Case Report

Perfusing the Bleeding Diathesis Patient
An Example of Protocol Flexibility

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Abstract

A patient who clearly fell within the parameters that define those who are likely to bleed excessively post-operatively presented for coronary artery bypass grafting. As this patient refused donor blood or blood products, the risk of excessive post-operative bleeding was a serious concern. This article elucidates the protocol modifications in equipment, blood management, heparin dosing, flow regulation, pressure control, and in the basic surgical approach that resulted in a successful operation and recovery with minimal blood loss.

Introduction

A 72-year-old white female presented for coronary artery bypass (CAB). A Jehovah’s Witness, the patient refused homologous blood transfusion. The patient weighed 50.2 kg, had a hemoglobin (Hgb) of 11.2 g/dl, and had been taking aspirin (ASA). An in-house study had identified the triad of being smaller (less than 63 kg), anemic (less than 12 g/dl Hgb), and older (greater than 68 years old) as universally bleeding excessively following cardiopulmonary bypass (CPB). Therefore, the patients in this bleeding diathesis group are now given at least two units of fresh frozen plasma (FFP) after protamine sulfate administration. In certain conditions (e.g. extremes of these indices, redo operation, direct visual assessment of poor hemostasis) four units are given. Additionally, five units of platelets may be given, particularly if the patient has been taking ASA; indeed, this subset of patients has presented the greatest challenge to hemostasis, frequently requiring additional FFP, platelets, cryoprecipitated proteins, epsilon aminocaproic acid, and/or desmopressin acetate.

Because we would not be able to give this patient blood or blood products, our protocol was substantially changed in an effort to minimize blood loss.

Case Description

First, efforts were aimed at improving this patient’s pre-operative status. Epoetin alfa (recombinant human erythropoietin, 4000 units QOD SC), with which we have had previous success (1), was initiated in an attempt to augment her low hemoglobin, as was ferrous sulfate (300 mg BID PO). Phytonadione (vitamin K, 30 mg, IM) was given to increase hepatic production of clotting factors. The patient was restricted from ASA therapy to allow the natural replenishment of functional platelets; this concern dictated a two week delay before surgery.

Operative Procedure

The greater saphenous vein was harvested and prepared, and the legs were closed and wrapped, prior to heparinization. Thus bleeding from the legs was all but eliminated.

An uncharacteristic proximal first approach helped reduce CPB time from our average of 1 hour 45 minutes for a triple CAB to 1 hour 9 minutes. Two anastomoses were performed prior to cannulation. A third proximal was completed after the removal of the aortic cross clamp, providing an ischemic recovery period. Dopamine hydrochloride (5 micrograms/kg/min) was started during this period for an early stimulation of myocardial contractility. A fourth diseased vessel, judged to be non-dominant, was not bypassed to reduce CBP time from our 2 hours 7 minutes average for a quadruple bypass.
Consultation with the anesthesia staff resulted in minimizing fluids given. Hypotension was treated with 0.1 mg aliquots of phenylephrine providing the systemic vascular resistance (SVR) was not elevated. The CPB circuit consisted of a vortex pump, a down-sized oxygenator, a 1/4” arterial line to a 18 Fr. aortic cannula, and a 3/8” venous line to a 32/40 Fr. two stage venous cannula. Priming volume was 900 ml of PlasmaLyte-A. Blood cardioplegia at a 4 to 1 blood to crystalloid ratio further addressed dilutional concerns. Also, because of the patient’s small heart size less cardioplegia was required.

A most significant adjustment of protocol was a reduction in heparin levels. After determining a baseline ACT of 164 seconds, 6,000 units of heparin were given. This brought the ACT (352 seconds) within target range (Figure 1). The only additional heparin given was 500 units late in the perfusion in response to an ACT of 227 seconds. Also, salvaged shed blood was washed with at least two liters of saline to avoid residual heparin.

The perfusion was conducted without our usual hypothermic protection (28°C rectal), although the rectal temperature was allowed to drift down to 35°C until the completion of the second distal anastomosis.

Five hundred milliliters of whole blood were sequestered upon initiation of bypass, requiring the addition of 500 ml of PlasmaLyte-A to maintain acceptable flows. Low blood flows had been anticipated because the patient was dehydrated and because of the low priming volume (900 ml). Blood flows averaged approximately 2.3 L/min. Flow rates were determined by monitoring venous blood gases via an in-line device. Their general deterioration (decreasing Svo₂ and decreasing pH, despite compensatory hypocarbia) was the chosen indicator for increasing flow by adding fluid. However, low flow periods were transient and were tolerated without further fluid addition since the venous blood gas values rebounded quickly to acceptable levels (Figure 2).

Upon completion of the final anastomosis, flow was decreased to 1 L/min. The vigorously contracting ventricles
enabled the venous cannula to be pulled and the line drained, despite a relatively empty heart. Blood from the circuit continued to be infused. Crystalloid was added immediately to displace the blood in the system. As a final conservatory step, the system was flushed with saline into the autotransfusion machine.

Protamine (300 mg) was promptly started. The chest was closed quickly to reduce heat loss. Salvaged shed blood and the sequestered whole blood were reinfused.

Discussion

The concerns associated with delaying the surgery on this patient were overshadowed by the need to improve her hematological profile. Given time and interventional therapy, red cell volume, functional platelet count, and clotting factor levels may be improved.

Although dietary iron supplements and epoetin alfa (2) have proven successful in enhancing hematocrits, this rise (Figure 3) is probably due to dehydration. On the morning of surgery, the patient weighed 46.6 kg, a 3.6 kg weight loss since her earlier work-up. A seven percent catabolic weight loss in two weeks is unlikely. Also, a negative fluid balance (-340 ml) measured during one eight hour shift (unfortunately the only period recorded) validates this assessment. The strong influence of respiration on the pulmonary artery pressure waveform also signaled a low vascular volume. This is not to say that the patient did not respond to therapy. The reticulocyte count hallmarks a solid response (Figure 4).

Platelet function is permanently compromised by ASA ingestion; but with a half-life of under fourteen days, functional platelets may be substantially replenished within this time (3).

Patients unable to maintain a normal hematocrit appear to behave hemostatically as though their clotting factor level or function is impaired. Therefore, vitamin K was given to ensure plasma levels were adequate for hepatic production of Factors II, VII, IX, and X.

Lower plasma heparin levels have been correlated with decreased post-op bleeding (4), so heparin dosage was restricted. Our target ACT range of 300-350 has been shown to be safe for CPB (5,6). The drop in ACT to 227 was considered too precipitous to be accurate and was immediately repeated. The subsequent ACT of 302, though within target range, was received with concern in light of the previous sample so heparin (500 units) was given.

Although the removal of 500 ml of blood resulted in severe anemia, hemostasis was of overriding concern. Sequestering blood clearly avoids the mechanical damage to platelets caused by the heart-lung machine and it helps to preserve platelet count (7,8). Reductions in the requirements for blood products have been demonstrated using this technique (7,9,10). Use of a vortex pump and prudent aspiration techniques can also help to preserve platelet count. The efficacy of these techniques is validated by the low chest tube drainage encountered (Figure 5).

References

