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Background

Pulmonary hypertension is associated with significant perioperative risk. The likelihood of death or need for major resuscitation has been reported at 6% in children under anesthesia for cardiac catheterization (1). Suprasystemic pulmonary hypertension has been shown to increase this risk eightfold (2).

Case Report

12 yo female presented with elevated BP of 192/139 and evidence of suprasystemic pulmonary hypertension on echo. History was remarkable for VSD (spontaneously closed), syncope at 6 yo, and general aversion to exercise. Urgent cardiac catheterization was required for further evaluation and initiation of pulmonary vasodilators. We obtained invasive access with local anesthesia and sedation to allow close hemodynamic monitoring and vasoactive support. We then performed a carefully titrated induction with a propofol/ketamine (ketofol) 1:1 mg infusion and placed an endotracheal tube. Doses used for ketofol ranged between 100-300 mcg/kg/min. Hemodynamic testing confirmed severe pulmonary hypertension (MPAP near 100mmHg) with minimal response to O₂, iNO and epoprostenol. Patient was subsequently extubated and brought to the cardiac ICU on epoprostenol. Treprostinil was then slowly titrated up as tolerated by her systemic BP, intermittently requiring phenylephrine infusion to maintain her SVR. Atrial septostomy was later performed to relieve right-sided pressures.

Discussion

This patient was thought to have severe primary pulmonary hypertension with resultant decrease in cardiac output, leading to increased SVR and systemic hypertension. Our anesthetic management goals included maintaining high SVR and avoiding further increase in PVR. Based upon preoperative echo, suprasystemic RV pressure resulted in displacement of her intraventricular septum towards the LV. During anesthetic induction, an acute decrease in SVR or increase in PVR could cause a greater degree of septal displacement. This could lead to obstruction of LV filling, reduced stroke volume, blood pressure and coronary perfusion, worsening of right-sided heart failure and ultimately cardiovascular collapse (3).

The safety and effectiveness of ketofol has been demonstrated in pediatric patients undergoing cardiac catheterization (4). Research in adults has demonstrated that ketamine causes an increase in PVR during spontaneous respiration (5); however, studies have shown that ketamine does not cause increased PVR in children while breathing spontaneously under anesthesia (6). One study of children undergoing cardiac catheterization found that propofol significantly decreased MAP, while ketamine increased MAP and had no effect on SVR, PVR or overall MPAP (7). In the setting of severe systemic and pulmonary hypertension, the hemodynamic effects of propofol and ketamine can counteract each other, potentially providing hemodynamic stability in patients at high risk for cardiovascular collapse.

References

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