The concern of renal specialists for urinary tract infections (UTIs) had declined with the passage of time. This trend is now being reversed, owing to new imaging techniques and to substantial progress in the understanding of host-parasite relationships, of mechanisms of bacterial uropathogenicity, and of the inflammatory reaction that contributes to renal lesions and scarring.

UTIs account for more than 7 million visits to physicians’ offices and well over 1 million hospital admissions in the United States annually [1]. French epidemiologic studies evaluated its annual incidence at 53,000 diagnoses per million persons per year, which represents 1.05% to 2.10% of the activity of general practitioners. In the United States, the annual number of diagnoses of pyelonephritis in females was estimated to be 250,000 [2].

The incidence of UTI is higher among females, in whom it commonly occurs in an anatomically normal urinary tract. Conversely, in males and children, UTI generally reveals a urinary tract lesion that must be identified by imaging and must be treated to suppress the cause of infection and prevent recurrence. UTI can be restricted to the bladder (essentially in females) with only superficial mucosal involvement, or it can involve a solid organ (the kidneys in both genders, the prostate in males). Clinical signs and symptoms, hazards, imaging, and treatment of various types of UTIs differ. In addition, the patient's background helps to further categorize UTIs according to age, type of urinary tract lesion(s), and occurrence in immunocompromised patients, especially with diabetes or pregnancy. Such various forms of UTI explain the wide spectrum of treatment modalities, which range from ambulatory, single-dose antibiotic treatment of simple cystitis in young females, to rescue nephrectomy for pyonephrosis in a diabetic with septic shock. This chapter categorizes the various forms of UTI, describes progress in diagnostic imaging and treatment, and discusses recent data on bacteriology and immunology.
**7.2 Tubulointerstitial Disease**

## Diagnosis

Urine test strips. Normal urine is sterile, but suprapubic aspiration of the bladder, which is by no means a routine procedure, would be the only way of proving it. Urinary tract infection (UTI) cannot be identified simply by the presence of bacteria in a voided specimen, as micturition flushes saprophytic urethral organisms along with the urine. Thus a certain number of colony-forming units of uropathogens are to be expected in the urine sample. Midstream collection is the most common method of urine sampling used in adults. When urine cannot be studied without delay, it must be stored at 4°C until it is sent to the bacteriology laboratory. The urine test strip is the easiest means of diagnosing UTI qualitatively. This test detects leukocytes and nitrites. Simultaneous detection of the two is highly suggestive of UTI. This test is 95% sensitive and 75% specific, and its negative predictive value is close to 96% [3]. The test does not, however, detect such bacteria as *Staphylococcus saprophyticus*, a strain responsible for some 3% to 7% of UTIs. Thus, treating UTI solely on the basis of test strip risks failure in about 15% of simple community-acquired infections and a much larger proportion of UTIs acquired in a hospital. immersed in the urine, shaken, and incubated overnight.

The most specific results, however, are provided by laboratory analysis, which allows precise counting of bacteria and leukocytes. Normal values for a midstream specimen are less than or equal to $10^5$ *Escherichia coli* organisms and $10^4$ leukocytes per milliliter. These classical “Kass criteria,” however, are not always reliable. In some cases of incipient cystitis the number of *E. coli* per milliliter can be lower, on the order of $10^2$ to $10^4$ [4]. When fecal contamination has been ruled out, growth of bacteria that are not normally urethral saprophytes indicates infection. This is the case for *Pseudomonas*, *Klebsiella*, *Enterobacter*, *Serratia*, and *Moraxella*, among others, especially in a hospital setting or after urologic procedures.
CAUSES OF ASEPTIC LEUKOCYTURIA

Self-medication before urine culture
Sample contamination by cleansing solution
Vaginal discharge
Urinary stone
Urinary tract tumor
Chronic interstitial nephritis (especially due to analgesics)
Fastidious microorganisms requiring special culture medium (Ureaplasma urealyticum, Chlamydia, Candida)

Leukocyturia. A significant number of leukocytes (more than 10,000 per milliliter) is also required for the diagnosis of urinary tract infection, as it indicates urothelial inflammation. Abundant leukocyturia can originate from the vagina and thus does not necessarily indicate aseptic urinary leukocyturia [1]. Bacterial growth without leukocyturia indicates contamination at sampling. Significant leukocyturia without bacterial growth (aseptic leukocyturia) can develop from various causes, among which self-medication before urinalysis is the most common.

Bacteriology

A. MAIN MICROBIAL STRAINS RESPONSIBLE FOR URINARY TRACT INFECTION

<table>
<thead>
<tr>
<th>Microbial Strain</th>
<th>First Episode or Delayed Relapse</th>
<th>Relapse Due to Early Reinfection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Escherichia coli</td>
<td>71%–79%</td>
<td>60%</td>
</tr>
<tr>
<td>Proteus mirabilis</td>
<td>11%–9.7%</td>
<td>15%</td>
</tr>
<tr>
<td>Klebsiella</td>
<td>—</td>
<td>20%</td>
</tr>
<tr>
<td>Enterobacter</td>
<td>1.0%–9.2%</td>
<td>—</td>
</tr>
<tr>
<td>Enterococcus</td>
<td>1.0%–3.2%</td>
<td>—</td>
</tr>
<tr>
<td>Staphylococcus saprophyticus</td>
<td>3%–7%</td>
<td>—</td>
</tr>
<tr>
<td>Other species</td>
<td>2%–6%</td>
<td>5%</td>
</tr>
</tbody>
</table>

Principal pathogens of urinary tract infection (UTI). A and B, Most pathogens responsible for UTI are enterobacteriaceae with a high pre-dominance of Escherichia coli. This is especially true of spontaneous UTI in females (cystitis and pyelonephritis). Other strains are less common, including Proteus mirabilis and more rarely gram-positive microbes. Among the latter, Staphylococcus saprophyticus deserves special mention, as this gram-positive pathogen is responsible for 5% to 15% of such primary infections, is not detected by the leukocyte esterase dipstick, and is resistant to antimicrobial agents that are active on gram-negative rods.

C, Acute simple pyelonephritis is a common form of upper UTI in females and results from the encounter of a parasite and a host. In the absence of urologic abnormality, this renal infection is mostly due to uropathogenic strains of bacteria [5,6], a majority of cases to community-acquired E. coli. The clinical picture consists of fever, chills, renal pain, and a general discomfort. Tissue invasion is associated with a high erythrocyte sedimentation rate and C-reactive protein level well above 2 mg/dL.

FIGURE 7-3

FIGURE 7-4

E. coli P. mirabilis Klebsiella Enterobacter Enterococcus S. saprophyticus Other
Virulence Factors of Uropathogenic Strains

**FIGURE 7-5**
Bacterial uropathogenicity plays a major role in host-pathogen interactions that lead to urinary tract infection (UTI). For *Escherichia coli*, these factors include flagella necessary for motility, aerobactin necessary for iron acquisition in the iron-poor environment of the urinary tract, a pore-forming hemolysin, and, above all, presence of adhesins on the bacterial fimbriae, as well as on the bacterial cell surface. (From Mobley et al. [7]; with permission.)

**FIGURE 7-6**
An electron microscopic view of an *Escherichia coli* organism showing the fimbriae (or pili) bristling from the bacterial cell.

**FIGURE 7-7**
*Proteus mirabilis* is endowed with other nonfimbrial virulence factors, including the property of secreting urease, which splits urea into \( \text{NH}_3 \) and \( \text{CO}_2 \).

**FIGURE 7-8**
Staghorn calculi. Ammonium generation alkalizes the urine, creating conditions favorable for build-up of voluminous struvite stones, which can progressively invade the entire pyelocalyceal system, forming staghorn calculi. These stones are an endless source of microbes, and the urinary tract obstruction perpetuates infection.
7.5 Urinary Tract Infection

FIGURE 7-9
Schematic representation of morphology and composition of type P and type 1 adhesive structures. Bacterial adhesins are paramount in fostering attachment of the bacteria to the mucous membranes of the perineum and of the urothelium. There are several molecular forms of adhesins. The most studied is the pap G adhesin, which is located at the tip of the bacterial fimbriae (or pili). This lectin recognizes binding site conformations provided by oligosaccharide sequences present on the mucosal surface [8].

FIGURE 7-10
Uropathogenic strains of Escherichia coli readily adhere to epithelial cells. This figure shows two epithelial cells incubated in urine infected with E. coli-carrying pap adhesins. Numerous bacteria are scattered on the epithelial cell membranes. About half of all cases of cystitis are due to uropathogenic strains of E. coli-carrying adhesins. Females with primary pyelonephritis and no urologic abnormality harbor a uropathogenic strain in almost 100% of cases [5].

FIGURE 7-11
Appropriate antibiotics for urinary tract infections (UTI). An appropriate antibiotic for treating UTI must be bactericidal and conform to the following general specifications: 1) its pharmacology must include, in case of oral administration, rapid absorption and attainment of peak serum concentrations; 2) its excretion must be predominantly renal; 3) it must achieve high concentrations in the renal or prostate tissue; 4) it must cover the usual spectrum of enterobacteria with reasonable chance of being effective on an empirical basis. Excluding special considerations for childhood and pregnancy, several classes of antibiotics fulfill these specifications and can be used alone or in combination. The choice also depends on market availability, cost, patient tolerance, and potential for inducing emergence of resistant strains.

### APPROPRIATE ANTIBIOTICS FOR URINARY TRACT INFECTIONS

<table>
<thead>
<tr>
<th>Antibiotics</th>
<th>General Indications</th>
<th>Pregnancy</th>
<th>Prophylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aminoglycosides</td>
<td>+</td>
<td>+†</td>
<td>-</td>
</tr>
<tr>
<td>Aminopenicillins</td>
<td>+†</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Carboxypenicillins</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Ureidopenicillins</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Quinolones</td>
<td>+†</td>
<td>-</td>
<td>+†</td>
</tr>
<tr>
<td>Fluoroquinolones</td>
<td>+</td>
<td>-</td>
<td>+†</td>
</tr>
<tr>
<td>Cephalosporins</td>
<td>+†</td>
<td>+</td>
<td>-†</td>
</tr>
<tr>
<td>First generation</td>
<td>+†</td>
<td>+</td>
<td>+†</td>
</tr>
<tr>
<td>Second generation</td>
<td>+†</td>
<td>+</td>
<td>+†</td>
</tr>
<tr>
<td>Third generation</td>
<td>+†</td>
<td>+</td>
<td>+†</td>
</tr>
<tr>
<td>Monobactams</td>
<td>+</td>
<td>+</td>
<td>-†</td>
</tr>
<tr>
<td>Carbapenem</td>
<td>+</td>
<td>+</td>
<td>-†</td>
</tr>
<tr>
<td>Cotrimoxazole</td>
<td>+</td>
<td>-</td>
<td>+†</td>
</tr>
<tr>
<td>Fosfomycin trometamol</td>
<td>+**</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Nitrofurantoin</td>
<td>+††</td>
<td>-</td>
<td>+††</td>
</tr>
</tbody>
</table>

* Aminoglycosides should not be prescribed during pregnancy except for very severe infection and for the shortest possible duration. With the exception of amoxicillin plus clavulanic acid, aminopenicillins should not be prescribed as first-line treatment, owing to the frequency of primary resistance to this class of antibiotics.
† According to antibiotic sensitivity tests.
†† Fluoroquinolones carry a risk of tendon rupture (especially Achilles tendon).
+ Oral administration only.
** Single-dose treatment of cystitis.
†† Simple cystitis; not pyelonephritis or prostatitis.
Classification of Urinary Tract Infection

Upper versus lower urinary tract infection

**FIGURE 7-12**
Cystitis in a female patient. In case of urinary tract infection (UTI), distinguishing between lower and upper tract infection is classical, but the distinction is also beside the point. The real point is to determine whether infection is confined to the bladder mucosa, which is the case in simple cystitis in females, or whether it involves solid organs (i.e., prostatitis or pyelonephritis). The dots in this figure symbolize the presence of bacteria and leukocytes (i.e., infection) in the relevant organ. Here, infection is confined to the bladder mucosa, which can be severely inflamed and edematous. This could be reflected radiographically by mucosal wrinkling on the cystogram. In some cases inflammation is severe enough to be accompanied by bladder purpura, which induces macroscopic hematuria but is not a particular grave sign.

**FIGURE 7-13**
Prostatitis. Anatomically, prostatitis involves the lower urinary tract, but invasion of prostate tissue affords easy passage of pathogens to the prostatic venous system—and, usually, poor penetration by antibiotics. Presence of bacteria in the bladder is also symbolized in this picture, but owing to free communication between bladder urine and prostate tissue, it can be accepted that pure cystitis does not exist in males.

**FIGURE 7-14**
A cute prostatitis can be complicated by ascending infection, that is, pyelonephritis.

**FIGURE 7-15**
Pyelonephritis in females. Essentially, this is an ascending infection caused by uropathogens. From the perineum the bacteria gain access to the bladder, ascending to the renal pelvocalyceal system and thence to the renal medulla, from which they spread toward the cortex. It has been shown that "pyelitis" cannot be considered a pathologic entity, as renal pelvis infection is invariably associated with nearby contamination of the renal medulla.
Urinary Tract Infection

CRITERIA FOR TISSUE INVASION

Clinical
Kidney or prostate infection is marked by fever over 38°C, chills, and pain. The patient appears acutely ill.

Laboratory
Tissue invasion is invariably accompanied by an erythrocyte sedimentation rate over 20 mm/h and serum C-reactive protein levels over 2.0 mg/dL. Blood cultures grow in 30%–50% of cases, which in an immunocompetent host indicates simply bacteremia, not septicemia. This reflects easy permeability between the urinary and the venous compartments of the kidney.

Imaging
When indicated, ultrasound imaging, tomodensitometry, and scintigraphy provide objective evidence of pyelonephritis. In case of vesicoureteral reflux, urinary tract infection necessarily involves the upper urinary tract.

Primary versus secondary urinary tract infection

Primary versus secondary urinary tract infection

Recurrent cystitis in females can be explained by hymeneal scars that pull open the urethral outlet during intercourse. Although rarely, other malformations that promote recurrent female cystitis are occasionally discovered, such as vesical diverticula (arrows).

Finally, it should be recalled that recurrent or chronic cystitis in an older woman can also reveal an unsuspected bladder tumor.
Tubulointerstitial Disease

FIGURE 7-20
Urethrocystogram of a man following acute prostatitis. In males, acute prostatitis may reveal urethral stenosis. Urethral stenosis is a good explanation for acute prostatitis. The beaded appearance of the stenosis (arrow) suggests an earlier episode of gonorrheal urethritis.

FIGURE 7-21
The severity of vesicoureteral reflux (VUR) as graded in 1981 by the International Reflux Study Committee. When children have pyelonephritis, the possibility of VUR should always be considered. Childhood vesicoureteral reflux is five times more common in girls than in boys. It has a genetic background; several cases occasionally occur in the same family. Unless detected and corrected early, especially the most severe forms of this class and when urine is infected (one episode of pyelonephritis suffices), childhood VUR is a major cause of cortical scarring, renal atrophy, and in bilateral cases chronic renal insufficiency. The International Reflux Study classifies reflux grades as follows: I) ureter only; II) ureter, pelvis, and calyces, no dilation, and normal calyceal fornices; III) mild or moderate dilation or tortuosity of ureter and mild or moderate dilation of renal pelvis but no or slight blunting of fornices; IV) moderate dilation or tortuosity of ureter and moderate dilation of renal pelvis and calyces, complete obliteration of sharp angle of fornices but maintenance of papillary impressions in majority of calyces; V) gross dilation and tortuosity of ureter, gross dilation of renal pelvis and calyces. Papillary impressions are no longer visible in the majority of calyces. (From International Reflux Study Committee [9]; with permission.)

FIGURE 7-22
Cystogram demonstrating left ureteral reflux (A). The consequences on the left kidney (B) consist of calyceal distension and a clubbed appearance due to the destruction of the papillae and of the adjacent renal tissue. The calyceal cavities are very close to the renal capsule, indicating complete cortical atrophy. This picture is typical of chronic pyelonephritis secondary to vesicoureteral reflux.
In case of bilateral, neglected vesicoureteral reflux, chronic pyelonephritis is bilateral and asymmetric. Here, the right kidney is globally atrophic. A typical cortical scar is seen on the outer aspect of the left kidney. The lower pole, however, is fairly well-preserved with nearly normal parenchymal thickness.

When intravenous pyelography discloses two ureters, the one draining the lower pyelocalyceal system crosses the upper ureter and opens into the bladder less obliquely than normally, allowing reflux of urine and explaining repeated attacks of pyelonephritis followed by atrophy of the lower pole of the kidney. Retrograde cystography is indicated for repeated episodes of pyelonephritis and when intravenous pyelography or computed tomography renal examination discovers cortical scars. In adults, retrograde cystography is obtained by direct catheterization of the bladder.

In children, isotopic cystography allows a diagnosis of vesicoureteral reflux with much less radiation than if cystography were carried out with iodinated contrast medium.

In the paraplegic, and more generally in patients with spinal disease, neurogenic bladder is responsible for stasis, bladder distension, and diverticula. These functional and anatomic factors explain the frequency of chronic urinary tract infection complicated with bladder and upper urinary tract infectious stones.
When acute pyelonephritis occurs in a sound, immunocompetent female with no history of urologic disease, imaging can be limited to a plain abdominal film (to rule out renal and ureteral stones) and renal ultrasonography. Ultrasonography typically discloses a swollen kidney with loss of corticomedullary differentiation, denoting renal inflammatory edema. Images corresponding to the infected zones are more dense than normal renal tissue (arrows).

The ultrasound procedure occasionally discloses the cavity of a small renal abscess, a common complication of acute pyelonephritis, even in simple forms.

Computed tomodensitometry. Simple pyelonephritis does not require much imaging; however, it should be remembered that there is no correlation between the severity of the clinical picture and the renal lesions. Therefore, a diagnosis of “simple” pyelonephritis at first contact can be questioned when response to treatment is not clear after 3 or 4 days. This is an indication for uroradiologic imaging, such as renal tomodensitometry followed by radiography of the urinary tract while it is still opacified by the contrast medium.

The typical picture of acute pyelonephritis observed after contrast medium injection [10] consists of hypodensities of the infected areas in an edematous, swollen kidney. The pathophysiology of hypodense images has been elucidated by animal experiments in the primates [11] which have shown that renal infection with uropathogenic Escherichia coli induces intense vasoconstriction.

Computed tomodensitometric images of acute pyelonephritis can take various appearances. The most common findings consist of one or several wedge-shaped or streaky zones of low attenuation extending from papilla to cortex, A. Hypodense images can be round, B. On this figure, the infected zone reaches the renal cortex and is accompanied with adjacent perirenal edema. Several such

(Continued on next page)
FIGURE 7-29 (Continued)
Images can coexist in the same kidney, C. Marked juxtacortical, circumscribed hypodense zones, bulging under the renal capsule, D, usually correspond to lesions close to liquefaction and should be closely followed, as they can lead to abscess formation and opening into the perinephric space, E and F. (E and F from Talner et al. [10]; with permission.)

FIGURE 7-30
Comparative sensitivity of four diagnostic imaging techniques for acute pyelonephritis. Renal cortical scintigraphy using ⁹⁹ᵐTc-dimethyl succinic acid (DMSA) or ⁹⁹ᵐTc-gluconoheptonate (GH) is very sensitive for diagnosing acute pyelonephritis. It entails very little irradiation as compared with conventional radiography using contrast medium. Some nephrologists consider ⁹⁹ᵐTc-DMSA cortical scintigraphy as the first-line diagnostic imaging method for renal infection in children. It is interesting to compare its sensitivity with that of more conventional imaging methods. (From Meyrier and Guibert [5]; with permission.)

Urinary Tract Infection

7.11
Tubulointerstitial Disease

**FIGURE 7-33** Histologic appearance of pyelonephritic kidney. A, The renal tissue is severely edematous and interspersed with inflammatory cells and hemorrhagic streaks. B, On another section, severe inflammation, comprising a majority of polymorphonuclear leukocytes, induces tubular destruction and is accompanied by a typical infectious cast in a tubular lumen (arrow).

**FIGURE 7-32** Renal pathology in acute pyelonephritis. Renal pathology of human acute pyelonephritis is quite comparable to what is observed in experimental pyelonephritis in primates [11]. However, our knowledge of renal pathology in this condition in humans is based mainly on the most catastrophic cases, which required nephrectomy, like the diabetes patient whose kidney is shown here. A, The surgically removed kidney is swollen, and its surface shows whitish zones. B, A section of the same organ shows white suppurative areas (scattered with small abscesses) extending eccentrically from the medulla to the cortex. There also were sloughed papillae (see Fig. 7-37).

**FIGURE 7-31** (see Color Plate) $^{99m}$Tc-DMSA cortical imaging of simple pyelonephritis in a female. The clinical signs implicated the right kidney. (Contrary to conventional radiology, the right kidney appears on the right of the image.) The false colors indicate cortical renal blood supply from red (normal) to blue (ischemia). The right kidney is obviously involved with pyelonephritis, especially its poles. However, contrary to the results of computed tomography, which indicated right-sided pyelonephritis only, a focus of infection also occupies the lower pole of the right kidney. This picture illustrates the greater sensitivity of renal scintigraphy for diagnosing renal infection. It also indicates that clinically unilateral acute pyelonephritis can, in fact, be bilateral.
FIGURE 7-34
A general algorithm for the investigation and treatment of acute pyelonephritis. Treatment of acute pyelonephritis is based on antibiotics selected from the list in Figure 7-11. Preferably, initial treatment is based on parenteral administration. It is debatable whether common forms of simple pyelonephritis initially require both an aminoglycoside and another antibiotic. Initial parenteral treatment for an average of 4 days should be followed by about 10 days of oral therapy based on bacterial sensitivity tests. It is strongly recommended that urine culture be carried out some 30 to 45 days after the end of treatment, to verify that bacteriuria has not recurred.

APN — acute pyelonephritis; ESR — erythrocyte sedimentation rate; CRP — C-reactive protein; UTI — urinary tract infection; IVP — intravenous pyelography. (From Meyrier and Guibert [5]; with permission.)
Renal abscess. Like acute pyelonephritis, one third of cases of renal abscess occur in a normal urinary tract; in the others it is a complication of a urologic abnormality. The clinical picture is that of severe pyelonephritis. In fact, it can be conceptualized as an unfavorably developing form of acute pyelonephritis that progresses from presuppurative to suppurative renal lesions, leading to liquefaction and formation of a walled-off cavity. The diagnosis of renal abscess is suspected when, despite adequate treatment of pyelonephritis (described in Fig. 7-34), the patient remains febrile after day 4. Here, necrotic renal tissue is visible close to the abscess wall. The tubules are destroyed, and the rest of the preparation shows innumerable polymorphonuclear leukocytes within purulent material.

Renal computed tomography (CT). In addition to ultrasound examination, CT is the best way of detecting and localizing a renal abscess. The abscess cavity can be contained entirely within the renal parenchyma, A, or bulge outward under the renal capsule, risking rupture into Gerota’s space, B.
Urinary tract infection (UTI) in the immunocompromised host. UTI results from the encounter of a pathogen and a host. Natural defenses against UTI rest on both cellular and humoral defense mechanisms. These defense mechanisms are compromised by diabetes, pregnancy, and advanced age. Diabetic patients often harbor asymptomatic bacteriuria and are prone to severe forms of pyelonephritis requiring immediate hospitalization and aggressive treatment in an intensive care unit.

A particular complication of upper renal infection in diabetes is papillary necrosis (see Fig. 7-32). The pathologic appearance of a sloughing renal papilla, A. The sloughed papilla is eliminated and can be recovered by sieving the urine, B. In other cases, the necrotic papilla obstructs the ureter, causing retention of infected urine and severely aggravating the pyelonephritis. C. It can lead to pyonephrosis (ie, complete destruction of the kidney), as shown on CT.

Urinary tract infection (UTI) in an immunocompromised host. Pregnancy is associated with suppression of the host's immune response, in the form of reduced cytotoxic T-cell activity and reduced circulating immunoglobulin G (IgG) levels. Asymptomatic bacteriuria is common during pregnancy and represents a major risk of ascending infection complicated by acute pyelonephritis.

(Continued on next page)
Petersson and coworkers [12] recently demonstrated that the susceptibility of the pregnant woman to acute UTI is accompanied by reduced serum antibody activity (IgG, IgA, IgM), reduced urine antibody activity (IgG, IgA), and low interleukin 6 (IL-6) response, A–C, respectively. The last may indicate that pregnant women have a generally reduced level of mucosal inflammation. These factors may be crucial for explaining the frequency and the severity of acute pyelonephritis during pregnancy. (From Petersson et al. [12]; with permission.)

Acute prostatitis as visualized sonographically. Acute prostatitis is common after urethral or bladder infection (usually by Escherichia coli or Proteus organisms). Another cause is prostate hematogenous contamination, especially by Staphylococcus. Signs and symptoms of acute prostatitis, in addition to fever, chills, and more generally the signs and symptoms of tissue invasion by infection described above, are accompanied by dysuria, pelvic pain, and septic urine. Acute prostatitis is an indication for direct ultrasound (US) examination of the prostate by endorectal probe. In this case of acute prostatitis in a young male, US examination disclosed a prostatic abscess (1) complicating acute prostatitis in the right lobe (2). Acute prostatitis is an indication for thorough radiologic imaging of the whole urinary tract, giving special attention to the urethra. Urethral stricture may favor prostate infection (see Fig. 7-20).
Xanthogranulomatous pyelonephritis (XPN). XPN is a special form of chronic renal inflammation caused by an abnormal immune response to infected obstruction [13]. This case in a middle-aged woman with a long history of renal stones is typical. For several months she complained of flank pain, fever, fatigue, anorexia and weight loss. Laboratory workup found inflammatory anemia and increased erythrocyte sedimentation rate and C-reactive protein levels. Urinalysis showed pyuria and culture grew Escherichia coli. CT scan of the right kidney showed replacement of the renal tissue by several rounded, low-density areas and detected an obstructive renal stone. Nephrectomy was performed. A. The obstructive renal stone is shown by an arrowhead. The renal cavities are dilated. The xanthogranulomatous tissue (arrows) consists of several round, pseudotumoral masses with a typical yellowish color due to presence of lipids. In some instances such xanthogranulomatous tissue extends across the capsule into the perirenal fat and fistulizes into nearby viscera such as the colon or duodenum. B. Microscopic view of the xanthogranulomatous tissue. This part of the lesion is made of lipid structures composed of innumerable clear droplets.

Malakoplakia. Malakoplakia (or malacoplakia), like xanthogranulomatous pyelonephritis, is also a consequence of abnormal macrophage response to gram-negative bacteria. A. Malakoplakia occurs in association with chronic UTI [14]. In more than 20% of cases, affected persons have some evidence of immunosuppression, especially corticosteroid therapy for autoimmune disease. In 13% of the published cases, malakoplakia involved a transplanted kidney. The female-male ratio is 3:1. Lesions can involve the kidney, the bladder, or the ureter and form pseudotumors. B. Histologically, malakoplakia is distinguished by large, pale, periodic acid–Schiff–positive macrophages (von Hansemann cells) containing calcific inclusions (Michaelis-Gutmann bodies). The larger ones are often free in the interstitium. Malakoplakia, an unusual form of chronic tubulointerstitial nephritis, must be recognized by early renal biopsy and can resolve, provided treatment consisting of antibiotics with intracellular penetration is applied for several weeks. (B, Courtesy of Gary S. Hill, M.D.)
References