Posterior reversible encephalopathy syndrome during severe acute pancreatitis

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a relatively rare central nerve condition. Here, we report a case of PRES that developed during acute pancreatitis in a previously healthy man in his 40s. On the third day, his respiratory status worsened during management of acute pancreatitis, requiring sedated ventilatory support. During ventilator management, his blood pressure was well-controlled, and no electrolyte abnormalities were observed; however, the patient was in a state of marked systemic inflammation. On the 12th day, after improvement in acute pancreatitis and respiratory status, with discontinuation of mechanical ventilation, the patient presented with mild disturbance of consciousness and visual field disturbance. Magnetic resonance imaging (MRI) showed diffuse vasogenic cerebral edema, mainly in the bilateral occipital and parietal white matter. The patient was provisionally diagnosed with PRES and was administered oral verapamil, along with supportive care. On the 23rd day, his neurological symptoms disappeared. Cerebral edema completely disappeared on MRI four months later. In the present case, it was speculated that endothelial dysfunction secondary to the severe inflammatory state accompanying acute pancreatitis caused PRES.

Keywords: acute pancreatitis; endothelial dysfunction; inflammation; posterior reversible encephalopathy syndrome

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a relatively rare central nerve condition that commonly causes headaches, blurred vision, seizures, and impaired consciousness. Preeclampsia/eclampsia, autoimmune diseases, cancer chemotherapy, transplantation, infection/sepsis/shock, and hypertension are known conditions associated with PRES.[1] Acute pancreatitis often has a serious course, and various complications, including cranial nerve system complications, have also been reported. There are some reports of PRES associated with acute pancreatitis, which in turn is associated with the conditions mentioned above.[2,3] However, there are few reports of PRES associated with acute pancreatitis in the absence of such conditions. We report a case of PRES during acute pancreatitis without specific conditions associated with PRES.

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Case Report

A 40s man without a specific past medical history did not receive any medication had epigastric pain for approximately two weeks and consulted a nearby clinic. Ultrasonography revealed pancreatic swelling, and the patient was diagnosed with acute pancreatitis. The patient was then referred to our emergency department. The patient was alert and showed no neurological symptoms. His blood pressure was 140/90 mmHg. Laboratory assessments showed increased levels of hepatic and pancreatic enzymes and triglycerides (aspartate aminotransferase, 61 U/L; alanine aminotransferase, 90 U/L; γ-glutamyl transpeptidase, 160 U/L; amylase, 1129 U/L; lipase, 3938 U/L; triglyceride, 3420 U/L). At the time of hospitalization, there were no observations of renal dysfunction or electrolyte abnormalities. Abdominal computed tomography revealed mild swelling of the pancreas, and a small amount of ascites. We initiated treatment for acute pancreatitis with crystalloid fluid infusion, gabexate

mesylate (39 mg/kg/day), and ulinastatin (300,000 units/day). Rosuvastatin (2.5 mg/day) and bezafibrate (400 mg/day) were administered orally for hypertriglyceridemia. On the third day, because of worsening respiratory distress, orotracheal intubation was performed and mechanical ventilation was initiated under midazolam sedation. During mechanical ventilator management, his blood pressure was well-controlled (Figure 1). The patient appeared to be in a highly inflammatory state from the second to the ninth day (Figure 1).

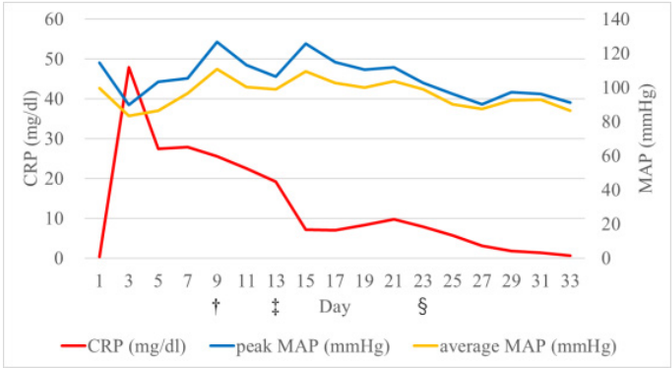

Figure 1: Changes in C-reactive protein levels and mean arterial pressure

peak MAP, highest mean arterial pressure of the day; average MAP, average mean arterial pressure over 24 hours
†: Improvement in pancreatic enzymes; ‡: Diagnosis with vasogenic cerebral edema; §: Neurological symptoms disappear
Abbreviations: MAP, mean arterial pressure; CRP, C-reactive protein

On the ninth day, the pancreatic enzyme levels and respiratory condition improved. Despite the discontinuation of sedation, the patient did not awaken. The patient awoke on the 12th day and was extubated. After extubation, his consciousness level was determined using the Glasgow Coma Scale (E4V4M6), and visual field impairment was observed. On the 13th day, magnetic resonance imaging (MRI) of the head was performed and suggested diffuse vasogenic cerebral edema (Figure 2).

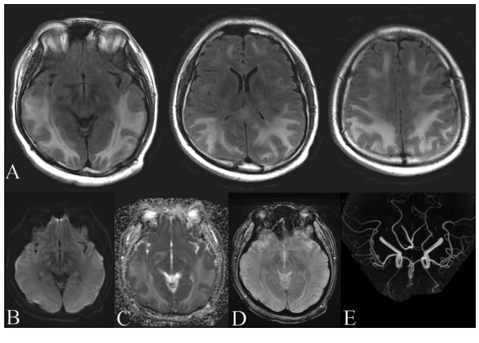

Figure 2: Magnetic resonance images on the 13th day

(A) Fluid-attenuated inversion recovery (FLAIR) shows hyperintensity in the subcortical region and the deep white matter symmetrically in the bilateral cerebral hemispheres, which is predominant in the bilateral occipital, parietal and temporal lobes. (B) Diffusion-weighted imaging shows no restricted diffusion in the FLAIR hyperintense area. (C) The apparent diffusion coefficient is elevated in FLAIR hyperintense area. (D) T2* weighted image shows a large number of cerebral microbleeds in the cortical and subcortical region, which is predominant in the bilateral occipital, parietal and temporal lobes. (E) Magnetic resonance angiography shows a diffuse, slight vasoconstriction.

We suspected PRES based on MRI findings. Supportive care was provided to the patient. On the 23rd day, his visual field impairment completely recovered and he became conscious (Glasgow Coma Scale (E4V5M6)). There was no recurrence of neurological symptoms thereafter. Follow-up MRI on the 24th day showed improvement in cerebral edema. The patient was finally diagnosed with PRES because vasogenic cerebral edema and neurological symptoms had improved after the treatment described above. Cerebral edema on follow-up MRI completely disappeared four months later (Figure 3). Subsequently, there has been no recurrence of cerebral edema or neurological symptoms.

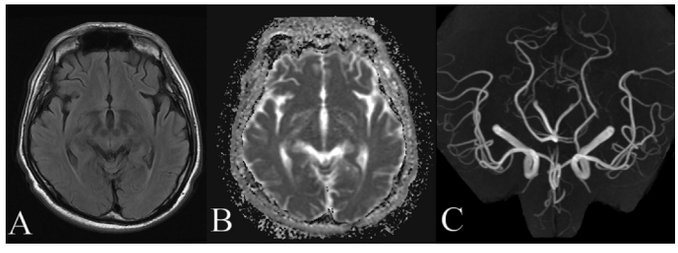

Figure 3: Magnetic resonance images obtained 4 months later

(A) Fluid-attenuated inversion recovery images shows no hyperintense areas. (B) Apparent diffusion coefficient normalizes in previously FLAIR hyperintense areas. (C) Magnetic resonance angiography shows improvement of vasoconstriction.

Discussion

It is commonly hypothesized that PRES arises either from vasogenic cerebral edema due to endothelial dysfunction caused by severe hypertension exceeding the limits of cerebral autoregulation (mean arterial pressure >140 to 160 mmHg), or from vasogenic cerebral edema secondary to cerebral hypoperfusion resulting from hypertension-induced autoregulatory vasoconstriction.[4,5] PRES is observed in the absence of hypertension in 20 to 40% of patients.[1] In the present case, no significant hypertension was observed (Figure 1). No severe vasoconstriction was observed on MR angiography (MRA) (Figure 2(E), 3(C)). Bartynski et al. reported that MRA or catheter angiography showed no vascular abnormalities in 13% of 46 patients.[6] Healthy vessel appearance is more frequently observed in normotensive patients.[6] Luo et al. noted from a literature review that PRES associated with acute pancreatitis mostly occurs in patients with no severe hypertension.[7] Among PRES-related conditions, T-cell activation and inflammatory cytokine production (tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1, interferon-alpha, and IL-6) are common.[4,5] Cytokines (TNF-α and IL-1) trigger endothelial dysfunction, and vasogenic cerebral edema.[4,5] In acute pancreatitis, the blood levels of inflammatory cytokines such as TNF and IL-6 are elevated,[8] and reportedly, the more severe acute pancreatitis, the

higher the blood level of TNF-α.[9] In the present case, endothelial dysfunction due to inflammatory cytokines in severe acute pancreatitis may lead to vasogenic cerebral edema.

Conclusion

It is speculated that the severe inflammatory state accompanying acute pancreatitis increases the possibility of PRES development. However, many cases of acute pancreatitis do not develop PRES, so there must be other unknown factors. Further research with more case studies is necessary to elucidate the pathophysiology of PRES during acute pancreatitis.

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