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Open Abdominal Management for Damage Control in Liver Transplantation: A Single-center Experience

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Background. Patients undergoing liver transplantation are in a state of coagulopathy before surgery because of liver failure. Intraoperative hemorrhage, massive transfusions, and post–reperfusion syndrome further contribute to coagulopathy, acidosis, and hypothermia. In such situations, temporary cessation of surgery with open abdominal management and resuscitation in the intensive care unit (ICU), which is commonly used as a damage control strategy in trauma care, may be effective. We assessed the outcomes of open abdominal management in liver transplantation and the corresponding complication rates. **Methods.** We retrospectively reviewed the outcomes of patients undergoing open abdominal management among 250 consecutive liver transplantation cases performed at our institution from 2009 to 2022. **Results.** Open abdominal management was indicated in 16 patients. The open abdomen management group had higher Model for End-stage Liver Disease scores (24 versus 16, $P < 0.01$), a higher incidence of previous upper abdominal surgery (50% versus 18%, $P < 0.01$), more pretransplant ICU treatment (31% versus 10%, $P = 0.03$), and more renal replacement therapy (38% versus 12%, $P = 0.01$). At the time of the damage control decision, coagulopathy (81%), acidosis (38%), hypothermia (31%), and a high-dose noradrenaline requirement (75%) were observed. The abdominal wall was closed in the second operation in 75% of patients, in the third operation in 19%, and in the fourth operation in 6%. Postoperatively, the frequency of early allograft dysfunction was predominantly higher in the open abdominal management group (69%), whereas the frequency of vascular complications and intra-abdominal infection was the same as in other patients. **Conclusions.** Open abdominal management can be a crucial option in cases of complex liver transplant complicated by conditions such as hypothermia, acidosis, coagulopathy, and hemodynamic instability. Damage control management minimizes deterioration of the patient's condition during surgery, allowing completion of the planned procedure after stabilizing the patient's overall condition in the ICU.

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Damage control is a surgical strategy used to promptly address life-threatening conditions, deferring definitive management until the patient is physiologically capable of withstanding repair. This strategy includes amelioration of acidosis, hypothermia, and coagulopathy in the surgical intensive care unit (ICU). The abdominal cavity is intentionally left open to facilitate reexploration and avert the onset of abdominal compartment syndrome (ACS). Open abdominal management (OAM) is an accepted strategy for patients with severe trauma and has recently been reported to be useful in nontrauma acute care surgery.[1] Typical examples include peritonitis, vascular emergencies, and acute pancreatitis are typical examples. OAM reduces operative time, avoids or treats ACS, allows observation of the extent of intestinal ischemia over time, and confirms control of the source of contamination with a second-look procedure.[1-3]

Patients undergoing liver transplantation (LT) are in a state of coagulopathy before surgery because of liver failure. Intraoperative hemorrhage, massive transfusions, and post-reperfusion syndrome further contribute to coagulopathy, acidosis, and hypothermia during LT. Insistence of hemostasis in such situations can prolong operative time and aggravate the vicious cycle perpetuated by hypothermia, acidosis, and coagulopathy. Additionally, if primary abdominal closure is performed despite massive blood loss or transfusion, ACS may develop, resulting in decreased graft blood flow, decreased cardiac output, and ventilatory failure because of increased intrapleural pressure.[4,5] OAM, including resuscitation in the ICU, may be beneficial in selected LT cases because it avoids a vicious cycle and restores normal physiological mechanisms.

Although OAM is widely used in trauma and acute care surgeries, few reports have described its use in LT.[6-10] There are no reports of living-donor LT (LDLT). This study was performed to evaluate the outcomes of OAM as damage control in LT and the associated complication rates.

PATIENTS AND METHODS

Patients

We retrospectively analyzed 250 adult patients who underwent LT at Nagasaki University Hospital from January 2009 to December 2022. Among them, the patients undergoing OAM were reviewed. The study was conducted in accordance with both the Declaration of Helsinki and Istanbul. The institutional review board of Nagasaki University Hospital approved the study (approval number: 20012022-2).

Damage Control Decision and OAM Technique

When facing uncontrollable medical bleeding after reperfusion or before abdominal closure, despite the use of available hemostatic agents and massive transfusions, the chief surgeon and anesthesiologist made a joint decision to perform damage control to stabilize the patient's hemodynamics and correct any coagulopathy. When making the decision, consideration was given to the presence of hypothermia (<35 °C), acidosis (pH <7.3), the necessity of massive transfusion, and the need for vasoconstrictor drugs to maintain a mean arterial pressure of 70 mm Hg. After completing all vascular anastomoses, the surgery was concluded with OAM, following gauze packing. OAM methods change over time. Initially, skin-only closure (SOC) was performed, and a handmade negative-pressure wound therapy (NPT) system was introduced in 2018. We used a method called the modified Barker's technique. Sterile surgical gauze with perforated polyester film was placed under the abdominal wall. Two silicone drains were then placed over the gauze and connected to a –20-cmH_2O suction source. The entire wound was then covered with a polyester drape.[11,12] A commercially available vacuum-assisted closure device, the 3M ABTHERA Advance (3M, Saint Paul, MN), is currently used. Reexploration for packing removal, hemostasis, biliary reconstruction if not already performed, and definitive closure was scheduled 48 to 72 h after LT. Early reoperation was performed when clinically indicated, such as when uncontrolled bleeding or signs of ACS appeared. During the OAM period, our goals were to first rapidly recover from shock, reduce vasopressor doses, and correct any coagulation abnormalities. We aimed to correct platelet counts to >50 000/μL and fibrinogen levels to >150 mg/dL. We actively used albumin preparations and blood transfusions as needed to prevent fluid overload. Additionally, we implemented fluid management with continuous venovenous hemofiltration in all cases. During periods of bleeding concern, the suction pressure of the 3M ABTHERA Advance device was set to 25 mm Hg. Once hemostasis was achieved, the pressure was gradually increased to 125 mm Hg.

Transplant Procedures and Perioperative Management

For patients who underwent LDLT, we selected a left lobe graft with the middle hepatic vein when the ratio of the graft volume to the recipient standard liver volume was >30%. A right lobe graft was the alternative for donation if the left lobe was not feasible. The ratio was calculated from the results of a volumetric study using computed tomography. In patients undergoing deceased-donor LT (DDLT), a piggyback technique was used for implantation. Arterial reconstruction was performed under a microscope in LDLT and under a surgical loupe in DDLT, with end-to-end anastomosis using interrupted sutures.[13] Duct-to-duct anastomosis was typically performed for biliary reconstruction. In cases in which staged biliary reconstruction was decided upon, a silicone drain was placed near the graft bile duct during the initial surgery. A biliary splint (2 mm, vinyl chloride tube) was placed beyond the anastomosis, and the splint was externalized through the upper edge of the duodenum with a Witzel-type fistula. The splint was removed approximately 3 mo after LDLT using a 2-step protocol.[14] The antimicrobial prophylaxis consisted of cefotaxime (4 g/d) and ampicillin (4 g/d). These drugs were injected 30 min before laparotomy and continued for up to 48 h after LDLT. During the period of OAM, we first continued administering these antibiotics and adjusted them to broad-spectrum antibiotics as necessary based on the cultured results. Additionally, micafungin was added as an empirical therapy. If there were no signs of fungal infection, the administration was discontinued after abdominal closure.

Immunosuppression Therapy

The standard immunosuppression regimen comprised tacrolimus and a steroid. The steroid was gradually tapered and discontinued by 3 mo after LT. The tacrolimus was started the day after LT by continuous intravenous injection and then changed to oral administration. The target trough level of tacrolimus was 10–15 ng/mL during the first month after LT. Mycophenolate mofetil was added for ABO-incompatible LDLT cases and patients whose trough levels of tacrolimus were intentionally kept lower because of renal dysfunction.[15] Even in cases of OAM, the timing of immunosuppressive drug initiation was not altered. However, we set the blood concentration of tacrolimus lower to 5–10 ng/mL, during OAM.

Definition of Early Allograft Dysfunction

Early allograft dysfunction (EAD) was defined by the presence of ≥1 of the following: (a) total bilirubin level of ≥10 mg/dL on postoperative day 7, (b) international normalized ratio (INR) of ≥1.6 on postoperative day 7, and (c) alanine aminotransferase or aspartate aminotransferase level of ≥2000 IU/mL within the first 7 postoperative days.[16]

Statistical Analysis

IBM SPSS Statistics 29 (IBM Corp, Armonk, NY) was used for the statistical analysis. The Mann-Whitney *U* test was used to analyze continuous data, and the Fisher test was used for categorical data. For all comparisons, the 2-sided significance level was set at a *P* value of <0.05.

RESULTS

Patient Characteristics

The preoperative characteristics of the 250 patients (139 men and 111 women) are summarized in Table 1. Of 250 patients, 22 (8.8%) underwent DDLT. The indications for LT were liver cirrhosis because of hepatitis C virus infection (n = 67; 26.8%), alcoholic liver cirrhosis (n = 40; 16.0%), nonalcoholic steatohepatitis (n = 36; 14.4%), liver cirrhosis because of hepatitis B virus infection (n = 28; 11.2%), primary biliary cirrhosis (n = 21; 8.4%), acute liver failure (n = 14; 5.6%), graft failure (n = 10; 4.0%), primary sclerosing cholangitis (n = 10; 4.0%), autoimmune hepatitis (n = 5; 2.0%), biliary atresia (n = 4; 1.6%), and other diseases (n = 15; 6.0%). The median (interquartile range [IQR]) recipient age was 58 (51–63) y, the Model for End-stage Liver Disease (MELD) score was 17 (12–24), and the Child-Pugh score was 10 (9–12). Twenty-nine patients (11.6%) were admitted to the ICU before LT to treat organ failure because of acute liver failure or acute-on-chronic liver failure. Thirty-three patients (13.2%) were undergoing dialysis, and 51 patients (20.4%) had a history of upper abdominal surgery.

OAM was performed in 16 patients (OAM group). The rationale for performing OAM was determined to be the necessity for damage control in all cases, and there were no cases where OAM was required owing to the large-for-size graft. DDLT consisted of 9 cases, with underlying diseases including hepatitis C–related cirrhosis with concomitant hemophilia in 3 cases, acute liver failure in 2 cases, graft failure after LDLT in 2 cases, acute-on-chronic liver failure in 1 case, and polycystic liver disease in 1 case. The median (IQR) MELD score was 34 (23–39). Five patients had a history of upper abdominal surgery, and all had extensive adhesions. The median cold ischemia time (CIT) was 567 min (522–585), and the amount of bleeding was 15 392 g (13 258–18 021). In contrast, among the 7 cases of LDLT, underlying diseases included hepatitis C–related cirrhosis in 3 cases, alcoholic cirrhosis in 2 cases, acute liver failure in 1 case, and nonalcoholic steatohepatitis in 1 case. The median MELD score was 18 (14–24). Three patients had a history of upper abdominal surgery, and extensive adhesions were also present. Although the median CIT was short at 106 min (88–169), the amount of bleeding was high at 26 100 g (15 781–26 965).

Compared with patients who did not require OAM as damage control (non-OAM group), the OAM group had higher MELD scores (24 versus 16, $P = 0.003$), a higher incidence of previous upper abdominal surgery (50% versus 18%, $P = 0.007$), more pretransplant ICU treatment (31% versus 10%, $P = 0.03$), more renal replacement therapy (38% versus 12%, $P = 0.01$), and more frequent DDLT (56% versus 6%, $P < 0.001$). The pretransplant platelet count, prothrombin time (PT)-INR, and fibrinogen levels were comparable between the groups. Donor age and body mass index were found to be higher in the OAM group ($P < 0.01$). No significant difference was observed in the percentage of graft steatosis between the non-OAM and OAM groups (Table 1). Of the 234 patients in the non-OAM group, 21 required early relaparotomy because of postoperative bleeding.

Intraoperative Factors

In the comparison of operative factors, the OAM group had more intraoperative blood loss (17.3 versus 6.1 L, $P < 0.001$), required more red blood cell (RBC) transfusions (46 versus 14 units, $P < 0.001$), fresh frozen plasma transfusions (54 versus 20 units, $P < 0.001$), and platelet component transfusions (40 versus 20 units, $P < 0.001$). The CIT in the OAM group was significantly longer because of the higher number of DDLTs ($P < 0.001$). Intraoperative portal vein thrombectomy was required in 6 cases (37.5%) in the OAM group and 25 cases (10.7%) in the non-OAM group, predominantly in the OAM group. In LDLT, there was no significant difference in the GRWR between the OAM and non-OAM groups (0.85 versus 0.76, $P = 0.26$). In DDLT, although there was a tendency for higher GRWR in the OAM group, there was no statistically significant difference (2.36 versus 1.83, $P = 0.06$).

TABLE 1.

Preoperative patient characteristics

Variable	OAM (N = 16)	Non-OAM (N = 234)	*P*
Recipient age	61 (54–64)	57 (51–63)	NS
Recipient sex, male	12 (75%)	127 (54%)	NS
Recipient BMI	23.0 (20.8–25.6)	23.8 (21.0–26.9)	NS
MELD score	24 (18–38)	16 (12–23)	<0.01
Prior upper abdominal surgery	8 (50%)	43 (18%)	<0.01
Resuscitation in ICU	5 (31%)	24 (10%)	0.03
Renal replacement therapy	6 (38%)	27 (12%)	0.01
Platelet count, $\times 10^4/\mu L$	6.4 (4.3–12.2)	5.6 (4.0–8.9)	NS
PT-INR	1.67 (1.40–1.89)	1.48 (1.29–1.73)	NS
APTT, s	50.5 (44.2–66.5)	43.1 (36.2–52.4)	0.01
Fibrinogen, mg/dL	171.5 (109.0–224.3)	144.0 (113.0–201.0)	NS
DDLT	9 (56%)	13 (6%)	<0.001
Donor age	51 (45–58)	39 (30–52)	<0.01
Donor sex, male	11 (69%)	126 (55%)	NS
Donor BMI	24.2 (22.9–25.6)	22.2 (20.3–24.1)	<0.01
Percentage of graft steatosis (%)	5 (4–20)	10 (5–19)	NS

Data are shown as n (%) or median (interquartile range).

APTT, activated partial thromboplastin time; BMI, body mass index; DDLT, deceased-donor liver transplantation; ICU, intensive care unit; MELD, Model for End-Stage Liver Disease; OAM, open abdomen management; PT-INR, prothrombin time-international normalized ratio.

At the end of the surgery, before abdominal closure or OAM, the platelet count was significantly lower in the OAM group (4.5 versus $8.0 \times 10^4/\mu L$, $P < 0.001$), whereas the PT-INR, fibrinogen level, body temperature, and lactate level did not differ between the groups. The OAM group also had progressive acidosis, and the noradrenaline doses were significantly higher in the OAM group (0.15 versus 0.02 µg/kg/min, $P < 0.001$; Table 2).

Resuscitation in ICU

Details of the 16 patients in whom damage control with OAM was performed are shown in Table 3. Upon deciding to finish the LT with OAM, we found coagulopathy in 81.3% of patients, acidosis in 37.5%, and hypothermia in 31.3%. In addition, a high dose of noradrenaline (>0.1 µg/kg/min) was required in 75.0% of patients. The method of OAM was SOC in 7 patients and NPT in 9; of the 9 NPT procedures, 6 were performed using a commercial device (3M ABTHERA Advance). The closure time was 34 (28–45) min for SOC and 14 (10–18) min for NPT, with NPT being significantly faster ($P < 0.001$). In the ICU, during the first 12 h after LT, 4 (2–5) units of RBC transfusion, 8 (8–8) units of fresh frozen plasma transfusion, and 10 (8–16) units of platelet transfusion were required. The volume of fluid from the abdominal drain was 1.5 (0.4–2.0) L. Of the 16 patients, 6 required an unscheduled reoperation (4 because of hemorrhage and 1 each because of ACS and decreased graft blood flow). ACS occurred in 1 patient who underwent SOC, and when the abdomen was opened, the patient's circulation immediately improved. At 12 h after resuscitation, the rate of coagulopathy decreased to 25.0%, acidosis decreased to 6.3%, hypothermia resolved, and high-dose noradrenaline requirement decreased to 18.8%. In the 4 cases of LDLT, staged biliary reconstruction was performed using duct-to-duct anastomosis. The abdominal wall was closed in the second operation in 12 patients, in the third operation in 3 patients, and in the fourth operation in 1 patient (Table 3).

Patient Outcomes

The morbidity and mortality rates are shown in Table 4. The duration of hospitalization did not differ between the 2 groups. The rate of EAD was significantly higher in the OAM group (68.8% versus 29.5%, $P = 0.003$). The rates of vascular, biliary, and intra-abdominal infectious complications were comparable between the groups. Five patients in the OAM group died within 1 y. The cause of death was graft failure in 2 patients and sepsis in 3 patients. The 90-d mortality rate was higher in the OAM than the non-OAM group (25.0% versus 8.1%, $P = 0.05$).

DISCUSSION

In the present study, we have reported our experience with 16 cases of OAM as a damage control strategy in LT. The OAM group had higher MELD scores, with many patients requiring treatment in the ICU and dialysis. Furthermore, the need for additional adhesion dissection during surgery because of previous abdominal surgeries likely contributed to increased intraoperative bleeding and transfusion requirements. In particular, among the DDLT cases, 9 out of 22 required OAM. The high rate of OAM in DDLT may be because of coagulation disorders and the need for vasopressors caused by postreperfusion syndrome associated with prolonged ischemic times. In the DDLT group, the MELD scores of the 9 OAM cases were higher compared with the 13 non-OAM cases (34 [23–39] versus 18 [14–24]). In addition, the 9 OAM cases included 3 cases of hemophilia, 2 cases of retransplant, 2 cases of acute-on-chronic liver failure grade 3,[17] and 1 case of multiple cystic liver disease with a history of liver resection. These

TABLE 2.

Intraoperative factors

Factors	OAM (N = 16)	Non-OAM (N = 234)	*P*
Operative time, min	747 (700–881)	775 (689–863)	NS
Cold ischemia, min	453 (120–572)	90 (66–119)	<0.001
Warm ischemia, min	44 (35–53)	40 (35–47)	NS
Intraoperative portal vein thrombectomy	6 (37.5%)	25 (10.7%)	0.01
GRWR, LDLT	0.85 (0.78–0.88)	0.76 (0.62–0.95)	NS
GRWR, DDLT	2.36 (2.24–3.12)	1.83 (1.41–2.30)	NS
Blood loss, g	17 261 (12 540–27 385)	6118 (3688–9566)	<0.001
RBC transfusion, units	46 (26–69)	14 (6–22)	<0.001
FFP transfusion, units	54 (41–86)	20 (9–32)	<0.001
PC transfusion, units	40 (30–50)	20 (10–30)	<0.001
Parameters before closure	4.5 (2.8–6.1)	8.0 (7.0–9.7)	
Platelet count, $10^4/\mu L$	4.5 (2.8–6.1)	8.0 (7.0–9.7)	<0.001
PT-INR	1.52 (1.30–1.66)	1.36 (1.29–1.48)	NS
APTT, s	57.0 (44.7–79.9)	68.2 (56.1–82.3)	NS
Fibrinogen, mg/dL	141.0 (122.5–163.5)	150.0 (139.0–166.0)	NS
Temperature, °C	36.2 (34.8–36.6)	36.8 (36.2–37.2)	NS
pH	7.32 (7.24–7.40)	7.39 (7.37–7.42)	0.01
Lactate, mmol/L	3.1 (1.9–5.6)	2.3 (1.7–3.2)	NS
Dose of noradrenalin, µg/kg/min	0.15 (0.07–0.20)	0.02 (0–0.05)	<0.001

Data are shown as n (%) or median (interquartile range).
APTT, activated partial thromboplastin time; DDLT, deceased donor liver transplantation; FFP, fresh frozen plasma; GRWR, graft-to-recipient weight ratio; LDLT, living donor liver transplantation; OAM, open abdomen management; PC, platelet component; PT-INR, prothrombin time-international normalized ratio; RBC, red blood cell.

TABLE 3.

Details of damage control with OAM cases

Variable	Case 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Age, y	52	65	41	60	56	65	61	65	64	61	63	47	59	64	53	60
Female/male	F	F	M	M	M	M	M	M	M	M	M	F	M	M	F	M
LDLT/DDLT	LDLT	LDLT	DDLT	DDLT	DDLT	LDLT	DDLT	LDLT	LDLT	DDLT	DDLT	LDLT	DDLT	LDLT	DDLT	DDLT
OAM method: (1) skin-only closure, (2) Barker's technique, (3) ABTHRA	1	1	1	1	1	1	1	2	2	2	3	3	3	3	3	3
Time required for TAC procedure, min	58	35	32	24	54	24	34	18	24	16	14	14	8	10	7	19
Intraoperative transfusion																
RBC transfusion, U	32	72	42	54	70	64	26	56	38	76	50	20	26	38	24	22
FFP transfusion, U	25	80	24	80	88	120	60	40	72	100	44	48	44	72	40	48
PC transfusion, U	20	40	40	60	50	40	30	40	50	70	30	30	40	50	10	30
At the time of DC decision																
Platelet count <5 × 10⁴/μL	−	+	−	+	−	+	+	+	+	+	+	−	+	+	−	−
PT-INR >1.5	+	−	+	+	−	−	−	−	+	+	+	−	+	+	−	+
Fibrinogen <150 mg/dL	+	+	+	+	−	−	−	+	+	+	+	−	+	+	−	+
pH <7.3	+	+	−	−	−	+	−	−	+	−	−	−	+	+	−	−
BT <35 °C	−	−	−	−	−	+	+	−	−	−	+	−	+	−	+	−
Dose pf noradrenalin >0.1γ	−	+	+	+	+	+	−	+	+	+	−	+	+	+	−	+
During 12 h of resuscitation in ICU																
RBC transfusion, U	8	4	2	2	8	0	6	4	4	2	2	0	4	6	2	4
FFP transfusion, U	6	20	8	8	12	8	8	8	8	4	8	8	8	8	0	8
PC transfusion, U	10	30	10	10	10	20	0	10	15	0	20	0	20	0	10	10
Amount of drain fluid, L	2.2	2.3	0.2	0.4	2.0	0	0.1	1.5	2.2	0.2	1.1	0.3	1.7	1.9	0.8	1.9
After 12 h of resuscitation in ICU																
Platelet count <5 × 10⁴/μL	−	+	−	−	−	−	−	−	+	−	+	−	−	−	−	−
PT-INR >1.5	+	−	−	−	−	−	−	−	+	−	+	−	−	−	+	−
Fibrinogen <150 mg/dL	+	−	−	−	−	−	−	−	+	−	+	−	−	−	+	−
pH <7.3	−	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−
BT <35 °C	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Dose pf noradrenalin >0.1γ	−	+	−	−	−	+	−	+	−	−	−	−	−	−	−	−
Unplanned relaparotomy (1) hemorrhage, (2) ACS, (3) decreased graft blood flow	1	1	−	−	1	2	−	−	−	−	−	−	−	−	3	1
Staged biliary reconstruction	−	+	−	−	−	−	−	−	+	−	−	+	−	+	−	−
No. of surgeries until definitive closure	1	2	1	1	1	1	1	1	1	1	1	2	1	1	2	3

ACS, abdominal compartment syndrome; BT, body temperature; DDLT, deceased-donor liver transplantation; F, female; FFP, fresh frozen plasma; ICU, intensive care unit; LDLT, living-donor liver transplantation; M, male; OAM, open abdomen management; PC, platelet component; PT-INR, prothrombin time-international normalized ratio; RBC, red blood cell; TAC, tacrolimus.

TABLE 4.

Patient outcome

	OAM (N = 16)	Non-OAM (N = 234)	P
Hospital stay, mo	1.4 (1.2–2.0)	1.7 (1.2–2.4)	NS
Early allograft dysfunction	11 (68.8%)	69 (29.5%)	0.003
Portal vein complication	2 (12.5%)	30 (12.8%)	NS
Arterial complication	1 (6.3%)	3 (1.3%)	NS
Biliary complication	2 (12.5%)	39 (16.7%)	NS
Intra-abdominal infection	1 (6.3%)	27 (11.5%)	NS
30-d mortality	1 (6.3%)	8 (3.4%)	NS
90-d mortality	4 (25.0%)	19 (8.1%)	0.05
1-y mortality	5 (31.2%)	40 (17%)	NS

OAM, open abdomen management.

conditions likely contributed to increased blood loss and the need for large-volume transfusions. Of course, not all patients with such backgrounds will require OAM. However, considering the higher rates of EAD and 90-d mortality in the OAM group, we believe that it is important to share risks with the team and ICU staff. Moreover, in cases where there is concern about the development of ACS because of intestinal edema associated with portal hypertension or increased intra-abdominal pressure because of liver packing, OAM becomes a realistic option. We believe that OAM can reduce the decreased organ blood flow and excessive tension on the anastomosis associated with increased intra-abdominal pressure. Although SOC was performed at the time of OAM introduction, the technique was changed to Barker's method after experiencing ACS and requiring an unscheduled relaparotomy. After switching to 3M ABTHERA Advance, the technique became simpler and drain complications were no longer experienced.

Regarding the indications for damage control, DiNorcia et al[9] stated that damage control surgery should be considered in patients with recurrent hypothermia (body temperature of <35 °C), acidosis (pH of <7.25), need for 20 to 25 units of RBC transfusion, and ongoing surface bleeding. We assessed coagulopathy based on the degree of hemostasis difficulty in the operative field and determined the indication for damage control in each individual case, along with the need for acidosis correction using sodium bicarbonate or trometamol, the transfusion volume, and any vasoactive drug requirements. Before closure, the OAM group had a lower platelet count and pH and higher noradrenaline doses than the non-OAM group, but the PT, activated partial thromboplastin time, and fibrinogen level were the same between the 2 groups. It was difficult to set a numerical cutoff for coagulopathy, which seemed to depend largely on the surgeon's impression during the procedure.

As in previous reports,[7-9] we found no differences in infection or vascular complications between the OAM and non-OAM groups in the present study. OAM requires multiple surgeries and continuous negative pressure; thus, intra-abdominal abscesses, difficulty in abdominal wall closure, and enteroatmospheric fistulas have been reported as associated complications. Among them, enteroatmospheric fistulas have been reported to be associated with increased mortality, prolonged hospitalization, and increased costs. The frequency of such fistulas has been reported to range from 5.7% to 17.2% in nontrauma cases and has been associated with the duration of OAM.[1] Since 2011, our institution has performed OAM in 80 trauma and nontrauma cases. In the trauma cases, there were 2 instances where closure was difficult because of abdominal wall damage, ultimately resulting in the complication of an enterocutaneous fistula. Additionally, in cases of severe abdominal sepsis, we experienced difficulties in the source control of contamination and in improving bowel edema, necessitating prolonged OAM. In contrast, in the LT cases analyzed in this study, there was no intra-abdominal contamination owing to the semi-clean nature of the surgery. Hemostasis through packing and correction of coagulopathy via transfusion allowed for early closure once negative fluid balance was achieved through continuous venovenous hemofiltration. As a result, the fascia closure rate after OAM was 100%. In the majority of cases, the fascia could be closed in the second operation, and even when closure was difficult in the second operation, we were able to minimize abdominal wall retraction and reduce the risk of complications by suturing a portion of the fascia and reducing the size of the abdominal opening.

Although our study faces several constraints, including its retrospective nature, the small sample size, potential timeframe bias, and the varied methods applied for OAM across the study duration, the results demonstrate that by determining damage control with OAM in the face of challenging physiologic abnormalities, LT can be possibly completed without increased complications after resuscitation in the ICU.

In conclusion, OAM as a damage control method can be crucial in complex LT cases complicated by hypothermia, acidosis, coagulopathy, and hemodynamic instability. Damage control management minimizes deterioration of the patient's condition during surgery, allowing completion of the planned procedure after stabilizing the recipient's overall condition in the ICU.

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REFERENCES

1. Coccolini F, Montori G, Ceresoli M, et al. The role of open abdomen in non-trauma patient: WSES consensus paper. *World J Emerg Surg*. 2017;12:39.
2. Weber DG, Bendinelli C, Balogh ZJ. Damage control surgery for abdominal emergencies. *Br J Surg*. 2014;101:e109–e118.
3. Khan A, Hsee L, Mathur S, et al. Damage-control laparotomy in non-trauma patients: review of indications and outcomes. *J Trauma Acute Care Surg*. 2013;75:365–368.
4. Malbrain MLNG, Cheatham ML, Kirkpatrick A, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. I. Definitions. *Intensive Care Med*. 2006;32:1722–1732.
5. Cheatham ML, Malbrain MLNG, Kirkpatrick A, et al. Results from the international conference of experts on intra-abdominal hypertension and abdominal compartment syndrome. II. Recommendations. *Intensive Care Med*. 2007;33:951–962.
6. Assirati G, Serra V, Tarantino G, et al. Vacuum-assisted closure therapy in patients undergoing liver transplantation with necessity to maintain open abdomen. *Transplant Proc*. 2016;48:383–385.
7. Chan T, Bleszynski MS, Youssef DS, et al. Open abdomen in liver transplantation. *Am J Surg*. 2018;215:782–785.

8. Hobeika C, Allard MA, Bucur PO, et al. Management of the open abdomen after liver transplantation. *World J Surg*. 2017;41:3199–3204.
9. DiNorcia J, Lee MK, Harlander-Locke MP, et al. Damage control as a strategy to manage postreperfusion hemodynamic instability and coagulopathy in liver transplant. *JAMA Surg*. 2015;150:1066–1072.
10. Komorowski AL, Li WF, Millan CA, et al. Temporary abdominal closure and delayed biliary reconstruction due to massive bleeding in patients undergoing liver transplantation: an old trick in a new indication. *J Hepatobiliary Pancreat Sci*. 2016;23:118–124.
11. Barker DE, Kaufman HJ, Smith LA, et al. Vacuum pack technique of temporary abdominal closure: a 7-year experience with 112 patients. *J Trauma*. 2000;48:201–206.
12. Montori G, Allievi N, Coccolini F, et al. Negative pressure wound therapy versus modified barker vacuum pack as temporary abdominal closure technique for open abdomen management: a four-year experience. *BMC Surg*. 2017;17:86.
13. Takatsuki M, Chiang YC, Lin TS, et al. Anatomical and technical aspects of hepatic artery reconstruction in living donor liver transplantation. *Surgery*. 2006;140:824–828.
14. Eguchi S, Takatsuki M, Hidaka M, et al. Two-step biliary external stent removal after living donor liver transplantation. *Transpl Int*. 2008;21:531–533.
15. Takatsuki M, Soyama A, Muraoka I, et al. Post-operative complications requiring hospitalization more than one yr after living donor liver transplantation. *Clin Transplant*. 2014;28:105–110.
16. Olthoff KM, Kulik L, Samstein B, et al. Validation of a current definition of early allograft dysfunction in liver transplant recipients and analysis of risk factors. *Liver Transpl*. 2010;16:943–949.
17. Moreau R, Jalan R, Gines P, et al; CANONIC Study Investigators of the EASL–CLIF Consortium. Acute-on-chronic liver failure is a distinct syndrome that develops in patients with acute decompensation of cirrhosis. *Gastroenterology*. 2013;144:1426–37, 1437.e1.