Death associated with disseminated intravascular coagulation after hip replacement

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Summary

Sudden-onset disseminated intravascular coagulation (DIC) occurred 3 h after uneventful polymethylacrylate bone cement insertion for a revision left Charnley hip replacement. Profuse bleeding caused a hypovolaemic state aggravating existing myocardial ischaemia; as a result death occurred secondary to myocardial infarction 6 h after operation. (Br. J. Anaesth. 1998; 80: 853–855)

Keywords: complications disseminated intravascular coagulation; complications myocardial ischaemia; surgery orthopaedic bone cement insertion

Immediate cardiopulmonary effects are the most common adverse reactions to methylmethacrylate (MMA) bone cement insertion. In the absence of hypovolaemia, hypotension is regarded as uncommon (incidence less than 5%) in elective hip surgery. Hypoxia and hypotension are more common in patients who are elderly, have pre-existing hypertension, uraemia or are having emergency surgery for transcervical femoral fractures. The aetiology of these reactions is thought to be pulmonary microembolism of fat and thromboplastic products.

A previously unreported manifestation of cement insertion, the delayed onset of sudden catastrophic haemostatic failure, is presented.

Case report

An 88-year-old woman was admitted electively for revision hip surgery. She had previously had two cemented prostheses inserted; loosening of the cemented prosthesis on the left had necessitated a revision.

On preoperative assessment she was noted to have a history of rheumatic fever, a cerebrovascular accident and a partial gastrectomy for drug-induced ulceration. She was receiving aspirin 75 mg. The patient weighed 35 kg with a body mass index of 15. On preoperative assessment she was noted to have a heart rate of 120 and blood pressure was 120/70 mm Hg. She was in atrial fibrillation with a heart rate of 120

The intraoperative course was unremarkable, apart from a rise in blood pressure to 200/100 mm Hg during the initial skin incision, with S-T segment depression on the electrocardiogram; the blood pressure and ischaemic changes were quickly controlled using an i.v. infusion of glyceryl trinitrate. There was no significant change in blood pressure, heart rate, arterial oxygen saturation (SpO2), or end-tidal carbon dioxide (PETCO2) with reaming or cement insertion into the acetabulum or femur. Central venous pressure was 7–10 mm Hg throughout the procedure. Blood loss during the 155-min procedure was estimated to be less than 500 ml. Intraoperative fluids were 2 l of Hartmann’s solution and 500 ml Haemaccel. A unit of blood was started immediately the operation was finished.

After an uneventful 90 min in the recovery room, the patient was transferred to an intensive care area for monitoring and management of extradural analgesia. On arrival there it was noted that her axillary temperature was 32.5°C and 400 ml of blood had drained from the wound drain since skin closure. A Bair Hugger warming blanket was applied and an epidural infusion started using fentanyl 15 μg h−1 and bupivacaine 7.5 mg h−1. Soon after transfer to the intensive care area, the patient’s blood pressure decreased to 64/32 mm Hg. This responded to a further unit of blood and 500 ml of Haemaccel, which increased the central venous pressure from 3 to 10 mm Hg, and discontinuing the glyceryl trinitrate infusion. However 25 min later the blood pressure was 85/60 mm Hg with associated S-T segment depression on the ECG. There was no evidence of a low intravascular volume, with central venous pressure 8 mm Hg, heart rate 80 beats min−1 and no

final dose of 0.25 mg was omitted, as the heart rate was 65 min−1 at 08:00 on the morning of surgery. No premedication was given. At induction of anaesthesia the blood pressure was 116/70 mm Hg and heart rate 100 beats min−1. After instigation of non-invasive monitoring and preoxygenation, anaesthesia was induced. An epidural catheter was sited at L1–L2 and a central venous pressure line inserted. Anaesthesia was maintained during intermittent positive pressure ventilation with nitrous oxide 66%, oxygen 33%, isoflurane 0.4–1.2%.

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new loss into the wound drains. The extradural infusion was stopped and metaraminol 1 mg in increments of 0.5 mg given i.v. This produced an immediate rise in blood pressure to 120/70 mm Hg.

Sudden reduction in systolic blood pressure to 40 mm Hg occurred 15 min later, and 1800 ml of unclotted, partially haemolysed blood was noted in the previously empty drains. A provisional diagnosis of disseminated intravascular coagulation (DIC) was made and a coagulation screen and fresh frozen plasma and platelets requested. In spite of vigorous fluid resuscitation with three units of blood and increments of epinephrine to a total of 0.5 mg, an asystolic cardiac arrest occurred. After a bolus of atropine 3 mg and a bolus of epinephrine 2 mg, a rhythm of atrial fibrillation with intermittent idioventricular rhythm was established. Arterial blood gases showed pH 7.473, \( P_{a\text{CO}_2} \) 3.42 kPa, \( P_{a\text{O}_2} \) 10.2 kPa, base deficit \(-3.2\) mmol \(\text{L}^{-1}\), and haematocrit 26%. An epinephrine infusion was started (15 \( \mu \text{g} \min^{-1} \)) and 1.5 l of hydroxyethyl starch 6% (Hespan) given to keep the central venous pressure at 8–10 mm Hg. Thirty min after the cardiac arrest the blood pressure was 110/60 mm Hg, central venous pressure 12 mm Hg and heart rate 120 beats \(\text{min}^{-1}\) (atrial fibrillation with a left bundle branch block), but there was oozing from venepuncture sites, haematuria and no clot in the wound drains. Clotting screen from the time of cardiac arrest showed a platelet count of \(<10 \times 10^9\) \(\text{L}^{-1}\), haemoglobin 9.9 g dl\(^{-1}\), prothrombin time (PT) seven times normal and thrombin time 40 s (control 14 s). Activated partial thromboplastin time (APTT) had no end point. Six units of platelets and four units of fresh frozen plasma were given.

Over the next 60 min, in spite of an epinephrine infusion (50 \( \mu \text{g} \text{min}^{-1} \)), the patient had repeated asystolic cardiac arrests. She was hypoxic and acidotic on 100% oxygen; arterial blood gases showed pH 7.08, \( P_{a\text{CO}_2} \) 5.3 kPa, \( P_{a\text{O}_2} \) 6.40 kPa, and base deficit \(-13.4\) mmol \(\text{L}^{-1}\). Repeat coagulation screen showed continuing consumption of clotting factors, with a haemoglobin of 5.5 g dl\(^{-1}\), platelets \(17 \times 10^9\) \(\text{L}^{-1}\), APTT >240 s, and PT 9.8 times normal. Three more units of blood were given while awaiting delivery of further clotting factors. Asystole progressed to electromechanical dissociation, and 215 min after the patient was admitted to intensive care it was decided to discontinue resuscitation as further attempts were unlikely to be beneficial.

The postmortem examination showed death to have been caused by myocardial infarction; the anterior two-thirds and posterior third of the left ventricle showed recent evidence of infarction, without demonstrable occlusive coronary atheroma. There was no significant aortic valve disease, but mitral valve thickening and incompetence consistent with rheumatic fever were noted.

**Discussion**

The cause of death was an extensive myocardial infarction. Although myocardial ischaemia was associated with the hypertension associated with skin incision, this was quickly controlled with glyceryl trinitrate. After operation, there were the additional stresses of hypothermia (axilla temperature 32.5 \(^{\circ}\text{C}\), anaemia and hypovolaemia. Hypovolaemic shock precipitated by a sudden onset coagulopathy was the main factor responsible for the myocardial infarction which resulted in her death.

The possible causes of disseminated intravascular coagulation in this patient are fat emboli from the bone cement and prosthesis insertion, delayed hypersensitivity reaction to bone cement, a transfusion of incompatible blood, and sepsis. In addition, the haemodilutional effects of the administration of 4.5 l of fluid, the effects on coagulation of administration of 1.5 l of Hespan and moderate hypothermia, need to be considered.

The contribution of direct microvascular occlusion by fat emboli to the myocardial ischaemia is unknown, as evidence for this was not specifically sought during the postmortem examination. Fat globules were, however, detected in the plasma and urine, indicating that fat embolism had taken place, although no petechiae were noted. None of the organ samples obtained at autopsy were stained for the presence of fat. Haematological derangement of this magnitude has not been previously noted in studies of fat embolism after lower limb fractures or hip surgery.

There have been conflicting reports of the magnitude and significance of histamine release in response to cement insertion. However Monteny felt that a severe hypersensitivity reaction to methylmethacrylate cement was unlikely to cause severe coagulopathy. Enquiry to the manufacturers of acrylic cement (Palcos R with gentamicin, Schering-Plough, Mildenhall, Suffolk) revealed no experience of this complication.

The blood bags of the transfused units were returned to the laboratory, and testing revealed no evidence of bacterial infection or incompatibility. The transfusion therefore appeared unlikely to be the cause of the haemolysis and DIC. There was no evidence of sepsis from the preoperative urine, nasal swabs or postmortem examination.

Of the other possible contributing factors, hypothermia is known to cause a thrombocytopenia proportional to its degree. At 32 \(^{\circ}\text{C}\) a 30–50% reduction in platelet count could be expected because of sequestration in the liver and gut. Before the coagulopathy became evident the patient had received 2 l of Hartmann’s solution and 1 l of Haemaccel, resulting in significant dilution (estimated blood volume being 2450 ml); the haematocrit at this point was 0.26. Haemaccel has not been associated with any interference with coagulation. However, after the onset of the coagulopathy, 1.5 l of Hespan was administered before blood became available. As well as diluting clotting factors, this has been observed to decrease the concentration of factor VIII and von Willibrand’s factor, prompting the recommendation to limit administration to 20 ml kg\(^{-1}\) day\(^{-1}\). Recent studies have not found any prolongation of clotting times or reduction in platelet count after using 1 l of Hespan in cardiac surgery, neurosurgery or gynaecology. However, Boldt and colleagues found a decrease in platelet aggregation associated with an increase in blood loss when they gave Hespan to cardiac surgery patients.

Profound coagulation failure occurred in the initial postoperative period. There was evidence of fat in the urine and sputum at postmortem examination. The combination of fat emboli, haemodilution,
hypothesis and possibly the effect of Hespan administration is most likely to have triggered the coagulopathy that resulted in haemorrhagic shock and myocardial infarction. The relative importance of each factor could not be ascertained. More rapid access to coagulation factors, which have to be requested and then sent by road from the regional transfusion centre, may have allowed the coagulopathy to be controlled earlier before major blood loss and myocardial ischaemia occurred.

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References