



Case Report

ISSN 2320-4818

JSIR 2018; 7(4): 85-87

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Received: 05-12-2018

Accepted: 30-12-2018

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A case of old Anterior wall MI Presenting with left Ventricular clot and systemic embolization in a young man

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Abstract

Patients having myocardial infarction with severe Left ventricular dysfunction should be followed up regularly as clot formation can be there in akinetic scarred areas. Other non-conventional risk factors should also be assessed. Our patient also had high homocysteine levels making such patients more prone for increased vascular risks, clot formation and embolization. Early anticoagulation, surgical intervention should be warranted in such patients so they can be saved from embolic complications especially in young age group.

Abbreviations: MI- myocardial infarction. NOAC- Non-vitamin K antagonist oral anticoagulants, TTE- trans thoracic echocardiography, TEE- trans esophageal echocardiography, LV- left ventricle, PVD- peripheral vascular disease

Keywords: Myocardial infarction, Peripheral vascular disease, Left ventricular dysfunction.

INTRODUCTION

Many patients previously having myocardial infarction on follow up present as cardio-embolic stroke or incidentally diagnosed to have Left ventricular clot on Echocardiography with high risk of embolization within akinetic areas mostly Antero-apical infarcts. Treating such patients Conservatively may be a challenge and What should be the best strategy for Choice of antiplatelets, anticoagulants on follow up always comes in mind. We are presenting a case of young man with stroke in whom timely management saved him from unwanted sequelae of stroke and acute peripheral vascular complications.

CASE REPORT

A 36 years old young man having previous history of anterior wall MI presented with weakness of left upper limb, slurring of speech, pain in both legs for one day. On admission Echocardiography was done which revealed a mobile pedunculated mass attached to akinetic apical septum [figure1 /2]. To confirm Trans esophageal echocardiography [figure3] followed by cardiac MRI was done. Cardiac MRI revealed ischemic involvement of left ventricular myocardium in LAD territory & ischemic scar with relative non-viability and intra cavitory thrombus. MRI brain was done revealed high right parietal infarct and lower limb Doppler revealed Peripheral vascular disease suggestive of cardio-embolic episodes. Previously twelve year back he had anterior wall myocardial infarction, then Coronary angiography was done which revealed recanalized LAD. His Stress ECHO done later revealed negative study and was on conservative medical management. Due to his age and having major cardiovascular events, other risk factors were also investigated and he was found to have high homocysteine level >50umol/L [CMIA]. Patient wanted himself to be managed conservatively, So IV heparin infusion was given in ICCU. He recovered having no focal neurological deficit and was followed with NOAC, aspirin, folic acid and other supportive drugs. Review ECHO was done after six months which revealed no remains of left ventricular clot. At present he is doing well with complete recovery [figure 4].

DISCUSSION

Left ventricular clot formation is a serious complication in patient with old anterior wall MI. The combination of blood stasis, endothelial dysfunction, hypercoagulability is responsible for the formation of thrombus (1). The Antero apical infarcts have large area of poorly contracting Left Ventricular muscle and intra cavitory blood movement is sluggish compared to normal areas. Many, but not all of these patients will have a Left ventricular apical aneurysm with akinesia or dyskinesia. Our patient has large

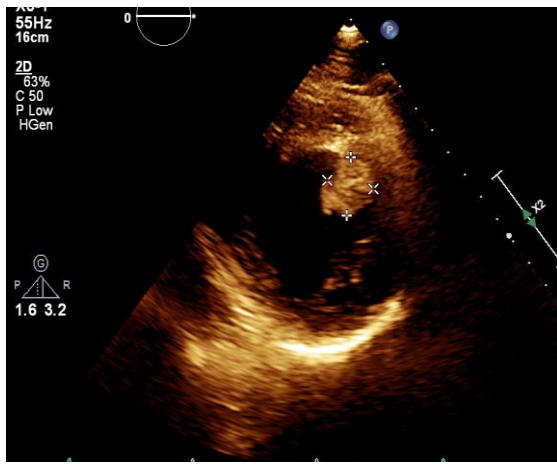


Figure 1: The PLAX view and short axis view showing thrombus attached to akinetic Apical/Septal wall

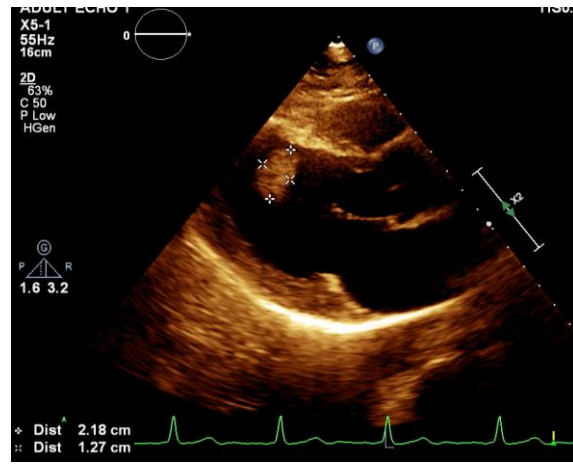


Figure 2: The PLAX view and short axis view showing thrombus attached to akinetic Apical/Septal wall

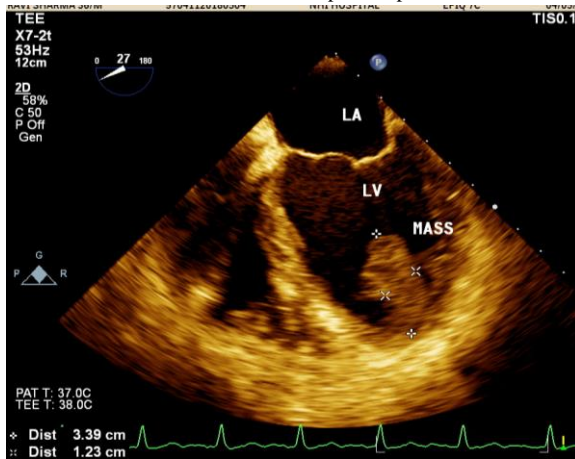


Figure 3: TEE image showing the mass attached



Figure 4: Plax view showing complete absence of left ventricular clot after six months of Apixaban treatment

mobile thrombus and high potential risk for more systemic embolization (2). Our ECHO finding was further confirmed by TEE followed by cardiac MRI which revealed mobile LV clot. ECHO is a very useful modality in such cases (3), but in our case we also had done cardiac MRI to confirm thrombus [4] and rule out other causes of Left ventricular mass and to start proper management as he is having ongoing thromboembolic phenomenon. Our patient also had homocysteine level >50 making him high risk for cardiovascular events. Hyperhomocysteinemia has been independently associated with LV clot formation. Current evidence from retrospective and prospective studies support the concept that higher total plasma homocysteine concentration is associated with increased risk of coronary artery disease, stroke, and venous thromboembolism [5]. Although major studies such as vitamin intervention for stroke prevention trial [VISIP], NORVIT, HOPE 2 trial [6/7/8] have been done but none has shown substantial benefit of homocysteine reduction. Despite lack of evidence that homocysteine reduction lowers risk, there remains specific patient population for whom homocysteine evaluation may prove appropriate including those lacking traditional risk factors with renal failure or with marked premature atherosclerosis, family history of myocardial-infarction and stroke at younger age group. The early and active recognition of Left ventricle thrombus is vital to unwanted sequelae of systemic thrombo-embolic events. In observational study and meta-analyses anticoagulant therapy is recommended in order to lower embolization risk. NOACS are not licensed for the treatment of Left ventricular thrombus however there are growing number of reported cases where use in Left ventricular thrombus has shown complete recovery [9/10/11]. In our case we have started NOAC [apixaban], aspirin and folic acid with regular follow up. After six months there is complete dissolution of left ventricular thrombus with no peripheral symptoms.

CONCLUSION

Thrombus formation is common after myocardial infarction in akinetic aneurysmal areas and severe LV dysfunction. Routine follow up in such patient with regular ECHO is necessary to diagnose LV clot early and avoid thromboembolic episodes. Further if needed, cardiac MRI can be done to rule out other form of LV mass. Evaluation of Non-conventional risk factors may play additive role in management and should be evaluated in such patients. NOACS can be considered with good results with complete resolution although more larger studies are needed.

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