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Severe hepatocellular damage and acute anuric renal failure following a grade V hepatic injury treated by plasmapheresis and hemodialysis

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Suvit Sriussadaporn, et al. Severe hepatocellular damage and acute anuric renal failure following a grade V hepatic injury treated by plasmapheresis and hemodialysis

A case of severe hepatocellular damage and acute anuric renal failure following a gradeV hepatic injury is presented. Renal shutdown began on the third admission day. The patient also developed progressive hyperbilirubinemia and acute liver failure. His condition deteriorated in spite of intensive supportive care and aggressive hemodialysis. Billirubin level reached 51.4 mg/dL on the 13th admission day and plasmapheresis with plasma exchange was considered necessary to decrease his bilirubin levels. Intermittent hemodialysis and plasmapheresis were undertaken until the kidneys recovered following which the hepatic function rapidly returned to normal. We conclude that although hepatic function after severe hepatic injury usually returns to normal with conventional supportive therapy and the benefit of plasmapheresis in this patient is unclear, plasmapheresis may have a role in desperate situation of liver and renal failure in which the renal failure may be further aggravated and never recover in the presence of very high bilirubin levels. Plasmapheresis reduces bilirubin levels and probably other harmful metabolites while awaiting recovery of the renal function.

Index : Plasmapheresis, post-traumatic liver failure

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Jaundice and deterioration of hepatic function are frequently seen in early postoperative period of severe hepatic trauma. However, bilirubin levels and other liver profiles usually gradually return to normal with conventional supportive measures owing to the excellent regenerative capacity of the liver. Nevertheless, when severe hepatocellular damage occurs together with acute anuric renal failure, mortality is exceedingly high. Rapidly and progressively rising bilirubin levels in severely injured patients imply a poor prognosis, especially when there is associated acute renal failure(1). Reducton of bilirubin levels by plasmapheresis with plasma exchange in addition to hemodialysis and other supportive therapy in such situation has been seldom, if ever successfully performed in critically injured patients.

Plasmapheresis is a process of removing plasma from withdrawn blood with retransfusion of the formed elements into the donor. Generally type specific fresh frozen plasma or albumin is used to replace the withdrawn plasma components or for therapeutic purposes. When type specific plasma or fresh frozen plasma is used to replace the withdrawn plasma, the term "plasmapheresis and plasma exchange" is employed.

We present a case of severe hepatocellular damage and acute anuric renal failure following a gradeV hepatic injury who was successfully treated by plasmapheresis with plasma exchange and hemodialysis.

Case Report

A 23-year-old man sustained a motor cycle accident 30 minutes before arriving at the Emergency Room. On arrival, the blood pressure was 80/50 mmHg, pulse rate 1OO/minute and respiratory rate 20/minute. Physical examination revealed a conscious patient with tenderness at the upper abdomen and swelling in his right thigh. Diagnostic peritoneal lavage was performed and a positive result was obtained on the basis of aspiration of frank blood from the lavage catheter. Fracture at midshaft of the right femur was also diagnosed. He underwent immediate exploratory laparotomy through a midline incision. At exploration, 400 ml of blood in the peritoneal cavity was recovered. A contused gall bladder and a 4 cm long laceration at anterior surface of the liver along the line dividing the liver into anatomicalright and left lobe with undetermined depth were noted. No active bleeding from the lacerated liver was observed. Cholecystectomy was performed without any surgical procedure on the lacerated liver. Immediate fixation of the fractured femur with plate and screws was also performed. The patient was subsequently admitted to the intensive care unit with stable vital signs. CT scan of the abdomen was scheduled to evaluate the extent of hepatic injury.

Six hours later while awaiting for CT scan, his blood pressure dropped to 70/50 mmHg and the abdomen was distended. The hematocrit dropped from 32% to22%. Intraabdominal bleeding was suspected and he was reoperated. At reexploration~ 2,000 ml of blood was found in the peritoneal cavity. Transection of the liver along the anatomical plane of the right and left lobe from anterior surface of the liver to the inferior vena cava and a 1 cm tear at the retrohepatic vena cava were found. Active bleeding from the torn inferior vena cava and branches of right hepatic artery and right portal vein was noted. A vascular clamp was immediately applied to the hepatoduodenal ligament followed by temporary packing of the liver wound with a large laparotomy pad. The midline incision was subsequently extended into the right 7th intercostal space for better exposure. Suture ligature of the bleeding points and direct suture repair of the inferior vena cava injury with a 5-0 prolene were performed. The right lobe of the liver which was severely damaged was also removed. After all bleeding points had been stopped, the thoracoabdominal wound was closed with a chest tube placed in the right pleural cavity and an active sump drain and 2 Penrose drains placed at the right subphrenic area. During the operation, the hepatic inflow occlusion was performed intermittently with the total occlusion time of 30 minutes. The mean arterial pressure was as low as 30 mmHg for a few minutes at the beginning and then was maintained at 50-60 mmHg throughout the operation. At the end of the operation, the body temperature was 33.5°C. Neither steroids nor local hypothermia was given to the patient during hepatic inflow occlusion.

Postoperatively, the urine output progressively decreased to a few milliliters per hour in spite of vigorous correction of hypovolemia and administration of large doses of furosemide and acute anuric renal failure was finally established.

Twelve hours after the second operation, he was brought to the operating room again because of persistent bleeding from the sump drain. Oozing of blood from the raw surfaces of the remaining liver and diaphragm was found and eventually treated by packing with roll gauze after failure to stop by hemostatic agents, electrocoagulation and suture ligatures. The packing was removed 24 hours later, however, at this time the abdomen could not be closed by conventional fascia to fascia closure owing to massive visceral edema. A polyglycolic acid mesh was sutured to the rectus sheath to cover the abdominal viscera and primary skin closure was performed after the skin and subcutaneous tissue from both sides of the abdominal incision had been mobilized from the rectus fascia (bilateral bipedicle advancement flaps) to prevent excessive loss of intraabdominal fluid and fistula formation.

After the last operation, the patient had progressive azotemia due to acute anuric renal failure and intermittent hemodialysis was started on the 4th admission day. Rapidly progressive jaundice with deterioration of consciousness supervened at the end of the first week of admission. In spite of intensive supportive care and aggressive hemodialysis; BUN, Cr and bilirubin levels continued rising. On the 10th day of admission; BUN was 160 mg/dL, Cr was 12 mg/dL and total bilirubin (TB) was 50 mg/dL. On the 13th day of admission; TB was 51 mg/dL, direct bilirubin(DB) 29 mg/dL, alkalinephosphatase (AP) 534 U/L, SGOT 283 U/L, SGPT 84 U/L and prothrombin time (PT) 30 seconds (control 12 seconds). Physical examination revealed a deeply jaundiced patient in comatose state being supported by a mechanical ventilator. The vital signs were within normal limits. At this point, it seemed to us that treatment with hemodialysis and other supportive care was not enough for this patient. Very high bilirubin levels probably aggravated the already damaged kidneys. Reduction of bilirubin levels was thought to be necessary and plasmapheresis with plasma exchange was subsequently performed (Equipment: CS-3000 Plus Blood Cell Separator; Baxter Healthcare Corporation, USA). Transient decrease in bilirubin levels (from 50 mg/dL to 30 mg/dL) and improvement of consciousness were observed after each time of plasmapheresis with plasma exchange. Bedside ultrasonography was also performed to exclude extrahepatic bile duct obstruction and no abnormal bile duct dilatation was observed. However, right subphrenic collection was detected and drained percutaneously under ultrasound guidance.

During treatment with plasmapheresis and hemodialysis nutrition was maintained by total parenteral nutrition during the first two weeks of admission and then by enteric feeding throughout the remaining period of hospitalization. The kidneys started producing urine on day 30 and the patient was removed from the mechanical ventilator on day32. The last plasmapheresis(total 13 times) and the last hemodialysis (total 18 times) were done on day33 and day34, respectively. Serum bilirubin levels gradually decreased while the renal function and general condition of the patient were improving. On day 74, the renal function returned to normal and jaundice was almost totally disappeared; liver function test revealed TB 2.1 mg/dL DB 1.3 mg/dL, AP 1,127 U/L, SGOT 46 U/L, SGPT 31 U/L and PT 12.5 seconds (control 13.0 seconds). He was discharged home on day 75 with near normal condition. The patient's clinical course is shown in the figure.

Discussion

Missed diagnosis of gradeV hepatic injury at the first operation resulted from the policy of not to disturb the non-expanding, non-bleeding intrahepatic hematoma. Exsanguination following such management confirmed the unpredictable course of intrahepatic hematoma. Geis et al[2] reported the 37.5% complication rate in 16 patients with intrahepatic hematoma treated conservatively and 2 of them underwent emergency operations because of expanding hematoma and blood loss. Close observation is imperative if the intrahepatic hematoma found at the operation is left undisturbed. Postoperatively, CT scan may be a very useful investigation for further management(3,4). Demonstration of the extent of hepatic injury and sometimes arterial flow into the intrahepatic hematoma is important for management plan. Patients who have a large intrahepatic hematoma or arterial flow into the hematoma may be further treated by angiography and selective embolization of the hepatic artery[5]. In our case report, CT scan was planned but the patient had exsanguinated before the procedure was done.

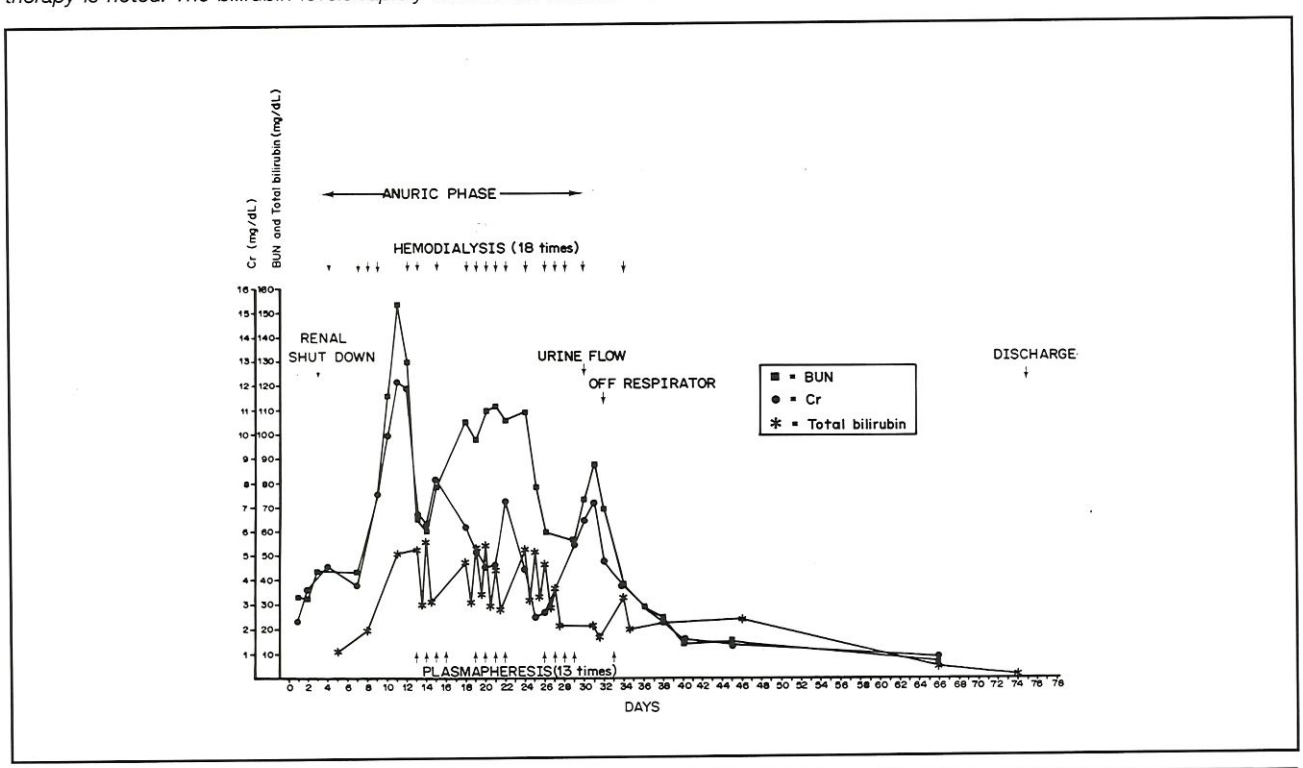
The decision to perform direct repair of the retrohepatic vena cava without intracaval shunt(6) or total

cm long) of the injury. Direct approach to the damaged retrohepatic vena cava by hepatic resection or hepatotomy concomitant with hepatic inflow occlusion(Pringle maneuver) has been reported with satisfactory outcome (8,9). Although the total duration of hepatic inflow occlusion performed in this patient was only 30 minutes, severe hepatocellular damage occurred. Hypoperfusion from massive blood loss, a 50% reduction of liver mass and intermittent hepatic inflow occlusion were the possible causes of severe deterioration of the hepatic function. The cause of progressive rise in bilirubin levels in such situations was thought to be intrahepatic cholestasis [1] not extrahepatic bile duct obstruction from operative injury as ultrasonography showed no evidence of bile duct dilatation. Although ERCP or transhepatic cholangiography can demonstrate bile ducts anatomy better than ultrasonography the patient was too sick to be moved from the intensive care unit for the procedures.

We did not give either steroids or local hypothermia to this patient during hepatic inflow occlusion. Local hypothermia and high dose steroids have been used to extend inflow occlusion time safely[10, 11]. In our opinion, steroids may be helpful before ischemic damage occurs to the liver cells. On the other hand, local hypothermia with ice saline may reduce patient's body temperature and aggravate the vicious cycle of hypothermia, acidosis and coagulopathy[12, 13].

Acute anuric renal failure in traumatized patients carries a high mortality. Early hemodialysis is of utmost important. Prevention of fluid overload, hyperkalemia and acidosis can be best managed by early hemodialysis. Furthermore hemodialysis helps to provide a space for nutritional support which is the key factor for recovery of the renal function and the patient's general condition. With optimum supportive care and no other aggravating factors, the duration of the renal recovery averages 10 to 25 days [14]. However, with concomitant severe hepatocellular damage, recovery of the renal function is unpredictable . Hyperbilirubinemia has been shown to cause several untoward effects on the kidneys ([15,16,17,18] and may make recovery of the renal function impossible. In our experience, recovery of renal functionn was possible in a multi-organ failure patient who had acute anuric renal failure and liver failure with total bilirubin up to 30 mg/dL when treated only with hemodialysis and intensive supportive care. In this reported case, total bilirubin level reached 50 mg/dL

Figure Showing clinical course of the patient during hospitalization. Fluctuation of serum bilirubin levels during plasmapheresis therapy is noted. The bilirubin levels rapidly returned to normal when renal function recovered.



before starting plasmapheresis. Deterioration of his condition despite aggressive hemodialysis and intensive supportive care urged us to consider plasmapheresis as an adjunctive therapy in the hope of temporary removal of bilirubin and probably other harmful metabolites while the liver and kidneys were non-functioning. Plasmapheresis with plasma exchange clearly reduced bilirubin levels. In addition, coagulopathy from severe hepatocellular damage was also corrected by replacing the withdrawn plasma with fresh frozen plasma. Plasma-pheresis has been used to treat several hepatocellular diseases i.e. fulminant hepatitis, Crigler-Najjar syndrome and intrahepatic cholestasis with variable outcomes (19,20,21).

Although the obvious benefit of plasmapheresis with plasma exchange in this patient can not be conclusively demonstrated, the clinical improvement after each and every time of intermittent plasmapheresis in our patient had encouraged us to continue such therapy while recovery of the renal and hepatic function was anticipated. We believe that plasmapheresis with plasma exchange had contributed toward the recovery of the organ functions in this patient.

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