Bangladesh Crit Care J March 2024; 12 (1): 50-54

Case Report

Severe Acute Pancreatitis Complicated by Acute Pulmonary Embolism: a case report

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DOI: https://doi.org/10.3329/bccj.v12i1.72395

Abstract:

Acute pancreatitis is a painful condition in which the pancreas becomes inflamed and edematous usually for a short period of time. Pulmonary embolism due to severe acute pancreatitis is a rare but serious condition. A 68-year-old patient got admitted in gastroenterology department of a tertiary care hospital of Dhaka, Bangladesh with chief complaints of severe abdominal pain for 5 hours and several episodes of vomiting for same duration. After admission relative investigation and examination were done. His USG revealed swollen oedematous pancreas with peripancreatic fluid collection, bilateral renal parenchyma changes with cortical cyst. Patient's Glasgow Imrie score was 3, Numeric pain score (NRS) was 4, Urine output was normal. The patient was categorized into severe acute pancreatitis. The patient was treated with antimicrobials, proton pump inhibitors, analgesic, and fluid resuscitation. But patient's medical condition deteriorated and oxygen demand increased. Due to susceptibility of pulmonary embolism and worsening of patient's condition a thrombolysis was done.

Severe acute pancreatitis complicating with pulmonary embolism is a fatal condition.[1] Early diagnosis and treatment are fundamental for treating this dreadful condition. When Patient with acute pancreatitis exhibits dyspnoea, leg oedema and thromboembolic events, pulmonary embolism should be suspected. Following early diagnosis, thrombolysis is necessary to combat this situation.

Key words: *Pancreatitis, Pulmonary embolism, Thrombolysis, Mortality.*

Introduction:

Acute pancreatitis (AP) is a painful condition in which the pancreas become inflamed and edematous usually for a [1]. In AP Systemic inflammatory response occurs which is usually triggered by self-digestion of pancreatic tissue.[2] Due to this inflammatory response many vascular and pulmonary complications arise. Depending upon transient organ dysfunction, local fluid collection and necrosis, acute pancreatitis is categorized into - mild, moderate, and severe pancreatitis. Severe acute pancreatitis is a fatal condition and comparing to mild and moderate pancreatitis, severe acute pancreatitis has a very high mortality rate 10 %.[3] Pulmonary embolism (PE) is serious condition which generally occurs due to clotted blood in lungs. PE is a thromboembolic vascular phenomenon. It occurs when a blood clot (usually from the leg or pelvis) travels to the lungs and blocks the pulmonary arteries. Main symptoms are sudden shortness of breath, sharp chest pain (may worsen with breathing), rapid heart rate, and cough. However, symptoms can be variable. Pulmonary embolism due to severe acute pancreatitis is a rare but serious condition .[4-5] Blood hypercoagulability and systemic inflammation due to acute pancreatitis may play an important role in the prognosis of pulmonary embolism. Comparing to undiagnosed PE (20%-30%), the death rate of diagnosed pulmonary embolism is very low (2%- 8 %).[6] So, physician needs to be very aware and observant in case of management these types of patients. Here we are presenting a case of pulmonary embolism with severe acute pancreatitis and review the relevant literatures.

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Case Report

A 68-year-old male patient got admitted in gastroenterology department of a tertiary care hospital in Dhaka with chief complaints of severe abdominal pain for 5 hours and several episodes of vomiting for same duration. Following admission routine investigations were done from which relevant investigation reports are as followed: Haemoglobin: 12.7, Total WBC count: 17.70 k/ul, red cell count 4.44 M/ul, Platelet: 204 k/ul, blood amylase: 4123 (U/L), blood lipase:

Received : February 10, 2024; Accepted : February 24, 2024

51416 (U/L), lactate dehydrogenase (LDH): 847 U/L , Calcium: 8.9 mg/dl, Alkaline phosphatise: 24 U/L, Urine routine microscopy report showed pus cells: 5-7/HPF, CRP: 42 mg/dl, & Procalcitonin: 0.983 ng/ml. His USG revealed swollen oedematous pancreas with peri-pancreatic fluid collection, B/L renal parenchymal changes with cortical cyst. Due to ongoing acute kidney injury (AKI) and peripancreatic fluid accumulation, the patient was shifted to Critical Care Medicine unit in 2nd day of his admission.

The patient was a known case of DM, HTN, hypothyroidism and had permanent pacemaker. The patient did not have any history of taking alcohol or tuberculosis. Following admission into ICU patients BP was 110/60 mm of Hg, pulse rate was 70b/min, SpO_2 was 94% with 4 L/min oxygen, patient's Glasgow Imrie score was:3, NRS score was 4, Urine output was normal. On Echocardiography the patient had LV systolic dysfunction (EF:50%) and no emboli was present. The patient was categorized into severe acute pancreatitis. The patient was treated with antimicrobials, proton pump inhibitors, analgesic, and fluid resuscitation. From the beginning of the treatment, thromboprophylaxis was ensured (Enoxaparin). Following 3rd day of patient's admission, a CT scan of abdomen with contrast of Hepatobiliary system with pancreas was done (Figure 1) which revealed swollen partially necrotizing pancreatitis (Stage- 2) with mild to moderate fluid collection. No haemorrhage was found. During treatment period on 7th day of hospital admission, the patient's oxygen demand suddenly increased. Following the event some routine emergency investigations were sent in which the D-Dimer was high 15. Immediately a CT Pulmonary angiogram was planned. The CT pulmonary angiogram (CTPA) revealed significant acute thrombus involving the right upper and descending pulmonary artery including it's segmental and distal segmental branches (Fig 2,3,4,5). Irregular saddle thrombus was present extending to right main pulmonary artery near it's bifurcation. Usually, clotted blood which blocks the pulmonary artery comes from the thrombus of deep veins in the legs.[7] So, following diagnosis of PE, a Doppler USG of lower and upper limb was arranged. Doppler USG of lower limb revealed no evidence of deep vein thrombosis, but a partially thrombosed, pseudo-aneurysm was present in right common femoral vein (Doppler lower limb includes common femoral vein also). As the patient didn't have high oxygen demand at that time, enoxaparin was started in therapeutic dose. But patient started de-saturate on 8thday after admission. So, thrombolysis was arranged following which patient's condition got improved. Patient's oxygen demand decreased drastically, chest pain relieved, tachycardia and hypotension resolved. On 12th day of his admission a repeat CT Abdomen with contrast and CT pulmonary angiogram were arranged to see the prognosis of the patient's condition. CT abdomen report revealed acute severe necrotizing pancreatitis with peri-pancreatic inflammation with phlegomnic (phlegmonous fluid collection) fluid extending to gastroepiploic areas, splenic hilum, small bowel mesentery, greater omentum, bilateral anterior and peri-renal spaces. CTPA revealed there is slow early progressive resolution of pulmonary thrombus.

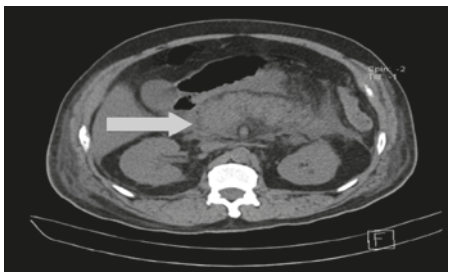

Figure-1: CT HBS with pancreas: swollen partially necrotizing pancreatitis (Stage- 2) with mild to moderate fluid collection. No haemorrhage was found.

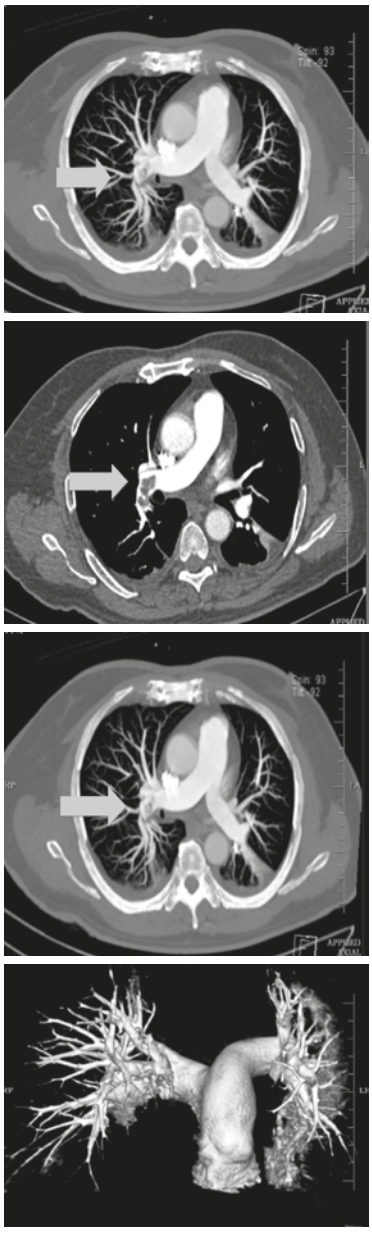

Figure 2, 3, 4, 5: Arrow in 2D CTPA images showing acute thrombus (filling defect) involving the right upper and descending pulmonary artery including it's segmental and distal segmental branches. Irregular saddle thrombus extending to right main pulmonary artery near its bifurcation.

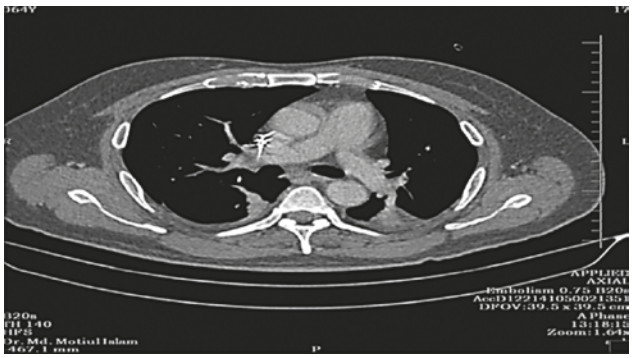

Fig 6: Post Thrombolysis CTPA

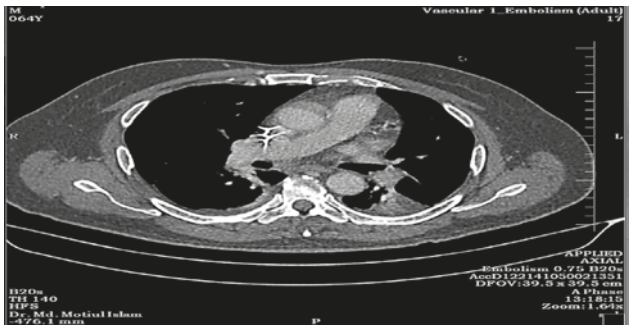

Figure 7: Arrow in 2D CTPA post thrombolysis showing resolution of pulmonary thrombus.

Discussion

In this report, we have described a case of acute pancreatitis with concomitant pulmonary embolism, and our experience suggests that pulmonary embolism may be an under-diagnosed complication of acute pancreatitis. Indeed, in our case, extensive pulmonary emboli were found incidentally after the diagnosis of severe acute pancreatitis was already made.

Acute pancreatitis is an inflammatory disease characterized by local tissue injury which can trigger a systemic inflammatory response. There is increasing evidence that endothelial dysfunction is one of the critical patho-physiological manifestations in patients with severe form of acute pancreatitis[8-11] and any factors that can cause venous blood flow stasis, vascular endothelial damage, and blood hypercoagulability can lead to venous thrombosis.[12] The clear mechanism of pulmonary embolism following severe acute pancreatitis is still unclear. But it is believed that by following mechanism this condition may occur.[13-15] If a pancreatic pseudocyst develops a fistulous tract or communication with the pancreatic duct, pancreatic juices could potentially leak directly into the cyst. This cyst may be originated from acute pancreatitis. In acute pancreatitis, pancreatic digestive enzymes leak out and damage the pancreas itself. This can lead to areas of tissue death (necrosis). Fluid released from damaged cells and inflammatory cells can accumulate and form a pseudocyst around the area of necrosis. If the cyst erodes into a blood vessel (pseudoaneurysm), the pancreatic juices could then enter the bloodstream. Once in the bloodstream, the activated pancreatic enzymes and acids have direct contact with the endothelial lining of blood vessels throughout the body. This pancreatic juice causes damage to the vascular endothelium. As a result, procoagulant substances got released and platelets get activated causing hypercoagulability state of the blood. [14,15]

Due to systematic inflammatory response during acute pancreatitis vascular endothelium got damaged. As a result, endothelial dependant acetylcholine relaxation reaction occurred in isolated mesenteric ring. Blood hypercoagulability might also play a major role in developing acute pancreatitis. Though one study stated that rather than blood hypercoagulability inflammation of venous endothelium, infiltration of cell and oedema are main fundamental cause of venous thrombosis in severe acute pancreatitis.[16] In addition to that the patient may have multiple risk factors to develop venous thrombosis.

Acute pancreatitis can rarely lead to pulmonary embolism due to increased clot formation (vascular thrombosis). Typical symptoms of pulmonary embolism in this setting include chest pain, shortness of breath, and sudden drops in oxygen saturation. Examination findings may reveal low oxygen levels, a bluish discoloration of the skin indicating poor circulation (peripheral cyanosis), rapid heart rate, or potentially death in severe cases. The diagnosis of pulmonary embolism should be suspected based on the patient's signs and symptoms. It can be confirmed via abnormal laboratory tests showing increased clotting tendency (e.g. prothrombin time, partial thromboplastin time, D-dimer). Imaging such as CT pulmonary angiography or CTPA directly visualizes clots in the pulmonary arteries. Echocardiography allows assessment of right heart strain from the embolism.

In summary, physicians should be aware that acute pancreatitis can, in rare cases, be complicated by pulmonary emboli. Characteristic clinical findings combined with supportive labs and imaging can confirm the diagnosis. Prompt diagnosis allows rapid treatment, as pulmonary emboli can seriously impact heart function and potentially lead to death in severe cases.

This patient with acute pancreatitis complicated by pulmonary embolism did not have any underlying blood disorders or hematologic diseases that could predispose to thrombosis. There was no history of recent surgery, trauma, or immobility that could have led to venous stasis and thrombosis development.Importantly, the patient had no prior thrombotic conditions or risk factors that would suggest a hypercoagulable state or increased clotting tendency. This includes a negative history for key precipitating factors for deep vein thrombosis like obesity, smoking, malignancy, oral contraceptive use, inherited thrombophilia, or recent lengthy travel.

In summary, the patient's lack of hematologic comorbidities, thrombotic medical history, recent triggers for immobility, or known risk factors for hypercoagulation, highlights acute pancreatitis as the likely inciting event for this pulmonary embolism. The severe inflammation and systemic response from the acute pancreatitis are the most probable precursor leading to vascular thrombosis and embolic complications in

the absence of other identifiable risks.

According to a recent literature these are the common sign and symptoms in acute pancreatitis complicating with pulmonary embolism and above-mentioned test should be made as soon as possible for confirmatory diagnosis .[17,18] Early diagnosis and management plan is very important to combat this dreadful situation. It can reduce mortality rate significantly. Early detection, oxygen support, pain management and thrombolysis (intravenous heparin, rTPA) are a must to do to.

Conclusion

In conclusions, severe acute pancreatitis complicating with pulmonary embolism is a fatal condition. Early diagnosis and treatment are fundamental for treating this dreadful condition. When Patient with acute pancreatitis exhibits dyspnoea, leg oedema and thromboembolic events pulmonary embolism should be suspected. Following early diagnosis thrombolysis is necessary to combat this situation. By doing this death rate can be reduced significantly.

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