ORIGINAL CLINICAL RESEARCH REPORT

Incidence of Postreperfusion Hyperfibrinolysis in Liver Transplantation by Donor Type and Observed Treatment Strategies

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BACKGROUND: Hyperfibrinolysis is a possible complication during liver transplantation, particularly immediately after reperfusion.
METHODS: We performed a retrospective study to examine the incidence, treatment, and resolution of postreperfusion hyperfibrinolysis in patients undergoing liver transplantation at Duke University Hospital from 2015 to 2020.
RESULTS: Out of 535 patients undergoing liver transplantation, 21 or 3.9%, 95% CI (2.5–5.9), had hyperfibrinolysis after reperfusion. Hyperfibrinolysis occurred in 16 of 511 (3.1%) patients receiving livers from DBD donors, 5 of 18 (27.8%) patients receiving livers from donation after circulatory death (DCD) donors, and 0 of 6 (0.0%) patients receiving livers from living donors. Fibrinolysis was treated with cryoprecipitate (12/21), a combination of cryoprecipitate and tranexamic acid (3/21), or neither (6/21) and resolved within several hours in all cases.
CONCLUSIONS: Anesthesiologists should be aware of the possibility of postreperfusion hyperfibrinolysis in liver transplantation, particularly with DCD donors, and may consider treatment with cryoprecipitate or tranexamic acid. Further work is needed to identify any potential differences, such as faster resolution of fibrinolysis, between different treatment modalities. (Anesth Analg 2023;136:518–23)

KEY POINTS

- **Question:** What is the incidence of postreperfusion hyperfibrinolysis during liver transplantation in conventional and donation after circulatory death (DCD) donors?
- **Findings:** In our cohort, the incidence of hyperfibrinolysis was 3.93% (95% CI, 2.45–5.94) in all liver transplants, 3.1% in patients receiving livers from DBD donors, 27.8% in patients receiving livers from DCD donors, and 0.0% in patients receiving livers from living donors.
- **Meaning:** Anesthesiologists should be aware of the possibility of postreperfusion hyperfibrinolysis in liver transplantation, particularly with DCD donors, and may consider treatment with cryoprecipitate or tranexamic.

GLOSSARY

AVR = aortic valve replacement; **CABG** = coronary artery bypass graft; **DBD** = donation after brain death; **DCD** = donation after circulatory death; **IQR** = interquartile range; **MELD** = model for end-stage liver disease; **OLT** = orthotopic liver transplantation; **ROTEM** = rotational thromboelastography; **STROBE** = Strengthening the Reporting of Observational Studies in Epidemiology; **tPA** = tissue plasminogen activator; **TXA** = tranexamic acid

End-stage liver disease is characterized by a significant imbalance in multiple aspects of the body's natural coagulation system, with clinically significant hypocoagulability and hypercoagulability both possible.[1] Hyperfibrinolysis is 1 manifestation of this imbalance and is a concern during liver transplantation, particularly immediately after reperfusion.[2] Some studies in the literature report an incidence as high as 84% after liver transplant from living donors,[3] although our own anecdotal experience suggests that this may be significantly lower but still of clinical concern. The incidence of fibrinolysis after reperfusion, overall and in subgroups based on donor source, is not well established. Furthermore, the natural history and treatment of fibrinolysis in this setting have not been well established and it is unknown if this fibrinolysis resolves untreated or if treatment with antifibrinolytics or cryoprecipitate is

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beneficial.[4] Coagulation and coagulopathy in end-stage liver disease is a complicated process requiring a delicate balance to avoid both unnecessary bleeding as well as a hypercoagulable state predisposing the newly anastomosed hepatic artery and portal vein to clot formation.[5,6]

Liver transplantation may provide a valuable opportunity to study the natural history of fibrinolysis. Thromboelastography has become more prevalent over the last decade, enabling us to study this process in the clinical environment.[7,8] However, the increased use of prophylactic antifibrinolytics in other settings where fibrinolysis is common such as cardiac and trauma surgery means that we do not often see fibrinolysis in the absence of antifibrinolytics.

Livers may be procured for transplantation from several types of donors. In a traditional donation after brain death (DBD) donor, the donor has been declared legally brain dead before procurement. In a donation after circulatory death (DCD) donor, the donor does not meet brain death criteria, but organs are procured shortly after a natural circulatory death. A living donor donates an anatomically viable portion of his or her liver, and both portions then hypertrophy to meet the needs of both the donor and the recipient.

During this time period, another innovation in liver transplantation was also under investigation at our institution. A number of livers were procured and then placed on the OrganOx pump for normothermic ex-vivo perfusion before transplantation.

As the incidence of fibrinolysis after reperfusion as well as the prevalence and effectiveness of potential treatments have been not well established, we designed a retrospective database study to examine the prevalence, treatment, and resolution of fibrinolysis in patients undergoing liver transplantation at Duke University Hospital from July 1, 2015, to June 30, 2020.

METHODS

Data Collection

Using our medical center's perioperative EPIC database, we performed a retrospective analysis of all patients who underwent orthotopic liver transplantation (OLT) at Duke University from January 1, 2015, to December 31, 2020. These data were supplemented and confirmed by data from our transplant center's records and data tracking our center's DCD program. Missing data were collected via chart review. The Duke Institutional Review Board approved the study and waived the requirement for written informed consent. This article adheres to the applicable STROBE guidelines.

Recipient and Donor Variables

Variables collected for analysis from the recipients included age, sex, surgery performed (multiorgan transplant versus OLT alone), lab-based model for end-stage liver disease (MELD) score at the time of transplant, surgical complications including portal vein or hepatic artery thrombosis, laboratory coagulation profiles for clotting time, clot formation, and clot breakdown including rotational thromboelastography (ROTEM) data, and medications and blood products administered perioperatively. For the donors, variables collected for analysis included donor type (DBD, DCD, and living donor), cold ischemic time, and whether the OrganOx pump was used.

Outcome Measures

The primary end point of the study was postreperfusion hyperfibrinolysis, as evidenced by an LI30 value of <90 on ROTEM data. ROTEM measures clot formation over time and reports out a graph showing evolving clot strength as amplitude on the y-axis over time on the x-axis. The LI30 value is a measure of clot strength 30 minutes after maximal clot formation. With normal coagulation, clot strength will maintain and LI30 will be 100%. A value of <90% when compared to the maximal clot formation was taken to represent hyperfibrinolysis

Secondary outcome measures focused on treatment modalities for fibrinolysis in the OLT population studied, resolution of fibrinolysis, and observed complications.

Statistical Analysis

Patient, surgical, and donor characteristics for each transplant in the full cohort were summarized as median and interquartile range for continuous variables and the number and percentage for categorical variables. For each covariate, the incidence of hyperfibrinolysis was described as the number and percentage for each level of that covariate. All statistical analysis was performed using R, version 4.1.2.

RESULTS

Patient Demographics and Transplant Characteristics

The study group identified included 535 patients who underwent OLT at Duke University Hospital from July 1, 2015, to June 30, 2020 (Table 1). The median (IQR [interquartile range]) recipient age was 56 [42–63] years, 75 recipients (14.0%) were pediatric (defined as patient age <18 years), 460 recipients (86.0%) were adult, 194 recipients (36.3%) were women, and 341 (63.7%) were men. The median [IQR] recipient pretransplantation MELD was 22 [15–29], with 409 recipients (76.4%) having a score of <30 and 126 recipients (23.6%) having a score ≥30.

In the donor population, 511 donor organs (95.5%) were identified as DBD, 18 donor organs (3.4%) were identified as DCD, and 6 donor organs (1.1%)

Table 1. Patient Demographics and Transplant Characteristics (N = 535)

Age (y)	56 [42–63]
Pediatric	75 (14.0)
Adult	460 (86.0)
Sex	
Female	194 (36.3)
Male	341 (63.7)
Donor type	
DBD	511 (95.5)
DCD	18 (3.4)
Living	6 (1.1)
Multiorgan	
Multiorgan	58 (10.8)
Single organ	477 (89.2)
MELD score	22 [15–29]
MELD score category	
<30	409 (76.4)
≥30	126 (23.6)
Cold ischemic time (min)	296 [243–375]
Cold ischemic time category	
<300 min	257 (48.0)
300–480 min	41 (7.7)
>480 min	201 (37.6)
Unknown	36 (6.7)
Organox system used	23 (4.3)

Median [IQR] or n (%).

Abbreviations: DBD, donation after brain death; DCD, donation after circulatory death; IQR, interquartile range; MELD, model for end-stage liver disease.

Table 2. Incidence of Postreperfusion Hyperfibrinolysis in Total Cohort and Subgroups

	Cases/N	% (95% CI)
Full population	21/535	3.9% (2.5–5.9)
Age		
Pediatric	0/75	0.0% (0.0–4.8)
Adult	21/460	4.6% (2.8–6.9)
Donor type		
DBD	16/511	3.1% (1.8–5.0)
DCD	5/18	27.8% (9.7–53.5)
Living	0/6	0.0% (0.0–45.9)
Multiorgan		
Yes	1/58	1.7% (0.0–9.2)
No	20/477	4.2% (2.6–6.4)
MELD score		
<30	16/409	3.9% (2.3–6.3)
≥30	5/126	4.0% (1.3–9.0)
Cold ischemic time (min)		
<300 min	8/257	3.1% (1.4–6.0)
300–480 min	8/201	4.0% (1.7–7.7)
>480 min	3/41	7.3% (1.5–19.9)
Unknown	2/36	5.4% (0.7–18.7)
Organox		
Organox system used	2/23	8.7% (1.1–28.0)
Organox system not used	19/512	3.7% (2.2–5.7)

Abbreviations: DBD, donation after brain death; DCD, donation after circulatory death; IQR, interquartile range; MELD, model for end-stage liver disease.

were identified as living donors. Of those identified, 58 donor organs (10.8%) were part of a multiorgan transplantation (12 combined liver/intestinal, 33 combined liver/kidney, 8 combined liver/heart, and 5 combined liver/lung), and 477 donor organs (89.2%) were single organ (liver only) transplants. No other major procedures, such as CABG or AVR, which might be expected to impact hyperfibrinolysis, were performed concurrently with liver transplant. Documented donor organ cold ischemia time had a median [IQR] time of 296 [243–375] minutes, with 257 organs (48.0%) experiencing <300 minutes of cold ischemia, 41 organs (7.7%) experiencing between 300 and 480 minutes of cold ischemia, 201 organs (37.6%) experiencing >480 minutes of cold ischemia, and 36 organs (6.7%) having an unknown length of cold ischemia time. The median [IQR] warm ischemia time for the DCD organs was 21.5 [19.3–25.0] minutes. Twenty-three patients (4.3%) received livers in which the OrganOx pump was used.

Hyperfibrinolysis Data and Characteristics

In the study population of 535 patients receiving liver transplantation, a total of 21 cases of hyperfibrinolysis were identified, or 3.9%, 95% CI (2.5–5.9) (Table 2). Of those patients who were identified to have postreperfusion hyperfibrinolysis, all were found to be in the adult population with an incidence of 21 cases in 460 patients (4.5%). Hyperfibrinolysis occurred in 16 of 511 liver transplants identified as DBD (3.1%), 5 of 18 liver transplants identified as donation after cardiac death (27.8%), and 0 of 6 liver transplants identified as living donation (0%). Hyperfibrinolysis occurred in 1 of 58 cases that were identified as part of a multiorgan transplantation (1.7%) and 20 of 477 cases that were identified as single-organ transplants (4.2%). Recipients who had a pretransplantation MELD score <30 experienced 17 cases of hyperfibrinolysis out of 409 patients (4.2%), while recipients who had a pretransplantation MELD score ≥30 experienced 5 cases out of 126 patients (4.0%). Hyperfibrinolysis occurred in 8 out of 257 cases with a documented cold ischemic time of <300 minutes (3.1%), 8 out of 201 cases with a documented cold ischemic time of between 300 and 480 minutes (4.0%), 3 out of 41 cases with a documented cold ischemic time of >480 minutes (7.3%), and 2 out of 36 cases without a defined cold ischemic time (5.4%). Patients for whom the OrganOx pump was used had hyperfibrinolysis in 2 out of 23 cases (8.7%) while patients for whom the pump was not used had hyperfibrinolysis in 19 out of 512 cases (3.7%).

Hyperfibrinolysis Treatment and Resolution Data

A total of 12 cases were treated with a cryoprecipitate infusion alone (57.1%), 3 cases were treated with a combination of cryoprecipitate and tranexamic acid (14.3%), and 6 cases were not treated with either cryoprecipitate or tranexamic acid (28.6%). Among the 15 patients who received cryoprecipitate, the mean dose was 1.6 ± 0.9 units. Among the 3 patients who received TXA, 1 received 850 mg and 2 received 1000 mg (Table 3). Of

Table 3. Treatment of Hyperfibrinolysis

	Hyperfibrinolysis cases (n = 21)
Treatment group, n (%)	
Cryoprecipitate only	12 (57.1)
Tranexamic acid only	0 (0.0)
Cryoprecipitate and tranexamic acid	3 (14.3)
Neither	6 (28.6)

the 21 cases where hyperfibrinolysis was identified, 17 cases (81%) had evidence of resolution of fibrinolysis on follow-up ROTEM (characterized by an LI30 of >90) whereas the remaining 4 cases (19%) did not have a follow-up ROTEM documented, presumably indicating that the patient did not have ongoing clinically significant coagulopathy.

Chart review of the 3 patients receiving antifibrinolytics (tranexamic acid) did not indicate any thrombotic complications.

DISCUSSION

Over the past decade, the ever-increasing demand for donor organs for liver transplantation has spurred on efforts to identify new donor sources and maximize the utilization of existing donors. Historically, DBD donors have supplied the majority of organs used for liver transplantation in the United States[9] with living donor transplants providing a small minority of available organs while placing the healthy donor at increased risk of complications and even death.[10] Therefore, alternative avenues for organ donation have been explored, with a focus on DCD organs for liver transplantation. In a traditional DBD donor, the donor has been declared legally brain dead, and with appropriate work up and consent, any viable organs may be procured for transplant with the heart beating and organs fully perfused. In a DCD donor, the donor does not meet brain death criteria, although is usually neurologically devastated, requires ongoing life support which the family has already decided to withdraw, and is expected to pass quickly upon withdrawal of this support. The donor is brought to the operating room and support is withdrawn by the existing care team, followed by an agonal phase characterized by hypoxia and hypotension. If cardiac arrest occurs within a narrow window of time, the patient is declared dead, and the organs may be procured for transplant.

Although the number of DCD livers available for transplant has increased rapidly over the past several years, these organs were initially associated with a significantly increased risk of graft failure that was unrelated to modifiable donor or recipient factors.[11] Subsequent work investigating this degree of graft failure suggested that DCD livers are injured by prolonged warm ischemic time due to the donation process, are more susceptible to postmortem clot formation, and have greater ischemic insult when compared to DBD donor grafts.[12] Further meta-analyses have suggested that DCD liver transplant recipients are also at a higher risk of developing ischemic cholangiopathy, further increasing the risk of graft failure and need for retransplantation.[13] Despite these complications, DCD livers provide otherwise unavailable organs and have been shown to reduce wait-list mortality and provide a definite survival benefit in patients with a high acuity of illness.[14] Further research aimed at reducing these complications has shown some potential benefit and includes extracorporeal and in-vivo perfusion[15,16] as well as pharmacological treatments to the donor graft such as tissue plasminogen activator (tPA).[17] While the use of tPA has been shown to reduce ischemic-type biliary complications[18] in the donor organ, it can cause further complications after transplantation. In the DCD population, the incidence of fibrinolysis is likely significantly higher when compared to other donor types because of the use of tPA in preserving the graft.

In this retrospective study, we observed an incidence of postreperfusion hyperfibrinolysis of approximately 4% across the whole cohort. The incidence was approximately 28% in those patients receiving livers from DCD donors. This is likely due to the tPA infused into the graft hepatic artery just prior to reperfusion in an attempt to minimize ischemic-type biliary complications, although it may also be due to the warm ischemia times associated with procurement under these conditions or other differences in the handling of the organs from these different donor types.

A majority of our patients had a pretransplant MELD score <30 (409 patients), and this group had an incidence of hyperfibrinolysis of 3.9%. Those patients with an MELD score ≥30 (126 patients) had an incidence of 4.0%. The relatively low MELD scores in our population may contribute to the demonstrated incidence of hyperfibrinolysis. The incidence of hyperfibrinolysis reported in the literature varies widely based on the population studied and the definition of hyperfibrinolysis used, with 1 study demonstrating an incidence in the pretransplant population ranging from 1.8% to 50% depending on the definition used.[19]

Of those 58 patients undergoing multiorgan transplant, only 1 case of fibrinolysis was identified (1.7%), which occurred in a combined liver-kidney transplant. Interestingly, we did not see any hyperfibrinolysis in the pediatric population, although this group had a small sample size (76). Two of 23 OrganOx cases (8.7%) had hyperfibrinolysis, 1 of which was from a DCD donor; however, further studies with larger sample sizes would be needed to further define the significance of this finding.

We were also able to study observed treatment modalities used in those patients experiencing postreperfusion hyperfibrinolysis during a time when there was not a standardized approach to this occurrence. Of these 21 cases, the majority received only cryoprecipitate (57.1%). The remainder of the cases either received both cryoprecipitate and tranexamic acid (14.3%) or neither (28.6%). No cases received tranexamic acid alone. Cryoprecipitate can be used to treat hyperfibrinolysis by repleting consumed fibrinogen and promoting clot stabilization while the new liver clears the tPA. Antifibrinolytics, such as tranexamic acid and aminocaproic acid, treat fibrinolysis by displacing plasminogen from binding to the fibrin surface and thereby inhibiting further fibrinolysis from occurring. Tranexamic acid is more commonly used at our institution, and no patients in our cohort received aminocaproic acid.

Posttreatment ROTEM results were available for 17 of the 21 cases, all of which showed resolution of the hyperfibrinolysis. The remaining 4 cases did not have any follow-up ROTEM data, presumably indicating that the patient did not have ongoing clinically significant coagulopathy. Thus, it appears that the hyperfibrinolysis resolved fairly quickly (within hours) in all patients, regardless of treatment modality. Further work is needed to identify any potential differences, such as faster resolution of fibrinolysis, in patients treated with cryoprecipitate or antifibrinolytics. Cryoprecipitate may offer a beneficial, low-risk option in patients in whom treatment is warranted. Antifibrinolytics are often avoided in liver transplant patients due to concern over thrombotic complications. However, in the 3 patients who received tranexamic acid, there were no documented thrombotic complications, suggesting that this may be safe if needed in certain patients with ongoing or severe fibrinolysis, although the number of observed cases here is admittedly very small.

This study does have limitations. Primarily, this retrospective study was done at a single institution (Duke University Hospital) and, therefore, reflects only the practices and patient population at this institution during the study period. Patient population and transplant practices vary widely between centers. The incidence of hyperfibrinolysis also varies widely depending on how it is defined. Hyperfibrinolysis, as defined in this study, was a relatively infrequent occurrence in this cohort and, as such, the number of actual cases, even over 5 years, is rather small and the number of cases in subgroups or any particular treatment group even smaller. Further studies, including robust multicenter data sets, could help further describe this problem and may identify additional risk factors that predispose to postreperfusion hyperfibrinolysis.

Liver disease is associated with significant abnormalities in coagulation, with both hypocoagulability and hypercoagulability a concern during liver transplantation. Postreperfusion hyperfibrinolysis is 1 significant manifestation of this imbalance. We observed this in 3.93% of all transplants but in 27.8% of those transplants with DCD donors. As DCD donation becomes more prevalent, it is important for anesthesiologists to be aware of the potential complications, such as fibrinolysis, and how to treat them. Cryoprecipitate was the most common treatment strategy observed in our group, and fibrinolysis resolved in all patients studied. Future, larger studies may help better define the risk factors for fibrinolysis and better delineate treatment strategies. ■

DISCLOSURES

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Contribution: This author helped with drafting manuscript as well as chart review and analysis.
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