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Endovascular management of splanchnic arteries bleeding in pancreato-biliary disease

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Background. *Splanchnic artery bleeding is a life-threatening condition, especially in high-risk patients. The purpose of this study is to evaluate the efficacy of endovascular treatment of splanchnic artery bleeding in pancreato-biliary disease, considered as survival at the 3-month follow-up.*

Patients and methods. *From 1992 to 2001 39 patients with upper splanchnic arterial lesion due to acute and chronic pancreatitis after surgery or percutaneous procedures, or as a complication of aneurysms or trauma, were treated using endovascular techniques. The patients underwent CT control immediately after the procedure, after seven days and then at the 3, 6 and 12-month-follow-up.*

Results. *In some patients, more than one angiography was necessary to identify the source of bleeding. Bleeding was stopped in all treated patients. Fatal re-bleeding occurred in 6 patients and, in the first part of the study, 2 patients died of hepatic failure after hepatic artery embolization.*

Conclusions. *Splanchnic artery bleeding is a life-threatening condition. Endovascular treatment can reach a clinical success rate of up to 75% at three months.*

Key words: pancreatitis - complications; biliary tract disease - complications; mesenteric arteries - angiography; haemorrhage - therapy; embolization, therapeutic

Introduction

Splanchnic artery bleeding is a life-threatening condition. Although mortality and morbidity associated with major pancreatic and biliary surgery have diminished with recent advances in surgical techniques, post-operative complications, especially following pancreatoduodenectomy, are still common.[1] The incidence of hemorrhagic complications due to arterial disruption after pancreatic surgery, especially pancreatoduodenectomy, has not changed in the last 20 years (2%-18%).[2] Bleeding is the second most serious complication after sepsis due to the dehiscence of the pancreatic anastomosis. When arterial disruption related to pancreatitis, ruptured aneurysm or trauma occurs, the mortality rate

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is high and can reach 70%.[3] Surgical intervention for upper splanchnic artery bleeding, including ligation of the proximal and distal portions of the lesion, may be critical in debilitated and high-risk patients. The upper splanchnic artery bleeding in pancreato-biliary disease can be managed by endovascular techniques.

Patients and methods

From 1992 to 2001, 39 patients with upper splanchnic arterial lesion due to acute and chronic pancreatitis after surgery or percutaneous procedures, or as a complication of aneurysms or trauma were treated using endovascular techniques.

The aetiology of the hemorrhage was assumed on the basis of the non-invasive vascular imaging (CT angiography, MR angiography, color-Doppler Ultrasonography) and the angiographic features as well as the case history (Table 1).

In 29 patients, the procedure was performed in emergency owing to unstable hemodynamic conditions. During the procedure, the hemodynamic parameters and pulse-oxymetry were monitored. In all patients, an intravenous infusion of acetate ringer was administered through a 16-gauge cannula needle inserted into a peripheral vein at an infusion rate of 15ml/kg/hr. In all patients, the procedure was carried out with anesthesiological support. A central venous catheter was positioned for infusions and pressure monitoring. Colloid solutions and packed red blood cells were administered as needed. Antibiotic prophylaxis was performed in all the patients by intravenous one-day administration of a second-generation cephalosporin.

Written consent was obtained from each patient before arteriography. At first, celiac arteriography, superior mesenteric arteriography and arterial portography were performed with a 5F catheter. The endovascular treatment was performed with the coaxial catheterism technique in all cases. For the embolization, 5F catheter was used for the selective catheterization of the vessel afferent to the arterial lesion and a microcatheter (Tracker-18 unibody Target Therapeutics, Fremont, CA, USA) for positioning the coils (Tornado Embolization Coils, W Cook Europe, Bjaeverskov, Denmark) or to inject the acrylic glue (Histoacryl B/Braun, Melsungen, Germany) added to Lipiodol UF (Guerbet, Aulnay-Sous-Bois, France). For artery repair a balloon-expandable covered stent (Jostent Peripheral Stent Graft, Jomed Implantate GmbH, Rangendingen, Germany) was used.

The patients underwent CT control immediately after the procedure, after seven days and then at the 3, 6 and 12-month-follow-up.

Table 1. Patient data: ethiology of haemorrage and distribution of the arterial lesion

Bleeding Artery	Acute Pancreatitis	P-B Surgery	Percutaneous Procedure	Chronic Pancreatitis	Aneurysm	Trauma	Total
Hepatic	-	3	7	-	2	1	13
Gastroduodenal	2	5	1	2	-	-	10
Splenic	5	-	-	1	1	-	7
Middle Colic	3	1	-	-	-	-	4
Pancreatico duodenal	1	-	-	2	-	-	3
Left Gastric	-	-	-	1	-	-	1
Gastroepiploic	1	-	-	-	-	-	1
Total	**12**	**9**	**8**	**6**	**3**	**1**	**39**

Results

From our experience, some patients required more than one angiography to identify the source of bleeding. The distribution of the arterial lesions is summarised in Table 1. The hepatic and gastroduodenal arteries were the most common locations of bleeding after surgery (Figures 1a, 1b). On the contrary, in pancreatitis, the distribution of the arterial lesions is quite uniform (Figures 2a, 2b, 3a, 3b, 3c). Bleeding was stopped in all treated patients. Different radiological endovascular techniques were used. The treatment modality depended on the vessel involved. Embolization, both proximal and distal to the bleeding site was performed whenever possible.

Embolization of the artery afferent to the arterial disruption using coils up and downstream of the arterial lesion (endovascular ligation) was performed in 21 cases (Figures 2c, 2d). The embolization with coils (Figures 2c 2d, 3d) was enough to stop the bleeding in about 50% of cases (Table 2). Acrylic glue with coils was used in 5 cases to achieve haemostasis more quickly. The embolization of the bleeding vessel just with acrylic glue was applied as endovascular treatment modality in 8 cases (Table 2). The embolization with acrylic glue alone was performed when selective catheterization downstream of the arterial lesion was impossible or in order to achieve an instant embolization (Figure 4). In 3 patients, an exclusion of the arterial disruption (Figures 1b, 1c, 1d) was achieved using a balloon-expandable covered stent (Jostent Peripheral Stent Graft, Jomed Implantate GmbH, Rangendingen, Germany). In acute pancreatitis, an artery dissection following selective catheterization resulted in bleeding stoppage in 2 cases of arterial lesion. These 2 cases were not considered as technical successes. After the procedure, fatal rebleeding occurred in 6 patients and, in the first part of the study, 2 patients died of he-

Table 2. Materials and techniques

Aetiology	Coils	Coils + glue	Glue	Covered stent	Dissection
Acute pancreatitis	6/12	1/12	3/12	0/12	2/12
P-B Surgery	2/9	3/9	1/9	3/9	0/9
Percutaneous procedure	8/8	0/8	0/8	0/8	0/8
Chronic pancreatitis	4/6	0/6	2/6	0/6	0/6
Aneurysm	0/3	1/3	2/3	0/3	0/3
Trauma	1/1	0/1	0/1	0/1	0/1
Total	**21/39 (54%)**	**5/39 (14%)**	**8/39 (20%)**	**3/39 (7%)**	**2/39 (5%)**

Table 3. Outcomes

Etiology	Mortality		Clinical Success
	Fatal Rebleeding	Fatal Complication	
Acute pancreatitis	4*/12	0/12	6/12
P-B Surgery	2/9	1/9	6/9
Percutaneous procedure	0/8	0/8	8/8
Chronic pancreatitis	0/6	0/6	6/6
Aneurysm	0/3	1/3	2/3
Trauma	0/1	0/1	1/1
Total	**6/39 (16%)**	**2/39 (5%)**	**31/39 (79%)**

* bleeding stoppage after artery dissection

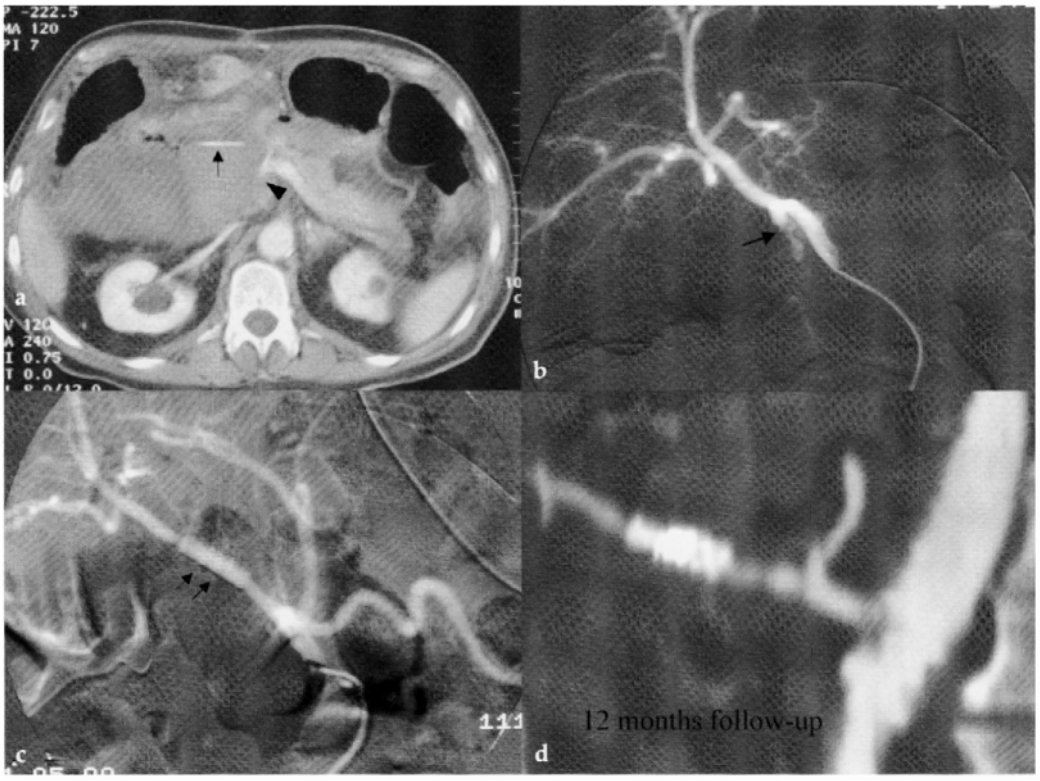

Figure 1a. Splanchnic artery bleeding after pancreatoduodenectomy (two cases). Axial CT scan, carried out in the venous contrastographic phase, highlights a voluminous subhepatic hematoma with intraperitoneal spread of contrast medium resulting in a contrast-blood level (arrow). The spleno-portal confluence (arrowhead) is compressed by the huge hematoma.

Figure 1b. Selective angiography of the hepatic artery, in another case, shows rupture of the hepatic artery involving the gastruoduodenal artery stump at the origin (arrow).

Figure 1c. In the same case, selective angiography after the placement of two covered stents (arrows) with a slight overlap in the hepatic artery for hepatic artery repair demonstrates bleeding stoppage and patency of the hepatic artery.

Figure 1d. In the same patient, at the 12-month follow-up the patency of the hepatic artery was still present at CT angiography.

patic failure after hepatic artery embolization (Table 3).

Discussion

Rupture of a pseudoaneurysm and bleeding into the abdominal cavity or gastrointestinal tract as a result of a different aetiology, although with a different clinical presentation, is often associated with massive, life-threatening haemorrhage.

The majority of pseudoaneurysms occur in pancreatitis, in association with or in close proximity to, pancreatic pseudocysts. In particular, although the natural history of pseudocyst in chronic pancreatitis is unpredictable, it can gradually erode the vascular wall of the adjacent vessels. This erosion has a double pathogenetic mechanism, enzymatic and mechanic. In the first case, the activated proteolytic enzymes in the liquid of the pancreatic pseudocyst cause necrotizing arthritis with a maceration of the vessel wall and

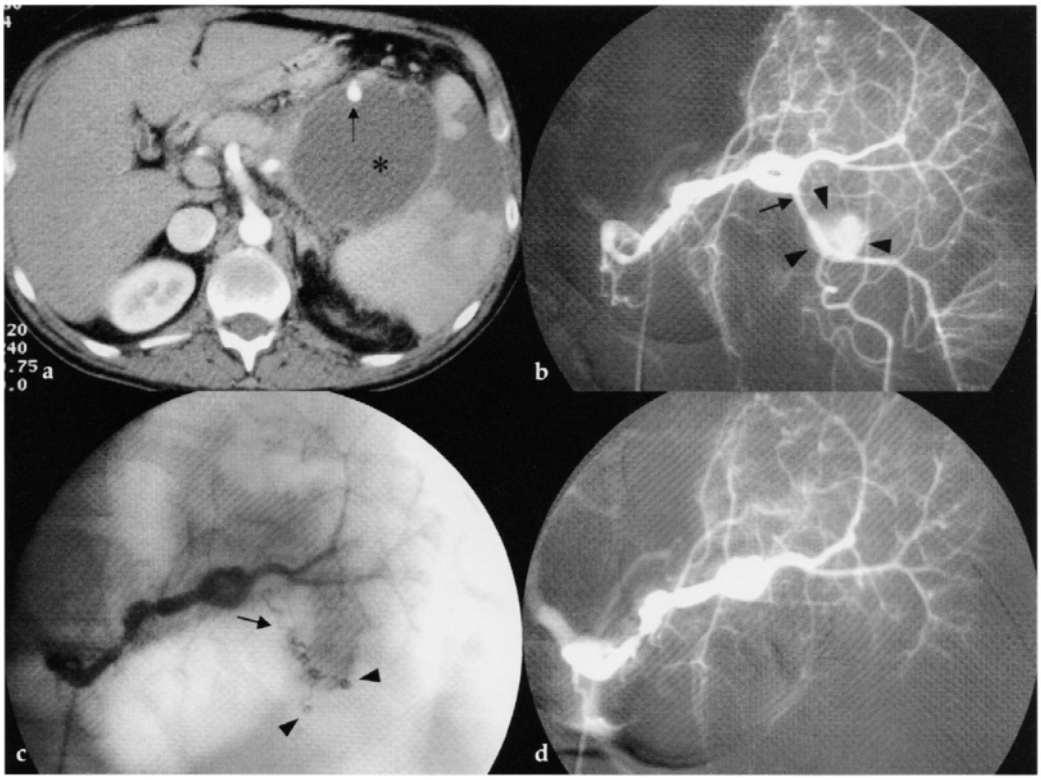

Figure 2a. Bleeding pancreatic pseudocyst. Axial CT scan in the arterial contrastographic phase demonstrates pseudocyst of the pancreatic tail (asterisk) involving the splenic artery. A small pseudoaneurysm is visible in front of the pseudocyst (arrow).

Figure 2b. Selective angiography of the splenic artery shows the pseudoaneurysm (arrowheads) slightly down-stream of the left gastroepiploic artery origin (arrow).

Figure 2c. After placing coils both up- (arrow) and down-stream (arrowheads) the pseudoaneurysm, at selective splenic arteriography.

Figure 2d. A complete exclusion of the lesion is present

bleeding inside the pseudocyst. The size of the pseudocyst is decisive in developing the type of the lesion: bleeding in a small pseudocyst is necessarily contained and more commonly ends up as a pseudoaneurysm; in case of a larger pseudocyst, rupture of the pseudoaneurysm and bleeding into the gastro-intestinal tract or into the peritoneal and/or retroperitoneal spaces can occur. Angiography reports the presence of pseudoaneurysm, without bleeding, in 10-21% of patients with chronic pancreatitis.[4] The occurrence is higher (10-31%) in patients with pseudocyst.[5,6] Hemorrhagic complications are expected in 6-31% of patients with pancreatic pseudocyst[7] and in 7-14% of those suffering from chronic pancreatitis.[8] The preventive vascular study of patients with pancreatic pseudocyst must be carried out since arterial pseudoaneurysm, although infrequent, is a potentially catastrophic complication. The risk vs. benefit balance when using angiography in this respect is, however, debatable and non-invasive vascular imaging is preferable (Doppler US, multidetectors CT, MRA).[9] Although in inflammatory pancreatic disease gastro-intestinal bleeding is frequently sustained by concurrent peptic disease, the

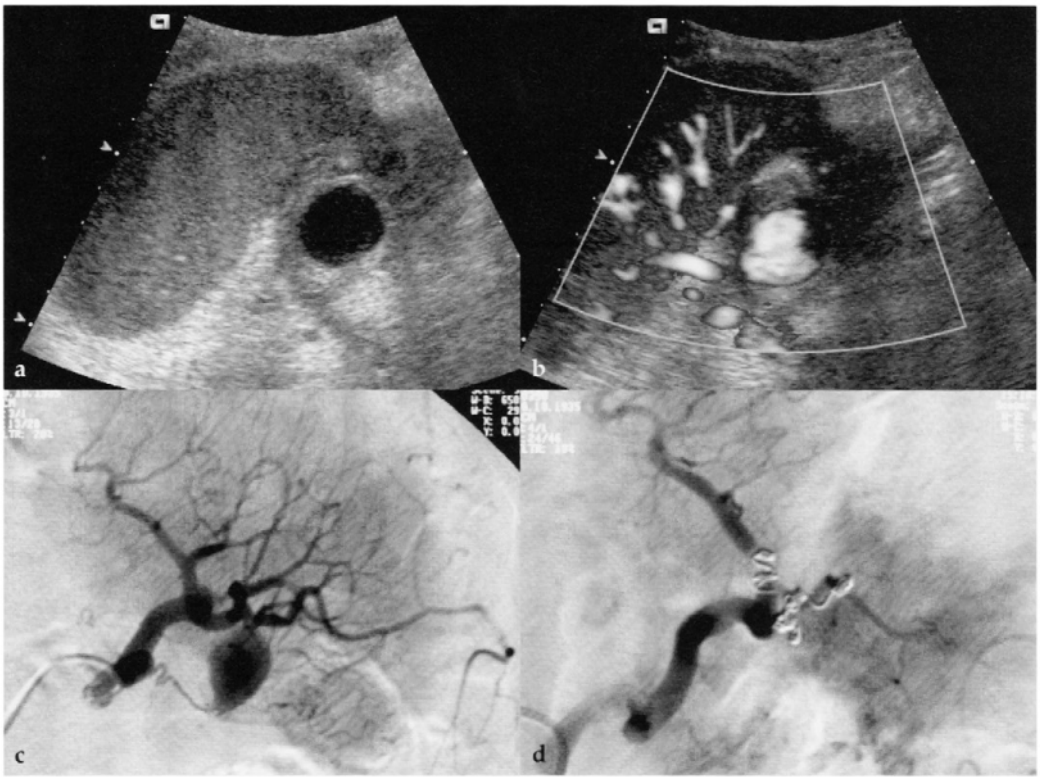

Figure 3a. Bleeding pancreatic pseudocyst. Ultrasonography shows a fluid-filled mass (asterisk) at the level of the pancreatic tail, adjacent to the splenic hilum.
Figure 3b. At power-Doppler, image blood flow within the pseudocystic lesion is present.
Figure 3c. Selective splenic angiography shows the presence of a pseudoaneurysm (asterisk) involving the splenic artery at the splenic hilum.
Figure 3d. after embolization with coils the selective angiographic control demonstrates the pseudoaneurysm exclusion

suspicion of vascular origin, apart from the negative findings of the endoscopic examination, above all correlates with the intermittence of the bleeding. Usually haemorrhagic shock is the clinical presentation of ruptured pseudoaneurysm, with a mortality rate of 50%.[10] Thanks to the most recent image reconstruction programs (MPR; MIP; SSD; VR), it is, nowadays, possible to obtain a well-defined arterial map of the pancreatic region thus identifying the lesion's vessel of origin with CT, particularly the multi-slice technique, or with MR, by means of 3D sequences in contrastographic phases. Angiography therefore plays no role in the diagnostic phase but is immediately used for treating lesions identified with non-invasive imaging. Embolization has a high possibility of success in the treatment of these lesions. With this aim it is above all necessary to define the afferent and efferent arteries involved. Only the occlusion of all efferences and therefore afferences to the pseudoaneurysm results in the certain and definite exclusion of the lesion from the arterial flow and its subsequent progressive collapse. The pseudoaneurysm has a pseudo-wall of variable thickness, which can derive from the fibrotic wall of the pseudocyst. It is better not to place coils inside the pseudoaneurysm, unless this is tech-

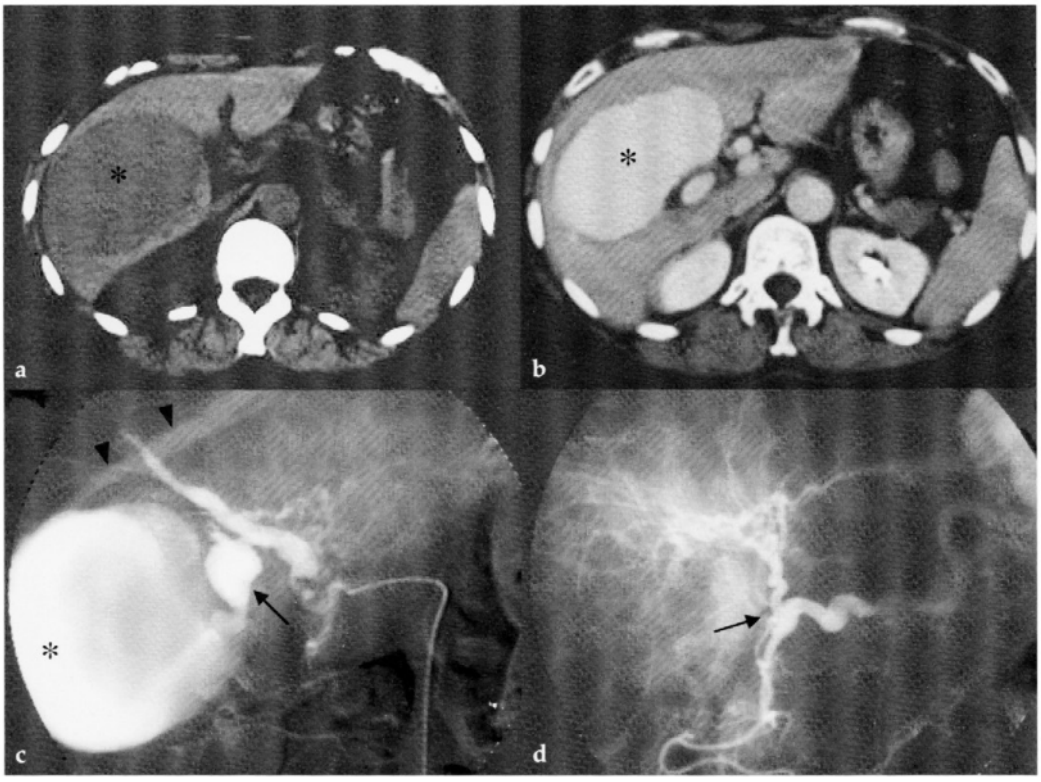

Figure 4a. Ruptured aneurysm of the right hepatic artery. CT scans in the pre- contrastographic phases show the presence of a huge intrahepatic hematoma (asterisk)

Figure 4b. Ruptured aneurysm of the right hepatic artery. CT scans in the post- contrastographic phases show the presence of a huge intrahepatic hematoma (asterisk)

Figure 4c. Selective common hepatic artery angiography demonstrates a ruptured aneurysm (arrow) arising from the right hepatic artery with the formation of a huge intrahepatic hematoma (asterisk) partially drained into the right hepatic vein (arrowheads).

Figure 4d. Selective common hepatic artery angiography following embolization with acrylic glue shows the occlusion of the right hepatic artery at the origin (arrow) with complete haemostasis.

nically indispensable to occlude the efferent vessels that would otherwise be inaccessible,[11] but to proceed to the occlusion of the afferent and efferent vessels with a technique similar to surgical tying.

Arterial bleeding in acute pancreatitis is a dramatic occurrence, subject to a high mortality rate. Stopping it may be very difficult, both surgically and radiologically. Emergency surgery is associated with a rather high degree of failure and mortality. In particular, there is more than an 80% occurrence of further bleeding,[12-14] while surgical resection is subject to more than 50% mortality.[8] The radiological treatment of acute bleeding from an arterial disruption in acute pancreatitis can fail due to the excessive extent of the maceration of the vessel or the involvement of several arterial branches. Once again the interventional procedure foresees the embolization of the bleeding vessel or vessels. The embolization of the vessel proximally or distally with respect to the site of bleeding is a condition for success not to be ignored. On the contrary, the embolization of the bleeding vessel with spongostan is subject to a high re-

lapse rate since the fibrin sponge is sensitive to the lytic action of the activated pancreatic enzymes.[15] The embolization with coils is a solution only when they are placed up and downstream of the breach in the bleeding arterial vessel where the wall is not affected by enzymatic erosion. From personal experience, when haemodynamic instability required a rapid embolic action, or when catheterization of the vessel downstream of bleeding was impossible, acrylic glue proved to be the most efficient embolizing agent.[16] Injected upstream of the arterial lesion and correctly diluted, the glue is able to reach the arterial tract downstream and, with its progressive polymerization, brings about a proximal and distal occlusion and, at the same time, fills the pseudoaneurysm.

The incidence of haemorrhagic complications due to arterial disruption after pancreatic surgery, especially pancreatoduodenectomy, has not changed in the last 20 years (2%-18%).[3] Bleeding is the second most serious complication after sepsis due to the dehiscence of the pancreatic anastomosis. Early bleeding, in the first two weeks, due to insufficient intra-operative hemostasis at the abdominal vessel level or in correspondence with the anastomosis, requires a second laparotomy. Late bleeding, after the first-second week, is more difficult to diagnose, and its treatment is less codified. It can follow arterial disruption by erosion after the dehiscence of the pancreato-jejunal anastomosis. More than two weeks after surgery, even modest bleeding must make anastomotic dehiscence a suspect. This slight bleeding is defined as »sentinel bleeding« because it often precedes, by 6 hours to 10 days, massive hemorrhage from the erosion of a large arterial branch. Rumstat *et al*[3] suggest immediate surgical intervention with a revision of the pancreatic-digestive anastomosis when »sentinel bleeding« appears at the drainage or the gastro-intestinal tract. When the dehiscence of the anastomosis occurs, the massive hemorrhage causes mortality in 15% to 58% of cases.[3,17] The frequent presence of anastomotic dehiscence requires an embolization technique similar to that used in acute pancreatitis. The most commonly involved vessels are the gastroduodenal artery stump or the common hepatic artery.[3,17-20] The embolization with coils leads to definite stoppage of the bleeding only when the gastroduodenal artery is occluded in a tract not involving septic maceration or, alternatively, the common hepatic artery is directly occluded. Embolization is sometimes used as a temporary procedure to stop or slow down bleeding so that the patient can be operated on electively rather than in emergency.

Considering the possible complication of the endovascular embolization, the vessels that can generally be embolized safely in this region include the left gastric, gastroduodenal, gastroepiploic, and pancreaticoduodenal arteries.[21] The occlusion of the common hepatic artery, with normal patency of the portal vein has no clinical consequences; embolization is, however, unadvisable in the presence of thrombosis or compression of the portal vein.[22] Moreover, the presence of a biliary-enteric anastomosis is considered a risk factor for developing a hepatic abscess following a hepatic artery embolization.[23] The treatment of bleeding from the common hepatic artery or from the short stump of the gastroduodenal artery following a pancreatoduodenectomy is often problematic for the surgeon, and the radiologist should decide to go ahead with classic embolization. But compression, even to the point of thrombosis, of the portal vein is common due to the presence of adjacent hematic collection. In these cases, the alternative treatment to embolization is positioning covered stents that maintain the patency of the hepatic artery in emergency (Figure 1). Although very significant, there are still only a few reports in literature concerned with the use of this technique.[24,25] Occlusion of the stent over time due to hy-

perplasia of intima is predictable. Nevertheless, the treatment stops the bleeding immediately and maintains the hematic contribution to the liver. The slow occlusion of the stent can then be compensated by the recruitment of collateral intra-hepatic arterial circulation and the return to normal portal flow or the growth of a collateral portal network.

Conclusions

Considering the high mortality rate of splanchnic artery bleeding in pancreato-biliary diseases and the poor results of surgical intervention, the endovascular approach, with embolization or repair of the bleeding vessel, has to be considered as the treatment of first choice. In our series, the endovascular treatment of splanchnic artery bleeding in pancreato-biliary disease resulted as clinically successful in up to 75% of cases at the three-month follow-up.

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