Brief Communication

Management of Iatrogenic Pulmonary Artery Injury during Pulmonary Artery Banding

Abstract

Pulmonary Artery banding (PAB) is limited to selected patients who cannot undergo primary repair due to complex anatomy, associated co-morbidities, as a part of staged univentricular palliation, and for preparing the left ventricle prior to an arterial switch operation. We report a catastrophic iatrogenic complication in which the pulmonary artery was injured during the PAB. We discuss its multi-pronged management.

Keywords: *Iatrogenic, pulmonary artery banding, pulmonary artery injury*

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The Editor,

In the current era, the indications of pulmonary artery banding (PAB) are limited to selected patients who cannot undergo primary repair due to complex anatomy, associated comorbidities, as a part of staged univentricular palliation, and for preparing the left ventricle before an arterial switch operation.[1] If no associated intracardiac procedure is needed, it can be safely performed without cardiopulmonary bypass (CPB) support. However, in the present era, due to the dwindling numbers of these procedures, surgeons and anesthesiologists have limited exposure in managing this operation and its complications. We report catastrophic iatrogenic complication in which the pulmonary artery (PA) was injured during the PAB. We discuss its multipronged management.

A 3-year-old female weighing 10 kg was diagnosed with complex congenital heart disease and pulmonary hypertension. As she was destined to undergo a final univentricular palliation, a PAB was planned.

Standard anesthesia protocols were adopted. At operation, the main PA was dissected free from the aorta. However, the basic precaution of looping the main pulmonary artery (MPA) by the subtraction technique[2] was omitted, instead a right-angled instrument to loop the MPA was used, resulting in PA perforation at two places, one at the point of entry and another at the point of exit of the instrument. This resulted in massive bleeding and exsanguination resulting in hypotension and cardiac arrest. Internal cardiac massage was instituted, and an unsuccessful attempt was made to repair the PA injury, due to its peculiar posterior location.

After systemic heparinization, emergency CPB was established by cannulating the aorta and the right atrial appendage, and the heart was allowed to beat at mild normothermia.

While preparations for instituting CPB were underway, more than 1 liter of Ringer lactate and three units of packed red blood cells were transfused. In addition, after heparinization, the autologous blood from the surgical field was obtained and rapidly transfused. Repeated boluses of epinephrine were also supplemented. While the perfusionist and the surgical team were making preparations for CPB that took approximately 12 min to accomplish, we were concerned about neuroprotection. To achieve the latter, the patient was placed in the Trendelenburg's position, and ice packs were placed on the head. After CPB was instituted, a few minutes of ventricular fibrillation were encountered followed by resumption of normal sinus rhythm. Injection thiopentone sodium (10 mg/kg) and injection methylprednisolone (30 mg/kg) were added to the pump. The temperature of the perfusate was allowed to drift to 32°C. Arterial blood gases,

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hematocrit, serum electrolytes, and blood glucose were maintained. However, the pupils were found to be dilated and fixed. The rent in the PA was repaired, and PAB was completed. Supportive CPB was continued for 103 min to ensure adequate systemic and brain perfusion and to ensure recovery of cardiac function. She was finally weaned off CPB on adrenaline and noradrenaline of 0.2 mcg/kg/min, dopamine and dobutamine of 20 mcg/kg/min each. After terminating CPB, pupils were found to be constricted with sluggish reaction to light. Postoperative recovery was uneventful, and she was discharged home on the 6th postoperative day with adequate band gradients and without any neurological deficit.

This case highlights the importance of having the perfusionist and CPB components ready in the operation room whenever dissection around great vessels is contemplated. This, however, does not argue for routine assembling of the pump in all closed-heart operations, but the anesthesiologists, perfusionist, and surgeons should be prepared for managing such an eventuality in the quickest possible time.

Residual neurocognitive deficits may occur and may increase postoperative morbidity, especially with massive blood loss leading to hypoxic brain injury. In such a scenario, timely volume infusion, judicious use of inotropes, and rapid institution of CPB are lifesaving and also minimize the risk of hypoxic brain injury. This should be combined with tight glycemic control, optimal hematocrit management, and pharmacological methods such as administration of steroids and thiopentone sodium, topical cooling of the brain by application of ice packs, maintenance of adequate perfusion pressures, ensuring adequate oxygenation, and avoidance of hypocarbia.[3]

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Conflicts of interest

There are no conflicts of interest.

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