ASK THE EXPERT

Perioperative Management of Pulmonary Hypertensive Crisis

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Pulmonary hypertensive crisis is characterized by an acute rise in pulmonary pressures, causing pressure overload of the right ventricle (RV) and decreased cardiac output. Reduced cardiac output through the pulmonary circuit leads to hypoxia, further exacerbating the increased pulmonary vascular resistance. Right ventricular dilation shifts the interventricular septum toward the left ventricle (LV), impeding LV filling and further compromising cardiac output. Systemic hypotension, metabolic and respiratory acidosis can ensue. In general, pulmonary hypertension (PH) patients have higher mortality during surgical procedures, ranging from 4%-24%.[1] Pulmonary hypertensive crisis in the perioperative setting is associated with even worse outcomes and mortality may surpass 50%.[2] Survival requires prompt recognition and intervention.

The best way to recognize and/or prevent perioperative pulmonary hypertensive crisis is to be prepared for it ahead of time. Preoperative assessment should include invasive hemodynamics if not recently performed. Recommendations to the surgical team prior to surgery should include local instead of general anesthesia if at all possible, utilization of a cardiac anesthesia team, and preferably one experienced with PH. Local anesthesia with either epidural or peripheral nerve block may avoid the excess morbidity and mortality of general anesthesia. Spinal anesthesia should be avoided in patients with PH due to the potential for systemic hypotension from vasodilation causing hemodynamic collapse. Perioperative invasive hemodynamic monitoring should be performed and will allow for early recognition of pulmonary hypertensive crisis. Continuation of PH medications throughout the perioperative period is also crucial.[3] Postoperatively, patients should be monitored in the intensive care unit. Risk factors for the development of pulmonary hypertensive crisis and poor surgical outcomes in PH patients can be found in Table 1.[4]

Prompt recognition of pulmonary hypertensive crisis is critical. Clinical parameters to identify include systemic hypotension, hypoxia, tachycardia, decreased urine output, and/or frank anuria. Confirmation of pulmonary hypertensive crisis is via invasive hemodynamics, thus the need for perioperative monitoring. Rising pulmonary and right atrial pressures with decreasing cardiac output is the hallmark. Echocardiography may be useful as an adjunctive measure to show worsening RV function and/or RV dilation.

Treatment options for perioperative pulmonary hypertensive crisis include supportive measures, therapies to treat the pulmonary vascular system, including inhaled or parenteral pulmonary vasodilators to lower pulmonary vascular resistance and inotropic support for the RV, and therapies to support the systemic vasculature. Supportive measures include 100% oxygen, hyperventilation to decrease CO_2, maintaining lung volumes near or at functional residual capacity (if on mechanical ventilation) to avoid hyperinflation or atelectasis (both of which may raise pulmonary vascular resistance), paralytics to decrease metabolic demand, maintenance of normal core body temperature, and treating acid/base disturbances.[5] Filling pressures should be optimized, which in some cases may require diuretics as severely elevated RV filling pressure may exacerbate renal dysfunction. Acute pulmonary vasodilators can include inhaled nitric oxide or prostacyclins. Prostacyclins can be delivered either via inhalation/nebulization or parenterally. Intravenous use of sildenafil has also been reported and may be of use.[6,7] Milrinone or dobutamine can be used for inotropic support of the RV. Milrinone has vasodilatory properties as well and so may be preferential to dobutamine. Milrinone has also been reported to be administered by nebulization.[4,8] Systemic hypotension should be addressed with pressors as needed, with an eye to increase perfusion pressure adequately but avoiding excessive increases in afterload. This can be achieved with low-dose dopamine, norepinephrine (which may also have some positive inotropic effect), or vasopressin.[9] If medical management is unsuccessful, mechanical support with extracorporeal membrane oxygenation (ECMO) can be considered.[10] ECMO may be useful in settings of inability to oxygenate and/or persistent systemic hypotension despite medical support with inotropes and pressors.

In summary, pulmonary hypertensive crisis can be a life threatening perioperative complication. In PH patients requiring surgery, preoperative assessment and planning may by preventive. Successful outcomes require perioperative vigilance and rapid intervention.

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Table 1. High-risk parameters for perioperative pulmonary hypertensive crisis.

Patient factors	Surgical factors
Clinical: Poor functional class (class III, IV greater risk than I, II) Low 6-minute walk distance (<330 meters) High BNP (>330) History of a pulmonary embolism	Type of surgery: Intermediate- to high-risk surgery (abdominal, orthopedic, thoracic, vascular, transplant) Emergent surgery at higher risk than planned procedures
Echocardiography: Preoperative RV dysfunction (global assessment, RV index of myocardial performance ≥0.75, TAPSE <1.6 cm, more recent measures of tissue Doppler or speckle tracking-derived strain) RV dilation and/or hypertrophy Flattened, or D-shape, septum with LV diastolic dysfunction Severe tricuspid regurgitation Pericardial effusion	Type of anesthesia: Spinal anesthesia high risk and should be avoided; may cause hemodynamic collapse from systemic vasodilation General anesthesia higher risk than local anesthesia (epidural or peripheral nerve block)
Invasive hemodynamics: Severe PAH (MPAP >55 mm Hg) Evidence of decompensated RV function (RAP >12 mm Hg, cardiac index <2.2 L/min/m^2)	Intraoperative events: Duration of surgery/anesthesia (>3 hours) Estimated blood loss (hemorrhagic shock leading to catecholamine surge and cardiovascular collapse; need for blood products causing fluid shifts and/or lung injury)

BNP, brain natriuretic peptide; RV, right ventricle; TAPSE, tricuspid annular plane systolic excursion; LV, left ventricle; PAH, pulmonary arterial hypertension; MPAP, mean pulmonary arterial pressure; RAP, right atrial pressure. See reference 4 for a good review.

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