Delayed Paraparesis: An Unusual Complication Following Coarctation of Aorta Repair

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ABSTRACT

Paraparesis following cardiac surgery is a manifestation of spinal cord injury (SCI). It can occur in any aortic surgery from the aneurysm to the coarctation of the aorta (CoA) where the cross-clamp of the aorta is applied. Though the incidence of paraplegia is low, its occurrence affects the morbidity and mortality of the patient. There are only sporadic case reports on the development of paraplegia following recurrent and technically challenging repair of CoA. However, the spontaneous development of paraplegia has also been reported in cases of unoperated CoA. The present report describes the case of delayed SCI in which paraparesis developed 5 days post a coarctation repair. The risk factors and strategies to protect the spinal cord during aortic surgeries are emphasized.

Keywords: Coarctation, paraparesis, spinal cord injury

INTRODUCTION

The incidence of spinal cord injury (SCI) in cardiac surgery ranges as high as 6.3% in descending thoracic aneurysm (DTA) surgery after implementing spinal cord protection strategies[1] and 0.4–1.5% following coarctation of the aorta (CoA) repair.[2] This divergence is evident in uncomplicated aortic coarctation where the extent of resection is limited to the isthmus, shorter cross-clamp time, and well-developed collateral circulation in contrast to aortic aneurysm surgeries. So, the emphasis on protecting the spinal cord from ischemic injuries should be in cases of expected prolonged cross-clamp time, surgical complexity, and poor collateralization.

Here, we report a case of delayed SCI following the CoA, patent ductus arteriosus (PDA), and ventricular septal defect (VSD) for corrective surgery.

CASE HISTORY

A 6-year-old male patient was admitted to our institute, with the diagnosis of CoA with VSD and PDA. On examination, the patient was conscious and cooperative, neurologically intact, with a blood pressure of 110/70 mmHg in the right upper limb and 70/40 mmHg in the lower limb, and with a pan systolic murmur in the apical area. Pre-operative transthoracic echocardiography showed peri-membranous VSD of 8 mm, CoA, and PDA distal to the left subclavian artery (LSCA).

The patient was planned for left postero-lateral thoracotomy and repair of the CoA with PDA ligation, followed by median sternotomy and pericardial patch closure of VSD. After connecting the standard monitors and securing intravenous lines, the patient was induced with fentanyl, ketamine, and vecuronium. The airway was secured with

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a tracheal tube, and the right internal jugular vein, right radial, and left femoral arteries were cannulated. Rt radial pressure was 116/68 and femoral pressure 80/44 mmHg, with a systolic gradient of 36 mmHg. Thereafter the patient was positioned for left thoracotomy. A 5 mm PDA was identified just distal to the coarcted segment of the aorta. It was ligated with a 2-0 ethibond suture, and the intercostal arteries were identified but were not dissected, looped, or divided. There was no unusual collateral communication across the coarcted segment of the aorta. Injection unfractionated heparin was given systemically @ 1 mg/kg, and activated clotting time reached 300 s. Now proximal descending thoracic aorta was clamped just distal to the origin of LSCA. DTA was also clamped a few millimeters away from the CoA segment, the coarcted segment of the aorta was excised, and an end-to-end anastomosis of the two ends of the apparently healthy lumen of the aorta was anastomosed with 3/8 5-0 polypropylene sutures in a continuous fashion. The aortic clamp time was 35 min. After achieving the hemostasis, the chest wall was closed in a standard manner. During the repair, the radial artery pressure was 128/70 mmHg and the femoral mean pressure was 25 mmHg. The nasopharyngeal temperature was maintained at around 34°C with the aid of a temperature controller machine blanket. Post-repair the gradient reduced from 36 to 5 mmHg across the anastomosis. The patient was repositioned from lateral to supine for median sternotomy. After adequate heparinization, the patient was subjected to cardiopulmonary bypass (CPB), and pericardial patch closure of VSD was performed in a cardioplegic arrested heart with the cross-clamp 46 min and CPB time of 92 min. The patient was weaned off CPB with minimal inotropic support.

The patient was extubated on postoperative day 1 (POD 1) with no neurological deficit. Bilateral lower limb weakness was noticed on POD 5. There was no sensory loss based on the pinprick method nor the involvement of the bowel and bladder. On examination, pupils were bilateral equal reacting to light, no facial or bulbar weakness, and hypotonia with the power of 3/5, in both lower limbs. The knee jerk was normal with sluggish ankle jerk and mute plantar reflex in both lower limbs. Magnetic resonance imaging (MRI) showed post-contrast enhancement along cauda equine nerve roots suggestive of ischemic insult. After a course of steroids (methylprednisolone 30 mg/kg for 3 days) and physiotherapy, the patient's tone and power started improving gradually and the patient started to walk with support on discharge.

DISCUSSION

The incidence of paraplegia following aortic aneurysm surgeries is significantly reduced due to a better understanding of the pathophysiology of SCI and its preventive measures. Whereas in coarctation repair though the incidence is already low, its existence cannot be denied and this imposes significant morbidity.

The mechanism attributed to the SCI is mainly the duration and degree of ischemia,[1] a delayed manifestation of SCI which may occur at any time between 1 and 21 days following surgery due to biochemically mediated reperfusion injury.[3] Generally, in coarctation surgeries, the duration of ischemia is limited by short cross-clamp time, and the degree of ischemia is counteracted by well-developed collaterals. Yet still, its occurrence following surgery and even spontaneous neurological deficit in coarctation of aorta patients mandates a further understanding of the mechanism in this particular cohort. Intercostal artery aneurysm compressing the anterior spinal artery or rupture into the vertebral canal is the postulated mechanism for the spontaneous occurrence of neurological deficit in a coarctation patient.[4] Either of these was not seen in the present case as evidenced by MRI findings.

The existence of a PDA in addition to a coarctation and recurrent coarctation repair increases the risk of paraplegia.[3] The present case had PDA and the clamp time was prolonged around 35 min due to unexpected surgical difficulties. Although Brewer *et al*.[5] suggested that clamping time is not associated with the occurrence of paraplegia, it should be interpreted cautiously in the context of the adequacy of collateralization. However, because of the anatomical heterogeneity in blood flow, even while the collateral circulation is adequate, the radicular artery may not be sufficient to allow even the shortest length of cross-clamping.[6]

Although we maintained the distal pressure at 25 mmHg during aortic clamping, various literature indicates that in mixed populations, measures to improve perfusion pressure or shorten clamp time to 20 min may prevent SCI if the distal aortic pressure is less than 40–60 mmHg,[7] whereas in pediatric cohorts, the minimum acceptable distal aortic pressure is still unknown. Regional oxygen saturation (rso2) monitoring during clamping might be helpful in identifying acceptable pressures to minimize ischemia.

Left heart bypass surgery and jump grafting[8] are the other preventative procedures used in these conditions to combat ischemia. Yet again difficulties in the prediction of those variations in blood supply or unexpected prolongation in clamp time limit the planning of these protective measures.

Hypothermia can increase the tolerance to ischemia by decreasing oxygen consumption and the metabolic rate

and it has been shown to have a protective effect on the spinal cord during clamping. On the other hand, deep hypothermia necessitates bypass, prolonging the surgical time and coagulopathy.[9] We maintained mild hypothermia to balance both issues.

Pharmacological interventions aim to decrease the metabolic requirements of the spinal cord or to decrease the inflammatory responses to ischemia or reperfusion. Steroids, naloxone, and *N*-methyl-D-aspartate antagonism by riluzole, memantine, and magnesium have shown benefits in animal studies in reducing the effects of SCI and are reported in some series as components of multimodal therapies.[10] Other interventions, such as reducing the toxic metabolites by superoxide dismutase, allopurinol, and deferoxamine, remote ischemic preconditioning,[11] may show promise in the future. In the present case, methylprednisolone 30 mg/kg once daily for 3 days was started once we recognized the complication.

So, monitoring during intraoperative and continued serial post-op neurological monitoring is vital in the early recognition so that protective measures can be undertaken. Traditionally, functional monitoring like somatosensory evoked potential or motor evoked potential is used to detect ischemic changes.[12] Metabolic includes cerebrospinal fluid (CSF) analysis for lactate, S-100, neuron-specific enolase, and glial fibrillary acidic protein;[13] physiological that includes lumbar CSF pressure, paravertebral muscle oximetry,[14] a polarographic technique for spinal cord oxygenation[15] are the other methods to monitor. After surgery, clinical monitoring of lower limb motor and sensory function, in addition to bowel and bladder continence, is imperative and may indicate a need for augmentation of blood pressure or lumbar CSF drainage depending on the circumstances.

In the present case, weakness of both lower limbs occurred as late as the fifth POD indicating the delayed SCI. The causative factor for delayed SCI could be (1) increased clamp time, (2) decreased collateralization, (3) association of PDA, and (4) VSD repair that mandated the patient to be subjected to CPB compounding the risk of ischemic reperfusion phenomenon and possibly an embolic phenomenon.

CONCLUSION

A delayed paraparesis post-coarctation of aorta repair increases the morbidity and early recognition by serial neurological examination and the use of computed tomography or MRI with timely management had a favorable outcome in the present case.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initial s will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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