

**SYMPTOMS, DIAGNOSIS AND TREATMENT OF ACUTE INFLAMMATORY
KIDNEY DISEASES IN PATIENTS WITH DIABETES MELLITUS**

Atoeva Munixon Nabievna

Bukhara State Medical Institute

Key words: infections urinary paths, pyelonephritis, antibacterial drugs, tactics treatments

Symptomatology diagnosis and treatment of acute inflammatory renal diseases in diabetic patients

Key words: urogenital infections, pyelonephritis, antibacterial agents, therapeutic strategy

Diabetes mellitus (DM), being one of the most common diseases in the world, is an acute medical and social problem, priority for the health care systems of all countries [1]. Indisputable progress in the treatment of diabetes has led, on the one hand, to an increase in the life expectancy of these patients, and on the other hand, to an increase in the frequency of various complications of this disease. Disruption of carbohydrate metabolism has a negative impact on almost on All organs And systems organism, V volume including the genitourinary system. Among the main urological complications at SD most often meet infections urinary tract infections (UTIs) and closely related inflammatory diseases kidneys.

Peculiarities development And currents IMP at sick SD

One from main specific processes at SD is increase concentrations glucose V urine on background hyperglycemia . Glucosuria is one from most important risk factors development IMP at patients With SD V result suppression of phagocytosis And, Maybe, cellular immunity, A Also contributing adhesion bacteria, Although V experiment Very high con- centration glucose V urine (over 55 mmol/l) suppresses their growth. In final as a result, long-term expressed glucosuria is a risk factor for damage to the serous- mucoid layer of the urothelium with the development of UTI. Pathogenetic factors that cause development IMP And inflammatory diseases kidneys in diabetes, are also [2, 3]:

toxic effects of hyperglycemia;

micro- And macroangiopathy , contributing renal ischemia ;

autonomic neuropathy leading to neurogenic bladder dysfunction and chronic urinary retention ;

flaw glycogen, leading To deterioration nutritional conditions of the glomerular-tubular apparatus;

hyperglycemia, which reduces the phagocytic activity of leukocytes and the bactericidal activity of the blood.

At SD1 development IMP promotes long-term course of the disease, more early (V flow first 4–5 years) And persistent development of peripheral and autonomic neuropathy. [4].

Spectrum pathogenic microorganisms, provocative IMP in patients with diabetes mellitus is similar to that in patients without diabetes mellitus [5]. It has been established that diabetes mellitus increases the risk of developing acute pyelonephritis (AP) ascending from the lower urinary tract, the causative agent of which is representatives of the genus *Enterobacteriaceae* and *Enterococcus faecalis*. Often at women, suffering SD, V this roles perform also representatives genus *Klebsiella* (25% at patients With SD By compared to 12% in patients without diabetes) [7].

A number of established bacterial factors are related to the virulence of a bacterial cell and its ability to attach to the surface of the mucous membrane before the moment of its introduction. clutches, so-called fimbriae . Fimbriae 1 type are present in almost all types of *Escherichia Coli*, which provide binding to receptors of vaginal mucosa cells, And V lesser degrees With receptors protective mu - copolysaccharide layer lining the mucous membrane of the urinary tract . . For bacteria with type 2 fimbriae (P- fimbriae), a tendency to adhere to glycolipids of substances of various blood groups that are secreted by the urothelium is more characteristic . P- fimbriae , in contrast to type 1 fimbriae , are more degrees have ability To clutch And defeat urinary tract , causing UTI and pyelonephritis. In the studies of A. Hopelman , S. Geerlings (2000), an increase in the adhesion of *E. coli* with type 1 fimbriae to the uroepithelium of women with diabetes was revealed compared to healthy people [8].

Important factor protection macroorganism from infection is the level of sex hormones, in particular: estrogens and progesterone for women, and testosterone for men. The urothelium produces and secretes onto the surface a mucopolysaccharide substance that forms an adhesive protective layer. The formation of this mucopolysaccharide layer is considered a hormonally dependent process: estrogens affect its synthesis , progesterone on his selection epithelial cells [9]. Confirmed Also And role testosterone V impact on urothelial receptors . It has been shown that in patients with diabetes, against the background of decreased levels of sex hormones, there is an increased risk of developing UTI [10, 11, 12].

Spicy inflammatory diseases kidneys at sick SD

Inflammatory diseases of the kidneys include acute pyelonephritis (AP) and chronic pyelonephritis (CP), which can be either a consequence of AP or an independent form of the disease. AP is determined How infectious and inflammatory disease of the renal parenchyma and renal pelvis with predominant defeat interstitial fabrics. Long- term decompensation carbohydrate exchange is risk factor development And progression acute infectious - inflammatory process in the kidneys, however, pyelonephritis itself is not complication SD. OP And his chronization are factors that aggravate the course of such a formidable specific complication as diabetic nephropathy.

Special attention clinicians addressed on acute purulent-destructive forms of the course of OP, due to the rapid progression of the disease and a real danger to life sick V cases development septic states. In case obstructions urinary paths (stones kidneys And ureters , tumor urinary bubble, adenoma prostate And etc.) algorithms diagnostics And

treatments IMP radically change , That's why these cases demand separate consideration. OP in patients with diabetes can be either asymptomatic (in case of immune system areactivity , or partial immunity) moon deficit), So And leak manifestly [14]. It is believed that decrease common immune resistance predisposed -

Gaet To persistence pathogens IMP [15].

In non-obstructive OP, microorganisms reach the urinary tract hematogenously or through the lymphatic pathways [16]. It has been established that What sick SD especially vulnerable For rapid progression infections V renal parenchyma And her subsequent complications. The combination of diabetes and OP leads to the emergence of a vicious circle, when the active inflammatory process in the renal parenchyma negatively affects the function of the glomerular-tubular apparatus, simultaneously aggravating insulin deficiency due to the increase in acidosis. In turn, metabolic disorders and a clear decrease in microcirculation worsen the course of purulent infection in the kidneys, What received Name "syndrome mutual burdens " (Fig. 2) [17] .

An additional factor that aggravates the development of AP is endothelial dysfunction, which, against the background of prolonged exposure to hyperglycemia, leads to disruption of angiogenesis and hemostasis [18]. In this case, there is a decrease in the synthesis of NO And increase concentrations endothelin-1 V renal vessels, What V result leads To their spasm, platelet aggregation, adhesion of mono- and platelets. Decompensated diabetes is often accompanied by a state of immunodeficiency, in which infectious processes occur more aggressively. Maybe, By this reason development of OP is Not so many result aggression microorganisms, but rather as a result of the state of the macroorganism predisposing it to infection [19].

AP often occurs as a severe infectious disease that poses a threat to the patient's life. Conflicting data on the clinical features, course of the disease and insufficient studied mechanisms development OP at sick With SD determine difficulties his diagnostics, treatments and prevention. On sectional material OP is revealed at 20% of patients with diabetes mellitus who died from various other causes, i.e. who did not have a primary diagnosis of AP [20].

In patients with diabetes, as in the general population, a unilateral inflammatory process predominates. However, with severe decompensation of diabetes, the risk of developing bilateral pyelonephritis increases . As a rule, the clinical onset of the disease is acute - with high temperature, fever, dysuria , pain in the lumbar region. Leukocytosis and an increase in ESR are noted in the blood . In patients with diabetes, urine testing reveals the presence of bacteria and a large number of leukocytes , however , in this category of patients, a normal cellular composition of urine sediment and the absence of bacteriuria.

In typical cases, diabetes mellitus affects the upper urinary tract, up to the development of a diffuse-purulent (apostematous pyelonephritis) or purulent-destructive form of the disease (carbuncle, abscess). The frequency of development of purulent forms is proportional to the severity of the course and the degree of carbohydrate metabolism disorder . exchange.

In typical cases, diabetes mellitus affects the upper urinary tract, up to the development of a diffuse-purulent (apostematous pyelonephritis) or purulent-destructive form of the disease (carbuncle, abscess). The frequency of development of purulent forms is proportional to the severity of the course and the degree of carbohydrate metabolism disorder . exchange.

In at least a third of patients, OP manifests itself in the form of a purulent process: apostematous pyelonephritis, carbuncle or abscess. It is known, What V 34% cases at sick without SD OP turns into purulent forms [21]. If diagnosis is not made in a timely manner , And belated therapy diseases at patients on background of diabetes, acute serous pyelonephritis develops into purulent forms in most patients [22]. In these cases, the disease is accompanied by severe intoxication and, if not treated in a timely manner, treatment Maybe develop septic shock, in which mortality exceeds 60% [23, 24, 25]. In diabetes mellitus , a powerful infectious process can become a prerequisite for the rapid development of pyogenic infection. Patients with diabetes mellitus are prone to the development of rarely diagnosed interstitial nephritis of an unusual form - nephritis caused by gas-forming microorganisms and accompanied by a high mortality rate (emphysematous pyelonephritis). Histologically, it is characterized by acute pyogenic infiltration with the formation of microabscesses and a high risk of further development of acute renal failure (ARF). The route of penetration of these microorganisms can be hematogenous. Even in the absence of obstacles outflow urine sharp infection V parenchyma is capable of progressing unnoticed until the development of intrarenal abscess, With distribution on perirenal fiber. It should be especially noted that emphysematous infection of the urological tract occurs almost always and only among patients with diabetes [13], and is a life-threatening condition , mortality at this form reaches 90% [26].

Difficulties V identification OP at SD arise V connections With the absence of pronounced local symptoms, and partly masking the clinical picture of AP by severe manifestations diabetes. Erased painful syndrome at sick With long -term decompensated SD And fever unclear genesis, in the absence of changes in urine tests, is the basis for a urological examination to exclude OP [27]. In patients with diabetes, the presence of OP may not be established at normal cellular composition urine, absence bacteriuria , proteinuria. A similar complex situation can be observed at apostematous pyelonephritis, carbuncle and abscess without drainage of purulent exudate into the renal pelvis and calyces . Therefore, it is necessary to use all available laboratory capabilities and modern radiation methods of examination (MRI) for timely diagnosis.

Traditionally diagnostics OP is based on accounting result -

of a comprehensive examination, including data from the anamnesis , clinical picture of the disease, laboratory and radiation methods research. Position With diagnostics OP has improved significantly with the advent of new high - tech diagnostic funds, such How ultrasonic and - research (ultrasound) and multi-spiral computed tomography (MSCT). These methods have changed the understanding of the possibilities of diagnostics of inflammatory kidney diseases. It has been established that early radiation diagnostics of purulent forms of OP at SD belongs main role V recognition pathological process, A Also V establishment stages And forms of the disease, V dynamic observation at conservative therapy and monitoring the condition of organs (kidneys and surrounding tissues) [28].

Antibacterial therapy for AP should be started without delay . Empirical therapy involves choosing antibiotics that are active against the most common pathogens of this type of infection. According to the recommendations of the European Association of Urologists (2008) for the treatment of urinary tract infections, patients with pyelonephritis at the stage of purulent inflammation are recommended to be treated with fluoroquinolones , aminopenicillins with - lactamase inhibitors , second- and third-generation cephalosporins, and aminoglycosides. However , it is known that the use of aminoglycosides in patients with diabetes mellitus, V connections With their expressed nephrotoxicity , possible only in exceptional cases and under careful control - lem of kidney function.

Special place V treatment OP occupy carbapenems . By According to the European Association of Urologists (2008), this group of drugs has super wide spectrum actions: active in relation majority gram-positive, literacy - negative And anaerobic microorganisms. That's why They are drugs second rows at treatment heavy forms of inflammatory processes of the urinary tract. In our opinion, at development OP at sick With SD, V separate cases, carbapenems can apply And How drugs first row.

Literature

Suntsov Yu.I., Dedov I.I., Shestakova M.V. Screening for complications of diabetes as a method for assessing the quality of medical care for patients. Moscow. 2008.

Levison ME, Pitsakis PG Effect of insulin treatment on the susceptibility of the diabetic rat to Escherichia with li-induced pyelonephritis. J. Infect Dis 1984; 150:554-60.

Shamkhalova M.Sh., Chugunova L.A. Infections urinary paths in patients with diabetes mellitus: diagnosis, prevention, treatment.

// Sugar diabetes. - 2001. - No. 3 (12), With. 24–29.

Svetlova G.N. Diabetic peripheral sensorimotor neuropathy in children: the role of clinical, metabolic and genetic factors. Dis . Cand. honey. sciences. Moscow. 2008.

Ludwig E. Urinary tract infections in diabetes mellitus // Orv Hetil . 2008 March 30;149(13):597–600.

Carton JA, JA Maradona, FJ Nuno, R. Fernandez-Alvarez, F. Perez- Gonzalez and V. Asensi . Diabetes mellitus and bacteraemia : a compara - tive study between diabetic and non-diabetic patients, Time J Add 1 (1992), pp. 281–287.

Geerlings SE, Stolk RP, Camps MJL, Netten PM, Hoekstra JBL, Bouter KP, Bravenboer B., Collet TJ, Jansz AR, Hoepelman IM Pre- valence and risk factors for asymptomatic bacteriuria in women with dia - betes mellitus. ICAAC 1999: abstr 607.

Hopelman A., Geerlings S. Urinary tract infections in diabetes mellitus// Clinical microbial and antimicrobial chemotherapy, 2000; No. 2 – pp. 40–45.

Laurent O.B., Sinyakova L.A., Kosova I.V. Treatment and prevention chronic recurrent cystitis in women. *Consilium medi - cum* , no. 7, volume 6, 2004.

Pradidarcheep W. Lower urinary tract symptoms and its potential relation with late-onset hypogonadism. *Aging Male*. 2008 Jun;11 (2):51–5.

Hassan J.M., Pope JC., Revelo P., Adams MC., Brock J.W., Demarco R.T. The role of postoperative testosterone in repair of iatrogenic hypospadias

in rabbits. *J. Pediatr Urol*. 2006 Aug;2 (4):329-32. Epub 2006 Apr. 27.

Strait S., Santti R., Gustafsson AND., Ma .. reel .. S. Co-localization of andro-

gen receptor with estrogen receptor beta in the lower urinary tract of the male rat. *J Urol*. 2001 Aug;166 (2):674–7.

Lopatkin N.A. *Urology.// M.: Medicine*. 1992.

Joshi N., G.M. Caputo, M.R. Weitekamp and A.W. Karchmer. Infections in patients with diabetes mellitus, *N Engl J Med* 341 (1999),

p. 1906–1912.