CHAPTER

24 Renal and Perirenal Abscesses

Neha D. Nanda • Louise M. Dembry

Bacterial infections of the kidney and perinephric space include a spectrum of pathologic conditions that can be divided into intrarenal and perirenal abscesses. Both conditions are suppurative infections localized either within the parenchyma of the kidney (intrarenal abscess, i.e., renal cortical abscess and corticomedullary abscess) or within the perirenal fascia external to the kidney capsule (perinephric abscess), and each can be identified by specific diagnostic techniques. The incidence of intrarenal and perirenal abscesses ranges from one to 10 cases per 10,000 hospital admissions. In the preantibiotic era, most cases were caused by hematogenous seeding from distant foci of infection and were predominantly in young males without an antecedent history of renal disease. Currently, most cases occur as a complication of urinary tract infection and affect males and females with equal frequency. The incidence increases with age and if an abnormality of the genitourinary tract exists. This chapter covers only the more common types of these renal and perirenal infections.

INTRARENAL ABSCESS

Renal Cortical Abscess (Renal Carbuncle)

Etiology

A renal carbuncle (from the Latin, carbunculus, or "little coal") is a circumscribed, multilocular abscess of the renal parenchyma, which forms from a coalescence of multiple cortical microabscesses (Fig. 24.1). It is most commonly caused by staphylococci (Staphylococcus aureus) and is the result of metastatic spread from a primary focus of infection elsewhere in the body, most commonly the skin. Renal carbuncles were first described by Israel in 1905 in a presentation before the Free Society of Berlin Surgeons.[1] Although numerous reports and reviews[2–8] have been published since Israel's initial description, the total number of reported cases of renal carbuncle remains relatively small.

Pathogenesis

A renal cortical abscess results from a primary focus of infection elsewhere in the body. Common primary foci are cutaneous carbuncle, furunculosis, cellulitis, paronychia, osteomyelitis, endovascular infection, and infection of the respiratory tract. Important predisposing conditions that increase the risk of bacteremia and hematogenous spread are injection drug use, hemodialysis, and diabetes mellitus. S. aureus is the most common causative agent (90%) and infects the cortex of the kidney by hematogenous dissemination from the primary focus, often resulting in several interconnecting furuncles or microabscesses. Coalescence may occur with progression of the infection to a lesion consisting of a fluid-filled mass with a relatively thick wall. Rarely, the process may extend to the periphery of the renal cortex and rupture through the capsule, leading to formation of a perinephric abscess. The majority of renal cortical abscesses are unilateral (97%) single lesions (77%) occurring in the right kidney (63%), and are not associated with perinephric abscesses (90%). The reason for unilateral localization is not clear, although diminished resistance of the kidney resulting from previous disease or injury, including trauma, has been cited as a predisposing factor.[9] Infrequently, ascending infection causes a renal cortical abscess.[10,11] Because the interval between the original staphylococcal infection and the onset of clinical symptoms of a renal cortical abscess may vary from a few days to many months (average time of approximately 7 weeks),[9] the primary focus of infection may have healed and is not apparent in one third of affected patients.[5,7]

Clinical Features

Renal cortical abscesses are three times more common in males than females. The disease occurs at all ages but is most common between the second and the fourth decades of life.[9] The clinical picture of a renal cortical abscess is nonspecific. Most patients have chills, fever, and abdominal or back pain.[5,7,9] Some may have a palpable flank mass. Others present with a clinical picture of fever of undetermined origin, with few or no localizing signs.[12] Most patients have no urinary symptoms[9] because the abscess occupies a circumscribed area within the parenchyma of the kidney, which may not communicate with the excretory passages.

Physical examination often reveals tenderness in or near the region of the kidney. Pain on fist percussion of the

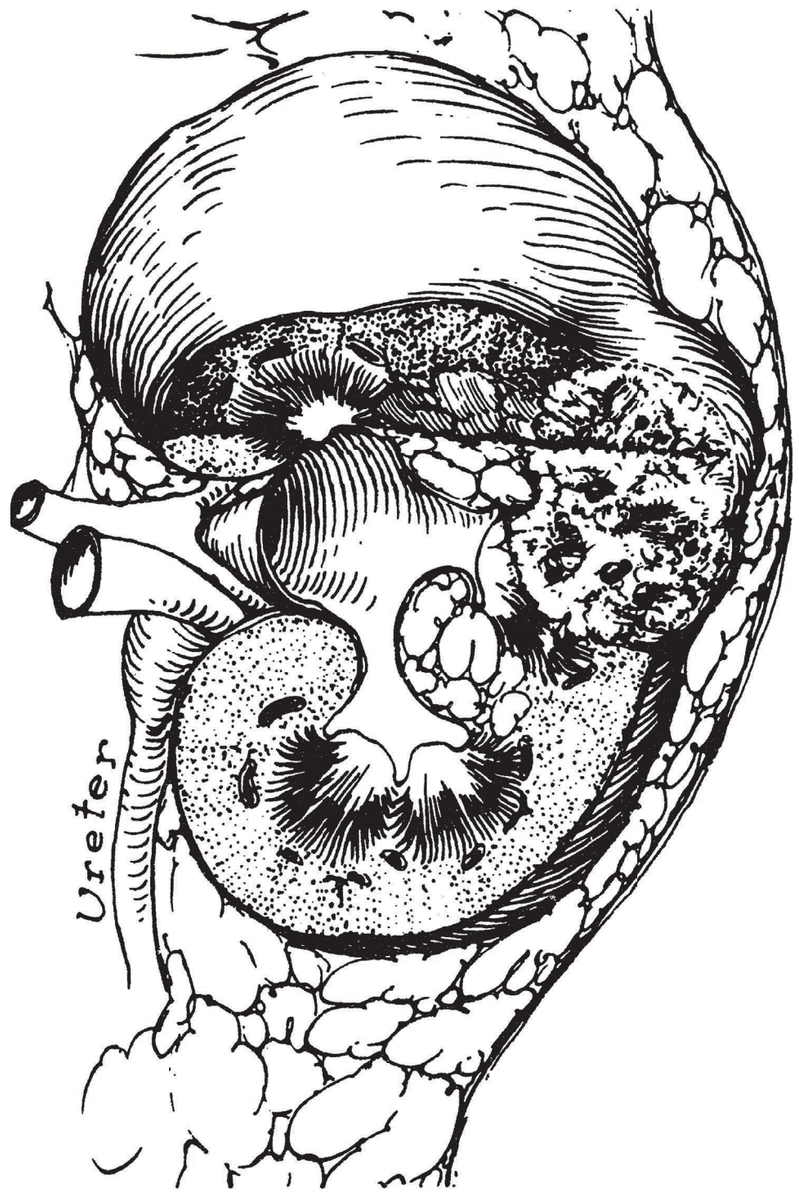


FIGURE 24.1 Diagram of the pathogenesis of a staphylococcal renal carbuncle. (From Andriole VT. Renal carbuncle. *Medical Grand Rounds*. 1983;2:259, with permission.)

costovertebral angle is the most constant physical finding, often accompanied by moderate muscle rigidity in the upper abdominal and lumbar muscles. A flank mass or a bulge in the lumbar region, with loss of the natural concave lumbar outline, may be present. Examination of the chest on the affected side may be abnormal, with decreased respiratory excursion, tenderness over the lower ribs, dullness, diminished breath sounds, increased fremitus, or rales.

Basic laboratory data are variable. Peripheral white blood cell counts are moderately elevated in 95% of patients.[9] The urinalysis usually presents no pathognomonic findings. Proteinuria, pyuria, or microscopic hematuria are usually present and a Gram stain of the urine will demonstrate the pathogen if the abscess communicates with the collecting system of the kidney. However, negative urinalyses are seen in most patients and blood cultures are usually negative.[9]

Diagnosis

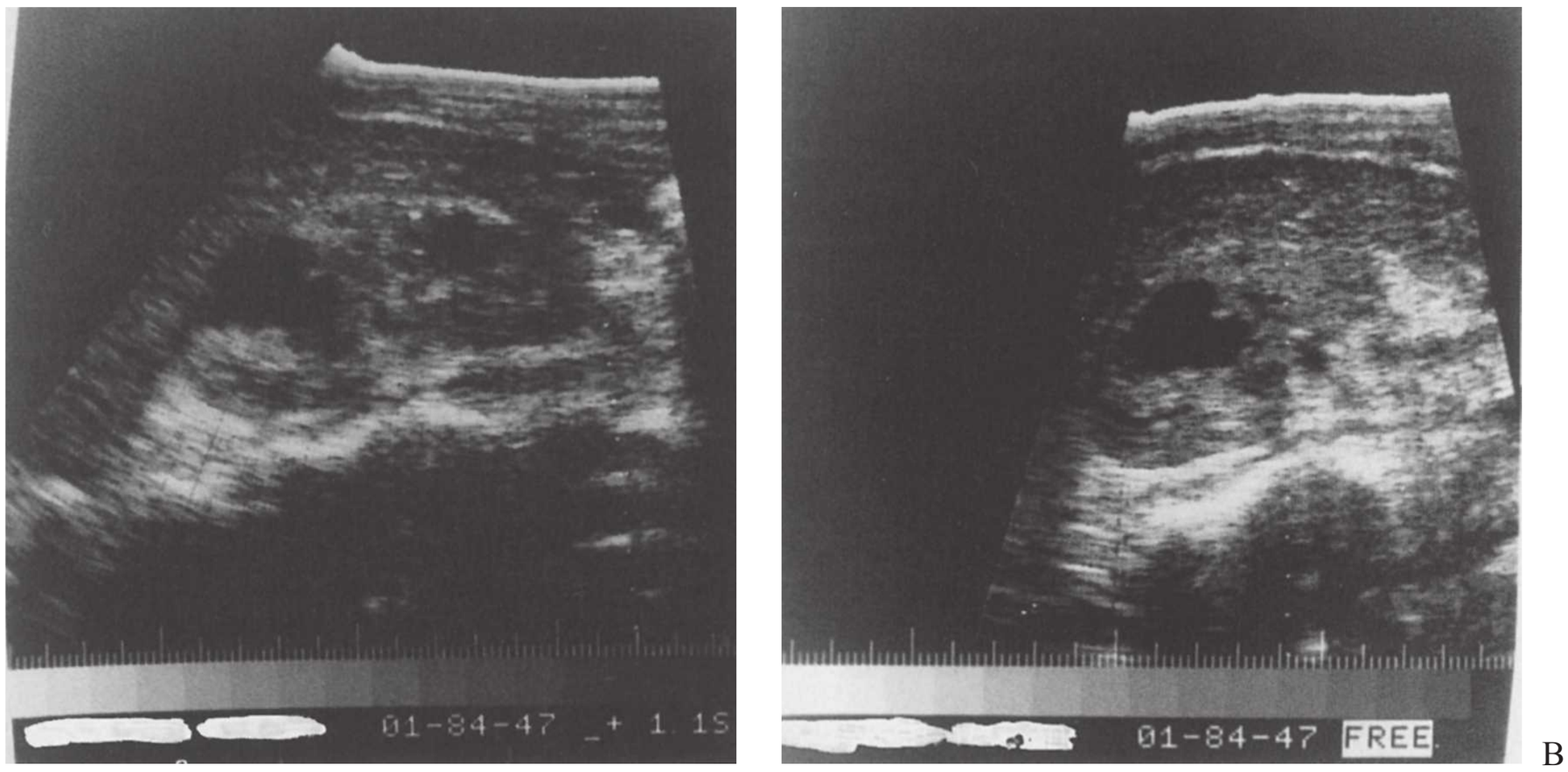
Renal cortical abscesses must be differentiated from other space-occupying lesions in the kidney. Renal tumors, cysts, intrarenal abscesses caused by aerobic gram-negative bacilli, and perinephric abscesses can mimic renal cortical abscesses. In the past, surgical exploration was performed to differentiate the renal mass from a carcinoma. The clinical presentation of a renal cortical abscess is nonspecific and not helpful in differentiating this disease from a renal tumor or perinephric abscess. Chills, fever, malaise, and back pain may be seen in each. A renal cortical abscess on the anterior surface of the kidney may produce abdominal symptoms and lead to an erroneous diagnosis of an intra-abdominal process. Renal cortical abscesses may also be confused with abscesses of the renal medulla, particularly in children.[7,9–11,13–15] Radiologic techniques can define the character of the renal mass and establish the correct diagnosis.[16–25]

In intravenous pyelograms, a renal cortical abscess appears as a mass of diminished density, frequently associated with distortion of the calyces, infundibulum, and renal outline. An abscess that extends to the periphery of the renal cortex may produce sufficient edema of the renal capsule to obliterate a segment of the perirenal fat shadow. However, there is no displacement of the kidney, as is frequently seen with a perinephric abscess. Thus, an abnormal intravenous pyelogram that demonstrates an intrinsic mass with calyceal distortion, but without displacement of the kidney in a patient with sterile urine, suggests a diagnosis of renal cortical abscess or tumor.

Ultrasonography has been extremely helpful in establishing the diagnosis of renal cortical abscess.[9] Renal ultrasonography is easily available, cost effective, and there is no exposure to radiation or contrast. Renal ultrasonography can provide morphologic detail of the kidneys; is capable of identifying cystic lesions, tumorlike masses, or abscess cavities; and can show the size and location of the lesion. Early in the development of a renal cortical abscess, however, internal echoes may be present, giving the appearance of a solid or semisolid mass. Because these findings are compatible with either a renal cortical abscess or tumor, computed tomography (CT) may be performed to define the lesion further and to establish the correct diagnosis.[26,27] Another disadvantage of renal ultrasonography is its dependence on the operator and the body habitus of the patient.[27] After coalescence, an abscess can be identified by ultrasound as a fluid-filled mass with a relatively thick wall (Fig. 24.2). Ultrasonography also can be used to guide aspiration of the lesion and to follow its resolution with antibiotic treatment[7,24,28] (Fig. 24.3).

CT is the most accurate noninvasive technique currently in widespread use and permits detection of abscesses smaller than 2 cm.[29–31] Contrast-enhanced CT is useful if ultrasonography is negative or equivocal and allows for the detection of pathologic lesions in the renal cortex and medulla in early stages. CT is also useful as a guide to percutaneous aspiration of an abscess and to follow a known lesion. An abscess appears as a sharply demarcated low-density lesion on CT. The abscess does not enhance with contrast because of its avascular nature; however, the wall of the abscess enhances because of the presence of dilated and inflamed vessels.[28,30,31] The finding of gas in a low density mass is pathognomonic for an abscess.[30]

Magnetic resonance imaging (MRI) is another noninvasive technique that is as accurate as CT to diagnose renal abscess and define the extent of involvement. With an MRI there is no exposure to radiation and ionizing contrast.[27] MRI with gadolinium and contrast-enhanced CT scans have comparable sensitivity to detect renal abscesses. Noncontrast CT and renal ultrasonography are not as good to detect renal parenchymal pathology.[32] To differentiate between a renal



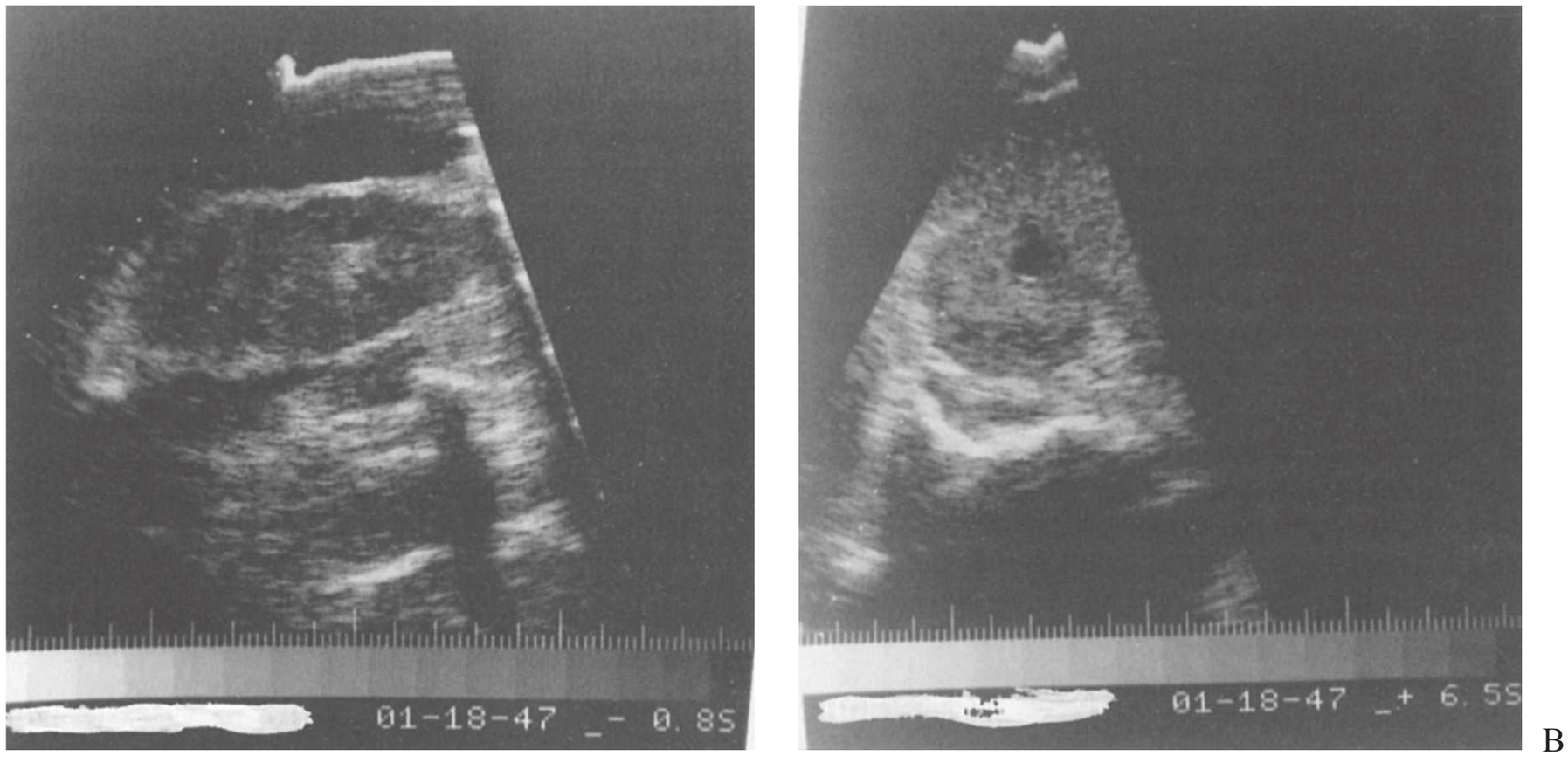
A B

FIGURE 24.2 Ultrasonogram of the right kidney on admission to the hospital. **A:** Longitudinal view, demonstrating two echolucent fluid-filled lesions. **B:** Transverse view, demonstrating fluid-filled masses with thickened margins. (From Andriole VT. Renal carbuncle. *Medical Grand Rounds*. 1983;2:259, with permission.)

malignancy and an isolated abscess in the kidney, MRI with diffusion-weighted images is helpful. The principle behind this is the diffusion of water molecules is reduced in the intracellular space compared to the extracellular space. Thus, highly cellular tumors may be more likely to have restricted diffusion than less cellular tumors/masses. This modality has been used extensively to characterize central nervous system lesions.[33] Several drawbacks to MRI are that it is expensive and time consuming to perform. Gadolinium use in patients with stage IV or V chronic kidney disease is also contraindicated.

Selective renal arteriography is an older modality used to differentiate renal cortical abscess from tumor. A renal cortical abscess can be identified angiographically as a mass that produces arcing, stretching, and attenuation of adjacent arteries, with the vessels located around the circumference rather than within the mass (Fig. 24.4). Early in the course, the rim around the abscess is poorly visualized, but arterial circulation to the periphery gradually increases with time so that a late study may identify a dense rim in the parenchymal phase. An untreated abscess may progress to a stage in which the rim is thick and poorly vascularized.

Renal and perirenal abscesses can be arteriographically distinguished from tumors because the major portion of an abscess is avascular whereas the wall of the abscess is hypervascular.



A B

FIGURE 24.3 Ultrasonogram of the right kidney after 4 weeks of antibiotic therapy (from the same patient as in Fig. 24.2). Longitudinal view **(A)** and transverse view **(B)** showing a decrease in the size of the fluid-filled echolucent lesions. (From Andriole VT. Renal carbuncle. *Medical Grand Rounds*. 1983;2:259, with permission.)

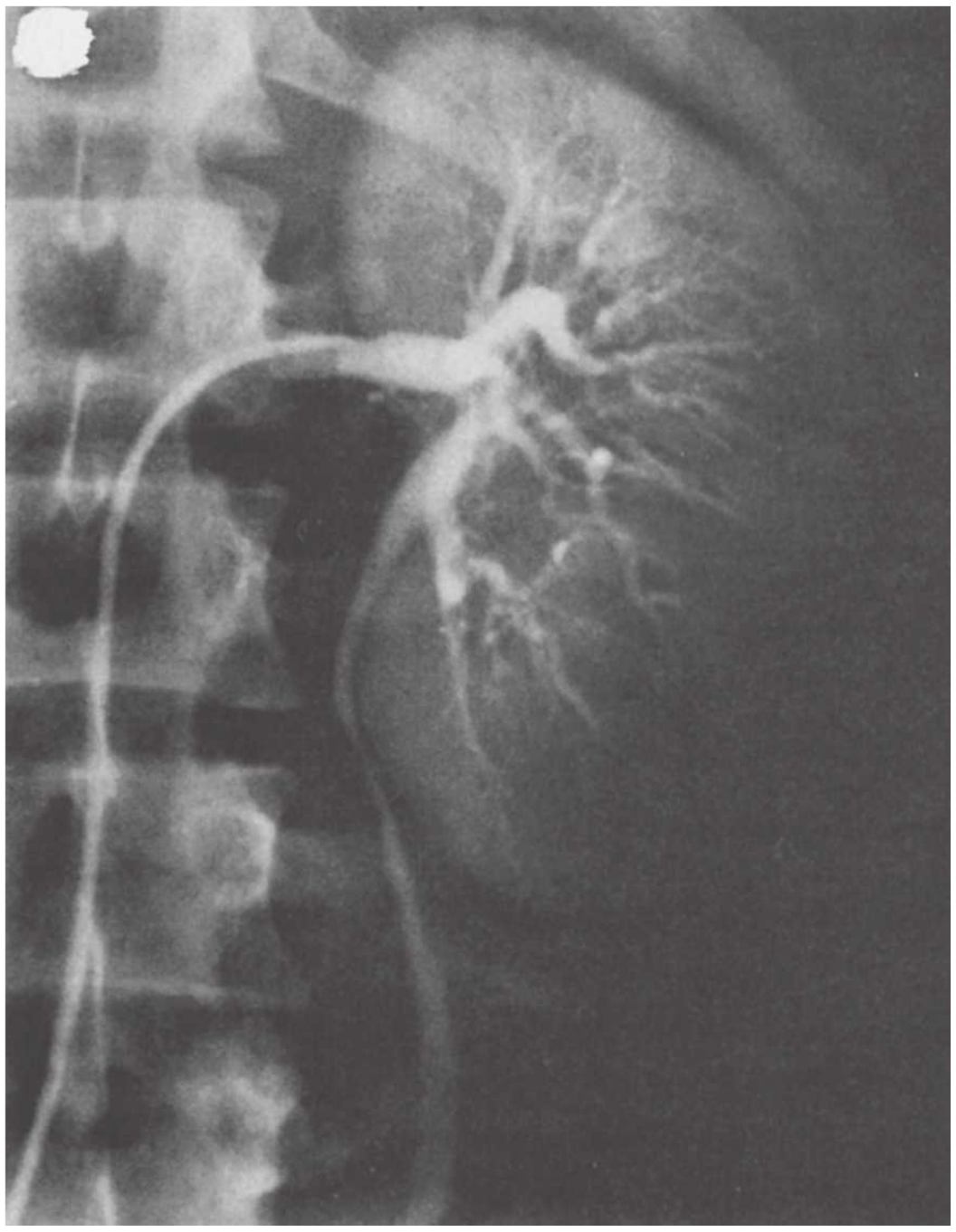


FIGURE 24.4 Arterial phase of left renal arteriogram. Peripheral vessels of the lower pole are attenuated and separated in comparison to normal vessels in the upper pole. No tumor vessels are present. (From Andriole VT. Renal carbuncle. *Medical Grand Rounds.* 1983;2:259, with permission.)

Renal carcinoma may be hypervascular or hypovascular (necrotic), but rarely both. In an abscess, the arteries retain their normal organization and branching pattern. Tumor neovascularity, in contrast, consists of abnormal vessels. Tumor vessels have no recognizable organization, may enlarge instead of taper as they course peripherally, and have an abnormal branching pattern.

Nuclear imaging was popular before CT and MRI became widely available. Renal scanning with gallium-67 (^{67}Ga) citrate (Fig. 24.5) also has been useful in localizing a renal abscess in adults.[17,28,34] A subtraction technique using ^{67}Ga citrate and technetium-99 (^{99}Tc) glucoheptonate can define the extent of perinephric involvement and eliminate any false-positive scans seen with gallium alone.[34] The latter may occur in patients with renal carcinoma, severe pyelonephritis without abscess formation, or ureteral obstruction. ^{111}In-labeled white cell scanning identifies a renal abscess but does not demonstrate renal carcinoma.

Noninvasive techniques such as ultrasound, CT, and MRI have reduced the need for intravenous pyelogram, selective angiography, and nuclear imaging to further define intrarenal masses.

Radiologic techniques can correctly establish a diagnosis of a renal cortical abscess only when this diagnosis is considered. Unfortunately, clinicians generally do not think of the diagnosis of renal cortical abscess early in its course. An average delay of 62 days before the correct diagnosis was established and proper treatment instituted has been reported.[9]

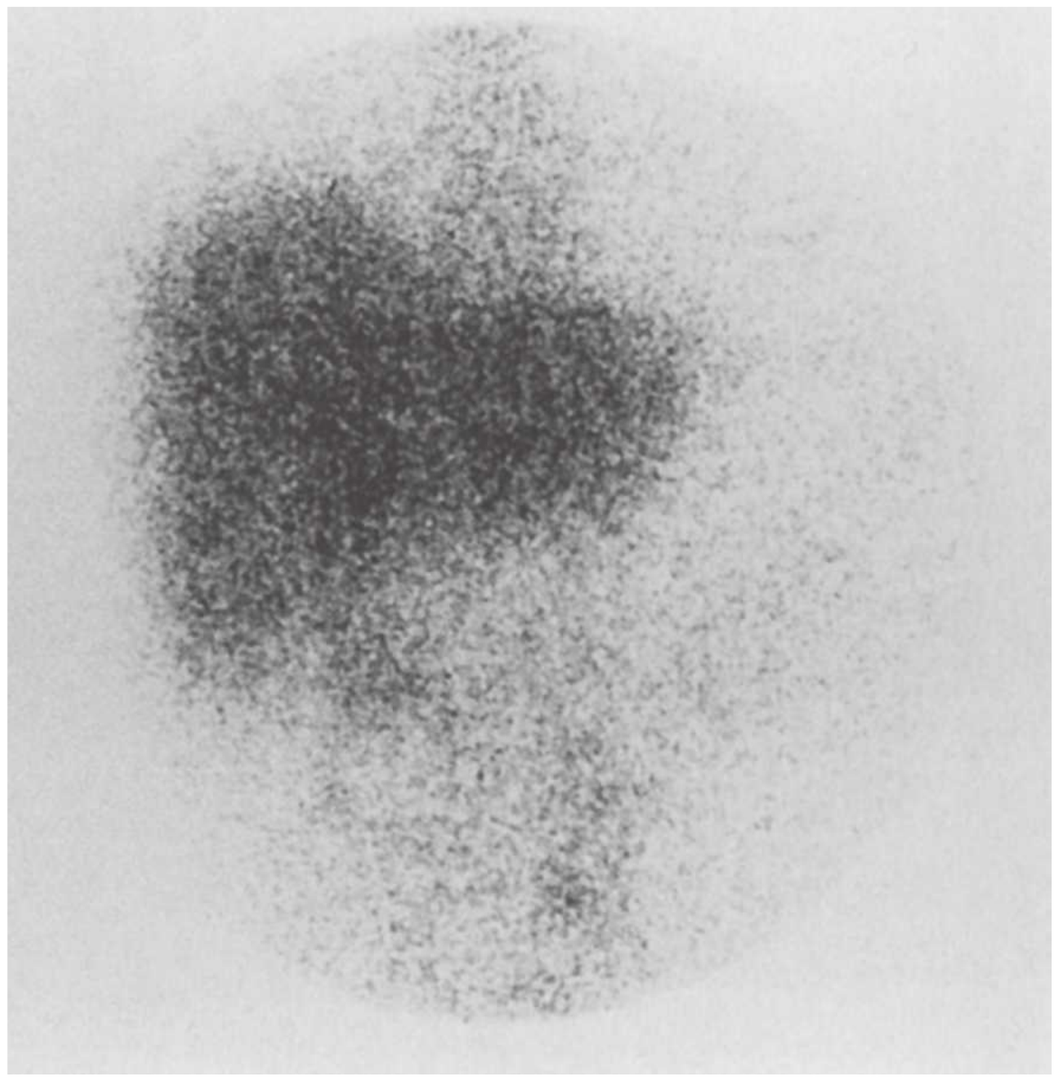


FIGURE 24.5 Radionuclide scan with ^{67}Ga citrate at 48 hours, showing abnormal uptake in the right upper quadrant, inferior to the liver and right in the area of the kidney. (From Andriole VT. Renal carbuncle. *Medical Grand Rounds.* 1983;2:259, with permission.)

Treatment

Historically, the treatment of a renal cortical abscess has been surgical and has varied with the condition of the patient.[8] However, because a renal cortical abscess is usually hematogenous in origin, and caused by S. aureus, it often responds to antistaphylococcal antimicrobial therapy alone, thus obviating the need for surgical intervention.[9]

If the diagnosis of renal cortical abscess is suspected from the history, physical findings, and renal ultrasonography (abscess localized to the renal parenchyma) or CT, and gram-positive cocci or no bacteria are seen on microscopic examination of the urine, antimicrobial therapy should be directed against S. aureus. Choice of empiric antistaphylococcal therapy depends on the susceptibility patterns of S. aureus in the community. If methicillin-sensitive S. aureus (MSSA) is prevalent in the community, oxacillin or nafcillin 1 to 2 grams intravenously (IV) every 4 to 6 hours is appropriate initial therapy. If a history of nonanaphylactic penicillin allergy (i.e., rash) is present, cefazolin (2 grams every 8 hours) is an alternative. Patients with severe immediate penicillin allergy may manifest cross-reacting allergy when a cephalosporin is administered and should receive vancomycin (1 gram every 12 hours) instead. If the prevalance of community-associated methicillin-resistant S. aureus (MRSA) is high, empiric therapy with vancomycin

is justified. Historically, MRSA infections have been associated with exposures to health care settings; however, since 2001, community-associated MRSA has become an increasingly recognized and prevalent pathogen.[35] With the indiscriminate use of glycopeptides, vancomycin-intermediate S.aureus (VISA) and vancomycin-resistant S. aureus (VRSA) are a growing concern. Daptomycin (6 mg per kg IV every 24 hours) is the agent of choice in this setting. The prevalence of VISA and VRSA is not high enough at this time to warrant empiric therapy with daptomycin. Renal cortical abscesses can be cured with parenteral antibiotic therapy administered for a minimum of 10 days to 2 weeks, followed by oral antistaphylococcal therapy for at least an additional 2 to 4 weeks. The decision to pursue percutaneous drainage for therapeutic and/or diagnostic purposes is guided by the size of abscess and response to antimicrobial therapy. Renal abscesses less than 5 cm in size can be treated with antibiotic therapy alone.[36,37] If there is optimal response, fever gradually subsides over a 5- to 6-day period without recurrence. Flank or back pain abates rather quickly, and patients display significant clinical improvement within 24 hours of initiating antibiotic therapy. A prompt response to treatment justifies continuing antibiotic therapy without surgical intervention, and serial ultrasound or CT examinations can be used to show progressive reduction and ultimate disappearance of the renal mass. A contrary clinical course should suggest misdiagnosis or uncontrolled infection, with the development of perinephritis, perinephric abscess, or infection with an organism resistant to the antibiotics being administered. In such cases, modification of therapy may be required, based on the results of cultures of blood and urine. Nevertheless, a trial of intensive antibiotic treatment is warranted in lesions measuring less than 5 cm that are localized to the renal parenchyma. If the patient does not respond within 48 hours, percutaneous, ultrasonically, or CT-guided needle aspiration of the intrarenal fluid-filled lesion can be attempted.[38–41] If the renal abscess measures more than 5 cm, therapeutic and diagnostic percutaneous drainage along with antibiotics should be attempted.[37] If this treatment is unsuccessful, operative intervention should be undertaken.

Renal Corticomedullary Abscess

Etiology

Enteric aerobic gram-negative bacilli, predominantly Escherichia coli, Proteus spp., and, less commonly, Klebsiella spp., Enterobacter spp., and Pseudomonas spp. are usually responsible for intrarenal corticomedullary infections in association with vesicoureteral reflux or other urinary tract abnormalities.

Pathogenesis

Renal corticomedullary bacterial infections include a variety of acute and chronic parenchymal inflammatory processes. The more severe forms of these infections include acute focal bacterial nephritis, acute multifocal bacterial nephritis, and xanthogranulomatous pyelonephritis, which almost always involve only one kidney.

Acute focal bacterial nephritis is an uncommon, severe form of acute infectious interstitial nephritis presenting with a renal mass caused by acute focal infection without liquefaction.[42] This entity is also referred to as focal pyelonephritis or acute lobar nephronia, because the pathology consists of a heavy leukocytic infiltrate confined to a single renal lobe with focal areas of tissue necrosis.

Acute multifocal bacterial nephritis is also a severe form of acute renal infection in which a heavy leukocytic infiltrate occurs throughout the kidney with frank intrarenal abscess formation. Acute focal bacterial nephritis may represent an early phase of acute multifocal bacterial nephritis.[43]

Xanthogranulomatous pyelonephritis is a very rare and atypical form of severe chronic renal infection. Schlagenhaufer initially described the pathologic features of xanthogranulomatous pyelonephritis[44] in 1916. Grossly, the entire kidney or its involved segment is enlarged and may be fixed by perirenal fibrosis or retroperitoneal extension of the granulomatous process, which often resembles an inoperable tumor. Xanthogranulomatous pyelonephritis is classified into three stages based on the extent of involvement of renal and adjacent tissue by the xanthogranulomatous process.[45] In stage I (nephric), the xanthogranulomatous inflammatory process is confined to the kidney. Stage II lesions (perinephric) involve the renal parenchyma and Gerota's fat, whereas stage III lesions (paranephric) involve the renal parenchyma and its surrounding fat with widespread retroperitoneal involvement. Each stage is further divided into focal or diffuse, depending on the amount of parenchymal involvement. Microscopically, the disease is characterized by massive tissue necrosis and phagocytosis of liberated cholesterol and other lipids by xanthoma cells (macrophages). These foamy xanthomatous histiocytes appear to simulate clear-cell renal carcinoma.[46,47]

Acute focal bacterial nephritis, acute multifocal bacterial nephritis, and xanthogranulomatous pyelonephritis most commonly occur as a complication of bacteriuria and ascending infection, associated with tubular obstruction or scarring from prior infections, renal calculi, vesicoureteral reflux, urinary tract obstruction, or other abnormalities of the genitourinary system or in association with the endocrinopathies of diabetes mellitus or primary hyperparathyroidism.[9,11,14,15,42,43,47–51] These predisposing factors, particularly vesicoureteral reflux in children and renal calculi or other forms of urinary obstruction in adults, lead to intrarenal reflux and provide an avenue for bacteria to inoculate the renal parenchyma. Parenchymal infection develops with abscess formation because the kidney is unable to clear the infection in the presence of reflux, urinary obstruction, medullary scarring, or other causes of tubular obstruction. In adults, two thirds of intrarenal abscesses caused by aerobic gram-negative bacilli are associated with renal calculi or damaged kidneys, whereas in children this process is often associated only with vesicoureteral reflux. The incidence of renal abscesses in patients with diabetes mellitus is twice that in nondiabetic persons. In contrast to the staphylococcal renal cortical abscess of hematogenous origin, the gram-negative bacillary corticomedullary abscess of the kidney frequently produces severe renal infection. Although renal corticomedullary infections are confined within the substance of the kidney, they may perforate the renal capsule and form a perinephric abscess, extend toward

the renal pelvis and drain into the collecting system, or develop into a chronic abscess.[50] The etiology of xanthogranulomatous pyelonephritis is undefined; however, it appears to be related to a combination of chronic urinary tract infection and renal obstruction. The majority of the cases have renal calculi with staghorn renal calculi being the most common type.[52] Additional predisposing factors include chronic segmental or diffuse renal ischemia resulting in alterations in renal or lipid metabolism or both, lymphatic obstruction, abnormal immune response, diabetes mellitus, and primary hyperparathyroidism.[47,53,54]

Clinical Features

Renal corticomedullary abscesses affect males and females with equal frequency except for xanthogranulomatous pyelonephritis in adults, where females are more frequently affected than males.[53,54] Although these infections occur in all age groups, the incidence increases with advancing age. Peak incidence for xanthogranulomatous pyelonephritis occurs in the fifth to seventh decade and has been reported to occur in transplanted kidneys as well as native kidneys.[55] Most patients with acute focal bacterial nephritis, multifocal bacterial nephritis, or xanthogranulomatous pyelonephritis experience fever, chills, and flank or abdominal pain. Two thirds of patients have nausea and vomiting but dysuria is not necessarily present thus mimicking an abdominal process. Some patients may have a palpable flank or abdominal mass. Clinical signs of severe urinary tract infection with urosepsis may be seen in patients with acute multifocal bacterial nephritis, half of whom have diabetes mellitus. Nonspecific constitutional complaints of malaise, fatigue, and lethargy are particularly common (74%) in patients with xanthogranulomatous pyelonephritis, who may also complain of weight loss (24%). Significant physical findings include a renal mass (60%), hepatomegaly (30%), and, rarely, a draining flank sinus in patients with a past medical history of recurrent urinary tract infection (65%), renal stones (30%), or prior urinary tract instrumentation (26%). Peripheral white blood cell counts are elevated in most patients. The urinalysis is often abnormal, with pyuria, proteinuria, bacteriuria, and occasionally hematuria. However, the urinalysis may be normal in as many as 30% of patients. E. coli, Proteus mirabilis, and Klebsiella spp. are the most common organisms recovered from urine cultures. Blood cultures are also frequently positive in patients with acute focal bacterial nephritis or acute multifocal bacterial nephritis. Anemia is present in 75%, abnormal liver function tests (bilirubin, asparate transaminase [AST], alkaline phosphatase, and prothrombin time) in 38% to 63%, hypoalbuminemia in 60%, hypergammaglobulinemia (α_1- and α_2-globulin) in 79%, and hyperuricemia in 50% of patients with xanthogranulomatous pyelonephritis.[47,56] In general, the clinical and laboratory findings may or may not point to the urinary tract as the focus of infection and may not distinguish renal abscess from other abnormalities of the urinary tract.

Diagnosis

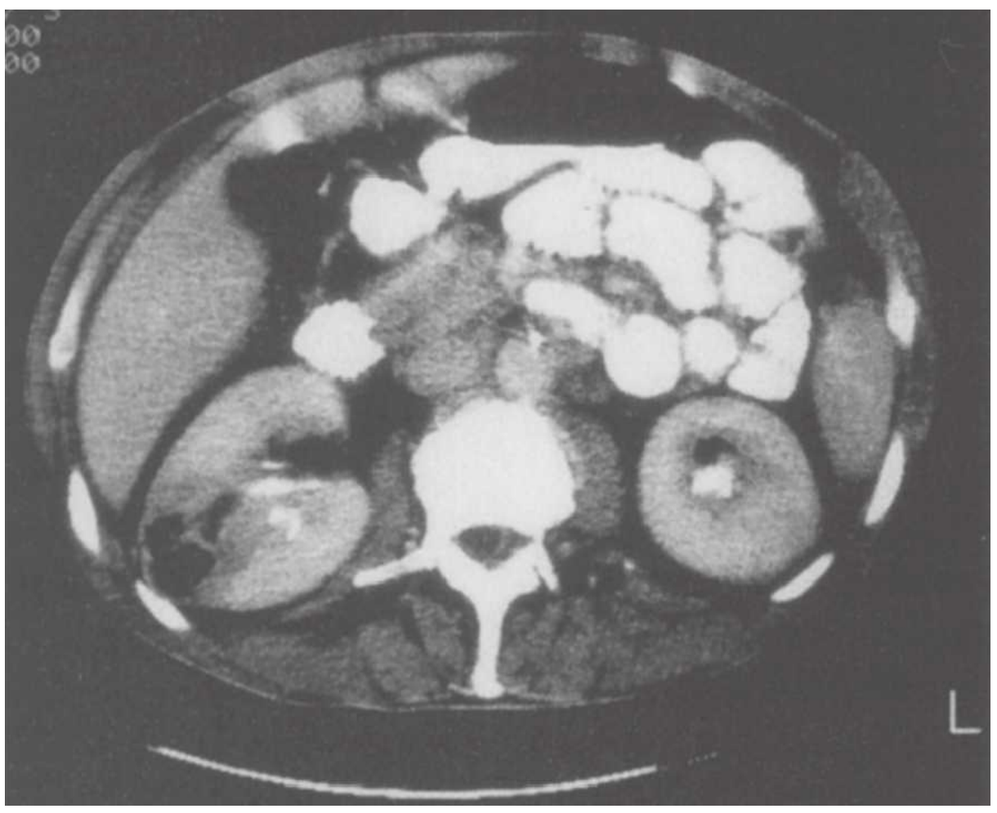
Renal corticomedullary abscesses must be differentiated from other space-occupying lesions in the kidney. Renal tumors, intrarenal cysts, renal cortical abscesses, and perinephric abscesses may mimic renal corticomedullary abscesses because the clinical presentation of each of these conditions is similar. Fever, chills, malaise, and back pain may be seen in each. Clinical signs of urosepsis may be present in patients with renal corticomedullary abscesses and, to a lesser extent, in patients with perinephric abscesses. In contrast, these signs are usually absent in patients with renal tumors, cysts, and renal cortical abscesses. Patients with renal corticomedullary abscesses often have an abnormal urinalysis (70%) with pyuria, proteinuria, and bacteriuria and blood cultures frequently are positive.[43]

Radiographic techniques are essential to identify renal corticomedullary abscesses. The urographic findings in patients with acute focal bacterial nephritis are: (1) a poorly marginated and relatively sonolucent ovoid mass disrupting the corticomedullary definition and producing some low-level echoes on ultrasound examination; (2) a solid-appearing mass on excretory urography, CT, or angiography; and (3) abnormal uptake of gallium at the location of the mass, which may be associated with increased activity elsewhere in the same or opposite kidney.[42] A fluid-filled mass or a fluid debris level typical of a frank renal abscess is not found in acute focal bacterial nephritis on ultrasonography.[42,43] In the pediatric population if there is evidence of nephromegaly (more than 3 standard deviations greater than the mean for age) on ultrasound or a focal mass is present, the probability of acute focal bacterial nephritis is high in the appropriate clinical setting.[57] On a non–contrast-enhanced CT, the lesion of acute focal bacterial nephritis is typically imperceptible. There are three characteristics seen with contrast-enhanced CT: lobar distribution of inflammatory areas, poorly defined wedge-shaped areas of diminished contrast enhancement without frank liquefaction, and masslike hypodense lesions in severe cases.[43,58] Renal abscesses, however, are usually round, have liquid centers, and are visible with and without contrast enhancement. The lack of a defined wall by ultrasound or CT in acute focal bacterial nephritis is an important factor distinguishing this entity from an abscess.[28] On angiography, narrowing and obstruction of veins within the mass, along with only minor arteriographic abnormalities, are characteristic of acute focal bacterial nephritis.[42] Focal abnormalities in the kidney on gallium images may be seen in some neoplasms and renal abscesses as well as acute focal bacterial nephritis. However, the diagnosis of acute focal bacterial nephritis is strongly suggested whenever the abnormalities are larger on the gallium image than on the urogram or sonogram or whenever bilateral abnormalities are seen on gallium images that correlate with a focal mass on the urogram or sonogram.[42] The distinction between acute focal bacterial nephritis, renal abscess, and tumor can be made also by needle aspiration. However, in most patients, a combination of imaging techniques is sufficient to diagnose acute focal bacterial nephritis and permit conservative medical therapy without confirmation by needle biopsy or surgery.[42,59] In this context, serial uroradiologic studies should be performed in 4 to 6 weeks, to follow the process to resolution.[26,51,60,61]

The urographic findings in patients with acute multifocal bacterial nephritis typically show severe impairment of

excretion of contrast material on the affected side, with renal enlargement, a diminished nephrogram, and a delayed pyelogram.[43,62,63] Ultrasonography may demonstrate areas of decreased echogenicity throughout the affected kidney. Poorly defined wedge-shaped areas of decreased contrast enhancement similar to those described in patients with acute focal bacterial nephritis can be seen on contrast-enhanced CT, except that multiple renal lobes are involved[43,64] (Fig. 24.6). If angiography is performed, the number and caliber of interlobar arterial branches are diminished, and fine linear stripes of alternating density and lucency in the angiographic nephrogram are present throughout the kidney.[43] CT is more sensitive than ultrasonography for the detection of intrarenal bacterial infections and defining the extent of disease.[26,28,65]

The radiographic findings in patients with xanthogranulomatous pyelonephritis are varied and uncharacteristic. The xanthogranulomatous process may occur in a localized (unifocal) or diffuse (multifocal) form in either a previously normal kidney or one that is obstructed, contains a stone, or has an anomalous collecting system or calyceal diverticulum.



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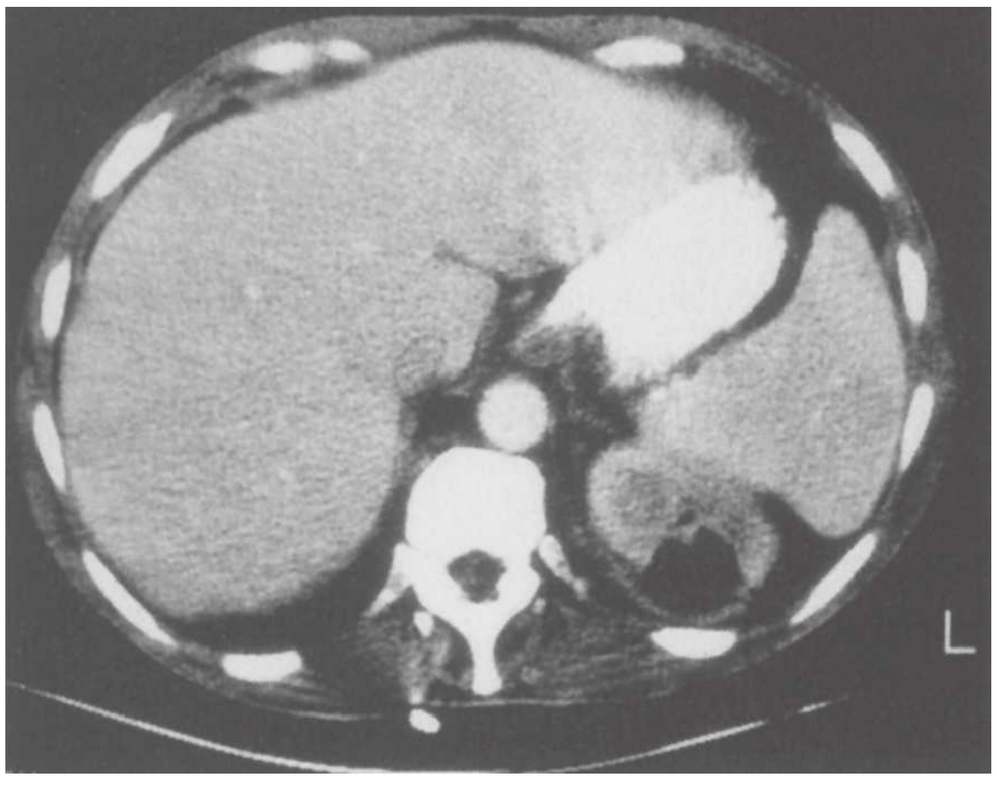

B

FIGURE 24.6 Computed tomography scans of a patient with bilateral intrarenal abscesses.

Perinephric extension of the xanthogranulomatous process produces obscure renal margins and ablation of the perinephric and paranephric fat.[47] Radiographically, xanthogranulomatous pyelonephritis appears as either localized or diffuse enlargement of one kidney with an indistinct renal outline. Urographically, the most frequent finding has been a stone-bearing (70%) and functionless (80%) kidney.[66] Calyceal deformity and irregularity (46%) may be present.[47,56] Neovascularity may be present on angiography, but most xanthogranulomatous renal masses are hypovascular or avascular, and the majority cannot be definitively distinguished from renal cell carcinoma.[47,67,68] There are no specific ultrasonographic findings that reliably distinguish renal tumors and abscesses from xanthogranulomatous pyelonephritis.[69] The sonographic features of xanthogranulomatous pyelonephritis include a diffusely enlarged kidney with multiple areas of increased anechoicity and a central echogenic focus with acoustic shadowing.[70] Although these findings are also seen in a hydronephrotic kidney, the presence of a central strongly echogenic focus suggests xanthogranulomatous pyelonephritis.[70,71] On CT, focal xanthogranulomatous pyelonephritis appears as low-density mass lesions, with wall enhancement surrounding dilated calyces, which may contain stones, or as a focal mass in one pole of a duplicated kidney.[72] In diffuse xanthogranulomatous pyelonephritis, CT demonstrates an enlarged kidney, often with central calcification in the renal pelvis without dilatation, and with multiple, rounded, low-density areas representing dilated calyces and abscess cavities. On enhanced scans, the walls of these cavities demonstrate a prominent blush because of increased vascularity within the granulation tissue and the compressed normal parenchyma.[66,72] Extension of the xanthogranulomatous process through the renal capsule, with involvement of the perirenal and pararenal spaces and the psoas muscle, when present, also can be observed on CT.[66,72] The diagnosis is suggested by CT findings in 44% of cases. This imaging study plays an important role in determining the extent of extrarenal disease and planning of operative treatment. Ultrasound examination is less specific than CT, and MRI generally offers no additional information over CT scans but may be useful in patients with renal insufficiency or allergy to iodinated contrast material.[28,73]

Treatment

In the past, surgical drainage, débridement, or nephrectomy was the accepted treatment for renal corticomedullary abscesses. Recent experience indicates that successful therapy of acute focal and multifocal bacterial nephritis with antimicrobial agents alone will produce a symptomatic response within 1 week in most patients and result in no sequelae.[29,34,41,43,49,50,74] Radiologic techniques should be used to document resolution of the infection[26]; nevertheless, the time at which the intrarenal infection is discovered and its degree of suppuration should guide its management. A well-established large abscess cavity may be more difficult to eradicate with antibiotics alone than an earlier lesion in the preabscess state; however, a trial of intensive antibiotic treatment is appropriate for lesions localized to the renal parenchyma before determining the need

for operative drainage, particularly in a promptly diagnosed and otherwise healthy person. Intravenous fluids and parenteral antibiotics should be started as soon as the diagnosis is entertained. In patients with acute focal or multifocal bacterial nephritis, initial antibiotic selection (empiric therapy), before the results of urine cultures and sensitivities are available, should be aimed at the most common uropathic, enteric gram-negative aerobic bacilli (e.g., E. coli, Klebsiella, and Proteus spp.). Monotherapy with a third generation cephalosporin (e.g., cefotaxime, ceftriaxone, or ceftazidime), an extended-spectrum penicillin (e.g., piperacillin), or ciprofloxacin is acceptable empiric therapy.

Combined therapy with a β-lactam, such as ampicillin or cefazolin, with an aminoglycoside is no more successful than single-agent therapy in the treatment of acute focal or multifocal bacterial nephritis.[41] Empiric therapy should be modified to the most effective single agent based on the results of the antibiotic sensitivities of the organisms recovered from cultures of urine, blood, or both. Although the duration of treatment has not been defined, current recommendations are to continue parenteral antibiotics for at least 24 to 48 hours after the resolution of fever and clinical improvement are attained. Oral antibiotic therapy, based on the results of antimicrobial susceptibility tests, can then be continued for an additional 2 weeks. Patients with acute focal bacterial nephritis typically respond to medical therapy alone (at least 14 days of an appropriate antimicrobial agent), and follow-up studies have shown resolution of the intrarenal lesion without the need for surgical drainage.[43,51,59] Although many patients with acute multifocal bacterial nephritis slowly improve with antibiotic therapy alone, some may require surgical intervention.

Patients who are likely to fail appropriate antibiotic therapy alone are those who have radiologic evidence of a large intrarenal abscess, significant obstructive uropathy, severe vesicoureteral reflux (primarily in children with gram-negative bacillary multifocal bacterial nephritis) with extensive parenchymal involvement, patients with diabetes mellitus with gas-forming infections, and patients of advanced age or with urosepsis.[14,43,48–50,75] In general, surgical intervention is indicated in the patient who has radiologic evidence of a large intrarenal abscess and persistent fever, with an absence of clinical response after 5 to 7 days of adequate antibiotic therapy. In patients requiring drainage, percutaneous aspiration of the abscess combined with systemic antibiotic therapy has been successful.[50,76,77] If a significant obstructive uropathy is present, prompt drainage, usually by percutaneous nephrostomy, is necessary with correction of the lesion, if possible, when the patient is afebrile and stable.[43] If surgical intervention is necessary, the abscess should be incised and drained, and nephrectomy should be reserved for diffusely damaged kidneys or for patients of advanced age who are septic and require urgent surgical intervention for survival.[48] Also, all children with renal parenchymal infection caused by gram-negative bacilli should undergo voiding cystourethrography to look for lower urinary tract abnormalities.[14] Thus, current clinical experience indicates that many patients with acute multifocal bacterial nephritis may not require surgery as they did in the past but may be treated successfully with antibiotics alone. The decision to drain the abscess mechanically should be based on the radiologic findings and response of the patient to initial drug therapy.

In contrast, patients with xanthogranulomatous pyelonephritis generally are not cured by antibiotic therapy alone. These patients often require surgical removal of the xanthogranulomatous process to cure this disease; however, there have been several case reports of successful treatment with antibiotics without surgical intervention.[53] The diagnosis of xanthogranulomatous pyelonephritis is not commonly made preoperatively; however, once the involved tissue is resected, the xanthogranulomatous process ceases and does not seem to recur. The prognosis in patients who have an otherwise normal urinary tract is excellent. Total nephrectomy is the usual procedure, but Malek and Elder[47] suggested that partial nephrectomy for selected localized disease, such as cases confined to the kidney (stage I) or involving the perinephric fat (stage II), may be sufficient. Partial nephrectomy is especially suitable in children, who usually present with localized disease.[47] In adults, the disease is frequently diffuse throughout most, or all, of the kidney and in advanced stages extend to the perinephric fat (stage II) and beyond (stage III). Although removal of the kidney and perirenal fat is preferred, it may be technically difficult and complicated by fistulization of adjacent bowel. Open surgical nephrectomy is preferred over laparoscopic nephrectomy as it is quicker, leads to less complications, and results in a similar postoperative course.[78] Even though xanthogranulomatous pyelonephritis does not recur following successful surgery, bacteriuria may continue in some patients and will require appropriate treatment.[47,56]

Infected Renal Cyst

Spontaneous infection of preexisting solitary renal cysts has been described.[43,79,80] In contrast, patients with autosomal dominant polycystic renal disease may have one or more cysts that become infected.[81] The most common etiologic agents are gram-negative uropathogens (especially E. coli) that are thought to infect the cysts as a consequence of bacteriuria and ascending infection.[82,83] Infection may also occur as a result of iatrogenic cyst instrumentation.[43] The clinical features of infected cysts are similar to those of an acute renal abscess and include nausea, chills, fever, flank or back pain, and dysuria. The diagnosis is made radiographically. Ultrasonography or CT may demonstrate a solitary renal mass that is compatible with an uncomplicated simple renal cyst or multiple lesions characteristic of polycystic renal disease. Gallium or indium imaging, gadolinium-enhanced MRI, or positron emission tomography may help to identify the infected cyst (or cysts) in patients with polycystic kidneys.[83,84] A definitive diagnosis can be made by ultrasound or CT-guided percutaneous cyst puncture with culture.

Effective treatment for infected solitary cysts includes percutaneous drainage combined with 2 weeks of appropriate antimicrobial therapy.[41] Fluoroquinolones and third generation cephaloporins have better penetration into the intrarenal cyst

compared to β-lactams.[83] Surgical drainage is rarely required. In contrast, the therapy of infected cysts in patients with polycystic renal disease is more difficult and not well defined. A conservative approach with long-term (6 to 8 weeks) oral antibiotic treatment directed against the most likely pathogens or those isolated from urine or blood cultures is successful in some cases.[82] In general, surgical drainage is generally avoided because of the difficulty in identifying which cyst is infected.

PERINEPHRIC ABSCESS

Etiology

S. aureus, E. coli, and Proteus spp. are the most common causes of perinephric abscesses. Other, less common causes include Klebsiella spp., Enterobacter spp., Pseudomonas spp., Serratia spp., and Citrobacter spp. Occasionally enterococci and streptococci are implicated, including two cases of S. pneumoniae[85,86] and one case of group B streptococcus in a diabetic patient.[87] Perinephric abscesses also may be caused by various anaerobic bacteria, including gram-negative bacilli and anaerobic cocci, Clostridium spp., and Actinomyces spp.[88] These anaerobic bacteria may be the pathogens in patients with abscess cultures reported to be sterile.[29,50] Mycobacterium tuberculosis is also an important cause of perinephric abscess, as are certain fungi, particularly Candida spp. Nocardia has been reported as a cause of perinephric abscess in immunocompromised patients.[89] More than one microorganism has been simultaneously recovered from perinephric abscesses in as many as 25% of patients.[48,50,90,91] Although bacteria isolated from the urine frequently correlate with those isolated from the abscess, in some patients, urine cultures are positive for microorganisms different from those subsequently isolated from the abscess material.[50,90,92] Blood cultures may be positive (20% to 40%) in some patients.[90]

Pathogenesis

A perinephric abscess is a collection of purulent material in the space between the capsule of the kidney and Gerota's fascia (Fig. 24.7). The abscess usually is confined to this space but may extend beyond Gerota's fascia into the pararenal space or even into the flank muscles or psoas muscle.[93] It may present as a draining flank abscess through Petit's triangle or as an abscess in the groin or paravesical area by extending caudally between the diverging layers of Gerota's fascia. It rarely perforates into the peritoneal cavity or ruptures into the colon. Cephalad extension may result in a subphrenic abscess, penetration of the diaphragm and empyema, lung abscess, or formation of a nephrobronchial fistula.[2,50,76,91] The most frequent initiating event is the direct extension or rupture of an abscess within the renal parenchyma into the perinephric space.[10,13] This is the most common mechanism responsible for staphylococcal perinephric abscesses that occur when a renal cortical abscess ruptures into the perinephric space.[9] Other causes include hematogenous or regional lymphatic seeding of the perinephric space, usually from sites of skin infection.[50,90] Rarely, spread of

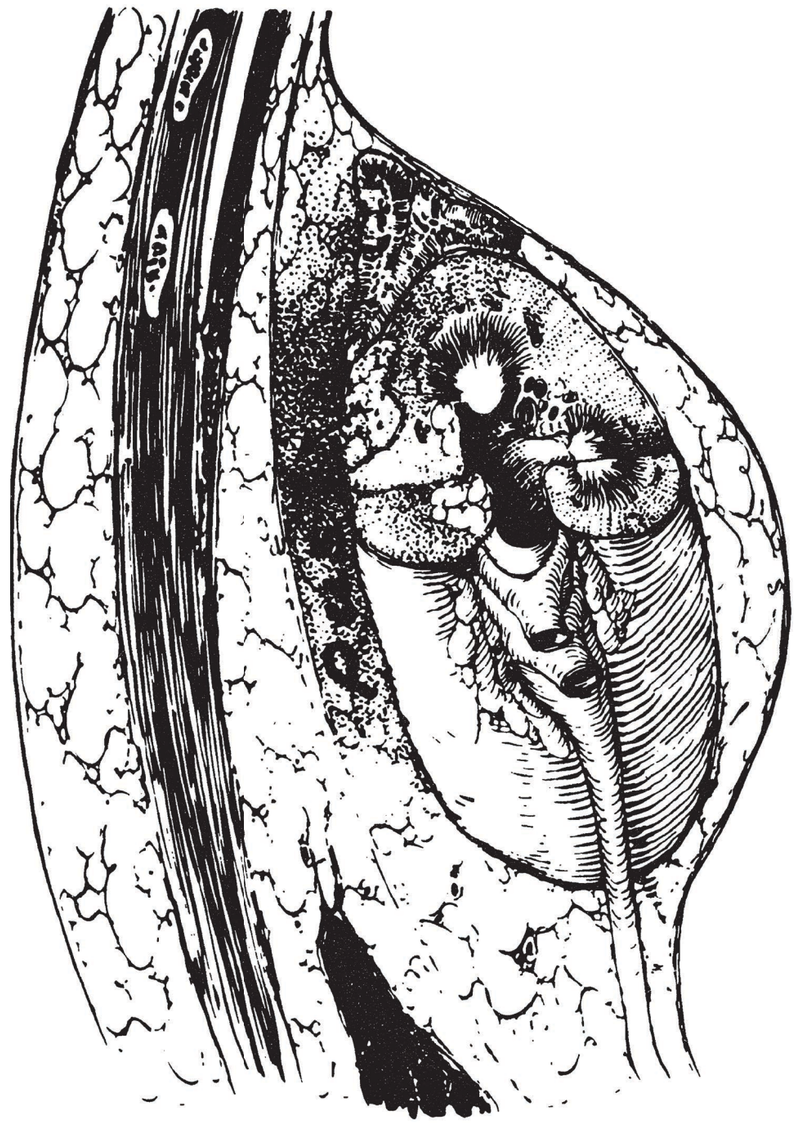

FIGURE 24.7 Diagram of the pathogenesis of a perinephric abscess.

infection from inflammatory lesions of the liver, gallbladder, pancreas, pleura, prostate, or female reproductive organs as well as diverticulitis, appendicitis, perforated carcinoma of the colon, and osteomyelitis of adjacent ribs or vertebrae has been implicated in the pathogenesis of perinephric abscess.[29,50,94–96] The majority of patients with perinephric abscesses have some form of obstruction to urinary outflow. Specific predisposing factors include renal or ureteral calculi, ureteral stricture, neurogenic bladder, vesicoureteral reflux, mechanical bladder outlet obstruction, neoplasm, renal papillary necrosis, polycystic kidney disease, genitourinary tuberculosis, immunosuppresion including renal transplantation, trauma (including urinary tract instrumentation, renal biopsy, or aspiration), and the associated conditions of diabetes mellitus (a major contributing factor), glucocorticoid therapy, and injection drug use.[43,50,81,85,97]

Clinical Features

The onset of perinephric abscess is characteristically insidious. Patients are often ill for 1 to 3 weeks before they seek medical care, and early recognition of this condition is difficult. The most common symptoms are fever, which occurs in almost all patients; unilateral flank pain, in 70% to 80%; and chills and dysuria, in 40% of patients.[50,90,98,99] Weight loss, nausea, and vomiting are less common. Diarrhea is very rarely a symptom associated with perinephric abscess.[100] Interestingly, in renal transplant recipients, the aforementioned symptoms are not masked despite immunosuppression.[101] On physical examination, flank and costovertebral angle tenderness are the most common findings, but abdominal tenderness may be present in about 60% of cases. In some patients, the pain may be

referred to the corresponding hip, thigh, or knee. Scoliosis, with splinting on the affected side, pain on bending toward the contralateral side and during either active flexion of the ipsilateral thigh against pressure, or extension of the thigh while walking may be present in some patients. A flank or abdominal mass is present in less than half the patients.[29,43,50,90] Routine laboratory studies are nonspecific. The peripheral white blood cell count is usually modestly elevated with associated neutrophilia. Anemia and azotemia may be present in 40% and 25% of patients, respectively.[98] Pyuria and proteinuria are common; however, hematuria is present in only 10% of patients and the urinalysis may be entirely normal in 25% to 30% of cases. Two thirds of patients have positive urine cultures, with more than 10^5 bacteria per mL of urine. Approximately 40% of patients are bacteremic.[29,43,50,90]

Diagnosis

A perinephric abscess must be differentiated from other infections of the urinary tract and from other occult abscesses. Patients with this disease may present with fever of undetermined origin or with unexplained peritonitis, empyema, or pelvic abscess resulting from extension of the perinephric abscess. The diagnosis should also be considered in patients with urinary tract infection who do not respond promptly to antibiotics and have an abnormality of the urinary tract or diabetes. Prompt diagnosis of this disease is made in less than one third of patients at the time of admission.[50,90] Up to 25% to 30% of patients are diagnosed only at autopsy.[50,90] This disease should be considered in the differential diagnosis of patients who present with the signs and symptoms described previously. Radiologic examinations with ultrasonography and CT are essential diagnostic aids in most cases. Roentgenogram of the chest may be normal or may show a pleural effusion, elevated hemidiaphragm either with or without decreased diaphragmatic excursion, or a lower lobe infiltrate.[50,90] Supine abdominal roentgenogram may demonstrate an upper quadrant mass, obliteration of the renal outline, vertebral scoliosis, or absent psoas shadows. However, obliteration of the psoas margin is not a reliable diagnostic sign for perinephric abscess.[9,13] Although uncommon, the presence of extraluminal, retroperitoneal gas bubbles in the area of the kidney suggests a perinephric abscess produced by gas-forming organisms. This condition, termed emphysematous pyelonephritis, occurs primarily in patients with diabetes mellitus, with or without urinary obstruction, and more rarely in nondiabetic patients who have urinary obstruction.[75,102,103] On excretory urography, important radiographic findings of perirenal abscesses include decreased renal mobility with respiration or position, absent or diminished renal function, caliectasis and other calyceal abnormalities, and displacement (usually medially and upward) of the kidney or ureter. Extrarenal extravasation of contrast material, although uncommon, is virtually diagnostic of perirenal abscess.[50,93] Also, fistula formation occurs occasionally between the perirenal space and other structures, such as the colon. Retrograde pyelography is usually not necessary, but it is occasionally helpful in identifying obstructive lesions distal to the renal pelvis. Ultrasonography may demonstrate an intrinsic mass in addition to the more characteristic findings of a perinephric fluid collection, along with displacement of the kidney, loss of a distinct renal outline, and renal fixation. These findings indicate extension of the inflammatory process to the perinephric space.[9,13] The sonographic appearance may vary from a nearly anechoic mass, displacing the kidney, to an echogenic collection that merges with normally echogenic fat within Gerota's fascia.[43] Ultrasound also may be useful to determine the extent of the abscess (Fig. 24.8)

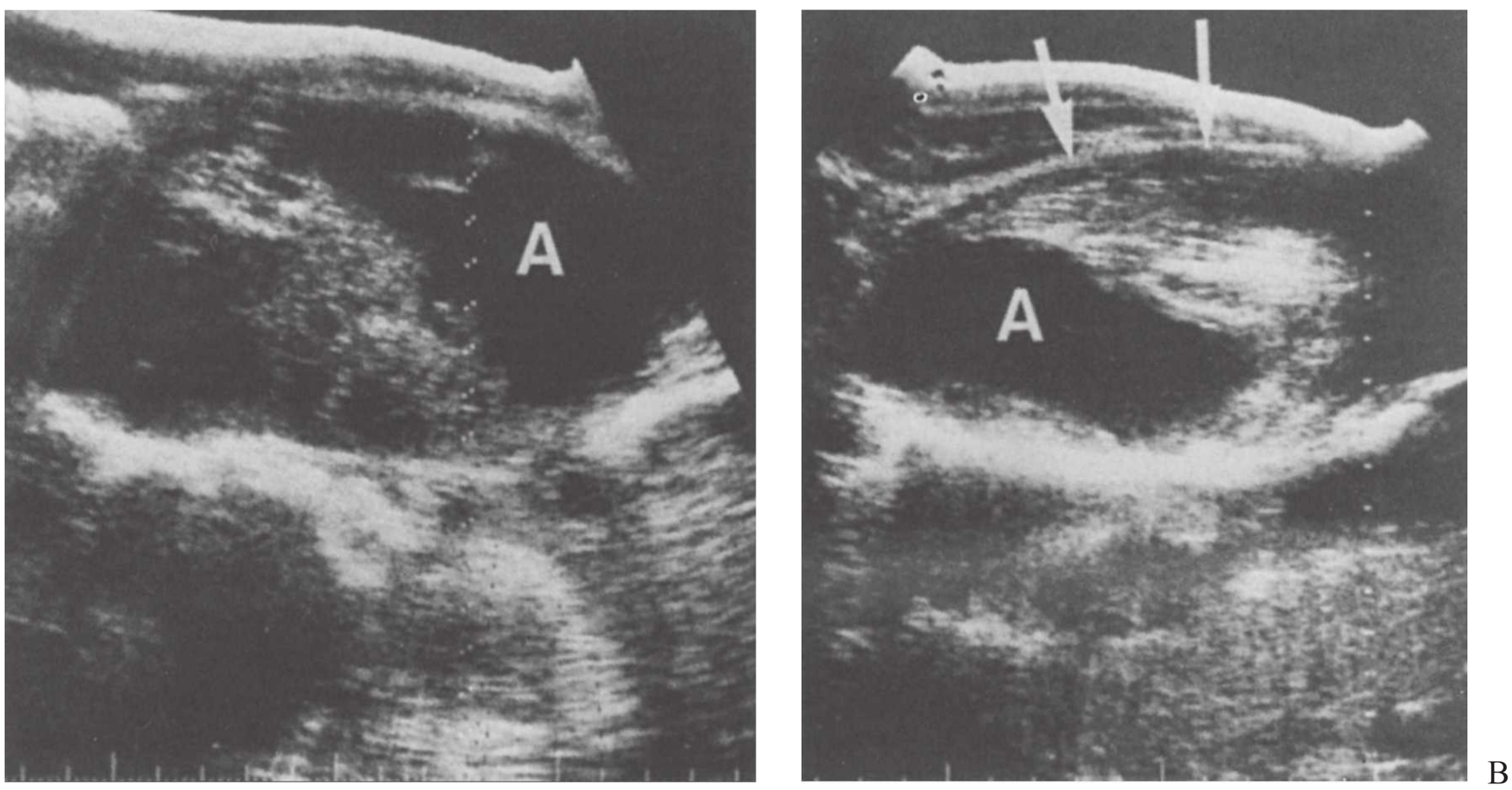

FIGURE 24.8 **A:** Longitudinal ultrasonogram down the right paravertebral region, demonstrating a huge complex mass with a large irregular fluid component, labeled *A*. **B:** Marked enlargement of the psoas major muscle *(arrows)* with a large contained fluid collection, labeled *A*. (From Andriole VT. The clinician's viewpoint. *Clin Diagn Ultrasound.* 1982;11:1, with permission.)

and detect associated obstruction of the collecting system. However, ultrasound may be falsely negative in 36% of cases as compared to CT.[98] Findings on CT include thickening of the renal fascia and perirenal fluid collection. In most cases, pus can be differentiated from other causes of perirenal fluid collections such as urine, blood, lymph, exudates, and transudates.[36] CT also provides the most precise anatomic information and can demonstrate the extent and route of the abscess beyond the renal capsule (e.g., extension to the flank or the psoas muscle) (Fig. 24.9). This detail is important in planning surgical drainage of the abscess. MRI has been used in conjunction with ultrasonography to detect perinephric abscess during pregnancy as CT is contraindicated.[104] Other modalities not used as frequently are ^{67}Ga or indium-111 (^{111}In) imaging and arteriography. Radionuclide imaging with ^{67}Ga or ^{111}In may be used occasionally to confirm the presence of renal or perirenal inflammation or evaluate renal function. Gallium or indium imaging may provide the first evidence of a perirenal abscess in patients with suspected infections but without localizing signs or symptoms. However, ^{67}Ga is not sufficiently definitive to exclude renal carcinoma, pyelonephritis, intrarenal abscess, or ureteral obstruction.[9,30,34] Thus a subtraction technique using ^{67}Ga citrate and ^{99m}Tc glucoheptonate has been used to define the extent of perinephric involvement as well as to eliminate any false-positive scans seen with gallium alone.[9,34] On angiography, characteristic findings of perinephric abscesses include an increase in number and size of the perforating arteries extending from the kidney into the abscess, stretching, and prominent tortuous capsular arteries around the abscess and a contrast blush.[7,9,21,22] Perirenal and renal abscesses can be arteriographically distinguished from tumors, as described previously; however, angiography is not necessary in most patients with perinephric abscess because of the availability of the newer, noninvasive imaging modalities.

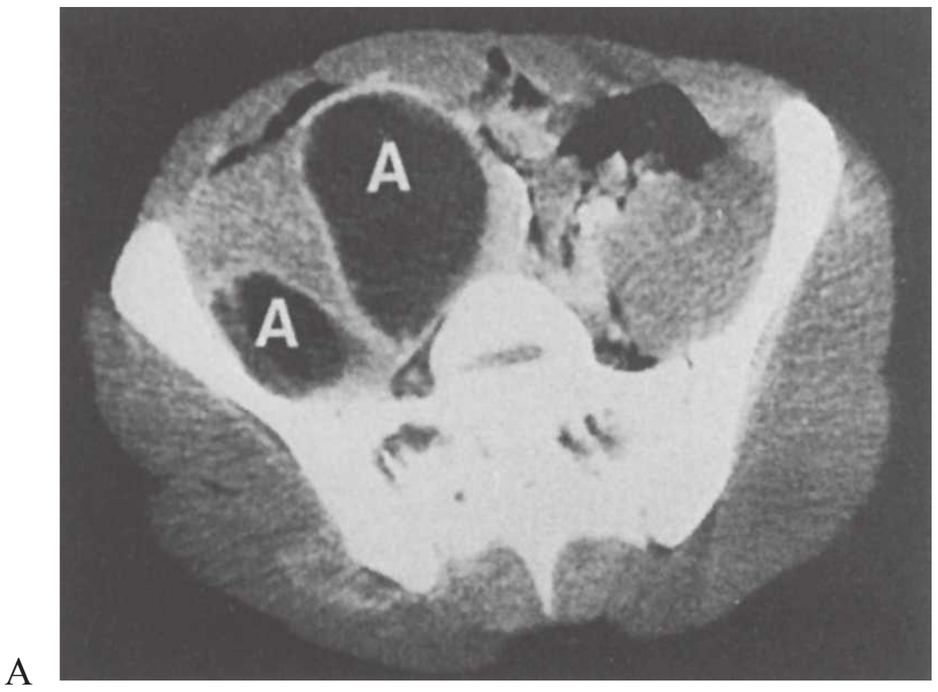

FIGURE 24.9 **A:** Computed tomography (CT) scan through midabdomen, demonstrating marked enlargement of the right psoas major muscle with a bilobed fluid-filled cavity, labeled *A* (from the same patient as in Fig. 24.8). **B:** Transverse CT scan through the level of the femoral head, showing the inferior extent of the abscess *(arrow)* pointing below the inguinal ligament. (From Andriole VT. The clinician's viewpoint. *Clin Diagn Ultrasound.* 1982;11:1, with permission.)

Treatment

Early surgical or percutaneous (under imaging guidance) drainage of the perinephric abscess is imperative.[43,50,90,105] Antibiotic therapy alone is inadequate and should be used as adjunctive treatment. In some patients, the perinephric abscess has been drained by percutaneous tube placement, aspiration of pus, and antibiotic irrigation prior to definitive surgery (nephrectomy), which is frequently necessary.[46,50] In others, acute nephrectomy is performed at the time of initial surgical drainage. There are rare case reports of immunocompetent patients with small (≤3.5 cm) abscesses who were successfully treated with prolonged courses of intravenous antibiotics without drainage[36,77]; however, immunocompromised patients or those with abscesses larger than 2 cm did poorly and had high mortality rates when treated with antibiotics alone. CT should be used to follow the response to treatment.

Initial antimicrobial therapy should be aimed at the most common uropathic gram-negative bacilli as well as against staphylococci, because some perinephric abscesses are a consequence of staphylococcal renal carbuncles. An aminoglycoside (gentamicin or tobramycin) in a dose of 1 to 1.5 mg per kg of body weight every 8 to 12 hours in patients with normal renal function should be combined with an antistaphylococcal β-lactam, oxacillin, nafcillin, or cefazolin, intravenously. The dose of gentamicin and tobramycin must be adjusted for those patients with compromised renal function. If suspicion of MRSA is high, vancomycin can be given empirically instead of a β-lactam. If an extended spectrum β-lactamase producing gram-negative organism is suspected, a carbapenem (e.g., doripenem, meropenem, and ertapenem) should be used in place of a β-lactam. Therapy should be modified based on the results of the antibiotic sensitivities of the organisms recovered from cultures of the abscess material. An antipseudomonal β-lactam (e.g., ticarcillin, piperacillin, cefoperazone, or ceftazidime) or a quinolone (ciprofloxacin) should be added to the aminoglycoside if Pseudomonas aeruginosa is the cause of the infection, and clindamycin or metronidazole should

be added if anaerobic bacteria are involved. A combination of penicillin or ampicillin plus gentamicin is the treatment of choice in enterococcal infections. Isoniazid plus rifampin and ethambutol or streptomycin is necessary for abscesses caused by M. tuberculosis and azoles (fluconazole or voriconazole) for those caused by fungi.

The prognosis in patients with perinephric abscess is poor. Even though there have been major improvements in diagnostic technology, surgical therapy, and antimicrobial treatment, the mortality associated with perinephric abscess remains high and is in a range from 20% to more than 50%.[29,48,50,77] Prompt diagnosis of perinephric abscess, immediate surgical or percutaneous drainage, appropriate antimicrobial therapy, followed by definitive surgical therapy for cases with poor response may be effective in reducing this high mortality rate.

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