

Unexpected Coagulation in the Bypass Pump Circuit

Case Report

Volume 2 Issue 4 - 2015

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Received: February 24, 2015 | **Published:** April 09, 2015**Abstract**

We present a case of premature coagulation in the bypass pump circuit. Fifty-nine years-old female (Euroscore: 5) presented with acute heart failure and pulmonary edema due to mitral valve stenosis. Following one vessel coronary bypass and mitral valve replacement (cross clamp time: 170 minutes), the blood collected before heparinization had to be transfused due to persistent bleeding. A short while after the transfusion, we had to initiate the backup bypass machine due to coagulation in the pump circuit. We are in the opinion that the whole blood with fresh coagulation factors activated the intrinsic pathway in the pump circuit and caused the coagulation.

Keywords: Coronary artery bypass surgery; Acute normovolemic hemodilution; acute/complication; Coagulation

Abbreviations: CPB: Cardiopulmonary Bypass; ACT: Activated Coagulation Time

Introduction

Hemodilution attenuates the adverse effects of hypothermia during cardiopulmonary bypass (CPB) on tissue perfusion [1]. While the priming volume of the bypass machine circuit provides hemodilution, acute normovolemic hemodilution is used to protect some portion of the patient's blood and to reduce need for packed blood products [2,3]. We present a case, where in our opinion; the whole blood collected from the patient caused premature coagulation in the bypass pump circuit.

Case Presentation

Fifty-nine years-old female (body mass index: 27.5 kg/m², body surface area 1.61 m²) presented with insulin-dependent diabetes mellitus, acute heart failure (EF: 65%) and pulmonary oedema, left ventricular concentric hypertrophy; coronary angiography demonstrated 80% stenosis in the proximal left anterior descending artery, and symptomatic mitral valve stenosis (Euroscore: 5). She was scheduled for mitral valve replacement and one vessel coronary bypass with general anesthesia. Preoperative hematocrit was 38%. A half liter of whole blood was collected, replaced with 500 ml isotonic fluid (0.09% NaCl) and stored in the refrigerator door, before heparinization with 350 U/kg (ACT 670 s, Hemo chron Celite). After normovolemic hemodilution, the hematocrit was 34%. CPB was established in a standard fashion (Dideco Compact flo Evo Phisio/M, body temperature: 28°C, hematocrit: % 28). Pump flow of 3.8 l/min and inspired oxygen fraction of 45% were required to provide sufficient perfusion at mean arterial pressure of 50 mmHg, monitored with cerebral pulse oximetry (basal value 81%). Following single coronary bypass grafting (Ao-OM1, 2 mm), left atriotomy was performed. Severe calcifications of the mitral annulus and apparatus were debrided and replaced with No: 25 SorinCarbomedics metal valve. Cross clamp time was 170 minutes, total urine during the perfusion was 1700 ml.

Due to a persistent leakage of blood, complete hemostasis could not be achieved for about an hour, despite topical use of tranexamic acid. The ACT was within 850-1000 s during this period, and two red blood cell packs were used to keep the

hematocrit between 21-23%. We ordered more red blood cell packs. A short trial to increase the mean blood pressure above 55 mmHg to increase the tissue perfusion aggravated the bleeding further. Therefore we kept the mean blood pressure at 50-55 mmHg. The patient became anuric during this period, therefore the patient was ultrafiltrated (1500 ml). Due to a delay in the blood bank, we had to transfuse the whole blood through the pump due to the continuous fall in the hematocrit (20.6%) and the cerebral perfusion pressure (39%). Before transfusing the whole blood, we measured the ACT and administered an additional 5000 IU of heparin. A short while after the transfusion, the perfusionist warned about high pump circuit pressure. An immediate check revealed an ACT of 310 s. Perfusion time was 269 minutes. We had to initiate the backup bypass machine; however the patient suffered from hypoperfusion despite subsequent placement of intra-aortic balloon pump and extra-corporeal membrane oxygenation.

Discussion

We experienced intra-operative clotting of the bypass circuit, which occurred in a very short time after transfusing the whole blood salvaged from the patient. We are in the opinion that the fresh clotting factors within the whole blood were responsible from the abrupt fall in ACT. We may argue that the high ACT (850-1000 s) was unnecessary and the cause of the bleeding. However, as stated in the "2011 update to the Society of Thoracic Surgeons and the Society of Cardiovascular Anesthesiologists blood conservation clinical practice guidelines", maintenance of higher heparin concentrations during CPB may reduce hemostatic system activation and reduce consumption of platelets and coagulation proteins in long CPB times [4]. This suggestion is based on Despotis' and colleagues' study, which showed significant reduction in perioperative blood loss and blood product use when higher heparin concentrations were used [5]. In our institution, we aim for an ACT of above 450 s, frequently check ACT, and apply additional heparin dose according to duration of perfusion and circuit pressure.

The patient's bleeding did not stop despite topical application of tranexamic acid, which is indicated for blood conservation [4,6]. Despite keeping the hemoglobin at 7 g/dl with two packed red blood cells, and hydrating the patient with 500 ml of lactated ringer, our patient suffered from end-organ ischemia of the brain and the kidneys. Therefore we had to transfuse the whole blood to keep the hemoglobin level above 7 g/dl as suggested by the guidelines [4].

Although there are conflicting reports about the protection of platelet functions by acute normovolemic hemodilution [7], we are in the opinion that the whole blood containing fresh coagulation factors caused the abrupt fall in ACT and coagulation of the circuit [8], due to the activation of the intrinsic pathway in the perfusion circuit.

Conclusion

In case of normovolemic hemodilution, the whole blood should not be used before terminating CPB. Nevertheless, we are in the opinion that the abrupt coagulation could be prevented by administering the whole blood via a peripheral vein over a longer period of time, serial measurements of the ACT, and administering heparin accordingly.

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