Extreme Clinical Presentations of Venous Stasis: Coronary Sinus Thrombosis


Abstract

Sixty six year old male with history of heart failure was admitted for dysphagia, weight loss. CT scan chest revealed diffuse oesophageal wall thickening. Upper endoscopy, oesophagogram confirmed diagnosis of achalasia. TTE revealed severely reduced biventricular systolic function with LVEF 10%; PASP 75-80 mmHg. Parasternal long views showed dilated coronary sinus with a visible, mobile 2.0 cm thrombus. Pro-thrombotic workup was negative.

Coronary sinus thrombosis has been identified as a rare complication to invasive cardiac procedures causing damage to coronary sinus endothelium and in hypercoagulable states. Typically acute thrombosis presents with chest pain, dynamic ECG changes, but chronic development does not present with ischaemic signs due to formation of efficient collateral circulation. We present a case report of stable primary coronary sinus thrombus incidentally diagnosed, secondary to chronic venous stasis in coronary circulation. Currently, there are no guidelines to assist physicians in long term management of such patients and thus warrants further investigations.

Introduction

Coronary sinus thrombosis is a rare clinical entity with a very high fatality rate. Invasive procedures of right heart including central venous line placements, pacemaker wire insertion, coronary sinus catheterisation for ventricular lead placement in resynchronisation therapy, heart surgeries can lead to damage of coronary sinus endothelium and subsequent thrombosis. Coronary sinus thrombosis has also been observed in patients with hyper-coagulable states. Primary thrombosis of coronary sinus without any preceding cardiac interventions and in absence of hyper-coagulable state very rarely reported in medical literature. We report the occurrence of primary coronary thrombosis which was noted incidentally on an echocardiogram.

Case Report

Sixty six year old man with history of hypertension, stroke, and chronic decompensated systolic heart failure since eight years prior to admission was admitted with complaints of epigastric pain, dysphagia, dyspnoea Grade C, decreased exercise tolerance and weight loss. Cardiac catheterisation done eight years prior to admission did not show evidence of coronary artery disease. There is no history of any invasive cardiac procedures or history of central venous line placement.

Vitals recorded were Blood Pressure 116/86; Pulse 68/min, Breathing 18/min, maintaining 97% saturation on two litres oxygen by nasal canula. Examination revealed a well built individual lying comfortable in bed. Chest examination revealed bibasilar crackles without any other abnormalities. Cardiac examination revealed tachycardia with irregular rate, systolic murmur grade III loudest in the mitral area and all

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peripheral pulses were well felt. There was pitting pedal oedema bilaterally extending till knee. Rest of the examination was unremarkable.

Electrocardiogram showed sinus rhythm with frequent premature atrial and ventricular contractions, incomplete left bundle branch block and left axis deviation. Trans-thoracic echocardiogram revealed severely reduced left and right ventricular systolic function with ejection fraction of 10%; Pulmonary artery systolic pressure 75-80 mmHg, moderate tricuspid regurgitation with moderate to severe mitral regurgitation and moderate aortic regurgitation. Parasternal long views revealed a markedly dilated coronary sinus with a visible, mobile two cm thrombus. Computed tomography of neck done for evaluation of dysphagia indicated thickening of the oesophageal wall suggesting an infiltrative process. Upper endoscopy and barium studies followed which confirmed the diagnosis of achalasia and ruled out any infiltrative process. Coagulation profile was normal and all prothrombotic factors were negative.

During the admission patient was treated for acute on chronic decompensated systolic heart failure, coronary sinus thrombosis and achalasia. Patient responded to furosemide and improved symptomatically. Patient was recommended Coumadin with Enoxaparin bridging for his coronary sinus thrombosis but he declined Coumadin, hence was anti-coagulated with Enoxaparin alone during inhospital stay. Patient was recommended defibrillator placement but he refused the same. Patient was continued and discharged on Aspirin, clopidogrel, lisinopril, carvedilol and furosemide with follow up in GI and cardiology clinic. At discharge patient was symptomatically stable with Grade C dyspnoea and able to tolerate medium consistency oral feeds.

**Discussion**

The most common mechanism to initiate thrombus formation in coronary sinus is endothelial damage, usually occurring as a secondary complication of procedures such as central venous line placement,\(^2\) pacemaker wire placement, cardiac surgery,\(^4\) heart transplantation, coronary artery bypass grafting,\(^6\) radiofrequency ablation around AV node.\(^1\) Apart from endothelial damage, hyper-coagulability (secondary to malignancy) and venous stasis also play a role in development of coronary sinus thrombosis although very few incidents have been reported.\(^6\) We present a case of Coronary sinus thrombosis diagnosed incidentally on an echocardiogram (Figure 1) in a patient with no history indicative of damage to coronary sinus endothelium or hyper-coagulability.

Venous stasis is long known to cause thrombosis in deep venous system of legs; however this factor has never been reported as a sole aetiological factor in formation of coronary sinus thrombus. The venous system of heart consists of three separate systems. The large bore coronary sinus and the anterior cardiac veins both empty in the Right atrium and drain 60% and 40% respectively of the total cardiac venous return. There are many anastomotic connections between tributaries of these two venous systems. Finally, the heart is also drained by minor Thebesian veins into all four chambers of heart. Wearn had described that thebesian veins in full capacity can drain up to 50% of cardiac venous return.\(^10\) This explains how coronary sinus thrombosis can present as a benign entity due to efficient collateral circulation.

Typically acute thrombosis of coronary sinus usually results in mortality and presents with chest pain, dynamic electrocardiogram changes and signs of acute cardiac decompensation.\(^1,5\) This probably results secondary to acute onset venous infarction of myocardium. However chronic development of thrombus does not present with ischaemic signs due to formation of efficient collateral circulation between coronary sinus, anterior cardiac and Thebesian veins. Thus chronic development may go unnoticed until it develops complications such as acute plaque rupture causing myocardial infarction,\(^5,6\) myocardial dysfunction without infarction potentially reversible with thrombectomy,\(^7\) cardiac tamponade.\(^2\) These cases are usually fatal\(^2,5,8,9\) and diagnosis is often made at autopsy; thus limiting the evidence reported in such instances.

Congestive heart failure is the leading cause of mortality among geriatric population and it is very important to focus on its potentially fatal complications. It is important for physicians to be alert and look for coronary sinus thrombosis in long standing surviving cases of heart failure who have chronic venous stasis in coronary circulation, as missing diagnosis can lead to sudden death. Currently, there are no long term guidelines to assist physicians in diagnosis and long term management of such patients. The role and efficacy of anticoagulation is unclear and thus warrants further investigations.

**Conclusion**

Considering the fact that coronary sinus thrombosis is a potential, though rare complication of invasive cardiac procedures; it is important to look for such an event in cases of chronic coronary venous stasis also. The evidence for the role of long term anticoagulation and diagnostic standards is unclear and further guidelines are warranted.
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