Early Spontaneous Graft Intra- and Perihepatic Hematoma after Liver Transplantation

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Rezumat

Hematom spontan precoce intra- și perihepatic al grefei după transplant hepatic

Hematomul grefei este o complicație cu risc vital în transplantul hepatic și nu există în literatură concluzii clare în privința tratamentului acestuia, cu excepția celor din rarele prezentări de caz publicate. Hematomul grefei poate fi intrahepatic sau subcapsular, și se poate dezvolta spontan sau ca urmare a unor injurii hepatice ori a unor manevre invazive transhepatice percutane. Prezentăm cazul unui pacient de 62 de ani care a beneficiat de un transplant hepatic ortotopic cu ficat întreg pentru hepatopatie cronică decompensată datorită unei ciroze etanolică. Procedura chirurgicală s-a desfășurat fără evenimente. Ecografia Doppler de rutină și examenul CT au depistat, la 7 zile posttransplant, un hematom extrahepatic paracav, care a fost tratat conservator și a fost stabil timp de 11 zile. În ziua a 18-a, la aproximativ 6 ore după un episod de diaree acută, pacientul a prezentat o scădere a hemoglobinei serice până la 6,6 mg/dl, iar examenele imagistice au evidențiat un hematom intrahepatic voluminos ocupând hemificatul drept, câteva alte hematoame extrahepatice, revărsat pleural în cantitate semnificativă și ascită hemoragică. Pacientul a fost tratat conservator cu succes, cu revenirea lentă a funcției hepatice și externat o lună mai târziu în stare generală bună.

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Cuvinte cheie: transplant hepatic, hematom hepatic subcapsular, hematom intraparenchimos hepatic, laceratie hepatică, hematom perihepatic

Abstract

Hematoma of the graft is a life threatening complication of liver transplantation (LT) and there has been no overt conclusion in the literature about optimal management except in scarcely reported cases. It may be either intrahepatic or subcapsular, then again it may develop spontaneously or following parenchimal injuries or transhepatic percutaneous invasive manoeuvres. In this report we describe a rare case of large spontaneous graft intra- and perihepatic hematoma. A 62 year-old man underwent a whole graft orthotopic liver transplantation (OLT) for decompensated chronic liver disease due to alcoholic cirrhosis. The surgical procedure was uneventful. During the early post-operative course, routine Doppler ultrasound examination and CT-scan revealed an extrahepatic paracaval hematoma, 7 days after transplantation, which was stable and conservatively managed until the 18-th postoperative day, when rapidly expanding intraparenchimal hematoma involving the right hemiliver, several other perihepatic hematomas, significant right pleural effusion and hemorrhagic ascites were described. The patient was successfully treated conservatively (non-surgically) with slow recovery of the liver allograft and discharged one month later in good general status.

Key words: liver transplantation, subcapsular liver hematoma, intrahepatic hematoma, hepatic laceration, perihepatic hematoma

Introduction

Liver allograft hematoma after OLT, although rare, is a serious underreported condition, requiring prompt management to avoid any devastating consequence (1,2,3). Asymptomatic bleeding can be detected on imaging follow-up, whereas significant bleeding may lead to shock, expanding hematoma, hepatic rupture (4,5), graft loss with need of retransplantation or even death (3). Early spontaneous liver graft hematoma is uncommon and the association between intra- and extrahepatic hematoma may even worsen the postoperative course after transplantation.

Case Report

A 62-year-old caucasian male with a past history of decompensated cirrhosis due to alcohol abuse (MELD 18), presented with fatigue, painless jaundice, hepatomegaly, dark coloured urine and weight loss. The patient's family history was noncontributory. Negative

viral, autoimmune serologies and metabolic markers were noted. His serum carbohydrate antigen CA 19-9 was 58 U/ml. Serum α -foeto-protein was normal. At the time of admission alkaline phosphatase, gamma glutamyl transferase, 5-nucleotidase and conjugated bilirubin were elevated. The hemoglobin level was 9,2 mg/dl, platelet count showed 50.000 /mmc, prothrombin time and renal function remained stable. As the patient had been listed for transplantation 6 months previously, he was transplanted in september 2016 receiving a full-sized liver allograft from a female donor after brain death occurred as a consequence of a cerebrovascular accident. The donor had no significant past medical history, except for back pain from a herniated lumbar disc and peptic ulcer disease. The surgical procedure was uneventful, the hepatectomy to the recipient being performed without the use of the veno-venous bypass. A large cavo-cavostomy was tailored by triangulation to secure adequate outflow to the graft. Portal, arterial and biliary reconstructions were undertaken according to the standard

techniques, respectively. Operative time was 375 min, cold and warm ischemia time were 250 min and 45 min., respectively. Autotransfusion and transfusion were 600 and 1450 ml, respectively, red blood cell (RBC) and fresh frozen plasma (FFP) 2850 ml. Colloid administration was 4000 ml and cristaloids 2000 ml. Intraoperative Doppler ultrasound of the vascular anastomoses confirmed normal inflow, outflow and resistivity index. Immunosuppression was initiated intraoperatively with methylprednisolone 1g and simulect (basiliximab) 20 mg and continued with tacrolimus and mycophenolic acid (MMF) afterwards. Valgancyclovir was used for prevention of viral infection in the day 2 postoperatively. Patient initial recovery was marked by a moderate anemia and persistent thrombocytopenia. Despite, anticoagulation therapy with enoxaparine (Clexane) 0,4 ml/24 h was carried on throughout the hospital stay. In the

7-th postoperative day patient's hemoglobin level dropped to 7,7 mg/dl, serum C-reactive protein rose to 25 mg/dl, and total bilirubin to 4,95 mg/dl. The abdominal MDCT disclosed bilateral moderate pleural effusion, normal vascular anastomotic flow, a subcapsular linear laceration 6'37 mm (thickness/caudal extension) in the segment VII of the allograft, paracaval and subhepatic hematoma 58'61'65 mm (AP/T/CC) with active bleeding but without overt arterial feeder (*Fig. 1*). White blood cell, platelet count, prothrombin time, hepato-renal function and patient's hemodynamic were stable. The patient was treated conservatively with analgesia, intravenous antibiotics and fluids, red cell blood (RCB) (2 units) and human albumin. Meanwhile the hemoglobin and hematocrite rose up to 10 mg/dl and 30 % respectively, the platelet count up to 200 '10⁶/mmc, the hepatic tests and coagulation were found normal. He remained stable

Figure 1. MDCT - 7th postoperative day after liver transplantation: thin subcapsular laceration in the segment VII of the liver graft (L) and paracaval hematoma (H) with active bleeding; the bleeding inside the hematoma (B) is not related to the arterial anastomosis nor to the pancreatico-duodenal vessels (d)

(including abdominal ultrasound findings) until the 18-th postoperative day when he presented an episode of acute diarrhoea, followed by another decline in the hemoglobin level to 6,6 mg/dl and Ht to 19 %. The test for *Clostridium difficile* toxins A and B in stools was positive. Abdominal Doppler ultrasound examination showed a huge intrahepatic hematoma, 11 cm in diameter, ill defined, located in the right hemiliver, with moderate compression over the right segmental portal branches and right hepatic vein, associated with perihepatic hematoma (pre-hepatic, right subphrenic and posterior paracaval) and right flank hematoma. A significant amount of free intraabdominal fluid was also noted. The peritoneal drain inserted under ultrasound guidance in the lower right quadrant removed hemorrhagic ascites. Bacteriologic test of the ascites identified *enterococcus faecalis* and *pseudomonas aeruginosa* requiring specific systemic antibiotherapy. Thoracoabdominal MDCT showed moderate right pleural effusion with passive collapse of the lower right lobe, non-homogeneous structure of

the right hemiliver with hematic densities associated to a subphrenic hematoma with hematic level (consistent with a recent bleeding), 10'6'10 cm (AP/T/CC) in diameter, 8'10 cm right flank hematoma fused to the previous reported paracaval hematoma, and a prehepatic collection with fluid density, 4 cm in thickness. No active bleeding was noticed (Fig. 2). All the vascular anastomosis of the graft as well as their intrahepatic branches were patent. The patient presented respiratory distress, increased abdominal pressure with tenderness and right lower limb oedema without thrombosis. Laboratory tests revealed sudden increase in liver enzymes (up to 5000 UI/dl) raising the issue of hepatic necrosis and surgical revision. However, the hemodynamic stability, the patency of the graft inflow and outflow, absence of intrahepatic vascular flattening, normal coagulation tests, rapid improvement of the hepatic function, allowed us to keep on a conservative treatment. The patient was given respiratory continuous positive pressure (CPAP), hemodynamic support and a thin

Figure 2. MDCT - 19th postoperative day after liver transplantation: Large laceration of the right lobe with intrahepatic hematoma, and subphrenic hematoma with hematic level (C); Arterial anastomosis, lobar and segmental arterial branches (A), portal anastomosis, lobar and segmental portal branches (B) and cavo-caval anastomosis with hepatic veins (C) are patent

catheter was inserted into the right pleural cavity to evacuate the hemorrhagic efusion. US-guided puncture and drainage of the prehepatic collection was performed several times, due to the loculated appearance on US, in order to rule out a secondary infection and to decrease the compression on the hepatic parenchyma. Intermittent pleural and peritoneal drainage were maintained for the next three weeks. Patient condition slowly improved. Close imaging follow-up (daily Doppler US) revealed progressive left hemiliver hypertrophy with slow resorbtion of the right intraparenchimal and perihepatic hematomas, also confirmed by MDCT examination performed after 2 weeks (*Fig. 3*). He was discharged on the 50-th post-operative day with good clinical and metabolic status. Six months after LT the patient has normal clinical, metabolic and imaging findings.

Discussion

Hematoma of the liver allograft is scarcely reported in the literature but is a serious complication of liver transplantation (1,2,3,4). It may present as intrahepatic, subcapsular or both and may develop spontaneously or subsequently to parenchimal injuries, either intraoperative (parenchimal laceration by compression) (5,6) or afterpercutaneous transhepatic invasive procedures, such as endoscopic retrograde cholangiopancreatography (7) or liver biopsy (8). Likewise, it may occur either after full-sized or partial-liver graft transplantation (1,2,3,4). Whether this rare phenomenon is more likely to occur after either whole or live donor liver transplantation remains unknown. Transplanted livers may be more sensitive to microtrauma and to blood flow compromise than the native liver as a consequence of loss of

Figure 3. Follow-up MDCT: Hypertrophy of the left lobe; partial resorbtion of the intrahepatic and perihepatic hematomas; patent intrahepatic vessels.

vascular autoregulation and of collateral flow (8). Therefore, the liver graft must be handled with special care to prevent potential mechanical injuries, either during organ procurement or transplantation procedure (5,6).

This case describes a large spontaneous intra- and perihepatic hematoma of a liver allograft occurred in the early course after LT. The US and MDCT initially described a paracaval and subphrenic hematoma with active bleeding but with no overt arterial feeder, which rendered the selective transarterial embolisation useless. Likewise, a subcapsular linear laceration in the segment VII of the allograft was described which was likely to result in the huge intra- and extrahepatic hematomas subsequently. As far as we didn't recognise any graft intraoperative damage, we should consider it a spontaneous occurrence. Another plausible contributing factor might be the presence of the low platelet count during the perioperative period. The patient was in significant respiratory and abdominal distress, however the vital functions were stable. Major intraparenchymal and perihepatic hematomas with sudden highly elevated liver enzymes (AST), even non specific, would have been consistent for liver cell necrosis and acute liver damage. Moreover, lack of traumatic element and of the active bleeding on repeated CT scan, would suggest rather necrosis than expanding hematoma. This was in fact the main diagnostic dilemma which rose the concern of surgical revision in order to secure the hemostasis and/or to resect necrotic hepatic areas. Another concern is linked to the balance between hemorrhage and risk of thrombosis, increased in septic conditions (infected ascites) our patient having ongoing anticoagulation therapy throughout the hospital stay. Hepatic capsule has been certainly damaged in the affected area whereas several large perihepatic hematomas have occurred. The severity of the complication should be related to the extension of the decapsulated area of the graft. However, an intact capsule would lead to an increased intrahepatic pressure by the expanding hematoma, resulting in hepatic vascular compromise and hepatic necrosis. In our reported case, it is

likely that perihepatic large hematomas behaved like a „perihepatic packing” with compressive effect on the liver but without vascular intrahepatic compromise or thrombosis. On the contrary, the „liver compartment syndrome” (8) leads to acute hepatic failure, usually requiring retransplantation.

Conclusions

This is, to our knowledge, the first reported case of early spontaneous huge intra- and perihepatic hematoma, after OLT. Timely diagnosis, close clinical and imaging follow-up and suitable management including vital function support, antibiotics, percutaneous US guided drainage can successfully salvage the patient and the liver allograft. Our emphasis is that patients who develop even massive intraparenchymal and perihepatic hematomas in the early course post OLT can be treated nonsurgically provided they are hemodynamically stable, they have patent graft inflow and outflow, no compression over the hepatic vasculature and stable hepato-renal function.

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