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Nontraumatic urinary tract emergencies include acute obstructive uropathy, infections which can become complicated, and acute renovascular abnormalities. Imaging plays an important role in evaluating the extent of the acute process and the location and in differentiating urinary tract emergencies from other acute conditions which may be clinical mimics.

Obstructive Uropathy

Obstructive uropathy is defined as structural or functional blockage of normal urinary outflow and may occur at any level of the urinary system. This leads to urine stasis, pressure buildup, and failure of excretion [1]. Etiologies include stones, blood clots, infection/inflammation, extrinsic compression, functional spasm, and primary or metastatic tumors. In healthy individuals, unilateral renal obstruction can be compensated for by the contralateral kidney, without measurable changes in overall excretory function. However, in patients with underlying chronic renal disease and/or bilateral obstruction, significant functional decompensation may occur. Additional complications include infection and collecting system rupture [2].

The clinical presentation of acute obstructive uropathy may include flank pain, dysuria, hematuria, oliguria, fever, and nausea/vomiting. In patients with fulminant uremia, lethargy and mental status changes may be present [2].

Imaging

Plain radiography may be used as initial screening for patients with acute abdominal/flank pain, although computed

tomography is the modality of choice. Radiography has 45–60 % sensitivity and 70 % specificity for detecting renal stones (Fig. 7.1a). Findings may be falsely negative for noncalcified stones, calcified stones less than 4 mm, and patients with large body habitus. In addition, false-positive interpretations may occur with gallstones, phleboliths, and granulomatous calcifications [1]. Other causes of obstruction, including masses and blood clots, are poorly characterized. Radiography may also be used for surgical planning and follow-up.

Multidetector computed tomography (MDCT) is the gold standard for imaging patients with suspected obstructive uropathy. Stone/lesion size, location, and degree of obstruction can be quantified. Non-contrast CT (NCCT) has a sensitivity of 95–98 % and specificity of 96–100 % for detecting urinary stones [3–5]. NCCT is relatively quick, without the use of intravenous or oral contrast, making it particularly useful in the emergency setting. Most stones appear radiodense on CT. Rarely, in HIV patients who are poorly hydrated and treated with indinavir, crystalline stones develop which are radiolucent on NCCT. When findings are negative or equivocal, intravenous contrast can be administered to assess for additional renal or abdominal processes. Besides urinary stones, other etiologies of intraluminal obstruction include blood clots, which are dense but lower in attenuation than calculus; masses, which measure soft tissue attenuation and enhance following contrast administration; and ureteral spasm, strictures, or injury. Extraluminal compression may be caused by retroperitoneal masses, abscess, inflammation, or posttraumatic hematoma [4, 5].

At CT evaluation for ureteral stone, one should focus at the most common locations; narrowing of the ureter occurs naturally at the ureterovesicular junction (UVJ), the pelvic brim as the ureter crosses iliac vessels, and at the ureteropelvic junction (UPJ) (Fig. 7.1b, c) [3]. Frequently, a transition point is visible, with urinary decompression more distally. Secondary CT findings in obstructive uropathy include hydroureter, hydronephrosis, perinephric stranding, and possible enlargement of the unilateral kidney

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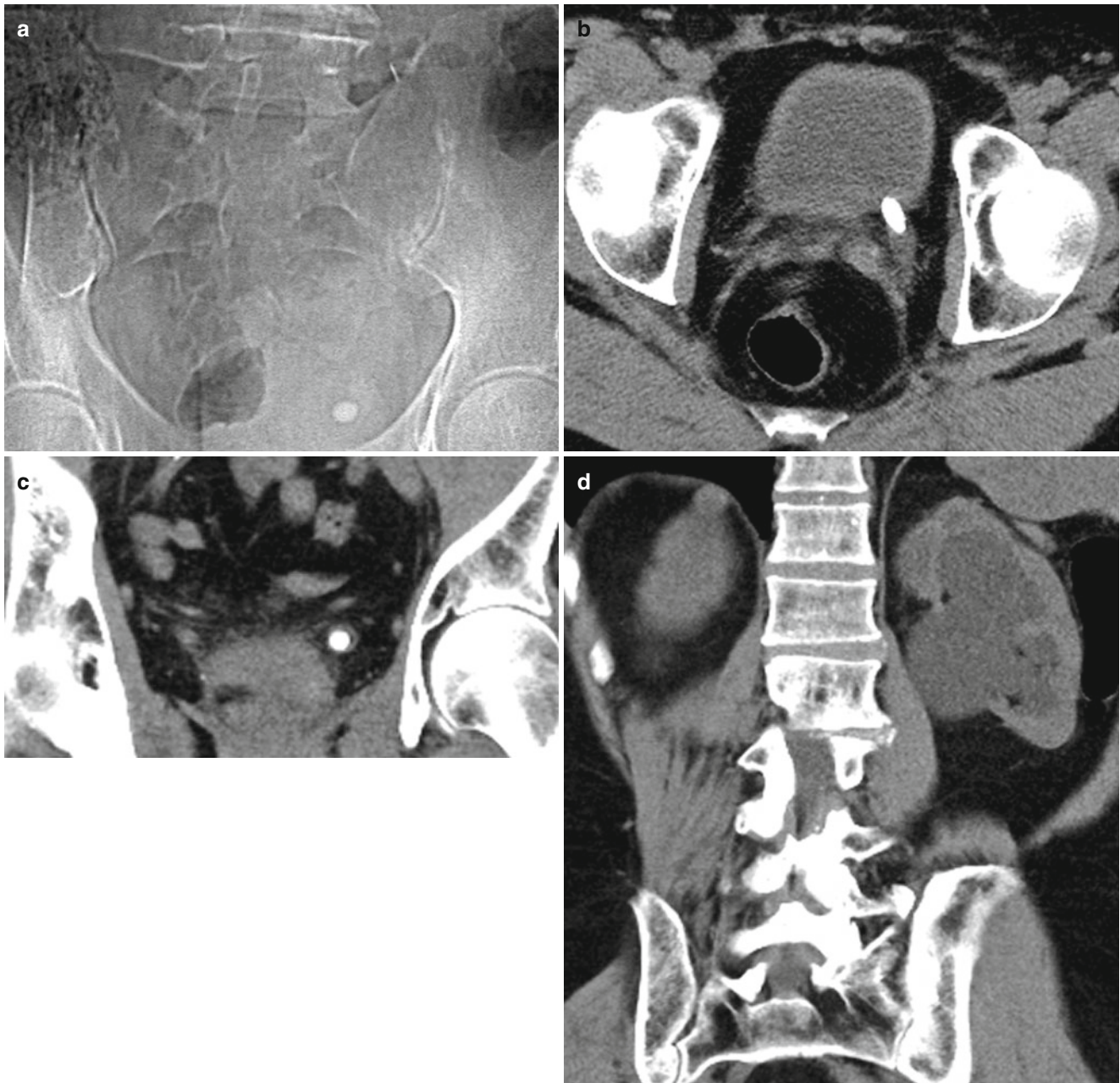


Fig. 7.1 Obstructing left UVJ stone in a 71-year-old male with elevated creatinine and hydronephrosis. (a) Scout view demonstrates a coarse calcification in the left pelvis corresponding to a left UVJ stone as confirmed

on subsequent CT images. (b–d) Non-contrast-enhanced CT axial (b) and coronal (c, d) images show a 14×6×4 mm left UVJ stone (b, c) with soft tissue rim sign (c) and upstream severe left hydronephrosis (d)

(Fig. 7.1d) (Table 7.1). Periureteral wall thickening and perinephric/periureteral fat stranding reflect acute inflammation. The “soft tissue rim sign” is due to edema of the ureteral wall at the site of stone impaction and may help differentiate a ureteral stone from a phlebolith in an adjacent vein (Fig. 7.1c) [6]. Renal edema may be appreciated, with decrease in Hounsfield attenuation on NCCT (“pale kidney” sign). Following contrast administration, there may be delayed excretion of contrast into the collecting system/ureter or beyond the level of obstruction (“delayed

nephrogram/urogram”), which may be partial or complete (Fig. 7.2). Pyelosinus or forniceal rupture manifests as a perinephric fluid (Fig. 7.3). In cases of extrinsic compression, the ureter appears abnormally deviated due to mass effect [4, 5, 7].

Ultrasonography (US) is a useful screening examination for pregnant and pediatric patients, in whom radiation exposure is a concern. The sensitivity for urinary obstruction is 60–70 %, but this is highly operator and patient dependent and involves limited ureteral assessment [8].

Table 7.1 CT findings in acute obstructive uropathy

1. Calcified stone in the GU tract (UVJ, UPJ, and pelvic brim are the most common locations)
2. Hydroureter
3. Hydronephrosis
4. Soft tissue rim sign
5. Perinephric/periureteral stranding
6. Unilateral enlarged kidney
7. Forniceal rupture
8. Pale kidney sign

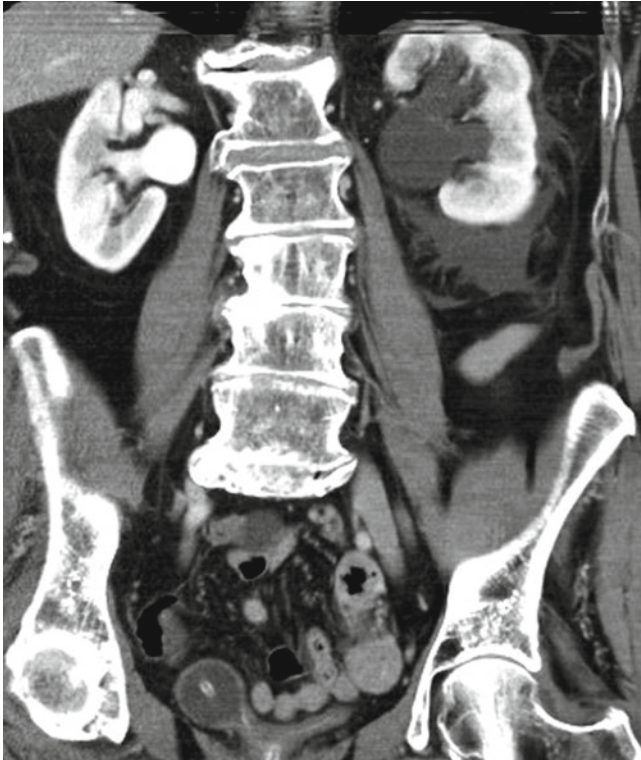


Fig. 7.2 Delayed excretion of contrast in an 87-year-old female with left abdominal pain. Contrast-enhanced coronal CT image shows left hydronephrosis and delayed excretion of contrast (delayed enhancement) from the left kidney as compared to the right in this patient with an obstructing 9 mm left UPJ stone

Renal stones are echogenic foci with or without shadowing, depending on size, composition, and technique (Fig. 7.4). Smaller stones may blend into the echogenic renal sinus and be missed. Ureteropelvic and ureterovesical junction stones, which lie superficially within good acoustic windows, may be seen. However, mid-ureteral stones are extremely difficult to detect. The finding of hydronephrosis is suggestive but may be absent in early obstruction (false negative) or present due to alternate causes such as pregnancy and reflux (false positive) (Fig. 7.5a). One must distinguish true hydronephrosis from mimics such as parapelvic cysts, extrarenal pelvis, and pelvicaliectasis. Additional signs include absent

or decreased ureteral jets and a Doppler arterial resistive index greater than 0.70 (or difference between kidneys greater than 0.10). However, these findings are neither sensitive nor specific [8].

Magnetic resonance imaging (MRI) is an alternative to CT, particularly in pediatric and pregnant patients for whom radiation exposure is a concern. MRI offers superior soft tissue contrast, albeit with increased costs and longer scan times. Ultrafast scanning protocols have been developed to image the urinary system with good spatial resolution in multiple planes. Fluid-sensitive T2-weighted sequences, such as single-shot fast spin echo (SSFSE) and balanced steady-state free precession (SSFP), utilize urine as an intrinsic contrast agent [9]. Fat-suppressed sequences are also acquired to enable differentiation from intraperitoneal/retroperitoneal fat. Urinary calculi, which are proton poor, may appear as hypointense filling defects (Fig. 7.5b, c). Stones smaller than 1 cm are frequently not seen – in which case diagnosis relies on signs of urinary obstruction, such as a ureteral transition point [9]. Acute obstruction frequently presents with soft tissue wall thickening, stranding, and edema, manifesting as high T2 signal and loss of renal corticomedullary differentiation. Multiphase gadolinium-enhanced imaging can be performed using a T1-weighted spoiled gradient echo sequence. This allows assessment of the renal parenchyma, improves urinary-calculi contrast, and quantifies collecting system transit time [9].

Complications and Treatment

In the early stages, acute obstructive uropathy is reversible with minimal renal parenchymal damage. Urinary stones smaller than 5 mm can be managed conservatively and usually pass spontaneously. Large and/or irregularly shaped stones may become impacted, predisposing to infection and collecting system rupture which can result in hematogenous dissemination of infection (urosepsis) [2].

In urgent situations, drainage procedures such as percutaneous nephrostomy, nephroureteral stenting, and suprapubic cystostomy can be performed to decompress the urinary system proximal to the site of obstruction [2].

Infections

Urinary tract infections have accounted for approximately one million emergency department visits annually in the USA [10]. Most of these are uncomplicated and only involve the urinary bladder. However, if the infection migrates proximally or is spread hematogenously, pyelonephritis may ensue.

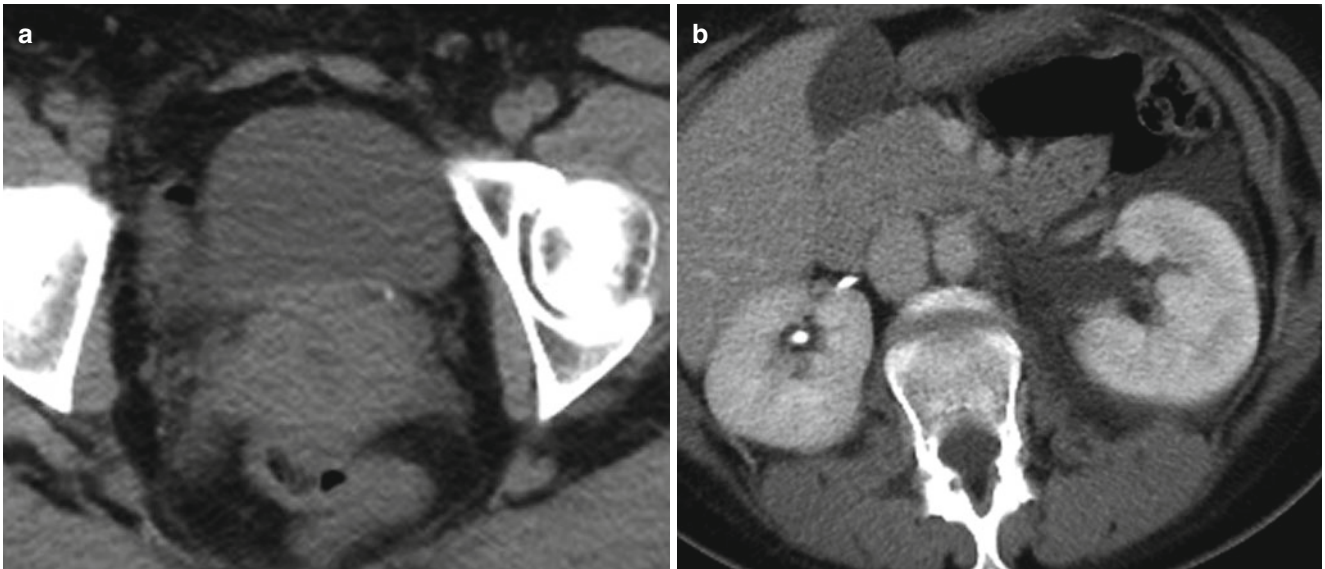


Fig. 7.3 Forniceal rupture in a 41-year-old female with left flank pain. (a) Non-contrast-enhanced axial CT image demonstrates 2 mm left UVJ stone. (b) Contrast-enhanced CT axial image demonstrates perinephric fluid consistent with fornix rupture

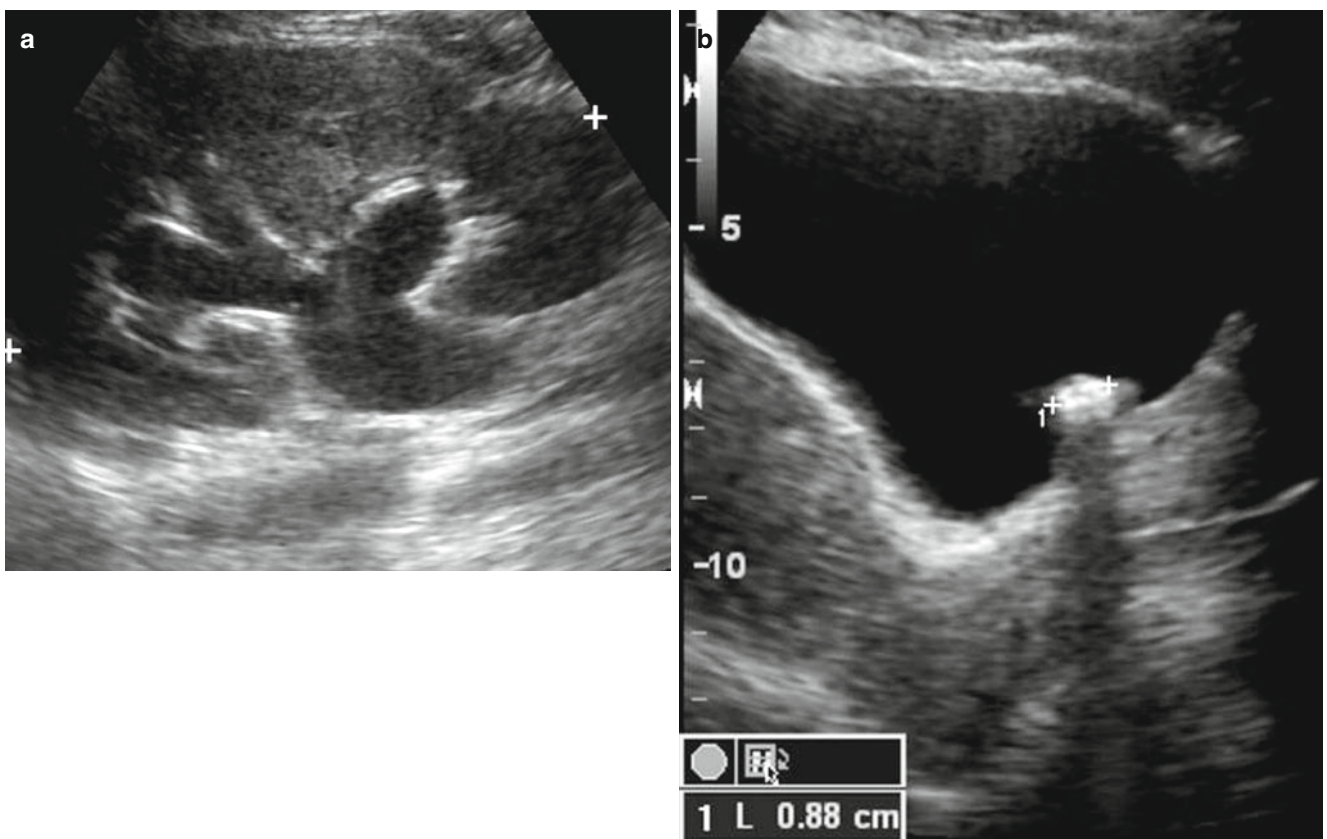


Fig. 7.4 Obstructing left UVJ stone with upstream left hydronephrosis. Ultrasound images show moderate left hydronephrosis (a). Shadowing echogenic focus at the left UVJ is consistent with obstructing stone (b)

Acute Pyelonephritis

Urinary tract infections typically begin in the urinary bladder and migrate proximally, leading to tubulointerstitial

inflammation. This ascending infection often occurs in the absence of reflux due to the virulence of the bacteria, most commonly gram-negative organisms, in particular *Escherichia coli*. Pyelonephritis can also occur if the urinary infection

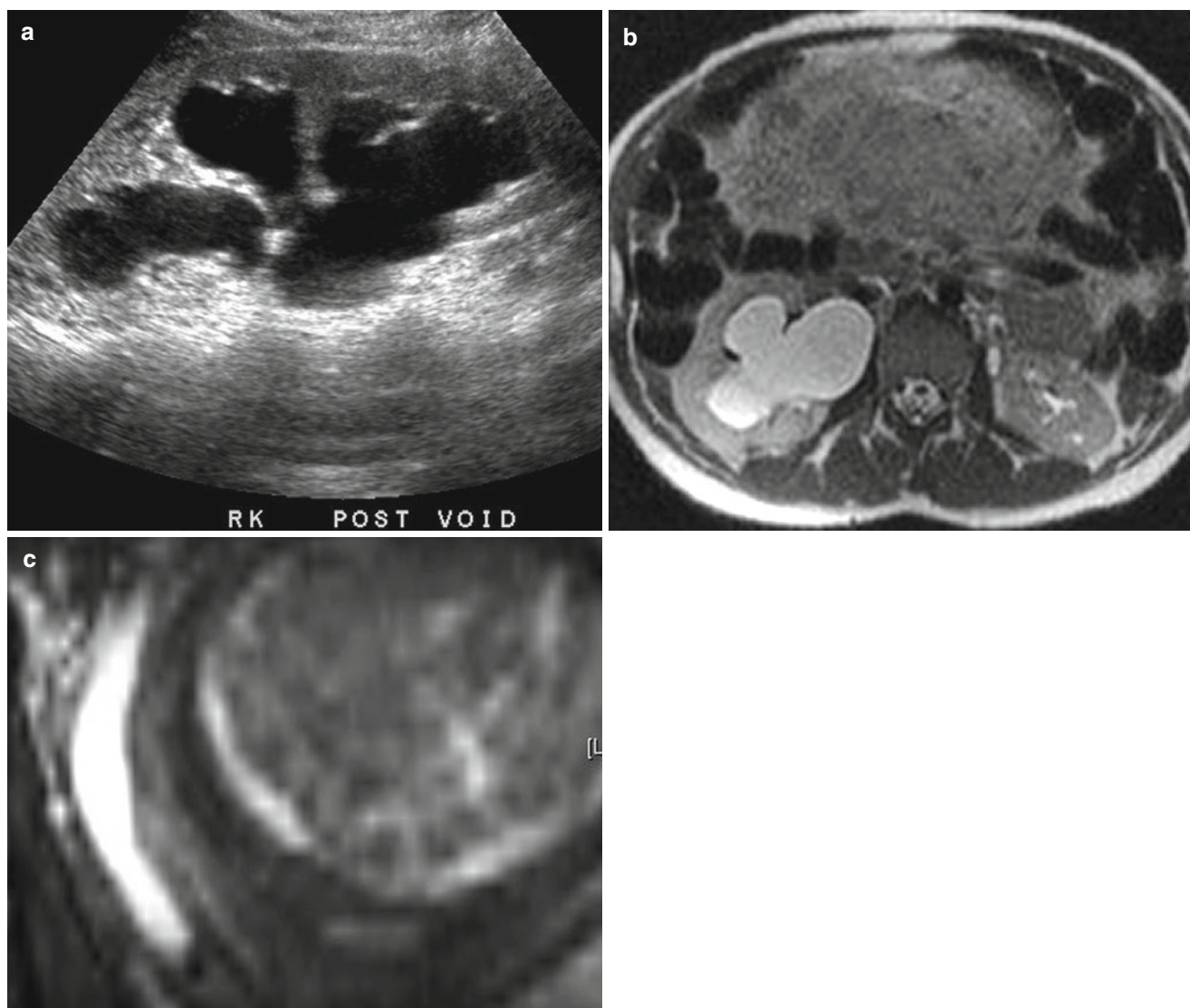


Fig. 7.5 A 27-year-old female, 34 weeks pregnant, with right abdominal pain. (a) Ultrasound shows moderate right hydronephrosis. Distal right ureter not visualized and etiology was unclear. (b, c)

Subsequent MR imaging shows moderate to severe right hydronephrosis on axial SSFSE HASTE imaging (b) secondary to distal right ureteral stone (c, coronal true FISP)

is spread to the kidneys hematogenously, from skin infection or endocarditis as may be seen in intravenous drug abusers.

Patients with pyelonephritis tend to present with fever, chills, and flank pain with costovertebral angle tenderness. Symptoms often also include dysuria, urinary frequency, and urgency [10]. Additional symptoms of acute pyelonephritis may include abdominal pain, nausea, and vomiting, which overlap with many other conditions, particularly gastrointestinal. Urinalysis findings include pyuria, bacteriuria, and positive urine culture.

Imaging

The majority of patients respond well to antibiotic treatment and imaging is often not required. However, in certain

circumstances, imaging can play an important role in differentiating acute pyelonephritis from other entities causing acute symptoms (particularly if the patient has failed 72 h of antibiotic treatment), detecting structural abnormalities, evaluating patients at high risk for complications, and characterizing the severity of infection and extent of possible organ damage.

MDCT is the preferred imaging modality to evaluate for acute bacterial pyelonephritis. Post-contrast imaging findings include one or more wedge-shaped hypodense areas extending from the papilla to the renal cortex, consistent with a striated nephrogram and representing decreased enhancement compared to the surrounding renal parenchyma (Fig. 7.6a). The findings result from stasis of contrast material within edematous tubules that demonstrates increasing attenuation over time. The decreased enhancement or striation is due to reduced perfusion secondary to obstruction of

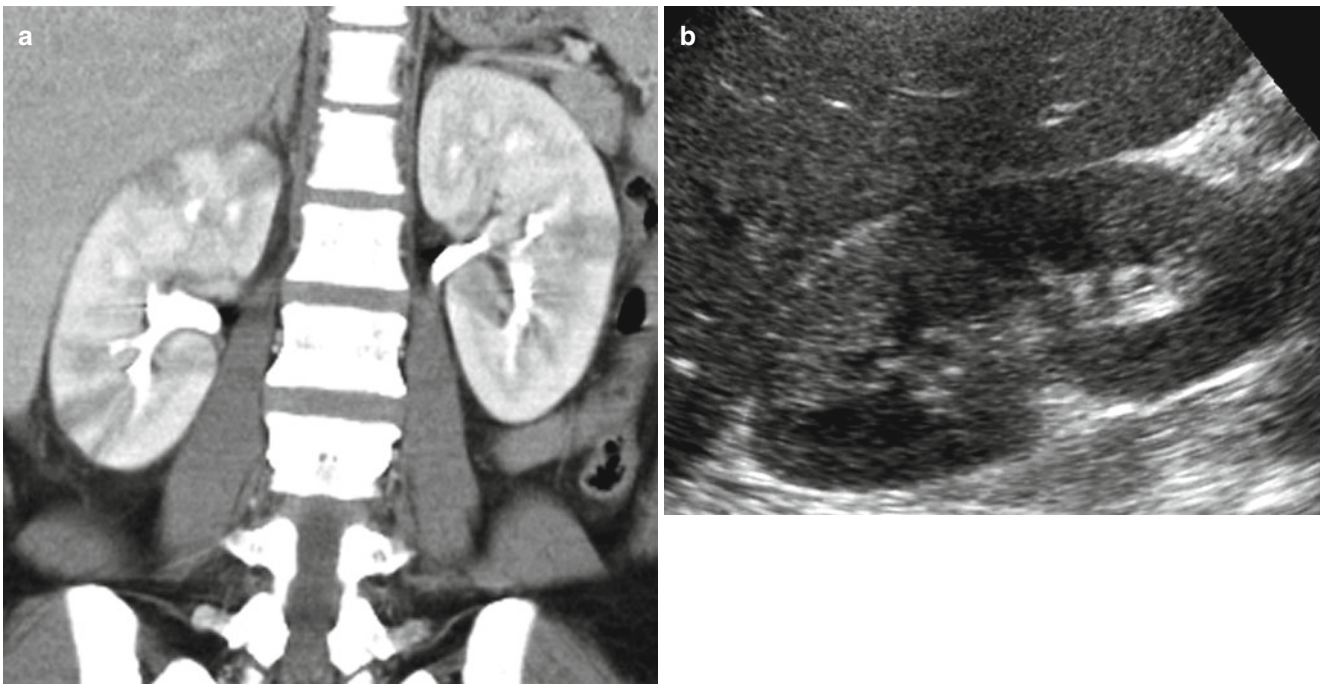


Fig. 7.6 Acute bilateral pyelonephritis in a 34-year-old female with fever, chills, and right upper quadrant pain. (a) Contrast-enhanced coronal CT image demonstrates bilateral, right greater than left, striated

nephrograms with alternating areas of hypodensity representing decreased enhancement. No drainable fluid collection. (b) Ultrasound image shows wedge-shaped echogenic area in the right kidney

Table 7.2 Differential diagnosis of striated nephrogram

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|--------------------------|
| 1. Acute pyelonephritis |
| 2. GU obstruction |
| 3. Renal vein thrombosis |
| 4. Contusion |

Table 7.3 Imaging of acute pyelonephritis

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|---|
| 1. CT and MRI: striated nephrogram |
| 2. US: often negative but can see hypoechoic or hyperechoic areas |
| 3. Enlarged kidney |

the renal tubules by intraluminal inflammatory debris, interstitial edema, and vasospasm [10]. A “striated nephrogram” can also be seen in GU obstruction, renal vein thrombosis, and contusion [11] (Tables 7.2 and 7.3). On non-contrast-enhanced CT, affected regions of the kidney may be lower in density due to underlying edema, and there may be loss of the renal pyramids. The affected kidney may be enlarged [12]. Renal calculi may be present. Secondary findings due to inflammation in acute pyelonephritis may also include perinephric stranding, thickening of Gerota’s fascia, and hydronephrosis [10, 12].

Ultrasound may be used to evaluate the urinary tract in cases of infection, in particular to assess for hydronephrosis or renal abscess. Occasionally findings of pyelonephritis may be found at ultrasound, although ultrasound is not as sensitive as CT and patients with clinically suspected

pyelonephritis often have negative ultrasound results. Changes in the echotexture of the renal parenchyma may be seen and include hypoechoic areas due to edema and hyperechoic regions due to hemorrhage (Fig. 7.6b) [10]. There may be hydronephrosis, renal enlargement, and/or loss of normal corticomedullary differentiation. Regions of hypoperfusion may be seen with color Doppler. Ultrasound is limited in assessing the full extent of pyelonephritis and perinephric extension and in visualizing microabscesses [10].

The use of MRI in the evaluation of acute renal infections tends to be limited to patients who cannot undergo intravenous contrast-enhanced CT (i.e., allergy to IV contrast material), patients in who radiation is a concern, or patients with inconclusive/equivocal CT results. MRI is much more susceptible to patient motion artifact than CT is, and it can be more difficult to obtain high-quality images in sick patients. In acute pyelonephritis, similar to that seen on CT, MRI shows wedge-shaped areas of decreased enhancement of the renal parenchyma on T1 fat-saturated post-contrast images and may show areas of decreased signal on T2-weighted images (Fig. 7.7). Renal enlargement and perinephric fluid may also be seen.

Treatment and Complications

Antibiotic therapy is the mainstay of treatment in acute bacterial pyelonephritis and most patients respond well. However, complications of acute pyelonephritis can include

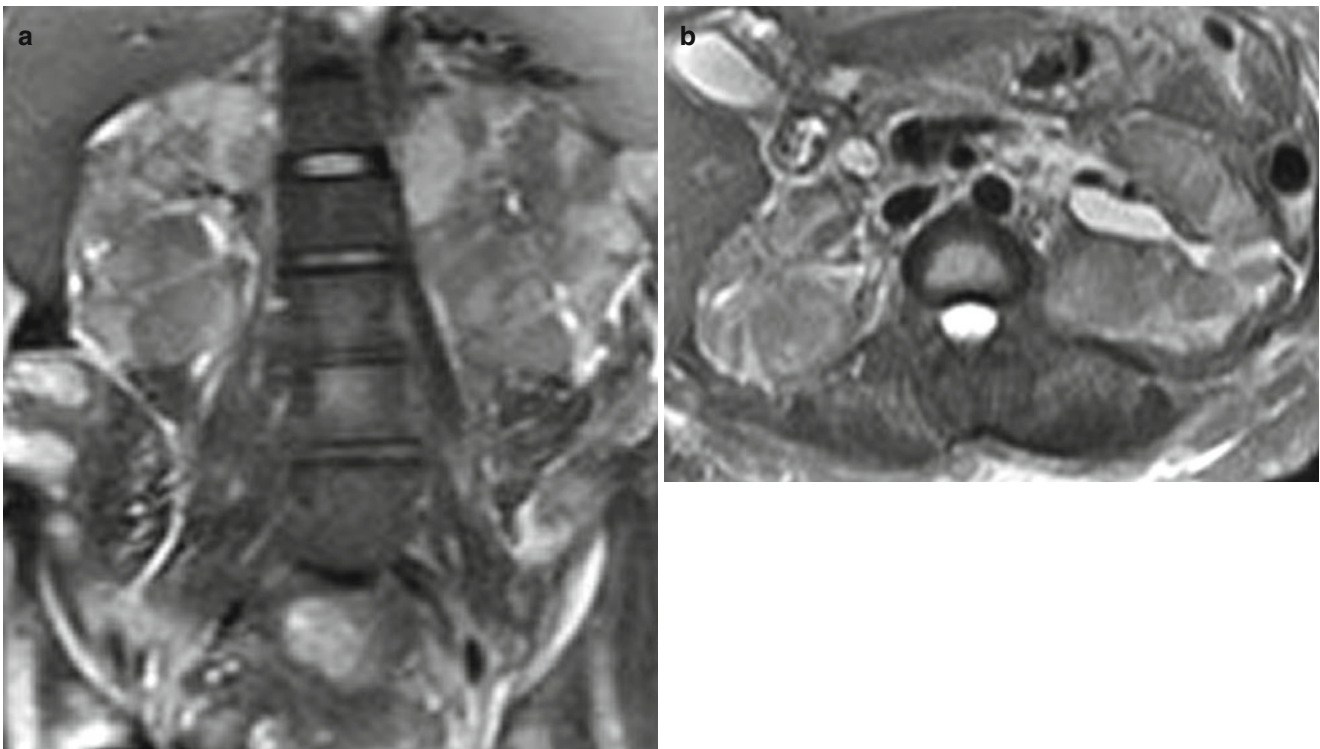


Fig. 7.7 A 62-year-old female with fever, chills, UTI, and *E. coli* bacteremia presenting with acute bacterial pyelonephritis. SSFSE (HASTE) MR coronal (a) and axial (b) images demonstrate diffuse heterogeneous signal in linear, wedge-shaped configuration in both kidneys

renal obstruction, renal or perirenal abscess, and emphysematous pyelonephritis.

Renal Abscess

Renal or perinephric abscesses typically develop from inadequately treated pyelonephritis more commonly due to ascending infection, with diabetic patients particularly prone (75 % of renal abscesses are seen in diabetic patients), and less often hematogenous spread [10]. The most common microorganisms are *E. coli* and *Proteus mirabilis*. Renal abscesses can be solitary or multiple, with multiple abscesses more commonly seen in hematogenous spread of infection [10].

Patients with renal abscess tend to have persistent symptoms of pyelonephritis despite at least 72 h treatment with antibiotics.

Imaging

CT is the preferred imaging modality to detect renal abscess in the emergent setting since some small abscesses are not seen on ultrasound. At CT, renal abscesses tend to be round and centrally hypodense, with central liquefaction, and may demonstrate significant rim enhancement at post-contrast imaging, but no internal enhancement (Fig. 7.8a). Gas may be seen within the abscess. A hypoenhancing halo may

surround the abscess in the renal parenchyma during the nephrographic phase. In many cases, the entire kidney is enlarged or there may be a focal mass/bulge in the renal contour. Perinephric inflammation and thickening of Gerota's fascia are also commonly seen. A perirenal abscess tends to manifest as a fluid collection in the perinephric space and may contain gas. Extra-parenchymal collections have been found to extend into the adjacent psoas muscle [10].

On ultrasound, a renal abscess tends to be a hypoechoic thick-walled structure with increased through transmission but less so than that seen with a simple cyst of similar size. Typically, no internal vascularity is seen. Debris may be seen within the abscess (Fig. 7.8b).

Renal abscesses on MRI are centrally T2 hyperintense and T1 hypointense and tend to have a thick wall which shows variable enhancement on T1-weighted post-contrast imaging (Fig. 7.9). Perinephric inflammatory stranding may also be seen. Findings may be difficult to differentiate from cystic renal cell carcinoma; the patient's clinical symptoms and subsequent resolution on follow-up imaging help make this distinction.

Emphysematous Pyelonephritis

Emphysematous pyelonephritis is a life-threatening emergency of the kidneys, caused by gas-forming bacteria which infect the renal parenchyma, collecting system, and/or

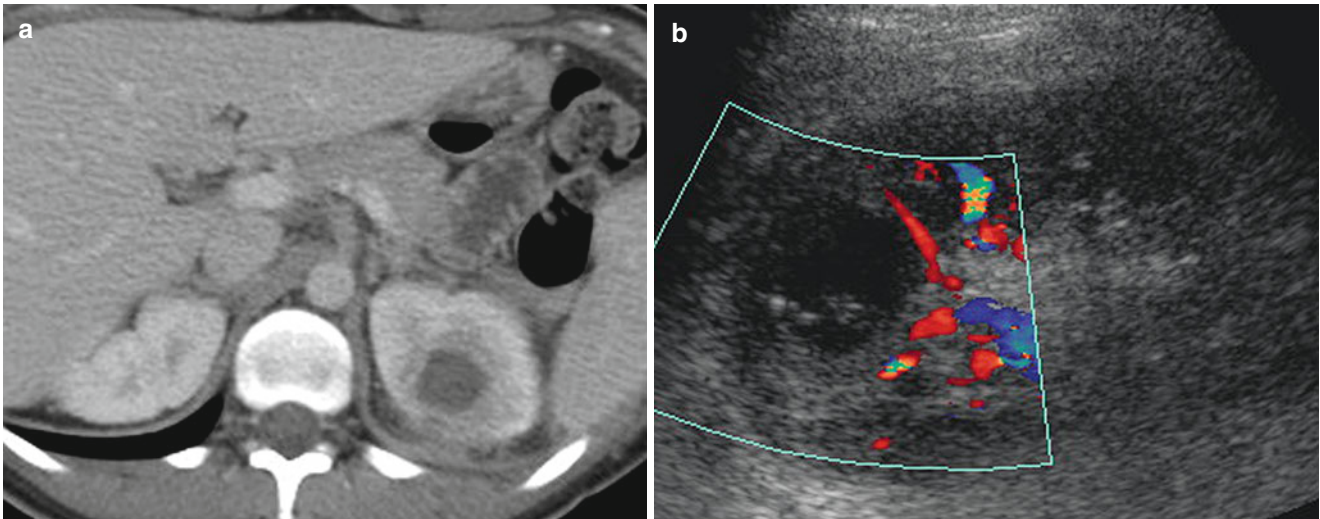


Fig. 7.8 Left renal abscess in a 26-year-old female with left flank pain and fever. **(a)** Contrast-enhanced axial CT image shows hypodense area in the left kidney with central more hypodense, necrotic core measuring

2.1×2.2 cm with adjacent perinephric fat stranding. **(b)**. Ultrasound images of the left kidney show 2.3×2.1 cm hypoechoic, thick-walled structure containing echogenic internal debris and no internal vascularity

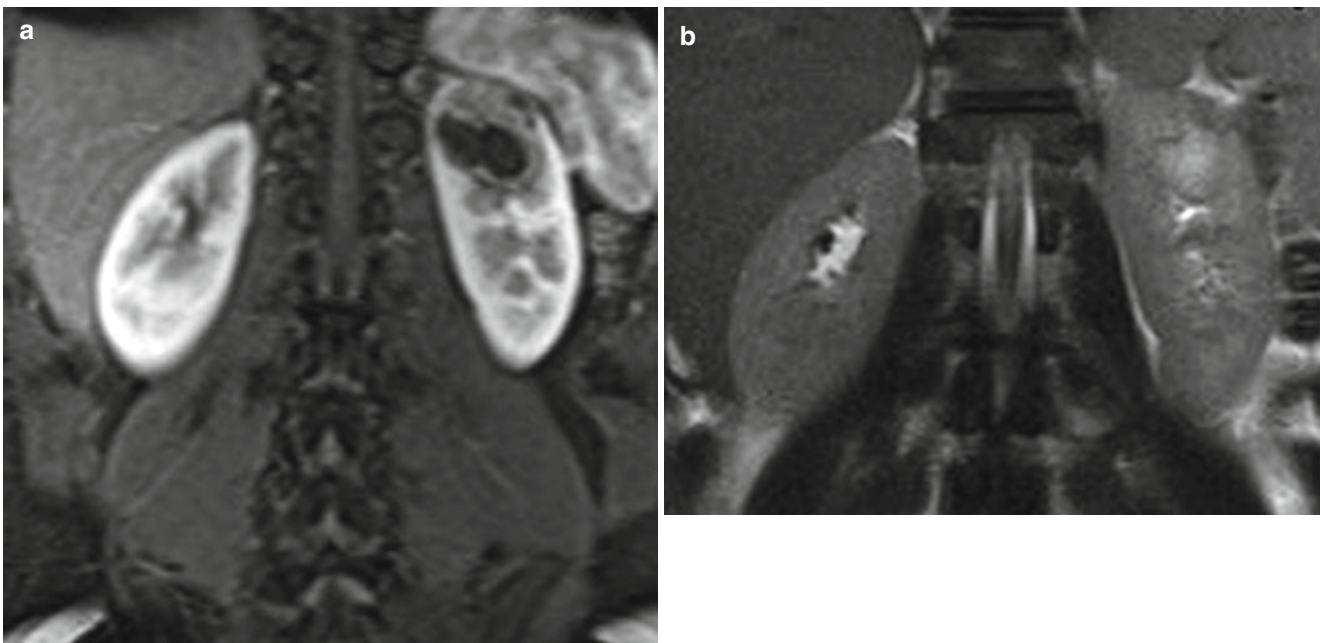


Fig. 7.9 Right renal abscess on MRI in an 18-year-old female with left upper quadrant pain and elevated WBC. **(a)** Contrast-enhanced T1-weighted coronal image demonstrates peripherally enhancing

thick-walled cystic lesion. **(b)** T2-weighted coronal image shows T2 hyperintense focus in the left renal upper pole

perinephric tissue. It is highly associated with poorly controlled diabetes mellitus, present in up to 90 % of cases. Obstruction of the corresponding urinary collecting system secondary to calculus, neoplasm, and/or sloughed papilla may also be present [13, 14]. The bacteria most commonly linked to emphysematous pyelonephritis are *E. coli* (~70 %) and *Klebsiella pneumoniae* [14]. If gas is localized to the renal collecting system and does not involve the renal

parenchyma, the term emphysematous pyelitis is used, which is less severe than emphysematous pyelonephritis, with lower mortality.

The presenting symptoms of emphysematous pyelonephritis include fever, flank pain, and pyuria, which are nonspecific and also seen in other urinary tract infections [14]. Patients may also present with lethargy/altered mental status, acid–base abnormalities, hyperglycemia, renal failure,

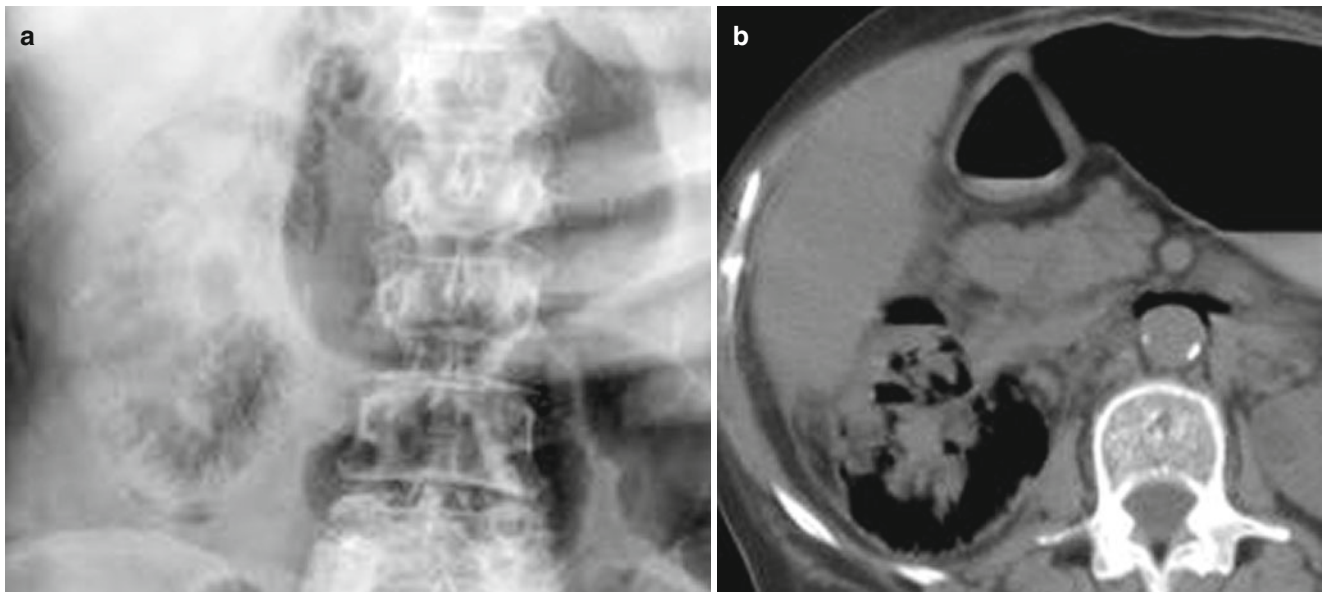


Fig. 7.10 Emphysematous pyelonephritis. (a) Scout image shows mottled lucencies projecting over the right renal shadow. (b) Non-contrast-enhanced axial CT image of the same patient demonstrates gas

within the renal parenchyma (Images courtesy of Leonora Mui at Columbia University Medical Center, New York, NY)

and, in severe cases, septic shock. Emphysematous pyelonephritis is seen more often in females than in males presumably because females are more susceptible to urinary infections. On physical exam, patients may have flank pain and, rarely, crepitus may be felt over the lower back [13].

Imaging

On radiography, evidence of gas overlying the renal shadows or radially oriented gas aligning with the renal pyramids may be seen (Fig. 7.10a). In severe cases, one may see crescent-shaped gas collection involving Gerota's fascia. Findings may also include obscuration of the ipsilateral psoas muscle shadow [13].

CT best demonstrates the extent of gas and destruction associated with emphysematous pyelonephritis. Imaging findings include foci of gas within the renal parenchyma/collecting system, renal enlargement and destruction, fluid collections with possible gas-fluid levels, and tissue necrosis (Fig. 7.10b) [10]. Two CT imaging types have been described: type I with streaky or mottled gas collections without fluid collections and type II with bubbly gas in the renal parenchyma/collecting system associated with renal/perirenal fluid collections [10, 13]. Mortality rates are 69 % for type I and 18 % in patients with type II [10, 13].

Sonographic evaluation of the kidneys in emphysematous pyelonephritis may demonstrate an enlarged kidney that contains echogenic foci with posterior dirty shadowing, indicative of gas within (Fig. 7.11). The echogenic foci representing gas may be confused with renal calculi. The true extent of

emphysematous renal involvement can be underestimated on ultrasound [13].

Treatment and Complications

Initial treatment includes aggressive IV hydration, correction of electrolyte imbalances, and broad-spectrum IV antibiotics [13]. If gas is diffuse throughout the kidney, nephrectomy is often indicated. If infection is more localized and preservation of kidney is a priority, percutaneous drainage can be attempted.

Emphysematous Cystitis

Emphysematous cystitis is a rare infection of the bladder caused by gas-producing organisms, most commonly *E. coli*, *Enterobacter aerogenes*, and *Klebsiella pneumoniae* [15]. It tends to be seen in elderly patients with diabetes, with diabetes reported in over half of cases. Elderly or debilitated patients with chronic urinary retention secondary to urinary bladder outlet obstruction, neurogenic bladder, or bladder structural abnormality, as well as patients with an indwelling urinary bladder catheter, are at increased risk of developing emphysematous cystitis in part due to urinary stasis. It has been speculated that organisms within the urinary bladder cause fermentation of urinary glucose, leading to release of carbon dioxide bubbles that collect in the submucosa or lumen of the bladder [15, 16].

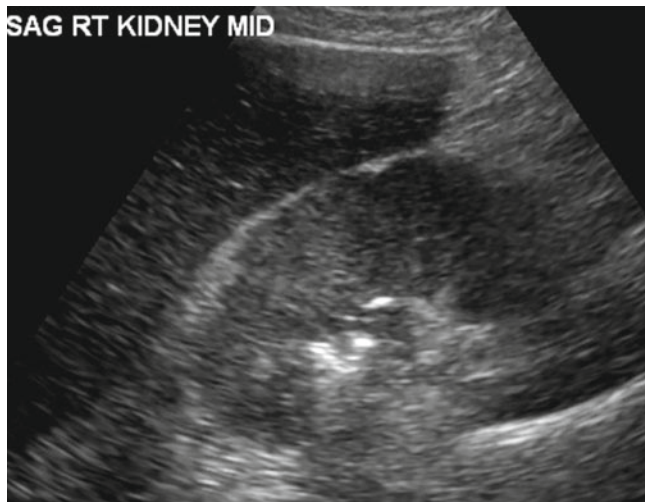


Fig. 7.11 Emphysematous pyelonephritis. Ultrasound image of the right kidney shows echogenic foci with dirty shadowing within the right kidney, consistent with gas

The presenting symptoms include dysuria, hematuria, and increased urinary frequency. The incidence of this infection is greater in females than males, 2:1 [13].

Imaging

Radiography is highly sensitive in detecting emphysematous cystitis (~97 %) [17]. On radiography, one may see curvilinear lucency, consistent with gas, outlining the bladder wall, with or without intraluminal gas (Fig. 7.12a).

In patients with emphysematous cystitis (EC), gas is seen within the wall of the urinary bladder and possibly intraluminal, particularly in the absence of recent instrumentation or fistulous communication with a hollow viscus (Fig. 7.12b). CT can detect cases of EC that are not apparent on radiography and can better depict the extent of disease. CT also helps differentiate emphysematous cystitis

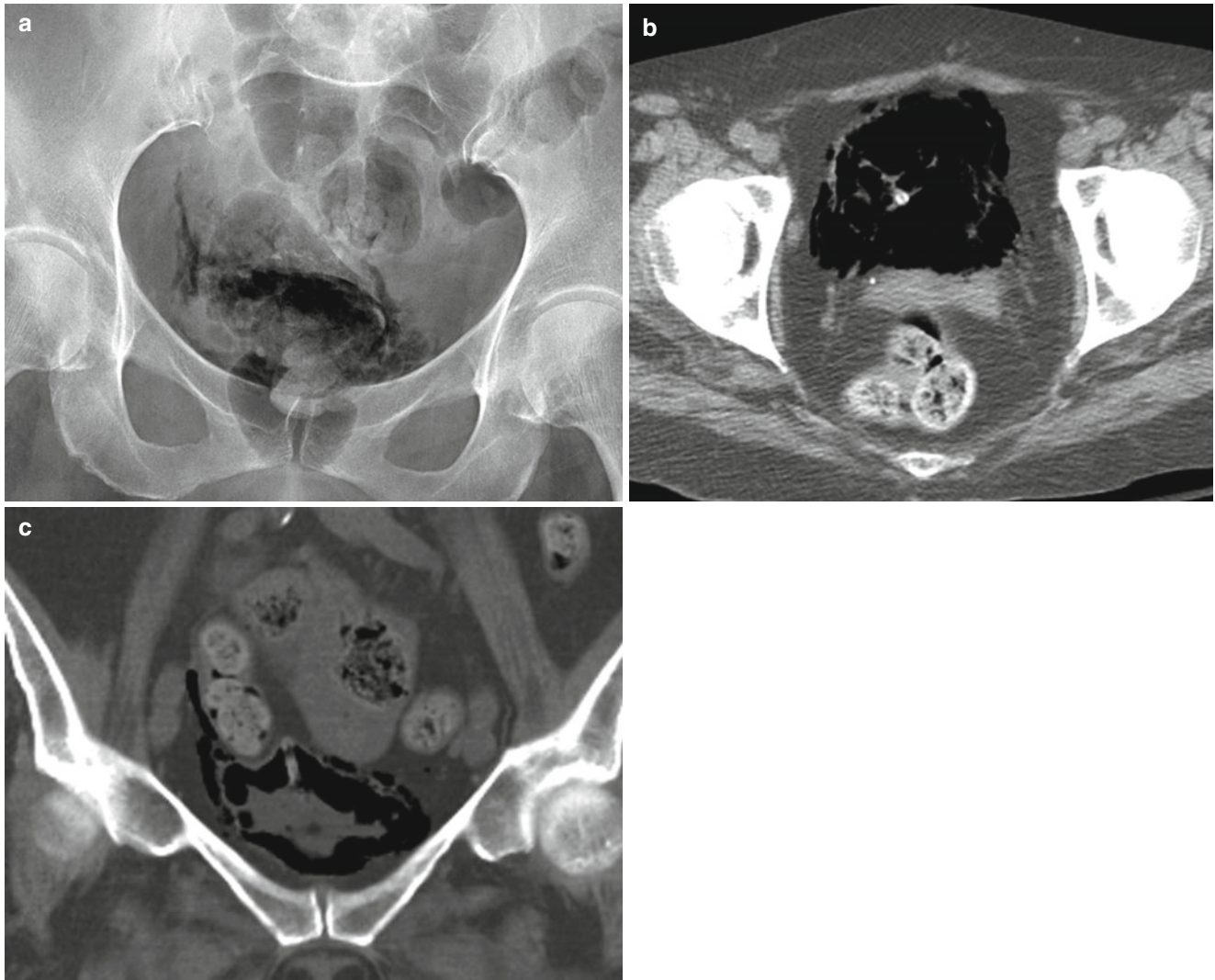


Fig. 7.12 Emphysematous cystitis in a 62-year-old female with right flank pain and UTI. (a) Pelvis radiograph shows pelvic lucencies consistent with bladder gas as confirmed on CT. (b) and (c) CT images

demonstrate extensive gas within the lumen and wall of the urinary bladder. Coronal image depicts dissection of gas in the right space of Retzius

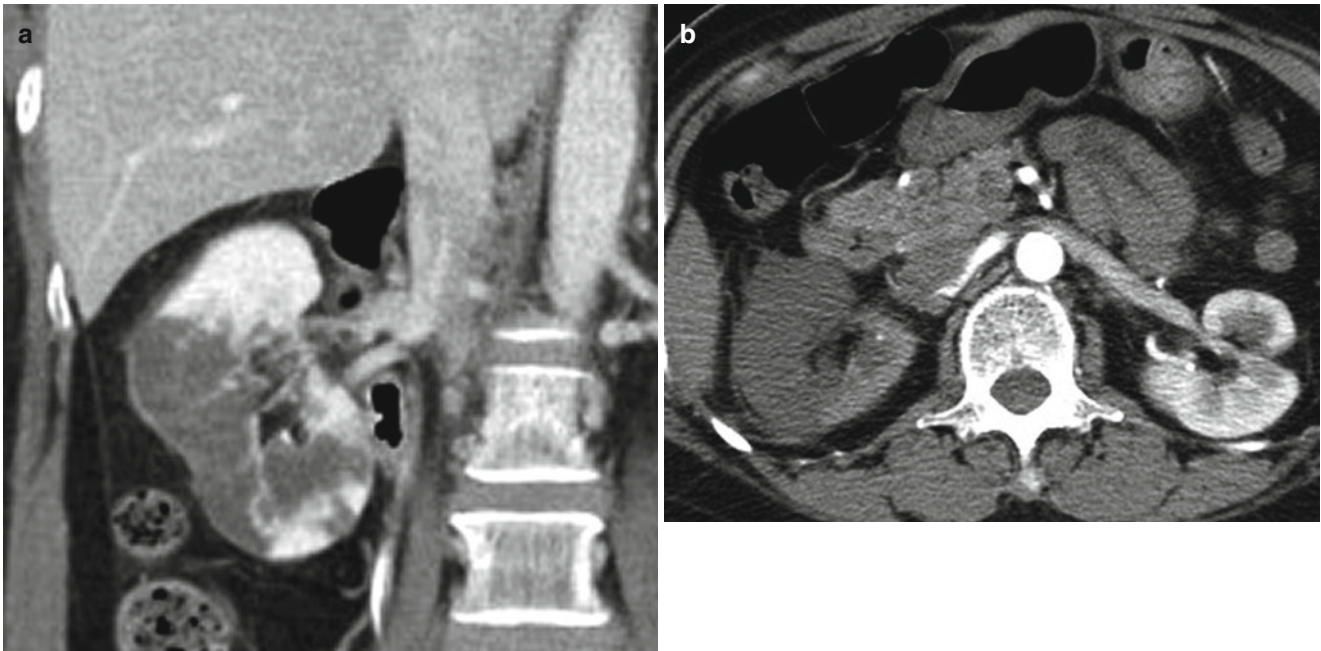


Fig. 7.13 Renal infarct in a 50-year-old female with right abdominal pain. **(a)** Contrast-enhanced coronal image of the right kidney shows wedge-shaped areas of hypodensity involving the cortex and medulla

with preservation of the capsule. **(b)** Contrast-enhanced axial CT image obtained 10 days later shows lack of flow in the distal right renal artery and significantly worsened lack of right renal perfusion/enhancement

from other causes of pelvic gas, including abscess or vesicocolic fistula [13].

Treatment

Emphysematous cystitis is typically treated with early broad-spectrum antibiotics and bladder drainage with Foley catheter. If disease progresses to involve the ureters or kidney(s), surgery may be required.

Renovascular Emergencies

Acute alterations in the renal vasculature can lead to various changes in the kidney, some of which require intervention. Renovascular emergencies include renal infarct and renal vein thrombosis.

Renal Infarction

Renal infarcts may develop secondary to thrombotic disease, embolism, aortic dissection, or trauma. Sources of thrombosis include arteriosclerosis, vasculitis, and aortic or renal artery aneurysm. Renal artery embolism is most commonly cardiac in nature but can also be iatrogenic, such as following catheterization.

The presenting symptoms in patients with renal infarction tend to be flank pain with possible hematuria [12].

Imaging

CT is the imaging modality of choice in evaluating for renal infarction. The size and location of the renal infarct depends on the size of the embolus, location of arterial occlusion, and the age of the event. At contrast-enhanced CT, findings in renal infarct are one or more wedge-shaped regions demonstrating lack of enhancement that involve both the renal cortex and medulla (Fig. 7.13) [18]. Cortical rim enhancement of the kidney can be seen adjacent to a renal infarct due to collateral capsular perfusion and usually appears several days after onset of the infarct [19]. Chronic renal infarct results in scarring and loss of renal cortex.

An acute renal infarct may have a normal grayscale appearance on ultrasound. Alternatively, a wedge-shaped hypoechoic area may be present in the renal parenchyma, which may be difficult to distinguish from pyelonephritis without the use of color Doppler. With color Doppler, there will be absence of flow to a portion of or to the entire kidney.

Treatment

The mainstay of treatment for acute renal infarct is anticoagulation.

Renal Vein Thrombosis

Renal vein thrombosis is commonly due to an underlying disorder of the kidney or of the clotting system. It can also be seen in dehydration, regional neoplastic processes (involving the kidney, adrenal gland, and/or ureter) with direct extension, and trauma [18, 19].

The clinical presentation of renal vein thrombosis can vary depending on the etiology of the thrombosis. Presenting symptoms include flank pain, gross hematuria, and decrease or loss of renal function [19]. Renal vein thrombosis is more often seen on the left, which is thought to be due to the greater length of the left renal vein compared to the right. Membranous glomerulonephritis is the most common underlying disorder in adults with renal vein thrombosis [19].

Imaging

Patients suspected of having renal vein thrombosis are often first imaged with ultrasound. On ultrasound, one may see enlargement of the affected kidney and a hypoechoic renal cortex due to underlying edema. Later, the kidney decreases in size and becomes hyperechoic. On Doppler ultrasound examination, the findings include absence of venous flow, echogenic material with the renal vein lumen consistent with thrombus, reversal of arterial diastolic flow, and elevated renal artery resistive index due to high resistance.

In acute renal vein thrombosis, CT imaging demonstrates a hypoattenuating filling defect consistent with thrombus within a thickened renal vein, and the vein may be distended (Fig. 7.14). Enhancement of the thrombus suggests tumor thrombus. The kidney on the affected side may be enlarged, and there may be delay in the renal cortical nephrogram and an imaging appearance of a diminished nephrogram [18, 19]. Additionally, perinephric edema may be present. Once chronic, renal vein thrombosis leads to attenuation of the vein due retraction of the thrombus and regional collateral vessels can be seen.

Treatment and Complications

Anticoagulation is the mainstay of treatment in renal vein thrombosis. Thrombolytic therapy has been used in cases of bilateral renal vein thrombosis with acute renal failure, large clot burden with high risk of additional embolic events, and pulmonary embolism. Complications of renal vein thrombosis include acute renal failure, extension of the thrombus into the IVC, and elevated risk of pulmonary embolism.

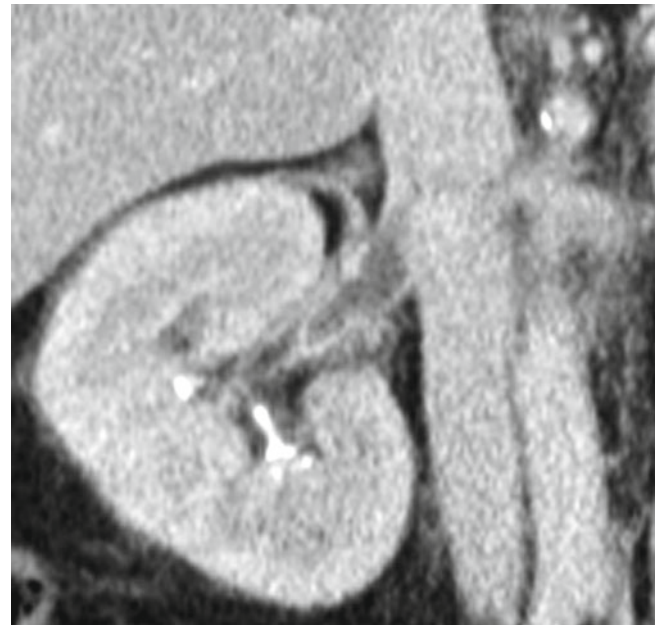


Fig. 7.14 Renal vein thrombosis in a 57-year-old female with abdominal pain and history of membranous glomerulonephritis. Contrast-enhanced coronal image shows filling defect within a distended right renal vein

Renal Transplant

With increase in renal transplantation in recent years, a greater number of renal transplant patients may present to emergency departments, and it is thus important to be aware of the potential transplant complications. In the emergent setting, evaluation of renal transplant is typically performed with ultrasound. Complications may be urologic or vascular and include peritransplant fluid collections.

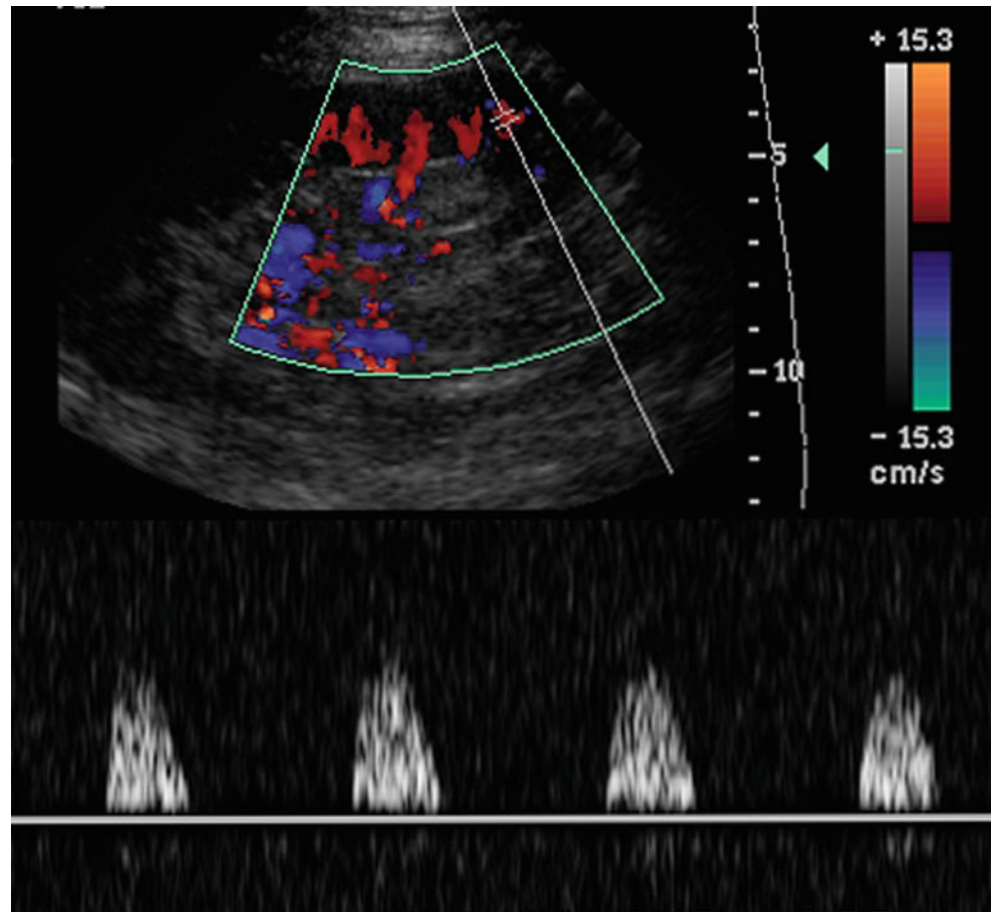
Ultrasonography

Peritransplant fluid collections can be partially differentiated based on time interval since transplantation. Small hematomas are commonly seen postoperatively and usually resolve spontaneously. They are echogenic when acute, and become less echogenic over time, and may contain septations [20].

Urinomas are well-defined, simple-appearing, anechoic collections that tend to occur between the urinary bladder and transplant kidney during the first 2 weeks after transplantation [20].

Lymphoceles are collections that tend to develop within 1–2 months posttransplantation, although may occur weeks to years thereafter and are usually seen between the transplant and the bladder. Sonographically, lymphoceles tend to be anechoic.

Fig. 7.15 Lack of diastolic flow. A 59-year-old female status post renal transplant with increasing creatinine. Doppler ultrasound image waveform shows lack of diastolic flow. Renal vein was patent. Differential diagnosis ATN vs. rejection



Large perinephric fluid collections can cause hydronephrosis, with lymphoceles being the most common [20]. All perinephric fluid collections can become infected and demonstrate more a complex cystic appearance with possible abscess formation. Peritransplant abscesses are rare and tend to develop during the first few weeks posttransplantation [20].

Renal transplant vascular complications include renal vein thrombosis and renal infarction, with imaging characteristics similar to that described above. Of note, in renal vein thrombosis, on color Doppler imaging, there may be lack of or reversal of diastolic flow in the intraparenchymal arteries, which may also be demonstrated in acute rejection or acute tubular necrosis (ATN). Renal vein thrombosis is differentiated from acute transplant rejection or ATN by lack of flow and thrombus in the main renal vein (Fig. 7.15).

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