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Case Report

Recurrent cardiac arrests due to severe aortic flow obstruction by aortic cannula: a fatal complication

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ABSTRACT

In neonates and children the blood vessels are small in calibre and the correct choice of arterial and venous cannulas together with accurate placement are crucial to avoid obstructing vascular branches and misdirecting flow or impairing venous drainage. Normally in children, to establish the cardiopulmonary bypass (CPB) one arterial cannula is inserted in the ascending aorta and single stage venous cannulae, where two cannulae inserted into the superior and inferior vena cava and joined by the Y-piece are used in the most open-heart surgeries to facilitate the circulatory, respiratory support along with the temperature management. Rarely aortic cannulation can lead to aortic dissection, aortic posterior wall rupture leading to trauma to the esophagus, dislodgement of the aortic atheroma, fatal bleeding leading even to cardiac arrest. In addition, SVC and IVC syndrome can also occur due to displacement or obstruction by the venous cannulae. However, repeated episodes of the cardiac arrests in children after successful weaning from CPB, even with the use of an appropriate size aortic cannula has not been reported in the literature to date. We wished to discuss this fatal complication after weaning from CPB, due to aortic flow obstruction by the aortic cannula in a one-year-old, 5.5 kg child following successful ventricular septal defect (VSD) repair. The institutional ethical committee clearance and clinical trial registrations are not applicable for this case report publication.

Keywords: Congenital heart disease, CPB, Aortic cannulation, Aortic obstruction, Cardiac arrest, TEE

INTRODUCTION

CPB circuits are essential for the repair of congenital heart defects in neonates and children. Single stage cannulae are used during most open-heart surgeries, where two cannulae are inserted in SVC and IVC and connected by Y-piece to establish CPB to divert the deoxygenated venous blood to the heart-lung machine for oxygenation and then back via the arterial cannula into the ascending aorta. Displacement of the venous cannulae can obstruct venous blood flow and may result with SVC and IVC syndrome.[1] Conversely, the aortic cannulation can cause complications like aortic dissection, aortic rupture, profuse bleeding and dislodgement of aortic atheroma resulting in systemic embolization.[2] We reported a rarer consequence of aortic cannulation, as a severe aortic blood flow obstruction resulting in recurrent episodes of the cardiac arrests in a child after successful VSD repair under CPB.

CASE REPORT

A one year old, 5.5 kg, with a BSA of 0.33 m^2, male child presented with hurried breathing since birth and recurrent

upper respiratory tract infection and easy fatiguability with decreased activity for the last three months of age. There was no history of cyanotic spells. On auscultation, a pansystolic murmur was present at the left fourth intercostal space. Chest X-ray showed pulmonary artery enlargement, cardiomegaly and increased pulmonary vascularity. Echocardiography revealed a large doubly committed VSD of 8.8 mm with moderate PAH, dilated RA, RV and pulmonary artery (2.6 cm) and normal biventricular functions with LVEF of 60% and TAPSE of 21 mm. He was put on oral digitalis, enalapril, sildenafil and furosemide and after obtaining informed consent from the parents, he was taken up for VSD patch closure under CPB. He was premedicated with oral midazolam (3 mg) two hours before surgery. In OR, standard ASA monitoring was started. His baseline heart rate was 140 bpm and arterial saturation was 99%. General anaesthesia was induced with fentanyl (50 mc), thiopentone sodium (5 mg), midazolam (0.5 mg) and vecuronium bromide (1 mg) was used to facilitate the endotracheal intubation with 4.5 mm cuff tube. After induction of anaesthesia, 20G Leadercath was inserted in the left femoral artery for continuous BP monitoring and intermittent ABG analysis. A 4.5 Fr triple lumen catheter was inserted via right internal jugular vein for CVP monitoring and administration of anaesthetic drugs and inodilators. His baseline BP and CVP were 85/50 mm Hg and 6 mmHg respectively and ABG revealed a pH 7.40, PO_2-176 mmHg, PCO_2-35 mmhg, Hb-11 gm%, HCO_3-25 mmol/l, SaO_2-99.8%. Anaesthesia was maintained with intermittent fentanyl, midazolam, vecuronium bromide and sevoflurane (1-2%) and oxygen in air with fraction of inspired oxygen (FiO_2) of 0.5-1. Anticoagulation with heparin (300 U/kg) was used to achieve an ACT of >480 Sec. Aortic cannulation was done using 12 Fr straight Styletted cannula (Medtronic), Venous drainage was achieved using single stage 14 Fr angled DLP cannula for SVC and 16 Fr angled DLP cannula for IVC. VSD was closed with Gore-Tex patch under standard moderate hypothermic CPB and potassium enriched cardioplegic (Den-Lido) myocardial protection. Weaning from CPB was easy with the use of infusion of milrinone (0.5 mc/kg/min), dobutamine (5 mc/kg/min) and NTG (1 mc/kg/min). On direct needle insertion, PA pressures were 22/7(10) mmHg, at cardioplegia line arterial pressure was 70/40 mmHg and at side port of the aortic cannula was 35/18 mmHg, as compared to femoral artery pressure of 30/17 mmHg. However, on visual assessment, the LV contractility deteriorated and progressed to distension and cardiac arrest and necessitated to reinstitute CPB. Patient had similar repeated three episodes of cardiac arrest on each successful weaning from CPB and LV contractility and hemodynamics could not be maintained even with the use of very high doses of inotropes. But we noted an unusual BP difference at proximal to aortic cannulation measured by direct needle insertion (90/40 mmHg) and femoral artery (30/17 mmHg). Therefore, it was decided to insert the TEE probe to assess the biventricular functions, rule out any other cardiac anomalies like extra VSD, mitral regurgitation, coartation of aorta and to assess the VSD patch closure, aortic flow obstruction and hemodynamics. TEE confirmed adequate VSD closure and absence of any other cardiac anomalies and good LV contractility while patient was on CPB support. Therefore, one more attempt for weaning off CPB was made under the TEE guidance. However, still there was a big difference between FA pressure and aortic pressure proximal to cannulation. Once again, the LV got distended and became almost akinetic. Consequently, the patient also developed severe mitral regurgitation with 2-3 MR jets and hemodynamic deterioration (Figure 1). Finally in a desperate scenario, it was decided to remove the aortic cannula even in severe hemodynamic instability (BP 31/17 mmHg), realizing that the aortic cannula might be the culprit for the deterioration of the LV function by obstructing the aortic blood flow. Following aortic decannulation, the LV contractility and hemodynamic improved gradually, (BP 90/42 mmHg) and maintained even with minimum infusion of dobutamine, milrinone (Figure 2). Utmost important to mention here that the similar sudden fall in blood pressure was also noted following aortic cannulation, however, that was managed as usual by volume administration through aortic cannula and promptly institution of CPB. Heparin was neutralized with protamine (1:1.3 ratio). Total CPB time was a sum of 188 min, 15 min, 22 min, 40 min and ischemia time was 140 min. Chest was closed after achieving proper haemostasis. His blood pressure and CVP before shifting to ICU were 90/45 mmHg and 7 mmHg respectively and ABG showed a Ph-7.47, HB-8.8 gm%, PCO_2-27, $PaO_2$244 mmHg and SaO_2-99%. Tracheal extubation was done on 2nd postoperative day and inodilators tapered slowly. Post-extubation, child was fully alert without any neurocognitive dysfunctions. Rest of the course was uneventful, and patient was discharged on 10th postoperative day.

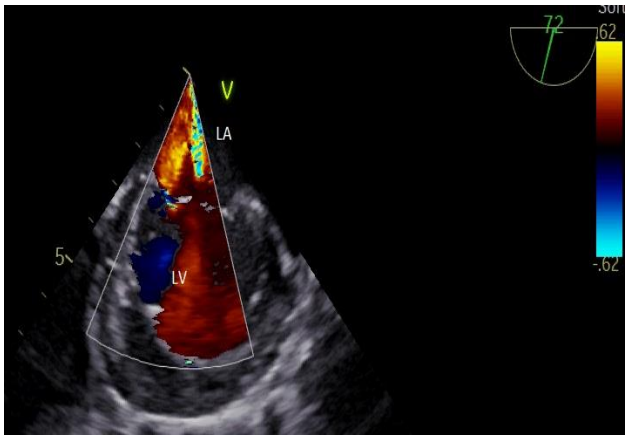

Figure 1: Mid esophageal 2 chamber TEE view (72 degree); color-Doppler revealed severe MR, with two jets reaching to the roof of the LA and LV was distended and hypokinetic (labelled).

MR-mitral regurgitation, LA-left atrium, LV-left ventricle.

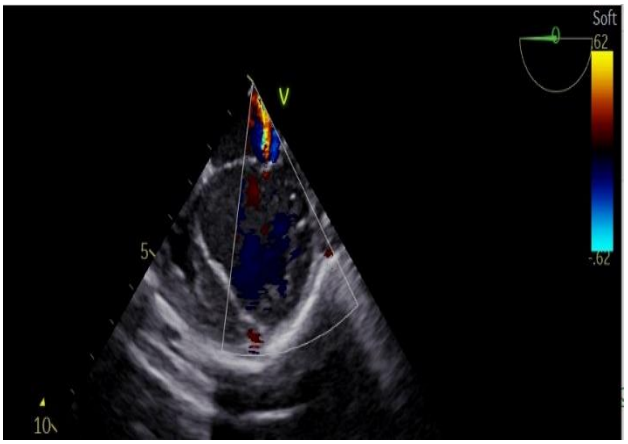

Figure 2: Modified mid esophageal 4-chamber TEE view (0 degree); color-Doppler revealed a mild MR after removal of the aortic cannula (labeled).

MR-mitral regurgitation, LA-left atrium, LV-left ventricle.

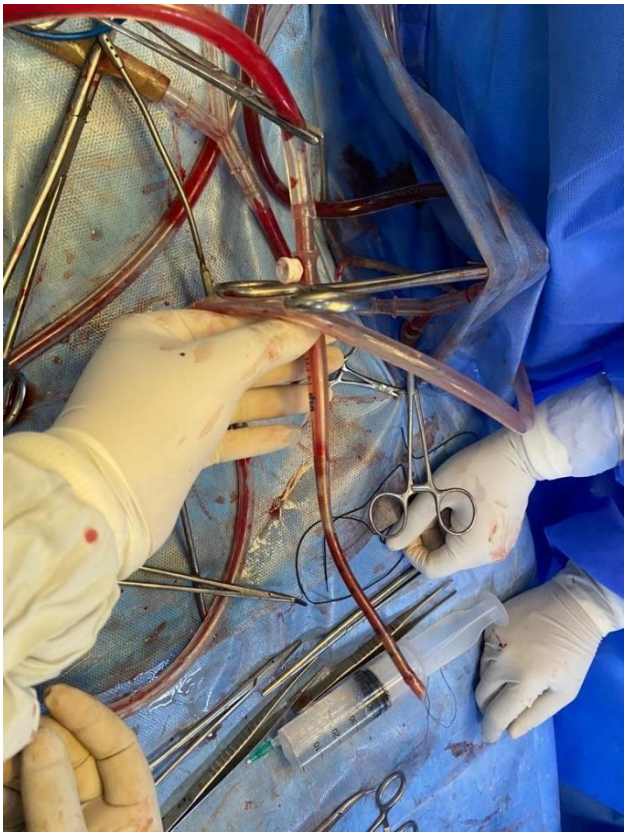

Figure 3: Intraoperative photograph shows the aortic cannula used during CPB, that caused the aortic blood flow obstruction and cardiac arrest.

DISCUSSION

CPB is a standard procedure in paediatric cardiac surgery.[3] The general principles of CPB are same for neonates and adults. It requires aortic and bicaval cannulations and some modifications are necessary to accommodate the multiple anatomical variations that may be encountered in congenital defects, that is, two aortic and three cava cannulas in associated interrupted aortic arch and a persistent left superior vena cava (LSVC) for adequate drainage during the CPB.[4] The aortic cannulation was primarily done at the distal ascending aorta, positioning the cannula close to the origin of the brachiocephalic trunk.[5] The appropriate arterial cannula size depended upon the required CPB flow, calculated using the formula,

CPB blood flow rate (litre/min)=body surface area (BSA) (m^2)×cardiac index (CI), (L m-2 /min).

Flows of 1.8 to 2.5 l/min/m^2 were commonly used for infants, children and adults during mild to moderate systemic hypothermia. The pump flow for this patient was calculated as weight in kg×150 ml/min=750ml/min and accordingly recommended aortic cannula of 12 Fr for 700-1000 ml flow was used (Figure 3). If a cannula was too large, it can obstruct native heart output, particularly in the ascending aortic position as this output was critical during cannulation and the initiation and weaning phases of bypass, also too large of a cannula may require an aortotomy that was difficult to close in a standard fashion. Therefore, aortic cannula size should be selected in conjunction with the perfusionist team to utilize the appropriate cannula for adequate flow during CPB. Table 1 shows general sizes of cannulas used for initiating CPB.

Table 1: Depicts general sizes of various venous and arterial cannulas used according to the weight of the patients for initiating CPB and to maintain optimum venous return and arterial flows.

Patient size (kg)	Arterial cannula (Fr)	Venous cannula (Fr)
2	8	8-10
3-6	10	10-12
6-8	12	14
8-16	14	17
16-30	17	19
30-40	17	21
>40	21	25

An arterial cannula that was too small, in addition to limiting flow, can cause high pressures gradient, cavitation, increased jet of flow velocities, jetting against the arterial wall and can increase the likelihood of dissection, and high shear forces which may damage the formed elements of the blood. However, diffusion-tip cannulas were available which provided multidirectional flow to reduce jets.[5] The tip of the cannula may be straight, tapered or angled as well as made from metal or plastic. Various tip modifications such as flanges or adjustable rings were available to prevent the cannula from being inserted too far into the aorta and impeding flow to the head vessels.[6] The inappropriately aortic cannulation can be associated with complications like

bleeding, aortic dissection, malposition of cannula tip, atheroma dislodgement causing systemic embolism, accidental decannulation, aortic posterior wall puncture causing fatal bleeding and esophageal damage.[7,8]

Our patient developed repeated LV distensions and cardiac arrests, even after repeated successful weaning from the CPB. Trans-oesophageal echocardiography (TEE) was used to confirm any unnoticed congenital anomalies like PDA, ASD, coartation of the aorta, extra VSD and VSD patch closure. After exclusion of the other possible causes of LV dysfunction and cardiac arrests, finally we reached to the decision that the repeated episodes of cardiac arrests were related to the rarer complication, the aortic blood flow obstruction by the aortic cannula. The LV dysfunction and cardiac arrests were even refractory to the very high doses of inotropes, inodilators and standard CPR. But at last, LV dysfunctions, MR and hemodynamic gradually improved only after aortic decannulation, and progressive improvement followed. The aortic blood flow obstruction by the aortic cannulation before institution, and after weaning from CPB in neonates and infants has been reported by some authors but no case of repeated cardiac arrests due to the LV distension and severe MR as a result of aortic blood flow obstruction by the cannula has been reported till date.[9] If it was suspected that a large aortic cannula was causing hypotension then a pressure gradient should be demonstrable across it, as it was observed in this patient also.

CONCLUSION

Aortic cannula size should be selected in conjunction with the perfusionist team to utilize the appropriate cannula for adequate flow during CPB. It is imperative to finally decide the aortic cannula size after observing the ascending aorta size after opening the chest. Even use of an appropriate size aortic cannula particularly in neonates and children can obstruct the aortic blood flow before and after CPB and patient may develop refractory LV distension and cardiac arrest.

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