Management of True Aneurysms of the Pancreaticoduodenal Arteries

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Objective

To review the authors' recent experience and that of the literature since 1973 and to provide management guidelines for true aneurysms of the pancreaticoduodenal arteries (PDA).

Summary Background Data

True aneurysms of the PDA are rare, with a total of only 52 cases reported since 1973.

Methods

Six patients were admitted to the authors' institution between 1985 and 1995 for rupture of a true aneurysm of the PDA. They were analyzed with regard to the mode of presentation, preoperative workup, management, and outcome.

Results

All patients had severe epigastric pain from retroperitoneal hemorrhage. Computed tomography scanning and angiography were performed in all cases. Aneurysms ranged from 0.7

to 1.2 cm (median 0.9 cm). The celiac axis was stenotic or occluded in five cases. Three patients underwent emergency pancreatoduodenectomy. Two of them survived. In one case, section of the median arcuate ligament was associated with the procedure, and the patient died from an aortic dissection. Embolization was performed in the last three patients. The procedure was definitive in two cases. In one, hemorrhage recurred 8 days later and required surgical ligation of the bleeding artery.

Conclusions

The authors recommend rapid treatment of all true aneurysms of the PDA. Because most of these aneurysms result from a stenosis of the celiac axis, selective embolization may help to preserve patency of the PDA and should, therefore, be the primary therapeutic choice in ruptured aneurysms. Close follow-up is mandatory because of possible recurrent bleeding. Appropriate and expeditious management of true PDA aneurysms should help reduce the mortality rate.

Aneurysms of the pancreaticoduodenal arteries (PDA) are rare and account for only 2% of all splanchnic artery aneurysms.¹ Although some PDA aneurysms cause persistent abdominal discomfort, leading to early diagnosis, 64% of reported patients presented after rupture, thus carrying a high mortality rate.² The increasing use of visceral angiography, and more recently the introduction of transcatheter arterial embolization, has helped to reduce the mortality rate.^{3,4} However, true and false PDA aneurysms should be distinguished: the latter result from pancreatitis, abdominal trauma, or septic emboli and often rupture into the gastro-intestinal tract, whereas the former are frequently associated

with a stenosis of the celiac axis and rupture into the retroperitoneal space. 1,5,6

To better analyze the outcome of patients with true aneurysm of the PDA and to provide management guidelines, we report our recent experience with six cases of rupture of true PDA aneurysms and review the experience with this uncommon entity reported in the literature since 1973.

PATIENTS AND METHODS

Computerized records of all patients admitted to our institution between 1985 and 1995 for a ruptured PDA aneurysm were reviewed. Aneurysms associated with a history of trauma, endocarditis, or pancreatitis were considered pseudoaneurysms and were excluded from the study. Six patients were identified, four men and two women, with a median age of 64 years (range 58 to 73). One patient had a history of systemic hypertension and atherosclerosis of the

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Figure 1. Patient 1. Abdominal CT scan showing a voluminous, highdensity paraduodenal fluid collection consisting of a recent retroperitoneal hematoma.

aorta, and two patients had had partial gastrectomy 26 and 35 years previously for duodenal ulcer. Neither of them had pancreatic or hepatic disease. Aneurysm of the PDA had not been suspected before rupture in any of these patients. All cases were analyzed with regard to the mode of presentation, preoperative workup, management, and outcome.

RESULTS

All patients had sudden, severe epigastric pain. Symptomatology had started 3 to 14 hours (median 9.5 hours) before hospital admission in five patients, and had lasted 7 days in another. On admission, five patients were hemodynamically stable and one required immediate resuscitation. Physical examination revealed a diffusely tender abdomen in all patients, with a nonpulsatile mass in the right upper quadrant in one of them.

Computed tomography (CT) scan and angiography were performed in all cases. CT revealed a voluminous retroperitoneal hematoma (Fig. 1), diffusing into the peritoneal cavity in three patients. Aneurysms of the PDA were suspected on contrast-enhanced CT scan in two cases. On angiography, aneurysms ranged from 0.7 to 1.2 cm (median 0.9 cm), and two of them were actively bleeding. In one case, a tortuous and dysplatic aspect of the anterior inferior PDA was associated with a posterior inferior PDA aneurysm, and in another a simultaneous asymptomatic splenic artery aneurysm of <1 cm was seen. The celiac axis was occluded in two cases, stenotic in three cases, and normal in one case. In the latter, however, occlusion of the gastroduodenal artery, resulting from previous gastric surgery, was observed. All stenoses of the celiac axis resulted from extrinsic compression from the median arcuate ligament and were associated with a retrograde flow in the gastroduodenal artery (Fig. 2).

Management consisted of volemic resuscitation followed by transcatheter arterial embolization and/or expeditious surgery (Table 1). Three patients underwent laparotomy immediately after angiography. In one of them, a temporary embolization with a large absorbable gelatin sponge (Gelfoam) had been performed to stabilize the patient. Ligation of the bleeding artery could not be achieved because of the massive retropancreatic hematoma, and pancreatoduodenectomy with (n = 1) or without (n = 2) preservation of the pylorus was performed. Postoperative course was uneventful in two patients, except for a transitory and moderate elevation of transaminases. In one case, however, section of the median arcuate ligament was performed in addition to the pancreatic resection, and the patient remained hypotensive, anuric, and acidotic after surgery. A repeat angiogram revealed aortic dissection, obstructing the celiac axis and the superior mesenteric artery. Aortic dissection could have resulted from a short trial clamping of the aorta (of a few seconds only) at the beginning of surgery. The patient underwent a repeat operation with venous bypass of the celiac axis, right colectomy, and left hepatic lobectomy, but died 4 days later. At necropsy, a medionecrosis of the aorta and a fresh thrombosis of the celiac axis were found.

Histologic examination of all resected aneurysms showed no abnormality, looking in particular for atherosclerosis and congenital dysplasia.

Coaxial catheterization and embolization with microcoils was performed in the last three patients (Fig. 3). Two of these procedures were successful. However, hemorrhage recurred 8 days later in one patient and required emergency



Figure 2. Patient 3. Angiography of the superior mesenteric artery showing a retrograde flow in the gastroduodenal artery with vascularization of the celiac territory. Note the small aneurysm of one of the PDAs.

Patient	Year	Size of Aneurysm	Celiac Axis Stenosis	Embolization	Surgery	Associated Procedure	Outcome
1	1985	1.2cm	Yes	_	Pancreatoduodenectomy	_	Alive
2	1986	0.9cm	Yes	Gelfoam	Pancreatoduodenectomy	Section of arcuate ligament	Dead
3	1990	0.8cm	Yes	_	Pancreatoduodenectomy	_	Alive
4	1991	0.8cm	Yes	Microcoils	Ligation of PDA for recurrent bleeding	_	Alive
5	1993	1 cm	Yes	Microcoils		-	Alive
6	1995	0.7cm	No*	Microcoils	_	-	Alive

Table 1. PATIENTS WITH RUPTURE OF A TRUE ANEURYSM OF THE PDA IN OUR INSTITUTION

surgery. Hemostasis was eventually obtained by ligation of the bleeding artery. Particular care was taken not to damage the superior mesenteric artery and the collateral circulation to the celiac territory.

A total of 68 units of blood were transfused, ranging from 2 to 27 (median 10) units per patient. No further hemorrhage were observed after a median follow-up of 4 years (range 2 to 9).

DISCUSSION

The incidence of true aneurysms of the PDA is extremely low but remains unknown because no large series have been published yet. A comprehensive review of the literature of the past 25 years yielded a total of 52 cases of true aneurysms of the PDA. Their characteristics are summarized in Table 2 (a complete list of references is available from the authors).

True aneurysms of splanchnic arteries are of particular interest because in contrast to large-vessel aneurysms, atherosclerosis may not be the primary causative factor. Evidence suggests that local hemodynamic events play an important role in the development of most of these aneurysms. Multiparity and portal hypertension have both been associated with an increased flow in the splenic artery, resulting in an increased flow in the splenic artery, resulting in an increased incidence of such aneurysms.⁷ Similarly, Sutton and Lawton⁸ postulated that stenosis of the celiac axis resulted in an increased flow through the PDA, which favored the development of PDA aneurysms. Hence, since their report in 1973, 33 PDA aneurysms have been reported in association with a stenosis of the celiac axis, whereas only 9 were reported with local atherosclerotic lesions. Other etiologies of true PDA aneurysms have been reported in a few cases and include congenital aberrations of the vessel wall⁹ or medial fibrodysplasia.¹⁰

In contrast to pseudoaneurysms,⁵ true aneurysms of the PDA rarely present with gastrointestinal hemorrhage. They usually rupture into the retroperitoneal space and cause acute abdominal pain, which may simulate gastroduodenal,





Figure 3. Patient 6. (A) Small aneurysm of one of the PDAs was recognized by angiography of the superior mesenteric artery. Note the occlusion of the gastroduodenal artery from previous gastric surgery. (B) Microcoil embolization of the aneurysm was subsequently performed.

Table	2. SU	MMAR	Y OF	REPORTED	
CASES	WITH	TRUE	PDA	ANEURYSM	
SINCE 1973*					

	Rupture (n = 27)	Nonrupture (n = 25)
Presentation		
Acute abdominal pain	19	_
Gastrointestinal bleeding	6	1†
Obstructing jaundice	1	2†
Persistent abdominal pain	1	8
Rim of calcification	NS	9
Bruits or pulsatile mass	NS	8
Incidental finding	_	10
(US/CT/angiography/surgery)		
Size		
<1 cm	0	1
1–1.5 cm	4	6
1.6–2 cm	4	5
>2 cm	5	11
NS	14	2
Possible etiology		
CA stenosis	13	20
Characteristics of the		
aneurysm:		
Atherosclerosis	4	5
Congenital anomaly	0	2
Fibrodysplasia	0	1
Treatment		
Surgery	20	17
Embolization	3	1
Dead before treatment	3	_
No treatment	1	7
Mortality rate		
Total (%)	26%	4%†
Operative (%)	20%	0%

CA, celiac axis; PDA, pancreaticoduodenal arteries; NS, not specified.

* The 6 patients reported in Table 1 are not included.

† Not related to the PDA aneurysm.

biliary, or pancreatic disease. The pain is usually intense and brings the patient to the hospital within 24 hours. Hemorrhage is initially contained by the retroperitoneal space, allowing time for an adequate diagnostic workup. However, this time may be short, because patients may rapidly become unstable and require emergency laparotomy.¹¹ Therefore, early angiography and rapid control of the hemorrhage are mandatory if PDA aneurysm is suspected.

Although Iyomasa et al² reported 19 pseudoaneurysms of the PDA, of which 16 were ruptured, we found that 48% of true aneurysms of the PDA were diagnosed before rupture. In a few patients, aneurysms were symptomatic by compressing the superior mesenteric artery or obstructing the common bile duct.^{12,13} Occasionally, a rim of calcification has been noted on a plain abdominal film and has led to early diagnosis.¹¹ However, unlike hepatic, splenic, or renal artery aneurysms, this is a rare feature and was not observed in any of our patients.¹⁴ Although most asymptomatic aneurysms of the PDA were incidentally discovered during angiography, contrast-enhanced CT scanning is an excellent diagnostic tool. It could localize aneurysms of <1 cm in our series. Hence, in the future the number of asymptomatic PDA aneurysms will certainly increase with the use of contrast-enhanced CT scanning.

Unlike true aneurysms of the splenic artery, which rarely rupture when they are <2 cm,⁷ true aneurysms of the PDA have not shown a clear correlation between size and propensity to rupture. In the literature, at least eight patients bled from aneurysms <2 cm, and in our experience four aneurysms were <1 cm and six were <1.5 cm when rupture occurred. Therefore, we recommend rapid treatment of all true aneurysms of the PDA, whatever their size, at the time of diagnosis.

Although it is tempting to bypass visceral angiography in favor of prompt surgical exploration when the patient is unstable, every attempt should be made to define the aneurysm before surgery. Aneurysms of the PDA are often located behind or within the parenchyma of the pancreas, and their detection at operation may fail in about 70% of cases.¹⁵ In addition, the patency of other visceral arteries and knowledge of the collateral flow is crucial to manage these aneurysms by embolization and/or surgery.⁶

Transcatheter arterial embolization of true aneurysms of the PDA has been reported in a few cases only.^{4,16,17,18} In this series, embolization of true PDA aneurysms was performed in four patients. In one case, the procedure was temporary, stabilizing the patient before surgery; in the three most recent patients, embolization was considered a definitive option. The advent of newer coaxial catheterization techniques has greatly improved embolization of small tortuous vessels. Therefore, selective embolization of branches of the pancreaticoduodenal arcades may preserve patency of the remaining PDA and, thus, preserve vascularization of the celiac territory. However, embolization is not always technically feasible because of the difficulty of selectively cannulating the feeding vessel of the aneurysm.¹⁷ In addition, embolization may be associated with aneurysmal rupture during the procedure or ischemic injury resulting from the absence of major collateral vessels.^{11,16,18} Although recurrent hemorrhage is less likely in true aneurysms than in pseudoaneurysms, which are usually associated with persistent pancreatic inflammation, such complications may still occur, and close observation of the patient is mandatory.

Surgical management usually involved ligation or resection of the PDA aneurysms (Table 3). However, in the present series, pancreatoduodenectomy had to be performed in three patients because the aneurysm could not be visualized consequent to the massive retroperitoneal hematoma. Before resecting the head of the pancreas in patients with a stenotic celiac axis, a trial clamping of the gastroduodenal artery can help determine if revascularization of the celiac territory is needed. Occasionally, measurement of the pressure gradient between the aorta and the hepatic artery is also

Table 3. SURGICAL PROCEDURES FOR TRUE PDA ANEURYSMS SINCE 1973*

	Rupture (n = 20)	Nonrupture (n = 17)
Section of arcuate ligament	0	2
Ligation of aneurysm	11	0
Resection of aneurysm	5	9
Reanastomosis PDA and SMA	2	4
Aortohepatic bypass	0	2
Section of arcuate ligament	1	0
Intraoperative death	1	0

PDA, pancreaticoduodenal arteries; SMA, superior mesenteric artery. * The 6 patients reported in Table 1 are not included.

helpful.⁶ Revascularization of the celiac territory, with section of the median arcuate ligament, reanastomosis of the PDA to the superior mesenteric artery, or through an aortohepatic bypass, has been reported in 11 patients over the past 25 years. Interestingly, in two cases, section of the median arcuate ligament and celiac axis revascularization entailed spontaneous resorption of the PDA aneurysms on follow-up angiography, probably by thrombosis of the aneurysmal sac.^{19,20}

Only one patient (17%) died in our series of ruptured true PDA aneurysms. This low mortality rate may result from early aggressive management, the use of preoperative angiography in all patients, and speedy treatment with embolization and/or surgery. Hence, appropriate and expeditious management of true PDA aneurysms should help reduce the mortality rate.

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