

Severe Mitral Regurgitation Complicates Risk of Pulmonary Edema in Severe Preeclampsia

Simone Sukhdeo, PhD; Gerardo Rodríguez, MD

Department of Anesthesiology, Rutgers Robert Wood Johnson Medical School, New Brunswick, NJ

Introduction

Acute pulmonary edema is a life-threatening complication that occurs in 3% of preeclampsia cases and represents the leading cause of maternal mortality from preeclampsia.¹ We report the management of a parturient with severe preeclampsia and newly discovered mitral regurgitation who developed acute pulmonary edema requiring emergent cesarean section.

Case Presentation

A 31-year-old patient, G2P0, with history of fetal demise associated with severe preeclampsia presented at **24 weeks gestation with severe features of preeclampsia**, including an episode of palpitations and dyspnea. PMH included hypertension and **systemic lupus erythematosus (SLE)**.

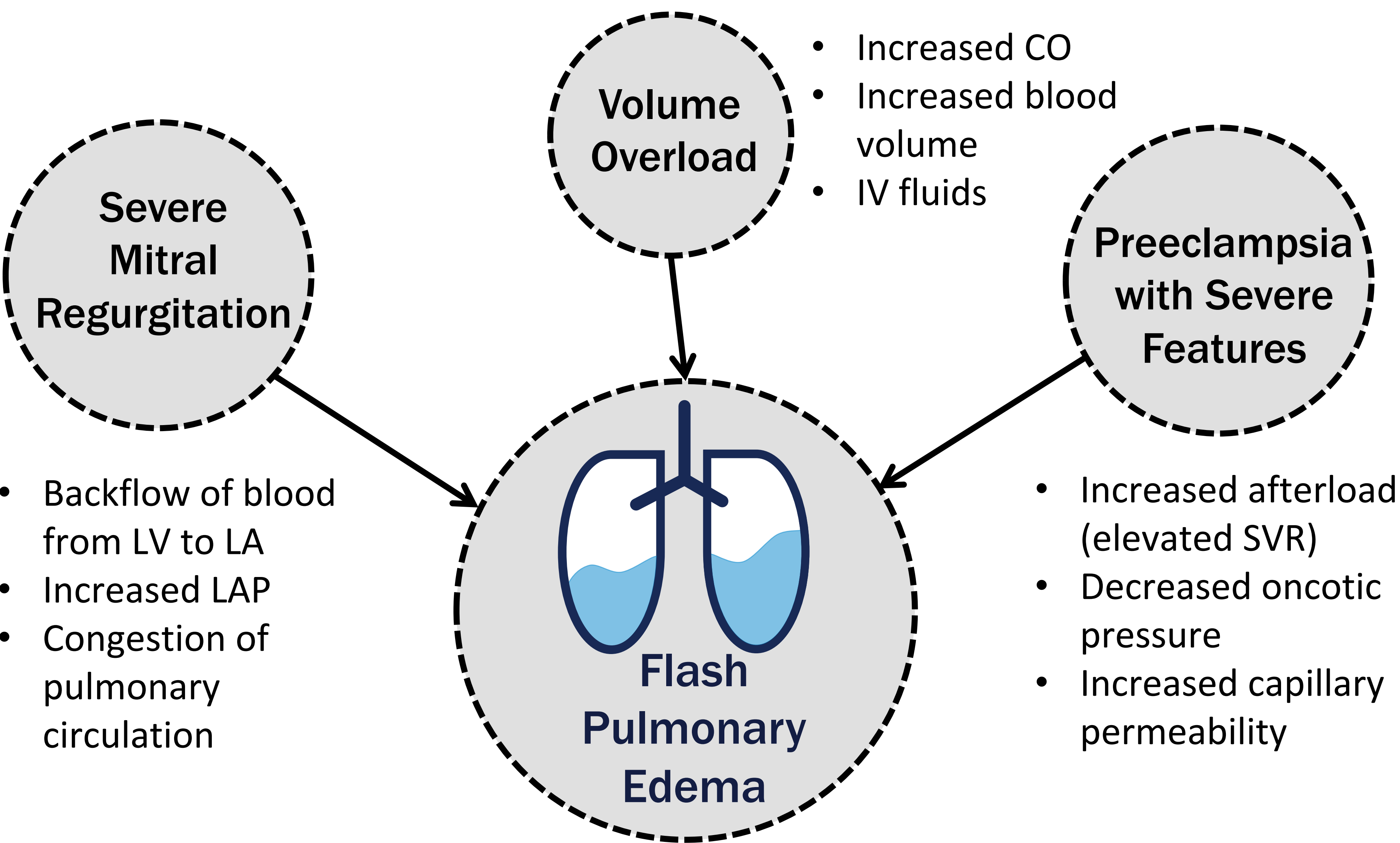
Lab findings were remarkable for BP 152/102, HR 131, proteinuria and elevated creatinine. Rapid progression to hypertensive crisis with severe headaches was stabilized on IV labetalol and magnesium sulfate infusion. A TTE was performed given history of SLE and palpitations, revealing **severe mitral valve regurgitation**.

On HD 3, the patient developed acute respiratory distress with hypoxia, jugular vein distention and inspiratory crackles bilaterally. Despite interventions with furosemide and prior to non-invasive ventilation support, the patient had **respiratory decompensation and fetal distress** requiring **emergent cesarean section**. Rapid sequence induction and intubation was performed with etomidate and succinylcholine. General anesthesia was maintained with sevoflurane, propofol and rocuronium. The infant was delivered and intubated for low Apgar scores.

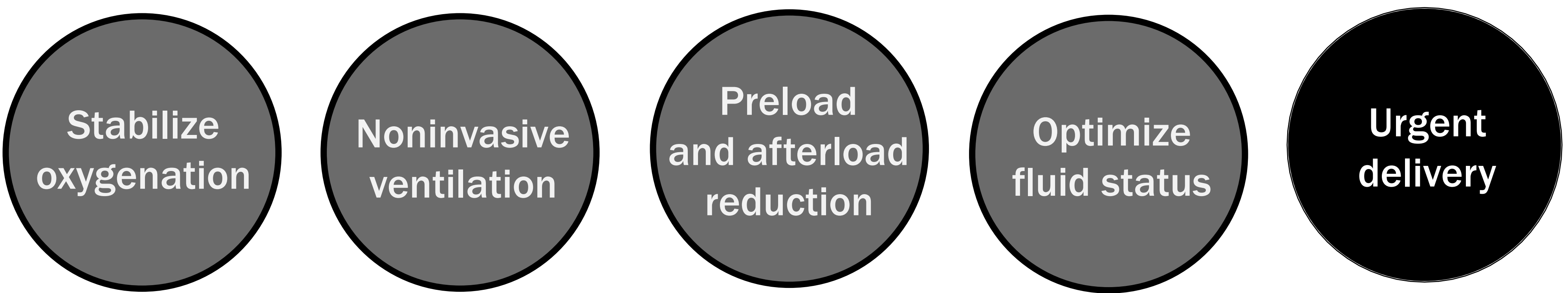
The patient remained intubated postoperatively. She was successfully extubated the next day after extensive diuresis and ventilator weaning, and discharged home on POD 4.

Mechanism and Management of Pulmonary Edema due to Severe Preeclampsia and Severe Mitral Regurgitation

Mechanism of Pulmonary Edema:



Peripartum Management:



Discussion

Preeclampsia and severe mitral regurgitation can be a high risk interaction of complex and opposing disease processes that promotes the development of acute pulmonary edema and congestive heart failure.

Mitral regurgitation (MR): SLE is an inflammatory autoimmune disorder that can result in valvular heart disease.² Severe MR causes congestion of pulmonary circulation as blood backflows to the left atrium and into the lungs. Hemodynamic stresses of pregnancy (i.e., increased blood volume and cardiac output) can unmask previously asymptomatic mitral valve disease.

Preeclampsia (PEC): Preeclampsia is characterized by hemodynamic and fluid balance derangements.³ PEC elevates systemic vascular resistance, decreases oncotic pressure, and increases capillary permeability which predisposes parturients with PEC to pulmonary edema. Endothelial dysfunction offers a pathway for extravasation of fluid into pulmonary interstitium and alveoli.

During pregnancy, PEC sets the stage for MR-induced flash pulmonary edema. The combination of systemic hypertension and volume overload of the pulmonary circuit creates a situation with significant danger of maternal and fetal compromise.

Early intervention by a multidisciplinary team, including anesthesiology, may mitigate the severity of complications by focusing on stabilization of oxygenation, noninvasive ventilation, hemodynamic stabilization via preload and afterload reduction, and fluid restriction.^{1,3} In the event of rapid decompensation, urgent delivery is the most effective therapeutic choice.³

References

1. Dennis AT, Solnordal CB. "Acute pulmonary oedema in pregnant women." *Anaesthesia* 67.6 (2012): 646-659.
2. Flutue A, Chaudhari S, Frishman WH. "Valvular heart disease and systemic lupus erythematosus: therapeutic implications." *Heart Dis* 5.5 (2003): 349-53.
3. Dua S, Maurtua MA, Cywinski JB et al. "Anesthetic management for emergency cesarean section in a patient with severe valvular disease and preeclampsia." *Int J Obstet Anesth* 15.3 (2006): 250-253.