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A Case Report on Left Gluteal Abscess with Necrotizing Fasciitis



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ABSTRACT

Necrotizing fasciitis (NF) is a rare, rapidly spreading infection that primarily affects deep fascia and can cause secondary necrosis of subcutaneous tissue, fascia, and muscle. The incidence of invasive *Streptococcus pyogenes* has recently increased significantly. Timely diagnosis and radical debridement are essential to prevent sepsis, multiple organ failure, and possible death. Once NF is diagnosed, the next steps include early removal of non-viable tissues and broad-spectrum treatment with intravenous antibiotics. This case describes an atypical case of NF of a gluteal abscess (L) with necrotizing fasciitis.



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INTRODUCTION

Necrotizing Fasciitis (NF) is a deadly soft-tissue infection that mostly affects the subcutaneous soft tissues of the fascial layer, causing progressive purulent necrosis of the skin and subcutaneous fascial tissues.^[1] NF is a rare but potentially fatal infection of the skin, soft tissues, and muscles that tends to spread quickly across the fascia planes, slowly destroying the fascia at a rate of up to 2-3 cm/h. The rapid clinical course of Fournier's gangrene, which can appear in the lower or higher extremities, perineum, vaginal region, and abdominal wall, is associated with polymicrobial infection and synergy, which typically coexist.^[2,3] There are four different forms of necrotizing fasciitis. Most cases (70–90%) fall under Type 1, which is characterized as polymicrobial NF. Gram-positive cocci, gram-negative rods, and anaerobes are frequently identified in patients with diabetes mellitus or peripheral vascular disease who are at risk for developing NF affecting the abdominal wall, trunk, and perianal regions. When there are no coexisting conditions, type 2, also known as monomicrobial, is typically brought on by community-acquired methicillin-resistant *Staphylococcus aureus* and group A *Streptococcus*. Type 3 is brought on by gram-negative bacteria such as *Clostridium* species and *Vibrio* species and is associated with minor wounds from a fish sting, infections from shellfish, or exposure to sea water. Finally, type 4 frequently occurs from a fungus infection, particularly in those with immunological problems, usually following trauma.^[4] In most cases, anaerobic bacteria produce gas that builds up in the gaps between soft tissues, creating the typical appearance of gas gangrene on normal X-rays and computed tomography (CT) scans.^[5] It is essential to diagnose NF quickly. Given that delays are linked to more complicated surgeries, higher rates of amputation, and higher fatality rates, even the smallest delay could prove catastrophic.^[6]

One of the most frequent causes of severe community-associated (also known as community-acquired) skin and soft tissue infections is *Staphylococcus aureus*, an omnipresent bacterium.^[7-10] *S. aureus* strains from infections in the community were frequently sensitive to penicillinase-resistant b-lactam antibiotics, such as methicillin and oxacillin, until recently. Antibiotics against MRSA are advised as part of empirical therapy for severe community-associated *S. aureus* infections. Group A streptococcus, *Clostridium perfringens*, or a combination of aerobic and anaerobic organisms, generally containing group A streptococcus, the Enterobacteriaceae, anaerobes, and *S. aureus*, typically cause these illnesses.^[11-15] Major clinical reviews on the subject as well as published microbiologic

research on the condition have not identified *S. aureus* as the only cause of Necrotizing Fasciitis.^[11,13-16] Because necrotizing fasciitis is typically polymicrobial in nature, the majority of authorities advise using broad-spectrum empirical antibiotic therapy for suspected cases. Vancomycin therapy, however, is not advised in the current standard requirements, potentially due to the rarity of this infection as a cause of necrotizing fasciitis.^[16-18]

Case Presentation

A 40-year-old female patient was brought to the emergency department with complaints of loss of consciousness and fever. She has a history of (L) Gluteal abscess status post – Incision and Drainage (S/P-I & D) from a local hospital and known medical history of Type 2 Diabetes Mellitus and was on Insulin. On arrival, the pulse rate, Blood Pressure (BP), and SPO₂ of the patient was noted as 114 beats/ minute, 90/60 mmHg, and 94% respectively. On systemic examination, the parameters of the cardiovascular system are found to be normal. The Glasgow coma scale (GCS) score is E₄V₂M₄. On examination, the peripheries were found to be cold and there is discoloration of skin on the (L) Gluteal region with pus draining from the I & D site.

Then She was admitted to Multidisciplinary Intensive Care Unit (MDICU), BP was controlled with dual inotropes, and Invasive ventilation, and insulin infusion were started. Emergency general surgery was consultation sought. Emergency wound debridement was done as a life saving measure in view of possible sepsis source. Procedure was done under general anesthesia and patient was weaned from dual inotropes and planned for further debridement.



Figure 1 shows (L) Gluteal Abscess with Necrotizing Fasciitis

Her initial laboratory data showed declined Haemoglobin (8.4 gm/dL), Serum Globulin (3.0 gm/dL), Serum Albumin (2.6 gm/dL), Serum Potassium (3.1 mmol/L), Serum Calcium (7.5 mg/dL). The elevated parameters include Total Count (34220 cells/cumm), Serum Creatinine (1.3 mg/dL), CRP (253.90 mg/L), Urea (45 mg/dL), ESR (120 mm/hr), HBA1C (15.6 %), INR (1.56), Ketone (6.6 mg/dL), Serum Amylase (161 U/L), Serum Lipase (731 IU/L), Serum LDH (276 U/L). Plain CT Brain shows Acute pontine hemorrhage in mid pons and hyperdense contents within right globe. Pus C&S report shows Staphylococcus aureus growth in culture. She was diagnosed with (L) Gluteal Abscess with Necrotizing Fasciitis, Mild pancreatitis, Sepsis related Trivial Pontine Hemorrhage, Septic shock, Diabetic Keto Acidosis and Type 2 Diabetes Mellitus.

Followed by emergency wound debridement, Post debridement was done. She was weaned from invasive ventilation, removed Ryle's tube, and started on orals and diet advanced as she improved. She was also managed with IV Meropenem 1gm Q8H and IV Teicoplanin 600mg Q12H. Pus culture showed MRSA and hence after Infection diseases (ID) consultation, decided to continue Teicoplanin for 14 days. She underwent daily dressing. As wound improved, she was discharged with an advice to follow up daily for dressings.

Advice on discharge include Tab. Linezolid 600mg twice daily for 7 days, Tab. Rabeprazole 20mg twice daily for 7 days, Tab. Paracetamol 650mg twice daily for 7 days, Inj. Isophane insulin + Soluble Insulin 50/50 thrice daily to continue and Tab. Repaglinide 0.5mg and Voglibose 0.3mg combination twice daily to continue for Type 2 Diabetes Mellitus.

DISCUSSION

NF is a rare infection of the superficial fascia and subcutaneous tissues that has a death rate of 30 to 60%, a fulminant course, and systemic toxicity. Early diagnosis and therapy are crucial to the prognosis of NF.^[19] According to this research, NF brought on by MRSA in the community has the potential to develop quickly and become clinically identical to necrotizing fasciitis brought on by infections like group A streptococcus. Since community-associated necrotizing fasciitis caused by MRSA is a new clinical syndrome, empirical therapy for this condition should include medications that are consistently active against the local MRSA strain. Included medicines with potent efficacy against this disease represent an important development in the empirical management of the infection.^[16,18] Surgical debridement of the afflicted tissues is the first line treatment for NF. Debridement and the rebuilding of soft

tissues may need surgery. A life-saving requirement is adequate surgical debridement. Debridement aims to eliminate all necrotic tissues, halt the transmission of infection, and minimize systemic toxicity. Debridement should be carried out again as necessary. Because thrombosis of the veins precludes sufficient penetration of antibiotics into the tissue, they are only used as a supplement to surgical debridement. Surgery is used to protect the healthy skin and achieve haemostasis by removing all necrotic tissues, including muscle, fascia, and skin. Considering the microbiological classification, treatment for NF comprises the intravenous infusion of broad-spectrum empirical antibiotics. Due to the purulent secretion and wound phlogistic symptoms, vancomycin in combination with a carbapenem, in this case "Meropenem", was the empirically chosen course of treatment. They were continued after culture results. The patient's survival depends on proper nutritional assistance and post-operative wound care. The traditional dressing has an enzymatic debriding effect when used in conjunction with surgical debridement.^[20] Treatment for NF is characterized by intensive surgical and medical therapy, which may include managing septic shock and giving intravenous fluids.^[21] For mixed aerobic and anaerobic bacterial infections, antibiotic therapy is necessary. Cefoxitin, clindamycin, imipenem, and mixtures of a b-lactamase inhibitor (i.e., clavulanic acid) and a penicillin (i.e., ticarcillin) are antimicrobials that usually target *S. aureus* as well as anaerobic microbes.^[22]

CONCLUSION

NF is a rare yet devastating condition due to its high mortality rate. Numerous conditions, including diabetes mellitus, immunosuppression, and liver cirrhosis, are linked to this pathology and may speed up the development of necrosis. If the condition remains untreated, septic shock will certainly happen, and a delay in diagnosis could be fatal. Clinical awareness, early diagnosis, prompt surgical debridement, prompt wound debridement, and intensive supportive care are all ways to reduce the mortality and morbidity linked to NF. Antibiotic therapy is the first step in the management of the infection. It is important to properly nourish the patient and proper management of the surgical incision after surgery. Septic shock management and intravenous fluid administration are also included in treatment.

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Informed Consent

Before taking this case the patient and their families were informed and informed consent was acquired.

Conflicts of Interest

The authors have no conflicts of interest to declare.

REFERENCES

1. Sarani B, Strong M, Pascual J, Schwab CW. Necrotizing fasciitis: current concepts and review of the literature. *J Am Coll Surg*. 2009;208(2):279-288.
2. Levine EG, Menders SM. Life-threatening necrotizing fasciitis. *Clin Dermatol* (2005) 23:144–7.
3. Urschel JD, Takita H, Antkowiak JG. Necrotizing soft tissue infections of the chest wall. *Ann Thorac Surg* (1997) 64:276–9.
4. Liu G, Zhang X, Fu W, Zhao L, Zhang C. Early treatment of necrotizing fasciitis secondary to perianal abscess. *Eur Surg*. 2012;44(2):120-123.
5. Kaafarani HM, King DR. Necrotizing skin and soft tissue infections. *Surg Clin North Am* (2014) 94:155–63.
6. Descamps V, Aitken J, Lee MG. Hippocrates on necrotising fasciitis. *Lancet* (1994) 344:556.
7. Sigurdsson AF, Gudmundsson S. The etiology of bacterial cellulitis as determined by fine-needle aspiration. *Scand J Infect Dis* 1989; 21:537-42.
8. Diekema DJ, Pfaller MA, Schmitz FJ, et al. Survey of infections due to *Staphylococcus* species: frequency of occurrence and antimicrobial susceptibility of isolates collected in the United States, Canada, Latin America, Europe, and the Western Pacific region for the SENTRY Antimicrobial Surveillance Program, 1997-1999. *Clin Infect Dis* 2001;32 Suppl 2:S114-S132.
9. Carratala J, Roson B, Fernandez-Sabe N, et al. Factors associated with complications and mortality in adult patients hospitalized for infectious cellulitis. *Eur J Clin Microbiol Infect Dis* 2003; 22:151-7.
10. Brook I, Frazier EH. Clinical features and aerobic and anaerobic microbiological characteristics of cellulitis. *Arch Surg* 1995; 130:786-92.
11. Wong CH, Chang HC, Pasupathy S, Khin LW, Tan JL, Low CO. Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality. *J Bone Joint Surg Am* 2003; 85:1454-60.
12. Bisno AL, Stevens DL. Streptococcal infections of skin and soft tissues. *N Engl J Med* 1996; 334:240-5.
13. Elliott D, Kufera JA, Myers RA. The microbiology of necrotizing soft tissue infections. *Am J Surg* 2000; 179:361-6.
14. Fontes RA Jr, Ogilvie CM, Miclau T. Necrotizing soft-tissue infections. *J Am Acad Orthop Surg* 2000; 8:151-8.
15. Brook I, Frazier EH. Clinical and microbiological features of necrotizing fasciitis. *J Clin Microbiol* 1995; 33:2382-7.
16. Swartz MN. Cellulitis and subcutaneous tissue infections. In: Mandell GL, Bennett JE, Dolin R, eds. *Mandell, Douglas, and Bennett's principles and practice of infectious diseases*. 5th ed. Philadelphia: Churchill Livingstone, 2000:1037-57.
17. Headley AJ. Necrotizing soft tissue infections: a primary care review. *Am Fam Physician* 2003; 68:323-8.
18. Gilbert DN, Moellering RC, Eliopoulos GM, Sande MA. *The Sanford guide to antimicrobial therapy*. Hyde Park, Vt.: Antimicrobial Therapy, 2004.
19. Sudavsky, L. A., J. C. Lashinger, G. F. Coppa, and F. C. Spencer. 1987. Improved results from a standardized approach in treating patients with necrotizing fasciitis. *Ann. Surg.* 206:661–665.

20. Zhao JC, Zhang BR, Shi K, et al. Necrotizing soft tissue infection: clinical characteristics and outcomes at a reconstructive center in Jilin Province. *BMC Infect Dis* 2017; 17:792.
21. Brook, I. 1989. Pediatric anaerobic infection: diagnosis and management. The C. V. Mosby Company, St. Louis.
22. Finegold, S. M. 1977. Anaerobic bacteria in human disease. Academic Press, Inc., Orlando, Fla.

