**Case Report**

- The patient was a 15-year-old male who was diagnosed with severe dilated cardiomyopathy approximately 4 months prior to this hospitalization.
- He was also found to have right ventricular thrombus on echocardiography, which was initially treated with heparin. HIT was diagnosed based on clinical and laboratory findings. Anticoagulation was changed to argatroban and then subsequently transitioned to warfarin.
- He required admission to the cardiac intensive care unit (CICU) with acute deterioration of cardiac function requiring inotropic support. He was then listed for heart transplantation and a protocol for anticoagulation with bivalirudin for CPB was planned in consultation with the hematology team. The patient was a 15-year-old male who was diagnosed with severe dilated cardiomyopathy approximately 4 months prior to this hospitalization.
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- He required admission to the cardiac intensive care unit (CICU) with acute deterioration of cardiac function requiring inotropic support. He was then listed for heart transplantation and a protocol for anticoagulation with bivalirudin for CPB was planned in consultation with the hematology team. His cardiac function continued to deteriorate and he was scheduled for a LVAD while waiting for a heart to be available.
- LVAD-Intraoperative management: Standard ASA monitoring was applied, arterial and central venous access was established. Anesthetic induction and maintenance were uneventful. Anticoagulation for CPB was initiated with bivalirudin as per guidelines in table 1. The postoperative ICU stay was stable and uneventful. He was extubated on post operative day 2 and subsequently discharged out of ICU on POD 4.

**Table 1: Bivalirudin dosing**

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<tr>
<th>Bivalirudin use in Heparin Induced Thrombocytopenia (HIT) for Pediatric Left Ventricular Assist Device (LVAD) and Heart Transplantation</th>
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<tr>
<td><strong>Surgery</strong></td>
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<td>LVAD</td>
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<td>Heart Transplant</td>
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**Discussion**

- HIT is diagnosed by integrating clinical features and laboratory testing; neither of these alone is sufficient. The diagnosis is often confirmed in the lab by either a strongly positive Enzyme-linked immunosorbent assay (ELISA) or a positive functional assay for HIT antibodies.
- Patients with a past history of HIT with absent HIT antibodies can theoretically be re-exposed to heparin as
  - HIT antibodies are short lived and usually (disappear at a median time of 50 to 90 days)
  - HIT antibodies do not often result in anamnestic responses when subsequently re-challenged.
- However, patients with acute or subacute HIT (HIT antibody present) requiring urgent surgery with CPB the management is often complex and alternative anticoagulants are used.
- Bivalirudin is one of the preferred options. It is a parenteral direct thrombin inhibitor and hirudin analog with a half-life of 25 to 36 minutes.
- No anticoagulation is available to reverse the anticoagulant effect of bivalirudin and its clearance is reduced by nearly 60% in patients with renal failure, and dose adjustments are recommended in these patients.
- In our cases we used a combination of activated clotting time (ACT), INR and partial thromboplastin time (aPTT) to monitor anticoagulation. Although there is no established monitoring strategy, prothrombin time (PT), INR, aPTT, thrombin time, and ACT all increase after the bolus administration of bivalirudin.
- Temperature management coming off CPB and post-CPB are important to enable the proteolytic degradation of bivalirudin. An anticoagulation strategy with regards to blood product use should also be planned.
- The CPB circuit should be primed with bivalirudin and the pump should continue to circulate to prevent clotting of the CPB circuit.

**References**