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Retroperitoneal Fasciitis: Spectrum of CT Findings in the Abdomen and Pelvis[1]

Christina M. Chingkoe, MD
Ali Jahed, MD, PhD
Michael P. Loreto, MD, FRCPC
Josée Sarrazin, MD, FRCPC
Caitlin T. McGregor, MD, FRCPC
Jason I. Blaichman, MD
Phyllis Glanc, MD, FRCPC

Abbreviation: ICU = intensive care unit

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[1]From the Department of Medical Imaging, University of Toronto, Toronto, Ont, Canada (C.M.C., A.J., J.I.B.); Department of Radiology, Health Sciences North, Sudbury General Hospital, Sudbury, Ont, Canada (M.P.L.); and Department of Medical Imaging, Sunnybrook Health Sciences Centre, 2075 Bayview Ave, MG160, Toronto, ON, Canada M4N 3M5 (J.S., C.T.M., P.G.). Presented as an education exhibit at the 2013 RSNA Annual Meeting. Received May 19, 2014; revision requested August 28 and received October 2; accepted October 8. For this journal-based SA-CME activity, the authors, editor, and reviewers have disclosed no relevant relationships. **Address correspondence to** P.G. (e-mail: *phyllis.glanc@sunnybrook.ca*).

SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Recognize the spectrum of CT findings in retroperitoneal fasciitis.
- Describe the pathways of spread of retroperitoneal fasciitis.
- Discuss the differential diagnostic procedure for this condition.

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Retroperitoneal fasciitis is a rare but potentially lethal complication of infection. Early diagnosis is crucial and is usually made when there is a high degree of clinical suspicion combined with characteristic imaging findings leading to early surgical intervention. Computed tomography (CT) can play a central role in demonstrating early findings, assessing the extent of disease to help determine the best surgical approach, identifying the primary source of infection, and evaluating the treatment response. The possible presence of retroperitoneal fasciitis should be considered in patients presenting with symptoms of sepsis, including pain that is disproportionate with the clinical abnormality. When retroperitoneal fasciitis is suspected, emergency CT can facilitate early diagnosis and evaluation of the extent of disease. Common findings at CT include fascial thickening and enhancement, muscular edema, fat stranding, fluid collections, and abscess formation. Gas tracking along fascial planes in the retroperitoneum is the hallmark of retroperitoneal fasciitis but is not seen in all cases. Another important clue to the diagnosis is asymmetric involvement of the retroperitoneal fascial planes and deep tissues. Fasciitis in the retroperitoneum may originate from infected retroperitoneal organs or from infection that spreads along indirect and/or direct pathways from a primary source elsewhere in the body. Findings of indirect tracking and transgression of fascial planes may indicate more severe infection associated with the necrotizing form of retroperitoneal fasciitis. Despite aggressive antibiotic treatment, early and repeated surgical débridement may be required to remove nonviable tissue in patients with the necrotizing form of retroperitoneal fasciitis. Awareness of the anatomy of the retroperitoneum, potential routes of spread of infection, and the spectrum of CT findings in retroperitoneal fasciitis is needed to achieve prompt diagnosis and guide treatment.

Introduction

Retroperitoneal fasciitis is a rare and potentially lethal rapidly progressive infection involving the retroperitoneal (extraperitoneal) deep soft tissues and fascial planes of the abdomen and pelvis. The incidence of retroperitoneal fasciitis is unknown as the literature is limited to case reports and case series. The largest described case series we are aware of involved 10 patients (1). Although necrotizing fasciitis of the integument has been well described, the retroperitoneal location of this infection is not as well documented.

The disease can range from a simple fasciitis or infection of the fascial planes to a fulminant necrotizing form, which is associated with thrombosed vessels and nonviable tissue. The distinction between the nonnecrotizing and necrotizing forms of retroperitoneal

TEACHING POINTS

- Important clinical clues for the diagnosis of retroperitoneal fasciitis include inordinate pain and rapid clinical deterioration, particularly in the postsurgical setting or in a patient with known lower extremity necrotizing fasciitis.
- Awareness of the spectrum of CT findings in retroperitoneal fasciitis is crucial, as early diagnosis may provide a window of opportunity in which to institute life-saving aggressive supportive and surgical measures.
- Findings at CT include asymmetric fascial thickening and enhancement, muscular edema, fat stranding, fluid collections, and abscess formation. Asymmetric involvement of the retroperitoneal fascial planes and deep tissues is an important clue to the diagnosis. Although gas tracking along fascial planes is considered a characteristic finding of retroperitoneal fasciitis, it is present only in a minority of cases in our experience.
- The necrotizing form of retroperitoneal fasciitis is characterized by the transgression of fascial planes (indirect tracking) and a more aggressive rapid clinical course. Additional findings include evidence of vascular thrombosis, hemorrhage, multifocal abscess formation, and frankly necrotic organs.
- In the necrotizing form of retroperitoneal fasciitis, early and repeated surgical débridement of the affected tissues in conjunction with aggressive administration of antibiotics are the key treatment elements.

fasciitis may not be possible on purely clinical or imaging criteria, ultimately requiring histologic confirmation. However, indirect tracking and transgression of fascial planes suggest the more severe necrotizing form of retroperitoneal fasciitis, as would frank breakdown of anatomic structures (Fig 1).

Unlike necrotizing fasciitis of the integument, retroperitoneal fasciitis is not associated with specific external clinical signs and often manifests with multiple comorbidities that may further complicate the diagnosis. Important clinical clues for the diagnosis of retroperitoneal fasciitis include inordinate pain and rapid clinical deterioration, particularly in the postsurgical setting or in a patient with known lower extremity necrotizing fasciitis. Awareness of the spectrum of CT findings in retroperitoneal fasciitis is crucial, as early diagnosis may provide a window of opportunity in which to institute life-saving aggressive supportive and surgical measures (2,3).

History

The first case report describing necrotizing retroperitoneal fasciitis was by Rush et al in 1991 (4). The subsequent report of a group of five patients by Woodburn et al in 1992 of necrotizing retroperitoneal fasciitis described a uniformly fatal disease despite surgical débridement (5). The actual incidence of retroperitoneal fasciitis is unknown and the mortality remains high, particularly in patients affected by the necrotizing form.

Predisposing Factors

Affected patients will often have systemic illnesses or immune-compromised states, such as diabetes mellitus, obesity, advanced age, liver cirrhosis, leukemia, human immunodeficiency virus (HIV), renal failure, or septicemia. Iatrogenic causes include steroid treatment, chemotherapy, and postsurgical states. In the largest case series by Mokoena et al, three of 10 patients with necrotizing retroperitoneal fasciitis had prior gynecologic procedures including cesarean section and intrauterine instrumentation (1).

Clinical Manifestation

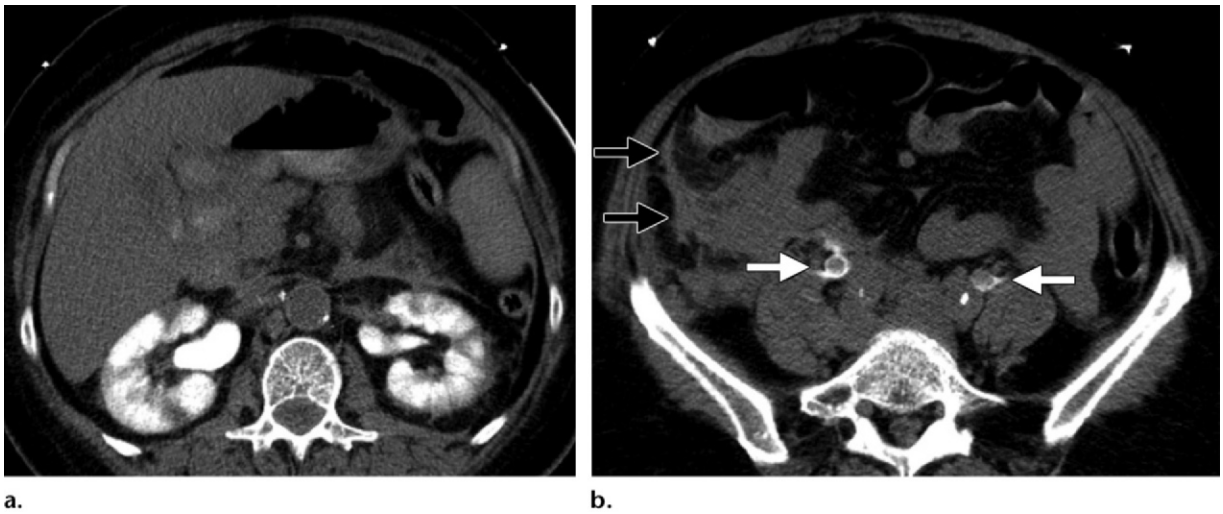
There is a paucity of information regarding the clinical manifestation of either simple retroperitoneal fasciitis or the necrotizing form of retroperitoneal fasciitis. Because of the lack of specific clinical signs in retroperitoneal fasciitis, there is often a delay in diagnosis, resulting in presentation at an advanced stage with symptoms of septic shock and hypotension (5,6).

The clinical diagnosis of necrotizing retroperitoneal fasciitis is challenging because findings at physical examination are limited. A high index of suspicion should be maintained for the diagnosis of retroperitoneal fasciitis in patients who present with inordinate abdominal pain and evidence of infection, particularly in the postsurgical period. It is believed that the disproportionate pain is related to a combination of nerve involvement and intramuscular edema with swelling resulting in compartment syndromes (2,7). Examples may include the thigh (when this is a source of the retroperitoneal fasciitis) or the psoas muscles. The clinical examination of the abdomen is nonspecific; however, palpation of the abdomen may elicit extreme tenderness. There are reported instances in which there may occasionally be visible extension of erythema and skin induration to the abdominal walls and flanks, reminiscent of retroperitoneal hemorrhage (8). It is important to search for a potential point of entry from the lower limb or perineum. Necrotizing fasciitis of the lower extremity, in particular the thigh, is commonly cited as an entry point in many cases of retroperitoneal fasciitis.

Laboratory findings are nonspecific at best, with variable elevation of white blood cells, erythrocyte sedimentation rate, and C-reactive protein (6).

Etiology

The majority of retroperitoneal fasciitis cases report identifiable primary sources of infection including appendicitis, diverticulitis, pyelone-



a. b.

c.

Figure 1. Necrotizing retroperitoneal fasciitis. A 61-year-old woman presented to the emergency department in extremis with rapidly progressive shock; resuscitation was unsuccessful, and she died within a short period of time. An indwelling intrauterine device associated with a right cornual abscess was believed to be the primary source of the extensive necrotizing retroperitoneal fasciitis identified at autopsy. β-hemolytic group A *Streptococcus* was cultured. **(a)** Axial computed tomographic (CT) image shows bilateral striated nephrograms as well as a flattened inferior vena cava, findings consistent with a diagnosis of shock. **(b)** Axial CT image shows bilateral ureteric contrast material extravasation secondary to ureteric necrosis (white arrows). In addition, there is extensive retroperitoneal fluid and stranding, asymmetrically greater on the right side (black arrows). **(c)** Coronal CT image shows right ureteric necrosis with contrast material extravasation (white arrow). There is extensive fluid within the retroperitoneal planes (black arrows).

phritis, perianal abscess, colon cancer, perforation, or postsurgical complications (9–22). Extra-abdominal primary sources of infection with retroperitoneal spread have also been described, most commonly involving necrotizing fasciitis of the extremities. However, not all cases have a definitive source of infection (23–26).

The majority of reported cases of retroperitoneal fasciitis are due to polymicrobial infections (5,15,25,27,28). Whether the inciting source is polymicrobial or monomicrobial may be partially dependent on the primary source of infection. Polymicrobial infections were more frequently cultured in cases of intraperitoneal or retroperitoneal sources of infection. Woodburn et al (5) demonstrated cases of polymicrobial cultures including *Bacteroides* species, *Escherichia coli*, and anaerobic streptococci in cases of retroperitoneal fasciitis from colon carcinoma, diverticular abscess, and intra-abdominal lymphoma. A review by Lancerotto et al (2) indicated that polymicrobial cultures were most commonly isolated in necrotizing fasciitis of the trunk, abdomen, and perineum. These consisted of at least one anaerobic and one facultative anaerobic bacterium, most commonly *Enterobacteriaceae* or non–group A streptococci (2). Other cultured organisms included several *Streptococcus* species (*S pyogenes, S pneumoniae,* and *Enterococcus faecalis*), in tandem with *E coli, Klebsiella pneumoniae, Staphylococcus aureus,* and *Clostridium* and *Bacteroides* species.

Single-organism *Streptococcus* (β-hemolytic group A or other) infections were more commonly isolated in cases of retroperitoneal fasciitis that are due to retroperitoneal extension from necrotizing fasciitis of the lower extremities (26,29). Not surprisingly, the most common causative agent of necrotizing fasciitis in the extremities is β-hemolytic group A *Streptococcus* (2,7), followed by methicillin-resistant *S aureus* (30).

Pathways of Spread

Understanding of the pathways of spread may be critical in achieving an early diagnosis of retroperitoneal fasciitis as infection tends to spread along the fascial planes. It is now recognized that these fascial planes represent potential spaces (31,32). The retroperitoneal space is extraperitoneal, located superficial to the parietal peritoneum and deep to the transversalis fascia (a fascial layer underlying the innermost muscular layer of the

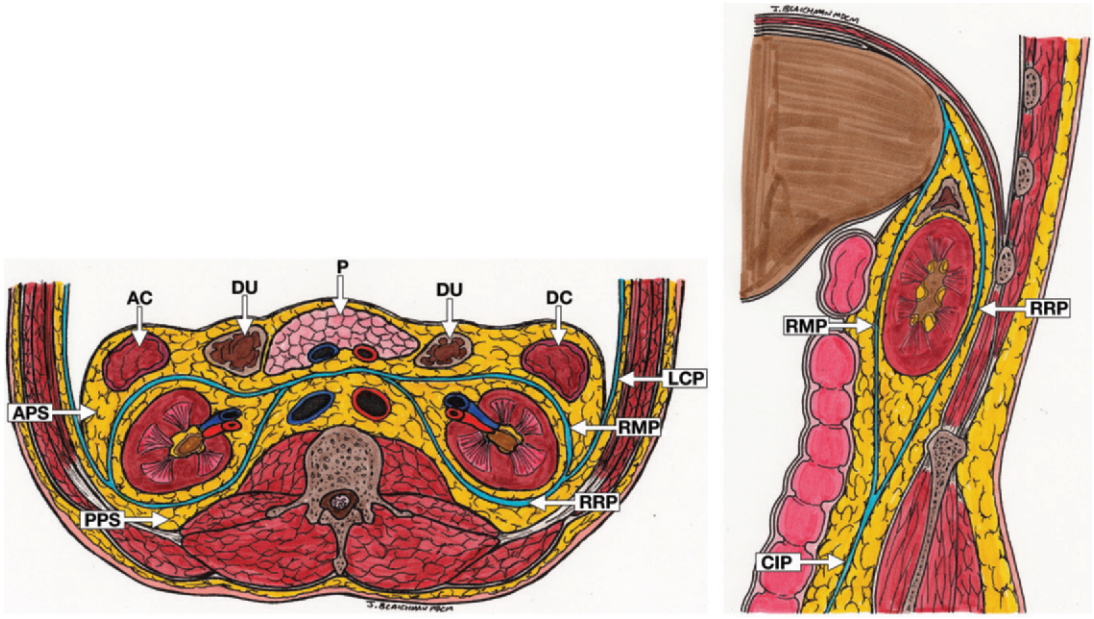


Figure 2. Retromesenteric spaces and planes. **(a)** Transverse drawing. There are three retroperitoneal spaces: the perirenal space, which surrounds the kidneys, adrenal glands, and proximal ureters bilaterally (depicted as the fat directly surrounding the kidneys in the drawing); the anterior pararenal space *(APS)* containing the pancreas *(P)*, ascending colon *(AC)*, descending colon *(DC)*, and parts of the duodenum *(DU)*, located anterior to the perirenal space and superficial to the parietal peritoneum; and the posterior pararenal space *(PPS)*, which is deep to the transversalis fascia (a fascial layer that underlies the innermost muscular layer of the abdomen) and posterior to the perirenal space. There are three retroperitoneal planes that act as potential spaces: the retromesenteric plane *(RMP)* between the anterior pararenal space and perirenal space; the retrorenal plane *(RRP)* between the perirenal space and posterior pararenal space; and the lateral conal plane *(LCP)*, a potential space communicating laterally with these two other planes. In the drawing, the innermost line of the lateral conal plane and anterior pararenal space represents the parietal peritoneum. **(b)** Sagittal drawing. Inferiorly, the retromesenteric plane *(RMP)* and retrorenal plane *(RRP)* merge to form the combined interfascial plane *(CIP)*.

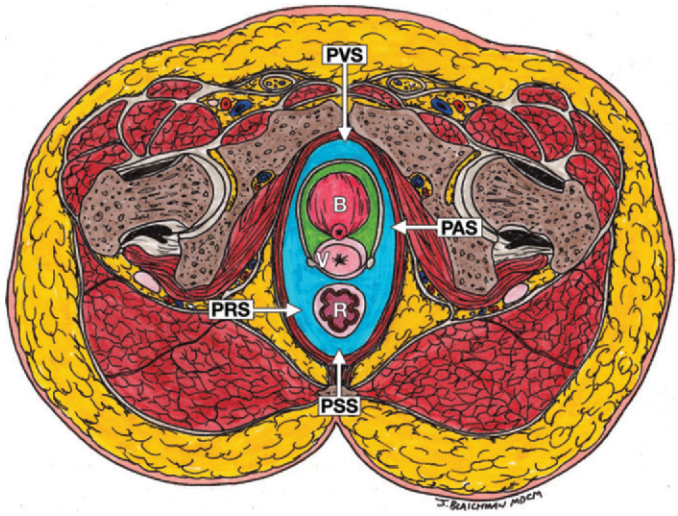
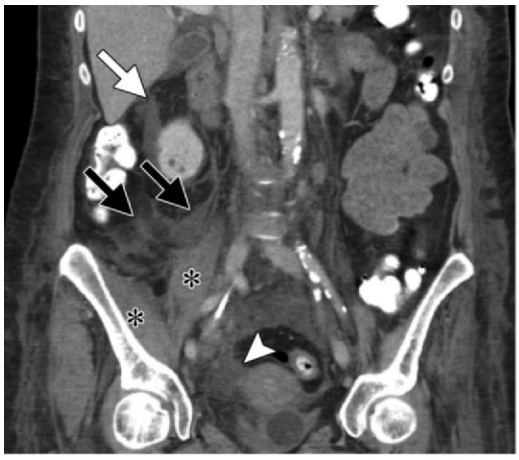


Figure 3. Axial drawing of the pelvic extraperitoneal spaces showing the prevesicular space *(PVS)*, paravesicular space *(PAS)*, perirectal space *(PRS)*, and presacral space *(PSS)*, which are in continuity with each other and with the retromesenteric spaces superiorly. Depicted centrally are the bladder *(B)*, vagina *(V)*, and rectum *(R)*.

abdomen). It is comprised of the anterior pararenal space, perirenal space, and posterior pararenal space (Fig 2). The perirenal spaces are cone-shaped structures, which are somewhat symmetric bilaterally and contain the kidneys, adrenal glands, and proximal ureters. These communicate through septa and lymphatics with the surrounding fascia classically known as Gerota fascia anteriorly and Zuckerkandl fascia posteriorly (33). As stated earlier, recent evidence shows that these fascia are laminar and potential spaces, which are now known as the retromesenteric space anteriorly and the retrorenal space posteriorly. These spaces communicate laterally with the lateral conal space, fuse inferiorly to form the combined interfascial space, and are in continuity with the pelvic extraperitoneal spaces (Fig 3). The anterior pararenal space lies anterior to the retromesenteric fascia and contains



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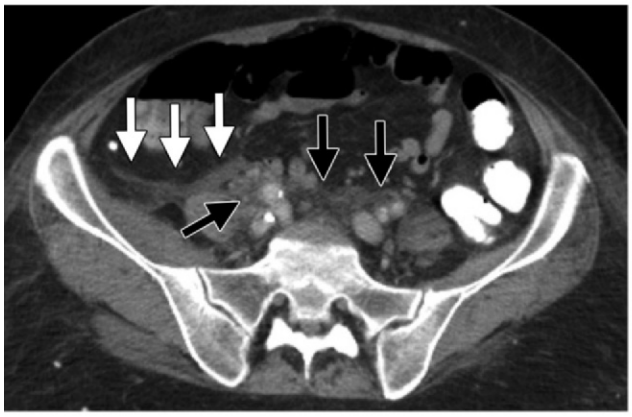

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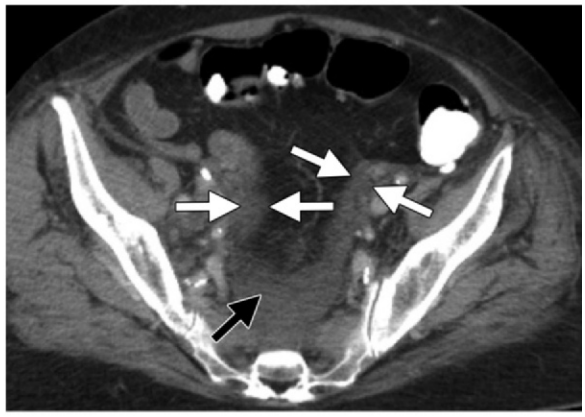

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Figure 4. Nonnecrotizing retroperitoneal fasciitis in a 62-year-old woman with pyelonephritis, fever, and right flank pain. Several courses of antibiotics had been discontinued because of drug reactions. Rapid deterioration in the patient's status required intensive care unit (ICU) admission with supportive therapy. Her recovery was uneventful. **(a)** Coronal CT image shows the right perinephric fluid (white arrow) associated with the pyelonephritis and asymmetric fascial thickening involving the lateral conal and combined interfascial planes (black arrows) with direct extension into the extraperitoneal pelvic spaces (arrowhead). Note the asymmetric thickening of the involved right psoas major and iliacus muscles (*). **(b)** Axial CT image shows inflammatory changes involving the right lateral conal plane and the combined interfascial plane (white arrows). Note bilateral inflammatory extension in the retroperitoneal fat along the iliac vessels (black arrows). **(c)** Axial CT image shows bilateral retroperitoneal tracking of inflammation into the pelvis along the combined interfascial plane (white arrows) into the presacral space (black arrow).

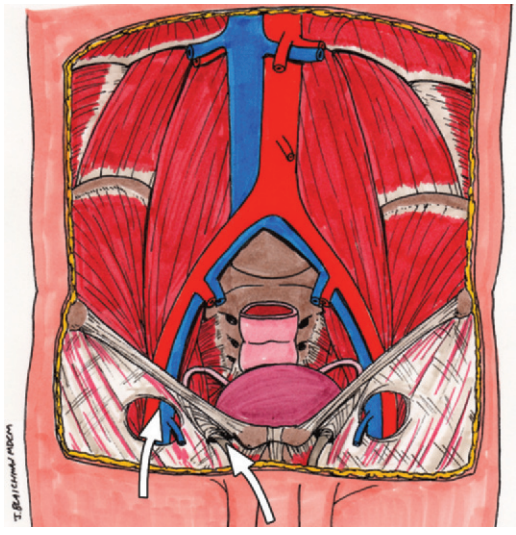

Figure 5. Coronal drawing of the pelvis shows potential direct communication routes into the extraperitoneal and retroperitoneal spaces through the femoral sheath and femoral vessels as well as the inguinal canal (arrows).

the pancreas, anterior and descending colon, and portions of the duodenum. The posterior pararenal space lies posterior to the retrorenal space and contains mostly fat. Also in the retroperitoneum are the aorta and inferior vena cava, which lie behind the retromesenteric plane (31,32,34).

Retroperitoneal fasciitis can originate within retroperitoneal structures, such as the kidneys, ureters, pancreas, duodenum, and ascending or descending colon. In these cases, access to the remainder of the retroperitoneum can be made by means of direct spread (35–37). This is well seen at cross-sectional imaging such as in cases of pancreatitis, complicated pyelonephritis, bowel perforations and abscesses, or complicated diverticulitis (36) (Fig 4).

Access to the retroperitoneum can also be made through direct or indirect fascial communications. Inferiorly, these communications are with the extraperitoneal pelvic spaces, and their associated organs such as the bladder, uterus, and rectum (31,32,38). The extraperitoneal pelvic spaces include the prevesicular space, perivesicular space, perirectal space, and presacral space. These pelvic extraperitoneal spaces are themselves in communication with extra-abdominal spaces (Fig 5), which can also serve as pathways of spread for infection,

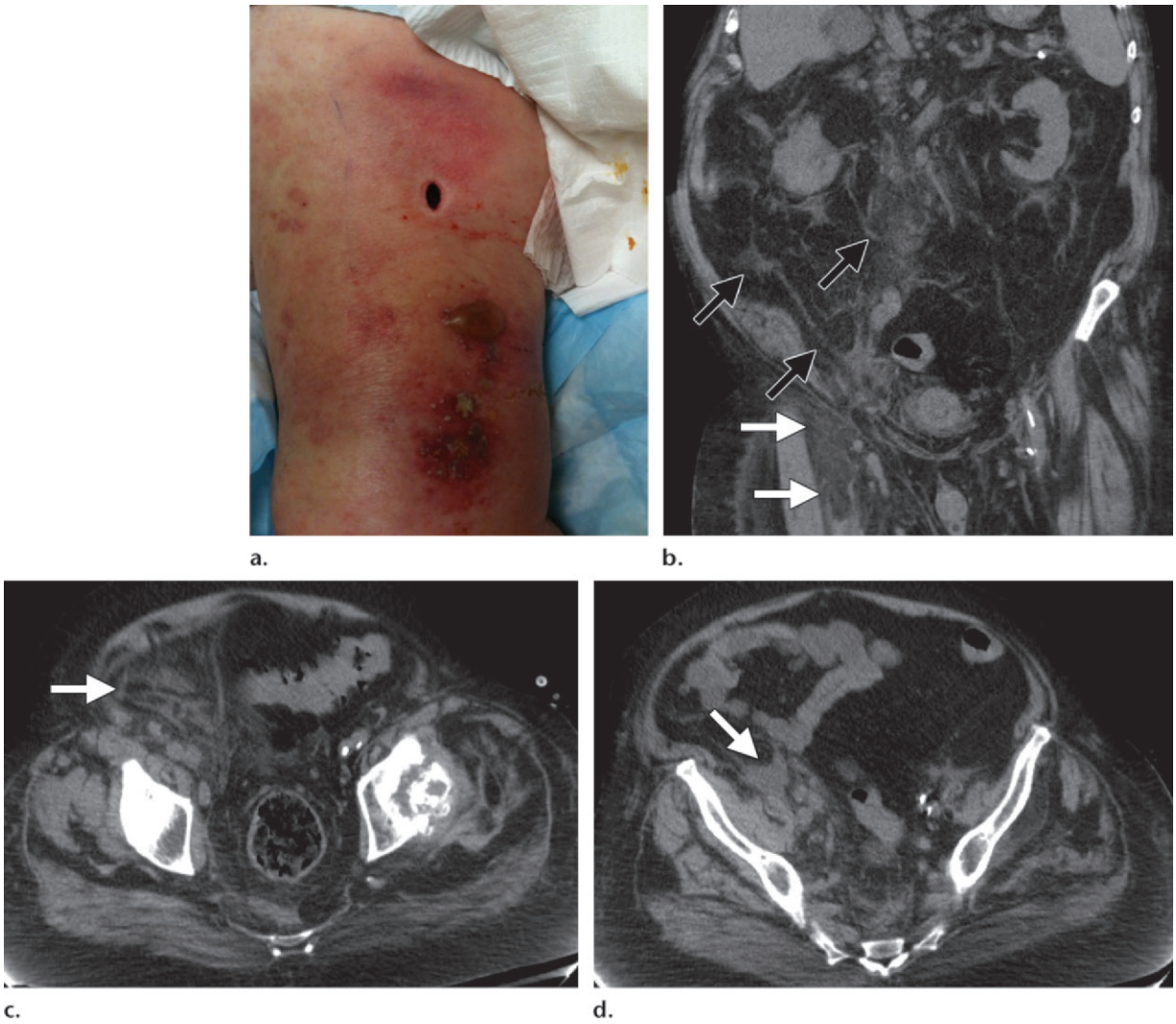

Figure 6. Necrotizing retroperitoneal fasciitis with direct spread from the thigh in a 73-year-old man who presented with severe right lower limb pain and swelling. Rapid deterioration in the patient's status led to his admission to the ICU, but his condition was refractory to supportive therapy, and he died without surgery. β-hemolytic group C *Streptococcus* was cultured. **(a)** Photograph of the swollen right thigh shows erythema, blistering, and mottling consistent with necrotizing fasciitis. **(b)** Coronal CT image shows fat stranding in the right anterior thigh (white arrows) with direct extension along the femoral vessels into the pelvis and along the right retroperitoneal fascial planes (black arrows). **(c)** Axial CT image shows fat stranding extending along the right external iliac vessels (arrow). **(d)** Axial CT image shows asymmetric thickening of the retroperitoneal combined interfascial plane (arrow).

such as via the inguinal canal or along the femoral vascular sheath space (31,37) (Fig 6). All other sources of spread, such as the perineum, do not directly communicate with the retroperitoneal space and are indirect pathways requiring compromise of fascial planes to allow access (Fig 7).

Imaging Features

CT is the first line of imaging in cases of suspected deep or retroperitoneal fascial infection. Emergency CT can lead to early diagnosis and provide evaluation of the extent of disease. For necrotizing soft-tissue infections, CT sensitivity has been described as 100% and specificity at 81% (39). Although early urgent CT may have an influential role in the diagnosis of retroperitoneal fasciitis, imaging should not be pursued if delay in diagnosis is outweighed by the need for immediate surgical management (3). Necrotizing fasciitis of the extremities, in particular the thigh, has been cited as the etiologic agent in many cases of necrotizing retroperitoneal fasciitis. An early staging CT scan should be ordered if the pain clinically extends to the pelvis or flank or close to the pelvic floor or inguinal regions. The process is usually extremely painful, which helps in determining the need to further extend imaging to the pelvis or abdomen (2,27,29).

Findings at CT include asymmetric fascial thickening and enhancement, muscular edema, fat stranding, fluid collections, and abscess formation. Asymmetric involvement of the retroperitoneal fascial planes and deep tissues is an important clue to the diagnosis. Although gas tracking along fascial planes is considered a characteristic finding

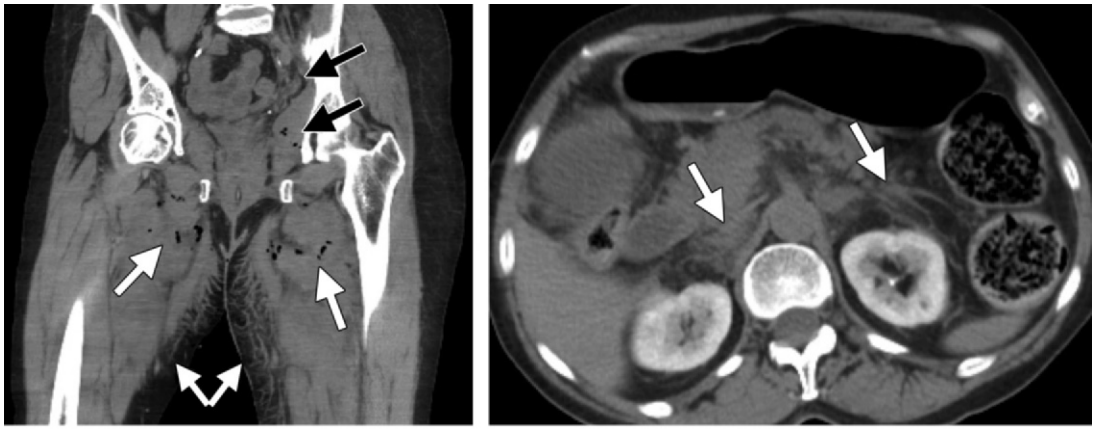

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Figure 7. Necrotizing retroperitoneal fasciitis with indirect spread from the thigh. A 68-year-old woman presented with severe acute bilateral medial thigh pain after insertion of a transobturator mesh (implant for urinary incontinence). Rapid deterioration with septic shock required her admission to the ICU. Despite aggressive surgical débridement, she did not survive the postsurgical period. β-hemolytic group A *Streptococcus* was cultured. **(a)** Coronal CT image shows gas locules, fascial thickening, and edema of bilateral adductor muscles with adjacent subcutaneous stranding (white arrows). There is extension of gas locules, fascial thickening, and stranding through the obturator canal involving the extraperitoneal pelvic space and the obturator internus muscle (black arrows). In a nonsurgical case, this would indicate transgression of fascial planes consistent with necrotizing fasciitis; however, the pathway of spread may be related to the recent surgical procedure. **(b)** Axial CT image shows extension into the retroperitoneum with asymmetric stranding along the retromesenteric plane (arrows), greater on the right side than on the left.

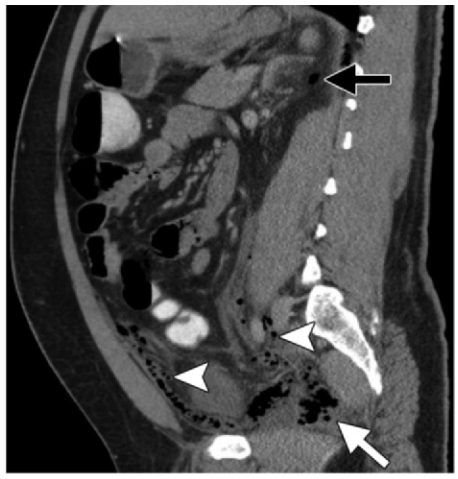
of retroperitoneal fasciitis, it is present only in a minority of cases in our experience (40) (Fig 8). Nonfocal collections along fascial lines and asymmetric involvement are suggestive of the diagnosis. Focal collections may indicate abscess or hematoma formation. An additional benefit of CT is that the examination may provide information on the underlying source of the retroperitoneal fasciitis—for example, identification of a perforated viscus or acute complicated diverticulitis. Fasciitis may originate in the retroperitoneum or track into the retroperitoneum through direct or indirect pathways. Immediate recognition and aggressive management may be life-saving.

The necrotizing form of retroperitoneal fasciitis is characterized by the transgression of fascial planes (indirect tracking) and a more aggressive rapid clinical course. Additional findings include evidence of vascular thrombosis, hemorrhage, multifocal abscess formation, and frankly necrotic organs (Fig 9). In aggressive forms, CT findings associated with septic shock and hypotension may be seen.

Ultrasonography (US) is considered to be of limited value in the diagnosis of retroperitoneal fasciitis. US may be used to identify focal fluid collections or abscess formation but is unlikely to be able to provide early diagnosis or appropriate staging of the extent of the disease (30,41,42). US has been found to be useful in the evaluation of integument necrotizing fasciitis, in looking for subcutaneous edema, fascial thickening, and fluid collections, particularly in the pediatric population (42).

Plain radiography is of limited value in assessing for retroperitoneal fasciitis. Gas may be identified along fascial planes, but this is nonspecific in isolation from the clinical context and does not adequately define the extent of the disease.

There is a paucity of literature examining the utility of magnetic resonance (MR) imaging in retroperitoneal fasciitis. The descriptions of MR imaging findings described later in this article are from the literature on integument necrotizing fasciitis. We would anticipate similar findings in the deep muscles and fascia of the retroperitoneum. Although CT is the better modality for detecting gas and its extent, the absence of gas does not exclude necrotizing fasciitis and, as stated, was present in only a minority of our cases. MR imaging, on the other hand, offers greater soft-tissue contrast because it is more sensitive in depicting the abnormal deep fascia and edema and their extent (43). MR imaging findings described in necrotizing fasciitis of the extremities include T2-weighted images with high signal intensity of the deep fascia and within the muscles (2). Deep fascial enhancement and thickening seen on gadolinium-enhanced images, as well as fluid collections involving deep fascial tissues, would also be findings consistent with necrotizing fasciitis (43). Gas bubbles, if present, will appear as a signal void on both T1- and T2-weighted images. These findings, when present, are reported to help differentiate necrotizing fasciitis from cellulitis, in which abnormal MR imaging findings are confined to the subcutaneous tissues and superficial fascia.



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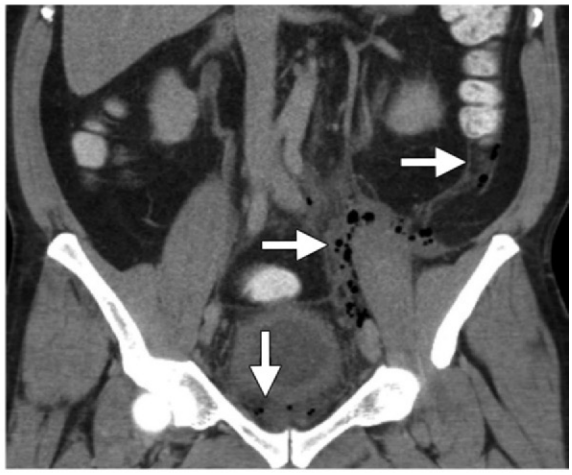

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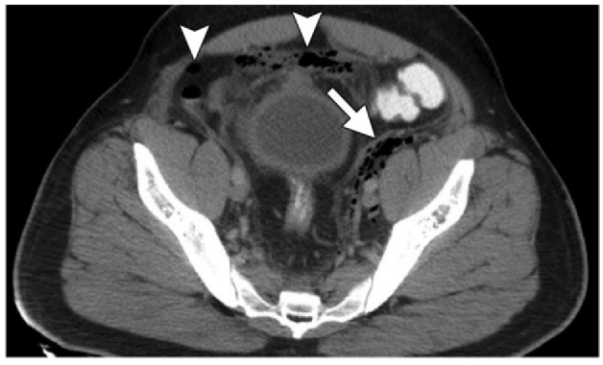

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Figure 8. Necrotizing retroperitoneal fasciitis tracking indirectly from the perineum. A 48-year-old man who underwent an incision and drainage of a perianal abscess returned 5 days later with necrotizing fasciitis of the perineum with extensive spread into the intraperiteoneal and retroperitoneal spaces, including abscess formation in the left ischioanal fossa, left iliopsoas muscle, and left subphrenic space. His treatment included multiple surgical débridement procedures with an extended ICU stay. *E coli* and *Streptococcus anginosus* were cultured. Gram-positive bacilli were identified at microscopy but not cultured. **(a)** Sagittal CT image shows an abscess with air at the level of the anus (white arrow) transgressing fascial planes to enter the peritoneum (black arrow) and the extraperitoneal pelvic spaces (arrowheads), findings in keeping with necrotizing retroperitoneal fasciitis. **(b)** Sagittal CT image shows tracking of air along the retroperitoneal fascial planes (arrows). **(c)** Coronal CT image shows fluid and air tracking asymmetrically along the left extraperitoneal pelvic spaces to the retromesenteric planes (arrows). **(d)** Axial CT image shows extension of the perianal abscess into the pelvis and through the fascial planes into the extraperitoneal pelvic spaces (arrows). **(e)** Axial CT image shows extension of the perianal abscess into the pelvis and through the fascial planes to the extraperitoneal pelvic spaces (arrow) and peritoneum (arrowheads).

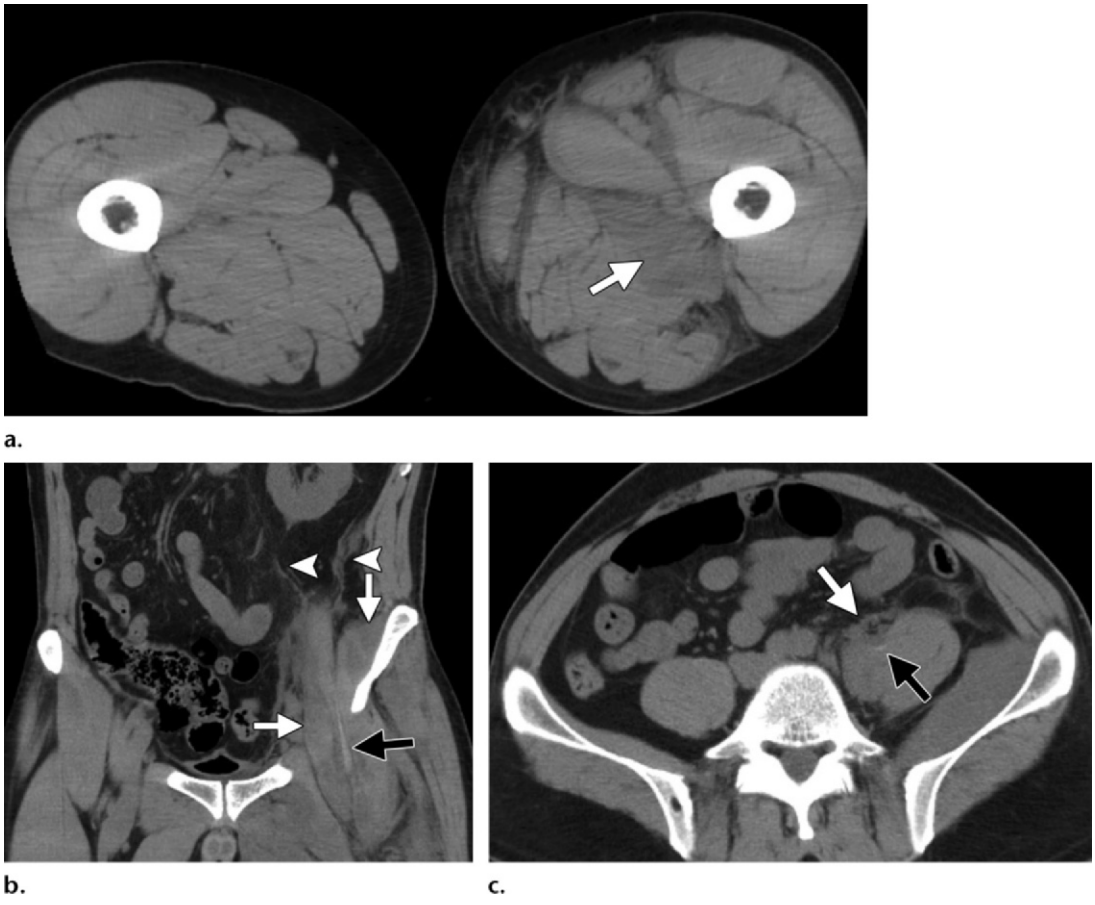


Figure 9. Necrotizing retroperitoneal fasciitis tracking indirectly from the left thigh in a 42-year-old man who presented with malaise, severe left thigh pain, and skin discoloration on the left thigh and left hemiscrotum. His status rapidly deteriorated, and he required ICU admission with supportive therapy. After several procedures involving aggressive surgical débridement of the left thigh and left aspect of the retroperitoneum, he recovered uneventfully. β-hemolytic group A *Streptococcus* was cultured in specimens from the thigh and retroperitoneum. **(a)** Axial unenhanced CT image of the left thigh shows asymmetric intermuscular fascial thickening and edema (arrow) along with subcutaneous stranding. **(b)** Coronal CT image shows asymmetric (left) heterogeneity and thickening involving the psoas major and iliacus muscles (white arrows). A linear region of high attenuation along the muscles represents a thrombosed vessel (black arrow). There is also asymmetric thickening of the lateral conal plane and retromesenteric planes (arrowheads) indicating transgression of fascial planes consistent with a necrotizing form of retroperitoneal fasciitis. **(c)** Axial CT image shows asymmetric thickening of the left psoas muscle (white arrow), with surrounding retroperitoneal fluid and stranding. There is a small focus of hyperattenuation consistent with the known thrombosed blood vessel (black arrow).

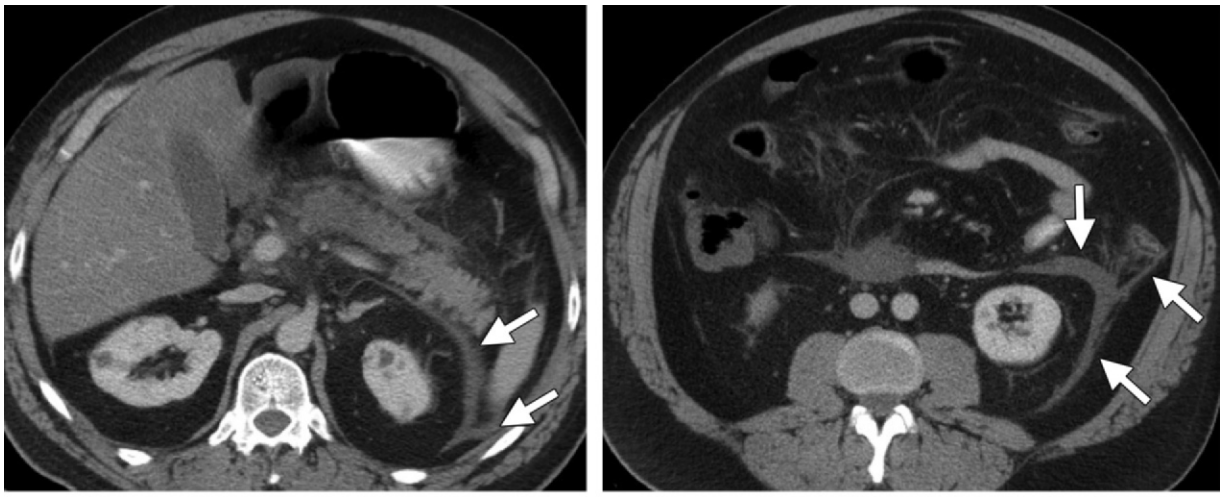
In their retrospective review of 30 patients with necrotizing versus nonnecrotizing infectious fasciitis of the extremity, Kim et al (44) found that MR imaging may be helpful in distinguishing between these two entities. On fat-saturated T2-weighted MR images, the necrotizing group had a higher frequency of deep fascial thickening greater than 3 mm and of abnormal low signal intensity (presumably air) in the deep fascia. On contrast material–enhanced T1-weighted MR images, the necrotizing group showed a higher frequency of focal or diffuse nonenhancement of the abnormal deep fascia. In addition, the necrotizing group showed more extensive involvement of the deep fascia and involvement of more than three compartments.

There is overlap, however, between the MR imaging findings of infected edematous nonnecrotic deep tissue and necrotic tissue, raising concerns about the potential oversensitivity of MR imaging for the diagnosis of necrotizing fasciitis (45). The extent of infection may be overestimated by MR imaging (43). The relatively limited access, longer duration of examination, and potential oversensitivity of MR imaging likely account for the lack of reported information for the diagnosis of retroperitoneal fasciitis. CT remains the first line of imaging.

Differential Diagnosis

The diagnosis of retroperitoneal fasciitis can be challenging because of the nonspecific signs and potential rapid clinical deterioration, which may limit the timeline and options available for diagnosis. Although clinical history and laboratory and imaging findings are helpful in making the diagnosis of acute pancreatitis, diverticulitis, retrocecal

Figure 10. Acute pancreatitis mimicking retroperitoneal fasciitis in a 50-year-old man. **(a)** Axial CT image shows peripancreatic stranding and heterogeneity of the pancreatic body in keeping with the clinical and biochemical diagnosis of acute pancreatitis. The inflammatory fluid tracking asymmetrically along the left retromesenteric plane and lateral conal plane (arrows) is noteworthy. **(b)** Axial CT image shows asymmetric inflammatory thickening of the retromesenteric plane, retrorenal plane, and lateral conal plane (arrows), a finding associated with the known clinical and biochemical diagnosis of acute pancreatitis.



a. b.

appendicitis, or pyelonephritis, it remains challenging to determine if associated retroperitoneal findings are reactive inflammatory changes or represent retroperitoneal fasciitis with infection. A number of conditions may mimic retroperitoneal fasciitis, including infected or inflammatory changes of the uroepithelium with associated periureteric stranding, thickening, and reactive fascial and fat stranding. Positive urinalysis and urine cultures may also be helpful in distinguishing a genitourinary infection. Acute pancreatitis may be distinguished by the clinical setting of gallstones or alcohol abuse, elevated amylase, and inflammation centered around the pancreas (Fig 10).

Gas tracking along the retroperitoneal fascial spaces may also be associated with viscus perforation, postsurgical changes (Fig 11), tracking from benign subcutaneous emphysema, or other causes of benign intra-abdominal gas (Fig 12) (9,46).

Intramuscular hemorrhage and abscess formation may be associated with retroperitoneal fasciitis, in particular the necrotizing form; however, these remain nonspecific signs and may be associated with multiple entities, including trauma and blood clotting disorders (Fig 13).

Third spacing can manifest with nonspecific fluid collections in the retroperitoneal and interfacial planes. In patients who are no longer mobile, the fluid may fall dependently, thus mimicking the asymmetric involvement typically associated with retroperitoneal fasciitis. The clue to the relatively benign diagnosis of third spacing would be associated subcutaneous edema or pleural effusions. Nonnecrotizing retroperitoneal fasciitis is particularly challenging to distinguish from third spacing as we may identify only fascial edema, thickening, and fluid collections without fat stranding.

Because the findings in retroperitoneal fasciitis are nonspecific, it is important to maintain a high index of suspicion for retroperitoneal fasciitis when there is inordinate pain and rapid clinical deterioration in a patient. Close or ongoing clinical communication between the referring and reporting physician may be helpful in identifying this high-risk patient group.

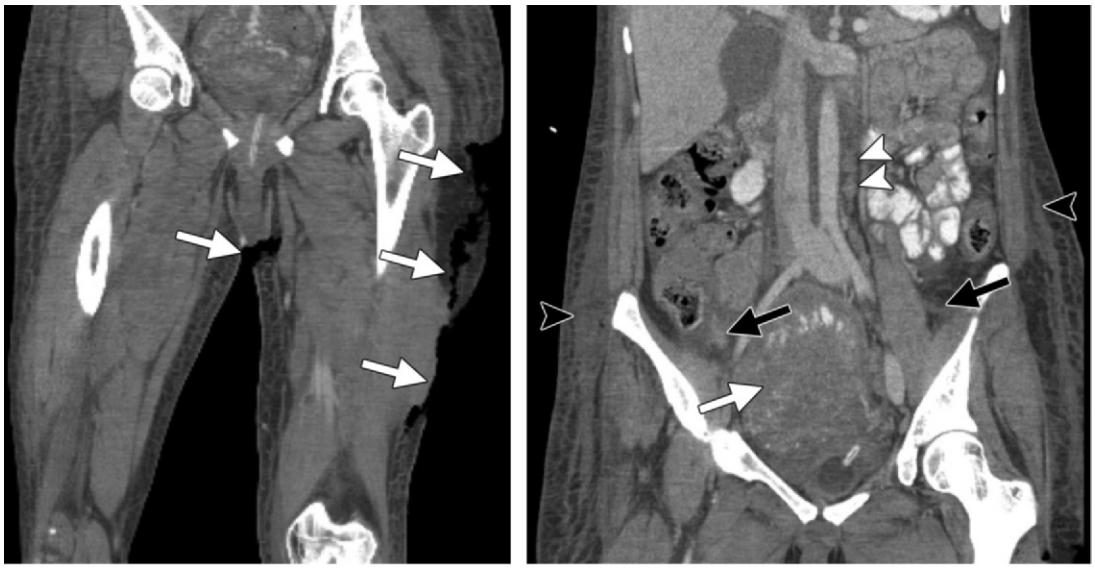
Treatment and Outcome

Treatment should be directed both at the origin of the infection and at the retroperitoneal fasciitis. Early aggressive empirical antibiotic administration should be broadly directed until the involved microbial organisms and their antibiotic susceptibility profiles are identified.

Imaging-guided aspiration in the most accessible area may be valuable if the diagnosis is uncertain or to obtain a sample for culture and sensitivity. In cases originating from the lower extremity, thigh, or groin, we have found it useful to sample the edematous soft tissue under US guidance. A sample may also be obtained when focal collections or abscesses are present. There is an emerging role for interventional radiology and percutaneous drainage of focal abscess collections; this procedure may be used in conjunction with conventional aggressive therapy. Therapeutic decisions should be reached on the basis of a multidisciplinary approach, with the indication for percutaneous drainage evaluated on a case-by-case basis (11,47).

The outcome will in part relate to the inciting event, comorbidities, and extent of disease

Figure 11. Necrotizing fasciitis of the left lower extremity. A 30-year-old woman with a term pregnancy was transferred for emergency cesarean section on the basis of fetal distress. She had fever, chills, and known cellulitis treated with antibiotics. After surgery, the findings on her thigh progressed from erythema to blistering and necrosis with rapid spread to the trunk, flank, buttocks, and left leg. She underwent several aggressive surgical débridement procedures of the thigh, and the cesarean section wound was reopened. The abdominal tissue appeared healthy, and culture was negative. Postsurgical CT findings were difficult to distinguish from retroperitoneal fasciitis. β-hemolytic group A *Streptococcus* was cultured from thigh tissue. The patient's recovery was uneventful. **(a)** Coronal CT image shows defects from surgical débridement of the left thigh (arrows), including thickening of muscles and loss of interfascial planes, with fascial thickening near the groin. **(b)** Coronal CT image shows the postpartum uterus (white arrow), intraperitoneal and retroperitoneal fluid and stranding (black arrows), small para-aortic reactive nodes (white arrowheads), and extensive anasarca in the subcutaneous tissues (black arrowheads). **(c)** Axial CT image shows a small volume of free fluid (*), gas locules (white arrow), and anasarca (black arrows), findings that were considered nonspecific.



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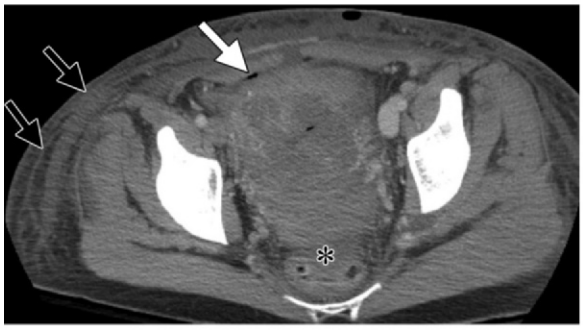


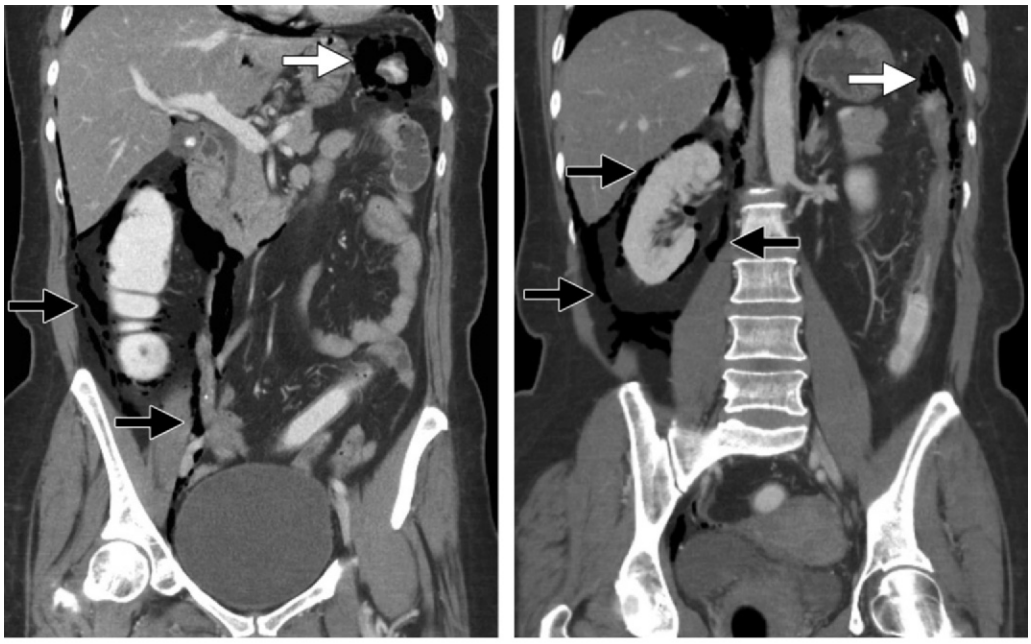
c.

at the time of diagnosis. Patients who develop retroperitoneal fasciitis commonly have considerable comorbidities such as immunosuppression or malignancy, which may contribute to the high mortality rate. In the necrotizing form of retroperitoneal fasciitis, early and repeated surgical débridement of the affected tissues in conjunction with aggressive administration of antibiotics are the key treatment elements (5,48). In some institutions, débridement protocols recommend iterative débridement every 24–48 hours until the infection is under control (7). Unfortunately, curative resection is not always feasible (5). Surgical fasciotomy and necrectomy may be required to relieve compartment syndrome.

There are reports of successful treatment of the nonnecrotizing form of retroperitoneal fasciitis with conservative supportive therapies and antibiotics (23). Necrotizing retroperitoneal fasciitis is associated with a higher mortality and has been considered an almost uniformly lethal infection with survival reported in only a few cases (5,25,26,48). In contrast, Mokoena et al (1) reported a remarkable 20% mortality in a group of 10 patients. They attributed this to the use of planned repeated laparotomy and débridement in addition to aggressive antibiotic and advanced supportive care measures for the critically ill. In our own institution, early aggressive therapy combined with surgical débridement has led to a survival rate approaching 50%. The surviving group was on average younger with less comorbidity and was identified at an earlier stage of infection than those who subsequently did not survive. This suggests that newer protocols with aggressive supportive intensive care, antibiotics, and surgery may contribute to improved survival of affected patients.

Conclusion

Retroperitoneal fasciitis is a rare but potentially lethal infection. Early diagnosis is crucial and is usually made when there is a high degree of



a. b.

Figure 12. Necrotizing retroperitoneal fasciitis mimicry in a 65-year-old woman with benign pneumatosis that tracked into the peritoneum and retroperitoneum. **(a)** Coronal CT image shows benign pneumatosis (white arrow) and air tracking along retroperitoneal planes (black arrows). **(b)** Coronal CT image shows benign intestinal pneumatosis (white arrow) and air tracking along retroperitoneal planes (black arrows).

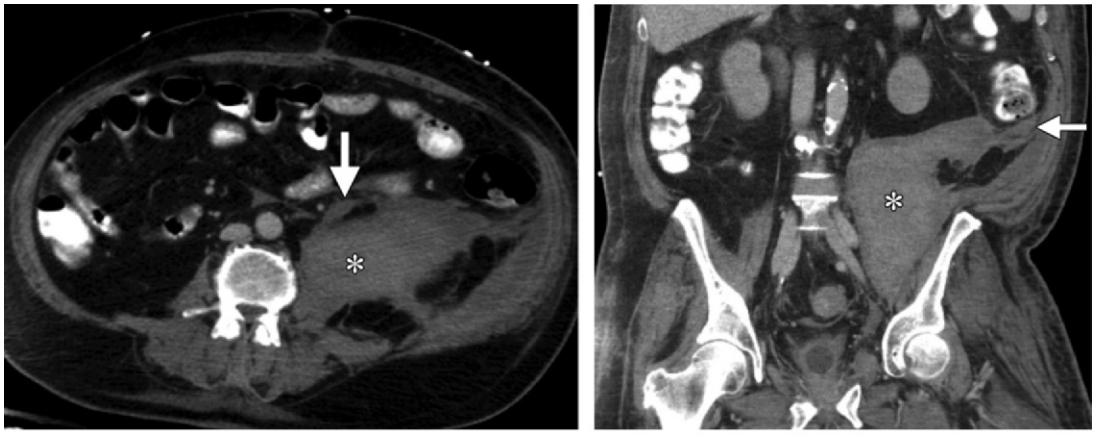


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Figure 13. Necrotizing retroperitoneal fasciitis mimicry in a 53-year-old man with left asymmetric thickening of the combined interfascial plane due to retroperitoneal hemorrhage. **(a)** Axial CT image shows left psoas hemorrhage (*) and asymmetric thickening of the combined interfascial plane (arrow). **(b)** Coronal image shows left psoas hemorrhage (*) and thickening of the lateral conal plane (arrow).

clinical suspicion combined with imaging findings leading to early surgical intervention. CT can play a crucial role in depicting early findings, assessing extent of disease to help determine the best surgical approach, identifying the primary source, and evaluating treatment response. Awareness of the spectrum of CT findings in retroperitoneal fasciitis is crucial for both diagnosis and guiding management.

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