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CASE REPORT

GASTRODUODENAL ARTERY PSEUDOANEURYSM DUE TO PANCREATITIS

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ABSTRACT: Gastroduodenal artery (GDA) aneurysms are rare but a potentially fatal condition if rupture occurs. They represent about 1.5% of all visceral artery (VAA) aneurysms and are divided into true and pseudoaneurysms depending on the etiologic factors underlying their development. Atherosclerosis and pancreatitis are the two most common risk factors. Making the diagnosis can be complex and often requires the use of Computed Tomography and angiography. The latter adds the advantage of being a therapeutic option to prevent or stop bleeding. If this fails, surgery is still regarded as the standard for accomplishing a definite treatment.

KEYWORDS: Gastroduodenal artery, Pseudoaneurysm, Pancreatitis, Endovascular coiling.

INTRODUCTION: Pancreatitis is a very common entity found in clinical practice but gastroduodenal artery aneurysm is a rare complication. It is usually asymptomatic in initial stage but may become fatal. So it should be cured as soon as possible according to its size before it gets ruptured. Endovascular and open surgery are two modalities of its treatment. Endovascular treatment is preferred nowadays.

CASE PRESENTATION:

ON ADMISSION: Patient came to CHA on 28th May, 2013 with complains of abdominal pain, vomiting, abdominal distension, altered bowel habits and jaundice. Patient was chronic alcoholic and cigarette smoker. No associated history of DM, TB, hypertension or previous surgery. Patient had same complain before 1 year for which he was managed conservatively. In September, 2012, patient had undergone EUS with FNAC and EUS guided aspiration which was suggestive of chronic pancreatitis. Aspiration fluid amylase and CEA levels were raised.

Patient's general body habitus was thin, icteric, nail & sclera with normal temperature, pulse, BP, RS, CVS and CNS findings. Abdomen was soft without signs of peritonitis and Intestinal obstruction. Tenderness was present in epigastrium and right subcostal region. No cyst or lump was palpable per abdomen.

CLINICAL COURSE: Patient was kept on broad spectrum antibiotics, analgesics and antacids. Total (D+I) S. Bilirubin 9 (5+4) mg/dl, SAP- 239 IU/L, SGPT- 18 IU/L, SGOT- 27 IU/L, S. Protein (A+G) 6.2 (2.5+3.7) d/dl, S. Amylase- 30 U/L, S. Lipase- 73 U/L, S. Calcium- 6.85 mg/dl, CA 19.9- 44 U/ml. Chest and Abdominal x-rays were normal. CECT Abdomen with Pelvis was done. It showed pancreatic parenchymal calcifications and MPD dilated & tortuous in body and tail region suggestive of chronic pancreatitis. Thrombosed pseudoaneurysm of gastroduodenal artery was found with mass effect on portal vein and CBD. Liver enlarged, IHBT dilated and GB was distended. Moderate ascites was present.

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Diagnosis of gastroduodenal artery pseudoaneurysm as a complication of chronic pancreatitis was made. Patient underwent endovascular coiling of gastroduodenal artery on 1st June, 2013. On 5th day of coiling patient had developed abdominal distension, abdominal pain and vomiting. Blood investigations were suggestive of S. Protein (A+G) 4.8 (2.7+2.1), S. Bilirubin (D+I) 9.5 (7.6+1.9), SGPT-8, SAP- 97.

CECT Abdomen with pelvis was done. It showed GB posterior wall defect suggesting perforation, changes of chronic pancreatitis and large thrombosed aneurysm of gastroduodenal artery with mass effect, metallic coil, gross ascites and bilateral pleural effusion.

ERCP suggest sloughed out mucosa with ruptured? pseudoaneurysm in duodenum, lots of altered blood and coiling in side cavity of pseudoaneurysm. Ascitic fluid examination suggested transudate fluid with significant presence of bile salt and bile pigment.

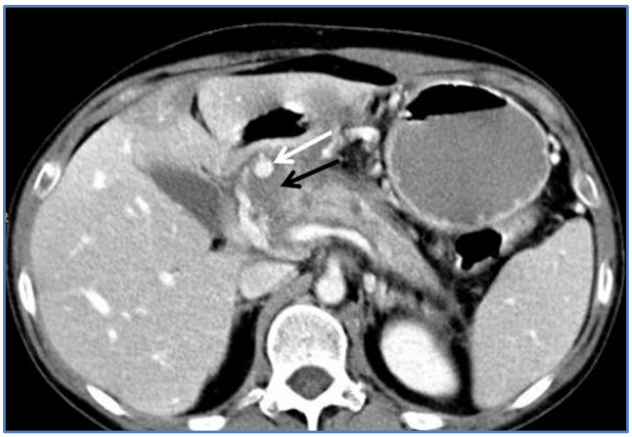

Figure 1

Diagnosis of GB perforation as a complication of endovascular coiling with biliary peritonitis was made. Patient underwent emergency exploratory laparotomy on 10th June, 2013. Intraoperative 1200 ml ascitic fluid was aspirated and GB was found to be perforated with fragile GB wall. Partial cholecystectomy and tube cholecystostomy was done. Patient developed post-operative diabetes insipidus. It was managed by daily fluid input/output monitoring and IV fluids. Patient was discharged on 9th post-operative day with normal vitals and urine output.

DISSCUSSION:

EPIDEMIOLOGY: Gastroduodenal artery (GDA) aneurysms are rare but a potentially fatal condition if rupture occurs. They represent about 1.5% of all visceral artery (VAA) aneurysms and are divided into true and pseudoaneurysms depending on the etiologic factors underlying their development. Atherosclerosis and pancreatitis are the two most common risk factors.

Making the diagnosis can be complex and often requires the use of Computed Tomography and angiography. The latter adds the advantage of being a therapeutic option to prevent or stop bleeding. If this fails, surgery is still regarded as the standard for accomplishing a definite treatment.

PATHOPHYSIOLOGY: The pathogenesis of GDA aneurysms is not fully understood. Trauma, hypertension and atherosclerosis have been cited as potential risk factors for true aneurysms. The

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pathophysiologic changes that underlay the development of true gastroduodenal artery aneurysms comprise mainly atherosclerosis of the celiac artery with subsequent stenosis but also rarely congenital absence of the celiac axis. These two circumstances can be distinguished by the morphology of the collaterals that develop.

The collateral vessels that form early on have usually parallel walls, are of uniform caliber and are limited to one or two vessels. In contrary, arteriosclerotic collaterals are more abundant, dilated and tortuous and hence more prone to aneurysmal formation within the vessel's wall. This occurs regardless of the location of the stenosis.

The pancreaticoduodenal artery is the main collateral pathway between the celiac axis and the superior mesenteric artery. Increased blood flow in the pancreaticoduodenal artery, as compensation for celiac artery stenosis, may cause a pancreaticoduodenal artery aneurysm. The same theory suggests that occlusion or stenosis of the superior mesenteric artery or celiac axis could be an etiologic factor predisposing to the formation of a gastroduodenal artery aneurysm.

As for pseudoaneurysms, inflammation with the most common cause being pancreatitis, results in vascular wall destruction that is mediated by pancreatic proteolytic enzymes leading to pseudoaneurysm formation[1].

CLINICAL PRESENTATIONS: After reviewing the literature extending from1956 to 2011, 74 cases describing GDA aneurysms were collected from the Japanese and English literature. A gastrointestinal hemorrhage secondary to rupture of the aneurysm was found to be the most common clinical presentation (52%) while only 7.5% of GDA aneurysms remained asymptomatic. Abdominal pain is the second most common symptom and occurs in 46% of cases. The mortality rate with rupture is about 40%[2] and depends on the severity, speed of the blood loss and the anatomical site of the rupture.

The highest mortality rate comes from rupture into the duodenum approaching 21%. These patients present with hematemesis, melena, and hemodynamic shock[3]. Less frequently, patients with GDA can present with retroperitoneal or intra-peritoneal bleeds with a 19% mortality rate. This could lead to gastric outlet obstruction and other nonspecific symptoms such as vomiting, diarrhoea and jaundice secondary to compressive hematoma or external pressure by the aneurysm.

In addition, bleeding into the pancreatic duct manifesting as recurrent episodes of hemosuccus pancreaticus have been reported as well as bleeding into the common bile duct. The presence of a pulsatile abdominal mass with or without a bruit on auscultation could be the sole warning sign and should raise the suspicion of a GDA aneurysm with prompt diagnostic work up to preclude the worst outcome.

DIAGNOSIS: Prior to the era of sophisticated imaging modalities the majority of cases of GDA aneurysms were undiagnosed until rupture occurred. Currently with the various imaging studies available, an increasingly larger number of cases are being incidentally detected in asymptomatic patients.

The gold standard diagnostic test is visceral angiography and it serves both diagnostic and therapeutic purposes by delineating the arterial anatomy and allowing therapeutic intervention. It has the highest sensitivity (100%) followed by computed tomography (CT) (67%) and ultrasonography (US) (50%). CT scan has the advantage of being non-invasive and localizing the

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aneurysm with its relations to surrounding structures. When performed in a patient with pancreatitis, CT scan can reveal a homogeneously enhancing structure within or adjacent to a pseudocyst which is highly suggestive of an associated pseudoaneurysm[4]. Three-dimensional CT adds to the accuracy of the study.

Doppler US may reveal turbulent arterial blood flow within or adjacent to a pseudocyst which is also suspicious for an aneurysm.

Plain X-ray of the abdomen is a rarely helpful study but may show shell-like calcifications in an atherosclerotic aneurysm.

New modalities such as Contrast-enhanced 3-dimensional magnetic resonance angiography or multi-detector row computed tomography have been reported to be as effective as visceral angiography in the diagnosis of abdominal vascular lesions. Other diagnostic studies are available including Pulse Doppler US, color Doppler US, endoscopic ultrasound and magnetic resonance imaging but are less frequently used.

TREATMENT: Once a GDA aneurysm ruptures, the patient faces a life threatening condition that could rapidly lead to death in 40% of cases.[2] Therefore, it is of utmost importance to diagnose and treat GDA aneurysms before a fatal complication occurs. Such a complication is not always related to the size of the aneurysm and therefore treatment should be planed as soon as a diagnosis is made.[5] Therapeutic strategies include surgical (revascularization, vessel ligature, aneurysmal sac exclusion) or endovascular interventions (coil embolization, stent placement).

The choice of the therapeutic procedure is made on individual basis and depends on the presenting symptom, the location of the aneurysm, and general condition of the patient and the risk of organ ischemia after the intervention.[5, 6] The traditional therapy of visceral artery aneurysms has been the surgical resection or ligation of the aneurysm. Recently, endovascular treatment, such as trans-catheter embolization, has been an alternative to open surgical repair and has become increasing popular.

Open surgical approach of patients suffering from a VAA is a safe and life-saving procedure. In 88.2% of patients, the treatment remained successful after a mean follow-up of 54 months. Moreover, emergent surgery is the treatment of choice in case of aneurysmal rupture in a hemodynamically unstable patient and consists of ligation, aneurysmorrhaphy or bypass surgery. If the condition of the patient allows it, the less invasive endovascular options should be exhausted before proceeding with surgery. If bleeding recurs or cannot be controlled, vascular surgery will still be a feasible alternative. Vascular reconstruction after exclusion of the aneurysm is not always necessary, as collaterals almost always exist between the visceral arteries.

For example, vascular supply to the stomach comes from the both the GDA and the Superior Mesenteric Artery (SMA). Hence, vascular reconstruction is not essential after resection of a GDA aneurysm unless there is celiac artery occlusion, as ligation of the GDA may cause gangrene of the gallbladder and stomach, splenic necrosis or other disastrous consequences.[6, 7] However, adequate collateral flow should be documented with preoperative imaging if permitted. CT and magnetic resonance angiography demonstrate excellent resolution for preoperative planning but angiography may allow better evaluation of real time flow dynamics.

For those patients with celiac artery or SMA stenosis, trans-luminal angioplasty would be one way of avoiding the risk of organ ischemia before or after surgical resection of the aneurysm. In cases

of erosion into the surrounding bowel structure, endoscopic techniques to identify and stop the source of bleed can be attempted initially as long as the patient's condition allows it.[6] Therefore, the endovascular option being a less aggressive approach, performed under local anesthesia offers a good therapeutic alternative for those patients who are unfit for surgical treatment due to incapacitating comorbidities and is associated with a shorter hospital stay.

Endovascular options include embolization of the aneurysms or stent graft deployment[8]. Some anatomical conditions are required though for technical feasibility of these procedures (saccular aneurysm with a narrow neck, fusiform aneurysm with adequate collateral flow, aneurysm of a vessel supplying an organ that has multiple arterial sources).[8]

Even though this less aggressive option plays an important role in high risk surgical candidates, it has its potential complications such as visceral ischemia resulting in sacrifice of the involved visceral vessel, end-organ thrombosis, and late-term vessel recanalization. Trans catheter embolization is the most popular endovascular intervention performed despite the potential risk of visceral ischemia and organ infarction.

Other complications include coil/stent migration, intra-procedural aneurysm dissection, or rupture[9], embolisms, access artery pseudoaneurysms and contrast-induced nephropathy. In contrast, surgical interventions have their own share of complications such as paralytic ileus, wound infection, massive bleeding, or acute pancreatitis. These complications have a significantly higher rate of occurrence in patients with previous abdominal surgery where adhesions are present, making the endovascular approach the preferred treatment option in those patients.[7]

Despite the fact that endovascular treatments do not represent a standard option and require both a specific training and a learning curve, the development of new technologies, such as the multilayer stent, could offer a new alternative to VAA treatment, particularly in high-risk patients.[9]

CONCLUSION: In conclusion, physicians might only encounter GDA aneurysms as an incidental finding on CT scans. In unfortunate patients, rupture might occur and lead to a fatal outcome if an emergent intervention is not made. Depending on the patient's condition, the decision to proceed with angiography or surgery should be taken without any delay to prevent the worst outcome.

REFERENCES:

1. John A. Windsor, Benjamin P.T. Loveday. Complications of Acute Pancreatitis. Maingot's Abdominal Operations, 2013, 12th edi.(1), 1139
2. Begert H, Hinterseher I, Kersting S, Leonhardt J, Bloomenthal A, Saeger HD. Management and outcome of hemorrhage due to arterial pseudoaneurysms in pancreatitis. Surgery 2005, 135(3):323-328. Epub 2005/03/05
3. Rowsell C, Moore TL, Streutkar CJ. Aneurysm of the gastroduodenal artery presenting as a bleeding duodenal ulcer. Clin Gastroenterology Hepatol Offic Clin Pract J Am Gastroenterological Assoc 2006, 4(10): xxviii. Epub 2006/06/08
4. Gary C. Vitale, Brian R. Davis. Pancreatic Pseudocyst: Laparoscopic or Endoscopic versus Conservative Surgery. Mastery of Surgery, 2012, 6th edi.(1), 1442
5. Koyazounda A, Jaillot P, Persico J, Thouret JM, Grand A. Aneurysm of the gastroduodenal artery ruptured into the peritoneum. Treatment by embolization. Presse Medicale (Paris, France 1983 1994, 239(14): 661-4

DOI: 10.14260/jemds/2014/2396

6. Quandalle P, Chambon JP, Marache P, Saudemont A, Maes B. Pancreaticoduodenal artery aneurysm associated with stenosis of the superior mesenteric artery. Ann Vasc Surg 1994, 8(3):281-284. Epub 1998/05/20
7. Germanos S, Soonawalla Z, Stratopoulos C, Friend PJ. Pseudoaneurysm of the gastroduodenal artery in Chronic pancreatitis. J Am Coll Surg 2004, 18(6):695-703. Epub 2004/12/16
8. Sachdey U, Baril DT, Ellozy SH, Lookstein RA, Silverberg D, Jacobs TS. Management of aneurysms involving branches of the celiac and superior mesenteric arteries: a comparison of surgical and endovascular therapy. J Vasc Surg Offic Publ Soc Vasc Surg Int Soc Cardiovasc Surg North Am Chapter 2006, 44(4):718-724. Epub 2006/10/03
9. Takahashi T, Shimada K, Kobayashi N, Kakita A. Migration of steel-wire coils into the stomach after transcatheter arterial embolization for a bleeding splenic artery pseudoaneurysm: report of a case. Surg Today 2001, 31(5):458-462. Epub 2001/05/31
10. Ferrero E, Ferri M, Viazzo A, Robaldo A, Carbonatto P, Pecchio A. Visceral artery aneurysms, an experience on 32 cases in a single center: treatment from surgery to multilayer stent.

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