

## PANCREATICODUODENAL ARTERIES ANEURYSMS: EVALUATION OF FREQUENCY AND ASSOCIATION WITH CELIAC ARTERY STENOSIS DUE TO COMPRESSION BY MEDIAN ARCUATE LIGAMENT.

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### ABSTRACT

The aim of the study is to evaluate the prevalence of pancreaticoduodenal arteries (PDA) aneurysms in a large population, and to define possible correlations with celiac artery stenosis due to compression by the median arcuate ligament. Radiological reports of abdominal contrast-enhanced CT scans of 18,180 patients were scrutinized to identify patients with true PDA aneurysms. Two abdominal radiologists classified the aneurysms according to size, location and morphology and scored the presence of celiac artery stenosis due to either median arcuate ligament compression or atherosclerotic disease. Eleven true PDA aneurysms were identified in 10 patients. Nine out of 10 patients had stenosis of the celiac artery, in which 8 cases (80%) was due to compression by the median arcuate ligament of the diaphragm, which likely represents the most common underlying etiology. In our population, the prevalence of reported PDA aneurysms was 0.055.

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### 1. Introduction

Pancreaticoduodenal artery (PDA) aneurysms are rare abdominal aneurysms, accounting for about 2% of all visceral aneurysms [1]. These aneurysms typically originate from the pancreaticoduodenal arcades, an anastomotic system connecting the celiac artery (CA) with the superior mesenteric artery (SMA) through four different pancreaticoduodenal arteries. The anterior and posterior superior PDAs arise from the gastroduodenal artery (GDA), which is a branch of the common hepatic artery (CHA), originating from the CA. The anterior and posterior inferior PDAs originate from the SMA. The anastomoses between these arteries create two pancreaticoduodenal arcades, one anterior and one posterior, which are located around the head of the pancreas and are responsible for blood perfusion of the head of the pancreas and part of the duodenum.

The majority of PDA aneurysms consist of false aneurysms (also known as pseudoaneurysms), secondary to local infection or inflammation related to surgery, trauma, endoscopic retrograde cholangiopancreatography (ERCP), acute or chronic pancreatitis or cholecystitis [2]. Aneurysms found in patients without an underlying history of these conditions or interventions may be considered true aneurysms [3].

Although their diagnosis remains challenging due to their small size, very low incidence and complex vascular anatomy, it carries a significant clinical relevance due to the high propensity for rupture, regardless of size, associated with a high mortality rate (up to 50%) [4,5].

Most of the current evidence regarding these rare PDA aneurysms are reported in the prior literature as sporadic case reports or small cohorts, diagnosed with conventional mesenteric angiography at the time of rupture [2,3,6–11], without a formal analysis of the possible frequency in a large population.

Some prior studies have suggested the possible correlation between true PDA aneurysms and stenosis of the celiac artery due to atherosclerotic disease, or celiac artery compression by the median arcuate ligament of the diaphragm [12]. This could have significant implications since treatment may consider both the embolization of the aneurysm and the removal of the stenotic celiac artery to correct the hemodynamic imbalance in the pancreaticoduodenal arcades [12].

The aim of our study was, therefore, to retrospectively evaluate the prevalence of true pancreaticoduodenal arteries aneurysms in a large population evaluated with contrast-enhanced CT imaging in a definite time span.

Moreover, we tried to confirm the correlation with celiac artery stenosis due to compression by the median arcuate ligament.

## 2. Material and Methods

### 2.1 Patient selection

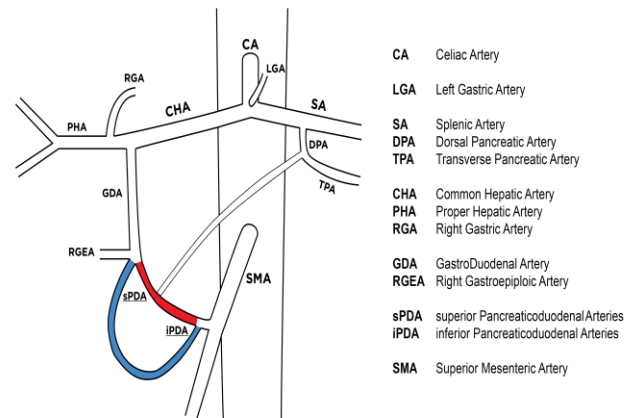
The institutional review board of our hospital approved this retrospective study, and the requirement for informed consent was waived. A search of the medical and radiological records was performed at our institution selecting abdominal contrast-enhanced CT scans acquired from January 2012 to December 2018. The initial population consisted of 18,180 consecutive patients imaged with multiphase contrast-enhanced CT. Reports were scrutinized when searching for abdominal visceral aneurysms with the following keywords: “aneurysm” AND (“visceral\*” OR “splanchnic\*” OR “pancreatic\*” OR “gastric\*” OR “duodenal\*” OR “hepatic\*” OR “mesenteric\*” OR “celiac\*”), to avoid selection bias due to misdiagnosed PDA aneurysms. Patients with medical records of possible pathologies associated with the development of false aneurysms, such as local infection or inflammation related to surgery, trauma, ERCP, acute pancreatitis, cholecystitis, as well as patients with collagens or congenital vascular abnormalities, were excluded from this study. Medical records were also scrutinized to determine demographic characteristics, clinical background, and presentation.

### 2.2 CT technique

Contrast-enhanced CT studies were performed using either multidetector 16-slice CT (General Electric Brightspeed Elite, GE Healthcare, Chicago, USA) or 64-slice CT (Siemens SOMATOM Definition AS+, Siemens Healthcare, Erlangen, Germany). CT scanning of the abdomen was performed from the diaphragm to the pubic symphysis using the following acquisition parameters: slice thickness 1.25-3.0 mm, tube potential 120-140 kVp and automatic mA modulation with a range of 250-300 mAs, pitch 1.5. All the selected studies included pre-contrast images of the upper abdomen. Post-contrast images were obtained during the arterial phase (20-30 seconds after injection of the contrast agent determined by a “bolus tracking” technique, using a threshold of 100 HU in the abdominal aorta) and portal venous phase (70-80 seconds) after the administration of 100-120 ml of a non-ionic iodinated contrast agent (Iomeprol, Iomeron 400, 400 mgI/ml, Bracco, Milan, Italy) at the flow rate of 3-5 ml/sec followed by 20-40 ml of saline flushing at the same injection rate. Images were reconstructed on coronal and sagittal planes and with the application of volume rendering (VR) and maximum intensity projection (MIP) algorithms.

### 2.3 Imaging analysis

Two radiologists (each with 5 years of experience in abdominal imaging) independently evaluated the corresponding CT images in a blinded fashion. Discordances among readers were resolved by consensus. The readers recorded the location and size of the aneurysms, caliber of the non-aneurysmatic visceral arteries (Figure 1), including the celiac artery, GDA, PDA and SMA measured in the arterial phase images. Multiplanar reconstructed images were adopted to measure the caliber of the vessels through their major axis. Arterial enlargement was considered to be present if its diameter was at least 25% larger than the normal range (normal size of celiac artery:  $0.79 \pm 0.04$  cm; GDA/PDA:  $0.4 \pm 0.03$  cm [13]).



**Figure 1. Graphic representation of the splanchnic circulation. The anterior pancreatic arcade, formed by the anterior branches of the superior and of the inferior pancreaticoduodenal arteries, is shown in red. The posterior pancreatic arcade, formed by the posterior branches of the superior and the inferior pancreaticoduodenal arteries, is shown in blue.**

The presence of atherosclerotic disease or compression factors of the celiac artery were also recorded. Aneurysms were classified according to the number (i.e. single or multiple), location (i.e. in the PDA or GDA) and morphology (i.e. fusiform or saccular).

Significant celiac artery stenosis was reported to be present if there was luminal narrowing greater than 50% of the proximal celiac artery [14]. Celiac artery compression by the median arcuate ligament was defined by the presence of a “hooked” appearance of the proximal celiac artery with a characteristic superior indentation in the upper portion of the vessel, few millimeters from the origin on sagittal images, associated with a subsequent post-stenotic dilatation of the celiac artery, in absence of atherosclerotic plaques or other causes of extrinsic compression. The severity of celiac artery stenosis caused by median arcuate ligament compression was further classified as follows: 1) mild if there was stenosis of the lumen lower than 50%, length of the stenosis  $\leq 3$  mm and no enlarged collateral; 2) moderate if there was stenosis of the lumen between 50-80%, length of the stenosis 5-8 mm and collaterals around the pancreatic head; 3) severe if there was stenosis of the lumen between 80-100%, length of the stenosis  $\geq 8$  mm and collaterals around the pancreatic head or body [15]. The celiac stenosis was attributed to atherosclerotic disease if there was significant intraluminal stenosis of the celiac artery ( $\geq 50\%$ ) in presence of atherosclerotic plaques and without a “hooked” appearance of the celiac artery. Atherosclerotic stenosis was also further classified in moderate (50-80% stenosis) or severe ( $\geq 80\%$ ).

### 2.4 Statistical analysis

Data of final cohort and qualitative contrast-enhanced CT analysis were reported as either continuous variables, summarized using mean and standard deviation with range or categorical variables using numbers and percentages.

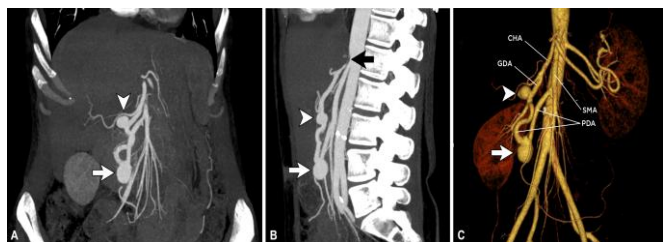
### 3. Results

Eleven true PDA aneurysms were detected in 10 patients (9 females and 1 male), with a mean age of  $64.4 \pm 16.0$  years (range 30-84 years). Characteristics of the final population and PDA aneurysms are summarized in Table 1. One patient had two PDA aneurysms measuring 16 and 28 mm in diameter, respectively. The mean PDA aneurysm size was  $18.3 \pm 8.8$  mm (range 9-35 mm). All aneurysms were saccular in morphology. None of the PDA aneurysms were ruptured at the time of initial diagnosis.

Patient	Gender	Age (years)	PDA aneurysm diameter (mm)	GDA aneurysm diameter (mm)	PDA max diameter (mm)	GDA max diameter (mm)	CA max diameter (mm)	SMA max diameter (mm)	Celiac artery stenosis	Pancreatic collaterals	Celiac stenosis severity	Ruptured aneurysm
1	F	62	9	8	4	3	3	8	MAL	Present	Severe	No
2	M	30	20	-	7	4	8	10	MAL	Absent	Moderate	No
3	F	52	18	15	6	7	4	8	MAL	Present	Severe	No
4	F	58	16 and 28	-	6	8	3	9	MAL	Present	Severe	No
5	F	63	28	-	9	8	6	8	MAL	Absent	Severe	No
6	F	81	35	-	5	6	9	8	MAL	Present	Severe	No
7	F	65	12	-	7	7	6	8	MAL	Present	Severe	No
8	F	84	9	-	5	5	3	8	ATS	Absent	Severe	No
9	F	69	9	-	5	6	6	7	MAL	Present	Severe	No
10	F	80	17	-	3	3	5	6	Absent	Absent	None	No
Mean	-	64.4	18.3	11.5	5.7	5.7	5.3	8.0	-	-	-	-

**Table 1.** Characteristics of the final population and aneurysms included in the current study. PDA: pancreaticoduodenal arteries; GDA: gastroduodenal artery; CA: celiac artery; SMA: superior mesenteric artery, MAL: median arcuate ligament; ATS: atherosclerosis.

In two (20%) patients, a coexistence of a second aneurysm in the GDA (mean diameter 11.5 mm) (Figure 2) was found. Eight patients (80%) had an associated enlargement of PDA (mean diameter  $5.7 \pm 1.7$  mm, range 3-9 mm). An enlargement of GDA (mean diameter  $5.7 \pm 1.8$  mm, range 3-8 mm) was also noted in 7 patients (70%).

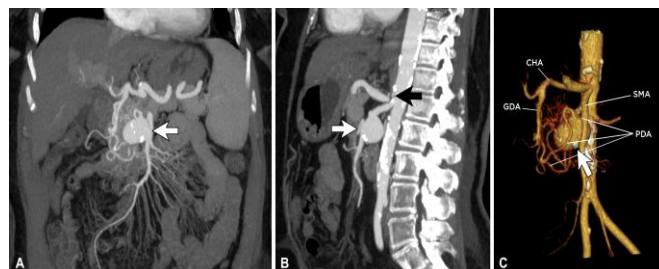


**Figure 2.** Contrast-enhanced CT scan of a 52-year-old woman with two aneurysms, one in the gastroduodenal artery (GDA, white arrowhead) and one in pancreaticoduodenal artery (PDA, white arrow). There is also severe stenosis of the origin of the celiac artery (black arrow). A, B: coronal and sagittal MIP reformatted images. C: volume rendering reconstruction. CHA: common hepatic artery; SMA: superior mesenteric artery.

Nine out of 10 patients (90%) had severe stenosis or occlusion of the celiac artery. In one patient, the stenosis was related to an atherosclerotic plaque determining severe intraluminal narrowing (greater than 80% reduction in caliber).

In the remaining eight patients (80%), the stenosis was considered to be secondary to a compression by the median arcuate ligament of the diaphragm. In those subjects, the median arcuate ligament stenosis was classified as severe (i.e. 80-100% stenosis or  $\geq 8$  mm in length) in 7 out of 8 patients, with associated multiple pancreatic head collaterals in 6 out of 8 patients (Figure 3).

In our population, the prevalence of true PDA aneurysms was 0.055% out of all consecutive patients imaged with contrast-enhanced CT.

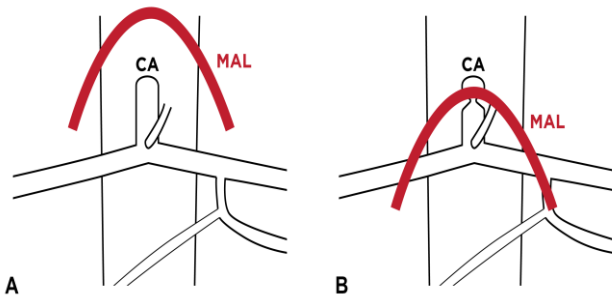


**Figure 3.** Contrast-enhanced CT scan of an 81-year-old woman with an aneurysm of the pancreaticoduodenal artery (PDA) (white arrows). There is also severe stenosis of the origin of the celiac artery with calcified atherosclerotic plaques (black arrow) and multiple pancreatic head collaterals. A, B: coronal and sagittal MIP reformatted images. C: volume rendering reconstruction. CHA: common hepatic artery; GDA: gastroduodenal artery; SMA: superior mesenteric artery.

### 4. Discussion and conclusions

We undertook this study to evaluate the prevalence of true PDA aneurysms and their association with celiac artery stenosis in a large cohort of patients evaluated with contrast-enhanced CT imaging. The main results of our study consisted of a high prevalence (90% of patients) of celiac artery stenosis in patients with true PDA aneurysms, concordant to the report in the prior literature [14,16,25,17-24]. Indeed, in eight out of ten patients (80%), the PDA aneurysms were associated with moderate-to-marked stenosis of the celiac artery due to compression by the median arcuate ligament of the diaphragm. Our results are concordant with the prior studies reporting the presence of median arcuate ligament compression of the celiac artery in 33-100% of the patients with true PDA aneurysms [14,16,25,17-24].

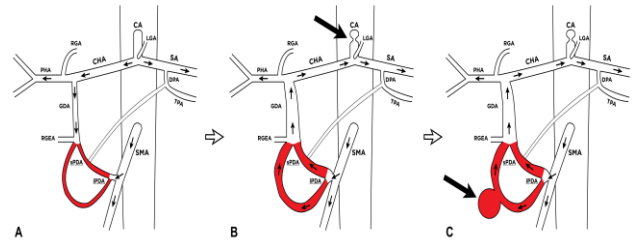
The median arcuate ligament is a fibrous arch that bridges the diaphragmatic crura on either side of the aortic hiatus. The ligament usually passes above the origin of the celiac artery; however, in up to 33% of the general population, it may insert lower, crossing the root of the celiac artery (Figure 4) [26].



**Figure 4. A: normal insertion of the median arcuate ligament (MAL). B: lower insertion of the MAL, with compression of the origin of the celiac artery (CA).**

This can lead to dynamic compression of the celiac artery, usually more severe in expiration, because of the cranial displacement of the aortic branches. This compression can be symptomatic in about 1% of the general population [6]. This uncommon clinical condition is known as median arcuate ligament syndrome (MALS), or *Dunbar syndrome*, and it is more frequently observed in young, thin women [27]. The classical diagnostic triad consists of chronic postprandial abdominal pain, weight loss and an upper abdominal arterial bruit, but these findings may be vague or absent in the majority of patients with compression of the celiac artery by the median arcuate ligament. In these patients with chronic celiac artery compression and stenosis by the median arcuate ligament, a possible complication may be the development of true aneurysms in the PDAs. Currently, the formation of PDA aneurysms is thought to be a consequence of altered flow and hemodynamic circulation due to celiac artery stenosis. In the conventional visceral circulation, blood arrives to the superior PDA from the gastroduodenal artery, through the common hepatic artery and the celiac artery, and to the inferior PDA from the SMA (Figure 5A). When the stenosis or occlusion of the celiac artery occurs, the two pancreaticoduodenal arcades function as collateral ways for blood perfusion to reach the gastroduodenal artery, and from there to all the other arteries arising from the celiac artery (Figure 5B) [28]. The walls of the PDAs and the GDA are not used to such a high blood flow, and therefore may be damaged by the increased shear stress [28]. Furthermore, the dynamic nature of the celiac artery compression can lead to an alternating anterograde and retrograde blood flow in the PDA, resulting in turbulent flow. This altered hemodynamic condition can lead to intimal damage and dysfunction of the medial layer, responsible for maintaining the structure and the elasticity of the vessel [28]. The medial layer dysfunction can ultimately lead to the development of a true aneurysm (Figure 5C) [29,30]. The evaluation of our population is confirming this hypothesis, since we noticed an enlargement of PDAs, an enlargement of GDA, and the presence of pancreatic head collaterals in 87.5%, 75% and 75%, respectively, of the patients with CA compression by MAL; findings that most probably reflect the increased blood flow through the pancreaticoduodenal arcades.

A recent study performed by Heo et al. [31] also reported that splanchnic artery aneurysms were present in 24% of patients with MALS, with bleeding due to an aneurysm rupture occurring in 2.7% of cases. Interestingly, our study also demonstrated the coexistence of GDA aneurysms in two patients with PDA aneurysm and stenosis of the celiac artery, supporting the hemodynamic hypothesis as an underlying cause.



**Figure 5. A: normal visceral circulation. B: visceral circulation with stenosis of the origin of the celiac artery (black arrow); there is a high flow in the pancreaticoduodenal arteries, and an inverted and increased flow in the gastroduodenal artery. C: aneurysm development in the posterior pancreaticoduodenal arcade (black arrow).**

Our study represents one of the largest populations of PDA aneurysms collected in a single institution (Table 2).

Study	Number of Patients	Gender	Mean age (years)	Number of aneurysms	Aneurysm mean size (mm)	Overall CA stenosis or occlusion	CA stenosis due to MAL	CA stenosis due to atherosclerosis	Ruptured aneurysms
Antonak et al.[16]	15	NR	58(31-84)	29 PDA: 3 GDA	13 (3-50)	15/15	14/15	1/15	NR
Bageac et al.[17]	9	4 M: 5 F	54(41-75)	9 PDA	16 (4-30)	3/9	3/9	0/9	7/9
Boll et al.[18]	20	9 M: 11 F	61(35-85)	16 PDA: 4 GDA	16 (3-35)	15/20	NR	NR	9/20
Chivot et al.[19]	10	6 M: 4 F	61(38-78)	10 PDA	5 (2-10)	10/10	10/10	0/10	10/10
Corey et al.[20]	35	21 M:14 F	60	25 PDA: 11 GDA	19 (10-48)	34/35	11/35	23/35	7/35
Flood et al.[21]	14	4 M: 10 F	46(26-50)	14 PDA	20 (13-70)	14/14	10/14	1/14	1/14
Katsura M et al.[22]	8	5 M: 3 F	62(45-82)	8 PDA	16 (10-20)	4/8	NR	NR	5/8
Murata et al.[23]	10	9 M: 1 F	57(45-72)	10 PDA	14 (5-33)	3/10	1/10	2/10	9/10
Nishiyama et al.[24]	7	5 M: 2 F	55 (40-70)	7 PDA	20 (10-35)	3/7	NR	NR	4/7
Orion et al.[14]	21	8 M: 13F	54	21 PDA	20 (8-32)	15/21	NR	NR	5/21
Vandy et al.[25]	15	5 M: 10 F	59	13 PDA: 3 GDA	21 (4-35)	15/15	15/15	0/15	0/15
Overall	164	76 M: 73 F	57 (31-85)	162 PDA: 21 GDA	16 (2-70)	132/164 (80.4%)	64/108 (59.2%)	108 (25%)	57/149 (38.2%)

**Table 2. Most relevant prior studies (including more than 5 PDA aneurysms each) reporting PDA aneurysms and their association with celiac artery stenosis. Number in parenthesis are ranges unless otherwise specified. NR: not reported; PDA: pancreaticoduodenal arteries; GDA: gastroduodenal artery; CA: celiac artery; MAL: median arcuate ligament.**

We estimated a prevalence of true PDA aneurysms of 0.055%. Although PDA aneurysms are considered to be 2% of all visceral aneurysms [1], to the best of our knowledge there were no data about the general prevalence of these aneurysms in current literature. True aneurysms have been reported in patients with a wide age range (mean, 57 years), with no differences in gender [3].

Although these conditions can be detected as incidental findings in asymptomatic patients, they manifest with various clinical symptoms, ranging from vague abdominal symptoms to hemorrhagic shock in case of rupture. The risk of rupture has been reported from 30 to 69% [10] and it carries a high mortality rate (up to 50%). Unlike other aneurysms of visceral arteries, no correlation has been reported between the size of true PDA aneurysms and their propensity to rupture [14]; and many cases of ruptured PDA aneurysms have been described with a size smaller than 10 mm. Therefore, treatment of the PDA should always be considered due to the high risk of rupture. The endovascular approach is considered the best treatment option, allowing exclusion of the aneurysm with a covered stent graft or coil embolization, with a high rate of success [3,23]. Open surgery is associated with a higher mortality rate [11]. If the aneurysm is associated with celiac artery compression from median arcuate ligament, elective open or laparoscopic surgery to decompress the artery may be considered to reduce the risk of developing other aneurysms [3].

Our study has multiple limitations that need to be acknowledged. First, it was conducted retrospectively in a large population, considering only the radiological reports to identify PDA aneurysms. Moreover, not all the exams were strictly performed with a CT angiography protocol. These limitations may have introduced selection biases and underestimate the prevalence of small PDA aneurysms. However, due to the rarity of this entity, a prospective study may be challenging to perform. Second, most of our patients did not undergo selective angiography to exclude the presence of additional smaller aneurysms. A long-term imaging follow-up should also be considered in a larger number of PDA aneurysms to assess the risk of rupture of these lesions.

Although true PDA aneurysms are a rare finding in an abdominal contrast-enhanced CT study, their diagnosis may have a critical impact due to the possible risk of rupture. Our study has demonstrated a low prevalence in a large population imaged with contrast-enhanced CT. These aneurysms are associated with a stenosis of the celiac artery, mostly secondary to the compression from the median arcuate ligament, which likely represents the most common underlying etiology.

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