“Suction Event” in a Patient with a HeartMate II Left Ventricular Assist Device

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Introduction: The use of axial pumps in ventricular assist devices is increasing in frequency. These pumps are sensitive to changes in both preload and afterload pressure and an abrupt change in either can result in excessive left ventricular suction. We present a case of a suction event that occurred in a patient with a HeartMate II left ventricular assist device.

Case Presentation: A 31 year old male with dilated cardiomyopathy and symptoms of New York Heart Association class IV heart failure presented for left ventricular assist device (LVAD) implantation as destination therapy. The patient was admitted to the hospital approximately one month prior with decompensation of his cardiomyopathy and associated liver failure. The patient was stabilized and was subsequently inotrope dependent requiring a continuous infusion of Dobutamine (5-10 mcg/kg/min) to maintain cardiac output. He was brought to the operating room for a planned left ventricular assist device implantation. After an uneventful operative placement of a HeartMate II (Thoratec Corporation, Pleasanton, CA) LVAD, the device was deaired and cardiopulmonary bypass was weaned. LVAD support was then initiated with the following inotropes: milrinone 0.5 mcg/kg/min, epinephrine 0.05 mcg/kg/min, and vasopressin 0.04 units/min. The LVAD support was slowly increased to 8,000 RPMs. TEE showed a left ventricle that was appropriately decompressed with no significant septal bulging, mild right ventricular hypokinesis, and appropriately placed LVAD inflow and outflow cannulas. At the conclusion of the procedure, LVAD flows ranged from 4.5 to 5.0 liters per minute with the mean arterial pressure in the 60’s. The patient was then transported to the SICU without any significant events.

While in the SICU, the patient began to have increasing chest tube output to the order of 200-300 ml per hour. This output continued over time and required multiple transfusions of PRBC’s and fresh frozen plasma. He then developed hypotension with a MAP in the 50’s, an elevated CVP, and LVAD flows that had decreased to below 4.0 liters per minute. A progressing cardiac tamponade was suspected and the patient was taken back to the operating room for reexploration. Upon arrival in the OR, a TEE was performed and a “sucked down” left ventricle was encountered with no evidence of tamponade (image 1). After volume loading and bolus injection of phenylephrine, the size of the left ventricle expanded (image 2). The mean arterial pressure was increased to the 50’s and the LVAD flow increased to the 4.5 to 5.0 liters per minute range. Given the amount of drain output and required transfusions, reexploration was still performed. A moderate amount of clotted blood was removed from the anterior mediastinum but no overt tamponade was encountered. Also, a small area of bleeding was noted from the LVAD outflow cannula insertion site on the ascending aorta, which was appropriately repaired. After chest closure, the patient was transported back to the SICU without further events.

Discussion: The HeartMate II is an axial flow LVAD and is known to be pressure sensitive. At a constant pump speed, its output varies, depending on the pressure differential between the pump inflow and outflow. Therefore, a change in either preload or afterload can change LVAD pump flow. In certain situations, when a significant change in preload or afterload occurs, excessive suction of the left ventricle can take place. This event will result in decreased flow through the LVAD and can potentially induce ventricular arrhythmias.

In this case, the patient was initially misdiagnosed with cardiac tamponade when in fact he was having an LVAD suction event. Once the suction event was diagnosed via TEE, it was corrected with volume loading and by increasing afterload via an increase in systemic vascular resistance. This case demonstrates the sensitivity of axial flow LVADs to changes in preload and afterload and also emphasizes the importance of being able to diagnose and treat a suction event.

References:
1) ASAIO 2008; 54:245-248
2) J Heart Lung Transplant 2007; 26:819-25
Anticoagulation using Tirofiban and Heparin during Cardiopulmonary Bypass in a Patient with Heparin-Induced Thrombocytopenia Type II

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Introduction: At this time, there is no recognized standard method of anticoagulation during cardiopulmonary bypass in patients with heparin-induced thrombocytopenia type II (HIT). We present a case of successful anticoagulation using the platelet glycoprotein IIb/IIIa inhibitor, tirofiban, with unfractionated heparin during cardiopulmonary bypass in a patient with HIT.

Case Presentation: A 73 year old male with severe mitral regurgitation, moderate aortic regurgitation, and coronary artery disease presented for mitral and aortic valve replacements and coronary artery bypass grafting. Two months prior, the patient was diagnosed with a deep venous thrombosis and pulmonary embolus for which he was treated with a heparin infusion. Subsequently, the patient developed HIT and continued to display positive serologic tests for HIT up to the day of surgery. According to the protocol at our institution, tirofiban 10 mcg/kg IV bolus followed by an infusion at 0.15 mcg/kg/min was administered prior to anticoagulation with 3 mg/kg of unfractionated heparin. The tirofiban infusion was discontinued approximately two hours prior to the cessation of cardiopulmonary bypass. The total cardiopulmonary bypass time was approximately four hours, during which time no evidence of thrombosis of the cardiopulmonary bypass circuit was noted. Following separation from cardiopulmonary bypass, heparin was reversed with protamine. Multiple transfusions of fresh frozen plasma, cryoprecipitate, and platelets in addition to recombinant factor VIIa were required to obtain adequate hemostasis. However, the bleeding was not excessive postoperatively, and the patient remained hemodynamically stable without evidence of thrombosis throughout the remainder of his hospital stay.

Discussion: Patients with HIT presenting for cardiac surgery represent an interesting clinical dilemma of how to obtain adequate anticoagulation for cardiopulmonary bypass. The incidence of HIT in patients undergoing cardiac surgery has been reported at 1%, with only approximately one-third of these patients developing thromboembolic complications.1 The formation of heparin and platelet factor 4 antigen-antibody complexes induce platelet aggregation, which ultimately leads to the decreased platelet count and thrombosis classically seen in HIT. These antibodies typically are not seen after 90 days, and thus patients re-exposed to heparin after this period have good outcomes. Many alternative anticoagulation strategies for patients who have active HIT have been successfully used during CPB, including heparinoids (danaparoid), direct thrombin inhibitors (hirudin, argatroban), and platelet inhibitors (prostacyclin, Gp IIb/IIIa inhibitors). The drawback of these strategies is that, unlike heparin, there are neither reversal agents nor available means to monitor level of anticoagulation and thus have been associated with both severe hemorrhage and inadequate anticoagulation.2

In this case, we chose to use heparin as the primary anticoagulant with tirofiban acting to block platelet aggregation. Tirofiban is a short-acting glycoprotein IIb/IIIa inhibitor eliminated via the kidneys with a plasma half-life of 2 hours. When used in combination with heparin in patients with HIT, tirofiban functions to inhibit platelet aggregation by HIT antibodies and thus prevent thromboembolic events during CPB. Despite tirofiban’s short half-life, as in this case, there have been reports of persistent platelet blockade resulting in bleeding post-protamine.3 Recombinant factor VIIa has been shown to overcome tirofiban’s antiplatelet action by increasing thrombin production on the platelet surface.3,4 In the end, this strategy of combining tirofiban with heparin provides a safe and successful mode of anticoagulation in patients with HIT requiring cardiopulmonary bypass.

References:
**Placement of an Apicoaortic Conduit in a Patient with Symptomatic Aortic Stenosis and Porcelain Aorta**

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**Introduction:** Aortic valve replacement is the traditional treatment for patients with severe symptomatic aortic stenosis. However, patients with porcelain aorta are at significant risk of perioperative stroke. This is a case of apicoaortic conduit (AAC) placement to effectively enlarge the left ventricular outflow area and avoid the risk of ascending aorta manipulation.

**Case Presentation:** A 76 year old female with progressive dyspnea was diagnosed with severe aortic valve stenosis. Her significant past medical history included hypertension, diabetes mellitus, end stage renal disease, and prior stroke. Chest CT showed a circumferential heavily calcified ascending aorta known as a “porcelain aorta”. It was felt that the risk of causing a stroke from cross clamping the ascending aorta was exceedingly high so instead of a traditional aortic valve replacement, she was taken to the operating room for AAC placement. Her monitoring and resuscitation lines were placed prior to entering the OR which included a large peripheral IV, radial arterial line, pulmonary artery catheter, and a double lumen catheter. Defibrillation pads were placed. After intravenous induction a 37 FR left double lumen tube was positioned. Her baseline intraoperative TEE showed concentric LVH, normal LV function, severe AS with calculated valve area of 0.9cm², and heavily calcified ascending aorta. The surgery was performed via left thoracotomy and selective right lung ventilation. First the descending aorta and LV apex were isolated. Simultaneously the AAC was created by suturing an apical connector and a tube graft to either end of a freestyle aortic valve/root. A partially occluding J clamp was placed on the descending aorta below the level of the LV apex and the distal end of the conduit was sutured to the aorta. The graft was then deaired. Next a needle and wire were placed through the LV apex. TEE was utilized to assess the wire was in the center of the LV and not caught in the papillary/subvalvular apparatus. Next, a foley balloon was placed through a LV coring device and these were placed over the wire. Epicardial leads were used to rapid ventricular pace at 180 beats/minute to both reduce the dynamic cardiac motion for coring, and also to decrease the cardiac output to near zero so the aortic valve would not open. This decreases the risk of systemic air embolization. The LV apex was cored out and hemorrhage was prevented by keeping the foley balloon in place until the apical connector was attached. TEE was again utilized to assess for laminar flow into the apical cannula and out the descending aorta tube graft. The maximal AV velocity was decreased from 4.0 to 2.3 m/s across her native valve. The surgery was performed without cardiopulmonary bypass and lasted approximately 3 hours. She was extubated on postoperative day #1 and discharged with uneventful hospital stay on postoperative day #5.

**Discussion:** The surgical options for this patient are limited due to her porcelain aorta. Traditional aortic valve replacement involves significant manipulation of the aorta. Our institution is involved in the PARTNER trial which compares placement of an aortic valve via catheter (either transfemoral or transapical) versus standard surgical AVR1. This patient does not qualify for this trial for two reasons: because she is dialysis dependent which is one of the exclusion criteria, additionally she could be randomized to standard AVR which was deemed too high of risk for stroke. If aortic valves via catheter placement become FDA approved in the future, placement via the transapical approach would have been an option in this patient. The transfemoral approach would be high risk as the catheters transverse the ascending aorta. Avoiding her heavily calcified valve and ascending aorta via placement of an apicoaortic conduit was deemed the safest choice.

There are a number of delayed morbidity risks associated with the AAC including thrombosis, valve dysfunction, and endocarditis2. Thrombosis within the foreign material can cause mechanical obstruction. The bioprosthetic valve will likely wear at a similar rate of one placed in the aortic valve position. Apical aortic conduits are difficult to assess for these complications using ultrasound postoperatively. The conduit sits beneath the left lung and the ultrasound has to pass through lung tissue and the conduit is obscured by the air interface. Additionally, the patient breathing contributes significant movement of the involved area. TTE or TEE should be used to screen for a worsening gradient across the native aortic valve. If this is significantly high or increasing over time, a cardiac MRI can detect most problems with the AAC.

This patient was able to have her aortic stenosis bypassed by placing an AAC via thoracotomy “off pump”. Although long term complications are a risk, this provided a safer alternative to traditional AVR with exceedingly high risk of stroke.

**References:**

1) http://clinicaltrials.gov/ct2/show/NCT00530894?show_locs=Y

2) American Heart Journal 2001;141:630-6
**A patient with Glanzmann’s Thrombasthenia for TVR and VSD Repair**

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**Introduction:** Glanzmann’s Thrombasthenia is an extremely rare, autosomal recessive disorder, where platelets lack integrin function in the glycoprotein IIb/IIIa complex essential for adequate hemostasis resulting in increased perioperative bleeding. Platelets appear structurally normal, but have varying deficiencies of Gp IIb/IIIa. During cardiopulmonary bypass assessment of platelet function is an ongoing dilemma, with only two case reports to date of cardiac surgery in patients with Glanzmann’s Thrombasthenia. With the advent of Gp IIb/IIIa inhibitors the VerifyNow® testing system has been developed. By analyzing the number of Platelet Aggregation Units (PAU), this system determines the percentage of active platelets in circulation. Though unproven, this testing system may be useful in patients with Glanzmann’s Thrombasthenia (GT) to both assess function of preexisting and transfused platelets.

**Case Presentation:** A 31-year-old female with GT presented for tricuspid valve replacement and ventricular-septal defect repair. The patient had a long-standing history of a small VSD and at age 16 developed bacterial endocarditis with resultant severe TR causing right atrial and ventricular enlargement. Now symptomatic, with increasing fatigue and shortness of breath, she presented to cardiothoracic surgery for definitive VSD closure and TVR. Preoperative evaluation revealed a 2:1 left to right shunt on right heart catheterization, severe TR with reversal of flow in the hepatic veins, enlarged right-sided chambers, no pulmonary hypertension, and preserved left ventricular function (EF of 60%). Extensive hematology workup and consultation was also performed prior to the operation confirming the diagnosis of GT, which showed the patient to have normal platelet numbers but sub-optimal function.

Preoperatively the patient received a two unit platelet transfusion resulting in a VerifyNow® Platelet Aggregation Unit (PAU) value of 38 (suggesting 38 percent of the circulating platelets were functional). The patient was taken to the operating suite, where a median sternotomy, bicalve cannulation and cardiopulmonary bypass commenced. Full systemic anticoagulation with intravenous heparin (400 U/kg) and aminocaproic acid were used (10 g bolus followed by a 2 g/hr infusion). A 10 mm VSD was found and repaired with a Teflon patch. The tricuspid valve with moderate septal/posterior leaflet thickening and severely foreshortened chordal structures was deemed irreparable and replaced with a 32 mm bioprosthetic mitral valve. Following completion of the procedure and cessation of CPB, post-procedural TEE showed no further VSD and an appropriately positioned and functioning bioprosthetic valve in the tricuspid position with no paravalvular leak. A post-CPB PAU value was found to be 33 and two additional units of platelets and a unit of fresh frozen plasma was administered. Adequate hemostasis was achieved and the patient was transferred to the intensive care unit. In the ICU, a follow-up PAU value was 74, suggesting improved platelet function following platelet transfusion. The patient was discharged on POD 8 without any complications and on follow had near total resolution of symptoms.

**Discussion:** Extracorporeal circulation, including cardiopulmonary bypass is known to cause platelet destruction and thrombocytopenia which can complicate cardiac surgery. Our patient presented to the OR with limited platelet function resulting in a high likelihood of post-operative coagulopathy. Using the VerifyNow® IIb/IIIa system to determine Platelet Aggregation Units (PAU) as a measure of platelet function was utilized in this case. This may present a new adjunct to tailor therapy for the bleeding patient. Though fairly uncomplicated, this case shows that even very complex procedures can be performed with appropriate pre-operative evaluation and treatment to best maximize a patients condition before entering the operating room. In addition, the use of the VerifyNow® system may prove to be beneficial in confirming adequate platelet function in cases where hemostasis is of utmost concern.

**References:**

5. VerifyNow® IIb/IIIa Assay Package Insert.
A Clot in the Venous Reservoir While Using Bivalrudin for Cardiopulmonary Bypass in a Heart Transplant Patient

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Introduction: Recent studies suggest that bivalrudin, a direct thrombin inhibitor, is a safe and efficacious alternative to unfractionated heparin for cardiopulmonary bypass in patients with heparin induced platelet antibodies. \(^1\)\(^2\) We present a case of a clot found in the venous reservoir of the cardiopulmonary bypass circuit while using bivalrudin for a patient with heparin induced platelet antibody undergoing orthotopic heart transplantation.

Case Presentation: A 51 yr old male with non-ischemic cardiomyopathy and recurrent ventricular tachycardia presents for orthotopic heart transplantation. Patient had been hospitalized for 4 months prior to surgery secondary to deteriorating heart function and developed heparin induced platelet antibodies with repeated assays, including one performed one week before surgery, returning “strongly positive”. No platelet aggregation test had been performed. The patient was being treated with Coumadin. On the morning of surgery, the INR was 2.0, PTT 40, and platelet count was 228K. The baseline activated clotting time (ACT) was 135 seconds. A 10 gm load of Aminocaproic acid followed by a 1gm per hour infusion was the antifibrinolytic regimen. Bivalrudin 2mg/kg load followed by a 2.5mg/kg/hr initial rate was used for anticoagulation. The cardiopulmonary bypass (CPB) pump was primed with 50mg of bivalrudin. The ACT was maintained between 421-514 seconds throughout the period of CPB. Prior to termination of CPB, modified ultrafiltration was performed with a Mintech hemofilter. During this period, a floating clot within the venous reservoir was noted, which eventually accumulated to form a 1-2 inch layer of contiguous clot at the top of the reservoir. The ACT was 439 seconds. There was no evidence of clot in the arterial circuit. Bivalrudin infusion was discontinued at the end of CPB. The patient weaned off CPB uneventfully. Two units of fresh frozen plasma were given for hemostasis. The post-operative course was uneventful. The patient was extubated the following morning after surgery, spent 3 days in the intensive care unit, and was discharged 10 days after surgery. There was no evidence of neurologic insult. His platelet count remained above 200K throughout his post-operative course. Coumadin, without concomitant direct thrombin inhibitor treatment, was restarted prior to discharge.

Discussion: The anticoagulation regimen with bivalrudin for cardiopulmonary bypass is yet to be fully defined. We believe our case illustrates two potential problems with using bivalrudin during CPB. First, the use of ACT to monitor adequacy of anticoagulation in the setting of bivalrudin may be misleading. Although small trials suggest a target ACT 2.5 times baseline provides adequate anticoagulation for CPB while using bivalrudin\(^1\)\(^2\), case reports have suggested that the ecarin clotting time (ECT) correlates better with clinical clotting than ACT when using a direct thrombin inhibitor.\(^3\) Second, care needs to be taken when performing modified ultrafiltration with bivalrudin. Modified ultrafiltration can be a means to remove bivalrudin, which is desirable at the end of CPB. In an in vitro experiment, the elimination of bivalrudin appears to be dependent on the hemofilter use, with the Mintech hemofilter having the highest elimination rate of bivalrudin.\(^4\)\(^5\)

References: