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A case of premature myocardial infarction

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Abstract

A 28 years old male admitted with anginal type of chest pain for 4 hours. Pain was left sided and radiating to jaw, and present even during rest. Patient was known case of Hyper Homocysteinemia on vitamin B12 and folic acid supplementation. Patient also had history of cerebral venous thrombosis in 2019, from then he was taking Warfarin and under regular follow up. On reviewing the records , the prothrombin time and INR (international normalized ratio) was however sub therapeutic recently. ECG was taken, which showed ST elevation in Lead I and a VL , suggestive of a high lateral wall myocardial infarction. Patient was taken up for primary angioplasty .Coronary angiogram showed total thrombotic occlusion of proximal Left anterior descending artery. LAD was wired and Balloon dilatation of the lesion was done. Post procedure LAD had TIMI grade IV thrombus. Thrombus aspiration was done followed by intracoronary streptokinase as the thrombus load was not reducing even after aspiration with aspiration catheters. Despite adequate measures, post procedure Thrombus burden was grade 4 and only TIMI II flow was established. Patient was hemodynamically stable. He was started on tirofiban infusion for 18 hours and managed in coronary ICU. Post procedure check angiogram was repeated after 48 hours which showed TIMI III flow and thrombus was absent. Pt was discharged with Antiplatelets and anticoagulant and was advised to review after 1 month.

Later he was followed up at 3 months , in functional class 1 and on optimum regular drugs

KEYWORDS: Hyperhomocysteinemia, Thrombus aspiration, TIMI thrombus burden

1.INTRODUCTION

Hyper homocysteinemia was recognized as the major risk factor for coronary events in the past. In patients with acute coronary syndrome, increased levels of homocysteine are associated with Prothrombotic state and increased platelet aggregation.[1]. Homocysteine is metabolized to methionine via remethylation pathway / trans sulfuration. Failure of which leads to Hyper homocysteinemia[2]. It is risk factor for both arterial and venous thrombosis. A C-to- T mutation at position 677 in the methylene tetrahydrofolate reductase gene (MTHFR) gives rise to thermolabile variant with reduced activity accompanied by elevated plasma homocysteine. Moderate Hyperhomocysteinemia is now recognized as a risk factor for arterial disease including carotid artery stenosis and stroke.[3]

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2.CASE PRESENTATION:

28 year, Male a known case of hyper homocysteinemia, admitted with complains of chest pain for 4 hours. Pain was left sided and radiating to jaw , and present even during rest. Patient was known case of Hyperhomocysteinemia on vitamin B12 and folic acid supplementation. Patient also had history of cerebral venous thrombosis on 2019, from then he was taking Warfarin and under regular follow up. INR at the time of admission was 1.25. No other conventional risk factors were present. Initial ECG showed ST elevation in Lead I and avL (figure 1). Patient was taken for Coronary angiogram immediately which showed Proximal LAD total thrombotic occlusion(figure 2). LAD was wired with Runthrough NS coronary wire and predilated with 3V Paulo 2x15mm balloon at 8 atm. And again predilated with Yukon Semi compliant balloon 2x15mm @12atm and Advaglide 2.5x12mm. Post Dilatation, LAD had TIMI grade IV thrombus (figure 4). Thrombus aspiration was done (figure 3) followed by intracoronary streptokinase of 1 lakh IU repeated twice. Despite adequate measures, post procedure Thrombus burden was grade 4 and only TIMI II flow was established(figure 4) at the end of the procedure . As patient was hemodynamically stable, he was started on tirofiban infusion for 18 hours and shifted to CCU. His serum Homocysteine level was 16.8 micromoles per litre (slightly high) and methyl malonic acid was 127micromoles per litre . Both were slightly high . Repeat ECG showed ST elevation in anterior leads suggestive of Proximal LAD occlusion(Figure 6).Post procedure check angiogram was repeated after 48 hours which showed TIMI III flow and thrombus was absent(Figure 5). Pt was discharged with Antiplatelets and anticoagulant and was advised to review after 1 month.

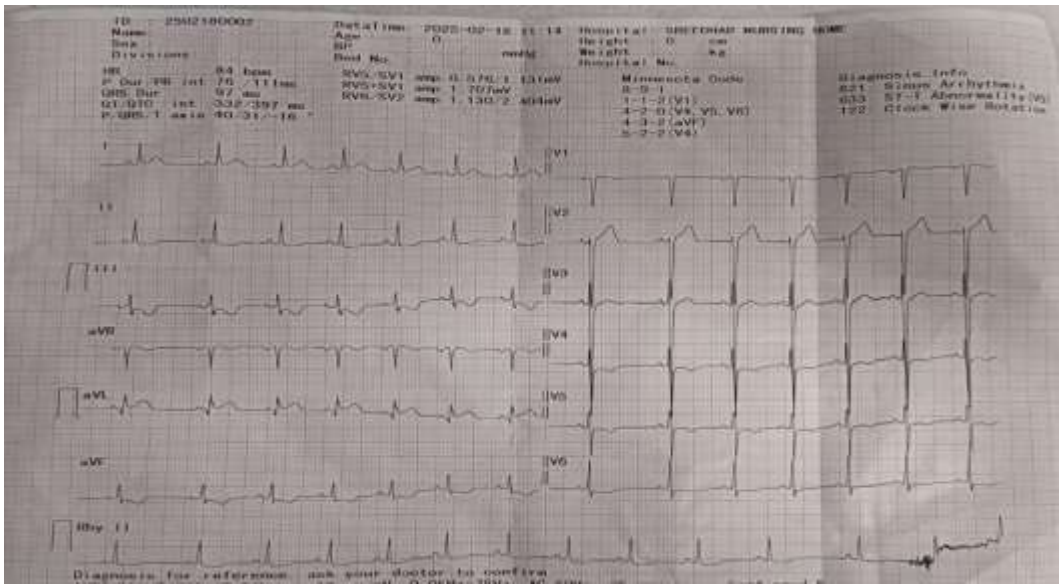

Figure 1: Preprocedural ECG; showing ST elevation in lead 1 and aVL

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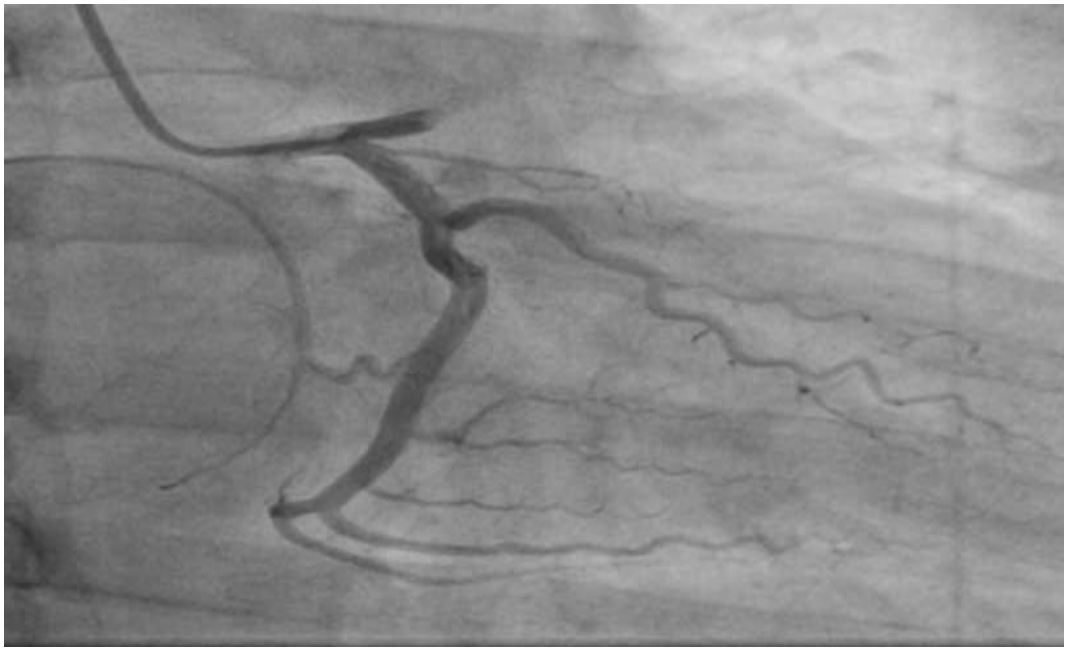

Figure 2: coronary angiogram showing Proximal LAD total occlusion

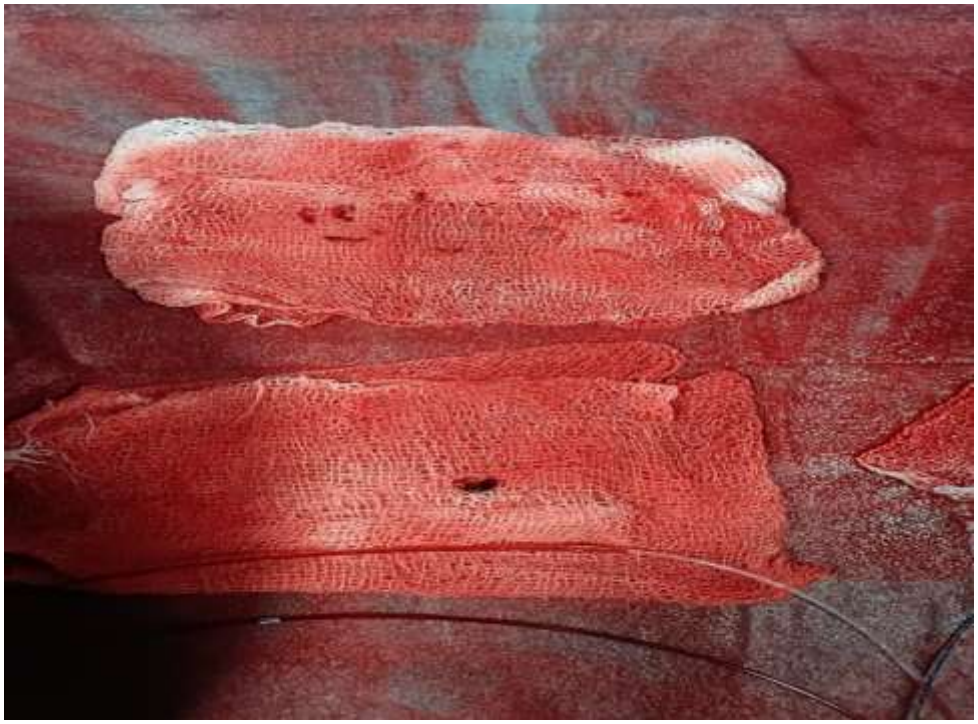

FIGURE 3: Thrombus aspirated

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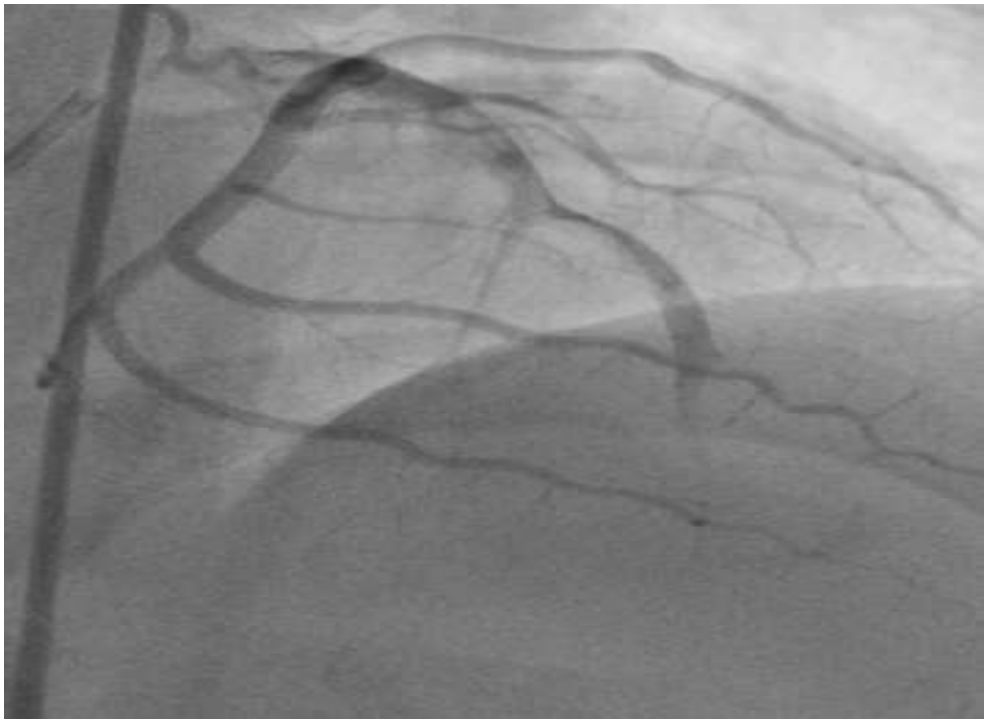

FIGURE 4: Post balloon dilatation with thrombus burden

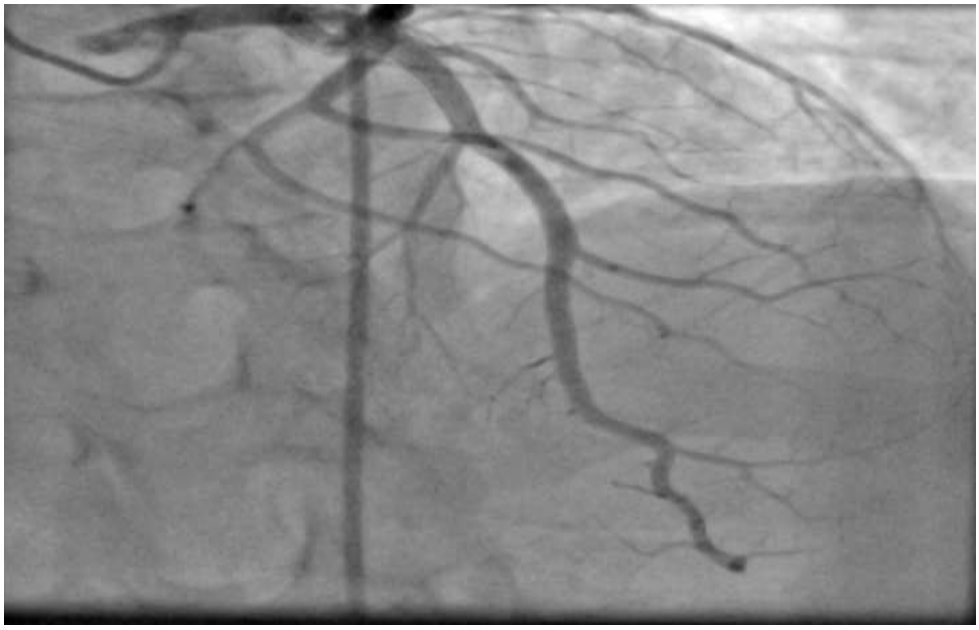

FIGURE 5: Check angiogram showing TIMI 3 flow

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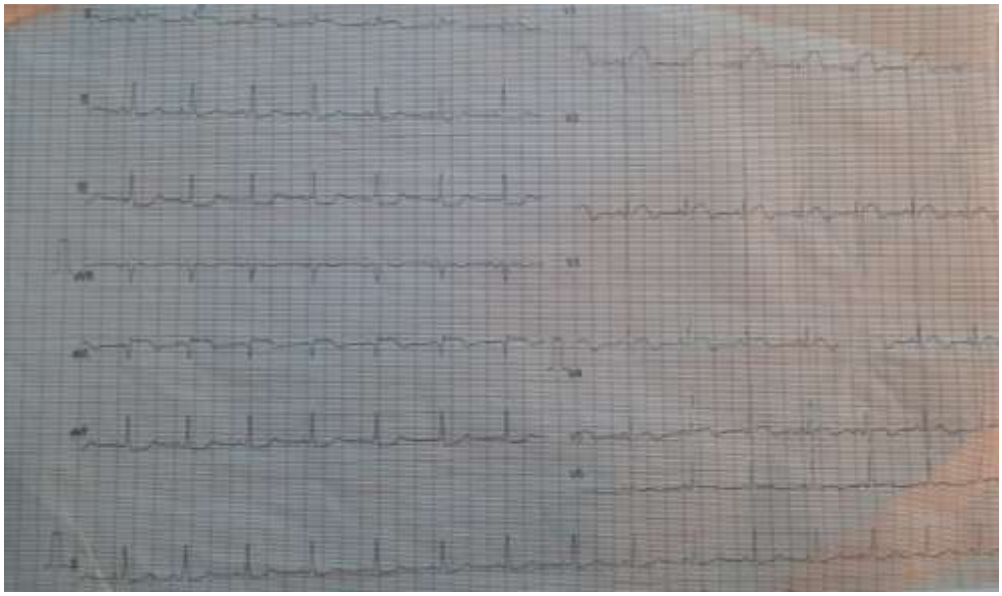

FIGURE 6: ECG-suggestive of proximal lad occlusion

3.DISCUSSION:

Premature Acute myocardial infarction, particularly in the setting of obstructive Coronary artery disease and/or female sex is an aggressive disease with high rates of recurrence and mortality, attributed largely to suboptimal control of modifiable risk factors[4][5]. Risk factors in young patients with MI with obstructive cad are same as in older patients but with significant differences in prevalence. Multiple studies have showed smoking, lipid disorders, and family history of premature CAD are more common in such patients[6]. Substance use is also prevelant in patients with premature CAD. DeFilippis et al reported cocaine and/or marijuana use was present in about 11% of $\leq$ 50 years individual and was associated with worse all cause and cardiovascular mortality [7]. Therefore screening of young patients with MI is mandatory[8].

Approximately more than one half of patients will have familial lipoprotein disorder and 20% have phenotypic diagnosis of heterozygous familial hyper cholesterolemia[9][10]. Rest of the individuals has inherited thrombophilias. Van de Water et al found an increased frequency of factor v leiden mutation and prothrombin G20210A polymorphism in 41 patients with MINOCA younger than 50 years. Another study reported that among the 84 consecutive patients with MINOCA, 23% had inherited thrombophilias and 5.7% had antiphospholipid antibody syndrome. The investigated genetic thrombophilia disorders included factor v Leiden mutation, Prothrombin G20210A polymorphism, deficiency of protein C and S and antithrombin III , Hyper homocysteinemia as well as APLA syndrome. Causes of Hyper homocysteinemia includes genetic enzyme polymorphism MTHFR, Methionine synthase, Cystathionine B synthase, dietary deficiency of folic acid, vitamin B12, B6, methionine, Renal failure, endstage Diabetes, renal failure. Therapeutic options to lower

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homocysteine include Vitamin B12 (1000-3000mcg)and Folic acid (500-5000mcg)supplementation in case of MTHFR gene mutation.

4.CONCLUSION:

Young patients with acute Myocardial infarction should be evaluated for thrombophilia if conventional risk factors are absent.

5.SOURCE OF FUNDING:

The work up of this case did not receive any specific grant from funding agencies in the public, commercial or non profit sectors.

6.ACKNOWLEDGEMENT:

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