A CLINICAL STUDY ON MESENTERIC VASCULAR OCCLUSIVE DISEASE

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CERTIFICATE

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DECLARATION

I solemnly declare that the dissertation titled "**CLINICAL STUDY ON MESENTERIC VASCULAR OCCLUSIVE DISEASE"** was done by me from September 2011 to August 2012 under the guidance and supervision of **Prof .Dr. P.V.VASANTHA KUMAR, M.S.**

This dissertation is submitted to the Tamilnadu Dr. M. G. R. Medical university towards the partial fulfillment of the requirement for the award of M.S. Degree in General Surgery (Branch I)

Place: Dr. SENTHILKUMAR

Date:

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A CLINICAL STUDY ON MESENTERIC VASCULAR OCCLUSIVE DISEASE :

Abstract:

Acute mesenteric ischemia is a condition that is very difficult to diagnose and is thought of atlast juncture with most cases resulting in increased morbidity & mortality. Smokers and known peripheral vascular occlusive disease patients with symptoms of post prandialangina are mandatory to screen with colour doppler and Further confirmed by CT Angiogram for early diagnosis of mesenteric ischemia. Due to obligue origin of superior mesenteric (SMA) artric from visceral aortic segment, SMA is most common mesenteric Vessel to undergo disease involvement. Hence high index of suspicion is necessary for earlier diagnosis and decreasing morbidity and mortality.

Keyword :

Mesenteric ischemia, superior mesenteric artery thrombosis, colour doppler, CT Angiogram.

CONTENTS

ABBREVATIONS:

AMI – Acute Mesenteric Ischemia

CMI – Chronic Mesenteric Ischemia

CI – Colonic Ischemia

SMA – Superior Mesenteric Artery

IMA – Inferior Mesenteric Artery

SMV – Superior Mesenteric Vein

IMV – Inferior Mesenteric Vein

MVT – Mesenteric Venous Thrombosis

INTRODUCTION:

Acute Mesenteric Ischaemia (AMI) is a condition that is very difficult to diagnose and is thought of at the last juncture with most cases resulting in increased morbidity and mortality. Though conditions like volvulus, intussception, hernias that are strangulated and intestinal obstruction in later stages can result in gangrene of the intestine, their number is minimal when compared to acute mesenteric ischemia as an inciting event.

The clinical presentation and the patients risk factor profile gives an idea of the underlying etiological process with severe abdominal pain out of proportion to the signs. . Patients with history of recent MI, atrial fibrillation, congestive heart failure,or peripheral arterial emboli are at a risk for an Superior Mesenteric Artery embolus.

Because of its oblique origin from the visceral aortic segment, SMA is the most common mesenteric vessel to undergo embolism. Thromboemboli tend to lodge in the proximal SMA, just beyond the few jejunal branches as the Superior Mesenteric Artery tapers. A minority (15%) may lodge at the SMA origin, but 50% lodge distal to the middle colic artery, creating a classic pattern of ischemia that spares the first portion of small intestine and the ascending colon. Atheroembolic emboli, in contrast, tend to be smaller and therefore lodge in the more distal mesenteric circulation. As a result, these emboli are likely to affect bowel perfusion less often and in more localised areas. Mesenteric venous

thrombosis (MVT) is rare and accounts for 5 to 15% of all acute mesenteric ischaemia.

Severe abdominal pain is the most common presentation of AMI. This can be sudden and dramatic in a previously well and asymptomatic patient (embolism), recurrent abdominal pain precipitating into unrelenting pain(thrombosis) or vague colicky abdominal pain which is progressive over 1 to 2 weeks (MVT). Copious vomiting ,diarrhoea and urge to defeacate are the associated symptoms. In the unconscious patient, abdominal distension, gastrointestinal bleeding,occult sepsis (leucocytosis or fever) and worsening metabolic acidosis may be the presenting features.

Physical examination may reveal non specific abdominal distension and non specific abdominal pain. Peritonism or blood in the stool or vomitus indicates advanced gastrointestinal ischaemia with likely intestinal gangrene and is mostly a recognised late feature. Mortality for AMI remains high, and patients requiring extensive gut resection are unlikely to survive. Patients surviving intestinal resection may develop short gut syndrome. The prognosis dramatically improves if revascularisation can be achieved prior to intestinal infarction.

The key to the successful management depends on the surgeon's ability to suspect the diagnosis, pursue appropriate investigations and institute

aggressive treatment. The mortality remains high due to difficulty and delay in the diagnosis . Delay in diagnosis contributes directly to ischaemic damage.

With the background of four to eight cases of bowel gangrene presenting with acute intestinal obstruction in a month in our hospital with unknown etiologies, a study to identify the cause and ways to improve the morbidity and mortality of the diseased population remained vital.

- **AIMS AND OBJECTIVES:**

a. to study the factors which lead on to mesentric vascular catastrophe.

b. to diagnose the disease early in patients with high risk factors before the onset of bowel ischemia by various modalities.

MATERIALS AND METHODS:

Materials:

Study data collection formats

Paper

Pencil

Personnel:

Radiologist

Surgeons of all Units in Department of General Surgery

Nursing and Theatre staff

Study population:

All the patients admitted in Coimbatore Medical College Hospital with the following criteria:

- Chronic smokers complaining of chronic abdominal pain especially post-prandial pain, food fear and weight loss not diagnosed by other modalities.
- Chronic abdominal pain with known peripheral vascular disease (atherosclerosis)

- Women taking contraceptive pills with chronic abdominal pain and hypercoagulable states.
- Patients with coronary heart disease (IHD) with or without bloody diarrhoea.
- Aged greater than sixty years with atherosclerotic changes.
- Patients presenting with acute intestinal obstruction due to gangrenous bowel.
- Patients admitted with thromboembolic manifestations.

Pregnant women and patients presenting with intestinal obstruction due to mass lesion or intraluminal obstruction were excluded from the study.

A total of 57 patients were part of the study over a period of one year.

Blood samples were collected from all the patients with the above criteria and complete hemogram, blood sugar urea, serum creatinine, serum electrolytes, serum lactate, lipid profile, PT INR were estimated. Chest x ray and ECG was taken.

Patients who were included in the study were subjected to Doppler ultrasound and a further confirmatory CT angiogram was done when mandatory for eluding a diagnosis of acute mesenteric vascular occlusion. Intra operative findings were noted for all the cases which were operated were taken and biopsy of the suspected vessel that was occluded was taken.

Further anticoagulant therapy using heparin was started for cases post operatively for patients having evidence of thrombosis intra operatively and patients with proven mesenteric vascular occlusion. Thrombectomy was done for patients intra operatively when the need calls.

- **STUDY PROTOCOL:**

1. Disease diagnosed in high risk population could be managed conservatively by life style modification like cessation of smoking, BPcontrol,lowering HbA1C<7%,statins, antiplatelet therapy,angioplasty with or without stenting.

2. Disease diagnosed in acute episodes could be managed by operative interventions like thrombectomy or embolectomy with resection and anastomosis of bowel with postoperative statins and antiplatelet therapy.

REVIEW OF LITERATURE:

Hypoxia of the small bowel and colon invariably lead to gastrointestinal ischemia and infarction (Tables 1 and 2). Ischemic bowel disease occurs as a result of insufficient vascular supply to the gut in acute or chronic episodes thus resulting in chronic small bowel and colonic ischemia (CI) and infarction. Vascular compromise of the gut is a complex multifaceted condition that depends on the (1) systemic circulatory state (2) extent of anatomic or functional compromise of the vasculature, (3) number of vessels affected and their calibre (4) vascular bed response to diminished perfusion (5) extent of collateral circulation, (6) time duration of ischemic insult, and (7) requirements of the involved bowel segment.[1–12] Patients who have intestinal ischemic disorders mostly present with pain abdomen and other nonspecific symptoms (nausea, diarrhoea, bloating and vomiting). Mesenteric ischemia is thought of as a final possibility after ruling out conditions like cholelithiasis, peptic ulcer disease, bowel obstruction, appendicitis, diverticulitis, and gastroenteritis. Accordingly, a high index of radiologic and clinical suspicion is required to diagnose ischemia and infarction of the gut.[1–12] Gastrointestinal tract ischemia can affect bowel viability with potentially catastrophic consequences, including intestinal necrosis and gangrene. Radiologic imaging modalities provide earlier

and more precise diagnosis and hence result in decreased morbidity and mortality of this life threatening condition.[1–12]

EPIDEMIOLOGY:

Vascular compromise of the gut is responsible for approximately 0.2% of hospital admissions and 1% of patients admitted with pain abdomen. The diagnosis of this disorder is on the increase for several reasons. Mesenteric Ischemia (MI) and infarction occur in old age and they mostly have an associated comorbid systemic dysfunction or cardiovascular disease . The population is aging, and the number of cases of Mesenteric Ischemia is expected to increase dramatically as the ‘‘Baby Boom’’ generation comes of age. Other factors include improved diagnostic techniques,heightened awareness of this diagnosis, and the efficacy of intensive care units to salvage critically ill patients.[13–28]

The causes of Mesenteric Ischemia are protean (Boxes 1–3).

Bowel ischemia most commonly occurs within the sixth and seventh decades of life. The age of onset depends on patient gender and the etiology of the ischemia. Primary mesenteric venous thrombosis (MVT) and non occlusive mesenteric ischemia (NOMI) present at the ages of 66.5 and 63 years, respectively.[8] Superior mesenteric artery (SMA) occlusion and non primary MVT present nearly a decade later, at the ages of 77.5 and 74 years, respectively.[8] Chronic mesenteric ischemia (CMI) presents in younger patients

than those with acute mesenteric ischemia (AMI), with a female prevalence as high as 4:1. This is in contrast to the overwhelming prevalence of men with peripheral vascular and aneurysmal disease. Generally, women present with ischemia at an earlier age than their male counterparts, which may contribute to the overall earlier age of onset of CMI.[13–28]

Prompt diagnosis of MI is facilitated by recognizing the various risk factors and comorbidities. Although there is significant overlap among the risk factors for the various vascular disorders of the intestines, certain etiology-specific risk factors have been described.

Acute Mesenteric Ischemia has been associated with recent myocardial infarction, intra-abdominal malignancies, and emboli to the extremities.[22] There is a greater than 50% association between CMI and coronary artery disease, peripheral vascular disease, hypertension, and smoking.

Tobacco use is strongly associated with gut ischemia, with some 70% to 90% of patients admitting to significant use.[6–9]

INTESTINALVASCULAR ANATOMY

Knowledge of mesenteric vascular anatomy and physiology is key to an appreciation of the causes and consequences of intestinal ischemia and infarction. The anatomy of the mesenteric circulation is complicated by the almost endless variations of blood supply to the gut.

Celiac Axis

Abdominal aorta has celiac artery as its largest branch, and it supplies the embryologic foregut. It leaves the abdominal aorta at an angle of 90^{0} at the level of the T12 or L1 vertebral body. After coursing ventrally and inferiorly 1 to 2 cm, the celiac artery branches into the splenic, common hepatic and left gastric arteries in most of the population. In 25%, there is a true trifurcation of these vessels, and in 1%, there is a common origin of the celiac and superior mesenteric branches—the celiacomesenteric trunk.[16]

The common hepatic artery gives rise to the gastroduodenal artery, which then becomes the right gastroepiploic artery and posterior and anterior superior pancreaticoduodenal arteries.

Typically, the left gastroepiploic artery arises from the splenic artery, which joins the right gastroepiploic artery.[16]

Superior Mesenteric Artery

The SMA (Fig. 2) is a large-caliber structure with a narrow takeoff from the aorta, making it the most susceptible of the major mesenteric vessels to embolic phenomena. It supplies the entire embryologic midgut and is the second largest intra-abdominal branch of the aorta.

There is great potential for distal anastomoses as there are more SMA branches to the distal small bowel than to the proximal portions.[12,26] The SMA originates 1 cm below the level of the celiac artery at the level of L1, and

course downwards toward the right and terminates as the ileocolic artery at the level of the caecum. The major branches of the SMA are the inferior pancreaticoduodenal artery, the middle colic artery, the right colic artery, 4 to 6 jejunal branches, and 9 to 13 ileal branches.[16]

Inferior Mesenteric Artery

The Inferior Mesenteric Artery (Fig. 3) is the smallest of the mesenteric vessels and arises 6 to 7 cm below the SMA at the level of L3. It supplies the hindgut: the distal transverse colon, splenic flexure, descending colon, and rectosigmoid. The IMA is a narrow-caliber artery (0.5 cm) that has a relatively acute takeoff angle from the aorta, rendering it much less susceptible to embolic events. The major branches of the IMA include the left colic, sigmoid, and hemorrhoidal arteries. The ascending branches of the left colic artery reach the splenic flexure in 80%to 85% of patients and extend to the midtransverse colon in 15% to 20% of individuals. At this point, they anastomose with branches of the middle colic artery from the SMA. The sigmoidal branches form arcades that anastomose with the left colic artery and superior hemorrhoidal artery.The superior hemorrhoidal artery supplies blood to the wall of the upper two thirds of the rectum and to the entire mucosa .[12,16]The middle hemorrhoidal artery arises from the anterior division of the internal iliac artery or from the vesical branch of this vessel. The middle hemorrhoidal artery traverses the infraperitoneal pelvis in the lateral ligaments and supplies the middle third of the rectum. The

inferior hemorrhoidal artery is a branch of Internal Pudendal Artery which in turn is a branch of the anterior division of the internal iliac artery. It is invested by endopelvic fascia as it exits the pelvis, below the piriformis muscle, through the greater sciatic foramen. It pursues a short course in the buttock and then re-enters the pelvis. It crosses the ischiorectal fossa, which may cause considerable bleeding if encountered during abdominoperineal resection of the rectum. This vessel supplies the levator ani and sphincters in addition to the lower rectum and anal canal.[12–16] The mesenteric vascular anatomy is well depicted on cross-sectional imaging (Fig. 4).

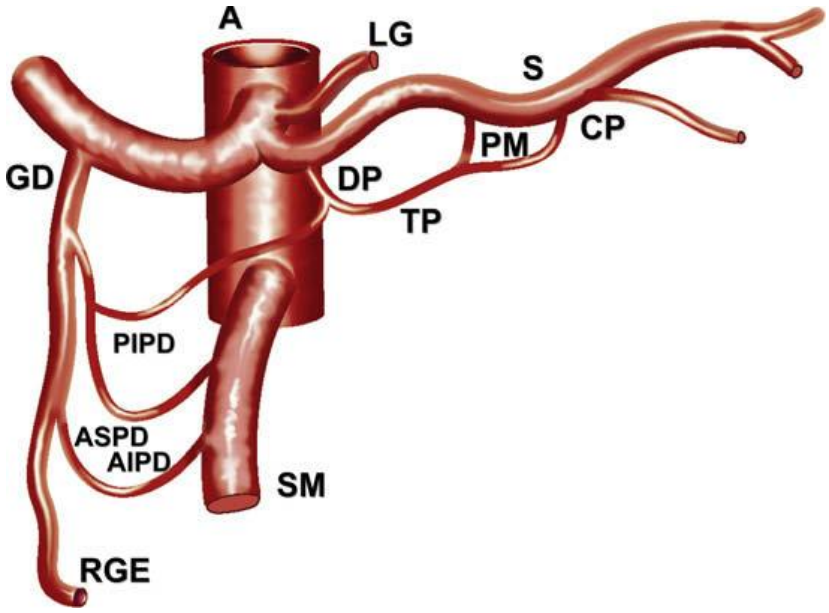


Fig.1. Diagram of typical celiac axis anatomy. A, aorta; AIPD, anterior inferior pancreaticoduodenal artery;ASPD, anterior superior pancreaticoduodenal artery; C, celiac axis; CP, caudal pancreatic artery; DP, dorsal pancreatic artery; GD, gastroduodenal artery; H, common hepatic artery; LG, left gastric artery; PIPD, posterior inferior pancreaticoduodenal artery; PM pancreata magna; RGE, right

gastroepiploic artery; S,splenic artery; SM, superior mesenteric artery; TP, transverse pancreatic artery.

Mesenteric Collateral Flow Patterns

There are numerous sources of collateral flow between the mesenteric vessels and nonmesenteric systemic vessels. This redundancy imparts substantial protection against intestinal ischemia and infarction after segmental vascular occlusion. As a result of these multiple potential sources of collateral flow, at least two of the three main vessels must be occluded or have critical stenoses for MI to develop.

Celiac axis–superior mesenteric artery collaterals

At autopsy, approximately 20% of individuals have greater than 50% stenosis of the celiac artery.[14,15] Most of these patients are asymptomatic because of the rich collateral vessels from the SMA (Fig. 5). The gastroduodenal and superior and inferior pancreaticoduodenal arteries are pathways of collateral flow between the SMA and celiac axis. The arc of Barkow forms potential communications between omental branches from the SMA and branches of the celiac axis.

SMA-IMA COLLATERALS:

At autopsy, it is not uncommon for the SMA (30%) and IMA (30%) to be stenotic[14,15] The marginal artery of Drummond, which lies in the subperitoneal space of mesocolon of the descending colon, consists of the ileocolic, right colic, middle colic, and left colic arterial branches. An anastomosis between the middle and left colic arteries is present in 95% of individuals and occurs at the splenic flexure—the so-called ‘‘Griffith’s point.’’

The arc of Riolan (meandering mesenteric artery) lies within the descending mesocolon as well but is more centrally located and usually joins the middle and left colic arteries.[12,16]

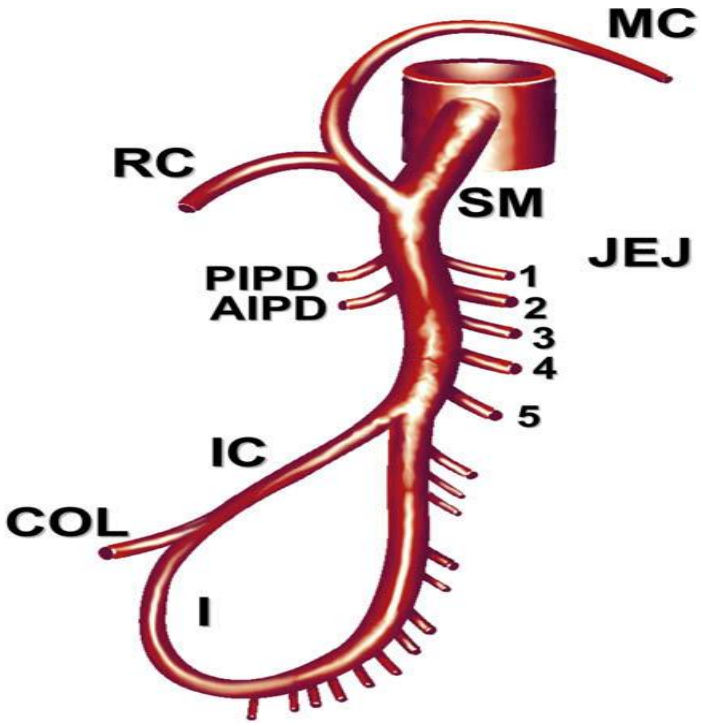


Fig. 2. Diagram of typical superior mesenteric artery anatomy. A, aorta; AIPD, anterior inferior pancreaticoduodenal artery; COL, colic branches; I, ileal branches; IC, ileocolic artery; JEJ, jejunal branches; MC, middle colic artery; PIPD, posterior inferior pancreaticoduodenal artery; RC, right colic artery; SM, superior mesenteric artery.

Inferior mesenteric artery–systemic circulation collaterals

There are collateral vessels within the rectum by way of the anastomoses of the superior rectal chronic celiac artery occlusion, a Whipple's operation can cause hepatic ischemia by interrupting collateral arterial flow from the SMA through the pancreaticoduodenal arcades. A segmental colectomy may interrupt critical anastomotic networks between the SMA and IMA and result in acute ischemia in patients who have severe mesenteric occlusive disease.

The IMA is the most frequently occluded mesenteric vessel by chronic vascular disease. This artery is usually sacrificed during abdominal aortic aneurysm repair. If the SMA is severely diseased and the midgut is receiving a large proportion of blood from the arc of Riolan, sacrificing this vessel during aneurysm repair can cause small bowel and
right colon infarction.[16]

Table 1

Types and approximate incidences of intestinal ischemia
Colonic ischemia 75%
Acute mesenteric ischemia 25%
Focal mesenteric ischemia 5%
Chronic mesenteric ischemia 5%
Mesenteric venous thrombosis Included in previous incidences

Table 2

Causes and approximate incidences of acute mesenteric ischemia
Embolus of Superior mesenteric artery 50%
NOMI 25%
SMA thrombosis 10%
MVT 10%
Segmental ischemia 5%

Box 2

Disorders associated with mesenteric venous thrombosis	
Antithrombin resistance Estrogen use Neoplasms Pregnancy Cirrhosis After sclerotherapy of esophageal varices Diverticulitis Pancreatitis Post operative states Splenectomy	Deficiency of Methyltetrahydrofolate replacement therapy) Thrombocytosis Peripheral deep vein thrombosis Portal hypertension Congestive splenomegaly Inflammation Appendicitis Perforated viscus Pelvic or intra-abdominal abscess Blunt abdominal trauma Decompression sickness

Causes of mesenteric ischemia	
ACUTE MESENTERIC	Emboli
ISCHEMIA	Valvular disease
Arrhythmias	Hypokinetic ventricular wall
Myocardial infarction	Aortic atherosclerotic disease
Cardiac aneurysm	Thrombosis
Iatrogenic	Nonocclusive
Atherosclerotic disease	Cardiac bypass
Heart failure	Renal failure
Sepsis	Pancreatitis
Medications	Venous occlusion
Burns	Sepsis
Hypercoagulable states	Portal hypertension
Malignancy	Pregnancy
Compression	Atherosclerotic disease
CHRONIC MESENTERIC	Inflammatory disease
ISCHEMIA	
Arterial hyperplasia or dysplasia	

Box.3 Causes of colonic ischemia	
IMA thrombosis	**Surgical**
Arrhythmia	Reconstruction of aorto iliac vessel
Congestive heart failure	Gynecologic surgeries
Vasculitis	**Medications**
Hematologic disorders	Related to vasoconstriction or
Sickle cell anemia	vasculitis
Factor I 2010A mutation (in	Digitalis
combination	Gold
with oral contraceptive use)	Pseudoephedrine
Polycythemia vera	Sumatriptan
Infections	Cocaine
Parasites	Methamphetamine
Angiostrongylus costaricensis	Nonsteroidal anti-inflammatory drugs
Entamoeba histolytica	Imipramine
Viruses	Related to hypovolemia or
Cytomegalovirus	constipation
Bacteria	Interferon-a
Escherichia coli O157:H7	Saline laxatives
Trauma	Estrogens
Long-distance running	Progestins
Pregnancy	Danazol
	Psychotropic medications
	Alosetron
	Vasopressin

MESENTERIC VENOUS THROMBOSIS

Mesenteric venous thrombosis (MVT) is responsible for 5% to 15% of bowel ischemia. It is more common in the fifth and sixth decades of life.

MVT includes acute, subacute, and chronic forms. Acute MVT occurs when the major mesenteric veins are occluded. Patients present with severe abdominal pain, distention, and positive fecal occult blood. Subacute MVT is usually caused by thrombosis of smaller mesenteric veins. This form has a slow insidious onset and evolves over

days with vague abdominal pain and nausea. Chronic MVT lasts several weeks or months. MVT is defined as primary in cases where no apparent cause of mesenteric venous occlusion is evident. Secondary MVT includes all mesenteric venous occlusions with known predisposing conditions.

Most cases have an identifiable cause with only 10% now characterized as idiopathic In 60% to 80% of MVT, a predisposing cause was identified. The causes of MVT include hypercoagulable states, venous stasis, and inflammation. Up to 50% of cases have had a deep venous thrombosis or pulmonary embolism before.

Bowel involvement in MVT is usually segmental with edema and hemorrhage of the bowel wall with focal exfoliation of the mucosa. Its presentation may be acute or chronic, generally presenting for evaluation 1 to 2 weeks after onset of symptoms. The diagnosis is difficult as

the symptoms are often nonspecific. Most patients will have abdominal pain (90%), but the onset is variable and location is inconsistent. Patients typically do not experience the classic symptoms of postprandial pain and sitophobia seen in arterial thrombosis.

Nausea and vomiting are present in 60% to 75% of patients and 30% will have altered bowel habits with either constipation or diarrhea. The diarrhea infrequently is bloody, but more than one half of patients are positive for occult blood. Fever is a common finding along with abdominal tenderness, distention with decreased bowel sounds, but peritoneal signs are seen in only two thirds of patients.

As with AMI, the laboratory parameters are nonspecific. Plain films of the abdomen often are normal at presentation or simply show a nonspecific ileus. These factors all contribute to the difficulty in making this diagnosis, but do not lessen the importance.Mortality is directly related to timing of diagnosis and institution of prompt therapy.The available data suggest that the most common disorder is the factorV Leiden mutation (causing resistance to activated protein C), which is present in 20% to 40% of patients. Other hypercoagulable states include such hematologic conditions as antithrombin III deficiency, protein C and S deficiency, polycythemia vera, thrombocytosis, hyperfibrinogenemia, and myeloproliferative disorders.

Hypercoagulability is also associated with use of oral contraceptives and neoplasms of the gastrointestinal tract, pancreas, lung, and ovary. Patients after

surgery are at increased risk for MVT. Splenectomy is the procedure with the highest risk for MVT. Most MVTs occur within 1 month after splenectomy. Abdominal trauma, abdominal inflammation, peritonitis, and portal hypertension are other risk factors for MVT.

Acute MVT has high incidence of recurrence (33%–40%) in the early postoperative period, so that many clinicians perform a second-look operation either routinely or selectively. In the case of selective second-look operations, the choice to perform a second-look procedure is based on the lack of clear demarcation zones at the first operation, leaving questionable zones of intestine not resected and left for observation.MVT without peritoneal signs can be managed conservatively. Early systemic anticoagulation with heparin is the main nonoperative treatment.

Non occlusive mesenteric ischaemia (NOMI)

NOMI accounts for 20% to 30% of acute mesenteric ischemic events, and, as with other types of AMI, carries a high mortality rate of 50% to 90%. NOMI refers to ischemia secondary to a low flow state in the absence of arterial or venous occlusion.

The decrease in cardiac output is associated with a diffuse mesenteric vasoconstriction which further reduces flow, leading to ischemia and ultimately necrosis. NOMI occurs mostly n the intensive care setting. These patients

suffer from either cardiogenic or septic shock. The use of inotropic agents such as noradrenalin (alpha agonist causing generalised vasoconstriction) aggrevate the mesenteric. Long term dialysis patients are another at high risk for NOMI. The severe and rapid fluid shift associated with the dialysis is contributory to the abdominal symptoms [27, 28].

Clinical symptoms are often missed as the patients with NOMI may be critically ill and the onset of symptoms is often insidious. The risk increases further if the patients are receiving enteral nutrition. The increased demand generated by the enteral feedings may exceed the capacity of blood flow to meet the metabolic requirements Watershed areas of mesenteric circulation in the region of the splenic flexure tend to be affected with NOMI. Another group which is vulnerable to NOMI is the surgical post-operative or polytrauma patient receiving enteral feeding in the intensive care unit.

The reported incidence of AMI in this group is 0.3 to 8.5% [11]. The increased demand from enteric feeding fails to be met by the systemic hypoperfusion and the mesenteric vasoconstriction. The diagnosis is difficult to make since the patients are often unable to alert the attention to the abdomen. Before this entity was recognised, the mortality was nearly 100% in cardiac patients following use of vasopressors, but the mortality associated with NOMI has decreased with increasing awareness and increasing use of vasodilators and afterload – reducing agents.

A series of autopsies were reviewed to assess the incidence of NOMI, the extent of involvement, and the potential risk factors.[18] The incidence was rare, yet increased in octogenarians from 2/100,000 person-years before age 80 to 40/100,000 personyears after age 80. Only 29% were correctly diagnosed before death, again verifying the difficulty in making an early diagnosis. The 3 major potential causes in this series were heart failure, atrial fibrillation, and recent major surgery. Forty percent of fatal cases of NOMI were found to have mesenteric stenosis which further contributed to the low flow state.

As with other forms of AMI, early laboratories and the abdominal flat plate show little change or are nonspecific. A high index of suspicion is required and NOMI should be considered in those patients who develop unexplained clinical deterioration or failure to thrive while recovering from a cardiac event or major surgery.

Iatrogenic intestinal ischaemia

Visceral malperfusion may result following post cardiac surgery related intra- aortic balloon pumps orother interventional radiological procedures of the aorta. Global involvement of the viscera, lowerlimbs, kidneys and pelvis may occur because of embolisation and hence is associated with high mortality.

There is no ideal treatment but can be supplemented with intravenous anticoagulation, prostacyclin infusion and appropriate fluid resuscitation. Esonophilia in the peripheral smear may evoke suspicion of cholesterol emboli.

This can occur in patients undergoing anticoagulation,systemic thrombolysis and aortic manipulation.With anticoagulation being contraindicated, statins are used to stabilise the plaque.Fever and systemic shock in the setting of leucocytosis with counts more than 20000 evokes a strong suspicion of severe colonic ischemia.

Bowel obstruction describes a wide variety of disease processes, some of which require urgent surgical management and others which may at least initially be managed nonoperatively. In 1994 more than 300,000 patients underwent adhesiolysis, most of which were for bowel obstruction. Considering that as many as 80% of cases of bowel obstruction are managed nonoperatively, this represents a small fraction of the patients admitted with bowel obstruction. Bowel obstruction is generally classified based on whether it involves the small or large bowel, whether it is a mechanical or functional obstruction, and, in the case of mechanical obstruction, if it is partial or complete.

Classification of the obstruction based on these 3 criteria helps formulate important management decisions. Small bowel obstruction usually presents with abdominal pain, often colicky,nausea, vomiting, and lack of flatus. If ischemia is present the pain may have the classic quality of being out of proportion to tenderness on physical examination. Small bowel obstruction is most commonly caused by adhesions (up to 75% of cases), but may also be caused by hernia, malignancy, inflammatory bowel disease, ischemia, intussusception, infections,

radiation, graft-versus-host disease (GVHD), intramural hemorrhage, stones (gallstones, enteroliths), Meckel's diverticulum, and other generalized inflammatory disorders such as lupus. Important historical questions include prior abdominal surgery, inflammatory bowel disease, cancer, radiation, pelvic inflammatory disease, endometriosis, or other intestinal disease. Examination is important to assess the degree of abdominal distension, tympany, tenderness, bowel sounds, and location and quality of scars, as patients are often poor historians in terms of prior surgery.

The presence of hernia should be carefully excluded. The degree of distension may give a clue as to the location of the obstruction, as distension increases with more distal obstructions. Hernia and distension are often difficult to assess in obese patients. Rectal or stomal digital examination is mandatory in patients who have not had recent surgery in these areas to identify stool or malignancies as the causative factors.

The diagnostic algorithm should focus on the classification of the obstruction as well as on determining the likelihood of ischemia. A complete blood count (CBC) to assess for leukocytosis and lactic acid assists in the identification of ischemia, although these are neither sensitive nor specific for mesenteric ischemia. Azotemia and electrolyte abnormalities are common and in the case of ileus may contribute to the pathophysiology.

The blood counts should be obtained to help assess the degree of volume depletion present in these patients and to assist in fluid resuscitation and electrolyte repletion. Women of child-bearing age should have at least a qualitative b-human chorionic gonadotropin (b-hCG) measurement.

Pathophysiologic changes secondary to aging cause an increased susceptibility to intra-abdominal diseases, as well as atypical clinical presentations. These changes occur from cellular to systemic levels, especially in the immune, genitourinary (GU), gastrointestinal (GI), nervous, and cardiovascular systems. Older adults are at a higher risk for more frequent and severe infections due to immunosenescence. Aging of B cells decreases the ability to develop humoral (antibody) immunity to new infections or antigens, thereby increasing the risk for recurrence. The T cell response also changes with aging, with decreased quantity and quality of the T cells and a decreased immune response to known antigens,possibly because of changes in the phenotype towards more immunosuppressive T cells.These derangements have consequences for interpretation of the white blood cell count; on the one hand, a low count does not exclude an acute inflammatory condition, on the other, an elevated count does not exclude functional immunodeficiency.

Aging is associated with a decreased response to pyrogens, lower basal body temperature, changes in thermal homeostasis, and a decreased production and conservation of heat. In one study, 30% of older adults who had surgical abdominal pain did not present with either a fever or

leukocytosis.Immunosenescence also results in decreasing immunosurveillance, the body's main defense against developing cancerous cells.

Renal changes with aging include decreased numbers of glomeruli and decreased glomerular function. These changes are caused by both long-term damage from comorbidities, such as hypertension and diabetes, and dysautoregulation of the afferent and efferent arterioles resulting in glomerular damage. Glomerular filtration rate decreases with age starting in the fourth decade and then diminishes by about 8 mL/min/decade, resulting in a reduction in the clearance of drugs and metabolites. Changes to the basement membrane and the development of small diverticula in the distal renal tubules promote urinary stasis and bacterial growth.The aging kidneys also have diminished ability to concentrate urine, making older adults more prone to dehydration. Hormonally, the kidneys have reduced production of epoetin, inclining the older adult toward anemia from slow losses of blood.

The effects of aging on the GI system also predispose patients to abdominal pathologic conditions. The stomach has a slightly decreased emptying time and fundal compliance. Acid secretion may increase secondary to decreased prostaglandin production. Liver mass and liver blood flow decrease with aging, resulting in decreased albumin synthesis and decreased phase 1 drug metabolism. The decrease in cytochrome P450 function and drug metabolism may be even greater in older men than in older women.[10] In the colon, the number of diverticula in the bowel increases with age. Because of

physiologic anorexia of aging, there is decreased fluid and nutrient intake, predisposing the older adult to constipation.[11,12] Reduced physical activity for just 2 weeks almost doubles total colonic transit time in older adults, which may contribute to the higher rate of postoperative ileus in this population.

Both the central and peripheral nervous systems are affected by aging. The prevalence of dementia and cognitive impairment increases, obscuring symptoms and obfuscating the medical history. Peripherally, pain and temperature sensation decreases as the type of pain sensing nerves slowly switches from A delta fibers (fast, sharp, prickly pain) to a reliance on slower-conducting C fibers.This sensation decrease may contribute to the lack of peritoneal signs in many older adults.

ISCHEMIC COLITIS

Intestinal ischemia presents itself in most cases as ischemic colitis representing about 50%-60% of cases. Ischemic colitis can be gangrenous or nongangrenous. The latter amounts to 81% to 85% of the cases and include transient and chronic forms.37. In the nongangrenous transient type, there is edema, hemorrhage, and possible necrosis of the mucosa and submucosa. This form usually resolves with a complete anatomic and functional recovery in 2 weeks. The nongangrenous chronic form usually extends to the muscularis propria. Fibrous tissue usually replaces the damaged muscularis propria, resulting in colonic strictures. Gangrenous forms comprise the 15% to 20% of

cases of ischemic colitis and are characterized by transmural necrosis, evolving in sepsis and usually requiring surgical resection.

An acute decrease in the colonic blood supply is usually the cause of ischemic colitis. In most cases of ischemic colitis, the cause is not apparent (idiopathic).

Known causes of ischemic colitis include:

Mesenteric vascular occlusion

Shock

Medications (digitalis, catecholamines, estrogen,danazol, gold, nonsteroidal anti-inflammatory medications, neuroleptics, diuretics and laxatives)

Cocaine

Colonic obstruction

Infection with Escherichia coli

Vascular occlusion may be related to thrombosis, embolization, trauma, radiologic procedures, or surgical procedures. Small vessel disease may be caused by diabetes mellitus, rheumatoid arthritis, or vasculitis.

Colon ischemia is described as a complication of abdominal aortic surgery in 0.2% to 10% of cases. It is five times more frequent after abdominal aortic aneurysm repair. In aortic occlusive disease, the IMA is usually occluded and an adequate collateral circulation to the left colon is already established before

surgery. In patients with aneurysm of the abdominal aorta, intraoperative IMA ligation can cause ischemia of the left colon in case of inadequate collateral flow. Risk factors for colon ischemia after abdominal aortic aneurysm repair include prolonged cross clamp time, reoperative procedures,ruptured aneurysms, hypoxemia, and hypotension. Colon involvement is usually segmental. The entire colon may be affected. The most commonly involved areas are the sigmoid colon and splenic flexure. Because of the vascular anatomy of the colon, two places are at particular at risk for ischemia: Griffith's point(splenic flexure) and Sudek's point (sigmoid). The sigmoid colon is involved in 75% of cases, the splenic flexure in 25%, and the right colon in 10%. A predisposing factor to colon ischemia is the absence of a marginal artery of Drummond at the splenic flexure.

This artery is absent in 7% of the population. Ischemic colitis affects mostly the retroperitoneal surface of the right colon in hypovolumic states. Patients with ischemic colitis are usually elderly and complain of acute abdominal pain, diarrhea, and hematochezia. The blood loss is usually small.The physical examination is remarkable for mild abdominal distention and localized tenderness. The definitive diagnosis of ischemic colitis is based on colonoscopy.

OTHER MESENTERIC ISCHEMIC SYNDROMES

The celiac artery compression syndrome includes all cases of celiac artery compression (external) because of median arcuate ligament or celiac ganglia involvement. Patients usually complain of postprandial abdominal pain, positional abdominal pain,nausea, and weight loss.

Mesenteric vessels may be involved in vasculitis with consequent bowel ischemia. Vasculitis may affect the main arteries, medium-sized arteries or the small vessels.Cocaine abuse is responsible for both acute and chronic ischemic episodes. Chronic ischemia may be due to occlusion of both the superior mesenteric artery and celiac artery.

Radiographic evaluation begins with supine and upright views of the abdomen. These films should be evaluated for bowel dilatation, colonic and rectal air, and air fluid levels. Often the transition point, that is, the location of obstruction, can be determined based on these radiographs alone,although the sensitivity of plain films is low (40%–80%). CT scanning has become a standard imaging modality for bowel obstruction as it has a sensitivity of 94% to 100% and specificity of 90% to 95%.Current CT technology with multiplanar reformats provides greater diagnostic confidence in terms of location and cause of obstruction. CT may determine whether the obstruction is complete or partial, and the presence of a closed loop obstruction or intestinal ischemia. Signs of ischemia on CT scan include thickened bowel wall, pneumatosis

intestinalis, absence of wall or vascular enhancement, portal venous gas, ascites, and inflammatory changes in the fat.

The results of CT imaging often change the management of bowel obstruction, and should therefore be used in cases where diagnosis is in question, or obstruction fails to resolve rapidly with nonoperative management. Small bowel follow-up studies have been reported to result in more frequent and rapid resolution of bowel obstruction, but this was not confirmed in a recent meta-analysis.

Initial management of the patient should focus on fluid resuscitation and the concept that the patient is being prepared for surgery. Medical management should focus on the patient's underlying comorbidities. Fluid resuscitation should be guided based on hemodynamic response and urine output. Monitoring of urine output is critical,and if there is any question of adequate urine output a Foley catheter should be placed. For patients with emesis, a nasogastric tube for decompression can provide symptomatic relief. Antiemetics can be administered judiciously, but for patients at risk for aspiration, nasogastric decompression is more prudent. Prophylaxis for deep venous thrombosis should be administered as these patients will often be bed-bound or destined for surgery. There are no data available regarding antibiotic administration except for immediate preoperative prophylaxis or in the setting of a known infectious cause. Standard therapy for mechanical small bowel obstruction is prompt surgery. This is especially true for patients with clinical signs of bowel ischemia

including fever, leukocytosis, peritonitis, acidosis, patients with radiographic signs of a closed loop obstruction or bowel ischemia, or patients with a complete obstruction. Patients with a partial obstruction, those in the immediate postoperative period, and those with obstruction from primary bowel inflammation such as inflammatory bowel disease or GVHD can generally be managed nonoperatively, as small bowel obstruction has been reported to resolve in up to 81% of cases without operation. Bowel obstruction that fails to resolve with nonoperative management after 48 hours will generally not resolve with more prolonged periods of observation. Although no study has demonstrated worsened mortality with delay in operation for patients without intestinal ischemia, it may be more likely to result in morbidity and the need for bowel resection.

Although laparoscopic or open approaches are feasible, laparoscopy has been reported to be a successful operative modality in 60% of patients requiring operative intervention, with a conversion rate to an open procedure of 20% to 65%.Large bowel obstruction is most commonly due to neoplasm. Other causes include inflammation/infection, volvulus, stricture, intussusception, and fecal impaction.Colonic obstruction typically presents with abdominal pain, abdominal distension,and obstipation. Complications such as ischemia and perforation are, as in small bowel obstruction, heralded by fever, leukocytosis, tachycardia, and peritonitis on physical examination. Diagnosis by plain radiographs has 84% sensitivity and 72% specificity, whereas CT scanning

provides valuable diagnostic information in terms of cause and location. Initial management is centered on resuscitation and correction of electrolyte abnormalities. Unlike with small bowel obstruction, emesis is uncommon and the need for nasogastric decompression is also less common. Definitive management is usually surgical, although for malignant obstructions colonic stents may be useful in cases of poor surgical risk or widely metastatic disease. Volvulus may be reduced endoscopically, which is usually successful in cases of sigmoid volvulus but less so in cecal volvulus. Surgical resection is mandatory in cases of recurrence and should be considered after successful reduction due to high recurrence rates. Patients with obstruction due to acute diverticulitis or other acute infectious causes should be treated with antibiotics, percutaneous drainage of abscesses, and careful monitoring of colonic diameter and serial abdominal examinations for signs of peritonitis.

Acute colonic pseudo-obstruction, Ogilvie syndrome, is a pancolonic dilatation without mechanical obstruction. The pathogenesis of this disorder is believed to involve autonomic dysregulation and occurs most commonly in association with another acute illness. Management generally involves fluid resuscitation and bowel rest with rectal decompression and nasogastric decompression. Narcotic and anticholinergic medications should be minimized. Although absolute cecal diameter is often implicated as determining the risk of perforation, the major risk factor is duration of overdistension in patients with a cecal diameter greater than 10 cm.

Therefore, in addition to the interventions mentioned earlier, therapy with an acetylcholinesterase inhibitor or colonoscopic decompression should be undertaken if there is no clinical improvement within 24 hours. In a randomized controlled crossover trial design, neostigmine resulted in a 94% success rate in improving abdominal distension and a low rate of recurrence, with 11% developing recurrent colonic distension.

Success rates for colonoscopic decompression vary from 61% to 95%. Surgery is indicated for failure of less invasive modalities and generally includes resection with or without reanastomosis if contamination is present at operation. Open and percutaneous cecostomy have been described but significant complications of these procedures have also been noted.

PATHOLOGIC FINDINGS OF INTESTINAL ISCHEMIA

Small Bowel

Pathologic evidence of small bowel ischemia and infarction (Figs. 6–8) may be diffuse and widespread or patchy and in multiple areas. The serosa of the affected small bowel often appears congested or blue and black. Perforations may be present but may not associated with well-developed fibrinous exudates if the surgical resection occurs within a short time of presentation. The mesentery is usually pale in arterial occlusions and congested and hemorrhagic in venous occlusion. The demarcation between normal and involved gut is

usually abrupt.The lumen of the intestine is always filled with blood and the surface of the mucosa may appear boggy, red and ulcerated and contains irregularly protruding islands of mucosa.The above phenomenon is responsible for the thumbprinting sign. Transmural hemmorrhage and pseudomembranes might be evident. The invoved segment shows friable and thin wall.Gross examination of the mesenteric veins in mesenteric venous thrombosis may show evident thrombi. Submucosal edema, congestion and hemorrhage may be seen along with preservation of the overlying mucosa in some instances.

Formation of pseudomembranes, luminal hemorrhage, necrosis of the mucosa with or without ulceration are seen in various degrees of submucosal involvement. There is absence of chronic inflammatory response in an acute episode, nevertheless neutrophils may be seen if significant time has passed since occlusion.[14,15]. Loss of villi occur progressing from villi tips to the cryptal bases and associated with varying degrees of congestion and edema.Neutrophils occur in the damaged areas within hours of the initial ischemic event. If the ischemic process stops, the changes in the mucosa may revert to normal, but it all depends on the extent and severity of the injury. When ischemia is persistent or severe, stricture formation and fibrosis may result in the process of tissue healing.[14,15]. Confusing are results of mesenteric vascular evaluation as thrombi may result due to congestion and prolonged stasis. When thrombi are present for a prolonged time, thrombi show evidence of organisation.Small arterioles may show evidence of fibrin thrombi in areas of necrosis but they do not

necessarily indicate vasculitis or a hypercoagulable state.[14,15]. Deeper portions of the colonic crypts show evidence of necrosis sparing the superficial portions in acute ischemic episodes of the colon.

Evident cytological atypia in the remaining crypts which show atrophic or withered appearance may resemble dysplasia. Lamina proprial hemorrhage , laminal proprial hyalinisation and pseudomembrane formations may also be seen. These lesions come down on their own or florid gangrene with perforation or formation of stricture can occur. The chronic phase of colonic ischemia may be very difficult to diagnose as non specific hiatological findings like stricture formation and submucosal fibrosis may only be evident.[14,15]

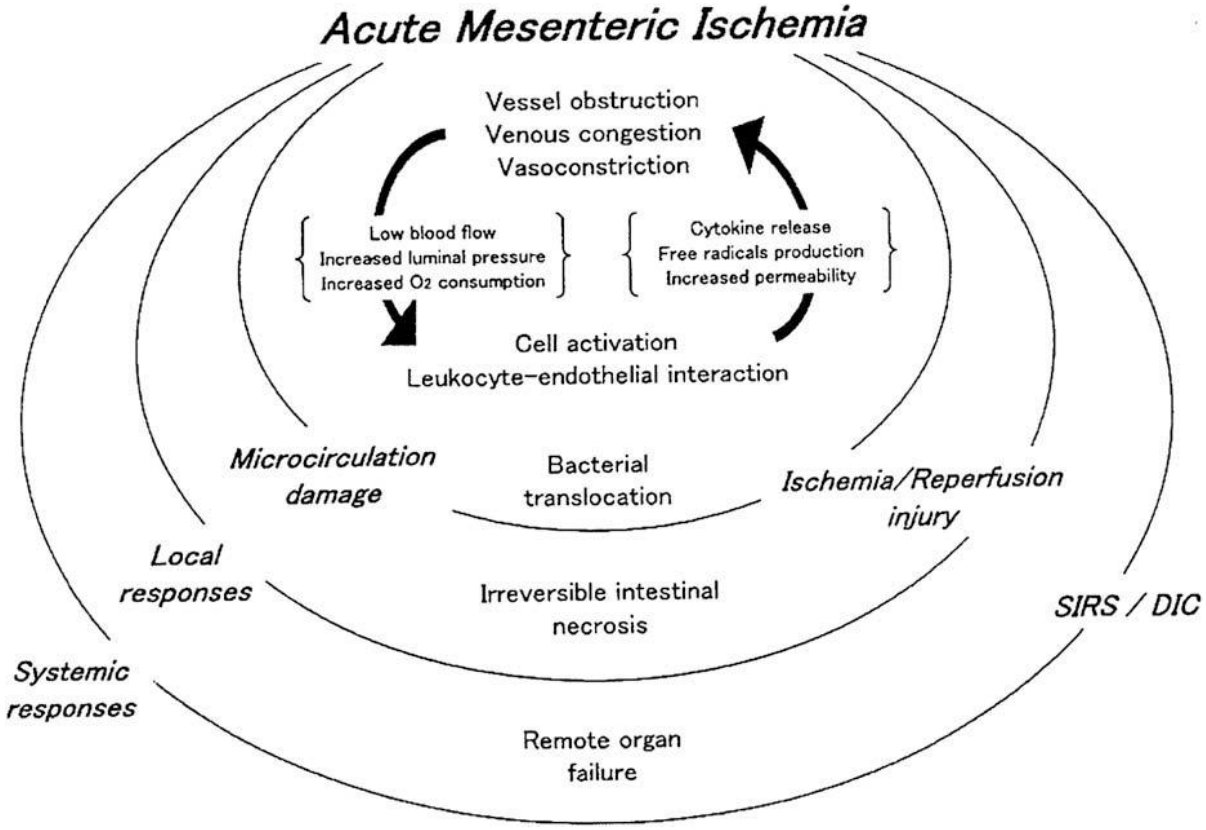
CLINICAL FEATURES OF MESENTERIC ISCHEMIA

No physiological or biochemical methods of detecting ischemia and predicting its outcome have been proven till date. Cell hypoxia may reveal itself as increased serum lactate but lactic acidosis occurs only with circulatory collapse and bowel necrosis in the later stages only.

Plasma D-dimer levels is coming up as an early marker in acute ischemic events. It has been shown to have good correlation with onset of ischemia and function thus indicating disease progression as evident in animal studies.

Alcohol dehydrogenase enzyme levels is a sensitive indicator of bowel ischemia when compared with generalised systemic hypoperfusion. Glutathione S-transferase with its several isoforms which are associated with bowel

specificity have been released with cell membrane damage. It increases with considerable rates with progressive tissue ischemia.



Acute Mesenteric Ischemia

Acute mesenteric ischemia is caused by an abrupt reduction of either arterial or venous blood flow to the gut, and is associated with a high mortality and morbidity rates requiring urgent diagnosis and treatment.32 Almost all patients present with severe pain abdomen. The onset of pain is usually sudden in patients with emboli as the cause. On the other hand, patients with thombotic etiology may have a more insidious onset of symptoms.

Nausea, vomiting, and diarrhea are also common complaints.[32]

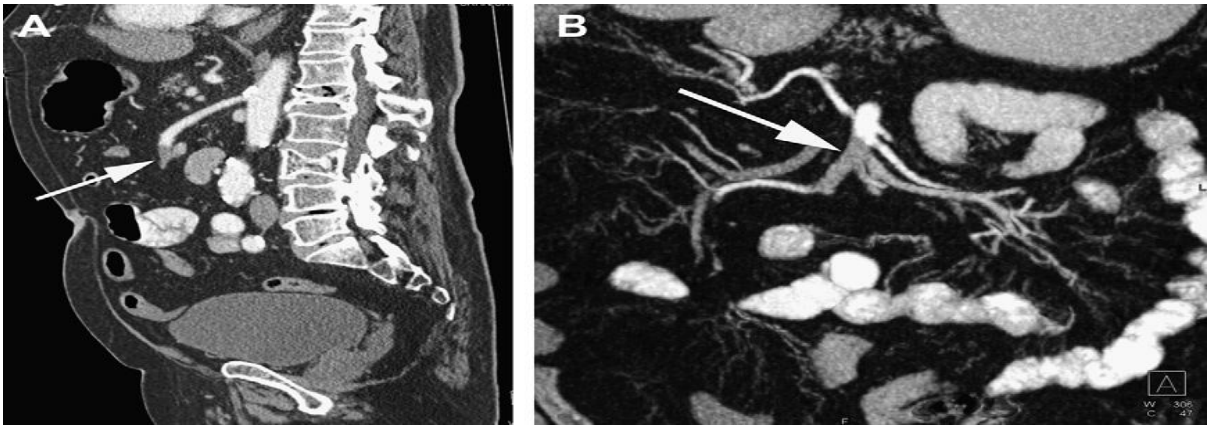
CTA is an excellent modality to evaluate patients with suspected acute mesenteric ischemia. CT in combination with 3D CTA can evaluate the mesenteric vasculature as well as bowel enhancement, and is therefore a comprehensive study. Thereare 4 major causes of acute mesenteric ischemia:

SMA embolus, SMA thrombus, mesenteric venous thrombus, and nonocclusive mesenteric33 ischemia.[34]

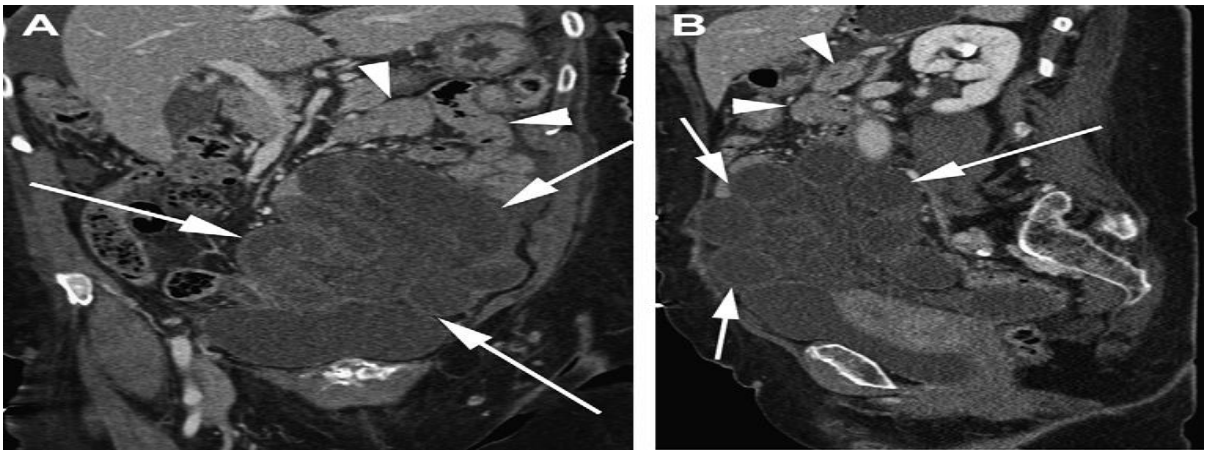
Acute emboli to the SMA are the most likely origin of AMI, occurring in 41% to 50% of cases.[35] Most of the emboli originate in the heart and will lodge in the Superior Mesenteric Artery a few centimetres distal to origin, mostlikely near the origin of the middle colic artery. Small emboli lodge more distally and mostly affect only small bowel segments.[35,36] The arterial thrombus is visible as a low-density filling defect on CT. Proximal thrombi are best visualized on the sagittal reconstructions,while distal thrombi may only be visible using volume rendering with comprehensive interrogation of all the distal mesenteric branches [37] Regardless of the cause of the ischemia, the affected small bowel loops may be dilated and fluid filled, as a result of an interruption in normal peristalsis and increased secretions. The wall may be thickened, but in some cases will actually be normal or thinned . Ischemia usually causes bowel wal circumferential thickening. The bowel wall that is ischemic is typically to 8 to 9 mm thick, but in some cases becomes as thick as 1.5 cm. Bowel wall thickening is important in cases of thrombosis of veins than in cases of arterial thrombosis. Therefore, a

bowel wall measuring 1.25 cm, in the setting of ischemia, most likely signals obstruction of venous blood flow. Complete lack of mural enhancement has been reported but is an unusual finding, given the redundant blood supply to the gut. The bowel wall may appear of low density, reflecting decreased perfusion.

Fig. 3. An 80-year-old man presenting with pain abdomen. (A) Sagittal MPR and (B) Coronal MIP show a large thrombus in the mid SMA (arrow); this was embolic, presumably from a cardiogenic source. Surgical embolectomy was performed.



F.g.3



F.g.4

Fig. 4. An 80-year-old woman with an acute small bowel obstruction. (A) Coronal MPR and (B) sagittal MPR show fluid-filled small bowel loops that are dilated. The wall appears thinned and has decreased enhancement (arrows) compared with the more proximal and unaffected small bowel loops (arrowheads).

Edema, or may appear increased in density relative to normal bowel loops,related to hemorrhage or hyperemia. The halo sign may be present. Intramural hemorrhage may be present,and is often only appreciated if noncontrast scans are obtained. After intravenous contrast is administered, intramural hemorrhage may be misinterpreted as enhancement. Pneumatosis is a late finding, indicating transmural infarction, and may be accompanied by air in the mesenteric veins and/or portal vein (Fig. 5). In patients with acute arterial ischemia, there may be stranding in the mesentery and ascites, also indicating severe ischemia and usually transmural infarction. Thrombosis of the SMA usually occurs in atherosclerotic disease, because of rupture of an unstable atherosclerotic plaque.

Thrombosis of the Superior Mesenteric Artery is thought to be responsible for up to 30% of all cases . Unlike emboli, thrombi typically develop at the origin of the SMA and within the first 2 cm, best visualized using sagittal reconstructions. There is usually a combination of calcified plaque with superimposed thrombus. Because SMA thrombosis often occurs in the setting of patients with chronic ischemia, there may be associated arterial collaterals,

which can be visualized well using CTA. Another well known cause of acute mesenteric ischemia is mesenteric venous thrombosis (see later discussion).

Nonocclusive mesenteric ischemia is thought to represent up to 25% of cases and is associated with a high mortality rate, up to 70%.43 NOMI occurs in hypotension or hypovolumic shock. Severe hypoperfusion of the gut will cause severe vasooconstriction of the mesenteric arteries. The findings on CT may be subtle.

Fig. 5. Postpartum female (37 years) with severe pain abdomen. Axial contrast-enhanced CT with soft tissues windows (A) and lung windows (B) shows a small bowel obstruction and pneumatosis (arrows). F, large necrotic uterine fibroid.

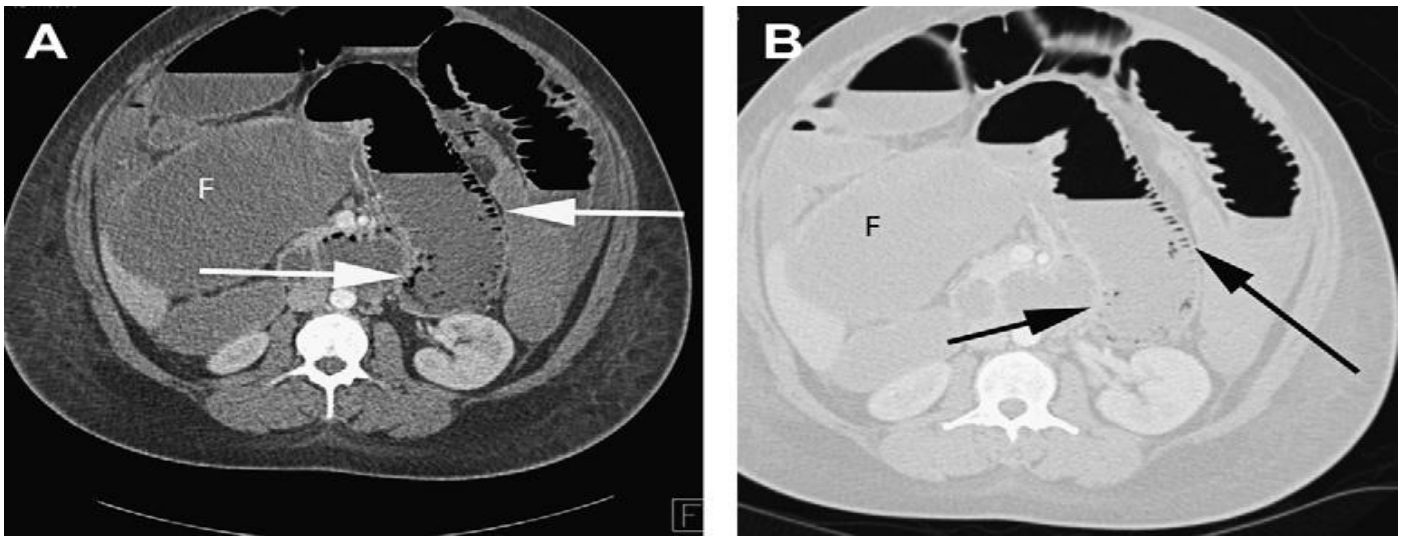


Fig. 6. An 83-year-old with pain abdomen . (A) Sagittal MIP shows extensive calcified atherosclerotic disease involving aorta and proximal SMA (arrow). (B) There is also a filling defect in the proximal SMA (arrows), which is acute thrombus that has form in a region of calcified plaque.

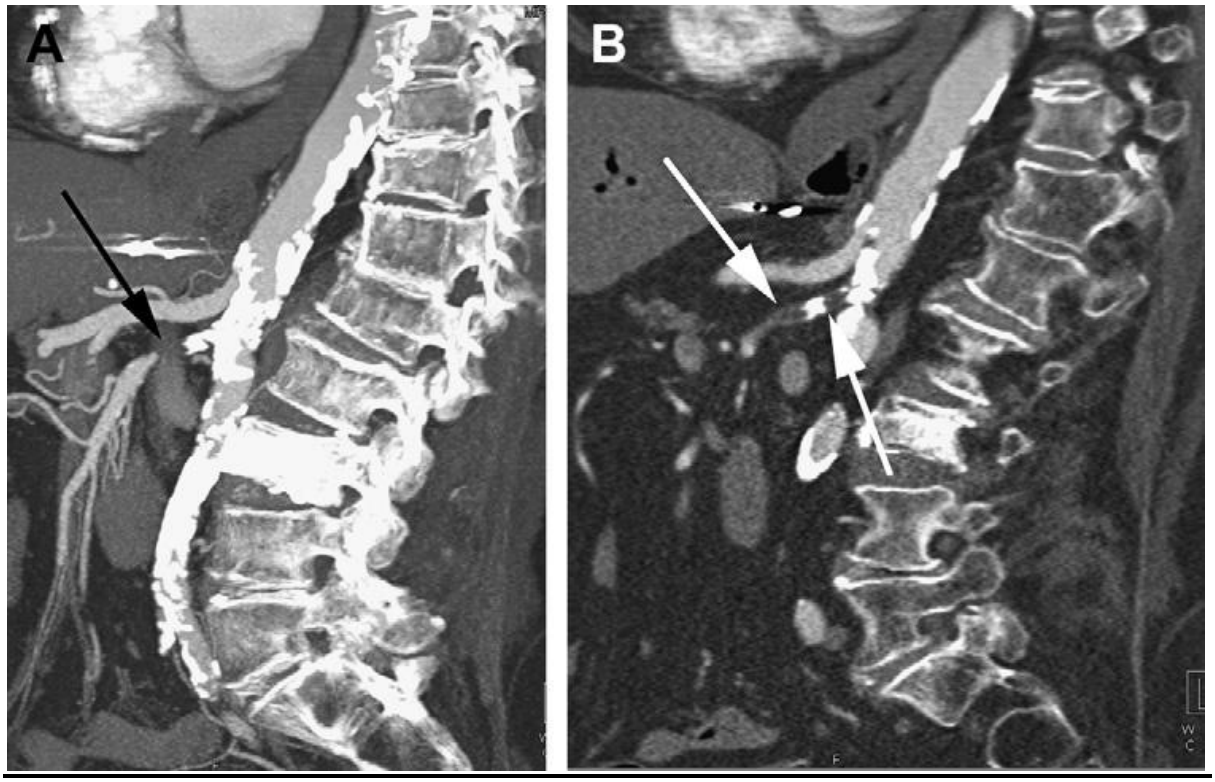
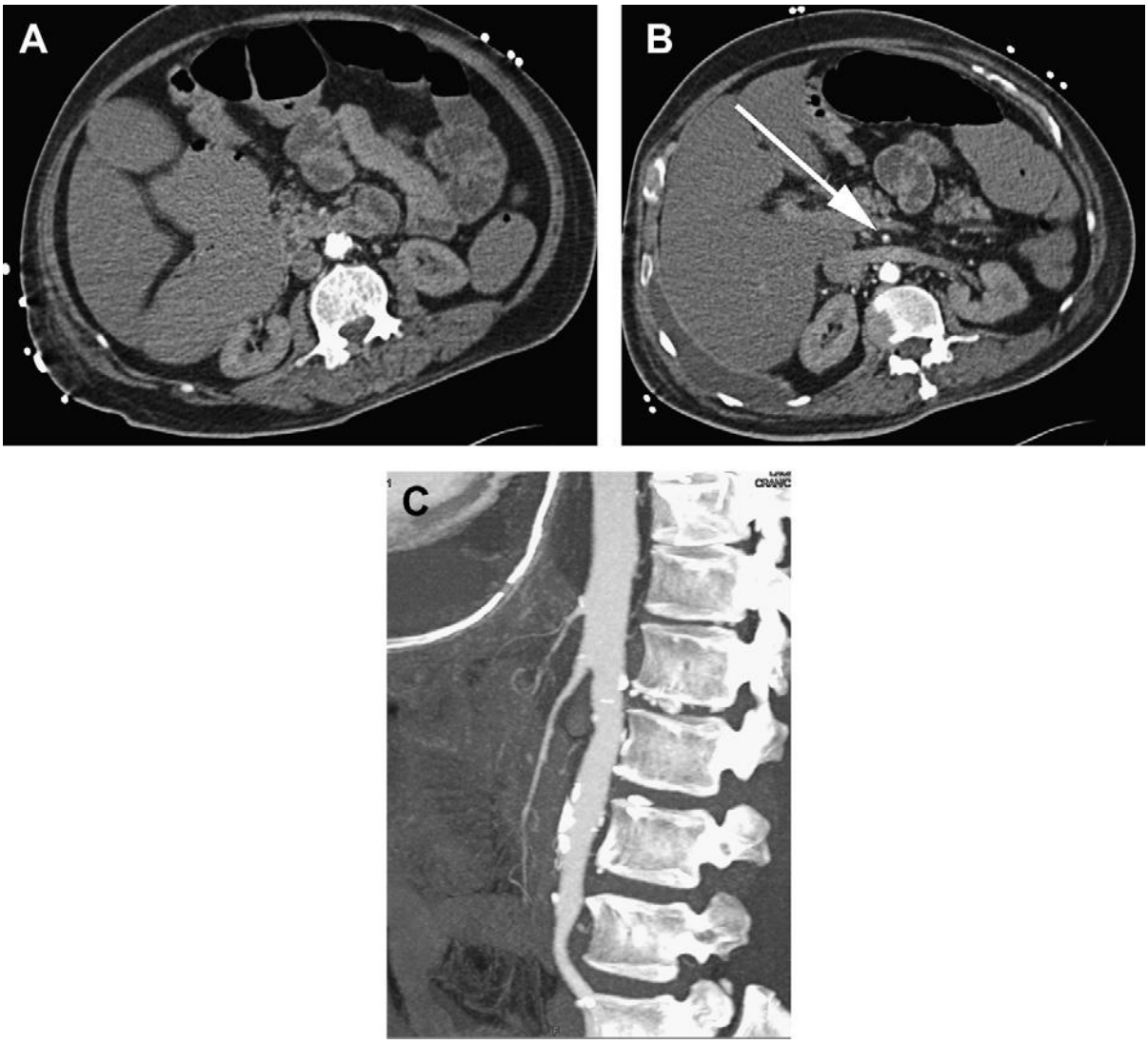


Fig. 7. A 65-year-old man in cardiogenic shock after an acute myocardial infarction. (A) Axial contrast-enhanced image through the mid abdomen shows dilated small bowel and colon as well as poor perfusion of the kidneys.(B) Axial contrast-enhanced image shows a small-caliber SMA (arrow). A right pleural effusion is also present. (C) Sagittal MIP image shows the small-caliber celiac axis and SMA, a typical finding in patients with hypotension.

Fig.7



its branches will appear small in caliber and there may be delayed opacification of the mesenteric veins. The bowel is often dilated and fluid filled. The bowel wall may also be thickened.[35–37] In severe cases pneumatosis or portomesenteric venous gas is present, indicating transmural infarction, which carries a dismal prognosis.

In patients with suspected infarcted bowel from emboli, explorative laparotomy with resection of the ischemic bowel and thus re establishing vascularity to the bowel.

However, advancements in interventional radiology techniques offer an effective, less invasive therapeutic alternative for patients with ischemia but no clear evidence of infarcted bowel. Arterial thrombolysis, angioplasty, and placement of stent are all available and effective. Venous thrombosis can be treated with systemic, anticoagulation, or percutaneous transhepatic delivery of thrombolytics.

Nonocclusive mesenteric ischemia can often by treated with selective arterial administration of vasodilating agents (ie, papaverine). Patients with ischemia resulting from thrombus forming in the setting of chronic mesenteric ischemia may require a combination of percutaneous and systemic therapies, and ultimately may need stenting or surgical revascularization.

Chronic Mesenteric Ischemia:

Atherosclerotic phenomenon in severe stages almost always results in chronic mesenteric ischemia , involving the mesenteric arteries, and therefore

occurs in older patients. It accounts for approximately 5% of all mesenteric ischemic illnesses, but ends in significant morbidity and mortality. Although atherosclerotic disease can often involve the mesenteric arteries, chronic mesenteric ischemia is actually a relatively uncommon but an important cause of abdominal pain in elderly patients. Even in the absence of symptoms, patients may have clinically significant atherosclerotic disease affecting the mesenteric arteries. Up to 18% of patients older than 65 years have greater than 50% stenosis of

a mesenteric artery, usually withosut symptoms. Patients with significant atherosclerotic stenosis of the mesenteric arteries will usually only become asymptomatic when least 2 of 3 major mesenteric vessels, typically the SMA and celiac artery, become severely stenotic or occluded.

Longterm studies have shown that as many as 86% of asymptomatic patients with greater than 50% stenosis of the mesenteric arteries eventually develop symptoms. Mortality is approximately 40%. Standard treatment for chronic mesenteric ischemia involves revascularization, which can be surgical or catheter based.

After surgical treatment, the recurrence rate of mesenteric ischemia symptoms at 3 years is approximately 11%. Percutaneous interventions include embolectomy,thrombolysis, angioplasty, and stenting. In patients with chronic mesenteric ischemia, CT will show significant occlusion of minimum of two of the major mesenteric arteries , usually the celiac trunk and SMA. The stenosis is

usually at the origin and may be a combination of calcified and noncalcified plaque. Because the process develops over a long period of time, collaterals are present. CTA and volume rendering in particular are especially valuable in detecting and quantifying the degree of stenosis and displaying the collaterals.[37] This technique can be used as a road map for the surgeon or interventional radiologist.

VENOUS PATHOLOGY

Mesenteric Vein Thrombosis

Mesenteric vein thrombosis (MVT) accounts for 5% to 15% of all mesenteric ischemias. Thrombosis usually involves the SMV, only rarely involving the IMV.[35] MVT can be classified as either primary or secondary.[35] Primary or idiopathic MVT results when no underlying etiology can be identified. Secondary MVT is more common. Common causes include underlying coagulopathy, either hereditary or acquired. Hereditary factors include Factor III deficiency, deficiencies in protein C, protein S, or antithrombin, or patients with polycythemia vera. Acquired coagulopathy is often related to cancer, intra-abdominal inflammatory conditions, postoperative patients, oral contraceptives, cirrhosis and portal hypertension, or patients with pancreatitis, sepsis, or after splenectomy.[35]

The increased incidence in postoperative patients may be the result of transient hypovolemia or release of tissue thromboplastins at surgery. In a retrospective study by Warshauer and colleagues. 11 of 43 patients with SMV thrombosis were status post partial or total colectomies. Clinical presentation varies depending on the location, extent, and cause of the thrombosis. Patients can present with acute, subacute, or chronic symptoms. Acute presentation can often mimic the presentation of acute arterial ischemia. In acute presentations, patients present with severe pain and there is a high risk of both ischemia and infarction. Outcomes vary, based 342 on the extent of thrombosis. Complete thrombosis carries a poor prognosis, as does extension of thrombus into other veins. Acute thrombosis can result in venous hypertension depending on the residual drainage from the intestines. Severe venous hypertension will compromise the viability and perfusion of the bowel.[35] On CT, thrombus will be visible in the mesenteric veins, typically associated with engorgement of the veins [35] The walls of the veins may be thickened with increased enhancement. Stranding in the mesentery and ascites are also often present. The bowel wall is usually thickening, often related to the venous obstruction. There may be decreased enhancement of the wall, or in some patients there may be increased enhancement due to hyperemia. A halo pattern has also been described.[35] Complete lack of bowel enhancement is uncommon, but does signify transmural infarction, especially when there is accompanying pneumatosis or

portomesenteric gas.[35] In subacute situations, patients may have abdominal pain, but typically do not show associated signs of ischemia, likely related to the development of collaterals.

Treatment in acute and subacute cases usually includes anticoagulation, alone or in combination with surgery.[35] Conservative treatment usually consists of anticoagulation, supportive measures, and bowel rest. Surgery may be required in patients with peritonitis and signs of ischemia/ infarcted bowel. Surgery may include resection of the small bowel, although the goal is to conserve as much bowel as possible. In select cases, thrombectomy or mechanical and percutaneous clot lysis can be performed. Overall, MVT carries a high mortality rate (20%–50%). The principal cause of the high mortality is delay in diagnosis. The condition is often misdiagnosed as gastroenteritis, small bowel obstruction, or inflammatory bowel disease. Even after treatment there is a high rate of occurrence, most commonly in the first 30 days after treatment. Chronic MVT, often in cirrhotic patients, typically causes little symptoms because of the development of an extensive collateral network.

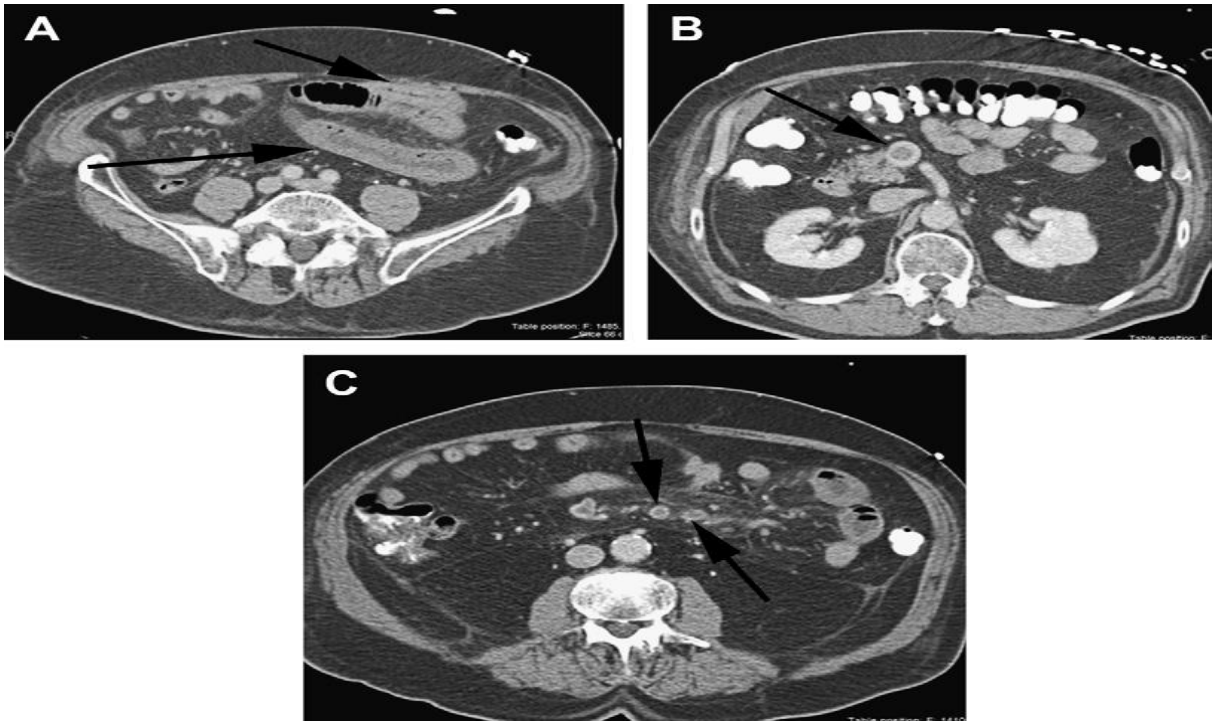
However, these patients are at increased risk for GI bleeding related to the presence of the collaterals. In patients with chronic MVT, treatment may include propranolol to help decrease the risk of variceal bleeding.

On CT, chronic MVT will appear as a low-density intraluminal clot. Enlargement of the vein and bowel wall thickening, as well as mesenteric

stranding and edema are typical associated findings. Multiple veins may be involved. Venous collaterals may also be present.

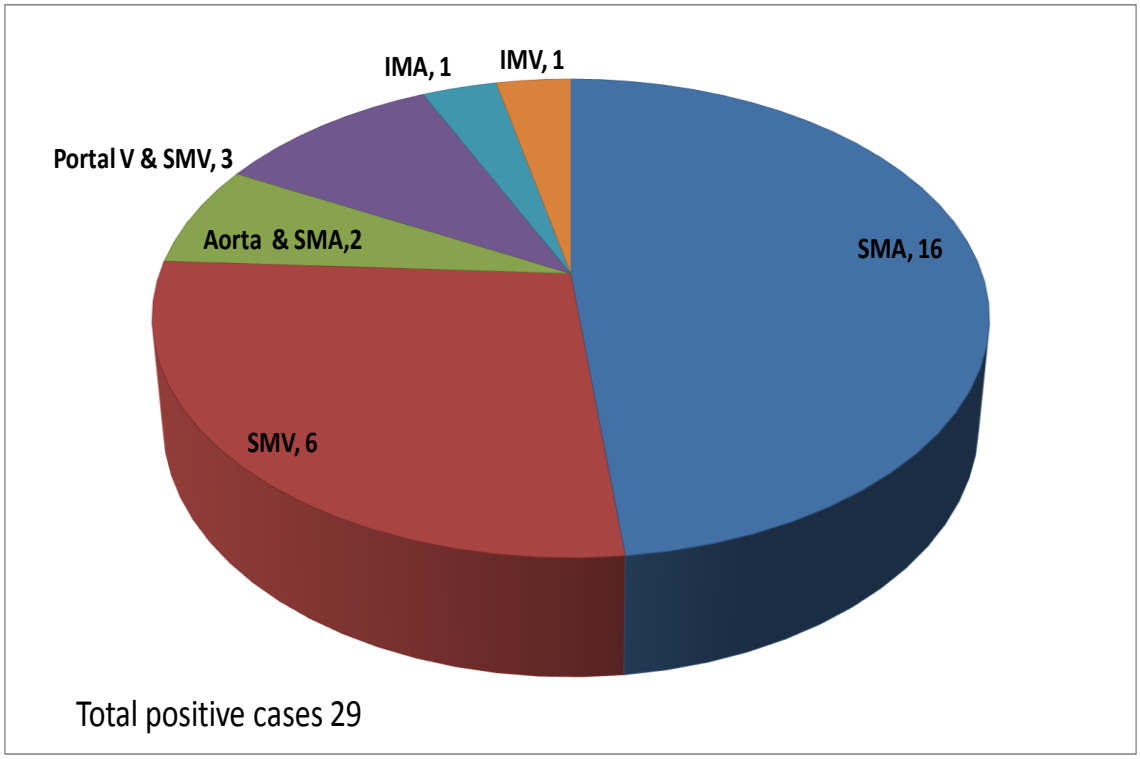
Fig. 8. An 80-year-old man with abdominal pain and history of Osler-Weber-Rendu syndrome. (A) Contrastenhanced axial CT shows small bowel thickening (arrows). (B) Axial image through the superior mesenteric vein shows a large clot (arrow). (C) Axial contrast-enhanced image through the mid abdomen shows extensive thrombus (arrows) in the branches of the SMV.

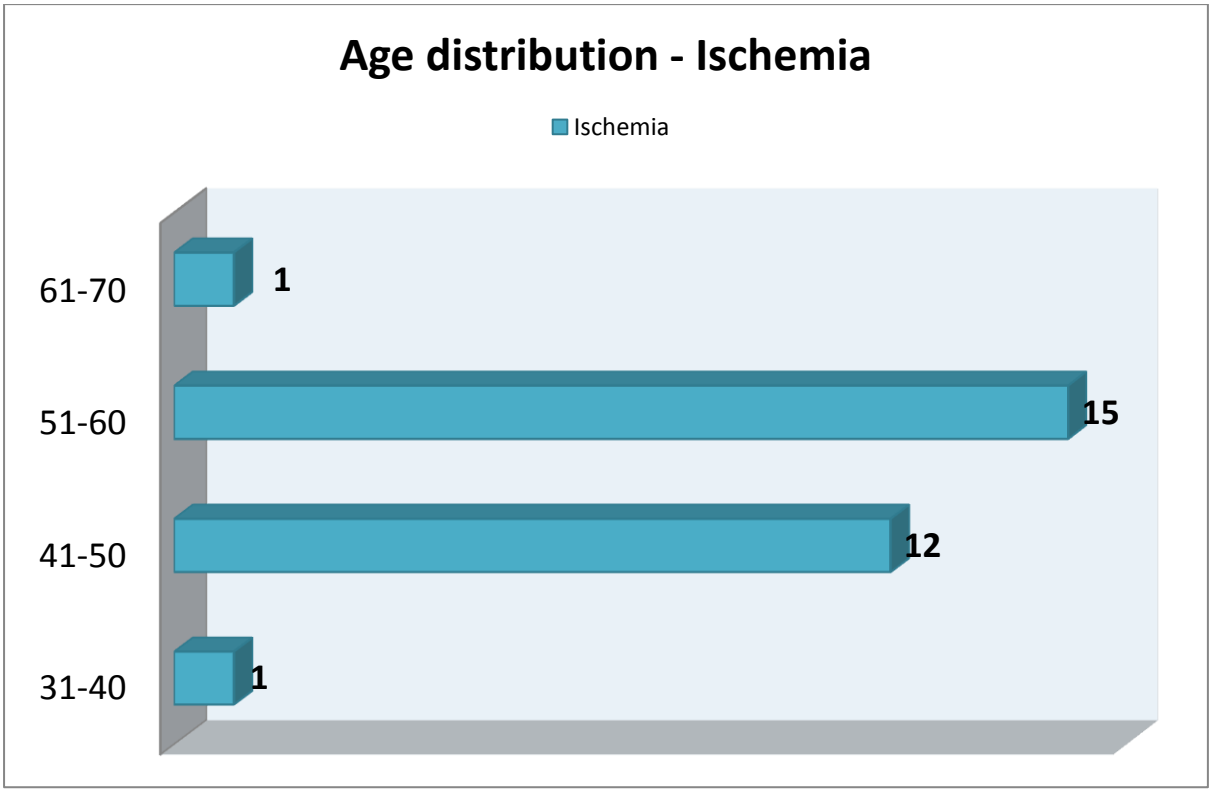
Fig.8



RESULTS:

The following pie diagram illustrates the disease distribution in various vessels.

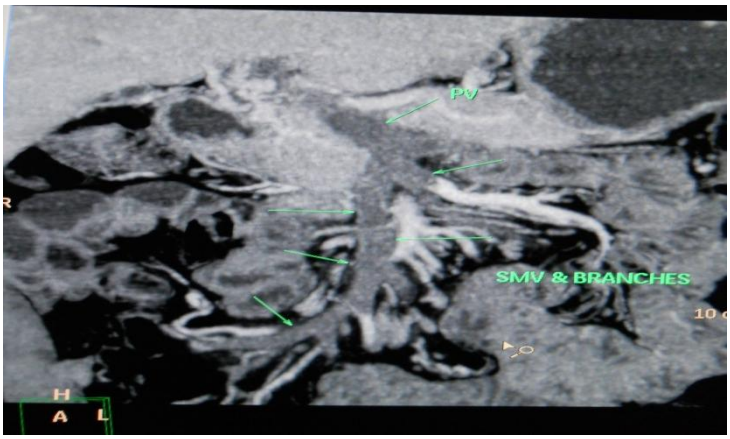




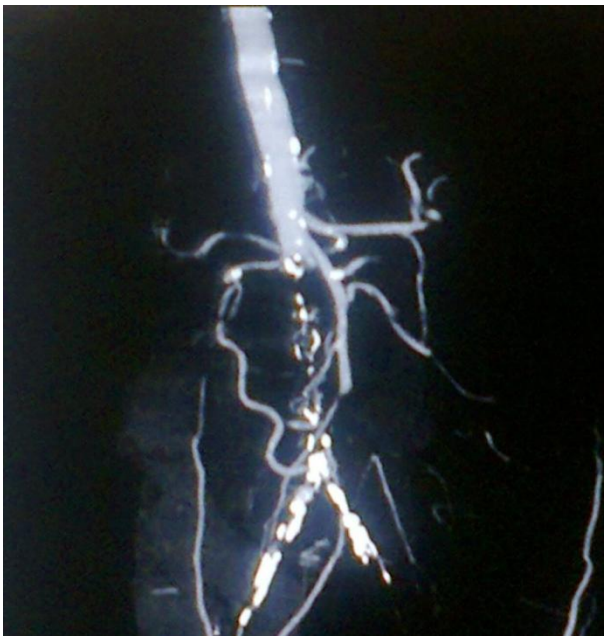
The agewise distribution of mesenteric oschemia is illustrated in the above table.

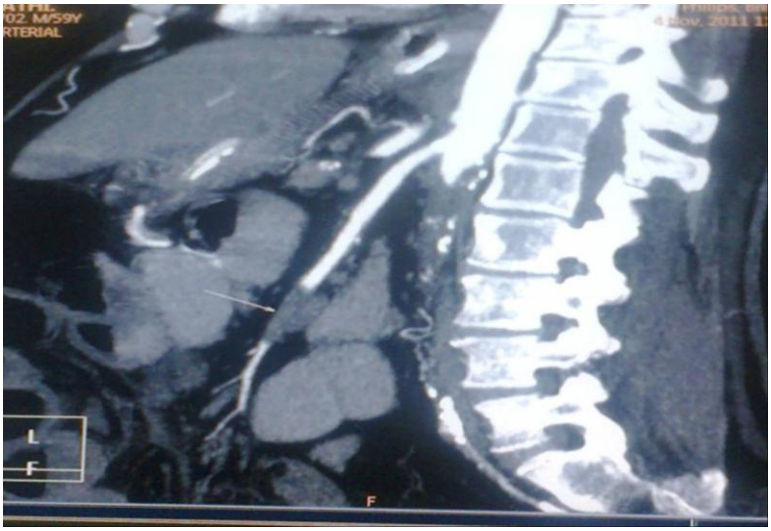
<u>Radiographic evidences of the involved vessels are to follow</u>:

SUPERIOR MESENTERIC VEIN AND ITS BRANCHES.

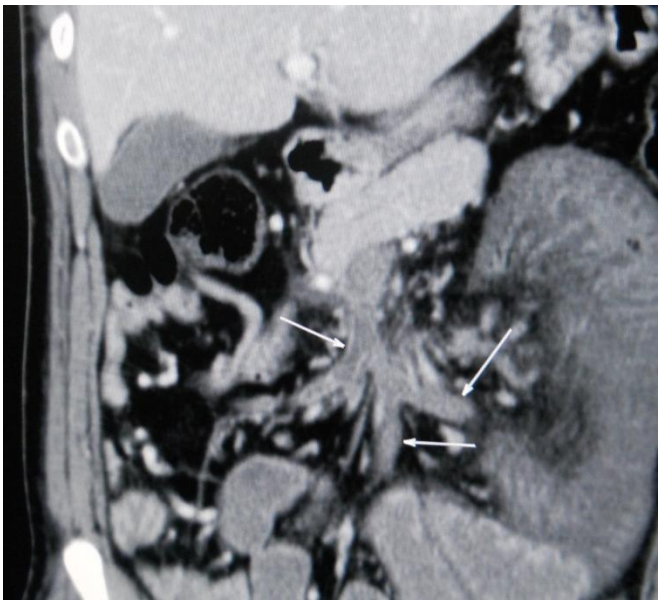


INVOLVEMENT OF SMA AND AORTA

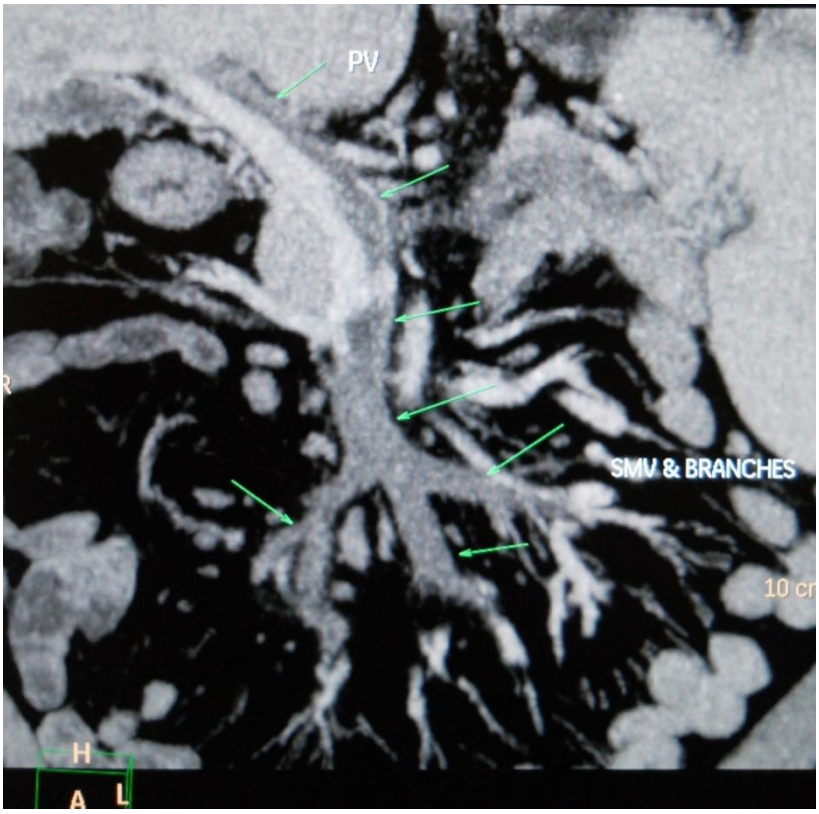




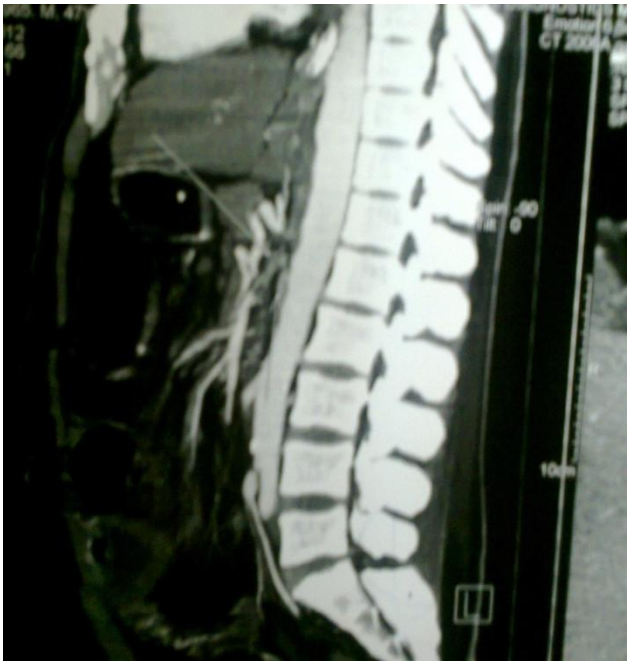
INFERIOR MESENTERIC VEIN



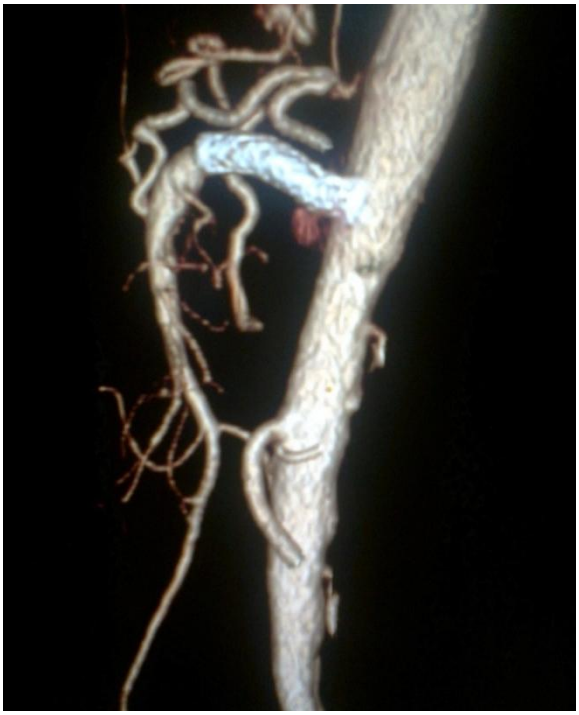
PORTAL VEIN & SMV INVOLVEMENT



SMA INVOLVEMENT

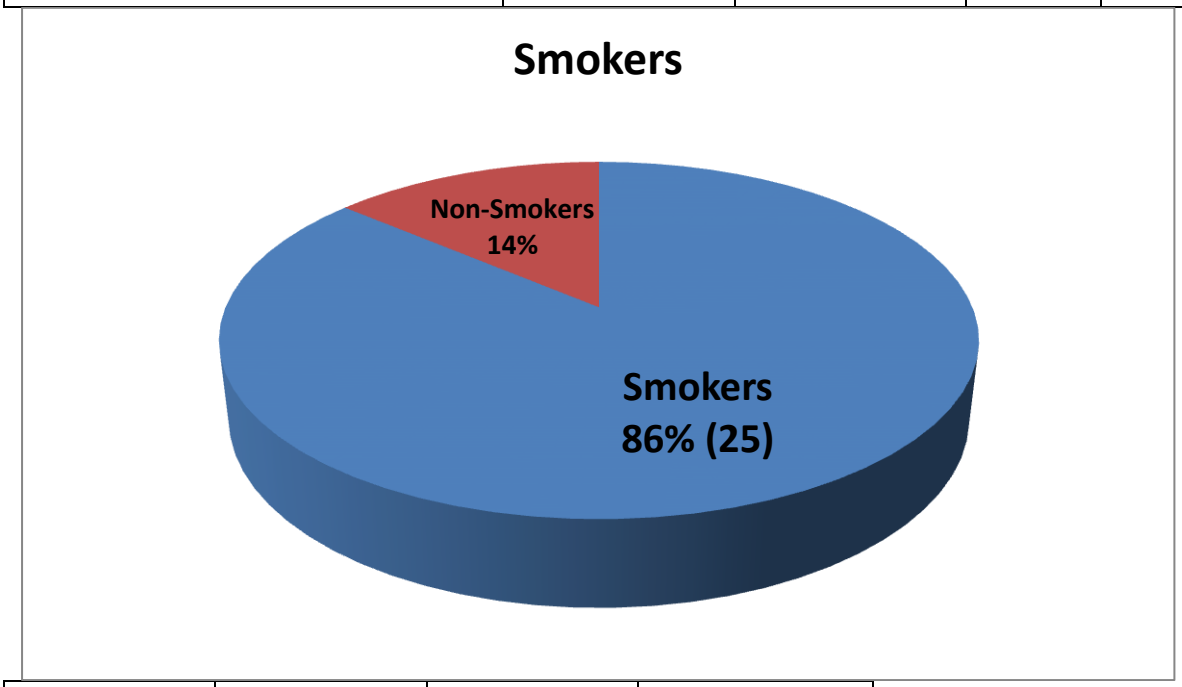


SMA AFTER STENTING



The statistical analysis illustrating the relationships between mesenteric ischemia and the various risk factors is illustrated below:

MESENTERIC ISCHEMIA				
		(+)	(-)	
	(+)	25	7	32
SMOKING	(-)	4	20	24
		29	27	56



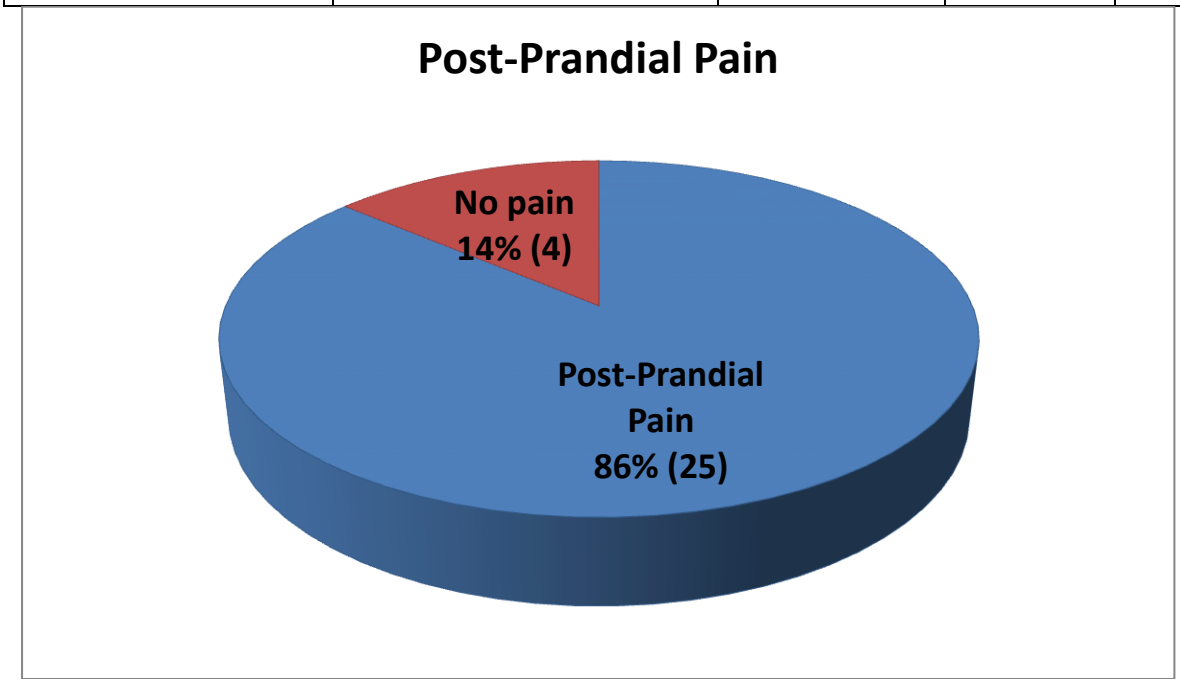
Test	Value	p-value(2-tail)
Yates corrected chi square	18.36	0.00001830

Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
CMLE Odds Ratio*	16.65	4.499, 74.16[1]
		3.946, 90.76[1]
Odds Ratio	17.86	4.575, 69.7[1]
Etiologic fraction in pop.(EFp|OR)	81.38%	63.94, 98.82
Etiologic fraction in exposed(EFe|OR)	94.4%	78.14, 98.57

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	86.21%	(69.44, 94.5[1])
Specificity	74.07%	(55.32, 86.83[1])
Positive Predictive Value	78.13%	(61.24, 88.98[1])
Negative Predictive Value	83.33%	(64.15, 93.32[1])
Diagnostic Accuracy	80.36%	(68.16, 88.66[1])
Likelihood ratio of a Positive Test	3.325	(2.482 - 4.455)
Likelihood ratio of a Negative Test	0.1862	(0.1102 - 0.3146)
Diagnostic Odds	17.86	(4.574 - 69.71)
Cohen's kappa (Unweighted)	0.6051	(0.3447 - 0.8655)
Entropy reduction after a Positive Test	16.72%	
Entropy reduction after a Negative Test	24.19%	
Bias Index	0.05357	

From the table, it is clearly evident that there is statistically significant correlation between smoking and mesenteric ischemia.

	MESENTERIC ISCHEMIA			
		(+)	(-)	
	(+)	25	1	26
POSTPRANDIAL PAIN	(-)	4	26	30
		29	27	56



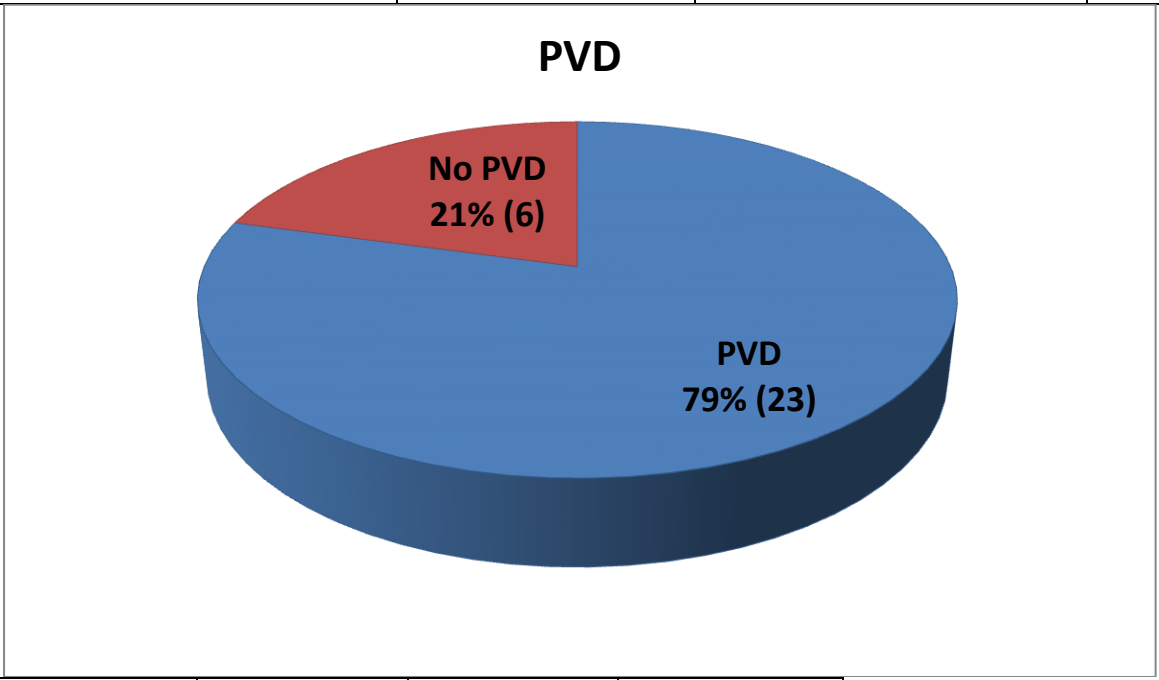
Test	Value	p-value(2-tail)
Yates corrected chi square	35.02	<0.0000001

Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
Risk in Exposed	96.15%	79.55, 100
Risk in Unexposed	13.33%	4.695, 30.29
Overall Risk	51.79%	39.01, 64.33
Risk Ratio	7.212	2.887, 18.01[1]
Risk Difference	82.82%	68.59, 97.05°
Etiologic fraction in pop.(EFp)	74.25%	53.23, 95.27
Etiologic fraction in exposed(EFe)	86.13%	65.36, 94.45

Point Estimates				Confidence Limits
Type			**Value**	**Lower, Upper**
CMLE Odds Ratio*			135.6	18.78, 3464[1]
				15.44, 6909[1]
Odds Ratio			162.5	16.98, 1556[1]
Parameter		**Estimate**	**I** 85.68 %	72.6, 98.75
Sensitivity		86.21%		
Specificity		96.3%		
Positive Predictive Value		96.15%		
Negative Predictive Value		86.67%		
Diagnostic Accuracy		91.07%		
Likelihood ratio of a Positive Test		23.28		
Likelihood ratio of a Negative Test		0.1432		
Diagnostic Odds		162.5		
Cohen's kappa (Unweighted)		0.8219		
Entropy reduction after a Positive Test		52.95%		
Entropy reduction after a Negative Test		29.98%		
Bias Index		-0.05357		
Etiologic fraction in pop.(EFp|OR)				
Etiologic fraction in exposed(EFe|OR)			99.38 %	94.11, 99.94

There is a statistically significant correlation between post prandial pain ad mesenteric ischemia.

		MESENTERIC ISCHEMIA		
		(+)	(-)	
PERIPHERAL VASCULAR DISEASE	(+)	23	2	25
	(-)	6	25	31
		29	27	56



Test	Value	p-value(2-tail)
Yates corrected chi square	26.41	0.000000276

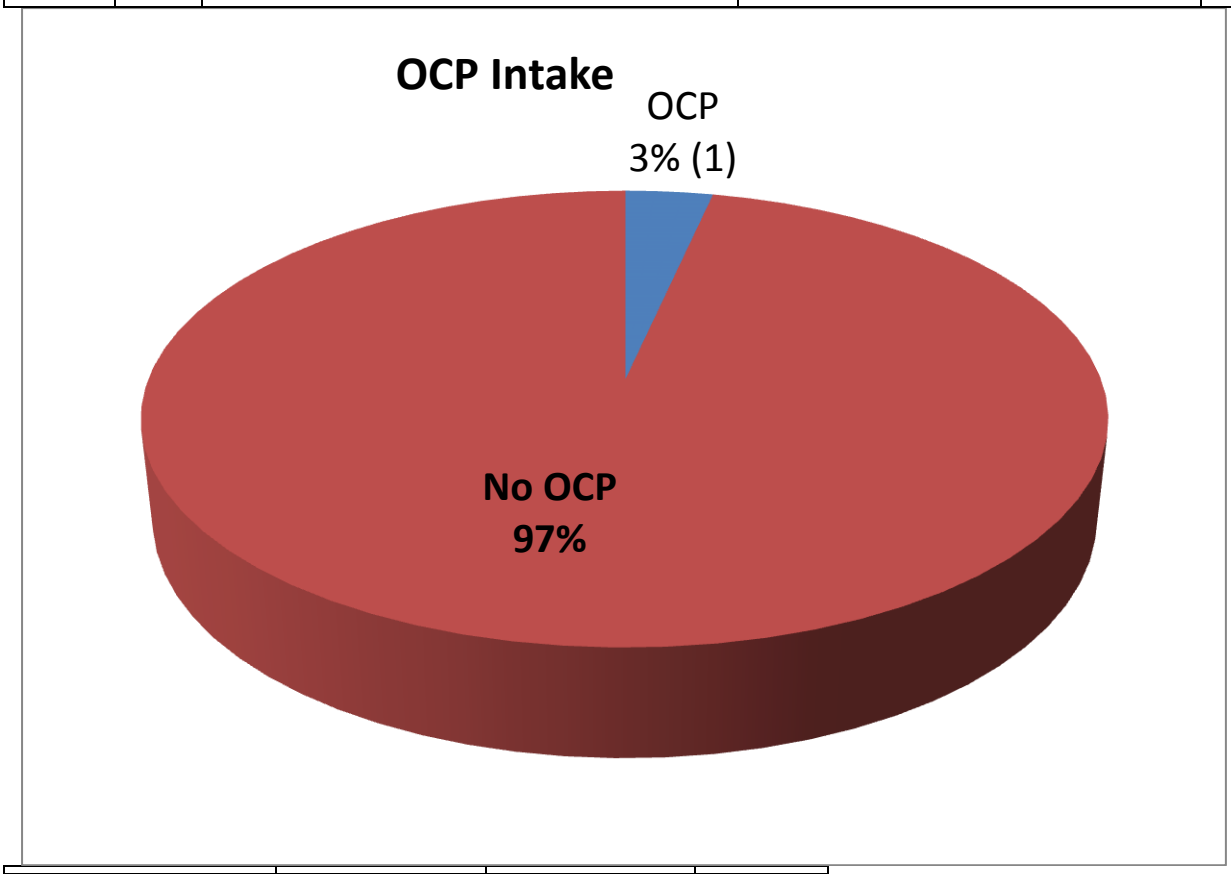
Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
Risk in Exposed	92%	73.9, 98.91
Risk in Unexposed	19.35%	8.818, 36.65
Overall Risk	51.79%	39.01, 64.33
Risk Ratio	4.753	2.296, 9.841[1]
Risk Difference	72.65%	55.14, 90.15°
Etiologic fraction in pop.(EFp)	62.63%	40, 85.25
Etiologic fraction in exposed(EFe)	78.96%	56.44, 89.84

Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
CMLE Odds Ratio*	43.03	8.943, 334.4[1]
		7.631, 475.3[1]
Odds Ratio	47.92	8.776, 261.6[1]
Etiologic fraction in pop.(EFp|OR)	77.66%	61.56, 93.75
Etiologic fraction in exposed(EFe|OR)	97.91%	88.61, 99.62

Parameter		Estimate	Lower - Upper 95% CIs	
Sensitivity		79.31%	(61.61, 90.15[1])	
Specificity		92.59%	(76.63, 97.94[1])	
Positive Predictive Value		92%	(75.03, 97.78[1])	
Negative Predictive Value		80.65%	(63.72, 90.81[1])	
Diagnostic Accuracy		85.71%	(74.26, 92.58[1])	
Likelihood ratio of a Positive Test		10.71	(3.93 - 29.17)	
Likelihood ratio of a Negative Test		0.2234	(0.1602 - 0.3117)	
Diagnostic Odds		47.92	(8.775 - 261.7)	
Cohen's kappa (Unweighted)		0.7154	(0.4561 - 0.9746)	
Entropy reduction after a Positive Test		41.37%		
Entropy reduction after a Negative Test		20.12%		
Bias Index		-0.07143		

There is a statistically significant correlation between PVD and mesenteric ischemia as evident from the diagram and analysis.

Single Table Analysis				
		MESENTERIC ISCHEMIA		
		(+)	(-)	
	(+)	1	2	3
OCP	(-)	28	25	53
		29	27	56



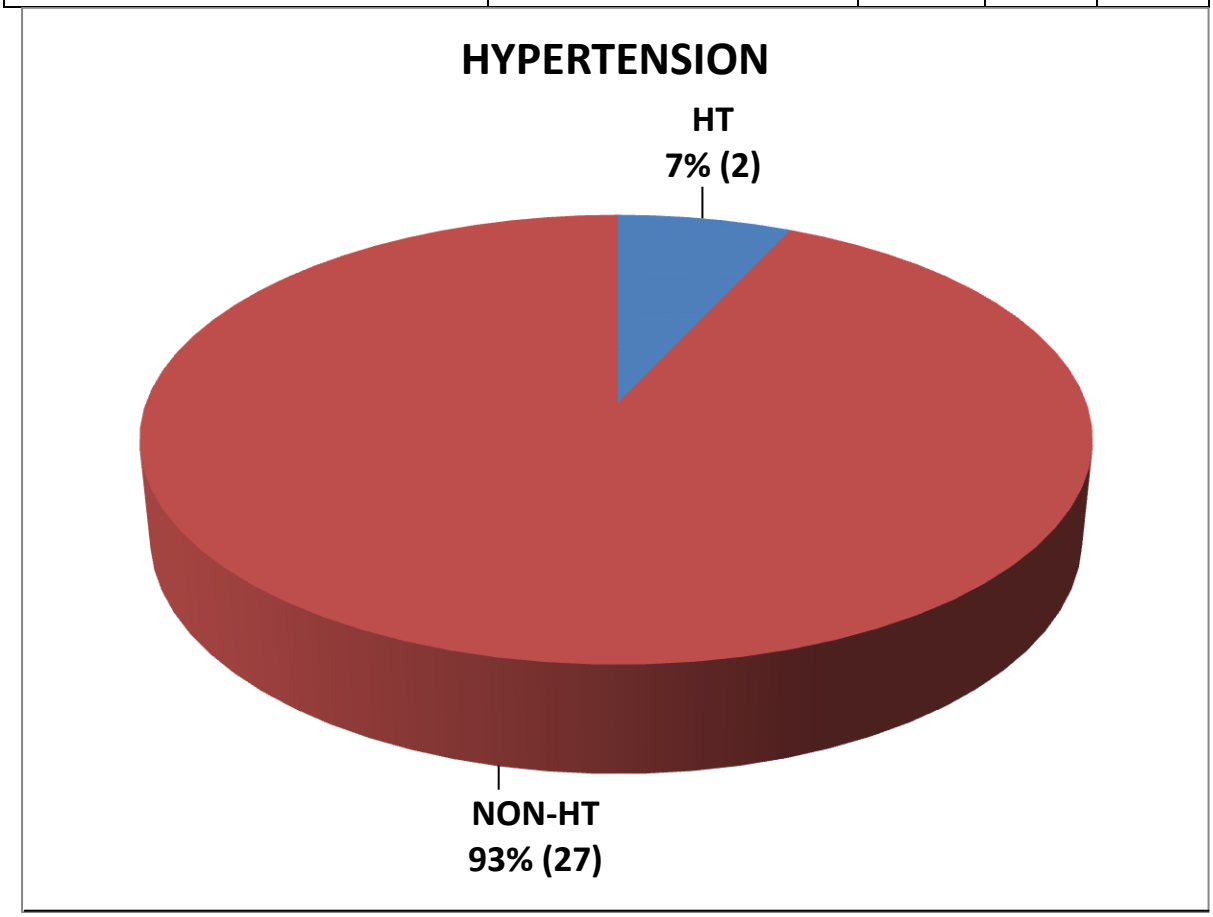
Test	Value	p-value(2-tail)
Yates corrected chi square	0.004048	0.9493

Point Estimates		Confidence Limits	
Type	**Value**	**Lower, Upper**	**Type**
Risk in Exposed	33.33%	5.628, 79.75	Taylor series
Risk in Unexposed	52.83%	39.66, 65.62	Taylor series
Overall Risk	51.79%	39.01, 64.33	Taylor series
Risk Ratio	0.631	0.1248, 3.189[1]	Taylor series
Risk Difference	-19.5%	-74.5, 35.51°	Taylor series
Prevented fraction in pop.(pfp)	1.977%	-4.293, 7.536	
Prevented fraction in exposed(pfe)	36.9%	-218.9, 87.52	

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	3.448%	(0.6113, 17.18[1])
Specificity	92.59%	(76.63, 97.94[1])
Positive Predictive Value	33.33%	(6.149, 79.23[1])
Negative Predictive Value	47.17%	(34.38, 60.34[1])
Diagnostic Accuracy	46.43%	(34.02, 59.3[1])
Likelihood ratio of a Positive Test	0.4655	(0.00000000000000000000000256 - 8465000000000000000000)
Likelihood ratio of a Negative Test	1.043	(0.9662 - 1.125)
Diagnostic Odds	0.4464	(0.03813 - 5.227)
Cohen's kappa (Unweighted)	-0.03832	(-0.1526 - 0.07591)
Entropy reduction after a Positive Test	5.6%	
Entropy reduction after a Negative Test	0.0965%	
Bias Index	-0.4643	

The sample size is too small to illustrate a significant correlation between ocp intake and mesenteric ischemia

MESENTERIC ISCHEMIA				
		(+)	(-)	
	(+)	2	8	10
HYPERTENSION	(-)	27	19	46
		29	27	56



Test	Value	p-value(2-tail)
Yates corrected chi square	3.498	0.06144

Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
CMLE Odds Ratio*	0.1813	0.02399, 0.8858[1]
		0.01695, 1.049[1]
Odds Ratio	0.1759	0.03356, 0.9223[1]
Prevented fraction in pop(PFpOR)	24.42%	-2.698, 40.2
Prevented fraction in exposed(PFeOR)	82.41%	7.769, 96.64

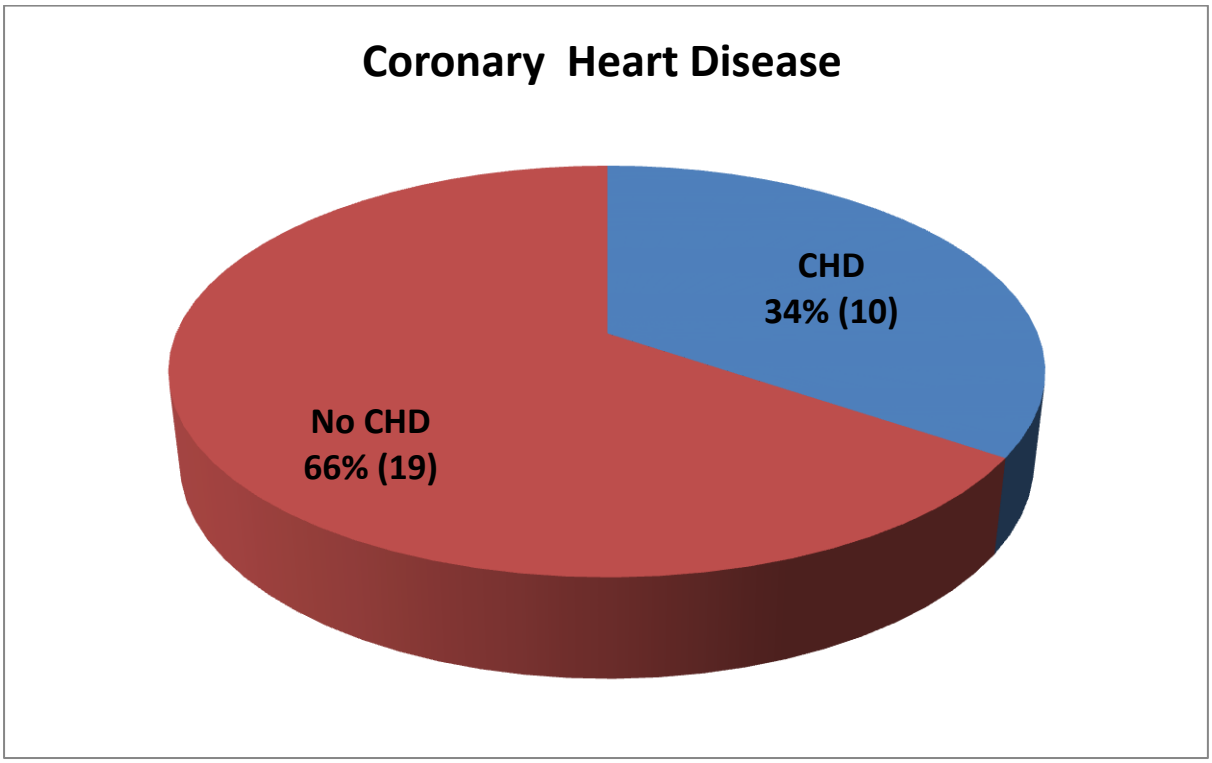
Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
Risk in Exposed	20%	4.589, 52.06
Risk in Unexposed	58.7%	44.33, 71.73
Overall Risk	51.79%	39.01, 64.33
Risk Ratio	0.3407	0.09636, 1.205[1]
Risk Difference	-38.7%	-67.28, -10.11°
Prevented fraction in pop.(pfp)	11.77%	0.318, 20.87
Prevented fraction in exposed(pfe)	65.93%	-20.49, 90.36

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	6.897%	(1.912, 21.96[1])
Specificity	70.37%	(51.52, 84.15[1])
Positive Predictive Value	20%	(5.668, 50.98[1])

The study reveals no significant correlation between mesenteric ischemia and hypertension as evident from the statistical analysis.

	MESENTERIC ISCHEMIA			
		(+)	(-)	
	(+)	10	8	18
CORONARY HEART DISEASE	(-)	19	19	38
		29	27	56

Negative Predictive Value	41.3%	(28.29, 55.66[1])	
Diagnostic Accuracy	37.5%	(26.01, 50.59[1])	
Likelihood ratio of a Positive Test	0.2328	(0.0000003272 - 165600)	
Likelihood ratio of a Negative Test	1.323	(1.178 - 1.486)	
Diagnostic Odds	0.1759	(0.03355 - 0.9224)	
Cohen's kappa (Unweighted)	-0.2219	(-0.4179 - -0.02595)	
Entropy reduction after a Positive Test	19.21%		
Entropy reduction after a Negative Test	1.456%		
Bias Index	-0.3393		



Test	Value	p-value(2-tail)
Yates corrected chi square	0.01046	0.9186

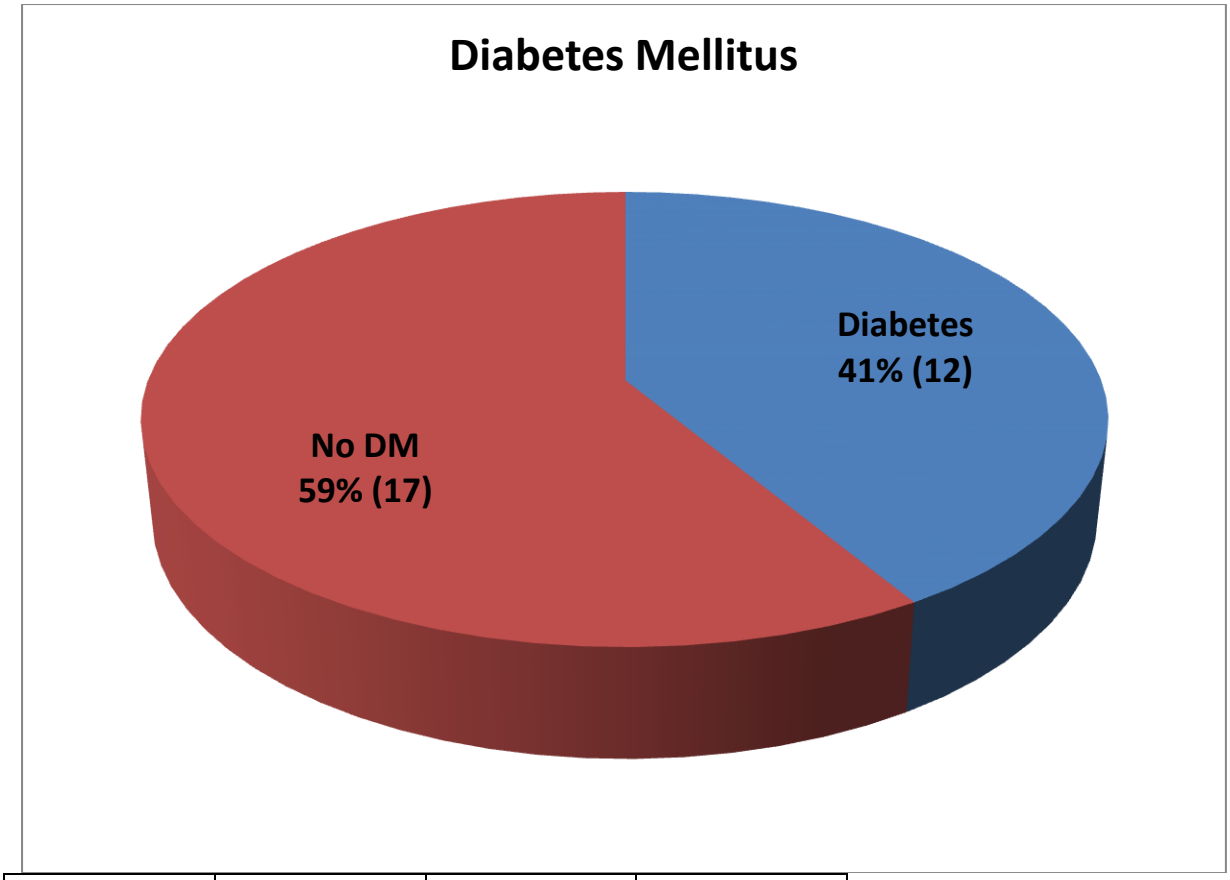
Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
Risk in Exposed	55.56%	33.7, 75.46
Risk in Unexposed	50%	34.85, 65.15
Overall Risk	51.79%	39.01, 64.33
Risk Ratio	1.111	0.6597, 1.871[1]
Risk Difference	5.556%	-22.37, 33.48°
Etiologic fraction in pop.(EFp)	3.448%	-13.96, 20.85
Etiologic fraction in exposed(EFe)	10%	-51.58, 46.56

Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
CMLE Odds Ratio*	1.245	0.3963, 3.987[1]
		0.352, 4.52[1]
Odds Ratio	1.25	0.4053, 3.855[1]
Etiologic fraction in pop.(EFp|OR)	6.897%	-26.62, 40.41
Etiologic fraction in exposed(EFe|OR)	20%	-100, 74.06

Parameter		Estimate	Lower - Upper 95% CIs	
Sensitivity		34.48%	(19.94, 52.66[1])	
Specificity		70.37%	(51.52, 84.15[1])	
Positive Predictive Value		55.56%	(33.72, 75.44[1])	
Negative Predictive Value		50%	(34.85, 65.15[1])	
Diagnostic Accuracy		51.79%	(39.01, 64.33[1])	
Likelihood ratio of a Positive Test		1.164	(0.6277 - 2.158)	
Likelihood ratio of a Negative Test		0.931	(0.8041 - 1.078)	
Diagnostic Odds		1.25	(0.4053 - 3.856)	
Cohen's kappa (Unweighted)		0.04786	(-0.1935 - 0.2893)	
Entropy reduction after a Positive Test		0.5548%		
Entropy reduction after a Negative Test		-0.06379 %		
Bias Index		-0.1964		

The study reveals no significant correlation between mesenteric ischemia and coronary heart disease as evident from the statistical analysis..

	MESENTERIC ISCHEMIA			
		(+)	(-)	
	(+)	12	12	24
DIABETES MELLITUS	(-)	17	15	32
		29	27	56



Test	Value	p-value(2-tail)
Yates corrected chi square	0.00149	0.9692

Point Estimates		Confidence Limits
Type	Value	Lower, Upper
Risk in Exposed	50%	31.43, 68.57
Risk in Unexposed	53.13%	36.45, 69.13
Overall Risk	51.79%	39.01, 64.33
Risk Ratio	0.9412	0.562, 1.576[1]
Risk Difference	-3.125%	-29.56, 23.31°
Prevented fraction in pop.(pfp)	2.521%	-23.95, 19.67
Prevented fraction in exposed(pfe)	5.882%	-57.63, 43.8

	Type	Value	
	CMLE Odds Ratio*	0.8843	
	Odds Ratio	0.8824	
	Prevented fraction in pop(PFpOR)	5.229%	
	Prevented fraction in exposed(PFeOR)	11.76%	

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	41.38%	(25.51, 59.26[1])
Specificity	55.56%	(37.31, 72.41[1])
Positive Predictive Value	50%	(31.43, 68.57[1])
Negative Predictive Value	46.88%	(30.87, 63.55[1])
Diagnostic Accuracy	48.21%	(35.67, 60.99[1])
Likelihood ratio of a Positive Test	0.931	(0.6274 - 1.382)
Likelihood ratio of a Negative Test	1.055	(0.8469 - 1.315)
Diagnostic Odds	0.8824	(0.3059 - 2.545)
Cohen's kappa (Unweighted)	-0.03046	(-0.2882 - 0.2273)
Entropy reduction after a Positive Test	-0.06379%	
Entropy reduction after a Negative Test	0.1317%	
Bias Index	-0.08929	

There is no significant correlation between mesenteric ischemia and diabetes mellitus as illustrated.

	MESENTERIC ISCHEMIA			
		(+)	(-)	
	(+)	2	19	21
CHRONIC ABDOMINAL PAIN	(-)	27	8	35
		29	27	56

Test	Value	p-value(2-tail)
Yates corrected chi square	21.4	0.000003721

Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
Risk in Exposed	9.524%	1.446, 30.12
Risk in Unexposed	77.14%	60.74, 88.17
Overall Risk	51.79%	39.01, 64.33
Risk Ratio	0.1235	0.03264, 0.467[1]
Risk Difference	-67.62%	-86.36, -48.88°
Prevented fraction in pop.(pfp)	32.87%	17.2, 43.55
Prevented fraction in exposed(pfe)	87.65%	53.3, 96.74

Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
CMLE Odds Ratio*	0.03391	0.004505, 0.158[1]
		0.003181, 0.1843[1]
Odds Ratio	0.03119	0.005949, 0.1635[1]
Prevented fraction in pop(PFpOR)	68.18%	-579.3, 74.04
Prevented fraction in exposed(PFeOR)	96.88%	83.65, 99.41

Parameter	Estimate	Lower - Upper 95% CIs	
Sensitivity	6.897%	(1.912, 21.96[1])	
Specificity	29.63%	(15.85, 48.48[1])	
Positive Predictive Value	9.524%	(2.652, 28.91[1])	
Negative Predictive Value	22.86%	(12.07, 39.02[1])	
Diagnostic Accuracy	17.86%	(10, 29.84[1])	
Likelihood ratio of a Positive Test	0.098	(0.0000001588 - 60500)	
Likelihood ratio of a Negative Test	3.142	(1.633 - 6.046)	
Diagnostic Odds	0.03119	(0.005948 - 0.1635)	
Cohen's kappa (Unweighted)	-0.6283	(-0.8795 - -0.3771)	
Entropy reduction after a Positive Test	37.8%		
Entropy reduction after a Negative Test	15.5%		
Bias Index	-0.1429		

There is a statistically significant correlation between chronic abdominal pain and mesenteric ischemia.

MESENTERIC ISCHEMIA				
		(+)	(-)	
	(+)	12	15	27
WEIGHT LOSS	(-)	17	12	29
		29	27	56

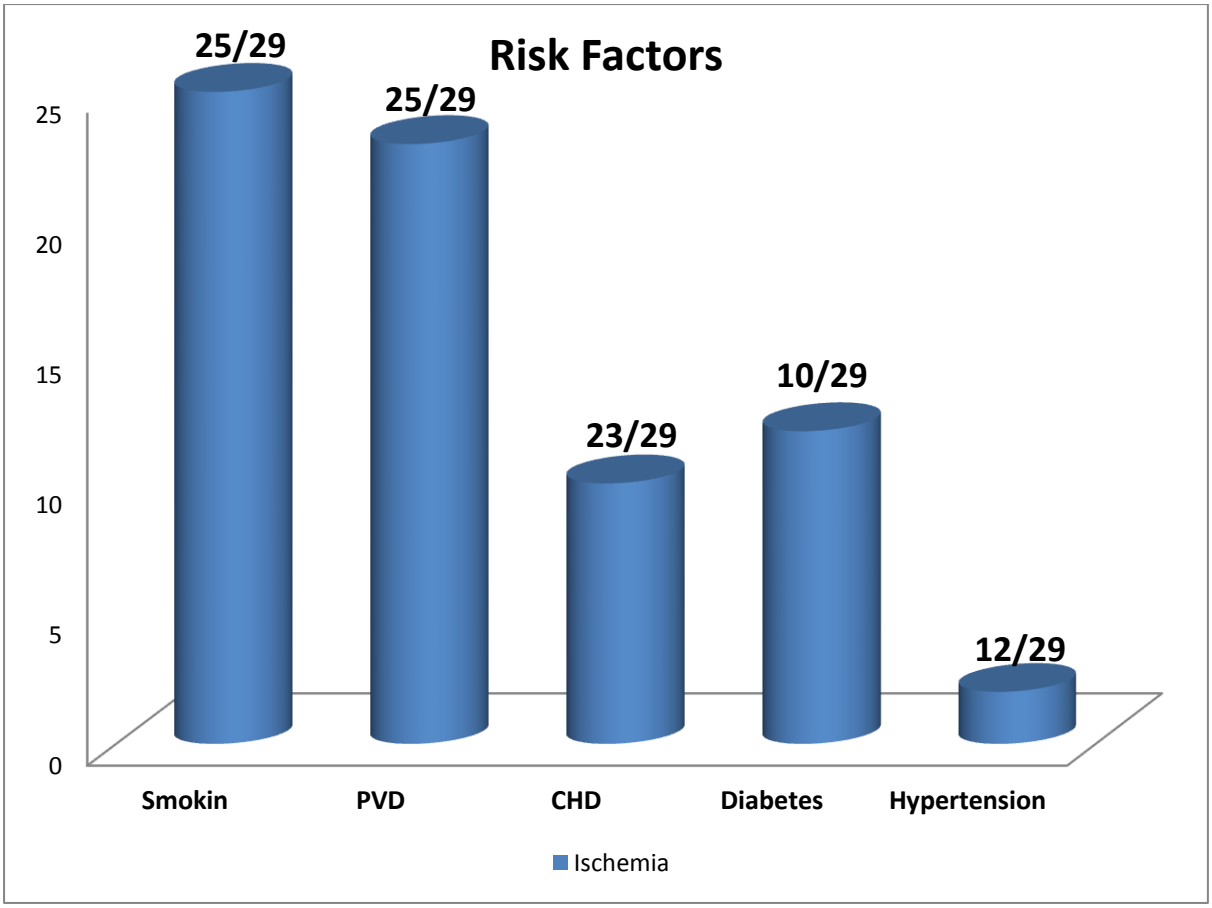
Test	Value	p-value(2-tail)
Yates corrected chi square	0.6292	0.4276

	Point Estimates		Confidence Limits
	Type	**Value**	**Lower, Upper**
	Risk in Exposed	44.44%	27.57, 62.7
	Risk in Unexposed	58.62%	40.71, 74.51
	Overall Risk	51.79%	39.01, 64.33
	Risk Ratio	0.7582	0.4504, 1.276[1]
	Risk Difference	-14.18%	-40.11, 11.76°
	Prevented fraction in pop.(pfp)	11.66%	-13.04, 27.5
	Prevented fraction in exposed(pfe)	24.18%	-27.64, 54.96

Point Estimates		Confidence Limits
Type	**Value**	**Lower, Upper**
CMLE Odds Ratio*	0.5706	0.1919, 1.66[1]
		0.1715, 1.844[1]
Odds Ratio	0.5647	0.1958, 1.629[1]
Prevented fraction in pop(PFpOR)	24.18%	23.84, 50.15
Prevented fraction in exposed(PFeOR)	43.53%	-62.89, 80.42

Parameter		Estimate	Lower - Upper 95% CIs
Sensitivity		41.38%	(25.51, 59.26[1])
Specificity		44.44%	(27.59, 62.69[1])
Positive Predictive Value		44.44%	(27.59, 62.69[1])
Negative Predictive Value		41.38%	(25.51, 59.26[1])
Diagnostic Accuracy		42.86%	(30.77, 55.86[1])
Likelihood ratio of a Positive Test		0.7448	(0.5186 - 1.07)
Likelihood ratio of a Negative Test		1.319	(0.9583 - 1.815)
Diagnostic Odds		0.5647	(0.1958 - 1.629)
Cohen's kappa (Unweighted)		-0.1414	(-0.4026 - 0.1198)
Entropy reduction after a Positive Test		0.5548%	
Entropy reduction after a Negative Test		1.43%	
Bias Index		-0.03571	

There exists a no statistically significant correlation between mesenteric ischemia and weight loss.



Out of the total 29 cases which were positive 9 cases had an acute presentation while 20 cases were identified while screening for risk factors.

This chart illustrates the various risk factors prevalent in acute cases and cases that were screened.

These patients were put on anticoagulant therapy with the treatment of the underlying disorders and were regularly followed up. This clearly revealed a significant reduction in morbidity in these patients thus signifying the importance of this study.

CONCLUSION:

This study clearly reveals a substantial association between mesenteric ischemia and smoking, postprandial abdominal pain and PVD.

Hence it is mandatory to screen symptomatic smokers, patients with postprandial angina and patients with PVD for mesenteric vascular occlusion.

Prophylactic anticoagulant therapy along with treatment of the underlying cause goes a long way in improving the lifestyle of these patients as evident from the study.

Our study also revealed stenosis of celiac and renal vasculature in 2 patients. Hence, apart from mesenteric ischemia, occlusion of other vasculature can also be diagnosed at an earlier stage and intervened at the right time to prevent future complications.

Hence , high index of suspicion is necessary for earlier diagnosis and decreasing morbidity and mortality in patients.

LIMITATIONS OF THE STUDY:

As our sample size is small, a future research involving a larger sample size is mandatory to validate the results of this project.

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Master Chart

S.no	Name	Age	Sex	Smoker	Post prandial pain	Weight loss	Pvd	Ocp intake	Hypertension	Coronary heart disease	Chronic abd.pain	Diabetes	Ischemia	Vessel (S-SCREENING; A-ACUTE)
1.	Rangasamy	54	m	No	No	No	No	No	Yes	No	Yes	No	No	
2.	Patteswaran	58	m	No	No	No	No	No	No	No	Yes	Yes	No	COELIAC(S)
3.	Marappan	62	m	No	No	No	No	No	No	No	*Yes*	Yes	No	
4.	Ramasamy	48	m	No	No	No	No	No	No	Yes	Yes	Yes	No	AORTA & RENAL (S)
5.	Rajaram	58	m	No	No	Yes	No	No	No	Yes	No	Yes	No	
6.	Jayaraman	63	m	No	No	No	No	No	No	No	Yes	Yes	No	
7.	Natarajan	60	m	No	No	Yes	No	No	No	Yes	Yes	Yes	Yes	SMA(S)
8.	Jeevanandan	55	m	No	No	Yes	No	No	Yes	No	Yes	No	No	
9.	Velammal	50	f	No	Yes	No	No	No	Yes	Yes	No	No	Yes	AORTA & SMA(S)
10.	Nageshwari	41	f	No	Yes	No	No	Yes	No	No	No	No	Yes	SMA(S)
11.	selvamuthukumaran	53	m	No	Yes	No	Yes	No	No	No	No	No	Yes	SMA(S)
12.	Uthiramani	54	f	No	No	Yes	No	No	Yes	No	Yes	No	No	
13.	Logeswari	37	f	No	No	No	No	Yes	No	No	Yes	No	No	
14.	Narayanan	50	m	No	No	Yes	No	No	No	Yes	Yes	No	No	
15.	Kalaiyarasan	53	m	No	No	Yes	No	No	Yes	No	No	Yes	No	
16.	marudammal	56	f	No	No	No	No	No	No	Yes	Yes	No	Yes	SMA(A)
17.	Vijayakumaran	52	m	No	No	No	No	No	No	Yes	No	Yes	No	
18.	maran	52	m	No	No	Yes	No	No	No	No	Yes	Yes	No	

Master Chart

19.	narayanamoorthy	48	m	No	No	No	No	No	Yes	No	Yes	No	No	
20.	venkateswaran	52	M	No	No	Yes	No	No	Yes	No	Yes	No	No	COELIAC (S)
21.	ramakrishnan	50	m	No	No	Yes	No	No	Yes	No	Yes	No	No	
22.	Kalaiselvi	42	f	No	No	No	No	Yes	No	No	Yes	No	No	
23.	mohammed hanifa	60	M	No	No	Yes	No	No	No	Yes	Yes	No	No	
24.	Thiruselvan	50	M	No	No	Yes	No	No	Yes	No	No	Yes	No	
25.	Sengottayan	60	m	Yes	No	No	No	No	No	Yes	No	No	No	
26.	Arumugam	58	m	Yes	No	Yes	No	No	No	No	Yes	Yes	No	COELIAC (S)
27.	Kumaran	42	f	Yes	No	No	No	No	No	No	Yes	No	No	
28.	Periyasamy	49	m	Yes	No	Yes	No	No	No	Yes	Yes	No	No	
29.	Arumugam	60	m	Yes	No	Yes	No	No	No	Yes	Yes	No	No	
30.	Kusalavan	57	m	Yes	No	No	No	No	Yes	No	No	No	Yes	SMA(A)
31.	Ramasamy	48	m	Yes	Yes	No	No	No	No	No	No	No	Yes	SMA(S)
32.	Arumugam	54	m	Yes	Yes	No	Yes	No	No	No	No	Yes	Yes	SMA(S)
33.	Patteswaran	58	m	Yes	Yes	No	Yes	No	No	No	No	Yes	Yes	SMA(S)
34.	Saravanan	38	m	Yes	Yes	No	No	No	No	No	No	Yes	Yes	IMV(A)
35.	Marappan	62	m	Yes	Yes	No	Yes	No	No	Yes	No	Yes	Yes	SMA(S)
36.	Arumugam	50	m	Yes	No	Yes	Yes	No	No	Yes	No	Yes	Yes	SMA(S)
37.	balasubramaniam	58	m	Yes	Yes	Yes	Yes	No	No	Yes	No	Yes	Yes	SMA(S)
38.	Mohammad	58	m	Yes	Yes	No	Yes	No	No	No	No	Yes	Yes	SMA(A)
39.	subramaniyam	55	m	Yes	Yes	Yes	Yes	No	No	No	No	Yes	Yes	SMA(A)
40.	senthilkumar	48	m	Yes	Yes	Yes	Yes	No	No	No	No	Yes	Yes	SMA(A)

Master Chart

41.	Venugopal	60	m	Yes	Yes	No	Yes	No	No	No	No	Yes	Yes	SMA(S)
42.	Ramanujam	60	m	Yes	Yes	Yes	Yes	No	No	Yes	No	Yes	Yes	AORTA & SMA(S)
43.	Palanisamy	41	m	Yes	Yes	No	Yes	No	No	Yes	No	No	Yes	SMV(A)
44.	thirumurugan	50	m	Yes	Yes	Yes	Yes	No	No	Yes	No	No	Yes	SMV(A)
45.	Murugeshan	47	m	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	AORTA (s))
46.	radhakrishnan	50	m	Yes	Yes	No	Yes	No	No	No	No	No	Yes	PORTAL VEIN & SMV(S)
47.	Ramesh	48	m	Yes	Yes	No	Yes	No	No	Yes	No	No	Yes	PORTAL VEIN & SMV S)
48.	Veeramani	50	m	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	SMV(S)
49.	Saleem	49	m	Yes	Yes	No	Yes	No	No	No	No	No	Yes	SMV(S)
50.	udhayakumar	45	m	Yes	Yes	No	Yes	No	No	No	No	No	Yes	SMV(S)
51.	selvaputhiran	53	m	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	PORTAL VEIN & SMV (S)
52.	Palanisamy	52	m	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	IMA(S)
53.	poutharaman	51	m	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	SMA(S)
54.	Dhandapani	48	m	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	SMV(A)
55.	Ramadoss	54	m	Yes	No	Yes	Yes	No	No	No	No	Yes	No	
56.	syed Ibrahim	57	m	Yes	No	No	Yes	No	No	No	No	Yes	No	

A CLINICAL STUDY ON MESENTERIC VASCULAR OCCLUSIVE DISEASE

Dissertation submitted in partial fulfilment of regulation

for the award of

M.S.DEGREE IN GENERAL SURGERY