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LETTER TO THE EDITOR

Bladder tamponade due to vesical varices during orthotopic liver transplantation

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Vesical varices due to portal hypertension and liver cirrhosis are extremely rare. Only four cases of such a phenomenon have been published [1–4]. We report a patient with primary biliary cirrhosis, who developed vesical varices accompanied by two episodes of hematuria. During orthotopic liver transplantation (OLT) the patient experienced massive urinary tract bleeding resulting in bladder tamponade requiring intraoperative hemostasis through a cystostomy.

The 58-year-old female patient was hospitalized with hematuria and urinary retention in a district hospital 5 month before OLT. Her personal history revealed an appendectomy in 1961 and a hysterectomy with adnexectomy due to ovarian cysts in 1988. Since 6 years, she was suffering from mild hyperbilirubinemia of unknown origin resulting in recurrent attacks of jaundice. Physical examination was only positive for hepatosplenomegaly. Her hemoglobin level was 6.8 mmol/l (normal 7.5–10.6), hematocrit 37% (normal 36–49), white blood count 3.9×10^9/l (normal 4–9), platelet count 140×10^9/l (normal 180–300) and thromboplastin time 23% (normal 80–110). Liver and cholestatic values included total bilirubin 152 μmol/l (normal <17), glutamic oxaloacetic transaminase 156 U/l (normal 11–50), glutamic pyruvic transaminase 107 U/l (normal 9–60), and alkaline phosphate 649 U/l (normal < 117).

Cystoscopic examination of the urinary bladder revealed massive varices up to 1.5 cm in diameter on the left lateral wall without active bleeding, but with fibrin on one varix, indicative for an inactive bleed. One week after admission, the bladder catheter was removed; the prothrombin time had normalized under vitamin K substitution.

Further examinations by duplex sonography and upper endoscopy revealed portal hypertension with hepatosplenomegaly, esophageal varices and ascites. Neither clinical examination nor duplex sonography of the abdomen showed evidence for an iliac vein thrombosis. Special hepatological examinations revealed an enormous elevation of antimitochondrial antibodies with a value of 1290 (normal <21), and the liver biopsy confirmed a primary biliary cirrhosis stage IV. Based on clinical and laboratory findings the patient was put on the waiting list for liver transplantation.

Only 1 week after evaluation for OLT, the patient had to be readmitted with hematuria, successfully treated by placement of a urinary catheter which was removed 2 weeks later.

Five months later, the OLT was performed with a model for end-stage liver disease (MELD) score of 24 points, and a Child Pugh score of 12 points (class C). At this time period, we used the classic transplantation technique with replacement of the caval vein and a femoro-porto-axillary venovenous bypass. During the venovenous bypass time (flow 1.4–3.2 l/min) a massive hematuria via transurethral Nelaton catheter (size: 14 French) was observed. Subsequently the patient became anuric due to a vesical tamponade manifesting as a huge palpable vesical 'tumor'. After the implantation of the liver graft, an additional lower midline incision for an extraperitoneal access was performed and 800 ml of partially clotted blood was removed from the bladder. As already seen 5 months earlier multiple 1.5 cm large and actively bleeding vesical varices on the left lateral wall of the bladder were detected, transfixed and ligated. With the exception of the intraoperative massive hematuria and bladder tamponade, no increased bleeding tendency was observed. Intraoperative values for prothrombin time were between 58% and 72% and for platelet count between 100 and 202×10^9/l. During the procedure, nine units of erythrocyte concentrates and 38 units of fresh-frozen plasma were given. The postoperative urologic course was uneventful with removal of the urinary catheter 2 weeks after OLT. Follow-up 48 months after transplantation with decompression of the portal hypertension was uneventful hepatologically as well as urologically with absent signs of urinary bladder varices.

Ectopic varices due to intrahepatic portal hypertension are predominantly located in the digestive tract. The prevalence is between 10% and 40% dependent on the cause of hypertension and the sensitivity of the diagnostic test [5]. Vesical varices are rare and seem to develop mainly after intestinal conduits, schistomosiasis and

retroperitoneal fibrosis [6–9]. Vesical varices as venous portocaval collaterals in portal hypertension due to liver cirrhosis are an extremely rare complication and described only in four cases [1–4]. Because the wall of the bladder is an uncommon collateral venous route, varices in this localisation seem to develop after interruption of the normal collaterals, i.e., after abdominal surgery [2].

Several treatment modalities for vesical varices are proposed: The administration of beta-blockers is indicated in patients without hematuria comparable to patients with esophageal varices. As well in patients with bleeding ectopic varices, octreotid application may be beneficial [5]. In symptomatic vesical varices surgical devascularisation, laser sclerosis and coagulation are often only of temporary effectiveness. The only successful long-term treatment is the surgical decompression of portal hypertension by a total or selective portocaval shunt, transjugular intrahepatic portosystemic shunt or OLT. Therefore, we propose the conservative management of vesical varices before OLT because surgical procedures are only of temporary success as long as portal hypertension is present.

In the present case, however, bladder tamponade due to bleeding vesical varices occurred as a result of coagulation disorders during liver transplantation and temporary changes in pressure (very likely elevated) in the portal vein probably during the setting up of the femoro-porto-axillary bypass. As urinary tract hemorrhage was observed about 3 h after placement of the urinary catheter, a traumatic insertion of the catheter may be excluded. Based on this case and other advantages, we changed our policy from liver transplantation with caval vein replacement and extracorporeal venovenous bypass to the piggy-back technique using a temporary end-to-side portocaval shunt during the anhepatic period so as to reduce portal blood pressure and to prevent bleeding from varices.

To summarize, we report a rare case of vesical varices due to portal hypertension with an exceptional bladder hemorrhage and tamponade during liver transplantation.

Uta Herden, Christian A. Seiler,
Daniel Candinas and Stefan W. Schmid
University Hospital of Bern,
Visceral and Transplantation Surgery,
Bern, Switzerland

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