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Letters to the Editor

Hypoxic hepatitis as diabetic ketoacidosis complication

Key words: Hypoxic hepatitis. Ischaemic hepatitis. Diabetic Keoacidosis.

Dear Editor,

Hypoxic hepatitis (HH) (also known as ischemic hepatitis or shock liver) is characterized by a dramatic but transient rise in serum concentrations of hepatic enzymes caused by anoxic necrosis of centrilobular liver cells (1). Although the pathogenesis is multifactorial –cardiac failure, respiratory failure and toxic-septic shock– (2), the final common pathway is the hepatocellular dysfunction secondary to low levels of oxygen for metabolic processes.

Case report

A 39-year-old man, with type 1 diabetes mellitus (T1DM) and dyslipidemia (taking simvastatin 20 mg/day for the last 10 years), went to emergency department. He was agitated and sweating profusely, with severe vomits. Serum glucose was 534 mg/dl and pH 6.91, additionally to ketone bodies in urine, so diabetic ketoacidosis (DKA) was diagnosed. He was treated with intravenous fluids and soluble insulin, normalizing the metabolic disturbances. According to liver parameters, ALT (447 U/L), AST (1,649 U/L) and LDH (378 U/L) were raised. Additionally to DKA, drug-induced liver injury (DILI) by simvastatin was suspected, so the patient was derived to Hepatology Department. We excluded viral infection (hepatitis A, B and C, cytomegalovirus, Epstein-Barr virus and HIV) and autoantibodies (including anti-mitochondrial antibody, anti-smooth muscle antibody, liver kidney microsomal type 1 antibody and antinuclear antibodies). Mild liver steatosis was found in ultrasound. Furthermore, CIOMS scale showed 2 points (improbable for DILI). A week after hospital admission, patient left hospital asymptomatic with laboratory values slightly impaired (ALT 297 U/L, AST 172 U/L, LDH 248 U/L), returning to normal fifteen days later.

Discussion

HH is recognized as the most frequent cause of acute liver injury. The presumed mechanism of hepatic injury is one of following: a) Drop in hepatic blood flow; b) passive congestion of the liver; and c) arterial hypoxemia. A transient (5-25 days) and marked (10-300 times the upper limit of normal) change in serum enzyme levels is usual. Although the histopathological finding is the essential element of HH, liver biopsy is usually not required for diagnosis. According to underlying conditions leading to HH, congestive heart failure, acute cardiac failure, exacerbated chronic respiratory failure and septic shock have been documented (3).

DKA is a potentially life-threatening complication in patients with DM, especially in T1DM. Clinical symptoms are nausea and vomiting, pronounced thirst, polyuria and abdominal pain. On the other hand, high levels of ketone bodies are associated with high serum and urine glucose levels, resulting in frequent urination. Combined with vomiting, the body quickly loses water and electrolytes (4). In our case, the raising of liver enzymes resulted from hypovolemia. This mechanism is closely related to severe hemodynamic change after the drop of hepatic blood flow which leads to hypoperfusion and hypoxia of the liver.

This case represents an interesting diagnostic challenge, due

to the patient was diagnosed of drug-induced liver injury when suffered from hypoxic hepatitis by diabetic ketoacidosis. We should to take into account this clinical entity to improve the poor prognostic of these patients.

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