

## Early Graft Dysfunction Post Coronary Artery Bypass due to Heparin Induced Thrombocytopenic Thrombosis

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**Abstract:** A 63 year old male presented to our hospital with NonST Elevated Myocardial Infarction and high grade stenotic lesions in distal left main artery and mid right coronary artery. He underwent an Intra Aortic Balloon Pump (IABP), heparin initiation, Coronary Artery Bypass Grafting (CABG) and got discharged. On 9<sup>th</sup> postoperative day he presented with STElevated MI, thrombosis of all grafts, thrombocytopenia due to heparin (Heparin Induced Thrombocytopenic Thrombosis). A rescue Per Cutaneous Intervention with Intra Aortic Balloon Pump of native vessels has been done with use of argotroban as alternative anticoagulant. Later Angiogram showed patent lumen with TIMI-3 flow. He was discharged with improved platelet count and stabilized condition.

**Keywords:** Intra Aortic Balloon Pump, Graft Failure, Heparin, Heparin induced thrombocytopenic thrombosis, Thrombosis

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### I. Introduction:

Postoperative Myocardial Infarction due to early graft failure following Coronary Artery Bypass Grafting as a result of Heparin induced Thrombocytopenic Thrombosis is often under-recognized, potentially life threatening complication of Heparin therapy. There is a reported incidence of 1-2.4% of all cardiac surgeries with higher incidence in venous grafts to arterial grafts. There are 2 types of HIT with TYPE-2 being more threatening than TYPE-1. It is an Immune mediated antibody formation against heparin complexed to platelet factor 4 (PF4). Coronary Angiogram has been demonstrated to be valuable tool for determining the cause. To date best approach for treatment remains unclear and controversial. The options could be PCI with IABP /redo CABG surgery / medical management. By writing this case we propose Intra Aortic Balloon Pumping as adjunct therapy to PCI in postoperative graft failure due to HIT.

### II. Case Presentation:

A 63 year old man with unremarkable past medical history except chronic smoking for 30 years presented to emergency with a 2 week history of exertional angina progressing to rest angina over past 24 hours. He complained of precordial chest pain, 8/10, radiating to both upper arms and shortness of breath. No significant family history, No known drug allergies.

On admission he was hemodynamically stable with normal range of vitals and normal physical examination (PE) findings. His EKG showed <1mm ST depression ("Fig 1"). There was mild elevation of initial set of cardiac biomarkers. Due to persistence of symptoms, he was referred to early diagnostic cardiac catheterization showing high stenotic lesions in distal left main artery and mid right coronary artery ("Fig 2 and Fig 3"). He was operated and discharged 4 days later.

After 5 days of post discharge, patient returned to emergency with history of 1 hour crescendo substernal chest pain which is radiating to left arm, jaw, 5/10 severity, diaphoretic with cold extremities and shortness of breath. No significant family history, No known drug allergies.

On examination he was found pale, hypotensive, tachypnoeic, normal S1S2, no murmurs, no JVD and rest of PE was normal. His EKG showed >2mm ST-segment elevation in Inferior Lateral distribution ("Fig 4"). Elevated cardiac biomarkers with Troponin T of 7.19 ng/ml, CK-MB Index of 9.9% and CKMB fraction of 147.2 ng/ml. His blood count showed anemia, thrombocytopenia and leukocytosis and elevated levels of HIT antibody (heparin-platelet factor 4). Urgent Coronary Angiogram revealed thrombosis of all grafts. ("Fig 5 and Fig 6") Chest XRay showed subpulmonary pleural fluid on right side ("Fig 7"). Computer Tomography (CT) of Lungs showed a subsegmental Pulmonary Embolism and large loculated pleural effusion. Trans Thoracic Echo (TTE) revealed a dense, pedunculated Left Ventricular Thrombus at apex. (Fig 8)

### III. Treatment:

On his first arrival with high stenotic lesions patient was treated with Intra Aortic Balloon Pump (IABP) insertion and initiation of Heparin followed by off pump Coronary Artery Bypass Grafting (CABG) on

the next day. Postoperative course remained uneventful and was discharged to a rehabilitation centre after 4 days with Metoprolol/Aspirin/Furesomide.

On 9<sup>th</sup> postop day when he presented with STEMI and thrombosis of all grafts, a rescue PCI with IABP was performed for the native vessels. Thrombolysis in MI flow grade 3 (TIMI-3) flow was established following revascularization and drug eluting stent placement of mid left Anterior descending and mid right coronary artery ("Fig 9 and Fig 10"). Cessation of all heparins, alternative anticoagulation with Argatroban was initiated. Transient bradyarrhythmias were treated with atropine and continuous intravenous dobutamine was initiated due to persistent hypotension. Aggressive intravenous fluid hydration and inotropic support were tapered by hospital day 2. IABP therapy was discontinued on hospital day 3 due to continued improvement in hemodynamics. Argatroban was transitioned to oral warfarin on hospital day 10.

#### **IV. Outcome And Follow Up :**

Patient doing well with improved platelet count (244) and improved physical condition was discharged after 23 days following rescue PCI on ASA/Plavix/Metoprolol/Enalapril/Crestor/digoxin/aldactone with follow up as outpatient.

#### **V. Discussion:**

- Type 2 HIT (Immune mediated) involves formation of multimolecular complexes between heparin, platelet factor 4 and antibodies (IgG) generated against heparin:PF4. These complexes result in endothelial activation, increased tissue factor synthesis, thrombin generation, platelet activation leading to thrombus formation. HIT is more severe and protracted form of HIT with macrovascular thrombosis and thrombus induced end organ dysfunction. Venous thrombosis is more common than arterial thrombosis. HIT following CABG may present as bypass graft occlusion, left atrial thrombus, valvular thrombosis or pulmonary embolism. Other clinical presentations could be heparin induced skin lesions, heparin resistance and adrenal vein thrombosis leading to hemorrhagic infarction.
- Coronary angiography is a safe and valuable tool for detecting graft failure due to thrombosis and postoperative MI.
- Following angiographic diagnosis, treatment strategy can be 1) immediate re-intervention by either PCI (with IABP) or redo CABG 2) conservative management.
- In presence of diffuse graft thrombosis, native vessel is likely the best target of intervention to minimize the risk of massive distal microvascular embolization.
- PCI could be preferred to redo CABG due to the latter causing increased myocardial damage leading to more frequently postoperative LCOS (low cardiac output syndrome).

#### **VI. Figures :**

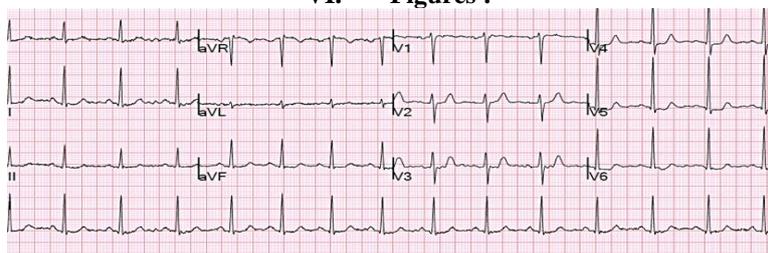


Fig 1 EKG showing  $<1\text{mm}$  ST depression

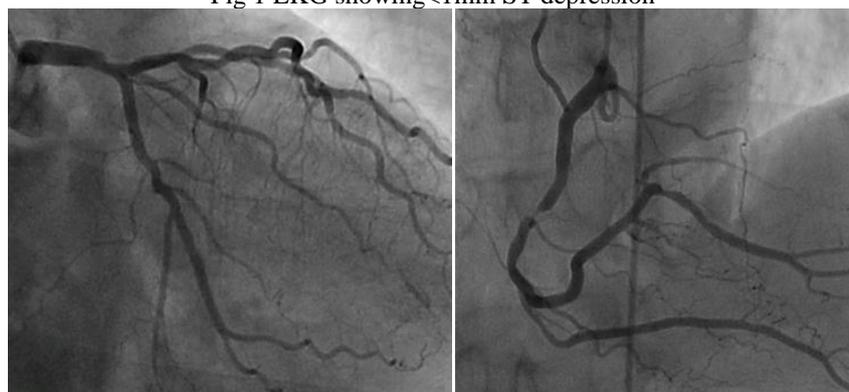


Fig 2 and 3 cardiac catheterization showing high stenotic lesions in distal left main artery and mid right coronary artery

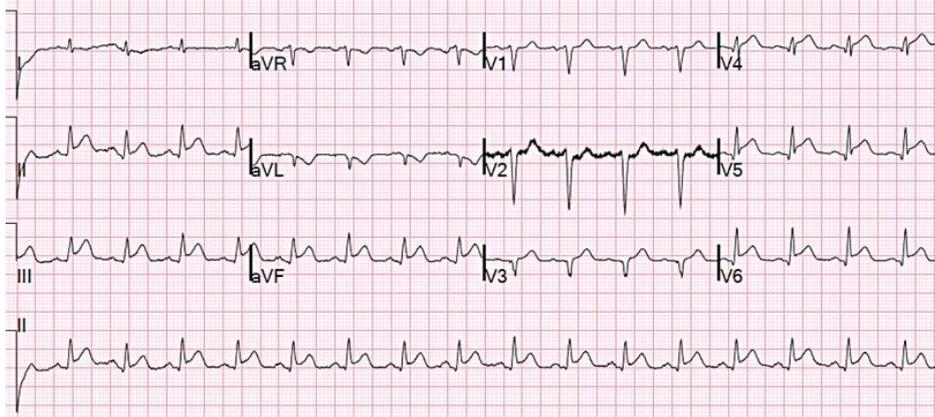


Fig 4 EKG showing >2mm ST-segment elevation in Inferior Lateral distribution.

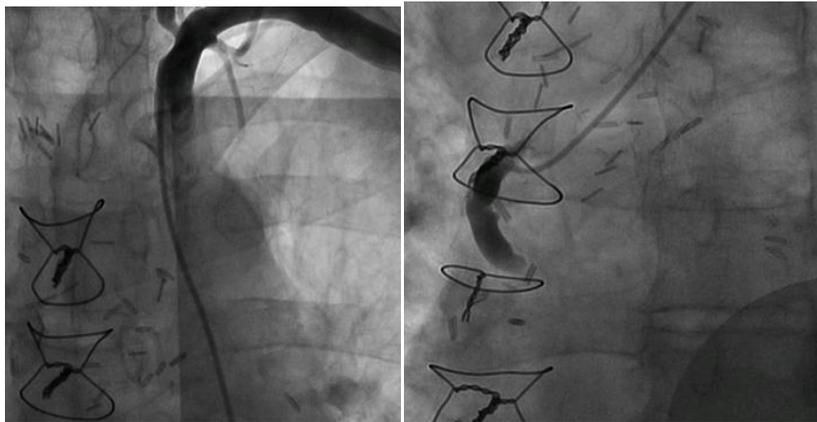


Fig 5 and Fig 6 Urgent Coronary Angiogram revealed thrombosis of all grafts



Fig 7 Chest XRay showed subpulmonary pleural fluid on right side



Fig 8 Trans Thoracic Echo(TTE) revealed a dense ,pedunculated Left Ventricular Thrombus at apex.



Fig 9 and Fig 10 Thrombolysis in MI flow grade 3 (TIMI-3) flow was established following revascularization and drug eluting stent placement of mid left Anterior descending and mid right coronary artery

## VII. Conclusion

- IABP adjunct to PCI can be used in cases of HIT based on following advantages of IABP use
  - IMPROVED VENTRICULAR FUNCTION
  - IMPROVED NATIVE VESSEL REPERFUSION BY COUNTERPULSATION
  - IMPROVED SYSTEMIC PERFUSION BY AUGMENTING AORTIC ROOT'S ELASTIC RECOIL.
- IABP therapy in postoperative MI due to graft failure after CABG is seldom described in literature and under utilized.

With favourable outcome of our case,we propose the use of IABP as adjunct therapy to PCI in management of postoperative graft failure after CABG.

## VIII. Learning Points ( Patients View):

- Elimination of all forms of Heparin is most essential element in treatment of HIT.
- Patients receiving heparin should have platelet count monitoring at baseline and at least every third day between day 5 and day14 of heparin exposure.
- IABP primary goal is to improve ventricular performance by improving coronary artery perfusion and decreasing myocardial oxygen demand.
- IABP adjunct to PCI can be preferred over redo CABG with successful outcomes as in our case.

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