Regional cerebral oxygenation monitoring – intraoperative management in a patient with severe left ventricular dysfunction

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Intraoperative near-infrared spectroscopy cerebral oxygenation monitoring assists intraoperative decision-making in environments without extracorporeal membrane oxygenation (ECMO), left ventricular assist device (LVAD) or access to cardiac transplantation. We report a case of an anomalous left coronary artery arising from the pulmonary artery (ALCAPA), undergoing cardiac surgery. A 4-month-old infant presented *in extremis* with cardiac failure. We discuss the pathophysiology and challenging intraoperative management of ALCAPA with extensive ischaemia and myocardial infarction.

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Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) is an uncommon congenital heart defect with an incidence of approximately 1 in 300 000 live births. The presentation and onset of symptoms typically occurs shortly after the neonatal period as the pulmonary vascular resistance falls and left coronary blood flow diminishes. Decreased coronary blood flow results in myocardial ischaemia, tissue infarction, mitral insufficiency and dilated ischaemic cardiomyopathy. Approximately 10 – 15% of patients remain asymptomatic and survive to adulthood and then present with coronary ischaemic syndromes or sudden death. The diagnosis is confirmed with echocardiography and occasionally cardiac angiography. Surgical therapy is indicated and should ideally take place before development of significant myocardial dysfunction. Anaesthetic haemodynamic management in the perioperative period must be meticulous because these infants are at significantly increased risk of myocardial ischaemia, cardiac arrest and sudden death.

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Case report

A 4-month-old, 5 kg girl presented *in extremis* to the intensive care unit. Electrocardiography revealed extensive ischaemic changes with the suggestion of established anteroseptal infarction. Chest radiography revealed significant cardiomegaly and pulmonary oedema. Transthoracic echocardiography showed a grossly dilated, poorly contracting left ventricle. The septal and posterior walls of the left ventricle, as well as the anteromedial papillary muscle, were echo-bright with associated severe mitral regurgitation. The origin of the left main coronary artery was difficult to visualise. The aortic arch was widely patent. Aortic root angiography confirmed the absence of the left coronary system from the aortic sinus.

The patient was stabilised and surgery scheduled 20 hours after ICU admission when further haemodynamic improvement was considered unlikely. In theatre monitoring was continued, including the use of near-infrared spectroscopy (NIRS) with the Somanetics (Troy, MI, USA) INVOS 5100 cerebral oxygenation monitor. Two sensors were used,[1] one placed at the level of T12 on the patient's back lateral to the vertebrae to measure regional somatic visceral oxygenation index ($rS0_{2i}$) and the other over the frontal cortex to measure regional cerebral oxygenation index ($rCS0_{2i}$). Surgery consisted of reimplantation of the left coronary artery onto the aorta, reconstruction of the pulmonary artery root with autologous pericardium, and a partial suture annuloplasty of the mitral valve.

The patient was separated from cardiopulmonary bypass with milrinone 1.5 µg/kg/min, nitroprusside 3 µg/kg/min and very high-dose adrenaline 3 µg/kg/min, the patient achieving haemodynamic stability. Because of oedema of the hypocontractile myocardium, the sternum could not be safely approximated and was left open. The skin wound was closed with a polytetrafloroethylene patch.

The INVOS cerebral oxygenation sensor placed over the right frontal cortex and regional somatic sensor placed at the level of T12 posteriorly were continuously displayed throughout the surgery and weaning from cardiopulmonary bypass. Throughout anaesthesia the mean and standard deviation saturation index for the cerebral sensor was $rCS0_{2i}$ (62 ± 7) and the mean somatic sensor index was $rS0_{2i}$ (77 ± 6) (Fig. 1). The continuous readout of these regional oxygenation trend indices reflected excellent oxygen delivery to the brain and somatic viscera as well as a normal oxygen extraction ratio during anaesthesia. They were the most important factor in determining the adequacy of red blood cell transfusion, systemic arterial afterload reduction and correct myocardial inotrope assistance needed to separate the child from cardiopulmonary bypass and achieve haemodynamic stability postoperatively. The peak postoperative lactate level of 1.8 mmol/l attested to the adequacy of cardiac output and oxygen delivery intra- and postoperatively.

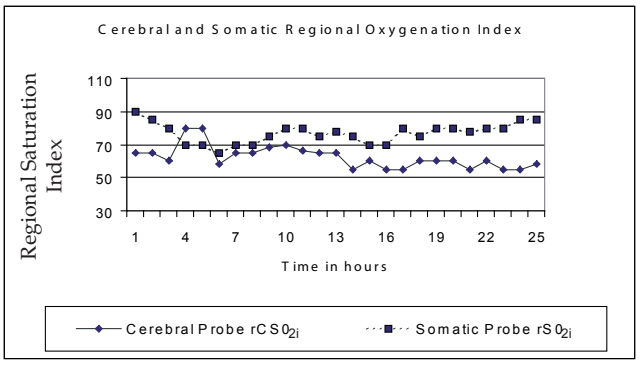

Fig. 1. Two site NIRS trends in a patient undergoing surgery for ALCAPA. Note the increase in cerebral oxygenation index during the time of cardiopulmonary bypass at 3 - 5 hours from the start of surgery.

The sternum was closed 4 days postoperatively, endotracheal extubation followed 2 days later with a further 2 days of continuous positive airway pressure (CPAP), and the child was discharged from the ICU on day 10.

Discussion

ALCAPA, also known as Bland-White-Garland syndrome, was first described in 1911 by Abrikossoff.[2,3] The clinical picture is usually an infant presenting with features of left ventricular dysfunction shortly after the neonatal period. The differential diagnosis includes critical coarctation of the aorta, severe aortic stenosis, myocarditis and non-ischaemic cardiomyopathy (metabolic, infection).

Blood in the coronary artery system flows in the direction of lowest resistance. In isolated ALCAPA, lower pulmonary artery pressure favours blood flow from the normal right coronary artery through myocardial coronary collateral vessels to the left coronary system and into the pulmonary arteries creating a 'steal' of blood flow from the myocardium.[4] Collaterals tend to develop early as arteriovenous fistulas, diverting blood from the myocardium to the pulmonary artery during the period of decreasing pulmonary vascular resistance in the weeks following birth. This causes myocardial ischaemia and progressive left ventricular dysfunction with infants presenting in congestive cardiac failure. Retrograde left coronary flow from inter-coronary collateral vessels and into the pulmonary artery can be demonstrated by cardiac echocardiography or cardiac angiography.

Without early surgical correction, poor myocardial blood flow leads to the development of severe ischaemic cardiomyopathy. Cardiac transplantation may be the only option in cases of end-stage ventricular failure.

Without extracorporeal membrane oxygenation (ECMO), left ventricular assist device (LVAD) or transplantation, the perioperative anaesthetic management of infants presenting *in extremis* with a diagnosis of ALCAPA is difficult. A haemodynamically stable intravenous induction with etomidate to minimise increased myocardial oxygen consumption may safely initiate induction of anaesthesia and intubation.[5] The main anaesthetic goal in ALCAPA is to ensure adequate right coronary artery perfusion and a normal systemic vascular resistance. The haemodynamic challenge of the myocardial depressant anaesthetic inhalational agents lies in treading a fine line between maintaining adequate diastolic blood pressure to optimise coronary blood flow and coronary perfusion with adequate afterload via a single right coronary artery and avoiding too high an afterload with increased left ventricular end-diastolic pressure and diminished coronary perfusion. A hypocontractile, ischaemic, dilated cardiomyopathy will not tolerate a high afterload with a low diastolic blood pressure.

Because of their small size, invasive cardiac output monitoring in children is technically challenging and not without morbidity. Current critical care management focuses on tissue oxygenation as a marker of adequate oxygen delivery, which is reflected by a normal mixed venous saturation (SvO_2) (normal 75%). The anaerobic threshold in adults occurs at SvO_2 of 50%, in infants and neonates it appears to be lower at 30 - 40%.[1] The patient did not develop an acidosis and this reflects adequate delivery of oxygen to the tissues as borne out by the regional NIRS values being greater than 50% during the perioperative period (Fig. 1). Our patient's somatic saturation index remained consistently higher than the cerebral saturation index. This has been noted in other studies and is thought to be due to higher oxygen extraction ratio in the brain.[1]

NIRS measures regional tissue oxygenation non-invasively and continuously. The INVOS 5100 monitor employs NIRS technology with an infrared light source and two sensors

applied to the skin. One is placed over the frontal cortex measuring regional cerebral tissue oxygenation and the other, in children, can be used over the somatic visceral area as a measure of somatic-renal tissue oxygenation.[1] Each sensor transmits two beams of infrared light. One sensor detects reflected infrared light superficially from skin and bone, and the other sensor analyses reflected light returning from deeper brain tissue or somatic organs (in particular liver or right kidney over the 12th thoracic rib posteriorly) at a depth of 2.5 cm.[1] The monitor displays an approximation of tissue oxygenation deep to the sensor as a regional saturation index (rSO_2). This index is a 3:1 venous weighted measure of the mixed arterial and venous blood saturation. NIRS should not be utilised as a diagnostic tool but rather as a physiological trend monitor providing a non-invasive measure of tissue oxygenation.[6]

References

1. Hoffman GM, Ghanayem NS, Tweddell JS. Noninvasive assessment of cardiac output. *Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu* 2005; 12-21.
2. Abrikossoff A. Aneurysma des linken Herzventrikels mit abnormer Abgangsstelle der linken Koronararterie von der Pulmonalis bei einem funsonatlichen Kinde. *Virchows Archiv für pathologische Anatomie und Physiologie und für klinische Medizin, Berlin* 1911; **203:** 413-420.
3. Bland EF, White PD, Garland J. Congenital anomalies of coronary arteries. Report of unusual case associated with cardiac hypertrophy. *Am Heart J* 1932-1933; **8:** 787-801.
4. Ikari NM, Diogenes TC, Gutierrez PS, *et al*. Mitral valve dysfunction caused by papillary muscle necrosis in patients with anomalous origin of the left coronary artery from the pulmonary trunk. *Arq Bras Cardiol* 1991; **57:** 129-132.
5. Schechter WS, Kim C, Martinez M, Gleason BF, Lund DP, Burrows FA. Anaesthetic induction in a child with end-stage cardiomyopathy. *Can J Anaesth* 1995; **42:** 404-408.
6. Hoffman GM. Pro: near-infrared spectroscopy should be used for all cardiopulmonary bypass. *J Cardiothorac Vasc Anesth* 2006, **20**(4): 606-612.

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