Imaging and Intervention in Gastrointestinal Hemorrhage and Ischemia

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5.3.1 Introduction

Acute gastrointestinal (GI) bleeding is a frequent cause of hospitalization of patients that commonly present melena, hematemesis, or hematochezia. The source of most bleedings can usually be identified through an initial work-up including barium studies and endoscopy; however, 5% of all patients with intestinal bleeding have obscure bleeding in which no definitive source has been identified though routine diagnostic examinations [1]. Detection and localization of the source of acute Intraabdominal hemorrhage is one of the major challenges in the early diagnostic work-up of acute intraperitoneal bleeding. It is very helpful in providing prompt hemostasis accomplished by means of transarterial embolization or surgery.

Helical computed tomography (CT) has been shown to be an effective method for detecting a wide variety of causes of obscure GI bleeding [2, 3]. Advantages over routine diagnostic exams include its ability to localize lesions, identify vascular abnormalities, and evaluate adjacent anatomical structures that may be related to the bleeding.

Bowel ischemia (BI) is not an uncommon disorder representing an increasing threatening abdominal condition as the overall population ages. Bowel and mesenteric ischemia occurs in a variety of conditions that carries out a reduction or an absence of blood supply [4, 5]. This may be acute or chronic depending on the onset and clinical presentation and may involve the small or large bowel, may be partial mural or transmural, and may be segmental or diffuse. Bowel ischemia is a challenging abdominal disease because of its wide range of clinical and pathological manifestations and its high mortality rate, which has remained high over the past 30 years despite technical and medical advances (50–90%) [6]. If suspected, early diagnosis and treatment is crucial because partial mural ischemia may progress to fatal infarction. Clinical onset and plain radiographic findings are nonspecific mainly in early stages of the disease. With recent technical advances improving its sensitivity CT has become the procedure of choice when mesenteric ischemia is suspected because of its capacity to show vascular occlusions, bowel changes, or other related abdominal signs [7]. Multidetector row CT are faster and allow thinner collimation than traditional spiral CT in imaging the entire mesenteric vessels, which is crucial in patients with suspected mesenteric ischemia [8]. In the same way CT and magnetic resonance (MR) may also be helpful in determining other primary causes of BI.

Other imaging techniques, such as ultrasound (US) and barium studies (BS), are less sensitive. The MR is an emerging diagnostic tool and in some studies is as reliable as CT for demonstrating bowel wall changes and vascular anomalies.

5.3.1.1 Vascular Anatomy

The arterial blood supply of the bowel loops is provided by three main arteries: the celiac trunk; the superior mesenteric artery; and the inferior mesenteric artery. Venous drainage is performed mainly by the superior and inferior mesenteric veins [9].

Gastrointestinal tract vascular flow from distal esophagus to the third portion of duodenum depends on the celiac trunk. The first branch of the common hepatic artery (gastroduodenal artery) represents an anastomosis between the superior mesenteric artery and the celiac trunk [6].

The superior mesenteric artery (SMA) arises from the abdominal aorta at the level of the L1 vertebral body, <1.5 cm below the celiac origin, and is just superior to the origin of the renal arteries. The SMA provides blood supply to the distal part of duodenum, the rest of small bowel loops, and the ascending and transverse large bowel to the splenic flexure. Main branches of the SMA are jejunal arteries, ileocolic artery, right colic artery (although absent in 80% of population it aids the ileocolic and middle colic in supplying blood to the ascending colon) and middle colic artery. Other branches include an artery for the right angle of colon and one for the transverse colon. The marginal arteries of Dwight and Drummond supply the vasa recta to the small intestine and colon and provide a channel of potential collateral blood supply to the entire gut. The vasa recta arise from the marginal artery and supply the bowel wall. The arc of Riolan is an inconstant artery parallel to a portion of the middle colic artery. Aberrant branches from the SMA are relatively common (common hepatic artery, right hepatic artery, splenic artery) [10-13].

The inferior mesenteric artery (IMA) arises from the aorta 7 cm below the origin of SMA at the level of L3. IMA supplies the splenic flexure, the descending colon, sigma and rectum. There are several anastomosis to lumbar, sacral and internal iliac arteries. Main branches are left colic artery (absent in 12% or may arise form the SMA), the colosigmoid artery, sigmoid branches and the superior rectal arteries. The branches of these arteries form several arcades to supply the muscularis propia, submucosa and mucosa.

The superior mesenteric vein (SMV) is a single trunk and receives several veins including the ileocolic, gastrocolic, right colic, and middle colic veins. The SMV lies at the right side of the SMA. In patients with complete malrotation of the gut this relationship is reversed [10, 12, 13].

The inferior mesenteric vein (IMV) receives blood flow from superior hemorrhoidal vein, sigmoid vein, and left colic vein. It may end at the splenic vein, at the splenoportal angle, or in the SMV [14].

5.3.2 Gastrointestinal Hemorrhage

5.3.2.1 Etiology

Intraabdominal hemorrhage may result from known conditions, such as bleeding diathesis or blunt abdominal trauma, or may be idiopathic. The latter condition may result from several incidentally discovered etiologies, including rupture of a vascular neoplasm, perforation of a duodenal ulcer, or other gastrointestinal pathologies; and inflammatory erosive processes, such as pancreatitis with subsequent pseudocyst or pseudoaneurysm formation.

Regarding lower GI bleeding, approximately 70% of cases are due to diverticular disease, neoplasms, and benign anorectal diseases that have a focal bleeding site.

5.3.2.2 Clinical Findings

The clinical presentation of spontaneous abdominal hemorrhage, although variable, frequently consists of sudden abdominal pain and distension associated with an acute drop in hematocrit. Uncommon signs include hypovolemic shock and discoloration around the umbilicus and flanks [15]. Signs and symptoms of abdominal hemorrhage may be equivocal, and hematocrit levels obtained during acute phase may not reflect its diagnosis [16]. In addition, clinical signs obtained during physical examination also may be independent from the severity and initiation of the intraabdominal hemorrhage, which is the reason why imaging plays a pivotal role in the diagnosis and assessment of this potentially lethal entity.

5.3.2.3 Diagnosis

Digestive endoscopy is highly effective and provides many solutions in the management of GI hemorrhage; however, it is necessary to point out that its efficacy depends on the experience of the operator and on good intestinal preparation. It should also be remembered that this technique is not useful in a large number of patients with hemorrhages whose origin is in the small bowel. For this reason, several angiodysplastic lesions and inflammatory or tumoral pathologies of this area of the intestine cannot be detected by endoscopy.

Nuclear Medicine

Although these are not equally well accepted in all the clinical management protocols, gammagraphic techniques constitute the next step after endoscopy for diagnosing digestive hemorrhage. They make it possible to detect bleeding with debits as low as 0.2 ml/min, and they are useful in locating the abdominal region in which the bleeding originates; however, they are not able to determine either the cause of the hemorrhage or the exact anatomical location.

Computed Tomography

Among the radiological techniques, CT and angiography are the most frequently used techniques for the study of patients with digestive hemorrhage. In 1989 Sivit et al. [17] were the first researchers to describe the CT appearance of active intraabdominal arterial extravasation in a patient with splenic rupture caused by blunt trauma. In 1991 Jeffrey et al. described 18 patients with active intraabdominal arterial hemorrhage diagnosed using dynamic contrast-enhanced CT [18].

Yamaguchi and Yoshikawa [19] believed that enhanced CT had the potential to show active GI bleeding getting

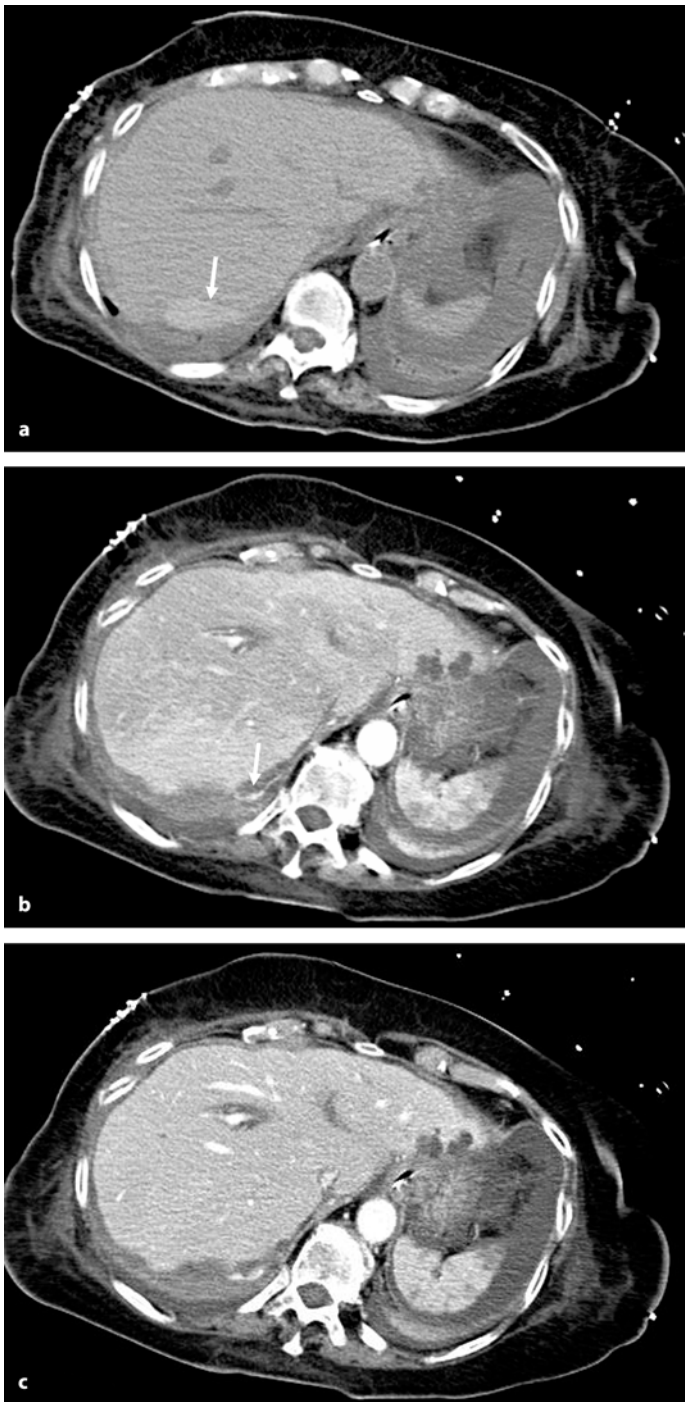

Fig. 1. A 74-year-old woman with hypotension after pancreatic, duodenal, renal, and adrenal resection due to malignant retroperitoneal fibrous histiocytoma. **a** Transversal section of unenhanced multidetector CT scan shows a perihepatic hematoma (*arrow*). **b** A clear jet of extravasated contrast material from a branch of the right phrenic artery, indicating active hemorrhage, is clearly depicted (*arrow*). **c, d** In addition, accumulation of extravasated contrast material is seen. **e** After the catheterization of the right phrenic artery two leaking points of contrast media were found. The bleeding point was successfully embolized with spherical particles (300–500 µm). (Although this interesting case is not a GI bleeding, it has been selected because the accurate information of the MDCT allowed a quick identification and embolization of the bleeding point in a hemodynamically unstable patient. The clinical results and the follow–up was good without any bleeding relapse.)

positive rates for GI bleeding on CT reached 80% [20]. Enhanced helical CT might provide information about the bleeding site [18, 21, 22] and indicates a specific diagnosis, but it is only a diagnostic tool and never therapeutic. When pooling of contrast material is found in the lower GI tract, the next strategy should be adopted immediately. For example, if the bleeding site is the rectum, proctoscopy or colonoscopy is chosen, followed by angiography, if found necessary. Conversely, if the bleeding site is in the right colon or small intestine, angiography is the first choice because colonoscopy has difficulties in reaching the bleeding portion. Then, when enhanced CT promptly provides a map of the bleeding site, an appropriate treatment method can quickly be selected (Fig. 1) [23].

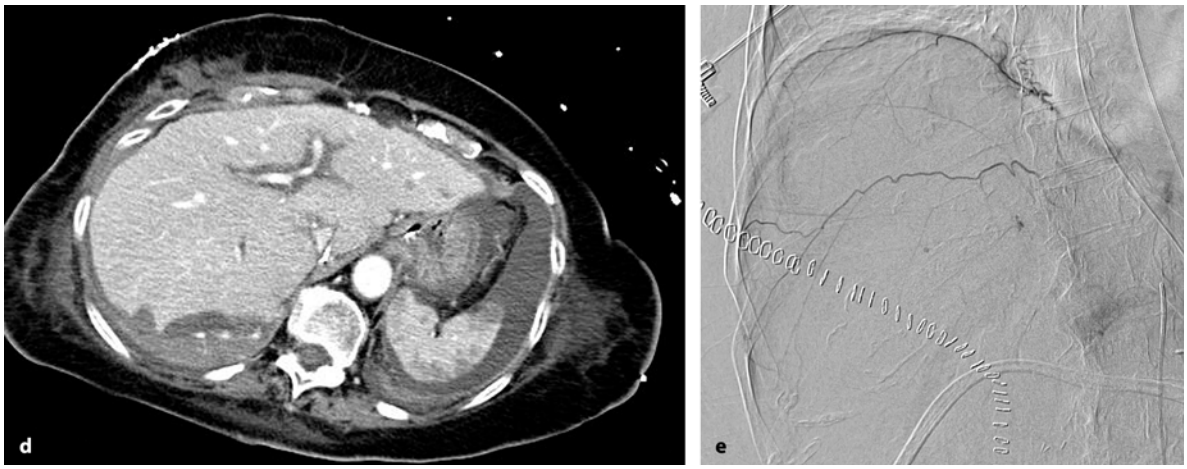

Fig. 1. (*continued*)

Computed tomography can provide indirect signs of bleeding, such as the hematoma, in the wall of the digestive tube, or the presence of localized or diffuse hematic collections. It can also pick up direct signs such as a jet of contrast ("active extravasation") exiting from the bleeding vessel. Active extravasation has been defined as CT evidence of a contrast material collection with attenuation similar to that of the aorta or major adjacent arteries and greater than that of a surrounding parenchymal organ [24].

Recently, with the advent of multidetector-row CT (MDCT) scanners, CT is undergoing further improvement, especially for the evaluation of vascular disease, and subsequently in the evaluation of hemorrhagic complications leading to intraabdominal bleeding.

Direct detection of the bleeding point depends on the technique used for exploration. When using MDCT and high-flow intravenous contrast injection, it is possible to identify the bleeding point directly in 20% of cases.

The administration of an oral contrast agent [24] is not considered to be useful in patients presenting at the emergency department with suspicion of active hemorrhage. The extravasated contrast agent in a patient with active hemorrhage may not be detected when a high concentration of oral contrast material is present within the intestine. In addition, oral application of contrast material, even via a gastric tube, is another time-consuming procedure, usually impractical in the acute clinical setting of these patients, and may interfere with another radiological techniques such as angiography.

Combined with the use of IV-administered contrast material, the faster scanning speed of MDCT and its ability to use a narrow collimation increases opacification of contrast material in the mesenteric, retroperitonel, and portal vasculatures [25, 26]. The superior spatial resolution provided by MDCT enables a more detailed assessment of the parenchymal organs and abdominal vasculature and improves the ability to identify and evaluate abdominal vessels on both axial source images and multiplanar reformations [27, 28]. Computed tomography also makes it possible to identify the lesion responsible (i.e., tumor) and obtain information about its morphology, the degree of affection of the intestinal wall, and possible distant repercussions [29].

Ultrasound

Although US is insensitive in detecting injury in solid organs, in some patients it has demonstrated a relatively high sensitivity in detecting free fluid within the lower abdomen. The sonographic appearance of acute intraperitoenal hemorrhage usually is that of hypoechoic fluid. There may be mixed echoes in this hypoechoic fluid, isolated echogenic clots, or fluid–fluid interfaces [30]. As clot formations occurs, the US appearance may vary, although the clot is usually hypoechoic compared with solid organs.

Magnetic Resonance Imaging

Magnetic resonance imaging also can be used to demonstrate intraperitoneal hemorrhage. A hematoma less than 48 h old may have nonspecific signal isointensity features [31]. Intraabdominal hematoma older than 3 weeks typically has a specific appearance referred to as the concentric ring sign, in which a thin peripheral rim, dark on all sequences, surrounds a bright inner ring, most distinctive on T1-weighted images. The MR imaging can readily distinguish blood from ascites. Acute blood, in the form of deoxyhemoglobin, is low in signal intensity on T2-weighted images. On the other hand, subacute blood, in the form of extracellular methemoglobin, has high signal intensity on T1- and T2-weighted images. The use of fat-suppression techniques accentuates this finding. In a chronic stage, a low signal intensity rim develops around the hematoma on both T1- and T2-weighted sequences. This rim corresponds to hemosiderim or fibrosis.

Angiography

Conventional angiography performed though direct intravascular catheterization is still for many specialists the gold-standard technique in the radiological diagnosis of digestive hemorrhage; however, it is an aggressive technique, which requires training, and the results of which are influenced by some factors including those caused by the patient movements or intestinal peristaltism.

Angiographic detection of the bleeding lesion is based on obtaining direct signs (visualizing the leak of contrast) and indirect signs (tumor vessels, vascular malformations, and others). It is important to remember that in order to detect the bleeding point, the patient has to be bleeding at the moment of the exploration, at least minimally. Although it depends on the equipment and technique used to perform the angiography, several authors have specified that it is necessary for a bleeding of at least 0.3–0.5 ml/min to be detected [32]. Obviously, when the angiography is more selective and precise, it is easier to detect the lesion. When the bleeding point is not found, other techniques can be applied; the first is to use lower-viscosity contrast, such as CO_2 [33]. Management of this contrast medium must be carried out carefully, not because of risks or complications, which are negligible or nonexistent, but because imaging artifacts may occur. Moreover, CO_2 is useful since it can detect very small leaks of contrast medium. Another technical possibility is to "induce" hemorrhage in cases when there is a high clinical suspicion that a particular artery can be the source of the bleeding but the angiography is normal. In those cases, bleeding can be provoked by mechanical stimulation [34] or with drugs. For this purpose, "bleeding stimulation" has been described with urokinase (50–100,000), tolazoline (25–200 mg i.a.), heparin (3–10,000 units i.v.), and tPA (10–50 mg i.a.) [35–39]. The aim of these technique is to open the bleeding point, temporarily occluded by the clot, confirm the precise site of the hemorrhage, and, ultimately, to apply embolizing agents to achieve secure and lasting occlusion of the lesion.

There are indirect signs that suggest the presence of a hemorrhage even without seeing the exact bleeding point. These signs may be clear as the presence of aneurysm, or subtle like the presence of neovascularization or a vessel network. Other signs are hard to interpret, such as the presence of premature venous drainage or a vessel-caliber increase.

Active GI bleeding is a potentially dangerous situation because patients with this condition may go into shock. Colonoscopy, angiography, and scintigraphy have been used widely to localize the source of bleeding, but time is needed to perform these examinations. Enhanced CT may be an alternative to more invasive procedures for evaluation of hemodynamically stable patients with suspicion of hemorrhage [18]. Its simplicity and its ability to detect a wide variety of causes of intestinal bleeding not possible using other methods makes helical CT, and specially MD-CT, an ideal method for detecting vascular lesions such as angiodysplasia and aortoenteric fistulas, small bowel masses, such as lipomas and stromal cell tumors, bowel wall thickening due to radiation enteritis and Crohn's disease, extraintestinal neoplasms, such as pancreatic cancer with metastases, and unusual lesions such as cholesterol emboli [40].

In the context of GI hemorrhage, CT has been proven to be an excellent imaging modality with a rapid diagnostic capability that contributes to a decrease in morbidity and mortality from patients presenting with suspicion of active hemorrhage.

5.3.2.4 Endovascular Treatment

The therapeutic approach to GI hemorrhage must be multidisciplinary and, at the same time, personalized to each patient's circumstance according to the clinical situation and the degree and source of the hemorrhage [41, 42]. Regardless of whether the method chosen is surgery, endoscopy, or endovascular treatment, the aims of therapy have to be the same: to obtain hemostasis and to treat the underlying lesion.

Below we shall analyze the techniques, results, and complications obtained with therapeutic embolization in gastrointestinal bleeding, depending on its origin and cause.

GI Hemorrhage of Hepatic Origin

A hepatic arterial lesion may present as a free intraperitoneal bleeding, as an intrahepatic or subcapsular hematoma, or as hemobilia that is clinically difficult to distinguish from other gastrointestinal hemorrhages [43–46]. If the lesion is in the common or proper hepatic artery, which is uncovered by liver parenchyma, the hemorrhage will generally be massive and difficult to control. The most common cause is the presence of a pseudoaneurysm related in most of the cases to earlier surgery (e.g., liver transplant) [34]. For its treatment, two therapeutic possibilities have been described: the first is to place a covered endoprostheses to seal the bleeding point and maintain the arterial flow at the same time; the second is to embolize the lesion as well as the hepatic artery. With the aim of preventing liver ischemia after the embolization, the gastro-duodenal artery should be left patent, to ensure hepatic arterial perfusion.

Intrahepatic vascular lesions causing hemobilia and GI hemorrhage can be divided mainly into four types:

1. Lesions of small terminal vessels. These lesions are generally caused by hepatic biopsy needles or biliary catheters, and can cause large hemorrhages. As these are distal vessels with a low possibility of connection and re-perfusion from other arteries, the treatment consists of superselective embolization using particles (100–500 μm) or microcoils.

2. Vascular lacerations. These lacerations are almost always caused by the insertion of drainage catheters, or by injuries or lacerating trauma. The artery must be embolized (coils or microcoils) first in the distal part to the lesion ("the back door"), the reason being to prevent "re-perfusion" distal to the bleeding point from occurring through intrahepatic collaterals [47]. Finally, the artery is embolized proximally to the lesion. In cases where there is a stable transhepatic access (e.g., from a biliary drainage), the lesion can be catheterized and treated using this approach. The therapeutic method is similar to the "endovascular," and at times this is the quickest and most useful solution in solving the problem.
3. Vascular lesions with arterio-portal shunt. Sometimes (e.g., after a liver trauma), when performing an arteriography in a patient with a massive hemobilia and arterioportal shunting, opacification of a bile duct is also seen (this sign reflects the massive passage of arterial blood to the bile duct); however, the arterio-portal shunt of traumatic/iatrogenic origin is in most cases the only angiographic manifestation of nonmassive, intermittent hemobilia. Since the bile duct is close to the portal vein, the arterio-portal connection is assumed to be accompanied by an arterio-biliary communication as a result of vascular laceration [48]. By occluding the arterio-portal connection, the bleeding point is thereby also sealed.

It is also important to realize that an arterio-portal shunt produces "arterialization" of the porta, with an increase in pre-sinusoidal portal pressure and therefore an increase in the porto-systemic gradient. This hemodynamic situation, in patients with previous portal hypertension (e.g., cirrhotics), results in a raised risk of bleeding from gastroesophageal varices.

For all of these reasons, it is clinically useful to close the "high-flow" arterio-portal connections. The technique consists of inserting the catheter either well within the shunt or in the artery closest to the lesion. Embolization is performed with coils or microcoils, and the aim is to close the connection directly. If the catheter cannot be advanced to the desired point, it is perhaps better not to embolize, as an excessively proximal occlusion would favor the opening of collaterals distal to the coils, leading to rechannalization of the shunt. In these technically difficult cases, which are uncommon, percutaneous treatment can be performed transparietohepatically by injecting alcohol or thrombin to sclerose the fistula, or by gaining transportal access to reach and occlude the fistula.

4. Vascular laceration with formation of pseudoaneurysm. The most frequent example is that of a lesion in the right hepatic or the proper hepatic artery that appears after open liver surgery, or especially after laparoscopic cholecystectomy [49–51]. In such large pseudoaneurysms, as in the case of vascular injury, the most suitable percutaneous treatment consists of distal and proximal occlusion of the arteries to exclude the arterial lesion while preserving distal flow. On occasions this procedure is not technically possible or is ineffective, so other possibilities must be considered such as direct puncture and sealing of the lesion using coils, gelatine sponge, or thrombin, which is perhaps the best option.

Hemorrhages of Pancreatic Origin

Vascular lesions associated with pancreatic diseases can sometimes manifest in the form of hemosuccus (bleeding through the duct of Wirsung) and then GI hemorrhage. They may originate after surgery and in these cases the treatment consists to embolize the bleeding vessel. Inflammatory lesions of the pancreas frequently cause vascular lesions (pseudoaneurysms), the clinical manifestation of which could be as a massive GI hemorrhage. The most usual endovascular treatment consists of the embolization/occlusion of the bleeding artery. As in lesions of the common hepatic artery, a possible treatment is to seal and exclude the lesion using covered metal stents; however, there is some controversy about this modality of treatment, since the insertion of an endovascular prosthesis in an inflammatory area can, at least theoretically, be contraindicated as the device may become infected.

Hemorrhages of the Upper GI Tract

Upper digestive hemorrhages are those which have their origin above the angle of Treitz. This section focuses on arterial hemorrhages that originate in the stomach and duodenum [52, 53].

Stomach

Gastric vascularization is characterized by the fact that it comes from many different arterial pedicles and, at least, the left gastric, the right gastric (from the hepatic arteries), the gastro-duodenal (with its right gastro-epiploic branch) and the splenic (with the short gastric and left gastro-epiploic arteries) are involved. In cases of digestive hemorrhage from a single bleeding point (e.g., stress ulcers), selective embolization, using segments of gelatine sponge, is highly effective, and necrosis due to post-embolization ischemia is very infrequent.

In cases of diffuse gastric hemorrhage, not controlled by endoscopy, owing to the above-mentioned network of collaterals, nonselective therapeutic embolization has been described as a possible treatment. The aim of this procedure is to reduce temporarily the blood supply to encourage hemostasis. It should not be carried out in patients who have undergone previous gastric surgery, as the stomach will have lost collaterals in some areas. The technique is performed using segments of gelatine sponge and embolizing two "main" pedicles, e.g., the left gastric and gas-

tro-epiploic arteries [54]. Finally, although there are series which contemplate the safe use of this type of nonselective embolization, other authors who have used similar techniques on similar patients have observed gastric necrosis [55].

Duodenum

An important feature of the duodenal vascularization is its dual supply from the hepatic artery and the SMA. Then the pancreato-duodenal arcades function like a true high-flow connection between the celiac trunk and the SMA; therefore, a hemorrhage with its origin in a vascular lesion (caused, for example, by a duodenal ulcer) must be treated bearing this double supply in mind (Fig. 2) [53]. This means that if the lesion is reached by catheterization of the gastro-duodenal artery the end of the catheter or microcatheter should be initially placed distal to the lesion so that a distal seal (e.g., coils) will prevent the entry of blood from another vessel ("re-perfusion"). Once the distal artery is sealed, coils, segments of gelatine sponge, particles (300–500 or 500–700 µm), or glues can be used to treat the bleeding point. The success rate of embolization in duodenal bleeding is around 80% [53]. After obtaining hemostasis, the artery can be closed proximally with coils, although there is some controversy in this regard, since, by doing this, access to the lesion is impeded should further bleeding occur. Similarly, it is not recommended to insert the embolic material inside the lesion since the intracavitary pressure rises and so does the risk of active bleeding during the procedure. Although embolization of the duodenal area is safe and effective, cases of duodenal estenosis have been observed in long-term follow-up, which are caused by fibrosis after ischemia [56].

In addition to the above-mentioned dual supply, the duodenum also has some highly peculiar anatomical characteristics such as aberrant arteries which originate from accessory hepatic arteries, or tumoral areas with their own vascular supply (other than the arcade). For these reasons, every case must be studied in detail so that the therapeutic decision has to be taken individually. The choice of material is also important, not only to prevent long-term complications, but also to achieve the primary aim of embolization, which is hemostasis. For example, in cirrhotic patients or patients with clotting disorders, the application of microcoils alone may be insufficient, and other materials such as gelatine sponge have to be used in addition to them in order to achieve immediate stopping of the bleeding [57].

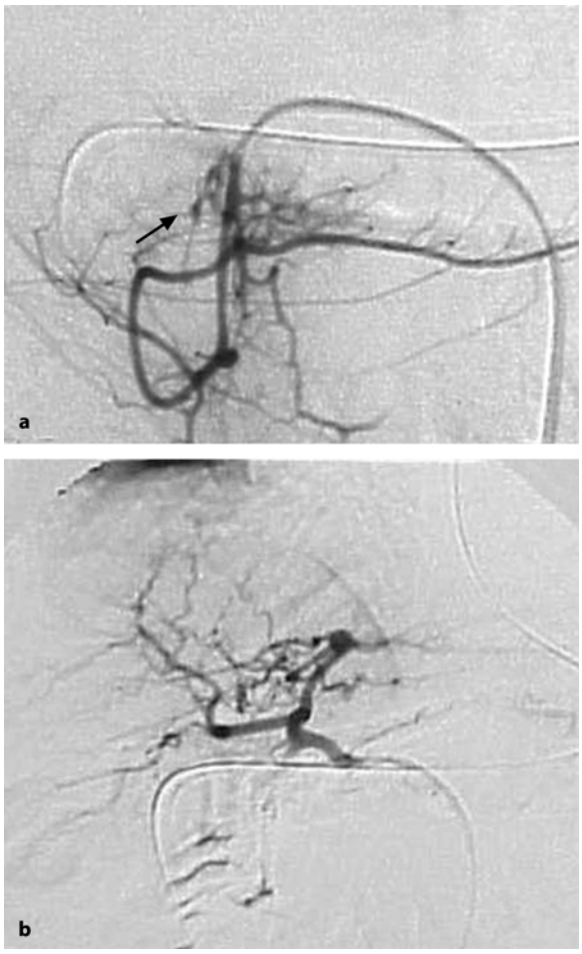

Fig. 2. Active hemorrhage in a patient with a duodenal ulcer. **a** Extravasation of contrast medium coming from the gastro–duodenal artery (*arrow*) is observed. **b** Hepatic arteriography after the embolization of the gastro–duodenal artery

Hemorrhages of the Lower GI Tract

As indicated previously, this section includes all the hemorrhages whose origin is distal to the angle of Treitz. The most common causes of bleeding in the large bowel are diverticula, tumors, and vascular malformations. The majority of these lesions are detected and treated by endoscopic techniques and only in a few cases are surgery or embolization needed. On the other hand, the small bowel is, at this moment, inaccessible to endoscopy, so other diagnostic techniques (such as angiography) and methods of treatment (such as embolization or surgery) acquire greater importance. When a therapeutic decision has to be taken, angiography and surgery do not have to be exclusive or competitive. In some cases of angiodysplasia, aneurysms, tumors, or metaplastic gastric mucosa, the lesion can be detected and identified using angiographic techniques before being surgically resected. For example, patients with multiple vascular lesions and a localized bleeding in just one can benefit from a "pre-surgical angiographic marking" with microcoils or superselective injection of dye in the bleeding vessel, which facilitates a highly economical and extremely precise resection of a short segment of the intestine [35, 58].

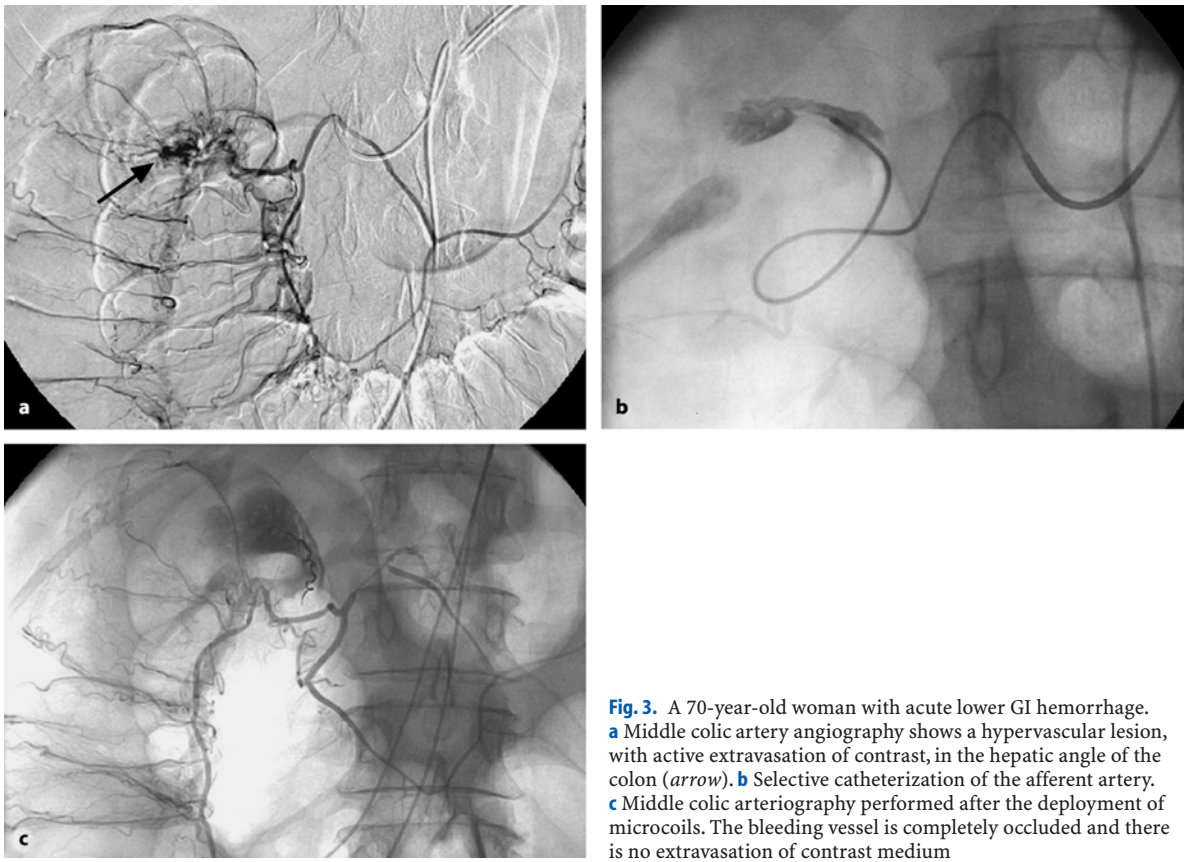

Fig. 3. A 70-year-old woman with acute lower GI hemorrhage. **a** Middle colic artery angiography shows a hypervascular lesion, with active extravasation of contrast, in the hepatic angle of the colon (*arrow*). **b** Selective catheterization of the afferent artery. **c** Middle colic arteriography performed after the deployment of microcoils. The bleeding vessel is completely occluded and there is no extravasation of contrast medium

In the early 1980s, different techniques and embolizing materials were developed to achieve safe hemostasis in a bleeding intestinal vessel; however, complications were observed, mainly ischemia and re-bleeding, probably due to poor technique and incorrect use of the embolizing agent. For this reason, at that time, other methods, such as the local infusion of vasoconstrictors (e.g., vasopressin), were developed. This treatment requires special hospital care, and is not free from cardiac (myocardial ischemia) and intestinal (caused by excessive vasoconstriction) complications. Although results vary according to the area of the bleeding, as it is more effective in colon hemorrhages than in the small intestine, recurrence of the bleeding has been described in up to 50% of cases [59, 60]. Owing to these obvious drawbacks, further attention has been paid to the development of new embolization techniques. New materials, such as microcatheters, spherical particles for embolization, and microcoils, have appeared on the market, making embolization safer and more accurate. As a result, embolization is presently an effective alternative to endoscopy and surgery, and can be recommended, from the technical point of view, for bleeding originating in any lesion of any localization. At present, the indications to perform an embolization for the treatment of a GI hemorrhage are: rebleeding after an unsuccessful endoscopic treatment, and when endoscopy is not feasible, for example, in the small intestine or in patients who have recently undergone abdominal surgery [61]. Another clinical indication is patients who present with massive bleeding and with an unstable hemodynamic situation. An urgent occlusion of the bleeding vessel can be performed in order to improve the clinical situation of the patient, even if embolization may generate intestinal ischemia. In such cases, the "exclusive" purpose of the procedure is to allow the patient to be treated for his or her underlying intestinal lesion in a better hemodynamic condition.

Once the decision to embolize has been taken attention should be paid not only to obtain hemostasis (which in itself is important), but also to the specific treatment of the underlying lesion (technique and materials will be different if, for example, the bleeding comes from an angiodysplasia or from a bleeding aneurysm). In general, for the vast majority of cases, it is recommended that the vascular occlusion be performed at a point, usually the "vasa recta," that is sufficiently distal to prevent rebleeding from collaterals, but proximal enough to prevent infarction of a segment of the intestinal wall. The "vasa recta" differ in morphology and characteristics according to whether it is in the jejunum, ileum, or colon [62]. In the jejunum, they are sparse, long (2–3 cm), and relatively thick, whereas in the ileum they are more abundant, well connected through col-

laterals, but also very thin, and in the colon they are less abundant, and with the origin from the marginal artery. Embolization of the "vasa recta" prevents, in most cases, ischemia of the intestinal wall and facilitates hemostasia by temporarily reducing the blood flow in the affected area. The materials that should be used are microcoils or particles such as "non-spherical" polyvinyl alcohol (500–700 µm) or, even better, the new "spherical" particles (700–900 µm; Fig. 3) [63–65]. The results obtained with embolization can be evaluated as "local success" (occlusion of the lesion) and "clinical success" (absence of bleeding and appearance of complications). Rebleeding ranges from 0 to 20% and complications from 0 to 42% [66]. This wide variation of figures is related with the time of the publication and, at this moment, using adequate materials and an accurate technique, both can be <5%.

The statement made in previous paragraphs that "any bleeding vessel can be embolized" is not completely correct since embolization can be ineffective in some situations and also the possibility of an ischemic complication is always present. This is particularly the case with two groups of patients. The first group are those with diffuse hemorrhage and clotting disorders. In such cases, infusion of vasoconstrictors could be a good option; however, another possibility to obtain "vasoconstriction" without drugs has been recently published. The technique consists in obtaining a temporary vasoconstriction by using catheters and guidewires manipulating them in such a way that they cause an arterial spasm. The spasm will produce a temporary decrease of the blood flow that facilitates hemostasia and the healing of the lesion [67]. The second group of patients are those who have a lesion (aneurysm, rupture, etc.) in a large artery, in whom surgery is contraindicated. In such cases, embolization should not be performed and covered stents could be used to seal the lesion and exclude the bleeding point; however these devices are still not flexible enough to be driven through tortuous curves of some splanchnic vessels.

5.3.3 Intestinal Ischemia

5.3.3.1 Acute Ischemia

Acute mesenteric ischemia may be manifested by the clinical triad of sudden onset of abdominal pain, diarrhea, and vomiting in a patient with pre-existing cardiac disease. These symptoms are nonspecific and result in delayed diagnosis and treatment [68].

Etiology

Acute BI may be secondary to occlusions of the arteries or veins or due to nonocclusive reduction of intestinal perfusion (NOMI). Acute occlusions of arterial trunks, mainly SMA, accounts for most cases whereas venous thromboses are involved in 5–10% of cases and NOMI conditions represent about 20–30% of the total [6].

Acute occlusion of SMA could be due to embolism (50%) or thrombus (25%). The most common source of embolism is the heart (atrial fibrillation, ventricular thrombi, or valvular lesions). About 95% of patients with SMA acute embolism occlusion have previous diagnosis of cardiac disease. Although embolism may be placed at the origin of the artery, the occlusion is usually placed in the middle or distal part of the SMA (Fig. 4). Thrombosis of SMA is most likely to occur proximal and is secondary to atherosclerosis. In these patients the degree of collateral vessels developed and the systemic blood pressure play an important role in the extent and severity of the bowel ischemia [6].

Acute occlusions may be related to other embolic conditions including embolisms form aorta, aortic dissection, cholesterol embolization, aortic surgery, or therapeutic embolization of mesenteric vessels [9, 10].

Antiphospholipid antibody syndrome affects females and among others causes major abdominal vascular thromboses, typically venous, and visceral infarctions [69]. Vasculitis comprises a diverse group of conditions that cause inflammation and necrosis of vessels walls. Pathogenic causes and mechanisms are multiple and not completely understood. The classification accepted presently is the Chapel Hill International Consensus Conference in 1994 in which vasculitis were classified in different categories depending on the size of the vessels affected. There are vasculitis affecting large vessels (aorta and the major trunks; Takayasu arteritis or giant cell arteritis), vasculitis of the medium sized vessels (visceral arteries and their branches; panarteritis nodosa or Kawasaki disease), and small vessel vasculitis (lupus erythematosus, Wegener's granulomatosis, Churg-Strauss syndrome, Schönlein-Henoch purpura), among others. Radiological findings in those patients often overlap. These diseases must be considered in young patients presenting ischemia at unusual sites, affecting small and large bowel and with systemic involvement (lung, genitourinary) [70].

Ischemia caused by the occlusion of mesenteric veins represents about 15–20% of cases and could be secondary to infiltrative tumours (colorectal or pancreas typically), inflammatory conditions (pancreatitis, diverticulitis, inflammatory bowel disease) various types of abdominal infections, blunt abdominal trauma, hypercoagulable states, portal hypertension, oral contraceptive use, or bowel obstruction (volvulus or intussusception). Primary venous thrombosis, when there is no predisposing condition, is unusual. When the thrombosis is proximal and isolated it probably will not cause a severe ischemia due to collateral drainage venous. Acute mesenteric venous thrombosis is defined when symptoms have been present for less than 4 weeks. Typical symptoms include abdominal pain, nausea and vomiting, and constipation with or without bloody diarrhea. Diffuse and intermittent abdominal pain

Fig. 4. Coronal maximum intensity projection (MIP) reconstruction shows an abrupt occlusion of the superior mesenteric artery due to embolism in a woman previously diagnosed with cardiac disease

may be present for days or even weeks. Abdominal distension is the most usual sign. All those symptoms and signs are not specific and may delay the diagnosis, and contribute to the high morbidity and mortality of this condition. The differential diagnosis must include all the causes of acute mesenteric ischemia (arterial and venous) and the radiological findings are similar [71].

Abdominal trauma may produce BI directly when mesenteric vessels and bowel wall are lacerated or indirectly when trauma leads to hemorrhagic shock and subsequent nonocclusive bowel ischemia. Radiological findings include focal bowel wall thickening, perienteric fluid collections, interloop mesenteric hematoma, and hemoperitoneum [72, 73].

Pathology

There are three stages of acute BI. In the first stage there are mucosal necrosis, erosions, and ulcers with hemorrhage. This condition is reversible and usually heals without consequences. If the ischemia extends more deeply into the bowel wall and leads to necrosis of the deep submucosal and muscular layers, strictures and stenosis may develop (second stage). The third stage represents a transmural bowel wall necrosis and infarction. This stage is associated with a high mortality and requires surgery [6].

When mural or mucosal necrosis is present, there is associated wall edema with hemorrhage being more prominent in patients with venous thrombosis, which also presents mesenteric fat edema not present in arterial vascular occlusion.

Imaging

Plain radiography findings are nonspecific with very low sensitivity. Most common findings include an unspecific ileus pattern with dilated loops of the bowel fluid-filled, but the image obtained, in some cases, can be "normal." Focal mural thickening secondary to submucosal hemorrhage (thumbprinting), separated bowel loops by mesenteric fat thickened, intramural gas (pneumatosis), and mesenteric or portal venous gas is seen rarely and indicates late-stage disease. If BI is detected with a plain film, it is usually at late stage of the disease representing severe intestinal changes [7].

Barium studies are useful in patients with atypical presentation, when BI is not suspected and in the postoperative period of an ischemic bowel loop resection in cases when ischemic loops were left in place. Findings include bowel dilatation, thumbprinting, fold thickened, abnormal mucosal pattern, ulceration, and stasis of barium. Barium studies should not be performed if an angiography study will be performed later [7].

Ultrasound

Findings on ultrasound studies are not specific. Vascular thrombus or blood flow changes in duplex and color Doppler studies, distended bowel, hypoechoic and thickened bowel walls, ileus, and fluid collections in peritoneal cavity are common findings in patients with bowel ischemia. Intramural gas can also be detected and, as occurs with the presence of fluid collections, represents a sign of transmural necrosis.

Detection of proximal vascular thrombus and complete occlusion may be diagnosed with color Doppler sonography, but the absence of occlusion in proximal vessels does not exclude the presence of small peripheral thrombus. Although ultrasound has several limitations, such as narrow window by distended bowel loops, its operator dependency or the poor correlation between Doppler flow index anomalies and the severity of ischemia can help identify patients who require angiography [7].

Computed Tomography

To obtain a high sensitivity CT study in a patient with suspected bowel ischemia, performing and following an accurate protocol is essential. Oral and rectal preparation is required, if possible: oral administration of 600–750 ml of high-attenuation contrast material or water (low attenuation) 30–120 min before scanning and rectal administration of 400–800 ml of contrast material or water. Water as oral

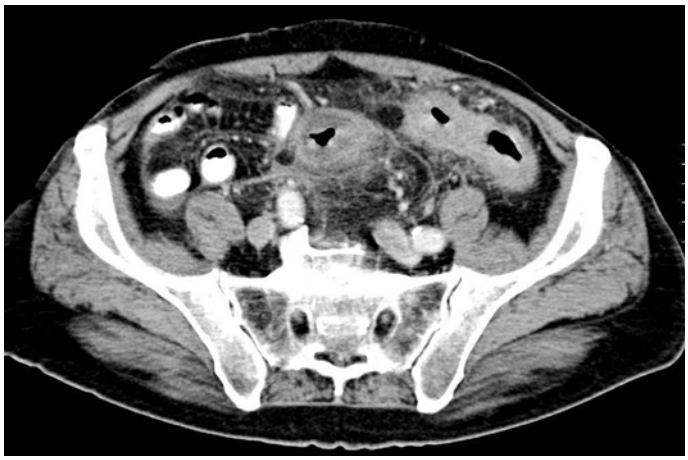

Fig. 5. Axial CT. Typical radiological findings of acute large bowel ischemia: circumferential wall thickening; low submucosal attenuation; and mesenteric fat stranding

contrast material offers two advantages respect to traditional oral contrast: it allows better visualization of the enhancing bowel wall and does not interfere with three-dimensional reformatting techniques (maximum intensity projection, volume rendering and shaded-surface display) [8]. Rectal contrast is essential when ischemic colitis is suspected, to distinguish focal areas of ischemic colitis from colonic contractions [6]. Intravenous administration of contrast material is necessary to show vascular occlusions and to evaluate bowel enhancement. From 120 to 150 ml of nonionic contrast agent are necessary at an injection rate of 3–5 ml/s. A biphasic acquisition is performed with the multidetector row CT. The arterial phase begins 25–30 s after initiation of contrast injection and the portal venous phase at 60 s. Multiplanar reformatted images are created at workstations [74].

The two main roles of CT when BI is suspected are to detect ischemic changes in bowel loops affected, and to determine, if possible, the cause of the ischemia (Fig. 4; vascular occlusion secondary to embolus, thrombus or atherosclerosis, vascular compression due to bowel occlusion, or vascular tumor invasion).

The most common sign in BI is the circumferential thickening of bowel wall. Normal small bowel wall thickness is 5 mm or less. Large bowel can measure in contracted areas >5 mm. The most common sign in BI is the circumferential thickening of bowel wall (Fig. 5). Normal small bowel wall thickness is 5 mm or less. Large bowel wall can measure in contracted areas more than 5 mm of thickness. The thickened wall will show low attenuation due to submucosal edema. If thickened bowel loops show high attenuation, it might be due to submucosal hemorrhage that is usually associated to ischemia The degree of bowel wall thickening does not reflect the severity of the ischemia. It has been demonstrated that reversal ischemia usually shows a much more significant bowel wall thickening than transmural infarction. Some authors have described the thickness of infarcted bowel wall as paper thin reflecting that wall thickening is not proportional to the severity of the ischemia. Reversible shock small bowel wall may manifest with a mild and diffuse wall thickened and nontransmural ischemic colitis may manifest with pronounced wall thickening due to submucosal hemorrhage, inflammation, and/or superinfection being indistinguishable in some cases from transmural colonic infarction.

Enhancement of ischemic loops is less significant compared with normal loops and is highly specific for acute mesenteric ischemia. Bowel walls may show hyperattenuation due to hyperemia or hyperperfusion. Hyperemia of bowel walls is secondary to mesenteric venous occlusion, whereas hyperperfusion appears during reperfusion following occlusive or nonocclusive bowel ischemia or as a result of superinfection and subsequent inflammation. Hyperemia or hyperperfusion of an ischemic bowel segment may be diffuse or segmental and involve the mucosa and submucosa [6–8]. Dilatation of bowel loops and air fluid levels are quiet common in acute bowel infarction and both signs are less common in patients with reversible ischemia and in superficial ischemic colitis. Mesenteric fat stranding, mesenteric fluid, and ascites are nonspecific CT findings but are related bowel obstruction complicated by mesenteric venous occlusion. Partial or transmural ischemia may present these signs [75].

Pneumatosis and portomesenteric gas are signs of late stage of the disease representing severe intestinal changes (Fig. 6). Both signs are less common than the signs previously described but are more specific of acute BI [76, 77].

Magnetic Resonance Imaging

Magnetic resonance imaging might be comparable to CT to show changes of bowel wall and mesenteric vascular anomalies associated to intestinal ischemia. Vascular visualization is markedly improved by the introduction of three-dimensional MR angiography. The MR studies for bowel ischemia are usually performed in patients who cannot undergo CT [7].

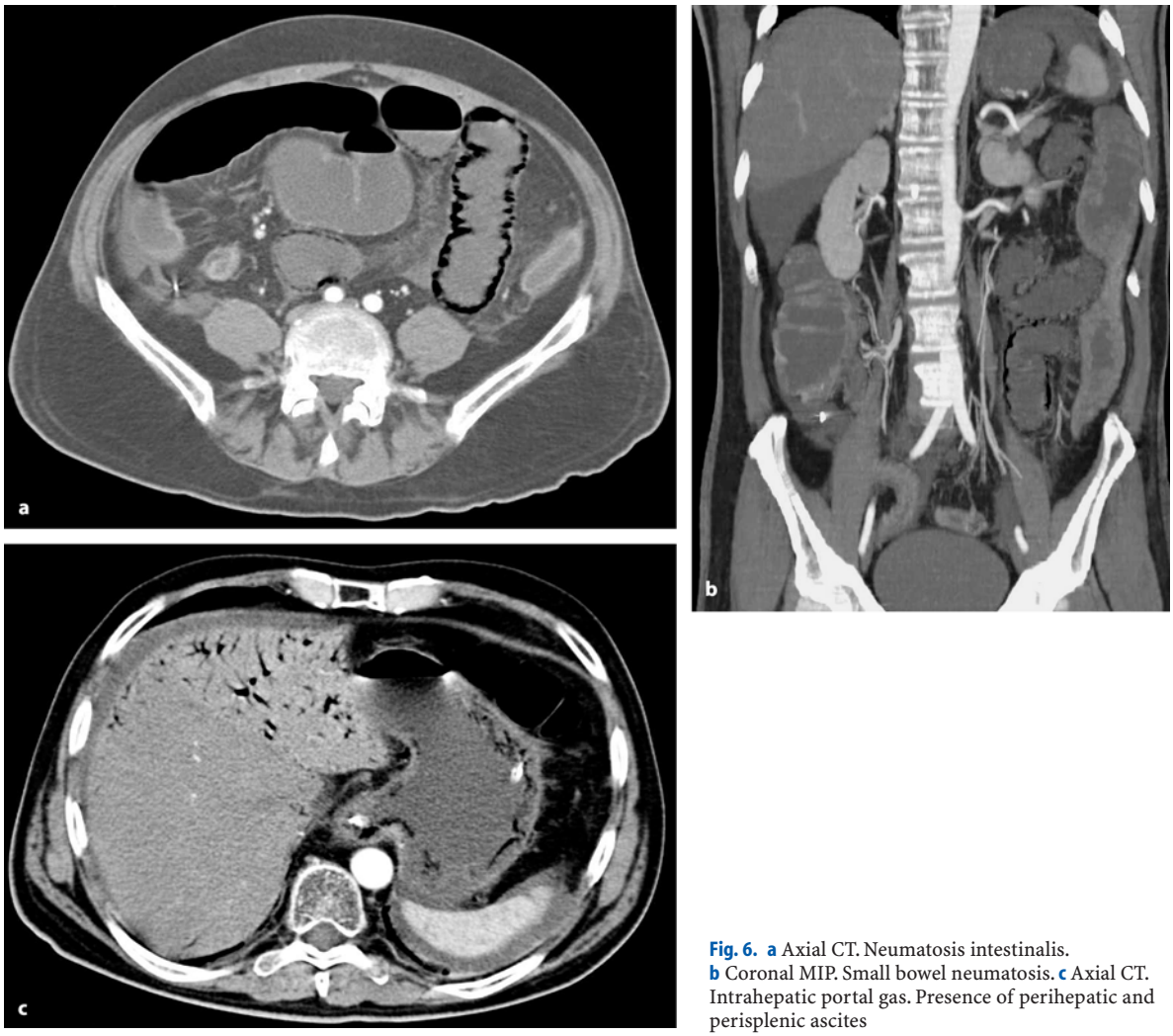

Fig. 6. **a** Axial CT. Neumatosis intestinalis. **b** Coronal MIP. Small bowel neumatosis. **c** Axial CT. Intrahepatic portal gas. Presence of perihepatic and perisplenic ascites

5.3.3.2 Chronic Ischemia

Chronic mesenteric ischemia (CMI), or "abdominal angina," is characterized by weight loss and abdominal epigastric pain that typically occurs 15–60 min after meals, due to the increased demand for splanchnic blood flow, and lasts for several hours. Other symptoms include constipation, flatulence, diarrhea, nausea, and vomiting. Chronic ischemia may produce mucosal damage leading to malabsorption. Symptoms develop insidiously contributing to diagnostic delay.

Etiology

Atherosclerotic disease is the main cause of CMI. In the majority of cases proximal segments of visceral arteries are usually involved. Infiltration of arterial walls by fatty plaques leads to stenosis or occlusion of one or more visceral arteries.

Other less frequent causes of vessel obstruction include fibromuscular dysplasia, Takayasu arteritis, tromboangiitis obliterans, radiation enteritis, drug-induced enteropathy, or extrinsic obstruction or vessel encasement by a tumor.

Pathology

The CMI is a slowly progressive disease. The degree of stenosis or obstruction capable of determining clinical symptoms depends on the anatomic configuration, the speed of progression, and the presence of collateral vessels. The number of arteries that must be involved before symptoms of abdominal angina appear is not clear but it has been suggested that while the three main supplying vessels are variably occluded or narrowed at least two vessels should be significantly compromised. Collateral vessels delay the clinical onset of the disease. These collateral can be divided into two major systems. The first system

connects celiac trunk to the SMA and is composed mainly of the pancreaticoduodenal arteries running between gastroduodenal artery and the proximal SMA. Blood can flow in both directions. The other system connects SMA and IMA and comprises the paracolic arcade (arch of Riolan) and the marginal artery of Drummond. When the three main trunks are occluded or stenotic, the phrenic, lumbar, and pelvic collateral vessels become prominent.

Patients with a single lesion can present pain, whereas patients that present lesions in all three trunks may be asymptomatic. This fact suggests that occurrence of ischemia also depends on the site of the lesion with respect to systems that connect the main trunks. Distal lesions to the collateral systems develop clinical symptoms of ischemia earlier than proximal lesions. Diffuse atherosclerosis of the distal visceral vessels is present in patients with diabetes or end-stage renal disease. These patients are unable to develop collateral vessels.

Another factor in the appearance of ischemia is the speed of progression of the lesions. Inflammatory lesions develop earlier and severe occlusion may be present before collateral vessels are present [78, 79].

Imaging

Ultrasound

Ultrasound is the preferred noninvasive screening test for SMA and celiac trunk artery stenosis. A peak systolic velocity >275 cm/s or greater is highly specific for significant SMA stenosis [80, 81].

Computed Tomography

The CT findings suggesting CMI include the presence of atherosclerotic calcified plaque at or near the origins of proximal splachnic arteries and focal vascular stenosis of proximal mesenteric vessels with prominent collateral development. These findings can be well demonstrated with MDCT. New computer reconstruction techniques allow accurate measurement of arterial stenosis [80, 81].

Magnetic Resonance Imaging

Magnetic resonance angiography shows a good agreement with conventional angiography for the evaluation of visceral arteries and evaluation of significant arterial stenosis. The advantage compared with CT is that it is a safe technique, even when patients have renal disease. The major disadvantage is that it is less sensitive for detecting calcified plaques and oversensitive for assessing vascular stenosis [7].

5.3.3.3 Endovascular Treatment of Intestinal Ischemia

As has been stated previously, intestinal ischemia can be classified into three major categories, as proposed by the American Gastroenterological Association (AGA) in 2000, according to the clinical features: AMI; CMI, also known as "abdominal angina"; and colonic ischemia (CI) or ischemic colitis. The AMI may be further classified into arterial thromboembolism, venous thrombosis, and NOMI [82].

This classification is important from a practical viewpoint because, for example, an arterial thromboembolism represents a true emergency, whereas CMI does not. Also, the mortality associated with each of them is different, and mesenteric venous thrombosis is not as lethal as an acute thromboembolism of the SMA or NOMI.

Acute Ischemia

Despite the progress made in the understanding of the pathophysiology, diagnosis, and treatment of this group of diseases, mortality still remains as high as decades ago, ranging between 59 and 93% in different published series [82, 83]. The mortality increases with age, the extension of bowel infarction and, most importantly, the time delay between onset of symptoms and intervention; the latter is the most important one, as it is entirely dependent on us. Thus, instituting an early diagnosis and treatment, before BI develops, results in marked improvement in survival rate [84].

The choice of treatment in many cases depends largely on the local availability for performing emergency angiography. Endovascular techniques do have a role to play, either alone or in conjunction with surgical treatment, but controlled trials are lacking because of the relative infrequency of these conditions.

Several therapeutic options are available from an endovascular viewpoint for the treatment of GI ischemia; these may be grouped into two broad categories depending on whether they are based on the use of mechanical devices (balloon catheters and stents) or pharmacological agents (fibrinolitics and vasodilators).

From a practical viewpoint there are four distinct entities that may result in acute mesenteric ischemia: SMA embolus; SMA thrombosis; and NOMI and mesenteric venous thrombosis.

In the presence of peritoneal signs and symptoms, there is uniform agreement that the treatment should consist of an exploratory laparotomy, together with resection of any necrotic portion of bowel. A primary anastomosis may be fashioned but, when blood perfusion of the anastomosis is not confirmed, it has been postulated that a second-look operation should be done within 12–24 h to confirm intestinal viability [85–92]. A recently published study, however, questioned the efficacy of the second-look operation in improving patient survival [93].

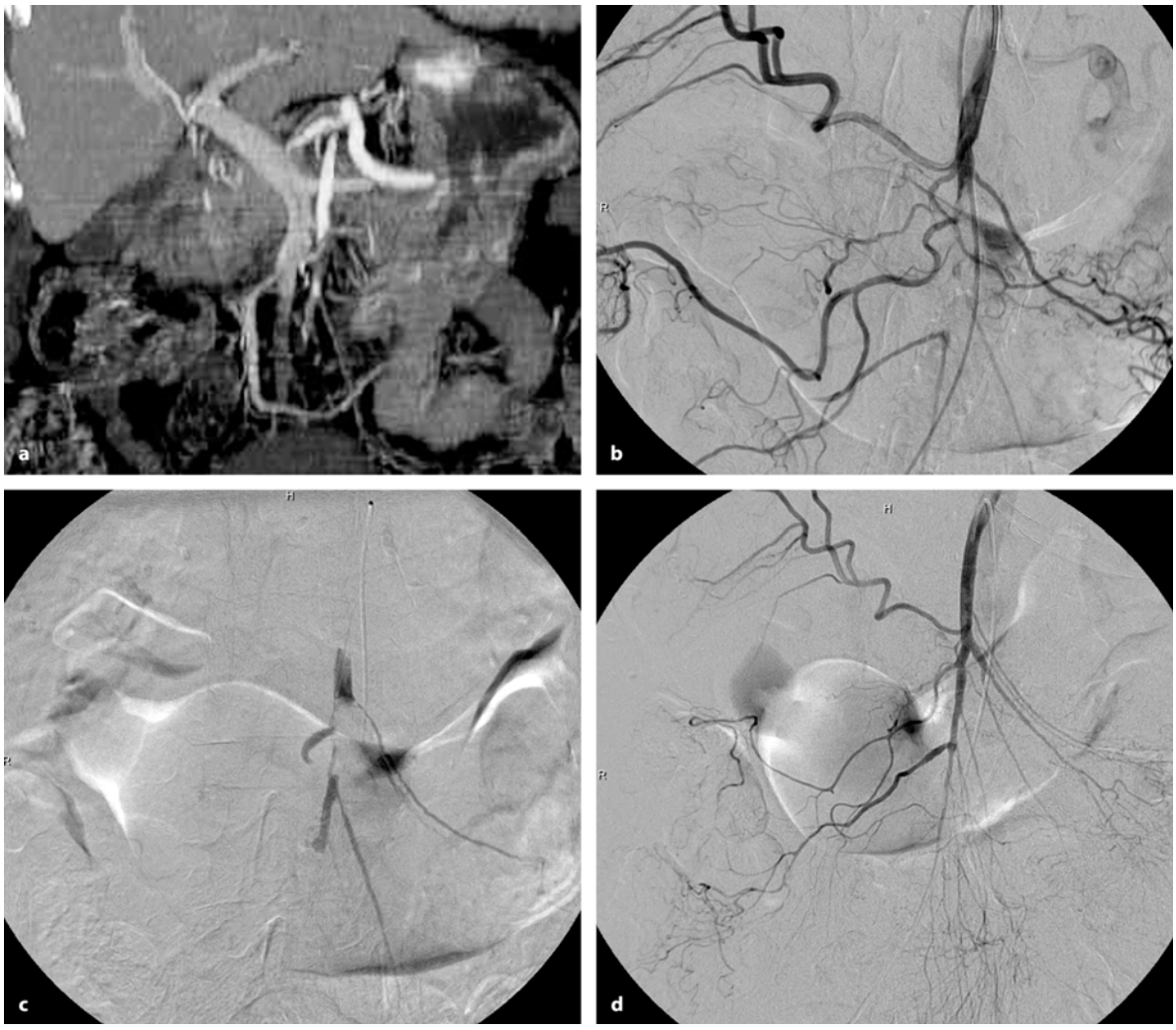

Fig. 7 a, b. A CT reconstruction and digital subtraction angiography show a filling defect within the superior mesenteric artery. The ileocolic artery is patent as well as the middle colic artery. The jejunal branches are underfilled with contrast. Note the absence of collateral circulation. A diagnosis of superior mesenteric embolus was made. **c** A microcatheter was placed with its tip just proximal to the thrombus and an infusion of urokinase (500,000 U in total) was started. **d** Six hours later, the embolus dissolved completely, and the arteries were patent. (Note, however, the severe vasospasm present, which mainly affects the jejunal and ileal branches.)

Establishing whether the occlusion is embolic or thrombotic may be difficult at times, as the angiographic findings may be similar. The presence of a meniscus sign or collateral circulation may help to point to one or the other. Differentiation between acute and chronic thrombosis can also be difficult at times, but visualization of collateral circulation with late filling of SMA branches favors the diagnosis of chronic disease.

In the presence of an embolus, an embolectomy with a Fogarty catheter may be performed through a SMA arteriotomy. A thrombosis, on the other hand, may be treated with an aortomesenteric bypass. Employing the saphenous vein as a graft has the advantage of reducing the risk of infection in comparison with a prosthetic one, but the risk of it kinking is higher [94].

When peritoneal signs are absent, in the presence of an embolus, an endovascular treatment consisting on the infusion of fibrinolitic agents (urokinase, streptokinase, or recombinant tissue plasminogen activator, t-PA) may be performed [95–102]. Different doses have been reported in case studies and series. Typically for streptokinase a 250,000-U intraarterial bolus followed by a low-dose continuous infusion of 5000–10,000 U/h. This is the least expensive of all but bears the drawbacks of potential allergic reactions (3%) and neutralization with antibodies to streptococcus. Urokinase, being an endogenous substance, does not have such shortcomings but is more expensive. The dosage used is 200,000–250,000 U as an intraarterial bolus followed by an infusion of 60,000–120,000 U/h. Finally, t-PA is the most expensive but has the advantage of being fibrin

selective, acting primarily on fibrin-bound plasminogen. A typical dose would be a 20-mg slow intraarterial bolus, followed by a subsequent 20-mg bolus 12 h later (Fig. 7) [103].

The outcome of this kind of treatment improves when the embolus is distal to the origin of the ileocolic artery or partially occluding the SMA trunk. Vasodilators have also been used on their own when the distal vascular bed is well founded; however, their main application is in conjunction with surgical embolectomy. In this context they have proved to be particularly helpful by relieving the vasoconstriction present in association with an embolus that may persist even after an embolectomy [104].

The use of angioplasty and stent placement for acute SMA thrombosis has also been reported [105, 106].

Patients suffering from NOMI may greatly benefit from the infusion of vasodilators on their own or in association with surgical resection of necrotic bowel. Early angiography before intestinal necrosis develops may prove useful in order to prevent an unnecessary laparotomy that would exacerbate vasoconstriction [94, 197]. An infusion of papaverine hydrochloride (60-mg bolus followed by an infusion at a dose of 30–60 mg/h) may be started at the time of angiography [108–111]. There are small series published in which the high mortality rate from this condition has been greatly reduced by this therapy [112]. This is in addition to any measures needed to revert the underlying condition, which predisposes to hypotension, such as hypovolemia, heart failure, or arrhythmia.

The treatment for mesenteric venous thrombosis depends on whether the diagnosis has been made incidentally in an asymptomatic patient or not. In asymptomatic individuals, in whom the diagnosis is an incidental finding, it has been proposed to rely either on no therapy or on a 3- to 6-month course of anticoagulation, although there are no studies published to support such a decision. Such regime consists of heparin for 7–10 days followed by oral anticoagulants for 3–6 months [113]. Symptomatic patients with peritoneal signs are candidates for urgent laparotomy with resection of necrotic bowel. Symptomatic patients with absence of peritoneal signs may be treated in the same way as asymptomatic patients, but the local infusion of fibrinolitic agents has also been proposed, either transarterially or directly into the vein through a transhepatic approach [114].

Chronic Ischemia

The CMI ("abdominal angina") does not require emergency treatment. It is usually the result of atheromatous involvement of the splachnic circulation, but there is a specific entity (extrinsic compression of the celiac trunk by the median arcuate ligament of the diaphragm) which tends to affect younger individuals that requires separate consideration.

The first consideration should be to decide which stenosis requires treatment, as it is not uncommon to find asymptomatic patients with atheromatous lesions in one or more vessels. It is generally accepted that patients with abdominal angina and unexplained weight loss, in whom other types of GI disease have been excluded, whose angiograms reveal stenosis in two of the three vessels, should be treated.

Traditionally, surgical revascularization has been the preferred therapy for these patients [115]. Presently, balloon dilatation and stent placement seem to be a reasonable alternative, although it is difficult to establish which treatment is better. From the published series the rates of clinical success seem to be similar; however, the recurrence rate of angioplasty (without stent placement) is higher [116]. It is likely that the use of stents diminishes the recurrence rate; therefore, making this the therapy of choice. The evidence to support this is still scanty but promising [117, 118].

Celiac artery compression syndrome is a controversial entity and is thought to be a syndrome of abdominal pain caused by compression of the celiac trunk by the median arcuate ligament and perhaps by dense encasement by periarterial neural tissue. Compression of the celiac artery is thought to cause intimal fibrosis that leads to luminal stenosis and impaired splachnic blood flow. This would result in symptoms similar to those of atherosclerotic mesenteric ischemia compression, which nearly always is caused by at least two major visceral artery occlusive lesions. Patients are typically young adults, especially women, and the clinical features are asthenic body habitus, intermittent abdominal pain, epigastric bruits, and rapid weight loss, but the patients often do not have symptoms. Symptoms may be the result of compression of a single visceral artery in the absence of adequate collateral vessels. Mesenteric steal or neurogenic mechanisms have been proposed as other possible causes. This entity does not benefit from angioplasty and stenting alone. In this situation surgical decompression together with arterial reconstruction or dilatation appears to be the preferred therapy, particularly when there is a persistent vessel deformation [119].

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