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Intestinal Ischemia and Gangrene

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1. Introduction

1.1 Historical background

Antonio Benivieni was the first to describe mesenteric ischemia as early as the 15th century. It was not before the mid 19th century during which the entity was extensively reported and researched after case reports by Virchow and others. The first successful surgical treatment of acute mesenteric ischemia (AMI) was performed by Elliot, who, in 1895, performed a resection of gangrenous portion of the bowel, followed by primary anastomosis of the viable parts. Dunphy correctly hypothesized that mesenteric ischemia was a manifestation of visceral atherosclerosis in the mid-twentieth century. During that time advancements in both the diagnostics and therapeutics of the disease as an entity was in full swing. Heparin was introduced for use in mesenteric venous thrombosis. In the 1950s, a major step in the vascular surgical repair to restore blood flow to ischemic bowel before gangrene occurred was introduced. The first successful embolectomy without bowel resection was performed in 1957. Nonocclusive mesenteric ischemia was first recognized as a subtype of AMI in the 1950s. By 1960, hypercoagulation status was identified as the apparent cause of most cases of mesenteric venous thrombosis and the combination of heparin administration and bowel resection became the standard treatment. In the 1970s, the use of angiography to diagnose and evaluate AMI, as well as the introduction of intra-arterial papaverine infusion, significantly improved the prognosis of patients by allowing early diagnosis and by elimination of residual arterial spasm.

1.2 Incidence

Intestinal ischemia is an uncommon condition presenting particular problems of diagnosis and management. The prevalence of the disease is difficult to establish. In the United Kingdom, approximately 2000 deaths a year are attributable to intestinal vascular insufficiency, with 1883 deaths in 2000. Of these, at least 833 (44%) were classified as acute (834 being unspecified as either acute or chronic). It affects women more than men by a ratio of 2:1. The incidence is rare below forty-five years of age and the majority of deaths occur after the seventh decade. Most cases are caused by emboli (40%-50%), followed by arterial thrombosis (25%-30%), venous thrombosis (10%), and non-occlusive mesenteric ischemia (20%). Mortality is high and has changed little since the 1970s, despite interventional advances.

2. Acute Mesenteric Ischemia

Abrupt interruption or diminution of blood flow to the intestine leads to acute mesenteric ischemia (AMI). The term AMI actually describes a wide spectrum of bowel injury ranging from reversible alterations in bowel function to transmural necrosis of the bowel wall. With better understanding of the clinical syndromes and the pathophysiological basis of AMI a multidisciplinary approach in management has emerged for patients with suspected mesenteric vascular disease. Not only is it important as a primary clinical problem causing high mortality and long-term morbidity, but also it frequently complicates other vascular conditions and operations; occurrence of colonic ischemia after aortic aneurysmectomy or aortoiliac reconstruction and the common association of superior mesenteric and peripheral arterial emboli. Acute ischemia is much more common than the chronic variant and ischemia of arterial origin is much more frequent than venous disease. AMI can result from both arterial and venous causes. The arterial forms include embolism, thrombosis, nonocclusive mesenteric ischemia and cases of focal segmental ischemia resulting from local atherosclerotic emboli or vasculitis. Acute mesenteric venous thrombosis and focal segmental ischemia caused by strangulation of the small intestine or by localized venous thrombosis comprise the venous forms of AMI.

AMI is a life-threatening vascular emergency and requires early diagnosis and intervention to adequately restore mesenteric blood flow and to prevent bowel gangrene and mortality. In spite of adequate understanding of the etiopathogenesis of the events over the past few decades, a high mortality in the range of 60%-80% associated with the disease unfortunately demonstrates a trend of continuous increase [1-5]. The main reason for this outcome is the persistent difficulty in recognizing the condition before bowel gangrene sets in [6,7].

Early clinical picture of AMI is usually nonspecific and in most cases can be characterized by an initial discrepancy between severe abdominal pain and paucity of clinical signs. Clinical examination can not differentiate an ischemic bowel from an infarcted one in most cases. This is because manifestations of acute abdomen, abdominal distention and gastrointestinal bleeding may also masquerade as other abdominal emergencies. Moreover, the risk factors for AMI and the clinical course differ according to the underlying pathologic condition [8,9]. With the progression of bowel ischemia to irreversible gangrene, severe metabolic derangements ensue, leading to a series of events that culminate to multiple organ dysfunction and finally death. The timely use of diagnostic and therapeutic methods to quickly restore blood flow to the bowel is the key to reduce the high mortality associated with this condition [5,8-10].

2.1 Pathophysiology

The splanchnic circulation receives approximately 25% of the resting cardiac output, which increases by about 10% postprandial [11,12]. The mucosal and submucosal layers of the bowel receive about 75% of this blood flow. This is made possible by various factors at the cellular and the molecular level, which interact to regulate the intestinal blood flow, a mechanism that is complex and not adequately understood. The probable mechanisms include the intrinsic (metabolic and myogenic) and the extrinsic (neural and humoral) regulatory systems [12,13]. The metabolic regulatory system causes adaptive changes in splanchnic circulation depending on the amount of oxygen delivery to the tissue rather than

the blood flow. An imbalance between tissue oxygen supply and demand leads to the accumulation of local metabolites (eg, hydrogen, potassium, carbon dioxide, and adenosine), which produces local vasodilatation. On the other hand, through the myogenic regulatory system, arteriolar tension receptors regulate vascular resistance in proportion to the 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 blood flow regulation comprises the activation of vasoconstrictor fibers through α -adrenergic stimulation, which results in small vessel constriction and a decrease in mesenteric blood flow. Of all the types of neural stimulation (cholinergic, histaminergic, adrenergic) that affect the gut, the adrenergic limb of the autonomic nervous system is predominant. Among the humoral factors, various endogenous (epinephrine and nor-epinephrine) and exogenous factors are responsible for affecting and regulating the splanchnic circulation. Various other humoral agents, chemical mediators and cytokines like histamine, nitric oxide, leukotrienes, thromboxane analogues and glucagon can cause splanchnic vasodilatation. Pharmacologic compounds, which decrease splanchnic blood flow, are vasopressin, phenylephrine, and digoxin [14]. Dopamine, however has both vasoconstricting and vasodilating effects. In low doses it causes splanchnic vasodilation, but at higher doses causes vasoconstriction probably by stimulating α -adrenergic receptors. Other molecules like papaverine, adenosine, dobutamine, and sodium nitroprusside also increase mesenteric blood flow. Thus the splanchnic circulation is variously regulated in a complex way. Both endogenous and exogenous neuro-humoral factors, drugs and chemical mediators have significant effects on the vascular flow regulation. It is of interest that the gut tolerates hypoxia to a fair degree and the degree of reduction in blood flow that bowel can tolerate without activating these mechanisms is remarkable.

At any given time, only one-fifth of the mesenteric capillaries are open, and normal oxygen consumption can be maintained with only 20% of maximal blood flow. However, when blood flow decreases below a threshold level, oxygen consumption is reduced and oxygen debt ensues. After an attack of ischemia, when splanchnic blood flow is restored, oxygen extraction increases, providing relatively constant oxygen consumption over a wide range of blood flow rates [12]. Therefore tissue damage due to alterations in mesenteric blood flow is not only due to ischemia but more importantly as a result of cellular injury associated with reperfusion [15,16]. Mesenteric ischemia and reperfusion leads to an increase in microvascular permeability and disruption of the intestinal mucosal barrier, primarily through the actions of activated polymorphonuclear neutrophils producing reactive oxygen species and other inflammatory mediators. The tissue damage induced by the oxygen free radicals is decreased in the presence of antioxidants, xanthine oxidase inhibitors, and free-radical scavenging substances; an observation, which conclusively establishes the role of reactive oxygen species in producing cellular injury during ischemia-reperfusion [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]. Reperfusion injury may be prevented by use of pharmacologic agents such as captopril and also carvedilol, which is an adreno-receptor blocker and a free-radical scavenger. It has also been demonstrated to have an anti-shock and endothelial-protective effect in a rat splanchnic ischemia reperfusion model [19,20].

2.2 Etiological factors

The etiology of acute mesenteric ischemia can be categorized into four specific types based on the cause of reduction of blood flow.

2.3 Arterial embolism

Emboli are the most frequent cause of AMI responsible for approximately 40% to 50% of cases [2,3]. In most situations the source of emboli is from the heart following myocardial infarction, atrial tachyarrhythmias, endocarditis, cardiomyopathies, ventricular aneurysms, and valvular disorders. These conditions can form mural thrombus, which can subsequently dislodge and embolize to the mesenteric arteries [21]. Other rare causes may include post-angiography of the coronary or cerebral circulation.

Most visceral arterial emboli lodge in the superior mesenteric artery (SMA); about 15% lodge at the SMA origin, while about 50% lodge distally at or beyond the origin of the middle colic artery [5,21]. Nearly 30% of these patients give a past history of an embolic event. The diagnosis of SMA embolism can be made intraoperatively based on the distribution of ischemia in the bowel. Since in most cases the origin the inferior pancreaticoduodenal branch is spared, perfusion of the proximal jejunum is maintained leaving the rest of the small bowel ischemic or gangrenous.

2.4 Arterial thrombosis

It is the second common cause of AMI accounting for 25% to 30% of all mesenteric ischemic events [22]. Mesenteric arterial thrombosis occurs commonly near the origin of the SMA in the setting of severe atherosclerotic disease [7]. Its slow progression allows the development of collaterals, which sustain the blood supply to a good extent. Bowel ischemia or gangrene ensues late only when the last remaining visceral artery or an important collateral artery occludes. The extent of bowel involvement is greater than that due to embolism, extending from the duodenum to the transverse colon, and results in high mortality in the range of 70% to 100% [22]. The need for more complex surgical revascularization procedures further complicates the situation.

2.5 Nonocclusive mesenteric ischemia

Non-occlusive disease accounts for about 20% of patients with mesenteric ischemia [8,23]. Its pathogenesis is poorly understood but often involves a low cardiac output state associated with diffuse mesenteric vasoconstriction possibly mediated by vasopressin and angiotensin. The mesenteric vasoconstriction commonly results from myocardial infarction, congestive heart failure, aortic insufficiency, cardiopulmonary bypass, renal or hepatic disease, major abdominal or cardiovascular surgery and vasopressor drugs [10,23]. However clear-cut risk factors may be absent. The resultant low-flow state causes intestinal ischemia and finally gangrene. It is further aggravated by endogenous and exogenous vasoconstrictors, disseminated intravascular coagulation, and cytokine-induced reperfusion injury. Both in-vitro and in-vivo studies have demonstrated the enhanced risk from the use of vasoactive drugs, particularly digoxin, in the pathogenesis of non-occlusive mesenteric ischemia (NOMI) by inducing contraction of splanchnic venous and arterial smooth muscle [14]. It has been observed that the watershed areas of circulation are more vulnerable. Patients under the stress of a surgical procedure or trauma, receiving enteral nutrition in intensive care units may also suffer from non-occlusive ischemia. The mechanism is

probably due to an imbalance created between demand from the enteral feed and supply in presence of decreased mesenteric perfusion. The reported incidence of AMI in these patients is 0.3% to 8.5% and manifests with signs of sepsis, abdominal distention and ileus, which closely mimics the systemic signs of a septic state which may be already present. The mortality rate in such situations is very high, almost up to 56%, which is understandable in the context of delay in diagnosis and the already compromised state of the patient [24].

2.6 Mesenteric Venous Thrombosis

Mesenteric venous thrombosis (MVT) is the least common cause of mesenteric ischemia, representing up to 10 to 15% of all patients with mesenteric ischemia [25]. Previously they were thought of as idiopathic in nature but with the advent of improved diagnostic techniques they are now shown to be secondary to clotting disorders. [26,27]. Segmental involvement of bowel occurs starting from the venous arcades and leading to edema and hemorrhagic necrosis. This is in contrast to the thrombosis caused by the intra-abdominal causes in which the thrombus starts in larger vessels and progresses to involve the smaller venous arcades. The superior mesenteric vein is usually more commonly involved than the inferior mesenteric vein. Hence distal large bowel involvement is much less common. The transition from ischemic to normal segment is more gradual with venous occlusion than with arterial embolism or thrombosis. The mortality rate ranges from 20 to 50 percent [27] and survival depends on age, the presence or absence of co-morbid conditions, delay in diagnosis and surgical intervention. A high rate of recurrence is observed especially within 30 days of presentation [28]. A combination of surgery and anticoagulation has a lesser recurrence rate than with anticoagulation alone. The observation that most of the recurrences occur at the site of bowel anastomosis is highly significant. This reflects inadequate bowel resection or propagation of the residual thrombus, both of which indicate a gradual ischemic transition zone spread over some distance from the area of gangrene, which is vulnerable to further necrosis probably due to the propagation of thrombosis and persistent ischemia.

2.7 Clinical presentation

The signs of AMI overlap considerably with the other common acute abdominal conditions like acute pancreatitis, acute diverticulitis, small-bowel obstruction and acute cholecystitis. Moreover, the clinical features of the underlying pathology causing AMI often coexist. The patients with SMA obstruction due to embolus or thrombus have an acute onset of symptoms and a rapid deterioration in their clinical condition. This is because of the lack of collaterals leading to rapid bowel ischemia and subsequent gangrene, whereas those with NOMI or MVT have a more gradual onset and a more protracted clinical course. The hallmark of AMI is unrelenting abdominal pain. This is frequently associated with nausea, vomiting, and urgent bowel evacuation. The classic picture of a patient with acute mesenteric ischemia involves severe abdominal pain with a paucity of abdominal examination findings and a history of risk factors. Excessive fluid loss in the third space leads to mental confusion, tachycardia, tachypnea, and circulatory collapse.

Non-occlusive mesenteric ischemia occurs most frequently in the critically ill patients who have compromised hemodynamics, are often intubated and on vasopressors. They may also have coexisting severe mesenteric atherosclerosis. With an acute hemodynamic insult in the background, non-occlusive ischemia may precipitate in these patients whose mesenteric

circulation is already compromised. Since they are too sick to manifest the bowel insult, delay of hours to days may occur before it is diagnosed or even suspected. A clinical clue to the problem may be an unexplained worsening in the clinical condition or a failure to thrive or a failure in having the anticipated recovery course. The typical presentation in a conscious patient not having a fulminant course is of diffuse, nonspecific abdominal pain associated with anorexia and diarrhea.

Compared with arterial thrombosis, MVT generates fewer prodromal symptoms with eating. Fever, abdominal distention, and occult blood in stool are the most common findings. Bloody ascites and large fluid losses in the third space may occur, leading to dehydration and hypotension, causing further propagation of venous thrombosis and worsening of the ischemic insult. The final outcome of all causes of mesenteric ischemia is bowel gangrene. When gangrene occurs, the patient develops peritoneal signs, hemodynamic instability, and signs of sepsis with multiorgan failure.

2.8 Diagnosis

As progression of bowel ischemia to frank gangrene is an irreversible event, prompt diagnosis and early treatment are paramount for favorable outcome in AMI. The clinician should have a high index of suspicion if the history and physical examination are suggestive of AMI [22]. Once it is suspected, prompt action to confirm the diagnosis or to exclude it should be initiated and if positive, appropriate treatment should be started without any delay. A differential diagnosis of AMI should always be kept in an elderly patient with a history of any cardiac event, like atrial fibrillation, recent myocardial infarction, congestive heart failure and arterial emboli, having postprandial abdominal pain that is out of proportion to the findings of physical examination. Survival drops rapidly from 50% when the diagnosis is made within 24 hours to 30% or less when the diagnosis is delayed [21].

The laboratory findings are nonspecific although abnormal values with clinical suspicion can help in further evaluation in line of AMI. The common laboratory abnormalities are hemoconcentration, leukocytosis, metabolic acidosis, with high anion gap and increased lactate concentrations. High levels of serum amylase, aspartate aminotransferase, lactate dehydrogenase, and creatine phosphokinase are frequently observed at presentation, but none is sufficiently specific to be diagnostic. Subacute presentation is usually seen with longer disease duration, however when ischemia from mesenteric thrombosis becomes acute, clinical presentation is similar to those who have an acute mesenteric embolic episode [29]. Hyperphosphatemia and hyperkalemia are usually late signs and are frequently associated with bowel gangrene [30]. Special laboratory tests, like serum alpha-glutathione S-transferase and intestinal fatty acid binding protein-I (I-FABP) are under evaluation [31,32].

An abdominal radiograph in AMI is also nonspecific as normal findings may be present in 25% of cases [33-35]. Characteristic radiographic abnormalities, such as thumbprinting or thickening of bowel loops, occur in less than 40% of patients at presentation. Presence of air in the portal vein is also a late finding associated with a grave prognosis [34]. The utility of a plain abdominal radiography helps to exclude other acute abdominal pathologies, such as intestinal obstruction or a perforated hollow viscus. Barium enema is contraindicated in a suspected case of AMI as increased intraluminal pressured due to barium and air can cause further reduction in perfusion, translocation of bacteria, and perforation. Moreover the presence of barium may compromise the findings of subsequent diagnostic tests, like

computed tomography (CT) and angiography. Although abdominal ultrasonography is the first-line investigation for acute abdominal conditions, it is of less help in AMI even when combined with Doppler because the interpretation is often technically limited by the presence of air-filled distended bowel loops. In addition, its sensitivity is limited in detecting more distal emboli or in the assessment of NOMI. The introduction of multidetector row CT (MDCT) is a big step ahead in the evaluation of mesenteric ischemia. The use of non-ionic iodine contrast-enhanced CT scan is rapidly replacing the conventional vascular imaging techniques. MDCT and 3-dimensional imaging characterized by high spatial resolution with a fast acquisition time provide a detailed examination of the small bowel and mesenteric vessels. The sensitivity of MDCT scan ranges from 96-100% and specificity from 89-94% [36-38]. The possible signs of mesenteric ischemia on a CT scan are thickened edematous bowel walls, hematoma, dilated bowel loops, engorged mesenteric vessels, pneumatosis, gas in mesenteric or portal veins, gangrene, and frank arterial or venous thrombosis [39,40]. Interestingly, CT is more sensitive in diagnosing MVT than other types of AMI and is considered the investigation of choice in suspected cases of MVT [41].

Selective angiography is considered to be the gold standard for the diagnosis of acute arterial occlusion with reported sensitivity ranging from 74% to 100% and a specificity of 100% [42]. Early angiography has been the major factor for the decline in mortality in patients with AMI over the past 30 years. Moreover, in the absence of mesenteric ischemia, angiography coupled with a plain abdominal radiograph may reveal the cause of abdominal pain in 25% to 40% of patients. Angiography must be bi-planar, the antero-posterior view demonstrating distal mesenteric blood supply and collaterals, and the lateral aortography for better visualizing of the origins of major visceral arteries that overlay the aorta [29]. Emboli usually lodge just after the middle colic artery where the SMA tapers. The thrombotic disease usually manifests as complete lack of visualization of the SMA origin on the lateral aortogram, associated with prominent collateral vessels in delayed antero-posterior views. Angiography also has the added advantage of being therapeutic by administration of intra-arterial thrombolytic agents for acute arterial thrombosis and intra-arterial papaverine infusion for all types of arterial ischemia. Some drawbacks of the routine use of mesenteric angiography include the technical difficulties in critically ill patients, a relatively high number of false-negatives in early course of the disease and potential renal toxicity of the contrast. Its limited availability in many centers also precludes its use as the investigation of first choice in mesenteric ischemia. MVT is characterized by a segmental slowing of arterial flow along with lack of opacification of the corresponding mesenteric or portal venous outflow tracts. In contrast NOMI has a diffuse involvement with normal venous runoff. NOMI also features a "string of sausages" sign due to multiple narrowing of the major SMA tributaries. Reflux of contrast material back into aorta is seen in venous occlusion and NOMI on selective SMA angiography.

Colonoscopy has been used to diagnose ischemic colitis but fails to visualize much of the small bowel. In addition, endoscopy may not detect subtle ischemic changes where full-blown gangrene has still not set in and these are the subset of patients who have maximum benefit from early and prompt diagnosis. Laboratory finding of elevated white blood cell counts, phosphate, lactate and lactate dehydrogenase levels in peritoneal fluid may be present in mesenteric ischemia but is usually non-specific [25,43]. Radionuclide imaging to identify infarcted bowel has been studied in animals, but clinical studies are lacking. Duplex ultrasonography has been used to detect a significant stenosis (>50%) in the mesenteric

vessels in patients with chronic mesenteric arterial occlusive disease, but its role in AMI seems limited [44-47]. Although magnetic resonance imaging (MRI) has shown promise in peripheral vascular diseases but its reliability in detecting altered flows in the superior mesenteric vessels in chronic ischemia has not been documented in controlled trials [48,49]. Magnetic resonance imaging also takes longer time making its use unlikely in this rapidly progressive disorder in contrast to MDCT and angiography. Peritoneoscopy may also be a useful tool for investigating AMI due to venous thrombosis [27]. Serosanguineous fluid in the abdominal cavity of an older patient with abdominal pain, hemoconcentration, and leukocytosis is strongly suggestive of MVT. These diagnostic methods have limited use because of low negative predictive value. Clinicians must be aware that undue delay caused by insensitive and nonspecific diagnostic techniques may worsen patient outcome.

2.9 Treatment

Treatment should start as soon as the diagnosis is made. The aim is initial resuscitation, prevention of further propagation of block, prevention of reperfusion injury and early restoration of blood flow. Underlying cause, if any, should also be simultaneously treated.

Fluid resuscitation should be started early, as hydration and perfusion is important to prevent progression of clot. Ideally it should be started before angiography and should always be guided with monitoring of the central venous pressure or pulmonary capillary wedge pressure. Inotropes should be added to improve cardiac output but with precaution in NOMI, where they can aggravate bowel ischemia. Vasoconstricting agents and digitalis should be avoided if possible since they can exacerbate mesenteric ischemia. If vasopressors are required, dobutamine, low dose dopamine, or milrinone are preferred as they have lesser effect on mesenteric perfusion. Parenteral broad spectrum antibiotics should also be added to prevent bacterial translocation and sepsis. Systemic anticoagulation should be started with intravenous heparin sodium unless the patient is actively bleeding [50]. The end point of adequate heparinization is to maintain the activated partial thromboplastin time (APPT) to twice the normal value. Pain should be promptly managed, as intense pain can be a trigger for shock.

After initial supportive treatment, efforts should aim at reducing the mesenteric vasospasm. If the diagnosis of AMI is made with angiography, the angiography catheter can be used for infusion of papaverine and other vasodilators. Papaverine (30 - 60 mg/h) use is recommended in cases of arterial embolism or nonocclusive disease and has shown to improve bowel salvage by reducing vasospasm. If mesenteric angiography is not done then infusion of intravenous glucagon at 1 µg/kg per minute and titrated up to 10 µg/kg per minute as tolerated may help to reduce the associated vasospasm [50].

The main goal of treatment in patients with acute mesenteric ischemia is the restoration of intestinal blood flow as rapidly as possible. This may be achieved by medical means, endovascular procedures and by surgery. The traditional treatment of mesenteric arterial embolism has been early surgical laparotomy with catheter-based embolectomy. The appropriate therapeutic option is guided by the etiology of ischemia. A less well established approach is local infusion of thrombolytic therapy through angiographic catheter, which has been successful in a selected number of reports [51-53]. Treatment decision is guided by the presence or absence of peritoneal signs, partial or complete arterial obstruction, and on whether the location of the embolus proximal to the origin of the ileo-colic artery or in more distal branches. Thrombolytic administration is risky as bleeding is the main complication but early infusion within 8 -12 hours of onset of symptoms in the absence of peritoneal signs

and distal emboli has the best outcome for successful reperfusion [54]. Surgical exploration is mandatory if clot lysis is not demonstrated within four hours or there is evidence of progressive ischemia. Long-term management is aimed at the prevention of future embolic events, typically with the use of oral anticoagulation [55].

The treatment of patients with acute mesenteric artery thrombosis is principally surgical. Surgical thrombectomy alone is unlikely to be successful in the long term. Persistence of thrombotic atherosclerotic plaques mandates surgical thrombectomy combined with a revascularization method (arterial reconstruction, bypass or endovascular stenting). In absence of peritoneal signs with angiographic evidence of good collateral blood flow, observation while on heparin anticoagulation may be justified. The use of aspirin in the perioperative period has not been well evaluated, but it may be justified in this setting if the risk of progressive ischemia appears to be greater than the risk of bleeding. After recovery antiplatelet agents such as aspirin may reduce the risk of recurrent mesenteric ischemia [56].

The management of NOMI is essentially pharmacological and is achieved by local selective infusion of vasodilators into the superior mesenteric artery (papaverine, tolazoline, nitroglycerin, glucagon, prostaglandin E and isoproterenol) [57]. The greatest clinical experience is with papaverine administered as a continuous infusion. This approach has resulted in a reduction in mortality rates from 70%–90% to 50%–55% during the last two decades. This is followed by the patient's clinical response to vasodilator therapy from repeated angiography (ranging from 30 minutes to every 24 hours) for evaluation of vasospasm and the decision to stop papaverine infusion. Immediate laparotomy is indicated if signs of acute abdomen develop or the patient condition is worsening [58].

Standard initial treatment for acute mesenteric venous thrombosis consists of heparin anticoagulation even in patients who have gastrointestinal bleeding if the bleeding risk is considered to be outweighed by the risk of intestinal gangrene. Intra-arterial infusion of papaverine during angiography to relieve the concomitant arterial spasm is also an option. Thrombolytic therapy is still experimental and has not a clear indication in superior mesenteric vein thrombosis. Prevention of recurrent venous thrombosis with oral anticoagulation is indicated for at least six months; a longer duration may be warranted if a thrombophilic state has been identified [59].

In the presence of peritonitis or worsening of the condition on non-operative treatment, laparotomy is indicated. Fluoresceine or the Wood's lamp may help better delineation of the necrotic tissue. If a revascularization procedure is intended and resection of necrotic tissue is not imminent, it is recommended to evaluate viability after the flow restoration. After that, efforts should be made in order to minimize the reperfusion injury by administering vasodilators and agents with free radicals neutralizing effect (allopurinol, angiotensin converting enzyme inhibitors).

A second-look laparotomy (after 24–48 hours) is recommended, even after successful primary intervention, because the intra-operative assessment of bowel viability is often inaccurate [60]. The rationale for this second look is based in part on the frequent occurrence of vasospasm after revascularization. Second-look laparoscopy has been advocated as a substitute for second-look laparotomy, but the reliability of this approach remains unproved [61, 62].

2.10 Outcome

Perioperative mortality in patients undergoing revascularization for acute mesenteric ischemia ranges from 44% to 90% [63]. 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. The most important prognostic factor is the early diagnosis. Recurrence is not uncommon, and it carries a poor prognosis. The small proportion of patients that survives massive bowel resection usually develop short-gut syndrome, requiring long-term total parenteral alimentation or small-bowel transplantation.

3. Chronic Mesenteric Ischemia

Chronic mesenteric ischemia CMI or intestinal angina is a clinical syndrome characterized by recurrent abdominal pain and weight loss due to repeated transient episodes of insufficient intestinal blood flow. This is because the increased metabolic demand falls short of the demand in the postprandial period. It occurs in the presence of severe atherosclerotic narrowing of one or more major splanchnic vessel. Approximately 90% of the patients complaining of intestinal angina have at least 2 out of 3 major mesenteric vessels occluded and 50% of them may have critical stenosis in all the 3 vessels [64]. Single vessel involvement is usually insufficient to cause intestinal angina because of the very efficient collateral circulation in the small bowel and colon. The disease is more prevalent in middle and elderly individuals with slight female preponderance in the setting of cardiovascular risk factors. The association with coronary artery, peripheral artery disease and cerebrovascular disease is seen in over 50% of patients.

3.1 Clinical presentation

The presentation of intestinal angina is due to a difference between the need of an increased blood flow required in response to food in the intestine and ability of the splanchnic circulation to meet the same. The magnitude of symptoms is dependent on the extent of atherosclerotic occlusion of the splanchnic artery. Another theory proposed is the steal phenomenon from the intestinal to the gastric circulation in response to food in the stomach [64]. Dull postprandial epigastric pain usually within the first hour after eating is the most common presentation. The intensity of pain can be of variable depending on the demand supply disparity and may occasionally radiate to the back. Heavy meals with high fat content also increase the intensity of pain. This leads to the development of a food aversion (sitophobia) and frequently, patients experience weight loss (80%). Approximately one-third of them have nausea, vomiting, and early satiety [65]. Bloating has also been observed. Constipation with fecal occult blood and ischemic colitis represent hindgut ischemia.

As with AMI the physical findings are mostly non-specific. Weight loss with signs of malnutrition, abdominal tenderness without rebound tenderness during an episode of severe pain and indirect signs of atherosclerotic vascular disease raises the suspicion of CMI. The diagnosis is mainly based on the characteristic clinical picture, the presence of an occlusive lesion in the splanchnic vessels documented by angiography and on the absence of other common causes of abdominal pain. In most cases, patients undergo an extensive workup for obscure chronic abdominal pain before the patient is seen by a vascular surgeon.

3.2 Imaging studies

A plain abdominal radiograph may suggest the diagnosis by showing calcification of the mesenteric vessels. Duplex ultrasonography of the mesenteric vessels is a useful initial test for supporting the clinical diagnosis of chronic intestinal ischemia. This should be

performed by patient in fasted state because mesenteric outflow resistance changes with food intake. It is also technically difficult due to presence of bowel gas but can be accomplished in more than 85% of subjects in the elective setting. The most frequent criterion to identify the celiac artery stenosis is a peak systolic flow of 200cm/sec or more. The test has an overall accuracy of approximately 90% for detection of greater than 70% diameter stenosis or occlusion of the celiac and superior mesenteric arteries when performed in highly experienced hands [66,67]. Angiography is recommended if the results of noninvasive testing are equivocal or if they suggest that intervention is required (establishing the feasibility of revascularization) and remains the diagnostic gold standard. In order to better appreciate the lesions, acquisition must also take bi-planar views [68]. Indirect evidence of CMI may be suggested by the presence of an enlarged arch of Riolan for proximal mesenteric arterial obstruction.

Multidetector CT scans and CT-angiography allow an extensive and noninvasive evaluation of mesenteric lesions. Visualization of the collateral vessels (Riolan's arch, pancreaticoduodenal arch) and of the change in caliber of the ischemic bowel loops (stenosis) may help the diagnosis [69]. Magnetic resonance angiography (gadolinium-enhanced MRA) is highly sensitive for detecting stenosis at or near the origin of the SMA or the celiac artery and avoids the risks of radiation exposure, dye allergy and renal toxicity associated with CT scan [70]. Recent advances in MRA technology have shortened acquisition times, so it is now possible to obtain successive images in the arterial and then the portal phase. MRA can be used as an adjunct to any MR examination. Acute mesenteric ischemia is an emergency in which CT scanning is the most appropriate imaging modality. Conversely, chronic mesenteric ischemia is best examined with contrast-enhanced MRA, which is almost as accurate as digital subtraction angiography [71].

Laboratory tests to evaluate malabsorption that accompany intestinal ischemia such as stool fat content, D-Xylose tolerance and Vitamin B12 absorption are non specific and generally not used in establishing the diagnosis.

3.3 Treatment

The goal of therapy for chronic mesenteric ischemia is revascularization. Taking into account the increased risk for thrombotic events with mesenteric gangrene in these patients, medical therapy alone is reserved for situation when the surgical risk is prohibitive and percutaneous revascularization is not feasible. On the other hand, medical treatment in the form of statins and antithrombotic therapy is given to all the patients except for the ones with contraindications. Oral anticoagulation may also be initiated. Analgesics and intravenous nitrates can be used for temporary pain control.

The means available for mesenteric revascularization are the surgical techniques of flow restoration and the more recently developed percutaneous transluminal procedures i.e., mesenteric angioplasty with or without stenting. As with any other vascular reconstruction patient selection and surgeon's decision combined with operative skill play an important role in overall success. The goals of these interventions are to relieve symptoms, to improve nutrition and to prevent mesenteric gangrene. Although most of the symptomatic patients have two out of three vessels involvement, the revascularization procedure should be considered in patients with documented critical stenosis in at least one major mesenteric vessel and in which other causes for chronic abdominal pain have been excluded. The indication for revascularization surgery in asymptomatic patients is not clear but it may be

considered in asymptomatic patients who are submitted to aortic reconstruction for aortoiliac occlusive disease with significant mesenteric occlusion as they have an increased risk of mesenteric gangrene after surgery [72]. Often bypass grafting of the superior mesenteric artery alone instead of complete vascularization is an effective and durable procedure for treatment of intestinal ischemia [73].

For many years, surgical revascularization has been the treatment of choice for chronic mesenteric ischemia. The techniques used include mesenteric endarterectomy and aortic reimplantation of the superior mesenteric artery. Perioperative mortality ranges from 0–11% and increases to 50% in patients with acute-on-chronic symptoms [74]. Primary graft patency rates at five years ranges from 57% to 69%, and 5-year survival rates ranges from 63% to 77% [75,76]. Postoperative morbidity rates are increased with concomitant aortic replacement, renal disease, and complete revascularization while advanced age, cardiac disease, hypertension and additional occlusive diseases influence overall mortality [77].

Percutaneous transluminal angioplasty, with or without stenting, has become an alternative to surgery for the patients, who are poor surgical candidates and for the patients in whom the diagnosis remains uncertain. The reported success rates are greater than 80% with residual stenosis of less than 50%. Relief of abdominal pain has been achieved in 75–100% patients and half of them experience weight gain. Long term follow up indicates clinical remission in approximately 80% of patients at two to three years [78]. Restenosis and recurrent symptoms occur in 17–50% of patients within the first year, making the short durability of the procedure its greatest limitation [79–83]. There has been no large randomized controlled trial comparing angioplasty, with or without stenting and surgical revascularization. As a general approach, revascularization is the treatment of choice in CMI and surgical revascularization is offered in patients with low surgical risk, while percutaneous angioplasty is preferred for high risk patients [84]. In patients who survive surgical revascularization, the prognosis is excellent and the 5-year survival rates approach 80%, with most patients becoming free of symptoms, resuming normal eating habits and gaining weight.

4. Conclusion

In summary, mesenteric ischemia is a challenging clinical problem with host of causes often diagnosed late and a strong clinical suspicion remains the key to early diagnosis. In the acute form, an aggressive approach should be adopted because the outcome crucially depends on rapid diagnosis and treatment, on the other hand, in chronic cases, the therapeutic decision should be carefully made, balancing risks and benefits, considering other comorbidities. With better understanding of the pathogenesis of mesenteric ischemia syndromes and the availability of a range of diagnostic and interventional techniques and adjuvant pharmacotherapy, an improved outcome may be achieved.

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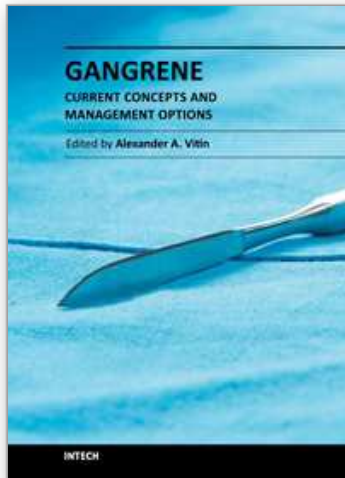
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Gangrene is the term used to describe the necrosis or death of soft tissue due to obstructed circulation, usually followed by decomposition and putrefaction, a serious, potentially fatal complication. The presented book discusses different aspects of this condition, such as etiology, predisposing factors, demography, pathologic anatomy and mechanisms of development, molecular biology, immunology, microbiology and more. A variety of management strategies, including pharmacological treatment options, surgical and non-surgical solutions and auxiliary methods, are also extensively discussed in the book's chapters. The purpose of the book is not only to provide a reader with an updated information on the discussed problem, but also to give an opportunity for expert opinions exchange and experience sharing. The book contains a collection of 13 articles, contributed by experts, who have conducted a research in the selected area, and also possesses a vast experience in practical management of gangrene and necrosis of different locations.

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