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Case Report

Posterior Chordae Rupture of the Tricuspid Valve due to Myocardial Infarction: A Rare Entity Among Tricuspid Valve Pathologies

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Abstract: The tricuspid valve is rarely affected by myocardial infarction. The right ventricle has a lower systolic pressure compared to the left ventricle making the papillary muscles of the tricuspid valve more resistant to ischemia. The right coronary artery is the main blood supply to tricuspid papillary muscles, chordae rupture of the tricuspid valve may occur following myocardial infarction. Here, we present a rare case of a 70-year-old male with severe tricuspid regurgitation, a completely occluded right coronary artery (RCA), 70% stenosis of the left main coronary artery (LMCA), and 100% occlusion of the left anterior descending artery (LAD). The patient's ejection fraction (EF) was reduced to 30%. Treatment included tricuspid valve repair using the bicuspidization technique and a five-vessel coronary artery bypass graft (CABG). This case demonstrates that, although rare, myocardial infarction can involve the tricuspid valve and that the surgery of the valve with full revascularization proved to be an effective treatment strategy in our patient.

Keywords: Myocardial Infarction; Papillary Muscle Rupture; Tricuspid Regurgitation.

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1. Introduction

Papillary muscle rupture is a relatively uncommon, yet frequently fatal complication following myocardial infarction (MI). Most cases involve rupture of the papillary muscles on the left ventricular side of the mitral valve apparatus. In contrast, papillary muscle rupture affecting the tricuspid valve after MI represents an exceedingly rare occurrence [1]. The etiology of ruptured tricuspid papillary muscle is predominantly traumatic, accounting for 62% of cases. Among these, 50% are related to blunt chest trauma, while the remaining 50% result from iatrogenic injury, specifically chordal severing during myocardial biopsy. Non-traumatic causes contribute to the remaining 38% of cases, with myxomatous degeneration accounting for 12%, infective endocarditis for 8%, and congenital anomalies for 3%. Additionally, 15% of patients present with idiopathic ruptures, for which no specific etiology can be identified [2].

We present a rare case of a 70-year-old male patient with severe tricuspid regurgitation due to papillary muscle rupture caused by myocardial infarction with a low ejection fraction of 30%. The uniqueness of our case lies both in the complexity of definitive diagnosis involving tricuspid valve pathologies and in the fact that surgical approach proved to be crucial in definitive diagnosis and treatment. Despite the low ejection fraction, full

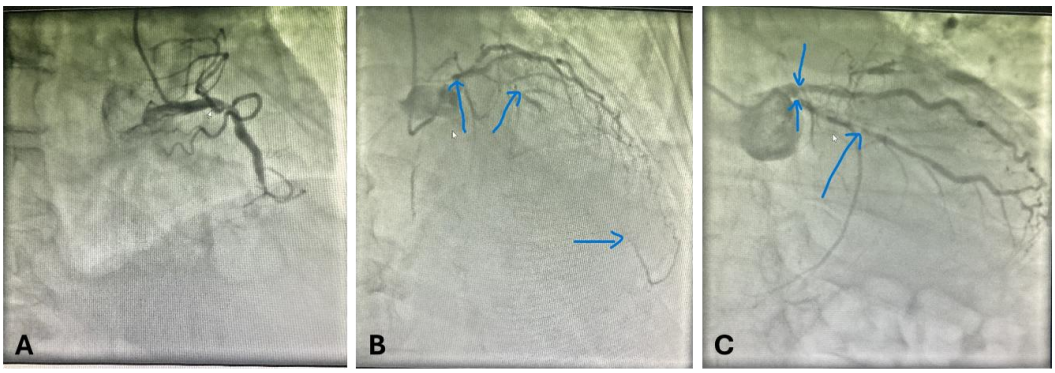
coronary artery revascularization and tricuspid valve repair proved to be effective in our case.

2. Case Report

2.1 Preoperative Findings and Management

A 70-year-old male presented with dyspnea on exertion. He had a history of coronary artery disease, heart failure classified as NYHA Class II, and was on a medical regimen that included nitrates, beta-blockers, diuretics, antihypertensive agents, and antiarrhythmics. The patient had no history of intravenous drug usage. On physical examination, his blood pressure was 109/76 mmHg, heart rate 64 bpm, and oxygen saturation 96% on 2 L/min of nasal oxygen. Coronary angiography revealed total occlusion of the RCA, 70% stenosis of the LMCA, and 99% occlusion of the LAD (Figure 1).

Figure 1. Coronary angiography of the patient showing multivessel disease (A). The blue arrows indicate the stenosis of the main vessels (B and C). Approximately 70% stenosis of the left main coronary artery (LMCA), 100% occlusion of the right coronary artery (RCA), and 99% occlusion of the left anterior descending artery (LAD).



Both transthoracic and transesophageal echocardiography showed severe tricuspid regurgitation (TR) with a flail posterior leaflet. Left atrium was measured 4,3 cm and the right atrium 4,3cm as well. A rupture of the posterior leaflet chordae was suspected, with a 1.1 cm structure consistent with vegetation observed at the tip of the chordae, moving into the right atrium during systole. On imaging no thrombus was seen on both the right and left atrium. The ejection fraction was reduced to 30%. Following the echocardiography report a preliminary diagnosis of infective endocarditis was fulfilled based on the detection of vegetation on transesophageal echocardiography and a positive peripheral blood culture for coagulase-negative Staphylococcus. The patient was started on teicoplanin as per the infective endocarditis protocol.

During preoperative follow up the patient had no fever but developed arrhythmia due to coronary artery disease which was managed by amiodarone. After consultation with the cardiology and infectious disease departments we planned to perform open surgery following patient's multiple coronary artery disease and severe tricuspid regurgitation and judged that a lesser invasive procedure is not appropriate given the patient's condition. The patient was not appropriate for stenting and severe tricuspid regurgitation could worsen patient's right ventricular functions and impact his quality of life. Therefore, no further imaging tools were employed.

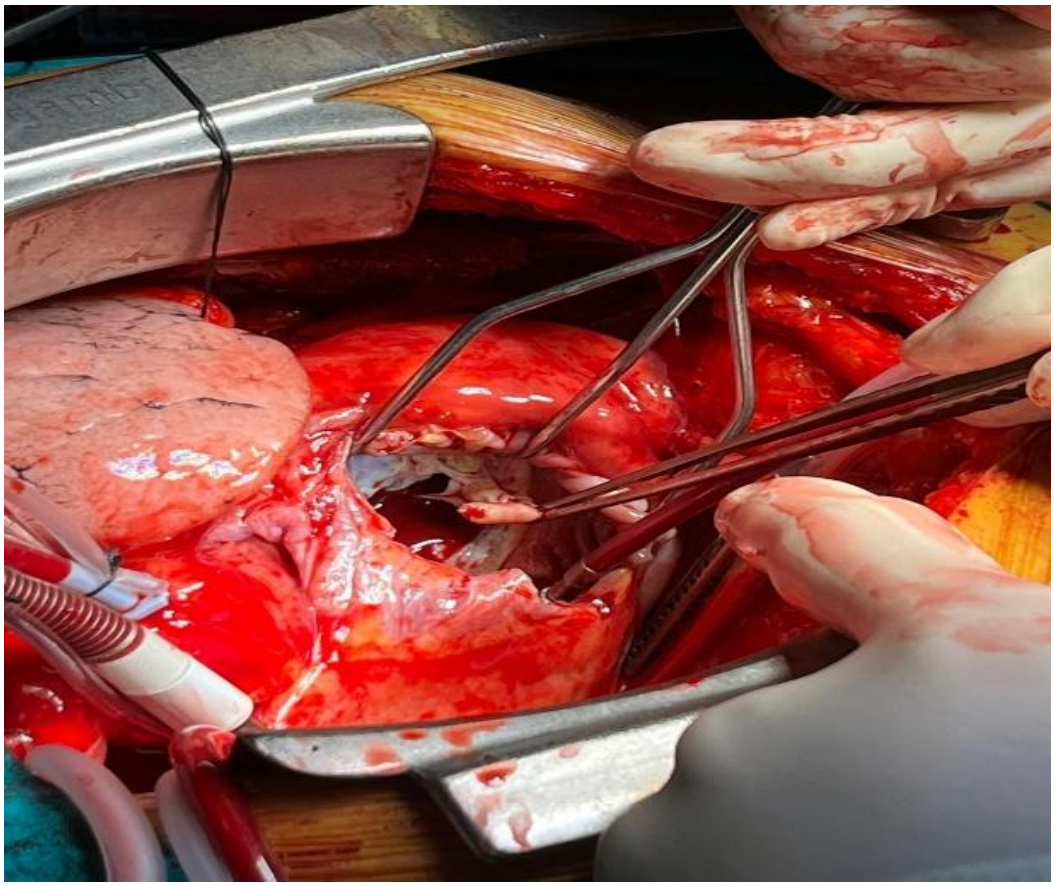
2.2 Operative Technique

We planned to perform a five-vessel coronary artery bypass grafting (CABG) and tricuspid valve repair. Under general anesthesia, a median sternotomy was performed. The aorta and superior vena cava were cannulated, while femoral venous cannulation was used for inferior vena cava (IVC) access to avoid lifting the heart, given the patient's low

ejection fraction (EF), thus reducing the risk of early fibrillation. Standard cardiopulmonary bypass (CPB) was initiated with moderate systemic hypothermia (28–30°C). Intraoperative findings showed no vegetation on the tricuspid valve but a ruptured papillary muscle of the posterior leaflet (Figure 2). Tricuspid valve repair was performed first, under CPB, without aortic cross-clamp placement. Specimens resected during the procedure were sent for both pathology and microbiology examination (Figure 2).

The anterior and septal leaflets of the tricuspid were sutured side-to-side, and De Vega annuloplasty was performed for annular dilatation. The water test confirmed no residual regurgitation. LAD was bypassed using the left internal mammary artery. Sequential aorto-coronary bypass was performed with a saphenous vein graft to the circumflex artery and intermediate branch. A separate saphenous vein graft was used for the first diagonal branch and anastomosed to the proximal saphenous graft of the circumflex artery. RCA was bypassed with a saphenous vein graft to the aorta. The procedure involved 74 minutes of aortic cross-clamping with antegrade hypothermic Custodiol cardioplegia and 274 minutes of CPB.

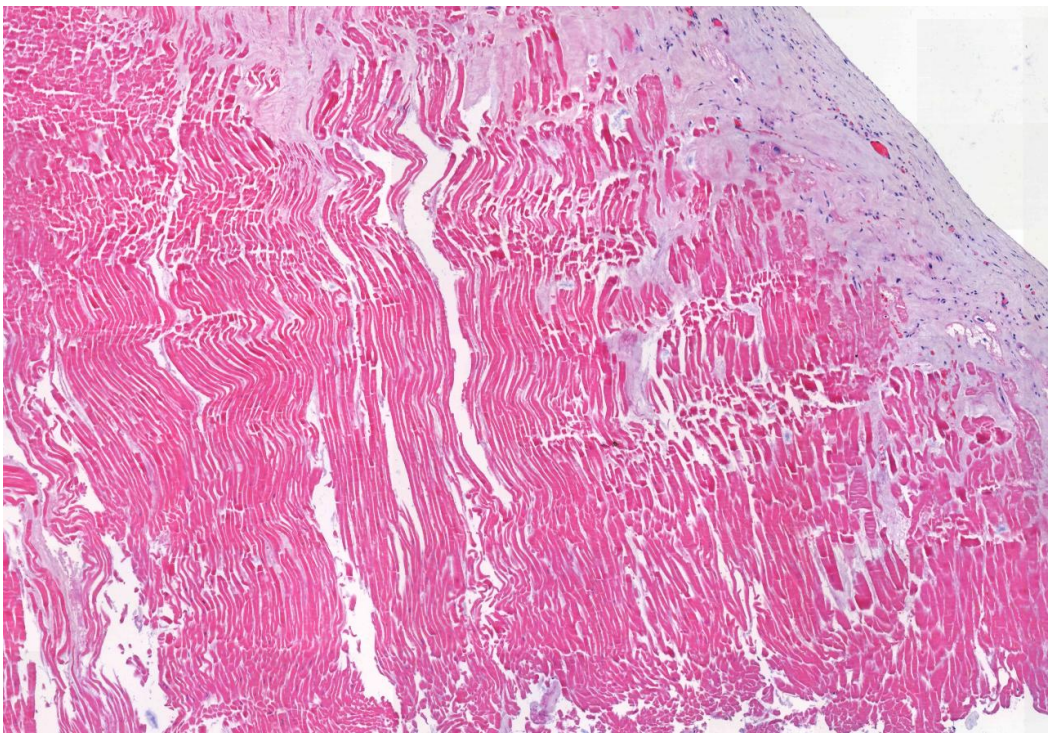
Figure 2. Ruptured papillary muscle of the posterior leaflet (tricuspid valve).



2.3 Postoperative Course

The patient required 3 units of packed red blood cells and 3 units of fresh frozen plasma. Patient's extubating was successfully completed after 18 hours postoperatively. Pathology (Figure 3) results showed myocardial necrosis due to myocardial infarction and microbiology showed no microorganisms. The patient was discharged from the ICU on postoperative day 5. Later, the patient developed pneumonia, which was confirmed by a CT scan. Prolonged intubation and hospital stay alongside heart failure were the main reasons for the development of pneumonia in our patient. Heart failure can be associated with lung edema which is a preoperative risk factor for pneumonia development.

Figure 3. Histopathology of tricuspid valve. Fibrosis and severe myocardial necrosis are seen (Hematoxylin-Eosin, x 100).



Following the pathology results and the intraoperative findings which showed no vegetation on the tricuspid valve, infective endocarditis was excluded, and the antibiotic regimen was adjusted according to infectious diseases protocol due to pneumonia. The patient was discharged from the hospital on day 17 post operatively after finishing antibiotic treatment as per infectious disease protocol with appropriate medical therapy and follow-up instructions. Postoperative echocardiographic findings (Figure 4) included an improvement in ejection fraction to 33%, no gradient on the tricuspid valve, no vegetation and mild regurgitation. Patient presented to the clinic for his second month follow up in good condition. His electrocardiogram was in sinus rhythm and C-reactive protein (CRP) levels were low. His heart failure symptoms were classified as NYHA class1. Patient had an improved ejection fraction with 36% and no gradient on the tricuspid valve with mild regurgitation.

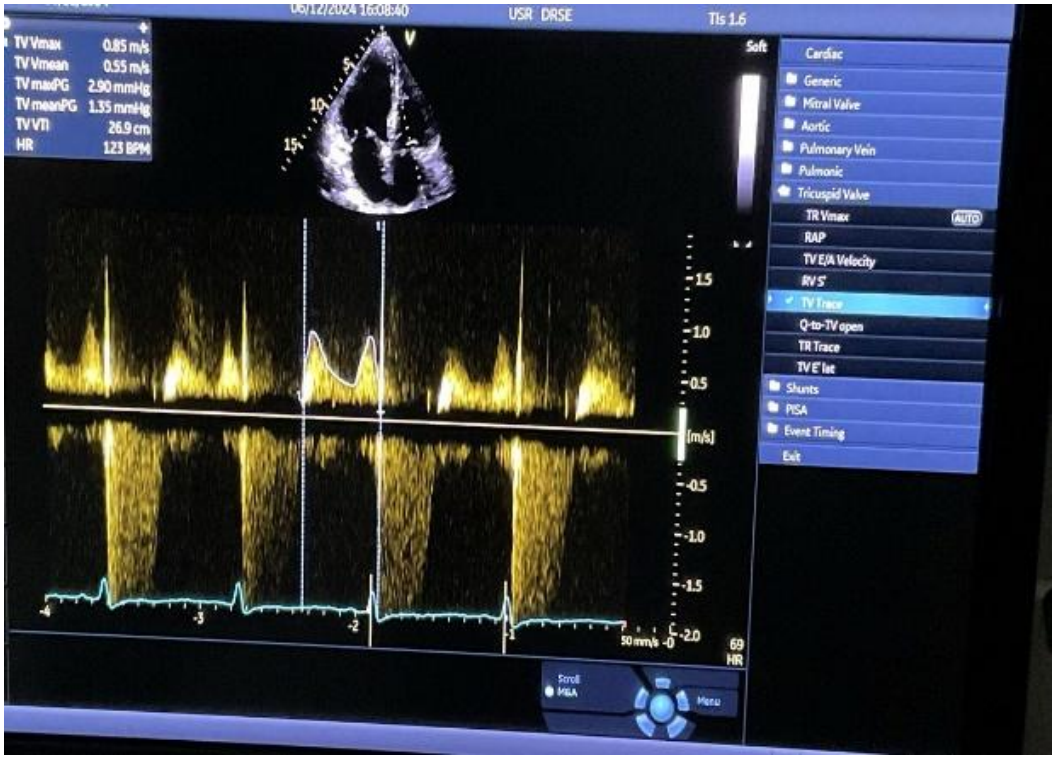
3. Discussion

Eisenberg et al. was the first to report a case of rupture of a tricuspid papillary muscle due to myocardial infarction in the literature [3]. Although papillary muscle rupture is rare, it is potentially a fatal, emergent complication, commonly associated with ischemic etiology [4]. Otherwise, causes related to structural, traumatic or infectious problems such as bacterial endocarditis, have been reported in especially young people between the ages of 20 and 30 years [5]. These conditions predominantly affect the left side of the heart, particularly the mitral valve. However, rupture of a papillary muscle of the tricuspid valve is possible but extremely rare [5]. There are also a few cases describing primary pulmonary hypertension and blunt trauma as causes of this condition [6].

Anatomically, each of the papillary muscles attaches to two adjacent leaflets of the tricuspid valve causing clinically insignificant resultant tricuspid insufficiency unless dilatation of the annulus supervened. The anterior and conus papillary muscles of the tricuspid valve are constant in position, while only the posterior one is inconstant [7]. Tricuspid papillary muscle rupture due to right coronary artery ischemia is rarely observed as in our case [8]. Our patient with a preliminary diagnosis of infective endocarditis was

started on the antibiotic regimen due to echocardiography and peripheral culture findings.

Figure 4. Post-operative echocardiographic findings showed no gradient across the tricuspid valve and mild regurgitation.



Preoperative echocardiography findings may mislead the definitive diagnosis as the papillary muscle rupture may be sometimes misjudged with vegetation so intraoperative findings and pathology had a crucial role in our patient's case. In heart valve surgeries, if suitable, valve repair is preferred over valve replacement. As part of the surgical plan, we managed this case using the bicuspidization technique for the tricuspid valve, which is an appropriate approach when the rupture involves only the posterior chordae. We did not consider replacing the tricuspid valve as the papillary muscle rupture only involved the posterior leaflet without compromising other parts of the tricuspid valve and by doing so, we also avoided warfarin usage for this patient in the future. For heart failure patients with low ejection fraction to minimize the progression of right heart failure not replacing the valve is a better choice as mild regurgitation on the tricuspid could relieve the pressure on the right ventricle.

The coexistence of coronary multivessel disease and reduced ejection fraction (EF) posed significant surgical challenges. However, a combined strategy of five-vessel coronary artery bypass grafting (CABG) and tricuspid valve repair proved effective. Infective endocarditis was ruled out after the pathology result. We recommend continuing antibiotic therapy until the pathology findings are finalized and then readjusting the treatment according to the patient's other comorbidities as in our case that presented with pneumonia.

Postoperatively, the patient had no positive blood cultures, and no vegetation was detected on echocardiography, strongly emphasizing the significance of intraoperative and pathological findings in identifying myocardial infarction as the definitive etiology in this kind of cases. Adjusting antibiotic therapy was important in reducing the patient's length of hospital stay. By surgical approach we better understood how papillary muscle rupture may sometimes be misjudged as vegetation in these kinds of rare cases. Decision

to treat this patient surgically was crucial as any other approach would have compromised his general condition given preoperatively ventricular arrhythmias or would have prolonged his hospital stay for antibiotic treatment as per infective endocarditis protocol according to preliminary diagnosis given first. The multidisciplinary evaluation and management of surgery, pathology, infectious diseases, cardiology and anesthesia departments is mandatory in this kind of cases for accurate diagnosis and successful treatment of the patient.

4. Conclusion

Papillary muscle rupture of the tricuspid valve due to myocardial infarction is a rare entity and should be considered in patients following myocardial infarction, especially in the presence of right coronary artery stenosis. Clinical decision-making is challenging in these types of cases, and in settings where advanced imaging tools are limited, a surgical approach with an experienced cardiac team is essential for the definitive diagnosis and treatment of the patient. Herein, we highlight a successful management of a rare case of papillary muscle rupture of the tricuspid valve and emphasize the importance of this infrequent diagnosis.

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Research Ethics Committee Approval: We declare that the patient approved the study by signing an informed consent form and the study followed the ethical guidelines established by the Declaration of Helsinki.

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Conflicts of Interest: The authors declare no conflicts of interest.

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