CASE STUDY OF 100 CASES OF INTESTINAL OBSTRUCTION

Dissertation submitted in partial fulfillment of the
Requirement for the award of the degree of

MS DEGREE EXAMINATION

GENERAL SURGERY

TIRUNELVELI

TIRUNELVELI MEDICAL COLLEGE

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

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CERTIFICATE

This is to certify that the dissertation titled “CASE STUDY OF 100 CASES OF INTESTINAL OBSTRUCTION “ is the original work done **by DR.VARGHESE THOMAS**,post graduate in Dept. Of General Surgery,Tirunelveli medical college,to be submitted to the TAMIL NADUDr.MGR Medical University Chennai-32 towards the partial fulfillment of the requirement for the award of MS Degree in General Surgery, April 2013.

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This is to certify that the Institutional Ethical Committee of this College unanimously approves the Thesis /Dissertation/ Research Proposal submitted before this committee by Dr. **VARGHESE THOMAS**, *a* **MS POSTGRADUATE IN GENERAL SURGERY** *in the Department of* **GENERAL SURGERY**, *of Tirunelveli Medical College /Hospital, Tirunelveli titled* **"A CASE STUDY OF 100 CASES OF INTESTINAL OBSTRUCTION."** *registered by the* IEC *as* **033/GS**/IEC/2010 *dated.* **15.12.2010**. *The Investigator is hereby advised to adhere to all the stipulated norms and conditions of this ethical committee.*

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LIST OF ABBREVIATIONS

AIO	Acute intestinal obstruction
BP	Computed tomography
CVP	Central venous pressure
ECF	Extracellular fluid
ECG	Electrocardiography
ESR	Erythrocyte sedimentation rate
GI	Gastrointestinal
GIT	Gastrointestinal tract
IMA	Inferior mesenteric artery
IV	Intravenous
IVC	Inferior venacava
Kcl	Potassium chloride
PVP	Polyvinyl propylene
RBC	Red blood cells
RIF	Right iliac fossa
RR	Respiratory rate
S	Sacral
SMA	Superior mesenteric artery

CONTENTS

INTRODUCTION

Intestinal obstruction is a common cause of acute abdomen handled by surgeons. Obstuction of inguinal hernias and adhesive obstructions make up a large number of these cases. The clinical presentation varies depending on the type, site ,duration etc. Timely evaluation, diagnosis and management plays an important role in disease outcome. This study deals with epidemiology, etiology, clinical features, morbidity and mortality.

AIM OF STUDY

1. To study various causes of intestinal obstruction.
2. To study various presentations of the cases.
3. To study epidemiology of cases.
4. To study various surgical procedures and its outcome.
5. To study factors affecting morbidity and mortality.

Inclusion criteria

1. All cases > 12 years age
2. Both males and females
3. Small bowel and large bowel obstruction
4. Cases of acute intestinal obstruction .

Exclusion criteria

1. All cases < 12 years age
2. Adynamicintestinalobstruction.
3. Sub acute cases.

REVIEW OF LITERATURE

HISTORICAL REVIEW

The attempts to treat acute intestinal obstruction dates back to centuries. In 6th century Sushrutha wrote oldest known descriptions of bowel surgery. Forms of intestinal obstruction like strangulated hernia, intussusceptions were known to the ancient Egyptians. Intestinal obstruction was observed by Hippocrates (460-370 BC).

The earliest operation recorded was performed by Proxogorous (350 BC), who created enterocutaneous fistula to relieve obstruction.

- Fabricusd'Aquopendente in 12 century described a procedure of intestinal repair the involving end-to-end anastamosis.
- Sanctus in 16 century treated intestinal obstruction by giving metallic mercury to the patients.
- John Arderence (1306-1390) was the first surgeon who wrote the book on "PassioIliaca" (Appendicitis or intestinal obstruction).
- Ambrosis Pare (1510-1590) was first to recognize obstruction as a pathological entity. For severe cases he used mercury in water, lead bullets smeared with mercury. (3)
- Franco (1561) did first Surgery on strangulated hernia.

- Kerckring in 1670 described the intestinal valvulaeconniventes.
- Bonetos in 1679 treated intussusception surgically.
- Amsterdam in 1676 suggested opening of abdomen to treat intussusception and volvulus.
- Mery in 1701, removed several feet of gangrenous bowel and established an artificial anus in woman suffering from strangulated hernia.
- La Peyronie in 1723, reported having excised the devitalized bowel and creating an artificial anus and mucous fistula, which was closed later on.
- Mensching in 1756 used repeated intestinal puncture to treat obstructed bowels.
- Planned Caecostomy was first described by Pillore (1776).
- Duret performed the first successful sigmoidostomy for the relief of obstruction in 1793.
- Dupytren in 1800 established enterostomy for obstruction.
- In 1826, Lipperance resected the lower rectum and anus and created colostomy, and in 1836, Diffenbach did resection anastomosis of small bowel in strangulated hernia.
- Duchenne in 1855 reported several successful instances where he treated intestinal obstruction with faradic current.

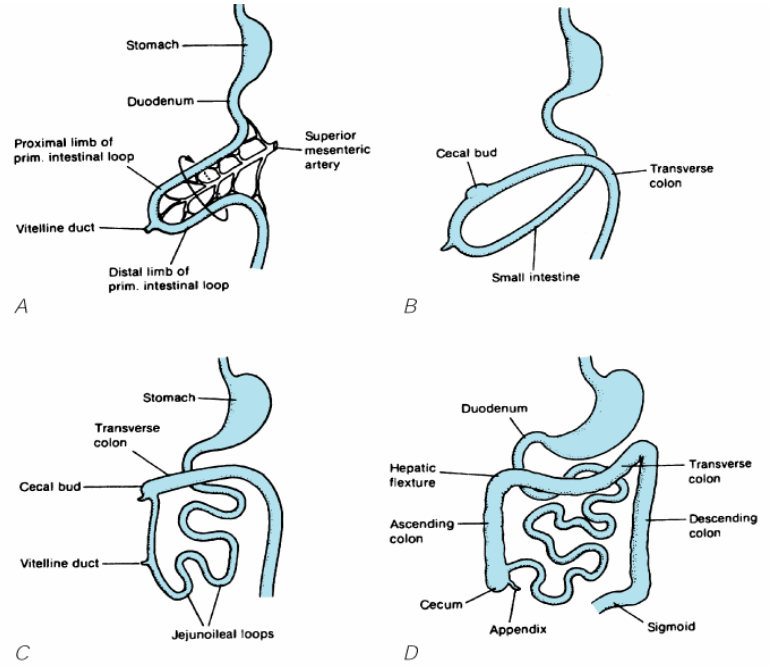
- Volvulus of caecum was first described by Rokintansky in 1841.
- Jonathan Hutchison performed first successful operation for intussusception in 1871 and Hirschprung in 1877 reduced intussusception by salt water and enema
- In 1875 Elliott performed bowel resection for mesentric vascular occlusion .
- In 1880 Block Parker and Kerr safely did exteriorisation, primary resection andanastamosis in different cases.
- Roentgen in 1893 discovered X-ray and Schwartz in 1911 pointed out the virtue of scout fIlm; Kloiber of Germany (1919) emphasized the importance of X-ray in locating the level of obstruction. (4)
- How toxic substances get accumulated bowel was described by Whipple and Williams (1926) described influence of anaerobic infection as a cause of toxaemia.
- Einborn (1909) passed a 18 F tube to study small bowel contents and in 1926 van der Reir and Schamber passed a tube through whole of GIT.
- In 1925, Gamble et al. proved that loss of fluid and solutes from the body into the bowel lumen was the factor responsible for death.
- Oppenheimer and Mann (1934) studied microcirculation,caecostomy was firstdescribed by Allen and

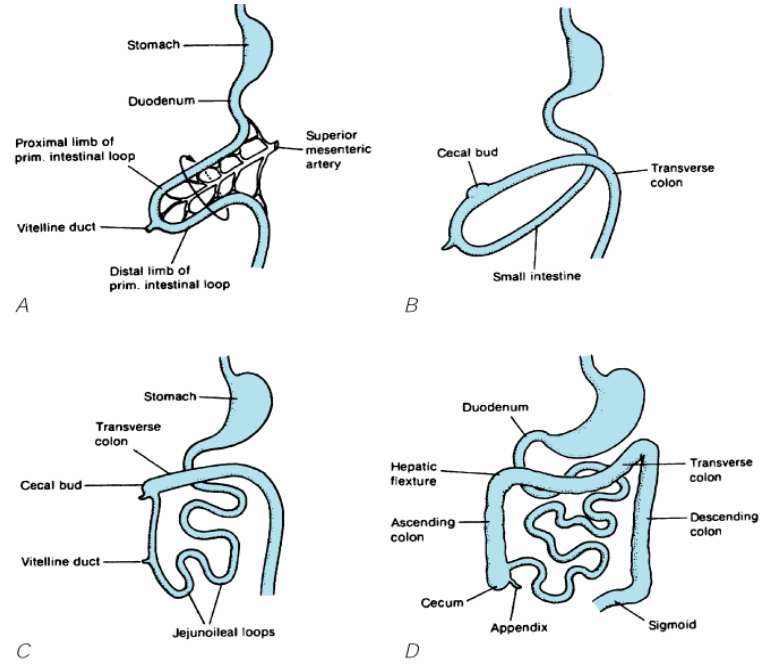
Welch in 1947. Noer et al in 1951 confirmed circulatory disturbances brought about by increased distension.

- Johnston in 1938, Harris in 1945, Cantor in 1946 and Gafton Smith in 1952 described various other tubes for gastrointestinal drainage.
- Bishop and Allock (1960) studied the bacteriology of gut above the obstruction.
- Bilgutay (1963) has encouraged the use of faradic current for intestinal paresisfollowing operations.
- The experimental use of Doppler ultrasonography to determine the viability of ischaemic intestine was first described by Wright and Gobson in 1975.
- Marfuggi and Greenspan in 1981 reported 93% accuracy of fluoroscopic dye injection technique for determining the viability of ischaemic bowel.
- Bookstein in 1982, used angiography to diagnose and treat small bowel bleeding.
- In 1996, Akgun gained more attention in mesosigmoplasty as definitive operation sigmoid volvulus.(5)
- In 1997, Yaco et al. evaluated diagnostic procedure, for diverticular disease (CT scan, contrast enema, ultrasonography).

EMBRYOLOGY OF SMALL INTESTINE

During the early stage of development, the primitive gut is in free communication with the rest of the yolk sac. In the cephalic and caudal parts of the embryo the primitive gut forms a blind ending tube the foregut and the hindgut and the middle part, the midgut remains temporarily connoted to the yolk sac. In the 5^{th}week embryo, there will be rapid elongation of the gut and its mesentery resulting in formation of the primary intestinal loop. At its apex, the loop remains in open connection with the yolk sac by way of the narrow vitelline duct. The cephalic limb of the loop develops into the distal part of the duodenum ,the jejunum and part of the ileum. The caudallimb becomes the lower portion of the ileum, the caecum, the appendix, the ascending colon and the proximal two-thirds of the transverse colon. The hindgut gives raise to distal third of transverse colon, the sigmoid, the rectum and part of anal canal.


Stomach
Duodenum
Proximal limb of prim. intestinal loop
Superior mesenteric artery
Vitelline duct
Distal limb of prim. intestinal loop
A
Cecal bud
Transverse colon
Small intestine
B
Stomach
Transverse colon
Cecal bud
Vitelline duct
Jejunoileal loops
C
Duodenum
Hepatic flexture
Transverse colon
Ascending colon
Descending colon
Cecum
Appendix
Sigmoid
D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CHRONOLOGY OF ROTATION OF THE MIDGUT LOOP

The loop has a prearterial or proximal segment and post arterial or distal segment. Viewed from the ventral side, the loop undergoes an anticlockwise rotation by 90°, so that it now lies in the horizontal plane. The pre-arterial segment comes to lie on the right side and the post-arterial segment on the left.

- Pre-arterial segment now undergoes great increase in length to form coils of the jejunum and ileum and the loops still lie outside the abdominal cavity to the right side of the distal limb.
- The coils of the jejunum and ileum (pre-arterial) return to the abdominal cavity. The coils of jejunum and ileum pass behind the superior mesenteric artery into the left half of the abdominal cavity.
- The post-arterial segment of the midgut loop returns to the abdominal cavity, it also rotates in an anticlockwise direction so that the transverse colon lies anterior to the superior mesenteric artery and the caecum comes to lie on the right side.
- Gradually the caecum descends to the iliac fossa and the ascending, transverse and descending parts of the colon become distinct.

FIXATION OF THE GUT

Small and large intestine are suspended from the posterior abdominal wall by mesentery. After the completion of rotation of the gut,

the duodenum, the ascending colon, the descending colon and the rectum become retroperitoneal by fusion of their mesenteries with the posterior abdominal wall. The original mesentery persists as the mesentery of the small intestine, the transverse mesocolon and the pelvic mesocolon.

There are three errors in the stages of rotation.

1. Non-rotation
2. Reversed rotation
3. Malrotation

PATHOLOGICAL CONSEQUENCES OF ANOMALIES OF ROTATION (6)

- No functional disturbance may result from abnormal fixation.
- Deficient fixation causes ptosis, torsion and volvulus.
- Excessive fixation may cause interference with mobility, kinks and compression of intestine.
- Abnormal rotation predisposes to volvulus, which causes intestinal obstruction.
- Volvulus of the ileocaecal segment is the typical lesion in later life resulting from imperfect rotation or deficient fixation of the gut.

ACCESSORY BANDS OF PERITONEUM

causes (1) Intestinal obstruction (2) Kinking (3) Angulation of bowel. Failure of part of the original membrane to disappear, minor

alterations in the development of secondary mesentery result in accessory peritoneal bands. These are:

- **Lane's ileal band:** The thickened peritoneal band extending from the right iliac fossa to the 5 cm of ileum which on continuous contraction causes kinking of the small bowel and resulting in obstruction.
- **Mesosigmoidmembrane** (Lane's first and last band): This is formed by thethickening of peritoneum extending from the pelvic brim of left iliac region to the junction of descending and sigmoid colon.
- **Genitomesenteric fold of Douglas:** causes kinking of appendix causing obstructive appendicitis as it extends from the back of the terminal mesentery to the region of the suspensory ligament or ovary or testis.
- **Jackson's membrane:** Lies between the posterior abdominal wall and caecum or ascending colon on the right side or from the hepatic flexure to caecum.

ANATOMY

SMALL INTESTINE

The small intestine is the longest part of the gastrointestinal tract and extends from the pyloric orifice of the stomach to the ileocaecal fold. This hollow tube, which is approximately 6-7 m long with a narrowing diameter from beginning to end, consists of the duodenum, the jejunum and the ileum.(7). The adult duodenum is 20-25 cm length and the name coined as duodenum because length is as long as width of 12 fingers. It is shortest, widest and most fixed part. It has nomesentery and partially covered by peritoneum. Its course presents a remarkable curve somewhat like horseshoe type, the convexity being directed towards the right and concavity to the left embracing the head of the pancreas. It has been divided into four portions. First part (superior portion), Second part (descending portion), third part (horizontal portion) and fourth part (ascending portion).

Blood supply and nerve supply

Arteries supplying the duodenum arise from the right gastric, supraduodenal, right gastroepiploic, and superior and inferior pancreaticoduodenal arteries.

Veins**:** These end in the splenic, superior mesenteric and portal veins.

Nerves**:** They come from the coeliac plexus.

Lymph nodes: Along interior and posterior pancreaticoduodenal artery.

Jejunum and ileum

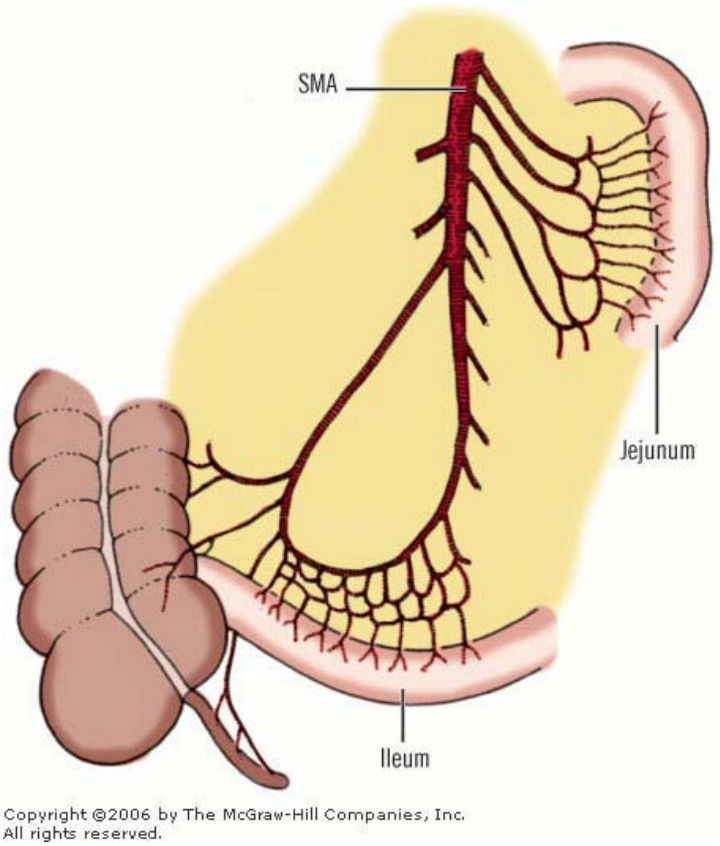
In small intestine excluding duodenum, upper 2/5 is formed by jejunum and lower 3/5 is ileum. Rest of small intestine extends from the duodenojejunalflexure to the ileocaecal valve, ending at the junction of the caecum and ascending colon. It is arranged in a series of coils attached to the posterior abdominal wall by the mesentery. The jejunal loops characteristically situated in the upper abdomen to the left of midline, whereas the ileal loops tend to lie in the lower right part of the abdomen and pelvis. Thisdistribution can be reversed during paralytic ileus or small bowel obstruction due to rotation around the mesenteric attachment following bowel distension.

The wall of jejunum and ileum is composed of serosa of visceral peritoneum, muscularis of longitudinal and circular smoothmusclefibres and a mucosa of connective tissue, smooth muscle and epithelium.

Blood supply

Blood supply is by superior mesenteric artery which is a branch of aorta, the branches of which, reaching the mesenteric border, extend between the serosal and muscular layers. After this, numerous branches traverse the muscle, supplying it and forming an intricate submucosal plexus from which minute vessels pass to glands and villi. The superior

mesenteric veins follow the arteries.



Nerve supply

Nerve supply by vagi and thoracic splanchnic nerves through the coeliac ganglia and superior mesenteric plexus.

LARGE INTESTINE

It is about 150 cm long, it extends from the terminal ileum to the anus. Its function is chiefly absorption of fluids and solutes and it differs in structure, size and arrangement from the small intestine in the following ways:

- It is for the most part more fixed in position.

- Its longitudinal muscle, though a complete layer, is concentrated into three longitudinal taeniae coli.
- The colonic wall is puckered into sacculations (haustrations) and appendices epiploicie by the taeniae.
- It has a great caliber.

The divisions are caecum, colon proper and the rectum.

Caecum

The caecum is a blind pouch lies in the right iliac fossa, its average axial length is about 6 cm and its breadth about 7.5 cm continues proximally with the distal ileum and distally with the ascending colon and is related posteriorly to iliopsoas muscle and femoral nerve, anteriorly to the abdominal wall, greater omentum and the coils of ileum. Almost the entire posterior surface of caecum is attached to the abdominal wall, in some cases it is wholly unattached.

Ileocaecal valve

The ileum opens on its posteromedial aspects of the caeco-colic junction and two flaps which project into the lumen of the colon. The valve is actually closed by sympathetic tone. It is mechanically closed by the distensions of caecum and prevents the reflux of caecal contents intoil;eum

Colon

The colon is conveniently considered in four parts: (1) Ascending, (2) Transverse, (3) Descending and (4) Sigmoid.

Ascending colon

It is normally fused with the posterior body wall and covered by peritoneum anteriorly. It is about 15 cm long and narrower than the caecum it ascends to the inferior surface of the right lobe of the liver, on which it makes a shallow depression, here it turns abruptly forwards and to the left; at right colic flexure.

Hepatic flexure

Anteriorly covered by peritoneum, posteriorly not covered by peritoneum and is in direct contact with renal fascia. It is related posteriorly to the inferolateral part of the anterior surface of the right kidney above, and anterolaterally is the right lobe of the liver, anteromedially are the descending part of the duodenum and fundus of the gallbladder.

Transverse colon

It extends from the right hepatic flexure to the left colic flexure measuring 50 cm. The transverse colon, unlike ascending and descending colon has a mesentery that had secondarily fused with posterior wall of the omental bursa. The transverse colon hangs in U or V shaped curve. Above

the transverse colon are the liver and gallbladder, the greater gastric curvature and the lateral end of spleen, below is the small intestine, in front are the posterior layers of the greater omentum and behind are the descending part of the duodenum, the head of the pancreas, the upper end of the mesentery, duodenojejunal flexure and coils of the jejunum and ileum. The transverse Colonsome times may be interposed between liver and diaphragm (Chilaiditi syndrome).

Splenic flexure

This is the junction of the transverse and descending colon in the left hypochondriac region. It is related to the lower part of the spleen and pancreatic tail above and medially with the front of the left kidney. It is attached to diaphragm by phrenico-colic ligament, which lies below the anterolateral pole of the spleen. It lies more superiorly and posteriorly than the hepatic flexure at the level of $10^{th}$and 11^{th}ribs.

Descending colon

It is about 25 cm long and extends from the splenic flexure to pelvic brim, and in the whole of its course is plastered to the posterior abdominal wall by peritoneum (like ascending colon). The descending colon is smaller in caliber more deeply placed and more frequently covered posteriorly by peritoneum. The descending colon lies on the lumbar fascia and iliac fascia. It ends at the pelvic brim about 5 cm above the inguinal

ligament.

Sigmoid colon

It is about 40 cm length. Sigmoid colon extends from the descending colon at the pelvic brim to the commencement of the rectum in front of the third piece of the sacrum. The sigmoid mesocolon has an inverted ‘V’ attachment to the posterior abdominal wall.

Blood supply

Blood supply is by branches of superior mesenteric artery and inferior mesenteric artery. Superior mesenteric artery supply uptothe junction of middle 1/3rdof transverse colon and colon beyond this is supplied by inferior mesenteric artery.

Nerve supply

Sympathetic to midgut from coeliac ganglion (T1–L1).Parasympathetic from vagus through coeliac plexus. Hindgut portion receives sympathetic supply from the lumbar sympathetic chain from L1–L2 and parasympathetic from the pelvic splanchnic nerves.

Rectum

The rectum is 12 cm long and is continuous with the sigmoid colon at S3. The human rectum follows the posterior concavity of the sacrum and shows three lateral curves or flexures that are most prominent when the viscusdistended, upper and lower curves convex to the right and a

middle curve convex to the left, the lowest part is slightly dilated as the rectal ampulla. It ends 2-3 cm in front and below the tip of the coccyx, turning abruptly downwards and backwards through levatorani muscle to become the anal canal 4 cm from the anal verge.

The mucosa of large intestine differs from small intestine in that it has no villi and consists of simple columnar epithelium which has only absorptive and goblet cells. The longitudinal muscles of muscularis mucosa are thickened to form taeniacoli, on contraction leads to formation of haustra necessary for haustral churning.

Blood supply

Blood supply mainly from the superior rectal artery, with contributions from the middle and inferior rectal and median sacral vessels.

Veins correspond to the arteries, but anastomose freely with one another, forming an internal rectal plexus in the submucosa and external rectal plexus outside the muscular wall.

Nerve supply

The sympathetic is derived by branches from the hypogastric plexus. The parasympathetic supply is from S2 and S3 by the pelvic splanchnic nerves.

Lymphatic drainage of colon

Lymph from the colon passes through four sets of lymph nodes:

(a) Epicolic lymph nodes, lying on the wall of the colon

(b) Paracolic nodes on the medial side of ascending, descending and mesocolic border of transverse and sigmoid colon

(c) Intermediate nodes along the main branches of vessels,

(d) Terminal nodes at the origin of SMA and IMA, finally drains to para-aortic nodes.

PHYSIOLOGY

The gastrointestinal system consists of the gastrointestinal tract and associated glandular organs that produce secretions.

The major physiological functions of gastrointestinal system are to digest food stuffs and absorb nutrient molecules into the blood stream. Mainly the small intestine and large intestinal carries out these functions by motility, secretion digestion and absorption.

Motility refers to the movements that mix and circulate the gastrointestinal contents and propel them along the length of the tract. The contents are usually propelled in the orthograde (forward) direction.

Secretion – refers to the processes by which the glands associated with the small intestine and large intestine release water and substances into the lumen.

Digestion – defined as the processes by which food and large molecules are chemically degraded to produce smaller molecules that can be absorbed along the wall of the intestine.

Absorption refers to the processes by which nutrient molecules are absorbed by cells that line the intestine and enter the circulation.

Functional Anatomy of Intestine

Intestinal villi – the villi are minute projections which are called as

enterocytes. Each enterocyte gives rise to hair like projections called microvilli, within each villi called lacteal. The lacteal opens into lymphatic vessels.

Crypts of Lieberkuhn are simple tubular glands of intestine. The three types of cells are interposed between columnar cells of the glands.

1. Argentaffin cells also known as enterochromaffin cells which secretsintrinsicfactor that is essential for the absorption of the vitamin B .
2. mucussecreated by goblet cells
3. Paneth cells -- cytokines called defensins.

Brunner's glands: These glands penetrate muscularis mucosa in the first part of the duodenum secretes mucus and traces of enzymes. The small intestine is presented with about 9 litres of fluid per day, 2 litres from dietary sources and 7 litres of gastrointestinal secretions, howeveronly 1-2 litres pass into the colon.

SUCCUS ENTERICUS

Water 99.5% Solids 0.5%

Reaction – Alkaline

pH – 8.3

Functions of SuccusEntericus

1. Digestive function – The enzymes of succusentericus act on the

partially digestedfood and convert them into final digestive products.

2. Protective function – The mucus present in the succusentericus protects the intestinal wall from the acid chime, which enters the intestine from stomach paneth cells secrete defensives which are the antimicrobial peptides.

3. Activator function – The enterokinase present in intestinal juice activates trypsinogen into trypsin.

4. Haemopoietic function – Intrinsic factor of castle which is present in the intestine,plays important role in erythropoiesis.

5. Hydrolytic process – Intestinal juice helps in all the enzymatic reactions of digestion.

Functions of small intestine

1. Mechanical function
2. Secretory function
3. Hormonal function
4. Digestive function
5. Activator function
6. Hemopoietic function
7. Hydrolytic function
8. Absorptive function

LARGE INTESTINE

Secretions

Large intestine juice

Water 99.5% Solids 0.5%

Organicsubstances

Inorganic substances

Functions of large intestine

1.Absorptive function – absorbs various substances such as water, electrolytes, organic substances like glucose, alcohol, drugs like anaesthetic agents, sedatives and steroids.

2. Formation of faeces
3. Excretory function
4. Secretory function
5. Synthetic function – synthesizes folic acid, vitamin B and vitamin K

Movements of small intestine

The movements of small intestine are essentials for mixing the chime with digestive juices, propulsion of food and absorption.

Four stages of movements occur in small intestine.

1. Mixing movements

a. Segmentation movements

b. Pendular movements

2. Propulsive movements

a. Peristaltic movements

b. Peristaltic rush

3. Peristalsis in fasting – Migrating motor complex

4. Movements of villi

Movements of large intestine

- Segmentation contractions
- Mass peristalsis

Intestinal bacteria

The bacteria include various strains of Escherichia coli and Enterobacteraerogenes. Pleomorphic organisms such as Bacteroidesfragilis and cocci of various types.

PATHOPHYSIOLOGY OF BOWEL OBSTRUCTION

Management of acute intestinal obstruction depends largely on early diagnosis, skillful management and an appreciation of the importance of treating the pathological effects of the obstruction just as much as the cause itself.

If detected early, the prognosis will be excellent on relieving the obstruction but in late cases where due to obstruction there is vascular compromise, where relieving of obstruction is not enough,it needs many other surgical procedures like resection, anastamosis, etc

Pathophysiological changes in acute bowel obstruction

The pathophysiological changes that occur with AIQ can be studied under following heads:

1. Intestinal distension

Although a constant feature of bowel obstruction, the mechanism underlying the intestinal distension has not been elucidated completely. Most of the gas distending the small bowel in early phases of obstruction accumulates from swallowed air. Other sources include: fermentation of sugars, production of carbon dioxide by interaction of gastric acid and bicarbonates in pancreatic and biliary secretions, and diffusion of oxygen and carbon dioxide from the blood. Followingdilatation and inflammation, activated neutrophils and macrophages accumulate in the bowel wall due

to increased blood flow to the gut, these release reactive proteolyticenzymes, cytokines, and other locally active substances which inhibit or damage the secretory and motor processes of the gut .The nitric oxide produced during the process causes smooth muscle relaxation, further aggravating the distension and inhibiting gut contractility The normal intraluminal pressure of 2 to 4 cm of water rises to 8 to 10 cm of water in obstruction, which may reach 30 to 60 cm of water in close loop obstruction. The reactive oxygen radicals produced during these changes not only affect gut motility but also its permeability.

During the first 12 hours of AIO, water and electrolytes accumulate within the lumen secondary to a decrease in absorption. By 24 hours, accumulation occurs more rapidly due to a further decrease in absorption and in addition to an increase in intestinal secretion secondary to mucosal injury and increased permeability. Although the role of neural or systemic humoral/hormonal mechanisms in aggravating distension remains likely, it is poorly investigated.

This decrease in the absorptive capacity of the gut with an increase in intraluminal secretion leads to excessive fluid losses which can lead to dehydration. Although the intestinal wall distal to the obstruction maintains normal function, the inability of the luminal content to reach the unobstructed gut compounds the dehydration.

2. Intestinal, Motility

After intestinal obstruction Intestinal motility, absorption and secretion are affected butwhen the normal bacterial barrier function of the viable gut fails is unclear.(10).

In the early phase of bowel obstruction, intestinal contractile activity increases in an attempt to move intraluminal contents past the obstruction. Later, it diminishessecondary to intestinal wall hypoxia and exaggerated intramural inflammation; however, the exact mechanisms have not been completely elucidated. Some investigators have suggested that the alterations in intestinal motility are secondary to a disruption of the normal autonomic, parasympathetic (vagal) and sympathetic, splanchnic innervation.

Cajalcells(interstitial cells)generate electrical slow waves which promote intestinal motility.These are lost on distension and regained on normalisation.

3. Circulatory Changes

Ischemia of the bowel wall can occur by several different mechanisms.Extrinsic compression of the mesentery by adhesions, fibrosis, mass, twisting or a hernia defect,extrinsic pressure on a segment of bowel (e.g., a fibrous band), or progressive distension in the setting of a closed-loop obstruction can all cause vascular compromise or

strangulation. The consequences of vascular compromise are more disastrous in large bowel obstruction, as in nearly a third of people ileocecal valve is competent,(11a), which functionally leads to closed-loop obstruction between the competent ileocaecal valve and the site of obstruction in the large bowel.

Progressive distension of the bowel lumen with a concomitant increase in intraluminal pressure results in increased transmural pressure on capillary blood flowwithin the bowel wall. In simple (non-closed loop) obstruction, this occurs rarely, as the obstructed distended bowel can decompress proximally. In severe intestinal distention, which is self-perpetuating and progressive, peristaltic and secretory derangements occur and the risks of dehydration and progression to strangulating obstruction increase. Strangulating obstruction involves compromised blood flow; occurring in nearly 25% of patients with small bowel obstructions. It is usually seen with hernia, volvulus, and intussusception.

In as little as 6 hours strangulating obstruction can progress to infarction and gangrene. Venous obstruction leads to arterial occlusion, causing rapid ischemia of the bowel wall,leading to gangrene and perforation. It is more common in cecum and ascending colon where the luminal diameter is greatest and (by Laplace's law) the wall tension (and ischemia.) is also maximum. This makes large bowel obstruction more of

a surgical emergency than small bowel obstruction. With strangulation, therecan also be blood loss into the infarcted bowel, which together with the preexistent fluid loss leads to further hemodynamic instability, exacerbating the already compromised blood flow of the intestinal wall.

4. Microbiological changes and Bacterial Translocation

The upper small intestine contains gram-positive facultative organisms in small concentrations, usually <10 ^6 colonies/mL. More distally, the bacterial count increases in concentration to about 10^8 times in distal ileum. Changing primarily to coliforms and anaerobes. In the presence of obstruction, bacteria proliferate rapidly proximal to the obstruction in direct proportion to the duration of obstruction, reaching a plateau of 10^9 -10^10 colonies/mL after 12-48 hours of obstruction.

The bowel distal to the obstruction tends to maintain its usual bacterial flora until ileus sets in, following which there is generalized bacterial proliferation. Toxins produced by these bacteria disrupt the mechanical integrity of the gut mucosa. Once the gut mucosal barrier is lost, bacterial translocation occurs as the luminal bacteria invade the submucosa and enter the systemic circulation via the portal venous and lymphatic systems. Reduction of perfusion of the intestinal wall further compromises the mucosal defences. All these changes have been well documented in animal models; however, documentation of true bacterial

translocation in humans is lacking. More recent work has shown that lipopolysaccharide and other inflammatory mediators, but not bacteria, can be recovered from the mesenteric lymphatics. The eventual drainage of these vasoactive substances into the systemic circulation may lead both to the systemic manifestations of sepsis and further disruption of the mucosal barrier function.

Due to these alterations in resident microbial flora, the risk of infective complications in bowel obstruction is increased markedly, especially if bowel resection is required or if an inadvertent enterotomy is made with intraperitoneal spillage of "obstructed" enteric contents. With strangulation, there is systemic entry of bacterial products, activation of immunocompetent cells, release of cytokines, and increased formation of reactive oxygen intermediates, this leads to systemic inflammatory response syndrome and multiple organ dysfunction.

Metabolic effects of obstruction

- In proximal obstruction - dehydration, hypochloremia, hypokalemia, metabolic alkalosis
- In distal obstruction - third space losses and dehydration
- Hypovolemia, hypotension
- Haemoconcentration
- Oliguria and azotemia

    - Increased intraabdominal pressure
    - Restricted ventilation and atelectasis
    - Impaired venous return
- Congestion of the bowel wall and seepage of blood into the lumen (significant if long segments involved)
    - Strangulation of the gut
    - Shock and death

PATHOLOGY

Distension occurs proximal to the obstruction and starts immediately after the obstruction occurs. Causes of distension are: fluid, gas and intestinal toxins.

(a) Fluid: made up by the various digestive juice about 8 litres/day.

Above pylorus 4 litres Saliva 1.5 litres Gastric 2.5 litresBelow pylorus 4 litres Bile and pancreatic 1 litre Succusentericus 3 litre

In obstructive pathology, there will be high enteric pressure leading to edema, shortening and clubbing of the villi leading to disturbed absorptive mechanism. Also there will be depletion of water and electrolytes due to vomiting, defective absorption, sequestration in lumen of bowel.

(b) Gas: Consists of swallowed atmospheric air, diffusion from blood into the bowel lumen 22% and the products of digestion and bacterial activity. When the O and CO has been absorbed into the blood stream, the resultant mixture is made up of N (90%) and H S.

(c) Intestinal toxins: In unrelieved strangulation, toxic substances appear in the peritoneal cavity only when the viability of the bowel wall is affected. When the obstruction is relieved these toxins may pass on to the bowel where absorption can occur. It is probable that the substances involved are endotoxins of Gram negative bacilli.

THE EFFECT OF OBSTRUCTION ON INTESTINAL MOTILITY

In the obstruction of the bowel, after some considerable period, peristaltic waves become incoordinated. Later with gross stretching, peristalsis stops and a stage of paralytic ileus occurs.

Progressive distension of the intestine leads to increase in intraluminal pressure and compression of capillaries. The slowing of local circulation and anoxic damage to capillary endothelium leads to leakage of fluid across the damaged capillaries, eventually accumulation of fluid above the site of obstruction occurs.

There after excessive distension of the intestine leads ischaemic necrosis and perforation at the antimesenteric border which is attributed to a combination of thinning wall, high pressure within the gut lumen and obliteration of its blood supply.

Some investigators have suggested that the alterations in intestinal motility are secondary to disruption of the normal autonomic, parasympathetic (vagal) and sympathetic splanchnic innervation. When the bowel is trapped by a hernia or band or involved in a volvulus or Intussusception in such a way that its blood supply is progressively interfered with leading to strangulation. Mesenteric vascular occlusion alone gives rise to gangrene without mechanical obstruction.

The first effect of strangulation is to compress the veins so as to cause ischaemia. When the venous return is completely occluded, the

colour of the intestine turns from purple to black. Due to increased edema at the point of obstruction, there will be capillary rupture with haemorrhagic infiltration. Thrombosis in intramural and mesenteric veins hastens the ischaemia. Mucosal necrosis first appears and spread towards the serosa causing moist gangrene of the bowel.

Increasing venous and arterial obstruction cause extravasation of blood under the serosa and effusion into the bowel lumen. In the case of strangulated external hernia only a small segment is involved, the blood that is sequestrated is minimal but when a large coil of intestine becomes strangulated, the blood loss is quite significant to make the patient hypovolemic and in severe cases leads to death of the patient.

In case of obstruction due to closed loop as seen in carcinomatous stricture of the colon.Proximally the competent ileocaecal valve prevents regurgitation of the contents whereas distally the colon is occluded by the neoplasm. This leads to high pressure in the caecum. If obstruction is not relieved, due to compression of the blood vessel in wall, ulceration, gangrene and perforation of the caecum will occur.

Adynamic obstruction

Adynamic obstruction is a condition where there is failure of neuro-muscular mechanism i.e. Auerbach’s and Meissner plexuses resulting in atony of the bowel producing loss of peristalsis and accompanied by abdominal distension.

CLASSIFICATION OF INTESTINAL OBSTRUCTION

Acute intestinal obstruction is most commonly a surgical disorder of small intestine and accounts for approximately 20% of all surgical admissions. Intestinal obstruction may be classified into two types.

1. Dynamic obstruction
2. Adynamic obstruction

DYNAMIC OBSTRUCTION: Where peristalsis working against a mechanical obstruction. Irrespective of aetiology or acuteness of onset, in dynamic obstruction the proximal bowel dilates and develops an altered motility. Below the obstruction the bowel exhibits normal peristalsis and absorption until it becomes empty at which point it contracts and becomes immobile. The causes of intestinal obstruction are:

Intraluminal

- Intussusception
- Bezoar
- Foreign bodies
- Gallstones
- Mucosal tumours

Intramural

- Stricture
- Malignancy: Carcinoid, Lymphoma, Leiomyosarcoma

- Inflammation: Crohn’s disease, Tuberculosis
- Haematoma
- Endometriosis

Extramural

- Bands/adhesions
- Hernia: External – Inguinal, Femoral, Incisional, Obturator

  Internal – Paraduodenal, Epiploic foramen, Diaphragmatic, Transmesenteric
- Tumours: Peritoneal metastasis, Desmoid
- Abscess: Diverticulitis, Pelvis inflammatory disease, Crohn’s disease

ADYNAMIC OBSTRUCTION: This may occur in two forms:

a. Peristalsis may be absent e.g.: Paralytic ileus

b. Peristalsis may be present in a non-propulsive form e.g.: (1) Mesenteric vascular occlusion (2) Pseudoobstruction.

CLINICAL FEATURES

Cardinal features of intestinal obstruction are:

1. Pain abdomen
2. Vomiting
3. Distension of abdomen
4. Constipation

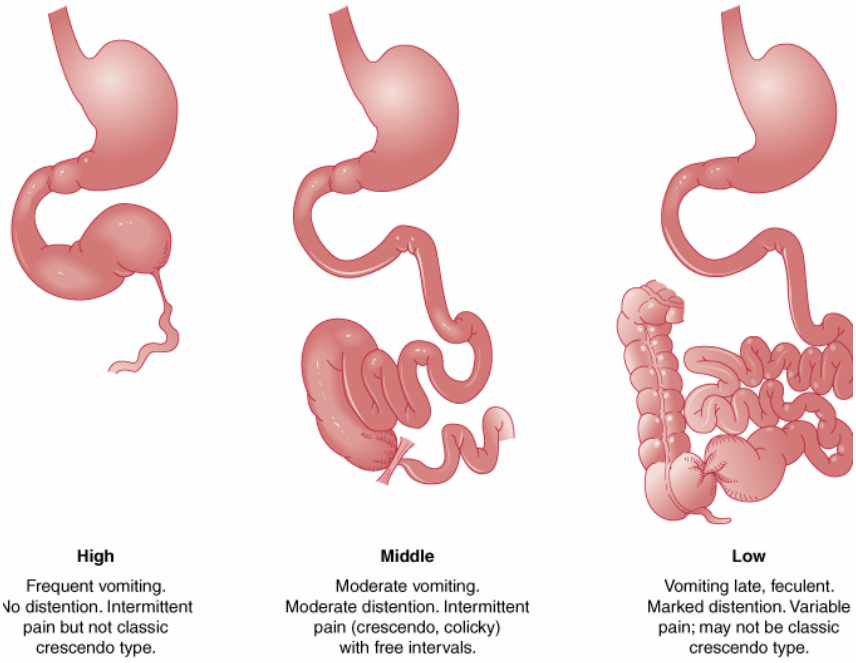
1. Pain abdomen

Abdominal pain is the first symptom. The onset may be insidious or abrupt in simple obstruction, but with strangulation the onset is usually sudden and severe. The pain is diffuse, poorly localized .

2. Vomiting

Vomiting is the next most common symptom. A constant symptom, the early vomiting is reflex in nature followed by quiescent period before real vomiting due to obstruction resumes. This quiescent period is of shorter duration in high-level obstruction and longer inlower small bowel obstruction. Initially it contains partly digested food, followed by bilious vomiting. Finally it is faeculent.

In low-level small bowel obstruction vomiting is less projectile and less frequent.. Reflex vomiting is unusual in colonic obstruction because the ileocaecal valve is competent. Colonic obstruction is associated with the brown vomit which becomes increasingly foul smelling as the obstruction persists.(13)



3. Distension

In early cases of obstruction of the small intestine abdominal distension is often slight or even absent. When the proximal jejunum is obstructed, the stomach becomes distended with gas and accumulated secretion, so that the epigastric region may, in later stages be more prominent and tense. When the ileum is involved, the central portion of the abdomen is moderately blown out and when the distal colon is blocked, there is considerable universal distension of abdomen, with well-marked bulging in the flanks. Visible peristalsis may be present.

4. Constipation

In complete intestinal obstruction, after the contents of the bowel below the obstruction have been evacuated, there is constipation and usually neither faeces nor flatus is passed i.e. absolute constipation. The

rule of constipation is present in intestinal obstruction does not apply in cases of Ritcher's hernia, gall stone obstruction, mesenteric vascular occlusion.

HISTORY AND PHYSICAL EXAMINATION

A detailed history and physical examination helps in diagnosis and management of intestinal obstruction. In simple mechanical obstruction there will be very few abdominal signs. Whereas in strangulated obstruction, patient will be toxic, tachycardia and hypotension will be there. Any past history of abdominal surgery suggests adhesion as a cause of obstruction. Hernia of long duration gives rise to strangulation,

Physical examination

Skin turgor: may be lost due to dehydration, may be cold and clammy.

Tongue:which may be dry and coated due to dehydration.

Nail and sclera: Anaemia, jaundice may be evident.

Rapid low volume pulse, low blood pressure, cold extremities, anxious look and increased respiratory rate are the evidence of shock and septicaemia

Examination of abdomen

Inspection: On inspection previous surgical scars which indicates adhesions orcancer. In early stage visible peristalsis may be seen. All hernial orifices haveto be inspected.

- **Type of abdominal distension:** Central in small bowel obstruction and upper abdominal in high-level obstruction. Distension will be more in the flanks in colonic obstruction.
- **Type of peristalsis:** Central step ladder type of peristalsis seen in distal small bowel obstruction and right to left over the umbilicus in the colonic obstruction.

Palpation

- Abdomen must be examined for presence of any palpable mass, localized abdominal tenderness, rebound tenderness and mass is suggestive of strangulation. In peritonitis there will be generalized rigidity and tenderness.

Auscultation: In simple mechanical obstruction, sounds become loud, high pitched and metallic. In late stages bowel sounds may be absent due to paralysis of bowel musculature. Bowel sounds may be absent in strangulation and ileus or low-pitched tingling sounds may be heard due to movements of fluid from one coil to another.

Rectal examination: To be performed in all cases of obstruction, may reveal faecal impaction, mass, red current bleeding in intussusception. A palpable pelvic mass or bulge due to collection in the Pouch of Douglas may be present. Ballooning of the rectum usually occurs in the intestinal obstruction may be due to obstruction to nerves causing sympathetic

paralysis.

LABORATORY INVESTIGATIONS

(i) Hematological tests

A full blood count, packed cell volume, serum electrolyte determination and blood urea level should be done. Leucocytosis is indicative of strangulation, extremely high counts are highly suggestive of mesenteric thrombosis. Serum electrolyte estimation will guide for fluid management which is the initial management of intestinal obstruction. Metabolic acidosis seen in distal intestinal obstruction due to combined effect of dehydration, ketosis and loss of alkaline secretions and metabolic alkalosis is seen in upper, intestinal obstruction due to considerable loss of acidic juice.

(ii) Urine examination

Specific gravity will give a rough idea of the amount of dehydration in case of intestinal obstruction.

(iii) Diagnostic aspiration

So important in the distinction between simple and strangulated obstruction, aspiration of peritoneal cavity with a fine needle in case of doubt and withdraw of blood stained fluid is diagnostic. of strangulation. Root finds that significant increase in the polymorphonuclear count of peritoneal fluid can be detected within three hours after peritoneal

invasion.

X-ray diagnosis

In all cases, ‘Scout’ film of the abdomen, with the patient standing and lying down must be taken. No enema should be given prior to plain X-ray because it may cause false fluid levels. The sensitivity of abdominal radiographs in the detection of small bowel obstruction ranges from 70 to 80%.(14)

The finding in erect abdomen X-ray for small bowel obstruction is the triad of dilated small bowel loops (>3 cm in diameter) air fluid levels seen on upright films and a paucity of air in the colon. (15).

Gas shadows : When the jejunum, ileum or the colon is distended with gas, each structure has significant radiological pictures. Jejunum is characterised by ‘valvulaeconniventes’ that pass from anti-mesenteric to mesenteric border in the regular fashion. Ileum radiography was described by Wangensteen as being ‘characterless’.

Large intestine shows haustral markings which unlike the valvulaeconniventes are spaced irregularly, do not completely traverse the circumference of the bowel and gas shadows of large intestine are located peripherally.

Fluid levels: In adults, two inconstant fluid levels are regarded as physiological, one at the duodenal cap and the other within the terminal

ileum. In obstruction, fluid levels appear later than the gas shadows; nearer the obstruction the ileocaecal valve fluid level is proportional to the degree of obstruction and to its site in small intestine. In upper small bowel obstruction, fluid levels will be in left upper quadrant and there will be few in number, multiple fluid level seen all over the abdomen in case of low small bowel obstruction. The presence of gas in the wall of the bowel is highly significant sign of intestinal necrosis, which was demonstrated by Schorr in 1963.

Volvulus of sigmoid shows greatly distended sigmoid loop filling the whole of the abdomen upto the diaphragm with the "Bent inner tube sign". Millin and Righler pointed out that "coffee bean" sign in pathognomonic sign of caecal volvulus.

Barium enema: In intussusception, barium is seen as a 'claw' around a negative shadow of intussusception whereas in sigmoid volvulus, barium column ends at the level of the distal sigmoid torsion in a characteristic Twisted Bird's Beak deformity.

Computerized tomography (CT)

CT demonstrates the cause of obstruction. Computerized tomography is very much useful in revealing the site, level and cause of obstruction and in displaying signs of threatened bowel viability. CT is most valuable when there are systemic signs suggestive of infarction, an

associated palpable mass. In these cases CT may confirm the presumptive diagnosis or reveal other causes such as appendicitis or diverticulitis.

It is a procedure of choice in patient who have a history of abdominal malignancy and clinical symptoms suggestive of bowel obstruction.In strangulated obstruction, target sign or pneumatosis and haemorrhage in the mesentery can be seen.

TREATMENT OF ACUTE INTESTINAL OBSTRUCTION

With some exceptions, an urgent surgical intervention is needed in a case of intestinal obstruction. Although it is difficult to differentiate between a simple and strangulated obstruction, the assessment of patient is done by taking detail history and clinical examination. Investigations are to find out whether the obstruction is mechanical or a dynamic and the level of obstruction.

The treatment has to be planned accordingly to the above assessment which includes supportive management and surgical management. There are four main measures in management of obstruction

- GI decompression
- Fluid and electrolyte replacement
- Relief of obstruction usually surgical
- Antibiotics to prevent complications from associated sepsis

The first two steps are always necessary prior to surgical relief of obstruction and are main stay of post-operative treatment. Surgical treatment is necessary for most cases of intestinal obstruction, but should be· delayed until resuscitation is complete provided there are no sign of strangulation or evidence of closed loop obstruction.

Conservative management

Patients with a partial intestinal obstruction may be treated conservatively with resuscitation and tube decompression alone. Resolution of symptoms and discharge without the need for surgery have been reported in 60-85% of patients with a partial obstruction.(16).

Simple obstruction caused by postoperative early adhesions or kinking may resolve spontaneously with conservative management and it is indicated in following conditions.

- Postoperative early adhesions
- Paralytic ileus of non-paralytic origin
- Inflammatory condition causing obstruction
- Obstruction due to worm impaction

The initial conservative management is used in above said conditions,facilitates spontaneous relief of obstruction and avoids more of adhesion formation due to surgery. The decision to intervene depends largely on the underlying cause, clinician involved and general condition

of the patient. The conservative management includes.

- GI decompression
- Fluid and electrolyte replacement
- Antibiotics to prevent complications

GI decompression

There are two types of aspiration tubes used in GI decompression and the decompression is achieved by the passage of non-vented (Ryle's tube) or vented Salem tube which are normally placed on free drainage with hourly aspiration but may be placed on continuous or intermittent suction decompression of the bowel proximal to obstruction and stomach will relieve certain amount of distension and toxic fluid accumulated in the bowel. Also avoids the aspiration pneumonia during induction of anaesthesia. It is also improves local bowel circulation and venous returnto the heart by relieving the pressure over the IVC. There are other special tubes used in decompression of the small bowel (long intestinal tube).

Fluid and electrolyte replacement

The individual patient has to be assessed according to general condition of the patient and underlying cause of obstruction. This should be routine in all cases of bowel obstruction before taking up for surgical intervention except in few case of early simple obstruction, which is

within 24 hours. The longer the duration of obstruction longer will be the time taken to get the patient ready for surgery. It is best to intervene when vital signs show a return to normal. It is unwise to operate early ina case of prolonged obstruction with poor general condition and it is also not wise todelay in case of strangulated and close loop obstruction.

The parameters like pulse rate, BP, Shock state, Degree of dehydration, Urine Output, Initial haematocrit value are taken into consideration in fluid management.

Electrolytes

The fluid loss should be corrected by colloidal replacement such as blood plasma, in most cases hyper or hyponatraemia which can be corrected by replacement of Hartman's solution or normal saline. The sodium deficit is estimated by multiplying the decrease in sodium concentration between the normal with total body water in liters. The composition of Ringer's lactate is almost as that of plasma and considered as physiologic and can be used to replace ECF and GI losses, in the absence of gross abnormalities of concentration and composition, if gastric juice loss is prominent, normal saline is used, the sodium chloride required on an average 80-110µ mols and is provided by 570 ml of isotonic saline solution. The KCI necessary for replacement should not be given until the normal renal output is established. Acid base balance is

corrected depending upon their determination. All patients with intestinal obstruction should have a central venous catheter in situ in superior vena cava, for frequent measurement of CVP and indwelling catheter into the bladder for measurement of urine output.

Antibiotics

Use of broad-spectrum antibiotics in adequate doses along with metranidazole are advised. It is important to give antibiotics pre and postoperatively till adequate recovery takes place.

Surgical Management

With regard to the timing of surgery, all patients should be operated on promptly after volume resuscitation if any evidence or suspicion araises that bowel is ischaemic.(17). Early operation indicated in (1) obstructed and strangulated hernia, (2) internal intestinal strangulation (3) acute obstruction, The classical clinical saying that the sun should not set and rise in case of unrelieved intestinal obstruction is sound and should be followed.

Laparotomy

In patients with small bowel obstruction who have not had previous abdominal surgery or in those with clinical evidence of ischaemia, a laparotomy is mandatory.(18)

When the cause of obstruction lies within the abdomen and but its

site is doubtful, right paramedian incision is advised, if left sided colonic obstruction is defined left mid or lower paramedian incision preferred, abdominal cavity is inspected which indicates the underlying pathology. Haemorrhagic fluid denotes strangulation; clear straw-coloured fluid denotes simple obstruction. The operative assessment is directed to:

- Site of obstruction
- Viability of the gut
- The nature of obstruction

The type of surgical procedure required will depend upon the nature of the cause, following relief of obstruction the viability of the involved bowel should be carefully assessed. In case of viable bowel, peritoneum will be shiny, mesentery bleeds on prick whereas nonviable bowel, peritoneum is lusterless, mesentery does not bleed on prick.

Doppler sonography can also help to test the circulation in the mesenteric vasculature (a most accurate method of testing viability). If viability of the bowel is in doubt, it should be dropped in warm moist pads for ten minutes along with 100% oxygen in the anaesthetic gas. Then after ten minutes it has to be reassessed, in doubtful cases resection has to be done.

PRINCIPLES OF LARGE BOWEL OBSTRUCTION

As most of the large bowel obstruction are due to malignancy, volvulus or secondary to adhesive bands, which commonly occur, in elderly patient. When lesion is operable and found in the caecum, ascending colon or proximal transverse colon, an emergency right hemicolectomy should be performed; if lesion is fixed a proximal stoma (colostomy. or ileostomy if ileocaecal wall is incompetent) or an ileo transverse colon bypass has to be considered, whereas obstruction lesions, of splenic flexure (malignant) should be treated by an extended right hemicolectomy. Ifone stage resection anastomosis is feasible a covering colostomy to protect the site of anastomosis is safe, where the distal segment could not be brought to the surface a proximal stoma and the distal end closed and returned to abdomen (Hartman's procedure) or both the ends brought outside, proximal as stoma and distal as mucus fistula, followed by second stage colorectal anastomosis can be planned when patient is fit. In very old or enfeebled patients when an obstructing carcinoma of rectum is fixed, left iliac colostomy is the best site for a permanent artificial anus. In rare circumstances, or if caecal perforation is suspected, wait for some time for improving the patient's condition and later relieving of obstruction can be done by doing an emergency caecostomy through a small incision in right iliac fossa.

OBSTRUCTION BY ADHESIONS AND BAND

It is the second most common cause of intestinal obstruction in developing countries.

The pathology lies with peritoneal irritation results in local outpouring of fibrin whichproduces adhesions between opposed peritoneal surfaces. The fibrinous adhesions may become vascularised and become mature fibrous tissue. Infection being an important cause. Also foreign materials like silk thread, barium sulphate, talc, results in fibrous formation.

These commonly occurs following laparotomy surgeries. Once adhesions have developed, progression to obstruction is inevitable in a significant proportion.Ileum is the commonest segment to be obstructed due to adhesions. After abdominal surgeries, about 5% of the cases subsequently develop obstruction due to adhesions, whereas operation in the colon carry high incidence of obstruction, about $1/5^{th}$cases. About 20% of the obstruction occurs in the first year after laparotomy and most of these occur during first few weeks after surgery and termed as early postoperative obstructions and most resolved by conservative treatment.

Type 1: Postoperative fibrinous adhesions cause incomplete obstruction. This occurs between 3-6 postoperative day. Also called as bread and butter fibrinous adhesions. These types of adhesions are

managed conservatively.

Type 2: Strong bands of postoperative fibrous adhesions and occur at an organ where there is deficient blood supply and takes additional blood supply through these adhesions, and these may cause intestinal obstruction at any time after abdominal operations.

Type 3: Adhesion of loops of intestine to an inflamed intraperitoneal structure, e.g. Tubercular mesenteric lymphadenitis, Salpingitis.

Type 4: This follows chemical irritation from material such as talc glove powder or powdered antibiotics placed during laparotomy.

Prevention for adhesion

The following factors may limit adhesions formation:

- A good surgical technique.
- Minimal contact with gauze
- Washing of the peritoneal cavity with saline to remove clots
- Covering the anastamotic and raw peritoneal surface with omentum.
- Many substances have been instilled in the peritoneal cavity to prevent adhesionformation like hyalurinidase, hydrocortisone, silicon, dextron, polylyvinyl propylene (PVP) chondritin, streptopyris and anticoagulants, antihistamine, NSAIDs, streptokinase. But no single agent found to be safe and effective.

Treatment

The treatment for adhesion is same as the general principles of management often it is curative in early type of adhesions, but conservative treatment should not prolong beyond 48-72 hours and should not be continue if symptoms and signs are progressive even after initial resuscitation. When laparotomy is done multiple adhesiolysis can be done.

Recurrent intestinal obstruction due to adhesion

Adhesions are a major cause of late morbidity. Approximately 10% of all patients who have laparotomy for adhesive obstruction will require a further operation at a later date for the same problem. A further 10% may require a third operation for adhesive obstruction.

There are often chances of recurrent obstruction after first adhesiolysis. The following procedures may be considered for recurrent obstruction

- Intestinal intubation
- Charles Phillips transmesenteric plication
- Noble plication
- Repeat adhesiolysis (enterolysis)

Noble plication

This procedure is time consuming and it involves release of all the involved coils of intestine are freed and adjacent coils are sutured with

serosal sutures to form gentle curves of average length of 15-20 cm. The mesentery of the corresponding bowel is sutured to prevent internal herniation.

Charles Phillips Operation

In this procedure, after adhesiolysis the bowel is placed in an orderly fashion and three synthetic sutures are passed through the mesentery of the plicated bowel, each doubled back upon itself and tied loosely. Stitches should pass a few centimeters away from the bowel wall. The resulting bowel should look like a pocket of sausages.

Intraluminal tube insertion

Several tubes are introduced into the small bowel through jejunostomy proximal to obstruction. Baker's tube or Miller Abbot tube is commonly used. The tubes are left in situ for 10-15 days. The tube is introduced to small bowel through proximal jejunostomyupto the caecum, where the balloon attached to this tube is distended and the proximal end of the tube is connected to a sterile drainage bag and drainage continued till the patient passes flatus. After 2 weeks the tube is graduallywithdrawn after deflating the balloon under sedation. This jejunostomy site heals by granulation tissue. The tube facilitates the formation of gentle smooth curved of small bowel. This technique was successful in relieving the obstruction in 80% of cases.

Internal hernias

When a segment of the small intestine herniates into one of the retroperitoneal fossae it is termed as internal hernias. This can occur in following sites:

- Supravesical hernia
- Foramen of Winslow
- Diaphragmatic hernia: Acquired/Congenital.
- Caecal or appendicaecal: Retroperitoneal fossae superior or retrocaecal
- A hole in the mesentery or mesocolon and defects in the broad ligament.
- Paraduodenal fossae: Right/left paraduodenal fossae.

  Internal herniation in the absence of adhesions is uncommon and a preoperative diagnosis is unusual. The standard treatment for a hernia is to release the constricting agent by divison. Internal herniations is treated by laparotomy and release of constriction ring. The distended loop first to be decompressed and reduced. Unviable segment of the bowel has to be resected and anastomosed.

Supravesical hernia

This hernia develops in a sac that is produced by a protrusion of the peritoneum of the supravesical fossa which extends into the

prevesicalspace. The symptoms occur because a loop of bowel becomes trapped in the hernial sac, producing a small bowel obstruction. Treatment consists of reducing the small bowel into the peritoneal cavity if possible and closing the neck of the sac. If reduction is impossible the neck of the sac may be incised to allow the small bowel to be reduced.

Foramen of Winslow hernia

The small bowel will herniate into the foramen but occasionally large bowel, particularly the mobile caecum. A preoperative diagnosis is usually difficult, at operation the findings are of bowel disappearing through the foramen of Winslow. Gentle traction on the small bowel associated with stretching of the mouth of the foramen allows the reduction of the hernia. If reduction is not possible, mobilization of the second part of the duodenum and head of thepancreas which allows much wider opening for reduction.

Paraduodenal hernia

The most common variety is the left duodenojejunal hernia. The proximal small bowel migrates retroperitoneally through the left duodenojejunal fossa and comes to lie behind the descending mesocolon and inferior mesenteric vessels. If the small bowel cannot be reduced through duodenojejunal fossa, inferior mesenteric vessels can be divided without compromising the circulation of left colon. Any adhesions that

hold the small bowel within the space behind the descending mesocolon are divided and the bowel is reduced through the duodenojejunal fossa.

Diaphragmatic hernia

Congenital	Acquired
1. Through oesophageal hiatus	1. Oesophageal hiatus hernia (98%)
2. Through foramen of Morgagni	2. Traumatic
3. Through foramen of Bochladek	3. Postoperative
4. Posterior hernia	
5. Eventration	

Treatment – Surgical repair is the treatment of choice and usually performed through

a low thoracotomy incision. The defect is closed by two layered non absorbable suturing.

VOLVULUS

Volvulus is axial rotation of a portion of alimentary tract on its mesentery. It may be primary or secondary. Primary volvulus occurs due to malrotation of the gut, abnormal mesenteric attachments or congenital bands. E.g. Caecal volvulus, volvulus neonatorum and sigmoid volvulus.

A secondary volvulus is due to actual rotation of a segment of bowel around an acquired adhesion or stoma. When it is completed, it forms a close loop of obstruction with ischaemia of the segment involved.

PRIMARY VOLVULUS

Volvulus neonatorum

Predisposed by arrested rotation of the gut causing narrow mesentery of the small bowel and caecum. Symptoms and signs are pain abdomen, bilious vomiting. On laparotomy, the whole of the midgut has to be delivered out. Volvulus usually occurs in clockwise direction, which has to be untwisted and if any secondary obstructing lesions such as congenital bands (transduodenal band of ladd) have to be released and Ladd procedure is performed. In this procedure, duodenum and upper jejunum is repositioned on the right side of the abdomen.

Small bowel volvulus

It usually occurs in the lower ileum, it may be spontaneous after consumption of plenty of vegetable food material or it may be due to adhesion passing to pelvic parities or pelvic organs in female. Treatment consists of laparotomy, derotation and causative adhesions has to be removed.

Caecal volvulus

It may occur when the right half of colon is lax and mobile. More common in female with classical features of obstruction like pain abdomen, nausea, vomiting, constipation. At first, it may be partial rotation and spontaneously released by passage of flatus and faeces.

Barium enema may show a “bird beak” appearance and absence of barium in the caecum. At laparotomy volvulus can be treated by caecal needle decompression followed by fixation of caecum to right iliac fossa or if caecum is gangrenous or perforated right hemicolectomy is carried out.

Sigmoid volvulus (19)

Sigmoid volvulus is a serious condition in which a redundant sigmoid loop rotates around its narrow, elongated mesentry, producing ischaemia and necrosis of the sigmoid colon, followed by rapid distension of the closed loop(26). Volvulus of the pelvic colon is more common in Eastern Europe, Russia and Mrica. It is rare in USA and Western Europe. In India, it is common in northern states like Punjab, UP, MP, Bihar and Maharashtra. This is rotation of sigmoid loop of the colon over mesentery, commonly occurs in adults and elderly males and one of the commonest causes of large bowel obstruction.

Aetiology includes high residue diet with physical inactivity, chronic constipation, psychiatric problems, prolonged use of sedatives, addiction to laxatives and acute psychotic drugs. Males are more affected who are middle aged or elderly. More prevalent in under developed and malnourished people. Pathology may be due to long pelvic meso colon, adhesions over the sigmoid colon, overloaded pelvic colon. twist has odd turns, ½, 1½, 2½, etc. After the loop has rotated 1½ turns the

veins involved in the torsion are compressed and the loop becomes greatly congested and if rotation is greater than 1½ times loop becomes gangrenous. It presents with signs and symptoms of acute large bowel obstruction, which may be intermittent initially and followed by passage of large quantity of faeces and flatus. Plain radiography of the abdomen shows an inverted “U” shaped shadow of distended bowel loop (or coffee bean appearance) that runs from right to left with two fluid levels one in each loop.

Management

Consists of conservative or surgical measures. Surgery is mandatory, if gangrene is suspected, resection anastamosis has to be carried out.

Conservative treatment

Sigmoidoscopy should be carried out, if the obstruction is reached a soft rectal tube is attempted tobe put into the twisted gut. This will immediately deflate the gut and surgery can be delayed until the patient is fit for surgery.

a. Oral medication

Oral liquid paraffin, 30-60 ml repeatedly given, it is said that liquid paraffin leads to relief of tension or oedema at the site of twist, thus loosening the constriction with further deflation, finally peristaltic wave

may untwist the colon.

b. Enema

Untwisting phenomena may occur if enema given in early stages.

Surgical treatment

In patients with sigmoid volvulus who do not have peritonitis on presentation, recurrence preventing surgery may be performed with minimal mortality rates.

After laparotomy through midline or left paramedian incision, the loop may be untwisted and deflation of the distended loop can be done through a stab incision under cover of purse string suture. The gangrenous bowel has to be resected and following procedure can be done.

a. Primary anastomosis after resection of gangrenous segment with proximal covering colostomy.
b. A Paul Mickulicz type double barrel colostomy.
c. Hartman's procedure: Proximal end colostomy with closure of the end of the distal colon or rectum in two layers and dropped into abdominal cavity. Later anastomosis can be done after one month.

Resective procedure

a. Resection with Paul Mickulicz procedures or Hartman's procedures followed by delayed anastomosis.
b. Resection and primary anastomosis: The redundant loop of sigmoid

colon can be resected and primary anastomosis can be done.

The mortality of patients with sigmoid volvulus treated surgically is closely related to the disease stage, a prompt surgical timing, the patient functional status and his collaboration with clinicians is essential in order to define a correct diagnosis and treatment.(28)

Non-resective procedures

a. **Mesocolopexy:** Plication of sigmoid colon by various methods, which make the long mesocolon into more broad based and shorter length, which avoids the torsion of the sigmoid colon on its mesentery Hall and Cragg's type, Prasad and Tiwari type had been advocated.

b. **Sigmoidopexy:** Involves fixation of sigmoid loop to the posterior wall of theabdomen or to the parietal peritoneum or to the transverse colon. The principle behind fixing the sigmoid loop to a various places is to curtail its free movements, which will avoid it from twisting.

c. **Extraperitonealization of sigmoid colon:** Sigmoid loop is placed in the space between lower abdominal peritoneum and abdominal musculature which may prevent or avoids rotation movements of the alimentary tract.

Ileosigmoid volvulus (compound volvulus)

Also known as ileosigmoidknotting.A rare condition where the long pelvic mesocolon allow the loop of ileum to twist over the root of

mesocolon of sigmoid resulting in gangrene of either or both segments of the bowel. On laparotomy depending upon the viability of the bowel loop, decompression and if gangrenous, resection anastamosis of the sigmoid volvulus has to be done.

SIMPLE VERSUS STRANGULATING OBSTRUCTION

Obstructed external hernia are the most common cause of intestinal obstruction in developing countries. Most patients with small bowel obstruction have simple obstructions that involve mechanical blockage of the flow of luminal contents.There is no compromised viability of the intestinal wall.But strangulating obstruction is obstruction with compromised blood flow.(25% patients of small bowel obstruction). Strangulation obstruction, commonly by incarceration of inguinal, femoral, epigastric, paraumbilical hernia can cause closed loop bowel obstruction in which the vascular supply is compromised, and lead to ischemia and gangrene.

In view of increased risk in strangulation early detection is required. Classic signs of strangulation have been described and include tachycardia, fever, leukocytosis, and a constant, noncramping abdominal pain. No clinical parameters or laboratory measurements can accurately detect or exclude the presence of strangulation in all cases. Hernias remain the most frequent cause of strangulation in patients presenting with this

condition.

Many laboratory tests, such as lactate dehydrogenase, amylase,ALP, and ammonia , have shown no real benefit.Some limited success in discriminating strangulation by measuring serum d-lactate, creatine phosphokinase isoenzyme (particularly the BB isoenzyme), or intestinal fatty acid–binding protein were described.A superconducting quantum interference (SQUID) magnetometer, a noninvasive method to detect mesenteric ischemia is used. Intestinal ischemia causesalterations in the basic electrical rhythm of the small intestine. Bowel ischemia and strangulation cannot be reliably diagnosed or excluded preoperatively in all cases by any known parameter, combination of parameters, or current laboratory and radiographic examinations.

Treatment

Fluid Resuscitation and Antibiotics

Patients with intestinal obstruction are usually dehydrated and depleted of sodium, chloride, and potassium, requiring aggressive intravenous (IV) replacement with an isotonic saline solution such as lactated Ringer's. Urine output should be monitored . On attaining adequate urine output, potassium chloride should be added to the infusion as needed. Serial electrolyte measurements, as well as hematocrit and white blood cell count, are needed to assess the adequacy of fluid

repletion. Broad-spectrum antibiotics are given prophylactically

Tube Decompression

Another important adjunct to the supportive care of patients with intestinal obstruction is nasogastric suction. This empties the stomach, reducing the hazard of pulmonary aspiration of vomitus and limiting further intestinal distention from preoperatively swallowed air. The use of long intestinal tubes (e.g., Cantor or Baker tubes) can be used. Patients with a partial intestinal obstruction may be treated conservatively with resuscitation and tube decompression alone. Enteroclysishelps in determining the degree of obstruction, higher-grade partial obstructions requiring earlier operative intervention. Even though an initial trial of nonoperative management of most patients with partial small bowel obstruction is warranted, clinical deterioration of patient or increasing small bowel distention on abdominal radiographs during tube decompression warrants prompt operative intervention.

Operative Management

A patient with a complete small bowel obstruction requires surgical intervention. A nonoperativeapproachhasbeen proposed by some, with arguements that prolonged intubation is safe in these patients ,such that no fever, tachycardia, tenderness, or leukocytosis is noted. Nevertheless, nonoperative management of these patients is undertaken at a risk of

overlooking an underlying strangulation obstruction and delaying the treatment of intestinal strangulation until after the injury becomes irreversible. Adhesive band intestinal obstruction may be treated with lysis of adhesions. Gentle handling of the bowel to reduce serosal trauma and avoiding unnecessary dissection and inadvertent enterotomies is essential. The hernia may be a groin, epigastric or paraumbilical hernia or it may be an incisional or parastomal hernia, the operative approach and initial dissection is similar to that in elective hernia repair. Incarcerated hernias can be managed by manual reduction of the herniated segment of bowel and closure of the defect. The treatment of patients with an obstruction and a history of malignant tumors can be undertaken by bypassing the obstructing lesion, itmay offer the best option than a long and complicated operation that may require bowel resection. An obstruction due to Crohn's disease will often resolve with conservative management if theobstruction is acute. In case of chronic fibrotic stricture being the cause of the obstruction, then a resection or stricturoplasty may be required. It can sometimes be difficult to evaluate bowel viability after the release of a strangulation. With questionable intestinal viability , the bowel segment should be completely released and placed in a warm, saline-moistened sponge for 15 to 20 minutes and then re-examined along with 100% oxygen administration.If normal color has returned and

peristalsis is evident, it is safe to avoid resection. In difficult borderline cases, fluorescein fluorescence may supplement clinical judgment. Second look laparotomy within 18 to 24 hours helps assess viability of bowel.

TUBERCULOSIS OF INTESTINE

Intestinal obstruction is the most common complication in the small bowel, affecting 60% of the patients with tuberculous enteritis. Common site is ileum, proximal colon and peritoneum. Approximately, 75% of patients with tuberculosis enteritis have involvement of the distal small bowel and ileocaecalregion(33).

Intestinal obstruction is the most common complication in the small bowel, affecting 60% of the patients with tuberculum enteritis.

There are two principal types:

1. Hyperplastic tuberculosis

The infection establishes itself in lymphoid follicles and the resulting chronic inflammation causes thickening of the intestinal wall and narrowing of the lumen. Pain abdomen with intermittent diarrhoea are usual symptoms. Sometimes mass in the right iliac fossa. In non-obstructed patients, treatment is with antituberculosis drugs and in patients with intestinal obstruction the presentation will be subacute intestinal obstruction which should be managed by resection of ileocaecal segment. When the patient presents with acute intestinal obstruction is treated with

ileotransverseanastomosis.

2. Ulcerative tuberculosis

In this condition there are multiple ulcers in the terminal ileum, serosalthickening, reddened and covered in tubercle. These patients are treated with antitubercular treatment, presents with intestinal obstruction secondary to stricture. There are treated by stricturoplasty or by resection of segment of bowel which contains multiple stricture or long segment stricture.

Other forms of strictures

Crohn's disease is among the most common aetiology of small intestinalstricture. Certain drugs are known to cause mucosal ulceration and strictures, drugs most notably are enteric coated potassium chloride preparations and NSAIDs.

Radiation therapy for intraperitoneal malignancy can lead to strictures, mesenteric ischaemia can lead to stricture formation, the distal ileum being at greatest risk as the ileocolic artery is the last branch of superior mesenteric artery. Various neoplasms including carcinoma, carcinoid, lymphoma can cause stricture within small intestine. Resection is the treatment whenever technically feasible.

GALL STONE ILEUS

Gall stone ileus accounts for 1-2% of cases of intestinal obstruction

usually occurs in older age group. To cause obstruction gall stone must be larger than 2.5 cm and enter the intestinal tract by a process of ulceration. The stone passes through duodenum, jejunum and colon. Obstruction is cause at distal ileum or at other areas of narrowing. The diagnosis is done by the presence of air in the biliary tree along with the signs of intestinal obstruction in scout film. Treatment is removal of stone via enterotomy or resection, if stone is severely impacted.

NEOPLASMS

Extrinsic tumour involvement from secondary spread is more likely causes of obstructing the lumen or by acting as nidus for intussusception. Although benign tumours are predisposing conditions for intussusception, malignant tumours like adeno carcinoma lymphomas and carcinoids rarely give rise to obstruction, wide resection and end-to-end anastamosis is the treatment.

Foreign bodies and bezoars

Luminal obstruction by the ingestion of foreign body commonly in children and psychotic patients. Bezoar may migrate into the small intestine causing obstruction. Small bowel obstruction can occur from bezoar arising from intestinal diverticulum. Bezoar or foreign body get impacted at the site where bowel is narrowed by previous surgery. Treatment is removal of foreign body or bezoar by enterotomy.

LARGE BOWEL OBSTRUCTION (29)

Colorectal cancer is the single most common cause of large intestinal obstruction in the United States, whereas colonic volvulus is the more common cause in Russia, Eastern Europe, and Africa. Intraluminal causes of colorectal obstruction include fecal impaction, inspissated barium, and foreign bodies. Intramural causes, include inflammation (diverticulitis, Crohn's disease, lymphogranulomavenereum, tuberculosis, and schistosomiasis), Hirschsprung's disease (aganglionosis), ischemia, radiation, intussusception, and anastomotic stricture. Extraluminal causes include adhesions (a common cause of small bowel obstruction, but rarely a cause of colonic obstruction), hernias, tumors in adjacent organs, abscesses, and volvulus

ETIOLOGY

CARCINOMA OF THE COLON

Carcinoma is the most frequent cause of large-bowel obstruction in developedcountries . The left colon is the most likely site of obstruction and the extraperitonealrectum the least. Signs of partial obstruction progress to those of complete obstructionwhen the narrowed colonic lumen is occluded by a fecal bolus. Since the right colon has semiliquid contents and a relatively wide lumen, obstruction occurs late in this segment and may be acute in its presentation, especially if the

ileocecalvalve is competent. The operative risk is increased considerably when perforation is present.

Signs and symptoms

The signs and symptoms of large bowel obstruction depend on the cause and location of the obstruction. Cancers arising in the rectum or left colon are more likely to obstruct than those arising in the more capacious proximal colon. Regardless of the cause of the blockage, the clinical manifestations of large bowel obstruction include the failure to pass stool and flatus associated with increasing abdominal distention and cramping abdominal pain. Colonic obstruction is associated with potentially serious complications such as perforation, only 4% of the tumours of the colon presnt with the perforation of the bowel, and the timing and selection of appropriate operative procedures are important. Symptoms can develop slowly and progressively or fulminantly. Among adults, elderly people are usually affected. The sigmoid colon is the usual site: this portion of the intestine is thick walled, not particularly distensible, and comparatively narrow.

Management

All patients with complete acute large bowel obstruction require prompt surgical intervention and should not undergo a trial of nonoperative management. Nasogastric decompression is also important

in patients with a large bowel obstruction to decrease the amount of air and gastric contents delivered to the bowel. Nasogastric decompression will help relieve intraluminal pressure, prevent further dilation of the proximal bowel, and possibly decrease the risk of perforation.

Antibiotics targeted at both skin and colonic flora should be administered. Exploration in patients with large bowel obstructions is best performed through a low midline incision. Patients with large bowel obstructions should be placed in the lithotomy or modified lithotomy position if access to the anus is anticipated. Obstructing lesions of the cecum and ascending colon should be resected via right hemicolectomy, usually with a primary anastomosis. Lesions in the transverse colon should be managed with an extended right hemicolectomy and again, with a primary anastomosis. Proximal diversion with an end ileostomy is not necessary in all patients; however, proximal diversion should be considered when there is any concern about bowel viability, if the patient is unstable, or in the case of substantial peritoneal contamination or peritonitis.

The management of obstructing lesions in the descending and sigmoid colon is a more classic approach with a Hartmann's procedure of segmental resection of the affected colon, an end colostomy, and a blind distal pouch or mucous fistula. An end colostomy at the time of operation

is safe and may decrease the incidence ofperioperative complications compared to an on-the-table bowel preparation with primary anastomosis.

Another option to consider in the early management of the patient with an obstructing lesion in the large bowel is the use of a self-expanding intraluminal metal stent (SEMS) to allow immediate colonic decompression and the ability to perform elective mechanical bowel preparation.The use of SEMS is becoming widely available and it can be a useful tool for the surgeon managing a large bowel obstruction. In experienced hands, a SEMS can be placed successfully in about 90% of patients with low complication rates. A SEMS can avoid the need for urgent or emergent operation by intraluminally decompressing the distended proximal colonand allowing distal passage of stool. A SEMS is also useful when palliating patients who might not tolerate surgical diversion or those with unresectable disease and a limited survival. With a locally advanced obstructing rectal cancer, after placement of a SEMS, the patient can undergo neoadjuvant therapy followed by surgical resection, again increasing the chances for a successful one-stage operation.

The cause of the obstruction needs to be managed individually. Thus a hernia is repaired once the obstructed colon is reduced, whereas an intussusception is reduced, with resection of the involved colon if necessary. Because it is so common, fecal impaction should be ruled out

in institutionalized or debilitated elderly patients.The cecum must be visualized to assess its viability. If an anastomosis is to be performed, the criteria for a good outcome must be met.

ADYNAMIC OBSTRUCTION

Adynamic obstruction, in which peristalsis may be absent as in paralytic ileus or it may be present in a non propulsive form as in mesenteric vascular occlusion or pseudoobstruction.

Paralytic Ileus

A paralytic ileus is defined as functionally impaired transit of intestinal contents because of decreased peristaltic activity of the gastrointestinal tract, in the absence of mechanical obstruction(34). An ileus can result from a number of causes.including drug induced, metabolic, neurogenic, and infectious.Patientshaving ileus continues to pass flatus and diarrheaoccassionally, and this

Helpsin distinguishishing these patients from those with a mechanical small bowel obstruction.Xray studies can help in distinguishing ileus from small bowel obstruction. Plain abdominal xrays reveals distended small bowel as well as large bowel loops.

Treating an ileus is supportive with nasogastric decompression and IV fluids. The most effective treatment to correct the underlying condition may be aggressive treatment of the sepsis, correction of metabolic or

electrolyte abnormalities, and stopping of medications that produce an ileus

Mesenteric Vascular Occlusion

Arterial embolism is more common than spontaneous thrombosis and the superior mesenteric vessels are implicated more frequently. Possible source of emboli include atrial fibrillation, a mural myocardial infarct, an atheromatous plaque or aneurysm, a vegetation of mitral valve, pulmonary vein thrombosis. The ischaemia of the bowel may be due to arterial or venous, the intestines and its mesentery. The intestines and its mesentery become swollen and oedematous, demarcation between healthy and infracted bowel being gradual. Blood stained fluid is exuded into the peritoneal cavity and the lumen of the infarcted bowel, which becomes filled with blood. Acute mesenteric ischaemia is a highly morbid event with reported mortality rates exceeding 60%. When the main branch of superior mesenteric artery is occluded, the whole of small intestine, caecum and a part of the ascending colon become infarcted.

Mesenteric vascular occlusion should be suspected, when a patient beyond middle age, giving a history of cardiac disease, it is suddenly seized with acute abdominal pain that is, however not colicky in character. He collapses and passes blood stained stools.

Clinical features

(i) Pain which is central abdominal in nature

(ii) Gastrointestinal emptying with persistent vomiting.

Investigations

Mesenteric angiography is a definitive diagnostic study. Duplex ultrasonography may be of some benefit in visualizing flow in the SMA. Ogata et al.reported that a kinetic dilated loop observed on real time ultrasonography has a high sensitivity 90% and specificity 93% for the recognition of strangulation. The positive predictive value was 73%.(1)

CT abdomen and pelvis shows focal or segmental bowel wall thickening.(39)

Treatment

Regardless of aetiology, the prognosis of patient with mesenteric ischaemia is dependent upon rapid diagnosis and initiation of treatment. Conservative management may be sufficient in selected cases; more often laparotomy is required and can be life saving.(40)Superior mesenteric embolectomy should be attempted and thrombo-endarterectomy or a bypass procedure from aorta or iliac arteries or the splenic artery to the more distal arterial surgery in mesenteric vascular occlusion. In late cases, the affected gangrenous bowel should be resected. Low molecular weight dextran protects the bowelagainst infarction, when superior mesenteric

artery is occluded. Lodgement of an embolus in themiddle colic artery should be treated by resection of the transverse colon with exteriorisationofboth ends.

Acute Colonic Pseudo-Obstruction

Acute colonic pseudo-obstruction also known as Ogilvie's syndrome is an often painless paralytic ileus of the large bowel characterised by rapidly progressive abdominal distension. Acute pseudo-obstruction of the colon is often suspected based both on abdominal radiography and the clinical setting, but it remains a diagnosis of exclusion.

Patient requires semi-emergent operative intervention, but only after appropriate resuscitation. Exclusion of a mechanical obstruction can be accomplished via either a careful, complete colonoscopy minimizing air insufflation, or by demonstrating freeretrograde flow of contrast without obstruction to the cecum on water-soluble enema. These water-soluble contrast enemas can also be therapeutic; some patients achieve decompression after contrast instillation because of stimulation of defecation. Initial management of pseudo-obstruction is the same as that for small bowel obstruction and includes placement of a nasogastric tube, nothing by mouth, rehydration, correction of electrolyte abnormalities, and if possible, discontinuation of narcotics.

The decision to pursue colonic decompression is based in part on the cecaldiameter as determined by abdominal radiographs, on symptomatology, and on the duration of obstruction. In general, the maximum accepted safe cecal diameter is12 cm; above this diameter, the risk of perforation increases substantially. When the cecum is less than 12 cm in diameter and the patient is not distressed, initial treatment should be continued, as immediate decompression is not necessary. If at any time during therapy the patient develops peritonitis or signs of ischemia, emergency surgical therapy is indicated.

Acute colonic pseudo-obstruction, with a markedly dilated proximal colon; endoscopy confirmed no distal obstruction, When the cecal diameter exceeds 12 cm or the patient has a significant amount of abdominal discomfort, decompression is indicated. The two methods of colonic decompression include intravenous neostigmineandcolonoscopic decompression. Neostigmine is the first line of treatment in the patient without contraindications. Neostigmine has been shown to be effective for treatment of acute colonic pseudo-obstruction, achieving success in about 90% of patients.

Colonoscopic decompression, once the first line of therapy for acute colonic pseudo-obstruction, is now considered the second-line treatment for patients with uncomplicated pseudo-obstruction.

Colonoscopicdecompression is indicated in those patients with contraindications to neostigmine administration or those who have failed neostigmine therapy. Another role for colonoscopy is to exclude a source of mechanical obstruction, in which case it may be both diagnostic and therapeutic. A long, large-diameter colonic tube can be placed at the time of colonoscopy, and although controversial, may aid in further decompression in those patients with refractory pseudo-obstruction. Most surgeons, however, do not place a long colonic tube at the time of first colonoscopic decompression, but do leave a tube should the patient require another colonoscopic decompression for recurrent obstruction. When a colonic tube is deemed necessary, the tube is left within the dilated segment, not just in the rectum.

Success must be confirmed by a decrease in the cecal diameter on post-therapy abdominal radiograph and by a notable decrease in abdominal girth. Serial exams and abdominal films must continue to ensure that the pseudo-obstruction does not recur or persist. If the cecal diameter is unchanged after colonoscopic decompression, then another treatment modality should be pursued and/or the diagnosis of pseudo-obstruction revisited and the diagnosis of mechanical obstruction re-entertained.

Operative intervention is indicated when signs and symptoms of

perforation or ischemia are present or when conservative measures have failed. In the presence of ischemia or when splitting of the teniae coli is evident, resection of the involved segment, usually the cecum and ascending colon, is warranted. Primary anastomosis can often be performed without a bowel preparation on the right side of the colon, as long as the residual tissues and medical condition of the patient are favorable. When surgical intervention is necessary after failed medical therapy and no evidence of ischemia exists, some authors recommend a surgical cecostomy tube be placed. The tube placed should have a large diameter (>32F) and be flushed frequently to prevent obstruction with stool, which can be quite problematic

METHODOLOGY

The materials for the clinical study of intestinal obstruction were collected from cases admitted to various surgical wards of Tirunelveli medical college hospital,during the period from February 2011 to October 2012,hundred cases of intestinal obstructionhave been studied. Patients belonged to the age groups ranging from 12 years to 88 years,paediatric age group is excluded from this study. The criteria for selection of cases was based on clinical history, physical findings, radiological and haematologicalinvestigations.

Patients who were having subacute intestinal obstruction treated conservatively,patients with adynamicobstructionandpaediatric patients were excluded from the study, and only those cases of acute intestinal obstruction which were managed surgically were studied to establish the aetiologyof intestinal obstruction with an aim to know the mode of presentation, physical findings, radiological and haematological findings, operative findings and outcome of acute intestinal obstruction.

After the admission of the patient, clinical data were recorded as per Proforma. The diagnosis mainly based on clinical examination and often supported by haematological and radiological examinations.

Methods

Study divided into

a. Clinical study

b. Investigations

c. Treatment

Study was conducted under the following headings:

a. History taking

b. Physical examination

c. Laboratory examination

d. Radiological examination – Plain X-ray erect abdomen

e. Ultrasound examination in selected cases

f. Surgical treatment and results

g. Follow-up

a. History taking

A complete history was obtained from the patient and the complaints entered in the proforma in a chronological order.

b. physical examination

(i) General physical examination – evidence of dehydration and the severity of it werelooked into it and vital parameters were recorded.

(ii) Local examination – Abdominal examination was done under standard headings inspection, palpation, percussion and palpation. Per rectal

examination was done and findings were noted.

(iii) Systemic examination – All other systems were examined carefully to rule outassociated anomalies and to assess the fitness for surgery.

c. Laboratory examination

(i) Haemoglobin

(ii) TC & DC

(iii) BT and CT

(iv) Blood grouping and Rh typing

(v) Urine for albumin and microscopy

d. Radiological examination

Erect abdomen X-ray done in all cases, ultrasound examination in selected cases.

SURGICAL MANAGEMENT

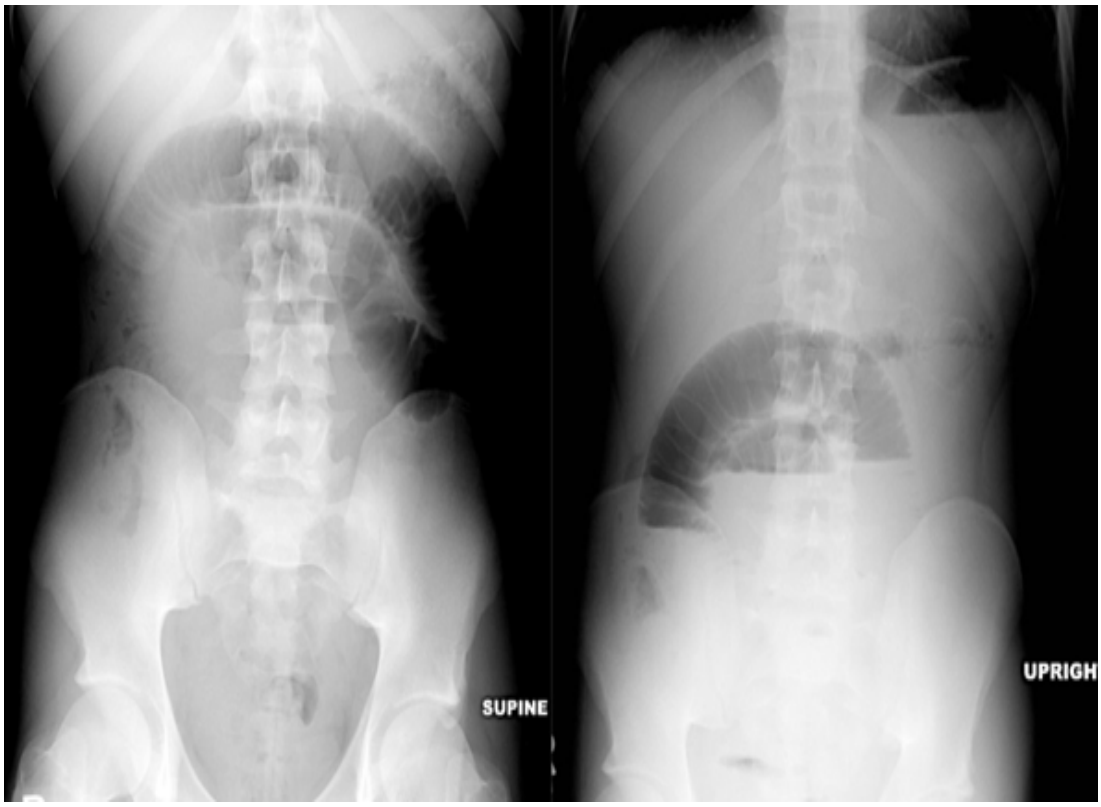
Immediately after the admission along with above procedure resuscitation with IV fluids especially ringer lactate and normal saline infusion started till the hydration and urine output improved. Nasogastric decompression with Ryles tube carried out and antibiotic prophylaxis started. And close observation of all bedside parameters (like pulse rate, BP, RR, urine output, abdominal girth, bowel sounds)was done. Patients who showed reduction in abdominal distension and improvement in generalcondition especially inindividuals with postoperative adhesions

conservative management was confined (by extending the supportive treatment) for next 24 hours, those who showed improvement by moving bowels, reduction in pain/tenderness were decided for conservative treatment, Such individuals are excluded from this study. Patients with clear-cut signs and symptoms of acute obstruction were managed by appropriate surgical procedure after resuscitation. Surgery adopted and criteria for deciding the procedure were noted, e.g. release of aband or adhesion, reduction and caecopexy for intussusception and release and repair for strangulated obstruction. The postoperative period was monitored carefully and all parameterswere recorded depending upon the patient's condition.

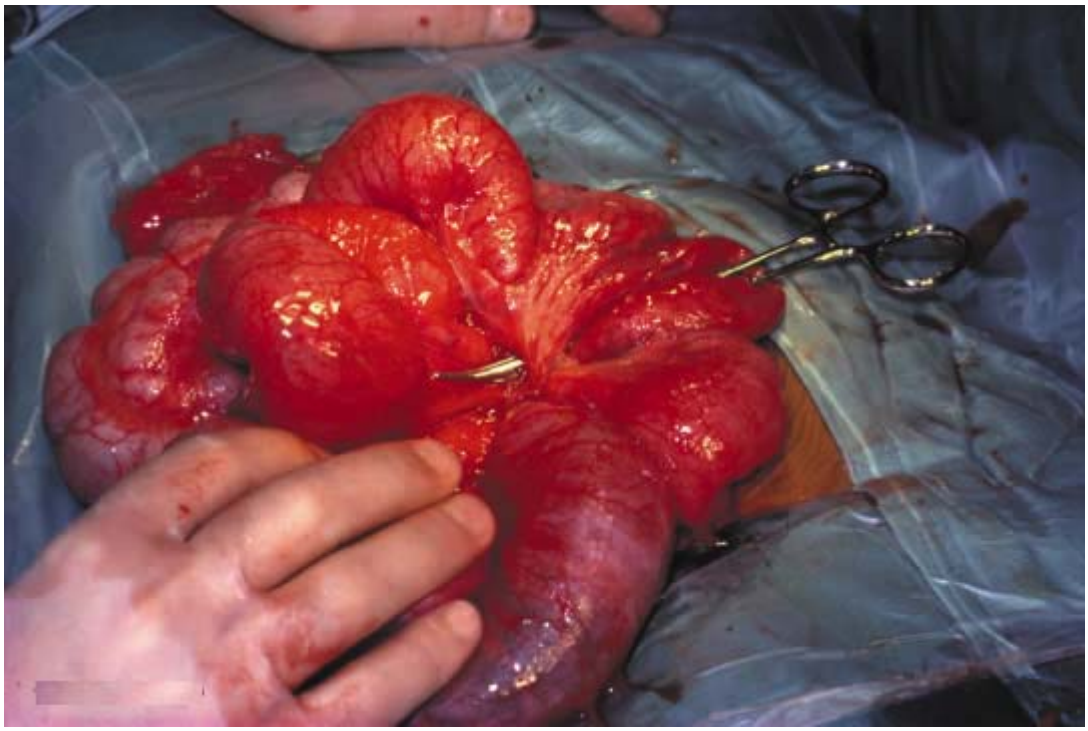
Postoperatively Ryle's tubeaspiration, intravenous fluids and antibiotics were administered. Any complications noted and treated accordingly.

Postoperative follow up after the discharge of patients was done in majority of the patientsupto 6 months. Most of the patients did not come for follow up after one or two visits.

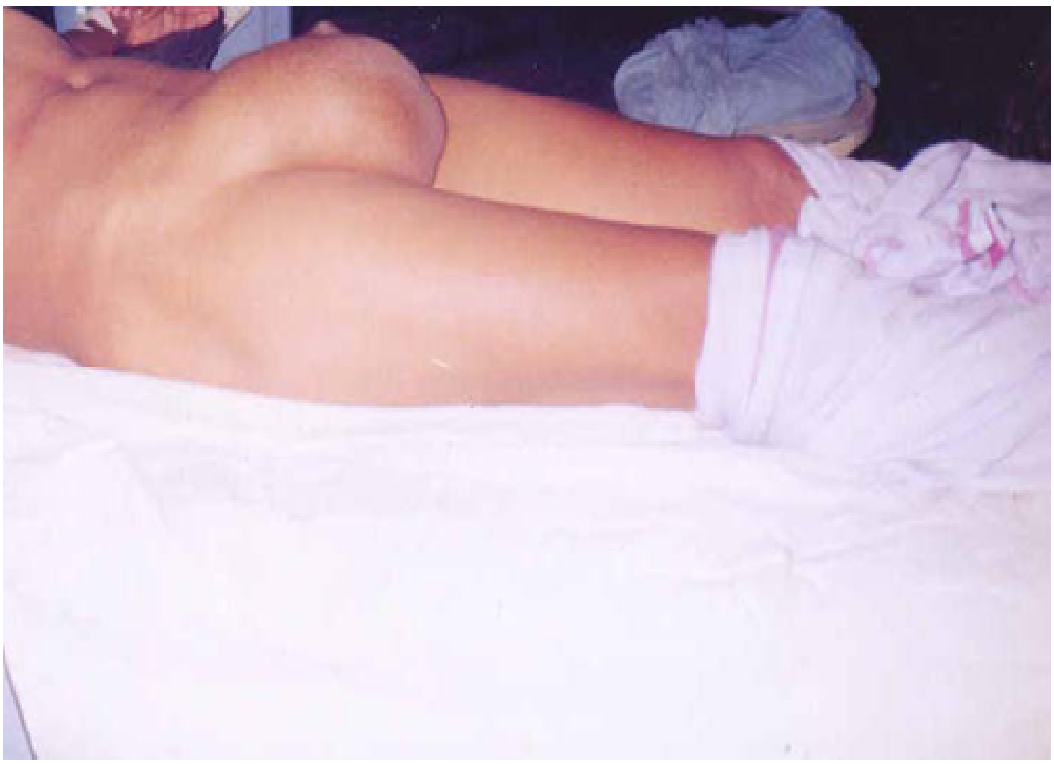
The results are tabulated stressing on following points age, sex, symptoms, examinationfindings, investigations, abnormalities, probable causative factors,operative findings and operative procedure adopted and complications if any.



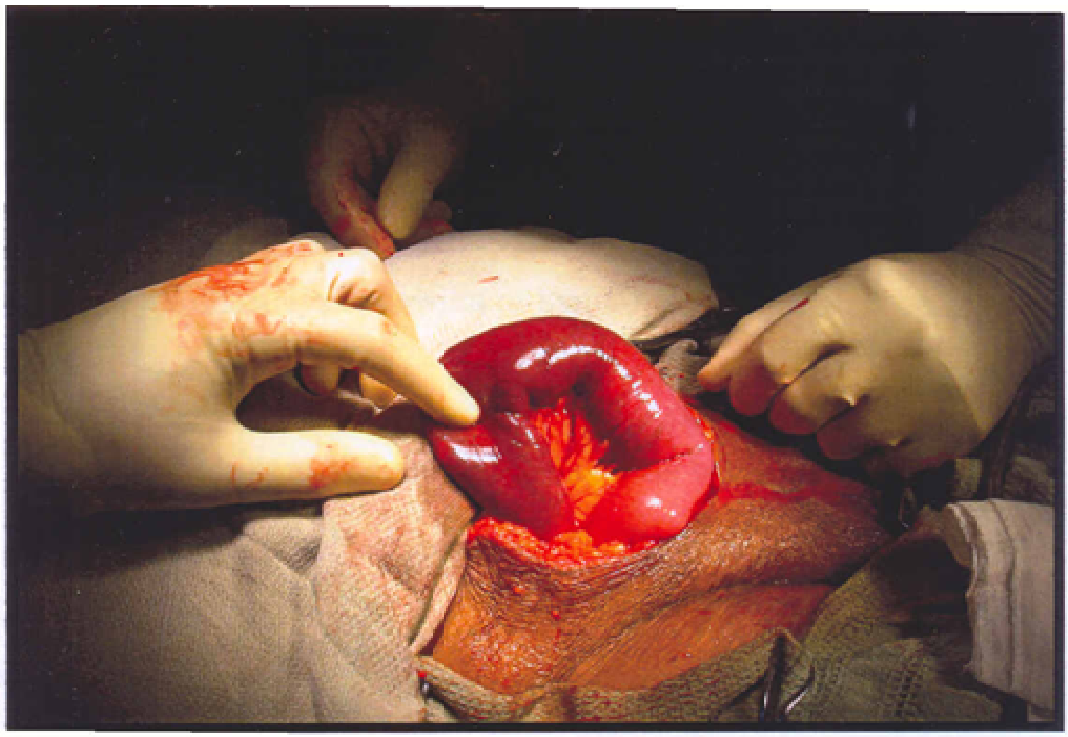
XRAY ABDOMEN SHOWING DIALATED BOWEL LOOP WITH AIR FLUID LEVELS.



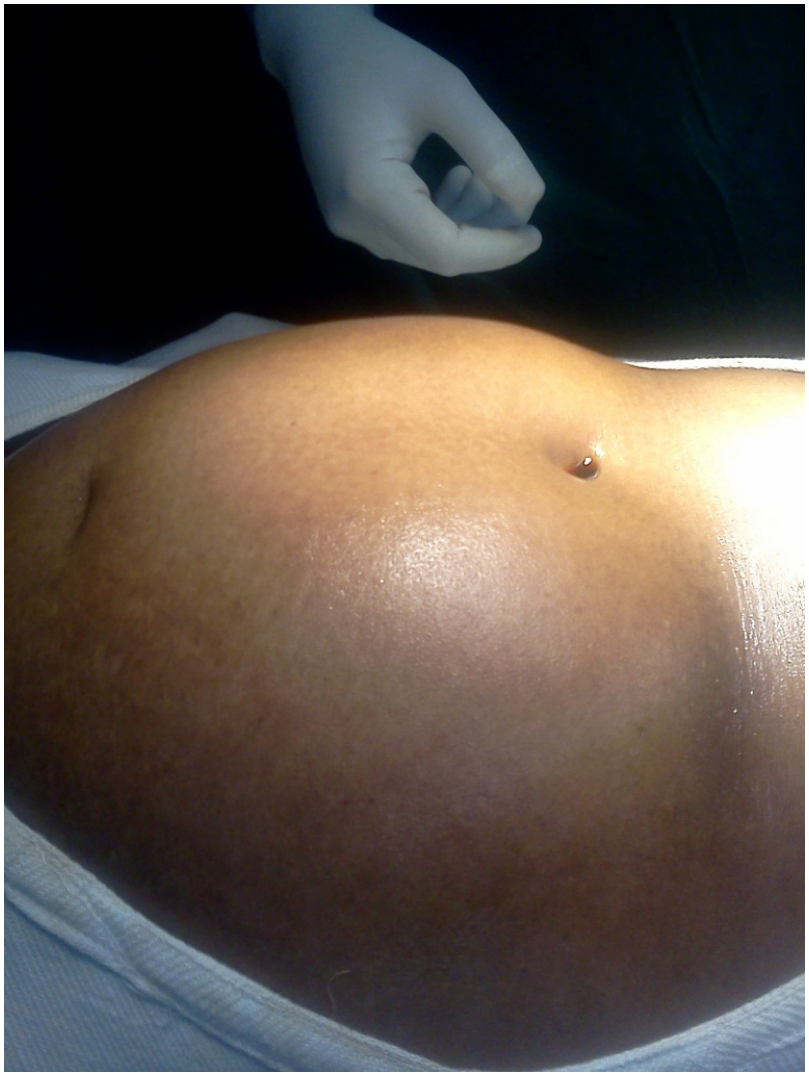
SMALL BOWEL ADHESIONS.



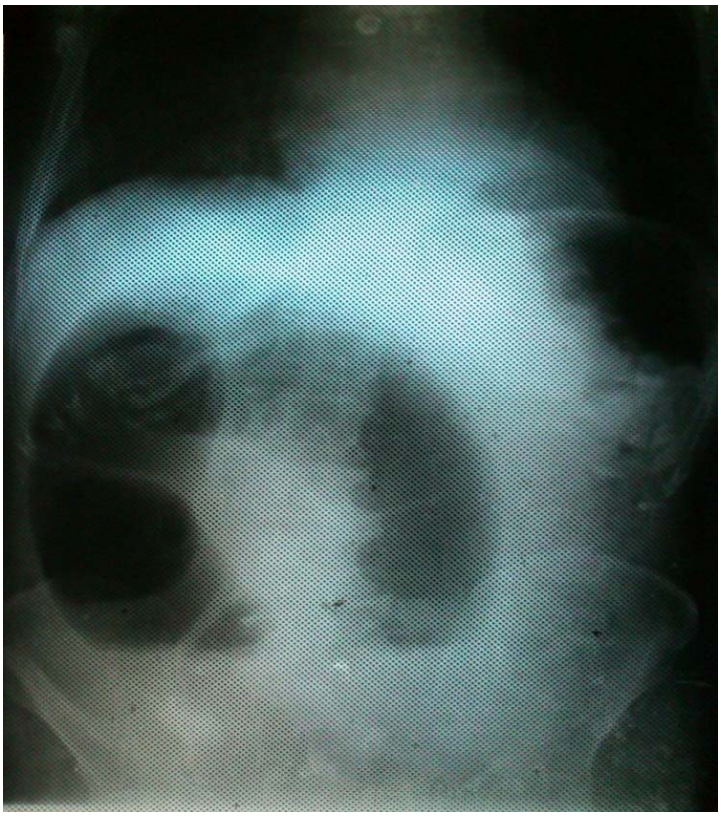
OBSTRUCTED INGUINAL HERNIA



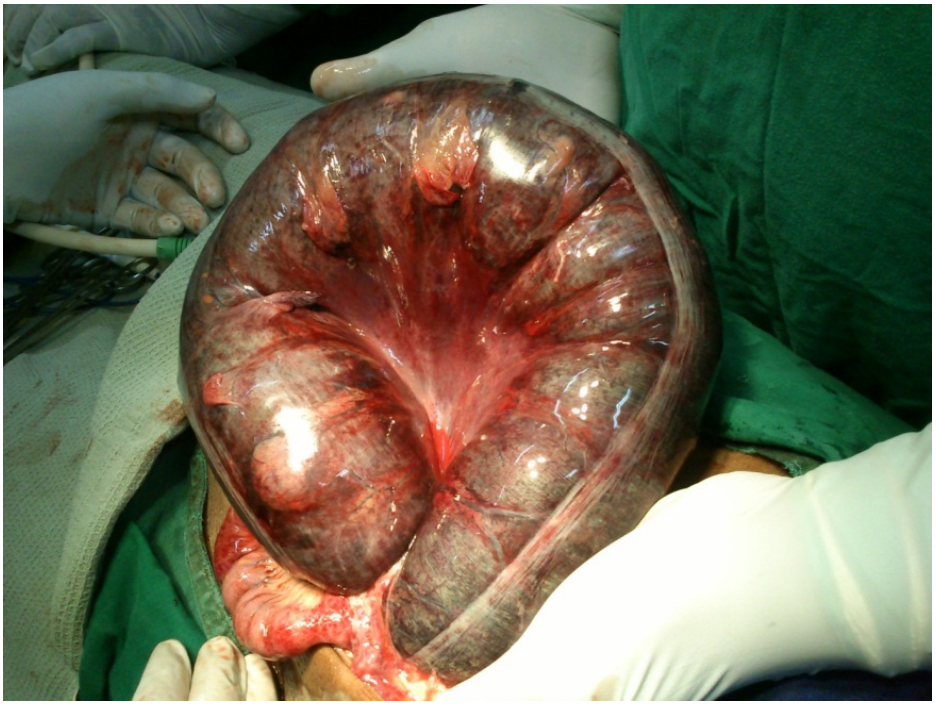
CONGESTED SMALL BOWEL IN OBSTRUCTED INGUINAL HERNIA.



LOWER ABDOMINAL DISTENSION IN ACUTE INTESTINAL OBSTRUCTION.



X - RAY FILM ABDOMEN ERECT APPEARANCE IN SIGMOID VOLVULUS



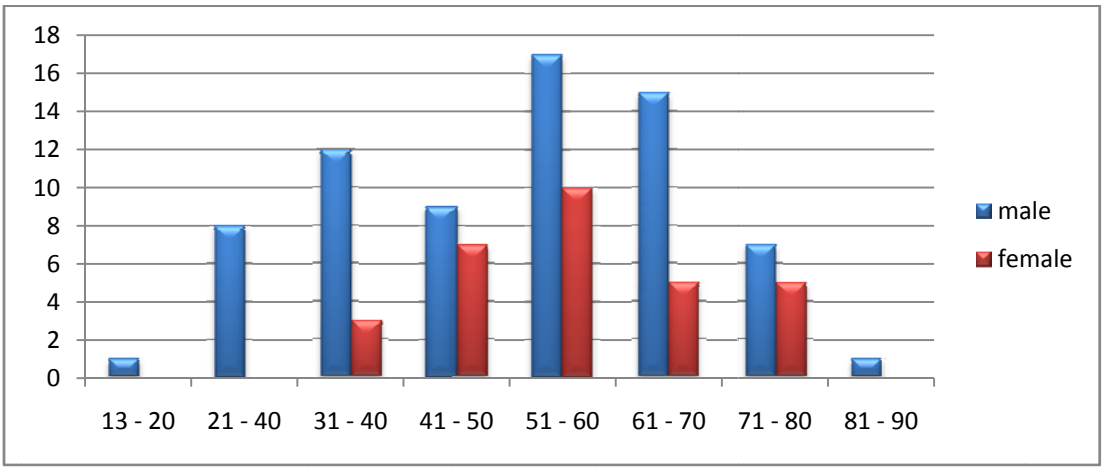
SIGMOID COLON GANGRENE FOLLOWING VOLVULUS

RESULTS

1)AGE DISTRIBUTION OF INTESTINAL OBSTRUCTION

AGE in years	MALE	FEMALE	TOTAL
13-20	1	0	1
21-30	8	0	8
31-40	12	3	15
41-50	9	7	16
51-60	17	10	27
61-70	15	5	20
71-80	7	5	12
81-90	1	0	1
TOTAL	70	30	100

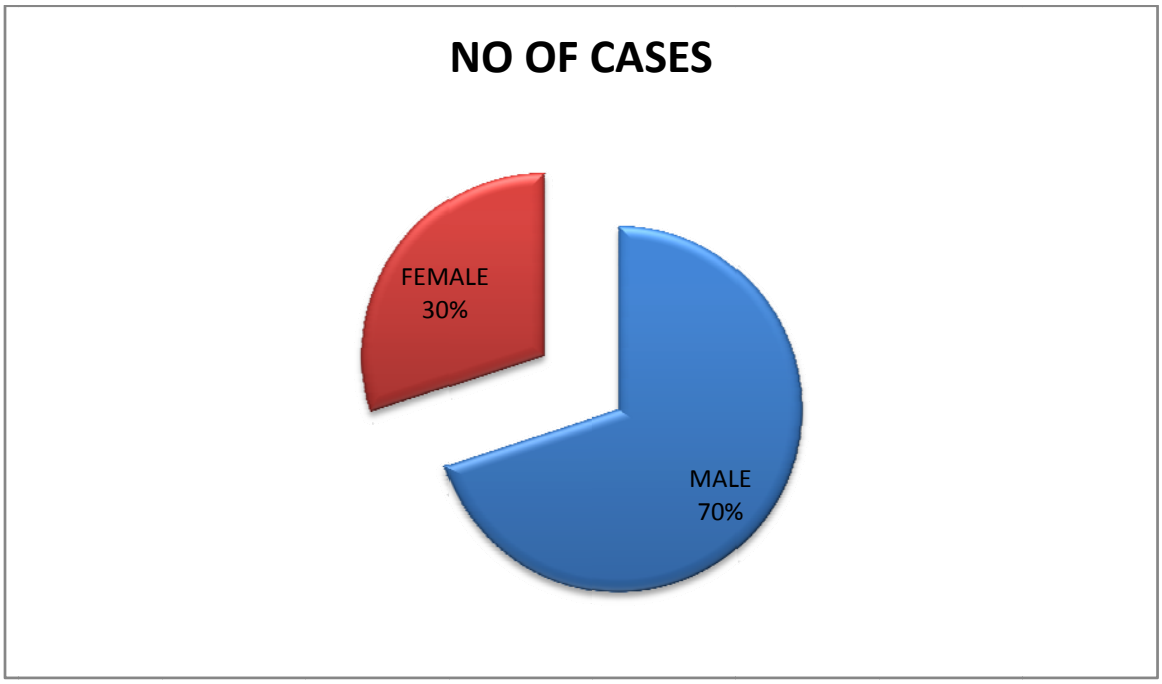
Age predominance from 40 – 70 years of age.



2)SEX WISE INCIDENCE OF INTESTINAL OBSTRUCTION

SEX	NUMBER OF CASES	PERCENTAGE
MALE	70	70%
FEMALE	30	30%

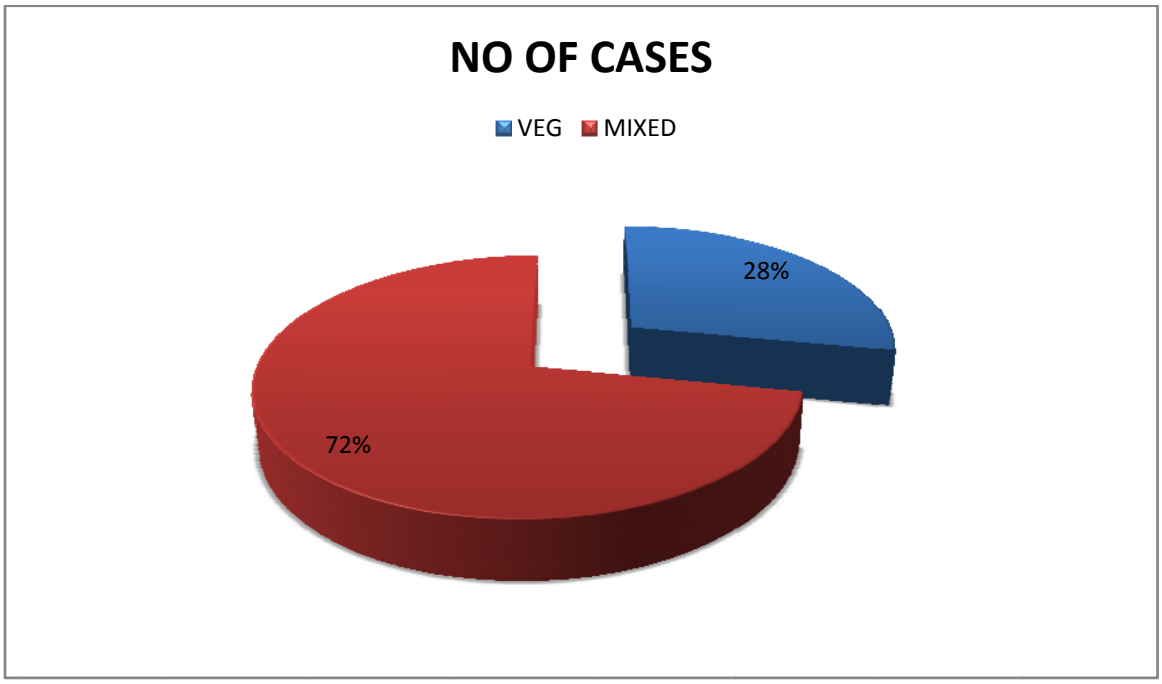
Male sex is predominantly affected.



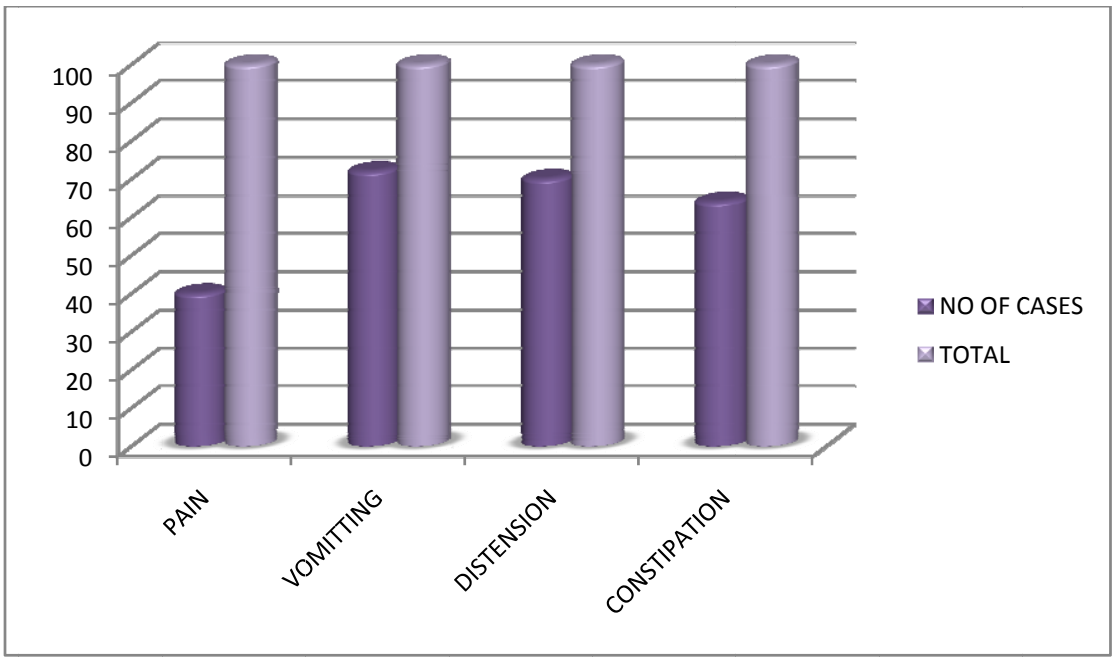
3)DIET WISE INCIDENCE OF INTESTINAL OBSTRUCTION

DIET	NO OF CASES	PERCENTAGE
VEGETARIAN	28	28%
MIXED	72	72%

People taking mixed diet has more predisposition,probably due to less fibre content of diet.

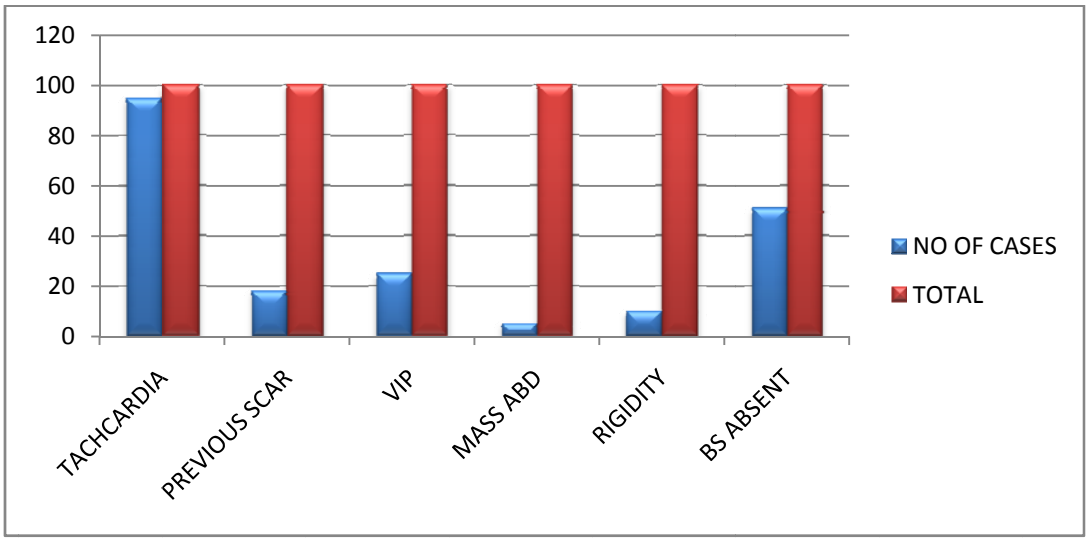


4)INCIDENCE OF SYMPYOMS



Vomiting and distension are the predominant symptoms in these patients.

5)INCIDENCE OF SIGNS IN THE CASES OF INTESTITAL OBSTRUCTION

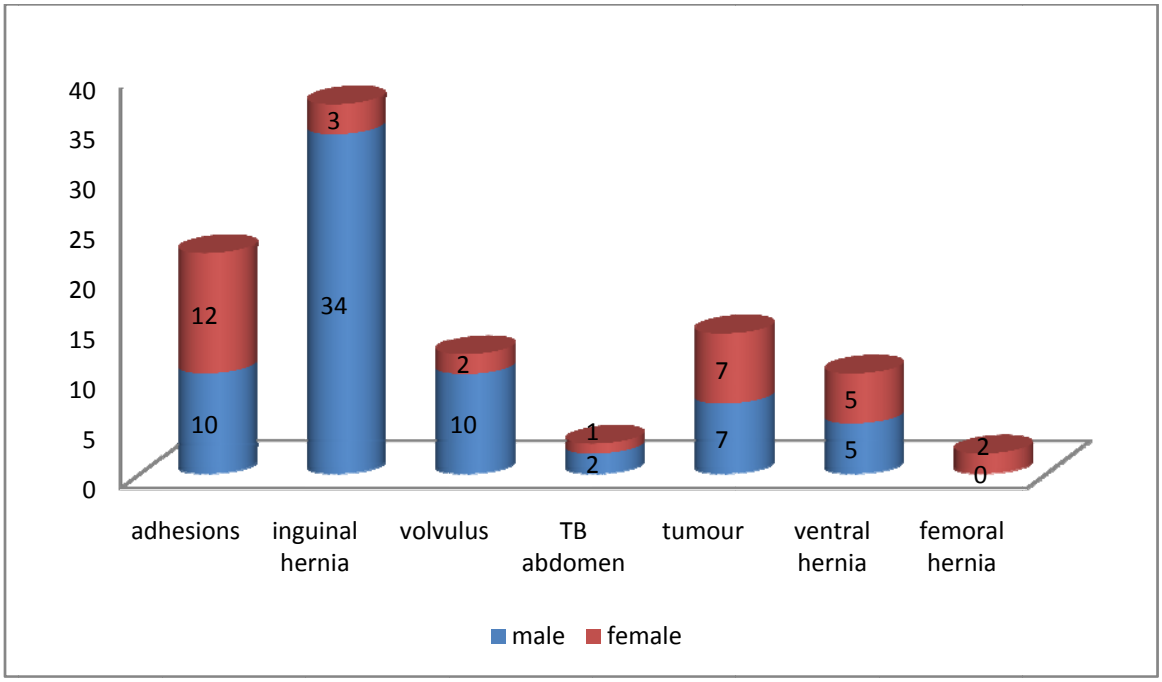


tachycardia is most common sign.

6)INCIDENCE OF VARIOUS ETIOLOGY OF INTESTINAL OBSTRUCTION

ETIOLOGY	NO OF CASES		TOTAL	PERCENTAGE
	MALE	FEMALE		
Adhesions	10	12	22	22
Inguinal hernia	34	3	37	37
Sigmoid volvolus	10	2	12	12
TB abdomen	2	1	3	3
Tumour	7	7	14	14
Ventral hernia	5	5	10	10
Femoral hernia	0	2	2	2

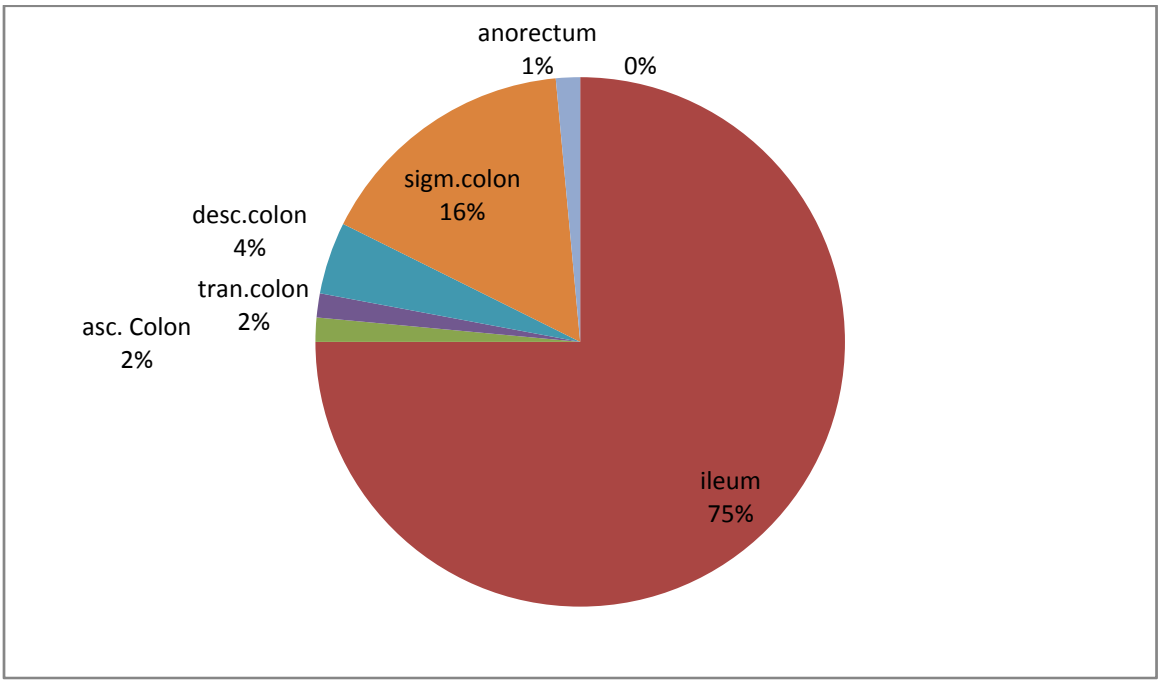
Inguinal Hernias form the most common group.



7)ANATOMICAL DISTRIBUTION OF INTESTINAL OBSTRUCTION

SITE	MALE	FEMALE
duodenum	-	-
jejunum	-	-
ileum	51	23
Ascending colon	1	1
Transverse colon	1	2
Descending colon	3	2
Sigmoid colon	11	3
anorectum	1	1

Ileum is the most commonly affected part due to its involvement in inguinal hernias and adhesive obstruction.



8)ANALYSIS OF THE SURGICAL PROCEDURE DONE

MANAGEMENT	NO OF CASES		TOTAL
	MALE	FEMALE	
RA & H	5	1	6
R & H	29	2	31
R & C	5	5	10
ROA	7	8	15
RA	21	10	31
DIVERSION	2	4	6

Resection and anastomosis was most commonly done procedure along with release and herniorrhaphy.

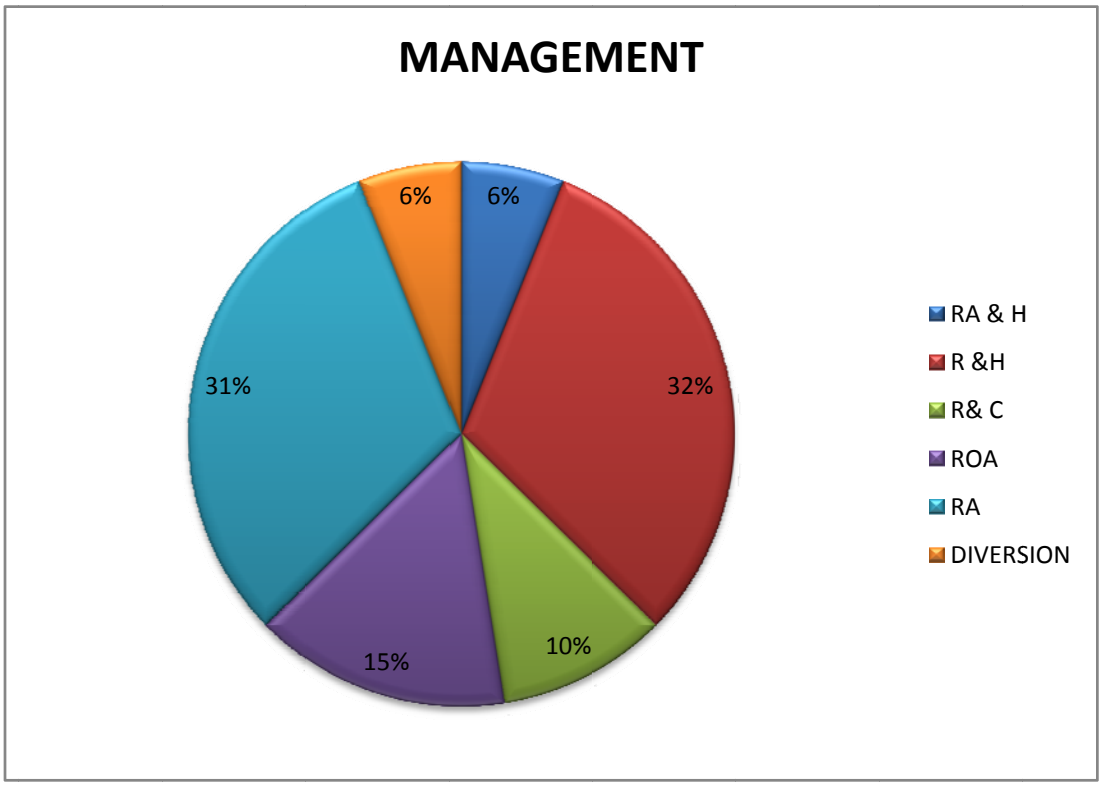
RA&H-resection anastomosis and herniorrhaphy

R & H- Release and Herniorrhaphy

R & C- Release and Closure.

ROA- Release of Adhesions.

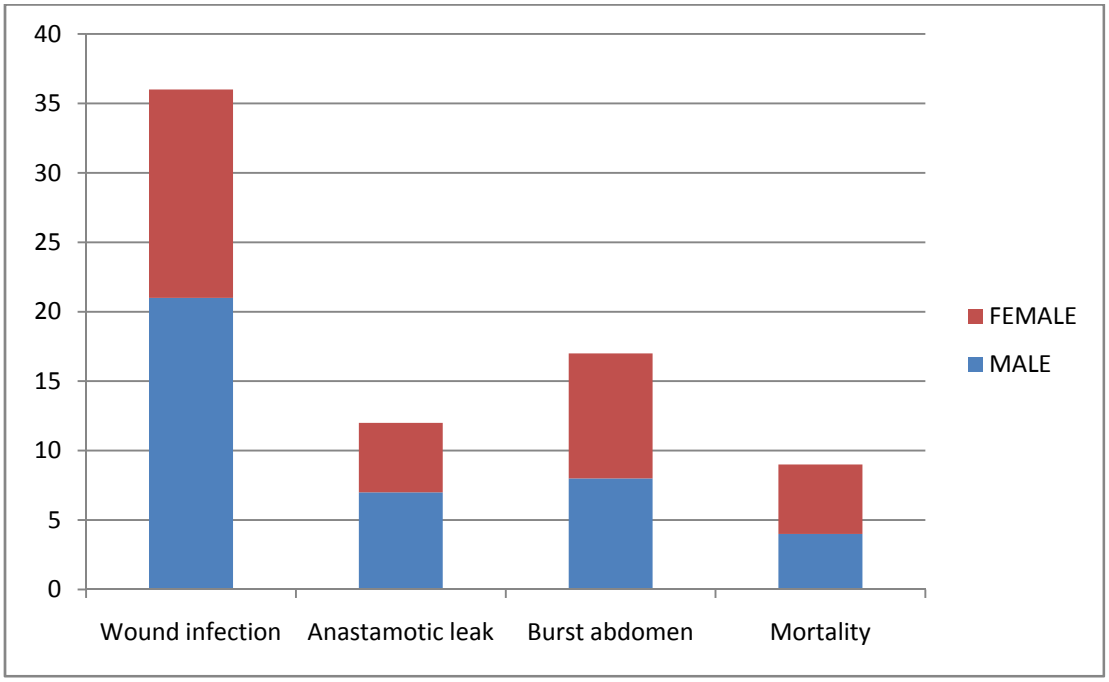
RA- resection anastomosis.



9) ANALYSIS OF POSTOPERATIVE COMPLICATIONS

COMPLICATION	MALE	FEMALE
Wound infection	21	15
Anastamotic leak	7	5
Burst abdomen	8	9
Mortality	4	5

Complication in proportion to cases was more in females.



DISCUSSION

Age incidence

Intestinal obstruction occurs across all ages.our study showed predominant involvement of 51- 60(27%) and 61-70(20%) which was comparable to studies by souvik et al and cole et al.

Age incidence was more in elderly as most cases of obstructed inguinal hernias occurred in these age groups.

Sex Incidence

In SouvikAdhikarietal.study male to female ratio was 4:1. In Osuigweet al. study male to female ratio was 2:1. In the present study male to female ratio 2.3:1.These are comparable to previous studies with malepredominance,probably due to increased incidence of obstructed inguinal hernias occurring in males.

Comparison of etiology with other studies

EtiologyasperArshad et al is adhesions 16 %,hernia 36%,volvulus 6 %,tumours 14%.Incidence as per souvik et al is adhesions 49%,hernia 34%,volvulus 5%,tumours 3%.

Incidence as per our study is adhesions 22%,hernia37%, volvulus12% ,tumours 14% .This is correlating with the fact that in third world countries where people present late for treatment forhernias,or where treatment is not easily available ,they constitute an important part

of intestinal obstruction.adhesions being the second most common etiology probably due to increasing operative interventions for various diseases than in the past.

Symptomatology studied showed incidence of vomiting and abdominal distension as predominant features.(72% & 70%).souvikadhikari et al showed 91% & 93% in their study,whereassarwar khan et al showed 93% &97% in their study.This might be due to early hospitalisation of patients for these complaints in our study.

Tachycardia was a uniform sign in a high percentage of patients.(95%).

Patients with mixed diet were found to have more predisposition for intestinal obstruction probably due to the less fibre content of diet.Wound infection was most common complication which was comparable to other similar studies.

Site of obstruction was commonly found to be ileum due to its involvement in hernias as well as postoperative adhesions and in tuberculosis.The result was comparable to those done by souvikadhikari et al.

Release of adhesions with herniorrhaphy along with resection anastomosis was the common surgical procedure done in our study.

Mortality rates

Our study showed a mortality rate of 9% which was comparable to following studies.Mortality was more in elderly patients ,with late presentations,malignancies.which all lead to the poor outcome.

Studies	**Year**	**No. of cases**	**Percentage**
SouvikAdhikari	2005	367	7.35%
Safian Matsu Moto	1975	171	19%
Jahangir-SarwarKhan	2001	100	7%
RamachandranCS	1982	417	12.7%

Gangrenous changes following late presentation was associated with increased post operative morbidity and mortality.

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CONCLUSION

Acute intestinal obstruction remains an important surgical emergency in the surgical field

Success in the treatment of acute intestinal obstruction depends largely upon early diagnosis skillful management and treating the pathological effects of the obstruction just as much as the cause itself.

Erect abdomen X-ray is valuable investigation in the diagnosis of acute intestinal obstruction.

1. The study showed that inguinal hernias are the most common cause of intestinal obstruction .
2. Males were predominantly affected.
3. Age group between 41 – 70 years was the most commonly affected.
4. Patients taking mixed diet was predominantly affected.
5. Most common symptom was vomiting .
6. Most common sign was tachycardia.
7. Mortality was found to be highest with colonic obstruction due to growth.
8. Total number of Anastamotic leak and Wound infection were more in males ,though a disproportionately higher incidence occurred in women inspite of their lower numbers.
9. Most common postoperative complication was wound infection.
10. Resection and anastomosis as well as release and herniorrhaphy were the most commonly done surgical procedures.

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PROFORMA

Sl.No. :Name : DOA:

Age :DOD : Unit :

Sex :Address :

Occupation :

Economic status:

1. Chief Complaints

2. History of Present Illness

- Pain
- Vomiting
- Distension
- Bowel habits
- H/o passing blood in stools
- H/o fever
- H/o Jaundice

3. Past History

- H/o tuberculosis
- H/o any surgeries
- H/o altered bowel habits

4. Personal History

- Micturition

- Bowel habits
- Sleep
- Weight loss
- Appetite
- Smoker
- Alcoholic

5. Menstrual History (females)

6. Obstetric History (females)

7. Family History

8. General Physical Examination

Vital Signs

- Pallor: Pulse:
- Cyanosis: Blood pressure:
- Clubbing: Respiratory rare
- Jaundice: Temperature
- Edema:
- LN:

9. Systemic Examination

Inspections

- Shape: Distension:
- Respiratory movements of each region:

- Peristaltic movements:
- Position of umbilicus:
- Scars:
- Any skin changes:
- Any mass:
- Hernial sites
- Renal angles:
- Supraclavicular fossa:

Palpation

- Cutaneous hyperaesthesia:
- Tenderness:
- Rebound tenderness:
- Muscular rigidity:
- Palpation of hernial orifice:
- Any mass palpable:
- Supraclavicular fossa:
- Testes:

Percussion

Auscultation

Per rectal

Per vaginal

Cardiovascular system

Respiratory system

Central nervous system

10. Investigation

Blood

Hb% TC: DC: BT: CT:

ESR Urea: RBS: Blood grouping and Rh typing:

Urine : Sugar: Albumin: Microscopy:

ECG :

X-ray : X-ray of the chest

X-ray of the abdomen (Erect):

11. Preoperative Diagnosis

12. Treatment

Preoperative treatment

Hourly

Pulse

Blood pressure

Temperature

Respiratory rate

IV fluids

RTA

Urine OutPut

Abdominal girth

Blood transfusion:

Drugs:

13.Operative management

Anaesthesia:

Incision:

Gross appearance: Peritoneum

Colour:

Small bowel: Gangrene:

Perforation:

Colour

Large bowel: Gangrene:

Perforation:

14. Pathology note

15. Surgical procedures

16. Postoperative treatment

Days

Pulse

Blood pressure

Temperature

Respiratory rate

Blood transfusion

IV fluids

RTA

Urine OutPut

Drain

Drugs

17. Postoperative complications

18. Histopathological report of the specimen

19. Condition at the time of discharge

20. Advice on discharge

21. Follow-up for any complaints

22. Remarks

MASTER CHART

S. No	Name	Age	Sex	IP.No	Diagnosis	Procedure	Anastamotic leak	Wound infection	Mortality
1	MURUGAN	58	M	38404	PH	R&C	NA	N	N
2	GANAPATHI	62	M	38634	IH	R&C	NA	N	N
3	MALA	65	F	22799	ING H	RAH	NA	N	N
4	ARUMUGAM	66	M	22980	TUMOUR	RAA	N	Y	N
5	MADASAMY	53	M	48692	ING H	RAH	NA	N	N
6	GOPAL	32	M	48901	ING H	RAH	NA	N	N
7	NALLASAMY	79	M	43520	SV	RAA	N	Y	N
8	SANKARI	45	F	44317	IH	R&C	NA	N	N
9	KRISHNAN	65	M	41217	TUMOUR	RAA	Y	Y	N
10	CHERMADURAI	70	M	40922	PH	R&C	NA	N	N
11	PETCHIAMMAL	40	F	42408	ADHES	R	NA	N	N
12	GANGA	46	F	42540	ADHES	R	NA	N	N
13	KARUPPAN	65	M	43012	ING H	RAH	NA	N	N
14	LAKSHMI	60	F	9722	*IC TB*	RAA	N	Y	N
15	GOPAL	57	M	11432	ING H	RAH	NA	N	N
16	MANOHAR	48	M	11590	ADHES	R	NA	N	N
17	SUBBIAH	67	M	13933	ING H	RAH	NA	N	N
18	SUDHA	53	F	11972	ADHES	R	NA	N	N

19	MURUGAN	38	M	11450	ING H	RAH	NA	N	N
20	ESAKKIAMMAL	75	F	13842	TUMOUR	TC	NA	Y	N
21	SANKILI	40	M	15723	ADHES	R	NA	N	N
22	CHELLIAH	60	M	16022	PH	R&C	NA	N	N
23	YAGAPAN	27	M	18445	ING H	RAH	NA	N	N
24	LAXMANAN	61	M	19791	SV	RAA	N	N	N
25	MAYILATHAL	54	F	18024	ADHES	R	NA	N	N
26	MARIAMMAL	55	F	17992	ADHES	RAA	N	Y	N
27	ANNATHAI	72	F	16818	TUMOUR	RAA	Y	Y	Y
28	MANICKAM	65	M	39860	TUMOUR	TC	NA	N	N
29	PONSELVAN	28	M	39996	ING H	RAH	NA	N	N
30	MOOKANDI	47	M	41931	ADHES	RAA		Y	N
31	MAHALINGAM	55	M	42446	ING H	RAH	NA	N	N
32	INDIRA	50	F	42668	TUMOUR	RAA	N	Y	N
33	JOHN	71	M	43586	ING H	RAH	NA	Y	N
34	MADASAMY	77	M	44618	TUMOUR	RAA	Y	Y	Y
35	MUTHIAH	88	M	44704	ING H	RAH	NA	Y	N
36	CHELLAPANDIAN	55	M	31676	SV	RAA	Y	Y	N
37	CHINNAMMAL	56	F	32891	ADHES	R	NA	N	N
38	MURUGAN	70	M	32886	ING H	RAH	NA	N	N
39	AYAN RAJ	38	M	33038	ADHES	R	NA	N	N
40	SANTHANAM	36	M	32603	ADHES	RAA	Y	Y	N
41	LAXMI	60	F	35309	ADHES	R	NA	N	N

42	KARUPPIAH	55	M	35634	ING H	RAA & H	N	Y	N
43	UDAIYAN	70	M	36912	ING H	RAA & H	N	Y	N
44	RAMAIAH	75	M	36473	ING H	RAH	NA	N	N
45	RAVI	49	M	36653	ING H	RAH	NA	N	N
46	DINESH	17	M	38100	ING H	RAH	NA	N	N
47	RAJENDRAN	52	M	39676	IC TB	RAA	N	N	N
48	SANKARAMMAL	55	F	20556	ADHES	RAA	N	N	N
49	KARUPPUSAMY	52	M	23979	SV	RAA	N	Y	N
50	KASIRAJAN	50	M	24309	ADHES	RAA	N	N	N
51	SUBBIAH	65	M	25137	ING H	RAH	NA	N	N
52	MARUTHU	32	M	27869	ADHES	R	NA	N	N
53	SHANMUGHAM	70	M	29263	TUMOUR	TC	NA	Y	N
54	PALANIKUMAR	36	M	29582	ING H	RAH	NA	N	N
55	MUNIAMMAL	75	F	29594	TUMOUR	RAA	Y	Y	Y
56	RANJITH	48	M	31780	ING H	RAH	NA	N	N
57	AUVADIAMMAL	80	F	44702	TUMOUR	TC	NA	Y	Y
58	THAVAPIRAN	78	M	48649	SV	RAA	N	Y	N
59	RAMASAMY	80	M	49287	ING H	RAA & H	Y	Y	Y
60	RAJAMOHD.	40	M	49355	ADHES	RAA	N	Y	N
61	ARUMUGAPERM	26	M	50512	SV	RAA	N	N	N
62	RANJITHAM	35	F	52195	IH	RAA	N	Y	N
63	MARISELVAN	23	M	4304	SV	RAA	N	N	N
64	AYYANKAR	65	M	4840	TUMOUR	RAA	Y	Y	N

65	RAMACHANDRN	35	M	4974	ING H	RAH	NA	N	N
66	VIJAYA	43	F	6168	FH	RAH	NA	N	N
67	PARAMESWRN	24	M	3964	ICTB	RAA	N	N	N
68	CHIDAMBARAM	52	M	6827	IH	R&C	NA	N	N
69	UMAIYAMMAL	58	F	7131	ADHES	R	NA	N	N
70	MURUGAN	44	M	7288	ING H	RAH	NA	N	N
71	PACHALAXMI	42	F	8273	ING H	RAH	NA	N	N
72	RAMACHANDRN	54	M	9602	PH	R&C	NA	N	N
73	CHELLAMMAL	70	F	11065	ADHES	RAA	Y	Y	Y
74	PONNAMAL	70	F	11586	FH	RAH	NA	Y	N
75	GOMATHI	32	F	12698	SV	RAA	N	Y	N
76	KOTTUSAMY	53	M	16488	ING H	RAH	NA	N	N
77	KOVIL	50	M	17484	ING H	RAH	NA	N	N
78	MURUGAN	35	M	17717	ADHES	R	NA	N	N
79	MURUGAN	30	M	52607	ING H	RAH	NA	N	N
80	PIRAMU	50	F	55131	ADHES	R	NA	N	N
81	MUNIAMMAL	60	F	53913	SV	RAA	Y	Y	Y
82	PARAMESWARI	70	F	55274	RH	RAA & H	N	Y	N
83	PONNUSAMY	55	M	55343	ING H	RAH	NA	N	N
84	POORNAM	50	F	50053	IH	R&C	NA	N	N
85	KARUPPIAH	32	M	56222	SV	RAA	N	N	N
86	ESSAKKIMUTHU	55	M	55629	ING H	RAH	NA	Y	N
87	KARUPAYEE	72	F	59357	TUMOUR	TC	NA	Y	N

88	MARIKONAR	60	M	60526	ING H	RAH	NA	Y	N
89	NATARAJAN	44	M	60772	SV	RAA	N	N	N
90	GANESHAN	55	M	1880	ADHES	RAA	N	N	N
91	VELU	70	M	2215	SV	RAA	Y	Y	N
92	MUSTHAFA	45	M	2709	ING H	RAH	NA	N	N
93	SRINIVASAN	72	M	3484	ING H	RAA & H	Y	Y	Y
94	VELLASAMY	58	M	15968	ADHES	R	NA	N	N
95	PATATHI	54	F	14630	IH	R&C	NA	N	N
96	NAINAR	55	M	17497	ING H	RAH	NA	N	N
97	GANESHAN	30	M	21546	TUMOUR	RAA	Y	Y	Y
98	SUBBIAH	70	M	26042	TUMOUR	TC	NA	Y	N
99	HARIHARAN	24	M	17224	ING H	RAA & H	N	N	N
100	SELVAKUMAR	37	M	27100	ING H	RAH	NA	N	N

ABBREVIATIONS IN MASTER CHART

PH – Para Umbilical Hernia

ING H- Inguinal Hernia

ADHES – Adhesions

IH – Incisional hernia

SV – sigmoid volvulus

ICTB – Ileocaecal TB