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# **Celiac axis stenosis and digestive disease: diagnosis, consequences and management**

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**Abbreviations used:** CA: Celiac axis; SMA: Superior mesenteric artery; PD: pancreatoduodenectomy

**Key words:** celiac axis, median arcuate ligament, atheromatous stenosis

**Summary:**

Arterial blood flow to the organs of the upper abdomen is provided by the celiac axis (CA) and the superior mesenteric artery (SMA) that communicate between each other *via* the gastro-duodenal artery, the anterior and posterior pancreatico-duodenal arcades, the branches of the dorsal pancreatic artery and inconsistently, though a supplementary arcade that connects the CA and the SMA (arcade of Bühler).

Celiac axis stenosis may or may not have a hemodynamic impact on the splanchnic circulation. Hemodynamically significant CA stenosis can be asymptomatic, or symptomatic with protean clinical consequences. Management depends on whether the mechanism of stenosis is extrinsic or intrinsic.

When upper gastrointestinal interventional radiology or surgery is indicated, stenosis can pose technical difficulties or create severe ischemia requiring good understanding of this entity in the planning of operative steps and adapted management. Management of CA stenosis is therefore multidisciplinary and may involve interventional radiologists, gastrointestinal surgeons, vascular surgeons as well as medical physicians.

Even though the prevalence of CA stenosis is relatively low (between 5 and 10%) and irrespective of its etiology, surgeons, radiologists and physicians must be aware of it because it can intervene in the management of upper gastrointestinal disease. It must be sought, and treatment must be adapted to each particular situation to avoid potentially severe complications.

Essential points:

- Celiac axis stenosis can have a hemodynamic impact on the splanchnic circulation; it can be symptomatic or not. The prevalence is between 5% and 10%.
- The most common etiologies are extrinsic stenosis caused by the median arcuate ligament and intrinsic stenosis, related to atherosclerosis.
- Establishment of a positive diagnosis, determination of the etiology and evaluation of the clinical impact all depend on contrast-enhanced abdomino-pelvic CT scan and echo-Doppler studies.
- If interventional radiology or surgery of the upper abdomen is planned, CA stenosis must be systematically sought in order to adapt management plans to avoid technical complications and/or severe ischemic phenomena.

## INTRODUCTION

The celiac axis (CA) and the superior mesenteric artery (SMA) provide the arterial blood flow to the upper abdominal viscera; they intercommunicate via the gastroduodenal artery, the anterior and posterior pancreaticoduodenal arcades, the branches of the dorsal pancreatic artery and inconsistently, though a supplementary arcade that courses between the CA and the SMA (arcade of Bühler) [1].

CA stenosis may or may not have a hemodynamic impact on the splanchnic circulation. Hemodynamically significant CA stenosis can be asymptomatic, or symptomatic with protean clinical consequences [2]. Management depends on whether the mechanism of the stenosis is extrinsic or intrinsic.

When upper gastrointestinal interventional radiology or surgery is indicated, CA stenosis poses technical difficulties [3] or can create severe ischemia [4,5], imposing the need for good understanding of this entity in the planning of operative steps and adapted management. The goal of this update is to describe the multidisciplinary management of CA stenosis, which involves interventional radiologists, gastrointestinal and vascular surgeons and medical vascular physicians.

## DEFINITION

### **Extrinsic stenosis: median arcuate ligament compression**

Compression of the CA by the median arcuate ligament of the diaphragm, described anatomically by Lipshutz in 1917 [6], is the most frequent cause of extrinsic CA stenosis. It is estimated to be present in 10% of the population [7]. The aorta passes from the chest to the abdomen through the diaphragm via the aortic hiatus; this is bordered posteriorly by the 12th thoracic vertebra and anteriorly by the intermingled fibers of the right and left diaphragmatic crura that form the pre-aortic median arcuate ligament. Thus constituted, the aortic hiatus is osteo-tendinous and relatively rigid, avoiding any influence of respiratory movements on aortic flow. However, this fibrous band can extrinsically compress the CA, angling the first centimeter of the axis downward and flattening it against the aorta. This compression increases during

expiration and chronic compression can cause defective development of the CA with CA hypoplasia. Contributing factors include hypertrophy of the median arcuate ligament or its low-lying insertion, high origin of the CA, abnormal trajectory, and more rarely, associated nervous compression or neuroma or the celiac plexus. Several classifications have been applied to median arcuate ligament-related stenosis, but they are rarely used in current practice. One of these classifications, described specifically for the repercussions of CA stenosis on pancreatoduodenectomy (PD), specifies the degree of stenosis, its length, the distance of stenosis from the aorta and the existence of collateral circulation [8].

### **Intrinsic stenosis: atheroma**

Atheroma, constituted by deposits of lipids and sub-endothelial inflammatory cells, can cause narrowing of the CA. Atheromatous plaques develop mainly in zones of turbulent blood flow, and therefore typically arise near the take-off of the CA.

### **Other causes**

Other much rarer extrinsic causes of CA stenosis include pancreatic tumor invasion of the CA, acute pancreatitis, granuloma formation, or nerve tumors. Other intrinsic causes of CA stenosis include fibromuscular dysplasia, inflammatory diseases (Horton's disease, Takayasu arteritis, polyarteritis nodosa, isolated gastro-intestinal vascularitis without systemic involvement, idiopathic aortitis), arterial elastic tissue anomalies (Type IV Ehler-Danlos syndrome, neurofibromatosis), arterial dissections (isolated, post-traumatic, extension of aortic dissection or syndromic) or other vascular diseases (segmental arterial mediolysis).

## **DIAGNOSIS**

### **Anatomy**

The arterial supply to the upper gastro-intestinal organs is provided by the CA, SMA and their numerous longitudinal and transversal anastomoses.

Several classifications of the anatomic variations of the CA exist for which at least 14 different configurations have been described [10]. The most common configuration, a

common trunk that divides into the left gastric artery and hepato-splenic trunk, is found in 50 to 75% of individuals. Other variations include trifurcation of the CA, a hepato-splenic trunk associated with the left gastric artery arising directly from the aorta, a hepato-mesenteric trunk associated with a gastro-splenic trunk or even a celiac-mesenteric trunk.

Several anatomic variations of the collateral anastomoses between the CA and SMA can co-exist and are important to identify when managing patients with CA stenosis, particularly in interventional radiology (*Figure 1*):

- At the level of the pancreatic head and isthmus: the division of the gastroduodenal artery into the anterior and posterior (superior) pancreaticoduodenal arteries forms the pancreaticoduodenal (Rio Branco) arcade that connects to the SMA *via* one or two inferior pancreatico-duodenal arteries [1]. Their configuration is variable; classically, there are two but there can be up to four different anterior and posterior pancreaticoduodenal arteries, and one or several pancreaticoduodenal arcades can arise directly from the common hepatic artery, sometimes, coursing through the parenchyma of the pancreatic head.

In the presence of modal hepatic artery anatomy, a communication may exist between the SMA and the right branch of the proper hepatic artery, running behind the pancreas, similar to when the right hepatic artery originates from the SMA [11]. Lastly, in presence of anatomic variations of the hepatic artery [12], and in particular, in the presence of a right hepatic artery originating from the SMA or a left hepatic artery originating from the left gastric artery, communications can arise between the proper hepatic artery and the supernumerary hepatic arteries around the portal vein, within the peri-biliary plexus and in the lesser omentum. A right hepatic artery originating from the SMA typically courses along the posterior aspect of the pancreas, or even within the pancreatic parenchyma [13].

- At the level of the pancreatic body: the dorsal pancreatic artery is the first branch originating from the splenic artery in 40% of individuals [1], but it can originate directly from the CA or one of its other branches. This artery divides into several branches that are variable in number and localization:

- Longitudinally, the dorsal pancreatic artery gives rise to a branch in **direct** communication with the SMA, forming the arcade of Bühler, which is independent from the pancreaticoduodenal arcades.
  - Transversally, it can provide a branch to the head and isthmus of the pancreas (arcade of Kirk) that joins the branches of the gastroduodenal artery and a branch that courses to the tail of the pancreas (inferior pancreatic artery) and anastomoses at different levels with branches of the splenic artery.
- At the level of the tail: the branches of the splenic artery anastomose with the transversal branches of the dorsal pancreatic artery (inferior pancreatic artery).

When CA stenosis is hemodynamically significant, the flow in the supra-mesocolic arteries is modified and blood flows preferentially retrograde from the SMA to the main branches of the CA through these collateral pathways (*Figure 2*).

## **Clinical findings**

Hemodynamically significant stenosis, while usually asymptomatic, can be responsible for symptoms such as post-prandial epigastric pain that appears approximately 15 minutes after meals, along with nausea, vomiting and weight-loss. Auscultation reveals a systolic bruit in the epigastric region, which is loudest at the end of expiration. These symptoms can be related to chronic digestive ischemia.

## **Imaging: positive diagnosis / etiology / impact / associated lesions**

Definitive diagnosis rests on CT scan with IV enhancement demonstrating CA stenosis associated with downstream arterial bed dilatation. The sensitivity and specificity of CT scan are 96% and 92%, respectively [4]. Currently, traditional arteriography is no longer used for the definitive diagnosis of stenosis.

CT scan can also point toward an etiologic diagnosis of extrinsic compression by identifying a notch at the origin of the CA suggesting median arcuate ligament

compression (*Figure 3*) or of intrinsic stenosis due to an ostial atheromatous plaque (*Figure 4*).

Evaluation of the hemodynamic impact of CA stenosis on down-stream flow is fundamental before any therapeutic decision. However, contrast-enhanced CT can over-estimate this if the study is performed in full expiration, because the median arcuate ligament impinges on the aorta and increases the image of stenosis [14]. Hemodynamically significant stenosis is better evaluated by echo-Doppler examination. Several authors have published criteria for stenosis. Aburahma *et al.* [15] defined « Peak Systolic Velocity » (PSV) > 320cm/s and « End Diastolic Velocity » (EDV) > 100 cm/s as being associated with > 70% stenosis. Indirect signs of CA stenosis are flow reversal in the gastroduodenal or common hepatic arteries fed by the SMA via the pancreaticoduodenal arcades. Nonetheless, echo-Doppler can yield false results when performed by inexperienced operators or because of variations in body habitus. MRI with contrast, if available, can provide a non-invasive and reliable measure of hemodynamic variations in the gastroduodenal artery related to CA stenosis [16]. Lastly, arteriography can demonstrate whether CA stenosis is hemodynamically significant but, because of its invasiveness, it is preferable to perform this investigation secondarily, notably when treatment of stenosis is envisioned by interventional radiology.

Associated findings supporting the diagnosis include post-stenotic dilatation of the CA, dilated gastroduodenal artery, collateral arterial circulation involving the pancreaticoduodenal arteries and the dorsal pancreatic artery, and aneurysms of the branches related to high arterial flow, most often at the level of the pancreaticoduodenal arcades [17] [18]. Of note, even in the absence of stenosis, enhanced CT scan can fail to visualize the gastroduodenal artery if the study is performed sub-optimally [19]. It is important to evaluate the associated involvement of other arterial pathways, particularly the SMA that can also impact the hemodynamics of upper gastro-intestinal circulation as well as clinical findings. With regard to stenosis of atheromatous origin, other atheromatous plaques should be sought at the level of the aorta and the ostia of its main branches (SMA, renal arteries, inferior mesenteric artery).

## **MANAGEMENT**

### **Management options**

The two main management modalities available to treat CA stenosis are surgical and endovascular. Surgical management consists of either division of the median arcuate ligament, (performed preferentially via laparotomy, although mini-invasive laparoscopic or robotic approaches have been described) or arterial bypass. Endovascular management consists of dilatation of the stenosis, with or without stent insertion. The choice of the modality depends on the etiology of the stenosis and the clinical context.

### **Extrinsic stenosis**

If management of extrinsic stenosis due to median arcuate ligament compression is indicated, management is surgical. Endovascular treatment should not be the first line choice. Harjola in 1963 [20] and Dunbar in 1965 [21] published the original descriptions of surgical treatment of median arcuate ligament compression. Laparotomy is preferred over mini-invasive approaches [22,]23]. Good exposure is critical to this procedure, either by midline extension of a bilateral subcostal incision or by extension of a previous incision, particularly in the obese. Median arcuate ligament compression can be released either via an anterior approach through the lesser omentum above the CA by following the common hepatic artery and back toward the CA after identification of the left gastric artery and the splenic artery. A finger palpating along the CA from top to bottom can identify the fibrotic band formed by the median arcuate ligament. It is then divided step by step over a right-angled dissector, ensuring hemostasis progressively with successive ligations or bipolar coagulation, as needed. Once the arcuate ligament is divided, the origin of the CA and the anterior aspect of the aorta must be freed and visible. This anterior approach can be difficult because of lack of space between the antero-superior aspect of the CA and the arcuate ligament, with an increased risk of injury to the CA during dissection due to its fragility. For this reason, a lateral approach has been described [24]. This second solution requires a Kocher maneuver to mobilize the duodenopancreatic block, exposing the origin of the CA, and also the left renal vein,



the left border of the supra-renal vena cava, the proximal segment of the SMA, as well as the lymph nodes and nerve branches covering the right diaphragmatic crus. The mesenteric lymph nodes to the right of the CA should be clipped or ligated to avoid post-operative chylous leakage while the right sided nerves of the celiac plexus are coagulated. The right crus is divided to the right of the origin of the SMA to expose the median arcuate ligament which presents as a pearly white fibrotic bowstring, 1 to 3 millimeters thick. The median arcuate ligament is then divided along the right border of the SMA, avoiding the superior aspect to facilitate repair in case of a vascular injury (*Figure 5*). The efficacy of the gesture is confirmed by a test clamping of the gastroduodenal artery or an arterial echo-Doppler of the branches (*Figure 6*). If CA blood flow remains insufficient despite this liberation, the median arcuate ligament should be separated from the left and superior aspect of the CA to visualize its origin on the aorta. This maneuver should be performed with extreme caution because of the fragility of the CA in the compression zone. If after this maneuver blood flow is still insufficient, one can consider abandoning arcuate ligament division in favor of a revascularization procedure according to the original surgical indications and the associated symptomatology. Surgical revascularization can be performed with venous autograft (saphenous vein), cryopreserved arterial **graft** or prosthetic bypass. The exact site of the bypass is dictated by local conditions: aorto-celiac, aorto-hepatic, aorto-splenic [25], aorto-gastroduodenal [8] or even resection-anastomosis without interposition [26]. While surgical revascularization is sometimes essential in the context of upper gastro-intestinal surgery (in particular for PD), it is not recommended in case of diagnostic doubt or atypical symptomatology. Several retrospective series have reported the use of laparoscopic or robotic mini-invasive approaches as an alternative to laparotomy for median arcuate ligament release, reporting similar blood loss, shorter hospital stay and cosmetic advantages [27-31] These mini-invasive approaches cannot be recommended because of the risk of injury during the release procedure, a rare but severe and possibly life-threatening complication that is, above all, difficult to repair by the same mini-invasive approach [32]. Endovascular angioplasty associated with stent insertion is not recommended after surgical median arcuate ligament release. Effectively, this procedure is often difficult and dangerous because the hypoplastic tissue is fragile, exposing the patient to the risk of CA rupture during the procedure [33].

## **Intrinsic stenosis**

Three arteries can be involved: CA, SMA and inferior mesenteric artery (IMA). Most patients with digestive artery atherosclerotic disease have aortic lesions that involve two or even all three arteries. Mikkelsen's rule [34,35] stipulates that at least two of the three arteries must be involved before circulatory insufficiency leads to chronic mesenteric ischemia. However, symptomatic mesenteric ischemia from stenosis of a single digestive artery is possible if the anastomotic intercommunication between the various arteries is poor. When symptomatic, atherosclerosis involves preferentially the SMA, then the CA, and less frequently, the inferior mesenteric artery. Most often SMA involvement results in mesenteric ischemia [36]. In a meta-analysis comparing 154 angioplasties to 355 surgical revascularizations of digestive arteries, the SMA was treated in 80% of cases, the CA in 25% and the inferior mesenteric artery in 5% of instances [37].

While atheromatous stenosis of digestive arteries is frequently observed, it is often difficult to establish a clear cause-and-effect relationship between non-specific abdominal symptoms and digestive tract artery anomalies in poly-vascular patients. Treatment of the stenosis without symptom relief is dangerous, submitting the patient to an unwarranted operative risk. Indications for prophylactic revascularization procedures to avoid acute mesenteric infarction are rare and should be performed within a strictly defined framework. Lastly, portal vein pulsed Doppler can be used to verify the responsibility of digestive artery stenoses in the genesis of symptoms; it should be performed in the fasting patient, and then ½ h, one h and 1 ½ hours after a meal. Portal vein flow (in relation to body surface) should increase; no increase or minimal increase in flow confirms mesenteric arterial insufficiency. This is the only functional test available.

When surgical treatment is indicated, the choice of technique depends on atheromatous plaque localization, the number of vascular axes involved, patient condition and symptoms leading to the indication for revascularization [10].

Per-cutaneous endovascular treatment can be performed in the interventional radiology suite or in a hybrid operating room. The CA can be approached via the brachial or femoral artery. The brachial approach is often preferred because it

presents the advantage of having to negotiate only two arterial curvatures to reach the CA while the femoral approach requires the passage of three curvatures, the last of which can be often impossible when the inverted connection angle is too acute [3]. For treatment of atheromatous stenosis by transluminal balloon angioplasty alone or combined with stent insertion, can provide early satisfactory results with low morbidity and mortality. There are no absolute recommendations for platelet anti-aggregant therapy after angioplasty, with or without stent insertion, but the overall consensus [38] is to prescribe double platelet anti-aggregant therapy for one month after the operation, followed by long-term single platelet anti-aggregant therapy. The potential complications of this procedure are plaque embolization (leading to intestinal and/or splenic necrosis), CA thrombosis, CA dissection, distal migration of the stent, and puncture site complications (hematoma, pseudo-aneurysm). Balloon angioplasty seems to provide satisfactory results for atheromatous CA stenosis, irrespective of associated stent placement; conversely, the results of angioplasty associated with stent placement in the SMA seem less durable [39]. Recurrences, if detected early, can be treated by a repeat endovascular procedure before other complications develop [40,41,42].

Otherwise, surgical revascularization should be preferred, notably in case of multiple revascularizations, long-segment stenosis, in young patients, in patients with a high risk of embolism or when exploratory laparotomy is indicted. In a meta-analysis of 43 retrospective studies that included 1795 patients treated for chronic mesenteric ischemia, the success rate was high for both techniques but statistically significantly higher in the surgical group (93% vs. 95%), although at the expense of higher morbidity and mortality (13% vs. 33% and 4% vs. 7%, respectively). The rate of symptom recurrence was higher in patients who underwent endovascular treatment (21% vs. 10%) with, however, the possibility of iterative treatment. There was no difference in 5 year survival [43].

## **INDICATIONS**

Management strategies for CA stenosis outside the context of associated surgery are summarized in *Figure 7*. Management strategies within the context of associated surgery are summarized in *Figure 8*.

### **Outside the context of upper gastro-intestinal surgery:**

- **Median arcuate ligament syndrome**

The median arcuate ligament syndrome is rare, involving 2/100,000 individuals with a 5:1 female predominance [44]. The symptoms leading to diagnosis are non-specific and inconstant (post-prandial epigastric pain, nausea, vomiting, loss of weight) and are not always related to ischemic phenomena, which means that surgical indications should be proposed with caution. The presence of a median arcuate ligament compression should be confirmed by imaging, its impact on blood flow confirmed by arterial echo-Doppler, and all other entities in the differential diagnosis should be eliminated by a gastroenterologist. The degree of stenosis and history of previous abdominal surgery have been reported as risk factors for symptomatic arcuate ligament syndrome [45].

After this exhaustive workup, surgical management consists in division of the median arcuate ligament; this gesture can be insufficient to restore adequate blood flow because of the chronic character of the compression resulting in CA hypoplasia. If arterial flow restoration on echo-Doppler imaging is insufficient, either surgical bypass or careful endovascular stent insertion can be considered [46], but the major character of surgery must be weighed against the severity of symptoms and the confirmed implication of the median arcuate ligament in the symptomatology. The functional results of this surgery seem to correlate with the restoration of arterial flow and the duration of symptoms.

- **Pancreatic arcade aneurysms**

Aneurysms of the pancreatic arcade are rare but warrant recognition because their rupture can lead to life-threatening hemorrhage. They develop due to increased blood flow through the arcades in patients with hemodynamically significant CA stenosis. The clinical picture of ruptured pancreatic arcade aneurysm is non-specific

abdominal pain associated with signs of intra-abdominal hemorrhage, occasionally important and poorly tolerated. The diagnosis is suggested when enhanced CT scan demonstrates an image of hematoma surrounding a pancreatic arcade aneurysm in the pancreatic head or mesocolon (Figure 9). In the absence of CT scan visualization of the aneurysm, traditional angiography can confirm the diagnosis. Non-surgical management relies on embolization of the pancreaticoduodenal arcades *via* the SMA, while surgery is reserved for failure of endovascular treatment. Incidental discovery of pancreatic arcade aneurysm related to CA stenosis requires embolization with the same technique. There is no consensus about division of the median arcuate ligament for this indication [47,48].

- **Necessity of the endovascular approach to the CA**

As indications for supramesocolic arterial interventional radiology increase, interventional radiologists must take CA anatomic variations into account. Many indications require an endovascular catheterization of the CA branches including chemo-embolization, intra-arterial chemotherapy and radio-embolization, as well as hepatic or splenic embolizations for trauma or indications for gastroduodenal artery embolization. When it is impossible to cannulate the CA ostium, the SMA can be catheterized to find a pathway to access the hepatic artery across the pancreaticoduodenal arcades and the gastroduodenal artery. Embolization procedures (simple embolization, chemo-embolization, radio-embolization) can be performed with microcatheters whose small size optimizes the success rate; passing *via* the SMA and the pancreaticoduodenal arcades should be envisioned only in case of failure *via* the CA [49]. In case of splenic trauma with active arterial bleeding, CA stenosis can impede endovascular access to the splenic artery and lead to the need for an emergency splenectomy. For intra-arterial chemotherapy of colorectal liver metastases, the diameter of the material is larger, and CA stenosis requires catheterization through the pancreaticoduodenal arcades *via* the SMA in order to attain the proper hepatic artery [50].

**Within the context of upper gastro-intestinal surgery:**

- **Esophago-gastrectomy (type Ivor Lewis-Santy)**

The Ivor Lewis-Santy operation, or esophago- (upper pole) gastrectomy with intra-thoracic anastomosis, is indicated for cancer of the middle and lower thirds of the esophagus. It requires a carcinologic resection that preserves the vascularization of the gastric tube used for reconstruction: necrosis of the gastric tube is a rare but severe, life-threatening complication. The team of Lainas *et al.* [5], in a series of 481 Ivor Lewis-Santy interventions, reported a 2.1% rate of necrosis, leading to anastomotic fistula in 90% of cases, re-operation for 70% of these patients with a 30% mortality rate. There were statistically significantly more CA stenoses in the group with necrosis (50% with extrinsic stenosis and 80% with intrinsic stenosis) compared with 0.4% and 2.3%, respectively, in the group without necrosis. CA stenosis (atheromatous or related to the median arcuate ligament) was an independent risk factor for gastric tube necrosis in multivariable analysis. CA stenosis should therefore be sought routinely and treated before performing esophago-gastrectomy, (division of the median arcuate ligament for extrinsic stenosis or stenting of intrinsic stenosis) in case of significant hemodynamic impact on the branches of the CA. Today, certain teams use indocyanine green fluorescence to visualize the gastric tube arterial vascularization intra-operatively [51,52,53].

- **Surgery for bleeding gastroduodenal peptic ulcer**

Bleeding gastroduodenal peptic ulcer is the most common cause of upper digestive hemorrhage; management is based on medical treatment combining proton pump inhibitors, vascular repletion and endoscopic hemostasis. In rare cases of refractory hemorrhagic shock or risk of recurrence as determined by endoscopic examination (Forrest stages Ia, Ib or IIa) associated with at least two failures of endoscopic treatment [54], surgery or alternatively, selective arterial embolization of the bleeding artery is indicated [55,56]. Surgical management is more effective than embolization to stop bleeding, with no difference in morbidity or mortality or risk of rebleeding [57]. Treatment can be conservative, consisting of duodenotomy, suture of the gastroduodenal artery at the base of the ulcer, or better, above and below the pylorus, and pyloroplasty closure, or more radically, oversewing of the gastroduodenal artery and antrectomy [58]. Arteriographic embolization can be impossible anatomically if hemodynamically significant CA stenosis renders access impossible; this imposes the need for surgical management.

- **Gastrectomy distal pancreatectomy with CA resection for locally advanced cancer (Appleby and modified Appleby techniques)**

The Appleby procedure was described in 1953 for locally advanced gastric cancers; it consists of *en bloc* total gastrectomy with left pancreatectomy, splenectomy and resection of the CA, in order to enable extended lymph node dissection [59]. The modified Appleby procedure was described in 1976 for locally advanced pancreatic cancers of the body and tail [60] and entails left spleno-pancreatectomy, resection of the CA and its branches, including the common hepatic artery out to the origin of the gastroduodenal artery [61].

While these operations allow resection of tumors that were long considered unresectable, few series of Appleby resection for gastric cancer have been published [62]. This intervention has developed and is now used essentially for pancreatic cancer. The principle behind this intervention relies on the presence of collateral circulation originating from the SMA with retrograde flow through the pancreaticoduodenal arcades and the gastroduodenal artery to assure hepatic and gastric arterial vascularization. Morbidity in this procedure is dominated by hepatic ischemia (abscess, infarction) and gastric complications (perforation, gastroparesis) [63,64], and every effort must be made to minimize these risks. Test clamping of the hepatic artery, searching for persistence of pulsations in the proper hepatic artery and an echo-Doppler investigation in its branches should be performed before dividing the common hepatic artery; the absence of hepatic artery flow contra-indicates CA resection. Pre-operative embolization of the common hepatic artery, alone or associated with embolization of the two other branches of the CA, can be performed [65,66] in order to enhance the development of collateral circulation to the hepatic artery via the pancreaticoduodenal arcades and to the stomach via the right gastric and right gastro-epiploic arteries and the arcades along the greater and lesser gastric curvatures. If, despite these measure, hepatic artery blood flow remains insufficient, arterial reconstruction (bypass) is necessary [67]. Embolization of the common hepatic artery seems to decrease the need for arterial reconstructions in patients undergoing the modified Appleby procedure [68,69,70]. When the CA is narrowed by the median arcuate ligament, collateral circulation *via* the duodeno-pancreatic arcades and the gastroduodenal artery develops and allows performance of the operation in certain patients without pre-operative embolization or arterial

reconstruction [71]. The same rules detailed above must be applied and, in the absence of development of a collateral arterial network on pre-operative imaging, it is preferable to embolize the CA, sometimes *via* the SMA and the pancreaticoduodenal arcades if the CA is not passable.

- **Pancreatoduodenectomy (PD)**

This operation requires division of the gastroduodenal artery and resection of the duodeno-pancreatic arcades. CA stenosis should be sought routinely pre-operatively because, if hepatic vascularization depends on backflow through the gastroduodenal [72], division of the gastroduodenal artery exposes the patient to hepatic ischemia, gastric and pancreatic complications, and menaces the gastro-jejunal, hepatico-jejunal and pancreatico-jejunal anastomoses [73-75]. The presence of a Dilatation of the pancreatic arcades has been identified as a risk factor for post-operative complications after PD [19]. The key pre-operative examination is enhanced abdomino-pelvic CT scan which can be complemented by echo-Doppler investigation of the digestive arteries and by contrast phase MRI [16] to evaluate the downstream arterial impact.

If there is atherosclerotic intrinsic CA stenosis, endovascular maneuvers should ideally be performed before surgery [76]. For extrinsic CA stenosis, division of the median arcuate ligament, preferentially via the lateral approach is facilitated by the duodeno-pancreatic mobilization. Otherwise, an arterial bypass must be performed during the operation. A classification to guide the revascularization strategy for extrinsic CA stenosis during PD has been published [8] that separates extrinsic stenoses into three types according the degree of stenosis, its length, the distance from the aorta and the existence of arterial collateral circulation.

In each situation, the impact of CA stenosis must be evaluated at surgery by cross-clamping the gastroduodenal artery at its origin and using echo-Doppler to look for decreased or abolished pulsations or decreased flow in the proper hepatic artery [77]. This test clamping must be repeated during the operation because a negative test at the beginning of dissection does not eliminate the possibility of a symptomatic arcuate ligament compensated by the persistence of collateral circulation. In a prospective study of 545 patients before PD, Gaujoux *et al.* [4] detected 62 CA



stenoses, 7 intrinsic (of which four were significant on arterial echo-Doppler) and 55 extrinsic (27 of which were significant). Intrinsic stenoses were treated by pre-operative stent or bypass while extrinsic stenoses were treated by intra-operative division of the median arcuate ligament with an 87% success rate. Ischemic complications were diagnosed in six patients, five of whom died, representing 36% of overall mortality. This underlines the importance of pre-operative detection of arterial stenoses to allow timely treatment.

- **Liver transplantation**

In case of liver transplantation, the hepatic collateral circulation is interrupted and the CA is the only source of arterial blood supply for the liver graft, unless the gastroduodenal artery has been preserved [78]. CA compression must therefore be identified and treated to avoid the severe complications of arterial thrombosis such as ischemic cholangitis with formation of intrahepatic abscess and biliary tract stenoses that can lead to loss of the graft and require repeat transplantation or even lead to death. In a recent series of 286 orthotopic transplantations, the prevalence of extrinsic CA stenosis was 12% while that of intrinsic stenosis was 6% [79]. Pre-operative analysis of receiver CT scans is therefore primordial. The only data in the literature are retrospective cohort studies [79-83] but the approach most commonly followed is to evaluate the flow in the hepatic artery after total hepatectomy. If flow is low, a median arcuate ligament compression must be routinely suspected and released to increase the graft flow. This release will also limit the risk of a vascular steal when the stenosis is associated with liver arterial hypoperfusion dependent on the splenic artery [84] and facilitate potential interventional radiology on the hepatic artery, should that be necessary post-operatively. This procedure is easier to perform before liver implantation when exposure is enhanced, allowing an anterior approach since the Kocher maneuver is often made difficult by portal hypertension. If arcuate ligament release does not restore sufficient arterial blood flow, an arterial bypass is necessary [81]. In case of collateral circulation around the CA making median arcuate ligament release risky or prone to morbidity, performance of an initial CA bypass during the arterial reconstruction of the transplant is recommended. If intrinsic atherosclerotic stenosis is present, it must be treated before liver transplantation; if not, an arterial bypass is necessary.

In the case of living donor liver transplantation, certain authors feel that CA stenosis in the donor is a contra-indication [85]. However, this stenosis does not influence the perfusion of the graft or the remnant because neither the gastroduodenal artery, nor the pancreaticoduodenal arcades are implicated in the hepatectomy. Whereas extrinsic stenosis related to a median arcuate ligament does not seem to impact the post-operative course [86,87] and does not constitute in itself a contra-indication, atherosclerotic intrinsic stenosis is a relative contra-indication [86] because it usually implies diffuse arteriosclerosis which represents a risk factor for biliary ischemia and retransplantation in the long term [88,89].

- **Pseudo-aneurysms**

Pseudo-aneurysm is a blood collection arising from arterial wall rupture but contained by adjacent structures. This complication, observed after acute necrotizing pancreatitis or upper gastro-intestinal (pancreatic, liver or gastric) surgery, is a potential source of life-threatening hemorrhage. Treatment is based on interventional radiology embolization of the pseudo-aneurysm. CA stenosis can require embolization *via* the SMA and the pancreaticoduodenal arcades.

## **CONCLUSION**

In spite of the relatively low prevalence, ranging from 5% to 10%, celiac axis stenosis, irrespective of its etiology, is an entity that surgeons, radiologists and physicians must be aware of, because of its importance in the management of upper digestive disease. CA stenosis has to be detected and treated in a manner adapted to each situation in order to avoid complications that can potentially be severe.

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## LIST OF FIGURES

### **Figure 1. Celiac axis anatomy and anastomoses with the superior mesenteric artery**

In red: anastomoses at the level of the head and isthmus of the pancreas. In blue, corporeal anastomoses. In green, distal anastomoses

1. Aorta; 2. Celiac axis; 3. Superior mesenteric artery; 4. Left gastric artery; 5. Right gastric artery; 6. Common hepatic artery; 7. Proper hepatic artery; 8. Gastroduodenal artery; 9. Anterior superior pancreaticoduodenal artery; 10. Posterior superior pancreaticoduodenal artery; 11. Inferior pancreaticoduodenal artery; 12. Right gastro-epiploic artery; 13. Left gastro-epiploic artery; 14. Splenic artery; 15. Dorsal pancreatic artery; 16. Transverse pancreatic arcade (arcade of Kirk); 17. Anastomosis between the celiac axis and the superior mesenteric artery (arcade of Bühler); 18. Inferior pancreatic artery

### **Figure 2. Schematization of blood flow secondary to compression of the celiac axis by the median arcuate ligament. Volume rendering (VR) CT reconstruction**

Supra-mesenteric blood runs off the aorta (1) via the superior mesenteric artery (2), through the pancreaticoduodenal arcades (3) enters the gastroduodenal artery retrograde (4) to vascularize the liver via the proper hepatic artery (5). Blood flow retrograde through the common hepatic artery (6) to vascularize the other branches of the celiac axis (7) the splenic artery (8) and the left gastric artery (9).

### **Figure 3. CT scan image after injection, maximum intensity projection reconstruction; sagittal and coronal slices. Median arcuate ligament compression of the celiac axis**

Arrow: celiac axis stenosis due to median arcuate ligament compression; \*: downstream impact with dilatation of the gastro-duodenal artery

### **Figure 4. CT scan image after injection, sagittal slice and VR reconstruction. Intrinsic stenosis of celiac axis due to arteriosclerosis.**

Arrow: celiac axis stenosis atheromatous plaque; \* associated involvement of the aorta and superior mesenteric artery

### **Figure 5. intraoperative view. Lateral approach: division of right crus toward the median arcuate ligament**

1. superior mesenteric artery; 2. aorta; 3. right crus of diaphragm and edge of median arcuate ligament; 4. celiac axis

**Figure 6. Clamping test by intra-hepatic arterial Doppler**

1. before clamping of the gastroduodenal artery; 2. After clamping of the gastroduodenal artery.

**Figure 7. Management strategy for celiac axis stenosis outside the surgical context.**

LAM= MAL median arcuate ligament; SMA=superior mesenteric artery

**Figure 8. Management strategy for celiac axis stenosis within the context of surgery**

OGPS: EG esophagogastrectomy; UGD: GDU gastroduodenal ulcer; DP= PD Pancreatoduodenectomy; LA = MAL median arcuate ligament

**Figure 9. Ruptured pancreatic arcade aneurysm**

A. CT image without injection, axial slice. Retroperitoneal and retro-duodenopancreatic hematoma

B. CT image with injection, VR reconstruction coronal and sagittal slice.

arrow: celiac axis stenosis due to median arcuate ligament compression; \*: downstream impact with dysplastic aspect of the gastroduodenal artery

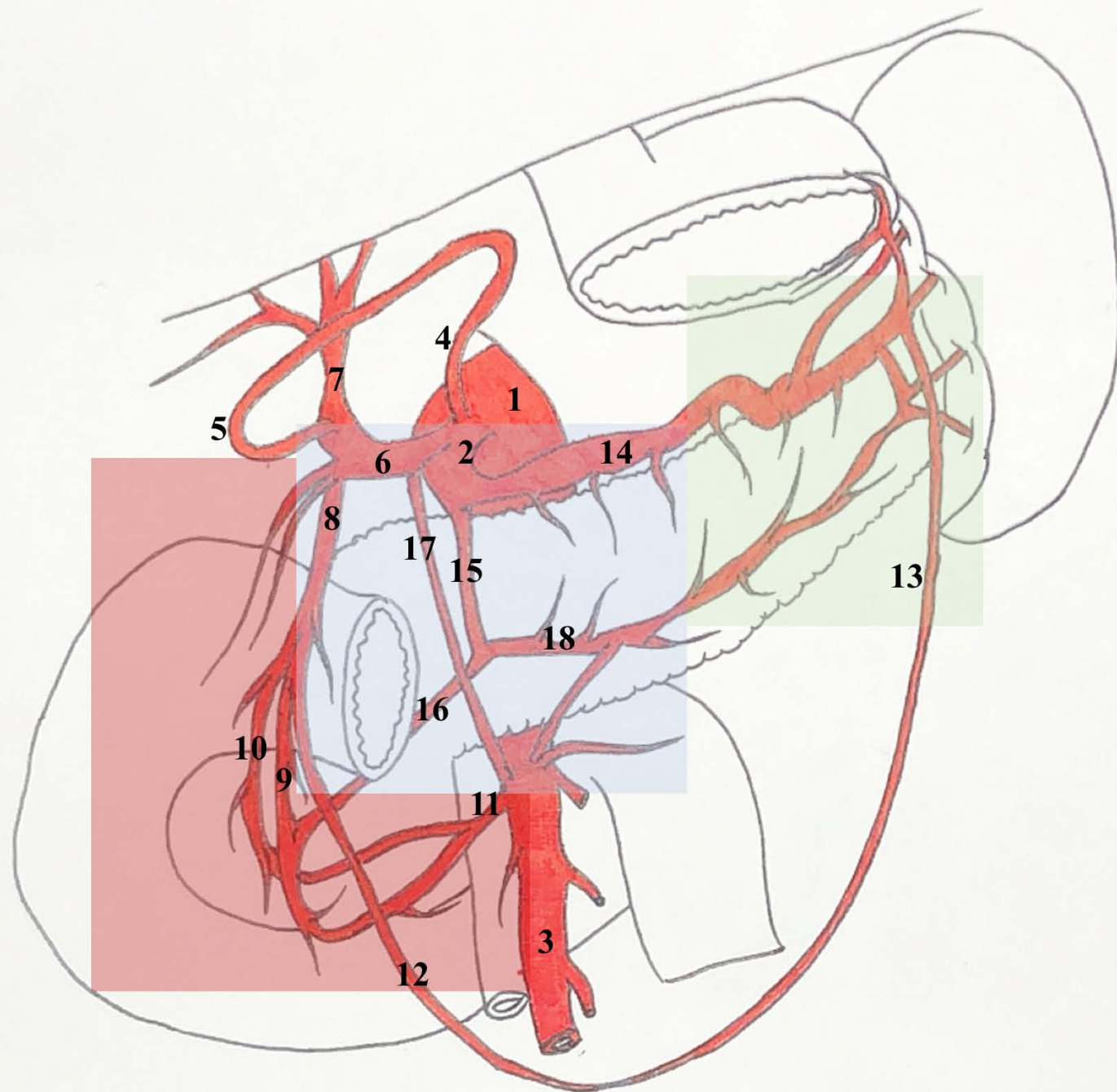
C. Celiac arteriography. Dysplastic aspect of the posterior pancreaticoduodenal artery

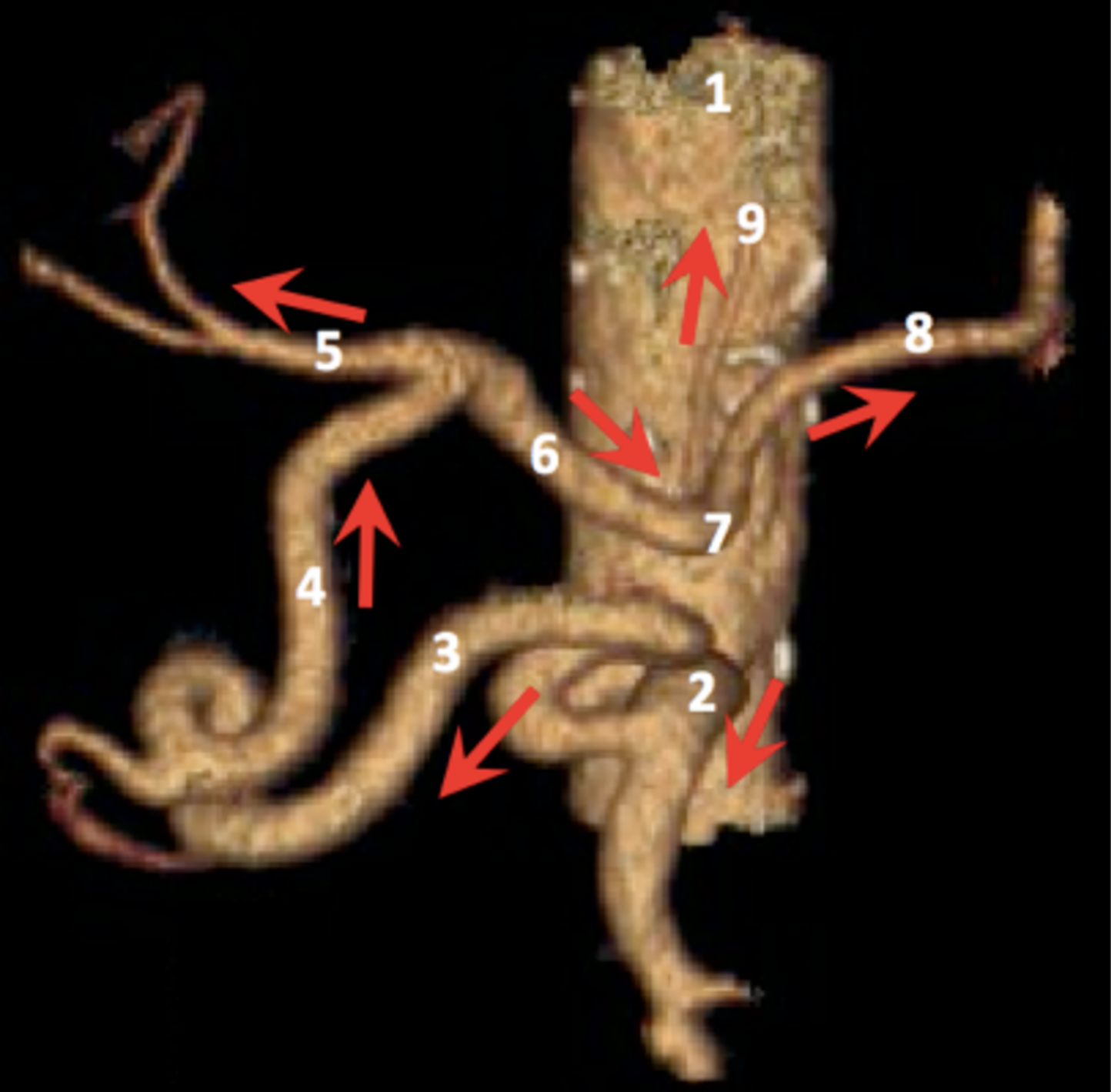
Arrow: micro-aneurysms of the posterior pancreaticoduodenal arcade

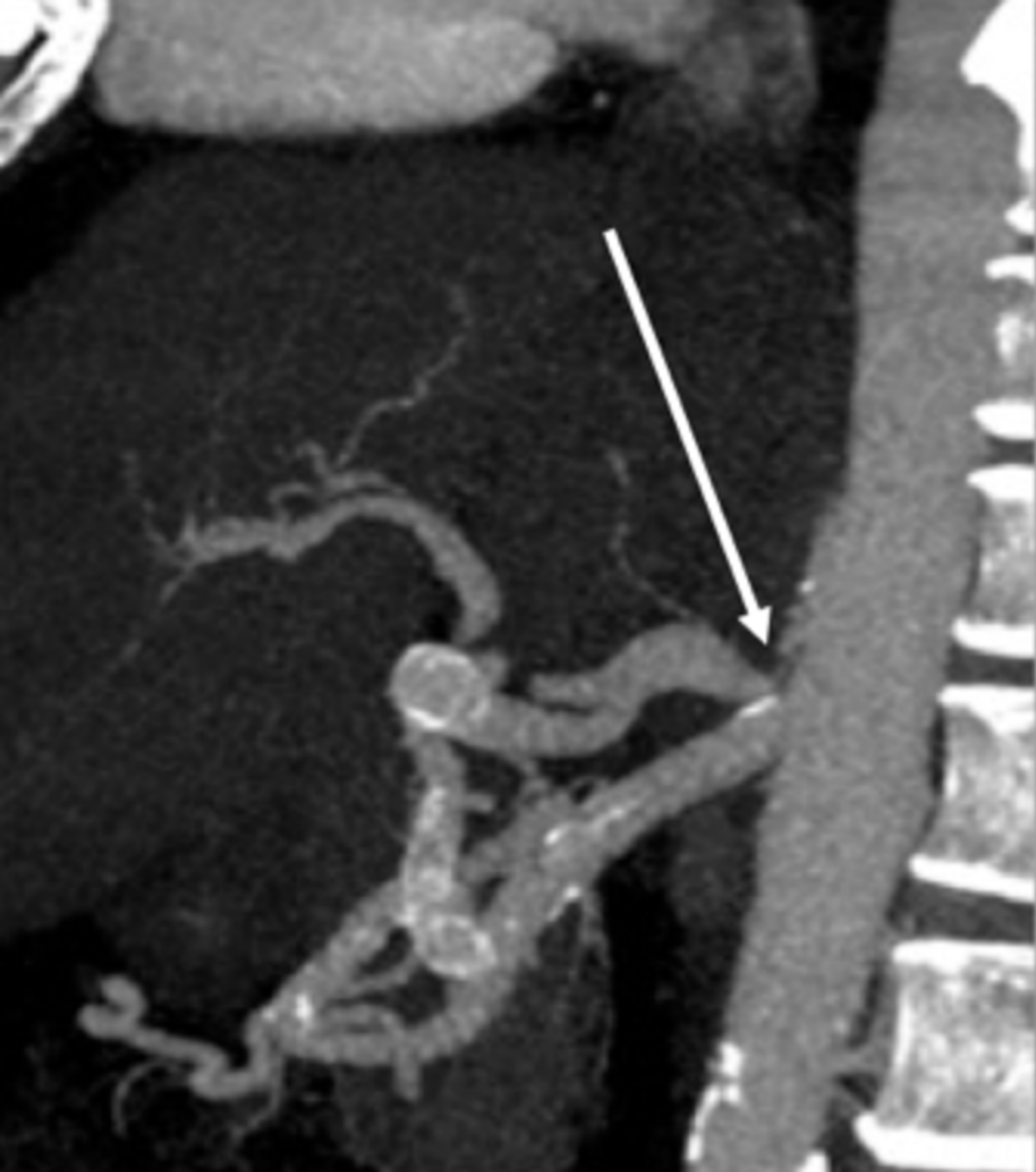
D. Celiac arteriography. Active bleeding shown by contrast extravasation, embolization of the pancreaticoduodenal arcade by micro-coils respecting the vascularization *via* the gastro-duodenal artery

Arrow: contrast material extravasation





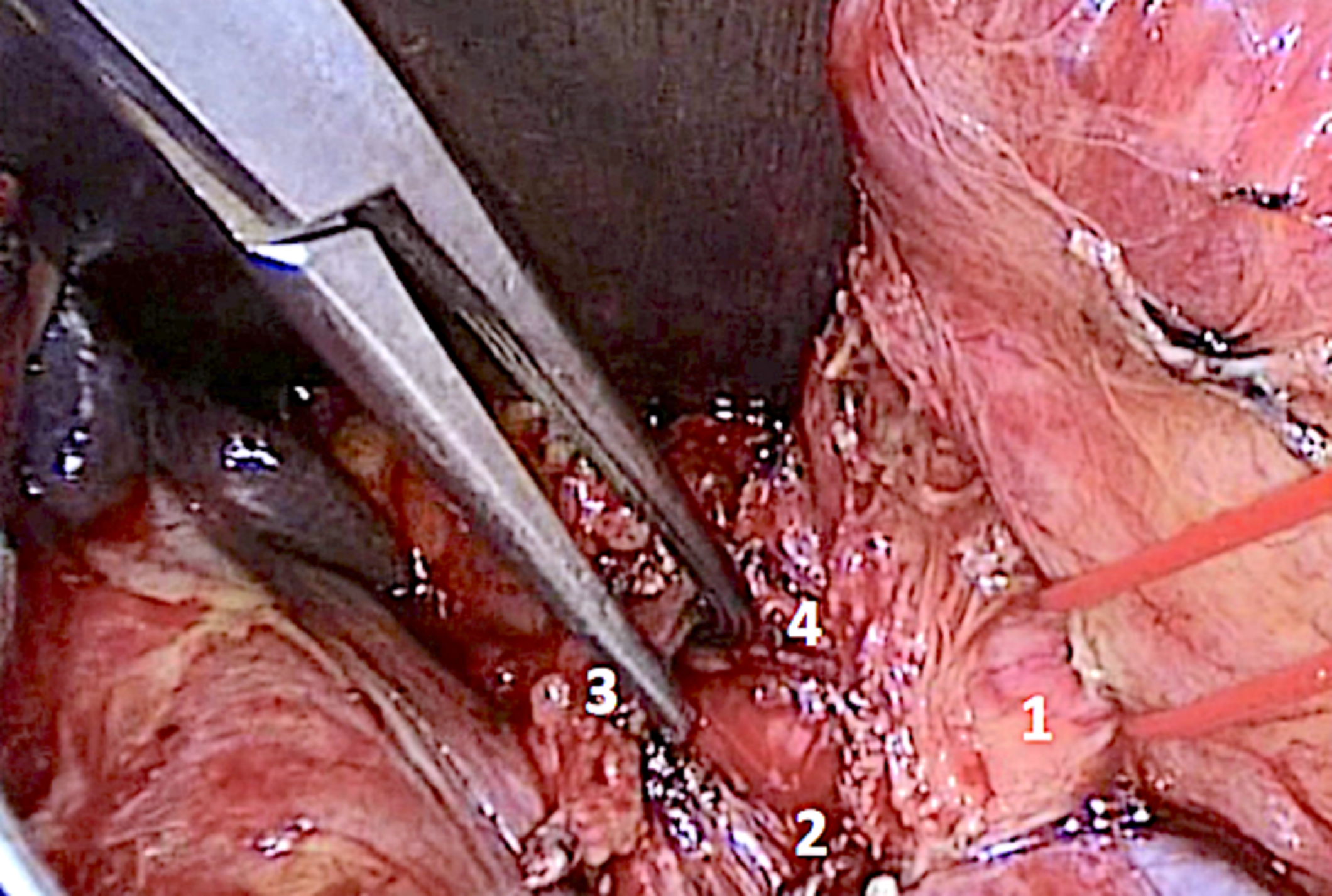












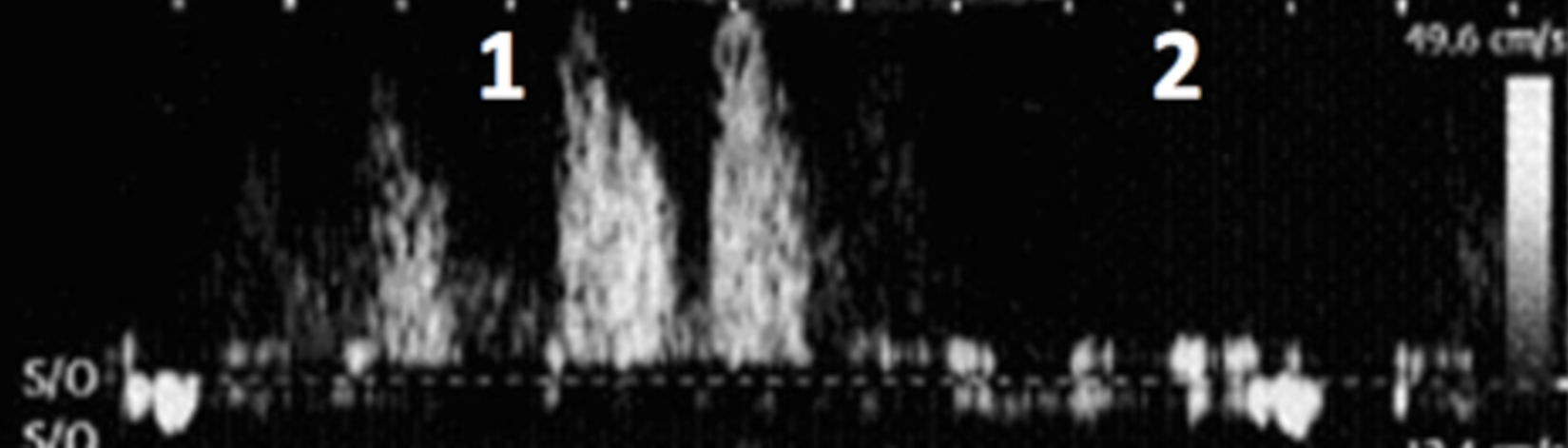


BK Medical  
 F: 1.00-1.50  
 M: 0.7-2.0  
 Rés./Hz 1/17 Hz  
 Général  
 Couleur  
 Doppler  
 Gain 50 %  
 PRF 2.0 kHz  
 1.72 cm 1.0 mm  
 60.0°



1 2

Mesures  
 S/O  
 S/O



	<b>Median arcuate ligament (MAL) syndrome</b>	<b>Pancreatic arcade aneurysm rupture</b>	<b>Celiac artery (CA), endovascular approach required</b>
<b>Stenosis</b>	<i>Extrinsic</i>	<i>Extrinsic</i>	<i>Extrinsic or intrinsic</i>
<b>Indication for management</b>	<ul style="list-style-type: none"> <li>- Symptomatology compatible with MAL</li> <li>- Hemodynamic impact</li> <li>- After extensive workup (search for differential diagnosis)</li> </ul>	<ul style="list-style-type: none"> <li>- Rupture of aneurysm</li> <li>- Incidental discovery of aneurysm</li> </ul>	Simple embolization of CA branches Intra-arterial chemo-embolization Intra-arterial radio-embolization Intra-arterial chemotherapy
<b>Therapeutic possibilities</b>	Surgical lateral approach to CA <i>via</i> laparotomy  Mini-invasive approach possible	Embolization of aneurysm <i>via</i> the SMA if CA catheterization is impossible Division of MAL considered	Use of microcatheters, embolization <i>via</i> the SMA if CA catheterization is impossible
<b>Alternatives</b>	In case of failure and valid indication, arterial bypass	Division of MAL if failure of embolization or recurrence	Surgery
<b>Consequences of no management</b>	Persistence of aspecific symptoms	Rupture, hemorrhage	Impossibility of performing what was planned

	<b>EG</b>	<b>Bleeding gastro-duodenal ulcer</b>	<b>Modified Appleby procedure</b>	<b>PD</b>	<b>Liver transplantation</b>	<b>Other</b> (inframesocolic surgery, hepatectomy)
<b>Stenosis</b>	<i>Extrinsic and intrinsic</i>	<i>Extrinsic and intrinsic</i>	<i>Extrinsic and intrinsic</i>	<i>Extrinsic and intrinsic</i>	<i>Extrinsic and intrinsic</i>	<i>Extrinsic and intrinsic</i>
<b>Indication for management</b>	Hemodynamic impact	Intractable hemorrhagic shock  Risk of re-bleeding and failure of endoscopic therapy	Absence of development of collateral circulation	Hemodynamic consequences	Hemodynamic impact  Extrinsic stenosis even without hemodynamic impact	None
<b>Therapeutic possibilities</b>	Intra-operative MAL release (lateral approach)  Pre-operative angioplasty of atheromatous stenosis	Embolization via the SMA  Surgical treatment (conservative or radical)	Intra-operative MAL release  Pre-operative angioplasty of atheromatous stenosis	Intra-operative MAL release (lateral approach)  Pre-operative angioplasty of atheromatous stenosis	Intra-operative MAL release (anterior approach) or initial bypass Pre-operative angioplasty of atheromatous stenosis	
<b>Alternatives</b>			Intra-operative arterial bypass	Intra-operative arterial bypass	Intra-operative arterial bypass	
<b>Consequences of no management</b>	Ischemic complications (gastric tube necrosis)		Complications of liver (abscess, infarction) and gastric (ischemia, gastroparesis) ischemia	Complications of liver and gastric ischemia  Anastomotic leak  Failure of embolization (hemorrhagic complications)	Thrombosis of arterial anastomosis  Complications of liver ischemia (abscess, cholangitis)  Re-transplantation	



