Updates in the Management of Non-traumatic Abdominal Vascular Emergencies (Abdominal Aortoiliac Aneurysms, Intestinal Ischemia, Splanchnic Aneurysms)

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36.1 Aortoiliac Aneurysms

Aneurysms involving abdominal aorta and iliac arteries are relatively common and potentially life-threatening.

Aneurysms usually result from degeneration in the media of the arterial wall, leading to a slow and continuous dilatation of the lumen of the vessel.

Most of the abdominal aortic aneurysms (AAA) are asymptomatic and detected as an incidental finding on diagnostic imaging obtained for other reasons. Rupture of an aortic aneurysm represents an emergency that can lead to 100% of mortality if not treated and needs prompt diagnosis and treatment.

Historically ruptured abdominal aortic aneurysms (rAAA) were treated with conventional open surgery. Despite significant advances in intensive care unit management and surgical techniques, mortality following repair of ruptured AAA remains high. Following the experience and good results with endovascular treatment in elective setting, operators suggest that also rAAA treatment may be improved using endovascular technique (endovascular aneurysm repair, EVAR). Theoretically, ruptured EVAR (r-EVAR) has several advantages, including the option to perform the entire procedure or parts of the procedure under local anesthesia, avoiding the hypotensive effects of general anesthesia, and the fact that a percutaneous approach minimizes blood loss and avoids laparotomy. But aortic endograft under emergency circumstances presents many challenges, and it’s far to be the “gold standard” for ruptured abdominal aneurysms. Among aortoiliac emergencies nowadays, we have to mention the failure of endovascular treatment with the need for open conversion. Surgical techniques of prosthetic explants in case of failure of EVAR are still debated.

36.1.1 Epidemiology, Risk Factors, and Natural History

AAA can be defined as an abdominal aortic diameter of 3.0 cm or more in either anterior-posterior or transverse planes. The prevalence of AAA increases with age; an incidence of 12.5% in men and 5.2% in women between 74 and 84 years of age is reported. A diagnosis of aortic aneurysm is performed in about 700,000 persons in Europe, 220,000 each year. It accounts for approximately 11,000 deaths every year in the United States, with mortality rates from ruptured AAAs reaching up to 90%. Risk factors for AAA are similar to those of other cardiovascular diseases: male sex, smoking, age older than 65 years, coronary artery disease, hypertension,
previous myocardial infarction, peripheral arterial disease, and a family history of AAA. Beyond the inherent risk of rupture, patients with AAA are also at an increased risk of cardiovascular disease and death [1]. The reported growth rate of AAAs is between 30 and 55 mm ranges from 0.2 to 0.3 cm per year, with a wide variation between patients. Larger AAA diameters are associated with higher AAA growth rates. The most important factor related to aneurysm growth rate is smoking. A larger initial aneurysm diameter is a significant and independent risk factor for AAA rupture. Other factors that have been associated with an increased risk of AAA rupture across several studies include female gender, hypertension, AAA expansion rate, and peak AAA wall stress [2].

Rupture of an aortic aneurysm represents an emergency that can lead to 100% of mortality if not treated. The risk of rupture depends on aneurysm diameter (1% between 40 and 49 mm up to 33% for AAA > 7 cm). The incidence of ruptured abdominal aortic aneurysms ranges between 5.6 and 17.5 per 100,000 person-years in Western countries and seems to have declined in the last decade. The overall mortality rate in all patients with a ruptured abdominal aortic aneurysm (rAAA) is around 80%. One-third of all patients with rAAA do not reach the hospital alive, and one-third do not have an intervention. The operative mortality of ruptured aortic aneurysm has not improved significantly in recent years, with mortality rates still ranging from 32 to 80% [3].

36.1.2 Clinical Presentation

The detection of an aortic aneurysm with physical examination is moderately sensitive (sensitivity of 68% and specificity of 75%). The most common finding is palpation of a pulsatile mass around the level of the umbilicus. Abdominal auscultation may reveal the presence of a bruit. Obesity, abdominal distention, and smaller aneurysm size can reduce the accuracy of abdominal palpation. Rarely symptoms due to compression of adjacent structures are described. Lower extremity edema may be related to compression of the inferior vena cava. Rarely aortocaval or aortoenteric fistulae can occur. Lumbar pain related to vertebral lesion may be due to a large dimension aneurysm erosion activity. Compression of bowel may cause gastrointestinal symptoms like dyspepsia, weight loss, or bowel obstruction, as well as ureteral compression that may lead to hydronephrosis. Distal embolization from aortic thrombus or aneurysm complete thrombosis could be the cause of lower limb acute ischemia. Otherwise, most of the aortic aneurysms are asymptomatic, and diagnosis of AAA is often made as an incidental finding on imaging studies, such as abdominal ultrasonography or computed tomography. AAA may be visible on plain radiography if the aneurysm wall is calcified [1].

A ruptured aortoiliac aneurysm is characterized by hypotension, shooting abdominal or back pain, and a pulsatile abdominal mass. This triad may be incomplete or absent, and misdiagnosis can occur in up to 60% of cases [4]. Abdominal pain or other symptoms occurring in a patient known to have or newly diagnosed with AAA can present a clinical dilemma. In surgical series, between 5 and 22% of AAA are asymptomatic (Fig. 36.1). In the absence of rupture, pain or other symptoms may indicate rapid expansion causing compression of adjacent structures, or an inflammatory or infected AAA. Symptoms that may be related to AAA include abdominal pain or back pain, signs of acute thromboembolism, and fever. A triad of chronic abdominal pain, weight loss, and elevated erythrocyte sedimentation rate in a patient with an AAA is highly suggestive of an inflammatory aneurysm. Patients with inflammatory aneurysms are often more symptomatic than patients with the more typical AAAs, but the incidence of actual rupture may be lower.

36.1.3 Management and Indication to Treat

The management of AAA depends on the size or diameter of the aneurysm and is a balance between the risk of aneurysm rupture and the operative mortality for aneurysm repair. For very small aneurysms between 3.0 and 3.9 cm, the risk of rupture is very low, and these aneurysms do not require surgical intervention. Two large multicentered randomized controlled trials of early open elective surgery versus surveillance, the UK
Small Aneurysm Trial (UKSAT) and the American Aneurysm Detection and Management study (ADAM), and a smaller trial of endovascular repair versus surveillance (CAESAR) [5] were performed in order to analyze the management of aneurysms from 4.0 to 5.5 cm in diameter. PIVOTAL trial [6] focused only on the 4.0 and 5.0 cm diameter range and compared early endovascular repair versus surveillance. In the UKSAT no significant difference in all-cause mortality at 5 years between the two groups was registered, and results were similar after 12 years of follow-up. The aneurysm rupture rate was 1% per year in the surveillance group, and the elective mortality rate for open surgery in the immediate repair cohort was 5.6%. Most patients in the surveillance group eventually underwent surgery because of aneurysm enlargement. Cost-effectiveness analyses suggested that surveillance was less costly than early surgery. In the ADAM study, rupture rate in the surveillance group (0.6% per year) and perioperative mortality rate in the surgery group (2.7%) were lower than in the UKSAT [7]. As with other studies, 60% of the surveillance group underwent operative AAA repair because of aneurysm enlargement. The findings of these two trials, summarized in a recent Cochrane review (at 6 years HR 1.11 [95% CI 0.91e1.34]), show the safety and hence benefits of a policy of ultrasonographic surveillance for aneurysms 4.0 e 5.5 cm in diameter [8].

When the diameter of 5.5 cm, measured by ultrasonography, in males is reached or symptoms develop or rapid aneurysm growth is observed (>1 cm/year), immediate referral to a vascular surgeon is recommended. Patients with a higher risk of rupture should be considered for surgery when the maximum aortic diameter reaches 5.0 cm. Females suffer AAA rupture at smaller aortic diameters than males and should be referred to vascular surgeons for assessment at a maximum aortic diameter of 5.0 cm, and aneurysm repair should be considered at a maximum aneurysm diameter of 5.2 cm in females.

Coexisting iliac aneurysms should be treated concurrently with AAA, and aortoiliac aneurysms comprise up to 43% of a specialist vascular surgeon’s workload. Isolated iliac aneurysms may be treated by either open or endovascular techniques. Intervention should be considered when the iliac diameter exceeds 3 cm.

In case of symptomatic aneurysms, it’s necessary to exclude other sources of symptoms. When no other cause is apparent, an urgent repair is suggested. In the presence of very large AAA (>6.0 cm), treatment is suggested even if the aneurysm is not felt to be the source of symptoms [9–11].

Although AAA repair should be offered to most patients with ruptured AAA, some patients may be at such high risk due to underlying comorbidities that comfort care is appropriate.

### 36.1.4 Symptomatic and Ruptured AAA

AAA rupture is defined as bleeding outside the adventitia of a dilated aortic wall (Fig. 36.2). Rupture is further classified into free rupture in the peritoneal cavity, often associated to shock and retroperitoneal rupture where the retroperitoneal tissue provides tamponade and reduces temporarily the volume of blood loss. Differentiation between symptomatic and ruptured aneurysms is critical. Symptomatic AAAs are those that have become painful but without breach of the aortic wall and without signs of shock. The inclusion of symptomatic AAAs in data on ruptured AAAs will artificially improve the results of outcomes.

The initial management of the patient with symptomatic (non-ruptured) or ruptured AAA is guided by the hemodynamic status. If a patient with a known aortic aneurysm presents signs of shock and symptoms linked to an aneurysm rupture, further diagnostic does not seem mandatory, and the patient should be immediately transferred to the operating room. Emergency ultrasound scanning can be done to confirm the suspected diagnosis. Most patients with a ruptured abdominal aortic aneurysm who reach the hospital alive are sufficiently stable to undergo computed tomography for further therapy setting. The timing of surgery for patients with symptomatic but unruptured aneurysms remains controversial. For patients determined to have a symptomatic AAA, but for whom repair will be delayed to optimize associated medical conditions, an observation period in intensive care unit setting is indicated.

Patients with AAA who require emergent or urgent aortic surgery for ruptured or symptomatic (non-ruptured) AAA should be treated at a facility where surgical expertise and/or the perioperative resources necessary for major aortic surgery are available (e.g., operating room personnel, an appropriately trained surgeon, perioperative intensive care). For patients who present to a facility where these are not available, transfer to a vascular center with higher levels of hospital resources is appropriate and may result in lower mortality. Outcomes for open surgical repair of ruptured AAA are correlated with surgeon experience with a higher annual caseload of open aneurysm repair per year (non-ruptured and ruptured) correlating with improved outcomes [12–14].

![Fig. 36.2 CT scan of retroperitoneal rupture of AAA](image-url)
36.1.5 Perioperative Management

The treatment of a patient with rAAA requires a multidisciplinary approach to ensure a quick diagnosis, appropriate preoperative and perioperative support, efficient aneurysm repair, and excellent postoperative care. The primary goal in the initial management of patients with rAAA is achieving hemodynamic stability to allow perfusion of the vital organs. Two large-bore peripheral intravenous catheters should be placed in all patients (symptomatic non-ruptured or ruptured AAA) for medication and fluid administration. In hemodynamically unstable patients with ruptured AAA, indirect evidence from the trauma population and one observational study in patients with AAA suggest that allowing a relatively low systolic blood pressure of 80–100 mmHg (permissive hypotension) may prevent further tearing of the aorta and limit blood loss. Dick et al. report that aggressive volume resuscitation of patients with rAAA before proximal aortic control resulted in an increased perioperative risk of death independent of systolic blood pressure [15–17].

Pain control is an important part of management. It is important to keep the patient comfortable, but conscious. In patients who remain severely hypertensive despite adequate pain control, short-acting intravenous beta-blockers can be used to reduce the blood pressure. Laboratory studies including complete blood count, electrolytes, blood urea nitrogen, creatinine, liver function tests, prothrombin time, partial thromboplastin time, and a type and crossmatch should be obtained. Similar to trauma patients with severe ongoing hemorrhage, patients with ruptured AAA often require massive transfusion.

36.1.6 Aneurysm Repair

Two methods of aneurysm repair are currently available: open surgery and endovascular aneurysm repair (EVAR).

Open AAA repair—Open aneurysm repair involves replacement of the diseased aortic segment with a tube or bifurcated prosthetic graft through a midline abdominal or retroperitoneal incision.

EVAR—EVAR involves the placement of modular graft components delivered via the iliac or femoral arteries, which line the aorta and exclude the aneurysm sac from the circulation.

36.1.6.1 Open Surgical Treatment

The most important step in rAAA surgery is the rapid, safe, and effective control of the proximal aorta with a consequent reduction of blood loss. After aortic clamping takes place, the anesthesia team should replace blood loss more aggressively. The aorta may be approached either transperitoneally or through the retroperitoneal space. Self-retaining retractors are used. The bowel is kept warm and, if possible, is not exteriorized. An infrarenal control and clamping if possible are performed. Often a suprarenal control is necessary and allows a quick and safe control of the aorta in a bloodless field. However, it causes a visceral ischemic injury that can contribute to the development of multi-system organ failure. In addition, suprarenal control can increase the cardiac afterload and promote myocardial ischemia. Another method for aortic control is balloon occlusion. Proximal control of the aorta can be carried out by placing a balloon in the proximal aorta without suprarenal exposure. An occlusion balloon can be inserted directly into the aorta or under fluoroscopic guidance through the femoral or brachial artery. Due to the risk of coagulation disorders, dissections should be minimized to reduce injury to blood vessels and other structures. The patient should be kept warm because hypothermia can lead to surgical bleeding and adverse cardiac events. Heparin administration is controversial [18].

Key Points

In rupture abdominal aortic aneurysms

- Hemodynamic status of the patient decides the following steps of treatment.
- Permissive hypotension is necessary to reduce massive bleeding and keep the patient alive.
- In case of hard proximal aortic clamping, use an intra-aortic balloon.
- If abdominal pressure is high, perform a temporary abdominal closure.

For symptomatic, non-ruptured AAAs, perioperative mortality rates are similar to those of elective repair. The mortality associated with ruptured AAA may be as high as 90% when patients who die at home or upon the arrival to the hospital are taken into account. In spite of obvious improvements in prehospital care, cardiovascular anesthesia, and critical care, surgical mortality following open repair of ruptured AAA has changed very little, remaining at approximately 30–50%. Factors that are associated with increased mortality following open repair of ruptured are reported in Table 36.1.

For patients with several prognostic factors for poor outcome, the incidence of serious comorbidities, such as dialysis dependence, colonic ischemia, and myocardial infarction, is high, and the need for surgery related to a complication is
Elective endovascular aneurysm repair demonstrates a reduction in perioperative (30-day) morbidity and mortality compared to open repair. There is accumulating evidence that morbidity and mortality following repair of symptomatic or ruptured AAA may also be reduced. The first successful endovascular repair of a rAAA was performed in 1994 by Marin et al. [23] EVAR is less invasive, avoids damage to periaortic and abdominal structures, reduces bleeding from surgical dissection, minimizes hypothermia, and lessens the requirement for deep anesthesia. Because of these potential advantages combined with reports of lower procedural mortality, EVAR has been regarded as superior to OSR for the treatment of rAAA. Observational studies have reported improved short-term survival after EVAR compared with OR, but methodological limitations lead to biased outcomes. Three randomized controlled trials have compared OSR and EVAR in the treatment of patients with rAAA. Hinchliffe et al. observed 32 patients with rAAA, finding a 30-day mortality rate of 53% in the EVAR group and 53% in the OSR group. Moderate or severe operative complications occurred in 77% of the patients in the EVAR group and 80% of the patients in the OSR group. The median total hospital stay in the EVAR group was 10 days, compared to 12 days in the OSR group. Reimerink et al. [24] randomized 116 patients with rAAA to treatment with either OSR or EVAR. The combined rate of death and severe complications at 30 days postsurgery was 42% in the EVAR group versus 47% in the OSR group. The 30-day mortality rate was 21% among the patients assigned to EVAR compared to 25 of the patients assigned to OSR in this study. In the IMPROVE (Immediate Management of the Patient with Rupture: Open Versus Endovascular repair) trial, 613 patients were prospectively recruited [25]. The 30-day mortality rate was 35.4% (112/316) in the EVAR group and 37.4% (111/297) in the OSR group. The 30-day mortality rate among patients with confirmed ruptures was 36.4% (100/275) in the EVAR group and 40.6% (106/261) in the OSR group. Although these trials had some limitations due to the relatively small number of patients who were recruited, no significant differences were found between EVAR and OSR regarding either the mortality rate or the complication rate. A systematic review and meta-analysis from van Beek et al. including a total of 3769 articles, 3 RCTs, 21 observational studies, and 8 administrative registries estimate the short-term (combined 30-day or in-hospital) survival after EVAR and OR for patients with rRAAA. The results indicate that EVAR is not inferior to OR in patients with a ruptured abdominal aortic aneurysm with regard to short-term survival. This supports the use of EVAR in suitable patients and OR as reasonable alternative [3]. The debate on the trial results is still open. We can say that in the presence of good anatomical features, endovascular expertise and availability of materials EVAR could be an alternative to open repair for rRAAA. For rAAA endovascular treatment, the institution must have a defined program for emergency endovascular surgery with rapid availability of high-quality computed tomography, availability of trained support staff, stock of available endovascular prostheses in a wide range of sizes, and available vascular surgeon appropriately trained in advanced endovascular techniques [26–29].

### Technical Notes

A preoperative contrast-enhanced CT scan is almost mandatory. The identification of appropriate proximal and distal sealing zones, as well as evaluation of the iliofemoral access, is critical to success.
In case of unsuitability of iliac access (for low diameters, calcification, or angulations) adjunctive procedures, low-profile delivery systems, or a uni-iliac device have to be considered. If it’s possible and available, a bifurcated graft is used to treat the ruptured aneurysm (Fig. 36.3).

This avoids crossover femoral-femoral bypass that usually requires general anesthesia and has a much higher risk of postoperative groin infections. Otherwise, an aorto-uniliac implant and femorofemoral bypass can be considered in the situation of hemodynamically unstable patients or when it is difficult to access the contralateral iliac artery due to stenosis, occlusion, or tortuosity (Fig. 36.4).

No studies have thus far have shown any one device to be superior in treating ruptured abdominal aortic aneurysms, and in fact, most devices have been shown to work sufficiently well. The important thing is to use a device that one is very familiar with [30]. Most centers perform r-EVAR under local anesthesia. It is very quick, and it does not affect patient hemodynamics, but patient discomfort and unrest may compromise the procedure and the precise

**Key Points**

In rupture abdominal aortic aneurysms

- In case of endovascular treatment, choose a device very familiar to surgeons
placement of the endograft. The use of percutaneous access is probably best reserved for patients with contained ruptures that are stable.

The use of heparin for anticoagulation during r-EVAR is debatable. Depending on the clinical status of the patient and the expected complexity of the planned repair, anticoagulation must be administered using careful judgment. Close communication with the anesthesiologist and monitoring of the activated clotting time are essential in all cases to individualize heparin use [31–34]. An aortic occlusion balloon is generally used only when severe hemodynamic instability occurs. In case of patient instability, a large balloon for aortic occlusion is positioned (Coda, Cook Medical; Reliant, Medtronic). Balloon inflation should only be carried out when truly necessary (approximately 19–27% of patients) because the use of an occlusion balloon is not without risk and “trash embolization” and aortic rupture from the balloon can occur. The transfemoral approach is the recommended access route for the aortic occlusion catheter. Initially, an axillary approach was suggested because of downward displacement in the aneurysm sac during femoral insertion. Actually, the contralateral femoral access serves as the access route for a sized sheath long enough to support an inflated balloon. Angiography is performed via the sheath, and deployment of the stent graft’s main body and ipsilateral leg can be done with the occlusion balloon inflated. Via the ipsilateral side, a new balloon is inflated inside the main body, and the primary suprarenal balloon may now be deflated and withdrawn via the sheath, thereby maintaining circulatory control and also restoring circulation to the visceral branches. Staged declamping upon completion of the procedure is good practice [35].

Severe angulation; short, conical, or wide aortic neck; and thrombosis at the landing zone are all factors that might prevent standard EVAR of an AAA. By using fenestrated or branched stent grafts, sealing can be accomplished at the suprarenal, supramesenteric, or supraceliac levels. But most of these systems are custom-made devices, which limits their use in practice to elective cases. In an acute setting, other techniques are reported.

A chimney or snorkel configuration requires antegrade catheterization from a transbrachial approach to facilitate placement of a covered stent into one or more branch vessels in a parallel course, adjacent to the main intra-aortic stent graft (Fig. 36.5).

The proximal portion of the snorkel stent extends above the proximal edge of the main aortic stent graft, thereby extending the proximal seal zone in a short or no-neck aortic aneurysm. Lachat et al. [36] have described the lift technique as an alternative to the chimney technique. Through a femoral access, followed by an 8-F sheath, the target renal vessel is cannulated, and a chimney graft is advanced 1–2 cm into the renal artery and deployed such that its proximal end faces downward. The distal end is fixed in place with an inflated angioplasty balloon. A stiff guidewire is inserted coaxially through the 8-F sheath, the guidewire is removed from the renal artery, and the 8-F sheath is carefully pushed over the stiff guidewire, lifting the chimney graft upward. With the chimney reoriented cranially, the aortic stent graft is immediately deployed. Use of physician-modified endovascular grafts have been described as a safe and effective alternative for treating patients with juxtarenal aneurysms who have no other alternatives for repair, as well as in ruptured aneurysms. Physician-modified endovascular grafts are performed under sterile conditions and the time required for device manufacture ranges from 30 to 80 min, depending on the number of fenestrations. The system is resheathed, depending on what system has been used [37–39].

Some authors report treatment of rAAA with the Nellix endovascular aneurysm sealing (EVAS) system (Endologix, Irvine, CA, USA). It consists of two identical catheter-based devices with a 10-mm flow lumen being created by two balloon-expandable polytetrafluoroethylene-covered cobalt-chromium stents. The stents are mounted on balloons for deployment and are surrounded by polyurethane endobags. The EVAS device fixes the two stents within the aneurysm sac using the endobags, which are filled with a polyethylene glycol (PEG)-based hydrogel that conforms to the aneurysm flow lumen and solidifies within minutes of delivery, providing fixation and seal at the aortic neck and iliac arteries. Potential advantages of EVAS are related to the possibility to treat a wider range of aortic morphology with a small inventory of devices. Preoperative sizing involving the selection of only a suitable length of the device and the avoidance of contralateral limb cannulation in EVAS may reduce the overall planning and
operative procedure duration in certain cases that are very important in the emergency setting. EVAS also provides an option for early hemostasis through endobag inflation with saline while maintaining limb perfusion. A final advantage is the avoidance of type II endoleaks with aneurysm sac sealing.

The potential disadvantages of the EVAS technique for ruptured aneurysm repair include a risk of enlarging a rupture or tearing the aorta with the pressure used to fill the endobags. In addition, there is the potential for failure to seal a ruptured aneurysm due to an inability to generate the required pressure within the ruptured aneurysm sac. The IFU for the Nellix devices specify that they are not suitable for patients with small or large common iliac arteries (<8 or >35 mm, respectively) or those with a large patent flow lumen (>60 mm) within the aneurysm sac.

During EVAR for rAAA, in the presence of inadequate distal landing zone in the common iliac artery, an extension to the external iliac artery is needed.

Preoperative or intraoperative embolization or plug positioning can also be used during EVAR for rAAAs (Fig. 36.6). In select cases, an iliac branch device can be used to preserve internal iliac artery flow.

Abdominal compartment syndrome (ACS) is the major cause of morbidity and mortality after a successful EVAR performed to treat rAAA. Persistent bleeding from the lumbar and inferior mesenteric arteries into the ruptured aneurysm sac in the situation of severe coagulopathy might also contribute to the development of ACS. As reported for open surgery after rAAA recognition and immediate treatment of compartment syndrome is mandatory.

### 36.1.7 Rupture After EVAR

Late failure of endovascular repair secondary to endoleaks, endotension, and sac enlargement, stent graft migration, tear and fracture, or infection continues to be a persistent problem that can result in delayed aneurysm rupture (Fig. 36.7) [40, 41]. Reported rates of abdominal aortic aneurysm (AAA) rupture after EVAR ranging from 0.5 to 1.2% per patient per year [42]. About aortic anatomical features before intervention, larger aneurysm diameter seems to be associated with an increased risk of graft rupture. Not only is the diameter the primary determinant of the risk of primary aneurysm rupture but also a strong predictor of late ruptures, type I endoleak, and aneurysm-related death [43].

Therapeutic approach to post-EVAR rAAA is dependent on the anatomic characteristics and the lesion. While it is ideal to remove all the endograft to eliminate any possible recurrent problems, complete excision is not an easy task. The presence of barbs or hooks, suprarenal fixation stents, or Palmaz stents that are well incorporated into the aorta and inflammatory changes around the aorta and vena cava, left renal vein, and iliac veins may complicate the matter, elevating the complexity of the operation. Complete excision of suprarenal fixation endograft may leave the aortic or arterial wall too thin and denuded, increasing the risk of anastomotic tear. In such cases, partial resection of endograft may significantly reduce the risk of renovisceral ischemia time and adverse outcomes. The endograft remnant can be incorporated in the suture line along with the aortic wall, and it is sewn to the surgical graft. When a total excision of endograft is required, a supraceliac clamping is also required. The reported operative mortality rates for post-EVAR rAAA range widely from 15 to 67%. With the progressive improvement of endovascular technology, its widespread use and long-term follow up, post-EVAR rAAA is
expected to increase in frequency and may lead to more complex endovascular repairs requiring suprarenal fixation or fenestrated endografts. The vascular surgeon should be well equipped to manage this highly lethal condition [43–47].

Nonocclusive mesenteric ischemia (NOMI) is a clinical status, typical of patients with low cardiac output or sepsis, frequently associated with the use of vasoactive/inotropic drugs (often multiple-drug therapies) [50, 51].

36.2 Acute Intestinal Ischemia

Acute intestinal ischemia is “a syndrome caused by inadequate blood flow through the mesenteric vessels, resulting in ischemia and eventual gangrene of the bowel wall. Although relatively rare, it is a potentially life-threatening condition” [48]: in fact, actually contemporary reviews show a high mortality, between 60 and 80% [49].

Acute mesenteric ischemia (AMI) can be caused by arterial or venous occlusion; the arterial thrombosis is divided in acute or chronic, in dependence of their onset.

Among acute mesenteric ischemia, we have different etiologies: SMA thromboembolism and mesenteric hypoperfusion syndromes (the most important are nonocclusive mesenteric ischemia and aortic dissection with mesenteric hypoperfusion) [50] (Fig. 36.8).

36.2.1 Epidemiology and Risk Factors

36.2.1.1 Thrombotic and Embolic AMI

Nowadays population-based studies are not complete because autopsies are rarely performed; actual data refer just to operated patients [50].

AMI frequency was reported to be 17.7% among patients requiring emergency laparotomy and 31% among non-traumatic patients who needed damage-control surgery [52].

Between 1972 and 1982, an epidemiological study conducted with a high autopsy rate (87%) in Sweden (Malmö population) estimated an overall incidence rate of AMI of 12.9% (95% confidence interval, 11.6–14.1) [53].

Figure 36.9 shows the different etiology distribution reported in that study [53].

Today we know that AMI is uncommon, 1–2 per 1000 hospital admissions; it seems to affect especially females (female/male ratio: 3/1), but adjusting for age and gender, we can get similar data.

Patients with AMI are typically between 60 and 70 years old, with some medical comorbidities [49, 54].

Two-thirds of patients with AMI present an acute SMA occlusion, whereas nonocclusive mesenteric ischemia and mesenteric venous thrombosis are less common [52].

Key Points

- Ruptured aneurysms require prompt diagnosis and treatment.
- If the patient is sufficiently stable, a CT scan should be performed.
- Aortic clamping is crucial in either possible surgical treatment, open repair, or EVAR.
emboli are considered clinical risk factors for embolic AMI [49, 54].

A history of postprandial abdominal pain, loss of weight, and food intolerance have to make us suspect for an acute thrombotic AMI [49].

Acosta et al. [55] realized a study based on 213 autopsies with cases of SMA occlusion, and they find a ratio embolus/thrombus of 1 [52, 56].

They identified an embolic origin in 80% of patients: the most common findings were atrial fibrillation and synchronous embolism, with embolic dislodgement to renal, splenic, common hepatic, and celiac trunk arteries in 41% of patients.

Thrombotic lesions were frequently associated with old cerebral infarction, aortic wall thrombosis, and disseminated cancer, occlusion, or severe celiac trunk stenosis (in 33% of patients) [55].

Seventy-three percent of patients with SMA thrombotic occlusion had previously shown symptoms of chronic mesenteric ischemia [55].

They also observed that thrombotic occlusions were more proximal than the embolic ones, with larger intestinal infarction [55].

36.2.1.2 Hypoperfusion Syndromes: NOMI

Being clinically not always well defined or easily recognized, NOMI’s prevalence isn’t clear, even if mixed populations with bowel gangrene show a percentage between 4 and 60%.

Malmö population study defined “NOMI” all those clinical cases where there were no signs of emboli and absence of mesenteric arteries thrombosis, dissection or significant stenosis.

NOMI’s incidence was 2/100,000 person-years (in spite of 8.6 for thromboembolic etiologies and 1.8 for MVT); they also reported that NOMI was as fatal as MVT and one-fifth deadly as SMA occlusion.

It’s reasonable to think that pharmacological improvements have to lead to a decrease of NOMI, but the rising number of cardiac pump devices has created a new source of NOMI [50, 51].

Another common finding between different experiences is the difficulty to divide NOMI from SMA thromboembolic occlusion without angiography, TC or autopic exam, because they have common risk factors, such as fatal cardiac failure, recent atrial fibrillation, or surgery.

Most of the patients (often the oldest ones) with NOMI also had SMA (40%) or celiac trunk concomitant stenosis.

About one-fifth presented synchronous infarction in the liver, spleen, or kidney [51, 53].

Mitsuyoshi et al. [57] defined four criteria for early diagnosis of NOMI (Table 36.2): a patient with three of them after cardiovascular surgery or renal replacement therapy is at risk of NOMI; we must require an urgent CT to get an early diagnosis [51].

36.2.1.3 Mesenteric Venous Thrombosis (MVT)

MVT in Malmö study and the most recent ones was 2.7/100,000 person-years, equally affecting males and females, especially from 70 to 79 years, with a mortality rate of 20%.

Risk factors for MVT are divided into three groups: direct injury, local venous congestion or stasis, thrombophilia (Table 36.2) [53, 58].

Table 36.2 Mitsuyoshi et al. criteria for early detection of NOMI

| Symptoms of ileus with slow appearance and an increasing trend |
| Need of catecholamine administration |
| An episode of hypotension |
| Slow increase of transaminase level (LDH included) |

Table 36.3 Risk factors for Mesenteric Venous Thrombosis

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<th>Direct injury</th>
<th>Local venous congestion/stasis</th>
<th>Thrombophilia</th>
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<td>Postsurgical trauma</td>
<td>Portal hypertension/liver cirrhosis</td>
<td>Activated protein C resistance</td>
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<td>Pancreatitis</td>
<td>Heart failure (EF &lt; 20%)</td>
<td>Protein S deficiency</td>
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<td>Obesity (BMI &gt; 40%)</td>
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<td>Metastatic cancer</td>
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<td>Oral contraceptive use/estrogen substitution</td>
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<td>Extramesenteric previous or synchronous VTE</td>
<td></td>
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</table>
In these patients, risk factors for early death are intestinal infarction, advanced cancer, pulmonary embolism and hepatorenal failure, elevated age of patient, conditions like malignancy or other comorbidities, and a delay in diagnosis [53, 58].

### 36.2.2 Diagnosis

The typical AMI clinical trial is characterized, in an elderly patient, by severe abdominal pain without pathological abdominal signs (pain “out of proportion”), bowel emptying, and a source of embolia [50, 52].

These classic symptoms can be absent in 20–25% of cases [52].

Frequently SMA occlusion and MVT are very difficult to be recognized promptly.

There are slight clinical differences among embolic SMA, thrombotic SMA, and MVT, as summarized in Table 36.4.

Independently from AMI etiology, laboratory findings are frequently hemoconcentration, leukocytosis, high anion gap, and lactic acidosis in advanced cases. We can also find elevation of amylase, aspartate aminotransferase, and lactate. All these blood tests are insensitive and nonspecific for AMI [49, 54].

### Table 36.4 Clinical different presentation of embolic SMA, thrombotic SMA, and MVT

<table>
<thead>
<tr>
<th></th>
<th>Embolic SMA</th>
<th>Thrombotic SMA</th>
<th>MVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal pain</td>
<td>Sudden onset</td>
<td>Insidious onset</td>
<td>Insidious onset</td>
</tr>
<tr>
<td>Vomiting</td>
<td>+ (71%)</td>
<td>+/-</td>
<td>+/-</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>+ (42%)</td>
<td>y</td>
<td></td>
</tr>
<tr>
<td>Bloody stools</td>
<td>+</td>
<td>+/-</td>
<td>+/-</td>
</tr>
<tr>
<td>Female genre</td>
<td>++</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Age (years old)</td>
<td>&gt;80</td>
<td>&gt;80</td>
<td>&lt;50</td>
</tr>
<tr>
<td>Anamnesis of atrial fibrillation</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anamnesis of myocardial infarction</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous artery embolism</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous deep vein thrombosis or pulmonary emboli</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Still-unknown source of emboli</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td>+/-</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>Synchronous embolism</td>
<td>++</td>
<td></td>
<td></td>
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<tr>
<td>Previous chronic mesenteric ischemia</td>
<td>++</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancreatic or hepatic pathologies</td>
<td>+</td>
<td></td>
<td></td>
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<tr>
<td>Coagulation deficit</td>
<td>+</td>
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</tbody>
</table>

### 36.2.3 Imaging

About 25% of patients are completely normal at plain x-rays. In advanced cases of AMI, we can see bowel wall edema (“thumb printing”) or pneumatosis; x-ray helps to exclude other etiologies of abdominal pain such as bowel obstruction or perforation.

Duplex ultrasonography in the abdominal district can evidence high-grade stenosis of the celiac trunk and SMA, but it’s not widespread because it’s highly operator-dependent and can’t be limited by intestinal gas, typical of nonfasted patients. Then duplex can reach just the proximal part of SMA, but emboli go further, in its distal segments, so this creates the risk of false negative results [49].

Contrast-enhanced computed tomography (CT) represents the examination of choice and has replaced angiography to get the diagnosis [49, 54].

In fact CT allows bowel evaluation and finding AMI etiology (embolic occlusion seems like an oval clot surrounded by contrast in a segment of artery located in the middle and distal part of the main trunk of SMA without significant atheromasic signs, thrombotic lesions present a clot overlying on an atheromasic occlusive lesion at the ostium of SMA) [49, 52].

Otherwise, preoperative CT scan can be used in an endovascular setting to assess the level and orientation of vessels (Fig. 36.10).

Angiography today is important especially for allowing many complementary or stand-alone treatments such as injection of intra-arterial vasodilators, thrombolysis, and angioplasty with or without stenting [49].

Less useful are other diagnostic exams like MRI or laparoscopy; in fact, the first has a CT-inferior resolution, limited to 1 mm (insufficient to see very distal emboli), overestimates the degree of stenosis, and requires significant post-processing. Moreover, the secondary signs of AMI (indurated fat, bowel wall thickening, etc.) are more difficult to recognize with MRI.

Laparoscopy is quite limited to assess intestinal viability because segmental ischemia can’t be seen because of a difficult bowel navigation [49].

### 36.2.4 Treatment

#### 36.2.4.1 Medical Treatment

Patients with AMI should be treated immediately with an isotonic crystalloid solution, and then, if necessary, invasive monitoring should be started, measuring hourly urine output, continuous central pressure and arterial pressure. A broad-spectrum antibiotic therapy has to be started together with intravenous heparin administration (doubling normal PTT), if not contraindicated [49].
If these patients develop sepsis and organ dysfunction, they are managed with the awareness that vasopressors can worsen bowel ischemia and visceral vasospasm, so before their administration, we have to verify adequate right heart filling pressures. These patients have a high percentage of fluid sequestration, so it’s mandatory to perform serial examination and bladder pressure monitoring to recognize an eventual abdominal compartment syndrome. Dobutamine, lower-dose dopamine, and less epinephrine are considered vasopressors of choice; pure alpha-adrenergic agents shouldn’t be used [49].

In MVT anticoagulant therapy is administered to stop the extension of thrombosis, and fibrinolytic drugs try to restore the patency of the affected venous segment [57].

So heparinization starts at the diagnosis, using unfractionated or low molecular weight heparin in therapeutic doses [58]. Literature reports that anticoagulant therapy increases recanalization up to 80% and recommends to consider lifelong treatment in case of prothrombotic risk factors.

It’s not well-known how long the patient must be anticoagulated, but in literature, many authors report at least 6 months; after that period, every decision will be tailored to the patient features, considering the MVT etiology.

Risk of bleeding is contained between 1 and 4%; a problem linked to anticoagulation is the risk of intestinal bleeding in case of mucosa necrosis.

A stand-alone medical treatment in MVT is successful in more than 90% of cases [58].

### 36.2.4.2 Surgical Treatment

Ideally, AMI treatment should require open or endovascular surgery or both.

After clinical and CT evaluation, it’s essential to exclude peritonitis and understand the etiology of occlusion (embolic or thrombotic); in case of peritonitis, laparotomy is mandatory.

It’s necessary to perform revascularization before sectioning any tract of the bowel, with exception made for cases of intestinal perforation, areas of frank necrosis, or peritoneal soilage, which requires resection of the affected bowel without reanastomosis [49, 52].

Bowel revascularization can be achieved with endovascular, open, or hybrid treatment.

### Open Surgery

Open revascularization, through midline laparotomy, can be obtained by surgical embolectomy, endarterectomy, patch angioplasty, or bypass grafting, with final assessment of bowel viability [49].

#### Key Points

In case of SMA bypass

- Perform a “lazy-C” bypass to avoid aortic clamping and prevent kinking.
- The more the length is, the more the graft has a good lie.
- Perform end-to-end anastomosis to improve graft patency.
- Choose vein graft in case of peritoneal contamination.

With a laparotomic access, it is possible to remove with a Fogarty catheter the thrombotic material even from the mesenteric vein, which runs parallel and right to its artery at the lower border of the pancreas, easily reached after lifting up the transverse colon. Laparotomy also allows to perform a bowel resection if required. In case of venous ischemia, it can be more difficult to distinguish the line between vital and ischemic tissue, as the border can be more diffuse than in the arterial ischemic case. In case of doubt or peritoneal contamination, it’s better to exteriorize the bowel ends and perform an anastomosis in a second time.
So a second look surgery could be reasonable, with good results also reported for laparoscopic second looks [49].

Patient has to be prepared and draped including both legs (at least until the knee) in case harvesting great saphenous vein or superficial femoral vein should be required [49].

A vertical midline incision is performed; an anterior or a more lateral surgical approach allows to expose SMA, depending on the etiology of AMI; the first is best for embolic etiologies or patent SMA, and the latter is useful for thrombotic occlusion and/or SMA disease [49].

**SMA Embolectomy**

SMA embolectomy requires exposition of the anterior wall of the vessel, reached elevating the omentum and transverse colon and mobilizing the small bowel to the right.

The peritoneum is sectioned with a horizontal incision at the base of the transverse mesocolon (without mobilizing the fourth portion of duodenum or Treitz ligament).

During dissection of the superior mesenteric vein (to the right of SMA), autonomic nerve fibers and small lymphatics are exposed, and they are sacrificed to expose SMA. To see the proximal segment of SMA, even the inferior pancreatic border can be carefully mobilized, preserving the splenic vein and its tributaries from the pancreas, too.

So SMA and all the branches included are isolated from the middle to the right colic branches; after systemic heparinization a transversal or longitudinal arterectomy is performed (the latter will require a patch angioplasty).

Ideally it would be better to extract thrombus just venting the proximal tract of SMA, but, if it’s necessary, an embolectomy balloon catheter can be also used (the most employed are 3 Fr or 4 Fr); distal segment can be treated with a 2 or 3 Fr catheter of the same kind. Sometimes passing through small distal multiple branches can be difficult; in alternative or adjunct, the surgeon can place a hand on either side of the mesentery and “milk” embolic material out of the arteries.

After complete thrombus removal, we can suture the arteriotomy as previously described [49].

**SMA Bypass**

In this case, a major tract of the artery is exposed, using a lateral access to SMA, passing above the fourth portion of the duodenum and opening the peritoneum lateral to the duodenum itself, along with the aorta and the left or right common iliac artery.

Graft orientation depends on the features of the inflow vessels (and their degree of atheromasic lesions) and the overall lie of the graft.

One of the most used graft orientations is the “lazy-C”; it’s a retrograde configuration, with a graft that originates from the right iliac artery and allows to avoid any aortic clamping and prevent kinking (Fig. 36.11).

It’s also possible to perform lazy-C grafts originating from the left iliac artery or the distal infrarenal aorta. The more the bypass length is, the more it has a good lie; graft patency results in better performing end-to-end graft to SMA anastomosis.

Anterograde bypass has to be considered if distal inflow sources are not ideal, because of their high degree of atheromasic disease, while the supraceliac aorta is generally relatively free of atheromasic lesion; another advantage is that this graft configuration is not easily prone to kinking.

Disadvantages are that supraceliac aorta is more complex to be isolated, so it requires a great technical effort, spending more time, and if partial clamping isn’t possible, with aortic cross clamping, a hemodynamic and physiological stress is produced [49].

Bypass is generally performed with prosthetic grafts of 6–8 mm in Dacron or PTFE; there’s no evidence that synthetic bypasses have a better patency than biological ones.

The degree of abdominal contamination leads to choice of graft; in particular, in case of significant peritoneal soilage, the use of vein is preferred; primary options are great saphenous vein or femoral veins. Veins require an additional effort for their harvesting that can be dangerous for critically ill patients; they also show a more elevated risk of kinking and external compression, because of their smaller diameters and the same features of their venous wall.

If there’s no alternative to synthetic grafts, Kazmer’s technique can be performed, consisting in covering a retrograde prosthetic bypass with an omental flap passed through the transverse mesocolon.

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![Fig. 36.11 The “lazy-C” bypass from SMA to the right common iliac artery](image-url)
Hybrid Approach: Retrograde Open Mesenteric Stent (ROMS)

It’s a hybrid technique proposed by Wyers et al. that consists in opening horizontally or longitudinally the peritoneum at the base of the transverse colon; SMA is isolated, and a local thromboendarterectomy is performed, and then the artery is closed with a patch (in vein or bovine pericardium) angioplasty that helps to easily realize a retrograde cannulation of SMA with a long flexible sheath to the aorta. In the end, SMA is stented to reduce the number of restenoses [49, 59].

This technique allows to reach a good result in SMA revascularization in a faster and easier way, exploring bowel viability at the same time [49].

36.2.4.3 Endovascular Techniques in Acute AMI

Endovascular treatment of AMI has widespread extensive use of multidetector CT angiography and the great progress in endovascular techniques reached in the last two decades [59].

Key Points

In endovascular treatment of AMI

• Move cautiously catheters to avoid dislodgment of proximal clot.
• After stenting, measurement of residual pressure gradient across the stent allows to avoid early restenosis.

The most used is a percutaneous femoral access, while a brachial one is used just when SMA takeoff is very sharp or when a proximal stenotic lesion is the origin of occlusion (in this case brachial approach gives us a better angle of access and less risk of dissection).

To get a good imaging, a 5 Fr pigtail catheter is generally preferred, positioned at T12 level. Generally Omnipaque 300 g/mL or Visipaque 270 mg/mL is used, but in patients with impaired renal function, we can choose CO2 at the base of the transverse colon; SMA is isolated, and a local thromboendarterectomy is performed, and then the artery is closed with a patch (in vein or bovine pericardium) angioplasty that helps to easily realize a retrograde cannulation of SMA with a long flexible sheath to the aorta. In the end, SMA is stented to reduce the number of restenoses [49, 59].

This technique allows to reach a good result in SMA revascularization in a faster and easier way, exploring bowel viability at the same time [49].

Aspiration embolectomy: If imaging shows a proximal SMA, embolus an antegrade approach is the first choice: the SMA will be catheterized with a reverse curve catheter and a hydrophilic 0.035-inch guidewire that has to be changed with a stiffer one. Sometimes it is possible to exchange directly the stiffer wire on the reverse curve catheter.

When the wire has been positioned, an introducer with the removable hub (a 7 Fr 45 cm or 8–9 Fr guiding catheter) can be left proximal to the embolus. Inside this, a 4–6 Fr catheter is introduced and aspiration with a manual 20-mL syringe starts, together with sheath withdrawal. In this way, the clot removal can be performed.

Many passages of the aspiration sheath in a to-and-from motion are required to achieve complete clot aspiration; for every passage of the wire out of the femoral sheath, it’s necessary to remove its hub to be sure that there’s no residual embolic material in the introducer. It’s also possible to use a double-lumen aspiration catheter, with one channel for the wire and the other for aspiration; they are useful for difficult access, while their limit is that they remove small amounts of clots compared with large lumen sheets alone [59].

SMA Thrombolysis

Thrombolysis is rarely used alone to treat AMI because it usually needs a long time to clear the obstruction and in case of bowel necrotic disease it could cause severe gastrointestinal bleeding [58].

In literature, there are many combined treatments of thrombolysis with other percutaneous thrombectomy devices [58].

For instance, frequently thrombolysis is performed together with mechanical methods of clot removal, to restore vessel patency rapidly; in fact it is useful to complete the effect of a mechanical removal of thrombotic material in residual or very distal clots.

Thrombolysis increases the risk of gastrointestinal bleeding, and it’s contraindicated when laparotomy is necessary; so this treatment is reserved just for those patients with low suspicion of transmural intestinal infarction [59].

In MVT it can be preferably administered as an intrathrombotic treatment using a transhepatic portography to place the catheter in the thrombus; in this way the risk of bleeding and the administered thrombolytic dose can be reduced; in alternative SMA it can be used to administer the drug.

In particular, the aspiration sheath can be left in the proximal SMA (catheter embolectomy has been realized previously) and placed a multi-side hole infusion catheter in the SMA, performing a local thrombolysis with rTPA at 0.5–1 mg/h rate. After 12 h the angiographic result can be controlled, and it can be decided to stop or continue the treatment [59].

Stenting

Stenting of visceral arteries takes place in case of proximal stenosis or occlusion; percutaneous transluminal angioplasty
(PTA) alone, for its low patency rate and its risk of dissection, is not recommended.

At first passing across the lesion is necessary; it can’t be easy with a femoral access; in this case, a brachial access can be used with a 4 Fr Headhunter catheter. The following step is introducing a 1.5 mm balloon on a Simmons catheter and dilating the lesion after aspiration and/or thrombolysis.

We have to choose the right balloon for the right lesion because the different features of the devices make them more or less adequate to a different degree of calcified lesions (e.g., Teflon-coated balloons are useful for lesions with difficult access, because of their slipperiness).

It’s wise to stent an occlusive lesion with a balloon-expandable stent, stronger than self-expandable nitinol stents, very useful for ostial stenosis and avoiding post-dilatation recoil. For SMA and celiac trunk, stents of 7–9 mm diameters are a good choice, while smallest devices can compromise long-term patency. After stenting, angiography and pressure measurement are performed: a residual pressure gradient across the stent of more than 12 mmHg requires additional PTA and/or stenting to avoid restenosis (Fig. 36.12) [59].

36.2.4.4 Assessment of Bowel Viability
After revascularization it’s necessary to assess the extent and severity of intestinal ischemia; before any decision, it’s necessary to wait at least 20–30 min of reperfusion [49].

Valuating bowel viability is not easy; in fact, intestinal segments prone to irreversible necrosis can appear quite normal, and segments bound to recover after revascularization can seem ischemic [49].

Laparotomy allows to see bowel condition, and it’s the first choice method because it’s safer and quicker than the same laparoscopy, which is limited in evaluating extensive intestinal paralysis with distended bowel loops.

Assessment of bowel viability is based mainly on clinical judgment and timely reevaluation, with the aim to save the greatest length of intestinal loops as possible [49].

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Fig. 36.12 Patient with AMI: at angiography significant AMS stenosis (a), stented with a balloon-expandable stent 6 × 18 mm, without reaching the nominal diameter (b). Residual pressure gradient across the stent is <12 mmHg; procedure is ended with clinical success (AMI resolution)
The surgeon has to remind that at first-look surgery it is difficult to determine bowel viability with certainty; at the end of the first operation, he has to decide if a second look is required to assess newly bowel viability and extend intestinal resection only if it’s strictly necessary. So he typically will leave bowel transected ends stapled shut without reanastomosis [49]. Rarely it’s advisable to perform a third look, before final bowel anastomosis is realized [49].

Bowel loops are evaluated looking for color and aspect of the bowel serosa, dilatation of intestinal loop, peristalsis, bleeding from cut surfaces, and visible or palpable pulsations in the mesenteric arcade [49, 50].

A continuous wave 9–10 MHz Doppler can be useful to detect nonviable segments.

Another method uses fluorescein and a perfusion fluorometer or a laser Doppler flowmeter to get an accurate quantification of a segment perfusion.

After revascularization, splanchnic vasospasm can persist for a variable period, worsening bowel ischemia; to reduce this phenomenon, papaverine can be administered into SMA.

36.3 Splanchnic Aneurysms

Splanchnic aneurysms are intra-abdominal aneurysms of the celiac trunk; gastroduodenal, pancreatic, splenic, superior, or inferior mesenteric artery; and renal arteries and their branches [60].

Key Points

- In VAAs endovascular treatment is important to occlude their collaterals to avoid carefully the aneurysmatic sac. continues to be perfused

36.3.1 Epidemiology

They are rare, often asymptomatic, incidental findings at TC or MRI, but sometimes they can require an emergency treatment in case of rupture [11, 21]. Rupture is documented in about 25% of them, with an elevated mortality rate of 70% [61].

They can involve visceral arteries with different incidence, as shown in Fig. 36.13 [60].

The rising number of endovascular procedure in the liver and kidney today has increased the number of intrahepatic and renal aneurysms [60].

True visceral aneurysms are divided from pseudoaneurysms: the first are dilatation involving all the three layers of the arterial wall; they can be sacciform or fusiform, mainly caused by atherosclerosis, fibrodysplasia, or connective tissue diseases [61].

36.3.2 Etiology

Thirty-two percent of VAAs are due to atherosclerosis, while medial degeneration is responsible for 24% of them, with its reduced number of smooth muscle cells and reduction or fragmentation of fibers. Other etiologies are abdominal and surgical trauma (22%) and infectious or inflammatory diseases (10%). In the pre-antibiotics era, most of VAAs had mycotic and/or inflammatory causes, often in association with endocarditis.

Other causes can be connective tissue diseases, such as Marfan or Ehlers-Danlos syndrome, hereditary hemorrhagic telangiectasia, Osler-Weber-Rendu disease, Kawasaki syndrome, and fibromuscular dysplasia.

Some systemic diseases can also give diffuse vascular anomalies and multiple aneurysms, such as Still disease, systemic LES, Hashimoto thyroiditis, and polyarteritis nodosa.

Finally, pregnancy and portal hypertension are considered risk factors for splenic and hepatic aneurysms [60, 62, 63].

Pseudoaneurysms can result from trauma, inflammation, infection, and vasculitis, or they can be iatrogenic. Moreover, pancreatitis, with the release of pancreatic enzymes, may produce destruction of the arterial wall,
resulting in pseudoaneurysms of the splenic, hepatic, gastroduodenal, and pancreaticoduodenal arteries [60].

Visceral aneurysm rupture is a life-threatening condition, with an elevated mortality rate of up to 30% in emergency; rupture can be associated with intra- or retroperitoneal bleeding, gastrointestinal bleeding, or bleeding into adjacent organs. An important number of ruptures occur after other medical procedures [16].

Many authors considered a diameter superior to 2 cm or three times greater than the respective normal artery as mandatory for the treatment of any visceral aneurysm, but different experiences show that the risk of rupture doesn’t depend only on aneurysm size but also from its localization, rate of growth, and underlying pathology. Some centers also consider their morphological aspect, believing that wall calcification indicates a long-standing lesion and can be a sign of aneurysmal stability. Instead a signal of impending rupture is rapid size increase [60, 62].

### 36.3.3 Diagnosis and Imaging

Computed tomography is the first-line imaging modality for patients with abdominal pain in the emergency department. All patients with a suspect of impending rupture of VAA should undergo to CT scan imaging [60].

### 36.3.4 Treatment

VAA can be treated with open or endovascular approaches. Management depends mainly on the location of the aneurysm, with an endovascular approach that is the first choice in aneurysms involving the parenchymal branches of the hepatic, splenic, renal, or pancreaticoduodenal arteries [60].

Open and endovascular treatments of VAA share the common goal of preventing aneurysm expansion and rupture or repairing the ruptured ones. This purpose is best accomplished by excluding the aneurysm from the arterial circulation and pressure. In case of adequate collateral flow, surgical management consists of ligation of the parent artery proximal and distal to the aneurysm. Otherwise, bypasses or grafts should be required [60].

Endovascular treatment can lead to isolation of the aneurysm from the arterial circulation: this goal can be achieved through different techniques.

**Aneurysmatic large arteries** can be “trapped” between coils placed first distally and then proximally to the aneurysm (Fig. 36.14a). For smaller aneurysms the distal artery or its branches can be occluded with large particles, placing also coils in the larger proximal part of the parent artery. In this way, the aneurysmatic lesion will be excluded from the circulation. We have to pay attention to occlude also other collaterals that could continue to perfuse the aneurysmatic sac (Fig. 36.14b) [60].

Coils are useful also for **saccular aneurysms with a narrow neck**: these devices can be used to fill the sac, constrained by its same neck; in case of **wider neck**, the lesion can be treated using a stent-assisted coil placement.

This technique consists of positioning a stent across the aneurysm neck (Fig. 36.14c) and then filling the sac with coils introduced through the interstices of the stent, which in turn avoid coils migration [60].

Glue or thrombin can be added to fill the sac into.

Thrombin injection can also be chosen to treat VAA: under CT or ultrasonographic guidance, a 20–22 gauge needle is introduced into the sac, and thrombin is injected until the aneurysmatic or **pseudoaneurysmatic lesion** has no more flow. This method is frequently used for small peripheral traumatic pseudoaneurysms of the liver, spleen, or kidney or for treating pancreaticoduodenal aneurysms [60].

For those VAA where **preserving arterial perfusion** is necessary, we can use covered stents, useful for arteries of at
least 6 mm of diameter; their use is limited by size and stiffness of their delivery systems, which make them not so advisable to be placed in distal tortuous vessels [60].

36.3.5 Follow-Up

Independently of treatment a careful postprocedural surveillance is necessary to exclude any enlargement or reperfusion of the aneurysmatic sac from collateral branches; a good follow-up can be realized with ultrasound and CT scan, performed at 1, 3, 6, 9, and 12 months [60].

36.3.6 Splenic Artery Aneurysms (SAAs)

36.3.6.1 Epidemiology
SAAs represent the 60% of VAAs, their real prevalence is difficult to establish because they are mainly asymptomatic. Autopic findings suggest a prevalence from 0.1 to 10.4% [62, 64, 65].

Concomitant visceral aneurysms have been detected in 3% of patients, while non-visceral aneurysmatic lesions have been found in 14% of patients (this percentage includes renal artery dilatations) [66].

They are four times more common in women than in men, in particular, in women with multiple pregnancies. In fact, the hormonal changes typical of pregnancy leads to structural weakness of the vessels wall, and portal congestion produces an increase of wall stress. These factors give a combination of medial hyperplasia and fragmentation of the elastic lamina. The same pathophysiology is associated with splenic arteries developed in patients with portal hypertension that counts a prevalence of 10% in lienal aneurysms [60, 62, 64–66].

Other different etiological factors attributed to aneurysm formation include angiodyplasia, pregnancy, atherosclerosis, diabetes, intracranial aneurysm, polyarteritis nodosa, alpha-1-antitrypsin deficiency, and infective factors.

Frequently they are asymptomatic, most of them are identified incidentally, or in case of rupture, this event is associated with a 25% mortality which increases to 75% among pregnant women with fetal mortality of 95%. In pregnant women with splenic aneurysms, two-thirds of them rupture in the third trimester followed by the second trimester; at the time of rupture, most lesions are more than 2.5 cm, but also smaller aneurysms have become symptomatic [67].

36.3.6.2 Clinical Diagnosis
Rupture can be sudden or in two stages, the latter (20–25% of cases) is characterized by an acute abdominal pain, with a brief stable period (from 6 to 96 h) followed by collapse. These clinical features are due to an initial rupture in the lesser sac, contained by omentum and/or blood clots that block foramen of Winslow, and then it is followed by free rupture into greater sac, when the tension within the lesser sac increases.

In 13% of patients, we assist to secondary erosion of the aneurysm into adjacent viscera, causing gastrointestinal bleeding due to rupture into the stomach, colon, or pancreatic duct. Rupture communicating with the splenic vein can lead to an arteriovenous fistula, with portal hypertension, or even mesenteric steal syndrome and ischemia of the small intestine [65–67].

In case of rupture, the patient refers acute abdominal pain in epigastrum or left hypochondrium, sometimes with the Kehr’s sign. Nausea, vomiting, and collapse can be associated. In pregnant women ruptured aneurysmatic lienal artery can be misdiagnosed with placental abruption [65, 67].

The natural history of untreated splenic artery aneurysms remains poorly defined; no consensus on a size criterion for aneurysm treatment in asymptomatic patients has been found. For many years splenic aneurysms greater than 2 cm in low-risk patients must be repaired; this dogma was based mainly on several small retrospective reviews of patients with splenic aneurysm rupture, but today has fallen into disuse [64–66].

Another common practice is that patients with childbearing potential or undergoing liver transplantation need aneurysm repair, independently of their size. Stanley et al. have proposed a reasonable approach that consists in treating elected patients with a mortality less than 0.5%, based on the fact that rupture risk is 2% associated with 25% of operative mortality in emergency [66]. Also enlarging aneurysms and aneurysms 2.5–3 cm or more in diameter may require treatment [67].

36.3.6.3 Diagnosis
In suspect of rupture of splenic aneurysm, CT scan or digital subtraction angiography are indicated, in order to define the exact site of the lesion, its collateral branches and the source of bleeding, and to find out other eventual visceral aneurysms [65].

Surgical treatment can involve open, laparoscopic, or endovascular surgery, in dependence of the presentation, location (many of them are placed in the mid-distal part of the artery), and size of the aneurysm.

It is important to preserve the spleen in order to save immunological function [65]. However, there is evidence that ligature or embolization of lienal artery alters the spleen function, even if it is preserved.

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P. Tracanelli et al.

[1] Pain in the tip of the left shoulder.
Surgical treatment of SAA includes ligation or resection for aneurysms in the proximal or middle segment of the splenic artery. Short gastric arteries and pancreatic arcade provide flow to the distal splenic artery, so bypass surgery is not necessary [65].

Complete or partial splenectomy can be performed in case of a lesion near the hilum or involving intrasplenic branches; sometimes in emergency partial, pancreatectomy can be necessary too. In this case, mortality rate rises to 20% versus 5% in elective surgery, with highest percentages in emergency.

The approach to the splenic artery can be through an anterior or a lateral route; the second has an increased risk of splenic infarction because it can compromise short gastric and left gastroepiploic vessels.

A safe and feasible treatment is the laparoscopic one, in the hand of a well-experienced surgeon with the help of intraoperative ultrasound. Even in this case, the operator can choose between an anterior and a lateral route, with lateral approach that is the first choice for central and distal aneurysms. Laparoscopic technique is contraindicated in unstable patients.

Endovascular trapping is a good alternative to open surgery for asymptomatic or symptomatic aneurysms without signs of frank rupture (Fig. 36.15) [60, 64, 66].

Complications include coils migration and distal infarction, abscess formation, and, rarely, rupture of the aneurysm. Recanalization can occur in up to 12.5% of patients. Embolization can also fail for technical reasons, in particular in case of extreme tortuous splenic artery [65].

After endovascular treatment we can also observe the so-called “postembolization syndrome,” a combination of fevers, abdominal pain, ileus, and pancreatic inflammation, observed in 30–80% of patients, that resolves within 4–5 days after the procedure [64, 66].

Fig. 36.15 Patient with symptomatic splenic aneurysm seen at CT and confirmed by angiography (a); plugs are positioned in the afferent and efferent vessel (b); at last we can see complete exclusion of the aneurysm, with revascularization from gastric artery and immediate perfusion of the superior segment of spleen (c, d)
36.3.7 Hepatic Aneurysms

36.3.7.1 Epidemiology
Hepatic artery aneurysms are the second most common kind of VAAs, with a major prevalence in men (3:2), involved in 66% of cases the extrahepatic tract of the artery, often with a degenerative or dysplastic etiology. Intrahepatic branch aneurysms (3%) instead show a traumatic or iatrogenic history or are associated with infections or vasculitis. Twenty percent of these VAAs can show both intra- and extraparenchymal involvement.

As for the splenic ones, true hepatic artery aneurysms are most commonly due to medial degeneration and secondary atherosclerosis.

They generally affect the patient in their sixth decade of life. Concomitant aneurysms in the visceral or non-visceral arteries are associated, respectively, for 31 and 42% [60, 66].

Rupture has been reported in 20–80% of hepatic aneurysms, with mortality rates from 21 to 35%; that’s why an aggressive approach is required to treat these lesions [60].

Pseudoaneurysms represent about 25% of all the hepatic aneurysm cases described in the literature; they are secondary to blunt or to penetrate traumatic injury or iatrogenic.

They become symptomatic medially 5.7 months after injury, with hemobilia, abdominal pain, and jaundice.

36.3.7.2 Diagnosis
Most of hepatic aneurysms are asymptomatic, incidentally diagnosed at CT scan or angiography, but some of them can become symptomatic: in fact, rapid expansion of the aneurysmatic sac produce severe abdominal or back pain in the patient, and large aneurysm can give an extrahepatic compression of the biliary tree.

Aneurysmatic decompression in the biliary tree is quite frequent, while rupture into the peritoneal sac or manifesting with gastrointestinal bleeding is rarer.

One-third of symptomatic patients present the so-called triad of Quincke, characterized by epigastric pain, hemobilia, and obstructive jaundice.

It’s not clear which risk factors can be associated to rupture, and its same prevalence is difficult to establish. Hypertension is the commonest comorbidity, while fibro-muscular dysplasia or polyarteritis nodosa puts the patient at high risk for hepatic artery aneurysm formation.

These conditions are associated with an increased risk of rupture; we find them in about 50% of ruptured hepatic aneurysms [60, 66].

36.3.7.3 Imaging and Treatment
As for splenic aneurysms, anatomical location of the lesion deeply influences its own surgical treatment. Angiography or CT scan can delineate potential anatomical variants and the exact position of the aneurysmatic sac, evaluating also collateral vessels [66].

When lesion involves the common hepatic artery, the aneurysmatic sac can be ligated or resected theoretically without any reconstruction, because the gastroduodenal and right gastric branches should provide a good perfusion; in literature are reported different experiences that describe central liver necrosis after treating a hepatic proximal lesion.

For this reason, in low-risk patients, arterial reconstruction with autogenous conduit has been suggested, in particular where the gastroduodenal artery is small. Many authors describe the use of intraoperative ultrasound to assess significant changes in hepatic vascularization, before performing a definitive ligation [60, 66].

Surgical treatment of more distal extrahepatic lesion, with the involvement of the proper hepatic or proximal right or left hepatic arteries, requires direct arterial reconstruction with autogenous vein.

Some authors also suggest repairing the aneurysm proceeding from the inside of the sac, in case of strong surrounding inflammation and proximity to the common bile duct [60, 66].

Endovascular management of intrahepatic aneurysms is the treatment of choice: transcatheter coil occlusion or embolization is less invasive than open surgery and allows to avoid hepatic resection.

Ischemic complications are minimal thanks to the collateral blood flow and oxygen delivery coming from the portal vein. We have to remind that patients with large segments of liver at-risk or baseline liver dysfunction are not good candidates for intrahepatic endovascular embolization [60, 66].

Endovascular repair is also for extrahepatic lesions: common hepatic artery aneurysms placed proximal to the origin of the gastroduodenal and right gastric arteries can be embolized by coils with a percutaneous access.

Key Points
In hepatic artery aneurysms

- Perform a temporary occlusion of the vessel; in case of signs of ischemia, reconstruct the artery, especially in low-risk patients with small-diameter gastroduodenal artery.
- In case of strong surrounding inflammation and proximity to the common bile duct, repair the aneurysm proceeding from the inside.
- In patients with large segments of the liver at risk or baseline liver dysfunction, intrahepatic endovascular embolization is contraindicated.

Treatment is the same for aneurysm or pseudoaneurysm; now we’ll indicate both lesions with the term “aneurysm.”
The proper hepatic artery can be treated with covered stent, but only in absence of excessive tortuosity and if we can find an adequate landing zone.

### 36.3.8 Gastroduodenal and Pancreaticoduodenal Aneurysms

Aneurysms and pseudoaneurysms often are complications of acute or chronic pancreatitis and pancreatic surgery or have a dysplastic or degenerative etiology. Mainly asymptomatic, some of them can lead to gastrointestinal or intra- or retroperitoneal bleeding. Their treatment today is mainly endovascular, through the sac embolization and a careful search and occlusion of all their collateral vessels.

### 36.3.9 Celiac Trunk Aneurysms

They represent 4% of all VAAs, often with a degenerative nature, frequently (20%) associated with aortic aneurysms and other visceral aneurysms (40%). They equally affect males and females in the fifth decade of life.

Their real risk of rupture is still unknown; today it is estimated to be about 13%, with a mortality of 100% in emergency, that has led to an aggressive surgical management that can include aneurysmectomy, aneurysmorraphy, or ligation, with or without arterial reconstruction or endovascular treatment\(^3\) (Fig. 36.16).

### 36.3.10 SMA Aneurysms

They represent the 5.5% of all VAAs, generally placed in the proximal 5 cm of the artery.

They affect mainly men in their fifth decade of life and present an increasing trend in prevalence. They can be mycotic or caused by inflammation, trauma, arterial dissection, vasculitis, or dysplasia or degenerative disease. These aneurysms are often symptomatic, showing acute and colic upper abdominal pain, associated to nausea or vomiting; about half of the patients present a palpable pulsating abdominal mass.

The high risk of rupture of this lesion (50%) makes them prone to be surgically treated; techniques are the same as that described for celiac trunk aneurysms [60].

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**Key Points**

**AMI and symptomatic VAAs**

- Because of their high mortality rate, they represent an emergency condition and should be treated urgently.
- CT scan is the exam of choice for all patients with severe abdominal pain and clinical suspect of AMI, renal artery ischemia, or symptomatic VAAs.
- The treatment of choice is the endovascular one, because it is effective and less invasive than open surgery, with exception made for AMI with bowel necrosis when laparotomic access is mandatory to assess intestinal viability.

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**Fig. 36.16** Celiac trunk aneurysm (a) treated with a covered stent in the hepatic artery and excluding with coils splenic artery (b)

\(^3\)As for other VAAs, their site and morphology condition the kind of treatment (mainly embolization or placement of covered stent).
The lesion frequently is in the proximal tract of SMA or celiac trunk: the lack of a landing zone limits the use of coils or covered stents. Simple ligation in these lesions is possible thanks to the extensive collateral circulation between the two vessels, through the pancreatic arcades. Revascularization with venous or prosthetic graft or direct arterial reconstruction is necessary for patients with a ruptured aneurysm and bowel ischemia or preoperative signs or symptoms of intestinal ischemia [60].

Case Scenario

A. A 60-year-old woman with persistent increasing abdominal pain for 2 months. She refers worsening epimesogastric pain in the last 2 days. Previous medical history: CAD, hypertension, and diabetes.

Vital signs and physical exam result are within normal limits except for abdominal pain.

ECG and screening for myocardial infarction are negative. The abdominal US reveal a turbulent blood flow at the origin of the celiac trunk, but the vessel wall is not well defined.

1. Which is your diagnostic suspect?
   A. Myocardial infarction
   B. Celiac trunk stenosis vessels disease
   C. Celiac trunk aneurysm
   D. Both C and D
   E. None of the previous answers

2. What is the right imaging test for this patient?
   A. No imaging but analgesic therapy should be started.
   B. Contrasted US abdomen imaging.
   C. MR abdomen imaging.
   D. CT abdomen imaging.
   E. All the previous answers

3. Imaging revealed… (Fig. 36.17)
   A. Abdominal aortic aneurysm
   B. Celiac trunk aneurysm
   C. Celiac trunk stenosis
   D. Both B and C
   E. None of the previous answers

4. Considering this diagnosis what would you also have to exclude also at CT scan?
   A. Signs of aneurysmatic rupture
   B. Associated VAs
   C. Associated abdominal aortic aneurysms
   D. All the previous answers

5. Considering CT findings and clinical assessment, how do you treat the patient?
   A. Conservative approach, as she has a 1.5 cm celiac trunk symptomatic aneurysm, but she’s not of childbearing age, and there are no signs of rupture.
   B. Urgent endovascular treatment (coils embolization) of the aneurysmatic lesion.
   C. Urgent open surgical treatment.
   D. None of the previous answers.

Please see Chap. 58 for the correct answer.

References


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