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A study of 25 patients on surgical management of acute mesenteric vascular thrombosis

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Abstract

Aim: To study outcome of surgical management of acute mesenteric vascular thrombosis like

- Extent of disease
- Extent of resection
- Post-operative survival

Patients and Methods: This study of 25 cases of diagnosed with acute mesenteric vascular thrombosis (MVT) from September 2016 to October 2018 in department of surgery, civil hospital Ahmadabad.

Inclusion criteria: Patients with acute abdominal pain who is diagnosed as MVT in either on CT-scan or on exploration.

Exclusion criteria: Patient with non-occlusive mesenteric ischemia that did not require laparotomy. Patient with only portal vein thrombosis.

Results: Among 25 patients, 23 patients underwent exploratory laparotomy. Two patients were expired before surgery. In present study, maximum number of patients is in the age group 41-50 years. In 23 patients exploratory laparotomy was done based on their clinical feature with peritoneal signs with CECT-abdomen pelvis finding. Mortality rate was 64% in my study while 9 (36%) patients are in follow-up, so early diagnosis and decision of intervention has saved 36% of patients.

Conclusion: Mesenteric vascular thrombosis is one of the most lethal vascular disorders. So, this type of moribund conditions require urgent use of abdominal CT-Scan and increasing use of anti-coagulative medication improved the outcome in patients. This disease entity is lethal prompt decisions should be taken for patients survival.

Keywords: Mesenteric vascular thrombosis (MVT), surgical resection of gangrenes bowel, patient outcome

Abbreviation: MVT - Mesenteric vascular thrombosis, SMV - Superior mesenteric vein, AMI - Acute mesenteric ischemia, NOMI - Non-occlusive mesenteric ischemia, SMA - Superior mesenteric artery, IMA - Inferior mesenteric artery, IMV - Inferior mesenteric vein, CMI - Chronic mesenteric ischemia, CRP - C-reactive protein, CTA - Computed tomography angiography, CECT - Contrast enhanced computed tomography

Introduction

Obliterative atheromatous disease of the mesenteric arteries usually occurs in elderly individuals who are medically debilitated with generalized atherosclerosis. Despite recent progress in perioperative management and better understanding in pathophysiologic, mesenteric ischemia is one of the most lethal vascular disorders with mortality rates ranging between 50 and 75 per cent. Delay in diagnosis and treatment are the main contributing factors in its high mortality. It is estimated that mesenteric ischemia accounts for 1 in every 1000 hospital admissions in the United States. The prevalence is rising due in part to the increased awareness of this disease, the advanced age of the population, and the significant comorbidity of these elderly patients. Early recognition and prompt treatment before the outcome.

There are four major syndromes of visceral ischemia involving the mesenteric arteries, which include: (i) acute embolic mesenteric ischemia, (ii) acute thrombotic mesenteric ischemia, (iii) chronic mesenteric ischemia, and (iv) non-occlusive mesenteric ischemia. Despite the variability of these syndromes, a common anatomic pathology is involved in these processes. The superior mesenteric artery is the most commonly involved vessel in acute mesenteric ischemia. Acute thrombotic mesenteric ischemia frequently occurs in patients with underlying mesenteric atherosclerosis, which usually involves the origin of the mesenteric arteries while sparing the collateral branches.

The development of collateral vessels is possible when the occlusive process is gradual rather than a sudden ischemic event. In acute embolic mesenteric ischemia, the emboli typically originate from a cardiac source and frequently occur in patients with atrial fibrillation or following myocardial infarction. Non-occlusive mesenteric ischemia is characterized by a low flow state in otherwise normal mesenteric arteries. In contrast, chronic mesenteric ischemia is a functional consequence of a long-standing atherosclerotic process which involves the main mesenteric vessels: the celiac, superior mesenteric, and inferior mesenteric arteries.

Several less common syndromes of visceral ischemia involving the mesenteric arteries can also cause serious debilitation. Chronic mesenteric ischemic symptoms can occur due to extrinsic compression of the celiac artery by the diaphragm, which is termed 'the median arcuate ligament syndrome'. Acute visceral ischemia may occur following aortic operation, due to ligation of the inferior mesenteric artery in the absence of adequate collateral vessels. Furthermore, acute visceral ischemia may develop in aortic dissection which involves the mesenteric arteries. Finally, other unusual causes of ischemia include mesenteric arteritis, radiation arteritis, and cholesterol emboli.

Normal mesenteric circulation

Three vessels constitute the major arterial supply to the intra-abdominal intestine:

1. The celiac axis for the stomach and duodenum,
2. The superior mesenteric artery for the jejunum, ileum, and right and transverse colon, and
3. The inferior mesenteric artery for the left and sigmoid colon.

It is unusual for an embolus to either the celiac axis or the inferior mesenteric artery to produce infarction, because of the obtuse angle of the celiac axis to the aorta, the small size of the inferior mesenteric artery, and the adequacy of collateral inflow to both. The superior mesenteric artery, in contrast, originates at an acute angle, is large and, at least when acute occlusion is proximal, seldom has the sufficient collateral flow to preserve viability of the entire area of intestine that it supplies. This artery is one of the most frequent sites of clinically apparent emboli to visceral vessels.

The first branches of the superior mesenteric artery are small, supplying the duodenum, pancreas, and proximal jejunum. The initial branch of any size is the middle colic artery. The superior mesenteric artery tapers rather rapidly distal to it, terminating in the arteries to the distal ileum. The usual sites at which an embolus lodges are at the origin of the middle colic artery, where it may or may not cause occlusion, and in the main trunk of the artery, within 5 cm distal to the origin of the middle colic artery. Occlusion at the origin of the superior mesenteric artery is almost invariably the result of thrombosis not an embolus.

As with other peripheral emboli, most mesenteric emboli come from the heart. Common sources of emboli are atrial fibrillation, infarction of the ventricle with mural thrombus, and valvular excrescences. A few emboli come from the aortic wall itself, and some are iatrogenic, following aortic surgery or catheterization.

Mesenteric venous thrombosis

When occlusion of the superior mesenteric artery occurs gradually, collateral inflow from the other visceral vessels usually protects the intestine from ischemic injury. When occlusion is acute this collateral supply is not sufficient. Depending on the site at which an embolus lodges, the resulting

ischemic injury may extend from the proximal jejunum to the left transverse colon or, with a more peripheral site of occlusion, may involve only the ileum and right colon. Anatomic variation in collaterals and the presence of fragments of emboli peripherally mean that the ischemic injury may be patchy and variable in its severity, especially in the early stages. However, the central area of supply, the ileum and cecum, usually sustains the most profound injury. A small embolus that enters a branch of the superior mesenteric artery may lead to a segmental infarct or produce no ischemic injury.

The mucosa is the only layer of the intestinal wall to have little resistance to ischemic injury, becoming damaged rapidly by even relatively mild degrees of ischemia. The first gross evidence of damage is submucosal edema; this proceeds to hemorrhage and sloughing of the mucosa, with a small amount of intra luminal bleeding. If the deeper layers remain viable, or if arterial perfusion is restored, the mucosa regenerates: this may take several weeks, during which there is usually diarrhea and sometimes a degree of malabsorption. In a few patients the presence of deep ulcers leads to major hemorrhage from regenerating areas.

The initial response of the muscularis propria to ischemia is spasm. Depending on the severity and duration of ischemia, atony, infarction, and perforation can follow. Full-thickness, irreversible infarction produces a characteristic, foul-smelling, bloody peritoneal exudates. With restoration of arterial perfusion the viability of the outer layers may be preserved and chronic ulceration may lead to the development of strictures, which may take several weeks to become apparent.

Visceral pain is an invariable symptom of mesenteric ischemia. Only in the obtunded or otherwise mentally impaired patient will it not suggest the diagnosis. In its absence, the diagnosis can be confidently excluded. Other symptoms and signs are less definite and depend on the extent, severity, and location of the ischemic injury.

The course of ischemic injury caused by an embolus is also variable. Short of removal of the embolus, the only factor likely to improve perfusion is remission of arterial spasm and the gradual enlargement of collateral vessels. Conversely, the ischemic injury may be worsened by factors that further decrease perfusion, including hypo perfusion due to systemic hypotension and spasm in visceral arteries. Local factors, including edema, hemorrhage, and distension, can also cause extension of the infarct. Although an embolus in the superior mesenteric artery can lead to immediate infarction and perforation within a few hours, this is not the usual course. Many patients will have symptoms for hours or even days before the development of irreversible infarction.

The collateral flow tends initially to lead to a gradient of severity of ischemic injury, the peripheral areas being most spared. There is a tendency for areas of full-thickness infarction to extend to include the marginal areas with the passage of hours. Eventually the initial patchy involvement will usually become an extended area of full-thickness infarction.

Pathogenesis of mesenteric vascular thrombosis

Depending upon the extent and severity of ischaemia, 3 patterns of pathologic lesions can occur.

1. Transmural infarction, characterised by full thickness involvement i.e. transmucosal ischemic necrosis and gangrene of the bowel.
2. Mural infarction, characterized by hemorrhagic gastro-enteropathy (hemorrhage and necrosis). The ischemic effect in mural infarction is limited to mucosa, submucosa and

superficial muscularis, while mucosal infarction is confined to mucosal layers superficial to muscularis mucosae.

3. Ischaemic colitis, due to chronic colonic ischaemia causing fibrotic narrowing of the affected bowel.

Transmural infarction

Ischaemic necrosis of the full-thickness of the bowel wall is more common in the small intestine than the large intestine.

Etiopathogenesis

The common causes of transmural infarction of small bowel are as under:

- i) Mesenteric arterial thrombosis such as due to the following:
 - Atherosclerosis (most common)
 - Aortic aneurysm
 - Vasospasm
 - Fibromuscular hyperplasia
 - Invasion by the tumour
 - Use of oral contraceptives
 - Arteritis of various types
- ii) Mesenteric arterial embolism arising from the following causes:
 - Mural thrombi in the heart
 - Endocarditis (infective and nonbacterial thrombotic)
 - Atherosclerotic plaques
 - Atrial myxoma
- iii) Mesenteric venous occlusion is less common cause of full-thickness infarction of the bowel. The causes are as under:
 - Intestinal sepsis e.g. appendicitis
 - Portal venous thrombosis in cirrhosis of the liver
 - Tumour invasion
 - Use of oral contraceptives
- iv) Miscellaneous causes:
 - Strangulated hernia
 - Torsion
 - Fibrous bands and adhesions.

Morphologic features

Grossly, irrespective of the underlying etiology, infarction of the bowel is haemorrhagic (red) type. A varying length of the small bowel may be affected. In the case of colonic infarction, the distribution area of superior and inferior mesenteric arteries (i.e. splenic flexure) is more commonly involved. The affected areas become dark purple and markedly congested and the peritoneal surface is coated with fibrinous exudate. The wall is thickened, oedematous and haemorrhagic. The lumen is dilated and contain blood and mucus. In arterial occlusion, there is sharp line of demarcation between the infarcted bowel and the normal intestine, whereas in venous occlusion the infarcted area merges imperceptibly into the normal bowel.

Microscopically, there is coagulative necrosis and ulceration of the mucosa and there are extensive submucosal haemorrhages. The muscularis is less severely affected by ischaemia. Subsequently, inflammatory cell infiltration and secondary infection occur, leading to gangrene of the bowel.

The condition is clinically characterised by 'abdominal angina' in which the patient has acute abdominal pain, nausea, vomiting, and sometimes diarrhea. The disease is rapidly fatal, with 50-70% mortality rate.

Mural and mucosal infarction

Mural and mucosal infarctions are limited to superficial layers of the bowel wall, sparing the deeper layer of the muscularis and the serosa. The condition is also referred to as haemorrhagic gastroenteropathy, and in the case of colon as membranous colitis.

Etiopathogenesis

Haemorrhagic gastroenteropathy results from conditions causing non-occlusive hypoperfusion.

These are as under:

1. Shock
2. Cardiac failure
3. Infections
4. Intake of drugs causing vasoconstriction e.g. digitalis, norepinephrine.

Morphologic features

Grossly, the lesions affect variable length of the bowel. The affected segment of the bowel is red or purple but without haemorrhage and exudation on the serosal surface. The mucosa is oedematous at places, sloughed and ulcerated at other places. The lumen contains haemorrhagic fluid.

Microscopically, there is patchy ischaemic necrosis of mucosa, vascular congestion, haemorrhages and inflammatory cell infiltrate. The changes may extend into superficial muscularis but deeper layer of muscularis and serosa are spared. Secondary bacterial infection may supervene resulting in pseudomembranous enterocolitis.

Clinically, as in transmural infarction, the features of abdominal pain, nausea, vomiting and diarrhoea are present, but the changes are reversible and curable. With adequate therapy, normal morphology is completely restored in superficial lesions, while deeper lesions may heal by fibrosis leading to stricture formation.

Ischaemic colitis

Although this condition affects primarily colon in the region of splenic flexure, it is described here due to its apparent pathogenetic relationship with ischaemic injury. Ischaemic colitis is characterised by chronic segmental colonic ischaemia followed by chronic inflammation and healing by fibrosis and scarring causing obstruction (ischaemic stricture).

Grossly, most frequently affected site is the splenic flexure; other site is rectum. Ischaemic colitis passes through 3 stages: infarct, transient ischaemia and ischaemic stricture. However, the surgical submitted specimens generally are of the ischaemic stricture. External surface of the affected area is fusiform or saccular. On cut section, there are patchy, segmental and longitudinal mucosal ulcers. Thus, the gross appearance can be confused with either of the two types of inflammatory bowel disease.

Microscopically, the ulcerated areas of the mucosa show granulation tissue. The submucosa is characteristically thickened due to inflammation and fibrosis. The muscularis may also show inflammatory changes and patchy replacement by fibrosis. The blood vessels may show atheromatous emboli, organising thrombi and endarteritis obliterans.

Diagnosis

As is the case for all types of AMI, prompt diagnosis is paramount if interventions are to be lifesaving. Given the nonspecific presenting symptoms of MVT, a high index of suspicion and willingness to perform diagnostic angiography or

laparotomy had been necessary to make the diagnosis of MVT in past decades.

Perhaps because of improvements in diagnostic testing, a heightened awareness of the condition, and the increased frequency of contrast CT use in emergency department patients with abdominal pain, one author showed that the time from presentation to diagnosis of MVT has decreased from an average of 1 week during the interval 1978-1995 to approximately 1 day during the interval 1995-2003;⁵⁹ other studies suggest that diagnosis within this shortened time frame has led to improved outcome.

Centers that have compared cases of MVT managed during different eras have shown that diagnosis has moved away from autopsy or surgery and towards less invasive modalities.

Laboratory testing

Classically, patients with AMI have leukocytosis, metabolic acidosis, elevated D-dimer, and elevated serum lactate. However, these laboratory tests are highly unspecific and do not exclude the possibility of AMI when negative.

Conventional inflammatory markers

The WBC count and CRP are usually elevated in AMI. However, WBC count was found to be negative in approximately 6-20% of AMI cases, and CRP in 15-25%. The inflammatory markers may be negative in the early phase of intestinal ischemia, for example, in a sudden embolic occlusion of the SMA.

WBC count is elevated earlier in the embolic AMI, while CRP tends to be higher in patients with thrombotic etiology.

Recently, it has been postulated that neutrophil/lymphocyte ratio could play a role in various ischemic conditions such as cardiac events, where high neutrophil counts reflect inflammation and low lymphocytes reflect poor general health and physiologic stress.

The sensitivity and specificity of neutrophil/lymphocyte level greater than 4.5 for AMI were 77% and 72%, respectively.

For procalcitonin, sensitivity ranged between 72-100% and specificity between 68-91%.

Imaging

The utility of plain abdominal radiography in AMI is limited; the signs to look for in the abdominal or chest X-ray are intestinal paralysis, intraperitoneal free gas, and portal venous gas.

Duplex ultrasound is being used for screening purposes for CMI and for follow-up imaging after mesenteric revascularization.

Duplex ultrasound

Visualizing the mesenteric vessels with duplex ultrasound is technically challenging and may be impossible in AMI due to extensive gas within one or two bowel loops. Ultrasound is not recommended for the initial evaluation of patients with ill suspected AMI, because it is time consuming and of limited diagnostic value. In the screening of CMI, the ultrasound assessment is performed during fasting by searching for luminal narrowing and atherosclerotic plaques followed by color

Doppler analysis

The sample volume (1.5 mm) is passed slowly from the aorta to the target vessel searching for elevated peak systolic or end diastolic velocities. 0 Peak systolic velocity cut-off value of 275 cm/s has been widely used for hemodynamically significant SMA stenosis and 200 cm/s for CA stenosis.

The most accurate cut-off values for >50% and >70% SMA stenosis were 2_295 cm/s (87% sensitivity, 89% specificity) and >400 cm/s (72% sensitivity, 93% specificity), respectively.

When assessing the dynamics of the mesenteric circulation, it is important to remember that the blood flow in the mesentery is susceptible to significant variation.

In fasting, approximately 20% of the cardiac output flows through the mesenteric arteries; the CA receives 800 ml/min and the SMA has a basal flow of 500 ml/min.

Postprandial, the CA flow increases approximately 30% while the SMA flow increases by more than 150%.

Furthermore, increased velocity in an unobstructed mesenteric artery may be consequence of a concomitant obstruction elsewhere in the splanchnic circulation.

A hemodynamically significant stenosis in either CA or SMA has been shown to increase flow velocities in the other unaffected artery especially when significant collaterals have developed.

Computed tomography^[10]

CT, or more precisely, CT angiography (CTA) has replaced conventional angiography as the gold standard diagnostic imaging modality in AMI.

Magnetic resonance angiography has a high sensitivity and specificity for detecting proximal obstruction of the SMA and the CA, but it has limited value in the evaluation of distal occlusions in the mesentery.

Diagnostic accuracy

Contrast enhanced CT with modern multi detector devices has proved to be the method of choice for detecting vascular pathology and associated intestinal changes in AMI.

The sensitivity and specificity of CT ranged between 89-100%. Contrast agent is used to highlight (i.e. enhance) specific tissues such as blood vessels and bowel wall during the abdominal CT. Contrast enhanced CT performed in both arterial and portal venous phases (biphasic protocol) is currently recommended as the first-line imaging technique in AMI.

The venous phase is required for the assessment of intestinal and solid organ perfusion pattern and other pathology of the abdomen, and the arterial phase enables more accurate detection of vascular pathology.

A single-phase image acquisition with biphasic contrast medium injection is called the "split-bolus" protocol, which enables diagnostic enhancement of arteries, veins, parenchymal organs, and bowel wall in a single series of images.

Mesenteric venous thrombosis in superior mesenteric vein. Absence of flow in superior mesenteric vein with ring enhancement is a sign of acute thrombus^[11].

However, in most cases of unknown etiology, the routine CT of the acute abdomen will be performed in venous phase alone, because in practice, AMI is not too often suspected prior to imaging.

Triphasic CT (constituting of unenhanced, arterial, and venous phases) has also been suggested as the standard imaging protocol in AMI.

The reasons for obtaining unenhanced CT are detection of submucosa hemorrhage and assessment of bowel wall enhancement comparing unenhanced and contrast enhanced images.

However, submucosa hemorrhage has low sensitivity for AMI and poor inter observer agreement. Furthermore, considering the larger radiation dose in Triphasic imaging and the fact that abnormal enhancement can be assessed by using normally

enhancing bowel as an internal reference, obtaining unenhanced CT is not required for the diagnosis of AMI.

Portal vein (a, coronal view; red arrow) and superior mesenteric vein thrombus [11].

Acute renal insufficiency has been reported in 52-72% of patients with acute SMA occlusion prior to imaging. The typical amount of non-ionic iodinated contrast material required for the abdominal CT is 120 ml.

It may be necessary to reduce the amount of contrast medium in patients with chronic renal insufficiency. However, performing only unenhanced CT because of the fear of contrast induced nephropathy will risk delaying the diagnosis, and the prognosis of patient will be worse if the patient is, indeed suffering from AMI.

Signs of bowel ischemia

Decreased or absent bowel wall enhancement is probably the most specific finding in acute intestinal ischemia (96% specificity), but it has been reported having a low sensitivity (18-62%). Increased enhancement of the bowel wall (33% sensitivity, 71% specificity) is caused by local hyperemia and hyper perfusion, for example, during reperfusion conditions following occlusive AMI or NOMI.

Impaired venous drainage of contrast medium in MVT may also cause hyper enhancement of the bowel wall. The normal bowel wall thickness in CT ranges from 3 to 5 mm, but it is strongly dependent on the degree of bowel distention or contraction.

Bowel wall thickening is the most common but also the least specific finding in AMI associated often with ischemic colitis and reversible intestinal ischemia. It is caused by edema or inflammation in the submucosa layer. Hemorrhage may also appear as hyper-attenuation of the bowel wall. If absent, bowel wall thickening does not exclude AMI, and in case of Trans mural infarction, the bowel wall may become paper thin.

Pneumatics intestine is and Porto-mesenteric venous gas have been reported in 6-28% and 3- 14% of cases with AMI, respectively. The specificity of these findings approach 100% for acute intestinal ischemia, but pneumatics does not yet prove irreversible bowel damage. The concomitant presence of pneumatics and Porto-mesenteric gas exhibit 83% specificity but only 17% sensitivity for Tran's mural bowel infarction.

Luminal dilatation is a common but unspecific CT finding in AMI and may result either from intestinal paralysis or from Trans mural bowel necrosis. The dilated bowel loops are often filled with gas and fluid. Bowel dilatation has been reported in approximately half of AMI patients.

Mesenteric fat stranding and ascites are nonspecific findings reported in 68-69% and 49-88% of AMI patients, respectively. Intra-abdominal free gas is a sign of intestinal perforation or associated with extensive pneumatics.

Arterial embolus or thrombus, or MVT are not direct signs of bowel ischemia but represent the etiology in AMI. Embolus or thrombus within mesenteric artery suggests an acute arterial occlusion.

Mesenteric atherosclerosis without sign of thrombotic occlusion may be an incidental finding or associated with CMI. However, CMI may eventually become acute, and thrombotic clot may not always be visible in CT. Intra luminal filling defect and engorgement of the SMV, venous collaterals and mesenteric fat stranding are indications of acute MVT.

In NOMI, the vascular findings are usually absent. In some cases, irregular narrowing (vasospasm) of the SMA, IMA, and mesenteric arterial arcades may be visible in CT but are difficult to detect.

Diminutive aorta and inferior vena cava in CT may reflect systemic shock in NOMI.

In the Malmo autopsy study, synchronous embolism (arm, leg, brain, abdominal viscera) was found in 68% of patients with SMA embolism but in none of the patients with SMA thrombosis.

Synchronous emboli within the visceral arteries are not always visible in the abdominal CT, but may be detected as a solid organ infarction in the kidneys, spleen or liver. Solid organ infarctions have been detected in 15-36% of patients with AMI, however, not exclusively in patients with embolism but also in patients with thrombosis.

Ultimately, the diagnosis of AMI is established by evaluating the patient's clinical condition, the laboratory findings, and the vascular CT findings together with the associated intestinal CT findings.

Management

Medical management

The initial management of AMI should be started with fluid resuscitation before the CT scan

Electrolyte imbalances (usually hyperkalemia) and anemia should be corrected. Bowel ischemia causes bacterial translocation through the bowel wall and broad-spectrum antibiotics are necessary to protect against bacteremia and sepsis.

If there are no contraindications, LMWH should be started to inhibit further development of thrombosis. If vasopressors are required, dobutamine should be preferred to norepinephrine. Post-procedural medical management includes anticoagulation in patients with arterial embolism or MVT for a minimum of six months, or lifelong depending on the underlying cause.

In 1946, heparin was first used postoperatively in a patient who underwent resection for intestinal infarction from MVT.

In 1965, Naitove performed a retrospective meta-analysis of anticoagulant use in 33 patients with MVT and showed that mortality was 50% for those who did not receive anticoagulants compared with 0% for those who received postoperative anticoagulation; this benefit has been demonstrated repeatedly in several other retrospective series.

Immediate heparinization upon diagnosis of MVT — intraoperative and even for patients who present with bleeding because of MVT-induced ischemia — is currently accepted as standard therapy.

Heparin has been shown to prevent recurrence of thrombosis after intestinal resection (14% versus 26% in one study; 0% versus 19% in another), and to be associated with lower mortality when recurrence does occur (22% versus 59%). In anticoagulated patients not undergoing surgery, most thrombosed veins will partially or completely recanalize over time.

In one study, 80% of anti-coagulated patients with mesenteric and/or portal vein thrombosis showed with vascular recanalization over a mean follow-up time of 5 months compared months of anticoagulation with warfarin, whereas lifelong therapy is advised for those whose thrombosis is believed to be idiopathic or who are expected to have persistent hypercoagulability because of thrombophilia or certain systemic conditions.

Systemic intravenous streptokinase and tissue plasminogen activator (tPA) have been successfully used in this setting, although most recent reports employ trans catheter routes of such therapy.

When acute large vessel thrombosis is identified, thrombectomy

is useful, as mechanical thrombectomy (with or without local thrombolysis) may result in a rapid and durable venous patency without the need for prolonged thrombolytic therapy.

Thrombectomy, trans-venous thrombolysis, or both, have been described via percutaneous Trans hepatic, Trans femoral and trans-jugular approaches; the Trans jugular approach is preferred in the setting of ascites.

For patients with thrombi in smaller veins, placement of an SMA catheter can be considered for thrombolysis via the trans-arterial route.

One author's experience suggested a worse outcome when thrombolytic therapy was initiated more than 24 hours after presentation. The largest two thrombolytic case series reported to date include a total of 28 patients with acute superior mesenteric venous thrombosis, of whom 82% achieved partial or complete lysis and 87% showed symptom improvement.

No patient required intestinal resection, although two patients died of refractory thrombosis in the setting of sepsis, and serious bleeding occurred in several patients.

No recurrence or late mortality was noted in either study.

Although no large or well-controlled studies exist to guide recommendations, aggressive intravascular therapy should be considered as an adjunct to anticoagulation in patients with acute MVT without infarction (or perhaps for poor surgical candidate seven with infarction), In centers where there are sufficient technical expertise and experience. Surgery should be considered in those who are not candidates for anticoagulation alone the post-procedure medication after EVT for AMI caused by vascular disease is comparable to that of other peripheral vascular interventions including acetylsalicylic acid, clopidogrel and statin.

It is generally accepted that dual anti platelet therapy should be continued for a minimum of 6-8 weeks after SMA Stenting, although early stent thrombosis in such a high-flow artery is rare.

The long-term patency is generally known to be significantly lower after PTA/S than after surgical bypass of the SMA. One suggested follow-up protocol included clinical examination and duplex ultrasound every 6 months during the first year and annually thereafter.

However, the evaluation of in-stent re stenosis with duplex ultrasound is demanding and subject to error and therefore, CTA might be preferred, at least in case symptoms recur. The recurrence of symptoms after PTA/S almost invariably means re stenosis, which can be treated percutaneous in most cases.

Surgical management

- The cornerstone of therapy of patients with AMI-MVT is removal of frankly gangrenous bowel, support, and prevention of clot propagation.
- After adequate resuscitation, appropriate investigations when diagnosis is confirmed and decision for emergency exploratory laparotomy is made then Patient is prepared, Blood Cross match is sent for need of transfusion.
- Patient & relatives counseled, explained & after Consent, under general Anesthesia.
- Midline laparotomy skin incision is made, subcutaneous tissue cut, linea Alba cut.
- Peritoneum held between pair of Kocher'shemostatics artery forceps, checked for absence of contents, cut with tissue cutting scissor, thorough exploration of abdomen was done.
- Free fluid drained, thorough examination of peritoneal cavity, organs, small & large Bowels done & appropriate

procedure is done depending upon extent of pathology i.e., Resection Anastomosis, Resection & Loop or Double barrel ileostomy, Resection & Jejunostomy on one side & Jejunostomy or Ileostomy another side. Specimen is sent for Histopathological examination.

- Thorough adequate peritoneal lavage is given.
- Proper Abdominal Closure is done in layers—from within outwards peritoneum, Posterior rectus sheath, Rectus muscle, Anterior Rectus Sheath, subcutaneous tissue & Skin.

Gangrenous changes after MVT

Embolism

Acute SMA embolism can be treated with open surgical embolectomy or with endovascular approach. The surgical approach to the SMA is performed via upper midline incision.

The SMA is exposed below the transverse mesocolon, and balloon embolectomy with no. 2 or 3 Fogarty catheters performed through a transverse arteriotomy.

In the past, intra-arterial thrombolysis was the primary mode of Endovascular treatment (EVT) in SMA embolism. The development of catheters suitable for endovascular aspiration has reduced the need for thrombolysis, and today, thrombolysis has been superseded by mechanical aspiration.

Gangrenous small bowel



Fig 1: Gangrenous changes after MVT



Fig 2: Resection segment of small intestine after MVT

However, catheter-directed continuous infusion of thrombolytic agent can still be used as an adjunct to clear out residual thrombi or distal embolization after endovascular mechanical aspiration.

In a septic patient with peritonitis, laparotomy is the primary intervention and surgical embolectomy is the fastest way to achieve intestinal reperfusion during emergency laparotomy.

In stable patients, endovascular mechanical aspiration has been technically successful in 73-100% of cases with 10-86% bowel

resection rate and mortality rate ranging between 10-33%. These results compare favorably or at least equally with the 30-49% mortality rate reported for open embolectomy in AMI.

If EVT is successful and the symptoms resolve quickly, laparotomy can be avoided in more than half of the patients with SMA embolism. However, if the abdominal pain persists after technically successful EVT, laparotomy should be performed without delay. Failure to resect, irreversibly damaged bowel will lead to sepsis and even larger bowel necrosis.

Thrombosis

The surgical approaches to revascularization in thrombotic (atherosclerotic) occlusion of the SMA are:-

- Ante grade bypass
- Retrograde bypass
- Thromboendarterectomy
- SMA Reimplanation
- Hybrid opens mesenteric stenting.

Patients with AMI are critically ill, and therefore, the procedure time to be as short as possible.

In emergency conditions, retrograde bypass to the SMA with a prosthetic graft is the fastest to perform. An iliac artery to SMA bypass using a C -shaped prosthetic graft has been shown to perform as well as an ante grade bypass in CMI.

Bypass to the SMA alone is strongly preferred in AMI as opposed to multi-vessel bypass grafting. Most authors prefer autologous vein in AMI due to the fear of bacterial contamination of the abdomen, while synthetic grafts seem to work very well in CMI due to the larger diameter of the graft and the excellent outflow of the SMA.

The endovascular approach to the treatment of thrombotic AMI is percutaneous Tran’s luminal angioplasty (PTA) with stenting (PTA/S) through femoral or brachial access.

Materials and Methods

This study was conducted at the department of General Surgery, Civil hospital, Ahmadabad, 25 cases of diagnosed with acute MVT from September 2016 to October 2018 in emergency that fell in inclusive criteria.

Patients were followed from the time of admission, per operative period, till the time of discharge or death, with pre-operative routine blood investigations, imaging (USG, CECT when required). Detailed Performa was developed to record information on demographic data, admission details, thrombus location, provisional diagnosis, treatment, surgical intervention, hospital stay, etiology were assessed, present history findings, past medical history, the operating details and follow-up done as per protocol.

Inclusion criteria

1. Patient presented with acute abdominal pain that is diagnosed as MVT in either on CT scan or on exploration.

Exclusion criteria

1. Patient with non-occlusive mesenteric ischemia. Who did not require laparotomy?

2. Patient with only portal vein thrombosis.

Mesenteric Arterial Thrombosis is defined as

- a) A thrombotic occlusion with superimposed calcified stenosis of the SMA, or
- b) A chronic occlusion or severe stenosis (70-99%) of the SMA, together with significant atherosclerotic obstruction of the CA or IMA in a patient with acute episode of prolonged symptoms and other findings consistent with the diagnosis of AML.

Mesenteric Venous Thrombosis is defined as acute abdominal pain and related symptoms together with major thrombosis of the SMV and surrounding mesenteric oedema indicating acute thrombosis. Patients with isolated portal vein thrombosis were not included. Venous mesenteric ischemia is defined as acute or subs acute MVT with CT findings suggesting intestinal ischemia (such as bowel wall thickening with ascites or abnormal bowel wall enhancement) or advanced intestinal injury requiring bowel resection. Patients presenting with acute abdomen suggesting mesenteric ischemia were evaluated with CECT Abdomen & pelvis after hemodynamic stabilization. Patients with peritoneal signs and unstable or stable after optimization of hemodynamic parameters underwent exploratory laparotomy.

On exploration if necrosis was found, bowel resection was performed, depending on intra-op findings appropriate surgical procedures done.

Like

1. Local resection +end to end anastomosis
2. 2 Resection + intestinal stoma

Resection + Jejunostomy on one side + Ileostomy/Colostomy on another side. All patients were treated with enoxaparin, a low molecular weight heparin (LMWH) (100 mg/kg, twice daily), after diagnosis with MVT until an oral anticoagulant (warfarin) could be administered, if indicated.

Once a surgical intervention seemed unlikely to be necessary, oral anticoagulation therapy was started with the aim of maintaining an international normalized ratio (INR) between 2.0 and 3.0.

Anticoagulation treatment was continued throughout each patient's follow-up and patients were followed up every three months.

Result

Table 1: Age

Age group (Yrs.)	No. of patient	Present study (%)
<31	1	4%
31-40	3	10%
41-50	9	34%
51-60	6	30%
>60	5	30%

In my study, maximum number of patients is in the age group of 41-50 years and means age is 52.5 year ^[12].

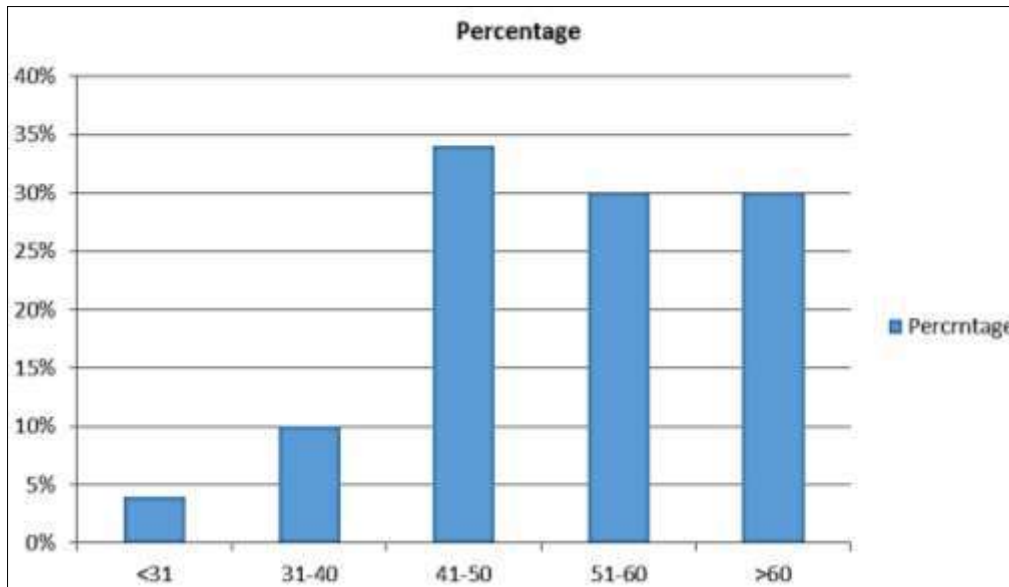


Fig 1: Age

Table 2: Sex

Sex	No. of patient	Present study
Male	18	72%
Female	7	28%

So, in my study 72% of male patients are involved so this disease is more common in male patients [12].

Table 3: Clinical features

Clinical features	No. of patient	Present study (%)
Abdominal pain	25	100
Nausea/vomiting	23	92%
Diarrhea	1	4%
Constipation	3	52%
Per rectal bleeding	2	8%
Abdominal distension	18	72%
Absent bowel sounds	24	96%

Duration of abdominal pain is ranging from the 2 days to 30 days; average is 7 days in my study, which is the most common symptoms followed by Nausea/Vomiting [13], while almost all patients had absent bowel sounds.

Table 4: Co-morbidities

Co morbidities	No. of patients	Percentage (%)
DM	07	28%
HTN	08	32%
IHD	04	16%
TB	04	16%
Total	23	92%

In my study most common co-morbidity was Hypertension followed by DM signifies involvement of vessels in MVT patients with co morbidities.

Table 5: Addiction

Addiction	No. of patients	Percentage (%)
Smoking	10	40%
Tobacco chewing	05	20%
alcohol	05	20%

Most common addiction was Bidi/Cigarette smoking involving

40% of patients followed by alcohol & tobacco chewing [13].

Table 6: Abdomen X-Ray finding

	No. of patients	Percentage (%)
Multiple Air-fluid levels	17	68%
Gaseous bowel or Normal	8	32%
Free gas	0	0%

68% patients in this series had multiple air fluid levels in Abdominal X rays done in emergency, whereas 32% patients had Gaseous or normal findings in X-ray.

Table 7: Abdomen-pelvis

	No. of patients	Percentages (%)
Patients with CECT	20	80%
Patients without CECT	05	20%
Total	25	100%

Majority of patients in my study were with CECT ABDOMEN PELVIS being 80% & pre-operatively helped in confirming the diagnosis while 5 patients were taken for exploration on clinical basis only.

Table 8: Vessels involved

	Number of patients	Percentages
CA	01	04%
SMA	13	62%
SMV	08	32%
IMA	00	--
IMV	00	--
PORTAL VAIN	03	12%

In my study, most common major vessel involved was SMA in 62% of patients followed by SMV involvement in 32% of patients which is similar to other studies. None of the patients had IMA, IMV involvement.

Table 9: Extent of bowel resection/disease

Length of bowel resection	No of patients	Percentages
<100 Cms	19	76%
>100 Cms	06	24%

In my case series, 76 percentage of patients required resection of bowel less than 100 Cms, out of that 36% patients out of those with resection more than 100 Cms two patients got expired and rest 4 patients fortunately survived. Extent of anastomosis is based on gross examination of viability of bowel; minimum length of bowel resected is 15 cms to maximum length of bowel resected is 190 Cms. Re-laparotomy was required in one patient only.

Table 10: Management, outcome and follow-up

Emergency exploratory laparotomy	23
Primary anastomosis	17
Fj/stoma	6
Jejunostomy on one side + ileostomy/Colostomy on other side	02
Need for relaparotomy	1
Hospital stay	Up to 46 days
Expired	16
Follow up	9

In 23 patients exploratory laparotomy was done based on their clinical features with peritoneal signs with CECT Abdo + pelvis findings Out of these patients, primary end to end anastomosis is done in 17 patients & double barrel stoma is formed in 6 patients based on intraperitoneal findings & surgeon's individual decision.

Extent of anastomosis is based on gross examination of viability of bowel; minimum length of bowel resected is 15 Cms to maximum length of bowel resected is 190 cms. Re-laparotomy was required in one patient only. Total 16 patients are expired. Out of these 9 pt expired in early postoperative period. Total 09 patients were discharged with regular follow-up.

Discussion

- In my study, maximum number of patients are in the age group of 41-50 years and mean age is 52.5 year ^[12].
- In my case series, 72% of male patients are involved so this disease is more common in male patients who are found common in almost all studies.
- In my study, 100% patients have abdominal pain as the most common symptom followed by Nausea/Vomiting which is seen in all Comparative studies while almost all patients had absent bowel sounds.
- In my study most common co-morbidity was Hypertension (32%) followed by DM (28%). The most common addiction was Bidi/Cigarette smoking involving 40% of patients followed by alcohol & tobacco chewing ^[13].
- 68% patients in this series had multiple air fluid levels in Abdominal X rays done in emergency whereas 32% patients had Gaseous or normal findings in X-ray. Not a single patient had free gas under diaphragm.
- Majority of patients in my study were with CECT ABDOMEN PELVIS (80%) pre-operatively helped in confirming the diagnosis while 5 patients (20%) were taken for exploration on clinical basis only.
- In my case series, 76 percentage of patients required resection of bowel less than 100 Cms out of those 10 patients (40%) expired, out of those with resection more than 100 Cms two [8%] patients got expired and rest 4 [16%] patients fortunately survived.
- Extent of resection is based on gross examination of viability of bowel. Minimum length of bowel resected is 15 cms to maximum length of bowel resected is 190 Cms. Re-laparotomy was required in one patient only. I

- In 23 patients exploratory laparotomy was done, Out of these patients, primary end to end anastomosis is done in 17 (73.91%) patients & double barrel stoma is formed in 6 (26.09%) patients based on intraperitoneal findings & surgeon's individual decision.
- Total 16 patients (64%) were expired. Out of these 9 (36%) patients expired in early postoperative period. Total 09 (36%) patient were discharged with regular follow-up.

Conclusion

- In my study, Acute MVT is more commonly seen in male population with more preponderance in middle aged & Smoking has significant role in development of MVT.
- Surgical management depends on peritoneal signs and patient's overall conditions. Surgical resection is a procedure of choice and Prompt laparotomy should be done for patients with peritonitis.
- In my study, in 23 patients exploratory laparotomy was done based on their clinical features with peritoneal signs with CECT Abdo + pelvis findings, mortality rate was 64% in my study while 9 (36%) patients are in follow-up so early diagnosis & decision of intervention has saved 36% of patients which is significant survival.
- Post-operative survival largely depends upon extent of disease, co-morbidities, level of Care & supportive treatment.
- Mesenteric vascular thrombosis is one of the most lethal vascular disorders. So, this type of moribund conditions require urgent use of abdominal CT-Scan and increasing use of anti-coagulative medication improved the outcome in patients.
- This disease entity is lethal. Prompt decisions should be taken for patient's survival.

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