A DISSERTATION ON

"CLINICAL STUDY ON SMALL INTESTINE OBSTRUCTION ON SEVERITY INDICATORS,ETIOLOGY, SURGICAL OUTCOME

SUBMITTED

TO

THE TAMILNADU DR.MGR MEDICAL UNIVERSITY

In partial fulfillment of the regulations for the award of the

DEGREE OF M.S (GENERAL SURGERY)

BRANCH-1

DEPARTMENT OF GENERAL SURGERY

STANLEY MEDICAL COLLEGE AND HOSPITAL TAMILNADU

DR.MGR MEDICAL UNIVERSITY, CHENNAI

MAY 2020

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This is to certify that this dissertation entitled **CLINICAL STUDY ON SMALL INTESTINE OBSTRUCTION ON SEVERITY INDICATORS, ETIOLOGY, SURGICAL OUTCOME"** is the bonafide work Done by the candidate **Dr. T. RAJKUMAR Post Graduate Student (MAY 2017 to MAY 2020)** in the Department of General Surgery, Stanley Medical College, Chennai-1,with registration number **221711066**under my guidance and supervision for the award of **M.S. Degree Examination** , Branch-I **(GENERAL SURGERY)** to be held in May 2020 under the Tamilnadu DR.M.G.R. Medical University, Chennai. I personally verified the urkund.com website for the purpose of plagiarism check**.** I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 18% of plagiarism in the dissertation**.**

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DECLRATION

I **Dr. T. RAJKUMAR,** solemnly declare that this dissertation entitled **"CLINICAL STUDY ON SMALL INTESTINE OBSTRUCTION ON SEVERITY INDICATORS, ETIOLOGY, SURGICAL OUTCOME** is a bonafide work done by me in the department of general surgery, Govt. Stanley Medical College and Hospital, Chennai under the supervision of **Prof. Dr. T. SIVAKUMAR M.S.,** This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the university regulations for the award of M.S, Degree (General Surgery), Branch – I Examination to be held in May 2020.

DATE:

PLACE:

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I am extremely thankful to my patients who consented and participated to make this study possible.

ABBREVIATIONS

SBO	-	Small intestine obstruction
TLC	-	Total leukocyte count
CRP	-	C Reactive protein
CT	-	Computer tomography

TABLE OF CONTENTS

INTRODUCTION

:

- **SMALL INTESTINAL OBSTRUCTION** is one of the common acute emergencies 12 to 16 % in surgical pratice,earlyrecogition and prompt intervention can prevent irreversible ischemia and therby decrease the mortality and long term morbidy

80% of small intestinal obstruction are due to benign cause.

Common cause:

- Adhesion(previous surgery,intra abdominal surgery)
- Strangulated hernia
- Tuberculosis
- Crohn"s disease
- Mesenteric ischemia
- Gall stones
- Bezoar
- Paralytic lleus

AIMS AND OBJECTIVE

- To study the incidence and various etiology of small intestinal obstruction
- To study the various modes of presentation, importance of early diagnosis and management
- To study The role of imaging studies in determining the site and etiology
- To study the mortality rate and morbidity rate in acute small intestinal obstruction

METHODOLOGY

STUDY DESIGN

PROSPECTIVE OBSERVATIONAL STUDY

PLACE OF STUDY

DEPARTMENT OF GENERAL SURGERY -GOVERNMENT STANLEY MEDICAL COLLEGE ,CHENNAI.

DURATION

12 MONTHS

PATIENT SELECTION

INCLUSION- Patients in intensive care units ,age15-80,
EXCLUSION- pediatric age

SAMPLE SIZE 60

Written informed consent will be obtained from all subjects before enrolment in the study

- All patients who are admitted in intensive care units

- All patients are thoroughly examined , SPECIAL relevance on palpatory finding guarding and provisional dignosis of intestinal obstruction is made

- All patients were inserted ryle's and foleys

- All patients were regularly MONITERED,routine investigation with crp

- AII Patient were screened by using Xray, usg,ct with contrast

- All patients were followed up for a period of 2 months.

- All details regarding the study will be recorded according to the pre designed proforma mentioned below

SPECIFIC SEVERITY INDICATORS- EACH ONE POINT

- CONTINOUS PAIN IN ABDOMEN > 4 DAYS
- ABDOMINAL GUARDING
- TLC> 11000 CELLS/CUMM (ON ADMISSION)
- CRP> OR = 10mg/l
- X RAY MULTIPLE AIR FLUID LEVEL ,CT ABOMEN SHOWING REDUCTION OF BOWEL WALL CONTRAST ENHANCEMENT

SCORE >=3 UNDERWENT EXPLORATION AND THOSE <3 CONSERVATIELY MANAGED

BASED ON INDIVIDULIZED SEVERITY SCORE AN OBSERVATIONAL STUDY TO ANALYZE SUGGICAL VS CONSERVATIVE MANAGEMENT

REVIEW OF LITERATURE

CLASSIFICATION

Intestinal obstruction may be classified into two types:

● Dynamic, in which peristalsis is working against a mechanical obstruction. It may occur in an acute or a chronic form

● Adynamic, in which there is no mechanical obstruction; peristalsis is absent or inadequate (e.g. paralytic ileus or pseudo-obstruction).

Causes of intestinal obstruction

Dynamic

Intraluminal

Faecal impaction

Foreign bodies

Bezoars

Gallstones

Intramural

Stricture

Malignancy

Intussusception

Volvulus

Extramural

Bands/adhesions

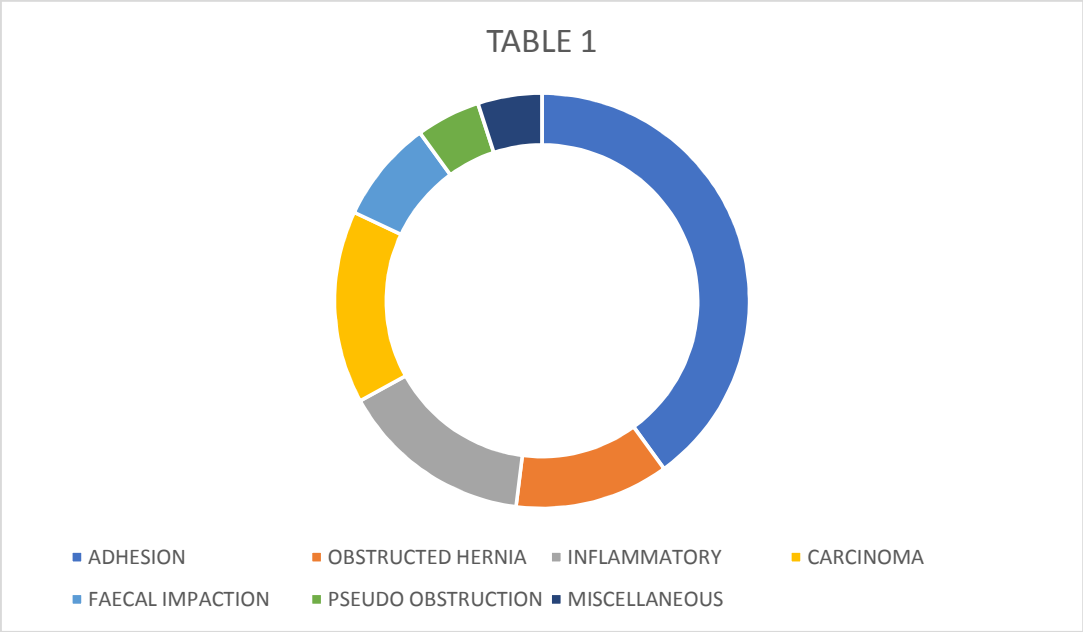
Hernia

Adynamic

Paralytic ileus

Pseudo-obstruction

Common cause of intestinal obstruction



PATHOPHSIOLOGY

Irrespective of aetiology or acuteness of onset, in dynamic (mechanical) obstruction the bowelproximal to the obstructiondilates and the bowel below the obstruction exhibitsnormalperistalsis and absorption until it becomes empty andcollapses. Initially, proximal peristalsis is increased in an attemptto overcome the obstruction. If the obstruction is not relieved,the bowel continues to dilate; ultimately there is a reductionthe bowel continues to dilate; ultimately there is a reductionin peristaltic strength, resulting in flaccidity and paralysis .

The distension proximal to an obstruction is caused bytwo factors ;

Gas: There is a significant overgrowth of both aerobicand anaerobic organisms, resulting in considerable gasproduction.Following the reabsorption of oxygen and carbondioxide, the majority is made up of nitrogen (90%) and hydrogen sulphide .

Fluid: This is made up of the various digestive juices.saliva 500 mL, bile 500 mL, pancreatic secretions 500 mL,gastric secretions 1 litre – all per 24 hours. This accumulatesin the gut lumen as absorption by the obstructed gut is retarded.

Dehydration and electrolyte loss are thereforedue to

-reduced oral intake;

- defective intestinal absorption;

-losses as a result of vomiting;

-sequestration in the bowel lumen;

- transudation of fluid into the peritoneal cavity

STRANGULATION;

It is important to appreciate that the consequences of intestinalobstruction are not immediately life-threatening unless

there is superimposed strangulation. When strangulationoccurs, the blood supply is compromised and the bowelobstruction are not immediately life-threatening unlessthere is superimposed strangulation. When strangulation occurs, the blood supply is compromised and the bowel becomes ischaemic.

Causes of strangulation

Direct pressure on the bowel wall

- Hernial orifices

- Adhesions/bands

Interrupted mesenteric blood flow

- Volvulus
- Intussusception

Increased intraluminal pressure

-Closed-loop obstruction

Ischaemia from direct pressure on the bowel wall from aconstricting band such as a hernial orifice is easy to understand.Distension of the obstructed segment of bowel results inhigh pressure within the bowel wall. This can happen when only part of the bowel wall is obstructed as seen in Richter'shernias. Venous return is compromised before the arterialsupply. The resultant increase in capillary pressure leadsto impaired local perfusion and once the arterial supply isimpaired, haemorrhagic infarction occurs. As the viability ofthe bowel is compromised, translocation and systemic exposure to anaerobic organisms and endotoxin occurs.

The morbidity and mortality associated with strangulationare largely dependent on the duration of the ischaemia andits extent. Elderly patients and those with comorbidities aremore vulnerable to its effects.

Although in strangulated external hernias the segment involved is often short, any lengthof ischaemic bowel can cause significant systemic effects secondary

to sepsis and obstruction proximal to the obstructioncan result in significant dehydration. When bowelinvolvement is extensive circulatory failure is common.

TYPES OF MECHANICAL INTESTINAL OBSTRUCTION

- **Obstruction by Adhesions and Bands:**

  Adhesions: Most commoncausefor intestinal obstruction.The lifetime risk of requiring anadmission to hospital for adhesional small bowel obstructionsusequent to abdominal surgery is around 4% and the risk ofrequiring a laparotomy around 2%. Adhesions start to formwithin hours of abdominal surgery. In the early postoperativeperiod, the onset of such a mechanical obstruction may be
  difficult to differentiate from paralytic ileus.

Any source of peritoneal irritation results in local fibrin production, which produces adhesions between apposed surfaces. Early fibrinous adhesions may disappear when the cause is removed or they may become vascularised and be replaced by mature fibrous tissue.

The common causes of intra-abdominaladhesions

Acute inflammation Sites of anastomoses-

reperitonealisation of rawareas, trauma, ischemia

Foreign material -Talc, starch, gauze, silk

Infection -Peritonitis, tuberculosis

Chronic inflammatory - Crohn's disease

Radiation enteritis

The region of the gall bladder should not be explored.

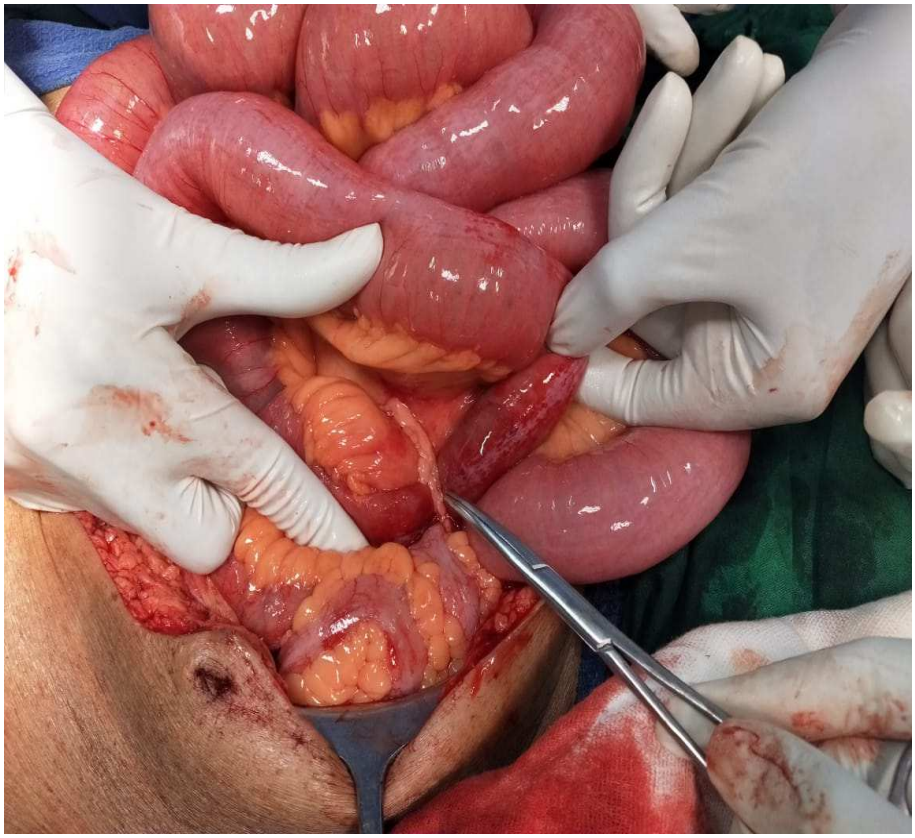


Figure1 Small bowel obstruction -Band

Prevention of adhesions

Factors that may limit adhesion formation include:

Good surgical technique

Washing of the peritoneal cavity with saline to remove clots

Minimising contact with gauze

Covering anastomosis and raw peritoneal surfaces

Adhesions may be classified into various types by virtueof whether they are early (fibrinous) or late (fibrous) or byunderlying aetiology. From a practica l perspective there areonly two types – ‘easy’ flimsy ones and ‘difficult’ dense. Postoperative adhesions giving rise to intestinal obstructionusually involve the lower small bowel and almost neverinvolve the large bowel.

Bands: Usually only one band is culpable. This may be:

-congenital, e.g. obliterated vitellointestinal duct;

-a string band following previous bacterial peritonitis

-a portion of greater omentum, usually adherent to parietes.

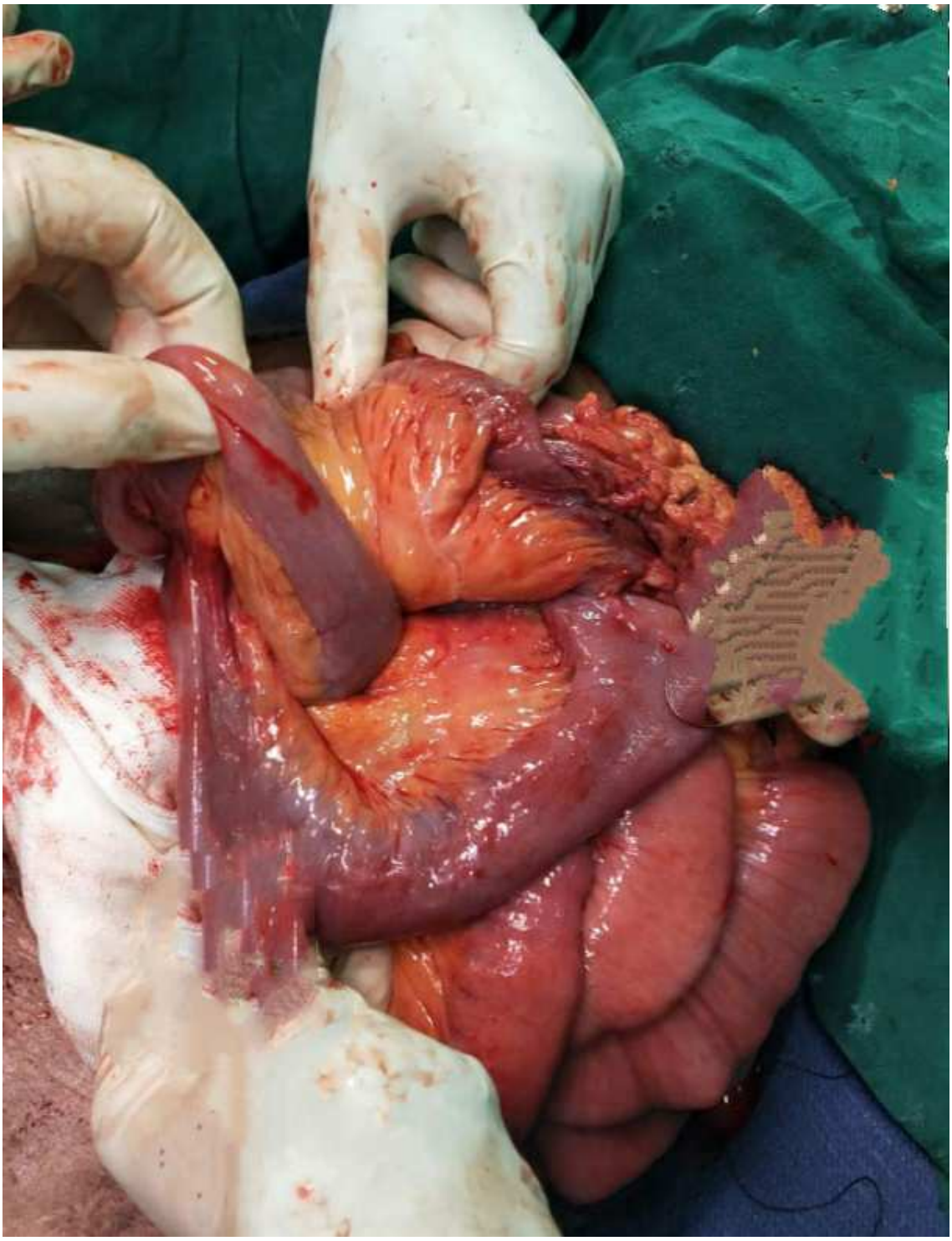


Figure 2 small bowel obstruction -Internal hernia

Internal hernia: Internal herniation occurs when a portion of the small intestinebecomes entrapped in one of the retroperitoneal fossae or in a congenital mesenteric defect .

-The foramen of Winslow;

-A defect in the mesentery;

-A defect in the transverse mesocolon;

-Defects in the broad ligament;

-Congenital or acquired diaphragmatic hernia;

-Duodenal retroperitoneal fossae – left paraduodenal and right duodenojejunal;

-caecal/appendiceal retroperitoneal fossae – superior, inferior and retrocaecal;intersigmoid fossa.

Internal herniation in the absence of adhesions is rare anda preoperative diagnosis is unusual. The standard treatmentof an obstructed hernia is to release the constricting agent bydivision. This should not be undertaken in cases of herniationinvolving the foramen of Winslow, mesenteric defects and

theparaduodenal/duodenojejunal fossae as major blood vesselsrun in the edge of the constriction ring. The distended loopin such circumstances must first be decompressed (minimisingcontamination) and then reduced.

- **Obstruction from enteric strictures**

Small bowel strictures usually occur secondary to tuberculosisor Crohn's disease. Malignant strictures associated with lymphoma are uncommon, whereas carcinoma and sarcoma arerare. Presentation is usually subacute or chronic. Standardsurgical management consists of resection and anastomosis.Resection is important to establish a histological diagnosis as this can be uncertain clinically. In Crohn's disease, strictureplastymay be considered in the presence of short multiplestrictures without active sepsis.

- **Bolus obstruction**

Gallstones:This type of obstruction tends to occur in the elderly secondaryto erosion of a large gallstone directly through the gall bladder into the duodenum. Classically, there is impactionabout 60 cm

proximal to the ileocaecal valve. The patientmay have recurrent attacks as the obstruction is frequentlyincomplete or relapsing as a result of a ball-valve effect.

Thecharacteristic radiological sign of gallstone ileus is Rigler'striad, comprising: small bowel obstruction, pneumobilia andan atypical mineral shadow on radiographs of the abdomen.The presence of two of these radiological signs has been consideredpathognomic of gallstone ileus and is encounteredin 40–50% of the cases (note than pneumobilia is common following endoscopic retrograde cholangiopancreatographyERCP) with sphincterotomy). At laparotomy, the stone ismilked proximally away from the site of impaction. It may bepossible to crush the stone within the bowel lumen; if not, theintestine is opened at this point and the gallstone removed.If the gallstone is faceted, a careful check for other entericstones should be made.

Food:Bolus obstruction may occur after partial or total gastrectomywhen unchewed articles can pass directly into the smallbowel. Fruit and vegetables are particularly liable to causeobstruction. The

management is similar to that for gallstone,with intraluminal crushing usually being successful .

Trychobezoars and phytobezoars: These are firm masses of undigested hair ball and fruit/vegetable fibre respectively. The former is due to persistenthair chewing or sucking, and may be associated with an underlyingpsychiatric abnormality. Predisposition to phytobezoars results from a high fibre intake, inadequate chewing, previous gastric surgery, hypochlorhydria and loss of the gastric pump mechanism. When possible, the lesion may be kneaded intothe caecum; otherwise open removal is required. A preoperative diagnosis is difficult even with high-resolution computed tomography (CT) scanning.

Stercoliths:

These are usually found in the small bowel in associationwith a jejunal diverticulum or ileal stricture. Presentation and management are identical to that of gallstones.

Worms: Ascaris lumbricoides may cause low small bowel obstruction,particularly in children, the institutionalised and those near the tropicsAn attack may follow the initiationof antihelminthic therapy. Debility is frequently out of proportionto that produced by the obstruction. If worms are notseen in the stool or vomitus the diagnosis may be indicated by eosinophilia or the sight of worms within gas-filled smallbowel loops on a plain radiograph (Naik). At laparotomy itmay be possible to knead the tangled mass into the caecum;if not it should be removed. Occasionally, worms may causea perforation and peritonitis, especially if the enteric wall is weakened by such conditions as ameobiasis.

Figure 3 SBO due to worms (Ascaris lumbricoides)

CLINICAL FEATURES OF INTESTINAL OBSTRUCTION:

Dynamic obstruction : The diagnosis of dynamic intestinal obstruction is based on the classic quartet of pain, distension, vomiting and absolute constipation.

Obstruction may be classified clinically into two types:

- Small bowel obstruction – high or low;
- Large bowel obstruction.

The nature of the presentationpresentation will also be influenced by whether the obstruction is:

-Complete;
- Incomplete.

Features of obstruction

-In high small bowel obstruction, vomiting occurs early, isprofuse and causes rapid dehydration. Distension is minimalwith little evidence of dilated small bowel loops on abdominalradiography.

-In low small bowel obstruction, pain is predominant withcentral distension. Vomiting is delayed. Multiple dilated smallbowel loops are seen on radiography.

Cardinal clinical features of acute obstruction

Abdominal pain

Distension

Vomiting

Absolute constipation

Presentation will be further influenced by whether the obstruction is:

Simple – in which the blood supply is intact;

Strangulating/strangulated – in which there is interference to blood flow.

The clinical features vary according to:

- The location of the obstruction;

-The duration of the obstruction;

-The underlying pathology;

-The presence or absence of intestinal ischaemia.

Late manifestations of intestinal obstruction that may beencountered include dehydration, oliguria, hypovolaemicencountered include dehydration, oliguria, hypovolaemicshock, pyrexia, septicaemia, respiratory embarrassment and peritonism. In all cases of suspected intestinal obstruction, the hernial orifices must be examined.

PAIN:

Pain is the first symptom encountered; it occurs suddenly

andis usually severe. It is colicky in nature and usually centred onthe umbilicus (small bowel) or lower abdomen (large bowel)

The pain coincides with increased peristaltic activity. With increasing distension, the colicky pain is replaced by a mildand more constant diffuse pain. If there is no ischaemia andthe obstruction persists over several days, pain reduces andcan disappear.

The development of severe pain is suggestive of the presence of strangulation, especially if that severe pain is continuous.

Beware the patient whose pain is not controlled withintravenous opiates.

Colicky pain may not be a significantfeature in postoperative simple mechanical obstruction andpain does not usually occur in paralytic ileus.

VOMITING:

The more distal the obstruction, the longer the interval between the onset of symptoms and the appearance of nauseaand vomiting. As obstruction progresses the character of thevomitus alters from digested food to faeculent material, as aresult of the presence of enteric bacterial overgrowth.

DISTENSION:

In the small bowel the degree of distension is dependent on thesite of the obstruction and is greater the more distal the lesion. Visible peristalsis may be present . This can sometimesbe provoked by 'flicking' the abdominal wall. Distensionis a later feature in colonic obstruction

and may be minimal orabsent in the presence of mesenteric vascular occlusion.

CONSTIPATION:

This may be classified as absolute (i.e. neither faeces nor flatusis passed) or relative (where only flatus is passed). Absolute constipation is a cardinal feature of complete intestinalobstruction. Some patients may pass flatus or faeces after theonset of obstruction as a result of the evacuation of the distalbowel contents. The administration of enemas should be avoided in cases of suspected obstruction. This merely stimulatesevacuation of bowel contents distal to the obstructionand confuses the clinical picture .

The rule that absolute constipation is present in intestinal obstruction does not apply in:

- Richter's hernia;
- Gallstone ileus;
- Mesenteric vascular occlusion;
- Functional obstruction associated with pelvic abscess;
- All cases of partial obstruction (diarrhoea)

OTHER MANIFESTATION:

DEHYDRATION:

Dehydration is seen most commonly in small bowel obstructionbecause of repeated vomiting and fluid sequestration. It results in dry skin and tongue, poor venous filling and sunkeneyes with oliguria. The blood urea level and haematocrit rise,giving a secondary polycythaemia

HYPOKALEMIA:

Hypokalaemia is not a common feature in simple mechanicalobstruction. An increase in serum potassium, amylase or lactate dehydrogenase may be associated with the presence ofstrangulation, as may leucocytosis or leucopenia.

PYREXIA:

Pyrexia in the presence of obstruction is rare and may indicate:

- The onset of ischaemia;

-Intestinal perforation;

- Inflammation or abscess associated with the obstructing disease.

Hypothermia indicates septicaemic shock or neglectedcases of long duration.

ABDOMINAL TENDERNESS:

Localised tenderness indicates impending or established ischaemia.The development of peritonism or peritonitis indicatesovert infarction and/or perforation. In cases of large bowelobstruction, it is important to elicit these findings in the rightiliac fossa as the caecum is most vulnerable to ischaemia.

BOWEL SOUNDS:

High-pitched bowel sounds are present in the vast majority ofpatients with intestinal obstruction. Normal bowel sounds are of negative predictive value. Bowel sounds may be scanty orabsent if the obstruction is longstanding and the small bowelhas become inactive.

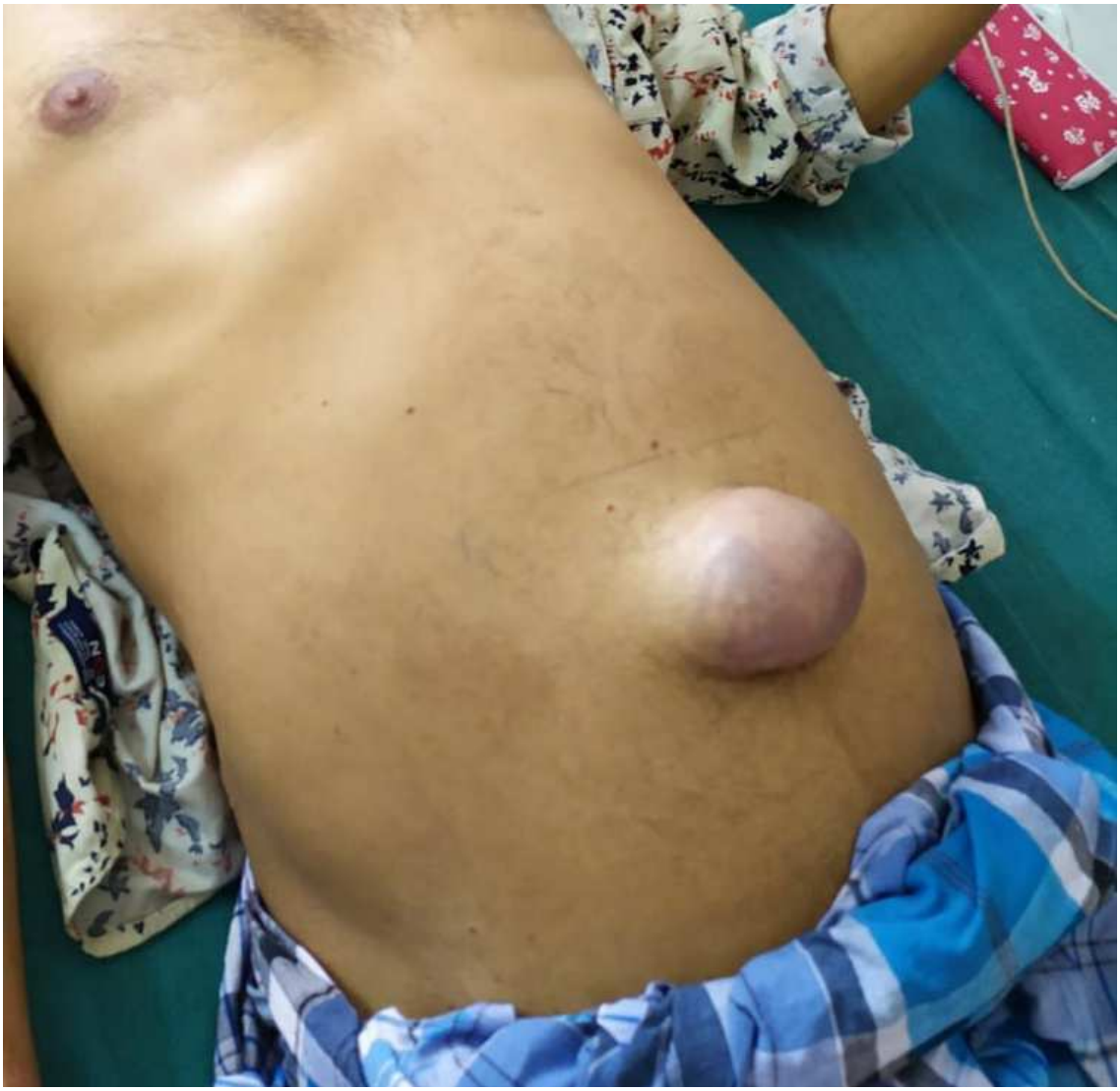


Figure 4 SBO due to strangulated umbilical hernia with distension

Clinical features of strangulation

It is vital to distinguish strangulating from non-strangulating intestinal obstruction because the former is a surgical emergency.

The diagnosis is almost entirely clinical

- Constant pain, severe pain
- Tenderness with rigidity and peritonism
- Shock

In addition to the features above, it should be noted that:

- The presence of shock suggests underlying ischaemia, especially if the shock is resistant to simple fluid resuscitation.
- In impending or established strangulation, pain is never completely absent.
- The presence and character of any local tenderness are of great significance and, however mild, tenderness requires frequent reassessment.
- Generalised tenderness and the presence of rigidity indicate

the need for early laparotomy.

-In cases of intestinal obstruction in which pain persistsdespite conservative management, even in the absence of the above signs, strangulation should be presumed.

-When strangulation occurs in an external hernia, the lump is tense, tender and irreducible and there is no expansile cough impulse. Skin changes with erythema or purplish discolouration are associated with underlying ischaemia.

IMAGING:

Erect abdominal films are no longer routinely obtained andthe radiological diagnosis is based on a supine abdominal film. An erect film may subsequently be requestedwhen further doubt exists. When distended with gas, the jejunum, ileum, caecum and remaining colon have a characteristic appearance inadults and older children that allows them to be distinguishedradiologically.

Radiological features of obstruction (on plain x-ray)

* The obstructed small bowel is characterised by straight segments that are generally central and lie transversely. No/ minimal gas is seen in the colon

*The jejunum is characterised by its valvulaeconniventes, which completely pass across the width of the bowel and are regularly spaced, giving a 'concertina' or ladder effect

* Ileum – the distal ileum has been piquantly described by Wangensteen as featureless

* Caecum – a distended caecum is shown by a rounded gas

shadow in the right iliac fossa

*Large bowel, except for the caecum, shows haustral folds, which, unlike valvulaeconniventes, are spaced.

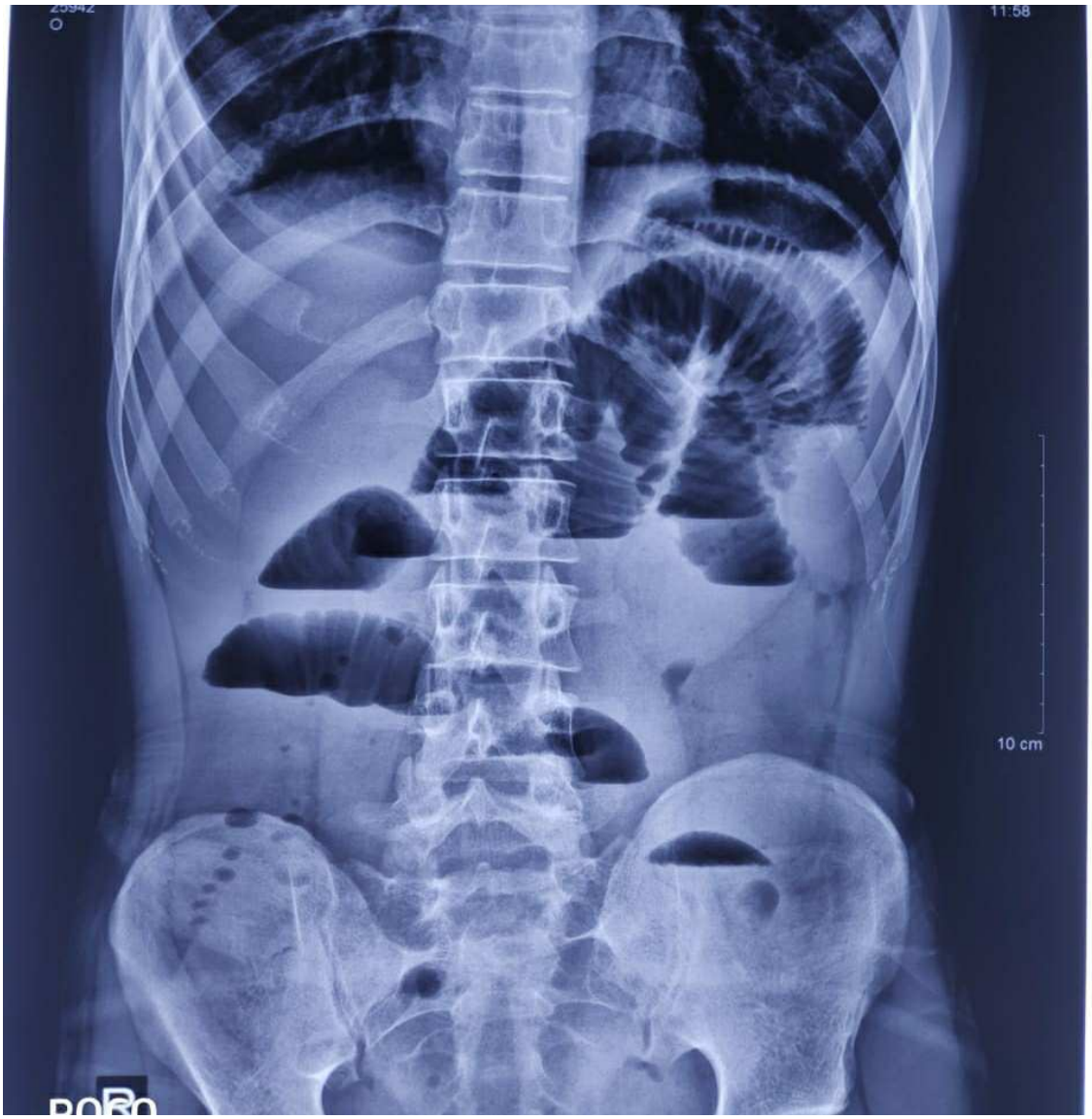


Figure 5 X ray abdomen showing Multiple air fluid levels in case of SBO

In intestinal obstruction, fluid levels appear later than gasshadows as it takes time for gas and fluid to separate These are most prominent on an erect film. In adults,two inconstant fluid levels – one at the duodenal cap and the other in the terminal ileum – may be regarded as normal. Ininfants (less than 1 year old), a few fluid levels in the smallbowel may be physiological. In this age group it is difficult todistinguish large from small bowel in the presence of obstruction,because the characteristic features seen in adults are not present or are unreliable.

During the obstructive process, fluid levels becomemore conspicuous and more numerous when paralysis hasoccurred. When fluid levels are pronounced, the obstructionis advanced. In the small bowel, the number of fluid levels is directly proportional to the degree of obstruction and to itssite, the number increasing the more distal the lesion.

In patients without evidence of strangulation there is arole for other imaging modalities. A recent systematic reviewand meta-analysis of the diagnostic and therapeutic role of50–100mL water-soluble contrast agent in adhesive small bowel obstruction included 14 prospective studies. Theappearance of contrast in the colon 4–24 hours after administrationhad a sensitivity of 96% and a specificity of 98% inpredicting resolution of small bowel obstruction. If contrastdoes not reach the colon, sugery is required in about 90% ofpatients. Administration of a water-soluble agent was alsoeffective in reducing the need for surgey (OR 0.62; p = 0.007)and shortening hospital stay.

In contrast, low colonic obstruction does not commonlygive rise to small bowel fluid levels unless advanced, whereashigh colonic obstruction may do so in the presence of anincompetent ileocaecal valve. Colonic obstruction is usually associated with a large amount of gas in the caecum. Alimited water-soluble enema should be undertaken to differentiatelarge bowel obstruction from pseudo-obstruction. Abarium follow-through is contraindicated in the presence ofacute obstruction and may be life-threatening.

The CT scan is now used very widely to investigate allforms of intestinal obstruction. It is highly accurate and itsonly limitations are in diagnosing ischaemia. Two CT scanfindings may be used in clinical practice when looking forintestinal ischaemia: reduced enhanced bowel wall is highly predictive of ischaemia and absence of mesenteric fluid is areliable finding to rule out strangulation. It is important toremember that even with the best imaging techniques, thediagnosis of strangulation remains a clinical one.

●Reduced bowel wall enhancement on CT increases the probability of strangulation 11-fold.

● Absence of mesenteric fluid on CT decreases the probability of strangulation 6-fold.

● The clinical reliability of other CT signs is doubtful for predicting strangulation

Impacted foreign bodies may be seen on abdominal radiographs.It is noteworthy that gas-filled loops and fluid levels inthe small and large bowel can also be seen in established paralyticileus and pseudo-obstruction. The former can, however,normally be distinguished on clinical grounds whereas the latter can be confirmed radiologically. Fluid levels may alsobe seen in nonobstructing conditions such as gastroenteritis,acute pancreatitis and intra-abdominal sepsis.

TREATMENT OF ACUTE INTESTINAL OBSTRUCTION:

There are three main measures used to treat acute intestinal obstruction.

Treatment of acute intestinal obstruction

-Gastrointestinal drainage via a nasogastric tube

- Fluid and electrolyte replacement

- Relief of obstruction

- Surgical treatment is necessary for most cases of intestinalobstruction but should be delayed until resuscitation iscomplete, provided there is no sign of strangulation orevidence of closed-loop obstruction.

The first two steps are always necessary before attempting

the surgical relief of obstruction and are the mainstay of postoperative management.

SUPPORTIVE MANAGEMENT:

Nasogastric decompression is achieved by the passage of a nonvented (Ryle) or vented (Salem) tube. The tubes arenormally placed on free drainage with 4-hourly aspiration butmay be placed on continuous or intermittent suction. As wellas facilitating decompression proximal to the obstruction,they are essential to reduce the risk of subsequent aspiration during induction of anaesthesia and post-extubation.The basic biochemical abnormality in intestinal obstructionis sodium and water loss, and therefore the appropriatereplacement is Hartmann's solution or normal saline. The volume required varies and should be determined by clinicalhaematological and biochemical criteria.Antibiotics are not mandatory but many clinicians initiatebroad-spectrum antibiotics early in therapy because ofbacterial overgrowth. Antibiotic therapy is mandatory for allpatients undergoing surgery for intestinal obstruction.

SURGICAL TREATMENT:

The timing of surgical intervention is dependent on the clinical picture. There are several indications for early surgical intervention.

Indications for early surgical intervention

- Obstructed external hernia
- Clinical features suspicious of intestinal strangulation
- Obstruction in a 'virgin' abdomen

Principles of surgical intervention for obstruction

Management of:

- The segment at the site of obstruction
-The distended proximal bowel
-The underlying cause of obstruction

The classic clinical advice that ‘the sun should not bothrise and set’ on a case of unrelieved acute intestinal obstructionwas based on the concern that intestinal ischaemia woulddevelop while the patient was waiting for surgery. If there iscomplete obstruction, but no evidence of intestinal ischaemia, it is reasonable to defer surgery until the patient has been adequately resuscitated. Where obstruction is likely to be secondaryto adhesions, conservative management may be continuedfor up to 72 hours in the hope of spontaneous resolution.

If the site of obstruction is unknown, adequate exposure is best achieved by a midline incision.

ASSESSMENT IS DIRECTED TO

- The site of the obstruction;

- The nature of the obstruction;

-The viability of the gut

In cases of small bowel obstruction, the first manoeuvreis to deliver the distended small bowel into the wound. Thispermits access to the site of obstruction. The small bowelshould be covered with moist swabs and the weight of thefluid-filled bowel supported such that the blood supply to the mesentery is not impaired.

Operative decompression should be performed wheneverpossible. This reduces pressure on the abdominal wound,reducing pain and improving diaphragmatic movement. Thesimplest and safest method is to insert a large-bore orogastric tube and to milk the small bowel contents in a retrogrademanner to the stomach for aspiration. All volumes of fluidremoved should be accurately measured and appropriatelyreplaced. It is important to ensure that the stomach is empty at the end of the procedure to prevent postoperative aspiration.

Rarely, decompression using Savage's decompressorwithin a seromuscular purse-string suture may be required. Its benefits should be balanced against the potential risk of septic complications from spillage and the risk of leakage from the suture line postoperatively. The type of surgical procedure required will depend upon the cause of obstruction – division of adhesions (enterolysis), excision, bypass or proximal decompression. Following relief of obstruction, the viability of theinvolved bowel should be carefully assessed Although frankly infarcted bowel is obvious, the viabilitystatus in many cases may be difficult to discern. If in doubt,the bowel should be wrapped in hot packs for 10

minutes withincreased oxygenation and then reassessed. The state of the mesenteric vessels and pulsation in adjacent arcades shouldbe sought. Viability is also confirmed by colour, sheen andperistalsis. If, at the end of this period, there is still uncertaintyabout gut viability, the gut should be resected if this doesnot result in short bowel syndrome. If the patient is septicsuch that they require inotropic therapy or would requirepostoperative level 3 intensive care treatment followingresection, consideration should be given to raising both endsof the bowel as stomas. This is not only safe but also allows regular assessment of the bowel.

Intestinal ischaemia/reperfusion injury has been describedfollowing reperfusion of ischaemic bowel with remote lunginjury resulting from the release of inflammatory mediators.This should be borne in mind when dealing with ischaemicbowel.

When no resection has been undertaken or there aremultiple ischaemic areas (mesenteric vascular occlusion), asecond-look laparotomy at 24–48 hours may be required.Special attention should always be paid to the sites of constriction

at each end of an obstructed segment. If of doubtfulviability they should be infolded by the use of a seromuscularsuture and can also be covered with omentum.

The surgical management of massive infarction is dependenton the patient's overall prognostic criteria. In the elderly,infarction of the small bowel from the duodenojejunal flexureto the right colon may be considered incurable, whereas inthe young, with the potential for long-term intravenous alimentation and small bowel transplantation, a policy of excisionmay be justified .

Whenever the small bowel is resected, the exact site ofresection, the length of the resected segment and that of theresidual bowel should be recorded .

As laparoscopic surgery is now so common, it is importantto note that small bowel obstruction and strangulationoccur in relation to port site hernias. The risk of port siteherniation is related to older age, higher body mass, trocar diameter and extension of the port site for tissue extraction.For laparoscopic cholecystectomy, the hernia rate is reportedto be around 2%. Obstruction and strangulation have evenbeen reported through 5-mm port sites. Complications

fromthese hernias may present in the early postoperative period

and as a Richter's hernia. They can be easily overlooked andcareful

examination of port sites in patients with small bowelobstruction is essential.

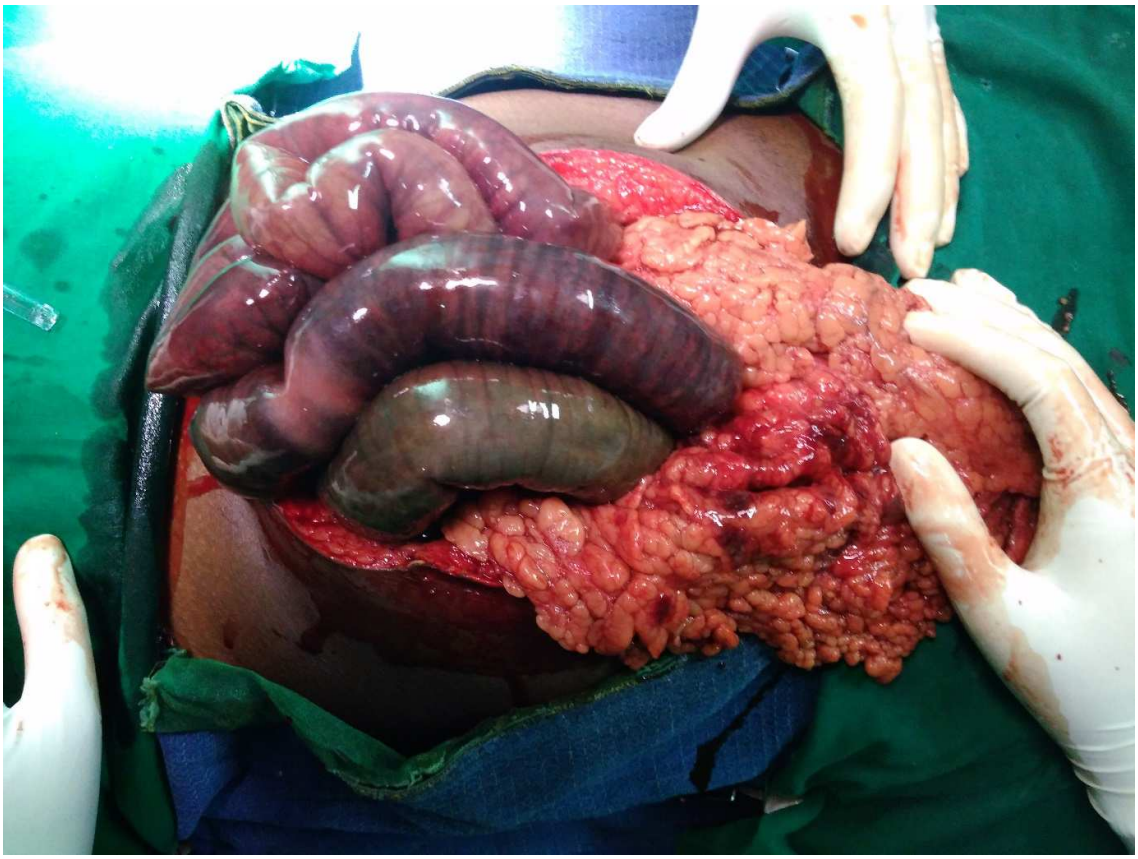


Figure 6 Mesenteric ischemia-intraoperative fiding entire small bowel gangrene, (Note-Dull and lustreless)

	viable	Non viable
Circulation	Dark colour becomes lighter Visible pulsation in mesenteric arteries	Dark colour remains No detectable Pulsation
General appearance	Shiny	Dull and lustreless
Intestinal Musculature	Firm	Flabby, thin and Friable
	Peristalsis may be observed	No peristalsis

Table 1 :Differentiation between viable and non-viable intestine

TREATMENT OF ADHESION:

Initial management is based on intravenous rehydration and nasogastric decompression; occasionally, this treatment iscurative. Although an initial conservative regimen is consideredappropriate, regular assessment is mandatory to ensurethat strangulation does not occur. Conservative treatment should not usually be prolonged beyond 72 hours.When laparotomy is required, although multiple adhesionsmay be found, only one may be causative. If there isabsolute certainty that this is the cause of the obstruction,this should be divided and the remaining adhesions can beleft in situ unless severe angulation is present. Division ofthese adhesions will only cause further adhesion

formation.

When obstruction is caused by an area of multiple adhesions,the adhesions should be freed by sharp dissection fromthe duodenojejunal junction to the caecum. Following therelease of band obstruction, the constriction sites that havesuffered direct compression should be carefully assessed and, if they show residual colour changes, invaginated with a seromuscularsuture. Laparoscopic adhesiolysis may be considered in highlyselected cases of small bowel obstruction. This is classed as anadvanced laparoscopic procedure and should only be undertakenby surgeons with advanced laparoscopic skills.

TREATMENT OF RECURRENT INTESTINAL OBSTRUCTION BY ADHESION:

Several procedures may be considered in the presence of recurrent obstruction including:

-Repeat adhesiolysis (enterolysis) alone;

- Noble's plication operation;

- Child–Phillips transmesenteric plication;

-Intestinal intubation.

POSTOPERATIVE INTESTINAL OBSTRUCTION:

Differentiation between persistent paralytic ileus and earlymechanical obstruction may be difficult in the early postoperative period. Mechanical obstruction is more likely if thepatient has regained bowel function postoperatively whichsubsequently stops. Obstruction is usually incomplete and themajority settle with continued conservative management. Postoperative intra-abdominal sepsis is a potent cause of postoperative obstruction; CT scanning with oral contrast is ofparticular value in the assessment of the postoperative abdomen.Instant gastrografin enemas are also

of value.

ADYNAMIC OBSTRUCTION:

PARALYTIC ILEUS

This may be defined as a state in which there is failure oftransmission of peristaltic waves secondary to neuromuscularfailure (i.e. in the myenteric (Auerbach's) and submucous(Meissner's) plexuses). The resultant stasis leads to accumulationof fluid and gas within the bowel, with associateddistension, vomiting, absence of bowel sounds and absolute constipation.

Varieties

The following varieties are recognised:

-**Postoperative:** a degree of ileus usually occurs after any abdominal procedure and is self-limiting, with a variableduration of 24–72 hours. Postoperative ileus may be prolongedin the presence of hypoproteinaemia or metabolicabnormality

-**Infection**: intra-abdominal sepsis may give rise to localised or generalised ileus.

-**Reflex ileus:** this may occur following fractures of the spine or ribs, retroperitoneal haemorrhage or even the application of a plaster jacket.

- **Metabolic:** uraemia and hypokalaemia are the most common contributory factors.

Clinical features

Paralytic ileus takes on a clinical significance if, 72 hours after laparotomy:

-There has been no return of bowel sounds on auscultation;

-There has been no passage of flatus

Abdominal distension becomes more marked and tympanitic.Colicky pain is not a feature. Distension increases painfrom the abdominal wound. In the absence of gastric aspiration,effortless vomiting may occur. Radiologically, the abdomenshows gas-filled loops of intestine with multiple fluid levels (if an erect film is felt necessary).

Management

Nasogastric tubes are not required routinely after elective intra-abdominal surgery. Paralytic ileus is managed with theuse of nasogastric suction and restriction of oral intake untilbowel sounds and the passage of flatus return. Electrolyte balancemust be maintained. The use of an enhanced recoveryprogramme with early introduction of fluids and solids is, however, becoming increasingly popular.

Specific treatment is directed towards the cause, but the following general principles apply:

- If a primary cause is identified this must be treated.

- Gastrointestinal distension must be relieved by decompression.

-Close attention to fluid and electrolyte balance is essential.

- There is no convincing evidence for the use of prokinetic drugs to treat postoperative adynamic ileus.

- If paralytic ileus is prolonged CT scanning is the most effective investigation; it will demonstrate any intraabdominalsepsis or mechanical obstruction and thereforeguide any requirement for laparotomy. Otherwisethe decision to take a patient back to theatre in these circumstances is always difficult. The need for a laparotomybecomes increasingly likely the longer the bowel inactivitypersists, particularly if it lasts for more than seven daysor if bowel activity recommences following surgery andthen stops again.

PSEUDO OBSTRUCTION:

This condition describes an obstruction, usually of the colon,that occurs in the absence of a mechanical cause or acuteintra-abdominal disease. It is associated with a varietmyopathy and a range of other factorsy of syndromes in which there is an underlyingneuropathy and/or myopathy and range of other

factors.

Small intestinal pseudo-obstruction

This condition may be primary (i.e. idiopathic or associated with familial visceral myopathy) or secondary. The clinicalpicture consists of recurrent subacute obstruction. The diagnosisis made by the exclusion of a mechanical cause. Treatmentconsists of initial correction of any underlying disorder.Metoclopramide and erythromycin may be of use.

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Factors associated with pseudo-obstruction

-**Metabolic**

Diabetes

Hypokalaemia

Uraemia

Myxodoema

Intermittent porphyria

- Severe trauma (especially to the lumbar spine and pelvis)

- Shock

Burns

Myocardial infarction

Stroke

-Idiopathic

-Septicaemia

-Postoperative (for example fractured neck of femur)

- Retroperitoneal irritation

Blood

Urine

Enzymes (pancreatitis)

Tumour

-**Drugs**

Tricyclic antidepressants

Phenothiazines

Laxatives

-**Secondary gastrointestinal involvement**

Scleroderma

Chagas' disease

RESULTS

FINDINGS

A prospective observation study – clinical study of small intestinal obstruction based on etiology,severity,indicators,surgical outcome was done for one year among 60 patients and following results were obtained**.**

Age distribution of patients

Mean age of patients: 44.18years

Standard deviation of age of patients: 11.88

Minimum age: 16 years

Maximum age: 72 years

Age distribution of Study participants(n=60)

Age category	Frequency (n)	Percentage (%)
11-20	1	1.64
21-30	8	13.11
31-40	12	19.67
41-50	18	29.51
51-60	17	27.87
>60	4	8.20

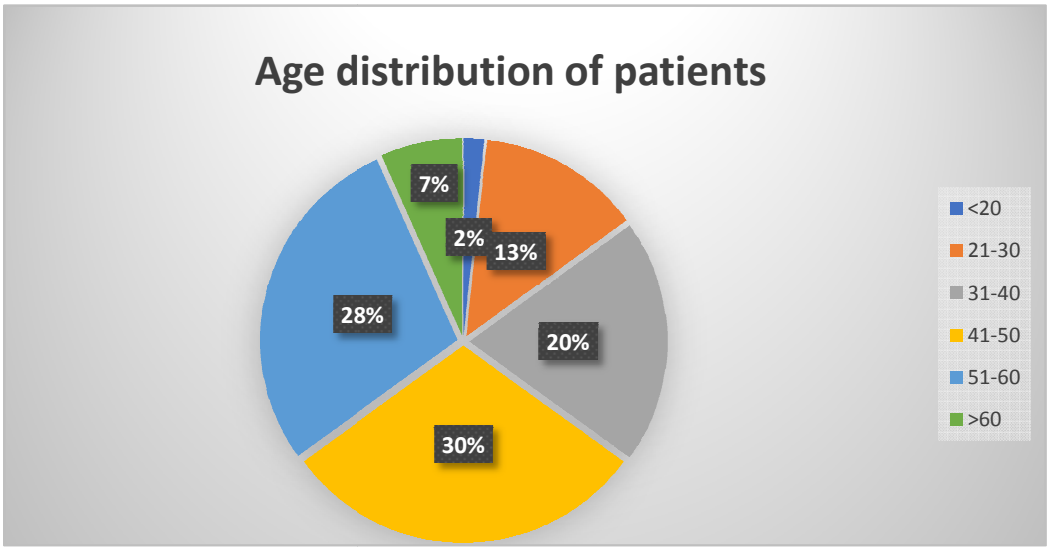

Figure 7: Age distribution of the patients

Gender distribution of the patients

Gender	Frequency	Percentage
Male	37	62%
Female	23	38%

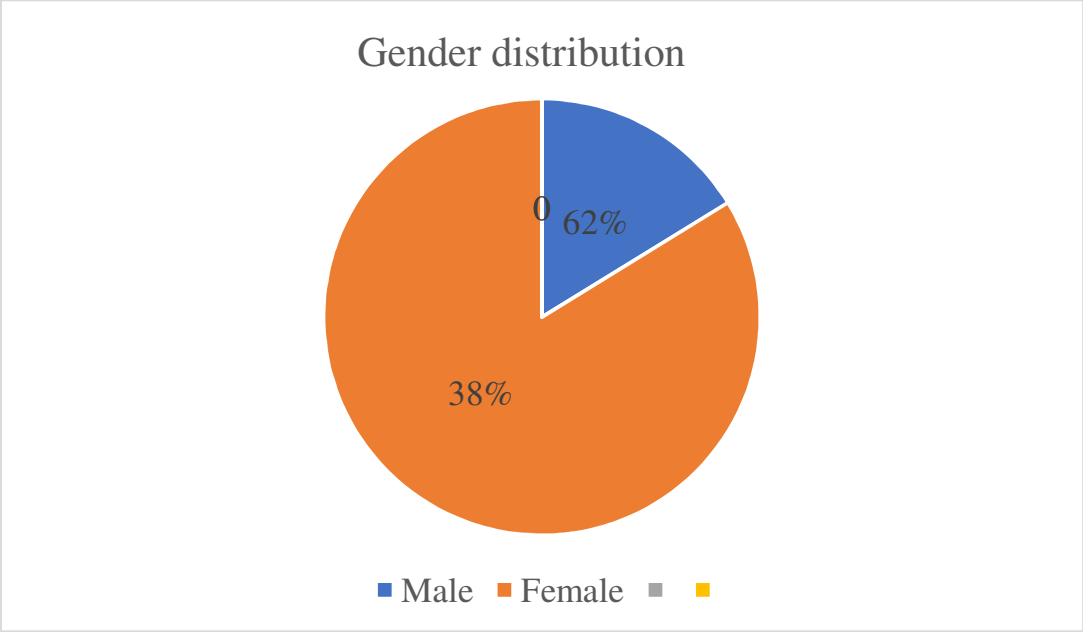

Figure 8 : Gender distribution

Majority of them were male 62%(n =38).The remainder was female 38% (n=23)

Distribution of study participants with abdominal pain > 4 days

Abdominal pain	Frequency	Percentage
Yes	49	81.67
No	11	18.33

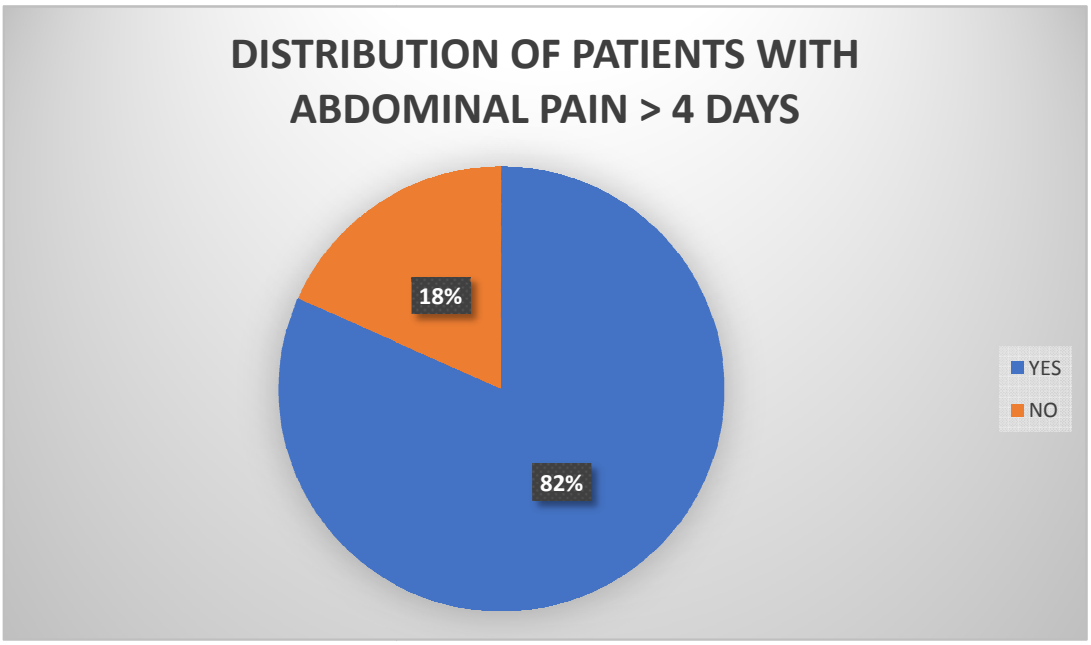

Figure 9 : Symptom abdominal pain > 4 Days

Figure 9 shows out of 60 study paticipants 81.67% (n=49) patients present with abdominal pain > 4days.

Distribution of study participants with constipation

Constipation	Frequency	Percentage
Yes	36	60
No	24	40

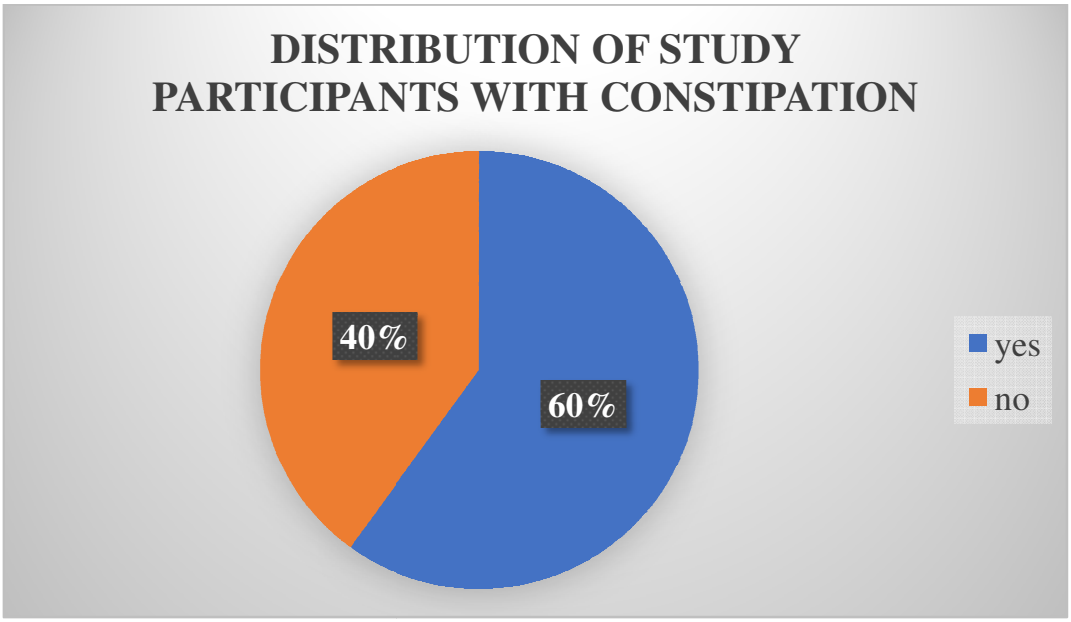

Figure10 :Distribution of study participants with constipation

Figure 10 shows distribution of patients in SBO presented with constipation.n=36(60%) presented with constipation.

Distribution of Patients with abdominal distension

Abdominal distension	Frequency	Percentage
Yes	30	50
No	30	50

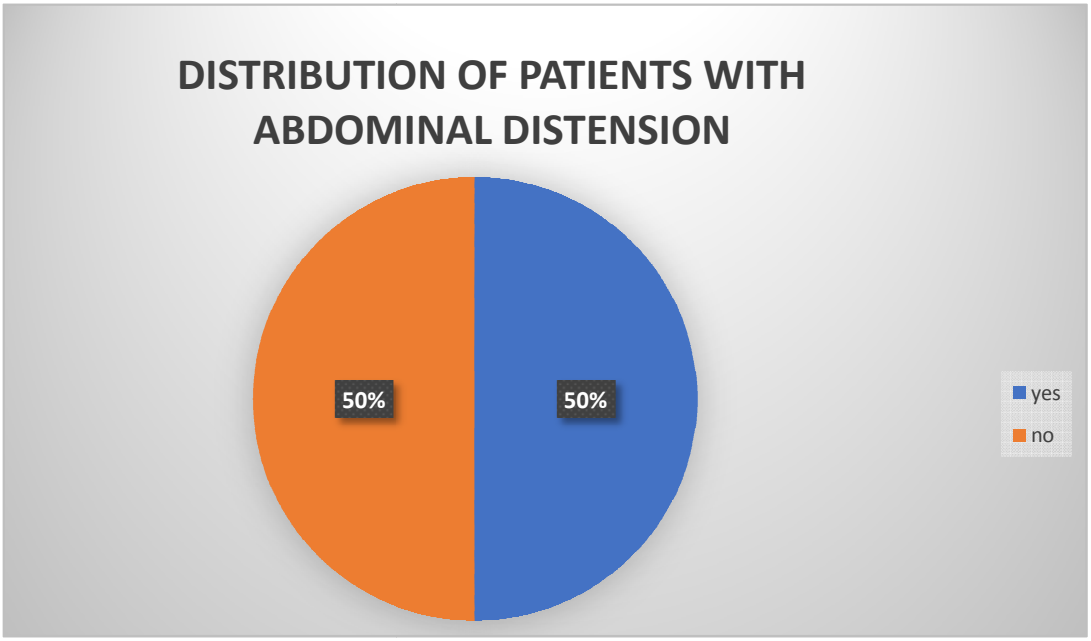

Figure 11 : Distribution of Abdominal distension

Figure 11 shows out of 60 patients,(n=30)50% were presented with abdominal symptoms and (n=30)50% not presented with abdominal symptoms.

Distribution of Patients with vomiting

Vomiting	Frequency	Percentage
Yes	55	91.67
No	5	8.33

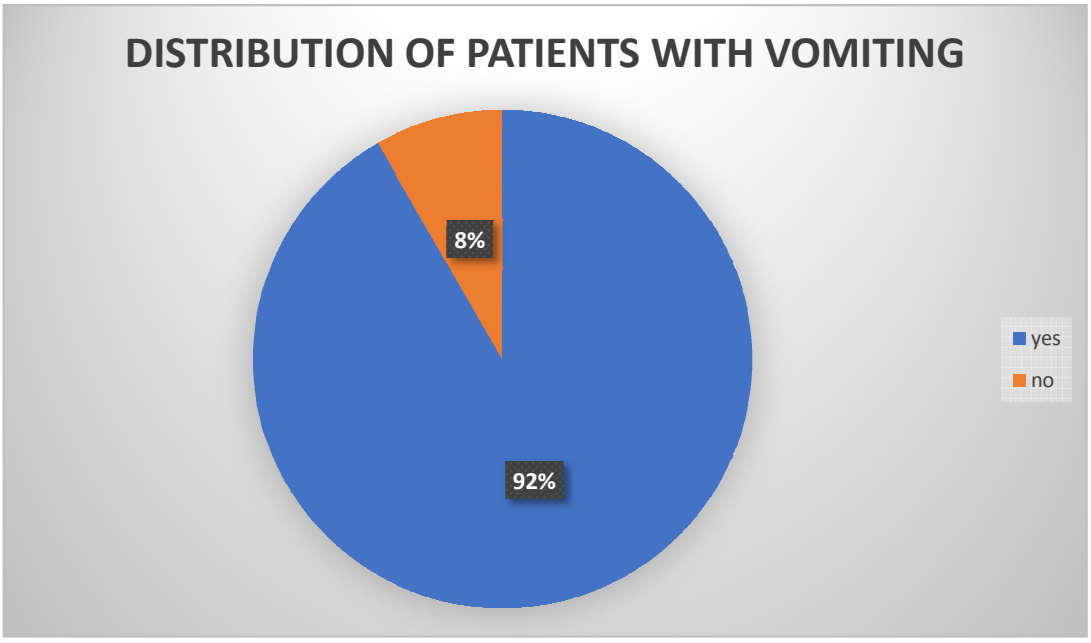

Figure12 :Distribution of vomiting

Figure 12shows out of 60 patients ,(n=55) 91.67% patients presented with vomiting,only n=5 8.33% were without it.

Distribution of Patients with Guarding

Guarding	Frequency	Percentage
Yes	22	36.67
No	38	63.33

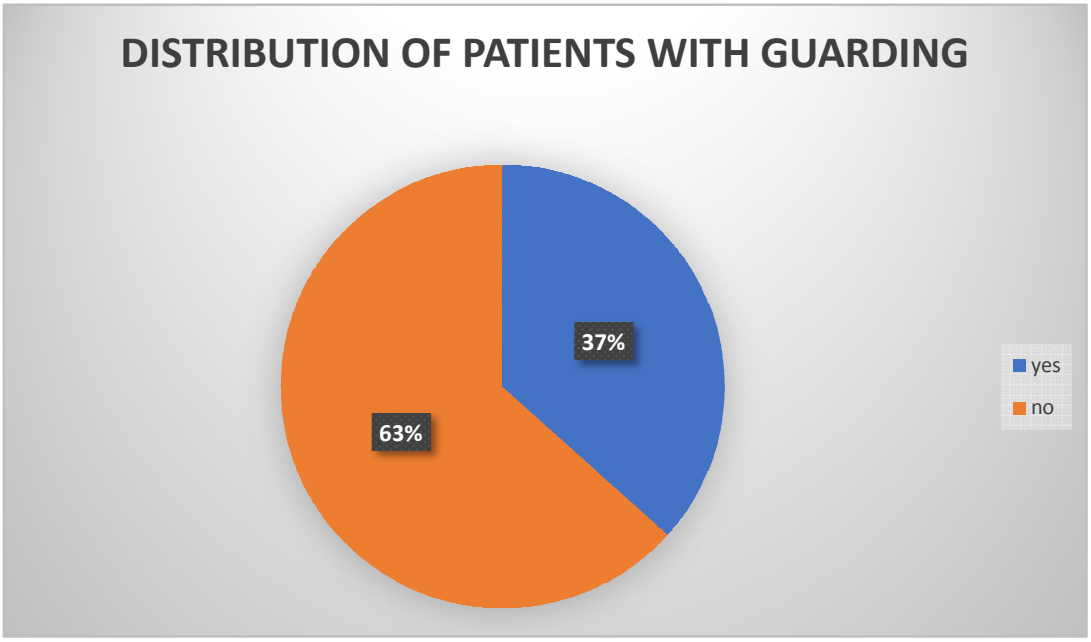

Figure 13: Distibution of Guarding

Figure 13shows out of 60 patients,only 37% n=22 were presented with abdominal guarding

Distribution of Patients with total leukocyte count >11000

TLC >11000	Frequency	Percentage
Yes	31	51.67
No	29	48.33

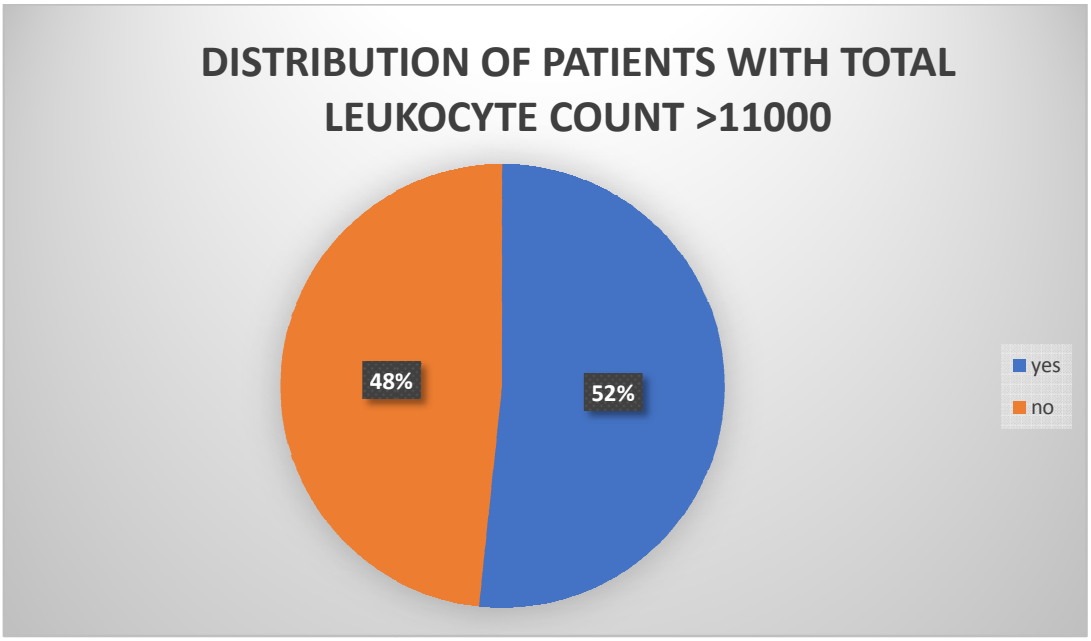

Figure 14 :Distribution of patients with >11000 TLC

Figure14 shows out of 60 patients ,(n=31) 51.67% pesented with TLC count >11000,remaining were (n=29) 48.33%

Distribution of Patients with CRP> 10

CRP>10	frequency	Percentage
Yes	29	48.33
No	31	51.67

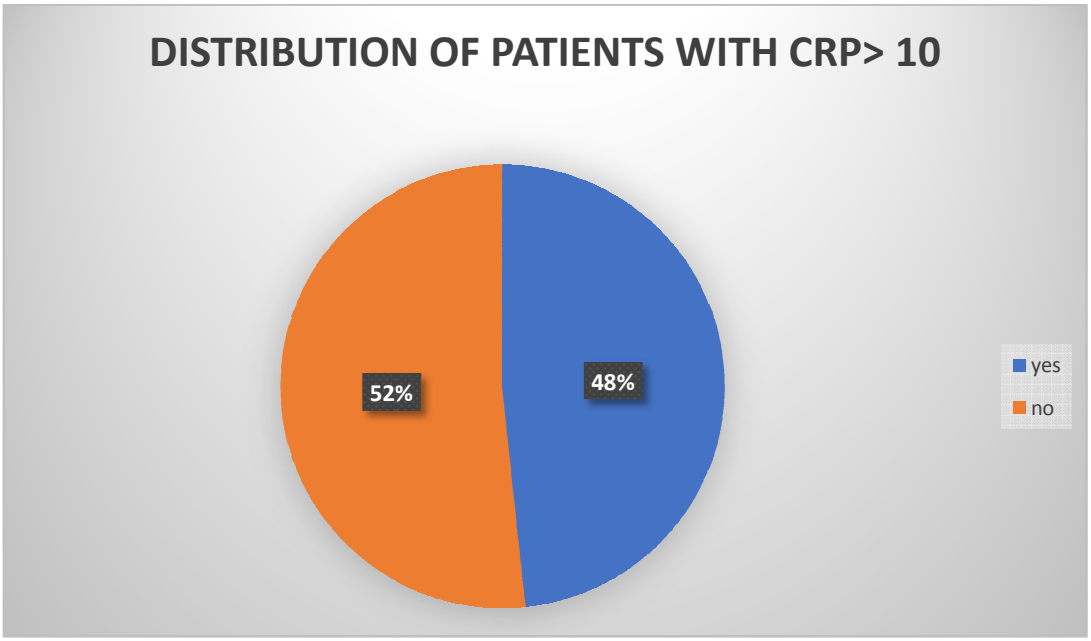

Figure 15 : Distribution of patients with CPR>10

Figure 15 shows out of 60 patients,(n=29) 48.33% were CRP >10 positive,(n=31)

51.67% negative

Distribution of Patient with X-ray air fluid level

Fluid level	frequency	Percentage
Yes	53	88.33
No	7	11.67

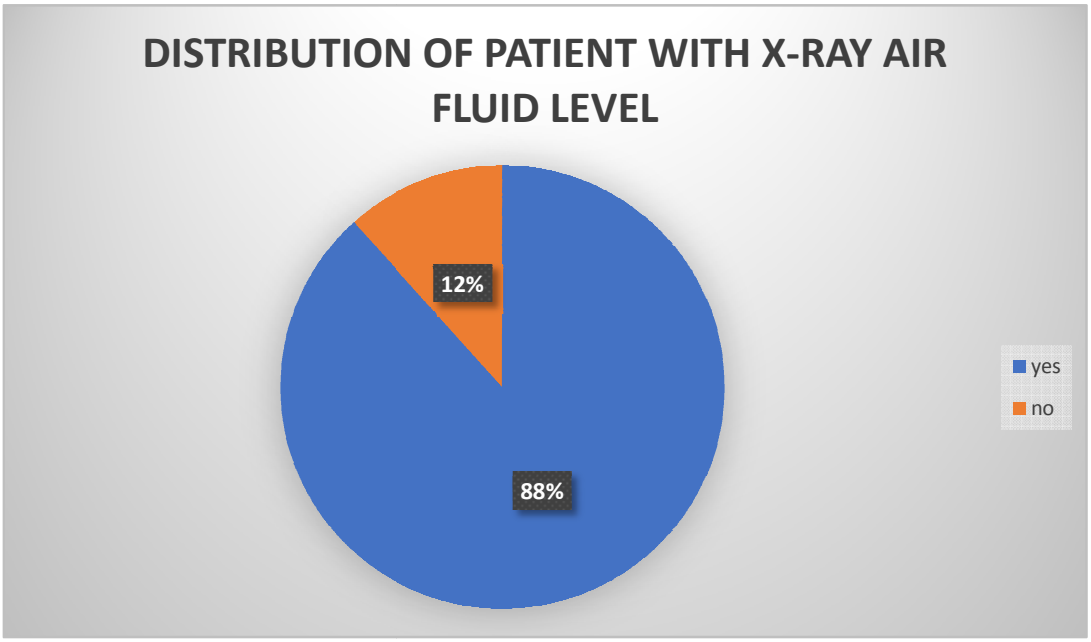

Figure 16 : Distribution of X-ray air fluid level

Figure 16shows out of 60 patients,(n=53) 88.33% were presented with multiple air fluid level on xray,only 11.67% (n=7) were not.

Distribution of Patients with CT reduced contrast enhancement

CT reduced contrast enhancement	frequency	Percentage
Yes	4	6.67
No	56	93.33

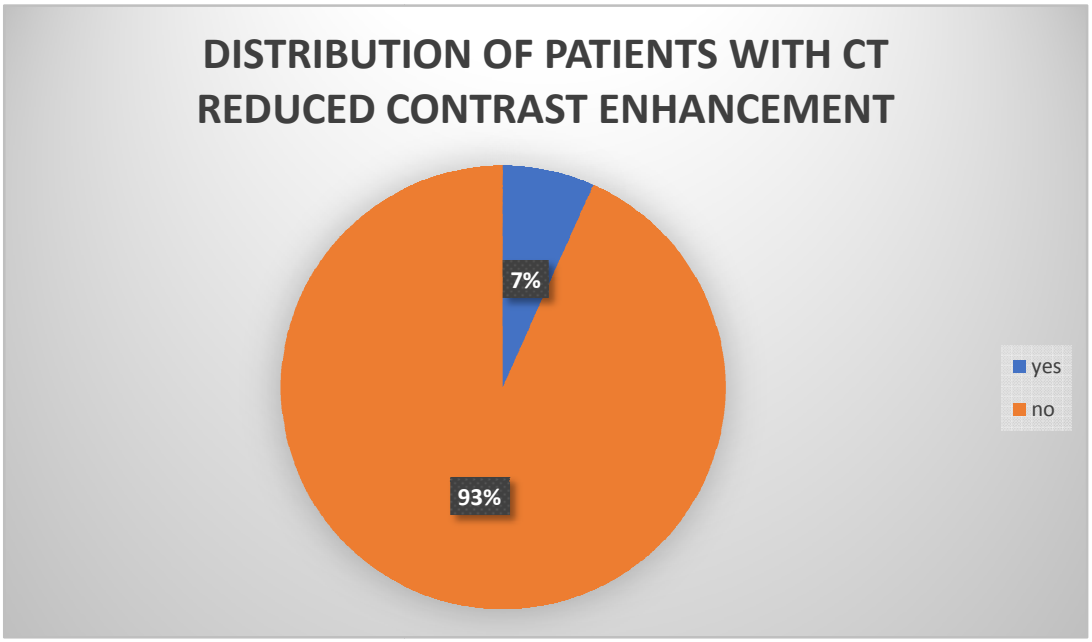

Figure17 : Distribution of patients with CT reduced contract enhancement

Figure 17shows out of 60 patients,only (n=4) 6.67% presented with CT reduced contract enhancement, (n=56) 93.33% presented were normal.

Distribution of Patients with history of previous surgery

previous surgery	frequency	Percentage
Yes	29	48.33
No	31	51.67

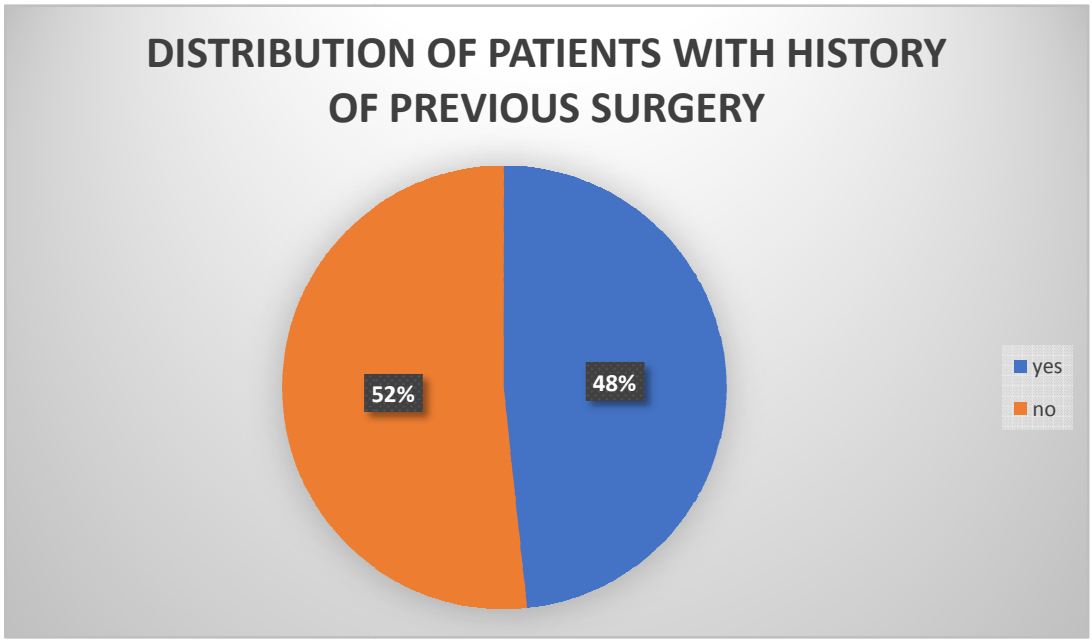

Figure 18 : Distribution with previous surgery .

Figure18 shows out of 60 patients ,(n=29) 48.33% were history of previous major abdominal surgery,(n=31) 51.67% were virgin abdomen.

Distribution of patients based on severity score

Score	Frequency	Percentage
1	9	15
2	11	18.33
3	16	26.67
4	15	25
5	9	15

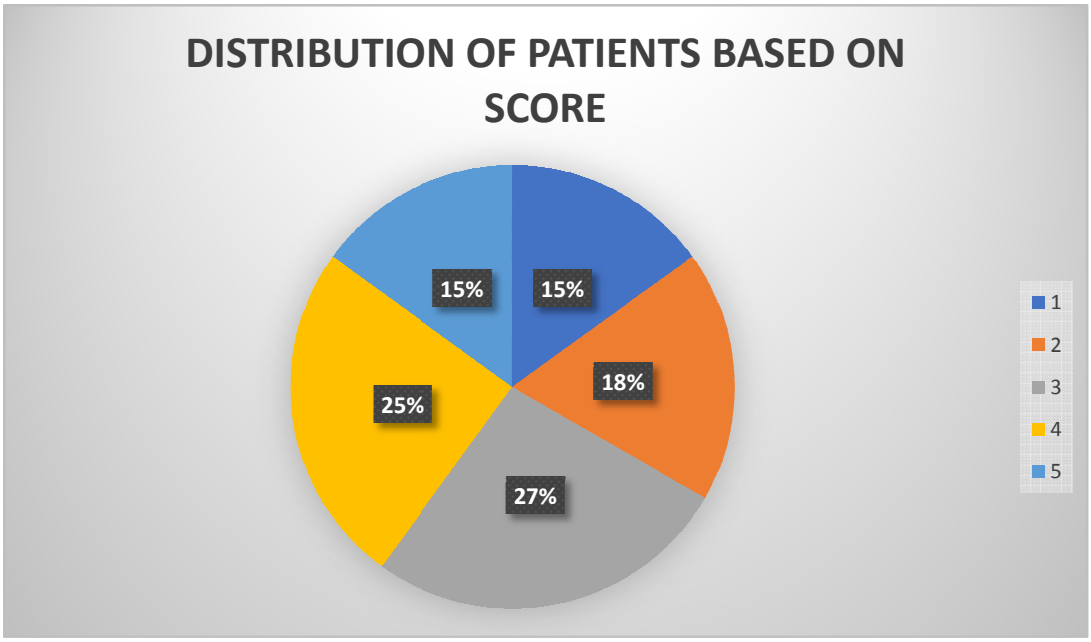

Figure 19 : Distribution of severity score

Figure19 shows distribution of severity score. Score 1 (n=9) 15%,score 2 (n=11) 18.33%,score 3 (n=16) 26.67%,score 4 (n=15) 25%,score 5(n=9) 15%.

Distribution of Patients based on etiology

Table2 : Distribution based on etiology

Etiology	frequency	Percentage
Adhesion	23	38.33
Appendicular perforation	1	1.67
Band	5	8.33
Incisional hernia	2	3.33
Inguinal hernia	5	8.33
Internal hernia	1	1.67
Mesenteric ischemia	4	6.67
Paralytic ileus	5	8.33
Phytobazar	2	3.33
TB abdomen	10	16.67
Umbilical hernia	1	1.67
Worm	1	1.67

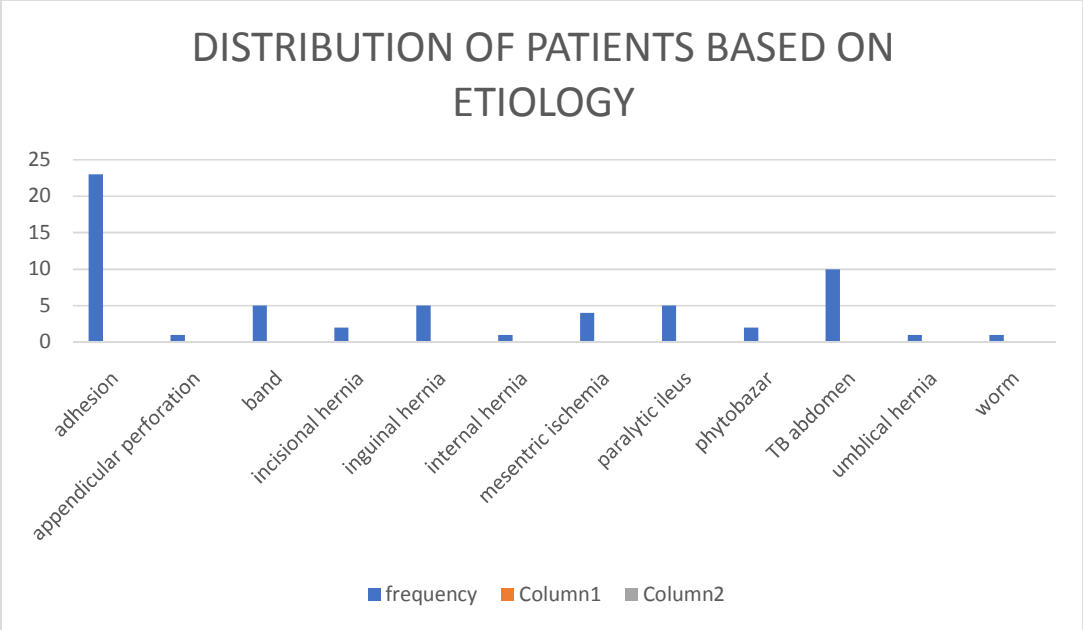

Table 2 shows distribution based on various etiology of small intestinal obstruction .one third of were (23) 38.33 due to adhesion,others (10) Tb abdomen,paralytic ileus (5) 8.33%,band (5) 8.33%,incisional hernia (2) 3.33%.

Distribution of Patients based on surgical procedure

Table 3 : Distribution of surgical procedure

Surgical procedure	Frequency	Percentage
Adhesiolysis	7	11.67
Ileostomy	1	1.67
Appendicectomy	1	1.67
Band release	5	8.33
Conservative	25	41.67
Enterotomy	2	3.33
Hernioplasty	6	10
Ileostomy	1	1.67
Resection	12	20.00

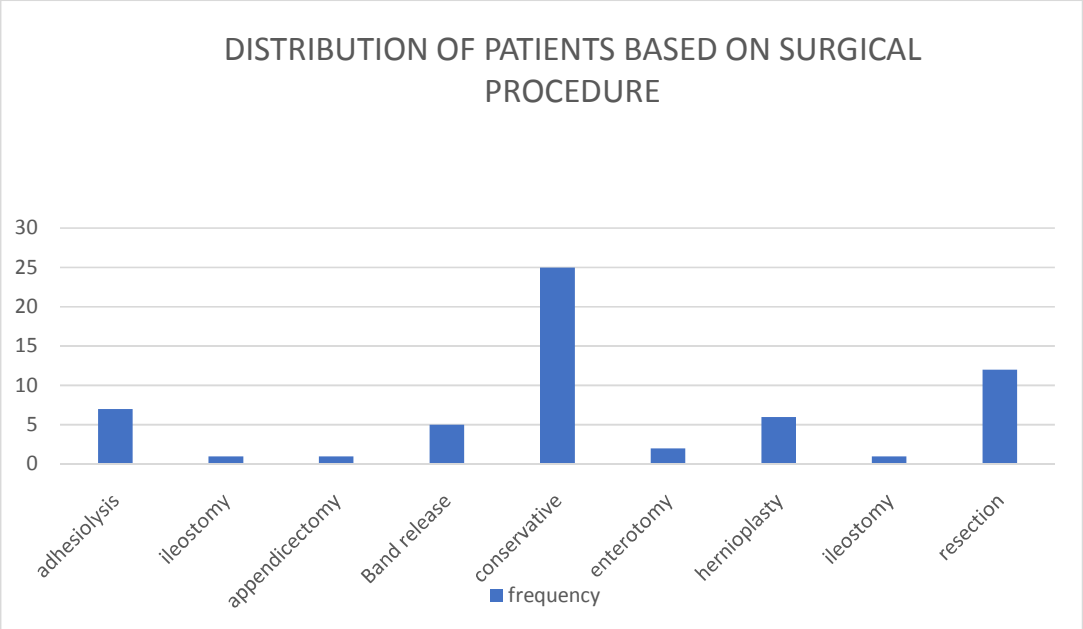

Table3shows comparsion of surgical mangenment on SBO . n=12 (20%)patient were went resection anastomosis, out of 23 adhesion (n=7) 11.67% were went for adhesiolysis,(n=16) 33.33% were managed conservatively.

Distribution of patients based on complications and outcome

Table 4: Distribution based on complication**.**

Complications	Frequency	Percentage
Brust abdomen	2	3.33
Death	3	5
Fistula	1	1.67
Recovered	51	85
Wound infection	3	5

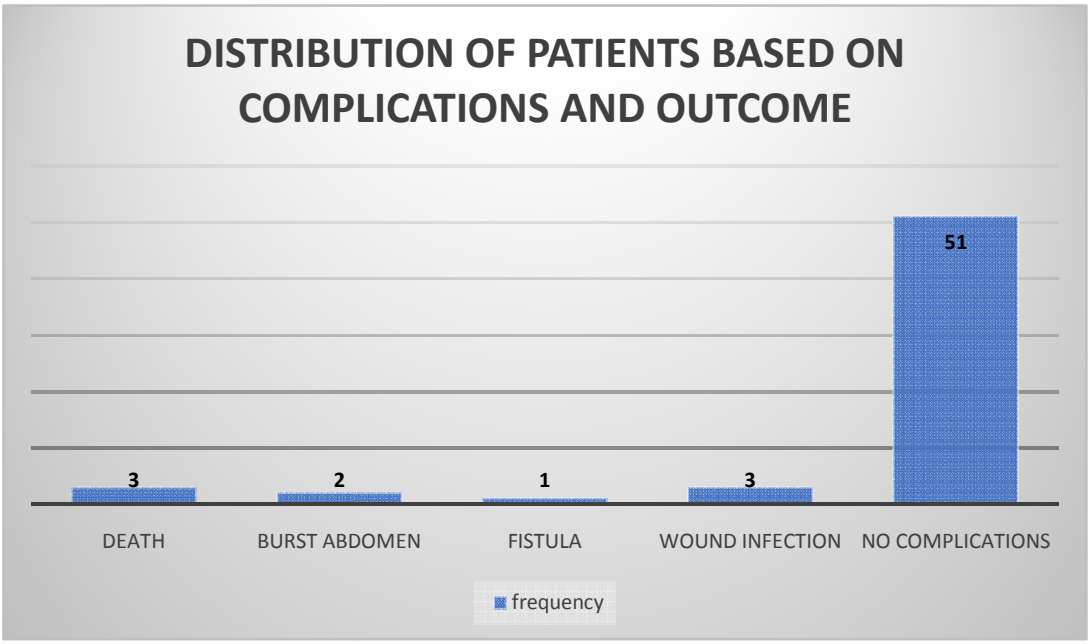

Table 4 shows distribution of complication and outcome on SBO.

No complication n=51 (85%),n=3(5%) Death, wound infection n=3 (5%).

Distribution of patients according to severity score on obstruction:

Table 5 : Distribution of severity score

Score	Surgery	Conservative	Total
<3	**0**	**20**	**20**
>3	**35**	**5**	**40**
Total	**35**	**25**	**60**

Table 5 shows distribution of patients in the study based on severity score and its management. 20 patients (33.33%) were score < 3 managed conservatively, while 35 patients (58.33%) were score >3 with surgery,only 5 patients (8.33%) were >3 managed with conservative.

Comparison of complications with age:

Table 6 :comparison of complications with age

Age category	Brust abdomen	Death	Fistula	Recovered	Wound infection
11-20	0	0	0	1	0
21-30	0	0	0	8	0
31-40	0	2	0	10	0
41-50	1	1	1	14	1
51-60	1	0	0	14	2
>60	0	0	0	4	0

Table 6shows comparison of complications with age . Age between 41-50 were more post operativecomplictions on SBO.

Comparison of complication with gender :

Table 7 : comparison of complications with age**:**

Gender	Brust abdomen	Death	Fistula	Recovered	Wound infection
Female	0	0	0	21	2
Male	2	3	1	30	1

Table 7 shows out of 60 patients n=37 (62%) male n= 6 (10%)were end upon high postoperative complications.

Comparison of complications with previous surgery

Table 8 : Comparison of complications with previous surgery

Previous surgery	Brust abdomen	Death	Fistula	Recovered	Wound infection
Yes	2	2	0	27	0
No	0	1	1	24	3

Table 8 shows Comparison of complications with previous surgery , n=2 patients were had brust abdomen and n=2 patients were died.

Comparison of score with complications:

Table 9 :Comparison of score with complications:

Score	Brust abdomen	Death	Fistula	Recovered	Wound infection
1	0	0	0	9	0
2	0	0	0	10	0
3	0	0	0	16	0
4	2	0	0	10	3
5	0	3	1	5	0

Table 9 showsComparison of score with complications: score >_ 4 were end upon high postoperative complications. P value 0.001(significant)

Comparison of etiology with complications:

Table 10 :Comparison of etiology with complications**:**

Etiology	Brust abdomen	Death	Fistula	recovered	Wound infection
Adhesion	0	0	1	1	0
Appendicular perforation	0	0	0	19	2
Band	0	0	0	0	1
Incisional hernia	0	0	0	5	0
Inguinal hernia	0	0	0	0	1
Internal hernia	0	0	0	0	5
Mesenteric ischemia	0	0	3	0	1
Paralytic ileus	0	0	0	5	1
Pytobazar	0	0	0	2	0
TB abdomen	2	0	0	8	0
Umbilical hernia	0	0	0	1	0
Worm	0	0	0	0	1

Table 10shows Comparison of etiology with complications,(n =3) patients died due to mesenteric ischemia . (n=2) patients with brust abdomen due to TB abdomen.

Comparison of surgical procedure with complications:

Table11 : Comparison of surgical procedure with complications

Surgical procedure	Brust abdomen	Death	Fistula	recovered	Wound infection
Adhesiolysis	0	0	1	5	0
Ileostomy	0	0	0	1	1
Appenicectomy	0	0	0	1	0
Band release	0	0	0	5	0
Conservative	0	0	0	24	0
Enterotomy	0	0	0	2	0
Hernioplasty	0	0	0	6	0
Ileostomy	0	0	0	1	0
Resection	2	3	0	5	2

P value 0.000(significant)

Table 11shows Comparison of surgical procedure with complications,

Total n=60 studied population n=6 patient of total n=9 post op complications were due to resection.

DISCUSSION

The present prospective observational study was carried out in our institute. 60 patients above 15 years admitted to the surgical wards with a provisional diagnosis of intestinal obstruction were taken for this study.

Age incidence

Small Intestinal obstruction although occurs in all age groups, the age spectrum in our clinical study was above 18 years. The study showed peak incidence in the age group 41-50 of 29.51% and 51-60 years of 27.87%

Sex Incidence

In this study male to female ratio is 1.6:1 .

Etiology

The cause of Small intestinal obstruction differs in different geographical locations. In present study of 60 cases of small intestinal obstruction, 38.33 % of the cases were due to adhesions .In this study, adhesion was the commonest cause of intestinal obstruction, which is comparable with the other study groups, Brooks and Butler with 23%,playfourth 54%.

Table 12

Cause	Present study	Brooks& butler	Playfourth
Adhesion	38.33%	23%	54%
Internal hernia	1.67%	25%	23%
Mesenteric ishaemia	6.67%	_	6%

Clinical features:

The common clinical feature of small intestinal obstruction are abdominal pain,vomiting,constipation,and abdominal distension.

Abdominal pain more than fourdays 81.67%

Constipation 60%

Distension 50%

Vomitig 91.67%

The finding of guarding on abdominal palpation cannot be ignored. Localised tenderness indicates impending or established ischaemia. The development of peritonism or peritonitis indicates impending or overt infarction and/or perforation. In this study 22 out of 22 patients with guarding were operated.

Laboratory investigation:

Total leukocyte count and C reactive protein are used.A TLC of more than 11,000 per cumm and a CRP of 10 mg/l or more was considered significant. Patients with bowel ischaemia often have marked leucocytosis**.**

In current study, most of the patients with a positive CRP value were operated on. Hence, CRP can be used as a severity indicator and is of value in deciding the timing of surgery**.**

Xray:

The erect Abdomen X-ray helps us in the diagnosis of intestinal obstruction as well as in differentiating the small bowel from large bowel obstruction. Multiple air fluid levels can be seen in small bowel obstruction whereas only gas shadows are seen in large bowel obstruction until the ileocecal valve is competent.in present study 88.33% shows multiple air fluid level.

Contrast enhanced Ct:

CT was used only when there was a diagnostic dilemma or to know the specific cause of obstruction.in present study 4 patient shows reduced contrast ienhancment on bowel werepreformed surgery

.

Management based on severity scoring system :

Every patient was given a score based on various parameters of the study. Whether the patient was conservatively managed or operated on was further analysed by using the scoring system. Maximum score was 5 and minimum 0. Score of 3 or more was significant. 20 patients (33.33%) having a score less than 3 were managed conservatively, while 35 patients (58.33 %) having a score of 3 or more where operated on.

A positive score of 3 or more had a sensitivity of 100 per cent and specificity 87.5 per cent. This allows early identification of strangulated SBO .

Sugical management:

The surgical management for the present study group includes release of adhesions, resection and anastomosis for many cases of bowel strangulation where the viability of the bowel was doubtful and for ischaemic bowel. Resection anastomosis was performed in 12 patients, adhesiolysis in 7, stoma creation in 1 and band release in 5, out of the 60 patients in our present study**.**

Complications:

Postoperative complications commonly occur in obstruction patients. Wound infection, burst abdomen, bowel fistula and death due to respiratory tract infection, septicaemia etc are a few common complications encountered. In the present study of 60 cases, complications like death occurred in 3 cases, wound infection 3, burst abdomen 2and bowel fistula one . Death occurred mostly due to septicaemia especially in mesenteric ischaemia cases, those that presented late and patients with other comorbid conditions**.**

CONCLUSION

SmallBowel obstruction continues to be one of the most common abdominal problems faced by general surgeons. Irrespective of the cause, it remains a major cause of morbidity and mortality.

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

Early recognition and aggressive treatment are crucial in preventing irreversible ischemia and transmural necrosis and thereby in decreasing mortality and long-term morbidity.

The evaluation of patients with suspected bowel obstruction for not only to confirm the diagnosis but also to determine the need for and timing of surgery.

Certain severity indicators and scoring systems can help to optimize this timing of surgery and prevent mortality

This study tries to use a severity scoring system to help identify the correct time to intervene in a case of smallintestinal obstruction. Most of the severity indicators have been found to be useful

Hence, this study emphasis the severity markers is necessary to prevent delay in operative intervention and thus prevent mortality and improve outcome of patients

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ANNEXURES

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01

INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK : CLINICAL STUDY ON SMALL INTESTINE OBSTRUCTION ON SEVERITY INDICATORS, ETIOLOGY, SURGICAL OUTCOME.

PRINCIPAL INVESTIGATOR : DR. T. RAJKUMAR
DESIGNATION : PG IN MS GENERAL SURGERY
DEPARTMENT : DEPARTMENT OF GENERAL SURGERY, GOVT. STANLEY MEDICAL COLLEGE.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 07.12.2018 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

Urkund Analysis Result

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https://www.slideshare.net/bineeshprakash/intestinal-obstruction-81991906
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https://msjonline.org/index.php/ijrms/article/download/3470/3237

Instances where selected sources appear:

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PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled **"CLINICAL STUDY ON SMALL INTESTINAL OBSTRUCTION BASED ON ETIOLOGY AND SURGICAL OUTCOME"** of the candidate **Dr. T. RAJ KUMAR** with **Registration Number 221711066** for the award M.S. Degree in the BRANCH-1 GENERAL SURGERY.I personally verified the urkund.com website for the purpose of Plagiarism check.

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- PAST HISTORY :
- CLINICAL FEATURE :
- RADIOLOGICAL FINDING AND CONTRAST STUIDES:
- SEVERITY SCORE:
- TREATMENT:
- OPERATIVE FINDINGS :
- COMPLICATION

CONDITION ON DISCHARGE

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INFORMED CONSENT

- **CLINICAL STUDY ON SMALL INTESTINAL OBSTRUCTION BASED ON ETIOLOGY AND SUGCIAL OUTCOME**

  PLACE OF STUDY: GOVT. STANLEY MEDICAL COLLEGE, CHENNAI

- NAME AND ADDRESS OF PATIENT:

  I, _____ have been informed about the details of the study in my own language.
- I have completely understood the details of the study.
- I am aware of the possible risks and benefits, while taking part in the study.
- I understand that I can withdraw from the study at any point of time and even then, I will continue to receive the medical treatment as usual.
- I understand that I will not get any payment for taking part in this study.
- I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.
- I know what I am supposed to do by taking part in this study and I assure that I would extend my full co-operation for this study.

Name and Address of the Volunteer:

Signature/Thumb impression of the Volunteer
Date:
Witnesses:
(Signature, Name & Address)
Date:
Name and signature of investigator:

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INFORMED CONSENT

DISSERTATION TOPIC:CLINICAL STUDY ON SMALL INTESTINAL OBSTRUCTION BASED ON ETIOLOGY,SEVERITY INDICATORS AND SURGICAL OUTCOME

PLACE OF STUDY: GOVT. STANLEY MEDICAL COLLEGE, CHENNAI

NAME AND ADDRESS OF PATIENT:

நான்,_____எனது சொந்த மொழியில் ஆய்வு விவரங்களை பற்றி தெரிவிக்கப்பட்டது.

நான் முற்றிலும் ஆய்வு விவரங்களை புரிந்து கொண்டேன். ஆய்வு பங்கெடுத்துக் கொண்டுள்ளநான், சாத்தியமான அபாயங்கள் மற்றும் பயன்களை அறிந்து இருக்கிறேன்.

நான் எந்த நேரத்திலும் ஆய்வு இருந்து திரும்ப முடியும் மற்றும் அதன் பின்னர், நான் வழக்கம் போல் மருத்துவ சிகிச்சை பெற தொடரும் என்று புரிந்து கொண்டேன்.

நான் இந்த ஆய்வில் பங்கு எடுத்து எந்த பணம் பெற முடியாது என்றும் அறிவேன்.

இந்த ஆய்வின் முடிவு, எந்த மருத்துவ இதழில் கிடைக்கும் என்றால், என் தனிப்பட்ட அடையாள வெளிப்படவில்லை வழங்கப்படும்.

நான் இந்த ஆய்வு பகுதியாக எடுத்து செய்ய வேண்டும் என்று எனக்கு நான் இந்த ஆய்வு என் முழுஒத்துழைப்பு நீட்டிக்க என்று உறுதியளிக்கிறேன்.

பெயர்மற்றும்தொண்டர்முகவரி:

தொண்டர்கையொப்பம் / பெருவிரல்ரேகை

நாள்:

சாட்சிகள்: ***(கையொப்பம், பெயர்மற்றும்முகவரி)***

நாள்:

பெயர் மற்றும் புலன் விசாரணை கையொப்பம்:

S.NO	NAME	AGE	SEX	IP NO	SYMPTOMPS				SIGNS	TLC	CRP	X RAY AIR FLUID LEVEL	CT REDUCED CONTRAST ENHANCEMENT	PREVIOUS SURGERY	SCORE	ETIOLOGY	SURGICAL PROCEDURE	COMPLICATION
					ABDOMINAL PAIN >4 DAYS	CONSTIPATION	DISTENSION	VOMITING	GUARDING	>11000	> 10mg/dl							
1	INBASEKAR	44	M	1822453	+	+	+	+	+	+	+	Y	N	Y	5	ADHESION	ADHESIOLYSIS	FISTULA
2	HARI	35	M	1829420	-	+	-	+	-	-	-	Y	N	N	1	TB ABDOMEN	CONSERVATIVE	RECOVERED
3	MUBINA	65	F	1825406	+	+	+	+	-	+	-	Y	N	Y	3	ADHESION	ADHESIOLYSIS/ILEOSTOMY	RECOVERED
4	KASIAMMAL	28	F	1825548	-	-	+	+	-	+	-	Y	N	N	2	PARALYTIC ILEUS	CONSERVATIVE	RECOVERED
5	RAMESH	38	M	1828454	+	+	+	+	+	+	+	N	Y	N	5	MESENTRIC ISCHEMIA	RESECTION	DEATH
6	KANNAN	32	M	1825143	+	+	-	+	-	-	-	Y	N	N	2	TB ABDOMEN	CONSERVATIVE	RECOVERED
7	ANDAL	55	F	1833461	+	+	-	+	-	+	-	Y	N	Y	3	UMBILICAL HERNIA	HERIOPLASTY	RECOVERED
8	DILI BABU	44	M	1829345	+	+	+	+	+	-	+	Y	N	N	4	TB ABDOMEN	RESERCTION	BRUST ABDOMEN
9	JOSEPH	58	M	1836541	-	+	+	+	-	-	-	Y	N	Y	1	ADHESION	CONSERVATIVE	RECOVERED
10	ASAITHAMBI	61	M	1835681	+	+	-	-	-	+	-	Y	N	N	3	INGUINAL HERNIA	HERNIOPLASTY	RECOVERED
11	FATHIMA	25	F	1839456	-	-	+	+	-	-	-	Y	N	N	1	PARALYTIC ILEUS	CONSERVATIVE	RECOVERED
12	RAJA	42	M	1839564	+	+	-	+	-	-	-	Y	N	Y	2	ADHESION	CONSERVATIVE	RECOVERED
13	PALANI	54	M	1845631	+	+	+	+	+	+	+	Y	N	N	5	TB ABDOMEN	ILEOSTOMY	RECOVERED
14	KUMAR	40	M	1845331	+	+	+	+	-	+	+	Y	N	N	4	BAND	BAND RELEASE	RECOVERED
15	GANAPATHI	55	M	1846732	+	-	-	+	-	+	-	Y	N	N	3	INGUINAL HERNIA	HERNIOPLASTY	RECOVERED
16	SULOCHANA	54	F	1846891	+	+	+	+	-	+	+	Y	N	Y	4	ADHESION	ADHESIOLYSIS	RECOVERED
17	PANDIYAN	36	M	1847934	+	-	+	+	+	+	+	Y	N	N	5	MESENTRIC ISCHEMIA	RESECTION	RECOVERED
18	KANCHANA	44	F	1856878	+	-	-	+	-	+	+	Y	N	N	4	BAND	BAND RELEASE	RECOVERED
19	MEENAKSHI	48	F	1856845	+	-	-	+	+	-	+	Y	N	Y	4	ADHESION	ADHESIOLYSIS	RECOVERED
20	RAEGINA	16	F	1854573	+	-	-	+	+	+	-	N	N	N	3	WORM	ENTEROTOMY	RECOVERED
21	MUTHU	45	M	1855671	+	-	-	+	-	-	-	Y	N	Y	2	ADHESION	CONSERVATIVE	RECOVERED
22	MARIAMMAL	58	F	1867340	+	+	+	+	+	+	+	Y	Y	Y	5	INTERNAL HERNIA	RESECTION	RECOVERED
23	HASINA	32	F	1875321	-	-	+	+	-	+	-	Y	N	Y	2	PARALYTIC ILEUS	CONSERVATIVE	RECOVERED
24	KARTHIK	23	M	1867342	+	+	-	+	-	-	+	Y	N	N	3	BAND	BAND RELEASE	RECOVERED
25	RAJADURAI	44	M	1865420	+	-	-	+	-	-	-	Y	N	N	2	TB ABDOMEN	CONSERVATIVE	RECOVERED
26	KUPPAN	54	M	1861894	+	+	+	+	+	-	+	Y	N	Y	4	ADHESION	ADHESIOLYSIS	RECOVERED
27	KASI	39	M	1861100	+	-	+	+	+	+	+	Y	Y	Y	5	MESENTRIC ISCHEMIA	RESECTION	DEATH
28	GANGA	48	F	1866239	+	+	-	+	-	+	+	Y	N	Y	4	INCISIONAL HERNIA	RESECTION	WOUND INFECTION
29	THIRUGYANAM	52	M	1876431	+	+	+	+	-	+	+	Y	N	Y	4	ADHESION	ADHESIOLYSIS	WOUND INFECTION
30	MOHAMED	37	M	1867001	+	+	-	+	-	-	-	Y	N	Y	2	ADHESION	CONSERVATIVE	RECOVERED
31	RATHIKA	52	F	1867430	+	+	-	+	-	-	-	Y	N	Y	2	ADHESION	CONSERVATIVE	RECOVERED
32	RAJI	32	F	1875421	+	-	+	+	-	-	-	Y	N	N	2	PARALYTIC ILEUS	CONSERVATIVE	RECOVERED
33	PARANTHAMAN	48	M	1876431	+	-	-	+	-	+	+	Y	N	N	4	INGUINAL HERNIA	HERNIOPLASTY	RECOVERED
34	PARTHASARATHY	53	M	1876541	+	+	+	+	+	-	+	Y	N	N	4	TB ABDOMEN	RESECTION	BRUST ABDOMEN
35	SYED	45	M	1877453	+	-	+	+	+	+	+	N	Y	N	5	MESENTRIC ISCHEMIA	RESECTION	DEATH
36	MICHEAL	22	M	1877832	+	+	-	+	+	+	-	Y	N	N	3	PENDICULAR PERFORATIC	APPENICECTOMY	RECOVERED
37	RAJAN	51	M	1877623	+	+	+	+	+	+	-	Y	N	Y	4	ADHESION	ADHESIOLYSIS	RECOVERED
38	RAJATHI	55	F	1878910	+	+	-	-	-	-	-	Y	N	Y	1	ADHESION	CONSERVATIVE	RECOVERED
39	BALU	32	M	1878765	+	+	-	-	-	-	-	N	N	N	1	TB ABDOMEN	CONSERVATIVE	RECOVERED
40	BANU	46	F	1878777	+	+	-	+	-	-	-	N	N	Y	1	ADHESION	CONSERVATIVE	RECOVERED
41	KATHIR	27	M	1934622	+	+	-	+	-	-	-	Y	N	N	2	TB ABDOMEN	CONSERVATIVE	RECOVERED
42	SYLAJA	55	F	1935689	+	+	+	+	-	+	+	Y	N	Y	4	ADHESION	RESECTION	WOUND INFECTION
43	JANANI	46	F	1937290	-	+	+	+	+	-	+	Y	N	Y	3	BAND	BAND RELEASE	RECOVERED
44	SAMSUN	60	M	1952409	+	+	+	+	+	+	-	Y	N	Y	4	ADHESION	ADHESIOLYSIS	RECOVERED
45	RAJENDRAN	63	M	1958013	-	+	-	+	+	+	+	N	N	N	3	INGUINAL HERNIA	HERNIOPLASTY	RECOVERED
46	SAGUNTHALA	35	F	1958721	-	-	+	+	-	-	-	Y	N	N	1	PARALYTIC ILEUS	CONSERVATIVE	RECOVERED
47	ESTHER	72	F	1958762	-	+	-	+	-	-	-	Y	N	Y	1	ADHESION	CONSERVATIVE	RECOVERED
48	SUBRAMANI	44	M	1957431	+	+	-	+	-	+	+	N	N	N	3	INGUINAL HERNIA	HERNIOPLASTY	RECOVERED
49	SUDKHAKAR	26	M	1958002	+	-	-	+	-	-	+	Y	N	N	3	TB ABDOMEN	CONSERVATIVE	RECOVERED
50	UNNI	43	M	1958541	+	-	-	-	-	-	-	Y	N	N	1	ADHESION	CONSERVATIVE	RECOVERED
51	MANOKAR	43	M	1959201	+	-	-	-	-	-	-	Y	N	Y	2	ADHESION	CONSERVATIVE	RECOVERED
52	BASKAR	52	M	1959452	+	-	+	+	-	-	+	Y	N	N	3	TB ABDOMEN	CONSERVATIVE	RECOVERED
53	KALIAMMAL	45	F	1967342	+	-	-	+	-	-	-	Y	N	Y	3	ADHESION	CONSERVATIVE	RECOVERED
54	KANNAN	38	M	1968421	+	-	-	+	-	-	+	Y	N	N	3	BAND	BAND RELEASE	RECOVERED
55	MALAR	28	F	1968460	-	+	+	+	+	+	+	Y	N	N	4	PYTOBAZAR	ENTEROTOMY	RECOVERED
56	YUSUF	55	M	1968340	+	-	+	+	-	+	-	Y	N	Y	3	ADHESION	CONSERVATIVE	RECOVERED
57	RANI	47	F	1968565	+	+	+	+	+	+	+	Y	N	Y	5	INCISIONAL HERNIA	RESECTION	RECOVERED
58	KANAGU	46	M	1975612	+	-	-	+	-	+	-	Y	N	Y	3	ADHESION	CONSERVATIVE	RECOVERED
59	JASMINE	29	F	1975551	-	-	+	+	+	+	+	Y	N	N	4	PYTOBAZAR	RESECTION	RECOVERED
60	SIVAN	55	M	1976124	+	+	+	+	+	+	+	Y	N	Y	5	ADHESION	RESECTION	RECOVERED