DIFFERENTIAL DIAGNOSTIC CRITERIA OF KIDNEY INJURY DUE TO LEPTOSPIROSIS

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ABSTRACT — Kidney injury in leptospirosis is a leading pathogenetic mechanism, due to development of degenerative changes in the tubular apparatus. A number of natural focal infections have similar pathognomonic symptoms that make it difficult to conduct differential diagnosis and provide timely ethiopathogenetic treatment. The article highlights the differential diagnostic criteria of kidney damage at leptospirosis, gives clinical examples.

KEYWORDS — leptospirosis, kidney damage, differential diagnostic criteria

INTRODUCTION

Almost all infectious diseases affected by severe fever and an intoxication syndrome are characterized by moderate proteinuria (within 0.3 g/l), caused by an increased permeability of the vascular wall [1, 3, 4, 7, 8].

There are a number of infectious diseases in which renal damage is the leading pathogenetic mechanism that often determines its outcome: malaria, hemorrhagic fever with a renal syndrome (HFRS), and leptospirosis [2, 5, 6].

At leptospirosis, there is a degenerative process in the epithelium of the tubular apparatus — a diffuse nephrosis in combination with hemolysis. This is a characteristic sign of leptospirosis, which occurs with necrosis of glomerular epithelial cells and the main membrane, followed by the involvement of renal tubules in the process [2, 5, 6]. In the first days of clinical manifestations, degenerative and necrotic changes develop in the affected organs and tissues, including the epithelium of the renal tubules. In some patients at the height of the disease, leptospira begin to accumulate,

multiply in the convoluted tubules of the kidneys and be excreted from the body with urine. Patients develop signs of acute renal failure, characterized by oligoanuria and uremic coma: diuresis decreases to 500 ml per day or less, proteinuria increases in the urine; there is an increase of the amount of leukocytes, erythrocytes, and cylinders in the urine sediment, and of the level of nitrogen-containing substances in blood. There is a decrease in both the secretory and excretory functions of the kidneys. Oliguria can change for anuria. The peculiarity of acute renal failure at leptospirosis is the absence of edema and arterial hypertension.

The aim of the study:
to estimate the incidence and single out differential diagnostic criteria of urinary tract involvement in leptospirosis.

METHODS

We made a clinical epidemiological analysis of 25 cases of leptospirosis of icterohemorrhagic form. The diagnosis of leptospirosis was confirmed by appropriate serological reactions and PCR diagnostics. The study involved patients aged 18 to 65 years. The majority of the cases were men of working age — 76%. More than half of those who developed leptospirosis were rural residents — 56%.

RESULTS

While collecting epidemiological anamnesis, we found out that the majority of patients with leptospirosis (76%) had a percutaneous mechanism of infection through the outer covers, contaminated water being the main factor of transmission of infection — when swimming in water tanks, fishing and hunting. Leptospirosis was registered mainly in the summer period (68%) with a maximum incidence rate in August (36%), although some cases were registered throughout the year.

In the initial period of the disease, the observed patients with leptospirosis often had a body temperature above 39,1° C, in 12% of cases — above 40° C. In 64% of cases, the temperature curve was remittent; a constant type of temperature curve was registered in every fifth patient; in single cases, with a severe disease, there was a double-wave fever.

The duration of the febrile period against the background of the antibiotic therapy was an average of 11.4 days. From the first days of the disease, the increase in body temperature in the patients observed was accompanied by severe symptoms of intoxication. All patients with leptospirosis complained of general weakness (100%), headache of a diffuse nature (92%).

We noted the development of renal syndrome in 12 patients with leptospirosis — 48%. Patients complained of pain in the lumbar region (48%), Pasternatsky symptom was positive in 10 patients (40%). In 9 patients (36%) there was a decreased urination. Anuria developed in 1 patient (4%) with a severe course of leptospirosis, which ended in a lethal outcome. Typical changes in urine were in 13 patients (52%): mild proteinuria, leukocyturia, hyaline and granular cylinders. An increased level of nitrogen-containing compounds was registered in the blood of these patients. The increase in creatinine was registered in each patient with the renal syndrome, the highest values were observed on the 7–8th day of the disease from 280 μmol/l at a moderate form to 517 μmol/l at a severe form. The level of urea was also increased — mean value was 12.0 mmol/l.

In severe leptospirosis, patients developed an acute medical emergency — acute renal failure. In the pathogenesis of renal insufficiency at this infection, the DIC-syndrome, intravascular hemolysis in combination with anemia and thrombocytopenia, and the metastatic spreading of pathogens to the tubular epithelium are important.

Clinically, it results in the development of acute nephrotic syndrome, manifested by proteinuria, edema and anuria.

Patient K., aged 64, resident of Astrakhan, was admitted to the Department of the Regional Infectious Clinical Hospital on September 26, 6 days after the onset of the disease with the directional diagnosis of *fever of unclear etiology*.

On admission he complained of weakness and fatigue, marked pain below waist, nausea, vomiting.

From the anamnesis: acute onset on the 29th of September, he felt weakness, body temperature increased to 39° C. At the same time, there was a darkening of the urine. The next two days he was running a fever of up to 39° C, suffered weakness, headache, severe muscle pain — the patient had difficulty walking around the room. He mentioned a single vomiting. Later, pain in the abdomen was becoming more intense and it acquired a permanent character; there was a twice repeated vomiting, jaundice on the skin, the reddish color of urine.

Epidemiological data — two weeks before the disease the patient went fishing to lakes in the Volga delta.

On examination — the condition is severe. The patient is running a fever, fragile. The face is hyperemic, sclero conjunctivitis is obvious (subscleral hemorrhages are revealed). The skin is icteric. On the skin of the body there is an abundant spotted and small-point rash of pinkish-red color.

In the lungs breathing is harsh. Respiratory rate is 24 per 1 min. Heart sounds are muffled, rhythmic. Pulse is 74 beats per minute. Arterial blood pressure is 120/60 mm Hg. Abdominal palpation is painful in all departments. Hepatosplenomegaly. Urination is seldom, in small quantities, urine is red. There are no meningitis signs.

Diagnosis: leptospirosis, an icterohemorrhagic form, severe course.

In the general blood test there is leukocytosis 16 · 10/ l, platelets — 24 thousand.

As clinically indicated, the patient was transferred to the intensive care unit.

The following day the patient's condition worsened. The jaundice increased. There was shortness of breath; weakened breathing, rhonchi crackles in the lungs. Respiratory rate was 30–32 per 1 min. Pulse — 88 beats per minute. Arterial blood pressure was 140/70 mm Hg. There was severe hepatosplenomegaly, oliguria, bleeding in places of injections. In the general blood test there was an increase in leukocytosis, stab shift, platelets — 36 · 10/l. The total bilirubin of blood was 107 μmol/l. In the coagulogram: fibrinogen B ++++, hypercoagulation. In the analysis of urine according to Nechiporenko: erythrocytes were 17500, leukocytes — 13500. The patient has registered complications of the underlying disease — acute renal failure, DIC syndrome, two-sided pneumonia.

From 29.09 — there is no fever, the symptoms of acute renal failure are increasing, the DIC syndrome is progressing, the patient is in sopor. Lumbar puncture was performed. In cerebrospinal fluid: cytosis — 57 cells 11 neutrophils, 46 lymphocytes, protein 0.75%, Pandy's reaction is weakly positive.

In dynamics: the patient's condition worsens, tachycardia is 120 per 1min, arterial blood pressure is 170/80 mm Hg, dyspnea is 40 per minute. Acrocyanosis. The skin is intensely icteric. The patient is transferred to the artificial lung ventilation. Coma I. Along the gastric tube — 200.0 of coffee-ground vomit.

On the 5th of October the condition is extremely serious. Coma IV. Hypothermia. Diffuse cyanosis. Pulse is 52 beats per minute, threadlike. Blood pressure is 80/50. At 15 o'clock there is cardiac arrest, resuscitation measures are without a positive result. Death was ascertained.

The diagnosis of leptospirosis was confirmed serologically by leptospira microagglutination Testing and lysis test with the serogroup of Icterohaemorragica 1/200, grippotyphosae 1/400.

CONCLUSIONS

kidney injury in leptospirosis is caused both by direct damaging action of the pathogen on the tubular apparatus of the kidneys, and indirectly as a result of hemolysis of red blood cells. Differential diagnostic criteria of leptospirosis are the absence of edema and arterial hypertension on the background of proteinuria of varying severity.

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