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Case Report

Total IVC Occlusion as an Unusual Cause of Acute Renal Failure After Orthotopic Liver Transplantation

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Renal dysfunction before and after orthotopic liver transplantation (OLT) has significant implications for morbidity and mortality of these patients. We describe the management of a 72-year-old male patient with history of alcoholic liver cirrhosis (MELD 38) undergoing OLT. The patient presented with declining renal function prior to OLT (baseline GFR <25 mL/min) due to diuretic therapy for refractory ascites, hypovolemia postgastrointestinal bleed, and possible hepatorenal syndrome. The intraoperative management was complicated by preexisting anemia (hematocrit, 22%), unusual RBC antibody (anti-JKa) and significant surgical blood loss. To achieve surgical hemostasis, temporary clamping of the inferior vena cava (IVC) caudal to the transplanted liver was necessary. Postoperatively, the patient remained anuric despite appropriate fluid resuscitation. Renal replacement therapy was initiated to balance volume and acid-base status. A venogram on postoperative day (POD) 5 indicated a complete IVC occlusion and caval thrombectomy was performed on POD 6. After restoration of venous renal drainage, renal function improved and renal replacement therapy was weaned. Renal function indicators normalized in 8 weeks, and remained unimpaired up to 3 months post-OLT. Unintended complete obstruction of the suprarenal IVC may occur during OLT to control surgical bleeding, and should be considered as a cause for acute renal failure after liver transplant. Despite the preexisting renal dysfunction, renal function quickly improved after restoration of blood flow drainage and normalized in less than 8 weeks post obstruction.

Key words: Liver transplant – Acute renal failure – IVC obstruction – Liver transplant complication

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Table 1 Perioperative laboratory values

Time	pH	PaCO2 mmHg	PaO2 mmHg	P/F ratio	Bicarb mEq/L	Hct %	SVO_2 %	Lactic acid mmol/L	K mEq/L	INR	Fibrinogen mg/dL	PLT 10^9/L
preOp					19	22		1.3	3.9	2.0	65	72
8:27	7.38	32	158	226	18	23	85	1.5	3.9			
9:40	7.37	33	178	254	18	27	87	1.9	4.1			
10:28	7.43	34	171	244	19	26	87	2.5	4.3			
11:04	7.42	34	175	250	22	23	92	2.4	3.7			
11:44	7.35	36	253	361	23	18	82	3.6	3.9			
12:11	7.35	36	210	300	19	9	60	3.8	3.4			
12:37	7.19	40	157	224	15	22	71	5.1	3.3	4.4	<20	10
13:56	7.20	59	120	171	22	22	82	6.1	4.0	1.8	152	91
14:41	7.21	60	148	228	24	23	80	5.4	3.7	1.3	246	121
ICU												
15:09	7.35	44	127	254	24	24	76		4.1	1.3	267	105
16:04	7.40	40	113	226	24	28	87	2.6	4.2	1.2		
POD 1	7.39	38	119	298	23	30	77	1.8	4.8	1.1		
POD 2	7.39	36	95	238	20	22	70	1.1	5.2	1.1	152	40

Anhepatic stage: 11.49 to 12.35; reperfusion: 12.36.

ABG: pH, p_aCO_2, p_aO_2, P/F ratio as calculated by p_aO_2/F_iO_2; Bicarb, bicarbonate; Hct, hematocrit; K, potassium; SVO_2, mixed venous oxygenation per pulmonary artery catheter.

Coagulation monitoring per INR; fibrinogen level; PLT, platelet count.

Acute kidney injury (AKI) is not an uncommon occurrence in the postoperative care after orthotopic liver transplantation (OLT) with significant implications for organ donor function and increased mortality.[1,2] The occurrence of AKI post OLT has been reported to occur in 9% to 78% of patients with multifactorial origin.[3–6] Considering consequences, postoperative AKI post-OLT requires a fast and focused workup toward underlying causes. Many patients with advanced liver disease present with diminished renal function so acute on chronic renal dysfunction might be the most commonly observed cause.[7] Intraoperative events, such as intraoperative blood loss, high transfusion requirements, or hypotension may also contribute to postoperative AKI.[3,8] Postrenal causes are rare but should be considered. We present a case of a patient undergoing OLT developing anuric renal failure postoperatively caused by significant acute obstruction of the intrahepatic inferior vena cava (IVC) resulting in blocked renal venous drainage.

Case Report

A 72-year-old male patient with alcoholic liver cirrhosis underwent orthotopic liver transplant (OLT) with a model for end-stage liver disease (MELD) score of 38 at transplant. Preoperatively the patient developed progressive renal dysfunction. Baseline creatinine 10 months before OLT of 1.2 mg/dL rose to 3.3 mg/dL 8 weeks before OLT, reflecting a glomerular filtration rate (GFR) decline from >60 to 18 mL/min 1.73 m^2, estimated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula. The renal dysfunction might have been caused by prerenal factors secondary to aggressive diuresis or hypovolemia with anemia related to recent GI bleed. Recent intra-abdominal infection requiring antibiotic therapy and hepatorenal syndrome were also considered. The intraoperative course was complicated by excessive surgical blood loss. Due to the patient's transfusion history and multiple alloantibodies, it was intraoperatively difficult to provide sufficient blood products for resuscitation. Cellsaver was not used because of the recent intra-abdominal infection. The majority of blood loss occurred at the end of the hepatectomy and anhepatic stage of OLT. Since not enough blood products were available and the patient displayed signs of insufficient oxygen delivery (shown in Table 1), the surgeon pursued surgical hemostasis by clamp and suture of obvious bleeders in the liver bed before successful reperfusion. Donor suprahepatic cava was anastomosed to recipient middle and left hepatic veins using the "piggy-back" anastomosis technique to preserve partial IVC blood return. After reperfusion, transesophageal echocardiography indicated that the venous return to the right heart had not normalized after removing clamps from supra- and infrahepatic IVC. The hepatic artery anastomosis to the donor liver was completed and the surgical field was reassessed. Due to

Fig. 1 Thrombus removed from patient's IVC on POD 6 post-OLT.

massive blood transfusions, hypothermia, and reperfusion, the patient was severely coagulopathic but hemodynamically stable. The surgical field was packed and patient was taken to the intensive care unit (ICU) for stabilization. On postoperative day (POD) 1, the patient returned to the operating room for re-exploration and abdominal closure. The coagulation status had normalized, the donor liver was functional, and there were no signs of active bleeding. Intraoperative hepatic sonography indicated patent hepatic and IVC circulation with minor stenosis of the IVC just caudal to the level of the suprahepatic caval anastomosis due to suture ligatures placed to control intraoperative hemorrhage. The transesophageal echocardiography exam indicated diminished right heart venous return and the suspicion of impaired infrahepatic IVC flow was discussed. The team decided not to explore the IVC any further at this time given concerns for possible bleeding. Bedside sonography was performed on POD 1 after abdominal closure, indicating antegrade IVC flow. Ultrasound exam did not reveal any abnormalities of renal circulation. The patient was extubated on POD 2, remained anuric over the next few days and required renal replacement therapy. Anticoagulation for venous thromboembolism prophylaxis with unfractionated heparin (UFH 5000 units subcutaneously, every 8 hours) was started on POD 2. Although IVC obstruction was discussed amongst the transplant surgeon, anesthesiologist, and intensivist, the renal consultant's assessment favored the AKI origin from pretransplant hepatorenal syndrome, aggravated by intraoperative hypotension and hypovolemia. It was not until fluoroscopic venogram on POD 5 demonstrated a high-grade infrahepatic IVC obstruction, therefore implying renal venous drainage obstruction as a likely cause of the postoperative AKI. The patient returned to the OR on POD 6 for IVC exploration. Sufficient amounts of compatible blood products were set aside for the surgical procedure. The acute occlusive thrombus shown in Fig. 1 extended from stenosis caudal to suprahepatic anastomosis to 0.5 cm below the renal veins and was removed. During the caval thrombectomy the IVC anastomosis was modified to a bicaval anastomosis to optimize IVC blood flow and renal vein outflow. After return to the ICU, the patient was extubated 12 hours post thrombus removal (POD 7 from OLT). Renal function indicated improvement shortly after thrombus removal by increased urine production (Fig. 2). The patient was weaned of renal replacement therapy, and renal function gradually normalized. The time course of renal function parameters from preoperative to 8 weeks post OLT are shown in Fig. 3. The patient was discharged home on POD 17 after OLT. Renal function indicators normalized in 8 weeks post-OLT, and remained unimpaired up to 3 months post-OLT.

Discussion

In the general population of hospitalized patients, acute kidney injury (AKI) is primarily caused by renal (46%) and prerenal causes (37%).[9] Similar distribution of causes for postoperative AKI has been observed after OLT.[1,2,10] Although mostly

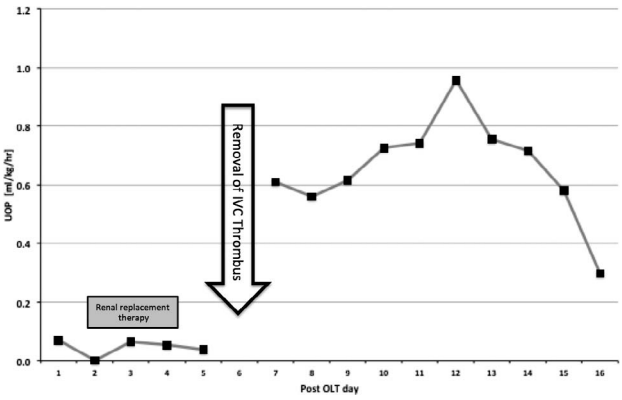

Fig. 2 The graph indicated the pattern of urine output as mL/kg/h as charted throughout hospitalization while Foley catheter for accurate measurement was in place.

multifactorial, postoperative AKI post-OLT causes include preexisting renal dysfunction before transplant, hepatorenal syndrome, intraoperative factors (e.g., blood loss, vasopressor therapy, surgical technique) and postoperative factors (e.g., surgical leaks, re-exploration, drug-induced toxicity).[7] Postrenal causes are less common and therefore rarely considered for AKI post-OLT, leading to delayed diagnosis and intervention.

The presented case illustrates this observation. In our case, aggressive surgical hemostasis was needed to control operative blood loss during the anhepatic stage of OLT. Without intent, the infrahepatic IVC was compromised and not restored after reperfusion. We speculate that the aggressive blood component therapy to treat the dilutional and consumptive coagulopathy might have decreased blood flow by thrombus formation additionally to suture related IVC narrowing. The complete thrombotic flow obstruction caused anuric renal failure. After flow restoration on POD 6, the renal function normalized over several weeks.

Our findings are consistent with the observations previously found for postrenal AKI. Postrenal AKI appears to be more frequently associated with severe renal function disturbances than other AKI causes, since the flow obstruction creates serious kidney injury in a very short time.[9,11] In animal studies, complete venous flow obstruction generated severe renal dysfunction despite well-maintained arterial perfusion.[12] Despite the experimental findings, it is rare that complete IVC obstruction is clinically observed as the sole the cause for acute renal failure.[13] The good news is that renal function appears to recover with appropriate timely intervention and restoration of renal circulation.[9] Although intervention is often required to relieve the obstructive component, postrenal AKI is less likely associated with mortality than other causes for AKI.[9,14] Compared to other causes for postoperative AKI, higher recovery rate of renal function has been observed in postrenal AKI.[9] The recovery of renal function after OLT depends on several factors (patient age, pretransplant diabetes, pretransplant need for renal replacement therapy, donor age) and recovery of renal function post-OLT has been observed in 68%.[2] Applying the described criteria for spontaneous recovery to our patient, his advanced age and elevated baseline creatinine would have predicted a less likely chance for return of

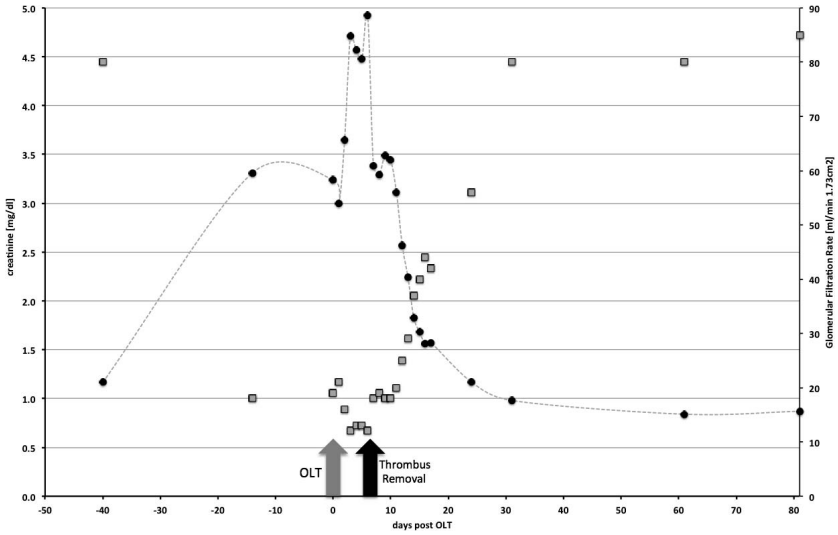

Fig. 3 Renal function parameters. The graph indicated the pattern of glomerular filtration rate, estimated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula (gray square) and serum creatinine (mg/dL; black circle) from baseline (41 days before OLT) to 3 months post-liver transplant (OLT), thrombectomy was performed on POD 6.

normal renal function. However, the postrenal origin and only short course of renal replacement therapy were encouraging.

While overall vascular complications occur in 8% to 15% after liver transplant, vascular complications related to the IVC anastomosis are a relatively rare observation, affecting approximately 0.9% to 2% of liver transplant recipients.[15,16] Clinical symptoms are nonspecific and vary with timing and degree of the stenosis from asymptomatic to lower extremity edema, renal function abnormalities, or Budd–Chiari syndrome.[17] The reported severity of renal dysfunction was defined by creatinine elevation >2 mg/dL in the first 30 days post-liver transplant.[18] Several case reports have reported IVC stenosis in liver transplant recipients in which the obstructions were successfully resolved with catheter-directed thrombolysis, balloon angioplasty, and or stent placement.[13,19–22] Interventional approach is preferred because the surgical intervention is associated with significant mortality.[22] The possibility of endovascular approach was not considered for our patient because of the severity and acuteness of obstruction. Therefore, an open approach was chosen as the best option to restore and prevent further flow impediment. To our knowledge, this is the first case report of a total IVC occlusion causing acute anuric renal failure post-liver transplant, which resolved completely after not immediate but complete normalization of blood flow.

Another observation in this case report is that bedside sonography failed to indicate the severe compromise of suprarenal IVC flow and renal circulation. Doppler sonography has good sensitivity and specificity to assess suprahepatic IVC, hepatic artery and portal vein anastomosis, and is therefore a part of the routine postoperative care after OLT.[17] However, as indicated in this case report, the infrahepatic and retrohepatic IVC portion can be difficult to isolate in the exam. Therefore, bedside sonography might not be able to rule out a narrowing of the vena caval infrahepatic anastomosis.[23,24] If clinical suspicion supports the diagnosis, venography, contract-enhanced CT or contrast-enhanced MRI are the gold standards to assess the infrahepatic circulation.[17,23]

In conclusion, this case emphasizes the need to consider all causes for renal failure for postoperative AKI after OLT, including venous flow obstruction. If recognized and restored early, renal function may recover and normalize quickly.

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