositis.**
6. **Intramuscular abscess.**

As we said before, "**NECROTIZING SOFT TISSUE INFECTIONS**" is defined as infection of **any layer within the soft tissue compartment** (dermis, subcutaneous tissue, superficial fascia, deep fascia or muscle) that are associated with necrotizing changes.

1. **NECROTIZING CELLULITIS.**

The normal subcutaneous layer appears hypoechoic on US, with two components: hypoechoic fat interspersed with hyperechoic linear echoes running mostly parallel to skin, which represent connective tissue septa (Fig 5). Veins and nerves may be visualized within the subcutaneous layer.

Cellulitis is a superficial soft tissue infection which involves the skin and subcutaneous tissues. Diagnosis is usually made clinically.
US in non-necrotizing cellulitis demonstrates diffuse thickening and increased echogenicity of the skin and subcutaneous tissue. A cobblestone appearance may be seen with anechoic strands randomly traversing the subcutaneous tissue (Fig 6).

Medical management of uncomplicated cellulitis is with antibiotics and correction of underlying metabolic abnormalities.

Necrotizing cellulitis also known as necrotizing bacterial dermohypodermitis, is characterized by the necrosis of the connective and adipose tissue but no lesions of the deep fascia.

In necrotizing cellulitis, it is possible to observe a loss of the normal pattern with disruption of the subcutaneous cellular tissue, which indicates necrosis. In many cases it is associated to other signs of NSTI such as thickening of the deep fascia or gas in the subcutaneous cellular tissue (Fig 7).

However, the US appearance of necrotizing cellulitis is nonspecific and can be indistinguishable from other causes of soft-tissue edema. For this reason, it is very important to correlate the radiological findings with the clinical findings in this kind of patients.

2. NECROTIZING FASCIITIS.

Normal fascia appears as a linear hyperechoic layer. Its thickness may vary depending on the location. (Fig 8).

Inflammation of the superficial and/or deep fascial planes compatible with fasciitis is characterized by the presence of fascia thickening and fluid tracking on US less than 4 mm. Superficial fasciitis is frequently seen associated with cellulitis and requires similar conservative treatment.

The US findings of Necrotizing fasciitis are:

1. Gas soft tissues.

The presence of gas in the fascial planes caused by gas-forming anaerobic organism is a specific and hallmark sign of NF. This finding is seen in 10-55% in some series. However, this finding appears when the disease is in an advanced stage.

Gas can also be found in subcutaneous tissue.
On US the small foci of gas appear as bright echogenic areas with posterior acoustic shadowing (Fig 9, Fig 10).

DIFFERENTIAL DIAGNOSIS.

There are other causes of subcutaneous emphysema and gas in the fascial planes. The main differential diagnosis is with gas related to trauma or iatrogenic. In these cases there are a history of surgery or trauma and the patients do not show systemic symptoms of sepsis. Sometimes differentiation of NSTI from postoperative changes is difficult. Typically, NSTI involves the fascial planes and sparing of the superficial epidermis. In this cases it is very important to correlate with clinical findings and in some cases serial US examination should show steady resolution.

2. Thickening and distortion of the deep fascia (more than 4 mm on US and more than 3 mm on MRI) and turbid collections or loculated abscess along the deep fascia.

The most common findings in NSTI is asymmetric thickening of the fascia with adipose-tissue infiltration of the skin and reticular infiltration of the hypodermal fat, which is seen in 80% of cases (Fig 11).

However, as thickening of the skin and reticular infiltration of the hypodermal fat occur in both superficial infections and necrotizing fasciitis, the diagnosis of necrotizing fasciitis rest on the concomitant clinical and laboratory findings (Fig 12). The more or less extensive focal inflammation of the hypodermal fat differentiates NSTI from simple stasis edema, which is more symmetric and more diffuse.

DIFFERENTIAL DIAGNOSIS.

There are other causes of thickening and distortion of the deep fascia, such as traumatic disorder (Fig 13). In this cases it is necessary to correlate the radiological findings with the clinical findings. Usually in these cases the thickening of the fascia is less than 3 mm and there are no clinical findings of shock (Fig 14).

3. GAS GANGRENE.

Normal muscle has low echo intensity. In the transverse plane, perpendicular to the long axis of the muscle, the muscle has a speckled appearance because of reflections of perimysial connective tissue, which is moderately echogenic (Fig 15A). In the longitudinal
plane (along the long axis of the muscle) the fascicular architecture of the muscle becomes visible. Reflections of the perimysial connective tissue results in linear, pinnate or triangular structure on the ultrasound image (Fig 15B).

It is very important to distinguish between pyomyositis and necrotizing muscle infection, since these are two different processes with different prognosis and require different treatment strategies.

**Pyomiositis** is pyogenic infection of skeletal muscle characterized by muscle swelling and its frequently leads to abscess formation, which is a focal collection of inflammatory cells, bacteria, and necrotic tissue debris contained by hypervascular connective tissue. The causative agent is **S. Aureus** in over 90% of cases. In the majority of patients, pyomiositis **usually involves a single muscle**, but multiple-site involvement is present in up to 40% of cases. The US appearances have been reported and correspond to the two stages of the disease. The first stage is **typically subacute**, and consists of a phlegmon, which is characterized by localized muscle edema and appears as nonspecific, hypoechoic, ill-defined area within one or more muscles. Later in the course of the disease, and intramuscular fluid collection of mixed echogenicity, which may be surrounded by thick hyperechoic and often hyperemic wall. Adjacent tissues are usually edematous and hyperemic consistent with phlegmon with increased color and Doppler signal. Septations are frequently present and may be sparse, thin, and incomplete, or numerous and thick. Increased through transmission is typical present. Air bubbles may be manifested as small, possibly mobile, hyperechogenic foci with dirty shadowing. Dynamic compression may demonstrate swirling of the contents (Fig 16). US is very sensitive in the detection of clinically occult fluid collections and may be used to guide aspiration.

Treatment for pyomiositis/abscess is surgical debridement and intravenous administration of antibiotics.

**Gas Gangrene.** This is the NSTI that affect the skeletal muscle.

It is usually caused by **Clostridium perfringes**. This infection occurs in a variety of setting, including traumatic wounds with soil contamination, surgery involving the bowel or biliary system or associated with septic abortions and unhygienic injections of medications or illicit drugs.

Less common, clostridial myonecrosis may occurs spontaneously without a prior wound. These cases are often due to Clostridium septicum bacteremia and usually involved and underlying gastrointestinal process, such as occult colon cancer, bowel infarction or neutropenic enterocolitis.
The patients show symptoms including **intense pain** and edema, which occur **several hours** after injury. Classic findings include the presence of gas in the tissues.

US can demonstrate the muscle hypo or hyperecogenic swelling (myositis) and hyperechoic foci with reverberation artifacts (gas). The **involvement of three or more compartments** in one extremity has also been describe as a NSTI indicator. A characteristic features of muscle is that its appearance on ultrasound changes with contraction; it appears thicker and more echoic on short-axis planes when contracted. An abscess-like delineation of the necrotic tissue has also been observed (Fig 17).

Treatment requires emergent surgical exploration, debridement and excision of the infected muscles and fasciotomies.

DIFFERENTIAL DIAGNOSIS.

- **Edema-like infiltration** of the muscle can occur in various noninfectious disorders such as idiopathic inflammatory myopathies, eosinophilic fasciitis, lymphedema, trauma to the muscles and aponeurosis, and myonecrosis.

Myonecrosis can occur in sickle cell crisis, compartment syndrome, crush injury, severe ischemia, intra-arterial chemotherapy (Fig 14), rhabdomyolysis and diabetic myonecrosis. Diabetic myonecrosis is a rare manifestation of long-standing (mean duration, about 15 years) and poorly controlled diabetes mellitus and can be seen in both type I and II diabetes. The most accepted cause for myonecrosis is diffuse microangiopathy. In diabetic myonecrosis the entire muscle is usually involved in edema, and the process may be bilateral. A history of poorly controlled diabetes or absence of leukocytosis suggests the presence of diabetic myonecrosis, which usually does not require aspiration (Fig 18).

- **Necrotic tissue with peripheral rim.**

Differential diagnosis at this stage includes other lesions resembling soft-tissue masses, such as neoplasm, intramuscular hematomas (Fig 19) myonecrosis (Fig 20) sarcoidosis and parasitic infections.

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The treatment of NSTI is urgent surgical debridement, decompression fasciotomy and intravenous administration of antibiotics. Hyperbaric oxygen can be used as a supplemental modality.
Fig. 5. Normal subcutaneous tissue appears hypoechoic on US, with two components: hypoechoic fat interspersed with hyperechoic linear echoes running mostly parallel to skin, which represent connective tissue septa (Fat lobules → Connective tissue septa)

Fig. 6. Cellulitis. 67-year-old female who had been beaten by her dog. She has an erythema in the volar aspect of the forearm that progressively increases. US demonstrates diffuse thickening and increased echogenicity of the skin and subcutaneous tissue associated to a cobblestone appearance, without thickening of the deep fascia and without soft tissue gas, consistent with cellulitis.
Fig. 7. Necrotizing Cellulitis. 53-year-old male with aplastic anemia and fever of unknown origin. As a casual finding CT demonstrates cellulitis in the perineal region and scrotum (A). Complementary US was performed at the bed side. US showed cellulitis (B) and a loss of the normal pattern with disruption of the subcutaneous cellular tissue, which indicates necrosis (C). The patient died 2 days after. Necropsy confirmed the presence of necrotizing cellulitis, with no evidence of necrotizing fascitis or affection of deep muscular planes.

Fig. 8. Normal fascia appears as a linear hyperechoic layer. (★ Superficial fascia → Deep fascia)
Fig. 9. NSTI. Gas soft tissue. 44-year-old male drug addict. History of heroin injection diluted with contaminated water. US shows small foci of gas appear as bright echogenic areas with posterior acoustic shadowing (A and B). US and CT also demonstrate an abscess located anterior to the Achilles tendon (C and D). Initial treatment was fasciotomy and subsequent amputation of the lower right limb (*GAS).

Fig. 10. NSTI. Gas soft tissue. 18-year-old girl diagnoses acute lymphocytic leukemia 20 days before. Severe pain in both legs after using a depilatory cream. US shows cellulitis (A) and multiple foci of gas with posterior acoustic shadowing (B). Treatment was urgent fasciotomy, however the patient died a few hours later.
Fig. 11. NSTI. Necrotizing fasciitis. Thickening and distortion of the deep fascia. 56-year-old male, previously healthy, comes to the emergency room due to severe pain in the lateral aspect of the right thigh. US was performed to rule out deep venous thrombosis. US showed thickening of the deep fascia more than 4 mm (*) associated with muscle hyperecogenic swelling (myositis). These findings are not specific but are highly suggestive of necrotizing fasciitis. A few hours later the patient has septic shock. Urgent fasciotomy is performed. The histological study demonstrated the presence of necrotizing fasciitis secondary to Group A Streptococcus pyogenes.

Fig. 12. Deep Fasciitis. 42-year-old male with history of depression. Severe pain in abdominal wall and septic shock. Abdominal CT did not show significant findings. Retrospectively an asymmetric thickening of the deep fascia in the abdominal wall is observed (*). Complementary US was performed at the bed side. US showed thickening of the deep fascia more than 4 mm (*). Emergency surgery was performed and abundant pus was obtained. The histological study demonstrated deep fasciitis without necrosis.
**Fig. 13**

Hematoma. 53-year-old woman with sport-related acute pain in the middle portion of the calf. US demonstrate a large collection / hematoma (H) between the gastrocnemius (G) and the soleus (S).

**Fig. 14**

Myonecrosis. 42-year-old female with history of Myeloid leukemia present acute pain in the middle portion of the calf after intraarterial chemotherapy. US shows thickening of the deep fascia less than 4 mm (*) associated of muscle hyperecogenic swelling (myositis) and mass lesion pattern. MRI demonstrates the same findings. The patient has no clinical or laboratory data suggesting NSTI, and is suspected myonecrosis due to intraarterial chemotherapy. Conservative treatment was performed with subsequent resolution.
Fig. 15. Normal muscle has low echo intensity. In the transverse plane, the muscle has a speckled appearance because of reflections of perimysial connective tissue, which is moderately echogenic (*Fig 15A). In the longitudinal plane the fascicular architecture of the muscle becomes visible. Reflections of the perimysial connective tissue results in linear, pinnate or triangular structure (→Fig 15B).

Fig. 15

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Fig. 16. Pyomyositis. 44-year-old female. History of psychiatric pathology. Inoculation of foreign body in left arm. On US we can see intramuscular fluid collection of mixed echogenicity with air bubbles (*) manifested as small, mobile, hyperechogenic foci with dirty shadowing. Surgical debridement was performed and abundant pus was obtained, secondary to pyomyositis by S. aureus.

Fig. 16

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Fig. 17

65-year-old male with history of Hodgkin's lymphoma and colostomy. He refers that in one of the changes of the colostomy bag, fecal contents fall in the surgical wound. Later begins with intense pain and swelling in the quadriceps muscle. US showed muscle hypoechoic swelling (myositis) and hyperechoic foci with reverberation artifacts (gas). The patient presents clinical symptoms of septic shock. Surgery was performed and the traumatologist observed necrosis in the fascia. The AP study confirmed the presence of necrotizing fasciitis and gas gangrene secondary to C. Septicum.

Fig 18

Diabetic myonecrosis. 50-year-old male with poorly controlled diabetes. He presented at the emergency room with chronic pain in both lower limbs that has worsened in the last few days. US showed bilateral increased thickness and echogenicity of the biceps tendon, semitendinous and semimembranous muscle. The patient did not present clinical symptoms of infection. Three months later, a follow-up US showed atrophy of the musculature in the posterior region of both limbs.
Fig. 19. Hematoma. 34-year-old male. Severe pain in the right calf after direct trauma. US shows an intramuscular, inhomogeneous mass in the lateral belly of the gastrocnemius muscle.

Fig. 20. Myonecrosis. Crush injury. 45-year-old male with history of drug abuse. The patient was found lying on the street. He present swelling on his left hip. CT scan angiography was performed to rule out Pulmonary embolism. Lab test showed increased of CPK, acute renal failure and metabolic acidosis. The gluteus medius muscle presented increased thickness with formation of pseudomasses (A). The US showed myositis and mass lesion pattern (B). The MRI confirmed these findings (C). The clinical diagnosis was crush injury. The patient was treated conservatively.
Conclusion

NSTI is highly lethal infections. They encompass a wide variety of soft tissue infections associated with necrosis that share the same diagnostic and treatment principles. Accuracy increases with familiarity of clinical findings, laboratory, macro and microscopic findings and imaging test.

US is a very useful methods of imaging. US provides important information in NSTI.

The most common US findings in NSTI is asymmetric thickening of the fascia (>3mm; fasciitis), but the absence of US abnormalities of the intermuscular fascia do not rule out other form of NSTI.

The presence of gas in the fascial plane is highly specific but rare.

This diagnosis capacity adds to other unrivalled advantages of US such as portability, availability, speed and patient comfort.
Personal information

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References


