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Outcomes After Open and Endovascular Repair of Non-Ruptured True Pancreaticoduodenal and Gastroduodenal Artery Aneurysms Associated with Coeliac Artery Compression: A Multicentre Retrospective Study

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WHAT THIS PAPER ADDS

Aneurysms of the pancreaticoduodenal arcades including the gastroduodenal artery (PDAAs) are rare, and best treatment evidence is lacking. This multicentre study suggests that in patients with PDAAs and compression of the coeliac axis (CA) by a median arcuate ligament (MAL), the choice between transcatheter embolisation or open exclusion of PDAAs should be tailored according to the location of the aneurysm, favouring open surgical repair for aneurysms located on the gastroduodenal and anterior pancreaticoduodenal arteries and embolisation for aneurysms located on the posterior pancreaticoduodenal artery. This study also demonstrates that in this setting CA stenting should be avoided.

Objective: True aneurysms of the peri-pancreatic arcade (PDAA) have been attributed to increased collateral flow related to coeliac axis (CA) occlusion by a median arcuate ligament (MAL). Although PDAA exclusion is currently recommended, simultaneous CA release and the technique to be used are debated. The aim of this retrospective multicentre study was to compare the results of open surgical repair of true non-ruptured PDAA with release or CA bypass (group A) *vs.* coil embolisation of PDAA and CA stenting or laparoscopic release (group B).

Methods: From January 1994 to February 2019, 57 consecutive patients (group A: 31 patients; group B: 26 patients), including 35 (61%) men (mean age 56 ± 11 years), were treated at three centres. Twenty-six patients (46%) presented with non-specific abdominal pain: 15 (48%) in group A and 11 (42%) in group B ($p = .80$).

Results: No patient died during the post-operative period. At 30 days, all PDAAs following open repair and embolisation had been treated successfully. In group A, all CAs treated by MAL release or bypass were patent. In group B, 2/12 CA stentings failed at < 48 hours, and all MAL released by laparoscopy were successful. Median length of hospital stay was significantly greater in group A than in group B (5 *vs.* 3 days; $p = .001$). In group A, all PDAAs remained excluded. In group B, three PDAA recanalisations following embolisation were treated successfully (two redo embolisations and one open surgical resection). At six years, Kaplan—Meier estimates of freedom for PDAA recanalisation were 100% in group A, and 88% ± 6% in group B ($p = .082$). No PDAA ruptured during follow up. In group A, all 37 CAs treated by MAL release were patent, and one aortohepatic bypass occluded. In group B, five CAs occluded: four after stenting and the other after laparoscopic MAL release with two redo stenting and three aortohepatic bypasses. Estimates of freedom from CA restenosis/occlusion were 95% ± 3% for MAL release or visceral bypass, and 60% ± 9% for CA stenting ($p = .001$). Two late restenoses following CA stenting were associated with PDAA recanalisation.

Conclusion: Current data suggest that open and endovascular treatment of PDAA can be performed with excellent post-operative results in both groups. However, PDAA embolisation was associated with few midterm recanalisations and CA stenting with a significant number of early and midterm failures.

Keywords: Embolisation, Median arcuate ligament, Pancreaticoduodenal artery, Visceral aneurysm
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INTRODUCTION

Aneurysms of the peri-pancreatic arteries, including the gastroduodenal artery and its pancreaticoduodenal branches (PDAA), account for only 2% — 3.5% of all visceral

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aneurysms.[1–3] They occur as either true or pseudoaneurysms secondary to pancreatitis or trauma. This study was limited to true PDAAs (Fig. 1) associated with coeliac axis (CA) stenosis by a median arcuate ligament (MAL).[4–6]

Despite their low incidence, these aneurysms deserve attention because, compared with other visceral aneurysms, they rupture at a disproportionate rate at a diameter sometimes < 10 mm.[2,7,8] For this reason, irrespective of size, prompt treatment is indicated.[2,9] For emergency cases, endovascular PDAA embolisation is the preferred technique.[8] However, with the widespread adoption of endovascular techniques, many intact PDAAs associated with CA stenosis are now treated electively by coil embolisation without CA release, or CA release by laparoscopy or CA stenting.[10–12] The authors of this study, who are accustomed to performing both open and endovascular techniques for visceral aneurysm repair, have different opinions regarding these techniques. Although all have agreed to perform transcatheter embolisation in the event of PDAA rupture, views on the management of unruptured PDA have been more divided. The literature on this aspect is not particularly helpful, as it consists essentially of case reports or small heterogeneous series mixing true PDAAs with false or ruptured aneurysms, and characterised by poor follow up, and lack of granularity. Hence, the authors of this report have pooled data from their centres to compare the results of open surgical repair of true PDAA with CA release or bypass *vs.* PDAA embolisation with laparoscopic release of MAL or CA stenting.

METHODS

From January 1994 to February 2019, 57 consecutive patients, 35 (61%) of whom were men, with a mean age of 56 $\pm$ 11 years presenting with an unruptured true PDAA associated with CA stenosis by MAL were analysed retrospectively in three academic centres: one in Italy (University of Rome, La Sapienza) and two in France (University Hospital of Poitiers and University Hospital of Toulouse). Each hospital's Institutional Review Board approved the study. Owing to its retrospective nature, patient consent was waived and, according to European Union regulations, the database was de-identified.

For the purposes of the analysis, the patients were divided into two groups. In group A, 31 patients underwent open surgical exclusion of PDAA followed by MAL release or CA revascularisation. In group B, 26 patients underwent endovascular repair with PDAA embolisation with laparoscopic release of MAL or CA stenting.

Pre-operative workup consisted of an abdominal ultrasound study and computed tomography angiography (CTA)

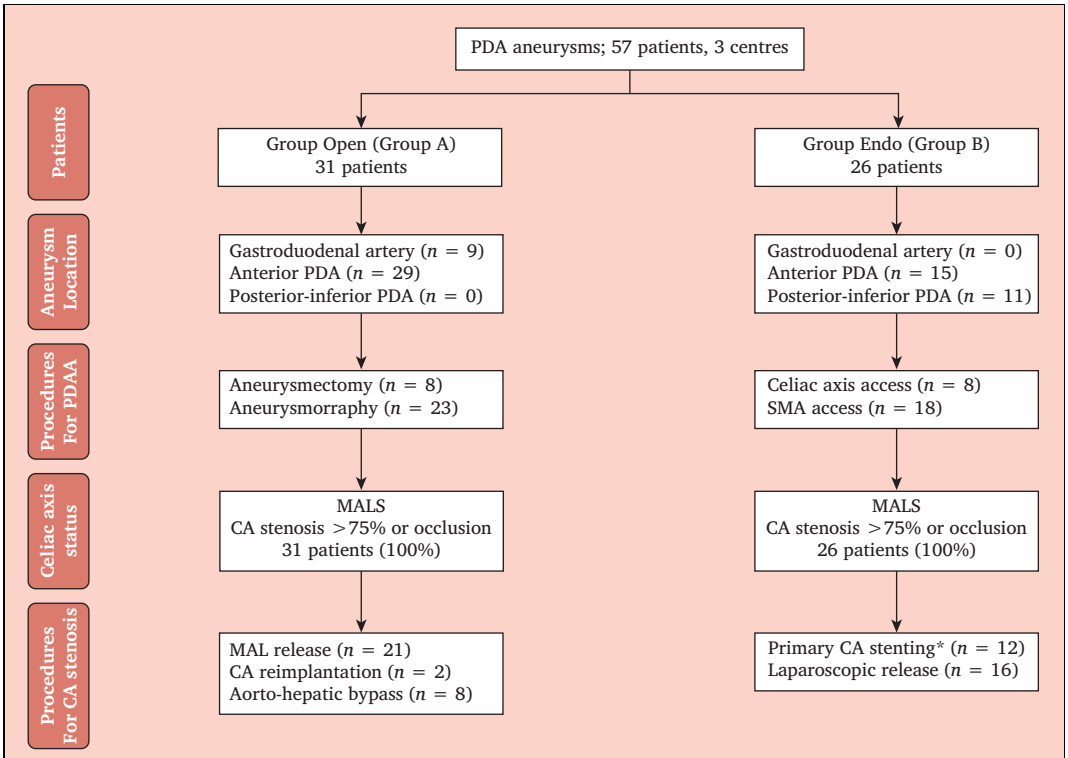

Figure 1. Flowchart of procedures for true pancreaticoduodenal artery (PDA) aneurysms (PDAA) associated with coeliac axis (CA) stenosis or occlusion in 57 consecutive patients treated between January 1994 and February 2019. In group A, 31 patients received open PDAA repair. In group B (26 patients), PDAA embolisation was associated with CA stenting in 12 patients and laparoscopic release of median arcuate ligament (MAL) in 16 patients. *Two patients with immediate failure of CA stenting received secondary laparoscopic MAL release.

with multiplanar and three dimensional reconstructions associated with a search for focal narrowing of the CA with retrograde filling of the pancreaticoduodenal arteries (Fig. 2). In all cases, pre-operative duplex ultrasound (DUS) evaluated the CA for MAL with inspiratory and expiratory data, which were positive in all cases.

In group A ($n = 31$), a surgical approach was made through bilateral subcostal incision with median extension toward the xiphoid process or by midline laparotomy. Depending on its size and location (Fig. 3), the PDAA was either resected (aneurysmectomy) with an eventual end to end arterial reconstruction or excluded (aneurysmorrhaphy; Fig. 4). MAL release was performed in 21 patients after gaining control of the coeliac aorta through section of the right crus of the diaphragm (Fig. 5). In the event of fibrosis, or occlusion, and depending on the extent of CA lesions, re-implantation of the CA into the coeliac aorta or revascularisation by an aortohepatic bypass was performed. No peri-pancreatic drainage was carried out.

In group B ($n = 26$), PDAA coil embolisation was achieved with different approaches: percutaneous transfemoral, or transbrachial with coeliac or superior mesenteric artery (SMA) catheterisation. No radial access was used in this series. Catheter configurations consisted primarily of a sheath guiding catheter, a 4 F catheter, and a microcatheter to achieve exclusion of inflow and outflow vessels, and to reduce the risk of PDAA recanalisation. According to surgeon preference, embolisation was followed by laparoscopic MAL release of or primary CA stenting. For primary stenting, a 4 F Cobra catheter and a 0.035 inch stiff guidewire were used. Angioplasty was achieved by a balloon catheter, followed by deployment of a balloon expandable stent. Laparoscopic MAL release was performed by collaboration with a surgeon familiar with laparoscopic techniques and according to the usual technique described elsewhere.[13]

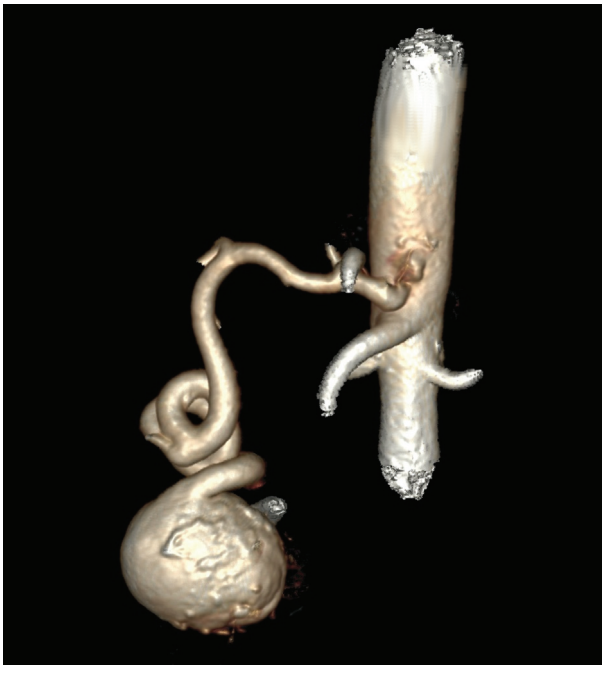

Figure 2. Computed tomography angiography of the aorta and visceral arteries of a patient in a series showing aneurysm of the anterior pancreaticoduodenal artery associated with proximal stenosis of the coeliac axis due to compression of the median arcuate ligament.

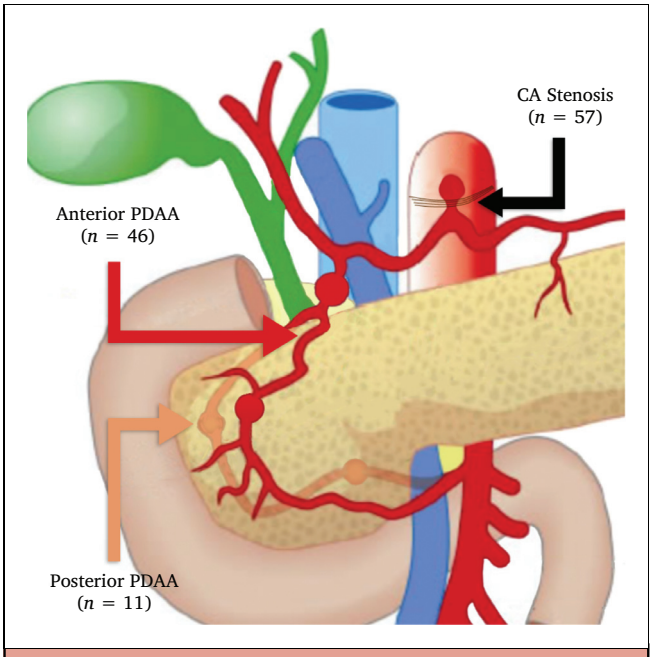

Figure 3. Schematic representation of the anatomy of the peri-pancreatic arteries in the 57 patients with pancreaticoduodenal aneurysms (PDAA) associated with coeliac axis (CA) stenosis or occlusion. Two aneurysms were located on the gastroduodenal artery, 44 on the anterior pancreaticoduodenal artery, and 11 at different locations on the posterior pancreaticoduodenal artery.

DUS of the pancreaticoduodenal arteries was performed prior to discharge in all patients. During follow up, all patients were seen on an outpatient basis, at one month and yearly thereafter, with aortic and the visceral artery DUS followed by CTA or preferably MRA after PDAA catheter embolisation if abnormal.

The primary study endpoints were post-operative mortality and morbidity, late PDAA recurrence, and re-operation. Secondary endpoints were post-operative hospital stay and late survival.

In each group, the following parameters were recorded: demographics; symptoms; imaging findings (DUS and CTA); site of PDAA; type of surgery; endovascular access route; SMA or CA catheterisation; embolisation; MAL release; visceral artery revascularisation; any revision; PDAA reperfusion; CA restenosis; morbidity; mortality rate; days in hospital; and median follow up time. All study data were cross checked and independently analysed by the clinical research department of the University of Poitiers.

The study was conducted according to the STROBE recommendations (see Supplementary Material). Univariable analysis was performed using *t* tests for normally distributed continuous variables and Mann—Whitney rank test for non-normally distributed variables with median and interquartile range (IQR). Fisher's exact test was used for

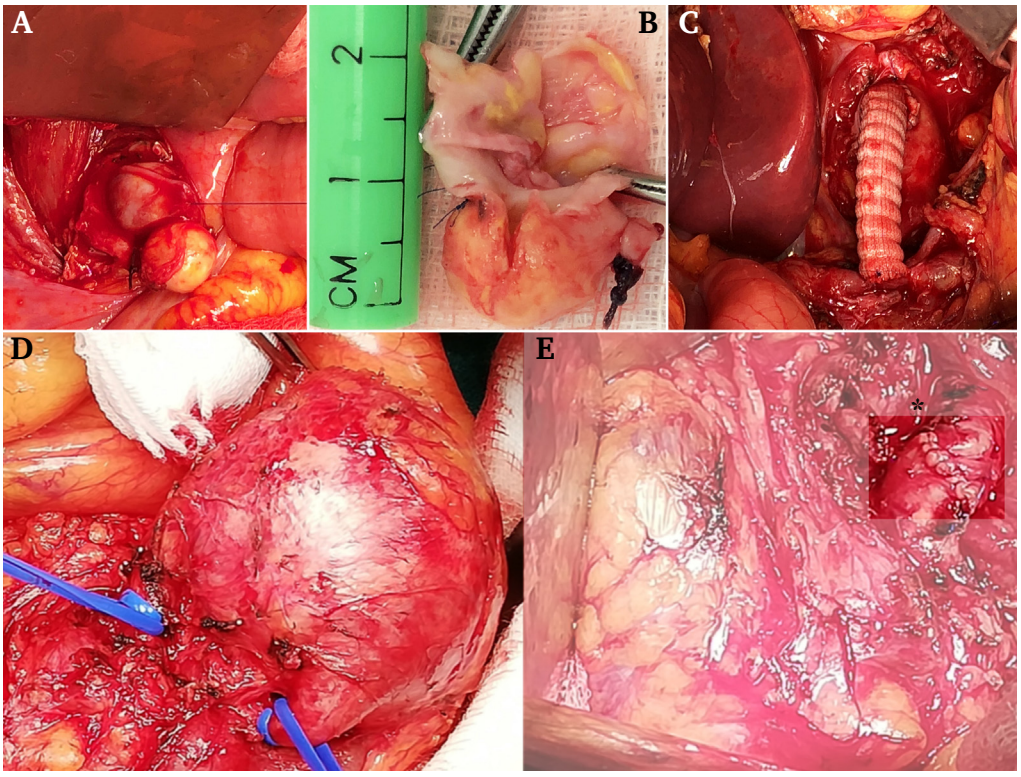

Figure 4. Open repair of two types (A–C and D–E) of pancreaticoduodenal aneurysms (PDAA). (A) First patient with a bilobed aneurysm of the gastroduodenal artery and an occluded coeliac axis. (B) Aneurysm resected and (C) revascularisation achieved by an aortohepatic bypass. (D) Second patient with a large aneurysm of the anterior pancreaticoduodenal artery with proximal and distal control (blue silastic slings). (E) Aneurysm resected with an end to end anastomosis of the anterior pancreaticoduodenal artery (asterisk).

categorical variables. All 30 day outcomes were calculated by standard counts. Midterm outcomes were estimated using the Kaplan–Meier time to event method. A p value $<$ .050 was considered to be statistically significant.

RESULTS

Between 1994 and 2019, 57 consecutive patients, including 35 men (61%), with a mean age of 56 $\pm$ 11 years underwent elective PDAA treatment. At presentation, all PDAAs were intact. No significant differences in baseline characteristics were observed between the two groups (Table 1). Twenty-six patients (46%) presented with non-specific abdominal pain: 15 (48%) in group A and 11 (42%) in group B ($p = .79$). PDAA location differed between groups (Table 2). All gastroduodenal aneurysms ($n = 2$) were treated by open repair. There were statistically significantly more aneurysms of the anterior pancreaticoduodenal artery in group A than in group B (94% *vs.* 58%; $p = .011$) and all posterior

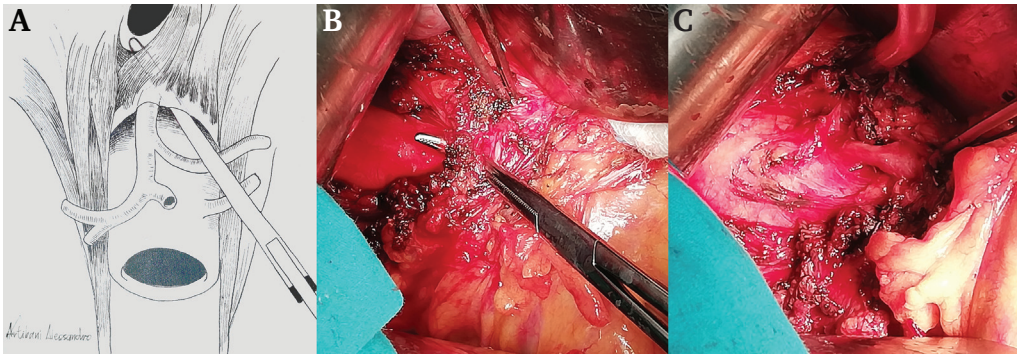

Figure 5. (A) Schematic representation of surgical release of the median arcuate ligament. (B) Corresponding operative view showing the median arcuate ligament on a dissector. (C) View of the coeliac axis after resection of the median arcuate ligament.

Table 1. Demographics, pre-operative cardiovascular risks and comorbidities in 57 patients with a non-ruptured aneurysm of the pancreaticoduodenal arteries and stenosis or occlusion of the coeliac axis, comparing group A and group B

Patient characteristics	Group A (n = 31)*	Group B (n = 26)†	p value	All patients (n = 57)
Age − y	54 ± 11	58 ± 11	.11	56 ± 11
Male sex	18 (58)	17 (65)	.59	35 (61)
CAD	8 (26)	7 (27)	.92	15 (26)
Hypertension	14 (45)	11 (42)	.82	25 (44)
COPD	3 (10)	3 (11)	.82	6 (10)
DM	6 (19)	5 (19)	.99	11 (19)
Chronic renal insufficiency	3 (10)	3 (11)	.82	6 (10)
Current smoker	9 (29)	10 (38)	.57	19 (33)
ASA class			.78	
II	16 (52)	15 (58)		31 (54)
III	13 (42)	10 (38)		23 (40)
IV	2 (6)	1 (4)		3 (5)

Data are presented as n (%) or mean ± standard deviation. ASA = American Society of Anesthesiologists; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; DM = diabetes mellitus.

* Group A consisted of patients who underwent open surgical repair of pancreaticoduodenal aneurysms (PDAA) and release of median arcuate ligament or coeliac axis (CA) bypass.

† Group B consisted of patients who had transcatheter PDAA embolisation with CA stenting or laparoscopic release of the median arcuate ligament.

pancreaticoduodenal artery aneurysms were treated by embolisation. Mean PDAA diameter was 29 mm (IQR 24 − 33 mm), PDAA diameters were significantly larger in group A (32 ± 8 mm [IQR 25 − 36 mm]) than in group B (27 ± 6 mm [IQR 22 − 29 mm]; p = .012).

Open procedure details, including MAL release or CA revascularisation, are presented in Table 3 and endovascular procedure details in Table 4. In group A, aneurysmorrhaphy (n = 23 [74%]) was the preferred technique (aneurysmectomy: n = 8 [26%]). CA compression was

Table 3. Open, and laparoscopic procedures in 31 patients of group A with non-ruptured pancreaticoduodenal aneurysm and stenosis or occlusion of the coeliac axis (CA)

Open repair of the aneurysm with surgical release of MAL by laparotomy or visceral bypass	Patients (n = 31)
Pancreaticoduodenal aneurysm	
Aneurysmorrhaphy	23 (74)
Aneurysmectomy*	8 (26)
MAL and CA revascularisation	
MAL release	21 (68)
Re-implantation of the CA to the aorta†	2 (6)
Aortohepatic bypass‡	8 (26)
Main outcomes	
Aortohepatic bypass occluded	1 (3)

Data are presented as n (%). MAL = median arcuate ligament.

* Two cases of direct arterial reconstruction of the pancreaticoduodenal artery.

† Fibrosis at the origin of the CA.

‡ Extensive fibrosis or occlusion of the CA.

treated by MAL section (n = 21 [68%]) or by revascularisation (n = 10 [32%]) in patients with fibrosis or CA occlusion.

In group B, transfemoral sheath access was used in 17 patients (65%), and transbrachial access in nine patients (35%) with CA or SMA catheterisation. Following successful PDAA embolisation, primary stenting of the CA was attempted in 12 patients with two immediate failures. Laparoscopic MAL release was performed in 16 patients, including two patients with immediate CA stenting failure, and was technically successful in all cases.

Early outcomes

Post-operative mortality was zero in both groups. Six patients (11%) presented transitory elevation of serum amylase/lipase concentration: two (6%) in group A and four (15%) in group B (p = .39). Two patients in group A developed an incisional hernia, and three patients in group

Table 2. Pre-operative presentation of 57 patients with non-ruptured pancreaticoduodenal aneurysm associated with stenosis or occlusion of the coeliac axis in group A and group B

Characteristics	Group A (n = 31)*	Group B (n = 26)†	p value	Total (n = 57)
Abdominal pain	15 (48)	11 (42)	.79	26 (46)
Pancreatitis	1 (3)	1 (4)	.89	2 (4)
Diameter of PDAA on CTA − mm	32 ± 8	27 ± 6	.012	29 ± 8
PDAA location			.001	
Gastroduodenal artery	2 (6)	0 (0)		2 (4)
Anterior pancreaticoduodenal artery	29 (94)	15 (58)		44 (77)
Posterior pancreaticoduodenal artery	0 (0)	11 (42)		11 (19)
MAL syndrome	31 (100)	26 (100)	–	57 (100)

Data are presented as n (%). PDAA = pancreaticoduodenal artery aneurysm; CTA = computed tomography angiography; MAL = median arcuate ligament.

* Group A consisted of patients who underwent open surgical repair of PDAA and release of median arcuate ligament or coeliac axis (CA) bypass.

† Group B consisted of patients who had transcatheter embolisation of PDAA with CA stenting or laparoscopic release of the median arcuate ligament

Table 4. Endovascular procedures in 26 patients of group B with non-ruptured pancreaticoduodenal aneurysm and stenosis or occlusion of the coeliac axis (CA)

Embolisation of aneurysm with stenting of CA or laparoscopic release of MAL	Patients (n = 26)
Pancreaticoduodenal aneurysm	
Transfemoral sheath access	17 (65)
Transbrachial sheath access	9 (35)
CA catheterisation	8 (31)
SMA catheterisation	18 (69)
Embolisation*	26 (100)
MAL and CA revascularisation	
CA stenting attempted	12 (46)
Stenting occluded within 48 h	2 (8)
Stented CA patent at 30 d	10 (38)
Laparoscopic release of MAL†	16 (62)
Main outcomes	
Pancreaticoduodenal aneurysm recanalisation‡	3 (11)
Late failure of CA stenting or MAL release by laparoscopy§	5 (19)

Data are presented as n (%). MAL = median arcuate ligament; SMA = superior mesenteric artery.
* Coil embolisation of aneurysm's afferent and efferent arteries.
† Including two patients with immediate CA stenting failure.
‡ Three redo procedures: redo embolisation (n = 1) and open surgical aneurysmorrhaphy (n = 2).
§ Four stents and one MAL release by laparoscopy failed with five redo procedures: redo stenting (n = 2) and aortohepatic bypass (n = 3).

B a brachial haematoma, all of which were managed conservatively. There was no mesenteric ischaemia, or biliary, or pancreatic fistula. Median hospital stay was longer in group A than in group B (5 *vs.* 3 days; p = .001). After 30 post-operative days, all PDAAs were successfully excluded after both open repair and embolisation. All CAs treated by MAL release or bypass were patent; two CA stentings failed but were followed by successful conversion with laparoscopic MAL release.

Midterm outcomes

Two patients (6%) in group A were lost to follow up after effective monitoring of five and six years, respectively. No patients in group B were lost to follow up.

In group A, after a median follow up of 11 years (IQR 7 — 19 years), no PDAA recurrence was observed. All CAs following MAL release were patent without restenosis on DUS, and one of the eight aortohepatic bypass grafts occluded at five years.

In group B, PDAA recanalisation was observed in three patients 2.9, 3.6, and 4.3 years after embolisation, respectively. Successful redo surgery was carried out in all cases with one redo embolisation, and two open surgical aneurysmorrhaphies. Five CA restenoses occurred, four of them between eight months and 4.3 years after CA stenting (n = 4) and one at five years after MAL release by laparoscopy. Two of the CA restenoses were associated with PDAA recanalisation. The five patients received redo stenting (n = 2) or aortohepatic bypass (n = 3), associated in two patients with aneurysmorrhaphy of a recanalised PDAA (Table 4).

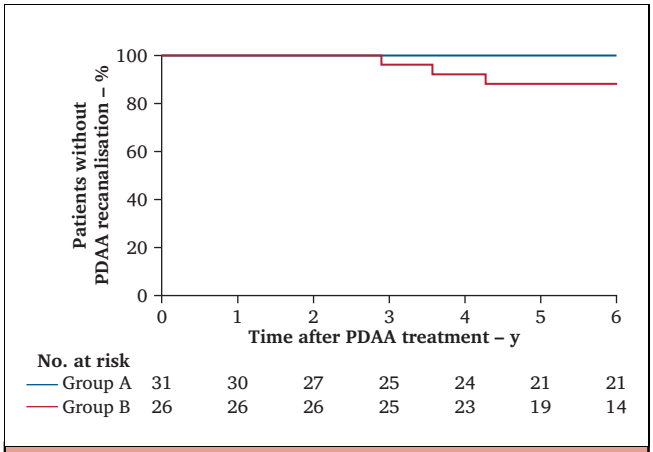

Figure 6. Cumulative Kaplan—Meier estimate of freedom for pancreaticoduodenal aneurysm (PDAA) recanalisation in group A with open repair by aneurysmorrhaphy or aneurysmectomy and in group B with transcatheter coil embolisation of the aneurysm. The number of patients at risk is indicated at each interval. At six years, freedom from PDAA recanalisation was 100% in group A, and 88% ± 6% in group B (p = .082).

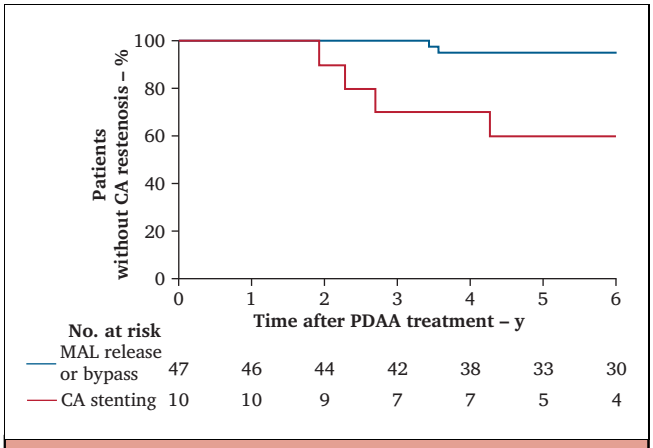

Figure 7. Cumulative Kaplan—Meier estimate of freedom from coeliac axis (CA) restenosis or bypass occlusion in patients with surgical release of median arcuate ligament (MAL) by laparotomy or by laparoscopy or bypass compared with patients with CA stenting for the treatment of pancreaticoduodenal aneurysm (PDAA). The number of patients at risk is indicated at each interval. At six years, freedom for CA occlusion after MAL release or visceral bypass occlusion was 95% ± 3% *vs.* 60% ± 9% for CA stenting (p = .001).

At six years, Kaplan—Meier estimates of freedom from PDAA recanalisation were 100% in group A and 88% ± 6% in group B (p = .082) (Fig. 6). For the same time period, estimates of freedom for CA restenosis/occlusion after MAL release or visceral bypass occlusion was 95% ± 3% *vs.* 60% ± 9% for CA stenting (p = .001) (Fig. 7). No PDAA ruptured during follow up. Six patients (10%) died of unrelated causes during follow up. Overall survival at six years was 97% ± 2%.

Table 5. Recent published studies (2010–2019) on elective treatment of true, non-ruptured pancreaticoduodenal and gastroduodenal aneurysms associated with coeliac axis (CA) compression

First author (year)	Cases (*n*)	Treatment (*n*)	Aneurysm diameter – mm (*n*)	Treatment of PDAA (*n*)	Treatment of CA stenosis (*n*)	Follow up – mo
Marone (2011)[20]	4	Endo (2) Open (2)	28	Aneurysmorrhaphy (1) Aneurysmectomy (1) PDAA embolisation (2)	MAL release	12 48
Brocker (2012)[14]	1	Endo (1)	10	PDAA embolisation (1)	NR (–)	6
Ghariani (2013)[21]	5	Open (5)	30	Aneurysmorrhaphy (5)	NR (–)	42
Wattez (2013)[22]	1	Hybrid (1)	20	PDAA embolisation (1)	Aortohepatic bypass (1/1)	8
Armstrong (2014)[23]	1	Endo (1)	14	PDAA embolisation (1)	MAL release (0/1)	12
Corey (2016)[11]	22	Endo (18) Open (4)	26	PDAA embolisation (18) Aneurysmectomy (4)	CA stenting (1/18) MAL release (1/4)	60
Sgroi (2015)[24]	2	Endo (2)	20	PDAA embolisation (2)	MAL release (0/2)	12
Tétreau (2016)[25]	7	Endo (5) Open (2)	22	PDAA embolisation (5) Aneurysmorrhaphy (2)	MAL release (0/5) Aortocoeliac bypass (2/2)	12
Boll (2017)[26]	11	Endo (8) Open (3)	16	PDAA embolisation (8) Aneurysmorrhaphy (3)	MAL release (0/8) Aortocoeliac bypass (3/3)*	6
Deser (2017)[27]	1	Open (1)	28	Aneurysmectomy (1)	Aortohepatic bypass (1/1)	6
Simon (2017)[28]	2	Hybrid (2)	20 (1) 40 (1)	PDAA embolisation (2)	Aortocoeliac bypass (1/2) Aortohepatic bypass (1/2)	12
Takeuchi (2017)[29]	2	Endo (1) Open (1)	12 (1) 30 (1)	PDAA embolisation (1) Aneurysmectomy (1)	CA stenting (1/1)† MAL release (1/1)	12 36
Vandy (2017)[4]	15	Open (15)	21	Aneurysmectomy (15)	Aortohepatic bypass (1/15) SMA re-implantation (1/15) MAL release (3/15)	52
Peyrotte (2018)[30]	2	Open (2)	30 (1) 10 (1)	Aneurysmectomy (2)	MAL release (2/2)	54
Present study	57	Endo (26) Open (31)	27 (26) 32 (31)	PDAA embolisation (26/26)‡ Aneurysmectomy (8/31) Aneurysmorrhaphy (23/31)	CA stenting (12/26)§ MAL release (21/31) Coeliac bypass (2/31) Aortohepatic bypass (8/31)*	72

PDAA = pancreaticoduodenal artery aneurysm; Endo = endovascular; MAL = median arcuate ligament release by open surgery; SMA = superior mesenteric artery; NR = not reported.
* Occlusion of bypass ($n = 1$).
† CA stenting occluded ($n = 1$).
‡ Late reperfusion of PDAA ($n = 3$).
§ CA stenting occluded within 30 days in two patients and in four additional patients during follow up.

DISCUSSION

This multicentre study shows that open and endovascular treatment of PDAA yield excellent post-operative results, with zero mortality and low morbidity in both groups. However, midterm follow up showed differences, with late recanalisation of three PDAAs (11%) following embolisation *vs.* none following open repair, and worrying results of CA stenting, with a crude 50% occlusion rate ($n = 6/12$) at six years.

Since 2000 endovascular procedures have been performed for PDAA with increasing frequency.[4] The increasing popularity of transcatheter PDAA embolisation is based on favourable comparison with open techniques in terms of post-operative complications, which can be as high as 19% for emergency open resection of PDAA *vs.* nearly none declared for endovascular techniques.[14] There is no doubt that transcatheter PDAA embolisation is the preferred technique in urgent situations and in patients with severe comorbidities.[8,15] However, endovascular treatment has some limitations. It exposes patients to the risk of late aneurysm reperfusion with the need for re-intervention approaching 15% in the study by Barrionuevo *et al.*[16] and 11% in this series. In addition, owing to complex arterial anatomy, embolisation is liable to be incomplete, with late recanalisation or extensive overflowing, with a risk of intestinal ischaemia or interruption of the anastomotic pathways related to low CA flow.[8] No such complications were observed in the present series, where open surgical repair was used for all gastroduodenal aneurysms and in 29/44 anterior pancreaticoduodenal aneurysms, with preserved vascularisation of the pancreas and bowel, and a complementary coeliac or hepatic artery bypass in 10 patients without significant morbidity.

Conversely, all aneurysms of the posterior pancreaticoduodenal artery were treated by embolisation, which was preferred to open surgical repair with a Kocher

manoeuvre and the attendant risk of pancreatic or biliary fistula. It is considered that the choice between transcatheter embolisation or open PDAA repair should be tailored according to the aneurysm's location and size.

Posterior pancreaticoduodenal artery aneurysms are most often easily accessible by catheterisation and should be embolised, except for large aneurysms of a broad based origin within millimetres of the SMA, which present the risk of small bowel and foregut structure ischaemia.

In this series, a MAL syndrome was present in all patients. These figures are comparable to those of Corey *et al.*,[11] who found it in 97% of CA stenoses in a relatively large series of 35 patients. Accumulating cumulative evidence from other studies has confirmed that 50% — 80% of PDAAs are associated with CA compression by MAL,[7,17–19] a fibrous arch that compresses the CA and leads (after years) to CA fibrosis and occlusion, as demonstrated in the 10 patients in this series in whom CA bypass was needed. Interestingly, while most PDAAs are degenerative, the most frequent cause of CA occlusion is not degenerative but rather related to compression by MAL. Upon CA occlusion, haemodynamic changes in the pancreaticoduodenal arcades may be responsible for initiation, growth, and rupture of a PDAA. The haemodynamic hypothesis has been confirmed by Mano *et al.*[19] using four dimensional magnetic resonance imaging. They found high retrograde flow with increased velocity and high shear stress in the pancreaticoduodenal and gastroduodenal arteries of patients with CA occlusion *vs.* controls. When associated with the results of clinical studies,[7,17,18] these objective haemodynamic findings are important physiological arguments in favour of MAL release, even if clinical demonstration is difficult owing to the rarity of relevant cases.

A survey of contemporary series (2010 — 2019) limited to elective treatment of true, non-ruptured PDAAs associated with CA occlusion was conducted (Table 5). In 14 recent publications,[4,11,14,20–30] excluding this one, 76 true PDAA cases were found, with 41 (54%) transcatheter embolisation and 36 (46%) open repair procedures. CA stenosis by MAL, which appeared in all of these patients, was treated in only 20 (29%). This short contemporary survey shows that many authors question the need for CA release or revascularisation in patients with PDAA. They reject the aforementioned haemodynamic hypothesis on the basis of favourable case reports or short series of PDA aneurysms without CA release.[24,26,31,32] However, these results ought not to mask the need for critical understanding of PDA haemodynamics when treating these aneurysms.[4,8,19]

In the present series, open surgical release of MAL offered satisfactory long term results in all patients without fibrosis or CA occlusion ($n = 21$). In the remaining 10 patients, fibrosis due to long lasting compression by MAL required direct reimplantation of the CA on the coeliac aorta or revascularisation by an aortohepatic bypass leading to durable results, except for one patient with asymptomatic bypass occlusion discovered at the five year follow up CTA.

In group B, primary CA stenting was carried out in 12 patients. The results, like those of several others,[16,24,29,33–35] were disappointing, with two immediate failures and four CA restenoses or occlusions occurring between eight months and 4.3 years, with freedom from CA restenosis of only 60% at six years. It is recognised that these small numbers make it difficult to draw definitive conclusions, but a crude 50% rate of short term failures renders the use of stents in this indication questionable. Of note, PDAA recanalisation occurred in two patients with CA occlusion following stenting.

Limitations of the study

This study has two limitations, first its retrospective nature, and second the limited number of patients, despite multicentre recruitment. However, true PDAAs are rare and the majority of reports in the literature consist of only two or three cases or small series with a short follow up. Although not a randomised protocol, an attempt has been made to present the results of a consecutive series of patients with decent midterm follow up and balanced management according to the PDAA characteristics and the limitations of each technique.

Conclusion

The current data suggest that, under elective conditions, both open and endovascular treatment for PDAA yield excellent post-operative results, with few midterm recanalisations after PDAA embolisation. In this series, CA stenting yielded worrying results, with a significant number of early and midterm failures. This study also demonstrates that in an elective setting, open surgical treatment of PDAAs according to their topography and associated with MAL release or visceral revascularisation can be performed safely, with minimal risk.

CONFLICT OF INTEREST

None.

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APPENDIX A. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2021.02.023.

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