Modern imaging of chronic renal infections

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Learning objectives

1. To illustrate the fundamental imaging features of the different forms of chronic renal infections;

2. To illustrate the modern imaging of renal tuberculosis with multidetector CT urography;

Background

From the pathological point of view the chronic renal infections include two types, interstitial chronic pyelonephritis and granulomatous pyelonephritis, namely xantogranulomatous pyelonephritis, renal tuberculosis, and malacoplakia. Imaging allows to depict both factors causing obstruction and infection such as stones and the renal and extrarenal extension of the disease. We shall focus on the following pathological entities: chronic pyelonephritis, renal tuberculosis, xanthogranulomatous pyelonephritis, renal replacement lipomatosis, malacoplakia, and cholesteatoma, and chronic renal infections in AIDS. We shall not deal with chronic pyonephrosis and of chronic abscess, because the features are common to the corresspective acute disease.

Imaging findings OR Procedure details

Chronic pyelonephritis

Chronic pyelonephritis is a somewhat controversial disease from a pathogenetic standpoint. Despite the ongoing debate on whether the condition is an active chronic infection, arises from multiple recurrent infections, or represents stable changes from a
remote single infection, its radiologic appearance is the same [1]. Chronic pyelonephritis is a chronic tubulo-interstitial fibrosing nephritis with long-standing recurrent infection and ongoing renal destruction, that may be unilateral or bilateral and involves renal parenchyma, renal calices and renal pelvis. On the other hand, the term chronic pyelonephritis does not fit with the residuum of old disease not presently active (e.g. reflux nephropathy). Chronic pyelonephritis is more frequent in diabetic population, with 20-40% of incidence compared to 2-6% in normal population according to autopsy series. Kidneys are dimensionally reduced with atrophy, parenchymal scars, irregular margins and cortical thinning and hypertrophy of residual normal tissue, and dilation of corresponding renal calices - caliceal clubbing secondary to retraction of the papilla from overlying scar (Figure 1 on page 10; Figure 2 on page 11), dilatation of the calyceal system [1], and increased parenchymal echogenicity with poor corticomedullary differentiation.

The goals of imaging in chronic recurrent pyelonephritis are the detection of chronic renal damage, and the detection of abnormalities that are often the cause of recurrence. However it should be stressed that there are no radiologic features that at a single point in time reliably indicate activity of the process. Therefore the goals of radiological investigations become the detection of abnormalities that are often the cause of the recurrence (i.e. infectious stones) and the detection of the chronic renal damage. In the past, intravenous excretory urography and nephrotomography, and nowadays CT-urography are often capable to acheive these two goals.

Findings related to the chronic renal damage (decrease of the kidney size, parenchymal scars, caliceal distortion - Figure 1 on page 10 -) are easily depicted at intravenous excretory urography.

Multidetector computed tomography (CT) urography and magnetic resonance (MR) imaging readily identify the chronic renal parenchyma damage and calyceal distortion and clubbing (Figure 2 on page 11).

**Renal tuberculosis**

Genitourinary *tuberculosis* is the most common manifestation of extrapulmonary *tuberculosis* [2, 3] accounting for 15%-20% of infections outside the lungs [4]. Approximately 4%-8% of patients with pulmonary tuberculosis will develop clinically significant genitourinary infection [4]. *Mycobacterium tuberculosis* reaches the genitourinary organs, particularly the kidneys, by the haematogenous seeding from disease in the lungs. The seeding occurs at the time of the initial lung infection with seeding of *Mycobacterium tuberculosis* in the periglomerular and peritubular capillary bed. Small granulomas form in the renal cortex bilaterally, adjacent to the glomeruli, and remain stable for many years [5]. A high rate of perfusion and favorable oxygen tension increase the likelihood of bacilli proliferating in this location [4]. In patients with intact cellular immunity, the disease remain confined to the renal cortex, while in some patients,
breakdown of host defense mechanisms leads to reactivation of the cortical granulomas with enlargement and coalescence and organisms spread into the real medulla causing a papillitis which may extend into the collecting system [5]. In fact, after capillary rupture the organisms migrate to the proximal tubule and loop of Henle with eventual development of enlarging, caseating granulomas and papillary necrosis. Granuloma formation, caseous necrosis, and cavitation are stages of progressive infection, which can eventually determine the loss of renal function and calcification of the entire kidney (autonephrectomy).

Despite haematogenous seeding of both kidneys, clinically significant disease is usually limited to one side and approximately 75% of renal tuberculous involvement is unilateral. The renal disease remains quiescent until there is an insult to the host's immunity at which time reactivation occurs. Patients with genitourinary tuberculosis typically have local symptoms including frequent voiding and dysuria. Hematuria can be either microscopic or macroscopic. Symptoms may also include back, flank, or abdominal pain [4, 6]. Constitutional symptoms such as fever, weight loss, fatigue, and anorexia are less common [4, 6]. Laboratory abnormalities include pyuria, proteinuria, and hematuria. Standard urine cultures can be normal. Furthermore, the presence of routine urinary tract pathogens can delay the diagnosis of coexistent tuberculosis [4].

*Mycobacterium tuberculosis* is isolated from the urine in 80%-95% of patients with genitourinary tuberculosis. In adults renal tuberculosis is the most known etiology of infundibolar strictures with consequent hydrocalyx. Obstruction may develop early or during the healing phase, even while the patient is receiving antituberculous therapy.

It must be underlined that each finding of renal tuberculosis can be caused by other diseases, but multiple abnormalities are usually present and allow a correct diagnosis. That's why renal tuberculosis is called the "great imitator".

The gray-scale US apperance of renal tuberculosis is not specific. US is advisable to evaluate the non-functioning kidney at intravenous excretory urography and for the follow-up during treatment. Kidney may appear large, normal sized or small, and calcifications are common. Hydronephrosis or hypoechoic parenchymal lesions, which correspond to parenchymal abscesses resulting from caseating necrosis, may be observed.

CT-urography allow a more accurate evaluation of the amount of residual functioning parenchyma and of the extrarenal spread.

On the plain film and unenhanced CT the kidney may appear large, normal sized or small. Calcifications [4] within the renal parenchyma are common (37-71 %) and follow different patterns. Calcifications may be amorphous, granular, lobar, or curvilinear and frequently extend beyond the kidney (e.g. psoas muscle).

Multidetector CT urography, as in the past intravenous excretory urography, is now considered the correct imaging technique to assess the upper urinary tract, including the
involvement in renal tuberculosis. CT-uroography allow a more accurate evaluation of the amount of residual functioning parenchyma and of the extrarenal spread.

The earliest morphologic alterations of renal parenchyma corresponding to calyceal alterations determined by tuberculosis include calyceal erosion ("moth-eaten calyx"), followed by medullary necrosis and papillary necrosis, or infundibular stricture with or without hydrocalyx (Figure 3 on page 12) Single or multiple calices may be involved in one or both kidneys [5]. CT-urography reveals early manifestations of renal tuberculosis, calyceal erosion with progression towards medullary or papillary necrosis (Figure 4 on page 14) The pathologic features of renal TB are extremely different and frequently different features coexist. Common sites of tuberculous strictures are the calyceal neck with hydrocalyx (Figure 5 on page 14; Figure 6 on page 16) phantom calyx (Figure 7 on page 16) infundibulum of a calyx with hydrocalyx or regional or focal hydrocalycosis (Figure 8 on page 17) the uretero-pelvic junction with dilatation of the entire renal pelvis, calyces and infundibola (Figure 9 on page 18) or the lower ureteral segment. Tubercular strictures often coexist with adjacent renal parenchyma scarring. The development of infundibular, pelvic, or ureteral strictures is nearly pathognomonic of renal tuberculosis [5]. An infundibular stricture may result in a "phantom calyx" when that segment of the kidney becomes non-functional. CT is very accurate in demonstrating parenchymal gross calcifications (Figure 8 on page 17).

Renal tuberculosis may manifest as extensive cavitation (open or extensive forms) or fi-brosclerosis (closed forms) [6, 7]. The open or extensive form (Figure 10 on page 18) corresponds to the extension of the caseified tissue necrosis to the intra-renal excretory tract (Figure 11 on page 20) Parenchymal masses can develop which may be calcified [5]. Communication of the granulomas with the collecting system can lead to regional spread of the bacilli into the renal pelvis, ureters, urinary bladder, and accessory genital organs. Extensive cavitation may determine renal caseation, whereas fibrosing reaction of the urinary tract results in obstructive hydronephrosis. When the process spreads into the collecting system, the three ways of evolution of the disease: 1) extensive cavitation (Figure 12 on page 20; Figure 13 on page 20) fibrosclerosis with resulting non-communicating cavities (Figure 14 on page 22; Figure 15 on page 24; Figure 16 on page 24); 3) recurrent "poussées".

The closed or fibrosclerotic form (Figure 17 on page 25) presents a better outcome to therapy and consists in the extension of the caseified necrosis toward the renal parenchyma. The host's healing response induces fibrosis with calcium deposition, focal fibrosis with progressive parenchymal scarring, stricture formation and dilatation of the intra-renal urinary tract and autonephrectomy (no functional contrast excretion). The fibrosclerotic forms of renal TB may appear as: 1) pure fibrosclerosis with parenchymal scar (Figure 18 on page 27) often with evidence of non-communicating cavities (Figure 19 on page 28) 2) reactivation of the granulomatous process over a permanent status of fibrosclerosis with caseous necrosis and cavitation, or mixed fibrosclerotic and cavitating form, and resulting communicating or non-communicating cavities with the intra-renal urinary tract (Figure 20 on page 28) Both forms determine
parenchymal calcifications, deformation of the adjacent renal calices from the simple narrowing of the calyx to medullary and papillary necrosis to obstructive hydronephrosis or hydrocalyx.

The end stage of renal tuberculosis corresponds to extensive renal parenchyma caseation and cavitation (Figure 21a on page 29) resulting in the putty kidney (Figure 21b on page 29) with the entire kidney becoming small, scarred, and densely calcified [8, 9] with autonephrectomy. In the putty kidney a calcified and thick materials fills a dilated collecting system (Figure 22 on page 30).

Xanthogranulomatous pyelonephritis

Xanthogranulomatous pyelonephritis (XGP) is a granulomatous infection characterized by destruction and replacement of renal parenchyma and surrounding tissues with lipid-laden macrophages (xanthoma cells). Females and diabetic patients are more frequently affected. The peak incidence age is the sixth decade of life. This chronic infection is attributed to chronic obstruction or to Proteus or E. Coli infection in 60% of cases. Clinically, XGP manifests with back pain, malaise, weight loss, and urinary tract symptoms such as frequency and dysuria which may be absent in up to 60% of the cases. XGP is almost always unilateral [10]. Diffuse XGP form [11] is much more frequent (85-90%) than focal (tumefactive) form [12]. Three XGP stages are described: 1) Confined to the kidney; 2) Extension to the Gerota's fascia; 3) Involvement of the paranephric spaces and other retroperitoneal structures.

US reveals a multifocal enlargement of the kidney or a pseudotumoral unifocal pattern [13, 14]. Furthermore multiple anechoic or hypoechoic areas with echoic content (dilated calyces and/or cavitary collections filled by inflammatory products) surrounded by a thin hyperechoic zone that represents the surrounding inflammatory reaction, and apparent parenchymal thickening (xanthomatous tissue) can be recognized. Stones and renal enlargement may also be identified. The focal (tumefactive form) of XGP may be considered a pseudotumoral lesion [15]. The principal differential diagnoses include renal cell carcinoma, transitional cell carcinoma of the kidney, renal lymphoma, and hypertrophic chronic pyelonephritis.

IVU can detect three main different features in the diffuse form: calcifications and stones (79% of cases); renal enlargement and absent excretion of contrast agent in the affected kidney (76%) [16]. Stones are usually large and centrally located, often staghorn in type.

CT shows the same three findings detectable by IVU, namely calcifications and stones - and renal enlargement (Figure 23 on page 32) and additional important features as well, i.e. spherical low-density non enhancing areas (from -15 to +30 H.U.) surrounded by enhancing rims corresponding to intrarenal collections (Figure 24 on page 32), possible finer calcifications within the xanthomatous mass, frequent involvement of
The focal form appears as a large hypodense non-enhancing mass possibly with rim enhancement and associated calculus, or as a focal area of renal enlargement with one or more hypo- or anechoic masses. In the focal form all modalities may fail in characterizing lesion. The newer investigative modalities and an increased awareness of XGP should make preoperative diagnosis possible in at least 2/3 of the cases. CT in particular appears to offer a reliable means of diagnosis and spread evaluation. MRI does not seem to give better information than CT. On cross-sectional imaging the focal form of XGP appears as a non-specific renal mass (Figure 26 on page 34). On examination of the macroscopic specimen, the focal (tumefactive) form manifests as a focal renal mass of yellow tissue with regional necrosis and hemorrhage mimicking renal cell carcinoma. Calculi are better depicted with CT but may be seen at MR imaging as areas of signal void within the collecting system. At MR imaging, the renal parenchyma is compressed by dilated calices and replaced by abscess cavities with intermediate signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Cavity walls may show marked enhancement after contrast material administration. Although the focal form of the disease may be misinterpreted as a renal neoplasm, the presence of a staghorn calculus, appropriate clinical presentation (e.g., chronic pyelonephritis in diabetic patients), and the characteristic imaging findings strongly suggest the diagnosis.

The perirenal space is commonly involved in a wide variety of neoplastic and nonneoplastic conditions including chronic fibrosis, often with xanthogranulomatous features (Figure 27 on page 35). The most frequent nontumoral pathology of the perirenal space is the secondary perirenal involvement from retroperitoneal fibrosis [19]. Perirenal fibrosis that occurs in association with retroperitoneal fibrosis or as part of multifocal fibrosclerosis is not difficult to detect at imaging. However, the imaging features of isolated perirenal fibrosis are nonspecific, and a biopsy may be required to achieve a definitive diagnosis.

**Renal replacement lipomatosis**

Renal replacement lipomatosis (RRL) is a rare reactive pathological entity that is characterized by focal or extensive fat tissue proliferation and renal parenchymal atrophy. It is usually associated with chronic inflammation and calculi. Large hyperplastic fat cells in the renal sinus are diagnostic of RRL. Fat tissue proliferation may be localized in renal sinus, renal hilus and in perirenal and periureteral spaces with severe atrophy of renal parenchyma and enlargement of the kidney. It is usually associated with chronic inflammation, calculi (75%) or XGP [20]. Association with renal tuberculosis was also reported [21]. Plain radiograph characteristically demonstrates a staghorn calculus and, sometimes, a lucent mass.
US reveals an enlarged kidney outlined by a thin hypoechoic rim corresponding to the residual renal parenchyma, stones with posterior acoustic shadowing and sometimes the extension of the echogenicity of the renal sinus to renal parenchyma [22]. Both XGP and RRL generally coexist and a complete distinction between these two entities is possible only by histological features.

Replacement lipomatosis of the kidney is characterized by extensive renal sinus lipomatosis with parenchymal atrophy. It is usually associated with chronic inflammation and with calculi. In addition to renal sinus, replacement lipomatosis may involve renal hilus and perirenal and sometimes periureteral spaces. Plain radiograph characteristically demonstrates a staghorn calculus and, sometimes, a lucent mass. IVU demonstrates a poorly functioning or nonfunctioning kidney, while US shows an enlarged kidney, outlined by a thin hypoechoic rim (the residual renal parenchyma), stones and the extension of the highly echogenic appearance of the sinus to the renal parenchyma. CT demonstrates stones and diffuse fatty replacement of parenchyma which is reduced to a thin rim of renal cortex (Figure 28 on page 35; Figure 29 on page 36) Differential diagnosis between replacement lipomatosis and XGP can be difficult. However in XGP the low attenuation material filling the dilated calyces typically ranges between -15 and +30 HU, while in replacement lipomatosis an attenuation of pure fatty tissue (between -60 and -100 HU) is observed. Sometimes the two diseases coexist.

Renal malacoplakia

Renal malacoplakia is a rare granulomatous infection [23] which occurs because of abnormal monocyte function with accumulation of bacteria incompletely destroyed forming the Michaelis - Guttman bodies [5, 24]. This pathologic entity is attributed to chronic renal infection by Gram negative bacteria, and most frequently E. Coli. Most frequently it involves the bladder, but it can be observed also in the kidney. There are two forms, the multifocal and the unifocal. The multifocal form consists in the diffuse enlargement of the kidney with evidence of intrarenal well-delimited yellowish masses with hemorrhagic and necrotic components. The unifocal form appears as a single mass with the same features of the multifocal form. Renal malacoplakia is also a rare cause of acute renal failure [25] and obstructive nephropathy due to ureteral and bladder involvement [26].

Excretory intravenous urography usually shows a large smooth kidney without hydronephrosis and often non visualization of collecting system. Sometimes small filling defects in the pelvis, ureters and bladder are detected.

US shows a large kidney with poorly defined masses of variable echogenicity. The central echo complex in the sinus is distorted and compressed. CT also shows a renal enlargement with hypodense solid masses (Figure 30 on page 36) which do not enhance, often with involvement of the renal excretory tract which appears compressed or infiltrated. Perinephric extension can be detected. Because of its rarity,
renal malacoplakia is usually not considered preoperatively, but the diagnosis could be suggested when above patterns are shown in a patient with urinary tract infection by *E. Coli*, especially with a known focus of non renal malacoplakia.

**Cholesteatoma**

Long-standing urinary tract infection (particularly with TB) can cause squamous metaplasia of the collecting system [27]. Desquamation of these epithelial cells forms an intraluminal collection of keratin, which is called a cholesteatoma. The nonspecific symptoms include dysuria, hematuria, and colic. On urography or retrograde pyelography a filling defect is seen with a characteristic laminated appearance caused by contrast material entering interstices in the mass. Calcification can occur, which may in part account for the fact that on CT the attenuation is higher than the usual soft-tissue filling defect (Figure 31 on page 37; Figure 32 on page 37). This is not a premalignant condition. Nevertheless, there may be progressive destruction and rapid reaccumulation of debris if the lesion is merely extracted.

**Chronic renal infections in AIDS patients**

The urinary tract is relatively spared from the effects of AIDS (urological symptoms occur in 16 % of all patients) and chronic infections are definitely unusual. In *Pneumocystis carinii, Citomegalovirus* and *Mycobacterium avium intracellulare* acquired immunodeficiency syndrome (AIDS)-correlated disseminated infections, US may reveal diffuse echogenic spots on renal cortex which correspond to punctate calcifications [28]. *Mycobacterium tuberculosis* may also involve the kidney in AIDS patients manifesting as renal abscesses on US, CT, and MR imaging and pyonephrosis in the acute setting. *Mycobacterium avium intracellulare* asymmetrically involved the renal cortex and medulla [29].

Disseminated infection by *Candida albicans* determine focal microabscesses in the liver, spleen, pancreas and kidneys, which appear as multiple small hypoechoic lesions on US. *Pneumocystis jirovecii* is a fungus that is most commonly associated with the AIDS-defining illness *Pneumocystis carinii* pneumonia [30]. The renal infection from *Pneumocystis jirovecii* include atypical cortical nephrocalcinosis [31].

In AIDS patients, *Mucormycosis* usually manifests as disseminated or focal invasion of renal parenchyma (Figure 33 on page 38) and of renal parenchyma vessels, with cortical infarcts and medullary necrosis. Diffuse calcifications are also visualized by CT and don't necessarily mean healed, inactive disease.
Images for this section:
Fig. 1: Figure 1 Chronic pyelonephritis. Typical calyceal distortion due to the underlying renal parenchyma damage and scarring.
Fig. 2: Figure 2a, b. Chronic pyelonephritis. Multidetector CT urography. Coronal reformations. (a) Right kidney. Calyceal distortion and clubbing (arrow) due to the underlying renal parenchyma damage with renal parenchyma scarring and focal reduction of renal parenchyma thickness. (b) Left kidney. Diffuse reduction of the renal parenchyma thickness with caliceal distortion (arrow).
Fig. 3: Figure 3. The early morphologic alterations of renal parenchyma determined by tuberculosis. The fundamental calyceal alterations in renal tuberculosis: calyceal erosion (a), medullary necrosis (b), papillary necrosis (c), and infundibular stricture without (d) or with hydrocalyx (e).
Fig. 4: Figure 4. Renal tuberculosis. CT urography shows also the existence of renal medullary necrosis in one of the renal calices of the lower group.
Fig. 5: Figure 5. Renal tuberculosis. Intravenous excretory urography. Tubercular stricture (small arrow) with evidence of hydrocalyx (large arrow) and adjacent calyceal erosion (long arrow).

Fig. 6: Figure 6a, b. Renal tuberculosis. CT urography. Transverse scan. (a) Infundibular stricture (arrow) due to fibrosclerosing tuberculosis with narrowing of the infundibulum. (b) Dilatation of an upper renal calyx (hydrocalyx).
**Fig. 7:** Figure 7. Renal tuberculosis. CT urography. Transverse scan. Infundibular stricture (arrow) due to fibrosclerosing tuberculosis with narrowing of the infundibulum and phantom calyx.

**Fig. 8:** Figure 8a - e. Renal tuberculosis. (a, b) Ultrasound scan, longitudinal view. Segmental dilatation of the upper urinary tract (arrow) with focal calcification within the renal parenchyma (calipers) and retraction of the renal profile. Unenhanced (c) and contrast-enhanced CT (d) show the renal parenchyma calcifications (arrow) with segmental dilatation of the intrarenal urinary tract. (e) CT urography, maximum intensity projection. Segmental dilatation of the upper urinary tract (arrow) due to fibrosclerosing tuberculosis with tuberculous stricture at the superior infundibulum with regional hydrocalycosis (arrow).
Fig. 9: Figure 9. 63-years old patients with renal tuberculosis. Contrast-enhanced CT. Fibrosclerosis of the ureteropelvic junction with consequent markedly dilated calices and renal parenchyma thinning.
Fig. 10: Figure 10. Renal tuberculosis with caseified tissue necrosis. Open forms. The extensive cavitation of renal parenchyma results in non-communicating (a) or
communicating cavities (b) with the intra-renal excretory tract with deformation of the adjacent renal calices from the simple narrowing of the calyx, medullary and papillary necrosis, up to obstructive hydronephrosis.

**Fig. 11:** Figure 11a - c. Evolution of the open forms of renal tuberculosis. The caseified tissue necrosis (a) progressively spreads to the renal urinary tract (b). The progressive fibrosclerosis results in a non-communicating cavity (c).

**Fig. 12:** Figure 12a, b. (a) CT urography, coronal reformation. (b) Maximum intentensity projection. A parenchymal cavity communicating with the renal excretory tract is visualized on the right kidney.
**Fig. 13:** Figure 13a - d. Diffuse cavitating open form of renal tuberculosis. (a-d) Contrast-enhanced CT during the excretory phase. The left kidney shows diffuse parenchymal cavitation with resulting communicating (arrows) and non-communicating cavities (caliper).
Fig. 14: Figure 14. Renal tuberculosis. CT urography. Coronal reformation of the right kidney. Fibrosclerosing tuberculosis with gross calcification (arrow) at the level of the renal parenchyma and adjacent non-communicating cavity.
**Fig. 15:** Figure 15. Open form of renal tuberculosis with diffusion into the intrarenal collecting system extensive cavitation and fibrosclerosis with resulting non-communicating cavities. Contrast-enhanced CT during the excretory phase. Extension of the caseified tissue necrosis to the intra-renal excretory tract with parenchymal fibrosclerosis with calcifications and irregularities of the renal margins (arrow) and resulting non-communicating cavities.
Fig. 16: Figure 16. Open form with diffusion into the intrarenal collecting system extensive cavitation and fibrosclerosis with resulting non-communicating cavities. Contrast-enhanced CT during the excretory phase. Renal parenchyma extensive necrosis with non-communicating cavities (arrows) in both kidneys, and parenchymal fibrosclerosis with calcifications and irregularities of the renal margins.
Fig. 17: Fig. 17. (a) Scheme. Closed form of renal tuberculosis. The pathologic process extends towards the renal parenchyma with progressive fibrosclerosis (arrow)
and distortion of the adjacent renal calices. (b) CT-uography. Typical parenchyma fibrosclerosis (large arrow) with adjacent gross parenchymal calcification (small arrow).

Fig. 18: Figure 18a, b. Closed form of renal tuberculosis. Contrast-enhanced CT during the excretory phase. The right kidney shows extensive parenchymal fibrosclerosis (large
arrows) with scar, dilatation of the intra-renal excretory tract (small arrows), and loss of excretory function with autonephrectomy (no contrast excretion is evident).  

**Fig. 19:** Figure 19a - d. Closed form of renal tuberculosis. (a) Intravenous urography. Dilatation of the upper (arrow) and lower urinary tract with no evidence of contrast excretion in the left kidney. (b - d) Contrast-enhanced CT during the excretory phase. (b) The right kidney shows parenchymal fibrosclerosis with clear irregularities of the renal parenchyma margins, calyceal deformation and dilatation (large arrow) due to the renal parenchyma sclerosis and one non-communicating cavity (small arrow). (c, d) The left kidney shows extensive fibrosclerosis with parenchymal scar, necrosis with cavitation and multiple non-communicating cavities due to reactivation of the caseating process over fibrosclerosis.
Fig. 20: Figure 20a-d. Closed fibrosclerotic form of renal tuberculosis. (a) Intravenous excretory urography. Tubercular infundibular stricture with phantom calyx (arrow). (b) Ultrasound, longitudinal scan. Evidence of an hypoechoic lesion (arrow) in the upper pole of the kidney within the renal parenchyma corresponding to a cavitation. (c, d) Contrast-enhanced CT during the excretory phase. (c) The same cavitation (arrow) appears non-communicating with parenchymal thinning. (d) Coexisting communicating cavitation (arrow) is also visualized.
Fig. 21: Fig. 21a, b. Putty kidney. Extensive cavitation determines diffuse renal parenchyma caseation and calcification. (a) Scheme; (b, c) anatomical macroscopic specimen.
**Fig. 22:** Figure 22. Putty kidney. Unenhanced CT, coronal reformations. The entire right kidney (arrow) appears small, scarred, and densely calcified. A calcified and thick material fills a dilated collecting system.

**Fig. 23:** Figure 23a - d. Diffuse xanthogranulomatous pyelonephritis confined to the kidney. CT shows multiple low-density non-enhancing areas surrounded by enhancing rims corresponding to intrarenal collections, and renal stone without involvement of perinephric region and Gerota's fascia.
**Fig. 24:** Figure 24a, b. Diffuse xanthogranulomatous pyelonephritis with extrarenal diffusion. CT shows multiple low-density non-enhancing areas surrounded by enhancing rims corresponding to intrarenal collections, and renal stone with involvement of perinephric region, Gerota's fascia, and pararenal posterior space of the retroperitoneum.

![Figure 24a](https://example.com/image1) ![Figure 24b](https://example.com/image2)

**Fig. 25:** Figure 25a, b. Diffuse xanthogranulomatous pyelonephritis with extrarenal diffusion and gas component. CT shows multiple low-density non-enhancing areas surrounded by enhancing rims corresponding to intrarenal collections, and renal stone with involvement of perinephric region, Gerota's fascia, and pararenal posterior space of the retroperitoneum. A gas component is evident within the renal collection.

![Figure 25a](https://example.com/image3) ![Figure 25b](https://example.com/image4)

**Fig. 26:** Figure 26a, b. Focal xanthogranulomatous pyelonephritis. Axial contrast-enhanced CT during the excretory phase. The focal form of xanthogranulomatous
pyelonephritis on the left kidney. The focal (tumefactive) form manifests as a focal renal mass with extension to the perinephric space.

**Fig. 27:** Figure 27a, b. Xanthogranulomatous fibrotic process of the perirenal space. Perirenal fibrosis that occurs in association with retroperitoneal fibrosis. Axial contrast-enhanced CT obtained during the corticomedullary phase depicts a rindlike soft-tissue layer surrounding both kidneys (arrows).

**Fig. 28:** Figure 28. Renal replacement lipomatosis of the right kidney with xanthogranulomatous pyelonephritis. (a) Gray-scale ultrasound, longitudinal scan. Renal enlargement (arrow) with heterogeneous appearance. (b) Plain radiograph. Extensive radiolucency in the right renal pelvis (arrows) with staghorn calculosis. (c) Contrast-enhanced CT, excretory phase. Extensive renal sinus lipomatosis with parenchymal atrophy and renal staghorn stones with renal hilus involvement. The renal parenchyma which is reduced to a thin rim of renal cortex. The replacement lipomatosis (arrow) invades the perirenal space, the anterior pararenal space, and the posterior pararenal.
space (small arrow). (d) Gross specimen. Diffuse fatty infiltration of the perirenal space and renal sinus with compression of the renal parenchyma (arrows).

**Fig. 29:** Figure 29. Renal replacement lipomatosis of the left kidney with xanthogranulomatous pyelonephritis. Replacement lipomatosis of the kidney is characterized by extensive renal sinus lipomatosis with parenchymal atrophy and renal staghorn stones with renal hilus and perirenal involvement. CT demonstrates stones and diffuse fatty replacement of parenchyma which is reduced to a thin rim of renal cortex.
**Fig. 30:** Figure 30a - c. Malacoplachia of the left kidney. (a) US shows focal renal mass (arrows). (b, c) CT shows a renal enlargement with hypodense solid masses with do not enhance, often with involvement of the renal excretory tract which appears compressed or infiltrated.

**Fig. 31:** Figure 31a, b. Cholesteatoma. Unenhanced (a) and contrast-enhanced CT (b). CT the attenuation is higher than the usual soft-tissue filling defect.
Fig. 32: Figure 32a, b. Cholesteatoma. CT urography, coronal reformations. (a) Diffuse thickening of the renal pelvis wall (arrow) is visualized in a patient with long-standing urinary tract infection due to reflux nephropathy depicted by renal parenchyma scar and calyceal distortion with a claviform shape; (b) a filling defect with a laminated shape (arrow) is evident within the renal pelvis corresponding to renal cholesteatoma.
**Fig. 33:** Figure 33. Renal mucormycosis in AIDS. Contrast-enhanced CT. Transverse plane. Involvement of the right kidney (arrow) which appears dimensionally increased and with heterogeneous density due to patchy areas also with diffusion to the perirenal space.
Conclusion

Chronic renal infections present some typical imaging findings which should be recognized to differentiate chronic renal infections from other renal pathologies.

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