Aneurysm of the pancreaticoduodenal arteries with a celiac trunk lesion: Current management

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Objective: **We report a ruptured aneurysm of the pancreaticoduodenal arteries without acute or chronic pancreatitis but associated with a median arcuate ligament division that compressed the celiac trunk, an exceptional event, thus far described in only 11 patients. We also conducted a targeted review to seek information about clinical presentation, to hasten diagnosis and assist in therapeutic management.**

Methods: **A 54-year-old man with retroperitoneal hemorrhage associated with an arcuate ligament division, documented on computed tomographic scans, underwent diagnostic arteriography and embolization to treat the bleeding aneurysm. In a second elective operation the causative arcuate ligament was sectioned to decompress the celiac artery, to prevent aneurysm recurrence.**

Results: **Embolization stopped the aneurysmal bleeding, and arteriograms showed that surgical sectioning resolved the celiac trunk stenosis. At 1-year follow-up the patient had no signs of complications or recurrence of disease.**

Conclusion: **Ruptured aneurysm of the pancreaticoduodenal arteries associated with stenosis of the celiac trunk is a surgical emergency. Although a literature review disclosed no significant difference between outcomes after open surgery and radiologic arterial embolization, our experience in this case suggests immediate embolization during arteriography as the most effective treatment. Later, to prevent recurrence, the arcuate ligament should be surgically sectioned and the celiac artery stenosis treated. (J Vasc Surg 2004;39:906-11.)**

Anatomically, the pancreatic head is encircled by the arterial vessels that make up the peripheral pancreatic circulation. This arterial network comprises the anterior and posterior pancreaticoduodenal arcades. These arteries connect the superior mesenteric artery system to the celiac artery system. Aneurysms involving these arteries develop spontaneously or arise from an associated disorder or a specific hemodynamic condition. The first case of a pancreaticoduodenal artery aneurysm was reported in 1895 by Ferguson.[1] Since then, fewer than 100 cases have been reported in the literature.[2,3] These aneurysms account for less than 2% of all aneurysms in the visceral arteries.[4] Pancreaticoduodenal artery aneurysms produce a variety of clinical manifestations, ranging from hemorrhagic collapse to vague abdominal symptoms. Our case of a ruptured pancreaticoduodenal artery aneurysm associated with a celiac artery lesion illustrates the difficulty in diagnosing these rare events in time, and in instituting prompt treatment to arrest the bleeding and performing an elective procedure to prevent recurrence. To seek information that might guide treatment and diagnosis, we reviewed the relevant publications of true ruptured and nonruptured pancreaticoduodenal artery aneurysms associated with a celiac artery lesion.

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Competition of interest: none.

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MATERIAL AND METHODS

We conducted a search of the literature in an attempt to locate all case reports and clinical reviews of pancreaticoduodenal artery aneurysms, especially those with associated stenosis or obstruction of the celiac axis. We used a combination of medical subheadings and free text searches. Terms searched for included pseudoaneurysm, visceral artery, aneurysm, visceral aneurysm, ruptured aneurysm, gastroduodenal artery, pancreaticoduodenal artery, and peripancreatic artery, with arcuate ligament, median arcuate ligament division, celiac artery, celiac stenosis, celiac occlusion, celiac obstruction, and celiac axis, and these terms with or without retroperitoneal hemorrhage. Databases searched included MEDLINE from January 1966 to December 2002, EMBASE from 1980 to December 2002, and Database of Abstracts of Reviews.

From more than 100 relevant results collected, we considered for analysis, only reviews of pancreaticoduodenal artery aneurysms and case reports that described true aneurysms of the pancreaticoduodenal arteries associated with a celiac artery lesion.

CASE REPORT

A 54-year-old man (weight, 120 kg; height, 1.92 cm) with no specific history of disease was transferred to our vascular surgery unit at the Catholic Institute in Lille, France, after a McBurney operation to treat suspected appendicitis syndrome disclosed a voluminous nonbleeding retroperitoneal hematoma. A computed tomography (CT) scan obtained after contrast material injection revealed a large intact retroperitoneal hematoma, with no bleeding from the aorta or the main visceral arteries, and a median arcuate ligament division that compressed the origin of the celiac trunk

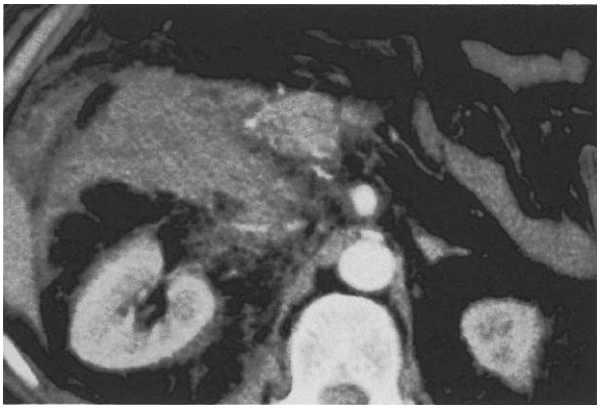

Fig 1. CT scan shows a large retroperitoneal hematoma and a median arcuate ligament division that compressed the origin of the celiac trunk.

(Fig 1). Because these findings suggested a ruptured pancreaticoduodenal artery aneurysm, arteriography was planned to confirm the diagnosis and treat the aneurysm with embolization. The patient, in normotension status, was kept under observation in the recovery room, then transferred to the vascular surgical unit.

On the following day a CT scan showed that the hematoma had enlarged. The patient was observed in our vascular unit, and arteriography was scheduled for the next day. During the night sudden hemodynamic collapse occurred, and the patient was promptly transferred to the radiology unit. While the patient was being prepared for radiovascular studies, a new CT scan showed that the hematoma had now increased in size and had spread to the intraperitoneal space, with perihepatic, perisplenic, and Douglas diffusion, providing evidence that the hematoma had ruptured into the peritoneum. The patient underwent selective aortic arteriography to visualize the stenosis caused by compression of the celiac axis, localize the bleeding pancreaticoduodenal artery aneurysm, and proceed to treatment with embolization. The arteriogram after the first contrast injection confirmed a tight stenosis involving the celiac trunk (Fig 2), and a particularly dense network of collateral vessels connecting the superior mesenteric artery to the celiac trunk. A selective superior mesenteric artery angiogram visualized the anterior and posterior pancreaticoduodenal arcades from the gastroduodenal artery. On the anterior arcade there was an aneurysm smaller than 6 mm. On the posterior arcade there was an aneurysmal malformation, with contrast leakage.(Fig 3) During the radiologic procedure the distal part of this malformation was embolized with two coils, and the proximal part of the malformation was then embolized with a single coil (Fig 4). These maneuvers achieved complete thrombosis of the malformation and the posterior pancreaticoduodenal arcade while preserving the gastroduodenal artery. The postoperative course was uneventful. A follow-up CT scan on day 6 showed a stable nonbleeding hematoma. Follow-up scans at 3 and 4 months showed that the hematoma had regressed.

Six months after the original operation the patient underwent surgery to decompress the celiac axis stenosis. Through a laparotomy over the umbilicus, the celiac trunk was decompressed by a single section of the large left pillar of the arcuate ligament.

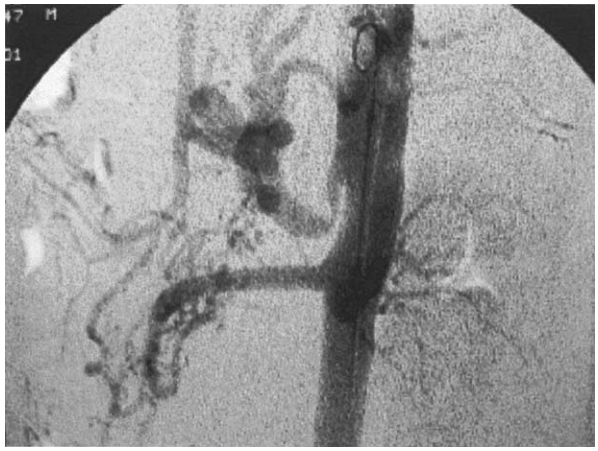

Fig 2. Global profile arteriogram shows stenosis of the celiac trunk and a particularly dense collateral arterial network connecting the superior mesenteric artery to the celiac trunk.

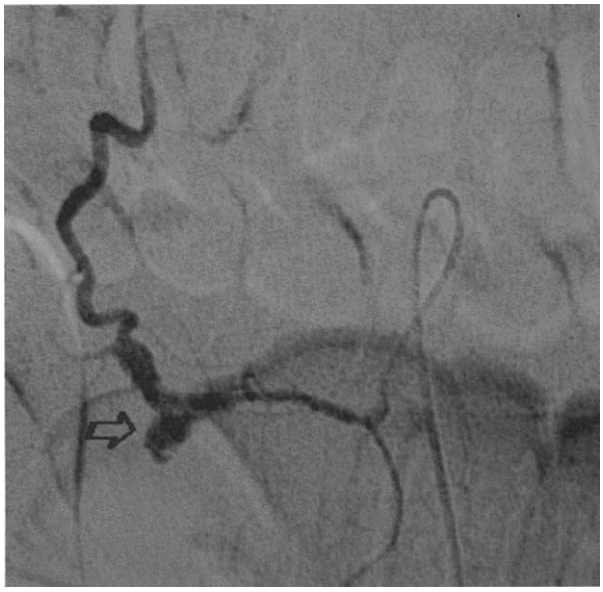

Fig 3. Selective catheterization of the posterior arcade shows an arterial malformation *(arrow)* with leakage of contrast material.

Palpation showed normal blood flow into the celiac axis, with satisfactory pulsation. Arteriography on postoperative day 3 confirmed that the celiac axis stenosis initially observed had regressed, and the aneurysmal malformation on the anterior pancreaticoduodenal arcade had disappeared (Fig 5). No contrast leaks were visible, nor were there signs of recurrent pancreaticoduodenal artery aneurysm. Short-term and mid-term follow up was uneventful.

DISCUSSION

The case we report is instructive for several reasons. First, true aneurysms of the pancreaticoduodenal artery associated with a celiac trunk lesion are extremely rare.

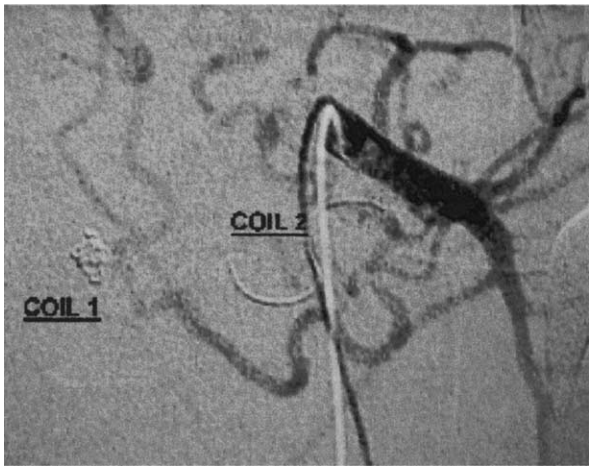

Fig 4. Complete thrombosis of the malformation (*coil 1,* accumulation of two coils) and the posterior pancreaticoduodenal artery after embolization of the proximal part *(coil 2)* and preservation of the gastroduodenal artery.

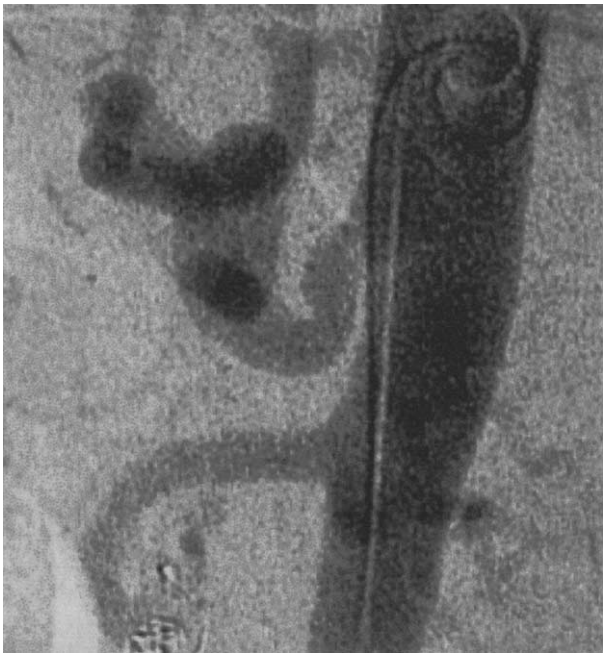

Fig 5. Postprocedural arteriogram after section of the median arcuate ligament confirms regression of the celiac axis stenosis initially observed (see Fig 2) and of the collateral network initially connecting the superior mesenteric artery and the celiac trunk.

Scarce information, mainly from more recent articles, is available on the causes, anatomy, presenting symptoms, and treatment.[2,3,5] Second, rupture develops slowly over 3 days after onset of symptoms. Third is the way the bleeding ruptured aneurysm was managed, and that surgical decompression of the celiac artery helped to prevent recurrence.

True aneurysms are often hard to differentiate from false aneurysms. Several findings suggested that our patient had a true aneurysm of the pancreaticoduodenal artery: the associated celiac trunk lesion, the absence of acute or chronic pancreatitis, and the dense arterial network connecting the superior mesenteric artery to the celiac artery. Conversely, false pancreaticoduodenal aneurysms, secondary to local injury, are principally observed with acute or chronic pancreatitis. The mechanism underlying their development involves biologic arterial erosion (by elastase and trypsin) or mechanical erosion (by pancreatic cysts or false cysts).[6] The incidence of false aneurysms is three to four times higher in men than in women; they develop on average at 45 years of age in patients with chronic alcoholism.[7,8] In as many as 10% of patients with chronic pancreatitis aneurysms are located on the pancreaticoduodenal arteries.[7] A pancreatic cause is implicated in 30% to 40% of cases.[2,9,10]

True aneurysms of the pancreaticoduodenal artery are especially rare. Various causes have been reported including arteriosclerosis, infection, fibrodysplasia,[11] and, principally, stenosis or occlusion of the celiac trunk. True aneurysms are found in patients with a wide age range (mean, 60 years.[2,3] They affect men and women equally. The relatively high average age at onset probably depends largely on underlying arteriosclerosis. Since Sutton and Lawton[12] in 1973 described a patient with a true aneurysm of the pancreaticoduodenal artery associated with a celiac trunk lesion, a celiac lesion is acknowledged as a major cause of development of an aneurysm of the pancreaticoduodenal artery. Its prevalence varies in different series. The first analysis and review of the literature in 1990[3] for this association demonstrated a prevalence of 68% of celiac lesions in true pancreaticoduodenal artery aneurysms. Later the same investigators, describing two further personal cases, reported a prevalence of 74%.[13] In 1999 other investigators reviewing 52 cases of pancreaticoduodenal artery aneurysms, excluding false aneurysms, reported that 63% were associated with a celiac trunk lesion.[5] To explain the association of a pancreaticoduodenal artery aneurysm with a celiac artery lesion, Sutton and Lawton[12] originally proposed that the increased blood flow in the peripancreatic arterial network provided collateral supply for revascularization of the celiac trunk, thus dilating the vascular walls until an aneurysm developed. Their hypothesis receives support from four reported cases that showed that simple revascularization of the celiac trunk with decompression, arcuate ligament section, or direct revascularization led to complete aneurysm regression at angiographic follow-up.[14-16] A radiologic study in 1991 designed to study the peripancreatic arterial network found peripancreatic artery dilatation in 80% of patients with celiac trunk compression and none of the control subjects.[17] The incidence of true pancreaticoduodenal artery aneurysms has been estimated at 3% by some,[18] and at 18% by others, in patients with median arcuate ligament division compressing the celiac trunk,[19] as in our patient.

Unlike previous reviews, our literature review focused on true aneurysms of the pancreaticoduodenal artery associated with a celiac trunk lesion. Considering a celiac trunk

Table I. Published cases of aneurysms of pancreaticoduodenal arteries

Author	*Date*	*No. of cases*	*Status*	*Treatment*			*Follow-up*
				Absence	*Surgical embolization*	*Revascularization alone*	
Sutton et al[12]	1973	2	NR	+			Lost
			NR		+		Good
Mora et al[15]	1976	2	NR			+	Good
			NR			+	Good
Scheflan et al[23]	1977	1	R		+		Good
Kadir et al[24]	1978	4	R (n = 1)		+		Dead
			NR (n = 3)		+		Good
Proud et al[16]	1978	1	NR			+	Good
Verminck et al[25]	1979	1	NR		+		Good
Mariano et al[26]	1981	1	R		+		Good
Vernhet et al[27]	1982	2	R		+		Good
Thevenet et al[28]	1983	2	NR		+		Good
Cangahar et al[30]	1985	1	R		+		Good
Partensky et al[29]	1987	1	NR		+		Good
Ambrosseti et al[31]	1987	2	R		+		Good (n = 1)
							Dead (n = 1)
Wrazidlo et al[32]	1987	1	NR		+		Good
Bersani et al[33]	1989	1	NR		+		Good
Quandalle et al[13]	1990	2	R (n = 1)	+			Dead
			NR (n = 1)		+		Good
Shibahara et al[34]	1993	1	R		+		Good
Suzuki et al[35]	1998	2	NR		+		Good
Nagano et al[14]	1997	1	NR		+		Good
Imamura et al[36]	1998	1	NR		+		Good
De Perrot et al[15]	1999	5	NR (n = 5)		+ (n = 3)		Good (n = 2)
							Dead (n = 1)
					+ (n = 2)		Good (n = 2)
Scesa et al[37]	2000	1	NR		+		Good
Ogino et al[38]	2002	1	R		+		Good
Current report	2003	1	R		+		Good

R, Ruptured; *NR*, nonruptured.

lesion as the major etiologic factor for development of a pancreaticoduodenal artery aneurysm, we analyzed the data for this type of aneurysm separately, discarding all other more exceptional causes for this localization (infection,[20,21] fibrodysplasia,[22] undetermined). The current literature consists of 23 publications that describe 36 aneurysms of the pancreaticoduodenal artery associated with stenosis or obstruction of the celiac axis[3,5,12,14-16,23-28] (Table). Our case report brings the number to 37.

Our patient had a relatively small aneurysm, 8 mm in diameter. Most pancreaticoduodenal artery aneurysms range in diameter from 8 to 30 mm (average, 9 mm).[5] Our review suggests that size is not a factor for rupture; all nonruptured aneurysms measured more than 10 mm in diameter, whereas most ruptured aneurysms measured less than 10 mm. The anatomic data from our review imply that some of these smaller aneurysms (17%) were multiple lesions.

Aneurysms of the inferior pancreaticoduodenal arteries vary in clinical presentation, and rupture is still the major complication. The frequency of rupture varies from 52%[3] to 69%.[13] Our review found a lower frequency: 30% (11 of 37) ruptured aneurysms and 70% (26 of 37) nonruptured aneurysms. Most ruptured aneurysms manifest clinically with nonspecific abdominal pain,[3,23,24,26,27,30,31,34,38] and in a few cases there is an acute abdominal syndrome associated with bleeding into the peritoneal cavity, and ultimately hemorrhagic collapse. These aneurysms usually rupture into the retroperitoneal space around the pancreas. More rarely, if treatment is delayed, as in our patient and in two reported cases, the aneurysm may ultimately rupture into the peritoneal cavity,[26,27] or, exceptionally, into the digestive tract, the duodenum,[24] or the Wirsung canal.[30]

Most nonruptured aneurysms[3,12,14-16,24,25,28,29,32,33,35-37] are discovered by chance, in particular during abdominal echography to investigate nonspecific abdominal pain.[36]

In our patient, as the first diagnostic step to identify the exact site of bleeding, we obtained a contrast-enhanced abdominal CT scan. With a ruptured aneurysm, CT scans invariably demonstrate the peripancreatic hematoma and enable measurement of the precise size of the collection. Contrast-enhanced CT scans also demonstrate eventual bleeding into the peritoneal cavity and show whether the aneurysm bleeding is persistent. CT scans enabled diagnosis of our patient's associated lesion of the celiac trunk, confirming this exceptional association and indicating the need for arteriography to confirm the provisional diagnosis, search for the cause, and guide treatment.

As our case report emphasizes, in a patient with a bleeding ruptured pancreaticoduodenal artery aneurysm, arteriography must be performed without delay. The investigation should begin with an adjunctive profile aortic injection to visualize and diagnose the celiac lesion. Selective superior mesenteric artery angiography will then visualize the collateral arterial network that is revascularizing the celiac branches, locate the aneurysmal formation, and specify the number of lesions. Finally, this imaging procedure localizes the bleeding from the ruptured aneurysm and, most important, visualizes the aneurysm and its feeding artery, thus enabling immediate radiologic embolization. In our patient, these procedures (Figs 2-4) confirmed the diagnosis and guided management, and recourse to surgery was avoided.

Our literature review showed that the current management of pancreaticoduodenal artery aneurysm consists of no treatment, surgery, embolization therapy, and treatment of the celiac trunk lesion. No treatment is an exceptional option, reported only twice (2 of 37)[3,12] for nonruptured aneurysms: in one case the patient refused therapy; in the second case there was life-threatening risk in a patient with cirrhosis, who died within a few days, of hepatic insufficiency.

During the 20 years covered in our review, when diagnostic and therapeutic imaging procedures were only beginning to be used, surgery was the most used technique (23 of 37), although since 1999 no new cases have been reported. The surgical procedure involved resection (14 of 23) or simple exclusion of the aneurysm from the circulation (6 of 23). The most common difficulty encountered in surgical treatment of a ruptured aneurysm arises when dissection shows that pancreatic dilacerations related to the hematoma necessitate duodenocephalic pancreatectomy. This technique was reported three times in our review (3 of 23). Surgery led to three deaths, all in patients with ruptured aneurysms: one after resection, one after aneurysm exclusion, and one after duodenocephalic pancreatectomy.

With the imaging techniques available today, the current reference standard for all hemorrhagic syndromes related to ruptured aneurysms and false aneurysms of the visceral arteries is radiologic treatment with embolization. In our review, this treatment was used in 8 of 37 of the most recent cases reported.[37,38] This technique is classically performed during the initial diagnostic arteriography. The main difficulty consists in avoiding coil migration and gastroduodenal or hepatic artery occlusion. In our review, all embolization procedures were successful, with no complications (six procedures to treat ruptured aneurysms, two procedures to treat nonruptured aneurysms). The failure rate for false aneurysm embolization in chronic pancreatitis was less than 20%.[39] As emphasized in a review published in 1990, including 39 aneurysms of the pancreaticoduodenal artery, associated or not with celiac trunk lesions,[3] if surgery or embolization fails, as they did in three cases each, neither technique is exclusive, because surgery can be followed by embolization, and embolization can be followed by surgery.

No epidemiologic studies are available to show whether nonruptured aneurysms of the pancreaticoduodenal artery should be treated or left alone. Arguments in favor of systematic treatment include the severity of rupture, with life-threatening risk, and especially the absence of a known relation between the size of the aneurysm and the risk for rupture. Inasmuch as patients necessarily undergo arteriography, a less aggressive procedure than surgery, with a good success rate, systematic treatment with embolization during arteriography, seems to be the treatment of choice.

In patients with pancreaticoduodenal artery aneurysms caused by a lesion of the celiac trunk, good management depends on resolution of the lesion surgically and prevention of recurrence. In our patient this was accomplished with a simple section of the median arcuate ligament. Our review identified no reported cases of endovascular treatment of celiac trunk stenosis. In agreement with the current literature, in the presence of occlusion of the celiac trunk, after an isolated aneurysm has been treated at surgery or embolization, direct celiac or hepatic artery bypass for revascularization seems an unnecessarily risky procedure in the absence of an associated multianeurysmal disorder.

In contrast to other reviews, we analyzed the results of treatment of ruptured aneurysms associated with a celiac trunk lesion, because mortality from nonruptured aneurysms in the series we reviewed was nil. The estimated mortality rate for aneurysms of the pancreaticoduodenal artery is 17.6% in the review conducted by Quandalle et al[3] in 1990 and 17% in the recent review by Coll et al[2] in 1998. In our review, which considered only aneurysms of the pancreaticoduodenal artery associated with a celiac lesion, mortality was 18.75% (three deaths in 23 treatments). In a large review of aneurysms of the pancreaticoduodenal artery of various causes, Coll et al[2] recently differentiated between cases reported before 1980 without embolization and those reported since 1980 treated surgically or with embolization. Mortality was lower in the most recently treated group. In this group of patients treated since 1980, embolization in 13 patients led to no deaths, whereas surgery in 15 patients led to five deaths. In our review, considering only ruptured aneurysms associated with a celiac lesion, 6 patients were treated with embolization, with no deaths, and no patient treated with embolization required repeat embolization or further surgery. Our review clearly suggests that, for ruptured aneurysms, mortality is higher after surgery than after embolization. Our case report and literature review nevertheless underline that embolization will generally resolve a hemorrhagic syndrome without major complications, has an excellent success rate, and the initial diagnostic arteriographic procedure combined with therapy is less aggressive for the patient than surgery.

CONCLUSION

Aneurysm of the pancreaticoduodenal arteries associated with a lesion of the celiac trunk is an exceptional event. A ruptured aneurysm is still an emergency, an requires prompt management. Our case report and literature review

suggest immediate arteriography to confirm the cause, establish the diagnosis, and enable nonsurgical treatment with embolization. Patients with stenosis of the celiac trunk caused by median arcuate ligament compression must then undergo elective surgical decompression to prevent the risk for recurrent aneurysm.

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