

## Pyelonephritis can be a source of a life-threatening necrotizing myofasciitis

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### Abstract

Necrotizing fasciitis is a progressive, rapidly spreading, inflammatory infection located in deep fascia. It may cause necrosis of skin and subcutaneous tissue and can even result in involvement of adjacent soft tissues such as muscles resulting in necrotizing myositis. We report the case of an adult male presenting with necrotizing myofasciitis secondary to left pyelonephritis. We also review the relevant literature.

### Introduction

Necrotizing fasciitis is a progressive, rapidly spreading, inflammatory infection located in deep fascia. It may cause necrosis of skin and subcutaneous tissue and can spread to adjacent tissues. The disease can spread via blood vessels and lymphatic can lead to shock, organ failure and death.<sup>1,2</sup>

The term “necrotizing soft tissue infection” now includes all forms of the disease process because the necrotizing infection involves all soft tissue and treatment is the same regardless of the anatomic location or depth of infection.<sup>3</sup>

Necrotizing fasciitis of the retroperitoneal space is uncommon and usually fatal. The lack of external clinical signs, the anatomical barriers to adequate debridement, and the concurrent comorbid conditions combine to make this entity very difficult to manage.<sup>4</sup>

### Case report

A 50-year-old male presented to emergency department with fever and generalized abdominal pain. He did not complain of urinary symptoms. On admission his vital signs were normal, except for a temperature of 38.5°C. Abdominal examination revealed no skin abnormalities and tender and rigid abdomen, more in the left flank and left iliac fossa.

Laboratory findings were leukocytosis at 21000/mm<sup>3</sup>, creatinine at (2.1 mg/dL) and random blood glucose at 116 mg/dL.

The patient had a history of hepatitis C virus, although current liver enzymes were normal. Abdominal ultrasonography revealed ectasia of the left pelvicalyceal system. A computed tomography scan showed no abnormalities, except slight fullness of the left pelvicalyceal system. The case was initially seen by a consultant of general surgery who diagnosed the case as acute diverticulitis with possible rupture of sigmoid colon. We, the urology team, were called to insert a double-J stent in the kidneys endoscopically to help the general surgeon explore without risking accidental ureteric injury. Surprisingly, inserting the double-J stent showed blackish discharge coming out of the left kidney (Fig. 1, Fig. 2, Fig. 3). Surgical exploration revealed necrosis of all fascial planes, peritoneal coverings and extending to the psoas muscle and perirenal space. Thorough debridement was performed. The left kidney did not appear healthy and we found that the kidney tissue would be macerated, with further dissection from the perinephric fat. We elected to limit the debridement instead of proceeding to nephrectomy. Urine culture grew *Escherichia coli*. The patient was then admitted to the intensive care unit, was administered a high dose of antibiotics and was started on aggressive fluid resuscitation. The patient showed gradual improvement and was discharged 2 weeks later. The double-J stent was removed after 6 weeks. An ultrasound done 3 months later was free of necrotizing myofasciitis.

### Discussion

Al Ammari and colleagues<sup>5</sup> described a case that was diagnosed preoperatively due to skin erythema and palpable crepitus. Their case was managed by nephrostomy draining the kidney, in addition to surgical debridement. Unfortunately, their patient died despite all possible supportive care.



**Fig. 1.** Black discharge through the ureteric catheter.

## Etiology

There are several predisposing factors that may lead to a patient's susceptibility to this disease. Primarily, immune deficiency, due to diabetes mellitus, complement C4 deficiency, AIDS, malignancies and drug use, is the main factor.<sup>6</sup>

Primary infection can penetrate due to skin trauma, infected needle in intravenous drug abusers or previous dermatological diseases, such as psoriasis and bed sores. Mucosal membranes in gastrointestinal or genitourinary systems are the other routes of infection.<sup>2</sup> Infection is often polymicrobial, which can be anaerobic or a mixture of aerobic and anaerobic.<sup>7</sup>

## Clinical picture

Classic symptoms, such as pain, anxiety and diaphoresis, worsen rapidly. As with other acute ischemia conditions (e.g., mesenteric ischemia), the pain is usually out of proportion to physical examination findings, but some patients might have little or no pain. The affected area can become insensate as additional tissue necrosis ensues.<sup>8</sup>

The only features that give out clues to early diagnosis are: early loss of muscular power (owing to early myonecrosis) unexplained by the other common conditions; precipitous course; and pain disproportionate to clinical signs (akin to mesenteric vascular infarction). A high index of suspicion is necessary to recognize this triad to diagnose this condition early.<sup>9</sup>

On clinical examination, the appearance of the overlying skin may vary, ranging from normal to resembling necrotizing fasciitis with signs of necrosis. Muscle strength may decrease with active and passive movement eliciting pain.<sup>10</sup>

## Investigations

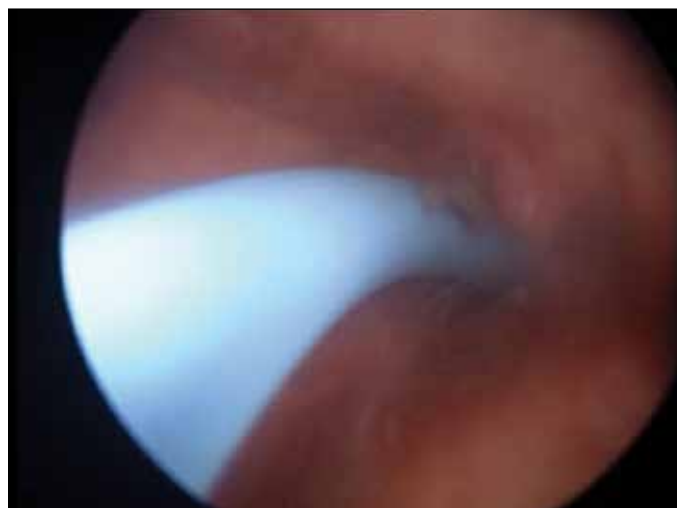
The most frequent misdiagnoses were flu/viral illness, deep vein thrombosis, or muscle strain/rupture/hematoma/abscess.<sup>11</sup>

The gold standard for detecting necrotizing soft tissue infections is tissue biopsy obtained at the time of wound exploration and surgical debridement. During wound exploration, tissue integrity and depth of invasion also can be evaluated. The findings of fascial necrosis and myonecrosis are indicative of necrotizing infection. Loss of fascial integrity along tissue planes and frank evidence of muscle involvement are also diagnostic. The use of frozen sections at the time of biopsy may not always provide accurate information about the depth of tissue involvement.<sup>12</sup>

In a retrospective study, Wall and colleagues<sup>13</sup> found that patients with necrotizing infection had either a white blood cell count  $>15\,400$  cells/mm<sup>3</sup> or a sodium level  $<135$  mmol/L on admission to the hospital.

Creatine phosphokinase (CPK) values were far higher in patients with group A streptococcal (GAS) necrotizing fasciitis than in those with non-GAS necrotizing fasciitis. Eleven of 18 (61%) patients with GAS necrotizing fasciitis had CPK levels of  $>600$  IU/L on admission, while none of the patients with non-GAS necrotizing fasciitis had high CPK levels. This indicates that muscle involvement is an early step in the course of necrotizing soft tissue infection caused by gas-forming organisms.<sup>14</sup>

Magnetic resonance imaging can be a helpful diagnostic adjunct because of its soft-tissue and multiplanar-imaging capabilities.<sup>15</sup>



**Fig. 2.** Persistent black discharge and gas through the ureteric catheter.



**Fig. 3.** Double J stent inserted for complete kidney drainage.

## Treatment

The mainstay of treatment is complete surgical debridement, combined with antimicrobial therapy, close monitoring, and physiologic support.<sup>16</sup> When the retroperitoneum is involved, excision may be delayed secondary to the lack of these clinical manifestations. The anatomy of the retroperitoneum is such that curative debridement is often not feasible, leading to a mortality rate that approaches 100%.<sup>4</sup>

Once the initial debridement has been done, management in an intensive care unit is recommended, and scheduled debridement at intervals of 6 to 48 hours should be performed until no further necrosis or infected tissue is seen. Close monitoring of the vital signs of the patient, as well as serial white blood cell counts (WBC), should be performed every 6 to 12 hours. Any additional physiologic derangement or increase in the WBC count before the planned debridement should prompt more frequent reoperations.<sup>17</sup>

The goal of pharmacotherapy is to eradicate the infection, avoid complications, and lessen morbidity and mortality. Broad-spectrum antimicrobial therapy should be empirically administered as soon as possible, and should cover likely pathogens. Combination therapy may be employed and involves the use of 2 or 3 antibiotics. Clindamycin is highly recommended at the earliest thought of group A streptococcal bacteremia as it is believed to inhibit both M-protein and exotoxin production. Clindamycin or metronidazole may be employed for anaerobes. Single antibiotic coverage involves the use of broad-spectrum drugs. Imipenem-cilastatin, for example, may be used alone or with the addition of vancomycin for methicillin resistant *S. aureus*. It provides coverage against anaerobes and aerobes, including *Pseudomonas* species for empiric therapy until microbiology results are available. Ampicillin sulbactam is another broad-spectrum antibiotic, but it lacks *Pseudomonas* species coverage. The

antibiotic coverage should be reassessed upon receiving culture and sensitivity results.<sup>12</sup>

Hyperbaric oxygen (HBO) therapy may be considered as an adjunct to other modalities, including surgical debridement and antibiotic administration, as long as it does not interfere with them. Increased tissue oxygenation following HBO may prevent the spread of the offending organisms. However, prospective studies demonstrating the efficacy of HBO are still lacking.<sup>12</sup>

The use of intravenous immunoglobulin (IVIG) may be a useful adjunct treatment in severe streptococcal infections. IVIG is thought to work against streptococci by neutralizing super antigen activity and reducing plasma levels of TNF and IL-6.<sup>12</sup>

Finally, appropriate early nutritional support, delivered enterally if possible, helps to control the catabolic response of these patients. Aggressive fluid resuscitation and blood component therapy are often required during the perioperative period.<sup>17</sup>

## Conclusion

Retroperitoneal necrotizing myofasciitis is a special entity that is hard to diagnose before surgical intervention, due to absent abdominal wall signs and non-conclusive radiological findings. Drainage of the affected kidney may be helpful, in addition to surgical debridement and broad spectrum antibiotics.

**Competing interests:** Dr. Kamel, Dr. Awed and Dr. Kotb all declare no competing financial or personal interests.

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