

Artigos de Revisão

Abcessos Renais - Revisão

António Murinello¹, Paula Mendonça¹, Ricardo Correia², Rocha Mendes²,
Margarida Albergaria³, Jorge Neta⁴, João Coelho¹, Sofia Lourenço¹

1. Unit of Internal Medicine I

2. Unit of Urology

3. Unit of Radiology

4. Unit of Histopathology

Curry Cabral Hospital. Lisbon. Portugal

Resumo

Baseados num caso de associação de abcessos renal, perirrenal e de quisto renal infectado, ocorrendo após biópsia prostática, os autores fazem revisão bibliográfica dos abcessos renais, classificados segundo características anatómicas – perinéfrico, cortical e cortico-medular (nefrite bacteriana aguda focal/multifocal, pielonefrite enfisematosa e pielonefrite xantogranulomatosa). Os abcessos renais são mais frequentes em diabéticos, menos vezes em alcoólicos, desnutridos e imunodeprimidos, estando associados geralmente a pielonefrites recorrentes, refluxo vesicoureteral, litíase urinária, ou resultam de êmbolos sépticos hematogéneos originados em focos extra-renais de infecção. A *Escherichia coli* é responsável pela maioria dos casos, e menos vezes a *Klebsiella*, *Proteus*, *Enterobacter*, *Pseudomonas* e *Staphylococcus aureus*. Descrevem-se a expressão clínica, complicações evolutivas e diagnóstico radiológico, enfatizando-se as infecções acompanhadas de formação de gás. Referem-se opções terapêuticas médico-cirúrgicas, cujas indicações dependem do diagnóstico precoce, complicações e factores predisponentes.

Palavras chave: Abcesso renal / Abcesso perinéfrico / Abcesso de quisto renal

Abstract

Based on a case of associated renal, perinephric and renal cyst abscesses, occurring after prostatic biopsy, the authors do a concise review of renal abscesses, classified by anatomic categories – perinephric, cortical and corticomedullary (acute focal/multifocal bacterial nephritis, emphysematous and xantogranulomatous pyelonephritis). Renal abscesses are frequent in diabetics, less so in alcoholics, undernourished and immunosuppressed patients, and commonly associated with recurrent pyelonephritis, vesicoureteral reflux, urinary lithiasis, or resulting of hematogenous septic emboli from extra-renal sources of infection. *Escherichia coli* is responsible for most cases, other agents are *Klebsiella*, *Proteus*, *Enterobacter*, *Pseudomonas*, *Staphylococcus aureus*. Clinical presentation, complications and imaging are considered, emphasizing gas-forming infections. Indications for medical and surgical therapeutic options depend on early diagnosis, complications and predisposing factors.

Key words: Renal abscess / Perinephric abscess / Abscess of renal cyst

Correspondência:

António Murinello
Av. Eng. António Azevedo
Coutinho, Lt. 8 r/c – dto.
2750-644 CASCAIS
Portugal
E-mail: amurinello@iol.pt
Tel. (351) 918 626 874

Introduction

Renal abscesses are considered a rarity amongst intra-abdominal abscesses^{1,2,3}. Symptomatology can be varied and misleading, their course frequently insidious for a time, with frequent elusive diagnosis, causing significant degree of morbidity and mortality. Renal abscesses are classified by anatomic categories – perinephric, cortical and corticomedullary abscesses². **Perinephric abscesses** usually are the result of rupture of renal abscess or infected hydronephrosis into the perirenal space, or more rarely from hematogenous seeding of septic emboli from extra-renal sources of infection. **Renal corticomedullary abscesses** occurs in confluent areas of the renal cortex and medulla, commonly as sequela of severe acute pyelonephritis, complication of vesicoureteral reflux, ureteral and pelvic calculi, being more frequent in diabetics⁴. **Renal cortical abscess (carbuncle)** results of hematogenous seeding of septic emboli from anywhere, being common in intravenous drug users, immunosuppression and patients with an extra-renal source of infection (endocarditis, perinasal sinusitis, skin or dental infections). **Infection of renal cysts** are rare as a cause of renal abscess, but infection of renal cysts is a possible etiology for a renal abscess^{5,6}. The authors present a patient with long history of bladder calculi and recurrent urinary infections, who after transrectal prostatic biopsy developed a clinical picture of serious urinary sepsis due to concomitant renal corticomedullary, perinephric and renal cyst abscesses. Surgery also showed pyonephrosis associated with almost obstructive pyeloureteral calculi. Cultures of urine and pus of all abscess areas revealed the same infecting microorganism (β -lactamase producing *E. coli*). Partial nephrectomy and post-operative drainage and antibiotherapy were successful.

Clinical Report

A 76 year old white man was admitted to our Unit (**I. Medicine 1**) on **22 JUL 06** due to undiagnosed total hematuria. He had a long history of hypertension, atrial fibrillation, and chronic liver disease due to heavy alcohol consumption until 10 years before. Urologic history began with a renal colic in 1980. From two years ago he had several episodes of painless hematuria, attributed to urinary stones. On **FEB 05** ultrasonography (US) showed: (1) cortical cyst in the left kidney lower pole (7cm on long axis) and bilateral renal microlithiasis; (2)

several bladder stones; (3) prostatic hypertrophy (59 gr). From **FEB 06** he had progressive dysuria, stranguria and frequent total hematuria. Blood tests revealed: no anemia, no leukocytosis, normal ionogram, glycemia and creatinine, urea 50 mg/dL (15.0-45.9); prostatic specific antigen (PSA) 10.98 ng/mL (N <4.00). Urinary sediment: 49 leukocytes/ μ L (1-10) and >1000 erythrocytes/ μ L (1-29). Proteinuria 35.00mg/24h. Uroculture was negative. On **JUN 06** an urologist in another Hospital tried an unsuccessful cystoscopy (pain and heavy hematuria). After that traumatic urologic intervention he had left renal colic several days later, being seen on **10 JUL 06** by an urologist in the Ambulatory Unit of our Hospital, who under prophylaxis with co-trimoxazole proceeded to transrectal prostatic biopsy, which revealed chronic prostatitis and no malignant tissue. Three days after prostatic biopsy the patient began to feel unwell, with nausea, anorexia, asthenia, and strong pain on the left lumbar region, with temporary amelioration with analgesics. Suddenly on the 10th day after prostatic biopsy (**20 JUL 06**), he had intense shivering and worsening of lumbar pain with irradiation to the right lumbar region and to the anterior abdominal wall, being assisted in the Emergency Unit of our Hospital. He had no fever and abdominal examination disclosed diffuse abdominal tenderness. There was no urinary retention. Urinary sediment had many leukocytes and erythrocytes and nitrituria. Blood tests showed: no anemia, no leukocytosis 7.700x10⁹/L (with 87.8% neutrophils); platelets 64.000x10⁹/L (N 130.000-400.000); I.N.R. 1.2 (0.90-1.20); APTT 46" (26.0-36.0); total/conjugated bilirubin 3.6 mg/2.5 mg/dL (0.20-1.3/0.00-0.30); potassium 3.1 mEq/L (3.60-5.00); creatinine 2.7 mg/dL (0.70-1.20), urea 119 mg/dL; PCR 21 mg/dL (N <1.00). Abdominal plain film was not informative. He was discharged with a diagnosis of renal insufficiency and hypokalemia and treated with ciprofloxacin. Two days later however, the patient came again to the Emergency Unit complaining of intense left lombalgia and total hematuria. Blood tests revealed: no anemia; leukocytes 6.800x10⁹/L (with 90.3% of neutrophils); platelets 34.000x10⁹/L; glycemia 160 mg/dL; creatinine 2.5 mg/dL; urea 126.2 mg/dL; potassium 3.2 mEq/L; γ -GT 129 U/L (15-73); alkaline phosphatase 286 U/L (38-126); normal AST/ALT; albumin 2.10 g/L (3.50-5.00); PSA 39.30 ng/mL. Urinary sediment revealed 500 leukocytes/ μ L, many erythrocytes and nitrituria. Abdominal US showed diffusely heterogenous hepatomegaly, gallbladder lithiasis, moderate splenomegaly, bilateral renal microlithiasis, kidneys measuring on long axis 13.3 cm (right) and 14.0 cm (left), this one with slight pyelocali-

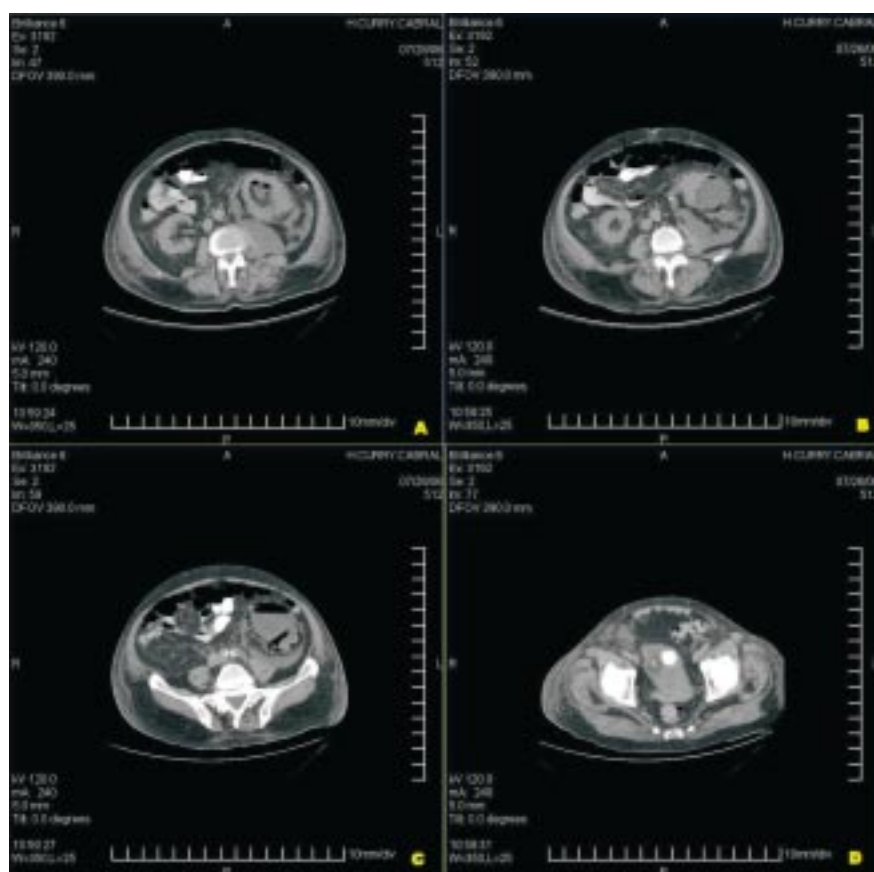


Fig. 1 – Non-contrast enhanced abdominal CT scan: **Fig. 1A:** gas in calyceal topography; suppurative collection in the posterior perirenal space causing anterior dislocation of the kidney and contacting the psoas muscle. **Fig. 1B:** urinary stone with topography in the lumbar left ureter; perirenal fluid collection; gas-forming renal abscess; perirenal fluid collection; cystic formation with non pure content and with internal gas. **Fig. 1C:** similar plane, but better definition of perirenal collection. **Fig. 1D:** voluminous bladder stone.

ceal ectasia and voluminous cortical cyst (8.4 cm of diameter) of the lower pole, with echogenic content at the bottom, attributed to intracystic hemorrhage. The patient was admitted to our Unit on **23 JUL 06** with a provisory diagnosis of hematuria due to thrombocytopenia. The patient appeared ill, was feverish (38.0°C), with atrial fibrillation (heart rate approximately 170/min) and respiratory rate 24/min. There were no signs of liver failure. On the abdomen a painful mass without definite limits and with firm consistency and lumbar contact was palpable at the left flank. There was moderate bilateral edema of the legs, and rectal touch detected slightly painful prostate, diffusely and moderately enlarged. While waiting for uroculture result, the patient was treated with intravenous levofloxacin for three days, without amelioration. As uroculture showed significant colony growth ($> 10^5$ colony-forming units/mL) of β -lactamase producing *E. coli*, only sensible to gentamicin and amoxiclav, the last one was prescribed

due to renal failure. At the same day hemogram revealed hyperleukocytosis ($30.700 \times 10^9/\text{L}$ with 91% neutrophilia) and abdominal non-contrast enhanced CT scan demonstrated a cyst (10x6 cm) on the lower pole of the left kidney with non pure intra-luminal content, with presence of gas pointing to infection with gas-forming bacteria. There was densification of the perirenal fat and also a fluid collection on the posterior perirenal space, contacting the psoas muscle and with a vertical extension measuring 20 cm. Urinary lithiasis was evident in the lumbar portion of the left ureter causing pyelocaliceal ectasia. The presence of gas in the calyces was in favor of a concomitant process of pyelitis. A voluminous calcified stone was shown in the bladder (Figs. 1 A, B, C, D). On the same day surgical catheter was inserted posteriorly in perirenal space, with drainage of abundant pus with cultural growth of *E. coli* with identical antibiotic susceptibility. Anaerobic culture was negative. After clinical stabilization and due to maintenance of sup-

purative collections as seen in CT scan done on **2 AUG 06**, operatory drainage was realized on **4 AUG 06**. We proceeded to drainage of posterior perinephric space as well as of the purulent collection in the renal cyst, the wall of which was resected. The parenchyma of the inferior pole of the left kidney was oedematous and enlarged, due to several areas of suppuration that were in contact with the cyst. Ureteropyelolithotomy to remove voluminous obstructive left ureteral lumbar stone and blood clots and partial nephrectomy of the inferior pole of the left kidney was necessary. Urine was turbid, showing characteristics of pyonephrosis. Post-operatory drainage was necessary for seven days. Indwelling urinary Folley catheter was maintained and the patient was discharged for a period of convalescence, before proceeding to prostatectomy and surgical removal of the bladder, because he was considered not to have medical conditions to support another type of surgery at the same general anesthesia. Oral amoxiclav was maintained until completing six weeks of therapy. One week after surgery blood tests revealed normal values of glycemia (101 mg/dL) and urea (39.2 mg/dL), and total/conjugated bilirubin 1.40/0,50 mg/dL. Meanwhile microscopical examination of the kidney revealed serious lesions of chronic interstitial nephritis with areas of acute pyelonephritis and suppuration in contact with a necrotic area of abscess. There were several cystic areas with structureless lining suggestive of gas spaces, one of which with voluminous hemorrhage and mixed inflammatory infiltrate. Lesions of glomerular and vascular sclerosis were conspicuous. The perirenal fat tissue showed areas of hemorrhage, steatonecrosis and organized panniculitis (Figs. 2 A, B, C, D, E, F, G, H).

Discussion

Perinephric abscess is usually the result of a rupture of a renal abscess or of pyonephrosis to the perirenal space⁷. Usually there are predisposing urologic conditions (renal or ureteral calculi with partial or total obstruction, noncalculous hydronephrosis, polycystic renal disease or infected renal carcinoma). *E. coli* and *Proteus* are the most common etiologic agents, but *K. pneumoniae* and *Ps. aeruginosa* can also be found⁸. Rarely perinephric abscesses are due to septic embolization from an infection elsewhere⁹ or the origin is a rupture of an infected lesion of the colon (inflammatory bowel disease, diverticulitis, infected colon carcinoma)⁷.

Renal corticomedullary abscess is considered a spectrum of disease encompassing various intra-renal

inflammatory processes, which include: **acute focal and multifocal bacterial nephritis, emphysematous and xanthogranulomatous pyelonephritis**. Renal corticomedullary abscess differs from renal cortical abscess because it develops from ascending rather than hematogenous spread of bacteria, and it is usually associated with underlying urinary tract abnormality (eg, vesicoureteral reflux, urinary tract obstruction), recurrent urinary tract infections and infected urinary lithiasis. Bacterial pathogens causing cystitis ascend to the upper urinary tract to infect renal medulla. Subsequent liquefaction of renal parenchyma and involvement of renal cortex is the postulated pathogenesis for development of corticomedullary abscess. Scarring of ureters and of ureteral-pelvic junction may occur, causing obstructive symptoms. Previous genitourinary instrumentation is a frequent initiating event leading to occurrence of corticomedullary abscess⁴.

E. coli are responsible for 75% of infections, 15-20% of cases are caused by *Klebsiella*, *Proteus*, *Enterobacter*, *Serratia* and *Pseudomonas* species, the remaining are due to gram-positive bacteria (*Streptococcus faecalis*, *Staphylococcus aureus*⁴). Obligatory anaerobes are ultimately being referred with greater frequency, isolated or in association with other gram-negative microbial agents, as responsible for genito-urinary suppurations^{10,11} (*Bacteroides fragilis*, pigmented *Prevotella* and *Porphyromonas* sp., anaerobic Gram-positive cocci and *Actinomyces* sp.) However, because of inconsistent use of adequate methods for their isolation and identification and also because of their fastidious nature with slow growth and also polymicrobial nature, anaerobic infection remains many times undiagnosed.

Gram-negative facultative anaerobes or obligatory anaerobes may produce gas by fermenting glucose in necrotic tissues¹². Facultative Gram-negative anaerobes include *E. coli*, *Klebsiella*, *Proteus mirabilis*, *Citrobacter* and yeasts. Patients with renal infections by these microorganisms are generally critically ill and their urologic problems compounded by serious medical problems, such as electrolyte imbalance, dehydration, acidosis, hyperglycemia. Diabetics have a high incidence of bacteriuria and pyelonephritis, as the hyperglycemia and ketoacidosis impairs host cellular defenses and protects bacteria from the bactericidal activity of lactic acid. Diabetes also predisposes to intrinsic vascular disease leading to papillary necrosis and renal infections, thereby reducing tissue absorption of gases produced by anaerobic bacteria and resulting in gas-forming (pneumaturia) pyelonephritis^{13,14}.

Acute focal bacterial nephritis is a well-localized inflammation of the kidney without frank abscess forma-

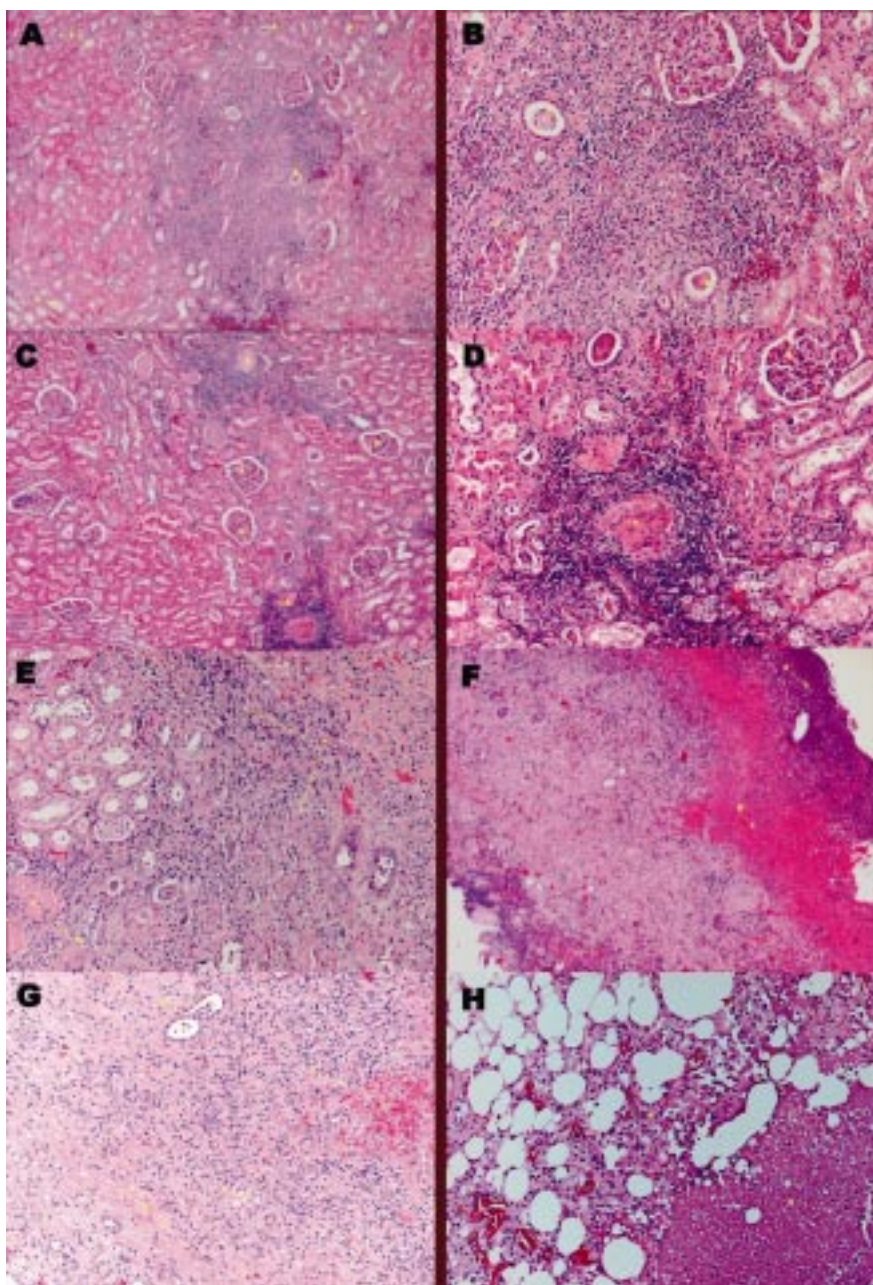


Fig. 2 – Histopathology: **Fig. 2 A:** HE (4x10): lower pole of left kidney – dilated and atrophic remnant tubules (A), some of it containing eosinophilic proteinaceous material (B). **Fig. 2 B:** HE (40x10): lower pole of left kidney – dilated and atrophic remnant tubules, some of it structureless, without epithelial lining, suggesting gas spaces (A), others with eosinophilic proteinaceous material in the lumen (thyroidization) (B), others with suppurative neutrophilic masses (C). **Fig. 2 C:** HE (10x10): lower pole of left kidney – hyalinized glomerules (A) and related areas of necrotic tubules, contrasting with other areas of preserved glomerulus (B). **Fig. 2 D:** HE (40x10): lower pole of left kidney – a higher amplification of the same aspects described on Fig 2 C. **Fig. 2 E:** HE (40x10): lower pole of left kidney – areas of tubular progressive invasion and substitution by chronic inflammatory reactive cells (A), with posterior deposition of collagen (B) in the cicatrization process. **Fig. 2 F:** HE (4x10): capsule of renal abscess - with areas of necrosis (A) and hemorrhage (B). **Fig. 2 G:** HE (40x10): capsule of the abscess – extensive inflammation of renal interstitium, partially occupied by fibrosis (A), with dilation (B) and obliteration (C) of renal tubules. Dilated tubules had epithelial lining cells and inflammatory cells in the lumen. Several areas of interstitial hemorrhage (D). **Fig. 2 H:** HE (40x10): perirenal abscess – perirenal fat with zones of panniculitis (A) and steatonecrosis (B)

tion and frequently progresses to the more severe **acute multifocal bacterial nephritis**, in which case there is heavy polymorphonuclear infiltrate through the kidney with areas of liquefaction and abscess formation.

Emphysematous pyelonephritis (EPN) is an uncommon severe necrotizing form of acute multifocal bacterial nephritis, which on abdominal radiography exhibits characteristic intraparenchymal (so-called “gas-gangrene of the kidney”) or perirenal gas and not, by definition, isolated within collecting system. The presence of gas suggests the presence of gas-forming anaerobic or facultative anaerobic pathogens. Although *E. coli* is the most common microorganism associated with EPN, any lactose-fermenting organism may be involved. Patients with severe EPN often present in *extremis* and require intensive medical treatment. The function of the affected kidney is often very poor. The disease occurs almost exclusively in patients with diabetes mellitus, with overall mortality of 45%. Occasionally it is seen in patients without diabetes mellitus but with obstruction of the renoureteral unit. Among diabetics with EPN, obstruction of affected kidney is seen in about 30% of the patients. It has also been reported in debilitated alcoholics and the severely immunocompromised¹⁵. Pathogenesis of EPN involves four factors: gas-forming microorganisms, high tissue glucose levels in the majority of cases, impaired tissue perfusion and defective immune response. Urinary obstruction aggravates these factors due to increase in pyelocalyceal pressure and compromised renal circulation. Pathological findings often include evidence of diabetic glomerulopathy, impaired tissue perfusion in the form of vascular thrombosis and infarction, glomerulosclerosis and papillary necrosis¹⁵. Renal interstitium commonly show diffuse infiltrative processes by acute suppurative and chronic inflammatory infiltrate forming multiple abscesses and large areas of infarction and multiple cysts in the necrotic areas, the lining of which are structureless thus suggesting gas spaces¹⁵.

Xantogranulomatous pyelonephritis (XGP) is a chronic long-term infection of a renal unit often associated with a renal calculus. As a result of long-standing infection, the kidney enlarges and it fixes itself to the retroperitoneum by peritoneal fibrosis and extension of the granulomatous inflammation. Native tissue planes, such as the plane between *Gerota fascia*, adjacent retroperitoneal structures and peritoneum, are disrupted. Histologically, granulomatous tissue containing lipid-laden macrophages (ie, foam cells) destroys and replaces renal parenchyma. Renal calculi are usually present in 75% of patients with XGP (50% are staghorn calculi).

Proteus mirabilis is the most common bacteria associated with XGP, but *E. coli* is also common. The histopathological process may evolve from an area of renal involvement to widespread extension into the retroperitoneum⁴.

Renal cortical abscess is a multilocular abscess involving the renal parenchyma as a result of coalescence of cortical microabscesses. Although *Staphylococcus epidermidis* can be a causative agent, *Staphylococcus aureus* is by far the commonest pathogen, spreading to the kidney through hematogenous dissemination from a focus elsewhere. The primary focus can be a carbuncle, a furuncle, a suppurative respiratory infection, or a cellulitis. Intravenous drug users are commonly affected by these infections as well as diabetics. Renal lesions usually coalesce to form a fluid-filled mass with a thick wall. The interval from the original staphylococcal infection and the onset of symptoms may be as long as several months, and the patient may have forgotten the primary focus of infection unless questioned closely. Other less commonly etiologic agents are *E. coli* and *Proteus*, and much more rarely *Brucella*¹⁶ and *Aspergillus*¹⁷. Urinary symptoms are commonly absent in these types of abscess, as well as urinalysis abnormalities.

Occasionally renal abscesses can be complicated by extension to nearby structures. Besides the rupture to the perirenal space of renal corticomedullary and cortical abscesses, extension of the infectious process can also cause peritonitis, subphrenic abscess, pleural empyema¹⁸, fistulous communication to skin, spleen, colon, pericardium¹⁹ or bronchus²⁰, sequelae calyceal diverticulum due to rupture into the collecting system²¹, papillary necrosis, loss of renal function, massive retroperitoneal hemorrhage due to abscess erosion into the renal vasculature²², psoas abscess²³, extension to the pelvis and groin, and epidural abscess²⁴. Bacteraemia due to these abscesses can result in spread of infection to other sites in the body.

Clinically renal abscesses can present as acute pyelonephritis, subsequently failing to respond to appropriate antibiotherapy. Alternatively, patients may have insidious onset of symptoms with absence of localizing signs such as flank pain, pyuria or bacteriuria, which most commonly occurs in patients with diabetes mellitus, alcohol or drug abusers, immunocompromised patients, and steroid therapy in patients with staghorn calculi. Generally clinical features of renal abscesses usually include fever, chills, and flank or abdominal pain. Dysuria and other urinary tract symptoms are variably present. Nonspecific constitutional symptoms (malaise, fatigue, weight loss) are common, as well as gastroin-

testinal symptoms (nausea, vomiting). Signs of renal abscesses vary greatly and have no specific characteristics to aid in diagnosis. Most patients with renal infection appear ill and in distress, febrile, and may have signs of haemodynamic instability with tachycardia and hypotension and a clinical picture of sepsis. A palpable flank mass is not consistently found, but costovertebral angle tenderness is usually detected⁴.

Most patients demonstrate leukocytosis with left shift. Urinalysis generally show signs of infection, but bacteriuria and pyuria may be absent if the ureter is completely obstructed and in cortical and perinephric abscess caused by bacteriemic emboli. Uroculture is frequently positive in corticomedullary and perinephric abscesses, but not in cortical abscess. Blood cultures are positive in more than 50% of the patients with renal abscesses, and are particularly useful in patients with urosepsis.

Plain X-ray of the abdomen has a sensitivity of only 33% to diagnosis the presence of gas in renal parenchyma. Renal abscesses can be evaluated with US, CT scan or MRI; however, CT is the imaging of choice. On US the abscess can appear similar to a cyst, but with some internal echoes and/or wall irregularity; as a solid mass mimicking a renal neoplasm; or the echo pattern of an abscess may be indistinguishable from adjacent renal parenchyma. US has no sensitivity to diagnose gas in the kidney. On CT, the abscess appears as a heterogeneous low-attenuation mass. There is often an irregular, enhancing wall secondary to hyperaemia or granulation tissue formation. A bulge in the renal cortex is typically present in peripheral abscesses. Inflammatory changes are seen in the adjacent fat. CT is the best imaging method to diagnose gas in the kidney. CT scans provide excellent anatomic details of renal or perirenal abscess collections to be surgically explored. Anaerobic metabolism of a microorganism produces insoluble gases, such as hydrogen, nitrogen and oxygen. Gases are produced as a result of fermentation, denitrification and deamination by a microorganism. Gases in renal parenchyma and perirenal space are well visualized in CT scan¹². On contrast-enhanced MRI, a liquefied portion of the abscess and enhancing wall are suggestive of the diagnosis²⁵.

The first line of treatment of renal abscesses includes correction of hyperglycemia, dehydration or electrolyte imbalance. Appropriate antibiotics against the most likely pathogens are given intravenously after obtaining blood and urine cultures, and changed accordingly to antibiogram susceptibilities. Appropriate antibiotics for use in treatment of infected renal cysts or

abscesses are agents that diffuse well into these closed sites, such as trimethoprim-sulfamethoxazole and fluoroquinolones. When the patient's general condition improves, the decision is then made either to wait the effect of antibiotherapy, to incise and drain the abscess percutaneously or to proceed to partial or total nephrectomy. When renal abscesses are yet small due to an early diagnosis, the preferred treatment would be image-guided percutaneous drainage, often performed under CT guidance. Additional microorganisms may be isolated at the time of drainage, in which case directed antibiotherapy may be necessary. Early diagnosis and aggressive medical antibiotherapy and surgical management of gas-forming infections of the genitourinary tract are vital¹².

Incision and drainage of a renal abscess is performed when the patient's general situation is too serious to attempt a nephrectomy, to preserve as much as renal tissue as possible, particularly in those patients with marginal renal function or when the renal parenchyma is only partially involved in the abscess process. Should the decision be made to incise and drain a renal abscess, all intra-renal loculi and abscess cavities must be adequately drained and also any complicating subphrenic abscess. A nephrectomy is performed when the renal parenchyma was replaced by an abscess cavity or when there is insufficient parenchyma worth conserving. Early diagnosis of EPN is necessary to try to avoid the necessity of a nephrectomy, which frequently offers the best outcome. Diabetic patients are easier to control medically following nephrectomy or incision drainage of a renal abscess. It is also important to consider the additional therapy of underlying disease (e.g., obstructive uropathy)¹⁵.

Although most reported patients had an associated urological abnormality, such as stones, obstruction or anatomic abnormalities, an interesting series by Tung Shu²⁶ refers to the possibility to find this type of abscesses in a group of patients with otherwise anatomically normal urinary tracts, and that with accurate diagnosis and minimally invasive therapy had excellent functional and anatomical outcomes.

Patients undergoing transrectal ultrasound-guided biopsies of the prostate are somewhat prone to bacteriuria and bacteraemia, despite partial protection by antibiotic prophylaxis²⁷. There are several predisposing factors favoring the occurrence of post-biopsy infections, namely former urinary tract infection, prostatitis, urinary catheterization, and diabetes mellitus^{27,28}. Our patient had a long history of previous urinary tract infections and urinary stones, and was submitted to

prostatic biopsy just preceding the development of the renal abscess, with rupture to perinephric space and a renal abscess in an old known renal cyst. It is however speculative to attribute the development of the process to prostatic biopsy, because the patient had strong reasons to develop a renal abscess (recurrent urinary tract infections, bladder stone, chronic prostatitis, ureteral stone with almost complete obstruction, old known voluminous renal cyst, chronic liver disease and impaired glucose regulation). Fortunately it was possible to preserve two thirds of the affected kidney at surgery and to adequately drain all suppurative collections, with recovery to normal values of blood urea and creatinine.

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