Current Problems in Diagnostic Radiology 48 (2019) 616–625

Current Problems in Diagnostic Radiology

journal homepage: www.cpdrjournal.com

Name That Nephrogram: Asymmetric Renal Enhancement in the Acute Care Setting

Staci M. Gagne, MD, Alex Newbury, MS, Kristina M. Nowitzki, MD, PhD, Byron Y. Chen, MD, Hao S. Lo, MD*

University of Massachusetts Medical School, Department of Radiology, Worcester, MA.

Disorders of the kidney and urinary collecting system are common encountered in the acute care setting. Computed tomography has progressively replaced intravenous pyelography for the evaluation of most urinary tract pathology including acute flank pain, suspected malignancy, congenital abnormalities, anatomical variants, and inflammatory/vascular conditions through evaluation of the "nephrogram" produced by intravenous contrast material filtering through the kidneys.

In this review, we describe the most common types of abnormal nephrograms seen on renal computed tomography, and highlight the salient features and conditions associated with them, in addition to a pictorial review with specific and interesting related cases. The types of abnormal nephrograms reviewed are absent, unilateral delayed, striated, spotted, and persistent.

Introduction

Disorders of the kidney and urinary collecting system are commonly encountered in the emergency and acute care setting. Over time, computed tomography (CT) has superseded intravenous pyelography for evaluation of the kidneys in the acute care setting. CT has become the study of choice due to its ubiquitous accessibility, fast image acquisition, and improved spatial resolution. Additionally, the contraindications for contrast-enhanced CT exam are minimal (ie, pregnancy, severe reaction to iodinated contrast, and renal failure). In 1 recent study of emergency department visits for "flank pain," 45.5% of patient underwent CT in 2008. In 2000, only 19.6% underwent CT for the same indication.[1] Normal renal physiology results in 3 patterns of contrast enhancement, termed "nephrogram." When the normal renal functioning is disrupted, the nephrogram can morph into one of several distinct enhancement patterns. In this article, we will review normal phases of renal contrast enhancement, pathophysiological categories leading to disrupted function, resulting abnormal patterns of enhancement, and differential diagnoses associated with each pattern.

Normal Renal Contrast Enhancement

In the absence of intravenous contrast, the cortex and medulla of the normal kidney have a density of 30-40 Hounsfield units (HU).[2] Once intravenous contrast is administered to the kidney, it is actively concentrated and secreted. Three distinct imaging (radiographic or CT) phases, correlate to the contrast traveling through the kidney. These phases follow a predictable progression after intravenous contrast administration (Table 1).

Intravenous contrast first briskly enters the kidneys through the main renal arteries. The contrast travels from the main renal arteries into the branching segmental arteries. In the renal sinus, the segmental arteries branch into lobar arteries. The segmental arteries divide into interlobar arteries which course through the columns of Bertin. At the corticomedullary junction, the interlobular arteries continue as arcuate arteries. Lastly, the arcuate arteries branch into interlobular arteries that divide into afferent arteriole.[3] At this stage, when contrast is in the rich distribution of small arteries, the cortex governs renal enhancement. Subsequently, contrast fills the tubules and collecting ducts, manifesting as medullary enhancement and distinction between the cortex and medulla disappears.[4] The contrast material is excreted by glomerular filtration, without tubular excretion or reabsorption, and exits via the renal collecting system.[5]

The first imaging phase, known as the *corticomedullary* phase, occurs 15-60 seconds after intravenous contrast administration. The cortex and medulla reach maximal density differentiation in this phase. The appearance of the kidney reflects contrast reaching the capillaries within the cortex. The maximal differentiation of the cortex and medulla is at 40-50 seconds. At this time, the cortex has a density of 145-185 HU, while the medulla has a density of 50-90 HU. Concurrently, the aorta is avidly opacified with contrast while the inferior vena cava (IVC) remains nonopacified.[2]

The next phase, termed the *nephrographic* phase, occurs at 90-120 seconds after intravenous contrast administration. The cortex and medulla demonstrate equal enhancement giving the

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflicts of interest: None.

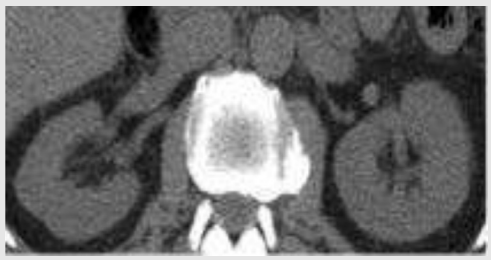
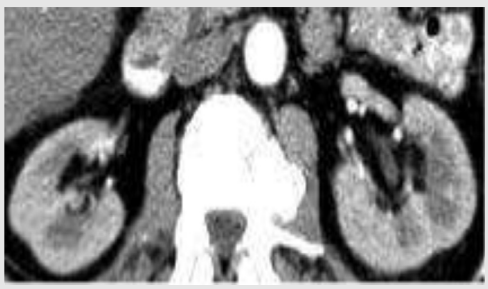
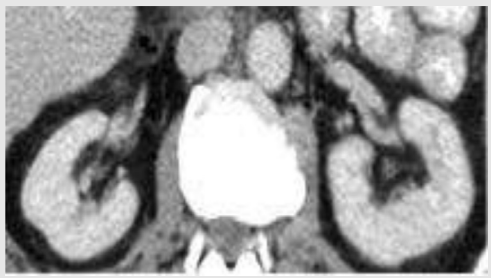
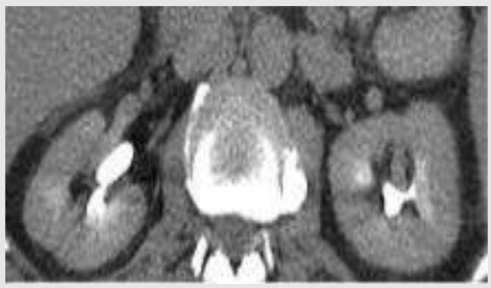
*Reprint requests: Hao S. Lo, MD, University of Massachusetts Medical School, Department of Radiology, 55 Lake Ave North, Worcester, MA 01655.

E-mail address: Hao.Lo@umassmemorial.org (H.S. Lo).

https://doi.org/10.1067/j.cpradiol.2018.10.004

0363-0188/

TABLE 1
Normal phases of renal contrast enhancement

Phase	Imaging appearance	Timing	Hounsfield units (HU)	Additional info
Noncontrast |(1a)		0s	Cortex, medulla: 30-40 HU	
Corticomedullary (1b)		15-60s	Cortex: 145-185 HU Medulla: 50-90HU	Cortex enhances briskly as contrast fills cortical capillaries.
Nephrographic (1c)		80-120s	Cortex and medulla: 120-170	Contrast is filtered by glomeruli, enters loops of Henle and collecting ducts.
Excretory (1d)		3-5 min	Cortex and medulla: Simultaneously decreases to 30-40 as contrast material leaves these areas	Contrast fills the calyces and renal collecting system

kidney a homogenous appearance. During this phase, the contrast is filtered by glomeruli and enters the loops of Henle and collecting ducts.[2] During this phase, the aorta and IVC show roughly equal density, due to similar intravascular contrast concentration.

The last phase is the called the *excretory* phase. This occurs at 3-5 minutes after contrast administration. The contrast is excreted, filling the calyces and renal collecting system. The cortex and medulla are homogeneous in density, but the attenuation is lower compared to the nephrogenic phase due to the movement of the contrast material out of the parenchyma. In this phase, contrast is no longer visible within the IVC or aorta.[2]

Abnormal Nephrogram

General

The 4 basic pathophysiologies causing the appearance of an abnormal nephrogram relate to vascular inflow, vascular outflow, nephron function, and urinary outflow (Table 2). The variety of specific pathologic conditions causing an abnormal nephrogram can be categorized into congenital, obstructive, vascular, traumatic, infectious, inflammatory, and neoplastic etiologies. The types of abnormal nephrogram patterns discussed here are absent, unilateral delayed, striated, spotted, and persistent.[6] Other previously described nephrogram patterns include rim, reverse rim, and unilateral hyperdense.

TABLE 2
Causes of absent nephrogram

Pathophysiology	Etiologies
No vascular inflow (Arterial occlusion)	Transection Dissection Hematoma Thromboembolic disease
No vascular outflow (Venous occlusion)	Hypercoagulable state Tumor invasion Nephrotic syndrome
No nephron function	Infiltrative masses (lymphoma, diffuse transitional cell carcinoma, metastasis) Acquired, nonmalignant (xanthogranulomatous pyelonephritis, tuberculosis autonephrectomy) Congenital (renal agenesis, hypogenesis, ectopia)
No urine output	Multicystic dysplastic kidney

Absent Nephrogram—Global versus Segmental

The absent nephrogram refers to the lack of functioning renal parenchyma, and can be categorized as global or segmented, depending on the pattern of decreased uptake of contrast material.[4,7] The most common cause for a globally absent nephrogram is complete arterial occlusion, classically occurring in the setting of blunt abdominal trauma with renal pedicle injury.[8] Common presentations of blunt trauma are motor vehicle accident, fall, or direct impact flank injury. In these circumstances, the renal pedicle undergoes a deceleration force, with compression against the rib cage or vertebral column.[8] The result is transection or tear of the main renal artery (Fig 1) or obstructing perinephric hematoma, resulting in compromised vascular inflow and a globally absent nephrogram. A nontraumatic dissection including the renal artery may also cause a similar result to a traumatic transection or tear (Fig 2). Other etiologies of complete arterial occlusion include thromboembolic diseases, most commonly

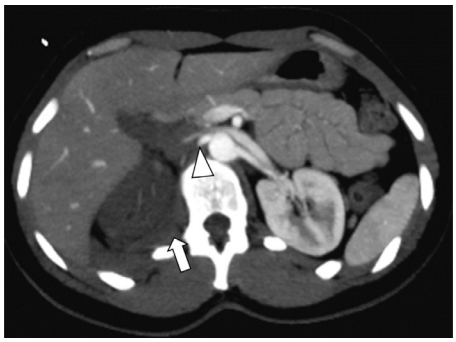


FIG 1. 33-year-old male presents after a motor vehicle accident. An axial arterial phase maximum intensity projection (MIP) image with essentially absent nephrogram in a normal size right kidney. There is a pararenal hematoma (arrow). Abrupt cutoff of the right main renal artery near its origin (arrowhead) indicates complete transection. A normal corticomedullary nephrogram is seen in the left kidney.

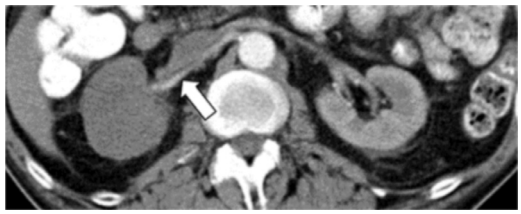


FIG 3. 69-year-old male presents with right flank pain. Axial arterial phase image with absent nephrogram in the right kidney. There is loss of the normal renal sinus fat. A normal right main renal artery is seen (arrow). Additional sections through the lung bases and liver (not shown) showed diffuse metastatic disease. A normal corticomedullary nephrogram is in the left kidney.

associated with atrial fibrillation. Several studies implicate atrial fibrillation in 40%-47% of renal infarction cases secondary to renal artery occlusion.[9,10] In general, the CT appearance of an absent nephrogram is a kidney of normal size and contour, with no parenchymal enhancement following contrast administration.[4] Additionally, there may be perinephric hemorrhage and absence of urine excretion into the ipsilateral ureter and collecting duct.[11]

The segmental absent nephrogram can be the result of several different pathologies. Consequently, it can present with multiple unique CT appearances. Space-occupying lesions such as neoplasm, cysts, and abscesses can displace the renal parenchyma, causing a disruption in the CT contrast uptake. Peripheral renal intravascular occlusive pathologies prevent contrast from reaching distalmost locations, resulting in a multifocal cortical abnormality.[4] These patchy wedge-shaped areas of decreased attenuation fail to enhance during the corticomedullary and nephrographic phases of a CT contrast study. They can result from local thrombus formation, vascular tumor invasion, or thromboemboli.[4,7,12] Other causes of segmented arterial occlusion include hypercoagulable states such as hyperhomocysteinemia and paroxysmal nocturnal hemoglobinuria.[13]

In the event of main renal vein complete occlusion (ie, renal vein thrombosis), blood flow is prevented from exiting the kidney resulting in an absent nephrogram. This condition is observed less commonly than renal artery occlusion. Complete renal vein occlusion can be seen in hypercoagulable disorders such as nephrotic syndrome and antiphospholipid syndrome, and tumor extension such as from renal cell carcinoma, Wilm tumors, and metastatic disease (Fig 3, 16, and 17).[7,14] Most cases result in incomplete occlusion because development of peripheral collaterals prevents complete loss of blood outflow.[15] The affected kidney appears enlarged and edematous, vis-a-vis the relatively normal-appearing kidney in acute arterial occlusion.[7]

The absent nephrogram arising from nephron absence or dysfunction includes acquired and congenital etiologies, such as renal agenesis, hypogenesis, ectopia, surgical nephrectomy, and chronic parenchymal atrophy/scarring.[7] Two uncommon, nonmalignant, acquired causes of absent nephrons mimic renal masses: They are xanthogranulomatous pyelonephritis (XGP) and tuberculous autonephrectomy. XGP is a chronic destructive granulomatous process

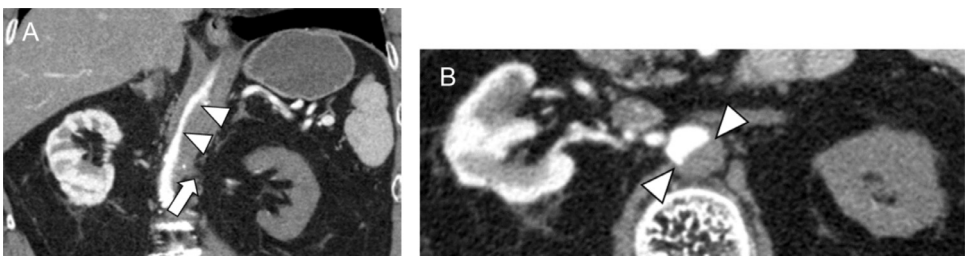


FIG 2. 69-year-old female presents with chest pain. (A) Coronal and (B) axial arterial phase images with absent nephrogram in the left kidney. There is a long segment thrombosed aortic dissection (arrowheads) involving the left renal artery (arrow). The right kidney demonstrates a normal corticomedullary nephrogram.

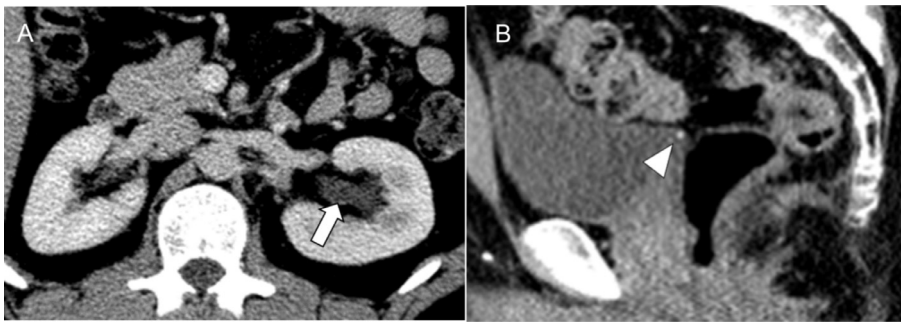


FIG 4. 28-year-old male presents with flank pain. (A) The venous phase of intravascular contrast, with contrast noted in the IVC on this axial CT image, indicates that the right nephrographic phase is normal with a delayed corticomedullary nephrogram on the left. Mild hydronephrosis is also seen in the left kidney (arrow). (B) Additional sagittal view of the bladder in the same patient demonstrates a punctate stone in the most dependent portion of the urinary bladder, settling there after having just passed through the left ureter (arrowhead).

thought to result from an atypical immune response to subacute recurrent bacterial infection. It is more common in the middle aged and women. XGP leads to the eventual destruction replacement of normal renal parenchyma with reactive tissue predominantly consisting of lipid-laden macrophages and often an associated staghorn calculus.[16,17] On CT, the normal renal borders are lost and enlarged as the renal pelvis contracts, and the calyces dilate in a multiloculated pattern containing fat density known as the "bear's paw sign" (Fig 20).[18]

Similarly, renal tuberculosis, also known as tuberculous autonephrectomy, leads to a loss of nephron function and an absent nephrogram. Renal involvement of tuberculosis can be from a localized urinary infection or from dissemination from a primary source, most often from the lungs.[19] While the granulomas can remain stable for several years, the *Mycobacterium tuberculosis* organisms will preferentially invade the renal medulla, resulting in extensive areas of papillary necrosis and vascular insufficiency.[20] As the disease progresses, infundibular and pelvic stenosis occur. The end result is parenchymal destruction, dystrophic calcification, and ultimately, loss of renal function.[21] Early imaging can show papillary necrosis and uneven caliectasis, progressing to mural thickening and enhancement with potential focal hydronephrosis. End-stage disease results in cortical thinning secondary to chronic, progressive hydronephrosis. CT shows amorphous dystrophic calcifications involving the whole kidney, resulting in a "putty kidney" appearance.[22]

Lastly, complete disruption in urine output is an uncommon pathophysiological cause of an absent nephrogram. It can be seen with multicystic dysplastic kidney. Multicystic dysplastic kidney is an acquired disorder seen in about 1:4300 live births, typically diagnosed via fetal ultrasound.[23,24] Noncommunicating benign cysts replace normal renal parenchyma, as the ascending ureteric bud fails to induce differentiation during embryogenesis.[25] The consequent loss of normal functioning nephrons leads to the complete lack urine output and an absent appearance on nephrogram.

Unilateral Delayed Nephrogram

Alterations in the normal temporal progression of the nephrographic process are the consequence of unilateral decreased blood or urine flow, resulting in a delayed nephrogram. The most common cause of slow urine output causing a unilateral delayed nephrogram is obstructive uropathy from ureteric calculus (Figs 4 and 18), blood clot, tumor, or extrinsic compression (Table 3). The nephrogram commonly shows a dilated renal pelvis proximal to the site of obstruction. Excretory phase imaging via CT urogram is particularly useful for differentiating intraluminal versus extrinsic compressive causes.[6]

An unilateral delayed nephrogram can also be caused by slow vascular inflow or outflow. Renal artery stenosis (RAS) and subcapsular perinephric hematomas are 2 causes of delayed

TABLE 3
Causes of unilateral delayed nephrogram

Pathophysiology	Etiologies
Slow vascular inflow	Renal artery stenosis, subcapsular hematoma
Slow vascular outflow	Renal vein compression
Poor nephron function	Unilateral pyelonephritis
Slow urinary outflow	Obstructive uropathy (stones, blood clot, tumor, lymphadenopathy)

inflow. RAS can be caused by several pathologies, including atherosclerosis, fibromuscular dysplasia, aortic dissection, and vasculitides (ie, polyarteritis nodosa and radiation fibrosis).[26] Most cases of RAS are unilateral and associated with secondary hypertension, resulting from an inappropriately activated Renin-Angiotensin-Aldosterone (RAA) system. The classic delayed nephrogram shows an asymmetric progression and prolongation of the cortical nephrographic phase and persistent corticomedullary differentiation on the affected side (Fig 19).[27] Subcapsular hematoma refers to the accumulation of blood products deep to the renal capsule. The high-pressure fluid collection decreases vascular inflow by compressing the adjacent arterial vasculature. Similar to RAS, patients with subcapsular hematomas can present with secondary hypertension as a result of RAA system activation[7]. Most commonly seen in the acute traumatic setting, these hematomas are identified by a smoothly crescentic distorted renal contour with an adjacent hyperdense collection on CT (Fig 5). Also, acute trauma may cause a vascular injury without fully compromising vascular flow to the kidney, resulting in a delayed, rather than absent, nephrogram (Fig 6).

Renal vein compression by any cause slows vascular outflow from the kidney, resulting in a unilateral delayed nephrogram. One such etiology is left renal vein compression between the superior mesenteric artery and aorta, known as "nutcracker syndrome" (Fig 7).

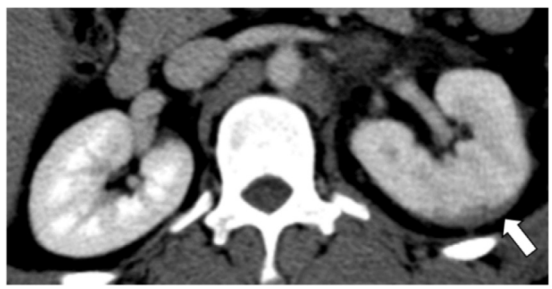


FIG 5. 35-year-old male presents after motor vehicle accident. There is a delayed nephrogram on the left with a small subcapsular hematoma (arrow). Irregularity of the left posterior cortex is consistent with laceration.

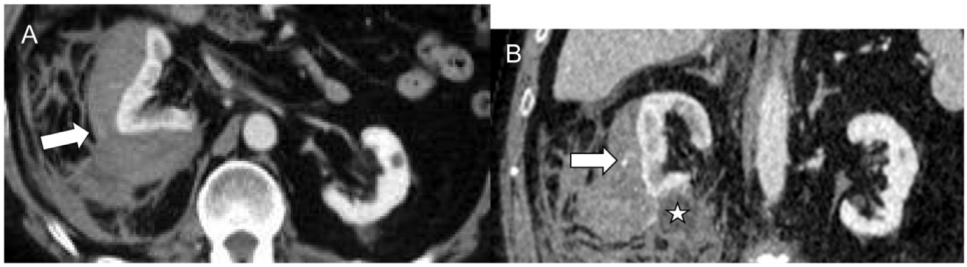


FIG 6. 73-year-old female on Rivaroxaban with acute flank pain. (A) On this axial contrast-enhanced CT image, there is a large subcapsular hematoma in the right kidney (arrow) with mass effect and causing a delayed corticomedullary nephrogram. (B) This coronal CT image shows extravasation of intravenous contrast (arrow), consistent with ongoing hemorrhage. An incidental small cyst is also present in the right lower pole (star).

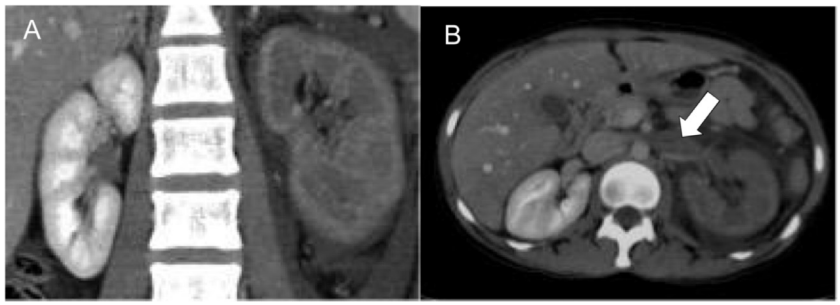


FIG 7. 32-year-old male presents with abdominal pain and hematuria. (A) A coronal CT image shows an enlarged, edematous left kidney with an extremely delayed corticomedullary nephrogram. The renal arteries are opacified, but no contrast is seen within the left renal vein. (B) Axial view confirms large filling defect within the left renal vein (arrow). External compression of the left renal vein by the superior mesenteric artery (i.e. Nutcracker syndrome) was suspected.

Elevated venous resistance causes flow disturbance and intimal injury within the veins draining the collecting system, leading to hematuria and the potential for renal vein thrombosis.[28,29] On CT, there is a reduced aortic-SMA angle (less than 45 degrees) and collateralization via the left gonadal vein, demonstrating early enhancement during the portal venous phase.[29]

Poor nephron function due to acute pyelonephritis can lead to a unilateral delayed nephrogram. It often presents with severe flank pain, high fevers, and potential sepsis.[30] It is 5 times more common in females than males. The most common causative agent is *Escherichia coli*, although other bacteria such as *Klebsiella, Proteus, Enterobacter*, and *Pseudomonas* have the necessary virulence factors to adhere to the urothelium.[31] CT urography is 1 option for imaging, although a single 45-90 second postcontrast acquisition usually provides enough information depending on the degree of pathology.[32] Postcontrast CT typically shows focal regions of edema, hypoenhancement and persistent delayed enhancement, which can last up to 6 hours, as a result of poor vascular flow through the affected region. The peripheral cortex is often included in the abnormal segment, helping to differentiate from an acute renal infarct which typically spares the subcapsular cortex.[32,33]

Striated Nephrogram

On CT, a striated nephrogram is seen as tubular and alternating degrees of attenuation in the corticomedullary region and has several unique etiologies (Table 4). The striations are the result of contrast material stasis, with accumulation in necrotic or edematous tubules. They can present unilaterally or bilaterally.[4]

Acute pyelonephritis is the most common cause of a striated nephrogram, in addition to being a common cause of a delayed nephrogram (see above section).[4] Tubular stasis via pus accumulation and interstitial edema is the histologic analogue of the striated nephrogram (Fig 8). CT urography shows initial regional hypoenhancement with nephrographic reversal on delayed imaging, due to hyperconcentrated urine accumulating in the inflamed collecting ducts.[34,35] Although more speculative, a striated nephrogram due to renal contusion follows a similar postulated mechanism—tubular stasis secondary to interstitial edema and inflammation leads to urine hyperconcentration (Fig 9).[35] Renal vein thrombosis and unilateral ureteral obstruction can also lead to increased intravascular and intratubular pressure, resulting in the subsequent increased interstitial edema, increased parenchymal pressure, and urine hyperconcentration.[36]

The causes for a bilateral striated nephrogram include acute pyelonephritis, tubular obstruction (proteinuria, myoglobinuria), hypotension (Fig 10), and autosomal recessive polycystic kidney disease. In common, they share the mechanism of generalized urine hyperconcentration.[4] Of note, CT urography in patients with autosomal recessive polycystic kidney disease can show bilateral renal enlargement with smooth contours. Significant parenchymal striations result from the collection of contrast material in large collecting

TABLE 4
Causes for unilateral vs bilateral striated nephrogram

Unilateral	Bilateral
Acute pyelonephritis	Acute pyelonephritis
Ureteric obstruction	Tubular obstruction (eg, proteinuria, myoglobinuria)
Contusion	Hypotension
Renal vein thrombosis	Autosomal recessive polycystic kidney disease

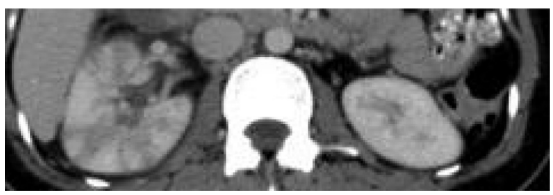


FIG 8. 23-year-old female with acute flank pain. An axial CT image demonstrates a striated nephrogram in the right kidney with radially oriented linear areas of reduced enhancement involving both cortex and medulla. The left kidney shows a normal nephrographic phase nephrogram. The diagnosis was acute pyelonephritis.

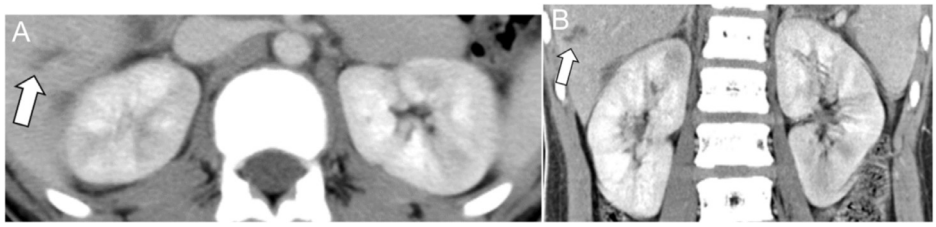


FIG 9. 15-year-old male status post motor vehicle accident. (A) Axial and (B) coronal images of segmental areas of delayed medullary enhancement in both kidneys give the appearance of a patchy striated nephrogram. In the acute traumatic setting this most likely represents areas of contusion. A portion of a liver laceration is also seen (arrows).

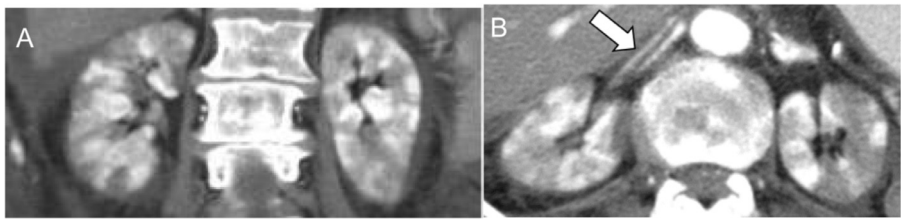


FIG 10. 69-year-old male with systolic hypotension. (A) Coronal CT view shows striated nephrograms in both kidneys. (B) Coronal image demonstrates that the IVC (arrow, adjacent to the right renal artery) is markedly flattened, consistent with severe hypotension. Perfusion abnormalities were also seen in the liver and spleen (not shown).

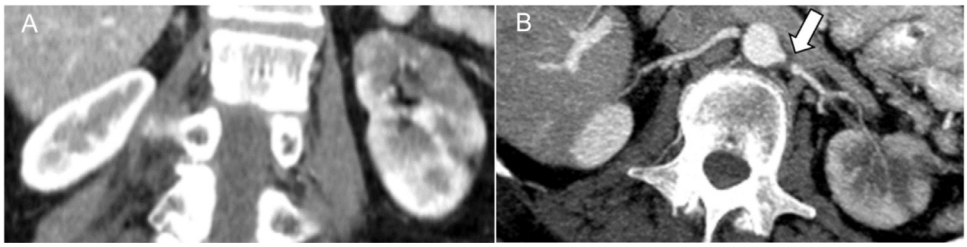


FIG 11. 82-year-old female with abdominal pain due to renal artery embolic disease. Spotted nephrogram in the left kidney, best appreciated on (A) the coronal CT image. (B) An axial MIP image from the same study shows a small filling defect within an accessory renal artery supplying the left upper pole (arrow).

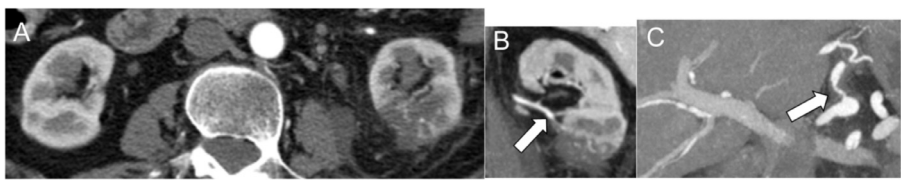


FIG 12. 66-year-old with abdominal pain due to renal ischemia. (A) An axial arterial phase CT images shows a wedge-shaped area of decreased perfusion in the lower pole of the left kidney. A normal corticomedullary nephrogram is seen in the right kidney. On (B) a coronal MIP image of the left kidney, there is mural thickening with abrupt narrowing of a left lower renal artery branch. (C) Similar segments of mural thickening and luminal narrowing seen in the left gastric artery. The patient was found to have polyarteritis nodosa.

duct cysts.[37]

Spotted Nephrogram

A spotted nephrogram refers to the patchy and irregular enhancement of the renal parenchyma because of small vessel occlusion and subsequent cortical ischemia and/or necrosis.[38] Segmental abnormality in perfusion or nephron function slows or prevents parenchymal contrast enhancement. Example etiologies include embolic disease (Fig 11) and intrarenal vasculitis, such as polyarteritis nodosa (PAN) (Fig 12). An uncommon cause of spotted nephrogram, embolic disease typically originates from cardiac sources (ie, valvular disease or left ventricular dyskinesia) or unstable atherosclerotic plaques in the aorta.[7] Pyelonephritis alters normal nephron function, as described

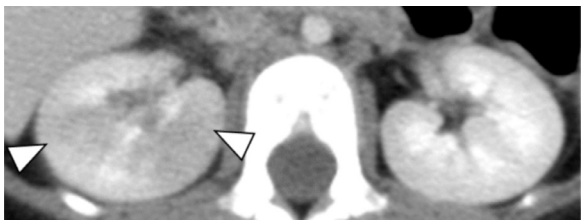


FIG 13. Spotted nephrogram appearance (arrowheads) in a patient with right-sided pyelonephritis.

TABLE 5
Causes of persistent nephrogram

Pathophysiology	Etiologies
No vascular inflow	Systemic hypotension Renal artery stenosis (atherosclerosis, focal obstructive masses)
No nephron outflow	Acute tubular necrosis (drug-induced, renal hypoxia) Mechanical obstruction (urate crystal nephropathy due to tumor lysis syndrome, Bence-Jones proteinuria, amyloid deposition)
No urine output	Stones

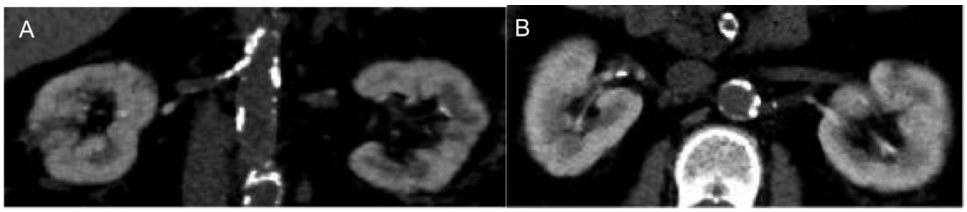


FIG 14. 54-year-old male with rising creatinine. (A) Coronal and (B) axial CT images show that even though no contrast is in the IVC or aorta, a corticomedullary nephrogram is present in both kidneys along with excretion of contrast. These findings represent retained contrast from a prior contrast enhanced study. This patient has acute tubular necrosis presumed secondary to contrast-induced nephropathy.

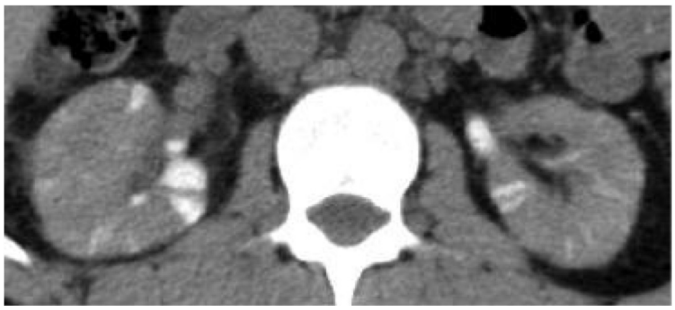


FIG 15. 35-year-old with acute renal failure and suspicious lucent bone lesions. Striated nephrograms are seen in both kidneys on this noncontrast study. The hyperdense areas represent hyperconcentrated retained contrast from a PE protocol chest CT performed earlier that day. The patient has tubular obstruction from multiple myeloma. The striated appearance of the delayed nephrogram is related to areas of tubular obstruction from Bence Jones proteins or amyloid deposits.

above, and severe cases can lead to enlarged kidneys with multiple patchy areas cortical enhancement (Fig 13).[39]

In cases of PAN, small- to medium-sized vessels become inflamed, resulting in intimal proliferation, luminal narrowing and eventual ischemia of the affected tissue. The ischemic renal parenchyma subsequently undergoes necrosis which presents as a spotted presentation on nephrogram. Furthermore, disruption of the medium-sized arteries can result in the inappropriate activation of the RAA system and secondary hypertension.[40] In patients with PAN, the renal arteries are the most common vessels involved and renal failure is a major cause of death.[41]

Persistent Nephrogram

The persistent nephrogram is defined by the retention of contrast in the cortex and/or collecting tubules for greater than 3 minutes, which is the typical duration for the pyelographic phase.[4] Delayed

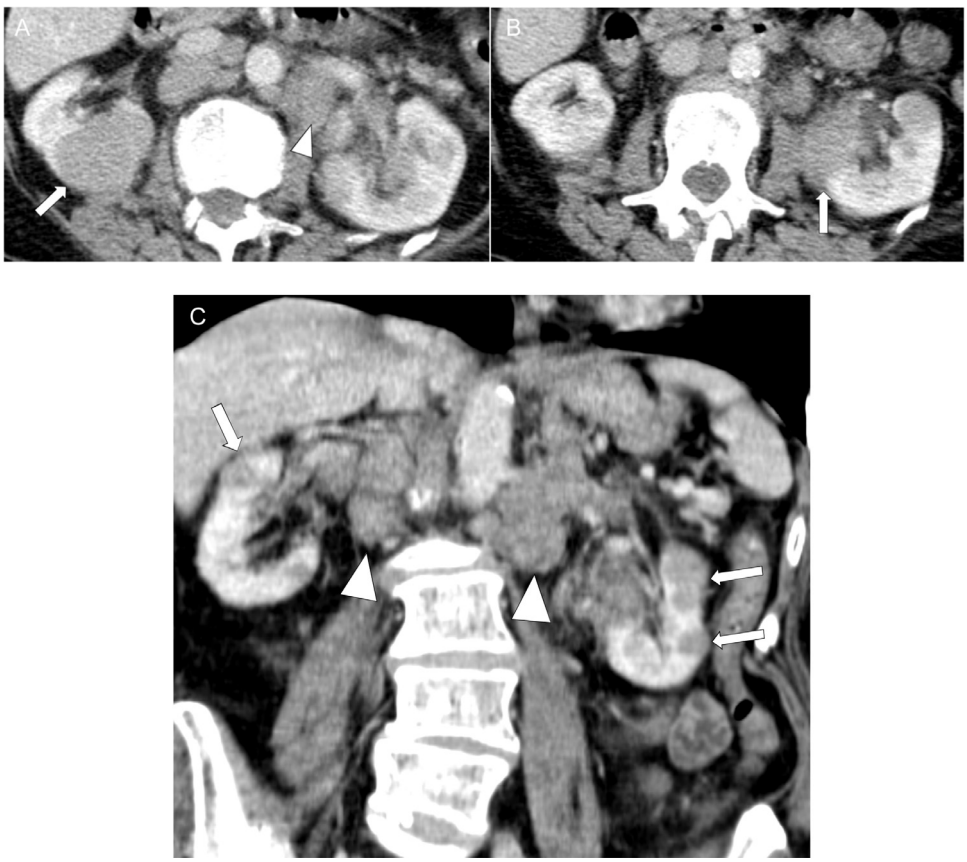


FIG 16. Segmental absent nephrogram in a patient with renal involvement of lymphoma. Multiple bilateral cortical regions of decreased enhancement (arrows) on (A) and (B) these 2 axial CT images at different levels and (C) coronal view represent intrarenal lymphomatous masses. Note extensive retroperitoneal lymphadenopathy (arrowheads).

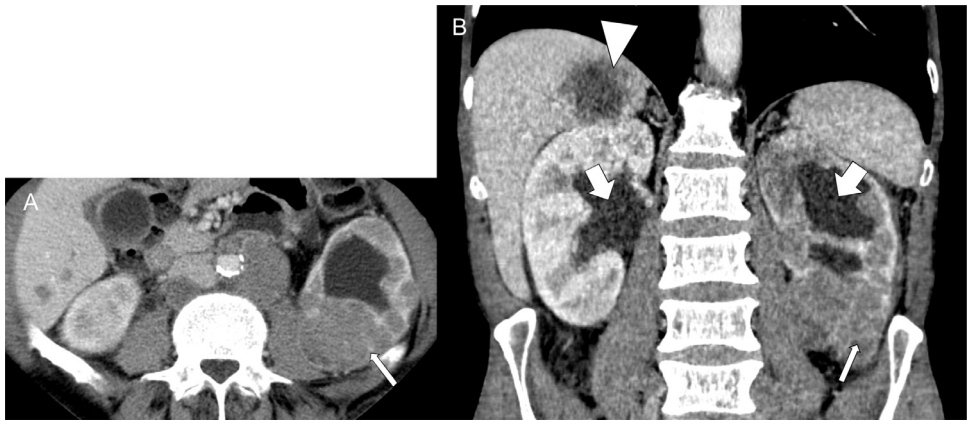


FIG 17. Segmental absent nephrogram in a patient with metastatic colon cancer. (A) An axial and (B) coronal CT image demonstrate multiple bilateral cortical regions of decreased enhancement (arrows) that represent intrarenal metastatic masses. Ureteral compression by distal pelvic metastatic disease (not shown) results in bilateral hydronephrosis (thick arrow). Note the extensive retroperitoneal lymphadenopathy and hepatic metastatic lesion (arrowheads).

excretion leads to the contrast agent remaining in the cortex and associated collecting system. Acute persistence of 3-10 minutes suggests systemic hypotension, while delay greater than 24 hours suggests complete tubular obstruction, as in the case of acute tubular necrosis (Table 5).[6]

Acute systemic hypotension is an emergent cause of persistent nephrogram. It typically coincides with classic clinical signs and symptoms, such as flattened IVC or aorta, hypoperfusion of the liver and spleen, and shock bowel.[42] When systolic blood pressure acutely decreases, the arterial pressure gradient across the glomerulus consequently decreases, leading to activation of the RAA pathway, salt and water reabsorption, and tubular stasis.[43] A common demonstration of acute hypotension on CT is a persistent nephrogram in the setting of adverse reaction to intravenous administration of iodinated contrast material (Fig 14).[4] The shunting of blood from the cortex to the medulla during acute hypotensive events can perpetuate the pyelographic phase for several hours following the acute window, generally improving with resolution of hypotension.[44]

Global persistent nephrograms lasting more than 1 day are typically due to tubular obstruction either from acute tubular necrosis or mechanical obstruction. Most commonly, acute tubular necrosis is the result of nephrotoxic agents such as antibiotics or iodinated contrast material, or renal hypoxia.[7] The persistence of cortical enhancement on CT lasting more than 24 hours postexposure accurately predicts the development of nephrotoxic exposure[45]. One cause of mechanical obstruction is urate crystal nephropathy secondary to the electrolyte abnormalities in tumor lysis syndrome. As tumor cells rupture during chemotherapy, intracellular contents are released into the serum, resulting in hyperphosphatemia, hypocalcemia, hyperkalemia, and hyperuricemia. Such electrolyte imbalances injure the collecting system and can lead to renal insufficiency. Similarly, multiple myeloma can cause Bence Jones proteinuria, amyloid deposition, and acute renal injury, manifesting as a persistent nephrogram on CT (Fig 15).

Segmentally persistent nephrograms are less common and can be seen in association with renal calculi in duplicated collecting ducts (Fig 18) and focal obstructions such as renal artery stenosis (Fig 19). It is the least commonly observed nephrogram overall and is typically due to transient increases in parenchymal pressure resulting in tubular stasis and a segmentally persistent image on CT[4]. For renal artery stenosis, segmentally persistent nephrograms are the result of relative hypotension that occurs in the renal tissue downstream from the

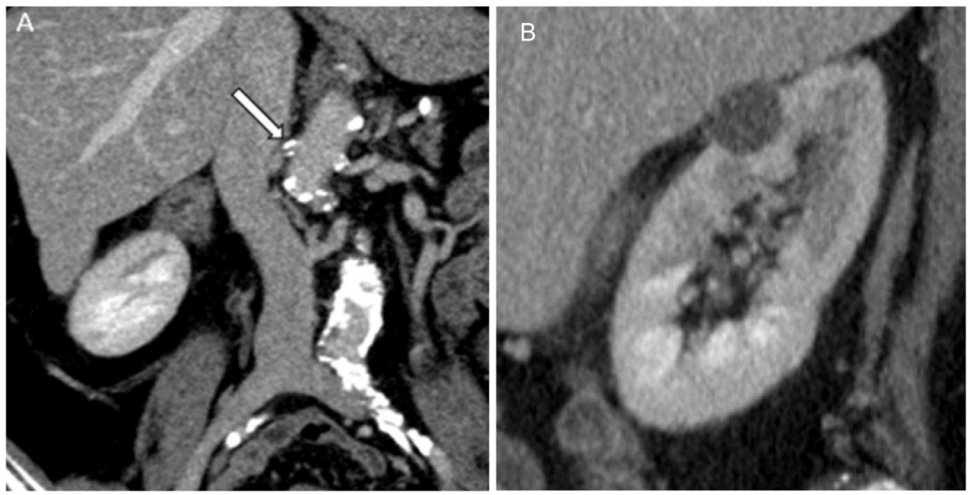


FIG 18. Segmental delayed nephrogram in a patient with renal artery stenosis (A) A coronal CT image of the abdomen shows 2 right renal arteries where the more superior renal artery (arrow) shows calcified and noncalcified plaque at the ostium resulting in renal artery stenosis. (B) A sagittal image demonstrates segmental delayed nephrogram of the upper kidney from impaired perfusion from the superior renal artery.

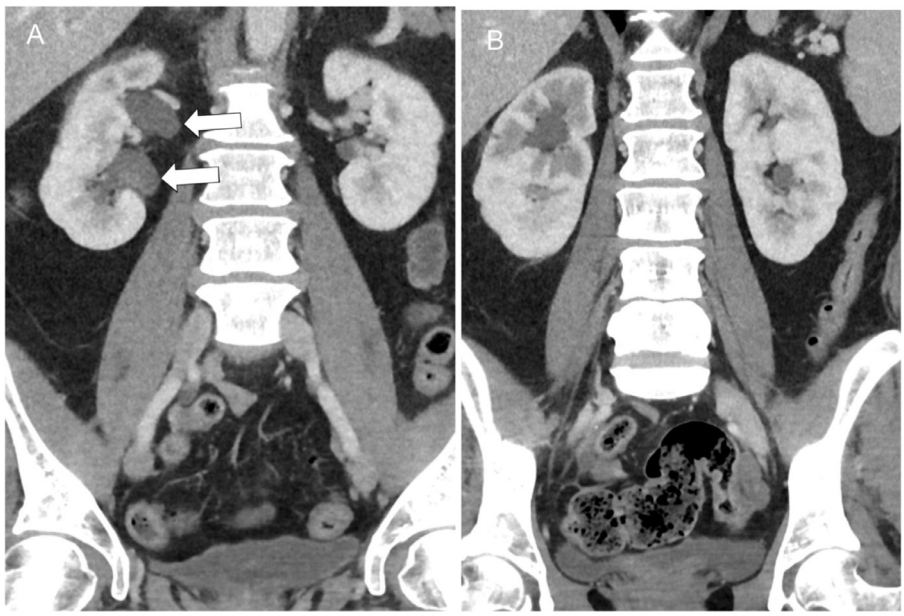


FIG 19. Ureteral stone causing right segmental delayed nephrogram. (A) A coronal CT shows a duplicated right renal collecting system (arrows). A distal ureteral stone in the upper pole moiety (not pictured) results in hydronephrosis and delayed nephrogram of the associated upper pole moiety seen on (B) this coronal view at a different level.

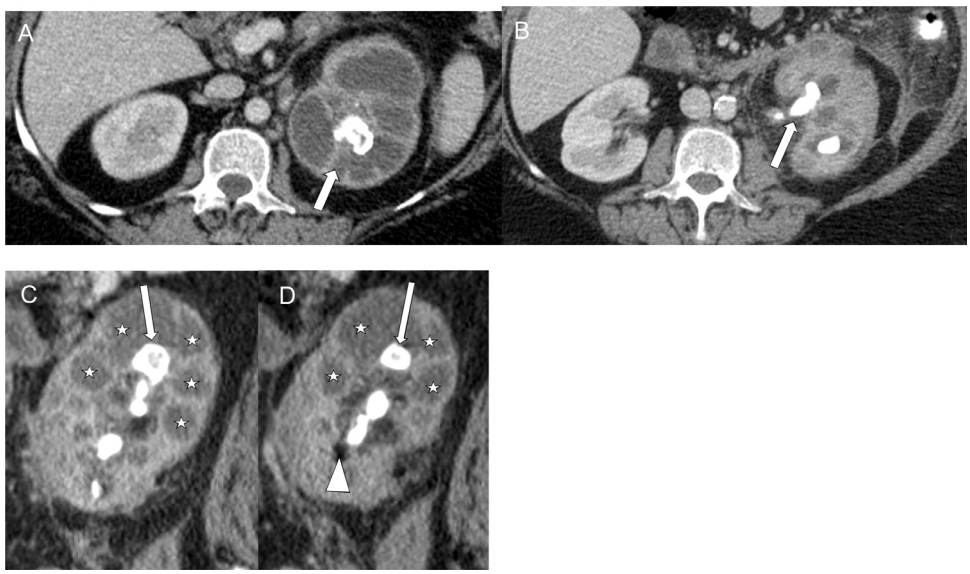


FIG 20. A patient presenting with flank pain and history of chronic renal infection. Findings in these (A and B) axial and (C and D) coronal CT images include left, unilateral delay nephrogram, staghorn calculi in the collecting system (arrows), dilation of the renal calyces in a multilocated pattern ("bear's paw sign") (asterisks) and foci of fat density representing the lipid-laded macrophages (arrowhead). Nephrectomy histology revealed xanthogranulomatous pyelonephritis.

stenotic lesion.[46] Other causes of focal obstructive lesions include enlarged lymph nodes, malignant growths, and parenchymal edema.

Conclusion

Contrast-enhanced CT is a crucial tool in the diagnosis of renal disease, used increasingly in the acute care setting. Its advantages are speed, accessibility, high-spatial resolution and few contraindications compared to other modalities. The nephrogram refers to patterns of renal enhancement as intravenous contrast courses through the renal parenchyma and collecting system, and when abnormal, can suggest specific diagnoses or disease etiologies. In this review, we present several cases illustrating abnormal nephrogram patterns, including the absent, unilateral delayed, striated, spotted, and persistent types. These can be used to better understand the differential diagnoses of acute renal pathology.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1067/j.cpradiol.2018.10.004.

References

1. Hyams ES, Korley FK, Pham JC, et al. Trends in imaging use during the emergency department evaluation of flank pain. J Urol 2011;186:2270–4 https://doi.org/10.1016/j.juro.2011.07.079.
2. Yuh BI, Cohan RH. Different phases of renal enhancement: Role in detecting and characterizing renal masses during helical CT. Am J Roentgenol 1999;173:747–55 https://doi.org/10.2214/ajr.173.3.10470916.
3. Wein A, et al. Campbell-Walsh Urology. 10th ed Philadelphia, PA: Elsevier/Saunders; 2012.
4. Saunders HS, Dyer RB, Shifrin RY, et al. The CT nephrogram: Implications for evaluation of urinary tract disease. Radiographics 1995;15:1069–85. discussion 1086-1088 https://doi.org/10.1148/radiographics.15.5.7501851.
5. Becker J, Babb J, Serrano M. Glomerular filtration rate in evaluation of the effect of iodinated contrast media on renal function. Am J Roentgenol 2013;200:822–6 https://doi.org/10.2214/AJR.12.8871.
6. Wolin EA, Hartman DS, Olson JR. Nephrographic and pyelographic analysis of CT Urography: Principles, patterns, and pathophysiology. Am J Roentgenol 2013;200:1210–4.
7. Wolin EA, Hartman DS, Olson JR. Nephrographic and pyelographic analysis of CT urography: Differential diagnosis. Am J Roentgenol 2013;200:1197–203.
8. Wong WS, Moss AA, Federle MP, et al. Renal infarction: CT diagnosis and correlation between CT findings and etiologies. Radiology 1984;150:201–5 https://doi.org/10.1148/radiology.150.1.6689761.
9. Bae EJ, Hwang K, Jang HN, et al. A retrospective study of short- and long-term effects on renal function after acute renal infarction. Ren Fail 2014;36:1385–9 https://doi.org/10.3109/0886022X.2014.947514.
10. Antopolsky M, Simanovsky N, Stalnikowicz R, et al. Renal infarction in the ED: 10-year experience and review of the literature. Am J Emerg Med 2012;30:1055–60. pp.
11. Steinberg DL, Jeffrey RB, Federle MP, et al. The computerized tomography appearance of renal pedicle injury. J Urol 1984;132:1163–4.
12. Harris AC, Zwirewich CV, Lyburn ID, et al. CT findings in blunt renal trauma. Radiographics 2001;21:S201–14.
13. Oh YK, Yang CW, Kim Y-L, et al. Clinical characteristics and outcomes of renal infarction. *Am. J. Kidney Dis* 2016 2016;67:243–50 https://doi.org/10.1053/j.ajkd.2015.09.019.
14. Morgan RJ, Fenely RC. Renal vein thrombosis caused by primary antiphospholipid syndrome. Br J Urol 1994;74:807–8.
15. Kawashima A, Sandler CM, Ernst RD, et al. CT evaluation of renovascular disease. Radiographics 2000;20:1321–40.
16. Craig WD, Wagner BJ, Travis MD. Pyelonephritis: Radiologic-pathologic review. Radiographics 2008;28:255–76.
17. Fan CM, Whitman GJ, Chew FS. Xanthogranulomatous pyelonephritis. Am J Roentgenol 1995;165:1008.
18. Dyer RB, Chen MY, Zagoria RJ. Classic signs in uroradiology. Radiographics 2004;24:S247–80 https://doi.org/10.1148/rg.24si045509.
19. Eastwood JB, Corbishley CM, Grange JM. Tuberculosis and the kidney. J Am Soc Nephrol 2001;12:1307–14.
20. De Francesco Daher E, Bezerra da Silva Junior G, Barros EJG. Renal tuberculosis in the modern era. Am J Trop Med Hyg 2013;88:54–64 https://doi.org/10.4269/ajtmh.2013.12-0413.
21. Muttarak M, ChiangMai WN, Lojanapiwat B. Tuberculosis of the genitourinary tract: Imaging features with pathological correlation. Singapore Med J 2005;46:568–74. quiz 575.
22. Jung YY, Kim JK, Cho K-S. Genitourinary tuberculosis: Comprehensive cross-sectional imaging. Am J Roentgenol 2005;184:143–50 https://doi.org/10.2214/ajr.184.1.01840143.
23. Katabathina VS, Kota G, Dasyam AK, Shanbhogue AK, Prasad SR. Adult renal cystic disease: A genetic, biological, and developmental primer. Radiographics 2010;30:1509–23.
24. Wood CG, Stromberg LJ, Harmath CB, et al. CT and MR imaging for evaluation of cystic renal lesions and diseases. Radiographics 2015;35:125–41 https://doi.org/10.1148/rg.351130016.
25. Hains DS, Bates CM, Ingraham S, Schwaderer AL. Management and etiology of the unilateral multicystic dysplastic kidney: A review. Pediatr Nephrol 2009;24:233–41.
26. Colyer WR, Eltahawy E, Cooper CJ. Renal artery stenosis: Optimizing diagnosis and treatment. Prog Cardiovasc Dis 2011;54:29–35 https://doi.org/10.1016/j.pcad.2011.02.007.
27. Birnbaum BA, Bosniak MA, Megibow AJ. Asymmetry of the renal nephrograms on CT: Significance of the unilateral prolonged cortical nephrogram. Urol Radiol 1991;12:173–7.
28. Takebayashi S, Ueki T, Ikeda N, Fujikawa A. Diagnosis of the nutcracker syndrome with color Doppler sonography: Correlation with flow patterns on retrograde left renal venography. Am J Roentgenol 1999;172:39–43 https://doi.org/10.2214/ajr.172.1.9888735.
29. Muraoka N, Sakai T, Kimura H, et al. Rare causes of hematuria associated with various vascular diseases involving the upper urinary tract. Radiographics 2008;8:855–67 https://doi.org/10.1148/rg.283075106.
30. Foxman B, Klemstine KL, Brown PD. Acute pyelonephritis in US hospitals in 1997: Hospitalization and in-hospital mortality. Ann Epidemiol 2003;13:144–50.
31. Jennette JC. Heptinstall's Pathology of the Kidney. 7th ed Philadelphia: Wolters Kluwer; 2015.
32. Johnson PT, Horton KM, Fishman EK. Optimizing detectability of renal pathology with MDCT: Protocols, pearls, and pitfalls. Am J Roentgenol 2010;94:1001–12 https://doi.org/10.2214/AJR.09.3049.
33. Zagoria RJ. Genitourinary radiology: The requisites. In: Louis: St, ed. 6th ed, Mosby; 1997.
34. Berliner L, Bosniak MA. The striated nephrogram in acute pyelonephritis. Urol Radiol 1982;4:41–4.
35. Rubin BE, Schliftman R. The striated nephrogram in renal contusion. Urol Radiol 1979;1:12–9. 1.
36. Wicks JD, Bigongiari LR, Foley WD, Walter J. Parenchymal striations in renal vein thrombosis: Arteriographic demonstration. Am J Roentgenol 1977;129:95–8.
37. Rabinowitz R, Segal AJ, Rao HKM, et al. Computed tomography in diagnosis of infantile polycystic kidney disease. J Urol 1978;120:616–7.
38. Winograd J, Schimmel DH, Palubinskas AJ. The spotted nephrogram of renal scleroderma. Am J Roentgenol 1976;126:734–8.
39. Dunnick NR, Sandler CM, Newhouse JH, et al. Textbook of Uroradiology. p 166, 4th ed Philadelphia, PA: Lippincott Williams & Wilkins; 2008. p. 166–8.
40. Stockigt JR, Topliss DJ, Hewett MJ. High-renin hypertension in necrotizing vasculitis. N Engl J Med 1979;300:1218 https://doi.org/10.1056/NEJM197905243002115.
41. Rhodes ES, Pekala JS, Gemery JM, et al. Case 129: Polyarteritis nodosa. Radiology 2008;246:322–6.
42. Ames JT, Federle MP. CT hypotension complex (shock bowel) is not always due to traumatic hypovolemic shock. Am J Roentgenol 2009;192:W230–5. [web].
43. Newhouse JH, Pfister RC. The nephrogram. *Radiol Clin N Am* 1979;17:213–26.
44. Kupic EA, Abrams HL. Renal vascular alterations induced by hemorrhagic hypotension. Invest Radial 1968;3:345–55.
45. Love L, Olson MC. Persistent CT nephrogram: Significance in the diagnosis of contrast nephropathology—An update. Urol Radiol 1991;12:206–8.
46. Dyer R, Miller S, Anderson BL, et al. The segmental nephrogram. Am J Roentgenol 1985;145:321–2 https://doi.org/10.2214/ajr.145.2.321.