Letter to the Editor

Dexmedetomidine in pulmonary hypertension

To the Editor,

We anesthetized a 35-year-old woman, weighing 71 kg, for laparoscopy-assisted vaginal hysterectomy and bilateral salpingectomy. On two-dimensional echography her pulmonary artery pressure was 80/40 mmHg (mean pressure: 60 mmHg). Ejection fraction was 64% and right atrium and ventricle were dilated with moderate tricuspid and pulmonary regurgitation. She was hypothyroid for 2 years and taking thyroxine 100 mg/day. For rheumatoid arthritis, she was on methotrexate 10 mg/week for 1 year. She was also taking spironolactone 30 mg, furosemide 20 mg and sildenafil 20 mg once daily. Other routine investigations including liver function tests were normal. After obtaining consent for a high risk surgery and explaining the possible consequences of general anesthesia, we induced anesthesia with 2 mg midazolam, 200 mg fentanyl, and 4 vol% sevoflurane, and 40 mg atracurium and 70 μg dexmedetomidine intravenously over 15 minutes prior to induction. Intraoperatively, we monitored oxygen saturation, electrocardiography (lead II, V5, V6), arterial blood pressure, end-tidal carbon dioxide, and temperature. We did not consider a pulmonary artery catheter or central venous catheter due to possible arrhythmia during this procedure. We had no facility of using transesophageal echocardiography intraoperatively. Dexmedetomidine infusion was continued intraoperatively at 36 μg/hour (i.e., 0.5 μg/kg/minute). There was no hemodynamic response seen with laryngoscopy, port insertion, or lithotomy positioning, intraoperatively or during extubation at the end of 90 minutes. Dexmedetomidine was continued throughout surgery and 3 hours after surgery in the intensive care unit. She was kept in intensive care for 24 hours, moved to the ward on the next day, and discharged home on Postoperative Day 3.

Surgical or procedural intervention of any kind under any anesthetic technique can lead to significant morbidity and mortality in patients with severe pulmonary hypertension. Hypoxemia, hypercarbia, acidosis (respiratory or metabolic), pain, increased intrathoracic pressure, hypothermia, hypervolemia, and intubation or extubation response increases pulmonary vascular resistance (PVR), leading to major adverse cardiac events. Precipitous hypotension during anesthesia induction should be avoided. Intermittent positive pressure ventilation can also affect the right ventricular preload and can have deleterious effects.

Dexmedetomidine is a centrally acting α2 agonist that causes sympatholysis, reduces heart rate, and attenuates hemodynamic response to intubation and extubation, thereby preventing increased PVR by reducing stress response. It does not decrease PVR by direct action. The requirement for narcotics is also reduced. Due to these desirable properties of dexmedetomidine, it is an ideal agent to be used as an adjunct in the anesthesia management of patients with pulmonary hypertension during general or regional anesthesia, and can be used as the sole anesthetic for procedural sedation as well. There are no known interactions of dexmedetomidine with drugs used in pulmonary hypertension such as sildenafil and diuretics.

References


Abhijit S. Nair*, Balasubramanyam Kandukuri, Thota Venkata Sanjeev Gopal Department of Anesthesia, Care Hospital, Axon Anesthesia Associates, Hyderabad, India

* Department of Anesthesia, Cellar 1, Care Hospital, Road no.1, Banjara Hills, Hyderabad 500034, India. E-mail address: abhijitnair95@gmail.com (A.S. Nair).