Title: Mitral Mayhem: Reperfused but Ruptured

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Introduction: Acute severe mitral regurgitation (MR) is a life-threatening complication following myocardial infarction (MI), commonly resulting from rupture of papillary muscle. The anterolateral papillary muscle (APM) has traditionally been considered to be “protected" because of its dual blood supply from the left anterior descending (LAD) and left circumflex (LCx) coronary arteries. However, in the presence of extensive coronary artery disease, this anatomical protection is not reflected at a functional level, leading to a rare, mechanical catastrophe following non-transmural MI.

Case: A 72-year-old male with history of hepatocellular carcinoma on tyrosine kinase inhibitor therapy (TKI) status post-chemoembolization, hypertension, and hyperlipidemia, who initially presented with complaints of chest pain. He was diagnosed with anterolateral myocardial infarction on ECG, with a troponin level of 2186 ng/dL and a proBNP of 13,000 pg/mL. The patient underwent emergent left heart catheterization, where a drug-eluting stent (DES) was placed in LCx, with residual 100% disease in proximal obtuse marginal 1 (OM1) and 80% disease in LAD. An intra-arotic balloon pump was also placed. LV gram and echocardiography (both transthoracic and transesophageal), revealed severe mitral regurgitation (MR 4+). The patient's hospital course was complicated by acute decompensation and cardiogenic shock, requiring intubation and initiation of inotropes.

Due to his prohibitive surgical risk, mitral transcatheter edge-to-edge repair (M-TEER) was performed, where 3 clips were placed. Post-clip, MR was mild, with a mean gradient of 4 mmHg. The patient then underwent successful revascularization with a DES to proximal and distal LAD. Next day, the patient decompensated and was found to have severe MR with APM rupture. Due to his poor prognosis, the family opted for a palliative extubation, and he passed away.

Discussion: This case points out the limitations of M-TEER as a rescue procedure in the context of acute PMR. While it provides immediate stabilization of cardiovascular status, it does not correct the biological process of infarct progression. The use of TKI may have also contributed to an increased risk of complications. TKIs have been shown to interfere with vascular endothelial growth factor (VEGF) signaling and have a negative effect on angiogenesis and collagen synthesis, which are important for wound healing after an MI. The infarcted muscle head is in a prolonged 'softening' phase and is not a reliable site for mechanical clip attachment.

The data from the 2025 IREMMI registry confirm our observations and demonstrate a significantly increased risk of in-hospital mortality (adjusted odds ratio 3.05) and a 22% risk of

conversion to surgery in patients undergoing TEER for PMR compared to those undergoing surgery for secondary functional MR. M-TEER is a 'bridge to stability' and not a 'cure' for PMR. High levels of vigilance are required for mechanical clip failure in the presence of multivessel disease and concomitant oncological treatments that impair the integrity of the infarcted myocardium.

Conclusion: APM rupture is a lethal complication that should be considered in cases with multivessel disease because dual blood supply does not automatically imply protection against global ischemic states. Although the application of M-TEER is a lifeline to the patient with inoperable cases, it is important to note that it is not the cure for the patient. It is important to consider the effects of systemic oncology therapies on the integrity of the cardiac structure because these therapies can affect the success of mechanical repair by inhibiting the healing process.

References:

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