CT Nephrogram - an overlooked sign that may help you reach the diagnosis

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Learning objectives

1. Review normal nephrographic physiology and pattern

2. Present and illustrate abnormal CT nephrographic patterns with the respective differential diagnosis

3. Correlate CT nephrogram with other imaging findings and clinical features, to reach the correct diagnosis.

Background

The urographic nephrogram (NG) is an important indicator of underlying functional and structural renal disease. CT nephrogram has an important role in the evaluation of urinary track disorders and is useful demonstrating both quantitative and qualitative nephrographic abnormalities, showing different patterns of enhancement. There are six major patterns of abnormal NG: absent NG, unilateral delayed pyelogram, bilateral persistent NG, rim NG, reverse rim NG and striated NG.

Imaging findings OR Procedure details

The nephrogram is defined as the radiographic image of opacified renal parenchyma after the administration of contrast material. Through extensive research and observation, the details surrounding the physiology and appearance of the normal kidneys in nephrograms, as well as nephrographic patterns and alterations associated with specific diseases have been elucidated. In this work we will describe the appearances of the normal nephrographic phases, the nephrographic patterns of specific entities and their differential diagnosis.

THE NORMAL CT NEPHROGRAM

Single-detector and multidetector spiral CT have dramatically refined the diagnostic evaluation of renal pathologic conditions by allowing rapid image acquisition through the entire kidney during various phases of contrast enhancement after the administration of a single bolus of intravenous contrast material. It is important to know the different phases of
a normal CT nephrogram and understand their value. Kidney CT study uses the following phases: unenhanced, corticomedullary, nephrographic and excretory (fig.1).

**Unenhanced CT**

Provides a baseline from which to measure the enhancement within the lesion after the administration of intravenous contrast material. This enhancement characteristic is important in distinguishing hyperdense cysts from solid tumors, because most tumors will enhance while cysts won't. Another reason is that urolithiasis or calcifications are best seen on unenhanced CT.

**Corticomedullary Phase**

The corticomedullary phase is the first and shortest phase of contrast enhancement. It occurs between 25 and 70 seconds after the initiation of contrast material injection. In this phase the contrast material is primarily within the cortical capillaries, peritubular spaces, and cortical tubular lamina and has not yet filtered through the more distal renal tubules. The renal cortex enhances brightly and is easily differentiated from the minimally enhancing renal medulla. Maximal opacification of the renal arteries and veins occurs allowing accurate delineation of the arterial anatomy.

**Nephrographic Phase**

As contrast material filters through the glomeruli into the loops of Henle and the collecting tubules, the nephrographic phase of contrast enhancement begins. This phase is best imaged after a scanning delay of at least 80 seconds and lasts up to 180 seconds after the start of contrast material injection. The renal parenchyma enhances homogeneously, allowing the best opportunity for discrimination between the normal renal medulla and masses. The nephrographic phase is the most valuable for detecting renal masses and characterizing indeterminate lesions.

**Excretory Phase**

The excretory phase begins approximately 180 seconds after the initiation of injection of iodinated contrast material. The contrast material is excreted into the collecting system, and as a result, the attenuation of the nephrogram progressively decreases. This phase is occasionally helpful to better delineate the relationship of a centrally located mass with the collecting system and define potential involvement of the calices and renal pelvis and helps evaluation of ureteral and bladder lesions.
Production of the normal CT nephrogram, with appropriate temporal progression through the phases defined above, requires a complex interaction of renal perfusion, normal glomerular filtration, tubular function and absence of urinary tract obstruction. Alterations of these processes may produce both quantitative and qualitative abnormalities in the perceived CT image. CT nephrograms will provides the radiologist with a powerful tool for assessing underlying renal disease.

NEPHROGRAPHIC PATTERNS OF ABNORMALITY

1. Absent Nephrogram
a. Global Absence of the Nephrogram

The most basic quantitative alteration of the CT nephrographic pattern is global absence (fig2 and fig3). It is defined as lack of contrast material enhancement in the kidney. Pathophysiologically, global absence is most easily equated with complete renal ischemia secondary to interruption of primary renal perfusion, most commonly due to total occlusion of the main renal artery.

Causes for this nephrographic pattern include:

- Trauma: with vascular pedicle injury
- Thromboembolic disease
- Renal artery dissection (spontaneous, traumatic, or iatrogenic).

This pattern is nearly always unilateral and is most often seen with blunt abdominal trauma with renal pedicle injury.

Usual CT appearance: kidney of normal size and contour, with no enhancement of the parenchyma after contrast material administration. Additional imaging features may include perirenal hemorrhage and lack of excretion of opacified urine into the ipsilateral collecting system and ureter.

Recognizing this pattern is extremely important because emergent surgical intervention is needed to correct vascular interruption and prevent irreversible hypoxic damage.
Fig. 2: 2. Axial enhanced CT images in the corticomedullary phase show unilateral global absence nephrogram of the right kidney (*) in a patient with traumatic kidney pedicle injury. On the left kidney we can see a striated pattern (arrow) due to contusion. Also notice hepatic lacerations (arrowhead).

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 3: 3. Axial enhanced CT images in the corticomedullary phase (a) and in the excretory phase (b) show unilateral global absence nephrogram in the same patient shown in fig.2 a few months later.

References: A. F. S. Simões; Porto, PORTUGAL

b. Segmental Absence of the Nephrogram

Focal defects in the CT nephrogram (fig 4 - 16) can be secondary to:

- Renal space-occupying processes: they displace or replace renal parenchyma such as neoplasms, cysts and abscesses.

- Local hemodynamic changes: major arterial interruption is responsible for global renal ischemia while local hemodynamic changes in more peripheral branches of the renal artery lead to segmental defects. Contrast material is prevented from reaching tissue beyond the site of vascular occlusion. These areas of underperfusion are depicted on contrast-enhanced CT examinations as focal or multifocal (patchy), peripheral, wedge-
shaped areas of decreased attenuation against a background of normally enhancing parenchyma. The relative size of the nephrographic defect correlates directly with the size of the obstructed feeding vessel and reflects the volume of renal parenchyma supplied.

- Focal renal infarction: segmental infarcts often appear as sharply demarcated, wedge-shaped areas of absent enhancement according to the distribution of the occluded vessel. They can have various causes, including arterial emboli, arterial thrombosis, renal vein thrombosis, vasculitis and septic shock. Embolic phenomena are the leading cause of multifocal segmental renal infarcts and can arise from a cardiac source or diseased peripheral vessels. Clinical signs and symptoms of acute renal infarction are often nonspecific, misleading, or absent. Many physical findings, such as flank pain, flank tenderness, hematuria, fever and leukocytosis, overlap significantly with the clinical characteristics of other renal processes such as nephrolithiasis, pyelonephritis, renal vein thrombosis, or even neoplasm.

Minimal nephrographic changes associated with small-vessel occlusive diseases such as vasculitis, collagen-vascular disease, and sickle cell anemia may be detected with contrast-enhanced CT.
Fig. 4: 4. Unenhanced (a) and enhanced (b, c) axial CT images show a focal absence of nephrogram (arrow). At first sight this wedge shape looks like a kidney infarction, but looking to image (a) we can see it has fat density. This is a focal absence secondary to the presence of an angiomyolipoma.

References: A. F. S. Simões; Porto, PORTUGAL

Fig. 5: 5. Unenhanced (a) and enhanced (b, c) CT images show a focal absence of nephrogram (arrow). Looking to image (a) we can see it has high density and it has no contrast enhancement in images (b) and (c). This is a focal absence secondary to the presence of high-density cyst. Note also caliectasis (*)secondary to bilateral junction syndrome.

References: A. F. S. Simões; Porto, PORTUGAL
**Fig. 6:** Enhanced CT images (a) and (b) show a focal nephrogram alteration secondary to the presence of a renal cell carcinoma (arrow).

**References:** A. F. S. Simões; Porto, PORTUGAL
**Fig. 7**: 7. Enhanced CT images (a) and (b) show a focal absence of nephrogram (arrow) due to a hemodynamic change secondary to the presence of an iatrogenic intrarenal pseudo-aneurysm (arrowhead). Note this patient has an axillary-femoral bypass graft (*) due to occlusion of the abdominal aorta.

**References**: A. F. S. Simões; Porto, PORTUGAL
Fig. 8: Unenhanced (a) and enhanced (b) CT images shows a focal absence of nephrogram (arrow) on (b) due to a hemodynamic change secondary to the presence of a traumatic intrarenal pseudo-aneurysm (arrowhead). Notice on (a) high-density area corresponding to recent bleeding leading, to perirenal fluid accumulation.

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 9: 9. Enhanced CT images (a) and (b) show a large segmental absence of nephrogram (*) on the right kidney secondary to traumatic renal artery injury (arrowhead). The left kidney, not visible in images (a) and (b) has an ectopic position in the pelvis (c) and shows a small segmental absence nephrogram (arrow) also secondary to traumatic vascular changes.

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 10: Enhanced CT images (a) and (b) show of a focal absence of nephrogram (arrow), at two different levels, secondary to traumatic kidney laceration. Also notice perirenal fluid.

References: A. F. S. Simões; Porto, PORTUGAL
**Fig. 11:** Unenhanced (a) and enhanced (b, c) CT images show of a focal absence of nephrogram (arrow) secondary to traumatic kidney rupture. Perirenal collection is also present (*).

**References:** A. F. S. Simões; Porto, PORTUGAL
Fig. 12: Enhanced CT images (a), (b), (c), and (d) show different phases and locations of a multiple segmental absence of nephrogram, bilaterally, secondary to underperfusion, in a patient with septic shock.

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 13: 13. Enhanced CT images (a), (b) and (c), show unilateral segmental absence nephrogram due to ischemic alterations in a patient submitted to ureteropyeloplasty. Image (d) shows lack of perfusion on the left kidney on the area corresponding to the absence of nephrogram. Note the presence of a kidney catheter on the left, fluid in the perirenal and anterior and posterior left pararenal spaces and ascites.

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 14. Enhanced CT images (a), (b), (c), and (d) show multiple segmental absence of nephrogram bilaterally due to arterial embolization (arrowheads) in a patient with endocarditis.

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 15: Enhanced CT images show an area of focal absence of nephrogram in the left kidney secondary to kidney infection (arrow). Notice perirenal fat stranding surrounding the infection site.

References: A. F. S. Simões; Porto, PORTUGAL
**Fig. 16**: Enhanced CT images (a), (b), (c) and (d) show two areas of focal absence of nephrogram in the left kidney secondary to kidney infection (arrows). Note adjacent to the most anterior lesion the presence of air and fat stranding. Fluid collection (*) in the anterior pararenal space is also present.

**References**: A. F. S. Simões; Porto, PORTUGAL

2. **Rim Nephrogram**

The rim nephrogram (fig 17 and fig 18) described at urography is a thin (2-4-mm) peripheral band of cortical opacification seen during the progression of the nephrographic phases. The rim pattern is believed to be the most specific indicator of renovascular compromise. It represents preserved subcapsular perfusion via collateral flow through capsular, peripelvic and periureteric vessels. This capsular collateral network is variably derived from adjacent retroperitoneal vessels such as the lumbar, intercostal, adrenal, phrenic, and iliac arteries. The increase in caliber of both renal capsular and extracapsular vessels is secondary to the presence of chronic ischemia. These observations imply that the amount of preserved cortical perfusion or thickness of the rim depend on preexisting renal ischemia to increase the extent of collateral blood supply.
Presence of either focal or global rim signs on CT images is associated with:

- Renal infarction.

- Renal vein thrombosis: increased interstitial pressure opposes perfusion pressure and results in impaired intrarenal arterial flow.

- Acute tubular necrosis: a similar mechanism as in renal vein thrombosis coupled with preglomerular vasoconstriction is thought to be the responsible for the rim pattern.

This pattern of subcapsular enhancement serves as a crucial point of differential diagnosis between infarction and pyelonephritis.

![Figure 17](image_url)

Fig. 17: Enhanced CT images (a), (b), (c) and (d) show a rim pattern nephrogram on the right kidney (arrow) and a focal absence of nephrogram (*) on the left. These alterations were secondary to bilateral infarction. The rim sign represents preserved subcapsular perfusion via collateral flow.

References: A. F. S. Simões; Porto, PORTUGAL
**Fig. 18**: Enhanced CT images (a) and (b) show a rim pattern nephrogram on the right kidney (arrow) in a patient with renal infarction secondary to traumatic injury.  

**References**: A. F. S. Simões; Porto, PORTUGAL

A "rim-like sign" (fig19) can be seen in chronic obstruction. It's not secondary to underperfusion as the rim pattern. It represents opacification of residual, functioning renal parenchyma surrounding a chronically dilated or obstructed collecting system. At CT, the rim of tissue may have variable thickness, is concave medially and surrounds the dilated calices in communication with the central pelvis.
Fig. 19: 19. Enhanced CT images (a) and (b) show a "rim-like" pattern on the right kidney (*). At first it can seem a "rim-sign" but looking carefully, we can see the image represents a kidney with chronic dilatation of excretory system with cortical thinning mimicking the rim sign. Note the slowed progression of nephrogram on the right secondary to this chronic obstruction.

References: A. F. S. Simões; Porto, PORTUGAL

3. Reverse Rim Nephrogram

The reverse rim nephrogram results from acute cortical necrosis and appears as a hypoattenuating zone circumscribing the kidneys adjacent to the renal capsule, with enhancement of the medulla. A rim of subcapsular cortical enhancement is also a characteristic finding because of collateral flow from the capsular vessels.

Acute cortical necrosis is a rare form of acute renal failure and results from ischemic necrosis of the renal cortex with sparing of the renal medulla. The pathophysiology of this condition is complex and has been attributed to ischemia due to vasospasm of small vessels, toxic damage to glomerular capillary endothelium and primary intravascular thrombosis. The process is either multifocal or diffuse; in most cases, it is bilateral. This
condition is associated with complications of pregnancy, including abruptio placentae and septic abortion. Other causes include sepsis, shock, venomous snake bite, severe dehydration, transfusion reaction and hemolytic uremic syndrome. Cortical necrosis can result from any condition that produces acute, prolonged shock.

4. Unilateral Delayed Pyelogram

Contrast material progressing from the renal arterial beds through glomeruli, nephrons, and finally the renal collecting systems should be evident in temporally synchronous and symmetric nephrographic phases at CT. Abnormalities in this evolutionary process are generally detected as unilateral alterations in the progression of the nephrographic sequence (fig20 - 25). Temporal delay may be attributed to a unilateral decrease in renal blood flow, with a corresponding decline in glomerular filtration.

This pattern is associated with:

- Renal artery stenosis: causes an asymmetric progression of the CT nephrogram, with prolongation of the cortical nephrographic phase and persistent corticomedullary differentiation on the involved side.

- Renal vein thrombosis: intravenous clot opposes the arterial perfusion gradient, slowing filtration and decreasing tubular transit. Additionally, interstitial edema will develop increasing the parenchymal pulp pressure, which leads to compression of tubular lumina. That will decrease the forward flow of contrast material originating a slowed temporal progression of the nephrogram. Characteristic imaging features of renal vein thrombosis, such as an enlarged renal vein containing a filling defect (thrombus), enlargement of the affected kidney caused by interstitial edema, and possibly retroperitoneal hemorrhage, should easily allow differentiation of this process from the characteristic small, smooth kidney of chronic renal ischemia.

- Obstructive uropathy: is the most common cause of asymmetric delay in the development of the nephrographic pattern. Obstruction to urine flow anywhere along the outflow tract increases internal pressure within the collecting system proximal to the blockage. Increased pressure in the static urine column is transmitted in a retrograde manner to the level of the nephron opposing the forward perfusion gradient of normal arterial pressure. Eventually, the rates of both glomerular filtration and tubular transit decrease. This delay is demonstrated at CT examinations by slowed progression of nephrographic phases in the obstructed kidney and persistence of the generalized nephrogram on delayed images.
When temporal abnormalities of the CT nephrogram are assessed, common causes of unilateral temporal delay, such as renal artery stenosis, renal vein thrombosis and urinary tract obstruction, should be considered. These entities, although obviously different in cause, relate to similar underlying pathophysiologic principles.

**Fig. 20**: 20. Enhanced CT images (a), (b), (c) show a slowed temporal progression of contrast enhancement on the left kidney associated with dilatation of the excretory system (*) secondary to chronic obstruction. Image (d) shows urothelial carcinoma of the bladder involving the left ureteral orifice (arrow) and causing the chronic obstruction.

**References**: A. F. S. Simões; Porto, PORTUGAL
**Fig. 21:** Enhanced CT images (a), (b), (c) show an asymmetrical progression of contrast enhancement on the left kidney secondary to aortic dissection (arrowhead) that reduces arterial flux. Note the presence of parapielic (*) cysts on the left kidney and cortical cysts (circle) on both kidneys.

**References:** A. F. S. Simões; Porto, PORTUGAL
Fig. 22: Enhanced CT images show a slowed temporal progression of contrast enhancement on the left kidney associated with renal vein malignant thrombosis (*). The renal cell carcinoma is seen on image (a) (arrow).

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 23: Enhanced CT images show a slowed temporal progression of contrast enhancement on the left kidney associated with dilatation of the excretory system (*) secondary to chronic obstruction, caused by an urothelial tumor of the ureter (not shown).

References: A. F. S. Simões; Porto, PORTUGAL
**Fig. 24:** Enhanced CT images show delayed pyelogram on the left kidney associated with dilatation of the excretory system (*) secondary to urolithiasis obstruction (not shown).

**References:** A. F. S. Simões; Porto, PORTUGAL
**Fig. 25:** Enhanced CT images show a slowed temporal progression of contrast enhancement on the right kidney in a patient with polycystic kidney disease, secondary to a pyelonephritis.

**References:** A. F. S. Simões; Porto, PORTUGAL

5. **Striated Nephrogram**

After contrast material administration, the striated CT nephrogram (fig2 and fig26-28) appears as linear bands of alternating high and low attenuation oriented parallel to the axis of the tubules and collecting ducts.

Unilateral causes of this pattern include:

- Ureteric obstruction: the striations are believed to represent areas of hyperconcentrated contrast material within clusters of dilated medullary rays outlined against a background of edematous renal parenchyma. This pattern is rare in acute extrarenal obstruction, thus
prior obstructive episodes are thought to be responsible for the preexisting dilatation of these medullary ducts.

- Acute pyelonephritis: the cellular inflammatory infiltrate selectively increases parenchymal pressure leading to increased interstitial edema within the affected medullary or cortical rays. These areas initially demonstrate decreased enhancement. With delayed imaging, the same areas may demonstrate attenuation that is increased relative to that of adjacent normal tissue. This nephrographic reversal is most likely caused by tubular stasis, in which contrast material-laden urine slowly enters and becomes hyperconcentrated in collecting ducts obstructed by inflammatory cells.

- Contusion: several possibilities were suggested as the cause of nephrographic striations including tubular stasis secondary to interstitial edema, focal areas of contrast material extravasation from collecting tubules and small vessels, and underperfusion resulting from underlying vascular spasm.

- Renal vein thrombosis: the cause of nephrographic striations associated with renal vein thrombosis is likewise speculative. One theory proposes that the striations represent unopacified tubular lumina outlined by stagnant, contrast material-laden blood in the peritubular capillary network. Given the association of renal vein thrombosis with increased parenchymal pressure, the concept of tubular stasis and urine hyperconcentration also seems appropriate in this setting.

Bilateral causes of this pattern include:

- Acute pyelonephritis

- Tubular obstruction (ie, Tamm-Horsfall proteinuria or rhabdomyolysis with myoglobinuria) can be explained by the mechanism of static urine within obstructed tubules and subsequent hyperconcentration.

- Hypotension: can be explained by the same mechanism as in tubular obstruction.

- Autosomal recessive polycystic kidney disease: the most radiologically striking example of a striated nephrogram is found in cases of autosomal recessive polycystic kidney disease. In this disorder, CT images depict smoothly contoured, enlarged kidneys bilaterally with extensive parenchymal striations relating to contrast material trapped within the characteristically large, dysplastic collecting tubules.
Fig. 26: Enhanced CT image shows a discrete bilateral striated pattern in a patient with kidney underperfusion secondary to severe hypotension.

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 27. Enhanced CT images show a striated pattern secondary to pyelonephritis in a renal allograft.

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 28: Enhanced CT images show a striated pattern secondary to pyelonephritis.

References: A. F. S. Simões; Porto, PORTUGAL

6. Persistent Nephrogram

Depending on the length of time between contrast material administration and imaging, asymmetries in development of the CT nephrogram can be perceived as zones of nephrographic absence, delay, or persistence. Unilateral nephrographic persistence at CT examinations corresponds directly to the obstructed side.

Common causes of unilateral persistence of the nephrogram include:

- Renal artery stenosis.
- Renal vein thrombosis.
- Urinary tract obstruction (previously discussed).
Causes of bilateral persistence of the nephrogram (fig 30) are not very common, and include:

- Systemic hypotension (shock): a life-threatening situation that can be responsible for a bilateral persistent high-attenuation CT nephrogram. As systolic pressure falls and the arterial pressure gradient driving glomerular filtration diminishes, the result is tubular stasis and increased resorption of salt and water at the tubular level via the renin-angiotensin-aldosterone pathway. The hypotensive nephrogram is most frequently demonstrated when CT is performed on a patient who is in shock or when a hypotensive contrast material reaction occurs during the CT examination.

- Intratubular obstruction: in normotensive patient, bilateral persistence of the CT nephrogram should prompt concern for intratubular obstruction as a result of protein (Tamm-Horsfall, Bence-Jones, or myoglobin) or crystal (urate) precipitation.

- Acute tubular necrosis: abnormalities in tubular function may also produce nephrographic persistence. Depending on the severity of tubular damage, the process of acute tubular necrosis may appear as high-attenuation CT nephrograms that persist for as long as several days after contrast material administration. Tubular damage can result from contrast material. The persistence of a nephrogram 24 hours after a contrast-enhanced study may be detected at CT and should alert the clinician to the fact that contrast material-induced nephropathy may be evolving. Acute renal failure induced by contrast media has been attributed to multiple factors, including tubular obstruction from precipitation of proteins or uric acid, vasoconstriction, sludging of erythrocytes related to contrast material osmolality, and possibly direct toxic effects on the renal tubular epithelium. Early recognition of persistent CT nephrograms on unenhanced CT images or abnormal persistence of CT nephrograms during the course of the CT study is important because this finding may precede the development of oliguria or a detectable rise in the level of serum creatinine, aiding the diagnosis of the patient's decline in renal function and prevent further administration of contrast material.
Fig. 29: Unenhanced CT image shows a bilateral persistent nephrogram 24h after the first CT. This patient developed a contrast material induced nephropathy. Note the absence of contrast in the aorta and the presence of contrast in the gallbladder indicating its biliary excretion.

References: A. F. S. Simões; Porto, PORTUGAL
Fig. 1: Normal CT nephrogram: Unenhanced (a) and contrast enhanced (b,c,d) axial CT images showing (b) normal corticomedullary phase (c) normal nephrographic phase and (d) normal excretory phase.
**Fig. 2:** Axial enhanced CT images in the corticomedullary phase show unilateral global absence nephrogram of the right kidney (*) in a patient with traumatic kidney pedicle injury. On the left kidney we can see a striated pattern (arrow) due to contusion. Also notice hepatic lacerations (arrowhead).
Fig. 3: 3. Axial enhanced CT images in the corticomedullary phase (a) and in the excretory phase (b) show unilateral global absence nephrogram in the same patient shown in fig.2 a few months later.
Fig. 4: 4. Unenhanced (a) and enhanced (b, c) axial CT images show a focal absence of nephrogram (arrow). At first sight this wedge shape looks like a kidney infarction, but looking to image (a) we can see it has fat density. This is a focal absence secondary to the presence of an angiomyolipoma.
Fig. 5: Unenhanced (a) and enhanced (b, c) CT images show a focal absence of nephrogram (arrow). Looking to image (a) we can see it has high density and it has no contrast enhancement in images (b) and (c). This is a focal absence secondary to the presence of high-density cyst. Note also caliectasis (*) secondary to bilateral junction syndrome.
Fig. 6: Enhanced CT images (a) and (b) show a focal nephrogram alteration secondary to the presence of a renal cell carcinoma (arrow).
Fig. 7: Enhanced CT images (a) and (b) show a focal absence of nephrogram (arrow) due to a hemodynamic change secondary to the presence of an iatrogenic intrarenal pseudo-aneurysm (arrowhead). Note this patient has an axillary-femoral bypass graft (*) due to occlusion of the abdominal aorta.
Fig. 8: Unenhanced (a) and enhanced (b) CT images shows a focal absence of nephrogram (arrow) on (b) due to a hemodynamic change secondary to the presence of a traumatic intrarenal pseudo-aneurysm (arrowhead). Notice on (a) high-density area corresponding to recent bleeding leading, to perirenal fluid accumulation.
Fig. 9: Enhanced CT images (a) and (b) show a large segmental absence of nephrogram (*) on the right kidney secondary to traumatic renal artery injury (arrowhead). The left kidney, not visible in images (a) and (b) has an ectopic position in the pelvis (c) and shows a small segmental absence nephrogram (arrow) also secondary to traumatic vascular changes.
**Fig. 10:** 10. Enhanced CT images (a) and (b) show a focal absence of nephrogram (arrow), at two different levels, secondary to traumatic kidney laceration. Also notice perirenal fluid.
**Fig. 11:** Unenhanced (a) and enhanced (b, c) CT images show of a focal absence of nephrogram (arrow) secondary to traumatic kidney rupture. Perirenal collection is also present (*).
Fig. 12: 12. Enhanced CT images (a), (b), (c), and (d) show different phases and locations of a multiple segmental absence of nephrogram, bilaterally, secondary to underperfusion, in a patient with septic shock.
Fig. 13: Enhanced CT images (a), (b) and (c), show unilateral segmental absence nephrogram due to ischemic alterations in a patient submitted to ureteropyeloplasty. Image (d) shows lack of perfusion on the left kidney on the area corresponding to the absence of nephrogram. Note the presence of a kidney catheter on the left, fluid in the perirenal and anterior and posterior left pararenal spaces and ascites.
**Fig. 14:** 14. Enhanced CT images (a), (b), (c), and (d) show of multiple segmental absence of nephrogram bilaterally due to arterial embolization (arrowheads) in a patient with endocarditis.
**Fig. 15:** Enhanced CT images show an area of focal absence of nephrogram in the left kidney secondary to kidney infection (arrow). Notice perirenal fat stranding surrounding the infection site.
Fig. 16: Enhanced CT images (a), (b), (c) and (d) show two areas of focal absence of nephrogram in the left kidney secondary to kidney infection (arrows). Note adjacent to the most anterior lesion the presence of air and fat stranding. Fluid collection (*) in the anterior pararenal space is also present.
Fig. 17: Enhanced CT images (a), (b), (c) and (d) show a rim pattern nephrogram on the right kidney (arrow) and a focal absence of nephrogram (*) on the left. These alterations were secondary to bilateral infarction. The rim sign represents preserved subcapsular perfusion via collateral flow.
**Fig. 18:** 18. Enhanced CT images (a) and (b) show a rim pattern nephrogram on the right kidney (arrow) in a patient with renal infarction secondary to traumatic injury.
**Fig. 19:** 19. Enhanced CT images (a) and (b) show a "rim-like" pattern on the right kidney (*). At first it can seem a "rim-sign" but looking carefully, we can see the image represents a kidney with chronic dilatation of excretory system with cortical thinning mimicking the rim sign. Note the slowed progression of nephrogram on the right secondary to this chronic obstruction.
Fig. 20: 20. Enhanced CT images (a), (b), (c) show a slowed temporal progression of contrast enhancement on the left kidney associated with dilatation of the excretory system (*) secondary to chronic obstruction. Image (d) shows urothelial carcinoma of the bladder involving the left ureteral orifice (arrow) and causing the chronic obstruction.
**Fig. 21:** Enhanced CT images (a), (b), (c) show an asymmetrical progression of contrast enhancement on the left kidney secondary to aortic dissection (arrowhead) that reduces arterial flux. Note the presence of parapielic (*) cysts on the left kidney and cortical cysts (circle) on both kidneys.
Fig. 22: Enhanced CT images show a slowed temporal progression of contrast enhancement on the left kidney associated with renal vein malignant thrombosis (*). The renal cell carcinoma is seen on image (a) (arrow).
Fig. 23: Enhanced CT images show a slowed temporal progression of contrast enhancement on the left kidney associated with dilatation of the excretory system (*) secondary to chronic obstruction, caused by an urothelial tumor of the ureter (not shown).
Fig. 24: Enhanced CT images show delayed pyelogram on the left kidney associated with dilatation of the excretory system (*) secondary to urolithiasis obstruction (not shown).
**Fig. 25:** Enhanced CT images show a slowed temporal progression of contrast enhancement on the right kidney in a patient with polycystic kidney disease, secondary to a pyelonephritis.
Fig. 26: 26. Enhanced CT image shows a discrete bilateral striated pattern in a patient with kidney underperfusion secondary to severe hypotension.
Fig. 27: Enhanced CT images show a striated pattern secondary to pyelonephritis in a renal allograft.
Fig. 28: Enhanced CT images show a striated pattern secondary to pyelonephritis.
Fig. 29: Unenhanced CT image shows a bilateral persistent nephrogram 24h after the first CT. This patient developed a contrast material induced nephropathy. Note the absence of contrast in the aorta and the presence of contrast in the gallbladder indicating its biliary excretion.
Conclusion

Given the great use of MDCT critical analysis of the CT nephrogram has assumed an important role in the evaluation of urinary tract disease. Recognizing these radiologic changes within the proper clinical setting allows the radiologist to suggest the correct diagnosis, in many instances before characteristic clinical findings become evident.

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