LETTER TO THE EDITOR

60 h of anhepatic state without neurologic deficit

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Two-stage liver transplantation (LT), i.e. emergent total hepatectomy with prolonged anhepatic state and subsequent LT, has been described as a means to stabilize patients with fulminant hepatic failure (FHF), primary nonfunction after LT, or massive hepatic trauma [1]. After total hepatectomy, patient survival only depends on the future availability of a liver graft. The pathophysiological consequences of prolonged anhepatic state are not fully known, as it is not known how long a patient may be anhepatic before it is too late for hope of survival. As FHF may lead to brain oedema, the cerebral outcome of patients submitted prolonged anhepatic state seems particularly critical.

A 34-year-old woman was suffering from end-stage liver cirrhosis with refractory ascites and portal hypertension from unknown origin. She underwent living-related LT with a left liver graft (segments II–IV). On postoperative day 1, INR rose from 1.7 to 5.3, and there was no arterial or portal flow within the graft at Doppler ultrasonography. At reintervention the graft was tense and ischemic, and both hepatic artery and portal vein were occluded. The necrotic graft was removed and the patient was registered for urgent cadaveric reLT. During the anhepatic state body temperature was maintained between 34°C and 37°C with warming blanket. Blood glucose level was controlled by intravenous glucose 20% infusion. The patient became anuric within 24 h and was supported with continuous veno-venous hemofiltration. Pulmonary and cardiac functions remained stable. Anaesthesia was maintained by continuous infusion of propofol and remifentanyl. The patient finally received a cadaveric liver that was reperfused 60 h after the first graft removal. The patient slowly woke up after anaesthesia interruption. She was extubated 7 days after reLT without neurologic sequel. She was fully conscious and had no motor deficits. However, she developed *Aspergillus fumigatus* infection and finally died from sepsis and progressive multiorgan failure.

The full awakening of this patient after 60 h of anhepatic state, and other cases published in the literature [1–3, demonstrate that survival without neurological sequel is possible after >2 days of anhepatic state. The reason for the absence of neurological complications in these anhepatic patients is unclear. It may be possible that anhepatic patients do not develop life-threatening neurologic complications, or might be at least less prone to develop brain oedema than patients suffering from hyperacute FHF who may develop brain oedema within 24 h. It was demonstrated that LT anhepatic phase is linked to lowering of intracranial pressure in FHF patients (4). In an animal model, the anhepatic pigs did not develop cranial hypertension, at the contrary of the ischemic FHF group (5). These observations may be interpreted as an argument in favour of the 'toxic liver hypothesis' that suggests that the presence of the diseased liver may be important to the genesis of brain oedema and elevation of intracranial pressure during FHF. The possibility and the reasons of absence of brain oedema in prolonged anhepatic state largely deserve further human observations and animal experiments.

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References

1. Ringe B, Lubbe N, Kuse E, Frei U, Pichlmayr R. Total hepatectomy and liver transplantation as two-stage procedure. *Ann Surg* 1993; **218**: 3.
2. Kim HB, Maller E, Redd D, *et al.* Orthotopic liver transplantation for inflammatory myofibroblastic tumor of the liver hilum. *J Pediatr Surg* 1996; **31**: 840.
3. Hammer GB, So SK, Al-Uzri A, *et al.* Continuous venovenous hemofiltration with dialysis in combination with total hepatectomy and portocaval shunting. *Transplantation* 1996; **62**: 130.
4. Detry O, Arkadopoulos N, Ting P, *et al.* Intracranial pressure during liver transplantation for fulminant hepatic failure. *Transplantation* 1999; **67**: 767.
5. Arkadopoulos N, Chen SC, Khalili TM, *et al.* Transplantation of hepatocytes for prevention of intracranial hypertension in pigs with ischemic liver failure. *Cell Transplant* 1998; **7**: 357.

