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A Case Report on VSD Closure + Lobectomy

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Overview

Case Presentation

DHCA

Case Presentation

Case

Infant patient 24 months, candidate for VSD closure who has been screened for recurrent hemoptysis. There was a large hemorrhagic sequestration in the lower left lobe of the left lung, which was closed by intervention but hemoptysis continued.

Past Medical History

- Favism

ECHO

- Premembranous VSD
- Narrow LPA
- Mild-to-Moderate TR

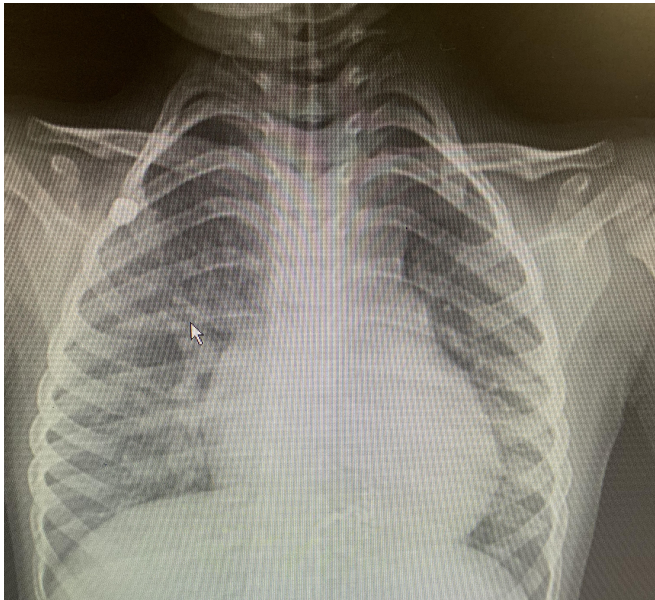
CT Angio

- Large Pulmonary Artery and Branches
- LPA is relatively Hypoplastic
- All PVs return to LA
- Alveolar Pulmonary opacity is seen in LLL due to pulmonary hemorrhage

Lab Test

- Hb = 12.3
- Hct = 35
- WBC = 8600
- Plt = 343000
- U/A = NL
- PT=13.2
- PTT=32
- INR =1.06

CXR



Induction

- Midazolam 3mg
- Fenta 1cc
- cis Atra 6mg
- arterial line : LR
- CV line : RF

ABG

مرکز آموزشی تحقیقاتی و درمانی قلب و عروق شهید رجایی
 یوف آزمايش های خون اتاق عمل

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شماره: تاریخ: شماره پرونده:

ABG		Lab							وزات پزشک						
Pump	PH	Po ₂	O ₂ sat	PCO ₂	HCO ₃	TCO ₂	BE	Na	K	Hb	Hct	BS	Lactate	CL	Ca
	7.40	464	100	39	24	25	0	138	3.3	11.2	368	9.6	11.0	2.4	
	7.29	607	100	46	22	24	-4	134	3.0	9.3	229	2.4	10.8	2.1	
	7.36	540	100	35	20	21	-4	133	4.8	8.5	27	183	0.9	11.5	2.0
	7.31	225	100	31	15	16	-10	134	5.1	7.4	24	170	2.5	11.5	2.1
	7.33	174	99	47	24	26	-1	140	4.1	9.6	31	226	3.0	11.4	1.9
	7.36	128	98	42	24	25	-1	140	3.9	9	29	263	3.6	11.1	2.1
	7.30	172	99	45	22	23	-4	138	2.8	8.1	2	62	3.4	11.5	1.7
	7.28	177	98	49	23	24	-3	148	3.4	8.4	27	269	3.5	11.7	1.8

نورانی ۱۰۰

ca + ptt + kel
 kcl -> ۳-۹

ک

Spiral Chest CT

- Right side pleural effusion
- MPA dilatation
- A few atelectatic band in mid and lower zone

CT Angiography

- Aortic annulus = $26mm$
- Aortic sinus = $39mm$
- Fusiform aneurysm in ascending thoracic aorta with $54mm$ diameter
- Maximum diameter of arch in mid arch is $41mm$

Induction

- BP = 160/90
- O₂Sat = 97
- Cerebral Oximetry = 50-70

- Midazolam = 5*mg*
- Etomidate = 20*mg*
- Cisatra = 20*mg*
- Maintenance = SAM

Lab Test after Induction

Air

- PH = 7.42
- PO₂ = 78
- O₂Sat = 96
- PCO₂ = 41
- HCO₂ = 30
- TCO₂ = 31
- BE = 5
- Na = 139
- K = 3.6
- Hb = 13.3
- HCT = 43
- BS = 90
- Lactat = 0.5
- Cl = 109
- Ca = 1.9

DHCA

From the second hour, the patient gradually became hypothermic to a temperature of 20 C for about an hour and a half and the patient's MAP decreased to about 20mmHg.

Cerebral Oximetry = 39/50 53/48 31/39

Medications

Medications that the patient received during hypothermia include:

- Phenobarbital = 200mg
- Thiopantal Sodium
- Dexamethazon = 8mg
- Topical Cerebral Cooling

Off Pump

- PH = 7.49
- O₂Sat = 100
- PCO₂ = 31
- HCO₂ = 24
- TCO₂ = 25
- BE = 2
- Na = 137
- K = 4.2
- Hb = 9.6
- BS = 166
- Lactat = 3.6
- Ca = 1.6
- Infusion Milrinone
- Infusion Epinephrin
- Fibrinogen 1gr
- Calcium 10cc
- Albumin 200gr

Introduction to DHCA

Most cardiac surgical procedures can be accomplished using cardioplegia-induced cardiac arrest and cardiopulmonary bypass (CPB) to maintain perfusion of other organs. In some situations, however, the underlying pathology or the nature of the surgery proposed necessitates complete cessation of the circulation. The use of profound systemic hypothermia to preserve organ function during cessation of the circulation is termed deep hypothermic circulatory arrest (DHCA).

DHCA

DHCA provides excellent operating conditions while reducing the consequences of organ ischaemia. As the brain is the organ most susceptible to ischaemia during circulatory arrest, it follows that other organs will also be protected by this strategy.

Indications for DHCA

Cardiac	Aortic surgery Pulmonary thromboendarterectomy Complex congenital surgery
Neurological	Cerebral aneurysms Arterio-venous malformations
Other	Renal cell carcinoma with caval invasion Other tumours with caval invasion

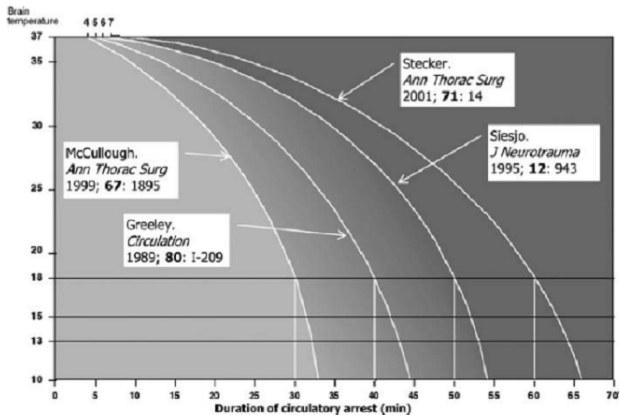
Table: Indications for DHCA

Adverse Consequences of Hypothermia

Cardiovascular	Arrhythmias secondary to potassium loss Increased plasma viscosity Vasoconstriction impairing microcirculation
Coagulation	Impaired coagulation Reduced platelet count
Renal and metabolic	Reduced glomerular filtration rate Metabolic acidosis Hyperglycaemia secondary to impaired glucose metabolism Effects on pharmacodynamics and pharmacokinetics
Cerebral	Vasoconstriction during cooling Brain injury from hyperthermia during rewarming

Table: Adverse consequences of hypothermia

Duration of Circulatory Arrest



Safe period for DHCA

At normothermia, brain injury occurs after around 4 min of circulatory arrest. Cerebral metabolism decreases by 6–7% for every 1 °C decrease in temperature from 37 °C; therefore, brain cooling results in a reduction in oxygen requirements. Circulatory arrest is typically undertaken at 18–20 °C and a range of safe periods for DHCA have been reported at this temperature. Most patients tolerate 30 min of DHCA without significant neurological dysfunction, but when this is extended to longer than 40 min, there is a marked increase in the incidence of brain injury. Above 60 min, the majority of patients will suffer irreversible brain injury, although there are still a small number of patients who can tolerate this. Longer periods of DHCA are tolerated in neonates and infants compared with adults.

Anaesthesia for DHCA I

All patients should undergo thorough preoperative assessment and be prepared for the extent of invasive monitoring and the potential for prolonged postoperative intensive care. When a patient requires emergency surgery, however, detailed assessment and prolonged discussion may be neither practical nor possible. Sedative premedication may be appropriate for patients undergoing an elective procedure involving DHCA, although care should be taken in patients in whom respiratory depression and hypoxia may induce haemodynamic disturbance—for example, patients with severe pulmonary hypertension.

Anaesthesia for DHCA II

The use of corticosteroids should be considered, as there is some evidence—albeit inconclusive—that they are neuroprotective. This is thought to occur by decreasing the release of inflammatory cytokines and preventing lysosomal breakdown during hypothermia. To be effective, corticosteroids (e.g. prednisolone 1 mg kg⁻¹) should be administered at least 6–8 h before surgery, any resultant hyperglycaemia is probably best treated using insulin.

Induction and Monitoring I

In addition to routine non-invasive monitoring, all patients undergoing surgery with DHCA require invasive arterial and central venous pressure monitoring. In some instances, femoral or bilateral radial arterial monitoring may be appropriate. In addition to allowing measurement of central arterial pressure, placement of a femoral arterial cannula may aid the surgeon should an intra-aortic balloon pump be required. Measurement of right heart pressures using a pulmonary artery catheter or the use of transoesophageal echocardiography (TOE) may be indicated in some patients. Temperature monitoring at two sites, typically the nasopharynx and bladder, is used to estimate brain and body temperatures, respectively. Studies have shown that bladder and tympanic temperatures correlate most closely with brain temperature. There is poor correlation between pulmonary artery catheter and rectal temperature measurements, and brain temperature.

Induction and Monitoring II

Despite limited evidence of outcome benefit, neurological monitoring is routinely used in many centres. Broadly speaking, these fall into one of two categories: monitors of cerebral substrate delivery [jugular bulb oximetry, transcranial Doppler sonography, and near infrared spectroscopy (NIRS)] and monitors of cerebral function [quantitative electroencephalography (qEEG) and evoked potential monitoring].

Induction and Monitoring III

Measurement of jugular venous oxygen saturation (SjO_2) provides an indication of the balance between cerebral oxygen supply and demand. Its use is limited as SjO_2 reflects global cerebral blood flow and may be unaffected by regional cerebral ischaemia. Low SjO_2 before the start of DHCA has been shown to be associated with adverse neurological outcome.

Induction and Monitoring IV

Transcranial Doppler is used to measure cerebral blood flow velocity (a surrogate for cerebral blood flow) in the basal cerebral arteries and to detect microemboli. Using paired optical sensors placed on the scalp, NIRS measures the oxygen saturation of blood in all vessels to a depth of 20–40 mm. Ease of use and limited potential for harm have prompted the increasing use of NIRS monitoring.

Induction and Monitoring V

qEEG is a sensitive monitor of cerebral ischaemia but may be affected by electromagnetic interference, hypothermia, and anaesthetic agents. In some centres, the EEG is used to assess the adequacy of cooling before DHCA. However, the temperature at which the EEG becomes isoelectric varies widely between patients—as low as 12.5 °C in some studies—and appears to be higher during cooling than rewarming.

Induction and Monitoring VI

The choice of anaesthetic drugs is largely a matter of personal and institutional preference. Theoretically, avoiding volatile agents, which uncouple cerebral blood flow from cerebral metabolism (autoregulation), may confer some benefit. The influence of hypothermia on drug metabolism and elimination should be considered and doses altered accordingly.

Anticoagulation

Unfractionated heparin ($3\text{--}5 \text{ mg kg}^{-1}$) is typically used to maintain an activated clotting time (ACT) $> 400 \text{ s}$. Inadequate anticoagulation during CPB increases the consumption of clotting factors and may paradoxically worsen postoperative coagulopathy. Lysine analogues (e.g. tranexamic acid, ϵ -aminocaproic acid) are frequently used to reduce fibrinolytic haemorrhage. It should be borne in mind that tranexamic acid may cause postoperative seizures.

General considerations

The duration of surgery conducted with DHCA mandates careful attention to the prevention of pressure sores and damage to the eyes, nerve plexuses, and peripheral nerves. Particular attention should be paid to cannulation sites, monitoring lines, airway connectors, and TOE probe which may cause pressure necrosis of the skin.

Measures should be taken to assist rewarming and prevent hypothermia after separation from CPB. An i.v. fluid warmer, heated mattress, and forced-air blanket should be considered in all cases.

Cerebral protection I

Systemic cooling is achieved during CPB by pumping water from a heater/cooler reservoir through a heat exchanger in the CPB oxygenator. The temperature gradient between water and blood is typically maintained at $< 10^{\circ}\text{C}$. Additional cerebral cooling can be achieved using a head-cooling jacket through which iced water is circulated or ice packed around the head. Although topical cooling of the head has been shown to be of some benefit in animal models, there is as yet no evidence of outcome benefit in humans.

Cerebral protection II

When CPB is resumed after a period of DHCA, hypothermic perfusion should be maintained for 10–20 min before rewarming commences. This is thought to reduce the risk of raised intracranial pressure which can occur during this period. Once rewarming commences, the gradient between core and peripheral temperatures should be $< 5^{\circ}\text{C}$. Excessively rapid rewarming with perfusion temperatures $> 37^{\circ}\text{C}$ may induce cerebral ischaemia secondary to an imbalance between oxygen supply and demand. Similarly, cerebral hyperthermia should be avoided as this may exacerbate neurological injury and increase the risk of adverse neurological outcomes.

Haemodilution

During hypothermia, the combination of increased plasma viscosity, erythrocyte rigidity, and progressive vasoconstriction leads to impairment of the microcirculation. Haemodilution, typically to a haematocrit of 20%, is thought to improve flow in the microcirculation.

Excessive haemodilution (e.g. haematocrit <10%) significantly reduces oxygen carrying capacity and causes tissue ischaemia. Normovoemic haemodilution is often achieved by removing heparinized blood via the arterial cannula immediately before commencement of CPB. Any blood removed is labelled and stored for retransfusion during rewarming.

Pharmacological Neuroprotection I

Drugs used for this purpose included thiopental (59%), propofol (29%), and others (48%), most commonly corticosteroids.

Thiopental and propofol have both been investigated at doses sufficient to cause burst suppression. Propofol does not appear to be associated with cardiac depression or delayed emergence from anaesthesia.

Although the use of thiopental is associated with dose-dependent myocardial depression and delayed emergence, it does not adversely impact on the ability to separate from CPB. There is no evidence that either drug improves neurological outcome in adults.

Glycaemic Control

There is evidence that hyperglycaemia during periods of hypothermia worsens the impact of ischaemia through increased glycolysis and intracellular acidosis. Virtually, all patients undergoing DHCA develop impairment of glucose metabolism and will require control of glucose with insulin. Concurrent corticosteroid administration is likely to increase the risk of perioperative hyperglycaemia.

The End