

Case Report

Acute Coronary Syndrome in a Young Patient with Dual Mechanical Valves and Therapeutic International Normalized Ratio - A Rare Occurrence

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ABSTRACT

Patients of valvular heart disease treated with mechanical valve replacement are required to be on lifelong medication with anticoagulants. Complications may arise when adequate International Normalized Ratio (INR) control is not maintained, leading to thromboembolic events, but very few cases have been reported where these complications arise despite therapeutic INR control. We, here, highlight a case of rare fatal complication of Acute Coronary Syndrome in a one month postoperative patient of Dual Mechanical Valve Replacement with therapeutic INR, which evaded correct diagnosis due to unawareness on the part of the treating team of physicians.

Keywords: Acute Coronary Syndrome; Arrhythmias; International Normalized Ratio; Valvular Heart Disease

Young patients (<60years) with valvular heart diseases, having longer life expectancy, are preferentially treated with valve replacement using mechanical valves as these valves have longer durability and lower need of reoperation, but also require lifelong use of anticoagulant medications [1,2]. The recommended range of International Normalized Ratio (INR) for patients of double valve replacement (DVR) is 2.0-3.0 [3], with the risk of embolism being lowest for INR range of 2.5-4.9 [4]. Patients maintaining a good compliance with their medication have a very low risk of embolism, estimated at 0.4-1.4% patient years [5]. We present an unusual complication of coronary thromboembolism, one month following mechanical valve replacement in a 34 year old male within therapeutic INR, which was diagnosed posthumously. We want to present this case due to the rarity of this fatal complication.

CASE REPORT

A 34 year old male patient presented to the emergency room with the complaints of shortness of breath and palpitations for a few hours duration, one month post-operatively after undergoing a surgery for dual heart valve replacement due to rheumatic heart disease (RHD) with severe mitral Stenosis (MS) and moderate aortic regurgitation (AR).

His vitals on re-admission were normal with SpO₂ of 98% on room air. He was diaphoretic and appeared in distress. His INR was 3.0 (Goal INR was 3-3.5). Electrocardiography showed ventricular tachyarrhythmia and ST-elevation with non-progression of R wave pattern, (**Fig. 1A & 1B**). Echocardiography showed global left ventricular hypokinesis with ejection fraction (EF) of 15%,

with both mechanical valves were functioning satisfactorily, and there was no evidence of pannus or thrombus on either valvular surface and no clots or collections in the pericardium. Coronary angiography was not performed as the patient had therapeutic range of INR ruling out the diagnosis of coronary thromboembolism.

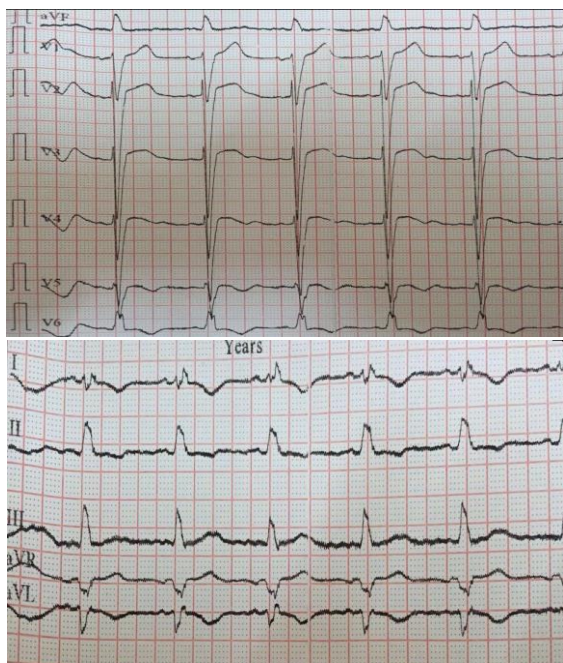


Figure 1A & 1B - ECG of the patient on the day of re-admission to the emergency room, showing ST segment elevation with Ventricular Tachyarrhythmia pattern.

Subsequently, the patient developed runs of ventricular fibrillation (VF), requiring defibrillation and Amiodarone loading with maintenance infusion. When the patient continued to have VF, he was started on Xylocard 2% infusion which led to cessation of the arrhythmia. His symptoms were thought to originate from left ventricular dysfunction, and ECG findings presumed to arise from pericardial changes. He died 48 hours after admission, due to intractable VF, despite all resuscitative measures and without an assertive diagnosis of his presentation.

His previous hospital stay during the valve replacement surgery had been uneventful, and his evaluation had shown no predisposing risk factors for thromboembolism. His pre-operative echocardiography had shown EF of 30%, anterior mitral leaflet was thickened and calcified, and posterior mitral leaflet was calcified and fixed, causing severe MS. Aortic valve (AV) cusps were thickened and retracted causing moderate AR,

and severe left ventricular (LV) systolic dysfunction with dilated LV dimensions of LVID(ed) = 6.3cm and LVID(es) = 5.8cm. He had undergone AVR surgery, during which his mitral valve (MV) was replaced using ON-X (25/33) conform mechanical valve, (internal orifice - 4.1 cm²), preserving the subvalvular apparatus and AV was replaced with 21mm conform ON-X valve, (internal orifice - 2.84cm²). His post-operative echocardiography had shown normally functioning mitral and aortic mechanical prosthesis with mean and peak gradients of 4 and 8 in mitral position, and 8 and 14 in aortic position, respectively. He had been discharged on 5th post operative day with an uneventful postoperative recovery, till he presented again to the emergency department one month later. His medications on discharge had included Metoprolol, Torsemide daily, Perindopril and Acenocumerol (equivalent of warfarin) in adequate doses and compliance was also good.

The post mortem examination showed evidence of coronary thromboembolism involving the left anterior descending (LAD) artery, prompting the treating physicians to the posthumous diagnosis of acute coronary syndrome (ACS), most likely arising from micro-thrombus on the mechanical valves, which he must have developed despite being on oral anticoagulation medication with good compliance and maintaining his goal INR.

DISCUSSION

Thromboembolic complications are not common events in patients with a good anticoagulant control and therapeutic INR. This was a young patient, who presented in his early post operative period, with thromboembolic complications in the coronary artery, which evaded correct diagnosis due to INR values being in the recommended range, and he, also, did not have any risk factors or a positive family history for ischemic heart disease or thromboembolism. Echocardiographic findings on neither of occasions suggested the presence of thrombus or pannus development on the mechanical valvular surfaces. In our extensive search of the published literature, we have not found a similar case presentation in so young patients, having good anticoagulant control and presenting in early post operative period.

A recent trial by Puskas et al has suggested that use of ON-X mechanical valves for AV replacement may be managed with lower doses of anticoagulant medication

which eventually results in significant lower risk of bleeding without affecting the risk of thromboembolism [6]. In a case series reported by Iakobishvili et al, found that the median time from implantation of a prosthetic valve to the development of the ACS was 8 years and that majority of such patients had 2 or more risk factors associated with atherosclerosis [7].

Wongrakpanich et al reported a case of coronary thromboembolism in a 54 year old male patient with dual prosthetic heart valves who was operated upon 20 years ago, and the patient received successful management by coronary angiography and thrombectomy with an eliminate aspiration catheter [8]. In a similar case reported by Sharma et al, a 35 year old female with DVR surgery, done 10 years prior and no coronary risk factors developed ACS. She had discontinued her oral anticoagulation medication three months earlier, but was successfully managed with balloon angioplasty [9].

Fernandez et al reported a case series of 4 patients who had developed massive thrombosis of Aortic Bjork-Shiley prosthesis despite a well maintained anticoagulation therapy [10]. Karakoyun et al reported a series of non-ST elevation ACS in three patients with single prosthetic valve replacement surgery done 5, 3, and 6 years after valve replacement, respectively [11]. All of them were successfully managed with thrombolytic therapy using low dose tissue plasminogen activator (t-PA). Levis et al reported a case of myocardial infarction in a 50 year old patient with tissue AV replacement done four years ago due to coronary artery embolism [12]. He was managed with percutaneous transluminal coronary angioplasty with balloon angioplasty and aspiration thrombectomy.

Conclusion

This case highlights the importance of entertaining a diagnosis of ACS in patient with suggestive findings, despite good anticoagulant control and therapeutic INR and ruling this out by timely and adequate cardiac imaging.

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