Bivalirudin in Off-Pump Coronary Artery Bypass Graft in a Patient With Heparin-induced Thrombocytopenia: A Case Report of its Use

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Heparin-induced thrombocytopenia (HIT) is an antibody-mediated reaction in which heparin administration causes a person to enter a pathological and highly prothrombotic state. When patients with known HIT undergo coronary artery bypass and grafting procedures, they must be appropriately anticoagulated. The dangers of heparin administration in this population necessitate the use of an alternative anticoagulant.

Keywords: Bivalirudin, CABG, coronary artery bypass and grafting, heparin-induced thrombocytopenia, off-pump artery bypass and grafting.

The case describes the successful use of bivalirudin for procedural anticoagulation during an off-pump coronary artery bypass and grafting.

Heparin-induced thrombocytopenia (HIT) is a pathologic state occurring in approximately 0.5%–5% of those exposed to heparin. The result is a highly prothrombotic state which places those affected at a high risk for thrombosis that may have drastic effects, including loss of life and limb. When patients affected by HIT require coronary artery bypass and grafting (CABG) surgeries, they present a unique challenge due to their need for anticoagulation coupled with the danger that heparin administration poses for them.

The greater incidence of heparin use for acute coronary syndromes has resulted in HIT becoming more commonplace. It is therefore necessary that alternative strategies for anticoagulation be identified and studied for patients with HIT who are undergoing CABG procedures in an effort to establish safe treatment modalities for future use. The goal is the avoidance of a prothrombotic state both during and after surgery, as well as adequate anticoagulation for the procedure itself. This case report describes the off-label use of bivalirudin, a direct thrombin inhibitor, to provide anticoagulation for a patient with known HIT undergoing an off-pump coronary artery bypass and grafting (OPCABG) procedure.

Case Summary
A 63-year-old male weighing 67 kilograms presented to the preoperative area for a one-vessel OPCABG. The patient’s allergies included latex, which caused a rash, and heparin, which caused heparin-induced thrombocytopenia. Past medical history included 2 myocardial infarctions, coronary artery disease with multiple previous coronary stents, angina at rest and with exertion, hypertension, ischemic cardiomyopathy, abdominal aortic aneurysm, chronic renal insufficiency, and chronic obstructive pulmonary disease. Of note, the patient’s laboratory findings included a blood urea nitrogen (BUN) level of 13 mg/dL, a creatinine level of 1.05 mg/dL, with a calculated glomerular filtration rate (GFR) of 91, and troponins >0.04 µg/L with all other results in normal ranges (white blood cells 6x10^3/mm^3, hemoglobin 13.9 g/dL, hematocrit 40%, platelets 144/L, sodium 138 mEq/L, potassium 4.2 mEq/L, glucose 103 mg/dL, prothrombin time 18.9 sec, international normalized ratio 1.6, partial thromboplastin time 51.3 sec). The patient was a former smoker of 2 packs per day for 40 years, having quit 1 month prior to surgery. A previous left ventricle apical thrombus required the patient to be anticoagulated in the past. Cardiac catheterization 5 days prior to surgery demonstrated an ejection fraction of 25% with severe multivessel coronary artery disease, including 100% occlusion of the left anterior descending coronary artery. Ultrasound demonstrated a 3.7 x 3.3 cm abdominal aortic aneurysm. A 12-lead electrocardiogram revealed normal sinus rhythm with premature ventricular contractions. The patient was classified as an American Society of Anesthesiologists (ASA) physical status class IV with a Metabolic Equivalents of Task (METS) rating of <4. Airway evaluation revealed an edentulous oropharynx with a Mallampati III, a thyromental distance of 3 fingerbreadths, and mouth opening of 3 fingerbreadths. Preoperative medications listed were fondaparinux sodium, hydralazine, clonidine, docusate sodium, carvedilol, aspirin, famotidine, clopidogrel bisulfate, tiotropium bromide, lisinopril, and simvastatin.

After a discussion between the anesthesia and surgical teams, it was decided that a bivalirudin infusion would be used for anticoagulation intraoperatively with activated
clotting time (ACT) monitoring. The bivalirudin infusion was initiated following induction of anesthesia. While the facility has a weight-based pharmacy protocol for bivalirudin use in anticoagulation medical management, it is not approved for cardiac surgery. The facility’s protocol includes a bolus dose of 0.75 mg/kg followed by an infusion at 1.75 mg/kg/hr. The protocol includes a reduced infusion dose for renally impaired patients. Because the patient’s current renal laboratory values demonstrated normal function, the infusion was not adjusted for renal impairment. Renal impairment is rated via calculated glomerular filtration rate and is classified as either moderate (20%), severe (60%), or dialysis (90%).

ACTs were performed approximately every 20–30 minutes during the case and recorded by the perfusionist, who was on standby in the room. The initial ACT revealed that the patient was sufficiently anticoagulated for OPCABG following the bolus and start of the infusion. The single vessel bypass was rapidly completed. The bivalirudin infusion did not require adjustment during the case.

The patient was induced with midazolam, etomidate, and fentanyl. Succinylcholine was used for intubation, which was uneventful. An arterial line, central venous catheter, and pulmonary artery catheter were placed post-intubation. Transeosophageal echocardiogram was used throughout the case. Standard ASA monitors were used, including a 5-lead ECG monitor, as well as a bispectral index monitor and a near-infrared regional cerebral perfusion oximetry monitor. Cisatracurium was used for maintenance of neuromuscular blockade and sevoflurane was used for anesthetic maintenance as well as concomitant doses of midazolam and fentanyl. The bivalirudin infusion was discontinued upon completion of the CABG anastomosis and the sternum was closed. The patient was started on a dexmedetomidine infusion and transported intubated to the ICU for fast-track extubation and recovery. Dexmedetomidine infusions in open-heart cases in this facility are typically started at a continuous rate of 0.5–0.8 µg/kg/hr intravenously without a loading bolus. No intraoperative complications were noted.

Discussion

• **Heparin-induced Thrombocytopenia.** The most common anticoagulant therapy in the clinical setting for OPCABG is heparin. Heparin is also essential in cardiopulmonary bypass and is found endogenously in basophils, mast cells, and the liver. Commercially prepared heparin, which parallels the activity of endogenous heparin, is derived from bovine lung and bovine or porcine gastrointestinal mucosa. Unfractionated heparin binds with antithrombin, a naturally occurring anticoagulant. Binding of heparin with antithrombin increases the ability of antithrombin to inactivate coagulation enzymes (including thrombin and activated factors X, XII, XI, and IX) by 1,000 times. Heparin therefore functions as an anticoagulant mainly by accelerating the normally occurring antithrombin-induced neutralization of clotting factors. Heparin therapy has become commonplace. It is used to treat a wide variety of thromboembolic disorders. Heparin’s beneficial properties include: proven efficacy, a rapid onset of action, laboratory monitoring easily performed, rapid neutralization with protamine, and low cost. Heparin has seen increased usage over the last 7 decades in medical practice due to an increasing number of vascular interventions and an aging population. With the more frequent use of heparin in recent years, the prevalence of HIT has prominently increased. Shaikh states that up to 30% of in-hospital patients need heparin in some form during their hospital stay. Approximately 600,000 new cases of HIT are reported every year.

**HIT** is a clinicopathologic, procoagulant condition characterized by thrombocytopenia seen in patients on heparin therapy. Platelet count decreases by 50% or to <100,000 /L after 5–14 days of heparin therapy. It is associated with increased length of hospitalization and increased morbidity and mortality. HIT is categorized into 2 types based on whether it is immunologically mediated or not. HIT type I exhibits nonimmunologically mediated thrombocytopenia. It is transient, characterized by early onset during heparin therapy, is associated with mild thrombocytopenia, is reversible, nonthrombotic, and is asymptomatic. HIT type II, which is commonly referred to simply as HIT, exhibits immunologically mediated thrombocytopenia. It is a severe reaction to heparin exposure which includes thrombotic events.

The prothrombotic state that occurs with HIT is caused by heparin-dependent antibodies that trigger platelet aggregation by binding to molecular complexes formed by platelet factor 4 (PF4), heparin, and immunoglobulin G (IgG)(Figure 1). Platelet activation, increased thrombin (IIa) formation, and activation of intrinsic tissue factor also occur, which renders the anticoagulant effect of heparin neutral. Thrombosis follows the release of procoagulant substances, endothelial and monocyte activation, and expression of tissue factor.

The most common preliminary evaluation to indicate HIT is a scoring system that uses the 4 T’s evaluation system: assessment of Thrombocytopenia, Timing, Thrombosis, and the absence of Thrombosis explanations for thrombocytopenia (Table). The score is based on a maximum of 8 points, with 6–8 points indicating a high probability of HIT, 4–5 points indicating a moderate probability of HIT, and 3 or less points indicating a low probability of HIT that allows exclusion of HIT as a diagnosis. Serologic or functional assay laboratory testing is used to confirm HIT diagnosis. HIT must be differentiated from disseminated intravascular coagulation (DIC), heparin toxicity, and hyperresponsive thrombocytopenia.

Once diagnosed, the important steps in managing
HIT patients are as follows: 1) suspend use of all heparin (including heparin flushes and heparin impregnated tubing); 2) suspend use of vitamin K antagonists; 3) begin alternative anticoagulant therapy; 4) investigate for lower-limb deep vein thrombosis (DVT); 5) upon recovery of platelet count, start or restart vitamin K antagonists; and 6) avoid prophylactic platelet transfusions as they can increase the thrombogenic effect. The alternative non-heparin anticoagulants for the treatment or prevention of HIT recognized by the American College of Chest Physicians (ACCP) are lepirudin, argatroban, bivalirudin, danaparoid, and fondaparinux.

- **Bivalirudin in Cardiothoracic Surgery.** Anticoagulation for patients with HIT requires an alternative to heparin. Alternative anticoagulants exist for patients to be treated medically; however, a heparin alternative has not been approved for use during cardiac surgery. A safe alternative is needed in both OPCABGs and cases requiring cardiopulmonary bypass (CPB). Ideally, an anticoagulant should minimize activation of clotting during bypass, have a simple and rapid method of intraoperative monitoring, and be completely reversible. Alternatives to heparin for intraoperative anticoagulation include thrombin inhibitors, danaparoid sodium, antiplatelet agents, and defibrogenating enzyme (See Table). Unfortunately, none of these agents meet all of the criteria for an ideal heparin alternative.

Bivalirudin has been approved for use as an anticoagulant in patients with HIT during percutaneous coronary intervention (PCI) and percutaneous transluminal coronary angioplasty (PTCA), and reports show that it has been successfully used off-label for both OPCABG and for CPB patients who have evidence of HIT. It was developed as a synthetic form of hirudin and is a dodecapeptide bivalent direct thrombin inhibitor. It incorporates binding affinity to both the catalytic active site and the anion-binding exosite of thrombin (Figure 2). The drug exhibits linear pharmacokinetics upon intravenous administration with a linear dose-response anticoagulant effect. It does not bind to plasma proteins, has a 13-liter volume of distribution, and a half-life of approximately 20–30 minutes in patients with normal renal function. It is cleared mainly via proteolytic cleavage (80%) with the remaining cleared renally (20%); therefore, doses may need to be adjusted in patients with abnormal creatinine clearance. It can be monitored via ACT, prothrombin time, thrombin time, and partial thromboplastin time. Antibody formation to bivalirudin is not documented. Also, it does not dem-
onstrate a hypercoagulable rebound state following dis-
continuation. In patients with normal renal function, if bivalirudin is infused for only 4 hours, the patient’s coagu-
lation function returns to baseline approximately 1 hour
after discontinuing the infusion. There is no antidote for
bivalirudin; therefore, it cannot be immediately reversed,
but reversibility has been demonstrated with combined he-
modialysis and modified ultrafiltration, administration of
recombinant Factor VIIa, and transfusion of fresh frozen
plasma (FFP)/cryoprecipitate.

Multiple case reports and multicenter trials have
been published in relation to the use of bivalirudin in cardiac
surgery. Bott et al published a case report in 2003 in
which bivalirudin was used for OPCABG procedures in
2 different patients undergoing cardiac surgery. The first
patient received a loading dose of 0.25 mg/kg, followed
by a continuous infusion beginning at 0.55 mg/kg/h
titrated to a goal of an ACT >200 seconds. Additional
boluses and infusion adjustments were made to maintain
an ACT >300 seconds for off-pump revascularization.
The second patient received a loading dose of 0.75 mg/
kg and then started on an infusion at 1.75 mg/kg/h. After
an initial ACT of 332, the infusion was reduced (by one-
third) to 1.15 mg/kg/hr and again titrated to a goal of
an ACT >200 seconds. In both cases, the infusion was
discontinued when protamine reversal would normally
be asked for at completion of the last anastomosis. The
authors concluded that bivalirudin was a predictable and
safe heparin alternative that deserved further randomized
trials to establish safety and efficacy.

The first comparative study between heparin with prot-
amine reversal and bivalirudin in OPCABG patients com-
pleted by Merry et al consisted of 100 patients who were
randomly assigned to receive either heparin with prot-
amine reversal or bivalirudin during surgery. The patients
on bivalirudin received a 0.75 mg/kg bolus, followed by an
infusion of 1.75 mg/kg/h, with additional adjustments as
indicated by ACT results to maintain an ACT of 300–350
seconds. Evaluation criteria were perioperative blood loss
and graft flow after 3 months. The patients who received
bivalirudin did not show a clinically significant difference
in perioperative blood loss, and had a greater incidence
of measured coronary graft flow than those who received
heparin. This was the first study to demonstrate the feasi-
bility of bivalirudin during OPCABG.

In 2006, Smedira et al performed the EVolution of
Patients during coronary artery bypass graft Operations:
Linking UTilization of Bivalirudin to Improved Outcomes
and New anticoagulant strategies (EVOLUTION) trial. It
was the first multicenter controlled evaluation of bivali-
rudin as a potential alternative to heparin in OPCABG.
This trial randomized 157 patients in 21 different centers
to receive anticoagulation with either bivalirudin or
heparin with a protamine reversal. Bivalirudin was dosed
first as a bolus at 0.75 mg/kg and then continued as an
infusion at 1.75 mg/kg/hr for the duration of the operation.
The practitioner had the option to increase or decrease
the infusion in 0.25 mg/kg/hr increments or administer
additional boluses of 0.1–0.5 mg/kg to maintain an ACT
>300 seconds. Comparisons were made between the 2

Figure 2. Heparin-induced Thrombocytopenia
methods of anticoagulation at the day 7 discharge mark, at 30 days, and at 12 weeks. Rates of complications were similar in both the heparin and bivalirudin groups. The authors of this small pilot study provided evidence that bivalirudin was a safe and effective alternative to heparin for OPCABG procedures.22

A multicenter study known as the Coronary Artery Bypass HIT/TS On and Off Safety and Efficacy (CHOOSE-OFF) trial was performed to evaluate anticoagulation with bivalirudin in patients with either known serological evidence of or risk factors for HIT. For the purposes of this study, an intraoperative bivalirudin anticoagulation protocol was developed. It was referred to as the EValuation of Patients during Coronary Artery Bypass Graft Operation: Linking Utilization of Bivalirudin to Improved Outcomes and New Anticoagulant Strategies (EVOLUTION-OFF). The protocol called for a loading dose of 0.75 mg/kg of bivalirudin followed by a continuous infusion of 1.75 mg/kg/hr with a target ACT of >300 seconds. The infusion was discontinued at the time the last anastomosis was created. This study found that the ACT was an effective tool for monitoring anticoagulation. In addition to being the largest trial (at the time it was conducted) of OPCABGs in patients with HIT or risk factors for HIT, it was also able to provide clinical evidence that bivalirudin was an effective anticoagulation choice in this population, while maintaining a postoperative safety profile similar to heparin.14

Palmer et al23 published a report on the use of bivalirudin in 243 consecutive patients who underwent OPCABG performed by a single surgeon. The anticoagulation protocol used during the study included a loading dose of 0.75 mg/kg of bivalirudin administered at the completion of the harvesting of the internal mammary artery, followed by a continuous infusion at 1.75 mg/kg/hr. The infusion was discontinued after the final proximal anastomosis. ACTs were routinely measured. If an initial ACT was >300 seconds, then no further measurements were taken. Saphenous vein grafts were filled with 0.2 mg/m² of bivalirudin solution on completion of distal anastomosis prior to completing the proximal anastomosis. In this study, if a patient required conversion to CPB, bivalirudin was discontinued and heparin was initiated. At the conclusion of the study, the authors stated that bivalirudin was an efficient and practical option that improved clinical outcomes.23

One of the most recent case reports citing the use of bivalirudin for OPCABG was published by Agrawal, Sayeed, Roy, and Somaraja24 in 2012. A bivalirudin loading dose of 0.75 mg/kg was administered over 10 minutes followed by an infusion at 1.75 mg/kg/hr. Again, the infusion was discontinued upon completion of grafting. Anticoagulation was monitored using ACT monitoring. This study found that 6 hours postinfusion, ACTs returned to normal.24

For patients with HIT that require CPB surgery, or conversion from OPCABG to CPB surgery, there are limited anticoagulation options available. Koster’s25 2007 article brings further light to the problem. There are 3 alternative anticoagulation protocols for patients undergoing cardiac surgery diagnosed with HIT. The first option includes avoiding heparin exposure through use of a direct thrombin inhibitor such as lepirudin for anticoagulation. The second option includes using a combination of heparin with a potent, yet short-acting antiplatelet medication such as a glycoprotein IIb/IIIa antagonist or a prostaglandin to prevent procoagulant activity. The third option includes waiting until the transient HIT antibodies are undetectable, performing the surgery with heparin, and then establishing alternative anticoagulation in the postoperative period.25

A case report by Vasquez et al26 chronicled a patient with suspected HIT undergoing CPB, during which bivalirudin was used for anticoagulation. The CPB circuit was not pre-primed with an anticoagulant. Instead, a bivalirudin infusion was initiated as a 1.25 mg/kg bolus, after which a continuous infusion was maintained at 2.75 mg/kg/hr. ACTs were monitored every 15–30 minutes and additional boluses were administered based on results with a goal of 500–600 seconds. The authors concluded that bivalirudin is a safe alternative to heparin for CPB cases.26

In 2004, Koster et al27 published a pilot study, which was the first prospective investigation using a heparin alternative for routine CPB. A total of 20 patients were enrolled in the United States and Europe. The CPB circuit was primed with 50 mg of bivalirudin and a bolus dose of 1.5 mg/kg of bivalirudin was administered to the patient, followed by an infusion at a rate of 2.5 mg/kg/hr. Ecarin clotting time (ECT) was monitored during bypass, with a target range of 400–500 seconds. Samples were tested 5 minutes after a bolus and every 15 minutes during the continuous infusion. After completion of bypass, the CPB machine was connected as a self-continuous circuit. The continuous circuit was dosed with 50 mg of bivalirudin followed by an infusion at 50 mg/hr. At the end of the surgery, when it was determined the patient would not need to return to CPB, the volume of the circuit was processed in a cell saver and retransfused to the patient. The findings suggested that bivalirudin use could benefit a patient population beyond those undergoing PCI and PTCA.27

ECTs are based on the ecarin reagent which is derived from snake venom. The ecarin reagent converts prothrombin to meizothrombin, which is a prothrombin intermediate that is sensitive to inhibition by direct thrombin inhibitors. ECTs are insensitive to heparin and are useful only for therapeutic drug monitoring. ACTs contain a coagulation activator such as celite, kaolin, or glass particles, as well as a magnetic stir bar. The ACT is a whole-blood clotting test.28
Another pilot study by Koster et al in 2005 assessed the effect of bivalirudin on hemoelastic activation during CPB; 10 patients for elective CABG were administered a bolus dose of 1 mg/kg of bivalirudin followed by a continuous infusion of 2.5 mg/kg/hr until 15 minutes before termination of CPB. The CPB machine was primed with a 50-mg bolus of bivalirudin. ECTs were obtained at 15-minute intervals and additional boluses were administered if the clotting time decreased to <400 seconds. It was found that hemoelastic activation was attenuated when cardiotomy suction was not used. The release and reinfusion of cardiotomy suctioned tissue factor enriched blood from the operative field secondary to surgical trauma and reperfusion of ischemic tissue, as well as CPB-induced inflammation and activation of the coagulation system secondary to blood contact with non-endothelial surfaces, produces a procoagulant effect. The use of cardiotomy suction intraoperatively resulted in increased post-CPB values for D-dimers, fibrinopeptide A, prothrombin 1 and 2 fragments, and thrombin-antithrombin. The authors suggested that cardiotomy suction should be restricted and replaced by cell saver technique when possible.

The EVolution of Patients during Coronary Artery Bypass Graft Operation: Linking UTilization of Bivalirudin to Improved Outcomes and New Anticoagulant Strategies (EVOLUTION-ON) multicenter trial of 101 patients compared heparin with protamine reversal to bivalirudin in patients undergoing CPB. They were randomized to bivalirudin vs heparin with protamine groups in a 2:1 ratio. For those patients randomized to the bivalirudin group, the CPB circuit was bolused with 50 mg of bivalirudin during priming. The patient received a bolus dose of 1.0 mg/kg, followed by a 2.5 mg/kg/hr infusion. Additional doses of bivalirudin were administered based on clotting times and provider discretion in the form of boluses of 0.1–0.5 mg/kg. Anticoagulation was monitored according to standard institutional practice, with a goal ACT of 2.5x the baseline. It was found that primary endpoints of procedural success were not significantly different between patients on bivalirudin vs heparin, which lead to the conclusion that bivalirudin is a safe and effective alternative to heparin.

The coronary artery bypass HIT thrombosis syndrome on- and off-pump safety and efficacy trial (CHOOSE-ON) was a multicenter study in both the United States and Germany in which 49 patients were treated with bivalirudin. Of these, 43 showed evidence of HIT and/or thrombosis syndrome. Following previously published guidelines, the CPB machine was bolused with 50 mg of bivalirudin. The patient was bolused with 1 mg/kg of bivalirudin followed by a continuous infusion of 2.5 mg/kg/hr until approximately 15 minutes before discontinuation of CPB. Additional boluses of 0.1–0.5 mg/kg were administered at the discretion of the provider to ensure an ACT of 2.5-fold or greater prolongation from baseline. At termination of CPB, a continuous circuit was created on the machine for recirculation. An additional bolus of 50 mg of bivalirudin was administered to the circuit, followed by a continuous infusion of 50 mg/hr. When it was determined that CPB was no longer needed, the circuit volume was processed with a cell saver. The CHOOSE-ON study was the first investigation that targeted regulatory approval for alternative anticoagulation during CPB in patients with HIT. It concluded that bivalirudin could be safely and effectively used for this indication.

A single-center experience with bivalirudin use in both CPB and OPCABG surgeries was reported in 2009. The data for 141 patients who underwent cardiac surgery with bivalirudin anticoagulation over an approximately 4-year span were reviewed. Anticoagulation for OPCABG followed the EVOLUTION-OFF dosing protocol in that patients received a bivalirudin bolus of 0.75 mg/kg followed by a continuous infusion of 1.75 mg/kg/hr during grafting. Additional boluses of bivalirudin were administered if the ACT decreased below 300 seconds. Anticoagulation protocols for CPB followed the EVOLUTION-ON and CHOOSE-ON trial guidelines in that patients received a bivalirudin bolus of 1 mg/kg, followed by a continuous infusion of 2.5mg/kg/hr until weaned from CPB. Cardiotomy suction was generally avoided if possible. The CPB machine was bolused with 50 mg of bivalirudin followed by a continuous infusion of 50 mg/hr into the recirculating circuit. At surgery conclusion, the circuit volume was processed via cell saver. This was the largest single-center experience report. It showed that bivalirudin anticoagulation had excellent procedural success rates with acceptable perioperative blood loss and transfusion rates.

The most recent case report was published by Koster et al in 2013 and described an emergency thrombectomy with CPB and deep hypothermic circulatory arrest using bivalirudin in a patient with HIT. Bivalirudin was administered as a bolus of 1 mg/kg followed by a continuous infusion of 2.5 mg/kg/hr. The CPB machine was primed with 50 mg of bivalirudin. Because proteolytic clearance of bivalirudin can lead to regional thrombosis in regions of stasis, the CPB machine was recirculated with a bivalirudin infusion. Of note is the fact that the presence of a visible thrombus is not indicative of the need for additional anticoagulation. A localized thrombus in an area of stasis reflects the local bivalirudin metabolism and does not correlate with intravascular levels. Bivalirudin is now undergoing phase II and phase III multicenter trials for both OPCABG and CABG with CPB in patients both with and without HIT. Previous promising results prompted the further investigation. Continued evaluation may show bivalirudin to be a viable choice for anticoagulation in cardiothoracic surgery.
Conclusion
The challenges of properly anticoagulating a patient with HIT who is undergoing CABG with CPB or OPCABG are very real, as are the dangers of complications from HIT should heparin not be avoided. If optimal surgical outcomes are to be achieved, it is necessary to identify patients with HIT as well as recognize the potential threats of the allergic process. While many potential alternatives to heparin exist, studies have not indicated that there is one clearly superior anticoagulant. Our case report is one incidence in which bivalirudin was successfully used to provide a satisfactory level of anticoagulation for an OPCABG using ACTs to monitor its effectiveness. Bivalirudin offers the advantage of having a relatively short half-life, while having the downfall of not having a reversal agent. Clinical trials that include establishing dosing and monitoring guidelines are currently underway for the use of bivalirudin in CABG and OPCABG procedures in the hopes of providing an alternative to heparin and better outcomes for patients who have HIT.

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The authors have declared they have no financial relationships with any commercial interest related to the content of this activity. The authors did discuss off-label use within the article.