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Aortic Valve Replacement for a Patient with Essential Thrombocythemia: A Case Report

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Abstract: A 51-year-old male patient with 3+ aortic insufficiency, hepatitis C, osteomyelitis right foot, and a preoperative platelet count 1.3 million/mm3 required cardiopulmonary bypass (CPB). Essential thrombocythemia is a relatively unknown entity with the utilization of CPB. After consulting with the surgeon, an anesthesiologist and another perfusionist, a team approach was used to discuss treatment for this patient during CPB. The treatment consisted of exchange transfusions, autotransfusion techniques, and current protocol for blood gas management. No incidence of hypercoagulability was observed during this procedure or postoperatively. Based on current knowledge of pathophysiology and review of the literature, plateletpheresis should be the current management of essential thrombocythemia. **Keywords:** aortic valve replacement, essential thrombocythemia, cardiopulmonary bypass. *JECT. 2004;36:166–168*

BACKGROUND

Essential thrombocythemia (ET) is the least common chronic myeloproliferative disorder. Other myeloprolific disorders include: chronic myelogenous leukemia, polycythemia vera, and myelofibrosis (1). Essential thrombocythemia is defined clinically when the following laboratory parameters are encountered: a platelet count greater than 600,000/mm^3, hyperplasia of marrow megakaryocytes, absence of the Ph chromosome, an expanded red cell mass, myelofibrosis, or any disorder causing reactive thrombocytosis (2)

Preventing or reducing the risk of such complications relating to ET as vaso-occlusion or hemorrhage is the treatment of ET. Though literature research, there are no predictive tests to determine who will develop these complications. The risks may vary according to age, platelet count, duration of disease, previous symptoms, and the absence or presence of other medical conditions (1). Current management therapy for the described disorders of ET include blood component removal, specifically plateletpheresis. The role of plateletpheresis in current management of ET is considered, based on current knowledge of pathophysiology and a review of the literature (3).

CASE DESCRIPTION

A 51-year-old male, 82.3 kg, 188 cm, body surface area (BSA) 2.08 m^2, was scheduled for an aortic valve replacement. He was diagnosed with a 3+ aortic insufficiency, hepatitis C, osteomyelitis on the right foot, and rheumatoid arthritis. His preoperative lab values were as follows: Hgb = 10.7g/dL; Hct = 30.4 %; platelet count = 1.3 M/mm^3; Pt = 11.2 sec; Ptt = 26 sec; Na = 136 mmol/L, K = 4.1 mmol/L; Bun = 19 mg/dL; Cr = 0.8mg/dL; and glucose = 135 mg/dL. The primary goal of the team approach consisted of reducing the patient's platelet count before initiating CPB (Table 1). The platelet count upon admission was 552,000/mm^3 and increased to 1.3 M/mm^3 on the day of surgery. Fifteen minutes following initiation of CPB the platelet count measured 679,000/mm^3. The platelet count was measured and monitored 2 days postop at 358,000/mm^3.

CASE MANAGEMENT

The patient was taken to the operating room where appropriate monitoring lines were placed, and routine general anesthesia was administered. The patient was then prepped and draped in the usual sterile fashion. Following intubation, anesthesia withdrew approximately 800 cc of packed red blood cells (RBCs) from the patient's central line. Four hundred (400) mLs were sent to the cell saver (Cell Saver 5 System, Haemonetics, Braintree, MA) to wash off the platelets. The washed RBCs were returned to the patient through the bypass circuit. The remaining 400 cc were saved for post-CPB utilization.

A SMARxT Cobe, (Cobe Cardiovascular, Arvada, CO) bypass circuit consisting of a Cobe oxygenator, a Cobe

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Table 1. Patient platelet counts.

Event	Platelet Count
Admission	552,000/mm^3
Day of surgery	1.3 M/mm^3
During CPB	679,000/mm^3
2 days post op	358,000/mm^3

open venous reservoir, a Cobe arterial line filter, a Sarns (Sarns Centrifugal System, Terumo Cardiovascular Systems, Ann Arbor, MI) centrifugal pump, and a Cobe venous saturation monitor were used. The circuit was primed with 1500 cc Plasmalyte-A (Baxter Healthcare Corp., Deerfield, IL) and recirculated. The baseline activated coagulation time (ACT) was 281 seconds, and the heparin protamine titration was 3.0 mg/kg. The baseline ACT was repeated because of the high results, possibly caused by inaccurate blood draw by anesthesia. The repeated baseline ACT was 118 seconds. ACTs were measured using the Hepcon (HemoTec, Inc., Englewood, CO) (Table 2).

A median sternotomy was performed, and the patient was systemically heparinized with a calculated loading dose by heparin dose response curve of 25,000 IU. An additional 20,000 IU added to the CPB circuit for an anticipated hypercoagulable state. The post-heparin ACT was 423 seconds, the surgeon was notified, and CPB was initiated. The blood gases results were within normal ranges throughout the CPB procedure. The Hgb = 7.2 g/dL, and the Hct = 21.3 g/dL 15 minutes following initiation of CPB. Additional hemodilution during CPB was not done. The patient was administered 675 cc from the cell saver, 400 cc collected from the patient preoperatively, in addition to excess volume from the bypass circuit. Table suctions were directed to the cell saver instead of the cardiotomy as routine. Heparinization and temperatures were monitored during the CPB procedure (Table 2). An additional 5000 IU of heparin was administered to the CPB circuit during the warming phase of the procedure as the ACT decreased to 523 seconds. The perfusionist determined this dosage because an HPT was not done at this time. The patient's aortic valve was replaced with a 27 mm St. Jude. Cross-clamp time was 46 minutes, and CPB time was 75 minutes. The patient was removed from the CPB circuit without incident. Anesthesia returned the 400 cc of blood collected preoperatively to the patient.

DISCUSSION

Fifteen minutes following initiation of CPB, the platelet count measured 679,000/mm^3. Two days postoperatively the platelet count measured 358,000/mm^3. The platelet counts continued to decrease, but was this attributable to the platelet pheresis or platelet sequestration. McCarthy, et al. described the dramatic reduction in the platelet count with ablation of symptoms by consecutive platelet apheresis (4). Griest stated platelet pheresis should be the current management of essential thrombocythemia (3). Upon further review, current literature indicates the decreased platelet count was relative to plateletpheresis. It is imperative to use the team approach when dealing with an unfamiliar patient diagnosis. Team members should consist of a surgeon, anesthesiologist, hematologist, and second perfusionist. Based on current knowledge of pathophysiology and review of the literature, plateletpheresis should be the current management of essential thrombocythemia.

Table 2. Anticoagulation results.

Time	Patient Temperatures Arterial (A), Bladder (B) Esophageal (E)	ACT (sec)	HPT (mg/kg)	Additional Heparin Administered
1235, 1240 baseline	B = 37°C E = 37°C	281 sec 118 sec	3 mg/kg	NA
1250 Post-heparin ACT	B = 37°C E = 37.2°C	423 sec	NA	NA
1310 15 minutes following initiation of CPB, cooling to 32°C	A = 30°C B = 36.2°C E = 31.8°C	719 sec	4 mg/kg	NA
1334 warming patient	A = 31°C B = 31°C E = 32°C	523 sec	NA	5000 IU
1355 15 minutes before termination of CPB	A = 35°C B = 34.5°C E = 37.8°C	686 sec	3.5 mg/kg	NA
1424 10 minutes post-protamine administration	B = 37.2°C E = 37.8°C	107 sec	0 mg/kg	NA

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