**ABSTRACT**

Heparin-induced thrombocytopenia (HIT) in patients undergoing open heart surgery has been reported with increasing frequency. Several strategies have been suggested to approach this difficult problem. However, the syndrome is still associated with significant morbidity and mortality. We describe an 82-year-old male with HIT who underwent coronary artery bypass grafting utilizing a heparin-bonded cardiopulmonary bypass circuit with very low systemic anticoagulation. Only one unit of packed red blood cells was transfused. The patient recovered uneventfully. This strategy is safe and effective, and, therefore, should be considered in patients with HIT.

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INTRODUCTION

In patients with heparin-induced thrombocytopenia (HIT), anticoagulation during surgical procedures requiring cardio-pulmonary bypass (CPB) presents a significant hazard (1). Immunologic response to unfractionated standard heparin may result in life-threatening bleeding or thromboembolic complications (1). In this report, we describe a new strategy to manage this clinical syndrome using heparin-bonded cardiopulmonary bypass circuits (HBC) with very low systemic heparinization.

CASE REPORT

An 82-year-old male was admitted to our facility with unstable angina and ruled in for a subendocardial myocardial infarction (MI). Past medical history was significant for previous MIs with ischemic cardiomyopathy, atrial flutter, hypertension, and chronic renal insufficiency. Treatment with intravenous infusion of heparin and nitroglycerin was started. Cardiac catheterization revealed a left ventricular ejection fraction of 25%, significant left main and three vessel coronary artery disease. The initial platelet count (PLC) was 182,000/mm$^3$ but within 24 hours, the platelet count dropped to 77,000/mm$^3$. HIT was suspected, heparin was stopped, and all heparin flushes were changed to normal saline. A disseminated intravascular coagulopathy (DIC) screen was negative. Thirty-six hours later, the PLC rose to 100,000/mm$^3$. However, recurrent anginal symptoms required heparin to be restarted, and within 24 hours the patient’s PLC again dropped to 60,000/mm$^3$. The diagnosis of HIT was confirmed by documenting a high level of serum IgG-platelet-associated antibodies (2).

The patient was taken to the operating room and a four-vessel coronary artery bypass graft (CABG) was performed. An ionically bound HBC circuita with very low systemic anticoagulation was used. All components of the circuit, which included a soft shell venous reservoir,b cardiotomy reservoir,c centrifugal pump,d membrane oxygenator,e arterial line filter,f cardioplegia system,g and cannulae,h were heparin coated. The Hemostasis Management Systemi was used for heparin titration. The patient was heparinized with 3000 IU of bovine heparin, which raised the ACT from a baseline of 125 to 194 sec. Cardiopulmonary bypass (CPB) was initiated with 2000 IU of heparin in the pump prime. An ACT was performed every 20–30 min and ranged from 234 to 284 sec. The patient was not actively cooled, and the systemic temperature was not allowed to drift below 34°C. Blood stagnation was strictly avoided during CPB, and perfusion flow rates were maintained at 2.0 to 2.4 L/m$^2$/min. The total perfusion time was 84 min. The protamine reversal dose was 40 mg. Based on careful visual inspection, no evidence of clot formation was noted in the surgical field or in the perfusion circuit. The patient was weaned from CPB without difficulty.

Thrombin antithrombin complexes (TAT), indicators of thrombin generation were measured before, during, and at the end of CPB. TAT, PLC, and hematocrit (HCT) values are depicted in Figure 1. Because TAT specimens must be prepared and interpreted, the intraoperative results were not available until after the surgical procedure.

After heparinization and before CPB, the PLC was 75,000/mm$^3$, and the TAT was 40.1 mcg/L. Once on CPB and just before aortic cross clamp removal, the PLC and the TAT dropped to 47,000/mm$^3$ and 32.2 mcg/L, respectively. At the conclusion of CPB, the PLC and the TAT continued to decrease to 42,000/mm$^3$ and 20.2 mcg/L, respectively. At 2 h after CPB, the PLC was 36,000/mm$^3$, but the TAT increased to 71.8 mcg/L. On postoperative day 1, PLC rose to 38,000/mm$^3$, and at discharge, was 104,000/mm$^3$. The patient received only one unit of packed red cells and no platelets during his hospital stay. His postoperative course was uneventful, and he was discharged on postoperative day 5.

DISCUSSION

Although heparin-induced platelet antibodies can be detected in up to 20% of patients requiring open heart surgery (3), the incidence of clinically important HIT has been reported up to 3% (4). Among these patients, up to 44% develop vascular complications secondary to thrombosis, with a 33% mortality (5).

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a Duraflo II, Baxter Healthcare Corp., Irvine, CA
b BMR 1900 Gold, Baxter Healthcare Corp., Irvine, CA
c BCR 3500 Gold, Baxter Healthcare Corp., Irvine, CA
d Sarns centrifugal pump, 3M Health Care, Ann Arbor, MI
e Spiral Gold, Baxter Healthcare Corp, Irvine, CA
f AF 1040 Gold, Baxter Healthcare Corp, Irvine, CA
g HE 30 Gold, Baxter Healthcare Corp, Irvine, CA
h Medtronic DLP, Grand Rapids, MI
i Medtronic Hemotec Inc., Englewood, CO

Figure 1: Hematocrit, platelet count, and serum thrombin-antithrombin (TAT) complexes values before, during, and post cardiopulmonary bypass.
HIT is often suspected in patients receiving intravenous heparin therapy when the platelet count drops by 50% or below 100,000/mm³ and rises after heparin is stopped, or if thrombotic events occur. Diagnosis is confirmed by platelet aggregation testing and/or serotonin platelet release testing (2).

Because large doses of heparin are required for CPB, alternatives to standard heparinization have been suggested for patients with HIT. These include beating heart surgery for CABG (avoiding anticoagulation altogether) or the use of alternatives to standard heparin. These include ancrod (6), hirudin (7), and low molecular weight heparin (LMWH) (8). The major caveat with heparin alternatives is that the anticoagulation effect cannot be completely reversed, leading to excessive postoperative bleeding (6–8). The use of HBC in a patient with HIT undergoing CABG was recently described in a case report (9). However, in this case, the covalently bonded HBC was utilized in conjunction with LMWH. The anticoagulation effect could not be fully reversed, resulting in increased postoperative bleeding and homologous blood product usage. Previous studies have demonstrated that the application of HBC with low systemic anticoagulation is safe and reduces postoperative bleeding, blood product utilization, and postoperative complications (10, 11). Reducing systemic heparinization minimizes heparin–protamine complexes. Improved biocompatible properties of HBC result in preserved platelet function and decreased activation of leukocytes and complement (10–12).

By using a closed reservoir system and minimizing areas of blood stagnation in the perfusion circuit, CPB can be safely conducted using minimal doses of heparin (12). Based on our circuit specifications, the surface of the ionically bound HBC used in this case was coated with 2400 IU of heparin, and only a portion of that amount is released from the surface into circulation (personal written communication from Mark Loar, Baxter Healthcare Corp. to Paul J. O’Gara May 3, 1999). In this case, patient safety was not compromised, because there was no visual evidence of clot formation in the surgical field or the perfusion circuit, and thrombin generation, measured by TAT complexes, did not increase during bypass. Using this strategy, platelet transfusion was avoided, and only one unit of packed red blood cells was used.

In summary, the use of HBC with very low doses of heparin with CPB in a patient with HIT was safe and effective. Because the experience described is limited to a single case report, more data are required to evaluate this approach critically. However, this strategy should be strongly considered in these patients.

REFERENCES


