

Emergency caesarean section delivery and puerperium in a patient with severe idiopathic pulmonary arterial hypertension — a case report

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Dear Editor,

We would like to present a case report of a 30-year-old primigravida with idiopathic pulmonary arterial hypertension who, while 31 weeks pregnant, was admitted to the department of cardiology due to a deteriorated general condition with the features of right ventricular failure, tachycardia, tachypnoea and low arterial blood oxygen saturation, as well as a respiratory infection. Although the treatment administered included passive oxygen therapy, inhalational sildenafil and iloprost, no noteworthy improvement was achieved.

CASE HISTORY

Due to genital tract bleeding, the patient was qualified for an emergency Caesarean section on hospitalisation day 5. The patient was transferred to the operating suite of the Department of Cardiac Surgery in cardiogenic shock with metabolic acidosis and respiratory failure. The Caesarean section was performed under general anaesthesia. A male newborn was delivered with a birth weight of 2,110 g and an Apgar score of 2/3/5/7. The intubated newborn was transferred to the Department of Intensive Care and Neonatal Pathology.

After the Caesarean section delivery, a Swan-Ganz catheter was inserted into the patient. The haemodynamic profile demonstrated a low cardiac index (CI: 1.7 L m⁻²), while the pulmonary artery pressure was comparable with the systemic pressure — 93/67 (mean 73) mm Hg. Levosimendan and inhalational nitric oxide were started and the infusion of epoprostenol was continued. Due to thrombocytopenia and bleeding to the airway, the dose of epoprostenol was not escalated. During the days which followed, cardiac output was normalised with a low stroke volume. Compensatory tachycardia, 130 beats per minute, was observed. The pul-

monary artery pressure remained at the level of the systemic pressure. The patient was mechanically ventilated with 45% oxygen in the respiratory mixture. Because of tachypnoea and poor tolerance of mechanical ventilation, the patient was sedated. Echocardiography still revealed a dramatically enlarged right ventricle, accompanied by limited filling of the left ventricle and severe tricuspid valve incompetence. No improvement in managing pulmonary arterial hypertension was achieved. Given progressive respiratory and circulatory failure, as well as the pulmonary artery pressure exceeding the systemic pressure, arteriovenous extracorporeal membrane oxygenation (AV ECMO) was decided upon on postoperative day 6. The right femoral vein and the left femoral artery were cannulated. ECMO therapy was uneventful until bleeding to the abdominal cavity occurred at the post-drain site. Haemorrhagic shock was diagnosed and massive transfusions were required. The site of bleeding was managed during an exploratory laparotomy. After surgery, the patient's clinical condition stabilised. A substantial amount of blood reoccurred in the drains on post-laparotomy day 2, which accelerated the decision of ECMO discontinuation after 7 days of support. Since the drainage persisted despite the withdrawal of anticoagulation, the patient required a re-laparotomy. The bleeding at the post-drain site was managed.

OUTCOME AND FOLLOW-UP

The further course of ICU treatment was uneventful. The haemodynamically stable patient was extubated on the day following the second laparotomy. On post-Caesarean section day 18, the patient, in general good condition and ambulated, was transferred to the department of cardiology for further treatment. After 20 days of hospitalisation in the cardiology department on combination therapy of pulmonary hypertension, the patient was discharged home in a satisfactory condition. ECHO performed prior to discharge demonstrated a decrease in the right ventricle cavity size and less severe tricuspid valve incompetence. At the same time, the newborn was discharged from the Department of Intensive Care and Neonatal Pathology. Regular rehabilitation and follow-ups in the ophthalmological outpatient clinic were recommended.

Pregnancy in women with pulmonary hypertension carries an extremely high risk of maternal mortality estimated in the range of 30–56%. Cardiologists strongly advise patients with pulmonary hypertension not to become pregnant; in cases of failed contraception, therapeutic abortion is recommended [1]. Physiological changes during pregnancy involve a 50% increase in circulating blood volume. Moreover, resting cardiac output increases due to increases in the stroke volume and heart rate [2]. To compensate for the increased blood flow, progesterone reduces systemic and

pulmonary vascular resistances. In women with pulmonary hypertension, the pulmonary vascular bed reacts to the increased blood flow by increasing the pulmonary artery pressure. This dangerous combination of increased cardiac output and pressure in the pulmonary vascular bed results in right ventricular failure [3].

The right ventricular systolic pressure in pregnant women with pulmonary hypertension is evaluated in three categories (30–50 mm Hg; 50–70 mm Hg; > 70 mm Hg). The right ventricular function is defined as normal, medium impaired or severe failure. The diagnostic tool is echocardiography. In 85–100% of cases, the delivery is preterm; atrophy is characteristic of 3–33% of foetuses and 7–13% of foetuses/newborns die. The highest mortality is observed in women with idiopathic pulmonary arterial hypertension and occurs within the first postpartum month [2].

Circulatory failure after delivery is caused by a reduction in preload due to bleeding, the quick return of vascular resistances to normal values and impaired systolic function of the right ventricle. The most common causes of maternal death during puerperium are right ventricular failure, arrhythmias or pulmonary embolism [4]. In idiopathic pulmonary hypertension, the disease affects the blood vessels. The dominant feature of the condition is vasoconstriction, which justifies the use of drugs reducing the tone of pulmonary vessel walls. The drugs applied include phosphodiesterase 5 inhibitors, endothelin receptor antagonists, prostacyclin analogues and guanyl cyclase stimulants. Phosphodiesterase 5 inhibitors (sildenafil, tadalafil) and inhalational, subcutaneous and intravenous prostacyclin analogues can be safely used in pregnancy. Intravenous epoprostenol is considered the drug of choice in pregnant women with pulmonary hypertension [4, 5].

There are no explicit recommendations regarding the methods of anaesthesia in women with pulmonary hypertension. The selection of anaesthesia for a Caesarean section is still under discussion because of insufficient material and no possibilities to compare the methods used over years due to advances in anaesthesia and the introduction of novel drugs. The priority is to avoid circulatory destabilisation during delivery. Scheduled Caesarean sections are performed most commonly to prevent emergency sections at night in haemodynamically unstable patients. When general anaesthesia is decided upon, it should be remembered that laryngoscopy and intubation provoke an increase in pulmonary artery pressure [6]. The recommended method of regional anaesthesia in haemodynamically stable patients is continuous epidural anaesthesia.

The most dangerous periods in pregnant women with pulmonary hypertension are the third trimester, delivery and puerperium. The highest mortality rate is noted during the first 10 postpartum days [7]. Treatment outcomes can

be improved thanks to strict intraoperative and puerperal monitoring. The basic tool for monitoring pulmonary hypertension is echocardiography. The use of the Swan-Ganz catheter is controversial. Instead, central venous pressure values can be recorded, which reliably reflect the changes in pulmonary artery pressures [8]. In our case, monitoring with the Swan-Ganz catheter was applied and the treatment was based not only on the pulmonary artery pressure but also cardiac output, systemic vascular resistances and saturation of venous mixed blood, which informed one in advance about the risk of haemodynamic destabilisation. An additional argument for using the Swan-Ganz catheter was the enormous experience of ICU physicians in this field.

The optimal dose of epoprostenol was not used due to thrombocytopenia and life-threatening bleeding. Epoprostenol is a potent inhibitor of platelet aggregation [7]. The patient twice required a laparotomy because of intraperitoneal haemorrhage from the site where the drain had been inserted during the Caesarean section. Immediately after the section, bleeding to the respiratory system was found, which was treated conservatively by discontinuing anticoagulants. Due to the AV ECMO applied, the patient received a continuous infusion of heparin, whose dose was modified. Since pregnancy creates the condition of hypercoagulation, we were concerned about the consequences of prolonged intervals in heparin infusion [9]. When the blood was observed in drains after the laparotomy, AV ECMO therapy was discontinued quicker than had been planned.

CONCLUSIONS

Pregnancy, delivery and puerperal care in patients with pulmonary hypertension should be managed by multidisciplinary teams consisting of cardiologists, pulmonary hypertension specialists, gynaecologists, anaesthetists experienced in obstetrics, neonatologists and intensive care specialists [10]. All of them should work in one hospital where extracorporeal therapies are available. When such teams and devices are lacking, things are left to chance or the goodwill of specialists and final outcomes depend on happy coincidences.

ACKNOWLEDGEMENTS

1. Source of funding: none.
2. Conflict of interest: none.

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Anaesthesiology Intensive Therapy
 2019, vol. 51, no 1, 72–74
 ISSN 1642–5758
 10.5603/AIT.a2019.0002
www.ait.viamedica.pl

Complications associated with nasotracheal intubation and proposal of simple countermeasure

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To the Editor,

Nasotracheal intubation is a widely used technique in anaesthesia management for procedures including oropharyngeal, dental, and maxillofacial surgeries [1–3]. It provides uninhibited access to the mouth and plays an important role when dealing with difficult airways [4–6]. It is also used in patients with cervical spine instability owing to injury [7] or in patients with a cervical spine fixation owing to disease or a previous operation [8]. Moreover, it is selected for patients who require prolonged intubation for intensive care [9]. However, nasotracheal intubation may lead to certain complications, with epistaxis being the most common. Epistaxis generally occurs due to damage to Kiesselbach's plexus in the anterior part of the nasal septum [10–12] where branches from several arteries, including branches of the ophthalmic, maxillary, and facial arteries, anastomose to form a vascular plexus. To avoid this complication, the tracheal tube should be inserted into the nasal cavity in such a manner that its bevel tip comes to the lateral side of the naris. However, if the bleeding occurs on insertion

of the tube, the nasotracheal intubation should be completed chiefly to protect the airway and also to tamponade the bleeding point. Risk of sinusitis is another disadvantage associated with nasotracheal intubation [13]. Sinusitis can induce oedema around the opening of the maxillary sinus. Mucosal oedema in the nasopharynx can also result in middle-ear problems. Superficial necrosis of the nasal ala is another common complication associated with nasotracheal intubation [3, 9, 14, 15]. Several measures have been suggested to avoid this necrosis problem [15–18]; however, these measures cannot always be applied in paediatric patients as their nares do not provide enough space for them. Nasotracheal intubation has also been reported to cause bacteraemia owing to abrasion of the nasal mucosa [19, 20]. Nasotracheal intubation-related carriage of bacteria into the trachea should be also avoided. It has been reported that prior treatment of the nostrils and anterior nasal septum with mupirocin is effective to avoid this complication [21, 22]. However, the cheapest and easiest countermeasure to avoid such a complication during a nasotracheal intubation for inducing anaesthesia involves the removal of nasal dirt from the tip of the tracheal tube; in short, the tracheal tube should be pulled out with the aid of a Magill forceps through the patient's mouth, while the dirt should be wiped away with a piece of clean cotton (Fig. 1). Additionally, dirt from the pharynx should be completely sucked out with the aid of a direct vision laryngoscope if required, before advancing the tracheal tube into the larynx. Once the tube tip and the pharynx are cleaned, the tube should be placed again into the oral cavity by pulling the proximal side of the tube near the patient's nostril. Subsequently, the tube tip can be advanced into the larynx with the aid of a Magill forceps. This series of treatment does not take longer than 10 seconds to