

Editorial

Coagulation monitoring and management during liver transplantation

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In this issue, Adachi [1] describes the anesthetic management of the liver transplant recipient from a living-related donor. Transplantation is indicated when the liver disease progresses to major functional impairment. Advanced hepatic failure produces complex physiological and biochemical effects; the surgical procedure is lengthy and often causes many problems. The management of such patient thus presents a redoubtable challenge for the anesthesiologist.

One of the most knotty problems that occurs during liver transplantation is hemostatic disturbance. Coagulopathy is often present before transplant and is characterized by decreases in coagulation factors, qualitative and quantitative platelet defects, and fibrinolysis. Massive surgical bleeding commonly leads to dilutional coagulopathy in addition to pre-existing problems. During the anhepatic stage, coagulation factors are further depleted, and clearance of activated factors and inhibitors is impaired. With reperfusion, coagulation is further exaggerated as a result of tissue plasminogen activator and heparin released from the donor liver, hypothermia, and additional dilutional effects [2–4].

To assess these abrupt changes of hemostatic status, the value of continual coagulation monitoring is widely accepted. Routine laboratory screening tests provide essential information, but results often are supplied too late to accurately reflect a changing situation. The problem of diagnostic delay may be overcome by the development of rapid bedside techniques for determining prothrombin time (PT), activated partial thromboplastin time (aPTT), and platelet count. Thromboelastography (TEG) [2–4] and use of the Sonoclot (Sienco, Wheat Ridge, CO, USA) [5,6], sensitive bedside techniques quantifying the rate and quality

of fibrin formation by measuring viscoelastic properties, provide excellent qualitative data. Although the maintenance of blood coagulability using TEG has been shown to reduce blood and fluid infusion volume [2], other monitors have not yet been fully evaluated.

To maintain sufficient hemostasis during the dissection and anhepatic phases, transfusion of fresh frozen plasma (FFP) is generally required to replace clotting factors that are lost during the surgical procedure. However, during the neohepatic stage, over-transfusion should be avoided because the use of FFP may lead to hepatic artery thrombosis (HAT) [7,8]. HAT is the second leading cause of graft failure in the immediate postoperative period, and the rates of HAT are increased in the pediatric population. When HAT is detected by ultrasonography or is highly suspected by clinical features, emergent revascularization or retransplantation is required for survival. Mortality rates for patients whose transplantation is complicated by HAT range from 20% to 60%. The literature suggests that urgent revascularization may salvage 20%–40% of grafts, but the majority of patients will require retransplantation [9]. Therefore, the prophylaxis of HAT is essential for the recipient's survival in Japan, where brain-dead grafts are rarely obtainable. In the immediate postoperative period, prophylactic infusion of heparin is done to prevent HAT while monitoring activated clotting time and aPTT.

However, during the operation, decisions about replacement therapy should always incorporate clinical factors. These include the presence or absence of generalized oozing and clot formation in the surgical field, and the likelihood of further surgical hemorrhage. Knowledge of the pathophysiology of severe hepatic disease and of the potential pitfalls of the operation is essential. During the neohepatic stage, transfusion of FFP might be avoided until the appearance of generalized bleeding in the operative field unresponsive to efforts of surgical hemostasis. Effective communication

with surgical colleagues is also vital, as is careful and comprehensive monitoring.

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