Extended Abstract Cardiovascular Investigation: Open Access 2020
Vol.4 No.4

Annual Cardiology 2017: Coronary occlusion & pulmonary embolism in the absence of thrombophiliat - S Wasif Hussain - Geisinger Medical Center, USA

S Wasif Hussain

Geisinger Medical Center, USA

We are presenting a case of 56-year-old man with no past medical history, presented with non-exertional chest pain, pressure like, constant, with no radiation, associated with lightheadedness & an episode of near syncope. He was not able to eat for 2 days prior to presentation. He has family history of hypertension in father but no history of thrombophilia. There is no history of smoking/alcohol use. On examination, he has bradycardia with balanced blood pressure. His cardiac examination showed bradycardia with regular rhythm. Lung examination showed clear lungs. Work up included EKG & labs. His EKG showed junctional rhythm. He was admitted to CCU. Telemetry checking showed heart block with junctional escape rhythm. There were ST changes in the right & inferior leads with mild elevation of troponins. After which, he was given aspirin 162 mg, clopidogrel 600 mg, heparin 5400 units IVP & started on heparin infusion. He was shifted to another facility for possible percutaneous coronary intervention. His troponins went up from 0.01 to 0.095 to 0.094 while his BNP was 182. Complete section of admission labs showed Na 141, potassium 4.2, Creatinine 1.05, Glucose 117. WBC 6.47, Hb 12.5, Hematocrit 37.9, Platelets 140. On subsequent day, CK was 1120, CKMB 151.2, troponin I 6.78 & later 11.38 & then 43.12. Lipid panel showed HDL 57, LDL 71, Triglycerides 47, Cholesterol 137. After transfer, he developed hypotension & started off on dopamine infusion. He had emergent cardiac catheterization after being transferred to the facility after 12 hours 24 minutes. His BP was 73/46 mmHg. He was given fluid bolus of 500 ml & BP improved to 95/60 mmHg. Cardiac catheterization showed two vessel disease with tubular stenosis 60% in mid LAD, CFX with discrete 10% stenosis, there was 100% stenosis of mid RCA. The lesion was associated with a large filling defect consistent with thrombus. There was TIMI grade 0 flow all through the vessel. The LVEDP was 14 mmHg. He was given heparin during the cardiac catheterization. PCI was unsuccessful due to large clot burden. Single bolus integrillin was given. He was moved to critical care unit. The dopamine infusion was discontinued on second day after cardiac catheterization. He was continued on IV fluids until the next day. Due to the RCA thrombus, eliquis was started. Initially, it was determined to be given for 3 months. Transthoracic Echo showed septal & posterior hypokinesis. Ejection fraction was 40%. There was possible McConnel's sign with right ventricular dilatation. The atrial septum bows from right to left consistent with increased right atrial pressure. He had CTA chest that revealed bilateral pulmonary emboli, RLL pulmonary infarct & flattening of IV septum. Hematology was consulted who recommended for life long anticoagulation. His cardiac rhythm spontaneously converted to sinus bradycardia. He was started on enalapril with clopidogrel. Beta blockers were avoided due to the bradycardia. He was subsequently discharged on enoxaparin & warfarin with lifelong anticoagulation. Hypercoagulable work up was sent including Antithrombin III antigen 89%, Factor V leidin is negative , PTT-LA 36.1 seconds & DRVVT 74.6 seconds, Lupus anticoagulant is not detected, protein C antigen 76 %, protein C functional 108 %, Cardiolipin Ab IgA <9 Units/ml, Cardiolipin Ab (IgG) <9 Units/ml, Phos. Serine Ab IgG 9, Phos Serine Ab IgM 5, Phos. Serine Ab IgA 1, B2 Glycoprotein I Ab (IgA) <9, Protein S functional 134 %, Protein S free 69 %, protein S total Ag 89 %. Coronary thrombus with possible embolic occlusion in the presence of Pulmonary embolism, is a unique case. There have been various case reports of acute myocardial infarction with coronary occlusion & pulmonary embolism. All those cases are secondary to paradoxical embolism due to patent foramen ovale. There is also a report of ischemic stroke associated with paradoxical embolism in a patient with acute myocardial infarction. In the absence of obvious hypercoagulable status, coronary thrombosis with pulmonary embolism has never been reported. In situ thrombosis at two separate locations can be a possibility. Embolism into the coronary vessel can also be due to other factors apart from hypercoagulable condition. Also, if there is coincidence of myocardial infarction & pulmonary embolism then the diagnosis can be particularly difficult due to the similarity in clinical signs & symptoms of both entities. There is another case reported in patient with antiphospholipid syndrome. This is secondary to hypercoagulable condition leading to thrombosis & embolism.

Deficiencies of protein S, protein C, & antithrombin III are the leading causes of thrombophilic defects. Patients with hereditary thrombophilia are at increased risk of acute PE, particularly among young individuals. Therefore, early detection of thrombophilic defects together with other unprovoked risk factors could reduce the risk of recurrent VTE.

Note: This work is partly presented at 19th Annual Cardiology Conference on August 31-September 01, 2017 held at Philadelphia, USA