Pulmonary endarterectomy under intermittent deep hypothermic circulatory arrest

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Received 18 August 2016
Accepted 29 December 2016

The Egyptian Journal of Cardiothoracic Anesthesia 2016, 10:60–63

Chronic thromboembolic pulmonary hypertension occurs in very few patients after acute embolism, and pulmonary thromboendarterectomy is the main and curative treatment. Here we have a patient with a chronic thrombus in the pulmonary artery that required thromboendarterectomy with deep hypothermic circulatory arrest.

Keywords:
deep hypothermic circulatory arrest, endarterectomy, pulmonary thromboembolism

Egypt J Cardiothorac Anesth 10:60–63

1687-9090

Introduction

Pulmonary artery (PA) embolism usually presents as an acute emergency. It has a low incidence rate, but it is associated with a high mortality rate. This case of subacute pulmonary embolism describes the use of intermittent deep hypothermic circulatory arrest (DHCA) during cardiopulmonary bypass (CPB) for the surgical excision of pulmonary embolism. This requires clear collaboration of anesthesiologists, cardiac surgeons, and perfusionists.

Case report

A 28-year-old man presented with shortness of breath and chest pain at rest of 1-week duration; he had similar complaints of intermittent shortness of breath and chest pain during the previous 6 weeks. He was bedridden for 6 months because of motor weakness and decreased sensation in the right lower limb. Clinical examination revealed a pulse rate of 110 beats/min, respiratory rate of 30/min, and blood pressure (BP) of 110/60 mmHg. Sensation of only crude touch and motor power 1/5 of right lower limb was present. Blood investigations revealed hemoglobin of 10 g/dl, total leukocyte count of 11 k/μl, platelet count of 3.96 k/μl, and random blood sugar level of 100 mg/dl. Liver and renal function tests were within normal limits. Oxygen saturation on room air was 87%, prothrombin time was 14.75/14, international normalized ratio was 1.34, and activated partial thromboplastin time was 30/28. Chest radiograph revealed a cavity in the right lower lobe of the lung. ECG showed a deep s-wave in lead I, q-wave in lead III, and T inversion in lead I (S1Q3T1), as well as sinus tachycardia. He was clinically diagnosed as a case of pulmonary embolism. Thrombolysis treatment with streptokinase failed to yield any clinical improvement.

Specific advanced investigations such as venous Doppler of both lower limbs revealed complete thrombotic occlusion of external iliac vein, common femoral vein, saphenous vein, and popliteal vein of the right lower limb, and partial thrombotic occlusion of the left distal external Iliac vein, common femoral vein, superficial femoral vein, and portal vein. Computed tomography angiography of chest revealed signs of thromboembolism of the right main pulmonary artery (MPA) and its branches. Cardiac catheterization revealed signs of pulmonary artery hypertension (PAH) with PA pressures of 90/30 mmHg and right ventricular (RV) pressures of 100/8 mmHg. Dilated left PA and flush occlusion of the right pulmonary artery (RPA) from the proximal stump were also observed. Two-dimensional echocardiography revealed right atrium and RV moderately dilated with paradoxical septal motion with severe PAH and clot protruding from the RPA to the MPA, and left ventricle ejection fraction of 60%. The patient was diagnosed as a case of pulmonary thromboembolism with severe PAH that failed thrombolysis and presented for elective surgical pulmonary embolectomy under general anesthesia with DHCA.

In the operating room, arterial blood gases showed PO_2 of 47.1 mmHg (SaO_2 82%) and PCO_2 of 28.7 mmHg. After preoxygenation with 100% O_2 for 3–5 min, induction was done with intravenous midazolam 3 mg, intravenous morphine 15 mg, intravenous fentanyl 500

mcg, and intravenous rocuronium 60 mg. Bispectral index (BIS) monitor was applied on the forehead, and after induction the reading was noted as 45. Two temperature probes were inserted (nasopharyngeal and rectal). Right internal jugular vein, left femoral artery, and left radial artery were cannulated. Maintenance of anesthesia was done with rocuronium, fentanyl, and isoflurane with MAC 0.8, as clinically indicated. Intravenous heparin 400 U/kg was administered. Heart was cannulated and CPB was initiated. The patient was gradually cooled down to 18°C and BIS reading was noted as 0. In addition, external cooling of the head was done using a head jacket filled with ice; the heart was cooled by filling the pericardial cavity with saline ice slush to prevent secondary rewarming. Intravenous methylprednisolone 1 g and intravenous thiopentone 1 g was administered through the CBP just before circulatory arrest. Circulatory arrests in deep hypothermia were performed four times, as depicted in Table 1.

First, the RPA was opened and a big thrombus was taken out. The total surgery time was 5 h and 38 min and DHCA time was ~25 min for each episode followed by circulation for 8 min, and antegrade cardioplegia was given twice as depicted in Table 1 to provide myocardial protection. A total of four episodes of DHCA were required.

The patient was rewarmed gradually and weaned off CPB. The BP was 100/52 mmHg. intravenous milrinone infusion was started (5 mcg/kg/h) after administering a loading dose of 50 mcg/kg/h. The rationale for starting milrinone was to support dilated and hypokinetic RV as per visual assessment. The BP improved to 120/70 mmHg and intravenous protamine was slowly administered.

The rest of the surgery was uneventful and the patient was transported to cardiothoracic vascular surgery (CTVS) ICU. He was extubated the same day and discharged 7 days postoperatively (Figs 1 and 2).

Discussion

PA embolism is a blockage of the MPA or its branches by an embolus, which has come mainly from deep vein thrombosis of the lower extremities. The thrombus breaks and lodges in the arteries. Because of obstruction of blood flow in the lungs, there is a back pressure on the RV and right atrium of the heart that leads to chest pain, difficulty in breathing, cough, and so on. Clinical signs include a fall in oxygen saturation, cyanosis, tachypnea, and tachycardia.

The basis of diagnosis is clinical findings, laboratory tests, and imaging, such as computed tomography pulmonary angiography. Treatment includes anticoagulants such as warfarin, thrombolytic drugs such as tissue plasminogen activator, and last surgical intervention and pulmonary thromboendarterectomy using CPB [1,2].

Anesthetic management includes opiod induction to avoid cardiovascular collapse. Ventilation strategy was to avoid hypoxia and hypercarbia to prevent rise in PAH. As in this case of RV hypertrophy, perfusion of the RV is limited to diastole and dependent on diastolic BP similar to left ventricular coronary perfusion. Adequate systemic vascular resistance is therefore necessary to maintain coronary blood flow to the RV and can be maintained by various catecholamine derivatives if necessary.

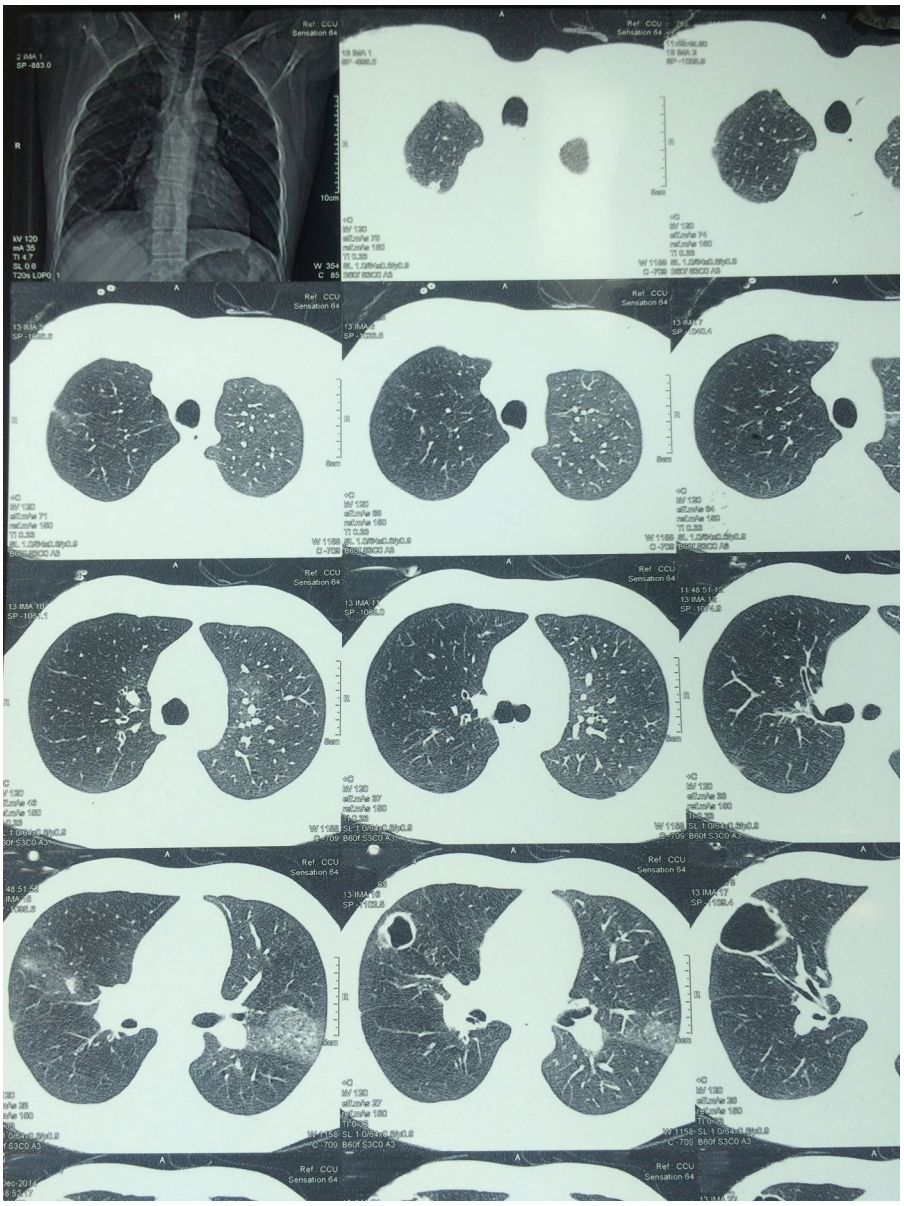
DHCA is done usually in surgeries of the aortic arch or brain, but in this case it was done to have a bloodless surgical access, because of adequate significant collaterals and bronchial blood flow in these patients. The theory of DHCA is to cool the patient, cease blood flow to the brain, and rely on the hypothermic protective effects of

Table 1 Sequential events during deep hypothermic circulatory arrest

Time period of DHCA sessions and flow sessions	Temperature 1 (°C) T1 (N)	Temperature 2 (°C) T2 (Re)	BIS score	RBS (mg/dl)	Systemic pressures during flow periods (mmHg)
DHCA-1 (13:45–14:07 h)	18	21	0	134	0
Onset of flow-1 (14:07–14:15 h) (antegrade cardioplegia given)	16.8	20.9	0		62/56 (MAP=59)
DHCA-2 (14:15–14:45 h)	17.1	21	0	140	0
Onset of flow-2 (14:45–14:54 h)	17.6	21	0		60/55 (MAP=58)
DHCA-3 (14:54–15:24 h)	17.8	21.1	0	154	0
Onset of flow-3 (15:24–15:33 h) (antegrade cardioplegia given)	17.6	21	0		63/56 (MAP=59)
DHCA-4 (15:33–15:50 h)	17.6	20.2	0	133	0
Onset of flow-4 (15:50 h)	17.8	21	0		63/56 (MAP=59)

BIS, bispectral index; DHCA, deep hypothermic circulatory arrest; MAP, mean arterial pressure; RBS, random blood sugar.

Figure 1



Preoperative computed tomography scan.

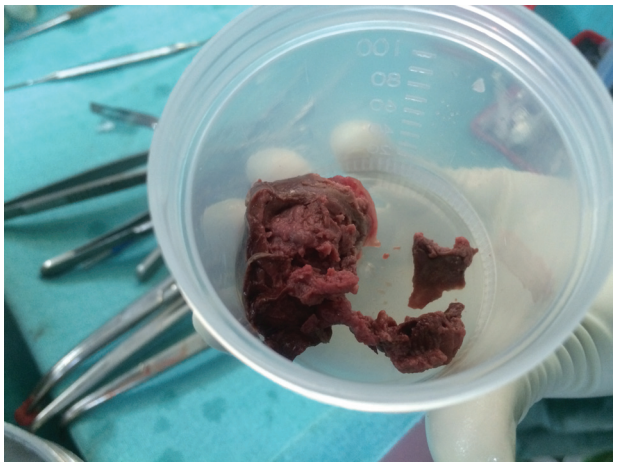
decreased cerebral metabolic rate of oxygen ($CMRO_2$), which decreases significantly with hypothermia. There is a 6–7% decrease in metabolism per 1°C decrease in body temperature. At 15°C, $CMRO_2$ is decreased to 15%.

Acceptable duration for DHCA should be limited to 30–40 min because of the increase in the risk of cerebral injury with prolonged duration. One retrospective analysis showed that the risk of stroke increases when the length of DHCA is longer than 40 min [3]. To prevent it, we used intermittent hypothermic circulatory arrest technique to allow cerebral circulation. Antegrade cerebral circulation can also be done to prolong DHCA duration, but our surgeons were not acquainted with this technique.

Rapid cooling of cerebral tissue can lead to heterogeneous temperatures and cerebral hypoxia. Slow cooling to ensure homogeneous hypothermia of the cerebrum is important. External cooling by head jacket comprising ice was also used to help inhibit secondary rewarming during the duration of DHCA. Rewarming should be accomplished over a similar period to a temperature of 34–35°C before weaning from CPB. Temperatures should be maintained between 35 and 37°C, and postoperative hyperthermia should be avoided in patients at risk for cerebral ischemia.

The incidence of stroke post-DHCA is ~10%.The etiology of cerebral injury post-DHCA is not completely defined; however, it is thought to be due

Figure 2



Embolus is removed.

to impairment in cerebral autoregulation, which is disrupted in hypothermic cardiac arrest [4]. We used BIS for neurologic monitoring with limited evidence of outcome benefits. In addition, it may be affected by anesthetic agents, hypothermia, and electromagnetic interference. Choice of anesthetic agents also varies from institution to institution. We avoid volatile anesthetic agents during DHCA, as it uncouples cerebral blood flow from metabolism.

Pharmacologic neuroprotective agent thiopentone 20 mg/kg was administered before DHCA to provide BIS 0 (Electroencephalographyb (EEG) silence). Steroids do provide anti-inflammatory role during CPB, but neuroprotection during DHCA has not been proven. They might lead to hyperglycemia and impair wound healing, but they were used because of their role in decreasing proinflammatory cytokines, which are thought to cause brain injury during ischemia [5,6].

It is theorized that hypothermia provides the neurologic protective effect by decreasing $CMRO_2$, reducing oxygen free-radical production, diminishing intracellular Ca^{2+} influx, and decreasing glutamine toxicity [7]. Hypothermia also causes significant coagulopathy. The use of centrifugal pump in extracorporeal circuit confers benefit by reducing hemolysis and preserving platelet function. Platelets and other factors are often needed during weaning from CPB. Prolonged CPB time, significant suture lines, and disruption of coagulation all are causes for increased blood loss.

For acid–base balance, we used α-stat. In addition, a meta-analysis on α-stat versus pH-stat showed that pediatric literature supports pH-stat, whereas α-stat may be better in the adult population [8]. Blood glucose levels less than 140 mg/dl should be maintained, as higher levels are suggested to worsen the impact of ischemia through increased glycolysis and intracellular acidosis.Postoperatively, these patients need close monitoring, as temperature may decrease by the time the patient arrives in the ICU. Hypoxemia and hypotension should be avoided. Cognitive dysfunction is common after DHCA [9].

Conclusion

Pulmonary thromboendarterectomy using deep hypothermia with intermittent periods of circulatory arrest is a high-risk surgery. Adequate cooling and careful rewarming with close monitoring of cerebral activities, deep anesthesia, and hemodynamic stability are desirable for a favorable outcome.

Acknowledgements

The authors are thankful to Dr Sanjeev Gupta, MCH for his contribution.

Conflicts of interest

There are no conflicts of interest.

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