Case Report

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Paraspinal Necrotizing Fasciitis Associated with Pressure Injury: An Unusual Case Report

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ABSTRACT

Necrotizing fasciitis is a type of soft tissue infection that destroys subcutaneous tissue. It is particularly dangerous for patients with chronic diseases and those who are bedridden while recuperating. Although necrotizing fasciitis is often caused by trauma or postoperative infection, in rare cases, it can be attributed to pressure injury (PI). The disease progression is very aggressive and can be lethal for patients who are bedridden or immunocompromised.

This case report describes a 47-year-old man with a history of diabetes and hypertension who became bedridden after a sudden status decline caused by nephrotic syndrome. He gradually developed an infection and rare deterioration of a PI on his upper back. After radiologic evaluation with magnetic resonance imaging and computed tomography, surgical intervention was performed and necrotizing fasciitis was confirmed. In this case of necrotizing fasciitis derived from a PI on the upper back, the infected area spread to the periphery at a rapid rate. The infection spread over his back and across the T1-T9 levels.

In this report, the authors describe the integrated system of the thoracolumbar fascia and the very aggressive spread of necrotizing fasciitis. Because of the anatomic structure of the back and the characteristics of this infection, only aggressive surgical debridement could prevent the spread of infection and reduce the systemic effects of the infection. Physicians should be aware of the possibility of PIs in bedridden patients and, in cases of exacerbation of the wound, consider rapid surgical intervention after prompt examination and diagnosis to reduce mortality.

KEYWORDS: debridement, necrotizing fasciitis, necrotizing soft tissue infection, nephrotic syndrome, pressure injury, wound care

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INTRODUCTION

Necrotizing fasciitis is a medical emergency; because this infection is severe and involves deep, soft tissue, it can easily lead to progressive disease.[1] Providers should carefully observe and track its rapid spread to prevent extensive soft tissue and skin necrosis. Necrotizing fasciitis can develop in many clinical situations, including in patients with major traumatic injuries or nonpenetrating soft tissue injuries, and those who are immunocompromised postsurgery.[2] However, particularly in patients who are older or nonadherent with treatment, necrotizing fasciitis may follow a pressure injury (PI).[3] Necrotizing fasciitis and PI may lead to comorbidity or fatality for the patient.

Pressure injuries commonly occur in places on the body where constant pressure and friction are applied, such as the sacrum, heel, greater trochanter, and lateral malleolar area, but can develop in any location of pressure loading. Although the upper back is a relatively rare location for a PI to develop, the authors have treated patients with upper back PIs and severe deep-tissue infections. In addition to the unusual disease location, these patients' outcomes have differed from those of patients with an ordinary PI infection. Herein, the authors describe a patient with paraspinal necrotizing fasciitis secondary to a PI on his upper back, discuss the patient's challenging clinical course, and comment on the difficulties of treatment. The following case is presented in accordance with the CARE reporting checklist.

CASE REPORT

A 47-year-old man with a history of diabetes mellitus, chronic kidney disease, hypertension, and dyslipidemia experienced general weakness, resulting from the abrupt development of nephrotic syndrome. He lacked insight into his medical condition and had no family; therefore, he was left unattended at home. Despite his young age, he was inactive in his daily life and developed a PI on his back. He was admitted to the nephrology department

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through the ED of the authors' hospital for evaluation and treatment.

His symptomatic nephrotic syndrome worsened, and systemic edema and severe pleural effusion were observed. At the time of his visit, poorly controlled diabetes was noted with a high glucose level (hyperglycemia) of 211 mg/dL (glycated hemoglobin, 7.3%). Continued conservative medical treatment with hemodialysis controlled his general edema well, but his high C-reactive protein (CRP) level and poor condition remained.

The following patient laboratory results were associated with a poor outcome based on the Laboratory Risk Indicator for Necrotizing Fasciitis scoring system: low hemoglobin level, 9.4 g/dL; low erythrocyte count, 3.25 × 106/μL; low serum sodium level, 125 mmol/L; high serum creatinine level, 3.64 mmol/L; high glucose level (hyperglycemia), 211 mg/dL; pain; high fever, >38.0° C; and fast heart rate (tachycardia), >100 beats/min. The patient's condition received a score of 7 points, indicating suspicion of necrotizing fasciitis (score > 6 on the scoring system). The patient also had a low serum total protein level (6.1 g/dL) and hypoalbuminemia (2.3 g/dL) caused by malnutrition during his long period of uncontrolled chronic medical history. These levels were monitored in cooperation and continuous follow-up with the nutrition team.

After evaluation for infection, the patient consulted with the plastic surgery department. Initially, the PI on his upper back was covered with a wet, whitish eschar that was assumed to be stage 4 (Figure 1A). Because of the patient's constant high CRP level (7.91 mg/dL), the authors initially tried to perform debridement for infection control. After the first incision, the surgeon could insert fingers down to the deep fascia and noticed gray dishwater-colored pus and a lack of bleeding, which are indicative of necrotizing fasciitis.

Once the eschar was debrided, massive amounts of pus were drained, and it was discovered that the entire back muscle was necrotized (Figure 1B). Despite continued tissue debridement, the wound bed could not be detected, which was unusual. Because the first debridement failed, contrast-enhanced computed tomography and magnetic resonance imaging were performed (Figures 2A-C). There was soft tissue swelling and fatty infiltration with internal air and fluid collection in the subcutaneous layer of the upper back from T1 to T9. His external wound size measured 10 × 8 cm, and radiologic findings suggested paraspinal necrotizing fasciitis; no findings were noted indicating osteomyelitis.

With cooperation from an orthopedic spine surgeon, serial radical debridement was performed with the patient under general anesthesia. An incision was made along the spine, and the Versajet Hydrosurgery System (Smith and Nephew, Key Largo, Florida) was used to conduct extensive debridement, followed by application of negative-pressure wound therapy.

Subsequently, the patient's CRP level decreased to 2.34 mg/dL. IV antibiotic treatment (piperacillin/tazobactam and ceftriaxone) targeted to the cultured bacteria—multidrug-resistant *Acinetobacter baumannii* and methicillin-resistant *Staphylococcus aureus*, pathogens associated with PI-related hospital-acquired infections—was continued for 4 weeks. The patient's initial high CRP level, polymicrobial wound culture result, extensive open wound, and wound location were obstacles to his recovery that made offloading and pressure distribution difficult. Therefore, the plastic surgeon performed a double rotational fasciocutaneous flap advancement to reconstruct the defect (23 × 16 cm; Figure 1C). Although the granulation tissue was healthy and the infection was relatively controlled, total flap loss occurred within 3 days postoperation because of suspected venous or arterial thrombosis and persistent wound bed infection (Figure 1D).

Figure 1. CLINICAL PHOTOGRAPHS OF THE CASE

A, Initial clinical photograph. The upper back area has eschar (8 × 10 cm), hyperpigmentation, and skin discoloration. B, Photograph of the wound after incision, drainage, and debridement. The wound bed is the paraspinal muscle, and slough tissue is visible. C, Immediate postoperative photograph after coverage with double rotational fasciocutaneous flap advancement. D, Because of recurrent wound bed infection, the double rotational flap failed. E, Photograph at 3 months after split-thickness skin graft coverage.

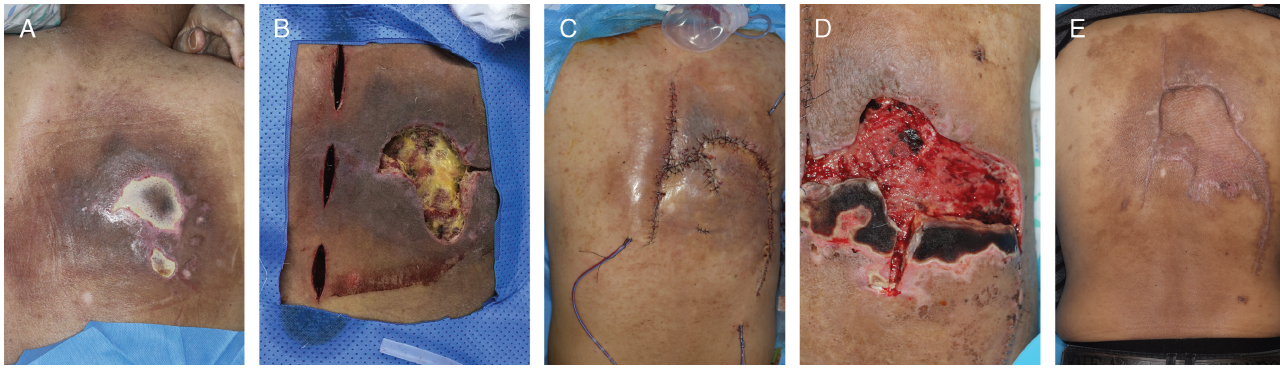
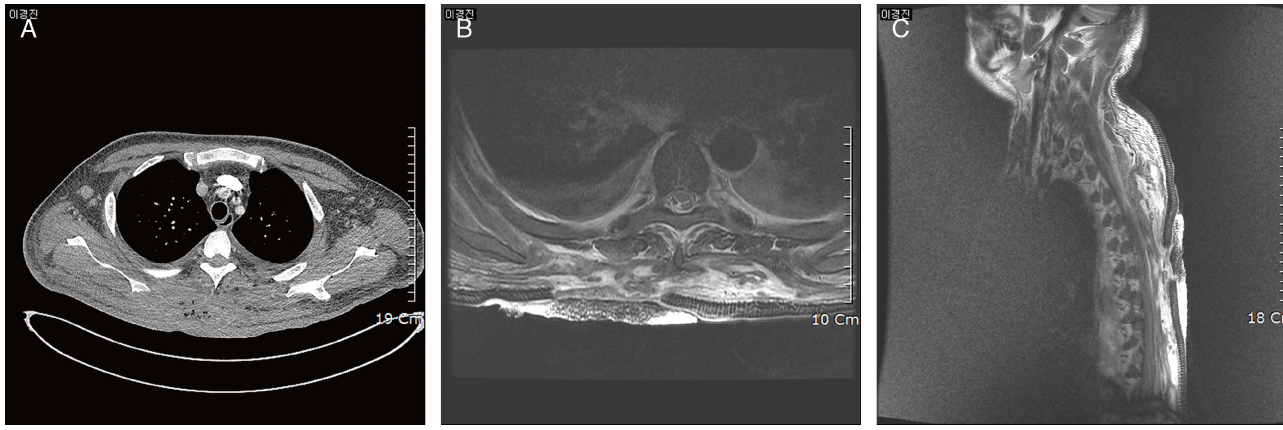


Figure 2. RADIOGRAPHIC EVALUATION WITH CONTRAST-ENHANCED COMPUTED TOMOGRAPHY AND MAGNETIC RESONANCE IMAGING

A, Initial contrast-enhanced computed tomography scan in the axial view. The most specific finding, gas formation, and asymmetrical fascial thickening and edema into the intermuscular septa are visible. Reticular attenuation of the subcutaneous fat in the suspected area is increased, which indicates infection. The infectious area is considered a lesion because it has lower attenuation than normal muscle, such as the latissimus dorsi. B and C, T2 axial and sagittal images showing an irregular signal below the skin lesion over the T1-T9 levels. A hypointense signal in an irregular signal area is suggestive of gas formation. The high intensity of the deep fascia is noted around the erector spinae and paraspinal muscles. Peripheral edema can also be seen. The T2 hyperintense signal of the deep intermuscular fascia is a significant finding for the diagnosis of necrotizing fasciitis.



For the third attempt at treatment, the authors chose careful conservative treatment for complete infection control. The necrotic lesion was debrided, and the wound was irrigated, then a povidone-iodine (Betadine)-soaked dressing and vacuum-assisted dressing were applied. Finally, the defect (20 × 15 cm) was covered using a split-thickness skin graft after confirming the infection had abated. Three months later, the patient recovered without any complications through outpatient observation (Figure 1E).

Written informed consent was obtained from the patient for publication of this case report and accompanying images. All procedures performed in studies involving human participants were in accordance with the ethical standards of Ajou University Hospital and with the Helsinki Declaration (as revised in 2013).

DISCUSSION

Necrotizing fasciitis is a medical emergency with high morbidity and mortality.[2] Spontaneous PIs in patients who are bedridden are common;[4] however, it is difficult to predict the concomitant development of necrotizing fasciitis. Few studies have reported the development of necrotizing fasciitis following a PI. Further, PIs arising from a lack of mobility may not have clear clinical features after they transition to necrotizing fasciitis. Mizokami et al[5] focused on the reasons for the rapid spread of necrotizing fasciitis. Among the many possible types of infection sources, rapidly progressive necrotizing fasciitis is caused by group A *Streptococcus*.[6] In this case, a combination infection of group A *Streptococcus* and Gram-negative *A baumannii* contributed to the patient's difficult clinical course. Given this infectious milieu, the pressure on a given anatomic location may be a critical factor in the development of necrotizing soft-tissue infection.[7]

The location of paraspinal necrotizing fasciitis in the present case differed from other common sites of necrotizing fasciitis. Necrotizing fasciitis invades the fascial planes and, in the current case, it spread along the spinal fascial plane. Anatomically, the thoracolumbar fascia has a posterior layer that consists of two laminae. The superficial lamina continues toward the aponeurosis of the latissimus dorsi, and the deep lamina continues toward the fibrotic band of the lumbar spine.[8] The integrated system of the thoracolumbar fascia contributed to the rapid spreading of necrotizing fasciitis.

Interestingly, the patient's infection extended from T1 to T9 and was caused by only a PI on the upper back. The thoracic skin-fascia interface is a space in which friction force between the skin and the underlying vertebra transmits nonnormal forces.[9] Regarding the mechanics of pressure loading on the upper back, weight can easily spread over this space through friction. Therefore, necrotizing fasciitis, especially following a PI on the upper back, would result in wide involvement of the paraspinal muscle through the skin-fascia interface.

The authors recommend rapid surgical intervention for patients with such an aggressive infection. Rapid spread along the spine can cause systemic toxicity. More than 50% of patients with necrotizing fasciitis have no specific related medical history, but risk factors include diabetes, chronic renal failure, and immunosuppression.[10,11] This

diversity of predisposing conditions makes it difficult to detect the disease and leads to a high mortality of up to 43%.[12] Therefore, prompt surgical exploration is extremely important.[13] Only through radical debridement can surgeons both confirm the extent of and control the infection. Assessment and debridement should be continued every 1 to 2 days until no necrotic tissue remains.[14] It may be a very long and difficult process for the patient to endure, but early trials of reconstruction could lead to failure, as was seen in this case report. Prompt debridement surgery within 24 hours of onset of necrotizing fasciitis increases patient survival.[15]

CONCLUSIONS

Patients with chronic disease who experience reduced mobility for extended periods are at high risk of PIs. This case confirmed that PIs can lead to paraspinal necrotizing fasciitis and be lethal to these individuals. Ambiguous features can lead to delay in early surgical intervention, which is an important factor for decreasing mortality. By closely observing and evaluating the possibility of PI in patients with chronic disease, appropriate early interventions can save the patient's life.

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