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Abdominal Pain as a Consequence of Dilated Cardiomyopathy - A Rare Case Report

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Abstract: ***A 35 year old male presented with acute pain in abdomen since 5 days. Pain was situated in left hypochondriac region with tenderness and rigidity present on per abdominal examination. Patient is a chronic alcoholic investigating further, On CECT A+ P, there were multiple Splenic infarcts with lung window showing incidental finding of LV thrombus. On transthoracic 2 Decho it revealed that patient is having dilated chambers with hypokinesia with a large mobile apical LV thrombus. Patient was given low molecular weight heparin bridging with warfarin. This case highlights a rare presentation of effect of alcohol on heart causing toxin induced dilated cardiomyopathy leading to formation of LV thrombus with consequence of splenic infarct due to systemic thromboemboli.***

Keywords: Spleenic infarct, lv thrombus, dilatedncardiomyopathy

1. Introduction

Alcohol induced cardiomyopathy is one of the most common type of toxin induced dilated cardiomyopathy seen in young males in India. It is also one of the most common cause of non ischemic type of dilated cardiomyopathy.They generally present with symptoms like chest discomfort, breathlessness on exertion, fatigue, palpitations and other symptoms of heart failure.[1] Due to decreased ejection fraction there is stasis of blood leading to thrombus formation. Thrombus formation is typically attributed to virchow's triad (reduced ventricular motion, local myocardial injury and hypercoagulability/stasis of flow). [2] This thrombus can lead to systemic emboli causing stroke, renal infarct, splenic infarct, etc.. Though this type of presentation is very rare, it is important for treating physicians to keep an open eye.

2. Case Presentation

A 35 year old male presented with chief complaint of pain in abdomen since 5 days. Pain was sudden in onset, present in the left hypochondriac region. It gradually progressed from pain while stretching of given area to pain at rest in 3 days. Pain was non radiating and was associated with tenderness and rigidity. There was no history of trauma. On digging the history further, patient also complaint of breathlessness, palpitation, fatigue and chest pain since 2 months which was gradual in onset and progressed from NYHA grade 2 to NYHA grade 3 in 2 months i.e.; patient getting the above mentioned symptoms on playing cricket or doing exertional work three months ago which progressed to having symptoms even while bathing or walking to bathroom (<100 mtr). Patient never approached any hospital or doctor for these symptoms because symptoms used to go away after rest. He also complained of occasional swelling of bilateral lower limb, pitting type,more in the evening which used to go away after limb raising or taking rest. Patient is a chronic alcoholic taking 360-720 ml of alcohol every day for the last 20 years.

On examination, he was conscious oriented to time, place and person with decubitus of left lateral position in flexion with right hand positioned over left hypochondriac region. Pulse was 120 bpm regular and blood pressure was 110/70 taken in the right arm in supine position. No pedal edema or cyanosis or icterus or pallor present. On per abdominal examination, there was tenderness guarding and rigidity on palpation in the left hypochondriac region. Respiratory cardiovascular and neurological examinations were within normal limits.

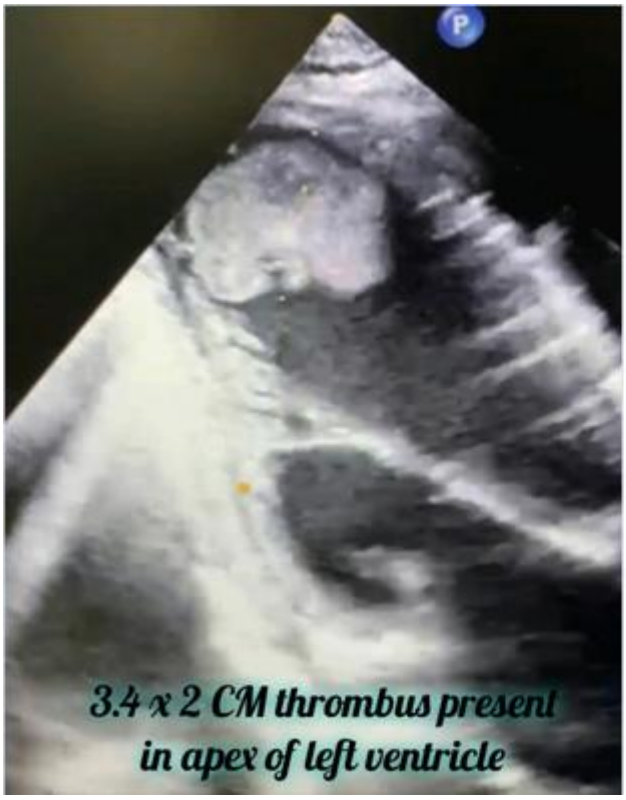
Investigation

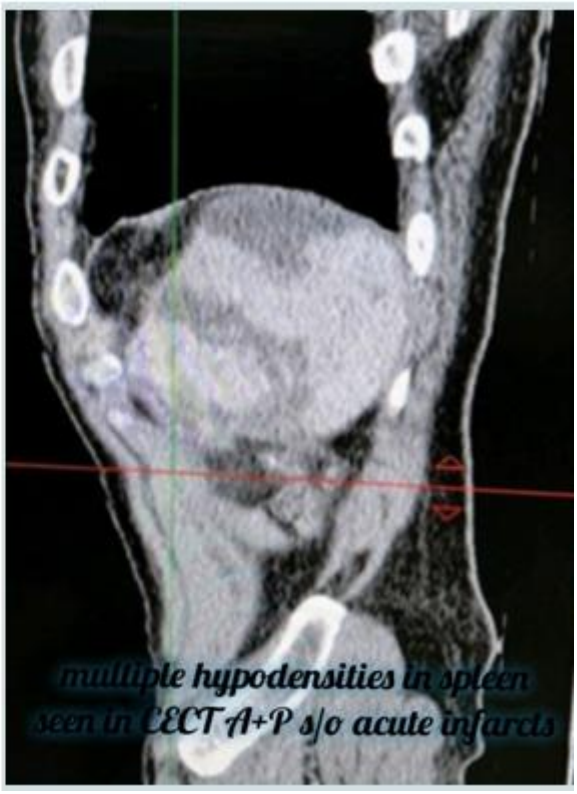
Patient was admitted and taken for urgent USG ABDOMEN +PELVIS which was suggestive of mild hepatomegaly with hypoechoic and mildly bulky head of pancreas.

Onn contrast enhanced CT of abdomen + pelvis, there were multiple patchy ill defined areas of non enhancing hypodensities involving spleen parenchyma suggestive of splenic infarcts however no obvious thrombus seen in splenic artery.

Surprisingly in the lung window, there was non enhancing lesion of approx size 28x27 mm in the left ventricle near the apex suggestive of? THROMBUS.

Due to the incidental finding and history suggestive of heart failure and personal history of chronic alcoholism, patient's 2Decho was done which showed a large left ventricular thrombus of size 3.4 X 2 CM in apex, global hypokinesia with LVEF pertaining to 10-15 %and dilated cardiomyopathy (all 4 chambers dilated).





3. Treatment

Given the LV thrombus, patient was started on low molecular weight heparin 5000 IU s/c twice a day for 5 days bridging with warfarin 5 mg OD from day 3. [3][4] Patient's INR to be kept in range of 2-3 while adjusting the dose of warfarin [5]. Warfarin is a vitamin K antagonist used as a prophylactic drug for thromboembolic events. For splenic infarct, the patient was given intravenous pain relieving medication. Due to the presence of heart failure with reduced ejection fraction, patient was started on diuretics to decrease preload, ACE inhibitor to decrease afterload, beta blocker to prevent remodeling and block sympathetic overactivity and digoxin to decrease cardiac overwork. [6] Patient was advised bed rest and follow up 2d echo after every 3 months till clot is resolved.

4. Discussion

Presentation of splenic infarct due to LV thrombus is very rare. Most commonly, splenic infarcts are due to myeloproliferative disorders or sickle cell disease. [7]

Alcohol is a huge burden on society in india. Alcohol affects multiple organs in the body like heart, liver, kidneys, blood and brain. There are multiple mechanisms by which alcohol causes dilated cardiomyopathy directly as well as indirectly. [8]Alcohol induced dilated cardiomyopathy is common in chronic alcoholics leading to heart failure with reduced ejection fraction. Chronic alcoholics tend to ignore symptoms leading to delay in proper investigation and treatment which in turn worsens the outcome and prognosis. Due to severe hypokinesia and dilation of heart, there is stasis of blood which in combination with local myocardial injury due to toxin effect results in formation of thrombus in heart. As a consequence of this thrombus, there is release of emboli in systemic circulation causing splenic infarcts.

Trans thoracic 2d Echo is standard investigation for diagnosing LV thrombus while contrast enhanced cardiac MRI remains gold standard. [9]

Anticoagulation with vitamin K antagonists remains mainstay prophylaxis and treatment of LV thrombus with duration of 3-6 months adjusted with follow up 2-d echos every 3 months. The prothrombotic effect of VKA warrants their co administration with parenteral anticoagulants initially.

5. Conclusion

Young patients presenting with acute left hypochondriac pain should be evaluated for splenic infarct as a potential cause. History should be dug in chronic alcoholics for the presence of heart failure symptoms and the cardiovascular system should be evaluated aggressively. Thus, it is appropriate to conclude in our patient that the cause of splenic infarcts is emboli from LV thrombus which is present as a consequence of toxin induced dilated cardiomyopathy causing stasis of blood in the left ventricle due to hypokinesia.

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