A RETROSPECTIVE CASE STUDY ON SMALL BOWEL OBSTRUCTION

This dissertation is submitted to PSG Institute of Medical Sciences and Research in partial fulfilment of the regulations for the M.S (General Surgery) Degree Examination, April 2016

By

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Done under the guidance of

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CERTIFICATE

This is to certify that this dissertation entitled **"A RETROSPECTIVE CASE STUDY ON SMALL BOWEL OBSTRUCTION"** is a record of bonafide research work done by Dr.GUHAN.R.J, under my guidance and supervision in the Department of General Surgery, PSG Institute of Medical Sciences and Research, Coimbatore – 641004.

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DECLARATION

I, **Dr. Guhan.R.J**, solemnly declare that this dissertation **"A RETROSPECTIVE CASE STUDY ON SMALL BOWEL OBSTRUCTION"** is a bonafide record of work done by me in the Department of General Surgery, PSG institute of Medical Sciences & Research, Coimbatore, under the guidance of **Dr.Rajesh kumar.S**, Professor of Surgery.

This dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfilment of the University regulations for the award of MS Degree (General Surgery) Branch-I, Examination to be held in April 2016.

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CONTENTS

INTRODUCTION

Intestinal obstruction is significant mechanical impairment or complete arrest of the passage of contents through the intestine. Symptoms include cramping pain, vomiting, obstipation, and lack of flatus. Diagnosis is clinical, confirmed by abdominal x-rays. Treatment is fluid resuscitation, nasogastric suction, and, in most cases of complete obstruction, surgery.

Mechanical obstruction is divided into obstruction of the small bowel (including the duodenum) and obstruction of the large bowel. Obstruction may be partial or complete. About 85% of partial small-bowel obstructions resolve with nonoperative treatment, whereas about 85% of complete small-bowel obstructions require surgery.

Location	Cause
Colon	Tumors (usually in left colon), diverticulitis (usually in sigmoid), volvulus of sigmoid or cecum, fecal impaction, Hirschsprung disease, Crohn disease
Duodenum	
• Adults	Cancer of the duodenum or head of pancreas, ulcer disease
• Neonates	Atresia, volvulus, bands, annular pancreas
Jejunum and ileum	
• Adults	Hernias, adhesions (common), tumors, foreign body, Meckel diverticulum, Crohn disease (uncommon), *Ascaris*infestation, midgut volvulus, intussusception by tumor (rare)
• Neonates	Meconium ileus, volvulus of a malrotated gut, atresia, intussusception

1. Causes of Intestinal Obstruction

In simple mechanical obstruction, blockage occurs without vascular compromise. Ingested fluid and food, digestive secretions, and gas accumulate above the obstruction. The proximal bowel distends, and the distal segment collapses. The normal secretory and absorptive functions of the mucosa are depressed, and the bowel wall becomes edematous and congested. Severe intestinal distention is self-perpetuating and progressive, intensifying the peristaltic and secretory derangements and increasing the risks of dehydration and progression to strangulating obstruction.

Strangulating obstruction is obstruction with compromised blood flow; it occurs in nearly 25% of patients with small-bowel obstruction. It is usually associated with hernia, volvulus, and intussusception.

Strangulating obstruction can progress to infarction and gangrene in as little as 6 hours. Venous obstruction occurs first, followed by arterial occlusion, resulting in rapid ischemia of the bowel wall. The ischemic bowel becomes edematous and infarcts, leading to gangrene and perforation. In large-bowel obstruction, strangulation is rare (except with volvulus).

Perforation may occur in an ischemic segment (typically small bowel) or when marked dilation occurs. The risk is high if the caecum is dilated to a diameter ≥ 13 cm. Perforation of a tumor or a diverticulum may also occur at the obstruction site.

Obstruction of the small bowel causes symptoms shortly after onset in the form of abdominal cramps centered around the umbilicus or in the epigastrium, vomiting, and in patients with complete obstruction may cause obstipation. Patients with partial obstruction may develop diarrhea. Severe, steady pain suggests that strangulation has occurred. In the absence of strangulation, the abdomen is not tender. Hyperactive, high-pitched peristalsis with rushes coinciding with cramps is typical. Sometimes, dilated loops of bowel are palpable. With infarction, the abdomen becomes tender and auscultation reveals a silent abdomen or minimal peristalsis. Shock and oliguria are serious signs that indicate either late simple obstruction or strangulation.

Supine and upright abdominal x-rays should be taken and are usually adequate to diagnose obstruction. Although only laparotomy can definitively diagnose strangulation, careful serial clinical examination may provide early warning. Elevated WBCs and acidosis may indicate that strangulation has already occurred, but these signs may be absent if the venous outflow from the strangulated loop of bowel is decreased.

On plain x-rays, a ladder like series of distended small-bowel loops is typical of small-bowel obstruction but may also occur with obstruction of the right colon. Fluid levels in the bowel can be seen in upright views. Similar, although perhaps less dramatic, x-ray findings and symptoms occur in ileus (paralysis of the intestine without obstruction); differentiation can be difficult. Distended loops and fluid levels may be absent with an obstruction of the upper jejunum or with closed-loop strangulating obstructions (as may occur with volvulus). Infarcted bowel may produce a mass effect on x-ray. Gas in the bowel wall (pneumatosis intestinalis) indicates gangrene.

In this study we have concluded our experience with small bowel obstruction and its common etiological factors and the management aspects to improve the current knowledge in the management of small bowel obstruction.

AIM OF THE STUDY

- To study the commonest etiological factors for small bowel obstruction in patients presenting to general surgery department in PSG Hospitals.
- To evaulate the validity of a scoring system for the line of management – surgical or conservative?

REVIEW OF LITERATURE

ANATOMY AND HISTOLOGY OF THE SMALL INTESTINE :

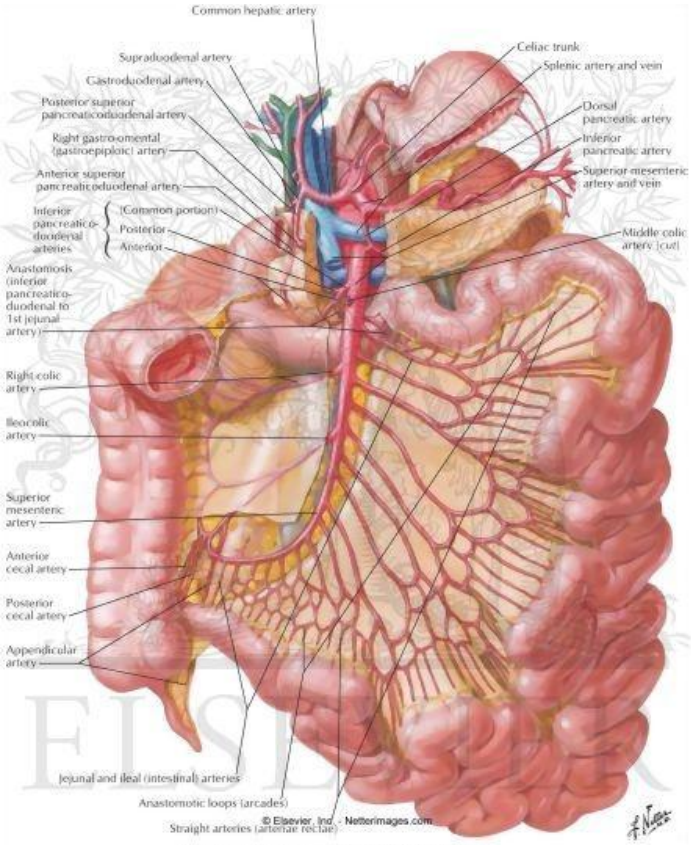
MACROSCOPIC FEATURES :

The small bowel is a tubular structure within the abdominal cavity with the stomach proximally and the colon distally. The small bowel increases 20 times in length with age, from 200 cm in the newborn to almost 6 m in adults. The duodenum, the most proximal portion of the small intestine, begins at the level of duodenal bulb, into the retroperitoneal space, near the head of the pancreas, and ends into the peritoneal cavity at the ligament of treitz. The remainder of the small bowel is suspended in the peritoneal cavity by a thin, broad based mesentery that is attached to the posterior abdominal wall and allows free mobility of the small intestine within the abdominal cavity. The proximal 40% of the small bowel is the jejunum, and the remaining 60% is the ileum. The jejunum occupies the left

abdomen, and the ileum is in the right abdomen and upper part of the pelvis. No distinct anatomic demarcation exists between jejunum and ileum. Visual examination of the luminal surface of the small intestine shows the plicae circulares. They are more in number in the proximal jejunum, and gradually decreases in number in the distal ileum and are absent in the terminal ileum. Lymphoid follicles are scattered throughout the small intestine but are in maximum concentration within the ileum, as Peyer's patches. Peyer's patches are more prominent during infancy and childhood. The small bowel continues with the colon at the ileo-caecal valve, which has 2 semilunar folds that protrude into the caecum. The ileo-cecal valve acts as a barrier to the retrograde flow of large bowel contents into the small intestine. This barrier is due to the angulation between the ileum and caecum maintained by the superior and inferior ileo-caecal ligaments, and a sphincter-type pressure does not appear to be present in this region.

INTESTINAL VASCULATURE:

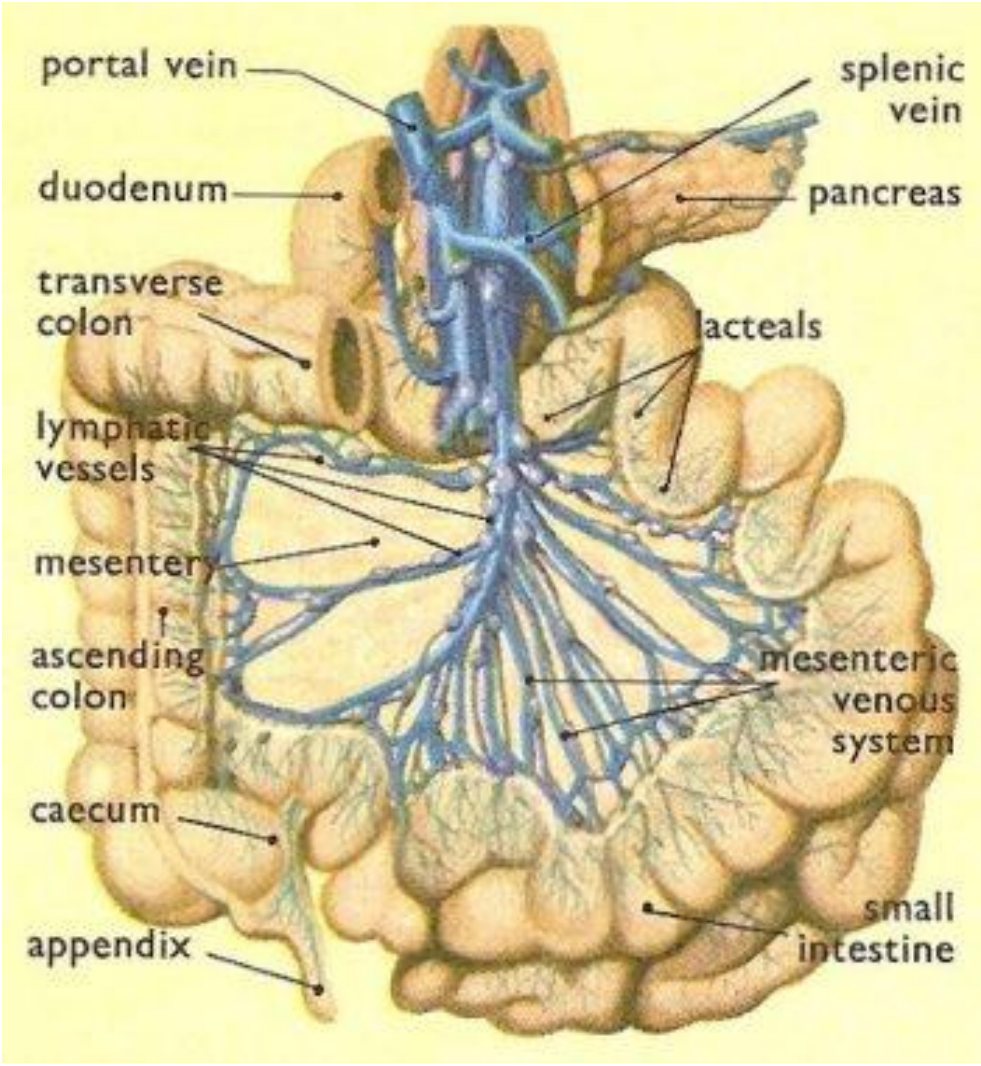
The superior mesenteric artery supplies distal duodenum, jejunum and ileum, the ascending colon, and the proximal two thirds of the transverse colon. Large arterial branches enter the muscularis propria and pass to the submucosa. In the small intestine, two types of branches arise from the submucosal plexuses: some arteries branch on the inner aspect of the muscularis mucosae and break into a capillaries that surround the crypts of Lieberkühn. Other arteries are destined for villi, each receiving one or two arteries, and this anatomic arrangement allows a counter-current mechanism during absorption. These vessels enter the base of the villi and forms a dense capillary network underneath the epithelium of the entire villus . One or several veins originate at the tip of each villus from the superficial capillary plexus and anastomose with the glandular venous plexus which then enter the submucosa joining the submucosal venous plexus.



BLOOD SUPPLY OF THE INTESTINES

INTESTINAL LYMPHATIC DRAINAGE:

The lymphatic drainage of the small bowel follows their respective blood supplies to lymph nodes in the celiac, superior mesenteric, and inferior mesenteric regions. Lymph flows into the cisterna chyli and then to the thoracic duct into the left subclavian vein. The small bowel lymphatics are called lacteals and become filled with milky-white lymph called chyle after eating. Each villi has one central lacteal, except in the duodenum where two or more lacteals per villi are present. The wall of the lacteal consists of endothelial cells, reticulum fibers, and smooth muscle cells. The central lacteals forms a network at the base of the villi with the lymphatic capillaries between the crypts of Lieberkühn. Branches of this plexus extend through the muscularis mucosae to form a submucosal plexus. Branches from the submucosal plexus penetrate the muscularis propria, where they receive branches from a neywork of vessels between the inner and outer layers.

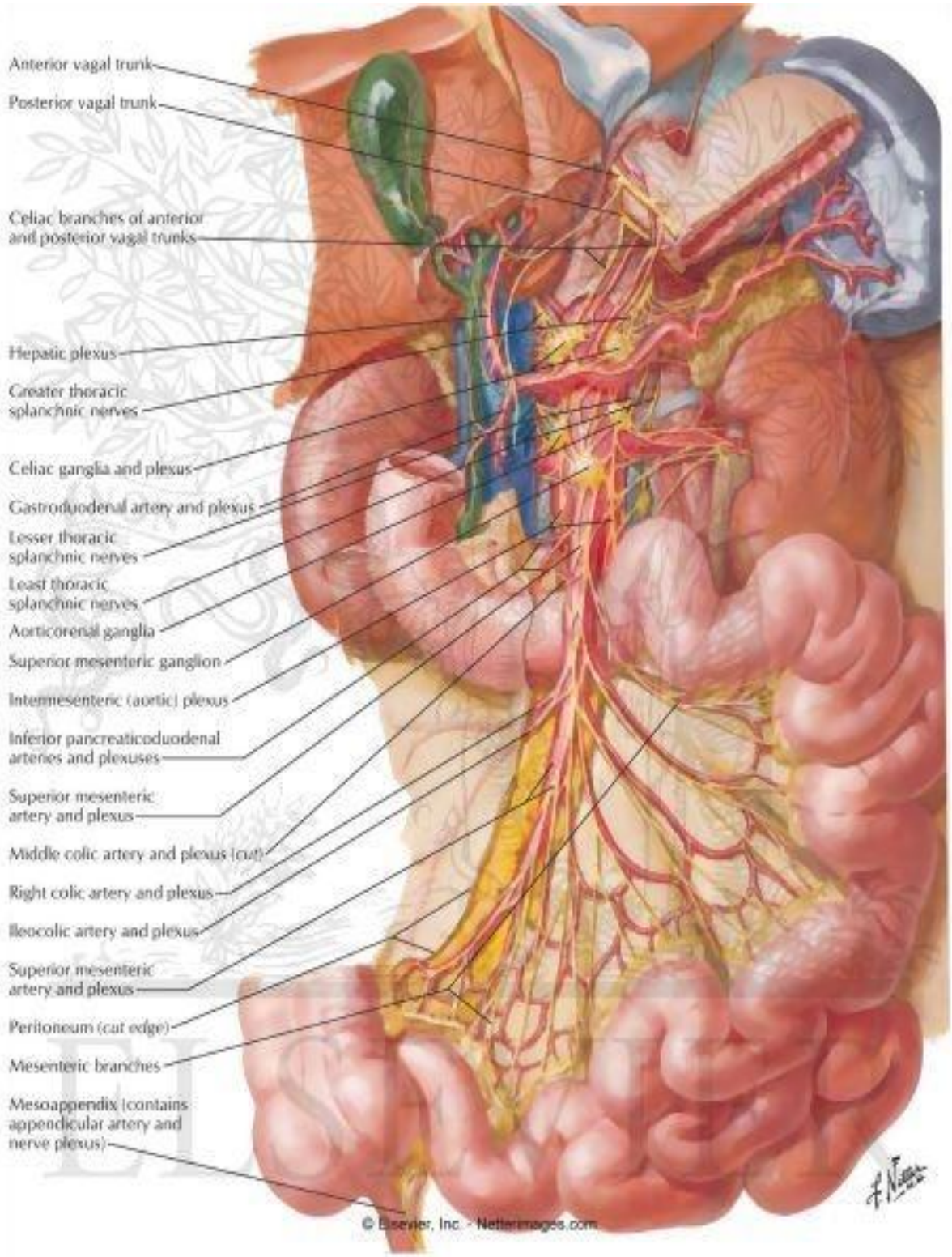


LYMPHATIC DRAINAGE OF THE BOWEL

INTESTINAL INNERVATION:

The autonomic nervous system including sympathetic, parasympathetic and enteric system innervates the gastrointestinal tract. The Extrinsic nerve supply is provided by the sympathetic and parasympathetic system and connects with the intrinsic nerve supply, made up of ganglion cells and nerve fibers within the wall. The intrinsic nervous system is made up of sub-serosal, muscular, and sub-mucosal plexuses. The sub-serosal plexus contains a network of thin nerves, without ganglia, that connects the extrinsic nerves with the intrinsic plexus. The Auerbach's plexus is situated between the outer and inner layers of the muscularis propria and it consists of ganglia and bundles of un-myelinated nerve fibres that connect with the ganglia. These axons originate from ganglion cells processes along with extrinsic vagus nerve and sympathetic ganglia. The deep muscular plexus is located on the mucosal aspect of the circular muscular layer of the muscularis propria. It does not contain ganglia; it innervates the muscularis propria and connects with the auerbach’s plexus.

The Meissner's plexus, innervate the muscularis mucosae and smooth muscle in the core of the villi. Fibers from this plexus also form a mucosal plexus that is situated in the lamina propria and provides branches to the intestinal crypts and villi. The ganglion cells of the submucosal plexus are distributed in two layers: One in the circular muscular layer of the muscularis propria and the other is contiguous to the muscularis mucosae. Ganglion cells are large cells, isolated or grouped in small clusters called ganglia. Ganglion cells have an abundant basophilic cytoplasm, a large vesicular round nucleus, and a prominent nucleolus. Ganglion cells are absent in the hypoganglionic segment 1 cm above the anal verge which is physiological.



NERVE SUPPLY TO THE INTESTINES

MICROSCOPIC FEATURES:

The small has certain histologic characteristics. The wall of the small intestine has four layers:

- Mucosa
- Submucosa
- Muscularis propria
- Adventitia / Serosa .

THE MUCOSA:

It is the innermost layer formed by glandular epithelium, lamina propria, and muscularis mucosae. The glandular epithelium forms crypts. The lamina propria that supports the epithelium consists of a layer of reticular connective tissue made of elastin, reticulin, and collagen fibers with lymphocytes, plasma cells, and eosinophilic granulocytes, as well as lymphatics and capillaries. The muscularis mucosae is a thin layer of smooth muscle at the boundary

of the mucosa and submucosa. The glandular epithelium is composed of various cell types:

1) Stem cells

2) Undifferentiated crypt cells

3) Absorptive cells

4) Goblet cells

5) Paneth cells

6) Enteroendocrine cells

7) M cells.

Stem cells are pleuri-potent cells located at the base of the crypt. The absorptive cells are high columnar cells with oval, basal nuclei, cytoplasm with abundant eosin and a periodic acid schiff (PAS) positive on the brush border. On electron microscopic examination, the brush border is seen to be composed of micro-villi, which are more numerous in the small intestines. Small bowel's microvilli increase the surface area of the cell 14 to 40

folds. Goblet cells are oval with flat basal nuclei and their cytoplasm is basophilic, metachromatic and PAS positive. Paneth cells are flask-shaped with an eosinophilic granular cytoplasm and a base positioned against the basement membrane. Paneth cells secrete lyzozymes and contain zinc, antimicrobial peptides, and growth factors. The mucosa contains neuro-endocrine cells. These neuroendocrine cells historically have been divided into argentaffin cells and argyrophilic cells. Argentaffin cells are also called enterochromaffin cells. These cells are oval or triangular and are also called halo cells and have a basal position in relation to the remaining epithelial cells and a pale cytoplasm filled with dark-stained granules. Variation in shapes and cell types has been detected with immunohistochemical staining. The APUD concept (amine precursor, uptake, and decarboxylation) provides common characteristics to these neuroendocrine cells. APUD cells are a group of cells with a common embryonic neural crest origin

and with similar cytochemical and electron microscopic features. Microscopically, entero - endocrine cells contain membrane bound granules with various sizes and electron dense cores, averaging 100 to 250 nm in diameter, and comprising large dense core vesicles and smaller, synaptic-type micro vesicles. Neuro secretory granules can be demonstrated specifically by immunofluorescence, with immunohistochemical stains such as neuron-specific enolase. Chromogranin enables visualization of the large-dense core vesicles and synaptophysin targets the small synaptic-like micro vesicles. ATP-dependent vesicular monoamine transporters has 2 isoforms in the form of VMAT1 and VMAT2. These antigens, derived from both the large and small dense-core vesicles, are expressed differentially in small dense core vesicles. Both are expressed in neuroendocrine cells, but VMAT1 is specific to serotinin cells, and VMAT2 is expressed in histamine-producing cells.

Specific immunohistochemical stains allow for identification of individual protein products of the neuroendocrine cells. Neuroendocrine cells also regulate secretion, absorption, motility, mucosal cell proliferation, and possibly immunobarrier control. Designation according to the nature of the stored peptide is preferable to characterization of neuroendocrine cells by letters. Serotonin-producing enterochromaffin cells, vasoactive intestinal polypeptide (VIP), and somatostatin D cells are distributed throughout the small and large intestine. M cells are special epithelial cells overlying the lymphoid follicles in the small bowel. M cells selectively bind, process, and deliver pathogens directly to the lymphocytes, macrophages, or other components of the lymphoid system. Interstitial cells of Cajal are present in the small bowel and are mesenchymal cells, located in the auerbach's plexus, the muscularis propria and the submucosa layer. The distribution of the interstitial cells of Cajal is similar in all age groups. They regulate intestinal motility as the pacemaker cells of the intestines. The interstitial cells of Cajal

are spindle-shaped, with long processes, and have large, oval, lightly stained nuclei with sparse peri nuclear cytoplasm. The interstitial cells of Cajal express the receptor for tyrosine kinase or CD117. Immuno histochemical stains that utilize antibodies against c-kit allow the interstitial cells of Cajal to be labeled.

THE SUBMUCOSA:

It lies between the muscularis mucosae and the muscularis propria, is a fibrous connective tissue layer that contains a nerve fiber plexus (Meissner's plexus) composed of nonmyelinated, postganglionic sympathetic fibers, and parasympathetic ganglion cells.

MUSCULARIS PROPRIA:

The muscularis propria responsible for contractility of small bowel has two layers of smooth muscle:

1) An inner circular coat
2) An outer longitudinal coat arranged in a helicoidal pattern.

A prominent nerve fiber plexus called the Auerbach's plexus, is found between these two muscle layers. Parasympathetic and postganglionic sympathetic fibers terminate in parasympathetic ganglion cells and postganglionic parasympathetic fibers terminate in smooth muscle. The adventitia is the outermost layer. When covered by a single layer of mesothelial cells, it is called the serosa.

SMALL INTESTINE:

The mucosa of the small intestine is characterized by mucosal folds known as plicae circulars or valves of Kerckring.

The mucosal folds are composed of mucosa and submucosa. Villi are mucosal folds that are of variable sizes and shapes in various parts of the small intestine. They may be broad, short, or leaf-like in the duodenum, tongue like in the jejunum, and finger like in distal ileum. The villous pattern also may vary in different ethnic groups. The height of the normal villus is 0.5 to 1.5 mm, villus height should be more than 1.5 times the thickness of the mucosa and 3 to 5 times the length of the

crypts. Two types of glands are present in the small intestine:

1)Brunner's glands

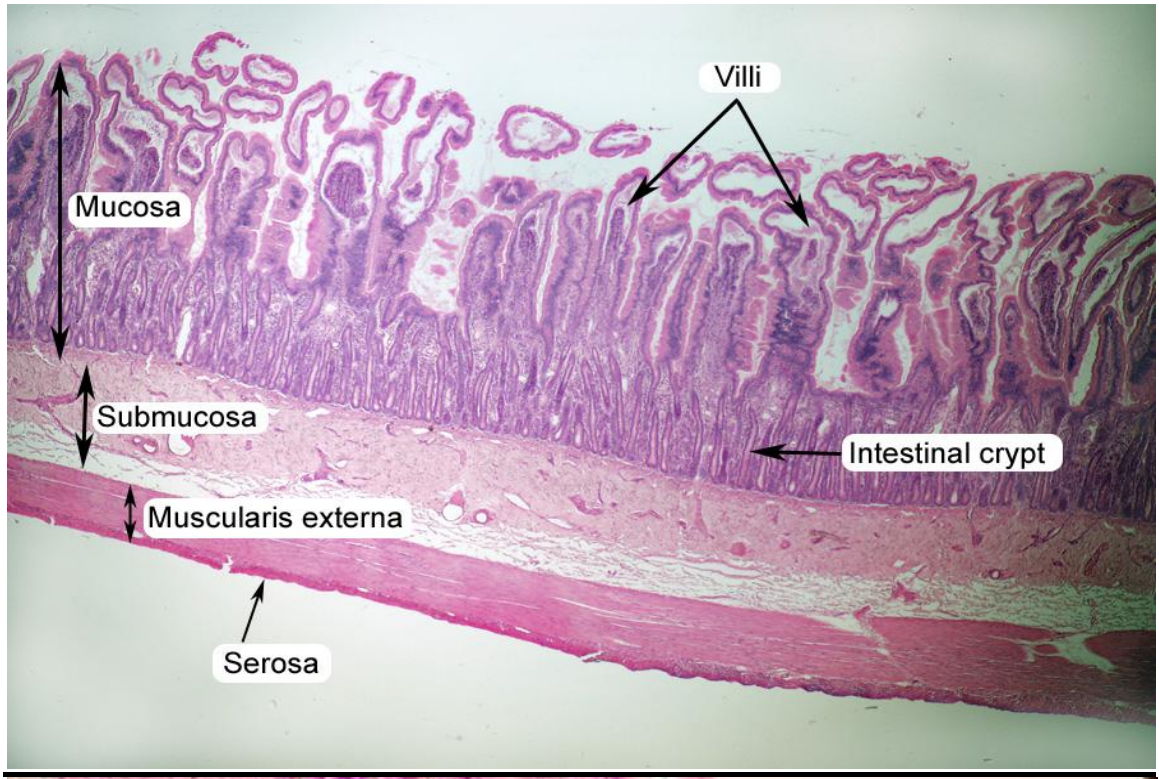
2)Crypts of Lieberkühn (intestinal crypts).

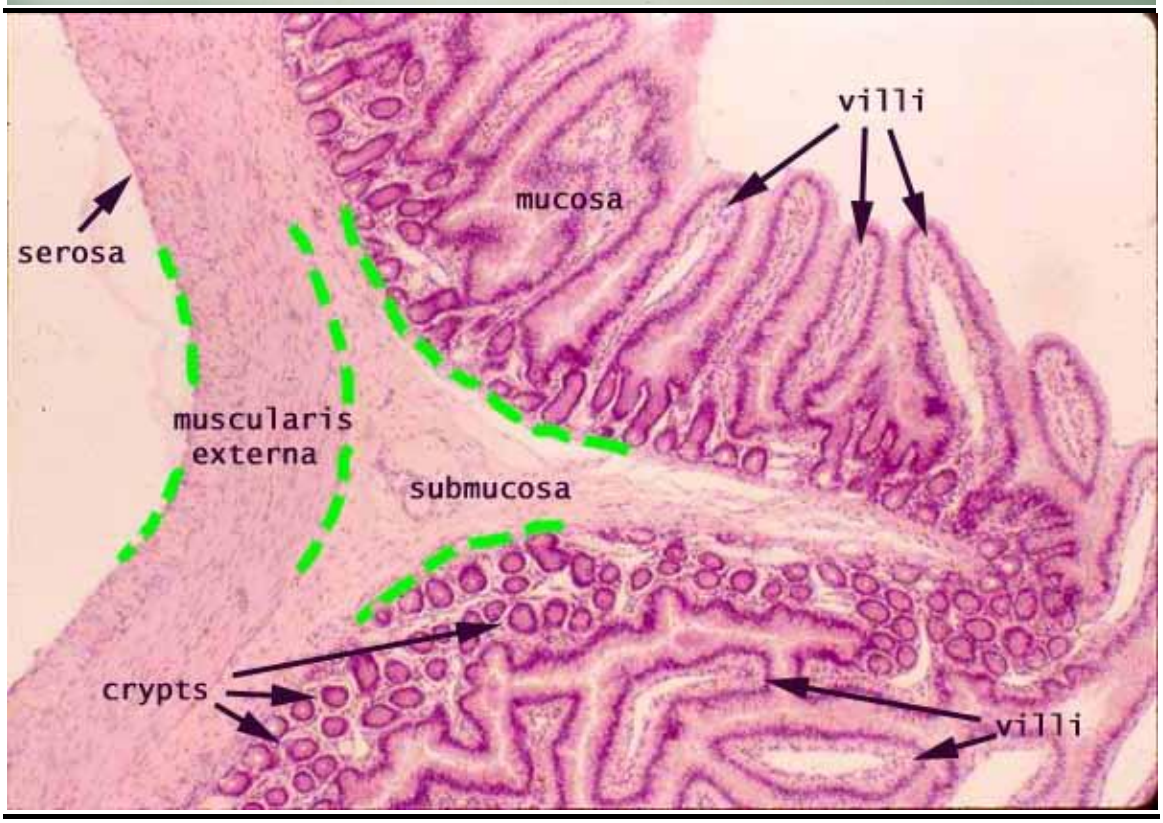
The first are submucosal glands found mainly in the first portion of the duodenum and in lesser numbers in the distal duodenum. In children, these glands also may be present in the proximal jejunum. Brunner's glands open into the intestinal crypts and resemble pyloric glands. Crypts of Lieberkühn are tubular glands that extend to the muscularis mucosae. Paneth and columnar cells are present in the base of the crypts. Above the base are absorptive cells and originate from undifferentiated cells and differentiate into goblet cells. Goblet cells predominate in the upper part of the crypt. Enteroendocrine cells are admixed with goblet cells. A certain number of CD3+ intraepithelial T lymphocytes normally are present in the villi. Smooth muscle is found in the lamina propria of the small intestinal villi. Plasma cells containing primarily IgA, and mast cells also are present. Lymphoid tissue is prominent in the lamina propria as solitary

nodules and as Peyer's patches seen in the submucosa. Peyer's patches are distributed along the anti-mesenteric border and are mostly present in the terminal ileum and decreases in number withage. Most enteroendocrine cells are present in the duodenum. Cells that produce ghrelin, gastrin, cholecystokinin, motilin, neurotensin, gastric inhibitory polypeptide and secretin are restricted to the small intestine. The proportions of these cells differ in the villi and crypts, as well as in different segments of the intestine. 90% of the villi epithelial cells are absorptive cells intermingled with goblet and enteroendocrine cells. The ratio of goblet to absorptive cells increases toward the proximal ileum. The interstitial cells of Cajal are maximum in the auerbach’s plexus of the small bowel

CROSS SECTION OF THE SMALL INTESTINE ON

ELECTRON MICROSCOPIC VIEW:





INTRODUCTION:

Small bowel obstruction (SBO) dates back the third or fourth century BC when Praxagoras created a enterocutaneous fistula to relieve small bowel obstruction. Till 1800's, Non-operative management of small bowel obstruction was the rule with the usage of laxatives, ingestion of heavy metals and manual reduction of hernias. After the introduction of aseptic surgical techniques and asepsis, surgical techniques for small bowel obstruction came to the fore. Despite a reduction in the mortality of patients with small bowel obstruction through the usage of isotonic fluids, small bowel decompression and antibiotics, A thorough clinical history and workup with awareness of the complications is a must in handling patients with small bowel obstruction.

CAUSES:

The causes of Small bowel obstruction in adults can be classified as follows:

1) **Lesions extrinsic to the intestinal wall:**

- Adhesion (commonly post - operative).
- Hernias:
    i) External hernias including ventral, inguinal, femoral or umbilical hernias.
    ii) Internal hernia through foramen of Winslow, paraduodenal and diaphragmatic hernias or post-operative mesenteric defects.
- Neoplastic causes like extra intestinal neoplasms, intra abdominal collections and carcinomatosis.

2) Lesions intrinsic to the intestinal wall:

- Congenital:
    i) Malrotations
    ii) Duplications

iii) Cysts

- Inflammatory:

  i) Tuberculosis

  ii) Crohn’s Disease

  iii) Actinomycosis

  iv) Diverticulitis

- Neoplastic

  Primary and metastatic malignancies.

- Traumatic

  Hematomas and ischemic strictures.

- Miscellaneous

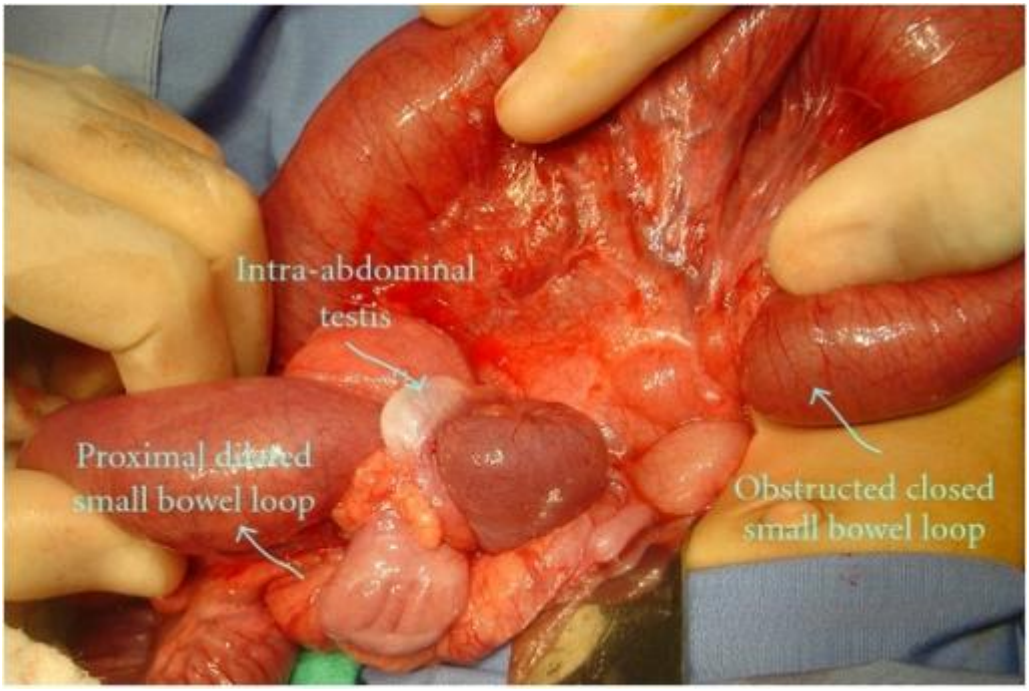
  i) Endometriosis

  ii) Intussusception

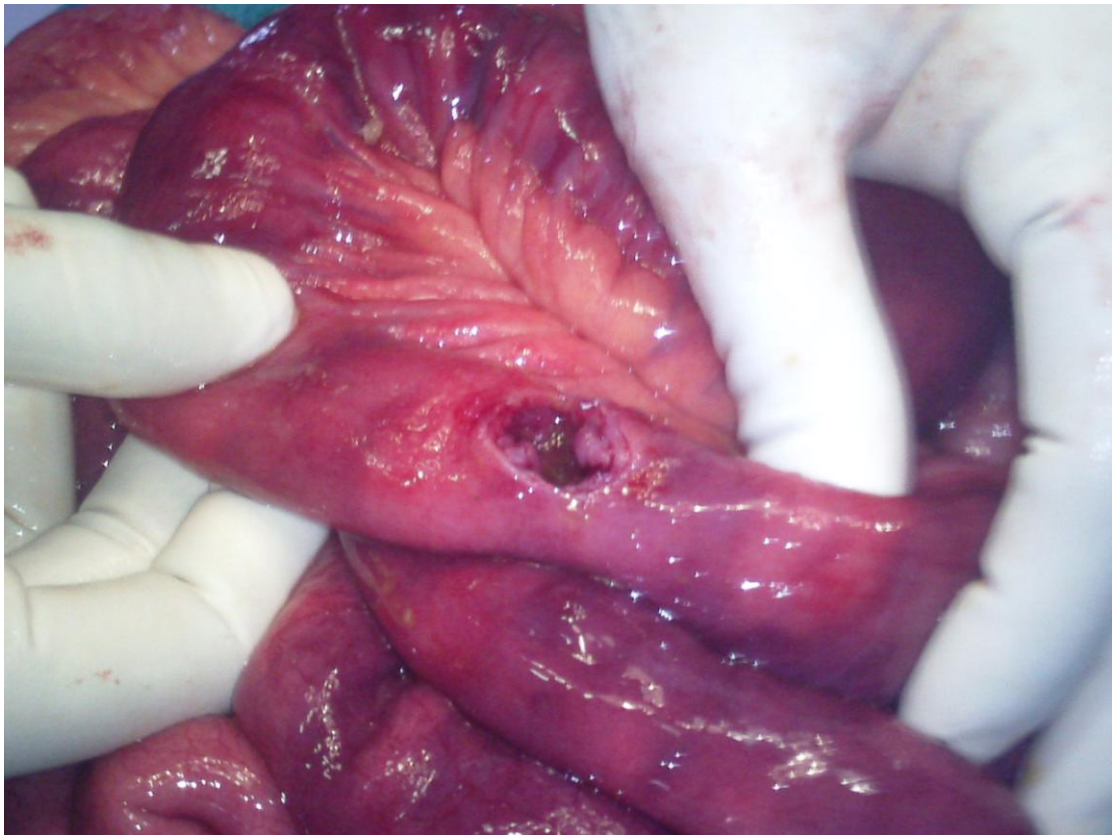
  iii) Radiation enteropathy and stricture.

3) Intraluminal obstruction

- Gallstone
- Bezoar
- Foreign body
- Enterolith

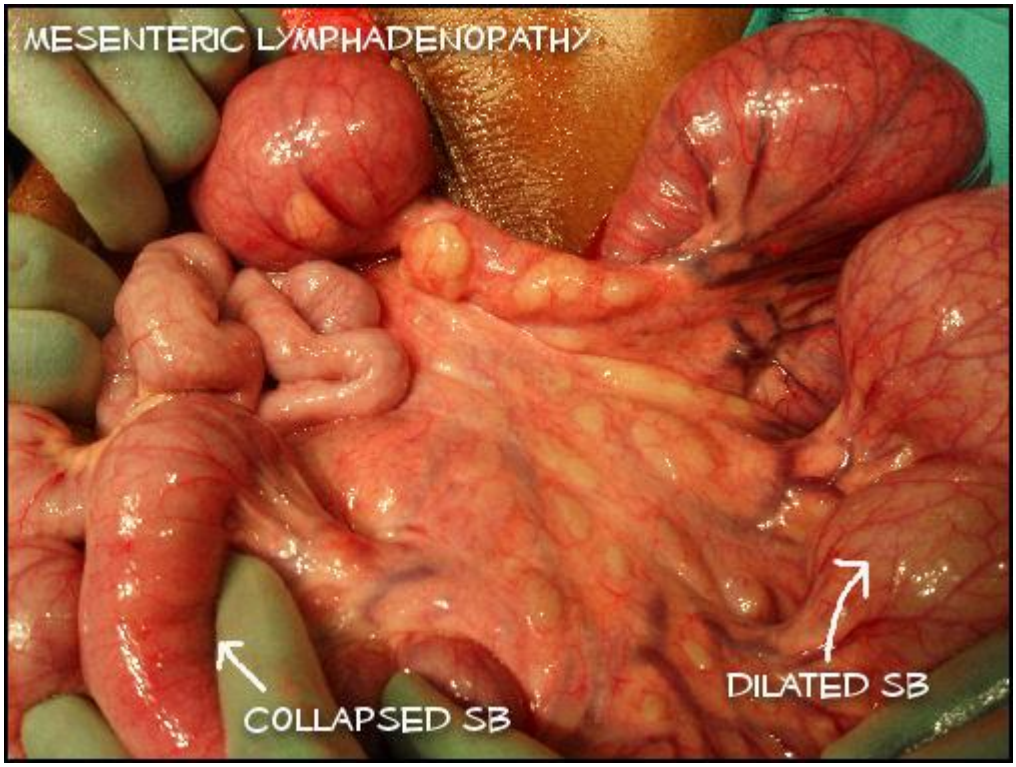


INTERNAL HERNIA PRESENTING AS SMALL BOWEL OBSTRUCTION

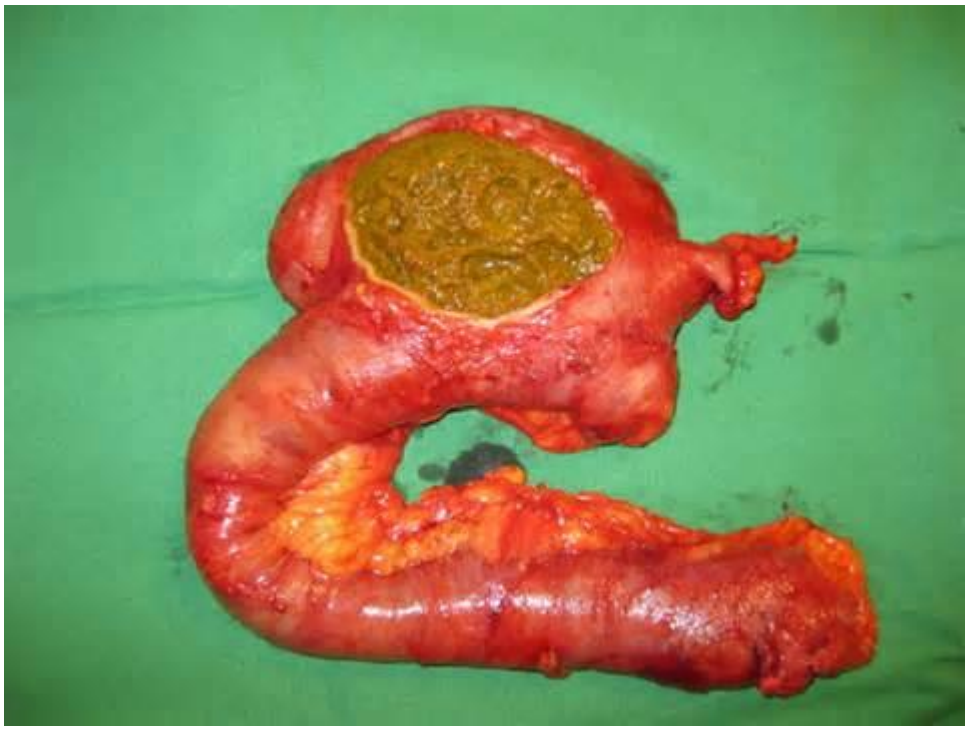


SMALL BOWEL PERFORATION PRESENTING AS

SMALL BOWEL OBSTRUCTION

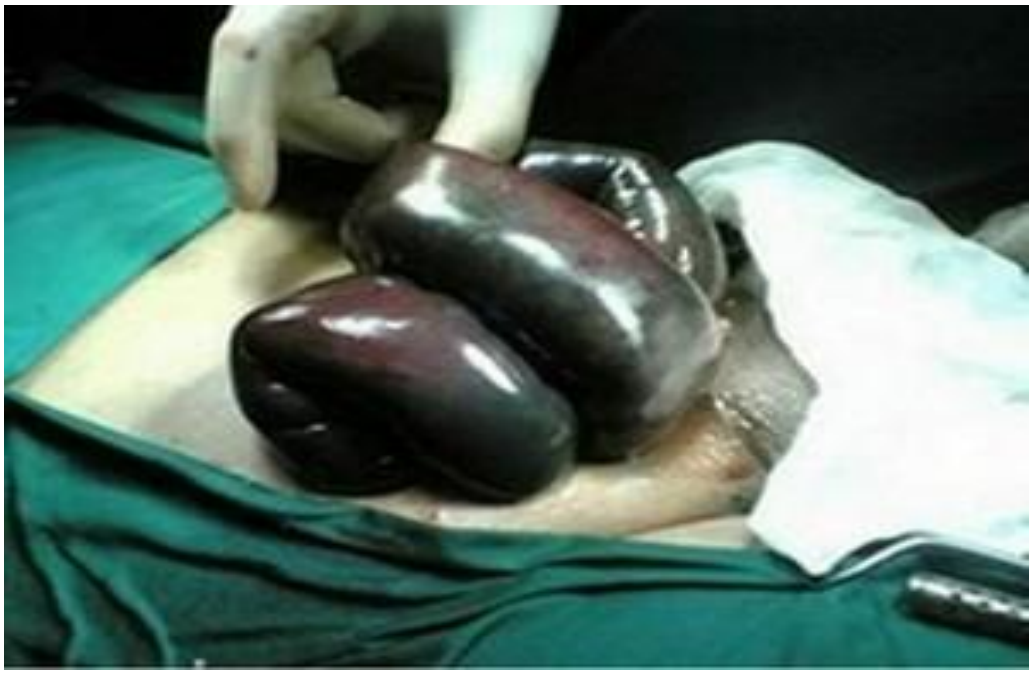


ABDOMINAL TUBERCULOSIS PRESENTING AS SMALL BOWEL OBSTRUCTION.



BEZOARS CAUSING SMALL BOWEL

OBSTRUCTION



STRANGULATED HERNIAS PRESENTING AS SMALL BOWEL OBSTRUCTION

Hernias have accounted for more than 50% of the cases of mechanical small bowel obstruction since the start of the 20th century. In industrialised countries, post operative adhesions have become the most common cause of small bowel obstruction. This change in trend can be attributed to the increase in number of elective hernia repairs. According to Moran et al, Postoperative adhesions are the commonest cause

of small bowel obstruction (SBO), a frequent surgical emergency. Adhesive obstruction is potentially lethal and a crucial aspect in management is to differentiate whether there is actual, or impending, small bowel ischaemia and therefore a need for emergency surgery. There are no completely accurate imaging or haematological techniques to exclude the requirement for surgery. Modern computerized tomography (CT) has been a significant advance in noninvasive assessment of SBO and may demonstrate the cause of the obstruction and suggest the presence of bowel ischaemia.

It is important to note that adhesions may not be the cause of SBO in a patient who has had abdominal surgery. Recurrent cancer, an obstructive colon lesion in the presence of an incompetent ileocaecal valve, an occult hernia, small bowel arterial or venous ischaemia, amongst others may be the cause and CT may elucidate some of these causes and help plan management.

Increasing utilization of laparoscopic surgery may reduce the extent and incidence of adhesions and laparoscopic adhesiolysis, in experienced hands, may be successful in managing acute obstruction or alternatively as a planned procedure when the obstruction has resolved. Adhesive SBO remains a common surgical emergency and there is no substitute for repeated examination by a surgeon, capable of performing a laparotomy, in the optimal management of these complex patients.

According to Chen et al based on a study in China done on 705 patients on the etiological factors and overall mortality of the patients with acute intestinal obstruction, and to explore the rational period of conservative therapy before operation, it was concluded that the epidemiological transition to adhesive obstruction still exists in China, and it is similar to that in Western countries. Nearly half of the patients with simple obstruction may achieve palliation by conservative therapy. Surgical intervention is indicated for the patients with prolonged and non-palliated simple obstruction, or strangulation disease within the first 24 hours. Adhesions secondary to pelvic,

gynaecological and colorectal procedures are responsible for more than 60% of the causes of small bowel obstruction. This can be attributed to the fact that bowel is more mobile in the pelvis and more or less a static organ in the upper abdomen.

About 20% of the cases of small bowel obstruction are due to malignancy. Most of them are metastatic deposits and peritoneal implants that spread from an intra-abdominal primary. Extrinsic compression of the small bowel may be due to large tumors resulting in small bowel obstruction. Interestingly, large bowel neoplasm of caecum and ascending colon also produce small bowel obstruction. Small bowel malignancies producing obstruction is an extremely rare phenomena.

10% of the cases of small bowel obstruction can be attributed to hernias. Majority of these cases are either ventral or inguinal hernias followed by internal hernias secondary to previous surgeries. Less common causes would be femoral, lumbar, obturator and sciatic hernias.

5 % of the cases of SBO are caused by Crohn's Disease. This is one of the causes of SBO where conservative management plays an important role as most of the obstruction is mainly caused by acute inflammation and edema. In case of long standing Crohn's disease, Strictures may develop needing surgical intervention.

Another important but overlooked cause is an intra-abdominal abscess, most commonly from an appendicular rupture or dehiscence of an intestinal anastomoses. It produces a local ileus adjacent to the abscess. Kinking of the small bowel may also happen as it occasionally forms the wall of the abscess cavity.

Intussusception, polyp, gallstones entering bowel through a cholecysto-enteric fistula, enteroliths, foreign bodies all account for 2-3% of the cases.

PATHOPHYSIOLOGY:

In the early phase of intestinal obstruction, increased effort by the bowel to push the contents beyond the point of obstruction occurs. This effort is seen both above and below the obstructing point and this can result in diarrhoea despite the patient having

obstruction. As obstruction worsens the intestines become fatigue resulting in dilation and contractions becoming less frequent and less intense.

Once the bowel starts to dilate intra luminal accumulation of water and electrolytes occurs. This massive third space fluid loss accounts for hypovolemia and dehydration in SBO. In case of proximal obstruction patient also has episodes of vomiting which results in dehydration associated with hypokalemia and metabolic alkalosis. Distal obstruction of small bowel produces less changes in electrolyte levels. Dehydration is accompanied by oliguria, azotemia, hypotension and shock. Intestinal obstruction results in increased intra luminal pressure which causes ischemia of the bowel. This phenomenon is most commonly seen in closed loop obstruction.

In the absence of intestinal obstruction the jejunum and proximal ileum are almost sterile. With SBO the flora of the intestines changes in both type (E.coli,S.fecalis and Klebsiella spp.) and the quantity(10^9-10^10/ml).

CLINICAL MANIFESTATIONS AND DIAGNOSIS:

The cardinal symptoms of SBO include colicky abdominal pain, abdominal distension, obstipation, nausea and vomiting. The typical crampy abdominal pain in SBO occurs in paroxysms at 4-5 minute interval and is more frequently associated with proximal obstructions. Nausea and vomiting are more commonly associated with high intestinal obstruction. As SBO progresses abdominal distension develops resulting in obstipation. As the obstruction becomes more complete the vomitus becomes more feculent indicate a late and established intestinal obstruction.

PHYSICAL EXAMINATION:

Patient presents with hypotension and tachycardia. Fever is a sign of strangulation. Abdominal distension may be present. Physical examination for previous scars should be done. Hyperactive bowel sounds with audible rushes associated with rapid peristalsis (Borborgymi) may be present. As obstruction

worsens, bowel sounds may be completely absent. Incarcerated hernias in groin, femoral triangle and obturator foramen should be carefully looked upon. Per rectal examination should be done for intraluminal masses and stool examination should be done for occult blood.

RADIOLOGICAL AND LABORATORY EVALUATION:

Plain radiographs of the abdomen may confirm clinical suspicion and demonstrates the level of obstruction. It has a diagnostic accuracy of about 60%. Features on the plain radiograph include dilated loops of small bowel, without evidence of colonic distension. Erect films of the abdomen may demonstrate step wise air fluid levels. Foreign bodies, gall stones can also be picked up on the abdominal x-ray. Further evaluation is needed in uncertain cases or in cases where we are unable to differentiate complete from partial obstruction.

The next line of investigations include a contrast enhanced computerized tomography of the abdomen (CECT). Its use is mainly to differentiate a partial from a complete obstruction and

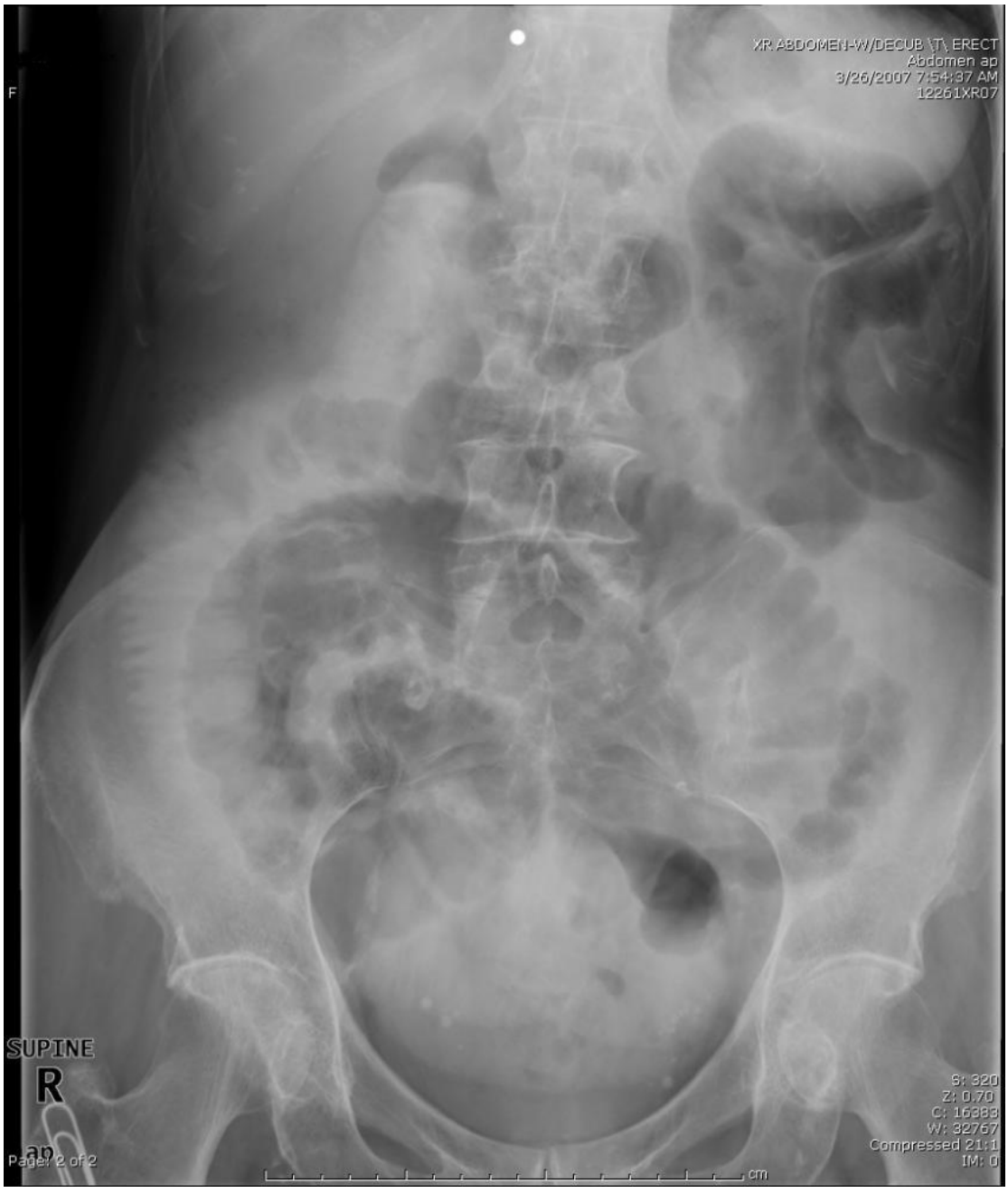
also to determine the location and cause of the obstruction. Its added benefit is in patients suspected to have extra-luminal cause of obstruction and suspected cases of strangulation.

Barium study is another adjunctive tool in patients with presumed obstruction. Enteroclysis is used to assess the level of obstruction and is the definitive study for patients with low grade, intermittent, recurrent SBO. The main drawbacks include the need for naso-enteric intubation, slow transit of the contrast material in an already paralyzed bowel and radiological expertise.

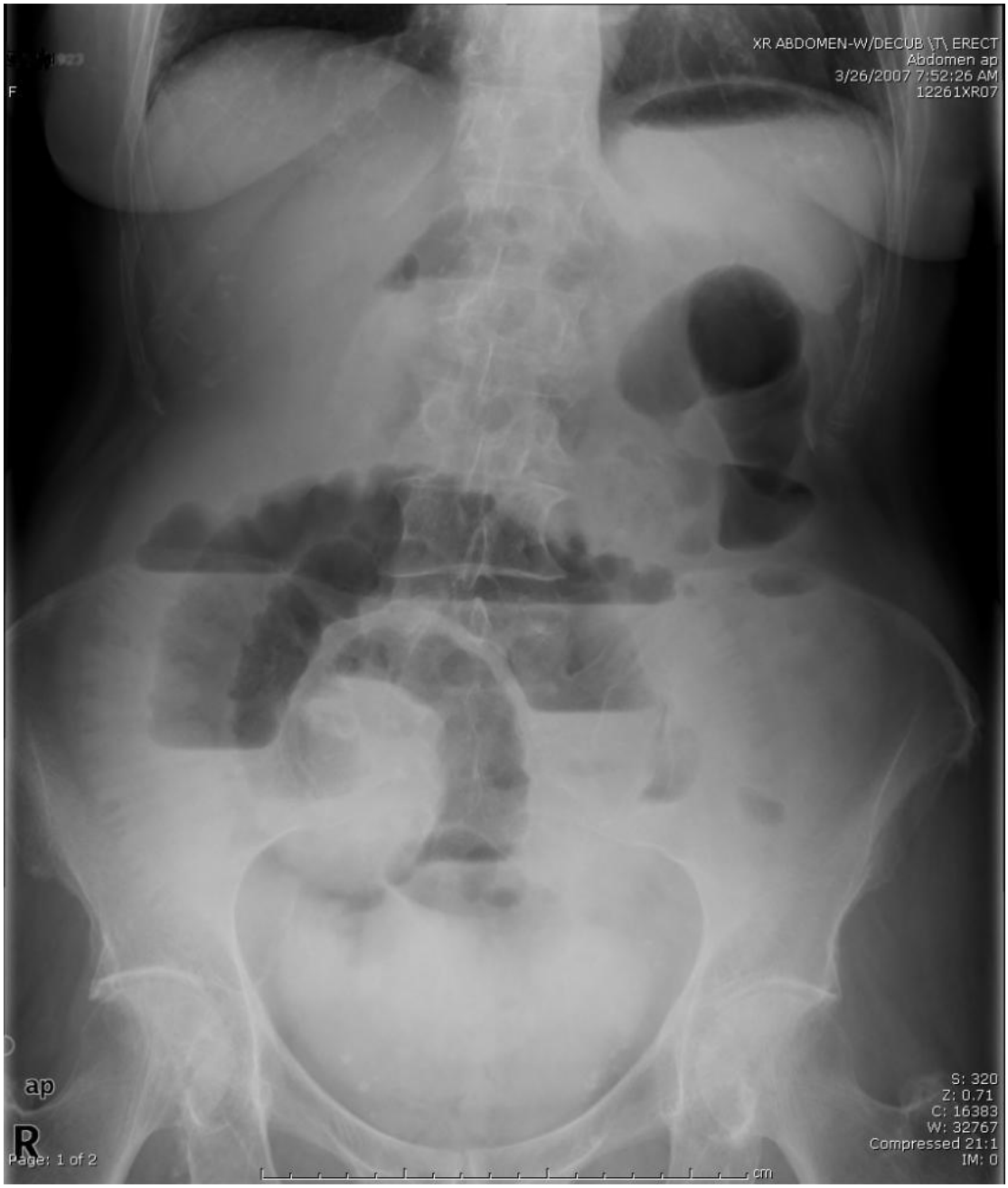
Ultrasonography of the abdomen(USG) is useful in pregnant patients to overcome the ill-effects of radiation. Magnetic Resonance Imaging (MRI) offers no better diagnostic efficacy as compared to CECT in SBO.

Laboratory investigations are mainly done to assess the severity of dehydration. Routine monitoring of serum electrolytes with creatinine is mandatory as it indicates the success of fluid resuscitation. Elevated hematocrit values suggest

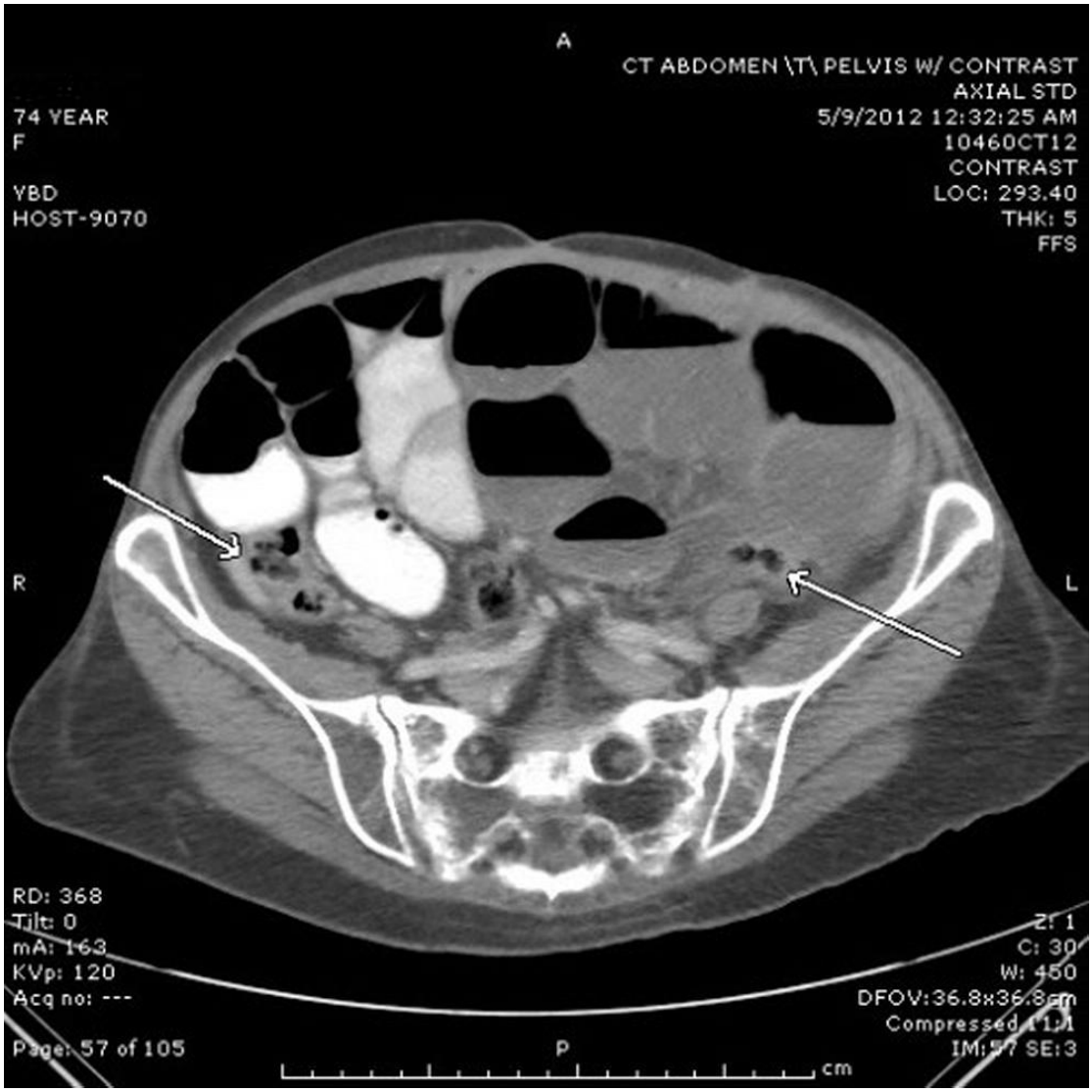
hemoconcentration. Leucocytosis indicates strangulation of the small bowel.



iii) Small bowel obstruction in supine film



iv) Small bowel obstruction on erect film



v) Small bowel obstruction on CT Abdomen

SIMPLE VERSUS STRANGULATING OBSTRUCTION:

Simple SBO's involve mechanical blockade of the flow of luminal contents without the compromise in the viability of the bowel wall. But, Strangulating SBO involves compromise in the blood flow leading to intestinal infarction. The main features differentiating from simple obstruction include tachycardia, fever, elevated white blood cell count and non-cramping abdominal pain. However, a number of studies have shown that no clinical parameters or laboratory measurements can accurately detect or exclude the presence of strangulation in all cases.

CECT Abdomen is useful in detecting late cases of irreversible ischemia (Pneumatosis intestinalis, portal venous gas). Newer laboratory measurements of Serum D-lactate, creatine phosphokinase isoenzyme (BB subtype) or intestinal fatty acid binding proteins are only investigational and cannot be applied widely. Newer Non invasive determinations of mesenteric

ischemia by a Super Conducting Quantum Interference Device (SQUID) is under trial.

TREATMENT:

Correction of dehydration remains the principal aspect in the management of intestinal obstruction. Aggressive replacement with Ringer's Lactate solution is the primary step. Strict urine output monitoring should be done. Central venous pressure monitoring and placement of a Swan-Ganz catheter may be used in case of old patients with large fluid requirements. Prophylactic broad spectrum antibiotics are preferred based on the principal of bacterial translocation that occurs in simple SBO's.

Naso-gastric suction through a Ryle's tube forms an important aspect of patient care. It empties the stomach, reduces the risk of pulmonary aspiration and further abdominal distension due to the swallowed air. Patients with partial intestinal obstruction can be treated with resuscitation and decompression alone. It is a satisfactory line of treatment in 60-85% of the cases. Higher

grades of obstruction may need an operative intervention. The decision to continue non-operative management of a patient with small bowel obstruction is based on the clinical acumen of the physician and constant vigilance regarding worsening of the patient.

Non-operative Management of small bowel obstruction is always a calculated risk with the possibility of overlooking strangulation of the bowel. Retrospective studies have shown that a 12-24 hours delay in surgery is safe but the incidence of strangulation along with other complications increases significantly after this period.

Post operative adhesions, being the most common cause of SBO may be treated conservatively/operative management. Great care must ne excised to avoid serosal injuries and enterotomies during adhesiolysis. Incarcerated hernias can be managed by manual reduction of the herniated bowel segment and closure of the defect.

In case of malignant tumors with an obstruction, non-operative measures are the ideal technique of management. In case of a complete obstruction, bypassing the obstructed point is the best option compared to bowel resection.

Acute SBO's secondary to Crohn's disease resolves spontaneously. If strictures develop, patients may need resection/ stricturoplasty.

Radiation induced SBO's can be managed by tube decompression and corticosteroids. In case of chronicity, laparotomies may be need needed to excise the necrosed bowel.

If viability of the bowel is an issue during laparotomy, bowel is generally taken out and placed in warm saline for 15-20 minutes and if the color and peristalsis is satisfactory, then it is safe to retain the bowel. Others methods to study viability including Doppler study, flourescein fluorescence may supplement clinical judgement. Second look laparotomy is done 18-24 hours after the primary surgery to assess bowel viability and is mainly

indicated in cases where the patient deteriorates after the initial surgery.

Laparoscopic management of acute small bowel obstruction is indicated in the following conditions:

- Mild abdominal distension with adequate visualisation.
- Proximal obstruction.
- Partial obstruction.
- Anticipated single band obstruction.
- Less than 3 previous surgeries.

In cases of recurrent SBO's, External plication procedures which were initially used have all been abandoned due to development of fistulas, gross leakage and peritonitis. Long intestinal tubes, gastrostomy or jejunostomy have been tried for recurrent SBO but have not been successful. According to Komatsu et al, among patients with adhesive small bowel obstruction (ASBO) initially managed with a conservative strategy, predicting risk of operation is difficult. On

investigating ASBO patients at 2 different periods to derive and validate a clinical prediction model for risk of operation, 154 patients were enrolled into the derivation cohort and 96 into the validation cohort. Based on the derived scoring, including **age >65 years, presence of ascites, and ryle's tube output >500 mL on day 3**, each patient was classified into 1 of 4 risk classes from low risk to high risk. When applied to the validation cohort, the positive predictive value (PPV) for operation in the high-risk class was 72%, while the negative predictive value (NPV) in the low-risk class was 100% with high sensitivity (100%) and specificity (96%) which led to a conclusion that the prediction model performs well for risk stratification of need for surgical intervention following conservative strategy among ASBO patients.

MATERIALS AND METHODS

MATERIALS

A Retrospective study conducted in PSG Institute of Medical Sciences and Research presenting with small bowel obstruction in 50 patients

QUESTIONNAIRE

To assess all patients according to the following:

- IP NO:
- CLINICAL PRESENTATION:
- AGE:
- SEX:
- RADIOLOGICAL FINDINGS:
- MODALITY OF TREATMENT:
- IF SURGERY; INTRAOPERATIVE FINDINGS

INCLUSION CRITERIA:

- All age groups above 16 years of age.
- Radiologically proven cases of small bowel obstruction.

EXCLUSION CRITERIA:

- Cases below 16 years of age.
- Large bowel obstruction.

METHODOLOGY:

In this study, 50 radiologically proven cases of small bowel obstruction were taken. The history was recorded by the principal investigator and the mode of presentation, duration and progression was recorded. Any history of previous surgeries was also taken into account.

The age of presentation, complete blood picture and ryle's tube output of these patients were recorded based on the case sheet of the patient.

Radiological findings in all these patients were taken into account and the modality of the management was looked upon.

The patients underwent either surgery or managed conservatively. If surgery was planned, the intra-operative findings were looked at and documented. The prognosis of either line of management was also documented. All details are documented in a questionnaire format and confidentiality preserved with the principal investigator of this study.

RESULTS AND OBSERVATIONS

OBSERVATION

ANALYSIS OF DATA

- Total of 50 cases were included in this study
- They were all radiologically proven cases of small bowel obstruction.
- All the patients case records were analysed for the age os presentation,ryles tube output and complete blood picture.
- Patients with adhesive SBO, a prognostic scale to predict the need for surgery was validated.

DIAGNOSIS	NO.OF CASES
Adhesive obstruction	14
Obstructed and strangulated hernias excludes internal hernias	8
Abdominal tuberculosis	7
Crohn’s disease with/without ileal perforation	5
Stricture – ileal,jejuna	5
Retroperitoneal tumors	3
Acute mesenteric ischemia presenting as gangrenous bowel	2
Volvulus	1
Intusussception	1
Gastrointestinal Stromal Tumors	1
Internal hernias	1
Small bowel diverticulum	1
Ogilve’s syndrome	1

DISCUSSION

A retrospective case study on small bowel obstruction was done in 50 patients and the study infers the following details.

This mean age of the study group is 52.76 years and is almost equal to the study conducted in 367 patients in eastern Indian population[4] .

80% of the study population lies within 40-70 years agr group[4] .

The sex predominance was more towards males(66%).

The most common cause of small bowel obstruction was post operative adhesions (28%) followed by tuberculosis abdomen and ileal perforation secondary to Crohn's disease. This is in correlation to most of the studies done on small bowel obstruction[1] but differs from a study in eastern india where obstructed hernias tend to be the most common cause of obstruction[4].

This study also tests the scoring system used for predicting the need for surgery in patients presenting with adhesive small bowel obstruction based on the study done by Komatsu et al.

Among the patients with adhesive small bowel obstruction, 8 patients(57.14%) were taken up for surgery.

Post operative complications were present in 4 patients(8%). The only complication noted was surgical site infection.

CONCLUSION

Acute intestinal obstruction is one of the most common cause for surgical admissions worldwide. The etiology varies; however, adhesions[1] appear to be the most common cause in india and in the western world as well as parts of asia and middle east. In our study, adhesions appeared to be the most important cause followed by tuberculosis abdomen and ileal perforation. The fact that intestinal tuberculosis has a major share of the cases can be attributed to the high prevalence of tuberculosis in the Indian population. The gender discrepancy can be attributed to the fact that most obstructed hernias are more common in males and women in rural india are mostly housewives which limit their exposure to tubercle bacilli in contrast to the males. Also, volvulus and malignancies of GIT are more in males as compared to females.

A critical factor in managing these patients is to determine whether patients can be subjected to conservative management or to emergency surgery. Conservative therapy was typically advocated for patients with pre-operative diagnosis of adhesive obstruction when the physiological parameters were within

normal limits (keeping a low threshold for surgery) as also in patients with intestinal tuberculosis who presented with sub-acute intestinal obstruction. Patients with adhesive obstruction were diagnosed based on the history (recurrent bouts of intestinal obstruction managed conservatively, history of laparotomy or appendicectomy in the past 2 years) and were included in the study only on radiological confirmation of the diagnosis.

There are several drawbacks in our study. Since our institution is a tertiary referral hospital, we mainly attended to cases which could not be managed under the primary or secondary level of health care; therefore, an accurate etiological assessment of acute intestinal obstruction might not have been reflected in our study. Also , most of our patients were from a poor socioeconomic status with a high prevalence of malnutrition; therefore, the morbidity and mortality are likely to be higher.

We also recommend the usage of a simple stratification model for patients with adhesive small obstruction as a predictor for surgery as it performs well for risk stratification.

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8. Pattern of acute intestinal obstruction: Is There a change in the underlying etiology?

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