

Necrotizing Fasciitis - A Dreaded Complication Following Laparoscopic Cholecystectomy – A Case Report With Review Of The Literature

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Citation

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Abstract

INTRODUCTION: Necrotizing fasciitis is a progressive infection of the fascia with secondary necrosis of subcutaneous tissues. The spread of disease is directly proportional to the thickness of the subcutaneous layer. Within a short span of time it destroys skin and subcutaneous tissue. **CASE PRESENTATION:** A 40-year-old female patient with history of acute cholecystitis was admitted for laparoscopic cholecystectomy. On the second postoperative day, she developed fever and pain with skin discoloration at the site of the drain kept due to biliary spillage during the procedure. Within 6-12 hours, her clinical condition deteriorated. She was diagnosed clinically as having necrotizing fasciitis. High-dose antibiotics and pressure supports were started. Wound debridement was done but the patient entered into multiple organ dysfunction syndrome for which she was kept on ventilator support. She expired on the 4th postoperative day. **DISCUSSION:** Necrotizing fasciitis is very rare in the era of laparoscopy and with the availability of broad-spectrum antibiotics. It is a nightmare for the surgeon if a patient has necrotizing fasciitis after laparoscopic surgery as mortality is very high. **CONCLUSION:** Skin discolorations with crepitus and disproportionate pain at the trocar site combined with rapid deterioration of vital parameters led to the diagnosis of necrotizing fasciitis in our patient. Early recognition, intensive care treatment, and aggressive wound debridement are the cornerstones for successful treatment. Mortality remains still high despite use of modern powerful antimicrobial drug regimens and advances in the care of critically ill patients. The available medical literature related to this problematic topic was reviewed and included in this presentation.

INTRODUCTION

Necrotizing fasciitis (NF) is commonly known as flesh-eating disease or flesh-eating bacteria syndrome. It is a serious bacterial infection that spreads rapidly and destroys the body's soft tissue. Superficial fascia and subcutaneous tissue are primarily involved with relative sparing of skin and underlying muscles. [1]

With the advent of broad-spectrum antibiotics this soft-tissue infection is uncommon nowadays. It is usually caused by virulent organisms. Different types of organisms may cause necrotizing fasciitis (e.g., Group A Streptococcus, Staphylococcus aureus, Clostridium perfringens, Bacteroides fragilis, Aeromonas hydrophila). [2]

Immunosuppressed patients, intravenous drug abusers, diabetics and alcoholics are prone to necrotizing fasciitis. The disease may affect young and healthy individuals. The most common sites of infection are abdominal wall, perineum, and extremities, where more subcutaneous tissue

is present. The entry of pathogens into the subcutaneous space happens through disrupted skin like trocar/drain sites or by hematogenous spread from a distant site of infection. Polymicrobial necrotizing fasciitis is usually caused by enteric pathogens, whereas monomicrobial necrotizing fasciitis is usually due to the patient's own skin flora. [3] The early symptom of NF is disproportionate pain at the skin wound associated with multiple blister/vesicle formation. A mottled, flaky appearance of the skin shows the tissue underneath begins to die. The patient develops flu-like symptoms, thirst, diarrhoea, vomiting, fatigue, frustration and/or altered consciousness due to release of cytokines and bacterial toxins. NF progresses rapidly and can cause death. NF needs prompt clinical diagnosis. Aggressive treatment like wound debridement, high-dose antibiotics and critical care support have been associated with improved survival. Mortality rates are as high as 76-80% with the cause of death being overwhelming sepsis syndrome and/or multiple organ system failure. [4]

Necrotizing Fasciitis - A Dreaded Complication Following Laparoscopic Cholecystectomy – A Case Report With Review Of The Literature

Necrotizing fasciitis after laparoscopic cholecystectomy is rare. We got this dreaded complication after laparoscopic surgery and the clinical deterioration of the patient was so fast that she landed up in multiorgan failure before we could understand the disease process.

CASE HISTORY

A 40-year-old female was brought by her relatives with a 3-day history of fever, chills, upper abdominal pain, jaundice and altered sensorium. She did not have diabetes or hypertension. On admission, she was drowsy, febrile and icteric. Her pulse rate was 110/minute and her systolic blood pressure was 80mmHg. Abdominal examination revealed tenderness over the right hypochondriac region with localized guarding and rigidity. She was admitted with a clinical diagnosis of acute cholecystitis with hypotension. Her hemoglobin level was 9g/dl, total leucocyte count was 25,000/cmm with 89% polymorphs and total serum bilirubin was 4.2mg/dl with a direct bilirubin of 2.3mg/dl. The alkaline phosphate level was 284 International Units. Bed-side ultrasonography of the abdomen was suggestive of acute calculous cholecystitis. The common bile duct diameter was 8mm. Intravenous fluid resuscitation and intravenous imipenem with cilastatin was started after sending the urine and blood culture. The patient improved with supportive therapy. There was no growth in blood or urine culture. On the 7th day of admission, magnetic resonance cholangio-pancreaticography was done. The image revealed cholecystitis with multiple stones in the gall bladder. Common bile duct and pancreatic duct were found to be normal. The patient was counseled for elective laparoscopic cholecystectomy. She was discharged with cefuroxime 500mg twice daily for 7 days.

After 6 weeks she was readmitted for elective laparoscopic cholecystectomy. On examination, she was afebrile with normal liver function tests. She underwent laparoscopic cholecystectomy. An intravenous cefoperazone-sulbactam combination was administered before the induction of anesthesia. The procedure was done with 4 standard ports. Calot's triangle was dissected with mild difficulty. There was perforation of the gall bladder and minimal spillage of bile and pus, for which a drain was placed through the right upper quadrant lateral trocar site. Pus was sent for culture. The immediate postoperative period was uneventful. The patient was mobilized and allowed oral nutrition on the first postoperative day. On the second postoperative day the patient developed a low-grade fever with severe pain at the drain site. Clinically, the abdomen was soft and bowel

sounds were present, with a drain output of 75ml in 24 hours. On examination, dark violet skin discoloration with blisters and edema was seen around the drain site [Fig. 1]. Crepitus was present at the dusky colored skin. In less than six hours she developed a fever of 104°F, tachycardia and tachypnea with a systolic pressure down to 80mmHg. The patient's condition deteriorated further with decrease in the level of consciousness.

Blood samples were collected for complete blood picture, erythrocyte sedimentation rate, blood glucose, liver and renal function tests. Fluid from the blisters was sent for culture and sensitivity. With clinical suspicion of necrotizing fasciitis, broad-spectrum antibiotics, fluid replacement and pressure supports were stated. The clinical condition did not permit to proceed to CT/MRI imaging; therefore, bed-side X-rays and abdominal ultrasonography were done. Soft-tissue ultrasonography showed a fluid collection in the subfascial and muscle plain. After fluid resuscitation, the patient was transferred to the operating room. The drain site was re-opened, revealing a large area of subcutaneous fat and fascia necrosis. Abdominal wall debridement was done and the wound was left open. The patient was kept on ventilator support and expired on the 4th postoperative day. E. coli was isolated from the spilled bile/pus. The necrotic tissues and fluid grew Group A Streptococcus and coagulase-positive staphylococci.

Figure 1

Skin discoloration at the drain site



DISCUSSION

In the era of broad-spectrum antibiotics, necrotizing fasciitis (NF) is rare after laparoscopic surgery. An online literature search was made and the published case reports of necrotizing fasciitis after different types of laparoscopic surgery were noted in table 1.

Necrotizing Fasciitis - A Dreaded Complication Following Laparoscopic Cholecystectomy – A Case Report With Review Of The Literature

Table 1

Laparoscopic surgery after which necrotizing fasciitis occurred

Laparoscopic surgery after which necrotizing fasciitis occurred	Authors	Patient status
After laparoscopic appendectomy	Ahmed et al. [12]	Expired
After laparoscopic colonic surgery	Tan et al. [13]	Survived
After laparoscopic left hemicolectomy for diverticulitis	Bradik G et al. [14]	Survived
After laparoscopic cholecystectomy	Mohammadhoseni et al. [4]	Expired
After laparoscopic cholecystectomy	Mittermair et al. [15]	Survived - hospital stay - 5 months
After laparoscopic appendectomy	Svaton et al. [16]	-
After diagnostic laparoscopy	Bharathan et al. [17]	-
After total extraperitoneal repair of a left inguinal hernia	Golash et al. [18]	Survived

The pathophysiology of NF is due to proliferation of virulent organisms within the superficial fascia and subcutaneous tissue. The released enzymes help the bacteria to spread through the fascia. There is microbial invasion with occlusion of nutrient vessels of the skin causing ischemia and necrosis. [2]

The clinical features of NF [2, 3] are (a) local symptoms: erythematous skin, blister and bulla formation with crepitus, skin gangrene (b) systemic symptoms: fever with flu-like symptoms, nausea, vomiting, diarrhea, fatigue and frustration (c) late symptoms: clinical deterioration of the patient's vital parameters, altered consciousness and hypotension.

Laparoscopic cholecystectomy is the procedure of choice for gall bladder diseases and NF after this type of surgery is a rare but dreaded complication. Postoperatively, the patient complains of trocar site pain and dusky coloration of the drain site should not be ignored. Any deviation from the normal postoperative course may signal a problem which could be aggressive NF. The mortality of this disease is high. A literature review shows that 4 out of 14 laparoscopic surgery cases with NF died. [4,8,9,10]

Diagnosis of NF starts with clinical suspicion only. Sudden deterioration of a healthy patient makes the surgeon think of postoperative intra-abdominal complications.

Ultrasonography, computerized tomography and magnetic resonance imaging of the abdomen help to exclude intra-abdominal postoperative catastrophes. Fascial thickening and fluid collection in the subcutaneous/muscle plain on CT/MRI images are the features of NF. [5]

The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score is based on C-reactive protein, total

leukocyte count, hemoglobin percentage, electrolytes, creatinine and blood glucose levels, and has high specificity [6]. Patients with LRINEC score of >6 have a higher rate of mortality. In our patient the LRINEC score was 11 (table 2).

Table 2

LRINEC score

Variables (unit)	Score	Points	Patient score	Points
Hemoglobin (g/dl)	>13.5	0		
	11-13.5	1		
Total leukocyte count/mm ³	<11	2	9.2	2
	<15,000	0		
Serum creatinine (mg/dl)	1.500-2.5,000	1	22,500	1
	>25,0000	2		
Glucose (mg/dl)	>1.6	2	2.7	2
	<180	0	90	0
Sodium (mmol/L)	>180	1		
	>135	0		
CRP (mg/dl)	<135	2	122	2
	<15	0		
Total	>15	4	17	4
	---		---	11

Various organisms have been isolated from patients with fascial necrosis and systemic toxicity. Culture from necrotic skin/blisters may identify causative organisms. The synergistic action of aerobic and anaerobic bacteria is responsible for the severity of the disease.

Bacteriologically, NF is divided into three types [7]:

Type I: polymicrobial; occurs after trauma

Type II: Group A Streptococcus; flesh-eating bacterial infection

Type III: clostridial myonecrosis or gas gangrene

In our case the organisms isolated from the blister fluid were Group A Streptococcus and coagulase-positive Staphylococcus.

Treatment of NF is primarily surgical and needs a multidisciplinary approach. Wound debridement of necrotic tissue is the priority. Use of broad-spectrum antibiotics, aggressive fluid resuscitation, nutritional support, and intensive supportive care are important treatment modalities. Survivors typically have a prolonged hospital stay. They often require extensive skin grafting or tissue transfer, but long-term morbidity is minimal. [11] In our case, we had clinical suspicions of NF but we never thought that the patient would deteriorate so rapidly.

CONCLUSION

Laparoscopy is the gold standard for cholecystectomy. With time and refinement of technique and standardization of the procedure, the incidence of complications has been reduced significantly, but necrotizing fasciitis following laparoscopic cholecystectomy is a night-mare complication for the surgeon and hard to digest for the patient relatives. Even if it

Necrotizing Fasciitis - A Dreaded Complication Following Laparoscopic Cholecystectomy – A Case Report With Review Of The Literature

is rare, a clinical scenario with disproportionate pain at the port/drain site and dusky skin changes should not be ignored and a differential diagnosis of necrotizing fasciitis should be kept in mind. Wound debridement and aggressive intensive care are the key in management. Even with all advances in antibiotics and organ support, the mortality rate is still high today.

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