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TOXIN-INDUCED ACUTE KIDNEY INJURY. CLINICAL OBSERVATION

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ABSTRACT — The article presents a clinical case of toxin-induced acute kidney injury. The authors analyzed the cause of the event, revealed the pathogenesis of toxin injury of the kidneys, and described the main principles of treatment. The authors used this clinical case to show the importance of timely renal replacement therapy and the influence of comorbid pathology on disease development. The onset of acute kidney injury in patients with surrogate alcohol intoxication manifested as combined kidney and liver injury was described.
The article may be of interest to doctors of all specialties, especially for therapists and surgeons who are the first to reveal acute kidney injury and their choice of adequate management ultimately determines the outcome.

KEYWORDS — acute kidney injury, surrogate alcohol, hemodialysis.

INTRODUCTION

Acute kidney injury (AKI) is a complex of symptoms that develops due to a sharp decrease in the glomerular filtration rate (GFR) that leads to an increase in the concentration of nitrogen and non-nitrogen metabolites in the blood with the disturbances in the levels of electrolytes, pH, and fluid volume excreted by the kidneys [1]. The mortality rate in patients with AKI was around 20–30% during the past years. In intensive care units, it reached 70% [2]. Prognostically unfavorable factors in patients with AKI are oligoanuria, persistent hypotension, hepatitis, a necessity for artificial lung ventilation, and impaired consciousness. One of the most significant risk factors of the mortality in AKI is sepsis. The lethal outcome rate from this complication is 75% [3]. It should be noted that in 75% of cases, renal AKI is provided by acute tubular necrosis (ATN) that can be of two types: ischemic ATN, which complicates shock of different genesis, comatose conditions, and dehydration, and nephrotoxic ATN that develops as a result of a direct toxic effect of chemical compounds and pharmaceutical drugs [4]. The most important role in the pathogenesis of toxin-induced AKI belongs to vasoconstriction caused by the effect of nephrotoxins that leads to alterations in renal microcirculation [5]. Nephrotoxic AKI is diagnosed in every tenth patient with AKI admitted to hospital for acute hemodialysis [6]. Timely diagnostics of AKI is crucial for the provision of specialized medical care. The treatment of AKI includes etiotropic therapy that aims to resolve or decrease the effect of the causal factors. Pathogenetic therapy targets the stabilization of systemic and regional hemodynamics, correction of electrolyte and pH disturbances, resolution of anemia and hyperhydration, limitation of protein diet, and also includes extracorporeal methods of treatment [4]. Often, timely diagnosis of AKI determines disease outcome.

The study aimed to present a clinical case of a patient with toxin-induced AKI and characteristics of disease p, its diagnosis, and treatment.

MATERIALS AND METHODS

A review of the literature on AKI was performed, clinical recommendations on the management of patients with AKI were analyzed, and the clinical case of a patient with AKI was presented.

RESULTS

Patient M. aged 65 years was admitted to the nephrology unit of the Tver Regional Clinical Hospital (Russia). The patient complained of pronounced leg weakness, inability to walk, decreased volume of the excreted urine to 100 ml per day, constipation for three days, whole body trembling, respiratory difficulty, loss of appetite, and dry mouth.

The anamnesis, collected from the patient's relatives, showed that the patient abused alcohol and surrogate alcohol for 12 days before the disease onset. The disease onset was sudden and started with fainting and a fall in blood pressure to 70/40 mmHg. The ambulance team started pre-hospital vasopressor support with dopamine. Considering the severity of the patient's condition, he was admitted to the Anesthesiology and Resuscitation Unit of the Tver Municipal Clinical Hospital No. 7 with the diagnosis *poisoning by unspecified substance*. At the pre-hospital stage, cerebral infarction was excluded by the results of computed tomography (CT) of the head. During the patient's stay at the intensive care unit for three days, laboratory tests were made. Biochemical blood

assay showed an increase in the concentration of urea nitrogen (from 8.1 mmol/L to 19.3 mmol/L) and creatinine (from 234 μmol/L to 380 μmol/L). The patient was examined by a nephrologist who diagnosed severe toxin-induced AKI (pre-renal combined with renal) at the stage of anuria. Ultrasonic examination of the abdominal cavity and kidneys revealed diffuse lesions of the parenchyma, pancreas, and kidneys. Thoracic CT revealed disc-like collapses in the inferior lobe of the left lung. Electrocardiography showed sinus node tachycardia, left axis deviation, signs of both ventricles overload, cicatricial changes of the myocardium of the left ventricle posterior wall. Echocardiography revealed diffuse hypokinesia of the left ventricle walls. The ejection fraction was reduced to 50%. Areas of hypokinesia of the left ventricle posterior wall were visualized. Conservative therapy was chosen that included cytoflavin, ademetionine, omeprazole, dexamethasone, Ringer's solution, and semisynthetic penicillins (Klamosar, levofloxacin hemihydrate, and furosemide). Despite the treatment, negative dynamics was observed: the level of azotemia increased and oliguria persisted. Considering the necessity to start hemodialysis, the patient was transferred to the nephrology unit of the Tver regional clinical hospital.

Within the first five days in the unit, the patient underwent renal replacement therapy with acute hemodialysis, which resulted in positive dynamics in biochemical blood parameters that reflect kidneys functioning: the level of urea nitrogen decreased from 28.0 mmol/L to 15.7 mmol/L; the level of creatinine decreased from 416.0 μmol/L to 240.0 μmol/L; GFR increased from 5.45 ml/min to 29.8 ml/min. The levels of potassium in the blood normalized from 3.4 mmol/L to 4.00 mmol/L. In the same period, hyperglycemia was observed (the level of glucose was up to 8.6 mmol/L). A decreased tolerance to glucose was revealed. Besides, pathological values of biochemical blood parameters that characterize kidney functioning were observed: an elevated level of total bilirubin to 36.2 μmol/L and its direct fraction to 9.0 μmol; a decrease in the total protein to 54 g/L; an increase in the activity of hepatic enzymes, hypoalbuminemia, and hypertriglyceridemia. From the first day of hospitalization, blood coagulation disturbances were revealed: prothrombin index — 36%, international normalized ratio — 1.86 units, D-dimer — 9.68 mg/L. At the beginning of the observation, clinical blood assay showed I degree anemia, leukocytosis, lymphopenia, neutrophilia, thrombocytopenia, erythrocyte sedimentation rate (ESR) up to 60 mm/hour. A common urine test revealed alterations typical for this pathology: low specific gravity, proteinuria to 0.26 g/L, leukocyturia 10–15 in the field of vision, microhematuria, bacteriuria, flat epithelium 3–5 in the field of vision, nebulous urine.

To control the status of vital organs and to reveal comorbid pathology, the patient underwent different instrumental investigations. Thoracic, abdominal, and brain CT revealed hepatomegaly, signs of fatty hepatosis, and dyscirculatory encephalopathy associated with cerebral atherosclerosis. An electrocardiogram showed sinus node tachycardia, horizontal axis deviation, and disturbance of repolarization in the inferior wall of the left ventricle. Abdominal and kidney ultrasonic examination revealed diffuse lesions of hepatic parenchyma and pancreas, diffuse lesions of both kidneys, and kidney cysts. The patient was examined by a gastroenterologist who diagnosed nonspecific reactive hepatitis associated with moderate steatosis and pancreatic lipomatosis. A psychiatrist diagnosed toxic encephalopathy.

Within the period of hospitalization (two weeks), apart from renal replacement therapy, the patient received multicomponent pharmacotherapy: losartan 50 mg in the morning daily; omeprazole 20 mg twice a day; insulin therapy when the level of glucose elevates higher than 11 mmol/L 3–4 units s/c; aminophylline 2.4–10.0% i.v. by drop infusion of 250 ml of saline solution daily; choline alfoscerate 4.0 i.v. by drop infusion of 250 ml of saline solution daily; furosemide 60 mg i.v. bolus daily with a decrease in the dose to 20 mg; Remaxol 400 mg i.v. by drop infusion daily, potassium chloride 4–30.0% i.v. by drop infusion of 250 ml of saline solution daily, Meldonium 5.0 i.v. by drop infusion N. 10; saline solution 500 ml i.v. by drop infusion daily for the first ten days.

Within two weeks, the indicated therapy led to the normalization of the level of blood urea nitrogen. The level of creatinine decreased to 141 μmol/L, GFR increased to 56.42 ml/min/1.73m^2. The level of transaminases also normalized. However, hypopotassaemia, hypoalbuminemia, hypoproteinemia, hyperglycemia, and a decrease in the tolerance to glucose remained. The level of glycated hemoglobin was 6% and there were disturbances in the blood coagulation system. Clinical blood assay still showed II-degree anemia and an increase in the ESR to 79 mm/hour. Clinical restoration of diuretic activity was observed. Further, the patient was transferred to the therapeutic unit of the Tver municipal clinical hospital No. 7 for the correction of anemia and treatment of comorbid pathology. The patient was transferred to the hospital with the diagnosis of severe toxin-induced acute kidney injury at the stage of restoration of diuresis. Renal replacement therapy with hemodialysis was performed daily from June 6, 2020 to June 10, 2020. Stage II anemia.

Comorbid diseases: moderate non-specific reactive hepatitis associated with liver steatosis. Pancreatic lipomatosis. Stage III hypertensive disease, II-degree arterial hypertension, risk 4. Ischemic heart disease: postinfarction cardiosclerosis of undetermined time of onset. Diabetes mellitus type II with multiple complications. Recommended level of glycated hemoglobin is less than 7.5%. Stage II encephalopathy of complex (toxin-induced and dyscirculatory) genesis with moderately expressed vestibular impairments and psychoorganic syndrome.

DISCUSSION

On the one hand, the described clinical case demonstrates classic development of toxin-induced AKI with anuria when conservative therapy was ineffective. On the other hand, it shows that timely indicated hemodialysis led to the restoration of the kidney functioning. It should be noted that the patient had numerous comorbid diseases that were not controlled by the patient. This was an aggravating factor for the development of the main disease. Besides, this clinical case shows that in patients with surrogate alcohol intoxication, kidney injury is combined with toxin-induced liver injury.

Up to now, the diagnosis and treatment of AKI remain a challenging task. The severity of AKI and its complications are determined by the timely diagnosis and complete volume of treatment measures, including renal replacement therapy. Patients with the developed AKI can be placed under the care of doctors practicing in a few specialties because its manifestations are diverse. We believe that dissemination of the knowledge and experience in the diagnosis and treatment of AKI will help doctors of primary care timely recognize patients at risk.

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