An induced mild hypothermia for an arrested patient during aortic valve replacement

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Abstract

A patient arrested during anesthesia was successfully managed with induced mild hypothermia and cardiopulmonary bypass (CPB). A woman, aged around 70 years, who had been diagnosed with severe aortic stenosis was scheduled to undergo aortic valve replacement. Soon after sternotomy, sudden bradycardia and low blood pressure developed, leading to ventricular fibrillation. Both CPB and systemic hypothermia were induced immediately. Low blood pressure and ventricular fibrillation continued for 10 min before CPB. The patient was cooled and maintained at target right tympanic and rectal temperatures of around 24°C during CPB. Aortic valve replacement proceeded uneventfully. She was weaned from CPB at right tympanic and rectal temperatures of 36.1 and 36.4°C, respectively. Postoperative induced hypothermia (target pulmonary artery temperature, 35°C) was applied over a period of 48 h with systemic natural cooling and ice packs around her head and neck. She was discharged to another hospital on post-operative day 19 with no neurological complications. Our experience suggests that induced hypothermia with systemic natural cooling and ice packs around the head and neck is a safe and feasible option for post-resuscitation intensive care, avoiding the need for emergency and intensive care teams.

Key words: aortic valve replacement, cardiac arrest, cardiopulmonary resuscitation, cardiopulmonary bypass, mild hypothermia

Introduction

Unpredictable cardiac arrest with widespread cerebral ischemia occasionally leads to catastrophic cerebral impairment during the perioperative period. Induced mild hypothermia appears to improve neurological outcomes after cardiopulmonary resuscitation in patients with out-of-hospital cardiac arrest.1,2 The optimal hypothermic management of in-hospital cardiac arrest, however, remains under investigation. We describe in-hospital cardiac arrest due to low blood pressure and ventricular fibrillation during aortic valve replacement that was treated using cardiopulmonary bypass (CPB) and induced mild hypothermia.

Case description

History

A woman, aged around 70 years, was transferred from another hospital to Kumamoto Chuo Hospital with the chief complaints of chest discomfort and fever. On admission, systolic blood pressure, diastolic blood pressure, and heart rate were 104 mmHg, 69 mmHg, and 104 bpm, respectively. She had been prescribed steroids for 3 months for idiopathic thrombocytopenic purpura. Infectious endocarditis was suspected and she was diagnosed with severe aortic stenosis (maximum aorta-left ventricular pressure gradient, 174 mmHg: aortic valve area, 0.4 cm²), although she had never experienced syncope. We
treated the infectious endocarditis with antibiotics and then scheduled her for aortic valve replacement surgery. Preoperative coronary computed tomography revealed no coronary artery stenosis. Severe left ventricular hypertrophy (interventricular septal thickness, 20 mm) was evident on echocardiogram. From a week before the surgery, the patient experienced discomfort and an unpleasant feeling in her chest almost everyday.

Anesthetic and operative management

The patient was classified as NYHA (New York Heart Association) functional classification III and ASA (American Society of Anesthesiologists) classification status P3. After premedication with scopolamine (0.2 mg) and establishing routine monitors of cardiac function (electrocardiography, pulse oximetry and invasive arterial blood pressure monitoring), anesthesia was induced with fentanyl (10 µg/kg) and diazepam (2.5 mg). Endotracheal intubation was facilitated with vecuronium (8 mg). Anesthesia was maintained with fentanyl (30 µg/kg) and infusions of propofol and vecuronium. Nicorandil (4 mg/h) was infused intravenously during surgery and intravenous dopamine and dobutamine were also continuously administered as needed. After orotracheal intubation, the lungs were ventilated with 100% oxygen. Standard monitoring included heart rate, arterial blood pressure, pulmonary artery pressure, central venous pressure, mixed venous oxygen saturation, cardiac output, regional cerebral oxygen saturation, bispectral index, urine output, rectal temperature and right tympanic temperature.

Cardiopulmonary perfusionists stood by before induction of anesthesia. Despite low, although stable, arterial pressure during the induction of anesthesia, the patient’s heart rate gradually increased. Cardiac output, mean pulmonary artery pressure, and central venous pressure immediately before surgery were 5.4 L/min, 22 mmHg, and 7 mmHg, respectively. Systolic blood pressure and heart rate were 85 mmHg and 130 bpm, respectively. Soon after sternotomy and during pericardiotomy, the patient developed sudden bradycardia and low blood pressure (below 60 mmHg) requiring immediate direct cardiac compression. Hemodynamic stability could not be maintained despite infusions of epinephrine (2 mg) and atropine (1 mg), and the patient subsequently developed ventricular fibrillation. After an intravenous injection of 300 µg/kg of heparin, the ascending aorta and right atrium were cannulated and CPB was induced immediately. Low blood pressure (below 60 mmHg) and bradycardia followed by ventricular fibrillation continued for 10 min before CPB. Systemic hypothermia was initiated after the induction of CPB, and the right tympanic and rectal temperatures were lowered to 18.1°C and 28.4°C, respectively, within 15 min of induction of CPB. Edaravone (30 mg) and thiamylal (250 mg) were administered for cerebral protection. The patient was cooled and maintained at target right tympanic and rectal temperatures of around 24°C with CPB during aortic valve replacement.

Aortic valve replacement proceeded uneventfully. The abnormal bicuspid aortic valve was calcified and did not open very well. The patient was weaned from initial CPB with norepinephrine (0.1 µg/kg/min), dopamine (7 µg/kg/min) and dobutamine (7 µg/kg/min) at right tympanic and rectal temperatures of 35.3 and 34.3°C, respectively, for cerebral protection. The bispectral index was 64% at this point. CPB, however, had to be restored because of uncontrolled multiple premature atrial contractions and hemodynamic instability 30 min after initial weaning off CPB. Thereafter, the patient’s blood pressure and arrhythmia were stabilized with milrinone (0.2 µg/kg/min) and an intraaortic balloon pump. She was weaned from subsequent CPB at right tympanic and rectal temperatures of 36.1 and 36.4°C, respectively. Cardiac output was 5.3 L/min after weaning from CPB, although landiolol was required for heart rate control. Right tympanic and rectal temperatures were 36.5 and 36.9°C, respectively, before transfer to the intensive care unit (ICU). The durations of surgery, anesthesia, aortic clamping, first CPB, and second CPB were 379, 435, 125, 185, and 38 min, respectively.

Mild hypothermia in the ICU

Our patient’s pupils were reactive to light and iso-
coric at the time of postoperative admission to the ICU. In the ICU, systemic hypothermia (target pulmonary artery blood temperature, 35°C) was induced and applied over a period of 48 h with systemic natural cooling and ice packs around her head and neck, for cerebral protection. Intracranial pressure and bispectral index were not monitored in the ICU. The temperature of the blood in the pulmonary artery, measured via the pulmonary artery catheter, was maintained between 34.5 and 35.5°C for 48 h from the start of cooling, followed by passive rewarming (Figure 1). Clinical treatment in the ICU was as shown in Figure 2. Sedation was induced with intravenous propofol, doses being adjusted as needed to manage mechanical ventilation. Cardiac output fluctuated between 3.4 and 5.8 L/min during hypothermia, with the aid of catecholamines. The need for cardiovascular support (catecholamine infusion and intraaortic balloon pump) gradually reduced and was discontinued within 96 h (Figure 2). Edoxanone (60 mg/day) was administered for 1 week.

The patient was naturally rewarmed for the next 24 h by removal of the ice packs around her head and neck (target pulmonary artery blood temperature, 36°C). She awoke on ICU day 4 after rewarming and discontinuation of sedation. The patient was free of clinically significant infections, shivering, convulsions, and active bleeding. She was extubated on ICU day 5 with no motor paralysis or sensory disturbances. We treated the infectious endocarditis with antibiotics until discharge. She was transferred to another hospital on post-operative day 19 without neurological complications.

Discussion

Although the cause of cardiac arrest can be difficult to determine, several clinical mechanisms could explain the cardiac arrest in this patient who experienced almost daily discomfort and an unpleasant feeling in her chest before aortic valve replacement surgery. Preoperative coronary computed tomography revealed no coronary artery stenosis. However, severe left ventricular hypertrophy was evident on preoperative echocardiogram. Sinus tachycardia and low blood pressure can lead to an imbalance between cardiac oxygen supply and demand, and result in sudden bradycardia and hypotension, followed by ventricular fibrillation. Thus, we had to

![Graph](image)

**Fig.1** Pulmonary artery temperature and right tympanic temperature of the patient while in the ICU. Target temperature, 35°C; duration of cooling, 48 h.
be extra vigilant of heart rate and blood pressure during the anesthetic management of this patient.

Several factors could have affected the positive outcome of the cardiac arrest in our patient. The sternotomy that was performed before cardiac arrest, which allowed direct cardiac compression and rapid induction of CPB, is one possible reason why our patient escaped neurological complications. Brain damage after cardiac arrest and resuscitation is determined by many factors, predominantly the duration of arrest (no flow), cardiopulmonary resuscitation (low flow), and body temperature. In this case, cardiac output for 10 min during direct cardiac compression was probably very low because of severe aortic stenosis. Extracorporeal cardiopulmonary resuscitation has short- and long-term survival benefits over conventional cardiopulmonary resuscitation in patients who develop in-hospital cardiac arrest of cardiac origin 8, 9. Our patient developed low blood pressure followed by cardiac arrest in an operating room environment during a surgical procedure. Therefore, the shorter duration of cardiac arrest likely facilitated a favorable outcome. Survival and neurological outcomes are reportedly related to the location and time of in-hospital cardiac arrest 9. This same clinical report also revealed that asystole and unwitnessed arrests are more frequent in hospitals.

The use of hypothermia after resuscitation from cardiac arrest has been studied in laboratory animals 6, 7. These studies demonstrated significantly improved outcomes when moderate hypothermia was induced after resuscitation, and showed that induced systemic hypothermia obviously mitigates brain damage after cardiac arrest in dogs. Hypothermia probably protects against various deleterious biochemical mechanisms; however, the exact mechanism of the cerebral resuscitation effect remains obscure. After cardiac arrest with no blood flow for over 5 min, the generation of free radicals and other mediators during reperfusion creates chemical cascades that result in cerebral injury 8. Although we rapidly induced hypothermia with CPB during surgery, followed by strict temperature management in the ICU in this case, it is difficult to conclusively comment on the effect of the induced hypothermia on the lack of neurological complications because only one case was observed.

Mild induced hypothermia appears to improve neurological outcomes in patients after cardiopulmonary resuscitation following out-of-hospital cardiac arrest 11, 12. Such patients have been vigorously cooled in an emergency room (or ICU if a bed was immediately

![Fig.2 Clinical treatment in the ICU. VCV, volume control ventilation; SIMV, synchronized intermittent mandatory ventilation; PS, pressure support; BiPAP, biphasic positive airway pressure.](image)
available). In our case, anesthesiologists and cardiovascular surgeons treated the patient with systemic hypothermia, since Kumamoto Chuo hospital does not have emergency or intensive care teams that specialize in hypothermic treatment. Therefore, we managed the patient in the simplest possible way to avoid complications of systemic hypothermia such as infection and hemodynamic instability. The time duration between restoration of circulation and induction of hypothermia, target core temperature, the duration of hypothermia and rewarming all affect neurological outcomes. Our simple hypothermia treatment can be applied to in-hospital cardiac arrest at institutions without emergency or intensive care teams and special equipment. Several reports have shown that inducing therapeutic hypothermia with large infused volumes of an ice-cold solution is feasible and safe for comatose survivors of out-of-hospital cardiac arrest. However, further studies are required to confirm these findings and determine the optimal hypothermic strategy for treating in-hospital cardiac arrest.

In conclusion, our findings suggest that induced hypothermia with systemic natural cooling and ice packs around the head and neck is safe and feasible in post-resuscitation intensive care, and obviates the need for emergency and intensive care teams. Alternative clinical approaches and safer strategies for the application of hypothermia should be investigated in more patients.

We reported this summary at the 27th Annual Meeting of the Japanese Society of Reanimatology (Nagasaki, 2008).

References