Case Report

Cerebral infarction leading to hemiplegia: A rare complication of acute pancreatitis

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Abstract

Peripancreatic vascular thrombosis is a known complication of acute pancreatitis (AP) and chronic pancreatitis. However, hemiplegia resulting from cerebral infarction due to cerebral arterial thrombosis is a rare complication of AP. Here, we report a case of alcohol related severe AP with multi-organ dysfunction, which was complicated by large left sided middle cerebral artery territory infarct - leading to right sided hemiplegia in a 48-year-old male patient. The neurological and vascular thrombotic complications of pancreatitis, their pathogenesis and management are discussed in brief.

Keywords: Complications, cerebral infarction, hemiplegia, pancreatitis, thrombosis

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Introduction

Systemic hypercoagulability and peripancreatic vascular thromboses are known complications of acute and chronic pancreatitis. Vascular complications occur in approximately a quarter of patients of pancreatitis,[1] adding to the associated morbidity and mortality. These include splanchnic vein thrombosis (SVT) and hemorrhage from direct arterial wall erosion or pseudoaneurysm formation.[1,2] However, cerebral infarction from arterial thrombosis is a rare complication of acute pancreatitis (AP). Here, we report a case of cerebral infarction resulting probably from left middle cerebral artery (MCA) thrombosis in a 48-year-old male patient of alcohol related AP. The neurological and vascular thrombotic complications of pancreatitis are reviewed in brief.

Case Report

A 48-year-old male, heavy alcohol consumer for the prior 6 years, presented with complaints of severe abdominal pain, vomiting and constipation for 3 days. There was no history of diabetes mellitus, hypertension, smoking, and previous abdominal pain. On admission to our hospital (on day 4 of illness), he was hemodynamically stable, had abdominal distention with diffuse tenderness and absent bowel sounds. Laboratory work up on day 4 revealed serum amylase: 898 U/l, lipase: 968 U/l, glucose: 116 mg/dl, Na^+: 132 mEq/l, K^+: 3.6 mEq/l, creatinine: 1.9 mg%, calcium: 8.6 mg/dl, bilirubin: 1.4 mg/dl, aspartate transaminase/alanine transaminase: 74/28 IU/l, alkaline phosphatase: 181 IU/l, serum proteins/albumin: 7.1/2.9 g/dl, serum cholesterol: 124 mg/dl, triglycerides: 130 mg/dl, Hb: 8.4 g/dl, leucocytes: 15,400/µl, platelets: 98,000/µl, prothrombin time: 18 s, activated partial thromboplastin time: 40 s. Computed tomography (CT) of abdomen [Figure 1] carried out with intravenous contrast on day 4 revealed enlarged, diffusely enhancing pancreas, multiple peripancreatic fluid collections and bilateral pleural effusions, confirming grade E AP (CT severity index = 4). He was kept nil per orally. Continuous nasogastric aspiration, intravenous fluids, stress ulcer prophylaxis and subcutaneous unfractionated heparin 5000 units twice/day for venous thromboprophylaxis were started. On day 5, he developed right sided hemiplegia. CT of brain revealed large left MCA territory infarct [Figures 2 and 3].

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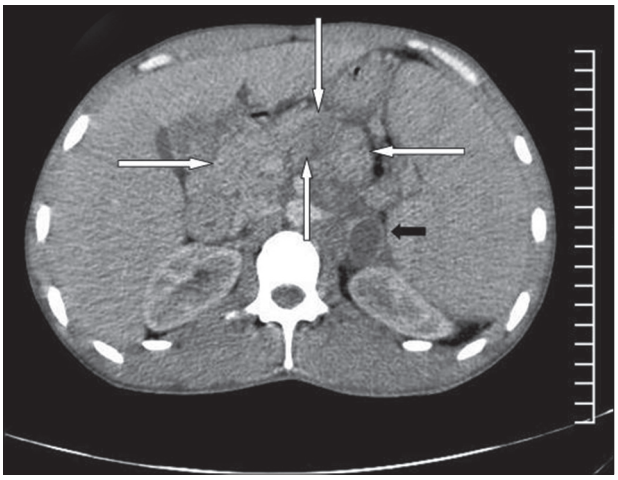

Figure 1: Computed tomography scan of the abdomen taken after intravenous contrast showing bulky, diffusely enhancing pancreas (indicated by white arrows) and a fluid collection (black arrow)

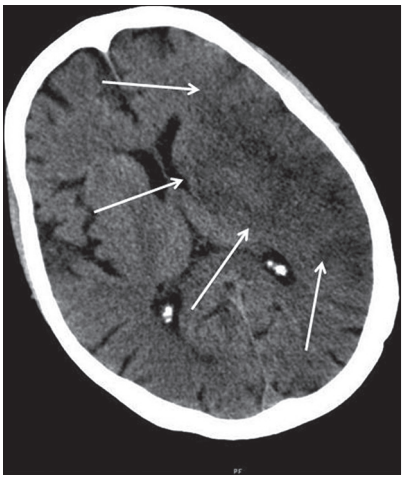

Figure 2: Plain computed tomography of the brain showing an area of hypodensity with poor gray-white differentiation in the left parieto-occipital regions, suggesting a large left middle cerebral artery territory infarct

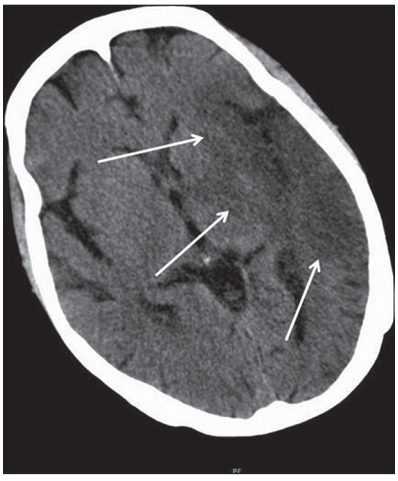

Figure 3: Plain computed tomography of the brain showing an area of hypodensity with poor gray-white differentiation in the left parieto-occipital regions, suggesting a large left middle cerebral artery territory infarct

Electrocardiogram (ECG) and echocardiography of the heart were normal. Work up for hyperhomocysteinemia, protein C and S deficiency, factor V Leiden, antithrombin III deficiency and antiphospholipid antibody syndrome was negative. Patient developed high grade fever and progressive acute renal failure from day 6. He was started on piperacillin + tazobactam and metronidazole. He was intubated on day 8 for progressive respiratory distress and hypoxia and was shifted to intensive care unit for mechanical ventilation. On day 9, he developed shock and was discharged against medical advice upon insistence of his relatives.

Discussion

Vascular thrombosis is a rare complication of AP. Splanchnic veins are most often the involved sites with a reported incidence of 1-2%, the consequences being portal hypertension, gastrointestinal bleeding, bowel ischemia and ascites.[1,2] Splenic vein is the most common site, followed by portal and superior mesenteric veins.[1,2] Thrombosis of inferior vena cava, renal veins and pulmonary thromboembolism have been reported rarely.[3,4] Arterial thromboses are less common than venous thromboses,[5] involve arteries in peripancreatic distribution – namely celiac trunk, perigastric, splenic and colonic arteries.[6] Microvascular thrombosis also plays a role in organ ischemia and infarction. Vascular complications are more commonly reported with alcohol related than with gall stone induced pancreatitis[6] and correlate with severity of AP.[2] The index patient had severe alcohol related AP as suggested by imaging [Figure 1] and the development of multi-organ dysfunction, but there was no evidence of SVT.

Neurological complications of AP include encephalopathy resulting from hypoxia or metabolic causes, uncommonly visual loss due to retinopathy or cerebral infarcts and polyneuropathy from nutritional deficiency (vitamin E) or prolonged critical illness.[7] Hemiplegia due to cerebral infarction resulting from vascular occlusion is rare,[7,8] as is spinal cord infarction leading to paraplegia.[9] Earlier reports of cerebral infarction in AP[7,8] have suggested fat embolism as the cause but left MCA thrombosis seems to be the most probable cause in our patient as he had a large infarct in the same territory [Figures 2 and 3]. Cardiac source for cerebral embolism is less likely in the index patient, considering that he had sinus rhythm throughout the course and ECG and echocardiography were normal. Other risk factors for ischemic stroke (hypertension, diabetes mellitus, obesity, smoking and advanced age) were absent in the index case and there was no

hypotension before development of cerebral infarction. Arterial thrombosis may lead to infarction of spleen, stomach and colon as well,[6,9] sometimes necessitating emergency surgeries. Acute limb ischemia of both lower limbs due to arterial thrombosis has also been reported in AP.[10]

The thrombotic tendency in AP may be related to systemic hypercoagulability resulting from inflammatory cytokines, endothelial dysfunction due to release of pancreatic enzymes into circulation, compression of vessels and direct vasculitis from surrounding inflammatory process.[5] The pathogenesis of fat embolism involves the release of chylomicrons and VLDL into the systemic circulation from omental fat necrosis in severe AP, along with elevated C-reactive protein which induce intravascular activation of the coagulation cascade and vascular occlusion.[7] Work up for other thrombophilic states was negative in our patient.

Dual phase contrast enhanced CT of abdomen, conventional or digital subtraction angiography, color Doppler ultrasonography and of late magnetic resonance angiography (MRA) are the modalities employed to diagnose vascular complications in AP.[2] CT cerebral angiography/MRA could not be carried out in the index case to confirm thrombosis because of worsening renal failure, progressive organ dysfunction and rapid clinical deterioration after development of cerebral infarction.

As there is increased risk of bleeding from pseudoaneurysms, frequent need for pancreatic debridement or drainage of infected collections, early anticoagulation (heparin followed by oral vitamin K antagonists to achieve an internationally normalized ratio of around 2.0)[2] for SVT in patients of AP can be problematic. Spontaneous recanalization was observed in about a third of patients of SVT irrespective of anticoagulation in a study.[2] Hence, the role of anticoagulation for SVT is unclear and the decision of anticoagulation has to be individualized, after considering the risk benefit ratio. Guidelines for management of arterial thrombosis are lacking, but heparin has been used in some cases.[10] Full therapeutic dose of heparin was avoided in the index patient considering the significant risk of hemorrhage into large MCA territory infarct.

To conclude, cerebral infarction can be a rare complication of AP and the treating physicians should be aware of vascular thrombotic and neurological complications of AP.

References

1. Mallick IH, Winslet MC. Vascular complications of pancreatitis. JOP 2004;5:328-37.
2. Gonzelez HJ, Sahay SJ, Samadi B, Davidson BR, Rahman SH. Splanchnic vein thrombosis in severe acute pancreatitis: A 2-year, single-institution experience. HPB (Oxford) 2011;13:860-4.
3. Ma SK, Kim SW, Kim NH, Choi KC. Renal vein and inferior vena cava thrombosis associated with acute pancreatitis. Nephron 2002;92:475-7.
4. Goenka MK, Kochhar R, Gulati M, Singh G, Khanna SK, Mehta SK. Acute pancreatitis complicated by pulmonary embolism secondary to inferior vena cava thrombosis. J Clin Gastroenterol 1994;19:85-6.
5. Mortelé KJ, Mergo PJ, Taylor HM, Wiesner W, Cantisani V, Ernst MD, *et al*. Peripancreatic vascular abnormalities complicating acute pancreatitis: Contrast-enhanced helical CT findings. Eur J Radiol 2004;52:67-72.
6. Challand C, Titcomb D, Armstrong CP. Pancreatic pseudocyst causing celiac artery trunk thrombosis. JOP 2008;9:512-4.
7. Bhalla A, Sachdev A, Lehl SS, Singh R, D'Cruz S. Cerebral fat embolism as a rare possible complication of traumatic pancreatitis. JOP 2003;4:155-7.
8. Flaggl E, Heer M, Hany A, Branda L. Loss of vision as a complication of acute pancreatitis. Schweiz Med Wochenschr 1988;118:722-5.
9. Wei SC, Kao JH, Lee WY, Lin JT, Wang TH. Acute pancreatitis complicated by infarction of the spleen and spinal cord. J Formos Med Assoc 1997;96:754-7.
10. Lee SY, Ng KH, Sebastian MG. Arterio-pancreatic syndrome. Case Rep Gastroenterol 2011;5:17-21.

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