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Clinical outcomes of cardiac surgery patients undergoing therapeutic plasma exchange for heparin-induced thrombocytopenia

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Abstract

BACKGROUND AND OBJECTIVES: Heparin-induced thrombocytopenia (HIT) is an antibody-mediated condition that leads to thrombocytopenia and possible thrombosis. Patients with HIT who require cardiac surgery pose a challenge as high doses of heparin or heparin alternatives are required to permit cardiopulmonary bypass (CPB). Intraoperative therapeutic plasma exchange (TPE) is a valuable adjunct in the management of antibody-mediated syndromes including HIT. The clinical impact of TPE on thromboembolic events, bleeding, and mortality after heparin re-exposure is not well established. We hypothesized that TPE with heparin re-exposure will not lead to HIT-related thromboembolic events, bleeding, or increased mortality after cardiac surgery with CPB.

MATERIALS AND METHODS: We reviewed 330 patients who received perioperative TPE between September 2012 and September 2017.

RESULTS: 24 patients received TPE for HIT before anticipated heparin use for CPB. Most patients were males (79%) scheduled for advanced heart failure therapies. Three patients (12.5%) died within 30 days after surgery but none of the deaths were considered HIT-related. Thromboembolic events (TE) occurred in 3 patients within 7 days of surgery; of those, two were possibly HIT-related.

CONCLUSION: TPE with heparin re-exposure was not strongly associated with HIT-related thrombosis/death after cardiac surgery with CPB.

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Contributions:

I. Moreno Duarte: Data collection, statistical analysis, data interpretation, manuscript writing.

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Keywords

cardiac surgery; heparin; plasma exchange; platelet factor 4; thrombocytopenia

BACKGROUND

Heparin-induced thrombocytopenia (HIT) is a prothrombotic condition in which antibodies to the antigenic complex of heparin and platelet factor 4 antigen (anti-heparin/PF4) activate platelets to degranulate and aggregate in the microcirculation [1]. These events lead to thrombocytopenia and possible thrombosis [1]. Seroconversion occurs in 8–17% of patients requiring heparin for medical indications, and in approximately 50% of patients exposed to heparin during the course of cardiac surgery. Patients with HIT who require cardiac surgery pose a challenge as high doses of heparin or heparin alternatives are required to permit cardiopulmonary bypass (CPB). The risks of thrombosis associated with early heparin re-exposure during CPB must be balanced against the adverse effects (i.e. bleeding and circuit thrombosis) that are a consequence from the use of irreversible heparin alternatives such as bivalirudin [2–4] for which there are no reversal agents or adequate intraoperative laboratory monitoring strategies to date.

Intraoperative therapeutic plasma exchange (TPE) is a valuable adjunct in the management of antibody-mediated syndromes including HIT. TPE permits heparin use by removing immune complexes and HIT antibodies [5]. Intraoperative TPE allows the use of heparin rather than irreversible heparin alternatives. In a retrospective study of 11 HIT or heparin/PF4 seropositive patients undergoing TPE in preparation for cardiac surgery, a single TPE treatment reduced heparin/PF4 titers by 50–84%, and 7 of 9 patients had normal anti-heparin/PF4 levels after treatment [6]. Following re-exposure to heparin, no serious adverse complications of HIT or to TPE were noted. HIT is associated with postoperative morbidity and mortality. Recent studies have shown that clinical complications of thrombosis and thrombocytopenia are highly correlated with antibody levels [7, 8]. Studies have also shown that TPE effectively removes heparin/PF4 antibodies [6, 9]. The clinical impact of TPE on thromboembolic events, bleeding, and mortality after heparin re-exposure is not well established. We hypothesized that TPE with heparin re-exposure will not lead to HIT-related thromboembolic events, bleeding, or increased mortality after cardiac surgery with CPB.

METHODS

After obtaining IRB approval, we retrospectively reviewed cardiac surgical patients undergoing TPE before CPB with heparin between September 2012 – September 2017. We identified adult patients > 18 years with a prior history of HIT (decrease in the platelet count of more than 50% from the highest platelet count value after the start of heparin with an onset 5 to 10 days after the start of heparin) or preoperative anti-heparin/PF4 antibody seropositivity (polyclonal ELISA OD > 0.4; GTI PF4 EIA, GTI diagnostics, Waukesha, WI) as their indication for TPE, which was performed as described in a previous study. [6] The American Society for Apheresis (ASFA) designates TPE for pre-CPB in HIT as

category III (optimum role of apheresis therapy is not established. Decision making should be individualized).[10] The level of evidence is grade 2C.

TPE was performed using the COBE Spectra® (from 2012 – 2015) or Spectra Optia® (2014 – 2017) after the induction of general anesthesia. TPE was performed with plasma replacement using a standardized protocol to exchange 1.0 plasma volume (approximately 3500–4500 mL based on the patient’s height, weight, gender, and hematocrit [11]). The timing of TPE was dependent on the hemodynamic stability of the patient. Ideally, it was performed before heparinization (400 U/kg as a bolus) but if necessary, heparin was given, CPB initiated to stabilize the patient, and TPE performed during CPB as previously described. [12] Because heparin is removed during TPE, additional heparin was infused during the exchange based on the following estimation. Assuming a hematocrit of 0.25, we replaced heparin lost during TPE as calculated by 4 U/mL of plasma removed administered as a 4000-U bolus after every liter removed with any remainder at the completion of treatment.

Daily minimum platelet counts were abstracted 7 days before to 30 days after TPE. Arterial/venous thromboembolic (TE) events within 7 days of TPE and survival status up to 30 days were determined. Patient demographic and clinical variables were summarized by frequency (%) for categorical and mean (SD) or median [IQR] for continuous variables.

RESULTS

Patient demographic and descriptive data are summarized in Table 1. We reviewed 330 patients who received intraoperative TPE between September 2012 and September 2017 (Figure 1). Of the 164 patients that underwent TPE during cardiac surgery using CPB, 24 patients received TPE for HIT before anticipated heparin use for CPB; the remainder received TPE in order to clear human leukocyte antigen (HLA) specific antibodies prior to heart or lung transplantation. All patients (24) had a history of HIT. Also, 7 patients (29%) had a history of prior VTE, but it was not clear from the records if this was HIT based on timing therefore we did not include this. Of the 24, 12 patients had preoperative anti heparin/PF4 antibody testing at our hospital; we had other patients transferred to us from outside hospitals with a history of HIT and a “positive ELISA” although we did not have these data in our records.

Most patients (79.0%, 18 patients, Table 1) were males scheduled for advanced heart failure therapies (i.e left ventricular assist device insertion or heart transplantation), they all received heparin for CPB. Twenty-one patients were anticoagulated with bivalirudin before surgery, one patient with argatroban, and one patient with coumadin. Postoperative anticoagulation for the LVAD patients used bivalirudin. The median preoperative HIT ELISA test optical density (OD) of 1.39 [0.67, 2.43]. Two patients with a positive polyclonal heparin/PF4 ELISA, were subsequently negative on the IgG specific assay.

Platelet counts typically reached a nadir on the day of surgery and steadily recovered for most patients during the first ten postoperative days (as shown in Fig. 2). For the 24 patients, in the cohort the preoperative median was $160 \times 10^3 / \text{mm}^3$. The lowest daily median

occurred on the day of surgery and had a median of $100 \times 10^3/\text{mm}^3$. Thereafter, the median values monotonically increased through postoperative day 10 to a median of $292 \times 10^3 \text{ mm}^3$. By day 16, all platelet counts were above $150 \times 10^3/\text{mm}^3$.

Only 11 patients had a postoperative HIT ELISA retested. These patients had a preoperative median OD of 1.99 and a single, intraoperative TPE treatment reduced the OD titer to a median of 0.34. One patient had a significantly increased OD titer after TPE (0.9 preoperatively to 1.41 postoperatively), although this was an increase in the polyclonal ELISA only. The IgG specific ELISA was negative. There was no difference between pre and postoperative OD titers in two patients, and for the remaining seven, the OD titers were reduced by 35–85% from the preoperative value. Similarly, seven patients decreased their postoperative OD to <0.4 . The median [Q1, Q3] change from pre-to-post surgery is $-1.57 [-2.01, 0.01]$, and the Wilcoxon Signed rank p-value is 0.037, suggesting a significant decrease in OD level in this patient group.

Three patients (12.5%) died within 30 days after surgery. One patient died after a hemorrhagic stroke with subsequent brain death. The platelet counts in the weeks preceding death were normal (Figure 3). The second patient died from respiratory failure secondary to pneumonia. The third patient had multiorgan failure secondary to sepsis; this patient presented with a TE event associated with a drop in his platelet count after an initial recovery, that could have been HIT related. He died later after resolution of thrombocytopenia; the cause of death was unrelated to the prior TE event and was not TE-related. None of the deaths were considered HIT-related, as the platelet counts were normal at the time of death. For these three patients, the preoperative median OD was 2.47 [0.233–2.487]. After TPE, the median OD was 0.467 [0.151–0.933].

TE events occurred in 3 patients within 7 days of surgery. Two were possibly HIT-related. One was a stroke with an ELISA OD four days before the TE of 0.47 and a PLT count of 54×10^3 . The second event was a deep venous thrombosis with an ELISA OD 1 day before the TE of 0.93 with a PLT count of 106×10^3 . A third TE occurred well after platelet count recovery to 330×10^3 , and was therefore unlikely HIT-related.

DISCUSSION

We present a series of 24 patients with current or recent HIT that were treated with TPE to remove heparin/PF4 antibodies and allow standard heparin anticoagulation for their complex cardiac surgery. These patients were successfully re-exposed to heparin, without intraoperative incident, to permit cardiopulmonary bypass. In our cohort, platelet counts reached a nadir on the day of surgery. Platelet counts typically decrease during cardiac surgery as a result of blood loss, transfusion, hemodilution, and activation with sequestration in the extracorporeal circuit (primarily the fibers of the oxygenator). It is challenging to differentiate this from HIT, other than it is an expected observation with a typical nadir on the day of surgery, or the day after, with subsequent recovery. The postoperative platelet count profile seems to follow two specific patterns in the setting of HIT. [13] One of the patterns is biphasic and shows an initial recovery of the platelet count post bypass with a subsequent decrease in the count of at least 40% compared to the maximum

postoperative value. The second pattern is monophasic with a low platelet count that persists in postoperative days 5–10. The Lillo-Le Louët score was created to diagnose HIT after CPB and includes the postoperative platelet profile as a variable.[14] Anti heparin/PF4 antibody titers were significantly reduced (by 35–85%) in the majority of the patients with available postoperative antibody testing data. A previous study also reported a reduction of 50%–84% after a single TPE treatment [6]. In our cohort, only two of the three TE events were possibly HIT-related, and none of the deaths were likely HIT-related. Therefore, TPE could be considered a reasonable option for patients with HIT requiring urgent cardiac surgery who were considered clinically unsuitable for heparin alternatives [2], due to complexity of surgery with anticipated long duration and therefore a large expected total bivalirudin dose in the setting of likely renal dysfunction and high bleeding risk.

None of the patients underwent additional TPE treatments aiming to achieve complete antibody removal, provided they had stable platelet counts postoperatively or could tolerate bivalirudin anticoagulation. Repeated postoperative TPE may be unwarranted due to risks of hemodynamic instability and exposure to additional blood products. However, it is unknown whether additional TPE based on repeat anti-heparin/PF4 testing could help further reduce TE complications in this morbid group. Serial PF4-dependent enzyme-immunoassay (EIA) testing for HIT antibodies may be useful especially if the IgG specific ELISA is unavailable. This is a simple, feasible method but is technically challenging as it only detects HIT antibodies indirectly [15]. There is no predetermined value for defining high-titer antibodies, and OD levels correlate poorly with antibody burden. The sensitivity and specificity for PF4/heparin optical density >0.40 is reported as 100% and 26% respectively [16], which means that some patients with positive test results in this assay may never develop HIT [17–19]. Some authors suggest that an OD >1.0–1.4 increases the risk of thrombosis [18, 20]. In our study, of the eleven patients with postoperative OD data, 10 had OD below 1, while one patient had a postoperative OD > 1.4. Interestingly, this patient neither presented with TE nor died in the first 30 days after surgery. The relationship between OD and antibody levels are expressed through a hyperbolic curve function [21]. At high ODs (>2–3), antibody saturation in the ELISA does not accurately reflect the antibody burden or actual titer. Serial dilutions of test samples over a wide range help to quantify the titer of antibody present.

Accuracy in the assessment of titers is particularly relevant for HIT TPE, as one exchange may not be sufficient to remove high titer antibodies [22, 23]. Some HIT patients with high ODs and high titers are likely to need more than one TPE to lower circulating PF4/H antibody burden. Functional assays such as a serotonin release assay (SRA) or heparin-induced platelet activation assay (HIPA), are more specific for immune-mediated HIT but take longer, are more technically demanding, and not widely available [23]. Achieving SRA negativity through multiple sessions of TPE can remove a sufficient amount of HIT antibodies despite persistent positive titers with ELISA [22]. In our study, the patients were not tested with functional assays after TPE, but previous studies have shown that the median time to a negative SRA after a single session is around 50 days [24, 25]. To date, TPE is scarcely used for HIT in most institutions, mostly because it is not recognized as an option. In a recent survey [26], only 37% of the surveyed institutions used TPE for HIT for indications such as cardiovascular surgery and HIT-associated thrombosis. There were no major complications from TPE. Hemodynamic instability due to TPE or any plasma reaction

was not apparent, although they may have been masked because patients were anesthetized and often already maintained on vasoactive medications. There were no technical issues encountered. Hypocalcemia is expected and was prevented with concomitant intravenous calcium gluconate administration (3–4 grams).

Intravenous immunoglobulin (IVIg) is emerging as a useful approach that can also help to treat patients with HIT. IVIg works by inhibiting HIT antibody-mediated platelet activation, apparently through competitive binding [24]. The inhibition is dependent on the constant domain of IgG (Fc) but not the antigen-binding portion (Fab), and the presence of the HH131 genotype. Other genotypes such as RR131 and HR131 responded favorably to high doses of IVIG, although not as well as the patients with the HH131 genotype. To date, there are no studies relating response to TPE to specific genetic phenotypes. Furthermore, IVIg had no effect on HIT antibody binding in a solid phase PF4-ELISA testing or in the SRA values, which were still strongly positive. In any case, IVIg treatment successfully achieved platelet count recovery [24]. Although laboratory assessments demonstrate antibody persistence after both TPE and IVIg, it is possible that the titer magnitude is not sufficient to trigger a pathological immune and platelet response. Another potential approach involves the concurrent use of cangrelor, a short-acting, intravenous P2Y12 inhibitor, and heparin for cardiopulmonary bypass in patients with HIT but there is limited literature at this time. [27]

This study is a case series without a control group and, consequently, has some limitations. It included a small number of patients, and not all patients had confirmatory tests (e.g SRA). We based the diagnosis on PF4/heparin EIA only, which may be insufficient as only 50% of antibodies causing a positive EIA have clinical relevance. Most of the available SRA results were reported late during the hospital stay, which made them clinically unhelpful. We also did not check for antibody re-emergence which may be a possibility after re-exposure to heparin.

Bivalirudin for complex redo surgery has not been studied, and the bleeding risk in this setting maybe even higher. TPE permits standard anticoagulation for complex procedures and maybe a feasible option for this type of procedures. Genetic assessments may also be useful in future studies to correlate the response to TPE to specific genotypes.

CONCLUSION

Intraoperative TPE is one strategy to facilitate standard heparin anticoagulation during CPB in patients requiring urgent cardiac surgery in the setting of acute HIT. Modifying the protocol to plan for additional TPE based on repeat anti-heparin/PF4 testing could further reduce thromboembolic complications, depending on the results of future studies.

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ADDENDUM

I. Moreno-Duarte: This author contributed to data collection and analysis, literature review, manuscript writing and preparation

M. Cooter: This author contributed to data collection and statistical analysis. O. A.

Onwuemene: This author contributed to manuscript writing and preparation.

K. Ghadimi: This author contributed to literature review, manuscript writing and preparation

I. J. Welsby: This author contributed to data collection and analysis, literature review, manuscript writing and preparation.

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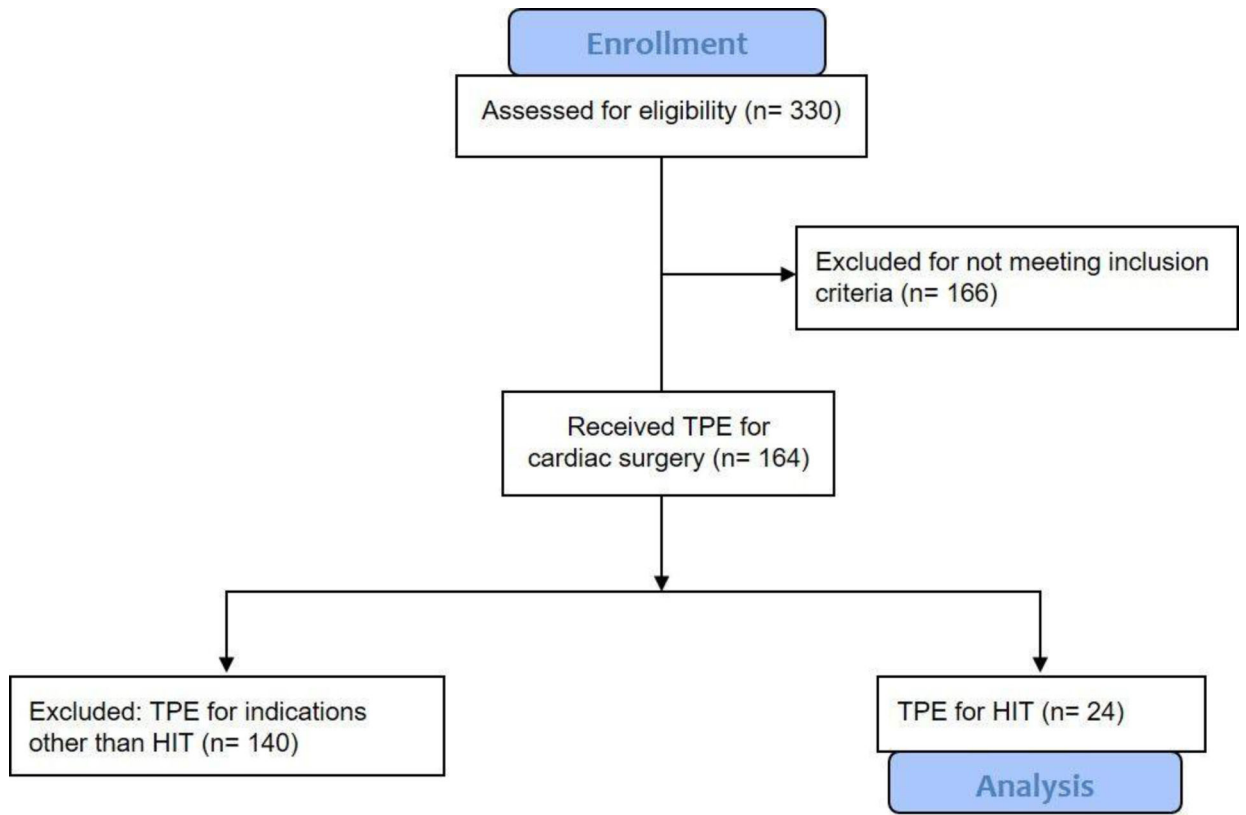


Figure 1.
CONSORT 2010 flow diagram

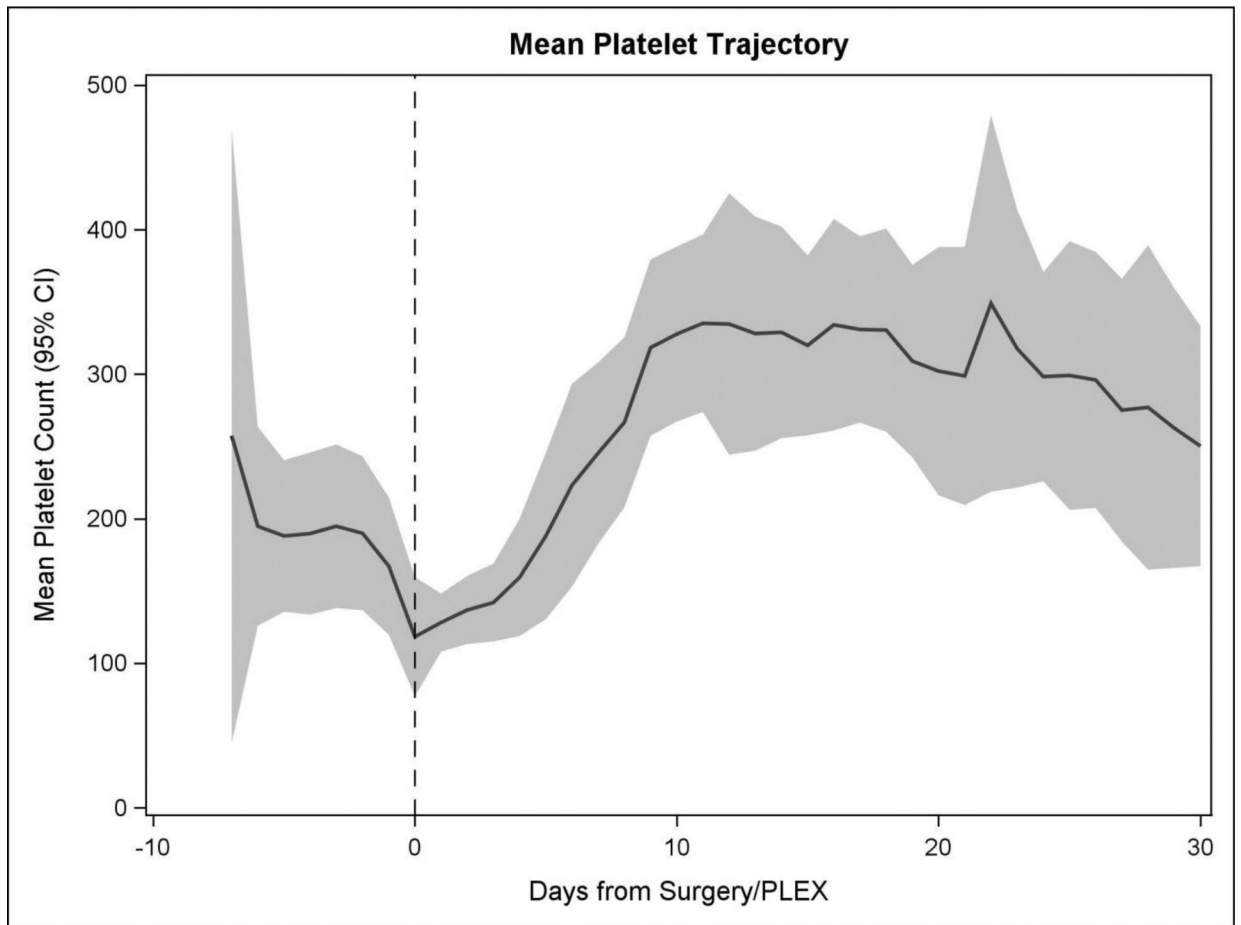


Figure 2. Platelet trajectories for all patients. This graph shows the daily mean platelet count. The band represents the 95% confidence interval.

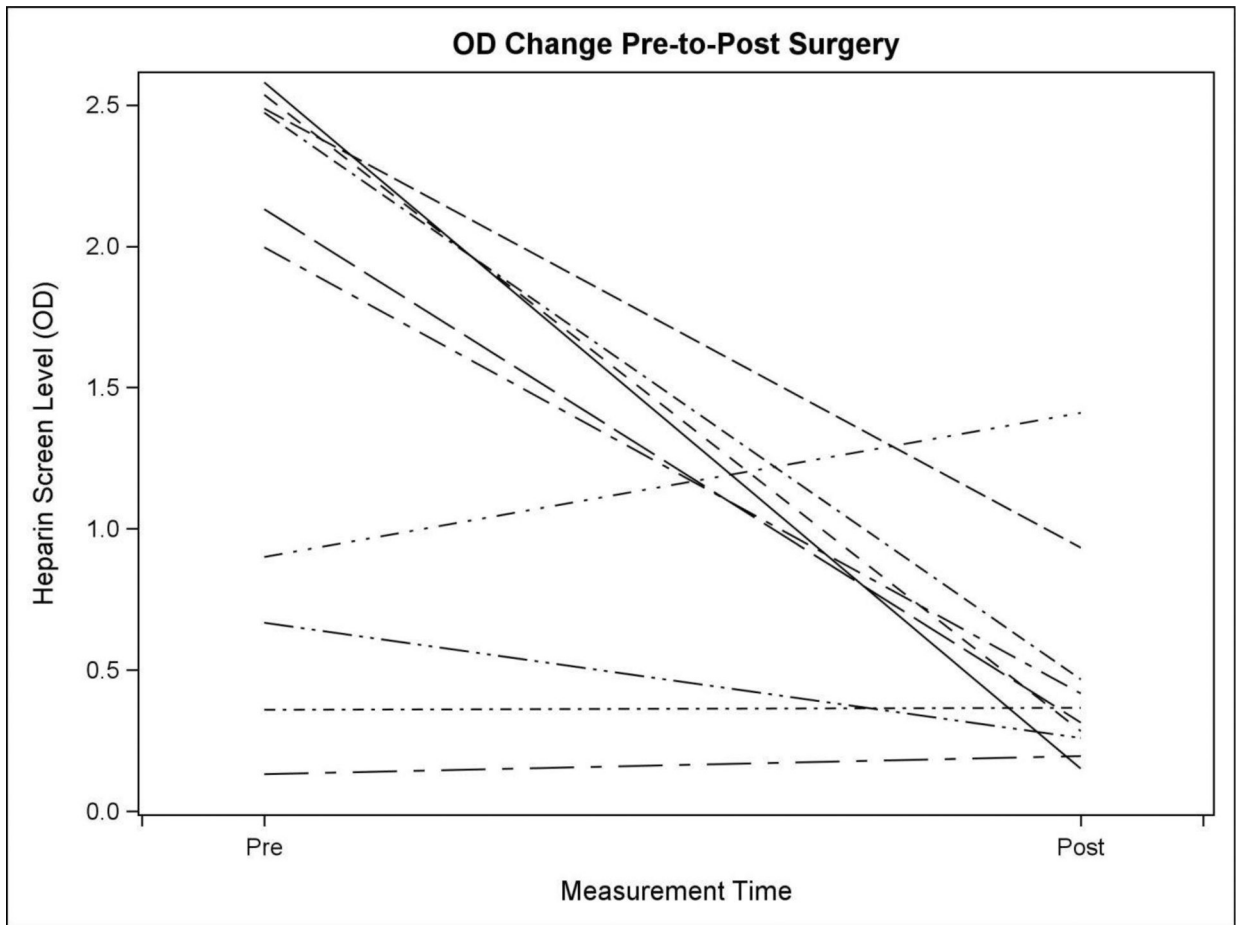


Figure 3. Median [Q1, Q3] change from pre-to-post surgery. the median [Q1, Q3] change from pre-to-post surgery is $-1.57 [-2.01, 0.01]$. The Wilcoxon Signed rank p-value is 0.037.

Table 1.

Demographic data

Variable (n=24 patients)	Mean \pm SD Percentage (n)
Age (years)	55.2 \pm 9.44
Gender (Male)	79% (19)
Procedure	
Heart transplant	21% (6)
VAD insertion/replacement	54% (13)
Other procedures	22% (5)
Race	
African American	37% (9)
Caucasian	54% (13)
Other	4% (2)
BMI	32.0 \pm 9.80
Height (m)	1.75 \pm 0.09
Weight (kg)	98.3 \pm 30.2
Preoperative Anti heparin/PF4 Antibody OD value	1.39 \pm 0.89
Nonischemic cardiomyopathy	62% (15)
Ischemic cardiomyopathy	29% (7)
Coronary artery disease	33% (8)
Mod-Sev Aortic stenosis/regurgitation	50% (12)
Atrial fibrillation/flutter	50% (12)
Diabetes	42% (10)
Hypertension	67% (16)
Smoker	8% (2)
Hypercholesterolemia	62% (15)
CKD	42% (10)
ESRD	4% (1)
Previous MI	25% (6)
Previous stroke	12% (3)
Prior VTE	29% (7)
Peripheral Vascular Disease	8% (2)
Mechanical circulatory support	67% (16)
preoperative IABP	58% (9)
preoperative VA ECMO	8% (5)
preoperative VV ECMO	4% (2)
Preoperative LVAD	25% (6)
Redo sternotomy	29% (7)