A Dissertation on

A STUDY ON RAISED SERUM LDH LEVEL IN ACUTE INTESTINAL OBSTRUCTION – A MARKER OF BOWEL GANGRENE

COIMBATORE MEDICAL COLLEGE HOSPITAL

Dissertation submitted to

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

CHENNAI - 32, TAMIL NADU

With partial fulfilment of the regulations

For the award of the degree of

M.S. DEGREE EXAMINATION

BRANCH I – GENERAL SURGERY

COIMBATORE MEDICAL COLLEGE HOSPITAL

COIMBATORE

APRIL 2016

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled " **A STUDY ON RAISED SERUM LDH LEVEL IN ACUTE INTESTINAL OBSTRUCTION – A MARKER OF BOWEL GANGRENE "** is a bonafide and genuine research work carried out by me under the guidance of **Dr. D. N. RENGANATHAN, M.S, FAIS., Professor, Department of GENERAL SURGERY, Coimbatore Medical College and Hospital,** Tamil Nadu, India.

DATE: Signature of the Candidate

PLACE: **Dr. S. SAKTHIVEL**

CERTIFICATE

This is to certify that dissertation entitled**, " A STUDY ON RAISED SERUM LDH LEVEL IN ACUTE INTESTINAL OBSTRUCTION – A MARKER OF BOWEL GANGRENE "** Submitted by Dr. S. Sakthivel in partial fulfilment for the award of the degree of master of surgery in GENERAL SURGERY by The Tamil Nadu Dr .M.G.R. Medical University, Chennai, is a bonafide record of the work done by him in the Department of general surgery, Coimbatore Medical College and Hospital, during the academic year 2013-2016.

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Name of the Candidate : Dr. S. Sakthivel

Course : MS General Surgery

Period of Study : 2013 - 2016

College : Coimbatore Medical College

Dissertation Topic : A study on raised serum LDH level in acute intestinal obstruction - A marker of bowel gangrene.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not accepted~~ and you are permitted / ~~Not permitted~~ to proceed with the above Study.

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CONTENTS

INTRODUCTION

Intestinal obstruction accounts for 20% of all surgical emergencies around the world. Intestinal obstruction can result from variety of causes. When strangulation superimposes and blood supply to the bowel is compromised , it may lead to bowel gangrene. The most common cause of bowel gangrene secondary to mechanical obstruction is strangulated hernia in India and post operative adhesions in developed countries.

Bowel gangrene is a major abdominal catastrophe associated with high mortality rate. This increased mortality is attributed to difficulty in diagnosing the condition early, the late presentation of the patient to the hospital and non availability of précised diagnostic tool for assessing bowel gangrene.

Lactate dehydrogenase is an enzyme found abundant in intestinal mucosa and when the intestinal mucosa undergoes tissue hypoxia/ischemia, it is released into the serum and its serum level increases. Hence serial estimation of the serum LDH helps us to identify whether the obstruction goes on for gangrene or not. This study was conducted to emphasise the significance of early estimation of rise in LDH levels followed by early intervention which helps in reducing the morbidity and mortality caused by bowel gangrene following obstruction.

LDH BASICS :

Other conditions where LDH is frequently encountered both clinically and on standardised test to :

- Diagnose ischemic hepatitis (LDH / ALT >1)
- Diagnose renal infarction (LDH 4 times the upper limit of Normal)
- Diagnose germ cell tumours
- To predict mortality in Patients with acute pancreatitis.

AIM OF THE STUDY

To study the raised level of LDH in patients with acute intestinal obstruction and correlate with bowel viability in CMCH, Sep 2014-2015.

OBJECTIVE OF THE STUDY

1. To estimate the levels of serum LDH in patients presenting with symptoms of intestinal obstruction
2. To identify the patients with elevated levels of serum LDH and correlate the elevated levels of LDH with viability of the bowel.

HISTORICAL HIGHLIGHTS AND

PIONEERS IN TREATMENT

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

(ii) Aretaeus the Cappadocian (81-138 A.D) described in detail the ileus secondary to incarcerated hernia.

(iii) Fabricius d'Aquapendente 12^{th} century. As reported by Duverger, he described the procedure of intestinal repair involving end to end anastomosis.

(iv) Lanfranc 13^{th} century used animal tracheas to connect the divided segments of the bowel.

(v) Ambrose pare , a French physician identified bowel obstruction first time and he reported a patient who died of twisted bowel.

(vi) Franco 1556 described his experience in surgically treating strangulated inguinal hernia. He made an incision over the swelling , dissected the constriction band, inserted a goose quill sized cannula and returned the bowel to the peritoneum.

(vii) Mery 1701 removed several feet of gangrenous bowel and established an artificial anus in a woman suffering from strangulated inguinal hernia.

(viii) Ramdohr 1727 removed two feet of gangrenous small bowel and invaginated the proximal end of the bowel into the lumen of the distal segment, securing the connections with a few sutures.

(ix) Travers 1812 while experimenting with suture techniques, he noted that wounds closed with sutures that passed through all layers of bowel wall healed well.

(x) Jobert 1824 performed end to side anastomosis in dogs and cats using continuous wax sutures.

(xi) Lembert 1826 developed a suture technique employing interrupted sutures that passed through entire bowel wall except mucous membrane.

(xii) Schwartz 1911 used the Xray films to determine the areas of intestinal distension.

(xiii) Klass 1950 diagnosed mesenteric ischemia before infarction. Performed embolectomy without intestinal resection(patient succumbed to acute cardiac failure).

(xiv) Shaw and Rutledge 1957 reported successful superior mesenteric vein embolectomy without bowel resection.

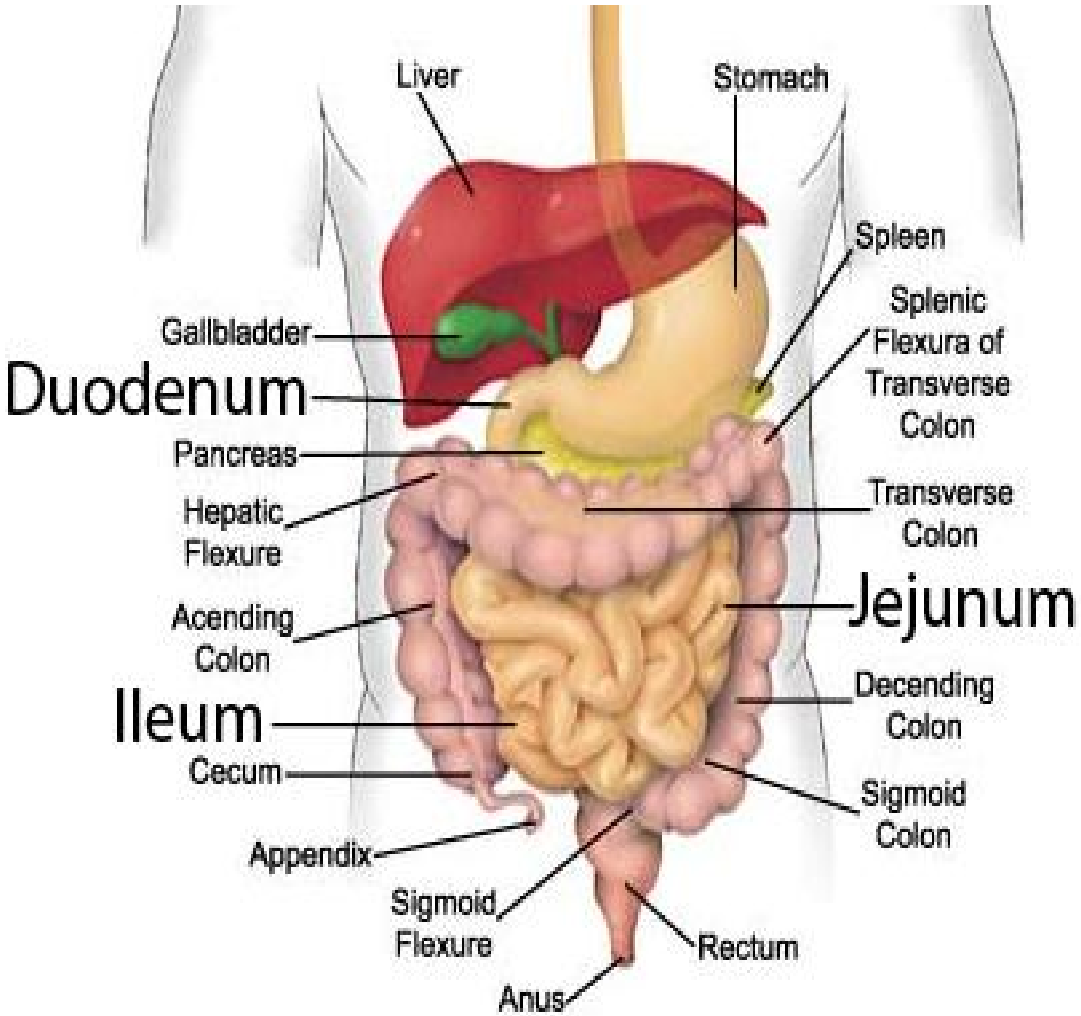
SURGICAL ANATOMY AND PHYSIOLOGY

GUT	PARTS	BLOOD SUPPLY	FUNCTION
foregut	Stomach, duodenum up to the major duodenal papilla	Coeliac axis	Digestion
midgut	Major duodenal papilla to the junction of middle and distal third of transverse colon	Superior mesenteric artery	Absorption
hindgut	Distal third of transverse colon, descending colon, Rectum and anal canal	Inferior mesenteric artery	

ANATOMY OF SMALL INTESTINE:

The small intestine is the longest part of the alimentary tract extending from the pylorus to the ileocecal junction. It is divided into three parts;

1. The duodenum
2. The jejunum
3. The ileum



DUODENUM

The duodenum is the C shaped part odf the intestine with a length of about 25cm. It connects the stomach to the jejunum. It receives openings of the bile and pancreatic ducts. The duodenum curves around the head of the pancreas.

The first 2.5 cm of the duodenum resembles the stomach in that it is covered on its anterior and posterior surfaces with peritoneum and has the lesser omentum attached to its upper border and the greater omentum attached to its lower border ; the lesser sac lies behind this short segment. The reminder of the duodenum is retroperitoneal, being only partially covered by peritoneum. Duodenum is divided into four parts.

First part of the duodenum:

The first part of the duodenum begins at the pylorus and runs upwards and backwards on the transpyloric plane at the level of the first lumbar vertebra.

Second part of the duodenum:

The second part of the duodenum runs vertically downwards in front of the hilum of the right kidney on the right side of the second and third lumbar vertebrae. About half a way down its medial border, the bile duct and the main pancreatic duct pierce the duodenal wall. The unite to form the

ampulla that opens on the summit of the major duodenal papilla. The accessory pancreatic duct(if present) opens into the duodenum a little higher up on the minor duodenal papilla.

Third part of the duodenum:

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

Fourth part of the duodenum:

The fourth part of the duodenum runs upwards and to the left to the duodenojejunal flexure. The flexure is held in position by a peritoneal fold, the ligament of Treitz, which is attached to the right crus of the diaphragm.

Blood supply of duodenum:

Arterial supply:

The upper half is supplied by the superior pancreaticoduodenal artery, a branch of gastroduodenal artery. The lower half is supplied by the inferior pancreaticoduodenal artery, a branch of superior mesenteric artery.

Venous drainage :

The superior pancreaticoduodenal vein drains into the portal vein and the inferior joins the superior mesenteric vein.

Lymphatic drainage :

The lymphatic vessels follow the arteries and drain upward via the pancreaticoduodenal nodes to the gastroduodenal nodes and then to the celiac nodes and downwards via pancreaticoduodenal nodes to the superior mesenteric nodes around the origin of the superior mesenteric artery.

Nerve supply :

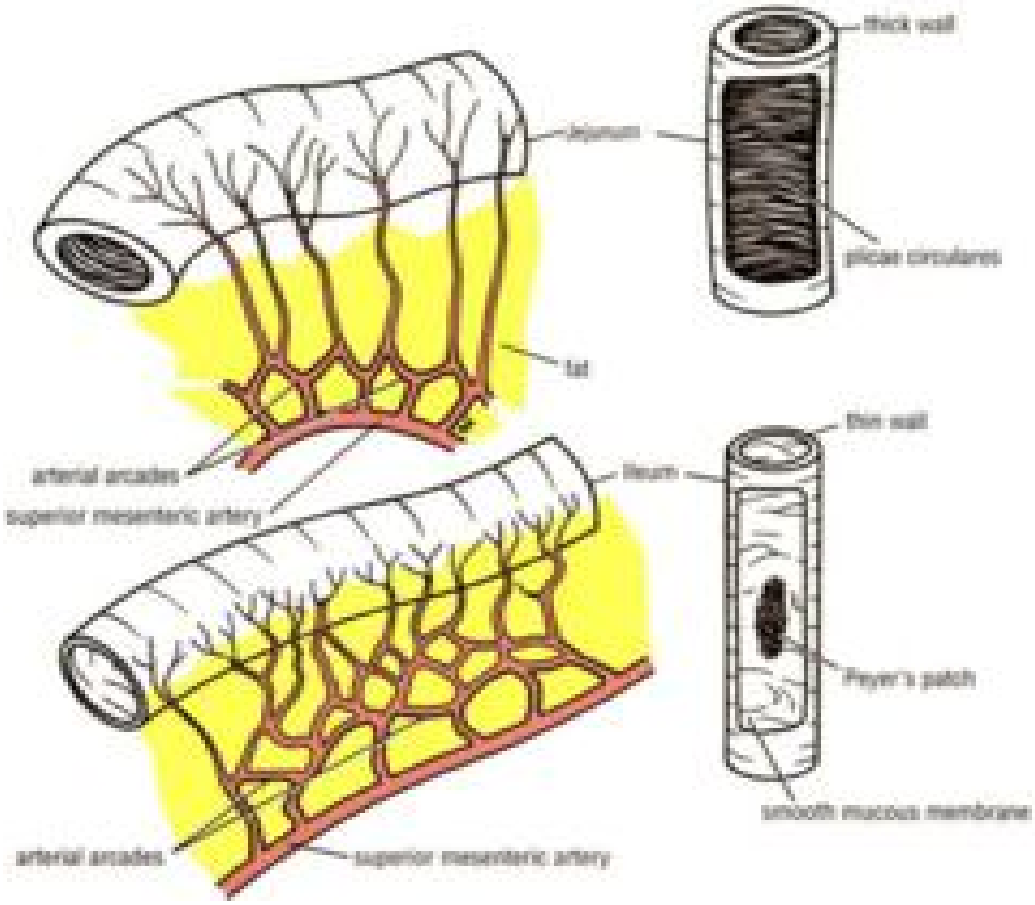
The nerves are derived from sympathetic and parasympathetic (vagus) nerves from the superior mesenteric and celiac plexuses.

JEJUNUM AND ILEUM :

The jejunum and ileum measure about 6m long. The upper two fifth is jejunum. Though jejunum and ileum have distinctive features, there is a gradual change from one to the other. The jejunum begins at the duodenojejunal flexure, and the ileum, ends at the ileocecal junction.

The coils of jejunum and ileum are freely mobile and are attached to the posterior abdominal wall by a fan shaped fold of peritoneum known as the mesentery. The long free edge of the fold encloses the mobile intestine. The short root of the fold is continuous with the parietal peritoneum on the posterior abdominal wall along a line that extends downwards and to the right from the left side of the second lumbar vertebra to the region of the right sacrolilac joint.

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



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

Blood supply :

Arterial supply :

Arterial supply is by superior mesenteric artery. They anastomose with one another to form a series of arcades. The lowest part of the ileum is also supplied by the ileocolic artery.

Venous drainage:

The veins correspond to the branches of superior mesenteric artery and drain into the superior mesenteric vein.

Lymphatic drainage :

Through many intermediate mesenteric nodes reach the superior mesentetric nodes which are situated around the origin of the superior mesenteric artery.

Nerve supply:

The nerves are derived from the sympathetic and parasympathetic nerves from the superior mesenteric plexus.

SUPERIOR MESENTERIC ARTERY :

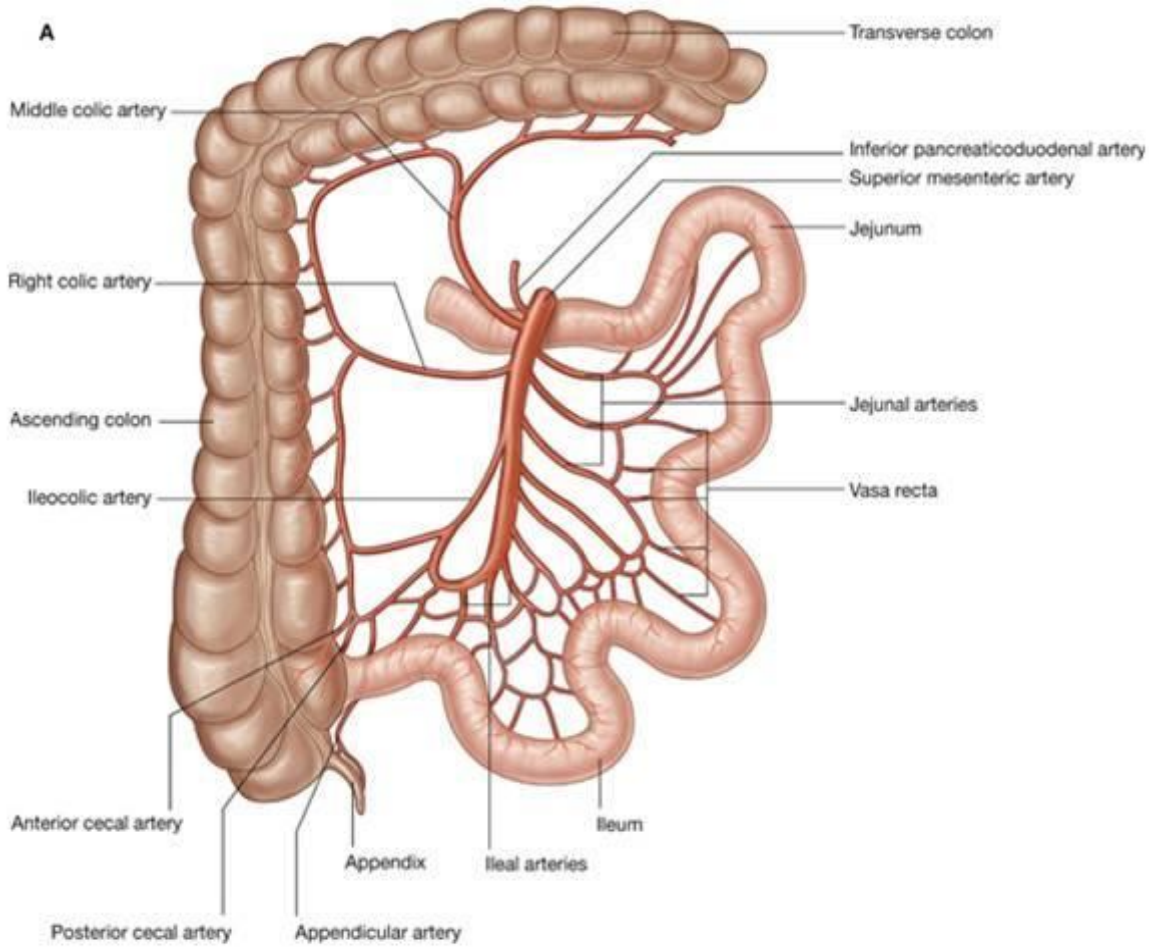
Origin:The superior mesenteric artery arises at an angle of $20^{o} – 30^{o}$ from the anterior aspect of the aorta opposite the upper third of the body of L1, 5-15mm caudal to the celiac artery.

At its origin, SMA measures 1cm in diameter.

Course:

As it passes forwards and downwards, it emerges beneath the inferior surface of the body of pancreas and courses anterior to the third portion of the duodenum and uncinate process of the pancreas

Branches : Constant branches of the SMA include the inferior pancreaticoduodenal artery, the middle colic artery , the right colic artery , the ileocolic artery and the intestinal arteries.



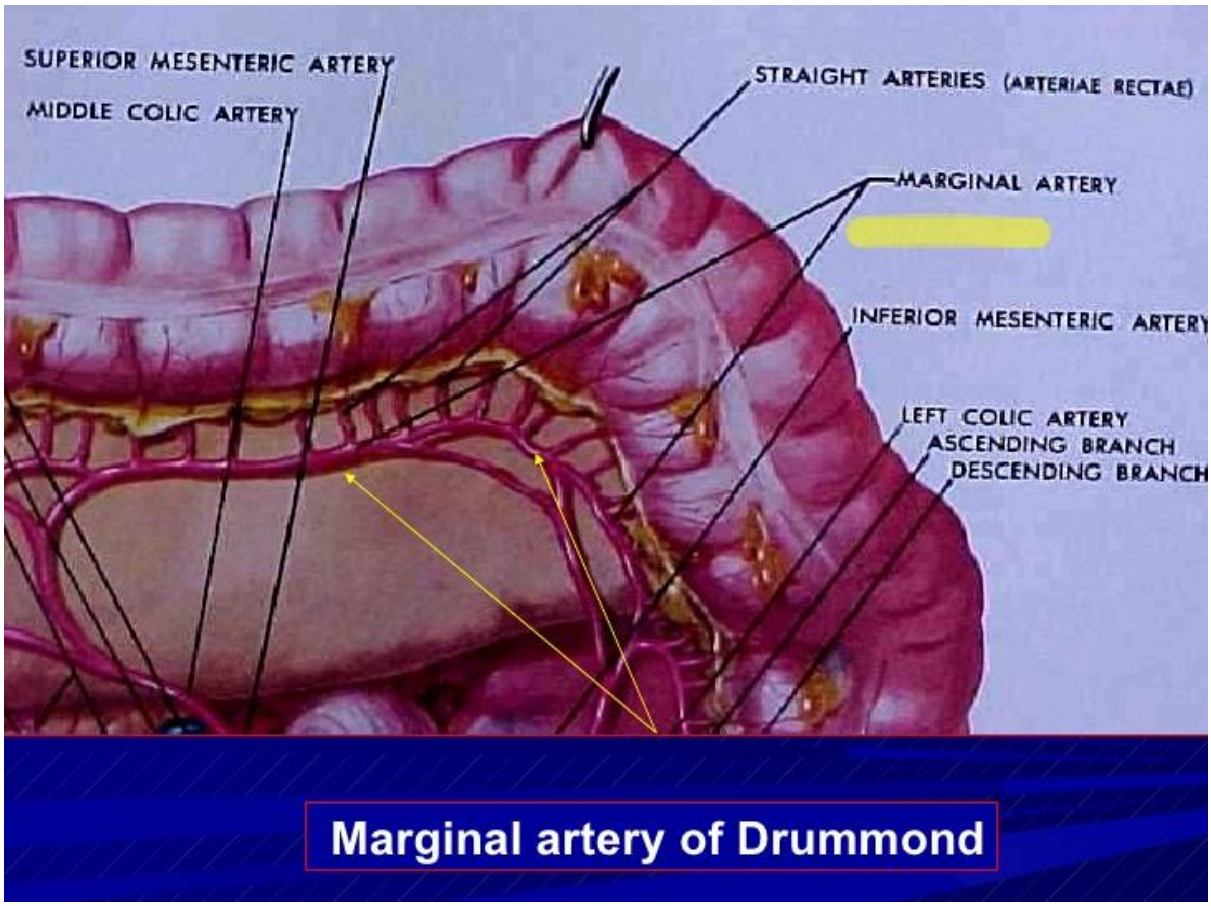
The inferior pancreaticoduodenal artery arises from the right side and communicates with the pancreaticoduodenal branches of the gastroduodenal branch of the hepatic artery. The middle colic artery arises just distal to the inferior pancreaticoduodenal artery along the inferior border of the pancreas.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

CELIAC – SUPERIOR MESENTERIC COMMUNICATIONS:

A common origin of the SMA and the celiac artery occurs in 1% of the individuals. In addition, between these two vessels may be a direct communicating channel known as the anastomotic artery of Buhler, whoch is a remnant of an embryological connection between these two arteries. Other important connections between individually arising superior mesenteric and celiac trunks are the superior and inferior pancreaticoduodenal arcades. Communication with middle colic arterial branches from the superior mesenteric artery occasionally occurs through

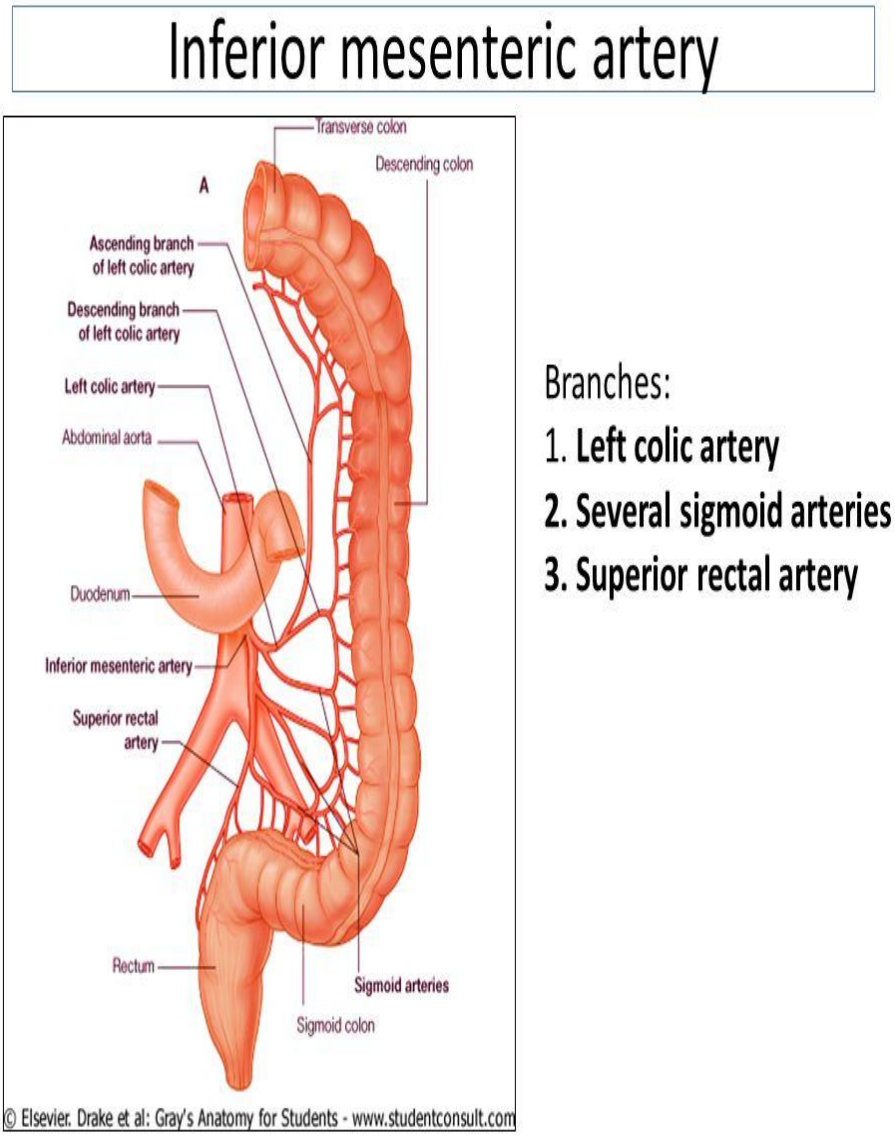
the dorsal pancreatic branch of the splenic artery. After complete occlusion of the celiac axis, generous communications by the pancreaticoduodenal loop maintain hepatic and gastric circulations.

SUPERIOR MESENTERIC – INFERIOR MESENTERIC ARTERIES COMMUNICATIONS:



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

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



REGULATION TO THE MESENTERIC BLOOD FLOW :

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

Extrinsic factors :

Autonomic nervous system:

Sympathetic stimulation causes vigorous contraction of of arteriolar smooth muscles resting in significant reduction in intestinal blood flow. Redistribution of capillary perfusion may result from sympathetic stimulation of precapillary sphincters

The major physiological role of sympathetic vasoconstriction of the gut is to decrease the splanchnic blood flow during activities that require increased blood flow to the skeletal muscle fibres, heart and brain.

Continuous sympathetic discharge may cause persistent mesenteric vasospasm even after the underlying cause of the mesenteric hypoperfusion is corrected. In addition, redistribution of blood flow away from the mucosa, mediated by sympathetic nervous activity may account in part of

susceptibility of mucosa to ischemic damage in various pathological conditions involving the mesenteric circulation.

Drugs :

Nor epinephrine , phenylephrine, methoxamine , metaraminol produces predominant alpha adrenoceptor stimulation and constricts mesenteric vasculature. Alpha blocking agents like phentolamine causes intestinal vasodilatation. Systemic administration of phentolamine may result in decreased intestinal blood flow even though local mesenteric vasodilatation occurs .

Isoproterenol a beta adrenoceptor stimulant increases intestinal blood flow. This action is blocked by propanolol a beta antagonist. Epinephrine which has both alpha and beta adrenoceptor stimulation , in low concentration causes intestinal vasodilation but causes vasoconstriction in high quantities. This is because alpha effects dominate beta mediated vasodilatation.

Dopamine causes vasodilatation by stimulating dopaminergic receptors in mesenteric vessels. Dopamine level >10mcg/kg/min produces an alpha stimulating effect that results in mesenteric vasoconstriction.

Histamine produces mesenteric vasodilation when it is administered intravenously or intra arterially. Histamine causes contraction of non

vascular intestinal smooth muscle, an effect that may limit the increase in intestinal blood flow produced by vasodilation. Bradykinin produces intestinal vasodilation.

Vasopressin and angiotensin II are peptides that produce potent vasoconstriction of intestinal circulation selectively affecting the splanchnic resistant vessels. Vasopressin is released as a result of hypotension and if the hypotension results from mesenteric ischemia, vasopressin may exacerbate vasoconstriction in mesenteric vessels.

Smooth muscle relaxant drugs causing vasoconstriction are tolazoline and papaverine, sodium nitroprusside, sodium nitrate , caffeine and aminophylline.

Digoxin produces significant intestinal vasoconstriction and diminishes mesenteric blood flow. Ergotamine causes increased vascular resistance.

Prostaglandin E1 stimulates the formation of cAMP and causes vasodilatation.

Gastrointestinal and pancreatic hormones :

The synthetic analogue of gastrin, pentagastrin, reduces mesenteric vascular resistance and increases mesenteric blood flow.

Cholecystokinin has been reported to produce both vasoconstriction and vasodilatation under varying conditions.

INTRINSIC REGULATION:

Metabolic regulation:

Conditions resulting in excessive oxygen demand relative to oxygen supply can cause accumulation of metabolites and diminished oxygen levels in interstitial fluid and this produces relaxation of arteriolar smooth muscles and increased perfusion of tissues. Oxygen supply and demand are thereby balanced.

Myogenic regulation :

Vascular smooth muscle tone is altered by arteriolar tension receptors in response to the changes in transmural pressure. Increased vascular transmural pressure results in arteriolar vasoconstriction, increased vascular resistance and diminished blood flow.

Conversely, a decrease in transmural pressure causes vasodilation, diminished vascular resistance and increased blood flow. The result of such regulation is maintenance of constant capillary pressure with minimal alteration in transcapillary fluid exchange. The myogenic control system is the principal factor in the protective mechanism termed autoregulation, which refers to the ability of the mesenteric circulation to maintain the

uniform total intestinal blood flow in the presence of widely varying systemic arterial pressures.

Blood flow in the intestinal villus vessels remain constant even when the perfusion pressure is lowered from 100mmHg to 30mmHg.

SPECIAL ASPECTS OF MESENTERIC CIRCULATION

Reactive hyperaemia:

Both metabolic and myogenic factors probably contribute to intestinal vascular dilation that characteristically occurs after cessation of brief periods of sympathetic stimulation or mesenteric arterial occlusion. The magnitude and duration of this reactive hyperaemia are directly related to duration of decreased perfusion.

The hyperaemic response occurs uniformly throughout the bowel wall after an occlusion period of less than one minute.Increasing the duration of ischaemia results in hyperaemia localised predominantly to the muscularis layer.

Post prandial hyperaemia:

During the initial phase of food in take, anticipation and ingestion of a meal are associated with increased mesenteric vascular resistance, probably caused by generalised sympathetic activity. In the second phase,

digestion of food and absorption of chyme result in decreased mesenteric vascular resistance.

This decreased resistance leads to an increase in superior mesenteric artery blood flow ,which may be double resting blood flow. Decrease in the iliac artery blood flow during digestion redistribution of cardiac output to the mesenteric circulation at the expense of limb blood flow. At maximal blood flow during digestion, the small bowel receives most of the blood,700ml/100g or more.

The stomach receives 300 to 400 ml/100g and the colon receives 200 -250 ml/100g. Post prandial intestinal hyperaemia correlates with the postprandial abdominal pain characteristically experienced by patients with chronic intestinal ischaemia.

Autoregulatory escape:

It is a compensatory mechanism that accounts for maintenance of intestinal blood flow at nearly normal levels, even in the presence of vasoconstrictor influences of continued sympathetic activity or prolonged catecholamine stimulation. Autoregulatory escape probably occurs because local metabolic vasodilator mechanisms ,elicited by ischaemia , become predominant over continued sympathetic vasoconstriction.Subsequent reversal of effects of vasoconstriction and restoration of required blood flow are greater in submucosa than in mucosa. Therefore, there may be a relative

distribution of blood flow away from mucosa. This phenomenon may explain preferential ischaemic damage to the mucosa.

MUCOSAL COUNTER CURRENT EXCHANGE MECHANISMS:

The architecture of microcirculation in the intestinal villus accounts for the existence of a counter current exchabe mechanism. Blood flow in the central part of the villus arteriole is parallel but opposite in direction to that in the subepithelial venous capillaries. Consequently, a gradient in oxygen tension exists between arteriole and venule.

This gradient is most prominent at the base of the villus.Diffusion along the gradient results in a progressive decrease in oxygen tension as blood flows from the base to the tip of the villus.This progressive diffusion gradient is accentuated by condtions that cause low mesenteric flow rates.The counter current exchange mechanism,therefore, tends to aggravate tiisue hypoxia in iachaemic conditions of the bowel.

Collateral blood flow:

When a major mesenteric artery becomes occluded,diminished arterial pressure distal to the obstruction stimulates collateral pathways to open promptly.Blood flow through collateral vessels continues as long as pressure in the vascular bed diatal to the occlusion remains lower than

systemic pressure.Likewise,if the major vessel occlusion is corrected,blood flow through collateral channels ceases.

Intestinal intraluminal pressure:

During an ischaemic insult ,the intraluminal pressure increases and this explains the colicky abdominal pain with early bowel ischaemia.Bowel distension caused by increased intraluminal pressure may diminish blood flow to the involved segment or to the entire intestine.An associated shunting of blood occurs away from the mucosa and muscularis propria.

The well-perfused serosa imparts a normal pink external appearance to the bowel,even though total intestinal blood flow may be markedly reduced.Diminution in flow may persist for hours after relief of distension.These findings emphasize the importance of nasogastric suction for decompression of the bowel in managing intestinal ischaemia.

Response to ischaemia:

Ultrastructural changes in mucosal cells are evident within 10 minutes of superior mesenteric artery occlusion and extensive histologic changes occur within 30 minutes.Progression of these changes ultimately results in bowel necrosis.

Important consequences of bowel ischaemia are increased transcapillary filtration,interstitial edema and ultimately the net movement

of fluid into the bowel lumen.The enzyme xanthine oxidase reacts with hypoxanthine ,which accumulates because of catabolism of ATP and molecular oxygen to produce the cytotoxic oxygen radicals.The cytotoxic effects of the oxygen radicals presumably result from peroxidation of the lipid components of cellular and mitochondrial membrane.

Polymorphonuclear leucocytes appear to contribute to reperfusion injury by releasing lysosomal enzymes at the site of ischaemic injury and by production of oxygen free radicals through neutrophil nicotinamide adenine dinucleotide phosphate oxidase.

THE LARGE INTESTINE

The large intestine is a muscular tube which extends from ileum to the anus. The length of the large intestine is about 135 cm. It has a circular muscle layer and a longitudinal muscle layer.

The circular muscle layer is continuous but the longitudinal muscle layer is arranged in three bands called taenia coli (taeniamesocolia, taeniaomentalis, taenialibera). In the rectum these three taenia coli fuses to form continuous layer.

Caecum is a blind sac which is starting portion of large intestine. It is 6 cm long and 7.5 cm broad .ascending colon is 15 cm long and fixed

posteriorly in the hepatic flexure. Transverse colon is approximately 40-45 cm long and it is fixed by phrenocolic ligament in splenic flexure.

Descending colon is about 20 cm long and fixed posteriorly. Sigmoid colon extends from the descending colon at the pelvic brim to the commencement of the rectum at the level of S3 vertebra and has the length of about 20-60 cm.

The taenia coli of sigmoid colon are wider than in other parts of colon and have appendices epiploicae.

The rectum is 12-15 cm in length and has no taenia coli, no appendices epiploicae,nosacculations and no haustration . the anal canal is the terminal portion of the large intestine is 3-8 cm in length and develops partly from endoderm and partly from ectoderm.

PERISTALTIC MOVEMENTS:

There are two types of peristaltic movements in the intestines.

1. Rhythmic contractions or segmentations
2. True peristaltic movements

Rhythmic contraction or segmentation is myogenic in origin and this movement helps in thorough mixing of food. This movement is best

developed in the ileum, less in the jejunum and rare in the duodenum. The true peristalsis occurs in the whole length of the intestines.

The amplitude and propagatory distance vary with the phase of digestion over the loop proximal to the obstruction. The amplitude and propagatory distance depends upon the loaded condition of the bowel. The rate of peristaltic wave remain constant and not dependent on the digestive phase and the loaded condition of the colon.

In acute intestinal obstruction, fluid accumulates in the lumen very slowly and hence the vomiting is delayed. If the stomach and small intestine become loaded with fluid, there will be considerable abdominal distension.

The abdominal distension is due to the accumulation of fluid and gases proximal to the obstructrion. The losses are water , sodium, potassium. RBC and plasma may be lost from the strangulated bowel segment. General factors contribute to the overall loss which is mainly from the extracellular compartment. The area of absorptive mucosa is unavailable for the process of absorption distal to the obstruction.

PATHOPHYSIOLOGY

CLASSIFICATION OF INTESTINAL OBSTRUCTION

TYPE	PERISTALSIS	OBSTRUCTION	EXAMPLE
Dynamic	present	mechanical	Adhesive bands
adynamic	Absent(eg.paralytic ileus) or present in non propulsive form	No mechanical element	Mesenteric vascular obstruction , pseudo obstruction

CAUSES FOR INTESTINAL OBSTRUCTION

DYNAMIC

Intraluminal

Impaction

Foreign body

Bezoars

Gall stones

Intramural

Stricture

Malignancy

Extramural

Bands and adhesions

Hernia

Volvulus

Intussuception

ADYNAMIC

Paralytic ileus

Mesenteric vascular occlusion

Pseudo obstruction

MECHANISMS OF OBSTRUCTION

Volvulus

Incarceration

Obstruction

Intussuception

Irrespective of etiology or acuteness of onset, in dynamic(mechanical) obstruction the proximal bowel dilates and develops an altered motility. Initially proximal peristalsis is increased to overcome the obstruction, in direct proportion to the distance of the obstruction. If the obstruction is not relieved, the bowel begins to dilate, causing a reduction in peristaltic strength, ultimately resulting in flaccidity and paralysis. This is a protective phenomenon secondary to increased intraluminal pressure.

Factors producing distension proximal to an obstruction :

1. gas
2. fluid

STRANGULATION

When strangulation occurs, due to compromised blood supply, the viability of the bowel is threatened.

Causes of strangulation

1. external

   hernial orifices

   adhesions /bands

2. interrupted blood flow

   volvulus

   intussuception

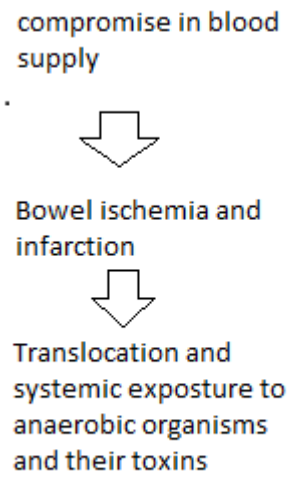
3. increased intraluminal pressure closed loop obstruction

4. primary

mesenteric infarction

The venous return is compromised before the arterial supply. The resultant increase in capillary pressure leads to local mural distension with loss of intravascular fluid and red blood cells intramurally and extraluminally.

Hemorrhagic infarction occurs once the arterial supply is impaired..



The morbidity and mortality associated with strangulation are dependent on age and extent. In strangulated external hernias, the segment involved is short and the resultant blood and fluid loss is small. When a bowel involvement is extensive the loss of blood and circulatory volume will cause peripheral circulatory failure.

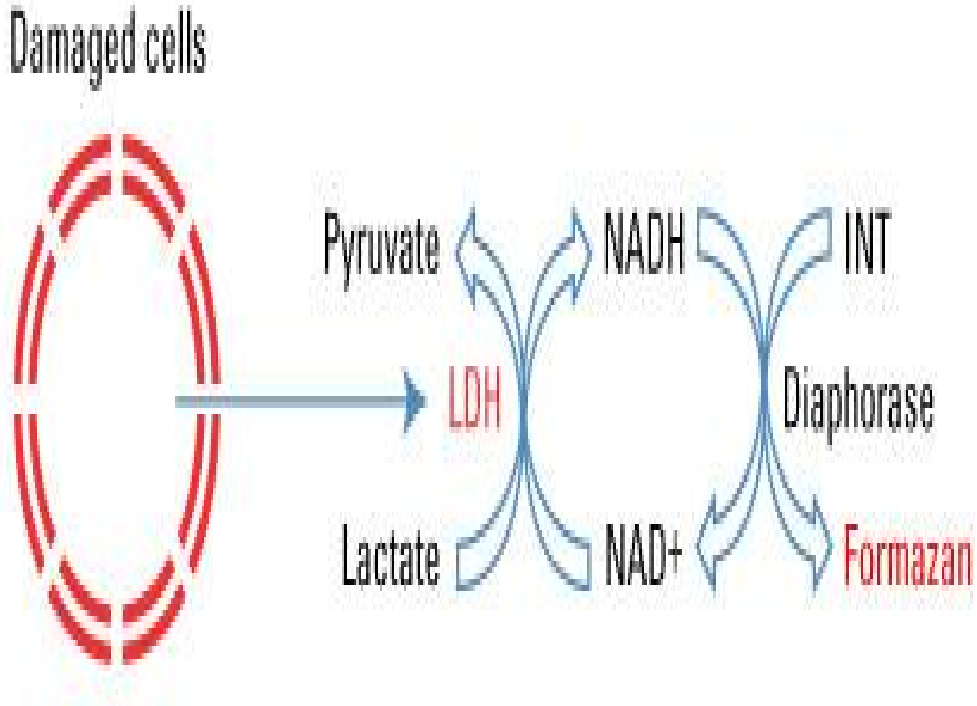
LACTATE DEHYDROGENASE:

Lactate dehydrogenase(LDH) is an enzyme that catalyses the oxidation of L-lactate to pyruvate having NAD+ as the acceptor of hydrogen ion.

L-lactate $\xrightarrow{\text{lactate dehydrogenase}}$ pyruvate (31)

LDH activity is present in all the cells of cytoplasm of the cytoplasm of the body predominantly in cytoplasm of the cell.

The level of LDH in the tissues is 500 times more than that in the serum.

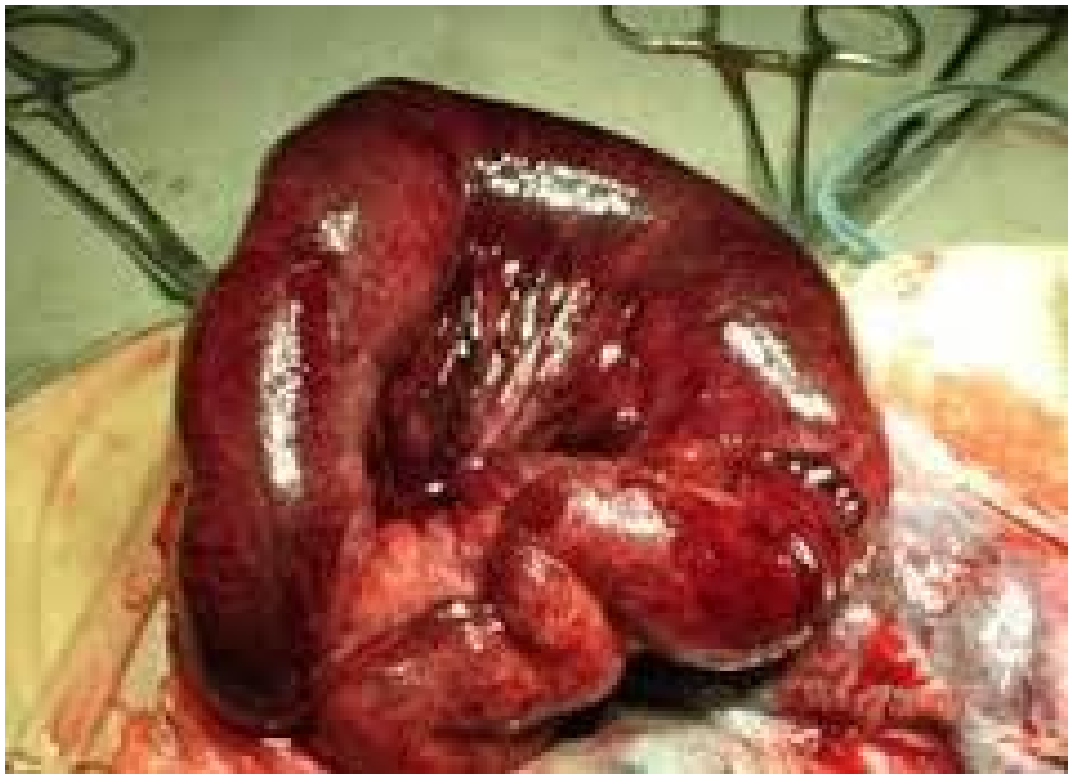


A mass of damaged tissue causes leakage of the LDH enzyme which in turn leads to the elevated levels of the enzyme in the serum.

Necrosis of the small bowel can lead to elevation in the levels of serum LDH and hence LDH can be considered as one of the markers for the onset of intestinal ischemia or gangrene.

STRANGULATED INGUINAL HERNIA

A hernia is said to be strangulated when the blood supply of its contents seriously impaired due to constricted neck of the sac. Gallegos and associates estimated the probability of strangulated hernia over time to be 2.8% over 3 months and 4.5% over 2 years because of enlargement of the neck of the sac.



Pathology :

The venous return is first impeded and the intestine becomes congested and bright red. As the venous stasis increases the arterial supply is also impaired. Ecchymosis appears in the serosa. Blood comes out into the intestinal lumen and into the hernial sac.

The viability of the intestine diminishes and migration of bacteria through the intestinal wall and the fluid within the sac becomes full of bacteria and toxins. The mesentery within the sac becomes congested and hemorrhagic. Thrombosis of its vessel occurs. Gangrene starts 5 to 6 hours after the onset of first symptom of strangulation.

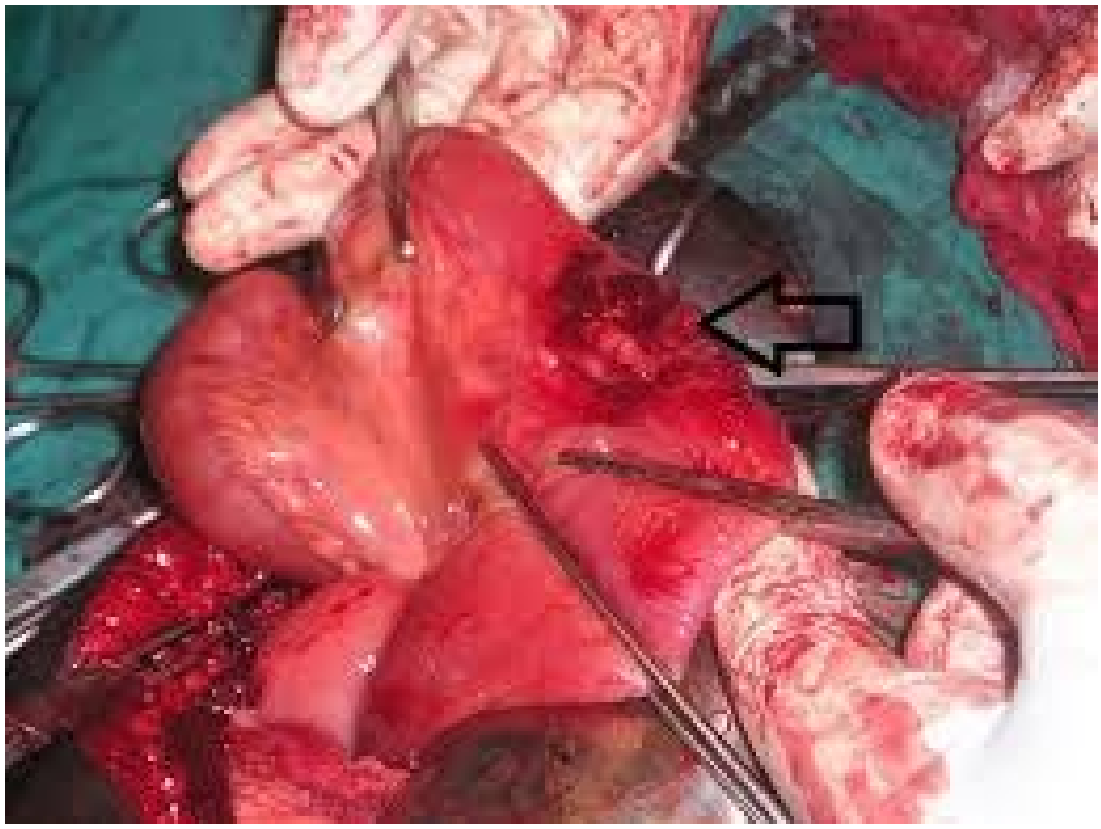
CLINICAL FEATURES :

Abdominal pain and vomiting are the main features. Pain is first at the hernial site and later spread all over the abdomen. If the strangulation is not relieved the paroxysm of pain continues. Such pain will cease only with the onset of gangrene and paralytic ileus. On examination the hernia is tense and tender. Cough impulse is absent.

STRANGULATED RICHTER'S HERNIA

When a portion of the circumference of the intestine becomes the content of the sac it is called Richter's hernia. Strangulation of such hernia often complicates a femoral hernia.

Clinical features mimic gastroenteritis and diagnosis becomes difficult. Vomiting if present is not that frequent. Intestinal obstruction may not develop until half the circumference of the bowel is involved. Intestinal colic may occur and sometimes there may also be associated diarrhoea.



When the paralytic ileus sets in there will be absolute constipation. For the above said reasons, diagnosis becomes delayed and operation performed mostly only when the gangrene and peritonitis sets in.

STRANGULATED FEMORAL HERNIA

In femoral hernia the contents pass through the femoral ring , traverse the femoral canal and comes out through the saphenous opening. After this it progress upwards in the subcutaneous tissue of the thigh and may even reach above the inguinal ligament. Due to the narrow passage of the canal , femoral hernia is more liable for strangulation. Its more common in females and accounts for 20% of hernia cases.

Clinical features

In case of strangulated hernia patient develops sudden pain at the local site which immediately spread all over the abdomen. Swelling is seen below and lateral to the pubic tubercle and below the inguinal ligament. Globular in shape and tense and tender on palpation.

STRANGULATED VENTRAL HERNIA

It is also known as incisional hernia or post operative hernia. This occurs through an acquires scar in the abdominal wall caused by previous surgery.

Etiology

1. Patient related factors

   Obesity with lax muscle , patient suffering from chronic cough , patient with severe anemia , hypoproteinemia or vitamin C deficiency.

2. Operative fallacies

   Injury to the motor nerves supplying the area can occur during incision. Certain incisions are vulnerable to cause nerve injury , for example Kocher's subcostal incision injures the 8^{th} 9^{th} 10^{th} intercostal nerve , battle's pararectal incision for appendicectomy may injure subcostal or ilioinguinal nerve. Inadequate care and hemostasis and keeping tube drainage through the laparotomy wound.

3. Post operative causes

   Infection , post operative cough and distension, post operative peritonitis , early removal of sutures, steroid therapy in post operative period.

PATHOLOGY

Often the incisional hernia starts un noticed and symptomless with partial disruption of the deeper layers of laparotomy wound during immediate or early post operative period. So careful closture of the wound is extremely important. Further wound infection often causes disruption of sutures. Thus the muscles are separated by weak scar tissue. A portion of the muscle may also be destroyed and heals by fibrosis which forms a weak scar.

CLINICAL FEATURES

Previous operation or trauma is usually noted. Ventral hernia occurs more common in fatty elderly females but can occur at any age . Commonly presents as swelling and pain with features of intestinal obstruction. Strangulation though uncommon is liable to occur at the neck of the small sac or in locule of a large hernia.

The old scar is seen within the swelling. The hernia may occur through a small portion often at the lower end. The swelling is irreducible and cough impulse absent and it is tense and tender.

TREATMENT OF STRANGULATED HERNIAS

Emergency operation is the treatment of choice to save the patient's life.

Initial management includes

- intravenous fluid administration,
- nasogastric tube inserted as intestinal obstruction is always associated
- Foley's catheterisation
- parenteral antibiotics is started immediately
- taxis and reduction of hernia sac should be avoided as it will result in reduction en masse, rupture of intestinal wall , rupture of sac extraperitoneally

Incision and opening of the sac is done according to the site of the hernia . the constricting narrow neck is divided. Viability of the bowel is the main thing to be considered. The following points indicate non viable bowel

1. The bowel becomes greenish or blackish in colour
2. It becomes flaccid and lustreless with thrombosis of mesenteric vessels
3. Low peristalsis in the gut
4. Blood stained or foul smelling fluid in the sac

It is always advisable that hot wet mops are placed on the involved bowel for ten minutes and again watched for viability. If the bowel is gently pinched with forceps , peristaltic movement starts and returning of pink colour indicates returning of viability

If the bowel is viable , it is pushed back into the peritoneal cavity and herniorrhaphy done. No form of hernioplasty should be attempted.

If the bowel is non viable

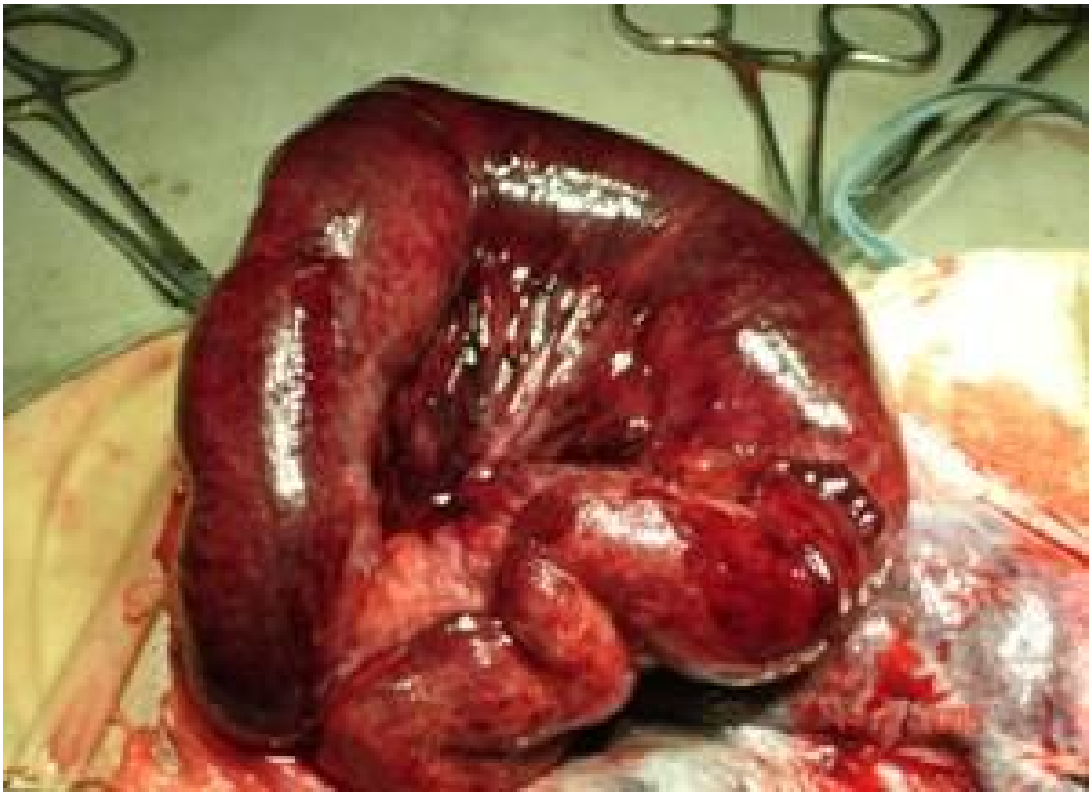
(i) A linear patch of gangrene at the constriction ring is best treated by invaginating it by means of Lembert's suture

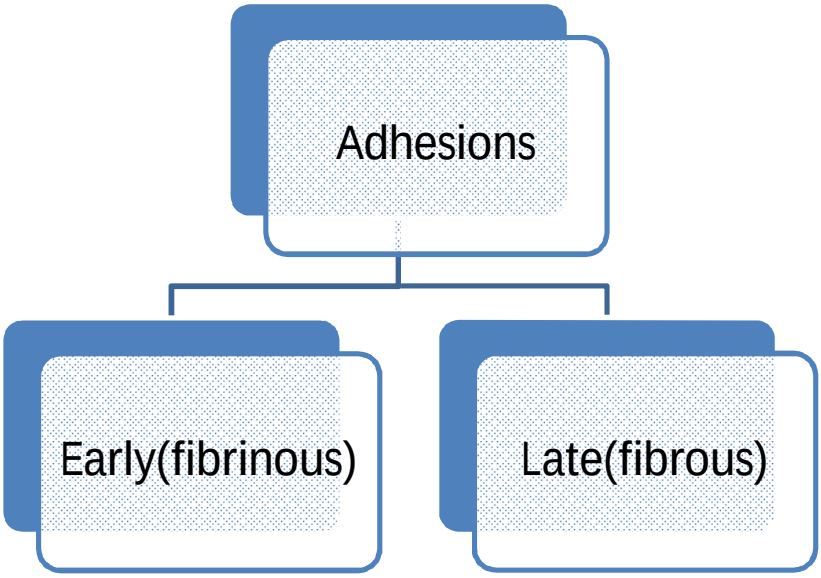
(ii) When the whole loop of the bowel is gangrenous and the condition of the patient permits , resection anastomosis should be done. If the patients condition is poor and the bowel above the strangulation is grossly distended , it is better to exteriorise the bowel and once the patient becomes fit , restoration of the continuity is attempted. Closure of the hernia defect done according to the hernial site.

ADHESIONS AND BANDS

ADHESIONS

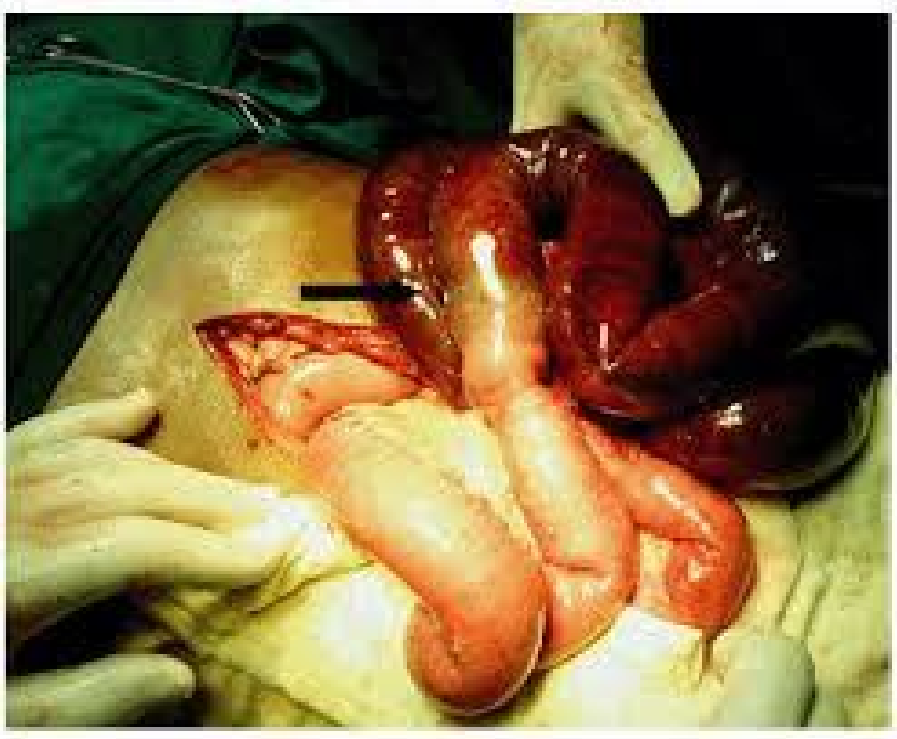
Adhesions accounts for 40% of all intestinal obstructions. Any source of peritoneal irritation will increase the local fibrin production that leads to adhesions between opposed surfaces. In western countries adhesions and bands are the most common cause of intestinal obstruction.





Post operative adhesions giving rise to intestinal obstruction usually involve the lower small bowel. Operations for appendicitis and gynaecological procedures are the most common precursors and are an indication for early intervention.

BANDS

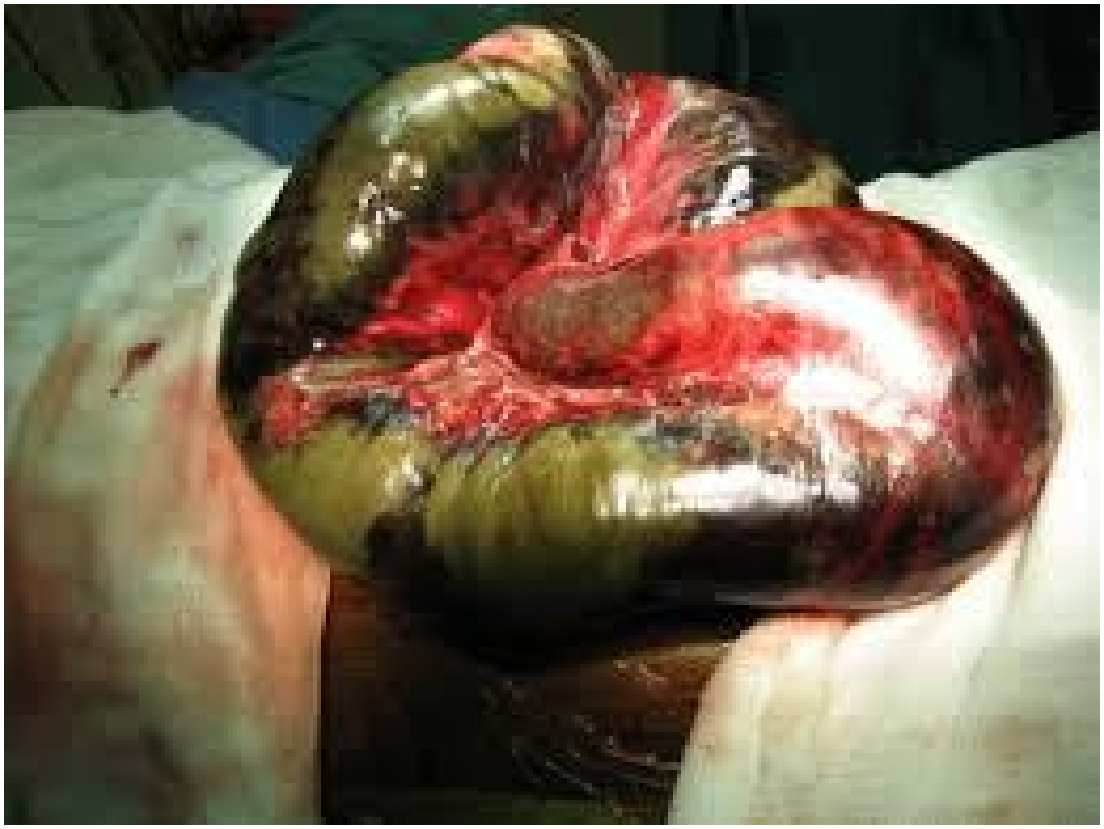


Usually only one band is culpable. This may be

1. Congenital eg obliterated vitellointestinal duct
2. A stream band following previous bacterial peritonitis
3. A portion of greater omentum usually adherent to the parietus.

VOLVULUS

A volvulus is a twisting or axial rotation of a portion of bowel about its mesentery. When complete, it forms a closed loop obstruction with resultant ischemia secondary to vascular occlusion.



Types of volvulus

TYPE	CAUSE	EXAMPLES
Primary	Secondary to congenital malformations of the gut, abnormal attachments of the mesentery, congenital bands	Volvulus neonatorum Caecal volvulus Sigmoid volvulus
Secondary (most common)	Rotation of piece of bowel around an acquired adhesion or stoma	Ileal volvulus due to carcinoid tumour

.

A volvulus may involve small intestine , caecum or sigmoid colon.

The commonest spontaneous type is sigmoid volvulus. Sigmoid volvulus can be relieved by decompression per anum.

Surgery is required to prevent or relieve ischemia.

INTUSSUSCEPTION

Intussusception occurs when one portion of bowel gets invaginated within an immediately adjacent segment almost invariably the proximal into the distal.

This condition is encountered more commonly in children of age 5 – 10 months as peak incidence. 90% cases are idiopathic but may be

associated with upper respiratory tract infection or gastroenteritis. Hyperplasia of Peyer's patches is considered the initiating event. Weaning , loss of passively acquired immunity and common viral pathogens have been implicated in the pathogenesis of intussusception in infancy.

Children with intussusception associated with lead points like Meckel's diverticulum , polyp , duplication , Henoch Schonlein purpura or appendix are usually older than the children with idiopathic disease.

Adult cases are invariably associated with a lead point which is usually a polyp eg Peautz-Jeghers syndrome , submucosal lipoma or an other tumor.

Pathology

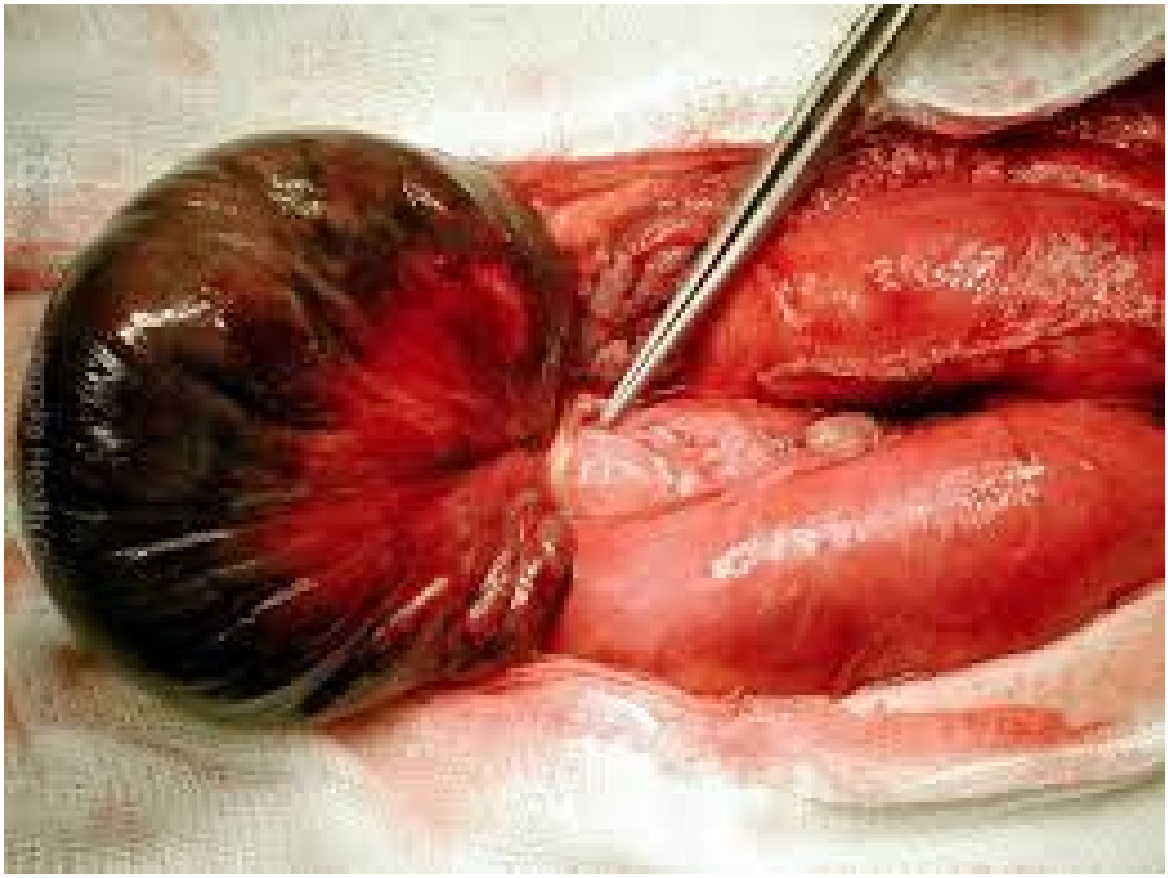
An intussusceptions consists of three parts

- entering or inner tube
- returning or middle tube
- sheath or outer tube (intussuscipiens)

An intussusception is an example for strangulating obstruction as the blood supply of the inner layer is usually impaired. The degree of ischemia depends on the tightness of invagination which is usually greater when it passes through the ileocaecal valve.

In most children the intussusception is ileocolic and in adults , the colocolic intussusception is common.

CLOSED LOOP OBSTRUCTION :



This occurs when the bowel is obstructed at both the proximal and distal ends. It is usually seen in intestinal strangulation and there won't be early proximal intestinal distension. When gangrene of the strangulated segment is imminent, retrograde thrombosis of the mesenteric veins result in distension of the both sides of strangulated segment.

A classic form of closed loop obstruction is malignant stricture of right colon with a competent ileocecal valve. The inability of the distended colon to decompress itself into small bowel results in an increased intraluminal pressure which is greatest at the caecum, with subsequent impairment of the blood supply, if obstruction is not relieved it will end up in necrosis and perforation.

MESENTERIC INFARCTION

Mesenteric vascular disease may be classified as acute intestinal ischemia-with or without occlusion – venous,chronic arterial , central or peripheral.

The superior mesenteric vessels are most likely affected by embolisation or thrombosis with the former being the most common. Occlusion at the origin of superior mesenteric artery is almost invariably the result of thrombosis whereas the emboli gets lodged in the origin of middle colic artery.

Inferior mesenteric involvement is usually clinically silent because of better collateral circulations.

Usually embolus arise from left atrium with fibrillation , mural MI , an plaque(atheromatous) from an aortic aneurysm and a mitral valve vegetation with endocarditis.

Primary thrombosis is associated with atherosclerosis and thrombangiitis obliterans. Primary thrombosis of the superior mesenteric veins may beassociated with factor V Leiden , portal hypertension , portal pyaemia and sickle cell disease and women taking oral contraceptive pills.

Irrespective of whether the occlusion is arterial or venous , hemorrhagic infarction occurs. The mucosa is the only layer of the intestinal wall to have little resistance to ischemic injury.

The intestine and its mesentery becomes swollen and edematous, exudation of hemorrhagic fluid occurs into the peritoneal cavity and bowel lumen.

If the main trunk of SMA is involved the infarction covers an area from just distal to the duodeno jejunal flexure to the splenic flexure. Usually a branch of the main trunk is implicated and the area of infarction is less .

Clinical features

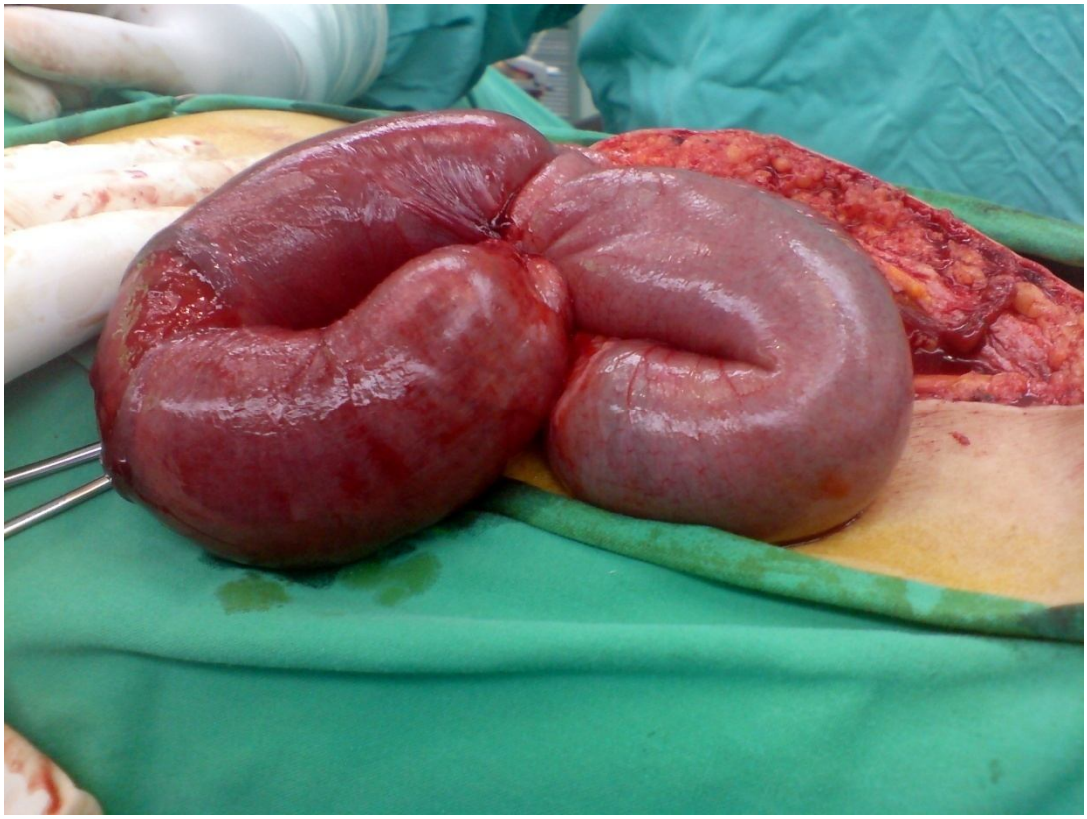
- abdominal pain – sudden onset in a patient with atherosclerosis or atrial fibrillation. The pain is central and out of proportion to clinical signs.
- In early stage, persistent vomiting and defaecation occur, with subsequent passage of altered blood .
- Hypovolemic shock occurs rapidly.
- Initially abdominal tenderness is mild and rigidity occurs late.

Investigations shows profound neutrophilic leukocytosis with absence of gas in the thickened small intestine on abdominal radiographs. The presence of gas bubbles in the mesenteric veins is rare but pathognomonic. Infarction of the large intestine alone is rare.

SMALL BOWEL VOLVULUS:

The cause of small bowel volvulus is either primary or secondary.

Primary small bowel volvulus :



The etiology is still poorly understood, several etiological factors are suggested.

Diet : : It may occur by taking large quantities of fibre after prolonged fasting.

Gut motility : Increased small bowel motility has a role in the etiology. Parasitic infestations alters the gut motility, increased

concentrations of 5 hydroxy tryptamine , a known stimulant of gut motility. SBV may occur in diabetic autonomic neuropathy.

The suggested mechanism behind the SBV is that a bulky bolus of food enters the proximal jejunum causing the loop of jejunum to descend into the pelvis. This displaces empty small bowel loop upwards initiating the rotation of the mesentery and leads on to volvulus.

For this to occur there must be a combination of long small bowel attached to a broad based fat free mesentery(which splints the bowel), very firm abdominal muscles (restricting the bowel movement to the coronal plane) and a diet with an exceptionally high bulk, eaten rapidly on a empty stomach.

Secondary small bowel volvulus :

The most frequently related conditions are bands, adhesions , Meckel's diverticulum , internal hernias, Ascariasis, pregnancy, ileal atresia, meconium ileus , enteroenterostomy, leiomyoma of the mesentery and surgeries like gastrostomy , gastrectomy.

PATHOPHYSIOLOGY :

In volvulus , a dangerous form of bowel obstruction called closed loop obstruction occurs in which a segment of intestine is obstructed both proximally and distally. In such cases , the accumulating gas and fluid cannot escape either proximally or distally from the obstructed segment , leading to rapid rise in luminal pressure, and a rapid progression to strangulation.

CLINICAL FEATURES :

SBV presents with the classical features of intestinal obstruction. The outstanding symptom is central abdominal pain, the severity of which may be out of proportion to the apparent degree of obstruction. The diagnosis should be particularly considered if the pain does not respond to narcotic analgesia, although in such cases frank gangrene is often already present. Associated with vomiting, distension, tenderness , peritonism, absent bowel sounds , elevated temperature, tachycardia and leukocytosis.

RADIOLOGICAL INVESTIGATIONS :

On barium studies , the small bowel loops may show typical ‘corkscrew’ or ‘spiral’ pattern suggesting SBV. On angiography , spiralling

of the branches of the twisted superior mesenteric artery produces a “barber pole” appearance that suggests the diagnosis. On CT or MRI characteristic findings include the ‘whirl’ sign of the rotated mesentery and ‘peacock tail’ sign due to torsion of bowel around mesenteric axis. Small bowel ischemia is suggested on ct by the presence of bowelwall thickening, intramucosal air and intraperitoneal fluid.

TREATMENT :

The surgical operations for SBV consists of derotation , with or without fixation, resection and anastomosis. In view of the excellent blood supply of the small bowel, some authors recommend resection and anastomosis in all cases of SBV regardless of whether gangrene is present or not

POST OPERARIVE COMPLICATIONS

Major complications

1. Anastomotic leak
2. Wound dehiscence
3. Septicaemia
4. Renal failure
5. Multi organ failure

Minor complications :

1. Faecal fistula
2. Prolonged ileus
3. Wound infection
4. Intra abdominal abscess
5. Respiratory tract infection
6. Urinary tract infection
7. Deep vein thrombosis

MATERIALS AND METHODS

In this study, the estimation of serum LDH was conducted in 45 cases of acute intestinal obstruction who were admitted in surgical wards of CMCH during the period of September 2014 to August 2015.

LDH was estimated in all the cases preoperatively on the day of admission.

INCLUSION CRITERIA :

1. Patients presenting with features of acute intestinal obstruction.
2. Patients with irreducible inguinal/femoral hernia.
3. Patients with features of intestinal obstruction diagnosed preoperatively as a case of SMA/SMV occlusion.

EXCLUSION CRITERIA:

1. Patients < 18 years and >80 years.
2. Pregnant women
3. Psychiatric patients.

All the patients eligible by inclusion and exclusion criteria were included in the study. All the cases were thoroughly examined. Time of presentation and onset of symptoms were noted.

Serum LDH was estimated in all the patients at the time of admission and the estimated value of serum LDH is compared with the viability of the bowel intra operatively.

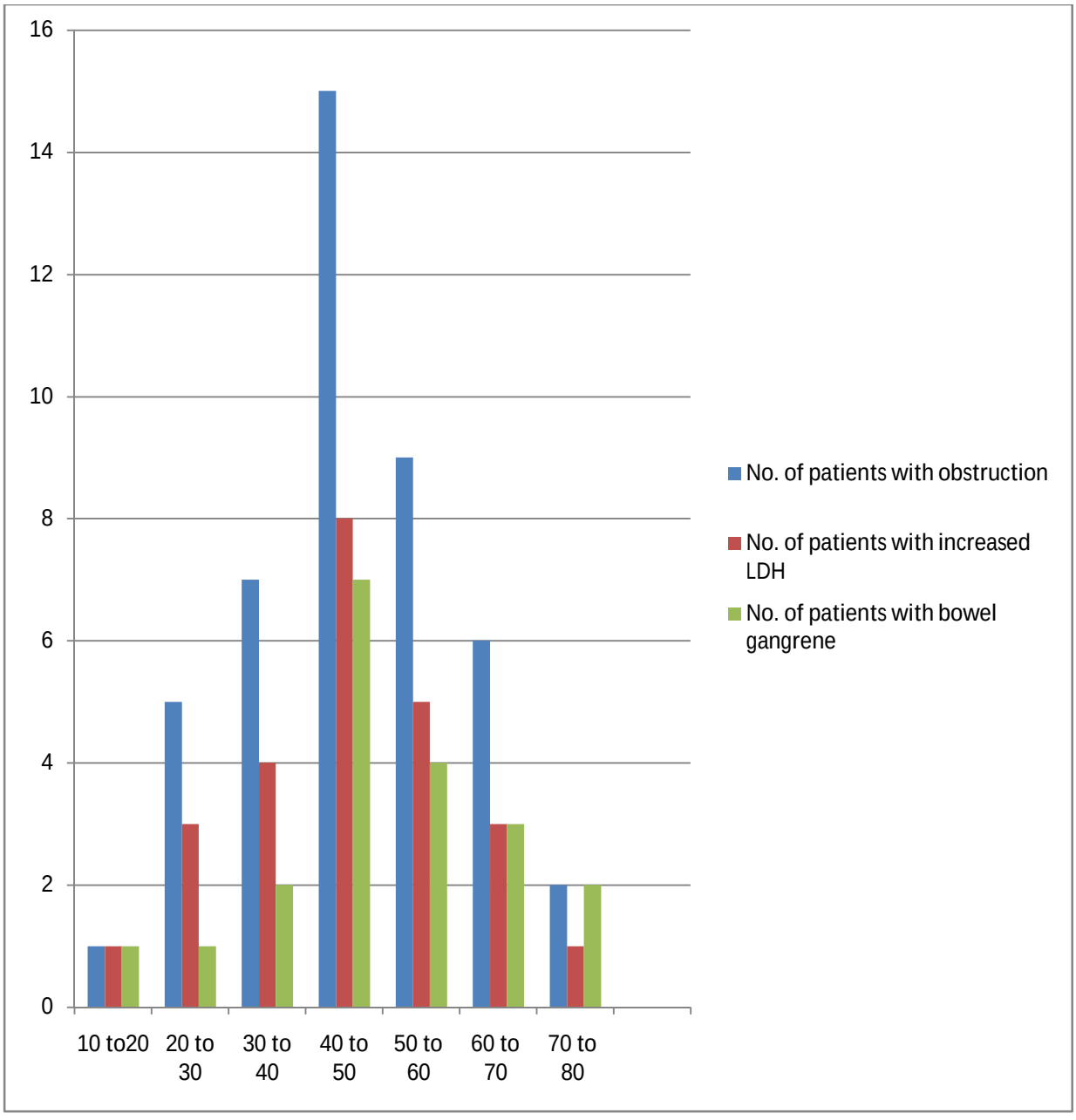
OBSERVATION AND RESULTS

The total number of patients presented with obstruction is 45 out of which 25 were found to have elevated LDH which is about 55.56%. Out of 25 patients presented with elevated LDH, 20 patients found to have gangrenous bowel. The age incidence in this study was between 19yrs to 80yrs.

AGE INCIDENCE

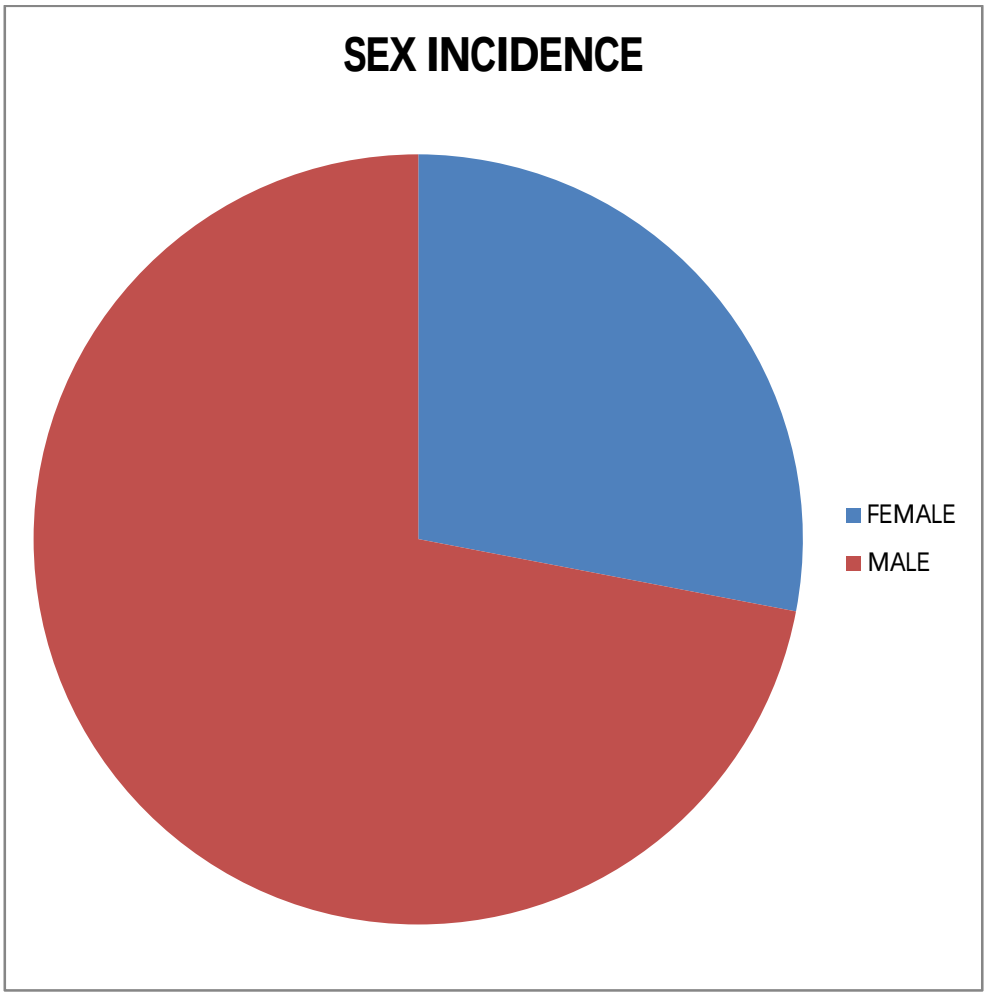
AGE	NO. OF PATIENTS WITH OBSTRUCTION	NO. OF PATIENTS WITH ELEVATED LDH	NO. OF PATIENTS WITH GANGRENOUS BOWEL
10-20	1	1	1
20-30	5	3	1
30-40	7	4	2
40-50	15	8	7
50-60	9	5	4
60-70	6	3	3
70-80	2	1	2
TOTAL	45	25	20

AGE INCIDENCE



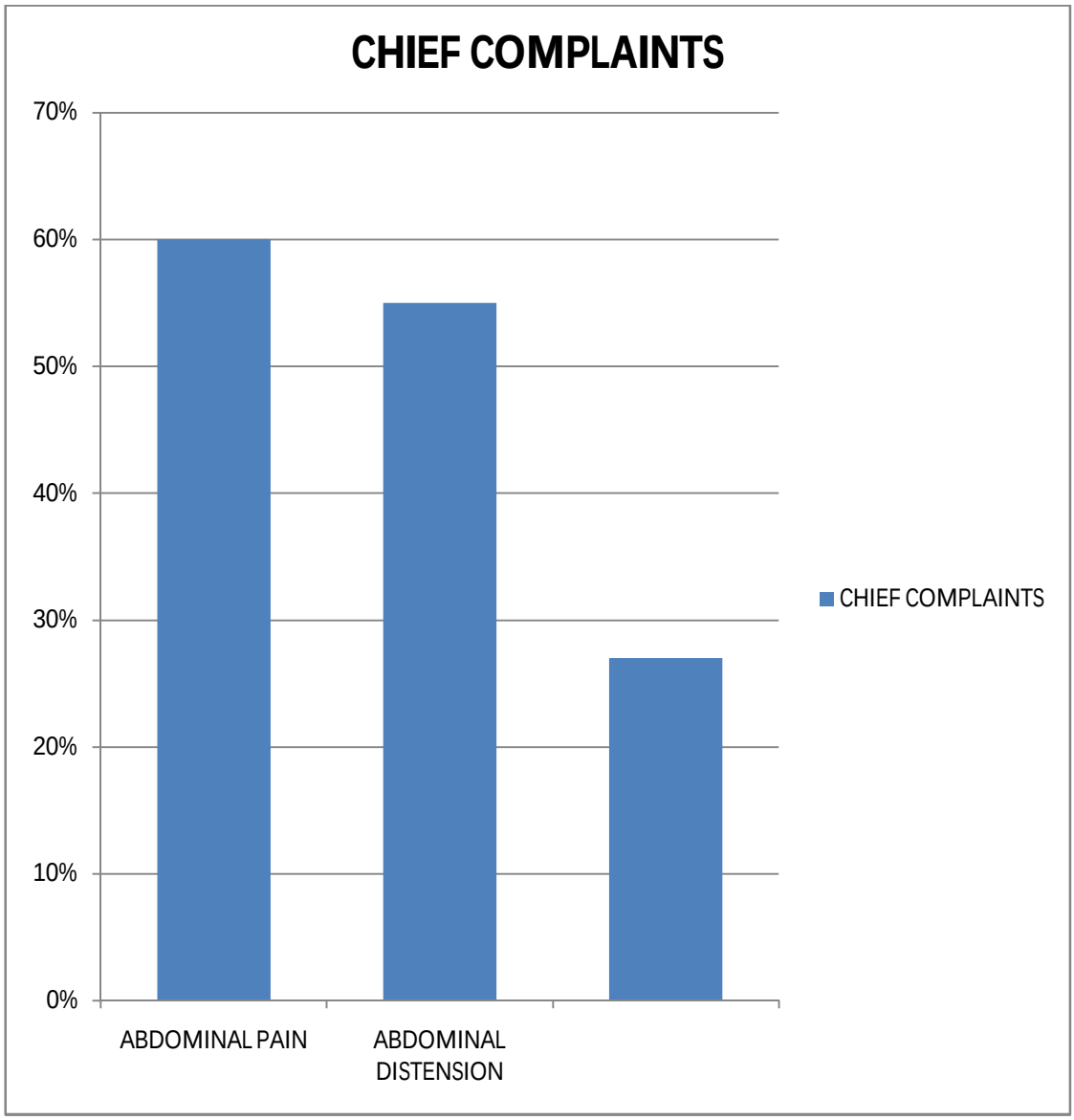
SEX INCIDENCE

Males are predominantly affected with male 86% and female 14%.



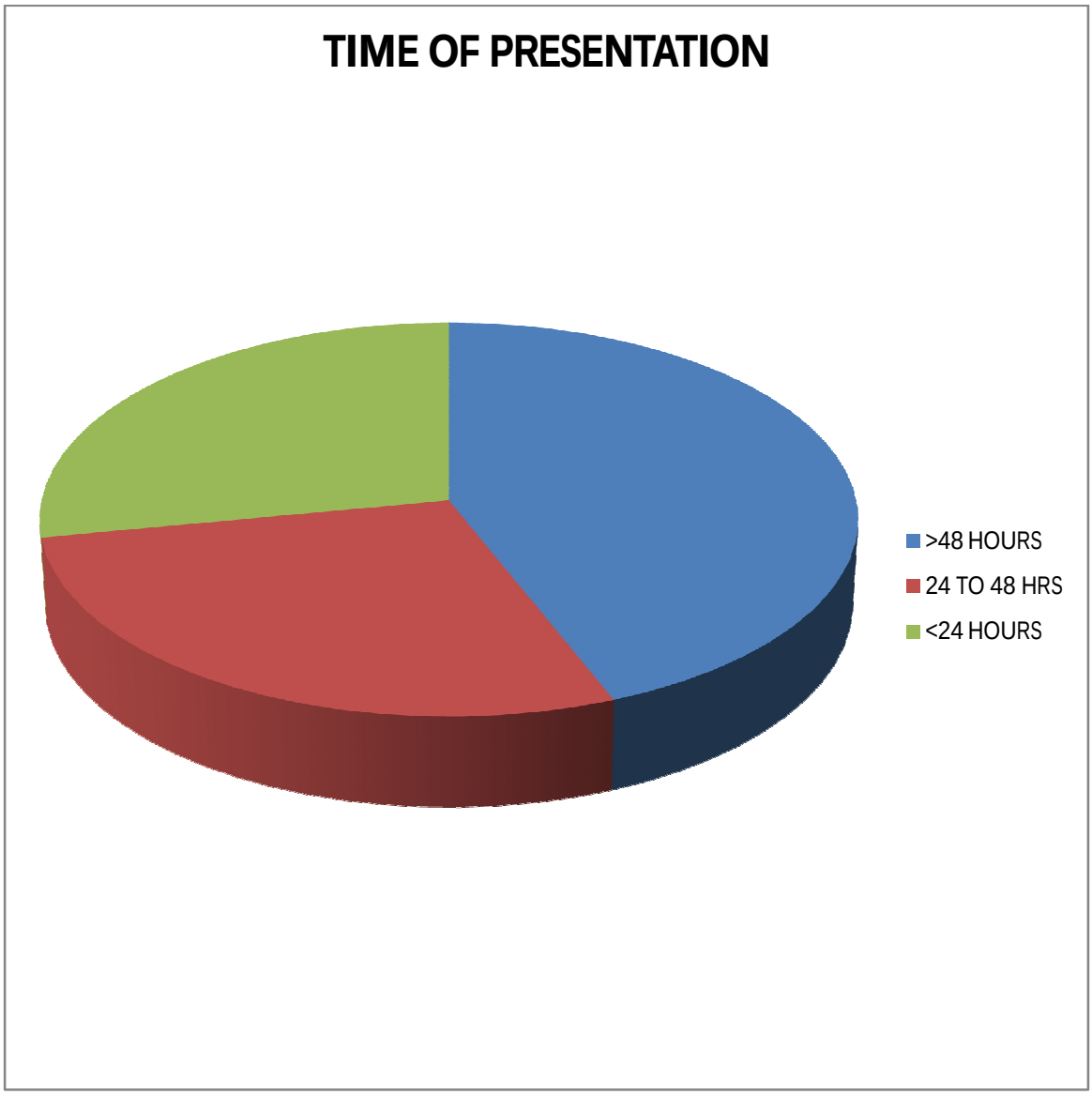
CHIEF COMPLAINTS

Most commonly presented with abdominal pain 60 % and irreducible swelling 40%.



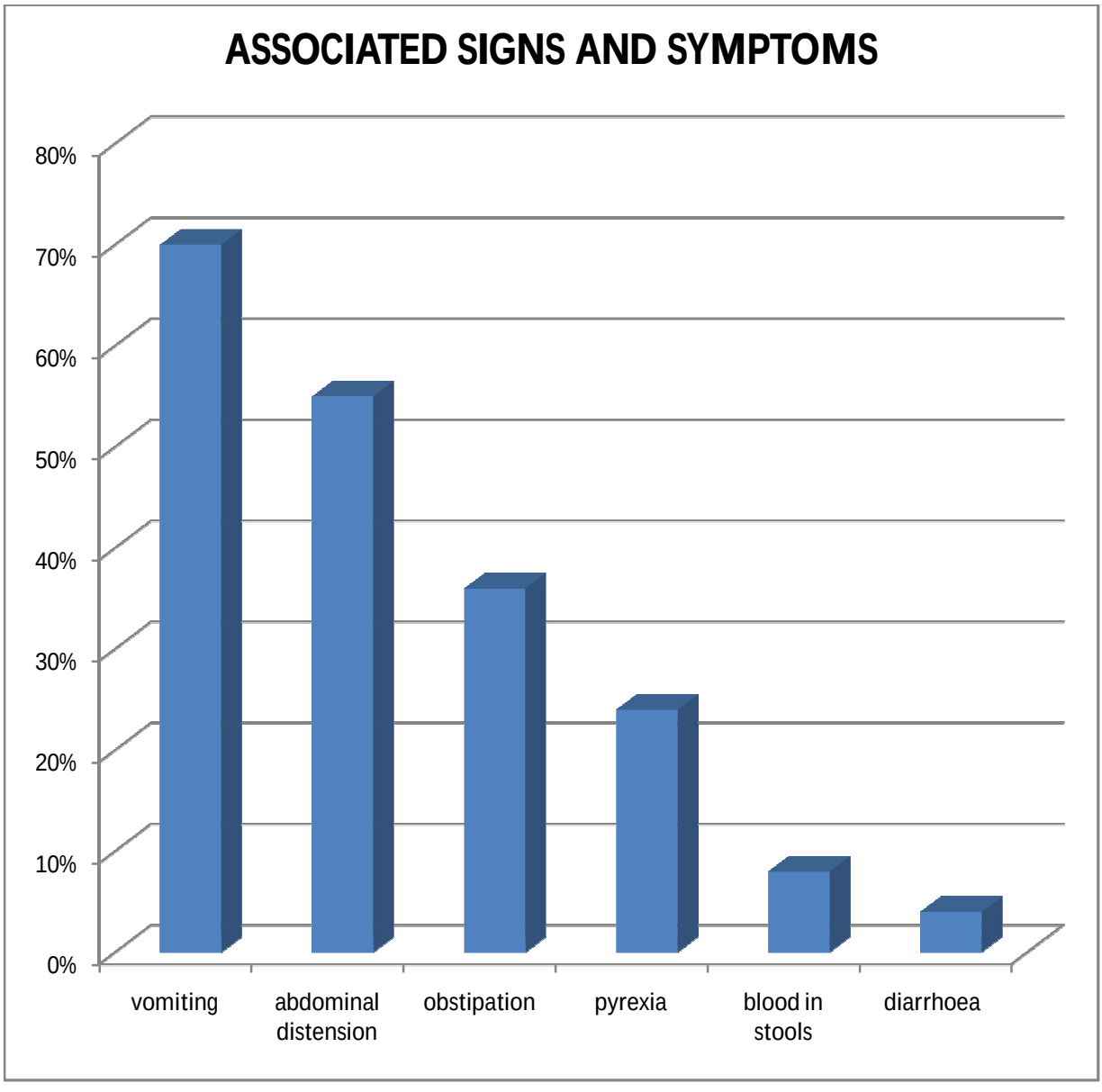
DURATION OF SYMPTOMS

44% of patients presented to the hospital >48 hours of onset of symptoms, 28% in 24 to 48 hours and 28% in <24 hours.



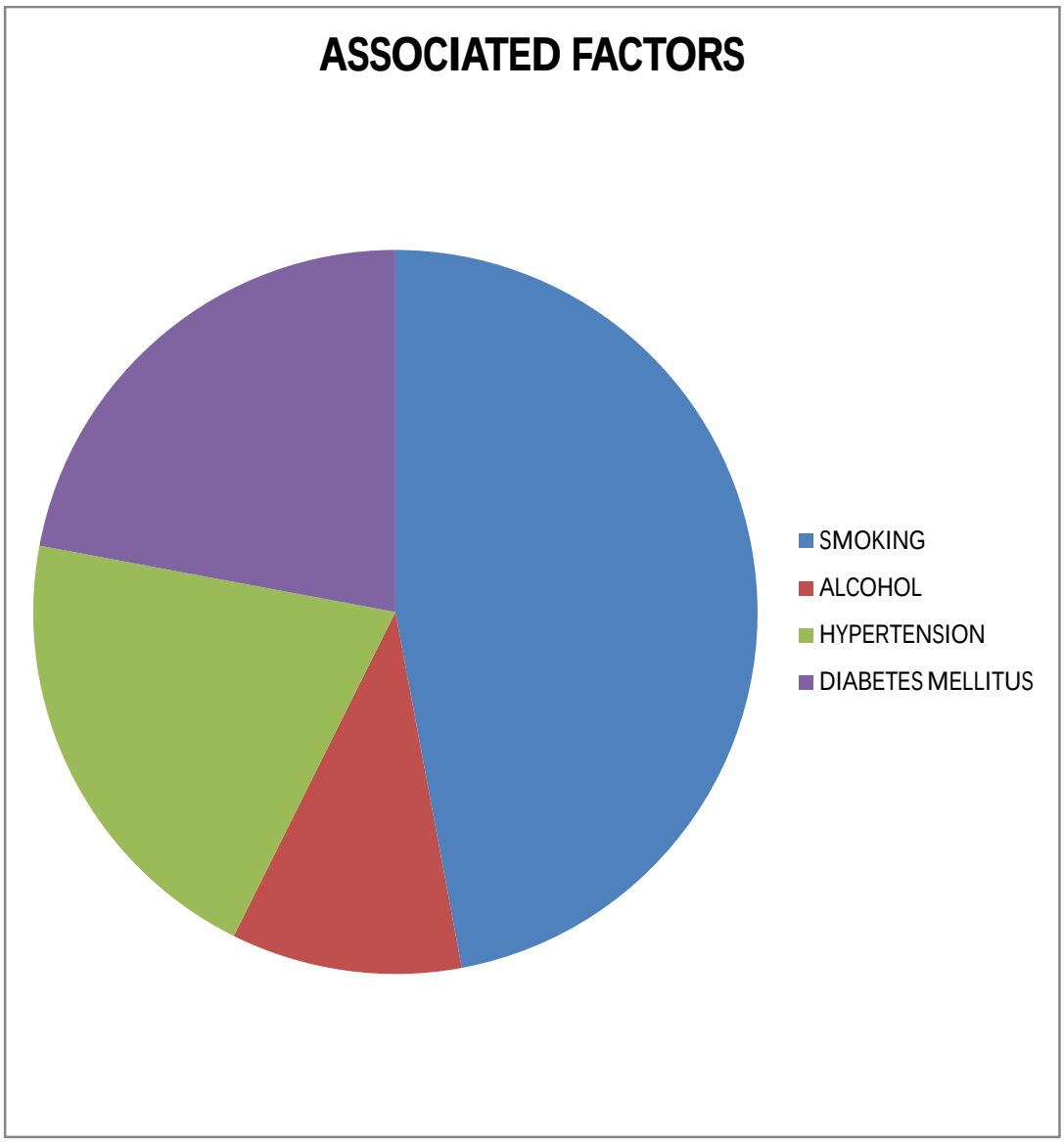
ASSOCIATED SYMPTOMS AND SIGNS

Most commonly pain associated with vomiting 84% followed by abdominal distension 40%, obstipation 36% , pyrexia 24%, blood in stools 8%, diarrhoea 4%.



ASSOCIATED FACTORS

Most commonly associated factor out of 25 cases with gangrene is smoking 64% , alcohol 14%, hypertension 28%,diabetes mellitus 30%.

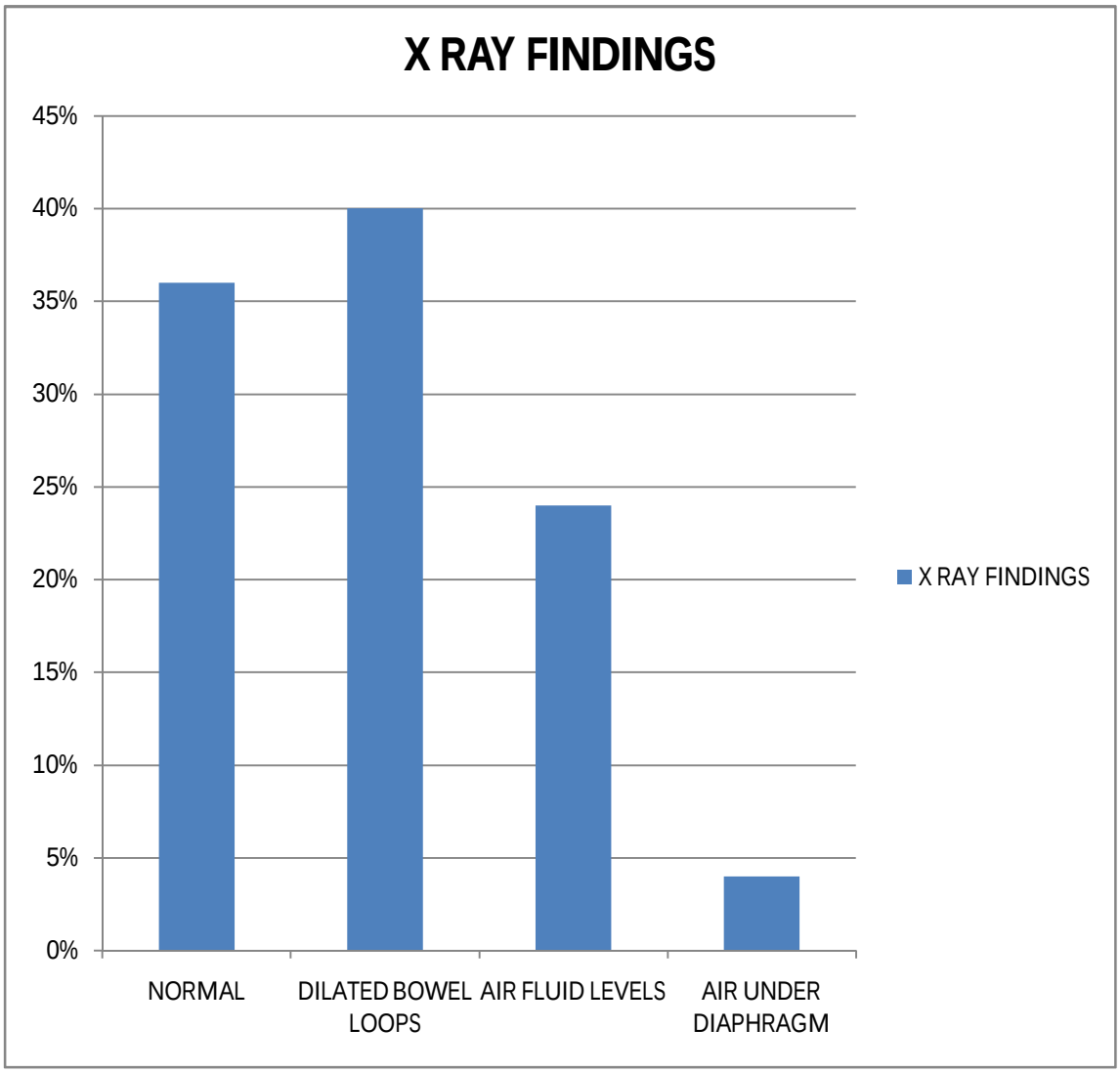


PER RECTAL EXAMINATION

76% normal faecal staining , 12% blood stained faeces , 8% empty.

X RAY ABDOMEN

Done in all patients. 36% no significant finding, 40% dilated bowel loops , 24% air fluid levels, 4% air under diaphragm.

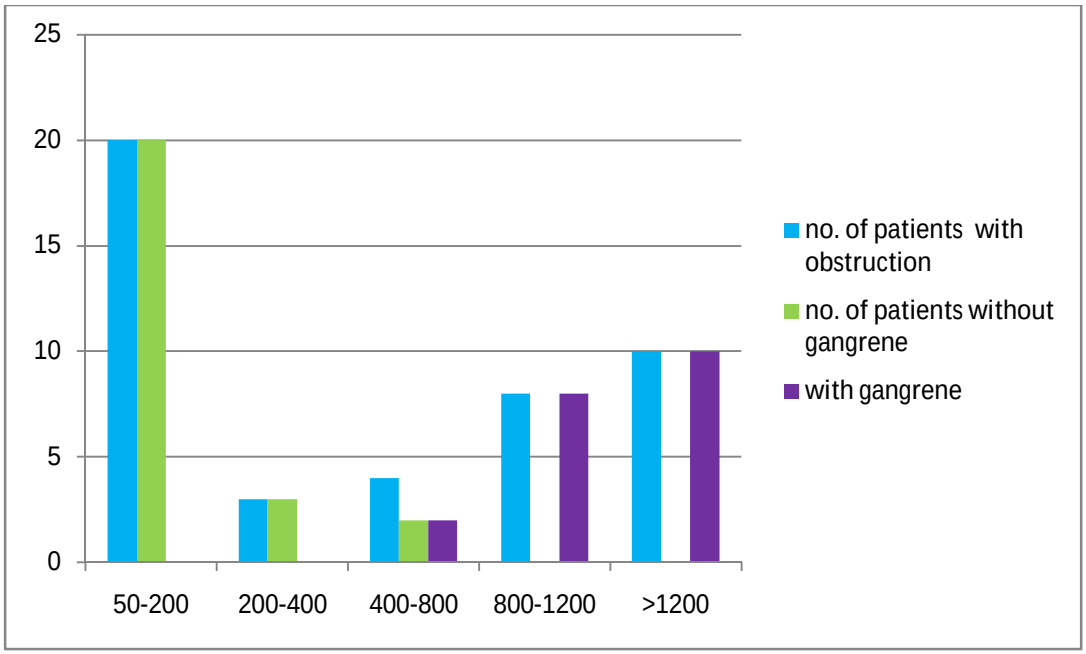


ETIOLOGY

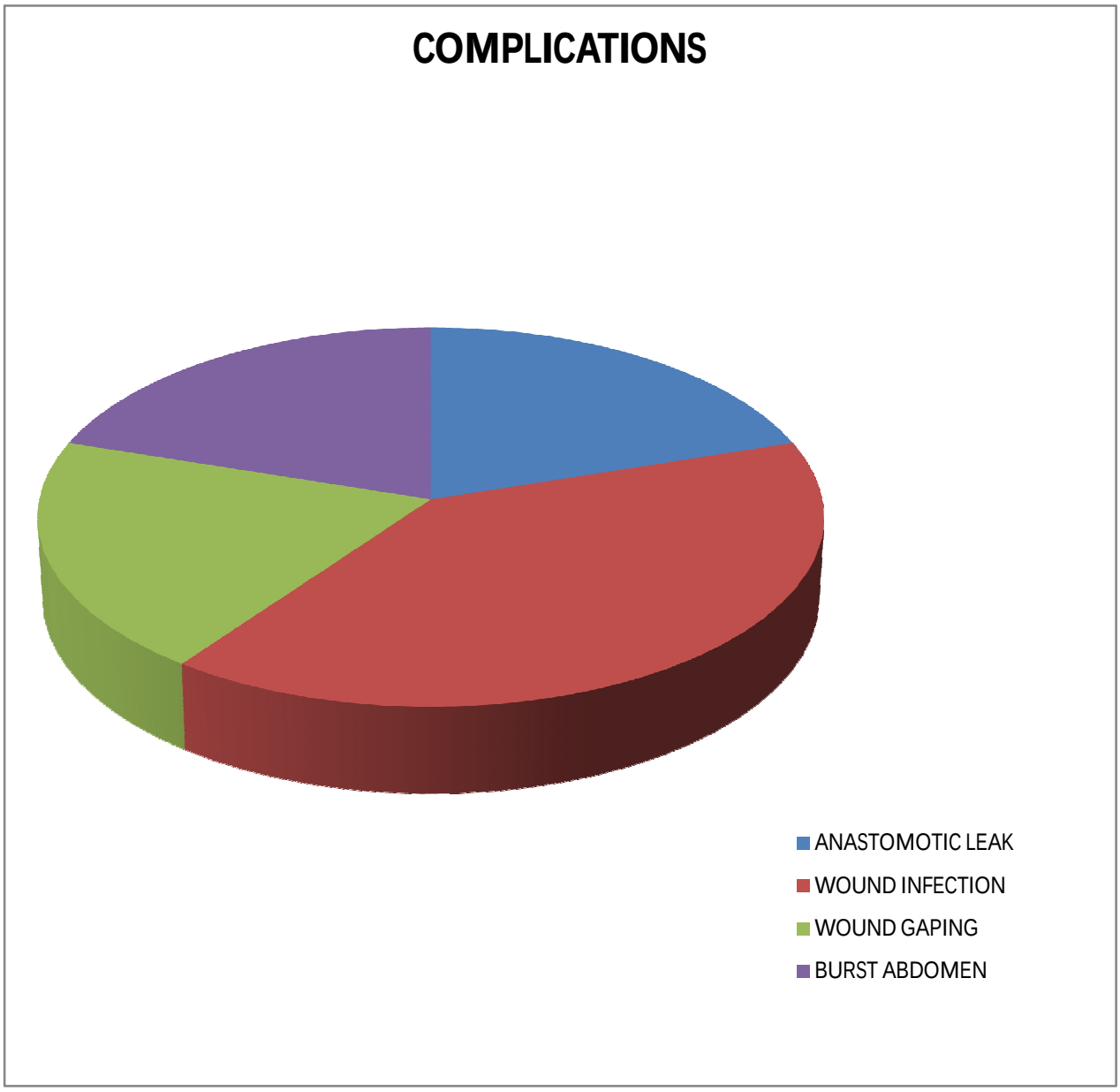
ETIOLOGY	No. OF CASES	No. OF CASES WITH INCREASED LDH	NO OF CASES WITH GANGRENOUS BOWEL
Adhesive intestinal obstruction	23	9	7
Strangulated inguinal hernia	8	6	4
SMA occlusion	6	4	4
Post operative constriction bands	5	3	2
Strangulated femoral hernia	2	2	2
SMV occlusion	1	1	1
TOTAL	45	25	20

LACTATE DEHYDROGENASE LEVELS IN VARIOUS PATIENTS:

The normal range of serum LDH is 50 to 200 IU/L.



Among 45 patients presented with obstruction 20 patients had LDH level between 50 to 200 and all the 20 patients had normal bowel viability and 3 patients had LDH level between 200 to 400 and all the three patients had normal bowel viability. 4 Patients had LDH level between 400 to 800 and two of them had normal bowel viability and two others had bowel gangrene. 8 Patients had LDH level between 800 to 1200 and all of them had bowel gangrene. 10 Patients had LDH level more than 1200 and all of them had bowel gangrene.



COMPLICATIONS

Anastomotic leak 8%, wound infection 16%, wound gaping 8%, burst abdomen 8%.

POST OPERATIVE FOLLOW UP

All the patients were given broad spectrum antibiotics and injection metronidazle for 7 days. 5 patients developed wound infection and were treated according to the wound culture and sensitivity.

Daily examination of laparotomy wound and drain site done. All patients catheterised until adequate output maintained and continuous RT aspiration done until <20 ml aspirated for 24 hours.

One patient required mechanical ventillatory support and extubated on first POD. The patients with systemic hypertension and diabetes mellitus managed accordingly.

DISCUSSION

Gangrenous bowel recognized as a potential cause of mortality. It occurs due to various causes. This study was undertaken with a notion to identify the patients with features of acute intestinal obstruction who are developing bowel gangrene at the earliest by estimating the serum LDH preoperatively

The level of lactate dehydrogenase elevates during an inflammatory process due to cellular function alterations and damage to the cells. The cell membrane permeability becomes altered due to ischemia and hypoxia and LDH is released into the circulation.

The normal level of serum LDH is 50 to 200 IU/L.

Out of 45 patients 25 patients had elevated serum LDH which is about 55.56% and among those 25 patients, 20 patients found to have bowel gangrene which is about 80% . In our study the mean value of elevated serum LDH in bowel gangrene patients is 1037 IU/L.

The youngest patient is 19 years old who presented with post operative constriction band causing gangrene of ileum. The oldest patient is 80 years old and presented with strangulated inguinal hernia.

The peak incidence is in the fifth decade with 33.3% and the most common cause of obstruction is found to be adhesive intestinal bands which are about 49% and among those 22 patients 8 patients had elevated LDH which is about 36.4%. Among those 8 patients, 6 patients found to have gangrene intraoperatively which is about 75%. 8 patients presented with strangulated hernia which is about 17.8% and among those 8 patients, 6 patients had elevated serum LDH which is about 75%.

And among those 6 patients, 4 patients were found to have gangrenous bowel which is 66.7%. 6 patients were found to have SMA occlusion and 4 patients among them were found to have elevated serum LDH. All the 4 SMA occlusion patients with elevated serum LDH were found to have gangrenous bowel, which is 100%. 4 patients were found to have post operative constriction band among whom 2 patients had elevated serum LDH which is 50%. Among those 2 patients with elevated serum LDH, one patient had bowel gangrene which is 50%.

2 patients presented with strangulated femoral hernia and both of them had elevated LDH and gangrene .

The incidence of bowel obstruction and gangrene is more common in males(72%) than females(28%).

Strangulated hernia occurs commonly in males than females due to increased physical activities and strenuous efforts.

Mesenteric vessel occlusion is also common in males than females may be due to thrombogenic factors like smoking and alcoholism.

The delayed time of presentation shown to influence the morbidity and mortality. Increased morbidity is seen in gangrene patients due to undue delay in presentation due to prolonged exposure of bacterial toxins leading to the onset of septicaemia.

Previously, an elevated level of serum LDH is considered as a indicator to assess the time of onset of myocardial infarction. Recently, an elevated level of serum LDH is visualised in patients who had bowel gangrene.

According to Muchas, an elevated level of LDH is found in 86% of patients who had bowel gangrene.[32]

"Lactate dehydrogenase is one of the markers of intestinal ischemia".

- *Thompson.*[33]

A study conducted by Lange.H, Jackel.R revealed an inference of 100% sensitivity and 42% specificity for increased LDH in patients presenting with acute abdomen who later are found to have intestinal ischemia and gangrene[34].

Dr Neil R Feins suggested that the level of LDH can be taken as a criteria for intestinal obstruction [35].

CONCLUSION:

From this study it is evident that ischemic changes in any part of the bowel can cause elevation in the serum levels of LDH and a higher value of >1000 IU/L strongly indicates an underlying gangrenous change.

It is a less invasive , cost effective and easily available diagnostic tool to diagnose bowel ischemia/gangrene. Hence it is more useful in centres where the diagnostic facilities are limited.

The pre operative estimation of serum LDH levels in patients presenting with features of acute intestinal obstruction helps in identifying the patients who undergo intestinal ischemia and gangrene at the earliest which makes an early intervention possible and helps in reducing the morbidity and mortality due to bowel gangrene.

Early diagnosis and early intervention is the key to reduce the mortality rate due to bowel gangrene.

This study was conducted in Coimbatore medical college hospital and the results are similar and comparable to the other studies.

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PROFORMA

Name: Age :

Sex:

Religion : Address :

Ip no. : case no. ;

Date of onset of symptoms :

Date of admission :

Date of operation :

Date of discharge :

PRESENTING COMPLAINTS :

1. Abdominal pain
2. Vomiting
3. Abdominal distension
4. Bowel - tennessmus, constipation

DETAILED HISTORY OF PATIENT

1. PAIN

a. Time and mode of onset :
b. Situation :
c. Character : type at onset/ type at present

Colicky / burning/ throbbing

Severe agonising pain

d. Progress :

e. Radiation :

f. Related to bowel and micturition

g. Relation to food

2. Vomiting

a. Present/absent

b. Character : projectile/regurgitation

c. Frequency

d. Quality - nature

- Color
- Odour
- Taste

e. Quantity

f. Associated materials with vomitus

g. Duration - continuous or intermittent

h. Progress

i. Relation to pain

j. Haematemesis : present/absent

- Colour
- Frequency
- Amount

3. Abdominal distension:

a. Mode of onset: gradual / sudden

b. Site

c. Duration

d. Progress

4. Bowels

a. Tenessmus

b. Constipation

c. Diarrhea

d. Dysentery

e. Melena

5. Fever since _____ days continuous/intermittent/remittent

6. Micturition

PAST HISTORY

a. Previous history of similar attacks

b. Any previous abdominal surgery

c. Hypertension

d. Diabetes mellitus

e. Tuberculosis

f. Jaundice

g. Epilepsy

h. Drug allergy

FAMILY HISTORY

a. Members - any family members suffering from similar illness

b. Health status

PERSONAL HISTORY

a. Diet - vegetarian/mixed

b. Habits

Smoking Alcohol

- Betel nut
- Bowel and bladder habits

- Sleep
- Appetite

MENSTRUAL / OBSTETRIC HISTORIES IN FEMALES

SOCIO ECONOMIC HISTORY

GENERAL EXAMINATION

a. Build

b. Nourishment

c. Eyes

d. Mouth

e. Nails

f. Cyanosis

g. Pedal oedema

h. Lymphadenopathy

i. Pulse

j- BP

k. Respiration

l. Temperature

m. Jaundice

n. Dehydration

LOCAL EXAMINATION

1. Abdomen

a. **Inspection**

(i) Shape

(ii) Movements

(iii) Umbilicus

(iv) Distension : local/ general

(v) Visible peristalsis

(vi) Visible mass

- Size
- Shape
- Surface
- Borders
- Impulse on coughing

(vii) Renal angle fullness +/-

(viii) Hernial orifices

b. **Palpation**

(i) Local rise of temperature

(ii) Tenderness

(iii) Swelling

- Situation
- Shape
- Kxtent
- Surface
- Consistency
- Movement

c. **Percussion**

d. **Auscultation**

2. External genitalia

3. P/R examination and P/V examination in females

4. Systemic examination

    a. Respiratory system

    b. CardiovascuVar system

    c. Central nervous system

5. Provisional diagnosis

6. Findings for diagnosis

7. Investigations

    (1) Blood investigations

        a. Serum LDH

        b. Hb%

        c. Blood grouping

        d. TC,DC,ESR

        e. Blood urea, serum creatinine

        f. Random blood sugar

        g. serum electrolytes

(2) Urine examination

    a. Albumin

    b. Sugar

    c. Microscopy

(3) Radiological investigations

    a. Plain X ray of abdomen

b. Contrast studies

c. Other relevant diagnostic investigations

d. Ultrasound

e. CT scan

f. Arteriography

g. Laparoscopy

h. 4 quadrant aspiration

i. Stool examination

(4) Final diagnosis

(5) Treatment

a. Treatment during resuscitation

b. Operation

c. Operative findings

d. Preoperative diagnosis

e. Post operative diagnosis

f. Post operative treatment

g. Post operative complications

h. Advice on discharge

i. Conclusion

j. Biopsy report

(6) Follow up

First follow up(1st week)

Second follow up(2nd week)

Third follow up (3rd week)

CONSENT FORM

Name :

Age :

Sex :

vdf;F ta;wpy; Fly;mi lgg[VwglL ,Ugg ija;k;mjw;F mWi trpfpr;ir bra;ant z;Lk; vdgija;k; kUj;J th; Tw mwpnt d; nkYk; vdf;F mWit rpfpr;ir bra;J bfhs;s t;k; mjw;F Kd;dh; ,uj;j ghpnrhjid bra;J bfhs;s t;k;rk;k jk;

Signature and name of the Vounlteer **Signature and Name of Witness**

Signature of the investigator :

Date :

Place : Coimbatore

PATIENT CONSENT FORM

STUDY: A STUDY ON RAISED SERUM LDH LEVEL IN ACUTE INTESTINAL OBSTRUCTION – A MARKER OF BOWEL GANGRENE COIMBATORE MEDICAL COLLEGE HOSPITAL

This study has been explained to me in my own language and I understood the following

1. What the study involves
2. That the refusal to participate will not affect my treatment in any way
3. That I may withdraw to take part in this study

Signature of the patient: Full name of the patient: Address: Date:

Witness: (should be a person not connected with the study)

I have been present while the procedure to be performed has been explained to the patient and I have witnessed his/her consent to take part.

Signature of the witness:

Full name of the witness:

Address:

Date:

MASTER CHART

Sl.NO	AGE	SEX	IP.No	ABDOMINAL PAIN	PALPABLE SWELLING	DURATION(HR)	DIABETIC	SHT	SMOKER	ALCOHOL	VOMITING	OBSTIPATION	DIARRHEA	AB.DISTENSION	GUARDING/RIGIDITY	TENDERNESS	BOWEL SOUNDS	ETIOLOGY	Sr.LDH	GANGRENE
1	64	M	12054	+	+	<48	+	-	+	-	+	+	-	+	+	+	+	SIH	1390	+
2	73	F	23843	-	+	48	-	-	-	-	+	+	-	+	+	+	+	SFH	950	+
3	35	M	65647	+	-	>24	-	-	+	+	+	-	-	+	+	+	+	AIO	896	+
4	40	M	30254	+	-	24	-	-	-	-	+	-	-	+	-	+	+	PCB	186	-
5	43	F	32865	+	-	24	-	+	-	-	-	+	-	+	-	+	+	AIO	158	-
6	48	M	23843	+	-	48	+	+	+	-	+	-	-	+	-	+	-	SIH	175	-
7	55	M	53678	-	+	24	+	-	-	+	+	+	-	+	+	-	-	AIO	470	-
8	58	F	50113	-	-	>48	+	-	-	-	+	-	-	+	-	+	+	SMA OCC	674	+
9	48	M	50568	+	-	>48	-	-	+	-	+	+	+	+	-	-	+	AIO	1224	+
10	55	M	30249	-	-	24	+	+	-	+	+	-	-	+	+	-	+	SIH	189	-
11	28	M	11496	-	+	24	-	-	+	-	-	-	-	+	+	-	-	AIO	184	-
12	36	F	12303	-	-	24	-	-	-	-	+	-	-	+	-	-	+	PCB	113	-
13	19	M	32007	+	-	>48	-	-	-	-	+	+	-	+	-	+	-	AIO	1326	+
14	63	M	45791	+	-	48	+	+	+	-	+	+	-	+	+	-	+	SIH	814	+
15	49	M	13616	+	-	24	-	+	+	-	+	-	-	+	+	+	+	AIO	723	
16	66	F	12004	+	+	48	+	+	-	-	+	+	+	+	+	+	+	AIO	1264	+
17	37	M	12415	+	-	24	+	+	-	+	+	-	-	+	+	+	-	SMA OCC	161	-
18	58	M	15939	+	+	48	-	-	+	-	+	-	+	+	+	+	+	PCB	1336	+
19	51	F	15320	+	-	>24	-	-	-	-	+	-	-	+	-	+	-	AIO	886	+
20	44	M	16828	+	+	24	+	+	-	-	+	-	-	+	-	+	+	PCB	197	-
21	28	M	34566	+	+	48	-	-	+	-	+	+	-	+	-	-	+	AIO	198	-
22	50	M	27701	+	-	24	+	+	+	+	-	+	-	+	-	+	+	SIH	924	+
23	54	M	28397	-	+	24	-	+	-	+	+	+	-	+	+	-	+	AIO	109	-

24	30	F	16829	+	+	24	-	-	-	-	+	+	-	+	+	-	-	AIO	163	-
25	46	M	67001	+	+	24	-	+	+	+	+	+	-	+	+	-	+	SMA OCC	195	-
26	52	M	63202	+	+	<24	+	+	-	-	-	+	+	+	+	+	+	AIO	567	-
27	35	F	16302	+	-	<24	-	-	+	-	+	+	+	+	-	-	+	AIO	145	+
28	65	M	43571	+	+	24	+	+	+	+	+	-	-	+	-	+	+	SIH	160	+
29	33	M	41578	-	-	>24	-	-	+	-	+	-	-	+	+	-	+	AIO	824	-
30	80	F	19572	+	+	48	+	+	-	-	+	+	-	+	+	+	-	AIO	892	+
31	36	M	45054	+	+	<24	-	-	-	+	+	-	-	+	+	+	+	SIH	96	-
32	46	M	49978	-	-	48	-	+	-	-	+	+	-	+	+	+	+	AIO	1287	+
33	52	F	35684	+	+	24	+	+	-	-	+	-	-	+	--	+	+	SMA OCC	561	-
34	55	M	64874	+	+	48	-	+	+	+	-	-	-	+	+	-	+	AIO	1552	+
35	50	M	37053	+	+	24	+	-	+	-	-	+	+	+	+	-	-	AIO	103	-
36	44	F	65969	+	+	48	-	+	-	-	+	+	-	+	+	-	+	AIO	786	+
37	38	M	10124	+	+	24	-	-	-	+	-	+	-	+	-	-	+	SMA OCC	292	-
38	48	M	13616	+	+	>48	-	+	+	+	+	+	-	+	-	+	+	AIO	1508	-
39	43	M	12415	+	+	24	-	-	+	-	+	-	-	+	-	-	+	AIO	326	-
40	72	F	15939	-	-	24	+	-	-	-	-	-	-	+	-	+	-	SMA OCC	179	+
41	49	M	54573	+	+	24	+	+	+	+	+	+	-	+	+	-	+	SFH	794	-
42	76	F	45791	-	-	>48	+	-	-	-	+	+	-	-	+	+	-	PCB	987	+
43	37	M	58396	+	+	24	-	-	-	+	+	-	+	-	-	-	-	PCB	384	-
44	57	M	44555	+	+	>48	-	+	+	-	-	-	-	+	+	+	+	SMA OCC	1278	+
45	72	M	51263	-	+	>48	+	-	-	+	+	+	+	+	-	-	+	SIH	1392	+

SIH- STRANGULATED INGUINAL HERNIA
AIO- ADHESIVE INTESTINAL OBSTRUCTION
SMA OCC- SUPERIOR MESENTERICE ARTERY OCCLUSION
SFH-STRANGULATED FEMORAL HERNIA
PCB-POST OPERATIVE CONSTRICTION BAND
SMV OCC- SUPERIOR MESENTERICE VEIN OCCLUSION

ABSTRACT

INTRODUCTION

Intestinal obstruction accounts for 20% of all surgical emergencies around the world. Intestinal obstruction can result from variety of causes. When strangulation superimposes and blood supply to the bowel is compromised , it may lead to bowel gangrene. The most common cause of bowel gangrene secondary to mechanical obstruction is strangulated hernia in India and post operative adhesions in developed countries.

Lactate dehydrogenase is an enzyme found abundant in intestinal mucosa and when the intestinal mucosa undergoes tissue hypoxia/ischemia, it is released into the serum and its serum level increases. Hence serial estimation of the serum LDH helps us to identify whether the obstruction goes on for gangrene or not. This study was conducted to emphasise the significance of early estimation of rise in LDH levels followed by early intervention which helps in reducing the morbidity and mortality caused by bowel gangrene following obstruction.

OBJECTIVE OF THE STUDY

1. To estimate the levels of serum LDH in patients presenting with symptoms of intestinal obstruction
2. To identify the patients with elevated levels of serum LDH and correlate the elevated levels of LDH with viability of the bowel.

MATERIALS AND METHODS

In this study, the estimation of serum LDH was conducted in 45 cases of acute intestinal obstruction who were admitted in surgical wards of CMCH during the period of September 2014 to August 2015.

LDH was estimated in all the cases preoperatively on the day of admission.

Serum LDH was estimated in all the patients at the time of admission and the estimated value of serum LDH is compared with the viability of the bowel intra operatively.

OBSERVATION AND RESULTS

ETIOLOGY	No. OF CASES	No. OF CASES WITH INCREASED LDH	NO OF CASES WITH GANGRENOUS BOWEL
Adhesive intestinal obstruction	23	9	7
Strangulated inguinal hernia	8	6	4
SMA occlusion	6	4	4
Post operative constriction bands	5	3	2
Strangulated femoral hernia	2	2	2
SMV occlusion	1	1	1
TOTAL	45	25	20

Among 45 patients presented with obstruction 20 patients had LDH level between 50 to 200 and all the 20 patients had normal bowel viability and 3 patients had LDH level between 200 to 400 and all the three patients had

normal bowel viability. 4 Patients had LDH level between 400 to 800 and two of them had normal bowel viability and two others had bowel gangrene. 8 Patients had LDH level between 800 to 1200 and all of them had bowel gangrene. 10 Patients had LDH level more than 1200 and all of them had bowel gangrene.

DISCUSSION

The normal level of serum LDH is 50 to 200 IU/L.

Out of 45 patients 25 patients had elevated serum LDH which is about 55.56% and among those 25 patients, 20 patients found to have bowel gangrene which is about 80% . In our study the mean value of elevated serum LDH in bowel gangrene patients is 1037 IU/L.

The incidence of bowel obstruction and gangrene is more common in males(72%) than females(28%).

According to Muchas, an elevated level of LDH is found in 86% of patients who had bowel gangrene.[32]

"Lactate dehydrogenase is one of the markers of intestinal ischemia".

- *Thompson.*[33]

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CONCLUSION:

From this study it is evident that ischemic changes in any part of the bowel can cause elevation in the serum levels of LDH and a higher value of >1000 IU/L strongly indicates an underlying gangrenous change.

It is a less invasive , cost effective and easily available diagnostic tool to diagnose bowel ischemia/gangrene. Hence it is more useful in centres where the diagnostic facilities are limited.

The pre operative estimation of serum LDH levels in patients presenting with features of acute intestinal obstruction helps in identifying the patients who undergo intestinal ischemia and gangrene at the earliest which makes an early intervention possible and helps in reducing the morbidity and mortality due to bowel gangrene.

Early diagnosis and early intervention is the key to reduce the mortality rate due to bowel gangrene.

This study was conducted in Coimbatore medical college hospital and the results are similar and comparable to the other studies.