Hard to Digest!

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Learning objectives

The aim of this poster is to present a comprehensive pictorial review of barium studies done to evaluate pathologies of the gastro-intestinal tract. This poster is targeted to serve as reference material for resident radiologists in understanding uncommon pathologies diagnosed on barium studies.
Background

BARIUM MEAL FOLLOW THROUGH

INDICATIONS:

1. Patients who have suspicion of small bowel disease- abdominal pain and diarrhoea.
2. Patients with suspected complete (or) near complete small bowel obstruction.
3. Patients who are suspected of suffering from Crohn's disease

If enteroclysis is the routine method, the barium follow through will only be used for
1. Elderly patients with suspected jejunal diverticulosis who present with malabsorption.
2. In patients who are unwilling or in whom it is not possible to perform intubation.

CONTRAINDICATIONS

1. Colonic obstruction
2. Suspected perforation
3. Paralytic ileus.

CONTRAST MEDIUM:

Medium density barium suspension (50-60 % w/v) containing a suspending agent (to maintain its stability and prevent flocculation) is used. High density barium (200-250 % v/w) may produce an appearance of fold thickening and clumping in the small bowel. Acid barium sulphate suspension produces spasm, enlarged folds and dilatation in duodenum and jejunum. Alkaline barium sulphate suspension may improve coating of the valvulae conniventes which increases diagnostic accuracy.

A water soluble iodine contrast agent such as gastrograffin is of limited value as it will be diluted and lose density in the small bowel. If they are used in cases of small intestinal obstruction, they may be so diluted by bowel content and their own osmotic action that they would fail to demonstrate either the site or the cause of the obstruction. In the old and frail patient or in young infants, there is additional hazard that their osmotic action
can seriously diminish blood volume. It is safe to use barium if small bowel obstruction is suspected and colonic obstruction is ruled out, to find the cause and site of the lesion. Therefore, the principal value of water soluble media is in the demonstration of leaks from the bowel.

**PREPARATION:**

Before any small intestinal study:

1. The colon should be cleaned by the administration of a suitable purgative. (purgative should be avoided in patient with suspected obstruction, acute exacerbation of Crohn’s disease or an Ileostomy.)

2. A low roughage diet and high fluid intake is also maintained for 48 hours prior to the investigation.

3. No food or fluid should be taken for 12 hours before the investigation. If the patient taking tranquilizers, antispasmodics and codeine, they should be stopped for 24 -48 hours before the examination.

**SMALL BOWEL FOLLOW THROUGH:**

Initially 150 ml of high density barium and effervescent agents are used to evaluate oesophagus, stomach and duodenum by means of double contrast examination. Later 200 ml of barium (20-25%) (to decrease the high density effect from double contrast study of upper GI study) followed by 250 ml of barium (40-45%) is given. Once this is completed, a series of overhead radiographs are obtained at half-hourly intervals till terminal ileum is reached.

**DEDICATED SMALL BOWEL FOLLOW THROUGH:**

- Single contrast technique.
- Double contrast technique

**Single contrast Technique:**

Single contrast technique is employed routinely in our department. Barium (600-900 ml) 50-60% is administered. Patient is asked to drink this as rapidly as possible. He is then put in the right side dependent position to aid rapid gastric emptying. After 15 to 20 minutes, a film is taken with the patient prone to separate the bowel loops, using high kV to demonstrated jejunum and proximal ileum. Subsequent films are taken at 15-30 minutes intervals till ileocaecal junction is opacified. To demonstrate ileocaecal junction,
supine right side up is the best position since ileum enters caecum in the posteromedial part.

**Advantages Of Prone Position:**

- Better separation and less overlap of bowel loops.
- In this position the centre of the abdomen is compressed making entire abdomen more uniform and thus more uniform x-ray penetration can be achieved.
- In this position loops of ileum tends to migrate cephalad and becomes less compacted in the pelvis which is often a common problem during procedure.

<table>
<thead>
<tr>
<th>Positioning</th>
<th>Purpose</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIRST Right side down dependent</td>
<td>To aid gastric emptying</td>
</tr>
<tr>
<td>SECOND Prone</td>
<td>To separate bowel loops</td>
</tr>
<tr>
<td>THIRD Right side up</td>
<td>To visualise IC junction</td>
</tr>
</tbody>
</table>

- Always empty the bladder prior to these spot films.
- Each overhead radiograph should be examined as soon as it is processed. Any suspected abnormalities should be evaluated with fluoroscopy and compression spot films
- Many authorities recommend that even in the absence of any abnormality, periodic fluoroscopic examination and compression spot images have to be taken.

Four spots of the ileo caecal junction should be taken with varying degrees of compression. Compression should be applied on the bowel loops to avoid overlap and to efface the mucosa so that the small lesions may not be missed and mobility of the loops can be well assessed. Spot films should be taken if required. The abnormalities must be shown atleast in two spot films taken at different times to demonstrate the persistence of the lesion.

Overlap of contrast filled bowel loops in the pelvis is often a problem. This can be overcome by the following methods.

1. Table head down
2. 30 degree caudal angled view of pelvis.
3. Emptying of urinary bladder prior to filming the ileal loops.

**IF DESIRED, GASTRIC AND BOWEL PERISTALSIS MAY BE INCREASED BY VARIOUS METHODS:**

1. **DRUGS:** Metaclopramide, neostigmine, Glucagon, Cholecystokinin. Of these, metaclopramide is most frequently used. This promotes bowel motility and can decrease the transit time of barium. Metaclopramide hydrochloride is available as:

   - Injection Perinorm or Injection Maxeron 5 mg/ml - 2 ml ampoule.
   - Tab. Perinorm or Maxeron- 10 mg tabs. Dose: 2 tabs given with the barium after stomach, duodenal study.

   **CAUTION:** In some patients it may lead to drowsiness (or) a parkinsonian like state. (Extra pyramidal reactions.)

2. **20-40 ml of sodium/meglumine diatrizoate** to the barium also reduces transit time.

3. **Use of cold water to dilute barium and intermittent ice cold water sips.**

   - Cooling makes the barium more palatable.
   - More importantly cold barium speeds the gastric emptying and passes more rapidly through the intestine than does the room-temperature barium.

4. **Preliminary cleansing of the colon and placing the patient in right lateral recumbent position. **

   **NOTE:**

   1. Polyposis: Films taken with collapsed loops show the polyps to best advantage.

   2. Diverticulosis: Delayed films may show persistence of barium in the diverticulae. Erect position will reveal any fluid levels caused by contrast media retained within the diverticulae.

   3. Large ulcers: Large collection of barium may be seen in the delayed film after the bowel loops have emptied the barium.
4. The transit time through the small bowel can vary greatly ranging between 15 minutes and 5 hours.
Findings and procedure details

PARAESOPHAGEAL HERNIA (Fig 1)

Paraesophageal hernia (PEH) (or rolling hernia) is an uncommon type of hiatal hernia (representing 5-15% of all hiatal hernias). The majority of the hiatal hernias being of the sliding type.

A paraesophageal hernia includes a peritoneal layer that forms a true hernia sac, distinguishing it from the more common sliding hiatal hernia. In PEH, there is an upward dislocation of the gastric fundus alongside a normally positioned gastroesophageal junction. The gastric fundus and, sometimes, abdominal viscera protrude into the mediastinum through the defect in the diaphragm. In contrast, a sliding hernia does not have a hernia sac and slides into the chest since the gastroesophageal junction (GE junction) is not fixed inside the abdomen.

Generally, a hiatus hernia is classified into four types.

**Type I = sliding hernia** - GE junction migrates into the posterior mediastinum through the hiatus.

**Type II-IV = paraesopahgeal hernias**

**Type II** - occurs when the fundus herniates through the hiatus alongside a normally positioned GE junction.

**Type III** - is a combination of types I & II hernias with a displaced GE junction as well as hernia sac containing portions of the fundus/body of stomach protruding through the hiatus.

**Type IV** - Characterized by displacement of the stomach along with other organs (colon, spleen, pancreas and small intestine) into the chest.

ZOLLINGER - ELLISON SYNDROME (Fig 2)

Zollinger-Ellison syndrome is a gastric hypersecretory condition caused by gastrinoma which leads to glandular hyperplasia and hypersecretion of HCl and pepsin resulting in thickened gastric rugae and multiple peptic ulcers within the stomach, duodenum and proximal jejunum.

Clinically, patients present with abdominal pain, diarrhea, GERD, GI bleeding, weight loss, nausea, and vomiting.

It manifests between the ages 20-50.
Treatment consists of control of gastrin hypersecretion, with proton-pump inhibitors and octreotide.

Surgical resection is the definitive treatment

**MENETRIER DISEASE (Fig 3)**

Menetrier’s disease is an idiopathic condition characterized by excessive mucus production, giant mucosal hypertrophy, hypo-proteinemia, and hypo-chlorhydria (due to the loss of parietal cells). The gastric rugae are typically wider than 25 mm and sometimes polypoid in appearance, the diameter of normal folds does not exceed 5mm.

The findings in Menetrier disease are non specific, the feature that sets Menetrier disease apart is that it produces the most impressive thickening.

The differential diagnosis of Ménétrier disease includes infectious causes (eg, cytomegalovirus, histoplasmosis, and H pylori), infiltrative diseases (eg, sarcoidosis and amyloidosis), malignancy (eg, Zollinger-Ellison syndrome, lymphoma, and gastric carcinoma), and varices.

It has bimodal age distribution. The childhood form is linked to cytomegaloviral infection and resolves spontaneously. It occurs in children younger than 10 years. The second peak is adulthood, and it progresses over time.

Males are affected more commonly than females.

**Clinical Features:**

Patients present with epigastric pain, hypoalbuminemia secondary to a loss of albumin into the gastric lumen, and an increased loss of enteric protein, which may manifest as an elevated fecal #1-antitrypsin level.

Other signs and symptoms are anorexia, asthenia, weight loss, nausea, gastrointestinal bleeding, diarrhea, edema, and vomiting.

It is associated with an increased risk for thromboembolic disease and gastric carcinoma.

**Treatment:**

Anticholinergics, prostaglandins, proton pump inhibitors, prednisone, histamine-2 blockers, and a high-protein diet.

If pharmacologic therapy fails or intractable pain or persistent loss of protein, and if malignancy could not be excluded, subtotal or total gastrectomy.

**GASTRIC POLYPS (Fig 4)**
Gastric polyps are the most common benign gastric tumor which are typically solitary, but may be multiple or diffuse.

Types of gastric polyps are-

**Hyperplastic:** Also known as an inflammatory polyp. Most common form, 75-90% of gastric polyps. They are multiple, small in size and may be located anywhere in the stomach. They have no malignant potential.

**Adenomatous:** Constitute 10-20% of all gastric polyps, with malignant potential. They are characterized by intestinal metaplasia and cellular atypia. They are solitary and located in the gastric antrum. They are larger and papillary in appearance.

**Villous:** Rare and have greatest malignant potential.

**Radiological findings:**

**Hyperplastic polyps:** Sessile or pedunculated. "Mexican hat sign" represents the stalk seen overlying the head of polyp. Usually < 2cm in diameter, Sharply marginated polyp with smooth circular border, Variable location and Often multiple.

**Adenomatous polyps:** Broad based elliptical polyp with or without pedicle, usually >2cm, lobulated but smooth surface and mostly located at the antrum.

**GASTRIC ADENOCARCINOMA (Fig 5)**

Gastric carcinoma is the third most common GI malignancy after colorectal and pancreatic cancer.

Predisposing factors include gastritis, adenomatous and villous polyp formation, gastrojejunostomy, partial gastrectomy, and pernicious anemia.

Most commonly located in the antrum or pylorus (50%); 60% are on the lesser curvature and 30% occur at the GE junction.

Adenocarcinoma is most common (95% of gastric malignancies).

Clinically patients present with abdominal pain, GI bleeding, and weight loss.

May infiltrate or spread superficially.

70% are ulcerating and infiltrating at the time of diagnosis.

Size correlates to prognosis.

**GASTRIC LYMPHOMA (Fig 6)**
The GI tract is the most common site of primary extra-nodal lymphoma (Non-Hodgkin > Hodgkin), involved in more than 50% of patients with disease.

Within the GI tract, the stomach is the site most frequently involved (40-75%)

Lymphoma accounts for only 3% of all gastric malignancy.

Gastric lymphomas are indistinguishable from carcinomas on UGIS, presenting as polypoid lesions, ulcerated masses, or thickened folds (differential for enlarged gastric folds). Unlike gastric carcinomas, lymphoma may also involve the small bowel, demonstrated by transpyloric spread.

Endoscopic biopsy is required for definitive diagnosis

**BEZOAR (Fig 7)**

Bezoars are concretions of accumulated undigested material unable to exit the stomach because of large size, indigestibility, gastric outlet obstruction, and decreased gastric motility.

Composed mainly of vegetable matter (phyto), hair (tricho), or milk curds (lacto).

Phytobezoars are the most common (55% of all bezoars).

Trichobezoars are seen more in pediatric patients and typically in young females with emotional problems.

Predisposing conditions include gastroparesis and prior gastric surgery.

Clinical symptoms include anorexia, abdominal pain, nausea, and vomiting.

A worrisome complication is mucosal ischemia and necrosis caused by mechanical compression and obstruction.

Management includes dissolution by oral ingestion of proteolytic enzymes, mechanical fragmentation via endoscopy, shock-wave lithotripsy (ESWL), or operative extraction.

Radiological findings:

Intraluminal filling defect not attached to bowel wall.

Upright view may show a mass at the air/fluid level interface (representing a bezoar floating on liquid within the stomach)

Partial or complete obstruction
JEJUNOGASTRIC INTUSSUSCEPTION (Fig 8)

Retrograde jejunogastric intussusception is a rare acute abdominal condition where the small bowel loops get incarcerated and may get strangulated inside the stomach.

Types of intussusception-

**Type I:** Afferent loop intussusception (antegrade)
**Type II:** Efferent loop intussusception (retrograde)
**Type III:** combined form.

Causes include a long afferent loop, jejunal spasm with abnormal motility, increased motility of efferent loop, adhesions leading to intussusception of a more mobile segment into fixed segment, widening of upper jejunum, causes of increased intra-abdominal pressure like vomiting, pregnancy and labor, dilated atonic stomach and retrograde peristalsis.

Clinically,

**Type 1** - Acute fulminant- sudden onset and colicky or constant upper abdominal pain associated with vomiting.

**Type 2** - Chronic intermittent- similar to the acute form but are milder and transient or sudden and spontaneous.

The presence of a mobile mass in association with pain and vomiting in a patient who has had a previous gastric surgery is considered virtually pathognomic of acute retrograde intussusception.

The mortality rate range from 10% for treatment within the first 48 hours to 50% within a 96 hour delay.

Treatment - manual reduction, resection of gangrenous bowel and revision of anastomosis, fixation of the jejunum to adjacent tissue like mesocolon, colon, or stomach may be added to prevent recurrence.

GASTRIC DIVERTICULUM (Fig 9)

The most common gastric form is a true diverticulum containing mucosa, submucosa, and muscularis propria and is caused by traction secondary to inflammation and scarring.

Located just below the level of the GE junction on the posterior aspect of the gastric fundus (75%). They are 3 or 4 cm large and rarely cause symptoms.

The second most common type is a partial antral gastric diverticulum (20%), a small 1-2 cm outpouching on the greater curvature of the distal gastric antrum. These are acquired
and are pulsion diverticula protruding through the muscularis propria. It results from a prior gastric ulcer.

Gastric diverticula are differentiated from ulcers by the absence of edema, mass, or wall rigidity.

**Radiographic Findings:**

Luminal outpouching with a broad neck, Normal mucosal pattern may be appreciated within the pouch, 75% near GE junction on posterior wall; 20% on greater curvature at the gastric antrum, Defect changes size and shape during imaging (vs. an ulcer which is rigid)

**EMPHYSEMATOUS GASTRITIS (Fig 10)**

Emphysematous gastritis is a rare and severe form of phlegmonous gastritis caused by mucosal disruption and is characterized by presence of gas in the stomach wall and clinical sepsis.

It is secondary to local spread through mucosa or due to haematogenous dissemination from a distant foci.

The stomach is a very uncommon site because of its acidity, abundant blood supply and an efficient mucosal barrier.

3 variants of gas within the wall of the stomach - interstitital gastric emphysema, cystic pneumatosis and emphysematous gastritis.

It is infectious in origin and the common isolated organisms are streptococci, e coli, enterobacter, pseudomonas, etc.

Predisposing factors are gastroenteritis, NSAID’s, diabetes, alcohol abuse, abdominal surgery, intake of corrosive substance, leukaemia and adenocarcinoma of the stomach.

Patients present with an acute abdomen with abdominal pain, vomiting, diarrhoea and haematemesis.

Gastric emphysema is asymptomatic and has benign course. The plain radiograph shows linear thin lucencies in the stomach wall.

In emphysematous gastritis, the radiograph shows an irregular mottled appearance with collection of gas in the stomach wall.

Treatment includes broad spectrum intravenous antibiotics and vigorous fluid support. Surgery is indicated in gastric infarction, perforation or failed medical management.
The mortality from emphysematous gastritis is more than 60% and the non-lethal complication like gastric strictures in up to 25%.

**GASTRIC VOLVULUS (Fig 11)**

Gastric volvulus is a rotational torsion of the stomach around either its longitudinal or transverse axis. >180 degrees of twisting produce complete luminal obstruction.

Predisposing factors are relaxed suspensory ligaments or mesenteries, diaphragmatic abnormalities, acute gastric dilatation, overfilling, prolonged vomiting, and sudden increases in intraabdominal pressure.

Occur at any age and are either an acute or chronic condition.

Acute volvulus presents with abrupt onset of epigastric pain and intractable retching. It is a surgical emergency as vascular occlusion with necrosis may ensue.

Two types of volvulus:

**Organoaxial**: Rotation around the gastric long axis extending from the cardia to pylorus. Most common and rarely associated with gastric ischemia.

**Mesenteroaxial**: Rotation around the gastric short axis extending from the greater to lesser curvature along the mesenteric attachment of the omentum (perpendicular to the long axis). This form is more serious due to an increased vascular compression and resulting tissue ischemia.

**Radiographic Findings**:  
Massively distended stomach with two air fluid levels on upright abdominal film, Incomplete or absent contrast filling the stomach, "Beak" sign at transition point

**Organoaxial**: The greater curvature will be situated superiorly with the lesser curvature lying inferiorly. The GE junction and pylorus will be in their normal locations.

**Mesenteroaxial**: The locations of the GE junction and pylorus are reversed relative to the body midline; the GE junction is located to the right, the pylorus to the left.

**PEPTIC ULCER DISEASE (DUODENAL ULCER) (Fig 12)**

Encompasses a number of entities, united by the presence of mucosal ulceration secondary to the effects of gastric acid. Since the recognition of Helicobacter pylori as a common causative agent, and the development of powerful anti-acid medications, peptic ulcer disease has become comparatively rare in western populations. Generally peptic
ulcer disease is encountered more frequently in males (M:F 3:1) and usually in the older population.

Risk factors include

• Helicobacter pylori infection
• NSAIDs
• Corticosteroids
• Severe physiological stress/illness (e.g. admission to intensive care)
• Zollinger Ellison syndrome.

Typically patients with upper abdominal pain and discomfort which is epigastric in location and 'gnawing' in character. Classically it is relieved by eating or antacids.

**CARCINOMA OF PANCREAS (Fig 13)**

Classified as

**A)** Ductal Adenocarcinoma

**B)** Nonductal neoplasms-

**C)** Cystic neoplasms: Mucinous macrocystic neoplasm and Serous microcystic neoplasm.

**D)** Endocrine tumors: Insulinoma, Gastrinoma, Glucagonoma, VIPoma, Somatostatinomas.

**E)** Solid & papillary epithelial neoplasm

**F)** Metastasis

Initial symptoms of pancreatic adenocarcinoma are abdominal pain, jaundice, and weight loss.

Most (60-70%) of these tumors are in the head of the pancreas, and 70% of these patients have obstructive jaundice as a result of common bile duct obstruction.

Advanced cancers have weight loss secondary to anorexia and pancreatic insufficiency.

Lab studies: elevated serum bilirubin, transaminases, alkaline phosphatase.

Levels of pancreatic enzymes (amylase, lipase, and elastase I) are elevated in only 30% of patients due to main pancreatic duct obstruction.
Tumor markers (CA19-9, DUPAN 2, Span 1, CEA) are indicators of advanced pancreatic carcinomas.

Tumors in the head of the pancreas are detected earlier, because they invade vital structures to cause symptoms. Carcinoma of the ampulla has the best prognosis.

**VILLOUS ADENOMA OF DUODENUM (Fig 14)**

The most common malignancy of the duodenum.

There is an increased incidence in patients with celiac disease (duodenum, proximal jejunum) and Crohn’s disease (ileum).

Signs and symptoms nonspecific, including abdominal pain, nausea, vomiting, weight loss, anemia.

**Radiological features:**

Occur within 25 cm of ligament of Treitz. Present as polypoid filling defects. These can ulcerate, leading to a "target" appearance on fluoroscopy. Some appear as infiltrative masses with annular luminal narrowing.

**GARDNER SYNDROME (Fig 15)**

Gardner syndrome is an autosomal dominant disease characterized by familial polyposis, osteomas of the long and short bones and cutaneous and subcutaneous lesions (epidermoid cysts, fibromas and desmoids tumors).

Osteoma occur in the sinuses and skull and its formation precedes polyposis and is the key to diagnosis.

It is associated with dental anomalies and must be considered in multiple impacted and unerupted teeth, supernumerary teeth, odontomas or dentigerous teeth.

Progression of polyps to malignancy is 100% and occurs by 30-50 years of age.

**PROLAPSING GASTRIC POLYPS (Fig 16)**

A huge hyperplastic gastric polyp can prolapse into the duodenum. The compression and obstruction of the ampulla of Vater by this polyp can cause acute pancreatitis.

**INTRADUODENAL DIVERTICULUM (Fig 17)**
The windsock sign is a typical appearance of intraluminal duodenal diverticulum described intraduodenal barium-contrast filled sac that is surrounded by a narrow lucent line (web or intraluminal mucosal diaphragm) which is well demonstrated as the barium in the duodenum passes distal to the diverticulum.

**SMA SYNDROME (Fig 18)**

Superior mesenteric artery (SMA) syndrome is characterized by compression of the third portion of the duodenum between the aorta and the superior mesenteric artery resulting in chronic, intermittent or acute complete or partial duodenal obstruction.

Patients present with vague abdominal symptoms, early satiety and anorexia or recurrent abdominal pain, vomiting.

It is associated with conditions causing severe weight loss-

- Anorexia nervosa
- Malabsorption
- Hypercatabolic states (burns, major surgery, malignancy)
- Severe congestive heart failure causing cachexia

**Radiological features:**

- Dilated fluid and gas filled stomach
- Dilatation of the first and second part of duodenum, extrinsic compression of third part, collapsed bowel distal to crossing of the SMA.

**SMALL BOWEL LIPOMA (Fig 19 and Fig 20)**

Small bowel is the 2\textsuperscript{nd} most common site for lipomas after colon. Most of the lipomas are found in ileum.

They are solitary but may be multiple, submucosal and pedunculated.

**Clinical features:**

- Abdominal pain, constipation, diarrhea, nausea and vomiting, GI hemorrhage (occult) when over 2cm, obstruction (30% patients), lead point of intussusceptions.

**Radiological Features:**

- Submucosal mass with obtuse borders with barium collections in central ulcerations
Differential diagnosis:

Adenomas

Carcinoid tumour

Intestinal polyposis syndromes

**SMALL BOWEL LYMPHOMA (Fig 21)**

The small bowel is the most-often affected intestinal site of lymphoma, representing secondary involvement of non-Hodgkin's lymphoma.

Most often affects the distal ileum, because of its predominance of lymphoid tissue.

**Radiological Features:**

Mucosal fold thickening and effacement, luminal narrowing, aneurysmal bowel wall dilatation, diffuse nodularity, extrinsic compression from mesenteric masses, solitary or multiple filling defects, ulceration, intussusceptions, etc.

**INTESTINAL HELMINTHIASIS (Fig 22)**

Ascariasis is due to infection with the Ascaris lumbricoides adult worm, and typically presents with gastrointestinal or pulmonary symptoms, depending on the stage of development. Gastrointestinal manifestations are due to adult worms. Most common complication of ascariasis is mechanical small bowel obstruction caused by a large number of worms.

**Radiological features:**

Intestinal ascariasis appears as individual worms seen as longitudinal tubular structures on barium enema. If the alimentary tract of the worm is empty, the worm may appear as a filling defect. If its alimentary tract is distended, the worm appears as parallel bands. On transverse sections, the worm appears as a target sign with body wall and a central dot representing its gut volvulus or intussusception.

**CROHNS DISEASE (Fig 24)**

Crohn's disease is also termed regional enteritis, is a chronic inflammatory condition that involves any area of the GI tract from the mouth to the anus.

Etiological factors include infection, altered immunity, and genetic predisposition.
Patients are under 30 years of age and present with abdominal pain, fever, nausea, vomiting, weight loss, and diarrhea (often bloody).

Classic features include:

Aphthoid lesions

Transmural inflammation with granulomas

Ulceration with "cobblestoning"

Fissure and fistula formation

Strictures

Sharply demarcated involved bowel segments with intervening normal areas (skip lesions)

Rectal sparing

The terminal ileum is the most frequently involved segment.

Treatment includes steroids, aminosalicylates, and immunomodulators for acute exacerbations and maintenance of remission.

Surgery is reserved for intractable/fulminant disease, or for complications such as abscesses, strictures, or perforation.

**Radiological features:**

Thick, distorted mucosal folds, Aphthoid lesions, Linear or serpiginous ulcers creating a mucosal "cobblestone" appearance, Funnel-shaped antral stenosis with narrowing of the proximal duodenum.

**GASTROINTESTINAL TUBERCULOSIS (Fig25)**

Causative organism is Mycobacterium tuberculosis. The most commonly affected site in the ileo-caecal junction.

Three types are known

Ulcerative

Hypertrophic

Ulcerohypertrophic
The **Stierlin sign** refers to repeated emptying of the caecum, seen radiographically as barium remaining in the terminal part of the ileum and in the transverse colon.

**INTRAMURAL PNEUMATOSIS (Fig 26)**

Intramural gas also known as pneumatosis intestinalis refers to gas within the wall of the bowel seen in intestinal ischaemia and infarction.

The gas is located submucosally (appears linear) or subserosally (rounded cystic collections).

Gas in the bowel in neonatal period is diagnostic of necrotizing enterocolitis.

They are of 2 types-

Primary pneumatosis intestinalis (15%) is a benign idiopathic condition with multiple thin-walled cysts develop in the submucosa or subserosa of the colon. This primary form is often termed pneumatosis cystoides intestinalis.

The secondary form (85% of cases) is associated with obstructive pulmonary disease, as well as with obstructive and necrotic gastrointestinal disease.

**MECKEL'S DIVERTICULUM (Fig 27)**

Meckel's diverticulum is the failed obliteration of intestinal end of omphalomesenteric duct. It is a true diverticulum (containing all three bowel wall layers) found 40-150 cm proximal to ileocecal valve, within the ileum.

Clinical symptoms include bilious vomiting, abdominal distension, periumbilical pain, and constipation.

Hemorrhage is the most frequent complication in the children, and it is associated with peptic ulceration from heterotopic gastric mucosa located within the diverticulum.

The bleeding is painless. Profuse bleeding is usually self-limited because of physiologic contraction of the splanchnic vessels in response to hypovolemia.

The stool of a patient with an actively bleeding Meckel diverticulum is classically bright red and has a texture that is comparable to "current jelly."

Intestinal obstruction is the second most common complication. It is usually seen in older children and adults.

Small intestinal obstruction occurs due to intussusception; volvulus or internal hernia from persistent attachment of the diverticulum to the umbilicus by the obliterated omphalomesenteric duct, mesodiverticular band, or adhesion.
Luminal obstruction from an inverted diverticulum, diverticulitis, or foreign body impacted in the diverticulum; inclusion of the diverticulum into a hernia.

Neoplastic obstruction; or rarely, the inclusion of a Meckel diverticulum in a true knot that forms between the ileum and sigmoid.

**ULCERATIVE COLITIS (Fig 28 and Fig 29)**

Ulcerative Colitis is an idiopathic chronic inflammatory disease that is characterized by superficial ulcerations, edema, and hyperemia of the colonic mucosa and submucosa.

Patients may present with bloody diarrhea, cramps, abdominal pain, fecal urgency, or tenesmus.

It begins in the rectum and extends proximally in a continuous pattern.

**Radiographic features:**

Confluent, circumferential, shallow ulcerations of the colon, granular mucosa, collar button ulcers, and "thumbprinting."

Complications include stricture formation, toxic megacolon, massive hemorrhage, polyps, a 1% increased risk of colorectal adenocarcinoma per year of disease.

Extraintestinal diseases associated with ulcerative colitis include sacroiliitis, uveitis, iritis, cholangitis, and thromboembolic disease.

Treatment consists of dietary modifications, antidiarrheal agents, 5-aminosalicylic acid agents, and topical/oral mesalamine or corticosteroids.

**TOXIC MEGACOLON (Fig 30)**

Toxic Megacolon is an extreme dilation of the colon in which the affected area of bowel loses all tone and contractility. Bowel sounds, peristalsis, and bowel movements cease.

Clinical features- progressive abdominal distension, significant discomfort, fever, and often leukocytosis.

Causes of toxic megacolon are ulcerative colitis (75% of cases), Crohn's disease, ischemic colitis, Chaga's disease, shigellosis, cholera, typhoid, pseudomembranous colitis, and amebic colitis.

The transverse colon is most often affected and may dilate up to 15cm in diameter.

**Radiographic features:**
Distension of the colon with absent haustra. Edematous and ulcerated mucosa may give rise to pseudopolyps.

**PNEUMATOSIS CYSTOIDES COLI (Fig 31)**

Pneumatosis cystoides coli is a rare condition defined as an abnormal location of gas within the colonic wall. It typically presents with multiple gas-filled cysts in the submucosa and/or subserosa of the colon. The size of the cysts may range from a few millimetres to several centimetres. It is regarded as the colonic variant of pneumatosis cystoides intestinalis.

**SCLERODERMA COLON (Fig 32)**

Scleroderma is a connective tissue disorder causing smooth muscle atrophy and connective tissue replacement.

Clinical manifestations include GERD, esophageal strictures, and impaired peristalsis throughout the GI tract.

Small bowel involvement never precedes skin and esophageal changes.

**Radiographic features:**

Esophageal erosions and strictures.

Luminal dilatation.

"Hidebound appearance" (closely spaced folds, which are normal in thickness).

Pseudodiverticula (prominent sacculations), most commonly seen in the colon.

Pseudo-obstruction from hypomotility with prolonged transit

**PELVIC LIPOMATOSIS (Fig 34)**

Pelvic lipomatosis is the overgrowth of benign, mature, white fat in the extraabdominal compartments of the pelvis along the perirectal and perivesicular spaces.

**Clinical features-**

Urinary tract symptoms (eg, frequency, dysuria, hematuria, urgency, and the sensation of incomplete emptying),

Gastrointestinal tract symptoms (eg, constipation, nausea, vomiting).
Nonspecific symptoms included lower abdominal pain, backache, and flank pain

**Radiographic features:**

Urinary bladder is displaced superiorly and anteriorly and compressed symmetrically, resembling pear shape or an inverted teardrop.

The lower ureters may be pinched medially.

With enough mass effect, obstruction of the ureters, with secondary hydroureter and hydronephrosis, can develop.

If lipomatosis coexists with cystitis glandularis, filling defects from edematous mucosa may also be observed along the bladder wall. If the edematous mucosa is positioned along the ureteral orifice, it too may lead to obstruction of the ureters.
Fig. 1: The gastro-esophageal junction is normally positioned, however a part of the stomach is seen herniating through the esophageal hiatus. Features are suggestive of a Para-esophageal Hernia

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Fig. 2: Thickened gastric rugal folds with a fairly large benign ulcer on the lesser curvature of the stomach. Findings are suggestive of Zollinger-Ellison syndrome

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Fig. 3: Marked thickening of the rugal folds with sparing of the antrum. Findings are suggestive of Menetrier's disease (also referred to as Hypertrophic Gastropathy)

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Fig. 4: Multiple, well defined filling defects are seen within the body of the stomach. Features are suggestive of Gastric Polyps

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Fig. 5: Clubbing, tapering irregularity and fusion of the gastric rugal folds is seen. Features are suggestive of Gastric Adenocarcinoma

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**Fig. 6:** Thickening and rigidity of the stomach with involvement of the pylorus and the duodenum is seen. Features are suggestive of Gastric Lymphoma

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Fig. 7: Solitary large filling defect occupying the entire stomach and which exhibits free movement of the mass on fluoroscopy. Features are suggestive of a Bezoar

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**Fig. 8:** Telescoping of the proximal jejunum into the stomach is consistent with a Jejuno-gastric Intussusception

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**Fig. 9:** Fairly large, smooth outpouching arising from the fundus of the stomach near the gastro-esophageal junction. Features are suggestive of a Gastric Diverticulum

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Fig. 10: Large, dilated stomach with air outlining its walls. Features are suggestive of Emphysematous gastritis

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Fig. 11: Complete, clockwise rotation of the stomach along an axis with the greater curvature and the fundus to the right of the gastro-esophageal junction. Features are suggestive of Gastric volvulus

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Fig. 12: Persistent fleck of barium within the duodenal cap on a double contrast study. Features are suggestive of a Duodenal ulcer

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Fig. 13: The reverse 3 sign or the Epsilon sign as described by Frostberg is suggestive of a Carcinoma of the Pancreas

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Fig. 14: A large, smooth, intraluminal filling defect in the C loop of the duodenum causing effacement of its folds. Histopathology revealed a Villous Adenoma.

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Fig. 15: Multiple, well defined filling defect scattered throughout the fundus and body of the stomach. Features are suggestive of Gardner's syndrome.

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Fig. 16: Multiple, well defined filling defects seen in the proximal duodenum. Features are suggestive of Prolapsing gastric polyps (infact, duodenal bulb polyps are actually gastric polyps that have prolapsed through the pylorus into the duodenum)

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Fig. 17: A large contrast filled, invagination within the lumen of the C-loop of the duodenum. Features are suggestive of an Intraduodenal Diverticulum.

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Fig. 18: An extrinsic linear impression is seen on the third part of the duodenum with proximal dilatation of the duodenal loop. Features are suggestive of a Superior Mesentric Artery Syndrome.

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Fig. 19: Multiple smooth filling defects are seen in the small bowel with fat attenuation of CT Scan. Features are suggestive of Lipomas of the Small Bowel

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Fig. 20: Multiple smooth filling defects are seen in the small bowel with fat attenuation of CT Scan. Features are suggestive of Lipomas of the Small Bowel

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Fig. 21: Effacement and spiking of the mucosal folds of the ileal loops. Histopathology revealed Lymphoma of the Small Bowel.

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Fig. 22: Long, tubular filling defect in the small bowel. Features are suggestive of Helminthiasis

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**Fig. 23:** Widening and rose-thorn like appearance of the mucosal folds of the ileum is seen. Features are suggestive of Intramural Haemorrhage.

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Fig. 24: Stricture in the terminal ileum with a small fistulous track seen arising from it. Features are suggestive of Crohn's Disease

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Fig. 25: The terminal ileum is dilated and deformed with a stricture proximal to the caecum with puckering of the caecum. Features are suggestive of Ileo-Caecal Tuberculosis

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Fig. 26: Air within the walls of the small bowel. Features are suggestive of Intramural Pneumatosis

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Fig. 27: Fairly large, smooth outpouching seen from the ante-mesentric border of the ileo-caecal junction. Features are suggestive of a Meckel's Diverticulum

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**Fig. 28:** Complete loss of haustral pattern with lead pipe rigidity of the entire colon. Features are suggestive of Ulcerative Colitis

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Fig. 31: Multiple sacculations involving the anti-mesentric border of the colon with strictures in the transverse colon. Features are suggestive of Scleroderma.

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Fig. 29: Complete loss of haustral pattern with lead pipe rigidity of the entire colon with multiple filling defects in the descending colon. Features are suggestive of Ulcerative Colitis with pseudopolyps.

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Fig. 30: Marked dilatation of the caecum (> 6cm). Features are suggestive of a Toxic Megacolon

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**Fig. 32:** Evidence of intramural air within the colon with gas filled cysts which do not communicate with the bowel lumen. Features are suggestive of Pneumatosis Cystoides Coli

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Fig. 33: Extravasation of barium into the peritoneal cavity.

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Fig. 34: Marked narrowing and stretching of the rectum with a lucency in the true pelvis. Features are suggestive of Pelvic Lipomatosis.

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Conclusion

**ADVANTAGES OF BMFT:**

1. Easily performed.
2. No discomfort / intubation to the patient unlike in enteroclysis.
3. It is a physiological process. Hence transit time can be assessed.
4. Direct real time visualization of the intestinal mucosa

**DISADVANTAGES OF BMFT:**

1. Overlapping of barium filled bowel loops in the pelvis.
2. Poor distension of bowel loops.
3. Inappropriate timing for visualisation of partial (or) intermittent small bowel obstruction.
4. Operator dependence.
5. Time consuming.
Personal information

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References


