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CASE REPORT

Dual antiplatelet agents and Rivaroxaban for massive intracoronary thrombus in STEMI

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Key Clinical Message

Management of intracoronary thrombus in patients presenting more than 12 hours after the onset of ST elevation myocardial infarction is challenging. We present such a case which had massive thrombus in left anterior descending artery. It was managed successfully with dual antiplatelet agents and factor Xa inhibitor rivaroxaban administered orally.

Keywords

Factor Xa inhibitor, massive thrombus burden.

Introduction

Cardiovascular disease burden is rising in Africa [1]. Early reperfusion is the modality of choice for treating STEMI (ST elevation myocardial infarction) [2]. Primary PCI (percutaneous coronary intervention) is the preferred method of reperfusion when it can be performed in a timely fashion by experienced operators [3]. There is a significant thrombus burden in nearly 30% of patient presenting with STEMI [4]. Management of thrombus during PCI for acute myocardial infarction could include aspiration thrombectomy, however, routine rheolytic thrombectomy has not shown clinical benefit [3]. We present here the case of a young African male presenting late with a large anterior and inferior wall myocardial infarction. He was found to have massive – extensive grade IV intracoronary thrombi [5] that was managed with dual antiplatelet agents and rivaroxaban.

Case report

We present the case of a 39-year-old African male who presented to Lutheran Medical Center in Arusha, Tanzania more than 12 hours after the onset of chest pain with anterior and inferior wall STEMI. He had no known risk factors for coronary artery disease, no history of drug abuse, or medical illness in the past. Since there was no cardiac catheterization facility available in the area, he was managed medically without fibrinolysis with aspirin, enoxaparin, and atenolol, and discharged home in 2 days. Two days after discharge he came to The Aga Khan Hospital in Dar es Salaam, Tanzania for coronary angiogram with possible PCI. On arrival, the patient had no chest pain. The ECG showed evolved anteroseptal and inferior wall myocardial infarction with persistent ST elevation in the anterior leads. (Fig. 1A). Because of the ECG evidence of anterior as well as inferior myocardial infarction it was felt that the patient probably had a large wrap around LAD (left anterior descending artery) which caused his index event. He received aspirin 300 mg, clopidogrel 600 mg, and atorvastatin 80 mg orally, and coronary angiography was performed the next day. The procedure was carried out by the right femoral approach, using six French sheath, six French Judkins coronary catheters, and six French pigtail catheter for left ventriculogram. At the end of the procedure, hemostasis was obtained by local pressure.

The coronary angiogram revealed a nondominant right coronary artery without stenosis. There was no right to

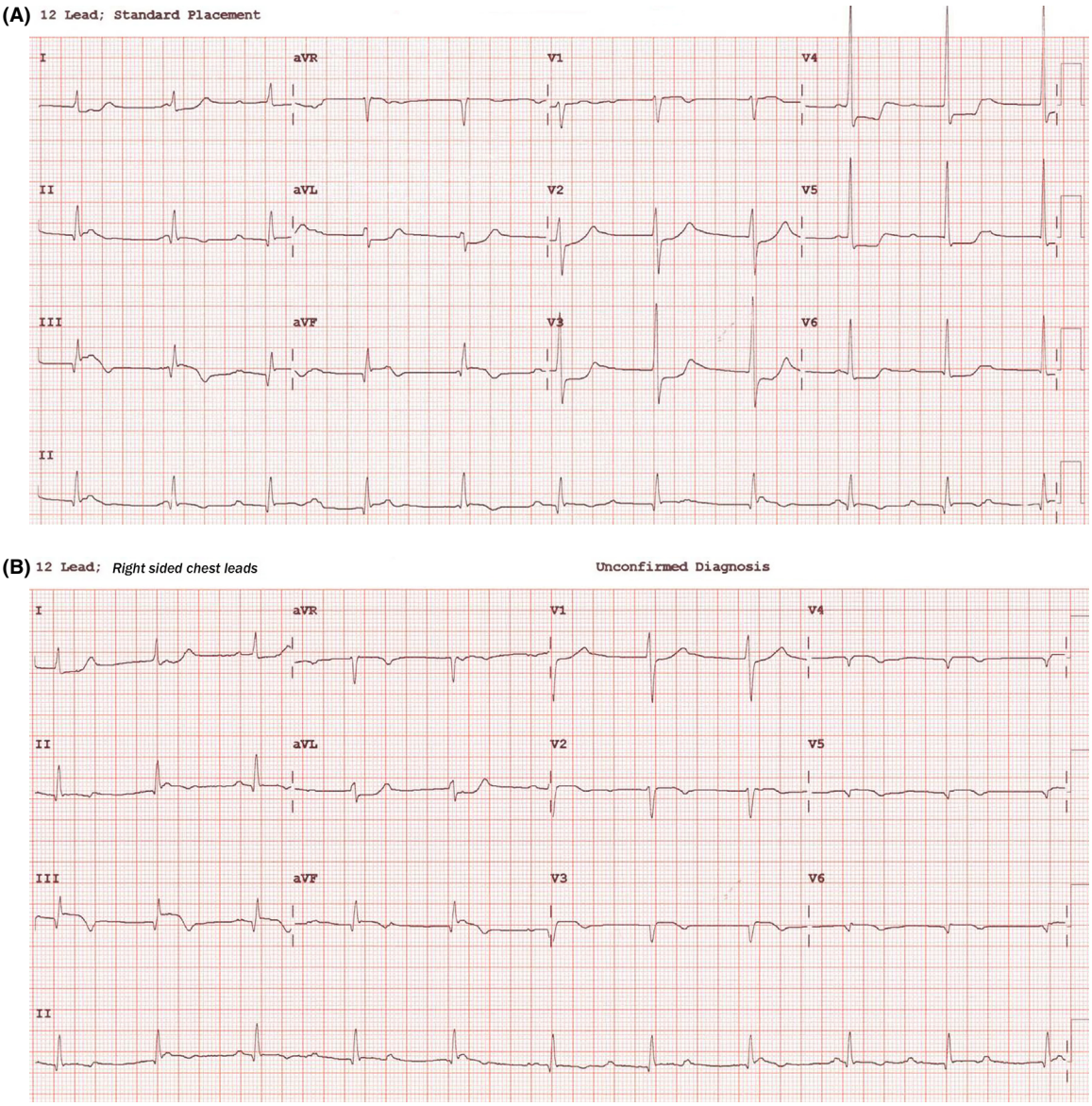

Figure 1. (A) ECG prior to the first coronary angiogram. (B) ECG prior to the follow-up coronary angiogram.

left collaterals. The LCA (left coronary artery) showed a large caliber left main trunk without stenosis. The LAD was of large caliber with a filling defect near the ostium which appeared to be a retrograde propagation from the proximal thrombus present at the site of probable ruptured plaque (Fig. 2A). The mid to distal LAD had a long linear filling defect compatible with thrombus. Distal LAD wrapped around the apex and continued as the PDA (posterior descending artery), which also appeared to have diffuse intraluminal thrombus (Fig. 2B). There was TIMI three flow in the LAD. The ramus intermedius was of moderate caliber reaching up to the apex without stenosis. The LCF (left circumflex) consisted essentially of a large posterolateral branch without stenosis. Left ventriculogram showed akinesia of left ventricular apex and adjoining distal anterolateral, and inferior walls with estimated ejection fraction (LVEF) of 30–35%. LVEDP (Left ventricular end diastolic pressure) was 22 mmHg.

Because of the massive thrombus burden in the LAD extending up to the ostium it was felt that the catheter aspiration extraction may not completely remove the thrombus and would carry a risk of embolization into the ramus and LCF. Moreover, the infarct appeared to be completed. Hence, it was decided to manage the patient conservatively and at a later date when the thrombus had cleared, PCI for any residual significant stenosis could be considered.

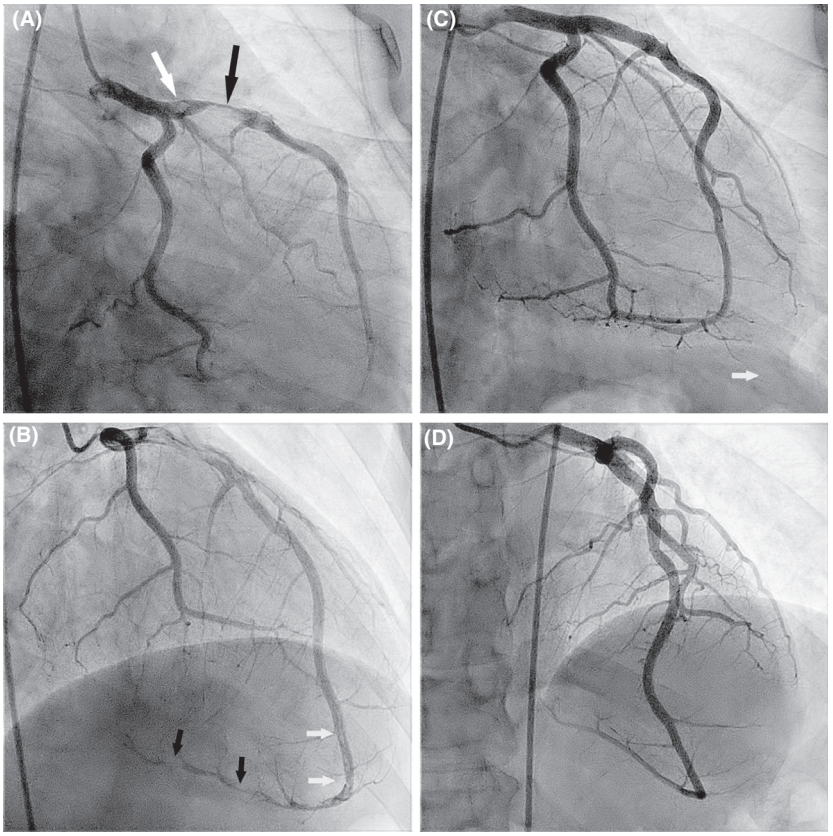

Figure 2. (A) RAO caudal view of LCA (left coronary artery) showing LAD (left anterior descending artery) ostial thrombus (white arrow), proximal thrombus, and ruptured plaque (black arrow). (B) RAO cranial view of LCA showing distal and apical LAD with linear thrombus (white arrows). The LAD wraps around the apex and continues into PDA (posterior descending artery) which also has thrombi (black arrows). (C) RAO caudal view of LCA during the follow-up angiogram showing complete resolution of LAD thrombi. (D) RAO cranial view of LCA during the follow-up angiogram showing complete resolution of LAD and PDA thrombi.

On the same day of the coronary angiogram, a 2-D echocardiogram showed normal LV chamber size with dyskinetic apex, akinetic anterior wall, and anterior interventricular septum. Estimated LVEF was about 35% with possible apical intramural thrombus.

The patient was placed on daily oral dose of aspirin 75 mg, clopidogrel 75 mg, spironolactone 12.5 mg, and atorvastatin 80 mg as well as carvedilol 3.125 mg twice a day. For anticoagulation, rivaroxaban 20 mg a day was chosen because anticoagulation with warfarin is problematic given the general difficulty and reliable monitoring of INR, as well as patient compliance with any complex warfarin dosing regimen. [6]

5 weeks later, the patient returned for a repeat coronary angiogram. He had complaint of mild chest discomfort when walking fast but no symptoms of heart failure. ECG showed further evolution of anteroseptal and inferior wall myocardial infarctions with less ST elevation and more T-wave inversions in precordial leads (Fig. 1B). Repeat coronary angiogram was carried out via right femoral approach using six French sheath and catheters. LVEDP was 12 mmHg. Coronary angiogram showed no change in RCA, ramus, and LCF. LAD was of large caliber going around the apex into a PDA. The ostium was widely patent without filling defect. The proximal LAD was also of larger caliber without filling defect or stenosis with mild plaque and some swirling of contrast (Fig. 2C). The mid, distal LAD and the PDA were patent without stenosis or filling defect (Fig. 2D). Echocardiogram showed hypokinesia of apical septum, apical lateral wall, and mid-interventricular septum with estimated LVEF about 40%. There was no evident intramural thrombus at the LV apex. The patient was discharged home on same medications except to stop rivaroxaban when the remaining 1 week supply of the medication was finished.

Discussion

ST elevation myocardial infarction is generally caused by a coronary artery plaque rupture, platelet aggregation at

the site of rupture followed by clot formation resulting in occlusion of a culprit coronary vessel. Primary PCI performed promptly in an experienced center is currently the preferred method of reperfusion of the occluded artery [3]. Management of thrombus during PCI for acute myocardial infarction could include aspiration thrombectomy, however, routine rheolytic thrombectomy has not shown clinical benefit [3].

During the performance of the PCI, occasionally the culprit vessel is found to have massive thrombus burden. The optimal management of massive thrombus burden is not known and the treatment is usually individualized. If the thrombus burden is very large as in the case of a saphenous vein graft occlusion, there is higher rate of acute failure (8.2%) [7]. For saphenous vein grafts, fibrinolytic therapy is no longer used for thrombus-containing lesions, but rheolytic or manual aspiration thrombectomy is sometimes employed [8]. When extensive thrombi are seen in aneurysmal coronary arteries, they have been variously managed. They have been treated with intracoronary thrombolysis followed by heparin and warfarin [9], aspiration thrombectomy followed by prolonged intravenous infusion of a glycoprotein IIb/IIIa inhibitor [10], guide catheter aspiration together with the use of distal protection devices, withdrawal of a distal filter vascular protection device together with adjunct glycoprotein IIb/IIIa inhibitor [11]. Recently in patients with large acute anterior wall STEMI, aspiration thrombectomy was not found to significantly reduce infarct size [12].

In our case, the patient had the coronary angiogram 5 days after the acute event. The risk of thrombectomy of massive thrombus burden outweighed potential benefit especially since the LAD had TIMI grade 3 flow despite the clots. Although there was a potential danger of recurrent occlusion from thrombus propagation, it was felt that medical management would be more prudent. Since the thrombus genesis has both platelet and the coagulation factor components, a triple anticoagulant therapy was chosen. In this part of the world, the use of warfarin and adjusting its dose to a therapeutic INR range is particularly challenging because of inconsistencies in laboratory results and lack of reliability of patient compliance. Hence, in addition to the guidelines recommended therapy of acute myocardial infarction including dual antiplatelet agents of ASA and clopidogrel, we chose to use rivaroxaban after confirming normal renal function. Rivaroxaban is approved for use in nonvalvular atrial fibrillation for the prevention of stroke and systemic embolism, treatment of pulmonary embolism, as well as treatment and prevention of DVT (deep vein thrombosis). In patients with a recent acute coronary syndrome, low-dose rivaroxaban was found to reduce the risk of the composite end point of death from cardiovascular causes, myocardial infarction, or stroke [13]. Because of the massive thrombus, we arbitrarily used the recurrent pulmonary embolism and DVT prophylaxis dose of this newer oral anticoagulant.

Rivaroxaban, because of its potent factor Xa inhibition reduces clotting. Besides its anticlotting activity, in experimental study it was shown to modify fibrin network with thicker fibers and larger pores, which resulted in greater permeation of flow through the clots [14]. Hence, it may facilitate resolution of thrombi.

In our case, the medical regimen used resulted in the resolution of the massive intracoronary thrombus. Incidentally, there was also resolution of left ventricular apical intramural thrombus which has been reported previously with the use of rivaroxaban [15]. Although it cannot be proven, it is plausible that rivaroxaban may have contributed to the resolution of the coronary thrombus through the naturally occurring intrinsic thrombolysis. More data need to be generated to confirm this.

Conclusion

Management of massive intracoronary thrombus burden is challenging despite the availability of various interventional and pharmacologic tools. In our case, a conservative approach of triple therapy with aspirin, clopidogrel, and rivaroxaban produced the desired outcome of resolution of extensive thrombus in the LAD without reocclusion. More studies are needed to address this problematic issue.

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Conflict of Interest

Nothing to report.

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