A 36-year-old man with severe alcoholic hepatitis was treated with plasma exchange combined with hemodiafiltration to remove endotoxins and inflammatory cytokines. During the treatment, he had critical arrhythmia (torsade de pointes [Tdp]). His laboratory data showed hypomagnesemia, which was suspected to be responsible for the development of Tdp. Patients with alcoholic liver disease tend to have hypomagnesemia and Q-T interval prolongation. Furthermore, hemodiafiltration may cause hypomagnesemia. Careful observation for electrolytic imbalance is necessary when clinicians treat patients with alcoholic liver failure with a liver support system.

Key words: alcoholic liver cirrhosis, torsade de pointes, hypomagnesemia

Introduction

Torsade de pointes (Tdp) is a rare, life-threatening arrhythmia, which is usually associated with a long Q-T interval. Hypokalemia and hypomagnesemia may cause Tdp. These conditions sometimes occur in alcoholic patients. We report a patient with acute exacerbation of alcoholic liver cirrhosis complicated with Tdp during plasma exchange therapy (PE) combined with hemodiafiltration (HDF).

Case report

A 36-year-old man was admitted to our hospital on February 15, 1998, because of jaundice, watery diarrhea, fever, poor appetite, and general fatigue. He had no unusual past history or any heart diseases. He had drunk about 80 g/day of alcohol for 15 years. Furthermore, for several months before admission, he had been drinking about 80–160 g/day of alcohol. Physical examination revealed a clear consciousness level, but the palpebral conjunctiva showed anemia and the bulbar conjunctiva showed icterus. His breathing sounds in both lungs were normal and his heartbeat was regular without murmurs. Abdominal examination revealed an enlarged liver, the edge of which was palpable four finger widths below the right costal margin. His legs showed no pretilial pitting edema.

Laboratory data were as follows: peripheral white blood cell count, 26.3 × 10^9/µl (normal range [NR], 3.9–9.8 × 10^9); hemoglobin, 11.1 g/dl (NR, 13.5–17.6 g/dl); hematocrit, 31.2% (NR, 39%–52%); platelets, 37.3 × 10^9/µl (NR, 13.1–36.2 × 10^9); prothrombin time, 45.6% (NR, 70%–120%); total serum bilirubin (T-bil), 28.3 mg/dl (NR, 0.2–1.2 mg/dl); direct serum bilirubin (D-bil), 12.9 mg/dl (NR, 0.2–1.2 mg/dl); alanine aminotransferase (ALT), 34 IU/l (NR, 7–42 IU/l); aspartate aminotransferase (AST), 125 IU/l (NR, 10–34 IU/l); alkaline phosphatase (ALP), 1616 IU/l (NR, 110–355 IU/l); ß-glutamyltranspeptidase (ß-GTP), 1031 IU/l (NR, 7–45 IU/l); Na, 126 mEq/ml (NR, 138–146 mEq/ml); K, 2.1 mEq/ml (NR, 3.5–4.9 mEq/ml); and Cl, 82 mEq/ml (NR, 98–108 mEq/ml). Serum immunoglobulin levels were as follows: IgG, 1590 mg/dl (NR, 800–1800 mg/dl); IgA, 756 mg/dl (NR, 150–310 mg/dl); and IgM, 161 mg/dl (NR, 80–230 mg/dl). Testing for antinuclear antibodies showed a negative result. Both hepatitis B surface antigen and antibody to hepatitis C virus (HCV) were negative.

Abdominal ultrasound (US) examination demonstrated an enlarged liver, moderate ascites, and a thickened gallbladder wall, but no splenomegaly. Computed tomography showed an enlarged liver, minimal ascites around the liver surface, and a dilated umbilical vein.
which indicated portal hypertension (Fig. 1). Radioisotope scanning (99mTc-labelled tin colloid of human albumin) revealed a decreased isotope uptake (Fig. 2).

We concluded that the patient's excessive alcohol intake before admission had induced exacerbation of liver dysfunction, and we diagnosed acute exacerbation of liver cirrhosis on the basis of the above findings.

After admission, he continued to complain of frequent watery diarrhea. Although, stool culture was negative, lipid staining of stool was positive.

His serum level of endotoxin (Et) was high (18.6 pg/ml; NR, <10 pg/ml). His serum interleukin-8 (IL-8) level was also high (197 pg/ml; NR, <15 pg/ml), but serum tumor necrosis factor-alpha (TNF-α) level was normal. Because his laboratory data showed severe liver dysfunction, glucagon-insulin therapy was administered for 4 days. However, his liver function did not improve. Therefore, PE combined with HDF was administered to remove the Et and inflammatory cytokines. However, during this combination therapy, he had critical arrhythmia (torsade de pointes [TdP]), and his heart began arresting, on March 4, 1998 (Fig. 3). Cardioversion was performed immediately. Electrolyte levels in the serum sample taken before the occurrence of TdP were as follows: Na, 129 mEq/ml; K, 3.5 mEq/ml; Cl, 102 mEq/ml; and magnesium (Mg), 1.26 mg/dl (NR, 1.45–2.71). Hypomagnesemia was suspected to be responsible for the development of TdP. After this episode of TdP, we administered magnesium sulfate to maintain the Mg level within the normal range (Fig. 4).