Simultaneous Aortic Valve Replacement and Splenectomy in the Thrombocytopenic Patient: Case Report

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Abstract

Splenomegaly associated with secondary hypersplenism is characterized by abnormal splenic function in which formed blood elements are sequestered and destroyed. A 47 year-old male diagnosed as having aortic insufficiency and hypersplenism secondary to bacterial endocarditis underwent a successful aortic valve replacement. Such patients present a higher risk, and patient management during cardiopulmonary bypass is complicated by the associated anemia and thrombocytopenia. Platelet concentrate and whole blood were administered after the splenectomy and just prior to cardiopulmonary bypass. The patient was perfused with a bubble oxygenator primed with two units of packed cells and 5% salt poor albumin in a crystalloid solution. Important parameters to monitor closely are activated clotting times, arterial line pressure, and numbers of circulating platelets. The platelet count prior to splenectomy was 49,000/mm^3. The number of platelets increased after the addition of platelet concentrate to 107,000/mm^3. Upon completion of the aortic valve replacement, the platelet count fell to 58,000/mm^3. This represents a 46% decrease in platelets during cardiopulmonary bypass. Leukocytosis is a typical finding after splenectomy, and the white blood cell count increased from 4,500/mm^3 presplenectomy to 16,600/mm^3 prior to cardiopulmonary bypass and fell slightly to 13,500/mm^3 after surgery.

Presented at Nineteenth International AmSECT Conference, San Francisco, California, March 2–5, 1981.

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Introduction

Hypersplenism is a term used to describe the inappropriate and abnormal sequestration and destruction of the formed blood elements by the spleen.[1] Hypersplenism may be classified into two categories, primary and secondary. Both categories exhibit the same clinical symptoms of splenomegaly and associated pancytopenia, the reduction in all the cellular elements of the blood. The normal function of the spleen is to remove damaged blood cells. Primary hypersplenism is caused by an enlarged spleen that has become over efficient at removing damaged cells, and in which there is no underlying pathological condition. In secondary hypersplenism, the spleen, because of some generalized disease state such as infection, becomes enlarged in response to the basic disease process, and there is a marked increase in the removal of normal cells. In patients with aortic insufficiency and diminished cardiac reserve the excessive removal of red blood cells from the circulation as observed in hypersplenism may further compromise the cardiac function and possibly result in cardiac failure. In patients undergoing cardiopulmonary bypass (CPB) for prosthetic replacement of the aortic valve and in whom underlying disease

conditions complicate patient management, it is necessary for the perfusionist to review the hematological parameters and the conditions that necessitate valve replacement.

Patients with hematologic disorders such as severe hemolytic anemias, sickle cell traits, or coagulation disorders will require constant monitoring of blood gases, hematocrit and activated clotting times (ACTs) to maintain satisfactory perfusion. The transient reduction in platelets and trauma to red blood cells associated with CPB in the patient already deficient in both platelets and red blood cells may produce significant postoperative complications. Heart surgery patients must be hemostatically competent to undergo systemic anticoagulation with heparin and its reversal with protamine. They also require both plasma clotting factors and platelets in adequate numbers. Bacterial endocarditis affects both of the above and may alter hemostasis.[2] The following case report describes the medical problems and the associated CPB management of a patient undergoing simultaneous splenectomy and aortic valve replacement for hypersplenism and aortic insufficiency secondary to endocarditis.

Case Report

A forty-seven year old, 70 kilogram white man was diagnosed as having aortic insufficiency and concomitant splenomegaly. Echocardiography indicated large vegetations on the aortic valve cusps. The patient related a history of rheumatic fever as a child, but had been healthy up until May, 1980. The patient was hospitalized at another institution for headaches and fever and subsequently underwent a craniotomy at that time for management of a subdural aneurysm which was later considered to have originated from mycotic emboli. Postoperatively a diastolic murmur was detected, and an echocardiogram and blood cultures were obtained. The echocardiogram indicated an aortic valve vegetation, but blood cultures throughout the patient's lengthy hospitalization proved negative. A preliminary diagnosis of subacute bacterial endocarditis (SBE) was made, and the patient was placed on antibiotic therapy. Prior to his admission to the University of Texas Medical Branch at Galveston, it was found that the patient's liver and spleen were enlarged. On admission thrombocytopenia (40,000/mm^3) and anemia (hematocrit 29%) were noted. In preparation for his impending surgery, platelet therapy and Phytonadione (Vitamin K) were instituted which resulted in a platelet count of 65,000/mm^3. The patient showed early signs of congestive failure, as exhibited by exertional dyspnea. His deteriorating hematologic status, along with incipient cardiovascular decompensation prompted surgical intervention.

TABLE 1
Blood Products Used During the Operative and in the Initial Twenty-Four Hour Postoperative Period

Time	Platelet Concentrate (Units)	Whole Blood (Units)	Packed Cells (Units)	Fresh Frozen Plasma (Units)
Post-splenectomy	10	1	0	0
CPB Prime	0	0	2	0
CPB	0	0	3	0
Post CPB	10	3	0	3
24 hrs. Postop	10	0	16	7
Total	30	4	21	10

Under general anesthesia with halothane and oxygen, the patient's thorax and abdomen were prepped and draped. A skin incision was made from the sternal notch to the umbilicus, and the spleen was exposed for dissection. The splenic artery and vein as well as the short gastric arteries were sequentially ligated and divided. The spleen was then elevated from its bed and removed. After the splenectomy the abdomen was closed, ten units of platelet concentrate (50–60 cc/unit) and one unit of whole blood were administered prior to median sternotomy (See Table 1). After the heart was exposed, sodium heparin (300 U/kg) was given, and ACTs were monitored to insure a safe level of anticoagulation (that is, greater than 400 seconds)[3]. The patient was perfused with a bubble type oxygenator,[a] primed with two units of packed cells (500 cc) in a 5% albumin and crystalloid[b] solution (1300 cc). In addition, 2000 U of heparin and 20 mEq. of sodium bicarbonate were added to the prime. Other components of the perfusion circuit included a cardiotomy reservoir[c] with an integral 27 micron (μ) filter, and a 40μ arterial filter.[d] The patient was systemically cooled to 26°C. A total of 1300 cc of potassium cardioplegia was administered, and the myocardial temperature was

[a] Model 42-221, Cobe Laboratories, Inc. Lakewood, CO 80215.

[b] Plasmalyte 148 pH 7.4, Travenol Laboratories, Deerfield, IL 60015.

[c] Model Q220F, Bentley Laboratories, Santa Ana, CA 92714.

[d] Model EC3840, Pall Biomedical Products Corp., Glen Cove, NY 11542.

maintained below 20°C during the time of aortic cross-clamping. An additional 1000 cc of crystalloid were administered while on bypass. The aortic valve and vegetations were removed, and a 27 mm Carpentier-Edwards aortic prosthesis was sewn in place. The patient was subsequently rewarmed to 38°C., and CPB was terminated without complication. The total urine output on bypass was 150 cc. ACTs were measured every 20 minutes while on bypass, and all the ACTs during bypass were greater than 600 seconds. Line pressures at the arterial filter were continuously monitored and no increases were observed. The hematocrit on pump fell to a low of 14 percent prompting the addition of three units of packed red blood cells. The blood gases remained within normal bypass parameters (average arterial pH 7.39, $PaCO_2$ 35 mmHg, PaO_2 232 mmHg, SaO_2 97.7%, SvO_2 84.2%). Using the ACT dose-response curve, heparin reversal was obtained with one milligram of protamine sulfate per milligram of heparin. Another ten units of platelet concentrate were adminstered immediately following CPB. The patient also required whole blood and fresh frozen plasma to control bleeding. The patient's chest was closed, and he was taken to the Intensive Care Unit (ICU). In the immediate postoperative period his hemodynamics remained stable, but bleeding presented a problem. His average chest tube drainage for the first four hours after surgery was greater than 180 cc per hour with a total of more than 1200 cc six hours after bypass. The bleeding was finally brought under control with the administration of ten more units of platelet concentrate and seven units of fresh frozen plasma. The total chest tube drainage after twenty-four hours was 2925 cc. The patient's remaining postoperative course was uneventful. He was weaned from ventilatory support after 28 hours and remained in the ICU for four days postoperatively. Although a culture of the removed valve proved negative, the patient remained on Tobramycin and Nafcillin antibiotic therapy. He was discharged twenty days after surgery in good condition.

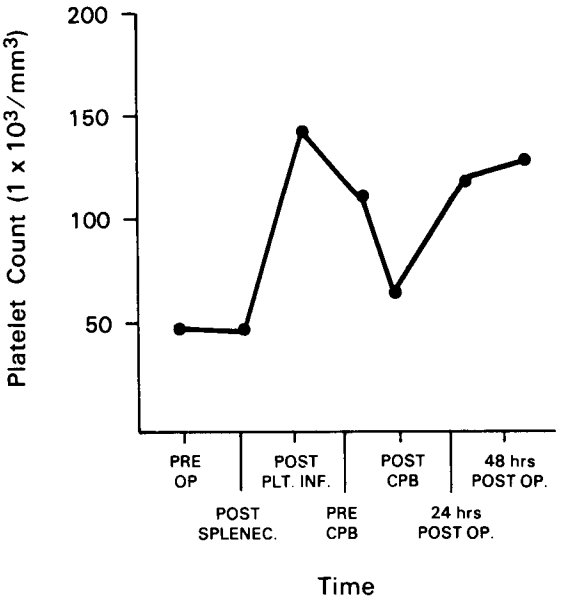

FIGURE 1. Platelet counts during the operation and in the initial forty-eight hour postoperative period.

Discussion

This patient's platelet count prior to cardiac surgery was 49,000/mm³ (See Figure 1). After the splenectomy the platelet count fell to 44,000/mm³. The platelet count increased to 144,000/mm³ with the administration of ten units of platelet concentrate prior to the sternotomy. It was thought that the administration of platelets prior to CPB would provide better control of postoperative bleeding, which is one of the major complications of heart surgery[4] and would prevent severe thrombocytopenia which might possibly result in future hemorrhage into the brain. Just before the initiation of CPB the platelet count decreased to 107,000/mm³. This reduction may be attributed to bleeding following the splenectomy or hemodilution due to volume replacement. At the completion of CPB the platelet count was further decreased to 58,000/mm³. This is a 46% decrease in platelets from the pre-bypass sample. Platelet reductions are well correlated with CPB[5,6] and the degree of cardiotomy suction.[7] Heparinization[8] and hypothermia[9] have also been documented to reduce the number of circulating platelets.

Studies on platelet sequestration in humans indicate that the normal sized spleen stores approximately one-third of the total platelet mass.[10] In hypersplenic patients this quantity may be markedly increased.[11] Thrombocytopenia due to prolonged immune complex generation from an infected valve can lead to the massive destruction of circulating platelets by the spleen.[12] Vegetations on the infected valve also act as a stimulus for platelet adhesion.[13] The subsequent embolization of these vegetations may eventually be transported to the spleen as well as other organs. Antibodies produced by the white pulp of the spleen respond by attacking the platelets contacted by the in-

fected valve. The spleen enlarges, and normal circulating red blood cells and leukocytes are thus pooled. The spleen may also begin to harbor the pathogen itself, enlarging even more and complicating the patient's hematological status.

The addition of platelet concentrate prior to CPB caused concern that these platelets might become adherent to the extra-corporeal filter or possibly abnormally alter the patient's coagulability. Mabry and associates[14] reported a case of heparin resistance and hypercoagulability in a patient with aortic insufficiency secondary to bacterial endocarditis. CPB in that case was terminated after the extra-corporeal circuit clotted. Careful attention to ACTs and arterial line pressures are essential for the detection and prevention of coagulation during CPB. Thrombocytosis is also frequently associated with splenectomy. This patient's platelet count became slightly elevated by the ninth postoperative day but never went above 450,000/mm^3.

The oxygenator was primed with two units of packed red blood cells to compensate for the patient's low prebypass hematocrit. His hematocrit prior to the splenectomy was 23.5% and before CPB fell to 18.7% due to intravenous fluid administration by the anesthesiologists for bleeding following the splenectomy. (See Figure 2). The addition of packed red blood cells during CPB brought the hematocrit up to 25% after the aortic valve replacement. The patient's hematocrit remained in the low to mid 30% range for several days, approaching 40% on the seventh postoperative day. The

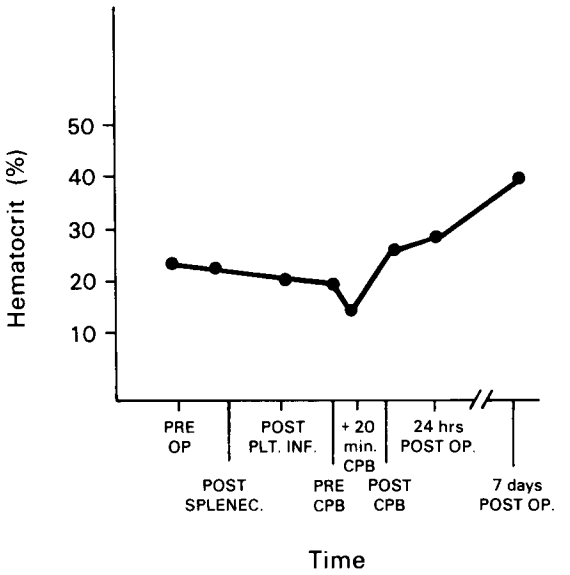

FIGURE 2. Hematocrits during the operation, in the initial twenty-four hours and on the seventh postoperative day.

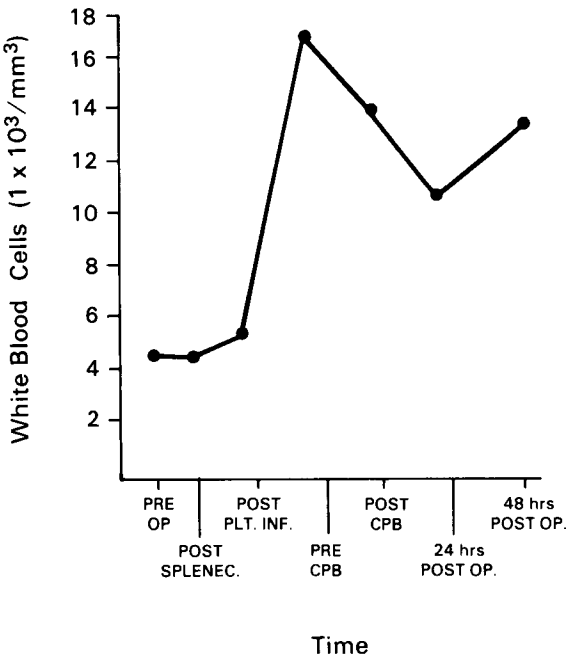

FIGURE 3. White blood cells during the operation and in the initial forty-eight hour postoperative period.

hematocrit was normal for the remainder of his hospitalization.

The removal of normal red blood cells by an enlarged spleen in a patient whose cardiac output is compromised by aortic insufficiency may result in eventual heart failure. In response to stress the heart must continue to pump an adequate amount of blood to perfuse the organ systems, but a lowered hematocrit and decreased oxygen carrying capacity of the blood may lead to peripheral vasoconstriction[15] and an increased afterload. This increase in afterload in the already failing heart may lead to severe heart failure.

A marked leukocytosis was observed after the splenectomy. The white blood cell (WBC) count increased from 4,500/mm^3 before splenectomy to 16,000/mm^3 before CPB (See Figure 3). This 370% increase in circulating WBCs may be attributed to the type of surgical procedure and the administration of whole blood. By differential count, the percentage of neutrophils showed a corresponding increase from 39% pre-splenectomy to 81% pre-CPB (See Figure 4). The WBC count declined after CPB to 13,500/mm^3, with a corresponding decrease in the percentage of neutrophils to 62%. A mild increase in the WBC count after routine CPB is a typical finding.[16,17] Leukocytosis and neutrophilia are also common findings after splenectomy.[18] This patient's WBC count upon discharge was

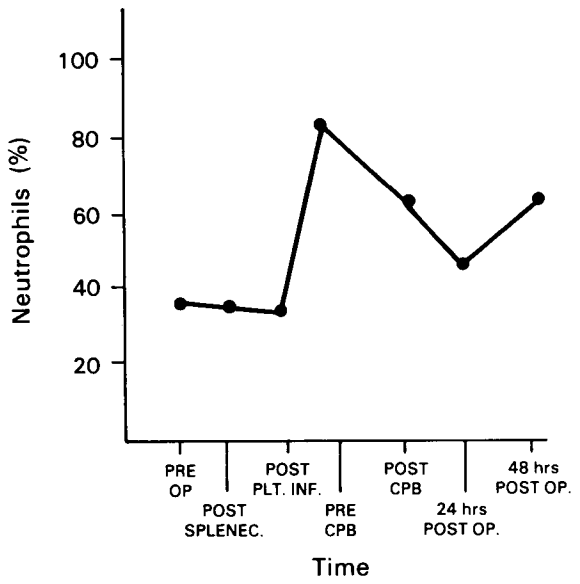

FIGURE 4. Neutrophils-percentage of white blood cells during the operation and in the initial forty-eight hour postoperative period.

increased over what it was prior to surgery, (8,100/mm^3 versus 3,200/mm^3), which is a finding considered normal in splenectomized individuals.

Splenic sequestration of platelets during CPB has been observed in dogs.[19,20] In animal studies the thick muscular capsule surrounding the spleen relaxes and expands in response to general anesthesia, thus, sequestering abnormal amounts of red blood cells.[11,21] Although this has not been directly observed, evidence seems to indicate that this may be true to a lesser extent in humans undergoing CPB. Platelet sequestration studies by Aster[10] indicate that after the infusion of radioactive chromium-tagged platelets in patients with normal spleens, the radioactive chromium was concentrated predominantly in the spleen and liver. In patients with enlarged spleens there is a striking contrast in spleen to liver ratios of labeled platelets ranging from 7:1 to 15:1. Hessell and associates[9] have demonstrated temporary thrombocytopenia in splenectomized dogs subjected to hypothermia with the liver serving as the exclusive site of sequestration when the spleen was removed. Recovery of the tagged platelets upon rewarming indicates that such sequestration is only temporary. Platelet kinetic and function studies in splenectomized dogs demonstrate that transient and reversible platelet sequestration occurs in the liver during CPB.[19] Dogs that were not splenectomized did not demonstrate this reversible sequestration of platelets in the spleen, and the spleen did not release sequestered platelets to the peripheral circulation after CPB.

The mechanism by which hypersplenic leukopenia occurs is poorly understood. The white pulp of the spleen contains a large collection of immunologically competent cells, and it is the site of antibody and immunoglobulin production. Hormonal effects of the spleen on myeloporesis are inconclusive in demonstrating whether this is responsible for the observed leukopenia.

Early valve replacement in patients with bacterial endocarditis and aortic insufficiency should be considered. In a study by Griffin[22] on patients with bacterial endocarditis, 64% of those patients with mild heart failure who received medical therapy died during treatment. Persanti and Smith[23] noted major embolic phenomena in culture negative bacterial endocarditis in 29% of their patients. Splenomegaly was also observed in 29% of the patients with culture negative bacterial endocarditis. Immediate antibiotic therapy and surgical intervention in the patient with bacterial endocarditis and aortic insufficiency are recommended to reduce the associated risks and complications.

Conclusion

In hypersplenism, the spleen serves as a massive reservoir that sequesters and destroys the blood elements. In the patient suffering from hypersplenism secondary to bacterial endocarditis, aortic valve surgery is contraindicated unless the underlying pathological condition is corrected. Patients undergoing simultaneous splenectomy and aortic valve replacement present a higher risk, and CPB may be complicated by the associated anemia and thrombocytopenia. A review of the preoperative hematological status is of value to the perfusionist in the management of such patients. Important parameters for the perfusionist to monitor are ACTs and arterial line pressures, as well as blood gases, hematocrits, and numbers of circulating platelets.

Acknowledgments

The authors would like to thank Barbara Nesmith for technical assistance in preparing the manuscript.

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