Case Report

Arterialisation of the Portal Vein with an Aortoportal Jump Graft for Portal Vein Thrombosis Following Liver Resection for Malignancy

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Fibrolamellar hepatocellular carcinoma (FHCC) is a variant of hepatocellular carcinoma, which mainly affects a young age group and carries a relatively good prognosis. It is widely accepted that aggressive curative resection is still the best option for FHCC. We report here a case of successful arterialisation of the portal vein with an aortoportal jump graft for portal vein thrombosis, which developed postoperatively in an already comprised portal vein with tumour invasion following an extensive liver resection for FHCC.

Keywords: Fibrolamellar hepatocellular carcinoma, liver resection, aortoportal jump graft

INTRODUCTION

Acute portal vein thrombosis can develop following abdominal surgery or neoplasm [1]. Although controversy exists with regard to treatment of this condition, it is widely believed that aggressive treatment should be administered quickly as this serious complication carries a high mortality and morbidity [2]. Current options for management of acute portal vein thrombosis and transjugular intrahepatic portosystemic shunt, thrombectomy and surgical reconstruction [3 – 6]. We report here a case of acute portal vein thrombosis in a patient following an extensive liver resection for FHCC and its successful management.

CASE REPORT

An 18-year-old girl with obstructive jaundice was referred to us for further investigation and management, having been diagnosed as suffering from FHCC. Initial examination was unremarkable except a 3 cm hepatomegaly below the right costal margin.

Imaging studies included an abdominal CT, visceral angiography and percutaneous transhepatic cholangiography. These investigations demonstrated as follows: 1) a space occupying le-
sion in the left lobe of the liver invading the medial sector of the right liver involving segments V and VII; 2) encasement of the left branch of the portal vein, middle and left hepatic veins by the tumour, together with a 90% obstruction of the right branch of the portal vein (Fig. 1); 3) presence of hilar lymphadenopathy; 4) obstruction of the left hepatic duct with compression of the right and common hepatic ducts.

In view of the nature of malignancy and her age, she underwent a left hepatectomy extended to segments V and VIII with preservation of the integrity of the right hepatic ductal system and the right hepatic artery. At operation, the portal vein was also explored and a Forgarty catheter was used to remove the invading tumour within the right branch of the portal vein. Hilar lymph node dissection was also carried out and a frozen section confirmed the presence of extrahepatic disease.

Postoperatively, she recovered well initially. However, 3 days later she developed acute thrombosis of the remaining right branch of portal vein which was confirmed on Doppler ultrasound. Faced with this difficult and serious postoperative complication, we decided to perform an urgent thrombectomy to unblock the portal vein. Unfortunately, rethrombosis occurred soon after thrombectomy. Therefore, we proceeded with resection of the thrombosed portal vein and complete reconstruction of the portal stumps. An end to side anastomosis was created between the mesenteric portal stump and the inferior vena cava by using an 8F PTFE ring graft. At the same time, the hepatic stump of the portal vein was anastomosed to the infrarenal aorta with an 8F PTFE ring prosthesis to form an aortoportal jump graft to achieve arterialisation of the hepatic portal vein (Fig. 2). Although postoperatively she developed a transient episode of ascites immediately after surgery that was managed conservatively, she made a full recovery and was discharged home on the 18th postoperative day. She enjoyed a good quality of life for 40 months but died subsequently from tumour recurrence.

DISCUSSION

Curative resection if the best option for hepatic tumours either primary or secondary [7, 8]. FHCC represents a different entity of hepatocellular carcinoma, carrying a good prognosis. It mainly affects a young age group and at presentation patients usually have evidence of extrahepatic involvement of malignancy [9]. It is widely accep-
ted that treatment for this group of tumours should be aggressive surgical resection and where deemed necessary liver transplantation can be performed following a total liver resection [10]. In our case, the patient presented with extrahepatic metastases with hilar lymphadenopathy, but with a resectable liver lesion.

The major cause of acute portal vein thrombosis in adults is malignant disease, mainly from pancreatic and hepatocellular carcinoma, constituting 5% to 6% of portal vein thrombosis in the West [2]. Acute portal vein thrombosis can also occur following blunt trauma or intra-abdominal surgery [1]. Without prompt treatment, it is invariably fatal in the acute situation especially when complicated by small bowel infarction. In our patient, the acute portal vein thrombosis developed as a result of a combination of direct invasion by the tumour and surgery within the portal vein.

Arterialisation of the hepatic portal vein with portocaval shunt has been performed successfully in patients with portal vein thrombosis undergoing liver transplantation [12]. Temporary arterialisation of the portal vein was also described during liver transplantation [13,14] and liver resection [15]. Here, we report the first of such cases following liver resection for neoplasm. We feel that although it is technically challenging, aortoportal jump grafting could be achieved as a salvage procedure for patients with portal vein thrombosis following liver surgery who would otherwise have an extremely grave prognosis. We also postulate that this technique might be useful in extreme cases of liver resection in order to achieve maximal tumour clearance.

References


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