"ROLE OF COMPUTED TOMOGRAPHY IN THE EVALUATION OF INTESTINAL OBSTRUCTION"

By

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"ROLE OF COMPUTED TOMOGRAPHY IN THE EVALUATION OF INTESTINAL OBSTRUCTION"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. N. RACHEGOWDA**, Professor & Head, Department of Radiodiagnosis, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of University regulation for the award "**M. D. DEGREE IN RADIODIAGNOSIS**", the examination to be held in April 2019 by SDUAHER. This has not been submitted by me previously for the award of any degree or diploma from the university or any other university.

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LIST OF ABBREVIATIONS

AIDS - Acquired Immunodeficiency Syndrome

BO - Bowel Obstruction

CT - Computed Tomography

FAP - Familial Adenomatous Polyposis

GIST - Gastrointestinal Stromal Tumor

GIT - Gastrointestinal Tract

GVHD - Graft –Versus-Host-Disease

HPE – Histopathology

ITB – Ileal / Ileocaecal TB

LBO - Large Bowel Obstruction

MALT - Mucosa-Associated Lymphoid Tissue

MDCT - Multidetector Computed Tomography

MPR - Multiplanar Reconstruction

MRI - Magnetic Resonance Imaging

NET – Neuroendocrinal Tumors

NPV - Negative Predictive Value

PPV - Positive Predictive Value

RSJ – Recto-Sigmoid junction

SBFS - Small Bowel Feces Sign.

SBO - Small Bowel Obstruction

SI - Small Intestine

SMA - Superior Mesenteric Artery

SMV - Superior Mesenteric Vein

SOBS - String Of Beads Sign.

TB - Tuberculosis

ABSTRACT

Background: Intestinal obstruction (IO) is one of the leading causes of admission in surgical and Emergency units. CT provides information on the viability of affected bowel tissue and helps in treatment planning.

Objectives: The objectives of the study were to determine the role of CT in the confirmation or exclusion of clinically suspected mechanical intestinal obstruction and to assess the location and cause of obstruction using CT.

Material and methods: This was a prospective observational study conducted over a period of one and half years (January 2017 to June 2018) and was performed on 33 patients suspected to have intestinal obstruction referred to the Department of Radio Diagnosis at R. L. Jalappa Hospital and Research Centre. CT scan was performed using 16-slice CT scanner machine. Follow up was undertaken for all patients undergoing surgery or those who were managed conservatively. Their surgical and histopathological findings were reported.

Results: More than 60% were females (n = 20) and remaining 13 patients were males (39.4%). There were 21 patients with final diagnosis of intestinal obstruction with 22 instances of bowel obstruction, as one patient had both small and large bowel obstruction. Intestinal obstruction was suspected on erect X-ray abdomen in nine patients (42.8%). Among SBO, the commonest level of obstruction was ileum and ileocecal junction (n = 8; 80%) of which three patients had intestinal tuberculosis, two

patients had postoperative adhesions and benign strictures due to Crohn's disease each. Among LBO the commonest site of obstruction was rectum in 41.67% of patients (n = 5), all of them are proved to be primary adenocarcinomas. The overall sensitivity, specificity, PPV, NPV and accuracy of CT in diagnosing bowel obstruction was 100%, 83.33%, 64.71%, 91.67% and 94.21% respectively.

Conclusion: CT provides accurate information in determining the cause and level of bowel obstruction. CT also helps to provide information on the viability of affected bowel tissue and help in treatment planning or identify the need for surgery. We recommend CT study as part of evaluation in patients presenting with bowel obstruction.

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Introduction

INTRODUCTION

Intestinal obstruction (IO) is one of the leading causes of admission in surgical and emergency units. Early diagnosis of bowel obstruction is critical in preventing complications, particularly perforation and ischemia. Previous studies have demonstrated computed tomography (CT) to be a valuable technique for imaging in intestinal obstruction[1].

The morbidity and mortality associated with acute small-bowel obstruction is significant accounting for 12–16% of all surgical admissions. Postoperative adhesions being the most common cause accounts for 70% cases. Other common causes include hernias, neoplasms, and Crohn’s disease. Mechanical large bowel obstruction is four to five times less common than small bowel obstruction[2].

Initial investigations such as plain radiographs have been shown to have a low sensitivity and specificity and therefore have a limited role in evaluation of bowel obstruction. Furthermore they are also limited in their ability to accurately discern the site and cause of obstruction. Other investigations such as enteroclysis may be contraindicated in patients with acute and complete or high-grade bowel obstruction and those with strangulation or suspected perforation. Its use is therefore limited in patients with markedly diminished intestinal peristalsis[3].

Given the relative lack of sensitivity and specificity of plain film findings in patients with symptoms of bowel obstruction, in acute settings, CT plays a central role in evaluation.

This study was carried out to evaluate the role of MDCT (multi detector computed tomography) in detecting etiology, diagnosis and management of intestinal obstruction. The MDCT diagnosis was confirmed by laparotomy findings/histopathology/clinical outcome.

Objectives

AIMS AND OBJECTIVES

The objectives of the study are as follows:

1. To determine the role of CT in the confirmation or exclusion of clinically suspected mechanical intestinal obstruction.
2. To assess the location and cause of obstruction using CT.

Review of Literature

REVIEW OF LITERATURE

Anatomy and function

The small bowel consists of duodenum, jejunum, and ileum with a length of about 6 to 8 m with a diameter of <2.5 cm, which can distend upto 4 cm[4]. It extends from pylorus superiorly to ileocecal valve inferiorly. The duodenum measures approximately 25 cm and is considered as the most predictable portion of the small intestine. Duodenum is primarily a retroperitoneal organ and only one-tenth (2.5 cm) is peritoneal. It is divided into four parts, which form a 'C'-shaped loop at the level of L1 to L3 vertebrae. The second and third parts of duodenum are about 8 to 10 cm long followed by first part (~5 cm). The fourth part of duodenum is the shortest segment and measures about 2.5 cm[4,5]. The duodenum terminates at the level of duodenojejunal junction. The rest of small bowel consisting of jejunum and ileum is more commonly referred to as 'small bowel'. The proximal 40% is comprised of jejunum and the distal 60% is comprised of ileum. The distinction between jejunum and ileum is indistinct; however one finds that intestinal morphology gradually changes as we traverse from proximal to distal portions. The term 'terminal ileum' refers to the distal 30 cm of ileum[5]. The terminal ileum is notorious for various infectious and inflammatory conditions such as intestinal tuberculosis and Crohn's disease[6].

Jejunum normally occupies the central and left part of abdominal cavity and ileum the lower and right part. These organs are peritoneal and attached to abdominal wall through mesentery. The mesenteric root is about 10 to 15 cm long and attached from right lower quadrant to left upper quadrant. Attachment to mesentery serves to facilitate bowel motility and ensure adequate blood flow to the intestines[4,5]. Abnormality of bowel mesentery such as malrotation or adhesions therefore increase risk of small bowel volvulus[4].

The large intestine begins from the ileocecal valve and ends at anus. It tends to form a border around small bowel loops and is usually present on the periphery. The large bowel is on an average 1.5 m in length, although there is considerable variation in among different individuals. It begins from cecum, which continues as ascending colon, hepatic flexure, transverse colon, splenic flexure, descending colon, sigmoid colon, rectum and anal canal. Large bowel enters the pelvis at the level of sigmoid colon till pelvic floor, where it ends as anus. The large bowel is fairly fixed in position and parts of ascending and descending colon are retroperitoneal in position. Unlike small bowel, large bowel has haustrations, which are incomplete infoldings of the luminal wall[7].

The vascular supply of the bowel loops depends on the development. The foregut (duodenum and stomach) receives blood supply from coeliac axis and its branches, the midgut (remaining of small bowel till proximal 2/3rd of transverse colon) from superior mesenteric artery (SMA) and hindgut (distal 1/3rd of transverse colon till anal canal) from inferior mesenteric artery (IMA). Therefore the primary blood supply for duodenum is from "superior and inferior pancreaticoduodenal arteries with contributions from several sources including right gastric, supraduodenal, right gastroepiploic, hepatic and gastroduodenal arteries"[4,5]. The fourth part of duodenum is also supplied by "terminal branch of anterior pancreaticoduodenal artery (branch of SMA)"[5]. The remaining small bowel is supplied by SMA and drained via superior mesenteric vein

(SMV)[4,5]. The duodenal veins and SMV drain into the portal vein forming the enterohepatic circulation[5]. The large bowel is drained by SMV (midgut part) and inferior mesenteric vein (IMV) (hindgut part), both of which ultimately drain into the portal vein. Part of venous drainage also occurs through middle rectal veins, which drain into internal iliac and pudendal veins[7].

Functional anatomy of GIT

The small bowel has an amazingly large absorptive surface area of about 4,500 m^2. This large absorptive area helps in optimal and effective absorption of nutrients and other materials from food. The reason behind such a large absorptive area is the presence of unique mucosal structure arranged in concentric folds, which appear as transverse ridges. These are also referred to as plicae circulares and are about 2 inches long and 3 mm thick[8]. These folds are not lost during physiological distension of bowel unlike the rugae in stomach, which are lost when stomach distends[5]. These folds can cover half to two-thirds of luminal circumference or may have more than one turn. They can be either horizontal or oblique in presentation[5,8]. These circular folds are seen maximum distal to major duodenal papilla and in proximal half of jejunum and reduce in size and frequency as one traverses bowel distally to ileum. These folds are almost completely absent in distal ileum and therefore this segment of bowel is thin. These folds increase absorptive area and also reduce the time taken for passage of contents[5]. It is believed that small intestine contains nearly 800 plicae circulares, which increase the mucosal surface area by 5 to 8 times more compared with outer surface area[8].

The entire mucosa of small bowel contains small projections referred to as villi, which further increase the surface area of intestinal lumen and also give a velvety texture of mucosa. Similar to mucosal folds, the villi are numerous and tall in duodenum and jejunum and reduce as one traverses distally and are short and fewer in number in ileum[5].

The variable number of plicae circulares and villi result in different rate of absorption in intestines. There are various specialized cells and receptors in particular sites, which explain the variable absorption at different sites. For example, iron and calcium are selectively absorbed in duodenum and proximal jejunum[8]. The presence of specialized cells and receptors in small intestine also ensure that maximum nutrient transport. Meanwhile, large colon is primarily responsible for water and electrolyte exchange. It is estimated that GIT handles nearly 8 to 10 L of fluid of which only 1.5 L is left for colonic absorption, while the rest of water and electrolyte absorption is handled by small bowel[9]. The bulk of pancreatic, biliary, intestinal and salivary secretions are also absorbed by the small bowel. Patients with small bowel obstruction often present with nausea and vomiting. There may also be persistence of symptoms despite reduced oral intake. Furthermore reduced transit due to SBO may lead to bacterial overgrowth with resultant feculent vomiting. SBO may also cause mucosal damage due to altered perfusion dynamics early in the course of disease[4].

BOWEL OBSTRUCTION

Patients with SBO may present with vague complaints such as crampy abdominal pain, vomiting, abdominal distension and absent or high-pitched bowel sounds. It is important to determine the cause for SBO so as to decide if patients can be treated conservatively or require surgery. Surgery is not recommended unless patients have a significant lesion causing high-grade intestinal obstruction, strangulation, vascular compromise or suspected to have intestinal perforation. Therefore it is important to determine the cause and location of bowel obstruction and to evaluate for presence or absence of complications[10].

Table 1. Types of Intestinal Obstruction and Description[10]

Type of Obstruction	**Description**
Complete or high-grade obstruction	No passage of fluid or gas beyond site of obstruction
Incomplete or partial obstruction	Some fluid or gas may pass beyond the obstruction
Strangulated obstruction	Compromised blood flow, may lead to intestinal ischemia, necrosis, and perforation.
Closed-loop obstruction	Obstruction of segment of bowel at two points along the course Isolated loop can show progressive accumulation of fluid in gas Risk of volvulus and subsequent ischemia

Table 2. Types and Causes of Small Bowel Obstruction[4,10,11]

Type of lesion	**Cause**
Extrinsic lesions	Adhesions
	Hernias
	Tumors
	Hematoma
	Endometriosis
Intrinsic lesions	Tumors
	Inflammatory lesions
	Vascular lesions
	Hematoma
Intussusception	Tumors
Intraluminal lesions	Gallstones
	Bezoars

The various causes for small bowel and large bowel obstruction have been enumerated in Table 2 and Table 3 respectively.

Table 3. Types and Causes of Large Bowel Obstruction[4,10,11].

Type of lesion	**Cause**
Intrinsic lesions	Tumors
	Inflammatory lesions
	Vascular lesions
Extrinsic lesions	Volvulus
	Hernias
	Adhesions
Intraluminal lesions	Stones
	Bezoars

Causes of SBO

Small bowel obstruction can be classified into complete or high-grade obstruction, incomplete or partial obstruction, strangulated obstruction and closed-loop obstruction, which are described in Table 1[10,11].

Adhesions

Adhesions are the leading cause of SBO in nearly 80% of patients with SBO[4]. It has been estimated that nearly 85% of adhesions are postoperative followed by peritonitis (10%), while the remaining 5% may be due to congenital causes or unknown etiology[12]. There is a positive correlation between history of abdominal surgery and adhesions. Studies have shown the incidence of adhesions to range from 67% to 93% in patients who have history of previous abdominal surgery compared to 10% to 28% in patients who did not have a history of abdominal surgery, suggestive of strong correlation[13]. Although postoperative adhesions are commonest cause for SBO, it was observed that only up to 4.6% may develop SBO. However, some surgeries have increased risk for adhesion-related SBO, which include open adnexal surgery (23.9%), ileal pouch-anal anastomosis (19.3%), followed by open total abdominal hysterectomy (15.6%) and lastly open colectomy (9.5%). It is possible that patients undergoing these surgeries are more at risk for adhesion-related SBO[14]. The risk of adhesions has reduced with introduction of laparoscopic surgery and this is one of the reasons why rate of adhesiolysis has remained stable despite increasing number of surgeries in the West[4].

Adhesions are not visible on CT studies and therefore the diagnosis is one of exclusion. Clinical history (previous history of surgery) is important. On CT a bowel loop showing kinking or tethering at the transition without identifiable cause is suggestive of adhesion[4].

Hernias

Hernias have been attributed as the second commonest cause for SBO accounting for up to 10% of SBO. Hernias are also considered as leading cause of SBO in some regions. Hernias can be external or internal. External hernias occur primarily through a defect through abdominal or pelvic wall usually at the site of congenital weakness or iatrogenic (post-surgery)[4,15]. External hernias include inguinal, umbilical, or incisional hernias, which can be readily identified on CT as herniation of bowel loops in the subcutaneous tissues or through abdominal wall[4]. Internal hernias can be defined as protrusion of bowel loops through peritoneum or mesentery into a different compartment in abdominal cavity. These hernias usually occur through pre-existing anatomic openings such as foramina, recesses or fossae. These hernias can also be caused as a result of defect due to congenital or acquired causes. Some of common acquired causes include surgery, trauma, or inflammation. Internal hernias are rare with a reported prevalence of 0.2 to 0.9% and have been reported in about 0.5% to 4.1% of intestinal obstruction. The commonest internal hernia is paraduodenal hernia seen in more than half of cases (53%). Other types of internal hernias include transmesenteric hernia, transomental hernia, pericaecal hernia, sigmoid mesocolon hernia, supravesical and pelvic hernias, hernia through broad ligament or through perirectal fossa[16]. On imaging paraduodenal hernia can be appreciated by the presence of sac-like configuration, which is situated directly to right of SMA or posterior to stomach. An important cause for internal hernia is Roux-en-Y gastric bypass surgery[4]. The potential sites of internal hernia following Roux-en-Y gastric bypass surgery are enteroenterostomy mesenteric defect (transmesoenteric), defect in transverse mesocolon (mesocolic window) and space between transverse mesocolon and mesentery of Roux limb (Petersen hernia)[17].

Challenge with internal hernias is that they can rarely be diagnosed clinically due to lack of specific clinical symptoms. An intermittent internal further poses a challenge in imaging diagnosis as the bowel loops may be in normal anatomical position during imaging study[16]. Although CT has good sensitivity, specificity and accuracy of 63%, 76% and 77% for diagnosis of transmesenteric hernias, diagnosis of internal hernias remain challenging task radiologically[18].

Hernias usually cause closed-loop SBO and therefore symptomatic hernias have high risk of developing ischemia (approximately 28%). It is therefore imperative that patients with symptomatic hernias undergo surgery. Asymptomatic or minimally symptomatic hernias may be treated conservatively initially or can be planned for elective surgical repair[4].

Tumors

The small intestine accounts for nearly three-fourths of length of GIT and >90% of the mucosal surface. Small intestine is however, a less common site for neoplasms of GIT constituting for only 2 to 6% of all GI tumours. The reason may be due to its physiological characteristics such as rapid transit of contents, alkaline environment, secretion of IgA and lymphoid tissue. Patients with small bowel neoplasms may present with non-specific symptoms such as GI bleeding, abdominal pain (60%), nausea and vomiting (50%), anaemia (50%), loss of weight (40%), diarrhoea and intestinal obstruction (30% each). A majority of patients are however asymptomatic until late stages. Small bowel neoplasms are notorious for late diagnosis and therefore do not have favourable prognosis with 5-year survival rates ranging from 0-28% for adenocarcinomas, 14-30% for lymphomas and 60% for carcinoid tumours[19].

Benign tumours

These neoplasms constitute about 1 to 2% of all GI tumours. The commonest tumours are leiomyomas and adenomas. Other benign tumours uncommonly seen in the GIT are lipomas and hamartomas. These lesions are usually asymptomatic and nearly half the patients may clinically asymptomatic[20].

Leiomyoma

These are the commonest benign tumours of small bowel with incidence ranging from 22 to 43%. These are usually solitary lesions, which originate from muscular layer of small intestine. Leiomyomas can be located in submucosal, intramural or subserosal region and can be appreciated on CT as homogeneously enhancing well-defined rounded mass and range in size from one to 10 cm. A submucosal leiomyoma may show small, focal mucosal ulcer. These tumours can be diagnosed based on their typical appearance, lack of mesenteric changes and no known malignancy elsewhere[20].

Adenomas

These neoplasms constitute about 14 to 20% of small bowel tumours. Adenomas are proliferative epithelial tumours and can be further classified similar to that of colonic adenomas. These tumours can be seen as tubular adenomatous polyps. These tumours are seen on CT as small-sized intraluminal masses (usually <2 cm). Adenomas may be solitary or multiple. Multiple adenomas are known to affect single intestinal segment; however, in case of familial polyposis, the adenomas tend to be distributed throughout GIT. Villous adenomas are broad-based tumours and are larger in size compared with tubular adenomatous polyps with an average size of about 3 cm. Adenomas are asymptomatic. Pedunculated polyps may cause intussusception and resultant SBO. Adenomas can be seen on CT as well-demarcated moderately enhancing soft tissue mass with maintained fat planes surrounding the lesion. Differential diagnoses include inflammatory fibroid polyps[20].

Lipoma

Lipomas account for about 8 to 20% of benign neoplasms of small bowel and are third common cause of benign small bowel tumours. Lipomas can be seen either in duodenum or distal part of small bowel. The most common presentation is intussusception. Lipomas are typically solitary lesions showing predominant fat density and may grow to large sizes. Lipomas can undergo cystic degeneration, necrosis and calcifications. On imaging, lipomas appear as well-defined ovoid lesions showing features of fat[20].

Hemangioma

These tumours are commonly seen in jejunum and the commonest presentation is intestinal bleeding. These tumours are challenging to detect on CT, unless they are sharply demarcated large enhancing mass in small bowel wall. The presence of feeding vessel if observed helps to clinch the diagnosis[20].

Polyposis syndromes

It is important to mention about polyposis syndromes. These include Peutz-Jeghers syndrome, juvenile polyposis, Cronkhite-Canada syndrome, Cowden's disease, Gardner's syndrome and familial adenomatous polyposis (FAP). These lesions are interspersed throughout the GIT. Some of the conditions are notorious for recurrence especially Gardner's syndrome and FAP, which may occur in small intestine once colon is resected[20].

Malignant tumours

Majority of small bowel tumours are malignant (up to 70%) (Table 4). Risk factors include history of inflammatory bowel disease (IBD), FAP, acquired immunodeficiency syndrome (AIDS) and neurofibromatosis. Small bowel malignancies have bad prognosis as more than half of these tumours are diagnosed late and present with advanced, where surgical resection is not an option.

Adenocarcinoma

Adenocarcinomas are the commonest malignancy of small bowel; however, it form <1% of all GI malignancy. The commonest site of adenocarcinoma is duodenum followed by proximal jejunum (proximal 25 to 30 cm beyond ligament of Treitz). Adenocarcioma due to Crohn’s disease is mostly seen in the terminal ileum. These tumours have varied appearance on CT studies. They are seen as heterogeneously enhancing focal wall thickening involving short segment of bowel and may cause luminal stenosis and SBO. These tumours can infiltrate beyond the bowel into omentum causing surrounding desmoplastic reaction. CT is helpful in evaluating extent of tumour spread, involvement of surrounding viscera, assess metastatic lymphadenopathy and to stage local spread and distant metastasis. It also helps to look for peritoneal carcinomatosis. The key imaging characteristics for diagnosis of adenocarcinoma are focal short segment lesion, proximal location of tumour, high-grade SBO, and solitary lesion[20].

Neuroendocrine tumours (NET) or carcinoids

NET is second most common malignancy (up to 25% of small bowel malignancy) followed by adenocarcinoma and originates from the enterochromaffine cells, which are present at the base of Liberkuhn crypts in bowel wall. These tumours are located in distal small bowel or proximal colon, typically involving appendix (50%) followed by ileum (33%). These are slow-growing neoplasms. On imaging they may be diagnosed as mural contrast-enhancing lesion, which extends to surrounding mesentery causing typical desmoplastic reaction with resultant spiculated margins. Calcifications are often seen (~70%). The desmoplastic reaction is classically described as “surrounding and encasing mesenteric vessels, and, in the worst case, leading to ischaemia of the affected small bowel loop.” These tumours are usually <2 cm and therefore are usually diagnosed late in the disease with patients often presenting with liver metastasis. Since these are hormonally active tumours, patients may present with symptoms such as cutaneous flushing, diarrhoea, intermittent hypertension and palpitations. Desmoplastic reaction usually causes SBO. Advanced cancers also demonstrate mesenteric malignant lymphadenopathy and peritoneal seeding. Hepatic metastasis are usually hypervascular and can be easily identified on triple-phase CT[20].

Lymphoma

Lymphoma is the third commonest small bowel malignancy (15 to 20% of small bowel malignancy) and may occur as primary bowel lymphoma or as part of systemic disease. The primary bowel lymphoma originates from the mucosa-associated lymphoid tissue (MALT). Lymphomas have also been associated as a complication of coeliac disease. Lymphoma is commonly seen in ileum and has several appearances on imaging. It may present as multifocal enhancing mucosal nodules affecting small segment of small bowel or may present as an enhancing single mass of variable size involving focal bowel segment with wall thickening and mucosal fold destruction with/without infiltration to surrounding mesentery. As these are soft tumours, they seldom cause small bowel obstruction; but are known to sometimes cause intussusception and SBO. Clinically, these patients present with non-specific symptoms and may have history of weight loss, fever, abdominal pain and diarrhoea. The key imaging findings of long segmental involvement typically in distal bowel without SBO are key findings, which help differentiate lymphoma from other malignancies[20].

Gastrointestinal stromal tumour (GIST)

GIST is commonly seen in the stomach and less likely in the small bowel, with jejunum being the commonest site. Although some authors classify GIST as benign tumour with malignant potential, most of the authors consider GIST as malignant neoplasm. GIST has been classified pathologically as “benign, borderline, low or highly malignant tumour”. On imaging it is difficult to differentiate benign from malignant lesion unless there are other signs of malignancy such as malignant lymphadenopathy. Therefore as a rule all GIST tumours are surgically resected. On imaging GIST can be as moderately enhancing exophytic mass with propensity for central necrosis or calcification. These tumours can displace adjacent bowel loops and may cause confusion about site of origin. Malignant GIST are known to cause hypovascular liver metastasis and peritoneal seeding[20].

Leiomyosarcoma

Leiomyosarcoma as the name suggests originates from the smooth muscle layer of bowel wall is an uncommon cause for small bowel malignancy (<15%). Leiomyosarcoma as a rule is seen as a single mass typically located in jejunum and ileum with Meckel’s diverticulum being the commonest site. These are slow growing extraluminal tumours and therefore have late presentation. On imaging these tumours appear as large rim enhancing heterogeneous density mass lesion showing central necrosis and also cystic changes. Associated findings include ulceration, cavitation or fistula formation. Hepatic metastases also show findings similar to primary lesion such as rim enhancement with central cystic/necrotic changes[20].

Metastases

These could be caused due to intraperitoneal seeding, haematogeneous spread or by direct extension from other tumours. Ovarian and colonic malignancies are the commonest cause for small bowel metastases. Causes of local infiltration may include pancreatic, biliary or colonic malignancies. Hematologic spread is from bronchial carcinoma, breast cancer, renal cell carcinoma or melanoma. On imaging metastases can appear as small enhancing nodules typically along serosal surface. There are no key imaging findings for diagnosis of metastasis. Presence of primary tumour with short segmental enhancing wall thickening or nodules/masses should raise suspicion for small bowel metastasis[20].

Table 4. Location and Typical Imaging Presentation of Common Small Bowel Malignancies

Malignancy	**Location**	**Typical signs**
Adenocarcinoma	Duodenum, proximal jejunum	Concentric lumen narrowing with irregular edges Complete bowel obstruction
Carcinoid	Ileum (appendix)	Heterogenous enhancement Small (<2 cm) single or multiple filling defects Desmoplastic reaction of the mesentery Hypervascular
Lymphoma	Ileum	Coarse segmental wall thickening with ulceration and necrosis Bulky lymphyadenopathies Aneurysmal dilatation of bowel loops
Malignant GIST	Jejunum (stomach)	Large regular mass with inhomogeneous enhancement Necrosis and/or ulceration Ileal localisation
Leiomyosarcoma	Jejunum, ileum, Meckel's diverticulum	Solitary large mass Cavitation, ulceration, fistula
Metastases		Intraluminal nodules develeloping from haematogenous spread to submucosal layers

Inflammatory disease – Crohn’s Disease

There has been an increasing incidence of Crohn’s disease as cause for SBO. In this condition SBO typically occurs due to acute inflammatory narrowing of bowel lumen or as a result of fibrostenosing disease, which causes narrowing and stricture. During the acute phase of Crohn’s disease there is presence of mural thickening and stratification, referred to as “double halo” appearance, mucosal thickening/ hyperemia, mesenteric inflammatory fat stranding or mesenteric vessel engorgement all of which can produce a typical appearance[4,21]. The fibrostenosing disease usually demonstrates homogeneous postcontrast bowel wall enhancement without mesenteric inflammation or mesenteric vessel engorgement[4].

Tuberculosis

The commonest site of intestinal tuberculosis is the ilcocecal region. Intestinal Koch’s is one of common cause for SBO. On imaging initial mild inflammation stage of disease may be seen as slight, symmetric wall thickening with small regional lymph nodes. During advanced stage of disease, imaging shows thickened bowel wall with heterogeneously appearing inflammatory mass. Multiple large lymph nodes may be present with hypoattenuating centers, consistent with caseation necrosis[21].

Appendicitis and Diverticulitis

Appendicitis is an uncommon cause for SBO. CT can accurately diagnose appendicitis and also show complications such as appendicular abscesses, peritonitis or phlegmon[21].

CT is the investigation of choice for evaluation of diverticulitis. On imaging uncomplicated diverticula may be seen as colonic diverticula, wall thickening of involved colon, mesenteric root edema and engorged mesenteric vessels. In complicated diverticulitis, imaging may show paracolonic or pelvic abscess, colovesical obstruction and peritonitis. Paracolonic inflammation may sometimes cause SBO. It is believed that close proximity of small bowel loops to areas such as sigmoid colon or periappendical region may cause secondary inflammatory changes which affect small bowel causing SBO. The small bowel loops may get trapped along the inflammatory changes in large colon resulting in SBO[21].

Radiation Enteropathy

Radiation enteropathy has been known to cause SBO in some patients, which is usually mild-to-moderate. In these cases, the underlying pathology is related to adhesions and to some extent radiation serositis related luminal narrowing and dysmotility. On imaging the changes are usually seen in the irradiated regions, usually involving large bowel in pelvis. Imaging, especially CT helps to evaluate extent of small bowel damage. In patients with severe radiation-induced enteropathy, imaging helps to evaluate full extent of bowel wall thickening and mesenteric fibrosis[21].

Intussusception

Intussusception has classically been defined as “the telescoping of a proximal segment of the GIT, called intussusceptum, into the lumen of the adjacent distal segment of the GIT, called intussuscipiens". Intussusception is one of uncommon causes for SBO. Intussusception in adults is rare and accounts for 5% all cases of intussusception. As a rule intussusception in adults is caused due to some bowel pathology (approximately 90% of cases show underlying pathology) and therefore has to be treated. The cause for intussusception in adults is a pathology, which acts as a lead point, such as neoplasms, lipomas, polyps, diverticuli, strictures, etc. As nearly two-thirds of cases of intussusception in adults is due to underlying malignancy, a definitive surgery is indicated, especially surgical resection. In small bowel about 30% of cases of intussusception is caused due to underlying malignancy, which more than doubles in large bowel (~66%) In contrast in children, intussusception in child is primary and benign and in about four of every five children, it can easily treated with pneumatic or hydrostatic reduction[22].

The commonest locations for intussusception is at the junction of free moving bowel segments and retroperitoneal or adhesionally fixed segments. Intussusception can be classified into four categories based on the site as: 1. enteroenteric, which is confined to small bowel, 2. colocolic, which is confined to large bowel only, 3. ileocolic, which is defined as "prolapse of terminal ileum within the ascending colon" and 4. ileocecal, which is causes when the ileocecal valve acts as a leading point. It may sometimes be difficult to distinguish between ileocolic and ileocecal intussusception. Other classification of intussusception is based on the etiology (idiopathic, benign or malignant lead point)[22].

The initial imaging is by radiographs and barium studies, which show “stacked coin” or “spring coil” appearance in upper GI barium studies and “cup-shaped filling defect” or “spiral” or “coil spring” appearance in barium enema. Radiographs have very low diagnostic yield in diagnosis of intussusception (ranging from 40 to 50%). Ultrasonography is often employed in evaluation of abdominal pain and is helpful in diagnosis of intussusception in both adults and children. The classical ultrasonography features are presence of “target” or “doughnut” sing on transverse view and “pseudo kidney” or “hay-fork” sing on longitudinal view. Ultrasonography however has its limits and has limited utility in obese patients and in those with excessive bowel gas. Currently, CT is considered the most sensitive investigation for evaluation of intussusception and has a diagnostic accuracy ranging from 58% to 100%. Imaging findings on CT shows telescoping of one segment of bowel loop into another giving an appearance of “target” or “sausage-shaped” mass with layering effect. Additionally, mesenteric vessels and omental fat are also seen within the bowel lumen. Other findings include presence of bowel wall edema (loss of 3-layer appearance resulting from impaired mesenteric circulation). CT has the ability to define and diagnose the lead point in secondary intussusception and also diagnose primary or idiopathic intussusception with confidence[22].

Vascular causes

Occlusion or stenosis of mesenteric vessels (SMA or SMV) may cause bowel ischemia. Other potential causes for bowel ischemia include vasculitic disorders secondary to endarteritis and reduced vascular supply to affected bowel segments. Imaging may show presence of thrombus or occlusion of mesenteric vessels. The ischemic segment of bowel may show non-circumferential or asymmetrical wall enhancement, or delayed enhancement or no enhancement. Bowel wall thickening can be consistently observed in bowel ischemia. Advanced stage of disease may show bowel infarct, which can be seen as gas in bowel wall (pneumotosis intestinalis) or may show gas in portal venous system[23].

Bezoars

Small bowel bezoars were once considered as unusual and uncommon cause of SBO. There has been an increasing incidence in SBO due to bezoars with increase in gastric surgeries for peptic ulcer disease. These patients present with complete mechanical obstruction and is commonly seen in jejunum or proximal ileum. CT is helpful in identifying bezoar in small bowel. Imaging findings include presence of intraluminal mass at the transition zone causing obstruction. The bezoar itself may appear as a mass lesion with mottled appearance with air foci within, akin to the "small bowel feces" sign. A pertinent clinical history helps to clinch the diagnosis[21].

Other Intraluminal Causes

Other uncommon causes for SBO may include presence of gall stone (gall stone ileus), foreign bodies, or intestinal worms (ascariasis) all of which may cause mechanical obstruction of small bowel resulting in obstruction[21].

Intestinal Malrotation

Intestinal malrotation can be defined as "anomaly of rotation and fixation of the midgut". Although intestinal malrotation is an isolated anomaly, it may sometimes be associated with other conditions such as congenital heart disease (CHD) or situs inversus. In adults intestinal malrotation is generally an incidental finding. On CT imaging hallmark findings of intestinal malrotation are of right-sided small bowel and left-sided colon along with abnormal relationship of SMA and SMV. Other finding includes aplasia of uncinate process. It may rarely cause SBO[21].

Small bowel volvulus

Small bowel volvulus can be caused secondary to conditions such as intestinal malrotation, congenital bands, internal hernia or postoperative adhesions. On imaging volvulus may show 'U'-shaped configuration or radial distribution of bowel loops, which are usually fluid filled and can be seen converging to point of torsion, where the mesentery gets twisted around the site of torsion. The mesenteric vessels give a distinctive "whirl" sign, caused due to twisting of vessels[21].

Large bowel obstruction

Large bowel obstruction (LBO) can present as an emergency and therefore it is important to have early and definitive diagnosis for prompt treatment. Nearly 80 to 85% of cases of LBO are secondary to colorectal cancer, volvulus and diverticulitis[24,25]. Unlike the small bowel, neoplastic etiologies constitute majority of cases of LBO. The non-neoplastic etiologies constituting for LBO include inflammatory bowel disease, colonic volvulus, diverticulitis and endometriosis. It is also important to look for mimickers of LBO such as paralytic ileus and toxic megacolon[24]. The neoplastic etiologies for LBO include colorectal carcinoma. Other causes for LBO are obstructive colitis and adult intestinal intussusception[25].

Non-neoplastic causes for LBO

Inflammatory bowel disease

The commonly afflicting inflammatory bowel disease in large bowel is ulcerative colitis (UC). Other inflammatory conditions such as Crohn's disease and intestinal Koch's can also affect large bowel. Although UC is common than Crohn's disease in large bowel, both these conditions rarely cause LBO. Crohn's disease is known to cause strictures due to prolonged disease, which affects morbidity; however strictures are more common in small bowel rather than large bowel[24].

Tuberculosis has been known to cause LBO. The commonest site for large bowel Koch's infection is cecum followed by ascending and transverse colon. Barium studies show short, rigid and distorted cecum, loss of normal mucosal pattern of terminal ileum and deformation of ileocecal valve. Ascending colon may show skip lesions. Sometimes concentric narrowing with fibrosing strictures may mimic malignancy. The presence or lack thereof shouldering with overhanging edge usually helps differentiate malignancy from non-malignant etiology. On CT studies, it may be difficult to differentiate between colonic Koch's and malignancy[24].

Diverticulitis

Diverticulitis is responsible for about 10% of LBO. Recurrent diverticulitis may cause abscess formation, edema and furthermore may cause strictures, which can all potentially lead to high-grade colonic obstruction. Although barium enema study can show concentric stenosis at the site of obstruction, it is not possible to differentiate it from malignancy. Features that help in diagnosis of diverticulitis include absence of diverticula near the site of obstruction, mucosal ulceration, irregularity of wall contour, overhanging margins and irregular edge. CT imaging may show recurrent inflammation and segmental involvement of large bowel (>10 cm), whereas colorectal cancer may show presence of pericolic lymphadenopathy and luminal mass[24].

Endometriosis

There is variable incidence of endometriosis of bowel ranging from 3% to 34%. The commonest site for endometriosis is the rectosigmoid region followed by small bowel, caecum and appendix. Patients with endometriosis are usually asymptomatic or may present with vague GI symptoms. Endometriosis causing bowel obstruction is very rare and when present may present with features of colonic obstruction and bleeding in the absence of obvious pelvic mass lesion. On barium enema, endometriosis may appear as broad-based mass or intramural defect. Sometimes secondary fibrosis may cause pleating of surrounding mucosa. A concentric endometriosis can mimic carcinoma or neoplastic etiology. MRI is considered as investigation of choice for evaluation of intestinal entometriosis[24].

Colonic volvulus

Colonic volvulus accounts for about 10 to 15% of all LBO and is the second commonest cause for non-neoplastic LBO. Colonic volvulus occurs at the junction of mobile colon and relatively fixed colonic loop along which the twisting can occur. The commonest sites for colonic volvulus are sigmoid colon (70%) followed by cecum (25%) and lastly transverse colon (5%)[24].

Sigmoid volvulus

Sigmoid volvulus is the most common of colonic volvulus. On radiographs, it may show “northern exposure” sign, which refers to large air-filled bowel loop representing sigmoid colon arising from pelvis and extending beyond transverse colon. Perhaps the most famous sign is the “coffee bean” sign, which refers to the coffee-bean appearance of dilated sigmoid colon. CT is particularly useful in diagnosis of sigmoid volvulus and may show “whorl sign”, referring to the twisting of mesentery and mesenteric vessels. The newer signs described on CT are “X-marks-the spot” sign and “split-wall sign”. “X-marks-the spot” refers to the two crossing sigmoid transition points, which are seen arising from single location. The “split-wall” sign refers to the apparent separation of sigmoid wall by the surrounding mesenteric fat caused due to incomplete folding/ twisting of sigmoid colon. CT also helps to assess the viability of the twisted bowel loop and guide further management[24].

Cecal volvulus

Cecal volvulus is the second most common cause of colonic volvulus. On radiography it is seen as dilated, gas-filled bowel loop located in the left upper quadrant or mid abdomen. There are two types of cecal volvulus, axial and loop-type. In the axial volvulus there is twisting of cecum in axial plane (clockwise or anticlockwise) along its long axis. The cecum therefore appears in the right lower quadrant. In the loop-type volvulus, there is twisting and inversion of cecum, which relocated cecum to right upper quadrant. On barium enema study, a beak sign may be appreciated at the level of twisting of volvulus. CT imaging helps to depict the type of cecal volvulus, helps to identify the site of twisting and also demonstrate mesenteric and mesenteric vessel twisting. CT further helps to assess the viability of affected colon. Unlike sigmoid volvulus, diagnosis of cecal volvulus is usually delayed resulting in significant damage and surgical resection is often required in these patients[24].

Disorders simulating LBO

Conditions such as toxic megacolon, chronic meteorism (in individuals with progressive systemic sclerosis), Ogilvie's syndrome and paralytic ileus can simulate LBO. In fact these disorders may cause adynamic ileus. Laparotomy remains the commonest cause for paralytic ileus followed by peritonitis secondary to conditions such as cholecystitis and pancreatitis, bowel ischemia, spinal injury etc[24].

Vascular causes

Large bowel ischemia is typically seen in elderly individuals and is usually secondary to occlusion or stenosis of SMA or SMV or IMA or IMV. Large bowel ischemia typically affects the watershed regions such as splenic flexure. CT imaging helps to evaluate the arterial or venous abnormality in these patients. The bowel wall may show wall thickening, submucosal edema and adjacent mesenteric reaction. The bowel wall may show hyperenhancing or hypoenhancing bowel walls. Post ischemic strictures may cause occlusion resulting in LBO[26].

Colorectal carcinoma

Colorectal tumours account for about 60% of all cases of LBO of which adenocarcinoma is the commonest. Malignant lesions cause luminal compromise and bowel wall thickening causing LBO. On radiographs a left-sided LBO may show dilated colonic loops, where as a right-sided LBO may show gasless colon and gasless small bowel loops, sometimes mimicking SBO. Barium enema may help in identifying the location of lesion but sometimes may be difficult especially in the proximally situated tumours. MDCT helps to assess the tumours, which appears as heterogeneously enhancing lesion. MDCT can also localize the site and level of obstruction, tumour extension to adjacent organs, malignant lymphadenopathy, and peritoneal carcinomatosis[25].

Obstructive colitis

Ischemic colitis (ulceroinflammatory disorder) occurring proximal to complete or partial occlusion of large bowel caused by colonic malignancy is referred to as obstructive colitis. The proximal ulcerated segment and the inflamed distal segment is usually separated by normal intervening colonic segment. The commonest cause for obstructive colitis is adenocarcinoma of colon. On barium study, obstructive colitis show thumb-printing sign, which is caused due to submucosal thickening. On CT, additional findings include presence of pericolonic vascular engorgement and surrounding fascial thickening. These findings can be confused for tumour infiltration resulting in overstaging of tumour. Obstructive colitis may be seen as a single discrete ulcer or as an extensive area showing fulminant colitis. On imaging obstructive colitis can be easily mistaken for ischemic colitis as enhancing lesion distal to ischemic colon is often overlooked[25].

Adult intestinal intussusception

Adult intestinal intussusception accounts for about 1% of all cases of bowel obstruction and up to 90% of patients have underlying pathological cause for intussusception. The classic CT imaging appearances include presence of target sign, psuedokidney sign, and sausage appearance. CT also helps to identify primary intestinal intussusception (no lead point) from secondary intussusception (caused due to lead point). More than half of large bowel intussusceptions are secondary to malignancy and are primarily caused by adenocarcinoma and less commonly by metastasis. Surgical excision is the treatment of choice as large bowel intussusception may undergo perforation causing infective peritonitis or peritoneal carcinomatosis[25].

Colonic obstruction caused by external neoplastic causes

Colonic obstruction may also result from external compression by adjacent neoplastic lesions. Peritoneal spread of tumours is by local factors such as direct invasion, intraperitoneal seeding or systemic factors such as hematogenic or lymphatic spread. In the presence of primary colonic carcinoma, sometimes it may be difficult to differentiate whether LBO is caused by primary tumour or by external neoplastic cause[25].

ROLE OF IMAGING IN EVALUATION OF BOWEL OBSTRUCTION

Diagnosis of SBO on radiographs

Radiography is the initial imaging modality performed in case of suspected SBO as it is inexpensive and readily available. The accuracy of radiographs for diagnosis range from as low as 50% up to 80%. The accuracy of SBO is improved if one performs X-rays in dependent (supine) and non-dependent (decubitus) views. The radiographic findings of SBO are shown in Table 5[10].

Table 5. Radiographic Signs in Small Bowel Obstruction[10]

Type of Radiograph	**Specific Signs**
Supine or prone	1. Dilated gas or fluid-filled small bowel (>3 cm)
	2. Dilated stomach
	3. Small bowel dilated out of proportion to colon
	4. Stretch sign
	5. Absence of rectal gas
	6. Gasless abdomen
	7. Pseudotumor sign
Upright or left lateral decubitus	1. Multiple air fluid levels
	2. Air fluid levels longer than 2.5 cm
	3. Air fluid levels in same loop of small bowel of unequal heights
	4. String of beads sign

Diagnosis of SBO on CT

Currently CT is considered as investigation of choice for evaluation of SBO with a reported sensitivity and specificity of 95% in diagnosing high-grade obstruction. CT however has lesser accuracy when demonstrating low-grade obstruction. There has been a debate on usage of oral contrast media in SBO. It is believed that if the oral contrast manages to pass through the site of obstruction, then high grade obstruction can be ruled out and this was one of key points for using oral contrast. However, in the recent times, there has an argument regarding the use of oral contrast media as radiologists are of opinion that the negative contrast in bowel (air and water) provide sufficient negative contrast to visualize bowel. Patients with SBO are also at risk of aspirating oral contrast media as they already feel nauseated and can throw up at any time. In cases of high-grade obstruction, the positive contrast rarely travels beyond the point of obstruction, thus limiting its usefulness. Additionally, one does not have to wait for 2-3 hour delay in CT examination when patient is having oral contrast. CT enterography is also not recommended in evaluation of SBO[10].

Some of the key points suggestive of SBO are dilated proximal small bowel loops (>2.5 cm) with decompressed distal small bowel loops and colon, presence of multiple air-fluid levels, “small bowel feces” sign and string of beads sign. The “small bowel feces” sign has been shown to have present in about 5 to 7% of patients. It is often observed in patients with high grade SBO. The importance is of “small bowel feces” sign is that it usually lies just proximal to the site of obstruction. The CT criteria for diagnosis of SBO are listed in Table 6[10].

Table 6. CT Criteria for Diagnosis of Small Bowel Obstruction[10]

<table>
<tr><th>Criteria</th><th>Specific Criteria</th></tr>
<tr><td rowspan="2">Major</td><td>Small bowel dilated to 2.5 cm or greater and colon not dilated (<6 cm)</td></tr>
<tr><td>Transition point from dilated to nondilated small bowel</td></tr>
<tr><td rowspan="2">Minor</td><td>Air fluid levels</td></tr>
<tr><td>Colon decompressed</td></tr>
</table>

CT is able to accurately determine the site transition of normal and abnormal bowel and consequently the site of obstruction, which is difficult to determine on radiographs. The accuracy of CT for determining site of obstruction ranges from 63 to 93%. Furthermore additional information on the viability of mesenteric vessels, bowel wall characteristics, and mesenteric changes can also be determined, which is not possible on radiographs. Although CT is not effective in identifying adhesions, other causes of SBO such as external and internal hernias, neoplasms, inflammatory conditions (diverticulitis, appendicitis, abscesses, Crohn's disease), gall stones can be identified by CT imaging[10].

Level of Obstruction

Computed tomography is useful to identify the anatomical level of SBO[3]. Identifying level of obstruction by observing mucosal fold pattern (to identify the jejunal or ileal loops) can be misleading as the mucosal folds tend to become effaced once loops are dilated. Additionally, distended small bowel loops tend to migrate from their normal anatomic location. The relative position of jejunum and ileum can become reversed and jejunal loops may be displaced to pelvis and the ileal loops may migrate to the upper abdomen[27]. Therefore, determination of obstructed site based on location of dilated small bowel loops alone is misleading. The ideal approach is to identify the site of obstruction and compare the relative lengths of dilated vs collapsed bowel loops[3]. This approach can be considered as more objective.

Diagnosis of LBO on radiographs

Abdominal radiographs are the initial modality of investigation in patients with suspected bowel obstruction. As in evaluation of SBO, both dependent (supine) and non-dependent (decubitis) radiographs should be taken. Although the sensitivity of radiographs in diagnosis of LBO is similar to SBO (84% versus 82% respectively), radiographs have lower specificity for diagnosing LBO when compared with SBO (72% vs 83%). It may therefore be challenging to differentiate between LBO and colonic pseudo-obstruction. Radiological findings helpful in diagnosing LBO are mentioned in Table 7[28].

Table 7. Radiographic Signs in Large Bowel Obstruction[28]

Type of Radiograph	**Specific Signs**
Dependent	Dilated colon >6 cm (>9 cm for cecum) proximal to site of obstruction
	Paucity or absence of gas distal to obstruction
Non-dependent	Air-fluid levels on erect or decubitus views
	Pneumatosis intestinalis
	Portal venous gas
	Pneumoperitoneum

Diagnosis of large bowel obstruction on CT:

On CT imaging the findings suggestive of LBO are similar to that noted on radiographs. The large bowel wall diameter varies widely from 3 to 8 cm and is largest at cecum. It is believed that cecal diameter > 8 cm is suggestive of LBO. Cecum is an easily distensible organ and therefore it gets distended with least pressure. This in turn increases the risk for ischemia and necrosis. Although the exact size of cecum at risk for perforation is not known, it is believed to lie between 9 to 12 cm. CT is able to determine accurately the presence of cecal ischemia, which puts it for risk of perforation. CT also helps to differentiate between pneumatosis intestinalis and air trapped between fecal matter and bowel lumen, which is not possible on radiographs. CT also helps to identify the cause of LBO, which is commonly due to volvulus and malignancy in our population. CT has shown sensitivity and specificity of 96% and 93% for detection of LBO[28].

CT IMAGING: BACKGROUND

There has been a significant improvement in the field of medical imaging in both the technologic and clinical areas following the discovery of X-ray in 1895 by Wilhelm Conrad Roentgen, a German Physicist. Innovations in technology are a norm in the Radiology Department, with introduction of new ideas and methods and refinements in existing techniques happening continuously. One such evolution is the invention of computed tomography (CT)[29].

The first idea of a computed tomography machine was conceived by Sir Godfrey Hounsfield in 1967 and the first patient was scanned for brain cyst in 1971[29].

Sir Godfrey Hounsfield, an electronic engineer working at the Central Research Laboratories of EMI in England commenced work on image reconstruction in 1968.His original apparatus consisted of a collimated isotope source mounted on a lathe bed. The objects examined were phantoms contained within a ten-inch water. The scan took nine days to complete because of the low intensity of the X-ray radiation source, and a further two and half hours to process the reading through a computer. The resulting image though of poor quality proved that the system worked. To provide sufficient intensity the equipment was modified by replacing the isotope with an industrial X-ray tube[29].

A prototype scanner was then developed and installed in Atkinson Morley Hospital in Wimbledon, England on 1st October 1971. The first patient scan was a 41 year old female with suspected frontal lobe tumor, the tumor was clearly demonstrated on the scan[29].

Hounsfield and Ambrose presented their paper on CT to the annual congress of the British Institute of Radiology on 20th April 1972 to great acclaim. The first CT papers, by these authors appeared in British Journal of Radiology in 1973. The invention of this technique resulted in the award of 1979 Nobel Prize in physiology and medicine to Sir G. N. Hounsfield, Central Research Lab., England (EMI), and A. N. Cormack of Physics Department, Tufts University, Massachusetts, U.S.A. Advanced Technological Developments. Over the last ten years, four different generations of CT scan equipment were produced. The most important improvements have been in the reduction in the single image generation time from five minutes to 2.5 seconds in the third and fourth generations scanners and an increase in spatial resolution and contrast[29]. The introduction of second generation CT scanners further reduced the scan time from about six minutes to about two minutes. Late second generation CT scanners with ≥ 20 detectors further reduced scanning time to about ≤ 20 seconds. This dramatically improved quality of body scans, which could not be performed previously within a breath hold. The third generation scanners further reduced the scan time to 5 seconds or less, which has now further improved to about 0.33 seconds[30].

Slip Ring Scanners

There was no significant improvement in CT technology following 4^{th} generation CT scanners in late 1980's. The only limitation at that time was interscan delays. Following one 360^0 rotation, the cables connecting rotating components (x-ray tube and detectors) to the rest of the gantry required rotation to be stopped and reversed for next slice, all of which added time of scan. All this changed with application of low-voltage slip rings. Slip rings provide electricity to the rotating components without fixed connections (Figure 1). Slip rings made it possible for continuous rotation, thereby reducing scan time. This technology also paved the way for introduction of spiral/helical CT scans[30].

“In the mid-1980s, another high speed CT scanner was introduced, which was referred to as the Electron Beam CT (EBCT) scanner used for imaging cardiovascular system. In 1989, Dr. Willi Kalender introduced volume scanning by using spiral / helical CT scanners. In spiral/helical CT Scanners, a thin X-ray beam traces a path around the patient and scans a volume of the tissue. Recently, dual slice spiral /helical CT scanner and multislice CT scanners were introduced which mainly increase the speed and volume of scan. Volume CT scanning has resulted in a wide range of applications such as CT fluoroscopy, CT angiography, 3D Imaging and virtual reality imaging”[29].

Figure 1. Slip-ring technology in Siemens Somatom Emotion CT scanner

CLINICAL STUDIES

Intestinal obstruction is responsible for approximately 20% of surgical admissions of patients with acute abdomen. The early diagnosis of bowel obstruction is critical in preventing complications, particularly perforation and ischemia[31].

The morbidity and mortality associated with acute small-bowel obstruction (SBO) is significant. Small-bowel obstruction caused by postoperative adhesions accounted for 70% of all cases. Other common causes include hernias, neoplasms, and Crohn's disease[32]. Mechanical large bowel obstruction is four to five times less common than small bowel obstruction, most common cause being neoplasms[33].

Plain films are usually obtained initially and have overall 69, 57, and 67% sensitivity, specificity, and accuracy, respectively. Its accuracy in diagnosing the site and cause of obstruction and the presence of strangulation is even lower. Given the relative lack of sensitivity and specificity of plain film findings in patients with symptoms of bowel obstruction, in acute settings, CT plays a central role in evaluation. CT can demonstrate pathologic processes involving the bowel wall as well as the mesentery, mesenteric vessels, and peritoneal cavity[3].

CT has a reported sensitivity of 81–94%, specificity of 96%, positive predictive value (PPV) of 91%, and negative predictive value (NPV) of 93% in the diagnosis of high-grade obstructions[34,35]. CT also has a high sensitivity and specificity in evaluation of bowel ischemia in the setting of SBO[35,36]. However, reliability of CT decreases when all grades of small-bowel obstructions are considered with a reported sensitivity of 64% and specificity of 79%[34], which is expected.

A study has shown that CT finding of partial SBO is likely to represent a clinical condition that will resolve with conservative management[35].

Sheikh et al conducted a prospective study in 60 patients to evaluate the role of CT in evaluation of various bowel pathologies over a period of 24 months. There were 12 cases of appendicitis and 28 cases of inflammatory bowel diseases of which seven were intestinal tuberculosis and four patients had IBD. Remaining 17 patients had non-specific colitis and were managed conservatively. There were three cases of bowel malignancy. CT showed 100% sensitivity and specificity in diagnosing appendicitis and sensitivity and specificity of 96.3% and 100% respectively for diagnosing inflammatory bowel and 100% accuracy in diagnosis of bowel perforation. The authors concluded that CT has good sensitivity and specificity for evaluation of bowel obstruction and also helps in revealing cause of obstruction (intraluminal, intramural and extrinsic factors). CT is helpful to further characterize bowel lesion as neoplastic or inflammatory. Complications such as strangulation and perforation can also be evaluated on CT. CT should be considered as investigation of choice in patients with suspected bowel obstruction, particularly in patients in whom clincal and radiographic findings are indeterminate[37].

A prospective follow-up study in 40 patients was conducted by Saini et al to evaluate the efficacy of CT in diagnosing the presence, level, degree and cause of intestinal obstruction, to evaluate the role of CT in detection of complications, and assess impact of CT in optimizing decision making and management. The authors reported that CT had 85% sensitivity and 70% specificity in diagnosing bowel obstruction. CT findings were consistent with intraoperative findings in 22 of 30 patients (73%), which was statistically significant (P = .003). The authors also observed that although CT is highly sensitive in diagnosing high-grade obstruction, the sensitivity reduces in low-grade obstruction, which is consistent with data from literature. The authors concluded that CT is sensitive and specific in determining presence of bowel obstruction and should be recommended as the investigation of choice in patients with suspected bowel obstruction as it can affect the management outcome in these patients. CT also helps to evaluate closed loop obstruction and to evaluate strangulation of bowel[38].

Pongpornsup S et al conducted a retrospective study in 35 patients to evaluate the role of CT in diagnosis of SB) and to identify the cause of SBO for further management. The authors reported that CT had diagnosed 25 cases of SBO of which one was false positive. The sensitivity, specificity, and accuracy of CT in diagnosis of SBO was reported to be 96%, 100%, and 97% respectively. The causes of SBO in their study were adhesions (n=10), metastases (n=4), internal hernia (n=3), ileitis (n=2), midgut volvulus (n=1), inguinal hernia (n=1), benign stricture secondary to chronic pancreatitis (n=1), submucosal hemorrhage (n=1), post-radiative enteropathy (n=1), and SMA syndrome (n=1). The authors concluded that CT is highly sensitive and specific in diagnosis of SBO[39].

Keoplung et al conducted a retrospective study in 75 patients (76 CT studies) to evaluate the benefit of addition of multiplanar reformations (MPR) in evaluation of bowel obstruction when compared to axial plane alone. The final diagnosis was confirmed in 57 patients (58 CT studies) and included 25 cases of SBO and 33 cases of LBO. The authors observed that CT diagnosis of bowel obstruction was made in 54 patients on axial planes alone and in 55 patients with axial and MPR with resulting in sensitivity and specificity of 93.1% and 77.8% respectively for axial planes alone and sensitivity and specificity of 94.8% and 72.2% with axial and MPR planes. Similarly accuracy of axial plane in diagnosis of SBO and LBO was 90% and 88.5% respectively and accuracy of axial plane with MPR was 90% and 92.3% respectively. The authors concluded that CT is an excellent modality for evaluation of bowel obstruction and addition of MPR helps to improve diagnostic confidence[40].

Beattie et al conducted a study in 44 patients to assess efficacy of CT in diagnosis of acute LBO. The patients presented with clinical features of LBO and abdominal radiographs were suggestive of LBO or pseudo-obstruction. The investigators took additional prone and/or decubitus CT scans in patients in whom site of obstruction was not well delineated on supine CT (n = 33). The authors observed that out of 44 patients only 50% (n = 22) had LBO with carcinoma as the commonest cause in 18 patients. The remaining 22 patients did not have mechanical obstruction. There were two false-negative CT studies with one study showing mural wall thickening. Mass was identified in 14 of 17 patients with true positive CT. The sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of CT for diagnosis of mechanical LBO was 91% each. The positive and negative likelihood ratios for CT were 10.1 and 0.1, respectively. The authors concluded that CT with additional selective prone and/or decubitus sections is highly effective in diagnosis of mechanical LBO and should be initial study of choice[41].

The advantage of current-generation multidetector CT (MDCT) scanners is increased anatomical coverage with thinner sections, which provide high-quality multiplanar (MPR) images and fewer motion artifacts. Multiplanar views help identify the site, level, and cause of obstruction when axial findings are indeterminate[2,10].

Materials and methods

MATERIALS AND METHODS

Source of data:

This was a prospective observational study that was performed on 33 patients suspected to have intestinal obstruction referred to the Department of Radio Diagnosis at R. L. Jalappa Hospital and Research Centre attached to Sri Devaraj Urs medical college, Kolar. Study was conducted over a period of one and half years (Jan 2017 - June 2018).

Inclusion Criteria:

All patients with clinically suspected intestinal obstructions, who were referred to our department for abdominal CT scan for evaluation of obstruction and whose follow up regarding surgical or conservative management was available.

Exclusion Criteria:

All patients in whom either CECT could not be performed or surgical / conservative follow up was not available.

Method of collection of data:

Informed consent was taken from all patients for their willingness to participate in the study. Baseline data was collected from the patients along with pertinent clinical history and relevant lab investigations.

X-ray erect abdomen was performed as a part of standard protocol for the patients with suspected intestinal obstruction.

CT scan was performed using 16-slice Siemens® Somatom Emotion® machine. Follow up was undertaken for all patients undergoing surgery or those who were managed conservatively. Their surgical and histopathological findings were recorded.

CT Protocol

CT was performed with patients either having oral contrast. The oral contrast used was non-iodinated contrast agent Iohexol 300 (Ultravist®) diluted with plain drinking water in the ratio 1:80 to 1:100. Patients were advised to drink 2 L of water prior to start of intravenous (iv) study. In patients who were nil per oral (npo) no oral contrast was given. Contrast enhanced CT was performed with multiphase study, which included arterial, venous and delayed sequences. The details were as follows:

Slice thickness – 5 mm plain and contrast

Pitch – 1.2

kVp – 130 kVp for plain study followed by 110 kVp for arterial phase and 130 kVp for venous and delayed phases.

mAs – CARE Dose 4D®, which is automated exposure control (AEC) provided by Siemens.

Scan area – From base of lungs to pelvis

Type of CT scan – spiral/ helical CT was done.

Contrast agent – Iohexol 300 (Ultravist®) was injected intravenously at the rate of 3.5 to 4 mL/s. Quantity of contrast used was based on body weight and ranged from 1.25 to 1.5 mL/kg body weight.

Bolus tracking was used to initiate the CT scan following injection of iv contrast.

Arterial phase: The arterial phase was calculated at 3-5 seconds following bolus trigger or about 15-20 seconds following contrast administration. Slice thickness of 5 mm was used, which was then reformed to 0.75 mm thin sections. The thin sections were used to create 3D images.

Venous phase: The venous phase was calculated about 25-30 seconds following completion of arterial phase or about 65-70 seconds following contrast administration. Slice thickness of 5 mm was used, which was reconstructed to 1.5 mm for 3D reformations.

Delayed phase: The delayed phase was calculated about 240 seconds following completion of venous phase or 300 seconds following contrast administration. Slice thickness of 5 mm was used, which was reconstructed to 1.5 mm for 3D reformations.

Image Assessment

The images were transferred to work station (Myrian ® or Osirix ®), where the studies were reported by two radiologists who were blinded to each other's findings. The radiologists were aware of the clinical question for the study and had access to patient's files, results of other imaging tests (such as ultrasound and X-rays) and results of any previous studies in the same patient. The radiologists had 10 years and 5 years of experience in reporting abdominal CT studies. The findings were compared with each other and with final diagnosis, which was either through surgery or histopathology. All the patients had not undergone surgery and in cases managed conservatively, the final diagnosis was compared with clinical outcome.

Statistical Analysis

Data will be entered into Microsoft excel data sheet and will be analyzed using SPSS 22 version software. Categorical data will be represented in the form of frequencies and proportions. Chi-square will be used as test of significance. Collected data will be analyzed by sensitivity, specificity, positive predictive value, negative predictive value, accuracy and test of significance. P value <0.05 will be considered as statistically significant.

Figure 2. SIEMENS® SOMATOM EMOTION 16® CT scanner used in the study.

Results

RESULTS

In our study we screened 87 patients who presented with complaints of intestinal obstruction. Among them, 24 patients had showed normal bowel loops and peristalsis on ultrasound and unremarkable erect abdominal radiograph and were managed conservatively and did not undergo CT abdomen. CECT study was performed in finally in 59 patients as four patients had altered renal function and therefore those patients were taken up for surgery. Among them, 13 patients who underwent were lost to follow up. There were 11 patients who were diagnosed with non-bowel pathological causing mass effect and causing pseudo-obstruction and therefore were not considered. Two patients refused to participate in the study and data from 33 patients was included for final analysis (Figure 3).

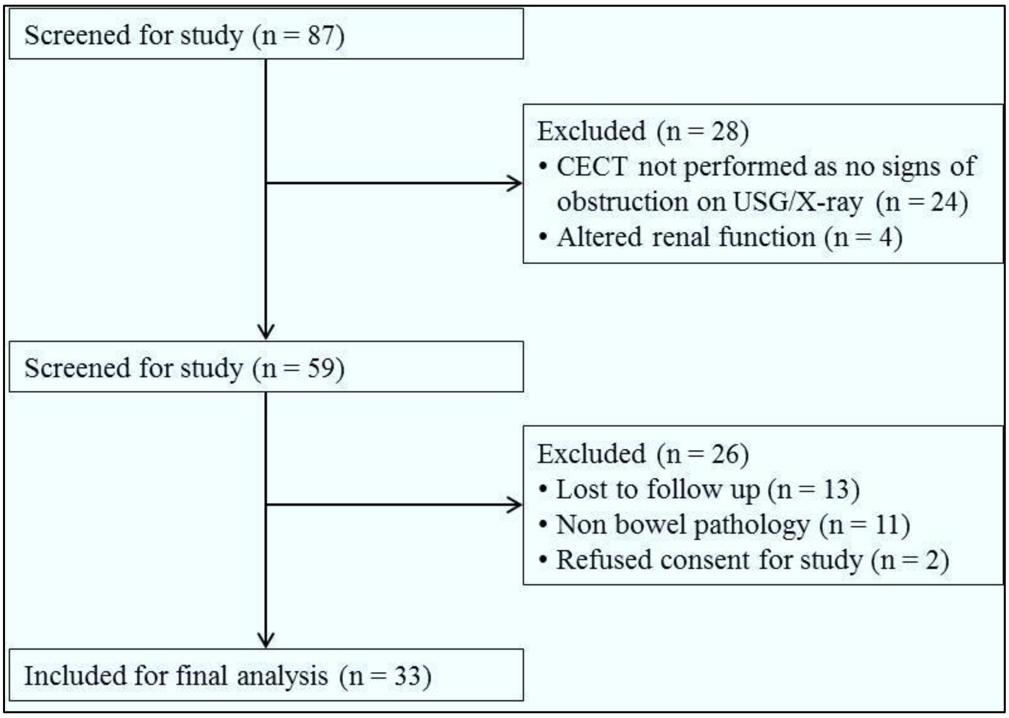

Figure 3. Study schematic.

Table 8. Age and Gender-wise Distribution of Patients

Age group (in years)	Male	Female	Total
11 to 20	0	1	1
21 to 30	2	2	4
31 to 40	0	2	2
41 to 50	4	3	7
51 to 60	2	4	6
61 to 70	4	5	9
71 to 80	1	3	4
Total	13	20	33

There were 33 patients with suspected bowel obstruction who were included in the final analysis. In our study there were more than 60% were females (n = 20) and remaining 13 patients were males (39.4%). The age and gender-wise distribution of patients is mentioned in Table 8. The commonest age groups belonged to patients of age 61 to 70 years (n = 9; 27.3%) (Figure 4) followed by 41 to 50 years (n = 7; 21.2%), 51 to 60 years (n = 5; 18.2%), 71 to 80 years and 21 to 30 years (n = 4 each; 12.1%), 31 to 40 years (n = 2; 6.1%) and lastly age group of 11 to 20 years (n = 1; 3%).

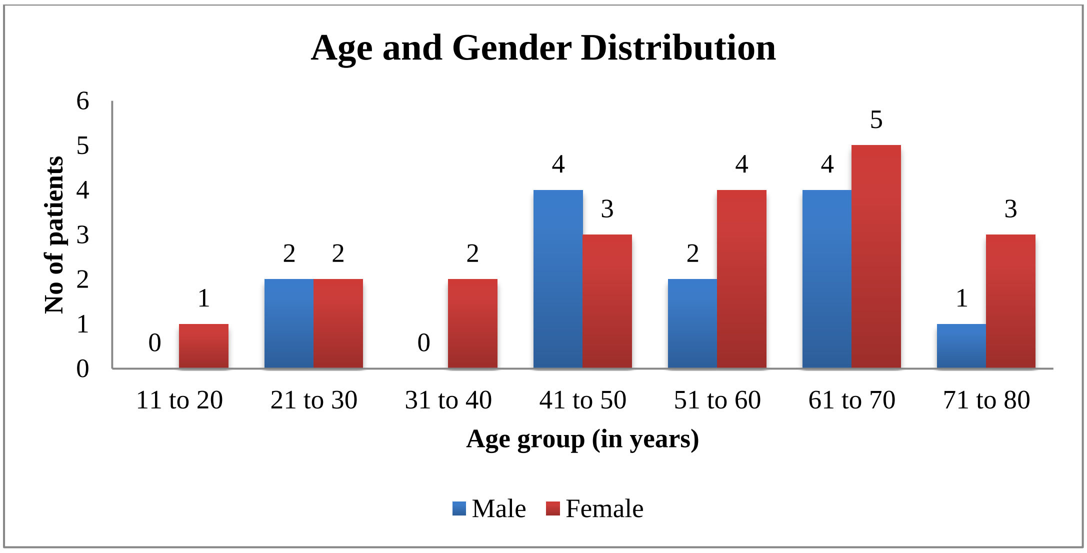

Figure 4. Age and gender-wise distribution of patients

Table 9. Clinical Presentation in Patients with Bowel Obstruction

Symptom	No of patients	%
Pain abdomen	33	100
Constipation	22	66.67
Abdominal distension	21	63.64
Vomiting	20	60.61
Obstipation	15	45.45
Bleeding per rectum	7	21.21

The commonest presenting complaint was pain abdomen, which was seen in all the patients followed by constipation in 22 patients (66.67%), abdominal distension (n = 21; 63.64%), vomiting (n = 20; 60.61%), obstipation (n = 15; 45.45%) and lastly bleeding per rectum in seven patients (21.21%) (Table 9). Majority of the patients presented with multiple complaints (Figure 5). Almost all the patients with SBO presented with pain abdomen, vomiting, abdominal distension and constipation. Majority of the patients with LBO presented with pain abdomen, constipation, obstipation, abdominal distension, and bleeding per rectum. None of the patients with SBO presented with bleeding per rectum. Similarly obstipation was also primarily a complaint in patients with LBO and only small percentage of patients with SBO had obstipation.

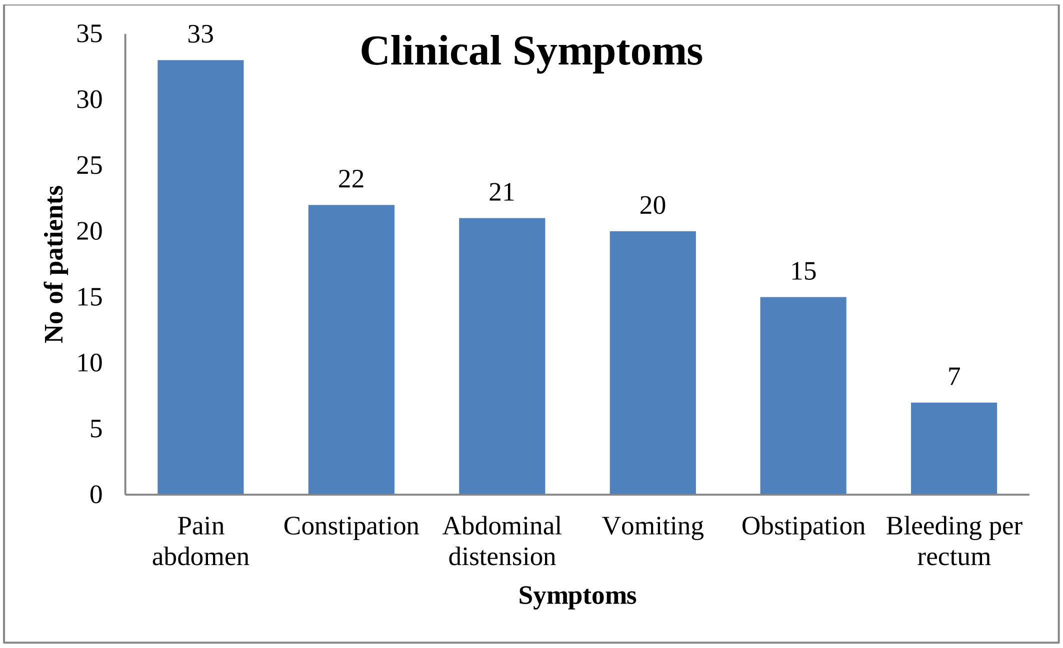

Figure 5. Clinical Presentation in patients presenting with bowel obstruction.

Table 10. Type of Modality & Bowel Obstruction

Type of modality showing bowel obstruction	**No of bowel obstruction**	**%**
X-ray	9	42.8
CT	24	109.09
Final diagnosis	22	100

At final diagnosis, there were 21 patients with intestinal obstruction (there were 22 instances of bowel obstruction, as one patient had both small and large bowel obstruction). In all of the patients with clinically suspected bowel erect X-ray abdomen was performed. On erect X-ray abdomen intestinal obstruction was suspected in nine patients (42.8%). The features seen in radiographs were multiple air-fluid levels in six patients (66.67%) followed by fluid-filled bowel loops (n = 4; 44.4%), and lastly gasless abdomen and dilated colon (n = 2 patients each; 22.2%). On CT there were 23 patients with 24 bowel obstruction (109.1%) who were suspected to have bowel obstruction (Table 10). CT over estimated two patients with bowel obstruction, which on surgery proved otherwise. Both these patients were diagnosed with small bowel obstruction. The finding seen in these two patients was dilated small bowel loops (>2.5 cm). The overall sensitivity, specificity, PPV, NPV and accuracy of CT was 100%, 83.33%, 64.71%, 91.67% and 94.21% respectively.

Table 11. CT Findings in Intestinal Obstruction

Finding	No of patients	%
Dilated bowel loops (>2.5 cm in small bowel and >6 cm in large bowel)	16	66.67
Air fluid levels	13	54.17
Ascites	7	29.17
Small bowel feces sign	3	12.50
Pneumatosis intestinalis	3	12.50
Absent / poor bowel wall enhancement	2	8.33
Strangulation	1	4.17
Gangrene	1	4.17
Engorged mesenteric vessels	1	4.17
High attenuation of bowel wall	1	4.17
Mesenteric haziness	1	4.17
Obliteration of mesenteric vessels	1	4.17

The commonest findings observed on CT were dilated bowel loops (>2.5 cm in small bowel and >6 cm in large bowel) in 16 patients (66.67%), followed by air fluid levels in 13 patients (54.17%), ascites in seven patients (n = 7; 29.17%), pneumatosis intestinalis and 'small bowel feces' sign in three patients each (12.5%), and absent or poor bowel wall enhancement in two patients each (8.33%) (Table 11). Other findings of strangulation, gangrene, engorged mesenteric vessels, high attenuation of bowel wall, mesenteric haziness and obliterated mesenteric vessels were seen in one patient each (4.17%) (Figure 6).

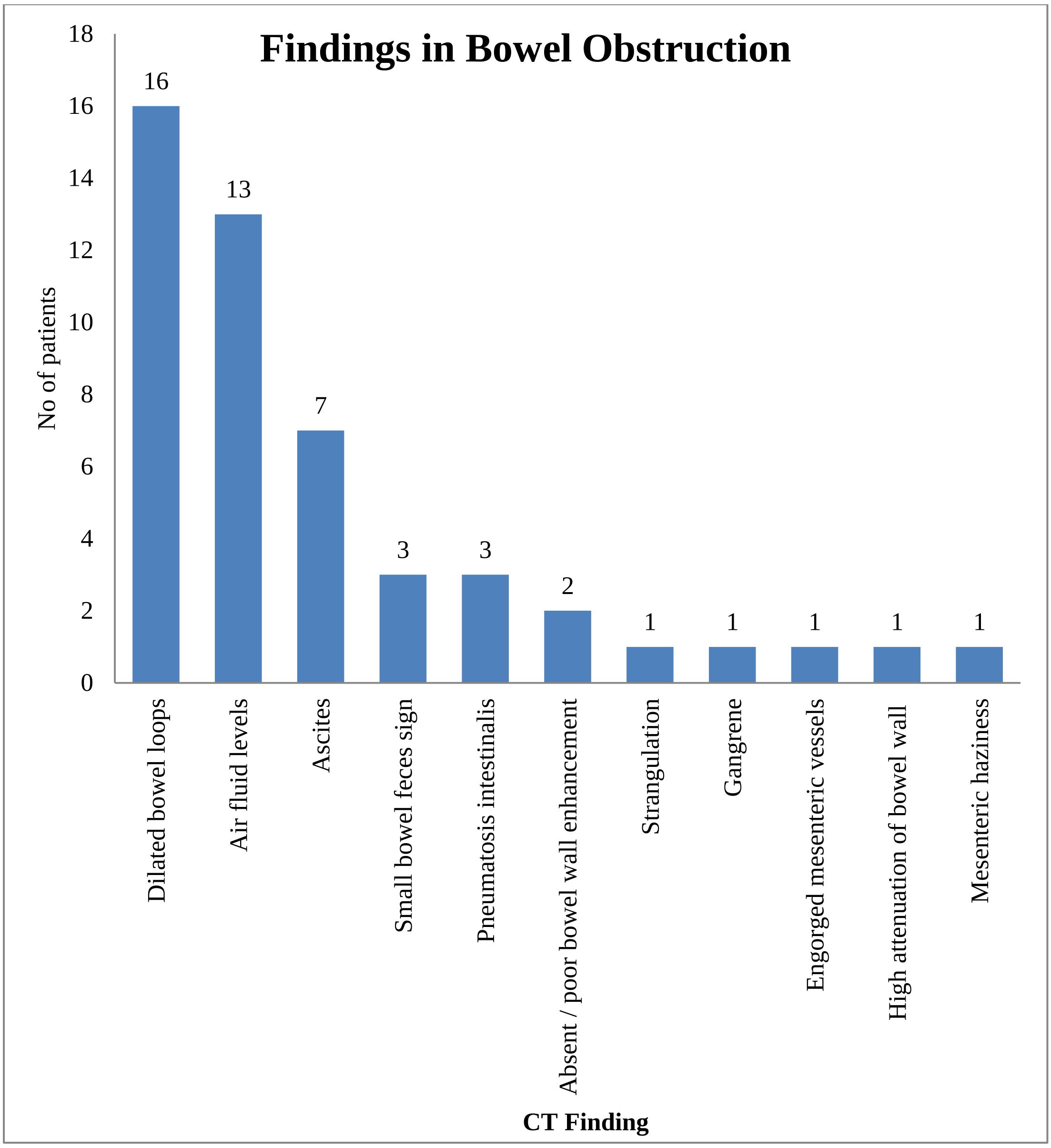

Figure 6. CT Findings in Intestinal Obstruction

Table 12. Level and Site of Bowel Obstruction

	Site	CT	Underwent surgery
Level of obstruction	Large bowel only	11	8
	Small bowel only	11	8
	Both	1	0
Sublevel of SBO	Duodenum	1	0
	Jejunum	1	0
	Ileum and ileocecal junction	10	8
Sublevel of LBO	AC	1	1
	TC	2	0
	DC	1	1
	SC & RSJ	3	2
	Rectum	5	4

In our study we observed a total of 23 patients (with 24 instances) with bowel obstruction of which 11 patients had SBO only, 11 patients had LBO only and one patient had both LBO and SBO, thus making 12 findings of SBO and LBO each. Surgery was performed in eight patients with SBO and LBO each (Table 12). Among patients with SBO only, those with intestinal Koch's and SMA syndrome were managed conservatively and were not operated. When only LBO was considered, patients with inoperable carcinoma rectal or colon were managed conservatively. One patient with peritoneal carcinomatosis had both SBO and LBO probably caused due to ascites and peritoneal adhesions. Because of peritoneal carcinomatosis, this patient was not operated and was managed conservatively. The lesion was causing obstruction in ileum & transverse colon.

Among SBO only, the commonest level of obstruction was ileum and ileocecal junction (n = 10; 83.3%). Duodenal obstruction and jejunal obstruction were seen in one patient each (8.33%). The patient with duodenal obstruction was diagnosed with SMA syndrome and was managed conservatively. The patient with jejunal obstruction was diagnosed on CT as primary carcinoma which was managed conservatively due to advanced disease and diagnosis was confirmed on biopsy. Among the 10 patients with ileal and ileocecal junction obstruction four patients were suspected to have adhesions of which two cases were confirmed during surgery. Two patients with CT diagnosed adhesions were found to have no evidence of intestinal obstruction on surgery. The patients improved post-surgery. Three patients had intestinal tuberculosis of which two patients were operated and one patient was managed conservatively. There were two patients who had benign stricture caused due to Crohn's disease. Both the patients were operated.. The remaining patient was diagnosed with peritoneal carcinomatosis and was managed conservatively (Table 13).

Among LBO the commonest site of obstruction was rectum in 41.67% of patients (n = 5) followed by sigmoid colon (n = 3; 25%) and transverse colon (n = 2; 16.67%). LBO at ascending colon and descending colon were observed in one patient each (8.3%). Four patients with carcinoma rectum were operated and the diagnosis was confirmed histopathologically. One patient with carcinoma rectum was managed conservatively due to advanced disease and diagnosis was confirmed with biopsy. There were 2 cases with carcinoma sigmoid colon and rectosigmoid junction of which one was operated and finding was confirmed histopathologically. One was managed conservatively due to advanced disease and diagnosis was confirmed on biopsy. There was one case of sigmoid volvulus, which was managed surgically and finding was confirmed. There were carcinomas once each in ascending, transverse & descending colon of which ascending and descending colon tumours were operated and their histopathology report came out to be adenocarcinoma. The carcinoma of transverse colon was managed conservatively due to advanced disease and diagnosis was confirmed as adenocarcinoma with biopsy.

Table 13. Correlation Between CT Diagnosis and Final Diagnosis

		CT Diagnosis						
		Primary bowel tumour	Adhesions	TB stricture	Benign stricture	SMA syndrome	Sigmoid volvulus	Peritoneal carcinomatosis
Final Diagnosis	Ca jejunum	1						
	Ca AC	1						
	Ca TC	2						
	Ca DC	1						
	Ca SC & RSJ	3						
	Ca rectum	5						
	Adhesions		2					
	No cause		2					
	ITB			3				
	CD				2			
	SMA syndrome					1		
	Sigmoid volvulus						1	
	Peritoneal carcinomatosis							1

AC = ascending colon; Ca = carcinoma; CD = Crohn’s disease; CT = computed tomography; DC = descending colon; ITB = ileal/ileocecal tuberculosis; RSJ = rectosigmoid junction; SC = sigmoid colon; SMA = superior mesenteric syndrome; TB = tuberculosis

IMAGES

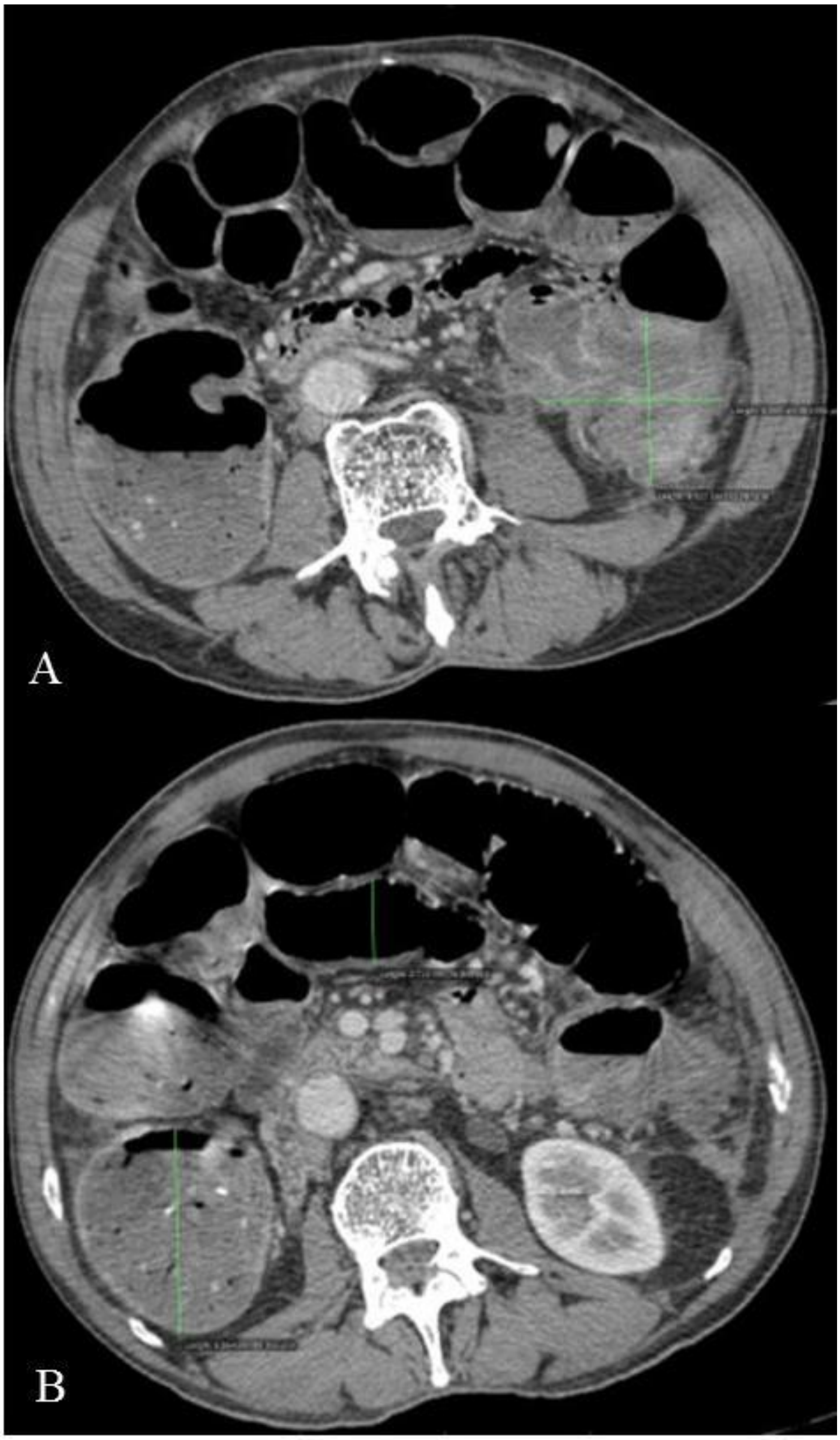

Figure 7. Large bowel neoplasm. Contrast enhanced images show a neoplastic lesion at the junction of descending and sigmoid colon with luminal narrowing and proximal bowel dilatation.

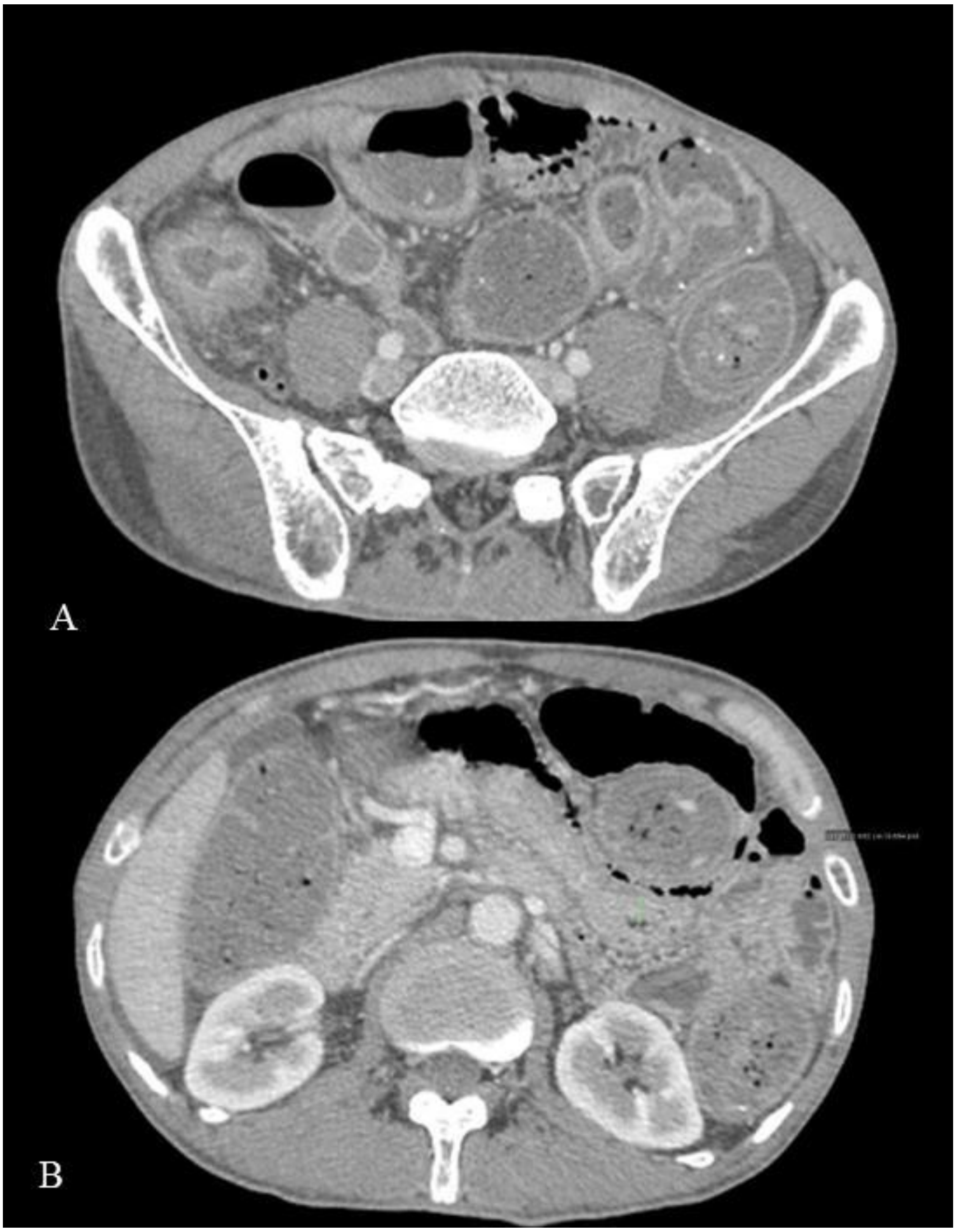

Figure 8. Crohn’s disease. Contrast enhanced images show ileocecal wall thickening with pericecal fat stranding. Long segment small bowel thickening with focal strictures also noted.

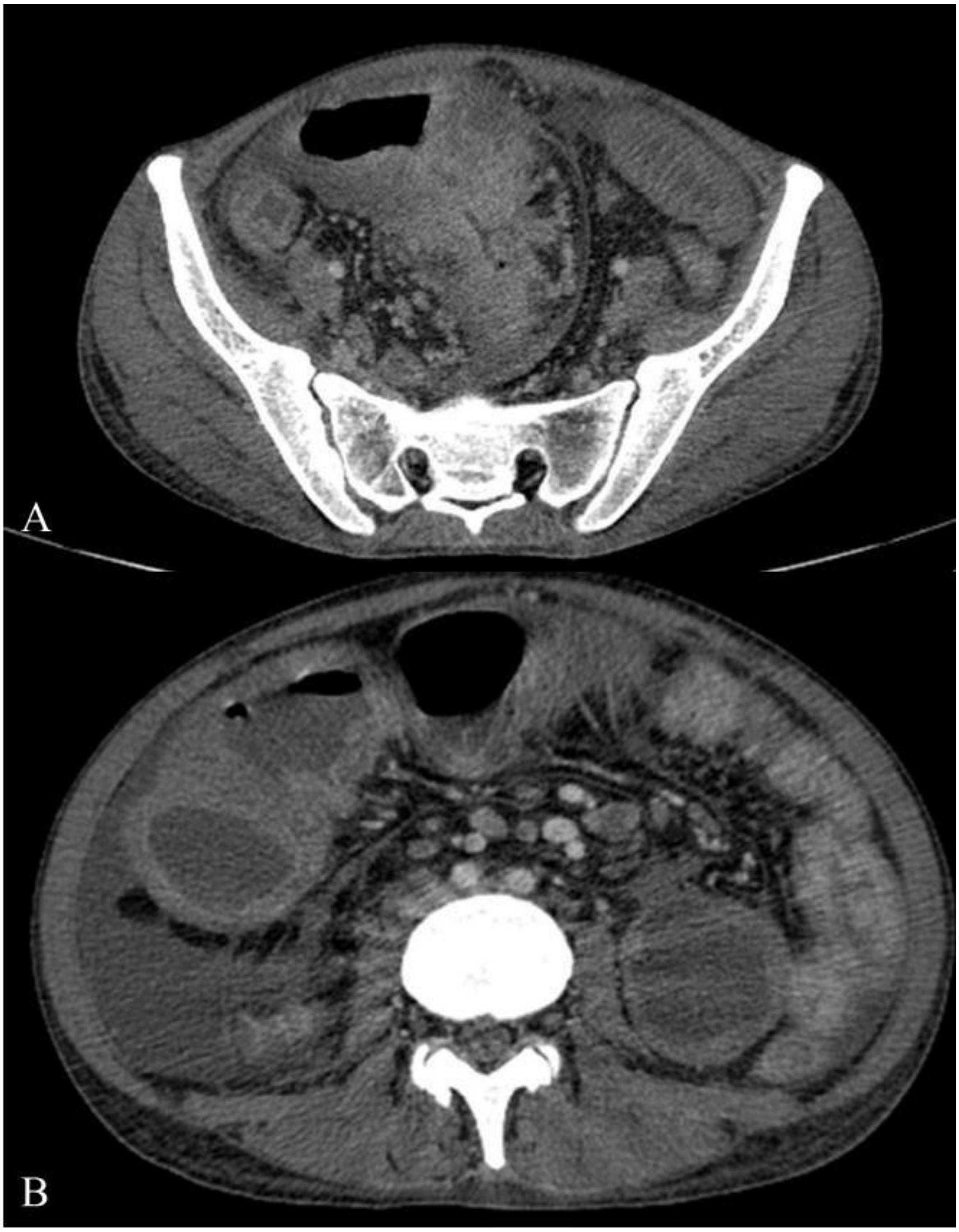

Figure 9. Contrast enhanced images show circumferentially enhancing mass in the terminal ileal region with features of proximal obstruction. Multiple lymph nodes also noted. Laparotomy and Histopathology showed stricture of tubercular etiology.

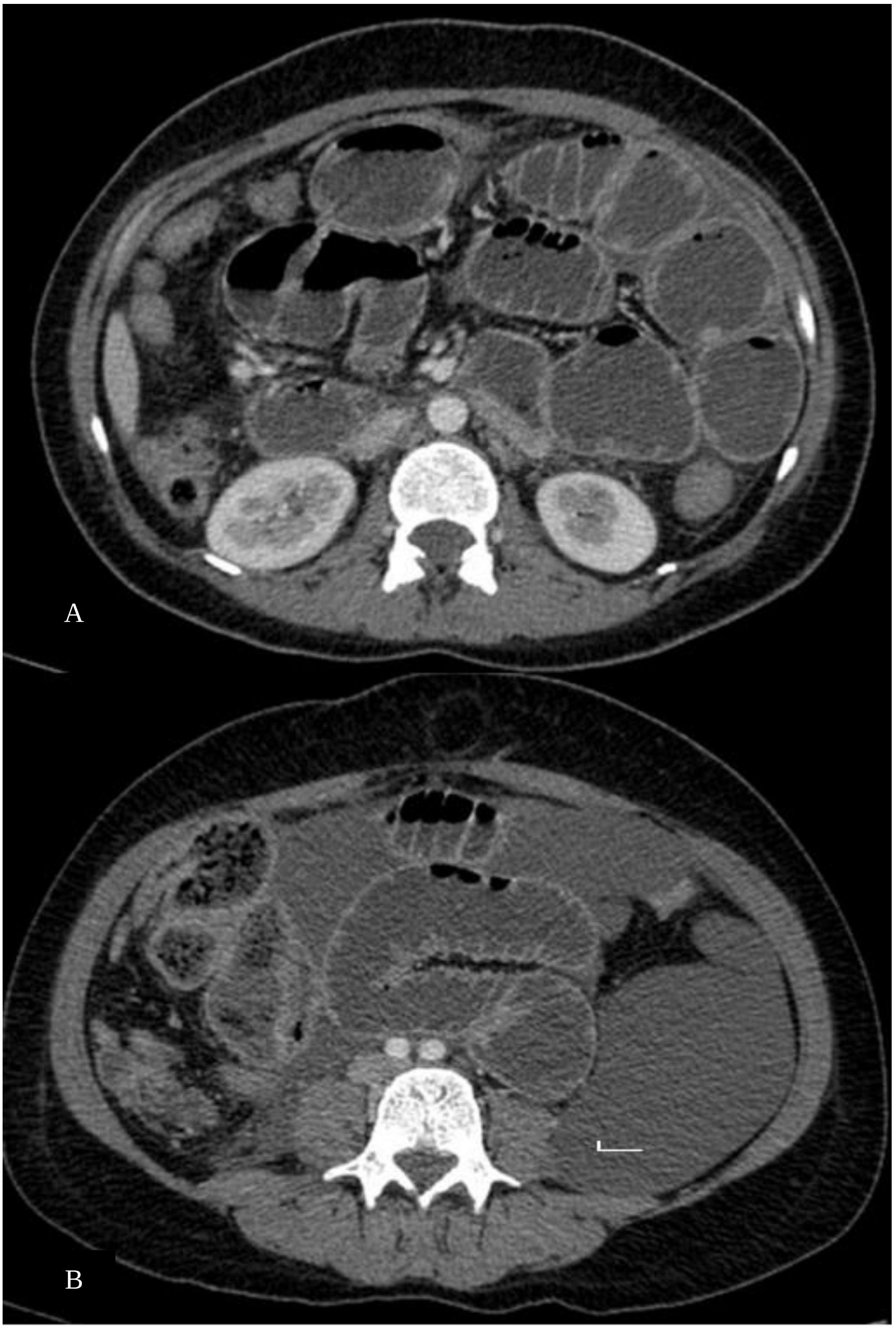

Figure 10. Case of Adhesions. Contrast enhanced images shows features of high grade mechanical obstruction with transition at distal ileum. Small bowel faeces sign noted.

Figure 11. Contrast enhanced CT images shows small bowel obstruction with abrupt transition in the distal ileum. Mild ascites also noted. Laparotomy proved postoperative adhesions.

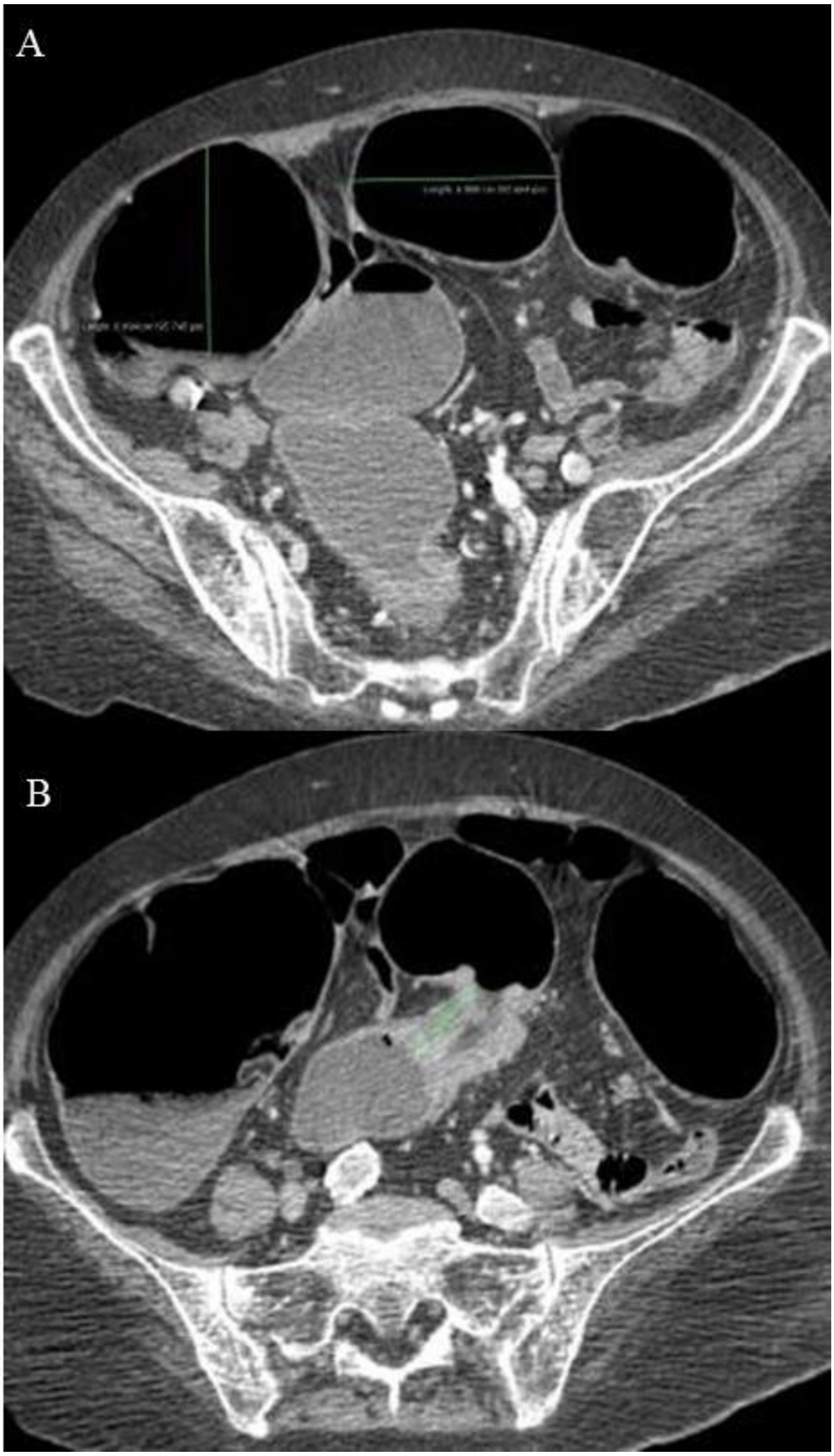

Figure 12. Case of carcinoma sigmoid colon. Contrast enhanced images sigmoid colon growth causing luminal stenosis with features of large bowel obstruction.

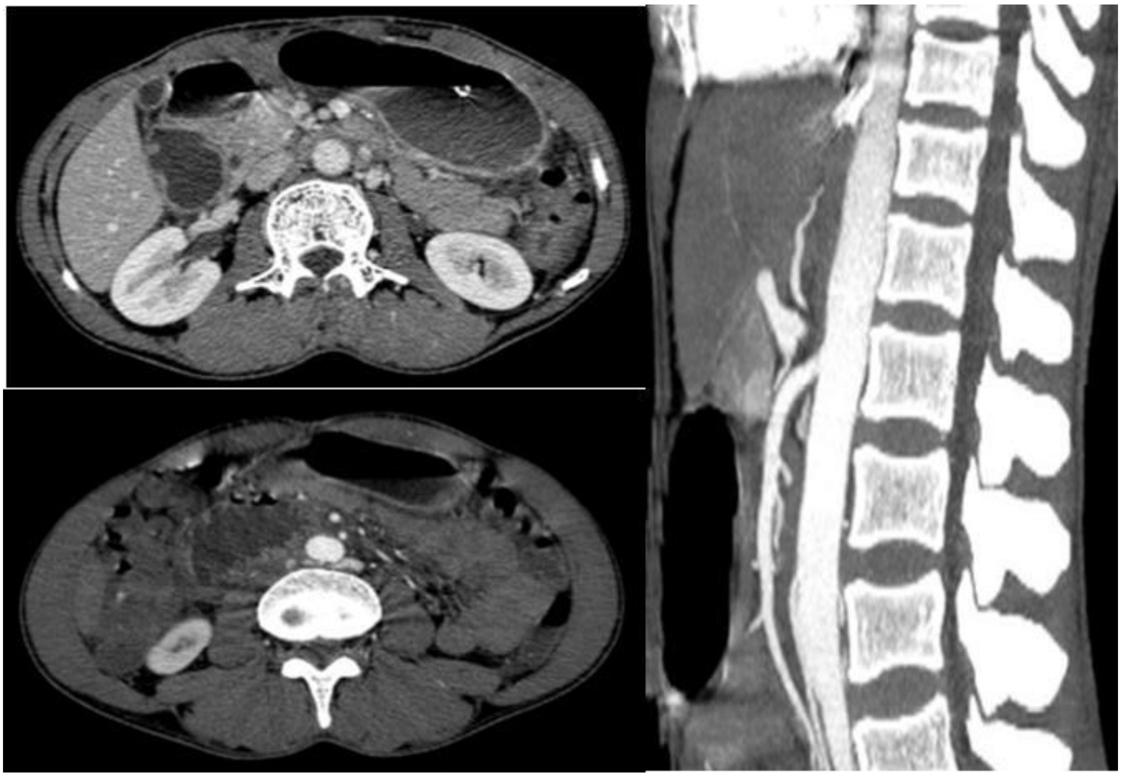

Figure 13. Case of superior mesenteric artery syndrome. Contrast enhanced images grossly distended stomach, 1st and 2nd part of Duodenum with transition at 3rd part. There is marginally reduced aorto –mesenteric distance.

Discussion

DISCUSSION

Intestinal obstruction is one of important differential diagnosis in a patient presenting with acute abdomen. Clinical diagnosis of intestinal obstruction can be challenging and imaging a significant role in diagnosis of intestinal obstruction. Currently CECT abdomen is considered as the most appropriate radiological investigation in evaluation of suspected small and large bowel obstruction. CT is able to demonstrate the level of bowel obstruction, diagnose common causes of bowel obstruction and to differentiate between high- and low-grade obstruction. Furthermore CECT also helps to assess complications of obstruction, such as strangulation[42].

In our study, intestinal obstruction was observed more frequently in females as compared with males (60.6% vs 39.4% respectively). The commonest age groups belonged to patients of age 61 to 70 years (n = 9; 27.3%) followed by 41 to 50 years (n = 7; 21.2%) and 51 to 60 years (n = 5; 18.2%). Least number of patients were in the age group of 11 to 20 years (n = 1; 3%).

The gender distribution in our study is different as compared with findings reported by Saini et al, who in their study of 40 patients in urban set up reported a male predominant populace with males constituting 67% (n = 27). They also reported the commonest age group of patients to be between ages 31 to 45 (n = 13; 33%) followed by 46 to 60 years (n = 12; 30%) and 15 to 30 years (n = 11; 27%). In our study we observed more number of cases of intestinal obstruction with increasing age, whereas Saini et al have reported more cases of intestinal obstruction in middle age group[38]. This difference in age-group and gender distribution could be attributed to different cultures and socioeconomic strata of patients observed in both the set ups.

The commonest presenting complaint in our study was pain abdomen, which was seen in all the patients followed by constipation in 22 patients (66.67%), abdominal distension (n = 21; 63.64%), vomiting (n = 20; 60.61%), obstipation (n = 15; 45.45%) and lastly bleeding per rectum in seven patients (21.21%). Majority of the patients presented with multiple complaints. Almost all the patients with SBO presented with pain abdomen, vomiting, abdominal distension and constipation. Majority of the patients with LBO presented with pain abdomen, constipation, obstipation, abdominal distension, and bleeding per rectum. None of the patients with SBO presented with bleeding per rectum. Similarly obstipation was also primarily a complaint in patients with LBO and only small percentage of patients with SBO had obstipation.

Data from various studies have also shown similar clinical complaints. Saini et al have reported pain abdomen in all the patients with bowel obstruction, abdominal distension in 82.5% of patients, vomiting in 67.5% of patients, followed by constipation/obstipation in 60% of patients, and abdominal tenderness in 65% of patients[38]. Singhania et al in the study on 53 patients with bowel obstruction reported abdominal distension in about 75.47% of patients, constipation in 73.58%, vomiting in 54.72% and abdominal pain in 56.6% of patients[43].

In our study, there were 21 patients with intestinal obstruction (there were 22 instances of bowel obstruction, as one patient had both small and large bowel obstruction). Erect X-ray abdomen showed intestinal obstruction in nine patients (42.8%). The features seen in radiographs were multiple air-fluid levels in six patients (66.67%) followed by fluid-filled bowel loops (n = 4; 44.4%), and lastly gasless abdomen and dilated colon (n = 2 patients each; 22.2%). On CT there were 23 patients (109.1%) (with 24 bowel obstruction) who were diagnosed with bowel obstruction. CT over estimated two patients with bowel obstruction, which on surgery proved otherwise. Both these patients were diagnosed with small bowel obstruction. The finding seen in these two patients was dilated small bowel loops (>2.5 cm). The overall sensitivity, specificity, PPV, NPV and accuracy of CT was 100%, 83.33%, 64.71%, 91.67% and 94.21% respectively. In our study CT evaluation was performed with axial sections and coronal and sagittal reformations for better understanding and delineation of bowel pathology.

Our study results are similar to data seen in other studies. Pongpornsup S et al in their study on 35 patients with SBO reported that CT diagnosed 25 cases with SBO of which one false positive and remaining were true positive. The authors reported that CT had overall sensitivity, specificity, PPV, NPV and accuracy of 96%, 100%, 100%, 90% and 97% respectively for diagnosing SBO[39]. Filippone et al reported 94% accuracy of CT in diagnosis of SBO. It was shown by the authors that addition of coronal reformations improved accuracy of CT in diagnosis of SBO (88% versus 94% in axial and axial with coronal reformations respectively). They also reported that when compared with final diagnosis axial sections alone were better at delineating SBO when compared with coronal reformations alone (92% vs 82% respectively). The authors also reported improved accuracy in diagnosis of LBO (88% versus 92% in axial and axial with coronal reformations respectively)[11]. Other studies have however reported lesser accuracy of CT in diagnosis of bowel obstruction. Singhania et al reported that CT identified 81.13% of cases of SBO. The sensitivity and specificity of CT was reported as 97.29% and 63.63% respectively. In their study of 53 patients, bowel obstruction was diagnosed in 43 patients on CT; however the final diagnosis revealed only 37 cases of intestinal obstruction[43]. Mallo et al conducted a review where they reported that the sensitivity of CT in diagnosis of SBO ranged from 81 to 100%, specificity 68 to 100%, PPV of 84 to 100% and NPV of 76 to 100%[35], which is consistent with our study. It is possible that studies having a lower sensitivity and specificity may be due to inherent selection bias in the studies. CT may show lower performance if the patient population in the study has relatively good number of patients with low grade obstruction. This can be concurred with data reported by Pongpornsup et al, who reported CT could identify all cases of high grade obstruction and could correctly identify only 58% of low grade SBO[39]. In our region we tend to get majority of cases with high grade obstruction, where the sensitivity and specificity of CT is high.

The commonest findings observed on CT in our study were dilated bowel loops in 16 patients (66.67%), followed by air fluid levels in 13 patients (54.17%), ascites in seven patients (n = 7; 29.17%), pneumatosis intestinalis and ‘small bowel feces’ sign in three patients each (12.5%). Other less common findings were strangulation, gangrene, engorged mesenteric vessels, high attenuation of bowel wall, mesenteric haziness and obliterated mesenteric vessels all of which were seen in one patient each (4.17%).

In our study the ‘small bowel feces’ sign was seen in three patients (12.5%) all of who had high grade bowel obstruction. ‘Small bowel feces’ sign is considered a highly specific sign for bowel obstruction. It is believed that in chronic or high grade obstruction, there is stasis and mixing of small bowel contents, which creates an appearance likened to feces in colon and hence the name ‘small bowel feces’ sign. It is usually present in high grade obstruction. The importance of this sign is that it is present just proximal to the site of obstruction/transition point and therefore helps in identifying the transition point in bowel obstruction[10,44]. Singhania et al reported presence of ‘small bowel feces’ sign in 5% of cases[43]. Lazarus et al reported a high ratio of small bowel feces sign in their study (n = 19 of 34 patients; 55.9%) in patients with SBO only. In their study they had relatively high number of moderate and high grade obstruction and this probably explains the unusually high percentage of ‘small bowel feces’ sign in their study[44].

In our study we observed a total of 23 patients (with 24 instances) with bowel obstruction of which 11 patients had SBO only, 11 patients had LBO only and one patient had both LBO and SBO, thus making 12 findings of SBO and LBO each. Surgery was performed in eight patients with SBO and LBO each. One patient with peritoneal carcinomatosis had both SBO and LBO probably caused due to ascites and peritoneal adhesions. Because of peritoneal carcinomatosis, this patient was not operated and was managed conservatively. The lesion was causing obstruction in ileum & transverse colon. Among SBO only, the commonest level of obstruction was ileum and ileocecal junction with duodenal obstruction and jejunal obstruction seen in one patient each. The patient with duodenal obstruction was diagnosed with SMA syndrome and was managed conservatively. The patient with jejunal obstruction was diagnosed on CT as primary carcinoma which was managed conservatively due to advanced disease and diagnosis was confirmed on biopsy. Among the 10 patients with ileal and ileocecal junction obstruction four patients were suspected to have adhesions of which two cases were confirmed during surgery. Two patients with CT diagnosed adhesions were found to have no evidence of intestinal obstruction on surgery. The patients improved post-surgery. Three patients had intestinal tuberculosis of which two patients were operated and one patient was managed conservatively. There were two patients who had benign stricture caused due to Crohn's disease. Both the patients were operated. The remaining patient was diagnosed with peritoneal carcinomatosis and was managed conservatively. Among LBO the commonest site of obstruction was rectum in 41.67% of patients followed by sigmoid colon (25%) and transverse colon (16.67%). LBO at ascending colon and descending colon were observed in 8.3%. Four patients with carcinoma rectum were operated and the diagnosis was confirmed histopathologically. One patient with carcinoma rectum was managed conservatively due to advanced disease and diagnosis was confirmed with biopsy. There were 2 cases with carcinoma sigmoid colon and rectosigmoid junction of which one was operated and finding was confirmed histopathologically. One was managed conservatively due to advanced disease and diagnosis was confirmed on biopsy. There was one case of sigmoid volvulus, which was managed surgically and finding was confirmed. There were carcinomas once each in ascending, transverse & descending colon of which ascending and descending colon tumours were operated and their histopathology report came out to be adenocarcinoma. The carcinoma of transverse colon was managed conservatively due to advanced disease and diagnosis was confirmed as adenocarcinoma with biopsy.

There are a variety of causes for bowel obstruction and these may vary in different studies. Filippone et al in their study in 73 patients reported SBO in 49 cases (67.1%) and LBO was seen in 24 cases (32.87%). Among SBO, 11 patients had jejunal obstruction, 13 patients had ileal obstruction and lastly 25 patients had terminal ileal obstruction, which is consistent with our findings. When the cause for SBO was evaluated, adhesions represented the majority of cases (n = 25; 51%) followed by hernia in 12 patients, small bowel tumour and bezoar in four patients each, closed loop hernia and internal hernia were seen in three and one patient respectively. The commonest location for LBO were descending colon in 37% of patients, cecum/ascending colon in 25% of patients, transverse colon in 17 of patients, and sigmoid colon in 21% of patients. When LBO was considered, neoplasms were cause in majority of patients (n = 17 of 21; 71%), which is consistent with our study. The other causes of LBO were volvulus in three patients and diverticulitis in four patients[11]. Singhania et al in their study on 53 patients reported SBO in 69.8% of patients and LBO in only 11.3% of patients. The commonest cause for SBO in their study was adhesions (22.92%) followed by inflammatory cause (9.3%), volvulus (8.33%), bowel neoplasm, hernia, and intussusceptions (6.25% each). Other uncommon causes were intestinal malrotation, extrinsic cause, and foreign body. No cause could be identified in about 16.28% of cases[43]. Pongpornsup et al in their study on 35 reported adhesions as commonest cause for SBO in 10 patients followed by metastases in four patients. Other uncommon causes were postradiative enteropathy, internal hernia, inguinal, submucosal hernia, midgut volvulus, SMA syndrome and benign stricture in one patient each[39]. Ali et al in their study in 40 patients demonstrated that both extrinsic and intrinsic causes were equally seen in SBO with commonest cause being adhesions, hernias followed by carcinoid tumour, appendicular cause, mesenteric vein thrombosis, Crohn’s disease, lymphoma, ileal carcinoma. Uncommon causes for SBO reported in their study were gall stone ileus, midgut volvulus and Ladd's band compressing duodenum. The unusual finding reported by the authors is probably due to the age group of patients (15 to 30 years) and the selective patient population who underwent CECT[45]. Megibow et al have also reported that adhesions were commonest cause for SBO followed by small bowel neoplasm, metastasis, Crohn's disease, hernia, hematoma, diverticulitis, intussusception, gall stone ileus and appendicitis[46]. The variation in the findings may be due to the native patient population and the disease demographics. While abdominal tuberculosis is common in our region and is a known cause of SBO in our population, the same may not be applicable in other patient population. We commonly receive patients with rectal carcinoma and this may the reason of high percentage of patients with rectal and rectosigmoid carcinomas in our studies.

Our study has certain limitations. Our patient population was limited and a more extensive patient population could have shown other factors causing bowel obstruction. Final diagnosis was not available in patients treated conservatively. This could theoretically affect the overall accuracy of CT in evaluation of bowel obstruction. It is possible that majority of patients who present with suspected intestinal obstruction have high grade obstruction and our data may have inadvertently been biased towards high grade obstruction and not general population.

Conclusion

CONCLUSION

Bowel obstruction is a fairly common encounter in clinical and radiological practice. There are various causes of bowel obstruction, which may make accurately diagnosing the cause of bowel obstruction a challenging task. CT provides accurate information in determining the cause and level of bowel obstruction. CT also helps to provide information on the viability of affected bowel tissue and help in treatment planning or identify the need for surgery. We recommend CT study as part of evaluation in patients presenting with bowel obstruction.

Summary

SUMMARY

Intestinal obstruction (IO) is one of the leading causes of admission in surgical and emergency units. Early diagnosis of bowel obstruction is critical in preventing complications, particularly perforation and ischemia. Previous studies have demonstrated computed tomography (CT) to be a valuable technique for imaging in intestinal obstruction.

This was a prospective observational study that was performed on 33 patients suspected to have intestinal obstruction referred to the Department of Radio Diagnosis at R. L. Jalappa Hospital and Research Centre attached to Sri Devaraj Urs medical college, Kolar. Study was conducted over a period of one and half years (Jan 2017 - June 2018). All patients with clinically suspected intestinal obstructions, who were referred to our department for abdominal CT scan for evaluation of obstruction and whose follow up regarding surgical or conservative management was available were included in the study. CT scan was performed using 16-slice Siemens® Somatom Emotion® machine. Follow up was undertaken for all patients undergoing surgery or those who were managed conservatively. Their surgical and histopathological findings were recorded.

There were 33 patients with suspected bowel obstruction who were included in the final analysis. In our study there were more than 60% were females (n = 20) and remaining 13 patients were males (39.4%). The commonest age groups belonged to patients of age 61 to 70 years (n = 9; 27.3%) followed by 41 to 50 years (n = 7; 21.2%),

51 to 60 years (n = 5; 18.2%), 71 to 80 years and 21 to 30 years (n = 4 each; 12.1%), 31 to 40 years (n = 2; 6.1%) and lastly age group of 11 to 20 years (n = 1; 3%).

The commonest presenting complaint was pain abdomen, which was seen in all the patients followed by constipation in 22 patients (66.67%), abdominal distension (n = 21; 63.64%), vomiting (n = 20; 60.61%), obstipation (n = 15; 45.45%) and lastly bleeding per rectum in seven patients (21.21%). Majority of the patients presented with multiple complaints. Almost all the patients with SBO presented with pain abdomen, vomiting, abdominal distension and constipation. Majority of the patients with LBO presented with pain abdomen, constipation, obstipation, abdominal distension, and bleeding per rectum. None of the patients with SBO presented with bleeding per rectum. Similarly obstipation was also primarily a complaint in patients with LBO and only small percentage of patients with SBO had obstipation.

At final diagnosis, there were 21 patients with intestinal obstruction (there were 22 instances of bowel obstruction, as one patient had both small and large bowel obstruction). In all of the patients with clinically suspected bowel erect X-ray abdomen was performed. On erect X-ray abdomen intestinal obstruction was suspected in nine patients (42.8%). The features seen in radiographs were multiple air-fluid levels in six patients (66.67%) followed by fluid-filled bowel loops (n = 4; 44.4%), and lastly gasless abdomen and dilated colon (n = 2 patients each; 22.2%). On CT there were 23 patients with 24 bowel obstruction (109.1%) who were suspected to have bowel obstruction. CT over estimated two patients with bowel obstruction, which on surgery proved otherwise. Both these patients were diagnosed with small bowel obstruction. The finding seen in these two patients was dilated small bowel loops (>2.5 cm). The overall sensitivity, specificity, PPV, NPV and accuracy of CT was 100%, 83.33%, 64.71%, 91.67% and 94.21% respectively.

The commonest findings observed on CT were dilated bowel loops (>2.5 cm in small bowel and >6 cm in large bowel) in 16 patients (66.67%), followed by air fluid levels in 13 patients (54.17%), ascites in seven patients (n = 7; 29.17%), pneumatosis intestinalis and ‘small bowel feces’ sign in three patients each (12.5%), and absent or poor bowel wall enhancement in two patients each (8.33%). Other findings of strangulation, gangrene, engorged mesenteric vessels, high attenuation of bowel wall, mesenteric haziness and obliterated mesenteric vessels were seen in one patient each (4.17%).

In our study we observed a total of 23 patients (with 24 instances) with bowel obstruction of which 11 patients had SBO only, 11 patients had LBO only and one patient had both LBO and SBO, thus making 12 findings of SBO and LBO each. Surgery was performed in eight patients with SBO and LBO each. Among patients with SBO only, those with intestinal Koch’s and SMA syndrome were managed conservatively and were not operated. When only LBO was considered, patients with inoperable carcinoma rectal or colon were managed conservatively. One patient with peritoneal carcinomatosis had both SBO and LBO probably caused due to ascites and peritoneal adhesions. Because of peritoneal carcinomatosis, this patient was not operated and was managed conservatively. The lesion was causing obstruction in ileum & transverse colon.

Among SBO only, the commonest level of obstruction was ileum and ileocecal junction (n = 10; 83.3%). Duodenal obstruction and jejunal obstruction were seen in one patient each (8.33%). The patient with duodenal obstruction was diagnosed with SMA syndrome and was managed conservatively. The patient with jejunal obstruction was diagnosed on CT as primary carcinoma which was managed conservatively due to advanced disease and diagnosis was confirmed on biopsy. Among the 10 patients with ileal and ileocecal junction obstruction four patients were suspected to have adhesions of which two cases were confirmed during surgery. Two patients with CT diagnosed adhesions were found to have no evidence of intestinal obstruction on surgery. The patients improved post-surgery. Three patients had intestinal tuberculosis of which two patients were operated and one patient was managed conservatively. There were two patients who had benign stricture caused due to Crohn’s disease. Both the patients were operated. The remaining patient was diagnosed with peritoneal carcinomatosis and was managed conservatively.

Among LBO the commonest site of obstruction was rectum in 41.67% of patients (n = 5) followed by sigmoid colon (n = 3; 25%) and transverse colon (n = 2; 16.67%). LBO at ascending colon and descending colon were observed in one patient each (8.3%). Four patients with carcinoma rectum were operated and the diagnosis was confirmed histopathologically. One patient with carcinoma rectum was managed conservatively due to advanced disease and diagnosis was confirmed with biopsy. There were 2 cases with carcinoma sigmoid colon and rectosigmoid junction of which one was operated and finding was confirmed histopathologically. One was managed conservatively due to advanced disease and diagnosis was confirmed on biopsy. There was one case of sigmoid volvulus, which was managed surgically and finding was confirmed. There were carcinomas once each in ascending, transverse & descending colon of which ascending and descending colon tumours were operated and their histopathology report came out to be adenocarcinoma. The carcinoma of transverse colon was managed conservatively due to advanced disease and diagnosis was confirmed as adenocarcinoma with biopsy.

Bowel obstruction is a fairly common encounter in clinical and radiological practice. There are various causes of bowel obstruction, which may make accurately diagnosing the cause of bowel obstruction a challenging task. CT provides accurate information in determining the cause and level of bowel obstruction. CT also helps to provide information on the viability of affected bowel tissue and help in treatment planning or identify the need for surgery. We recommend CT study as part of evaluation in patients presenting with bowel obstruction.

Bibliography

BIBLIOGRAPHY

1 Khurana B, Ledbetter S, McTavish J, Wiesner W, Pablo RR. Bowel Obstruction Revealed by Multidetector CT. Am J Roentgenol *2*002; 178:1139–44.

2 Nicolaou S, Kai B, Ho S, Su J, Ahamed K. Imaging of acute small-bowel obstruction. AJR Am J Roentgenol 2005;185:1036-44.

3 Furukawa A, Yamasaki M, Takahashi M, Nitta N, Tanaka T, Kanasaki S, Yokoyama, et al. Small Bowel Obstruction: Scanning Technique, Interpretation and Role in the Diagnosis. Semin in Ultrasound CT MR October 2003; 24(5):336-52.

4 Santillan CS. Computed tomography of small bowel obstruction. Radiol Clin North Am 2013;51:17-27.

5 Small intestine. In: Strandring S, editor. Gray's Anatomy. The anatomical basis of clinical practice. 40th edition. Chapter 66. Churchill Livingstone Elsevier. 2008. Page 1124-35.

6 Sharma R, Madhusudhan KS, Ahuja V. Intestinal tuberculosis versus crohn's disease: Clinical and radiological recommendations. Indian J Radiol Imaging 2016; 26: 161–172.

7 Large intestine. In: Strandring S, editor. Gray's Anatomy. The anatomical basis of clinical practice. 40th edition. Chapter 67. Churchill Livingstone Elsevier. 2008. Page 1136-62

8 Anatomy of the lower digestive tract. In: Rogers K, editor. The Human body. The digestive system. 1st edition. Chapter 3. Britannica Educational Publising. New York:NW. 2011. Page 61-73.

9 Kiela PR, Ghishan FK. Physiology of intestinal absorption and secretion. Best Pract Res Clin Gastroenterol 2016; 30:145–159.

10 Paulson EK, Thompson WM. Review of small-bowel obstruction: the diagnosis and when to worry. Radiology 2015;275:332-42.

11 Filippone A, Cianci R, Storto ML. Bowel obstruction: comparison between multidetector-row CT axial and coronal planes. Abdom Imaging 2007;32:310-6.

12 Assenza M, De Gruttola I, Rossi D, Castaldi S, Falaschi F, Giuliano G. Adhesions small bowel obstruction in emergency setting: conservative or operative treatment? G Chir 2016;37:145-149.

13 Margenthaler JA, Longo WE, Virgo KS, Johnson FE, Grossmann EM, Schifftner TL, et al. Risk factors for adverse outcomes following surgery for small bowel obstruction. Ann Surg 2006;243:456-64.

14 Barmparas G, Branco BC, Schnüriger B, Lam L, Inaba K, Demetriades D. The incidence and risk factors of post-laparotomy adhesive small bowel obstruction. J Gastrointest Surg 2010;14:1619-28.

15 Silva AC, Pimenta M, Guimarães LS. Small bowel obstruction: what to look for. Radiographics 2009;29:423-39.

16 Takeyama N, Gokan T, Ohgiya Y, Satoh S, Hashizume T, Hataya K, et al. CT of internal hernias. Radiographics 2005;25:997-1015.

17 Sunnapwar A, Sandrasegaran K, Menias CO, Lockhart M, Chintapalli KN, Prasad SR. Taxonomy and imaging spectrum of small bowel obstruction after Roux-en-Y gastric bypass surgery. AJR Am J Roentgenol 2010;194:120-8.

18 Crispín-Trebejo B, Robles-Cuadros MC, Orendo-Velásquez E, Andrade FP. Internal abdominal hernia: Intestinal obstruction due to trans-mesenteric hernia containing transverse colon. Int J Surg Case Rep 2014;5:396-8.

19 Anzidei M, Napoli A, Zini C, Kirchin MA, Catalano C, Passariello R. Malignant tumours of the small intestine: a review of histopathology, multidetector CT and MRI aspects. Br J Radiol 2011;84:677-90.

20 Sailer J, Zacherl J, Schima W. MDCT of small bowel tumours. Cancer Imaging 2007;7:224-33.

21 Boudiaf M, Soyer P, Terem C, Pelage JP, Maissiat E, Rymer R. CT evaluation of small bowel obstruction. Radiographics 2001;21:613-24.

22 Marinis A, Yiallourou A, Samanides L, Dafnios N, Anastasopoulos G, Vassiliou I, et al. Intussusception of the bowel in adults: a review. World J Gastroenterol 2009;15:407-11.

23 Sinha R, Verma R. Multidetector row computed tomography in bowel obstruction. Part 1. Small bowel obstruction. Clin Radiol 2005;60:1058-67.

24 Hayakawa K, Tanikake M, Yoshida S, Urata Y, Inada Y, Narumi Y, et al. Radiological diagnosis of large-bowel obstruction: nonneoplastic etiology. Jpn J Radiol 2012;30:541-52.

25 Hayakawa K, Tanikake M, Yoshida S, Urata Y, Yamamoto E, Morimoto T. Radiological diagnosis of large-bowel obstruction: neoplastic etiology. Emerg Radiol 2013;20:69-76.

26 Sinha R, Verma R. Multidetector row computed tomography in bowel obstruction. Part 2. Large bowel obstruction. Clin Radiol 2005;60:1068-75.

27 Burkill GJ, Bell JR, Healy JC. The utility of computed tomography in acute small bowel obstruction. Clin Radiol 2001;56:350-9.

28 Jaffe T, Thompson WM. Large-Bowel Obstruction in the Adult: Classic Radiographic and CT Findings, Etiology, and Mimics. Radiology 2015;275:651-63.

29 Sreeram E. Computed tomography: Physical principles, Clinical applications and Quality control. 2nd ed. Philadelphia PA. W B Saunders 2001.p1-28.

30 Goldman LW. Principles of CT and CT technology*. J Nucl Med Technol 2007; 35:115–28.

31 Khaled AM, Mohamed ES, Role of multislice CT in emergency department, Alexandria J medicine, June 2014; (50):171-8.

32 Szucs RA, wolf EL. Miscellaneous abnormalities of the colon. In: Gore RM, Levin MS, eds. Textbook of gastrointestinal radiology 2nd edition Philadelphia, Pa: Saunders, 2008; 1089-1102.

33 Desser TS, Megan G. Multidetector Row Computed Tomography of Small Bowel Obstruction. Semin_Ultrasound_CT MR 2008;29:308-21

34 Burkill GJC, Bell JRG, Healy JC. The utility of computed tomography in acute small bowel obstruction. Clin Radiol 2001; 56:350–59.

35 Mallo RD, Salem L, Flum DR. Computed tomography diagnosis of ischemia and complete obstruction in small bowel obstruction: a systematic review. J Gastrointest Surg 2005; 9(5):690–4.

36 Scaglione M, Grassi R, Pinto A, et al. [Positive predictive value and negative predictive value of spiral CT in the diagnosis of closed loop obstruction complicated by intestinal ischemia]. Radiol Med (Torino) 2004; 107:69–77.

37 Sheikh MT, Sheikh MT, Jan M, Khan HA, Vashisht GP, Wani ML. Role of multi-detector CT (MDCT) in evaluation of bowel diseases. J Clin Diagn Res 2017;11:TC11-TC13.

38 Saini DK, Chaudhary P, Durga CK, Saini K. Role of multislice computed tomography in evaluation and management of intestinal obstruction. Clin Pract 2013;3:e20.

39 Pongpornsup S, Tarachat K, Srisajjakul S. Accuracy of 64 sliced multi-detector computed tomography in diagnosis of small bowel obstruction. J Med Assoc Thai 2009;92:1651-61.

40 Keoplung S, Teerasamit W, Suvannarerg V. Diagnosis of bowel obstruction: added value of multiplanar reformations from multidetector CT in comparison with axial planes alone. J Med Assoc Thai 2013;96:1569-77.

41 Beattie GC, Peters RT, Guy S, Mendelson RM. Computed tomography in the assessment of suspected large bowel obstruction. ANZ J Surg 2007;77:160-5.

42 Stoker J, van Randen A, Laméris W, Boermeester MA. Imaging patients with acute abdominal pain. Radiology 2009;253:31-46.

43 Singhania KV, Mehta R, Kazi Z. Role of multidetector computed tomography in bowel obstruction. Int J Sci Stud 2017;5(5):131-134

44 Lazarus DE, Slywotsky C, Bennett GL, Megibow AJ, Macari M. Frequency and relevance of the "small-bowel feces" sign on CT in patients with small-bowel obstruction. AJR Am J Roentgenol 2004;183:1361-6.

45 Ali SA, Mansour MGE, Farouk O. Utility of 64-row multidetector computed tomography in diagnosis and management of small bowel obstruction. Egyptian J Radiol Nucl Med 2017;48:839-46.

46 Megibow AJ, Balthazar EJ, Cho KC, Medwid SW, Birnbaum BA, Noz ME. Bowel obstruction: evaluation with CT. Radiology 1991;180:313-8.

Annexures

ANNEXURE I

"ROLE OF COMPUTED TOMOGRAPHY IN THE EVALUATION OF INTESTINAL OBSTRUCTION"

PATIENT PROFORMA

Demographic details:

Name:

Age:

Clinical History:

Relevant lab investigation findings:

Histopathological findings with diagnosis:

Previous CT findings if available:

Present CT Findings with diagnosis:

Impression:

Final diagnosis:

ANNEXURE II

INFORMED CONSENT FORM

STUDY TITLE: **ROLE OF COMPUTED TOMOGRAPHY IN THE EVALUATION OF INTESTINAL OBSTRUCTION.**

CHIEF RESEARCHER/ PG GUIDE'S NAME: Dr. N. RACHEGOWDA

PRINCIPAL INVESTIGATOR: Dr. GNANA SWAROOP RAO POLADI

NAME OF THE SUBJECT:

AGE :

GENDER :

a. I have been informed in my own language that this study involves CT and use of contrast material as part of procedure. I have been explained thoroughly and understand its complication and possible side effects.
b. I understand that the medical information produced by this study will become part of institutional record and will be kept confidential by the said institute.
c. I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation at any time without prejudice to my present or future care at this institution.
d. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).
e. I confirm that ____________________ (chief researcher/ name of PG guide) has explained to me the purpose of research and the study procedure that I will undergo and the possible risks and discomforts that i may experience, in my own language. I hereby agree to give valid consent to participate as a subject in this research project.

ANNEXURE II

Participant's signature/thumb impression

Signature of the witness: Date:

1)

2)

I have explained to ___________________________ (subject) the purpose of the research, the possible risk and benefits to the best of my ability.

Chief Researcher/ Guide signature Date:

ANNEXURE II
ROLE OF COMPUTED TOMOGRAPHY IN THE EVALUATION OF INTESTINAL OBSTRUCTION

Patient Information Sheet

Principal Investigator: Dr. Gnana Swaroop Rao Poladi / Dr. N. Rachegowda

I, Dr. Gnana Swaroop Rao Poladi, post-graduate student in Department of Radio-Diagnosis at Sri Devaraj Urs Medical College. I will be conducting a study titled "Role of computed tomography in the evaluation of intestinal obstruction" for my dissertation under the guidance of Dr. N. Rachegowda, Professor & Head of department of Radio-Diagnosis. In this study, we will assess the confirmation or exclusion of clinically suspected mechanical intestinal obstruction, to assess the location & cause of obstruction and to look for its complications using CT.

You would have undergone CT/CECT abdomen before entering the study. You will not be paid any financial compensation for participating in this research project.

All of your personal data will be kept confidential and will be used only for research purpose by this institution. You are free to participate in the study. You can also withdraw from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution

Name and Signature of the Principal Investigator

Date

ANNEXURE III

KEY TO MASTER CHART

AD - Abdominal Distension

ADH – Adhesion

BO – Bowel Obstruction

BPR - Bleeding Per Rectum

BWE - Bowel Wall Enhancement

C – Constipation

COO – Cause of Obstruction

DL - Dilated Loops

EMV – Engorged Mesenteric Vessels

FLOO – Final Level of Obstruction

FLSBO – Final Level of Small Bowel Obstruction

FLLBO – Final Level of Large Bowel Obstruction

FCOO – Final Cause of Obstruction

HABW - High Attenuation of Bowel Wall

HP - Histopathology

HS - History of abdominal Surgery

LOO - Level of Obstruction

MH - Mesenteric Haziness

NBO – No Bowel Obstruction

OMV – Obliteration of Mesenteric Vessels

ANNEXURE III

PA - Pain Abdomen

PC – Peritoneal carcinomatosis

PI - Pneumatosis Intestinalis

SBFS - Small Bowel Feces Sign

SLLBO - Sublevel of Large Bowel Obstruction

SLSBO - Sublevel of Small Bowel Obstruction

SMAS – Superior Mesenteric Artery Syndrome

STR - Strangulation

SV - Sigmoid Volvulus

TBS - TB Stricture

V - Vomiting

Masterchart

Sl. No	Trial ID	Age (in years)	Age Group (years)	Sex	Clinical presentation							BO on X-rays	CT Findings															Management	HP				
					PA	V	C	OBS	AD	BPR	AS		DL	LOO	SL SBO	SL LBO	SBFS	COO	STR	Gangrene	EMV	BWE	HABW	PI	MH	OMV	ASC			FLOO	FLSBO	FLLBO	FCOO
1	488893	50	41 to 50	M	Y	Y	Y	N	Y	N	N	Y	Y	N	I&IC	NBO	N	PBT	N	N	N	N	N	N	N	N	N	S	BS	SB	I&IC	NBO	BS
2	373213	45	41 to 50	F	Y	N	Y	Y	Y	Y	N	N	Y	Y	NBO	NBO	N	PBT	N	N	N	N	N	N	N	N	Y	C	PBT	LB	NBO	DC	PBT
3	358986	55	51 to 60	M	Y	Y	Y	N	N	N	N	N	Y	N	I&IC	NBO	N	TBS	N	N	N	N	N	N	N	N	N	S	TB	SB	NBO	NBO	TBS
4	369964	65	61 to 70	F	Y	Y	Y	N	Y	N	Y	Y	Y	N	I&IC	NBO	Y	BS	N	N	N	N	N	N	N	N	N	S	BS	SB	I&IC	NBO	BS
5	345226	56	51 to 60	F	Y	N	N	N	N	N	N	Y	N	NBO	NBO	S&RSJ	N	NBO	Y	Y	N	N	N	N	N	N	Y	S	NA	LB	NBO	S&RSJ	SV
6	351466	13	11 to 20	F	Y	N	N	N	N	N	N	N	Y	N	I&IC	NBO	N	TBS	N	N	N	N	N	N	N	N	N	C	TB	SB	I&IC	NBO	TBS
7	329928	65	61 to 70	F	Y	Y	Y	N	Y	N	Y	Y	Y	N	I&IC	NBO	Y	ADH	N	N	N	N	N	N	N	N	N	S	NA	SB	I&IC	NBO	ADH
8	334801	28	21 to 30	M	Y	Y	N	N	Y	N	Y	Y	Y	N	I&IC	NBO	Y	ADH	N	N	N	N	N	N	N	N	N	S	NA	SB	I&IC	NBO	ADH
9	335647	29	21 to 30	F	Y	Y	N	N	N	N	N	N	Y	N	J	NBO	N	PBT	N	N	Y	N	N	Y	N	N	N	C	PBT	SB	J	NBO	PBT
10	319509	65	61 to 70	M	Y	N	Y	Y	Y	Y	N	Y	Y	Y	NBO	NBO	N	NBO	N	N	N	N	N	N	N	N	N	C	NA	NBO	NBO	NBO	NBO
11	305655	50	41 to 50	F	Y	N	Y	Y	Y	N	N	N	Y	Y	NBO	DC	N	PBT	N	N	N	N	N	N	N	N	N	C	NA	NBO	NBO	NBO	NBO
12	309589	30	21 to 30	F	Y	N	Y	N	N	N	N	N	N	NBO	NBO	NBO	N	NBO	N	N	N	N	N	N	N	N	N	C	NA	NBO	NBO	NBO	NBO
13	310080	50	41 to 50	F	Y	N	N	N	N	N	N	N	N	NBO	NBO	NBO	N	NBO	N	N	N	N	N	N	N	N	N	C	NA	NBO	NBO	NBO	NBO
14	152890	68	61 to 70	F	Y	N	Y	N	N	N	N	N	N	NBO	NBO	NBO	N	NBO	N	N	N	N	N	N	N	N	N	C	NA	NBO	NBO	NBO	NBO
15	242182	51	51 to 60	F	Y	Y	Y	Y	Y	N	N	N	Y	Y	NBO	TC	N	PBT	N	N	N	N	N	N	N	N	Y	S	NA	LB	NBO	TC	PBT
16	304481	50	41 to 50	M	Y	Y	Y	Y	Y	N	N	N	Y	NBO	NBO	NBO	N	NBO	N	N	N	N	N	N	N	N	N	C	NA	NBO	NBO	NBO	NBO
17	304816	76	71 to 80	F	Y	Y	Y	Y	Y	N	N	N	Y	Y	NBO	S&RSJ	N	PBT	N	N	N	N	N	N	N	N	Y	S	NA	LB	NBO	S&RSJ	PBT
18	288375	50	41 to 50	M	Y	Y	N	N	Y	N	N	N	N	N	I&IC	NBO	N	ADH	N	N	N	N	N	N	N	N	N	S	ADH	SB	NBO	NBO	NBO
19	363659	65	61 to 70	F	Y	Y	Y	Y	Y	N	Y	N	Y	N	I&IC	NBO	N	ADH	N	N	N	N	N	N	N	N	N	S	NA	SB	I&IC	NBO	NBO
20	533467	46	41 to 50	M	Y	Y	Y	Y	Y	N	N	Y	N	B	I&IC	TC	N	PC	N	N	N	Y	N	Y	Y	Y	Y	C	NA	Both	I&IC	TC	PC
21	541581	68	61 to 70	F	Y	N	Y	Y	Y	N	N	N	N	Y	NBO	S&RSJ	N	PBT	N	N	N	N	N	N	N	N	N	S	PBT	LB	NBO	S&RSJ	PBT
22	548440	70	61 to 70	M	Y	Y	N	N	N	N	N	N	N	NBO	NBO	NBO	N	NBO	N	N	N	N	N	N	N	N	N	C	NA	NBO	NBO	NBO	NBO
23	469944	34	31 to 40	F	Y	Y	Y	Y	Y	N	N	N	N	NBO	NBO	NBO	N	TBS	N	N	N	N	N	N	N	N	N	C	NA	SB	I&IC	NBO	NBO
24	548316	70	61 to 70	M	Y	N	Y	Y	Y	Y	N	N	Y	Y	NBO	R	N	PBT	N	N	N	N	N	N	N	N	N	S	PBT	LB	NBO	R	PBT

AD - Abdominal Distension, ADH – Adhesion, BO – Bowel Obstruction, BPR - Bleeding Per Rectum, BS – Benign Stricture, BWE - Bowel Wall Enhancement, C – Constipation, COO – Cause of Obstruction, DL - Dilated Loops, EMV – Engorged Mesenteric Vessels, FLOO – Final Level of Obstruction, FLSBO – Final Level of Small Bowel Obstruction, FLLBO – Final Level of Large Bowel Obstruction, FCOO – Final Cause of Obstruction, HABW - High Attenuation of Bowel Wall, HP – Histopathology, HS - History of abdominal Surgery, LOO - Level of Obstruction, MH - Mesenteric Haziness, NBO – No Bowel Obstruction, OMV – Obliteration of Mesenteric Vessels, PA - Pain Abdomen, PC – Peritoneal carcinomatosis, PI - Pneumatosis Intestinalis, SBFS - Small Bowel Feces Sign, SLLBO - Sublevel of Large Bowel Obstruction, SLSBO - Sublevel of Small Bowel Obstruction, SMAS – Superior Mesenteric Artery Syndrome, STR – Strangulation, SV - Sigmoid Volvulus, TBS - TB Stricture, V - Vomiting

Masterchart

Sl. No	Trial ID	Age (in years)	Age Group (years)	Sex	Clinical presentation							BO on X-rays	CT Findings															Management	HP				
					PA	V	C	OBS	AD	BPR	AS		DL	LOO	SL SBO	SL LBO	SBFS	COO	STR	Gangrene	EMV	BWE	HABW	PI	MH	OMV	ASC			FLOO	FLSBO	FLLBO	FCOO
25	550703	56	51 to 60	M	Y	N	Y	Y	Y	Y	N	N	N	Y	NBO	R	N	PBT	N	N	N	N	N	N	N	N	N	S	PBT	LB	NBO	R	PBT
26	553372	30	21 to 30	M	Y	N	N	N	N	Y	N	N	N	Y	NBO	R	N	PBT	N	N	N	N	N	N	N	N	N	C	PBT	LB	NBO	R	PBT
27	553782	78	71 to 80	F	Y	Y	N	N	N	N	N	N	N	NBO	NBO	NBO	N		N	N	N	N	N	N	N	N	N	C	NA	NBO	NBO	NBO	NBO
28	560276	65	61 to 70	M	Y	Y	Y	Y	Y	N	Y	Y	Y	N	I&IC	NBO	N	TBS	N	N	N	N	N	N	N	N	N	S	TB	SB	I&IC	NBO	TBS
29	561853	60	51 to 60	F	Y	Y	Y	Y	Y	Y	N	N	Y	Y	NBO	R	N	PBT	N	N	N	N	N	N	N	N	N	S	PBT	LB	NBO	R	PBT
30	567600	78	71 to 80	F	Y	Y	N	N	N	N	N	N	N	NBO	NBO	NBO	N	NBO	N	N	N	N	N	N	N	N	N	C	NA	NBO	NBO	NBO	NBO
31	586388	55	51 to 60	F	Y	N	N	N	N	N	N	N	N	N	D	NBO	N	PBT	N	N	N	N	N	N	N	N	N	C	NA	SB	D	NBO	SMAS
32	602354	33	31 to 40	F	Y	Y	Y	Y	Y	Y	N	N	N	Y	NBO	R	N	PBT	N	N	N	N	N	N	N	N	Y	C	PBT	LB	NBO	R	PBT
33	382669	75	71 to 80	M	Y	Y	Y	N	Y	N	N	Y	N	Y	NBO	AC	N	PBT	N	Y	N	Y	Y	Y	N	N	Y	S	PBT	LB	NBO	AC	PBT

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