

## CASE REPORT

# Anaphylactic reaction after autologous blood transfusion: A case report and review of the literature

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## ABSTRACT

Autologous blood transfusion as a cause of intraoperative anaphylaxis is very rare. We encountered one such life-threatening event in a 72-year-old patient undergoing laminectomy and pedicle screw fixation. The probable cause identified was the floseal mixed autologous blood transfusion. Review of literature has been done, and measures to avoid such an event in the future are discussed.

**Key words:** Anaphylaxis, autologous, blood transfusion, cell saver, neurosurgery

## Introduction

Anaphylactic reaction may occur intraoperatively, generally on reexposure to a specific antigen and requires the release of proinflammatory mediators. Anaphylactoid reactions occur through a direct non-immunoglobulin E-mediated release of mediators from mast cells or from complement activation. Autologous blood transfusion as a cause of intraoperative anaphylaxis is very rare. We encountered one such life-threatening event in a 72-year-old patient undergoing laminectomy and pedicle screw fixation.

## Case Report

A 72 years male, American Society of Anesthesiologists Grade 2 was scheduled for L2–L3 laminectomy and pedicle screw rod fixation under general anesthesia (GA) for L2–L3 Posterior inter vertebral disease. He had a history of L3–L4–L5 laminectomy, discectomy and pedicle screw rod fixation done under GA 6 years back. Patient was known

hypertensive controlled with medications since last 15 years and had a history of left anterior coronary artery stenting 3 years back. The patient was on tablet amlodipine (2.5 mg), losartan (50 mg), atorvastatin (10 mg), aspirin (325 mg) and clopidogrel (75 mg) OD. Preoperatively he had 10% sensory loss over L5 dermatome; otherwise his vital parameters were within normal limits. The patient's electrocardiogram (ECG) was normal and had a left ventricular ejection fraction of 55% with no regional wall motion abnormality in two-dimensional echo. We premedicated the patient with inj glycopyrrolate 0.2 mg intramuscularly. The patient had a baseline pulse rate, noninvasive blood pressure (BP) and SpO<sub>2</sub> of 60/min, 130/76 mmHg, and 100%, respectively. Anesthesia was induced with fentanyl, propofol, rocuronium and was maintained with fentanyl, vecuronium, sevoflurane, nitrous oxide and oxygen. Intra-arterial and central venous cannulation were done after anesthesia induction and patient was made prone after the induction. During the course of surgery there were 3 episodes of hypertension with tachycardia, all managed with ensuring adequate depth of anesthesia. In view of expected major blood loss, cell saver was used during the surgery. Blood loss during the surgery was approximately 2200 ml, and we administered 4 L crystalloid and 1500 ml of hexa ethyl starch (HES) and 3 units of packed red blood cell. There were no hemodynamic changes during allogeneic blood transfusion. Toward the conclusion of surgery at the time of hemostasis, floseal (floseal<sup>®</sup> hemostatic matrix [it consists of a bovine-derived gelatin matrix component, a human-derived thrombin component. Baxter; Deerfield, IL, US]<sup>[1]</sup>) was used for hemostasis. A thorough wash was given but at this time a sudden venous bleed occurred. In view of a hemodynamic threat, we had to administer 430 ml of autologous blood collected from the compact advanced cell saver surgical

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autotransfusion machine (Dideco Electa [Sorin Group, Italy]).<sup>[2]</sup> The patient had stable hemodynamics throughout the surgery but toward the end of the surgery (around 2 h after administration of allogeneic blood and HES) and after few minutes of starting autologous blood, there was sudden fall in BP (70/46 mmHg) and tachycardia (heart rate [HR] 130/min) was observed. We did not notice any change in end-tidal CO<sub>2</sub> and ECG, but the airway pressure increased to 20 mmHg from 17 mmHg. Immediately blood transfusion was stopped, fluid bolus, 100% oxygen and injection phenylephrine was administered (100 µg increments, total 500 µg over 10 min). With these interventions, the BP increased to 78/50 mmHg but did not reach the base line and still there was tachycardia (125/min). By the time skin closure was completed, and when the surgical drapes were removed, we noticed erythematous patches throughout the body in addition to facial edema. However, no hematuria was noticed. With a strong suspicion of anaphylaxis 1000 ml of crystalloid solution and intravenous hydrocortisone (300 mg), chlorpheniramine (25 mg) and epinephrine (100 µg) were administered. The BP increased to 80/51 mmHg with HR of 132/min; the patient was still prone, and infusion of epinephrine was started at 5 µg/min. Arterial blood gas report at that time was normal and the patient was made supine from the prone position, and the BP improved to 107/66 mmHg, with HR of 106/min. The troponin T done in the operation room was normal. The patient observed for 30 min more after starting adrenaline infusion and the BP increasing to 128/76 with HR 96/min. The patient was shifted to the Intensive Care Unit (ICU) where he was kept on mechanical ventilation. The patient's vitals were stable in ICU, and the infusion of epinephrine was tapered off over 3 h. A 12 Lead ECG and troponin T done in ICU was normal. The patient's trachea was extubated in the next morning; he remained stable in the ICU for next 24 h. The patient was shifted from ICU on the 2<sup>nd</sup> day and was discharged from the hospital on 7<sup>th</sup> day.

## Discussion

Cardiovascular symptoms (73.6%), cutaneous symptoms (69.6%), and bronchospasm (44.2%) are the most common clinical features of intraoperative anaphylaxis.<sup>[3]</sup> Intraoperatively under anesthesia the early cutaneous signs of anaphylaxis are often unrecognized, leaving bronchospasm and cardiovascular collapse as the first recognized signs of anaphylaxis.<sup>[4]</sup> In our case also it was cardiovascular symptoms that raised the alarm first. The possible causes of the clinical presentation in our case could have been either mismatched transfusion reactions or an anaphylaxis. We ruled out transfusion reaction due to allogeneic blood to be the cause as there were no signs of hemodynamic alteration even after 2 h of allogeneic blood transfusion, and there was no hematuria. In our case, we suspected an anaphylactic reaction just after transfusion of autologous blood from the cell saver. The common agents reported responsible for intraoperative anaphylaxis are muscle

relaxants (mainly succinylcholine, rocuronium, atracurium, natural rubber, antibiotics like penicillin and beta-lactams, hypnotics like propofol and thiopentone, colloid solutions such as dextran and gelatins and opioids like morphine and meperidine.<sup>[3]</sup> Muscle relaxants and HES cannot be implicated to be the cause for anaphylaxis as there were no signs of hemodynamic disturbance after their repeated administration intraoperatively. The possible cause of anaphylaxis in our case could be the hemostatic agent used during the surgery, which when mixed with the autologous blood, remained unfiltered and subsequent transfusion resulted in an anaphylactic reaction. All precautions were taken to avoid this mixing but due to the inadvertent intraoperative situation, there is a possibility of miniscule quantity of floseal getting sucked into the cell saver. Anaphylaxis may occur because of animal gelatin components of topical hemostatic agents. Spencer *et al.* reported occurrence of an abrupt hypotension, ventilatory difficulty and erythematous rash after use of floseal hemostatic matrix in patient of spinal fusion, which was treated with epinephrine and dexamethasone.<sup>[5]</sup> In another case report, an anaphylactic reaction was noticed 1/2 h after autologous blood transfusion in an 11-year-old female. The postoperative laboratory data in that case revealed an elevated bovine gelatin specific IgE.<sup>[6]</sup> The fragments of collagen-based hemostatic agents can pass through 40 µm transfusion filters of blood scavenging systems (Dideco Electa cell saver). Hence, in our case probably the fragments of floseal were aspirated into the cell saver at the time of application which were not filtered properly by the 40 µm transfusion filter and subsequently caused anaphylaxis when administered along with autologous blood. Previous reports have focused on the thrombin components, and care should be taken in the administration of these products, particularly in the atopic individual and not to aspirate floseal matrix into autologous blood salvage circuits.<sup>[5-8]</sup> Thus with this case report we want to emphasize upon the fact that hemostatic agents can pass through the cell saver unfiltered and can mix with the autologous blood causing subsequent anaphylaxis upon transfusion. Caution should be exercised while transfusing autologous blood to a patient from cell saver. The hazardous complications can be avoided. Surgeons should avoid suctioning into the cell saver suction tubing after they have applied the floseal or any other such hemostat, which might be suctioned along with the blood and can in turn be transfused to the patient without being filtered. The cell saver suction should not be used at the time of bone drilling and when using bone cement, as these also can have the same outcome as floseal has when transfused along with the autologous blood from cell saver. The blood from cell saver should be transfused slowly, and caution should be exercised as when transfusing the allogeneic blood.

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