



A CASE OF ACUTE CORONARY SYNDROME PRESENTING AS ACUTE ABDOMEN

NARENDRAKUMAR V

Department of Cardiology,
MADRAS MEDICAL COLLEGE AND GOVERNMENT GENERAL HOSPITAL

Abstract :

Cardiovascular disease is one of the leading causes of mortality in both developed and developing nations. One of the most dreaded complications of myocardial infarction is thrombo embolic manifestation. Here we are reporting an unusual presentation of acute anterior wall myocardial infarction presenting as mesenteric ischemia and acute pancreatitis due to embolic manifestation.

Keyword : Acute Coronary Syndrome
Acute Abdomen

45 year old male was referred from surgical gastroenterology, a case of acute abdomen for routine cardiac evaluation. He was admitted with complaints of breathlessness and abdominal pain for two days. He denied h/o chest pain. Known smoker and alcoholic for 20 years. He was not a known case of Coronary Artery disease. Not a known DM/ HT/CVA pt. No family h/o premature CAD / sudden cardiac death. General examination was normal. Vitals were stable; Cardiovascular examination revealed normal

heart sounds, no added sounds and murmur. ECG showed an evolved AWM. His Investigation revealed WBC count was 18,600 with polymorphs 80% lymphocytes 10% eosinophil 10%, Urea- 48 mg%, Creatinine- 1.9 mg% , LFT – WNL, Sr. Amylase- 97 iu/ L, CPK (T) – 1670 iu, HIV-NR, HBsAg- Negative.

Echocardiogram showed RWMA of left ventricle – LAD territory with large LV apical clot measuring 6.8 x 2.3 cm extending from LV apex to LVOT, Moderate LV systolic dysfunction EF- 36%, Valves normal and normal RV function. USG Abdomen showed features s/o pancreatitis. Contrast CT Abdomen showed celiac, splenic, renal artery thrombus with mesenteric ischemia, acute pancreatitis / pseudo cyst, splenic and renal infarct. OGD – Ulcerated globular mass in stomach- pancreatic pseudocyst abetting stomach. Pancreatic pseudocyst was managed conservatively using broad spectrum antibiotics. Patient was treated conservatively with Heparin, DAPT, Acenocoumarin, Diuretics, ACE inhibitors and Beta blockers.

Patient was on follow up in our department. His follow up echo revealed decrease in size of LV apical clot and mild LV systolic function. Ultrasound abdomen revealed reduction in size of pancreatic pseudocyst.

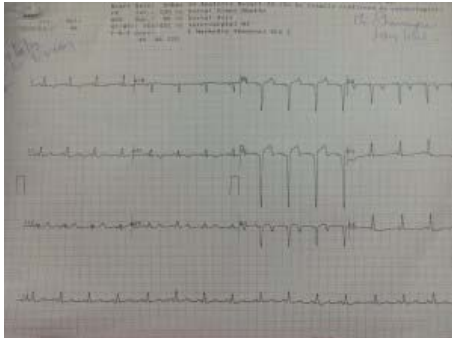


Fig 1: ECG



Fig 2: Echo - PLAX View



Fig 3: Echo A5C view

Fig 4: Echo - M Mode

Fig 5: CT - LV clot

Fig 6: CT Abdomen



DISCUSSION:

The incidence of clinically evident systemic embolism after MI is lower than 2%. The incidence increases in patients with anterior wall MI. The overall incidence of mural thrombus after MI is approximately 20%. Large anterior MI may be associated with mural thrombus in as many as 60% of patients. Most emboli arise from the left ventricle as a result of wall motion abnormalities or aneurysms. Atrial fibrillation during ischemia also contribute to systemic embolisation.

The combination of blood stasis, endothelial injury and hypercoagulability, often referred as Virchow's triad, is a prerequisite for in vivo thrombus formation. LV regional wall akinesia and dyskinesia result in blood stasis, often recognised on two dimensional echocardiography by the occurrence of spontaneous LV contrast. Prolonged ischemia leads to subendocardial tissue injury with inflammatory changes. Patients with an acute coronary syndrome display a hypercoagulable state- increased concentrations of prothrombin, fibrinopeptide A, and von Willebrand factor, and decreased concentrations of the enzyme responsible for cleaving von Willebrand factor. This triad can result in the formation of LV thrombus composed of fibrin, red blood cells, and platelets. The most common clinical manifestation of embolic complications is stroke, rare presentation of limb ischemia, renal infarction or mesenteric ischemia. Mesenteric ischemia manifests with postprandial abdominal pain out of proportion to clinical findings. Risk factors for the development of LV thrombus are consistently irrespective of infarct treatment and include large infarct size, severe apical asynergy (ie, akinesis or dyskinesis), LV aneurysm, and anterior MI. The risks of systemic embolisation in patients with LV thrombus are: (1) severe congestive heart failure, (2) diffuse LV dilatation and systolic dysfunction, (3) previous embolisation, (4) advanced age, and (5) presence of protruding or mobile thrombi in LV. Two dimensional echocardiography provides excellent specificity (85–90%) and sensitivity (95%) in detecting LV thrombus. LV thrombus on echocardiography is defined as a discrete echodense mass in the left ventricle with defined margins that are distinct from the endocardium and seen throughout systole and diastole. It should be located adjacent to an area of the LV wall which is hypokinetic or akinetic and seen from at least two views (usually apical and short axis). Our patient had large LV apical clot with thrombus in celiac axis presenting

as mesenteric ischemia, acute pancreatitis and the patient was managed conservatively.

Conclusion:

We have presented this case of an evolved AWMi presenting as acute abdomen due to emboli phenomenon for its rarity. The diagnosis should be considered in patients with acute coronary syndrome presenting as acute abdomen and timely intervention is life saving for the patient.

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