



Case Report

GASTRODUODENAL ARTERY ANEURYSM - A CLINICAL CASE REPORT, BRIEF LITERATURE REVIEW

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ABSTRACT

The aneurysm of the gastroduodenal artery (AGDA) is a rare, but potentially fatal vascular disease. Chronic pancreatitis is considered to be the leading etiological cause. The most common complication is rupture, which is accompanied by extremely high mortality, due to the rapidly occurring massive blood loss.

In such situations, a high index of clinical suspicion, followed by urgent imaging diagnosis and active treatment strategy in case of emergency can prevent a potentially fatal outcome.

In the following paper, we present a case of a successfully treated ruptured aneurysm of the gastroduodenal artery (AGDA), that started with minor gastrointestinal bleeding. Endoscopic hemostasis was attempted, followed by an episode of massive recurrent bleeding, requiring emergency operation.

A brief literature review, related to the diagnosis and treatment of this rare, but fatal complication of AGDA, is also presented.

Key words: gastroduodenal artery aneurysm, visceral aneurysms, endovascular treatment, coil embolization, endoaneurysmorrhaphy.

INTRODUCTION

The term "visceral aneurysms" (VA) refers to all intra-abdominal aneurysms, except those located in the aorto-iliac segment. Visceral aneurysms are a rare pathology (0.01-0.2%), but in patients with chronic pancreatitis, their frequency reaches up to 10% (1). VAs are associated with a risk of rupture in 25% of cases, and the mortality rate for the development of this complication reaches 75% (2).

Aneurysms of the gastroduodenal artery (AGDA) are among the rarest. Their frequency is about 1.5% of all VAs and is second in position after the aneurysms of the superior mesenteric artery (3). Despite their rare morbidity, they represent an important part of

the VAs, as they have a potential risk of rupture, reaching 75% and clinically evident bleeding from the gastrointestinal tract in 52% of cases (4).

With this article, we add one more to these rarely described cases of AGDA, complicated with rupture, with a brief discussion of the etiology, pathogenesis, clinical presentation and innovations in the contemporary treatment of AGDA.

THE CLINICAL CASE

An 81-year-old male was urgently admitted to the Emergency Department of Trakia Hospital, Stara Zagora, after haematemesis, occurred twice at home. He denies abdominal pain and clinical evidence of melaena. The patient reported years of chronic ischemic heart failure disease and chronic atrial fibrillation. At the time of admission, he was on therapy with Trombex 75 mg. daily and Diclofenac Duo

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(from 10 days), due to complaints of gonarthrosis.

From the clinical examination- abdomen was soft, non tender. Slightly accelerated peristalsis, without palpation evidence of organomegaly or tumor formations.

Haemodynamics - SBP -120/80, HR- 80 beats./min. Blood count - Hb. 139 g/l, Htc. 0.39, INR- 0,9.

After an emergency consultation with a gastroenterologist, the patient was hospitalized for urgent diagnostic evaluation, endoscopic verification of the source and extent of bleeding, and endoscopic hemostasis.

During the emergency video gastroscopy (VGS), a moderate amount of clear blood, regurgitating from the duodenum was found in the stomach. At the transition isthmus - D 2 segment, a large, fresh coagulum was visualized, under which clear blood flowed. After irrigation of the coagulum in the duodenal wall, an oval, pulsatile formation, lined with

intact mucosa, approximately 35 mm in diameter, was visualized. Presence of a central, ulcerated umbilicus, with active arterial bleeding - suspicion of AGDA, with the initial rupture to the gastrointestinal tract. After the insertion of a single Pentax macroclip, the bleeding stopped.

According to the findings from the VGS, urgent CT angiography was considered in order to specify the diagnosis, the localization of the process, the type of the complicated VA and to determine the subsequent treatment strategy.

The performed contrast enhanced CT angiography visualized in the hilus of the right kidney an aneurysmal expansion of 4 cm in diameter, with parietal thrombosis and a real lumen of 2.5 mm, correlating with the gastroduodenal artery (GDA). (**Figure 1**). Coeliac trunk - with ostial stenosis, branches - dilated, highly kinked, with intramural calcifications. Conclusion – AGDA!

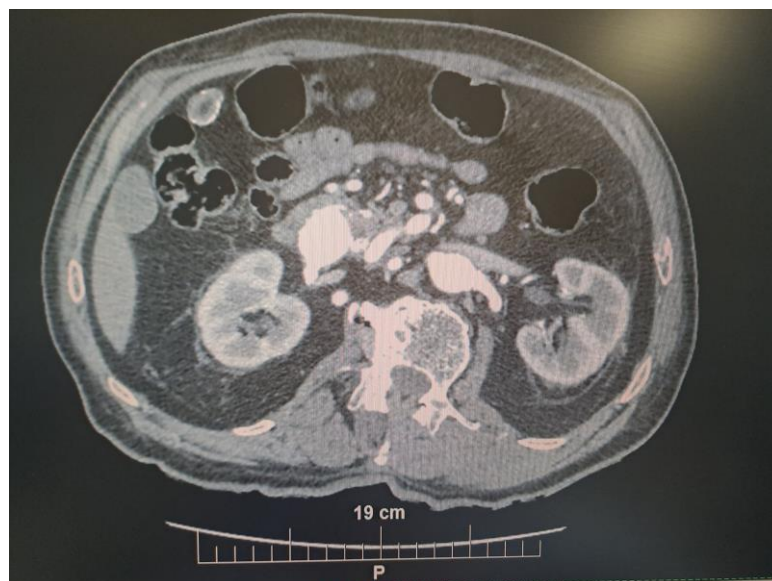


Figure. CT angio of AGDA

Followed a subsequent consultation with a vascular surgeon. A detailed analysis of the angiographic study was performed in order to plan a minimally invasive, endovascular treatment. Detailed examination revealed multiple afferent and efferent vessels to and from the AGDA, with significant kinking and coiling (**Figure 2**). The specific anatomical substrate would make it difficult to selectively cannulate the aneurysm with guidewires and

catheters, through which to perform stent-graft isolation of the AGDA. Coil embolization, aiming for definitive haemostasis would only be efficient if performed in all afferent and efferent vessels. However, such an aggressive maneuver could seriously disrupt the collateral blood circulation in this extremely dangerous area, with a potentially high risk for the development of ischemia in the structures, adjacent to the aneurysm.



Figure 2. CT angio of AGDA, with visualization of significant kinking and coiling of afferent and efferent vessels in the region of the aneurysm.

After analyzing all options for the type of intervention, it was decided that the patient requires stabilization of haemodynamic and haematological indicators, conducting relevant interdisciplinary consultations - cardiologist, anesthesiologist, general surgeon and planning a major abdominal, open surgical operation to isolate AAGD and complete definitive haemostasis.

COURSE OF THE DISEASE

Despite active medical treatment with Terlipressin in maximum doses, controlled hypotension and administration of a full amount of haemostatic medications, the patient again represented signs of acute GIT rebleeding. Clinically, haematemesis, haematochezia, collapse in haemodynamic and haematological parameters and development of a picture of progressive haemorrhagic shock were registered. This necessitated carrying out volume resuscitation, transfusion of two units isogroup blood in order to prepare the patient for an emergency, open surgical intervention by a joint team of specialists from the Vascular Surgery Department and the General Surgery Department.

OPERATIVE INTERVENTION

With longitudinal, midline abdominal incision, through the omental bursa, the duodenum was reached and mobilized by a Kocher maneuver, left to the duodenojejunal ligament. The aneurysm was located in a triangle, formed by the upper part of the duodenum, its descending part and the hepatoduodenal ligament. Several tortuous, enlarged arterial vessels, communicating with the GDA were located ventrally and dorsally. Both ventral and one dorsal vessels were ligated with 1/0 Prolene suture, at a selective site, under the AAGD. Pulsatile blood flow in the aneurysm continued. To ligate the proximally located GDA, followed mobilization of the common bile duct and its selective, supraduodenal transection. Selective ligation of the GDA, again with 1/0 Prolene suture was performed, resulting in cessation of pulsations in the aneurysm. Aneurysmotomy, evacuation of parietal thrombus, endoaneurysmorrhaphy and package with Surgicell were performed. The common bile duct was reanastomosed over a T- tube, Kehr drain, with single interrupted sutures, 3/0 Polydioxanone suture. Intraoperatively, through the inserted in the stomach nasogastric tube, after lavage with cold 0,9 % NaCl solution, was confirmed that there was currently no evidence

of ongoing active bleeding. During the intraoperative examination at the end of the intervention - no signs of end organ ischemia of the structures in the gastroduodenal region, located in the area of definitively ligated arteries, were detected.

The postoperative period went on relatively slight, without postoperative complications. No recurrent GIT bleeding was detected on the performed postoperative VGS after restoring passage. Haematological and haemodynamical indicators remained stable. The patient was discharged on the 11th postoperative day, with recommendations for trans T-tube drain cholangiography and removal of the Kehr drain within 1 month.

DISCUSSION

After Starlinger's first announcement of a diagnosed AGDA (5) in 1930, an increasing number of cases of this type of aneurysms were gradually reported, most likely due to the development and improvement of imaging diagnostics and the desire for similar pathology to be actively sought and diagnosed.

Depending on the morphology and etiology, AGDA is divided into true and false, with predominating of the latter (6). Pancreatitis and atherosclerosis are indicated as the leading etiological reasons for the development of AGDA.

During the development of acute pancreatitis, the pooling of proteolytic enzymes into the surrounding tissues can cause destruction of the wall of the adjacent arterial vessels, which is the reason for the subsequent formation of false aneurysms. The disturbed structure of the vessel wall in progressive atherosclerosis is considered the leading pathogenetic mechanism in the formation of true aneurysms of the visceral arteries. Some authors discuss another pathophysiological mechanism for the formation of AGDA. According to them, atherosclerotic stenosis of the coeliac trunk leads to an increased retrograde blood flow through the superior mesenteric artery and the pancreaticoduodenal artery, which generates increased pressure in the GDA, leading to the formation of true aneurysms along its course (7). Other, rare etiological causes, such as agenesis of coelic trunk, peptic ulcer disease, iatrogenic lesion after pancreatic head biopsy, tuberculosis are also discussed.

In our case, the most likely etiologic cause for the development and establishment of AGDA,

is the combination of atherosclerotic genesis with an existing arterial vascular malformation. The reason for the acute haemorrhage from the GIT was the gradually weakening of the duodenal wall from the expanding AGDA, followed by erosion, necrosis and massive GIT haemorrhage.

Clinically, uncomplicated AGDA are usually asymptomatic. However, their presence can be suspected in dull epigastric pain, mechanical jaundice, chronic post-haemorrhage anemia (occult bleeding). Depending on the direction of the pathological process, rupture of AGDA in the adjacent gut, is presented with haematemesis, melaena, haemobilia. When a rupture in the free peritoneal cavity occur, the clinical outcome is most often presented by severe, sudden abdominal pain and signs of haemorrhagic shock. Although very rare, AGDA can rupture into the superior mesenteric vein, with the clinical presentation of bleeding oesophageal varices (8).

Currently, with the continuous improvement of non-invasive imaging diagnostics, more and more visceral aneurysms, including AGDA, are discovered incidentally during examination on another occasion, and here ultrasound diagnostics is a reliable and inexpensive method for diagnosis and follow-up. Occasionally, the presence of acute GIT haemorrhage, in which the performed VGS cannot establish conclusive evidence of a ruptured AAGD as the source of bleeding, can become a serious diagnostic challenge. In this situation, CT angiography or direct selective angiography are the appropriate next diagnostic steps. In recent years, selective angiography has increasingly become the first choice when such type of pathology is suspected. This is due to its high specificity (up to 100%) and the possibilities for simultaneous diagnosis and endovascular treatment (9).

Once established, AGDA is amenable to definitive treatment, regardless of whether it is asymptomatic or small in size, because the increased potential for rupture is not related to size. Optimal management of AGDA depends on the clinical presentation and haemodynamic status of the patient. In asymptomatic patients or "bleeding but stable" ones, endovascular treatment - coil embolization or exclusion of the aneurysm with a "covered" stent (stent graft), provides the best therapeutic result, combined with the lowest morbidity and mortality (10, 11). Percutaneous injection of thrombin under endoscopic ultrasound control in aneurysms of

visceral arteries is a possible choice, most suitable in cases of "false" aneurysms (pseudoaneurysms), when endovascular treatment is impossible or after failed attempt (12, 13).

In a hemodynamically unstable patient, with active bleeding due to a ruptured AGDA in the GIT, endovascular treatment is also the first choice treatment. In case of failure or impossibility to conduct it (as was in our case), an emergency laparotomy is scheduled. The intervention carried out, should exclude the aneurysm from the blood flow by ligation of all related vessels. Particular attention should be paid to the intraoperative review, because in the presence of an arterial malformation, ligation of only the afferent and efferent arteries may be insufficient to interrupt the blood flow in the aneurysm. Subsequent aneurysmotomy is mandatory, as leaving an unrecognized branch with an active blood supply to the aneurysm, may eventually lead to rebleeding. The intraoperative assessment of available ischemia of the structures, vascularised from the GDA basin, determines whether the intervention should be completed without (ligation of the aneurysm) or with vascular reconstruction (resection with end-to-end anastomosis, resection with interposition, preferably with vein, less often with vascular prosthesis).

CONCLUSIONS

Due to their rarity, AGDA is rarely suspected clinically as an etiological cause of acute GIT bleeding. The lack of a conclusive finding of the source of bleeding on VGS could raise serious doubt about the presence of an unusual cause of the incident. In a case with a ruptured AGDA, recurrent minimal bleeding from the GIT may be a predictor of catastrophic haemorrhage, and therefore diagnostic efforts and therapeutic behavior should be directed toward active measures to prevent such a life-threatening situation.

Today, endovascular procedures in the case of AGDA are the preferred first choice treatment method. Transcatheter coil embolization or endograft placement are reliable, safe, and effective treatment options in patients with preserved, stable haemodynamics. In our case, despite the detailed preoperative analysis of the CT angiography results and the available endovascular treatment options, the specific anatomic substrate due to an aneurismal arterial malformation with several arterial branches emerging from it and the progressive

deterioration of the patient's status and haemodynamic failure, necessitated urgent surgery. This fact supports the thesis that, when the expected result from endovascular intervention is questionable, open surgery is the only alternative for a successful outcome, despite the higher operative risk involved. The case presented by us is an evidence for the need to be mastered both endovascular and open surgical interventions equally, in order to provide the most appropriate team solutions in such difficult and life threatening situations.

ABBREVIATIONS

AGDA- Aneurysm of gastroduodenal artery
 VA- Visceral aneurysms
 VGS- Videogastroscopy
 GDA- Gastroduodenal artery
 GIT- Gastrointestinal tract
 SBP- Systolic blood pressure
 HR- Heart rate

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