

PCI for in-stent restenosis in a patient with acute coronary syndrome, cardiogenic shock and thrombocytopenia secondary to myelodysplastic syndrome

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Abstract

Cardiogenic shock occurs in 2.5% patients with non ST elevation myocardial infarction as compared to 5-8% of patients with ST elevation myocardial infarction. Percutaneous coronary intervention for ischemia related cardiogenic shock in patients with myelodysplastic syndrome and thrombocytopenia presents a unique challenge for the interventional cardiologist and hematologist. The patient in our case report presented with non ST elevation myocardial infarction, severe thrombocytopenia and cardiogenic shock. He had in-stent restenosis in prior bare metal stents and was declined for coronary artery bypass grafting (CABG), limiting the treatment options. This is the first case report of percutaneous coronary intervention for in-stent restenosis in myelodysplastic syndrome. The procedure was successful and patient did well on one year follow up.

Keywords: percutaneous coronary intervention (PCI); thrombocytopenia; myelodysplastic syndrome (MDS)

Case presentation

66-year-old gentleman was admitted with a history of upper abdominal discomfort and progressively worsening shortness of breath on exertion for the past three weeks. The patient presented to the emergency department with shortness of breath at rest along with chest pain and unable to lay flat. The patient's known co-morbid conditions include myelodysplastic syndrome, hypertension, diabetes mellitus and ischemic heart disease. Two months prior to this admission the patient was admitted with non ST elevation myocardial infarction and pulmonary edema. He underwent percutaneous intervention of left anterior descending artery with bare metal stents due to low platelets.

On examination, the patient was vitally stable with bibasilar crepitations. His ECG on admission revealed sinus rhythm with left axis deviation, ST segment elevation in aVR and ST depressions in leads I, aVL and V4-V6 (Figure 1). The chest X-ray showed bilateral hilar congestion consistent with heart failure. His workup showed raised troponin-I levels, creatinine 1.3, hemoglobin 8.9 and platelet count of 27,000. An echocardiogram was done that revealed severe left ventricular (LV) dysfunction with segmental wall motion abnormality of LAD territory. A diagnosis of non ST elevation myocardial infarction with heart failure was made. The patient did not respond to initial treatment including dual antiplatelets and diuretics. Subsequently he

developed worsening symptoms of chest pain and more marked ECG changes including ST elevation in aVR and V1 and ST depression with T wave inversions in leads V2-V6. The patient was intubated due to pulmonary edema and respiratory distress. His urine output decreased and blood creatinine level jumped up. His blood pressure also decreased; requiring inotropes norepinephrine and dopamine. Two units of regular platelets and one unit mega platelets were transfused to keep platelet count more than 50000 and coronary angiogram performed. The coronary angiogram revealed an 80% stenosis in ostio-proximal segment of left anterior descending artery (LAD) and significant in-stent restenosis in the proximal and mid LAD stents (Figure 2). There was a 70% stenosis in proximal segment of left circumflex artery (LCx) with haziness. The

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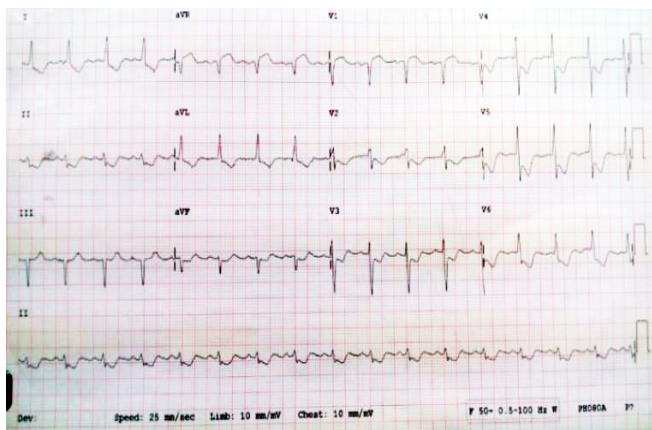


Figure 1 Electrocardiogram showing ST segment depressions in leads & ST segment elevations in leads.

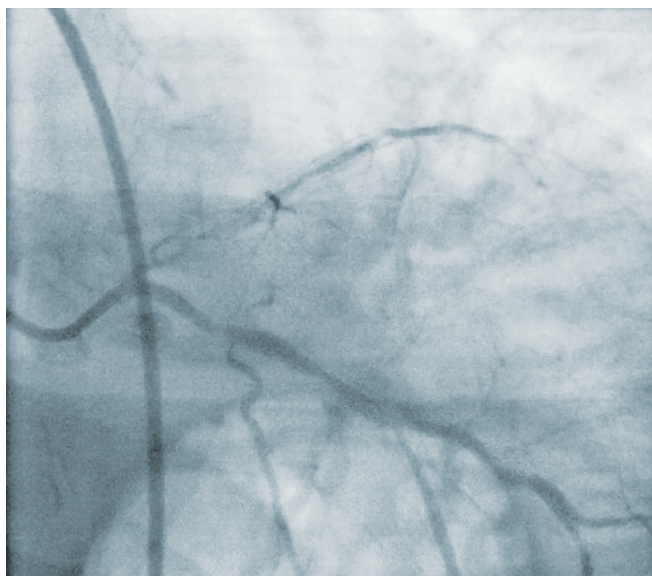


Figure 2 AP caudal view showing in-stent stenosis in proximal left anterior descending artery; significant disease in ostio-proximal segment of left anterior descending artery.

right coronary artery (RCA) angiogram was not done; as it was chronic total occluded (CTO) as per the prior angiogram performed two months ago. The hematology team was of opinion that coronary artery bypass grafting (CABG) cannot be done due to advanced myelodysplastic syndrome. Not many options left because patient did not do well with the bare metal stents and developed in-stent restenosis. As there is now enough data about safety of polymer free drug eluting stents and newer generation stents regarding short duration of antiplatelets. We decided to percutaneously revascularize with everolimus eluting stents, which were available in our cardiac catheterization laboratory. Polymer free drug eluting stents were not available at the time of procedure. Left main (LM) coronary artery was engaged with Voda left™ 3 (Boston scientific) 7 French guiding catheter. LAD and LCx wired with Runthrough Intermediate wire (Terumo). The prior stents and proximal segment of LAD predilated with semi-compliant balloon 2.5 × 15 mm (Saphire, OrbusNeich) at 6 -10 atm and noncompliant balloon 3.0 × 15 mm (NC Trek, Abbott Vascular) at 8-18 atm. An everolimus eluting stent 3.5 × 23 mm (Xience, Abbott Vascular) was deployed from LM to LAD at 12 atm. The prior bare metal stents were treated with drug coated balloons (Sequent Please 2.5 × 26 mm and Sequent 3 × 17

mm). The LM to LAD stent post dilated with noncompliant balloon 3.5 × 15 mm (NC Trek, Abbott Vascular) at 8-16 atm. An everolimus eluting stent 3.5 × 12 mm (Xience, Abbott Vascular) deployed in the proximal LCx at 12 atm and post dilated with noncompliant balloon 3.5 × 15 mm (NC Trek, Abbott Vascular) up to 16 atm (Figure 3). The intra-aortic balloon pump (compatible with 7.5 French sheath) inserted from same right femoral artery access in the end of procedure to prevent multiple arterial punctures. IABP was the only percutaneous cardiac assist device available in our country, although the effectiveness of IABP in high risk PCI patients was under discussion during the time of this procedure. Post procedure patient remained stable. Heparin infusion was given for IABP, maintaining an APTT between 50-70 sec. The blood count was regularly checked and our aim was to keep platelets above 50000 and hemoglobin above 10 gram/dl. The patient was transfused one mega unit of platelets 24 h post procedure and then 72 h post procedure.

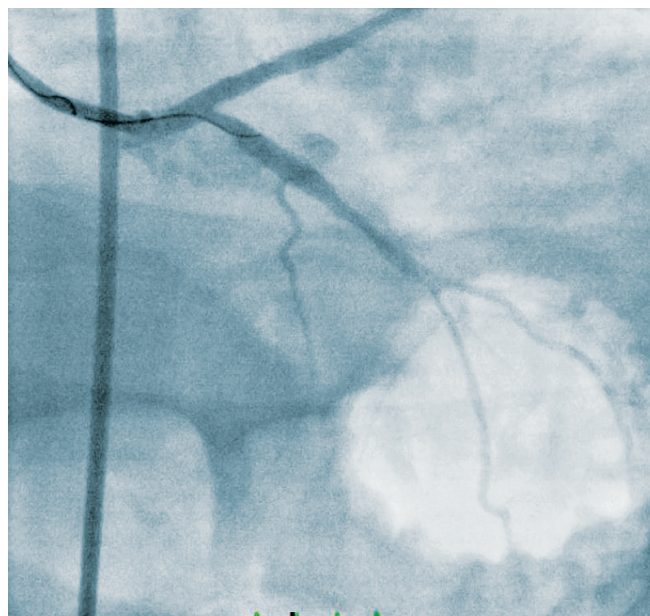


Figure 3 RAO Caudal view showing final angiogram result after PCI of left anterior descending artery and left circumflex artery.

Post procedure patient's blood pressure improved and urine output picked up which allowed us to taper off the inotropes. The patient's renal function improved. The intra-aortic balloon pump (IABP) was removed 48 hours post procedure after transfusion of two units of regular platelets. The patient was subsequently extubated after 72 h. He remained stable throughout his hospital stay. During his hospital stay the patient did not have any incidence of bleeding. He was subsequently discharged on aspirin 75 mg QD, clopidogrel 75 mg QD, atorvastatin 40 mg HS, Carvedilol, ACE Inhibitor and spironolactone. The patient had regular follow up with cardiologist and hematologist. The clopidogrel was continued for six months then reduced to every other day and discontinued at one year. The intension was to stop clopidogrel, if patient develops further drop in platelet count, bleeding or decrease in hemoglobin. The patient remained asymptomatic and did not require blood products. Although there is literature as mentioned above about short duration of dual antiplatelets; but in our patient the lesion was not

denovo and patient developed in-stent restenosis (ISR) after placement of bare metal stents. Moreover it was complex PCI of LAD and LCx including left main artery with multiple overlapping stents. The chances for late stent thrombosis were still considerable. His platelet counts and hemoglobin were regularly checked. The platelet count remained between 21000 to 36000 and hemoglobin above 10 g/dl. This encouraged us to continue dual antiplatelets for more than three months to reduce the risk of late stent thrombosis.

Discussion

This case report presents a unique challenge. In a patient who has significant coronary artery disease and myelodysplastic syndrome (MDS) with thrombocytopenia, dual antiplatelet therapy (DAPT) would place him at a very high risk of developing bleeding [1, 2]. In such a situation placement of a bare metal stent (BMS) would seem to be the most appropriate choice in terms of percutaneous coronary artery revascularization and coronary bypass grafting [3]. However here is a patient who developed in-stent restenosis (ISR) after placement of bare metal stents a few months ago and now has cardiogenic shock with pulmonary edema secondary to NSTEMI, requiring mechanical ventilation. ISR can be defined clinically as presentation of recurrent angina or objective evidence of myocardial ischemia, whereas angiographically it is defined as > 50% diameter stenosis in the stented segment [4]. ISR after bare metal stent implantation is characterized by vascular injury from implantation of metallic stent followed by neointimal proliferation and inflammation [5]. There is now data which suggests that we can stop dual antiplatelets at one month in case of polymer free drug coated stent and at three months with new generation drug eluting stents [6-8]. Since the patient did develop ISR after BMS, we decided to put drug eluting stents. Although with thorough search of literature, we could not find a single case report regarding managing ISR in MDS patient with thrombocytopenia.

We committed this patient to dual antiplatelet therapy, the safety of which is not established in presence of thrombocytopenia secondary to MDS. Cardiovascular morbidity and mortality in MDS is secondary to anemia [9]. Anemia in these patients causes a decrease in afterload, an increase in preload and in turn leads to cardiac remodeling, left ventricular hypertrophy and worsening of congestive heart failure [10]. Anemia itself would worsen angina due to supply demand mismatch. Patients who are on DAPT and have thrombocytopenia are at increased risk of developing anemia secondary to bleeding. Thrombocytopenia does not place patients at lower risk of developing significant coronary artery disease. Due to hematopoietic dysregulation the platelets in MDS may be of increased or decreased size. That theoretically would provide a greater surface area for adhesion to endothelial surface [11], predisposing to coronary artery thrombosis.

Percutaneous coronary artery revascularization in presence of thrombocytopenia is very rare. Bare metal stents have been used in most cases. Most of these patients have had idiopathic thrombocytopenic purpura (ITP) that led to a low platelet count [12]. Patients who have undergone PCI have

been pretreated with prophylactic platelet transfusions, intra-venous immunoglobins (IVIG) or rituximab. Rarely has PCI been done in a patient with MDS and thrombocytopenia [3]. Thus use of drug eluting stents is an option in patients with thrombocytopenia and cardiogenic shock; however the decision should be undertaken with a multidisciplinary approach and on a case by case basis.

Conflicts of interest (--B--)

Authors declare no conflicts of interest.

References

- [1] Moretti C, Teresa Lucciola M, Morena L, Biondi-Zoccai G, Laudito A, et al. Idiopathic thrombocytopenic purpura and percutaneous coronary stenting: a dangerous duo? *Int J Cardiol.* 2008; 130(3):e96-97.
- [2] Tefferi A, Vardiman JW. Myelodysplastic syndromes. *N Engl J Med.* 2009; 361(19):1872-1885.
- [3] Miyag Y, Yamauchi S, Suzuki S, Kitagawa A, Masaki Y, et al. Investigation of coronary artery bypass grafting for a patient with myelodysplastic syndrome. *Ann Thorac Cardiovasc Surg.* 2001; 7(4):250-253.
- [4] Dangas GD, Claessen BE, Caixeta A, Sanidas EA, Mintz GS, et al. In-stent restenosis in the drug-eluting stent era. *J Am Coll Cardiol.* 2010; 56(23):1897-1907.
- [5] Demircelik B, Altinsoy M, Bozduvan F, Gunes M, Cakmak M, et al. Coronary intervention for acute coronary syndrome in a 51-year-old man with immune thrombocytopenic purpura: A case report. *J Med Case Rep.* 2014; 8:214.
- [6] Urban P, Meredith IT, Abizaid A, Pocock SJ, Carrié D, et al. Polymer-free drug-coated coronary stents in patients at high bleeding risk. *N Engl J Med.* 2015; 373 (21):2038-2047.
- [7] Yusuf SW, Iliescu C, Bathina JD, Daher IN, Durand JB. Antiplatelet therapy and percutaneous coronary intervention in patients with acute coronary syndrome and thrombocytopenia. *Tex Heart Inst J.* 2010; 37(3):336-340.
- [8] Oliveira W, Meireles GCX, Longhi A, Beltrão P, Pimenta J. In-hospital and late outcomes after coronary stenting in patient with unstable angina and myelodysplastic syndrome. *Arq Bras Cardiol.* 2006; 87(5):e168-e171.
- [9] Oliva EN, Schey C, Hutchings AS. A review of anemia as a cardiovascular risk factor in patients with myelodysplastic syndromes. *Am J Blood Res.* 2011; 1(2):160-166.
- [10] Germing U, Kobbe G, Haas R, Gattermann N. Myelodysplastic syndromes: Diagnosis, prognosis, and treatment. *Dtsch Arztebl Int.* 2013; 110(46):783-790.
- [11] Germing U, Platzbecker U, Giagounidis A, Aul C. Platelet morphology, platelet mass, platelet count and prognosis in patients with myelodysplastic syndromes. *Br J Haematol.* 2007; 138(3):399-400.
- [12] Besa EC. Myelodysplastic syndromes (refractory anemia). A perspective of the biologic, clinical, and therapeutic issues. *Med Clin North Am.* 1992; 76(3):599-617.