Ventricular Septal Rupture (Defect), Postmyocardial Infarction

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Risk

- Historically seen in 1–3% of MIs prior to era of acute revascularization.
- Incidence is 0.2% in current era of acute percutaneous intervention.
- Most occur within 1 wk of MI; 20–30% occur in first 24 h post-MI.
- Rarely occurs >2 wk post-MI.
- Medical management alone results in a mortality >90%.

Perioperative Risks

- Accounts for 5% of MI-related deaths,
- Without surgical therapy, survival is less than 10% at 1 mo.
- Surgical short-term survival 40–81%.
- Increased mortality seen in the setting of urgent repair (due to tissue fragility), posterior VSD, preop dialysis, mitral regurgitation, and redo cardiac surgery.
- Improvements in surgical techniques have enabled earlier surgery prior to hemodynamic deterioration, with associated increase in survival.
- Percutaneous device closure with GA and TEE has similar mortality.

Worry About

- Associated papillary muscle rupture
- Poor systemic perfusion and end-organ dysfunction
- Pulm congestion with massive L-to-R shunt

Overview

- Sudden onset of holosystolic murmur with thrill and hemodynamic deterioration (hypotension and pulm congestion).
- Despite advances in periop management, expect increased morbidity and mortality.
- Expect a complicated postop course with prolonged ICU stay.

Usual Treatment

- Repair of new VSD with hemodynamic deterioration using pericardial or prosthetic patch material.
- Support preop with inotropic agents/intra-aortic balloon counterpulsation.
- Percutaneous device closure as an alternative to surgery.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Low forward cardiac output due to massive L-to-R shunt	Sudden onset of hypotension and shock	Loud holosystolic murmur and thrill	ECHO, cardiac cath
RESP	Congestion/edema	Respiratory distress	Rales	CXR
RENAL/HEPATIC	Dysfunction due to cardiogenic shock	Anuria		ABGs, Foley cath

Key References: Arnaoutakis GJ, Zhao Y, George TJ, et al.: Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database, *Ann Thorac Surg* 94(2):436–443, 2012; Jeppsson A, Liden H, Johnsson P, et al.: Surgical repair of post infarction ventricular septal defects: A national experience, *Eur J Cardiothorac Surg* 27:216–221, 2005.

Perioperative Implications

Preoperative Preparation

- Consider elective tracheal intubation and PEEP.
- Support cardiac output using inotropic agents.
- Lower resistance to forward cardiac output using afterload reduction, including intra-aortic balloon counterpulsation.
- Obtain coronary angiogram. Concurrent revascularization can potentially improve outcome, although recent studies have not found this.

Anesthetic Technique

- High-dose opioid/muscle relaxant technique common
- Prior to CPB, use minimal FIO_2 and PEEP (maximizes PVR) to decrease L-to-R shunt across VSD.

Monitoring

- Intra-arterial line.
- Most use PA cath owing to pulm Htn and for shunt quantitation; step up saturation between right atrium and PA to measure degree of shunting.
- Thermodilution cardiac output may be falsely elevated.
- TEE to define anatomy, diagnose assoc papillary muscle rupture, monitor ventricular function (including stroke volume), and assess adequacy of surgical repair.

Airway

- High airway pressures and frequent suctioning in the setting of pulm edema.

Induction

- High-dose opioid technique to maintain hemodynamic stability. Avoid vasodilation assoc with volatile anesthetics.

Maintenance

- If pt is hypertensive, titrate low doses of volatile agent or benzodiazepines.

Surgical Stages

- Pre-CPB:
  - Median sternotomy with aortic and biatrial cannulation.
  - Vein or internal mammary artery harvest may be required for concomitant myocardial revascularization.
  - Lowest FIO_2 consistent with adequate oxygenation.
- CPB:
  - Maintain Hct using hemofiltration and transfusion.
- Post-CPB:
  - Inotropic support almost universally required for LV failure.
  - RV failure common.
  - Assess ventricular repair using TEE or right atrial-to-pulm O_2 sat ratio.
  - FIO_2: 1.00 to minimize PVR.
  - May require ventricular assist devices.
  - Rule out residual shunting by TEE.
  - Emergent surgery is associated with residual shunt.
- Blood loss/volume concerns:
  - Antifibrinolytic therapy (beginning pre-CPB).
  - Transfuse coagulation factors based on results obtained from point-of-care testing (TEG, platelet function analyzers).

Postoperative Considerations

- Postop renal/hepatic/neurologic dysfunction
- Postop LV, RV, or biventricular failure

Anticipated Problems/Concerns

- Cardiogenic shock with MODS
- Prolonged ventilatory dependency and ICU stay
- Course not dramatically improved with percutaneous device closure

Ventricular Tachyarrhythmias

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Risk

- VTach/VFIB are uncommon but potentially fatal dysrhythmias requiring urgent diagnosis and management.
- Risk increases with age owing to the higher incidence of structural and ischemic heart disease and cardiac failure.
- Primary cause of sudden death and accounts for 75–80% of sudden cardiac death. Incidence in USA is about 300,000/y and similar in other developed nations.
- Males at greater risk (46% vs. 34%).
- Pts under 30 with HOCM, myocarditis, RV dysplasia, or long-QT syndrome are at higher risk for VTach/VFIB.

Perioperative Risks

- Cardiac and vascular surgery (up to 50% incidence) does not influence late mortality if LV function is preserved.
- Low cardiac output after CABG (requiring pressors) predicts life-threatening VTach/VFIB within 72 h postop.
- Cardiac ischemia.
- Uncorrected electrolyte and/or acid-base disturbances, hypoxia, hypercarbia, hypothermia.
- Use of class 1 and 3 antiarrhythmics, sympathomimetics, QT-prolonging drugs.
- Placement of central venous catheters.

Worry About

- Electrolyte imbalance (particularly hypokalemia and hypomagnesemia), acid-base disturbances, hypoxia, hypotension, fluid overload, ongoing myocardial ischemia, and metabolic disturbances.
- Use of IV epinephrine and other catecholamines/sympathomimetics.
- Drugs that prolong QT (organophosphates, antipsychotics, tricyclics) may precipitate PVT, particularly in Brugada and other long-QT syndromes.
- Poor cardiac function.
- Modulation of neuroendocrine stress responses.