Dissertation on

"PROSPECTIVE STUDY ON VARIOUS FACTORS (DEMOGRAPHIC, CLINICAL, BIOCHEMICAL) IN PATIENTS WITH ACUTE MESENTERIC ISCHEMIA"

Dissertation submitted to

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI.

in partial fulfilment of the regulations for the Award of the degree of

M.S. (General Surgery)

Branch – I

THE TAMILNADU Dr. MGR MEDICAL UNIVERSITY

CHENNAI

MAY 2020

CERTIFICATE

This is to certify that, the dissertation entitled

"PROSPECTIVE STUDY ON VARIOUS FACTORS (DEMOGRAPHIC, CLINICAL, BIOCHEMICAL) IN PATIENTS WITH ACUTE MESENTERIC ISCHEMIA"

Is the bonafide work done by **DR.SHAYEE KALYEE SHANJEEV.P.B** during his **M.S. (General Surgery)** course **2017-2020**, done under my supervision and is submitted in partial fulfilment of the requirement for the M.S.(BRANCH-I)- General Surgery of The Tamilnadu Dr.MGR Medical University, May 2018 examination.

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DECLARATION

I, certainly declare that this dissertation titled **"PROSPECTIVE STUDY ON VARIOUS FACTORS (DEMOGRAPHIC, CLINICAL, BIOCHEMICAL) IN PATIENTS WITH ACUTE MESENTERIC ISCHEMIA"** represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice, and are acknowledged.

I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other University board, either in India or abroad. This is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfilment of the rules and regulations for the award of Master of Surgery Degree Branch I (General Surgery).

Place: Chennai (**DR.SHAYEE KALYEE SHANJEEV.P.B)**

Date: Post Graduate

ACKNOWLEDGEMENT

As I walk down the memory lane, I realize with a deep sense of humility that what I have done now would not have materialized, but for certain luminaries, who have enlightened my path to wisdom.

"Surgery is learnt by apprenticeship and not from textbooks, not even one profusely illustrated"- Ian Aird.

While I put these words together it is my special privilege and immense pleasure to record my deep sense of gratitude and indebtness to my retired chief **Prof. A. AFFEE ASMA M.S., DGO,** Institute of General Surgery, Madras Medical College, Rajiv Gandhi Government General Hospital, Chennai. Her valuable and critical suggestions, constant inspiration and encouragement, timeless efforts, unstinted cooperation and mental support throughout have made this marathon task a smooth journey. Words cannot simply express my gratitude to her for imparting to me the surgical skills that I have acquired.

I place on record my profound gratitude to my guide, **Prof. V. RAMALAKSHMI M.S., D.A.,** for her support, keen interest and constant encouragement she has given during the course of this thesis work.

I thank my Director of Institute of General Surgery **Prof. R.KANNAN, M.S.,** for leading an example and being constant source of motivation and support during all struggles that occurred during this study.

I thank my beloved Dean, Madras Medical College & Rajiv Gandhi Government General Hospital for having permitted me to carry out this study in this esteemed institution.

An ostentatious use of words will not be sufficient to express my heartiest thanks to my ever friendly Assistant Professors Dr. Joyce Prabakar M.S, Dr. Padmanaban M.S, Dr. Vimala M.S., D.G.O., and Dr. Karthikeyan M.S for all of them have given me invaluable advice, guided me on and have been so kind and patient to me.

My sincere thanks to all other teaching staffs of Institute of General Surgery for their understanding, inspiration and affectionate encouragement right from the budding stage of this works.

My sincere thanks to the Institute of Biochemistry and The Institute of Pathology for helping me in completing my study.

I take this opportunity to thank my seniors, my colleagues and my friends for their continuous help and support which gave strength to my thoughts and mapped my way to find methodology for myself.

Sometimes it is not easy to express in words especially when you have to say thanks to your family for their constant undemanding love, dedication, sacrifice, affectionate encouragement and never ending enthusiasm, without which this study would not have been possible.

My heartfelt thanks go to each and every patient who agreed to be a part of this study and my sincere apologies to them in case any inconvenience caused.

CONTENTS

PATIENT PROFORMA:

PATIENTS NAME

IP NO

AGE/SEX

PREVIOUS HISTORY

BODY TEMPERATURE

SIGNS OF PERITONITIS

WBC COUNT

PLATELET COUNT

MEAN PLATELET VOLUME

RBC DISTRIBUTION WIDTH

SERUM AMYLASE

D-DIMER

METABOLIC ACIDOSIS

INFORMATION SHEET

TITLE: **"PROSPECTIVE STUDY ON VARIOUS FACTORS (DEMOGRAPHIC, CLINICAL, BIOCHEMICAL) IN PATIENTS WITH ACUTE MESENTERIC ISCHEMIA"**

Name of Investigator:

Name of Participant:

Purpose of Research: To Analyse Various Demographic, Clinical And Biochemical Factors In Patents Diagnosed With Acute Mesentric Ischemia , So These Factors May Help In Diagnosing Acute Mesentric Ischemia In Patients Coming With Acute Abdominal Pain In Emergency Settings In The Absence Of Imaging Modality

Study Design: Prospective Observational Study

Study Procedures: patients who were admitted as acute abdominal pain and diagnosed as acute mesenteric ischemia will be subjected to detailed history taking, general and systemic examination, routine and specific blood investigations, and the course of their hospital stay and the data analysed

Possible Risks: No risks to the patient

Possible benefits

To patient : Early diagnosis of the disease permits early treatment which in turn improves survival rates, mortality and morbidity.

To doctor & to other people: If this study gives positive results, it can help in early diagnosis of acute mesenteric ischemia in emergency settings among patients presenting with acute abdominal pain in the absence of radiological investigations.

Confidentiality of the information obtained from you: The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared

Can you decide to stop participating in the study: Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time

How will your decision to not participate in the study affect you: Your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant

Date :

Place :

PATIENT CONSENT FORM

Study Detail : **"PROSPECTIVE STUDY ON VARIOUS FACTORS (DEMOGRAPHIC, CLINICAL, BIOCHEMICAL) IN PATIENTS WITH ACUTE MESENTERIC ISCHEMIA"**

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Patient's Name :

Patient's Age :

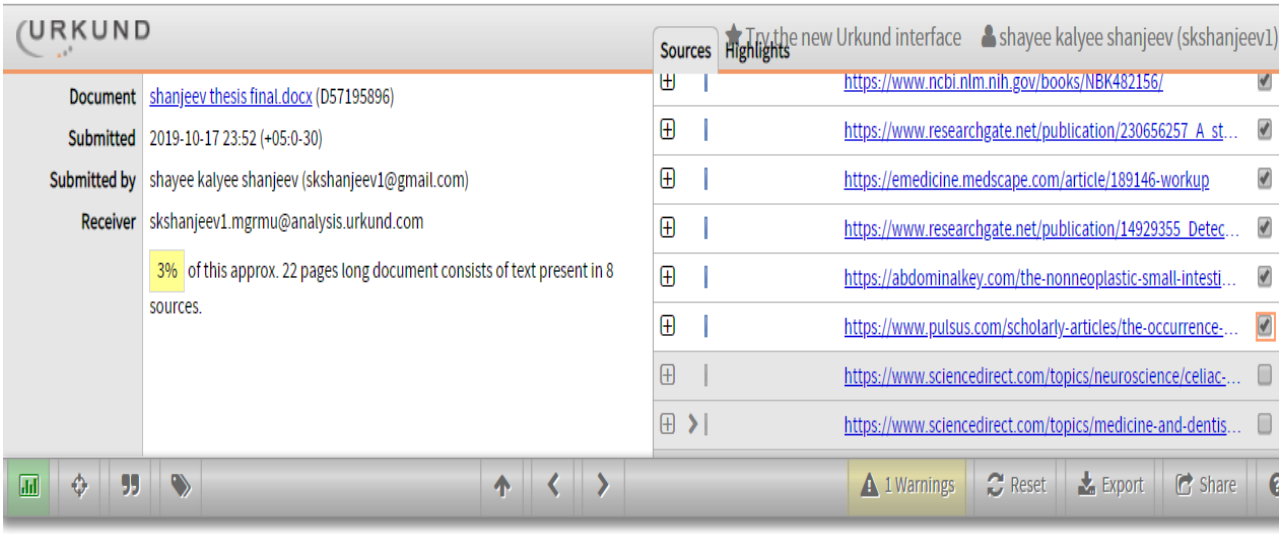
In Patient Number :

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.	❑
I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.	❑
I understand that sponsor of the clinical study, others working on the sponsor's behalf, the Ethics committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.	❑
I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.	❑
I hereby consent to participate in this study	❑
I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests and to undergo treatment	❑

Signature/thumb impression

Patient's Name and Address:



INTRODUCTION Mesenteric ischemia is an acute or chronic perfusion

84% #1 Active

Acute mesenteric ischemia (AMI) is a life-threatening vascular emergency, with a mortality

External source: https://www.sciencedirect.com/topics/medicine-... 84%

Acute mesenteric ischemia is a life-threatening vascular emergency associated with a 60–80% mortality

of up to 80%. However, there has been a slight improvement in recent years, perhaps due to wider use of contrast-enhanced CT allowing earlier definitive diagnosis and better control of atrial fibrillation, reducing systemic thromboembolism, one of the major underlying causes. Chronic mesenteric ischemia (CMI) is a more insidious disease, which can cause severe cachexia and result in a significant reduction in patient quality of life. Around 20% of patients with CMI go on to develop AMI, offering a high-risk population in whom earlier diagnosis and treatment could potentially reduce the catastrophic impact of AMI. Unfortunately however, many patients' first presentation is with bowel infarction, posing the question of how these patients can be identified earlier. The diagnosis of mesenteric ischemia is clinically difficult, due to the non-specific symptoms and signs. However, AMI is an important diagnosis to consider typically in elderly patients with acute abdominal pain out of proportion to clinical signs, particularly those with a previous history of vascular disease or atrial fibrillation. CMI is a diagnosis of exclusion, but a history of postprandial pain and weight loss is suggestive, highlighting the importance of taking a careful patient history. Early diagnosis is important to improve patient symptoms and to prevent bowel infarction with its associated morbidity and mortality. AIMS AND

Urkund Analysis Result

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https://www.sciencedirect.com/topics/medicine-and-dentistry/inferior-mesenteric-artery
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Instances where selected sources appear:

8

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.Shayee Kalyee Shanjeev.P.B.
Post Graduate in MS General Surgery
Institute of General Surgery
MMC/Chennai

Dear Dr.Shayee Kalyee Shanjeev.P.B.,

The Institutional Ethics Committee has considered your request and approved your study titled **"PROSPECTIVE STUDY ON VARIOUS FACTORS (DEMOGRAPHIC, CLINICAL, BIOCHEMICAL) IN PATIENTS WITH ACUTE MESENTERIC ISCHEMIA " - NO.16122017**

The following members of Ethics Committee were present in the meeting hold on **12.12.2017** conducted at Madras Medical College, Chennai 3

1. Prof.P.V.Jayashankar :Chairperson
2. Prof.R.Narayana Babu,MD.,DCH., Dean,MMC,Ch-3 : Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3 : Member Secretary
4. Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch : Member
5. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3 : Member
6. Prof.A.Pandiya Raj,Director, Inst. of Gen.Surgery,MMC : Member
7. Prof.Shanthy Gunasingh, Director, Inst.of Social Obstetrics,KGH : Member
8. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai : Member
9. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3 : Member
10. Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3 : Member
11. Prof.Bharathi Vidya Jayanthi,Director, Inst. of Pathology,MMC,Ch-3: Member
12. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
13. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
14. Thiru K.Ranjith, Ch- 91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary - Ethics Committee

INTRODUCTION

Mesenteric ischemia is an acute or chronic perfusion abnormality of the gastrointestinal tract. Acute mesenteric ischemia (AMI) is a life-threatening vascular emergency, with a mortality of up to 80%. However, there has been a slight improvement in recent years, perhaps due to wider use of contrast-enhanced CT allowing earlier definitive diagnosis and better control of atrial fibrillation, reducing systemic thromboembolism, one of the major underlying causes. Chronic mesenteric ischemia (CMI) is a more insidious disease, which can cause severe cachexia and result in a significant reduction in patient quality of life. Around 20% of patients with CMI go on to develop AMI, offering a high-risk population in whom earlier diagnosis and treatment could potentially reduce the catastrophic impact of AMI. Unfortunately however, many patients' first presentation is with bowel infarction, posing the question of how these patients can be identified earlier.

The diagnosis of mesenteric ischemia is clinically difficult, due to the non-specific symptoms and signs. However, AMI is an important diagnosis to consider typically in elderly patients with acute abdominal pain out of proportion to clinical signs, particularly those with a previous history of vascular disease or atrial fibrillation. CMI is a diagnosis of exclusion, but a history of postprandial pain and weight loss is suggestive, highlighting the importance of taking a careful patient history. Early diagnosis is important to improve patient symptoms and to prevent bowel infarction with its associated morbidity and mortality.

AIMS AND OBJECTIVES

To Analyse Various Demographic, Clinical And Biochemical Factors In Patents Diagnosed With Acute Mesenteric Ischemia , So These Factors May Help In Diagnosing Acute Mesenteric Ischemia In Patients Coming With Acute Abdominal Pain In Emergency Settings In The Absence Of Imaging Modality

REVIEW OF LITERATURE

INTRODUCTION:

Abrupt interruption or diminution of blood flow to the small intestine or the colon leads to acute mesenteric ischemia (AMI). It describes a wide spectrum of bowel injury ranging from reversible alterations in bowel function to transmural necrosis of the bowel wall.

GROSS ANATOMY:

Small intestine extends from the pylorus to the caecum and it is a tubular structure. Length is variable from person to person and measures on an average between 4 to 6 metres. It has three segments: Duodenum- most proximal and lies in the retro peritoneum. Starts after pylorus and demarcated from the jejunum by ligament of treitz. Jejunum and Ileum- lie within the peritoneal cavity and are attached to the retro peritoneum by a broad based mesentery. There is no anatomical landmark that delineates jejunum from ileum. Proximal 40% is arbitrarily divided as jejunum and distal 60% is considered as ileum. Ileum is demarcated from the caecum by the ileocaecal valve.

Internal mucosal folds of the small intestine are known as **valvulae coniventes or plicae circulars.** They are visible on gross inspection. On gross examination small intestinal mucosa also reveals aggregates of lymphoid

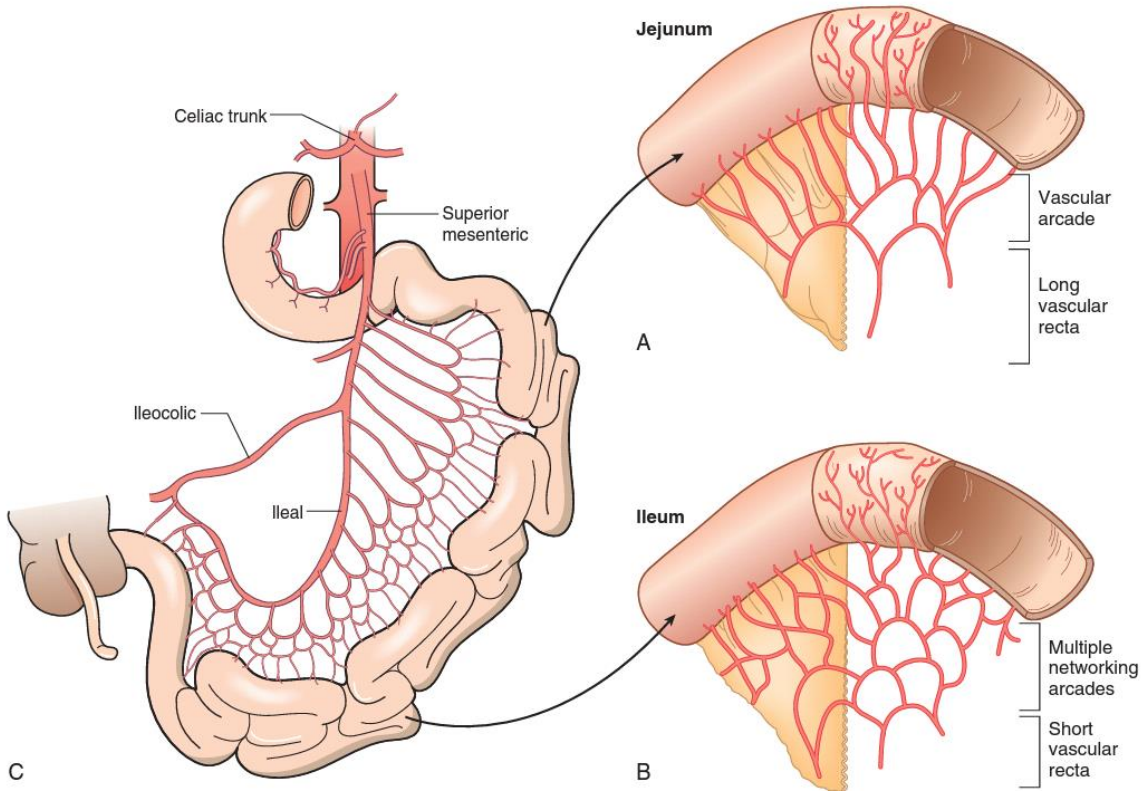
follicles called as **payer's patches**. Difference between jejunum and ileum on gross inspection are as follows

GROSS INSPECTION	**JEJUNUM**	**ILEUM**
Valvulae coniventes	More prominent	Less prominent
Circumference	Larger	Smaller
Wall	Thickened	Thin
Mesentery	Less fatty	More fatty
Vasa recta	Larger	Smaller

BLOOD SUPPLY:

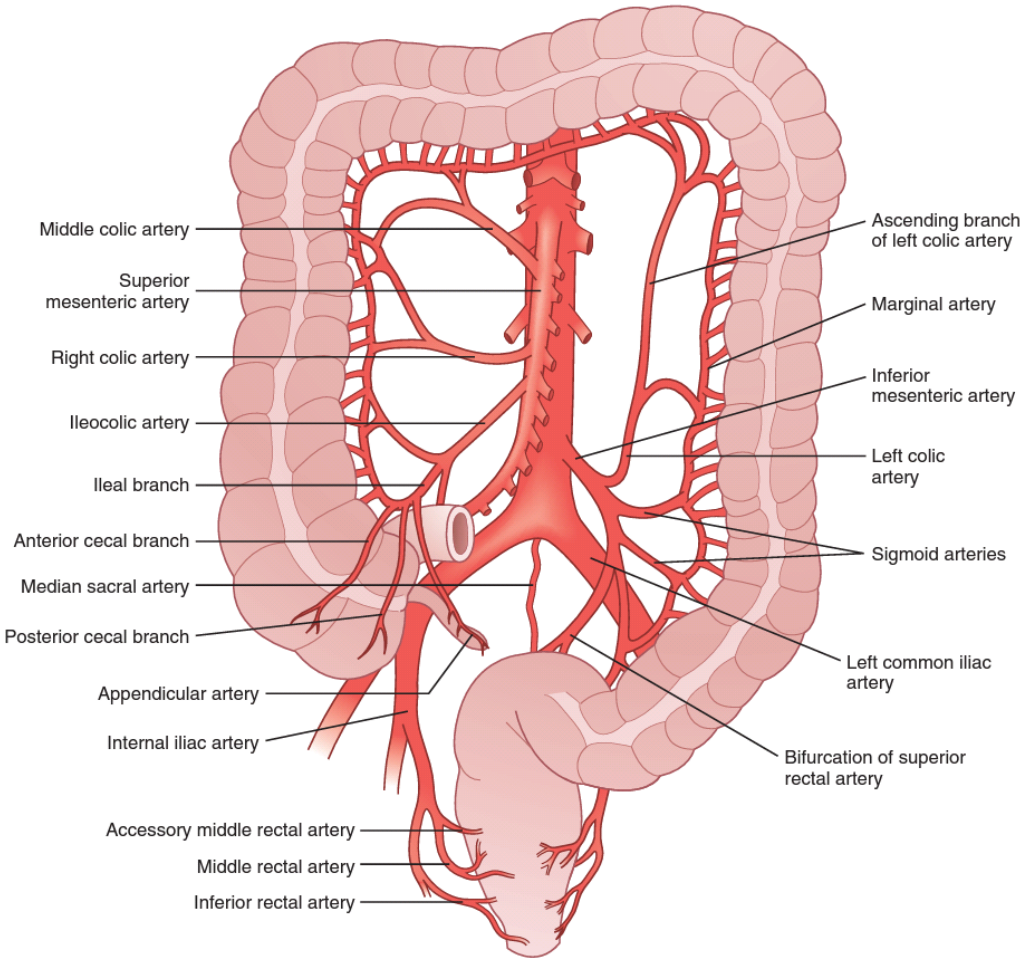
Duodenum- The main vessels supplying the duodenum are the superior and inferior pancreaticoduodenal arteries. The first and second parts also receive Contributions from other sources, including the right gastric, supraduodenal, Right gastroepiploic, hepatic and gastroduodenal arteries.

Jejunum and ileum –receives blood supply from the jejunal and ileal branches of SMA



LARGE INTESTINE

The arterial supply of the large intestine is derived from both the superior and the inferior mesenteric arteries .The caecum, appendix, ascending colon and proximal two-thirds of the transverse colon (derived from the midgut) are supplied from ileocolic, right colic and middle colic branches of the superior mesenteric artery. The distal third of the transverse colon, descending and sigmoid colon, rectum and upper anal canal (hindgut derivatives) are supplied predominantly from the inferior mesenteric artery via the left colic, sigmoid and superior rectal arteries, with a small contribution from branches of internal iliac artery (the middle and inferior rectal arteries).



Superior mesenteric artery:

The artery of the midgut is the superior mesenteric artery, which arises from the anterior surface of the aorta at the level of the lower border of L1. It then enters the root of the mesentery of the small intestine and passes obliquely downwards and to the right, giving off several branches to the large intestine.

1. **Ileocolic artery** arises near the root of the mesentery, descending within the mesentery to the right towards the caecum, and divides into superior and inferior branches, the superior branch running up along the left side of the ascending colon to anastomose with the right colic artery. The

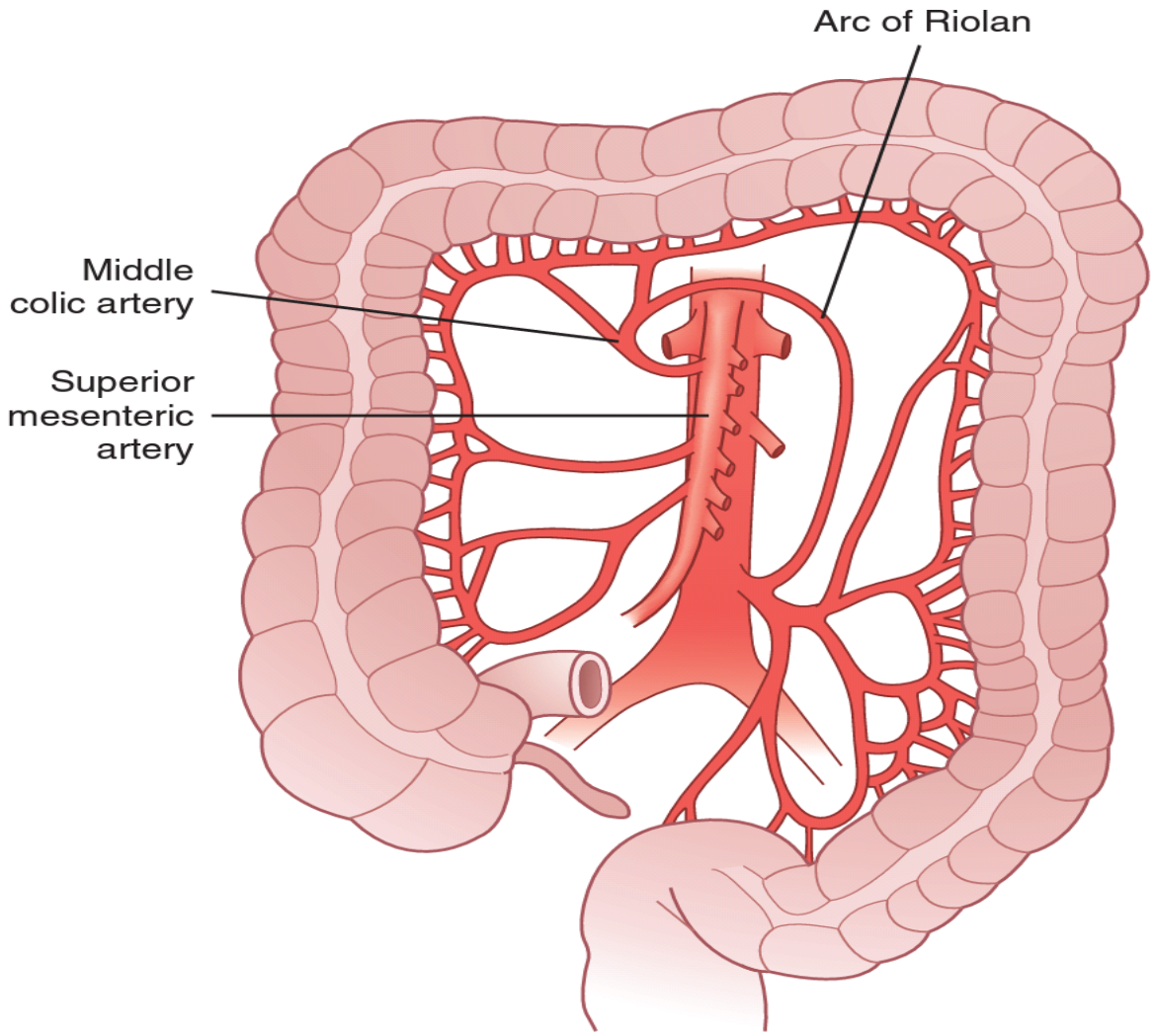
inferior branch runs to the ileocolic junction and divides into anterior and posterior caecal arteries, the appendicular artery, and an ileal branch that passes to the left in the ileal mesentery to anastomose with a terminal ileal branch of the superior mesenteric artery

2. **Right colic artery** usually arises as a common trunk with the middle colic artery, but may originate directly from the superior mesenteric artery, or from the ileocolic artery. Near the left side of the ascending colon, it divides into a descending branch, which runs down to anastomose with the superior branch of the ileocolic artery, and an ascending branch, which passes where it anastomoses with a branch of the middle colic artery.
3. **Middle colic artery** arises either separately or in common with the right colic Artery. As it approaches the colon, it usually divides into right and left branches. The right branch anastomoses with the ascending branch of the right colic artery. The left branch supplies the terminal part of the midgut and anastomoses with a branch of the left colic artery near the splenic flexure.

THE MARGINAL ARTERY OF DRUMMOND

The marginal artery is formed by the main branches and arcades arising from the ileocolic, right colic, middle colic and left colic arteries. It is most apparent in the ascending, transverse and descending colons and poorly developed in the sigmoid colon. The marginal artery in the region of the splenic

flexure may be absent or of insufficient calibre to be of functional significance. It along the inner margin of the colon and gives off short terminal branches to the bowel wall Nevertheless, it may dilate considerably when one of the main visceral arteries is compromised since it then provides a collateral arterial supply to the colon.



ARC OF RIOLAN

It runs a meandering course in the colonic mesentery between the main trunk of the middle colic artery and the ascending branch of the left colic artery. When present, this vessel is usually only prominent when there is occlusion of the superior or inferior mesenteric artery.

INFERIOR MESENTERIC ARTERY:

The artery of the hindgut is the inferior mesenteric artery which arises from the anterior or left anterolateral aspect of the aorta 3–4 cm above the aortic bifurcation, at the level of L3. It runs obliquely down to the pelvic brim. Beyond which it continues in the root of the sigmoid mesocolon as the superior rectal artery.

1. **Left colic artery** usually arises shortly after its origin, ascends within the left colic mesentery and divides into an ascending and a descending branch. The ascending branch passes upwards across the left psoas major, gonadal vessels, ureter and left kidney, and is crossed by the inferior mesenteric vein; its terminal branches anastomose with those of the left branch of the middle colic artery within the transverse mesocolon. The descending branch passes laterally and downwards, and anastomoses with branches from the ascending branch and the highest sigmoid artery to form part of the marginal artery. The arterial arcades thus formed supply the distal third of the transverse and the descending colon. The left colic artery may originate from or in common with a

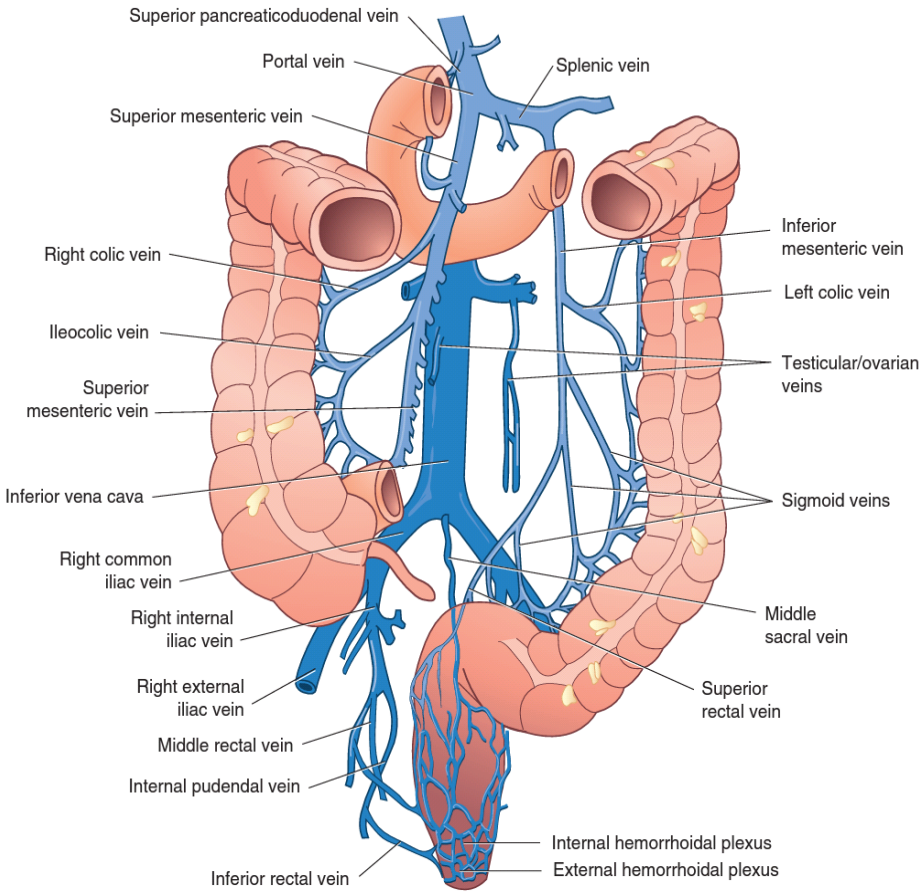
sigmoid artery. The left colic artery may itself give rise to an accessory left middle colic artery. The dominant arterial supply of the splenic flexure is usually from the left colic artery but may be from the left branch of the middle colic artery.

2. **Sigmoid arteries:** The inferior mesenteric artery gives rise to between two and five sigmoid arteries, which descend obliquely in the sigmoid mesocolon anterior to the left psoas major, ureter and gonadal vessels. They supply the distal descending colon and sigmoid colon, and anastomose superiorly with the left colic artery and inferiorly with the superior rectal artery. Unlike the arrangement in the small intestine, arterial arcades do not form until the arteries are close to the wall of the colon, when small branches arise that supply the sigmoid colon directly. A true marginal artery is less obvious in the sigmoid colon.

3. **Superior rectal artery:** The inferior mesenteric artery crosses the left common iliac vessels medial to the ureter and descends in the medial limb of the sigmoid mesocolon, straddled by the inferior hypogastric nerves on either side. As it crosses the pelvic brim, it becomes the superior rectal artery and supplies upper 2/3 of rectum. At the level of the third sacral vertebra, where the rectum begins, the artery enters the upper mesorectum in the midline and divides into two branches that descend, initially posterolaterally, and then on each side of the rectum.

Terminal branches pierce the rectal wall and anastomose with branches of the middle and inferior rectal arteries within the rectal submucosa.

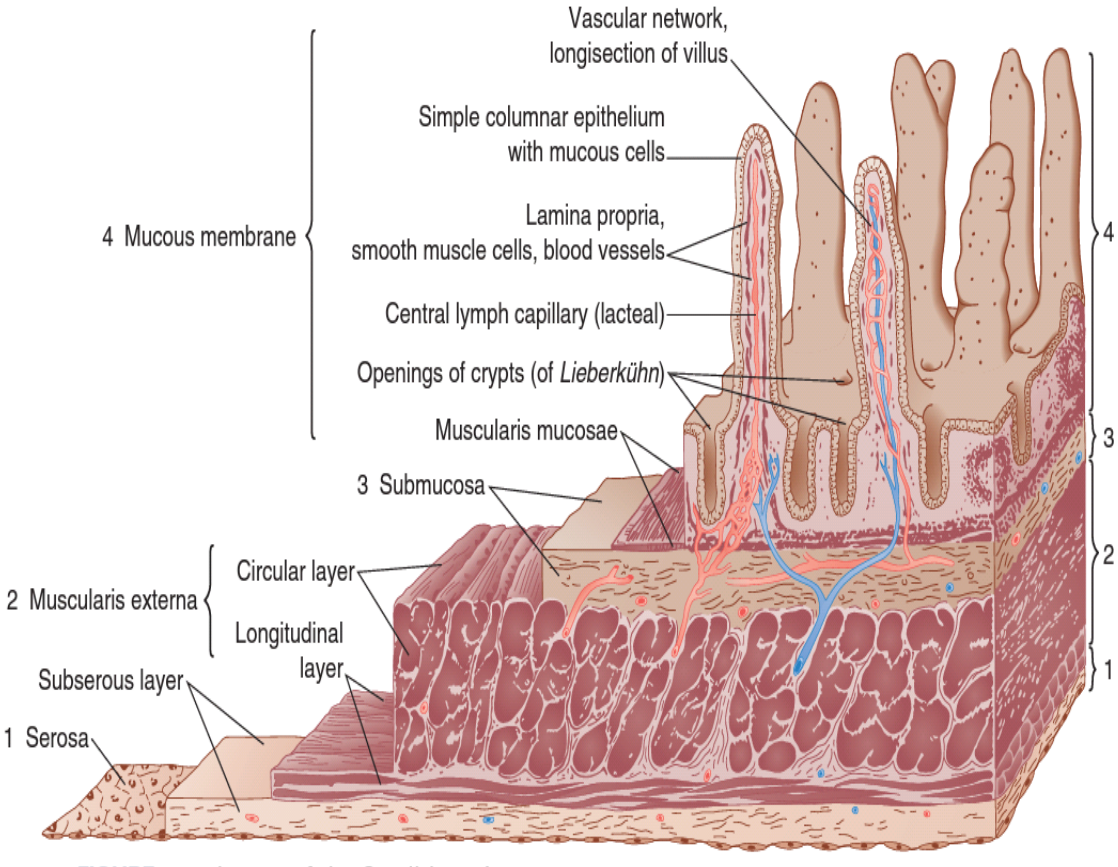
SUPERIOR MESENTERIC VEIN



The superior mesenteric vein drains the small intestine, caecum, ascending and transverse parts of the colon, and parts of the stomach and greater omentum. It is formed in the mesentery of the small bowel by the union of tributaries from the terminal ileum, caecum and vermiform appendix. It joins the splenic vein behind the neck of the pancreas in the transpyloric plane to

form the portal vein. The superior mesenteric vein receives jejunal, ileal, ileocolic, right colic, middle colic, right gastroepiploic and inferior pancreaticoduodenal veins. Although the inferior mesenteric vein usually drains into the splenic vein, it may join the superior mesenteric vein directly or its confluence with the splenic vein.

HISTOLOGY:



The wall of small intestine has four distinct layers:

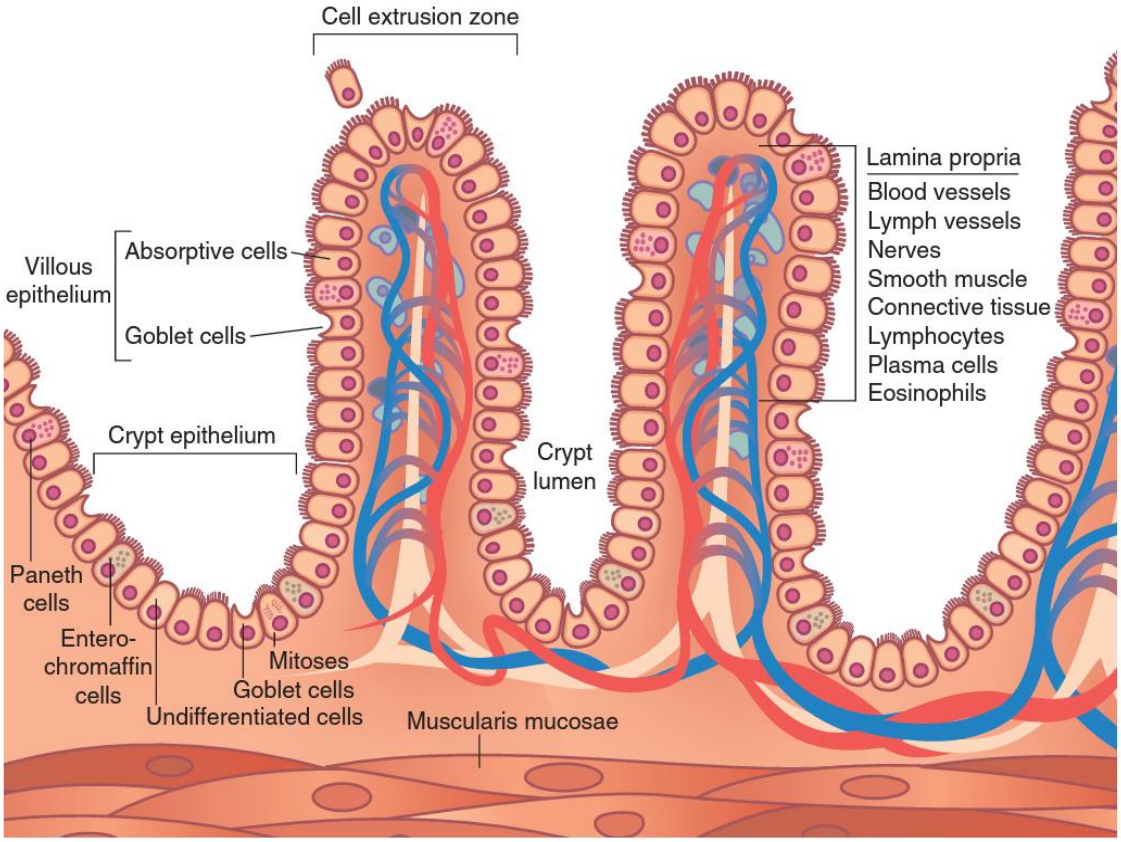
1. **Mucosa: is** organized into villi and crypts. Villi are finger like projections extending into the intestinal lumen. Epithelial cellular proliferation rapidly expanding and confined to the crypts of liberkuhn It is the innermost layer and has 3 layers
    - Epithelium helps in absorption and secretion.
    - Lamina propria has connective tissue and a variety of cells.
    - Muscularis mucosa is a thin sheet of smooth muscle cells.

Intestinal stem cells can differentiate along one of four pathways and yield enterocytes and goblet, enteroendocrine and paneth cells. The epithelium undergoes continuous renewal and one of the most dynamic tissues in the body. Because of the high cellular turn over, mucosa is highly resilient and also susceptible to certain therapeutic forms of injury namely chemotherapy and radiotherapy. Enterocytes are predominant absorptive cells and contain digestive enzymes, transporter mechanisms and microvilli which increases the absorptive surface area of the small intestine to as much as 40 folds. Goblet cells produce mucin which has defensive function. Enteroendocrine cells contain secretory granules which serve regulatory function. Located in the base if the crypts, Paneth cells contain growth factors, digestive enzymes and antimicrobial peptides. Other cells are M cells and intraepithelial lymphocytes.

2. **The sub mucosa** has heterogeneous population of cells and dense connective tissue. Cells include leukocytes and fibroblasts. It also contains submucosal (meissner's plexus)

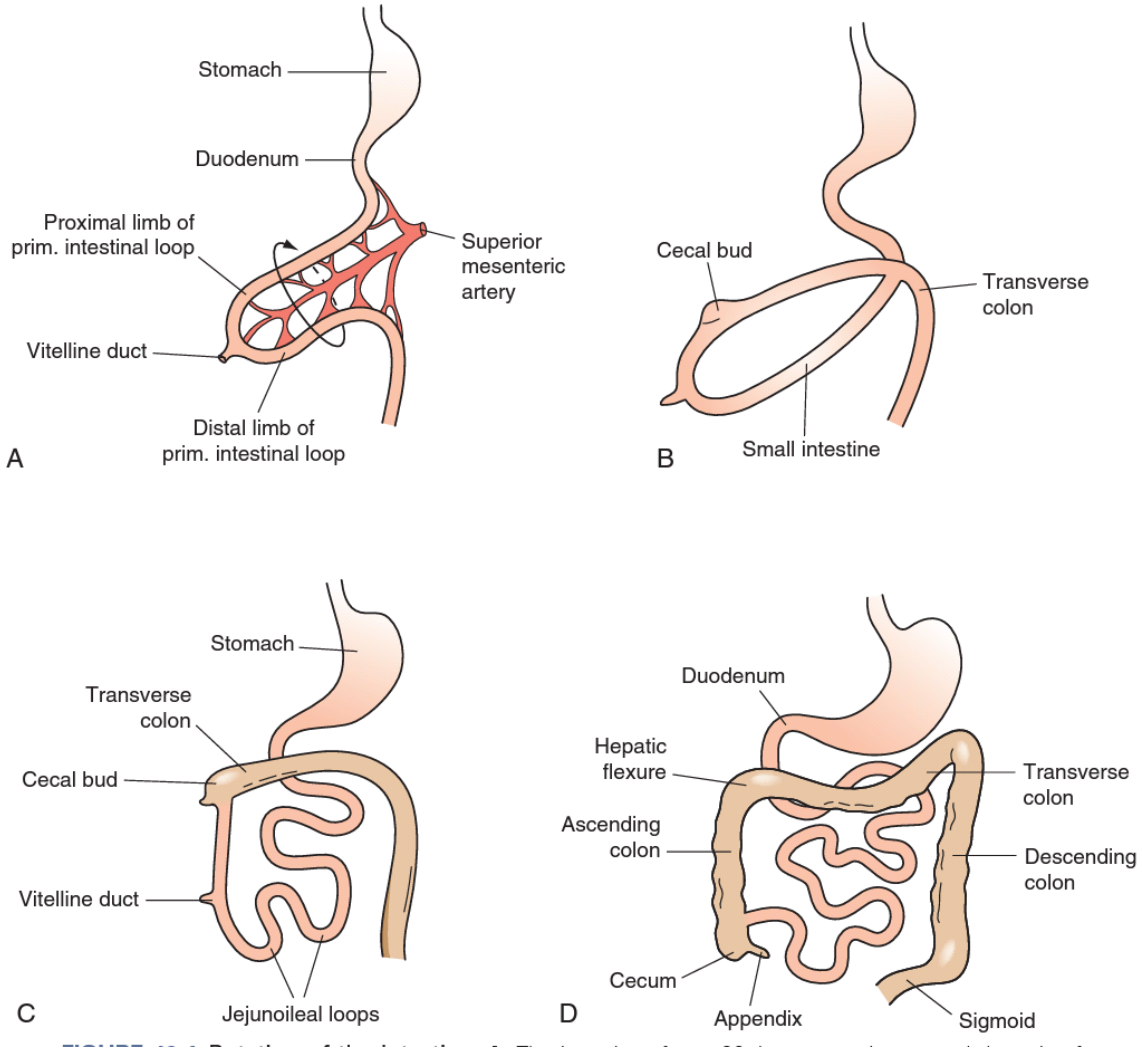
3. **The muscularis propria** has an outer longitudinal and an inner circular muscle layer which has smooth muscle fibres. Myentric or auerbach's plexus is present at the interface between these two layers.

4. The **serosa** has a single layer of smooth muscle cells which is a component of visceral epithelium.



EMBRYOLOGY OF INTESTINE:

At 4 weeks of gestational age embryonic gut tube is formed from the endoderm which is the first recognizable precursor of the small intestine. Duodenum is a foregut structure and others are derived from the midgut. The narrowed communication between the yolk sac and the intestine is the vitelline duct which gets obliterates by the end of gestation. Incomplete obliteration of the vitellointestinal duct is associated with meckel's diverticulum.



Visceral peritoneum is formed from the portion of mesoderm attached with the endoderm. Parietal peritoneum is formed from the portion of mesoderm attached with the ectoderm. Coelomic cavity is formed from this mesodermal division which gives rise to peritoneal cavity.

Extracoelomic herniation of the developing bowel happens approximately at fifth week of gestation. This is because the abdominal cavity cannot accommodate the enormously lengthened bowel. Lengthening of the bowel continues in the subsequent weeks. And returns back to the abdominal cavity by 10^{th} week of gestation and as a result duodenum becomes a retroperitoneal structure.

During this bowel herniation and retraction the bowel totally undergoes a 270 degree counter clock wise rotation around the superior mesenteric axis relative to the posterior abdominal cavity. As a result caecum is present in the right lower quadrant and the duodenaljejunal junction to the left of midline.

EPIDEMIOLOGY

Incidence is low, estimated at 0.09–0.2% of all acute surgical admissions. Although the entity is an uncommon cause of abdominal pain, diligence is always required because if untreated, mortality has consistently been reported in the range of 70-80%. Early diagnosis and timely surgical intervention are the cornerstones of modern treatment and are essential to reduce the high mortality associated with this entity

HISTORY:

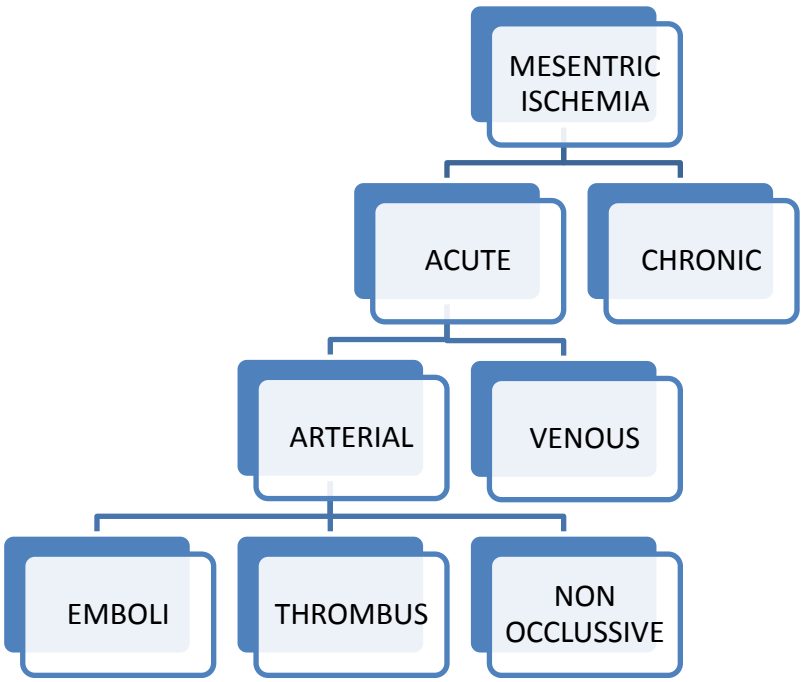
The first description of mesenteric thrombosis is attributed to Antonio Beniviene of Florence in the fifteenth century, but Tiedenman's clinical case report in 1843 first stimulated interest in this problem. The first successful bowel resection for intestinal infarction was reported by Elliott in 1895. In 1906 Delbet suggested the possibility of revascularization for SMA occlusion. Although Ryvlyn in 1943 and Blinov in 1950 described patients in whom SMA embolectomy was unsuccessfully attempted, Klass is generally credited with establishing the feasibility of this operation. In 1951 he described two patients who underwent successful embolectomy but died of cardiac causes postoperatively. One year later Stewart performed the first SMA embolectomy with survival. Successful operative approaches to acute SMA thrombosis as well as chronic mesenteric ischemia were reported in the 1950s. Ende in 1958 first described nonocclusive mesenteric ischemia, and during the 1960s various attempts to treat this condition using local and regional anaesthetic blocks as well as systemic and intra-arterial vasodilator were reported.

PATIENT POPULATION

An increasing incidence has been attributed to the aging of the population, since AMI occurs predominantly, but not exclusively, in geriatric patients, especially those with significant cardiovascular and systemic disorders. Similarly, the widespread use of coronary and surgical intensive care units and other extraordinary means of cardiopulmonary support has salvaged

patients who previously died rapidly of cardiovascular conditions, only to have them go on to develop AMI as a later consequence of their primary disease. A decline in the incidence of nonocclusive ischemia has been noted, possibly due to the increasing use of systemic vasodilators, such as the calcium channel blocking agents and nitrates, in coronary intensive care units.

TYPES:



Acute mesenteric ischemia is much more common than chronic, and ischemia of arterial origin is much more frequent than venous disease. In the chronic forms, the viability of the bowel is not compromised, but the blood flow is inadequate to support the functional demands of the intestine, while with the acute forms, intestinal viability is threatened. Atherosclerotic narrowing or occlusion of the mesenteric arteries, producing intestinal angina,

and gradually evolving mesenteric venous thrombosis are the common forms of chronic ischemia.

SPECIFIC TYPES OF ACUTE MESENTERIC ISCHEMIA:

ARTERIAL CAUSES:

1. SUPERIOR MESENTERIC ARTERY EMBOLI:

It accounts for 40–50% of episodes of acute mesenteric ischemia. The emboli usually originate from a left atrial or ventricular mural thrombus. The thrombus embolizes after being dislodged or fragmented during a period of an arrhythmia. Many patients with SMAE have a history of previous peripheral artery embolism, and approximately 20% are associated with concurrent emboli to another arterial bed including the spleen, or kidney.

Emboli to the superior mesenteric artery tend to lodge at points of normal anatomical narrowing. The majority of emboli lodge 3 to 10 cm distal to the origin of the SMA, thus classically sparing the proximal jejunum and colon. In 10–15% of patients the emboli lodge peripherally in branches of the SMA, or in the SMA itself distal to the origin of the ileocolic artery. The embolus may completely occlude the arterial lumen, but often only partially occludes the vessel. Experimental studies suggest that initially the collateral circulation is adequate to maintain intestinal viability following most acute SMA occlusions, but after a period of partial occlusion and diminution of blood flow distal to the embolus, vasoconstriction develops in arteries both proximal and distal to the embolus. This vasoconstriction can sufficiently impair the

collateral blood flow to the SMA and its branches distal to the embolus to cause or exacerbate an ischemic injury.

The onset of symptoms is usually dramatic as a result of the poorly developed collateral circulation, and it is characterized by the abrupt onset of severe abdominal pain associated with diarrhea, which may become bloody. Frequently, the diagnosis of SMA embolism can be made intraoperatively based on the distribution of ischemic bowel. In most SMA emboli the proximal jejunum is spared, whereas the rest of the small bowel is ischemic or infracted.

2. **NONOCCLUSIVE MESENTERIC ISCHEMIA:**

It causes 20–30% of episodes of AMI and is thought to result from splanchnic vasoconstriction initiated by period of decreased cardiac output associated with hypotension caused by

- cardiogenic shock, (Arrhythmias, acute myocardial infarction, congestive heart failure, aortic insufficiency)
- hypovolemic shock
- hepatic diseases, renal diseases, especially in patients requiring haemodialysis,
- major cardiac or intraabdominal operations
- vasoactive medications
- trauma and are receiving enteral nutrition in intensive care units

The vasoconstriction may persist even after the precipitating cause has been eliminated or corrected. Frequently, a more immediate precipitating cause is present, although the consequent intestinal ischemia may not become manifest until hours or days later.

3. SUPERIOR MESENTERIC ARTERY THROMBOSIS:

Patients with this condition can tolerate most major visceral artery obstruction because the slow progressive nature of atherosclerosis allows the development of important collaterals. Bowel ischemia or infarction ensues when the last remaining visceral artery or an important collateral artery occludes. The extent of bowel ischemia or infarction is typically greater than that with embolism, extending from the duodenum to the transverse colon. Perioperative mortality ranges from 70% to 100%, in part because of the delay in diagnosis, the extensive nature of the bowel ischemia-infarction, and the need for more complex surgical revascularization.

It occurs at areas of severe atherosclerotic narrowing, most often at the origin of the SMA. The acute ischemic episode is commonly superimposed on chronic mesenteric ischemia, hence approximately 20–50% of these patients have a history of abdominal pain with or without evidence of malabsorption and weight loss during the weeks to months preceding the acute episode. Additionally, most patients with SMAT have severe and diffuse atherosclerosis with a prior history of coronary, cerebrovascular, or peripheral arterial

insufficiency. SMA thrombosis may also occur due to vasculitis, mesenteric dissection, or a mycotic aneurysm.

4. VENOUS CAUSES:

This represents less than 5% of all cases of AMI. The thrombosis is attributed to a combination of Virchow's triad, i.e., stagnated blood flow, hypercoagulability, and vascular inflammation. Mesenteric venous thrombosis is usually segmental, with edema and haemorrhage of the bowel wall and focal sloughing of the mucosa. Thrombi usually originate in the venous arcades and propagate to involve the arcuate channels. Hemorrhagic infarctions occur when the intramural vessels are occluded. The thrombus is usually palpable in the superior mesenteric vein. Involvement of the inferior mesenteric vein and large bowel is uncommon. The transition from normal to ischemic intestine is more gradual with venous embolism than with arterial embolism or thrombosis

The additional components altering blood flow include portal hypertension, pancreatitis, inflammatory bowel disease, sepsis, and trauma. In these situations, the consequences of bowel edema and increased vascular resistance secondary to venous thrombosis result in reduced arterial blood flow, leading to bowel ischemia.

Hypercoagulable states

1. Pregnancy
2. Polycythemia Vera
3. Thrombocytosis
4. Neoplasm
5. Antithrombin III deficiency
6. Protein C deficiency
7. Protein S deficiency
8. Oral contraceptive use
9. Peripheral deep venous thrombosis

Inflammation

1. Inflammatory bowel disease
2. Pelvic or intraabdominal abscess
3. Pancreatitis
4. Peritonitis (e.g., appendicitis, perforated viscus)
5. Diverticular disease

Portal hypertension

1. Congestive splenomegaly
2. Cirrhosis
3. Following sclerotherapy of oesophageal varices

Postoperative state or trauma

1. Blunt abdominal trauma
2. Splenectomy and other postoperative states

Other

1. Decompression sickness
2. Idiopatic (around 20%)

PATHOPHYSIOLOGY OF MESENTERIC VASOCONSTRICTION:

The intestines are protected from ischemia by their abundant collateral circulation. Communications between the celiac, superior mesenteric, and inferior mesenteric vascular beds are numerous. Collateral flow around occlusions of smaller arterial branches in the mesentery of the small intestine and right colon is made possible by the branching primary, secondary, and tertiary arcades. Within the bowel wall there is a network of communicating submucosal vessels that can maintain the viability of short segments of intestine where the extramural arterial supply has been compromised. Collateral pathways open immediately upon occlusion of a major vessel in response to arterial hypotension distal to the obstruction. Increased blood flow through the collateral pathways continues as long as the pressure in the vascular bed distal to the obstruction remains below systemic pressure. If, however, vasoconstriction develops in the distal arterial bed, the arterial pressure rises due to increased resistance, which ultimately impairs collateral

flow to the dependent segment. Despite adequate collateral vasculature in most cases, acute interruption or diminution of blood flow in the mesenteric circulation caused by emboli or hypotension results in intestinal ischemia secondary to persistent vasospasm. A decrease in SMA flow initially produces local mesenteric vascular responses that tend to maintain intestinal blood flow, but if the diminished flow is prolonged, active vasoconstriction develops, which may persist even after the primary cause of decreased flow is corrected.

In NOMI it had been presumed that the bowel injury occurs during the period of diminished cardiac output or hypotension, and that with correction of these cardiovascular problems the mesenteric blood flow returns to normal. This simplistic concept is contradicted by operative findings of persistent bowel ischemia when no arterial or venous obstruction is found and cardiac function has been optimized. It is known that in patients with NOMI the onset of abdominal signs and symptoms caused by intestinal ischemia may actually begin after the correction of the primary systemic problem. This paradox can be explained by the experimental observations that an episode of low mesenteric flow, as short as 2 hours in duration, can produce mesenteric ischemia as a result of vasoconstriction, and that the vasoconstriction and ischemia may continue even after the correction of the initiating problem. The end result of an ischemic episode is dependent on many factors. Thus the extent of intestinal injury is a function of the

- state of the systemic circulation;
- the degree of functional or anatomical compromise;

- the number and calibre of vessels affected;
- the response of the vascular bed to diminished perfusion;
- the nature and capacity of the collateral circulation to supply the needs of the dependent segment of bowel;
- the duration of the insult
- the metabolic needs of the dependent segment as dictated by its function and bacterial population.

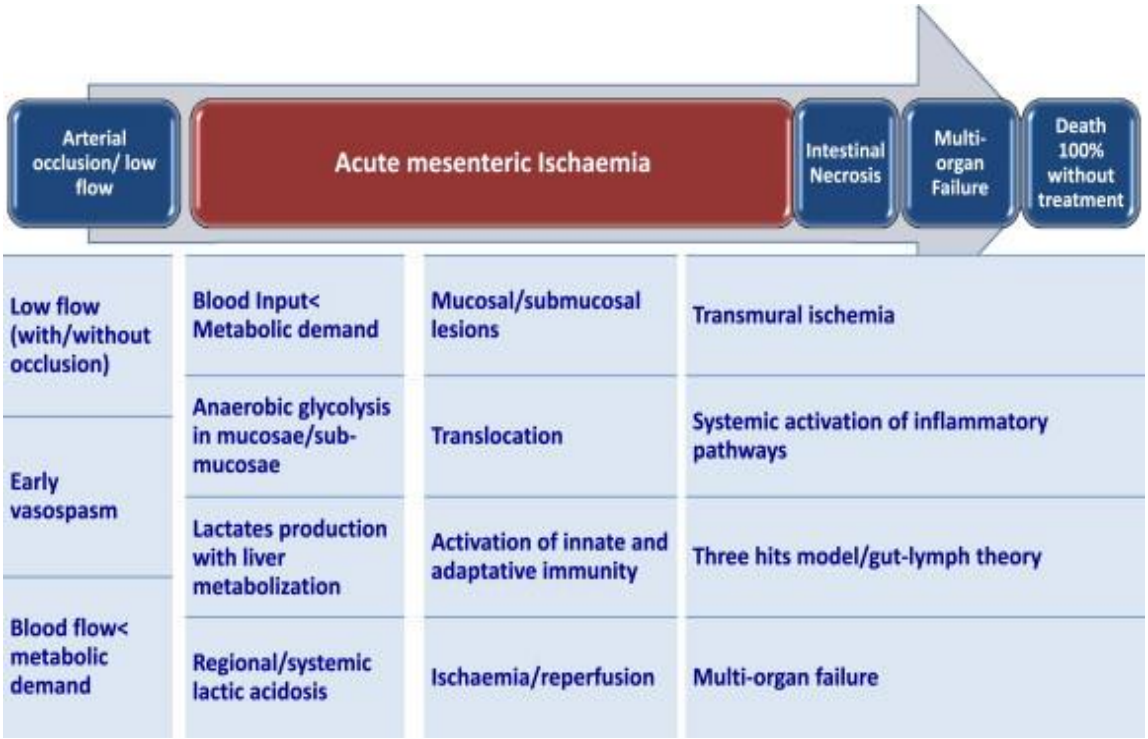
Because vasospasm may persist even after the initial cause of the ischemia is corrected, bowel injury continues unless the vasospasm is relieved. An aggressive radiological and surgical approach to these diseases targets both the cause and the persistent vasospasm

FACTORS INFLUENCING GUT MICROCIRCULATION:

The splanchnic circulation receives approximately 25% of the resting and 35% of the postprandial cardiac output. 70% of the mesenteric blood flow is directed to the mucosal and submucosal layers of the bowel, with the remainder supplying the muscularis and serosal layers. The physiologic characteristics of splanchnic blood flow are complex and incompletely understood. Multiple major elements interact to provide the intestinal tract with an appropriate share of the blood supply, including the intrinsic (metabolic and myogenic) and the extrinsic (neural and humoral) regulatory systems.

Pressure-flow auto regulation, reactive hyperaemia, and hypoxic vasodilation are considered intrinsic controls and are responsible for

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



The extrinsic **neural component** of splanchnic circulatory regulation comprises the α-activated vasoconstrictor fibers. Intense activation of vasoconstrictor fibers through α-adrenergic stimulation results in vasoconstriction of small vessels and a decrease in mesenteric blood flow.

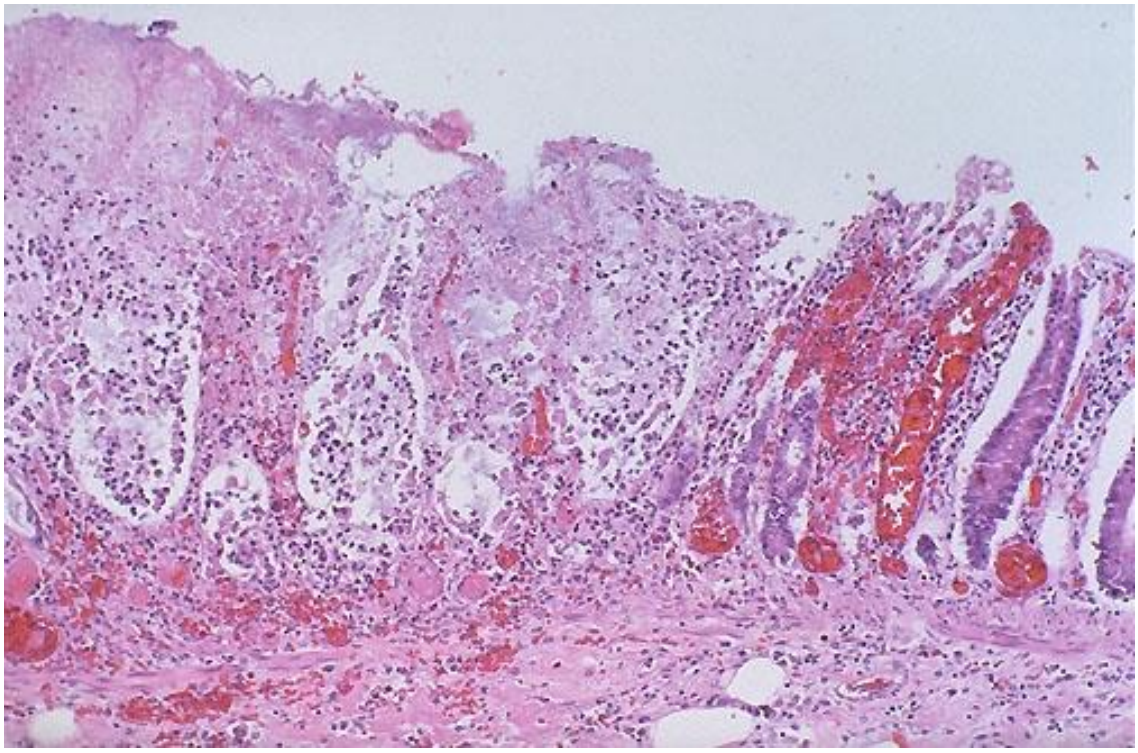
After periods of prolonged α-adrenergic vasoconstriction, blood flow increases, presumably through β-adrenergic stimulation, which has a protective response. After cessation of α-adrenergic stimulation, brief hyperaemia makes the response triphasic. Although various types of neural stimulation (e.g., vagal, cholinergic, histaminergic, and sympathetic) can affect the gut, the adrenergic limb of the autonomic nervous system is the predominant and possibly the sole neural influence on splanchnic circulation.

Numerous endogenous and exogenous **humoral factors** are capable of affecting the splanchnic circulation. Norepinephrine and high levels of epinephrine produce intense vasoconstriction through the stimulation of adrenergic receptors. Other pharmacologic compounds that decrease splanchnic blood flow include vasopressin, phenylephrine, and digoxin. Low-dose dopamine causes splanchnic vasodilation, whereas higher doses lead to vasoconstriction by stimulating α-adrenergic receptors. Papaverine, adenosine, dobutamine, fenoldopam mesylate, and sodium nitroprusside are exogenous agents that increase mesenteric blood flow. In addition, various naturally occurring agents can serve as splanchnic vasodilators, including acetylcholine, histamine, nitric oxide, leukotrienes, thromboxane analogues, glucagon, and an assortment of gastrointestinal hormones.

CELLULAR CHANGES:

Intestinal vascular insufficiency leads to hypoxia, first with mucosal and submucosal consequences. The hypoperfusion of the intestinal mucosa is

responsible for an early hypoxic cellular desquamation of the intestinal villi. The early major actors are neutrophils that adhere and migrate to the ischemic site to ensure the removal of tissue debris during necrosis. Mucosal and submucosal cells switch to anaerobic glycolysis with local production of lactate which is initially fully metabolized by the liver.



The increase in intracellular acidosis blocks anaerobic metabolism and the membrane pumps of ionic and acid-base regulation. This leads to a profound alteration of cellular homeostasis and ultimately cell death by apoptosis. Initially, there is dissociation between high porto-mesenteric blood lactate levels and normal peripheral blood lactate levels due to the active liver metabolism. Systemic lactic acidosis is, therefore, a late phenomenon, which often indicates intestinal necrosis and the onset of a multi-visceral failure.

Associated endothelial lesions can lead to platelet, pro- and anti-thrombotic agent (protein C, S, and anti-thrombin) consumption, which causes hemorrhagic syndrome.

Furthermore, the intestinal neuro-hormonal regulation of associated with the activation of the renin-angiotensin-aldosterone system, which maintains the mucosal oxygen extraction rate. This induces a reflex splanchnic arterial vasospasm, irrespective of the initial vascular mechanism, that may prolong and worsen ischemia despite therapeutic revascularization.

INFLAMMATORY RESPONSE:

These mucosal alterations discussed above leads to disruption of the epithelial barrier and interactions among microorganisms, bacterial antigens, endotoxins of the intestinal lumen with the mucosal and submucosal immune system. The stimulation of innate immunity will result in local and then systemic inflammatory pathways activation such as TLR, NF-κB or TNF. Through the bloodstream, bacteria, endotoxins, cells degradation products and activated immune cells translocate and promote SIRS (systemic inflammatory response syndrome). Cytokines, chemokines, cellular and bacterial debris can also reach the pulmonary circulation from the lymphatic circulation and thus cause acute respiratory distress syndromes (ARDS). The absence of a rapid recovery of this perfusion defect leads to irreversible transmural necrosis and

then to peritonitis. Without intestinal resection, the SIRS rapidly evolves to multiple organ dysfunction syndrome and death.

THEORIES OF INFLAMMATION

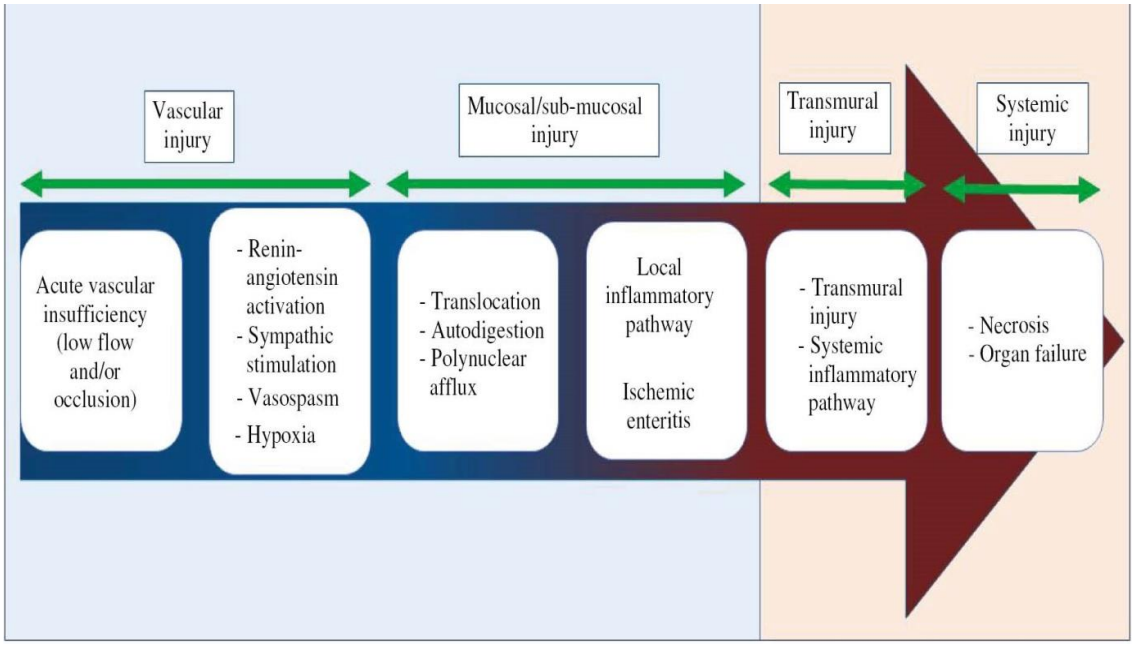
1. In **gut origin of sepsis** model, the gut was considered to be the engine driving multi-organ failure Aside from its barrier function, the gut contains growth factors, adenosine and hormones, which are potential mediators of the modulation of intestinal inflammation and repair, due to their roles in cellular proliferation, differentiation, migration, apoptosis and autophagy. Physiologically, the gut could initiate and propagate sepsis due to the ability of bacteria, endotoxins, and other antigens to translocate, along with the production of pro-inflammatory cytokines and toxins.

2. In the "**gut-lymph**" theory, bacteria, cellular components, immune cells, cytokines and chemokines generated by the injured gut travel via the lymphatic's to reach the pulmonary circulation, activating alveolar macrophages and contributing to acute lung injury, acute respiratory distress syndrome and multi-organ failure

3. The "**three hits model**" combines the above two models and also adds the phenomenon of reperfusion injury.

INTESTINAL AUTO DIGESTION

It describes the effect of pancreatic enzymes on the intestinal barrier altered by ischemia. Self-digestion contributes to the worsening of ischemic lesions and the development of the related systemic inflammatory response. Degradation products of pancreatic enzymes, residues of bacterial products pass through the lymphatic, haematogenous or peritoneal barrier and are likely to induce not only a loco-regional but also a systemic reaction. In animal models, inhibition of these enzymes results in a decrease in intra-parietal micro-bleeding, systemic inflammatory response, and even mortality in some studies. The action of these enzymes would involve degradation of inter-enterocytic tight junction's proteins such as E-cadherin. Moreover, these enzymes also induce cleavage of the inactive prometalloproteinases into active metalloproteinases.



REPERFUSION INJURY:

The systemic consequences of bowel ischemia and necrosis are lethal in most patients in the absence of curative treatment including revascularization. However, reoxygenation of the digestive mucosa can also paradoxically worsen epithelial and vascular lesions. Brief periods of mesenteric ischemia lead to an increase in micro vascular permeability, whereas prolonged ischemia leads to disruption of the intestinal mucosal barrier, primarily through the actions of reactive oxygen metabolites and polymorphonuclear neutrophils.

The role of oxygen free radicals in reperfusion injury is demonstrated by the reduction of tissue damage in the presence of antioxidants, xanthine oxidase inhibitors, and free-radical scavenging substances. Polymorphonuclear leukocytes contain enzymes that reduce molecular oxygen to superoxide anions and produce hypochloric acid, providing an additional source of reactive oxygen metabolites. Epithelial cells may produce xanthine oxidase–derived oxidants and initiate the production of proinflammatory agents that attract polymorphonuclear leukocytes. In addition, phospholipase A_2 is activated during reperfusion, increasing the formation of cytotoxic lysophospholipids within the ischemic tissue and up-regulating the production of prostaglandins and leukotrienes. Further understanding of the role of reperfusion injury may present opportunities for protective pharmacologic therapies with agents such as captopril (ACE inhibitor) and carvedilol (β-adrenoreceptor blocking agent and a free-radical scavenger, has been demonstrated to have an antishock and endothelial-protective effect in a rat splanchnic ischemia reperfusion model).

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

CLINICAL PRESENTATION:

Early identification of acute mesenteric ischemia requires a high index of suspicion by the clinician in those patients who have significiant risk factors associated with this disease. **Sudden severe abdominal pain** accompanied by rapid and often forceful bowel evacuation especially with **minimal or no abdominal signs** strongly suggests an acute arterial occlusion in the mesenteric circulation. Unexplained abdominal distension or gastrointestinal bleeding may sometimes be the only features of acute intestinal ischemia. Distension, while absent early, is usually a sign of impending intestinal infarction. The stool contains occult blood in 75% of patients, and this bleeding may precede any other symptom of ischemia. Right-sided abdominal pain associated with the passage of maroon or bright red blood in the stool, though characteristic of colonic ischemia, also suggests the diagnosis of acute mesenteric ischemia. Although there are no abdominal findings early in the course of intestinal

ischemia, as infarction develops, increasing tenderness, rebound tenderness, and muscle guarding develope which reflects the progressive loss of intestinal viability and the progression of transmural gangrene. Significant abdominal findings strongly indicate the presence of infarcted bowel. Nausea, vomiting, hematochezia, hematemesis, massive abdominal distension, back pain, and shock are other late signs often indication compromise of bowel viability. Bloody ascites and large fluid losses with third-spacing may occur, leading to dehydration and hypotension, causing further worsening of the mesenteric ischemia. The final common pathway of all the specific causes of mesenteric ischemia is bowel infarction. When infarction occurs, the patient has peritoneal signs, hemodynamic instability, and signs of sepsis with multi-organ failure

With **SMA embolism**, the onset of symptoms is usually dramatic because of lack of collateral circulation, and it manifests as severe and unrelenting abdominal pain, nausea, vomiting, and urgent bowel evacuation.

Patients with **SMA thrombosis** frequently report a prodromal symptom complex of postprandial pain, nausea, and weight loss associated with chronic intestinal insufficiency. Patients with a sub acute onset tend to seek medical care much later than those with arterial emboli. However, when ischemia from mesenteric thrombosis becomes acute, patients present similarly to those who have acute SMA embolism.

NOMI occurs most frequently in elderly, critically ill patients and in those with an acute hemodynamic insult. Such patients are often intubated and sedated and, therefore, are unable to alert the clinician to their symptoms. In

these circumstances, the intestinal ischemia may not become clinically evident until hours or days after the initial hemodynamic insult. These patients frequently experience unexplained worsening in their clinical condition or a failure to thrive or to follow their anticipated recovery course.

Except in the most fulminant cases, patients with **MVT** typically present late (i.e., 1-2 weeks after onset), complaining of diffuse, nonspecific abdominal pain associated with anorexia and diarrhoea. If the pain is localized, it is most often in the lower quadrants. Compared with arterial thrombosis, MVT generates fewer prodromal symptoms with eating or postprandial pain.

LABORATORY FINDINGS

Improving the prognosis of AMI requires the discovery of early, sensitive and specific diagnostic biomarkers. In the last decade, some potential biomarkers have emerged from the literature. Some of these markers have been studied with particular interest, because of their higher presumed enterocyte specificity.

Common laboratory findings

Variations in common biological blood parameters (base deficit, lactate dehydrogenase, aspartate aminotransferase, creatinine phosphokinase, alkaline phosphatase, phosphate, and amylase) have frequently been observed

More than 90% of patients will have an abnormally **elevated leukocyte count**.

The second most commonly encountered abnormal finding is metabolic acidosis with **elevated lactate level**, which occurred in 88%. Patients may present with lactic acidosis due to dehydration and decreased oral intake. Thus, differentiation of early ischemia versus irreversible bowel injury based upon the lactate level alone is not reliable unless accompanied by other clinical evidence. Elevated serum lactate levels >2 mmol/L was associated in irreversible intestinal ischemia

Regarding haematological parameters, attention has been given to platelet indices, particularly platelets volume, but also to the various combinations of neutrophils, lymphocytes and platelets ratios high platelet volume would be a poor prognosis indicator. As a whole, the use of such indices appears to be difficult to translate into clinical practice.

BIOLOGICAL MARKERS OF THROMBOSIS:

D-dimer, an enzymatic degradation product of fibrin, has been found to be the most consistent highly sensitive early marker, but has low specificity. Moreover, D-dimers are usually increased either in arterial or venous occlusive forms although they remain in the normal range in NOAM.

BIOLOGICAL MARKERS OF HYPOXIA AND OXIDATIVE STRESS

L-lactate is a ubiquitous product of glycolysis in the context of anaerobia. L-lactate elevation in plasma could not differentiate intestinal ischemia from the other etiologies of abdominal emergencies or intensive care diseases. Its

elevation better reflects the late stage of the disease, with extensive transmural necrosis, anaerobic metabolism due to systemic hypoperfusion.

Glutathione S-transferases are enzymes involved in the detoxification of a wide variety of endo- and xeno-biotics, conjugating them to glutathione. These enzymes are sensitive biomarkers of cytolysis, with a very short half-life, and are currently used in the diagnosis of hepatic cytolysis. A-GST showed a significant increase in 50% of AMI, as compared with 12 other types of unclear acute abdominal pain suspected as being ischemic, with a negative predictive value of 100%. However it also increases in non-specific hypotensive patients with multiple organ failures.

During acute ischemic conditions, albumin's metal-binding capacity is reduced, leading to the appearance of a metabolic variant known as **ischemia-modified albumin**. It is a sensitive but non-specific marker of myocardial and muscle ischemia, pulmonary embolism and stroke. IMA is usually measured in the plasma or serum by ELISA or using a spectrophotometric method that measures altered cobalt-human serum albumin binding

BIOMARKERS OF INFLAMMATION:

The **C-reactive protein** is commonly increased in AMI. Acute inflammatory mediators such as interleukin-2 and 6 and tumor necrosis factor (TNF) are non-specific of intestinal injury, although interleukin-6 (IL-6) has been proposed

BIOMARKER OF INFECTION:

Procalcitonin is a precursor of the calcitonin is currently used in clinical practice for the differential diagnosis of infection of bacterial origin. PCT is usually elevated during sepsis, in specific bacterial infections, and in various types of ischemia, but lacks specificity.

GUT BARRIER DYSFUNCTION BIOMARKERS:

D-lactate, the second stereoisomer of lactate, is a by-product of bacterial fermentation, with only a small amount being produced by human cells. It can be found in the circulation after ischemic injury, increased intestinal permeability, or bacterial overgrowth. AMI is associated with growth of the resident bacterial microbiota that releases D-lactate into portal and systemic circulations. The analysis of D-lactate concentration requires strict pre-analytical conditions, which are comparable to the one needed for the assessment of L-lactate concentrate-ion. D-lactate is usually assayed using an enzymatic UV spectrophotometric method on deproteinized plasma. As the sensitivity of this marker in either plasma or serum ranged between 67% and 90%, whereas the specificity reached 87%.

BIOMARKERS OF VILLI INJURY:

Fatty acid-binding proteins are cytosolic proteins involved in the uptake and intracellular transport of fatty acids. The mature enterocyte expresses three isoforms:

- intestinal FABP (I-FABP),
- ileal bile acid-binding protein (I-BABP) and
- liver FABP (L-FABP).

I-FABP is specific of the ileum. I-FABP is a 15-kDa soluble protein expressed by enterocytes located at the tips of the intestinal mucosal villi, the anatomical region that is first affected by ischemic injuries. In physiological conditions, I-FABP is low in peripheral circulation and is cleared via the urine. After mucosal tissue injury, and especially enterocyte necrosis, the protein is quickly released into the bloodstream. In clinical settings, I-FABP concentrations measured in peritoneal fluid, plasma and urine. In peritoneal fluid, whose presence reveals late and severe disease, high levels of I-FABP were detected in patients with intestinal diseases .Urine I-FABP could be a biomarker with high specificity and sensitivity. Serum I-FABP has sensitivity of 80% and a pooled specificity of 85%, and an area under the ROC curve of 0.86 in the diagnosis of AMI.

BIOMARKER OF ENTEROCYTE MASS AND INTESTINAL FAILURE:

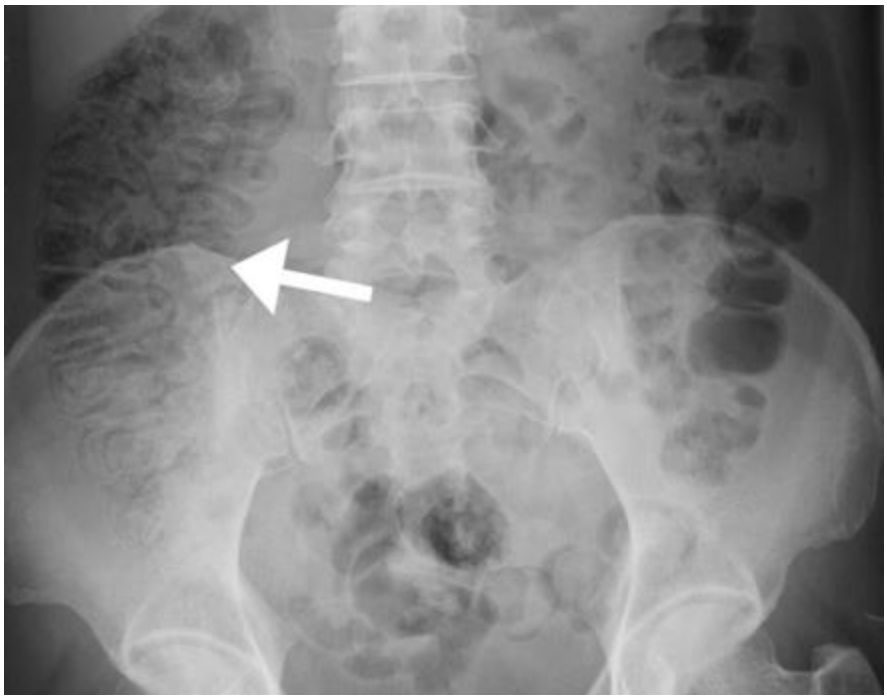
Citrulline is a non-proteinogenic amino acid synthesized from glutamine by small bowel enterocytes. This amino acid is a precursor of nitrogen oxide and participates in the transformation of ammonia into urea, and in the synthesis of

arginine. Its plasmatic concentration depends on gut synthesis and renal elimination, decreasing in short bowel conditions and thus known as a functional marker of enterocyte mass, correlated with remnant small bowel length and home parenteral nutrition dependence.

Citrulline is usually measured in plasma or serum, using ELISA methods, high-performance liquid chromatography or mass spectrometry. Critically ill patients with shock may have an AMI resulting in a reduction of enterocyte mass and related citrulline synthesis, leading to low plasma citrulline concentrations as compared to those in patients with other acute abdominal conditions. Acute renal failure induces high plasmatic citrulline concentrations by decreasing renal clearance and citrulline transformation into arginine, which may complicate the interpretation of the results in severe patients with multiorgan failures. Moreover, post prandial samples are associated with a 10%–20% decrease in blood concentration .These results suggest that citrulline is probably more promising as a prognostic than a diagnostic marker of AMI. Moreover, the interpretation of a ratio between plasmatic citrulline and creatinine could help minimize the effect of acute renal failure leading to possible false-negative results.

DIAGNOSTIC STUDIES:

X-RAY:



A radiograph is usually the initial test ordered in patients with acute abdominal pain but has a limited role in the diagnosis of mesenteric ischemia, especially in the early setting. A negative radiograph does not exclude mesenteric ischemia. Late in the course of the disease, formless loops of small intestine or small intestinal “thumb printing” can suggest the diagnosis of AMI. Ischemic intestinal perforation manifests as free intraperitoneal air.

BARIUM STUDIES:

It has no place in the diagnosis of AMI. The introduction of barium and air may increase intraluminal pressure, causing reduced perfusion to the bowel wall, translocation of bacteria, and, potentially, perforation. In addition, the presence of barium may compromise subsequent diagnostic tests, such as computed tomography (CT) and angiography

ULTRASOUND

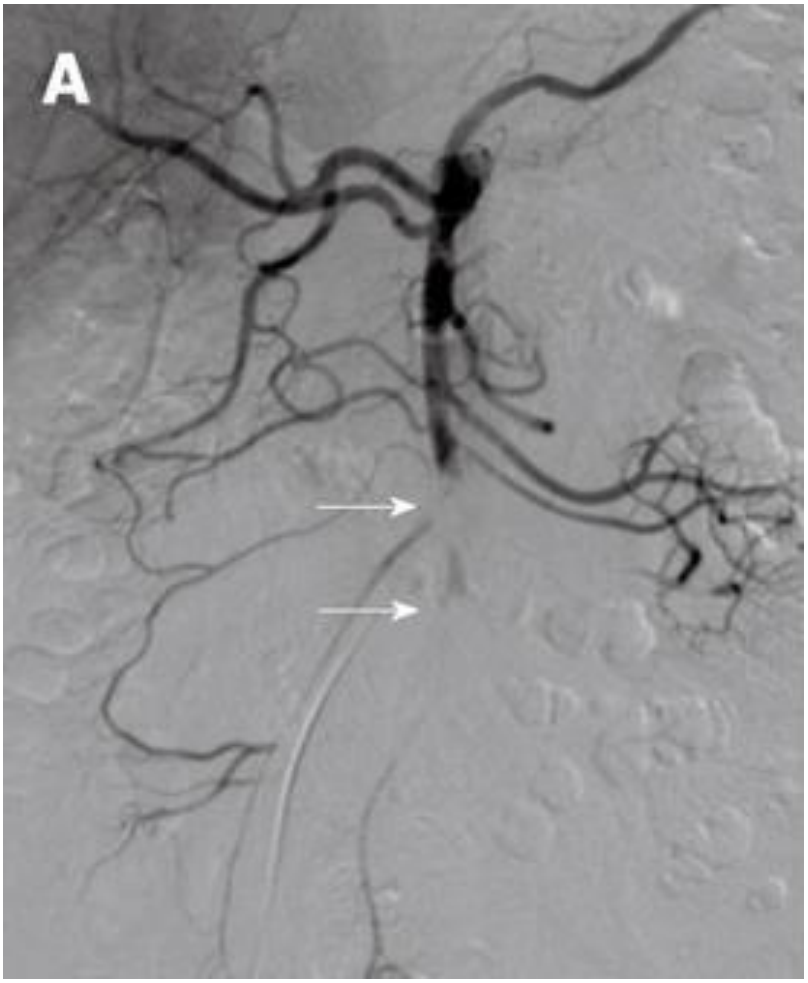
Ultrasound has little role in the diagnosis of AMI but forms a basic investigation to rule out other causes of acute abdomen. The proximal arteries only can be visualised in skilled hands, and this is often challenging due to overlying bowel gas (e.g., ileus) or patient body habitus. The finding of a proximal vessel stenosis/occlusion may be misleading as this is a common finding in the asymptomatic elderly population. Intra-operative ultrasound may be utilised in assessing the response of treatment and bowel viability. Ultrasound may have a role in the non-acute presentation, for instance, in identifying portal vein or SMV thrombus.

ANGIOGRAPHY:

Historically, angiography was limited to identifying arterial occlusions by embolus or thrombosis. Currently, selective angiography is the mainstay of diagnosis and initial treatment of both occlusive and non-occlusive forms AMI.

Four reliable angiographic criteria for the diagnosis of mesenteric vasoconstriction the cause of NOMI have been identified;

1. narrowing of the origins of multiple branches of the SMA
2. alternate dilatation and narrowing of the intestinal branches -string of sausage sign
3. spasm of the mesenteric arcades
4. impaired filling of intramural vessels.

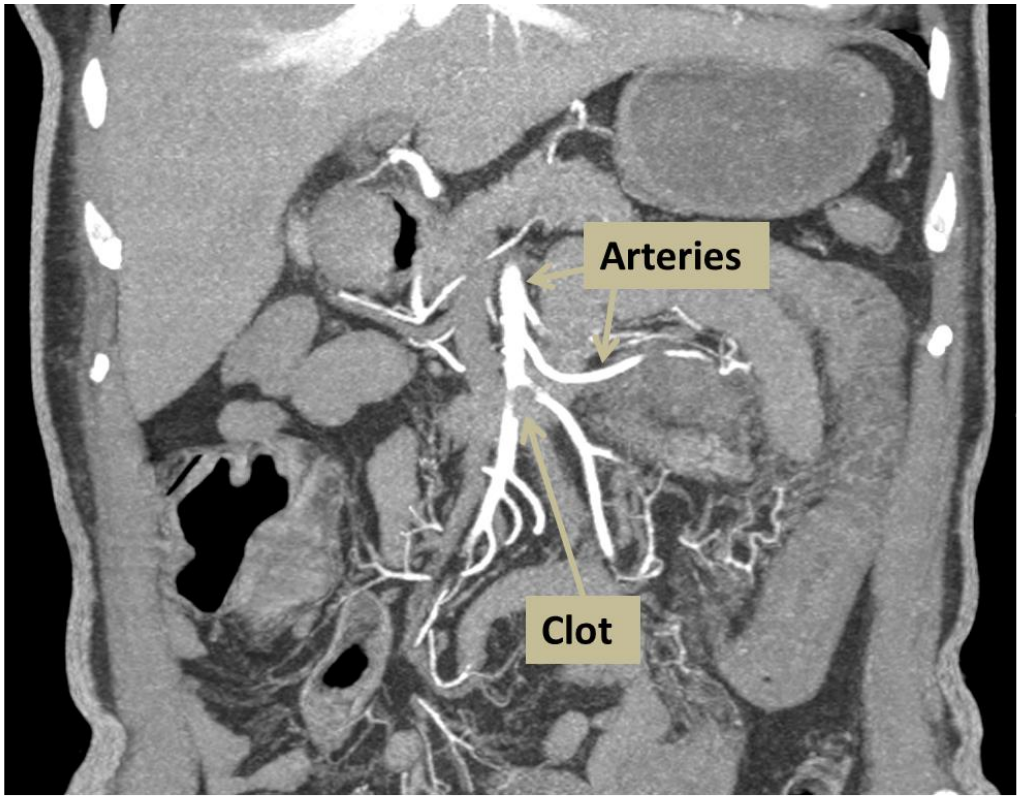


Therefore, if angiography is performed sufficiently early in the disease, patients with occlusive and nonocclusive AMI can be identified before bowel infarction develops and before clinical and radiologic signs of infarction make the diagnosis of intestinal ischemia evident

CT ANGIOGRAPHY:

Delay in diagnosis is the dominant factor that accounts for continued mortality rates as high as 30–70% despite vast clinical experience and recognition of this entity. The multi-detector CTA has supplanted formal angiography as the diagnostic study of choice. Multi-detector computed tomography (MDCT) scanners are essential for the early diagnosis of AMI, but often require specialized personnel to perform and interpret the findings. 3D reconstruction is frequently helpful. Volume rendering as in this image is now a semi-automatic workflow component of many CT machines. These can aide remote communities with less experienced staff. In advanced AMI, the CTA findings reflect irreversible ischemia include

1. intestinal dilatation and thickness,
2. reduction or absence of visceral enhancement,
3. pneumatosis intestinalis,
4. portal venous gas
5. free intraperitoneal air

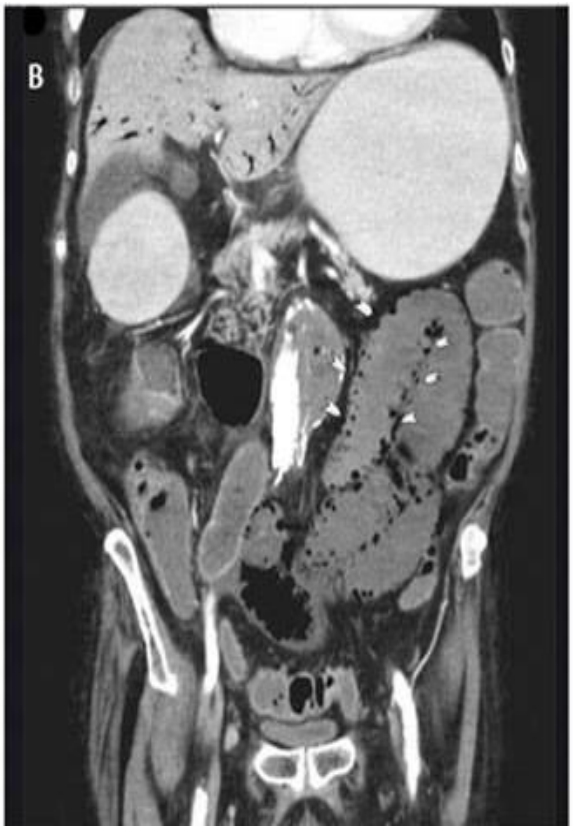


Clot in the superior mesenteric artery obstructing blood flow to the bowel.

Comprehensive biphasic CTA includes the following important steps:

1. Pre-contrast scans to detect vascular calcification, hyper-attenuating intravascular thrombus and intramural haemorrhage.
2. Arterial and venous phases to demonstrate thrombus in the mesenteric arteries and veins, abnormal enhancement of the bowel wall, and the presence of embolism or infarction of other organs.
3. Multi-planar reconstructions (MPR) to assess the origin of the mesenteric arteries

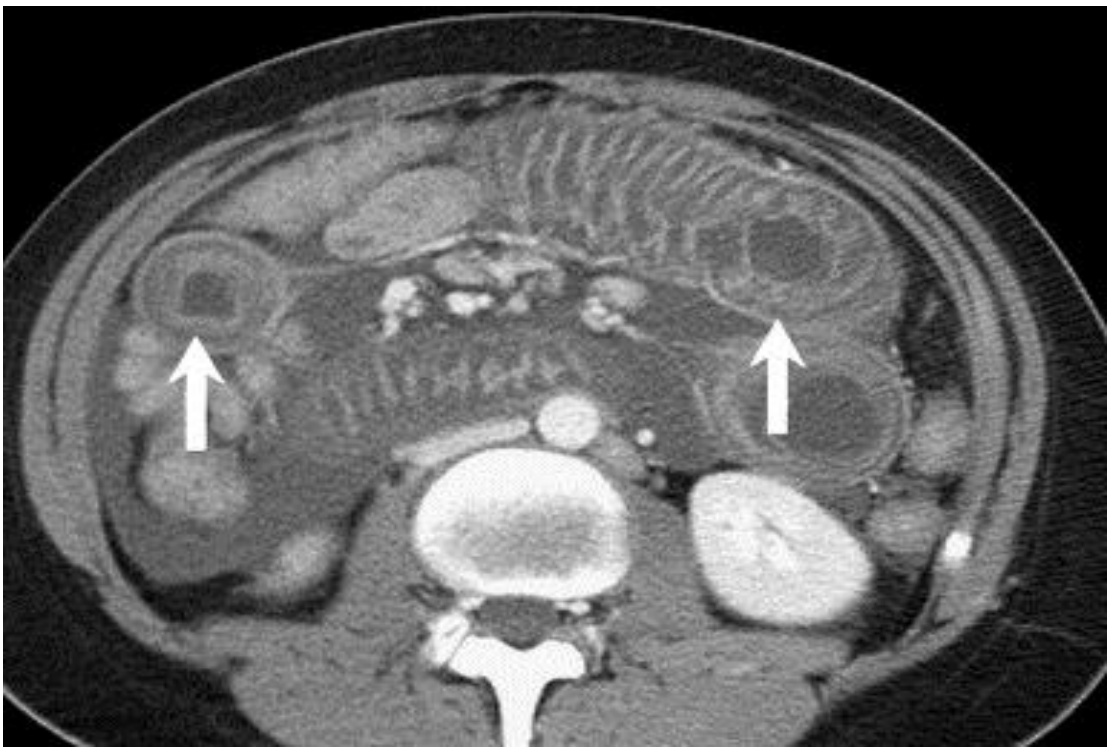
CECT abdomen showing pneumatosis intestinalis and intraportal air



CTA should be performed despite the presence of renal failure, as the consequences of delayed diagnosis, missed diagnosis, or mismanagement are far more detrimental to the kidneys and the patient then exposure to the iodinated contrast agent. A sensitivity of 93%, specificity of 100%, and positive and negative predictive values of 100 and 94%, respectively, have been observed.

In MVT, the most common positive radiological finding on venous phase CTA is thrombus in the superior mesenteric vein on venous phase CTA. This has been described as the target sign .Associated findings that suggest MVT include bowel wall thickening, pneumatosis, splenomegaly, and ascites

Ct Abdomen Showing Target Sign



COLONOSCOPY:

It does not have adequate sensitivity and specificity in detecting ischemic changes but can identify infarction of colon. It does not visualize much of the small bowel, which is frequently involved in AMI.

RADIONUCLIDE IMAGING

It has been used to identify infarcted bowel in animals, but clinical studies in humans have yet to be performed. Also the lack of widespread availability may impair its wider clinical use

DOPPLER ULTRASONOGRAPHY

It has been used to detect a significant stenosis (>50%) in the mesenteric vessels in patients with chronic mesenteric arterial occlusive disease, but its role in AMI seems limited

MAGNETIC RESONANCE IMAGING:

It has shown promise in detecting altered flows in the superior mesenteric vessels in chronic ischemia, but its reliability has not been documented in controlled trials. The relatively long time needed by most medical centers for scheduling and performing magnetic resonance imaging has made its use impractical in this rapidly progressive disorder.

PERITONEAL FLUID ANALYSIS:

Though it may yield abnormal results (elevated white blood cell counts and phosphate, lactate dehydrogenase, and lactate levels) in mesenteric ischemia, its role in the diagnosis of AMI is neither well studied nor widely accepted.

DIAGNOSTIC LAPAROSCOPY:

It may be useful in patients whose clinical status precludes angiography. However, laparoscopic examination of the bowel is limited to the serosal surface, making it unreliable for diagnosing early mucosal necrosis at a time when the serosa still appears relatively normal

TREATMENT:

Once the diagnosis of AMI is made, treatment should be initiated without delay. This should include

- ❖ active resuscitation and treatment of the underlying condition,
- ❖ reducing the associated vasospasm,
- ❖ preventing propagation of the intravascular clotting process,
- ❖ minimizing the reperfusion injury
- ❖ salvaging as much bowel as possible

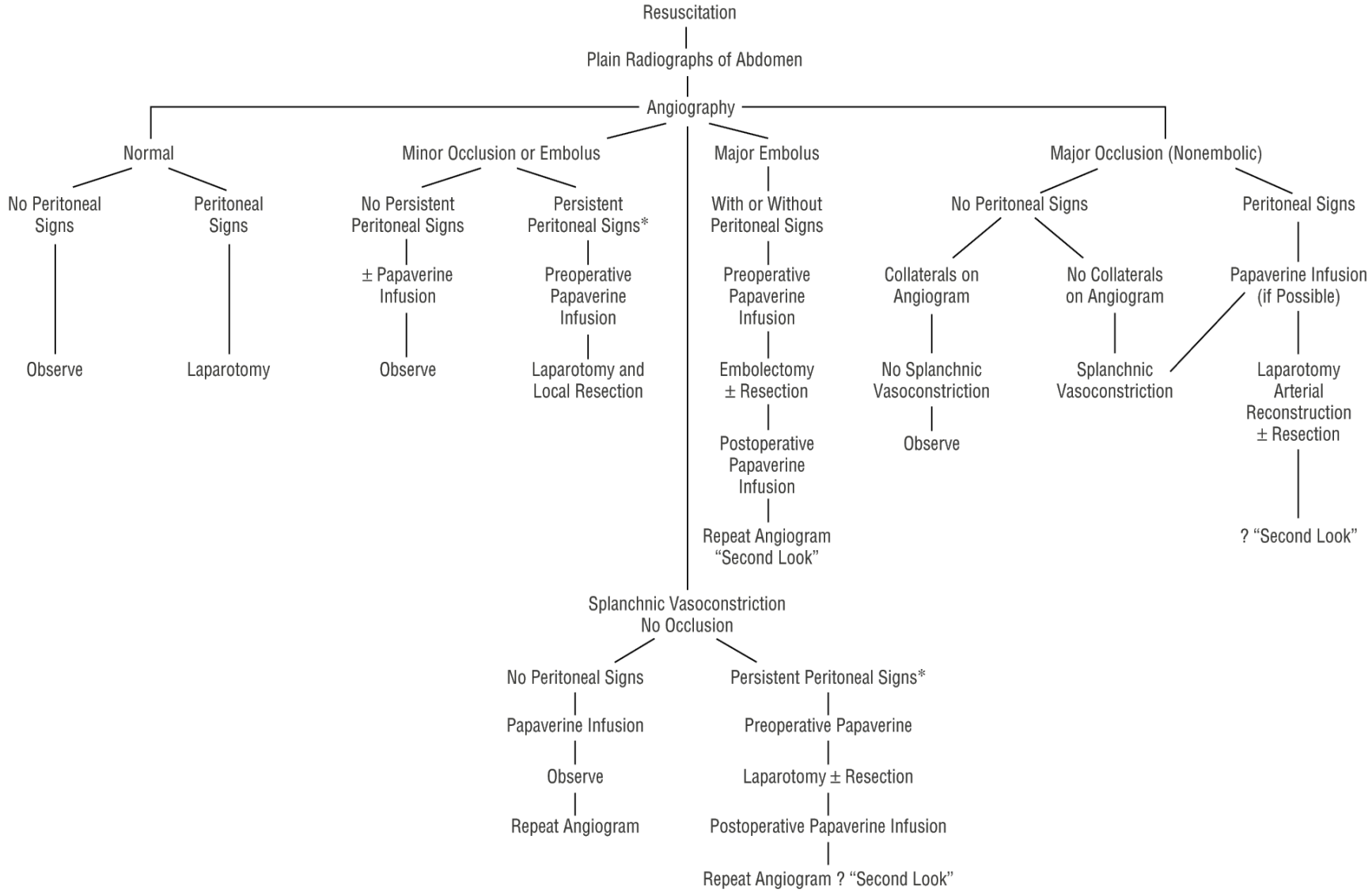
Initial Managment:

1. Intravenous fluid resuscitation with crystalloids and blood products should be started promptly to correct the volume deficit and metabolic derangement. Placement of a Swan-Ganz catheter may be required for judicious fluid resuscitation and hemodynamic monitoring, especially in critically ill patients. Ideally, fluid resuscitation should begin before angiography, and crystalloids may be administered in amounts as high as 100 mL/kg. Supranormalization of hemodynamic values has been attempted, with equivocal results, and it remains to be proven whether such an approach offers an advantage to patients with AMI.
2. Broad-spectrum antibiotics should be given as early as possible.
3. If there are no contraindications to anticoagulation, therapeutic intravenous heparin sodium should be administered to maintain the activated partial thromboplastin time at twice the normal value.

4. After the patient's hemodynamic condition has been optimized and anticoagulation therapy has been initiated, efforts should aim at reducing the mesenteric vasospasm. If the diagnosis of AMI is made without the use of mesenteric arteriography, intravenous glucagon infused initially at 1 µg/kg per minute and titrated up to 10 µg/kg per minute as tolerated may help reduce the associated vasospasm.

PAPAVARINE:

When angiography is used to establish the diagnosis, the angiographic catheter should be left in the SMA for infusions of papaverine or other vasodilators. Papaverine, a phosphodiesterase inhibitor, increases mesenteric blood flow to marginally perfused tissues and may considerably improve bowel salvage. The usual dose is 30 to 60 mg/h. Papaverine use is recommended in cases of arterial embolic or nonocclusive disease because in both conditions the arterial vasospasm persists even after successful treatment of the precipitating event.


Resuscitation
Plain Radiographs of Abdomen
Angiography
Normal
No Peritoneal Signs
Observe
Peritoneal Signs
Laparotomy
Minor Occlusion or Embolus
No Persistent Peritoneal Signs
± Papaverine Infusion
Observe
Persistent Peritoneal Signs*
Preoperative Papaverine Infusion
Laparotomy and Local Resection
Major Embolus
With or Without Peritoneal Signs
Preoperative Papaverine Infusion
Embolectomy ± Resection
Postoperative Papaverine Infusion
Repeat Angiogram "Second Look"
Major Occlusion (Nonembolic)
No Peritoneal Signs
Collaterals on Angiogram
No Splanchnic Vasoconstriction
Observe
No Collaterals on Angiogram
Splanchnic Vasoconstriction
Peritoneal Signs
Papaverine Infusion (if Possible)
Laparotomy Arterial Reconstruction ± Resection
? "Second Look"
Splanchnic Vasoconstriction No Occlusion
No Peritoneal Signs
Papaverine Infusion
Observe
Repeat Angiogram
Persistent Peritoneal Signs*
Preoperative Papaverine
Laparotomy ± Resection
Postoperative Papaverine Infusion
Repeat Angiogram ? "Second Look"

SURGICAL MANAGMENT:

Even in the absence of bowel necrosis, surgical procedures are generally required, except in NOMI, in which the management is primarily medical. Therapeutic improvement during preoperative resuscitation may offer a false sense of security, but bowel infarction, sepsis, and multiple organ failure usually follow unless laparotomy, revascularization, and excision of infarcted bowel segments are performed. Visceral revascularization (embolectomy, thrombectomy, endarterectomy, or bypass) should precede bowel resection in almost all patients with occlusive AMI.

Treatment of MVT is somewhat controversial and depends on the extent of intestinal ischemia. Patients without evidence of bowel infarction often recover spontaneously without operative intervention, and many are treated with anticoagulation alone. The presence of peritoneal signs necessitates emergency laparotomy. Cases of successful treatment with intravascular thrombolytic agents have also been reported. However, thrombolysis is contraindicated when bowel infarction is suspected.

Revascularisation Procedures:

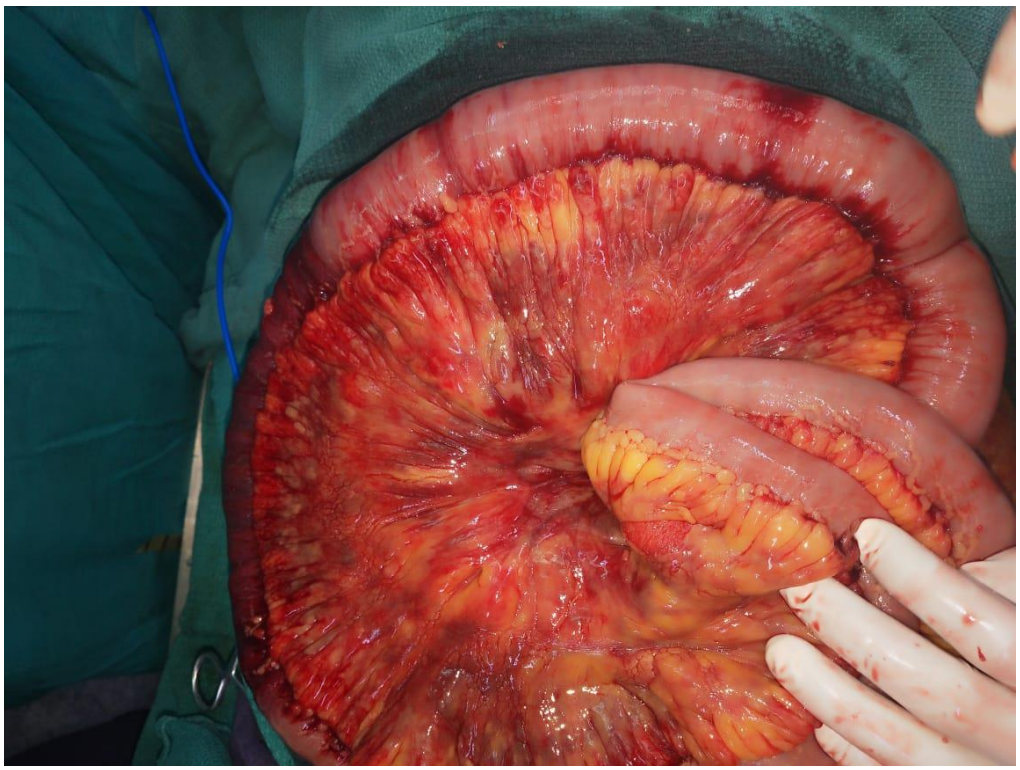
For acute mesenteric embolism, a **standard embolectomy** via a transverse arteriotomy in the proximal SMA should be performed. After embolectomy, the arteriotomy is closed primarily with interrupted nonabsorbable sutures. If the cause of an acute mesenteric embolism is in doubt or if SMA thrombosis is suspected, then a longitudinal arteriotomy is preferred.

With this approach, if a bypass is necessary, the longitudinal arteriotomy can be used as the site for the **distal bypass graft anastomosis**. If flow is adequately restored without a bypass, then the longitudinal arteriotomy can be closed by patch angioplasty to ensure that the luminal diameter is not compromised.

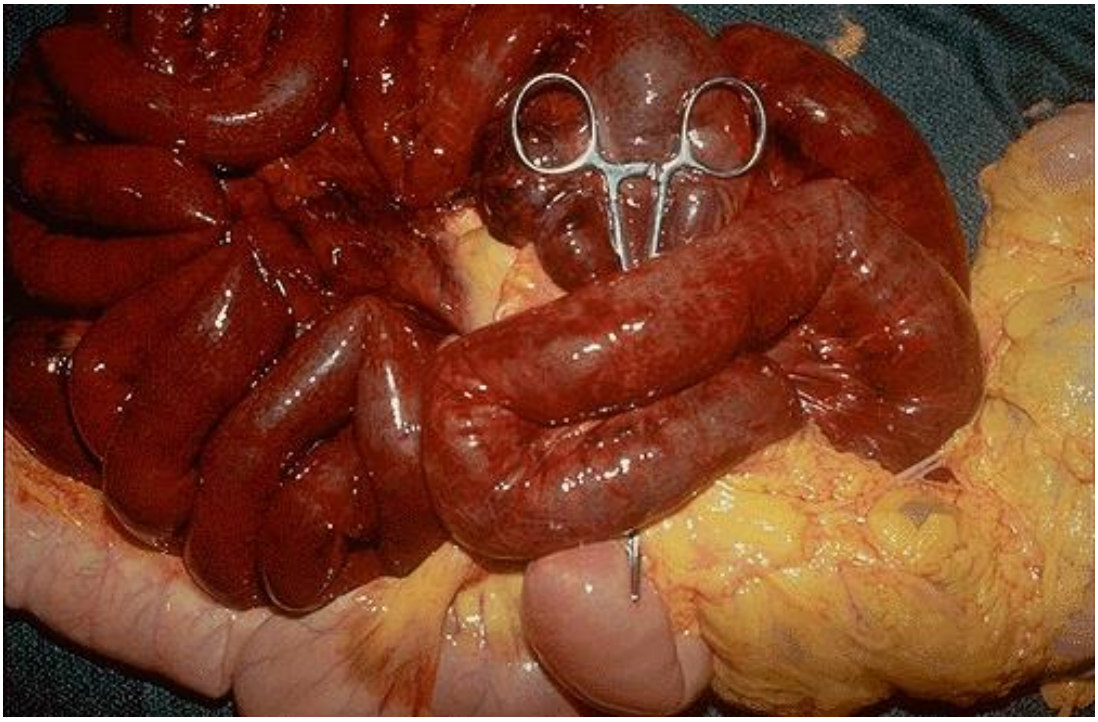
For AMI from arterial thrombosis due to atherosclerotic disease, a vascular bypass graft is usually necessary. The graft can originate from the infrarenal or supraceliac aorta. To avoid potential intra-abdominal contamination from perforated, infarcted bowel, revascularization should be performed using an autologous vein.

Effective treatment of NOMI largely depends on the underlying cause. Initial therapy should aim at removing the offending stimulus and correcting the underlying medical condition. Logically, any infarcted bowel must be resected. Vasodilators, anticoagulation, and **mesenteric regional blockade** have been used in cases in which infarction has not yet occurred. Occasionally, direct transcatheter infusion of papaverine into the SMA restores normal blood flow within minutes. Unless the original provocation or insult is reversed, mortality in NOMI is similar to that in other forms of AMI.

BOWEL SURGERY:



As in embolic disease, revascularization should be performed first, with subsequent resection of clearly nonviable bowel. This allows preservation of potentially viable gut and reduces the possibility of creating a "short-gut syndrome." If the adequacy of perfusion to the bowel is in question, the ends of the bowel may be brought out as stomas. Although nonspecific, the presence of arterial pulsations and the return of bowel peristalsis and normal bowel colour suggest intestinal viability.



Visual examination of the exterior of the bowel is unreliable, especially in cases of NOMI, in which the serosa may appear viable despite the presence of infarcted mucosa. The use of intravenous fluorescein and inspection under a Wood lamp has been shown to be more sensitive and specific, but this method is not widely accepted. Uniform uptake of fluorescein generally suggests bowel viability. Patchy uptake suggests questionable bowel viability, and these segments are better left in situ, with a plan for a "second-look" operation. Intraoperative Doppler ultrasonography has not demonstrated any advantage over clinical judgment in assessment of bowel viability.

Even when the primary operation is successful, the intraoperative assessment of bowel viability is often inaccurate, and few reliable signs are available to detect persistent ischemia or developing infarction in the postoperative period. For this reason a **second-look laparotomy** after 24 to 48

hours is usually recommended. The rationale for this second look is based in part on the frequent occurrence of vasospasm after revascularization. Second-look laparoscopy has been advocated as a substitute for second-look laparotomy, but the reliability of this approach remains unproved.

Postoperative Managment:

Postoperatively, patients treated for AMI are invariably critically ill. Metabolic acidosis and hyperkalemia should be aggressively corrected. Persistent acidosis, especially in the absence of renal failure, should raise concerns about ongoing uncorrected bowel ischemia or infarction. Adequate volume resuscitation is essential to avoid persistent mesenteric hypoperfusion. The mesenteric capillary leak syndrome after mesenteric revascularization is well recognized. Frequently, patients with this condition require 10 to 20 L of crystalloid resuscitation during the first 24 to 48 hours after surgery.

After successful revascularization, efforts should be directed toward limiting any reperfusion injury that may cause progressive mesenteric ischemia or infarction. If the patient's hemodynamic condition allows, infusion of vasodilators should be considered (intravenous glucagon or intra-arterial papaverine). The use of allopurinol, angiotensin-converting enzyme inhibitors, and other free oxygen scavengers may help reduce the reperfusion syndrome. Postoperatively, recurrence and progression of thrombosis are common. Heparinization has been shown to reduce the recurrence of thrombosis.

Sepsis and multiple organ dysfunction syndromes occur in many patients with AMI. The presentation and management of such complications are similar to those of complications from other causes; however, the use of vasopressors may worsen ischemia in marginally viable bowel and exacerbate the condition. Vasopressor options include dopamine (3-8 μg/kg per minute) and epinephrine (0.05-0.10 μg/kg per minute); pure α-adrenergic agents should be avoided, if possible. After initial treatment of the acute event, the possibility of thrombophilia should be investigated. If a prothrombotic condition is detected, long-term warfarin therapy may be necessary.

DIFFERENTIAL DIAGNOSIS:

The list is endless as many of the signs and symptoms associated with AMI are also seen in more common causes of acute abdomen presenting in emergency (pancreatitis, acute diverticulitis, small-bowel obstruction, acute cholecystitis). Therefore in conclusion a high degree of suspicion is required to clinically suspect and do appropriate confirmatory investigation so that treatment can be started at the earliest.

MATERIALS AND METHODS

MATERIALS:

STUDY CENTRE: Institute of General Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital.

TYPE OF STUDY: Observational study.

STUDY DURATION: January 2018 to May 2019

SAMPLE SIZE: 30.

SUBJECT SELECTION:

INCLUSION CRITERIA:

1. Patients admitted to the emergency ward for suspected AMI in whom diagnosis of AMI was confirmed by laparotomy, CT angiography or mesenteric angiography.
2. Age :18-80 yrs

EXCLUSION CRITERIA:

1. Chronic Liver Disease
2. Chronic Renal Disease
3. Hematological Diseases

As these may cause significant abnormal test results of serum markers

METHODS:

All Patients of ages 18-80 yrs of age who presented to the emergency department of madras medical college were admitted. Detailed history taking and clinical examination was done. Patients were adequately resuscitated following which routine blood investigations and imagings were done. Among them 30 consecutive patients in whom acute mesenteric ischemia was identified by explorative laparotomy or CECT angiogram or mesenteric angiogram were included in this study. The following parameters were observed in the selected patients:

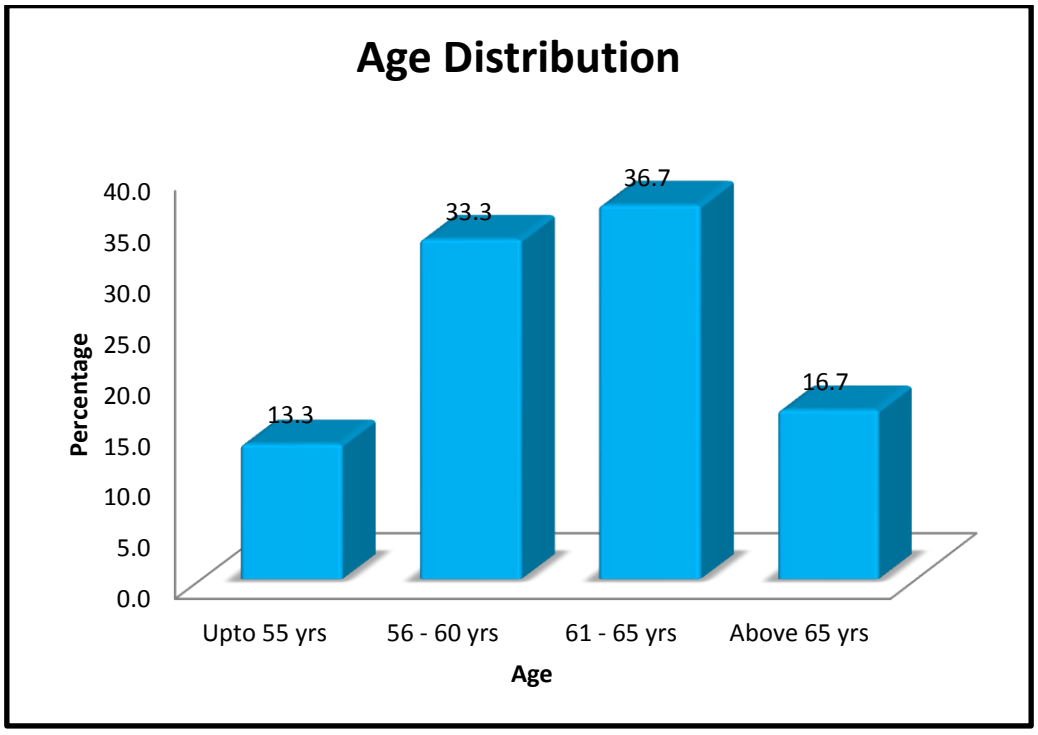
- Demographics(age, sex)
- Previous history(thrombotic disease, atrial fibrillation)
- Clinical signs(body temperature, signs of peritonitis)
- Laboratory investigations
    - ❖ WBC
    - ❖ Platelets
    - ❖ Mean platelet volume
    - ❖ red cell distribution width
    - ❖ Serum amylase
    - ❖ D-dimer
    - ❖ Metabolic acidosis

All collected data will be analysed and conclusions will be drawn based on the above parameters.

DATA ANALYSIS

AGE

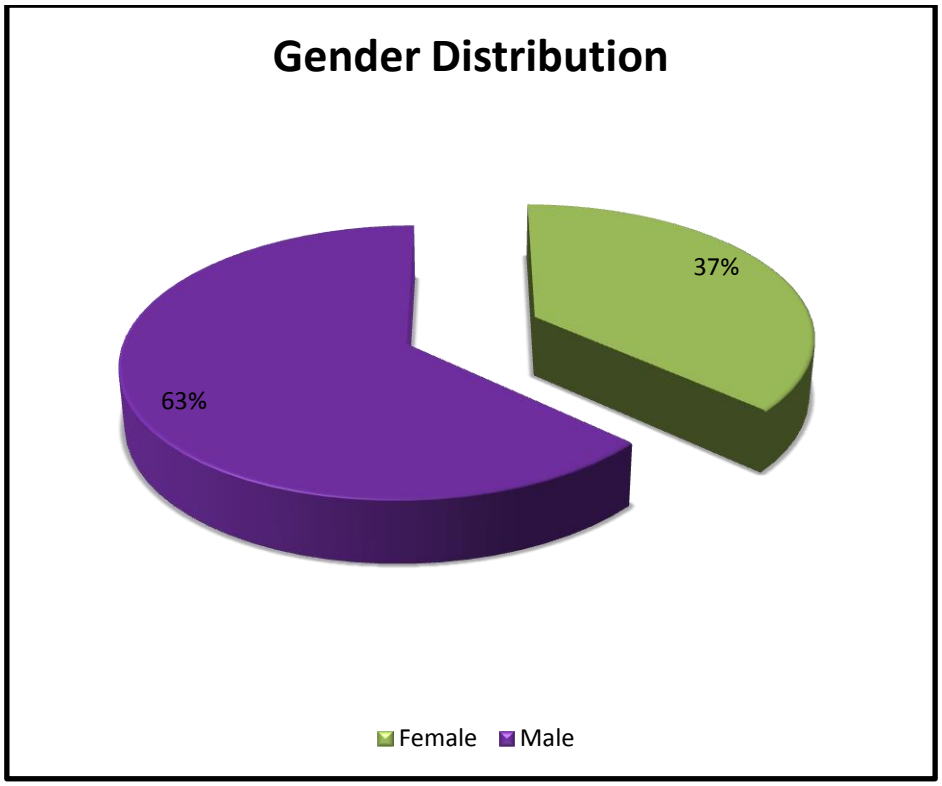
Age	Frequency	Percent
Less Than 55 Years	4	13.3
56- 60 Years	10	33.3
61- 65 Years	11	36.7
Above 65 Years	5	16.7
Total	30	100



Among the 30 patients the majority (11) belonged to the 61- 65 age group, whereas the least (4) were less than 55 years of age.

GENDER

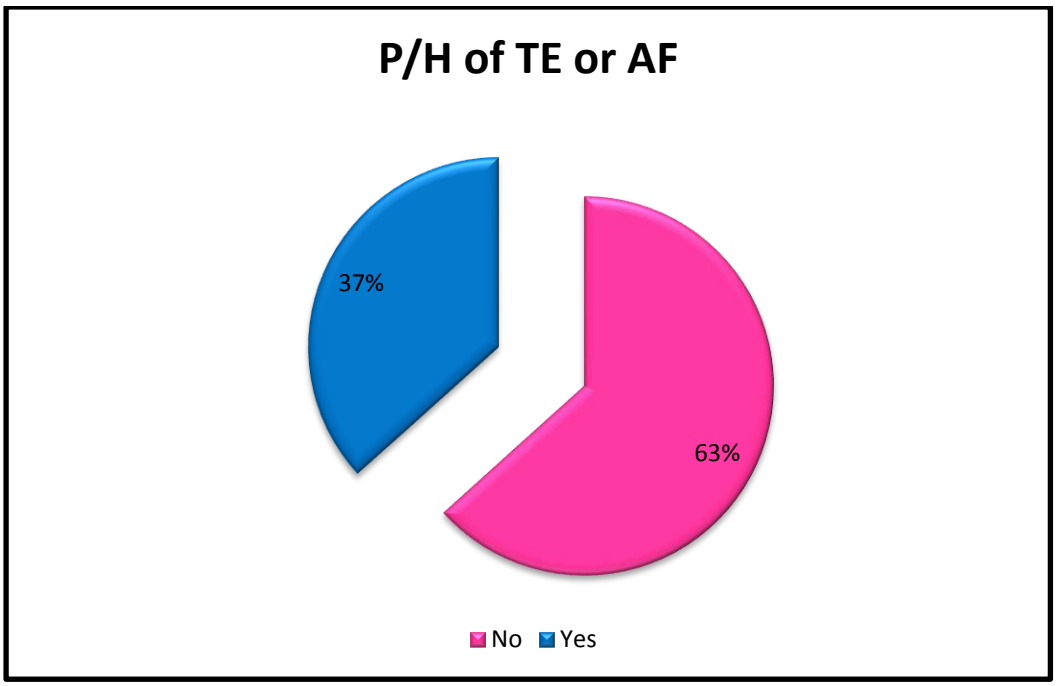
Gender	Frequency	Percent
Female	11	36.7
Male	19	63.3
Total	30	100



In our study majority of the patients were males.

PREVIOUS HISTORY OF THROMBOEMBOLIC EVENTS OR ATRIAL FIBRILLATION

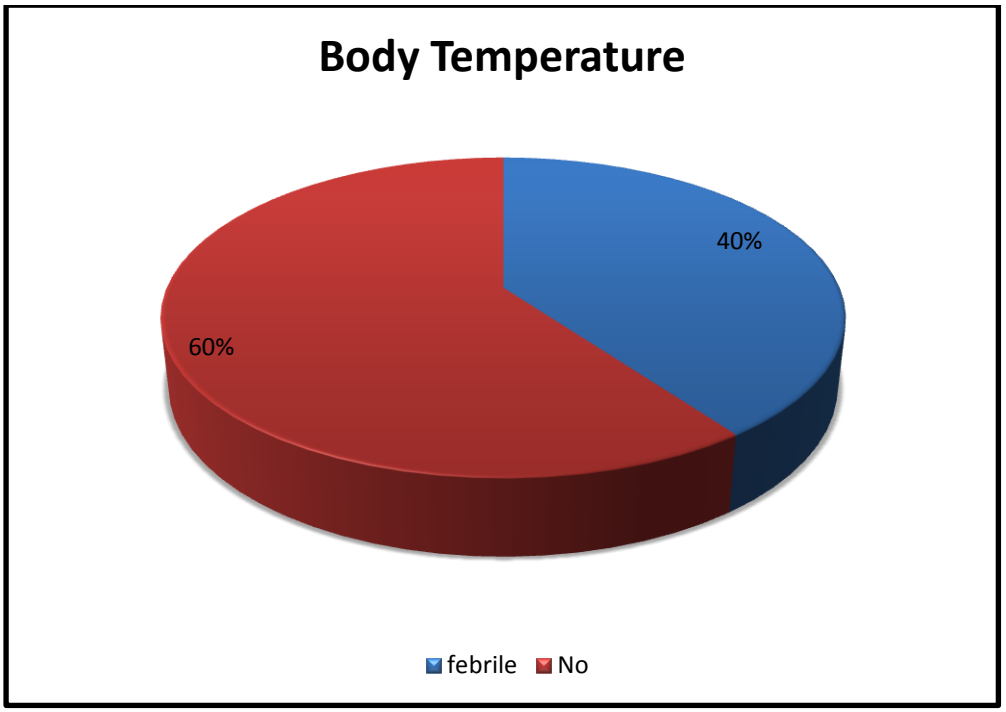
Previous History of Thromboembolic Events or Atrial Fibrillation	Frequency	Percent
No History	19	63.3
Prior History Present	11	36.
Total	30	100



In our study only 37 per cent gave previous history of thromboembolic events or atrial fibrillation.

BODY TEMPERATURE

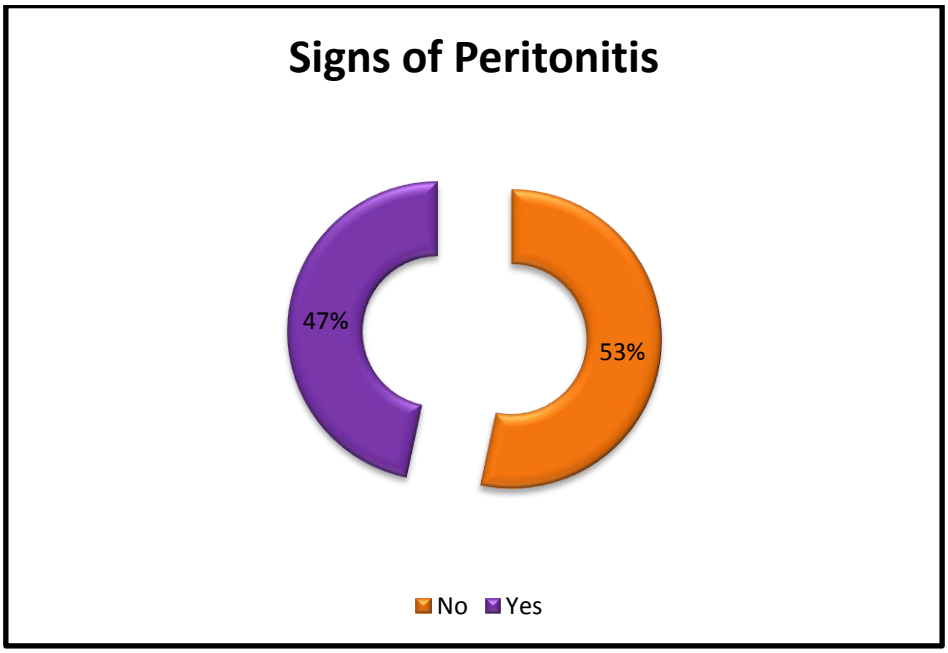
Body Temperature	Frequency	Percent
Febrile(> 37.5 C)	12	40
Afebrile (36.5-37.5 C)	18	60
Total	30	100



60% of the study patients were afebrile while remaining 40% were febrile

SIGNS OF PERITONITIS

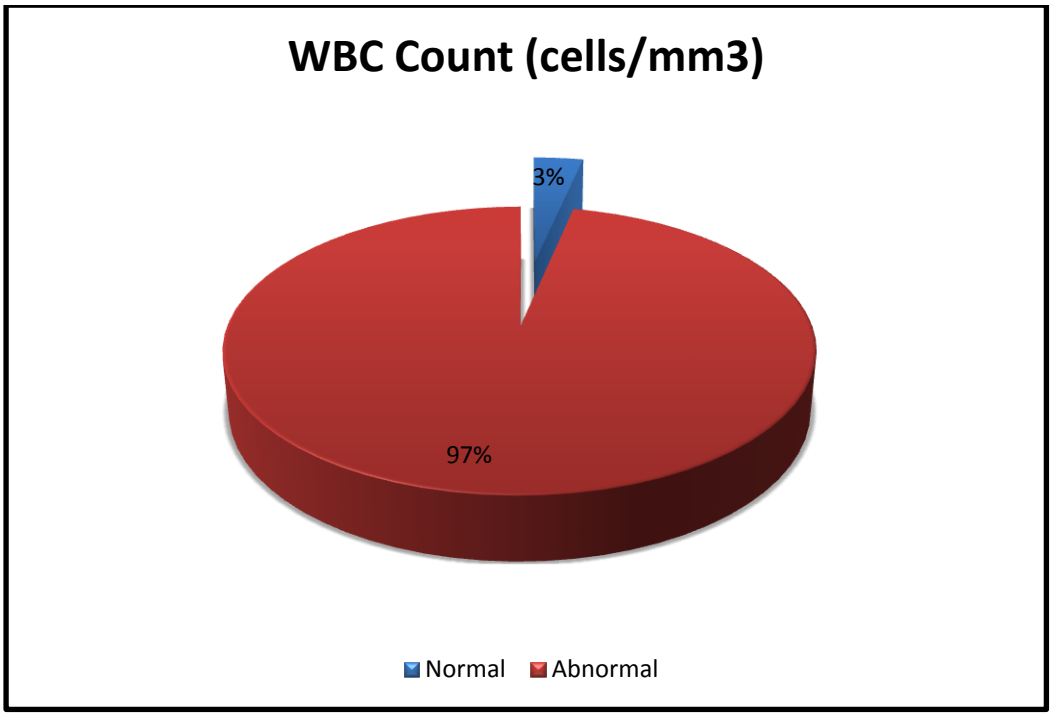
Signs of Peritonitis	FREQUENCY	PERCENT
PRESENT	16	53.3
ABSENT	14	46.7
TOTAL	30	100



53.3% of the patients showed signs of peritonitis while the remaining 46.7% did not show any signs.

WHITE BLOOD CELL COUNTS

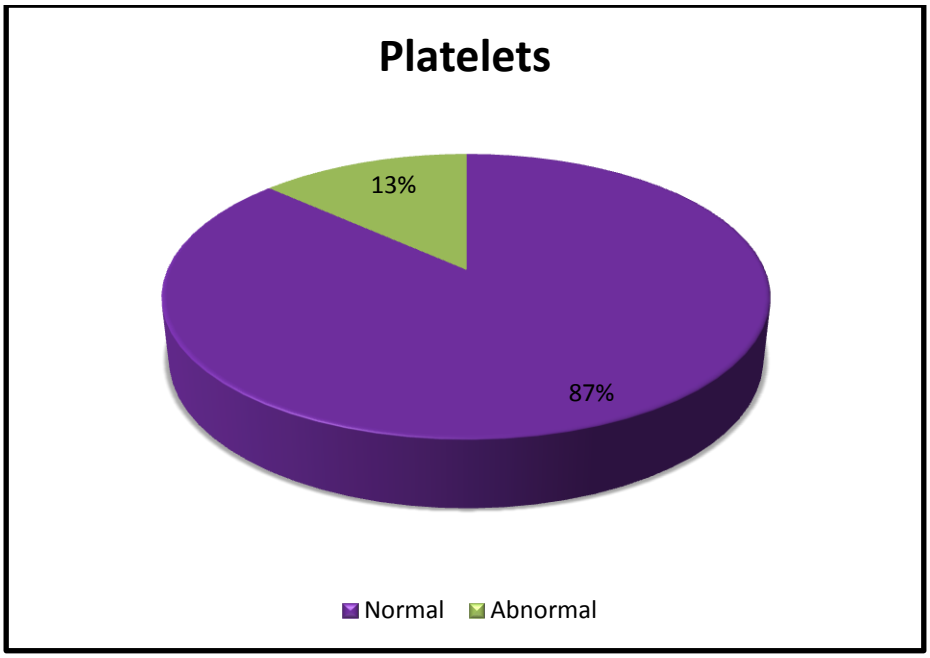
WBC Counts	Frequency	Percent
Normal	1	3.3
Elevated	29	96.7
Total	30	100



29 out of 30 patients showed elevated WBC counts.

PLATELET COUNT

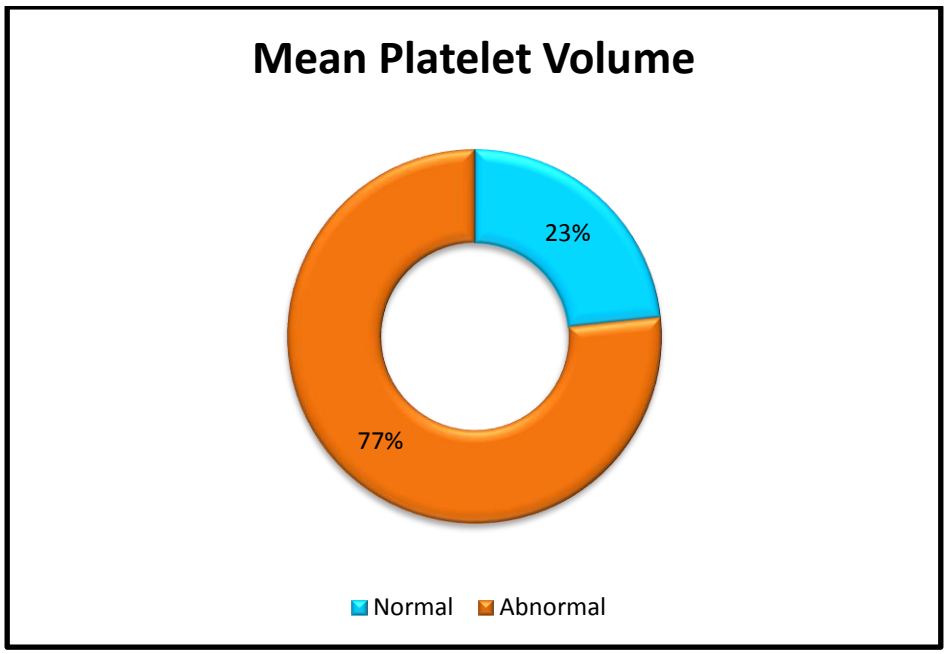
Platelet Count	Frequency	Percent
Normal	26	86.7
Abnormal	4	13.3
Total	30	100.0



26 out of 30 patients showed normal platelet counts while the remaining 4 showed abnormal platelet counts.

MEAN PLATELET VOLUME

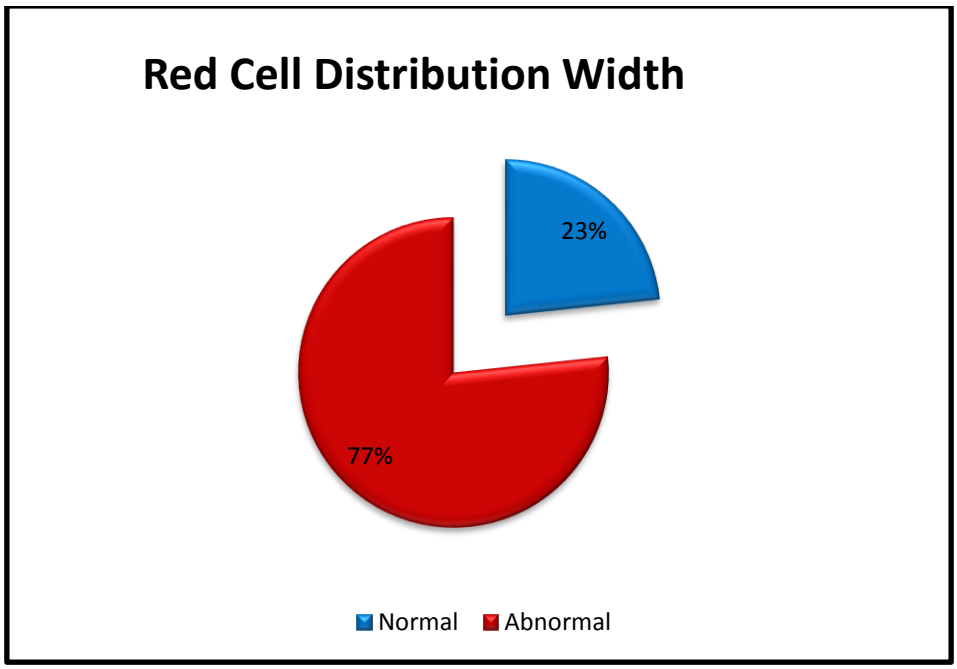
Mean Platelet Volume	Frequency	Percent
Normal	7	23.3
Elevated	23	76.7
Total	30	100



Mean platelet volume was elevated in 76.7% of the patients.

RED CELL DISTRIBUTION WIDTH

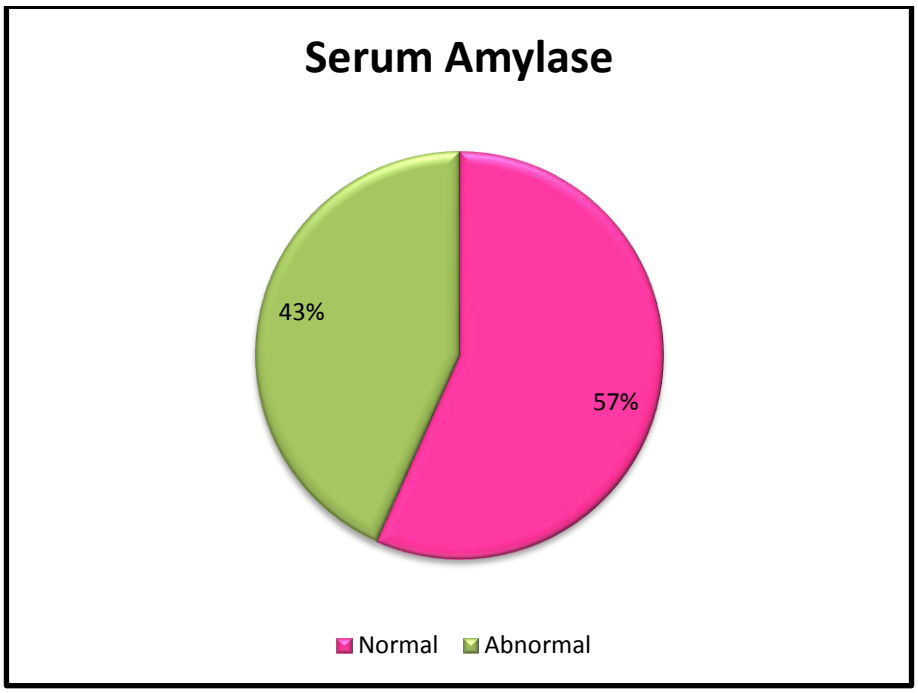
Red Cell Distribution Width	Frequency	Percent
Normal	7	23.3
Abnormal	23	76.7
Total	30	100.0



23 out of 30 patients showed elevated red cell distribution width.

SERUM AMYLASE

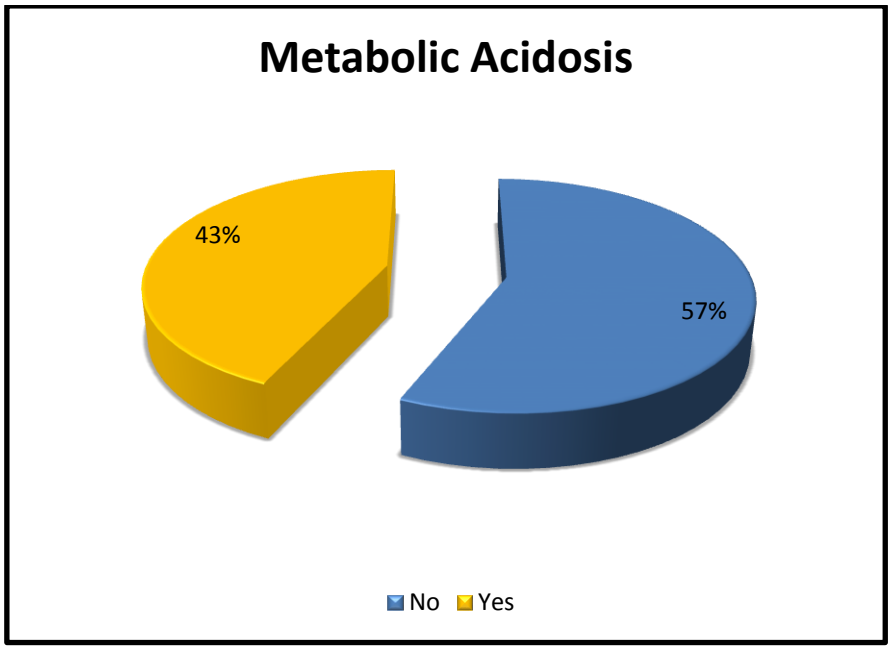
Serum Amylase	Frequency	Percent
Normal	17	56.7
Abnormal	13	43.3
Total	30	100.0



Serum amylase was normal in 56.7% while the remaining 43.3% showed abnormal values.

METABOLIC ACIDOSIS (pH<7.37)

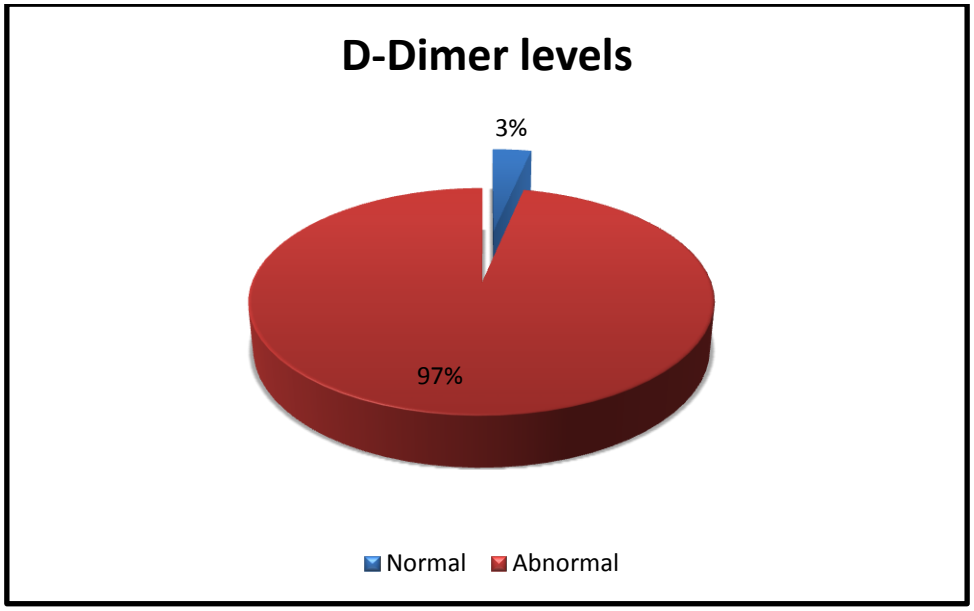
Metabolic Acidosis (pH <7.37)	Frequency	Percent
No	17	56.7
Yes	13	43.3
Total	30	100.0



Metabolic Acidosis was present in 17 out of 30 patients.

D-DIMER

D-Dimer	Frequency	Percent
Normal (< 550 ng/ml)	1	3.3
Elevated (> 550 ng/ml)	29	96.7
Total	30	100



Majority (29 out of 30) showed elevated D-Dimer levels in Serum.

Descriptive Statistics

	N	Minimum	Maximum	Mean	S.D
Age	30	54.0	72.0	61.533	4.5541
WBC count	30	9500.0	26000	20043	3237
Platelets	30	87000.0	510000	282900	117208
MPV	30	1.5	16.0	12.147	3.2250
RCDW	30	11.0	19.0	15.817	1.7883
Serum Amylase	30	23.0	1400.0	206.233	335.1813
D-Dimer	30	450.0	1600.0	983.333	251.7433

DISCUSSION

In this study 12 variables were analysed for each patient

1. Age: it is a disease which occurs predominately but not exclusively in elderly age group. In this study the majority of patients were in the 61-65 years group followed by 56-60 year age group whereas the lowest group were aged less than 55 years
2. Gender: there is no known gender predilection, but in this study the majority of the patients were male (63%). Sex has not been found to be associated with mesenteric ischemia in other studies.
3. Previous history of thromboembolic events or atrial fibrillation: it a proven risk factor of thrombotic/ embolic type of acute arterial mesenteric ischemia but not associated with other types of AMI. Furthermore AMI can be the first presentation of this disease and not necessarily proceeded by these events. 37% of study subjects gave a positive history.
4. Body temperature: hypothermia or hyperthermia is one of the features of systemic inflammatory response syndrome (SIRS) criterion. In AMI due to gangrene of the bowel SIRS develops in late stages. In early stages temperature alterations may not be present. Only 40% of patients were febrile at presentation.

5. Signs of peritonitis: clinical features of peritonitis like guarding, rigidity, abdomen distension, reduced urine output developed when the ischemia became transmural which was observed in 47% of the patients. This can occur in peritoneal inflammation of any aetiology like perforation.
6. WBC counts: Counts more than 11000/mm^3 and less than 4000/mm^3 occur in late stages of bowel gangrene when SIRS develope. Abnormal counts can also occur even in mucosal ischemia. Abnormal counts were observed in 96.7% of sample patients.
7. Platelet count: normal platelet counts were observed in 87% of patients. Low platelet levels are expected once patient develops disseminated intravascular coagulation and hence are associated with poor prognosis.
8. Mean platelet volume: is a marker of the size and activation of platelets, and elevated levels reflects increased production and activation of platelets. It was thought that increased MPV could be associated with increased vascular inflammation and thrombogenicity, and a direct association has been shown between increased MPV and acute thrombotic events, such as acute myocardial infarction, unstable angina, and stroke. Furthermore, increased MPV was found to be an independent predictor factor of mortality in ischemic vascular events. Elevated MPV was noted in 77% of study patients.
9. Red cell distribution width: Increased RDW at admission was a predictor of the extent of necrosis and mortality in AMI patient. Elevated RDW was observed in 77% of the patients.

10. Serum amylase: elevated serum amylase was observed in 43.3% of the patients. Hyperamylasaemia was found in association with all aetiologies of infarction. The magnitude of the hyperamylasaemia appeared to be related to the extent of the bowel infarction, the highest levels occurring when infarction involved the small bowel and colon. It is also elevated in a number of acute abdominal pathologies.
11. D-Dimer: elevated levels were noted in 96.7% of the patients. They are present in the blood after a blood clot is degraded by fibrinolysis. While a negative result practically rules out thrombosis, a positive result can indicate thrombosis but does not rule out other potential causes. Its main use, therefore, is to exclude thromboembolic disease where the probability is low. In addition, it is also elevated in disseminated intravascular coagulation
12. Metabolic acidosis: it is a component of advanced AMI with bowel gangrene accompanied by systemic sepsis. 43.3% of patients showed metabolic acidosis in biochemical analysis.

CONCLUSION

Frequency of WBC counts, D-Dimer, Red cell distribution width and Mean platelet volume were found to be significantly elevated in patients with acute mesenteric ischemia. The frequency of non biochemical parameters in our study such as Demographics(age, sex), Previous history(thrombotic disease, atrial fibrillation) and Clinical signs(body temperature, signs of peritonitis) were found to be low in AMI indicating that cannot be relied in making or excluding the diagnosis. Multiple studies have been previously done analysing the various biochemical parameters and their role in disease process and how they can be exploited for use in regular clinical practise to diagnose and identify the prognosis of AMI. However this study is a single centre study done with a small sample size, more multicentre comparative studies involving large sample are required to validate these results for use in clinical practise.

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patient no	age	sex	previous history of thromboembolic events or atrial fibrillation	body temperature >37.5 celcius	signs of peritonitis	wbc count (cells/mm3)	platelets 15000-450000/mm3	mean platelet volume 9.4–12.3 fL	red cell distribution width 11.5-14.5%	serum amylase (U/L) normal-23 to 85	D-dimer (ng/ml)	metabolic acidosis (pH < 7.37)
1	59	m	yes	febrile	yes	19000	250000	8.5	13	23	650	yes
2	60	f	no	afebrile	no	18400	210000	12.5	15	86	690	no
3	55	m	no	afebrile	yes	20100	190000	11.9	16	110	770	yes
4	64	m	yes	afebrile	no	22000	110000	12	15.5	45	980	no
5	66	m	no	febrile	no	18000	450000	7.8	18	1400	1250	yes
6	61	f	no	afebrile	yes	19800	510000	13.2	17	300	1300	no
7	55	m	yes	afebrile	no	19000	230000	13.5	11	58	860	yes
8	54	f	no	febrile	yes	21000	250000	15.9	15	79	880	yes
9	59	m	no	afebrile	no	16500	190000	10.2	14	80	900	no
10	59	m	yes	febrile	yes	19900	90000	9	15	34	1180	yes
11	60	f	no	afebrile	no	17800	160000	13.6	16	90	1250	no
12	61	m	yes	afebrile	no	20100	440000	1.5	12	76	1050	yes
13	72	m	no	febrile	yes	22000	340000	15.6	16	145	680	no
14	63	m	no	afebrile	no	21000	390000	14.8	17	880	940	yes
15	60	f	yes	afebrile	yes	20900	290000	9.8	16.5	55	790	no
16	65	m	no	febrile	no	18900	410000	9.1	16	67	900	no
17	63	f	no	afebrile	yes	20100	390000	13.9	15.5	790	450	yes
18	65	m	yes	afebrile	no	19900	420000	16	16	65	1150	no
19	70	m	no	febrile	no	23000	350000	14.9	18	86	990	no
20	54	m	no	afebrile	yes	22900	300000	14.2	17	230	1010	no
21	69	f	yes	afebrile	no	17000	87000	15.6	16	950	1600	yes
22	65	f	no	febrile	no	26000	200000	14.9	13	66	1100	no
23	66	m	no	afebrile	yes	22500	190000	11	16	49	1450	no
24	60	m	yes	afebrile	no	15900	370000	8.7	17	56	790	yes

25	63	m	no	febrile	no	19000	180000	13.7	16	30	750	no
26	58	f	no	afebrile	yes	18000	290000	15	18	90	850	no
27	63	m	yes	febrile	no	25000	450000	14.2	19	59	980	yes
28	61	f	no	afebrile	yes	9500	350000	13.5	17	45	1250	no
29	60	f	no	febrile	yes	26000	210000	10.5	17	38	1150	no
30	56	m	yes	febrile	yes	22100	190000	9.4	16	105	910	yes