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Pediatric Anesthesia

Ventricular fibrillation in an ex-premature neonate following reperfusion of ischaemic gut incarcerated within an inguinal hernia.

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Ventricular fibrillation in an ex-premature neonate following reperfusion of ischaemic gut incarcerated within an inguinal hernia.

Summary

We describe the case of a neonate who underwent surgery for bowel obstruction. The child was born at 25 weeks post conception and at the time of surgery he had a postconceptual age of 44 weeks. He had undergone two previous laparotomy procedures for necrotizing enterocolitis. At laparotomy there was unexpected extensive compromise to gut perfusion. The child developed ventricular fibrillation following the reperfusion of a segment of ischaemic gut found incarcerated in an inguinal hernial orifice. We discuss the pathophysiology of intestinal ischaemia-reperfusion (I-R) injury. We have reviewed the interventions that may be employed to minimise the systemic impact of I-R.

Keywords: Hyperkalaemia; Hyperkalaemia in infants; Ventricular fibrillation; Reperfusion injury.

Introduction

Ventricular fibrillation is a well documented complication following the reperfusion or revascularisation of a previously ischaemic region. It has been observed in children following limb reperfusion, in transplant surgery and following release of gut volvulus. Ventricular fibrillation has not been previously reported to occur following reperfusion of ischaemic bowel within an incarcerated hernia.

Case report

SECTION REDUCED TO EXCLUDE UNNECESSARY HISTORY

The child described in this case was born after 25 weeks gestation with a birth weight of 900 grams. Within the first few days of life he developed signs and symptoms consistent with necrotising enterocolitis. A laparotomy was performed on day 3 which identified a perforation in keeping with the diagnosis of necrotizing enterocolitis. A second laparotomy was required within a few days of the first. After these initial episodes he required a period

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3 of ventilator support and total parenteral nutrition. He was discharged from the neonatal
4 intensive care unit a few weeks later, and whilst he continued to require total parenteral
5 nutrition, he required only minimal oxygen therapy without ventilatory support.
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10 **Nineteen weeks after birth, at a post-conceptual age of 44 weeks he became acutely unwell**
11 **with a grossly distended abdomen.** Despite aggressive resuscitation, his condition
12 deteriorated further and it was necessary to use a high frequency oscillating ventilation. It
13 was considered that despite the infant's poor condition, he had been optimised as much as
14 possible through medical intervention. Following surgical consultation, it was decided that
15 surgical intervention by laparotomy provided the only possibility of survival.
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22 The child was transferred to theatre where high frequency oscillating ventilation was
23 resumed. In addition to the central venous access already in situ, a further peripheral
24 cannula was placed in the right foot. Anaesthesia was maintained with an infusion of
25 morphine 40ug/kg/hr, intravenous ketamine 1mg/kg, given at 30 minute intervals and
26 pancuronium 0.1mg/kg. The consultant surgeon raised concerns about the possibility of
27 abdominal compartment syndrome, and, prior to incision, intravenous mannitol 0.5mg/kg
28 was administered. A midline incision was performed and the peritoneum was entered
29 without any change in the patient's haemodynamic state. After extensive and prolonged
30 division of adhesions, it was apparent that the majority of the bowel had a dusky
31 appearance and its viability was in doubt. The child remained stable throughout this initial
32 period of dissection (SaO₂ 89-94%, HR 140-150, SBP 60-70) and only required a bolus of
33 10ml/kg/hr 4.5% human albumin solution (HAS) to maintain haemodynamic stability. After
34 an hour and a half, it was necessary for the surgeon to release a segment of bowel that had
35 passed down the left inguinal canal. Within 1 minute, the peripheral oxygen saturation
36 began to fall and after a brief run of ventricular tachycardia, he went into ventricular
37 fibrillation (VF). Cardiopulmonary resuscitation was commenced with the surgeon providing
38 chest compressions, and whilst ventilation was provided via a T-piece, the defibrillator was
39 brought into theatre. During the brief hiatus awaiting the defibrillator, it seemed
40 appropriate to treat suspected underlying cause of VF with dose specific resuscitation drugs
41 which had been prepared in advance. The cardiac arrest was treated with boluses of
42 0.1ml/kg 1:10000 adrenaline, 0.5ml/kg calcium gluconate 10% and 20ml/kg 4.5% HAS. A
43 rhythm consistent with cardiac output was rapidly restored but quickly deteriorated back
44 into VF. Further boluses of adrenaline and calcium were immediately administered, in
45 addition to sodium bicarbonate and furosemide 1mg/kg. A wide complex tachycardia was
46 observed which resolved into sinus tachycardia and cardiac output was confirmed by
47 presence of femoral pulsation. It was no longer possible to aspirate blood from the central
48 venous access in order perform blood gas analysis, and the surgeon performed a femoral
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3 cut down to cannulate the femoral artery. Initial blood gas analysis indicated a metabolic
4 acidosis, elevated lactate but normal potassium.
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7 The division of adhesions was a complex procedure and it proved impossible to maintain
8 haemostasis. During the operation, 120ml/kg packed red cells, 20ml/kg platelets and 40
9 ml/kg fresh frozen plasma in addition to 100ml/kg 4.5% HAS were transfused. Towards the
10 end of the operation, there was a brisk and substantial haemorrhage, the arterial blood
11 pressure fell to 30/10 and external chest compressions were initiated. After a rapid
12 transfusion and 0.1ml/kg 1:10000 adrenaline, the blood pressure returned to within
13 acceptable limits. After complete division of the adhesions, it was concluded that the
14 majority of the bowel was not viable. The decision was made to close the abdomen, to
15 administer no further blood products and return to the child to his parents on NICU. He
16 passed away within an hour of returning to NICU.
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25 Discussion

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30 Ischaemia-reperfusion injury is a multifaceted pathophysiological process that is set in
31 motion following the restoration of blood flow to an ischaemic organ. Certain biochemical
32 consequences of I-R are easily recognizable to the clinician. Following revascularization
33 there is an immediate increase in the plasma concentration of potassium ions, hydrogen
34 ions, lactate and partial pressure of carbon dioxide. These changes alone can induce life
35 threatening arrhythmias such as ventricular fibrillation. Whilst restoration of blood flow to
36 the bowel is essential for tissue viability, the histological changes observed after I-R exceed
37 those observed after the same period of ischaemia alone (1). There is a delayed systemic
38 impact of I-R that extends beyond the effected organ. A cascade can be activated which
39 results in systemic inflammatory response syndrome (SIRS) and can progress to multi-organ
40 dysfunction syndrome (MODS) (2).
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49 The cellular changes associated with I-R are less readily identifiable. With a reduction in
50 available oxygen, there is a failure in mitochondrial oxidative phosphorylation, resulting in
51 reduced levels of adenosine 5'-triphosphate (ATP). A reduction in ATP synthesis results in
52 failure of ATP-dependent membrane pumps and cellular accumulation of calcium, sodium
53 and water, associated with cellular injury (3). Failure of normal ATP metabolism also results
54 in accumulation of hypoxanthine. This would normally be converted to xanthine by
55 xanthine dehydrogenase and then to uric acid by xanthine oxidase. Under ischaemic
56 conditions, xanthine dehydrogenase is converted to excessive quantities of xanthine
57 oxidase. Upon reperfusion, the large quantity of xanthine oxidase uses hypoxanthine and
58 the newly available oxygen as substrates to form hydrogen peroxide and other reactive
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3 oxygen species (ROS). ROS such as superoxide (O_2^-), hydroxyl radicals (OH^-) and peroxide
4 not only cause direct damage to the reperfused tissue by lipid membrane peroxidation, but
5 can activate leukocytes and complement pathways. C5a and to a lesser extent C3a are
6 responsible for the increased expression of pro-inflammatory mediators such as tumour
7 necrosis factor α , interleukin-1, and interleukin-6 (1), implicated in the progression of SIRS
8 to MODS (2).
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15 Numerous strategies have been suggested to reduce the impact of I-R, and these can be
16 categorised into physical and pharmacological interventions. Physical interventions include
17 induced hypothermia, which has been demonstrated experimentally to reduce the impact
18 of gut I-R injury both locally and at distant organs (4). Another example of physical
19 intervention is ischaemic preconditioning, where exposure to short periods of ischaemia and
20 reperfusion can protect organs from prolonged periods of I-R (5). It is this same mechanism
21 that is postulated for the protective effects of isoflurane anaesthesia in coronary artery
22 bypass graft surgery (6). Other pharmacological interventions include the administration of
23 agents known to have antioxidant and ROS scavenging properties such as mannitol and n-
24 acetylcysteine (7), both commonly deployed in transplant surgery. More recently, there has
25 been interest in a possible role for ketamine in the prevention of I-R in an animal model of
26 bowel I-R injury (8).
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36 We would like to highlight a number of issues that arose during the management of this
37 case. We took the decision to proceed without arterial blood pressure monitoring. This
38 decision was based on the likelihood of successful cannulation in a child with such
39 pronounced peripheral oedema and abdominal distension, the relatively stable
40 unsupported cardiovascular state, available access to central venous blood sampling and the
41 need for prompt surgery.
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45 Due to the surgeon's concerns regarding the possibility of abdominal compartment
46 syndrome, we felt it appropriate to cautiously administer mannitol, despite the child's
47 serious condition. At the time of administration, the child was relatively stable from a
48 haemodynamic perspective, and the systemic impact of abdominal compartment syndrome
49 could well have resulted in fatality.
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53 Because of the precise timing of the VF arrest, immediately following the release of
54 incarcerated bowel, we suspected that the cause was due to a reperfusion injury. The
55 reperfusion could have released hydrogen ions, potassium and inflammatory mediators into
56 the systemic circulation. These initially might cause myocardial stunning, decreased cardiac
57 output and explain the fall in peripheral oxygen saturation. The same pro-arrhythmogenic
58 metabolites and mediators could result in the development of VT and VF. The episode
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3 occurred suddenly and during a period of relative cardiovascular stability. We did not feel
4 the arrhythmia was as a result of gradually worsening condition. We used sodium
5 bicarbonate and furosemide in an attempt to obtund the effects of these metabolites.
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11 To summarise, in cases where there is a clear concerns regarding reperfusion injury, there
12 are various therapeutic options to consider. These primarily focus upon the protection
13 against reactive and damaging metabolites. Interventions include the use of mannitol and
14 other antioxidant therapies such as n-acetylcysteine. If reperfusion results in cardiovascular
15 compromise, early treatment of hyperkalaemia and metabolic acidosis should be
16 considered.
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For Peer Review

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