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## Mesenteric Vascular Disease

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### 1. Introduction

Mesenteric ischemia occurs when perfusion of the visceral organs fails to meet normal metabolic requirements. This disorder is categorized as either acute and chronic, based on the duration of symptoms. Acute mesenteric ischemia (AMI) occurs rapidly over hours to days and frequently leads to acute intestinal infarction requiring resection.

### 2. Anatomy of the visceral arteries

The celiac artery arises from the abdominal aorta just caudal to the diaphragm at the level of L1 and is bordered by the median arcuate ligament at the aortic hiatus superiorly and the superior border of the pancreas inferiorly. Traditionally, the three branches from this common trunk include the left gastric, splenic, and common hepatic arteries. However, multiple variations of the true "trifurcation" can exist. Most frequently, the common hepatic artery and its branches arise from the SMA or directly from the abdominal aorta (1).

The SMA arises a few centimeters caudal to the celiac trunk, and its origin is crossed by the neck of the pancreas and the splenic vein.

The IMA is usually located 3 to 4 cm cephalic to the aortic bifurcation, just to the left of midline, and usually arises at the level of the third lumbar vertebra.

#### 2.1 Acute mesenteric ischemia

##### 2.1.1 Embolism

The most common cause of AMI is embolization to the SMA. Arterial emboli are responsible for 40% to 50% of cases of AMI (2, 3, 4). The proximal source of the embolus is frequently intracardiac mural thrombus. Mural thrombus in proximal aneurysms in the thoracic or proximal abdominal aorta can also serve as embolic sources. Because the SMA arises at a less acute angle from the abdominal aorta compared with the other mesenteric vessels, it appears to be the most common final destination for mesenteric emboli. Additionally, such emboli tend to lodge several centimeters from the vessel's origin, usually distal to the middle colic artery.

##### 2.1.2 Arterial thrombosis

Arterial thrombosis constitutes the next most common cause of AMI and occurs in 20% to 35% of cases (4, 5). Preexisting atherosclerotic plaque affecting all visceral vessels is the

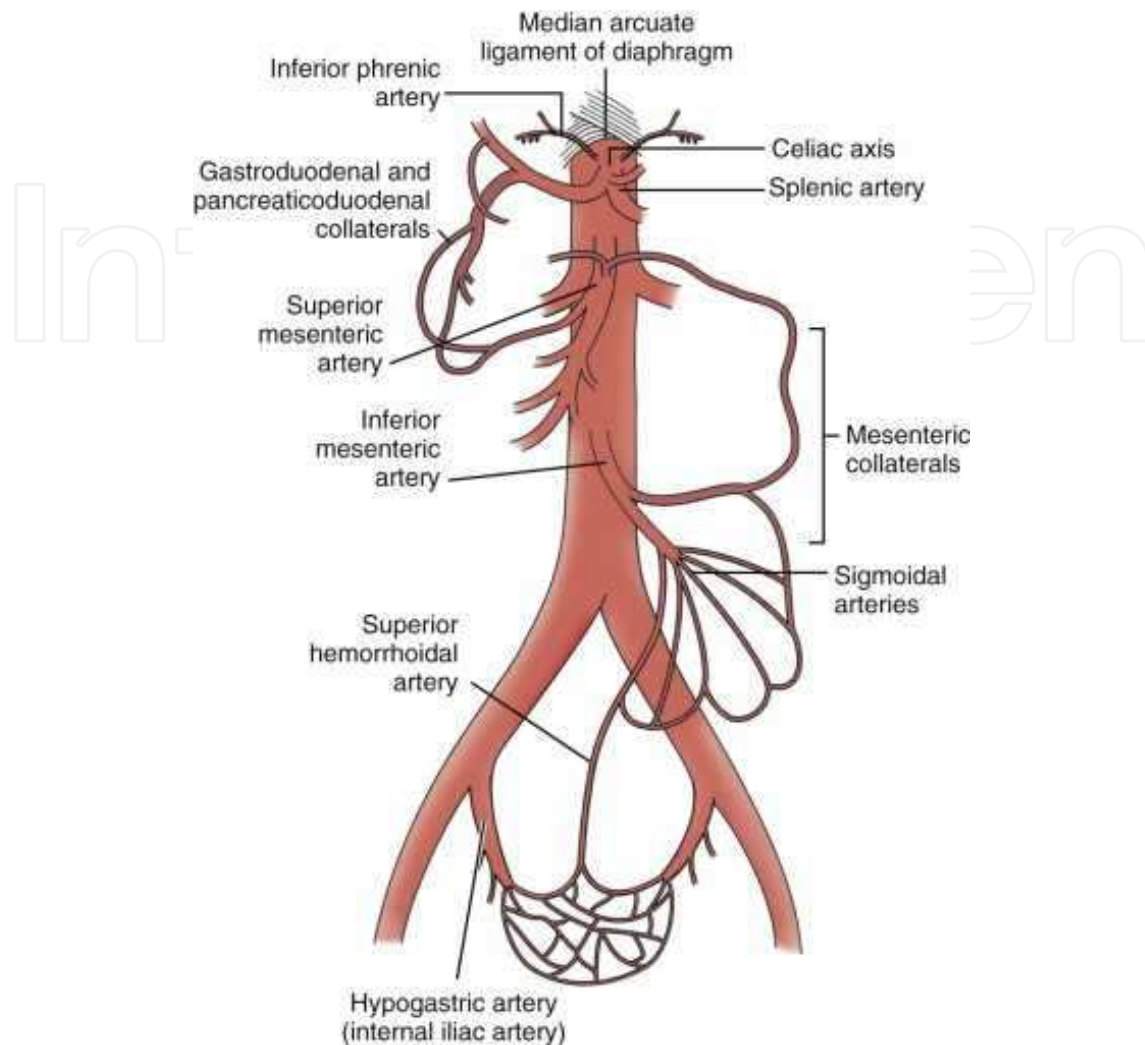


Fig. 1. Anatomy of the visceral arteries

most common finding. The affected segment of artery is usually its origin at the level of the aorta. Patients with acute arterial thrombosis frequently have preexisting symptoms of CMI. Acute extension of an aortic dissection can also serve as a mechanism for abrupt mesenteric vessel occlusion and thrombosis. The degree of intestinal infarction was significantly greater in patients with SMA thrombosis compared with embolus.

### 2.1.3 Nonocclusive mesenteric ischemia

Impaired intestinal perfusion in the absence of thromboembolic occlusion is termed nonocclusive mesenteric ischemia (NOMI). Symptomatic patients are frequently found to have extensive atherosclerosis, with involvement of all three visceral arteries. However, NOMI can also occur in patients without mesenteric arterial occlusive disease (6, 7, 8). Visceral ischemia can occur due to low-flow states, especially in conjunction with intestinal atherosclerotic disease. NOMI most commonly occurs secondary to cardiac disease, particularly severe congestive heart failure (9).

### 2.1.4 Mesenteric venous thrombosis

MVT constitutes 5% to 15% of all cases of mesenteric ischemia (10). Involvement is usually limited to the superior mesenteric vein but can also involve the inferior mesenteric vein and portal vein. The extent of bowel ischemia depends largely on the degree of venous involvement. Inherited or acquired hypercoagulable diseases, including protein-C and -S deficiency, polycythemia vera, antithrombin III deficiency, antiphospholipid antibody syndrome, and factor V Leiden mutation, are frequent causes.

## 3. Clinical presentation

### 3.1 Acute mesenteric ischemia

The most common symptom of AMI associated with arterial thromboembolic disease is the sudden onset of abdominal pain. Lack of collateral flow to the visceral organs leads to a more dramatic presentation in AMI, with severe, rapid clinical deterioration. Nausea, vomiting, diarrhea, emptying symptoms, and abdominal distention can also occur. Patients with NOMI or MVT typically present with a slower clinical course. Frequently, patients with NOMI are critically ill, hospitalized, intubated patients who experience a sudden deterioration in their clinical condition.



Fig. 2. Acute Mesenteric Ischemia (intraoperative photograph)

### 3.2 Chronic mesenteric ischemia

Postprandial abdominal pain and progressive weight loss are the most common symptoms in patients with CMI. Pain is often described as dull and crampy and located in the midepigastic region. The course of symptoms can be equated with intestinal claudication. Lack of energy leads to failure of the intestinal smooth muscle to relax, which intensifies the cramping pain. Pain often occurs 15 to 45 minutes after a meal, and the severity varies according to the size and type of meal. Patients typically develop “food fear” and decrease their oral intake in anticipation of severe pain after meals. Changes in bowel habits, nausea,

and vomiting are less common findings. CMI is believed to be more prevalent in elderly women (11). The variable nature of symptoms often makes the diagnosis confusing and can result in delayed treatment. The traditional risk factors for atherosclerosis are usually present. A heavy smoking history is frequently obtained. The majority of patients also have a history of symptomatic manifestations in other vascular beds, most commonly cerebrovascular, coronary, and peripheral arteries.

Physical examination findings are usually nonspecific. Patients are commonly undernourished and cachectic. An abdominal bruit can sometimes be auscultated but is not always present. Bowel sounds are frequently hyperactive. Guarding and rebound tenderness are usually absent. Low prealbumin and albumin levels are often seen, owing to the patient's chronic malnourished state.

## **4. Diagnostic evaluation**

### **4.1 Noninvasive evaluation**

Duplex ultrasonography is a useful tool for the early, noninvasive diagnosis of visceral ischemic syndromes. Color Doppler scanning can be used to assess the flow velocities and resistance index in the splanchnic arteries and their arterial beds, as well to evaluate end-organ vascularity (12).

Computed tomography (CT) is an accurate, noninvasive imaging modality for diagnosing mesenteric ischemia, CTA diagnosed AMI with a sensitivity of 96% and a specificity of 94%.

Magnetic resonance angiography (MRA) is useful for diagnosing mesenteric occlusive disease. Because MRA takes significantly longer to perform than CTA, its role in evaluating patients with AMI is limited.

### **4.2 Invasive evaluation**

Conventional angiography remains the "gold standard" in the diagnosis of mesenteric ischemia. Anteroposterior and lateral views of the visceral aorta as well as selective catheterization of the celiac trunk, SMA, and IMA, provide the most accurate and specific localization of stenotic and occlusive lesions. Therapeutic alternatives such as balloon angioplasty, stenting, and thrombolysis and percutaneous thrombus extraction can all be used to restore luminal visceral blood flow.

## **5. Treatment of acute and chronic mesenteric ischemia**

Medical treatment alone is not effective in these patients. Preventive risk factor modification helps control the progression of atherosclerosis in the mesenteric circulation as well as other vascular beds. Patients with known risks for inheritable hypercoagulable disorders should undergo screening and should be treated with systemic anticoagulation if indicated.

### **5.1 Endovascular treatment**

Advances in endovascular techniques have greatly expanded the role of percutaneous interventions for patients with mesenteric ischemia in recent years. However, endovascular



management remains largely limited to patients with CMI. Balloon angioplasty and stenting are the most common interventions, and recent reports have documented excellent technical results with low patient morbidity. Endovascular therapy should be the treatment of choice in high-risk patients with CMI (13, 14, 15, 16). High technical success rates and decreased patient morbidity and mortality rates have been reasonably well established in such individuals.

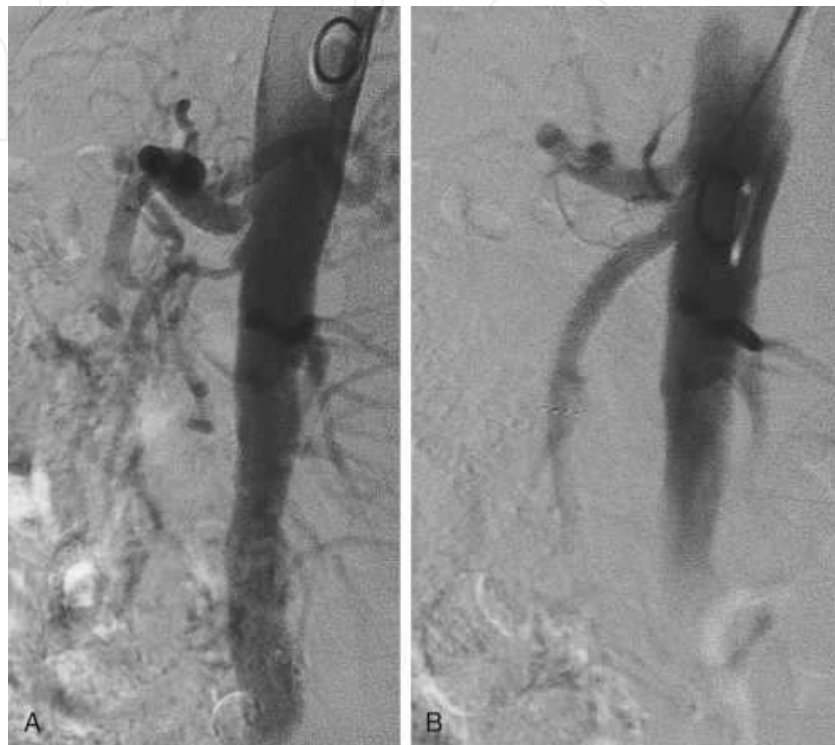


Fig. 3. A, Lateral arteriogram of the celiac axis and superior mesenteric artery. Note the mild orificial stenosis in the celiac axis and the severe stenosis in the proximal superior mesenteric artery. B, Completion study after angioplasty and stenting of the superior mesenteric artery stenosis. Note the widely patent superior mesenteric artery, with no evidence of stenosis

## 5.2 Surgical treatment

Laparotomy with visceral revascularization can be used to treat patients with both AMI and CMI. Patients presenting with signs and symptoms of AMI require urgent abdominal exploration, assessment of bowel viability, and revascularization. Several techniques for the restoration of intestinal perfusion are available to the vascular surgeon, and familiarity with a variety of options is crucial. Before revascularization, large segments of both small and large intestine may appear dusky, ischemic, or necrotic.

## 5.3 Acute mesenteric ischemia

### 5.3.1 SMA embolectomy

Perfusion of the mesenteric arteries is assessed by palpation and Doppler evaluation. In cases in which the obstruction is caused by an embolus, a proximal SMA pulse is often

appreciated. Systemic heparinization is established. If the artery feels relatively soft and free of atherosclerotic disease, a transverse arteriotomy is performed distal to the area of obstruction, and the arterial lumen is assessed for thrombus. Balloon-tipped embolectomy catheters are gently passed proximally and distally until no more clot can be removed. Care must be taken not to overinflate the balloons and dissect the arterial intima. Distally, mesenteric vessels are very thin, and overinflation can result in rupture and intramesenteric extravasation. The transverse arteriotomy is then closed primarily with simple interrupted Prolene sutures if no endarterectomy is necessary. In cases in which a flow-limiting plaque is present, the arteriotomy is converted to a longitudinal one, and a local thromboendarterectomy is performed. Patch angioplasty with autogenous vein is the preferred method of revascularization owing to potential contamination from concomitant bowel resection. The arteriotomy site can also be used for distal anastomosis of an antegrade or retrograde bypass if necessary.

## 5.4 Chronic mesenteric ischemia

### 5.4.1 Transaortic endarterectomy

Advantages of this operation include removal of atheroma from the aorta and both visceral arteries simultaneously. Limitations include the need for extended exposure of the upper abdominal aorta via medial visceral rotation and incomplete plaque removal if the atheroma extends to the distal artery or if transmural calcification is present. It is suitable for selected patients with CMI undergoing elective revascularization (17).

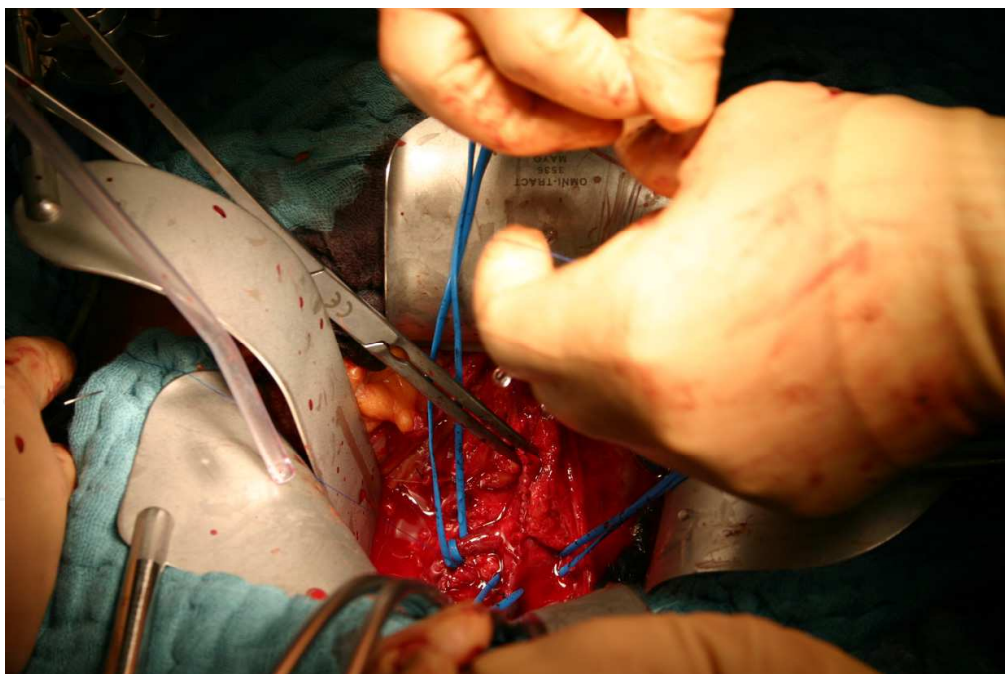


Fig. 4. Endarteriektomie of the SMA

### 5.5 Antegrade mesenteric bypass

Reconstruction of the celiac artery and the SMA with a bifurcated prosthetic graft originating from the supraceliac aorta. The operation is done through an upper midline or

bilateral subcostal incision, depending on the patient's body habitus and costal cartilage flare. Supraceliac-origin grafts are a poor choice in patients with compromised cardiac or pulmonary function or those with extensive atherosclerosis or circumferential calcification of the supraceliac aorta. In these cases, infrarenal sources of inflow are preferred (18, 19, 21).

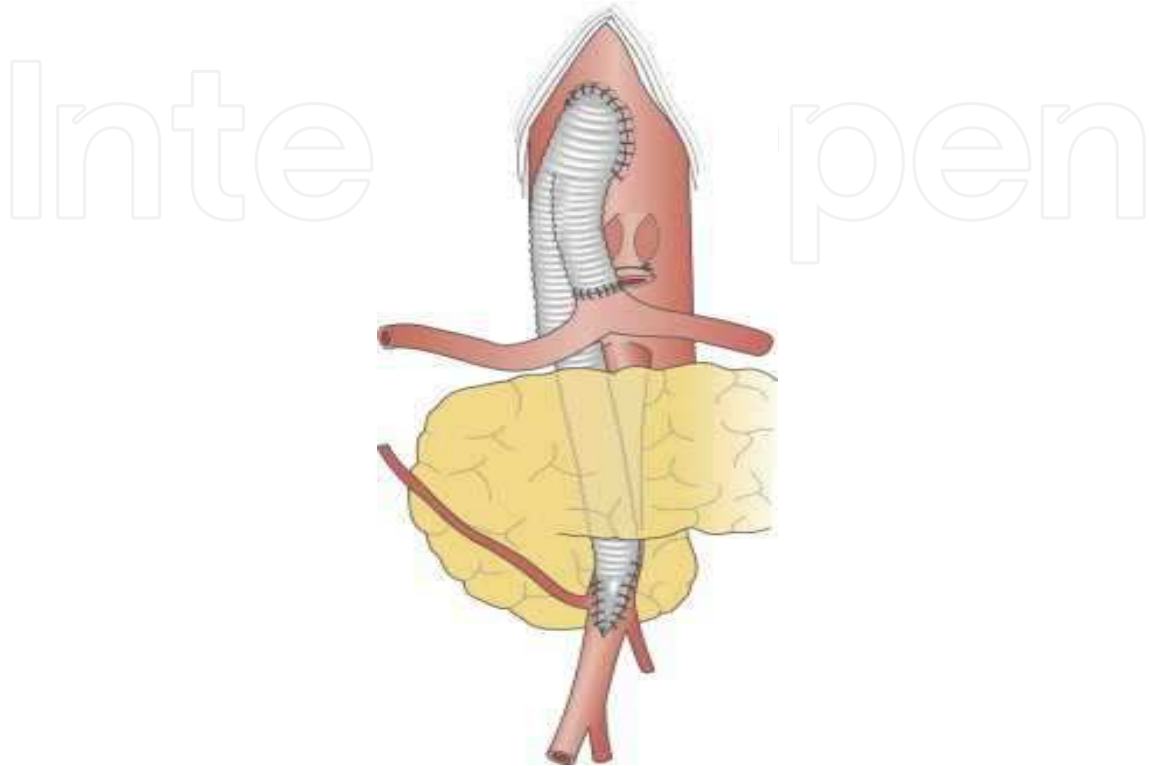


Fig. 5. Antegrade aortoceliac-superior mesenteric artery bypass

### 5.6 Retrograde mesenteric bypass

The infrarenal aorta, a prior infrarenal aortic graft, or the iliac artery are excellent inflow sources. Two-vessel reconstructions can be performed with retrograde grafts by doing a side-to-side anastomosis to the SMA and an end-to-side anastomosis to the common hepatic artery. These grafts may be passed on top of or beneath the pancreas and curved in a C shape toward the hepatic artery.

## 6. Treatment of nonocclusive mesenteric ischemia

The primary treatment for NOMI is medical, with extensive critical care support and prompt arteriography. Operative exploration is reserved for signs of peritonitis that suggest the presence of gangrenous bowel that requires excision. Interventional therapies can be initiated at the time of the diagnostic arteriogram and are targeted at relieving vasospasm using intra-arterial infusions of vasodilator medications. The most common intra-arterial agent is phosphodiesterase inhibitor papaverine and prostaglandin (22). Surgical exploration is required for all patients who have evidence of any threatened bowel, regardless of the underlying cause. The prognosis is poor, despite the absence of organic obstruction in the principal arteries.



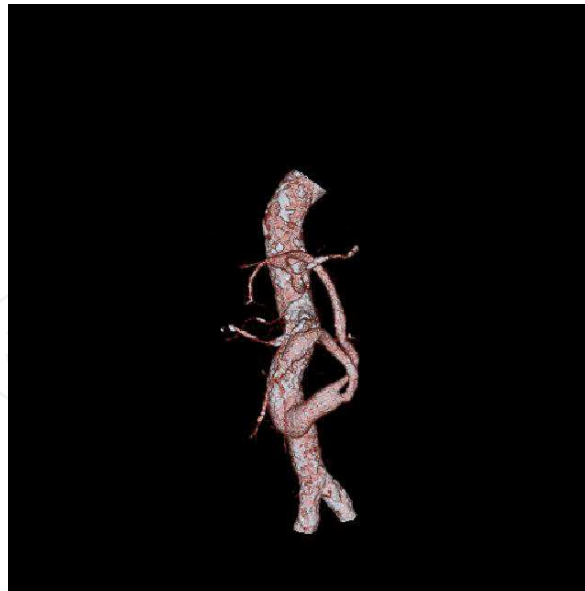


Fig. 6. Retrograde aortoceliac-superior mesenteric artery bypass

## 7. Treatment of mesenteric venous thrombosis

Initial anticoagulation with heparin is the treatment of choice in patients without peritonitis. After initial anticoagulation, continued treatment with low-molecular-weight heparin (LMWH) or VKA is advocated. Uncertainties about bowel viability are assessed through laparotomy or laparoscopy; it is safer to perform a laparotomy to check for bowel viability in patients with signs of peritonitis and rebound tenderness. Endovascular treatment in combination with heparin infusion, with or without bowel resection, is an additional treatment tool (23, 24, 25, 26, 27). The indications for surgery are peritonitis, severe gastrointestinal bleeding, late small bowel perforation, and intestinal stricture; the last is often associated with chronic diarrhea.

### 7.1 Splanchnic artery aneurysms

True aneurysms of splanchnic arteries are less common than visceral artery pseudoaneurysms, but they remain an important vascular disease. Nearly 22% of these present as clinical emergencies, including 8.5% that result in death (28). The pathogenesis and natural history of these aneurysms have been reassessed, and in most instances redefined, within the past three decades as advances in imaging technology and endovascular treatments have begun to influence diagnostic and management strategies. Recognition of splanchnic artery aneurysms has increased because of the greater availability and widespread use of advanced imaging capabilities such as high-resolution computed tomography (CT) scanning, magnetic resonance angiography (MRA), sophisticated ultrasonography, and angiography. Selective arteriography remains the most valuable examination in planning therapy but noninvasive imaging techniques for diagnosis and operative planning are becoming increasingly important.

Although surgery remains the mainstay of therapy for most splanchnic aneurysms, especially in the setting of rupture, many aneurysms (particularly those involving solid organs) are now treated with catheter-based interventions. Endovascular approaches are

commonly used to control the bleeding that accompanies aneurysm rupture (29), and prophylactic treatment of incidentally discovered intact aneurysms has become common (particularly those well-collateralized aneurysms that are imbedded within the pancreatic or hepatic parenchyma). Embolization has become the preferred treatment in patients at high surgical risk or for aneurysms in locations that are difficult to approach surgically.

Arterial Location	Incidence of Aneurysms
Splenic	60.0%
Hepatic	20.0%
Superior mesenteric	5.5%
Celiac	4.0%
Gastric or gastroepiploic	4.0%
Jejunal, ileal, or colic	3.0%
Pancreaticoduodenal or pancreatic	2.0%
Gastroduodenal	1.5%
Inferior mesenteric	Rare

Table 1. Incidence of Aneurysms of the Splanchnic Arterial Circulation (30, 31)

### 7.2 Splenic artery aneurysm

The most common of the splanchnic artery aneurysms and account for as many as 60% of all reported splanchnic aneurysms (32). The most common clinical risk factors reported in association with Splenic Artery Aneurysm are female gender, a history of multiple pregnancies, and portal hypertension. A classic calcified ring may be noted in the left upper quadrant on a plain x-ray film of the abdomen. The patients may have an abdominal bruit, the majority of physical examinations are normal in patients with asymptomatic lesions. When rupture occurs, patients usually complain of acute left-sided abdominal pain. Shock, abdominal distention, and death can result from free intraperitoneal rupture of an Splenic Artery Aneurysm. The overall mortality of ruptured Splenic Artery Aneurysm is high (33).

Splenic aneurysms that have ruptured or are symptomatic require urgent treatment. Additionally, aneurysms in pregnant women or those of childbearing age also absolutely warrant treatment. Less stringent indications for treatment include aneurysms that are noted to be enlarging or those greater than 2 cm in diameter.

Endovascular exclusion of has been used more recently with general success. Treatment options include coil embolization of the splenic artery both proximal and distal to the aneurysm itself, thereby effectively "trapping" the lesion (34, 35).

### 7.3 Hepatic artery aneurysms

The hepatic artery is the second most common location for aneurysmal degeneration in the splanchnic circulation. The causes are degenerative ("atherosclerotic"), medial degeneration,

fibrodysplasia, trauma, infection, biliary diseases and percutaneous or endoscopic procedures, polyarteritis nodosa, and congenital disorders.



Fig. 7. Splenic Artery Aneurysm (intraoperative photograph)

Symptoms can include epigastric or right upper quadrant pain and subsequent gastrointestinal hemorrhage and jaundice. Treatment options depend to a large extent on the anatomic location and morphology of the Hepatic Artery Aneurysms, underlying etiology, and status of the end organ (36, 37, 38, 39).

#### **7.4 Celiac artery aneurysms**

In contrast to Splenic Artery Aneurysms are Celiac Artery Aneurysms more commonly found in men. The causes are medial degeneration or atherosclerotic disease. Less common causes include trauma, collagen vascular disease, arterial dissection, anomalous splanchnic circulation, and mycotic aneurysms. Open surgical options included aneurysmectomy, aneurysmorrhaphy, and ligation (40).

#### **7.5 Superior mesenteric artery aneurysm**

Most commonly found within the first 5 cm of the artery,, these aneurysms are particularly dangerous because complications such as aneurysm rupture, acute thrombosis, or distal embolization may jeopardize the entire small bowel.

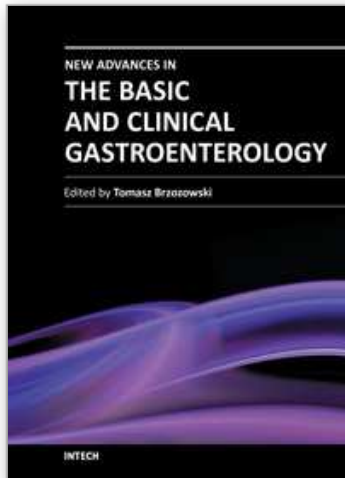
Superior Mesenteric Artery Aneurysm associated with an infectious etiology in the majority of cases (Mycotic aneurysms). The majority of Superior Mesenteric Artery Aneurysms are symptomatic at initial evaluation. Treatment of Superior Mesenteric Artery Aneurysms should be considered regardless of size or symptomatology because of the high mortality risk associated with potential rupture. treatment must be individualized and based on the etiology, size, and anatomic location of the lesion; co-morbid conditions of the patient; and the potential morbidity of the proposed procedure (41, 42).

## 8. References

- [1] Rosenblum JD, Boyle CM, Schwartz LB: The mesenteric circulation: anatomy and physiology. *Surg Clin North Am* 1997; 77:289-306.
- [2] Foley MI, Moneta GL, Abou-Zamzam AM, et al: Revascularization of the superior mesenteric artery alone for treatment of intestinal ischemia. *J Vasc Surg* 2000; 32:37-47.
- [3] Stoney RJ, Cunningham CG: Acute mesenteric ischemia. *Surgery* 1993; 114:489-490.
- [4] McKinsey JF, Gewertz BL: Acute mesenteric ischemia. *Surg Clin North Am* 1997; 77:307-318.
- [5] Park WM, Gloviczki P, Cherry Jr KJ, et al: Contemporary management of acute mesenteric ischemia: factors associated with survival. *J Vasc Surg* 2002; 35:445-452.
- [6] Bradbury AW, Brittenden J, McBride K, et al: Mesenteric ischaemia: a multidisciplinary approach. *Br J Surg* 1995; 82:1446-1459.
- [7] Mansour MA: Management of acute mesenteric ischemia. *Arch Surg* 1999; 134:328-330.
- [8] Oldenburg WA, Lau LL, Rodenburg TJ: Acute mesenteric ischemia: a clinical review. *Arch Intern Med* 2004; 164:1054-1062.
- [9] Thomas JH, Blake K, Pierce GE, et al: The clinical course of asymptomatic mesenteric arterial stenosis. *J Vasc Surg* 1998; 27:840-844.
- [10] Kumar S, Sarr MG, Kamath PS: Mesenteric venous thrombosis. *N Engl J Med* 2001; 345:1683-1688.
- [11] Schwartz LB, Gewertz BL: Mesenteric ischemia. *Surg Clin North Am* 1997; 77:275-507.
- [12] Dietrich CF, Jedrzejczyk M, Ignee A: Sonographic assessment of splanchnic arteries and the bowel wall. *Eur J Radiol* 2007; 64:202-212.
- [13] Sivamurthy N, Rhodes JM, Lee D, et al: Endovascular versus open mesenteric revascularization: immediate benefits do not equate with short-term functional outcomes. *J Am Coll Surg* 2006; 202:859-
- [14] Sharafuddin MJ, Olson CH, Sun S, et al: Endovascular treatment of celiac and mesenteric arteries stenosis: applications and results. *J Vasc Surg* 2003; 38:692-698.
- [15] Sarac TP, Altinel O, Kashyap V, et al: Endovascular treatment of stenotic and occluded visceral arteries for chronic mesenteric ischemia. *J Vasc Surg* 2008; 47:485-491.
- [16] Brown DJ, Schermerhorn ML, Powell RJ, et al: Mesenteric stenting for chronic mesenteric ischemia. *J Vasc Surg* 2005; 42:268-274.
- [17] Lau H, Chew DK, Whittmore AD, et al: Transaortic endarterectomy for primary mesenteric revascularization. *Vasc Endovasc Surg* 2002; 36:335-341.
- [18] Jimenez JG, Huber TS, Ozaki K, et al: Durability of antegrade synthetic aortomesenteric bypass for chronic mesenteric ischemia. *J Vasc Surg* 2002; 35:1078-1084.
- [19] Kansal N, LoGerfo FW, Belfield AK, et al: A comparison of antegrade and retrograde mesenteric bypass. *Ann Vasc Surg* 2002; 16:591-596.
- [20] Kougias P, Lau D, El Sayed HF, et al: Determinants of mortality and treatment outcome following surgical interventions for acute mesenteric ischemia. *J Vasc Surg* 2007; 46:467-474.
- [21] Mateo RB, O'Hara PJ, Hertzner NR, et al: Elective surgical treatment of symptomatic chronic mesenteric occlusive disease: early results and late outcomes. *J Vasc Surg* 1999.821-832.
- [22] Mitsuyoshi A, Obama K, Shinkura N, et al: Survival in nonocclusive mesenteric ischemia: early diagnosis by multidetector row computed tomography and early

- treatment with continuous intravenous high-dose prostaglandin E1. *Ann Surg* 2007; 246:229-235.
- [23] Lopera JE, Correa G, Brazzini A, et al: Percutaneous transhepatic treatment of symptomatic mesenteric venous thrombosis. *J Vasc Surg* 2002; 36:1058-1061.
- [24] Kim HS, Patra A, Khan J, et al: Transhepatic catheter-directed thrombectomy and thrombolysis of acute superior mesenteric venous thrombosis. *J Vasc Interv Radiol* 2005:1685-1691.
- [25] Zhou W, Choi L, Lin PH, et al: Percutaneous transhepatic thrombectomy and pharmacologic thrombolysis of mesenteric venous thrombosis. *Vascular* 2007; 1:41-45.
- [26] Grisham A, Lohr J, Guenther JM, et al: Deciphering mesenteric venous thrombosis: imaging and treatment. *Vasc Endovasc Surg* 2005; 39:473-479.
- [27] Semiz-Oysu A, Keussen I, Cwikiel W: Interventional radiological management of prehepatic obstruction of [corrected] the splanchnic venous system. *Cardiovasc Intervent Radiol* 2007; 30:688-695.
- [28] Pasha S.F., Glociczki P., Stanson A.W., Kamath P.S.: Splanchnic artery aneurysms. *Mayo Clinic Proc* 2007; 82(4):472-479.
- [29] Gabelmann A., Gorich J., Merkle E.M.: Endovascular treatment of visceral artery aneurysms. *J Endovascular Therapie* 2002; 9(1):38-47.
- [30] Ruiz-Tovar J., Martinez-Molina E., Morales V., et al: Evolution of the therapeutic approach of visceral artery aneurysms. *Scand J Surg* 2007; 96(4):308-313.
- [31] Huang Y.K., Hsieh H.C., Tsai F.C., et al: Visceral artery aneurysm: risk factor analysis and therapeutic opinion. *Eur J Vasc Endovasc Surg* 2007; 33(3):293-301.
- [32] Miani S., Arpesani A., Giorgetti P.L., et al: Splanchnic artery aneurysms. *J Cardiovasc Surg (Torino)* 1993; 34(3):221-228.
- [33] Carr S.C., Mahvi D.M., Hoch J.R., et al: Visceral artery aneurysm rupture. *J Vasc Surg* 2001; 33(4):806-811.
- [34] Grego F.G., Lepidi S., Ragazzi R., et al: Visceral artery aneurysms: a single center experience. *Cardiovasc Surg* 2003; 11(1):19-25.
- [35] Kanazawa S., Inada H., Murakami T., et al: The diagnosis and management of splanchnic artery aneurysms: report of 8 cases. *J Cardiovasc Surg (Torino)* 1997; 38(5):479-485.
- [36] Lumsden A.B., Mattar S.G., Allen R.C., Bacha E.A.: Hepatic artery aneurysms: the management of 22 patients. *J Surg Res* 1996; 60(2):345-350.
- [37] Abbas M.A., Fowl R.J., Stone W.M., et al: Hepatic artery aneurysm: factors that predict complications. *J Vasc Surg* 2003; 38(1):41-45.
- [38] Parangi S., Oz M.C., Blume R.S., et al: Hepatobiliary complications of polyarteritis nodosa. *Arch Surg* 1991; 126(7):909-912.
- [39] Erskine J.M.: Hepatic artery aneurysm. *Vasc Surg* 1973; 7(2):106-125.
- [40] Bailey R.W., Riles T.S., Rosen R.J., Sullivan L.P.: Celiomesenteric anomaly and aneurysm: clinical and etiologic features. *J Vasc Surg* 1991; 14(2):229-234.
- [41] Stone W.M., Abbas M., Cherry K.J., et al: Superior mesenteric artery aneurysms: is presence an indication for intervention?. *J Vasc Surg* 2002; 36(2):234-237.discussion, 7
- [42] Geelkerken R.H., van Bockel J.H., de Roos W.K., Hermans J.: Surgical treatment of intestinal artery aneurysms. *Eur J Vasc Surg* 1990; 4(6):563-567.





## **New Advances in the Basic and Clinical Gastroenterology**

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The purpose of this book was to present the integrative, basic and clinical approaches based on recent developments in the field of gastroenterology. The most important advances in the pathophysiology and treatment of gastrointestinal disorders are discussed including; gastroesophageal reflux disease (GERD), peptic ulcer disease, irritable bowel disease (IBD), NSAIDs-induced gastroenteropathy and pancreatitis. Special focus was addressed to microbial aspects in the gut including recent achievements in the understanding of function of probiotic bacteria, their interaction with gastrointestinal epithelium and usefulness in the treatment of human disorders. We hope that this book will provide relevant new information useful to clinicians and basic scientists as well as to medical students, all looking for new advancements in the field of gastroenterology.

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