

Acute Pancreatitis Complicated By Vascular Emboli

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ABSTRACT

This was a case of acute pancreatitis who complicated by left side hemiplegia and dysarthria. It diagnosed as Lacunar infarction and therapy with intravenous heparin started according to neurologist order. In third day of admission, the patient suddenly got dyspnea, tachycardia and tachypnea. Infusion of heparin continued with clinical diagnosis of pulmonary emboli and also warfarin added to his therapeutic regimen. The clinical condition of patient gradually got better and on 7th day of admission, he could tolerate oral regimen without any respiratory difficulty and his hemiplegia improved. On the following day, he discharged from ward with prescription of warfarin.

Keywords: Acute pancreatitis; Lacunar infarction; Pulmonary emboli

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INTRODUCTION

Acute pancreatitis in an inflammatory condition characterize by local damaging of pancreas and potentially could result in systemic inflammation(1,2). One of the causes of acute pancreatitis morbidity and mortality was vascular side effect(3-6). Arterial emboli was a rare and potentially mortal side effect of acute pancreatitis(7,8) and can occlude the major arteries of involved organs like lung or brain(9,10).

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These embolies usually result from deep venous thrombi but potentially can derive from any part of blood circulatory system(11,12). Vascular complication of acute pancreatitis could be related to arterial or pseudo aneurysmal bleeding and/or ischemic side effects(13,14).

CASE REPORT

The patient was a 77 Year old man suffering from epigastric pain since 5 days before admission. His pain aggravated by eating or lying down and partially subsided with sitting and bending forward. He has no significant past medical or drug history. There was no history of drinking alcohol or smoking. The only positive finding on clinical examination was epigastric tenderness. His vital signs included: Blood pressure 100/70, Pulse rate 84, Respiratory rate 16 and Temperature 37.2 °C.

His laboratory profile include: BUN =13 mg/dl, Cr = 1 mg/dl, PTT= 37 Sec, PT = 13 Sec, ESR= 72 mm/h, WBC= 7900 , Hb= 9.9gr/dl, MCV= 95 fL , AST= 233

U/L, ALT= 221 U/L, Alk-P= 236 U/L, Ca = 8 mg/dl, P = 2.9, Amylase = 89 U/L, Lipase = 39 U/L and TG = 105 mg/dl. An ultrasonography requested which reported the liver size and parenchymal echo normal and gall bladder wall thickness as 3mm with some sludge. For further clarification an abdominal CT scan requested. The abdominal CT scan findings were compatible with retroperitoneal fat accumulation around pancreas and heterogeneous pancreatic enlargement with irregular borders and suggested pancreatitis as paraclinical diagnosis (Figure 1).

The patient admitted by diagnosis of acute pancreatitis, transferred to ICU and supportive therapy including hydration with Ringer Lactate started. On the second day of admission, the patient suddenly complicated by left side hemiplegia and dysarthria. The neurologic consultation requested and based on clinical examination and brain CT scan and MRI finding, the neurologist diagnosed the case as Lacunar infarction and therapy with intravenous heparin started accordingly.

In third day of admission, the patient suddenly got dyspnea, tachycardia and tachypnea. On auscultation, there was minor wheezing on apex of right lung. A CT angiography requested which supported the clinical diagnosis of pulmonary emboli. Infusion of heparin continued and warfarin added to his therapeutic regimen. The clinical condition of patient gradually got better and on 7th day of admission, he could be able to tolerate oral regimen without any respiratory difficulty and his hemiplegia also improved. On the following day, he discharged from ward with recommendation of continuing warfarin consumption.

On follow up visit 2 weeks later, he was feeling good and the warfarin dose adjusted to keep the INR between 2 and 3 and. The patients followed up to 3 months later without any significant problem.

DISCUSSION

Thromboembolic events are one of the rare complications of acute pancreatitis which is not completely explained physiologically(7,8). It seems that thrombophilia and resultant vascular thrombosis be partly due to release of proteolytic enzymes from pancreas and its vascular complications(7,8). Some researchers have resumed various mechanisms including: 1. Presence of relational cysts between pancreatic ducts and vasculum. 2. Leakage of pancreatic secretions to vessels and predisposing them to thrombosis. 3. Thrombophilia due to liver dysfunction and raising of serum levels of trypsin that potentially can cause elevation of serum concentration



Fig. 1: CT scan of patient suggestive of acute pancreatitis.

of fibrinogen and factor VIII. 4. Vascular changes due to proteolytic damage(5,7,8,15-18).

The release of pancreatic enzymes damage the vascular endothelium and acinar cells which consequently leads to expression of endothelial adhesion molecules(19-21). Vascular injury could lead to local microcirculatory failure and release of free radicals and inflammatory cytokines into the circulation(22). These substances further interact with the pancreatic microcirculation to increase vascular permeability, which could induces thrombosis(19). Some patients with severe pancreatic damage develop systemic complications, including vascular thrombosis and embolization although a full blown hypercoagulable state is very uncommon(3-5,23).

Vascular thrombotic events can cause various signs based on the site of involvement(4,5,19). Pancreatitis in combination with vascular complications is dangerous and potentially lethal. The survival of patients with concomitant pancreatitis and vascular complications depends on the early diagnosis of these complications(5,23). Rapid diagnosis of these complications and commencing therapeutic interventions has a major role in management and preventing morbidity and mortality. Overall thrombotic events in the clinical course of acute pancreatitis are very rare but potentially mortal(3-5,7,11,23) that necessitate close clinical monitoring and follow up.

One of the rare and potentially lethal complication of acute pancreatitis is vascular thrombosis and emboli. Being cautious and keeping its possibility in mind is necessary for prompt diagnosis and proper intervention.

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