RISK FACTORS EFFECTING MORTALITY IN ACUTE MESENTERIC VASCULAR OCCLUSION - A SINGLE EXPERIENCE

Dissertation submitted in partial fulfillment of regulation for the award of
M.S.Degree in General Surgery (Branch I)

THE TAMILNADU

DR.M.G.R.MEDICAL UNIVERSITY

APRIL 2016

CERTIFICATE

Certified that this dissertation entitled **"RISK FACTORS EFFECTING MORTALITY IN ACUTE MESENTERIC VASCULAR OCCLUSION - A SINGLE EXPERIENCE"** is a bonafide work done by Dr.B.Venkatesh Prabhu, post graduate student of the Department of General Surgery, Coimbatore Medical College, Coimbatore, during the academic year 2013 - 2016. This work has not previously formed the basis for the award of any degree or diploma.

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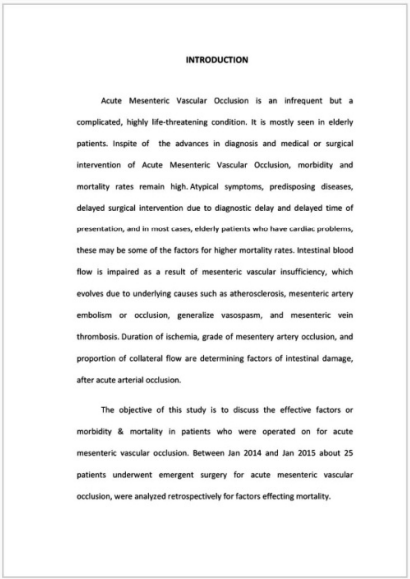

INTRODUCTION

Acute Mesenteric Vascular Occlusion is an infrequent but a complicated, highly life-threatening condition. It is mostly seen in elderly patients. Inspite of the advances in diagnosis and medical or surgical intervention of Acute Mesenteric Vascular Occlusion, morbidity and mortality rates remain high. Atypical symptoms, predisposing diseases, delayed surgical intervention due to diagnostic delay and delayed time of presentation, and in most cases, elderly patients who have cardiac problems, these may be some of the factors for higher mortality rates. Intestinal blood flow is impaired as a result of mesenteric vascular insufficiency, which evolves due to underlying causes such as atherosclerosis, mesenteric artery embolism or occlusion, generalize vasospasm, and mesenteric vein thrombosis. Duration of ischemia, grade of mesentery artery occlusion, and proportion of collateral flow are determining factors of intestinal damage, after acute arterial occlusion.

The objective of this study is to discuss the effective factors or morbidity & mortality in patients who were operated on for acute mesenteric vascular occlusion. Between Jan 2014 and Jan 2015 about 25 patients underwent emergent surgery for acute mesenteric vascular occlusion, were analyzed retrospectively for factors effecting mortality.


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INTRODUCTION

Acute Mesenteric Vascular Occlusion is an infrequent but complicated, life-threatening condition, mostly seen in elderly patients. Despite the advances in diagnosis of Acute Mesenteric Vascular Occlusion, morbidity and mortality rates remain high. Atypical presenting symptoms, presence of predisposing diseases, delayed surgical intervention due to diagnostic difficulties and due to delayed presentation, and in most cases, elderly patients who have cardiac problems, these may be some of the factors for higher mortality rates. Intestinal blood flow is impaired as a result of mesenteric vascular insufficiency, which evolves due to underlying causes such as atherosclerosis, mesenteric artery embolism, generalize vasospasm, and mesenteric vein thrombosis. Duration of ischemia, grade of mesentery artery occlusion, and proportion of collateral flow are determining factors of intestinal damage, after acute arterial occlusion.

The objective of this study is to discuss the effective factors or morbidity & mortality in patients who were operated on for acute mesenteric vascular occlusion. Between Jan 2014 and Jan 2015 about 25 patients underwent emergent surgery for acute mesenteric vascular occlusion, were analyzed retrospectively for factors effecting mortality.

HISTORICAL REVIEW

1. **Sushrutha 6th** century B.C wrote the oldest known descriptions about bowel surgery. Described using a chemical cautery over the swelling of strangulated hernias. Used the tentacles of black ants to clamp the edges of bowel wounds together.

2. **Fabricius d'Aquapcndente** 12th century As reported by Duverger, he described a procedure of intestinal repair involving end-to-end anastomosis

3. **Lanfranc** 13th century Used animal tracheas to connect divided segments of bowel.

4. **Ranidchr** 1727 Removed two feet of gangrenous small bowel and invaginated the proximal end of the bowel into the lumen of the distal segment, securing the connection with a few sutures.

5. **Travers** 1812 While experimenting with suture techniques, he noted that wounds closed with sutures that passed through all layers of the bowel wallhealed well.

6. **Jobert** 1824 Performed end-to-side anastomosis in dogs and cats using continuous wax suture,

7. **Lembert** 1826 Developed a suture technique employing interrupted sutures that passed through the entire bowel wall except for the mucous membrane.

8. **Schwartz** 1911 Used x-ray films to determine areas of intestinal distension

9. **Klass** 1950 Diagnosed mesenteric ischemia before infarction. Performed embolectomy without intestinal resection (patient died of acute heart failure).

10. **Shaw & Rutledge** 1957 Reported successful superior mesenteric vein embolectomy without bowel resection.

11. **Endc** 1958 First description of non occlusive mesenteric ischemia.

AIM OF STUDY

Objectives:

1. To identify risk factors effecting mortality in acute mesenteric vascular occlusion.
2. To study the level and extent of bowel involvement in acute mesenteric vascular occlusion.
3. To study the morbidity and mortality after surgical intervention for acute mesenteric vascular occlusion.

REVIEW OF LITERATURE

ANATOMY OF SMALL INTESTINE

The small intestine is the longest part of the alimentary canal. It extends from the gastric pylorus to the ileocecal junction .It is divided into three parts: duodenum, jejunum, and ileum.[27]

DUODENUM

The duodenum is a C-shaped tube, which joins the stomach to the jejunum is about 25 cm long. The duodenum curves around the head of the pancreas. The first 2.5 cm of the duodenum resembles the stomach in that it is covered on its anterior and posterior surfaces with peritoneum and has the lesser omentum attached to its upper border and the greater omentum attached to its lower border; the lesser sac lies behind this short segment.[27] The remainder of the duodenum is retroperitoneal, being only partially covered by peritoneum. Duodenum is divided into four parts.

FIRST PART OF THE DUODENUM

The duodenal first part begins at the pylorus and runs upward and backward on the transpyloric plane at the level of the first lumbar vertebra.

SECOND PART OF THE DUODENUM

The second part of the duodenum runs vertically downward in front of the hilum of the right kidney on the right side of the second and third lumbar

vertebrae. About halfway down its medial border, the common bile duct and the pancreatic duct pierce the duodenal wall[27]. They unite to form the ampulla of vater that opens on duodenal papilla at the level of its summit.

THIRD PART OF DUODENUM

The third part of the duodenum runs horizontally to the left on the subcostal plane, passing in front of the vertebral column and following the lower margin of the head of the pancreas.

FOURTH PART OF THE DUODENUM

The fourth part of the duodenum runs upward and to the left to the duodenojejunal flexure. This flexure is held in position by a fold of peritoneum, called the ligament of Treitz,[27] which originates from the right crus of the diaphragm.

BLOOD SUPPLY OF DUODENUM

ARTERIES

Arterial supply of the duodenum is by the superior pancreatico duodenal artery, which supplies the upper half and a branch of gastro duodenal artery. The lower half of the duodenum is supplied by the inferior pancreatico duodenal artery which is a branch of superior mesenteric artery.

VEINS

The superior pancreatico duodenal vein drains directly into the portal vein, where as the inferior pancreatico duodenal vein drains into the superior mesenteric vein.

LYMPH DRAINAGE

The lymphatic drainage courses along the course of the arteries, and drain upward via pancreaticoduodenal nodes, which drain to the gastroduodenal nodes and then to the celiac nodes and downward via pancreaticoduodenal nodes to the superior mesenteric nodes[27] where the superior mesenteric artery originates.

NERVE SUPPLY

The nerves are derived from sympathetic and parasympathetic (vagus) nerves from the celiac and superior mesenteric plexuses.

JEJUNUM AND ILEUM

The jejunum and ileum measure about 20 ft (6 m) long; the upper two fifths is jejunum. Each has distinctive features, but there is a gradual change from one to the other. The jejunum begins at the duodenojejunalflexure, and the ileum ends at the ileocecal junction.[27]

The coils of jejunum and ileum are freely mobile and are attached to the posterior abdominal wall by a fan-shaped fold of peritoneum known as the mesentery. The long free edge of the fold encloses the small and large bowels. The short root of the fold is continuous on the posterior abdominal wall with the parietal peritoneum that extends downward and to the right from the left side of the second lumbar vertebra to the region of the right sacroiliac joint.[27]

The root of the mesentery permits the entrance and exit of the branches of the superior mesenteric artery and vein, lymphvessels, and nerves into the pace between the two layers of peritoneum forming the mesentery.

DIFFERENCE BETWEEN JEJUNUM AND ILEUM	
JEJUNUM	**ILEUM**
Thick wall	Thin wall
Large lumen	Small lumen
Fat on mesentery	Fat on ileum & mesentery
Prominent plicae circulars	Less prominent plicae
Single arterial arcade	Multiple arterial arcade
Sparse lymph node aggregate	Frequent lymph aggregate

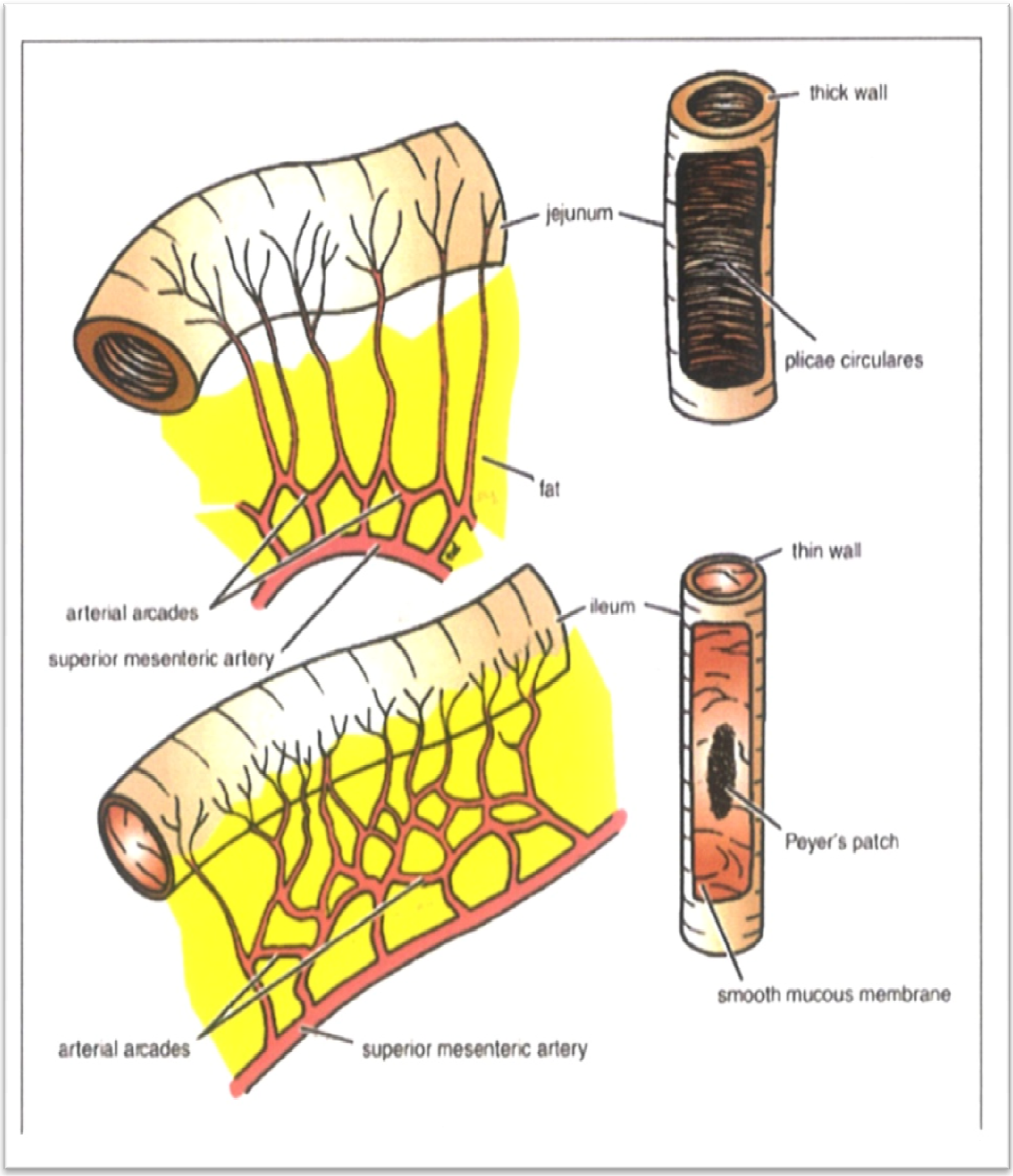


Figure : 1 Difference Between Jejunum and Ileum

Anatomic divisions of the large intestine:

1. Colon
2. Rectum
3. Anal canal

Anatomical Layers;

1. Mucosa
2. Submucosa
3. The inner circular muscle layer which unites didtally to form the internal anal sphincter.
4. The outer longitudinal muscle layer which condenses to form three teniae coli over the colon. The teniae converge proximally at the appendix and distally at the rectum.
5. Serosa – the outermost layer which covers the intra peritoneal colon and the proximal one third of rectum.[27]

1. SMA branches;

a. Ileocolic artery supplies blood flow to the distal ileum and proximal part of ascending colon.

b. Right colic artery and the middle colic artery supplies the ascending colon and the transverse colon respectively.[27]

2. Inferior mesenteric artery branches

a. Left colic artery and the sigmoidal artery supply the descending colon and the sigmoid colon respectively.

b. The proximal rectum is supplied by superior rectal artery.

The final branches of these arteries anastamose with the branches of the adjacent artery and communicate via the marginal artery of Durammond.

3. Internal iliac artery branches in to

a. Middle rectal artery

b. Internal pudendal artery

LYMPH DRAINAGE

Through many intermediate mesenteric nodes reach the superior mesenteric nodes, which are situated where the SMA originates.

NERVE SUPPLY

The nerves are derived from the sympathetic and parasympathetic (vagus) nerves from the superior mesenteric plexus. Sympathetic arise from the roots T6-T12 and L1-L3.

2. Parasympathetic innervation is from the vagus nerve and sacral nerves S2–S4.[27]

SUPERIOR MESENTERIC ARTERY (SMA)

The SMA usually arises at a 20- to 30-degree angle from the anterior aspect of the aorta opposite the upper third of the body of LI, 5 to 15 mm caudal to the celiacartery. At its origin, the SMA measures about 1 cm in diameter, AS it passes forward and downward, it emerges from beneath the inferior surface of the body of thepancreas and courses anterior to the third portion of the duodenum and uncinate process of the pancreas.[27]

Constant branches of the SMA include the inferior pancreaticoduodenal, the middle colic, the right colic, the ileocolic, and the intestinal arteries. The inferior pancreaticoduodenal artery arises on the right side and communicates with pancreaticoduodenal branches from the gastroduodenal branch of the hepatic artery. The middle colic artery arises just distal to the inferior pancreaticoduodenal along the inferior border of the pancreas. This vessel is an important landmark when managing SMA occlusive problems. The right branch of the middle colic artery anastomoses with the ascending limb of the right colic artery and with the left branch of the middle colic artery, which comes from the inferior mesenteric circulation. The ileocolic artery may arise from the SMA either separately or in a common trunk with the right colic artery. Intestinal arterial branches supply the jejunum and ileum and vary from 12 to 20 in number. They originate from the left side of the SMA after it enters the mesentery.[27,28]

BLOOD SUPPLY OF SMALL INTESTINE BY SUPERIOR MESENTERIC ARTERY

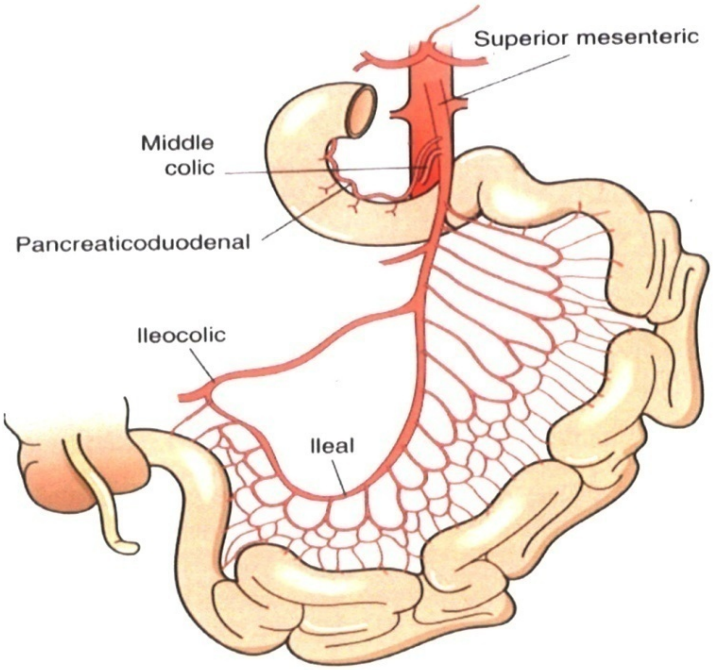
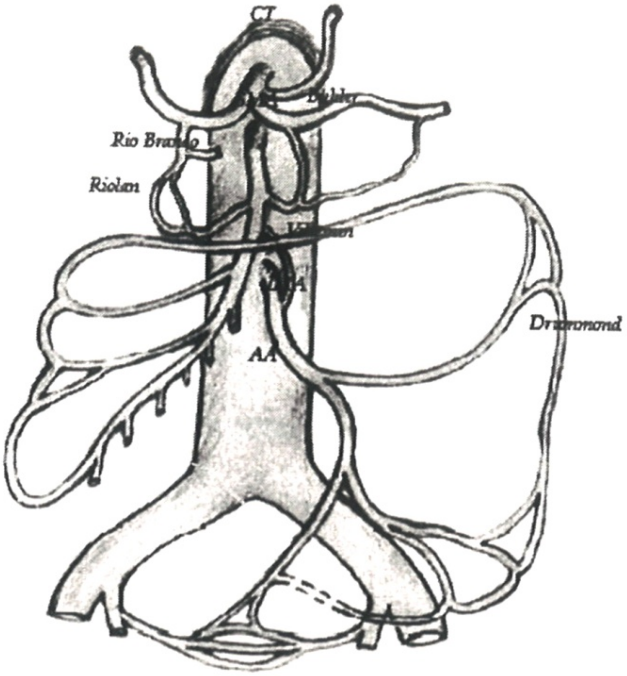


Figure : 2 Branches of Superior mesentery artery



CELIAC-SUPERIOR MESENTERIC COMMUNICATIONS

A common origin of the SMA and the eeliac artery occurs in 1% of individuals. In addition, between these two vessels may be a direct communicating channel, known as the **anastomotic artery of Buhler,** which is a remnant of anembryologic connection between these two arteries. Other important connections between individually arising superior mesenteric and eeliac trunks are the superiorand inferior pancreaticoduodenal arcades. Communication with middle colic arterialbranches from the SMA occasionally occurs through the dorsal pancreatic branch of the splenic artery. After complete occlusion of the eeliac axis, generousnimunications by the pancreaticoduodenal loop maintain hepatic and gastric circulations.[27]

SUPERIOR MESENTERIC – INFERIOR MESENTERlARTERIES COMMUNICATION

The Meandering Mesenteric Artery connects the ascending branch of the left colic artery directly by a central anastomotic vessel to the SMA circulation with a branch arising from the SMA just proximal to the origin of the middle colic artery The meandering mesenteric artery is potentially present in about two thirds of normal individuals.

The marginal artery of Drummond, first described by Von Haller in 1786 connects the left branch of the middle colic with the ascending branch of the left colicartery. At the splenic flexure, the left branch of the middle colic artery and the leftcolic artery from the IMA anastomose to provide continuity to the marginal artery of Drummond. This anastomotic site is Griffiths' point.[27]

REGULATION OF MESENTERIC BLOOD FLOW

Active vasodilation results from lysis of basal intrinsic smooth muscle tone. Vasoconstriction results from changes in opposing constrictor and dihtor forces favoring smooth muscle contraction. Changes in intestinal blood flow are influenced by numerous extrinsic and intrinsic factors operating simultaneously.

EXTRINSIC CONTROL

AUTONOMIC NERVOUS SYSTEM

Sympathetic stimulation causes vigorous contraction of arteriolar smoothmuscle resulting in significant reduction in intestinal blood flow. Redistribution of capillary perfusion may result from sympathetic stimulation of precapillary sphincters. The major physiologic role of sympathetic vasoconstriction of the gut is to decrease splanchnic blood flow during activities that require increased blood flow to skeletal muscle, heart, and brain. Continued sympathetic discharge may cause persistent mesenteric vasospasm even after the underlying cause of intestinal hypoperfusion has been corrected. In addition, redistribution of blood flow away from the mucosa, mediated by sympathetic nervous activity may account in part for susceptibility of mucosa to ischemic damage in various pathologic conditions involving the mesenteric circulation.[28]

DRUGS

Norepinephrine, methoxamine, metaraminol, and phenylephrine produces predominant alpha-adrenoreceptor stimulation and constricts mesenteric vasculature. Alphablocking agents such as phentolamine cause intestinal vasodilation. Systemic administration of phentolamine may result in decreased intestinal blood flow even though local mesenteric vasodilation occurs.

Isoproterenol, a beta-adrenoreceptor stimulant, increases intestinal blood. This action is blocked by propranolol, a beta-antagonist. Epinephrine, which has both alpha- and beta-adrenoreceptor stimulating action, in low concentrations causes intestinal vasodilation, whereas higher concentrations produce vasoconstriction as the alpha effects dominate beta-mediated vasodilation. Dopamine causes vasodilation by stimulating dopamincrgic receptors in mesenteric vessels. Dopamine levels greater than 10 ug/kg/min produce an alpha-stimulating effect that results in mesenteric vasoconstriction.

Histamine produces mesenteric vasodilation when it is administered ravenously or intra-arterially. Histamine causes contraction of nonvascularintestinal smooth muscle, an effect that may limit the increase in intestinal blood flow produced by vasodilation. Bradykinin produces intestinal vasodilation.[33]

Vasopressin and angiotensin II are peptides that produce potent vasoconstriction of the intestinal circulation selectively affecting the

splanchnic resistance vessels. Vasopressin is released as a result of systemic hypotension, and if the hypotension results from mesenteric ischemia, vasopressin may exacerbate vasoconstriction in the mesenteric vessels.

Smooth muscle relaxant drugs causing vasodilatation are tolazoline and papavcrine, sodium nitroprusside, sodium nitrite, caffeine and aminophylline.

Digoxin produces significant intestinal vasoconstriction and diminishes mesenteric blood flow.[26] Ergotamine causes increased vascular resistance. Prostaglandin E_1 stimulates formation of cAMP and causes vasodilation.[33]

GASTROINTESTINAL AND PANCREATIC HORMONES

The synthetic analog of gastrin, pentagastrin, reduces mesenteric vascular resistance and increases intestinal blood flow. Cholecystokinin has been reported to produce both vasoconstriction and vasodilation under varying conditions.

INTRINSIC REGULATION

Metabolic Regulation

Conditions resulting inexcessive oxygen demand relative to oxygen supply cause both accumulation of metabolites and diminished oxygen level in interstitial fluid and this produce relaxation of arteriolar smooth muscle and increase tissue perfusion. Oxygen supply and demand are thereby balanced.[28]

MYOGENIC REGULATION

Vascular smooth muscle tone is altered by arteriolar tension receptors in response to changes in transmural pressure. Increased vascular transmural pressure results in arteriolar vasoconstriction, increased vascular resistance, and diminished blood flow. Conversely, a decrease in transmural pressure causes vasodilation, diminished vascular resistance, and increased blood flow. The result of such regulation is maintenance of constant capillary pressure with minimal alterations in transcapillary fluid exchange. The myogenic control system is the principal factor in the protective mechanism termed autoregulation, which refers to the ability of the mesenteric circulation to maintain uniform total intestinal blood flow in the presence of widely varying systemic arterial blood pressures. Blood flow in intestinal villus vessels remains constant even when perfusion pressure is lowered from 100 mmHg to 30 mmHg.[33]

SPECIAL ASPECTS OF MESENTERIC CIRCULATION

REACTIVE HYPEREMIA

Both metabolic and myogenic factors probably contribute to intestinal vascular dilation that characteristically occurs after cessation of brief periods of sympathetic stimulation or mesenteric arterial occlusion. The magnitude and duration of this reactive hyperemia are directly related to duration of decreased perfusion. Thehyperemic response occurs uniformly throughout the bowel wall after an occlusionperiod of less than 1 minute.

Increasing the duration of ischemia results in hyperemia localized predominantly to the muscularis layer.

POSTPRANDIAL HYPEREMIA

During the initial phase of food intake, anticipation and ingestion of a meal are associated with increased mesenteric vascular resistance, probably caused by generalized sympathetic activity. In the second phase, digestion of food and absorption of chyme result in decreased mesenteric vascular resistance. This decreased resistance leads to an increase in superior mesenteric artery blood flow, which may be double resting blood flow. Decrease in iliac artery blood flow during digestion suggests redistribution of cardiac output to the mesenteric circulation at the expense of limb blood flow. At maximal blood flow during digestion, the small bowel receives most of the blood, 700 ml/100 g or more. The stomach receives 300 to 400 ml/100 g. and the colon receives 200 to 250 ml/100 g. Postprandial intestinal hyperemia correlates with the postprandial abdominal pain characteristically experienced by patients with chronic intestinal ischemia.[25]

AUTOREGULATORY ESCAPE

Autoregulatory escape is a compensatory mechanism that accounts for maintenance of intestinal blood flow at nearly normal levels, even in the presence of vasoconstrictor influences of continued sympathetic activity or prolonged catecholamine stimulation. Autoregulatory escape probably occurs because local Metabolic vasodilator mechanisms, elicited by ischemia, become

predominant over continued sympathetic vasoconstriction. Subsequent reversal of effects of vasoconstriction and restoration of required blood flow are greater in submucosa than ucosa. Therefore, there may be a relative redistribution of blood flow away from mucosa. This phenomenon may explain preferential ischemic damage to the mucosa.[33]

MUCOSAL COUNTERCURRENT EXCHANGE MECHANISMS

The architecture of the microcirculation in the intestinal villus accounts for the existence of a countercurrent exchange mechanism. Blood flow in the central part of the villus arteriole is parallel but opposite in direction to that in the subepithelialvenous capillaries. Consequently, a gradient in oxygen tension exists between arteriole and venule. This gradient is most prominent at the base of the villus. Diffusion along the gradient results in a progressive decrease in oxygen tension as blood flows from the base to the tip of the villus. This progressive diffusion gradient is accentuated by conditions that cause low mesenteric flow rates. The counter-current exchange mechanism, therefore, tends to aggravate tissue hypoxia in ischemic conditions of the bowel.[33]

COLLATERAL BLOOD FLOW

When a major mesenteric artery becomes occluded, diminished arterial pressure distal to the obstruction stimulates collateral pathways to open promptly. Blood flow through collateral vessels continues as long as pressure in the vascular bed distal to the occlusion remains lower than systemic pressure. Likewise, if the major vessel occlusion is corrected, blood flow through collateral channels ceases.

INTESTINAL INTRALUMINAL PRESSURE

During an ischemic insult the intraluminal pressure increases and this explainecolicky abdominal pain associated with early bowel ischemia. Bowel distensionsea by increased intraluminal pressure may diminish blood flow to the involvedsegment or to the entire intestine. An associated shunting of blood occurs away frommucosa and muscularispropria. The well-perfused serosa imparts a normal pink external appearance to the bowel, even though total intestinal blood flow may be markedly reduced. Diminution in flow may persist for hours after relief of distension. These findings emphasize the importance of nasogastric suction for decompression of the bowel in managing intestinal ischemia.[33]

RESPONSE TO ISCHEMIA

Ultrastructural changes in mucosal cells are evident within 10 minutes of acute superior mesenteric artery occlusion, and extensive histologic changes

occur within 30 minutes. Progression of these changes ultimately results in bowel necrosis. Important consequences of bowel ischemia are increased transcapillary filtration, interstitial edema, and ultimately the net movement of fluid into the bowel lumen. The enzyme xanthine oxidase reacts with hypoxanthine, which accumulates because of catabolism of ATP, and molecular oxygen to produce the cytotoxic oxygen radicals. The cytotoxic effects of the oxygen radicals presumably result from peroxidation of the lipid components of cellular and mitochondrial membrane.[25]

Polymorphonuclcar leukocytes appear to contribute to repcrfusion injury by releasing lysosomal enzymes at the site of ischemic injury and by production of oxygen free radicals through ncutrophil nicotinamide adeninc dinucleotide phosphate oxidase.

Acute mesenteric vascular occlusion is a syndrome, an acute life threatening condition causing necrosis and eventually gangrene of the bowel due to occlusion of the vascular tree .

AMI can be classified as being due to arterial cause or venous cause. The arterial causes of AMI can be further divided into nonocclusive mesenteric ischemia (NOMI) and occlusive mesenteric arterial ischemia (OMAI). Further OMAI can be due to acute mesenteric arterial thrombosis (AMAT) and acute mesenteric arterial embolism (AMAE). Venous cause involves mesenteric venous thrombosis (MVT). Thus practically there are 4 types of AMI clinically which are as follows:

- NOMI
- AMAE
- AMAT
- MVT

Secondarily mesenteric ischemia can occur due to mechanical obstruction (eg: from, intussusceptions, volvulus, strangulated internal hernia), dissection of the aorta and tumor compression. Rarely, blunt trauma can lead to intestinal infarction due to isolated **SMA** injury.[1]

ETIOLOGY

Acute mesenteric arterial embolism

Causes includes:

- Cardiac emboli - Auricular thrombus from atrial fibrillation, Mural thrombus following myocardial infarction, or less frequently valvular endocarditis leading to septic emboli.
- Ruptured atheromatous plaque leading to emboli from aortic thrombus fragments.
- Dislodged plaques by surgery or arterial catheterization.
- The most common site of emboli in the SMA is mostly where the middle colic artery originates. Thrombosis is more frequently seen with

ischemic disease than embolisation. Optimal treatment of embolisation has a good survival rate.[7]

ACUTE MESENTERIC ARTERIAL THROMBOSIS

Causes includes:

- Atherosclerotic vascular disease (most common)
- Dissection of aorta
- Aneurysm of the aorta
- Arteritis
- MI or CCF causing reduced cardiac output.
- Dehydration due to any cause[1]

Mesenteric venous thrombosis

Causes include –

Hypercoagulability from protein C and S deficiency, antithrombin III deficiency, factor V Leiden mutation, plasminogen abnormality, dysfibrinogenemia, polycythemia vera thrombocytosis,pregnancy and oral contraceptive use

- paraneoplastic syndromes
- Intra-abdominal infections such as appendicitis or diverticulitis.

- portal hypertension leading to venous congestion.
- Venous trauma (surgery or accident)
- Elevated intraabdominal pressure I laparoscopic surgery.
- Pancreatitis
- Decompression sickness[2]

PATHOPHYSIOLOGY

Inadequate blood supply of the small and large intestine may be caused due to arterial occlusion thrombotic or embolic, thrombotic venous occlusive or nonocclusive disease like vasospasm or due to low cardiac output. Approximately about 50% of all cases involve embolic pathology, about 25% arterial thrombosis, NOMI involves about 20%, and MVT less than 10%.

Severity of injury is inversely related to the blood flow in the mesentery and directly depends on blood pressure, vessels involved, collateral circulation and ischemia duration. SMA and SMV has good collateral circulation, hence its blockade is silent.

The bowel may be affected varying from reversible ischemia to transmural infarction with necrosis and perforation. Gastrointestinal tract bleeding can be seen due to mucosal sloughing.

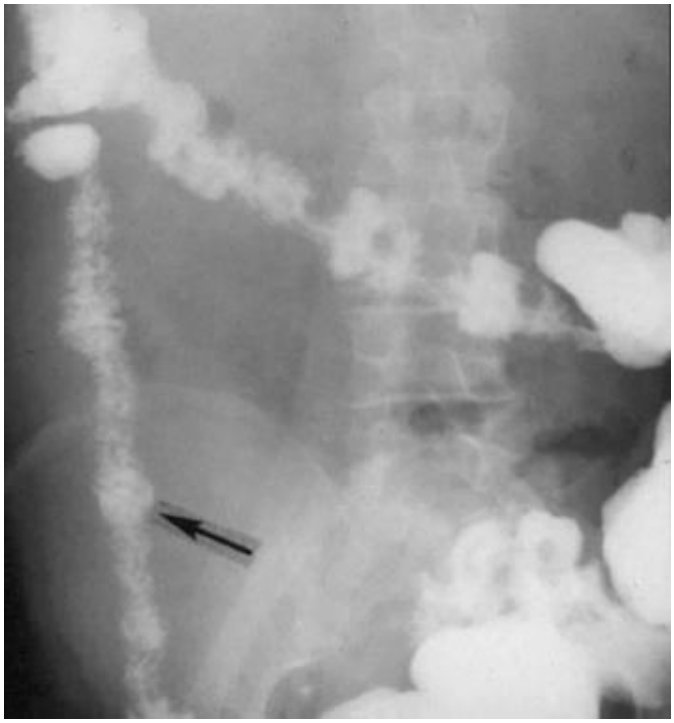


Figure : 3 X ray shows spasm of the bowel

As the ischemia progress the mucosal barrier gets disrupted leading to release of bacteria, toxins and vasoactive substances into the systemic circulation which can lead to death due to septic shock ,multisystem organ failure or cardiac failure or before bowel necrosis actually occurs.[6]

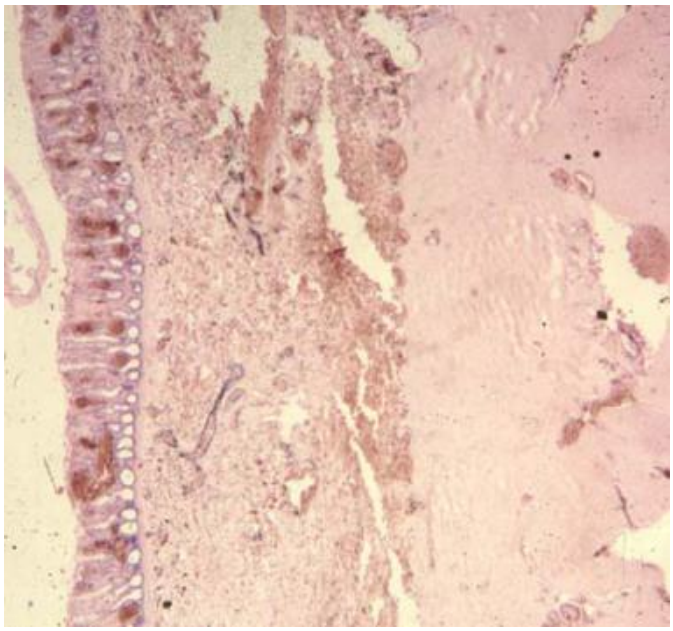


Figure : 4 Pathologic changes after three hours after onset of ischemia of bowel

The bowel mucosa becomes edematous releasing toxic fluid into the peritoneal cavity. This is the fluid that is taken while doing a diagnostic peritoneal lavage.

. About 8-12 hours from the onset of symptoms necrosis of bowel can occur .

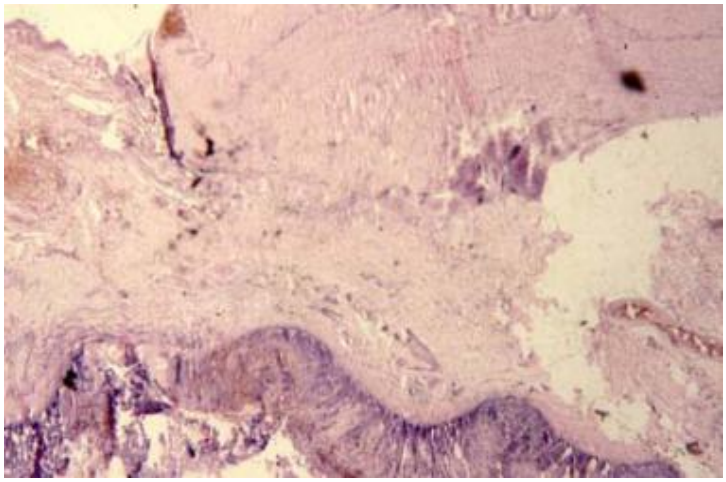


Figure : 5 Post ischemia findings

Acute mesenteric arterial embolism

Cardiac embolus , the usual cause of AMAE which include mural thrombi, atrial thrombi from atrial fibrillation and mitral stenosis and, endocarditis, mycotic aneurysm and thrombi at the site of aortic plaques or at aortic prosthetic graft sites.[9]

As vascular occlusion is acute, in onset, compensatory increase in collateral flow is difficult to develop because of which ischemia is worse than compared to the patients with AMAT. SMA is the visceral vessel most susceptible to embolibecause of its small takeoff angle from the aorta and higher flow. IMA is rarely involved.[1]

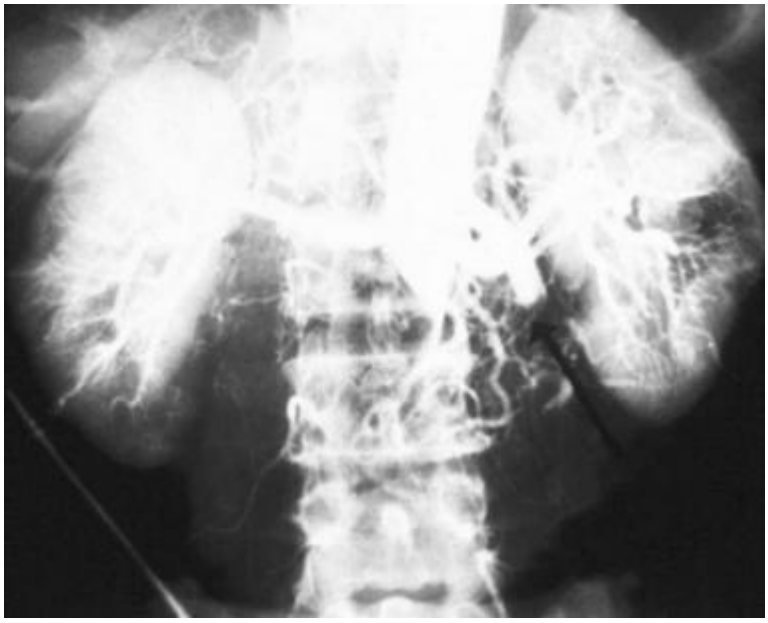


Figure : 6 Complete aortic occlusion with acute embolism of SMA

Acute mesenteric arterial thrombosis

AMAT is a delayed complication of preexisting atherosclerosis. Symptoms are usually seen only after involvement of two of the three arteries. They are either completely blocked or stenosed.

A thrombus is formed in low flowing blood stream which results in acute obstruction of blood flow to the bowel. The mucosa dies first as it is most sensitive to the reduction in blood flow. Patient usually presents with bloody stools. The bowel gradually becomes necrotic eventually bacterial overgrowth occurs resulting in bowel perforation leading to death.[6]

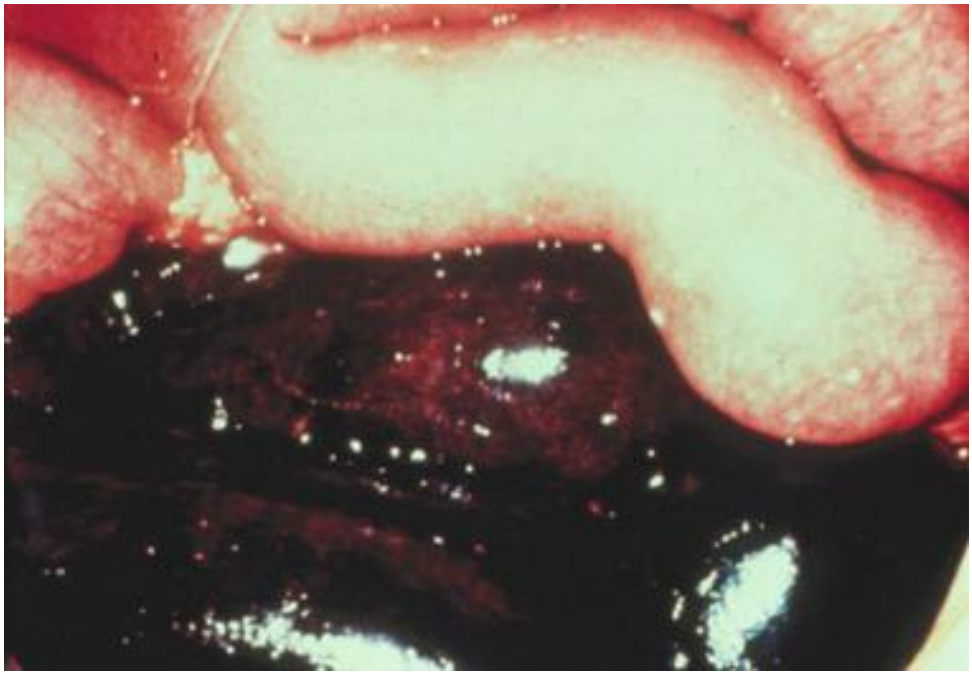


Figure : 7 Specimen of necrosed bowel

AMAT may also be as a result of complication of aneurysm of arteries or due to dissection, trauma, and thromboangitis obliterans. In, smaller diameter vessels are affected. Thrombosis usually occurs at the origin of the SMA.[7]

Mesenteric venous thrombosis

MVT often is the caused due to some processes where there is a clots in the mesenteric veins. MVT can also be seen in patient after splenectomy or ligation of the portal vein or the SMA.

Ischemia in this is due to accumulation of fluid into the bowel wall and lumen which leads to hemoconcentration and hypovolemia. This results in edema of the bowel mucosa and a decrease in the flow of blood due to venous thrombosis which eventually leads to bowel ischemia.[4]

MVT usually seen in young people.

EPIDEMIOLOGY

Age, sex and race related demographics

AMI is often seen in older people in 6 th decade. But can also be seen younger people who have hypercoagulable states.

As such there is no sex preference for AMI. Men are at higher risk for AMI due to higher incidence of atherosclerosis in them. It involves all races

equally. However, people with higher incidence of atherosclerosis may be at risk for developing AMI, such as African American people.[1]

Prognosis

Despite improved survival rates in the recent times, AMI carries a grave prognosis. If bowel wall infarction has occurred mortality is high. Even with good treatment, as many as 40-80% of patients die. For those patients who do survive, the risk of re thrombus is high.

The mortality predictors include older age, hepatic and renal impairment, hypoxia, intramural pneumatosis, acidosis and sepsis. Mortality is highest for AMAT then followed by NOMI, AMAE and MVT.[8]

Early diagnosis and intervention reduces rate of mortality only if the diagnosis is made before signs of peritonitis sets in. It is essential to act early when there is clinical suspicion rather than waiting for full evidence. Survivors of extensive bowel resection have a significant long-term morbidity because of less absorptive surface area. Mortality can be reduced with early treatment.

PATHOPHYSIOLOGY

The earliest microscopic changes are seen in the mucosa within 10 minutes after injury. Histologically inflammatory cell infiltration can be seen. Mucosal edema occurs as a result of increased capillary permeability. Bacterial translocation can occur leading toendotoxemia and exudation of fluid into the bowel lumen. Later the damaged mucosa sloughs offleaving back

ulerations of the bowel wall. Although the bowel mucosa is viable but due to prolonged inadequate bloody supply can ultimately leads tonecrosis, of the

In mesenteric vein thrombosis (MVT) both portal and superior mesenteric venous pressures are raised. The arterial response to mesenteric venous thrombosis may persist well after the venous obstruction has been corrected.[4]

PATHOLOGY GROSS APPEARANCE

Bowel turns to black in colour. Mucosa is denuded exposing the muscle fibres. The fibres are destroyed showing haemorrhagic patches and some time perforation of bowel wall.[5]

MICROSCOPIC APPEARANCE

Plenty of acute inflammatory cell are seen. Mucosal and sub mucosal edema is present. Characteristically thrombi in mucosal and submucosal capillaries are seen. Muscle fibres shows loss of nuclei but muscularispropria is spared.[25]

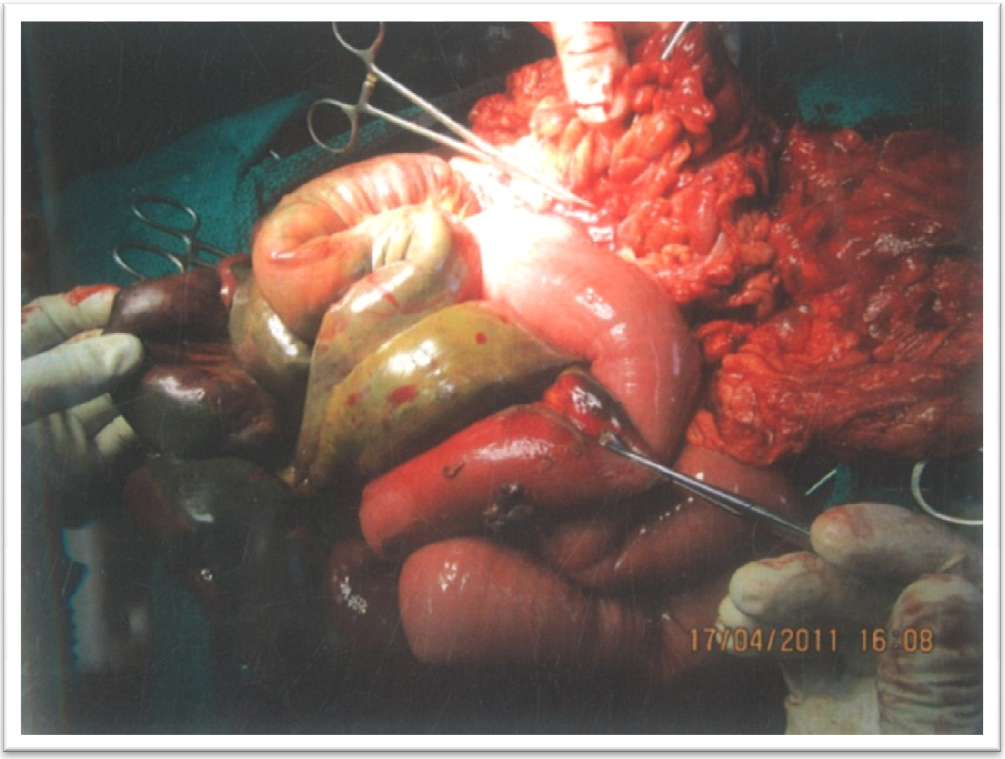


Figure : 8 Intra Operative picture of small bowel gangrene

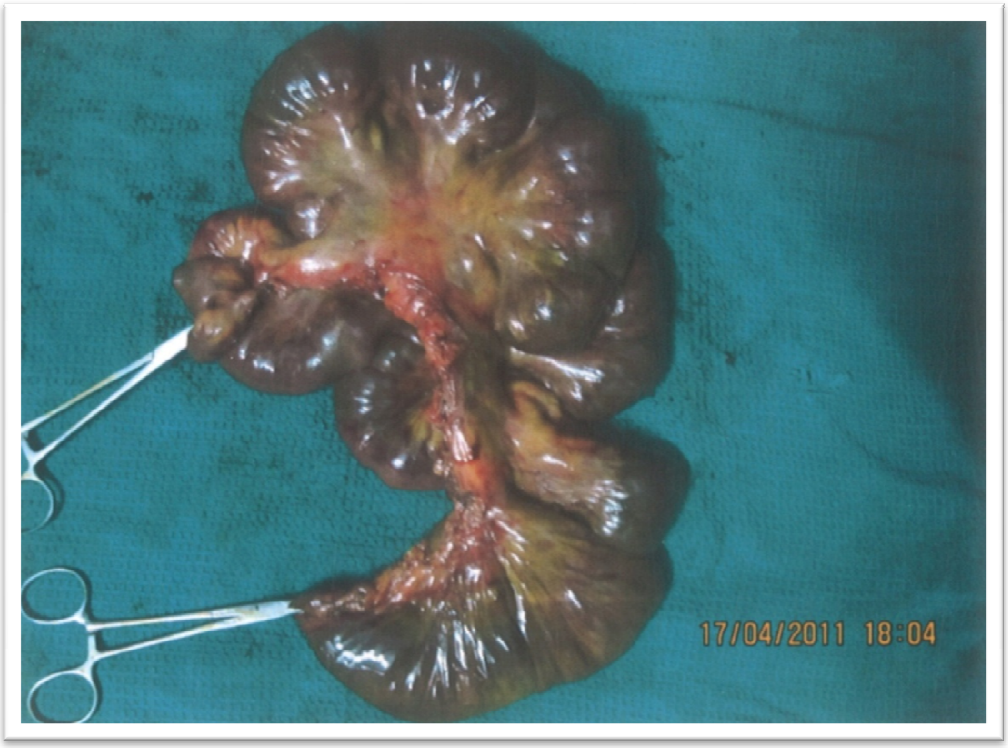


Figure : 9 Resected segment of small bowel gangrene

CLINICAL PRESENTATION

Patients present with severe generalized abdominal pain. Pain is often associated with vomiting. The location of pain may vary initially but once the ischemia progresses to infarction the patient will develop generalized pain. Patients can also present with symptoms of nausea, vomiting and diarrhea in early course of the disease. Progression of intestinal ischemia to transmural bowel infarction leading to peritonitis may be signaled by fever, bloody diarrhea and shock.

Early diagnosis of acute mesenteric ischemia (AMI) requires a high index of suspicion in any patient who are at high risk for embolic or thrombotic events such as those with cardiac disease, peripheral vascular disease, cardiac arrhythmias or recent history of myocardial infarction, examination of the abdomen may reveal relatively normal findings or only slight abdominal distension in the early stages of AMI. As the disease the abdomen becomes grossly distended with absent bowel sounds and peritoneal signs will develop.[5]

EVALUATION OF INTESTINAL BLOOD

FLOW PHYSICAL SIGNS AND SYMPTOMS

It is rapid and cost free but has low specificity and low sensitivity and needs other confirmatory tests.

2. LAB INVESTIGATIONS

Elevated WBC count, amylase level, pH level are seen in bowel ischemia but has low specificity. Venous and peritoneal phosphate level and elevated creatinine phosphokinase are also seen but many prospective studies have shown that it could not find difference between viable and non viable bowel.[8]

3. PLAIN X RAY ERECT ABDOMEN

Gasless abdomen due to small bowel spasm which later lead to distensionand ileus but the disadvantage is that its non specific. Portal venous gas is a late finding not useful in early diagnosis. On barium examination thumb printing sign is seen due to submucosal haemorrhage and superficial mucosalulceration.

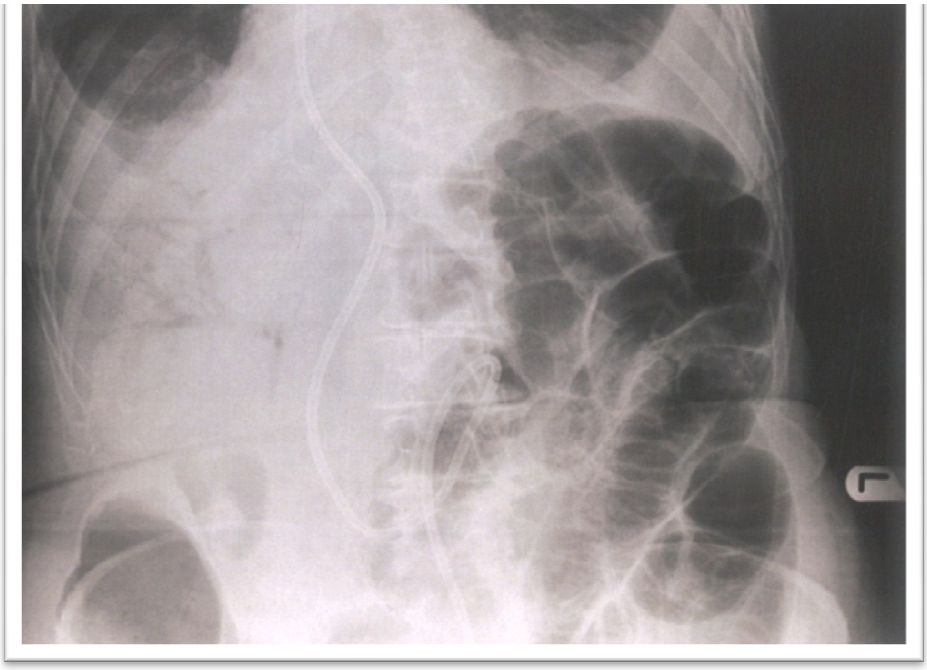


Figure : 10 X ray showing portal venous gas

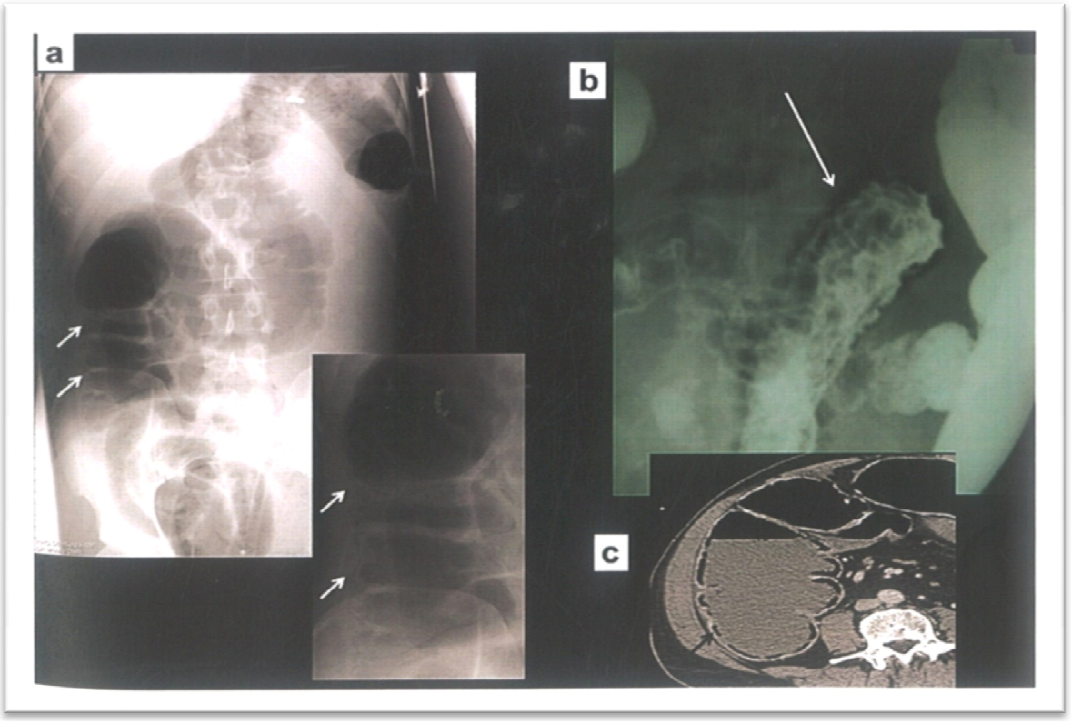


Figure : 11 X ray showing intramural gas shadow

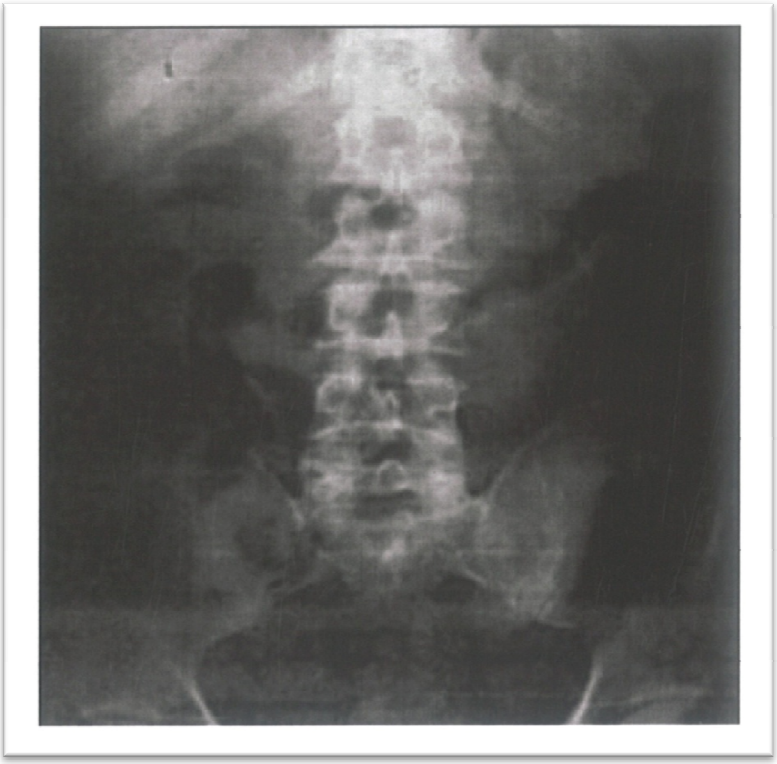
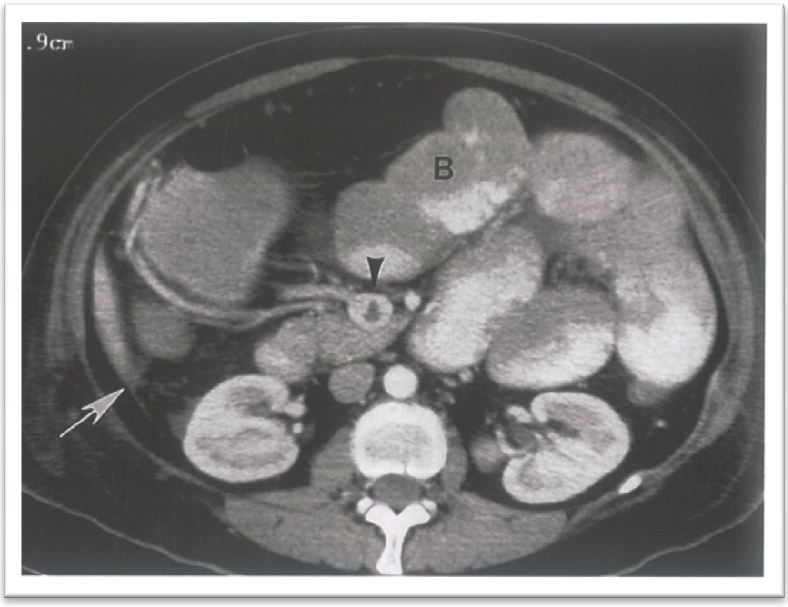


Figure : 12 X ray showing thumb printing sign in transverse colon

4. COMPUTER TOMOGRAPHY OF ABDOMEN:

CT has become the investigation of choice for diagnosis of acute mesenteric vein thrombosis (MVT) and has sensitivity more than 90%. Enlarged SMV or portal vein with low attenuation in the central area is indicative of thrombus. Contrast CT, shows rim enhancement showing bulls eye appearence. Presence of ascites and bowel wall thickening are suggestive of the MVT. It is not helpful to differentiate between acute and chronic occlusion.[13]

Figure : 13 CT – abdomen shows block in superior mesentery artery



5. ARTERIOGRAPHY

Acute thrombosis involve origin of any or all the three vessels i.e. superior mesenteric, inferior mesenteric and celiac artery. Emboli affects

mainly superior mesenteric artery often at the orifice of middle colic artery and visualized as an inverted meniscus sign.

In non occlusive ischemic disease segmental mesenteric arterial constriction with associated proximal stenosis of superior mesenteric artery. Anteriolateral and anterioposterior view is essential. The information obtained helps to select appropriate procedure for the patient. Characteristic string of sausages or string of lake sign is seen.[1]

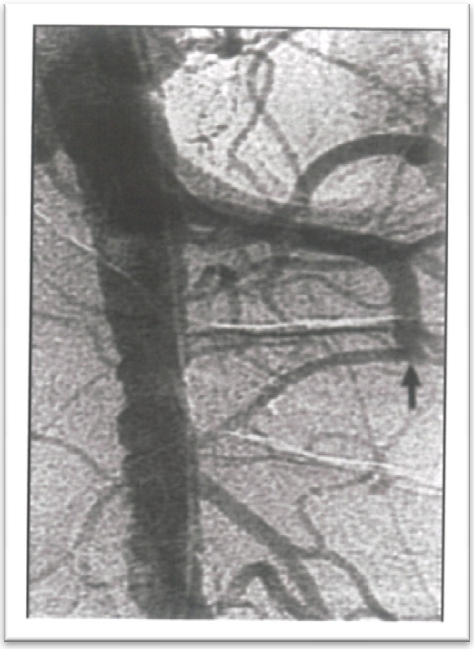


Figure 14 Arteriogram showing Superior Mesentery Artery Occlusion

6. RADIOLABELLED ISOTOPE STUDIES

Intravenous **Te^{99m} labelled pyrophosphate** binds to extravasated extracellular calcium from ischemic cells and **Te^{99m}sulphur colloid labelled**

leucocyte migrate to area of inflammation. Major disadvantage is that they need advanced bowel ischemia i.e necrosis to obtain hot spot in isotope imaging.

Intra peritoneal injection of Xe^{133}: Injected Xe^{133} quickly and equally absorbed by passive transperitoneal diffusion into ischemia and normal bowel . It is promptly cleared by normally perfused bowel and the poorly perfused bowel retainXe^{133} which is detected by gamma cameras. The advantages are its safety, rapidity of results, ability to detect early ischemia, lack of interruption by adhesions and Moderate ascites.[1]

7. DOPPLER STUDY

Pre operative: It is valuable in evaluation of chronic mesenteric arterial occlusive disease. Even with flow in the proximal SMA or celiac artery an embolic cause cannot be ruled out. Absent flow in the portal and mesenteric venous system and the presence of ascites are highly suggestive of Mesenteric vein thrombosis. The visceral aorta can be visualized by transgastric ultrasonography.

Intra operative: pencil like hand held Doppler ultrasound probe applied to the anti mesenteric border of bowel wall and discrete mesenteric vessels. Its shows typical pulsatile sound indicating adequate blood flow. Drawback is lack of sensitivity for small patchy areas of inadequate circulation and no use in venous occlusion. Advantage is its quickness and ease of application. Sensitivity is 82% and specificity is 91%.[7]

8. FLOURESCEN DYE

Sodium flourescen dye emits gold-green flourescen when exposed to UV light of wave length 3600°A to 4000°A. It is injected intravenously over 30-60 seconds. The dye enters into the viable tissue within minutes of injection. After dimming the operation room lights, woods lamp used to illuminate the field. The viable bowel show confluent fluorescence and non viable bowel show absence of florescence and Perivascular fluorescent pattern. Its rapid transudation into peritoneal cavity and its prolonged presence after injection prevent it from using frequently atleast within 48 hours. Over all sensitivity and specificity is 100% [37]

9. MAGNETIC RESONANT ANGIOGRAM

MRA are excellent non invasive screening techniques for patients suspected of having mesenteric ischemia of all causes. MRA has higher spatial resolution, allowing assessment of the peripheral visceral branches and the inferior mesenteric artery with greater accuracy. In addition, it allows the identification of calcified plaques. The lack of radiation and iodinated contrast agents make it the technique of choice for children and patients with azotemia.[33]

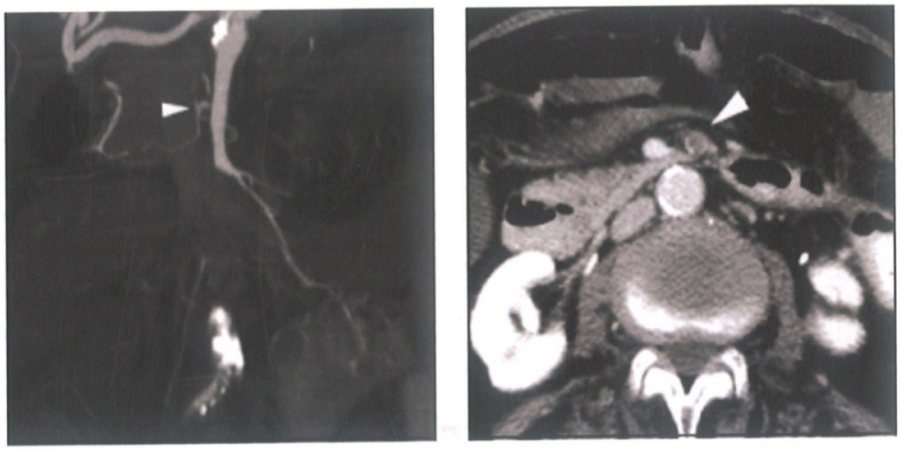


Figure : 15 MR angiogram showing superior mesentery artery block

10. MAGNETIC RESONANT OXIMETRY

MR oximetry is capable of detecting oxygen desaturation caused by segmental ischemia. A loss of oxygen saturation in the SMV relative to that in the inferior vena eava provides a convenient marker of mesenteric ischemia.

MANAGEMENT

Fluid management is very important in these patients and can be monitored by foleys catheter and also a central venous catheter in the patient with significant cardiac disease. If significant hemodynamic instability is present then an arterial line for systemic blood pressure monitoring is adviced. Dopamine is a drug of choice in inotropic agent for patients with AMI because at its low doses it causes mesenteric vasodilator and vasoconstriction rarely seen at higher dose. A nasogastric tube should be inserted to decompress the bowel. Early institution of broad-spectrum antibiotics including for anaerobics compulsory as to prevent bacteremia due to bacterial

translocation from weakened bowel barrier. Postoperatively anti coagulants to be administered to reduce the risk of reccurance.[37]

INTERVENTIONAL RADIOLOGY

Interventional radiologic techniques recently used are catheter-directed thrombolysis, percutaneous transluminal angioplasty (PTA) and fenestration of aortic dissection.

SURGERY

Operative intervention remains the treatment of choice. The surgeon's aim is to confirm of mesenteric ischemia and assess the viability of the intestine and to determine the cause, to revascularise if possible and resection of nonviable bowel. Proximal vessel pulsation which weakens distally, is suggestive of an embolus. Absent proximal pulsation is most probably due to arterial thrombosis.

REVASCULARISATION EMBOLUS

An SMA arteriotomy is done distal to the middle colic artery origin. A transverse arteriotomy is preferred if diagnosis of embolus is certain and longitudinal incision should be made so that it can serve as the site for the distal anastomosis of a bypass graft.

THROMBOSIS

Thromboendarterectomy procedure is done through suprarenal aortic and proximal mesenteric arterial. In acute SMA thrombosis the distal anastomosis can be done at the level of the middle colic artery. In case of celiac arterty occlusion, anastamosis can be performed just distal to its origin from aorta. When MVT is confirmed at exploration and if there is a clot in the superior mesenteric vein although the primary treatment is anticoagulation, thrombectomy should be attempted.

RESECTION

Once revascularization is done, should observe for some 30 to 45 minutes to assess the viability of intestine and decided the need for resection. Clinical signs such as absent peristalsis, mucosal edema, mucosal haemorrhage, bowel discolouration, and unhealthy cut edges of bowel are better markers and may lead to resection .The viability of the bowelcan also be assessed by a continuous-wave Doppler ultrasound probe or sodium fluorescein dye. The part of the bowel that isnonviable must be resected. Primary anastomoses can be done if patient is stable and with viable bowel.[33]

Atypical symptoms, presence of predisposing diseases, delayed surgical intervention due to diagnostic difficulties and elderly patients who have cardiac problems are some of the factors for high mortality rates. Intestinal blood flow is impaired as a result of mesenteric vascular

insufficiency which can be due to underlying causes such as atherosclerosis, mesenteric artery embolism, generalized vasospasm and mesenteric vein thrombosis. Duration of ischemia, grade of mesentery artery occlusion and proportion of collateral flow are the factors determining the intestinal damage after acute arterial occlusion. Leukocytosis is common in AMI but it is a nonspecific marker for inflammation and infection. About 50% of patients have metabolic acidosis a late finding which shows intestinal infarct and 25% of them have hyperamylasemia. Prerenal azotemia, lactate level, increased levels of phosphate and alkaline phosphatase may also be present. The role of radiologic imaging in AMI diagnosis is limited. Findings in direct abdominal radiographs are nonspecific. Abdominal ultrasonography and mesenteric Doppler ultrasonography are the preferred imaging modalities in the diagnosis. Computed tomography (CT) angiography has high sensitivity and specificity. Although mesenteric vessels can be visualized with magnetic resonance (MRI) angiography but its evaluation of the mesenteric arteries is primarily limited to the proximal celiac and superior mesenteric artery (SMA) only. Angiography must be performed early when mesenteric artery occlusion is suspected. When there is suspicion of AMI and emergency angiography cannot be done then emergency laparotomy must be performed. In patients with findings of peritonitis and suspicions of AMI need of angiography is controversial. In centers without sufficient radiologic imaging techniques, diagnostic peritoneal lavage may be helpful to evaluate the intestinal activity. In the past 30 years with the use of diagnostic and conventional angiography

and the progress in intense care units new approaches such as SMA bypass, SMA embolectomy and retrograde open mesenteric stent have been used. Especially when there are signs of peritonitis an emergency surgical intervention is adviced in patients with mesenteric arterial embolism. To reestablish sufficient blood flow to the intestine and to reduce the extent of ischemia-reperfusion damage and the risk for definitive bowel infarction, embolectomy should be applied immediately and whenever it is possible. If there is not sufficient pulse after embolectomy, a translocation of the SMA onto the infrarenal aorta can be performed or a bypass between the aorta or iliac vessels and the mesenteric artery can be done as an alternative.[33]

MATERIALS AND METHODS

Source of Data:

- 25 patients admitted in Coimbatore Medical College and Hospital with Acute Mesenteric Vascular Occlusion.

Study Place:

- Coimbatore Medical College and Hospital

Study Design:

- Prospective Observational Study

Sample Size:

- 25 Patients

Study Period:

- September 2014 – September 2015.

Inclusive Criteria:

- Patients admitted with suspected Mesenteric Vascular Occlusion.
- Age > 18 yrs

Exclusion Criteria

- ❖ Patients admitted with bowel gangrene due to other causes.

25 patients admitted in general surgery department in Coimbatore Medical College with a probable diagnosis of Acute Mesenteric Vascular Occlusion will be evaluated as follows. Demographic features (age, gender, time elapsed to laparotomy), serum values of leukocytes, amylase, alkaline phosphatase, and urea, liver enzyme levels, radiologic imaging techniques, surgical techniques, complications, mortality, and hospitalization period were evaluated. Acute Mesenteric Vascular Occlusion was diagnosed on clinical examination supported with laboratory and imaging techniques. Elapsed time between the onset of symptoms and the surgery is defined as 24 hours and more than 24 hours. All Patients underwent emergent laparotomy. This was determined by prediagnositic contributory techniques. Patients were allocated to three groups according to the place of the necrosis. Resection and Anastamosis was done according to bowel viability. Comparing the factors effecting mortality between groups, the data on the group with total necrosis were disregarded because all patients died.

Followed up post operatively and observed for any complications.

Risk factors associated with various complications and mortality will be analysed and studied.

AGE

Age	Frequency	Percent
28	1	4.0
30	1	4.0
35	1	4.0
40	4	16.0
41	1	4.0
47	2	8.0
49	1	4.0
50	4	16.0
53	1	4.0
55	2	8.0
57	1	4.0
60	3	12.0
62	1	4.0
65	1	4.0
74	1	4.0
Total	**25**	**100.0**

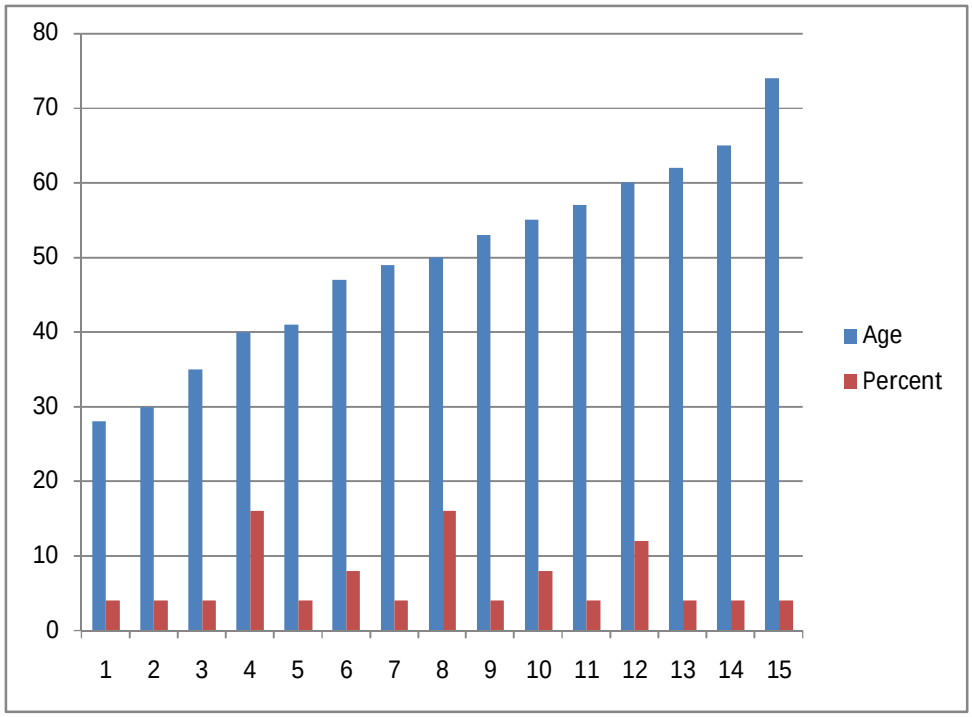


Chart 1 : Age

SEX

Sex	Frequency	Percent
MALE	22	88.0
FEMALE	3	12.0
Total	**25**	**100.0**

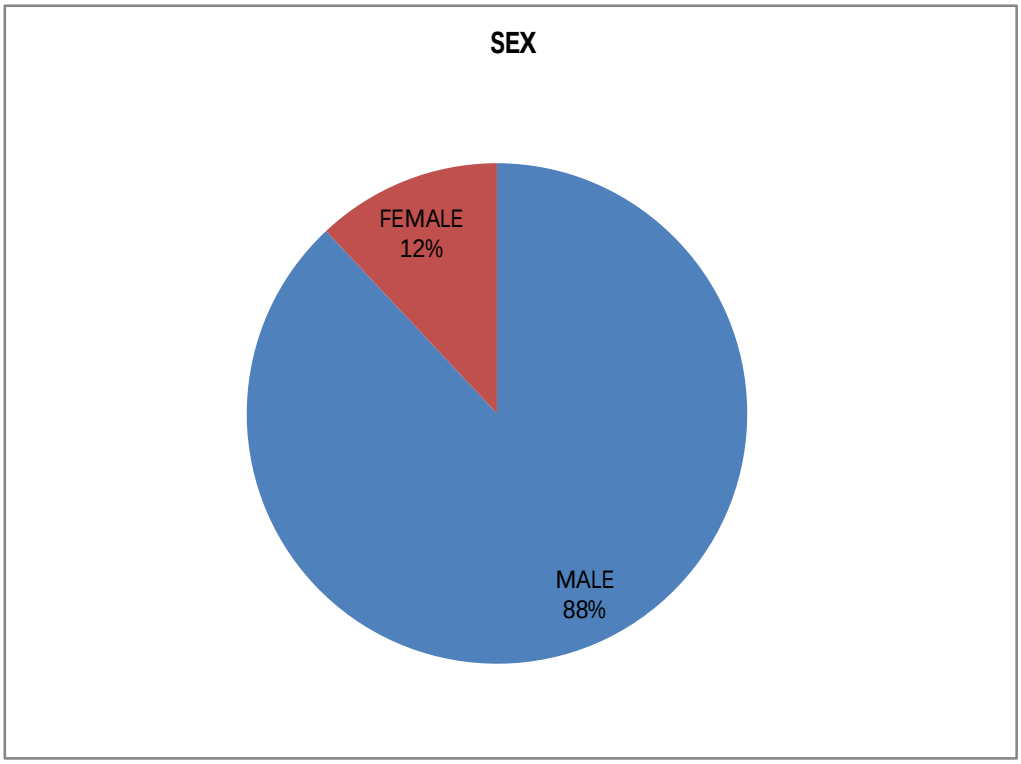


Chart : 2 Sex

ABDOMEN PAIN

	Frequency	Percent
Positive	19	76.0
Negative	6	24.0
Total	**25**	**100.0**

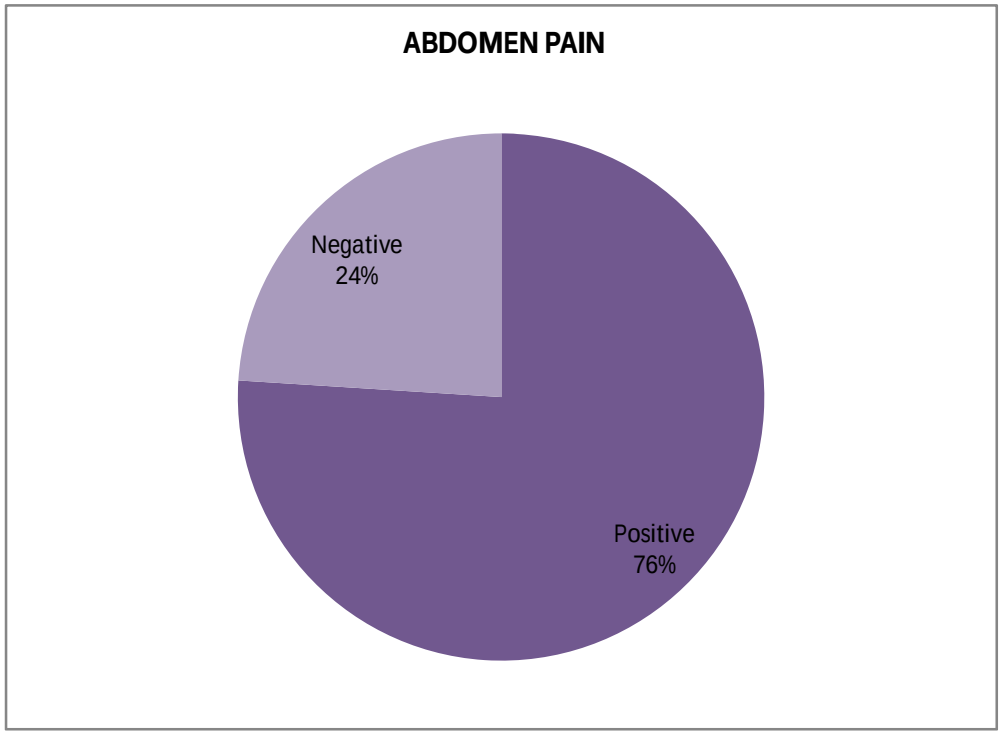


Chart : 3 Abdomen Pain

HISTORY (Duration of symptoms in hours)

	Frequency	Percent
<24	5	20.0
>48	17	68.0
<48	3	12.0
Total	**25**	**100.0**

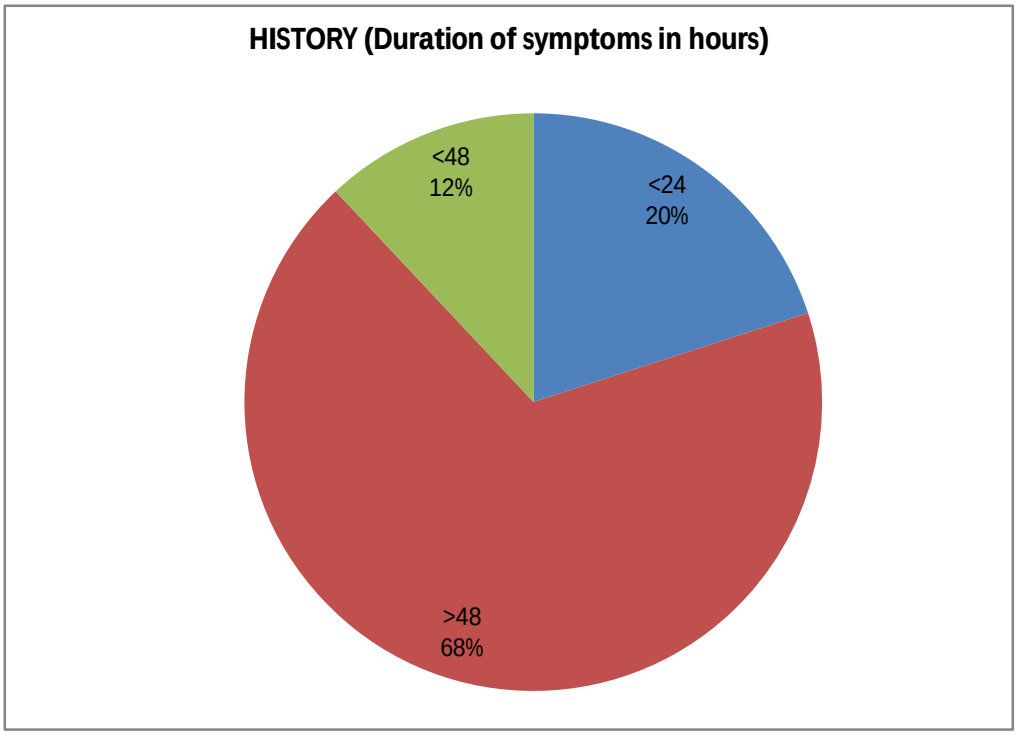


Chart : 4 History (Duration of symptoms in hours)

DIABETES

	Frequency	Percent
Positive	12	32.0
Negative	13	68.0
Total	**25**	**100.0**

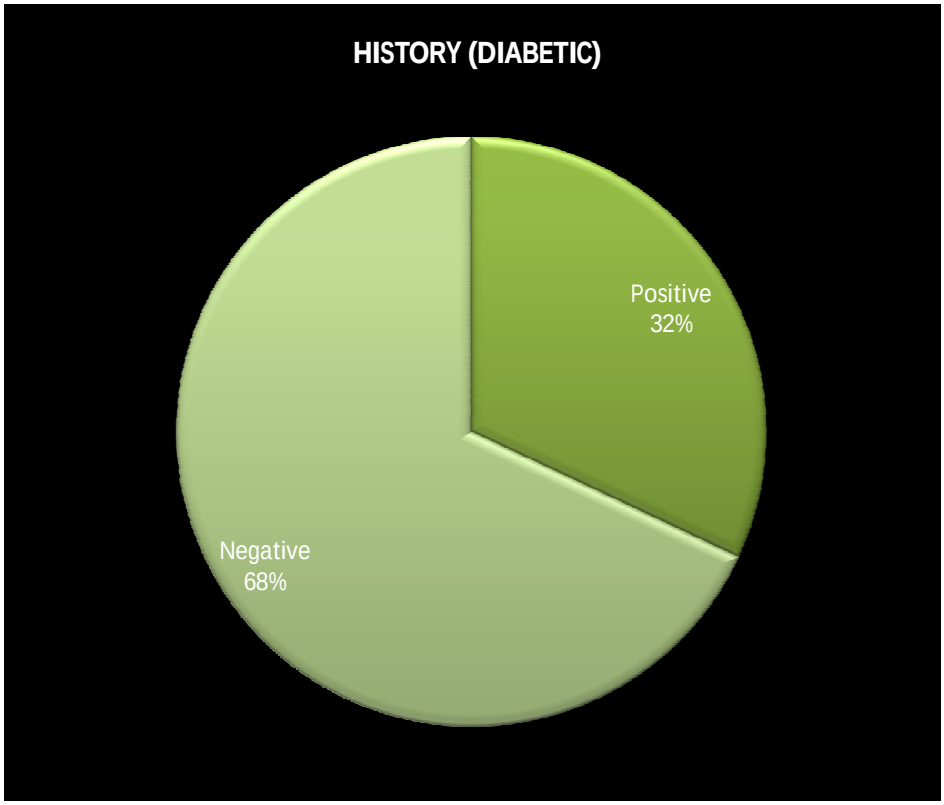


Chart : 5 Diabetes

SYSTEMIC HYPERTENSION

	Frequency	Percent
Positive	13	32.0
Negative	12	68.0
Total	**25**	**100.0**

SHT

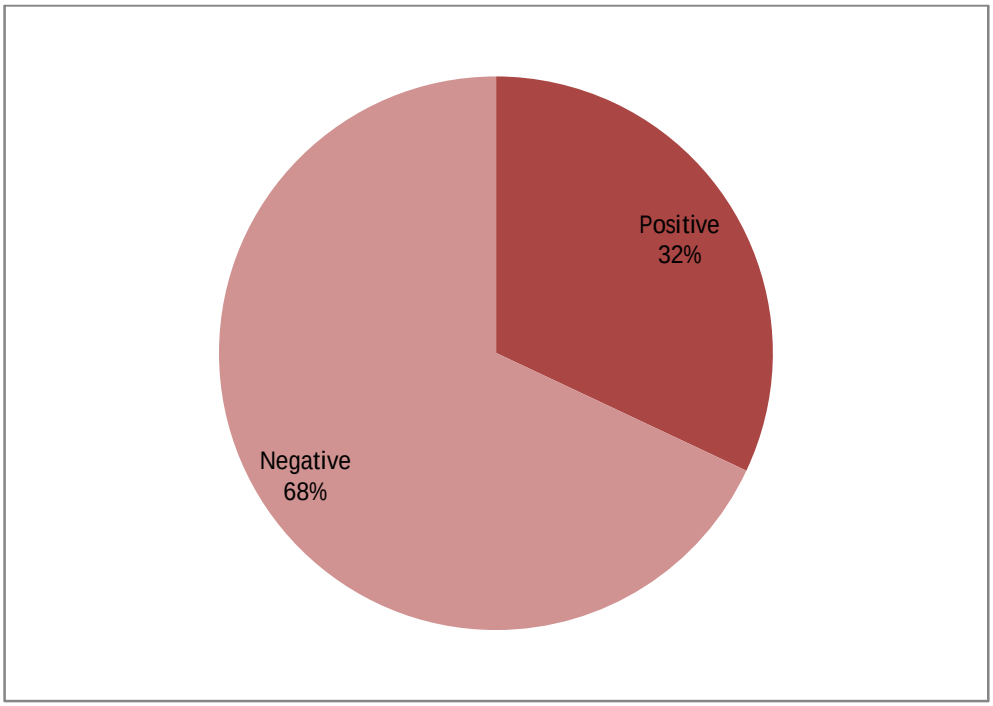


Chart : 6 SHT

SMOKING

	Frequency	Percent
Positive	20	80.0
Negative	5	20.0
Total	**25**	**100.0**

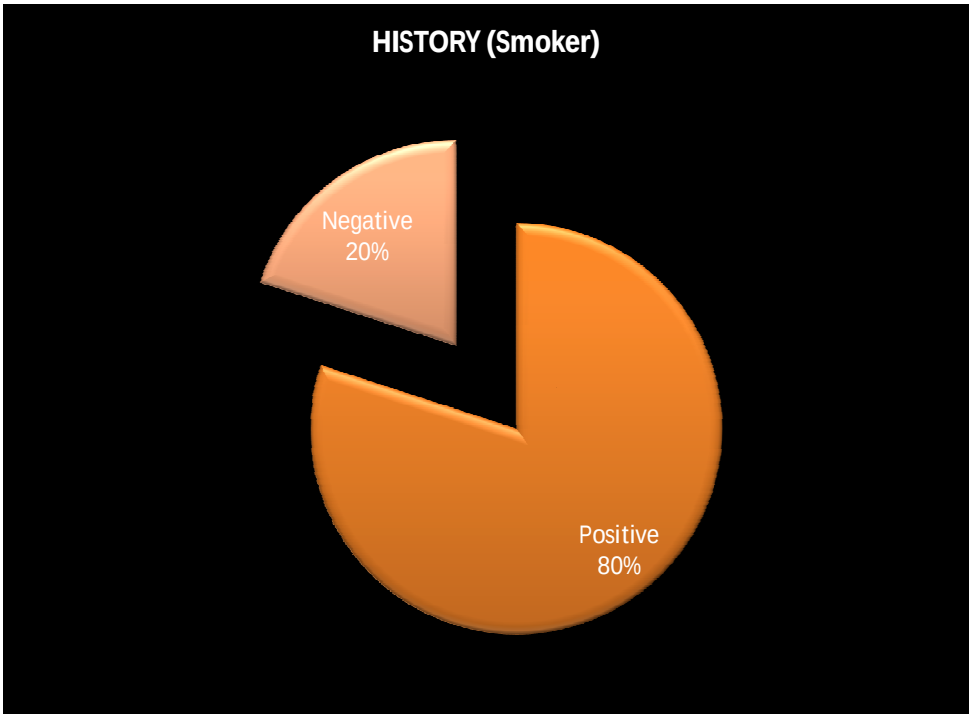


Chart : 7 Smoking

ALCOHOLISM

	Frequency	Percent
Positive	18	72.0
Negative	7	28.0
Total	**25**	**100.0**

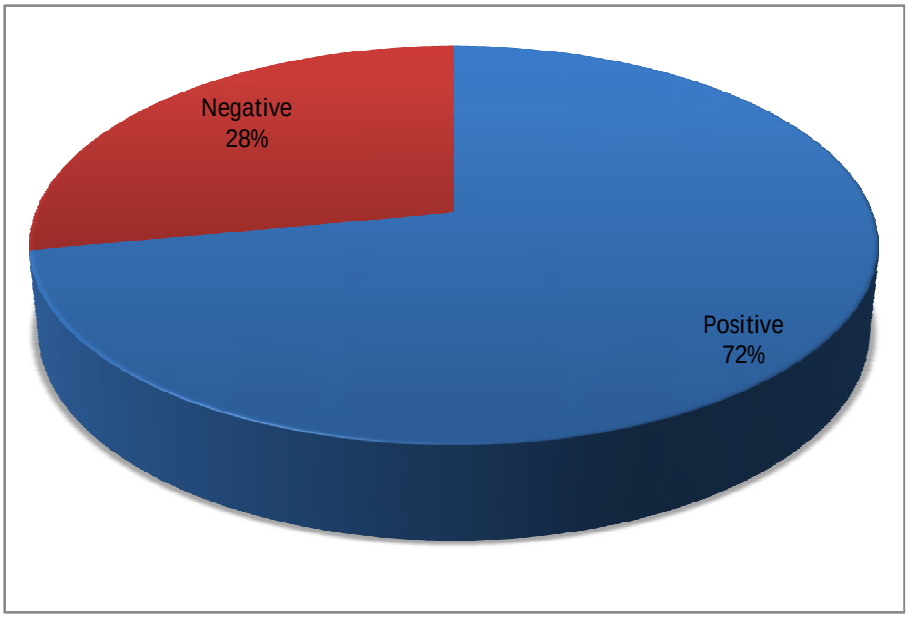


Chart : 8 Alcoholism

THROMBO EMBOLIC EPISODES

	Frequency	Percent
Positive	4	16.0
Negative	21	84.0
Total	**25**	**100.0**

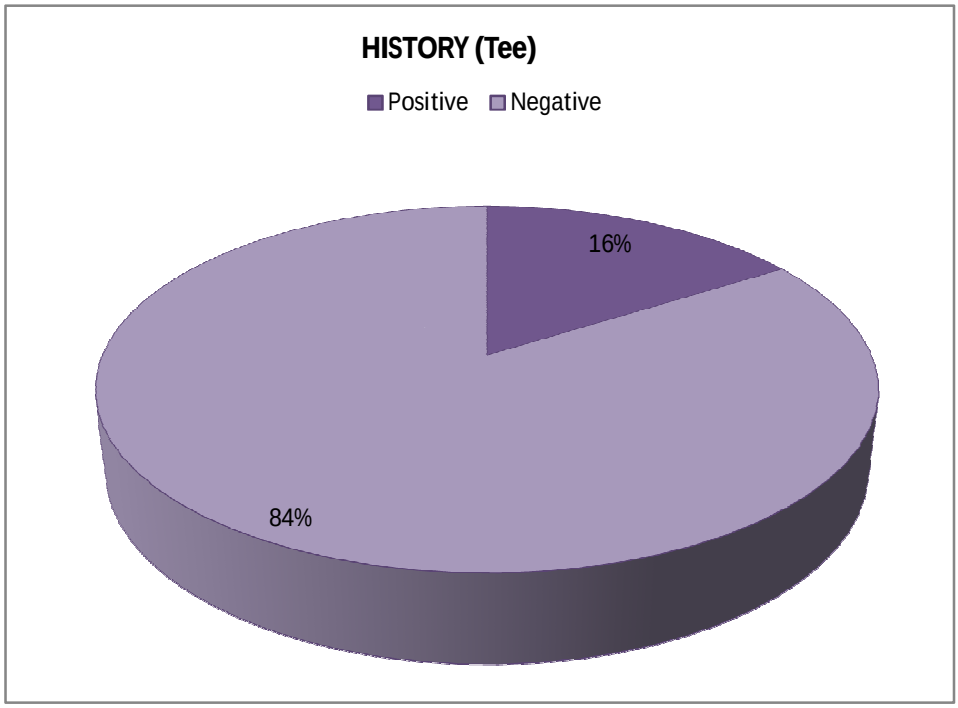


Chart : 9 Thrombo Embolic Episodes

VOMITING

	Frequency	Percent
Positive	17	68.0
Negative	8	32.0
Total	**25**	**100.0**

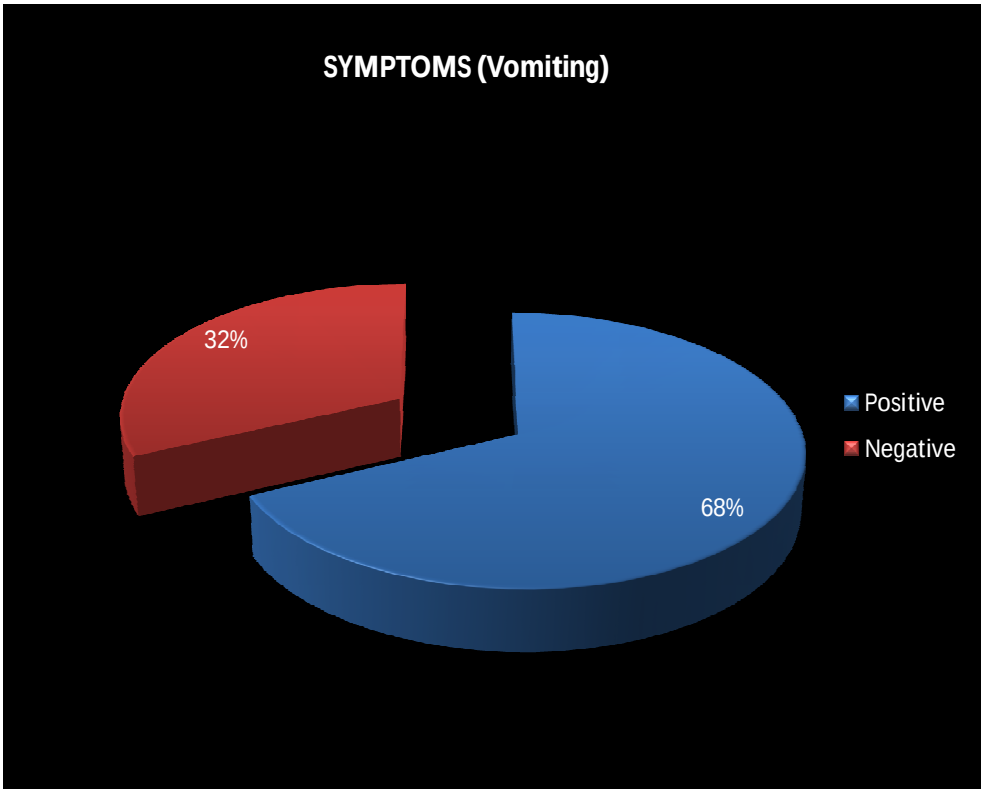


Chart : 10 Vomiting

DIARRHOEA

	Frequency	Percent
Positive	5	20.0
Negative	20	80.0
Total	**25**	**100.0**

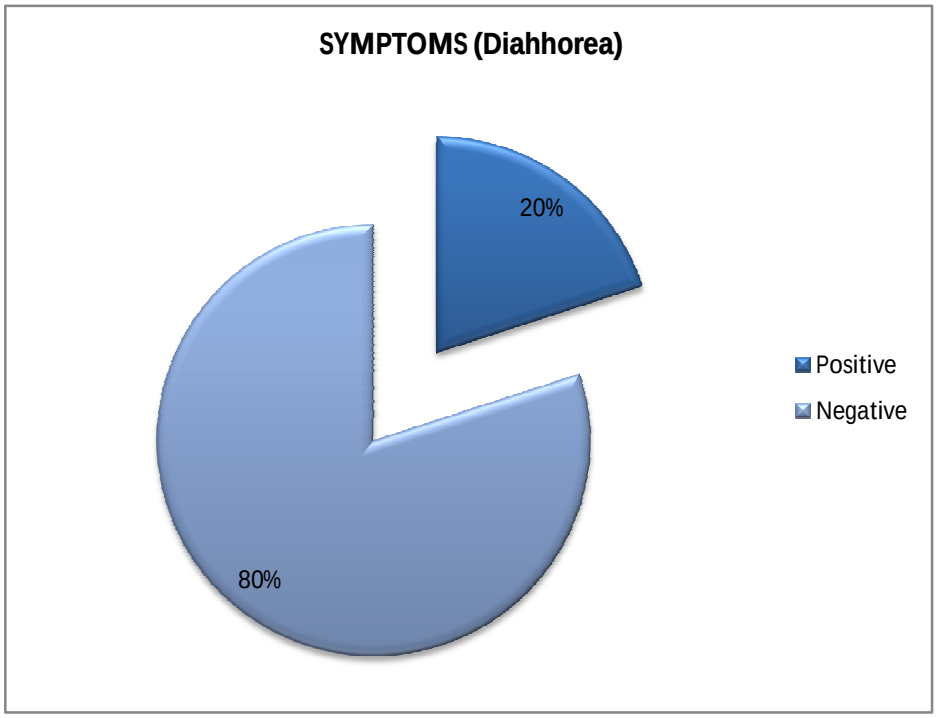


Chart : 11 Diarrhoea

BLOOD IN STOOLS

	Frequency	Percent
Positive	4	16.0
Negative	21	84.0
Total	**25**	**100.0**

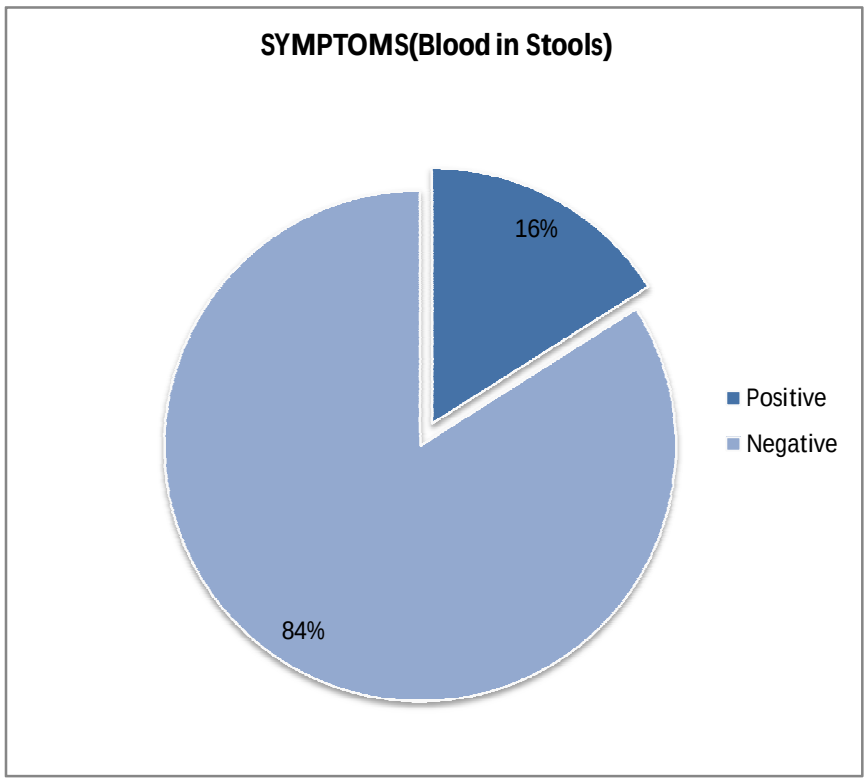


Chart : 12 Blood in Stools

OBSTIPATION

	Frequency	Percent
Positive	19	76.0
Negative	6	24.0
Total	**25**	**100.0**

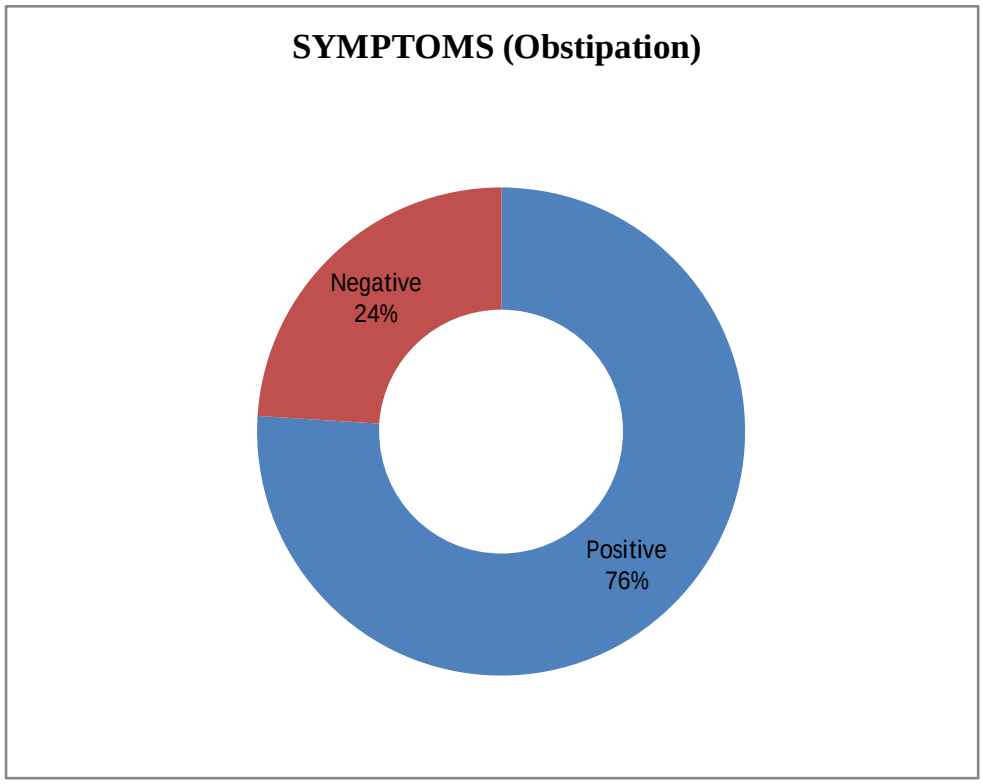


Chart : 13 Obstipation

SYMPTOMS (Pyrexia)

	Frequency	Percent
Positive	6	24.0
Negative	19	76.0
Total	**25**	**100.0**

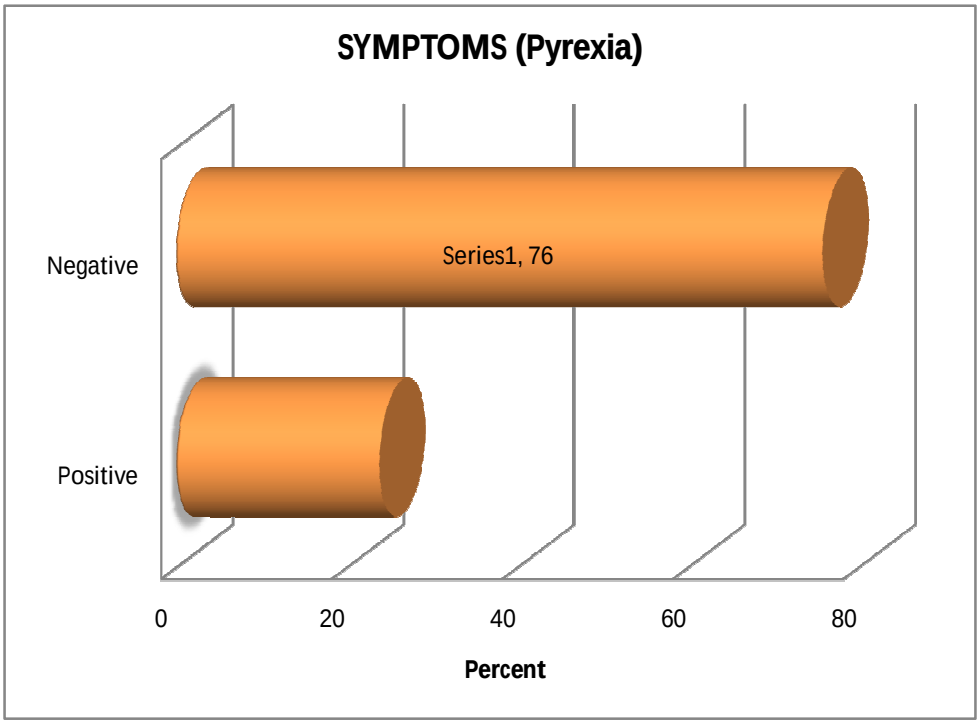


Chart : 14 Symptoms (Pyrexia)

ABDOMINAL DISTENSION

	Frequency	Percent
Positive	17	68.0
Negative	8	32.0
Total	**25**	**100.0**

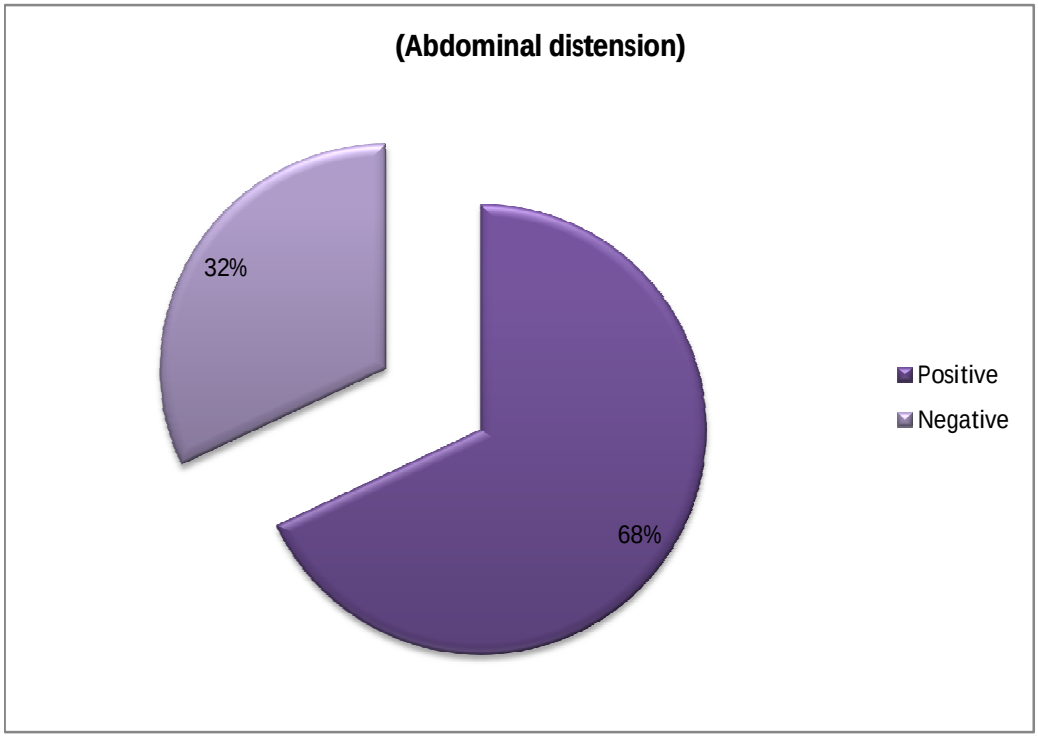


Chart : 15 Abdominal Distension

GUARDING/RIGIDITY

	Frequency	Percent
Positive	18	72.0
Negative	7	28.0
Total	**25**	**100.0**

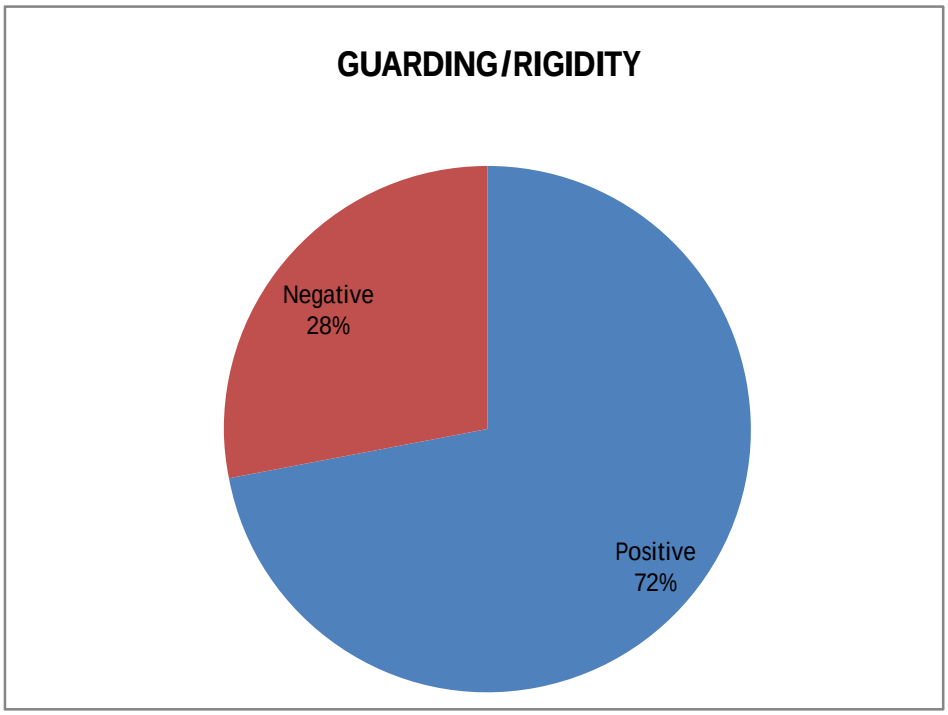


Chart : 16 Guarding/Rigidity

TENDERNESS

	Frequency	Percent
Positive	24	96.0
Negative	1	4.0
Total	25	100.0

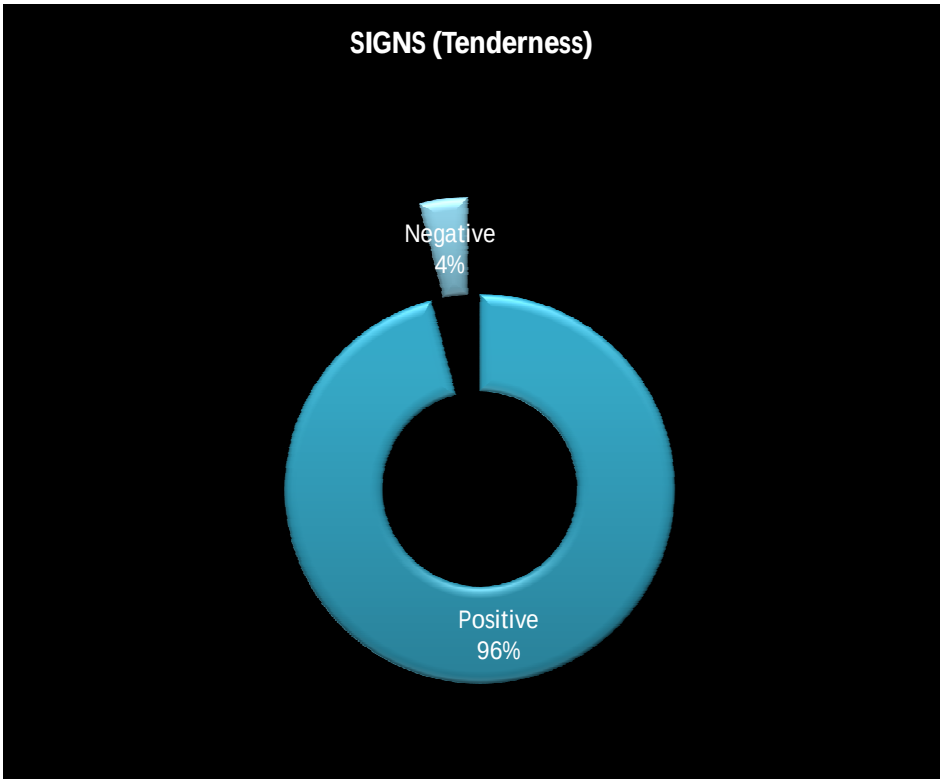


Chart : 17 Tenderness

SIGNS (Bowel sounds)

	Frequency	Percent
Positive	1	4.0
Negative	24	96.0
Total	25	100.0

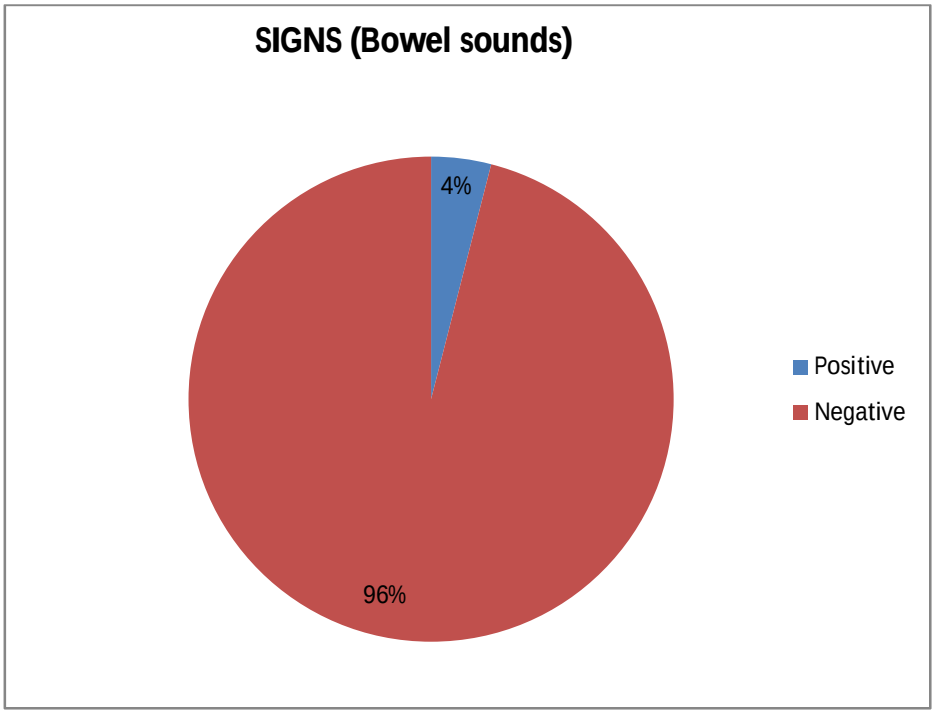


Chart : 18 Signs (Bowel sounds)

SIGNS (Per Rectal)

	Frequency	Percent
N	13	52.0
RECTUM ROOMY	9	36.0
MALENA	3	12.0
Total	**25**	**100.0**

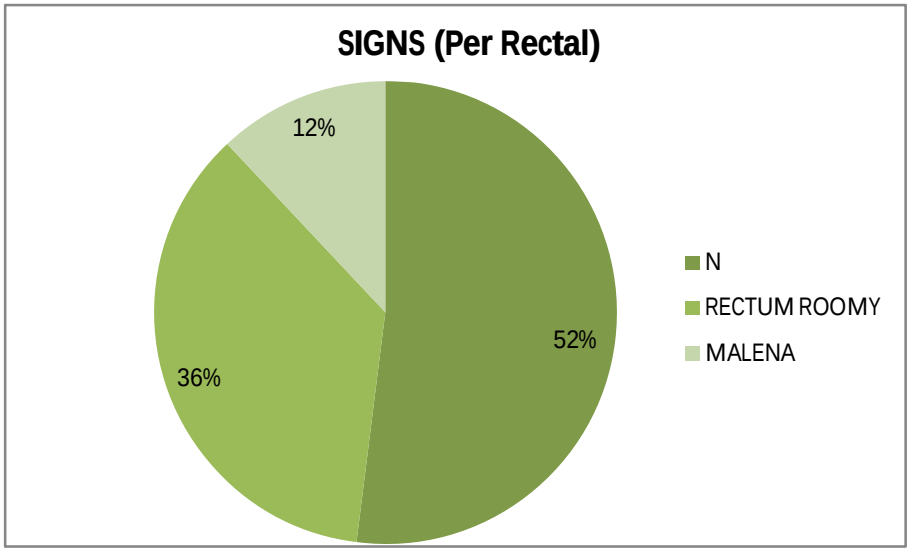


Chart : 19 Signs (Per Rectal)

SHOCK

	Frequency	Percent
Positive	19	76.0
Negative	6	24.0
Total	25	100.0

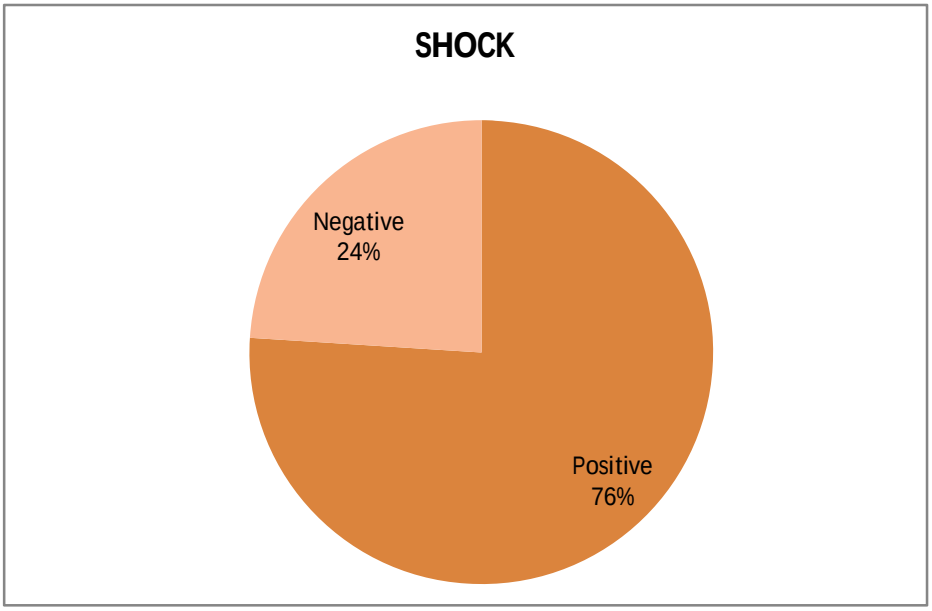


Chart : 20 Shock

INVESTIGATION (Leucocytosis)

	Frequency	Percent
Positive	25	100.0

INVESTIGATION

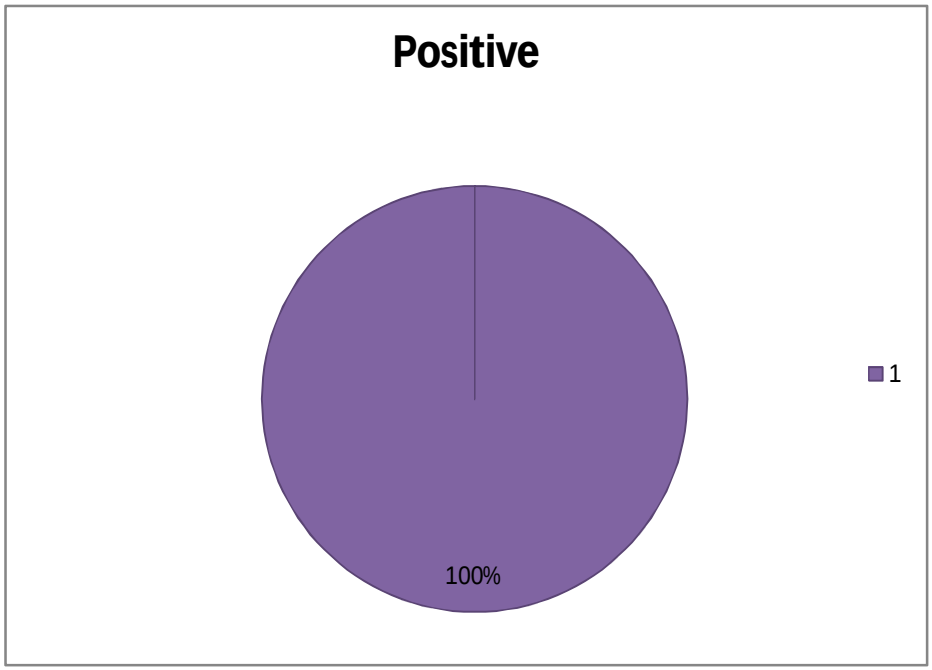


Chart : 21 Investigation (Leucocytosis)

INVESTIGATION (Urea / Creatinine)

	Frequency	Percent
Positive	19	76.0
Negative	6	24.0
Total	25	100.0

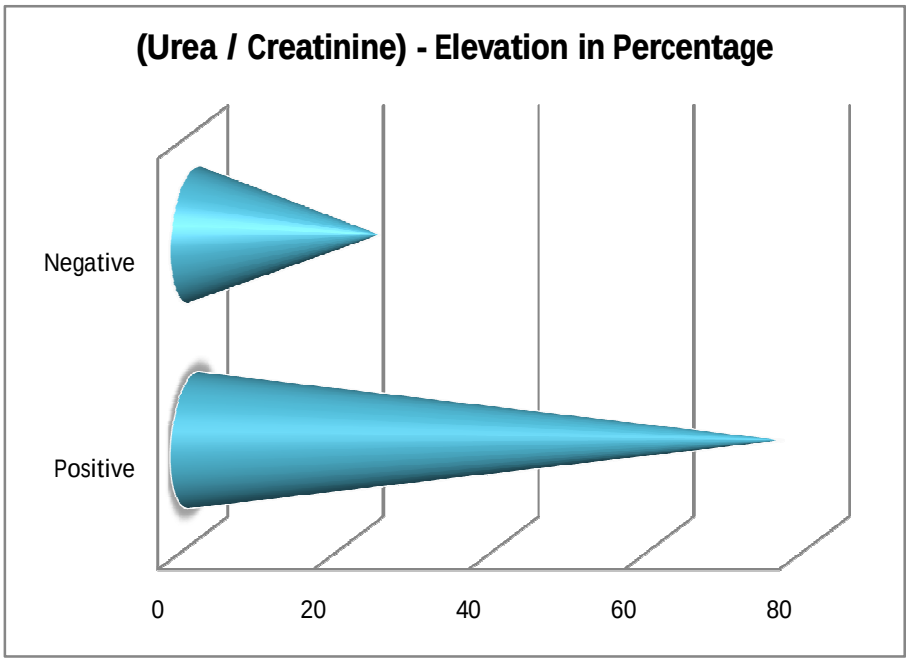


Chart : 22 Investigation (Urea / Creatinine)

INVESTIGATION (X Ray ABD)

	Frequency	Percent
NS	3	12.0
DBL	5	20.0
DBL + AFL	16	64.0
DBL + AVD	1	4.0
Total	**25**	**100.0**

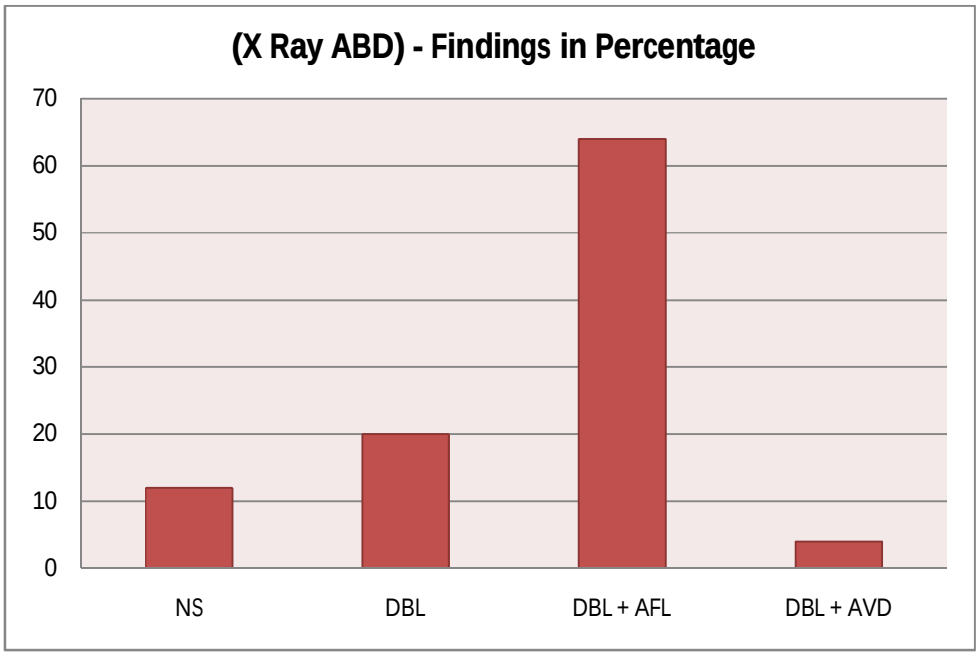


Chart : 23 X –Ray Findings

INVESTIGATION (Four Quadrant Aspiration)

	Frequency	Percent
Positive	4	16.0
Negative	21	84.0
Total	**25**	**100.0**

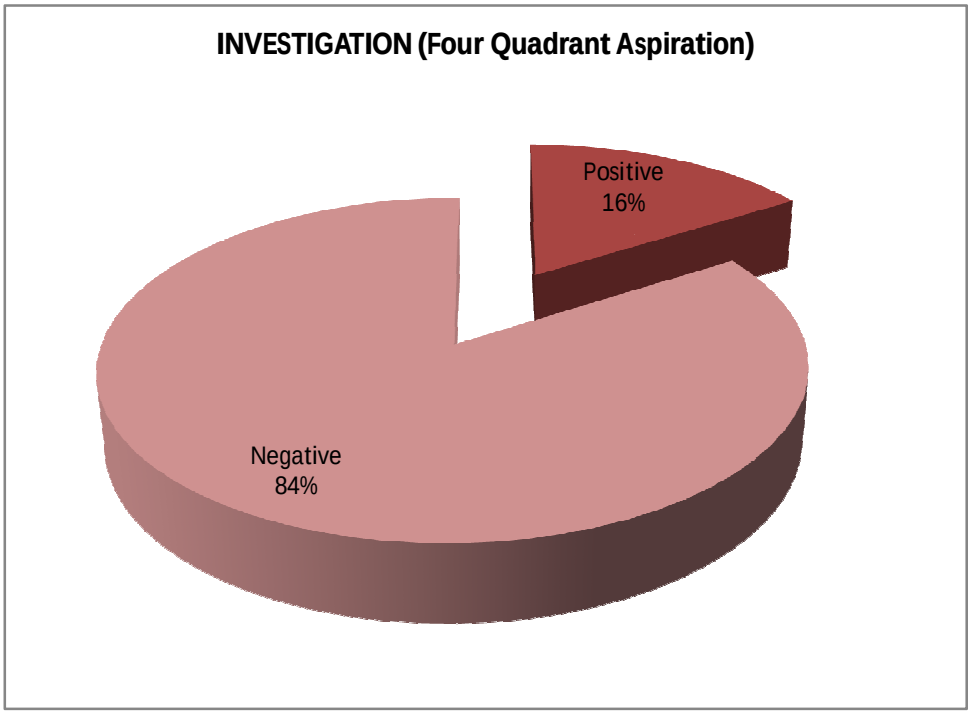


Chart : 24 Investigation (Four Quadrant Aspiration)

INVESTIGATION (USG ABD)

	Frequency	Percent
Positive findings of obstruction	22	88.0
N	3	12.0
Total	**25**	**100.0**

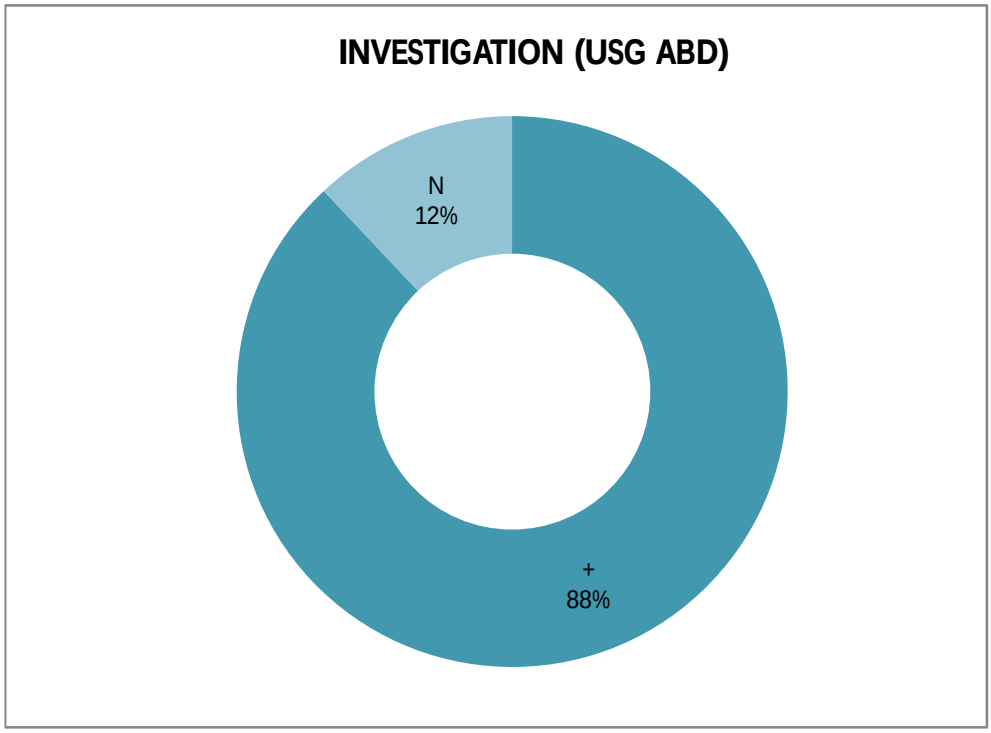


Chart : 25 Investigation (USG ABD)

INVESTIGATION (ECG)

	Frequency	Percent
Abnormal	8	32.0
N	17	68.0
Total	**25**	**100.0**

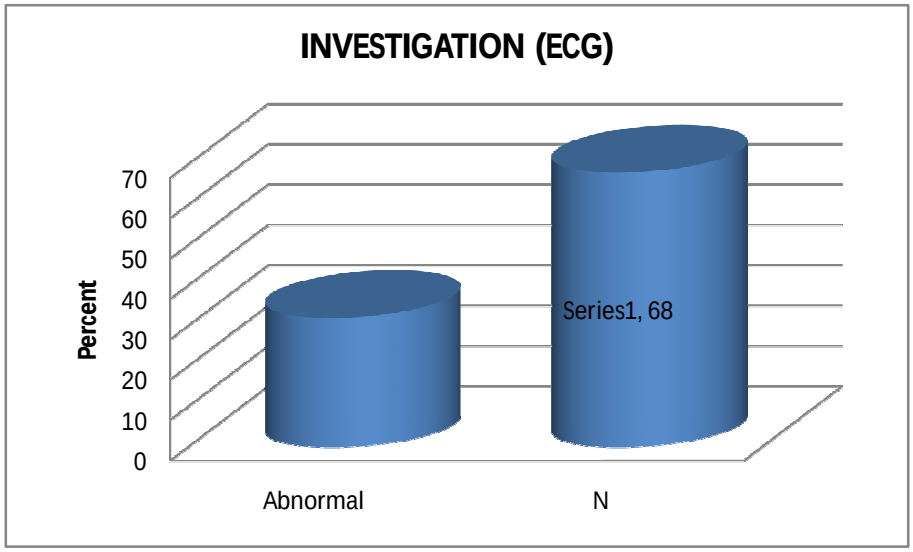


Chart : 26 Investigation (ECG)

GANGRENE SEGMENT

	Frequency	Percent
JEJUNUM	3	12.0
ILEUM + AC	5	20.0
ILEUM + AC + TC	1	4.0
ILEUM + AC + TC + DC	2	8.0
JEJUNUM + ILEUM	7	28.0
JEJUNUM+ILEUM+AC	2	8.0
JEJUNUM + ILEUM + AC +TC + DC + SC	5	20.0
Total	**25**	**100.0**

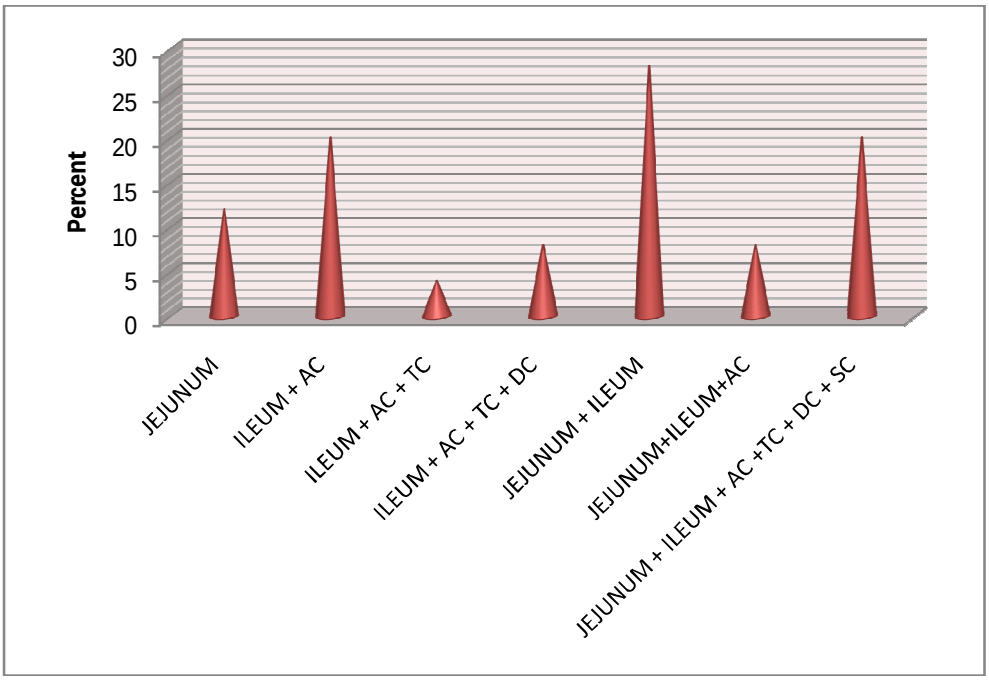


Chart : 27 Gangrene Segment

LENGTH OF GANGRENE SEGMENT (METER)

	Frequency	Percent
-	2	8.0
1.5M	2	8.0
1M	1	4.0
2.5M	4	16.0
2M	1	4.0
3.5M	2	8.0
3M	7	28.0
4.5M	1	4.0
4M	1	4.0
5M	2	8.0
6.5M	2	8.0
Total	**25**	**100.0**

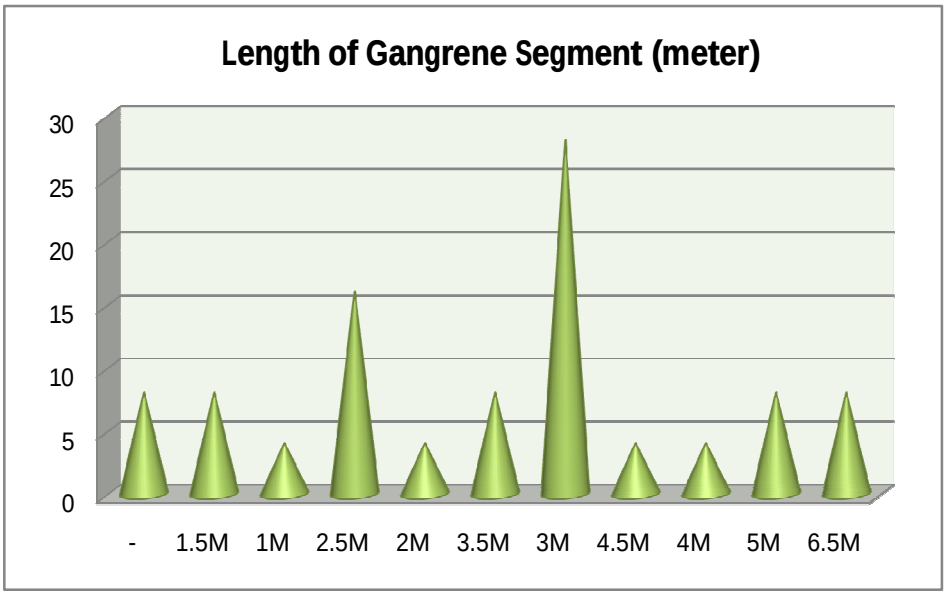


Chart : 28 **Length of Gangrene Segment (Meter)**

DIAGNOSIS

	Frequency	Percent
SMAE	2	8.0
SMAT	5	20.0
SMAT + IMAT	4	16.0
SMAT + SMVT	1	4.0
SMAT+SMVT+IMAT	1	4.0
SMVT	12	48.0
Total	**25**	**100.0**

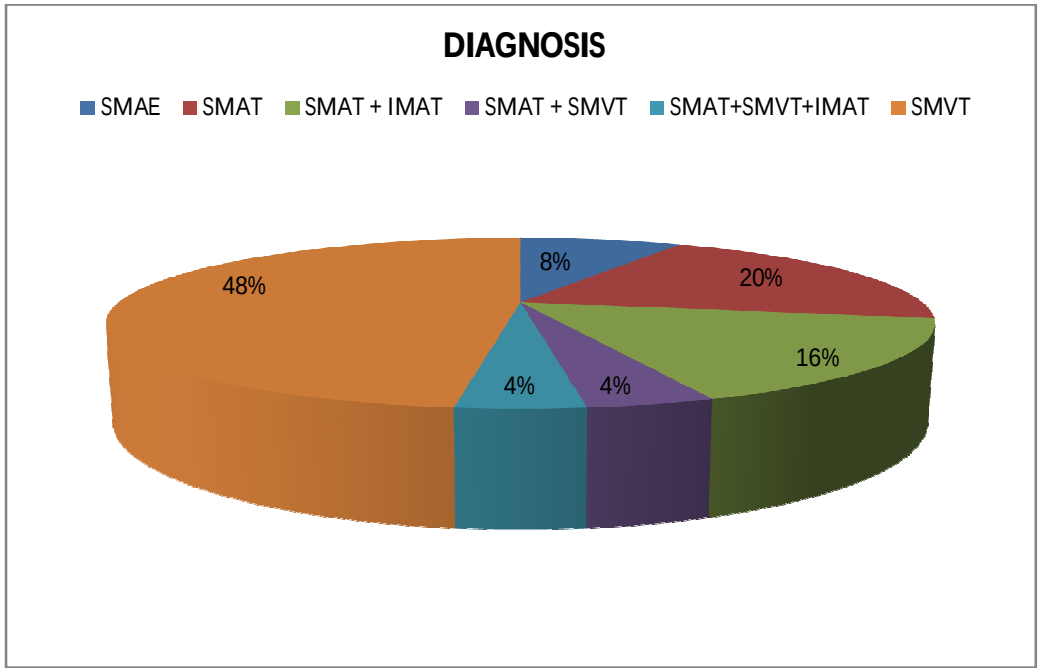


Chart : 29 Diagnosis

SURGERY

	Frequency	Percent
PRECEDURE DEFERRED	5	12.0
RESECTION+ILEOSTOMY	2	8.0
RESECTON+ILEO-TRANSVERSE ANASTAMOSIS	5	20.0
RESECTON+JEJUNUNO ILEAL ANASTAMOSIS	6	24.0
RESECTON + JEJUNO-JENUNAL ANASTAMOSIS	3	12.0
RESECTON + JEJUNO TRANSVERSE ANASTAMOSIS	4	20.0
Total	**25**	**100.0**

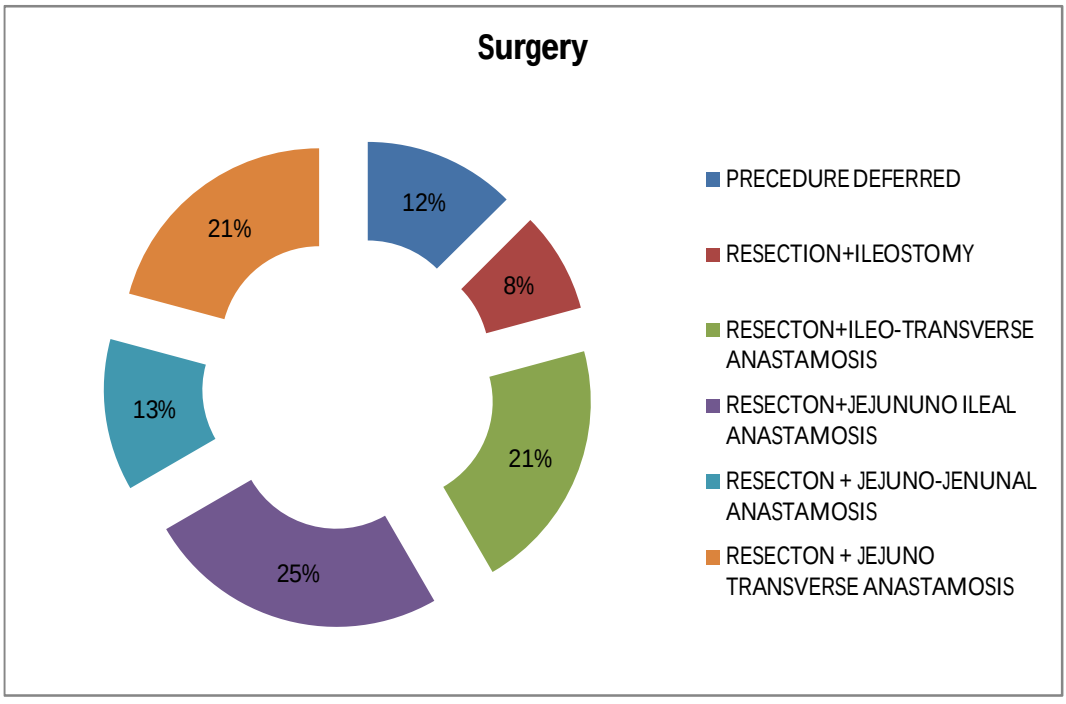


Chart : 30 Surgery

CARDIAC COMORBIDITY

	Frequency	Percent
N	15	60.0
AF	3	12.0
CCF	1	4.0
MR	6	24.0
Total	**25**	**100.0**

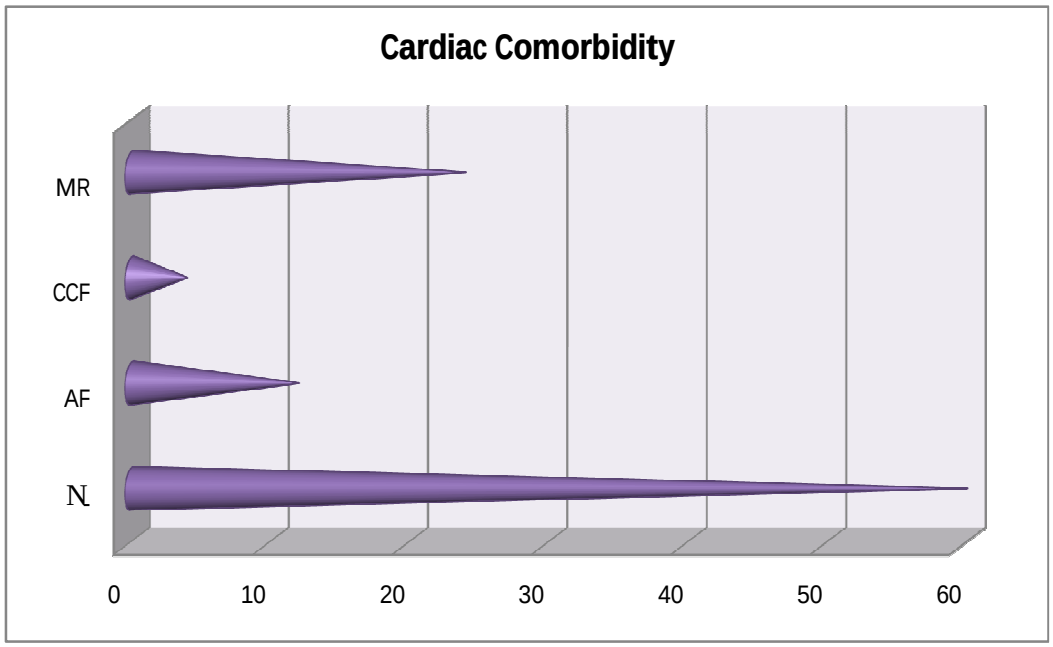


Chart : 31 Cardiac Comorbidity

ELAPSED TIME

	Frequency	Percent
<24HRS	6	24.0
<48HRS	1	4.0
>48HRS	18	72.0
Total	**25**	**100.0**

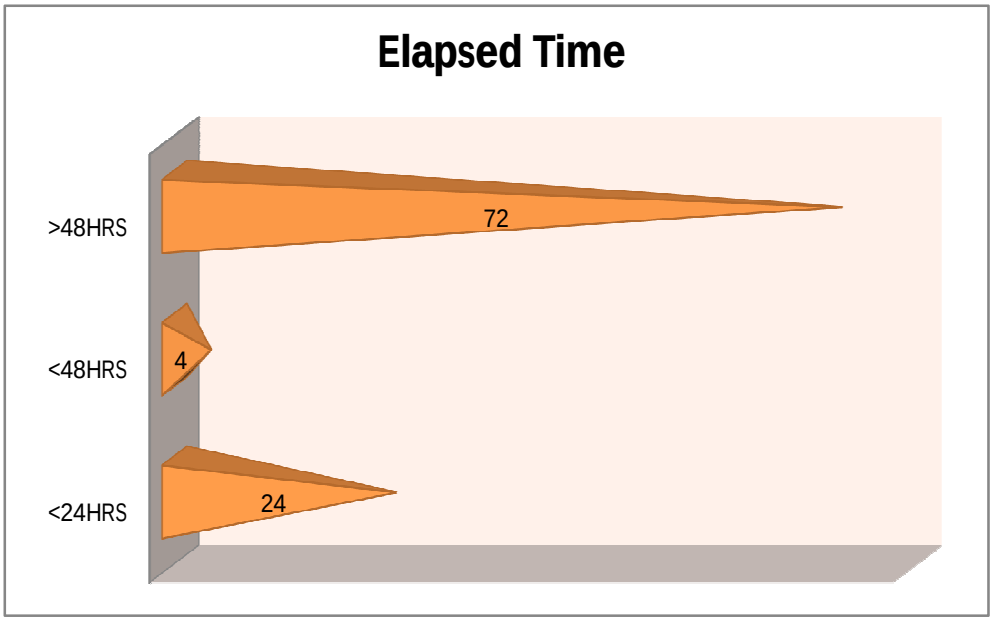


Chart : 32 Elapsed Time

MORBIDITY

	Frequency	Percent
-	4	16.0
AL	1	4.0
AL , S	1	4.0
AL, FF,S	1	4.0
AL,S	2	8.0
BA, AL,S	1	4.0
BA, FF	1	4.0
BA,S	1	4.0
BA/ AL	1	4.0
FF	1	4.0
FF, BA,S	1	4.0
FF,S	1	4.0
S	8	32.0
WI,S	1	4.0
Total	**25**	**100.0**

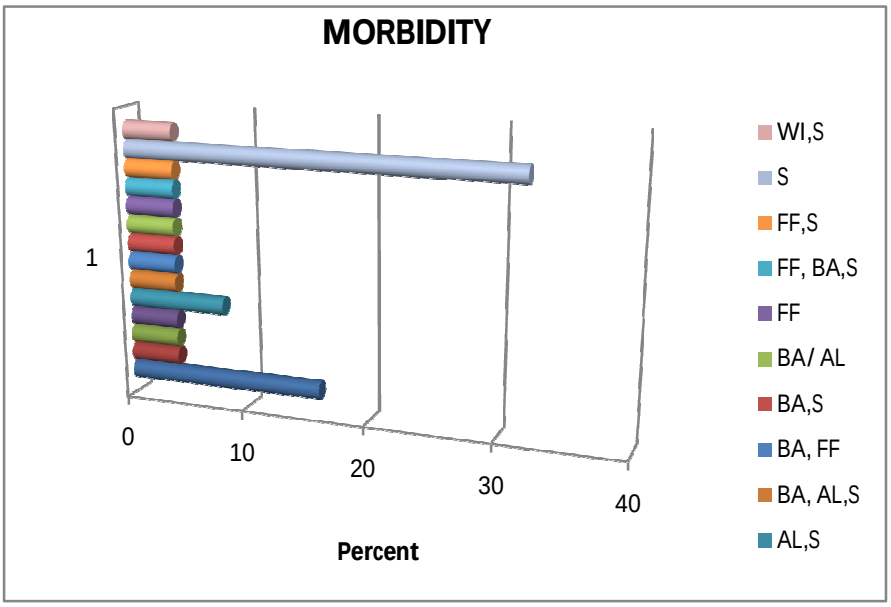


Chart : 33 Morbidity

MORTALITY

	Frequency	Percent
-	8	32.0
+	17	68.0
Total	25	100.0

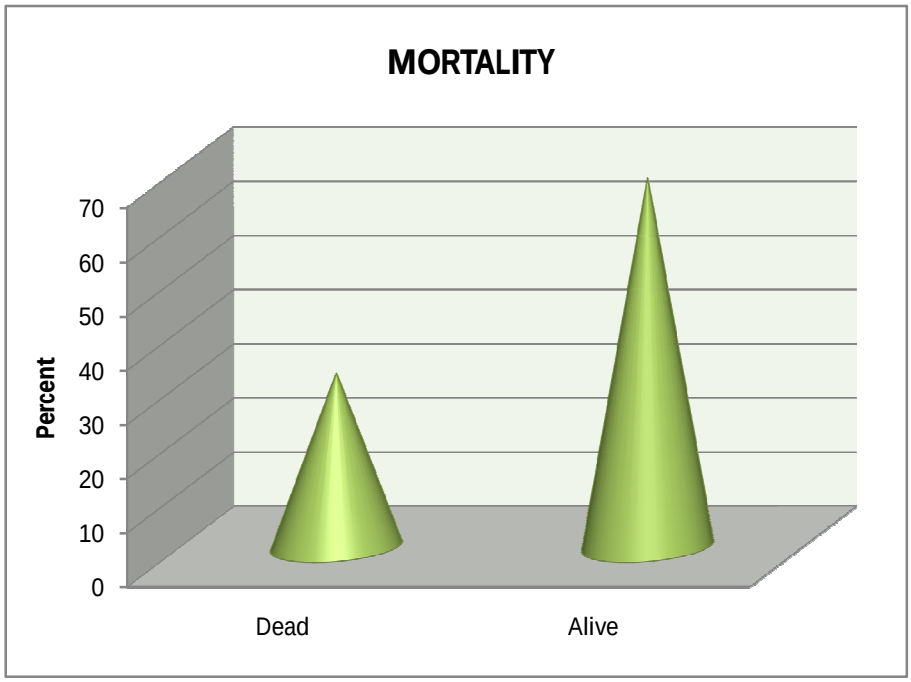


Chart : 34 Mortality

DISCUSSION

Acute Mesenteric Vasccular Occlusion is an infrequent but complicated, life-threatening condition. It is mostly seen in elderly patients. Despite the advances in diagnosis of Acute Mesenteric Vasccular Occlusion, morbidity and mortality rates remain high. Atypical symptoms, presence of predisposing diseases, delayed surgical intervention due to diagnostic difficulties, and in most cases, elderly patients who have cardiac problems, these may be some of the factors for higher mortality rates.

The youngest patient is 28 years old presented with SMA thrombus and the oldest is 74 years who presented with SMV thrombus, the peak incidence seen in 5 th to 6 th decade, with 64%.

Bowel gangrene is more common in males with 86%, when compared with females which was 12%, Mesenteric vascular occlusion may also be due to associated thrombogenic factors like, smoking and alcohol. 80% were smokers and 72% were alcoholics.

Diabetes and hypertension has a strong association in mesenteric vascular occlusion. Among the patients, 68% were diabetics, and 68% were hypertensives.

The most common presentation was abdominal pain in 76%. The pain started in one quadrant initially, later became diffuse to spread all over the abdomen, indicating the progression of bowel infarction. Predominant

symptom was obstipation seen in 76 %, vomiting in 68%, malena in 16% and pyrexia as a symptom in 24 % of patients.

Regarding signs, abdominal distension was a predominant sign in 68%, guarding and rigidity in 72%, tenderness in 96%, making it the most common sign among patients.

Bowel sounds was absent in 92% of patients. Per rectal examination revealed a normal finding in 52%, rectum was roomy in 36 %, and in 12% of patients, malena was seen.

Hemodynamic shock was an important sign in most of the patients, which was positive in 76%.

Lab investigations showed an elevated leucocytes in all the patients, making it the most constant marker in acute mesenteric vascular occlusion. Nearly 70 -80% of patients showed, elevated alkaline phosphatase levels too.

Azotemia was also predominant in patients. 76% of the patients presented with elevated urea and creatinine levels, mostly , pre- renal azotemia due to increased third space loss.

Four quadrant aspiration was done in all patients, and in 4 patients, a toxic hemorrhagic fluid was aspirated. All those four patients were cases of extensive small and large bowel necrosis.

X ray abdomen erect posture was done in all patients, 12% showed a normal study, 20% showed dilated bowel loops, predominantly 64% showed a

combination of dilated bowel loops with air fluid levels. One patient showed dilated bowel loops with minimal air under the diaphragm, thus presenting as a case of bowel necrosis with perforation. That patient had extensive gangrene of all the small and large bowel loops.

Ultrasound abdomen was also done in all the patients, which showed dilated bowel loops with sluggish peristalsis in 88% of the patients .

A superior mesenteric arterial and venous ultrasonic Doppler was done in about 15 patients, which showed no significant flow in SMA, with a suspected emboli in 2 patients, suspected arterial thrombosis in 4 patients, mesenteric venous thrombosis in 8 patients, 5 patients came with CT angiographic evidence of mesenteric arterial or venous thrombosis from private hospitals. Five patients were adviced CT angiogram for proving mesentric vascular occlusion as ultrasonographic Doppler was not much contributory.

ECG was taken in all patients, which showed a normal ECG in 68% and an abnormal ECG in 32% with atrial fibrillation and left ventricular failure changes.

Emergency laparotomy was done in all patients, with gangrenous segments involving only jejunum in 12%, ileum and ascending colon in 20%, ileum, ascending colon, transverse colon, and descending colon involvement in 8%, ileum, ascending colon, and transverse colon involvement in one patient, jejunum, ileum, ascending colon in 8% and extensive gangrene from jejunum to sigmoid colon was found in 20% of the patients.

Resection anastomosis was done in 20 patients with ileo-transverse anastomosis in 20%, jejuno-ileal anastomosis in 29%, jejuno-jejunal anastomosis in 12%, and jejuno-transverse anastomosis in 16% of the patients. Resection and ileostomy was done in 2 patients of whom three were descending colon and sigmoid colon involvement.

After laparotomy, the resection procedure was deferred in 5 patients as they had complete small and large bowel gangrene starting from jejunum to sigmoid colon. So, abdomen was closed again without doing any further.

During laparotomy, superior mesentric artery thrombosis was seen in 20% of the patients leading to bowel gangrene from jejunum to transverse colon. Both SMA and IMA thrombosis was seen in 16% leading to involvement of descending colon also, inaddition to other bowel loops. SMA embolism was seen in 8% of the patients, who already had atrial fibrillation and had segmental bowel infarction. Both SMA and IMA thrombosis,combined with superior mesenteric vein thrombosis was seen in one patient.

Combined SMAT and SMVT which is a rare entity was seen in one patient. The predominant finding in all the laparotomies which showed segmental bowel necrosis was SMVT.

The length of the resected segment varied from 1.5 meters to 6.5 meters with an average resection length of 2.5 to 3.5 meters.

The cardiac co- morbidity present in the patients were atrial fibrillation in 12% of which 8% patients presents with arterial embolism, congestive cardial failure in 4% , and mitral regurgitation with low cardiac ejection fraction in 24%.

The delayed time of presentation has shown to influence the morbidity and mortality.

In this study, mortality was reserved only to patients presenting more than 48 hours, of onset of symptoms and was 68%, 28% of patients who presented with in 24 hours of symptoms and one patient who presented from 24 to 48 hours of onset of symptoms survived. The increased mortality in late presentation is due to prolonged exposure of bacterial toxin into the peritoneal cavity leading to onset of septicemia.

In a report from from Madrid, 21 patients with SMA embolus , intestinal viability was achieved in 100% if the duration was less than 12 hours, 56% for more than 12 hours.

Sepsis was a predominant factor seen in all the patients who died. Increased morbidity was seen in patients with more than 48 hours delay , predominantly such as wound infection, wound gaping, respiratory infections, fecal fistula, post operative ventilator support for reduced respiratory effort, burst abdomen for which 4 patients went in for resuturing of anterior abdominal wall and anastamotic leak.

In this study, mortality was highly seen in patients presenting with isolated SMA or combined SMA and IMA thrombosis patients with SMV thrombosis had good survival rates. Four patients out 12 patients having SMV thrombosis died where as all the patients with SMA or SMA and IMA thrombosis died(11 patients), postoperatively due to complications making SMV involvement a favourable prognostic sign when compared to arterial thrombosis.

The two patients who presented with emboli of the SMA also died due to complications.

Considering the high mortality rate of patients with intestinal gangrene, prevention seems to be the most logically, proven strategy, to improve outcome if the high risk groups can be identified which include

1. History of previous thrombo embolic events
2. Rheumatic heart disease
3. Arrythmias
4. Patients taking digitalis, diuretics, estrogen and danazol
5. Patients with protein C and S and anti thrombin deficiency
6. Sickle cell disease
7. Diabetes and hypertension

In this case, three patients (12 %) presents with, atrial fibrillation of which in 2 patients, emboli of the SMA was evident. Treatment of chronic.

Atrial fibrillation with anti coagulants or cardio version and treatment of congestive cardiac failure have been demonstrated to significantly reduce the thrombo – embolic complications.

Patients should be educated regarding the benefits of regular treatment and complication of stopping the drug without physicians advice. Physician at the primary health care level, should be educated about the possibility of mesenteric vascular occlusion in high risk patients and refer them to higher centre immediately.

After the patient reaching the hospital, the following should be done to reduce mortality and morbidity.

1. Early antibiotic therapy- this will postpone the irreversible stage of septicemia. They protect against the emerging bacteremia.

2. No unnecessary delay in surgery if peritonitis of unknown etiology suspected because this will lead to irreversible hypotension due to prolonged exposure of bacterial toxins.

3. Judicial decision should be made between early surgical management to terminate exposure to gangrenous bowel toxin versus the adequate pre operative time in maximally correcting fluid and electrolyte imbalance.

4. Proper and correct technique of resection anastamosis of the bowel to be followed and the gangrenous part should be resected out immediately. The operative time should be reduced as much as possible.

CONCLUSION

1. Highest incidence was seen in 5 th decade.
2. Males are more predominantly affected than females.
3. Smoking and alcoholism are strong predisposing factors for the disease.
4. Advanced age is an unfavourable factor for good prognosis.
5. High leucocyte levels correlates with the disease sevearity and high mortality.
6. Increased elapsed time between the symptoms and the surgical intervention has an increasing effect on mortality.
7. Superior mesenteric vein thrombosis is more common than arterial thrombosis.
8. Superior mesenteric vein thrombosis carries a good prognosis when compared to arterial thrombosis or emboli.
9. Mortality is directly proportional to the length of the bowel involved.
10. Mortality is high in both intestinal and colon gangrene when compared to small bowel alone.

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20. Cuscheri's essential surgical practice

21. Maingot's abdominal surgery

22. Hamilton bailey emergency surgery.

23. Gray's anatomy

24. The Surgical Clinic of North America- intestinal ischemia.

25. Pathological basic of disease Robbin's

26. Human embryology- Inderbir Singh

27. Regional anatomy- snell's

28. Surgical clinics of north America-abdominal emergencies-acute mesenteric ischemia dec 1997.

29. Master of surgery Fischers General surgical operation by R.M Kirk

30. Copes early diagnosis of acute abdomen

31. Acute mesenteric ischemia-recent advances.

32. Curran's atlas of histopathology

33. Current surgical diagnosis and management 2015.

34. Diagnostic radiology- hepato-biliary and gastro intestinal imaging- Manarama Reddy.

35. Schwartz textbook of surgery

36. Rob and smith operative surgery

37. Schakelford alimentary tract surgery

38. Physiological basis of modern surgical care Thomas A Muller.

PROFORMA

I. Name : I.P.No: Case No:

Age : Date of symptom onset:

Date of admission:

Sex : Date of operation:

Religion: Date of discharge:

Address:

II. PRESENTING COMPLAINTS:

1.

2.

3.

4.

III. DETAILED HISTORY OF PRESENTING ILLNESS:

1. PAIN:

   a. Time and mode of onset:

   b. Situation:

   c. Character: type at onset/type at present

Colicky /burning /throbbing/ Sever agonizing pain.

d. Progress:

e. Radiation:

f. Related to bowel and micturition:

g. Relation to food:

2. VOMITING:

a. Present /absent.

b. Character: Projectile / regurgitation.

c. Frequency.

d. Quality. - Nature

- Colour

  Taste

- Odour.

e. Quantity

f. Associated material with vomitus.

g. Duration - continuous/ intermittent,

h. Progress.

i. Relation to pain:

j. Haematemesis: Present/absent

Colour

Frequency

Amount

3. DISTENSION OF ABDOMEN:

   a. Mode of onset: gradual /sudden.

   b. Site

   c. Duration

   d. Progress

4. BOWELS:

   a. Tenesmus

   b. Constipation

   c. Diarrhoea

   d. Dysentery

   e. Melena

5. FEVER: since days

   Continuous / intermittent/ remittent.

6. MICTURATION.

IV. PAST HISTORY:

a. Previous history of similar attacks:

b. Any previous abdominal surgery:

c. Hypertension

d. Tuberculosis

e. Diabetes

f. Jaundice

g. Drug allergy

V. FAMILY HISTORY:

a. Members - any member of family suffered from similar attacks.

b. Health status

VI. PERSONAL HISTORY:

a. Diet: vegetarian/mixed.

b. Habits: smoking yes/no

Alcohol yes/no

Betel nut yes/no

d. Sleep:

e. Appetite:

VII. MENSTURAL /OBSTETRIC HISTORY IN FEMALES:

VIII. SOCIO-ECONOMIC HISTORY

IX GENERAL EXAMINATION:

a. Build

b. Nourishment

c. Eyes:

d. Mouth:

e. Nails:

f. Cyanosis:

g- Pedal oedema:

h. Lymphadenopathy

i. Pulse:

j- B.P. mm of hg :

k. Respiration:

1. temperature:

m. jaundice

n. dehydration

X. LOCAL EXAMINATION:

Inspection :

1. Shape:
2. Movements:
3. Umbilicus:
4. Distension: local/general.
5. Visible peristalsis:
6. Visible mass
7. renal angle: fullness:+/-
8. hernia sites:

b. Palpation:

1. local rise of temperature
2. tenderness site

C. PERCUSSION:

- Obliteration of Liver Dullness

D. AUSCULTATION:

- Bowel Sounds

XI. EXTERNAL GENITALIA:

XII. P/R EXAMINATION AND P/V EXAMINATION IN FEMALES:

XIII. SYSTEMIC EXAMINATION:

a. Respiration system

b. Cardiovascular system:

c. Central nervous system.

XIV. PROVISIONAL DIAGNOSIS:

XV. FINDINGS FOR DIAGNOSIS

XVI. INVESTIGATIONS

1. BLOOD INVESTIGATION:

   a. Hb%

   b. Blood grouping

   c. TC,DC,ESR

   d. Blood urea, serum creatinine

   e. Random blood sugar

   f. Serum electrolytes

2. URINE EXAMINATION

a. Albumin

b. Sugar

c. Microscopy

3. RADIOLOGICAL INVESTIGATIONS

a. Plain x ray of abdomen

b. Contrast studies

c. Other relevant radiological investigations

4. Ultrasound examination, USG Doppler

5. CECT scan, CT Angiogram

6. M.R.I

7. Arteriography

8. Laproscopy

9. 4 quadrant aspiration

10. stool examination

XVII. FINAL DIAGNOSIS

XVIII. TREATMENT

1. Treatment during resuscitation

2. Operation
3. Operative findings
4. Preoperative diagnosis
5. Post operative diagnosis
6. Post operative treatment
7. Post operative complication
8. Advice on discharge
9. Conclusion
10. Biopsy report

XIX. FOLLOW UP

IST Follow up (1st week) 2nd follow up(l month) 3rd follow up(3months)

MASTER CHART

S.No	Name	Age	Sex	IP No	Abdomen pain	HISTORY						SYMPTOMS					SIGNS					
						Duration of symptoms in hours	Diabetic	SHT	Smoker	Alcoholic	Tee	Vomiting	Diahhorea	Blood in Stools	Obstipation	Pyrexia	ABD distension	Guarding/rigidity	Tenderness	Bowel sounds	Per Rectal	Shock
1.	SAMBATH	53	M	59808	+	>48	+	+	+	-	-	+	-	-	+	-	+	+	+	-	RR	+
2.	KUPPAN	57	M	77734	+	>48	+	+	+	+	-	+	-	-	+	-	+	+	+	-	M+RR	+
3.	DEVARAS	74	M	18562	+	<24	+	+	+	-	-	+	-	-	-	-	+	-	+	-	RR	-
4.	CHINNUPALANI	50	M	25541	+	<48	+	-	-	-	-	+	-	+	-	-	-	-	+	-	N	-
5.	MURUGESH	40	M	31176	-	>48	-	+	+	+	+	-	+	-	+	+	+	-	+	-	N	+
6.	ITTUSAMY	50	M	32357	+	<48	-	-	+	+	-	-	-	-	-	-	+	+	+	+	RR	+
7.	RAJAN	60	M	56179	-	>48	+	+	+	+	-	-	-	-	+	-	-	+	-	-	M+RR	-
8.	PACANIAMMAL	65	F	63758	+	>48	+	+	-	-	-	+	-	-	+	-	-	+	+	-		+
9.	ANANDHA KUMAR	50	M	75476	+	>48	-	+	+	-	-	+	+	-	+	+	-	-	+	-	N	+
10.	RAMASAMY	60	M	80346	+	>48	-	-	+	+	-	-	-	-	+	+	+	+	+	-	N	+
11.	AYYASAMY	49	M	81489	-	>48	-	+	+	-	-	+	-	+	+	-	+	+	+	-	N	+
12.	PALANIAMMAL	62	F	23027	+	>48	+	-	-	-	+	+	-	-	-	-	+	+	+	-	RR	+
13.	SOUNDAR RAJA	30	M	45433	+	>48	-	-	+	+	+	-	-	-	+	-	+	+	+	-	N	+

14.	DEVANDRAN	50	M	49488	+	<24	+	-	+	+	-	-	-	-	+	+	+	+	+	-	RR	-
15.	RENGANATHAN	47	M	64583	+	>48	+	+	+	+	-	+	-	-	+	-	+	+	+	-	M+RR	+
16.	VELLAISAMY	35	M	31894	+	<48	-	-	+	+	-	+	-	-	+	-	+	-	+	-	RR	+
17.	GOVINDARAJ	41	M	58081	-	<24	+	+	+	+	-	+	-	-	+	+	-	-	+	+	N	+
18.	RADHAKRISHNAN	55	M	24918	-	>48	-	-	+	+	-	+	-	+	+	-	+	+	+	-	RR	-
19.	KRISHNAVENI	47	F	24254	+	<24	-	+	-	+	+	+	+	-	-	-	-	-	+	-	N	+
20.	KANDHASAMY	60	M	27852	+	<24	+	+	+	+	-	+	-	-	-	-	+	+	+	-	N	+
21.	DURAISAMY	40	M	20952	+	>48	-	-	+	+	-	-	-	-	+	-	+	+	+	-	RR	+
22.	JOHN PONNUSAMY	55	M	21207	+	>48	+	+	+	+	-	-	-	+	+	-	+	+	+	-	N	+
23.	KAJAMOHIDEEN	28	M	41718	-	>48	-	-	+	+	-	+	+	-	+	+	+	+	+	-	RR	-
24.	SURENDRAN	40	M	38418	+	>48	-	-	+	+	-	+	-	-	+	-	-	+	+	-	N	+
25.	MURUGAN	40	M	61241	+	>48	-	-	-	+	-	+	+	-	+	-	-	+	+	-	N	+

RR – RECTUM ROOMY
M – MALENA

INVESTIGATION								Gangrene segment	Length of Gangrene Segment (meter)	Diagnosis	Surgery	Cardiac Comorbidity	Elapsed Time	Morbidity	Mortality
S.No	Name	Leucocytosis	Urea /	X Ray ABD	Four	USG ABD	ECG								
1.	SAMBATH	+	+	DBL+AFL	-	+	N	J+I	3M	SMVT	R+JIA	MR	>48HRS	AL, FF,S	+
2.	KUPPAN	+	+	DBL	-	+	-	I+AC	3.5M	SMAE	R+ITA	AF	>48HRS	WI,S	+
3.	DEVARAS	+	+	DBL+AFL	-	+	N	J+I	2.5M	SMVT	R+JIA	-	<24HRS	BA/ AL	-
4.	CHINNUPALANI	+	-	DBL	-	N	N	J	1.5M	SMVT	R+JJA	-	<48HRS	-	-
5.	MURUGESH	+	+	DBL+AFL	-	+	-	I+AC	2M	SMAE	R+JTA	AF	>48HRS	S	+
6.	ITTUSAMY	+	+	NS	-	N	N	J	1M	SMVT	R+JJA	MR	>48HRS	-	-
7.	RAJAN	+	+	DBL	+	+	N	J+I+AC+TC+DC+SC	6.5M	SMAT+ IMAT	PD	-	>48HRS	S	+
8.	PACANIAMMAL	+	+	DBL+AFL	-	+	N	J+I+AC+ TC+DC	5M	SMAT+ SMVT +IMAT	PD	-	>48HRS	S	+
9.	ANANDHA KUMAR	+	-	DBL+AVD	+	+	N	I+AC	3M	SMVT	R+ITA	-	>48HRS	AL , S	+
10.	RAMASAMY	+	+	DBL	-	+	-	I+AC	3M	SMAT	R+ITA	-	>48HRS	S	+
11.	AYYASAMY	+	-	DBL+AFL	+	+	-	I+AC+TC +DC+SC	6.5M	SMAT +IMAT	PD	MR	>48HRS	S	+

12.	PALANIAMMAL	+	+	DBL+AFL	-	+	N	J+I	3.5M	SMVT	R+JIA	CCF	>48HRS	BA, AL,S	+
13.	SOUNDAR RAJA	+	+	DBL+AFL	-	+	N	J+I+AC	4.5M	SMAT	R+JTA	MR	>48HRS	FF,S	+
14.	DEVANDRAN	+	+	NS	-	+	-	J	1.5M	SMVT	R+JJA	AF	<24HRS	FF	-
15.	RENGANATHAN	+	+	DBL+AFL	-	+	N	J+I+AC+TC+DC+SC	6.5M	SMAT +IMAT	PD	-	>48HRS	BA,S	+
16.	VELLAISAMY	+	-	DBL+AFL	+	N	N	J+I	2.5M	SMVT	R+JIA	-	>48HRS	FF, BA,S	+
17.	GOVINDARAJ	+	+	DBL+AFL	-	+	N	J+I+AC	3M	SMAT	R+JTA	-	>48HRS	AL,S	+
18.	RADHAKRISHNAN	+	+	DBL	-	+	N	J+I	2.5M	SMVT	R+JTA	MR	<24HRS	-	-
19.	KRISHNAVENI	+	-	DBL+AFL	-	+	-	I+AC+TC	2.5M	SMAT	R+ITA	MR	>48HRS	S	+
20.	KANDHASAMY	+	-	DBL+AFL	-	+	N	I+AC+TC +DC	4M	SMAT	R+I	-	>48HRS	S	+
21.	DURAISAMY	+	+	DBL+AFL	-	+		J+I	3M	SMVT	R+JIA	-	<24HRS	BA, FF	-
22.	JOHN PONNUSAMY	+	+	DBL+AFL	-	+	N	J+I	3M	SMVT	R+JIA	-	<24HRS	AL	-
23.	KAJAMOHIDEEN	+	+	DBL+AFL	-	+	N	J+I+AC+TC+DC+SC	6M	SMAT +IMAT	PD	-	>48HRS	AL,S	+
24.	SURENDRAN	+	+	NS	-	+	N	I+AC+TC +DC	5M	SMAT+SMVT	R+I	-	>48HRS	S	+
25.	MURUGAN	+	+	DBL+AFL	-	+	-	I+AC	3M	SMVT	R+ITA	-	<24HRS	-	-

J

-JEJUNUM, I-ILEUM, AC-ASCENDING COLON, TC-TRANSVERSE COLON, DC-DESCENDING COLON, SC-SIGMOID COLON, R-RESECTON, JIA-JEJUNO-ILEAL ANASTAMOSIS, JJA-JEJUNO-JENUNAL ANASTAMOSIS, ITA-ILEO-TRANSVERSE ANASTAMOSIS, PD-PRECEDURE DEFERRED, ISA-ILEO-SIGMOID ANASTAMOSIS, SMAT-SUPERIOR MESENTERY ARTERIAL THROMBOSIS, SMVT-SUPERIOR MESENTERIC VEIN THROMBOSIS, IMAT – INFERIOR MESENTERIC ARTERY THROMBOSIS

S-SEPSIS, I-ILEOSTOMY, DLB – DILATED BOWEL LOOPS, AMI – ACUTE MESENTERIC ISCHEMIA, AFL – AIRFLUID LEVELS, NS – NORMAL STUDY, FF – FECAL FISTULA, AL – ANASTOMOTIC LEAK, BA – BURST ABDOMEN, MR – MITRAL REGURGITATION, AF – ATRIAL FIBRILLATION.

STATEMENT OF CONSENT

I, _____, do hereby volunteer and consent to participate in this study being conducted by **Dr. P.VENKATESH PRABHU**. I have read and understood the consent form / or it has been read and explained to me in my own language. The study has been fully explained to me, and I may ask questions at any time.

Signature / Left Thumb Impression of the Volunteer Date: Place:

Signature and Name of witness Date: Place:

Signature of the investigator:

Name of the investigator:

xggpjy;gotk;

bgah; :

ghypdk; :

Kfthp : taJ :

muRnfhit kUj;Jtf;fy;Yhpapy; bghJ mWit rpfpr;ir Jiwapy; gl;l nkwgogg[gapYk; khzth; **gh. bt'Tfnl#] gpug[** mth;fs; nkwbfhs;Sk; **" RISK FACTORS EFFECTING MORTALITY IN ACUTE MESENTERIC VASCULAR OCCLUSION - A SINGLE EXPERIENCE"** vdw nrhjidapd; brayKiw kwWk; midj;J tpgu';fisak; nfl;Lfbfhz;lJld; vdJ midj;J renjf';fisak; bjspthpggLj;jpfbfhz;nld;vdgij bjhptpj;Jf;bfhs;fpnwd;

ehd; ,e;j Ma;tpy; KG rkkjj;JlDk; Ra re;jidak;lDk; fye;J bfhs;s rkkjpf;fpnwd;

,e;j Ma;tpy; vd;Dila midj;J tpgu';fSk; ghJfhf;fggLtJld; ,jd; Kot[fs; Ma;tpjHpy; btspapl ggLtjpy; vdf;F ve;j Mlnrgidak;, y;iy vdgij bjhptpj;Jf;bfhs;fpnwd; ve;j neuj;jpYk; ,e;j Ma;tpy; ,Ue;J tpyfpfbfhs;s vdf;F chpik cz;Lvdgijak;mwpntd;

,lk; :

njjp : **ifbahggk;/ nuif**