"A CORRELATIVE STUDY OF CLINICAL, BIOCHEMICAL, RADIOLOGICAL, DIAGNOSIS WITH OPERATIVE DIAGNOSIS OF ACUTE ABDOMEN"

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BRANCH – I

Register No: 221711366

DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

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I hereby declare that the dissertation titled **"A CORRELATIVE STUDY OF CLINICAL, BIOCHEMICAL, RADIOLOGICAL, DIAGNOSIS WITH OPERATIVE DIAGNOSIS OF ACUTE ABDOMEN"** is a bonafide and genuine research work carried out by me at Tirunelveli Medical College hospital, Tirunelveli under the guidance of **DR. B.M. PABITHA DEVI M.S.,** Associate Professor, Department of General Surgery, Tirunelveli Medical College, Tirunelveli.

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PRINCIPAL INVESTIGATOR: POST GRADUATE STUDENT
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THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of The Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

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1. The approval is valid for a period of 2 year/s or duration of project whichever is later
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This is to certify that this dissertation work titled **"A CORRELATIVE STUDY OF CLINICAL, BIOCHEMICAL, RADIOLOGICAL, DIAGNOSIS WITH OPERATIVE DIAGNOSIS OF ACUTE ABDOMEN"** of the candidate **Dr. PREETHI KRISHNARAJ** with registration Number **22171166** for the award of **M.S.** Degree in the branch of **GENERAL SURGERY.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **8 Percentage** of plagiarism in the dissertation.

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INTRODUCTION

The term acute abdomen refers to signs and symptoms of abdominal pain and tenderness, a clinical presentation that often requires emergency surgical therapy. This challenging clinical scenario requires a thorough and expeditious workup to determine the need for operative intervention and to initiate appropriate therapy. The acute abdomen may be caused by an infection, inflammation, vascular occlusion, or obstruction. The patient will usually present with sudden onset of abdominal pain with associated nausea or vomiting.

The approach to a patient with an acute abdomen should include a thorough history and physical examination. Many diseases, some of which are not surgical or even intra-abdominal can produce acute abdominal pain and tenderness. Therefore, every attempt should be made to make a correct diagnosis so that the chosen therapy is appropriate.

AIM AND OBJECTIVES

1. This study is aimed to correlate the clinical examination, biochemical investigations and radiological findings with operative diagnosis of acute abdomen.
2. To estimate the sensitivity, specificity and accuracy of biochemical diagnosis in acute abdomen.
3. To estimate the sensitivity, specificity and accuracy of radiological diagnosis in acute abdomen.
4. To know the importance of clinical examination in acute abdomen.

REVIEW OF LITERATURE

HISTORICAL ASPECTS OF ACUTE ABDOMEN :

Hippocrate's observations of manifestations of acute abdominal conditions has been a master piece about life and its meaning.

Among the conditions that relate peculiarly to small intestine is intestinal obstruction recognized as early as eighth century BC. At that time sushrutha recommended that obstruction be treated by incision of intestine, replacement of organs after moistening them with honey and butter and sewing up of the intestine.

Sir Zachary Cope in his book 'The history of acute abdomen' reports that William Ballonius in his consiliorum Medicinalium (Geneva 1934), recorded a case of gangrenous appendicitis.

In 1893 Roentgen described x-rays and diagnostic significance of gas shadows by Schwartz (1911). Schwartz in Vienna was the first to give a description in 1911 of gas distended bowel loops in intestinal obstruction and also noted that fluid levels could be seen in films taken with horizontal tube.

Beniwal Udai Singh et al 2003 concluded that repair of typhoid perforation is better than temporary ileostomy in enteric perforation. Ileostomy and ileotransverse bypass should be considered as the treatment option in patients with unhealthy gut.

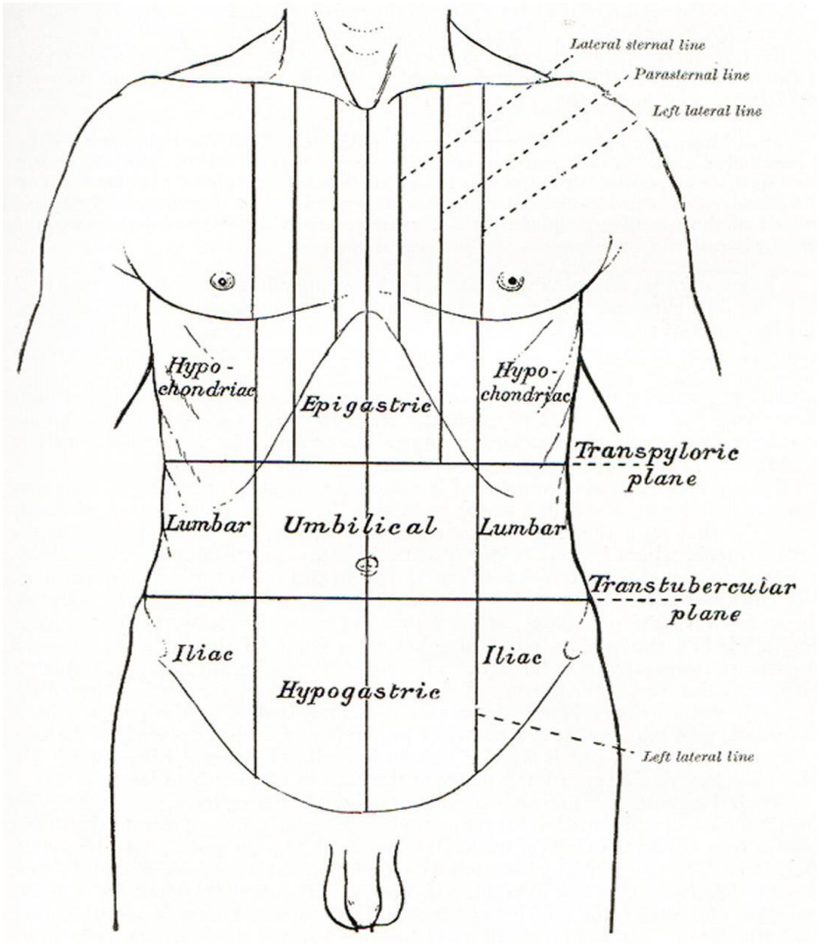
SURGICAL ANATOMY :

Under normal conditions, the peritoneal cavity is a potential space that has a lining consisting of mesothelial layer, the serosal surface and subserosal layer which contains blood vessels and lymphatics. There are two major divisions of peritoneal cavity, the general peritoneal cavity which contain intra-abdominal viscera and lesser peritoneal cavity or lesser omental bursa. The foramen of Winslow is the opening through which these cavities communicate. For descriptive purposes the anterior abdominal wall is divided into four quadrants, with imaginary horizontal and vertical lines crossing at umbilicus. This permits the examiner to indicate that patient complained of pain in right upper quadrant or that tenderness was felt in left lower quadrant etc. The use of quadrants of abdomen for topographic location of pain, tenderness, or masses is helpful for the clinician, but it is important to recall that this external division of abdominal wall has little anatomic basis.

The anatomic relationships of abdominal viscera and visceral attachments have a significant influence upon the localization of signs and symptoms of acute abdominal diseases. The visceral attachments influence the location of spread of blood, purulent material and intra abdominal fluid within peritoneal cavity.

The abdomen is divided into 9 regions by 2 horizontal and 2 vertical lines.

The upper horizontal line or transpyloric runs midway between xiphisternum and umbilicus. Lower horizontal or trans tubercular line lies at the level of tubercles on iliac crests about 2” behind the anterior superior iliac spines. The vertical lines are drawn on either side, through midpoint between anterior superior iliac spine and pubic symphysis.



The different regions are:

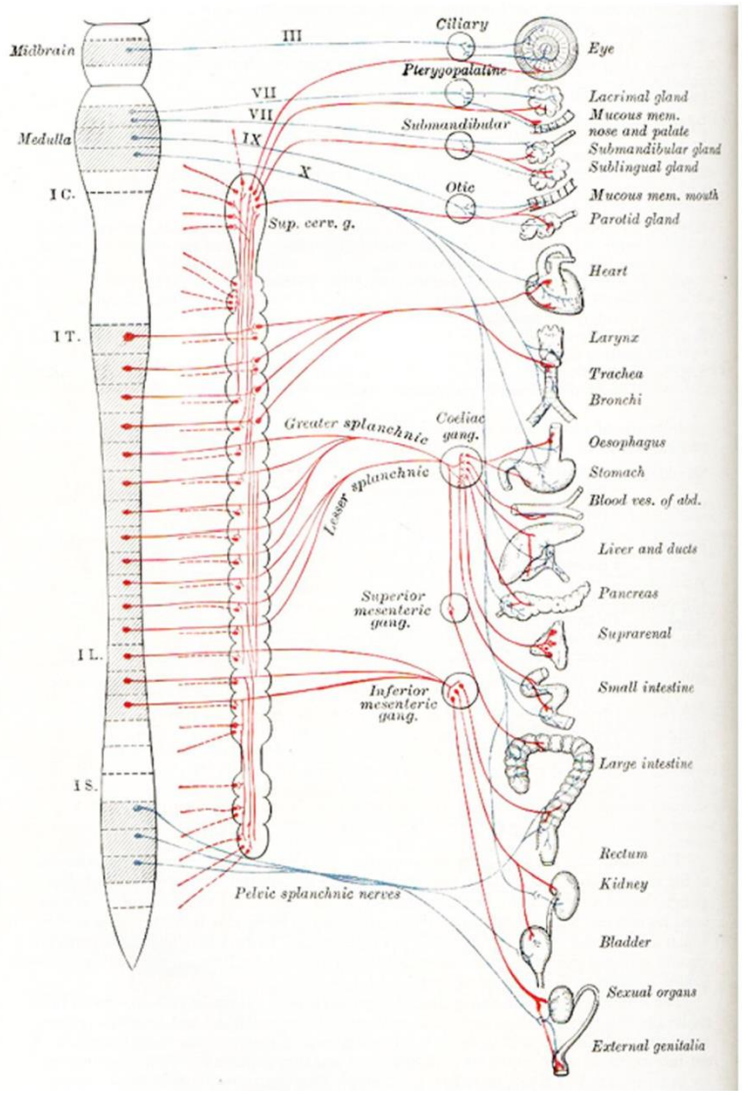
1. Right hypochondrium
2. Epigastric
3. Left hypochondrium
4. Right lumbar
5. Umbilical
6. Left lumbar
7. Right iliac
8. Hypogastrium
9. Left iliac

PATHOPHYSIOLOGY OF ABDOMINAL PAIN:

A surgeon should be able to recognize the physiologic difference between visceral, somatic and referred pain to understand clinical significance of anorexia, nausea and vomiting to correlate interrelationships of symptoms (ie., pain and vomiting).

ABDOMINAL PAIN :

The peritoneal cavity is a potential space, lined by visceral and parietal peritoneum. Each of these two surfaces have distinctive pain pathways and characteristics. A third type of pain, called referred pain, causes sensation at a distant site from the affected organ. This type of pain sometimes helps, but often hinders and confuses the diagnosis.



VISCERAL PAIN :

The visceral peritoneum receives its pain sensation through the splanchnic nerves of the autonomic nervous system. This sensation is poorly localized and may be felt in the abdomen at some distance from the affected organ. It is caused by stretching or ischemia but not by cutting or burning. Visceral afferent fibre from the capsule of the liver, Spleen, central portion of the

diaphragm and crura reach the central nervous system through the sympathetic sensory fibres in the phrenic nerves. Fibres from the gall bladder, stomach, small bowel and pancreas travel to the celiac plexus and via the splanchnic nerves to the sixth through the ninth thoracic segments of spinal cord. Afferent fibres from colon, appendix and pelvic organs reach tenth and eleventh thoracic segments via the mesenteric plexus.

Visceral pain arising from the foregut structures, such as the stomach, pancreas and duodenum is perceived in the epigastrium. Pain originating from the midgut structures, such as the small bowel at the periumbilical area. Whereas pain originating from hindgut structure, the left colon is perceived in the hypogastrium.

The location of this pain also depends on the embryonic origin of the viscus, for example, testicular pain is associated with renal colic. Despite complaints of severe pain, the patient may have a soft abdomen without significant tenderness, as in the case of an early small bowel obstruction without associated peritoneal inflammation. Severe visceral pain causes autonomic reflexes such as sweating, tachycardia, bradycardia, hypotension, cutaneous hyperaesthesia and involuntary contraction of the abdominal musculature.

PARIETAL PAIN :

The parietal peritoneum and abdominal wall receive somatic pain fibres from the spinal nerves. Sensation from these fibres is sharp, well localized

and severe and is usually caused by inflammation of the peritoneum. Movement aggravates this pain involvement, usually due to an underlying visceral inflammation. The vagus nerves do not carry pain sensation from the abdominal cavity, even though they contain many afferent fibres. Pain from the oesophagus and the lower portion of the bladder and rectum is conducted by the vagus nerve and sacral plexus respectively.

REFERRED PAIN :

Somatic and visceral nerves often synapse in the spinal cord. Stimuli coarsing along the visceral afferents stimulate the synapsing somatic nerves and cause sensation to be perceived in areas remote from the stimulated viscus. Common examples are the referred pain from the diaphragm to the supra clavicular area by the third cervical nerve, Gallbadder pain perceived at the tip of the Scapula by the seven spinal nerve etc.

CAUSES OF ATRAUMATIC ACUTE ABDOMEN:

1) **INFLAMMATION AND INFECTION:**

- Acute appendicitis
- Acute cholecystitis
- Acute diverticulitis

- Acute pancreatitis

- Salphingitis, Septic abortion, Mesenteric adenitis, Primary peritonitis, Crohn's disease, Meckel's diverticulitis, Pyelonephritis and cystitis.

2) PERFORATION:

- Gastric ulcer

- Duodenal ulcer

- Carcinoma of the colon

- Diverticular disease

- Acuteappendicitis

- Perforation of a segment of strangulated bowel, Acute cholecystitis, Crohn's disease, Ulcerative colitis, Lymphoma Foreign body perforation, Perforation of the oesophagus (Boerhaave's syndrome), Perforation of the urinary bladder.

3) OBSTRUCTION:

- Small bowel : Congenital bands/atresia
    - Hernia
    - Adhesions from previous surgery
    - Intussusception
    - Tumour
- Largebowel
    - Malrotation of thegut.
    - Tumor
    - Volvulus
    - Inflammatory Stricture

4) **INFARCTION:**

- Arterial thrombosis or embolus
- Venous thrombosis
- Torsion of testis/ ovary,
- Dissecting aortic aneurysm

5) HAEMORRHAGE:

- Ruptured abdominal aortic aneurysm
- Ruptured ectopic pregnancy
- Aneurysm of mesenteric vessels,
- Dissecting aneurysm of the aorta, Ruptured ovarian cyst, Ruptured ovarian cyst, Ovulatory bleed, Endometriosis, Spontaneous rupture of liver tumour, Rectus sheath haematoma.

PATHOLOGICAL PROCESSES CAUSING THE ACUTE ABDOMEN: INFLAMMATION AND INFECTION :

These conditions are usually characterized by a febrile illness with localized signs of peritonitis. The origin of inflammation can frequently be determined from the history and onset of the pain and by the site of maximum tenderness. The common intra abdominal inflammatory conditions has been listed above

PERFORATION:

Perforation of an abdominal viscus usually results in the rapid onset of severe abdominal pain. The viscus which has perforated can often be determined from a history of previous abdominal pain. In the early stages, the site of maximum tenderness may also indicate the organ which has perforated. However, the usual end point of generalized peritonitis is a rigid abdomen in which selective tenderness can no longer be elicited. Commonest aetiologies include perforated peptic ulcer of either the stomach or duodenum, diverticular disease, typhoid perforation, perforated carcinoma of colon.

OBSTRUCTION:

Obstruction of any hollow viscus within the abdominal cavity can cause acute abdominal pain. Obstruction of the small or large bowel usually cause intermittent colicky pain, while obstruction of the ureter or gall bladder presents with continuous pain which is punctuated by acute exacerbations. Unless a complication such as strangulation or perforation has occurred, there will not, in most instances,

be signs of peritonitis. The obstructed viscus can often be determined from the history of the pain.

INFARCTION :

MESENTERIC ISCHAEMIA

Mesenteric ischaemia can be of acute (90%) or chronic type (10%). In acute type the causes are arterial embolism, arterial thrombosis, non-occlusive form or venous occlusion.

Acute occlusions of the superior mesenteric artery due to thrombosis or embolisation are responsible for approximately 60%–70% of cases of acute bowel ischaemia, whereas non-occlusive conditions account for approximately 20%–30% of cases and mesenteric venous thromboses account for 5%–10% of the total.

Embolic and thrombotic acute mesenteric ischaemia (EAMI/TAMI)

Arterial inflow occlusion most commonly results from thromboembolism, where the embolus originates from the left atrium as a consequence of atrial fibrillation. Emboli preferentially affect SMA because of its small take-off angle compared with those of the coeliac and IMA. While thrombi and large emboli may occlude the proximal SMA and ostia of major mesenteric vessels resulting in extensive small bowel and colon ischaemia, smaller emboli may lodge in the distal portions of the vessel and cause smaller regions of segmental ischaemia. Acute arterial thrombi and emboli may appear as obvious low-attenuation filling defects in the luminal vessels

There are various causes of occlusion of the mesenteric arteries besides embolism. In younger patients thrombotic microangiopathies and antiphospholipid antibody syndrome are also frequent causes of occlusion of the mesenteric arteries.

The development of intestinal ischaemia from an arterially obstructing lesion depends upon the location of the obstruction, the patient's collateral vasculature, acuity and degree of the obstruction. As said before, the presence of collateral arcades allows bidirectional flow, which can bypass obstructing lesions. In the presence of obstructions involving all three major arteries (coeliac, SMA and IMA), the phrenic, lumbar and pelvic collateral arteries may dilate to provide accessory visceral blood flow. However, if the lesion is distal to the point of collateral flow, the collateral supply is ineffective and ischaemia is more likely to ensue.

Clinically, EAMI is characterized by a sudden onset of abdominal pain in patients over 70 years old and a history of atrial fibrillation. In the early course of the disease, it can be characterized by an initial discrepancy between the severity of abdominal pain and minimal findings on physical examination. Patients can also present with symptoms of nausea, vomiting and initial forced evacuation. The location of pain varies, but as ischaemia progresses to infarction, it becomes diffuse and signs of

peritoneal irritation appear. The development of transmural infarction may also be signalled by fever, bloody diarrhoea and shock.

Thrombotic arterial mesenteric ischaemia (TAMI) has a more indolent course. TAMI patients undergo gradual progression of arterial occlusion; therefore, many report symptoms of mesenteric angina (postprandial abdominal pain lasting up to 3 hours and nausea), which results in **"food fear"**, early satiety and weight loss. In the acute setting, however, the clinical symptoms are similar to those found in patients with EAMI

The main risk factors for TAMI are atherosclerotic disease and dyslipidaemia.
There may be a history of other vascular events and previous vascular surgery.

Venous acute mesenteric ischaemia (VAMI)

Mesenteric venous thrombosis may be caused by infiltrative, neoplastic or inflammatory/infectious conditions VAMI appears in younger patients, over 40, sometimes with several days of mild symptoms. Although occasionally idiopathic, up to 50% of patients have an identifiable risk factor, such as previous deep venous thrombosis or pulmonary embolism. Other risk factors include a hypercoagulability state such as Leiden factor V mutation, oral contraceptive use, cirrhosis and advanced malignancy.

As in arterial occlusion, isolated proximal mesenteric venous thromboses usually do not lead to severe bowel ischaemia because of the extended collateral network between the mesenteric and systemic veins. In contrast, thrombosis of very distal mesenteric veins usually leads to severe haemorrhagic infarction of the bowel wall.

The onset of VAMI is characterised by subacute abdominal pain that may develop over a period of up to 2 weeks. VAMI is not usually associated with postprandial syndrome, although bloating, abdominal distension, fever and occult blood in stools may be present

Non-occlusive mesenteric ischaemia (NOMI)

In the setting of non-occlusive causes such as septic, haemorrhagic or cardiogenic shock, a profound drop of systemic blood pressure results in a reflexive mesenteric arterial vasoconstriction with diversion of blood flow to the brain and heart. As a consequence, intestinal perfusion will decrease dramatically and nonocclusive bowel ischaemia may develop.

Risk factors for NOMI include age over 50, history of acute myocardial infarction, congestive heart failure, aortic insufficiency, cardiopulmonary bypass, kidney or

liver disease or major abdominal surgery. Notably, many patients with NOMI may have none of these factors.

The diagnosis of NOMI is the most challenging, first because it is often silent as it occurs in patients that are critically ill and often ventilated and second because CT findings overlap with those of other forms of bowel disease such as infectious and inflammatory enteritis and colitis.

The diagnosis should be suspected in patients with mesenteric hypoperfusion secondary to circulatory shock or vasoactive drugs when there is a significant unexpected deterioration in their clinical course. Acute or insidious pain (without defecation), bloating, abdominal distension and the presence of occult blood in the stools are all consistent with NOMI in a critically ill patient.

There has been an overall decrease in the incidence of this syndrome with improved management of haemodynamic instability.

Chronic mesenteric ischemia is a rare condition that was first described in 1918 as **“abdominal angina”** by Goodman.1 Its onset is gradual and it is often diagnosed late in its course. Treatment of the underlying lesion is necessary to prevent the development of acute mesenteric ischemia, which may result in bowel infarction

and death. Shaw performed the first successful open repair for chronic mesenteric ischemia in 1958.2 Since then, surgical repair has been the standard treatment for chronic mesenteric ischemia. However, angioplasty and stenting of the mesenteric arteries for treatment of chronic mesenteric ischemia has gained popularity because of its effectiveness and relatively low rates of morbidity and mortality compared with open surgical repair.

PATHOGENESIS AND CLINICAL PRESENTATION

The etiology of chronic mesenteric ischemia is often multifactorial. The most common cause is atherosclerosis involving the proximal portions of the celiac, superior mesenteric, or inferior mesenteric artery. Less common etiologies include dissection, vasculitis, fibromuscular dysplasia, radiation, and cocaine abuse. Factors that predispose patients to atherosclerosis are associated with increased risk for chronic mesenteric ischemia. These include smoking, hypertension, diabetes mellitus, and hypercholesterolemia. Chronic symptoms are caused by the gradual decrease in blood flow to the intestines. Because total blood flow to the intestine can vary from 25% when fasting to 35% after eating, symptoms are more prevalent after eating. The normal circulation to the bowel includes blood supply from the celiac artery, the superior mesenteric artery, and the inferior mesenteric artery. There is, however, the potential for collateralization between the vascular territories of these vessels.5 The abundant mesenteric blood supply and slow progression of

atherosclerosis allows these collateral pathways to develop. Because of this collateral circulation within the mesenteric vasculature, patients may not experience symptoms until two or three major mesenteric vessels are involved. Classic symptoms of chronic mesenteric ischemia include postprandial abdominal pain associated with significant weight loss, food fear, nausea, vomiting, or diarrhea. The abdominal pain classically starts 15 to 30 minutes after a meal and typically lasts for 30 minutes. As the obstructive process progresses, chronic, dull abdominal pain ensues. Chronic mesenteric ischemia generally presents in patients older than 60 years of age and is 3 times more frequent in women.

TREATMENT

After the diagnosis of chronic mesenteric ischemia is made, patients should undergo definitive treatment because of the risk of continued weight loss, acute infarction, perforation, sepsis, and death.

Medical treatment is usually reserved for patients who are not healthy enough to be treated, either surgically or endovascularly. This would consist of long-term anticoagulation, such as warfarin. In addition, some patients may find short-term relief with nitrate therapy; however, this is not curative.

Open surgical repair includes transaortic endarterectomy, direct reimplantation on the aorta, and antegrade or retrograde bypass grafting. Several series evaluating surgical repair for chronic mesenteric ischemia have reported high technical success rates and symptom improvement in 90 to 100% of patients. However, they have also demonstrated that open surgical repair has also been associated with significant morbidity (5 to 30%) and mortality (5 to 12%). This is at least in part related to the weight loss and malnutrition, including low albumin, in this patient group, which are all predictors of increased morbidity and mortality after major surgery. Symptom recurrence rates following open surgical repair for chronic mesenteric ischemia ranged between 9% and 35% in these studies. Recurrent symptoms and thus the reintervention rate were higher in patients treated endovascularly when compared with those treated with open surgery.

HAEMORRHAGE :

Blood within the peritoneal cavity produces the symptoms and signs of an 'acute abdomen'. This may initially be localized to the site of the bleed, but rapidly becomes more generalized. The history and original site of abdominal pain should give some indication of the source of the haemorrhage.

DIAGNOSIS OF ACUTE ABDOMEN:

Diagnosis of acute abdomen includes :

1. History taking
2. General physical examination
3. Local examination of abdomen
4. Investigations

HISTORY

The common symptoms which a patient with acute abdomen complaints are :

1. Abdominal pain
2. Vomiting
3. Distension of abdomen
4. Constipation

ABDOMINAL PAIN:

Pain is usually the predominating and presenting feature of acute abdomen. In order to elucidate its cause, the location, mode of onset, progression and character of pain must be determined. The patient should be asked about the exact site of pain, onset, duration, nature, relation to posture, food, bowels and micturition. Always enquire about radiation of pain.

Absence of pain at the time of examination, does not exclude an acute condition. For example, remission after perforation of appendix or three to four hours after perforation of peptic ulcer, are well recognized. Similarly in intestinal obstruction there may be a quiet phase during which colic is relieved, or relief may imply that there is paralytic ileus.

a) Location of pain:

Because of complex dual visceral and parietal sensory network subserving the abdominal area, pain is mediated primarily by afferent C fibres located in walls of hallow viscera and in capsule of solid viscera. It is elicited either by distention, inflammation or ischemia, stimulating receptor neurons or by direct involvement of nerve as in malignancy. The centrally perceived pain will be slow in onset, dull, poorly localized and protracted. Increased wall tension or forceful smooth muscle contraction leads to diffuse deep seated pain felt in mid-epigastrium, peri umbilical and lower abdomen, but most often felt in midline because of bilateral sensory supply to spinal cord.

By contrast, parietal pain is mediated by both C and A nerve fibre, the latter being responsible for more acute, sharper, better localized pain sensation. Direct irritation of somatically innervated parietal peritoneum by pus, bile, Urine or GI secretions is associated with more exact localization of pain (from T6-LI). Parietal pain is more easily localized because somatic afferent fibres are directed to only one side of nervous system.

Abdominal pain may be referred or may shift to sites far away from primarily affected organs.

The term referred pain denotes noxious sensation perceived at a distant site from a strong primary stimulus. For example pain felt in shoulder by irritation of diaphragm.

Spreading or shifting pain parallels course of underlying condition. For example pain in appendicitis shifting from epigastric region to right iliac fossa.

b) Mode of onset and progression of pain:

The mode of onset reflects nature and severity of inciting process. Onset may be explosive (within few seconds) progressive (with in 1 to 2 hours) or gradual over hours. Excruciating generalized pain suggests an intra abdominal catastrophe such as perforated viscus or rupture of aneurysm, ectopic pregnancy or abscess. Accompanying systemic signs like tachycardia, tachypnoea, shock underscore the need for prompt

resuscitation and laparotomy.

A less dramatic clinical picture is steady mild pain, centred in a well defined area in 1 to 2 hours. Typical of acute cholecystitis, acute pancreatitis, strangulated bowel, mesenteric infarction, proximal small bowel obstruction etc.

Some patients may have slight, at times only abdominal discomfort. Eventually pain and abdominal findings become more pronounced and well localized. This reflects slowly developing condition or body's defensive effort to cord on and off a acute process. This occurs in acute appendicitis, incarcerated hernia, small bowel and large bowel obstruction, walled off visceral perforations etc.

c) Character of pain:

Nature, severity and periodicity of pain provide clues to underlying cause. Steady pain is most common. Sharp superficial constant pain due to peritoneal irritations typical of perforated ulcer and ruptured appendix.

The gripping, mounting pain of small bowel obstruction is usually intermittent, vague, deep seated and cresendo first but soon becomes sharper, unremitting and better localized.

Pain is appropriately referred to as colic, if there is pain free intervals that reflect intermittent smooth muscle contractions, as in ureteric and biliary colic.

The 'aching discomfort' of ulcer pain and 'stabbing breath taking pain' of acute pancreatitis and mesentric infarction and searing pain of ruptured aortic aneurysm remain apt descriptions.

Movements tends to aggravate the pain in peritonitis but conditions associated with severe colic at times relieves the patient of pain.

Previous ingestion of drug's sedatives and any injection may modify or minimize signs to some extent.

NAUSEA :

This symptom of nausea is common in any acute illness. In its milder form it is merely a distaste for food. This symptom like that of vomiting is of no specific diagnostic value but usually indicate some derangement in gastro intestinal function. Usually found in acute illness with distention of abdomen or local visceral dilatation. Extreme nausea usually occurs with severe blood loss.

2) Vomiting:

Pain in acute abdomen usually precedes vomiting. In medical conditions reverse is true. When sufficiently stimulated by secondary afferent visceral fibres, the medullary vomiting centres activate efferent fibres to induce reflex vomiting.

The details to be extracted are, its relation to onset of pain, character of vomitus, frequency and volume of vomitus, contents and presence or absence of nausea.

In patients with biliary colic or upper small bowel obstruction, vomiting occurs soon after the onset of pain. Distal small bowel obstruction may be manifested by cramping pain 2 to 4 hours before vomiting while vomiting is a late complication of large bowel obstruction.

Vomiting is 'reflex', occurs in early stages of acute abdomen associated with severe pain abdomen, distortion of hollow viscus, increased tension in mesenteries, torsion with local or generalized inflammation of peritoneal or retro peritoneal tissues. Toxic vomiting occurs with development of gross infective conditions and uremia.

In acute appendicitis initial vomiting is reflex, later due to ileus and rarely to organic obstruction. Both nausea and vomiting are characteristic complaints of pre or post ileal appendicitis. In intestinal obstruction vomiting first is reflex, later due to obstruction, later due to strangulation .In some patients

vomiting is surprisingly absent , for example there may be signs and symptoms of peritonitis or intestinal obstruction, but no history of vomiting. In such conditions a nasogastric aspiration of large quantities of foul fluid converts a doubtful clinical picture to certainty.

A number of serious acute abdominal condition are not associated with vomiting. Vomiting is not important symptom of perforated ulcer although nausea and retching may occur after perforation, vomiting is infrequent. Vomiting relieves pain in case of peptic ulcer but in colics it relieves pain temporarily so that it reappears immediately. Vomiting may not occur in cases of large bowel obstruction or acute appendicitis. The latter problem is associated with loss of appetite and frequent nausea, even though emesis has not occurred. Vomiting may be infrequent or absent in intra abdominal haemorrhage.

3) DISTENTION OF ABDOMEN:

This is a complaint of patient with peritonitis (especially late stages) and of intestinal obstruction. This is contributed by collection of peritoneal fluid in chemical peritonitis, pus, GI contents in perforations, blood in haemoperitoneum and gaseous in paralytic ileus.

4) BOWEL HABITS:

Evaluation of status of patient must include appraisal of bowel function. While diarrhoea may suggest the presence of gastro enteritis, in a patient with abdominal pain, appendicitis may be present. In some patients, obstipation with abdominal distention supports the diagnosis of large bowel obstruction. However, in patients with pancreatitis, cholecystitis and inflammatory problems of abdominal viscera, ileus may develop with distention and failure to pass flatus and stools. The character of stools must be noted. The presence of blood, dark or fresh, tarry stools, and other abnormalities should be sought for, as well as presence of diarrhoea. Enquiry concerning tenesmus and decreased caliber of stools should be made in patients whom a lesion of large bowel is suspected. In intussusception red currant jelly stools is of significance.

5) ASSOCIATION WITH FOOD INTAKE:

History of onset of acute abdomen after the ingestion of meal is recognized in some cases of perforated peptic ulcer. The character of meal is also of help, as in cases of acute pancreatic, patient will give history of having had rich fatty food or alcohol.

Occasionally ingestion of fish bones or indigestible fruits or vegetables may be causative for perforation and intestinal obstruction.

6) MENSTRUAL HISTORY:

The relationship of menstruation to acute abdominal symptoms may be of diagnostic importance. It is crucial in diagnosis of ectopic pregnancy, mittelschemerz and endometrosis. If amenorrhoea is present, possibility of pregnancy will necessarily influence diagnosis and management. Irregular vaginal bleeding may occur with any acute pelvic condition or abortions. History of amenorrhoea and shock will give the suspicion of ruptured ectopic gestation.

7) DRUG HISTORY:

Important not only in peri operative management but also because it may offer a diagnostic value. Anticoagulants have been implicated in retroperitoneal and intramural duodenal and jejunal hematoma, oral contraception information of benign hepatic adenomas and mesenteric arterial infarction. Corticosteroids may mask signs even in advanced peritonitis.

GENERAL PHYSICAL EXAMINATION:

The surgeon should develop a systematic approach to the examination of acute abdomen. The customary vital signs- Temperature, Pulse, Respiratory rate and Blood pressure are essential in appraisal of patient who complains of abdominal distress.

GENERAL APPEARANCE :

The appearance of the patient will frequently give some general clues as to the severity of the illness, look for toxicity, colour of skin, cyanosis of lips or mucosa, distention of veins in neck or trunk, respiratory rate and effort, position of limb etc.

The position of patients may provide a clue. The patients with renal or biliary colic is restless and been described as 'climbing the wall with pain'. In contrast, patients with peritoneal irritation, prefers to be immobile because movement increases discomfort. Small bowel obstruction produces intermittent cramping pain and patient will 'Double up' at intervals. Similarly a child with intussusception will draw up the legs and cry from time to time with pain. Flexion of right hip is adopted in case of inflamed retrocaecal appendix.

SHOCK :

Always signs of shock should be sought for. It suggests an underlying grave disorder. Development of shock at an early stage suggests internal haemorrhage or gangrene of bowel. In intestinal obstruction the patients will go on to shock by fluid and electrolytic imbalance due to vomiting or later by paralytic ileus or gangrenous bowel. In late cases septic shock may supervene because of bacterial peritonitis.

DEHYDRATION :

Sunken eyes, loss of skin turgour, dry tongue, decreased urinary output suggests dehydration

COLOUR :

Extreme pallor is noted in acute conditions with severe pain due to primary shock. Extreme pallor with shallow respiration and abdominal crisis is suggestive of intra abdominal haemorrhage.

A flushed appearance is frequently associated with pyrexia in a inflammatory condition. A cyanotic or greyish complexion has been regarded as suggestive of pancreatic inflammation.

PULSE :

The pulse rate by itself has no diagnostic value, but serial observations help in assessing the progression of conditions or circulatory collapse.

Moderate tachycardia with or without temperature denotes some inflammatory pathology. Extreme tachycardia of low volume with cold clammy skin, perspiration denotes shock or hypovolemia or septicemia.

BLOOD PRESSURE :

The recording of blood pressure is of great value in general assessment of condition of patient, especially in cases of shock and haemorrhage.

Serial recordings of blood pressure is utmost importance in management of any patient.

RESPIRATION :

Both respiratory rate and character of respiratory effort should be observed. Patients with severe abdominal pain associated with perforation of peptic ulcer or with acute pancreatitis often have shallow respiratory exertions. Marked abdominal distention also interferes with respiration.

A rapid respiratory rate in advanced peritoneal disease may be a sign of increased toxemia or a pulmonary complication.

TEMPERATURE :

Low grade fever is common in inflammatory conditions such as diverticulitis, acute cholecystitis and appendicitis.

High fever with lower abdominal tenderness in young women without systemic illness suggest acute salphigitis.

Disorientation or extreme lethargy combined with very high fever (> 30^0C) or swinging temperature or with chills and rigors signifies impending septic shock, most often due to advanced peritonitis, acute cholangitis or

pyelonephritis.

After intraperitoneal haemorrhage, the circulatory collapse may lead to subnormal temperature first, later high temperature because of absorption of blood.

ABDOMINAL EXAMINATION:

Inspection :

The abdomen should be carefully inspected before palpation. Surgical scar noted on the abdomen may have a bearing on acute abdomen. A tensely distended abdomen with old surgical scar indicate both the presence and cause (adhesions) of small bowel obstruction. A previous operation may carry a risk or specific complication, for example stomal ulcer may follow gastro-jejunostomy for peptic ulcer.

An operation may have already be done for a condition known to recur, like perforated peptic ulcer, volvulus of gut etc.

Discoloration or bruising of abdominal wall may be important. Echymotic discoloration of umbilicus or flanks are after seen in acute pancreatitis, circum umbilical echymosis may occur in intra abdominal haemorrhage, particularly in ruptured ectopic gestation.

Distention of abdomen, evident on inspection may be generalized in

intestinal obstruction, peritonitis and ascitis. Local or symmetrical distention may indicate enlargement of a particular viscera. Emptyness in one part may be recognized as intussusception. A scaphoid contracted abdomen is seen in perforated peptic ulcer. Visible peristalsis suggests obstruction. The direction and pattern of peristaltic waves may occasionally be of significance.

Sluggish or no respiratory movement of abdominal wall indicates wide spread irritation of peritoneum as occurs in diffuse peritonitis or haemorrhage into peritoneal cavity. similarly localized limitation of respiratory movement occurs in localized inflammation of peritoneum of underlying organs. Eg: Acute appendicitis, Acute cholecystitis.

The potential hernial orifices should be examined at umbilicus, inguinal canals and femoral region. A visible swelling at a hernial site in lying down position is always significant, as it is usually an obstructed or strangulated hernia.

In males, testis and scrotum should be inspected for any enlargement, displacement etc. Displacement may indicate maldescent or it may be due to cremasteric muscle spasm which may occur in intra peritoneal

inflammation, absence testis may be of importance if the organ is intra abdominal, as it may be the cause of acute symptoms.

PALPATION :

When inspection is complete, the patient is asked to point with his finger where the pain has started and the site of persisting pain, to get an objective localization of pain and also the site of maximum tenderness. The patient is asked to cough and the site of maximum pain he gets is noted.

Palpation is performed with patient resting in a comfortable supine position and hyperaesthesia may be demonstrated in abdominal wall disorders or localized peritonitis, but is more prominent in herpes zoster, spinal root compression and other neuromuscular disorders. This can be elicited by gently picking up a fold of skin or scratching the abdominal wall with finger. Ex :- presence of hyperaesthesia in Sherren's triangle in gangrenous appendicitis and an area between $9^{th}$and 11^{th}ribs posteriorly on right side is known as Boa's sign suggestive of acute cholecystitis.

Tenderness that can localize peritoneal inflammation is perhaps the most important finding in patients with a acute abdomen. Beginning away from area of cough tenderness and gradually advancing towards it.

Rebound tenderness is an important sign of peritoneal irritation and signifies that peritoneal surfaces are involved by an inflammatory process. It is detected by sudden removal of palpating hand from abdomen and should be done without warnings, so that it is not expected by patient.

The most important sign elicited is GUARDING. Guarding is assessed by placing hand over abdominal muscles and depressing them gently, while the patient will voluntarily contract the abdominal muscles. If there is involuntary spasm, which remains taut and rigid (board like) throughout the respiration, it is called rigidity. Unlike peritonitis, renal colic induce spasm confined to ipsilateral rectus muscle.

ABDOMINAL MASSES :

Are usually detected by deep palpation. Deeper masses may be adherent to posterior or lateral abdominal wall and are often partially walled of by omentum and small bowel. As a result, their borders are ill defined and only dull pain elicited.

SPECIFIC SIGNS OF DIAGNOSTIC IMPORTANCE:

McBURNEY'S SIGN : Maximum tenderness felt over Mc Burneys point

ROVSING SIGN : When pressure is applied over left iliac fossa, pain is felt in right iliac fossa, due to shift of coils of intestines to right impinging on inflamed appendix.

COPE'S PSOAS TEST : When inflamed focus lies on psoas muscle, to relieve pain patient often flexes the corresponding thigh. Hyperextension then causes pain. Positive in cases of psoas **abscess arising from perinephric abscess, perforated Crohns enteritis and acute appendicitis (retrocaecal).**

OBTURATOR TEST :

On flexing the hip joint and externally or interally rotating the thigh, obtutator internus will stretch and causes pain. Positive in pelvic appendicitis, strangulated obturator hernia.

MURPHY'S SIGN :

The patient is asked to take deep breath as the examiner gently palpates the right coastal margin on the lateral border of the right rectus (gall bladder point). With the descent of diaphragm an acutely inflamed gall bladder comes in contact with fingers, the patient will wince with a catch in the breath.

BALANCE'S SIGN :

Persistent dullness on the left side of the abdomen due to early coagulation of splenic blood and shifting dullness on the right side. Seen in splenic rupture or injury.

KEHR'S SIGN :

Referred pain to the left shoulder due to irritation of left dome of diaphragm occurs in splenic injuries or rupture spleen.

BALDWING'S TEST :

A hand is placed over the flank of the patient, the patient is asked to rise the right lower limb off the bed, keeping knee extended. The patient will immediately complain of pain in case of retro caecal appendix.

GREY TURNER'S SIGN :

Discoloration in the left flank seen in acute haemorrhagic pancreatitis.

CULLEN'S SIGN :

Discoloration around umbilicus seen in late cases of acute pancreatitis.

THE SIGN DE DANCE :

A feeling of emptyness in right iliac fossa in intussusception.

PERCUSSION :

Percussion of the abdomen should begin in the quadrant free of pain and should be performed lightly so as to avoid eliciting pain at the onset of the examination. Percussion is performed for the detection of free peritoneal fluid, bowel distention, mass and obliteration of liver dullness associated with a perforated viscus.

Although percussion is of vital importance, many a times it is not possible to perform percussion in an acutely rigid and tender abdomen.

AUSCULTATION :

Auscultation for audible peristalsis in an important step in examination of acute abdomen. It is necessary to listen to two or three minutes to establish absence of peristalsis.

Auscultation of the abdomen should include all four quadrants, with special attention given to the frequency and pitch of bowel sounds and rushes of gas audible to the examiner that correlate to the facial expression of pain by the patient.

Absence of bowel sounds indicates diffuse peritonitis and paralytic ileus occasional tinkles suggest air and fluid in the intestine and the presence of a

severe paralytic ileus. Peristaltic rushes, synchronous with colic are heard in a mild small bowel obstruction and in early pancreatitis. They differ from the high pitched hyper peristaltic sounds unrelated to the crampy pain of gastro enteritis, dysentery and fulminent ulcerative colitis.

Early mesenteric arterial occlusion is associated with increased activity and bowel sounds are loud and active within few hours. The ischemic bowel loses its peristaltic function.

HERNIAL ORIFICES

Hernial orifices in both sexes should be examined. Incarcerated inguinal hernia can cause intestinal obstruction.

EXTERNAL GENITALIA :

External genitalia should be examined.

PER VAGINAL EXAMINATION :

Acute abdomen is incorrectly diagnosed more often in women than in men, particularly in young age group. Hence pelvic examination by per speculum, digital and bimanual examination is a must.

PER RECTAL EXAMINATION :

No examination of an acute abdominal case is complete without the digital

examination of the rectum.

1) TENDERNESS:

The right wall may be tender in pelvic type appendicitis, which may not show tenderness or rigidity of the anterior abdominal wall. Tenderness is often elicited in the rectovesical pouch in perforated peptic ulcer.

2) BULGING:

The bulging of the anterior wall of the rectum with tenderness is significant of a pelvic abscess.

3) ANY GROWTH:

In lower third of rectum causing obstruction. Though rare, is usually missed if rectal examination is not done routinely.

In intussusception, after rectal examination has been finished one will find the gloved finger, to be smeared with mucus blood (red-currant jelly) but not faecal odour. In cases of obstruction it is important to note whether the faeces is present (impaction) or whether rectum in widely dilated and empty (ballooning) as in colonic obstruction, higher up.

Examination of the other systems :

RESPIRATORY SYSTEM :

Examination of chest is essential as preoperative assessment and to

differentiate between chest disease and acute abdomen. Acute pneumococcal pneumonia may mimic acute abdomen quite often.

CARDIOVASCULAR SYSTEM :

Sometimes we have to rule out acute abdomen from myocardial infarction.

INVESTIGATIONS :

Blood investigations :

Haemoglobin, hematocrit and white cell counts taken on admission are highly informative. Only a rising or marked leucocytosis indicates inflammatory conditions. Its role in the diagnosis of acute appendicitis is considered very high. Besides this, it is almost always present in acute cholecystitis, acute pancreatitis etc. Acute intestinal obstruction when complicated by strangulation may show presence of leucocytosis.

Serum electrolytes, sugar, urea and creatinine are important, especially if hypovolemia is expected. Diabetic crises may mimic an acute abdomen. Uremia may present itself with persistent vomiting accompanied by increasing distention of the abdomen to make this condition confused with acute intestinal obstruction.

A raised serum amylase level corroborates a clinical diagnosis of acute pancreatitis. Moderately elevated values must be interpreted with caution,

since abnormal levels frequently accompany strangulated or ischemic bowel, twisted ovarian cyst or perforated ulcer. Moreover a normal or even low amylase value may be seen in haemorrhagic pancreatitis or pseudocyst.

RADIOLOGICAL STUDIES:

PLAIN X-RAY ABDOMEN

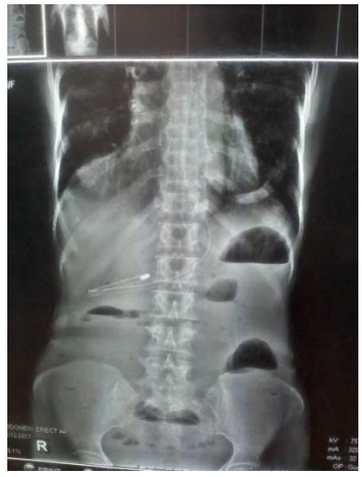
Plain supine and erect films of the abdomen or lateral decubitus view in patients should be obtained.

X-RAY FINDINGS IN GENERAL PATHOLOGICAL CONDITIONS:

1) **AIR AND FLUID LEVELS:**

An enormously dilated stomach suggests gastric outlet obstruction due to a chronic duodenal ulcer or acute gastric dilatation. Displaced gastric shadow indicates sub-diaphragamtic abscess, hematoma or pancreatic mass.

Although gas patterns in bowel loops is an important finding. Small amount of gas are detected commonly in stomach and colon, with smaller amounts in small intestines. Abnormal gas patterns are seen in motor disturbances of GIT, as in paralytic ileus, obstruction or associated with circulatory impairment with diminished gas absorption.



Ileus is characterized by gas being seen throughout the gastrointestinal tract.

Bowel obstruction is manifested by multiple air and fluid levels with dilatation of the bowel proximal to the obstruction. A high small bowel obstruction may be difficult to diagnose because repeated vomiting will effectively decompress the obstructed segments. Small bowel obstruction is differentiated from large bowel by identifying, circular valvulae conniventes, folds of mucous membrane of small bowel and the haustral folds.

Gas may be detected in biliary tract when there are fistulous tract between bowel and gall bladder or common bile duct or gallstone ileus.

Along with clinical findings, the distinctive radiologic appearances of colonic dilatation in toxic megacolon or volvulus, establishes the diagnosis. Thumb print impression on colonic walls are noted in half the cases of

ischemic colitis.

Abdominal radiographs are only 50-60% sensitive for small bowel obstruction. In most cases, the abdominal radiograph will have the following features:

Dialated loops of small bowel proximal to the obstruction (see 3-6-9 rule)

Predominantly central dilated loops

Three instances of dilatation >2.5-3 cm

Valvulae conniventes are visible

Air-fluid levels

However, obstruction may also present with the following features:

Gasless abdomen: gas within the small bowel is a function of vomiting, NG tube placement and level of obstruction

String-of-beads sign: small pockets of gas within a fluid-filled small bowel

Abdominal radiographs will show a large, dilated loop of the colon, often with a few gas-fluid levels. Specific signs for sigmoid volvulus are:

Coffee bean sign

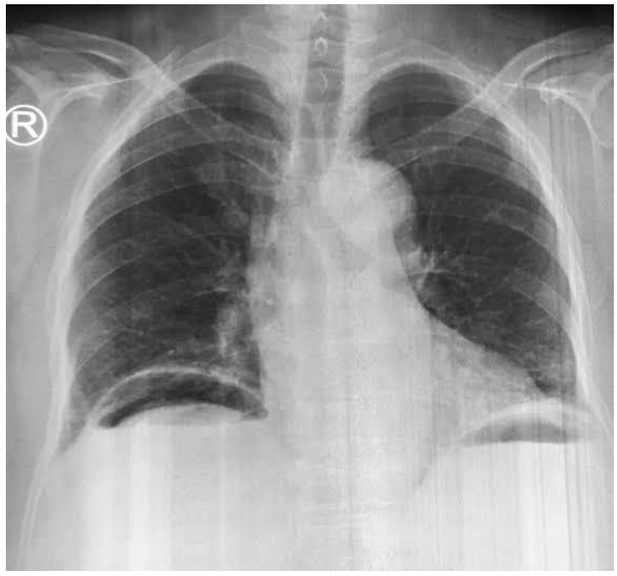
Frimann-Dahl sign - three dense lines converge towards the site of obstruction

Absent rectal gas

2) PNEUMO PERITONEUM:

Free air in peritoneum is usually detected when x-ray is taken in erect posture. The film must include diaphragm. If the patient is too ill, he may be placed in lateral position, that is lying on one flank up, and X-ray obtained as lateral view of abdomen.

Free air develops in associations with a perforated viscus, commonest being perforated duodenal ulcer. Here gas collects in moderate quantities which escape under the dome of diaphragm.. In perforations of the stomach and colon may be associated with much larger quantities. In contrast, in small intestine perforations, only a small quantities escape. In perforations of the stomach and colon may be associated with much larger quantities of free intraperitoneal air.



The other conditions are typhoid perforation, diverticulitis with perforation. Pneumo-peritoneum is unusual in appendicular perforation. Free gas under hemidiaphragm is present in approximately 80% of perforated ulcers and corroborates the clinical diagnosis.

Plain abdominal radiography should be the first diagnostic modality used in suspicion of a perforated viscus. It is possible, using careful radiographic technique, to demonstrate as little as 1 mL of free gas on an erect chest or left lateral decubitus abdominal film.20 The high percentage of missed cases is due to technical imperfections rather than limitations of the test (poor quality of plain abdominal radiography, excluding the uppermost portion of the peritoneal cavity of the image)

Plain radiography is infrequently able to give the diagnosis, however, is useful for identifying free gas, and may show an appendicolith in 7-15% of cases 1. In the

right clinical setting, finding an appendicolith makes the probability of acute appendicitis up to 90%.

If an inflammatory phlegmon is present, displacement of caecal gas with mural thickening may be evident.

Small bowel obstruction pattern with small bowel dilatation and air-fluid levels is present in ~40% of perforations.

An erect chest x-ray is probably the most sensitive plain radiograph for the detection of **air under diaphragm**. If a large volume pneumoperitoneum is present, it may be superimposed over a normally aerated lung with normal lung markings.

BOWEL-RELATED SIGNS

Double Wall Sign (Also Known As Rigler Sign Or Bas-Relief Sign)

Telltale Triangle Sign (Also Known As The Triangle Sign Or Telltale Triangle)

Peritoneal Ligament-Related Signs

Football Sign

Falciform Ligament Sign

Lateral Umbilical Ligament Sign (Also Known As Inverted "V" Sign)

Urachus Sign

Right Upper Quadrant Signs

Cupola Sign

Fissure For Ligamentum Teres Sign

Hepatic Edge Sign

Lucent Liver Sign

Morison Pouch Sign (Doge Cap Sign)

Periportal Free Gas Sign

OBLITERATIONS OF PSOAS MARGIN:

Obliteration of psoas margin usually indicates retroperitoneal disease. Contraction of right flank, obliteration of psoas margin, curving of spine with concavity toward right are important findings in cases of perforated duodenal ulcer, acute appendicitis, right ureteric calculus. Similar findings on the other side is due to left ureteric calculus or ruptured spleen.

ABNORMAL SHADOWS AND ABNORMALITY OF NORMAL SHADOWS:

Calcified deposits may be of significance. It may be of diagnosis according to location as Gall stones, Renal stone, Ureteric stone, Lymph node, pancreatic calcification, calcified aortic aneurysm, phelobolith, prostatic calculi etc. Some are related to acute abdomen and some are incidental findings. A fecolith in the area of appendix with presence of right lower quadrant pain indicates appendicitis.

ULTRA SONOGRAPHY:

High resolution ultrasonography has also been used as a method of improving diagnostic accuracy. The appearances used to diagnose acute appendicitis on ultra sonography are the presence of a non compressible peristaltic tubular structure with a dilated lumen and a thickened wall. This investigation when performed by an expert it may be of value in more difficult cases, particularly in young women. Patient without a sonographically visible appendix recognition of loculated fluid and prominent periappendiceal fat may be a useful indirect clue to the diagnosis of perforated appendicitis.

Currently, diagnostic criteria used for the diagnosis of acute appendicitis by ultrasound are

1. Blind ending, immobile, non-compressible, aperistaltic, tubular structure. Mural

thickness is assessed by measuring the distance from the echogenic mucosa to the outer edematous wall that shows few echoes.

2. Cannot be displaced on pressure.

3. Bull's eye or target lesion visualized in the transverse plane with diameter > 6mm.

4. Faecolith in the lumen.

5. Periappendiceal collection.

6. Hypo or hyperperistaltic loops in the right iliac fossa.

7. Miscellaneous signs:

'Cockade' around target lesion. Tubular structure > 50 mm in length.

Jeffrey et al8 in a study, pointed out the sonographic pitfalls in the diagnosis of acute appendicitis, in which they observed a that a dilated fallopian tube or hypertrophied fibers of the psoas muscle could be mistaken for a target lesion, while a gas containing appendix could be mistaken for a bowel loop.

In acute cholecystitis, ultrasonography is the current most commonly used, imaging method, can rapidly assess the caliber of the biliary tree, the presence or absence of biliary calculus and the appearance of the gall bladder wall and contents.

In case of acute pancreatitis ultrasound can be done to assess the suspected

complications. Because of the frequent accompanying a dynamic ileus, CT is more reliable.

The indications for the use of ultrasound in the evaluation of the acute abdomen have increased dramatically in the past decade. Ultrasonography can provide rapid morphologic evaluation of liver, spleen, pancreas and kidney, with the advent of pulsed Doppler ultrasound, the blood vessels of the abdomen can be studied with remarkable precision.

Because of the frequently occurring adynamic ileus in patients with an acute abdomen large areas of the abdomen are inaccessible for ultrasound evaluation owing to the interposed gas, which transmits a sound wave poorly.

Ultrasonography and diagnostic peritoneal lavage are similar in terms of sensitivity and specificity for fluid detection, but ultrasongoraphy has clear advantage as it is non-invasive, results in fewer non-therapeutic laparotomies, can be repeated, allows resuscitative efforts to proceed while the patient is being scanned, does not interfere with subsequent imaging and, rather simply diagnose the presence of blood in peritoneal cavity, may identify which organ has been injured.

The FAST exam evaluates the pericardium and three potential spaces within the peritoneal cavity for pathologic fluid. The right upper quadrant visualizes the hepatorenal recess, also known as Morrison's pouch, the right paracolic gutter, the hepato-diaphragmatic area, and the caudal edge of the left liver lobe. Position the probe in the sagittal orientation along the patient's flank at the level of the 8 to 11 rib spaces. This view is the most likely to detect free fluid with an overall sensitivity of 66%. Recent retrospective evidence suggests the area along the caudal edge of the left lobe of the liver has the highest sensitivity, exceeding 93%.

Next, subxiphoid (or subcostal) views to evaluate the pericardial space. Ultrasound detects as little as 20 cc of pericardial fluid and studies have shown excellent sensitivities and specificities approaching 100%. Traumatic pericardial tamponade happens rapidly with as little as 50cc to 100 cc preventing the pericardial compliance from accommodating as it does with gradually accumulating effusions common in numerous chronic medical conditions. There are several sonographic findings of cardiac tamponade. Right ventricular collapse during ventricular diastole and inferior vena cava plethora are the easiest and most frequently observed. The subcostal view helps differentiate between pleural and pericardial effusions as well since there is no pleural reflection present.

Following the subxiphoid view, image the left upper quadrant to inspect the splenorenal recess, the subphrenic space, and the left paracolic gutter, as well as the left lower hemithorax when performing an Extended FAST exam (eFAST). Similar views of the right hemithorax are obtained when scanning the right upper quadrant. The presence of the hyperechoic vertebral bodies, or **"spine sign,"** aids in identifying pleural fluid. Sensitivities and specificities of ultrasound for the detection of hemothorax are 92% to 100%. Finally, suprapubic images evaluate for free fluid in the rectovesical pouch in males and the rectouterine (Pouch of Douglas) and vesicouterine pouches in females.

In addition to the anatomy described above, the eFAST incorporates views of the right and left anterior hemithorax to detect the presence of a pneumothorax. Typically, a small amount of pleural fluid lines the interface between the parietal and visceral pleurae, allowing for synchronized lung and chest wall expansion and contraction during inhalation and exhalation, respectively. The sonographic appearance is described as pleural lung sliding or the **"ants marching"** sign.

ARTIFACTS AND PITFALLS

Bowel imaging is subject to artifact from air that can disperse and distort the image. Potential pitfalls include:

1. Misinterpretation of images. Patients with ascites may have prominent bowel walls. Chronic inflammatory bowel disease, amyloidosis, and Behcet's disease may have chronic bowel thickening.

2. Not applying enough pressure to displace gas and compress normal bowel

3. Not imaging in orthogonal planes to clearly distinguish bowel from other cystic structures

4. Mistaking large bowel for small bowel or vice versa

5. Measuring the posterior wall of the bowel where posterior acoustic enhancement could lead to falsely enlarged measurements

6. Misidentifying adjacent structures such as the gallbladder, stomach, or bladder as evidence of local free fluid.

7. Mistaking ascites for local free fluid

CT SCAN :

CT has evolved as the premier technique for triaging most patients. CT has earned this role because it can provide a global perspective of the gut, mesenteries, omentum, peritoneum, retroperitoneum, subperitoneum, and extraperitoneum uninhibited by the presence of bowel gas and fat. Helical scanning allows thinner contiguous images to be obtained without increasing radiation exposure and without respiratory misregistration. The rapidity of scanning allows several acquisitions to be obtained during different phases of a single IV contrast bolus.

CT Examination

Use of CT in the evaluation of acute abdominal pain has increased to a large extent. This increase was related to the high accuracy of CT in the diagnosis of specific diseases that can be achieved with use of multidetector CT scanners.

The CT technique used to examine patients with acute abdominal pain generally involves scanning of the entire abdomen after intravenous administration of an iodinated contrast medium. Although abdominal CT can be performed without contrast medium, the intravenous administration of contrast material facilitates good accuracy—with a positive predictive value of 95% reported for the diagnosis of appendicitis —and a high level of diagnostic confidence, especially in rendering diagnoses in thin patients, in whom fat interfaces may be almost absent . Although rectal or oral contrast material may be helpful in differentiating fluid-filled bowel loops from abscesses in some cases, the use of oral contrast material can markedly increase the time these patients spend in the emergency department. The lack of enteral contrast medium does not seem to hamper the accurate reading of CT images obtained in patients with acute abdominal pain as it does in postoperative patients. For example, in a series of 1021 consecutive patients with acute abdominal pain in whom only intravenous contrast medium was administered, there were no inconclusive CT scans due to the lack of enteral contrast medium . Multiplanar reformation is beneficial, especially in cases of equivocal CT scans, and it increases the radiologist's level of confidence in the diagnosis.

Studies to evaluate the accuracy of abdominal CT performed in patients with acute abdominal pain generally are scarce. In the cohort study of 1021 consecutive patients with acute abdominal pain, USG and CT were compared for the determination of urgent diagnoses . CT was significantly more sensitive than USG (89% vs 70%, $P < .001$). The highest sensitivity (only 6% missed urgent cases) was obtained with a diagnostic strategy involving the use of initial USG, followed by CT, only in negative or inconclusive USG cases. Use of this approach also led to a reduction in radiation exposure because CT was needed for only 49% of the patients. Alternative strategies based on the body mass index or age of the patient or on the location of the pain resulted in a loss of sensitivity. In the literature, there are two randomized controlled trials in which standard practice was compared with early CT—in one study, early CT was performed within 1 hour of presentation, and in the other study, it was performed within 24 hours—in patients who presented with acute abdominal pain. In these two studies, standard practice involved conventional abdominal and chest radiography and, if necessary, additional CT. CT was requested in half the patients in the standard practice group.

Prospective studies involving the examination of patients for whom the clinician ordered CT scanning have shown that CT findings have a significant effect on diagnoses. In one study, the accuracy of the clinical diagnosis made before CT was performed improved from 71% to 93% after CT was performed. The accompanying change in treatment management was 46%. Another study revealed a significant

increase in the level of confidence of the diagnosis made with CT: The treatment management for 60% of patients was changed. Abdominal CT reportedly yields good overall interobserver agreement and very good interobserver agreement for the determination of specific urgent diagnoses, with reported κ values of 0.84, 0.90, and 0.81 for agreement regarding the diagnoses of appendicitis, diverticulitis, and bowel obstruction, respectively.

Exposure to ionizing radiation is a disadvantage of CT. The use of intravenous contrast medium is a drawback in patients with imminent renal insufficiency.

PITFALLS IN CT SCAN

Not recognising ischaemic bowel

Bowel ischaemia is often fatal if unrecognised, and can be a difficult clinical diagnosis to make. When assessing this on CT it is vital to give IV contrast to assess vascular patency and bowel wall enhancement—both arterial and portal venous phases are recommended. A pre-contrast scan may help to identify intramural haemorrhage, which can mimic mural enhancement on post-contrast images alone, but is not always necessary as other post-contrast features will usually indicate the diagnosis. It is also important not to give positive oral contrast, as this will mask mucosal enhancement (in fact, positive oral contrast is generally not recommended in the setting of the acute abdomen because of the risk of missing bowel ischaemia). In some cases, the CT features are clear cut (i.e. mural oedema, poor mural

enhancement, intramural gas, free fluid and associated vascular filling defects +/- the presence of gas in the portal system).

The features present can differ depending on the cause–venous occlusion tends to cause more mural oedema and mesenteric congestion than arterial occlusion, whereas arterial occlusion tends to reduce mural enhancement earlier and also causes earlier transmural infarction. The mesenteric arteries and veins should always be carefully assessed for the presence of filling defects representing an embolus (in arteries) or a thrombus (in veins or arteries). In the mesenteric arteries, thrombosis usually occurs near the origin of the superior mesenteric artery (SMA)/inferior mesenteric artery (IMA), whereas emboli tend to wedge at branching points. Occasionally in cases of arterial embolism, small infarcts may be seen in the spleen or kidneys, and in rare instances a thrombus may be visible in the left atrial appendage acting as a source for the emboli.

Venous thrombosis has many different causes, such as thrombophilia, myeloproliferative disorders, malignancy, inflammation, recent surgery/trauma, portal hypertension and oral contraceptives. It is not uncommon to see typical features of ischaemia without a visible arterial/venous occlusion—in these cases the differential diagnosis also includes vasculitis (e.g. polyarteritis nodosa, Henoch–Schönlein purpura, systemic lupus erythematosus and Behçet syndrome), overdistension of the bowel (e.g. due to bowel obstruction, faecal impaction or

paralytic ileus) and low-flow states (e.g. hypovolaemic shock, heart failure or drug-induced splanchnic vasoconstriction). Ischaemia due to low-flow states usually occurs at watershed areas between vascular territories (e.g. at the splenic flexure, at the rectosigmoid junction and, rarely, in the caecum).

In some cases of bowel ischaemia the CT features are subtle—bowel dilatation without a discrete transition point can occasionally be the only sign of ischaemia. Furthermore, there may be paradoxical hyperenhancement of the bowel wall rather than reduced enhancement , due to hyperaemia and/or reperfusion via collaterals. Intramural and portal system gas are ominous signs in the presence of bowel ischaemia, indicating transmural infarction; however, intramural gas does not always imply ischaemia and is also seen in benign pneumatosis. In these cases, the patients will usually be asymptomatic and other features of ischaemia will be absent.

Not recognising a closed loop small bowel obstruction

CT is the imaging test of choice when investigating small bowel obstruction. One of the most important considerations is whether a closed loop obstruction is present (i.e. two transition points at a single location creating a bowel loop that is obstructed at both ends. In most cases an adhesive band (usually related to previous surgery) has crossed over a loop of bowel, thereby obstructing the afferent and efferent limbs. However, volvulus and hernias (both external and internal) may also be responsible.

Closed loop obstruction requires urgent surgical intervention because of the risk of strangulation at the point of obstruction, causing mesenteric venous occlusion and subsequent venous ischaemia and infarction. When features of venous ischaemia are present, it is usually straightforward to diagnose closed loop obstruction on CT, as the oedematous dilated bowel and congested mesentery stand out from the rest of the dilated thin-walled bowel.

In cases secondary to band adhesions, the point of obstruction can be difficult to identify, as the adhesions are not usually visible (except in rare cases where a little fat becomes entrapped within the band. **The small bowel faeces sign** (semisolid content in the small bowel lumen), if present, can help to identify the point of obstruction. The cardinal signs of closed loop obstruction include two tightly angulated bowel loops in close proximity with beaked tapering and convergence at the point of obstruction, focal narrowing/obliteration of mesenteric veins as they pass through the point of obstruction followed by venous engorgement within the closed loop mesentery, a cluster of stacked oedematous bowel loops, and **a 'whirl' sign** within the mesentery as it approaches the point of obstruction. The 'whirl' sign can be seen in any cause of closed loop obstruction, but is particularly prominent in cases of volvulus. Patients with small bowel volvulus also usually have a predisposing congenital intestinal malrotation.

Internal hernias are a rare cause of closed loop obstruction and occur through peritoneal defects, foramina and recesses (e.g. foramen of Winslow, paraduodenal/pericaecal fossae, perirectal/supravesical recesses, and transomental/transmesenteric/broad ligament defects), which may be congenital or acquired (e.g. the Petersen's defect in the transverse mesocolon in patients who have had a retrocolic roux-en-Y anastomosis).

MAGNETIC RESONANCE IMAGING :

MR imaging is not yet widely used in the diagnostic work-up of patients who present with acute abdominal pain. The major advantage of MR imaging is the lack of ionizing radiation exposure. The high intrinsic contrast resolution rendered with MR imaging is another advantage, as intravenous contrast medium may not be required. The high intrinsic contrast resolution has the potential to be particularly valuable for assessment and diagnosis of pelvic disease in female patients, but this has not been substantiated. In the past, MR imaging required long examination times. Currently, with recently introduced high-speed techniques, MR imaging protocols for patients with acute abdominal pain involve examination times shorter than 15 minutes. However, the lack of around-the-clock availability of MR imaging is still a logistic problem at many hospitals.

MR imaging has demonstrated promising accuracy for the assessment and diagnosis of appendicitis, albeit in a relatively small series of patients, who often were pregnant. MR imaging is also accurate in the diagnosis of diverticulitis. MR imaging

is more accurate than CT for the diagnosis of acute cholecystitis and the detection of common bile duct stones . However, the body of scientific research on the use of MR imaging in patients with acute abdominal pain is relatively limited. Therefore the availability of and expertise with this examination are limited, and the cost-effectiveness has not been studied. MR imaging has contraindications, including claustrophobia, which may prevent MR imaging from being performed.

LAPAROSCOPY :

Laparoscopy has an established role prior to laparotomy in women in whom the diagnosis of appendicitis is uncertain. A ruptured graffin follicle, pelvic inflammatory disease or other tubo-ovarian conditions can be readily diagnosed thereby averting an unnecessary laparotomy. Laparoscopy is also useful diagnostic tool in managing obtunded, elderly or critically ill patients who may have atypical manifestations of an acute abdomen.

MATERIALS AND METHODS

This study has been conducted in Department of General Surgery Tirunelveli Government Medical College. Based on the analysis of 162 cases of acute abdomen admitted to Tirunelveli Medical College Hospital, Tirunelveli, fulfilling the criteria were selected for the study.

INCLUSION CRITERIA:

- All cases of acute abdomen will be taken up for study.

EXCLUSION CRITERIA:

- All cases of acute abdomen deceased before taken up for surgery.

COLLECTION OF DATA:

An elaborate study of patients with acute abdomen with regard to history which includes the onset, character, location, duration, radiation and chronology of the pain experienced. The intensity and severity of the pain, worsening and relieving factors along with similar history in the past will be questioned. Other symptoms like nausea, vomiting, constipation, diarrhea, pruritus, melena, hematochezia and hematuria will be questioned. The physical examination beginning with general examination of the patient followed by inspection, palpation, percussion and auscultation of the abdomen. Digital rectal examination along with examination of external genitalia will be done.

Biochemical test like hemoglobin, white blood cell count with differential, electrolytes, urea, creatinine, liver function test, urinalysis are to be done in relevant cases.

Radiological investigations including plain chest radiograph, erect abdominal radiograph, abdominal ultrasonography and CT scan of the abdomen in needed patients will be done for patients with acute abdomen.

RESULTS

TABLE 1: SYMPTOMS

SYMPTOMS	NUMBER	PERCENTAGE
PAIN	162	100.0
VOMITING	107	66.0
FEVER	95	58.6
ABDOMINAL DISTENTION	20	12.3
CONSTIPATION	35	21.6
DIARRHOEA	5	3.1

TABLE 2: SIGNS

SIGNS	NUMBER	PERCENTAGE
TENDERNESS	162	100.0
GUARDING	79	48.8
RIGIDITY	29	17.9
DECREASED BOWEL SOUNDS	64	39.5
FREE FLUID	26	16.0
LIVER DULLNESS	35	21.5
SHOCK	1	0.6

TABLE: 3 LEUCOCYTOSIS

	LEUCOCYTOSIS	
	Yes	No
Acute Appendicitis	55	23
Appendicular Perforation	16	4
Perforative Peritonitis	28	12
Intestinal Obstruction	14	10

TABLE 4: INVESTIGATIONS SUGGESTIVE OF DIAGNOSIS

INVESTIGATIONS SUGGESTIVE OF DIAGNOSTIC	NUMBER	PERCENTAGE
X-RAY	54	33.3
USG	113	69.8
CT SCAN	79	92.8

TABLE 5: CLINICAL DIAGNOSIS

CLINICAL DIAGNOSIS	NUMBER	PERCENTAGE
ACUTE APPENDICITIS	83	51.2
APPENDICULAR PERFORATION	15	9.3
INTESTINAL OBSTRUCTION	29	17.9
PERFORATION PERITONITIS	35	21.6
TOTAL	162	100.0

TABLE 6: INTRAOPERATIVE DIAGNOSIS

INTRAOPERATIVE DIAGNOSIS		NUMBER	PERCENTAGE
ACUTE APPENDICITIS		78	48.1
APPENDICULAR PERFORATION		20	12.3
INTESTINAL OBSTRUCTION		23	14.2
PERFORATION PERITONITIS 41(25.4)	DU	22	13.6
	GBP	2	1.2
	GP	10	6.2
	IP	4	2.5
	JP	3	1.9
TOTAL		162	100.0

TABLE 7: ACUTE APPENDICITIS

CLINICAL	INTRAOPERATIVE		TOTAL	CHI-SQUARE
	YES	NO		
YES	78	5	83	143.18 DF=1 P<0.001
	94.0%	6.0%	100.0%	
NO	0	79	79	
	.0%	100.0%	100.0%	
TOTAL	78	84	162	
	48.1%	51.9%	100.0%	

TABLE 8: APPENDICULAR PERFORATION

CLINICAL	INTRAOPERATIVE		TOTAL	CHI-SQUARE
	YES	NO		
YES	15	0	15	117.37 DF=1 P<0.001
	100.0%	.0%	100.0%	
NO	5	142	147	
	3.4%	96.6%	100.0%	
TOTAL	20	142	162	
	12.3%	87.7%	100.0%	

TABLE 9: INTESTINAL OBSTRUCTION

CLINICAL	INTRAOPERATIVE		TOTAL	CHI-SQUARE
	YES	NO		
YES	23	6	29	122.9 DF=1 P<0.001
	79.3%	20.7%	100.0%	
NO	0	133	133	
	.0%	100.0%	100.0%	
TOTAL	23	139	162	
	14.2%	85.8%	100.0%	

TABLE 10: PERFORATION PERITONITIS

CLINICAL	INTRAOPERATIVE		TOTAL	CHI-SQUARE
	YES	NO		
YES	35	0	35	131.76 DF=1 P<0.001
	100.0%	.0%	100.0%	
NO	6	121	127	
	4.7%	95.3%	100.0%	
TOTAL	41	121	162	
	25.3%	74.7%	100.0%	

TABLE 11:COMPARISON OF CLINICAL AND OPERATIVE DIAGNOSIS

OPERATIVE DIAGNOSIS	**CLINICAL DIAGNOSIS**							
	Acute Appendicitis		Appendicular Perforation		Perforative Peritonitis		Intestinal Obstruction	
	No	%	No	%	No	%	No	%
Acute appendicitis	78	93.9	-	0	-	0	-	0
Appendicular perforation	5	6.02	15	100	-	0	-	0
Perforative peritonitis	-	0	-	0	35	100	5	17.24
Intestinal obstruction	-	0	-	0	-	0	24	82.7

TABLE 12:

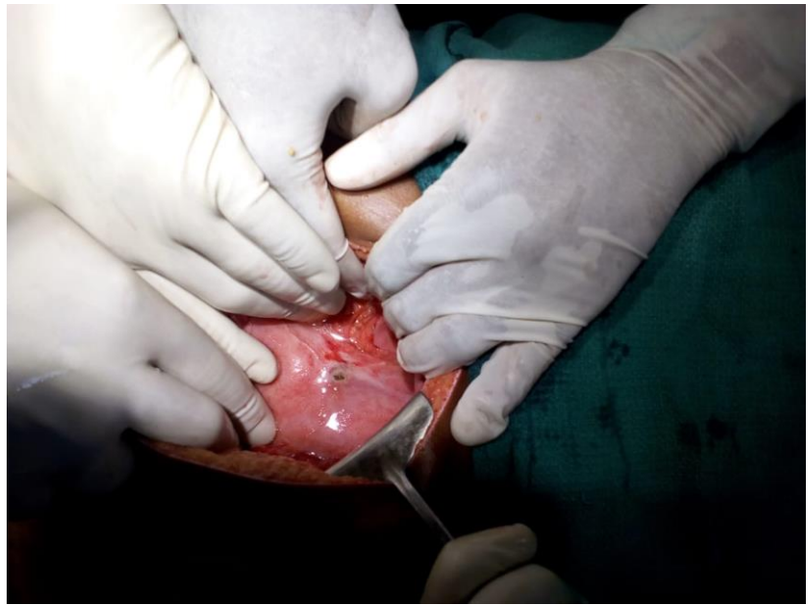
VARIABLES	SENSITIVITY	SPECIFICITY	PPV	NPV	LR+	LR-	ACCURACY
ACUTE APPENDICITIS	100%	94.05%	93.98%	100%	16.80	0.00	96.91%
APPENDICULAR PERFORATION	75%	100%	100%	96.60%	0.00	0.25	96.91%
INTESTINAL OBSTRUCTION	100%	95.68%	79.31%	100%	23.17	0.00	96.30%
PERFORATION PERITONITIS	85.37%	100%	100%	95.28%	0.00	0.15	96.30%

DISCUSSION

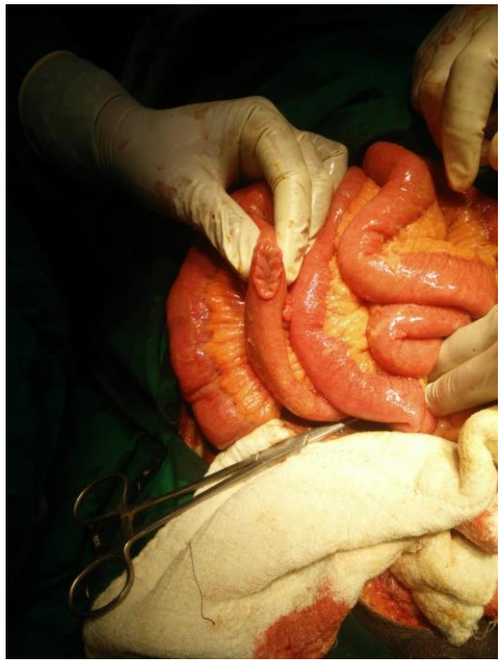
Out of 162 patients, 83 patients who were clinically diagnosed as acute appendicitis, intra operatively acute appendicitis was found in 78 patients (93.9%), and 5 patients had appendicular perforation (6.02%).

Out of 15 patients clinically diagnosed to have appendicular perforation, all had appendicular perforation intraoperatively (100%).

Out of 35 patients with clinical diagnosis of perforative peritonitis, all had found to have perforation intraoperatively (100%) (duodenal perforation-22; gastric perforation-10; ileal perforation-3).

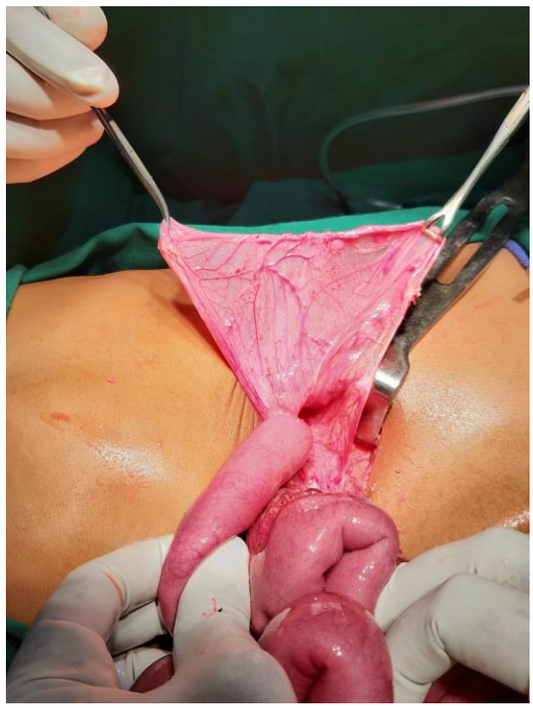


Gastric Perforation

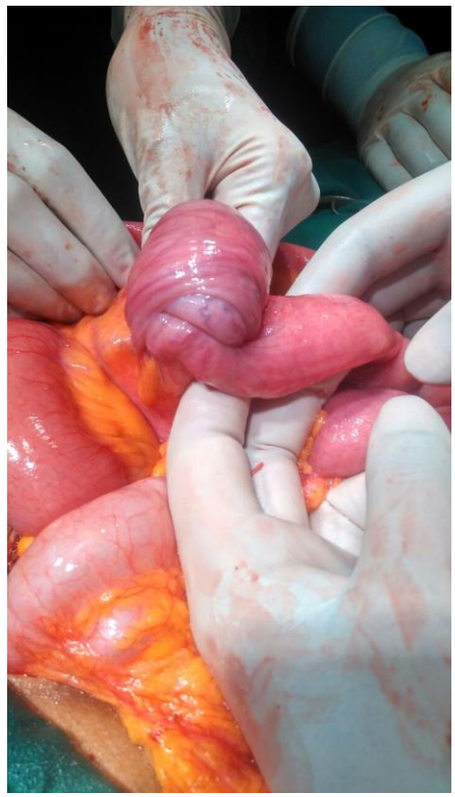


Ileal Perforation

Out of 29 patients with clinical diagnosis of intestinal obstruction, 12 had small bowel obstruction and 10 had large bowel obstruction (82.7%) including adhesions due to bands(6), Meckel's divericulum due to the presence of mesodiverticular band (3), tuberculous abdomen with obstruction due to strictures in the small bowel (3), sigmoid volvulus (2), mesenteric ischemia (2), paraduodenal hernia (2), colonic malignancy (sigmoid colon growth-2 and transverse colon growth-1 and caecal growth-1) and intususception at jejuno-jejunal level. (1).

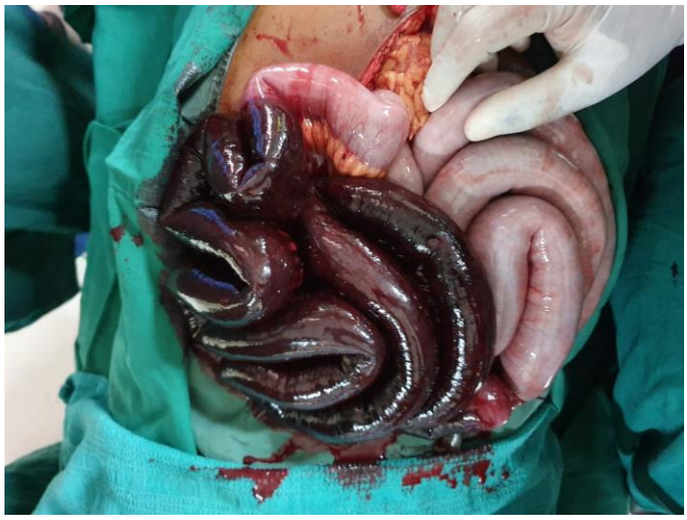


Paraduodenal Hernia

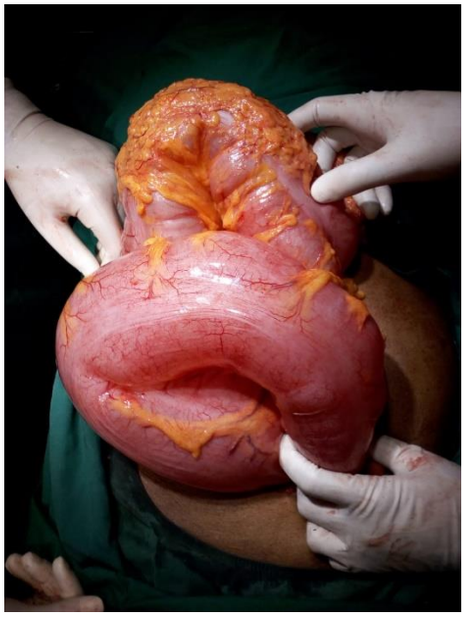


Intussception

2 patients who were clinically diagnosed to have intestinal obstruction, were intraoperatively diagnosed to have mesenteric ischemia resulting in bowel gangrene. One patient who was clinically diagnosed to have intestinal obstruction was found to have hemorrhagic pancreatitis intraoperatively.

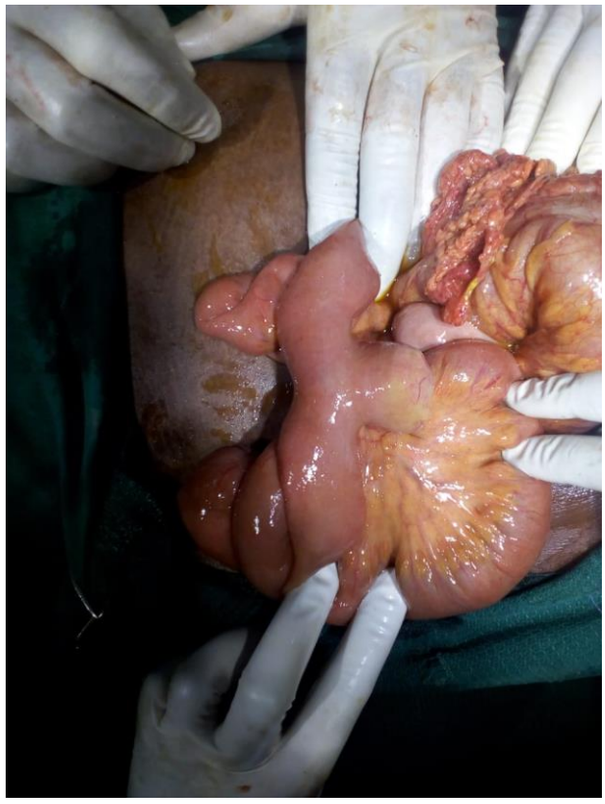


Mesenteric Ischemia



Sigmoid Volvulus

The other 5 patients (17.24%) who were diagnosed clinically as intestinal obstruction were found to have perforation intraopertively (jejunal perforation-2, GB perforation-2, ileal perforation-1).



Meckel's Diverticulum

Total leukocyte count was elevated in 55 patients of acute appendicitis (70.5%), 16 patients with appendicular perforation (80%), 28 patients with perforative peritonitis (70%) and 14 patients with intestinal obstruction (58.3%).

In a study of 493 patients with acute appendicitis, Pieper and associates in 2002 noted that 66.7% had a leukocyte count of 11,000 or more and 5.5% had a raised count of more than 20,000.

Vermeulen et al after evaluating 221 adult patients admitted with right lower

abdominal pain have concluded that the white cell count did not significantly influence surgical decision-making in cases of suspected acute appendicits. Coleman et al. reported that WBC is a poor predictor of the severity of the disease. With appendicitis the white cell count has been variously reported as being either reliable or unreliable. Thus although a raised white cell count is highly sensitive test for acute appendicitis, it is rendered almost useless due to its low specificity and it has little diagnostic value. Where the white cell count is at variance with the clinical features, the latter should take precedence. The only value of white cell count would seem to be to prompt observation rather than operation in a patient who has equivocal features of appendicitis together with a normal count.

Radiological test such as X-ray was found to be accurate especially in the diagnosis of perforative peritonitis and intestinal obstruction with an accuracy of 87.5% and 79.1% respectively.

The diagnostic utility of USG abdomen and pelvis was not statistically significant between these three clinical variables i.e. appendicular perforation, perforative peritonitis and intestinal obstruction but showed 66.6% accuracy for acute appendicitis which is the highest and is in accordance with other studies.

Among the patients who had undergone CT abdomen and pelvis, the diagnostic accuracy was 94.7% for acute appendicitis, 85.7% for appendicular perforation, 100% for perforative peritonitis and 84.2% for intestinal obstruction.

The accuracy of clinical diagnosis in acute abdomen cases are 96.91% for acute appendicitis, 96.91% for appendicular perforation, 96.30% for perforative peritonitis and intestinal obstruction.

The accuracy of clinical diagnosis in acute abdomen is better when compared with the accuracy of biochemical and radiological diagnosis.

CONCLUSION

- Clinical examination was found to be statistically correlating with the intraoperative findings.
- Radiological investigations like USG had high sensitivity for appendicitis but overall low specificity.
- Erect x-ray was diagnostic of perforation and intestinal obstruction.
- CT scan was superior when compared with x-ray and USG.
- Thus clinical judgement is key to the diagnosis of acute abdomen with biochemical and radiological investigations only aiding in its management and cannot replace the clinical decision.
- Despite the improvements in technology and time constraints, clinical examination of the patient remains noteworthy in making a diagnosis.
- Thus clinical examination remains gold standard in making a diagnosis of acute abdomen.

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PROFORMA

1. Case No :
2. Name :
3. Age Sex :
4. Address :
5. I.P. No :
6. Unit / Ward
7. Date of admission
8. Date of Surgery
9. Date of discharge
10. Chief complaints
    i. Pain – Onset, character, location. Duration, radiation, worsening & relieving factors.
    ii. Nausea / vomiting
    iii. Constipation / diarrhea
    iv. Others like pruritus, melena, hematochezia, hematuria
    v. Similar complaints in the past / previous surgeries
11. General physical examination

    Pallor / Icterus

    BP

    PR

12. Examination of abdomen (including external genitalia)
    i. Inspection
    ii. Palpation
    iii. Percussion
    iv. Auscultation
    v. P/R
13. Clinical diagnosis
14. Biochemical investigation

    CBC

    RFT

    LFT

    RBS

    Urine routine
15. Radiological investigation
    i. Chest X-Ray
    ii. Abdominal X-Ray Erect
    iii. Abdominal ultrasonography
    iv. CT Abdomen
16. Surgery done:
17. Post-operative diagnosis

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

Sl. No.	NAME	IP NO	AGE/SEX	PAIN	VOMITING	FEVER	ABD. DIS	CONSTIPATION	DIARRHOEA	TACHYCARDIA	TENDERNESS	GUARDING	RIGIDITY	DEC. BS	FREE FLUID	LIVER DULLNESS	SHOCK	RFT	LEUCOCYTOSIS	X-RAY	USG	CT	CLINICAL DIAGNOSIS	OPERATIVE DIAGNOSIS
1	mydeen	61081	43/M	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
2	essakkiappan	61119	55/M	Present	P	Present	A	Present	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Diagnostic	PP	DU
3	mariappan	59483	48/M	Present	P	Present	A	A	A	Present	Present	Present	A	Present	Present	Obliterated	A	N	Present	Diagnostic	Diagnostic	Diagnostic	PP	DU
4	sumithra	59456	31/F	Present	P	Present	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Diagnostic	AA	AA
5	mohammed khaja my	59469	17/M	Present	A	A	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
6	shara	56056	15/M	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
7	karthick	56053	24/M	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
8	petchimuthu	55987	21/M	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Diagnostic	AA	AA
9	manonmani	54488	49/F	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
10	murugan	52701	20/M	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
11	subramanian	50992	55/M	Present	P	Present	A	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	A	ND	ND	ND	IO	GB P
12	bhuvaneshwari	51011	21/F	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
13	venkatesh	47494	20/M	Present	P	Present	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
14	jenath nisha	47431	21/F	Present	A	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AP
15	selvi	45565	19/F	Present	A	Present	A	A	A	Present	Present	Present	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AP	AP
16	kasthuri	45548	50/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
17	ariyan kavoo	43754	60/M	Present	P	Present	A	Present	A	Present	Present	Present	Present	Present	A	Obliterated	Present	Elevated	Present	Diagnostic	ND	Diagnostic	PP	DU
18	velsamy	43653	40/M	Present	A	Present	A	A	A	Present	Present	Present	A	A	A	A	A	Elevated	Present	ND	ND	ND	AP	AP
19	mayandi	41801	32/M	Present	P	Present	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
20	naveen kumar	40220	15/M	Present	A	A	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
21	vasantha kumar	40046	13/M	Present	P	Present	A	A	Present	Present	Present	A	A	A	A	A	A	N	A	ND	ND	Not done	AA	AA
22	akbar	40177	48/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Not done	PP	DU
23	manoselvaraj	38264	27/M	Present	P	A	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
24	vignesh	36584	25/M	Present	P	Present	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
25	muppidathi	36456	19/F	Present	P	A	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
26	ramalingam	34287	45/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	A	Diagnostic	Diagnostic	Diagnostic	PP	GP
27	paul raj	32648	34/M	Present	P	Present	A	A	A	Present	Present	Present	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AP	AP
28	prem kumar	32711	19/M	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
29	peerkan beevi	30786	38/F	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
30	jeyaraj	30860	32/M	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
31	pappathi	25942	70/F	Present	P	A	Present	A	A	Present	Present	Present	A	Present	Present	A	A	Elevated	A	ND	ND	ND	IO	GB P
32	ilaiyaraja	27682	27/M	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	ND	AA	AA
33	mariammal	27726	50/F	Present	P	Present	Present	Present	A	Present	Present	Present	A	Present	A	A	A	N	Present	Diagnostic	Diagnostic	Diagnostic	IO	IO
34	karuppan	27669	36/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Diagnostic	PP	GP
35	shanmuga devar	27706	70/M	Present	P	Present	A	Present	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	A	Diagnostic	Diagnostic	Diagnostic	PP	GP
36	karthick	26070	18/M	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
37	karunakaran	25987	63/M	Present	A	A	A	A	A	Present	Present	Present	A	A	A	A	A	Elevated	A	ND	ND	Diagnostic	AP	AP
38	subramani	24309	62/M	Present	P	Present	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	Present	Diagnostic	Diagnostic	ND	IO	IO
39	akneal irsanth	22638	13/M	Present	P	Present	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
40	vijayalakshmi	21023	26/F	Present	A	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
41	kaja mydeen	20957	40/M	Present	P	Present	A	A	A	Present	Present	Present	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AP	AP
42	divya	19159	24/F	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
43	manikavel	9026	19/M	Present	A	A	Present	Present	A	No	Present	Present	A	Present	Present	A	A	N	Present	Diagnostic	Diagnostic	Diagnostic	IO	IO
44	kalai sankar	8980	18/M	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
45	rajam	7282	37/F	Present	A	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
46	anbuselvam	3638	13/M	Present	P	A	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
47	ramu	1983	51/F	Present	P	Present	A	Present	A	Present	Present	Present	A	Present	Present	Obliterated	A	N	Present	Diagnostic	ND	Diagnostic	PP	GP
48	pon essakki	93706	31/F	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
49	anbalagan	91859	69/M	Present	P	Present	A	Present	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	Diagnostic	Diagnostic	PP	DU
50	valli	89841	59/F	Present	P	Present	Present	A	A	Present	Present	Present	A	Present	Present	A	A	N	A	Diagnostic	Diagnostic	Diagnostic	IO	IO
51	ganesan	91780	42/M	Present	P	A	A	A	A	Present	Present	Present	A	A	A	A	A	Elevated	Present	ND	ND	Not done	AP	AP
52	parameshwari	91721	24/F	Present	P	Present	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	N	Present	Diagnostic	Diagnostic	Diagnostic	IO	IO
53	selvi	86094	43/F	Present	A	Present	A	A	A	Present	Present	Present	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AP	AP
54	shanmugaiah devar	86040	62/M	Present	P	A	A	Present	A	Present	Present	Present	A	Present	A	A	A	Elevated	A	Diagnostic	Diagnostic	Diagnostic	IO	IO
55	chinnadurai	86010	42/M	Present	P	Present	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	N	Present	Diagnostic	Diagnostic	Not done	IO	IO
56	athithya murugesh	86099	16/M	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Not done	AA	AA
57	muthusamy	86035	70/M	Present	P	A	A	Present	A	Present	Present	Present	A	Present	A	A	A	Elevated	A	ND	Diagnostic	Diagnostic	IO	IO
58	poomari	85975	24/F	Present	P	Present	A	A	A	Present	Present	Present	A	A	A	A	A	N	Present	ND	ND	Not done	AP	AP
59	murugammal	83884	32/F	Present	A	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
60	antochristi doss	83823	13/M	Present	P	A	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
61	muthukutty	81573	19/M	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
62	marimuthu	77700	57/M	Present	P	A	A	Present	A	Present	Present	Present	Present	Present	Present	A	A	Elevated	Present	Diagnostic	Diagnostic	Not done	IO	IO
63	nageshwari	77556	31/F	Present	A	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Diagnostic	AA	AA
64	subramani	77607	62/M	Present	P	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	N	Present	Diagnostic	Diagnostic	Diagnostic	IO	IO
65	dinesh	75304	17/M	Present	P	Present	A	A	A	No	Present	Present	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AP	AP
66	anandha bala	75276	15/M	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
67	appannaswamy	75263	63/M	Present	A	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	ND	Diagnostic	PP	DU
68	karthiga	24037	19/F	Present	P	Present	A	A	Present	No	Present	Present	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AP	AP
69	ekanath	23852	27/M	Present	A	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
70	selvamani	24028	54/M	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
71	chellapandi	24144	23/M	Present	P	A	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA

Sl. No.	NAME	IP NO	AGE/SEX	PAIN	VOMITING	FEVER	ABD. DIS	CONSTIPATION	DIARRHOEA	TACHYCARDIA	TENDERNESS	GUARDING	RIGIDITY	DEC. BS	FREE FLUID	LIVER DULLNESS	SHOCK	RFT	LEUCOCYTOSIS	X-RAY	USG	CT	CLINICAL DIAGNOSIS	OPERATIVE DIAGNOSIS
72	thangamani	26145	35/F	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
73	priya	27128	30/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
74	essakkiappan	28141	23/M	Present	A	Present	A	A	Present	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Not done	AA	AP
75	kumar	29303	40/M	Present	P	Present	A	Present	A	Present	Present	Present	A	Present	Present	A	A	N	Present	Diagnostic	Diagnostic	Diagnostic	IO	IO
76	selvaraj	31376	29/M	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
77	mariammal	31385	35/F	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Diagnostic	AA	AA
78	jaya	31476	57/M	Present	P	A	A	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	A	Diagnostic	Diagnostic	Diagnostic	IO	IO
79	chellathai	31400	55/F	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
80	thangalakshmi	35493	25/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
81	sankaravadivoo	35506	90/F	Present	P	A	A	Present	A	Present	Present	Present	A	Present	A	A	A	Elevated	Present	ND	ND	Not done	IO	JP
82	bharath	37383	25/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Diagnostic	PP	DU
83	abdul kadhar	39381	53/M	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
84	arun raj	40951	30/M	Present	P	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	A	Diagnostic	Diagnostic	Diagnostic	IO	IO
85	manikavel	41138	34/M	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	ND	ND	AA	AA
86	subash	41077	22/M	Present	A	Present	A	A	A	No	Present	Present	A	A	A	A	A	N	Present	ND	ND	Not done	AP	AP
87	mariammal	42939	32/M	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
88	kalimuthu	43149	66/M	Present	P	Present	Present	Present	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	Diagnostic	Diagnostic	PP	DU
89	sudalaikannu	43182	17/M	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	ND	Diagnostic	AA	AA
90	ramar	43181	50/M	Present	A	Present	A	A	A	Present	Present	Present	A	Present	A	A	A	Elevated	Present	ND	ND	Not done	IO	JP
91	murugan	44950	61/M	Present	P	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	A	Diagnostic	Diagnostic	Not done	IO	IO
92	kadarkarai	45053	37/M	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
93	ganesan	45700	36/M	Present	A	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
94	marimuthu	46977	40/M	Present	A	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	Diagnostic	Not done	PP	DU
95	murugammal	46385	38/F	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Diagnostic	AA	AA
96	esther	48865	45/F	Present	A	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	Present	Diagnostic	Diagnostic	Not done	IO	IO
97	elavarasan	52436	18/M	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
98	pooja	54236	13/F	Present	P	Present	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	ND	Not done	AA	AA
99	jeganath	54343	34/M	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
100	alagu durai	41831	62/M	Present	P	Present	A	A	A	Present	Present	Present	A	Present	A	Obliterated	A	Elevated	Present	Diagnostic	ND	Diagnostic	PP	DU
101	rama moorthi	43458	44/M	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	Elevated	Present	ND	ND	Diagnostic	AA	AP
102	vellasamy	43701	60/M	Present	A	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	ND	Not done	PP	IP
103	murugan	43620	50/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	Diagnostic	Not done	PP	DU
104	kavitha	64712	37/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
105	abdul kadhar	44624	42/M	Present	P	Present	A	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	Present	Diagnostic	Diagnostic	Diagnostic	IO	IO
106	kasi viswanathan	43617	62/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	A	Diagnostic	Diagnostic	Not done	PP	DU
107	nisar	45515	17/M	Present	P	A	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
108	poolthai	45527	45/F	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
109	murugaiah	45592	70/M	Present	P	Present	A	A	A	Present	Present	Present	A	Present	A	Obliterated	A	Elevated	Present	Diagnostic	Diagnostic	Diagnostic	PP	GP
110	maniraj	45460	46/M	Present	P	Present	A	A	A	Present	Present	Present	A	A	A	A	A	Elevated	Present	ND	Diagnostic	Diagnostic	AP	AP
111	prema	45587	40/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	Elevated	Present	ND	ND	Not done	AA	AA
112	muthu lakshmi	47371	68/F	Present	A	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	A	Diagnostic	Diagnostic	Not done	IO	IO
113	ramalingam	47560	28/M	Present	A	Present	A	A	A	No	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
114	mano ranjitham	47307	50/F	Present	P	Present	A	Present	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	ND	Diagnostic	PP	IP
115	parvathy	47303	46/F	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	ND	Not done	AA	AA
116	ponnuthai	47719	45/F	Present	P	Present	A	A	A	Present	Present	Present	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AP	AP
117	aruna	48650	47/F	Present	A	Present	A	A	A	Present	Present	A	A	A	A	A	A	Elevated	Present	ND	Diagnostic	Diagnostic	AA	AP
118	kalimuthu	48975	20/M	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
119	chithrakani	47376	25/F	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
120	sekar	47313	58/M	Present	P	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	Present	Diagnostic	ND	Diagnostic	IO	IO
121	sudalaimani	45598	29/M	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Diagnostic	AA	AA
122	muthu jeya jothi	47727	37/F	Present	A	Present	A	A	Present	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
123	panjavarnam	47290	62/F	Present	A	A	A	A	A	Present	Present	Present	A	Present	Present	A	A	Elevated	A	ND	Diagnostic	Not done	IO	JP
124	selvamani	49564	23/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	ND	Not done	AA	AA
125	susila	47540	53/F	Present	P	A	Present	Present	A	Present	Present	Present	A	Present	A	A	A	N	A	Diagnostic	Diagnostic	Diagnostic	IO	IO
126	saravanan	47588	42/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	Present	Obliterated	A	N	A	Diagnostic	ND	Not done	PP	DU
127	murugan	47352	45/M	Present	P	Present	A	A	A	Present	Present	Present	A	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Not done	PP	DU
128	gurupatham	47406	70/M	Present	P	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	N	A	Diagnostic	Diagnostic	Diagnostic	IO	IO
129	moosa	47341	58/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	Diagnostic	Not done	PP	DU
130	jeyalakshmi	47404	35/F	Present	P	A	A	A	Present	No	Present	Present	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AP	AP
131	arulraj	49200	52/M	Present	P	A	A	Present	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	ND	Not done	PP	DU
132	ramasamy	47118	75/M	Present	A	A	A	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	A	ND	Diagnostic	Diagnostic	IO	IO
133	sankaran	47412	62/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Not done	PP	GP
134	antony raj	49155	40/M	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
135	rajammal	47312	35/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	ND	Not done	AA	AA
136	ganesan	49262	50/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Not done	PP	DU
137	chithambaram	49062	35/M	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
138	muthu kannan	50586	14/M	Present	A	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
139	arumugam	50911	25/M	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	ND	Diagnostic	AA	AA
140	oorkavalan	50350	65/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	A	Diagnostic	Diagnostic	Not done	PP	DU
141	vijay	51132	21/M	Present	A	A	A	A	A	No	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Diagnostic	AA	AA
142	parameshwari	50817	35/F	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA

Sl. No.	NAME	IP NO	AGE/SEX	PAIN	VOMITING	FEVER	ABD. DIS	CONSTIPATION	DIARRHOEA	TACHYCARDIA	TENDERNESS	GUARDING	RIGIDITY	DEC. BS	FREE FLUID	LIVER DULLNESS	SHOCK	RFT	LEUCOCYTOSIS	X-RAY	USG	CT	CLINICAL DIAGNOSIS	OPERATIVE DIAGNOSIS
143	murugan	61460	45/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	ND	Diagnostic	PP	IP
144	pitchammal	49111	45/F	Present	P	A	A	A	A	Present	Present	Present	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AP	AP
145	mookammal	49017	35/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Diagnostic	AA	AA
146	ganapathy	49068	60/M	Present	P	Present	Present	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Not done	PP	GP
147	jeyalakshmi	47404	35/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Not done	AA	AA
148	ponnusamy	47335	46/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	A	Diagnostic	Diagnostic	Diagnostic	PP	DU
149	muthu duraichi	50645	50/F	Present	A	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	N	Present	ND	Diagnostic	Diagnostic	IO	IO
150	esakiammal	61300	40/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Diagnostic	AA	AP
151	rajasekar	50862	39/M	Present	P	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	ND	Not done	AA	AA
152	subbaiah	50859	50/M	Present	P	Present	A	Present	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	ND	Diagnostic	PP	GP
153	chellapandian	50825	60/M	Present	P	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	N	A	ND	Diagnostic	Not done	IO	IO
154	thayammal	52671	38/F	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Diagnostic	AA	AA
155	kirubamani	56710	15/F	Present	A	A	A	A	A	Present	Present	A	A	A	A	A	A	N	Present	ND	Diagnostic	Not done	AA	AA
156	vasanthi	59186	35/F	Present	P	Present	A	A	A	Present	Present	A	A	A	A	A	A	N	A	ND	Diagnostic	Diagnostic	AA	AA
157	selva raj	52746	65/M	Present	P	Present	A	A	A	Present	Present	Present	A	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Not done	PP	GP
158	sankar	50815	65/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	Diagnostic	Diagnostic	PP	DU
159	amudha	62942	85/F	Present	P	A	Present	Present	A	Present	Present	Present	A	Present	Present	A	A	Elevated	A	ND	ND	Diagnostic	IO	IP
160	mariammal	54425	50/F	Present	P	Present	A	A	A	Present	Present	Present	A	Present	A	Obliterated	A	N	A	Diagnostic	Diagnostic	Diagnostic	PP	GP
161	mookan	54435	59/M	Present	P	A	A	Present	A	Present	Present	Present	Present	Present	A	Obliterated	A	Elevated	Present	Diagnostic	Diagnostic	Not done	PP	DU
162	maharajan	56050	50/M	Present	P	Present	A	A	A	Present	Present	Present	Present	Present	A	Obliterated	A	N	Present	Diagnostic	Diagnostic	Diagnostic	PP	DU