Ventricular septal rupture presenting with hyperosmolar hyperglycemic nonketotic coma

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Hyperosmolar hyperglycemic nonketotic coma (HHNC) is a life-threatening emergency manifested by marked elevation of blood glucose, hyperosmolarity, and little or no ketosis. With the dramatic increase in the prevalence of type 2 diabetes and the aging population, physicians may encounter this condition more frequently in the future. Although the precipitating causes are numerous, underlying infections are the most common. Other causes include certain medications, undiagnosed diabetes, substance abuse, acute myocardial infarction (AMI), and coexisting disease. Although the coexistence of AMI with other clinical manifestations of diabetes has been well described, few data exist on the concomitant occurrence of HHNC and myocardial infarctions. This article aims to discuss the clinical course and treatment strategies of this rare condition.

The mortality rate of HHNC is between 10-50%, a considerably higher rate than that of diabetic ketoacidosis. Age, degree of dehydration, hemodynamic instability, underlying precipitating causes, and degree of consciousness all are powerful predictors of a fatal outcome.[1] Coronary artery disease is the leading cause of mortality in patients with diabetes mellitus, which accounts for a considerable amount of comorbid conditions of patients suffering an AMI.[2] At hospital admission, these patients may demonstrate a large clinical spectrum of disease, varying from newly diagnosed diabetes to a diabetic coma. Diabetic ketoacidosis and HHNC are 2 types of life-threatening diabetic comas that are associated with or precipitated by an AMI in rare conditions. Although AMI has been reported to be a frequent cause of death in diabetic ketoacidosis, there is little on the association of HHNC with acute coronary syndromes. Herein, we describe the clinical courses and treatment approach of a patient with ventricular septal rupture after AMI.

A 60-year-old woman was admitted to our emergency department with the complaints of severe shortness of breath and angina radiated to the left arm and hand. No heart disease, hypertension, diabetes mellitus, or drug use was present in her history. She was orthopneic and cyanotic. Blood pressure was 90/60 mm Hg; heart rate was 106 bpm. Jugular venous distention, third heart sound, continue murmur through the left sternal line and inspiratory crepitant pulmonary rales were detected on her physical examination. An electrocardiogram (ECG) showed diagnostic ST segment elevations in DII, DIII, aVF and V5-V6. On echocardiography, ventricular septal rupture, and akinesia in the inferior, posterior and lateral walls of the left ventricle were seen. Blood glucose was 885 mg/dl, creatine kinase, myocardial bound was 99 U/L, and leukocyte count was 16000/mm^3. She was transferred to the coronary care unit with the diagnosis of inferolateral AMI and HHNC. Serum osmolality was 350 mosm/kg. Urine analysis revealed glycosuria associated with only mild ketonuria. Arterial blood gas analysis showed mild acidosis. Thrombolysis was started with streptokinase. However, the patient developed cardiopulmonary arrest during the infusion and did not respond to cardiopulmonary resuscitation. Type 2 diabetes is an important cause of cardiovascular morbidity and mortality accounting for 20% of the total number of patients admitted for suspected myocardial infarction. Patients with diabetes have a 2-fold increase in hospital mortality when compared with those without diabetes. Long-term follow-up reveals a continuously increasing excess mortality, mostly due to fatal re-infarctions and congestive heart failure.[2] Acute MI causes a dramatic increase in adrenergic tone, which stimulates lipolysis, thereby increasing the levels of free fatty acids. Several hormonal mechanisms contribute to a decrease in insulin sensitivity and glucose utilization during acute myocardial ischaemia. Recently, published data have suggested that acute hyperglycemia and poor glycemic control independently contribute to the increased cardiovascular risk of diabetes mellitus.[3] The DIGAMI study,[4] which demonstrated a linear relationship between blood glucose tertiles and long-term mortality, suggested that strict insulin treatment with improved metabolic control seems to reduce the adverse effect of an initially poor metabolic control. Acute hyperglycemia impairs coronary microcirculatory response to myocardial ischemia, attenuates the endothel-dependent (stem from the endothel) vascular response, inhibits nitric oxide production, and increases free oxygen radicals.[4] Both hyperglycemia and hyperosmolality that are characteristic features of HHNC might associate to an increased extent of an AMI. In diabetic and acutely hyperglycemic dogs, infarct size was found to be linearly related to blood glucose concentration.[5] Although hyperosmolality has been reported to increase left ventricular contractility and oxygen consumption, it's independent contribution to the infarct size is controversial, and even a decrease in infarct size by increasing serum osmolality has been observed.[5] Our patient suffering both AMI with ventricular septal rupture and HHNC had a complicated clinical course at the acute phase. Interestingly, diabetes mellitus was not in her history.

In conclusion, underlying precipitating causes should be investigated in patients presenting with HHNC. Mortality seems high in patients presenting with HHNC and AMI. Perhaps, it is necessary to investigate new treatment approaches to reduce mortality in these patients.

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A forgotten complication following pancreatic resection. *Visceral artery pseudo-aneurysms*

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Postoperative bleeding after pancreaticoduodenectomy (PDR), with an incidence of 7.5%, is not an uncommon complication.[1] Most of the bleeders comprised operative field and gastrointestinal ulcers at the anastomotic margin. Visceral artery aneurysms as a bleeding source following PDR, although rare, are clinically important, and potentially lethal without prompt recognition.[2,3] Despite recent advances in therapeutic techniques and diagnostic tools, the management of visceral artery aneurysms following PDR remains clinically challenging. We analyzed the initial presentations, therapeutic interventions, and outcomes of 5 patients with complicating visceral artery aneurysms after PDR, followed by a review of pertinent clinical information. Data were collected retrospectively on all patients receiving PDR and diagnosed with visceral artery aneurysms that occurred between June 2000 and June 2005. **Table 1** lists the demographics and clinical outcomes of these 5 patients. Four patients received one-stage PDR (Whipple's operation) for pancreatic head cancer. Duodenum-preserving pancreatic head resection was deployed in one patient with benign pancreatic lesion. Four out of 5 (80%) patients presented as gastrointestinal bleeding. One patient (20%) presented as bleeding from abdominal drains, and had no additional episode before visceral artery pseudo-aneurysms was proven. The interval from pancreatic resections to definitive diagnosis of visceral artery pseudo-aneurysms was 20.6±7.8 days (range, 10-30 days). Three common hepatic artery, and 2 gastroduodenal artery pseudo-aneurysms were proven in the diagnostic angiography. Common hepatic artery pseudo-aneurysms were treated by coil embolizations of the common hepatic artery (**Figure 1**). Pseudo-aneurysms in the gastroduodenal artery obliterated all feeding arteries as in case 4, or occluded the pseudo-aneurysm per se as in case 5. The only in-hospital death had received PDR for pancreatic cancer and was discharged on post-operative day 14th. The patient returned to the emergency department on post-operative day 26th owing to jaundice, severe upper gastrointestinal bleeding, and hypovolemic shock. Emergency arterial angiography disclosed a common hepatic pseudo-aneurysm, which ruptured into the gastrointestinal tract. Although hemostasis was temporarily achieved by transcatheteric coil embolization, the patient eventually succumbed as a result of multi-organ failure. There were 2 deaths in the follow-up; one died of intra-abdominal abscess in the 3rd month, and the other died of liver abscess in the 22nd month. The mean follow-up (excluded one in-hospital mortality) was 21.25±16.58 months, range 3-43 months.

Brodsky and Turnbull[4] reported a "sentinel bleed" after PDR that was attributed to vessel erosion. Fresh bloody discharge from abdominal drains comprised most initial presentations of "sentinel bleed." Relevant clinical research also had similar observations, with an average presentation of 19 days after PDR.[5] In this