CASE REPORT

Hyperbaric oxygen therapy in the management of severe acute anaemia in a Jehovah’s Witness

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Summary

A case is described in which a Jehovah’s Witness patient who refused blood transfusion suffered massive antepartum haemorrhage, her haemoglobin falling as low as 2.0 g dl−1. She was treated on an intensive care unit with intermittent positive pressure ventilation and general supportive measures, pulsed hyperbaric oxygen therapy and recombinant human erythropoietin.

Keywords Religion; Jehovah’s Witness. Oxygen therapy; hyperbaric. Anaemia.

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There follows a description of the management of severe acute blood-loss anaemia in a Jehovah's Witness patient who refused blood transfusion and whose haemoglobin fell to a minimum of 2.0 g.dl⁻¹. The principal problem throughout was maintaining adequate tissue oxygenation while the haemoglobin concentration recovered. Therefore care was taken to minimise blood loss and oxygen requirements, and maximise oxygen delivery and erythropoiesis.

Case history

A 38-year-old woman, para 2, gravida 3, presented to the labour ward following ambulence transfer at 39 weeks' gestation following sudden, profuse and painful vaginal bleeding at home. She was clinically pale and tachycardic (120 beat.min⁻¹), with blood pressure 165/95 mmHg. She was alert and orientated and announced that she was a Jehovah's Witness and did not want any blood even if she would otherwise die. After being assured that her wishes would be respected she was immediately taken to theatre. Blood taken at this time showed her haemoglobin concentration (Hb) to be 7.4 g.dl⁻¹.

Rapid sequence induction of anaesthesia was performed with pre-oxygenation, cricoid pressure, etomidate 20 mg and suxamethonium 100 mg. Tracheal intubation was performed with a size 8.0 cuffed tube. Anaesthesia was maintained with enflurane in 100% oxygen, vecuronium and incremental doses of fentanyl and morphine once the baby was delivered. Emergency lower segment Caesarean section confirmed massive placental abruption, and resuscitation of the stillborn baby by the paediatric team was unsuccessful. Haemostasis was encouraged by administration of syntocinon (intravenous infusion), ergometrine 500 µg intramuscularly and carboprost 250 µg by intra-uterine injection. Hysterectomy was decided against as haemostasis was acceptable and the abdomen was closed in layers leaving two large drains. Total blood loss was estimated to be in excess of 31 L.

There was good cardiovascular stability throughout, blood pressure 120–140 mmHg systolic with a persistent tachycardia of 120 beat.min⁻¹. A total of 3.5 l of synthetic colloid and 6.5 l of crystalloid was given intra-operatively. Postoperatively she was transferred to the intensive care unit (ICU), sedated, paralysed and ventilated.

On arrival at the ICU her Hb was 4.2 g.dl⁻¹, platelet count was 71 × 10⁹.l⁻¹, and clotting studies showed the International Normalised Ratio to be 1.8 and the activated partial thromboplastin time ratio 1.3. Aprotinin, tranexamic acid and vitamin K were administered until the coagulation improved. To minimise oxygen consumption she was kept sedated (with propofol and morphine infusions), paralysed (with a pancuronium infusion) and ventilated. Blood sampling was kept to a minimum and, when required, paediatric size sample tubes were used.

On day 2, the Hb fell to 3.0 g.dl⁻¹. The patient developed a persistent metabolic acidosis (pH 7.17; base excess —10.5 mmol.l⁻¹) despite boluses of sodium bicarbonate, and ischaemic changes were seen on the ECG, despite ventilation with 100% oxygen. A pulmonary artery flotation catheter was introduced and cardiac output studies were performed. She required inotropic support with an adrenaline infusion at 6 µg.min⁻¹. Treatment with erythropoietin was commenced (20 000 units three times per week). The marrow was also supported by administration of haematinics (vitamin B 12 1 mg i.m. once daily, folic acid 10 mg i.v. once daily, and iron 100 mg i.m. once daily). Nasogastric feed was not tolerated initially so parenteral nutrition was instituted 36 h after ICU admission.

In view of the persistent metabolic acidosis, evidence of myocardial ischaemia and probable gut ischaemia, it was decided that treatment with hyperbaric oxygen (HBO) was indicated. The treatment was pulsed HBO for 90 min at 2 atm absolute (ATA). This was administered three times a day initially, then twice daily as the patient stabilised. The HBO treatments were associated with a reversal of the ischaemic ECG changes, an increase in urine output, a decrease in the cardiac output, an increase in systemic vascular resistance and a reduction in inotrope requirements (Table 1). Systemic acidosis was monitored by the base excess, and reversal of metabolic acidosis was seen during HBO treatment. Base excess improved from — 5.6 to — 0.8 mmol.l⁻¹ during the first treatment. After the first HBO treatment, bowel sounds returned and enteral feeding was recommenced. These improvements persisted beyond the duration of the HBO treatments. The patient was transferred to the hyperbaric unit on a daily basis by ambulence with full intensive care support and accompanied by a consultant intensivist. No problems were encountered during transfer. HBO treatments were discontinued after 16 days and a total of 22 treatments at which time the Hb was 3.6 g.dl⁻¹ and the patient showed no significant deterioration without treatment.

The haemoglobin had continued to fall, reaching 2.0 g.dl⁻¹ by day 4. The subsequent rise in haemoglobin was slow initially (Fig. 1), despite a good reticuloocyte response. The delay in response of the marrow was suspected to be due to sepsis, the white cell count and temperature being raised throughout this period. Paracetamol and surface cooling were used to control the temperature. A methicillin-resistant Staphylococcus aureus was identified in sputum and from a central venous catheter tip. Appropriate antibiotics were administered.

From day 4 to day 20 the serum sodium concentration was consistently raised (150–164 mmol.l⁻¹). We have no good explanation for this phenomenon but note that it has
Table 1 Changes in cardiac output and systemic vascular resistance before, during and after hyperbaric oxygen (HBO) treatment

<table>
<thead>
<tr>
<th></th>
<th>Adrenaline (µg min⁻¹)</th>
<th>Heart rate (beat min⁻¹)</th>
<th>Cardiac index (L min⁻¹ m⁻²)</th>
<th>SVRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-HBO</td>
<td>6</td>
<td>114</td>
<td>7.6</td>
<td>721</td>
</tr>
<tr>
<td>First treatment at 2 ATA</td>
<td>6</td>
<td>107</td>
<td>5.4</td>
<td>1007</td>
</tr>
<tr>
<td>Immediately post treatment</td>
<td>4</td>
<td>104</td>
<td>4.6</td>
<td>1139</td>
</tr>
<tr>
<td>Second treatment at 2 ATA</td>
<td>4</td>
<td>104</td>
<td>4.3</td>
<td>1340</td>
</tr>
<tr>
<td>Immediately post treatment</td>
<td>3</td>
<td>102</td>
<td>4.4</td>
<td>1364</td>
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<tr>
<td>2 h post treatment</td>
<td>3</td>
<td>92</td>
<td>5.2</td>
<td>962</td>
</tr>
<tr>
<td>10 h post treatment</td>
<td>3</td>
<td>113</td>
<td>7.3</td>
<td>614</td>
</tr>
</tbody>
</table>

SVRI = indexed systemic vascular resistance (dynes cm⁻⁵ m⁻²).

been observed previously in the literature [1]. On day 15, a percutaneous tracheostomy was performed and on day 22 weaning was commenced. On day 28, sedation was weaned, and by day 31, 1 month from admission, the patient was able to sit out in a chair.

Neurological assessment at this time revealed lower limb weakness, greater on the right than on the left, with pronounced right foot drop, marked discomfort and dysesthesia under the right foot, and loss of position and vibration sense at the right halluc. There were no other neurological symptoms or signs above the knees and she was mentally alert and surprisingly cheerful. A magnetic resonance scan and EMG studies were performed. The EMG studies showed evidence of pressure damage to the sciatic nerves, principally right-sided. The magnetic resonance scan of the brain showed a small right cerebellar infarct, and unexpectedly, appearances in the left parietal region consistent with a meningioma.

Erythropoietin was discontinued after 38 days at which time her Hb was 7.6 g dl⁻¹. On day 39, the patient was stable enough to be discharged to a high-dependency unit, where she remained for a further week before being moved to a general ward. The rest of this woman’s hospital stay was uncomplicated and consisted principally of physiotherapy and occupational health input. One hundred and fourteen days after admission she was discharged home. At this point there was still pain from the right foot and the patient needed a walking stick for mobilisation. Haemoglobin concentration on discharge was 11.3 g dl⁻¹. She has subsequently had the meningioma removed uneventfully.

Discussion

The prime consideration in severe acute anaemia is maintaining an adequate tissue PaO₂, despite the reduced oxygen-carrying capacity of the blood. Care is taken therefore to minimise oxygen consumption and maximise oxygen delivery.

Blood loss was minimised postoperatively in this patient by aggressive correction of clotting times, the use of paediatric-size blood-sampling tubes and rationalisation of the frequency of sampling. Tranexamic acid acts by competitively inhibiting the activation of plasminogen to plasmin. Aprotinin is an inhibitor of proteolytic enzymes including trypsin, plasmin and both plasma and tissue kallikrein. To maximise erythropoiesis the patient was fed early and given iron supplements (parenterally initially then enterally as gut function recovered). Haematinics were given, as was erythropoietin [2, 3]. Despite this, as can be seen from Fig. 1, the rise in haemoglobin was disappointingly slow.

Oxygen consumption was decreased by sedation, muscle relaxation and treatment of pyrexia with paracetamol and surface cooling. Oxygen delivery may be improved by an increase in the cardiac output or increasing the oxygen content of blood. This is normally achieved by increasing haemoglobin concentration. When this facility is not available, oxygen content can be increased by increasing inspired oxygen partial pressure. The amount of oxygen dissolved in plasma in physical solution is determined by Henry’s Law, and the content may be increased to useful values by the use of HBO therapy. At 3 ATA absolute of 100% oxygen the physically dissolved oxygen can supply the

![Figure 1](image)

Figure 1 Haemoglobin and reticuloocyte response. (●) [Hb], g dl⁻¹; (■) reticulocytes, %.
basic metabolic needs of the body and the Hb of the mixed venous blood is therefore completely saturated [4].

One of the main potential adverse effects of hyperbaric oxygenation is oxygen toxicity [1, 5]. This has two main clinical manifestations. Acute oxygen toxicity affects the central nervous system (CNS) and results in convulsions. This effect generally only occurs at higher partial pressures of oxygen (> 1.8 ATA) and is dose/time dependent. The risk can be minimised by limiting the duration of continuous oxygen exposure. Usually this is achieved by employing 'air breaks' at regular intervals during the HBO treatment. Subacute or pulmonary oxygen toxicity affects the respiratory system and can result in a picture of acute lung injury with reduced vital capacity, reduced compliance and impaired gas exchange. These effects can occur at lower oxygen partial pressures (above 0.5 ATA) but require longer exposures. Risk of pulmonary oxygen toxicity can be assessed by measuring the unit pulmonary toxic dose (UPTD), a method which expresses hyperbaric oxygen exposure in terms of an equivalent exposure in minutes at 1.0 ATA [6]. A UPTD of 615 produces an average reduction in vital capacity of 2%, which is completely reversible. A UPTD of 1425 produces an average reduction in vital capacity of 10%, which although reversible, would require a longer period for recovery [7]. Effects of repetitive exposures are cumulative, but this is reduced by an interval between exposures [8, 9].

In order to optimise oxygen delivery while minimising risks of toxicity, a treatment protocol using HBO at 2 ATA lasting for 90 min (with a 5-min air break) was selected. This produced a UPTD of 232 for each treatment. Treatments were given three times per day initially then, as haemoglobin concentration recovered, treatment frequency was reduced. There was no evidence at any time of oxygen toxicity either in terms of sympathetically mediated signs of convulsive activity, or deterioration of respiratory function. On treatment blood pressure rose, heart rate decreased and cardiac output fell. ECG ischaemic changes resolved and acidosis on arterial blood gas measurement improved. These changes persisted for some time after each treatment, presumably because HBO reverses the oxygen debt and saturates the tissues with oxygen. There is a demonstrated association between duration of oxygen debt and both organ failure and death [10, 11]. Several patients have been treated on similar regimens in the USA and their experience has been similar; in general the treatments were well tolerated and no oxygen toxicity was reported [12, 13].

Another potential adverse effect of hyperbaric oxygen treatment is barotrauma. Care must be taken to avoid pulmonary barotrauma and tension pneumothorax during 'ascent' and particular vigilance needs to be maintained to detect and treat this if it should happen. Middle ear and sinus barotrauma are more common, but less serious complications. One concern is that many HBO units are not set up adequately to deal with a critically oxygenated ICU patient [14]. Transfer and treatment should be carried out with the same level of care maintained as on an ICU. This requires invasive haemodynamic monitoring, measurement of oxygen saturation, availability of blood gas analysis and other tests, syringe drivers safe for use at pressure and a ventilation system designed to be used at pressure. Most importantly, the patient should be accompanied at all times by an ICU specialist with experience of HBO treatment.

According to the Report on Confidential Enquiries into Maternal Deaths in the United Kingdom 1991–93 [15], three women who refused blood transfusion on religious grounds died from obstetric haemorrhage during this period. The report contains a chapter dealing specifically with this issue and making a number of recommendations regarding the management of pregnant women who will not accept blood transfusion, and their management in the event of obstetric haemorrhage. The potential role of hyperbaric oxygen in this situation is acknowledged.

The mainstay of care in a severely anaemic patient who refuses transfusion is sedation and artificial ventilation to minimise oxygen requirements, while at the same time optimising oxygen delivery and supporting erythropoiesis. Despite this, oxygen debt may arise. Hyperbaric oxygen therapy can be a useful adjunct acting by reversing oxygen debt by an easily understood mechanism with a good safety record. We believe that without this treatment this patient would have continued to deteriorate as she had done prior to its institution.

References
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CASE REPORT

Profound motor blockade with epidural ropivacaine following spinal bupivacaine

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Summary

Ropivacaine, a relatively new amide local anaesthetic, reputedly produces less motor block than equivalent doses of bupivacaine, potentially combining high-quality analgesia with the ability to ambulate. We report two cases of prolonged, profound motor block with patient-controlled epidural analgesia using 0.1% ropivacaine, following spinal bupivacaine for Caesarean section. As there was no evidence of inadvertent intrathecal ropivacaine administration or of any neurological injury, we hypothesise that epidural ropivacaine may interact with intrathecal bupivacaine to prolong its effect.

Keywords Anaesthesia; spinal, epidural. Complications; motor block. Local anaesthetics; ropivacaine, bupivacaine.

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Ropivacaine is the most recent amide local anaesthetic to be introduced into clinical practice. It was developed with a view to reducing systemic toxicity and increasing differential sensitivity to sensory nerves in comparison with bupivacaine, the current standard local anaesthetic for epidural administration [1, 2]. Previous studies have suggested that continuous epidural administration of 0.2% ropivacaine compares favourably with bupivacaine and can produce satisfactory postoperative analgesia with relative sparing of motor blockade, potentially facilitating earlier