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Anesthetic Management of the Parturient with Systemic Lupus Erythematosus, Pulmonary Hypertension, and Pulmonary Edema

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Coexistence of pulmonary hypertension with pulmonary parenchymal and vascular inflammation secondary to systemic lupus erythematosus (SLE) severely limits the cardiac and pulmonary reserves of parturients, predisposing mother and fetus to hypoxemia, and increasing the morbidity and mortality rates associated with anesthetic interventions. We report the successful anesthetic management of a parturient with pulmonary hypertension complicated by SLE pneumonitis and vasculitis, pulmonary edema, and severe orthopnea.

Case Report

Our patient, a 28-yr-old, gravida VII, para I, ab V, with an intrauterine pregnancy at 31 weeks' estimated gestational age, was admitted to the hospital by her pulmonologist. Her chief complaint was worsening dyspnea at rest, associated with paroxysmal nocturnal dyspnea, 3-5 pillow orthopnea, and bilateral lower extremity edema. She claimed to have no drug allergies. The surgical history was significant for a postpartum cholecystectomy 7 yr prior with general anesthesia that required postoperative ventilatory support for 7 days. Her medical records suggested that an acute, multilobe pneumonitis, combined with decreased respiratory effort secondary to upper abdominal postsurgical pain, resulted in severe hypoxemia and was slow to resolve. A subsequent lung biopsy was diagnostic for SLE, lupus pneumonitis, and vasculitis. Moderately restrictive and severe interstitial lung disease developed and required immunosuppressive therapy from 1995-1997. Medications included home oxygen 2-4 l/min *via* nasal cannula and betamethasone 35 mg intramuscularly each day for 3 days. Antepartum pulmonary function parameters included total lung capacity of 2.67 l, forced expiratory volume in 1 s of 1.62 l, and DLCO 7.7 $ml \cdot min^{-1}\ mmHg^{-1}$, which were 48%, 51%, and 33% of predicted, respectively.

She tolerated minimal exercise with mild dyspnea until 27 weeks estimated gestational age, when she developed severe dyspnea at rest that required home oxygen therapy of 3 l/min during waking hours. Upon admission, she had severe dyspnea, tachypnea, and orthopnea at rest. Her blood pressure was 130/80 mmHg; maternal heart rate 120 beats/min; respiratory rate 40-45 breaths/min; temperature 36.1°C; weight 70 kg; and height 65 inches. Pulmonary examination revealed inspiratory crackles from the base to midthorax bilaterally; cardiac examination showed a regular, tachycardic rhythm with an S_4 gallop. Moderate, dependent, pretibial edema was present bilaterally despite a mild diuresis from furosemide. Arterial blood-gas analysis revealed *p*H 7.39, pO_2 178 mmHg, and pCO_2 52 mmHg while the patient received oxygen at 6 l/min by face mask. Compared with the patient's previous echocardiograms, a transthoracic echocardiogram on admission revealed increased right-atrial enlargement, new right-ventricular dilation, and higher pulmonary arterial pressure, estimated at 40-50/25-35 mmHg. Direct measurements revealed pulmonary arterial pressure of 48/25 with a mean of 32 mmHg. An anteroposterior chest radiogram was obtained prior to delivery and demonstrated bilateral pleural effusions, fluid in the fissure between the right middle and lower lobes, air bronchograms, and patchy infiltrates in all lung fields consistent with severe, diffuse, interstitial pulmonary edema. Despite declining maternal respiratory function, the fetus continued to have frequent spontaneous movements and a reassuring fetal heart rate pattern without decelerations.

After receiving metoclopramide 10 mg intravenously and sodium bicitrate 30 ml orally, the patient moved onto the operating table in the semisitting position. We applied routine monitors (noninvasive blood-pressure cuff, five-lead electrocardiogram, continuous-pulse oximetry), transduced pressures from *in situ* radial and pulmonary arterial cath-

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Received from the Department of Anesthesia and Critical Care, University of Chicago, Chicago, Illinois. Submitted for publication October 29, 1998. Accepted for publication April 1, 1999. Support was provided solely from institutional and/or departmental sources.

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Key words: Cardiovascular physiology; choice of anesthesia; critical care consultation.

Table 1. Perioperative Hemodynamic and Respiratory Parameters

	Perioperative Times (h)								
Parameter	−4	−2	L	D	1.5	4.5	6.5	11	16 (E)
HR (bpm)	122	127	102	102	90	112	102	93	124
SAP									
Systolic (mmHg)	122	123	135	132	132	128	130	132	128
Diastolic (mmHg)	70	77	70	71	80	77	82	86	74
Mean RAP (mmHg)	1	10	8	10	8	7	5	4	4
PAP									
Systolic (mmHg)	32	44	41	40	34	40	36	36	32
Diastolic (mmHg)	10	24	22	20	10	18	16	14	16
PCWP (mmHg)						12	10	8	4
$F_{I_{O_2}}$ (%)	28	28	100	30	100	50	40	40	40
pH	7.38	7.35			7.32	7.32	7.39	7.40	7.39
Pa_{O_2} (mmHg)	119	146			355	208	143	167	166
Pa_{CO_2} (mmHg)	44	51			55	59	49	52	52
O_2 saturation (%)	99	99	100	100	99	99	99	98	99
HCO_3^- (mEq/l)	26	27			28	20	29	32	31
BE (mEq/l)	1	1			1	2	3	6	5

At each perioperative time, relative to delivery, a set of hemodynamic and respiratory parameters is presented. For perioperative times, −4 and −2 h = preoperative baseline values; L = laryngoscopy and endotracheal intubation; D = delivery of the infant, 1.5, 4.5, 6.5, 11, and 16 h are postoperative values with E = extubation.

HR = heart rate; SAP = systemic arterial pressure; RAP = right atrial pressure; PAP = pulmonary arterial pressure; PCWP = pulmonary capillary wedge pressure; BE = base excess.

eters, preoxygenated the patient with 100% O_2, applied cricoid pressure, and then induced anesthesia with fentanyl 7 μg/kg intravenously, etomidate 0.05 mg/kg intravenously, and succinylcholine 1.5 mg/kg intravenously. Laryngoscopy and intubation were performed after reclining the patient to the supine position with leftward uterine displacement. Anesthesia was maintained with 100% oxygen and isoflurane 0.8–1.6%, titrated to maintain preanesthetic hemodynamic values (table 1). Following delivery of a 2,685-g female neonate with Apgar scores of 6 and 8 at 1 and 5 min, respectively, isoflurane was decreased to 0.3% in nitrous oxide 70% and oxygen 30%, and fentanyl 250 μg intravenously and morphine 7 mg intravenously were given. Total intravenous fluid volume was 400 ml, estimated blood loss was 800 ml, and total urine output was 100 ml. The patient was sedated overnight with an intravenous propofol infusion (20–30 $\mu g \cdot kg^{-1} \cdot min^{-1}$) until extubation the next morning. Postcesarean patient-controlled analgesia was provided as intravenous morphine 1.0 mg bolus with a 10-min lockout period.

The patient's infant daughter showed no signs of respiratory depression, required no ventilatory support, and received routine care in the normal newborn nursery. Mother and daughter were discharged home on the fourth postoperative day with no adverse sequelae.

Discussion

The principal goal of cardiovascular management of a parturient with pulmonary hypertension and diffuse interstitial pulmonary edema from SLE-induced pulmonary parenchymal and vascular disease is to optimize pulmonary blood flow. Flow is optimized by maintaining a normal sinus rhythm, minimizing pulmonary vascular resistance (PVR), avoiding significant decreases in systemic vascular resistance, and regulating right-ventricular preload and pulmonary arterial pressures in the narrow ranges sufficient to overcome a high pulmonary vascular resistance without exacerbating pulmonary edema.[1]

Pulmonary vascular resistance may be increased by hypoxia, hypercapnia, endogenous catecholamines (as a result of stress or pain), or Valsalva maneuvers. Although vaginal delivery avoids the potential complications of surgery (trauma, hemorrhage, infection, postoperative pain, and delayed recovery), we anticipated a prolonged and difficult course of labor with exacerbations of preexisting tachypnea, tachycardia, pulmonary hypertension, pulmonary edema, and supply-demand imbalances in maternal and fetal oxygen.[2] Because fetal and maternal well-being could not be predictably controlled during a trial of labor and vaginal delivery, our obstetricians agreed to perform an elective cesarean delivery.

Epidural anesthesia for cesarean delivery, although described in parturients with pulmonary hypertension,[3–9] results in sympathetic blockade, which diminishes venous return to the right atrium, thus eliminating the critical driving force for adequate pulmonary blood flow. The onset of hypotension and hypoxemia can be lethal because pharmacologic interventions and efforts at closed chest compressions do not reliably overcome

pulmonary hypertension to restore pulmonary blood flow. In addition, spontaneous ventilation in our patient required 60-degree head elevation that not only presented an unacceptably difficult surgical approach for our obstetricians but also would severely exacerbate the effects of sympathectomy, volume shifts, fluid requirements, and perioperative hemodynamic risks. In contrast, ventilation, oxygenation, and systemic and pulmonary hemodynamics can be better regulated with general anesthesia in the supine position necessary for elective cesarean delivery.

For intravenous induction of anesthesia, etomidate was chosen to maintain systemic vascular resistance and minimize hypotension; fentanyl was chosen to prevent untoward hemodynamic responses to laryngoscopy, intubation, and intraoperative pain. Isoflurane maintained pulmonary and systemic pressures within preoperative ranges, and nitroglycerin and dobutamine were immediately available.

Intrathecal and epidural opioids were considered for postcesarean analgesia[8,10]; however, our patient refused to consent. After extubation, cautious administration of patient-controlled morphine with supplemental oxygen allowed for satisfactory analgesia, normal oxyhemoglobin saturation, and only a slight increase in baseline carbon dioxide retention with no episode of severe respiratory depression or hypoxemia, as expected.[11]

Pregnancy complicated by pulmonary hypertension has a high mortality rate. Unique to our patient was her coexisting, SLE-related restrictive lung disease, pulmonary edema, and orthopnea that severely reduced the margin of safety normally associated with routine obstetric and anesthetic interventions during labor and delivery. In this setting, elective cesarean delivery using general anesthesia with invasive cardiovascular monitoring successfully prevented serious adverse sequelae.

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