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BISMUT-INDUCED ONSET OF ACUTE TUBULOINTERSTICIAL NEPHRITIS: A CLINICAL CASE

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ABSTRACT — This article presents a clinical case of drug-induced acute TIN that developed in response to self-administered intake of bismuth tripotassium dicitrate. This clinical case was characterized by an acute onset, presence of urinary syndrome (low specific gravity of the urine and proteinuria), occurrence of azotemia in the setting of preserved diuresis, lack of hyperkaliemia, and quick and complete restoration of renal functioning after the discontinuation of a bismuth-containing drug. It can be suggested that the presented clinical case will allow for a better understanding of the causes and clinical features of acute TIN and the choice of the management tactics for patients with this pathology.

KEYWORDS — tubulointerstitial nephritis (TIN), medicinal nephritis, acute kidney damage, chronic kidney disease, bismuth trisalium citrate.

BACKGROUND

Drug-induced renal injury is one of the most acute problems in modern nephrology that is caused by the development of tubulointerstitial nephritis (TIN). As a rule, acute TIN is the main cause of *unspecified kidney injury* in patients with preserved diuresis and normal kidney sizes [1–5, 8]. TIN is an inflammatory condition of renal interstitium and tubules that is associated with the development of acute renal injury in 10–25% of cases. In 20–40% of cases, it leads to the development of chronic kidney disease (CKD). One of the drugs, which administration can be complicated by the development of TIN, is bismuth tripotassium dicitrate that exerts anti-ulcerous and anti-inflammatory effects combined with bactericidal activity against Helicobacter pylori. This drug is widely used in the treatment of ulcerous disease and chronic gastroduodenitis in the exacerbation phase [6–13]. This drug contains bismuth oxide that is primarily eliminated by kidneys. It can be suggested that the expansion of the knowledge on the peculiarities of this pathology can contribute to its timely diagnostics.

The study aimed to describe a clinical case of drug-induced acute TIN that developed in response to intake of bismuth tripotassium dicitrate and expand the doctors' knowledge about this pathology for the prevention, timely diagnostics, and treatment of acute TIN.

MATERIALS AND METHODS

The authors reviewed medical publications on clinical recommendations on drug-induced TIN for the past decade and analyzed the medical history of a patient from the Nephrology Department of the Tver Regional Clinical Hospital (Russia).

CASE DESCRIPTION

Patient K., 56 years old, female, was admitted to Nephrology department of the tertiary hospital complaining about pains in the epigastrium, feeling of abdominal borborygmi and bloating, loose stool up to 2–3 times per day, bilateral lumbar pain, and nausea.

Antibiotics were not prescribed in this case, because the patient self-medicated herself recognizing her symptoms as gastritis. She decided to consult a doctor only after taking the drug during 4 days, when her condition began to deteriorate.

The patient said that the condition developed quickly. The symptoms intensified within several days. The patient began to take bismuth tripotassium dicitrate 2 tablets twice a day without prescription. On day 4 after the beginning of the drug intake the patient developed face edema. The daily urine output decreased. During the examination at the local clinics, biochemical blood assay showed elevated levels of creatinine to 889.5 μmol/L and BUN to 21.6 μmol/L. The specified condition was defined as acute renal injury developed on day 4 after the drug intake. On day 6, the patient was hospitalized.

The patient's condition was satisfactory at admission to the hospital. Face edema and pastosity of the lower third ankles and feet were observed. The pulse was 80, rhythmic. BP was 16090 mmHg. The abdomen is soft, slightly painful in the epigastrium. The kidneys were not palpable. The diuresis was 1.0 L.

The clinical blood analysis revealed light normochromal anemia (hemoglobin 105 g/L), anisocytosis, light thrombocytopenia (167×10^9/L), eosinopenia,

ESR acceleration to 27 mm/h. Common urine analysis showed urine low specific gravity (1008), insignificant proteinuria (to 0.06 g/L), a large amount of flat epithelium, and 2–4 leukocytes in FOV. Biochemical blood assay at admission showed hyperazotemia (creatinine — 850 μm/L, BUN — 15 mmol/L). Ultrasonic investigation revealed diffuse alterations in the renal parenchyma with an increase in their size. ECG registered sinus bradycardia, horizontal electrical axis of heart, and signs of left ventricular overload. Esophagogastroduodenoscopy revealed esophagitis, mixed gastritis, duodenogastric reflux, and indirect signs of cholecystopancreatitis. EchoCG protocol showed insignificant hypertrophy of the basal area of the interventricular septum, left ventricular diastolic dysfunction, and preserved ejection fraction. Gastroenterologist and nephrologist examined the patient and evaluated the necessity of urgent hemodialysis. However, considering preserved diuresis, positive dynamics of creatinine levels decrease in the blood, and satisfactory condition, conservative management of the patient was chosen.

Clinical diagnosis was made based on the results of laboratory-instrumental tests. The primary diagnosis was drug-induced acute tubulointerstitial nephritis developed after intake of bismuth tripotassium dicitrate. Complications: moderate acute renal injury. Comorbid diagnosis: gastro-esophageal reflux disease: I degree esophagitis. Chronic gastroduodenitis in the exacerbation phase. Fatty hepatosis. Pancreatic lipomatosis. Hypertensive disease, II degree, high risk.

In the Nephrology Department, the patient received conservative therapy that included discontinuation of bismuth tripotassium dicitrate, indication of euphylline infusions (2.4% — 10.0 i/v drop infusion BID, daily), omeprazole (40 g i/v drop infusion before dexamethasone infusion), dexamethasone (16 mg i/v drop infusion daily with a gradual dose decrease to complete discontinuation), prednisolone (20 mg per os starting a week after the beginning of treatment at hospital), furosemide (40–60 mg i/v). During the therapy, the patient's condition improved and positive dynamics of laboratory parameters were observed.

At the patient's discharge a significant decrease in the levels of creatinine (to 150 μmol/L) and BUN (to 11 μmol/L) were observed. Clinical blood analysis showed normalization of hemoglobin, erythrocyte, and platelet levels, and a decrease of ERS to 16 mm/h. A common urine test revealed insignificant proteinuria (0.03 g/L). For outpatient therapy, it was recommended to take antiaggregants (pentoxifylline) 100 mg BID, per os, for 1 month. 1 month after the discharge, levels of creatinine and BUN were normalized.

DISCUSSION

The presented clinical case included the major manifestations of drug-induced acute TIN complicated by acute kidney injury developed in response to self-medication of bismuth tripotassium dicitrate. The clinical features of this case included acute onset (4 days after the self-medication), lack of oligo-anuria, presence of urinary syndrome (low specific gravity of the urine and proteinuria), increase in the level of creatinine in the blood with preserved diuresis, lack of hyperkaliemia, and quick and complete restoration of renal functioning after discontinuation of the bismuth-containing drug [10–13].

It can be suggested that the presented clinical case will allow better understanding of the causes and clinical features of acute TIN and the choice of management tactics for patients with this pathology.

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