

Necrotizing Fasciitis of the Abdominal Wall Caused by Invasive Group D Enterococcus faecalis Secondary to Infected Intra-abdominal Drainage

J Carvajal Balaguera, J Archilla Estevan, A Smaranda, J Camuñas Segovia, S Martín Alcrudo, C Cerquella Hernández

Citation

J Carvajal Balaguera, J Archilla Estevan, A Smaranda, J Camuñas Segovia, S Martín Alcrudo, C Cerquella Hernández. *Necrotizing Fasciitis of the Abdominal Wall Caused by Invasive Group D Enterococcus faecalis Secondary to Infected Intra-abdominal Drainage*. The Internet Journal of Surgery. 2007 Volume 17 Number 1.

Abstract

Necrotizing fasciitis (NF) is a relatively rare soft-tissue infection that is characterized by rapidly widespread necrosis of the fascia and subcutaneous tissue.

We discuss the successful saving of an 80-year-old woman with necrotizing fasciitis of the abdominal wall caused by invasive group D Enterococcus faecalis, secondary to postoperative drainage inserted during right hemicolectomy for colon cancer. Other factors such as a long hospital stay before surgical intervention, chronic anemia secondary to intestinal haemorrhage due to angiodysplasia, multiple preoperative transfusions of blood and chronic hepatopathy secondary to virus hepatitis B favoured its development.

The patient underwent repeated procedures of debridement, and was treated with respiratory and hemodynamic support, broad-spectrum antibiotics, parenteral and enteral nutrition. She recovered forty days after admission to the Intensive Care Unit. We discuss the need to recognize necrotizing fasciitis as a potential complication of infected postoperative drainage, as once necrotizing fasciitis occurs, mortality is more likely. It is also important to point out that intraabdominal drainage should be placed with great care due to the severity of possible complications.

We conclude that recognition and aggressive surgical debridement of NF is mandatory. We also address the issue of the importance of respiratory and hemodynamic support and alimentation in these patients.

INTRODUCTION

Necrotizing fasciitis (NF), is defined as a relatively rare soft-tissue infection which causes widespread necrosis of fascia and subcutaneous tissue while sparing muscle and skin ¹.

The trunk (37,1%), groin, perineum and extremities are the most frequently involved sites. The eyelids, scalp, face and neck are only rarely involved ¹. Late diagnosis and an impaired immune response, such as seen in diabetes mellitus, help account for the high mortality and morbidity associated with this entity ².

This entity of the abdominal regions may arise from infections coming from urogenital disease, colorectal pathology or infected wounds. In some occasions, the agent gains entry through intact skin and in many cases the source is unknown ³.

Known iatrogenic causes of NF that have been published in

the medical literature are traumatic urinary catheterization ^{4,5,6}, prostatic massage ⁵, prostatic biopsy ⁵, percutaneous cardiac revascularization ⁷, transthoracic percutaneous biopsy ⁸, liposuction ⁹, intramuscular injection of NSAIDS ¹⁰, laparoscopic colonic surgery ¹¹, artery cannulation ¹², and along the surgical sutures or at the drain site in the abdominal wall after operation in the abdomen ¹³, as in our case.

To our knowledge, this is the first case of NF associated with postoperative intraabdominal drainage after colon surgery.

This article reviews the epidemiology, aetiology, pathogenesis, clinical diagnosis, radiological examinations, laboratory tests and management principles of this entity.

CASE REPORT

An 80-year-old woman was admitted to our hospital one month before surgery because of significant anemia; she had

Necrotizing Fasciitis of the Abdominal Wall Caused by Invasive Group D Enterococcus faecalis Secondary to Infected Intra-abdominal Drainage

a previous history of chronic liver disease secondary to virus hepatitis B, hypopituitarism requiring steroid replacement, allergy to betalactamics, mitral regurgitation and paroxysmic atrial fibrillation.

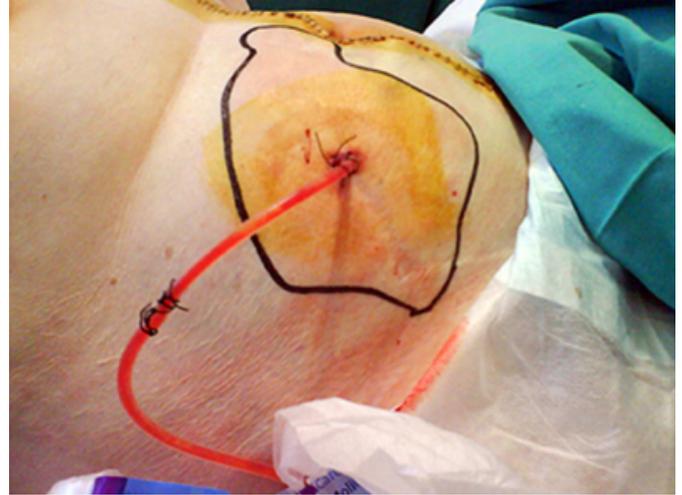
She was diagnosed with anaemia secondary to digestive haemorrhage due to colon neoplasia and intestinal angiodysplasia and required several blood transfusions.

After antibiotic prophylaxis with Gentamicin and Metronidazole, an open right hemicolectomy was uneventfully performed and a drain (Jackson - Pratt, Fortune Medical Instrument Corp.) was left in place in proximity to the surgical area to rule out an anastomosis leak, inadequate hemostasis or injury to adjacent organs. Histological study of the surgical specimen was compatible with a Dukes B stage or B2 Astler and Staller stage colon cancer.

During the first 5 days, the postoperative course was uneventful. Six days after surgery, the patient had to be admitted to the Intensive Care Unit (ICU), because she developed abnormal vital signs with tachypnea, tachycardia, chills, and general malaise. On admission to the unit, she looked pale, was confused and in mild distress. Blood pressure was 95/40 mmHg, pulse was weak at a rate of 122 beats/min, respiratory rate was 28 breaths/min, axillary temperature was 38°C, and pulse oximetry was 98% with supplemental oxygen at 3 l/min per nasal cannula. Auscultation of the lung revealed reduction in breath sounds; the heart was arrhythmic and hyperdynamic without murmurs. The abdomen was slightly distended with decreased bowel sounds. An erythematous, warm, painful and swollen area could be observed around the abdominal drainage (Fig. 1). No crepitation was palpable across the compromised area. There was no guarding or rebound and both the pelvic and rectal exams were normal. Bilateral lower extremity oedema was also present.

Figure 1

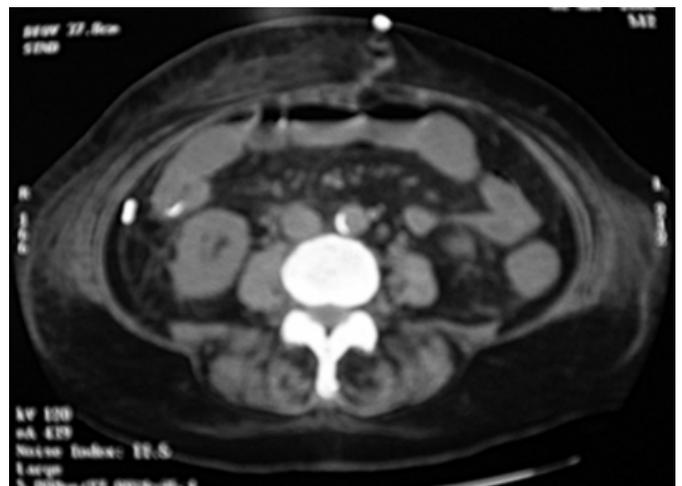
Figure 1: Photography showing the external aspect in the first days; an extensive cellulitis is appreciated around the drain site.



Laboratory data revealed a hemoglobin of 8.7 g/dL; and a hematocrit of 26.7%. The white blood cell count was 30,400/cc with 97% neutrophils. Serum bicarbonate was 118 mEq/L, glucose 112 mg/dL, sodium 129 mmol/L, and creatinine 2.1 mg/dL. Liver function studies were within normal limits and the urinalysis was unremarkable. The chest radiograph did not show alterations. Abdominal and pelvic CT showed diffuse infiltration in subcutaneous tissue of the right abdominal wall without signs of suture dehiscence (Fig. 2).

Figure 2

Figure 2: Abdominal CT showed diffuse infiltration in subcutaneous tissue of the right abdominal wall.



Diagnosis of abdominal wall cellulitis was made at the time of admission to the Intensive Care Unit and the patient was

Necrotizing Fasciitis of the Abdominal Wall Caused by Invasive Group D Enterococcus faecalis Secondary to Infected Intra-abdominal Drainage

started on intravenous (IV) fluids, broad spectrum antibiotics including imipenem/cilastina (3x3g daily dose) and linezolid (2x600 mg daily dose); non-invasive ventilation was also used intermittently and vasopressors were initiated. Two days after admission to the Intensive Care Unit, the cellulitis with necrosis had extended rapidly and affected the whole right abdominal wall. It had a circumferential area of ecchymosis and necrosis with macerated margins, and involved deeper structures of subcutaneous fat. A serosanguineous fluid drained out of the surgical wound. Incisions were made in the abdominal wall and a discharge of purulent material was obtained. The extensive necrotic tissue was debrided along the fascial planes (Fig. 3). Cultures of the wound taken at this time revealed an infection with group D Enterococcus faecalis in quantities $>10^6$ CFU/g of tissue, sensitive to ciprofloxacin, imipenem, linezolid, teicoplanin and vancomycin. Echerichia coli sensitive to aztreonam, cefepime, gentamicin, tobramycin and imipenem also grew in the infected wounds.

Figure 3

Figure 3: Status of the abdominal wall after extensive surgical debridement and drainage.



Three days after admission the patient developed septic shock with respiratory failure requiring intubation and mechanical ventilation support; necrotizing fasciitis was presumed and intravenous teicoplanin, meropenem and gentamicin were started; the management at this point also included dobutamine, paracetamol, metamizol, omeprazol, and enoxaparin. Hyperbaric oxygen therapy was not an option at our institution.

The histopathological examinations of surgically obtained biopsy specimens revealed suppurative fasciitis, widespread

myonecrosis, and thromboses of the vessels, all of which were compatible with a diagnosis of necrotizing fasciitis.

During her stay at the Intensive Care Unit, the patient required extensive daily debridements. Afterwards the wounds were allowed to heal by secondary intention (Figures 4,5).

Figure 4

Figure 4: Photography showing evolution of the lesions in a period of 3 week.



The following microorganisms were isolated during her stay at the Intensive Care Unit:

- Enterococcus faecalis in urine samples; sensitive to ampicilin, ciprofloxacin, linezolid, vancomycin, linezolid, imipenem.
- Coagulase negative Staphylococcus in central catheter tip cultures sensitive to linezolid and vancomycin.
- Klebsiella pneumoniae in blood culture sensitive to amoxicillin/CA, aztreonam, gentamicin, imipenem.
- Enterobacter cloacae in pharyngeal exudate sensitive to amikacin, aztreonam, ciprofloxacin, imipenem and piperacillin/tazobactam.

The patient required 20 days of mechanical ventilation support; tracheotomy was performed to facilitate weaning. During her stay at the ICU she also required hemodynamic support (dopamine, norepinephrine, dobutamine), treatment with diuretics, parenteral and enteral nutrition and blood transfusion.

Necrotizing Fasciitis of the Abdominal Wall Caused by Invasive Group D Enterococcus faecalis Secondary to Infected Intra-abdominal Drainage

The patient was discharged from the Intensive Care Unit 40 days after admission. She was discharged home 24 days later.

Figure 5

Figure 5: Photography showing evolution of the lesions in a period of 6 weeks. Local status of the wounds prior to discharge from the hospital.



DISCUSSION

In 1871, Joseph Jones¹⁴ was the first to describe necrotizing fasciitis (NF) when he referenced more than 2600 cases during the civil war. This lesion has also been called “hospital gangrene”, “necrotizing erysipelas”, “streptococcal gangrene”, and “suppurative fasciitis”. In 1952 the term NF was first used by Wilson¹⁵; this term is the most accurate to describe the disease.

The first clinical case of NF was described by Meleney in 1924; since then and despite the advent of sophisticated antibiotics and imaging modalities, the mortality rate of this serious infection has remained unchanged at approximately between 50-80%². The most important factor determining prognosis is early diagnosis and surgical debridement, with little reliance on medical therapy.

NF is a rare soft-tissue infection that affects people of a wide range of ages. The males are affected more than the females (53,2%). It is rapidly progressing, affects the cutaneous microcirculation, subcutaneous fascia and dermis, and characteristically spares the underlying muscle^{16,17}.

Necrotizing soft tissue infections can be divided into necrotizing cellulitis, necrotizing fasciitis and myonecrosis; these infections can be nonclostridial or clostridial¹³.

NF is mostly found in immunocompromised patients such as patients with diabetes mellitus, cancer, alcoholism, chronic liver disease, vascular insufficiency, organ transplantation, human immunodeficiency virus, malnutrition, and neutropenia and other immunocompromised patients¹⁸. In the case we describe, predisposing factors were the long hospital stay before surgery, chronic anaemia, multiple transfusions of blood, an immuno-compromised status, old age, chronic liver disease, colon neoplasia and taking nonsteroidal anti-inflammatory drugs.

The pathogenesis of necrotizing fasciitis is still not completely understood. Beginning at the original site, the infection spreads; initially subcutaneous fat and fascia are affected with little evidence of infection at the skin surface. Afterwards, nutrient vessels that perfuse the skin become thrombosed, causing ischemia, hypoxia and vascular cutaneous and subcutaneous necrosis. Drop in soft-tissue oxygen pressure allows the proliferation of anaerobe microorganisms. The localized necrosis of skin secondary to thrombosis of nutrient vessels as they pass through the zone of involved fascia can progress to frank cutaneous gangrene if the fasciitis is left untreated^{16,19}.

The onset of symptoms is usually several days after the initial insult. The patient may present marked leukocytosis, hemoconcentration and fulminant shock. The affected skin becomes warm, erythematous and edematous, so that the infection may be mistaken for cellulitis or erysipelas but patients fail to respond clinically despite 24 to 48 hours of appropriate antibiotics. The tense and shiny skin has no sharp demarcation and may develop a dusky decoloration with poorly defined borders. A central zone of necrosis may develop and it can eventually ulcerate. Anesthesia of the skin is caused from involvement of the cutaneous nerves as they pass through necrotic subcutaneous tissue. Soft tissue crepitance is common from gas formation. Intra-abdominal disease such as perforated colon or appendix should always be considered as the source of abdominal wall NF²⁰.

These patients often have a low-grade fever and can be anemic and jaundiced from bacterial hemolysis. Massive amounts of fluid can be sequestered with resultant hyponatremia, hypoproteinemia, and dehydration. Hypocalcemia can develop from necrosis of subcutaneous fat and subsequent saponification. Important complications of NF are acute renal insufficiency with a frequency of 31.6% and mortality of 50%, acute respiratory distress

Necrotizing Fasciitis of the Abdominal Wall Caused by Invasive Group D Enterococcus faecalis Secondary to Infected Intra-abdominal Drainage

syndrome (ARDS) with a frequency of 29% and mortality of 59% and multiorgan failure (MOF) with a frequency of 21% and mortality of 77.5%²¹.

The causative organism or organisms in necrotizing fasciitis vary with the site and cause of infection. It is important to identify it and to test for antimicrobial susceptibility to correctly choose antibiotic treatment.

Monomicrobial infections account for 18.6% and polymicrobial for more than 80% of cases of NF²²; most commonly involved microorganisms are bacteria of the gastrointestinal tract and perineum area like group A and B *Streptococcus pyogenes*^{23,24}, group D *Enterococcus*, *Echerichia coli*¹, *Proteus* spp¹, anaerobic bacteria (*Clostridium perfringens*)²⁵, *Pseudomonas aeruginosa*²⁶, *Klebsiella*²⁷, *Salmonella* group C²⁸ or *Staphylococcus aureus*²⁹.

A prolonged hospital stay before surgery or preoperative antibiotic treatment has been associated with the emergence of multidrug-resistant microorganisms such as *Pseudomonas aeruginosa*, *Enterobacter* spp, and *Proteus* spp, Methicillin-resistant *Staphylococcus aureus* and *Candida* spp³⁰.

The nature of the disease is exemplified by the patient in our case report. The onset of symptoms was several days after the surgery. The beginning of the disease was misleading due to the sequence in which tissues were affected by bacteria during necrotizing fasciitis. On admission at the ICU, although she had extensive subcutaneous damage, her skin was only mildly erythematous. During the first two days at the ICU, the correct diagnosis was not made due to the fact that the clinical presentation was masked by cellulitis of the abdominal wall.

The clinical symptoms and signs of sepsis and the lack of response to antibiotics were important for differential diagnosis.

In this case, the infection was probably initiated at the point of insertion of the surgical drainage, as the wound culture at the insertion site was initially positive for invasive group D *Enterococcus cloacae*. The infection possibly was secondary to drainage contamination during colon surgery. Later, wound cultures were positive for *E. coli* and *Pseudomonas aeruginosa*.

In elderly patients, as our patient, the symptoms usually are not specific; therefore, the diagnosis in these patients is more

difficult.

Although the diagnosis of NF is often made clinically, emergency computed tomography (CT) can lead to early diagnosis with accurate assessment of disease, help evaluate the structures that can become involved. CT is invaluable for detecting deep complications of cellulitis and pinpointing the anatomic compartment that is involved by an infection. CT provides a higher specificity for the diagnosis of NF than the conventional radiology and ultrasonography. CT helps guide therapy toward emergency surgical debridement in cases of necrotizing fasciitis and toward percutaneous drainage in cases of abscess formation^{31,32}.

Magnetic resonance imaging (MRI), can be useful for differentiating necrotizing fasciitis from non-necrotizing soft-tissue infection and for planning the treatment of necrotizing fasciitis³³.

The importance of early and aggressive management of NF must be emphasized. Once diagnosis is made, immediate surgical evaluation and repeated debridements are usually required to make sure all the necrotic tissue has been removed. Once blood culture and wound culture have been obtained, broad spectrum antibiotics should be started to avoid the spread of infection. A daily wound exam and dressing change must be performed. Skin grafts should be considered to cover the wound only if it is definite that the infection has been eradicated^{34,35,36,37,38}.

In patients with NF, it is also important to begin and maintain resuscitation and supportive therapy with fluids. Other adjunctive wound therapies such as hyperbaric oxygen therapy have been proposed for improving the outcome. The proposed mechanisms of this therapy are the bactericidal effect on anaerobes and improved tissue oxygenation³⁹.

In our patient, repeated debridements were carried out, and these may have been the keys to success in avoiding widespread infection. Aggressive management including resuscitation with fluids and pressors (Norepinephrine, Dopamine and Dobutamine), immediate surgical evaluation, and intravenous antibiotics was also performed. Other important adjuncts in the treatment of our patient were non-invasive and invasive ventilation, enteral and parenteral nutrition and broad-spectrum antibiotics. Hyperbaric oxygen therapy was not possible at our institution.

Probably, the main responsible factor of the elevated mortality in elderly patients is the delay in diagnosis and

treatment; because in these patients the clinical manifestations are usually atypical, therefore NF is not suspected.

This report serves to alert surgeons and their team of the importance of the careful management of the instruments and of the systems of surgical drainage, mainly in gastrointestinal surgery; because an inappropriate management may result in extensive tissue destruction and require extensive surgery.

In conclusion, NF is a potential complication of inserting a postoperative drainage after abdominal surgery. Because of the poor prognosis and the high mortality, aggressive surgical debridement and appropriate antibiotics are the cornerstones of effective therapy.

CORRESPONDENCE TO

Josué Carvajal Balaguera Calle Téllez, 30, Escalera 12, 2ª planta, Puerta 3. 28007 Madrid Spain Tel. +34915518018 Fax Hospital. +9134915345330 E-mail: josuecarvajal@yahoo.es

References

1. Eke N. Fournier's gangrene: a review of 1726 cases. *Br J Surg* 2000; 87: 718-728.
2. Comin Novella L, del Val Gil JM, Oset García M. Gangrena de Fournier: presentación de 6 casos sin mortalidad. *Cir Esp* 2008; 84: 28-31.
3. Levine EG, Manders SM. Life-threatening necrotizing fasciitis. *Clin Dermatol* 2005; 23: 144-47.
4. Karim MS. Fournier gangrene following urethral necrosis by indwelling catheter. *Urology* 1984; 23: 173-5.
5. López JC, Sánchez JM, Piñeiro MC, Bouso M, Parra L, García J. Gangrena de Fournier secundaria a cateterismo uretral. *Arch Esp Urol* 2005; 58: 15-20.
6. Conn IG, Lewi HJE. Fournier's gangrene of the scrotum following traumatic urethral catheterization. *J R Coll Surg Edinb* 1987; 32: 182.
7. Calderon E, Carter E, Ramsey KM, Vande JA, Green WK, Alpert MA. Necrotizing fasciitis: a complication of percutaneous revascularization. *Angiology* 2007; 58: 360-6.
8. Nouira K, Bourkhis S, Maalej S, Ben J, Azaiez O, Ben M, Rahal K, Menif E. Necrotizing fasciitis complicating transthoracic biopsy. *Rev Pneumol Clin* 2007; 63: 273-6.
9. Gonzalez I, Marin D, Palao R, Barret JP. Necrotizing fasciitis after liposuction. *Acta Chir Plast* 2007; 49: 99-102.
10. Orlando A, Marrone C, Nicoli N, Tamburello G, Rizzo A, Paglioro L, Cottone M. Fatal necrotizing fasciitis associated with intramuscular injection of nonsteroidal anti-inflammatory uncomplexed endoscopic polypectomy. *J Infect* 2007; 54: 145-8.
11. Tan LG, See JY, Wong KS. Necrotizing fasciitis after laparoscopic colonic surgery: Case report and review of literature. *Surg Laparosc Endosc Percutan Tech* 2007; 17: 551-3.
12. Kapadia S, Polenakovik H. Cutaneous zygomycosis following attempted radial artery. *Skinmed* 2004; 3: 336-8.
13. Huljev D, Kucisec-Tepes N. Necrotizing fasciitis of the abdominal wall as a post-surgical complication: a case report. *Wounds* 2005; 17: 169-177.
14. Jones J. Investigation upon the nature, causes and treatment of hospital gangrene as it prevailed in the confederative armies. In: Hamilton FH, ed. *United States Sanitary Commission, Memoires: Surgical II*. New York, NY: Riverside Press; 1871: 146-170.
15. Wilson B. Necrotizing fasciitis. *Am Surg* 1952; 18: 416-31.
16. Verit A, Verit F. Fournier's gangrene: the development of a classic pathology. *BJU* 2007; 100: 1218-20.
17. Urdaneta E, Méndez A, Urdaneta A. Fournier's gangrene. Current perspectives. *An Med Interna* 2007; 24: 190-4.
18. Yanar H, Taviloglu K, Ertekin C, Guloglu R, Zorba U, Cabioglu N. Fournier's Gangrena risk factors and strategies for management. *World J Surg* 2006; 30: 1750-4.
19. Thaichinda S, Kositpantawong N. Necrotizing skin soft-tissue infections associated to septicemia: 7 cases report and review. *J Med Assoc Thai* 2008; 91: 117-23.
20. Yenyol CO, Suelozgen T, Arslan M, Ayder AR. Fournier's gangrene: experience with 25 patients and use of Fournier's gangrene severity index score. *Urology* 2004; 64: 218-222.
21. Golger A, Ching S, Goldsmith CH, Pennie RA, Bain JR. Mortality in patients with necrotizing fasciitis. *Plast Reconstr Surg* 2007; 119: 1803-7.
22. Legbo JN, Legbo JF. Bacterial isolates from necrotizing fasciitis: a clinical and pathological perspective. *Niger J Med* 2007; 16: 143-7.
23. Mehta S, McGerr A, Low DE, Hallet D, Bowman DJ, Grossman SL, Stewart TE. Morbidity and mortality of patients with invasive group A streptococcal infections admitted to the ICU. *Chest* 2006; 130: 1679-86.
24. Sendi P, Johansson L, Norrby A. Invasive group Streptococcal disease in pregnant adults: a review with emphasis on skin and soft-tissue infections. *Infection* 2008; 36: 100-11.
25. Dylewski J, Drummond R, Rowen J. A case of clostridium septicum spontaneous gas gangrene. *CJEM* 2007; 9: 133-5.
26. Akamine M, Miyagi K, Uchilara T, Azuma M, Tara S, Higa F, Haranaga S, Tate A. Necrotizing fasciitis caused by pseudomonas aeruginosa. *Intern Med* 2008; 47: 553-6.
27. Oishi H, Kagawa Y, Mitsumizo S, Tashiro Y, Kobayashi G, Udo K, Aoki S, Takaya Z, Araki K. A fatal case of necrotizing fasciitis due to bacteria translocation of *Klebsiella oxytoca*. *J Infect Chemother* 2008; 14: 62-5.
28. Khawcharoenporn T, Apisarthanarak A, Kiratisin P, Mundy LM. *Salmonella* group C necrotizing fasciitis: a case report and review of the literature. *Diagn Microbiol Infect Dis* 2006; 54: 319-22.
29. Miller LG, Perdreau-Remington F, Rieg G, Mehdi S, Perlroth J, Bayer AS. Necrotizing fasciitis caused by community associated Methicillin-resistant *Staphylococcus aureus* in Los Angeles. *N Eng J Med* 2005; 14: 1445-1453.
30. Tellado JM, Stiges-Serra A, Barcenilla F, Palomar R, Serrano R, Barberán J, Moya M, Martínez M, García JA, Mensa J, Prieto J. Pautas de tratamiento antibiótico empírico de las infecciones intraabdominales. *Rev Esp Quimioterap* 2005; 18: 179-186.
31. Levenson RB, Singh AK, Novelline RA. Fournier gangrene: role of imaging. *Radiographics* 2008; 28: 519-28.
32. Fayad LM, Carrino JA, Fishman EK. Musculoskeletal infection: role of CT in the emergency department. *Radiographics* 2007; 27: 1723-36.
33. Sato T, Hagiwara K, Matsumo H, Chiyokura Y,

Necrotizing Fasciitis of the Abdominal Wall Caused by Invasive Group D Enterococcus faecalis Secondary to Infected Intra-abdominal Drainage

Morimoto S, Kunogi J, Akiyama O. A case of necrotizing fasciitis caused by coagulase-negative staphylococcus: utility of magnetic resonance imaging for the preoperative diagnosis of necrotizing fasciitis. *J Infect Chemother* 2005; 11: 160-3.

34. Carter PS, Banwell PE. Necrotising fasciitis: a new management algorithm based on clinical classification. *Int Wound J* 2004; 1: 189-198.

35. Ryszel H, Germann G, Riedel K, Köllensperger E. Surgical concept and result of necrotizing fasciitis. *Chirurg* 2007; 78: 1123-9.

36. Fajdic J, Bukovic D, Hrgovic Z, Habek M, Gugic D, Jonas D, Fassbender WJ. Management of Fournier's gangrene. Report of 7 cases and review of the literature. *Eur J Med Res* 2007; 12: 169-72.

37. Bosoglu M, Ozbey I, Atamanalap SS, Yildirgan MI, Aydinli B, Palat O, Oztuek G, Oren D. Management of Fournier's gangrene: review of 45 cases. *Surg Today* 2007; 37: 558-63.

38. Anwar MU, Haque AK, Rahman J, Morris R, McDermott J. Early radical surgery and antimicrobial therapy with oxygen in necrotizing fasciitis. *Plas Reconstr Surg* 2008; 121: 360-1.

39. Escobar SJ, Slade JB Jr, Hunt TK, Cianci P. Adjuvant hyperbaric oxygen therapy (HBO2) for treatment of necrotizing fasciitis reduces mortality and morbidity. *Undersea Hyperb Med* 2005; 32: 437-43.

40. Arenal JJ, Bengoechea M. Mortality associated with emergency abdominal surgery in elderly. *Can J Surg* 2003; 46: 111-6.

***Necrotizing Fasciitis of the Abdominal Wall Caused by Invasive Group D Enterococcus faecalis
Secondary to Infected Intra-abdominal Drainage***

Author Information

J. Carvajal Balaguera

Surgeon Assistant, Service of General and Digestive Surgery, Hospital Central de la Cruz Roja San José y Santa Adela

J. Archilla Estevan

Physician Assistant, Anesthesiology Service, Hospital Central de la Cruz Roja San José y Santa Adela

A. Smaranda

Physician Assistant, Anesthesiology Service, Hospital Central de la Cruz Roja San José y Santa Adela

J. Camuñas Segovia

Surgeon Assistant, Service of General and Digestive Surgery, Hospital Central de la Cruz Roja San José y Santa Adela

S. Martín Alcrudo

Physician Assistant, Anesthesiology Service, Hospital Central de la Cruz Roja San José y Santa Adela

CMA Cerquella Hernández

Chief, Service of General and Digestive Surgery, Hospital Central de la Cruz Roja San José y Santa Adela