

Cardiogenic Shock Secondary to Rupture of the Papillary Muscle.

Acute Presentation and Challenges with VA ECMO Management.

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INTRODUCTION

- Papillary muscle rupture (PMR) is a rare but life-threatening complication of myocardial infarction or infective endocarditis, leading to severe mitral regurgitation, cardiogenic shock, and pulmonary edema.
- The rupture causes abrupt valvular incompetence and backward flow, resulting in acute heart failure.
- PMR occurs in only 0.07%–0.26% of post-MI cases but contributes to ~5% of MI-related mortality.
- Partial ruptures cause milder regurgitation, whereas complete ruptures—typically within the first week post-MI—lead to rapid clinical collapse.
- Posteromedial PMR is 6–12× more common due to its single-vessel supply from the posterior descending artery.
- Most cases arise from small infarcts with poor collateral flow, where preserved contractility increases shear stress on the weakened papillary muscle.
- VA-ECMO provides rapid cardiopulmonary support, reducing LV load and improving perfusion, and serves as a bridge to surgery; CVVHD can be added for optimized fluid and metabolic control.

CASE PRESENTATION

- 56-year-old male with atrial fibrillation, hypertension, and migraines presented with acute altered mental status after sudden decline marked by vomiting, weakness, and confusion.
- In the ED, he was hypotensive and hypoxic with rising troponins. An EKG was suggestive of an inferior STEMI with RV involvement; he developed acute respiratory failure requiring intubation and cardiogenic shock requiring multiple pressors, with severe acidosis and leukocytosis.
- Bedside TTE showed severe MR with possible vegetation. CT surgery consulted, shock team called and recommending ECMO support. Pt taken urgently to cath lab for TEE and ECMO. LHC showed 70% dRCA/ostial RPDA disease w/o PCI.
- Patient was cannulated for VA-ECMO and intraoperative TEE confirmed posteromedial papillary muscle rupture with torrential MR.
- He was transferred to the CTICU on vasoactive support and later underwent mitral valve replacement, CABG ×2, and CFA/SFA repair, with ECMO subsequently moved to the right axilla (due to North-South syndrome) and CVVHD initiated for renal failure.
- Postoperatively, he was managed on heparin, aspirin, and amiodarone and eventually weaned off milrinone and levophed.
- CT head later showed multiple bilateral embolic strokes with poor prognosis; neurosurgery recommended no intervention, and the family elected comfort care and terminal extubation.

VA ECMO/CABG/MVR

INTRAOPERATIVE EVENTS

- **OR Visit I:**
 - **Anesthesia:** Intubated in situ; uncomplicated induction; given midazolam and maintained low-dose propofol.
 - **Hemodynamics:** Vasopressin and norepinephrine titrated; vasopressin discontinued, stable on norepinephrine alone.
 - **Procedure:** VA-ECMO successfully placed; no intraoperative events; patient remained intubated postoperatively.
- **OR Visit II:**
 - **Induction & Maintenance:** Underwent MVR and CABG for North–South syndrome; induced with midazolam, fentanyl, rocuronium; maintained on isoflurane then propofol.
 - **Intraoperative Course:** On norepinephrine and epinephrine; severe biventricular dysfunction from cardiogenic shock; on CPB (3 hr 2 min, cross-clamp 1 hr 26 min).
 - **Outcome:** Difficulty weaning due to myocardial stunning; ECMO initiated with aortic cannula moved to right axilla; surgery completed successfully.

INTRAOPERATIVE IMAGES



Figure 1: Image above shows mid esophageal 4 chamber view. Papillary muscle rupture

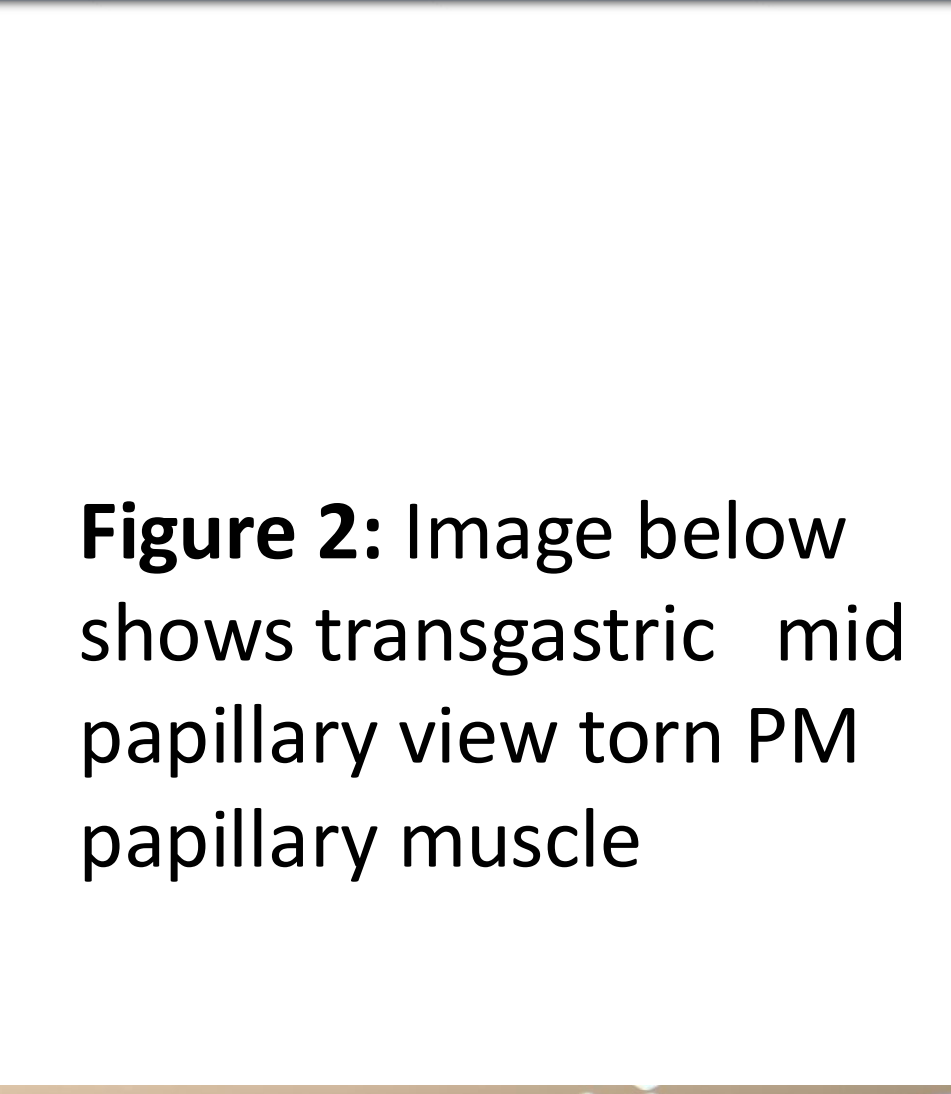


Figure 2: Image below shows transgastric mid papillary view torn PM papillary muscle

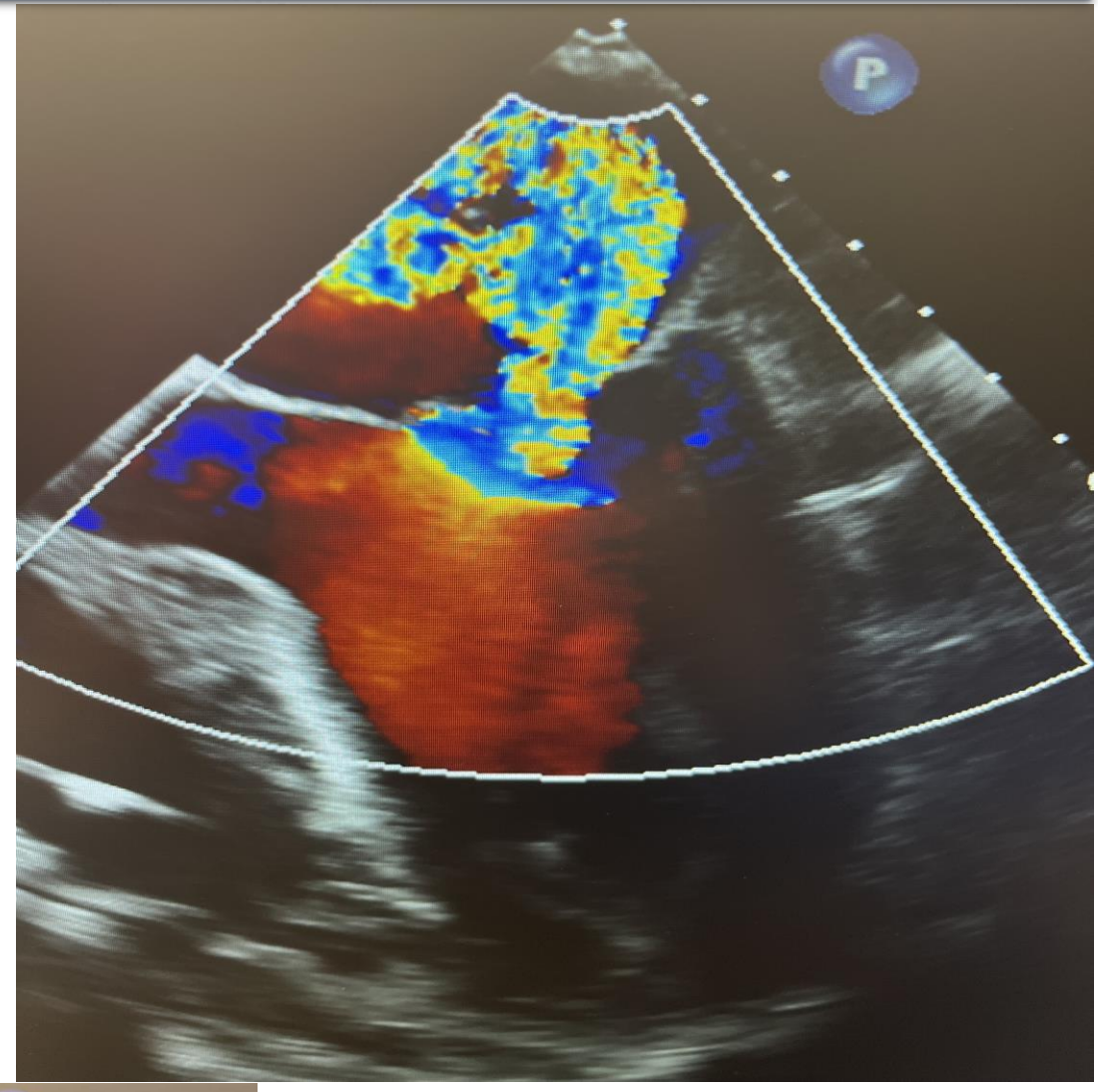


Figure 3: Image above shows mid esophageal 4 chamber severe MR

POSTOPERATIVE EVENTS

POSTOPERATIVE EVENTS

- **Post-op I:**
 - **Initial Course:** Arrived to CTICU with pulse pressure ~10mmHg; lost pulsatility within 45 min (MAP 63–65 mmHg on norepinephrine).
 - **Interventions:** TEE showed minimal AoV opening: dobutamine started for LV venting—improved pulsatility but caused North–South syndrome at 5 mcg/kg/min, resolved when weaned to 2.5 mcg/kg/min.
 - **Findings:** Increased ECMO flows improved oxygenation but reduced pulsatility; TEE revealed LV “smoke,” poor AoV opening, and rising PA pressures (25/20 → 67/31 mmHg).
 - **Outcome:** Neurologically intact on sedation holiday; taken to OR for mitral valve replacement due to worsening LV distention and oxygenation imbalance.
- **Post-op II:**
 - **Neurologic:** CT head showed bilateral embolic strokes and hypoxic injury; no neurosurgical intervention indicated. Prognosis for meaningful neurologic recovery is extremely poor.
 - **Renal:** Oliguric from ischemic ATN secondary to cardiogenic shock; on CRRT with improving filling pressures.

DISCUSSION AND CONCLUSIONS

North-South syndrome/Harlequin Syndrome/Differential Hypoxemia

- **Mechanism:** In femoral VA-ECMO, retrograde ECMO flow competes with desaturated LV output, creating a mixing zone that results in dual circulation—oxygenated lower-body perfusion and hypoxic upper-body perfusion.
- **Clinical Effects:** Severe pulmonary dysfunction can lead to differential or fulminant differential hypoxia, causing upper-body cyanosis, cerebral and myocardial ischemia, neurologic changes, arrhythmias, and hemodynamic instability.
- **Diagnosis:** Identified by marked SaO₂ discrepancies between the right radial and femoral arteries; right-arm pulse oximetry and targeted ABGs are key for detecting cerebral hypoxia.
- **Management:** Includes increasing ECMO flow, optimizing ventilator settings, reducing native cardiac output, and, if persistent, reconfiguring the circuit (e.g., V-AV ECMO or added SVC/central drainage).

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