Renal Replacement Therapy for Patients with Acute Liver Failure Awaiting Orthotopic Hepatic Transplantation

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Dear Sir,

As hepatic transplantation becomes more accepted, the number of centres performing such major operations is likely to increase. Clinical teams will experience a learning curve, not only hepatologists and hepatic transplant surgeons, but also supporting nephrologists. Currently patients with fulminant hepatic failure are referred to specialist centres for further management, including the assessment of the need for emergency orthotopic hepatic transplantation. In time decisions regarding further management are likely to be made locally. This group of patients typically develop acute renal failure either by the direct effect of toxic substances, such as [A](#)-benzoquinomine after paracetamol (acetaminophen) self-poisoning [1], or in the clinical setting of multiple organ failure, sepsis and/or following the administration of nephrotoxic drugs. They present the nephrologist with different problems than those encountered in patients undergoing hepatic transplantation for end-stage liver disease who require temporary renal support [2].

Although patients with fulminant hepatic failure die with multiple organ failure, the most common mode of death remains cerebral oedema [3]. As the mortality of this group of patients has remained high despite advances in intensive care support, it is relevant to attempt the prediction of patient survival soon after admission [4], so that an urgent appeal for a donor can be made before the general condition of the patient deteriorates making any attempt at transplantation impossible. Hepatic transplantation is technically easier in patients with fulminant hepatic failure than those with severe portal venous hypertension [5]. However, the intracranial pressure (ICP) has been shown to increase intraoperatively following hepatic reperfusion [6], and in some preliminary reports up to a third of such patients have required active treatment for increased ICP during surgery [6–8]. Persistently increased ICP may be a major problem for up to 24 h following successful orthotopic hepatic transplantation. The increased ICP and cerebral oedema observed in these patients probably reflects an increase in total cerebral blood flow associated with major intracranial shunting of blood, and results in local neuronal hypoxia. The cerebral pattern mirrors the systemic circulation which shows an increased cardiac output, a markedly reduced peripheral vascular resistance [9], and tissues exhibiting an oxygen uptake that is supply dependent.

Under these circumstances treatment of renal failure with conventional machine haemodialysis [10] or haemo-filtration [11] may provoke an increase in ICP. Since these treatments also tend to produce some degree of cardiac instability, then the net effect can be both a reduction in total cerebral blood flow/oxygen delivery and also a reduction in the cerebral perfusion pressure, so resulting in death or neurological morbidity [12]. These patients therefore need to be treated by

renal replacement systems designed to minimise changes in both ICP and haemodynamic status [13]. In addition, membrane bio-incompatibility is likely to be a factor in reducing haemodynamic and intracranial stability [14]. Other factors must also be considered, such as the nature of the extracorporeal anticoagulant and the anionic base of the substitution/dialysis fluids [15].

Patients presenting with fulminant hepatic failure often show a severe hypophosphataemia despite impaired renal function [16], and the use of continuous renal replacement systems may aggravate, or result in further, electrolyte imbalances [17].

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The presence of a liver transplant unit is likely to increase the work load of supporting nephrologists but, with meticulous attention to detail, the patient can usually be supported whilst a donor liver is found, with subsequent transfer to the transplant surgeon. However, we have noted that, in the interval of discontinuing a continuous haemofiltration/dialysis circuit and movement to an adjacent operating theatre suite, a previously controlled ICP may increase to values requiring prompt action. Control of increased ICP in the anuric patient in this situation may be very difficult and the majority of such cases have either died from increased ICP on the operating table or within the first 12 h postoperatively. This experience suggests that it may be of value to maintain continuous forms of renal support peroperatively [18], emphasising the role of the indispensable nephrologists in acute as well as chronic situations of liver failure and transplantation.

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