Anesthetic considerations on pulmonary hypertension: A case report and minireview

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Abstract: Pulmonary hypertension presents a great challenge to the anesthesiologist. The number of patients suffering from pulmonary hypertension presenting for surgery is increasing because of increased age and the modern medical and surgical therapy as well. We present a case of a patient suffering from pulmonary hypertension scheduled for left radical nephrectomy due to left kidney tumor. Preoperative optimization and prevention of deterioration of pulmonary hypertension and right side heart failure are the problems the anesthesiologist must deal with. A good collaboration among cardiologist, anesthesiologist, and the surgeon can guarantee the patient’s outcome.

Keywords: General Anesthesia, Pulmonary Hypertension, Right Heart Failure

1. Introduction

Pulmonary hypertension is a rare disease, but with increased preoperative morbidity and mortality. PH is divided in the primary form and the secondary one. Primary PH is a rare condition without any evident causes (1), whereas the secondary PH is often due to cardiac and/or pulmonary diseases. Chronic pulmonary diseases, mitral valve stenosis/regurgitation, and left side ventricular diseases, are the commonest causes of secondary PH. The pathophysiological mechanisms include hypoxia-induced vasoconstriction (2), elevated left atrial pressure, and increased pulmonary venous and arterial pressure. PH can progress in atrial fibrillation, and right side heart failure with tricuspid valve regurgitation, further increasing the gravity of the disease (3). The anesthesiologist’s role consists on preoperative optimization, prevention of further deterioration of PH, and finally prevention or treatment of acute right side heart failure.

2. Case Report

The patient V.B, number 550754, 72 years old white man, with no previous cardiac medical history, was recovered in our institute diagnosed with left kidney tumor. In the preoperative consult, unexpected atrial fibrillation was clinically diagnosed confirmed by ECG as well. Echocardiography examination found aortic valve stenosis 1, 7 cm², moderate mitral valve regurgitation (40-60% regurgitation fraction), left ventricle and left atrium dilatation with normal function (EF 61%, SHF 33%), tricuspid valve regurgitation, and systolic pulmonary hypertension (PsAP 52 mmHg). Thoracic X-Ray examination revealed cardiomegaly and a decreased cardiopulmonary ratio. Blood gases and the other biochemistry examination were within normal limits. Atenolol, digoxine, captopril, nitrates, forusemid, and LWMH, were administered at least one week before surgery. The patient was premedicated with oral Diazepam 10 mg the night before the surgery, and morfine sulfate 10 mg intramuscularly 30 minutes before the induction of anesthesia. In operating room after peripheral venous cannula was inserted, right radial artery and right internal jugular vein were cannulated with a 20G arterial cannula and 7.5 F central venous catheter. A fast track anesthesia was made (Fentanyl 5 mcg/kg, Propofol 2 mg/kg, cis-atracurium 0.15 mg/kg). Monitoring included invasive arterial monitoring, central venous pressure, ECG, temperature, urine output, and end-
Tidal carbon dioxide. The anesthesia was maintained with sevoflurane, propofol infusion, fentanyl and cis-Atropine as needed. Priority was given to pulmonary hypertension, mitral regurgitation and possibly right cardiac failure. There was performed mild hyperoxigenation, infusion of millrinone and adrenaline, small bolus doses of phenylephrine and nitroglycerine. The patient was discharged uneventful on 8-th postoperative day, being advised for surgical correction of mitral and aortic valve diseases.

3. Discussion

3.1. Preoperative Optimization

The pulmonary artery pressures vary from 18-25 mmHg (systolic) and 6-10 mmHg (diastolic). The pressure over 25 mmHg at rest or more than 30 mmHg with exercise is considered to be PH. PH can be usually presented with difficulties in breathing, weakness, fatigue, and rhythm disturbances. Chest X-ray shows prominent pulmonary artery, whereas ECG (4) presents right axis deviation. Pulmonary function test can reveal the respiratory origin of PH. Echocardiography (5, 6) may be helpful in determination of valve malfunctions, right atrial enlargement, elevated pulmonary pressure, and tricuspid regurgitation. The pathophysiology (4-6) changes are complex. Right ventricle ejects in pulmonary bed which is characterized by one tenth of systemic pressure. The pulmonary vasoconstriction can increase right ventricle pressure and decreases left ventricular filling resulting in low cardiac output. The increasing right ventricle afterload can precipitate ventricular failure. Right ventricle enlargement results in annular dilatation of tricuspid valve and tricuspid regurgitation. Tricuspid regurgitation can further increase right ventricle wall stress, resulting in myocardial ischemia and infarction. General therapy’s goals are correction/prevention of hypoxia, hypercapnia, and acidosis. Maintaining right ventricular function by increasing its inotropism and by decreasing afterload is the principal goal of the anesthetic practice. Digoxine, calcium channel blockers, diuretics, and low molecular weight heparin are the commonest used drugs in the preoperative period.

3.2. Anesthetic Care

After preoperative evaluation and preparation, the patient is scheduled for surgery. Arterial line, central line, pulmonary artery catheter, and occasionally transesophageal echocardiography are mandatory. Besides of anesthetic technique (general or regional anesthesia), ensuring oxygenation and ventilation remain the principal goals. The anesthesiologist must beware of negative effects of inhalator drugs on cardiovascular system. Using large opioid doses can minimize volatile anesthetics. All the situations that can increase right ventricle afterload must be avoided. Prophylactic low dose of dobutamine or millrinone combined or/not with norarenaline may be helpful. The anesthesiologist must avoid hypoxemia, hypercarbia, acidosis, hypovolemia, and increasing afterload of right ventricle. Adequate anesthesia’s depth must be ensured. Table 1 summarizes the drugs usually helpful for PH.

### Table 1. The common drugs used for PH.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine</td>
<td>5-10 mcg/kg/minute</td>
</tr>
<tr>
<td>Millrinone</td>
<td>50mcg/kg bolus, 075mcg/kg/min infusion</td>
</tr>
<tr>
<td>Dopamine</td>
<td>5-10 mcg/kg/minute</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>2-20 mg/minute</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td>2-20 mcg/minute</td>
</tr>
<tr>
<td>NO</td>
<td>20-40 ppm</td>
</tr>
<tr>
<td>Prostacicine</td>
<td>4-10 ng/kg</td>
</tr>
</tbody>
</table>

4. Medical Therapy

Medical treatment includes intravenous and volatile vasodilator drugs. Intravenous vasodilators are milrinone, dobutamine, dipiradomole, calcium channel blockers, and nitroglycerine. Volatile drugs include prostaglandins (prostacycline, epoprostenol, treprostinil, iloprost), and nitric oxide. Oral pulmonary vasodilators are presented by endotheline receptors antagonists (bosentan), and Viagra (sildenafil). Milrinone reduces systemic and pulmonary pressure, while increasing right ventricle inotropism. Exaggerated systemic hypotension presents an eminent risk for further deterioration of right ventricle function and ischemia. The combination of milrinone and vasopressors as noradrenaline or phenylephrine, can minimize this side effect (7). Calcium channel blockers reduces right ventricle afterload by smooth musculature releasing. Nitric oxide produces pulmonary vasodilatation without systemic hypotension through cyclic guanosine monophosphate (8). Prostacycline is available in both forms: inhaled and intravenous. Prostacycline and other prostaglandins (9) decrease pulmonary pressure, right atrial pressure. It is recently reported that combination with nitric oxide is more effective than alone therapy (10). Levosimendan is a new inotropc drug, increasing inotropy of right ventricle mediated induced calcium sensitized cells activation.

The principal anesthetic care issues (11) remain reducing right ventricle afterload (vasodilators, deep anesthesia), maintaining its inotropism (inotropes and regional anesthesia if available), and avoiding all deteriorating situations (hypoxia, hypercapnia, acidosis, light general anesthesia).

5. Conclusions

Several problems are faced by anesthesiologist managing a patient suffering from PH undergoing to non cardiac surgery. Prevention of exacerbation of PH, and right heart failure are the crucial steps that can improve the patient’s outcome.

References


