Subcapsular Liver Hematoma in a Patient on Chronic Hemodialysis

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Dear Sir,

Chronic hemodialysis is associated with spontaneous hemorrhage occurring mostly in the gastrointestinal tract [1], subdural space [2], pericardium [3], pleura [4] and retroperitoneum [5]. In the literature, only 2 cases of subcapsular liver hemorrhage in hemodialysis patients were reported.

We describe a patient undergoing chronic hemodialysis in whom a liver mass – most probably a subcapsular hematoma – has been followed for the last 5 years.

Case Report

A 50-year-old woman of Georgian origin was admitted in October 1977 to the surgical ward in a state of sepsis secondary to a perforated gallbladder, which was subsequently removed. As her blood tests revealed persistently elevated levels of urea and creatinine (64.2 mmol/l, 90 mg% and 274 μ mol/l, 3.1 mg%, respectively) her renal function was evaluated at a later stage, and the diagnosis of chronic pyelonephritis was made, based upon the characteristic radiological findings in her kidneys and by the patient's account of recurrent urinary infections over the past years. One year later, the patient was readmitted with end-stage renal failure with severe dyspnea and generalized anasarca. Blood tests showed a creatinine of 1,352.5 μ mol/l, 15.3 mg%, urea 224.9 mmol/l, 315 mg% and a creatinine clearance of 2.7 ml/min. As recovery of renal function seemed unlikely at that time, the patient was later started on chronic intermittent hemodialysis (3 times a week for 4 h with the Travenol RSP artificial kidney).

In January 1979, the patient developed a massive pericardial effusion. A wide pericardium excision was performed and 1,500 cm³ of serofibrinous fluid (acid fast-negative) were evacuated from the pericardial cavity. Serum laboratory data included the following: globulins 42 g/l, albumin 32 g/l, γ -globulins 2,600 units, SGOT and SGPT 18 and 33 units, respectively; paraproteins were negative. On physical examination, the liver extended 2 cm below the right costal margin and a liver scan showed slight enlargement without space-occupying lesions. Fig. 1. Iliac angiography demonstrating deflection of the celiac plexus (P) and related arteries from the midline to the left side of the body. The branches of the hepatic artery (H) running parallel, distorted and stretched by the large avascular mass in the right lobe region.

In December 1981, continuous hypotension, not related to the hemodialysis treatment, became a prominent feature. A large, firm, irregular mass was palpated in the right abdomen. Blood tests at this stage were as follows: globulins 60 and albumin 35 g/L; SGOT, SGPT, LDH and CPK levels remained within normal limits. The clinical picture of hypotension and a steadily growing right abdominal mass continued until July 1983, accompanied by decreasing hemoglobin and hematocrit levels, so that frequent blood transfusions were necessary, averaging 5–6 units of washed packed red cells (PWC) per month. Coagulation studies showed normal one-stage prothrombin time (14 s) and normal serum fibrinogen levels (2.1–4.4 g/l). No fibrin-split products were detected. Partial thromboplastin time was 20 s and platelets counts were within the range of

324

Smetana/David/Pellet/Bar-Khayim

180,000–240,000 mm². On the basis of the clinical picture, “Tc Caphytate colloid radio-isotope liver scans done since June 1982, computerized tomography and celiac angiography (fig. 1), a subcapsular liver hematoma was diagnosed and confirmed in a blood pool study performed with “Tc pyrophosphate in vivo labeled red cells, disclosing that the mass was avascular. At this point, surgery was not recommended because of the patient’s definite refusal and also because, in the surgeon’s opinion, the mass was exerting homeo-static self-pressure on the possible sources of bleeding, and any operative intervention might thus have lead to a massive and possibly fatal bleeding. Fortunately, however, the patient’s clinical condition gradually improved, and since July 1983, the abdominal mass decreased in size, while the systolic blood pressure stabilized at 95–115 mm Hg. Transfusions were reduced to 1–2 WPC every 5–6 weeks, as prior to December 1981.

Discussion

Intrahepatic and/or subcapsular hematomas of the liver have been diagnosed following liver biopsies [6] and traumatic events to the abdomen [7], during pregnancy [8] and as complications of the use of oral contraceptives [9]. Treatment is dependent on the extent and location of the hematomas. Those located centrally can be simply evacuated, whereas a subcapsular tear with bleeding may require partial hepatectomy. Although patients on chronic hemodialysis have an increased tendency of developing bleeding disorders, subcapsular liver hematomas in these patients are extremely rare and have been described previously in 2 cases. One of the reported patients expired a short while after surgical evacuation of the hematoma, the other patient had been treated with a combined anticoagulant regimen of warfarin and dipyridamole for 3 months before developing the intrahepatic hematoma. This patient was treated conservatively. Our patient received regular heparin treatment of 5,000 units, used during hemodialysis only, without additional anticoagulant supplement. Thus, it remains unclear what could have triggered the hemorrhage in this patient after years of regular hemodialysis, particularly when scans revealed no structural liver abnormalities.

The conservative course pursued in our and in the other case reported, with favorable ultimate clinical out-

come, raises the question as to the optimal approach to subcapsular liver hematomas in chronically hemodialyzed patients. This approach should be individualized, dependent upon the extent and location of the liver hematoma. Surgical intervention may be indispensable in cases of uncontrolled intrahepatic bleeding. In other cases, cautious transfusions and dialysis with careful

regional heparinization may be successful in the management of these patients, rendering the need for surgical intervention unnecessary.

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