Positive and differential imaging diagnosis in renal vascular ischemia

Poster No.: C-0899  
Congress: ECR 2013  
Type: Educational Exhibit  
Authors: A. Ionescu\textsuperscript{1}, I. G. Lupescu\textsuperscript{1}, R. A. Capsa\textsuperscript{2}; \textsuperscript{1}Bucharest/RO, \textsuperscript{2}Bucuresti/RO  
Keywords: Kidney, Vascular, CT, MR, Computer Applications-Detection, diagnosis, Ischemia / Infarction, Embolism / Thrombosis, Transplantation  
DOI: 10.1594/ecr2013/C-0899

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method ist strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Learning objectives

1. List the epidemiologic, etiologic and clinical features of renal vascular ischemia.

2. To review and illustrate the diagnostic imaging (CT and MRI) findings in acute and chronic renal vascular pathologies.

3. To recall the most important differential diagnoses for renal vascular ischemia.

Background

Renal vascular anatomy

In the majority of the human subjects, each kidney is supplied by one renal artery arising from the abdominal aorta, but in approximately 30% of individuals more than one artery can be present (1-2). The main renal artery divides into four anterior branches at the renal hilum: apical, upper, middle and lower anterior segmental arteries. Further divisions include the lobar, interlobar, arcuate and interlobular arteries. (Fig. 1 on page 6, Fig. 2 on page 7) Accessory renal artery is the most common variation; they usually arise from the aorta or iliac arteries and perfuse the upper or lower renal pole. Prehilar branching is another common variant; it represents the branching of the main renal arteries into segmental branches at a more proximal level than the renal hilum (3).

Renal veins course anterior to the renal arteries. The renal cortex is drained sequentially by the interlobular, arcuate, interlobar and lobar veins, and then they converge to form the main renal vein (Fig. 3 on page 8).

Renal ischemia

Definition. Renal ischemia is defined as blood volume deficiency in one or both kidneys usually due to functional constriction or actual obstruction of a blood vessel, which, if untreated, can lead to cortical and medullary necrosis. Renal infarction can be due to injuries to the renal artery, renal vein or, in cases of abdominal trauma, injuries to the renal pedicle.
Incidence. In autopsy study, the incidence of renal infarction is 0.48-1.4%. The mean age of the patients is 67.4 ± 21.1 (range 30±87 years) (4-6). The main cause of renal infarction due to arterial impairment is atherosclerotic vascular disease and the main cause due to vein impairment is tumoral thrombosis.

Etiology (7).

Causes affecting the artery:
- Thromboembolism (arterial fibrilation, miocardial infarction, mixoma, bacterial endocarditis)
- Aneurysm of the aorta or renal artery
- Dissection of the aorta or renal artery
- Atherosclerosis
- Transcatheter embolization and other endovascular procedures
- Fibromuscular dysplasia
- Blood clotting disorders
- Vasculitis: - Polyarteritis nodosa
  - Takayasu arteritis
  - Systemic lupus erythematosus
  - Drug-induced vasculitis
- Trauma: - Avulsion / Occlusion of the renal artery
  - Penetrating vascular injury
- Congenital pathology
- Sepsis
- Paraneoplastic syndrome
- Kidney transplant
- Retroperitoneal pathology

Causes affecting the vein:
- Trauma: - Acute venous occlusion / avulsion
- Tumoral causes: - Grawitz tumor
- Wilms tumor
- Retroperitoneal tumors
- Adrenal tumors
- Transitional cell carcinoma

- Non tumoral causes: - Nephrotic syndrome
- Antiphospholipid syndrome
- Amyloidosis
- Systemic lupus erythematosus
- Sickle cell disease
- Diabetic nephropathy
- Dehydration
- Blood clotting disorders
- Paraneoplastic syndrome
- Kidney transplant

- Retroperitoneal pathology

**Physiopathology.** Acute kidney injury (AKI) induced by ischemia can result in damage to both the tubular as well as the microvascular compartment which is leading to sustained reductions of glomerular filtration rate (GFR). Of central importance in this process is the activation of inflammatory pathways that are influenced by factors released by damaged proximal tubules as well as adhesion of damaged microvascular cells. Infiltrating leukocytes may impinge on renal blood flow (RBF) either by secreting vasoactive factors or by contributing to the disruption of flow by physical interference (11-13). In addition, exacerbated hypoxia leading to tubular obstruction may contribute to reductions in GFR - Fig. 4 on page 9.

**Clinical features.** The commonest presenting features are flank pain, tenderness in the epigastric region and the costovertebral angle, pallor, nausea, vomiting, fever, hematuria, hypertension, signs of peripheral ischemia, proteinuria, peripheral edema and, rarely, oliguria and other uremic signs. Gradual onset causes symptoms of nephrotic syndrome.

**Laboratory tests.** The laboratory tests showed elevated levels of serum LDH, leucocytes, blood urea nitrogen and serum creatinine.

All patients presenting with the triad: high risk of a thromboembolic event, persisting flank/abdominal/lower back pain, elevated serum levels of lactate dehydrogenase and/or hematuria within 24 hours after pain onset, should undergo imaging examination to rule out or to prove acute renal infarction.
The main clinical differential diagnostics are renal colic, pyelonephritis and renal carcinoma (8-10).

**Imaging.** The first line of imaging modality for the suspicion of renovascular disease is *abdominal ultrasound* which may demonstrate focally increased echogenicity and absence of intrarenal arterial sign on color flow Doppler (12). *Contrast enhanced CT* and *MRI* are considered a safe non-invasive, easily available and highly accurate methods for detecting and assessing the causes for renal infarction (1,2,3).

*Renal angiography* is not usually required unless an operative approach is considered (2,15).

*99 M Tc DMSA* scan demonstrate photon deficient area in relation to infarcted region. (14-15)

**CT findings** (16-22)
CT finding depend on both the extent and the age of the infarction. The size and the shape of a renal infarction are determined by the size of the occluded artery or vein.

If a *major renal artery branch* is occluded, an acute focal infarct results, which manifests as a wedge-shaped, low-attenuation renal parenchymal lesion on contrast-enhanced CT (Fig. 5 on page 10 ). The base of the wedge is contiguous with the renal capsule, and the apex is directed toward the renal hilus. There is usually sharp margination between infarcted tissue and the adjacent normal nephrogram. Emboli and vasculitis cause multiple, often bilateral, focal renal infarcts. When *smaller intrarenal arteries* are occluded, the CT findings are less specific and consist of multiple low-attenuation defects that are scattered throughout the nephrogram, although peripherally located. Vascular thrombosis in sickle cell disease causes multiple foci of "slitlike" areas of attenuation. (22)

If the *main artery* is occluded, global infarction of the kidney ensues. On contrast enhanced CT scans the effected kidney shows lack of enhancement and high density cortical rim which represents perfusion supplied by collaterals to the outer rim of cortex (2-4 mm) - the "cortical rim" sign, which is considered a diagnostic sign.

The "cortical rim" sign is reported to be present in #50% of focal or global infarcts and is thought to be due to an intact renal collateral circulation (renal capsular vessels, peripelvic vessels and periureteral vessels). In spite of the presence of renal collateral circulation, it takes a minimum period of 8 h for this circulation to expand and become apparent on CT after 1 week. The "cortical-rim" sign has been reported in arterial renal infarction, renal vein thrombosis and acute tubular necrosis. Fig. 6 on page 11
"Flip-flop enhancement" pattern has also been described in renal infarction. This refers to a hyperdense region on delayed CT images in the same region as a hypodense region on the nephrographic phase images. This sign is due to extravasation of contrast material in areas of ischemia caused by disruption of the glomerular membrane. This sign is suggesting ischemia rather than necrosis and may be seen in inflammatory states (18-19, 21).

Acute focal and global renal infarctions can be associated with perinephric fluid collections, hemorrhage and thickening of the renal fascia.

**Old infarcts** manifest on CT as renal cortical scars or as a small, shrunken kidney with smooth or irregular contours (Fig. 7 on page 12).

**Renal vein thrombosis** may affect both kidneys and may occur in an acute or a chronic form. Renal vein thrombosis is more common on the left side, presumably because of the longer left renal vein. With **acute renal vein thrombosis**, the kidneys appear enlarged and excretory function diminishes. With **chronic thrombosis**, ureteral indentations resulting from collateral venous channels may be present.

**MRI findings:** (23-26) Table 1 on page 13

Renal infarcts can demonstrate a variety of signal intensities on native T1- and T2-weighted images depending on the time between the onset of the vascular insult and the MR examination.

MR imaging can demonstrate renal swelling, indistinct corticomedullary differentiation on T1-weighted images, and decreased signal intensity of the renal cortex and medulla. A band of low signal intensity in the outer part of the medulla is typically present, though this finding can also be noted in patients with hemorrhagic fever with renal syndrome (HFRS).

Gadolinium-enhanced MR is an excellent imaging modality for the evaluation of the renal arteries and veins and parenchymal changes (which are similar to contrast enhanced CT).

While a nonenhancing filling defect within the renal vein is consistent with bland thrombus, demonstration of thrombus enhancement on post-contrast images is characteristic of tumoral thrombus.

**Images for this section:**
**Fig. 1:** Normal renal segmental arterial anatomy. Drawings of the kidney from an anterior (a) and posterior (b) perspective demonstrate the apical, anterior, posterior, and basilar branches of the renal artery.
Fig. 2: Coronal volume rendering images show anterior and posterior views of normal segmental anatomy of the renal artery. The first division is the posterior branch (large red arrow). The posterior branch gives two segmental branches (black arrows), supplying the posterior central portion of the kidney. The main renal artery then continues its course before branching into four anterior branches (small white arrows) at the renal hilum.
**Fig. 3:** A. Drawings of the normal renal vein anatomy. B. MPR images show the normal renal vein anatomy. The renal veins (large blue arrow) flows into the inferior vena cava (blue asterisk). The left renal vein courses anterior to the aorta (red asterisk).
Fig. 4: Schematic representation of tubular and vascular damage leading to reduction of glomerular filtration rate (GFR) in ischemic acute kidney injury.
Fig. 5: Acute thrombosis of the left renal artery with acute renal infarction from arterial fibrillation - contrast-enhanced CT evaluation in a male patient, age 45 y ; renal infarction demonstrated by a hypodense wedge-shape area (orange arrow) in the left kidney determined from occlusion of a major branch of the renal artery.
**Fig. 6:** "Cortical-rim" sign (orange arrow): abdominal trauma with thrombosis of renal artery, lack of enhancement of renal parenchyma and perfusion of the outer rim of the cortex.
**Fig. 7:** Aspect of old renal infarcts (orange arrow) in different patients - small, shrunken right kidney (a) and left kidney (b) due to renal artery stenosis; multiple bilateral renal cortical scars (c) with a small left renal kidney due to systemic atherosclerosis with calcified plaque on both renal arteries.
<table>
<thead>
<tr>
<th>Time</th>
<th>T1</th>
<th>T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;6 hours</td>
<td>• HYPOINTENSE</td>
<td>• HYPOINTENSE</td>
</tr>
<tr>
<td></td>
<td>• OBLITERATED CORTICOMEDULLARY DEMARCATION</td>
<td></td>
</tr>
<tr>
<td>&gt;24 hours</td>
<td>• HYPERINTENSE</td>
<td>• HYPOINTENSE</td>
</tr>
<tr>
<td></td>
<td>INTERSTITIAL HEMORRHAGE</td>
<td></td>
</tr>
<tr>
<td>&gt;3 days</td>
<td>• HYPERINTENSE</td>
<td>• HYPERINTENSE</td>
</tr>
<tr>
<td></td>
<td>COAGULATIVE NECROSIS</td>
<td></td>
</tr>
<tr>
<td>2-4 weeks</td>
<td>• HYPOINTENSE</td>
<td>• HYPOINTENSE</td>
</tr>
<tr>
<td></td>
<td>ORGANIZING FIBROSIS</td>
<td></td>
</tr>
</tbody>
</table>

**Table 1:** Native MRI aspects of renal infarction depending on time.
Material and Methods:

Retrospective study over a period of five years (2007-2011) of CT and MRI imaging studies in the Radiology and Medical Imaging Department of Fundeni Clinical Institute performed in patients with acute renal failure, hypotension, specific signs of heart disease or trauma and in which ultrasound evaluations have revealed changes in the renal artery or vein, incompletely characterized. The study included patients with renal transplant showing impaired vascular pedicle of the graft.

CT protocol:

- unenhanced and enhanced multidetector spiral CT of the abdomen (collimation: 5; pitch: 1-1,5 )
- contrast media injection of nonionic iodinated contrast material 1,5 ml/kg at a flow rate of 3-4 ml/sec
- postcontrast CT acquisition:
  - corticomedullary phase at 25-35 sec after the start of the injection when renal arterial and venous opacification is greatest
  - nephrografic phase at 100-120 sec after the contrast material administration
  - excretory phase at 5-7 min after the contrast material administration.

MRI protocol:

- MR imaging was performed an a 1.5 T using a Phased-array Torsopa coil;
  - respiratory gating SE sequences with slice thickness 7 mm and spacing 2 mm
  - 3D FSPGR T1 with gadolinium injection (0,2 ml/kg, flow 2 ml/s) with bolus detection and multiplanar reconstructions.

Results: Table 2 on page 20

The imaging retrospective analysis includes 107 patients of which 65 presented vein thrombosis (from which most associated with malignancy) and 39 patients had impaired renal artery (total or partial) and 3 patients had impaired both renal artery and vein.
ASPECTS OF RENAL INFARCTION DUE TO ARTERIAL IMPAIRMENT:

We detected 39 patients with renal artery impairment from which 7 cases were in patients with renal transplant; 19 patients had total renal artery thrombosis (associated with renal trauma or thrombembolism) and 20 patients had partial renal artery thrombosis. Acute renal artery thrombosis was found in 19 cases and chronic renal artery impairment was found in 20 patients (form which 19 cases were associated with sistemic atherosclerosis).

A. IN ACUTE STAGE (19 cases):

1. Renal artery thrombembolism (Fig. 8 on page 21)

The most common cause for renal infarction is thromboembolism or in-situ thrombosis, which is less common. In patients with preexisting atrial fibrillation, the incidence of renal thromboembolism is said to be approximately 2%. The bilateral occurrence in this context responsible for acute renal failure is exceptional.

The mechanism may be embolic, typically caused by blood or cholesterol clots occluding the renal artery or branch vessels; this is the most common cause and is often of cardiac origin, such as atrial fibrillation, myocardial infarction with left ventricular thrombus, mitral stenosis, atrial myxoma or infective endocarditis.(23-25)

2. Renal artery involvement in partially thrombosed aortic aneurysms or aortic dissection.

Renal infarction can appear in aortic aneurysm with intraluminal thrombus (Fig. 9 on page 21) or when acute dissection develops and obstructs the renal arteries (Fig. 10 on page 22, Fig. 11 on page 23). The dissection may extend into a renal artery or a normal renal artery arising from the true lumen may have reduced flow due to collapse of the true lumen. The intimal flap may intermittently cover the renal arterial origin or may extend into the main and segmental renal arteries, thus interrupting renal blood flow. (30-31)

3. Abdominal trauma Fig. 12 on page 24

Primary vascular injury of the kidney following blunt trauma occurs when there is occlusion of the main renal artery by an intimal flap as a result of deceleration. Occlusion of a segmental renal artery branch results in a segmental renal infarct. On occasion, the diagnosis may be delayed because hematuria is absent. Renal pedicle injury is rarely associated with thrombosis or laceration of the renal vein. (26-29)
Typical parenchymal findings in posttraumatic occlusion of the main renal artery include an absent nephrogram with or without the "cortical rim" sign. Absence of perinephric hematoma is a hallmark of renal artery occlusion unless associated renal lacerations are present. Hemorrhagic infiltration or minimal hematoma is present around the proximal renal artery.(29)

B. IN CHRONIC STAGE (20 cases):

1. Atherosclerotic renal artery disease (19 cases) - Fig. 13 on page 25

Atherosclerotic renal artery stenosis is the most common primary disease of the renal arteries, and it is associated with two major clinical syndromes, ischemic renal disease and hypertension. It shows a frequency of 4-12% in the general population. Renovascular hypertension accounts for 1%-4% of all patients with hypertension. (32-33)

Atherosclerotic lesions usually occur at the origin of the renal artery or within the proximal 2 cm and in 20-50% can be bilateral.

Lesions of atherosclerosis are either eccentric or concentric with respect to the artery. Poststenotic dilatation of the artery may be present.

2. Takayasu arteritis (1 case) - Fig. 14 on page 26

Takayasu disease is a rare form of granulomatous arteritis of unknown cause that commonly involves the aorta, its major branches, and the pulmonary arteries. It results in stenosis, occlusion, dilatation or formation of aneurysms in the involved blood vessels. Of these, stenoses or obstructions are the most common, frequently involving the abdominal aorta and the renal arteries, and often resulting in renovascular hypertension. (34) Bilateral damage is more common.

Contrast-enhanced CT and MRI may demonstrate thickening of the arterial wall with crescents and indistinct outlines. (35-36)

ASPECTS OF RENAL INFARCTION DUE TO VENOUS IMPAIRMENT:

We detected 65 cases of renal vein thrombosis from which 57 patients had tumoral type thrombosis (mostly associated with Grawitz tumor) and 8 patients had acute non-tumoral type thrombosis from which 4 patients had nephrotic syndrome, 1 patients had renal trauma and 3 patients had a septic shock.

A. TUMORAL TYPE THROMBOSIS (57 cases):
Renal vein thrombosis associated with tumor is frequently caused by direct tumor extension and occurs most commonly in cases of renal cell carcinoma (Fig. 15 on page 27, Fig. 16 on page 28) and occasionally in cases of transitional cell carcinoma or Wilms tumor. Tumor thrombus in the renal vein may also result from a left adrenal tumor. Left ovarian vein thrombosis may extend into the left renal vein.

Contrast-enhanced CT shows thrombus in a thick-walled renal vein with or without extension into the IVC. The presence of inhomogeneous enhancement in the thrombus is indicative of tumor involvement. (39-40)

B. NON-TUMORAL TYPE THROMBOSIS (8 cases)

Non-tumoral thrombosis of the renal vein is usually caused by an underlying abnormality of the clotting system or the kidney itself or, in infants, dehydration. Non-tumoral type of venous thrombosis can be acute (Fig. 17 on page 29, Fig. 18 on page 30) in which case contrast-enhanced CT or MRI shows a thrombus in a thick-walled renal vein with or without extension into the IVC and increased kidney size with smooth contours, or chronic in which case the affected renal vein becomes attenuated due to retraction of the clot along with development of extensive collateral vessels along the proximal to middle ureter and around the kidney; the kidney shrinks in size, but with smooth contours.

ASPECTS OF RENAL ISCHEMIA DUE TO IMPAIRMENT OF BOTH RENAL ARTERY AND VEIN (3 cases)

Renal ischemic phenomena can appear in context of renal trauma with avulsion of renal pedicle, in case of massive retroperitoneal tumors or retroperitoneal fibrosis that can include the both the artery and vein maintaining vascular permeability, but with reduced blood flow. (Fig. 19 on page 31)

ISCHEMIC VASCULAR DISEASE IN RENAL TRANSPLANT (41-42)

We detected 8 cases with renal vascular pedicle impairment in the transplanted kidney, from which 7 cases had renal artery thrombosis and 1 case had both renal artery and vein thrombosis (Fig. 20 on page 32).

Renal artery thrombosis is an early complication in renal transplant (within the first week), while renal artery stenosis is considered a late complication (after one month). (Fig. 21 on page 33)
Complete renal vein thrombosis (RVT) after renal transplantation has an incidence of 0.55-3.4% and accounts for up to one-third of early allograft losses. RVT develops gradually, presumably beginning as partial vein thrombosis thus there is a narrow but important window for early diagnosis of partial RVT, which would allow for timely intervention.(42)

**DIFFERENTIAL DIAGNOSIS:**

The main differential diagnosis of acute renal infarction is acute pyelonephritis because both conditions often demonstrate wedge-shaped, low-attenuation renal lesion on CT and MRI and manifest as acute onset of flank pain and fever. Pyelonephritis lesions demonstrate delayed enhancement (from stasis of contrast material in edematous tubules) and lack of "cortical-rim" sign. (Fig. 22 on page 34)

Small renal infarcts may also be confused with focal lymphomatous lesions or metastasis. (Fig. 23 on page 35)

Different types of renal tumors or transitional cell tumors that extend into the renal parenchyma can be included in the differential diagnosis because they can demonstrate low-attenuation areas with low-enhancement. (Fig. 24 on page 36)

**IMAGING RECOMMENDATIONS.**

- When a patient is suspected of a renovascular disease the first line of imaging modality is abdominal ultrasound which can either exclude or confirm the presence of renal parenchyma changes and also visualize the renal vascular pedicle permeability using Doppler color.
- The next line of imaging modality is CT because of highly accurate results in detecting and assessing the causes for renal infarction; MRI is used in non-emergency cases and in patients with intolerance to iodinated contrast-media.
- Renal angiography should be considered for interventional treatment.

**THERAPEUTIC OPTIONS**

The four current therapeutic options available to treat patients with renal artery disease include (1) medical management, (2) surgical revascularization, (3) percutaneous transluminal renal angioplasty (PTRA), and (4) stents. Guidelines indicate that when renal artery stenosis is less than 50 % and captopril renal scintigraphy is negative, then medical therapy should be followed and imaging reevaluation every six month; if renal artery stenosis is more than 50% and captopril renal scintigraphy is positive, then PTRA or stenting should be taken into consideration.
For acute renal artery thrombosis, thrombolisis is the first line of treatment and then, according to the experience of the operating team, stenting the obstructed artery. Open surgery is not recommended other than in the case of trauma, where other problems may indicate the need for surgery anyway.

For **acute renal vein thrombosis**, the line of treatment are anticoagulants. This method has high success rate in acute renal vein thrombosis and even preserved total renal function, but it is discussed in chronic renal vein thrombosis. (43-45)

**PROGNOSIS**

The most common sequel to renal infarction is loss of renal function and persistent hypertension. However, many patients go on to have normal kidney function with no permanent hypertension.

A small percent will need dialysis, 8% in one case series, related to the extent of renal parenchyma alteration (9).

**Images for this section:**
**Table 2:** Etiology distribution date in study group.

**Fig. 8:** Acute thrombosis of the right renal artery, anterior branch (red arrow) with renal infaction on the anterior surface, the upper part (orange arrow)- contrast-enhanced CT examination in a 56 years old female patient; reevaluation in 3 months demonstrates renal parenchyma scar (orange arrow).
Fig. 9: Aortic aneurysm with intraluminal thrombosis with chronic renal ischemic alteration in the left kidney - contrast-enhanced CT evaluation in a male patient age 78 with systemic arterial hypertension; multiple left renal arteries (seen in coronal MPR - d); one renal artery (red arrow) branches from intraluminal thrombosis with other multiple left renal arteries permeable (orange arrow). Note the reduced dimensions of the left kidney with irregular contours.
Fig. 10: Aortic dissection type B with left renal artery dissection (red arrow) with minimal left renal parenchyma ischemia - contrast-enhanced CT evaluation in a male patient, 63 years old; note that the right renal artery (orange arrow) emerges from the true lumen (t); minimal delayed left renal nephrogram.
Fig. 11: Aortic dissection type B with left renal artery dissection (red arrow) - contrast-enhanced CT examination in a 76 years old patient; note the right renal artery (green arrow) which emerges from the false lumen (f) with a faint opacification of the apical branch and reduced dimensions of the right kidney with perirenal fat stranding (orange arrow).
**Fig. 12:** Thrombosis of the right renal artery (red arrow) with almost entirely right kidney ischemia - contrast-enhanced CT evaluation of a 61 years old female patient with right kidney trauma; note the increase in size of the right kidney, with smooth contours and lack of enhancement of the renal parenchyma with "cortical-rim" sign (orange arrow) with maintaining permeability of small intrarenal branches.
Fig. 13: Atheromatosis plaques with partial thrombosis of the left renal artery (red arrow) with chronic ischemic renal parenchyma alteration - contrast-enhanced CT evaluation in a 67 years old female patient with systemic atheromatosis; note the reduced dimensions of the left kidney with smooth contours.
Fig. 14: Partial thrombosis at the proximal region of the left renal artery (red arrow) - MRI examination of a female patient age 35 years diagnosed with Takayasu disease with ischemic renal parenchyma alterations; the left kidney has reduced dimensions compared to the right one, with indistinct corticomedullary differentiation on T1-weighted images.
Fig. 15: Tumoral thrombosis of the right renal vein (red arrow) extended into the inferior vena cava - contrast-enhanced CT examination of a 51 years old male patient with right kidney Grawitz tumor (blue arrow); note the apical part of the right kidney has indistinct corticomedullary differentiation and delayed nephrogram.
Fig. 16: Tumoral thrombosis of the left renal vein (red arrow) - gadolinium-enhanced MRI examination in a 72 years old male patient diagnosed with left kidney Grawitz tumor (blue arrow).
**Fig. 17:** Non-tumoral acute left renal vein thrombosis (red arrow) with partial left renal kidney parenchyma ischemia (blue arrow) - contrast-enhanced CT examination in a 17 years old female patient diagnosed with nephrotic syndrome; note the enlarged left kidney with smooth contours.
Fig. 18: Non-tumoral acute partial right renal vein thrombosis (red arrow) - gadolinium-enhanced MRI examination in a 32 years old male patient with septic shock; note the perirenal inflammatory alterations.
**Fig. 19:** Ischemic changes in both renal parenchyma (orange arrow)- contrast-enhanced CT examination 67 years old female patient with non-Hodgkin’s lymphoma; the retroperitoneal tumor includes the renal arteries on both sides (red arrow) and left renal vein (blue arrow). Note the extensive collateral vessels in perirenal space (green arrow).
Fig. 20: Total thrombosis of renal artery and vein with renal parenchyma necrosis (orange arrow) in a 34 years old male patient with renal graft; note the indistinct corticomedullary differentiation on both T2- and T1-weighted images.
Fig. 21: Renal artery thrombosis with renal parenchyma ischemic changes due to a pseudoaneurism at the arterial anastomosis - contrast-enhanced CT of a 67 years old male patient with kidney transplant; note a small anterior apical branch permable (red arrow), renal parenchyma ischemia (orange arrow) and normal renal parenchyma on the posterior side (yellow arrow);
**Fig. 22:** Acute pyelonephritis - contrast-enhanced CT examination in a 25 years old female patient with septic shock. The inflammatory changes in left renal parenchyma (orange arrow) appear hypodense in the cortico-medullary (a) phase and the nephrografic phase (b) and show delayed contrast uptake (c).
Fig. 23: Focal lymphomatous renal lesions (orange arrow) - contrast-enhanced CT examination of a 45 years old female patient with Hodgkin’s lymphoma.
Fig. 24: Transitional cell carcinoma (green arrow) - contrast-enhanced CT examination in a 68 years old female patient. Hypodense area in upper left renal parenchyma (orange arrow) due to extension of the renal pelvis tumor into the parenchyma.
Conclusion

1. Renal infarction is a rare and the diagnostic is easily missed or delayed because it can clinically mimic other conditions, the use of imaging diagnostic being indispensable.

2. The main cause for renal artery ischemia is atherosclerotic pathology that can be correlated with secondary hypertension. For renal venous ischemia, the main cause is tumoral type thrombosis related to Grawitz tumor.

3. In assessing reno-vascular pathology, angio-CT and angio-MRI plays an important role both in the evaluation and management of primary disease and the secondary manifestations.

4. CT is considered the first line of imaging modality for renovascular disease; MRI is used for patients with intolerance to iodinated contrast media or in case of limited kidney function for evaluation of renal parenchyma without the use on contrast media (sequences as phase-contrast MR angiophagy or state-free precession MR angiography).

References


17. Urban BA, Fishman EK. Tailored helical CT evaluation of acute abdomen. Radiographics. 20 (3): 725-49


**Personal Information**

andreea_goia@yahoo.com

ilupescu@gmail.com

Radiology and Medical Imaging Department Fundeni Clinical Institute, University of Medicine and Pharmacy " Carol Davila" Bucharest, Romania