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Olgu Sunumu

Unexpected Coagulation in the Bypass Pump Circuit

Başar ERDİVANLI*, Şahin BOZOK, Şaban ERGENE**, Tahir ERSÖZ*, Nebiye TÜFEKÇİ****

ABSTRACT

We present a case of premature coagulation observed in the bypass pump circuit. Fifty-nine-year-old female patient (Euroscore: 5) presented with acute heart failure and pulmonary oedema 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 coagulation.

Key words: *activated coagulation time, agglutination, cardiopulmonary bypass*

ÖZ

Baypas Pompa Devresinde Beklenmeyen Pıhtılaşma

Baypas pompa devresinde erken koagulasyon gözlenen 1 olguyu sunduk. Elli dokuz yaşında kadın hasta (Euroscore: 5), mitral kapak darlığına bağlı akut kalp yetmezliği ve pulmoner ödemle başvurdu. Tek damar greftleme ve mitral kapak replasmanı sonrasında (kros klemp süresi: 170 dk.), inatçı bir kanama nedeniyle, heparinizasyon öncesi hastadan alınan 500 ml tam kanı vermek zorunda kaldık. Transfüzyondan kısa süre sonra, devrede pıhtılaşma nedeniyle yedek baypas pompasına geçmek zorunda kaldık. Taze pıhtılaşma faktörleri içeren tam kanın, pompa devresinde intrinsik yolağı aktive ettiği ve pıhtılaşmaya neden olduğu düşüncesindeyiz.

Anahtar kelimeler: *aktive pıhtılaşma zamanı, aglütinasyon, kardiyopulmoner baypas*

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.

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*Recep Tayyip Erdoğan Üniversitesi Eğitim ve Araştırma Hastanesi, Anesteziyoloji ve Reanimasyon Anabilim Dalı
**Recep Tayyip Erdoğan Üniversitesi Eğitim ve Araştırma Hastanesi, Kalp ve Damar Cerrahisi Anabilim Dalı
Yazışma adresi: Yrd. Doç. Dr. Başar Erdivanlı, İslampaşa Mah., Şehitler Cad. 53100 Rize
e-mail: berdivanli@gmail.com

CASE PRESENTATION

Fifty-nine year-old female (body mass index: 27.5 kg/m^2, body surface area 1.61 m^2) presented with insulin-dependent diabetes mellitus, acute heart failure (EF: 65%), pulmonary oedema, left ventricular concentric hypertrophy, angiographically 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 the whole blood was collected with a blood bag shaker and weighed on a pediatric scale. Intravascular volume was replaced with 500 ml isotonic fluid (0.09% NaCl) and stored in the refrigerator door, before heparinization with 350 U/kg (ACT 670 s, Hemochron Celite). After normovolemic hemodilution, the hematocrit was 34%. CPB was established in a standard fashion (Dideco Compactflo 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 Sorin Carbomedics metal valve. Cross-clamp time was 170 minutes, total urine during the perfusion was 1700 ml. Prior to removing the cross-clamp, the patient's body temperature was 36°C, and arterial blood gas values were within normal levels (pH 7.37, pCO_2 35.7 mmHg, PaO_2 187 mmHg, BE -3.9, serum lactate concentration 2.1 mmol/l). Due to a persistent blood leakage, complete hemostasis could not be achieved for about an hour, despite topical application of 1.5 grams of tranexamic acid in 100 ml serum physiologic. In order to prevent an abrupt coagulation in the circuit, the pericardial collection of blood and tranexamic acid were aspirated with a spare aspirator instead of the suction. 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%. The patient was anuric during this period, where she received 500 ml of lactated ringer via intravenous route. In order to compensate for blood losses, and to preserve the pump flow and the mean perfusion pressure at 50-55 mmHg, the patient received a total of 1500 ml via bypass pump, which had to be ultrafiltrated due to anuria and concerns of hemodilution. At this point, the patient could not be weaned from CPB, despite receiving triple inotropes at high doses. Due to a delay in blood bank, we had to transfuse the whole blood through the pump due to continuous fall in hematocrit (20.6%), cerebral oxymetry (39%), and increasing serum lactate concentration (4.2 mmol/l at this point). ACT level measured before the transfusion was 650 s, and the patient received 5000 U of heparin before the transfusion of the autologous blood. A short while after the transfusion, the perfusionist was 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. Postoperatively, the patient received hemodialysis due to renal failure, but she died due to liver failure at the postoperative ninth day.

DISCUSSION

We experienced intra-operative clotting of the bypass circuit, which occurred 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 for the abrupt fall in ACT.

We may argue that the achievement of high ACT (850-1000 s) was unnecessary and it is also the cause of bleeding. However, as stated in the "2011 update of 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 to achieve an ACT of above 450 s. We also 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 kidneys. Increasing the mean blood pressure above 55 mmHg aggrevated the bleeding. 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.

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