Case Reports

Adjunctive Ischemic Lower-Limb Perfusion during Aortic Repair

with Cardiopulmonary Bypass Prevents Fatal Reperfusion Injury

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Acute aortic dissection can be complicated by malperfusion syndromes, including ischemia of the lower limbs. In some cases, delayed correction of leg ischemia leads to reperfusion injury, potentially resulting in renal failure. We describe the case of a 64-year-old woman who presented with acute aortic dissection manifesting itself as lower-limb ischemia. During and after aortic surgery with cardiopulmonary bypass, the patient developed myonephropathic metabolic syndrome. Hyperkalemia was corrected and acute kidney injury was prevented by infusing large volumes of intravenous fluids and administering human atrial natriuretic peptide. Peripheral bypass surgery was unnecessary. This case suggests that restoring blood flow to an ischemic leg by means of adjunctive perfusion during aortic repair with cardiopulmonary bypass is a viable way to overcome the biochemical instability associated with prolonged ischemia, especially hyperkalemia in the early phase of reperfusion. ***(Tex Heart Inst J 2019;46(2):130-2)***

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Acute aortic dissection can impair or stop blood flow to the lower limbs.[1,2] In rare cases, delayed correction of the ischemia can lead to reperfusion injury.[3-7] Myonephropathic metabolic syndrome (MNMS) is characterized by the influx of harmful metabolites into the systemic circulation after reperfusion of the ischemic limbs. Restoring blood flow to the legs has been protective against this syndrome; however, management protocols have not been well established. Investigators fail to agree on the timing of initiating leg perfusion and the necessity of cardiopulmonary bypass (CPB) support.[3-7] We describe the case of a patient in whom MNMS developed during and after replacement of the ascending aorta, and our use of CPB to prevent reperfusion injury.

Case Report

A 64-year-old woman with a medical history of hypertension and hyperlipidemia experienced sudden pain and numbness in her left lower leg and was diagnosed with acute aortic dissection. She was transferred to our hospital, where she presented with left-lower-leg ischemia manifesting itself as absent femoral pulse, paresthesia, and paralysis. Chest radiographs showed a mildly enlarged superior mediastinal shadow (cardiothoracic ratio, 56%). Computed tomograms revealed aortic dissection from the ascending aorta to the terminal branches of the abdominal aorta, with a partially thrombosed false lumen (Fig. 1A) and occluded right renal and left common iliac arteries (Fig. 1B). The primary entry tear appeared to be in the ascending aorta.

At this point, MNMS was a major concern; consequent to the time taken to transfer the patient, the estimated time for reperfusion of the ischemic leg was >6 hours. We planned to do the following: 1) concomitantly restore blood flow to the ischemic leg and apply systemic perfusion through CPB; 2) perform peripheral artery bypass surgery if ischemia persisted after aortic repair; 3) establish ultrafiltration to treat hyperkalemia during CPB; and 4) prevent acute kidney injury by infusing large volumes of intravenous fluids and administering human atrial natriuretic peptide[3] (hANP) (Daiichi Sankyo Co., Ltd.) with adjunctive hemodialysis, if necessary.

The right axillary and left femoral arteries were simultaneously prepared for vascular access. A conventional median sternotomy was performed, and a small amount of

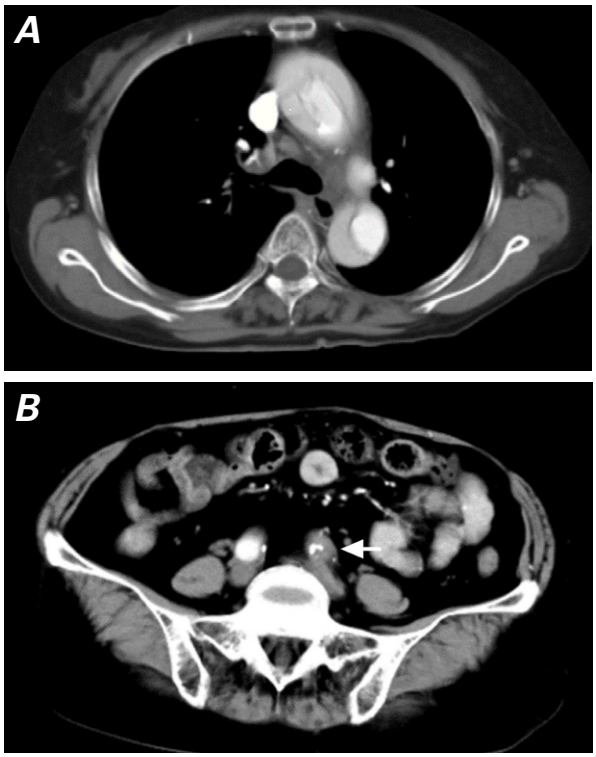

Fig. 1 *Preoperative computed tomograms show* ***A****) type A aortic dissection with* ***B****) contrast medium in the right but not the left (arrow) common iliac artery.*

nonhemorrhagic fluid was drained from the pericardial space. After vascular grafts were sutured to the right axillary and left femoral arteries in an end-to-side fashion,[8] CPB was established with use of these arteries for arterial perfusion; a 2-stage cannula was used for atrial drainage. Left femoral artery pressure was monitored only during CPB, to ensure adequate perfusion to the left leg. Blood flow to the patient's lower leg was restored >8 hours after presentation.

Antegrade and retrograde infusions of cold-blood cardioplegic solutions were used for myocardial protection. After systemic hypothermia was used to initiate circulatory arrest (rectal temperature, 21.6 °C), the aorta was opened. Selective antegrade cerebral perfusion was established through a vascular graft anastomosed to the right axillary artery, with perfusion catheters directly cannulated into the left common carotid and left subclavian arteries. The primary entry tear was identified in the ascending aorta. Replacement of the ascending aorta was performed with use of a 26-mm J graft (Japan Lifeline Co., Ltd.). Because femoral artery pulses were palpable bilaterally after the patient was weaned from CPB, peripheral bypass surgery was deemed unnecessary.

The patient's serum potassium level reached a peak of 7.5 mmol/L immediately after initiation of CPB. Ultrafiltration with infusion of 7,300 mL of saline solution decreased her potassium level to approximately 6 mmol/L. Metabolic acidosis with a minimal base excess of –6.2 mmol/L was corrected by infusing sodium bicarbonate. In the intensive care unit, hANP was administered at 0.04 μg/kg/min until day 2, then decreased to 0.02 μg/kg/min and discontinued on day 3. Intravenous fluids were infused at rates of approximately 400, 200, and 100 mL/hr on days 1, 2, and 3, respectively. The patient's urinary output was approximately 4,000 mL/d during the 4 days after the procedure. She was weaned from the respirator on day 4. Her creatine kinase levels increased markedly (peak level, 77,692 IU/L) 24 hours after surgery. Her renal function was slightly affected (peak creatinine level, 159 μmol/L on day 5); however, hemodialysis was not necessary. The patient's electrolyte levels were maintained within normal ranges after she was transferred to the intensive care unit. Thereafter, she had an uneventful postoperative course and was discharged to a rehabilitation hospital with mild palsy of her left lower leg and no sensory disturbance. Computed tomograms confirmed patency of her bilateral common iliac arteries.

Discussion

Our experience demonstrates that restoring blood flow to an ischemic leg by means of adjunctive perfusion concomitantly with systemic perfusion by CPB can improve the clinical outcomes of patients who present with MNMS during or after aortic surgery.

Iliofemoral malperfusion has been reported to occur in 9.7% to 15.2% of cases of acute type A aortic dissection.[1,2] Although no adverse events occur when blood flow is successfully restored early, MNMS resulting from rhabdomyolysis can develop when reperfusion is delayed.[3-7] Because this syndrome results from the influx of harmful metabolites into the systemic circulation after reperfusion, potential death is a concern even in the early phase of reperfusion. Peripheral revascularization without CPB support can be fatal when MNMS occurs; our experience suggests that adjunctive perfusion of the ischemic limb during CPB support, followed by aortic repair, can reduce the risk of death.

We easily corrected our patient's critical hyperkalemia that peaked immediately after reperfusion. Her serum potassium would probably have reached even higher levels without the dilution effect related to the priming volume of CPB. In a previous case of bilateral leg ischemia, the authors reported perfusing one leg with CPB support and the other leg without CPB support.[4] Potassium levels after the reperfusion were well controlled with CPB support but not controlled at all without CPB support. The patient survived this condition after perfusion was discontinued and the ischemic limb was amputated.[4] This result clearly supports the approach that we have described.

For perfusion, we chose to cannulate the graft sewn onto the artery, instead of direct cannulation, to avoid arterial injury and to ensure peripheral circulation.[8] This practice enables peripheral bypass from the contralateral femoral artery without arterial clamping, when necessary.

The efficacy of hANP in the treatment of MNMS has not been established. Sezai and colleagues[3] noted that hANP eliminates myoglobin deposition and reported a case of MNMS in which hemodialysis was not necessary. Although the role of this peptide in maintaining renal function is unclear, our experience suggests that it has adequate diuretic effect.

We conclude that restoring the blood flow to ischemic legs by means of adjunctive perfusion concomitantly with CPB support, followed by aortic repair, can minimize the risks associated with MNMS when prolonged reperfusion is a concern.

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