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Severe hyperglycemic shock associated with hepatic portal venous gas

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Abstract
We report a case of hyperglycemic shock associated with hepatic portal venous gas. A 79-year-old woman with post-stroke depression developed severe tachycardiac atrial fibrillation and hypotension due to hypovolemia caused by severe hyperglycemia, as well as showing disseminated intravascular coagulation (DIC). Continuous intravenous infusion of insulin and volume loading with normal saline gradually achieved normalization of the serum glucose level and hemodynamic stability. However, the DIC did not resolve, and abdominal computed tomography (CT) revealed hepatic portal venous gas (HPVG) in the left lobe of the liver. Surgery was thus considered mandatory. However, because severe hemodynamic lability occurred again immediately after the CT examination, and persisted, surgery could not be performed, and the patient died of septic shock due to bowel perforation. It was concluded that the underlying causes of DIC should be sought promptly, without delay.

Key words Hyperglycemia · Hypovolemic shock · Hepatic portal venous gas

Introduction

Hepatic portal venous gas (HPVG) occurs in a variety of clinical settings and pathological conditions, such as bowel ischemia/necrosis, bowel distension, intra-abdominal sepsis [1,2], and long-term hemodialysis [3]; the presence of this finding often suggests a poor prognosis [1,2]. We present a case of severe hyperglycemic shock associated with HPVG.

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Case report

A 79-year-old woman (height, 142 cm; body weight, 35 kg) who had post-stroke depression was transferred to the intensive care unit (ICU) with the aim of managing disturbance of consciousness, disseminated intravascular coagulation (DIC), and severe hemodynamic lability. She had undergone a gastrectomy 15 years before and had presented with diabetes mellitus (DM), hypertension, atrial fibrillation, and depression during the 8 years before the present admission. The occurrence of right hemiparalysis after a cerebral infarction 2 months before this admission had made her more depressive. Oral medications given after the diagnosis of post-stroke depression included milnacipran ($50\,mg\cdot day^{-1}$) and mianserin ($10\,mg\cdot day^{-1}$). Although these oral treatments were discontinued after 3 days (6 weeks before the ICU admission), because they had tended to make her stuporous, her level of consciousness had not changed after discontinuation of this medication. However, there were no abnormal findings on cerebral computed tomography (CT) and magnetic resonance imaging (MRI). On admission to the ICU, neurological examination revealed that she had a Glasgow coma scale of grade 3 (E1, V1, M1) with pupils unreactive to light. Physical examination showed respiratory rate, 40–45 breaths per min; arterial blood pressure (ABP), 55/36 mmHg; heart rate (HR), 140–180 bpm (tachycardiac atrial fibrillation); central venous pressure (CVP), $-1\,cmH_2O$; and urine output, $0–10\,ml\cdot h^{-1}$. Laboratory examinations showed a severe rise in fasting blood sugar (FBS), at $1290\,mg\cdot dl^{-1}$ and serum osmotic pressure of $416\,mOsm\cdot l^{-1}$. A specimen of arterial blood revealed that pH was 7.32; oxygen partial pressure, 99 mmHg; carbon dioxide partial pressure, 47 mmHg; and base excess, $-2.4\ mEq\cdot l^{-1}$ under supplemental oxygen given at a rate of $5\,l\cdot min^{-1}$ via a face mask. Serum electrolyte values were: sodium, $152\,mEq\cdot l^{-1}$; and potassium, $5.7\,mEq\cdot l^{-1}$. Coagulation tests disclosed:

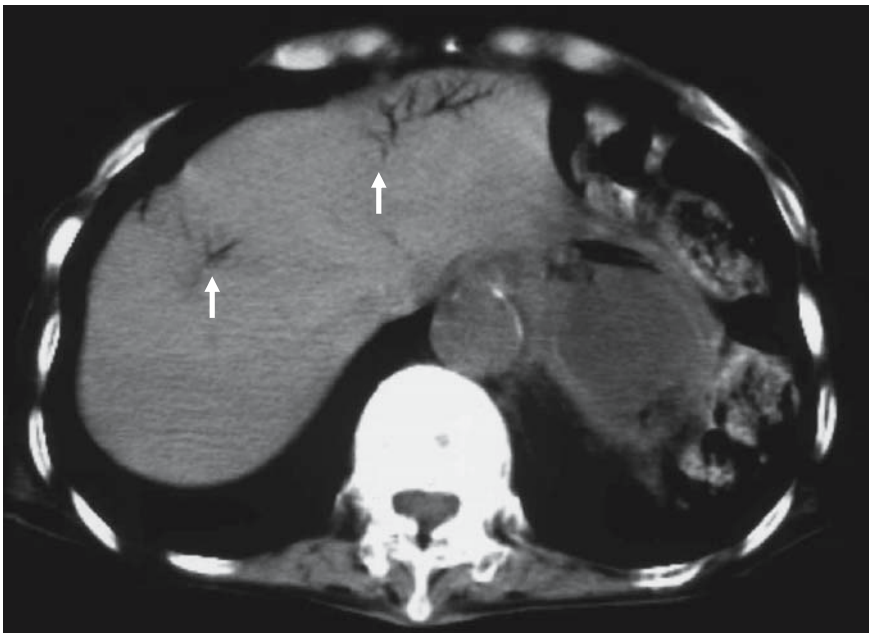

Fig. 1. Hepatic portal venous gas. Unenhanced computed tomography (CT) scan 36 h after admission to the intensive care unit (ICU) showed branched areas of decreased attenuation in the left hepatic lobe (*arrows*)

platelet count, 70 000 per mm^3; prothrombin time, 64% of normal; and fibrine/fibrinogen degradation products (FDP), 43.5 $\mu g \cdot ml^{-1}$. These clinical findings and laboratory data suggested marked dehydration due to severe hyperglycemia, and the presence of systemic inflammatory response syndrome (SIRS) and DIC.

After admission to the ICU the patient received a rapid infusion of 300 $ml \cdot h^{-1}$ of normal saline. In addition to the loading dose of intravenous fluid, she received a continuous intravenous infusion of 10 units$\cdot h^{-1}$ of insulin. Fifteen hours after the insulin infusion, as the FBS values had decreased to 250 $mg \cdot dl^{-1}$, the administration of insulin was discontinued. At this time, intravenous infusion of normal saline was maintained at 200–300 $ml \cdot h^{-1}$ because the hypovolemia had not yet resolved (ABP, 80/60 mmHg; HR, 140–160 bpm; CVP, 0 cmH_2O; and urine output, 10–20 $ml \cdot h^{-1}$). Over 20 h after the application of the rapid volume loading, ABP was kept at 100–120/60–80 mmHg and HR was maintained at 100–140 bpm, and hemodynamics gradually tended to improve. However, because the DIC did not resolve despite the resolution of the hyperglycemia and the hemodynamic instability, the causes of the DIC were reevaluated. Physical examination revealed a distended abdomen with decreased bowel sounds 22 h after ICU admission, and abdominal unenhanced CT scan at this time revealed the presence of HPVG (Fig. 1). The HPVG showed a pattern of branching radiolucency extending almost to the periphery of the left lobe of the liver. Signs of bowel perforation were not detected by CT scan at this time. Although it was considered that surgery was mandatory, the patient again developed marked hypotension immediately after return to the ICU from the CT examination, and the families' consent to surgery could not be obtained; the patient was therefore treated conservatively. Fifty hours after the patient's admission to the ICU, although she received an hour of cardiopulmonary resuscitation because of cardiopulmonary arrest, she died.

Autopsy was not carried out because written informed consent could not be obtained from the families. An hour after death (52 h after admission to the ICU), virtual autopsy imaging by CT scan, performed as an alternative to autopsy to determine the cause of death [4], detected extraluminal air in the peritoneal cavity (Fig. 2), suggesting bowel perforation.

Discussion

Hepatic portal venous gas (HPVG) is a rare condition that is often associated with extensive bowel necrosis, and has a fatal outcome [1,2]. Although the etiology of HPVG has been unclear, Liebman et al. [1] reported that the causes of HPVG included: (a) mucosal damage such as that due to inflammatory bowel disease and mesenteric ischemia; (b) gastrointestinal distension of spontaneous, traumatic, and iatrogenic causes; and (c) intraabdominal sepsis. As for the causes of bowel ischemia, Butler et al. [5] suggested that shock-induced circulatory disturbances in the superior mesenteric artery (SMA) could produce bowel ischemia and/or necrosis. Our patient had presented in a state of hypovolemic shock secondary to severe hyperglycemia on admission to the ICU. Therefore, it was speculated that bowel necrosis due to circulatory depression had

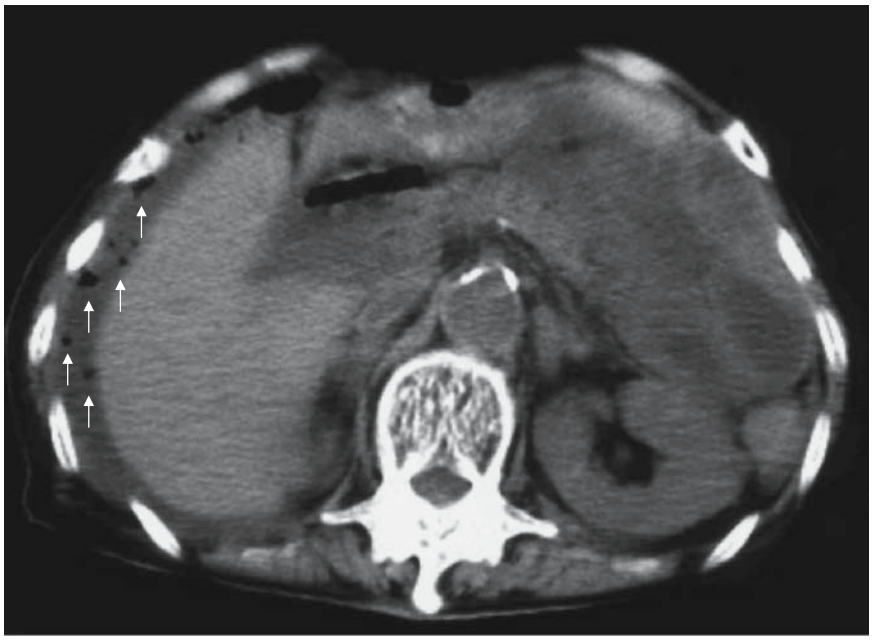

Fig. 2. Pneumoperitoneum. Unenhanced CT scan 74 h after the patient's admission to the ICU revealed extraluminal air in abdominal ascites in the peritoneal cavity (*arrows*)

caused the HPVG, although autopsy findings were not obtained.

Upon arrival in the ICU, although the patient had already presented with DIC, close examination of her abdominal condition to determine the cause of the DIC was not performed, because the treatment of severe hyperglycemia, hypovolemic shock, and tachycardiac atrial fibrillation took precedence over all other procedures for saving her life. In this patient, the absence of complaints of abdominal pain or discomfort because of the patient's reduced level of consciousness may also have delayed the decision to perform an abdominal examination. The decision to manage the severe hyperglycemia and hypovolemia may have been valid in consideration of the possibility of their contribution to the occurrence of DIC. However, bowel ischemia or necrosis with HPVG has a poor prognosis, with a mortality rate of 75%–90% [1,2]. Therefore, when abdominal CT demonstrates HPVG and clinical fidings suggest the presence of bowel ischemia or necrosis, prompt surgical management is mandatory. In our patient, however, DIC was the only finding suggestive of the presence of severe underlying disease immediately after her ICU admission, because the marked hemodynamic lability prevented the performance of CT, and subjective symptoms of the patient, such as abdominal pain, were not available, due to the patient's reduced level of consciousness. This case leads us to the conclusion that the underlying cause of DIC should be sought promptly, without any delay.

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