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CLINICAL REPORT

Hypothermic cardiopulmonary bypass for minimally invasive mitral valve plasty in adult moyamoya disease

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Abstract A 43-year-old man underwent minimally invasive mitral valve plasty of a flail mitral valve. Four years previously, he had been diagnosed with moyamoya disease (MMD) by cerebral magnetic resonance imaging/angiography findings. In MMD, risk factors for cerebral stroke include changes in arterial carbon dioxide partial pressure, blood pressure, and body temperature. And during cardiopulmonary bypass (CPB), these hemodynamic changes can be challenging. However, hypothermia during CPB can decrease cerebral oxygen consumption and have a cerebral protective effect. We performed a minimally invasive mitral valve plasty, using hypothermic CPB, in a patient with MMD, without any neurological deficits.

Keywords Hypothermia · Cardiopulmonary bypass · Moyamoya disease

Introduction

Moyamoya disease (MMD) is a chronic cerebrovascular disease involving stenosis or occlusion of the terminal portion of the internal carotid arteries [1]. Clinically, children with MMD present with ischemic attacks, and adults present with either ischemic or hemorrhagic events. In MMD, risk factors for ischemia include hypocapnia, hypotension, hypovolemia, and hypothermia [2–6]. During cardiopulmonary bypass (CPB), these hemodynamic changes can be challenging. Thus, maintaining a normothermic state contributes to prevention of hypoperfusion of the brain. However, hypothermia during CPB can decrease cerebral oxygen consumption and have a cerebral protective effect. We report hypothermic CPB for minimally invasive mitral valve plasty performed in a patient with MMD.

Case report

A 43-year-old man was admitted to the hospital with exertional dyspnea and palpitation. Physical examination revealed a grade 4 systolic murmur in the mitral area. Transthoracic echocardiography showed severe eccentric mitral regurgitation (IV/IV) due to a flail mitral valve (P2 pathology). He was referred for minimally invasive cardiac surgery.

He had a history of seizure events and anticonvulsive medication 4 years previously. MMD was diagnosed by cerebral magnetic resonance imaging/angiography and transfemoral cerebral angiography, which revealed chronic occlusion of the right distal internal carotid artery and collateral vessels (Fig. 1). Brain single-photon emission computed tomography showed a mild perfusion decrease in the right cerebral cortex with normal vascular reserve state.

After premedication of glycopyrrolate 0.2 mg, anesthesia was induced with etomidate 15 mg and rocuronium 50 mg intravenously and was maintained with sevoflurane and remifentanil. The radial arterial pressure, capnograms, bispectral index, central venous pressure, body temperature, EKG, oxygen saturation, and cerebral oximetry (INVOS

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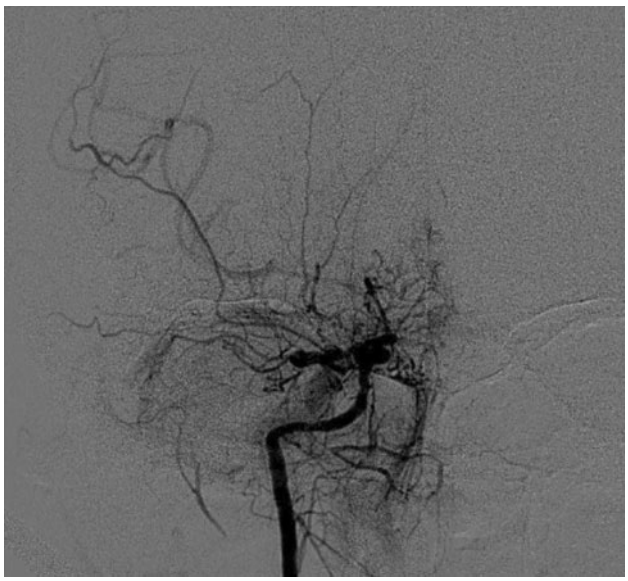

Fig. 1 Occlusion of the right distal internal carotid artery (ICA) on transfemoral cerebral angiography

5100; Somanetics, Troy, MI, USA) were continuously monitored throughout the surgery. Minimally invasive cardiac surgery was performed via a right anterolateral minithoracotomy and a 6-cm incision at the 4th intercostal space.

By serial arterial blood gas analysis, arterial carbon dioxide partial pressure was maintained at 35–45 mmHg, and arterial oxygen tension was maintained at 100–350 mmHg. The total operation time was 3 h 30 min and total CPB duration was 1 h 18 min. The patient presented in the operating room with an esophageal temperature of 37.2°C. His temperature was maintained at 37–37.2°C in the prebypass period, and was reduced to as low as 28.9°C during the bypass. The temperature was raised above 36°C in the postbypass period. Cerebral regional oxygen saturation (rSO_2) was 75–81% in the prebypass period, 70–76% during the bypass period, and 74% in the postbypass period. Fluctuation of rSO_2 values was within 15% (Table 1). The radial mean arterial pressure was constantly kept above 70 mmHg during the bypass. On completion of the operation, the patient was transferred to the intensive care unit (ICU), intubated, and ventilated.

Three hours after the operation, there was one tonic-type seizure event. Cerebral magnetic resonance imaging showed no abnormal findings. Seven days later, he was discharged from the hospital without any complaints. No other cerebral ischemic episodes were noted.

Discussion

In patients with MMD vasoconstriction in the moyamoya vessels causes perioperative cerebral ischemia. Thus,

Table 1 Series of regional cerebral O_2 saturation

	T basal	T-30	B-0	B-15	B-30	B-45	PB-15
rSO_2 (left, %)	81	75	74	70	73	73	74
rSO_2 (right, %)	80	79	76	73	75	76	80

rSO_2 Regional cerebral O_2 saturation, *T basal* before anesthesia induction, *T-30* 30 min after induction, *B-0* bypass start, *B-15* 15 min after bypass start, *B-30* 30 min after bypass start, *B-45* 45 min after bypass start, *PB–15* min after bypass weaning

hypocapnia is reported to be a major risk factor for the occurrence of stroke in MMD. Furthermore, hypercapnia induces cerebral steal from the collateral vessels to the normal vessels, and this can cause ischemia in MMD [2–6]. Therefore, normocapnia was maintained throughout the surgery in our patient, and normotension was maintained for the preservation of perioperative cerebral perfusion pressure.

To control mean arterial pressure, we did not use a vasoconstrictor. Instead, we employed a perfusionist to control the flow rate, and if mean arterial pressure rose, we infused nimodipine. This agent has a selective effect on the cerebral vessels [7].

Body temperature also contributes to perioperative cerebral ischemia. Hypothermia is related to the development of ischemic complications in patients with MMD, because it increases the risk of a stroke, as the result of constriction of the collateral vessels [6]. And CPB is a high-risk procedure for patients with MMD, due to decreased cerebral perfusion and non-pulsatile flow. There have been some case reports of normothermic CPB surgery in patients with MMD [8–12]. However, hypothermia during CPB can decrease cerebral oxygen consumption and have a cerebral protective effect [13, 14]. Decrease of the core temperature can reduce cell injury by suppressing excitotoxins and oxygen radicals, stabilizing cell membranes, and reducing the number of abnormal electrical depolarizations. Few cases of cardiac surgery employing hypothermic CPB in MMD have been reported;one such case was the repair of an atrial septal defect in pediatric MMD [15]. Another was the case of a 33-year-old man with MMD undergoing the repair of an aortic coarctation [16]; thiopental infusion and a pH–stat strategy were used and there were no neurologic sequelae. But our strategies during CPB were based on an α-stat strategy with the close monitoring of cerebral tissue oxygen saturation. The monitoring of cerebral tissue oxygen saturation during CPB allows the detection of clinically important cerebral desaturations and can help to predict perioperative neurological sequelae. We aimed to maintain cerebral saturation at 75% of the baseline value by following a specified treatment algorithm (Table 2). Theoretically, hypothermia can decrease cerebral oxygen

Table 2 Cerebral desaturation treatment algorithm

1.	Increase inspired oxygen to 100%
2.	Check head and cannula position to ensure adequate venous drainage
3.	If $PaCO_2$ <40 mmHg, increase $PaCO_2$ to >40 mmHg
4.	If mean arterial pressure (MAP) <50, increase MAP to >60 mmHg
5.	If hematocrit <20%, transfuse packed red blood cells (PRBCs)
6.	If none of the above interventions improve cerebral saturation, decrease cerebral oxygen consumption by increasing anesthesia depth

consumption and should increase rSO_2. But in our patient, the rSO_2 decreased during the hypothermic CPB (Table 1). We suspected that one of the reasons for this decrease was that we used the α-stat strategy; this strategy can induce hypocapnia and this is surely the main cause of decreased cerebral oxygen saturation. However, as the pH–stat strategy can induce hypercapnia, which is surely the main cause of the cerebral steal phenomenon, we conclude that the pH–stat strategy cannot be more beneficial than the α-stat strategy in MMD.

In summary, we were able to perform a minimally invasive mitral valve plasty in a patient with MMD without any neurological deficits, using hypothermic CPB based on the α-stat strategy. The patient benefited from the cerebral protective effects while ischemic complications were avoided by our use of a specific treatment algorithm in response to decreases in cerebral tissue oxygen saturation.

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