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Use of sophisticated intra-operative monitoring in resuscitation of unexpected cardiovascular collapse during general anaesthesia

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ABSTRACT

INTRODUCTION: The diagnosis of intraoperative anaphylaxis is important but can be difficult as the symptoms can be varying and dependent on patient factors.

PRESENTATION OF CASE: We describe an acute, unexpected and life threatening cardiovascular (CV) collapse, presumed to be due to an acute anaphylactic reaction secondary to gelatin administration, following induction of general anaesthesia (GA), in an ASA 3 patient scheduled for axillo-bifemoral bypass.

DISCUSSION: The management of the profound cardiovascular (CV) collapse was greatly assisted by sophisticated haemodynamic, depth of anaesthesia and cerebral oximetry monitoring.¹ As far as we are aware this is the first such case where the full haemodynamic, depth of anaesthesia and cerebral oxygenation changes during CV collapse, presumed due to an acute anaphylactic reaction under GA have been fully documented.

CONCLUSION: The use of advanced monitoring intraoperatively proved extremely useful in guiding the resuscitation of a life threatening allergic reaction under general anaesthesia.

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1. Introduction

Allergic reactions and histamine release following the use of modified gelatins have been well described in the literature following the pioneering work of Lorenz et al.² The diagnosis of intraoperative anaphylaxis is important but can be difficult as the symptoms can be varying and dependent on patient factors.³ Also, the different shock states can present in a similar way and GA can disguise some of the symptoms. This case report is a useful illustration of the value of invasive monitoring in high-risk cases.

2. Presentation of case

Mrs. A was a 59-year-old ASA 3 female patient who presented for elective axillo-bifemoral bypass surgery. She was a known smoker with chronic obstruction pulmonary disease (COPD) and asthma and had suffered a recent myocardial infarction (MI) requiring insertion of a drug eluting stent 3 months earlier. However, she had a moderate exercise tolerance and had received several previous uneventful GAs for other vascular surgeries. She had no known allergies. Her medications included aspirin, clopidogrel, beta-blockers, calcium antagonists and bronchodilators. Her pre-operative blood tests were in the normal range. Her ECG showed ST elevation in the inferior leads.

In addition to conventional monitoring, as specified in national guidelines, it is our practice in high-risk vascular patients to

commence monitoring of haemodynamics (LiDCOrapid, LiDCO PLC, UK), depth of anaesthesia and cerebral oximetry (BIS and Invos, Covidien, USA) prior to induction of anaesthesia. The use of these monitors in combination during high-risk surgery has been the subject of a recent review.¹

Anaesthesia was induced and maintained with total intravenous anaesthesia (TIVA) using a target controlled infusion (TCI) of remifentanyl and propofol. Following 200 µg of glycopyrronium, remifentanyl was administered to achieve an estimated effect site concentration (Ce_{ff}) of 2 ng ml⁻¹ and propofol was then titrated to achieve a BIS level of 45 (Ce_{ff} of 2.5 mcg ml⁻¹) (Fig. 1). 6 mg of cisatracurium was then given. A size 4 laryngeal mask airway (LMA Supreme, Intaventdirect, UK) was inserted 3 min later and her lungs were mechanically ventilated using a tidal volume of 8 ml kg⁻¹, at a rate of 8 times per minute.

Mean arterial blood pressure (MAP) fell from 94 mmHg pre-induction (5, Fig. 2) to 53 mmHg 9 min post induction. Since stroke volume variation (SVV, an indicator of fluid responsiveness⁴) had risen to 15%, the decision was taken to administer a fluid challenge of 200 ml (5 ml kg⁻¹) of gelatin (Volplex®, UK) by rapid intravenous injection using a 50 ml syringe (6, Fig. 2). Following the gelatin, Mrs. A's MAP dropped further with a big fall in SV associated with profound tachycardia. The latter initially resulted in an increase in cardiac output (CO) but as the SV continued to fall together with the heart rate, the CO fell. Anaphylaxis was the presumptive diagnosis (7, Fig. 2). An infusion of adrenaline was commenced (8, Fig. 2) with little effect. Noradrenaline infusion was then started (12, Fig. 3).

Propofol TCI was reduced to a target of 1 mcg ml⁻¹ to maintain BIS in the desired range of 40–55 (Fig. 1), on the assumption

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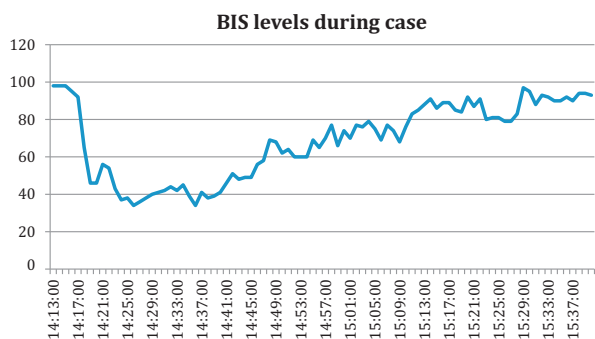


Fig. 1. BIS values for whole case (for explanation, see text).

that propofol metabolism would be significantly decreased due to the reduction in CO and liver blood flow.⁵ Cerebral oxygenation (rSO_2) diminished markedly (Fig. 4) and reached its lowest level (50% below baseline) when systolic blood pressure fell to 32 mmHg. Noradrenaline infusion restored haemodynamics (Fig. 3) and cerebral rSO_2 (Fig. 4) to pre-induction values suggesting a full return of cerebral perfusion. The decision was taken not to continue with the procedure so the propofol and remifentanyl infusions were stopped and she was allowed to awaken. She was extubated and taken to the high dependency unit (HDU) where she remained overnight. She returned to the ward the following day and was discharged from the hospital 1 day later.

Blood samples were taken for mast cell tryptase and the immediate sample showed a raised value of 80 with subsequent values falling back to baseline confirming that the cardiovascular collapse was most likely to be due to anaphylaxis.⁶ She was also referred to the allergy clinic and informed of the importance to attend the clinic to confirm that this reaction was caused by gelatins and not by any of the other anaesthetic agents given

to her. Unfortunately she did not attend her clinic appointment on 3 separate occasions, so a definitive cause of her anaphylactic reaction could not be made. However, gelatin seemed the most likely cause as she had received propofol, opioids and cisatracurium previously during her other GAs with no problems.

Mrs. A eventually underwent a successful axillo-bifemoral bypass under local anaesthesia one month later.

3. Discussion

The differential diagnosis of the sudden and profound CV collapse in this patient following gelatin made anaphylaxis the most likely cause. Besides anaphylaxis, myocardial ischaemia causing cardiogenic shock was also considered due to her recent MI. However, the rapid recovery of the patient suggests that this was not the case. Also, troponin levels obtained post operatively in HDU were in the normal range. An anaphylactic reaction should always be considered if there is continuation or exacerbation of hypotension following the administration of gelatin since it is a common cause of anaphylaxis. Patients with significant cardiac disease are obviously less able to withstand the haemodynamic insult caused by anaphylaxis as in this case.

According to the Association of Anaesthetists of Great Britain and Ireland (AAGBI) guidelines of treatment of anaphylaxis, adrenaline is the drug of first choice in management. However, since adrenaline did not produce improvement in cardiovascular status, noradrenaline infusion was commenced. Vasopressin should also be considered when attempting to reverse refractory hypotension in this situation and is less likely to exacerbate the tachycardia.⁷

The management of profound CV collapse was greatly assisted by sophisticated haemodynamic, depth of anaesthesia and cerebral oximetry monitoring.¹ Having a BIS reading was very useful

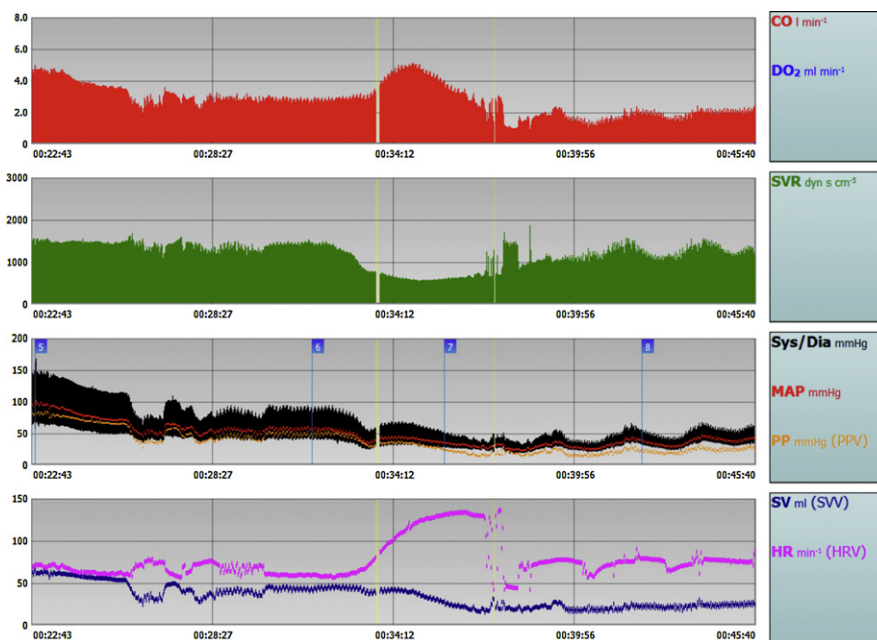


Fig. 2. LiDCO values during profound cardiovascular collapse following gelatin administered at point (6), third trace A presumptive diagnosis of anaphylaxis was made at (7) and adrenaline infusion commenced at (8). Data obtained post hoc using LiDCO view pro® software. Key to this figure. Haemodynamic values are displayed on the Y axis and time in minutes on the X axis. The top trace (red) indicates nominal (n) cardiac output in lpm. The second trace (green) indicates systemic vascular resistance (SVR). The third trace (black) indicates blood pressure, systolic, diastolic and mean (red line). The fourth trace indicates nominal (n) stroke volume (blue) and heart rate (purple).

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