How to manage splenic rupture during major liver resection?

Mehdi OUAÏSSI (1), Stéphane BENOIST (1, 2), Christophe PENNA (1, 2), Bernard NORDLINGER (1, 2)

(1) Assistance-Publique-Hôpitaux de Paris, Hôpital Ambroise Paré, Department of Surgery Boulogne ; (2) Université Versailles Saint Quentin en Yvelines, Versailles.

SUMMARY

Spontaneous splenic rupture is a rare but life threatening complication of major liver resection with only five reported cases during major liver resection under hepatic vascular occlusion. We report two cases of splenic rupture during liver resection including the first case during portal triad clamping. In both patients, the hemorrhage was stopped by removing the vascular clamp. A splenectomy was performed in both patients and liver resection was completed under vascular clamping without complications. Although very rare, physicians should be aware of the possibility of splenic rupture during liver resection because instead of increasing vascular occlusion, clamp removal usually stops the hemorrhage.

Introduction

Splenic rupture is a rare but life threatening complication of major liver resection that must be recognized and managed properly to avoid massive hemorrhage. Only five cases of splenic rupture during liver resection have been reported [1-3]. In all these cases, splenic rupture occurred during liver resection under hepatic vascular exclusion. There are no reported cases with only portal triad clamping [1-3]. We report two cases of splenic rupture during major liver resection including the first case of splenic rupture during portal triad clamping.

Case reports

Patient 1

A 72-year-old man was operated on for metachronous colorectal liver metastasis. Intraoperative ultrasonography showed three liver lesions in the right lobe with one lesion close to the cavo-hepatic junction. A right hepatectomy under hepatic vascular exclusion was planned. Five minutes after liver transection, there was a rapid drop in mean arterial pressure from 93 to 45 mm Hg and a sudden and severe intra-abdominal hemorrhage occurred. The overall intraoperative fluid balance was 2000 ml and the central venous pressure was 8 mm Hg just before the intra-abdominal hemorrhage. The first hypothesis was injury to the vena cava or hepatic veins by the vascular clamps but no vascular injury was found. There was no injury to the right adrenal vein, which had been ligatured and divided before the vascular clamps were applied. There was no hemorrhage from the cut surface of the liver. Exploration of the peritoneal cavity revealed several lacerations of the splenic capsule with intense bleeding. Hemorrhage was stopped by removing the vascular clamps resulting in a rapid decrease in splenic vein pressure. A splenectomy was performed and the liver resection was performed under hepatic vascular exclusion with no further hemodynamic difficulties. Overall, six units of pack red cells were administered intraoperatively. The postoperative course was uneventful. Pathological examination of the spleen revealed congestion but no another abnormalities and the non malignant liver parenchyma was normal.

Patient 2

A 67-year-old man without any underlying liver disease was operated for a 10 cm diameter hepatocellular carcinoma located in the right lobe of the liver. A right hepatectomy (segments V, VI, VII, VIII) was planned. At surgery, the non malignant liver was normal and there was no evidence of portal hypertension. Ten minutes after liver transection began under portal triad clamping, a sudden and massive intra-abdominal hemorrhage occurred. The overall intraoperative fluid balance was 1500 ml, the central venous pressure was 6 mm Hg and the mean arterial pressure was 102 mmHg just before the intra-abdominal hemorrhage. The absence of portal vein injury by vascular clamp was checked. There was no hemorrhage from the cut surface of the liver and no injury of the retrohepatic vena cava was found. Finally, exploration of the peritoneal cavity revealed a ruptured spleen with several lacerations of the splenic capsule. The hemorrhage was stopped by removal of the vascular clamp. A splenectomy was performed and the liver resection was completed under portal triad clamping with no complications. Four units of...
pack red cells were administered intraoperatively. The patient experienced postoperative left pleural effusion requiring drainage. Pathological examination confirmed a complete resection of the hepatocellular carcinoma on a non-cirrhotic liver. The spleen was congested with no other abnormalities.

Discussion

Massive bleeding during liver transsection can be life threatening if the cause is not rapidly recognized and treated. The most common etiology of massive bleeding is injury to the hepatic vessels in the cut surface of the liver. Massive bleeding can also be related to injury to the major hepatic vessels injury such as the portal vein, the hepatic veins or the inferior vena cava. In these situations, the control of acute hemorrhage is achieved by applying or replacing the vascular clamp to control and stitch the injured vessel. In contrast, in case of splenic rupture, the vascular clamps must be removed, resulting in a rapid decrease of intrasplenic pressure to control massive bleeding. This is simpler and more effective than supra-celiac aortic and splenic artery clamping. For these reasons, although it is very rare, the possibility of splenic rupture should be known because instead of increasing vascular occlusion, removing the clamps usually stops the hemorrhage.

As previously reported [1-3], we performed a splenectomy after stopping the hemorrhage by removing the vascular clamp. Conservative management such as splenopexy is not recommended because it prevents the use of vascular occlusion while finishing the major liver resection and exposes the patient to a high risk of further postoperative bleeding.

To our knowledge, this is the first case of splenic rupture during portal triad clamping. After portal triad clamping, there is a moderate increase in portal venous pressure and most of the portal blood flow is redirected to the left gastric vein and the short gastric vein via the splenic vein and the spleen [4]. In our second non-cirrhotic patient, these portosystemic collateral channels were probably insufficiently developed and after portal triad clamping, the retrograde blood flow in the splenic vein may have caused severe congestion of the spleen elevating intrasplenic pressure and causing rupture of the splenic capsule. This hemodynamic hypothesis is supported by the fact that acute hemorrhage was stopped by removing the clamps.

REFERENCES