A STUDY ON ASSESSMENT OF SERUM LACTATE LEVELS IN INTESTINAL OBSTRUCTION

A DISSERTATION SUBMITTED TO

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulation for the award of the

Degree of M.S (general surgery)

BRANCH – I

DEPARTMENT OF GENERAL SURGERY

STANLEY MEDICAL COLLEGE AND HOSPITAL

TAMILNADU DR.M.G.R MEDICAL UNIVERSITY, CHENNAI

MAY- 2020

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This is to certify that dissertation "**A STUDY ON ASSESSMENT OF SERUM LACTATE LEVELS IN INTESTINAL OBSTRUCTION**" is a bonafide record of work done by **Dr. JOE PRAVEEN KUMAR. J**, in the Department of General Surgery, Stanley Medical College, Chennai, during his Post Graduate Course from 2017-2020. This is submitted in partial fulfillment 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.**

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DECLARATION

I **Dr. JOE PRAVEEN KUMAR . J** solemnly declare that this dissertation titled "A STUDY ON ASSESSMENT OF SERUM LACTATE LEVELS IN INTESTINAL OBSTRUCTION", 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.,** and **Prof. Dr. A.ANANDI M.S.**

This dissertation is submitted to the Tamilnadu Dr MGR Medical university, Chennai in partial fulfillment of the university regulations for the award of M.S,degree (General Surgery), branch – 1 examination to be held in May 2020.

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PLAGIARISM CERTIFICATE

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TITLE OF THE WORK : A STUDY ON ASSESSMENT OF SERUM LACTATE LEVELS IN INTESTINAL OBSTRUCTION.

PRINCIPAL INVESTIGATOR : DR. J. JOE PRAVEEN KUMAR,
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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 11.01.2019 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.
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6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

TABLE OF CONTENTS

INTRODUCTION

- Intestinal obstruction is one of the commonest clinical problems encountered in surgical practice.
- Strangulation is one of the grave complications of intestinal obstruction that requires emergency laparotomy.
- Ischemia, that complicates 7 to 42% of bowel obstructions, significantly increases mortalities associated with bowel obstruction
- Time is an essence, with an earlier diagnosis of strangulation favoring increased patient's survival
- The diagnosis of strangulation is primarily clinical with a sudden onset of pain i.e. continuous rather than colicky, the early appearance of shock, and the presence of fever, tachycardia, marked abdominal tenderness, guarding, rebound tenderness and a tender abdominal mass are all in favor of the diagnosis of strangulation.
- Various biochemical markers such as serum tumor necrosis factor α, C reactive protein, interleukin 6, lactate, intestinal fatty acid binding protein(I-FABP), creatine kinase B, isoenzymes of lactate dehydrogenase have been studied
- Therefore, studies investigating the role of biomarker in predicting strangulation in patients of acute bowel obstruction are needed.

- Lactic acid is the normal endpoint of the anerobic breakdown of glucose in the tissues.

- L-lactate and D-lactate are the two optical isomeric forms of lactate

- L – lactate is the final end product of anaerobic glycolysis. During this process it is formed out of pyruvic acid by the enzyme lactate dehydrogenase (LDH). During ischemia the cells will start anaerobic dissimilation and the serum lactate rises

- D-lactate is not produced in mammalian tissue, but it is detected in a situation of an abnormal proliferation of enteral bacterial flora due to mucosal injury following mesenteric ischemia.

AIMS AND OBJECTIVES

The aim of this prospective observational study is to evaluate the role of Serum Lactate as a marker of strangulation in bowel obstruction

MATERIALS AND METHODOLOGY

DESIGN OF STUDY

Non randomized prospective observational study

SAMPLE SIZE

50 cases admitted in Emergency General Surgery ward with clinical suspicion of intestinal obstruction for a period of 6 months (February 2019 to July 2019)

INCLUSION CRITERIA:

- ✓ Patients of age group > 12 years and both sex
- ✓ Patients with clinical suspicion of acute intestinal obstruction

EXCLUSION CRITERIA

- × Patients with co-existing medical illness such as chronic kidney disease, diabetes mellitus, any cardiac ailment and coagulopathies as they lead to false positive results.
- × Patients with any intraoperative finding apart from simple or strangulated bowel obstruction

METHODOLOGY

- Patients with clinical suspicion of acute intestinal obstruction who are admitted in Department of General Surgery are chosen (by criteria mentioned above), who may undergo laparotomy.

- Written and informed consent will be sought.

- Blood samples are taken at the time of presentation in the emergency within 20 minutes of their arrival

- By eliciting comorbid history and its duration

- Blood sample will be collected and sent to the Department of Biochemistry for separation of sera by centrifugation and storage in sterile vials at -20 degree Celsius and measurement of serum lactate levels are done

- Normal serum lactate value is less than 2mmol/L

- Cut off value for strangulation is 4mmol/L

- Values between 2-4mmol/L indicates strong suspicion

- Assessment and comparison of serum lactate levels in various outcomes of intestinal obstruction is done

REVIEW OF LITERATURE

DEFINITION

Bowel obstruction is defined by the lack of aborad transit of intestinal contents, regardless of etiology. Bowel obstruction may involve only the small intestine (small bowel obstruction), the large intestine(large bowel obstruction),or both via systemic alterations in metabolism, electrolyte balance or neuroregulatory mechanisms (generalised ileus). Traditionally surgeons perspective of a bowel obstruction represents a mechanical obstruction that is due to physical stenosis or occlusion of the intestinal lumen. In the broader context, however, ineffective motility, without any physical obstruction, causes a functional obstruction or ileus of the intestine. Furthermore, intestinal obstruction can be classified based on duration of presence(acute vs chronic obstruction), extent(partial vs complete),type of obstruction(simple vs closed loop) and risk of bowel compromise(incarcerated vs strangulated). Bowel obstruction continues to be one of the most common intraabdominal problems faced by general surgeons. In a 2010 global burden of disease study, bowel obstruction and ileus were responsible for 2.1 % deaths, 54 years of life lost and 54 disability adjusted life years per 100000 population, respectively, second only to peptic ulcer disease for all abdominal conditions for each of these parameters. Early recognition and aggressive treatment are crucial in preventing irreversible ischemia and transmural necrosis, thereby decreasing mortality and long term morbidity. Despite multiple recent advances in diagnostic imaging and marked advances in our treatment armamentarium, intestinal obstruction remain a significant surgical problem given the lack of treatment options to manage adhesions, hernias and malignancies.

MECHANICAL BOWEL OBSTRUCTION

Mechanical obstruction is defined as a physical narrowing or occlusion of the intestinal lumen. This blockage may be intrinsic or extrinsic to the wall of the intestine or secondary to luminal obstruction arising from the intraluminal contents. Partial obstruction implies that the intestinal lumen is narrowed and some intestinal content can transit distally. In the presence of a complete obstruction, the lumen is obliterated, and no intestinal content can pass beyond the point of obstruction. The risk of strangulation that is, vascular compromise of the intestine

increases markedly in the presence of a complete obstruction, especially when caused by extraluminal etiology such as hernia defect or an adhesive band compressing the small bowel mesentery. Accordingly, complete obstruction can be categorised further as simple, closed loop and strangulated obstruction. A simple obstruction has no associated vascular compromise and the intestine can be decompressed proximally. Closed loop obstruction occurs when both ends of the involved intestinal segment are obstructed(eg. Volvulus or compressive adhesive band) and results in increased intraluminal pressure secondary to increased intestinal secretion and accumulation of fluid in the involved intestinal segment. Closed loop obstruction carries a substantial risk of vascular compromise and irreversible intestinal ischemia of the involved bowel and thus requires emergent operative attention. Finally, strangulation occurs when the blood supply to the affected intestinal segment is compromised leading to focal or segmental transmural necrosis. The affected segment may involve only a portion of the bowel wall compressed by a tight adhesive band or an entire intestinal segment as occurs with a strangulated hernia or a closed loop. If viability of the bowel is maintained after release of the obstruction, strangulation can be reversed(reversible strangulation obstruction). In contrast, irreversible strangulation occurs if the vascular compromise has caused irreversible transmural necrosis whether or not the strangulation is relieved. All irreversible strangulated obstruction start as reversible strangulated obstructions and thus early diagnosis is paramount in rescuing the compromised intestine.

TABLE 1: MECHANICAL BOWEL OBSTRUCTION

LESIONS EXTRINSIC TO THE INTESTINAL WALL	LESIONS INTRINSIC TO THE INTESTINAL WALL
Adhesions Postoperative Congenital Postinflammatory **Hernia** External abdominal wall (congenital of acquired) Internal Incisional **Congenital** Annular pancreas Malrotation(rotational abnormality) Omphalomesenteric duct remnant **Neoplastic** Carcinomatosis Extraintestinal neoplasm **Inflammatory** Intra abdominal abscess Starch peritonitis **Miscellaneous** Volvulus Gossypiboma Superior mesenteric artery syndrome	**Congenital** Intestinal atresia Meckel's diverticulum Duplications/cysts **Inflammatory** Crohn's disease Eosinophilic granuloma Infections Tuberculosis Actinomycosis Complicated diverticulitis Appendicitis **Neoplastic** Primary neoplasms Metastatic neoplasms **Miscellaneous** Intussusception Endometriosis Radiation enteropathy/stricture Intramural hematoma Ischemic stricture **Intraluminal/obturator obstruction** Gallstone Enterolith Phytobezoar Parasite infestation Swallowed foreign body(magnets,illicit drug mules,sharp objects that perforate the bowel,etc)

FUNCTIONAL BOWEL OBSTRUCTION OR ILEUS

Functional obstruction or ileus occurs when the bowel, small or large, fails to propel content distally in the absence of a mechanical obstruction. The pathophysiology of ileus involves electrolyte disturbances,impaired neuroregulatory innervation,imbalanced hormonal input and other less common causes. The most common form of functional obstruction is postoperative ileus,because it is present to some extent after nearly all intra abdominal operative procedures. A group of rare,chronic,progressive,gastrointestinal PSEUDO OBSTRUCTIONS are related either to hereditary or acquired visceral myopathies,neuropathies or a poorly understood disruption of myoneural coordination of organized contractile activity. Generally the small bowel recovers effective motor function within hours after an abdominal operation and in fact,transient focal intestinal peristalsis is often visualised during an abdominal operation. In contrast,stomach regains motor function 24 to 48 hours after an operation leading to delayed gastric emptying and the colon exhibits the slowest recovery response and may take 3 to 5 days to recover effective propulsive activity postoperatively.

EARLY POSTOPERATIVE (MECHANICAL) BOWEL OBSTRUCTION

Early postoperative bowel obstruction is defined as bowel obstruction occurring within the first 6 weeks postoperatively. This type of intestinal obstruction represents a distinct clinical entity with a unique pathophysiology and it should be differentiated from both the classic mechanical bowel obstruction and postoperative ileus. The formation of acute adhesions is the responsible cause in 90% of early postoperative bowel obstructions necessitating surgical management. Other causes include internal herniation,fascial herniation after laproscopic surgery,intra abdominal abscess,intramural intestinal hematoma and anastomotic edema or leak.

TABLE 2 : FUNCTIONAL BOWEL OBSTRUCTION,ILEUS,PSEUDO OBSTRUCTION

INTRA ABDOMINAL CAUSES	EXTRA ABDOMINAL CAUSES
Intraperitoneal Peritonitis(chemical,infections) Intra abdominal abscess Contained anastomotic leak Postoperative Chemical: Gastric juice Bile Blood Autoimmune: Serositis Myositis Vasculitis Neuropathy Intestinal ischemia: Arterial or venous Sickle cell disease **Retroperitoneal problems** Urolithiasis Pyelonephritis Metastasis Pancreatitis Retroperitoneal trauma/hematoma	**Thoracic problems** Myocardial infarction Severe congestive cardiac failure Pneumonia Thoracic trauma **Metabolic abnormalities** Electrolyte imbalance Sepsis Lead poisoning Porphyria Hyperglycemia/ketoacidosis Hypothyroidism Hypoparathyroidism Uremia **Medicines** Opiates Anticholinergics Alpha adrenergic agonists Antihistaminics Psychotropic drugs Catecholamines **Miscellaneous** Acute spinal cord injury Pelvic fracture Head trauma Chemotherapy Radiation therapy Hip arthroplasty Renal transplantation Acute megacolon(ulcerative colitis,clostridium difficile infection)

EPIDEMIOLOGY

In recent decades, the overall incidence of small bowel obstruction has been stable over time as noted by a study that examined the incidence from 1988 to 2007 when it ranged from 579 to 654 diagnoses for bowel obstruction per 100000 population. The etiology and frequency of obstruction was altered markedly throughout the 20th century when repair of hernias became commonplace and thus the etiology of bowel obstruction related to incarceration in a hernia defect decreased and was replaced by adhesive obstruction as the most common cause of bowel obstruction. In the underdeveloped world, bowel obstruction still manifests with a clinical picture resembling that found in the early 20th century in western societies with incarcerated hernias leading the list in frequency. The wider application of minimally invasive surgical procedures with fewer adhesions may decrease the frequency of bowel obstruction secondary to postoperative adhesions, particularly in cholecystectomies and hysterectomies. Obstetric, gynecologic and other pelvic surgical procedures represent important etiologies for the development of postoperative adhesions, therefore, a slightly greater frequency of bowel obstruction is observed in women. About 80% to 90% of bowel obstructions occur in the small intestine and the other 10%to 20% occur in the colon. Colorectal cancer is responsible for 60% to 70% of all large bowel obstructions, while diverticulitis and volvulus account for majority of the remaining 30%. In contrast, small bowel obstruction is most commonly attributed to adhesions, hernias or neoplasms. Bowel obstruction results in substantial overall mortality and morbidity. Mortality rates range from upto 3% for simple obstructions to as great as 30% when there is vascular compromise or perforation of the obstructed bowel. Bowel obstruction is frequently a recurrent problem adding to the overall morbidity of an operation or even repetitive successful nonoperative management. Recurrence rates vary according to method of management (conservative or operative). Intestinal obstruction recurs in about 12% of patients after a successful primary conservative treatment and in 8% to 32% of patients after operative management for adhesive bowel obstruction.

PATHOPHYSIOLOGY

Mechanical bowel obstruction results in numerous alterations of the normal intestinal physiology, including motility and absorption. The pathophysiology of bowel obstruction remains incompletely understood despite numerous investigations both clinically and experimentally. Bowel distension, decreased absorption, intraluminal hypersecretion, and alterations in motility are found universally, but the mechanisms mediating these relatively dramatic pathophysiologic derangements remain unclear. In addition, bowel obstruction is accompanied by considerable disruption of mechanisms of neural and hormonal control, the type and quantity of endogenous bacterial flora, and the innate immunity of the gut.

The older, classic literature addressing the pathophysiology of bowel obstruction considered a decrease in blood flow as the sentinel event leading to most of the observed pathophysiologic changes. More recent experimental work, however, suggests that an increase in blood flow in association with an intense intramural inflammatory reaction and subsequent mucosal production of reactive oxygen species mediate many of the pathophysiologic changes observed in the early phase of bowel obstruction.

DISTENSION, ABSORPTION AND SECRETION

Bowel distension is a characteristic, fundamental, and constant pathophysiologic response to mechanical bowel obstruction. Accumulation of swallowed air is responsible for much of the small bowel distension in the early phases of obstruction. As would be expected, intramural gas consists of approximately 75% nitrogen in the obstructed bowel. Fermentation of sugars, production of carbon dioxide from the blood are other sources of gas in early obstruction. Dilation and inflammation of the bowel wall cause accumulation of activated neutrophils and stimulation of resident macrophages within the muscular layer of the bowel wall, impairing the secretory and motor processes by release of reactive proteolytic enzymes, cytokines and other locally active substances. Local release of nitric oxide, a potent inhibitor of smooth muscle tone and contractility by the inflammatory response, aggravates intestinal dilation through inhibition of contractile activity. Notably, a correlation

between the amount and activity of nitric oxide synthesis, and the severity of intestinal dilation observed exists. Furthermore, experimental data demonstrate a relationship between distension and the intramural production of reactive oxygen metabolites. In addition to disrupting gut motility, these metabolites also modulate permeability of the vasculature and the gut mucosa.

Along with the intraluminal accumulation of gas, the bowel also has a secondary decrease in net absorption resulting in the addition of water and electrolytes into the lumen during the first 12 hours of small bowel obstruction. By 24 hours, intraluminal water and electrolytes accumulate more rapidly because of a further decrease in absorptive flux; this decrease in net absorptive reflux occurs via stimulation of a concomitant increase in net intestinal secretion (secretory reflux). These changes are caused by increased permeability due to secondary mucosal injury resulting in intraluminal neural or systemic humoral/hormonal mechanisms aggravate this upregulation of unidirectional secretory flux also remain likely but poorly investigated or explained.

This net secretion of fluid into the lumen of the obstructed bowel is exacerbated further by the accumulation of intraluminal bacteria derived toxins, bile acids, prostaglandins, vasoactive intestinal polypeptide and mucosa derived oxygen free radicals. With a more chronic obstruction, bacterial proliferation occurs in the lumen, further disrupting absorption, secretion and mucosal integrity. The decrease in the absorptive capacity and increase in secretion lead to important fluid loses(enterosecretion) that my result in profound dehydration. Although the intestinal wall distal to the obstruction maintains relatively normal function, the inability of luminal content to reach the unobstructed small bowel and colonic absorptive surface is an important component of overall dehydration.

INTESTINAL MOTILITY

In an attempt to propel intraluminal contents past the obstruction, intestinal contractile activity increases in the early phase of bowel obstruction, probably in large part related to the intestinal distention. Later in the course of the bowel obstruction, however, contractile

activity decreases likely secondary to a relative hypoxia of the intestinal wall and enhanced intramural inflammation. Although the exact mechanisms have not been described adequately, these responses may be similar to the changes found early after an abdominal operation, again related to the inflammation of the intestinal wall. Some investigations have suggested that the alteraltions in intestinal motility are secondary to a disruption of the normal autonomic parasympathetic (vagal) and sympathetic splanchnic innervation, while others relatele these changes more to a local effect of inflammation of the intestinal wall.

Splanchnic innervation has been the focus of extensive research, and especially so in the pathogenesis of paralytic ileus. Chemical sympathectomy has been successful in ameliorating ileus in several experimental models. Other pharmacologic approaches have focusses on blocking the neural sympatholytics and cholinergic agonists. Still other experimental approaches have been designed to prevent or inhibit the inflammatory response that accompanies the "physiologic" response to celiotomy or the abnormal inflammatory response accompanying generalised ileus. More recent investigative attention has been directed to impaired intestinal motility in the face of opioid administration postoperatively. The mu receptor antagonist alvimopan appears to inhibit opioid induced intestinal impairment and enhance motility.

CIRCULATORY CHANGES

The resident and transient flora of the upper small intestine consists mainly of gram positive, facultative, anerobic organisms in small concentrations, usually less than 10^6 colonies/ml. The bacterial count increases distally to about 10^8 colonies/ml in the distal ileum. In addition to this increase in number of bacteria, a change of flora to primarily coliform and anaerobic organisms is apparent. In the presence of obstruction, however, a rapid proliferation of bacteria occurs proximal to the point of obstruction, consisting

predominantly of faecal type organisms. The proliferation of this faecal flora, proportional to the duration of obstruction, reaches a plateau of 10^9 to10^10 colonies/ml after 12 to 48 hours of an established obstruction. The bowel distal to the obstruction tends to maintain its usual bacterial flora until the onset of a generalised inflammatory provoked ileus, resulting only then in bacterial proliferation distal to the point of obstruction. Bacterial toxins play an important role in the mucosal response to bowel obstruction. Experiments in germ free dogs with mechanical bowel obstruction have shown that net intraluminal accumulation of fluid and electrolytes does not occur and net absorption continues.

Experiments, primarily in rodents, have shown that bacterial translocation occurs secondary to impairment of the barrier function of the intestinal mucosa if bowel obstruction persists. The disruption of the mucosal barrier function begins early after the onset of bowel obstruction. The cellular response to obstruction is multifactorial. In the enterocyte, the endoplasmic reticulum dilates as early as 4 hours after onset of bowel obstruction. Mitochondrial swelling, focal epithelial necrosis, intracellular ballooning, an degenerative changes in the nucleus of epithelial cells (apoptosis) have been demonstrated as early as 6 to 12 hours after the onset of obstruction in this experimental model. The mucosal defence is compromised further by a decrease in perfusion of the intestinal wall. The loss of mucosal integrity allows luminal bacteria to both translocate as well as to invade the submucosa and enter the systemic circulation via the portal venous and lymphatic systems. Several bacterial substances can be retrieved from peritoneal fluid and lymphatic channels even in the absence of perforation. In the recent model, bacteria can be cultured from the spleen, liver and mesenteric lymph nodes, indicating a marked increase in bacterial translocation. Concomitant with bacterial translocation, lymph fluid contains numerous bacterial proteins and lipoproteins that further disrupt normal gut function.

The demonstration of bacterial translocation in these elegant studies with rodent models led to the erroneous assumption of the existence of a similar bacterial translocation in humans. Reproducible documentation of true bacterial translocation in man is notably lacking, and existence of a true bacterial translocation seems unlikely. Several studies have unsuccessfully tried to document the presence of bacteria in intra-abdominal lymph nodes, spleen, liver and even lymphatics. In contrast, more recent work has shown that lipopolysaccharide and other inflammatory vasoregulatory mediators, but not bacteria, can be recovered from the mesenteric lymphatics. The eventual drainage of these inflammatory substances into the systemic circulation may lead both to the systemic manifestation of sepsis and further disruption of the mucosal barrier function.

The change in the intraluminal bacteriology in simple intestinal obstruction is important clinically, because it markedly increases the risk of infectious complications, especially if an intestinal resection is required or if an inadvertent enterotomy occurs with intra peritoneal contaminated of highly inoculated, bacterial-laden enteric contents. In contrast, with irreversible strangulation obstruction, a myriad of local and systemic alterations, such as release of cytokines and increased formation of reactive oxygen intermediate, can promote the systemic inflammatory response syndrome and progress to multiple organ dysfunction with all its consequences.

ETIOLOGY

ADHESIONS

Adhesions are inflammatory-derived, fibrous attachments of connective tissue that adhere to organ surfaces. Adhesions may be congenital or acquired through post inflammatory and/or postoperative processes. Congenital or inflammatory adhesions are less frequent causes of bowel obstruction than post-operative adhesions, except in certain circumstances

such as rotational disorders(malrotation) or a persistent urachus. The leading cause of small bowel obstruction in western societies is postoperative adhesions, which are responsible for 40% to 80% of bowel obstructions in hospitalized patients. This wide variation in incidence of adhesive obstruction varies with referral patterns, community practice settings, racial cultures, and regional preferences.

Adhesion formation is nearly universal after celiotomy and starts within hours of an intra-abdominal operation, since the inflammatory phase is the first requirement for adhesion development. While the exact pathogenesis of adhesion formation remains incompletely understood, experts agree that adhesion formation is a surface event associated with peritoneal injury. This inciting trauma triggers a local inflammatory response leading to activation of the complement and coagulation cascades along with exudation of fibrinogen rich fluid; the full establishment of this fibrinous inflammatory response is present 5 to 7 days after the trauma of a celiotomy. Recent findings have identified the presence of sensory nerve fibers in human peritoneal adhesions, suggesting that these structures may be capable of conducting pain or other neural responses.

Peritoneal healing(mesothelialisation) appears to differ from the response in skin, where re-epithelialisation occurs from the periphery inward. In the peritoneum, operative or traumatic defects are reperitonealised by implantation of mesothelial cells in multiple areas of the defect. This mesothelialisation takes place quite rapidly, and resurfacing is often complete by 2 to 5 days after the injury, depending upon the local conditions.

Normal peritoneal healing, however is a complex, interrelated programmed inflammatory process. The initial response involves infiltration of the wound area with polymorphonuclear leukocytes and lymphocytes. During the ensuing 24 to 36-hours, circulating and local macrophages are recruited by various chemokines. By 48 hours, a fibrin scaffold overlying

the defect has been established, covered by macrophages and a few mesothelial cells. These mesothelial cells then coalesce to fully cover the defect over the next 2 to 5 days. Fibroblasts and other mesenchymal cells populate the underlying fibrin scaffold and begin to lay down a basement membrane. By 8 to 10 day, a single layer of mesothelial cells resting on a continuous basement membrane has been established. This process describes the simple resurfacing of an uncomplicated peritoneal defect.

In comparison to the previously described physiologic process of normal peritoneal healing, adhesion formation is a pathologic process. Studies suggest that adhesion form in response to the initial fibrin gel matrix in response to the local, inflammatory microenvironment. This fibrin gel matrix consists of numerous types of cells such as platelets, mast cells and erythrocytes, in conjunction with surgical debris, non-viable tissue, foreign bodies and possibly bacteria. The resultant spectrum of fibro inflammatory changes between physiologic mesothelial healing versus pathologic adhesion formation varies not only among individuals but is dependent also on many other conditions such as inflammation,infection,devitalised tissue and foreign bodies.

If the fibrin gel allows apposition of adjacent surfaces, a band or bridge mahy form(ie, an adhesion). This process of adhesion formation is dynamic, consisting predominantly of macrophages early, butby 2 to 4 days, larger strands of fibrin begin to appear along with fibroblasts. By 5 days, distinct bundles of collagen are apparent, and the fibroblasts begin to form a syncytium within the matrix. These cells predominate thereafter, and eventually the fibrin matrix and cellular elements are replaced by a vascularised, granulation type tissue containing macrophages, fibroblasts, giant cells, and a rich vascular supply. Eventually the surface of the adhesions is covered by a mesothelial layer, but only after formation of the underlying fibrous scar leading to surface opposition and transperitoneal fibroinflammatory bands of varying severity and extent.

An important factor in the spectrum of adhesion formation that contributes to the risk of future adhesive bowel obstruction is the type of surgical procedure performed. Operations involving structures in the inframesocolic compartment and those in the pelvic region such as colonic, rectal, and gynaecological procedures impart the higher risk. Open procedures, use of gloves containing starch granules, gallstone spillage during cholecystectomy, and separate peritoneal closure were also correlated with adhesive SBO in a review article. Adhesive bowl obstruction may occur at any time postoperatively after a celiotomy, with reports ranging as early as within the first postoperative month to more than eight decades after the index operation. A study by Menzies and Ellis found that about 20% of adhesive bowel obstructions occur within 30 days after the initial celiotomy, about 20% occur between 1 and 12 months postoperatively, another 20% tend to occur between 1 and 5 years postoperatively, with the remainder(-40%) occurring after 5 years. A Norwegian study of patients requiring as operation for adhesive bowel obstruction found that most episodes of recurrent bowl obstruction occurred within 5 years after the previous episode, but the risk of obstruction persisted for more than 20 years after a prior episode reaching an incidence as great as 29% at 25 years. Therefore, a common predisposition to adhesive obstruction is the presence of a prior episode of adhesive obstruction. Numerous surgical attempts to decrease or prevent the development of postoperative adhesions have been reported and are discussed subsequently. The literature on pharmacologic prophylaxis against postoperative adhesion formation is extensive and riddles with numerous false claims of benefit. Suffice it to say that no reliable or truly effective pharmacologic agent has been developed to augment mesothelialisation and prevent adhesion formation. Several proprietary barrier products of variable efficacy have been developed and will be discussed.

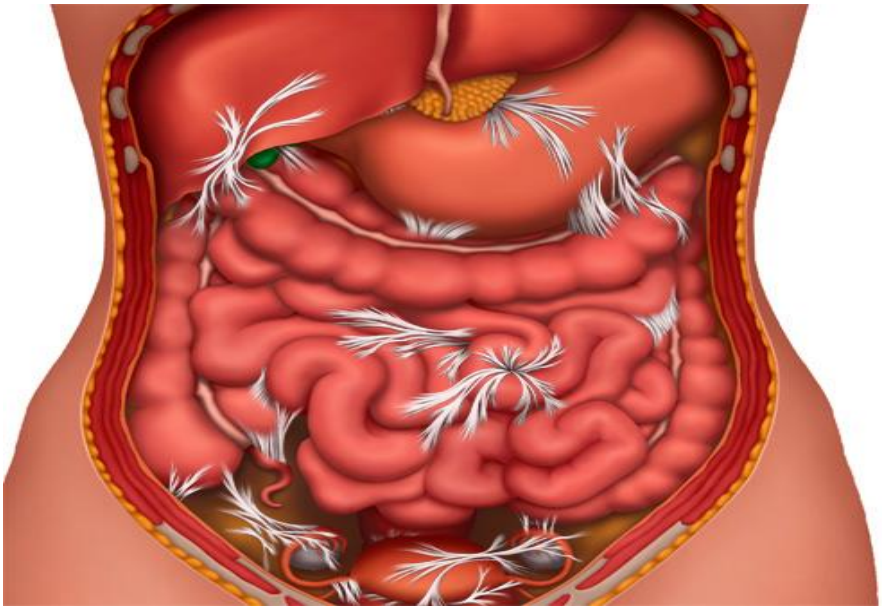


FIGURE – 1 : ADHESIONS

HERNIA

Hernias are the second most common cause of bowel obstruction in most reported series. Inguinal hernias and hernias acquired postoperatively most frequently lead to intestinal obstruction, but congenital abdominal wall or internal hernia may on occasion cause a bowel obstruction by incarcerating intestinal contents. Hernias as an etiology are more common in males than in females, primarily because of the predominance of inguinal hernias in men. In contrast, incarcerated femoral or obturator hernias are more common in women.

Approximately 5% of external hernias will require emergency operation if they are repaired electively. These hernias are usually incisional hernias, umbilical hernias and indirect inguinal hernias or femoral hernias. Inguinal hernias rarely incarcerate, which has changed their management from repair of all minimally symptomatic patient. The presence of acute incarceration should prompt emergent operative management, because 10% to 15% of incarcerated hernias contain necrotic bowel at exploration. Chronically incarcerated hernias

can develop strangulation, but most chronically incarcerated hernias can be managed electively

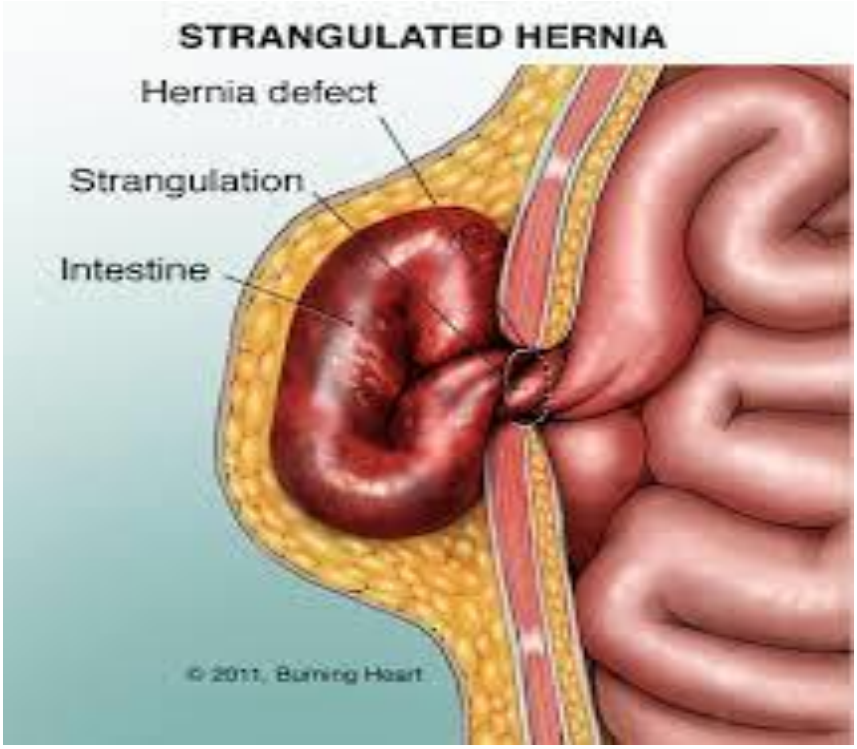


FIGURE – 2 : STRANGULATED HERNIA

INTERNAL HERNIA AFTER LAPARASCOPIC GASTRIC BYPASS

Minimally invasive surgery has brought new etiologies of intestinal obstruction. The reported incidence of internal hernia after intestinal surgery and especially after Roux-en-Y gastric bypass (RYGB), is 0.2% to 3%, a significantly increased incidence compared with the open approach. Factors contributing to the increased risk of internal hernia after a laparoscopic approach include lack of adhesion formation, increased small bowel motility, marked weight loss-induced increased opening, and failure to close all mesenteric defects appropriately. There are two or three mesenteric defects created during RYGB, depending on whether the retrocolic or antecolic technique is used. Petersen's defect or space is the best-

known site of herniation and can arise with either an antecolic or retrocolic position of the alimentary limb. It is named after Petersen, who in 1990 described two cases of internal herniation posterior to a loop gastrojejunostomy. Internal hernias are often with non-specific or intermittent symptoms (periumbilical pain, nausea, vomiting, anorexia, abdominal distension) Spontaneous reduction in the hernia can occur and CT, upper GI contrast series and plain abdominal films may be non-diagnostic. Symptoms of intermittent bowl obstruction after laparoscopic gastric bypass should raise suspicion for the presence of an internal hernia especially after weight loss. The best measure to prevent these hernias is the meticulous closure of the created mesenteric defects and suspicion of an internal hernia may itself be appropriate justification for operative exploration, especially via a diagnostic laparoscopy.

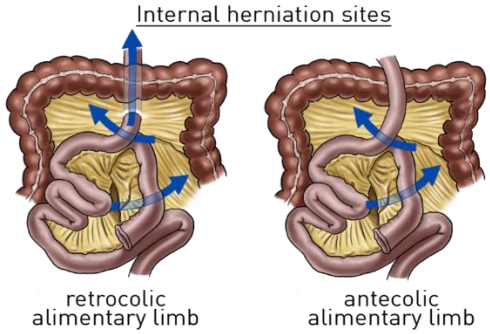


FIGURE – 3 : INTERNAL HERNIATIONS

TROCAR SITE HERNIA

The reported incidence of trocar site herniation is 0.2% to 3%; the true long-term incidence, however might even be greater. Trocar site hernias are observed rarely with 5mm trocars but more frequently with the use of 10mm,12mm or bigger trocars and especially with the “cutting” or bladed trocars. Closure of the fascial defect and the use of non-cutting, radial expanding trocars are recommended to decrease the risk for formation of trocar site hernias. Trocar site hernias can lead to small bowel obstruction early or late after a minimal access, intra-abdominal procedure.

Following a laparoscopic procedure, patient complaints of pain in the region of a trocar site, nausea or vomiting should lead to investigation for a bowel obstruction. In these cases, the bowel obstruction may be partial or complete. Commonly, the antimesenteric portion of the bowel wall will be incarcerated in the small fascial defect, resulting in a partial obstruction. These hernias are dangerous, because they may result in strangulation and necrosis in the absence of intestinal obstruction. Reduction of necrotic bowl during hernia repair can result in missed perforation and peritonitis. Although trocar associated hernias are rare, with the widespreaed use of laparascopy, they have become a well-known complication.

MALIGNANT BOWEL OBSTRUCTION

Primary intra abdominal neoplasms are a common cause of both large and small bowel obstruction. Colorectal, gastric, small bowel and ovarian neoplasms are the most frequent causes of malignant bowel obstruction, either from primary lesion(colon and small bowel neoplasms) or from peritoneal metastases(ovarian, colonic and gastric neoplasms) In many of these patients, bowel obstruction is associated with a high rate of recurrence and morbidity and may often be a terminal event.

Metastatic cancer can also cause bowel obstruction usually small bowel obstruction, usually small bowel obstruction. The most common form of obstructing metastatic lesion is peritoneal carcinomatosis related to one of the aforementioned primary, intra-abdominal malignancies, but localized hematogenous metastases to the wall of the small intestine from melanoma and carcinoma of the breast, kidney or lung can also cause intraperitoneal metastases that can obstruct the bowel.

CROHN'S DISEASE

Crohn's disease is a chronic,transmural,inflammatory ailment of the gastrointestinal tract that may affect any part of the alimentary tract from the mouth to the anus. Despite often

intense involvement of the bowel wall, Crohn's disease is responsible for fewer than 5% of cases of small bowel obstruction. When true mechanical obstruction is present, the cause is usually secondary to the inflammatory process or to chronic stricture formation. Other granulomatous diseases causing obstruction such as tuberculosis and actinomycosis are much less in western countries but in the developing world where AIDS is endemic,intra abdominal tuberculosis must be entertained in the diagnosis of intestinal obstruction.

INTUSSUSCEPTION

Intussusception is a relatively frequent cause of bowel obstruction in infancy, but it accounts for only 2% of bowel obstruction in the adult population. The median age of presentation in adults with intussusception is the sixth to seventh decade. The etiology of intussusception differs greatly between adult and pediatric patients. In the vast majority of adult intussusceptions, there is a demonstrable inflammatory lesion or a neoplasm that serves as the lead point of the intussusception. 20% of adult cases are idiopathic. Neoplasms causing intussusception in adults are malignant in almost 50% of patients.

VOLVULUS

Volvulus represents an axial twist of the bowel and its mesentery. This entity is an infrequent cause of small or large bowel obstruction in the western world. Volvulus is encountered more frequently in the geriatric population, in individuals with a long history of constipation, or in institutionalised, neurologically impaired or psychiatric patients. Colonic volvulus comprises about 1% to 4% of all bowel obstructions and about 10% to 15% of all large bowel obstructions. The volvulated segment must be mobile to allow the degree of freedom necessary to permit an axial twist of mesentery. The affected segment has either a long, narrow mesentery(malrotation or cecal volvulus) and/or a lack of bowel wall fixation

(floppy caecum syndrome). Overall, sigmoid volvulus accounts for 75% of all patients with volvulus. Cecal volvulus accounts for 25% of bowel volvulus and is the most common cause of large bowel obstruction in pregnancy. The “cecal bascule” is a unique form of cecal volvulus that occurs when the true anatomic cecum folds anteriorly over onto the ascending colon obstructing the lumen. This form of cecal volvulus may be intermittent and recurrent and is especially difficult to diagnose.

Primary volvulus in the small intestine is generally rare but it is quite prevalent in central Africa and India. The etiology has been related to abrupt dietary changes that occur during the religious holiday when the people celebrating Ramadan fast during day and then consume a large meal after dark. Some claim that this racial group has an exceedingly long, floppy small bowel mesentry that permits generous mobility of bowel.

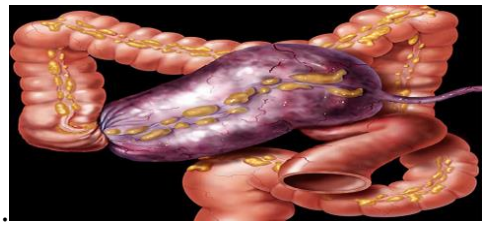


FIGURE – 4 : CECAL VOLVULUS

DIAGNOSIS

The diagnosis of bowel obstruction is highly suspected clinically based on careful history taking and physical examination and it may be confirmed by imaging, such as abdominal radiography or CT. The etiology of the obstruction can often be determined by astute history taking complemented by physical examination and imaging studies.

HISTORY AND PHYSICAL EXAMINATION

The classic clinical picture of a patient suffering from bowel obstruction includes intermittent crampy abdominal pain, distension, acute obstipation, nausea and vomiting.

Abdominal pain and then distension usually precede the appearance of nausea and vomiting by several hours. The more proximal the obstruction, the earlier and prominent are the symptoms of nausea and vomiting, distension is usually less prominent. Conversely, the more distal the obstruction, the more prominent is the abdominal distension. The abrupt onset of symptoms may herald the presence of a closed loop obstruction. The location and character of pain may be helpful in differentiating mechanical bowel obstruction from ileus. Ileus tends to have a more diffuse and mild pain, often without waves of colic, while mechanical bowel obstruction usually presents as severe, truly colicky pain. Recurrent paroxysms occurring in short (10-30 secs) crescendo-decrescendo episodes is often associated with mechanical small bowel obstruction, while in mechanical large bowel obstruction episodes are usually spaced farther apart and tend to last longer(1-2 min). Pain is usually described as visceral and poorly localised. Classically, the presence of constant or localised pain has been regarded as a sign of strangulation.

Obtaining a complete medical history is of paramount importance to make the diagnosis and determine the etiology. The fundamentals of history taking including the type and location of pain, the temporal association of symptoms, associated symptoms and aggravating and alleviating factors are all important components. The past medical history may also be critical in both making the diagnosis and establishing the cause of bowel obstruction. It is essentially important to inquire about previous episodes of bowel obstruction, recent and distant abdominal operations, current medications, a history pf chronic constipation, recent changes in the calibre of stools and a history of cancer and its treatments. Other cause of chronic intestinal obstruction such as Crohn's disease or Tuberculosis should be discussed.

A thorough physical examination is mandatory and should include assessment of vital signs and hydration status as part of the initial resuscitation. Tachycardia, hypotension and oliguria are signs of advanced dehydration that mandate aggressive resuscitation. Fever may

be associated with an infective cause or with strangulation. In abdominal examination it is important to look closely for potential hernia defects and previous surgical incisions. Auscultation can determine the presence, frequency and quality of obstructed bowel sounds. Bowel obstruction may have the metallic tinkling sounds of water dripping into a large hollow container indicative of dilated bowel with an air fluid interface. Functional obstruction may present with absence of bowel sounds. Mechanical bowel obstruction presents with an increase in the frequency of bowel sounds but more specifically the high pitched rushes and groans followed by the metallic tinkling sounds. A succussion splash may be heard in the presence of a dilated stomach or markedly dilated small bowel filled with an air fluid interface.

Abdominal palpation should reveal the presence of peritoneal signs such as rebound, localised tenderness and involuntary guarding that herald vascular compromise or perforation. The presence of these findings is suggestive of the need for an emergent operation. Abdominal masses should be noted. Digital rectal examination is required to demonstrate empty or roomy rectum and to rule out fecal impaction or a low lying rectal cancer as a cause of obstruction.

LABORATORY

Laboratory tests are essential in patients with bowel obstruction because they may aid in the diagnosis and more importantly any underlying metabolic defects should be corrected prior to operative therapy. While no rest in sensitive and specific enough to diagnose mesenteric ischemia reliably, a spectrum of laboratory tests may be helpful in determining the condition of the patient and should guide resuscitation. A complete blood cell count and differential, electrolyte panel, blood urea nitrogen, creatinine and urinalysis should be obtained to evaluate fluid and electrolyte imbalance and to assess the possibility of sepsis.

Arterial blood pH, serum lactate concentrations and amylase and lactic dehydrogenase activity may be useful tests in the evaluation of bowel obstruction, especially when trying to exclude the presence of strangulation or underlying bowel necrosis. An increase in serum lactate concentrations should raise the suspicion of intestinal ischemia. Intestinal fatty acid binding protein (I-FABP) is a highly sensitive marker for extensive mesenteric infarction. Also serum concentrations of phosphate, isoforms of creatine phosphokinase (isoform B), ischemia modified albumin, gut luminal tyrosine concentrations, alpha glutathione S transferase may identify the presence of intestinal cell necrosis.

RADIOLOGIC FINDINGS

The management of small bowel obstruction remains heavily reliant on excellent clinical acumen and appropriate imaging. The clinician is faced with answering critical questions, " Is this complete obstruction," and "is the intestine ischemic?" the literature is replete with clinical studies examining the prognostic value of various forms of imaging in terms of predicting the need for operative management or the presence of intestinal ischemia.

FLAT AND UPRIGHT ABDOMINAL RADIOGRAPHY

Plain radiographs including a chest x-ray and flat and upright films of the abdomen remain a viable initial imaging modality in patients with clinical bowel obstruction. An initial chest x-ray may reveal extra abdominal processes such as pneumonia that could be associated with an ileus rather than bowel obstruction. The presence of free air from a perforated viscus may indicate a diagnosis other than small bowel obstruction requiring emergent operation. Flat and upright films of the abdomen in patients with a small bowel obstruction characteristically have multiple air fluid levels in dilated loops of bowel and a paucity of gas in the distal

decompressed small bowl and colon. The location of the obstruction in the proximal or distal small intestine greatly influences the findings on the plain abdominal films. A very proximal small bowel obstruction may be associated with relatively small gastric air fluid level resulting from a fluid filled stomach. Conversely a distal small bowel obstruction will have multiple air fluid levels with dilated loops of small bowel stacked on one another. On a plain abdominal film, the small bowel lies centrally and intestinal markings from the valvulae conniventes or plicae circulars encompass the entire diameter of the bowel, whereas large bowel lies at the periphery of the abdomen and haustral markings only partially cross the bowel. The appearance of the bowel gas may also give a clue as to the duration of the obstruction. Fecalization of the small bowel content whereby the luminal contents show less of an air fluid level and more of an appearance of semisolid content with pockets of gas, suggests a more chronic obstruction. Rarely a plain radiograph will contain a pathognomonic sign of intestinal obstruction from gallstone ileus. Importantly, plain films are notoriously poor indicators of bowel involved with vascular compromise unless devastating signs of portal venous gas and intestinal pneumatosis are evident. Closed loop bowel obstructions are also difficult to diagnose on plain x-rays because the involved bowel with a proximal and distal occlusion may be fluid filled and lack any gas. Thus additional imaging procedures should be obtained in patients with any suspicion on compromised bowel.

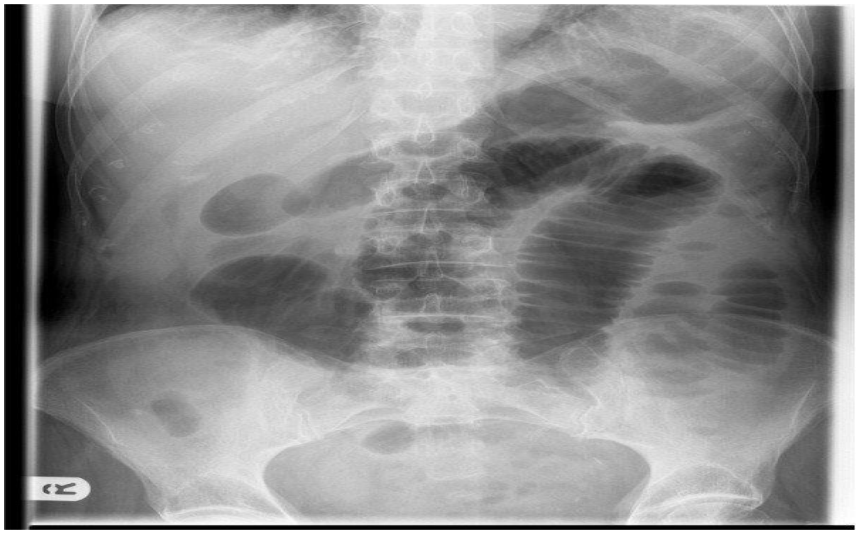


FIGURE – 5 : MUTIPLE DILATED BOWEL LOOPS

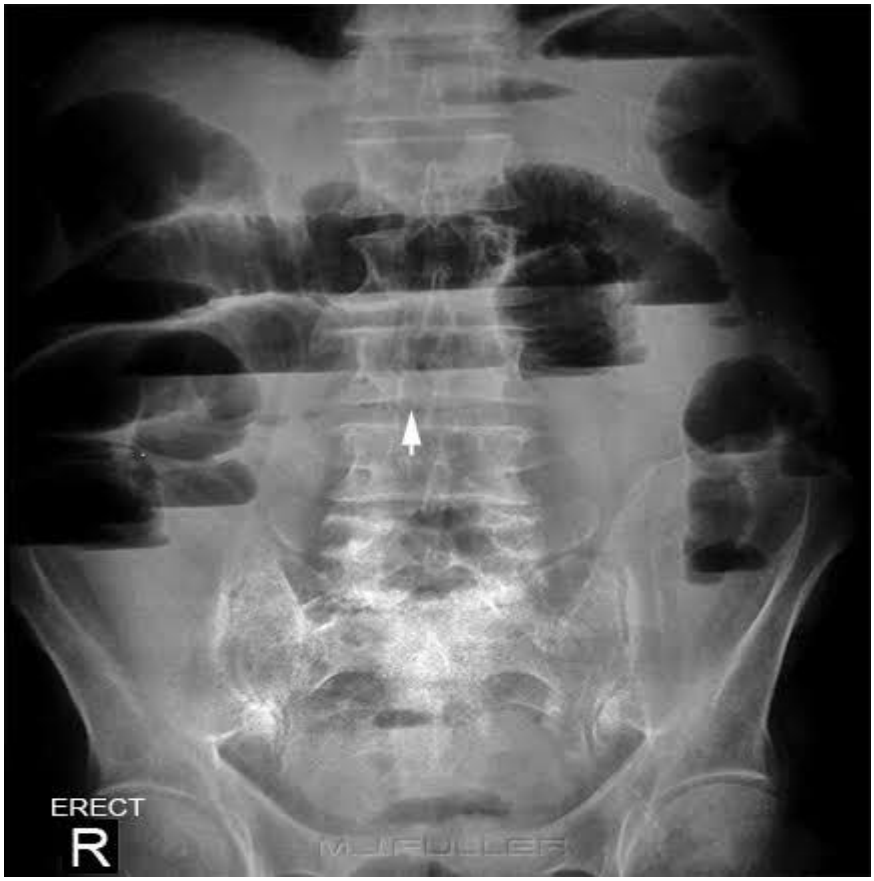


FIGURE – 6 : MULTIPLE AIR FLUID LEVELS

CONTRAST STUDIES

Although contrast studies using dilute barium or hyperosmotic, water soluble contrast of the small and large bowel have been an integral component of the diagnostic evaluation, enthusiasm for these studies has waned substantially. The radiologic literature and various guidelines developed by the radiologic community supports strongly the use of contrast-enhanced CT as the diagnostic imaging modality of choice. Nonetheless, in specific clinical situations, such as in a patient with an obstructing sigmoid or rectal tumor, a radiograph with rectally administered contrast may provide diagnostic information that is timely, economical, and clinically important.

On occasion, a small bowel follow though series may be helpful in distinguishing between mucosal inflammation and extraluminal compromise from adhesions as the etiology of bowel obstruction in a patient with crohn's disease. This diagnostic information may alter the therapeutic approach but generally small bowel follow through studies have little if any advantage over CT.

When contrast agents are utilized, the risks of each agent must be considered carefully. The primary side effects of barium include inspissation in the obstructed large bowel. Barium results in severe intraperitoneal infection/barium peritonitis when extravasated in the face of small intestinal perforation. Gastrografin if aspirated, can cause severe pneumonitis also it becomes diluted rapidly with an established small bowel obstruction, and thereby yields little information in a distal small bowel obstruction. Finally most surgeons agree that contrast studies are contraindicated in patients with a clear diagnosis of complete bowel obstruction and when strangulation or perforation is suspected.

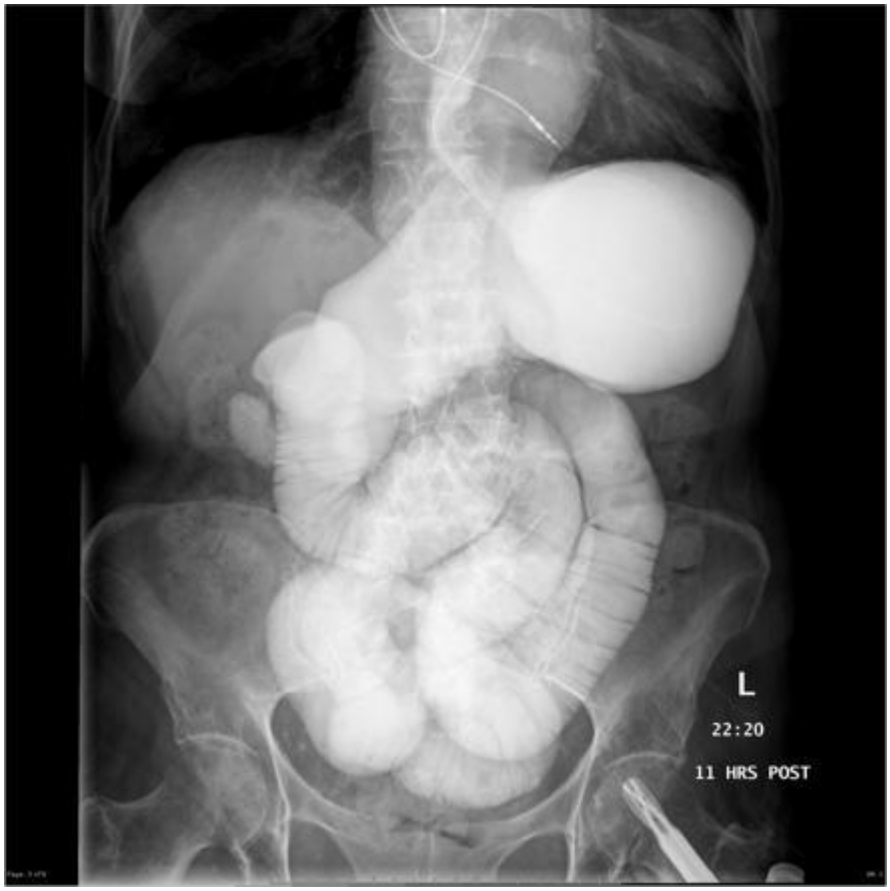


FIGURE - 7 : CONTRAST STUDY SHOWING SMALL BOWEL OBSTRUCTION

COMPUTED TOMOGRAPHY

CT has become the primary diagnostic imaging modality for the diagnosis of suspected intestinal obstruction in many centers. The increased use of CT reflects the preference of clinicians for the additional diagnostic information garnered from this examination. CT not only provides information about the presence or absence of a luminal obstruction,, but it can also define both the site of obstruction and the existence of extraluminal processes, a small bowel transition point, associated inflammation, fluid collections, masses, abdominal wall or internal hernias, and free intra peritoneal fluid. CT can expedite the diagnosis of strangulation obstruction if findings including mesenteric edema, free peritoneal fluid, intestinal wall thickness and the absence of fecalisation of the small bowel are present. Early detection of bowel ischemia is paramount to successful surgical management of obstruction. Several

studies have reported a diagnostic accuracy of greater than 90% with the use of CT in intestinal obstruction. The presence of two or more beak signs, whirl signs, a C- or U- shaped appearance of bowel loop and a high degree of obstruction were assosciated with non surgical treatment failure. Among studies utilizing IV contrast, reduced small bowel enhancement had a 95% specificity in determining ischemia and absence of mesenteric fluid had an 89% sensitivity in ruling out strangulation. In setting where iodinated contrast is contraindicated the finding of increased bowel wall attenuation on unenhanced images is concerning for bowel ischemia, with a 100% specificity and 56% sensitivity. A recent study suggested that CT findings of free peritoneal fluid, thickened bowel and mesenteric edema, combined wih vomint were predictive of the need for eventual operative management, but though relatively sensitive for ischemia, not very specific. It is important to remember that CT is better at identifying rathen than excluding the presence of ischemia.

Overall the current preference for the use of CT is associatied with an increased likelihood of operative intervention and decreased mortality however, these associaions are causal or coincidental remains unknown.

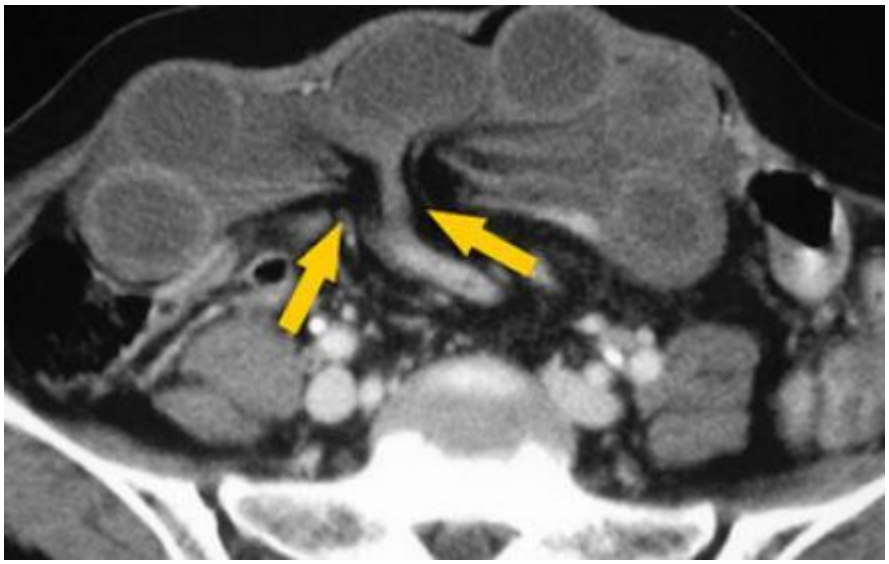


FIGURE - 8 : CT SHOWING CLOSED LOOP SMALL BOWEL OBSTRUCTION

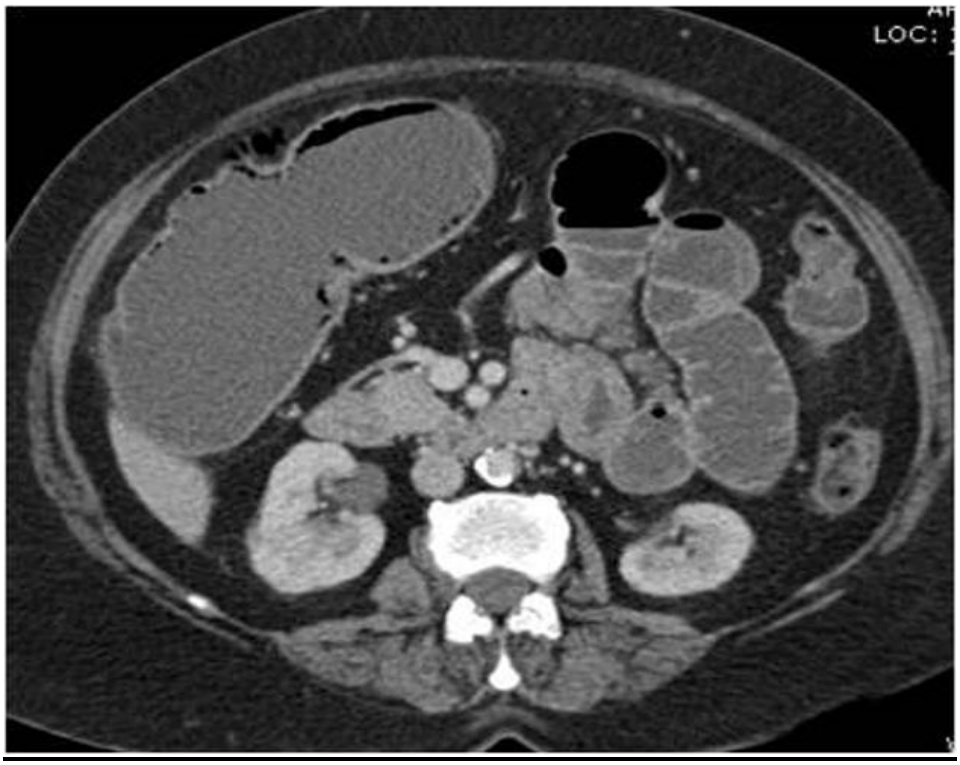


FIGURE – 9 : CT SHOWING LARGE BOWEL OBSTRUCTION

ULTRASONOGRAPHY

Ultrasonography(US) is used infrequently in the diagnosis of intestinal obstruction. Features concerning for strangulated bowel include akinetic bowel loops, hyperechoic and thickened mesentery and presence of peritoneal fluid. US has been reported to be useful for the early recognition of strangulation obstruction in several studies however in absence of an experienced ultrasonographer, the reliability of US remains questionable. Also it is difficult to perform in obese patients, and extensive bowel gas may obscure the pattern of intestinal obstruction.

MAGNETIC RESONANCE ENTEROGRAPHY

Magnetic resonance enterography (MRE) has not been utilised as frequently as CT because performance of this examination is more time consuming and requires substantial expertise in interpretation. In general practice, it does not have a greater diagnostic accuracy

than CT. MRE may have an advantage of distinguishing benign from malignant bowel strictures in patients with suspected malignant bowel obstruction. In centers that use MRE frequently, diagnostic accuracy exceeding 90% is achievable.

VIDEO CAPSULE ENDOSCOPY

Video capsule endoscopy (VCE) may be a valuable diagnostic tool in patients with subacute or chronic intestinal obstruction where other imaging techniques have not revealed an etiology. VCE is particularly helpful in patients with obstruction related to a stricture caused by inflammation or malignancy. VCE may provide a diagnosis in nearly 40% of previously undiagnosed patients. Retention or impaction of the capsule either at a stricture or in any area of severe kinking related to adhesions in a patient who otherwise may have resolution of the obstruction without an operation, is a major concern with use of VCE. The incidence of this circumstance is infrequent, but when happens may require celiotomy.

DETECTION OF ISCHEMIA

It is critical to diagnose the strangulation obstruction caused by ischemia of the intestine, because the mortality associated with strangulated bowel obstruction is 9% to 40% compared to less than 5% in non-strangulated intestinal obstruction. Clinical and imaging parameters claimed to permit early detection and operative intervention remain unreliable and in fact do not help in early diagnosis. Historically, acidosis, increased serum amylase activity and increased serum lactate levels were also claimed to be indicators of strangulation. Given the conflicting evidence, the importance of integrating physical exam, imaging and other clinical parameters (e.g. Worsening acidosis) when assessing a patient with bowel obstruction cannot be overemphasized.

MANAGEMENT

The initial management of patients with small bowel obstruction should focus on aggressive fluid resuscitation and nasogastric decompression of the stomach to prevent further accumulation of intestinal fluid and air. In addition, nasogastric decompression decreases the potential for aspiration and relieves vomiting. These therapies should be instituted in all patients, whether they are treated operatively or undergo a trial of non-operative management. Blood should be analysed for serum electrolyte concentrations, complete blood count, lactate concentration, typed and screened for potential transfusion, and when necessary, arterial blood gases should be analysed as well. The most important initial step in management is crystalloid fluid resuscitation that aims to replete fluid losses. Patients with small bowel obstruction often present with profound volume depletion and may require several litres of isotonic crystalloid solutions, such as normal saline (0.9% NaCl) or lactated Ringer solution with additional potassium as urine output is restored. Resuscitation should be guided by urine output, provided the patient is hemodynamically stable and has normal renal function. Patients who are hemodynamically unstable or have impaired cardiac, pulmonary, or renal function may require monitoring of central venous pressure to better evaluate their volume status. Colloid solutions, such as 5% albumin or hetastarch, have little or no role in the resuscitation of patients with a small bowel obstruction. Proper fluid resuscitation includes correction of metabolic or electrolyte imbalances, which may be severe. Specifically, in patients who have experienced prolonged vomiting, potassium and chloride should be measured to diagnose hypokalemic, hypochloremic alkalosis and replacement therapy started after resuscitation with normal saline. Though potassium replacement is a critical component of therapy, replenishment of this electrolyte should begin only after renal function has been established by good urine output. Volume resuscitation, electrolyte replacement, and establishment of adequate urine output are critical before operative therapy is undertaken. Broad-spectrum antibiotics should be given to patients within an hour of the

incision as prophylaxis against surgical site infection, but otherwise, antibiotics have no defined role postoperatively or in patients managed nonoperatively.

Nasogastric decompression helps to prevent aspiration during vomiting and on induction of general anaesthesia. Symptomatically, gastric decompression helps relieve abdominal distension and can improve respiratory function in patients with respiratory compromise. Long intestinal tubes placed distal to the pylorus were also used historically, to relieve small intestinal distention under the assumption that intestinal decompression may be therapeutic if related to adhesions, because the decompressed bowel may detort and thereby relieve the mechanical obstruction. Success rates of up to 90% have been reported in some series of patients treated with a long nasointestinal tube. In contrast, however, most prospective and retrospective studies have failed to demonstrate the superiority of nasointestinal versus nasogastric intubation, making the added expense of fluoroscopic or endoscopic placement of a nasointestinal tube unwarranted. Use of these long intestinal tubes has fallen out of favor, and they are of historic interest only in the preoperative treatment of small bowel obstruction.

NON-OPERATIVE MANAGEMENT

Nonoperative management of intestinal obstruction should be considered only in patients with uncomplicated intestinal obstruction in the absence of peritonitis, a progressive leucocytosis, or impaired bowel wall perfusion on imaging. When indicated, this approach is reported to be successful in 62% to 85% of patients. The rate of success of nonoperative management is influenced by patient selection, type of bowel obstruction (complete vs partial), etiology (e.g., adhesions, hernia, or neoplasm), and the surgeon's threshold for conversion to operative management. Patients successfully managed nonoperatively require fewer hospital days and avoid the morbidity or convalescence necessitated by an operation. Few studies have compared the long-term outcomes of patients with a small bowel obstruction treated nonoperatively versus operatively.

When patients with a small bowel obstruction are initially managed nonoperatively, vigilant attention must be paid to volume resuscitation, electrolyte homeostasis, and nasogastric decompression. Patients managed nonoperatively require the same aggressive resuscitation and replacement of daily losses with an appropriate crystalloid solution and electrolyte replacement as patients who are managed operatively. Fluid replacement should take into consideration the volume and electrolyte loss in the output of the nasogastric tube, urinary output, and insensible losses. Electrolytes should be monitored frequently and corrected as necessary. Delayed correction of potassium and magnesium concentrations may lead to delayed return of bowel function and misdiagnosis of obstruction versus ileus. Adequate proximal decompression is important to allow the bowel an opportunity to decompress. This concept is accomplished by maintaining a functioning nasogastric tube. If the patient becomes progressively more distended or develops vomiting, tube placement should be evaluated and tube function confirmed by bedside evaluation. Standard nasogastric tubes should be inserted, such that the second of four marks is evident at the tip of the nares. The first mark is 40 cm from the tip of the tube-that is, the normal distance from the nares to the esophagogastric junction. Thus, if all four marks are outside the nares, the tube most likely is not in the stomach. Likewise, if no marks are visible, the tube is coiled within the stomach or is in the duodenum. On occasion, an abdominal radiograph is necessary to confirm placement. If the tube is noted on a radiograph to be out of position, it should be repositioned and imaged again for proper placement. On evaluation, the tube should be connected to the suction apparatus, sumping properly (if the tube has a sump port), and should be checked for patency by flushing and aspirating water through the suction lumen. Oral intake should be nil in the presence of a nasogastric tube. In addition, the tube should never be “clamped” for prolonged periods of time, because by traversing the esophagogastric junction, the tube will lead to an incompetent lower esophagogastric sphincter and potential aspiration. Connection of the tube to a drainage bag for a brief trial is an appropriate alternative to clamping and may be used as a test to determine patient readiness for

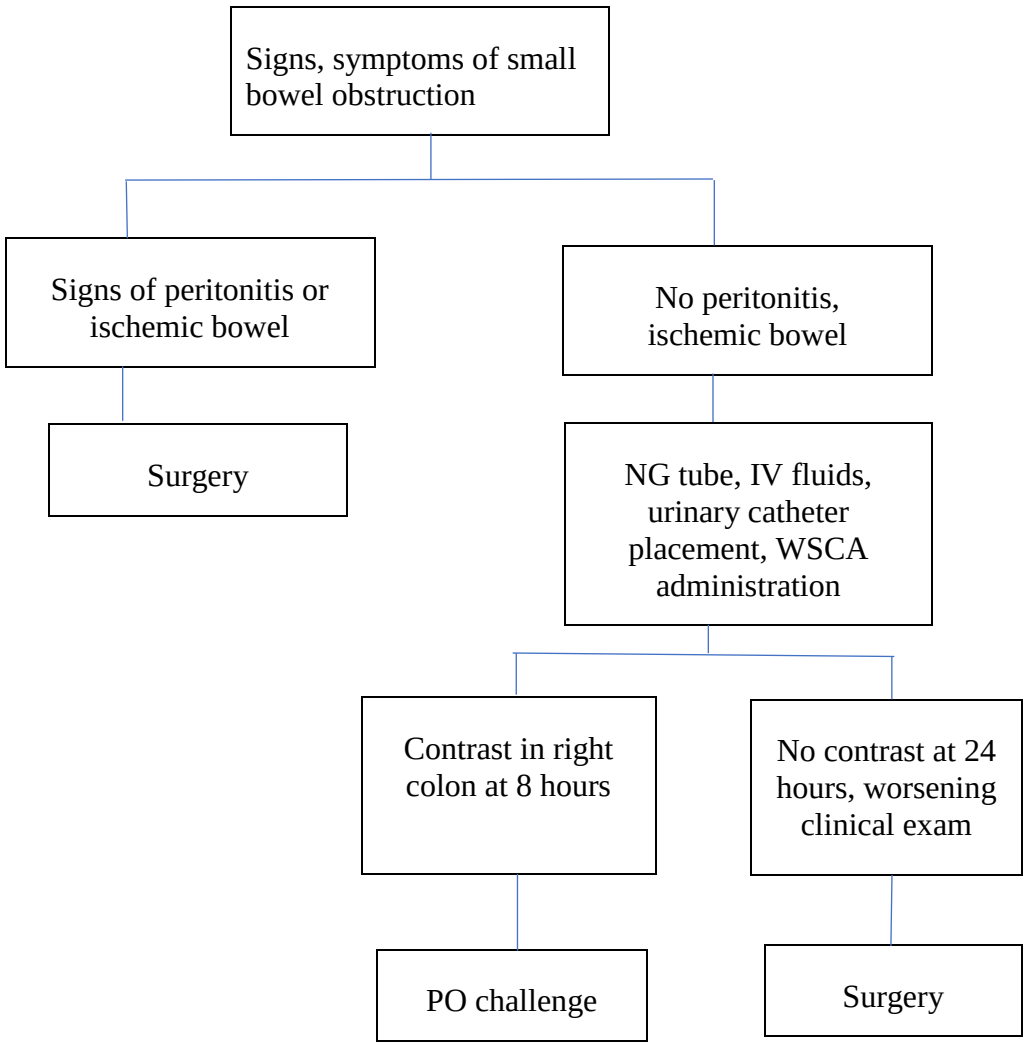
nasogastric tube removal.

Absolute contraindications to nonoperative management include suspected ischemia, large bowel obstruction, closed-loop obstruction, acutely incarcerated or strangulated hernia, and perforation. A relative contraindication to nonoperative management is complete small bowel obstruction- that is, dilated small intestine with no air in the bowel distally. To better delineate partial and complete obstruction, studies have adopted a protocol-driven approach to utilize water-soluble contrast agents (WSCA) in nonoperative management. Among the protocols described in the literature, patients presenting with signs and symptoms of small bowel obstruction were assessed clinically and on CT imaging. Those demonstrating features concerning for ischemia underwent operative exploration immediately following appropriate resuscitation. The remaining patients receiving nonoperative treatment underwent gastric decompression, fluid resuscitation, urinary catheter placement, and WSCA administration.

Following WSCA, abdominal plain films were taken at 8 hours after or 1, 2, 4, and 8 hours after administration, depending on the study. Patients passed WSCA challenge if contrast reached the right colon by times ranging from 8 hours to 24 hours after WSCA. Patients who developed worsening signs and symptoms consistent with peritonitis underwent exploratory laparotomy. Among the patients who failed WSCA challenge but did not have a worsening signs, time from WSCA administration to operative management varied from 24 hours to 4 to 5 days. Success rates using WSCA protocols have ranged from 57 to 90.5%.

Based on a recent meta-analysis of 14 prospective trials, presence of contrast in the colon predicted resolution of obstruction with 96% sensitivity, 98% specificity, 99% positive predictive value, and 90% negative predictive value. The authors supported use of WSCA as both a diagnostic and therapeutic tool and demonstrated a decreased need for surgery and decreased hospital length of stay, although results from individual studies remain mixed regarding length of stay and frequency of laparotomy. These studies support use of WSCA protocols in adhesive small bowel obstruction and suggest that protocols decrease use of non-therapeutic laparotomies while diminishing delays in surgical care when indicated.

If non operative management is attempted in a patient with complete obstruction, the decision should be made with the understanding that there is a definite risk of overlooking a underlying strangulation obstruction and thus there should be a low threshold for operative intervention in patients with complete obstruction.



PROTOCOL FOR USING WATER SOLUBLE CONTRAST AGENTS IN NONOPERATIVE MANAGEMENT OF SMALL BOWEL OBSTRUCTION

When all the clinical and imaging findings are equivocal in a patient, other parameters like arterial pH, serum lactate concentrations, amylase and lactate dehydrogenase activity, D-dimer, Intestinal fatty acid binding protein (IFAP), plasma level of ischemia modified albumin, gut luminal tyrosine concentration and alpha glutathione S transferase will aid towards operative management of the condition.

WHEN TO CONVERT TO OPERATIVE MANAGEMENT

Prompt operative intervention is mandatory in patients who develop signs and symptoms suggestive of a strangulation obstruction. These parameters include fever, tachycardia, leucocytosis, localized tenderness, continuous abdominal pain, and peritonitis. The presence of any three of these signs has an 82% predictive value for strangulation obstruction. Similarly, the presence of any four of the above signs has a near 100% predictive value for strangulation obstruction. Obviously, patients who develop free air, signs of a closed-loop obstruction on abdominal radiograph, or gross peritonitis require operative exploration. If CT demonstrates evidence of ischemia, such as pneumatosis intestinalis, bowel wall thickening, portal venous gas, generalized ascites, or nonenhancement of the bowel wall, operative intervention should be strongly considered.

The timing of conversion to operative management in a patient with a small bowel obstruction who is not improving with nonoperative management is more controversial. Some surgeons advocate operative intervention in any patient who fails to show improvement within 48 hours of initiating therapy. Others advocate a more liberal use of nonoperative therapy, citing a mean time to successful resolution of up to 4.6 days. The authors believe that nonoperative management can be continued greater than 48 hours with the understanding that delaying inevitable place the patient at increased risk for perioperative morbidity. As mentioned earlier, implementation of a protocol-driven approach with use of water-soluble contrast agents may be of diagnostic benefit in this setting, though further studies are needed to identify the optimal time to pursue operative care. It is

important for the surgeon to remember that nonoperative management always carries a calculated risk of overlooking an underlying strangulation obstruction.

OPERATIVE MANAGEMENT

Once the decision has been made to pursue operative management, steps should be taken to prevent peri- and postoperative complications. Preoperative preparation includes assessing the medical fitness of the patient, and as time allows, taking steps to optimize the patient's medical status. Special consideration should be given to ensure that the patient has been resuscitated adequately by establishing adequate urine output, appropriate antibiotics have been administered, and any electrolyte abnormalities have been addressed. Consideration should be given to the administration of β- blockers to patients with cardiovascular comorbidities and especially to those who were on β-blockers prior to admission. A nasogastric tube should already be in place to decrease the risk of aspiration during the induction of anaesthesia; nevertheless, a rapid-sequence anaesthetic induction will be necessary to protect the airway during intubation, even in the presence of a nasogastric tube. Several decisions must be made with regard to operative planning to provide the safest approach that will afford the best outcome for each individual patient. The choice of operative approach and incision is important to allow the surgeon adequate exposure and visibility. A laparoscopic approach should be considered in some patients. When an obstruction develops in the early postoperative period; the original incision should be reopened provided extensive adhesions were not present originally. Safe entrance into the peritoneal cavity may be best achieved by approaching this from the extremes of the previous incision rather than going directly through the mid-portion of the incision. In patients without a history of prior abdominal operation or those who are remote from their original operation, a midline celiotomy affords the best exposure to all four quadrants of the abdomen. For example, patients with upper oblique, transverse, or subcostal type incisions may have pelvic adhesions that are difficult to address from the upper abdomen, especially

through a high transverse incision.

Once within the abdominal cavity, the first step is to identify the site and cause of obstruction. If the point of obstruction is not obvious, decompressed bowel distal to the obstruction can be identified and followed proximally to the point of obstruction. Care should be taken when handling the obstructed bowel at or near the point of obstruction when acutely obstructed, especially if it is fixed at an apparent site of obstruction or if it is ischemic. This region is at high risk for strangulation and infarction, making it more likely to rupture with spillage of bacteria-laden enteric contents into the abdomen. The dilated bowel proximal to the offending obstruction is often thin-walled and at increased risk for perforation if obstruction is acute. After the offending obstruction has been corrected, a thorough exploration of all four quadrants should always be undertaken to ensure that all intestinal injuries are repaired, nonviable segments are resected, and a second site of obstruction or fixation is not overlooked. This concept is especially true for volvulated segments of small bowel where two points of fixation are often present. Occasionally, obstructing bands traversing a sizeable part of the peritoneum can affect more than one loop of bowel. When a small bowel resection is necessary, intestinal continuity of the small bowel can be accomplished generally with a primary anastomosis unless there is generalized peritonitis and the edges of the remnant bowel are of questionable viability. When an intestinal anastomosis is performed, the surgeon must assess the discrepancy in bowel diameter and wall thickness between the obstructed proximal bowel and decompressed distal bowel when choosing anastomotic techniques. The surgeon may consider a side-to-side or end-to-side anastomosis in situations where massive dilation of the proximal bowel makes an end-to-end anastomosis difficult technically. In addition, a stapled anastomosis may be less safe in cases where a large discrepancy in bowel wall thickness exists or when there is bowel wall oedema, because uniform approximation of the tissue for a given staple height may not be possible. Abdominal closure may be difficult to achieve when the small bowel is massively dilated. In these cases, intraoperative intestinal decompression will facilitate closure. Techniques

described for intraoperative decompression include manual retrograde decompression into the stomach (with careful handling of the obstructed bowel), intraoperative passage of a long nasointestinal tube and, rarely, performance of a controlled enterotomy with passage of a decompressing tube. The latter technique is strongly discouraged except under very select circumstances, such as tremendous intestinal distention preventing abdominal closure or distention threatening bowel viability. Manual retrograde decompression of luminal contents around the ligament of Treitz, through the pylorus, and into the stomach allows for aspiration through the nasogastric tube by the anaesthetist. This manoeuvre is the safest and quickest technique because it allows closure of the abdominal wall while avoiding an enterotomy and excessive manipulation of the bowel. When decompressing the bowel, the inflamed and distended bowel must be handled gently, because experimental studies have demonstrated an increased rate of bacteraemia after extensive manipulation of obstructed bowel. In addition, the anaesthesia team should be alerted to the manoeuvre to be certain that their nasogastric tube is functioning well. Although intraoperative decompression has not been shown to decrease the rate of postoperative complications or the speed of return of bowel function, it certainly, does make abdominal closure easier, faster, and safer.

Nonviable bowel needs to be identified and resected. Resection should be undertaken with caution, especially in patients with a limited length of bowel from a previous resection or those with large sections of ischemia. Adjuncts for determining bowel viability include the use of Doppler US and intravenous fluorescein. These tests are relatively subjective, should be used with caution, and are only adjuncts to sound clinical judgment. In patient who would otherwise be left with less than two-thirds of their original bowel length after resection of all bowel of questionable ischemia, consideration may be given to resecting all the grossly necrotic or obviously nonviable bowel but preserving bowel of questionable viability and performing an end ostomy or a second-look procedure 12 to 24 hours later, particularly if the viability of the ends to be anastomosed is in question.

BYPASS VERSUS RESECTION

In patients with an incurable malignant small bowel obstruction, if the offending obstruction is unable to be released or it is deemed unsafe to attempt to dissect out the site of obstruction, intestinal bypass can be performed. Bypass relieves the obstruction while re-establishing intestinal continuity and preventing a closed-loop obstruction; however, the advisability of a bypass procedure should be considered. For instance, in the presence of carcinomatosis, a bypass may prove fastest and safest, because patient survival will be short.

In contrast, patients with certain chronic inflammatory diseases will remain at risk for ongoing problems (e.g., Crohn's disease or tuberculosis) related to the inflammation in any segment that has been skipped by bypassand these patients may require resection than simple bypass. The surgeon should at least consider an initial laparoscopic, minimal access approach in patients with uncomplicated small bowel obstruction. Laparoscopy is known to cause fewer adhesions than open laparotomy for treatment of adhesive small obstruction. Several studies have shown laparoscopy to be a safe and effective means of access for the operative treatment of small bowel obstruction. When successful, a laparoscopic approach decreases both the duration of hospital stays, and the complication rate. Patients successfully treated laparoscopically appear to have more rapid return of bowel function. These reports show a large benefit to laparoscopic treatment for small bowel obstruction, but need to be interpreted carefully. Many series compare patients treated laparoscopically to those who failed initial laparoscopic treatment. Those patients unable to be treated laparoscopically likely had more extensive adhesions or complicated pathology possibly requiring resection. Operative intervention in these patients would be more involved and complex whether done open or laparoscopically. One would expect these patients to have greater hospital stays, greater complication rates, and slower return of bowel function independent of the method of abdominal access. In addition, the skill and confidence level of the surgeon should weigh in the decision to approach the obstruction laparoscopically. First,

if the surgeon lacks skill in using moderately advanced laparoscopic techniques, an open operation may be a better choice. Similarly, if the patient is known to have a frozen abdomen or has either a severely distended, tense abdomen with markedly distended bowel or multiple dense adhesions at the time of insertion of the laparoscope, conversion to an open procedure is wise. Initial access for creating the pneumoperitoneum in a patient with a small bowel obstruction is achieved best by a fully open approach under total visual control, but limited data support this concept.

RECURRENT SMALL BOWEL OBSTRUCTION

Although the results of individual studies vary, between 4% and 34% of patients will experience recurrent small bowel obstruction regardless of management modality. This wide range of recurrence rates likely results from variations in both the duration and quality of follow-up between studies as well as the etiology of the original bowel obstruction. Recurrent obstruction is more common in patients with multiple adhesions, matted adhesions, previous admissions for small bowel obstruction, and previous pelvic, colonic, and rectal surgery.

In the past, numerous attempts have been made by surgeons to control the formation of adhesions in an effort to prevent future mechanical obstruction. A simple technique to prevent adherence of the bowel to the undersurface of the fascial incision is to interpose the omentum between the bowel and the incision. Theoretically, when adhesions from the posterior surface of the anterior abdominal wall form after omental interposition, they will involve the omentum and not the underlying bowel. Other more intricate techniques, such as the Noble plication and the Childs−Phillips trans mesenteric plication, have been described in the more distant past. These procedures involve the suturing adjacent loops of small bowel into an orderly pattern in an attempt to plicate the bowel permanently in a position that will not allow mechanical obstruction. Although initial reports were encouraging, the Noble and Childs−Phillips procedures have multiple complications and are of historic interest only. The

problems associated with plication procedures have included prolonged operative times and high rates of enterocutaneous and enteroenteric fistula, abdominal abscess, and wound infection; moreover, the rate of recurrent obstruction is as great as 19%, bringing into question their efficacy. Attempts to "plicate" the bowel with a long intestinal tube, so-called

intraluminal plication, have not proved effective. In some patients, complete or adequate adhesiolysis is not possible or may risk vascular injury to a substantial segment of bowel because of the acute inflammatory nature or tenacity of the adhesions. This situation is especially common when celiotomy is deemed necessary or performed too soon after a previous intra-abdominal procedure (see the following section on early postoperative small bowel obstruction). This situation is especially common when the previous operation involved an extensive adhesiolysis. In such situations, it may be important to control any bowel injuries present, end any further dissection, and conclude the operation to prevent further bowel injury and its potential sequelae. This "conservative" approach may allow the acute inflammatory process to resolve or regress (often 3-6 months); should the obstruction not resolve by 6 months, the plan should be to reoperate at a time when the adhesions have matured, allowing a more controllable and much safer adhesiolysis.

In some situations, the mature decision might be to provide proximal diversion with a proximal enterostomy if the obstruction has no chance for resolution (e.g., due to malignancy or radiation) or if a more distal bowel repair is tenuous, or to place a tube gastrostomy for diversion and patient comfort. Pursuing a futile attempt to complete the adhesiolysis puts the patient at risk for serious bowel injury or devascularization injury necessitating resection of otherwise normal bowel with the risk of enterocutaneous fistula or subsequent short bowel syndrome.

ADHESION PREVENTION

Over the last 100 years, multiple approaches have been employed in an attempt to prevent the formation of unwanted postoperative adhesions. These attempts include, among others, the use of cow cecum, shark peritoneum, sea snake venom, and fish bladder, as well as multiple fluids, mechanical barriers, and gels. The concept of separating injured surfaces mechanically to prevent adhesions is attractive. The formation of fibrin bridges (and thus adhesions) may be preventable by separating injured surfaces in the postoperative interval during the critical period of healing and mesothelialisation by application of an absorbable biofilm. Estimates of the minimum amount of time necessary for an impermeable or semipermeable barrier to prevent adhesion formation appear to be about 36 hours. Some authors have placed a Silastic sheet between two injured peritoneal surfaces and when left in place for 36 hours, no adhesions formed between these surfaces thereafter. Others have postulated that separating the surfaces at risk for the first 5 to 7 days until full mesothelialisation occurs would seem to be most effective; however, the barrier should not incite its own inflammatory response and should not decrease fibrinolytic activity or suppress access to oxygen. The ideal product, therefore, should be bioabsorbable, last only 5 to 7 days, be easy to apply, be interposed between all injured surfaces, and not itself incite an inflammatory reaction.

The most effective method to date has been the application of a sheet of bioresorbable hyaluronate membrane. This approach has been shown to decrease the formation of adhesions at the site of application. Multiple reviews have supported that use of this product decreased adhesion incidence of reoperation for adhesive small bowel obstruction remains unclear. Based on various studies and assumptions, the use of hyaluronate membranes in elective abdominal surgery does decrease the amount of postoperative adhesions at the site of application but does not decrease the incidence of intestinal obstruction or the need for future reoperation for obstruction. Use of these products requires careful consideration, because they are expensive and their clinical benefit appears to be

relatively low. Other materials or substances are being developed that may someday move to the forefront of adhesion prevention. These include gel and liquid preparations such as hyaluronic acid and carboxymethylcellulose, hydrogel, fibrin sealant, and protein polymers. Other adhesion barriers include oxidized regenerated cellulose (ORC). ORC has been well studied and does help prevent adhesion formation, but its use requires a blood-free field that at times is not practical to achieve. The use of ORC, like hyaluronate membranes, has not been shown to decrease the incidence of subsequent adhesive small bowel obstruction. Strategies including use of postoperative hyperbaric oxygen, peritoneal cell transplantation, and use of foetal-liver mesothelial cells have been described in animal models but have yet to be applied in a clinical setting.

EARLY POSTOPERATIVE SMALL BOWEL OBSTRUCTION

Early postoperative small bowel obstruction, herein defined as within 6 weeks of the original operation, is a relatively uncommon problem but remains a real dilemma encountered in every practice performing abdominal operations. It is often difficult, if not impossible, to distinguish early obstruction from postoperative ileus, but fortunately the management is usually quite similar. Patients with suspected early mechanical small bowel obstruction should be managed initially by nasogastric decompression, fluid resuscitation and correction of any electrolyte abnormalities. After a thorough physical examination and the decision that emergent intervention is not indicated, a search for the cause of obstruction should be undertaken. CT can be helpful in determining the etiology of an obstruction but is notoriously unreliable at differentiating ileus versus partial obstruction. Obstructions caused by extrinsic bowel compression amenable to percutaneous correction, including fluid collections, abscesses, and hematomas, may be diagnosed and treated by percutaneous drainage. CT may be able to detect those causes of obstruction that will likely require operative intervention, such as internal hernia, fascial dehiscence, and uncontrolled anastomotic leak. Early CT may be warranted in patients who had a laparoscopic operation

and have signs of early obstruction, because a port site hernia may be evident and would require prompt operation.

Generally, two categories of patients with early postoperative small bowel obstruction have been recognized. The first category includes those in whom the obstruction becomes evident within 10 days of an abdominal operation. Conservative management is advised usually as long as signs and symptoms of ischemia and strangulation obstruction are not present and other remediable causes have been excluded. Patients within this time frame are not at a substantially increased risk of bowel-related complications after celiotomy, provided there are no internal hernias and, if the original operation was done laparoscopically, that port site hernias can be excluded. It is important to rule out correctable causes of extrinsic compression and reverse any electrolyte abnormalities, especially if ileus is also suspected. Strangulation obstruction, albeit rare, can occur in this group of patients, and thus a high index of suspicion must always be maintained. The etiology of a strangulation obstruction in this group is almost never related to adhesions but rather to some surgical misadventure, such as internal hernia, an overlooked segment of ischemia at the original celiotomy, bowel entrapped in the fascial closure, or an unsuspected abdominal wall hernia. The second category of patients is those presenting between 10 days and 6 weeks after operation.

Conservative management is advised whenever possible for patients in this category as well. The risk of iatrogenic bowel complications during and after reoperation so early after celiotomy increases dramatically in this group secondary to the dense adhesions often present during this period after abdominal operation. The time period from 7 to 10 days up until 6 to 12 weeks postoperatively represents the window when the greatest inflammatory reaction is present intraperitoneally. The developing adhesions are highly vascular and friable. If the patient had no or very minimal adhesions at the time of celiotomy, reoperation is warranted; however, in a small, unpredictable group of patients without any previous adhesions, and reliably so in those with dense adhesions that had required substantial adhesiolysis at the time of original celiotomy, an acute inflammatory reaction involving the

peritoneal surfaces may agglutinate adjacent loops of bowel, often involving the omentum and mesenteric surfaces. Operations performed during this period have a much greater rate of iatrogenic injury and subsequent fistula formation. Those patients not responding to conservative management during this period are best placed on parenteral nutrition until the obstruction resolves or they are more than 6 to 12 weeks out from their last celiotomy. At this time, the decision to reoperate is made based on several considerations. First, if the patient had relatively few adhesions at the time of celiotomy, reexploration at 6 weeks to 3 months postoperatively may be warranted. In contrast, in those patients who required an extensive adhesiolysis at the time of original celiotomy, many experienced surgeons wait for a full 6 months prior to reoperation for several reasons:

1.by 6 months, the adhesions are reliably less vascular and more mature;

2. reoperation prior to 3 months may reveal a frozen abdomen in which the obstruction may be unable to be dissected free safely;

3.the obstruction may resolve as the adhesions mature.

BOWEL OBSTRUCTION AFTER ROUX-EN-Y GASTRICBYPASS SURGERY

As with all other operations and maybe more so in the current era of laparoscopic Roux-en-Y gastric bypass (RYGB), bowel obstruction is a worrisome complication after bariatric surgery for morbid obesity. Estimates of the rate of bowel obstruction after RYGB vary within a reported range of 0.3% to greater than 9% depending on the technique used to perform the operation. The rate of bowel obstruction appears to be less after open RYGB, but there are no large prospective studies comparing laparoscopic to open procedures at this time. Bowel obstruction after RYGB can occur secondary to a variety of aetiologies; however, the four most common aetiologies, in decreasing order of frequency, are internal hernia, adhesive obstruction, stenosis at the jejunojejunostomy, and incisional hernia. The diagnosis of bowel obstruction after laparoscopic RYGB is more difficult than after other surgical procedures secondary to the altered gastrointestinal anatomy created by the

procedure and the often less typical response of the patient with morbid obesity. After RYGB, the symptoms of bowel obstruction can be vague, and because the most common etiology is internal hernia, the symptoms are often intermittent. Abdominal pain is the most common symptom present in 82% of patients in one large series, and importantly, nausea and vomiting were seen in fewer than 50% of patients in this series. All three symptoms were present in only 28% of patients.

Unfortunately, imaging studies also have a lesser sensitivity for bowel obstruction in patients after RYGB, with reported sensitivities of 51%, 57%, and 33% for CT, UGI contrast study, and plain abdominal radiography, respectively. When patients with unexpected gastrointestinal symptoms after RYGB are assessed, a high index of suspicion for bowel obstruction is warranted. Given the frequency of internal hernia as a cause of postoperative bowel obstruction and the low sensitivity of radiologic evaluation for bowel obstruction in patients after RYGB, a low threshold for laparoscopic exploration is warranted in patients with suspected bowel obstruction. Internal hernia is the most common cause of bowel obstruction after RYGB. Anatomically, there are three different types of internal hernias seen after RYGB. All three types of internal hernias are trans mesenteric defects created during the formation of the Roux limb. The so-called Peterson hernia occurs in the infracolic compartment through the potential space between the mesentery of the Roux limb, the transverse mesocolon, and the retroperitoneum, and can be seen with either an antecolic or retrocolic Roux limb. Herniation through the mesenteric defect created by the jejunojejunostomy is the second site of internal hernia observed after RYGB and can occur with both antecolic and retrocolic gastric bypass. Herniation through the mesenteric defect in the transverse mesocolon created by passage of the retrocolic Roux limb is the third type of internal hernia observed in RYGB and is only seen in retrocolic gastric bypass; this type of internal hernia was the most common type before the importance of meticulous closure of this defect was appreciated. Most authors believe that bowel obstruction after RYGB is substantially more common after laparoscopic retrocolic bypass, with reported rates of 3.2%

to 5.1% after retrocolic and 0.3% to 1.7% after antecolic bypass reported in the largest series. Meticulous closure of all potential hernia spaces with nonabsorbable suture at the time of RYGB is the best way to prevent internal hernia; however, care must be taken when closing the mesocolic defect, because obstruction at the mesocolic window from tight scar formation has also been reported as a cause of bowel obstruction after RYGB. When operating on a patient with internal hernia after RYGB, careful closure of the hernia defect with nonabsorbable suture after reduction in the hernia is the treatment of choice.

RADIATION ENTEROPATHY

The management of radiation enteropathy is often difficult and frustrating. The clinical presentation can be quite diverse with recurrent intermittent small bowel obstruction, a true, chronic, persistent partial small bowel obstruction, or chronic diarrhoea/malabsorption. Operative management is often extremely challenging secondary to the dense adhesions and chronic inflammatory reaction present after radiation. These patients also tend to develop recurrent areas of enteropathy consistent with progression of disease in bowel that appeared normal previously, because this ischemic disease is an ongoing and progressive chronic process. The need for operative correction with a resection and anastomosis has been reported to have a mortality rate as high as 21% in some series. Patients with radiation enteropathy also have a high rate of anastomotic leak and fistula formation after operation because of the compromised vascular supply to the bowel. These effects are magnified in patients with atherosclerosis, hyperlipidaemia, or type 2 diabetes. For these reasons, a cautious, conservative approach to the patient with radiation enteropathy is warranted whenever possible. When operative management is necessary, the surgeon must decide between resection, bypass of the affected segment, or adhesiolysis. As noted earlier, resection has been reported to have a high mortality rate, with a 36% incidence of leak after primary anastomosis. Surgeons advocating aggressive resection back to healthy bowel, however, have reported leak rates between 0% and 8% when confounding conditions (abscess, fistula,

necrosis, or recurrent cancer) were absent; such an aggressive approach may require an extensive resection but often involves resection of non-functional bowel anyway. Given the complexities in managing radiation enteropathy, implementation of a scoring system may help direct care and improve outcomes. Short bowel syndrome is always a concern, especially because the involved bowel is usually the distal ileum.

Most surgeons approach the treatment of radiation enteropathy cautiously. In those patients with recurrent cancer and radiation enteropathy, treatment should consist of palliative bypass of the diseased segment with creation of an anastomosis in visibly normal tissue. If the obstructive process is localized, wide resection back to healthy, non-irradiated tissue (if possible) with primary anastomosis is acceptable, provided adequate absorptive area is preserved. Usually this involves anastomosis from small bowel to the ascending colon, because the terminal ileum has usually been within the radiation field. While ideally a complete resection of the entire involved small bowel is optimal, the surgeon must consider the extent of the resection necessary as well as the anatomic segment involved. Because the distal ileum is commonly involved, major resection back to reliably normal, nonirradiated small bowel may require a total or subtotal ileal resection that carries its own nutritional complications. Thus, the surgeon is faced with a decision concerning preservation of mildly involved but functional ileum versus complete resection. In contrast, if the bowel is severely involved and non-functional, resection, despite its side effects, may be the best option. When the affected area contains dense adhesions or is stuck deep within the pelvis, bypass may be a better choice to avoid the very real concern of potential iatrogenic injury to the bowel, bladder, pelvic organs, and ureters, however, if there is a localized abscess or associated septic process, bypass is not a good option because the ischemic inflammatory process will continue. Attempts at complete lysis of adhesions alone without resection are controversial due to the risk of traumatizing the intestine with potential fistula formation. For the patient with advanced disease who presents years after irradiation, adhesiolysis may not be a good option, especially if the bowel is matted and agglutinated. In contrast, in the case of isolated

adhesive bands and the patient being early (<2 years) after irradiation, lysis alone may be warranted; much of the decision needs to be based on the quality of the involved bowel and the site of obstruction. If the bowel is thickened, nonpliable, and strictured, resection or bypass is best.

CARCINOMATOSIS AND MALIGNANT OBSTRUCTION

Bowel obstruction in the setting of carcinomatosis often represents the terminal phase of the malignant disease. Operative management is entirely palliative and needs to be selectively. In the case of limited life expectancy and malignant cachexia or ascites, nonoperative palliative measures are advised because operative intervention would be unnecessary and associated with a poor quality of life due to the convalescence required after a non-curative celiotomy. However, some patients with a good performance status may have a long-life expectancy, and in this case, operative bypass with the idea of permitting renewed oral intake may be indicated. Patients and their families should be counselled that the relief of their obstruction will not affect disease progression but may improve quality of life. addition, the surgeon should keep in mind that up to one-third of bowel obstructions presenting in the setting of carcinomatosis are due to adhesions and not to malignant obstruction. Therefore, a short trial of conservative therapy with rehydration and nasogastric decompression is usually advisable, although many patients with carcinomatosis will fail this intervention. In addition, depending on the location and extent of the malignant disease involving the gastrointestinal tract, a palliative endoscopic stent placement may relieve the obstruction. An initial minimal access, laparoscopic approach should at least be entertained in patients with a malignant obstruction, provided the access to the peritoneal cavity is safe. The least invasive approach is best for these patients, and if palliation, such as a bypass or gastrostomy tube, can be achieved laparoscopically, the patient would benefit substantially

with decreased pain, possibly a shorter convalescence, and decreased duration of hospital stay, all of which are important considerations in the palliative care of patients with a limited life expectancy. At exploration, multiple scenarios may be encountered. Some patients will have an isolated area of adhesions and require only adhesiolysis. Others will have a solitary metastasis causing either an intra- or extraluminal obstruction that can be corrected with a limited resection or bypass. If multiple areas of adhesions are present or the affected area is adherent to the abdominal wall or intra-abdominal structures in the patients with incurable malignant obstruction, bypass of the involved segment will provide symptom relief and the fewest opportunities for complication.

One should consider placement of a tube gastrostomy if there is any question of the success of the operation, if impending obstruction seems imminent, or if relief of the obstruction is not possible. In the event of recurrent obstruction, a tube gastrostomy can be used to decompress the stomach and avoid the discomfort associated with a nasogastric tube. The decision to place a palliative, decompressive, tube gastrostomy is more difficult in the presence of ascites. In this situation, better option would be a tube pharyngostomy. In addition, if histologic diagnosis of the neoplasm had been obtained previously, a repeat biopsy should be entertained to ensure that the neoplasm has histologic characteristics consistent with the original biopsy.

SERUM LACTATE

Lactate is a conjugate base (anion) of lactic acid. It is the primary endogenous agonist of hydroxy carboxylic acid receptor 1(HCA1), a G protein coupled receptor.

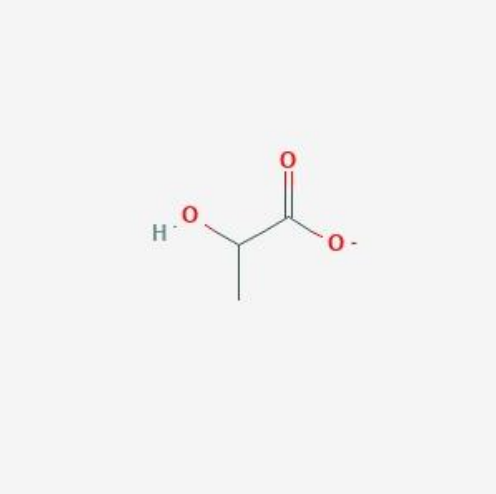


FIGURE - 9 : LACTATE ION

Lactate exists as two different isomers : D – lactate and L – lactate. L – lactate is the end product of anaerobic glycolysis. During this process it is formed out of pyruvic acid by the enzyme lactate dehydrogenase (LDH). During ischemia the cells will start anaerobic dissimilation and the serum lactate rises.

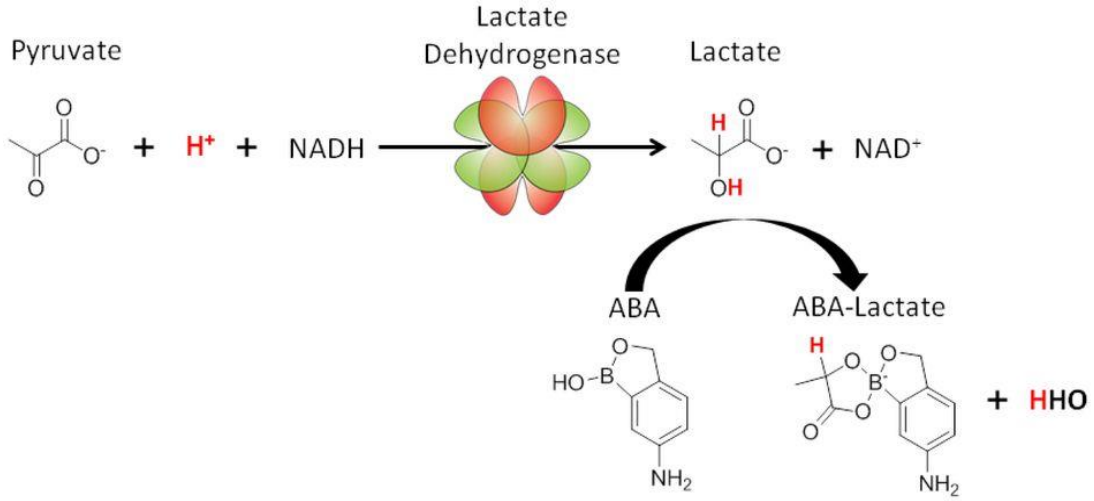


FIGURE - 10 : LACTATE FORMATION

Lactate is absorbed mostly by liver but partially by the kidney. Liver accounts for

approximately 50% and the kidney for about 30% of whole body lactate uptake. In the liver lactate is converted back to pyruvate and by gluconeogenesis to glucose. Thus an increased serum lactate can be a result of tissue hypoperfusion as well as a decreased lactate metabolism in the liver or kidney. Experimental studies have shown that the liver is able to increase lactate uptake in case of excessive mesenteric lactate production so that an increased serum L – lactate can be compensated. D – lactate is not produced by the human body but released by intestinal bacteria. Increased D – lactate during intestinal ischemia might be caused by overgrowth of these bacteria. D isomer is metabolised by D – LDH enzyme. In daily care, lactate measurement includes L – lactate which is measured using an enzymatic reaction between L – lactate and either L – lactate oxidase or L – LDH. Both of these enzymes are specific for L – lactate. Therefore D – lactate is not routinely obtained when measuring L – lactate. D -lactate is measured by using an enzymatic reaction with D – LDH, which is not available in most hospitals.

OVERVIEW OF LACTATE METABOLISM

Lactate is the end product of anaerobic glycolysis and is converted from pyruvate by lactate dehydrogenase (LDH). Upon its release from peripheral tissues, it arrives in the liver and undergoes LDH mediated conversion to pyruvate, which can then be recruited for gluconeogenesis or oxidative phosphorylation in the liver, the latter being catalysed by pyruvate dehydrogenase (PDH). Hence in case of strenuous exercise, it serves as a fuel during such periods of increased anaerobic metabolism.

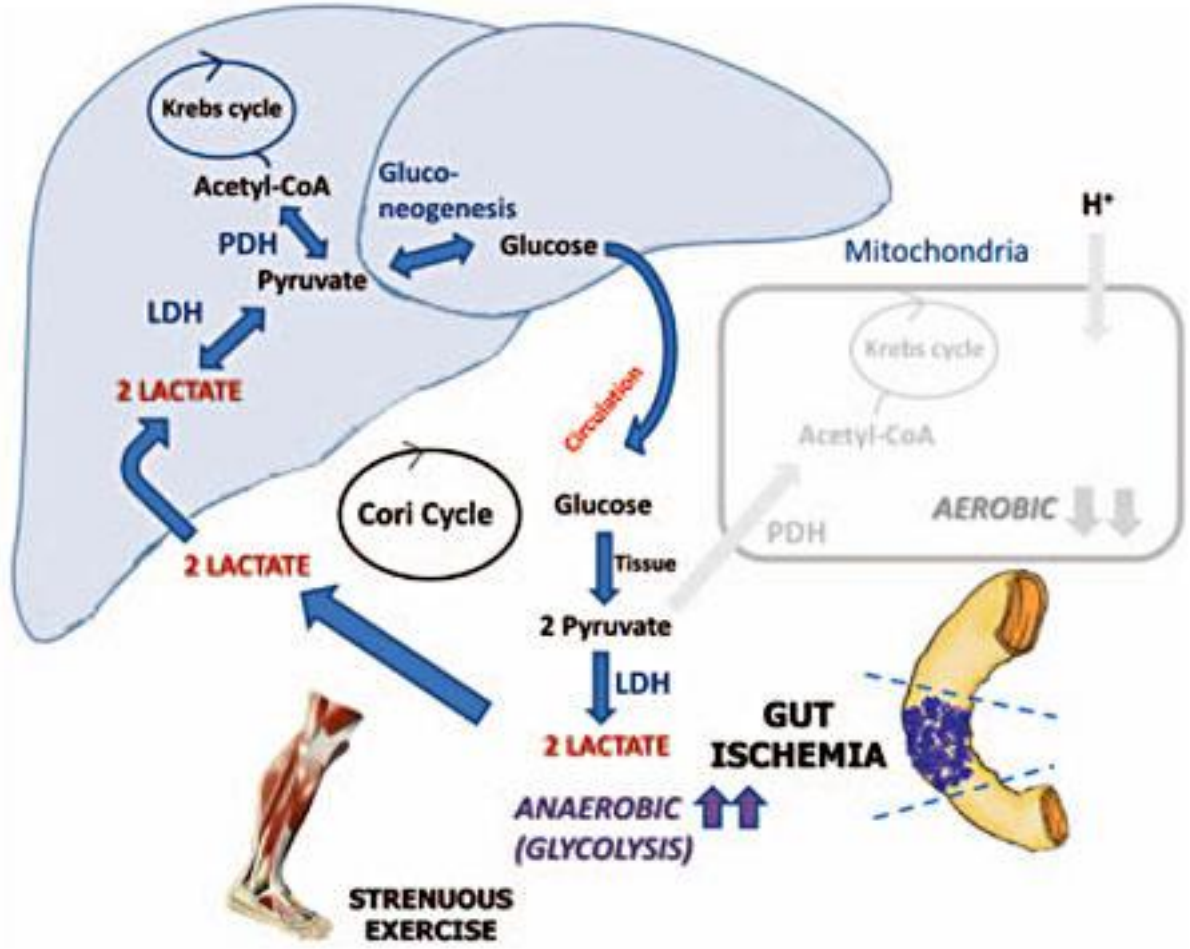


FIGURE - 11 : LACTATE METABOLISM

Serum lactate does not cause acidosis per se. Rather, an acidotic state results from the diminished recruitment of H+ ions into the mitochondria during states of suppressed oxidative phosphorylation.

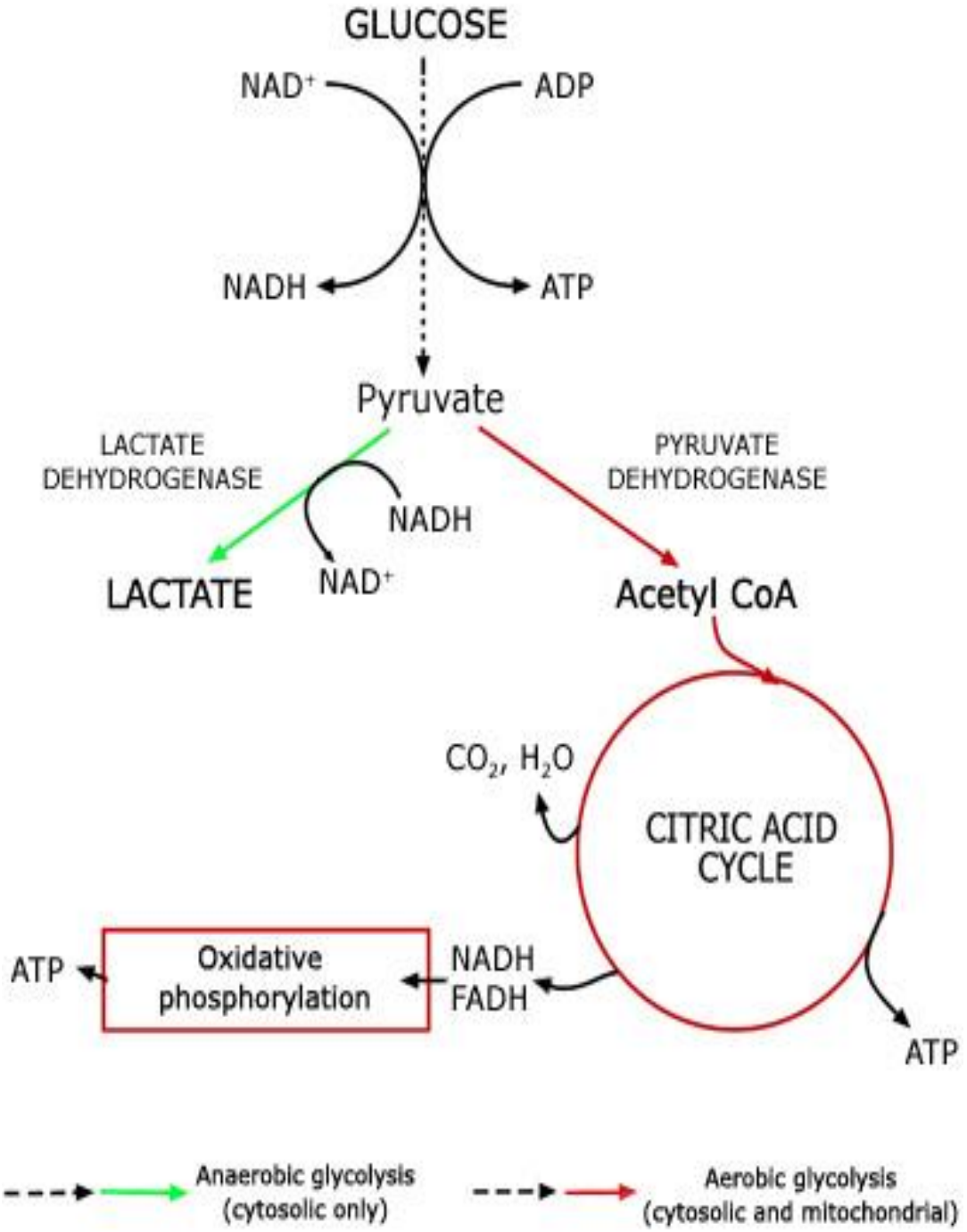


FIGURE - 12 : LACTATE METABOLISM BETWEEN METABOLIC PATHWAYS

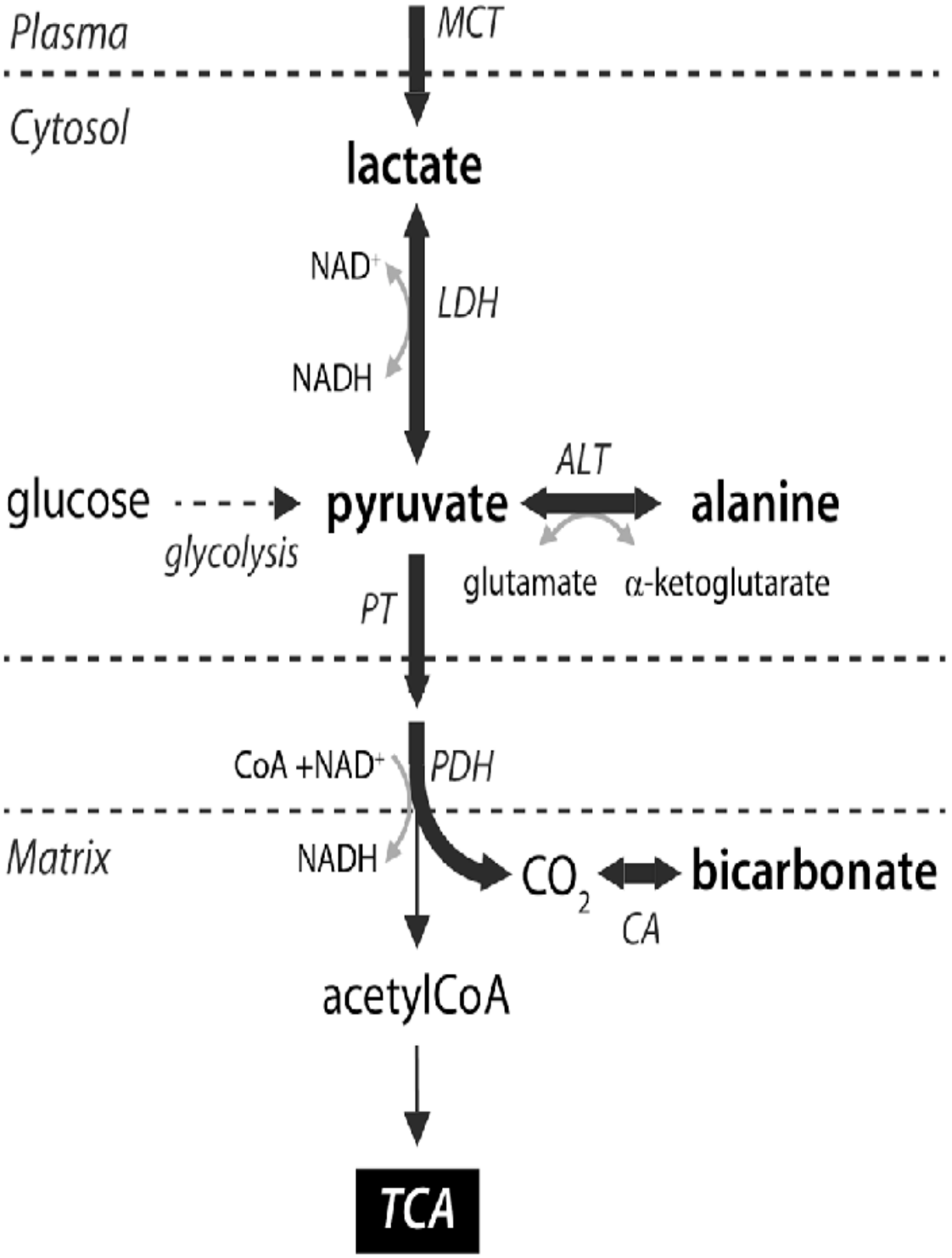


FIGURE - 13 : LACTATE METABOLISM AT INTRACELLULAR LEVEL

An anaerobic metabolism is also assumed to take place in the ischemic gut during acute mesenteric ischemia, which would result in increased lactate release from the gut into the portal vein. For serum lactate in the general circulation to be elevated, the amount of released lactate from the ischemic gut must exceed the conversion capacity of the liver. Therefore increased serum lactate is rather a marker of anaerobic metabolism and can undergo elevation during hepatic failure but also other non hypoxic states such as diabetes, malignancies or congenital disorders of lactate metabolism.

SERUM LACTATE AND STRANGULATION : A HISTORICAL VIEW

Increased serum lactate levels were initially reported by studies on intestinal vascular occlusion during surgical interventions involving reconstruction of aortic or intestinal vessels. From the end of the 1980s to the mid 1990s, measurement of serum lactate was shown to be beneficial in diagnosing acute mesenteric ischemia in some European case series. Among the earliest case series reporting elevated serum lactate, the study by Janda et al. demonstrated tenfold increased serum lactate levels among patients who developed postoperative occlusion of intestinal arteries as opposed to uncomplicated cases. Among patients with acute abdomen and intestinal vascular occlusion there was 7 fold increase in serum lactate as opposed to patients without intestinal ischemia. Nutz and Sommer noted that the rationale of measuring serum lactate is the high demand of the intestine for blood and oxygen and thus its high ischemic vulnerability. They postulated that in the presence of ischemia, intestinal cells would depend on anaerobic metabolism, resulting in acidosis and a serum lactate increase even before necrosis of the tissue.

Table 1. *Diagnostic value of L-lactate for acute mesenteric ischaemia*

Study	Design	Results	Conclusion
Lange 1994[8]	Prospective, patients with acute abdominal symptoms, n = 85	Serum L-lactate before diagnosis or preoperatively increased in all patients with MI (n = 20), but also in many other patients	**Sensitivity 100%** **Specificity 42%**
Meyer 1998[9]	Retrospective, patients operated for AMI, n = 46	Serum L-lactate preoperatively increased in > 90%* of patients with AMI. Only 19 patients operated < 6 hours, 12 > 24 hours after presentation	**Sensitivity > 90%***
Lange 1997[10]	Prospective, patients with abdominal pain, n = 120	Serum L-lactate increased in 24 of 25 patients with MI, but also in many other patients	**Sensitivity 96%** **Specificity 36%**
Gearhart 2003[11]	Prospective, patients clinically suspected for AMI, n = 58	Serum L-lactate increased in 24 of 31 patients with AMI, 8 false-positively increased	**Sensitivity 78%** **Specificity 53%**
Cronk 2006[12]	Prospective, patients admitted for mechanic bowel obstruction, n = 21	Serum L-lactate increased in 1 of 3 patients with gut necrosis, 5 false-positively increased	**Sensitivity 33%** **Specificity 72%**
Acosta 2012[13]	Retrospective, patients with AMI, n = 55	Serum L-Lactate increased in 14 of 27 patients (other patients had no L-lactate measurement)	**Sensitivity 52%**
Van der Voort 2014[14]	Prospective, patients on ICU clinically suspected of AMI, n = 44	Serum L-lactate increased in 18 of 23 patients with AMI	**Sensitivity 78%** **Specificity 48%**

N = number of patients; (A)MI= (acute) mesenteric ischaemia; ICU = Intensive Care Unit. *Not further specified

ARTERIAL VS VENOUS LACTATE

Lactate valves differ for venous and arterial blood. Normal ranges vary among laboratories. Normal value for venous blood is 0.5 to 2.2 mmol/L and for arterial blood is 0.5 to 1.6 mmol/L. Elevated lactate of any variety is considered abnormal and the same should be evaluated. Either collection is appropriate for bundle compliance. Lactate elevations may be influenced by various medications, hepatic insufficiency or hyperlactatemia due to primary cardiac causes of hypoperfusion. Strenuous exercise, metformin ingestion, iodine containing contrast media for radiological studies may elevate serum lactate. Lactate measurements from arterial blood are thought to be more accurate because tourniquet is not used, they are not generally affected by the collection process.

COHEN – WOODS CLASSIFICATION

The Cohen-Woods classification categorises causes of lactic acidosis as follows

Type A : Decreased perfusion or oxygenation

Type B : B1 : Underlying disease (sometimes causing type A)

B2 : Medications or intoxicated

B3 : Inborn error of metabolism

RESULTS

AGE DISTRIBUTION

The mean age of the patients is 54.44 years with standard deviation of 16.13 years, ranging between 15 years to 86 years. The following table and figure shows the age distribution of the participants.

Characteristics	Age in years
Mean	54.44
Median	54.00
Mode	65
Std. Deviation	16.134
Minimum	15
Maximum	86

TABLE 1 : AGE DISTRIBUTION

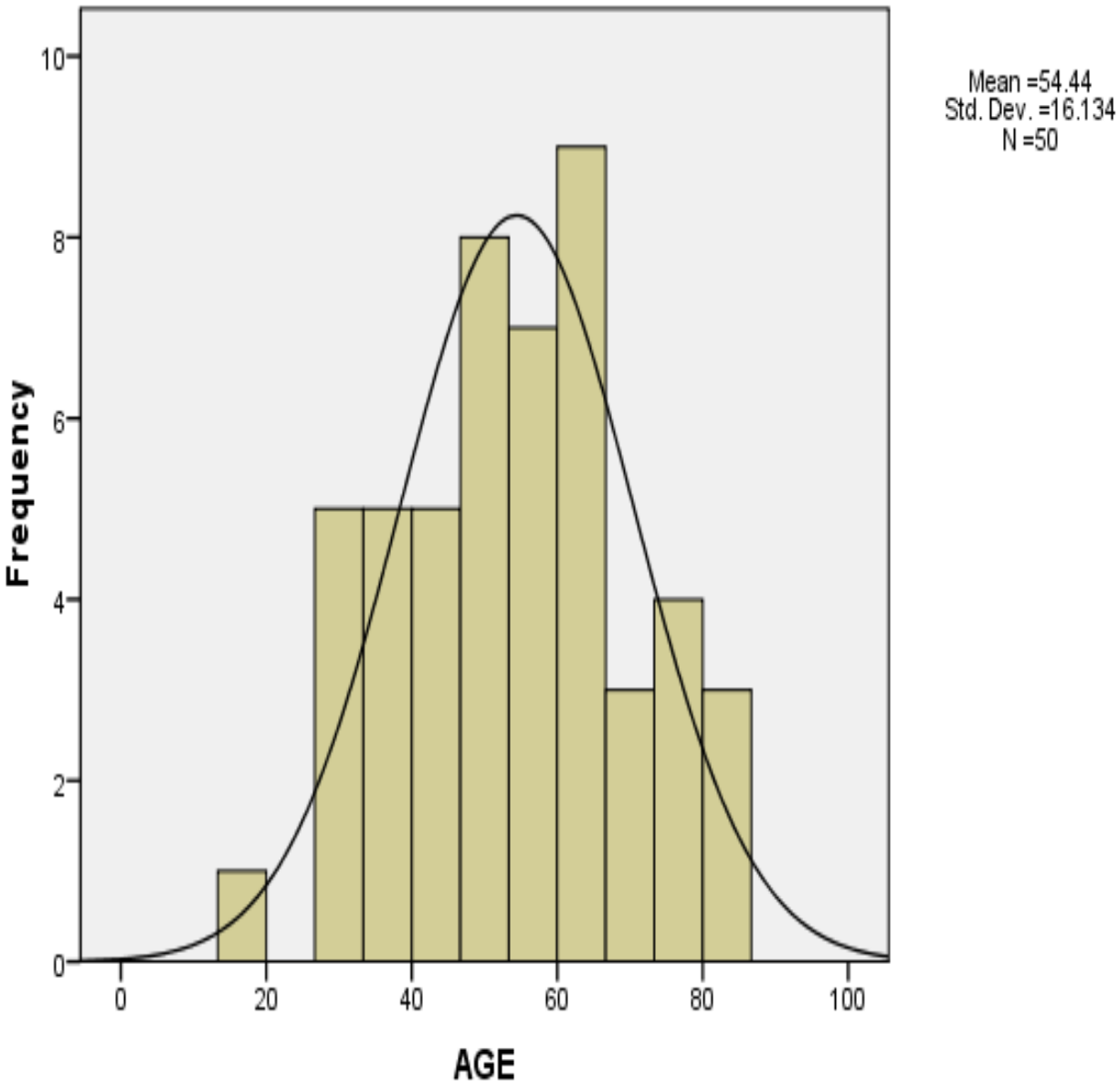


FIGURE 14: AGE DISTRIBUTION

GENDER DISTRIBUTION

Out of 50 patients, 29 (58%) of them are males and 21 (42%) of are females.

Gender	Frequency	Percent
Female	21	42.0
Male	29	58.0
Total	50	100.0

TABLE 2: GENDER DISTRIBUTION

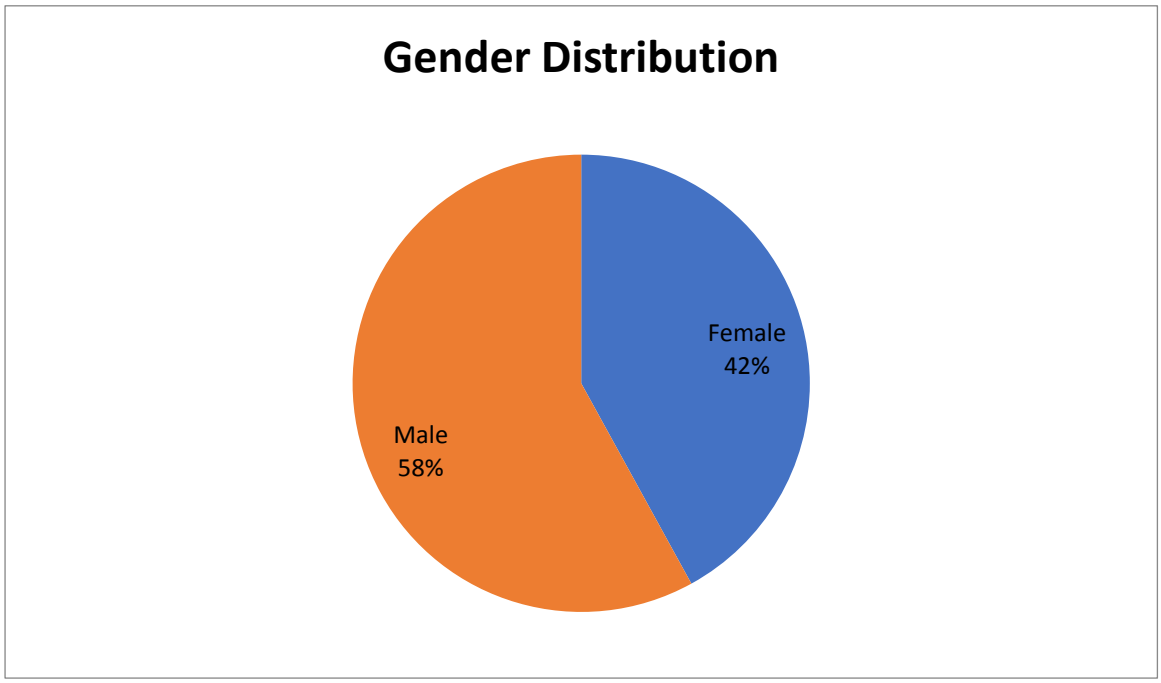


FIGURE 15: GENDER DISTRIBUTION

SYMPTOMS

The following table and figure shows the symptoms of the patients. All of them had tenderness, eight (16%) of them had shock, 35 (70%) had guarding and 32 (64%) of they had exaggerated bowel sounds.

Symptom	Frequency	Percentage
Shock	8	16
Tenderness	50	100
Guarding	35	70
Bowel Sounds		
Absent	18	36
Exaggerated	32	64

TABLE 3: SYMPTOMS

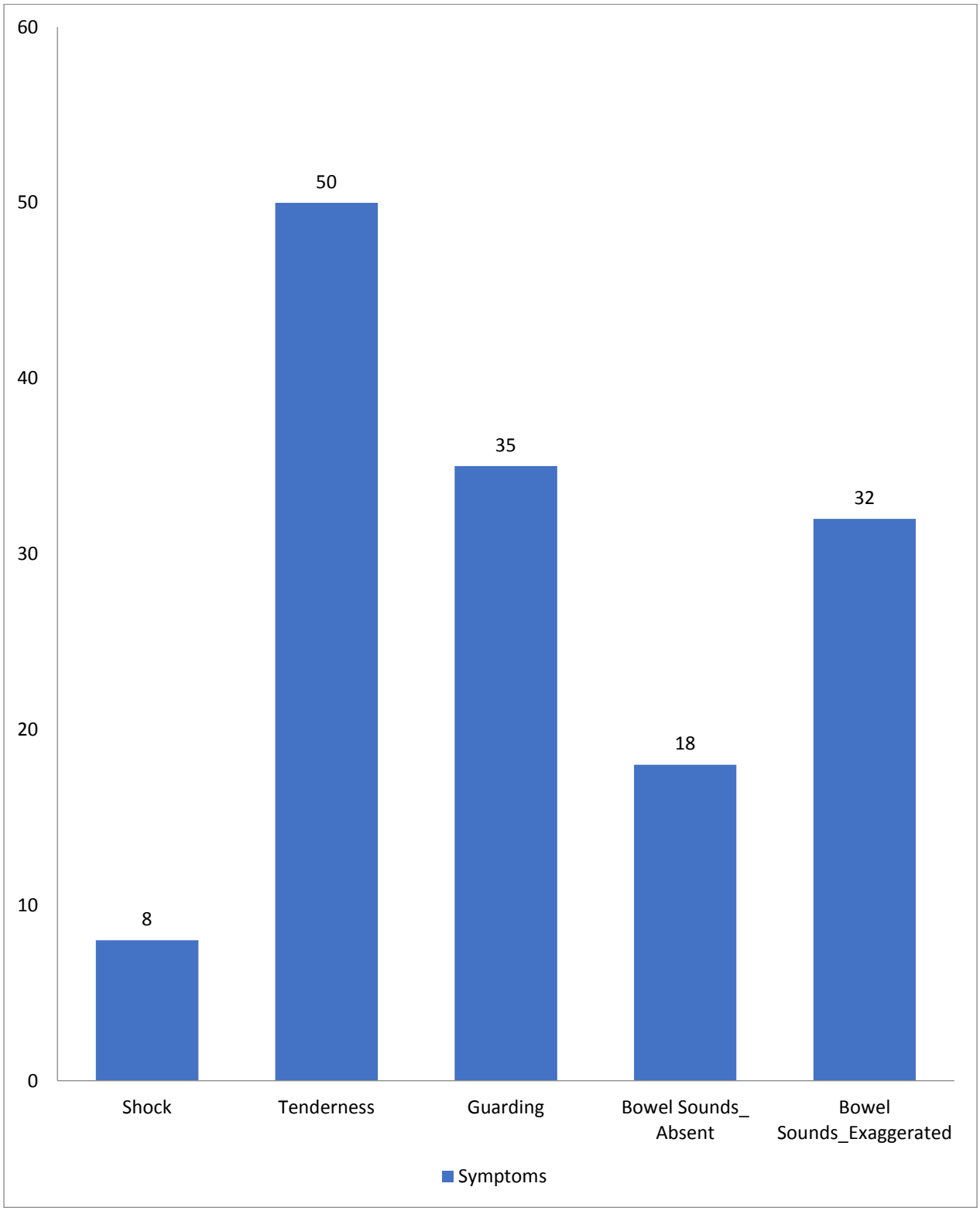


FIGURE 16: SYMPTOMS

DIGITAL RECTAL EXAMINATION

The digital rectal examination shows that it was roomy in 24 (48%) of the patients while in 26 (52%) of the patients, it was empty.

Digital rectal Examination	Frequency	Percent
EMPTY	26	52.0
ROOMY	24	48.0
Total	50	100.0

TABLE 4: DIGITAL RECTAL EXAMINATION

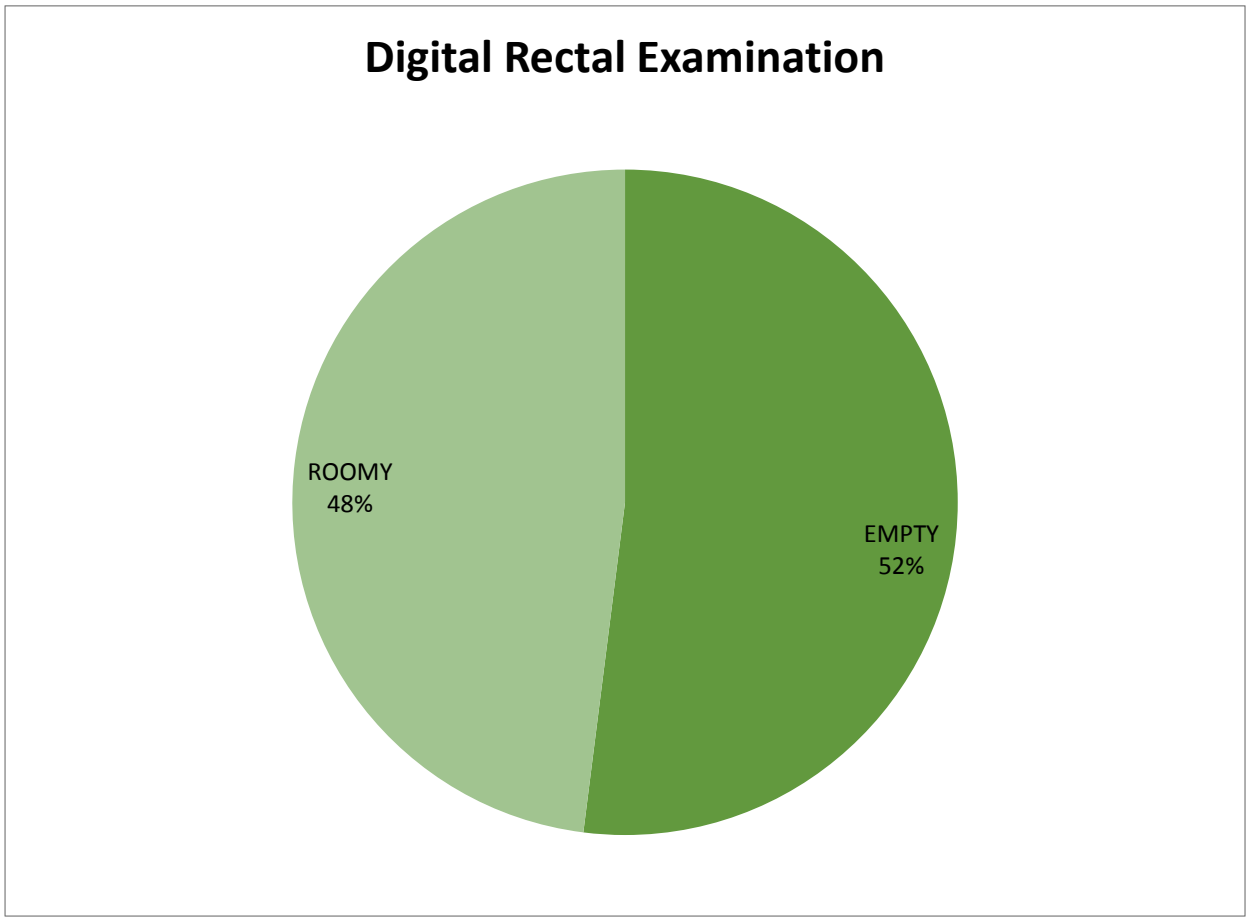


FIGURE 17: DIGITAL RECTAL EXAMINATION

COMORBIDITIES

None of them had any comorbidity

HISTORY OF PREVIOUS SURGERY

Around 36% (n=18) of the patients had history of previous surgery.

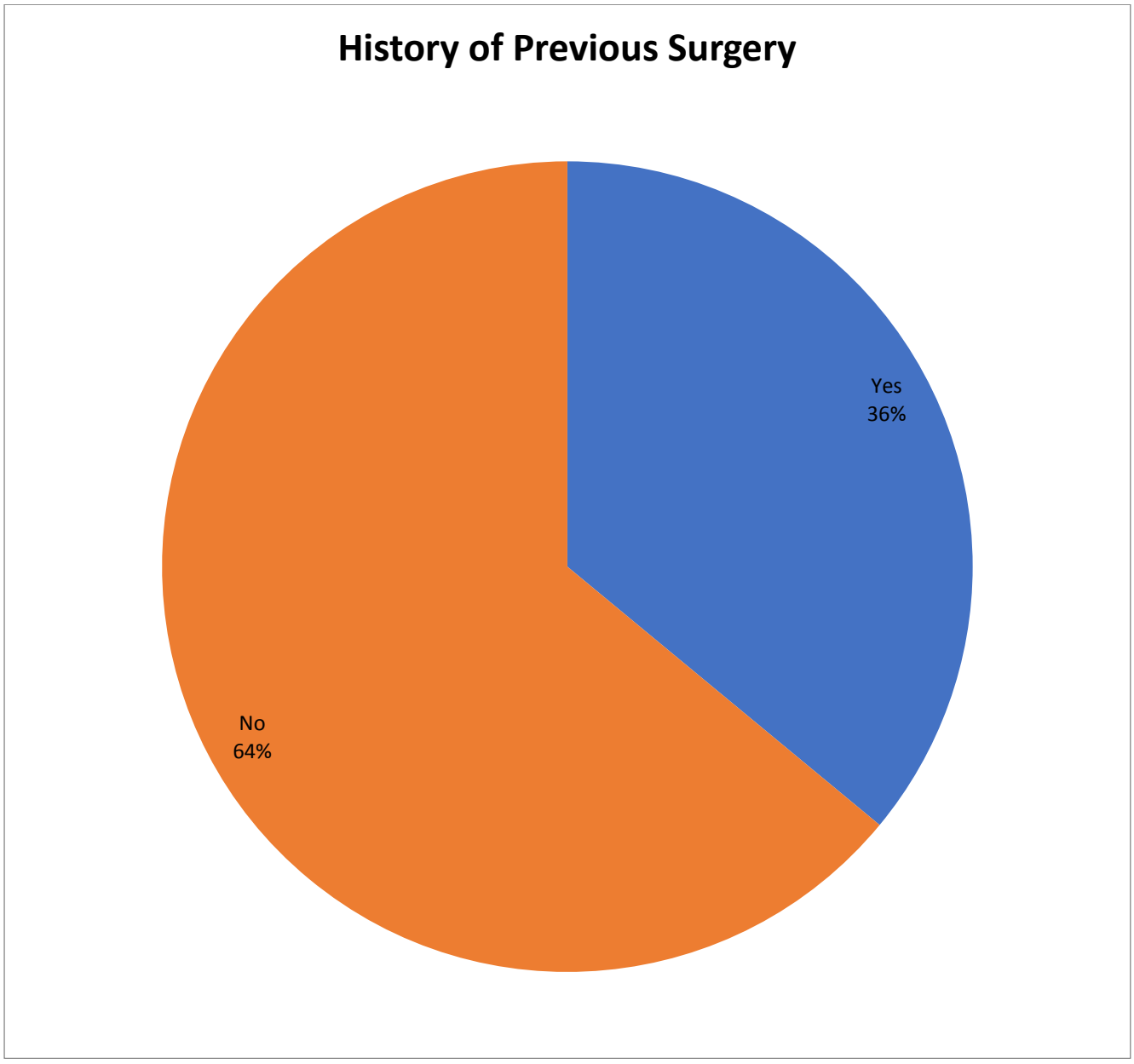


FIGURE 18: HISTORY OF PREVIOUS SURGERY

DIAGNOSIS

The following table shows the diagnosis of the patients.

DIAGNOSIS	Frequency	Percent
ACUTE INTESTINAL OBSTRUCTION	21	42.0
OBSTRUCTED INCISIONAL HERNIA	6	12.0
OBSTRUCTED LEFT INGUINAL HERNIA	4	8.0
OBSTRUCTED RECURRENT INCISIONAL HERNIA	1	2.0
OBSTRUCTED RIGHT INGUINAL HERNIA	6	12.0
OBSTRUCTED RIGHT PARAUMBLICAL HERNIA	1	2.0
OBSTRUCTED UMBILICAL HERNIA	6	12.0
SUBACUTE INTESTINAL OBSTRUCTION	5	10.0
Total	50	100.0

TABLE 5: DIAGNOSIS

ETIOLOGY

Out of the 50 cases of obstruction, 40% are due to hernia,12% are due to adhesive bands and 6% are due to postoperative adhesions.

	Intraoperative Findings	Frequency	Percent
	Postoperative Adhesions	4	8.0
	Adhesion Bands	6	12.0
	Hernia	40	80
	Total	50	100.0

TABLE 6: ETIOLOGY

LEVEL OF OBSTRUCTION

Out of the 50 cases, 96% are small bowel obstruction and remaining 4% are large bowel obstruction.

Level of obstruction	Frequency	Percent
Small Bowel	48	96
Large Bowel	2	4

TABLE 7 : LEVEL OF OBSTRUCTION

STRANGULATION

Out of 50 patients, 72% of them (n=36) had strangulation while the rest had no strangulation.

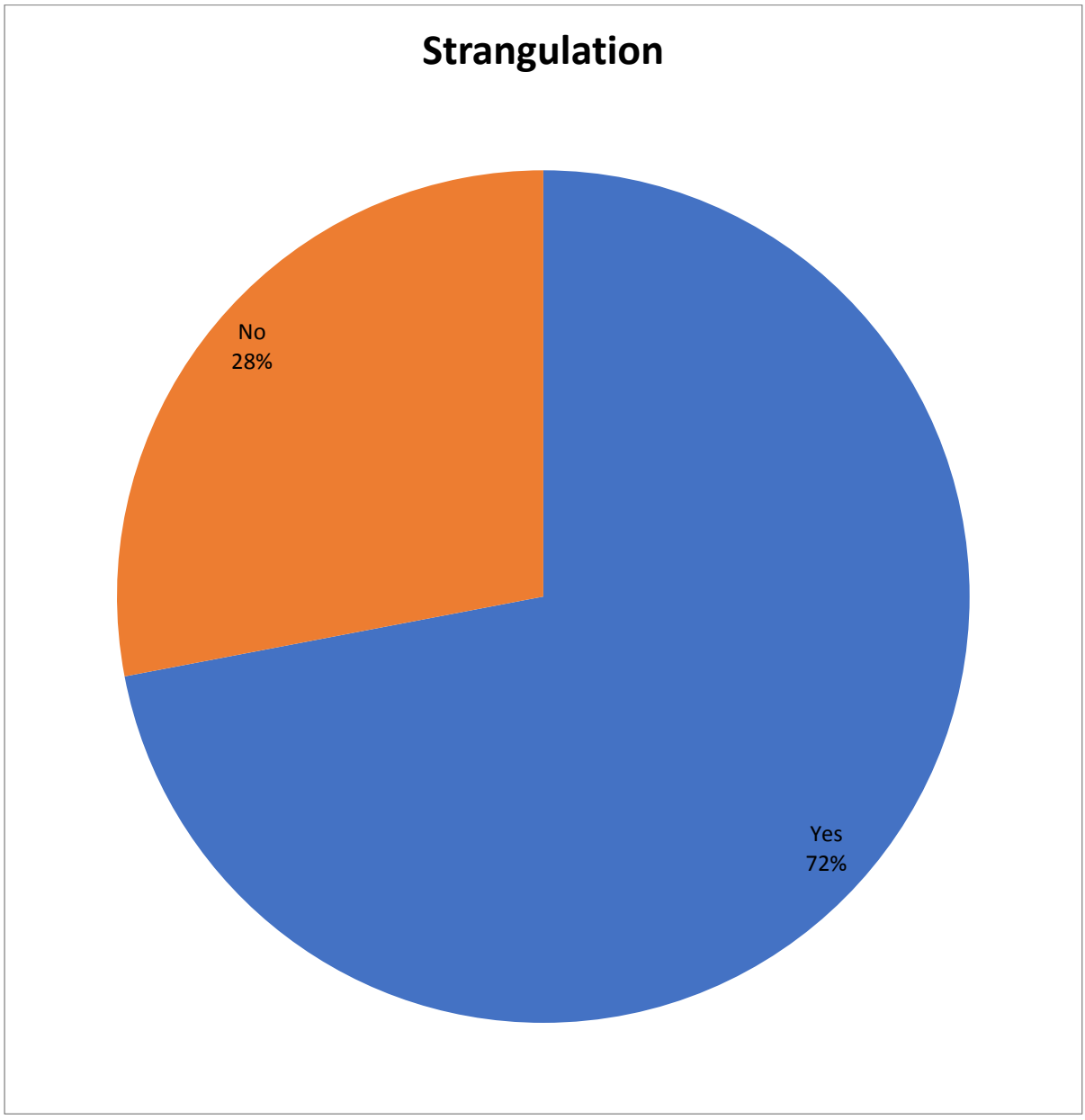


FIGURE 19: STRANGULATION

SERUM LACTATE LEVEL

The mean serum lactate level in the patients are 5.16 mmol/L (S.D=1.708) ranging between 2-9 mmol/L.

Characteristic	Serum lactate mmol/L
Mean	5.16
Median	5.30
Mode	6
Std. Deviation	1.708
Minimum	2
Maximum	9

TABLE 8: SERUM LACTATE LEVELS

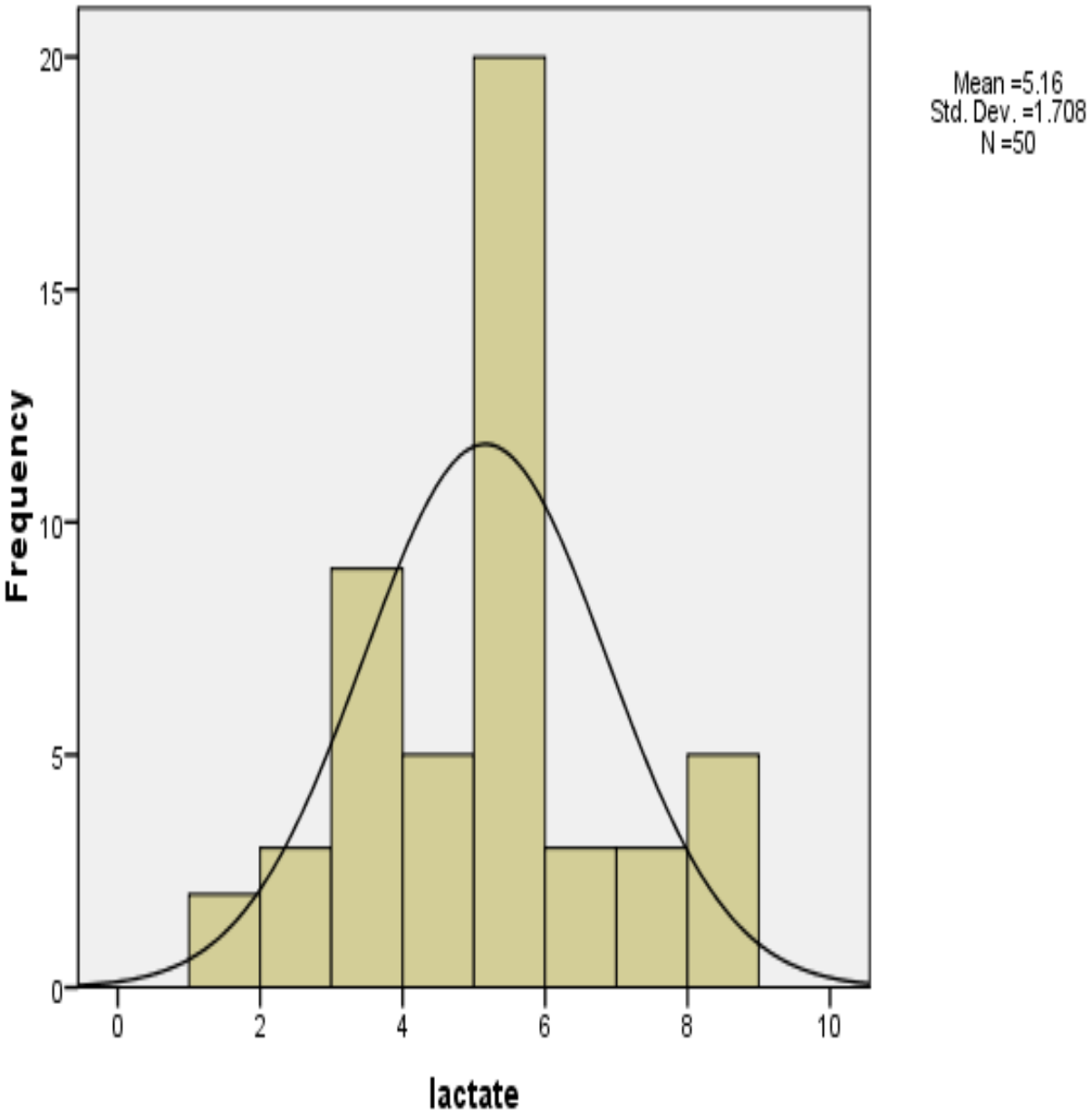


FIGURE 20: SERUM LACTATE LEVELS

CLASSIFICATION OF PATIENTS BASED ON SERUM LACTATE VALUES

Based on the serum lactate levels, 72% of them were classified as strangulation, 6% of them as normal and 22% of them with strong suspicion.

Classification	Frequency	Percent
Normal	3	6.0
Strangulation	36	72.0
Strong Suspicion	11	22.0
Total	50	100.0

TABLE 9: CLASSIFICATION OF PATIENTS BASED ON SERUM LACTATE VALUES

COMPARISON OF SERUM LACTATE CLASSIFICATION WITH ACTUAL STRANGULATION

Chi-square analysis shows that serum lactate levels significantly differ in groups with and without strangulation.

		STRANGULATION Present		Total	Chi-Square p-value
		NO	YES		50.00 P=0.00653 Highly Significant
Classification	Normal	3	0	3	
	Strangulation	0	36	36	
	strong suspicion	11	0	11	
Total		14	36	50	

TABLE 10: CHI-SQUARE ANALYSIS

COMPARISON OF SERUM LACTATE VALUES BETWEEN DIFFERENT GROUPS

Kruskal-Wallis Test for comparison of serum lactate values across the three groups with normal, strangulation and strong suspicion shows that the results are significant with a chi-square value of 30.23 with p=0.00123 (highly significant).

		Normal	Strong Suspicion	Strangulation
Patients		3	11	36
Mean		1.8333	3.4091	5.9722
Median		1.8000	3.6000	5.6000
Mode		1.70[a]	3.50[a]	5.60
Std. Deviation		.15275	.61230	1.17731
Minimum		1.70	2.10	4.60
Maximum		2.00	3.90	8.60

TABLE 11: COMPARISON OF SERUM LACTATE VALUES BETWEEN DIFFERENT GROUPS

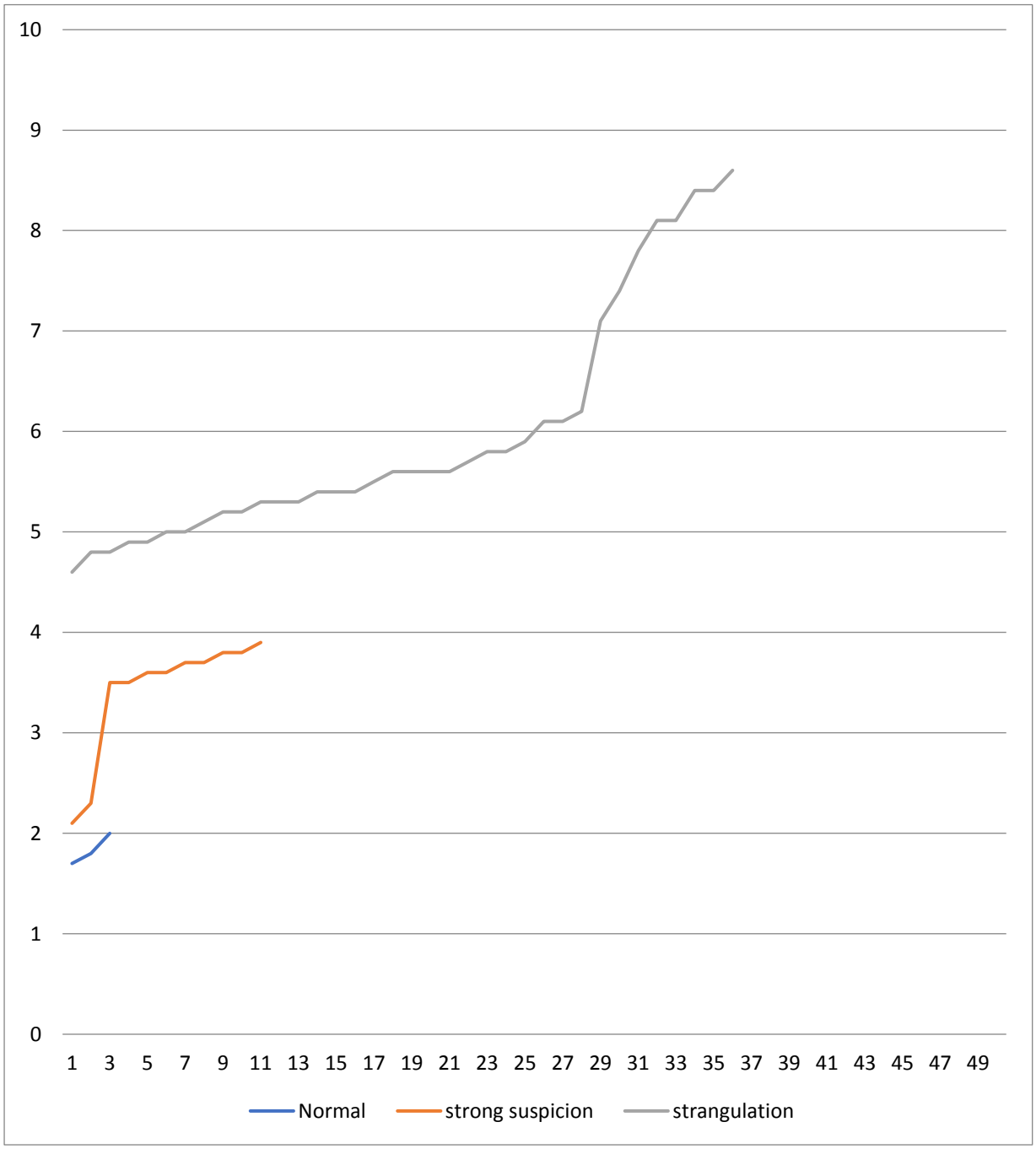


FIGURE 21: COMPARISON OF SERUM LACTATE VALUES BETWEEN DIFFERENT GROUPS

DISCUSSION

Acute intestinal obstruction possess strangulation as a grave complication and requires prompt diagnosis. This is easier said than done, especially in an emergency setting. Acute intestinal obstruction with reported mortality rates have found association with delay in surgical management with progression to strangulation in many cases.

Most of the lactate found in the human body is L – lactate. Van Noord studied 49 patients with chronic gastrointestinal ischemia and found that L – lactate elevation was significantly increased as compared with the nonischaemic group. Markogiannakis et al had also reported finding in favour of serum lactate as predictor of ischemia and strangulation.

In this study a group of 50 cases of intestinal obstruction with no comorbidities had been taken for assessment of various outcomes of obstruction and their relation to levels of serum lactate. Out of the 50 cases, 96% were small bowel obstruction and 4% were large bowel obstruction with a mean age of distribution 54.44 years. About 36% of the sample, had previous history of surgery of which 6% had postoperative adhesions causing obstruction or strangulation. With regard to operative findings, hernia and adhesions were the most common findings. Out of 50 patients, 72% had strangulation and bowel gangrene as intraop findings and the rest were simple obstructions. The mean serum lactate value in the patients were 5.16 mmol/L ranging between 1.70 to 8.60 mmol/L. The cut off values of strangulation, simple obstruction and those subacute cases managed conservatively were 4.3,3.4 and less than 2.3 respectively. Based on these values, 72% were classified as strangulation,22% with strong suspicion and 6% of them were normal. Kruskal-Wallis test for comparison of serum lactate values across the three groups with normal, strong suspicion and strangulation shows that the results are significant with a chi-square value of 30.23 with p=0.00123(highly significant).

The study has shown that serum lactate levels were significantly raised in strangulated bowel obstruction as compared with simple bowel obstruction.

The main strength of this study lies in the fact that it is a prospective study with applicability in an emergency setting in a developing country, where availability of computed tomography (CT) and other costly biomarkers is beyond the reach of poor patients. Also, this study included both small and large bowel obstruction.

LIMITATIONS

1. Study participants were less in number
2. Patients of paediatric age group were not a part of this study
3. Only serum lactate had been studied as a marker which is naturally inferior when compared to a study with combination of other parameters
4. D-lactate had not been studied due to the high cost of the kit
5. Radiological, postoperative outcome, length of hospital stay and follow up were not assessed by markers with regard to diagnosis of strangulation.

CONCLUSION

A positive correlation between elevated serum lactate and strangulation bowel obstruction had been established via this study. Further studies incorporating various biomarkers and their correlation with clinical presentation and radiological findings should be sought. Such studies would help in reducing the time interval to surgery in cases of acute intestinal obstruction with strangulation as well as decreasing unwarranted laparotomy in those cases of intestinal obstruction without strangulation, that can be managed conservatively depending on other parameters and clinical findings. These biomarkers can be made readily available in the emergency setting after due consideration given to their clinical relevance at the institutional level. This study does add to the current literature regarding the need of decision-making policy for management of acute intestinal obstruction incorporating the role of biomarkers for predicting strangulation at the time of presentation.

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ANNEXURES

PROFORMA

Name:

Age:

Gender:

Duration of symptoms :

1. Abdominal pain -

2. Abdominal distension –

3. Vomiting –

4. Fever –

5. Obstipation –

H/O previous abdominal surgery

SIGNS AT THE TIME OF PRESENTATION

- SHOCK (SBP < 90mmHg) – YES / NO
- ABDOMINAL TENDERNESS – YES / NO
- GUARDING/ RIGIDITY – YES / NO
- EXAGGERATION OF BOWEL SOUNDS – YES / NO

GOVT.STANLEY MEDICAL COLLEGE, CHENNAI- 600 001
INFORMED CONSENT

DISSERTATION TOPIC:
A STUDY ON ASSESSMENT OF SERUM LACTATE LEVELS IN INTESTINAL OBSTRUCTION
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:

அரசு ஸ்டான்லி மருத்துவக்கல்லூரி மருத்துவமனை, சென்னை
ஒப்புதல்படிவம்
இடம் :அரசு ஸ்டான்லி மருத்துவ கல்லூரி மருத்துவமனை , சென்னை

DISSERTATION TOPIC:

A STUDY ON ASSESSMENT OF SERUM LACTATE LEVELS IN INTESTINAL OBSTRUCTION

தன்னார்வலரின் பெயர் மற்றும் முகவரி:

_____ஆகியநான், எனக்கு இந்த ஆய்வின் விவரங்களை என் சொந்த மொழியில் எனக்கு தெரிவிக்கப்பட்டுள்ளது

ஆய்வின் விவரங்களை முழுமையாக புரிந்துகொண்டேன்.

ஆய்வில் பங்கெடுக்கும் போது சாத்தியமான அபாயங்கள் மற்றும் நலன்களை நான் அறிவேன்.
நான் எப்போது வேண்டுமானாலும் படிப்பிலிருந்து பின்வாங்க முடியும் என்பதை புரிந்து கொள்கிறேன், அதோடு கூட வழக்கமாக மருத்துவ சிகிச்சையைப் பெறுவேன்.
இந்த ஆய்வில் பங்கெடுத்துக் கொள்ள எனக்கு எந்த கட்டணமும் கிடைக்காது என்று புரிந்து கொள்கிறேன்.
இந்த ஆய்வின் முடிவுகள் ஏதேனும் மருத்துவ இதழில் வெளியிடப்பட்டால், என் தனிப்பட்ட

அடையாளத்தை வெளிப்படுத்தவில்லை என்று புரிந்துகொண்டேன்.

இந்த ஆய்வில் பங்கெடுத்துக் கொள்வதன் மூலம் நான் என்ன செய்ய வேண்டும் என்று எனக்குத் தெரியும், இந்த ஆய்விற்கான எனது முழு ஒத்துழைப்பையும் நான் விரிவாக்குவதாக உறுதியளிக்கிறேன்.

தன்னார்வலரின் பெயர் மற்றும் முகவரி:
தன்னார்வலரின் கையொப்பம் / கட்டைவிரல் தோற்றம்
நாள்:

சாட்சிகள்(கையொப்பம், பெயர்&முகவரி):
நாள்:

பெயர் மற்றும் புலன் விசாரணை கையொப்பம்:

MASTER CHART

S.NO	NAME	AGE/SEX	SHOCK	TENDER ABDOMEN	GUARDING/RIGIDITY	BOWEL SOUNDS	DIGITAL RECTAL EXAMINATION	COMORBIDITIES	PREVIOUS ABDOMINAL SURGERY	DIAGNOSIS	INTRAOP FINDINGS	STRANGULATION	MANAGEMENT	SERUM LACTATE mmol/L
1	SUBAITHA BEGAM	67/F	YES	YES	YES	ABSENT	ROOMY	NO	NO	SMALL BOWEL OBSTRUCTION	ILEAL GANGRENE 40CM FROM IC JUNCTION	YES	ILEAL RESECTION WITH DOUBLE BARREL ILEOSTOMY	7.1
2	KUMAR	65/M	NO	YES	NO	EXAGGERATED	EMPTY	NO	NO	OBSTRUCTED LEFT INGUINAL HERNIA	ILEAL GANGRENE 55CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS	5.2
3	USMAN	56/M	NO	YES	NO	EXAGGERATED	EMPTY	NO	YES	SMALL BOWEL OBSTRUCTION	ADHESION BAND AT MIDJEJUNUM	NO	ADHESIOLYSIS	3.5
4	RAJENDRAN	63/M	NO	YES	YES	EXAGGERATED	EMPTY	NO	NO	OBSTRUCTED RIGHT INGUINAL HERNIA	ILEAL GANGRENE 35CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS	4.9
5	CHELLAPAN	40/M	NO	YES	YES	EXAGGERATED	ROOMY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	BAND AT PROXIMAL ILEUM	NO	BAND RELEASE	3.7
6	MADAPPAN	45/M	NO	YES	YES	ABSENT	EMPTY	NO	NO	OBSTRUCTED UMBILICAL HERNIA	JEJUNAL GANGRENE 50CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS, ANATOMICAL REPAIR	5
7	THESAPATTU	75/F	YES	YES	YES	ABSENT	EMPTY	NO	YES	ACUTE INTESTINAL OBSTRUCTION	GANGRENOUS DISTAL DESCENDING COLON AND SIGMOID COLON	YES	RESECTION OF GANGRENOUS SEGMENT WITH END COLOSTOMY	8.4
8	MUNUSAMY	80/M	NO	YES	YES	EXAGGERATED	ROOMY	NO	NO	OBSTRUCTED RIGHT INGUINAL HERNIA	ILEAL GANGRENE 30CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS	5.4
9	MUNUSAMY	50/M	YES	YES	YES	ABSENT	ROOMY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	GANGRENOUS CAECUM AND ASCENDING COLON	YES	RIGHT HEMICOLECTOMY WITH END ILEOSTOMY	8.1
10	BHAKIYAMMAL	54/F	NO	YES	YES	EXAGGERATED	EMPTY	NO	YES	ACUTE INTESTINAL OBSTRUCTION	ILEAL GANGRENE 40CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS	4.6
11	SAMSUN	60/M	NO	YES	YES	EXAGGERATED	EMPTY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	BAND AT ILEUM 35CM FROM IC JUNCTION WITH ILEAL GANGRENE	YES	BAND RELEASE WITH ILEAL RESECTION AND ANASTOMOSIS	5.2
12	PUSHPA	50/F	NO	YES	NO	EXAGGERATED	EMPTY	NO	YES	OBSTRUCTED UMBILICAL HERNIA	OBSTRUCTED MID JEJUNUM AS CONTENT WITH VIABLE BOWEL	NO	MESH REPAIR	3.8
13	RANGANATHAN	53/M	NO	YES	NO	EXAGGERATED	EMPTY	NO	NO	OBSTRUCTED RIGHT INGUINAL HERNIA	ILEAL GANGRENE 40CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS WITH HERNIORRHAPHY	5.6
14	DHAKSHANAMOORTHY	62/M	NO	YES	NO	EXAGGERATED	ROOMY	NO	NO	OBSTRUCTED LEFT INGUINAL HERNIA	OBSTRUCTED PROXIMAL ILEAL SEGMENT AS CONTENT WITH VIABLE BOWEL	NO	EMERGENCY HERNIOPLASTY	3.8

15	NATARAJAN	65/M	YES	YES	YES	ABSENT	EMPTY	NO	NO	OBSTRUCTED RIGHT PARAUMB LICAL HERNIA	ILEAL GANGRENE 45CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS WITH	7.4
16	KASIM	55/M	NO	YES	NO	EXAGGERATED	ROOMY	NO	NO	SUBACUTE INTESTINAL OBSTRUCTION	NIL	NO	CONSERVATIVE MANAGEMENT/PT RELIEVED OF SYMPTOMS	2.1
17	MANMAL JAIN	65/M	NO	YES	YES	EXAGGERATED	ROOMY	NO	NO	OBSTRUCTED LEFT INGUINAL HERNIA	JEJUNAL GANGRENE 55CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS WITH HERNIORRHAPHY	5.5
18	SIVAKUMAR	78/M	NO	YES	NO	EXAGGERATED	EMPTY	NO	NO	OBSTRUCTED RIGHT INGUINAL HERNIA	OBSTRUCTED MID ILEAL SEGMENT AS CONTENT WITH VIABLE BOWEL	NO	EMERGENCY HERNIOPLASTY	3.9
19	DEVI	30/F	NO	YES	YES	ABSENT	ROOMY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	ILEAL GANGRENE 35CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS	6.2
20	VATCHALA	47/F	YES	YES	YES	ABSENT	EMPTY	NO	YES	OBSTRUCTED RECURRENT INCISIONAL HERNIA	ILEAL GANGRENE 40CM FROM IC JUNCTION	YES	ILEAL RESECTION WITH DOUBLE BARREL ILEOSTOMY	8.6
21	NATHAN	85/M	NO	YES	NO	EXAGGERATED	ROOMY	NO	NO	SUBACUTE INTESTINAL OBSTRUCTION	NIL	NO	CONSERVATIVE MANAGEMENT/PT RELIEVED OF SYMPTOMS	1.8
22	MANJULA	38/F	NO	YES	YES	ABSENT	ROOMY	NO	YES	ACUTE INTESTINAL OBSTRUCTION	JEJUNAL GANGRENE 40CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS	5.3
23	SUMATHI	40/F	NO	YES	NO	EXAGGERATED	ROOMY	NO	YES	ACUTE INTESTINAL OBSTRUCTION	ADHESION BAND AT ILEUM 40CM FROM IC JUNCTION WITH ILEAL GANGRENE	YES	ADHESIOLYSIS WITH ILEAL RESECTION AND ANASTOMOSIS	5.8
24	BARNABAS	52/M	NO	YES	NO	EXAGGERATED	EMPTY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	BAND AT MID JEJUNUM	NO	BAND RELEASE	3.6
25	MUTHUKUMARAN	33/M	NO	YES	YES	EXAGGERATED	EMPTY	NO	NO	OBSTRUCTED RIGHT INGUINAL HERNIA	ILEAL GANGRENE 40CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS WITH HERNIORRHAPHY	5.6
26	MUMTAZ	42/F	NO	YES	YES	ABSENT	ROOMY	NO	YES	OBSTRUCTED INCISIONAL HERNIA	JEJUNAL GANGRENE 50CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS WITH ANATOMICAL REPAIR	5.4
27	EDWIN	45/M	NO	YES	NO	EXAGGERATED	ROOMY	NO	NO	OBSTRUCTED UMBILICAL HERNIA	OBSTRUCTED MID JEJUNUM AS CONTENT WITH VIABLE BOWEL	NO	MESH REPAIR	3.5
28	RAVI	48/M	NO	YES	YES	ABSENT	EMPTY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	JEJUNAL GANGRENE 55CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS	4.9
29	RAJA	52/M	NO	YES	YES	EXAGGERATED	EMPTY	NO	NO	OBSTRUCTED UMBILICAL HERNIA	ILEAL GANGRENE AT 40CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS WITH ANATOMICAL REPAIR	5.1

30	FASULUN BEE	65/F	NO	YES	YES	ABSENT	ROOMY	NO	YES	OBSTRUCTED UMBILICAL HERNIA	JEJUNAL GANGRENE 50CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS WITH ANATOMICAL REPAIR	5.3
31	THIRUMURUGAN	36/M	NO	YES	YES	EXAGGERATED	ROOMY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	BAND AT ILEUM WITH GANGRENE AT 35CM FROM IC JUNCTION	YES	BAND RELEASE WITH ILEAL RESECTION AND ANASTOMOSIS	4.8
32	INDIRA	45/F	NO	YES	NO	EXAGGERATED	ROOMY	NO	NO	SUBACUTE INTESTINAL OBSTRUCTION	NIL	NO	CONSERVATIVE MANAGEMENT/PT RELIEVED OF SYMPTOMS	2
33	KASTHURI	65/F	NO	YES	YES	ABSENT	EMPTY	NO	YES	OBSTRUCTED INCISIONAL HERNIA	JEJUNAL GANGRENE 40CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS WITH ANATOMICAL REPAIR	5.4
34	RAJASIMMAN	50/M	NO	YES	YES	ABSENT	ROOMY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	ILEAL GANGRENE 45CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS	5
35	KRISHNAN	72/M	YES	YES	YES	ABSENT	EMPTY	NO	NO	OBSTRUCTED RIGHT INGUINAL HERNIA	ILEAL GANGRENE 55CM FROM IC JUNCTION	YES	ILEAL RESECTION WITH DOUBLE BARREL ILEOSTOMY WITH HERNIORRHAPHY	7.8
36	JEGADEESWARI	29/F	NO	YES	NO	EXAGGERATED	EMPTY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	JEJUNAL GANGRENE 50CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS	5.6
37	GAJENDIRAN	58/M	NO	YES	YES	EXAGGERATED	EMPTY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	ILEAL GANGRENE 55CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS	4.8
38	KUPPAMMAL	54/F	NO	YES	YES	EXAGGERATED	ROOMY	NO	YES	ACUTE INTESTINAL OBSTRUCTION	ADHESION BAND AT PROXIMAL ILEUM WITH ILEAL GANGRENE 60CM FROM IC JUNCTION	YES	BAND RELEASE WITH ILEAL RESECTION AND ANASTOMOSIS	5.7
39	MURUGAN	45/M	NO	YES	YES	EXAGGERATED	ROOMY	NO	NO	OBSTRUCTED UMBILICAL HERNIA	OBSTRUCTED MID JEJUNUM AS CONTENT WITH VIABLE BOWEL	NO	MESH REPAIR	3.6
40	SELVI	36/F	NO	YES	YES	EXAGGERATED	EMPTY	NO	YES	OBSTRUCTED INCISIONAL HERNIA	OBSTRUCTED PROXIMAL ILEAL SEGMENT WITH GANGRENE AT 45CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS WITH ANATOMICAL REPAIR	5.9
41	LAKSHMI	65/F	YES	YES	YES	ABSENT	ROOMY	NO	YES	OBSTRUCTED INCISIONAL HERNIA	ILEAL GANGRENE 50CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS WITH ANATOMICAL REPAIR	8.1
42	ARUN	15/M	NO	YES	YES	ABSENT	ROOMY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	BAND AT MID JEJUNUM WITH JEJUNAL GANGRENE 50CM FROM DJ FLEXURE	YES	BAND RELEASE WITH JEJUNAL RESECTION AND ANASTOMOSIS	6.1
43	LOGAMMAL	70/F	NO	YES	NO	EXAGGERATED	EMPTY	NO	YES	SUBACUTE INTESTINAL OBSTRUCTION	NIL	NO	CONSERVATIVE MANAGEMENT/PT RELIEVED OF SYMPTOMS	1.7

44	KULLAMMA	75/F	NO	YES	YES	EXAGGERATED	EMPTY	NO	YES	ACUTE INTESTINAL OBSTRUCTION	ADHESION WITH OBSTRUCTED MID JEJUNUM AS CONTENT WITH VIABLE BOWEL	NO	ADHESIOLYSIS	3.7
45	SHANMUGAM	77/M	NO	YES	NO	EXAGGERATED	ROOMY	NO	NO	SUBACUTE INTESTINAL OBSTRUCTION	NIL	NO	CONSERVATIVE MANAGEMENT/PT RELIEVED OF SYMPTOMS	2.3
46	RATHI	32/F	NO	YES	YES	EXAGGERATED	EMPTY	NO	YES	ACUTE INTESTINAL OBSTRUCTION	ADHESION WITH JEJUNAL GANGRENE AT 45CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS	6.1
47	SAHUL HAMEED	86/M	YES	YES	YES	ABSENT	ROOMY	NO	YES	OBSTRUCTED INCISIONAL HERNIA	ILEAL GANGRENE 45CM FROM IC JUNCTION	YES	ILEAL RESECTION WITH DOUBLE BARREL ILEOSTOMY WITH ANATOMICAL REPAIR	8.4
48	CHINNARAJ	60/M	NO	YES	YES	ABSENT	EMPTY	NO	NO	OBSTRUCTED LEFT INGUINAL HERNIA	ILEAL GANGRENE 40CM FROM IC JUNCTION	YES	ILEAL RESECTION AND ANASTOMOSIS WITH HERNIORRHAPHY	5.3
49	NIROSHA	27/F	NO	YES	YES	EXAGGERATED	EMPTY	NO	NO	ACUTE INTESTINAL OBSTRUCTION	BAND AT MID JEJUNUM WITH JEJUNAL GANGRENE 55CM FROM DJ FLEXURE	YES	BAND RELEASE WITH JEJUNAL RESECTION AND ANASTOMOSIS	5.6
50	PAPPAMMAL	65/F	NO	YES	YES	EXAGGERATED	ROOMY	NO	YES	OBSTRUCTED INCISIONAL HERNIA	JEJUNAL GANGRENE 55CM FROM DJ FLEXURE	YES	JEJUNAL RESECTION AND ANASTOMOSIS WITH ANATOMICAL REPAIR	5.8