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INFLAMMATIVE DISEASES OF UROGENITAL SYSTEM

Educational manual

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The study guide "Inflammatory diseases of the genitourinary system" presents materials for preparing for practical classes in urology and for increasing the level of theoretical training of English-speaking students of the 4th year of the medical faculty.

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CONTENT

CONDITIONAL ABBREVIATIONS

UTI - Urinary tract infection

TMP-SMX - Trimethoprim-sulfamethoxazole

CT - Computed tomography

XGP - Xanthogranulomatous pyelonephritis

APRT - adenine phosphoribosyltransferase

KUB - kidney-ureter-bladder

ESWL - extracorporeal shock wave lithotripsy

IVP - intravenous pyelogram

PREFACE

Urology is a branch of clinical medicine, in particular surgery, which studies diseases of the urinary organs and the male reproductive system. More than two millennia passed from the time of Hippocrates, when doctors used uroscopy to diagnose any disease, to the separation of urology into an independent discipline. Over the past century, and especially in recent decades, thanks to the rapid general scientific and technical development, urology has evolved into one of the most progressive high-tech branches of modern clinical medicine. This, accordingly, requires from doctors an increasingly in-depth study of the discipline, and from teachers of medical universities - to improve the educational process.

The main task of the discipline of urology is the study of the main sections of urology, clinic and diagnosis of urological diseases, methods of conservative and operative treatment. The main feature of this manual is that it was developed in accordance with the standard and working program for the study discipline "Urology" for students of higher medical institutions of education of Ukraine of the III-IV levels of accreditation, approved by the Ministry of Health of Ukraine.

The purpose of creating the training manual was to increase the theoretical training of students on the topic "Inflammatory diseases of the genitourinary system". It was a difficult task: to equip students with modern basic knowledge of urology within a rather concise curriculum, which would allow in practice to correctly assess the patient's condition and provide him with assistance within the limits of the doctor's capabilities, without making a mistake.

In the manual, most of the illustrative material is created by the authors, otherwise - a link to the source is added. Tasks for test control can be used during extracurricular and classroom training.

NONSPECIFIC INFLAMMATORY DISEASES OF THE KIDNEYS AND PARANEPHRAL TISSUE. ACUTE PYELONEPHRITIS, ACUTE PARANEPHRITIS.

Bacterial Infections of the Genitourinary Tract: Introduction.

Urinary tract infection (UTI) is a term that is applied to a variety of clinical conditions ranging from the asymptomatic presence of bacteria in the urine to severe infection of the kidney with resultant sepsis. UTI is one of the more common medical problems. It is estimated that 150 million patients are diagnosed with a UTI yearly, resulting in at least $6 billion in health care expenditures (Stamm and Norrby, 2001). UTIs are at times difficult to diagnose; some cases respond to a short course of a specific antibiotic, while others require a longer course of a broad-spectrum antibiotic. Accurate diagnosis and treatment of a UTI is essential to limit its associated morbidity and mortality and avoid prolonged or unnecessary use of antibiotics. Advances in our understanding of the pathogenesis of UTI, the development of new diagnostic tests, and the introduction of new antimicrobial agents have allowed physicians to appropriately tailor specific treatment for each patient.

Epidemiology

The epidemiology of UTI grouped by age and sex is shown in Table 1. In newborns up to 1 year of age, bacteriuria is present in 2.7% of boys and 0.7% in girls (Wettergren, Jodal, and Jonasson, 1985). The incidence of UTI in uncircumcised males is higher than in circumcised males (1.12% compared to 0.11%) during the first 6 months of life (Wiswell and Roscelli, 1986). In children between 1 and 5 years of age, the incidence of bacteriuria in girls increases to 4.5%, while it decreases in boys to 0.5% (Randolph and Greenfield, 1964). Most UTIs in children younger than 5 years are associated with congenital abnormalities of the urinary tract, such as vesicoureteral reflux or obstruction. The incidence of bacteriuria remains relatively constant in children 6–15 years of age. However, the UTIs in these children are more likely to be associated with functional abnormalities

of the urinary tract, such as dysfunctional voiding. During adolescence, the incidence of UTI significantly increases (to 20%) in young women, while remaining constant in young men (Sanford, 1975).

Table 1. Epidemiology of UTI by Age Group and Sex.

	Incidence (%)		
Age (y)	**Female**	**Male**	**Risk Factors**
< 1	0.7	2.7	Foreskin, anatomic GU abnormalities
1–5	4.5	0.5	Anatomic GU abnormalities
6–15	4.5	0.5	Functional GU abnormalities
16–35	20	0.5	Sexual intercourse, diaphragm use
36–65	35	20	Surgery, prostate obstruction, catheterization
> 65	40	35	Incontinence, catheterization, prostate obstruction

Approximately 7 million cases of acute cystitis are diagnosed yearly in young women (Schappert, 1999); this likely is an underestimate of the true incidence of UTI because at least 50% of all UTIs do not come to medical attention. The major risk factors for women 16–35 years of age are related to sexual intercourse and diaphragm use. Later in life, the incidence of UTI increases significantly for both males and females. For women between 36 and 65 years of age, gynecologic surgery and bladder prolapse appear to be important risk factors. In men of the same age group, prostatic hypertrophy/obstruction, catheterization, and surgery are relevant risk factors. For patients older than 65 years, the incidence of UTI continues to increase in both sexes. Incontinence and chronic use of urinary catheters are important risk factors in these patients. In those younger than 1 year and those older than 65 years, the morbidity and mortality of UTI are the greatest (Shortliffe and McCue, 2002).

Pathogenesis. Bacterial Entry.

Understanding of the mode of bacterial entry, host susceptibility factors, and bacterial pathogenic factors is essential to tailoring appropriate treatment for the

diverse clinical manifestations of UTI. There are 4 possible modes of bacterial entry into the genitourinary tract. It is generally accepted that periurethral bacteria ascending into the urinary tract causes most UTI. Most cases of pyelonephritis are caused by the ascent of bacteria from the bladder, through the ureter and into the renal parenchyma. Consequently, the short nature of the female urethra combined with its close proximity to the vaginal vestibule and rectum likely predisposes women to more frequent UTIs than men (Nicolle et al, 1982).

Other modes of bacterial entry are uncommon causes of UTI. Hematogenous spread can occur in immunocompromised patients and in neonates. *Staphylococcus aureus, Candida* species, and *Mycobacterium tuberculosis* are common pathogens that travel through the blood to infect the urinary tract. Lymphatogenous spread through the rectal, colonic, and periuterine lymphatics has been postulated as a cause for UTI; however, currently there is little scientific support to suggest that dissemination of bacteria through lymphatic channels plays a role in the pathogenesis of UTI. Direct extension of bacteria from adjacent organs into the urinary tract can occur in patients with intraperitoneal abscesses or vesicointestinal or vesicovaginal fistulas. Relapsing infection from an inadequately treated focus in the prostate or kidney may seed other parts of the urinary tracts.

Host Defenses

Host factors have an essential role in the pathogenesis of UTI. Unobstructed urinary flow with the subsequent washout of ascending bacteria is essential in preventing UTI. In addition, the urine itself has specific characteristics (its osmolality, urea concentration, organic acid concentration, and pH) that inhibit bacterial growth and colonization (Sobel, 1997). It also contains factors that inhibit bacterial adherence, such as Tamm-Horsfall glycoprotein (Duncan, 1988; Pak et al, 2001). Urinary retention, stasis, or reflux of urine into the upper urinary tract can promote bacterial growth and subsequent infection. Consequently, any anatomic or functional abnormalities of the urinary tract that impede urinary flow can increase the host's susceptibility to UTI. These abnormalities include obstructive conditions at any level of the urinary tract, neurologic diseases affecting the function of the

lower urinary tract, diabetes, and pregnancy. Similarly, the presence of foreign bodies (such as stones, catheters, and stents) allows the bacteria to hide from these host defenses.

In response to the presence of bacteria, cells lining the urinary tract secrete chemoattractants such as interleukin-8 to recruit neutrophils to the area and limit tissue invasion (Frendeus et al, 2001). Specific serum and urinary antibodies are produced by the kidney to enhance bacterial opsonization and phagocytosis and to inhibit bacterial adherence. The protective role of both cellular and humoral-mediated immunity in preventing UTIs remains unclear; deficiency in B-cell or T-cell function has not been associated with the increased frequency of UTI or altered the course of the infection (Schaeffer, 2001; Svanborg Eden et al, 1988).

Many studies have demonstrated that there is selectivity in bacterial adherence to cells lining the urinary tract, and the degree of adherence correlates with colonization and infection. Women with recurrent UTIs have higher adherence of bacteria to their mucosal cells in vitro compared to women who never had an infection (Navas et al, 1994). The increased adherence may be due to having more binding sites for bacterial adhesins on their mucosal cells. Alternatively, these patients may not secrete soluble compounds, which normally compete for the same receptors that bind bacterial adhesins. Blood group antigens may constitute one group of these soluble compounds that inhibit bacterial adherence (Lomberg et al, 1986). These findings would suggest a genetic predisposition for UTI.

Other important host factors include the normal flora of the periurethral area or the prostate and the presence of vesicoureteral reflux. In women, the normal flora of the periurethral area composed of organisms such as lactobacillus provide a defense against the colonization of uropathogenic bacteria (Osset et al, 2001). Alterations in the periurethral environment (such as changes in the pH or estrogen levels or the use of antibiotics) can damage the periurethral flora, allowing uropathogens to colonize and subsequently to infect the urinary tract (Schaeffer et al, 1999). In men, the prostate secretes fluid containing zinc, which has potent antimicrobial activity (Fair, Couch, and Wehner, 1976). Finally, in children, the presence of vesicoureteral

reflux does not increase their susceptibility to UTI but does allow bacteria to be inoculated into the upper tract and the infection to progress.

Aging is associated with an increased susceptibility to UTI, in part because of the increased incidence of obstructive uropathy in men (Matsumoto, 2001; Nicolle, 2002) and alteration in the vaginal and periurethral flora from menopause in women (Foxman et al, 2001). Other causes include soiling of the perineum from fecal incontinence, neuromuscular diseases, increased instrumentation, and bladder catheterization (Ronald, 2002).

Bacterial Pathogenic Factors

Not all bacteria are capable of adhering to and infecting the urinary tract. Of the many strains of *Escherichia coli,* the uropathogens belong to a limited number of O, K, and H serogroups. They have increased adherence properties to uroepithelial cells (Blanco et al, 1996; Hovanec and Gorzynski, 1980; Orskov et al, 1982), resistance to the bactericidal activity of human serum (Bjorksten and Kaijser, 1978), production of hemolysin (Hughes et al, 1983; Koronakis and Hughes, 1996), and the increased expression of K capsular antigen (Whitfield and Roberts, 1999). The ability of *E coli* to adhere to epithelial cells is mediated by ligands located on the tips of the bacterial fimbriae (pili). The ligands bind to glycolipids or glycoprotein receptors on the surface membrane of uroepithelial cells. The pili are classified by their ability to cause hemagglutination and the type of sugar that can block this process. P pili, which can agglutinate human blood, bind to glycolipid receptors on uroepithelial cells, erythrocytes (P-blood group antigens), and renal tubular cells (Svenson et al, 1983). Type 1 pili, which can agglutinate guinea pig blood, bind to mannoside residues on uroepithelial cells (Ofek et al, 2000). P pili are observed in over 90% of the *E coli* strains causing pyelonephritis but less than 20% of the strains causing lower urinary tract infections (Kallenius et al, 1981; JA Roberts et al, 1997). In contrast, type 1 pili may help bacteria to adhere to bladder mucosa (Connell et al, 1996; Martinez et al, 2000). Most uropathogenic *E coli* have both types of pili. Once attachment to the uroepithelial cells occurs, other bacterial pathogenic factors become important. Most uropathogenic *E coli* strains produce

hemolysin, which initiates tissue invasion and makes iron available for the infecting pathogens (Hughes et al, 1983; Koronakis and Hughes, 1996). The presence of K antigen on the invading bacteria protects them from phagocytosis by neutrophils (Bortolussi et al, 1979; Evans et al, 1981). These factors allow the infecting pathogens to escape the various host defenses (Svanborg et al, 1996).

Causative Pathogens

Most UTIs are caused by a single bacterial species. At least 80% of the uncomplicated cystitis and pyelonephritis are due to *E coli*, with most of pathogenic strains belong to the O serogroups (Orskov et al, 1982). Other less common uropathogens include *Klebsiella*, *Proteus,* and *Enterobacter* spp. and enterococci. In hospital-acquired UTIs, a wider variety of causative organisms is found, including *Pseudomona*s and *Staphylococcus* spp. (Wagenlehner and Naber, 2000). UTIs caused by *S aureus* often result from hematogenous dissemination. Group B - hemolytic streptococci can cause UTIs in pregnant women (Wood and Dillon, 1981). *Staphylococcus saprophyticus*, once often thought of as urinary contaminants, can cause uncomplicated UTIs in young women (Hovelius and Mardh, 1984). In children, the causative bacterial spectrum is slightly different from that of adults, with *Klebsiella* and *Enterobacter* spp. being more common causes of UTI (Jeena et al, 1996; Ronald, 2002; Schlager, 2001). Anaerobic bacteria, lactobacilli, corynebacteria, streptococci (not including enterococci) and *Staphylococcus epidermidis* are found in normal periurethral flora. They do not commonly cause UTIs in healthy individuals and are considered common urinary contaminants.

Diagnosis

The diagnosis of UTI is sometimes difficult to establish and relies on urinalysis and urine culture. Occasionally, localization studies may be required to identify the source of the infection. Most often, the urine is often obtained from a voided specimen. In children who are not toilet-trained, a urine collection device, such as a bag, is placed over the genitalia, and the urine is cultured from the bagged specimen. These 2 methods of urine collection are easy to obtain, but potential contamination

from the vagina and perirectal area may occur. There is a high false-positive rate, especially from bagged specimens (Al-Orifi et al, 2000). Suprapubic aspiration avoids potential contamination; however, due to its invasiveness, it is rarely used except in children and selected patients. Urine obtained from a urinary catheter is less invasive than a suprapubic aspiration and is less likely to be contaminated than that from a voided specimen. If a patient has an indwelling catheter, a urine specimen should be obtained from the collection port on the catheter.

Urinalysis

Urinalysis provides a rapid screen for UTIs. The urine can be immediately evaluated for leukocyte esterase, a compound produced by the breakdown of white blood cells (WBCs) in the urine. Urinary nitrite is produced by reduction of dietary nitrates by many gram-negative bacteria. Esterase and nitrite can be detected by a urine dipstick and are more reliable when the bacterial count is greater than 100,000 colony-forming units (CFU) per milliliter. Microscopic examination of the urine for WBCs and bacteria is performed after centrifugation. When bacteria counts are greater than 100,000 CFU/ mL, bacteria can be detected microscopically (Jenkins, Fenn, and Matsen, 1986). More than 3 WBCs per high-power field suggests a possible infection. The sensitivity and specificity of these tests are shown in Table 2. The urinary nitrite test is highly specific but not sensitive, whereas the other 3 tests have a sensitivity and specificity approximately 80%. A combination of these tests may help to identify those patients in whom urine culture will be positive.

Table 2. Sensitivity and Specificity of Urinalysis.

Tests	**Sensitivity (%)**	**Specificity (%)**
Esterase	83 (67–94)	78 (64–92)
Nitrite	53 (15–82)	98 (90–100)
E or N	93 (90–100)	72 (58–91)
White blood cells	73 (32–100)	81 (45–98)
Bacteria	81 (16–99)	83 (11–100)
Any above	99.8 (99–100)	70 (60–92)

Urine Culture

The gold standard for identification of UTI is the quantitative culture of urine for specific bacteria. The urine should be collected in a sterile container and cultured immediately after collection. When this is not possible, the urine can be stored in the refrigerator for up to 24 hours. The sample is then diluted and spread on culture plates. Each bacterium will form a single colony on the plates. The number of colonies is counted and adjusted per milliliter of urine (CFU/mL). Defining the CFU/mL that represents clinically significant infection can be difficult. It is dependent on the method of collection, the sex of the patient, and the type of bacteria isolated (Table 3). Traditionally, greater than 100,000 CFU/mL is used to exclude contamination. However, studies have clearly demonstrated that clinically significant UTI can occur with less than 100,000 CFU/mL bacteria in the urine (Stamm et al, 1982).

Localization Studies

Occasionally, it is necessary to localize the site of infection. For upper urinary tract localization (Lorentz, 1979), the bladder is irrigated with sterile water and a ureteral catheter is placed into each ureter. A specimen is collected from the renal pelvis. Culture of this specimen will indicate whether infection in the upper urinary tract is present. In men, infection in the lower urinary tract can be differentiated (Meares and Stamey, 1968). A specimen is collected at the beginning of the void and represents possible infection in the urethra. A midstream specimen is next collected and represents possible infection in the bladder. The prostate is then massaged and the patient is asked to void again. This specimen represents possible infection of the prostate.

Antibiotics

Treatment with antimicrobial agents has minimized the morbidity and mortality associated with UTIs. The goal in treatment is to eradicate the infection by selecting the appropriate antibiotics that would target specific bacterial susceptibility. However, choosing the appropriate antimicrobial agents is often difficult. Many antibiotics are available, and the lowest effective dose and length of

therapy are not well defined. Many conventions for the treatment of UTI are arbitrary. The general principles for selecting the appropriate antibiotics include consideration of the infecting pathogen (antibiotic susceptibility, single-organism vs. poly-organism infection, pathogen vs. normal flora, community vs. hospital-acquired infection); the patient (allergies, underlying diseases, age, previous antibiotic therapy, other medications currently taken, outpatient vs. inpatient status, pregnancy); and the site of infection (kidney vs. bladder vs. prostate). Because most antibiotics are cleared from the body by the liver or the kidney, certain antimicrobial agents need to be adjusted in the presence of liver or renal diseases (Table 5). Table 6 lists the common uropathogens and the recommended oral and intravenous antimicrobial agents for treatment. Table 7 lists the common sites of UTI, the recommended treatment, and the duration of therapy. In patients with recurrent UTIs or those who are at risk for UTI (such as children with vesicoureteral reflux), prophylactic antibiotics may be used. Table 4 lists common prophylactic regimens.

Table 4 . Prophylactic Antibiotics Regimen.
Nitrofurantoin, 50 or 100 mg daily
Nitrofurantoin macrocrystals, 100 mg daily
TMP-SMX, 40/200 mg daily
Cephalexin, 250 mg daily
Ciprofloxacin, 250 mg daily
Trimethoprim, 100 mg daily

Table 5. Antibiotics That Require Dosage Adjustments for Liver and Renal Diseases.
Renal diseases (Creatinine clearance < 30 mL/min)
Aminoglycosides
-Lactams
Cefoxitin, Ceftizoxime
Cefonacid, Ceftazidime
Cefuroxime, Cefepime
Cepirome, Moxalactam
Carbenicillin, ticarcillin, ticarcillin-clavulanate
Vancomycin
Tetracycline (except doxycycline)
Sulfonamides
Hepatic diseases (with elevated bilirubin)
Chloramphenicol
Tetracyclines
Clindamycin, rifampin, pefloxacin
Renal-hepatic diseases
Ceftriaxone
Cefoperazone
Carbenicillin
Ticarcillin
Azlocillin
Mezlocillin
Piperacillin

Table 6. Recommended Antimicrobial Agents for Common Genitourinary Pathogens.		
Bacteria	**Oral Therapy**	**Parenteral Therapy**
Gram-positive cocci		
Staphylococcus aureus	Nafcillin, nitrofurantoin, ciprofloxacin	Nafcillin, vancomycin
Staphylococcus epidermidis	Ampicillin, nitrofurantoin, ciprofloxacin	Ampicillin, penicillin G
Staphylococcus saprophyticus	Ampicillin, nitrofurantoin, ciprofloxacin	Ampicillin, penicillin G
Streptococcus, group D		
S faecalis (enterococci)	Ampicillin, nitrofurantoin	Ampicillin plus gentamicin
S bovis	Penicillin G, ampicillin	Ampicillin, vancomycin
Streptococcus, group B	Ampicillin, cephalosporin	Ampicillin, cephalosporin
Gram-negative cocci		
Neisseria gonorrhoeae	Ciprofloxacin plus doxycycline	Ceftriaxone
Gram-negative rods		
Escherichia coli	TMP-SMX, ciprofloxacin, nitrofurantoin	Gentamicin
Enterobacter sp.	TMP-SMX, ciprofloxacin, nitrofurantoin	Gentamicin plus piperacillin
Gardnerella vaginalis	Metronidazole, ampicillin	Metronidazole
Klebsiella sp.	TMP-SMX, ciprofloxacin	Gentamicin plus cephalosporin
Proteus sp.	Ampicillin, TMP-SMX, ciprofloxacin	Ampicillin, gentamicin
Pseudomonas aeruginosa	Carbenicillin, tetracycline, ciprofloxacin	Gentamicin plus piperacillin
Serratia sp.	TMP-SMX, carbenicillin	TMP-SMX, amikacin
Other pathogens		
Chlamydiae	Tetracycline, erythromycin	Tetracycline, erythromycin
Mycoplasmas, ureaplasmas	Tetracycline, erythromycin	Tetracycline, erythromycin
Obligate anaerobes	Metronidazole, clindamycin	Metronidazole, clindamycin

TMP-SMX, trimethoprim plus sulfamethoxazole.

Table 7. Recommended Antimicrobial Agents and Duration of Therapy Based on the Type of UTI.			
Diagnosis	**Pathogen**	**Choice of Antibiotics**	**Duration of Therapy**
Cystitis	*E coli*	1st:TMP/SMX	1–3 days
	Klebsiella	2nd:Fluoroquinolone	
	Proteus		
Pyelonephritis	*E coli*	1st:Gluoroquinolone	7–10 days
	Proteus	2nd:2ndgeneration cephalosporin	
	Klebsiella	3rd: Aminopenicillin/BLI	
	Enterobacteria		
Complicated UTI	*E coli*	1st Fluoroquinolone	3–5 days
	Enterococci	2nd: Aminopenicillin/BLI	after afebrile
	Pseudomonas	3rd: 3rd generation cephalosporin	
	Staphylococci	Aminoglycosides	
Prostatitis	*E coli*	1st:Fluoroquinolone	Acute: 2 weeks
	Enterobacteria	2nd: 2nd generation cephalosporin	Chronic:
	Pseudomonas	3rd: 3rd generation cephalosporin	4–6 weeks
	Enterococci		
Epididymitis	*E coli*	1st: Fluoroquinolone	2 weeks
	Enterobacteria	2nd: 2nd generation cephalosporin	
	Enterococci		
	Chlmaydia	1st:Doxycycline	
	Ureaplasma	2nd:Macrolide	

BLI, beta-lactamase inhibitor; TMP/SMX, trimethoprim-sulfamethoxazole.(Adapted from table 2 from Wagenlehner and Naber, 2000.)

TMP-SMX, trimethoprim-sulfamethoxazole.

Trimethoprim-Sulfamethoxazole

Trimethoprim-sulfamethoxazole (TMP-SMX) is commonly used to treat many UTIs, except those caused by *Enterococcus* and *Pseudomonas* spp. It interferes with the bacterial metabolism of folate. Trimethoprim-sulfamethoxazole is highly effective and relatively inexpensive. Adverse reactions occur in 6–8% of patients using this medication; they include hypersensitivity reactions, rashes, gastrointestinal upset, leukopenia, thrombocytopenia, and photosensitivity. Trimethoprim-sulfamethoxazole should not be used in patients who have a folic acid deficiency state, glucose-6-phosphate dehydrogenase deficiency, or AIDS, or in pregnant patients. It is the most frequently prescribed antibiotic for uncomplicated UTI (Huang and Stafford, 2002). Recently, the use of TMP-SMX has declined due to the increased incidence of bacterial resistance (Brown et al, 2002) and physicians' preference for other newer antibiotics (Huang and Stafford, 2002).

Fluoroquinolones

Fluoroquinolones have a broad spectrum of activity, especially against gram-negative bacteria. Although they have adequate activity against *Staphylococci* species, fluoroquinolones do not have good activity against *Streptococci* species and anaerobic bacteria. They interfere with the bacterial DNA gyrase, preventing bacterial replication. Although they are highly effective in the treatment of UTI, fluoroquinolones are relative expensive. Adverse reactions are infrequent and include mild gastrointestinal effects, dizziness, and lightheadedness. Fluoroquinolones should not be used in patients who are pregnant and should be used judiciously in children because of potential damage to developing cartilage. Due to their broad spectrum of activity, fluoroquinolones have gained popularity in the empiric treatment of both uncomplicated and complicated UTIs (Schaeffer, 2002).

Nitrofurantoin

Nitrofurantoin has good activity against most gram-negative bacteria (except for *Pseudomonas* and *Proteus* spp.), *Staphylococci,* and *Enterococci* species. It inhibits bacterial enzymes and DNA activity. Nitrofurantoin is highly effective in the treatment of UTI and is relative inexpensive. Adverse reactions are relatively common and include gastrointestinal upset, peripheral polyneuropathy, and hepatotoxicity. Long-term use may result in pulmonary hypersensitivity reaction and interstitial changes. With increasing awareness of this antibiotic and its activity against common uropathogens, nitrofurantoin usage in the treatment of uncomplicated UTIs has increased from 14% to 30% in the past 5 years (Huang and Stafford, 2002).

Aminoglycosides

Aminoglycosides are commonly used in the treatment of complicated UTI. They are highly effective against most gram-negative bacteria. When combined with ampicillin, they are effective against enterococci. They inhibit bacterial DNA and RNA synthesis. The principal adverse effects of aminoglycosides are nephrotoxicity and ototoxicity. Aminoglycosides are primarily used in patients with complicated UTIs who require intravenous antibiotics (Santucci and Krieger, 2000). Aminoglycosides can be given as a single daily dosing; this regimen is directed toward obtaining higher peak and lower trough levels in order to achieve more effective microbial killing while reducing toxicity (Carapetis et al, 2001).

Cephalosporins

Cephalosporins have good activity against most uropathogens (Garcia-Rodriguez and Munoz Bellido, 2000). First-generation cephalosporins have good activity against gram-positive bacteria, *E coli,* and *Proteus* and *Klebsiella* spp. Second-generation cephalosporins have increased activity against anaerobes and *Haemophilus influenzae.* Third-generation cephalosporins have broader coverage against gram-negative bacteria but less against gram-positive bacteria. The cephalosporins inhibit bacterial cell wall synthesis. Adverse reactions include hypersensitivity and gastrointestinal upset. Oral cephalosporins have been used

effectively in the empiric treatment of uncomplicated UTIs (Lawrenson and Logie, 2001); in children with febrile UTI/pyelonephritis, oral third-generation cephalosporins such as cefixime have been shown to be safe and effective (Hoberman et al, 1999).

Penicillins

First-generation penicillins are ineffective against most uropathogens and are not commonly used in the treatment of UTI. However, the aminopenicillins (amoxicillin and ampicillin) have good activity against *Enterococci, Staphylococc*i, *E coli,* and *Proteus mirabilis*. However, gram-negative bacteria can quickly develop resistance to many aminopenicillins. The addition of -lactamase inhibitors such as clavulanic acid makes the aminopenicillins more active against the gram-negative bacteria. Although penicillins and aminopenicillins are inexpensive, the addition of the -lactamase inhibitors makes them more expensive. Adverse reactions include hypersensitivity (which can be immediate or delayed), gastrointestinal upset, and diarrhea. In general, penicillins are not commonly used in the treatment of UTI unless they are combined with -lactamase inhibitors (Sotto et al, 2001).

Clinical Presentation. Kidney Infection

Acute Pyelonephritis

Acute pyelonephritis is defined as inflammation of the kidney and renal pelvis, and its diagnosis is usually made clinically.

Presentation and Findings

Patients with acute pyelonephritis present with chills, fever, and costovertebral angle tenderness. They often have accompanying lower-tract symptoms such as dysuria, frequency, and urgency. Sepsis may occur, with 20–30% of all systemic sepsis resulting from a urine infection. Urinalysis commonly demonstrates the presence of WBCs and red blood cells in the urine. Leukocytosis, increased erythrocyte sedimentation, and elevated levels of C-reactive protein are commonly seen on blood analysis. Bacteria are cultured from the urine when the culture is obtained before antibiotic treatment is instituted. *E coli* is the most

common causative organism, accounting for 80% of the cases. *Klebsiella, Proteus, Enterobacter, Pseudomonas, Serratia,* and *Citrobacter* spp. account for the remaining cases. Of the gram-positive bacteria, *Streptococcus faecalis* and *S aureus* can be important causes of pyelonephritis.

Radiographic Imaging

Contrast-enhanced computed tomography (CT) scans can accurately demonstrate findings, confirming the diagnosis of pyelonephritis (Dacher et al, 1993). Acute bacterial infection causes constriction of peripheral arterioles and reduces perfusion of the affected renal segments. Perfusion defects, which can be segmental, multifocal, or diffuse, are seen as areas of reduced signal density (Figure 13–2). Renal enlargement, attenuated parenchyma, and a compressed collecting system are other characteristic findings on CT scan. However, CT scan is not necessary unless the diagnosis is unclear or the patient is not responding to therapy. Radionuclide study with ^{99m}Tc-dimercaptosuccinic acid is equally sensitive in detecting the perfusion defects of pyelonephritis (Levtchenko et al, 2001). In patients with acute pyelonephritis, renal ultrasonography is important to rule out concurrent urinary tract obstruction but cannot reliably detect inflammation or infection of the kidney

Figure

Acute pyelonephritis. Computed tomography scan with intravenous contrast demonstrates a perfusion defect (white arrow) and enlargement of the affected kidney.

Management

The management of acute pyelonephritis depends on the severity of the infection (Ghiro et al, 2002; Nickel, 2001). In patients who have toxicity because of associated septicemia, hospitalization is warranted. Empiric therapy with intravenous ampicillin and aminoglycosides is effective against a broad range of uropathogens, including enterococci and *Pseudomonas* species. Alternatively, amoxicillin with clavulanic acid or a third-generation cephalosporin can be used. Fever from acute pyelonephritis may persist for several days despite appropriate

therapy. Parenteral therapy should be maintained until the patient defervesces. If bacteremia is present, parenteral therapy should be continued for an additional 7–10 days and then the patient should be switched to oral treatment for 10–14 days. In patients who are not severely ill, outpatient treatment with oral antibiotics is appropriate. For adults, treatment with fluoroquinolones or TMP-SMX is well tolerated and effective. Therapy should continue for 10–14 days. Some patients in whom acute pyelonephritis develops will require follow-up radiologic examination such as voiding cystourethrogram or cystoscopy.

Emphysematous Pyelonephritis

Emphysematous pyelonephritis is a necrotizing infection characterized by the presence of gas within the renal parenchyma or perinephric tissue. Eighty to ninety percent of patients with emphysematous pyelonephritis have diabetes; the rest of the cases are associated with urinary tract obstruction from calculi or papillary necrosis (Shokeir et al, 1997).

Presentation and Findings

Patients with emphysematous pyelonephritis present with fever, flank pain, and vomiting that fails initial management with parenteral antibiotics (Tang et al, 2001). Pneumaturia may be present. Bacteria most frequently cultured from the urine include *E coli, Klebsiella pneumoniae,* and *Enterobacter cloacae.*

Radiographic Imaging

The diagnosis of emphysematous pyelonephritis is made after radiographic examination. Gas overlying the affected kidney may be seen on a plain abdominal radiograph (KUB). CT scan is much more sensitive in detecting the presence of gas in the renal parenchyma than renal ultrasonography.

Management

In the management of emphysematous pyelonephritis, prompt control of blood glucose and relief of urinary obstruction is essential, in addition to fluid resuscitation and parenteral antibiotics. The mortality rate is 11–54% (Michaeli et al, 1984). Poor prognostic factors include high serum creatinine level, low platelet

count, and the presence of renal/perirenal fluid in association with a bubbly/loculated gas pattern or gas in the collecting system (Wan et al, 1998). In combination with medical treatment, percutaneous drainage appears to be helpful in accelerating resolution of the infection and minimizing the morbidity and mortality of the infection (Chen et al, 1997). Nephrectomy may be required if there is no function in the affected kidney. Three to four weeks of parenteral antibiotic therapy is usually required.

Renal Abscesses

Renal abscesses result from a severe infection that leads to liquefaction of renal tissue; this area is subsequently sequestered, forming an abscess. They can rupture out into the perinephric space, forming perinephric abscesses. When the abscesses extend beyond the Gerota's fascia, paranephric abscesses develop. Historically, most renal/perinephric abscesses result from hematogenous spread of staphylococci, in particular from infected skin lesions. Patients with diabetes, those undergoing hemodialysis, or intravenous drug abusers were at high risk for developing renal abscesses. With the development of effective antibiotics and better management of diseases such as diabetes and renal failure, renal/perinephric abscesses due to gram-positive bacteria are less prevalent; those caused by *E coli* or *Proteus* species are becoming more common (Merimsky and Feldman, 1981; Thorley, Jones, and Sanford, 1974). Abscesses that form in the renal cortex are likely to arise from hematogenous spread, whereas those in the corticomedullary junction are caused from gram-negative bacteria in conjunction with some other underlying urinary tract abnormalities, such as stones or obstruction.

Presentation and Findings

The most common presenting symptoms in patients with renal/perinephric abscesses include fever, flank or abdominal pain, chills, and dysuria. Many of the symptoms have lasted for more than 2 weeks. A flank mass may be palpated in some patients. Urinalysis usually demonstrates WBCs; however, it may be normal in approximately 25% of the cases (Thorley, Jones, and Sanford, 1974). Urine

cultures only identify the causative organisms in about one-third of cases and blood cultures in only about half of cases (Edelstein and McCabe, 1988).

Radiographic Imaging

Renal abscesses can be accurately detected using ultrasonography or CT scans. There is a wide range of ultrasonographic findings ranging from an anechoic mass within or displacing the kidney to an echogenic fluid collection that tends to blend with the normally echogenic fat within Gerota's fascia (Corriere and Sandler, 1982). With high sensitivity, CT scans can demonstrate an enlarged kidney with focal areas of hypoattenuation early on during the course of the infection. Once the inflammatory wall forms around the fluid collection, the abscess appears as a mass with a rim of contrast enhancement, the "ring" sign. CT scans may also demonstrate thickening of Gerota's fascia, stranding of the perinephric fat, or obliteration of the surrounding soft-tissue planes (Dalla Palma, Pozzi-Mucelli, and Ene, 1999). Intravenous pyelogram and kidneys, ureter, and bladder tests are less sensitive tests in detecting renal/ perinephric abscesses, with results being normal in about 20% of the cases (Thorley et al, 1974).

Management

The appropriate management of renal abscess first must include appropriate antibiotic therapy. Because it is often very difficult to identify the correct causative organisms from the urine or blood, empiric therapy with broad-spectrum antibiotics (ampicillin or vancomycin in combination with an aminoglycoside or third-generation cephalosporin) is usually recommended. If the patient does not respond within 48 h of treatment, percutaneous drainage under CT or ultrasound guidance is indicated (Siegel, Smith, and Moldwin, 1996). The drained fluid should be cultured for the causative organisms

If the abscess still does not resolve, then open surgical drainage or nephrectomy may be necessary. Follow-up imaging is needed to confirm resolution of the abscesses. These patients will also require evaluation for underlying urinary tract abnormalities such as stone or obstruction after the infection has resolved.

Xanthogranulomatous Pyelonephritis

Xanthogranulomatous pyelonephritis (XGP) is a form of chronic bacterial infection of the kidney. The affected kidney is almost always hydronephrotic and obstructed. In most cases, XGP occurs unilaterally. Severe inflammation and necrosis obliterate the kidney parenchyma. Characteristically, foamy lipid-laden histiocytes (xanthoma cells) are present and may be mistaken for renal clear cell carcinoma (Iskandar, Prahlow, and White, 1993; Lorentzen and Nielsen, 1980).

Presentation and Findings

Patients with XGP commonly present with flank pain, fever, chills, and persistent bacteriuria. A history of urolithiasis is present in about 35% of the patients (Malek and Elder, 1978). On physical examination, a flank mass can often be palpated. Urinalysis commonly demonstrates WBCs and protein. Serum blood analysis reveals anemia and may show hepatic dysfunction in approximately 50% of the patients (Malek and Elder, 1978). Because XGP primarily occurs unilaterally, azotemia or renal failure is not often seen (Goodman et al, 1979). *E coli* or *Proteus* species are commonly cultured from the urine. However, one-third of patients with XGP have no growth in their urine, most likely because they have recently received antibiotic therapy. Approximately 10% of the patients with XGP have mixed organisms or anaerobic bacteria identified in their urine. Culture of the affected renal tissue can reliably identify the causative organism.

Radiographic Imaging

CT scan is the most reliable method in imaging patients suspected of having XGP. It usually demonstrates a large heterogeneous, reniform mass. The renal parenchyma is often marked with multiple water-density lesions, representing dilated calyces or abscesses (Figure 13–5 A–B) (Goldman et al, 1984). On contrast-enhanced images, these lesions will have a prominent blush peripherally, while the central areas, which are filled with pus and debris, do not enhance. An area of central calcification surrounded by a contracted pelvis may also be seen (Eastham, Ahlering, and Skinner, 1994). The inflammatory process may be seen extending to the perinephric fat, the retroperitoneum, and adjacent organs such as the psoas

muscle, spleen, colon, or the great vessels. Because of the association of urolithiasis and XGP, renal calculi may be seen (Parsons, 1993). Renal ultrasonography can also be used in performing imaging on patients with XGP (Tiu et al, 2001). It usually reveals an enlarged kidney with a large central echogenic area and anechoic parenchyma. However, ultrasonography does not provide comparable anatomic details to those obtained from CT scan. It is not uncommon for XGP to be misdiagnosed as a renal tumor because of their similar appearances on radiologic imaging (Zorzos et al, 2002).

Management

The management of XGP is dependent on accurate diagnosis. In some cases, XGP is misdiagnosed as a renal tumor. A nephrectomy is performed and a diagnosis is made pathologically. In those in whom a diagnosis of XGP is suspected, kidney-sparing surgery such as a partial nephrectomy is indicated. However, when the infection is extensive, a nephrectomy with excision of all involved tissue is warranted. There are reported cases of treating XGP with antibiotic therapy alone (Brown, Dodson, and Weintrub, 1996) or in combination with percutaneous drainage; however, these treatments are not likely to be curative in most patients and may lead to complications such as renal cutaneous fistula.

Chronic Pyelonephritis

Chronic pyelonephritis results from repeated renal infection, which leads to scarring, atrophy of the kidney, and subsequent renal insufficiency. The diagnosis is made by radiologic or pathologic examination rather than from clinical presentation.

Presentation and Findings

Many individuals with chronic pyelonephritis have no symptoms, but they may have a history of frequent UTIs. In children, there is a strong correlation between renal scarring and recurrent UTIs (Wennerstrom et al, 2000). The developing kidney appears to be very susceptible to damage, and this susceptibility appears to be age-dependent. Renal scarring induced by UTIs is rarely seen in adult kidneys. Because patients with chronic pyelonephritis often are asymptomatic, the

diagnosis is made incidentally when radiologic investigation is initiated to evaluate for the complications associated with renal insufficiency, such as hypertension, visual impairments, headaches, fatigue, and polyuria. In these patients, urinalysis may show leukocytes or proteinuria but is likely to be normal. Serum creatinine levels reflect the severity of the renal impairment. Urine cultures are only positive when there is an active infection.

Radiographic Imaging

Intravenous pyelogram or CT scan can readily demonstrate a small and atrophic kidney on the affected side. Focal coarse renal scarring with clubbing of the underlying calyx is characteristic. Ultrasonography similarly can demonstrate these findings. DMSA is the best imaging modality to look for renal scarring (Stoller and Kogan, 1986). Areas of scarring can be seen as photopenic areas.

Management

The management of chronic pyelonephritis is somewhat limited because renal damage incurred by chronic pyelonephritis is not reversible. Eliminating recurrent UTIs and identifying and correcting any underlying anatomic or functional urinary problems such as obstruction or urolithiasis can prevent further renal damage. In children, evaluation for vesicoureteral reflux with a voiding cystourethrogram is important to eliminate a risk factor for recurrent pyelonephritis and renal scarring. Long-term use of continuous prophylactic antibiotic therapy may be required to limit recurrent UTIs and renal scarring. Rarely, removal of the affected kidney may be necessary due to hypertension or having a large stone burden in a nonfunctioning kidney.

Chronic pyelonephritis, as a rule, is a I consequence of acute pyelonephritis. Of greatB value in diagnostics of chronic pyelonephritis I are laboratory, X-ray and tracer techniques of I research.

Leukocyturia is one of the most relevant and frequently occurring signs of chronic pyelonephritis.

An essential help in diagnostics of chronic pyelonephritis is rendered by X-ray method of testing. Main X-ray signs of the disease are: (1) changes of dimensions

and contours of the kidneys; (2) disturbance of excretion • by the kidney of radio-opaque matter; (3) I pathological values of renocortical index I (RCI); (4) deformations of the pyelocaliceal i system; (5) Hodson's symptom; (6) changes of I angioarchitectonics of the kidney.

On a survey urogram in chronic I pyelonephritis we find a decrease of the size of I one of the kidneys, a noticeable rising of density of a shadow and vertical position of the axis of I the affected kidney.

In excretory urography an X-ray pattern of I chronic pyelonephritis differs by polymorphism I and asymmetry of changes, which depend on the ratio of infiltrative-inflammatory and cicatricial processes. When infiltrative processes dominate, on urograms we find separation of calices, spastic stricture of their necks and pelvis. In cicatricial changes there are signs of the decrease of tone of calices, pelvis and the upper third of the ureter in the form of their moderate extension and the symptom of the edge of the lumbar muscle. At the place of contact of the pelvis and ureter with the edge of the lumbar muscle we observe as even flattening of their contour parallel to it. There are different deformations of calices: they gain a fungoid, mace-shaped form, become displaced, their necks are elongated and narrowed, the papillae are smoothed out.

Of tracer techniques of research in chronic pyelonephritis we apply isotope renography as a method of separate definition of renal function and establishing the side of the greatest affection. The method allows also to execute: a dynamic check over recovery of renal function in the process of treatment. To quantify and define the quality of functioning parenchyma it is expedient to apply dynamic scintigraphy. In segmental affection of the kidney dynamic scintigraphy reveals a delay of 99mTcDTPA transport in a zone of cicatricial changes. In pyelonephrotically contracted kidney static and dynamic scintigraphy allows to establish the size of the kidney, nature of drug accumulation and its distribution in it. Indirect renoangiography, thus, allows to determine the condition of renal blood supply and its recovery during treatmenIn patients with accompanying Hthogenesis (struvite stones), connected with infection with the microorganisms splitting the urea, such as

Proteus group, xanthogranulomatous pyelonephritis may develop. It is an atypical form of infectious affection of the kidneys in which upon microscopical research the occurrence of cellular infiltrates consisting of mononuclear macrophages laden with fat cells is typical. Macroscopic ally this disease looks as volumetric formation, which may be circumscribed or widespread, depending on a degree of gravity of the disease and volume of renal injury. More often this condition is mistaken for nephrocellular cancer.

Chronic pyelonephritis should be differentiated more often from nephrophthisis and glomerulonephritis. In favor of nephrophthisis the past history of tuberculosis of other organs, disuria, hematuria, cicatricial narrowing of the upper urinary paths, proteinuria, a less expressed dominance of leukocyturia over hematuria testify.

Reliable signs of nephrotuberculosis are: (1) presence of mycobacteria of tuberculosis in urine; (2) representative picture of tubercular affection of the urinary bladder at cystoscopy; (3) characteristic X-ray signs of the disease.

Chronic glomerulonephritis differs from pyelonephritis by predominance in urine of erythrocytes over leucocytes, glomerular type of proteinuria (infiltration of proteins with a high molecular weight into the urine), cylindruria, etc.

Treatment of chronic pyelonephritis should envisage the following main measures: (1) removal of causes producing the disturbance of urine passage or renal circulation, venous in particular; (2) administering antibacterial drugs in consideration of the data of antibiogram; (3) rising an immune reactivity of the organism.

Restoration of the outflow of urine should be reached, first of all, by applying this or that kind of surgical intervention (removing calculi from the kidneys and urinary tract, nephropexy, in case of nephroptosis, adenomectomy, plasty of the urethra or pyeloureteral segment, etc.). Xanthogranulomatous pyelonephritis is a surgical indication for partial nephrectomy or nephrectomy.

Without restoring the passage of urine to a great extent, the applying of antibacterial drugs usually does not give a prolonged remission of the disease.

The initial continuous course of antibacterial treatment makes up 6-8 weeks, since during this time it is necessary to achieve the suppression of the infectious agent in the kidney and destruction of purulent inflammatory process in it without complications, in order to prevent the formation of a cicatricial connective tissue. After achieving the stage of remission of the disease in the patient the antibacterial treatment should be prolonged by intermittent courses.

In the interval between the intakes of antibacterial drugs we prescribe cranberry juice 2-4 glasses per day, infusion of herbs possessing diuretic and antiseptic properties, Sodium Benzoatum and Methionine. Sodium Benzoatum and cranberry juice with Methionine increase synthesis of hippuric acid in the liver, which is being excreted with urine and has a strong bacteriostatic effect on causative agens of pyelonephritis. If the infection is resistant to antibacterial drugs, for the treatment of chronic pyelonephritis we apply large doses of Methionine (up to 6 g per day) with the purpose to create a sharply acid reaction of urine. As stimulating agent of a nonspecific immunological reactivity for patients with chronic pyelonephritis we apply Methyluracil or Pentoxyl within 1045 days monthly.

Sanatorium treatment of patients with chronic pyelonephritis is also carried out. Intake of low-mineralized waters boosts diuresis that promotes excretion of products of inflammation from the kidneys and urinary paths.

The prognosis in chronic pyelonephritios is in direct dependence on duration of the disease, activity of its course and frequency of repeated attacks of acute pyelonephritis. The prognosis is especially unfavorable if the disease starts in children's age due to the anomalies of the kidneys or urinary paths. Chronic pyelonephritis is the most often cause of chronic renal failure and nephrogenic arterial hypertension. The prognosis becomes particularly unfavourable in combination of both these complications.bacteriological research of urine is inoculation of its sediment into special culture media, possessing a selective sensitivity to the originator of tuberculosis. At present, inoculation on Lowenshtein's potato-egg medium and an accelerated inoculation on Price-Shkolnikova's blood medium are used. While the first method is applied the outcomes are received

within 2-2.5 months, the second — within 2-4 weeks, but the first method is more sensitive.

It is accepted to consider that the most sensitive test for Mycobacteria tuberculosis is biological: a hypodermic or intraperitoneal inoculation of the patient's urinary sediment to guinea pig — the animal possessing a high receptivity to the originator of tuberculosis.

More reliable information on functional disturbance in the kidneys in tuberculosis, including the earliest, is received due to tracer techniques of research (isotope renography, scanning and scintigraphy of the kidneys). Isotope renography does not reveal changes characteristic of nephrophthisis, however, if upon establishment of tuberculosis in one of the kidneys and finding functional disturbances in the opposite kidney, it is possible to suppose the presence of tuberculosis in it in the most initial, "subclinical" phase, which is not X-ray detected. Scintigraphy allows to judge the prevalence of destructive changes in the kidney, i. e., about the stage of the disease.

In the urinary bladder cystoscopy gains the relevant diagnostic value. Upon the appearance of specific tuberculous changes in the mucosa of the bladder the early phase of tuberculosis may cause edema , ulceration or polypi resembling a tumour on cystoscopy. Biopsy shows characteristic tubercles, giant cells and acid-fast bacilli. The detection in the urinary bladder of tuberculous ulcers, cicatrical retraction, deformation and hiatus of the ureter orifice confirms the diagnosis of renal tuberculosis and approximately indicates the side of the disease. More often these changes are localized in the circumference of the orifice of ureter of the affected kidney, or at any rate, in the corresponding half of the urinary bladder.

Treatment. Wide application of antituberculous drugs has marked a new epoch in complex treatment.

The purpose of chemotherapy of the patient with tuberculosis is the following: (1) suppression of reproductive duplication of Mycobacteria tuberculosis and prevention of their dissemination in the organism; (2) termination of bacteria excretion in order to prevent possible infection of healthy persons; (3) achievement

of a status of clinical treatment with a minimum of functional losses and residual changes; (4) prevention of palindromia. Medicamental treatment of patients with tuberculosis of the urinary system is represented by the complex of measures, including, besides a specific chemotherapy/ the basic method of treatment, also agents of pathogenetic influence, directed to normalization of the broken functions of the patient's organism, correction if undesirable effects of antituberculous drugs,prevention of posttuberculous cicatrization.

Chemotherapy of patients with tuberculosis of the urinary system consists in the combined, long and regular application of antituberculous nigs.

Thus, influence on the originator of the disease and tuberculous process is reached, (development of drug resistance is prevented, the Increase and maintenance of bacteriostatically Actlve drug concentration in the blood, urine Ind, that is especially important, in the foci of lesion are noted.

However, healing of tuberculous process ecurs only in correct treatment, i. e., in a rational chemotherapy. Success of antibacterial therapy is defined by conformity of the administered treatment with the form and phase of the process and with peculiarity of clinical manifestations of the disease in the patient.

Now the principle of two-phase chemotherapies has strongly come into clinical practice: more intensive antibacterial treatment with a daily, as a rule, introduction of drugs in the initial stage of the basic course and less saturated therapy in the subsequent stage, after a stable termination of specific bacteria excretion and before stabilization of tuberculosis.

All drugs are grouped in consideration of activity of their influence on Mycobacterium tuberculosis and clinical course of the disease.

Group A — Isoniazidum and Rifampicin and accordingly their pharmacological analogues are referred to this group.

Group B — Streptomycin, Kanamycin, Phlorimycin, Capriomycin, Biomycin, Ethambutol, Pyrazinamid, Quinolones, Prothionamid.

Group C — PAS (A) and Thioacetazone.

For the last 15 years Quinolones — chemotherapeutic drugs of a wide spectrum have taken a leading place among new antimicrobial agents. Quinolones have a very wide spectrum, of action, including all Gram-negative and Gram-positive aerobic bacteria, the majority of anaerobes. They are highly active in the infection of the urinary paths, intestine, in gonorrhea. They render bacteriostatic and bactericidal action on Mycobacterium tuberculosis of both typical and atypical forms located intra- and extracellularly. They have a long period of elimination and retention of activity that provides a prolonged action. Only 10% of drug is metabolized, basically it is excreted with urine in a pure state. They are administered only in combination with other antituberculous drugs in the dose of 400 mg (1-2 times per day within 14-28 days). The opportunity to administer low doses and a single dose is of great advantage. It is necessary to avoid ultraviolet radiation and staying in the sun during treatment with Quinolones and 3-4 days after its completion.

The prolonged antituberculous chemotherapy increases a risk of development of arterial hypertension. Pathogenetic hypotensive therapy with an individual selection of medicines and knowledge of mechanisms of changes in the vasopressor system in tuberculosis allows to lower or normalize the arterial pressure in more than 80% of patients.

Surgical treatment is an integral part of the general complex of medical measures in the patients with tuberculosis of the genitourinary system. Improvement in patients with tuberculosis of the kidneys and urinary paths observed in more than 60% of cases results in an operative measure. To achieve clinical treatment of patients is possible only in case of focal process in the kidney, called tuberculosis of the renal parenchyma, or in destruction of renal papillae — tuberculous papillitis.

Results of treatment are much worse in patients with circumscribed cavernous process and are especially adverse in specific lesion of the ureters and bladder.

Absolute indications to nephrectomy are pyonephrosis, polycavernous nephrophthisis with a decrease of renal function and retained bacteria excretion, despite a specific therapy not less than 12 months, secondary contracted kidney with

renal hypertension caused by it, cavernous nephrophthisis in combination with ulcerative urethritis and cystitis, when there is a substantial threat of development of contracted bladder.

Before operation it is necessary to count relative indications such as a total cement kidney, a non function ing kidney of small size without bacteria excretion, without exacerbation of pyelonephritis, hypertension and ulcerative tuberculous cystitis.

Now combined organ-preserving operations are successfully applied on the kidney, i. e., resection of a segment with subsequent cavernotomy or cavernoectomy made in one stage. The resection of two segments, top and bottom, is possible. The variant of intervention is defined by prevalence and localization of the process.

Organ-preserving operations are expedient only in patients with clinicoradiological signs of segmentary disconnecting a tuberculous focus or with the caverns having an apparent tendency to disconnection.

It is especially important to perform an operation when, despite an intensive and long enough (3-4 months) antibacterial therapy, the bacteria excretion is still preserved.

Indications to nephrectomy arise in the presence of a cavern or caverns communicated through a thin passage with the pelvis, in connection with disturbance of evacuation of contents from a cavern.

Cavernotomy is indicated in case of lesion in the medium segment of the kidney in connection with features of angioarchitecture in this part, and also if there are huge caverns of other segments of the kidneys. These changes lead to chronic disorders of circulation, compression of the parenchyma and in the final analysis to the development of nephrogenic hypertension. Cavernoectomy should be carried out in patients with caseous-necrotic changes and thick/ sclerosed wall of a cavern.

If the ureter is affected/ organ-preserving operations on the kidney are possible only, when a high-grade passage of urine is provided from the kidney. The internal splintage is carried out with the use of a catheter-stent and performance of plastic operation on the ureter before operation on the kidney that is the performance of

one-stage operation, on the kidney and ureter. Appearance and introduction into practice of such techniques as modelling on stents, baloon dilatation/ puncture nephrostomy allow to reduce quantity of operations concerning tuberculosis of the ureters and to lower percentage of complications.

Contraindications to organ-preserving operations are generalized processes retaining their activity and also accompanying somatic changes: blood diseases, lardaceous toxic nephritis, glomerulonephritis, diseases of the cardiovascular system in the stage of decompensation, neoplasms, etc.

In the performance of organ-preserving operations it is necessary to solve the following basic problems: to carry out a dissection of the affected parenchyma cuneiformly or on a plane; to make a dissection of a tissue under conditions of disconnection of renal circulation or without disconnection; what method is the safest and reliable to carry out hemostasis.

Cavernotomy and cavernoectomy are low traumatic. These operations, up to now, have not lost their value, and are performed particularly in patients with a widespread destructive tuberculosis of the kidneys. They allow maximally to keep a functioning parenchyma, and that is their advantage in comparison with nephrectomy

Variety of courses of tuberculosis of kidneys, its various localization and extents of kidney and urinary path damage have caused also a variety of surgical interventions. First of all, it concerns the ureter as it determines the destiny of an overlying kidney. Canker of mucosa of the ureter, its cicatrical narrowings break uropoiesis, lead to rising intrapelvic pressure, hemodynamic changes, expansion of the pyelocaliceal system. Strictures of the orifice of the ureter settle down, mainly, 1-10 cm above.

Tactics of treatment depends on the status of the kidney, its function reserves, character of the process in the ureter, as well as on the changes in the bladder. Three kinds of therapy, being frequently combined are probable: medicamental, endoscopic modelling on a catheter and surgical. All authors consider that in fresh processes, the so-called mucous urethritis with prevalence of edema and infiltration

of the mucous membrane, medicamental treatment is rather efficient and should be carried out according to the principles of modern antituberculous therapy.

Variants of operative measures on the ureter are various and depend on the level and extent of lesion. They can be divided into some kinds: anastomoses of the ureter with the bladder, various kinds of a scrappy plasty, interstitial plasty — replacement of a part or the whole ureter with an intestinal graft. Ureterocystoneostomy in stenoses of pelvic portion of the ureter and plasty. according to Boari or Demel are used more often. Technical performance of operations may be various. All of them are directed to the prevention of stenosis of anastomosis and development of vesicoureteral reflux.

Operative treatment of multiple stenoses of the ureter represents appreciable difficulties. The whole ureter or its part is replaced with a piece of a small bowel. Indications to the given kind of intervention being limited are irreversible bilateral lesions of the ureters or the ureter of solitary kidney.

In the true contracted bladder any kinds of therapy appear inefficient. The problem of possible and most efficient methods of drainage of urine remains disputable. As the evidence of this there is an existing variety of ways of drainage of urine in the contracted bladder. The majority of surgeons use the isolated fragment of the intestinal tract.

For replacement of the urinary path terminal and preterminal parts of the ileum, blind gut and sigmoid colon are serviceable. It is necessary to select the loop of the intestine physiologically closest to the changed piece of urinary paths. The twisted intestine is more applicable for replacement of the ureter as constant peristalsis actively transfers the contents in direction of the bladder. The sigmoid intestine surgical approach for reduction of volume of the J bladder, as it Is closer to it, is more preferable and | has a big capacity. The sigmoid colon is voided by consecutive tonic contractions arising when a disconnected loop is filled with urine.

The prognosis concerning convalescence in nephrophthisis is the most favourable in the absence of far advanced destructive changes in the renal parenchyma and cicatrical deformation of the urinary paths. The prognosis

nephrophthisis, that is associated with their surveyed as preoperative treatment or treatment 1 inferior vascularity. Besides, under the influence ex juvantibus. of Streptomycin and other drugs the cicatrical In tuberculosis of the epididymis usually I changes at the place of tuberculous foci in the its extirpation is made — epididymectomia, but 1 tissue of a testicular sinus and its epididymis in a partial affection of this organ its resection I develop, that leads to a disturbance of can be carried out. Similarly, at a total affection I permeability of the deferent paths and makes it of the testicle it is usually removed (orchiectomy) I inexpedient to preserve the affected epididymis. and at partial — resection of the testicle is

In tuberculosis of the prostate and seminal I antibacterial drugs of a wide spectrum of vesicles treatment is predominantly conservative. action (Penicillin, Gentomycin, Ampicillin, The exception represents a tubercular abscess in Biseptol, Kanamycin) in combination with these organs. Opening the abscess is executed glucocorticoids, immobilization of the scrotum through the anterior wall of the rectum or1 at sparing regimen. In case of intense pain through the perineum under the control of the novocaine blockade of the spermatic cord, cold forefinger, introduced into the rectum. First, one I are indicated. Epididymites of a nonspecific should make sure of the presence of abscess by etiology under the influence of nonspecific puncturing the site of the greatest ramollissement antibacterial therapy, as a rule, within 1-14 days with a needle. In empyema of the are subject to an appreciable involution, and seminal vesicles vesiculectomy is performed, differs from tuberculosis. Absence of positive dynamics after 2 weeks of chemotherapy conducted by courses gives reasons to suspect tuberculosis and to start antitubercular therapy with Streptomycin in combination with Isoniazidum and Ethambutol in routine doses. The inefficiency of antitubercular rnedicamental treatment within 30-40 days allows to offer the patient explorative diagnostics or operative treatment. First of all, these are patients, in whom the data of clinicolaboratory researches are insufficient for the reliable diagnosis of tuberculosis, and changes in the epididymis or testicle urgently demand clarity in the

diagnosis for the further treatment. If the diagnosis of tuberculosis does not cause doubt, treatment should begin at once with three antitubercular.

Patients with tuberculosis of male genital If there is a formation of abscess organs are subject to a prolonged observation in the scrotum, it is necessary to open and drain by phthisiourologist in the antituberculous it, and add to the treatment the introduction of clinic and systematic chemotherapy up to a Streptomycin or Kanamycin in the zone of the complete convalescence. Sanatorium treatment, abscess. Thus, rnedicamental treatment of acute climatotherapy, regimen and diet are of great epididymitis or orchiepididymitis should be significance.

Pyonephrosis

Pyonephrosis refers to bacterial infection of a hydronephrotic, obstructed kidney, which leads to suppurative destruction of the renal parenchyma and potential loss of renal function. Because of the extent of the infection and the presence of urinary obstruction, sepsis may rapidly ensue, requiring rapid diagnosis and management.

Presentation and Findings

Patients with pyonephrosis are usually very ill, with high fever, chills, and flank pain. Lower-tract symptoms are not usually present. Bacteriuria and pyuria may not be present when there is complete obstruction of the affected kidney.

Radiographic Imaging

Imaging with renal ultrasonography can be performed to rapidly diagnose pyonephrosis. Ultrasonographic findings include persistent echoes in the inferior portion of the collecting system, fluid-debris level with dependent echoes that shift with positional changes, strong echoes with acoustic shadowing from air in the collecting system, and weak echoes throughout a dilated collecting system. Renal or ureteral calculi may also be identified on ultrasonography.

Management

Management of pyonephrosis includes immediate institution of antibiotic therapy and drainage of the infected collecting system. Broad-spectrum

antimicrobials are indicated to prevent sepsis while the causative organism is being identified; antibiotics should be started before manipulation of the urinary tract. Performing drainage of the obstruction through the lower urinary tract (such as using a ureteral stent) should be reserved for patients who are not septic. Extensive manipulation may rapidly induce sepsis and toxemia. In the ill patient, drainage of the collecting system with a percutaneous nephrostomy tube is preferable. Once the infection is treated, additional imaging evaluation is required to identify the cause of the urinary obstruction, such as urolithiasis or ureteropelvic junction obstruction.

Bladder Infection

Acute Cystitis

Acute cystitis refers to urinary infection of the lower urinary tract, principally the bladder. Acute cystitis more commonly affects women than men. The primary mode of infection is ascending from the periurethral/ vaginal and fecal flora. The diagnosis is made clinically. In children, the distinction between upper and lower UTI is important. In general, those in whom acute cystitis developed do not usually require any extensive radiologic investigation (such as a voiding cystourethrogram), but those in whom pyelonephritis developed do (American Academy of Pediatrics, 1999).

Presentation and Findings

Patients with acute cystitis present with irritative voiding symptoms such as dysuria, frequency, and urgency. Low back and suprapubic pain, hematuria, and cloudy/foul-smelling urine are also common symptoms. Fever and systemic symptoms are rare. Typically, urinalysis demonstrates WBCs in the urine, and hematuria may be present. Urine culture is required to confirm the diagnosis and identify the causative organism. However, when the clinical picture and urinalysis are highly suggestive of the diagnosis of acute cystitis, urine culture may not be needed. *E coli* causes most of the acute cystitis. Other gram-negative (*Klebsiella* and *Proteus* spp.) and gram-positive (*Staphylococcus saprophyticus* and enterococci) bacteria are uncommon pathogens (Gupta et al, 1999).

Radiographic Imaging

In uncomplicated infection of the bladder, radiologic evaluation is often not necessary.

Management

Management for acute cystitis consists of a short course of oral antibiotics. Trimethoprim-sulfamethoxazole, nitrofurantoin, and fluoroquinolones have excellent activity against most pathogens that cause cystitis. Trimethoprim-sulfamethoxazole and nitrofurantoin are less expensive and thus are recommended for the treatment of uncomplicated cystitis (Huang and Stafford, 2002). However, it is estimated that resistance to TMP-SMX by *E coli* isolates causing uncomplicated acute cystitis is approximately 20%, compared to less than 2% to nitrofurantoin (Gupta, Scholes, and Stamm, 1999). In adults and children, the duration of treatment is usually limited to 3–5 days (Abrahamsson et al, 2002; Naber, 1999). Longer therapy is not indicated. Single-dose therapy for the treatment of recurrent cystitis/UTI appears to be less effective (Philbrick, 1986); however, fluoroquinolones with long half-lives (fleroxacin, pefloxacin, and rufloxacin) may be suitable for single-dose therapy (Naber, 1999). Resistance to penicillins and aminopenicillins is high and thus they are not recommended for treatment.

Recurrent Cystitis/UTI

Presentation and Findings

Recurrent cystitis/UTI is caused either by bacterial persistence or reinfection with another organism. Identification of the cause of the recurrent infection is important, because the management of bacterial persistence and reinfection are distinct. If bacterial persistence is the cause of recurrent UTI, the removal of the infected source is often curative, whereas preventative therapy is effective in treating reinfection.

Radiographic Imaging

When bacterial persistence is the suspected cause, radiologic imaging is indicated. Ultrasonography can be obtained to provide a screening evaluation of the

genitourinary tract. More detailed assessment with intravenous pyelogram, cystoscopy, and CT scans may occasionally be necessary. In patients who have frequent, recurrent UTI, bacterial localization studies and more extensive radiologic evaluation (such as retrograde pyelograms) is warranted. When bacterial reinfection is the suspected cause of recurrent cystitis, the patient should be carefully evaluated for evidence of vesicovaginal or vesicoenteric fistula. Otherwise, radiologic examination is often not necessary in these patients.

Management

Management of recurrent cystitis, again, depends on its cause. Surgical removal of the infected source (such as urinary calculi) is needed to treat bacterial persistence. Similarly, fistulas need to be repaired surgically to prevent bacterial reinfection. In most cases of bacterial reinfection, medical management with prophylactic antibiotics is indicated. Low-dose continuous prophylactic antibiotic has been shown to reduce the recurrences of UTI by 95% compared to placebo or historical controls (Mangiarotti, Pizzini, and Fanos, 2000; Nicolle and Ronald, 1987). Alternatively, intermittent self-start antibiotic therapy can be used in treating recurrent cystitis in some women. Motivated patients self-identify episodes of infection on the basis of their symptoms and treat themselves with a single dose of antibiotics such as TMP-SMX. This regimen has been shown to be effective and economical in selected patients (Pfau and Sacks, 1993; Raz et al, 1991). When the recurrent cystitis/UTI is related to sexual activity, frequent emptying of the bladder and a single dose of antibiotic taken after sexual intercourse can significantly reduce the incidence of recurrent infection (Pfau and Sacks, 1994). Alternatives to antibiotic therapy in the treatment of recurrent cystitis/UTI include intravaginal estriol (Raz and Stamm, 1993), lactobacillus vaginal suppositories (Reid and Burton, 2002), and cranberry juice taken orally (Lowe and Fagelman, 2001).

Malacoplakia

Malacoplakia is an uncommon inflammatory disease of the bladder that can also affect other parts of the urinary tract, including the ureters and kidneys (Stanton and Maxted, 1981). In the bladder, it manifests as plaques or nodules made of large

histiocytes (von Hansemann cells) with laminar inclusion bodies (Michaelis-Gutmann bodies) (McClure, Cameron, and Garrett, 1981).

Presentation and Findings

Malacoplakia more commonly affects women than men (Stanton and Maxted, 1981) and is associated with a history of UTI. Patients with malacoplakia often have chronic illness or are immunosuppressed. In patients with malacoplakia of the bladder, irritative voiding symptoms (urgency and frequency) and hematuria are common (Curran, 1987). When the disease affects the ureter or kidney, the patient may present with fever, flank pain, or flank mass. When it affects both kidneys, signs or symptoms of azotemia or renal failure may be present (Dobyan, Truong, and Eknoyan, 1993).

Radiologic Imaging

Radiologic imaging with ultrasonography or CT may demonstrate a mass in the bladder and evidence of obstruction if the disease extends to the ureter (Vas et al, 1985). When the disease involves the kidney, focal or diffuse, hypodense, parenchymal masses may be seen on CT imaging (Frederic, D'Hondt, and Potvliege, 1981). It is often difficult to distinguish malacoplakia from malignancy (transitional cell or renal cell carcinoma) with radiologic imaging. The diagnosis is often established after biopsy.

Management

Management of malacoplakia primarily consists of antibiotic therapy, in particular those that produce high intracellular levels. Consequently, TMP-SMX and fluoroquinolones are recommended in the treatment of malacoplakia. Bethanecol and ascorbic acid, which enhance phagolysosomal activity, may have some benefits (Stanton and Maxted, 1981; Trujillo-Santos et al, 1999). In patients with malacoplakia limited to the lower urinary tract, antibiotic therapy alone is usually sufficient. However, when malacoplakia involves the ureter or kidney, surgical excision may be needed in addition to the antibiotic therapy (Dasgupta et al, 1999; Long and Althausen, 1989). The prognosis is poor and the mortality rate is high in patients who have bilateral renal involvement, regardless of treatment.

URETHRITIS, PROSTATITIS, EPIDIDYMITIS. TUBERCULOSIS OF GENITOURINARY ORGANS.

Prostate Infection

Acute Bacterial Prostatitis

Acute bacterial prostatitis refers to inflammation of the prostate associated with a UTI. It is thought that infection results from ascending urethral infection or reflux of infected urine from the bladder into the prostatic ducts. In response to bacterial invasion, leukocytes (polymorphonuclear leukocytes, lymphocytes, plasma cells, and macrophages) are seen within and surrounding the acini of the prostate. Edema and hyperemia of the prostatic stroma frequently develop. With prolonged infection, variable degree of necrosis and abscess formation can occur.

Presentation and Findings

Acute bacterial prostatitis is uncommon in prepubertal boys but frequent affects adult men. It is the most common urologic diagnosis in men younger than 50 years (Collins et al, 1998). Patients with acute bacterial prostatitis usually present with an abrupt onset of constitutional (fever, chills, malaise, arthralgia, myalgia, lower back/rectal/perineal pain) and urinary symptoms (frequency, urgency, dysuria). They may also present with urinary retention due to swelling of the prostate. Digital rectal examination reveals tender, enlarged glands that are irregular and warm. Urinalysis usually demonstrates WBCs and occasionally hematuria. Serum blood analysis typically demonstrates leukocytosis. Prostate-specific antigen levels are often elevated. The diagnosis of prostatitis is made with microscopic examination and culture of the prostatic expressate and culture of urine obtained before and after prostate massage. In patients with acute prostatitis, fluid from the prostate massage often contains leukocytes with fat-laden macrophages. However, at the onset of acute prostatitis, prostatic massage is usually not suggested because the prostate is quite tender and the massage may lead to bacteremia. Similarly, urethral catheterization should be avoided. Culture of urine and prostate expressate usually identifies a single organisms, but occasionally, polymicrobial infection may occur. *E coli* is the most common causative organism in patients with acute

prostatitis. Other gram-negative bacteria (*Proteus, Klebsiella, Enterobacter, Pseudomonas,* and *Serratia* spp.) and enterococci are less frequent pathogens. Anaerobic and other gram-positive bacteria are rarely a cause of acute prostatitis (RO Roberts et al, 1997).

Radiologic Imaging

Radiologic imaging is rarely indicated in patients with acute prostatitis. Bladder ultrasonography may be useful in determining the amount of residual urine. Transrectal ultrasonography is only indicated in patients who do not respond to conventional therapy.

Management

Treatment with antibiotics is essential in the management of acute prostatitis. Empiric therapy directed against gram-negative bacteria and enterococci should be instituted immediately, while awaiting the culture results. Trimethoprim and fluoroquinolones have high drug penetration into prostatic tissue and are recommended for 4–6 weeks. The long duration of antibiotic treatment is to allow complete sterilization of the prostatic tissue to prevent complications such as chronic prostatitis and abscess formation (Childs, 1992; Nickel, 2000). Patients who have sepsis, are immunocompromised or in acute urinary retention, or have significant medical comorbidities would benefit from hospitalization and treatment with parenteral antibiotics. Ampicillin and an aminoglycoside provide effective therapy against both gram-negative bacteria and enterococci. Patients with urinary retention secondary to acute prostatitis should be managed with a suprapubic catheter because transurethral catheterization or instrumentation is contraindicated.

Chronic Bacterial Prostatitis

In contrast to the acute form, chronic bacterial prostatitis has a more insidious onset, characterized by relapsing, recurrent UTI caused by the persistence of pathogen in the prostatic fluid despite antibiotic therapy.

Presentation and Findings

Most patients with chronic bacterial prostatitis typically present with dysuria, urgency, frequency, nocturia, and low back/perineal pain. Others are asymptomatic,

but the diagnosis is made after investigation for bacteriuria. In patients with chronic bacterial prostatitis, digital rectal examination of the prostate is often normal; occasionally, tenderness, firmness, or prostatic calculi may be found on examination. Urinalysis demonstrates a variable degree of WBCs and bacteria in the urine, depending on the extent of the disease. Serum blood analysis normally does not show any evidence of leukocytosis. Prostate-specific antigen levels may be elevated. Diagnosis is made after identification of bacteria from prostate expressate or urine specimen after a prostatic massage, using the 4-cup test. The causative organisms are similar to those of acute bacterial prostatitis. It is currently believed that other gram-positive bacteria, *Mycoplasma*, *Ureaplasma*, and *Chlamydia* spp. are not causative pathogens in chronic bacterial prostatitis.

Radiologic Imaging

Radiologic imaging is rarely indicated in patients with chronic prostatitis. Transrectal ultrasonography is only indicated if a prostatic abscess is suspected.

Management

Antibiotic therapy is similar to that for acute bacterial prostatitis (Bjerklund Johansen et al, 1998). Interestingly, the presence of leukocytes or bacteria in the urine and prostatic massage does not predict antibiotic response in patients with chronic prostatitis (Nickel et al, 2001). In patients with chronic bacterial prostatitis, the duration of antibiotic therapy may be 3–4 months. Using fluoroquinolones, some patients may respond after 4–6 weeks of treatment. The addition of an alpha blocker to antibiotic therapy has been shown to reduce symptom recurrences (Barbalias, Nikiforidis, and Liatsikos, 1998). Despite maximal therapy, cure is not often achieved due to poor penetration of antibiotic into prostatic tissue and relative isolation of the bacterial foci within the prostate. When recurrent episodes of infection occur despite antibiotic therapy, suppressive antibiotic (TMP-SMX 1 single-strength tablet daily, nitrofurantoin 100 mg daily, or ciprofloxacin 250 mg daily) may be used (Meares, 1987). Transurethral resection of the prostate has been used to treat patients with refractory disease; however, the success rate has been

variable and this approach is not generally recommended (Barnes, Hadley, and O'Donoghue, 1982).

Granulomatous Prostatitis

Granulomatous prostatitis is an uncommon form of prostatitis. It can result from bacterial, viral, or fungal infection, the use of bacillus Calmette-Guérin therapy (Rischmann et al, 2000), malacoplakia, or systemic granulomatous diseases affecting the prostate. Two-thirds of the cases have no specific cause. There are two distinct forms of nonspecific granulomatous prostatitis: noneosinophilic and eosinophilic. The former represents an abnormal tissue response to extravasated prostatic fluid (O'Dea, Hunting, and Greene, 1977). The latter is a more severe, allergic response of the prostate to some unknown antigen.

Presentation and Findings

Patients with granulomatous prostatitis often present acutely, with fever, chills, and obstructive/irritative voiding symptoms. Some may present with urinary retention. Patients with eosinophilic granulomatous prostatitis are severely ill and have high fevers. Digital rectal examination in patients with granulomatous prostatitis demonstrates a hard, indurated, and fixed prostate, which is difficult to distinguish from prostate carcinoma. Urinalysis and culture do not show any evidence of bacterial infection. Serum blood analysis typically demonstrates leukocytosis; marked eosinophilia is often seen in patients with eosinophilic granulomatous prostatitis. The diagnosis is made after biopsy of the prostate.

Management

Some patients respond to antibiotic therapy, corticosteroids, and temporary bladder drainage. Those with eosinophilic granulomatous prostatitis dramatically response to corticosteroids (Ohkawa, Yamaguchi, and Kobayashi, 2001). Transurethral resection of the prostate may be required in patients who do not respond to treatment and have significant outlet obstruction.

Prostate Abscess

Most cases of prostatic abscess result from complications of acute bacterial prostatitis that were inadequately or inappropriately treated. Prostatic abscesses are

often seen in patients with diabetes; those receiving chronic dialysis; or patients who are immunocompromised, undergoing urethral instrumentation, or who have chronic indwelling catheters.

Presentation and Findings

Patients with prostatic abscess present with similar symptoms to those with acute bacterial prostatitis. Typically, these patients were treated for acute bacterial prostatitis previously and had a good initial response to treatment with antibiotics. However, their symptoms recurred during treatment, suggesting development of prostatic abscesses. On digital rectal examination, the prostate is usually tender and swollen. Fluctuance is only seen in 16% of patients with prostatic abscess (Weinberger et al, 1988).

Radiologic Imaging

Imaging with transrectal ultrasonography or pelvic CT scan is crucial for diagnosis and treatment.

Antibiotic therapy in conjunction with drainage of the abscess is required. Transrectal ultrasonography or CT scan can be used to direct transrectal drainage of the abscess (Barozzi et al, 1998). Transurethral resection and drainage may be required if transrectal drainage is inadequate. When properly diagnosed and treated, most cases of prostatic abscess resolve without significant sequelae (Weinberger et al).

Urethritis

Types of Urethritis

Infection/inflammation of the urethra can be categorized into those types caused by *Neisseria gonorrhoeae* and by other organisms (*Chlamydia trachomatis*, *Ureaplasma urealyticum*, *Trichomonas vaginalis*, and herpes simplex virus) (Dixon, Pearson, and Clutterbuck, 2002). Most cases are acquired during sexual intercourse.

Presentation and Findings

Patients with urethritis may present with urethral discharge and dysuria. The amount of discharge may vary significantly, from profuse to scant amounts. Obstructive voiding symptoms are primarily present in patients with recurrent

infection, in whom urethral strictures subsequently develop. It is important to note that approximately 40% of patients with gonococcal urethritis are asymptomatic (John and Donald, 1978). The diagnosis is made from examination and culture of the urethra. It is important to obtain the specimen from within the urethra, rather than from just the discharge. Approximately 30% of men infected with *N gonorrhoeae* will have concomitant infection with *C trachomatis.*

Radiologic Imaging

Retrograde urethrogram is only indicated in patients with recurrent infection and obstructive voiding symptoms. Most patients with uncomplicated urethritis do not require any radiologic imaging.

Management

Pathogen-direct antibiotic therapy is required. In patients with gonococcal urethritis, ceftriaxone (250 mg intramuscularly) or fluoroquinolones (ciprofloxacin 250 mg) (David, Wildman, and Rajamanoharan, 2000) or norfloxacin 800 mg) may be used. For patients with nongonococcal urethritis, treatment is with tetracycline or erythromycin (500 mg 4 times daily) or doxycycline (100 mg twice daily) for 7–14 days (O'Mahony, 1999). However, the most essential component of treatment is prevention. Sexual partners of the affected patients should be treated, and protective sexual practices (such as using condoms) are recommended.

Epididymitis

Causes of Epididymitis

Infection and inflammation of the epididymis most often result from an ascending infection from the lower urinary tract. Most cases of epididymitis in men younger than 35 years are due to sexually transmitted organisms (*N gonorrhoeae* and *C trachomatis*); those in children and older men are due to urinary pathogens such as *E coli.* In homosexual men who practice anal intercourse, *E coli* and other coliform bacteria are common causative organisms. The infection in the epididymis may spread to involve the testis.

Presentation and Findings

Patients with epididymitis present with severe scrotal pain that may radiate to the groin or flank. Scrotal enlargement due to the inflammation of the epididymis/ testis or a reactive hydrocele may develop rapidly. Other symptoms of urethritis, cystitis, or prostatitis may be present before or concurrent with the onset of scrotal pain. On physical examination, an enlarged and red scrotum is present, and it is often difficult to distinguish the epididymis from the testis during the acute infection. A thickened spermatic cord can occasionally be palpated. Urinalysis typically demonstrates WBCs and bacteria in the urine or urethral discharge. Serum blood analysis often reveals leukocytosis.

Radiologic Imaging

Frequently, it is difficult to distinguish epididymitis from acute testicular torsion based on the history and physical examination alone (Petrack and Hafeez, 1992). Scrotal Doppler ultrasonography or radionuclide scanning can be used to confirm the diagnosis (Paltiel et al, 1998). The presence of blood flow in the testis on Doppler ultrasonography or uptake of the tracers into the center of the testis on radionuclide scanning rules out torsion. On scrotal ultrasonography, patients with epididymitis commonly have an enlarged epididymis with increased blood flow. A reactive hydrocele or testicular involvement may also be seen. Prepubertal children who are diagnosed with epididymitis will require radiologic investigation for urinary tract anomalies such as reflux or ureteral ectopia (Likitnukul et al, 1987). Postpubertal children who are diagnosed with epididymitis should be educated about sexually transmitted diseases and safe sexual practices.

Management

Oral antibiotic treatment is directed against specific causative organisms, as mentioned in the previous sections on urethritis and UTI. In addition, bed rest, scrotal elevation, and the use of nonsteroidal anti-inflammatory agents are helpful in reducing the duration of the symptoms. In patients with epididymitis caused by sexually transmitted organisms, treatment of their sexual partners is recommended to prevent reinfection. For patients with sepsis or severe infection, hospitalization

and parenteral antibiotic therapy may be needed. Open drainage is indicated in cases in which an abscess develops. Occasionally, patients with chronic, relapsing epididymitis and scrotal pain may require epididymectomy for relief of their symptoms.

Special Circumstances

Urinary Tract Infection Related to Pregnancy

With pregnancy, there are anatomic and physiologic changes to the urinary tract due to compression by the gravid uterus and alterations in the hormonal milieu. Renal length increases approximately by 1 cm during normal pregnancy as a result of increased vascular and interstitial volume (Waltzer, 1981). The glomerular filtration rate increases by 30–50%, most likely secondary to the increase in cardiac output (Waltzer, 1981). Typically, there is significant ureteral dilation with resultant urinary stasis during the second and third trimesters of gestation. This hydroureter is attributed to the smooth muscle–relaxing effects of progesterone and the mechanical compression of the ureters by the uterus at the level of the pelvic brim (Waltzer, 1981). The bladder is also affected, both physically and physiologically. The enlarged uterus displaces the bladder superiorly and anteriorly. The bladder becomes hyperemic, and its capacity is increased, most likely due to the effects of progesterone (Waltzer, 1981).

Because of these changes in the urinary tract during normal pregnancy, bacteriuria is a clinically relevant finding in pregnant women. It is estimated that the prevalence of bacteriuria is 4–6% (Sweet, 1977), which is not significantly different from that in nonpregnant women of comparable age. Interestingly, approximately 30% of those who have bacteriuria on screening evaluation later have pyelonephritis, compared to only 1–2% in those who do not have bacteriuria (Sweet, 1977). Treatment of bacteriuria decreases the incidence of pyelonephritis during pregnancy to approximately 3%. Overall, the incidence of acute bacterial pyelonephritis is 1–4% in pregnant women (Gilstrap, Cunningham, and Whalley, 1981; Wing, 1998). Sixty to seventy percent of the episodes of pyelonephritis occur during the second and third trimesters of pregnancy, when urinary stasis is the

greatest. In 10–20%, recurrent episodes of pyelonephritis develop before delivery (Gilstrap, Cunningham, and Whalley, 1981). Significant maternal risk factors include diabetes and history of UTI. When left untreated, pyelonephritis during pregnancy is associated with a high rate of infant prematurity and its associated perinatal mortality (Locksmith and Duff, 2001; McGregor and French, 1998; Schieve et al, 1994). It remains unclear whether treated pyelonephritis during pregnancy has any effects on the developing fetus (Gilstrap and Ramin, 2001).

Consequently, it is recommended that women be screened for bacteriuria during pregnancy to prevent the development of pyelonephritis. A voided urine specimen should be obtained at the first prenatal visit and at 16 weeks of gestation (Stenqvist et al, 1989). For asymptomatic individuals, significant bacteriuria is defined as 2 voided urine cultures with greater than 10^5 CFU/mL of a single organism. For symptomatic pregnant women, greater than 10^3 CFU/mL is considered to be significant (Rubin, Beam, and Stamm, 1992). Pregnant women who are found to have bacteriuria should be treated with penicillins, oral cephalosporins (Christensen, 2000; Wing et al, 1999), or fosfomycin trometamol (Minassian et al, 1998). Table 13–9 lists the antibiotics and their effects on pregnancy. However, amoxicillin is not recommended because of the rate of bacterial resistance (Hart et al, 2001). A 3-day course is suggested, although single-dose therapy may be effective in some patients (Tincello and Richmond, 1998). Repeat urine culture to document eradication of bacteriuria is necessary in all patients. Patients with acute bacterial pyelonephritis should be treated with parenteral cephalosporins, penicillins with -lactamase inhibitors, or monolactams (Rubin, Beam, and Stamm, 1992). Periodic surveillance urine culture is recommended because many of these women will have recurrent episodes of pyelonephritis.

UTI in Patients with Human Immunodeficiency Virus or Acquired Immunodeficiency Syndrome

Human immunodeficiency virus alters the normal host defense against bacterial infection. When the CD4 lymphocyte count falls to less than 200/mm^3, the

risk of bacterial and opportunistic UTI increases dramatically (Evans et al, 1995; Hoepelman et al, 1992). In addition, antiretroviral medications used to treat HIV (eg, zidovudine) can further suppress normal immune response and increase the risk of UTI in these patients.

UTI/Cystitis

Hoepelman et al (1992) obtained urine cultures from HIV-positive men prospectively and when they had symptoms suggestive of a UTI. They observed that positive urine cultures were identified in 30% of HIV-infected men with CD4 < 200/mm^3 and in 11% with CD4 = 200–500/mm^3, while none with CD4 > 500/mm^3 had evidence of a urine infection. Gugino et al (1998) similarly observed that the incidence of bacteriuria in asymptomatic HIV-infected women was the same as that in uninfected women. Causative organisms include common uropathogens such as *E coli* and *Klebsiella and Enterococcus* spp. Urinary infection with *S aureus* and *Pseudomonas aeruginosa* is more common in HIV-infected patients (Schonwald, Begovac, and Skerk, 1999). Because of the common prophylactic use of TMP-SMX to prevent *Pneumocystis carinii* pneumonia in AIDS patients, the incidence of UTI in this group is decreased. However, when a UTI develops in these patients, the infecting organism is typically resistant to TMP-SMX (van Dooyeweert et al, 1996).

Prostatitis

In HIV patients, the incidence of bacterial prostatitis is approximately 3% and is 14% in patients with AIDS, compared to 1–2% in noninfected men of similar age (Leport et al, 1989). Causative organisms include common prostatitis pathogens such as *E coli* and *Proteus* spp. and other less common organisms such as *Salmonella typhi, S aureus, P aeruginosa, and N gonorrhoeae* (Staiman and Lowe, 1995). Prolonged treatment (4–6 weeks) with fluoroquinolones may be necessary because of a high risk of reinfection and lower immunity status in these patients. Prostatic abscess is more common in patients with AIDS compared to that in the general population (Staiman and Lowe, 1995; Trauzzi et al, 1994). Causative organisms include *E coli* and other gram-negative bacteria or opportunistic fungus

or mycobacterial infection (Lee, Dinneen, and Ahmad, 2001). Effective drainage and prolonged antimicrobial or antifungal therapy are needed.

Epididymitis and Urethritis

In HIV-infected men, epididymitis may be caused by *N gonorrhoeae* and *C trachomatis.* However, infection by coliform bacteria such as *E coli* is more common, especially in patients having unprotected anal intercourse (Berger et al, 1987). In HIV-infected patients with suppurative or antibiotic-resistant epididymitis, infection with fungi or mycobacteria should be considered (Desmond et al, 1993). In HIV-infected men who present with urethritis, treatment for both *Chlamydia* and *N gonorrhoeae* is indicated even when gonococcus is only isolated from culture. Due to increased viral shedding with genital infections, it is recommended that HIV-infected patients abstain from sexual intercourse until 7 days after treatment is completed.

Infection by Uncommon Organisms

Urinary infection with *Mycobacterium* species can develop in HIV-infected patients. The kidneys are first infected and the infection spreads to the lower urinary tract. In patients with AIDS, it is estimated that 6–23% have renal tuberculosis (Marques et al, 1996). *M tuberculosis* is the most common pathogen, with *M avium* and *M intracellulare* being less common (Sepkowitz et al, 1995). In HIV-infected patients who present with irritative/obstructive voiding symptoms but have no evidence of bacterial infection on culture, infection of the lower urinary tract by *Mycobacterium* species should be considered. Treatment with at least 2 antituberculosis agents is needed for 6–9 months.

TUBERCULOSIS OF GENITOURINARY ORGANS

We can distinguish two principally different forms of tuberculosis of genitourinary organs: acute and chronic. The acute form has no independent clinical value: it represents miliary tuberculosis arising simultaneously with miliary tuberculosis of other organs by dissemination of the process. In recent years the acute form of tuberculosis of genitourinary organs is marked extremely seldom in connection with modern capabilities of a specific chemotherapy and prophylaxis of generalized tuberculosis.In their practical activities the doctors, as a rule, encounter "with the chronic form of tuberculosis of genitourinary organs, which has its own clinical picture and is an independent nosological form.

Tuberculosis of genitourinary organs is secondary, the so-called organic tuberculosis. It differs from tuberculosis of the majority of other organs since it represents comparatively later occurrence of tuberculous illness of the organism. The disease develops in the definite period after the patients suffered from tuberculosis of other organs, and in recent years this interval has considerably increased. In this connection the average age of patients with tuberculosis of genitourinary organs is enlarged. The disease can be isolated, i. e., not accompanied by clinical manifestations of tuberculosis of other organs, or combined with tuberculous affection of the lungs, osteoarticular system, etc.

Tuberculosis of Renal and Urinary System

The etiology of tuberculosis of organs of the urinary system, as well as tuberculosis of other organs, is well known: it is caused by a specific originator — Mycobacterium tuberculosis.

The basis of suggested classifications of tuberculosis of the kidneys and urinary paths is the pathomorphologic clinical course of tuberculosis: from the initial, infiltrating changes in the depth of the renal parenchyma to the development of polycavernous nephrophthisis, tuberculous pyonephrosis. The most convenient and simple is a tetrastage clinical and roentgenological classification of

nephrophthisis, the basis of which is a single criterion — a degree of the renal tissue destruction.

stage — not destructive (infiltrative) nephrophthisis.

stage — initial destruction: papillitis or small (no more than 1 cm in diameter) single caverns.

stage — restricted destruction: a cavern of large size or polycavernous tuberculosis in one of the segments of the kidney.

stage — total or subtotal destruction (polycavernous tuberculosis of two segments, tuberculous pyonephrosis, atrophy and calcification of the kidney).

There are no pathognomonic clinical signs of nephrophthisis and urinary paths that makes the identification of this disease considerably difficult. Frequently, at the beginning it proceeds clinically under the mask of other urological diseases (chronic pyelonephritis, nephrolithiasis, tumour of the kidney, etc.), less often — it is asymptomatic at all. When the process spreads to the urinary bladder, the disorders of emiction are usually estimated as signs of nonspecific cystitis.

Of the first subjective clinical manifestations of the disease the most frequent now are nephralgias, more often dull ache, but sometimes acute, like that of renal colic, which is usually taken for the sign of nephrolithiasis. Dysuria is in the second place by frequency. Dysuria in nephrophthisis poorly responds to nonspecific therapy, fast recurs and progresses. In the third place by frequency, among the first clinical manifestations of nephrophthisis it is possible to place hematuria, more often not accompanied by any other signs. Such hematuria, first of all suggests the idea of tumour of the kidney.

Besides, nephrophthisis can clinically proceed under a mask of other diseases, quite often it is combined with them. Very often, as it was already indicated, nephrophthisis is accompanied by chronic nonspecific pyelonephritis, A combination of nephrophthisis with nephrolithiasis is quite often, observed, therefore, if there are symptoms of pyelonephritis and nephrolithiasis it is necessary to remember the possibility of simultaneous presence of tuberculosis. The past

history of tuberculosis of the lungs, lymphatic nodes, bones and joints, exudative pleuritis, etc., substantially reinforces the suspicion of nephrophthisis.

A body temperature for the majority of patients is normal. The temperature rise more often does not fall outside the limits of subfebrile values, but sometimes can accept hectic nature, being accompanied by chill and resembling the attack of acute pyelonephritis. The arterial hypertension comparatively seldom may be encountered as a symptom of nephrophthisis, mainly, in the far advanced two-sided process or affection of solitary kidney. More often local objective signs (morbidness upon deep palpation in the field of the kidney, palpated kidney, positive Pasternatsky's sign) are observed, however, they are not pathognomic for nephrophthisis; of the relevant value is palpation of the external genital organs, prostate and seminal vesicles, as nephrophthisis is watched approximately in half of men suffering from genital tuberculosis.

Of the objective signs of nephrophthisis and urinary paths, changes of urine are the most typical of this disease. The most common sign is pyuria (leukocyturia). This sign is encountered in the uncured common of kidneys almost for 100% patients. Persistent leukocyturia, which does not respond to nonspecific antibacterial therapy, is especially characteristic of nephrophthisis. Rather often a laboratory sign of nephrophthisis is also proteinuria. Characteristic of this disease is that the contents of protein in urine, as a rule, does not exceed 1 g/1 and protein is not found without presence of cellular elements in urinary sediment. It allows to consider proteinuria in nephrophthisis as "false", i. e., extrarenal. Sometimes erythrocyturia proves to be an earlier manifestation of this disease than leukocyturia. In such cases the disease, at first, proceeds under the guise of chronic nephritis.

An important objective sign of nephrophthisis and tuberculosis of the urinary tract is a characteristic picture of the pyelocaliceal system, ureter and urinary bladder on roentgenograms.

In the initial not destructive stage of the process it is eformation developed in the depth of the renal parenchyma at the expense of the infiltrate of the calices and pelvis (prelum or forcing them back, narrowing of the necks of calices and

expansion of calices). In the starting destruction of the parenchyma we observe the corrosion of contours of the renal papillae and

The narrowings of the ureter, quite often being multiple, are characteristic of tuberculosis of the ureter. They are localized more often in pyeloureteral segment, in the upper third of the ureter and especially frequently in its pelvic portion. In the far advanced tuberculosis of the ureter its multiple strictures give it a characteristic picture of the so-called bead-like ureter pelvic curvature is also representative of tuberculosis. Its decreases in size/ deformation, slant are characteristic of tuberculous lesion of the urinary bladder.

Urogenital tuberculosis

Urogenital tuberculosis can rightfully be classified as the most common pathology of the genitourinary system. This is evidenced by statistical data of both domestic and foreign authors. Tuberculosis of the kidneys makes up 13-15 percent of the total number of all surgical diseases of the upper urinary tract, second in frequency only to kidney stone disease, and tuberculosis of the external genitalia in men - 20-25 percent of diseases of the scrotum.

Modern methods of X-ray examination make it possible to recognize tuberculosis of the genital organs in the early stages of its development, and thus to start rational treatment in a timely manner.

Gender, age, side of lesion. Tuberculosis of the kidney occurs with the same frequency in persons of both sexes. It usually affects people of younger and blooming age. The largest number of patients occurs between 20 and 40 years of age. It is generally accepted that it occurs less frequently in children under 10 years of age and in the elderly. According to the literature, it is mostly impossible to establish kidney damage on one or the other side. Bilateral kidney damage was noted by a number of authors in 33-35 percent of cases.

Pathogenesis. According to many authors, kidney tuberculosis is a secondary process in the body and it arises as a result of hematogenous metastasis from the existing primary complex in the lungs, intestines or lymph nodes. Thus,

kidney tuberculosis is considered as a local manifestation of a general tuberculosis infection in the body.

Tuberculosis mycobacteria, as a rule, penetrate into the kidney by a hematogenous route, without causing a liquid lymphogenic route from adjacent organs affected by tuberculosis (intestine, mesenteric lymph nodes, spine).

However, in many patients with kidney tuberculosis, during a difficult clinical examination, it is not possible to establish a specific process in other organs from which hematogenous transmission of the infection was possible.

BY. Lebedeva (1952), A.I. Myants (1954), A.N. Chystovich (1960) recognize that tuberculosis mycobacteria can enter the kidney simultaneously with lung damage or from the lymph nodes during primary infection of the body. However, while tuberculosis mycobacteria find favorable conditions for their development in the lungs or intestine, these conditions are less favorable in the kidney parenchyma, where special sensitization of the kidney tissue, its susceptibility, is necessary for their vital activity.

According to A.I. Mayantsa, this sensitization sometimes occurs 3-10 years after the initial clinical manifestation of tuberculosis in the body. As soon as the infectiousness of tuberculosis was proven, features were found that distinguish it from other infectious diseases.

It was established that the nature of the course and morphological manifestations of TB in many cases depend on how the tubercle bacillus first penetrated the body, or the process takes place in an organism that has already been exposed to tuberculosis infection. Depending on this, 2 periods were distinguished in the development of TB: 1) the primary complex 2) the result of reinfection.

In 90-95 percent, the primary infection is through the respiratory tract.

The main feature of primary tuberculosis is its development against the background of active foci of primary infection - a constant source of sensitization of the body.

The primary focus is the source of hematogenous tuberculosis. It is characterized by the presence of foci in various organs and is characterized by

increased activity of the body. At the same time, the occurrence of a focus - metastasis in the kidney and the development of a progressive process in them do not coincide in time: metastasis occurs in childhood, and the development of tuberculosis in the kidney begins after 10-15-20 years, when tuberculosis is no longer present in other organs.

Hematogenous involvement in the kidney usually occurs during bacellemia. Sometimes this happens when a tuberculosis focus is aggravated in any location, in other cases new tuberculosis foci appear. The body as a whole and its individual organs are sensitized to tuberculosis infection. Clinical studies have established that hematogenous transfer of tuberculosis infection to the kidney occurs in 5-14 percent of pulmonary tuberculosis, and in 2-12.7 percent of bone tuberculosis. These data should be taken into account during preventive examinations of the population.

More recently, some studies have recognized the possibility of the infection entering the kidney by an ascending route (urinogenically) from the bladder affected by tuberculosis. Today, this way of tuberculosis infection entering the kidney is considered a rare phenomenon. It is possible only in some cases as a result of backflow of urine infected with mycobacterium tuberculosis and bladder into the ureter with spasmodic contractions of the bladder and insufficiency of the vesical mouths of the ureters due to their specific lesions. This phenomenon was called vesicoureteral reflux. The upward path of spread of tuberculous infection from the affected bladder to the kidney is possible, both with a banal infection through the opening of the ureter, and through the lymphatic pathways located in the tissue surrounding it.

The penetration of mycobacterium tuberculosis into the parenchyma of the kidney does not always cause the development of a specific process in it. The nature of the changes that occur depends on the degree of natural or acquired immunity from these conditions, the following options for the development of the pathological process in the kidney are possible: a) tuberculosis mycobacteria that have penetrated the kidney are reduced by the body's protective forces and die; b) tuberculosis mycobacteria in the kidney in a latent state, without causing any deviations from the

parenchyma; c) tuberculosis mycobacteria find favorable conditions for their life activity, as a result of which a specific tubercular process develops in the kidney.

Until recently, there was an opinion of the authors that in most patients, the tuberculous process first affects one kidney and that the simultaneous impression of both kidneys is noted only in 15-20 percent (R.M. Fronshtein, I.M. Epshtein, etc.). However, today such a point of view is denied by many authors. So A.I. Mayants and A.N. Chistovych and others. it is believed that in most patients with tuberculosis, two kidneys are affected at the same time, but the degree of damage in each of them is not equally intense. In one of the kidneys, the tuberculous process is in a latent state and is not clinically noted, and in the future it is even eliminated, while in the other kidney, a blooming focus develops with the progression of the process. The validity of these statements is confirmed in practice - in the rapid outbreak of a tubercular process in a number of patients in the other kidney after nephrectomy, although the previous clinical examination of this kidney before the operation did not establish a tubercular process in it.

Veselovskyi (1955) believes that all modern diagnostic methods are not sufficient to assert that a kidney considered healthy is not affected by tuberculosis and that renal tuberculosis in its initial form is always bilateral.

For a long time, there was an almost unanimous opinion, however, that the hematogenous deposition of a specific embolus carried into the kidney occurs in the medullary layer at the top of the papillae, since the most pronounced specific changes in the parenchyma of the removed kidney are found in this layer. However, at the autopsy of the corpses of patients who died from pulmonary tuberculosis, the main mass of tubercles in the cortical layer of the kidneys and, to a lesser extent, in the medullary layer, were found in the early stages of the development of a specific process in the kidneys. In later periods, specific changes prevail in the brain layer.

The tuberculous process developing in the parenchyma of the kidney, during its spread, reaches the tops of the cups, and, undergoing disintegration, breaks into the lumen of the urinary tract. There is a permanent connection of the focus with the cups, bowl and bladder.

Tuberculosis infection of bowls and cups, according to rich authors, takes place through contact. A.I. Mayants does not rule out the spread of tubercle bacilli through lymphatic vessels located in the submucosal layer of the pelvis or ureter.

With further progression, the tuberculous process spreads to the urinary bladder, which is affected by tuberculosis only with a long process in the kidneys, from where it spreads downward.

In the presence of a tuberculous process in the urinary bladder, significantly pronounced destructive changes are usually noted in the parenchyma of the kidneys. However, in some cases, when a widespread ulcerative process of the mucous membrane of the urinary bladder is detected, only a specific infiltrate without signs of decay is detected in the affected kidney.

For a long time, the bladder remains intact until tuberculosis infection. According to V.D. Ground, such a condition is explained by the fatigue of the nerve trunks passing through its wall.

Pathological anatomy. Touching on the acute miliary form of renal tuberculosis, it should be said that it is clinically unrecognizable and can be detected only on section. Therefore, chronic tuberculosis of the kidney should be discussed in more detail.

When mycobacterium tuberculosis settles in the parenchyma of the kidney, the presence of the necessary allergic state of the body and, in particular, sensitization of the renal parenchyma, the tuberculous process (tuberculosis nodule) begins to develop.

Further, diverse dynamics of the tuberculosis process are possible.

In some cases, tubercles disintegrate, and disintegration cavities (caverns) are formed from the fusion of adjacent disintegrated tubercles in the kidney parenchyma, sometimes these caverns are single, in other cases there are many of them, occupying large areas of the renal parenchyma (polycavernous process) if the tubercles disintegrate occurs on the mucous membrane of the pelvis or ureter, then specific tuberculous ulcers are formed here. The wall of the cavern is covered with necrotic tissue, and its cavity is filled with caseous contents or thick pus. In the

center of specific foci, nonspecific inflammatory changes develop. Zones of perifocal inflammation are sometimes closed over a long period of time.

Under favorable protective conditions in the body, the reverse development of the formed tubercles, their resorption, scarring, and petrification is possible. The dynamics of these changes are especially pronounced in relation to the kidney, where, along with the costal tubercles, they can be found in the medulla. Petrification of tuberculous changes is possible not only in nodules, but also in separate caverns and even in massive kidney lesions - a frozen kidney.

As a result of scarring and specific ulcerative changes from antibacterial treatment or under the influence of the body's protective reactions, a narrowing of the lumen (passage) of the ureter often develops, with consequent impaired motility of the pelvis. As a result of the expansion of the bowl and calyces and stagnation of what can be contained in the caverns, a specific tuberculous pyonephrosis develops in a number of patients: the parenchyma of the kidney thins, and the organ itself turns into a purulent bag. In some cases, with complete obliteration of the lumen of the ureter, exclusion of the entire kidney-autonephrectomy may occur. False healing occurs: urine becomes transparent, dysuric phenomena disappear.

Sometimes, instead of pyonephrosis, atrophy of the kidney develops, in which only a lump of sclerosing tissue is determined at the level of its location, in the center of which there are remnants of the renal parenchyma with the phenomena of a specific tubercular process. In some cases, the atrophied parenchyma of the kidney is replaced by hyperplastic, growing adipose tissue and turns into a lump of fat with scant remnants of renal tissue. These changes were called fatty replacement of the kidney. Extremely rarely, in the presence of pyuria and mycobacterium tuberculosis, the urogenital examination of the removed kidney does not establish specific changes characteristic of tuberculosis. The histological picture of the kidney in these patients corresponds to a chronic inflammatory process such as nephrosclerosis. This form of kidney tuberculosis S.P. Fedorov called it Koch's nephrocirrhosis. The lesion of paranephritis manifests itself in two variants. In some cases, it is accompanied by purulent melting of adipose tissue, in others, which

occurs much more often, sclerosing paranephritis develops, as a result of which the kidney is surrounded by a scar shell. Such changes in the perirenal tissue force the surgeon to use the subcapsular method when removing the kidney.

When the process spreads to the urinary tract, the lacrimal bowl, ureter, and bladder develop specific tubercular changes - tubercles, and as a result of their disintegration, one or another severity of ulcerative changes develops. Simultaneously with these changes in the mucous membranes, a sclerosing process develops in the adipose tissue surrounding the pelvis and urethra.

Scarring of specific ulcers on the mucous membrane of the ureter, as well as the periurethral tissue, contributes to the formation of narrowing of the lumen (passage) of the ureter, and on this basis, a motility disorder of the pelvis and ureter occurs. Changes occurring in a tuberculous lesion of the urinary bladder are identical to those described in the pelvic lesion, tubercles, specific ulcers, sclerosing process in the peribladder tissue.

As a result of scarring of ulcerative changes and infiltration of peribladder tissue, the development of a shrunken so-called "small bladder" is possible, which is usually accompanied by insufficiency of the bladder mouths of the ureters. The last circumstance can cause a very serious complication - ureteral reflux.

Classification. To date, there is no proven and generally recognized classification of kidney tuberculosis. Most of the proposed classifications of tuberculosis of the kidneys and urinary tract are based on the principle of staging. They are based on the pathomorphological course of the tubercular process: from the initial infiltrative changes in the depth of the renal parenchyma to the development of polycavernous tuberculosis of the kidney of tuberculous pyonephrosis.

Before the antibacterial era, the classification of renal tuberculosis was mainly clinical. For example, S.P. Fedorov identified two forms of kidney tuberculosis: 1 – acute or subacute, miliary form, 2 – chronic kidney tuberculosis. He, in turn, divided chronic kidney tuberculosis into two subgroups: a) with specific

tubercular changes, b) without specific changes - according to the type of chronic nephritis.

In the past, when the main method of treatment was early nephrectomy, any classification was considered to solve the main diagnostic task - recognition of diseases at an early stage. At present, the following classification is the most acceptable and sufficiently appropriate for practical purposes:

I. Clinical and radiological forms of renal tuberculosis:

1) tuberculosis of the renal pelvis (without existing X-ray changes)

2) tuberculosis of the renal papilla (papellitis)

3) cavernous tuberculosis of the kidney

4) tuberculous pyonephrosis

II. Phase of the tuberculosis process:

1) Open tubercular process

2) Exclusion

3) Total segmental scarring

4) Disclosure of one cup

III. Bacillary: BK + BK-2

IV. Functional state of the kidney:

1) The function is broken

2) The function is reduced

3) The function is missing

V. Complications:

Pyelonephritis, nephrolithiasis, hypertension, amyloidosis, etc.

Each of these forms is easily determined by classical methods, has its own clinical and radiological characteristics, its prognosis and methods of treatment.

Semiology. In the clinic of tuberculosis of the kidneys and urinary tract, unfortunately, there are not enough specific pathognomonic symptoms, quite often, tuberculosis of the kidneys can appear under the guise of a completely different disease.

Brief clinical assessment of individual symptoms.

a) Bacillus

This term should be understood as the excretion of tuberculosis mycobacteriuria in the urine in the absence of pyuria. Bacilluria is a symptom of great practical importance, as it can be the first manifestation of latent, flowing tuberculosis of the kidneys. Occurring without any clinical manifestations, bacilluria appears suddenly and indicates that there is damage to the parenchyma of the kidneys, mycobacterium tuberculosis (Kilpoitner) does not pass through. However, he established that mycobacterium tuberculosis under certain conditions can pass through the renal filter without causing changes in the kidney. He claims that bacilluria is a temporary phenomenon and occurs periodically. His opinion is that bacilluria should be recognized only when it manifests itself due to some complications or aggravation of the tubercular process in the lungs (hemoptysis, pyothorax, accompanied by a large influx of tuberculosis mycobacteria into the blood). Emphasizes that the usual research methods are not enough for this - it is necessary to examine the urine daily for several weeks. These data were confirmed by M.M. Chaussovsky

B) Pyuria.

Manifestation of pyuria - proved the connection of the tuberculosis center with the excretory urinary tract. At the first stage of the symptom, due to the small lumen of the course, due to the connection, the intensity of pyuria is quite insignificant. It can occur periodically and even disappear for a while. As the passage connecting the bowl and calyces with the tubercular focus expands, the amount of pus in the urine increases accordingly - pyuria becomes sharply pronounced. In cases where, in the course of antibacterial treatment, scar processes in the neck of the calyx or ureter cause their lumen to become overgrown, progress from the tuberculous focus stops and pyuria is not determined. Since pyuria or kidney tuberculosis passes for a long time in the absence of pain, it is often incorrectly interpreted as chronic pyelitis or pyelonephritis, which is why patients undergo long-term and unsuccessful treatment. Pyuria is the main and most constant symptom of kidney tuberculosis. The amount of pus in the urine can vary widely:

from barely expressed turbidity to the formation of a strong sediment, similar to what is observed in pyelonephritis. The intensity of pyuria depends both on the degree of the destructive process in the kidneys and on the conditions of emptying the tuberculosis focus. In cases of "asymptomatic" pyuria, the possibility of kidney tuberculosis should be considered and a long-term urological examination should be carried out.

C) Hematuria

Of clinical interest is the total hematuria occurring in the initial stage of the disease, of course, as the first symptom of a kidney tubercle, which is associated with their disintegration and the involvement of the walls of blood vessels in this process. Sometimes they are so intense that sometimes they force urologists to perform nephrectomies. Its intensity depends on the caliber of vessels affected by tuberculosis (tuberculosis manifested).

Sudden onset, irregular flow, intense nature, rapid disappearance - make these hematurias similar to those in kidney neoplasms.

Total hematuria can also be noted in the later stages of a tuberculous impression of the urinary system - when the process spreads to the mucous membrane of the urinary bladder (this is due to the presence of bleeding ulcers). Therefore, in the presence of only total hematuria, one should also think about kidney tuberculosis, especially in young people.

D) Urinary disorder.

Dysuric disorders occur when the bladder is damaged and those pathological changes on the part of the mucous membrane of the bladder that are observed in tuberculosis. However, urination disorders can be noted even in the absence of changes on the part of the mucous membrane of the urinary bladder. In such cases, they should be considered as a manifestation of the renal-bladder reflex of I.M. Epstein believes that similar phenomena occur under the influence of tuberculosis intoxication.

The intensity of diuresis of these disorders in renal tuberculosis depends on the nature of specific changes in the bladder, the depth of the lesion and the

localization of the process. Urinary disorders include: imperative urges, often nocturnal and then daytime urination, pain during and at the end of urination, urinary incontinence.

However, dysuria is not an early sign of the disease. Urinary disorder in kidney tuberculosis progresses rapidly, becomes painful and exhausting for patients. The frequency of urination fluctuates in a wide range: the majority of patients hold urine for an average of 1.5-2 hours, sometimes the frequency of urination is significantly increased - the urge occurs after 10-20 minutes (bladder neck, area of the Lieto triangle, a sharp decrease in urinary volume bladder, insufficiency of the closing apparatus).

E) Pain syndrome.

Pain in kidney tuberculosis can be either dull in the area of the affected kidney or sharp in the nature of renal colic.

Dull pains are caused by an increase in the kidney and its pinching in a poorly pliable capsule. They are localized in the hypochondrium or in the corresponding half of the lumbar region. They develop gradually, have a long-term course. Pains that are aching in nature can have a typical irradiating pain in the groin, thigh, scrotum, not related to the position of the patient's body or physical exertion. Sometimes pain can radiate to a healthy kidney.

In 21 percent of cases with kidney tuberculosis, acute pains such as renal colic are noted. They are no different from those in other kidney diseases. These pains are based on the following three factors: spastic contractions of the wall of the bowl or ureter, cicatricial changes in the lumen of the ureter and blockage of the lumen of the ureter by pus with fecal matter or blood clots - renal colic can end with its discharge. Cases of kidney tuberculosis with renal colic as the only symptom of the disease are of considerable interest.

F) General condition of patients.

The general condition of patients with a tubercle of the renal organs can be satisfactory even with a significant destructive process in the kidney. Sometimes

patients have an increased appetite, are in excellent condition, without loss of working capacity, are engaged in physical education and sports.

Disturbance of the general condition occurs when the process spreads to the urinary bladder with nodular rash in the kidney parenchyma and does not go parallel to the intensity of the destructive process. With small foci, a poor general condition can be noted, and with large ones, good. The severe general condition of the patient should cause suspicion of the possibility of bilateral kidney damage.

G) Temperature of the patient.

Fever is not a characteristic symptom of kidney tuberculosis. The second appearance can be related to many points:

1) an additional infection that joined the tubercular process

2) nodular rash in the parenchyma with an ascending infection of the kidney

3) retention of the contents of the caverns in case of insufficient emptying of them with subsequent absorption of toxic products into the blood

4) the influence of extrarenal tuberculosis foci, mainly the process in the lungs.

In most cases, observing an increase in temperature has the character of a low-grade fever. Often, a high temperature (running along a hectic curve) is the first symptom of a process in the kidney that has already progressed far. The duration of the febrile period can be limited to one or several days, in other cases it lasts for weeks and months, which is accompanied by an exacerbation of the process in the kidney, increased pain and dysuric disorders.

In those cases when the cause of high temperature cannot be established, with a proven unilateral process in the kidney with significant destructive changes in the parenchyma, it is necessary to perform a nephrectomy: the operation quickly brings him out of the state of intoxication and preserves his life.

Combined kidney damage with tuberculosis and stones.

Combined kidney damage by tuberculosis and stones cannot be considered as a case study. According to L.P. Kreiselburg, this condition is observed in 8

percent of cases. Until now, the following issues of this combination are considered debatable:

Is there any dependence between these processes or these lesions should be considered as a random combination of two independent diseases

Which of these processes is primary in the kidney and whether one of them can be recognized as a predisposing factor.

Forms of combined lesions according to Hotstein

Tuberculosis of the kidney and stones in the kidney of the same name.

Tuberculosis in one, and stone in the other kidney.

Stones in both kidneys, tuberculosis in one.

Tuberculosis in both kidneys, and stones in one.

Tuberculosis and stones in both kidneys.

Most often, there is the first group, in second place is the second.

Diagnostics. Diagnosis of cavernous tuberculosis of the kidneys when using modern methods of endoscopic examination and contrast radiography of the urinary system does not present any particular difficulties. Difficulties in diagnosis mainly arise in the initial stages - the infiltrated form of tuberculosis of the kidney in the absence of destructive changes determined on an X-ray.

The main reason for this problem is insufficient familiarity of general practitioners with the clinical manifestations of tuberculosis of the urinary system, weak study of analysis and insufficient use of all possible methods of bacteriological and biological examination of urine for the presence of tuberculosis mycobacteria.

Diagnosis of kidney tuberculosis is designed to solve the following tasks:

1) to confirm the presence of a tuberculous lesion in the urinary system or to rule out the specific nature of the purulent-inflammatory process. It must be supported by evidence;

2) determine the spread of the process in the urinary system: is there damage to one or two kidneys, damage to the ureter and bladder;

3) to establish the volume of destructive changes in the kidney parenchyma, since their severity determines the choice of a conservative or operative treatment method.

4) Determine the functional state of the diseased and healthy kidney, as the acceptance of operative treatment largely depends on this. In addition, it is necessary to rule out the presence of tuberculosis foci in the systems, in particular, in the genital organs in men.

The diagnosis of kidney tuberculosis should be based on indirect signs, since these indicators do not determine the nature of the disease and cannot become elements of a specific diagnosis. But sometimes the presence of such non-specific criteria (aseptic pyuria, history of tuberculous lungs or other organs, chronic cystitis that does not respond to conventional therapy). It is often a valuable diagnostic aid, and sometimes the only possibility to make a diagnosis.

Palpation of the area of the location of the kidneys in the issue of specific diagnosis of tuberculosis of the urinary system is not of great importance. Positive results in some cases determine the condition of the diseased kidney (its enlargement, mobility) and perirenal tissue, which can be taken into account in predicting the complexity of surgical intervention. An enlarged kidney is palpable only with pyonephrosis. As long as the membranes are not involved in the tubercular process, the kidney is mobile. When the perirenal tissue is involved, the mobility of the kidney is sharply limited, sometimes a dense infiltrate is determined at this level. In some cases, when kidney tuberculosis is suspected, when it is not possible to establish specific symptoms of this disease, palpation of the thickened ureter during vaginal examination in women can to a certain extent speak in favor of tuberculosis.

Urine study. The data of the general analysis of urine do not make it possible to make a diagnosis of kidney tuberculosis in patients. They testify only to the presence of a purulent-inflammatory process in the urinary organs without determining its etiology. The only proven diagnosis of kidney tuberculosis is the finding of mycobacterium tuberculosis in purulent urine.

Not being specific symptoms, some deviations found in the urine of these patients, however, give the right in some cases to speak in favor of kidney tuberculosis.

A) Persistent acidic reaction of urine.

This symptom is recognized by a number of authors as pathognomonic for kidney tuberculosis. According to H.M. Epshtein, the acidic reaction of urine with tuberculosis of the urinary tract can sometimes persist for up to 3 months. According to L.P. Kreisalburg's alkaline reaction of urine occurs in only 1.3 percent of patients. However, in his opinion, the acidic reaction of urine is not specific for tuberculosis of the urinary tract, as it can be noted in other diseases. However, a persistently pronounced acid reaction of purulent urine, which does not contain microbes, if even then mycobacterium tuberculosis is not found, is highly suspicious for tuberculosis of the urinary organs.

B) Proteinuria

According to R.M. Kronshtein, proteinuria should lead to thinking about kidney tuberculosis.

The diagnostic value of proteinuria is considered from several points of view:

1) albuminuria as an early symptom of kidney tuberculosis, which can occur long before the disease manifests

2) cylinderless albuminuria as a specific sign of renal tuberculosis

3) albulinuria as a manifestation of accompanying nephritis of the second kidney.

In the last category of patients, the amount of protein exceeds 1-2 percent and is usually accompanied by the presence of enzyme elements characteristic of nephritis. The amount of protein in the urine of patients with kidney tuberculosis, as a rule, does not reach a high level, it rarely exceeds one percent. In some cases, a large amount of protein should be attributed to accompanying hematuria.

C) Tuberculosis mycobacteria in urine.

Clear evidence of tuberculosis kidney disease is the detection of mycobacterium tuberculosis in purulent urine. But their absence in urine does not deny this diagnosis. The fact is that the existing methods of their determination do not provide an absolute opportunity to detect tuberculosis mycobacteria in urine in all cases.

There are three methods of detecting tuberculosis mycobacteria in urine:

1) bacterioscopic - in fixed and specially colored smears from urine sediment

2) bacteriological - by sowing urine on special media

3) biological - infection of a guinea pig with infectious urine of a patient.

These three methods complement each other.

It should be noted that the biological method is also not always correct. A positive result can sometimes be obtained even in the absence of tuberculosis in the kidneys. S.D. Fedorov described a case from his practice when a nephrectomy was performed based on a positive result. Histology revealed a kidney tumor.

Sometimes inoculation does not give a negative effect, if few tuberculosis bacteria were found in the urine used for urine analysis or they are not virulent enough, in particular, under the influence of antibacterial treatment. A positive result of a biological test to detect the specific nature of the disease is determined within 84-90%.

D) Aseptic pyuria.

In the antibacterial period of aseptic pyuria, great attention was paid. Nowadays, the question of the diagnostic significance of aseptic pyuria in renal tuberculosis is being revised. Clinicians have observed the frequent combination of kidney tuberculosis with non-specific pyelonephritis. Most likely, as a result of the use of antibiotics, their bacteriological effect is reflected in the weakening of the activity of tuberculosis mycobacteria, which entails the growth of pus of harmful microbes.

True aseptic pyuria should include those cases where the absence of flora was detected by cultures.

Aseptic pyuria is not a specific sign of renal tubercle, it is also possible in other diseases. However, in case of kidney tuberculosis, aseptic pyuria is stable, permanent, while in other diseases it is determined only periodically. It is determined in 85% of patients with kidney tuberculosis (L.P. Kryselburg).

Blood test. Changes in the blood, which are noted in patients with tuberculosis of the urinary organs, are not specific only for this disease. They are important mainly in assessing the effectiveness of antibacterial treatment, prognosis and in establishing indications and contraindications for surgical intervention in exhausted and weakened patients.

A) ***Specific blood changes*** (bacteremia, serological reactions). The source of bacillemia can be any active tuberculosis focus in the body. Of course, it is not possible to determine which source caused this condition.

The opinions of different scientists differ regarding the meaning of bazallemia. We also received definitions and serological reactions of the type of complement rejection.

Acceleration of ROE, leukocytosis, lymphopenia are also not specific for renal tuberculosis.

B) ***Protein functions of blood plasma.***

Recently, in the diagnosis of tuberculosis of the urinary organs, the determination of the level of protein functions in the patient's blood plasma by the electrophoresis method is widely used. In the exudative stage of the disease, in the presence of tissue decomposition processes, the globulin reaction increases. Acute exudative inflammatory processes are characterized by an increase in the globulin fraction. Currently, the study of the state of protein fractions in the blood plasma is carried out after the previous administration of tuberculin in 24-48 hours. Based on this, the indicators found are more characteristic of tuberculosis infection, allowing differential diagnosis with non-specific processes in the urinary organs.

C) ***C-reactive protein.***

In recent years, research articles devoted to the study of C-reactive protein in tuberculosis infection have been published. Normally, this protein is not detected in humans.

Not being specific for any infection, it serves as an early indicator of developing acute inflammatory and destructive processes in the patient's tissues. C-reactive protein in patients with kidney tuberculosis appears early, is the most stable compared to changes in the blood protein formula, and can be used as one of the early signs of the disease.

Endoscopic studies. Cystoscopy is one of the leading methods for diagnosing tuberculosis of the urinary organs. It can detect specific elements of tuberculosis infection in the bladder and confirm the diagnosis by the same documenting method. At the same time, it is possible to find out the side of the infection, and in combination with the indigo carmine test, the functional state of the sick and healthy kidney. Sometimes the bladder is so reduced that cystoscopy becomes impossible, the minimum capacity of the bladder is sufficient for cystoscopy (40-50).

Specific changes in the bladder can be expressed in the form of tubercular tubercles and ulcers. This is enough to determine the diagnosis. Tubercles have the appearance of yellowish formations the size of a pinhead or hemp seed, surrounded by an auxiliary rim located along blood vessels, are concentrated more often in the area of the mouth (mouth) of the ureter of the affected kidney. Moderate progression of the process in the bladder, tubercles are much less common.

Tuberculosis ulcers are a product of the decay of tubercles, they correspond to a later stage of bladder damage. Their peculiarity is that they are linear according to the localization of former tubercular nodules.

The mouths of the ureters affected by tuberculosis often undergo a number of changes. Accompanied by infiltration of the mouths and scarring processes, they are so characteristic that many authors give them the meaning of specific: gaping of the mouth, its retraction, star shape with uneven edges, bullous edema. The mucous membrane of the urinary bladder can be covered with a fibrinous plaque in some

areas. Cystoscopy usually reveals changes in the mucous membrane of the urinary bladder on the side of the kidney lesion.

Chromocystoscopy sets itself the task of:

Identify the side of kidney damage in the absence of subjective complaints.

Determination of the functional state of each kidney, which is very important when deciding on the choice of a treatment method, and in particular, an operative one.

However, the intensity of indigo carmine excretion does not always correspond to the severity of kidney damage. Sometimes, when the excretory function is preserved, massive destructive changes are observed and vice versa.

Then, in addition to the above-mentioned methods, according to S.P. Fedorov, it is necessary to perform catheterization of the ureters. This achieves:

1) obtaining urine directly from the kidneys to confirm the source of pyuria and the possibility of detecting tuberculosis mycobacteria.

2) checking the patency of the ureter of the affected side and thereby identifying narrowings that are important in the diagnosis of kidney tuberculosis.

X-ray examination.

X-ray examination for tuberculosis of the kidneys and ureter occupies a central place among other diagnostic methods.

X-ray diagnostics for tuberculosis of the urinary organs should solve the following tasks:

a) to confirm or exclude the tubercular nature of the lesion.

b) determine the volume of destructive changes in the urinary organs

c) specify the prevalence of the process in the urinary system

d) determine the functional state of both the diseased and healthy kidney

Only after that, you can decide on the method of treatment. In the diagnosis of tuberculosis of the kidneys, the following can be used: inspection radiography, ascending pyelography, internal urography, tomography, urokymography and renal angiography.

Survey radiography in the diagnosis of urinary organs can become a very valuable method for recognizing tuberculosis of the kidneys. On the pictures, you can find petrifications, the presence of silted areas, the presence of accompanying concretions, etc.

Ascending pyelography gives a complete picture of destructive changes in the kidney (one or many cavities), changed pelvic cups, etc.

When the process spreads to the ureter, characteristic specific changes occur in this organ.

a) straight direction of the ureter - it loses its curvature and takes the form of a stretched string between the bladder and the kidney

b) narrowing of the course of its lumen above which a number of consecutive expansions appear. In some cases, the anatomical condition of the entire ureter is revealed, its lumen sometimes reaches a significant width.

Excretory urography is less important for recognizing kidney tuberculosis than ascending pyelography, especially in the early stages of the disease. However, it becomes the only invaluable method in diagnostics when ascending pyelography is technically impossible to perform in the presence of an ulcerative lesion of the urinary bladder with a decrease in its capacity and in the case of insurmountable obstacles along the course of the ureter, which have developed as a result of tuberculous strictures.

It indicates the destructive changes in the kidneys and the degree of preservation of kidney functions.

Indications for excretory urography in renal tuberculosis can be divided into two groups: absolute and relative. Those cases in which it is not possible to perform an ascending pyelography should be classified as absolute (strictures of the urethra, small capacity of the bladder, ureter obstruction), and all cases of kidney tuberculosis are relative.

TOMOGRAPHY - can be performed to obtain auxiliary data.

a) performance of the shadow of the kidney, when it is not achieved by inspection radiography or excretory urography.

b) detection of specific changes in the parenchyma of the kidney and in the bowls, when ascending pyelography is impossible, when the usual excretory orography does not give a clear contrasting shadow of the bowls or cavities

c) to detect in the kidney parenchyma large caverns covered with a shadow, expanded cups or bowls.

UROKIMOGRAPHY - reflects a violation of the motor and evacuation function of the affected organs (ureter, character, frequency, rhythm and direction of the palsy).

CYSTOGRAPHY - allows you to detect the limitation of distension of the urinary bladder and the presence of vesicoureteral reflux (if present). It is possible to establish the side of the lesion when cystoscopy and catheterization of the ureters are impossible.

Renal angiography. The disease can be established only with a developed process. The number of vessels at the level of the tuberculous focus decreases, their hummingbird becomes diverse, contours are uneven. The terminal vessels of the branch disappear, roughly breaking off near the very focus of the pathological process. Renal angiography acquires great diagnostic value in the case of extensive so-called tubercular infiltrates of the kidney.

Renal angiography can be very useful for clarifying the limits of a specific process in patients who are proposed to undergo kidney resection.

Treatment. Until relatively recently, in relation to the treatment of unilateral tuberculosis of the kidney, there was a unanimous opinion that the only rational method is the early removal of the affected kidney.

S.P. Fedorov R.M. Fronshtein in all his writings claimed that in the case of confirmation of unilateral tuberculosis of the kidney, it is necessary to perform nephrectomy. At the same time, it is recommended to remove the kidney as early as possible, before the tuberculous process has spread to the bladder or the second kidney.

The discovery of antituberculosis drugs and their introduction into the clinic created a new era in the treatment of tuberculosis.

The latter are divided into two groups:

I) main antibacterial drugs (1st line) and 2-reserve drugs (second line). First-line drugs include strentomycin PASK and hydrazyl derivatives of isonicotinic acid (ftivazid).

Treatment begins, as a rule, with the use of first-line drugs. using two or three drugs of this series at the same time. Such combined use of the drug increases the effectiveness of therapy and prevents the development of drug resistance in bacteria.

Streptomycin is administered intravenously, usually 0.5 g per day.

Ftivazid is initially prescribed up to 0.1 g three times a day, intravenously, and then, if the drug is well tolerated, 0.3 g three times a day. Ftivazid is well tolerated by patients and does not cause side effects. Only some patients have peresthesias of the intercostal nerves and nerves of the limbs, convulsions and angiotic attacks. Therefore, ftivazide is contraindicated in coronary insufficiency, after a myocardial infarction, in epilepsy, heart defects, with decompensation and organic diseases of the central nervous system.

Ftivazid reduces blood coagulation.

Tubazid is prescribed internally at 0.15-0.2 g per day. Has significant toxicity. Duration of treatment is 3-4 months.

Saluzide 0.5 2-3 times a day (10% -5-10 ml intramuscularly, intravenously, subcutaneously)

Metazid 0.2-0.5 g 2 r.v.d. Less toxic.

Larusan 0.1-0.3 g 2-3 times per day.

Side effects: dyspeptic disorders, headaches, changes in white blood, in particular significant zosinophilia.

Contraindications: Liver disease, organic diseases of the central nervous system, glomerulonephritis.

Due to the great success of antibacterial therapy, indications for surgical treatment of tuberculosis of the urinary organs have narrowed significantly. Absolute indications for operative treatment today are polycavernous tuberculosis of

the kidney, tuberculous pyonephrosis. A. I. Mayants refers to a non-functioning kidney that is osmolated as an indication for nephrectomy.

The main method of surgical intervention for kidney tuberculosis is its removal. In addition to this radical operation, organ-sparing ones such as kidney resection and cavernotomy have also been used today.

Contraindications to nephrectomy. Absolute: a) tuberculous cavernous process in the second kidney with bilateral damage; b) significant insufficiency of the second (healthy) kidney; c) generalization of the tuberculosis process in the body.

Relative: severe general condition of the patient, tuberculous process in the lungs in the stage of an infiltrative outbreak, pregnancy in the second half or passing with complications, exhaustion of the patient, if it is associated with the tuberculous process in the kidney, is not only not a contraindication to nephrectomy, but dictates its implementation.

Plastic operations on the ureter. Replacement of the ureter in case of narrowing of the pelvic-ureteral segment by a loop of the small intestine (A.P. Frumkin).

2) Technique of surgery for structures in the pelvic segment of the ureter according to Bang-Huk-Boar.

Bladder plastic surgery.

a) Intestinal plastic surgery (loop of the ileum of the small intestine) (S.D. Holigorskyi, A.M. Gasparyan) or segment of the sigmoid colon (Kyuss, A.P. Frumkin).

Tuberculosis of genital organs.

We considered the pathogenesis above. What factors contribute to the development of a tuberculous focus in the organs of the reproductive system in men.

a) Injury. As recognized by A.I. Mayantz, hemorrhage and disruption of innervation mobilizes the essential infection. Currently, it is recognized that trauma gives an impetus to the development of an already organized patent focus.

b) Transferred gonorrheal inflammation of the urethra and gonads. However, this is a debatable issue.

c) Sexual excesses. Theoretically, the influence of an active sexual life on the development of genital tuberculosis is completely permissible. The predominance of the disease in young people during the period of increased sexual function is a confirmation of the stated assumption.

Tuberculosis of the genital system in men is rarely localized in one of its organs. In most patients, a tubercular process can be detected in a number of gonads during clinical examination.

They are most often found in the combined lesion of the epididymis in combination with tuberculosis of the prostate gland and the vas deferens.

More recently, external organs - testicles and epididymis - were recognized as the primary localization of tuberculosis in the genital area. However, Oppenheim's experiments and Lev's confirmations proved that the primary focus in the reproductive system is the prostate gland and seminal vesicles, hence the process of hematogenous, or more often via the seminal pathways antiperistaltically spreads to the testicle and epididymis.

The third group of authors B. N. Holtsov, Gohen admit the possibility of primary infection of any organ of the sexual sphere without any selective tendency of one or another of them.

Patho-anatomical picture of the prostate and seminal vesicles.

Specific changes in the prostate gland develop both in the follicles themselves and on the walls of the excretory ducts. As a result of the fusion of tuberculous nodules and their curdled decay in the parenchyma of the gland and seminal vesicles, decay cavities (caverns) can form. In the future, the contents of these cavities break through and enter the urethra or the near-prostatic tissues, forming persistent purulent fistulas on the perineum or buttocks. In rare cases, the contents of the caverns open into the lumen of the rectum. In rare cases, petrification of caseous contents is possible, which should be distinguished from prostate stones in the differential diagnosis.

Semiology. The clinical course of tuberculosis of the external genital organs does not always develop in the same way.

There are two clinical forms of the disease: acute and chronic. Why there is such a division is unknown to this day. The acute form begins with sudden, pulling pains in the scrotum with irradiation along the spermatic cord and in the sacrum. 3 swelling of the affected side of the scrotum will appear. The skin reddens, becomes shiny and tense. The spermatic cord and especially the vas deferens thickens and becomes sharply painful, the body temperature rises, the patient's condition is extremely depressed. Usually, by the end of the second week, the acute symptoms subside and the disease turns into a chronic form.

The chronic form develops gradually, and is not rarely determined during a random examination of the patient. Painful, dull compression along the course of the seminal canal, in the body and tail of the epididymis, a bumpy dense formation is noted. The boundary between the appendix and the testicle is delineated, which does not happen in the acute form, in which the appendix and the testicle merge into one conglomerate.

In the future, the density merges and is subject to melting. Abscesses grow together with the skin of the scrotum, the skin reddens, thins, the abscess comes out with the formation of a persistent, long non-healing fistula. Tuberculosis mycobacteria are rarely found in pus. In some patients, fistulas sometimes heal with a dense retracted scar.

Diagnostics. Diagnosis of epididymitis or orchoepididymitis does not cause special difficulties. The main task is to clarify the nature of the lesion of the appendix, that is, to differentiate tuberculosis of the external genital organs from gonorrheal processes and especially from the category of so-called non-specific epididymitis, which is often encountered in our country and in our time.

The rational choice of the treatment method depends on the establishment of such a diagnosis, and in particular, the solution to the issue of indicators for surgical treatment-removal of the appendix.

It is necessary to know that non-specific epididymitis in most patients has a sluggish, torpid, chronic course and it is very difficult to distinguish them from specific (tubercular) lesions. Unconditional evidence of tubercular damage to the appendix or testicle is the presence of fistulas in the scrotum. The finding of the tubercular process in two organs and especially in the urinary system can be very essential in determining the tubercular nature of the lesion of the appendix.

Is a specific diagnosis of tuberculosis of the external genital organs possible?

A positive Perke reaction in adults indicates only the presence of a tuberculosis focus in the body, without determining the degree of its activity and, of course, localization.

Serological reactions-rejection of complement according to Bordet-Chat (however, it is not universal).

Determination of tuberculous bacteremia by bacteriological examination of blood for the presence of the causative agent of tuberculosis (Lakhtenstein).

Treatment. Specific therapy (tuberculin treatment) is not justified. Patients in whom the diagnosis was made early from the onset of the disease and in whom there is a tendency to predominate productive changes, should be treated only conservatively (but at least 7-9 months).

X-ray therapy. If in the 1930s X-ray therapy was used quite widely, today, with the availability of antibiotics, the wide use of X-ray therapy is inappropriate.

Surgical treatment. Surgical treatment is the main and most rational method of treatment. It should be recommended for the following reasons.

a) tuberculosis-affected appendix as an organ has lost its functional significance.

b) a tubercular focus in the epididymis has a constant threat of spreading the process in the testicular parenchyma.

c) the presence of a unilateral tuberculosis lesion of the external genital organs very dangerously reflects on the opposite side, both in terms of the possibility of its damage by a specific process, and in terms of the toxic effect on the function of the not yet affected organs of the genital system of the opposite side.

Four types of surgical intervention:

1) removal of only the epididymis

2) removal of the appendix together with the testicle

3) organ-preserving operations, resection of the testicle, opening and treatment of the cavern

4) radical operations on pelvic genital organs.

Prostate tuberculosis.

Unfortunately, tuberculosis of the prostate cannot be considered a rare disease (about 10 percent of cases). Always hit the seminal vesicles.

Most patients with tuberculosis of the prostate gland are completely asymptomatic. However, when the process spreads to the thickness of the organ, the following occur: frequent, painful or difficult urination, terminal hematuria. Dull pains in the perineum are possible. When opening the cavity in the posterior urethra, pyuria is manifested. Ulcers are formed, indicating the presence of tuberculosis in the prostate. The general condition of patients in the early stages of the disease is usually satisfactory, with cavernous lesions general weakness, rapid fatigue, subfibrous or elevated temperature, weak appetite are noted.

Taking into account the fact that the prostate gland is more often affected in men with pulmonary tuberculosis, a mandatory rectal digital examination, as well as prostate secretion, is indicated in all these patients.

The prostate gland in such patients is enlarged. Its surface may be bumpy in some areas, the consistency is dense and elastic.

In all these patients, it is necessary to examine the urine in two portions - the presence of leukocytes in the second portion and the absence in the first. It is necessary to investigate the sperm culture for mycobacterium tuberculosis.

In some cases, the diagnosis can be made after ascending urethrography. The contrast mass penetrates the parenchyma of the prostate gland and reveals decay cavities.

Vesuculography can be of great help (insufficient filling of the testicles of the seminal vesicles, eaten contours of the urinary tract). Differential diagnosis is considered in detail in practical classes.

Treatment. Tuberculosis of the prostate gland and seminal vesicles is currently treated only conservatively, by using antibacterial chemopreparations actively and for a long time.

Effective antibacterial treatment of the prostate gland in sanatorium-resort conditions in combination with rational nutrition, vitamins, climate and the use of koumiss. The treatment is periodically repeated.

Surgical treatment is used only in limited cases - when a decay cavity is formed in the thickness of the prostate gland or when specific abscesses break into the paraprostatic tissue with the formation of spotting abscesses, purulent or urinary fistulas. In these cases, caverns are opened with their subsequent drainage and the use of antibiotics and chemotherapy drugs.

A number of authors (A.I. Mayants, V.D. Grund) prescribe vitamin D in an alcohol solution - 25-30 units per day for 3 months, along with treatment with streptomycin, in the case of a normal state of the urinary tract.

Indications and contraindications for sanatorium-resort treatment of tuberculosis and the approximate length of stay of these patients at the resort:

1) Bilateral tuberculosis of the kidney without detection of destructive changes in the parenchyma on the x-ray. The period of stay at the resort is 2-3 months.

2) Unilateral tuberculosis of the kidney with destructive changes in the papillae, with one or two small caverns. Duration of medication. at the resort for 3-4 months.

3) Tuberculosis unites the kidneys. The period of stay at the resort is determined individually, but at least 3 months.

4) Patients referred after nephrectomy for kidney tuberculosis. It is advisable to send them to a sanatorium-resort treatment within 6-12 months after the operation.

The term of stay is 2-3 months. After returning, these patients must be observed in local anti-tuberculosis dispensaries for 2 years.

5) Patients with bilateral tuberculosis of the kidneys with moderately pronounced changes in each of them with satisfactory function. The term of stay is 4 months.

6) Patients with residual changes in the bladder after nephrectomy. Stay at the resort for at least 3 months.)

7) Tuberculosis of testicles and their appendages. These patients can be referred to the resort both before and after surgery.

8) Prostate tuberculosis. The duration of stay at the resort is 2 months, with repeated treatment after one year.

INTRAVENOUS UROGRAPHY IVU

The IVU consists of a series of plain films taken after administration of an intravenous injection of a water-soluble iodine containing contrast medium.

Indications

1. Tuberculosis of the urinary tract

Traditionally the patient was prepared with a period of 4 h starvation and fluid deprivation and the bowel purged with a strong laxative. Occasionally the patient will feel nauseated after the IVU injection and rarely there will be a severe reaction with the need for cardiovascular and occasionally cardiopulmonary support. With this in mind, it seems reasonable to persist with avoidance of food for 2-4 h prior to the procedure.

Radiological anatomy

The kidneys are typically located at the level of the upper lumbar spine with the right kidney slightly lower than the left. They generally lie with their axes along the psoas muscles with the upper pole slightly more medial than the lower. Alterations in position and orientation of the kidneys may be related to congenital anomalies such as pelvic kidneys or may be secondary to mass effect from an adjacent lesion.

The size of the kidneys is somewhat variable depending on age and sex of the patient, but on the intravenous urogram, the kidneys normally range from 11 to 14 cm. The right kidney is typically slightly smaller than the left.

The kidneys should be symmetric in size with a discrepancy greater than 2 cm requiring an explanation. There are a number of causes of abnormal renal size, ranging from incidental anomalies such as congenital renal hypoplasia to significant conditions such as renal artery stenosis (small kidney) or infiltrating renal neoplasm (large kidney).

The kidneys should have a reniform shape and a smooth contour.

The intrarenal collecting system consists of calyces, infundibula, and the renal pelvis. Normally, each kidney consists of 7 to 14 evenly distributed calyces.

The normal ureters exhibit continual peristalsis and on a single film, it is uncommon to demonstrate the entire length of both (or even either) ureters.

They will often demonstrate smoothly narrowed areas (especially at the pelviureteric junctions and as they cross the iliac vessels in the pelvis) and more relaxed capacious areas. This is normal. Proximally, the ureter passes over the psoas muscle and should generally lay just lateral to the lumbar spine. The midportions of the ureters course over the lateral sacrum with the distal portion gently curving laterally in the pelvis before entering the bladder.

The ureter should be inspected for filling defects, which can be caused by stones or tumor, and should be symmetric in size. Evaluation of the ureteral course is important. Deviations of the normal ureter generally suggest extrinsic diseases, such as mass lesions. However, in patients with large psoas muscles the ureters may be displaced laterally as an incidental result.

The bladder is an oval to rounded structure that normally lies just above the pubic symphysis on the IVU. In women, the dome of the bladder may normally be indented by the uterus. These normal findings must be differentiated from abnormal extrinsic mass effects. Bladder wall thickness can sometimes be visualized and assessed, especially if thickened. Additionally, the bladder mucosa should be scrutinized for irregularity or filling defects that may suggest a mass.

Patient preparation

- blood urea and serum creatinine level should be within normal limits
- if patient is asthmatic premedication in the form of steroids is administered two days prior
- fasting after 10 pm (previous night) (as contrast injection sometimes induces nausea which might lead to vomiting and aspiration)
- patient should be well hydrated (dehydrated patients are prone for renal damage)
- bowel preparation is necessary, as gas and fecal matter filled bowel loops will obscure the kidney shadows
- low residue diet with plenty of oral fluids, the day previous to the IVU

Contrast media

Contrast materials currently in use are excreted almost exclusively by glomerular filtration, with subsequent concentration in the renal tubules and progressive opacification of the urinary tract.

They are two types:

1. ionic (urograffin, angiograffin)
2. non-ionic(omnipaque, ultravist)

Ionic contrast media have a higher incidence of reaction but they are cheaper as compared to the non-ionic contrast media.

Procedure

Patient is placed in supine position. The patient is asked to void the bladder before the procedure.

Contrast media is injected intravenously into a prominent vein in the arm. Test injection of 1ml of contrast is given and patient observed for 5 min for any contrast reactions. Then the rest of the contrast is rapidly injected within 30-60 seconds.

The dose of contrast media is 2 ml/kg body wt.

Intravenously injected iodinated contrast is excreted primarily by glomerular filtration in the kidney, opacifying the urinary tract as it progresses from the kidney through the ureter and to the bladder. Capturing this sequential "opacification" on

radiographs is the fundamental basis of the IVU. There are many variations in the filming sequence for the urogram that are acceptable as long as it optimizes visualization of specific anatomy of the urinary tract during maximum contrast opacification. Optimal visualization of the kidney is accomplished very early in the examination. Within 1 to 3 minutes after injection, the contrast bolus is filtered by the glomeruli and fills the nephron, resulting in intense opacification of the renal parenchyma; this phase of contrast opacification is called the ***nephrogram*.**

The kidneys should be evaluated for:

- their position
- orientation
- size
- contour
- radiographic density.

Soon after the nephrographic phase, contrast begins filling the intrarenal collecting system including the calyces and renal pelvis. This portion of the study is termed the ***pyelographic phase***.

5-10 min film

Shows nephrogram, renal pelvis

15-20 min film

A complete visualization of the pelvicalyceal system entire ureters is possible in this film, especially with the patient in prone position as the ureters will be antedependent in prone position.

30-35 min film

A complete visualization of the urinary tract: kidney, ureter, bladder can be done and bladder distension can be evaluated in the later film.

The series is varied according to the individual patient. Renal obstruction may require a delayed study up to 24 hours to outline the pelvicalyceal system.

Advantages

- IVU is low cost
- anesthesia is not needed

- detailed anatomy of the collecting system
- rapid overview of the entire urinary tract
- demonstration of calcifications
- demonstrate renal function and allow for verification that the opposite kidney is functioning normally
- it is sensitive for obstruction
- can show non opaque stones as filling defect
- IVP is an excellent modality to diagnose medullary sponge kidney and papillary necrosis .

Disadvantages

- contrast material must be avoided in patients with a history of allergy, hay fever or asthma until steroid cover has been given; those on metformin must stop this drug for 24 h before any contrast. These groups cannot safely undergo an emergency IVU.
- the differentiation from a phlebolith is difficult, especially when there is no ureteric dilatation proximally
- contraindications renal insufficiency
- contraindications hepatorenal syndrome, thyrotoxicosis, pregnancy
- do not differentiate solid or cystic lesion
- requires contrast medium and radiation
- missing small stones
- quality of study may be limited by inadequate bowel preparation
- inconvenience of a long filming sequence

Retrograde and antegrade pyelography

Direct injection of water-soluble iodinated contrast material is a useful method of examining various regions of the urinary tract. The advantage of this method of evaluation is the direct control over the contrast injection rather than reliance on secondary excretion from the kidney.

CYSTOGRAPHY

Imaging of the bladder is performed with a cystogram.

Indications

- the extent of vesicoureteral reflux
- urinary stress incontinence can be assessed
- urinary tract infections
- suspected obstruction
- suspected bladder trauma or rupture
- detection tumor
- detection diverticula
- detection stones
- to investigate suspected fistulas involving the bladder (usually into the gastrointestinal tract, occasionally elsewhere such as the vagina)

Procedure

A catheter is placed into the bladder and contrast material is then injected. The contrast material is optimally injected under fluoroscopic observation but occasionally is performed with only static conventional radiographs, such as in the trauma setting. Anatomic considerations and evaluation are similar to the IVU with a few caveats.

The method is useful for outlining tumors of the bladder when intravenous urography has been unsuccessful or equivocal.

One advantage to cystography is that vesicoureteral reflux can be evaluated during the conventional cystogram unlike during IVU.

Cystography can be classified into three groups:

- micturiting cystourethrography (MCUG)
- dynamic cystography
- simple cystography.

The MCUG is primarily performed for an investigation of childhood.

Dynamic cystography is part of the urodynamics investigation of the lower urinary tract.

Simple cystography is a relatively frequently performed and straightforward investigation in the adult.

Patient preparation

For two days before the examination, medical experts recommend limiting intake of products that provoke flatulence. On the eve of the research (in the evening), as well as immediately before cystography (morning) held enema.

Advantages

- these imaging tests provide the basic anatomy of the bladder and urethra
- show urethral movement
- low cost
- wide availability
- general familiarity

Disadvantages

- cystography is contraindicated spend in acute inflammation of the bladder, urethra, scrotum, prostate and seminal vesicles (If the research is still necessary, the doctor can perform a downward cystography)
- the catheter could damage the urethra, bladder or nearby structures
- they require catheterization
- the images contain no information about the pelvic musculature and adjacent soft tissue structures
- only structures in direct contact with the urethral and bladder lumen opacify with contrast.

Contraindication.

1) Unilateral polycavernous tuberculosis of the kidneys.

2) Tuberculous pyonephrosis.

In addition, the presence of marked changes on the part of the urinary bladder is also a contraindication for the referral.

Practical task: describe x-ray images of virtual patients.

Fig. 1. Tuberculosis. Cavern (tubercular track). JPG

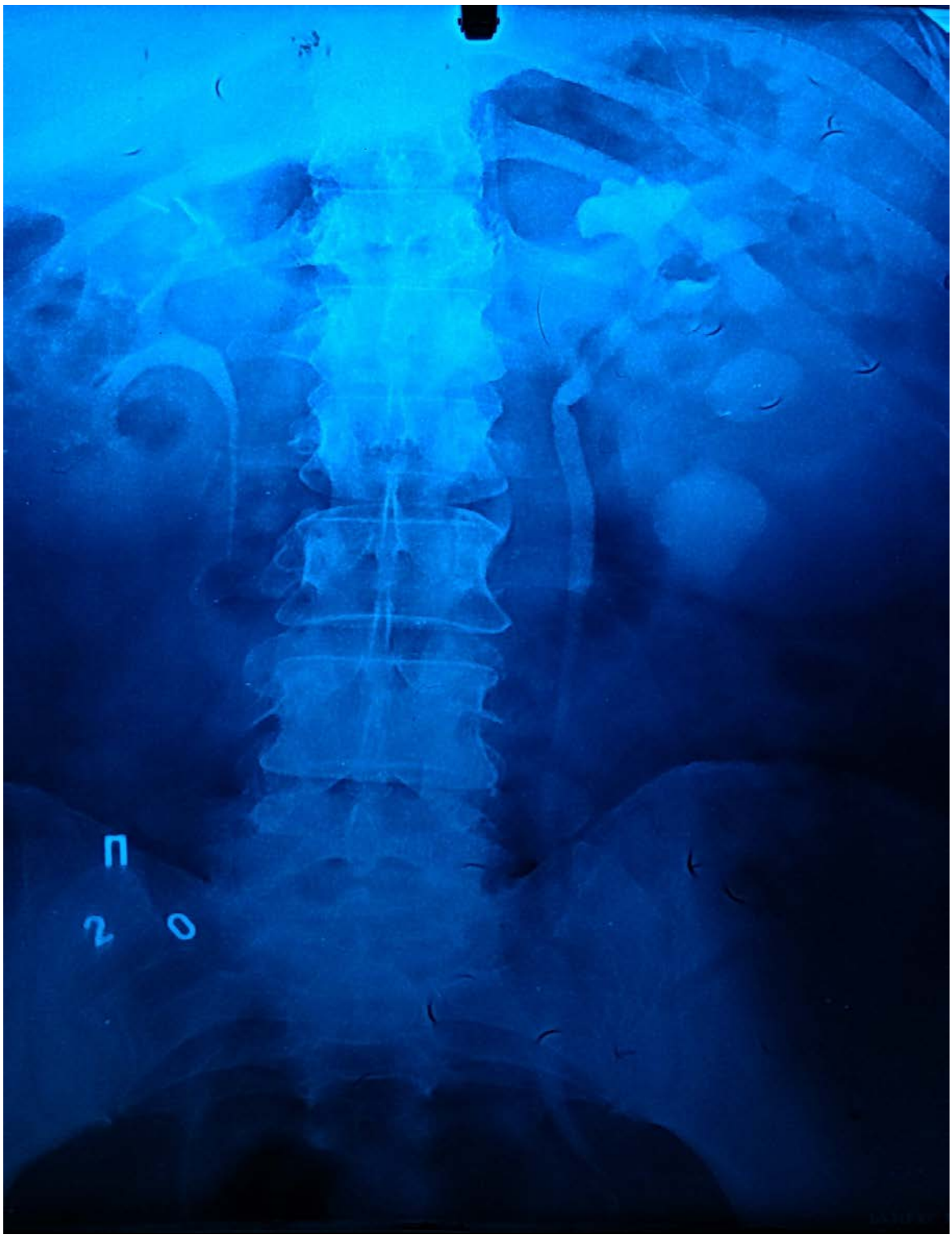

Fig. 1. Tuberculosis. Cavern (tubercular track). JPG

TESTS

1.What is a place of pyelonephritis in the structure of human's inflammatory diseases?

a) first
b) second
c) third
d) fourth
e) the last

2. Pyelonephritis is defined as:

a) specific kidney parenchyma inflammation
b) autoimmune glomerular capillary injury
c) infectious non-specific inflammation of kidney parenchyma
d) infectious non-specific inflammation of kidney parenchyma and kidney pelvis
e) all answers are correct

3.What is a causative germ of pyelonephritis?

a) Staphylococcus
b) Escherichia coli
c) streptococcus
d) proteus
e) Pseudomonas aureginosa

4.Pyelonephritis more often occurs :

a) men
b) women
c) older people
d) children
e) youth

5.What factor plays a driving value for the occurrence of pyelonephritis?

a) carbohydrate metabolism disorder
b) hemostasis violation
c) blood circulation disorder
d) lack of sleep
e) vitamin deficiency

6.What are the triad of clinical signs of acute pyelonephritis?

a) body temperature increase
b) abdominal pain
c) blood leucocyte count increase
d) weakness
e) fever with chills, intensive sweat, pain in the kidney area and changes in the urine.

7. Patient complains on the constant, aching pain in the perineum and suprapubic region, stagnant urine stream, frequent, difficult, painful urination, nocturia. Suffer for such a symptoms for several months while gradually started difficulties with urination, pain appeared in the perineum area. While rectal examination - prostate increased in size (more by right lobe), hard on palpation, asymmetric, central sulcus is smoothened, right lobe has a cartilage density, painless, bumpy. What kind of disease should we think of?

a) prostate cancer
b) prostate sclerosis
c) stone disease, right prostate lobe stone
d) prostate tuberculosis
e) chronic congestive prostatitis

8.What urine changes are typical for acute pyelonephritis ?

a) low specific gravity

b) leukocyturia
c) slats in urine sediment
d) bile pigments in urine
e) glucose in urine

9. Plain radiographs of acute pyelonephritis find:
a) lack of lumbar muscle contour
b) vertebral osteophytes
c) the smooth kidney contour
d) aerocoly reduction
e) phlebolithes

10.What antibiotics are prescribed in acute pyelonephritis:
a) cephalosporins
b) penicillins
c) are not prescribed
d) prescribed due bacterial urine test
e) tetracyclines

11. Before antibiotics administering in case of acute secondary pyelonephritis doctor should:
a) not restore urine passage
b) restore urine passage
c) recommend amplipuls as physiotherapy
d) do not take into account urine passage obstruction
e) provide kidney parenchyma fine needle biopsy

12.Chronic pyelonephritis pain in the kidneys most often are:
a) acute
b) spasmodic

c) occasional, dull and aching
d) pain while moving
e) burning pain

13.What are the reasons for the transformation of acute pyelonephritis to chronic?

a) high body mass index
b) long term of antibiotic therapy
c) inadequate antimicrobial therapy along with untreated urinary tract obstruction
d) diabetes mellitus
e) immune suppression

14.The patient complains on spasmodic sharp pain in the right lumbar region with genitals irradiation within three days. On the second day fever increased up to 40°C with chills, intensive profuse sweats. Right kidney is palpable, lumbar muscles tense, positive Pasternatsky symptom. Urine analysis: protein 0.033 g / L, white blood cells- lots WBCs/HPF. Ultrasound: right kidney is increased, pelvis and ureter are dilated. What is your diagnosis?

a) acute appendicitis
b) ileus
c) acute secondary right side pyelonephritis
d) acute cholecystitis
e) right side renal colic

15.The optimal term of chronic pyelonephritis treatment

a) 1 month
b) 3 months
c) 9 months
d) 1,5 – 2 years
e) 3 weeks

16.Patient 40 years, had a furunculus on a back region 2 weeks ago. Three days ago high body temperature appeared up to 39°C with fever and pain in the right lumbar region. The objective state is of moderate severity, pulse rate - 100 beats in 1 min. Arterial blood pressure 130/80, tongue is dry. Abdomen is soft and non-tender, painful in the right lumbar region. Lower back muscles are strained and swollen. Right hip is tightened to abdomen, its extension is painful. Urination is painless. Urine analysis - protein 0.033 g / l, leukocytes 4-5 WBCs/HPF, no bacteria found, erythrocytes 0-1 RBCs/HPF. General blood test - leukocytosis -15×109/l, stabs/bands neutrophil cells -15%, Blood sedimentation test - 28 mm / h. What pathological process led to this disease?

a) acute right side pyelonephritis
b) acute appendicitis
c) acute cholecystitis
d) acute pancreatitis
e) acute right side paranephritis

RECOMMENDED LITERATURE

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КАФЕДРА УРОЛОГІЇ

А.О. ГУБАРЬ

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