FIGURE 3. Two stent profiles were seen on the long-axis view. Fully deployed stents had a biconic (*left*) or a conic (*right*) aspect. Note the waist zone (*) on the left.

indirect evidence of leaflet tissue to stent mismatch in at least 17.5% of cases. The clinical impact of these new data will have to be determined.

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Novel use of plasmapheresis in a patient with heparin-induced thrombocytopenia requiring urgent insertion of a left ventricular assist device under cardiopulmonary bypass

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The management of heparin-induced thrombocytopenia (HIT) remains a major clinical challenge in adults requiring cardiac surgery, especially when present preoperatively. Patients with end-stage heart failure are particularly vulnerable to the development of HIT owing to their frequent and long-term exposure to heparin. We report a case in which plasmapheresis was successfully used to eliminate the circulating antiheparin platelet factor 4 (PF4) antibodies (Ab) to implant a left ventricular assist device (LVAD) under cardiopulmo-

nary bypass (CPB) with full heparinization, as routinely performed.

CLINICAL SUMMARY

The patient was a 47-year-old man with mild chronic obstructive pulmonary disease and no significant medical history except for multiple recent hospitalizations for respiratory decompensation, presumed to be secondary to exacerbation of chronic obstructive pulmonary disease as well as pneumonia. He was admitted to an outside institution with acute cardiogenic shock, presumed to be secondary to acute viral myocarditis, without any evidence of myocardial ischemia (negative electrocardiographic findings, negative troponin levels). He also had acute renal failure, hepatic ischemia with significantly elevated transaminase levels, and rapid development of respiratory failure necessitating emergency intubation. He was started on inotropic support. An echocardiogram revealed severe global biventricular hypokinesis with a left ventricular ejection fraction of 7%, severe mitral and tricuspid

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TABLE 1. Antiheparin platelet factor 4 antibody optical densities

Date	Postop day	Platelet count	PF4 OD	Comments
8/25/09	Preop	205	1.572	
8/26/09	Preop	184	1.946	
8/27/09	Preop	204	—	
8/28/09	0	123	—	← CentriMag LVAD insertion without CPB or heparin
8/29/09	1	81	—	Plasmapheresis day 1
8/30/09	2	86	—	Plasmapheresis day 2
8/31/09	3	87	1.175	Plasmapheresis day 3
9/1/09	4	78	0.925	Plasmapheresis day 4
9/2/09	5	91	0.730	Plasmapheresis day 5
9/3/09	6	98	0.832	Plasmapheresis day 6
9/4/09	7	120	0.931	Plasmapheresis day 7
9/5/09	8	149	0.411 (negative)	
9/6/09	9	148	—	
9/7/09	10	172	0.511 (indeterminate)	
9/8/09	11/1	187	0.463	← HeartMate II device insertion on CPB with heparin
9/9/09	12/1	103	0.797	
9/10/09	13/2	98	1.045	
9/11/09	14/3	129	0.979	
9/12/09	15/4	150	—	
9/13/09	16/5	171	—	
9/14/09	17/6	209	—	
9/15/09	18/7	292	—	
9/16/09	19/8	380	—	
9/17/09	20/9	478	1.467	

Antiheparin platelet factor 4 antibodies were considered negative if the optical density (OD) was less than 0.5. CPB, Cardiopulmonary bypass. CentriMag left ventricular assist device, Levitronix, Waltham, Mass; HeartMate II device, Thoratec Corporation, Pleasanton, Calif.

regurgitation, severe pulmonary hypertension, and a 2 × 1-cm laminar left ventricular apical thrombus. Systemic heparinization was immediately initiated. Cardiac catheterization revealed no coronary artery disease. Two days after initiation of the heparin drip, the patient became profoundly thrombocytopenic with a platelet count of 40,000/ μ L. Rapid-onset HIT was subsequently diagnosed with a positive antiheparin PF4 Ab assay, which was confirmed by a positive serotonin release assay. Heparin was immediately discontinued, and the patient was started on a regimen of lepirudin.

The patient was extubated in approximately 1 week with resolution of acute renal failure and liver injury, but he continued to receive high-dose inotropic support with minimal improvement of the left ventricular ejection fraction for 3 weeks (10%–15% on follow-up echocardiograms). At this time, the patient was transferred to our center for evaluation of heart transplant versus LVAD placement. Soon after his arrival, the hemodynamics deteriorated despite his receiving 2 inotropic drugs and intra-aortic balloon pump support, with a cardiac index of 0.8 L/ m^2 . Repeat antiheparin PF4 Ab and serotonin release assay were persistently positive on bivalirudin.

Inasmuch as the patient required urgent cardiovascular support, the decision was made to place a temporary LVAD (CentriMag; Levitronix, Waltham, Mass) off-pump with inflow from the left atrium and outflow to the aorta. The chest was closed. Bivalirudin was started 2 hours after

CentriMag LVAD insertion. Daily plasmapheresis was initiated the following day for 7 consecutive days until the anti-heparin PF4 Ab test results became negative (Table 1). He was extubated and continued to receive bivalirudin during this time. On postoperative day 8, the patient underwent explantation of the CentriMag LVAD, placement of a HeartMate II device (Thoratec Corporation, Pleasanton, Calif), and removal of the left ventricular apical thrombus under CPB with full heparinization and without any complications. The patient did well postoperatively and was eventually discharged to his home receiving warfarin sodium (Coumadin) without any further complications of HIT.

DISCUSSION

HIT has become an enormous clinical entity in managing patients undergoing cardiac surgery. HIT develops at a rate of 1% to 3% postoperatively.¹ When HIT is diagnosed preoperatively in patients who need urgent or emergency cardiac surgery that necessitates CPB, surgeons are faced with a major dilemma: currently no consensus or data exist to support the best strategy for anticoagulation for CPB during the operation. Although alternative strategies for systemic anticoagulation for CPB exist, they are all associated with significant drawbacks.²

Owing to the urgent or emergency nature of this case, waiting for antiheparin PF4 Ab negativity to place the LVAD on CPB with heparin was not an option. We chose

not to place the LVAD using bivalirudin or any other direct thrombin inhibitors because this is associated with potentially lethal bleeding complications.³ Some centers have reported the use of unfractionated heparin and concomitant potent platelet inhibitors for CPB in the setting of HIT. However, this approach has not been adopted at our institution.

The use of plasmapheresis for HIT is not a novel conception. Few studies have reported using plasma exchange in the management of HIT with thrombosis.⁴ However, the surgical literature contains only 1 case report in which plasmapheresis was successfully used preoperatively in a HIT-positive patient who required coronary artery bypass grafting under CPB with heparin.⁵ To our knowledge, this is the first reported case of plasmapheresis for a HIT-positive patient requiring an LVAD.

This isolated experience suggests that plasmapheresis may be used successfully to seroconvert a patient to

HIT-negative status. This allows for the patient to safely undergo cardiac surgery with CPB using systemic heparinization. The more widespread use of this strategy should be corroborated in a larger patient population.

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Hemophagocytic syndrome: A rare but specific complication of lung transplantation

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Hemophagocytic syndrome (HPS) is a rare but occasionally life-threatening complication after solid organ transplantation.^{1,2} This syndrome is characterized by phagocytizing histiocytes that infiltrate tissues and phagocytize blood elements and their precursors. The cause of this syndrome is unknown.³ The development of HPS has been associated with an uncontrolled excessive inflammatory response, and the timely initiation of potentially life-saving therapy with immunoglobulins and steroids is mandatory. Nonetheless, the reported prognosis of this syndrome remains poor.¹⁻³ There is no published information regarding HPS in lung transplantation.

We report a case of severe HPS related to *Pseudomonas* infection in a lung transplant recipient.

CLINICAL SUMMARY

A living-donor single lobar lung transplant was performed for a patient with pulmonary veno-occlusive disease. The primary immunosuppression consisted of 2 single doses of basiliximab, daily doses of methylprednisolone, followed by maintenance immunosuppression with tacrolimus (target trough level: 10–15 ng/mL), mycophenolate mofetil, and prednisolone. Results of blood tests for hepatitis C virus, hepatitis B virus, cytomegalovirus, and Epstein–Barr virus of the donor and the recipient were negative.

Pancytopenia (hemoglobin 8.3 g/dL, platelets $5 \times 104/\mu\text{L}$, and neutrophils $0.6 \times 103/\mu\text{L}$) was first noted as a clinical problem 8 weeks after transplant (Figure 1). Low-grade fever and mild splenomegaly were present. Elevated levels of interleukin-2 receptor (2355 U/mL [normal range < 650]), ferritin (6338 ng/mL [<138]), triglycerides (193 mg/dL [<150]), aspartate aminotransferase (48 IU/L [<35]), alanine aminotransferase (73 IU/L [<42]), alkaline phosphatase (630 IU/L [<360]), γ -glutamyl transferase (182 IU/L [<40]), and fibrinogen (420 mg/dL [<390]) were also noted. Bone marrow aspiration revealed massive hemophagocytic

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