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Revascularization Renaissance: A Case Series of conservative triumph over Ischaemic Mitral Catastrophe in Acute Myocardial Infarction

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Abstract

Acute severe ischaemic mitral regurgitation (MR) following acute myocardial infarction (AMI) is an uncommon but life-threatening complication associated with high mortality. Although surgical mitral valve intervention and transcatheter edge-to-edge repair (TEER) represent established management strategies, surgery in the acute MI setting carries substantial procedural risk, and TEER may be limited by cost and availability. In selected patients in whom MR is attributable to papillary muscle dysfunction and hemodynamics are borderline but potentially reversible, prompt coronary reperfusion with intensive medical therapy may offer a viable non-surgical alternative. We describe a case series of eight patients aged 43–73 years who presented with AMI (anterior, inferior, or lateral wall involvement) complicated by moderate-to-severe ischaemic MR, with left ventricular ejection fraction ranging from 28% to 48%. All patients underwent coronary revascularisation by percutaneous coronary intervention (PCI) or thrombolysis followed by facilitated PCI, along with aggressive medical management, including antiplatelet agents, anticoagulation, inotropes, and guideline-directed anti–heart failure therapy. None of the patients required surgical mitral valve intervention or TEER. At 6-month follow-up, all patients were haemodynamically stable and asymptomatic, with regression or stability of MR severity. This series suggests that timely revascularisation combined with optimal medical therapy may ameliorate severe ischaemic MR secondary to papillary muscle dysfunction in carefully selected AMI patients, potentially avoiding high-risk interventions; prospective studies are needed to refine selection criteria and validate outcomes.

Keywords: Ischemic mitral regurgitation, Acute myocardial infarction, Coronary revascularization, Medical management

Introduction

Severe mitral insufficiency is among the most feared mechanical complications after acute myocardial infarction (AMI) because it can precipitate abrupt pulmonary oedema, cardiogenic shock, and multi-organ hypoperfusion. Although echocardiographic evidence of ischaemic mitral regurgitation (MR) after acute coronary syndromes is relatively common, clinically significant (moderate–severe) MR is reported in a smaller but important subset, variably estimated at 3–12% across cohorts and definitions.(1-3) The pathophysiology of acute ischaemic MR spans a continuum from "organic" leaflet malcoaptation due to papillary muscle rupture (PMR) to "functional" regurgitation driven by post-infarction ventricular geometry. Rupture—typically partial and most often involving the posteromedial papillary muscle because of its single coronary blood supply—is rare in the reperfusion era (roughly 0.05–0.26%

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after MI) but carries a fulminant clinical course.(4) In contrast, papillary muscle ischaemia/dysfunction and left ventricular (LV) remodelling can displace papillary muscles apically and laterally, enlarge the annulus, and tether the mitral leaflets, producing regurgitation even when the valve apparatus is structurally intact.(4, 5)

Outcomes remain poor when acute severe ischaemic MR is treated with medical therapy alone. Contemporary reviews of mechanical post-MI complications highlight very high short-term mortality with uncorrected defects, and case series of PMR consistently show that conservative management is associated with rapid deterioration and death.(6-8) Urgent surgery (repair or, more commonly in PMR, replacement) is therefore considered definitive therapy, but operative mortality remains substantial and is strongly influenced by haemodynamic status (shock, need for mechanical support), infarct size, and comorbidity burden, with reported early mortality commonly in the 20% range in meta-analyses and large series and higher in extreme-risk presentations.(9, 10)

In parallel, transcatheter edge-to-edge repair (TEER) using MitraClip has expanded the armamentarium for patients deemed prohibitive surgical candidates, with registry and observational data supporting feasibility as a stabilising strategy in acute MR following MI, including in cardiogenic shock settings.(11, 12) Nevertheless, an important clinical grey zone persists: patients with severe MR predominantly from papillary muscle dysfunction (rather than frank rupture) and "borderline" but potentially reversible hemodynamics. In this subgroup, rapid reperfusion and infarct-limiting therapy can improve regional wall motion, reduce tethering forces, and lessen regurgitation severity, particularly when MR is dynamic and worsens with ongoing ischaemia.(13) Mechanistically, functional ischaemic MR is increasingly understood as a ventricular disease rather than a primary valvular disorder. Asymmetric LV remodelling after MI alters annular shape and papillary muscle position, producing leaflet malcoaptation through tethering; the degree of MR may therefore fluctuate with loading conditions and ischaemia severity.(5, 14) Early, successful reperfusion—especially primary percutaneous coronary intervention (PCI)—may prevent MR progression or promote regression by limiting infarct expansion and restoring papillary muscle synchrony.(15) Similarly, when severe MR occurs episodically during active ischaemia and improves after revascularisation and haemodynamic optimisation, a non-surgical strategy may be sufficient in carefully selected patients.(16)

Against this background, the present case series describes eight patients with moderate to severe ischaemic MR complicating AMI who were managed with timely coronary revascularisation and intensive medical therapy alone, without surgical correction or TEER. Our experience supports the concept that, when MR is driven predominantly by papillary muscle dysfunction and functional tethering (rather than mechanical rupture) and when hemodynamics can be stabilised, rigorous patient selection may identify candidates for conservative management, potentially avoiding high-risk valve intervention in the acute MI setting.

Case Series

Case 1: A 55-year-old man with diabetes mellitus and hypertension presented to the emergency department with acute-onset chest pain and breathlessness of one day's duration; examination showed hypoxemia with hemodynamic compromise. Electrocardiography demonstrated features of an evolved lateral wall myocardial infarction. Two-dimensional transthoracic echocardiography revealed a lateral wall regional wall motion abnormality with a left ventricular ejection fraction of 40% and severe mitral regurgitation with an eccentric jet (**Video**

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1). He was admitted to the coronary care unit and started on high-flow oxygen, dual antiplatelet therapy, unfractionated heparin, and aggressive anti–heart failure measures including diuretics, nitrates, and beta-blockers, while diabetic ketosis was managed concurrently with an insulin infusion. Coronary angiography showed single-vessel disease with total occlusion of the major obtuse marginal branch and a co-dominant left circumflex artery with significant stenosis involving its major obtuse marginal branch. The patient underwent successful primary percutaneous coronary intervention with stent implantation to the major obtuse marginal branch, achieving TIMI III flow. After revascularization, optimal medical therapy was continued with dual antiplatelets, beta-blockers, ACE inhibitors, statins, and diuretics as required. Over the ensuing days, he showed progressive hemodynamic recovery and no longer required oxygen support; repeat echocardiography demonstrated stable mitral regurgitation with improved left ventricular function. He was discharged on medical therapy and remained hemodynamically stable and asymptomatic at 15-day and 6-month follow-up.

Case 2: A 46-year-old man with diabetes mellitus and a chronic smoking history presented to the emergency department with acute-onset chest pain of 4–5 hours' duration, with markedly elevated and poorly controlled blood glucose at presentation. Electrocardiography demonstrated acute ST-segment elevation in the anterior leads, consistent with an extensive anterior wall myocardial infarction. Two-dimensional echocardiography showed regional wall motion abnormality with severe left ventricular systolic dysfunction (LVEF 28%), moderately severe eccentric mitral regurgitation **(Figure 1a)**, Grade III left ventricular diastolic dysfunction, and an 8-mm pericardial effusion adjacent to the right atrium (**Video 2**). He was hypoxemic but normotensive on arrival; owing to financial constraints, he received thrombolysis with streptokinase. After thrombolysis, symptoms improved, but hypoxemia persisted, necessitating supplemental oxygen. He was managed with dual antiplatelet therapy, anticoagulation with unfractionated heparin, aggressive diuresis, and inotropic support as required, while glycaemic control was achieved using an insulin infusion. Coronary angiography performed 24 hours after thrombolysis revealed triple-vessel coronary artery disease with 80% stenosis of the mid-left anterior descending artery and mid-left circumflex artery. Urgent percutaneous coronary intervention with stent implantation was undertaken to both the left anterior descending and left circumflex arteries, achieving TIMI III flow in both vessels. Following successful revascularization, optimal medical therapy was continued; his oxygen requirement gradually reduced and was eventually discontinued, with progressive improvement in haemodynamic parameters. He was discharged on optimal medical management and remained hemodynamically stable, in functional class II at 6-month follow-up, with moderate mitral regurgitation.

Case 3: A 59-year-old man with hypertension presented to the emergency department with acute-onset chest pain of 8 hours' duration, with elevated blood pressure at presentation. Electrocardiography showed features of an evolved inferior wall myocardial infarction. Two-dimensional echocardiography demonstrated hypokinesis of the inferoseptum and basal inferior wall with a left ventricular ejection fraction of 48% and moderately severe mitral regurgitation (**Video 3**). He was admitted and underwent urgent coronary angiography, which revealed total occlusion of the mid-right coronary artery and, in addition, an ectatic proximal left anterior descending artery with a saccular coronary aneurysm and a focal 70% stenosis in the mid-left anterior descending artery. Percutaneous coronary intervention with stent deployment was performed to the mid and distal right coronary artery, achieving TIMI III flow with complete resolution of stenosis. After successful revascularization, he was maintained on optimal medical therapy including dual antiplatelets, beta-blockers, ACE inhibitors, statins, and diuretics, and no complications necessitating surgical intervention occurred. Following

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haemodynamic stabilization, he was discharged on medical management and remained hemodynamically stable and asymptomatic at 15-day and 6-month follow-up, with stable or improved mitral regurgitation severity.

Case 4: A 53-year-old man with a chronic smoking history presented with acute-onset chest pain and breathlessness of one day's duration. Electrocardiography showed an acute anterior wall myocardial infarction. Echocardiography revealed a regional wall motion abnormality with moderate left ventricular systolic dysfunction (EJEF 35%) (**Video 4**), moderate-to-severe mitral regurgitation, and a left ventricular apical thrombus (**Figure 1b**). He was hypoxemic and required supplemental oxygen, although blood pressure remained relatively stable. Owing to financial constraints, thrombolysis with streptokinase was administered; symptoms improved thereafter, but hypoxemia persisted, necessitating continued oxygen support. He was maintained on dual antiplatelet therapy, anticoagulation with unfractionated heparin followed by enoxaparin, and comprehensive anti–heart failure treatment. Coronary angiography demonstrated 95% stenosis of the proximal-to-mid left anterior descending artery. Percutaneous coronary intervention with stent implantation to the left anterior descending artery was performed, achieving TIMI III flow. With revascularization and aggressive medical therapy, he showed progressive haemodynamic recovery, gradual resolution of hypoxemia, and eventual discontinuation of oxygen support. He was discharged on medical management and remained hemodynamically stable and asymptomatic at 15-day and 6-month follow-up evaluations.

Case 5: A 66-year-old man with hypertension, chronic obstructive pulmonary disease, and a history of smoking presented to the emergency department with acute-onset chest pain of one day's duration. Electrocardiography showed features of an evolved inferior wall myocardial infarction. Two-dimensional echocardiography demonstrated hypokinesis of the basal and mid inferoseptum, inferior, and inferolateral walls with a left ventricular ejection fraction of 48% and moderate-to-severe mitral regurgitation (**Figure 1c, Video 5**). He was admitted immediately and underwent urgent coronary angiography, which revealed 90% stenosis of the mid-right coronary artery. Percutaneous coronary intervention with stent implantation to the right coronary artery was performed, achieving TIMI III flow. After revascularization, he was continued on optimal medical therapy with dual antiplatelets, beta-blockers, ACE inhibitors, statins, and diuretics as required, with no complications necessitating surgical intervention. Following haemodynamic stabilization, he was discharged on medical management and remained hemodynamically stable and asymptomatic at 15-day and 6-month follow-up.

Case 6: A 43-year-old obese man with suspected obstructive sleep apnea and a 3-year history of gastroesophageal reflux disease presented with acute-onset chest pain for two days. Electrocardiography showed an evolved anterior wall myocardial infarction. Two-dimensional echocardiography demonstrated hypokinesis of the distal septum, apex, anterior wall, apical anterolateral wall, basal and mid inferoseptum, and basal inferior wall, with a left ventricular ejection fraction of 45% and moderate-to-severe mitral regurgitation (**Video 6**). He was admitted to the coronary care unit and underwent urgent coronary angiography, which revealed total occlusion of the mid left anterior descending artery. Percutaneous transluminal coronary angioplasty (POBA) without stent implantation was performed to the left anterior descending artery, achieving TIMI II flow. After revascularization, he was maintained on optimal medical therapy, and no complications necessitating surgical intervention occurred. Following haemodynamic stabilization, he was discharged on medical management and remained hemodynamically stable and asymptomatic at 15-day and 6-month follow-up evaluations.

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Case 7: A 51-year-old woman with hypertension and diabetes mellitus presented to the emergency department with acute-onset chest pain of two days' duration, with poorly controlled blood glucose at presentation. Electrocardiography demonstrated an evolved inferior wall myocardial infarction. Two-dimensional echocardiography revealed hypokinesis of the basal and mid inferoseptum, inferior, and inferoposterior walls with a left ventricular ejection fraction of 36% and moderate-to-severe mitral regurgitation (**Video 7**). She was admitted immediately and underwent urgent coronary angiography, which showed tandem proximal left anterior descending artery lesions with a maximum 90% stenosis, 80% stenosis of the major obtuse marginal branch, and a mid-right coronary artery with tight stenosis and heavy thrombus burden extending into the mid and distal segments. Percutaneous coronary intervention with stent implantation was performed to the right coronary artery, left anterior descending artery, and major obtuse marginal branch, achieving TIMI III flow in all treated vessels. After successful multivessel revascularization, she was continued on optimal medical therapy including dual antiplatelets, beta-blockers, ACE inhibitors, statins, diuretics, and insulin for glycaemic control; no complications requiring surgical intervention occurred. Following haemodynamic stabilization, she was discharged on medical management and remained hemodynamically stable and asymptomatic at 15-day and 6-month follow-up evaluations, with stable mitral regurgitation.

Case 8: A 73-year-old man with diabetes mellitus presented to the emergency department with acute-onset breathlessness for two days; on admission, hypertension was newly documented and blood glucose was poorly controlled. Electrocardiography showed ST-segment depression in leads V2 to V5. Transthoracic echocardiography demonstrated regional wall motion abnormality with a left ventricular ejection fraction of 40%, moderate mitral regurgitation, a dilated left ventricle, and mild pulmonary arterial hypertension with a pulmonary artery systolic pressure of 38 mmHg (**Video 8**). Cardiac biomarkers were markedly elevated, confirming a non–ST-elevation myocardial infarction. He presented in decompensated heart failure requiring high-flow oxygen support and was admitted to the coronary care unit, where he was started on diuretics, nitrates, dual antiplatelet therapy, statins, and insulin. Coronary angiography revealed total occlusion of the major obtuse marginal branch with only mild disease in the left anterior descending and right coronary arteries. Urgent percutaneous coronary intervention with stent implantation to the major obtuse marginal branch was performed, achieving TIMI III flow with complete resolution of stenosis. After successful revascularization, he showed prompt haemodynamic stabilization with resolution of oxygen requirement; repeat electrocardiography demonstrated resolution of ST depression, and follow-up echocardiography showed regression of mitral regurgitation to a mild degree. He was discharged on optimal medical therapy and remained hemodynamically stable and asymptomatic at 15-day and 6-month follow-up, with only mild residual mitral regurgitation.

Discussion

Severe ischaemic mitral regurgitation (IMR) complicating acute myocardial infarction remains a time-critical clinical problem because it can abruptly convert a coronary event into refractory pulmonary oedema, cardiogenic shock, and multi-organ hypoperfusion. In contemporary practice, clinically important IMR is less common than mild MR detected incidentally on early echocardiography, yet it continues to account for disproportionate morbidity, prolonged critical care use, and high early mortality. The syndrome is heterogeneous, and its behaviour depends on whether the dominant mechanism is structural failure of the subvalvular apparatus or functional malcoaptation driven by ventricular disease. Conceptually, the pathophysiology is

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usually framed around two primary mechanisms: (i) mechanical disruption of the mitral apparatus—classically papillary muscle rupture (PMR)—and (ii) functional regurgitation secondary to myocardial ischaemia, papillary muscle dysfunction, and adverse left ventricular (LV) remodelling with leaflet tethering.(13, 17)

Complete PMR is the most dramatic end of this spectrum. It typically occurs in the first week after infarction—often cited around 2–5 days—when infarcted tissue undergoes softening and is vulnerable to tearing, and it produces sudden severe MR with rapid rises in left atrial pressure, fulminant pulmonary oedema, and shock. Without prompt haemodynamic stabilisation and definitive correction, outcomes are poor, and historical and contemporary reviews consistently emphasise extremely high mortality with purely conservative management in this scenario.(5, 18) Importantly, however, complete rupture is uncommon in the reperfusion era; more frequently, clinicians encounter partial rupture or, even more often, functional IMR in which the valve leaflets are intrinsically normal but fail to coapt because the ventricle and papillary muscles have been geometrically displaced.(13)

Functional IMR after AMI is not simply a "papillary muscle problem." Although papillary muscle infarction and stunning can contribute to acute MR, the regurgitant orifice is typically created by a constellation of LV changes: regional systolic dysfunction with dyssynchrony, asymmetric LV remodelling, annular dilatation (especially posterior), and papillary muscle displacement that tethers the leaflets apically and laterally, restricting closure.(19, 20) This ventricular–valvular interaction explains why MR severity can fluctuate with loading conditions and the ischaemic burden: increases in afterload, tachycardia, transient ischaemia, or worsening congestion can intensify tethering forces and enlarge the effective regurgitant orifice, while haemodynamic optimisation may reduce it. It also explains why two patients with similar papillary muscle involvement can exhibit very different MR grades depending on LV size, regional remodelling pattern, and annular geometry.

These mechanistic distinctions matter because prognosis and optimal therapy differ. In PMR, the natural history is catastrophic and definitive intervention is generally required, often alongside temporary mechanical circulatory support in shock. In functional IMR, by contrast, the regurgitation may partially reflect reversible myocardial stunning and acute remodelling that can improve if perfusion is restored and remodelling is attenuated. Experimental and clinical observations support the idea that even modest acute changes in regional contractility or LV geometry can generate clinically significant MR, and that the regurgitation can improve substantially once perfusion is re-established and progressive remodelling is prevented.(21) This pathophysiological "reversibility window" underpins the conservative strategy highlighted by the present series. The first pillar of such an approach is early reperfusion. Primary percutaneous coronary intervention (PCI) has been associated with a lower incidence of IMR after ST-elevation MI compared with thrombolysis alone, plausibly because prompt and durable reperfusion limits infarct expansion, preserves regional function, and reduces geometric distortion.(15, 22) Beyond incidence, restoration of coronary flow can also reduce MR severity in patients whose regurgitation is driven by ongoing ischaemia or stunning. Revascularisation reduces ongoing necrosis and mitigates progressive LV dilatation, which in turn lessens papillary muscle displacement and leaflet tethering; this infarct-limiting effect is central to the rationale for prioritising coronary correction when the primary lesion is amenable to intervention.(13, 17) The second pillar is optimal medical therapy directed at both hemodynamics and remodelling. In the acute phase, stabilisation often requires diuretics and vasodilators to reduce pulmonary congestion and afterload, and inotropes when low output predominates. Once blood pressure and perfusion allow, guideline-directed therapies—particularly beta-blockers and agents that modulate the renin–angiotensin–aldosterone

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system—can reduce wall stress, favourably influence LV remodelling, and indirectly improve leaflet coaptation by limiting LV and annular dilatation.(23) These effects are clinically relevant because, in functional IMR, the regurgitant orifice is a dynamic expression of LV geometry rather than a fixed structural defect. A particularly important nuance is that some patients experience episodic MR during active ischaemia, with improvement after reperfusion; recognising this phenotype requires careful, repeated assessment of the haemodynamic and echocardiographic response to revascularisation and medical optimisation.(21)

Against these potential benefits must be weighed the risks and limitations of valve-directed intervention in the acute MI setting. Surgical series have long reported wide ranges of operative mortality, reflecting heterogeneity in case mix, urgency, shock status, and institutional expertise; even in contemporary cohorts, early mortality remains substantial, particularly for unstable patients or those requiring mechanical support.(24) In ischaemic MR, repair is theoretically attractive, yet durability concerns and the underlying ventricular pathology have led many centres to favour chordal-sparing replacement in selected acute presentations, with ongoing debate over optimal surgical strategy.(25) TEER with MitraClip has emerged as an important alternative for prohibitive surgical risk, including in post-AMI acute MR; however, procedural complexity, access and cost constraints, and anatomical limitations in distorted functional MR (marked tethering, large coaptation gaps, unfavourable leaflet angles) can restrict applicability and effectiveness in some patients.(26)

Within this landscape, our case series highlights a clinically meaningful subgroup: patients with severe-to-moderate IMR predominantly due to papillary muscle dysfunction and functional tethering rather than frank rupture, and with near-stable hemodynamics (even if oxygen support is required). In such patients, a strategy centred on timely coronary revascularisation and meticulous medical optimisation may allow MR to regress or stabilise without immediate surgical or transcatheter valve intervention. This approach does not argue against surgery or TEER when indicated; rather, it emphasises that the “default” assumption of irreversibility may not hold in all post-AMI MR, and that careful selection—integrating mechanism on echocardiography, response to reperfusion, and trajectory of hemodynamics—may identify candidates in whom avoiding high-risk procedures is both feasible and beneficial.

Several limitations must temper interpretation. The cohort size is small, MR severity grading is partly qualitative, and quantitative measures (e.g., vena contracta, regurgitant volume/effective regurgitant orifice area) were not uniformly available; moreover, there is no matched control group treated with early surgery or TEER, and the experience reflects a single centre, limiting generalisability. Nevertheless, the consistent clinical stabilisation and favourable mid-term follow-up in this mechanistically defined group support the need for prospective studies that compare conservative, valve-sparing pathways with early intervention strategies, and that refine selection criteria—particularly the echocardiographic and haemodynamic markers that predict reversibility after revascularisation and medical therapy.

Conclusion

This case series indicates that severe-to-moderate ischaemic mitral regurgitation attributable to papillary muscle dysfunction in the setting of acute myocardial infarction can improve with timely coronary revascularisation and optimal medical therapy alone, without the need for surgical correction or percutaneous transcatheter intervention, and that, in carefully selected patients with near-stable hemodynamics, such a conservative strategy may achieve clinical improvement and haemodynamic stabilisation while potentially avoiding the substantial morbidity and mortality associated with high-risk procedures. This approach is not universally

applicable, and rigorous patient selection remains critical, as patients with mechanical complications such as complete papillary muscle rupture, acute severe haemodynamic collapse, or cardiogenic shock that does not respond to revascularisation and medical optimisation should still be prioritised for urgent surgical or transcatheter intervention. Prospective studies using standardised MR severity quantification and systematic comparisons of outcomes across treatment pathways are needed to better define the role of conservative management in this complex population.

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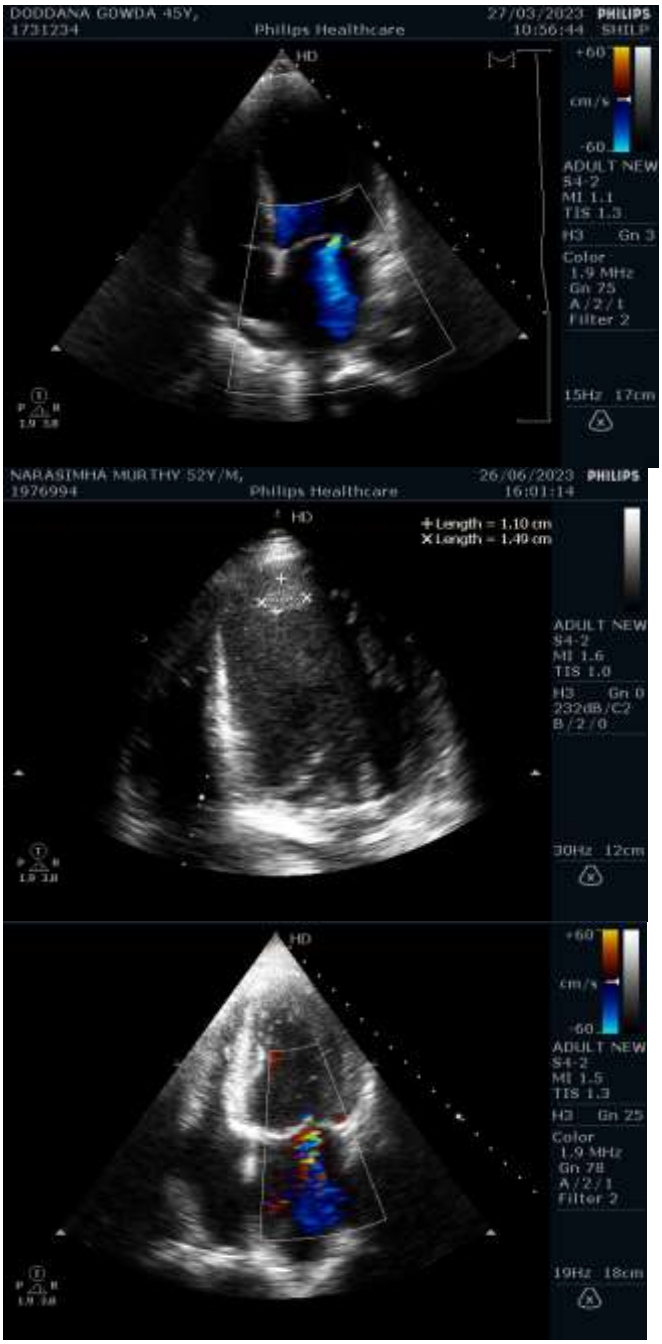

Apical four-chamber colour Doppler echocardiographic view demonstrating systolic jet of mitral regurgitation directed posteriorly from the mitral valve into the left atrium in a 45-year-old male patient

Apical four-chamber echocardiographic view showing left ventricular apical thrombus measuring 1.10 × 1.49 cm in a 52-year-old male patient

Apical four-chamber colour Doppler echocardiographic view demonstrating severe mitral regurgitation with systolic turbulent jet directed laterally into the left atrium

Figure 1: Apical four-chamber colour Doppler echocardiographic view

Attachments (https://drive.google.com/drive/folders/1498-rdyufnBzrFzQAMv5HBuVRsx1W0U1?usp=drive_link)

Video 1: Apical four-chamber colour Doppler 2D echocardiographic cine-loop demonstrating moderate mitral regurgitation with systolic colour jet into the left atrium

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Video 2: Apical four-chamber colour Doppler echocardiographic cine-loop demonstrating moderately severe mitral regurgitation with systolic turbulent jet (yellow-red mosaic pattern) directed into the left atrium

Video 3: Apical four-chamber colour Doppler echocardiographic cine-loop illustrating severe mitral regurgitation with high-velocity systolic jet filling the left atrium

Video 4: Apical four-chamber colour Doppler echocardiographic cine-loop showing prominent mitral regurgitation jet originating from the mitral valve during systole

Video 5: Apical four-chamber colour Doppler echocardiographic cine-loop depicting moderate to severe mitral regurgitation with eccentric systolic jet and vena contracta visualization

Video 6: Apical four-chamber colour Doppler echocardiographic cine-loop revealing moderate-to-severe mitral regurgitation across multiple cardiac cycles

Video 7: Apical four-chamber colour Doppler echocardiographic cine-loop highlighting moderate to severe mitral regurgitation jet with proximal isovelocity surface area (PISA) formation

Video 8: Apical four-chamber colour Doppler echocardiographic cine-loop demonstrating moderate mitral regurgitation with dense systolic mosaic jet extending into the left atrium