

Chronic Mesenteric Ischemia: Imaging and Percutaneous Treatment¹

CME FEATURE

See accompanying test at http://www.rsna.org/education/rg_cme.html

LEARNING OBJECTIVES FOR TEST 5

After reading this article and taking the test, the reader will be able to:

- Describe the pathophysiology of CMI on the basis of anatomic considerations.
- List the clinical and radiologic findings in CMI and recognize the differential diagnoses.
- Discuss the indications for, techniques of, and results of PTA with stent placement in CMI.

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Chronic mesenteric ischemia (CMI) is rare and is often diagnosed late. Fatal malabsorption-related complications or acute ischemic events occur in the absence of treatment. Diagnosis depends on careful acquisition of a medical history and elimination of other conditions. No sensitive and specific tests are available for functional diagnosis of CMI. If other causes of abdominal pain and weight loss have been confidently ruled out, evidence of visceral artery occlusion at noninvasive imaging (Doppler ultrasonography, computed tomographic angiography, and magnetic resonance angiography) suggests CMI. Until the 1990s, open surgery was considered the treatment of choice; percutaneous transluminal angioplasty (PTA) was reserved for patients for whom surgery carried a high risk. However, open surgery carries a nonnegligible risk of morbidity and mortality. In recent years, PTA with stent placement has been recognized as a minimally invasive means of obtaining good long-term results with an acceptable recurrence rate and consequently has been suggested for primary treatment of CMI. New treatments including administration of fibrinolytic agents before PTA of chronic occlusions, routine revascularization of one or more arteries, and stent placement will probably be validated in the near future. Similarly, new data on selection of the best approach will become available soon.

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Abbreviations: CA = celiac artery, CMI = chronic mesenteric ischemia, IMA = inferior mesenteric artery, PTA = percutaneous transluminal angioplasty, SMA = superior mesenteric artery

Index terms: Arteries, mesenteric, 95.72 • Arteries, stenosis or obstruction, 95.72 • Arteries, transluminal angioplasty, 95.128 • Mesentery, ischemia, 95.761

RadioGraphics 2002; 22:863–880

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See the commentary by Coldwell following this article.

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Table 1
Characteristics of 16 Patients with CMI

Patient/ Age (y)/ Sex	Other Vascular Disease*	Signs and Symptoms	Weight Loss (kg)	Time to Diagnosis (mo)	Site of Significant Stenosis or Occlusion†	Cause
1/85/F	LLOAD	Angina	14	1	SMA‡	Atheroma
2/41/M	LLOAD	Angina	2	24	CA, SMA, IMA	Atheroma
3/69/M	LLOAD	Ischemic gastropathy	0	6	CA, SMA	Atheroma
4/56/M	LLOAD, CAD, SAI	Angina	6	2	CA, SMA	Atheroma
5/34/M	LLOAD	Angina	30	56	CA, SMA	Buerger arteritis
6/92/F	None	Angina	8	24	CA, SMA	Atheroma
7/75/M	LLOAD, CAD	Diarrhea, constipation	5	11	SMA‡	Atheroma
8/73/M	SAI	Angina	7	8	CA, IMA	Atheroma
9/72/M	LLOAD, CAD, SAI	Angina	0	3	CA, SMA	Atheroma
10/62/M	None	Angina	15	9	SMA	Atheroma
11/90/F	None	Angina	5	3	SMA‡	Atheroma
12/54/F	LLOAD, SAI	Angina	0	3	CA, SMA	MAL§ syndrome, Takayasu arteritis
13/75/F	LLOAD	Ischemic gastropathy	0	9	CA, SMA	MAL syndrome, atheroma
14/75/F	None	Angina	0	3	SMA, IMA	Dysplasia, atheroma
15/45/M	None	Angina	20	18	CA, SMA	Atheroma
16/53/M	LLOAD	Angina	7	6	CA, SMA	Atheroma

*CAD = coronary artery disease, LLOAD = lower limb occlusive arterial disease, SAI = supraaortic involvement.

†CA = celiac artery, IMA = inferior mesenteric artery.

‡Lesion located distally in the SMA (at least 4 cm from the ostium).

§MAL = median arcuate ligament.

Introduction

Chronic mesenteric ischemia (CMI) was described in 1894 by Councilman (1). Goodman (2) in 1918 and Dunphy (3) in 1936 recognized that abdominal angina was a vascular disease. In 1958, Shaw and Maynard (4) performed a successful surgical revascularization of the superior mesenteric artery (SMA), demonstrating that full recovery was achievable.

Percutaneous transluminal angioplasty (PTA) of the SMA was reported in 1980 by Uflacker et al (5) and Furrer et al (6). Since then, this procedure has gained acceptance but has not yet been established as the treatment of choice.

The first part of this article reviews the pathophysiology and diagnosis of CMI. Then, the role of noninvasive imaging is extensively discussed. Finally, the techniques and results of PTA and stent placement are presented, with abundant reference to 16 consecutive cases managed at our institution during the past 10 years.

Diagnosis of CMI

Etiologic Setting

Atheroma is the main cause of CMI. In the overwhelming majority of cases, the proximal segments of the visceral arteries are involved. Fatty

infiltration of the arterial wall leads to stenosis or occlusion of one or more visceral arteries.

However, the proportion of arterial occlusions caused by nonatheromatous lesions is probably higher in the visceral arteries than at other sites:

Takayasu arteritis, dysplastic lesions, thromboangiitis obliterans, and radiation-induced lesions can be encountered (Table 1). These nonatheromatous causes, particularly Takayasu arteritis, have been reported in many studies (7).

Many patients with CMI have atheroma at other sites: Lower limb occlusive arterial disease and lesions of the coronary and carotid arteries are prevalent in most cases (Table 1) (8). This high prevalence among CMI patients of atheroma at other sites may contribute to the poor prognosis of CMI in patients whose risk factors are not controlled.

Obstructive Lesions of the Visceral Arteries and CMI

The number of arteries that must be involved before symptoms of chronic ischemia occur remains a matter of debate. It has been suggested that involvement of at least two vessels should be required for a diagnosis of CMI. Autopsy or ultrasonographic (US) studies found that multiple severe visceral artery lesions were common in

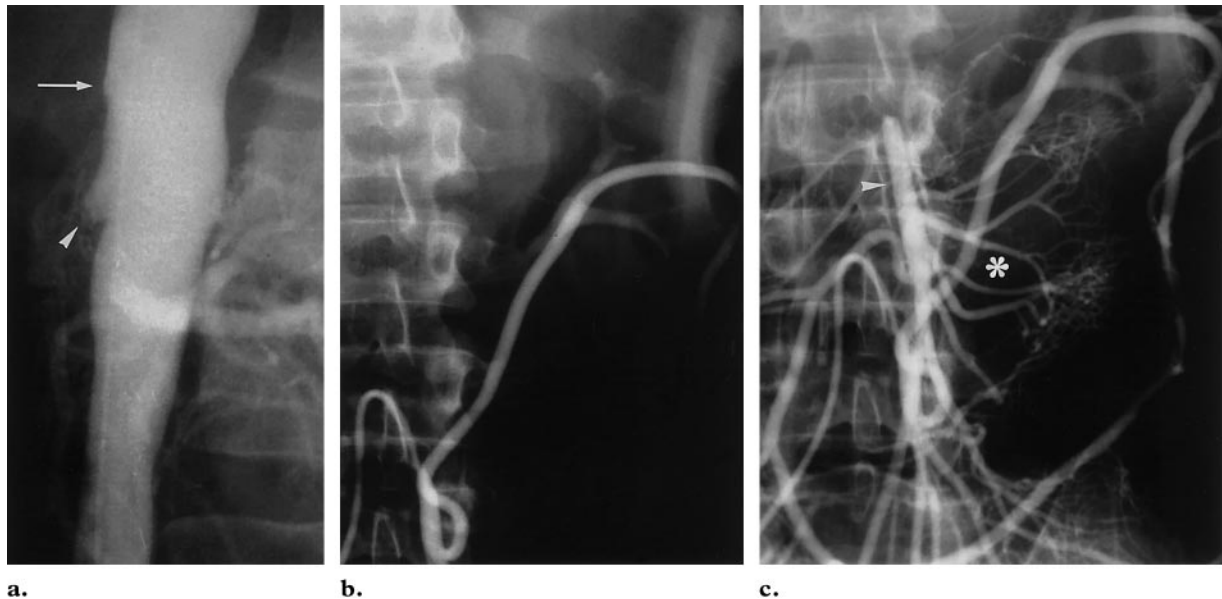


Figure 2. Collateral vessels in a 79-year-old patient with thrombosis of the two main visceral arteries but no abdominal symptoms. **(a)** Global lateral angiogram shows thrombosis of the SMA (arrowhead) and CA (arrow). **(b)** Image from selective angiography of the IMA shows a prominent marginal artery of Drummond connected to the paracolic arcade. **(c)** Angiogram shows that the paracolic arcade is connected to the SMA (arrowhead) downstream from the occlusion and feeds the jejunal arteries (*). Note that the paracolic arcade is located low in the abdomen due to ptosis of the transverse colon, which is common in elderly patients.

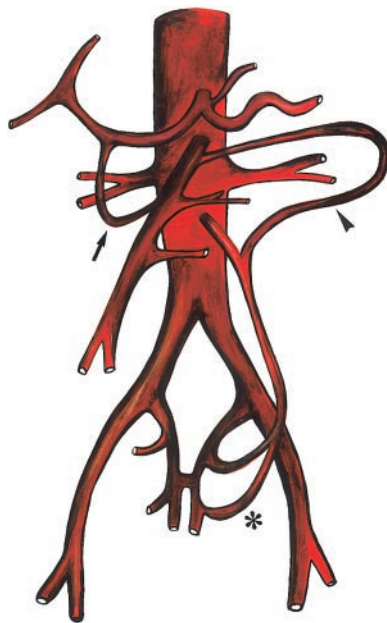


Figure 1. Drawing shows the main collateral vessels in occlusion of the CA or SMA. The pancreaticoduodenal arteries (arrow) connect the CA and the proximal SMA. The marginal artery of Drummond and the paracolic arcade (arrowhead) run between the SMA and IMA. When the IMA is also occluded, the systemic vessels (mainly the internal iliac artery) can feed the IMA (reverse flow) and the other vessels via previously described anastomoses (*).

patients without symptoms of chronic ischemia (9–11).

However, whether ischemia occurs depends mainly on the amount of blood that flows into the diseased artery from other arteries (the CA and IMA). Thus, the bowel vasculature should be

viewed as a single functional unit. The collateral vessels that develop in patients with visceral artery stenosis have been described accurately. They can be divided into two major systems (Fig 1). One system connects the CA to the SMA and is composed mainly of the pancreaticoduodenal arteries running between the gastroduodenal artery and the proximal SMA. As with most visceral anastomoses, the blood can flow in either direction according to the site of the occlusion. The other system connects the SMA to the IMA and comprises the paracolic arcade (or Riolo arch) and the marginal artery of Drummond. When all three visceral arteries (CA, SMA, and IMA) are stenotic or occluded, phrenic, lumbar, and pelvic collateral vessels become prominent. Typical collateral networks in patients with single or multiple lesions are shown in Figures 2 and 3, respectively.



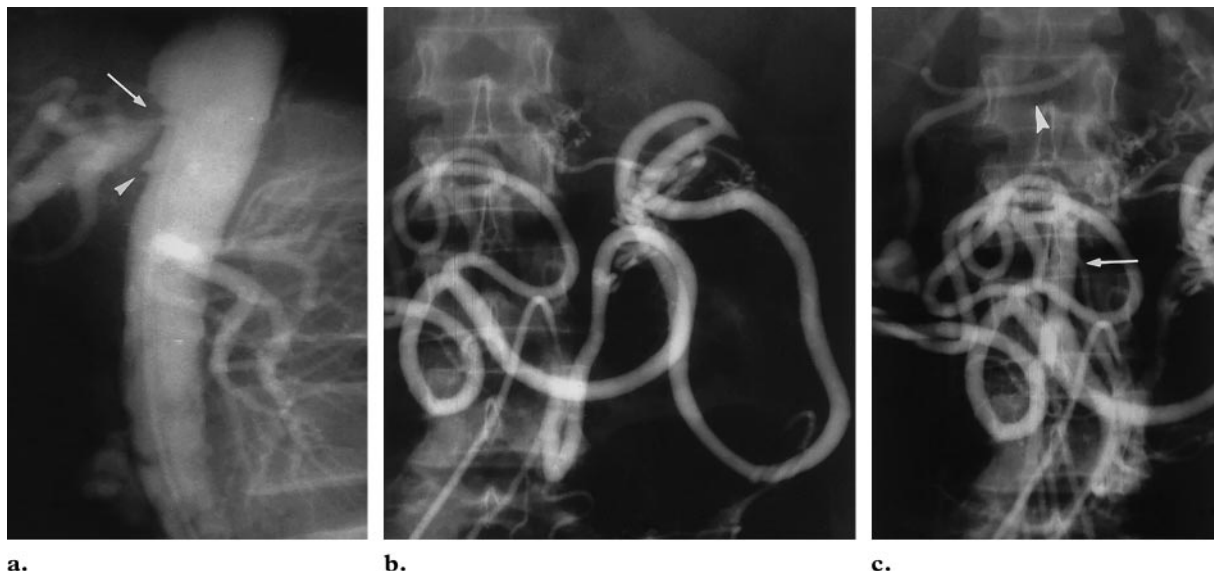


Figure 3. Collateral vessels in a 66-year-old patient with abdominal angina. **(a)** Lateral angiogram shows thrombosis of the SMA (arrowhead) and severe stenosis of the CA (arrow). **(b)** Angiogram obtained with selective injection of contrast material into the IMA shows unusual serpentine collateral vessels emerging from the marginal artery of Drummond, which is not connected to the paracolic arcade. **(c)** Late-phase angiogram shows the SMA (arrow) and CA (arrowhead).

Some patients with a single lesion have pain, whereas others with up to three lesions are asymptomatic. This suggests that the occurrence of painful ischemia does not depend only on the number of proximal arterial lesions. One of the other factors involved may be the site of the lesion or lesions. For instance, when an SMA lesion is downstream from the anastomosis with the gastroduodenal artery (three cases in our study), ischemia may occur more rapidly than with a more proximal lesion. Nevertheless, distal stenosis is uncommon because atheroma rarely develops in the distal parts of the mesenteric vessels (9). Diffuse atherosclerosis of the distal visceral vessels is occasionally present, particularly in patients with diabetes or end-stage renal disease. These patients may be unable to develop collateral vessels and consequently may experience ischemia even with a single mild stenosis.

Another factor in the occurrence of ischemia may be the tempo of progression of the lesions. In inflammatory diseases, such as Takayasu disease, severe occlusion may occur before an efficient collateral network has time to develop.

Thus, the occurrence of ischemia may be a multifactorial event dependent on the pace of lesion progression, the ability of the individual patient to develop collateral vessels, and the site of the lesion.

Clinical Presentation

Abdominal angina, which was first described at the beginning of the 20th century, is defined as

postprandial abdominal pain with weight loss and anorexia. Changes in bowel habits and vomiting are less common.

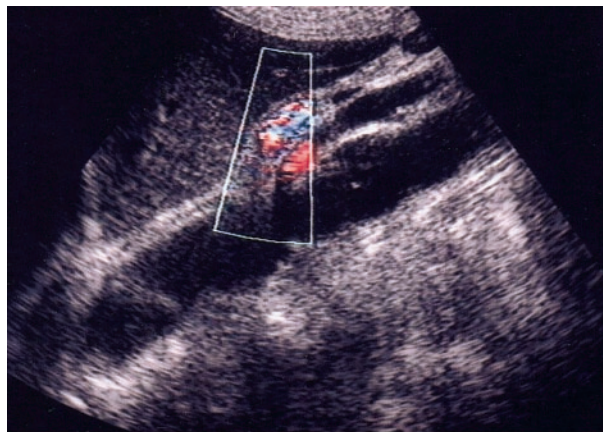
In two of our patients, ischemic gastropathy was the final diagnosis. One of these patients had incapacitating gastroparesis with vomiting, postprandial heaviness, and delayed gastric emptying at barium study. The other had severe, endoscopically documented gastroduodenitis unresponsive to pharmacotherapy. In both patients, prompt resolution of the symptoms after PTA supported the diagnosis of ischemic gastropathy. A few reports of reversible gastric symptoms after revascularization have been published (13,14). We agree with Casey et al (13) that the visceral arteries should be investigated, at least with Doppler US, in patients with chronic gastric symptoms.

Cachexia suggesting a malignancy can occur if there is severe malabsorption or if the patient eats less to avoid triggering the pain.

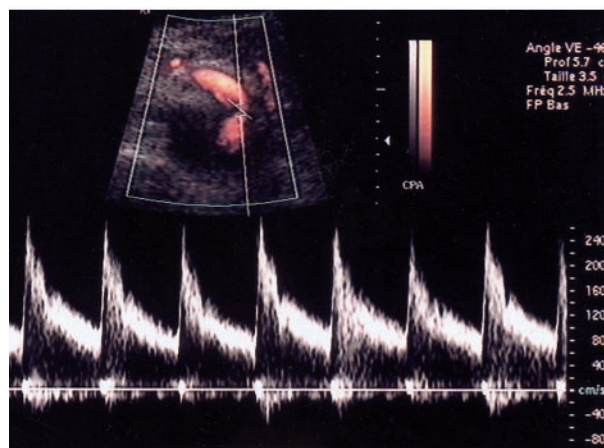
Because abdominal pain and weight loss are common symptoms and CMI is an uncommon condition, the diagnosis of CMI is often made late (Table 1).

The symptoms usually develop insidiously. This contributes to the diagnostic delay. Thus, in all age groups, many patients are seen late, at a stage when they have severe weight loss.

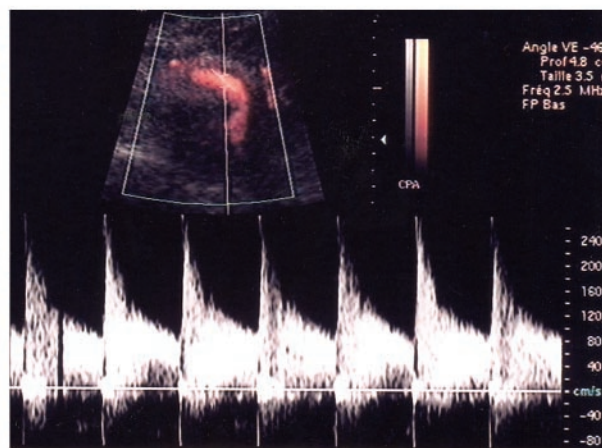
The prevalence of CMI seems to increase with age, in agreement with the age-related increase in the prevalence of visceral artery occlusion in autopsy studies (13,14). However, CMI can occur in younger patients and the weight loss can suggest a malignant disease.



a.



b.



c.

Figure 4. Duplex US findings in isolated stenosis of the CA. (a) Lateral US image obtained in color mode shows color aliasing. (b) Lateral US image obtained in Doppler mode shows signs of moderate stenosis with increases in systolic and diastolic velocities, as well as mild turbulence. (c) Lateral US image obtained in Doppler mode shows major poststenotic turbulence and Doppler aliasing, which indicate a stenosis of greater than 75%.

Imaging in CMI

Abdominal Imaging.—Because occlusive visceral artery lesions are not specific for the diagnosis of CMI, imaging studies of the abdomen should be performed to rule out other causes. Contrast material-enhanced computed tomography (CT) of the entire abdominal cavity has proved capable of providing accurate information on inflammatory, neoplastic, and vascular diseases of the abdomen. Moreover, it shows the small bowel walls, whose normal appearance in CMI rules out an acute event (15). In patients with good renal function, multi-detector row technology can be used to obtain CT angiograms of the mesenteric vessels during the same investigation.

In patients with pain alleviated by meals (an unusual feature in CMI), endoscopy is in order to look for an ulcer of the stomach or duodenum.

If liver function tests show abnormalities (particularly cholestasis), a US study and magnetic resonance (MR) cholangiography should be considered to look for bile duct obstruction caused by a pancreatic carcinoma or other lesion.

Imaging of the Visceral Arteries.—When the main nonvascular causes of abdominal pain have been ruled out, the bowel vasculature should be studied. Several techniques are capable of showing stenosis or occlusion of the proximal visceral arteries.

Duplex US has yielded encouraging results over the past decade. Numerous studies, many of which used arteriography as the standard of reference, found that duplex US was an accurate screening test for proximal arterial stenosis or occlusion. A peak systolic velocity greater than 275 cm/sec seems highly specific for significant SMA stenosis (16), although an end-diastolic velocity greater than 45 cm/sec may be more accurate (17,18) (Fig 4).

Most studies of differences in velocities and resistive index between the fasting state and the postprandial state were conducted in patients with cirrhosis of the liver or inflammatory diseases (19). However, blunting of fasting-postprandial differences in peak systolic velocity has been reported in patients with visceral artery stenosis

Figure 5. Acute mesenteric ischemia in a 43-year-old man with acute abdominal pain but no peritoneal signs. (a, b) CT scans obtained in the emergency department. (a) CT scan shows a thrombus in the SMA (arrow) and nonspecific jejunal wall thickening (arrowhead), which produces the target sign. (b) CT scan obtained 2 cm lower shows enhancement of the distal SMA (arrow), which is fed by collateral vessels, and thinning of the mesenteric wall of a jejunal loop (arrowhead), which is a more specific sign of acute ischemia. (c) Image from selective angiography of the SMA shows a thrombus in the trunk of the artery (arrow). (d) Image from selective angiography of the IMA shows that the distal SMA (arrow) is fed by a prominent marginal artery of Drummond (arrowhead). Acute thrombosis complicating preexistent distal stenosis of the SMA was suspected, and urokinase was given as an intraarterial injection of 600,000 U in 6 hours. (e) Control angiogram shows an irregular persistent stenotic lesion (arrow). (f) Angiogram shows a balloon-expandable stent, which was placed to prevent reocclusion and distal migration. Surface irregularities (arrowheads) are visible proximal and distal to the stent and may represent sites of restenosis. The patient returned home on day 5 and was asymptomatic 8 months later.

(20). This finding suggests failure of the mesenteric blood flow to adapt to meal-related needs. However, this blunting was not strictly correlated with pain severity.

MR angiography and, more recently, CT angiography have both been reported to provide accurate data on bowel vascularization. As with renovascular disease, evaluation of the proximal part of the visceral arteries is generally sufficient for making the diagnosis and selecting the best treatment.

Over the past decade, contrast-enhanced MR angiography has benefited from technological advances. In particular, improved gradient technology allows ultrafast volume acquisitions with computer-assisted multiplane reformatting or volume rendering (21,22). This technique has shown excellent agreement with conventional angiography for evaluation of the visceral vessels (23) and detection of significant arterial stenosis (24). An important advantage (particularly compared to CT angiography) is that it is entirely safe even when there is severe renal disease, which is not unusual in patients with atheroma.

Although spiral CT has proved useful for assessing the visceral arteries (25), multi-detector row CT, which was introduced a few years ago, provides even better information on the abdominal aorta and its branches. New computer-assisted reconstruction techniques allow accurate measurement of arterial stenoses (26,27).

Because correlations are poor between CMI and the presence at vascular imaging of one or more arterial stenoses, we agree with McShane et al (28) that diagnosis of CMI requires confident elimination of other conditions. Although the above-mentioned noninvasive diagnostic investi-

gations are very promising, we believe they should be viewed as screening tests that are useful for avoiding unnecessary angiograms. Because no functional test capable of detecting visceral ischemia is available, the diagnosis of CMI continues to rest on a careful medical history.

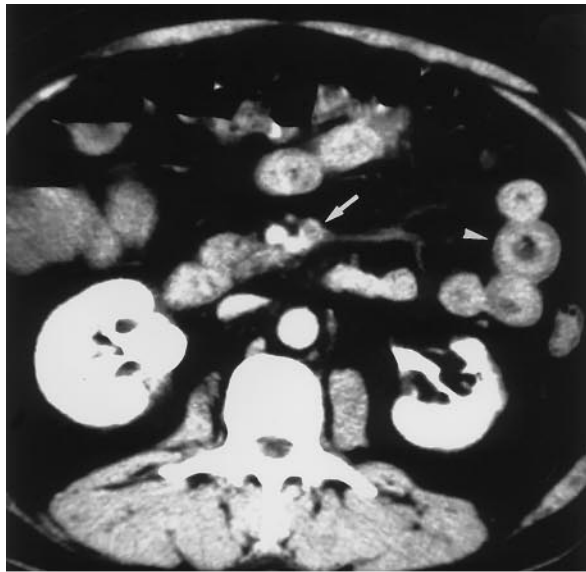
Differential Diagnoses

Acute Mesenteric Ischemia.—A crucial point is differentiation of stable chronic symptoms from symptoms that have worsened recently. If the pain has recently become permanent or if peritoneal symptoms have developed, acute ischemia should be suspected and an emergency laparotomy should be performed. In patients who are seen very early, angiography prior to surgery may deserve discussion as a means of establishing the diagnosis and allowing local fibrinolytic treatment of an acute thrombus or embolus (29). An intraarterial infusion of papaverine should be given to patients with nonocclusive acute mesenteric ischemia (30).

Figure 5 shows a case of acute ischemia that was successfully managed with PTA at our institution. This method was selected because of the absence of peritoneal signs at evaluation in the emergency department.

Retroperitoneal or Celiomesenteric Malignancy.—A high index of suspicion for pancreatic carcinoma should be maintained. Major weight loss and insidious onset of pain caused by celiac plexus compression are typical. Endoscopic US, CT, and MR imaging can help one make the diagnosis (Fig 6).

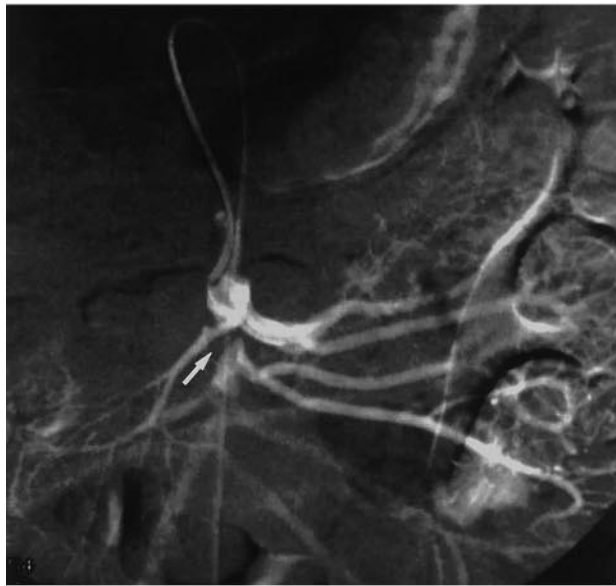
Retroperitoneal lymphomas can also cause epigastric pain by putting pressure on the celiac plexus. In most cases, CT readily allows one to make the diagnosis.



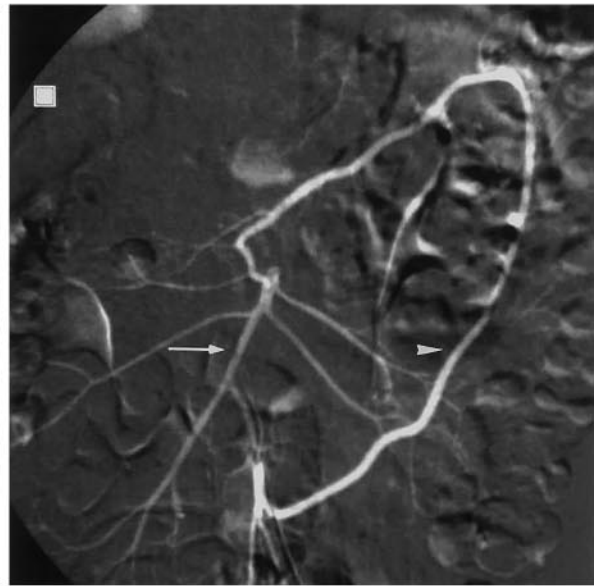
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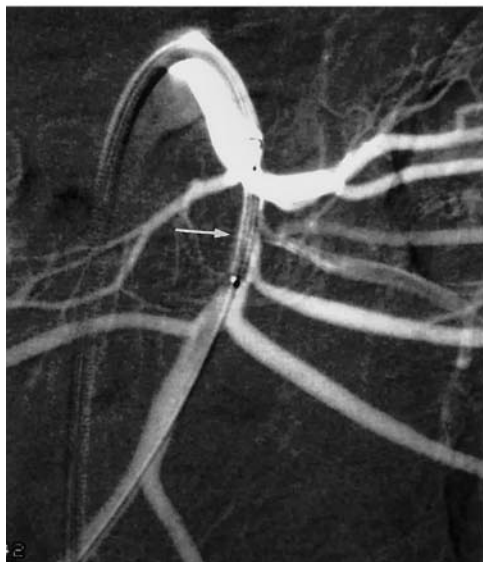
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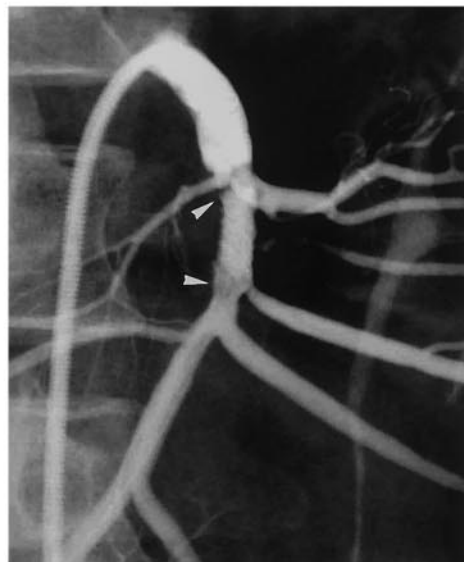
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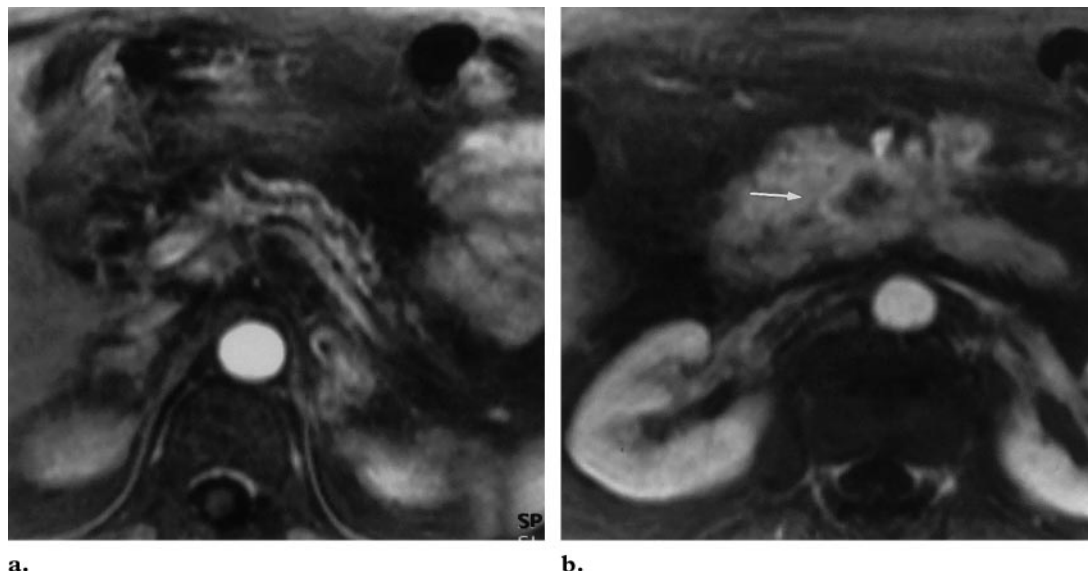


Figure 6. Infiltrating adenocarcinoma of the pancreatic head. **(a)** Axial gadolinium-enhanced T1-weighted gradient-echo MR image of the pancreas shows a thin biliary tract. **(b)** Axial gadolinium-enhanced T1-weighted gradient-echo MR image of the pancreas clearly shows the tumor with peripheral enhancement (arrow).

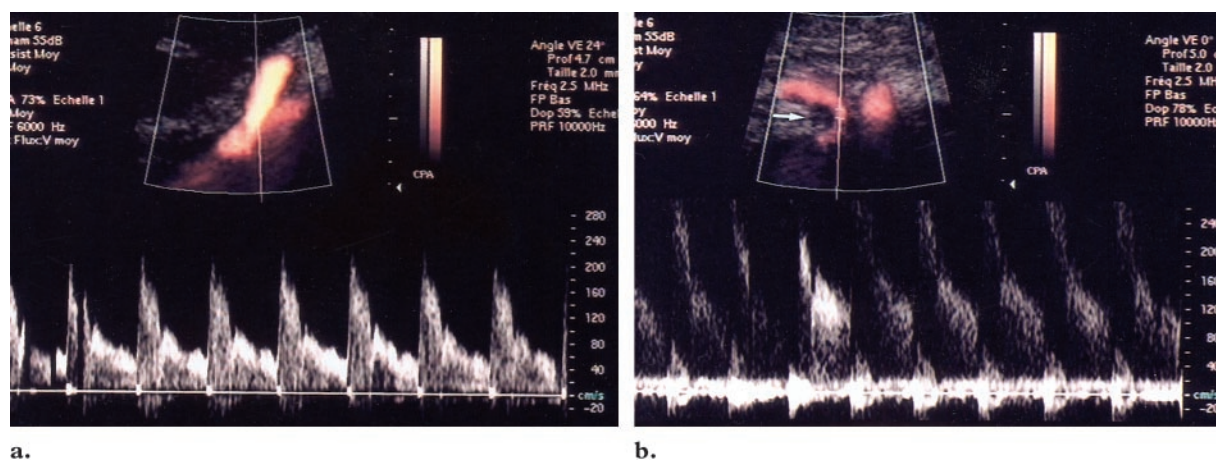


Figure 7. Median arcuate ligament syndrome. **(a)** Lateral US image obtained with energy and pulsed mode imaging during inspiration shows that the CA and SMA course downward. The velocities in the CA are slightly increased. **(b)** Lateral US image obtained with energy and pulsed mode imaging during expiration shows that the proximal part of the CA is lowered and impinged on by the median arcuate ligament (arrow). The velocities are markedly increased, and aliasing associated with turbulence is seen.

Median Arcuate Ligament Syndrome.—

The median arcuate ligament syndrome is still controversial. It has been described as a combination of epigastric pain, sometimes alleviated by inspiration, and abdominal bruits occurring almost always in young women.

Although some authors have argued that the pain is caused by gastroduodenal tract ischemia (31), others believe that it arises from celiac plexus compression and consequently is not an indication for PTA or stent placement (32–34). We share this point of view.

Angiography can show compression of the CA. On lateral projections, there is a concave imprint on the cranial surface of the proximal CA. The stenosis generally increases during deep expiration and can disappear completely during inspiration.

Duplex US, which is used more routinely, shows the breathing-related changes in the degree of stenosis, with increased turbulence or flow velocity during expiration (31) (Fig 7).

CT has been found useful in diagnosing this syndrome (32): The median arcuate ligament can be identified easily as a hypoattenuating structure crossing the aorta above the departure of the CA.

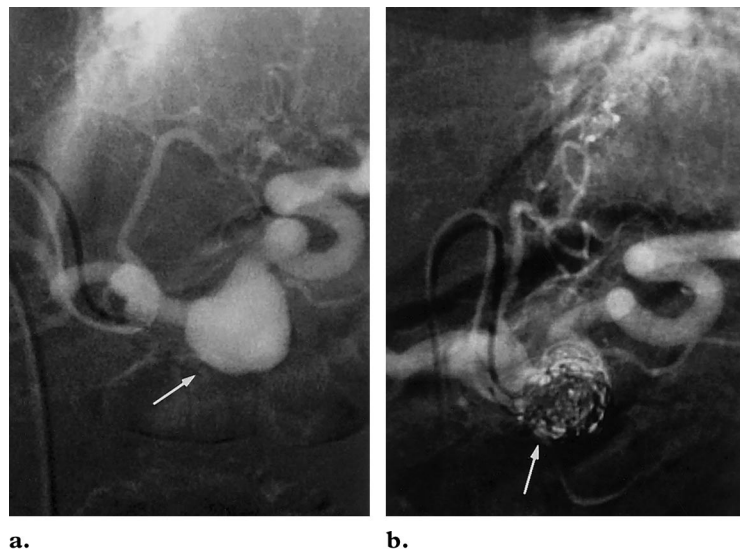


Figure 8. Aneurysm of the splenic artery in a 68-year-old patient with end-stage renal disease who had unexplained abdominal pain. **(a)** Selective angiogram shows a sacciform aneurysm (arrow). **(b)** Angiogram shows that embolization was performed with coils (arrow), with a good final result.

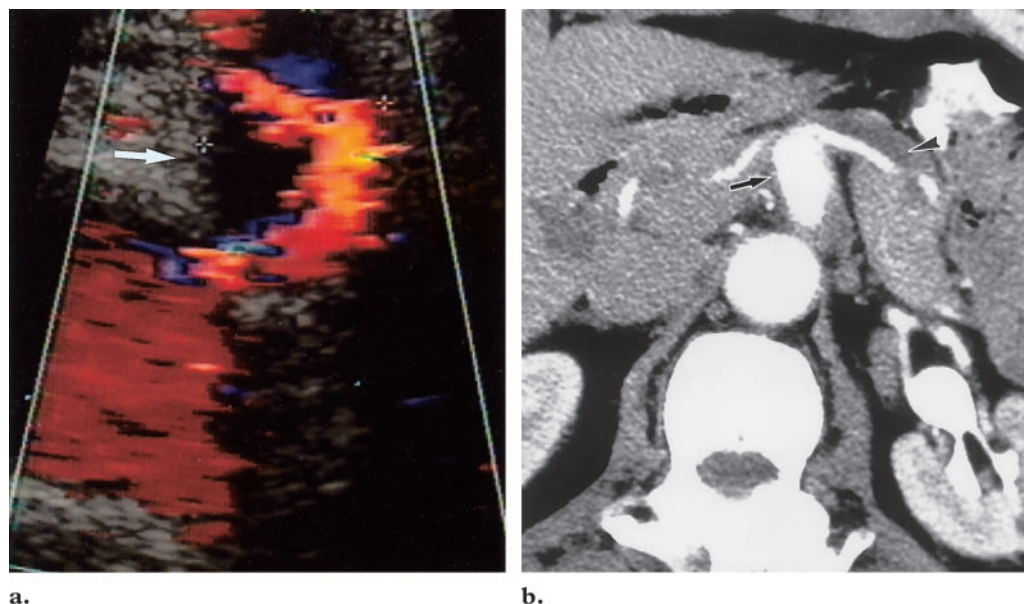


Figure 9. Aneurysmal dissection of the CA in a 59-year-old patient who had severe epigastric pain for several years. **(a)** Color Doppler US image shows an aneurysm of the celiac trunk (arrow). **(b)** Contrast-enhanced CT scan also shows the aneurysm (arrow). The very thin aspect of the splenic artery and the hypoattenuating crescent anterior to the splenic artery (arrowhead) are suggestive of an associated splenic artery dissection.

Treatment of this condition nowadays takes the form of laparoscopic division of the median arcuate ligament (35).

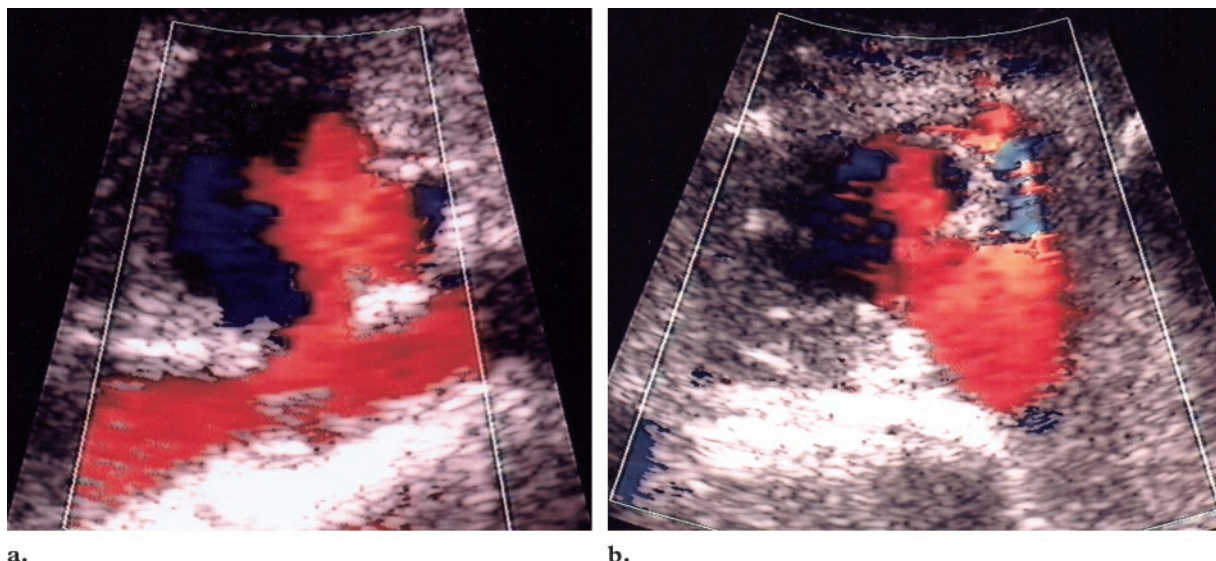
Gastroduodenal Ulcer.—The diagnosis of gastroduodenal ulcer is suspected when the pain is alleviated by meals, and this diagnosis is confirmed with endoscopy. Ischemic gastropathy is extremely uncommon. This diagnosis should be suspected only when the lesions are progressive or refractory to treatment.

Nonocclusive Vascular Lesions: Aneurysms and Dissections.—Aneurysms or false aneurysms of the visceral arteries are uncommon (36).

Embolization is gaining ground as the treatment of choice for these abnormalities (Fig 8) (37,38).

Rarely, the aneurysm is a complication of isolated dissection of CA branches (Fig 9). A few other cases of painful isolated dissection of the visceral arteries have been reported (39–41).

False aneurysms of the visceral arteries are easier to differentiate from CMI because a history of blunt abdominal trauma or chronic pancreatitis is usually present (37).



a.

b.

Figure 10. Mycotic sacciform aneurysm of the abdominal aorta in a 53-year-old man with unexplained abdominal pain. (a) Lateral color Doppler US image shows a sacciform aneurysm of the anterior wall of the abdominal aorta, under the renal arteries. (b) Anteroposterior color Doppler US image also shows the aneurysm. Treatment consisted of open surgery after resolution of the inflammation.

Less uncommon vascular causes of abdominal pain are aneurysms or dissection of the abdominal aorta (Fig 10). However, the symptoms are generally less progressive than in CMI.

Percutaneous Treatment

Angiographic Findings

When the diagnosis of CMI has been confidently established, angiography of the visceral arteries should be performed prior to PTA. Global lateral views are needed to confirm the findings of non-invasive imaging studies (Fig 2a). Then, routine selective catheterization of the visceral arteries provides information on the severity of the stenoses and on the blood supply. Collateral vessels are usually best visualized on anteroposterior projections (Fig 3c). Catheterization of the CA, if it is patent, can be used to measure the length of an occlusion located in the SMA. This information is important for planning the treatment. On the other hand, selective catheterization of the IMA should be replaced by nonselective angiography performed with the catheter placed at the level of the IMA ostium. Indeed, accidental catheter trauma and occlusion would be very poorly tolerated, since the IMA represents the main feeding collateral vessel for the whole intestine.

Techniques of PTA

Choice of Route and Catheters.—Since the first reports of PTA of the SMA or CA in 1980, less aggressive, more slender catheters have been developed and advances in the design of the balloon and stent catheter have made the procedure easier to perform. This explains why the axillary route has been discarded by most teams. However, the humeral route is still used, particularly for PTA and stent placement in the CA; there are three curves in opposite directions when the CA is approached by the femoral route but only two when it is approached by the humeral route. In our experience, the best catheter geometries for selective catheterization of the visceral arteries via the femoral approach are the sidewinder configuration, the hook configuration, and, more rarely, the cobra configuration. With the humeral route, the vertebral configuration seems effective.

Control angiograms can be obtained by means of a long-armed sheath positioned in front of the mesenteric vessels (Fig 11). The coaxial technique, which uses materials developed for coronary artery angiography, is an alternative that provides optimal procedure comfort.

As in other endovascular procedures, heparin as a 5,000-U bolus should be injected as soon as selective catheterization is started. This has been shown to reduce the complication rate.

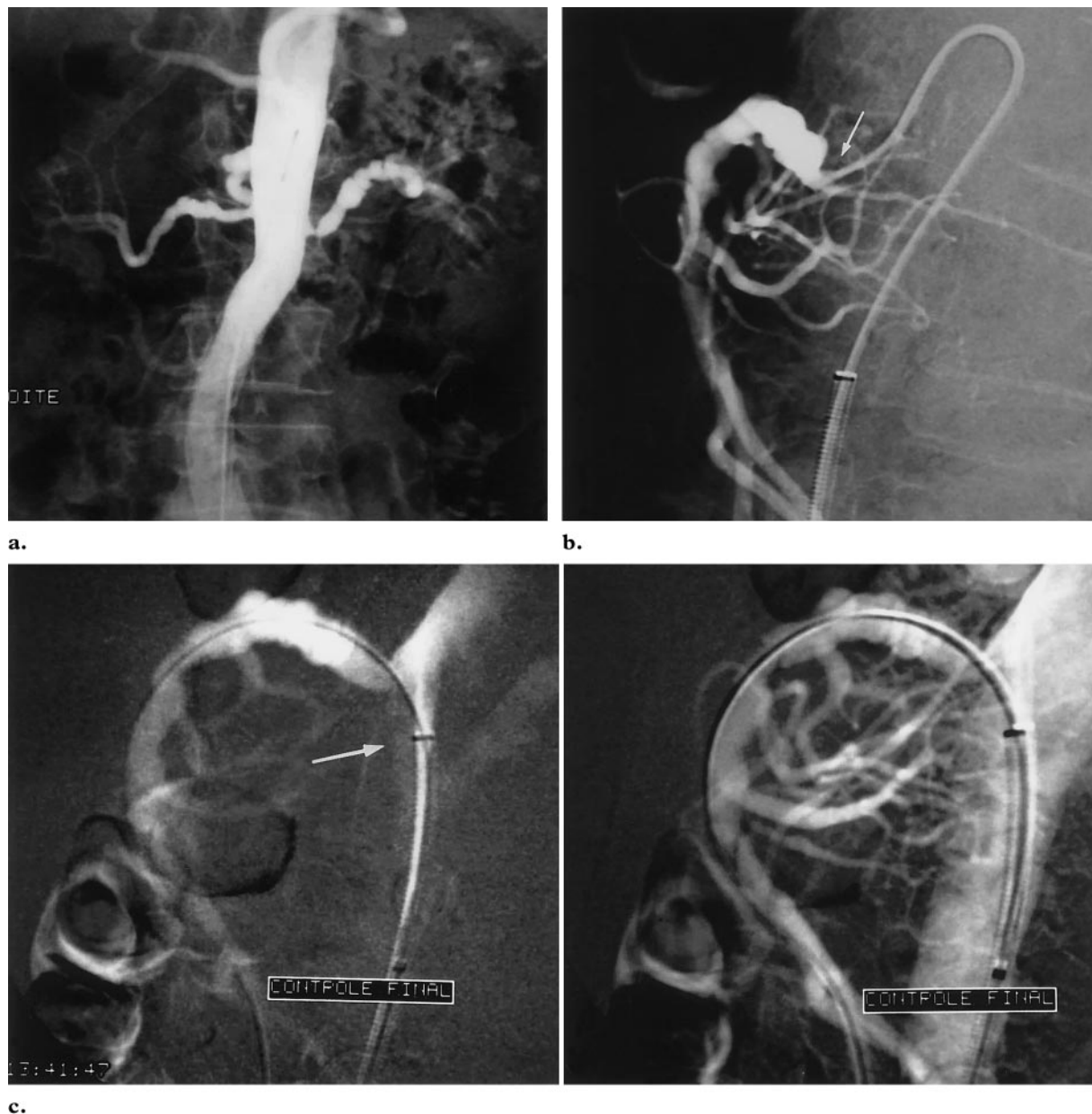


Figure 11. CMI in a 75-year-old woman with arterial dysplasia of the renal and mesenteric arteries. **(a)** Global angiogram. **(b)** Angiogram obtained with selective catheterization of the SMA by using a 5-F sidewinder catheter shows an ostial stenosis (arrow). **(c)** Postangioplasty completion arteriograms obtained by using a long and armed 6-F sheath, which is pushed to the level of the mesenteric ostium (arrow).

Single-Vessel or Multiple-Vessel Revascularization?—According to the literature, revascularization is generally performed in only one obstructive lesion. Our opinion is that revascularization of the SMA should be attempted first. Revascularization of the CA could be reserved in case of failure of the SMA revascularization attempt. This is consistent with the experience of other teams, in which revascularization was far more difficult at the CA than at the SMA because of the more sinuous course of the CA in most patients. Because a humeral route conversion is generally required for CA revascularization, certain authors advocate this route as the first step.

In all 16 of our patients, at least one PTA procedure was successful. However, PTA of both the CA and SMA was successful in only one of seven patients. In the three cases of failed SMA revascularization, the CA could be treated successfully (Table 2).

There have been few reports of multiple-vessel revascularization to achieve symptom relief (Table 3). Single-vessel revascularization of the CA or SMA usually ensures a favorable long-term outcome.

Table 2
Procedures Performed, Results, and Outcomes in 16 Patients with CMI

Patient	Site of Significant Stenosis or Occlusion	Procedures Performed by Artery*		Complications	Immediate Outcome	Recurrence of Pain	Long-term Outcome
		SMA	CA				
1	SMA	PTA	...	None	Improvement, 4-kg weight gain	None	11-kg weight gain at 57 mo
2	CA, SMA, IMA	Failure [†]	PTA	Surgically repaired femoral hematoma	Clinical remission, 3-kg weight gain	None	No symptoms at 48 mo
3	CA, SMA	PTA	Failure	None	Clinical remission	None	No symptoms at 46 mo
4	CA, SMA	Failure	PTA	None	Clinical remission	None	Death (lung infection) at 12 mo
5	CA, SMA	PTA	NA	None	Clinical remission	None	No symptoms at 10 mo
6	CA, SMA	Failure	PTA	Surgically repaired humeral hematoma	Clinical remission	At 4 mo [§]	No symptoms at 7 mo, 5-kg weight gain
7	SMA	PTA	...	None	Clinical remission, 3-kg weight gain	None	No symptoms at 54 mo
8	CA, IMA	...	PTA, stent	None	Clinical remission, 4-kg weight gain	None	No symptoms at 14 mo
9	CA, SMA	PTA	NA	None	Clinical remission	None	No symptoms at 33 mo
10	SMA	PTA, stent [†]	...	None	Clinical remission, 5-kg weight gain	None	No symptoms at 3 mo, lost to follow-up
11	SMA	PTA, stent	...	None	Clinical remission	None	No symptoms at 3 mo, lost to follow-up
12	CA, SMA	PTA, stent	NA [‡]	None	Clinical remission	None	No symptoms at 26 mo
13	CA, SMA	PTA	Failure	None	Clinical remission	At 12 mo	No symptoms at 18 mo
14	SMA, IMA	PTA	...	None	Clinical remission	None	No symptoms at 15 mo
15	CA, SMA	PTA, stent [†]	Failure [†]	Painful dissection of CA	Clinical remission, 12-kg weight gain	None	No symptoms at 14 mo
16	CA, SMA	PTA, stent	PTA, stent	None	Clinical remission	None	No symptoms at 8 mo

*Failure = failed revascularization attempt, NA = not applicable, stent = stent placement.

[†]Case of occlusion.[‡]Patient had median arcuate ligament syndrome.[§]Treated with PTA of the SMA and CA.^{||}Treated with PTA of the CA and stent placement in the SMA.

However, in young patients with progressive atheromatous or inflammatory disease, two-vessel revascularization may reduce the risk of symptom recurrence (42). A reasonable strategy may be to revascularize the SMA first, then to attempt CA revascularization via the humeral route when the patient has gained weight and is feeling better.

Occlusion.—Although a few recent reports indicate that recanalization of the SMA is feasible, the techniques for this procedure have not been precisely defined.

Our experience suggests that a notch on the lateral angiogram predicts successful recanalization because it is usually associated with a short, nonostial occlusion (Fig 12). Routine administration of fibrinolytic agents prior to passing the

Table 3
Patient Data and Procedure Success Rates from Studies of PTA

Study and Year	Patients with Signs of CMI	Male-Female Ratio	Percentage of Patients with Solitary Lesions	Procedure Success Rate per Patient (%)	Procedure Success Rate per Artery (%)	Percentage of Successful Multiple-Vessel Procedures
Golden et al (53), 1982	7	4/3	0 (0/7)	86 (6/7)	86 (6/7)	0 (0/7)
Odurny et al (47), 1988	10	5/5	0 (0/10)	100 (10/10)	89 (17/19)	50 (5/10)
McShane et al (28), 1992	6	3/3	17 (1/6)	100 (6/6)	100 (10/10)	50 (3/6)
Matsumoto et al (33), 1995	11/19	10/9	5 (1/19)	79 (15/19)	80 (16/20)	0 (0/19)
Rose et al (48), 1995	7/8	2/6	0 (0/7)	38 (3/8)	33 (3/9)	12 (1/8)
Hallisey et al (52), 1995	14/16	3/13	25 (4/16)	88 (14/16)	84 (21/25)	25 (4/16)
Allen et al (34), 1996	19	3/16	...	95 (18/19)	96 (23/24)	16 (3/19)
Maspes et al (12), 1998	23	5/18	35 (8/23)	96 (22/23)	90 (37/41)	61 (14/23)
Nyman et al (32), 1998	4/5	3/2	0 (0/4)	100 (5/5)	83 (5/6)	0 (0/5)
Current study, 2001	16	10/6	25 (4/16)	100 (16/16)	74 (17/23)	6 (1/16)
All studies	117	48/81	17 (18/108)	89 (115/129)	84 (155/184)	24 (31/129)

Note.—Values in parentheses are raw data.

**Figure 12.** CMI in a 45-year-old man with occlusion of both the CA and the SMA. (a) Lateral angiogram clearly shows a notch at the SMA (arrow). (b) Angiogram shows the occlusion being crossed by using a hydrophilic wire (arrow). (c) Control angiogram obtained after angioplasty shows good circulation in the SMA (arrowhead), but persistent recoil necessitated use of a stent (arrow). (d) Final angiogram shows a good result after stent placement.

Table 4
Mortality, Morbidity, and Clinical Success Rates from Studies of PTA

Study	PTA-related Mortality Rate (%)	Major Morbidity Rate (%)	Initial Clinical Success Rate (%)	Rate of Recurrence of Ischemic Symptoms (%)	Long-term Clinical Success Rate (%)*	Length of Follow-up (mo) [†]
Golden et al (53)	0 (0/7)	0 (0/7)	100 (6/6)	0 (0/6)	100 (6/6)	25
Odurny et al (47)	10 (1/10) [‡]	0 (0/10)	78 (7/9)	71 (5/7)	71 (5/7)	24
McShane et al (28)	0 (0/6)	0 (0/6)	83 (5/6)	40 (2/5)	100 (5/5)	21
Matsumoto et al (33)	0 (0/19)	16 (3/19)	80 (12/15)	17 (2/12)	92 (11/12)	25
Rose et al (48)	0 (0/8)	25 (2/8)	100 (7/7)	14 (1/7)	100 (7/7)	2–13
Hallisey et al (52)	0 (0/16)	0 (0/16)	100 (14/14)	25 (3/12)	83 (10/12)	4–48
Allen et al (34)	5 (1/19) [§]	5 (1/19)	79 (15/19)	20 (3/15)	93 (14/15)	39
Maspes et al (12)	0 (0/23)	0 (0/23)	77 (17/22)	24 (4/17)	82 (18/22)	27
Nyman et al (32)	0 (0/5)	40 (2/5)	100 (4/4)	25 (1/4)	100 (4/4)	8–36
Current study	0 (0/16)	12 (2/16)	100 (16/16)	12 (2/16)	100 (14/14)	26
All studies	1.6 (2/129)	7.8 (10/129)	87 (103/118)	23 (23/101)	90 (94/104)	26.3

Note.—Values in parentheses are raw data.

*Primary assisted.

[†]Values are mean or range.

[‡]Death caused by acute limb ischemia and septicemia.

[§]Death caused by occlusive dissection and bowel infarction.

catheter across the lesion has been advocated (43,44). In our population, fibrinolysis was not used in the two cases of SMA occlusion in which PTA was successfully performed, and no periprocedural signs of distal migration occurred. In our opinion, fibrinolysis is not appropriate in patients with stable symptoms of CMI because collateral vessels from the CA or IMA supply blood to the SMA close to its origin, so that the occlusion is usually very short, even when this is not obvious on the angiogram. In one of our two cases, contrast-enhanced CT clearly showed that the SMA occlusion was less than 2 cm long. Moreover, in chronic occlusions there is no recent blood clot and consequently the efficacy of fibrinolysis is debatable. However, because migration is a serious event, we recommend fibrinolysis in patients with recent onset or worsening of symptoms.

Stent Placement.—Recent reports have shown that stent placement in visceral arteries is feasible (45,46). Primary stent placement is now the standard of reference for renal ostial lesions, which represent 70% of renal artery stenoses. Because celiac stenoses and most mesenteric stenoses frequently have an ostial component, we believe that primary stent placement should be the rule, as for renal stenoses. Besides, we share the opinion of Sheeran et al (45) that routine stent placement is in order to achieve recanalization or treat procedure-related dissection. Because new-generation stents are more flexible, easier to implant, and

smaller in diameter, they will probably be increasingly used to prevent restenosis of lesions with moderate recoil.

Selection of the stent depends on the site of the lesions and the experience of the operator. At our institution, the balloon-expandable stent was preferred for ostial lesions because of its greater radial force, and autoexpanding stents were reserved for arterial trunk lesions because of their greater flexibility. However, the new balloon-expandable stents are more flexible, and the introduction of coaxial catheterization by using a 0.14- or 0.18-inch guide wire has substantially reduced the external diameter of the sheath at the puncture site.

Routine heparin therapy for 2 days after the procedure followed by aspirin or ticlopidine therapy has been recommended. At our institution, 100 mg of aspirin is given on the day of the procedure and continued indefinitely, but heparin is not used after the procedure.

Complications.—Puncture site hematomas still represent the most frequent serious complication of PTA, and proper compression of the site after the procedure is essential to prevent development of a false aneurysm. There is general agreement that puncture site complications, particularly spasm and thrombosis, are more common at the humeral artery than at the femoral artery.

A few cases of PTA-induced dissection have been reported (34,47); stent placement was usually successful. However, in one of our patients, dissection of the CA occurred during attempted

Table 5
Results of Surgery for CMI in the Past Decade

Study and Year	Patients with Signs of CMI	Postsurgical Mortality Rate (%)	Major Morbidity Rate (%)	Rate of Recurrence of Ischemic Symptoms (%)	Symptom-free 3-year Survival Rate (%)	Late, Acute, and Fatal Thrombosis Rate (%)
Mateo et al (49), 1999	85	8	33	19	76	11
Calderon et al (50), 1992	20	0	20	0	100	0
McAfee et al (42), 1992	58	10	42	10	66*	4
Johnston et al (51), 1995	21/34	0	19	14	86	9

*Symptom-free 5-year survival rate.

recanalization. Two stents were implanted, but Doppler US performed the next day showed that the artery was occluded (Table 2).

One case of fatal post-PTA acute ischemia has been reported (34). However, this complication seems less common with PTA than with open surgery.

Comparison with Outcomes after Surgery

Early Outcome.—Our 16 patients were able to eat the day after the procedure and were able to leave the hospital within 1 week. At the 3-month evaluation, they all reported improvement in symptoms (Table 2). This dramatic improvement in symptoms after PTA has been reported consistently. The improvement in symptoms seems somewhat slower after surgery, probably because of the ileus induced by anesthetics.

Midterm Outcome.—The earliest reports of PTA used to treat occlusive SMA lesions were published in 1980 (5,6), and a few series comprising more than five cases have been published (Tables 3, 4). These reports show that outcomes after PTA compare favorably with outcomes after surgery (Table 5). First, the mortality rate seems lower after PTA than after surgery, probably because of the lower rate of postprocedure infarction: After surgery, acute early thrombosis of the bypass may be precipitated by the hemodynamic instability that can occur during general anesthesia and open surgery. Early acute occlusion seems less common after PTA, even with stent placement. A possible explanation is that the major sources of blood supply are left untouched and can become functional should thrombosis develop. The same is not always true of bypasses (particularly with end-to-end anastomosis). In addition, the other complications of PTA are less common than in the past (use of smaller-diameter catheters at the puncture site). Finally, PTA does not expose the patient to the risks inherent in gen-

eral anesthesia: The first patients treated with PTA for CMI had severe cachexia, contraindicating surgery with general anesthesia.

It has been suggested that surgery should be the treatment of choice, at least in selected patients, because it provides a better long-term patency rate (48–51). In the medium term, only two of our 16 patients experienced recurrent symptoms, and in both repeat PTA was successful (Table 2). The data from our patients and other studies show that most recurrences take place within 1 year after PTA and that mortality is related primarily to comorbidities (Table 2).

However, the patency of the treated vessels has not been systematically assessed in our patients, as in most studies. Therefore, it would be interesting to obtain data from a randomized trial that would comprise a patency study over the mid- and long term. Actually, relief of pain does not necessarily equate with patency.

Comparison of the data in Tables 4 and 5 shows that the need for a repeat procedure because of symptom recurrence caused by restenosis may be slightly higher in the PTA-treated population, but the risk of acute obstruction and death is probably lower than with bypass grafting. The rates of late, fatal, acute thrombosis in surgical series are reported in Table 5.

Conclusions

Since the beginning of the 20th century, a large body of data on CMI has accumulated from case reports and small patient series. Because CMI is rare, its pathophysiology remains poorly understood. In particular, the relation between symptoms and arterial lesions is unclear. There is no specific diagnostic test, and the diagnosis continues to rest on clinical grounds. A high index of suspicion should be maintained in patients with postprandial pain and weight loss. An important

step is to eliminate other conditions, even in patients with occlusive visceral artery lesions.

Since the introduction of PTA in 1972, this procedure has become increasingly safe and effective. This very promising technique needs further evaluation, especially in the case of younger patients, before being proposed as the initial treatment in all patients with CMI.

References

1. Councilman WT. Three cases of occlusion of the superior mesenteric artery. *Boston Med Surg J* 1894; 130:410–411.
2. Goodman GH. Angina abdominis. *Am J Med Sci* 1918; 155:524–528.
3. Dunphy JE. Abdominal pain of vascular origin. *Am J Med Sci* 1936; 192:109–112.
4. Shaw RS, Maynard EP 3rd. Acute and chronic thrombosis of the mesenteric arteries associated with malabsorption: a report of two cases successfully treated by thromboendarterectomy. *N Engl J Med* 1958; 258:874–878.
5. Uflacker R, Goldany MA, Constant S. Resolution of mesenteric angina with percutaneous transluminal angioplasty of a superior mesenteric artery stenosis using a balloon catheter. *Gastrointest Radiol* 1980; 5:367–369.
6. Furrer J, Gruntzig A, Kulgemeier J, Goebel N. Treatment of abdominal angina with percutaneous dilatation of an arteria mesenterica superior stenosis. *Cardiovasc Intervent Radiol* 1980; 3:43–44.
7. Ha HK, Lee SH, Rha SE, et al. Radiologic features of vasculitis involving the gastrointestinal tract. *RadioGraphics* 2000; 20:779–794.
8. Moawad J, Gewertz BL. Chronic mesenteric ischemia. *Surg Clin North Am* 1997; 77:357–369.
9. Järvinen O, Laurikka J, Sisto T, Salenius JP, Tarkka MR. Atherosclerosis of the visceral arteries. *Vasa* 1995; 24:9–14.
10. Croft RJ, Menon GP, Marston A. Does “intestinal angina” exist? A critical study of obstructed visceral arteries. *Br J Surg* 1981; 68:316–318.
11. Roobottom CA, Dubbins PA. Significant disease of the celiac and superior mesenteric arteries in asymptomatic patients: predictive value of Doppler sonography. *AJR Am J Roentgenol* 1993; 161:985–988.
12. Maspes F, Mazzetti di Pictralata G, Gandini G, et al. Percutaneous transluminal angioplasty in the treatment of chronic mesenteric ischemia: results and 3 years of follow-up in 23 patients. *Abdom Imaging* 1998; 23:358–363.
13. Casey KM, Quigley TM, Kozarek RA, Raker EJ. Lethal nature of ischemic gastropathy. *Am J Surg* 1993; 165:646–649.
14. Libevski SM, Koch KL, Atnip RG, et al. Ischemic gastroparesis: resolution after revascularization. *Gastroenterology* 1990; 99:252–257.
15. Rha SE, Ha HK, Lee SH, et al. CT and MR imaging findings of bowel ischemia from various primary causes. *RadioGraphics* 2000; 20:29–42.
16. Moneta GL, Lee RW, Yeager RA, Taylor LM, Porter JM. Mesenteric duplex scanning: a blinded prospective study. *J Vasc Surg* 1993; 17:79–84.
17. Perko MJ. Duplex ultrasound for assessment of superior mesenteric artery blood flow. *Eur J Vasc Endovasc Surg* 2001; 21:106–117.
18. Zwolak RM, Fillinger MF, Walsh DB, et al. Mesenteric and celiac duplex scanning: a validation study. *J Vasc Surg* 1998; 27:1078–1087.
19. Giavagnorio F, Picarelli A, Di Giovambattista F, Mastracchio A. Evaluation with Doppler sonography of mesenteric blood flow in celiac disease. *AJR Am J Roentgenol* 1998; 171:629–632.
20. Gentile AT, Moneta GL, Lee RW, Masser PA, Taylor LM, Porter JM. Usefulness of fasting and postprandial duplex ultrasound examinations for predicting high-grade superior mesenteric artery stenosis. *Am J Surg* 1995; 169:476–479.
21. Shirkhoda A, Konez O, Shetty AN, Bis KG, Elwood RA, Kirsch MJ. Contrast-enhanced MR angiography of the mesenteric circulation: a pictorial essay. *RadioGraphics* 1998; 18:851–861.
22. Gilfeather M, Holland GA, Siegelman ES, et al. Gadolinium-enhanced ultrafast three-dimensional spoiled gradient-echo MR imaging of the abdominal aorta and visceral and iliac vessels. *RadioGraphics* 1997; 17:423–432.
23. Ernst O, Asnar V, Sergeant G, et al. Comparing contrast-enhanced breath hold MR angiography and conventional angiography in the evaluation of mesenteric circulation. *AJR Am J Roentgenol* 2000; 174:433–439.
24. Meaney JF, Prince MR, Nostrant TT, Stanley JC. Gadolinium-enhanced MR angiography of visceral arteries in patients with suspected chronic mesenteric ischemia. *J Magn Reson Imaging* 1997; 7:171–176.
25. Cikrit DF, Harris VJ, Hemmer CG, et al. Comparison of spiral CT scan and arteriography for evaluation of renal and visceral arteries. *Ann Vasc Surg* 1996; 10:109–116.
26. Horton KM, Fishman EK. 3D CT angiography of the celiac and superior mesenteric arteries with multidetector CT data sets: preliminary observations. *Abdom Imaging* 2000; 25:523–525.
27. Konen E, Amitai M, Apter S, et al. CT angiography of superior mesenteric artery syndrome. *AJR Am J Roentgenol* 1998; 171:1279–1281.
28. McShane MD, Proctor A, Spencer P, Cumberland DC, Welsh CL. Mesenteric angioplasty for chronic intestinal ischaemia. *Eur J Vasc Surg* 1992; 6:333–336.
29. McKinsey JF, Gewertz BL. Acute mesenteric ischemia. *Surg Clin North Am* 1997; 77:307–318.
30. Bassiouny HS. Nonocclusive mesenteric ischemia. *Surg Clin North Am* 1997; 77:319–324.
31. Erden A, Yurdakul M, Cumhur T. Marked increase in flow velocities during deep expiration: duplex Doppler sign of celiac artery compression syndrome. *Cardiovasc Intervent Radiol* 1999; 22:331–332.
32. Nyman O, Ivancey K, Lindle M, Uher P. Endovascular treatment of chronic mesenteric ischemia: report of five cases. *Cardiovasc Intervent Radiol* 1998; 21:305–313.
33. Matsumoto AH, Tegtmeier CJ, Fitzcharles EK, et al. Percutaneous transluminal angioplasty of visceral arterial stenoses: results and long-term clinical follow-up. *J Vasc Interv Radiol* 1995; 6:165–174.
34. Allen RC, Martin GH, Rees CR, et al. Mesenteric angioplasty in the treatment of chronic intestinal ischemia. *J Vasc Surg* 1996; 24:415–423.

35. Roayaie S, Jossart G, Gitlitz D, Lamparello P, Hollier L, Gagner M. Laparoscopic release of celiac artery compression syndrome facilitated by laparoscopic ultrasound scanning to confirm restoration of flow. *J Vasc Surg* 2000; 32:814–817.
36. Yeh TS, Jan YY, Jeng LB, Hwang TL, Wang CS, Chen MF. Massive extra-mesenteric gastrointestinal haemorrhage secondary to splanchnic artery aneurysms. *Hepatogastroenterology* 1997; 44:1152–1156.
37. Gambiez LP, Ernst OJ, Merlier OA, Porte HL, Chambon JP, Quandalle PA. Arterial embolization for bleeding pseudocysts complicating chronic pancreatitis. *Arch Surg* 1997; 132:1016–1021.
38. Yamakado K, Nakatsuka A, Tanaka N, Takano K, Matsumura K, Takeda K. Transcatheter arterial embolization of ruptured pseudoaneurysms with coils and n-butyl cyanoacrylate. *J Vasc Interv Radiol* 2000; 11:66–72.
39. Chaillou P, Moussu P, Noel SF, et al. Spontaneous dissection of the celiac artery. *Ann Vasc Surg* 1997; 11:413–415.
40. Takeda H, Matsunaga N, Sakamoto I, Obata S, Nakamura S, Hayashi K. Spontaneous dissection of the celiac and hepatic arteries treated by transcatheter embolization. *AJR Am J Roentgenol* 1995; 165:1288–1289.
41. Matsuo R, Ohta Y, Ohya Y, et al. Isolated dissection of the celiac artery: a case report. *Angiology* 2000; 51:603–607.
42. McAfee MK, Cherry KJ, Naessens JM, et al. Influence of complete revascularization on chronic mesenteric ischemia. *Am J Surg* 1992; 164:220–224.
43. Tytle TL, Prati RC. Percutaneous recanalization in chronic occlusion of the superior mesenteric artery. *J Vasc Interv Radiol* 1995; 6:133–136.
44. Maleux G, Wilms G, Stockx L, Vancleemput J, Baert AL. Percutaneous recanalization and stent placement in chronic proximal superior mesenteric artery occlusion. *Eur Radiol* 1997; 7:1228–1230.
45. Sheeran S, Murphy T, Khioaja A, Sussinan S, Hallisey M. Stent placement for treatment of mesenteric artery stenoses or occlusions. *J Vasc Interv Radiol* 1999; 10:861–867.
46. Waybill PN, Enea NA. Use of a Palmaz stent deployed in the superior mesenteric artery for chronic mesenteric ischemia. *J Vasc Interv Radiol* 1997; 8:1069–1071.
47. Odurny A, Sniderman KW, Colapinto RF. Intestinal angina: percutaneous transluminal angioplasty of the celiac and superior mesenteric arteries. *Radiology* 1988; 167:59–62.
48. Rose S, Quigley T, Raker E. Revascularization for chronic mesenteric ischemia: comparison of operative arterial bypass grafting and percutaneous transluminal angioplasty. *J Vasc Interv Radiol* 1995; 6:339–349.
49. Mateo RB, O'Hara PJ, Hertzner NR, Masch EJ, Beven EG, Krajewski LP. Elective surgical treatment of symptomatic chronic mesenteric occlusive disease: early results and late outcomes. *J Vasc Surg* 1999; 29:821–831.
50. Calderon M, Reul GJ, Gregoric ID, et al. Long-term results of the surgical management of symptomatic chronic intestinal ischemia. *J Cardiovasc Surg* 1992; 33:723–728.
51. Johnston KW, Lindsay TF, Walker PM, Kalman PG. Mesenteric arterial bypass grafts: early and late results and suggested surgical approach for chronic and acute mesenteric ischemia. *Surgery* 1995; 118:1–7.
52. Hallisey MJ, Deschaine J, Illescas FF, et al. Angioplasty for the treatment of visceral ischemia. *J Vasc Interv Radiol* 1995; 6:785–791.
53. Golden DA, Ring EJ, McLean GK, Freiman DB. Percutaneous transluminal angioplasty in the treatment of abdominal angina. *AJR Am J Roentgenol* 1982; 139:247–249.