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Tirofiban and rivaroxaban use in the management of right coronary artery thrombus as a primary manifestation of essential thrombocythemia

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CASE REPORT

Tirofiban and Rivaroxaban use in the management of right coronary artery thrombus as a primary manifestation of essential thrombocythemia

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Abstract

A 45 year old male with hypertension was presented to our centre with a recent inferior wall myocardial infarction (IWMI) and post infarction angina. Invasive coronary angiography revealed an occluded proximal right coronary artery (RCA) with high thrombus burden, in the absence of obstructive disease in the remaining coronary vasculature. Based on raised platelet counts of 923,000/microliter and positive Janus kinase (JAK 2- V617) mutations tested by polymerase chain reaction (PCR), a diagnosis of essential thrombocythemia (ET) was made. A therapeutic strategy of aspiration thrombectomy along with I/V Tirofiban was used for three days, followed by reassessed angiogram and percutaneous coronary intervention (PCI) with drug eluting stent (DES) placement was applied.

In addition to dual antiplatelet and statin therapy, patient was treated with Rivaroxaban 15 mg once daily for a month and Hydroxyurea 500mg twice daily. At one month follow up, patient was asymptomatic, with decreasing platelet counts and no bleeding complications.

Keywords: Inferior wall MI High thrombus burden Essential thrombocythemia Tirofiban Rivaroxaban.

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Introduction

Essential thrombocythemia (ET) is characterized by JAK 2 or Cal-reticulin mutations endowing unchecked bone marrow megakaryocytic proliferation and raised peripheral blood platelet counts.[1] Coronary arterial thrombus presenting as acute coronary syndrome (ACS) can be a rare presenting feature in ET.[2]

After obtaining informed consent from the patient to utilize his medical records, we present a case of late arrival Inferior Myocardial Infarction (MI) and post infarction angina with underlying high thrombus burden in the right coronary artery as the first clinical presentation of ET in a 45 year old male in the absence of major CAD risk factors.

Case Report

A 45 year old male, was referred to our centre from a remote northern district. Ten days ago, he reported sudden onset, severe, nonspecific retrosternal pain radiating to the epigastric area, had no exacerbating or relieving factors lasting greater than thirty minutes. He went to the local hospital, where a twelve lead electrocardiogram (EKG) was performed.

ST elevation in inferior leads II, III and AVF were observed. Being devoid of any thrombolytic or primary PCI facilities, patient was managed medically with dual anti platelet and heparin therapy, with referral to our centre for further management.

Over the next few days, patient had recurrent episodes of angina at rest and minimal activity, not alleviated by prescribed nitrates and beta blocker therapy. Prior medical history was remarkable for hypertension (HTN) well controlled on once daily Losartan. No family history of premature coronary artery disease was reported.

Systemic examination was unremarkable.

Clinical examination revealed a well -built, middle aged male, haemodynamically stable. General physical examination was unremarkable, cardiovascular, gastro-intestinal and other system's examination revealed no abnormalities. Lab parameters were remarkable for a raised platelet count (table). Echocardiogram revealed an ejection fraction of 55% and an inferior basal wall motion hypo kinetic abnormality with mild mitral regurgitation.

Given patient's post MI angina; invasive coronary

Table: Laboratory tests.

Haemoglobin: (gm/dl)	14.2 gm/dl (Normal range:11-14.5 gm/dl)
Total Leukocyte count: (cells x10E9/L)	7000 cells x10E9/L) (Normal range:4.6-10.8 cells x10E9/L)
Platelets:(cells x10E9/L)	923 cells x10E9/L (Normal range: 154-433 cells x10E9/L)
Prothrombin time: (seconds)	10.9 seconds (Normal range: 9.3-12.8 seconds)
INR:(ratio)	1.1(Normal range:0.9-1.2)
APTT (seconds):	29 (Normal range:22.9-34.5 seconds)
Cre:(mg/dl)	1.1 mg/dl (Normal range:0.6-1.2 mg/dl)
Peripheral film:	Hypersegmented neutrophils

APTT: activated Partial Thromboplastin Time; Cre: creatinine; INR: International normalized ratio

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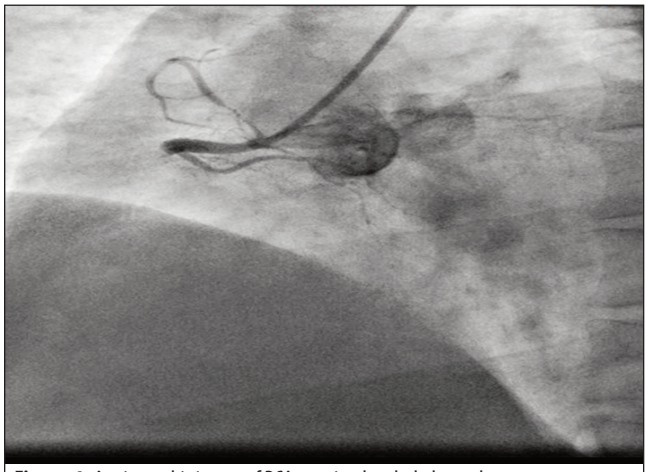

Figure-1: Angiographic image of RCA, proximal occluded vessel seen.

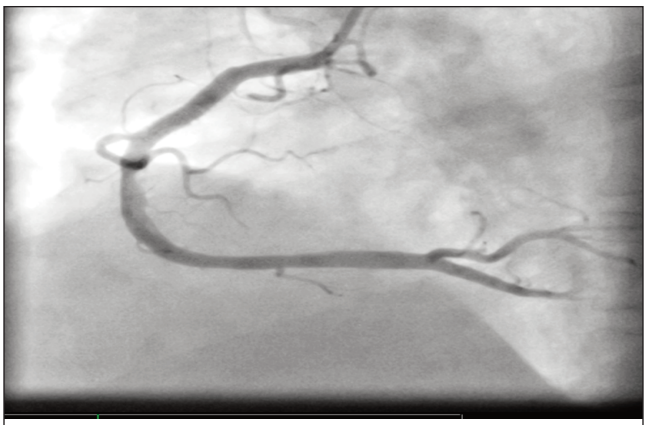

Figure-2: Angiographic image of RCA, Right anterior oblique projection after i/v Tirofiban infusion and successful PCI with drug eluting stent, TIMI III flow seen.

angiogram(ICA) with intent of revascularization was performed using right radial approach employing Tiger 5 French diagnostic catheter (Optitorque^{R}, Terumo Interventional systems), JR4 6 French guiding catheter (Boston Scientific^{R}) , to engage left main and right coronary arteries, respectively. ICA showed 100% proximal RCA stenosis (Image A), with mild plaquing in left anterior descending and left circumflex arteries. The RCA occlusion was crossed with a BMW 0.014 guide wire (Abbot^{R}). A high thrombus burden at that time precluded stent placement; hence only aspiration thrombectomy using Thrombuster II (Kaneka Medical^{R}) was performed, and patient managed with Tirofiban infusion for next three days.

Later, repeat angiography was performed with successful angioplasty of proximal RCA using DES (Image B).

A right radial approach was employed, with Sion 0.014 guide wire (Asahi^{R}) and JR4 French guiding catheter (Boston Scientific^{R}). Pre dilation of proximal RCA was done using NC Trek 4.0 * 12 mm and 4.5 * 12 mm balloons (Abbot^{R}), single Xience Prime DES 4.0 * 28 mm (Abbot^{R}) were deployed. Post dilation was achieved using a Sapphire II 3.0* 15 mm balloon (Orbusnich^{R}).

Haematology consultation for raised platelet count was sought. They advised PCR (Polymerase Chain Reaction) assay for JAK 2-V216 mutation, which was positive. Based on this, along with raised platelet count and hyper segmented neutrophils on peripheral film, a diagnosis of ET was established. Patient was discharged home on dual antiplatelet and Rivaroxaban 15 mg once daily for a month, along with Capsule Hydroxyurea 500 mg twice daily as par Haematology. Patient was stable, asymptomatic with decreased platelet counts at 656,000/ micro litre at one month follow up in the Cardiology clinic.

Discussion

Conventional ACS comprises pre-existing plaque rupture and the ensuing activated, aggregated adhesive platelet rich thrombus which may in order of progressive severity variably obstruct or occlude coronary blood flow.[3] ACS occurred in 9.4 percent patients with ET in the Rossi et al case series of 170 patients; albeit 75% of them had risk factors of CAD.[4]

In ET; implicated mechanisms are abnormally activated, pro coagulant behaviour of platelets with altered glycoproteins and add on endothelial dysfunction causing the culprit thrombosis.[5]

This is a case of premature coronary artery disease (CAD) occurring in a healthy 45 year old male with HTN, albeit well controlled, as his sole risk factor for CAD. ET in this patient presented as an occlusive high thrombus burden disease in a single vessel, thus highlighting this haematological disorder as an unconventional, significant risk factor for STEMI in young adults.[6]

The involvement of RCA represents a rare culprit vessel in ET associated STEMI, which reportedly occurs in Left anterior descending artery.[7] The high thrombus burden attendant of this disorder required expectant medical and interventional strategies, clearly lacking in guidelines.

Our strategy was initial aspiration thrombectomy, I/V Tirofiban to treat the thrombus burden, later followed by DES (Drug Eluting Stent) placement, comparable to literature reports of aspiration thrombectomy, and glycoprotein IIb/IIIa inhibitor use.[8] Medical management comprised additional novel anticoagulant to continue in the first month post angioplasty.

ET represents a non-traditional risk factor for premature CAD, with regard to this case and previous similar reports in international literature.[9,10]

Conclusion

ST elevation myocardial infarction can be the index manifestation of essential thrombocythemia. Right coronary artery represents a rare culprit vessel implicated in ET associated STEMI. A therapeutic approach targeting attendant high thrombus burden comprising glycoprotein II b/III a inhibitor use, deferred angioplasty and novel anticoagulant with good clinical and angiographic outcomes may serve as a useful therapeutic strategy with regard to attendant high thrombus burden as in our case.

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Conflict of interest: None.

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