**Necrotizing Soft Tissue Infection after Laparoscopic Bariatric Surgery. Case report and review**

Jorge Franco, Juan del Castillo, Álvaro Velásquez

Bariatric Surgery Department, Nuestra Señora de los Remedios Clinic, Cali, Colombia

**ABSTRACT:** Background: Necrotizing Soft Tissue Infection (NSTI) is a rare and aggressive form of infection of the fascia and subcutaneous tissue without pathognomonic signs. No single organism or combination of organisms is consistently responsible for NSTI. This paper reports the first case of fatal NSTI after an uneventful Laparoscopic Bariatric Surgery (LBS). A PubMed/Medline database search (1985-2010) did not identify any reports of NSTI after LBS. Early diagnosis and an aggressive approach to treatment with initial debridement followed by planned repeat debridement in conjunction with antibiotic and multidisciplinary support remains the mainstay of treatment.

**Key words:** Necrotizing soft tissue infection, Laparoscopic Bariatric Surgery.

**Aim:** Systematic review of the literature on NSTI after LBS and report of a case encountered in our experience of over 1650 patients. **Patients and Methods:** We have used PubMed to search MEDLINE for articles published between January 1, 1985, and May 31, 2010, using the search terms necrotizing soft tissue infection, laparoscopic bariatric surgery. Additional articles culled from references were obtained. The inclusion criteria were documented NSTI by imaging studies such as, magnetic resonance imaging (MRI) or deep incisional skin frozen section biopsy, or surgery following LBS. We include the case from our institution. **Results:** No identifiable reports were found of NSTI after LBS. **Conclusions:** Laparoscopic surgeons should be aware of the risk of NSTI after LBS, although it is a very rare complication, because it may be fatal. A high index of suspicion is required for early surgical and medical intervention.

**INTRODUCTION**

Necrotizing Soft Tissue Infection (NSTI) includes a broad category of bacterial and fungal skin infections characterized by aggressive infection syndrome with rapid necrosis of subcutaneous fat and fascia that is associated with high mortality rates. NSTI has traditionally represented an uncommon but particularly pathogenic disease entity since its original description by Meleney in 1924.

There were no cases reported after LBS. Our patient represents the first report of NSTI after LBS without risk factors or iatrogenic gastrointestinal complications. Despite surgical advances and the introduction of antibiotics, reported mortality rates for NSTI range from 6 to 76%.

**CASE REPORT**

A 44-year-old male (BMI 40) in 2007 had an uneventful gastric MiniBypass for Morbid Obesity. The surgical procedure lasted 75 minutes, and he was discharged in stable condition after 2 days in the Clinic. 20 months later, the patient had Laparoscopic conversion to Gastric Sleeve due to chronic diarrhea and malnutrition. His BMI was 19 and surgical time was 1 hour (resection of gastrojejunal anastomosis and anastomosis of the pouch gastric to the antrum, with extraction of fundus and gastric body). He was discharged after 1 day in the Clinic. The patient was readmitted to the Clinic 3 days later, presenting to the emergency department with complaints of back and right flank pain, as well as abdominal pain at the site of subcutaneous application of the heparin. He was admitted with dysnea, tachycardia. TA: 86/54, HR: 144, T:36°C and pain in abdominal wall right flank. Abdominal ultrasound showed right flank hematoma without free liquid in the abdominal cavity. The patient was started on
Anatomic factors are important in explaining the facility with which NSTI cause damage. Most bacteria and fungi can multiply within viable tissue, but fibrous attachments or "boundaries" between subcutaneous tissues and fascia (scalp, hands, etc.) can help limit the spread of infection. The natural lack of fibrous attachments in the larger areas of the body (e.g., trunk, extremities) facilitates widespread infection. Generally, bacterial and toxin-related effects converge to cause skin necrosis, shock, and multisystem organ failure.

The time course for NSTI varies. Infection can progress over days to weeks; more often, however, limb-threatening or life-threatening sequelae manifest within only a few hours after the infection begins. There are three general types of NSTI identified. The fulminant form is characterized by shock over several hours and is associated with a high mortality. The acute form is associated with symptoms that develop over more than one to several days with large areas of necrosis; finally, the subacute form has a more indolent initial course, gradual tissue necrosis, with progression despite the use of antibiotics and is followed by sudden deterioration. The clinical courses of laparoscopic-related NSTI often suffered the acute form. Symptoms manifested within the first week after the operation.

Reported risk factors for NSTI include age greater than 50 years, peripheral vascular disease, diabetes mellitus, malnutrition, atherosclerosis, high co-morbid index scores (i.e., APACHE or Surgical Infection Stratification System), obesity, hypoalbuminemia, chronic alcoholism, and intravenous drug abuse. Many of these risk factors reflect an immunocompromised state.

Although NSTI can be monomicrobial, they usually are synergistic polymicrobial infections. In fact, the synergistic action of aerobic and anaerobic bacteria could be responsible for the fulminant course of this disease. A study found that only 28 of 182 patients developed necrotizing skin infections from single pathogens; the other 154 patients had polymicrobial infections. In this study, the majority of monomicrobial infections were caused by streptococcal isolates such as B-hemolytic streptococci (namely group A streptococci or streptococcus pyogenes). Other frequently cited causes of monomicrobial NSTI include staphylococcus aureus and Clostridium perfringens. Fungal infections also have been reported. In another study, 66% of NSTI were found to be polymicrobial, and 29% were caused by single pathogens.

The Physical Examination should cover all body surfaces. As necrotizing skin infections begin in deep tissue planes, the epidermis may appear relatively unscathed until late in the course of infection. However, some clinical clues are available: in skin: erythema,
tense edema, grayish or other discolored wound drainage, vesicles or bullae, necrosis, ulcers, crepitus. The presence of crepitus is variable. In one study, crepitus was present in only 18% of patients with necrotizing fasciitis and was a late clinical sign. Because of the paucity of distinct findings, NSTI may still be missed. Bullae and skin necrosis, for example, may not be present in 66 to 70% of patients with occult infections. 1) Pain that extends past the margin of apparent infection; severe pain that appears disproportionate to physical findings; decreased pain or anesthesia at apparent site of infection. 2) General symptoms: fever, tactile temperature, diaphoresis, tachycardia, toxic delirium (systemic infection), they may become hypotensive and show signs of renal failure and hemolytic anemia. 

Early diagnosis and aggressive surgical intervention are critical to patient outcomes. There are no established diagnostic criteria and no standard definitions for NSTI to date. Diagnosis is primarily clinical. Most importantly, severe pain disproportionate to local findings along with systemic toxicity should raise suspicion for the possibility of NSTI. Tenderness extending beyond the port site, erythema and swelling appear first. Foul smelling discharge from the port site may become apparent. The mortality of this disease remains high, reportedly from 6 to 76%. Physical findings are not sufficient to identify the organisms that cause the infections. The gold standard for detecting NSTI is tissue biopsy obtained at the time of wound exploration and surgical debridement. The findings of fascial necrosis and myonecrosis are indicative of necrotizing infection. Others diagnostic tests with negative results cannot exclude necrotizing skin infections, for example, an absence of soft tissue gas on radiographs does not rule out as a possibility these infections. Different radiological methods have been used for the diagnosis of NSTI. Plain radiography typically shows no specific abnormality until advanced stages of NSTI are present with internal gas formation or soft tissue thickening. Both CT and MRI facilitate the detection of fascial thickening, focal fluid and soft tissue abnormalities. If findings such as tense skin edema, crepitus, bullae, and radiologic and laboratory abnormalities are present, they provide additional impetus to urgently start wound exploration. Wong developed a simple and objective scoring system, called the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score, based on routine laboratory investigations including C-reactive protein, WBC, hemoglobin, serum sodium, creatinine, and glucose that can distinguish NSTI from other soft tissue infections with high specificity even in early cases. The finger test to determine whether tissues are dissected with minimal resistance and a deep incisional skin frozen section biopsy can be used to confirm the diagnosis of NSTI.

TREATMENT

Treatment of NSTI is primarily surgical. Controlled surgical debridement of necrotic and diseased tissues remains the cornerstone of treatment and can increase survival in patients with NSTI. In various studies, patients who underwent surgical debridement more than 12 hours after hospital admission had higher amputation and mortality rates. Definitive surgery, debridement of all necrotic tissue, should be performed during the first operation regardless of the extent of involvement. Postoperative wound re-evaluation and debridement must be performed frequent. The possibility of adjacent or deeper sites of occult necrosis and infection must be excluded. With the resolution of the necrotizing infection and the establishment of granulation tissue, surgical attention can be directed toward coverage of tissue defects caused by the infectious process. They often require extensive skin grafting or tissue transfer. Empiric antibiotic therapy can be employed until wound culture isolates are identified. Because most NSTI are polymicrobial, broad-spectrum coverage is advisable. Hyperbaric oxygen therapy has been a controversial adjunct in the management of NSTI. It is not recommended as a replacement for surgical debridement or intravenous antibiotic therapy. Surgery, use of broad-spectrum antibiotics, aggressive fluid resuscitation, nutritional support, and intensive supportive care are the most important treatment modalities. Survivors typically have a prolonged hospital stay.

CONCLUSION

NSTI after LBS has not been reported yet and it can be fatal. Any deviation from a normal postoperative course may signal a problem, which could be aggressive NSTI. Unfortunately, there are no pathognomonic signs. As the infection progresses, their pain may decrease. No single organism or combination of organisms is consistently responsible for NSTI. Early diagnosis and an aggressive approach to treatment with initial debridement followed by planned repeat debridements in conjunction with broad spectrum antibiotics and nutritional support remains the mainstay of treatment.

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AUTHOR INFORMATION

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