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Review Article
ACUTE AND CHRONIC ISCHEMIA ,CLINICAL REVIEW

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ABSTRACT

Acute mesenteric schemia almost always involves the small bowel and may follow superior mesenteric artery thrombosis or embolism and almost associated with hypecoagable dieases and tend to affct smaller lenghthes of bowel .chronic mesenteric ischemia usually follows low flow in he inferior mesenteric artery territory and ranges from mild ischemia to gangerous colitis.

1. Introduction

Acute mesenteric ischemia is a life-threatening vascular emergency that requires early diagnosis and intervention to adequately restore mesenteric blood flow and to prevent bowel necrosis and patient death. The underlying cause is varied, and the prognosis depends on the precise pathologic findings . despite the progress in understanding the pathogenesis of mesentric ischemoa and the development of modern treatment modalities, acute mesentric ischemia remains a diagnostic challenge for clinicians. and the delay in diagnosis contributes to the continued high mortality rate. Early diagnosis and prompt effective treatment are essential to improve the clinical outcome.(58)

Acute mesenteric ischemia (AMI) is a potentially fatal vascular emergency with overall mortality of 60% to 80%,1-5 and its reported incidence is increasing.3 Acute mesenteric ischemia comprises a group of pathophysiologic processes that have a common end point bowel necrosis. The survival rate has not improved substantially during the past 70 years, and the major reason is the continued difficulty in recognizing the condition before bowel infarction occurs.(5)

REVIEW

PURPOSE OF REVIEW

Mesenteric ischemia (MI), both acute (AMI) and chronic (CMI), is a challenging diagnosis to make, and early diagnosis and treatment are vital to improve outcomes. This manuscript summarizes the most up to date information on diagnosis and treatment of these disorders.

Acute mesenteric ischemia (MI) results from insufficient splanchnic blood flow that is unable to meet the metabolic requirements of the small intestine. Despite MI being an uncommon cause of abdominal pain, failure to urgently recognize and intervene is associated with a grim prognosis. The last decade has seen a revolution within the interventional and surgical management of this multidisciplinary disease; however, despite these advances and reductions in overall mortality, MI still continues to be associated with poor outcome. This review discusses the various types of mesenteric ischemia, their presentation, diagnosis, prognosis, and varying modalities of treatment, with special emphasis on the recent developments in its management.(2)

MESENTERIC VASCULATURE ANATOMY AND PHYSIOLOGY

The mesenteric arterial circulation comprises three principal branches of the abdominal aorta, namely, the celiac axis (CA), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA). The CA supplies blood to the stomach, the first and second part of the duodenum, part of the pancreas, the liver, and the spleen. The SMA supplies blood to the remainder of the duodenum, jejunum, ileum, ascending colon, and the proximal third of the transverse colon. The IMA delivers blood to the distal colon including the distal transverse, descending, and sigmoid colon and the proximal rectum. The SMA is the primary artery for the small intestine with collateral supply from the CA via the pancreaticoduodenal artery [1]. An increase in the collateral circulation generally occurs when there is a stenosis of over 70% within the mesenteric arteries. The mesenteric blood flow accounts for about 15–20% of the cardiac output in the fasting state and up to 35% in the postprandial state. Following food intake, a surge in blood flow occurs within about 10–20 min, and its magnitude depends on the size and composition of the meal. Large, fatty meals cause the largest increase in blood flow. Apart from collaterals, the mesenteric circulation is able to autoregulate blood supply through several complex mechanisms. As a result, the small intestine can tolerate up to a 75% reduction in overall blood flow for as long as 12 h [2]; however, with complete occlusion, irreversible ischemia can occur in as short as 6 hours.

Acute mesenteric ischemia is interruption of intestinal blood flow by embolism, thrombosis, or a low-flow state. It leads to mediator release, inflammation, and ultimately infarction.(11)

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ACUTE MESENTERIC ISCHEMIA PATHOPHYSIOLOGY

The intestinal mucosa has a high metabolic rate and, accordingly, a high blood flow requirement (normally receiving 20 to 25% of cardiac output), making it very sensitive to the effects of decreased perfusion. Ischemia disrupts the mucosal barrier, allowing release of bacteria, toxins, and vasoactive mediators, which in turnleads to myocardial depression, systemic inflammatory response syndrome (see Sepsis and Septic Shock), multisystem organ failure, and death. Mediator release may occur even before complete infarction. Necrosis can occur as soon as 6 h after the onset of symptoms.(13).

The splanchnic circulation receives approximately 25% of the resting and 35% of the postprandial cardiac output(.3,4) Seventy percent of the mesenteric blood flow is directed to the mucosal and submucosal layers of the bowel, with the remainder supplying the muscularis and serosal layers. The physiologic characteristics of splanchnic blood flow are complex and incompletely understood. Multiple major elements interact to provide the intestinal tract with an appropriate share of the blood supply, including the intrinsic (metabolic and myogenic) and the extrinsic (neural and humoral) regulatory systems.(4,5)

Pressure-flow autoregulation, reactive hyperemia, and hypoxic vasodilation are considered intrinsic controls and are responsible for instantaneous fluctuations in splanchnic blood flow. In the metabolic theory, oxygen delivery rather than blood flow causes adaptive changes in splanchnic circulation. An imbalance between tissue oxygen supply and demand will raise the concentration of local metabolites (eg, hydrogen, potassium, carbon dioxide, and adenosine), resulting in vasodilation and hyperemia. In contrast, the myogenic theory suggests that arteriolar tension receptors act to regulate vascular resistance in proportion to transmural pressure. An acute decrease in perfusion pressure is compensated for by a reduction in arteriolar wall tension, thereby maintaining splanchnic blood flow.

The extrinsic neural component of splanchnic

Circulatory regulation comprises the α-activated vasoconstrictor fibers. Intense activation of vasoconstrictor fibers through α-adrenergic stimulation results in vasoconstriction of small vessels and a decrease in mesenteric blood flow. After periods of prolonged α-adrenergic vasoconstriction, blood flow increases, presumably through β-adrenergic stimulation, which acts as a protective response. After cessation of α-adrenergic stimulation, brief hyperemia makes the response triphasic.

Although various types of neural stimulation (eg, vagal, cholinergic, histaminergic, and sympathetic) can affect the gut, the adrenergic limb of the autonomic nervous system is the predominant and possibly the sole neural influence on splanchnic circulation.

Numerous endogenous and exogenous humoral factors are capable of affecting the splanchnic circulation. Norepinephrine and high levels of epinephrine produce intense vasoconstriction through the stimulation of adrenergic receptors. Other pharmacologic compounds that decrease splanchnic blood flow include vasopressin, phenylephrine, and digoxin.(6) Low-dose dopamine causes splanchnic vasodilation, whereas higher doses lead to vasoconstriction by stimulating α-adrenergic receptors.

Papaverine, adenosine, dobutamine, fenoldopam mesylate, and sodium nitroprusside are exogenous agents that increase mesenteric blood flow. In addition, various naturally occurring agents can serve as splanchnic vasodilators, including acetylcholine, histamine, nitric oxide, leukotrienes, thromboxane analogues, glucagon, and an assortment of gastrointestinal hormones. The effects of prostaglandins are variable.

In summary, the splanchnic circulation is regulated by a complex array of physiologic and pharmacologic factors.

Reperfusion Injury

Tissue damage due to alterations in mesenteric blood flow is often the result of cellular injury associated with reperfusion(.7,8) Brief periods of mesenteric ischemia lead to an increase in microvascular permeability, whereas prolonged ischemia leads to disruption of the intestinal mucosal barrier, primarily through the actions of reactive oxygen metabolites and polymorphonuclear neutrophils.

The role of oxygen free radicals in reperfusion injury is demonstrated by the reduction of tissue damage in the presence of antioxidants, xanthine oxidase inhibitors, and free-radical scavenging substances. Polymorphonuclear leukocytes contain enzymes that reduce molecular oxygen to superoxide anions and produce hypochloric acid, providing an additional source of reactive oxygen metabolites. Epithelial cells may produce xanthine oxidase–derived oxidants and initiate the production of proinflammatory agents that attract polymorphonuclear leukocytes.(17) In addition, phospholipase A2 is activated during reperfusion, increasing the formation of cytotoxic lysophospholipids within the ischemic tissue and up-regulating the production of prostaglandins and leukotrienes.18 Further understanding of the role of reperfusion injury may present opportunities for protective pharmacologic therapies with agents such as captopril and carvedilol.(19,20) Carvedilol, a new β-adrenoreceptor blocking agent and a free-radical scavenger, has been demonstrated to have an antishock and endothelial-protective effect in a rat splanchnic ischemia reperfusion model.(20)

The degree of reduction in blood flow that the bowel can tolerate without activating these reperfusion mechanisms is remarkable. Only one fifth of the mesenteric capillaries are open at any given time, and normal oxygen consumption can be maintained with only 20% of maximal blood flow. When splanchnic blood flow is restored, oxygen extraction increases, providing relatively constant oxygen consumption over a wide range of blood flow rates.12 However, when blood flow decreases below a threshold level, oxygen consumption is reduced and oxygen debt ensues

AETIOLOGY

The common causes of acute mesenteric ischaemia can be classified into:

Thrombus-in-situ (Acute Mesenteric Arterial Thrombosis, AMAT)

Embolism (Acute Mesenteric Arterial Embolism, AMAE)

Non-occlusive cause (Non-Occlusive Mesenteric Ischemia, NOMI)

Venous occlusion and congestion (Mesenteric Venous Thrombosis, MVT)

TYPE	Proportion of Cases	Underlying CAUSE Atherosclerosis
AMAT	25%	Cardiac causes* or abdominal / thoracic aneurysm
AMAE	50%	Hypovolemic Shock, Cardiogenic Shock Coagulopathy, Malignancy
NOMI	20% MVT<10%	Inflammatory Disorders

*Cardiac causes include arrhythmias (e.g. AF), post-MI mural thrombus, or prosthetic heart valve

Rarer causes for acute mesenteric ischaemia include Takayasu's arteritis, fibromuscular dysplasia, polyarteritis nodosa, and thoracic aortic dissections.(22)

RISK FACTORS

The risk factors for acute mesenteric ischaemia depend on the underlying cause.

Specifically, however for AMAE, the main reversible risk factors are smoking, hyperlipidaemia, and hypertension, much the same as for chronic mesenteric ischaemia.

Other potential causes are atrial fibrillation, intracardiac thrombus, or thoracic aneurysm / thrombus.(3)

RISK FACTORS FOR POOR PROGNOSIS IN ACUTE MESENTERIC ISCHEMIA

In those with AMI, older age, delayed diagnosis, elevated lactic acid levels at diagnosis and 24 h following diagnosis, and NOMI are all associated with increased risk of 30-day colectomy and mortality [63, 89, 90]. Worsened outcome is associated with the vessel involved and distribution. AMI from arterial occlusion has a significantly higher mortality rate than that from MVT. The latter has the lowest mortality among the four common etiologies. Occlusions of the CA and IMA can be compensated by autoregulation and collateralization from the SMA, whereas occlusion of the SMA almost always results in ischemic damage. Proximal arterial occlusions usually result in more extensive ischemic insult than peripheral occlusions with the severity and extent of ischemia (transmural or not) and the intestinal segments involved also being associated with poor outcome Global ischemia of the small and large bowel results in higher mortality [73].

Also, findings on a CTA correlate with the ischemic damage and can predict prognosis in those with AMI. Arterial occlusion, bowel wall hyperdensity, and the absence of thickening and enhancement mark earlier stages of AMI and are associated with a better prognosis. Pneumatosis linearis, portal venous gas, pneumoperitoneum, and loop dilatation occur late in the disease and are associated with higher mortality [36, 38]. By considering these factors, clinicians will be able to better triage patients who might require more aggressive interventions earlier in the treatment course

CAUSES

Acute mesenteric ischemia can be categorized into 4 specific types based on its cause (Table 2).

Arterial Embolism

Arterial emboli are the most frequent cause of AMI and are responsible for approximately 40% to 50% of cases.2,3 Most mesenteric emboli originate from a cardiac source. Myocardialischemia or infarction, atrial tachyarrhythmias, endocarditis, cardiomyopathies, ventricular aneurysms, and valvular disorders are risk factors for the development of mural thrombus, which can subsequently embolize to mesenteric arteries(9).Rarely, a mesenteric artery embolus can occur during or after angiography of the coronary or cerebral circulation. Most visceral arterial emboli preferentially lodge in the superior mesenteric artery (SMA) because it emerges from the aorta at an oblique angle.

Whereas 15% of arterial emboli occur at the origin of the SMA, 50% lodge distally to the origin of the middle colic artery, which is the first major branch of the SMA(10,8).Nearly one third of all patients with an SMA embolus have a history of an antecedent embolic event.

The onset of symptoms is usually dramatic as a result of the poorly developed collateral circulation, and it is characterized by the abrupt onset of severe abdominal pain associated with diarrhea, which may become bloody. Frequently, the diagnosis of SMA embolism can be made intraoperatively based on the distribution of ischemic bowel. Because most SMA emboli lodge distally to the origin of the middle colic artery, allowing the inferior pancreaticoduodenal branches to be perfused, the proximal jejunum is spared, whereas the rest of the small bowel is ischemic or infarcted.

Arterial Thrombosis

Acute mesenteric thrombosis accounts for 25% to 30% of all ischemic events(.2,11,12)Almost all mesenteric ischemia due to arterial thrombosis occurs in the setting of severe atherosclerotic disease, with the most common site near the origin of the SMA.7 Frequently, patients with this condition can tolerate major visceral artery obstruction because the slow progressive nature of atherosclerosis allows the development of important collaterals. Bowel ischemia or infarction ensues when the last remaining visceral artery or an important collateral artery occludes. The extent of bowel ischemia or infarction is typically greater than that with embolism, extending from the duodenum to the transverse colon.

Perioperative mortality ranges from 70% to 100%(3,11,12)in part because of the delay in diagnosis, the extensive nature of the bowel ischemia-infarction, and the need for more complex surgical revascularization. (11)

Nonocclusive Mesenteric Ischemia

Approximately 20% of patients with mesenteric ischemia have nonocclusive disease.(13,14) The pathogenesis of nonocclusive mesenteric ischemia (NOMI) is poorly understood but often involves a low cardiac output state associated with diffuse mesenteric vasoconstriction.

Splanchnic vasoconstriction in response to hypovolemia, decreased cardiac output, hypotension, or vasopressors best explain the difference between this entity and other forms of AMI. The resultant low-flow state causes intestinal hypoxia and necrosis. Endogenous and exogenous vasoconstrictors, disseminated intravascular coagulation, and reperfusion injury may also contribute. Vasoactive drugs, particularly digoxin, have been implicated in the pathogenesis of NOMI. Digitalis preparations induce contraction of splanchnic venous and arterial vascular smooth muscle in vitro and in vivo.(15) Watershed areas of circulation are more vulnerable in NOMI.

Conditions predisposing to NOMI include age older than 50 years, myocardial infarction, congestive heart failure, aortic insufficiency, cardiopulmonary bypass, renal or hepatic disease, and major abdominal or cardiovascular surgery. However, patients may not have any clear risk factors.(16,14) Because this condition frequently affects critically ill patients who have considerable comorbidities, the onset may be insidious, and the mortality rates are high.

Between 1960 and 1980, because of the frequent use of vasopressors in cardiac patients, mortality was nearly 100%. With increasing use of afterload-reducing agents and vasodilators, the mortality rate associated with NOMI has declined.

An unusual form of nonocclusive ischemia has been described in patients who have undergone the stress of a surgical procedure or trauma and are receiving enteral nutrition in intensive care units.(17) The reported incidence of AMI in these patients is 0.3% to 8.5%. The proposed mechanism is an imbalance between demand (created by the enteral feedings) and supply (decreased by systemic hypoperfusion and mesenteric vasoconstriction). Most patients manifest signs of sepsis, with abdominal distention as a late clinical sign. Survival is poor (56%). (17)

Mesenteric Venous Thrombosis

Mesenteric venous thrombosis (MVT) is the least common cause of mesenteric ischemia, representing up to 10% of all patients with mesenteric ischemia and 18% of those with AMI. In the past, most cases were thought to be secondary to other intra-abdominal pathologic conditions (such as malignancy, intra-abdominal sepsis, or pancreatitis) or were classified as idiopathic. With improved diagnostic techniques, more cases have been shown to be related to primary clotting disorders, with only 10% of cases now being classified as idiopathic(3,17,18)

Mesenteric venous thrombosis is usually segmental, with edema and hemorrhage of the bowel wall and focal sloughing of the mucosa. Thrombi usually originate in the venous arcades and propagate to involve the arcuate channels.

Hemorrhagic infarctions occur when the intramural vessels are occluded. The thrombus is usually palpable in the superior mesenteric vein.(18) Involvement of the inferior mesenteric vein and large bowel is uncommon. The transition from normal to ischemic intestine is more gradual with venous embolism than with arterial embolism or thrombosis.

Mortality depends on the type of MVT (acute vs chronic) and the extent of venous involvement. Patients with acute disease with involvement of the superior mesenteric or portal vein have a 30-day mortality approaching 30%. Long-term in those with the chronic form(18)

CLINICAL PRESENTATION

The “classic” presentation for mesenteric ischemia will be in a patient over the age of 60. Women are three times more likely than men to have acute mesenteric ischemia. Patients will present with sudden abrupt onset of abdominal pain which may be associated with nausea, vomiting, and diarrhea. The abdominal pain will initially be severe and diffuse without any localization. One of the distinctive findings in mesenteric ischemia is that the abdominal pain is out of proportion to their physical exam. The patient may be screaming in pain, but their initial abdominal exam can be soft with no guarding or rebound. This is because the ischemia is in the wall of the hollow viscus of the intestine and therefore does not cause the same peritoneal signs that would be present in appendicitis, cholecystitis, and other more localized processes. As the disease progresses and the bowel infarcts, the patient will develop abdominal distension with guarding, rebound, and absence of bowel sounds. They may develop abdominal wall rigidity. Bloody diarrhea and heme-positive stools are a late finding after bowel has infarcted.

The aforementioned description is the classic presentation, often seen on standardized tests. Unfortunately, in practice patients may present with postprandial pain or generalized abdominal pain that can mimic other disease processes making the diagnosis of mesenteric ischemia less obvious. The postprandial pain is similar to exercising with unstable angina in the myocardium: with decreased blood supply to the intestines, when patients eat and demand higher oxygen utilization during digestion, the flow is insufficient and causes ischemic pain.

This is why this type of pain is often referred to as abdominal angina. To truly understand the real-life presentations of mesenteric ischemia, the four different etiologies of this disease must be analyzed: mesenteric artery embolism, mesenteric artery thrombosis, mesenteric vein thrombosis, and non-occlusive ischemia.

DIFFERENTIAL DIAGNOSES

Acute Mesenteric Ischemia.

A crucial point is differentiation of stable chronic symptoms from symptoms that have worsened recently. If the pain has recently become permanent or if peritoneal symptoms have developed, acute ischemia should be suspected and an emergency laparotomy should be performed. In patients who are seen very early, angiography prior to surgery may deservediscussion as a means of establishing the diagnosis and allowing local fibrinolytic treatment of an acute thrombus or embolus(19). An intraarterial infusion of papaverine should be given to patients with nonocclusive acute mesenteric ischemia (,20).

Retroperitoneal or Celiomesenteric Malignancy.—

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

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

Median Arcuate Ligament Syndrome

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

Although some authors have argued that the pain is caused by gastroduodenal tract ischemia (,21), others believe that it arises from celiac plexus compression and consequently is not an indication for PTA or stent placement (,22–23). We share this point of view.

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

Duplex US, which is used more routinely, shows the breathing-related changes in the degree of stenosis, with increased turbulence or flow velocity during expiration (,21) (,,,Fig 7).

CT has been found useful in diagnosing this syndrome (,22): The median arcuate ligament can be identified easily as a hypoattenuating structure crossing the aorta above the departure of the CA.

TREATMENT OF THIS CONDITION NOWADAYS

takes the form of laparoscopic division of the median arcuate ligament (,24).

Gastroduodenal Ulcer

The diagnosis of gastroduodenal ulcer is suspected when the pain is alleviated by meals, and this diagnosis is confirmed with endoscopy. Ischemic gastropathy is extremely uncommon.

This diagnosis should be suspected only whenthe lesions are progressive or refractory to treatment.

Nonocclusive Vascular Lesions: Aneurysms and Dissections.—

Aneurysms or false aneurysms of the visceral arteries are uncommon (,25). Embolization is gaining ground as the treatment of choice for these abnormalities (,26,27).

Rarely, the aneurysm is a complication of isolated dissection of CA branches . A few other cases of painful isolated dissection of the visceral arteries have been reported (,28,29).

False aneurysms of the visceral arteries are easier to differentiate from CMI because a history of blunt abdominal trauma or chronic pancreatitis is usually present (,26).

Less uncommon vascular causes of abdominal pain are aneurysms or dissection of the abdominal aorta .However, the symptoms are generally less progressive than in CMI.(20)

DIAGNOSTIC TESTING

Labs

Generally, labs are not helpful in making the diagnosis of mesenteric ischemia from other abdominal pathologies as no single lab has the sensitivity and specificity to rule in or rule out the disease. The white blood cell count is commonly elevated, but is a nonspecific finding and a normal white count does not rule out the disease. Hemoconcentration, elevated amylase levels, and metabolic acidosis may also be found in mesenteric ischemia, but are non-specific findings. An elevated lactate level is sensitive for mesenteric ischemia however is frequently seen in late disease after bowel infarction. Lactate cannot be used as a negative screening test. It can be followed during the disease to determine is bowel loss is occuring. One study did show some promise with D-dimer testing. In that study, no patient presenting with a normal D- dimer had intestinal ischemia. Further studies focusing on D-dimer use in mesenteric ischemia are required.

Plain Radiography

Plain films of the abdomen will typically be normal early in the course of the disease. An upright abdominal x-ray can be used to rule out free air from a perforated viscus. As the ischemia progresses, subtle signs such as thickening of bowel wall and distended loops of bowel can be seen, but like the labs are non-specific signs. Pneumatosis of the intestinal wall can afterwards be seen on plain film, but is a late finding when bowel has become necrotic.

Angiography

Mesenteric angiography was the gold standard for mesenteric ischemia but has been replaced in recent years by multidetector CT angiography of the abdomen and pelvis due to advances in CT imaging. Mesenteric angiography can identify the site and type of occlusion including non- occlusive ischemia. Medications such as papaverine (a vasodilator) and thrombolytics can also be infused during mesenteric angiography allowing for interventions on emboli and thrombi. The downsides of angiography are that it is an invasive and lengthy procedure and may not be readily available at all hospitals or all times of day. In addition, angiography will not evaluate other causes for abdominal pain such as bowel obstruction, renal colic, genitourinary disorders and gynecologic diseases. (62)

Multidetector CT Angiography (CTA) of Abdomen/Pelvis

CTA of the abdomen/pelvis has largely replaced mesenteric angiography as the tool to diagnose mesenteric ischemia. Recent studies have shown CTA to have a sensitivity of 93% and specificity up to 100% for mesenteric ischemia. In comparison to angiography, CTA is fast, less invasive, and more readily available. In addition to the vascular findings of thrombus and emboli, CTA can also demonstrate subtler signs of mesenteric ischemia such as circumferential thinking of the bowel wall, bowel dilatation, bowel wall attenuation, and mesenteric edema which may not be seen on angiography. Other pathologies such as colitis, appendicitis and bowel obstruction, which may mimic mesenteric ischemia, can also be diagnosed on CTA.

It is for these reasons that CTA is the initial test to obtain for patients in who the diagnosis of mesenteric ischemia is being considered.

Unlike angiography, CTA cannot provide therapy, but, can help triage patients towards those who can undergo angiography and those who should go to the operating room immediately. CT angiography requires intravenous contrast to be administered, and contraindications to iodinated contrast should be considered prior to obtaining the examination.

Renal insufficiency is often comorbid with patients who have multisystem disease and the theoretical risk of contrast induced nephropathy should be weighed against the risk of missing the diagnosis if angiography is not performed.

Oral contrast is not indicated.(78)

Approach to a patient with suspected acute mesenteric ischemia (AMI). Resuscitation, antibiotics, anticoagulation and other supportive care should be provided to all patients. (PTA: Percutaneous transluminal angioplasty, LMWH: Low-molecular-weight heparin, DCS: Damage control surgery, PG E1: Prostaglandin E1, ICU: Intensive care unit.

TREATMENT

Initial treatment in mesenteric ischemia must focus on stabilization and resuscitation. Two large bore IVs with crystalloid fluids are necessary in patients, especially those who are hypotensive. Continuous monitoring of vital signs is paramount. Broad spectrum antibiotics covering bowel flora, such as ceftriaxone and metronidazole, should be started. Early surgical consultation is highly recommended so that the surgeons can closely follow the patient, do serial abdominal examinations, review the CT imaging with radiology, and take the patient to the OR rapidly thus saving as much bowel as possible.

Anticoagulation should be considered and discussed with the surgeons. A shorter-acting anticoagulant such as an unfractionated heparin infusion may be optimal as it can be shut off quickly if the patient is to go to the OR.

The ultimate management of acute mesenteric ischemia is challenging, ever-changing and diverse. Treatment can range from non- operative management with medications, intravascular thrombolytics, percutaneous angioplasty, operative revascularization, resection of bowel, or a combination of therapies. The treatment for each patient must be individualized depending on the patient's state of health, cause of ischemia, and resources that are available. Listed below are the treatments based on the four etiologies of mesenteric ischemia.

Mesenteric Artery Embolism

The treatment of choice for mesenteric artery embolus is embolectomy and bowel visualization to assess for signs of necrosis.

Percutaneous treatment with thrombolytics directly infused into the artery containing the embolism during angiography is another option for patients who do not have peritoneal signs or are non-operative candidates. The drawback is that bowel viability generally assessed during laparotomy cannot be done. In addition, contraindications to thrombolytics including recent surgery or GI bleed, recent stroke, and peritoneal signs indicating bowel infarction must be considered.

If operative management is decided, revascularization is done first so that ischemic- looking bowel can recover with the return of blood flow. Once blood flow is reestablished, any bowel that remains infarcted and necrotic is then resected. A “second look” procedure 24-48 hours later may be done if the viability of a section of bowel was in question during the first surgery. Mesenteric Artery Thrombosis

Patients who develop peritoneal signs must go to the OR(17)

In this etiology, heparin should be started as soon as the diagnosis is made and prior to surgery. The corrective operative measures for mesenteric artery thrombus are the same as for mesenteric artery embolism. For non-operative candidates, percutaneous transluminal angioplasty with stenting is an option. In patients with chronic mesenteric ischemia and mesenteric artery thrombosis, there has been complete resolution of symptoms after intervention.

Mesenteric Vein Thrombosis

If there are signs of infarction, then operative care is required. Otherwise thrombectomy with endarterectomy or distal bypass is the first choice of treatment. Anticoagulation is routinely administered to prevent thrombus reoccurrence. These patients will generally require life-long anti-coagulation.

Non-occlusive Mesenteric Ischemia

The treatment is to diagnose the underlying cause of the low flow state to the bowel such as sepsis or decreased cardiac output. Patients who develop peritoneal signs must go to the OR(17)

CHRONIC MESENTERIC ISCHEMIA

Many patients with CMI are asymptomatic due to collateral circulation within the mesenteric arterial tree. The prevalence of asymptomatic stenosis of either CA or SMA is 3% in those younger than 65 years and 18% in those older than 65 years [24]. Postprandial epigastric or mid-abdominal pain starting soon after a meal and lasting approximately 30 min to 2 h is most common [25]. The classic terminology used to describe this pain, arising from the inability of the mesenteric vasculature to keep up with the increasing metabolic oxygen demand in the postprandial state is “abdominal angina” or “intestinal angina” [26, 27]. With time, patients learn to alleviate their pains by eating smaller meals and avoiding fatty foods. This gradually leads to an aversion to or fear of food (e.g., sitophobia), and weight loss [28]. CMI might also present with chronic diarrhea, recurrent peptic ulcers, or proximal colitis. One must be mindful of this diagnosis in any patient with abdominal pain since CMI is frequently missed; the average time from presentation to diagnosis is 20–25 months [29, 30]. With progression of disease, the pain changes from intermittent to persisting for longer after meals and eventually continuous discomfort. In advanced stages, patients with CMI are prone to an evolution to acute severe symptoms and AMI. AMI requires prompt recognition and intervention.

Collateralization of the SMA most commonly results in MALS being asymptomatic. In 10– 20%, ischemic symptoms can result from CA compression, which is also known as celiac artery compression syndrome (CACS) or Dunbar syndrome [31]. This is most commonly seen in women between 30 and 50 years of age. Having a similar presentation to CMI, CACS mostly presents with postprandial abdominal pain but vomiting, bloating, and poor appetite are also seen [32]. The classical triad comprises postprandial pain, weight loss, and an abdominal bruit.

CLINICAL PRESENTATION

Abdominal angina, which was first described at the beginning of the 20th century, is defined as postprandial abdominal pain with weight loss and anorexia. Changes in bowel habits and vomiting are less common.

In two of our patients, ischemic gastropathy was the final diagnosis. One of these patients had incapacitating gastroparesis with vomiting, postprandial heaviness, and delayed gastric emptying at barium study. The other had severe, endoscopically documented gastroduodenitis unresponsive to pharmacotherapy. In both patients, prompt resolution of the symptoms after PTA supported the diagnosis of ischemic gastropathy. A few reports of reversible gastric symptoms after revascularization have been published(36,37). We agree with Casey et al (,13) that the visceral arteries should be investigated, at least with Doppler US, in patients with chronic gastric symptoms.

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

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

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

The prevalence of CMI seems to increase with age, in agreement with the age-related increase in the prevalence of visceral artery occlusion in autopsy studies (,13,,14). However, CMI can occur in younger patients and the weight loss can suggest a malignant disease (37)

COMPLICATIONS

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

A few cases of PTA-induced dissection have been reported (,34,,47); stent placement was usually successful. However, in one of our patients, dissection of the CA occurred during attempted recanalization. Two stents were implanted, but Doppler US performed the next day showed that the artery was occluded (,Table 2).

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

DIAGNOSIS

As is the case with AMI, the role of serum markers in the diagnosis of CMI is very limited [31]. Postprandial elevation in D-dimer and L-lactate has been associated with this diagnosis but this is non-specific. I-FABP, CRP, LDH levels, and leukocyte counts are of little value.

Duplex ultrasonography (US) is a useful screening tool for suspected mesenteric stenosis. It is not associated with any radiation exposure, has no adverse effects, and can be reliable in experienced centers. When diagnosing SMA stenosis of greater than 70%, it has a sensitivity of 72–100% and a specificity of 94–98% [32,33]. It is also widely used as follow- up imaging to confirm open blood flow after endovascular and surgical interventions. This technique is operator dependent, and in some low-volume centers, the accuracy of this modality might be lower.

CTA, with its ability to reconstruct images, provides maximum details about the mesenteric vasculature and other intraabdominal organs.

With a sensitivity of 100% and specificity of 95%, it has largely superseded digital subtraction angiography as the imaging of choice in the diagnosis of mesenteric artery stenosis (MAS) [34]. The latter is still used in some limited settings, like intra-operatively during endovascular interventions, and in preoperative assessment of patients with MALS to determine the extent of the lesion and dynamic changes with respiration.

Magnetic resonance angiography (MRA) is a useful modality for the diagnosis of mesenteric stenosis. There is very limited data comparing MRA with CTA in those with CMI. The advantages are that it has no radiation exposureand can allow a more accurate estimation of flow velocity and volumes than CTA. In patients where CTA is contraindicated, MRA is the next preferred imaging for CMI. This is not preferred in those with AMI given the extensive time it takes to complete an MRA study.

Selective mesenteric angiography is rarely used for diagnosis in modern times; however, given the complexity of patients with NOMI, the absence of clear radiological signs and potential interventions via angiography, this diagnosis may require this technique. Functional testing for mesenteric ischemia is indicated in those with asymptomatic mesenteric stenosis and in single vessel disease [38]. It is not required in patients with typical symptoms of CMI and multi-vessel involvement. Visible light spectroscopy (VLS) [39] is the technique most commonly used. With this assessment, during endoscopy, a fiber-optic catheter measures the oxygen saturations achieved in the upper gastrointestinal mucosa. For the diagnosis of CMI, VLS has a sensitivity of about 90% and a specificity of 60% [43]. Air tonometry is another functional test used to measure the partial pressure of carbon dioxide (pCO_2) and the pH in the gastrointestinal mucosa (19,40]. Provocative testing after exercise [41,42] and eating is useful in the initial diagnosis of CMI and follow-up of those after revascularization.

When clinically recognized, the diagnosis of CMI should be straightforward in those with symptoms typical of ischemia and multi-vessel obstructive disease identified on CTA. However, in those with single vessel stenosis, it is imperative to rule out other etiologies of abdominal pain. Abdominal US and CT are useful in the assessment of the gallbladder and pancreas. Though upper endoscopy and colonoscopy have no direct role in the diagnosis of CMI, they can rule out other underlying causes as well. Functional testing, although not readily available in many institutions, might also be used to differentiate CMI from other diagnoses.

MANAGEMENT

Medical Management

The initial step to reduce the metabolic demand of the mesenteric vasculature should be to advise more frequent, smaller, low-fat meals. A nutritionist can help ensure a well-balanced diet avoiding weight loss and vitamin deficiencies that can frequently be seen. Aggressive medical atherosclerotic risk factor reduction plays an

important role in managing CMI. Weight reduction, smoking cessation, and appropriate management of comorbidities such as diabetes mellitus, hypertension, and hyperlipidemia reduce the risk for thrombosis and a more severe acute presentation.

Surgical Management Revascularization

In patients with CMI, the question of "whom to treat" is frequently challenging for the clinician [44]. A clinical approach for those with CMI is summarized in Fig. 3. The primary considerations include the presence of typical symptoms, single vessel or multi-vessel stenosis, and overall functional status of the patient.

Multi-vessel disease is defined as stenosis of both SMA and CA of more than 70%.

Symptomatic multi-vessel disease should always be treated [45]. In symptomatic patients with single vessel stenosis, those with typical symptoms without another etiology for the pain should be considered for treatment. In these patients, if functional testing is available, a positive functional ischemia test (VLS or tonometry) is a good indication for treatment since they are most likely to benefit [19]. When these tests are not available, empiric treatment should be strongly considered. Most patients with asymptomatic mesenteric stenosis do not require intervention. Those with a good functional status and multi-vessel asymptomatic disease should be considered for treatment since most of these patients will progress to AMI if no intervention is made. Another subsets of asymptomatic patients who may benefit from treatment are those planned for major abdominal surgeries since these individuals are likely to have a more complicated peri-operative course without intervention(46)

Approach to a patient with suspected chronic mesenteric ischemia (CMI). Aggressive risk factor management is an important part of Percutaneous

TREATMENT

Angiographic Findings

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

TECHNIQUES OF PTA

Choice Of Route And Catheters

Since the first reports of PTA of the SMA or CA in 1980, less aggressive, more slender catheters have been developed and advances in the design of the balloon and stent catheter have made the procedure easier to perform. This explains why the axillary route comfort.

has been discarded by most teams. However, the humeral route is still used, particularly for PTA and stent placement in the CA; there are three curves in opposite directions when the CA is approached by the femoral route but only two when it is approached by the humeral route. In our experience, the best catheter geometries for selective catheterization of the visceral arteries via the femoral approach are the sidewinder configuration, the hook configuration, and, more rarely, the cobra configuration. With the humeral route, the vertebral configuration seems effective.

Control angiograms can be obtained by means of a long-armed sheath positioned in front of the mesenteric vessels. The coaxial technique, which uses materials developed for coronary artery angiography, is an alternative that provides optimal procedure As in other endovascular procedures, heparin as a 5,000-U bolus should be injected as soon as selective catheterization is started. This has been shown to reduce the complication rate.

Single-Vessel or Multiple-Vessel Revascularization?—

According to the literature, revascularization is generally performed in only one obstructive lesion. Our opinion is that revascularization of the SMA should be attempted first.

Revascularization of the CA could be reserved in case of failure of the SMA revascularization attempt. This is consistent with the experience of other teams, in which revascularization was far more difficult at the CA than at the SMA because of the more sinuous course of the CA in most patients. Because a humeral route conversion is generally required for CA revascularization, certain authors advocate this route as the first step.

In all 16 of our patients, at least one PTA procedure was successful. However, PTA of both the CA and SMA was successful in only one of seven patients. In the three cases of failed SMA revascularization, the CA could be treated successfully . There have been few reports of multiple-vessel revascularization to achieve symptom relief Single-vessel revascularization of the CA or SMA usually ensures a favorable long-term outcome.

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

Occlusion

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

Our experience suggests that a notch on the lateral angiogram predicts successful recanalization because it is usually associated with a short, nonostial occlusion. Routine administration of fibrinolytic agents prior to passing the catheter across the lesion has been advocated (,43,,44). In our population, fibrinolysis was not used in the two cases of SMA occlusion in which PTA was successfully performed, and no periprocedural signs of distal migration occurred. In our opinion, fibrinolysis is not appropriate in patients with stable symptoms of CMI because collateral vessels from the CA or IMA supply blood to the SMA close to its origin, so that the occlusion is usually very short, even when this is not obvious on

the angiogram. In one of our two cases, contrast- enhanced CT clearly showed that the SMA occlusion was less than 2 cm long. Moreover, in chronic occlusions there is no recent blood clot and consequently the efficacy of fibrinolysis is debatable. However, because migration is a serious event, we recommend fibrinolysis in patients with recent onset or worsening of symptoms.

Stent Placement

Recent reports have shown that stent placement in visceral arteries is feasible (,45,,46). Primary stent placement is now the standard of reference for renal ostial lesions, which represent 70% of renal artery stenoses. Because celiac stenoses and most mesenteric stenoses frequently have an ostial component, we believe that primary stent placement should be the rule, as for renal stenoses. Besides, we share the opinion of Sheeran et al (,45) that routine stent placement is in order to achieve recanalization or treat procedure-related dissection. Because new-generation stents are more flexible, easier to implant, and smaller in diameter, they will probably be increasingly used to prevent restenosis of lesions with moderate recoil. Selection of the stent depends on the site of the lesions and the experience of the operator. At our institution, the balloon-expandable stent was preferred for ostial lesions because of its greater radial force, and autoexpanding stents were reserved for arterial trunk lesions because of their greater flexibility. However, the new balloon-expandable stents are more flexible, and the introduction of coaxial catheterization by using a 0.14- or 0.18-inch guide wire has substantially reduced the external diameter of the sheath at the puncture site.

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

COMPARISON WITH OUTCOMES AFTER SURGERY

Early Outcome

Our 16 patients were able to eat the day after the procedure and were able to leave the hospital within 1 week. At the 3-month evaluation, they all reported improvement in symptoms (,Table 2). This dramatic improvement in symptoms after PTA has been reported consistently. The improvement in symptoms seems somewhat slower after surgery, probably because of the ileus induced by anesthetics.(72)

Midterm Outcome

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

It has been suggested that surgery should be the treatment of choice, at least in selected patients, because it provides a better long-term patency rate (,48–,51). In the medium term, only two of our 16 patients experienced recurrent symptoms, and in both repeat PTA was successful (,Table 2). The data from our patients and other studies show that most recurrences take place within 1 year after PTA and that mortality is related primarily to comorbidities (,Table 2). However, the patency of the treated vessels has not been systematically assessed in our patients, as in most studies. Therefore, it would be interesting to obtain data from a randomized trial that would comprise a patency study over the mid- and long term. Actually, relief of pain does not necessarily equate with patency.

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

TABLE 2. Procedures Performed, Results, and Outcomes in 16 Patients with CM

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

†Case of occlusion.

‡Patient had median arcuate ligament

syndrome.

§Treated with PTA of the SMA and CA.

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

TABLE 3. Patient Data and Procedure Success Rates from Studies of PTA

Note.—Values in parentheses are raw data

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

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

Note.—Values in parentheses are raw data.

*Primary assisted.

†Values are mean or range.

‡Death caused by acute limb ischemia and

septicemia.

§Death caused by occlusive dissection and bowel infarction

PROGNOSIS

Perioperative mortality in patients undergoing revascularization for AMI ranges from 44% to 90%.(35) Published data on long-term results after successful revascularization are few, and, in general, prognosis is not as favorable as that for patients with chronic mesenteric ischemia. Recurrence is not uncommon, and it carries a poor prognosis. A small proportion of patients survive massive bowel resection and develop short-gut syndrome, requiring long-term total parenteral alimentation or small-bowel transplantation.

Primary survey and ipitial actions

Mesenteric ischemia is a time-sensitive disease process as delays in diagnosis will lead to increased morbidity and mortality, especially in elderly patients. The first and most important initial action is to consider mesenteric ischemia in the differential diagnosis of all elderly patients with abdominal pain. The importance of early consideration and diagnosis of mesenteric ischemia cannot be overemphasized. Other initial actions will include large bore intravenous access, fluid resuscitation, and telemetry monitoring. Obtain an ECG to see if the patient has atrial fibrillation which can put them at risk for an embolic cause of mesenteric ischemia. Discuss the case with the surgeons as early as possible so that they can monitor for changes in the patient's abdominal exam. An initial benign, soft abdominal exam can become peritoneal and that may lead the surgeons to take the patient to the operating room rapidly in order to preserve as much bowel as possible.

Consider aggressive fluid administration early in the patients ED course as well as addressing any other abnormalities in the primary survey. If the patient is becoming hypoxic or has dyspnea due to fluid resuscitation, apply oxygen via nasal cannula, a non-rebreather mask, or non-invasive positive pressure ventilation via BiPAP. Consider intubation if their breathing worsens despite those. (70)

SUMMARY

Acute mesenteric ischemia is a challenging clinical problem with diverse causes, which often results in delayed diagnosis and treatment. A strong clinical suspicion and an aggressive approach should be adopted in dealing with this condition because the outcome crucially depends on rapid diagnosis and treatment. With better understanding of the pathogenesis of AMI and the availability of a range of diagnostic and interventional techniques and adjuvant pharmacotherapies, an improved outcome can be achieved.

Mesenteric ischemia is a rare cause of abdominal pain, but it should remain on the differential diagnosis for any patient presenting with abdominal discomfort. With better recognition and tools for intervention, mortalityfrom AMI has decreased from ~ 50–75% previously to ~ 25% more recently. Treatment should be guided by clinical stability and extent of ischemia with a multidisciplinary approach, including internists, intensivists, gastroenterologists, interventional radiologists, and surgeons. Though treatment strategies have improved with advances in pharmacotherapy, percutaneous interventions, and critical care, diagnosing AMI before bowel necrosis remains central to improve outcomes. CMI is a slowly progressive entity and prompt recognition and intervention are essential to optimize outcomes as well.

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

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

CONCLUSION

AMI is a true surgical emergency. First and foremost, important evidence is a high index of suspicion based on the combination of history of abrupt onset of abdominal pain, acidosis, and organ failure. This clinical scenario should prompt imaging (CTA) in order to establish the diagnosis. In parallel with rapid resuscitation and after careful assessment of the CTA, the patient should be explored to assess bowel viability, re-establish vascular flow, and resect non-viable bowel. Subsequently, the employment of damage control techniques and continued critical care resuscitation is essential. Planned re-assessment of the bowel with further resection or anastomosis and stoma as needed is integral. Close cooperation between acute care surgeons, radiologists, anesthetists, and the vascular surgeons is essential.(56)

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Since the introduction of PTA in 1972, this procedure has become increasingly safe and effective. This very promising technique needs further evaluation, especially in the case of younger patients, before being proposed as the initial treatment in all patients with CMI.(46)

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