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Brain death organ donor supported by a left ventricular assist device showing unexpected congestive liver fibrosis: A case report

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ABSTRACT

INSTRUCTION: Organ transplantation from a brain death donor on mechanical circulatory support is rare. We report a case in which a brain death donor, supported by a left ventricular assist device (LVAD), unexpectedly displayed significant congestive fibrosis of the liver.
PRESENTATION OF CASE: The potential organ donor was diagnosed 23 years previously as having dilated-phase of hypertrophic cardiomyopathy. He had undergone implantation of an LVAD as a bridge to heart transplantation. Laboratory tests and imaging studies performed during the follow-up for his cardiac disease and donor evaluation confirmed that he was suitable for donation of liver. During organ procurement, special attention was paid to preserving LVAD and its device's drive lines and the exposure of the surgical fields was restricted by those devices. Thoracotomy and laparotomy were performed, and the aorta and inferior vena cava were encircled successfully. The gross appearance of liver, however, suggested significant fibrosis. Therefore, the decision was made not to use this liver. Subsequent trichrome-stained permanent sections revealed advanced fibrosis (stage F3–4).
DISCUSSION: As previously reported, organ procurement from donors with LVAD was thought to be demanding procedure because of the limited exposure of surgical field. In addition, it would be difficult to predict severe liver fibrosis in patients with an LVAD without a pathological examination.
CONCLUSION: Donors with mechanical circulatory support systems can be candidate to expand the donor pool, but technical difficulty should be expected owing limited exposure during the donor operation. For liver transplantation, subclinical advanced liver fibrosis should be noted.

1. Introduction

Organ shortage remains one of the major limitations in the field of liver transplantation (LT). Hence, an effort has been made to increase the existing brain death donor pool by including liver allografts from extended-criteria donors. There is still reluctance, however, to accept grafts from donors on mechanical circulatory support systems because of their continued need for anticoagulation, the potential for embolic disease, and the possibility of organ injury due to cardiogenic shock [1,2] and LT cases with donors on ventricular assist devices remain rare [3–5]. We report herein the case of a brain death donor supported by a left ventricular assist device (LVAD) whose liver was unexpectedly found to be affected by significant congestive fibrosis. This work has been reported in line with the SCARE criteria [6].

2. Presentation of case

The potential donor was a brain-dead man in his forties who had been evaluated and deemed eligible for organ donation that included his lungs, liver, pancreas, and kidneys. He was 173 cm in height and weighed 56.3 kg. He had been medically diagnosed 23 years previously as having dilated-phase of hypertrophic cardiomyopathy. Since then, he had been followed by a cardiologist and his cardiac function was worsening. In 2015, he had undergone implantation of an LVAD as a bridge to heart transplantation. However, he developed a motor function deficit affecting his right upper extremity 8 months after the LVAD was implanted. During the

Abbreviations: ALT, alanine transaminase; AST, aspartate transaminase; CT, computed tomography; LT, liver transplantation; LVAD, left ventricular assist device.

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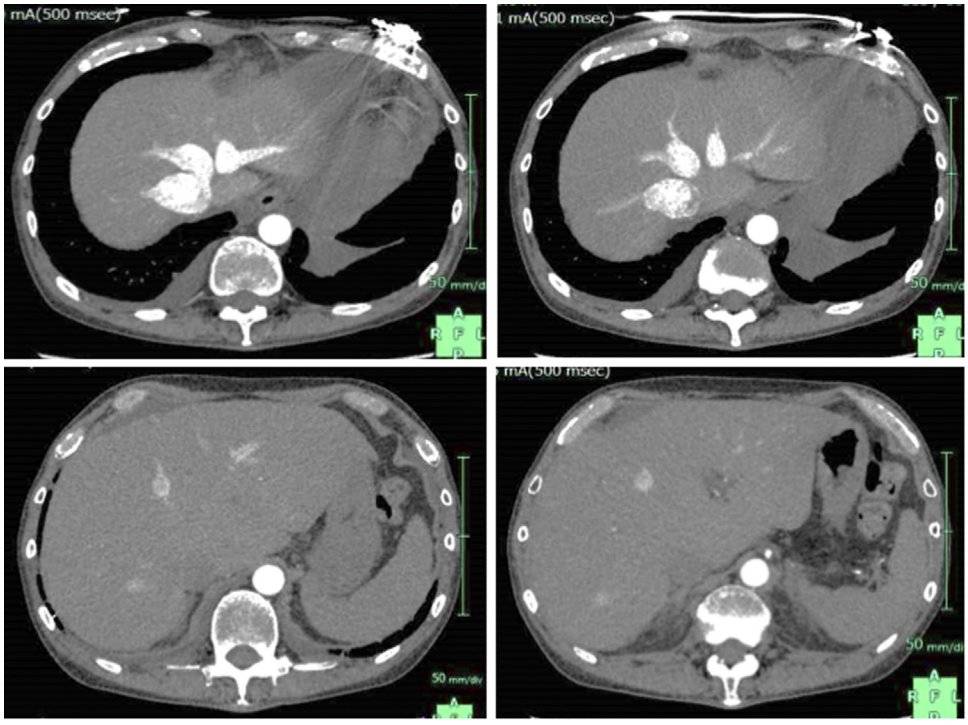

Fig. 1. Abdominal CT scan at 8 months prior to the donor surgery. There was no significant liver abnormality except for early enhancement of dilated hepatic veins.

evaluation of this episode, intracranial hemorrhage was demonstrated on computed tomography (CT). His neurological condition continued to deteriorate, and he was eventually declared brain dead.

Other than the cardiac disease, he had no medical history and no family history of liver disease. During the follow-up for his cardiac disease, laboratory tests showed that his bilirubin had occasionally been slightly elevated but basically remained under 2.0 mg/dl. The elevated bilirubin was thought to be due to constitutional jaundice. Other liver function tests, including Aspartate transaminase (AST), Alanine transaminase (ALT), albumin, and coagulation tests, were consistently normal. Abdominal CT scan at 8 months prior to the donor surgery showed no significant liver abnormality except for early enhancement of dilated hepatic veins (Fig. 1). In abdominal CT scan at 2 months prior to the donor surgery, there was no obvious findings of liver fibrosis, although liver appeared to be decreased in size (Fig. 2). The results of laboratory tests related to liver function performed just before procurement are shown in Table 1. His AST and ALT levels were within their normal ranges. Although his bilirubin level was elevated at the time of donor surgery, it remained under 2.0 mg/dl until 2 days prior to donor surgery. An enlarged and pulsatile liver was not seen in this donor.

Table 1
Laboratory data of the donor before operation.

Variable	Normal range	
Red-cell count (per mm^3)	4,000,000–5,500,000	3,690,000
White-cell count (per mm^3)	3800–8500	12500
Hemoglobin (g/dl)	13–18	11.7
Hematocrit (%)	37–52	33.3
Platelet count (per mm^3)	160,000–410,000	105,000
AST (U/l)	13–33	31
ALT (U/l)	6–30	21
Total bilirubin (mg/dl)	0.3–1.2	4.5
Direct bilirubin (mg/dl)	0–0.2	0.9
Total protein (g/dl)	6.7–8.3	6.5

AST, Aspartate transaminase; ALT, Alanine transaminase.

Donor surgery was performed without stopping the LVAD. A midline incision was made from the jugular notch to the symphysis pubis as usual fashion. However, the left upper quadrant was occupied by the LVAD, and the device and drive lines were severely encapsulated by dense fibrous tissue. Because special attention was paid to preserving the LVAD and the drive lines, initial abdominal exposure was limited. During this thoracotomy and laparotomy process, the cardiovascular surgeon who implanted this device attended the surgery to support transplant surgeons. Thoracotomy and laparotomy were then successfully carried out without damaging the LVAD. A moderate amount of serous, yellow ascites was observed.

The appearance of the liver was slightly reduced in size and multiple macronodular lesions were detected (Fig. 3). Moreover, significant fibrosis was suspected because the liver edge felt stiff on palpation. The pathologist at the donor hospital evaluated the subcapsular wedge frozen biopsy, and diagnosed mild microvesicular steatosis (<10%) with lymphoid filtration. Significant fibrosis was not pointed out from the frozen section. Judging from all information we obtained during operation, we decided, not to transplant this liver because of the fibrotic conditions. Other organs such as bilateral lungs, pancreas, and bilateral kidneys were successfully procured and transplanted.

When the procurement team returned to our hospital, additional histological evaluation on subcapsular wedge biopsy was made to confirm the liver condition. A trichrome-stained permanent section of the liver demonstrated advanced fibrosis (stage F3–4) as shown in Fig. 4, which supported the intraoperative liver appearance and our decision.

3. Discussion

Thoracic and abdominal organ transplantation from donors supported by various ventricular assist devices is still rare [3–5]. Because the donor in the current case was a heart-beating brain death donor, specific attention was given not to damage the LVAD system during donor surgery. Although we ultimately did not pro-

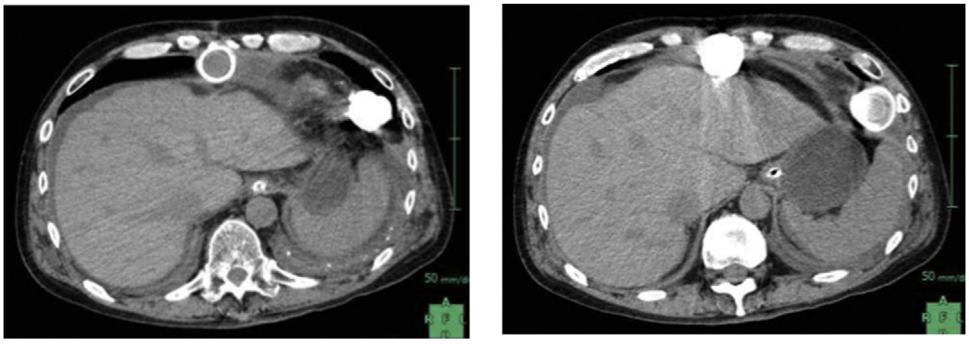

Fig. 2. Abdominal CT scan at 2 months prior to the donor surgery. There was no obvious findings of liver cirrhosis, although liver appeared to be decreased in size compared to previous CT.

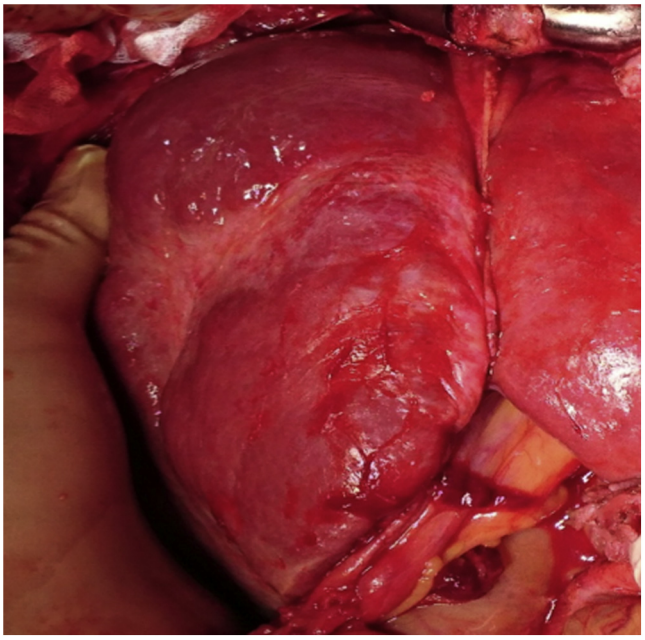

Fig. 3. Intraoperative findings of liver. The liver was decreased in size with multiple macronodular lesions. Liver edge was irregular and rounded.

cure the liver graft in the current case, difficult procedures were anticipated because of the limited exposure of surgical field due to the drive lines across and above the liver. In a case report, Misra et al. noted that effort was necessary to minimize the amount of dissection performed prior to cross-clamping because of the presence of the drive line and full anticoagulation of the donor [3].

The histopathology of cardiac hepaotopathy was initially described in liver necropsy specimens in 1951. Since then, it has been reported that chronic right heart failure predisposed to hepatic passive congestion and centrizonal necrosis, which could lead to hepatic fibrosis [7–9]. In a series of 59 patients awaiting cardiac transplantation or LVAD placement, congestive changes were seen in all patients, with 19% having histological changes consistent with cirrhosis [10]. Although hepatomegaly is the most common manifestation of acute or chronic heart failure [11], loss of the pulsatile liver in patient with chronic cardiac disease is more concerning as it implies progression to cardiac fibrosis or cirrhosis, as was seen in our case [12]. Although ascites was noticed prior to donor surgery in this case, ascites is not specific to liver cirrhosis, and sometimes it is also seen with right-sided heart failure or congestive hepatopathy without cirrhosis. Also, the stage of congestive heart failure does not seem to be correlated with hepatic fibrosis or cirrhosis [9]. Dai et al. reported that the patients' characteristics or liver tests (i.e. serum AST, ALT, ALP, and total bilirubin) were not associated with the severity of hepatic fibrosis [7]. They also reported that it was difficult to distinguish liver cirrhosis by imaging studies such as ultrasonography or CT [7]. Therefore, it would be difficult to predict severe liver fibrosis in patients with an LVAD without a pathological examination.

Histologically, the portal tracts are relatively spared from fibrosis, which is a distinguishing factors of cardiac cirrhosis compared with other etiologies of cirrhosis, and its presence likely contributes to the lack of symptoms associated with portal hypertension [13]. Histological changes in the setting of heart failure show a unique

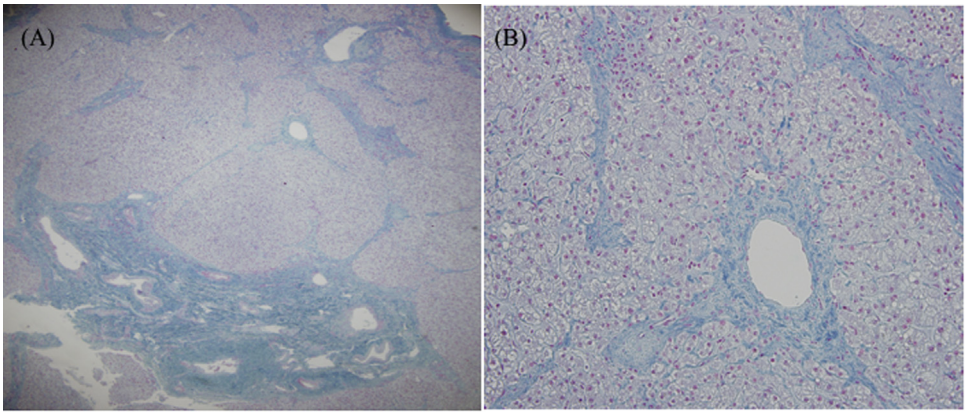

Fig. 4. Histological findings of liver biopsy. (A) low-magnification view and (B) high-magnification view of trichrome stain showing periportal fibrotic expansion with marked portal to portal and portal to central bridging. Lymphocyte were scattered within this collagenous tissues.

pattern of progression. Inflammation is minimal to absent and pericellular fibrosis occurs first, mostly of central veins, followed by central-central or central-portal bridging. Regenerative nodules are observed after the development of bridging fibrosis [8].

4. Conclusion

Donors with mechanical circulatory support systems could be an option for expanding the donor candidate pool, although organ procurement can be more difficult than usual because of the limited exposure of surgical field. Specific attention must be paid to preserving the drive lines during thoracotomy and laparotomy, taking care not to damage the devices. It also should be noted that chronic heart failure can lead to subclinical advanced liver fibrosis. Advanced congestive fibrosis of a potential liver graft may be difficult to diagnose before donor surgery. Therefore, the decision should be based on the gross appearance and pathological examination of the liver during the donor surgery.

Conflicts of interest

We have no conflicts of interest.

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Ethical approval

Ethical approval has been exempted by our institution.

Consent

We don't have signed consent from a deceased patient or family. The head of our medical team take responsibility that exhaustive attempts have been made to contact the family and that the paper has been sufficiently anonymised not to cause harm to the patient or his family.

Author contribution

HK participated in surgical procedure and draft the report. YO, MK, NK, SO, TH, MT contributed surgical procedure. SE and YO supervised this report. All authors read and approved the final manuscript.

Registration of research studies

I registered my research at Research Registry. Research Registry UIN is researchregistry3656.

Guarantor

The guarantor of this manuscript is Hideya Kamei, corresponding author.

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