# Successful Management of Necrotizing Fasciitis of Perianal Region due to Kobiraji Maltreatment for Hemorrhoids

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#### Abstract

Necrotizing fasciitis (NF), an invasive soft-tissue infection that affects the fascia superficially and spreads quickly along subcutaneous tissue. It is a medical-surgical emergency. The patient may have symptoms that are not consistent with the clinical findings, such as a temperature, evidence of local inflammation, and acute pain. Patients typically present at a later stage, but because there are few cutaneous signs in the early stages, diagnosis might be difficult. Thus, a high index of suspicion plays a crucial role in the NF diagnosis. Furthermore, intraoperative diagnosis frequently serves as a confirmation. The cornerstones of the treatment include analgesia, hydration and electrolyte control, intravenous antibiotics and prompt surgical debridement.

**Keywords:** Necrotizing Fasciitis, Perianal Region, Kobiraji Maltreatment, Hemorrhoids.

#### Introduction

A rare but potentially fatal soft tissue infection, necrotizing fasciitis is characterised by quickly spreading inflammation and necrosis of the skin, subcutaneous fat and fascia. Local and systemic signs and symptoms are correlated to diagnose a necrotizing illness. NF is characterised by nonspecific symptoms such as tenderness, warmth, erythema, swelling, pain, and necrosis in a tissue area. The patient's life can only be preserved by an expedient diagnosis and vigorous surgical intervention. We reported a case of perineal necrotizing fasciitis after Kobiraji treatment. The case showed significant tissue necrosis that required immediate surgical intervention, and the patient was effectively managed with the best possible outcome.

# **Case Summary**

A male patient, age 21, was brought to the hospital with complaints of perianal secretion and pain which had been accentuated over the previous three days. He had previously used indigenous medicinal plants for self-medication in the perineal area to alleviate haemorrhoids. He had tachycardia and was very worried upon admission. He was not feverish.



Figure-1: Initial presenting features-Necrozed perianal area



Figure-2: After extensive surgical debridement of perineal area



Figure-3: Deviational Colostomy

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Figure-4: Healthy granulated wound on 14th POD



**Figure-5(a):** Reconstruction of perineal area by rotation flap.



Figure-5(b): Reconstruction of perineal area by rotation flap.

Local physical examination revealed discoloration, swelling of whole perineal region and on palpation temperature was found raised but there was no tenderness. Within 24 hours of admission the area of cutaneous necrosis extended from 50 cm<sup>2</sup> to 500cm<sup>2</sup> (Figure-1). Laboratory investigation findings of hematological and biochemical investigations C-reactive protein (CRP) was very high (357mg/ml), CPK Level was 1698 U/L but blood culture yield no bacterial growth. Signs and symptoms of systemic toxicity revealed tachycardia (>90min) and tachypnea (>20min). Immediately after admission the patient received intravenous 3rd generation cephalosporin-ceftriaxone (2gm) and Metronidazol antibiotic (500) tds for extended antimicrobial coverage. Despite of antibiotherapy swelling, pain and necrosis expanded rapidly. So, on the basis of a high clinical suspicion the case was diagnosed as a case of necrotizing necrotizing fasciitis (NF). And urgent decision was made for surgical debridement of the wound. So, he was counseled about the gravity of this condition, need of extensive operation and the risk of increased mortality if surgical debridement is not performed. On the next day of admission, patient underwent extensive surgical debridement with removal of all devitalized tissue, muscle, nerve, necrosectomy, fasciotomy of the perineal region with proper toileting (Figure-2). Left deviational colostomy was established in the patient to control infection and to enhance perineal healing (Figure-3).

Intraoperative findings revealed frankly gangrenous, gray necrotic tissue, lack of bleeding, thrombosed vessels, pus, non-contracting muscle with extensive sloughing. A positive finger text was the most significant finding, which was characterized by lack of resistance to finger dissection in normally adherent tissue. Eventually, anatomopathogy revealed a necrotizing fasciitis and ischemic muscular necrosis.

Antimicrobial therapy was chosen as per culture and sensitivity results. Extended coverage with intravenous antibiotic was started with Ceftriaxon and Metronidazol. To obtain optimal care adequate metabolic, nutritional, hemodynamic support and intravenous ketorolac for analgesia were the mainstays of support for patient.

Wound left open after debridement and sterile dressing with iodine gauze was performed. Surgical wound was reevaluated in frequent basis to see the progress of wound healing and any extension of infections (Figure-4). Daily wound care was maintained until the appearance of healthy granulation tissue. After 14 days, wound coverage done by reconstruction of perianal area repaired by rotation flap (Figure-5-a,b). After 3 months reversal of colostomy was performed. Patient was observed for 7 days after reversal of colostomy.

### **Discussion**

Wilson coined the name "Necrotizing fasciitis" in 1952 to describe the salient aspects of the infectious process. With an incidence rate of 4 to 13/1000000 per year, it is a rare disorder. Necrotizing fasciitis (NF) is a severe soft tissue illness that progresses quickly, mostly due to polymicrobial infections. The infection enters the body via the subcutaneous layer, travels along the fascial planes, and eventually destroys tissue, with a 3cm/h pace of tissue destruction reaching the fascia. It might be difficult to identify NF and distinguish it from cellulitis at the time of admission. Only 15% to 34% of patients with NF get a correct admission diagnosis, according to research. Additionally, a 30% adult death rate was discovered. At first, the patient could merely have a fever and crescendo pain, which is sudden, intense pain that has to be treated



with ketorolac or another medication. Anorexia, diarrhoea, myalgia, and malaise may also manifest in the first 24 hours. Because there are no cutaneous symptoms at first, the infection is often misdiagnosed or the right diagnosis is not detected until later. 7,8

Type 1 or type 2 necrotizing fasciitis is classified according to the presence of an organism. 9,10 The majority of NF cases (80-90%) are type 1 which includes facultative and anaerobes as well as polymicrobial infections including non-group A streptococci. The most frequent causes of this kind of NF are local infections, wounds and vascular lesion surgery. Edoema, erythema, bullae, necrotic and ulcerated lesion are examples of cutaneous symptoms. Its progressive character is mild. Muscular infection and deep fascia involvement may or may not be evident.10 Type 1 often includes fasciitis of the perineum and abdomen. The two species that are most often implicated are enterococci and Gram-aerobic enteric bacilli. The Group A streptococcus infection known as Type 2 NF is monomicrobial. Leg NF is often type 2.11 This kind of NF progresses quickly (1-3 days), presenting with edoema, erythema and necrotic bullae. The NF is caused by trauma, surgery, burns, skin lesions, erysipelas and varicella.10

Although NF may affect any part of the body, it most often affects the perineum, extremities and abdominal wall. The following conditions may have a significant impact on the etiologic aspect: hemorrhoidal banding rectal cancer, pilonidal abscess, vulvar abscess, episiotomy, coital injury, salpingogectomy, prostatic surgery, genitourinary infection and bartholonitis. <sup>12,13</sup> Hematogenous dissemination from a distant location, blunt or penetrating trauma, and direct inoculation of the subcutaneous tissue from a superficial site are risk factors of NF that lead to the spread of pathogens in the subcutaneous tissue.

Because there are few cutaneous findings in NF, early diagnosis is very difficult. Patients with NF may first exhibit sepsis symptoms (such as fever, tachycardia, altered mental state, or diabetic ketoacidosis) on their own or in conjunction with signs of skin inflammation. Since NF first develops in the deep tissue planes, there may not be much epidermal involvement at first, making the diagnosis challenging to distinguish from cellulitis and non-necrotizing skin infections. Systemically toxic symptoms might include tachycardia, fever (temperature over 38°C), altered mental state, or even diabetic ketoacidosis. A thorough inspection of everybody area is necessary to investigate any kind of skin infection. This has to be guaranteed for septic patients when the source is not immediately apparent since the mouth cavity and perineum are often disregarded. <sup>16</sup>

The usual presentation of necrotizing fasciitis is a patchy skin darkening accompanied by pain and edoema. Yet when the illness worsens, vesicles, bullae, necrosis, crepitus, and a grayish-brown discharge appear. Crepitus and hemorrhagic bullae are warning indicators of NF. Although it is a delayed symptom, only around 18% of NF sufferers have crepitus. 17,18 Crepitus and blistering have been identified by several studies as the most distinctive indicators of necrotizing soft tissue infection at the time of first presentation. Another NF indication related to a deep-seated infection is localised discomfort. On the other hand, NF patients often endure excruciating discomfort, and they may become quite terrified while having an examination. Nonetheless, a patient with diabetic neuropathy may experience discomfort and lack of feeling, which might result in an incorrect diagnosis. This is particularly common in areas where infections are hidden, including the mouth cavity or perineum. 18,20 The key to diagnosing systemic poisoning is to recognise its signs and symptoms. Early observations include tachypnea (>20min), tachycardia (>90min), and high temperature. Severe septic syndrome, characterised by a reduction in arterial pressure (less than 90 mmHg or less than 40 mmHg below the baseline pressure), occurs as the infection worsens. Renal impairment is indicated by elevated creatinine and oligouria (<30 ml/h).<sup>21</sup>

The diagnosis of NF is difficult; only a strong index of suspicion is required. Early surgical exploration is required if there is no resistance to dissection of typically adhering fascia in order to confirm the diagnosis. Normal laboratory tests, such as those measuring C-reactive protein and white blood cell count, may help differentiate non-necrotic soft tissue infection from necrotizing fasciitis. CPK concentration is a helpful indicator of muscle necrosis as well.<sup>22</sup>

Per-operative culture with antibiogram is helpful for proper antibiotic therapy. A biopsy is a useful substitute for traditional diagnostic techniques. Even while radiologic investigations are helpful, surgical treatment should never be postponed in instances that are really suspect. Additionally, foreign bodies, localised fluid collection abscesses, and subcutaneous and fascial thickening are all detected by CT scanning. Surgery, which involves the immediate debridement of all necrotic tissues and the thorough fasciotomy draining of the affected fascia planes, is still the first-choice therapy for NF. Page 1979.

Since the death rate sharply rises after 24 hours after presentation, surgery should be done as soon as possible. Reevaluating surgical wounds on a regular basis is necessary to look for signs of disease extension. Urinary diversion and/or colostomy diversion are often required in perineal NF to manage infection. Sometimes amputation of the limbs is required especially in individuals who have peripheral vascular disease and/or diabetes. It is often necessary to do reconstructive surgeries such as skin grafting, flap, or free flap. 28

According to Wong et al, delaying the surgery for more than 24 hours after hospital admission results in a nine-fold increase in mortality. Incomplete vs thorough debridement was observed to increase mortality by Bilton et al by 4% to 38% whereas Voros et al reported a sharp rise in mortality with delay and insufficient surgical intervention. <sup>22,29,30</sup>

It is advisable to provide antimicrobial medication based on the findings of susceptibility and culture. Even if enteric pathogen resistance is rising, beta-lactam/beta-lactamase combinations like ampicillin/sulbactam, piperacillin/tazobactam or ticarcillin/clavulinate remain fair empirical options. It is also advised to use third- or fourth-generation cephalosporins that include either clindamycin or metronidazole for anaerobe coverage.<sup>31</sup>

Patient complained of perianal secretion and discomfort for three days, and she had previously used local selfmedication in the perianal region to cure haemorrhoids using local medicinal herbs (Kobiraji). This was a fastmoving case with both systemic and local NF symptoms. The next day, after prompt and thorough surgical debridement, all adhering tissues in the perianal region could be easily dissected with the fingers which supports the diagnosis. In this instance, the medicinal plant's chemical burn caused NF to develop. However, there is no evidence of bacterial growth in culture. Metronidazole and Ceftriaxon were used as antibiotherapy treatments for the patient. Colostomies were performed in order to hasten the healing process since all tissues from the perianal region had been removed. Additionally, a rotation flap and reversal colostomy were used to repair the perianal region after the incision had sufficiently healed. Since these cases are rare, as far as we know, we are sharing this one in order to raise awareness among surgeons who could treat this kind of skin infection.

#### Conclusion

Necrotizing fasciitis is always a clinical diagnosis. It should be suspected in every skin infection with fever, signs of systemic toxicity and severe pain. A high clinical suspicion followed by prompt surgical exploration can save the life of patient. Reconstruction of affected area is a needed for better outcome. Despite antibiotic therapy and surgical intervention, the mortality and morbidity of NF remain high if early identification of the necrotizing process is not done. So, the most important determinants of mortality are the timing and adequacy of debridement.

## Acknowledgment

The author is immensely grateful to Dr. Mahmood Kamal Vaskor for his comments on an earlier version of the manuscript to improve it.

### References

- 1. Trent JT, Kirsner RS. Diagnosing necrotizing fasciitis. Advances in Skin & Wound Care. 2002; 15(3):135-8.
- Brun-Buisson CA. Stratégie de prise en charge des fasciites nécrosantes. In Annales de dermatologie et de vénéréologie. 2001; 128 (3):394-403.
- 3. Wilson B. Necrotizing fasciitis. Am Surg. 1952; 18:416.
- 4. Levin EG. Manders SM. Life threatening necrotizing fascitis. Clin Dermatol. 2005; 23:144-7.
- 5. Wong CH, Chang HC, Pasupathy S, Khin LW, Tan JL, Low CO. Necrotizing fasciitis: Clinical presentation, microbiology and determinants of mortality. JBJS. 2003; 85(8):1454-60.
- 6. Childers BJ, Potyondy LD, Nachreiner R, Rogers FR, Childers ER, Oberg KC et al. Necrotizing fasciitis: A fourteen-year retrospective study of 163 consecutive patients. The American Surgeon. 2002; 68(2):109-16.
- 7. Bisno AL, Stevens DL. Streptococcal infections of skin and soft tissues. New England Journal of Medicine. 1996; 334(4):240-6.
- 8. Adams EM, Gudmundsson S, Yocum DE et al. Streptococcal myositis. Arch Intern Med. 1985; 145:1020-102.
- 9. Guilianio A, Lewis Jr, Hardley L, Blaisdell FW. Bacteriology of necrotizing fascitis. Am Surg. 1977; 134:52-7.
- 10. Bruin-Buisson C. Strategies de prise en charge des fascitis necrosantes. Ann Dermatol Venereol. 2001; 128:394-404.
- 11. Chelsom J, Halstensen A, Haga T, Høiby EA. Necrotising fasciitis due to group A streptococci in western Norway: Incidence and clinical features. The Lancet. 1994; 344 (8930):1111-5.
- 12. Moss RM, Kunpittaya S, Sorasuchart A. Cervical necrotizing fasciitis: An uncommon sequela to dental infection. Annals of Otology, Rhinology & Laryngology. 1990; 99(8):643-6.
- 13. Maisel RH, Karlen R. Cervical necrotizing fasciitis. The Laryngoscope. 1994; 104(7):795-8.
- 14. Zerr DM, Rubens CE. NSAIDS and necrotizing fasciitis. The Pediatric Infectious Disease Journal. 1999; 18(8):724-5.
- 15. Aronoff DM, Bloch KC. Assessing the relationship between the use of nonsteroidal anti-inflammatory drugs and necrotizing fasciitis caused by group a streptococcus. Medicine. 2003; 82(4):225-35.
- 16. Hill MK, Sanders CV. Necrotizing and gangrenous soft tissue infections. The skin and infection: A color atlas and text. Baltimore, MD: Lipincott, Williams & Wilkins. 1995:62-75.
- 17. Headley AJ. Necrotizing soft tissue infections: A primary care review. American Family Physician. 2003; 68(2):323-8.
- 18. Hsiao CT, Lin LJ, Shiao CJ, Hsiao KY, Chen IC. Hemorrhagic bullae are not only skin deep. Am J Emerg Med. 2008; 26(3):316-9.
- 19. Wang YS, Wong CH, Tay YK. Staging of necrotizing fasciitis based on the evolving cutaneous features. International Journal of Dermatology. 2007; 46(10):1036-41.
- 20. Elliott DC, Kufera JA, Myers RA. Necrotizing soft tissue infections. Risk factors for mortality and strategies for management. Annals of Surgery. 1996; 224(5):672.



- 21. Das DK, Baker MG, Venugopal K. Risk factors, microbiological findings and outcomes of necrotizing fasciitis in New Zealand: A retrospective chart review. BMC Infectious Diseases. 2012; 12(1):1-8.
- 22. Wong CH, Khin LW, Heng KS, Tan KC, Low CO. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: A tool for distinguishing necrotizing fasciitis from other soft tissue infections. Critical Care Medicine. 2004; 32(7):1535-41.
- 23. Fisher JR, Conway MJ, Takeshita RT et al. Necrotizing fasciitis: Importance of roentgen graphic studies for soft-tissue gas. JAMA. 1979; 241(8):803-6.
- 24. Wysoki MG, Santora TA, Shah RM, Friedman AC. Necrotizing fasciitis: CT characteristics. Radiology. 1997; 203(3):859-63.
- 25. Rahmouni A, Chosidow O, Mathieu D et al. MR imaging in acute infectious cellulitis. Radiology. 1994; 192(2):493-6.
- 26. McHenry CR, Piotrowski JJ, Petrinic D et al. Determinants of mortality for necrotizing soft-tissue infections. Annals of Surgery. 1995; 221(5):558.

- 27. Freischlag JA, Ajalat G, Busuttill RW. Treatment of necrotizing soft tissue infections: The need for a new approach. The American Journal of Surgery. 1985; 149(6):751-5.
- 28. Burge TS, Watson JD. Necrotising fasciitis. British Medical Journa. 1994; 308(6942):1453-4.
- 29. Bilton BD, Zibari GB, McMillan RW, Aultman DF. Aggressive surgical management of necrotizing fasciitis serves to decrease mortality: A retrospective study/discussion. The American Surgeon. 1998; 64(5):397.
- 30. Voros D, Pissiotis C, Georgantas D, Katsaragakis S, Antoniou S, Papadimitriou J. Role of early and extensive surgery in the treatment of severe necrotizing soft tissue infection. Journal of British Surgery. 1993; 80(9):1190-1.
- 31. Childers BJ, Potyondy LD, Nachreiner R, Rogers FR, Childers ER, Oberg KC, Hendricks DL, Hardesty RA. Necrotizing fasciitis: A fourteen- year retrospective study of 163 consecutive patients. The American Surgeon. 2002; 68(2):109-16.