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A STUDY ON SMALL BOWEL OBSTRUCTION

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CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF SMALL BOWEL OBSTRUCTION AND ITS VARIOUS CAUSES AND THE MANAGEMENT IN ADULTS"** is the bonafide record work done by **Dr. K.MANIVANNAN** submitted as partial fulfillment for the requirements of **M.S. Degree Examinations Branch I, General Surgery, SEPTEMBER 2006.**

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INTRODUCTION

Acute small bowel obstruction continues to be a frequent emergency till date. It accounts for 2/3 of abdominal cat atrophies. Delay in diagnosis has a direct bearing on the morbidity and mortality.

Physical signs and their interpretation reach a high pinnacle of importance in the diagnosis. Frequently an urgent and all-important decision has to be reached by their aid alone. It is one of the emergencies where as quickly as possible we act, the result will be remarkable.

It is one of the gravest emergencies that reveal the talents of the surgeon in all aspects.

Investigations and resuscitation protocol for all patients were identified. All the cases in the present study were treated surgically.

AIM OF THE STUDY

1. TO EVALUATE THE COMMON CAUSES OF ACUTE SMALL BOWEL OBSTRUCTION

2. TO IDENTIFY THE ETIOPATHOGENSIS

3. TO EVALUATE THE VARIOUS MODE OF PRESENTATION

4. TO STUDY THE VARIOUS MODALITIES OF TREATMENT IN THIS CENTER.

5. TO EVALUATE THE MORBIDITY AND MORTALITY OF ACUTE SMALL BOWEL OBSTRUCTION

REVIEW OF ANATOMY AND EMBRYOLOGY

Exact cause of intestinal obstruction cannot be predicted in certain cases because it is well said the fact abdomen is a magic box.

A surgeon who is opening on a case of obstruction must be able to tackle any eventuality that comes across in the odd hours. For this detailed knowledge of anatomy and basic knowledge of embryology is essential.

At an early stage of development, alimentary canal is represented by a tube suspected in the midline of abdominal cavity by a ventral and dorsal cavity. It consists of tree portions.

	PARTS	**BLOOD SUPPLY**	**FUNCTION**
FOREGUT	STOMACH+ Duodenum as for as the major duodenal papilla	COELIAC AXIS	DIGESTIVE FUNCTION
MIDGUT	Ampulla to the junction of middle with the left third of transverse colon	SUPERIOR MESENTERIC ARTERY	ABSORPTIVE FUNCTION
HINDGUT	Left third of transverse colon+left colon	INFERIOR MESENTERIC ARTERY	EXCRETORY FUNCTION

ROTATION OF GUT

I STAGE(5-10 WEEKS OF EMBRYO)

1) Review from ventral side, the loop undergoes an anticlockwise rotation by 90* so that it now lies in a horizontal plane.

2) The prearterial segment now undergoes greater increase in length of form coils of jejunum and ileum. Intestinal loops still be outside the abdominal cavity to the right side of the distal limp.

II STAGE(10-11 WEEKS)

1) The coils of small bowel now to return to abdominal cavity.As they do so, the midgut loops undergoes a further anticlockwise rotation.As a return jejunum and ileum pass behind the superior mesenteric artery into left half of abdominal cavity. The duodenum comes to lie behind the artery and coils of jejunum and ileum occupy the posterior and left side of abdominal cavity.

2) The colonic portion then returns to abdominal cavity with anticlockwise rotation so that transverse colon lies anterior to superior mesenteric artery and caecum lies on right side at subhepatic portin. Ascending colon cannot b e demarcated at this stage.

III STAGE

Caecum descends to right iliac fossa and ascending colon, caecum, descending colon, rectum become retroperitoneal by fusion of their mesenteries with the posterior abdominal wall.

SURGICAL ANATOMY OF SMALL INTESTINE

The jejunum and ileum measures about 20feet(6meters)long. The upper two fifths of the length make up the jejunum. Each has distinctive features, but there is a gradual change from one to the other. The jejunum begins at the duodenojejunal flexure, and the ileum ends at the ileocaecal junction.

The coils of jejunum and ileum are freely mobile and are attached to the posterior abdominal wall by a free shaped fold of peritoneum known as mesentery of the small intestine. The long free edge of the fold encloses the mobile intestine. The short root of the fold is continuous with the parietal peritoneum on the posterior abdominal wall along a line that extends downward and to the right from the left side of 2nd lumbar vertebra to the region of the right sacroiliac joint. The root of the mesentery permits the entrance and exit of the branches of the superior mesenteric artery and vein, lymph vessels and nerve into the space below the two layers of peritoneum forming the mesentery.

BLOOD SUPPLY

The arterial supply is from branches of superior mesenteric artery. The intestinal branches arise from the left side of the artery and run in the mesentery to reach the gut. They anastomose with one another to form a series of arcades. The lowest part of the ileum is also supplied by ileocolic artery.

VENOUS DRAINAGE

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

LYMPH DRAINAGE

The lymph vessels pass through many intermediate mesenteric nodes and finally reach the superior mesenteric nodes, which are situated around the origin of superior mesenteric artery.

NERVE SUPPLY

The nerves are derived from the sympathetic and parasympathetic (vagus) nerves form the superior mesenteric plexus.

DIFFERENCES BETWEEN JEJUNUM AND ILEUM		
FEATURES	**Jejunum**	**ILEUM**
1. Location Walls	Occupies upper and left parts of the intestinal area	Occupies lower and right parts of the intestinal area
2. Walls	Thicker and more vascular	Thinner and less vascular
3. Lumen	Wider and often empty	Narrower and often loaded
4. Mesentery	a)windows present b)Fat less abundant c)Arterial arcades,1 or 2 d)vasa recta longer and fewer	a)No windows b)Fat more abundant c)Arterial arcades,3 or 6 d)vasa recta shorter and more numerous
5. Circular Muscular folds	Longer and more closely set	Smaller and sparse and more closely set
6. Villi	Longer,thick and more abundant	Longer,thick and more abundant
7. Peyer's patches	Absent	Absent
8. Solitary lymphatic follicles	Fewer	Fewer

CLASSIFICATION AND ETIOLOGY

Intestinal obstruction is divided into two main types.

Intestine obstruction

Mechanical **Neurogenic**

(DYNAMIC) (ADYNAMIC)

In Mechanical obstruction the intestinal contents are prevented from Passing along the bowel by acute obstruction of the lumen of the gut.

In neurogenic obstruction the peristalsis ceases and no true propulsive Waves occur and so the intestinal contents do not transverse the bowel.

ACCORDING TO THE ETIOLOGY:

This is the most useful type of classification, which reveals the underlying cause of obstruction.

1.**Causes in the lumen:** Gall stone, food bolus, Fecal impaction, Barium, bezoar, worms etc.,

2.**causes in the wall:** Congenital atresia, Bowel neoplasms, inflammatory strictures etc.,

3.**causes outside the wall**: Strangulated internal hernia, external hernia, obstruction due to adhesions, volvulus intussception

The etiological factor for intestinal obstruction is diverse and shows variation from country to country, decade to decade.

AGE INCIDENCE

As per world literature, intestinal obstruction may occur at any age. Its incidence rises in middle age and reaches a plateau in those over 50.Compatively rare in children and young adults.

COMMON CAUSES OF INTESTINAL OBSTRUCTION AT EACH AGE GROUP

Neonate - congenital atresia,volvulous neonatorum, Meconeum ileus, Hirschprung's disease,ano-rectal anamalies.

Infant -strangulated inguinal hernia, intussception, complications Of Meckel's diverticulum, Hirschprung's disease

Young adult -Adhesions and bands,strangulated inguinal hernia.

Middle age - Adhesions and bands, strangulated inguinal hernia, strangulated Femoral hernia, carcinoma of colon.

Elderly - Adhesions and bands, strangulated inguinal hernia, strangulated Femoral hernia, carcinoma of colon, Diverticulitis, Impacted faeces.

SEX INCIDENCE

Acute small bowel obstruction occurs mainly in the 45to 60 age group, that too in male gender.

PATHOPHYSILOGY

Though simple mechanical obstruction, strangulated obstruction and ileus have much in common, there are important differences in pathophysilogy and management. Also, colon obstruction differs in some aspects from small bowel obstruction.

A) SIMPLE MECHANICAL OBSTRUCTION OF THE SMALL INTESTINE

The principle physiological derangements of the mechanically obstructed intestine with intact blood supply are:

i) Accumulation of Fluid and Gas above the point of obstruction

ii) Altered Bowel motility

i) FLIUD AND ELECTROLYTE DISTURBANCES

The bowel immediately above the obstruction is the most affected initially. The ileum above an obstruction ceased to absorb sodium and water. So these substances accumulated in the intestinal lumen and, as time passed, the rates of their secretion increased. Potassium, normally secreted by ileum, was secreted

at an even greater rate after gut had been obstructed. .So accumulation of water, within the bowel in early stage,and K+,Na+,due to retarded absorption .After 48 hours, the water, K+, Na+, get accumulated into the lumen above the obstruction at an increasing rate due to accelerated secretion of those substances. Prostaglandin release in response to bowel distention is thought to be a mechanism by which secretion into obstructed loop is increased..

The ileum below the obstruction showed only moderate changes in its absorptive and secretary capacity. The most striking change was a two-fold increase in K+ secretion into lumen.

Since the fluid lost into the intestinal lumen was isotonic with the body fluids, the concentration of electrolytes in the serum was not altered until later in obstruction.

The fluid is made up by whatever fluids the patient ingests as well as the various digestive juices -about 8000 ml per 24 hours.

Above pylorus 4000 ml	Saliva 1500 ml
	Gastric 2500 ml
Below pylorus 4000 ml	Bile and pancreatic 1000 ml
	Succus entericus 3000 ml

In obstruction, absorption from the gut is retarded but excretion of water and electrolytes especially Sodium, Potassium, Chloride and Bicarbonate, the exact concentration depending on the particular site of Intestinal obstruction.

Approximate Electrolyte content of GI tract

Fluid	**mmo1/1**		
	Sodium	**Potassium**	**Chloride**
Gastric juice	60	10	100
Bile	145	5	100
Pancreatic juice	140	5	75
Small bowel contents	110	5	100

The severity of depletion and speed with which it becomes manifest depends upon the level of obstruction. It is most severe and occurs early in high intestinal obstruction, later in ileal obstruction and is slow to appear in colonic obstruction.

The second route of fluid and electrolyte loss into the wall of the involved bowel, accounting for the boggy edematous appearance of the bowel. Thirdly, some of this fluid exuded from the serosal surface of the bowel, resulting in free peritoneal fluid. Fourthly and most obvious route of fluid and electrolyte loss in by vomiting or Nasogastric tube aspiration after treatment is initiated.

Above said causes rapidly depletes the extra cellular fluid space, leading progressively to haemo concentration, Hypovolemia, Metabolic acidosis, renal insufficiency, shock and death unless treatment is prompt and resolute.

(ii) Intestinal Gas

This is also responsible for distensions of bowel above the obstruction. This consists of swallowed atmosphere air (68%) diffusion from blood into the bowel lumen (22%) and the products of digestion and bacterial activity (10%). The O2 and Co2 (8%) has been absorbed into the blood stream, the resultant mixture is made up of Nitrogen (90%) and Hydrogen Sulphide. The enormous increase in the intestinal gas is mainly due to the market increase in the gut bacteria both anaerobes and aerobic organisms.

(iii) Bowel motility

Initially, the bowel proximal to the obstruction shows increase in the peristaltic activity to over come the obstruction, initiated by stimulated of stretch reflexes. These contractions are account for the severe colicky abdominal pain. Increased peristalsis continues for a period from 48 hours to several days. The more distal the point of obstruction, the longer does it remains vigorous. If the obstruction is not relieved the increasing distension causes peristalsis to become feebler; Finally the peristalsis ceases, and the obstructed intestine become flaccid and paralysed.

The intestine below the point of obstructed exhibits normal peristalsis and absorption from it continues for 2 to 3 hours following the obstruction, until the residue of its contents has been passed onwards. Then the distal empty intestine becomes immobile, contracted and pale.

B) STRANGULATED OBSTRUCTION

Occlusion of blood supply to a segment of bowel in addition to obstruction of the lumen is usually referred to as **strangulated obstruction.** The first effect of strangulation is to become blue and congested. When the venous return is completely occluded, the colour of the intestine turns from purple to black. There is marked increase in the capillary pressure results in escape of intravascular fluid and red blood cells into the bowel wall, its lumen and the

hernial sac or peritoneal cavity. About this time, owing to increased edema at the point of obstruction, the arterial supply is jeopardized.. The peritoneal coat loses its glistening appearance, mucous membrane becomes ulcerated and Gangrene is imminent. Large amount of blood get sequestered in the strangulated segment, which is proportional to the length of the segment.

Unlike non-strangulating obstruction, early distension of the proximal intestine is absent. For a time varying from a few minutes to several hours, vigorous peristalsis continues in the proximal segment but is still unaccompanied by distension. By the time gangrene of the strangulated segment is imminent, retrograde thrombosis is proceeding along the along the related tributaries of the mesenteric vein. Distension then appears both on the proximal and distal sides of strangulation. The greatest distension occurring when the venous return is completely obstructed while the arterial supply remains unimpaired.

When the wall of the intestine becomes partly devitalized, both bacterial toxins and products of tissue autolysis pass into the peritoneal cavity there to be absorbed into the circulation. This is followed by the migration of bacteria and peritonitis follows. Delay in the recognition and treatment of intestinal strangulation significantly enhances the mortality.

BACTERIOLOGY

The normal upper small intestinal contents are virtually sterile. The distal small gut fluid may yield a scantly growth of faecal flora. The situation is quite different in the presence of obstruction. The bowel above the level of obstruction contains profuse bacterial colonies, predominantly faecal in type (Both aerobic an anaerobic) and increase in the anaerobic organisms especially **Bacteriodes.** Experimental studies demonstrate that **Clostridium perfringens** exotoxins contribute to the lethal activity of filter-sterilized strangulation fluids but direct clinical evidence is lacking. The longer the period of obstruction, the higher up the bowel this contamination extended.

The major threat to life in intestinal obstruction is the possible absorption of toxins, mainly from Gram Negative organisms in the presence of damaged bowel, particularly when strangulation is present. In unrelieved strangulation, toxic substances appear in the peritoneal cavity and absorption in the systemic circulation takes place. However, when obstruction is relieved, these toxins may pass on to the normal bowel where rapid absorption can occur. This factor stresses the need for intestinal decompression before and during operation, early surgical correction, and for adequate prophylactic antibiotic cover against Gram Negative organisms.

C.CLOSED -LOOP OBSTRUCTION

When both afferent and efferent limbs of a loop of bowel are obstructed, closed -loop intestinal obstruction exists. This is clinically dangerous form of obstruction because of the propensity for rapid progression to strangulation of the blood supply before the usual manifestation of intestinal obstruction become obvious.

In its typical form it is seen in carcinomatous stricture of the colon. Distally the colon is occluded by the neoplasm, while in one -third of cases the lieo-caecal valve prevents regurgitation of the contents of the large intestine into the ileum, and consequently that part of colon proximal to the neoplasm is closed at both ends. As a result of anti peristalsis the pressure within the caecum becomes so high as to compress the blood vessels within its wall. If the obstruction is unrelieved, stercoral ulceration gangrene and **Pistol -shot** perforation of caecum will eventually occur.

CLINICAL FEATURES AND DIAGNOSIS

There are **Four** common complaints of patients with Acute obstruction:-

1. Pain
2. Vomiting
3. Constiopation/obstipation
4. Abdominal distension

There may all exists in a particular case (or) occur in any combination.

1. PAIN

Pain is the first and most common symptom. The onset may be insidious or abrupt. The pain is colicky and each attack last from 3 to 5 minutes. If the obstruction lies in the jejunum or high in the ileum the characteristic attacks of intestinal colic come on at interval of from 3 to 5 minutes. In obstruction to the terminal ileum or the large intestine, the interval of freedom tends to be longer-from 6 to 10 minutes.

2. VOMITING

Usually occurs almost immediately after obstruction of bowel. This early vomiting is **reflex vomiting** and is followed by variable quiescent period before vomiting resumes. Then quiet interval is short in high obstruction and vomiting is more frequent and copious leads more rapid reduction in extra cellular fluid volume. With low small bowel obstruction,

vomiting is less frequent and less productive so patient with low obstruction may look well even when he has quite marked physical signs in the abdomen.

As low obstruction progresses, the vomit begins to assume faeculant character. In closed loop obstruction, vomiting and small bowel distension may never occur. Reflex vomiting is unusual in colon obstruction. Vomiting results only after retrograde distension of small bowel occur due to incompetent ileo-caecal valve.

3. **CONSTIPATION/OBSTIPATION**

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 (absolute constipation). This rule does not apply in cases of Richter's hernia. Gall stone obstruction, Mesenteric vascular occlusion, and intestinal obstruction associated with a pelvic abscess, acute intussusception and there may be diarrhoea of various types.

4. ABDOMINAL DISTENSION

In early cases of obstruction, abdominal distension is often slight or even absent. When the proximal jejunum is occluded, the stomach becomes distended and so epigastric region may 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 the abdomen with well marked bulging in the flanks. Volvulus of the sigmoid may be accompanied by distension of stupendous proportions.

CLINICAL SIGNS

There is little in the usual clinical findings that help in the early case. The presence of an abdominal scar, whether recent or old, always suggests an underlying band or adhesions. In the early stage, the vital parameters are normal. At a late state, the patient becomes anxious and pale, with a feeble rapid pulse, falling temperature and blood pressure, and typical feature of dehydration-a dry skin, dry tongue, sunken eyes may be present. Shock may be more marked in the strangulated case.

PALPATION

Usually reveals tenderness and release tenderness. This, together with muscle guarding, tends to be more marked in the strangulated case. A mass may be detected on palpation, such as a carcinoma of the colon, diverticulitis of the sigmoid or an intussusception. The hernial orifice should be methodically palpated and an rectal examination should be performed. Typically, in an intestinal obstruction the rectum is ballooned. Occasionally a low-lying obstructive tumour or an impacted mass of faeces may be found or a pelvic tumour may be palpable through the rectal mucosa or there may be telltale blood or slime on the examining finger.

BORBORYGMI

Synchronous with the colic, it is often possible to hear borborygmi with the aid of a stethoscope, establishes the diagnosis in nine out of ten cases. The clinician should listen for at least a minute. Obstruction is indicated by high-pitched Splashing, rushing or tinkling sounds lasting at least a second and having a characteristic gurgling quality.

DIFFERENTIAL DIAGNOSIS

Simple mechanical obstruction has to be distinguished from the following causes of acute abdomen which include:-

1. Acute Gastro enteritis
2. Pancreatitis
3. Appendicitis
4. Perforated peptic ulcer
5. Renal or Biliary colic
6. Torsion of ovarian cyst
7. Medical causes e.g. Diabetic coma.

DIAGNOSTIC STUDIES

1) Laboratory Tests:- The loss of large amounts of essentially isotonic extra cellular fluid into the intestine is principally responsible for the laboratory findings in simple mechanical obstruction.

a)	Urine	-	Mild proteinuria or acetonuria
		-	Specific gravity 1.025 to 1.030
		-	Reduced urine output
b)	Hb%	-	Elevated due to haemo concentration
	PVC	-	Elevated due to hameo concentration
c)	WBC court	-	increased count of 15.000/mm3-in case of simple mechanical obstruction. 15,000 to 25,000/mm3 increased neutrophils in strangulated many immature cells obstruction.
d)	Blood urea	-	increased
e)	Serum Na+, K+, Cl-, Hco3-lowered		
f)	Serum Amylase		increased

2) RADIOLOGICAL FINDINGS

As we take plain x-ray abdomen as only the radiological investigation to diagnose the intestinal obstruction, prime importance has been given to this and dealt with in detail. It is always preferable for patients to be x-rayed in the radiological department and not in bed. Erect films diagnose obstruction by showing multiple fluid levels, particularly if these are stepladder. Supine films allow the nature of the distended bowel to be assed.

Gas shadows

When the jejunum, the ileum or the colon is distended with gas, each has a characteristic appearance. Obstruction of the small intestine is revealed by relatively straight loops that generally lie more or less transversely in step-ladder fashion. Obstructed large intestine is disclosed by its haustration markings; a distended caecum is shown by a rounded gas-shadow.

Jejunum is characterised by its valvulae conniventes that passes from the anti mesenteric to the mesenteric border, spaced regularly, giving rise to a **concertina** effect. **The distal ileum has been rightly described by wangensteen as characterless Large Intestine** Shows has haustral folds, which are spaced irregularly, not traverse the complete width of the bowel, and the indentation are not placed opposite one another.

FLUID LEVELS

In infants under the age of 2 years a few fluid levels in the small intestine are of normal occurrence. In the adult, three fluid levels are sometimes seen and must be regarded as physiological. The first is in the gastric fundus. The second is at the duodenal cap. The third-a fluid level in the terminal ileum-is rare.

In intestinal obstruction it takes a little time for the gas to separate from the fluid; consequently, fluid levels appear later than gas shadows. When paralysis of the intestine has occurred, fluid levels are become more conspicuous and more numerous. The number of fluid levels is proportionate to degree of obstruction and to its site in the small intestine. Differential Air-fluid level in the same loop of 20mm or greater was moderately suggestive that a bowel obstruction was mechanical in nature (Harlow et al)

GENERAL OUTLINE OF TREATMENT

There are four measures for combating and over coming the effect of intestinal obstruction. They are

1. **1.Naso gastric aspiration** through Ryle's Tube - used to decompress the distended bowel by aspirating the fluid as well as swallowed air.

2. Replacement of fluid and electrolytes

If acid gastric juice loss is prominent, then normal saline solution is used. Otherwise lactated Ringer's solution and 5% Dextrose in water in about equal proportions are preferred to replace the lost fluid. Potassium chloride also will be necessary but should not be given until a good urine out put is established.

3. **Antibiotics** to Prevent complications from associated sepsis, either locally (peritonitis) or peripherally (chest complications) or generally (septicemia).

4. Relief of obstruction by surgery

The surgical procedures for the relief of intestinal obstruction may be divide into seven categories (Goar et al,Chaib et al).

a) Exploratory laparotomy for obstructions if uncertain origin.

b) External drainage of the intestine proximal to the obstruction-enterostomy, caecotomy, colostomy.

c) Short-circuiting anastomosis around an obstruction eg. Entero anastomisis or ileo colic anastomosis.

d) Resection of bowel either to remove the obstructing lesion eg. A carcinoma of the colon or because a strangulated segment of bowel has undergone irreversible ischaemic change.

e) Lysis of bands or adhesion.

f) Planned operations for specific obstructive lesions eg. A strangulated external hernia or laparotomy for Intussusception in a child.

g)Enterotomy for removal of obstruction-Gall stones, Bezoars.

MATERIALS AND METHODS

A total of 109 cases of acute intestinal obstruction admitted in all surgical wards, THANJAVUR MEDICAL COLLEGE HOSPITAL, THANJAVUR from November 2004 to MARCH 2006. Both men and women more than 12 years of age totaling 109 cases with features of acute intestinal obstruction were chosen. The paediatric patients were not included in this study.

The characteristic clinical features of acute intestinal obstruction like abdominal pain, vomiting, constipation/obstipation, abdominal distension and clinical sings including Fever (37.2^{o}). Tachycardia (>100/mt), Palpable abdominal mass if any were noted.

All the patients were subjected to investigation while resuscitative measures were on progress.

1. Urine-albumin, sugar
2. Hb%
3. Blood Grouping with typing
4. WBC count
5. Blood-urea, sugar
6. Serum Electrolytes-in cases with severe dehydration.
7. X-ray chest PA view
8. ECG
9. Plain X-ray abdomen erect and supine position. and result were recoded.

Investigations 4 and 9 were taken as a tool to identify the pathology while others were meant for assessing the patient's fitness for surgery.

After adequate preoperative preparation all patients were subjected to surgical Procedures appropriate to the condition and preoperative pathology were noted.

The etiological incidence, Sex incidence, Age incidence, incidence of Strangulation, value of plain X-ray Abdomen in diagnosis of acute intestinal obstruction, importance of early treatment were studied.

STRANGULATION OBSTRUCTION

Definition

Occlusion of the blood supply to a segment of bowel in addition to obstruction of the lumen is usually referred to as **strangulated obstruction**. Interference with the mesenteric blood supply is the most serious complication of intestinal obstruction.

Etiology: This is frequently occurs secondary to

a) External hernias like Inguinal, Femoral, Paraumbilical, and Incisional hernias.

b) Intra abdominal hernias.

c) Adhesive band obstruction.

d) Volvulus of -Sigmoid colon, Small bowel, Caecum etc.

DIFFERENTIATION BETWEEN SIMPLE AND STRANGULATED OBSTRUCTION:-

a. A sudden onset: The symptoms nearly always begin suddenly and

b. Shock: Occurs early in severe strangulating obstruction.

c. Pain: In both strangulating and non-strangulating obstruction this is typically colicky but it become continuous and sometimes unrelenting.

d. Elevation of **Temperature (37.2^{0} C)** and **Tachycardia (100/mt)**

e. Increase in WBC count in the range of **15,00** -25,00/mm3 with polymyorphonuclear predominance with many immature forms strongly suggest the obstruction is strangulated but this is not a sensitive indicator.

f. Metabolic acidosis-is present after 12 hours in at least half of the patients with strangulation.

g. In strangulation there is always tenderness over and intra-abdominal strangulated coil and the **Rebound tenderness** is distinctive sign of strangulation.

h. Presence of an **Abdominal of Pelvic swelling** which is tense and tender in 10% of strangulating obstruction.

The clinical parameters, like **Continues pain, Fever (>37.2^{0}C)**, **Tacchycardia (> 100mt), Leukocytosis (15,00-25,00 cells /mm3), Palpable abdominal mass,** enable us to detect the presence of strangulation in about **38%** of cases that too mainly in external hernias. **Shatila** and **Chamberalin, sarr et al** in a large series highlighted the failure of these clinical parameters of differentiate between simple and strangulated obstruction.

Despite the advances made in the diagnosis and management of intestinal obstruction, its mortality rate is still high in case of strangulated obstruction. Cohn has shown that bacteria and bacterial toxins play a role in causing mortality. For this reason adequate dose of antibiotics is indicated.

MORTALITY IN CASE OF STRANGULATED OBSTRUCTION CAN BE REDUCED BY

a) Adopting immediate resuscitative measures to combate dehydration, electrolyte loss and blood transfusion.

b) Preoperative and preoperative nasogastric/gastro intestinal suction and antibiotics.

c) Early operation.

d) Resection of dead segment of the bowel and adequate lavage of peritoneal cavity with normal saline with or without antibiotics so as to remove and dilute the toxic intra peritoneal fluid.

OPERATIVE FINDINGS IN STANGULATED OBSTRUCTION

On opening the abdomen, blood stained fluid present in the peritoneal cavity, the fluid should be removed by suction or mopped up as completely as possible, for it is toxic and infected. After the relief of strangulation a decision must be reached as to whether the segment that was strangulated is viable. When it is black and the peritoneal coat has lost its sheen, when the mesentery shows a lack of arterial pulsation, or there is thrombosis of its veins, it is non-viable, if not already gangrenous, and if practicable, resection follows by anastomosis is carried out. In doubtful cases when the intestine is blue, purple or dark red, the effect of wrapping it is a warm moist abdominal pack is noted. At the same time the anesthetist administers pure oxygen for 3 minutes. By these means viable is differentiated from non-viable intestine. -- as shown in the table.

Special attention should be paid to the sites of pressure rings at each end of the segment, which it of doubtful viability, should be infolded by seromuscular sutures and covering the area with omentum. When strangulated intestine is deemed non-viable, It is resected and the continuity of the alimentary canal restored by end-to-end anastomosis.

SMALL BOWEL OBSTRUCTION

In this series the following are major causes of small bowel obstruction.

A. External hernia

B. Adhesion and bands

C. Tuberculous Adhesion

D. Small bowel volvulus

E. Meckel's Diverticulum

F. Intussusception

G. Ileo-Sigmoid Knotting

H. Miscellaneous

Secondary Deposits

Small bowel Tumours

OBSTRUCTION CAUSED BY EXTERNAL HERNIAS

Definition :

A hernia is the protrusion of a viscus or part of a viscus through an abnormal opening in the walls of its containing cavity. Most common External hernias are Inguinal, Femoral, paraUmbilical.

In this study External hernias contributes to **44.30% (48 cases)** of total occurrence. Among External hernias, **79.16% (38 cases)** is contributed by inguinal hernia and remaining by other hernias. Males are predominantly affected especially 3rd to 6th decade of life.

ETIOLOGY : Three factors are involved in precipitation of hernia.

1. **Presence of pre formed sac:** A patent processes vaginalis is held to be prime cause of indirect inguinal hernia in infants, children and probably in adults.
2. **Repeated elevations in the intra abdominal pressure:** Occur in
   - **(a)** Chronic cough
   - **(b)** Straining and defecation (constipation)

(c) Straining on micturation (BPH, stricture urethra)

(d) Pregnancy

(e) Ascites

(f) Intra Abdominal malignancy

3. Weakening of the body muscles and tissues

Occur in old Age and obesity

Pathology:

The hernia either reduces itself when the patient lies down or can be reduced by the patient with expansible impulse on coughing is the common presentation.

It became **irreducible** when the contents cannot be returned to the abdomen and there is no evidence of other complications. It is due to adhesions between the sac and its contents or from over crowding within the sac. Any degree of irreducibility predisposes to strangulation.

An irreducible hernia-containing intestine, which is obstructed from without or within, but there is no interference to the blood supply to the bowel, with absence of cough impulse, become **obstructed Hernia.** When the blood supply of contents of obstructed hernia impaired, **Strangulated Hernia** results. Pathological features of strangulated hernia are as that of strangulating obstruction already discussed.

The constricting agent causing obstruction and strangulation of the contents are

In **Indirect Inguinal Hernia**

1. Neck of the sac
2. The external abdominal ring in children
3. Adhesions within the sac-rarely

In **Femoral Hernia**

1. Narrow, unyielding femoral ring.
2. Narrow neck of the sec
3. Gimbernat's 9lacunar) ligament.

In **Parambilical Hernia**

1. Narrow neck
2. Fibrous edge of the linea Alba.

Structures commonly involved in strangulations are

1. Small intestine-commonest

2. Omentum

3. Portion of a circumference of small intestine (Richter's hernia)

4. Large intestine-rarely involved.

Clinical features

Sudden pain, at first situated over the hernia, is followed by generalized abdominal pain, paroxysmal in character and often located mainly at the umbilicus. Vomiting is forcible and usually often repeated. The patient may say that the hernia has recently become larger.

On examination, the hernia is **tense,** extremely **tender, and irreducible** and there is **no expansible impulse** on coughing.

Treatment

Vigorous manipulation (**taxis)** has no place in modern surgery. Its dangers include:-

1. Contusion or rupture of the intestinal wall.
2. Reduction -en -masse
3. Return of already devitalized bowel.
4. Reduction into a loculus of the sac.
5. Sac may rupture and its contents are reduced extra peritoneally.

In obstructed/Strangulated hernia **operative treatment is mandatory.** Strangulated omentum must always be resected. Strangulated small bowel resected and end-to-end anastomosis is carried out.

For Indirect Inguinal hernia with liberal Inguinal incision skin, subcutaneous on tissue incised and deliver the body and fundus of the sac together with its covering and the testis (in male) onto the surface. Incise the sac, the fluid there in is mopped up or aspirated. Then the external oblique and superficial inguinal ring divided.

A finger passed into the opening made in sac, and employing the finger as a guide, the sac is slit along its length (this will divide two constriction lies ate external ring or in the Inguinal canal).

When the constricting agent is at the deep ring it may possible to continue slitting up the sac over the finger beyond the point of constricting or a grooved director is inserted and the neck of the sac is divided with a knife in an upward and inward direction under vision. Now draw down the content and examine and dealt accordingly. After transfixing the sac, repair the hernia.

In case of Femoral hernia

Modified **Lotheissen** procedure is operation of choice with inguinal incision, skin, subcutaneous tissue divided. As soon as external oblique has been exposed, the inferior margin of the wound is retracted strongly, there by displaying the swelling. The covering of sac are incised and peeled off. Sac incised and drain out the fluid. Open the Inguinal canal. The transversal's fascia is incised to the

medical side of the epigastric vessels and the opening is enlarged. Once open the peritoneum above the inguinal ligament, we can inspect the content of sac. Should the obstruction lie in a narrow neck of sac, the beak of a hemostat is insinuated and neck is stretched (may injure abnormal obturator artery). The contents of the sac are delivered and dealt accordingly and repair the hernia.

OBSTRUCTION CAUSED BY ADHESIONS AND BANDS

Adhesions or solitary bands are the most common cause of intestinal obstruction in the western world. **Mucha** (1987) of Mayo clinic, **Mc Entee** et al of United Kingdom showed that adhesions and bands are important prime cause for intestinal obstruction which accounts for 49%, 32% in their studies respectively, In our study adhesion contributes to 39.44% (4**cases)** and stands second, of which strangulation occurred in 25**% (12 cases).** Males are commonly affected. About 2 cases were due to post-surgical adhesion

Etiology

The causes of intra peritoneal adhesions are shown below:-

1.	Ischemic areas	-Sites of anastomosis Reperitonealization of raw areas
2.	Foreign bodies	-Talc, starch granules, gauze lint, cellulose, non-absorbable sutures.
3.	Infective disease	-Peritonitis -Tuberculosis
4.	Inflammatory disease	-Crohn's disease
5.	Radiation enteritis	
6.	Sclerosing Peritonitis	-usually drug induced (eg.Practolol)

The number **1** and **2** constitute post-surgical (operative) adhesion and it is the most common category. Appendicectomy, Gynecological surgery and surgery of large bowel were the most common preceding surgical procedure. Postoperative adhesion giving rise to Intestinal obstruction usually involve the lower ileum.

Pathology

Following abdominal surgery, fibrinous adhesions always from between the intestinal loops. In the majority of cases, this fibrinous material becomes

organized with the in growth of both fibroblasts and capillaries to form adhesions. Adhesions are best regarded as an attempt by

the line of a bowel anastomosis or to a laparotomy scar as a result of the strangulating effects of sutures.

The earlier belief in maintaining the intactness of peritoneal endothelium to prevent fibrinous adhesions is replaced by the fact that large peritoneal defects, which are left open and bleeding, heal within a few days into a smooth glistening new serosa. However if the injury is accompanied by vascular damage (if the tissue are crushed, sutured or lighted) then adhesions develop.

The peritoneum reacts in a similar manner to foreign material as it does to ischaemic tissue. Therefore, granulomas and adhesions may result from fragments of gauze, unabsorbed suture material or talc or starch glove powder introduced at the time of laparotomy.

Clinical features :

Obstruction may present in early post-operative period or may occur at any time after abdominal surgery or an intra abdominal inflammatory episode. When symptoms arise shortly after abdominal operation, it has to differentiated from Post-operative ileus.

The patient with late adhesive obstruction may give a history of number of previous episodes of subacute obstruction. The clinical features are those of classic low small gut obstruction with severe central colicky abdominal pain, nausea, and vomiting and abdominal distension. Examination reveals central distension of the abdomen, nearly always with an abdominal with an abdominal scar from previous surgery, with rebound tenderness, increased bowel sounds. Plain X-ray abdomen usually demonstrates multiple fluid levels.

Treatment:

1. **Non operative Treatment** with Gastro intestinal suction drainage combined with intravenous fluid therapy is extremely beneficial for all type of adhesion and occasionally it is curative in those cases develop obstruction within 3rd and 6th postoperative period. Patient who will respond to tube decompression of the GIT usually do so in the first 48 hours (Bizer et al, Brolin et al. Peetz et al, Wolfson et al, Cox et al.) There is no convincing evidence that long intestinal tubes are more efficacious than Nasogastric tube in the decompression of small bowel obstruction.

2. Adhesiolysis

The abdomen opened through previous wound after excising the scar. Although many adhesions are often present, frequently only one of those found to be cause of obstruction, which was divided near the insertion where it is least vascular. At other times the insertine is angulated by adherence to the parietes, to the mesentery, to the nearby structures. In these circumstances it is sometimes possible to free the obstructed intestine by dissection. In order to prevent recurrence the bare areas should be covered with omental grafts.

3. In cases of Recurrent wide spread adhesions

(a) Noble's plication operation

(b) Child's -Philip's transmesenteric plication was recommended.

Prevention of Adhesion formation

The following precautions are aimed at minimizing adhesion formation after abdominal surgery:

1. Meticulous surgical Technique

2. Washing the peritoneal cavity with **saline or Dextran (Fabri et al)** solutions at the end of the procedure to remove clots and implanted foreign bodies (glove power, suture material).
3. Avoidance of excessive packing with gauze.
4. Covering anastomosis and raw peritoneal surface with the greater omentum.
5. Leaving raw peritoneal areas unstitched. Numerous substances have been instilled into the peritoneal cavity in the hope of preventing adhesion formation. Hyaluronidases, Hydrocortisone, Silicone, Fibrinolysin, Dextran, Poly vinylpyrolidine (PVP), Sreptomycin solutions have been tried. No single substance has been demonstrated to be completely effective and safe in clinical use.

OBSTRUCTION BY CONGENITAL BAND

A band (usually one band only is culpable) is occasionally the cause of acute obstruction.

Such a band may be,

1. Congenital-often an obliterated vitello intestinal duct.
2. A string -like band, frequently thin and fragile, following previous bacterial peritonitis.

3. A portion of greater omentum, adherent usually to the parietes, constitutes obstruction band.

Treatment:

1. If involved intestine viable, division of the band and release of the obstructed loop is the simplest surgery.
2. After releasing the band the entrapped intestine regains its colour usually. If the involved bowel is found to be gangrenous, resection and end-to-end anastomosis is the choice.

If the constriction sites that have suffered direct compression by the band show any residual colour changes, they should be invaginated.

OBSTRUCTION CAUSED BY ABDOMINAL TUBERCULOSIS

Intestinal tuberculosis is a common disease prevailing in our country. This study includes only those cases of abdominal tuberculosis, which presented as acute intestinal tuberculosis and account for 6% (**6 cases)** in total Main source of infection is swallowed sputum.

Pathology

Commonly the **Fibrous (Plastic)** form present as acute intestinal obstruction characterized by production of widespread fibrous adhesion.

In the acute phase the bowel in the ileo-caecal is red, edematous and friable with visible tubercles on the serosa. In the more chronic phase the wall of the intestine is thickened due to granulomatous infiltrates and fibrosis and forms a mass in the right iliac fossa. On naked eye examination, the lesion may be indistinguishable

from Crohn's disease of the Terminal ileum and caecum. Histologically there is little difference between the lesions in the small intestine and Hyperplasic mass found in the ileo-caeal region. The mucosal ulcers do not usually penetrate the muscularis mucosa, but deep to the layers there are masses of granulomatous follicles often with central caseation and lymphocytic infiltration. The Granulomata are scattered through all the layers of the intestine from the mucosa to the serosa. Unlike Crohn's disease, abscess and fistula formation are uncommon in tuberculosis. Sometimes there may be combination of tuberculosis peritonitis with stenotic lesions in the small intestine. In tuberculous enteritis the typical granulomatous lesion leads to the formation multiple stricture and intestinal obstruction.

Clinical features

Most common presentation of abdominal tuberculosis in our country is acute or chronic obstruction.

Abdominal pain (90%) is the commonest symptom in both the obstructive and non-obstructive group (**Das et al)** Vomiting is also a frequent symptom in obstructive group. Although perforation of the small intestine is uncommon, it may occur in a dilated loop of ileum proximal to a stenotic tuberculous lesion causing obstruction. Anorexia, loss of weight with abdominal distension also present.

Distended coils acts as **blind loop** and give rise to steatorrhoea. In the chronic intestinal obstruction there are usually symptoms of increasing constipation and loss of weight. The obstruction may be due to stricture of small intestine either single or multiple.

Investigations:

A Plain X-ray abdomen erect may show multiple fluid levels with or Without Ascites. There may be diffuse calcification or evidence of localized abscess.

Treatment

1. If a tuberculous abdomen is opened due to a mistaken diagnosis unless there is any mechanical obstruction present, only an omental or Lymph node biopsy must be taken and sent for Histopathological examination Exploration of abdomen should be followed by a course of antituberculous drugs viz. **Rifamycin, INH, Ethambutol, pyrazinamide.**
2. Division of band or adhesiolysis is sufficient if band or adhesions only causing obstruction and followed by a course of antituberculous drug.
3. For ileo-caecal lesion, a limited resection of terminal ileum and caecum is adequate. A formal Right Hemicolectomy is not required (Pujari). A bypass ileotransverse anatomisis should be avoided for fear of creating blind loop.
4. If a singly stricture is found as culprit, Stricturoplasty is sufficient (Joshi)
5. If adhesions are accompanied by multiple fibrous strictures of the ileum are situated close to each other, it is best to excise the affected bowel only (limited resection).

OBSTRUCTION CAUSED BY VOLVULUS OF SMALL INTESTINE

Volvulus of small intestine account for about 20% of all cases of intestinal obstruction (excluding hernia) that have been reported in (Agarwal and Misra) in literature. In this study 2 cases were reported (2%) all of them required resection.

Predisposing factor

Small intestine volvulus often follows a previous pathological lesion such as

a. Congenital anomalies like Mekel's diverticulum
b. Local peritonitis
c. Tuberculous mesentenric-lymphadenitis
d. An abdominal operation

These favours formation of adhesion passing from the antimesenteric border of an intestinal loop to the parietes.

In Africa, consumption of a large meal of maize and vegetables seems to predispose this condition.

Pathology: The loops of intestine generally twist in a clockwise direction. The lower quarter of ileum is most commonly involved, although at times a large part

of small gut (or) whole of the jejunum and ileum and its mesentery may undergo rotation.

Clinical features:

The signs and symptoms are more of small gut obstruction with strangulation. The distended coils of small intestine may be felt through the abdominal wall.

Investigations:

Plain X-ray abdomen erect shows multiple fluid levels and gas shadow pertaining to the small bowel obstruction.

Treatment

1. Laparotomy and untwist the bowel if it is found to be viable. Divide the causative band

2. If Gangrene of bowel present and the mesentery cannot be untwisted, resection and end-to-end anastomosis should be done to maintain the intestinal continuity.

MECKEL'S DIVERTICULAM

It is one of the rare causes for acute intestinal obstruction. In this series 2 cases were reported contributing 1.83% and all of them were males.

It is present in 2% of human race, situated upon the anti mesenteric border of small intestine 2 feet from the Ileo caecal valve and it is usually 2 inch long. Being congenital diverticulum, it poses all three coats of the intestinal wall.

The presence of a band between the apex of the diverticulum and the umbilicus may cause obstruction either by band itself or by a volvulus around it or obstruction with in a hernia.

CLINICAL FEATURES

In most cases a Meckel's diverticulum is an incidential finding and usually asymptomatic. All features of Intestinal obstruction occurs.

X-ray shows features of small bowel obstruction but fails to identify the cause.

TREATMENT

1. In Non Gangrenous obstruction – Meckelian Diverticulectomy is the choice (Volko ve al).

2. In Gangrenous obstruction resection of a segment of the ileum containing the diverticulum followed by end to end anastomosis.

OBSTRUCTION CAUSED BY INTUSSUSCEPTION

In intussusception can occur at any age. In the adult, Intussusceptions is comparatively rare, accounting for about 5 present of all obstruction. It is Common in some African communities.

In this study 4 cases were reported (3.27%) of which 3 being Ileo-colic type, one being Ileo-ileal type.

ETIOLOGY

This is usually a tumour forming the apex of the intussuception which tend to be a begin lesion in small bowel intussuceptions and a malignant tumour in intussusceptions of the colon.

Submucous lipoma is the most frequent benign lesion causing intussusception. It also due to polypus, papilliferous growth, Meckel's diverticulum's, Leiomyoma etc.

Clinical manifestations

Usually dominated by the general signs and symptoms of intestinal obstruction. Presence of lump and passage of blood per rectum often suggest the diagnosis. During the height of an attack, an obvious mass may be present but may disappear completely when the patient reexamined just a few hours later.

RADIOLOGICAL EXAMINATION

Plain X-ray abdomen - shows dilated loops of small bowel with Multiple fluid levels

Barium enema - used to diagnosis an intussuscepting large Bowel tumour. Indeed, this may produce at least temporary reduction of intussusceptions

CT scan: extremely useful to detect the involved segment and a mass lesion intussusceptions Three concentric circles that form as one segment of bowel invaginates into another an Central circle by entering layer of intussusceptions, and 2nd circle by entrapped mesentery and 3rd circle represents the intussusceptions (IKO et al).

Treatment of adult intussusceptions is invariably surgical.

Large bowel intussusception risk of malignancy as the cause is high and so proceed resection without any attempt at reduction.

INTUSSUSCEPTION OF SMALL INTESTINE

Reduction should be attempted. If bowel is Gangrenous-resection and end-to-end anastomosis must be carried out. In idiopathic case, nothing further than reduction need be performed. Benign lesion causing intussusception-enterotomy and removal of lesion (polypus)

ILEO SIGMOID KNOTTING

A loop if ileum wraps around the base of and elongated sigmoid colon or vice viz. It is a variant of midget volvulus. Also called as compound volvulus, double volvulus, valvulus associate; intestinal knot syndrome or ileo-sigmoid interwining results in two-closed loop obstruction. In this study 2 cases were reported contributing 1.63%.More common in Eastern India.

ETIOLOGY:

May be due to genetic, dietary, and habitual. In South India labour class people drink large quantities of GANJI, a preparation containing jawar, buttermilk and water may predispose. An abnormally long colon with a lean and lengthy ileal mesentery.

CLINICAL FEATURES:

Dramatic on set of obstructive symptoms with rapid deterioration of general condition.

INVESTIGATIONS :

Plan x-ray abdomen

i) Shows distension of both ileal loops and sigmoid colon.

ii) Disproportionately dilated loops of the bowel with their limbs directed downwards into their lower quadrants.

iii) Small bowel air fluids levels.

iv) Unintended but faecally loaded bowel proximal to the pelvic colon.

v) Medial deviation of the descending colon.

Immediate surgery is the choice to prevent mortality from gangrene ileum 80% and sigmoid 50%.

MISCELLANEOUS

In this study one case of secondaries in the omentum adherent and infiltrating the ileum causing the obstruction and one case of small bowel tumour, contributing 0.91% in small bowel obstruction.

Secondaries Causing intestinal obstruction

Patient harboring metastatic intraperitioneal carcinoma who presents with bowel obstruction provides a unique challenge. This event will merely herald the terminal phase. One third of this patient harbors an adhesive rather than malignant obstruction. Breast, renal cell carcinoma and melanoma often give solitary metastatic deposits within the peritoneal cavity leading to bowel obstruction.

Carcinomatosis presents much greater problems, since there are multiple sites of peritoneal obstruction that are not amenable to either resection or bypass. So tube gastrostomy is worthwhile.

SMALL BOWEL TUMOURS:

Rare, both benign and malignant types collectively account for less than 10% of all Gastro Intestinal Tract neoplasms.

Etiology-unknown

Risk factors. Chron's disease, celiac disease, dermatitis herpetiformis, PeutzJeghbers syndrome, radiation enteritis and adenomas.

BENIGN:

60% of small bowel neoplasm.

Mostly asymptomatic and are incidental findings.

Types:

Epithelial tumours, tubular and villous adenomas, lipomas haemangiomas and neurogenic tumours. May occur in association with any of the various types of familial polyposis..

Gardener's syndrome: Familial polyposis and epidermoid cysts.

Turcot's syndrome: Familial polyposis and brain tumours.

Villous tumour is prone for malignant change.

Commonest presentation is intestinal obstruction due to intussusception, iron deficiency anaemia and Gastro Intestinal bleedings.

MALIGNANT :

Rare, less than 5% of all Gastro Intestinal neoplasms.

Types: **Adenocarcinoma-40%**

Carcinoid tumours-30%

Lymphoma-25%

Smooth muscle tumour-5%

It occurs in patients with hereditary polypoid syndromes, familial polyposis peutz-Jehgher's syndrome, and chron’s disease.The sequence is hamartoma-adenoma-displasia-carcinoma. Adeno carcinomas are well-differentiated mucus secreting tumours.

INCIDENCE:

Duodenum040% Jejunum-40% IIeum-20%

CLINICAL FEATURES:

Age-over 40 to 50 years Sex-equal distribution.

Epigastric or periumbilical discomfort or pain.

Postprandial colicky pain, nausea, vomiting, weight loss and Gastro Intestinal bleeding.

Anaemia, guaiac positive stools and jaundice in periambullary lesion. Intestinal obstruction is an advanced stage.

Thos tumour does not response to chemotherapy or radiotherapy surgical resection is the choice.

TREATMENT

INGUINAL HERNIA:

All the obstructed inguinal hernias were operated surgically. The obstruction was relieved and the contents were inspected and herniorrahphy done. The postoperative period was uneventful.5 cases were Infected and secondary suturing done.

For strangulated inguinal hernia in 5 cases, resection and anastomosis of the non-viable intestine was done and end-to-end anastomosis was done.

FEMORAL HERNIA:

For the Femoral hernia with intestinal obstruction in 3 cases, the contents were inspected and the guts were viable and reduced the contents and followed by raphy was done by Modified **Lotheissen** procedure.

PARAUMBLICAL HERNIA:

For the five paraumblical hernias with simple obstruction, MAYO'S REPAIR done.Vaccum drainage was kept for 2 days. Their postoperative period was uneventful.

INCISIONAL HERNIA:

All the incisional hernia patients were with simple obstruction and underwent repair and treated with intravenous fluids and antibiotics. Their post operative period was uneventful.

POSTINFLAMMATORY:

Post surgical cases with intestinal obstruction were operated. In all the cases who underwent laprotomy with simple adhesive obstruction, simple adhesiolysis was done. All were intensively treated with appropriate antibiotics and with intravenous fluids.

CONGENITAL BANDS:

Laprotomy was done and simple adhesiolysis was done. The postoperative period was uneventful.

TUBERCULOSIS ABDOMEN:

Laprotomy was done and found to have simple obstruction. So biopsy was taken from omentum, lymph nodes and sent for Histopathological examination. Subsequently they were put on anti tuberculosis drugs.viz. Rifamycin, I.N.H., Ethambutol and pyrazinamide.All the cases were successfully treated.

SMALL BOWEL VOLVULUS:

The nonviable bowel was resected followed by end to end anastomosis.

MECKEL'S DEVERTICULUM:

Meckelion Diverticulectomy was done. End to end anastomosis was done. Their postoperative period was uneventful.

INTUSSUSCEPTION:

Submucosal lipoma with intussusception for which enterotomy and excision of the same was done. One case who had ileocaecal type, limited hemicolectomy was done.

ILEOSIGMOID KNOTTING:

Resection of the knot along with ileum and sigmoid enbloc with end to end anastomosis was done.In our study 2 cases were operated with resection and anastomosis.

MISCELLANEOUS

SECONDARIES CAUSING INTESTINAL OBSTRUCTION:

Adenoma of ileum with intussuception was resected and end to end anastomosis

SMALL BOWEL TUMOURS:

One case of leiomyoma arising from the ileum was operated and end-to-end anastomosis was done. Patient was admitted with obstructive features.

DISCUSSION

In our study of 109 patients with acute Intestinal obstruction, about 48 cases of Small Bowel obstruction due to external hernias and 42 cases of small bowel obstruction due to adhesions were recorded contributing 44.03% and 39.44% respectively. In contrast to Western literature, where 22.1% of small bowel obstructions occur due to external hernias and 47.4% of small bowel obstruction due to adhesions. The difference of contribution of small bowel obstruction is due to early intervention and awareness of the patients as compared with the western world.

In this study, External hernia contributes to 44.03% (48 cases) of Acute Intestinal obstruction. Inguinal hernia contributes to 79.16% (38 cases) to external hernia and 34.86% to total small bowel obstruction. It ranks FIRST among the causes of acute intestinal bowel obstruction. This is in contrast with the world literature were adhesive obstruction is the prime cost (40%), and hernia becomes the second cause of obstruction (25%).

We have encountered only 3 cases of femoral hernia and 5 cases of Paraumbilical hernia and 2 cases of incisional hernia, which together contributes only 20.83% of external hernia and 9.17% of in total.

The high prevalence of inguinal hernia causing obstruction is attributed to

a) Inadequate knowledge about the disease proper because of low literacy.

b) Reluctance of patient to undergo Elective repair of hernia

c) High prevalence of chronic cough (viz.tuberculosis etc.)

Even though the prevalence of inguinal hernia causing obstruction is Early arrival of patient once obstruction occurs even though he doesn't care it before.

a) Early recognition and immediate treatment.

In our study Adhesive obstruction accounts for 39.44% (42cases) of acute intestinal obstruction ranks second. Among this 2 cases were due to Post-surgical adhesion, 42 cases due to post inflammatory adhesion and 4 cases due to Meckel's devrticulum . Among this strangulation occurred in 8 cases involving the small bowel only. In adhesive obstruction, in western countries is genetic predisposition. Increased incidence of Caesarean section, Hysterectomies and P.I.D accounts for more incidence of adhesive obstruction in females.

Still the Abdominal tuberculosis account for 6.00% (6 cases) in total as a cause small bowel obstruction. Even with advent of potent anti tuberculosis drugs the reason for failure of improvement of situation is not known to certain. The occurrence of small bowel volvulus was 2 % (2 cases) in contrast to Agarwal and Misra's observation whom reported 20%. Surprisingly we have come across 4 cases of Intussusceptions (IIeo-Colic type-1, Ileo-ileo -3) and 2 cases of Meckel's diverticulum (all of them are male) causing obstruction. Surprisingly we have note 2 cases of secondaries in the omentum adherent to and infiltrating ileum, causing the obstruction, in this case we took biopsy. One case of small bowel tumour was encountered.

The clinical parameters like Continuous pain, Fever (>37.2*^{o} C), Taccycardia (>100/mt), Palpable abdominal mass enable us to detect the presence of strangulation in about 8% of cases, that too mainly in external hernias. Shatila and Chamberlain. Sarr et al in a large series highlighted the failure of these clinical parameters to differentiate between simple and strangulated obstruction.

Plan X-ray Abdomen is still valuable in diagnosing the bowel obstruction. In our study we able to get multiple fluid levels in radiological picture in about 85% of cases. We haven't got positive air-fluid level in early stage of obstruction in cases of inguinal hernia. But exact localization of obstruction necessitates other complementary investigations

Early reorganization by the patient and prompt treatment by surgeon gives good reward and decreases the mortality.

CONCLUSION

1. The major cause of acute intestinal obstruction is still External hernia (44.03%)here. Among this, inguinal hernia alone accounts for 79.16% in total.
2. Adhesive obstruction accounts for 39.449% in total, of which the Post-inflammatory adhesion is the major cause.
3. Abdominal tuberculosis accounts for 6%.
4. The incidence of small bowel volvulus is 2%.
5. Morbidity in our study is 11% and Mortality is 3.66%
6. Plain X-ray abdomen is a valuable in the diagnosis of the acute obstruction (73%) and hence it is considered as minimal investigation before surgery.
7. Early surgical intervention and antibiotics has reduced the mortality of the simple bowel obstruction.
8. In Strangulated obstruction, the mortality rate is still significantly more, due to aged, associated disease like diabetic mellitus, heart disease, renal the mortality.
9. Early diagnosis and early surgical intervention is the key to reduce the mortality.

Prof .Dr.RMN Unit

A CASE STUDY OF SMALL BOWEL OBSTRUCTION IN TMCH, THANJAVUR

PROFORMA

NAME:	AGE:	SEX: IP NO:
OCCUPATION:	PLACE:	DOA:
		DOS:
		DOD:
PRESENT COMPLAINTS:		DURATION:

1.
2.
3.
4.

HISTORY OF PRESENT OLLNESS

1.Abdominal pain

2.Vomiting

3.Distension

4.Constipation/Obstipation

5.Hemetemesis/Melena

6.pyrexia/Diarrhoea/Blood&mucus

7.Post Prandial Abdominal Pain

PAST HISTORY

1.H/o Surgery

2.H/o Exposure to TB

3.H/o Hypertension/Ischemic Heart Disease/Diabetes

FAMILLY HISTORY

H/O Similar illness in family members

PERSONAL HISTORY

1.Diet

2.Smoking

3.Alcohol consumption

ON EXAMINATION

1.Build

2.Nutrition

3.Hydration Status

. 4.Anaemia

5.Jaundice

6.Lymphatics

7.Pedal Edema

8.Pulse

9.Blood Pressure

10.Respiratory Rate

11.Temperature

EXAMINATION OF THE ABDOMEN

INSPECTION

1.Distension -central

-peripheral

2.Visible Intestinal Peristalis

3.hernial sites -Inginal/Femoral/Umblical

PALPATION

1.Rigidity

2.Guarding

3.Tenderness

4.Lump

5.Ascites

PERCUSSION

ASCULTATION

PER RECTAL EXAMINATATION

1. Empty
2. Red current Jelly
3. Blood stained
4. Lump
5. Anterior wall Bulge
6. Ballooned out

OTHER SYSTEM EXAMINATION

INVESTIGATIONS

URINE EXAMINATION

1.urine output

2.albumin

3.sugar

4.deposits

BLOOD EXAMINATIONS

1. Hb%
2. Blood Urea
3. Blood sugar
4. Serum creatinine

5. Serum Electrolytes
6. Blood Grouping/Typing
7. Bleeding time
8. Clotting time

RADIOLOGY

1.

2.

3.

4.

5.

DIAGNOSIS

TREATMENT

1.Oerative findings

2.Procedure done

3.Post operative period

FOLLOW UP:

BIOPSY REPORT

BIBLIOGRAPHY

1. Lee McGregor's Synopsis of Surgical Anatomy 12th Edition 1999 Pages 24-39.
2. David C.Sabiston:Text Book of Surgey 17th edition, 2004 P.1323-1380
3. Charles V.Mann in Bailey and Love's Short Practice of Surgery 24th Edition, 2004. P.1058-1075.
4. Cuschieri A.in Essential Surgical Practice 4th edition, 2004. P. 1423-1431.
5. Dudley HAF, Fielding LP in Hamilton Bailey's Emergency Surgery 13th edition, 2000, P.435-473.
6. Harlod Ellis in Maingot's Abdominal Operations 10th edition, P.1159-1172, 1391-1402. p.479-492
7. Human embryology VI edition-By INDERBIR SINGH
8. Chaurasia BD-Human Anatomy-12th Edition P.218 to 224,1995
9. Agarwal RL,Misra MK: Volvulus of the small intestine in Nortine in Northern India. Am.J.Surg.120:366,1970
10. Bizer LS, Liebling RW, Delaney HM: Delaney HM: small bowel Obstruction. Surgery 89:407-413, 1981.

11. Brolin RE: The role of GI tube decompression in the treatment of Mechanical intestinal obstruction. Am.Surgeon 49:131-137, 1983.

12. Chaib F, Toniolo CH, Figueira NC, Santanail: Surgical Treatment of Intestinal obstruction. Arn.Gastroenterol 1990. Oct.-Dec. 27(4): 182-6.

13. Cox MR, Gunn IF, Eastman MC, Hunt RF, Heinz AW: the Safety and duration of non-operative treatment for adhesive small bowel obstruction. Aust.NZ.J.Surg 1993 May:63(5) : 367-71

14. Das, P.Shukla HS: Clinical diagnosis of Abdominal tuberculosis Brit. J.Surg.63: 941, 1976.

15. Fabri PJ. Ellison EC, Anderson ED: High molecular weight dextran effect on adhesion formation and peritonitis in rats. Surgery 94:336-341, 1983.

16. Goar Iav, Barmiia NN, Domanski BV, Esebua BA: Surgical treatment of acute intestinal obstruction. Khir 1991: (4) :1-3.

17. Harlow CL, Sterar RL, Archer PG: Diagnosis of bowel obstruction on plain abdominal radiographs: Significance of air-fluid levels at different hights in the same loop of bowel AJR Am.J.Roentgenol 1993. Aug.161 (2) : 291-5.

18. Iko Bo, Teal JS, Siran SM: Computed Tomography of adult intussusception clinical and experimental studies, AJR 143: 769-772, 1984.

19. Joshi MJ: Surgical management of intestinal tuberculosis.

20. A conservative approach Ind.J.Surg.40.78.

21. Liu mY. Lin HH WU CS, Jn YY, Wang KL: Etiology of Intestinal Obstruction-4 years experience. Chang Kang I H such 1990 Sep. 13(3): 161-6.

22. McEntee G, Pender D,Mulvin D:Current spectrum of intestinal obstruction. Br.J.Surg 74:976, 1987.

23. Mucha P.Small Intestinal obstruction. Surg. Clin.North Am.67:597, 1987.

24. Peetz OJ, Gamelli RL, Pilcher DB: Intestinal intubation in Acute mechanical small bowel obstruction. Arch Surg. 117:334-336, 1982.

25. Pujari BD: Modified Surgical Procedures in intestinal tuberculosis Br.J.surg 66:180, 1979.

26. Saar MG, Bulkley GB, Zuidema GD: Pre-operative recognition of intestinal strangulation obstruction. Am.J.Surg. 145:176-182, 1983.

27. Schwartz, Shires, Spencer. Principles of Surgery 6th edition, P.1153-1306, 1994.

28. Shatila AH, Chamberlain BE, Webb WR: Current status in disgnosis and management of strangulation obstruction of the small bowel. Am.J.Surg. 299:132, 1976.

29. Vokov Elu, Mura EV AV: Surgical Treatment of Mekel's diverticulum. Sov Med 1001: (3): 80-2.

30. William O.Richards, Lester F.Williams: Acute Intestinal obstruction, Surg. Clin. North. Am.Vol.68/No.2 Apr. 1988.P.355-373.

31. Wolfson PJ, Bauer JJ, Gelernj IM: Use of the long tube in management of patients with small intestinal obstruction due to adhesion. Arch.Surg.12:1001-6, 1985.

32. Weil Bacher D, Bolin JA et al, Intussusception in Adults Amer.Journ. Surgery 121.531,1971.

33. Oxford textbook of Surgery:2nd Edition vol.II pages 1867-1876

34. Management of Abdominal Hernias:3rd Edition 1998

    a. Andrew.N.Kingsnorth,Karl.A.Leblanc

35. Nyhus LM.Iliopubic tract reapir of inguinal and femoral hernia.The postirior (prepretonial) approach:Surgical Cli.North America 1993:73:487

36. Maingot's Abdominal operations:Michael.J.Zinner,Seymour

37. I.Schwartz,Harold Ellis 10th Edition pages 479-492