Hemolytic-uremic Syndrome Complicating Acute Pancreatitis

Narinder Pal Singh, Neeru P. Aggarwal, Hardik R. Shah, Laxmi Kant Jha, Anish Kumar
Department of Internal Medicine and Nephrology, Max Super Speciality Hospital, Ghaziabad, Uttar Pradesh, India

Abstract

Hemolytic-uremic syndrome (HUS) is characterized by acute kidney injury with hemolytic anemia and thrombocytopenia. It has diverse etiologies, clinical manifestations, and risk factors. Acute pancreatitis as a cause of HUS is rare in adults. We report a case of 32-year-old male who presented with ethanol-induced acute pancreatitis complicated with hemolytic-uremic syndrome managed with hemodialysis and plasmapheresis.

Keywords: Acute kidney injury, hemodialysis, plasmapheresis, thrombotic thrombocytopenic purpura

Introduction

The incidence of acute kidney injury in patients with acute pancreatitis carries a poor prognosis. Hemolytic-uremic syndrome (HUS) is characterized by nonimmune hemolytic anemia, thrombocytopenia, and renal failure caused by platelet thrombi in the microcirculation of the kidney. HUS as a result of alcoholic pancreatitis is scarcely reported. We report a case of alcoholic pancreatitis complicated with renal dysfunction followed by HUS, successfully managed with plasmapheresis after early recognition.

Case Report

A 32-year-old male was admitted to hospital with complaints of abdominal pain, distension, vomiting, and reduced urine output. He had consumed alcohol in a substantial amount at a party, night before the onset of symptoms. He was a known hypertensive, controlled on low-dose amlodipine, otherwise healthy. On examination, the patient was afebrile, pale, dehydrated with tender, distended abdomen, and hypoactive bowel sounds. Ultrasonography of the abdomen revealed mild fatty liver and mild ascites with pancreas obscured by overlying gas-filled loops. Over the next 2 days, he became jaundiced and oliguric. Renal functions deteriorated with increase in urea and creatinine. He had an abrupt decrease in hemoglobin and platelet count and increased in serum bilirubin [Table 1]. Further evaluation revealed raised levels of procalcitonin, normal coagulation profile, and negative stool for occult blood.

His peripheral blood film revealed numerous fragmented red cells and schistocytes.

In view of Coombs-negative hemolytic anemia, thrombocytopenia, and acute renal failure with schistocytes on blood smear, a diagnosis of HUS was made complicating acute pancreatitis. On the 3rd day, he was initiated on plasmapheresis: 2 L of plasma exchange per cycle (30 ml/kg) supplemented with albumin and fresh frozen plasma product transfusions as replacement fluids. In due course, three sessions of hemodialysis and four sessions of plasmapheresis were provided to him on alternate days, also requiring additional two units of blood transfusions. He was managed with empirical intravenous antibiotics and total parenteral nutrition (TPN) for initial 5 days. Culture (blood and urine) reports were sterile.

By the 7th day, there was substantial improvement in clinical condition with significant increase in urine output. Laboratory parameters improved with hemoglobin, increased platelet count, reduction in urea, creatinine, and total bilirubin level. By the 10th day, the patient was shifted out to wards and was discharged in stable condition by the 14th day on oral antibiotics.

Address for correspondence: Dr. Narinder Pal Singh,
Department of Internal Medicine and Nephrology, Max Super Speciality Hospital, Vaishali, Ghaziabad - 201 012, Uttar Pradesh, India.
E-mail: nanusingh58@gmail.com

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How to cite this article: Singh NP, Aggarwal NP, Shah HR, Jha LK, Kumar A. Hemolytic-uremic syndrome complicating acute pancreatitis. Indian J Crit Care Med 2017;21:534-6.
coagulation and also proteolise the fibrinogen resulting in dysfibrinogenemia. It is proposed that in cases of HUS-TTP secondary to pancreatitis, circulating proteases may modify circulating von Willebrand factor (vWF) molecules enabling spontaneous binding to platelet membrane glycoproteins with subsequent platelet aggregation.[8] Serum estimation of vWF-cleaving protease (ADAMTS-13) has been used to differentiate between TTP and HUS as patients with TTP have deficient ADAMTS-13 activity in plasma compared to patients with HUS.[9] However, in our patient, levels could not be measured due to unavailability of test at center.

The treatment of HUS following acute pancreatitis is primarily supportive. Exchange plasmapheresis has been reported to be an effective treatment with a response rate of 79%.[10] Rituximab (anti-CD20 antibody) is an alternative treatment option in refractory or relapsing HUS.[11]

CRRT/SLED is the preferred treatment for acute kidney injury in intensive care units. It removes metabolic waste products and eliminates inflammatory mediators such as IL-1, IL-6, and IL-8 which contributes to early recovery, as in our case.[12,13]

In conclusion, it is important that physicians recognize thrombotic microangiopathy (HUS/TTP) as one of the potential causes of acute renal failure among adult patients with acute pancreatitis, especially in the setting of anemia and thrombocytopenia. Till date, a total of 22 cases have been described to develop HUS/TTP following pancreatitis of varied etiology. Our patient had an excellent response to hemodialysis and plasma exchange with complete resolution of his microangiopathy and recovery of renal function.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**


