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Simultaneous Liver-Kidney Transplantation in Patient with a History of Heparin-Induced Thrombocytopenia: A Case Report and Literature Review

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 58
Final Diagnosis: Heparin-induced thrombocytopenia
Symptoms: Liver and kidney failure
Medication: —
Clinical Procedure: Simultaneous liver-kidney transplantation
Specialty: Transplantology

Objective: Unknown ethiology

Background: Heparin-induced thrombocytopenia (HIT) is the development of thrombocytopenia caused by exposure to heparin. Here, we report the case of a 58-year-old male with HIT who then underwent a successful simultaneous liver-kidney transplantation (SLKT).

Case Report: The patient had end-stage hepatitis due to a hepatitis C virus (HCV) infection and was on hemodialysis due to nephropathy related to HCV. Furthermore, he was diagnosed with HIT caused by the administration of heparin for hemodialysis during these treatments. Fortunately, there was no evidence of thromboses and HIT antibody converted negative immediately. Four years after the occurrence of HIT, SLKT was performed for liver and kidney failure. Although the donor was heparinized, the donor grafts were flushed on a backtable by an organ preservation solution without heparin to reduce residual heparin. The subsequent transplantation was uneventful. After the operation, anticoagulation with argatroban, a direct thrombin inhibitor, was started instead of heparin. In the postoperative course, neither thrombosis nor graft dysfunction occurred.

Conclusions: SLKT in a patient who had a history of HIT could be performed safely.

MeSH Keywords: Heparin • Iatrogenic Disease • Kidney Transplantation • Liver Transplantation • Thrombocytopenia

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Background

Heparin-induced thrombocytopenia (HIT) is an iatrogenic disorder that is the immune-mediated development of thrombocytopenia due to the administration of various forms of heparin. It is caused by the formation of abnormal antibodies that activate platelets and predispose the patient to thrombosis, and this abnormal formation of blood clots inside a blood vessel sometimes become serious and life-threatening. HIT can be a common occurrence in surgical patients who receive heparin therapy and it has been recognized extensively in the cardiac, vascular, and orthopedic surgery population [1–3]. Hemodialysis is also a factor causing HIT due to constant heparin exposure [4–6].

In organ transplantation, anticoagulant therapy by the administration of heparin is a mandatory procedure to prevent graft thrombosis during the perioperative period. Because avoidance of heparin is required in patients with HIT, alternative therapies should be considered when performing organ transplantation for such patients. Furthermore, transplant recipients are frequently exposed to heparin due to the underlying end-stage disease that leads to transplantation [7]. However, an appropriate anticoagulant therapy in a patient with HIT is still undefined in organ transplantation. In such patients, the management of acute or subacute incidence of HIT should be deliberate due to the high thrombotic risk, and it might be difficult to manage anticoagulant therapy even in patients following completion of HIT after a decline of HIT antibody. In addition, although the organ donor is generally anticoagulated with heparin before organ explantation to avoid thrombosis, it remains unknown whether it is acceptable for patients with HIT to use the graft of a heparinized donor.

We successfully performed a simultaneous liver-kidney transplantation (SLKT) in 58-year-old male with HIT. Herein, we report this case and a review of other cases of organ transplantation with HIT.

Case Report

The patient was a 58-year-old male with a history of HIT. He had end-stage hepatitis due to a hepatitis C virus (HCV) infection and was receiving hemodialysis due to nephropathy related to HCV. Twelve years ago, before transplantation, he was diagnosed with hepatitis type C and started interferon therapy. Four years before transplantation, his liver function worsened, with a Child Pugh score of 10 and model of end-stage liver disease (MELD) score of 12, and began hemodialysis because his nephropathy had progressed. For dialysate, unfractionated heparin was used as an anticoagulating agent. Three months later after starting hemodialysis, the platelet count had decreased and platelet factor 4 (PF4)/heparin-enzyme-linked immunosorbent assay (ELISA) (HIT antibody) showed a high level. Nafamostat mesilate was immediately substituted for unfractionated heparin on hemodialysis without reinstitution. Fortunately, there was no evidence of thromboses, his platelet count reverted, and the titer of HIT antibody converted negative.

Four years later after the occurrence of HIT, progressive liver failure with an increased MELD score of 36 required liver transplantation, and the patient underwent SLKT. The donor was a male in his forties that had taken a standard systemic anticoagulation that was carried out with a bolus of 15 000 international unit (IU) of unfractionated heparin (230 IU/kg body weight), which was central-venously administered 12 minutes before clamping the abdominal aorta. For organ preservation, cold aortal perfusion with 5000 mL of organ preservation solution (University of Wisconsin solution, Viaspan, Bristol-Myers Squibb, Brussels, Belgium) (77 mL/kg body weight), which included 10 mL of heparin was performed over 20 minutes. It was considered that the liver and kidney from the heparinized donor would be accepted even with the increased risk of thrombosis triggered by potential residues of heparin within the graft because the patient couldn't wait against the risk of ongoing end-stage liver disease. The backtable preparation of both the liver and kidney grafts was done with University of Wisconsin solution that did not include heparin, and the grafts were directly flushed through the great vessels to wash out possibly remaining heparin. The volume of University of Wisconsin solution was chosen according to the weight of each graft, at 1 mL/g graft weight. Before reperfusion of the grafts, washout of University of Wisconsin solution was performed by 5% albumin that did not include heparin and its volume was same to University of Wisconsin solution. Subsequently, the transplantation was performed.

We performed the liver transplantation and kidney transplantation. The operation time was 14 hours and 54 minutes. In regard to liver graft, the cold ischemic time (CIT) was 8 hours and 36 minutes and the warm ischemic time (WIT) was 40 minutes. For the kidney graft, CIT and WIT were 9 hours and 21 minutes and 61 minutes, respectively. During operation, the patient was on continuous hemodiafiltration (CHDF). The amount of intraoperative blood loss was 3500 mL and required transfusions of 6 units of packed red blood cells and 12 units of fresh frozen plasma.

After the operation, the liver and kidney showed good primary function and the postoperative course was uneventful (Figure 1). We mainly monitored activated clotting time (ACT), activated partial thromboplastin time (APTT), and the international normalized ratio of prothrombin time (PT-INR) to evaluate coagulation status, and we controlled this status with keeping over 100 seconds in ACT during first 10 days after

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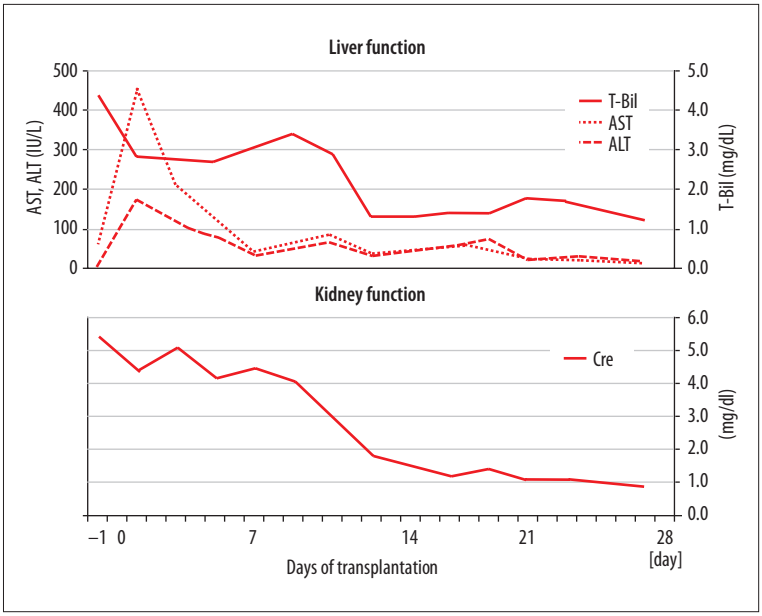

Figure 1. A 58-year-old male who had a history of heparin-induced thrombocytopenia (HIT) underwent simultaneous liver-kidney transplantation (SLKT) due to both liver and kidney failure. The liver and kidney showed good function after transplantation. T-Bil – total bilirubin; AST – aspartate transaminase; ALT – alanine transaminase; Cre – creatinine.

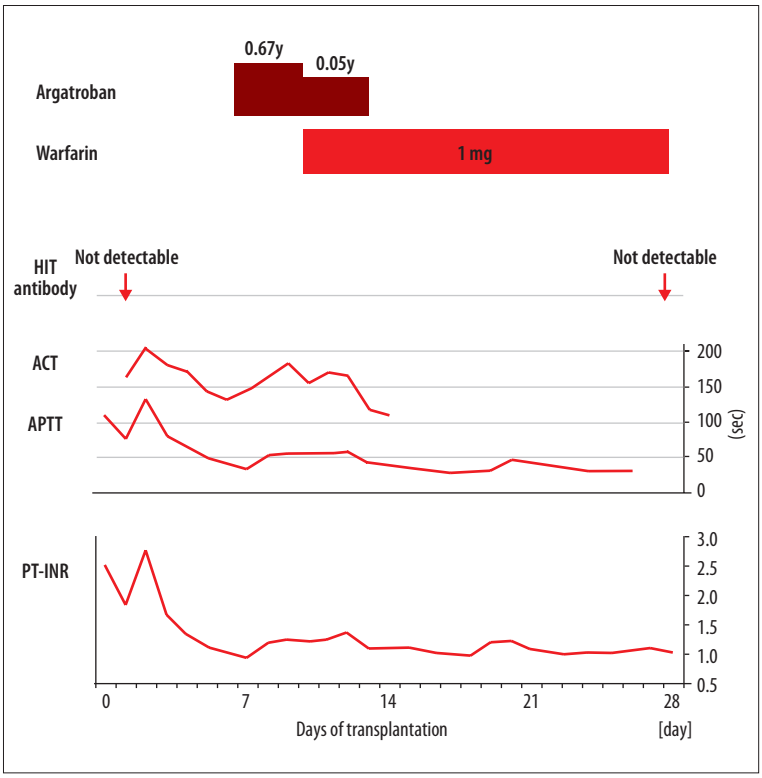

Figure 2. Coagulation status in the patient was monitored by activated clotting time (ACT), activated partial thromboplastin time (APTT), and international normalized ratio of prothrombin time (PT-INR). For the first 7 days after transplantation, anticoagulating reagents weren't administrated due to the patient's hyperfibrinolytic status. On day 7, the intravenous administration of argatroban was started at 0.067 μg/kg/minute (γ) as the patient's hyperfibrinolytic status was declining. On day 10, oral administration of warfarin was started and continued until 4 weeks after transplantation while argatroban was gradually tapered off. The titer of HIT antibody could not be detectable on either day 1 or day 28.

transplantation. Just after the operation, the patient was in hyperfibrinolytic status, therefore, anticoagulation wasn't performed, and no symptoms related to HIT occurred and the titer of HIT antibody did not convert positive (Figure 2). Because the activity of coagulation gradually increased, intravenous anticoagulation was started at 7 days after the operation with an intravenous administration of argatroban, a direct thrombin inhibitor (0.067 μg/kg/minute). Furthermore, oral administration of warfarin (1 mg/day) was started at 10 days after operation and argatroban was gradually tapered off while monitoring coagulation status. Administration of warfarin was finished at 4 weeks after transplantation and no thrombosis occurred for 48 months after transplantation.

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Discussion

We reported on the case of a 58-year-old male with heparin-induced thrombocytopenia (HIT) who underwent simultaneous liver-kidney transplantation (SLKT).

HIT is classified as either type I or II [8]. HIT type I is a transient thrombocytopenia, caused directly by heparin itself, and is mostly mild and naturally reversible by interruption of heparin use. In contrast, HIT type II is a thrombocytopenia caused by an immune response where there is the formation of abnormal antibodies that induce activated platelets, which predisposes to thrombosis and the abnormal formation of blood clots inside a blood vessel. Such thrombosis sometimes becomes serious in HIT type II.

Immunoglobulin (Ig) G antibodies bind with the complexes of negatively charged heparin molecules and a positively charged PF4, and form immune complexes that activate platelets via the platelet Fc-receptor. The clinical pretest probability for the 4 T-Test (Thrombocytopenia, Timing of platelet fall, Thrombosis, and oTher cause of thrombocytopenia) was 6 points, indicating a high probability for HIT [1]. In this case, the antibody screening test showed a high level of heparin-induced thrombosis antibody. Furthermore, the platelet count had decreased by more than 50%, and the timing of the platelet count fall was less than 1 day. Although, there was no evidence of thrombosis, the 4 T-Test score for HIT was 6 points, and therefore the patient was diagnosed as HIT type II.

It has been reported that the frequency of HIT is 0.5% to 5% [9,10], and many factors have been reported as risks for HIT, including being female [11] and using unfractionated heparin [12]. Furthermore, some reports have demonstrated that single nucleotide polymorphisms (SNPs) could involve HIT [13–15]. The frequency of HIT in patients with cardiac disease increases due to more opportunities for exposure to heparin. In addition, due to continuous exposure to heparin, dialysis patients are also a risk group, and up to 12% develop HIT [4]. We performed a SLKT for a patient with HIT caused by hemodialysis. As noted, HIT might frequently occur in transplant recipients because they have many chances for exposure to heparin due to the underlying end-stage disease. Unfortunately, most organ transplantations require anticoagulation to prevent graft thrombosis during the perioperative period, therefore, it is important to have strategies to manage HIT in organ transplantations. We reviewed the PubMed database from 1998 to 2018, using the keywords “heparin induced thrombocytopenia” and “transplantation” and found total 81 cases of HIT in transplantation in which the patient was diagnosed preoperatively. These cases and our case are summarized in Table 1. Among of these cases, the largest number of cases were reported in heart transplantations, with 68 cases [16–35]. As noted, patients with cardiovascular disorders frequently need anticoagulation for diagnostic studies, surgical procedures, and therapy, therefore HIT is a relatively common complication in this population. Our review found lung transplantation with HIT had 2 cases [36,37]. There were 3 cases diagnosed with HIT preoperatively in kidney transplantation, although HIT frequently occurs in patients undergoing hemodialysis [38–40]. Although the reason is unclear, patients who were diagnosed with HIT might not achieve kidney transplantation due to avoidance of the perioperative risks of thrombosis. However, such candidates might be able to avoid strict heparin exposure by planning preemptive transplantation or continuous ambulatory peritoneal dialysis (CAPD). Currently, however, there are no alternative therapies to transplantation for severe liver failure, and patients cannot select any other treatment other than transplantation, and thus SLKT is also inevitable.

Chronic end-stage liver disease is frequently associated with coagulation disorders and secondary thrombocytopenia due to portal hypertension. These preexisting disorders in liver transplant candidates make clinical recognition of HIT difficult because a significant drop in the platelet count according to the 4 T-Test tends to be rather small when the baseline value is already reduced below the normal range. Therefore, when liver transplantation is performed, it is important to distinguish whether the decline in platelet count was caused by liver dysfunction or HIT, when considering the possibility of HIT. With regard to SLKT, the frequency of HIT would be deservedly high because hemodialysis for renal failure increases the chance of heparin exposure. There was 1 report of a case with HIT in SLKT, in which HIT occurred just after the start of hemodialysis [41]. In our case, the patient was diagnosed with HIT 3 months later, after starting hemodialysis and being exposed to heparin.

We could not determine an indication for transplantation without discretion due to the severity of thrombosis caused by HIT. However, there are no alternative treatments to transplantation in patients with severe liver failure. Referring to reports describing cases with HIT in liver transplantation, there were 3 cases who had liver transplantation within 3 days of a diagnosis of HIT [42–44]. All of these cases were urgent, and transplantation was given a priority as a part of intensive therapies. Fortunately, there were no complications related to thrombosis. Although it still remains unresolved whether transplantation would be acceptable in such cases with an acute or subacute incidence of HIT, some reports have shown that a case with the negative conversion of HIT antibody could be acceptable if the administration of heparin is avoided and other anticoagulants are selected as a substitute for heparin [45,46]. The titer of HIT antibodies usually declines rapidly within a few weeks after the cessation of heparin, and becomes below detection levels. After a several months interval, patients with a history of HIT who are shortly re-exposed to heparin are usually not

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Table 1. Review of literature on organ transplantation with HIT.

Organ	Case	Diagnosis of HIT before Tx	HIT Ab at Tx	Anticoagulant therapy	Thrombotic complication	Author	Year
Heart	2 cases	N/A	N/A	Heparin	None	Brozena SC	1998
	40 y.o. male	6 months ago	N/A	Enoxaparin	None	Prifti E	2000
	34 y.o. female	15 days ago	N/A	Danaparoid	Thrombosis of the jejunal veins	Christiansen S	2000
	55 y.o. male 40 y.o. female	11 days ago 23 days ago	N/A N/A	Danaparoid Danaparoid	None None	Pamboukian SV	2000
	59 y.o. female 41 y.o. female	9 months ago 1 month ago	N/A Negative	Ancrod Heparin	None None	Robitaille D	2001
	10 cases	–	–	–	–	Hourigan LA	2002
	27 y.o. male	16 days ago	N/A	Bivalirudin	None	Mann MJ	2005
	39 y.o. male	10 months ago	N/A	Lepirudin	None	Schenk S	2006
	5 y.o. female	54 days ago	Negative	Argatroban	None	Almond CS	2006
	55 y.o. female	5 days ago	N/A	Lepirudin	None	Saravanan P	2007
	24 cases	–	–	–	–	Schroder JN	2007
	51 y.o. female	14 days ago	N/A	Lepirudin	None	Wadia Y	2008
	55 y.o. male 55 y.o. male 44 y.o. male	7 days ago 1 month ago 7 days ago	Negative Negative Negative	Argatroban Lepirudin Lepirudin	None None None	Selleng S	2008
	3 cases	73 days ago 48 days ago 66 days ago	N/A N/A N/A	Lepirudin Lepirudin Lepirudin	None None None	Nocera P	2008
	39 y.o. male	12 days ago	N/A	Bivalirudin	None	Simsir SA	2010
	9 cases	–	–	–	–	Zucker MJ	2010
	61 y.o. male	29 months ago	N/A	Argatroban	None	Genzen JR	2010
	51 y.o. male	2 months ago	Positive	Bivalirudin	None	Wong JK	2010
	62 y.o. male	5 days ago	Positive	Bivalirudin	None	Pazhenkotti AP	2015
	6 y.o. child	N/A	N/A	Argatroban	Circuit thrombus formation	Latham G	2016
	55 y.o. male	13 days ago	N/A	Bivalirudin	None	Chen E	2016
Lung	40 y.o. male	114 days ago	Positive	Argatroban	None	Dolch ME	2010
	58 y.o. male	21 days ago	N/A	Bivalirudin	None	Koster A	2017
Liver	37 y.o. female	3 months ago	Negative	Lepirudin	None	Fretschner R	2005
	39 y.o. male	2 years ago	Negative	Bivalirudin	None	Andregg BA	2008
	57 y.o. female	2 days ago	Positive	Argatroban	None	Bachmann R	2011
	56 y.o. male	3 days ago	Positive	Argatroban	None	Pannicke N	2012
	51 y.o. female	3 days ago	Positive	Lepirudin	None	Biagioni E	2013

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Table 1 continued. Review of literature on organ transplantation with HIT.

Organ	Case	Diagnosis of HIT before Tx	HIT Ab at Tx	Anticoagulant therapy	Thrombotic complication	Author	Year
Kidney	17 y.o. male	5 months ago	Negative	Lepirudin	None	John U	2006
	50 y.o. male	5 months ago	Negative	Coumadin	Bilateral lower extremity DVT	Muzaffar M	2012
	28 y.o. female	2 years ago	Negative	Bivalirudin	Acute tubular necrosis with areas of infarction in graft	Podolak B	2014
Simultaneous heart-kidney	49 y.o. female	4 months ago	Positive	Bivalirudin	None	Choxi A	2017
Simultaneous pancreas-kidney	36 y.o. female	16 months ago	Negative	Dabigatran	None	Jozwik A	2018
Simultaneous liver-kidney	51 y.o. female	48 days ago	N/A	Refludan	None	Bachmann R	2014
	58 y.o. male	2 years ago	Negative	Argatroban	None	Our case	2019

Tx – transplantation; HIT – heparin-induced thrombocytopenia; Ab – antibody; N/A – not available; DVT – deep vein thrombosis; y.o. – year old.

at risk of developing early-onset HIT (< 5 days) [47]. Actually, Potzsch et al. reported use of heparin during cardiopulmonary bypass in patients with a history of HIT [48]. Furthermore, it has been reported that patients with a history of HIT and negative antibody titers have been re-exposed to heparin in liver transplantation procedures without any problems [45].

In organ transplantation, the acceptability of a graft which is taken from a donor treated with heparin should also be considered. It remains to be discussed whether heparin residuals within the graft are influenced by quantity, route of administration (aortal and/or portal venous), and duration of rinsing with a preservation solution during explantation. Fretschner et al. showed that for liver transplantation, very low titers of HIT antibodies (optical density <0.7 in the PF4/heparin/ELISA) or a long lag time (>35 minute) in the heparin-induced platelet activation test were not prohibitive for using a heparin-rinsed organ [45]. In addition, Bachmann et al. reported a successful case of liver transplantation with acute HIT from a heparinized donor [42]. Assuming heparin residuals within the graft, the donor organ in our case was flushed on a backtable with increased amounts of preservation solution without heparin. Although there are few reports of multi-organ transplantation [41,49,50], we performed the treatment of organ grafts based on that protocol for reducing re-exposure to heparin, and no thrombotic events occurred, and the titer of HIT antibody did not convert positive. Therefore, even in SLKT it was possible to transplant an organ from a donor treated with heparin safely to a patient with a history of HIT before transplantation. Considering the coagulating risk caused by using preservation solution without heparin, heparin-rinsed organ might be acceptable in patients with a history of HIT and negative antibody titers, because for such patients, re-exposure to heparin is not at risk for developing HIT.

Conclusions

SLKT could be performed safely for a patient with a history of HIT and a negative conversion of HIT antibody.

Conflicts of interest

None.

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