"CLINICAL PROFILE OF PATIENTS WITH ACUTE ABDOMEN AND MANAGEMENT AT RURAL REFERRAL CENTRE"

By

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Dissertation Submitted to the

Sri Devaraj Urs Academy of Higher Education

And Research Centre, Tamaka, Kolar, Karnataka,

in partial fulfilment of the requirements for degree of

MASTER OF SURGERY
IN
GENERAL SURGERY

Under the guidance of

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SRI DEVARAJ URS MEDICAL COLLEGE,
TAMAKA, KOLAR

2012

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ACKNOWLEDGEMENT

First and foremost, I express my sincere and heartfelt gratitude to my respected Professor ***Dr. Madan M, M.S,*** Professor, Department of General Surgery, Sri Devaraj Urs Medical College, Kolar for his constant encouragement and valuable guidance throughout the course and the present study.

I express my sincere and heartfelt gratitude to ***Dr. A. Bhaskaran*** M.S., Prof and Head of the department of General Surgery, SDUMC, Kolar, for his constant encouragement and valuable guidance throughout the course and the present study.

With a deep sense of gratitude, I thank **Prof. Mohan Kumar, Prof. Sreeramulu P N,** for their constant encouragement and help throughout my course.

I express my sincere thanks to **Dr. Krishna Prasad, Dr Venkat Krishna, Dr. Basavarajapp, Dr. Nischal, Dr. Praveen, Dr. Mahesh M S, Dr Srikanth Reddy, Dr Mahesh Babu, Dr. Naveen, Dr. Vasanth, Dr. Prathibha, and Dr. Shashi Rekha** for their invaluable timely suggestions and support.

I acknowledge my thanks to all my teachers of the Department of Surgery for their heartfelt support at all the times.

I also thank my batch mates, **Dr. Asadulla Baig, Dr. Harsha K., and Dr. Supreeth C.S.** for helping me in many aspects.

The support I got from my seniors **Dr. Harish Kakilaya, Dr. Naveen TK, Dr. Seema, Dr. Nikhil Shetty, Dr. Jyotindar** cannot be expressed in words.

I would like to thank my juniors **Dr. Pavan, Dr. Iram, Dr. Sathiadev, Dr. Ananth Raju, and Dr. Vijay Agrawal** for their constant support.

I am infinitely obliged to my beloved Parents **Dr. Madanlal Chunilal Mandhane** and **Dr. Jayashree Madanlal Mandhane** and brother **Dr. Hrishikesh** who are always my pillars of strength. I am truly indebted to my dear wife **Dr. Sneha** for her unwavering support, understanding and her untiring patience.

I am also thankful to all nursing staff, **OT Staff, Paramedical staff, Mr. Aanand and Mr. Krishna Murthy** for their invaluable help.

Last but not the least, I would also like to thank all the **patients** without whom, this study would not have been possible.

I would like to thank everyone who helped me in my study for all their help throughout the preparation of this dissertation.

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LIST OF ABBREVIATIONS USED

Ac.	—	Acute
Alb	—	Albumin
BUN	—	Blood Urea Nitrogen
CBD	—	Common Bile Duct
CT	—	Computerized Tomography
D.U	-	Duodenum
GE	—	Gastroenteritis
GIT	—	Gastrointestinal tract
H2	—	Histamine-2
IV	—	Intravenous
IVP	—	Intravenous pyelogram
LDH	—	Lactate dehydrogenase
LFT	—	Liver function test
Mic	—	Microscopy
MRI	—	Magnetic Resonance Imaging
PID	—	Pelvic inflammatory disease
PR	—	Per Rectal
PV	—	Per Vaginal
RIF	—	Right iliac fossa
SGOT	—	Serum glutamate Oxaloacetate transferase (AST)
SGPT	—	Serum glutamate Pyruvate transferase (ALT)
Sug	—	Sugar
USG	—	Ultrasound
WBC	—	White Blood Cells

ABSTRACT

BACKGROUND

Acute abdomen means, the patient complains of acute abdominal symptoms that suggest a disease, which definitely or possibly threatens life and may or may not demand immediate operative interference. The diagnosis and management of acute abdomen forms a large part of routine duties of a general surgeon throughout his career.

OBJECTIVES **t**o study the incidence of non-traumatic acute abdominal pain, to analyse the nature & presentation acute abdominal emergencies and to study the mortality and morbidity rate in the analysed cases.

METHODS

The study was conducted in R. L. Jalappa Hospital and research centre attached to Sri Devaraj Urs Medical College, from December 2009 to April 2011. 100 cases with acute abdominal pain have been studied. Patients who underwent surgery have been only included in this study to come to correct diagnosis, whereas acute abdomen of traumatic origin, in paediatric age group, due to gynaecological or medical cause also recurrent pain abdomen was excluded. Pre-operative history of all acute abdominal emergencies has been taken to arrive at pre-operative diagnosis. In all the cases, operative findings and post-operative diagnosis were recorded.

RESULTS

Acute abdomen was more common in 2^{nd}, 3^{rd} and 4^{th} decade of life and in males. Hollow viscus perforation forms the commonest cause of acute abdomen. Acute appendicitis being the 2^{nd} and acute calculus cholecystitis forms the 3^{rd} commonest cause of acute abdomen.

CONCLUSION

In our study acute abdomen was found more commonly 2^{nd} to 4^{th} decade of life

with male : female :: 65 : 35. Most common presenting symptom was pain abdomen. The commonest cause being Hollow viscus perforation. Commonest position of appendix was retrocaecal and pathological type was non-perforated inflamed appendix, for which emergency appendectomy was done and mortality was nil. Acute appendicitis 2nd commonest and intestinal obstruction was 3rd commonest cause of acute abdomen.

KEY WORDS

Perforation, Acute appendicitis, Appendectomy, Acute cholecystitis, Leucocytosis, Neutrophilia.

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"Actual operative skill cannot be gained by observation, any more than skill in playing the violin can be had by hearing and seeing a virtuoso performing on that instrument."

Allen O. Whipple (1881–1963)

INTRODUCTION

The term acute abdomen designates symptoms and signs of intra-abdominal disease usually treated best by surgical operation. Many diseases, some of which do not require surgical treatment, produce abdominal pain, so the evaluation of patients with abdominal pain must be methodical and careful. The proper management of patients with acute abdominal pain requires a timely decision about the need for surgical operation. This decision requires evaluation of the patient's history and physical findings, laboratory data, and imaging tests. The syndrome of acute abdominal pain generates a large number of hospital visits and may affect the very young, the very old, either sex, and all socioeconomic groups. All patients with abdominal pain should undergo evaluation to establish a diagnosis so that timely treatment can minimize morbidity and mortality.

The first principle is that the necessity of making a serious and thorough attempt at diagnosis, usually predominantly by means of the history and physical examination. Abdominal pain is one of the most common conditions that call for prompt diagnosis and treatment. Usually, though by no means always, other symptoms accompany the pain, but in most cases of acute abdominal disease pain is the main symptom and complaint. The very terms "acute abdomen" and abdominal emergency", which are constantly applied to such cases, signify the need for prompt diagnosis and early treatment, by no means always surgical. The term "acute abdomen" should never be equated with the invariable need for operation.

The abdomen has been referred to as Pandora's magic box. Very often an accurate diagnosis cannot be made without surgery and many wonders are revealed on opening the abdomen. So it is often the last court of appeal in investigating abdominal cases.

The general rule can be laid down that the majority of severe abdominal pains that ensue in patients who have been previously well, and that last as long as six hours, are caused by conditions of surgical import.

AIMS AND OBJECTIVES

- To study the incidence of non-traumatic, acute abdominal emergencies.
- To analyze the nature & presentation of the non-traumatic acute abdominal emergencies treated in surgical units.
- To study the mortality and morbidity rate in the analyzed cases.

REVIEW OF LITERATURE

HISTORICAL REVIEW

An acute abdominal problem has been recognized since the era of Hippocretes. A variety of terms were given to acute abdominal problems among which iliae passion Ileus were familiar to celsus and Hippocretes, iliae passion that can be considered synonymous with acute abdomen.

Hippocrates advocates deflation of intestines and the use of enemas in intestinal obstraction.

Paraxegoras is believed to have performed the first record exterostomy.

As early as on 1556 operation was recommended, by Pierre franeo for inguinal hernia. He proposed surgical intervention in the treatment of strangulated inguinal hernia. It was not until 1836 that **Jaharn Friedrich dieffenbach** reported the successful account of resection of gangrenous bowel in a strangulated Hernia.

In 1710 **Lirarature** suggested that the colon should be opened when obstructed.

Jean Julima of Amasset developed the use of lumbar colostomy.

Rowlinson in 1727 gave the accurate description of signs and symptoms of perforated ulcer. Surgery for treatment of perforated ulcer was undertaken somewhere during the year 1883. The earliest published successful intervention for the perforated ulcer is that by **Hensher** in 1891 and **Krige** in 1892.

A progressive improvement in the mortality rate has been reported by adopting surgical line of treatment for acute duodenal ulcer perforation with introduction of antibiotics and relaxant anaesthesia mortality rate fall to 4% in 1953 to 2% in 1960.

Acute inflammation of pancreas was not recognized until 1842.

ANATOMICAL CONSIDERATIONS

The peritoneal cavity is a potential space that has a lining consisting of a mesothelial layer, the sub serosal layer. The peritoneal cavity divided into general peritoneal cavity and lesser omental bursa. They communicate through the foramen Winslow.

For the purpose of description anterior abdominal wall is divided into 9 regions, by 2 horizontal and 2 vertical lines. The use of quadrants for topographic location of pain. Upper horizontal or transpyloric line, runs middling between umbilicus and Xiphisternum lower horizontal line joins tubercles of iliae creates about 2" behind the iliac spines vertical lines drawn through midpoint of iliac spine and symphysis pubis.

Table 1: The division of abdomen in 9 regions

1.	Rt. Hypochondrium		Lt. Hypochondrium
2.	Epigastric		Rt. Lumbar
3.	Umbilical region		Lt. Lumbar
4.	Rt. Iliac region		Hypogastrium
5.	Lt. Iliac region		

Tenderness, or masses is helpful for the clinician but it is important to recall that this external division has little anatomic basis.

The anatomical relationships of abdominal viscera have influence upon the localization signs and symptoms. The visceral attachments influence the localization and spread of blood, purulent material and fluid within the cavity.

The peritoneal peritoneum with invests the abdominal wall diaphragm and the pelvic surfaces. In innervated by T5-11 spinal nerves and phrenic nerve. Pain sensation from the abdomen radiates through spinal and autonomic nerves.

Pain full stimuli from structures derived from fore gut refers to epigastrium because these structures innervated by splanchnic nerves. Mid gut pain felt in the pericumbilical regions, hindgut pain is a suprapubic region, pain is not well localized with visceral peritoneum but if disease process involves partial peritoneum precise localization occurs because of spinal nerves innervation.

ANATOMY AND PHYSIOLOGY

DEVELOPMENT ANATOMY[1, 3]

The developmental anatomy of the abdominal cavity and of its viscera determines normal structure and influences the pathogenesis and clinical manifestations of most abdominal diseases. Peritoneal attachments and visceral sensory innervation are particularly important to the evaluation of acute abdominal disease. After the 3rd week of fetal development, the primitive gut divides into foregut, midgut, and hindgut. The superior mesenteric artery supplies the midgut (the fourth portion of the duodenum to the midtransverse colon). The foregut includes the pharynx, the esophagus, the stomach, and the proximal duodenum, whereas the hindgut comprises the distal colon and the rectum. The afferent fibers accompanying the vascular supply provide sensory innervation to the bowel and associated visceral peritoneum.

Thus, disease in the proximal duodenum (foregut) stimulates celiac axis afferents to produce epigastric pain. Stimuli in the caecum or appendix (midgut) activate afferent nerves accompanying the superior mesenteric artery to cause periumbilical pain, and distal colon disease induces inferior mesenteric artery afferent fibers to cause suprapubic pain. The phrenic nerve and afferent fibers in C3, C4, and CS dermatomes accompanying the phrenic arteries innervate the diaphragmatic musculature and the peritoneum on its undersurface. Stimuli to the diaphragm therefore cause referred shoulder pain. The parietal peritoneum, abdominal wall, and retroperitoneal soft tissue receive somatic innervation corresponding to the segmental nerve roots.

The richly innervated parietal peritoneum is particularly sensitive. Parietal peritoneal surfaces sharply localize painful stimuli to the site of the stimulus. When visceral inflammation irritates the parietal peritoneal surface, localization of pain occurs. Maneuvers that exacerbate this irritation then intensify the pain. The many "peritoneal signs" useful in the clinical diagnosis of the acute abdomen originate in this fashion. Dual-sensory innervation of the abdominal cavity by both visceral afferents and somatic nerves produces clinical pain patterns that aid in diagnosis. For example, the pain of acute appendicitis originates with poorly localized periumbilical pain progressing to sharply localized right lower quadrant pain when the inflammation involves the parietal peritoneal surface.

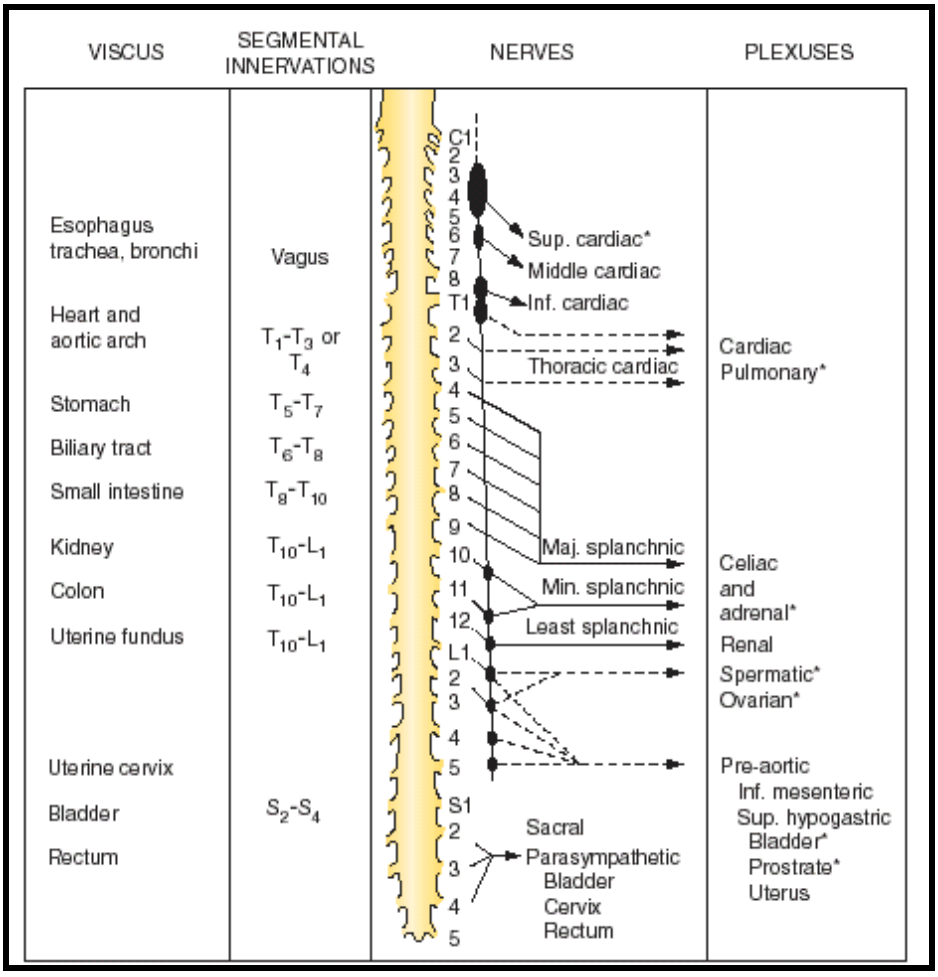


Fig 1: Sensory Innervation of the viscera[2, 3]

Peripheral nerves mediate sharp, sudden, well-localized pain. Sensory afferents involved with intraperitoneal abdominal pain transmit dull, sickening, poorly localized pain of more gradual onset and protracted duration. The vagus nerve does not transmit pain from the gut. Small, unnamed sympathetic afferent nerves transmit pain from the esophagus to the spinal cord. Afferent nerves from the liver capsule, the hepatic ligaments, the central portion of the diaphragm, the splenic capsule, and the pericardium enter the central nervous system from C3 to C5. The spinal cord from T6 to T9 receives pain fibers from the periphery of the diaphragm, the gallbladder and the stomach, the pancreas, and the small intestine. Pain fibers from the colon, appendix, and pelvis viscera enter the central nervous system at the 10th and 11th thoracic segments. The sigmoid colon, rectum, renal pelvis and capsule, ureter, and testes pain fibers enter the central nervous system at T11 and L1. The bladder and the rectosigmoid colon send afferent nerves to the spinal cord from S2 to S4.

Cutting, tearing, crushing, or burning usually does not produce pain in the abdominal viscera. However, stretching or distention of the peritoneum produces pain. Bacterial or chemical peritoneal inflammation produces visceral pain, as does ischemia. Cancer can cause intraabdominal pain by invading sensory nerves. Abdominal pain may be visceral, parietal, or referred. Visceral pain is dull and poorly localized, usually in the epigastrium, periumbilical region, or suprapubic region, and it usually does not lateralize well. Patients with visceral pain may also experience sweating, restlessness, and nausea. The parietal or somatic pain associated with intraabdominal disorders may be more intense and precisely localized. Referred pain is perceived at a site distant from the source of stimulus. For example, irritation of the diaphragm may produce pain in the shoulder. Disease in the bile duct or gallbladder may produce shoulder pain. Distention of the small bowel can produce pain referred

to the back.

During the 5th week of fetal development, the bowel outgrows the peritoneal cavity, protrudes through the base of the umbilical cord, and undergoes a 180-degree counterclockwise rotation. During this process, the bowel remains outside the peritoneal cavity until approximately the 10th week, when it returns to the abdomen, and an additional 90-degree counterclockwise rotation occurs. This embryologic rotation places the viscera in their adult positions, and subsequent fusion of the portions of the colonic and duodenal mesenteries with the mesothelium of the posterior abdomen forms the normal ultimate peritoneal attachments. Knowledge of these attachments is clinically important during me evaluation of patients with the acute abdomen because of variation in the exact position of the viscera (e.g., pelvic or retrocecal appendix) and the compartmentalization of the abdomen by mesenteric attachments. The latter, for example, may channel duodenal or gastric contents from the site of a perforated ulcer to the right lower quadrant.

PERITONEAL PATHOPHYSIOLOGY

Mesothelial cells cover the visceral and parietal peritoneal surfaces. Openings into radially arranged lymphatics penetrate the diaphragmatic peritoneal surface.[4] Introduction of bacteria into the peritoneal cavity can cause an outpouring of fluid from me peritoneal membrane. This loss of fluid from me circulation may lead to dehydration and may produce the clinical signs of resting or orthostatic hypotension and tachycardia. Diaphragmatic lymphatics are the major route for the clearance of bacteria and cellular debris from the abdominal cavity. This process leads to an intraperitoneal circulation of fluid toward the subdiaphragmatic regions bilaterally. Fluid not cleared in this fashion tends to accumulate in me deep end of the

pelvis. Thus, subdiaphragmatic, subhepatic, paracolic, or pelvic fluid collections can accompany visceral perforation. The peritoneal surfaces localize bacteria and the products of inflammation. The peritoneum responds to inflammation by increased blood flow, increased permeability, and the formation of a fibrinous exudate on its surface. The bowel also responds to inflammation with localized or generalized paralysis. The fibrinous surface thus created, aided by decreased intestinal movement, causes adherence between bowel and omentum and effectively walls off inflammation. An abscess may produce sharply localized pain with normal bowel sounds and gastrointestinal function, whereas a disseminated process, such as a perforated ulcer, produces generalized abdominal pain with a quiet abdomen. Peritonitis may affect the entire abdominal cavity or a portion of the visceral or parietal peritoneum. Transudation can produce an increase in the peritoneal fluid, which is rich in protein and leukocytes that facilitate the formation of fibrin on peritoneal surfaces.

Peritonitis denotes peritoneal inflammation from any cause. Primary or spontaneous peritonitis can occur as a diffuse bacterial infection without an obvious intra. abdominal source of contamination. Primary peritonitis, most commonly caused by Pneumococcus or hemolytic Streptococcus, occurs more commonly in children than in adults. However, adults with ascites and cirrhosis are susceptible to spontaneous peritonitis resulting from Eschericia coli and Klebsiella.

The more common secondary peritonitis results from perforation, infection, or gangrene of an intraabdominal organ,[4] usually of the gastrointestinal tract. Gastrointestinal secretions, pancreatic secretions, bile, blood, urine, and meconium cause chemical peritonitis when in contact with the peritoneum. A common form of chemical peritonitis follows perforation of a peptic ulcer. Bile peritonitis may result

from perforation of the gallbladder or leakage from the bile ducts. Ordinarily, slow bleeding into the abdominal cavity produces relatively few signs of inflammation; the addition of bacteria to blood produces suppuration. The sickest postoperative patients may have tertiary peritonitis that kills 30% to 64% of affected patients. The syndrome of poorly localized intra-abdominal infection, an altered microbial flora, progressive organ dysfunction, and high mortality define tertiary peritonitis.

Peritonitis causes abdominal pain, either generalized or localized, depending on the disease. Appendicitis usually causes localized pain. Perforated peptic ulcer usually produces generalized abdominal pain. Acute cholecystitis causes right upper quadrant pain referred to the right scapula or shoulder. Physical findings of patients with peritonitis are abdominal tenderness, guarding, and rebound tenderness.

CAUSES OF ACUTE ABDOMEN

I. EXTRAABDOMINAL CAUSES MIMIKING ACUTE ABDOMEN [2, 5]

a. Abdominal wall, intramuscular hematoma

b. Cardiopulmonary

1. Pneumonia
2. Empyema
3. Myocardial Ischaemia
4. Acute rheumatic heart disease

c. Blood

1. Leukemia
2. Sickle cell anaemia

d. Neurogenic

1. Spinal cord tumours
2. Ostemyelitis of the spine
3. Tabes Dorsalis
4. Herpes Zoster
5. Abdominal epilepsy

e. Metabolic

1. Uremia
2. Diabetic Acidosis
3. Porphyria
4. Addisonian crisis

f. Toxic

1. Venoms
2. Drugs

3. Insect bites

4. Led Poisoning

5. Insect bites

g. Pyogenic

h. Traumatic

1. Perforating injury

2. Blunt injury

II. CONGENITAL CAUSES

1. Congenital pyloric atresia or stenosis
2. Duodenal atresia stenosis
3. Ampulla of Vatar stenosis
4. Persistence of ventral mesentery or band on duodenum
5. Congenital duodenal and obstraction or stenosis
6. Meckel's diverticulum
7. Stenosis or atresia of anus and rectum
8. Omphalomesentric duct
9. Malrotation of midgut
10. Tumours
11. Intussuception
12. Primary peritonitis
13. All types of hernias
14. Abnormal meconium
    1. Meconium ileus
    2. Meconium play syndrome

III. INTRAABDOMINAL CAUSES OF PAIN

A. Inflammation

a. Solid organs

1. Pancreatitis

2. Hepatitis

3. Hepatic abscesses

4. Splenic abscesses

B. Hollow organs

1. Appendicitis

2. Cholecystitis

3. Regional ileitis

4. Diverticulitis

C. Peritoneum

1. Bacterial peritonitis

a. Primary peritonitis

1. Pneumococcal

2. Streptococcus

3. TB

b. Perforated hollow Viscous

c. Non-bacterial and chemical

1. Ruptured Ovarian Cyst

2. Gall Bladder perforation

3. Peptic Ulcer

D. Mesentry

1. Lymphadinitis

2. Twist

3. Ischemia

E. Pelvic Organs

1. Pelvic Inflammatory Disease

2. Pelvic Endometriosis

3. Twisted Ovaries

4. Twisted Fibroid

IV. MECHANICAL OBSTRUCTION

A. Adhesions

a. Hollow Intestinal organs

1. Adhesions

2. Intussusception

3. Volvusus

4. Tumours

5. Hernia

B. Pelvic

1. Ovarian Cyst

2. Degeneration of Fibroid

3. Pregnancy Ectopic

C. Mesentry

1. Omental torsion

D. Solid Viscera

1. Acute Spleenomegaly

2. Acute Hepatomegaly

V. VASCULAR

A. Retroperitoneal bleeding

1. Dissection of Aorta

2. Rupture of Aneurysm

3. Pancreatitis

B. Intraperitoneal Bleeding

1. Ruptured Liver

2. Ruptured Spleen

3. Ruptured Mesentry

4. Ruptured Ectopic Pregnancy

5. Aneurysm

C. Ischaemia

1. Infarction

2. Embolism

3. Thrombosis

4. TAO

VI. EXTRAPERITONEAL

1. Nephritis

2. Pyelitis

3. Perinephric Abscess

4. Epididymitis

5. Vasculitis Seminal

6. Porostatitis
7. Urethral obstruction
    1. Calculus
    2. Tumours

Causes of Haemoperitoneum

Spontaneous

1. Haemorrhagic diseases
2. Haemorrhage from Tumours
3. Peptic Ulcer
4. Mesentric Thrombosis, Strangulation
5. Pancreatitis
6. Torsion of Omentum
7. Rupture of Mesentric arteries
8. Rupture of Aneurysm
9. Rupture of Ovarian Cyst

NEUROLOGIC UNDERPINNINGS

The location of pain is critical to aiding in making the correct diagnosis, and a physician must be versed in its interpretation.[6, 7] This requires a fundamental understanding of the three basic forms of pain: visceral, somatic, al referred.

Table 2: Possible Causes of Pain by Location

Location of Pain	**Associated Diseases**
Right upper quadrant (liver, kidney, gallbladder)	Acute cholecystitis, biliary colic, acute hepatitis, duodenal ulcer, right lower lobe pneumonia
Right lower quadrant (ascending colon, appendix, ovary, fallopian tube)	Appendicitis, caecal diverticulitis, ectopic pregnancy, tubo-ovarian abscess, ruptured ovarian cyst, ovarian torsion
Left upper quadrant (pancreas, spleen, kidney)	Gastritis, acute pancreatitis, splenic pathology, left lower lobe pneumonia
Left lower quadrant (sigmoid and descending colon, ovary, fallopian tube)	Diverticulitis, ectopic pregnancy, tubo-ovarian abscess, ruptured ovarian cyst, ovarian torsion
Midline or periumbilical	Appendicitis (early), gastroenteritis, mesenteric Lymphadinitis, myocardial ischemia or infarction, pancreatitis
Flank	Abdominal aortic aneurysm, renal colic, pyelonephritis
Front to back	Acute pancreatitis, ruptured abdominal aortic aneurysm, retrocecal appendicitis, posterior duodenal ulcer
Suprapubic or lower abdominal	Ectopic pregnancy, Mittelschmerz, ruptured ovarian cyst, pelvic inflammatory disease, endometriosis, urinary tract infection

HISTORY

The common symptoms in acute abdomen are:[7,8]

1. Nausea
2. Constipation
3. Obstipation
4. Distension of abdomen
5. Altered bowel habits
6. Pain abdomen
7. Bladder functions
8. Menstrual History
9. Drug History
10. Previous ingestion of food or drinks

Abdominal pain

This is the commonest and the invariable symptom of a patient with acute abdomen.

Characteristics of pain

• Site

• Time and mode of onset Severity

• Nature

• Progression

• Duration Exacerbating/relieving factors Radiation

Site of Pain

The site of abdominal pain is perhaps the most valuable pointer to the

underlying diagnosis. In order to describe the site of pain, the abdomen is traditionally either divided into quarters or ninths.

Nature of Pain

There are two main pathological mechanisms in the development of abdominal pain; obstruction and inflammation. Inflammation produces a constant pain made worse by local or general disturbance, and pain, which is made worse by movement or coughing, suggests inflammation of the parietal peritoneum. In this situation the patient will often be/ seen to tie very still in order not to exacerbate the pain.

Obstruction of a muscular viscus produces a-colicky pain. This pain comes and goes in 'spasm', often only lasting a few minutes at a time but returning at frequent intervals. It may be described as 'gripping' in nature, and between spasms the patient is usually pain free. The pain itself is severe and may be helped by moving around or drawing the knees up towards the chest. Underlying inflammation must be suspected when a colicky pain does not disappear between spasms, or becomes continuous. In the case of intestinal obstruction this might mean strangulation and urgent surgery is required.

Radiation is the site to which the pain extends while the initial pain persists. When a pain radiates, it signifies that other structures are becoming involved. For example, pain from a duodenal ulcer may radiate through to the back, indicating that inflammation through the wall of the duodenum to involve structures of the posterior abdominal wall, such as the pancreas, has occurred. Ureteric pain radiates to the tip of the penis in men and to the labium majoris in women.

Onset of pain

The onset of pain can be sudden or gradual. Typically, pain from a perforation

is sudden and that from inflammation is gradual. Patients with the former can usually remember exactly what they were doing at the time of onset, whereas in the latter localization of time is more difficult

Severity of pain

A patient's description of the severity of pain is very subjective. Every individual has a different reaction to pain, and this is often more reflective of the patient's personality than of the underlying pathology. A better indication is to assess the affect of the pain on the patient's life. For example, did they call their GP? Were they unable to attend work? Did the pain interfere with their sleep? Furthermore, it is often useful to ask the patients to give the severity of the pain as a score on a numerical or pictorial scale.

Progress of pain

Once a pain has occurred it may remain exactly the same, gradually improve or worsen, or may fluctuate.

Movement of pain

It is also useful to note whether the pain moves. The classic example of this is acute appendicitis, which starts as a vague central 'referred' pain and then moves to the right iliac fossa as the adjacent parietal peritoneum becomes inflamed.

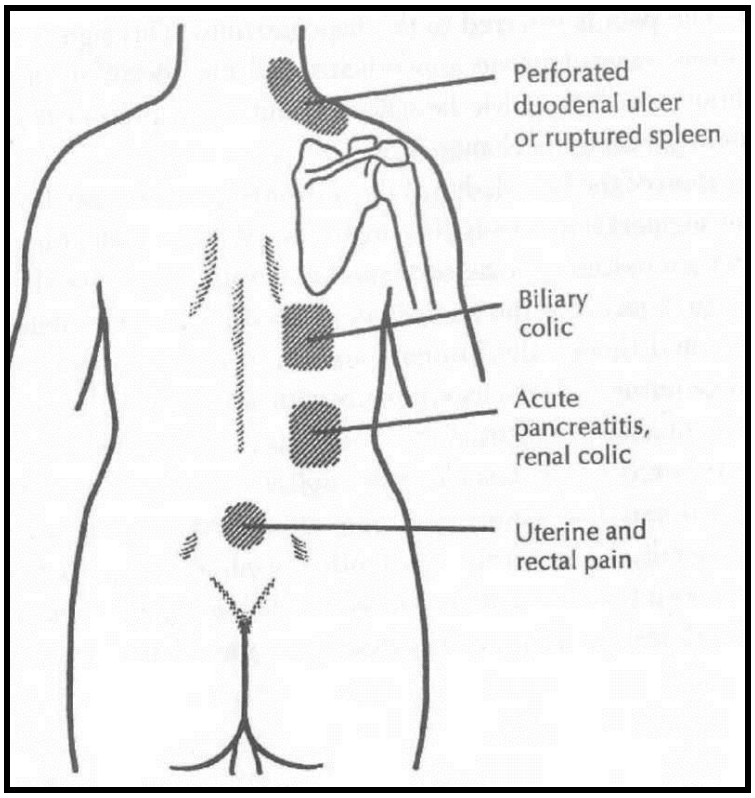


Fig 2: The sites on the posterior surface of the body to which pain is referred in acute abdominal conditions

Vomiting

Pain in acute abdomen usually precedes vomiting. In medical conditions reverse is true. When sufficiently stimulated by secondary afferent visceral fibers. The medullary vomiting centres activates efferent fibers 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, renal colic or upper small bowel obstruction. Vomiting occurs soon after 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.

Passage of vomitus upwards from stomach and intestine results from a combination of active reversed peristalsis and simple overflow. Therefore they vomit first gastric contents and partially digested food, and than bile stained fluid, and later dark coloured foul smelling contents referred to as faecal. But is seldom for colonic obstruction.

Vomiting is a reflex occurs in early stages of acute abdomen associated with severe pain, distension of hollow viscus, increased tension, in mesenteries, local or generalized inflammation of peritoneal or retroperitoneal tissue. Toxic vomiting occurs with development of gross infective conditions.

In acute appendicitis initial vomiting is reflex, later due to ileus, and rarely to organic obstraction. In intestinal obstruction vomiting first is reflex 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 conditions are not associated with vomiting. Vomiting is not an important symptom of perforated ulcer although nausea and retching may occur after perforation vomiting is infrequent.

Vomiting may not occur in cases of large bowel obstruction, or acute appendicitis. The later 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.

Nausea

This symptom of nausea is common in any acute illness. In its milder from it is

merely a dis-taste for food. This symptom like that of vomiting is of no specific diagnostic value. It usually indicates some derangement in gastro intestinal function. Usually founding acute illness with distension of abdomen or local. Visceral dilatation. Extremely nausea usually occurs with severe loss of blood.

Distension of abdomen

This is a complaint of patient with peritonitis and of intestinal obstruction.

Bowel Function

Diarrhoea may suggest GE in a patient with abdominal pain appendicitis may be present. In some patients obstipation with abdominal distension supports the diagnosis of large bowel obstruction. However in patients with pancreatitis cholecystitis and inflammatory problems of abdominal viscera ileus may develop with distension and failure to pass flatus and stools. The character of stools must be noted. The presence of blood dark or fresh tarry stools and abnormalities should be sought for as well as presence diarrhoea enquire concerning.

Tenesmus and decreased caliber of stools should be made in patients whom a lesion of large bowel is suspected. In intussusception red current jelly stools is of significant.

Previous Ingestion of Food or Drink

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

Bladder Function

Presence of dysuria frequency, difficulty or retention of urine suggests pathology is in genet urinary system.

Similar symptoms may occur in P.I.D. due to contiguity of bladder. Patients with renal stone give history of previous attacks, presence of sand or gravel in urine haematuria.

Menstrual History

The relationship of menstruation to acute abdominal symptom may be of diagnostic importance. It is crucial to diagnosis of ectopic pregnancy endometriosis.

Drug History

It is important not only in peri-operative management but also may offer a diagnostic value. Oral contraceptives in formation of benign hepatic adenomas and mesentric arterial infarction. Steroids may mask even in advanced peritonitis.

Travel History

May rise the possibility of amoebic liver abscess or hydatic cyst of spleen, T.B., Salmonella or dysentery.

GENERAL PHYSICAL EXAMINATION [7, 8]

General Inspection

This is the right of individual impression obtained by physician at patient it is subjective nature. If he is ambulatory and has more serious abdominal lesion he will probably press his hands on most pain full area.

Examination of face

The face reliable mirror of acute process i.e., in abdominal cavity.

Deeply set sunken eyes tired suffering look, pointed nose, tightly stretched skin over bones, dry chipped lips breathing and dilation of the alae nasi occurs only in some cases, like, progressed peritonitis cachexia.

Dejected expression, half closed eyelids to shrunken eyes cyanotic lips acute shock.

Examination of tongue

Brownish Black Coffee ground Greenish Yellow - after high bile vomiting. Dull, dry, rough and coated in peritonitis.

Observation of breath and respiration

Smell of breath, feculent in ileus, sweetish putrid in pyloric stenosis. Metallic in Bleeding patient.

Rapid breathing, fever, anemia shock, breathing dilated alae nasi in Diffuse peritonitis, anemia, Kussmaul's breathing in acidosis.

Examination of heart and pulse

Acute abdominal conditions frequently directly consequence of a heart lesion. Rapid pulse low B.P. in intraabdominal bleeding shock etc.

Examination of Chest

Empyema, heart dilatation, bronchitis and other conditions presents as acute abdomen.

Pulse

Moderate tachycardia with or without increased temperature denotes some inflammatory pathology. Tachycardia, with low volume pulse; with cold clammy skin and perspiration denotes hypovolemia and septicaemia.

Colour

Pallor seen in anemia hypovolemia, shock and severe pain. Flushed appearance in Hyperpyrexia. Cynotic or Grayish complexion pancreatitis.

Blood pressure recording is of great value in cases of shock, haemorrhage fall in blood pressure in acute strangulation of the intestine indicates more serious prognosis.

Temperature

An inflammatory process is usually associated with pyrexia but absence of raise in temperature does not exclude severe acute intraperitoneal inflammation. Sub normal temperature in advanced peritonitis. Temperature chills in UTI salpingitis. In aged temperature reaction may be negligible.

Abdominal Examination

Inspection:

The abdomen should be carefully inspected before palpation. Surgical scar noted on the abdomen may have 0 bearing on acute abdomen. A previous operation may carry a risk or specific complication for example, stomal ulcer after G.J. for peptic ulcer.

An operation may have already been done for a condition known to recur like perforated peptic ulcer Volvulus etc. Discoloration or bruising of abdominal wall may be of importance. Ecchymatic discoloration of umbilicus or flanks are often seen in haemorrhagic pancreatitis.

Local or symmetrical distension may indicate enlargement of a particular viscera Emptiness in one part may be recognized in intussusception. A scaphoid contracted in perforated D.U visible peristalsis suggest obstruction. The direction may occasionally of significant movements on respiration are restricted in peritonitis.

The hernial orifices should be examined at umbilicus; inguinal canal and femoral region in males, testis and scrotum should be inspected cremasteric muscle spasm, which may occur, in intraperitoneal inflammation.

Palpation

Palpation is performed with the patient resting in a comfortable supine position.

Pressure should be slight when palpation is started and degree of muscular resistance should be assessed palpation is accomplished with fingertips and fingers fully extended.

It is the utmost importance of differentiate voluntary from involuntary muscular rigidity. While involuntary muscular rigidity or muscle rigidity. While involuntary muscular rigidity or muscle guard indicates underlying parietal peritonitis and is one which the clinician is very much looking for, the voluntary muscular rigidity is simple rigidity of the abdominal musculature brought about by the patient himself due to fear of being hurt and resentment due to exposure of the abdomen.

Method of eliciting involuntary muscle guard is to use both hands during palpation one above the other. The hand in contact with abdominal wall remains passive and wholly utilized to feel the condition of the abdominal musculature while the hand above is used to exert a slight and steady pressure to assist the hand below for better palpation. Patient's confidence must be gained by being gentle in your behaviour. You may ask the patient to open his mouth and breathe deeply in and out. Unlike the involuntary muscle guard, the voluntary muscular rigidity will disappear during expiration and helps the clinician to palpate in a better way. The muscle guard usually corresponds to the area of tenderness.

Tenderness

Its extent and severity are determined first by one finger palpation beginning away from area of tenderness and gradually advancing towards it.

When patient raises his or her head from the bed the abdominal muscles will be tensed. Tenderness persists in abdominal wall condition whereas deeper peritoneal pain is lessened, hyperaethesia may be demonstrated in abdominal wall disorders or localized peritonitis.

Abdominal Masses

Are usually detected by deep palpation, sometimes a swelling may be lost on deeper palpation as it increases muscle guarding.

Deeper masses may be adherent to posterior or lateral abdominal wall and often partially walled of by omentum and small bowel pain elicited Ex. , pancreatic phlegm on, ruptured aortic aneurysm.

Percussion

Percussion serves several purposes. Tenderness on percussion is a kin to eliciting Rebound tenderness. Both reflect peritoneal irritation and parietal pain.

It is useful in the following ways:

1. A local mass may be delineated for example appendicular abscess or mass in R.I.F.
2. Obliteration of liver dullness. - Right midaxillary line is percussed from above downwards. The percussion note will be resonant in the upper part of the mid-axillary line. At the upped border of the liver the resonant note is replaced by the dull note. If the liver dullness is replaced by a resonant note it indicates presence of free gas under the diaphragm as occurs in perforation of the gastro-intestinal tract. It must be remembered that absence of this sign does not exclude perforation since this sign will only be present when there is sufficient leakage of air.

   Fallacy - Considerable distension of the gut and emphysema of the lung may obliterate the area of normal liver dullness.
3. Tympany near midline in a distended abdomen denotes air trapped within distended bowel loops.
4. A tumour may be recognized.

5. Enlargement of liver spleen and kidney can be made out.
6. Free peritoneal fluid may be made out by shifting dullness. Ex., in peritonitis and intra-peritoneal haemorrhage.
7. Free fluid and a local mass related to a particular viscus may indicate intra-peritoneal blood and local haematoma formation around that viscus. For ex. , ruptured spleen.

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

Peristaltic rushes synchronous with colic are heard in a mid 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 fulminant ulcerative colitis.

The 'silent abdomen' is a pathognomonic feature of diffuse periotonitis. Even localized absence of peristaltic sound will be evident around acute inflammation of the organ concerned. To the contrary a 'noisy abdomen' is a feature of acute intestinal obstruction. Normal intestinal sound is heard as clicks and gurgles but in intestinal obstruction distinct metallic tinkles or borborygmi can be heard. In case of peritonitis or paralytic ileus when the intestinal sounds are absent peculiar respiratory and cardiac sounds may become audible.

Except for these more extreme patterns many auscultatory variants heart in paralytic ileus and other conditions render them largely useless for specific diagnosis.

Early mesentric arterial occlusion is associated with increased peristaltic

activity and bowel sounds are loud and active within few hours. The ischaemic bowel looses its peristaltic function.

Table 3: Important Signs in Patients with Abdominal Pain [6, 7, 8]

Sign	Finding	Association
Cullen's sign	Bluish periumbilical discoloration	Retroperitoneal hemorrhage (hemorrhagic pancreatitis, abdominal aortic aneurysm rupture)
Kehr's sign	Severe left shoulder pain	Splenic rupture Ectopic pregnancy rupture
McBurney's sign	Tenderness located 2/3 distance from anterior iliac spine to umbilicus on right side	Appendicitis
Murphy's sign	Abrupt interruption of inspiration on palpation of right upper quadrant	Acute cholecystitis
Iliopsoas sign	Hyperextension of right hip Causing abdominal pain	Appendicitis
Obturator's sign	Internal rotation of flexed right hip causing abdominal pain	Appendicitis
Grey-Turner's sign	Discoloration of the flank	Retroperitoneal hemorrhage (hemorrhagic pancreatitis, abdominal aortic aneurysm rupture).
Chandelier sign	Manipulation of cervix causes patient to lift buttocks off table	Pelvic inflammatory disease
Rovsing's sign	Right lower quadrant pain with palpation of the left lower quadrant	Appendicitis

SPECIAL METHODS OF EXAMINATION

Diagnostic Enema

The enema is used in, assessing the probability of complete obstraction. The first enema may produce flatus and only a small amount of flatus and fecal matter and second anemia produces only a small amount of flatus.

In contrast if colon is obstructed completely some faeces and little flatus may be produced by first anemia and nothing is likely to result from second anemia.

Diagnostic Aspiration [6, 9]

A 22 number gauge spinal needle is inserted through a kin wheal of local anaesthetic a point is chosen about 3 cms above and 3 cms medial to anterior superior iliac spine. This point is below the umbilicus and near the outer border of rectus. The patient is tilted towards the needle any fluid present is aspirated the procedure is repeated on the other side Injury to bowel is negligible risk. Results were indicated as positive when abnormal fluid (clear, turbid, purulent, bloody, serosanguinous, bile-stained, urine etc.) was aspirated. Accurate diagnosis was made in 76.47% in non-traumatic acute abdomen. High incidence of accurate results was obtained in gastroduodenal perforations (92%).

Laparoscopy [6]

It is indicated only when clinical picture is uncertain or the diagnosis abs cure in traumatic or non-traumatic acute abdomen. It is not done when surgery is mandatory, useful in acute pancreatitis where it cannot be distinguished from a condition requiring surgery and where signs and symptoms are localized to lower abdomen to reveal a pathology of reproductive organs in females.

Examination under GA [6]

The abdomen should always be palpated under GA, before an incision is made so that previous findings may be confirmed.

In a child mass due to intussuception is better felt under anesthesia.

Per Rectal Examination - The following are noted:

1. Tenderness

Pelvic tenderness is an important finding. Diffuse tenderness is non-specific found in diffuse inflammatory conditions. Effusion in peritoneum is indicative of pelvic irritation appendicitis.

2. Any Growth

3. In lower 3/4 or rectum causing obstruction.

4. In all cases of obstruction it is important to note whether the faeces is present or whether rectum is dilated and empty as in colonic obstruction.

5. On withdrawing the finger in every case, assessment should be made about colour, presence of blood or mucus.

6. In elderly males if B.P.H. is present post operative care should be given for retention of urine.

7. In advanced case apex of intussusception may be felt.

8. In females on applying pressure forwards high up in rectum recognition is made of tubo-ovarium or uterine disease.

9. In gross peritoneal infection the tissues may be felt as boggy and edematous.

Particularly in vicinity of rectovesical or rector uterine pouch. In more advanced cases of pelvic infection definite abscess may be felt in resolving cases the pouch involved is diffuse with firm infiltration.

Per Vaginal Examination

In ectopic pregnancy non-clotting blood from cul-de sac doughy pelvic mass with recovery of blood by colpocentesis is evidence of haemoperitoneum. There is bleeding from vagina, which is, thicker and darker than normal menstrual flow and cervix is softer than normal.

There may be or may not be much findings in a ruptured corpus luteal cyst.

In torsion of tube or ovary concentrate on determining whether the swelling is attached to or part of uterus whether it moves with cervix, whether uterus or ovary can be felt apart from it. In difficult cases a good guide is presence or absence of a sharp cleft between uterus and lower pole of tumour.

If uterine vessels are felt pulsating strongly through one or both vaginal fornices the tumour is probably uterine.[36]

In acute salphingitis on bimanual examination the cervix may be soft and uterus slightly enlarged frequently it is tender. Adnexal swelling will never be there in acute salphingitis.

In rupture of tubo-ovarium abscess drainage by posterior colpotomy may help in arriving at diagnosis. In twisted or ruptured ovarian cyst there mayor may not be any findings.

INVESTIGATIONS

Blood Studies [4, 5]

Hb%, Haematocrit and WBC counts taken on admission are highly informative. Only arising or marked leucocytosis especially in presence of a shift to the left on blood smear is indicative of serious infection. Moderate leucocytosis, commonly encountered in inflammatory condition is non-specific and even may be absent. A low white cell count is feature of viral infection such as mesenteric adenitis or gastro-enteritis.

Serum electrolytes, urea and creatinine are important especially if hypovolemia is expected. Arterial blood gas determination should be obtained in patients with hypotension, peritonitis, pancreatitis, possible isecllemic bowel and septicemia unsuspected metabolic acidosis may be the first clue to serious disease.

A raised serum amylase level corroborates clinical diagnosis of acute pancreatitis.

Clotting studies should be done if history hints a haematological disorder.

Urine Tests

Dark urine reflect mild dehydration. Tea colored urine in Hyperbilirubinemia, Microscopic Haematuria or pyuria in UTI.

Stool Test

For malaena, ova cyst, stool culture.

Plain Erect X-ray Abdomen [6, 12]

Plain films are indicated in patients who have appreciable abdominal tenderness. Distension on in case of suspected obstruction or perforated viscus renal ureteric calculi acute cholecystitis.

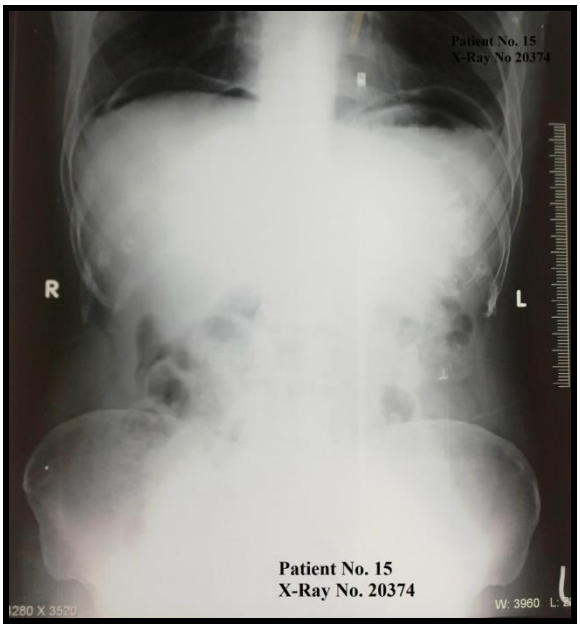


Fig 3: Erect X-ray abdomen showing gas under the diaphragm

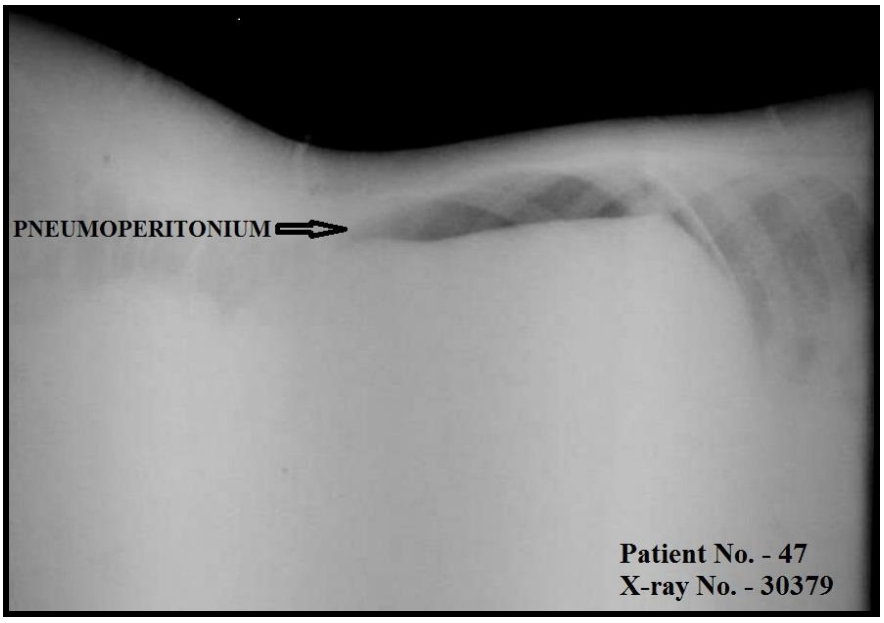


Fig 4: Lateral decubitus X-ray abdomen showing pneumoperitonium

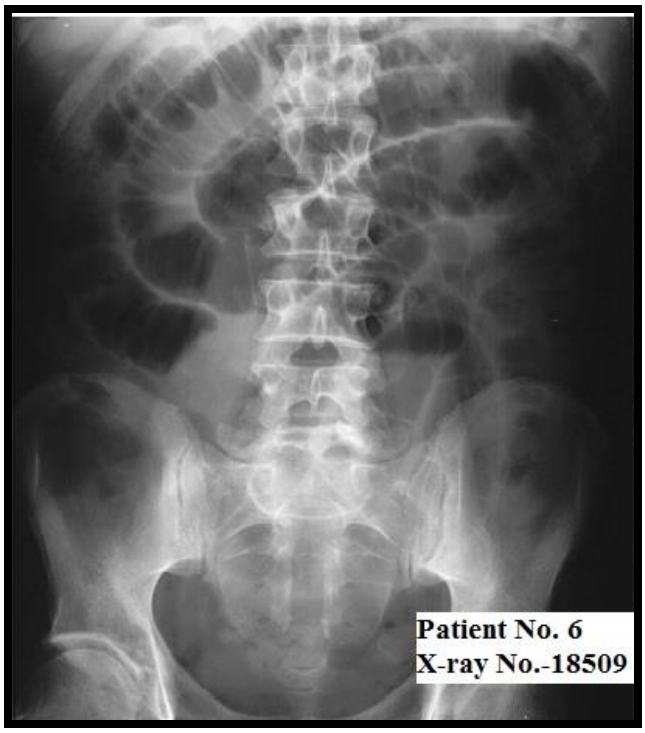


Fig 5: X-ray showing small bowel Obstruction

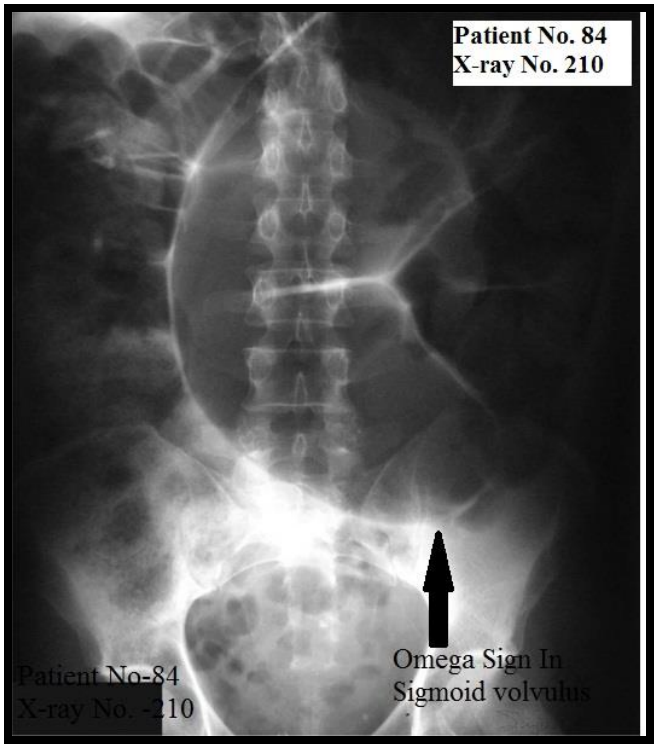


Fig 6: X-ray showing large bowel obstruction

X-ray findings in Pathological Conditions

1. **Muscular Rigidity**: This can be demonstrated by studying flunk and curvature of spine.
2. **Obliteration of psoas margin**: Indicates retroperitonial disease, curving of spine with concavity towards right are findings in perforated, duodenal ulcer, Appendicitis, ureteric colic right.
3. **Gas in intestine:** Small amount of gas is detected in stomach and colon, with smaller amounts in small intestines, abnormal gas patterns are seen in motor disturbances of GIT as in ileus, obstruction or circulatory impairment with diminished absorption of gas.

Gas may be detected in biliary tract when there are fistulous tract between intestine and gall bladder. Gas may be seen in bowel wall and portal vein in advanced mesentric infarction.

Air fluid levels are usually seen in disital small bowel obstruction and a distended caecum with small bowel dilatation in large bowel obstruction.

Dilated loop more than 6 cm, marked distention of caecum, general peripheral position of bowel, several incomplete haustral folds, typical of colonic obstruction.

Thumb print impressions on colonic wall are noted in half the cases of ischemic colitis. Displaced gastric colonic air shadow may be the only sign of sub-capsular splenic haemetoma.

4. **Pneumoperitoneum:** Free air develops in association with a perforated hollow viscus. Pneumoperitoneum is unusual in appendicular perforation.
5. **Fluid in Peritoneal Cavity**

The density of which nearly corresponds to that of parenchymatous organs. In pelvis minimal collection shows wedge shaped shadows between loops of intestine.

Small collections produce a density similar to half moon and larger collections as full moon.

6. **Diaphragmatic Signs**

The movements of diaphragm should be seen under fluoroscopy. Movements on right side are limited in appendicular perforation and perforated peptic ulcer in diffuse peritonitis movements limited bilaterally.

7. **Pulmonary Signs**

Reactionary effusion in peritonitis pancreatitis atelectasis in peritonitis presents as horizontal sharp formed densities.

Special Radiological Studies [6, 12]

1. Barium Examination

Barium meal seldom done because of possibility of ingested barium spilling over the peritoneal cavity or precipitating obstruction. Often used in stromal obstruction diagnosis after gastric surgery.

Enema useful in large bowel obstruction to demonstrate, site and nature of obstruction. It is of therapeutic value in intussuception.

2. IVP used in renal or uretric colic.

3. Angiography

Angiographic studies are indicated if intra abdominal intestinal ischemia or haemorrhage is suspected. Selective visceral angiography is a reliable method of diagnosing mesentric infarction ruptured. Liver adenoma or aneurysm of splenic artery.

It is contraindicated in unstable patients and shock or sepsis.

4. Ultrasound

Ultrasonography saves time and money can be performed in emergency department show high specificity and sensitivity.[11] Most useful in diagnosis of cholecystitis, cholangitis, pancreatitis, intraabdominal abscesses.

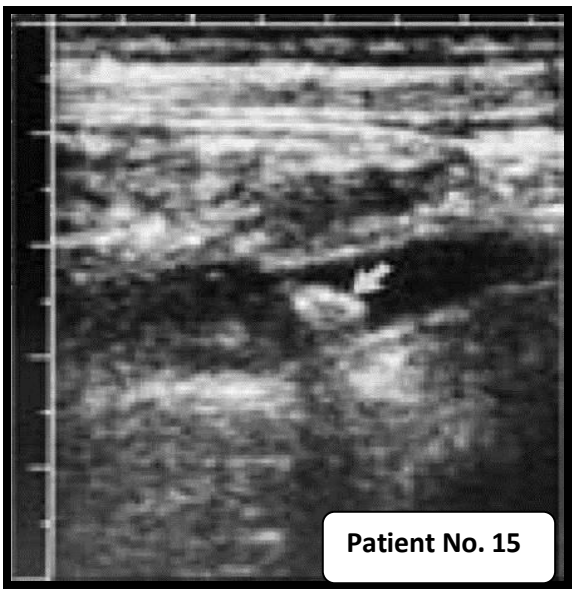


Fig 7: Acute appendicitis. On ultrasonography, a radiographically nonopaque appendicolith (arrow) is evident within a thick-walled, distended appendix (longitudinal view).

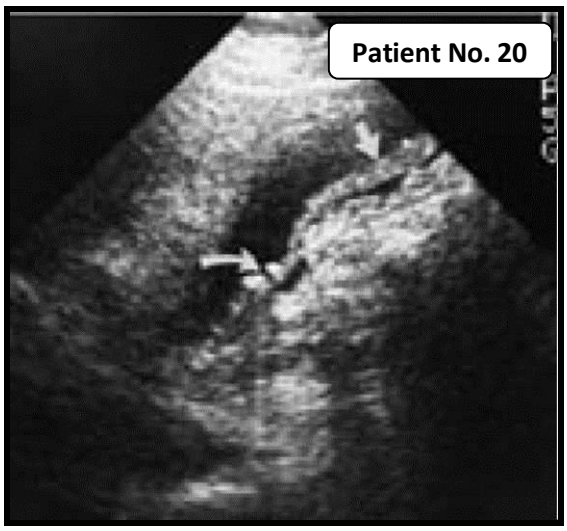


Fig 8: Acute cholecystitis. Ultrasound evaluation shows two small stones (curved arrow) present in the neck of the gallbladder. The wall of the gallbladder in the fundus (straight arrow) is thickened, and pericholecystic fluid is present.

5. CT Scan

Useful in pancreatitic and retroperitoneal lesions.

Radionuclide Scans

Useful in locating, intraabdominal abscess, intestinal bleeding, detection of ectopic gastric mucosa.

Video laparoscopy [10]

The laparoscopy procedures were done with four main purposes diagnosis (i.e. enteritis) diagnosis and treatment (i.e. Acute cholecystitits) treatment only (Appendicitis).

SPECIFIC TYPES OF ABDOMINAL DISEASES

APPENDICITIS

General Considerations

It is essential that the earliest signs and symptoms of appendicitis be appreciated clearly; the view is accepted by most surgeons of experience that every patient with acute appendicitis should be operated on within the first twenty-four hours from the onset or as soon thereafter as possible.

It is desirable, and in many cases possible, to diagnose appendicitis before peritonitis has set in, or at least before there is any more than that slight amount of irritation of the peritoneum that is commonly associated with any inflammatory process within the gut.

Anatomical position of the appendix in relation to symptom [3,5]

The vermiform appendix, though usually described as being situated behind ileocecal junction with the tip directed toward the spleen, is not al, found in that situation. To know the common positions is of great importance in diagnosis, for the signs and symptoms may thereby vary considerably. The accompanying diagram shows the more common positions For descriptive purposes it is well to recognize the ascending appendix, the iliac appendix, and the pelvic appendix. However, because of differences in degree of embryological rotation of the caecum, the appendix might lie in any position, even in the left upper quadrant. When the appendix lies by the side of the ascending colon or in the iliac fossa, there will be the most definite local signs, while if it is situated behind the caecum or behind the distal portion of the ileum and the common mesentery, the inflammatory process will be somewhat masked by the gut lying in front. If the appendix hangs over the right brim of the true pelvis, the

disease may give rise to few signs in the suprapubic region of the abdomen, resulting in a dangerous condition to which we shall call attention below.

Many of the mistakes made in the diagnosis of appendicitis are due to a failure to realize the very great difference in signs and symptoms that follow from the varying positions and relations of the appendix.

The symptoms and local signs of the attack

The signs and symptoms are as follows: Pain (epigastric or periumblical then right iliac fossa) /vomiting/nausea/ acute loss of appetite/ local tenderness (Per abdomen or per rectum) / Local rigidity of muscles (in constant)/ Superficial hyperaesthesia (in constant)/ Fever (in constant)/ Constipation (in constant)/ Testicular symptoms (uncommon).

The order of occurrence of the symptoms

1. Pain, usually epigastric or umbilical **2.** Anorexia, nausea, or vomiting **3.** Tenderness-somewhere in the abdomen or pelvis **4.** Fever **5.** Leukocytosis

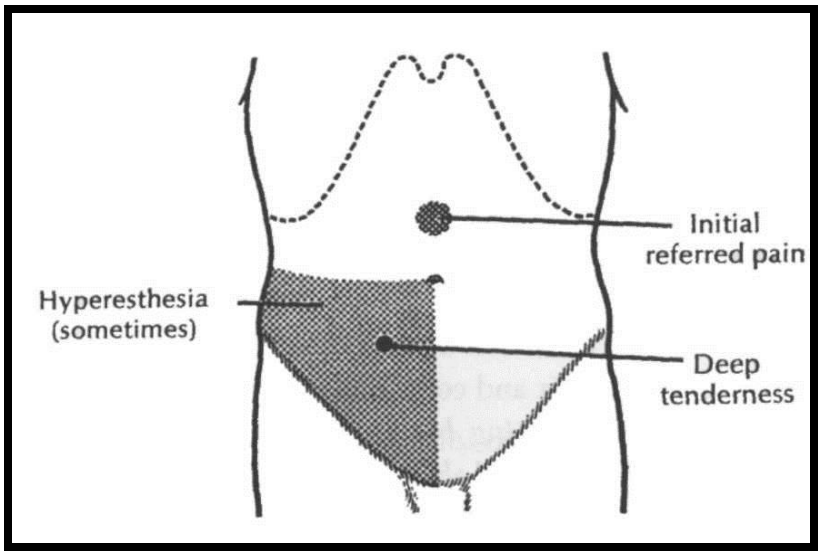


Fig 9: The common position of initial referred pain, the position of deep tenderness (nearly always to be elicited when the abdominal will is not rigid, and the iliac triangle of hyperesthesia found in some cases of appendicitis.

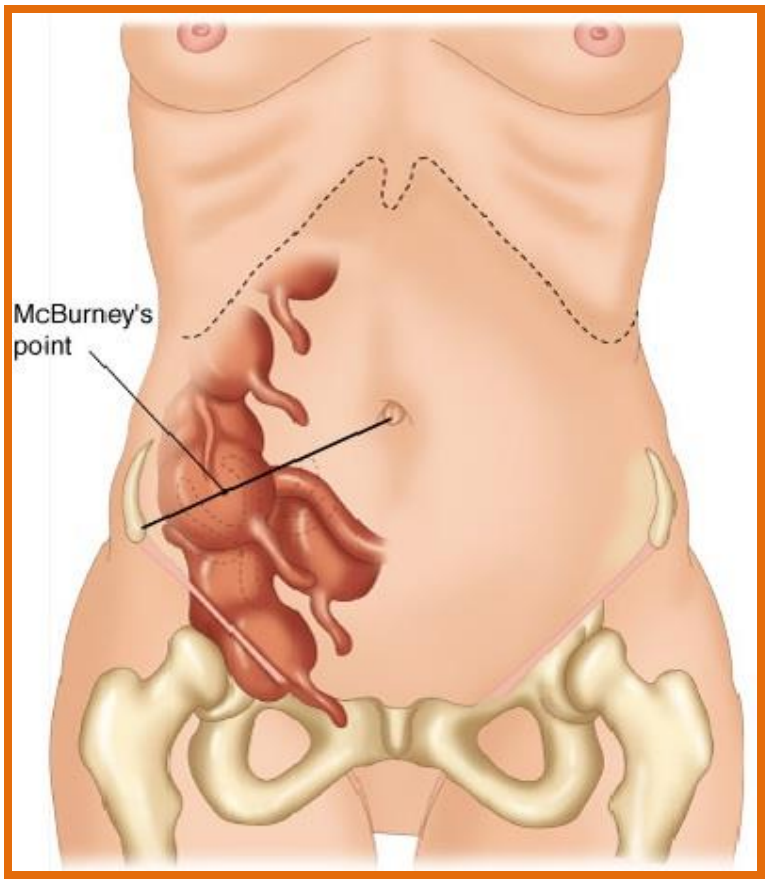


Fig10: Various possible positions of the appendix

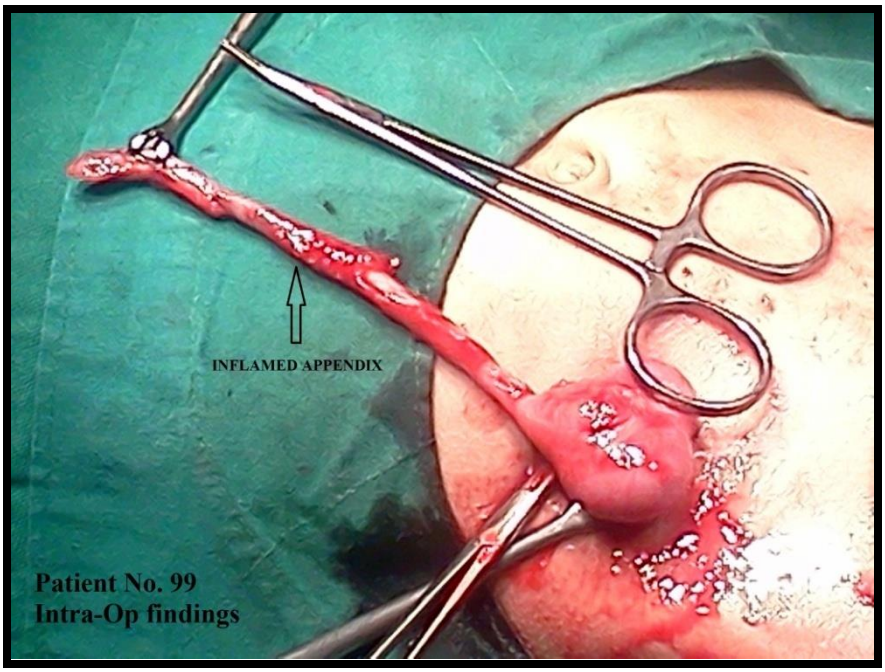


Fig 11: Acutely inflamed elongated appendix

Investigations [5]

The diagnosis of acute appendicitis is essentially clinical, however a decision to operate based on clinical suspicion alone can lead to removal of normal appendix in 1530% of cases. A number of clinical and laboratory based scoring systems have been devised to assist diagnosis. The mostly widely is Alvarado score. A score of 7 or more is strongly predictive of acute appendicitis. Abdominal ultrasound examination is more useful in children and thin adults, particularly if gynaecolological pathology is suspected. Contrast enhanced CT scan is most useful in patients in whom there is diagnostic uncertainty particularly in older patients.

Table 5: The Alvardo Score

Symptoms	Score
• Migratory RTF pain	1
• Anorexia	1
• Nausea and vomiting	1
Signs	
• Tenderness (RIF)	2
• Rebound tenderness	1
• Elevated temperature	1
Laboratory	
• Leucocytosis	2
• Shift to left	1
Total	10

Treatment

The treatment of acute appendicitis is appendicectomy. There is a perception that urgent operation is essential to prevent the increased morbidity and mortality of peritonitis.

Appendicular Mass

It is treated by Oschner and Sherrens Regime, patient is kept nil orally,with naso gastric aspiration to give rest to the gut.Intravenous Antibiotics are startwd with I.V fluids and analgesics. Temp, pulse &blood pressure charts are maintained. After 6-8 weeks,patient is advised Interval Appendicectomy.

Appendicular Abscess

Is drained by extraperitoneal approach.An incision of 5-6 cm is made in the right iliac fossa and all muscles are divided. However,peritoneum is not opened.It is swept medially and pus is drained outside. Appendicectomy is done at a later date.

ACUTE CHOLECYSTITIS

Most cases of acute cholecystitis are caused by gallstones obstructing cystic duct.[2,5] Gallstone obstructing the duct orifice leads to vigorous contraction of the gall bladder resulting in the pain of biliary colic if the stone fails to pass through the duct contractions increase and edema begins in the walls of the gall bladder. As edema and obstruction persist damage to the epithelial cell layer of the mucosa of the gall bladder occurs followed by release at enzymes. Such as phospolipase with break down the phospholipids. Acute calculous cholecystitis is frequently seen the setting of severe stress multiple trauma and critical illness.

Clinical Presentation

Common in middle-aged patient with previous history of biliary colic approximately 25% of patients will have no previous history of biliary symptoms. Once the pain of biliary colic is persisted beyond 4to 6 hours clinical diagnosis of cholecystitis is made. The pain is crampy yet does not cease between cramps (Small bowel colic). The radiation is generally to epigastrium and occasionally to the back rarely to the flank or lower abdomen. Nausea vomiting is common fever is generally low.

The physical findings are right upper quadrant tenderness; guarding and murphys sign A mass is found in 20% of cases laboratory findings are WBC count commonly between 12.000 - 15.000. More than 15.000 indicate gangrene or perforation.

Bilirubin levels less than 4 mg per dl are encountered frequently. More than 4 mg indicates CBD stones. A rapid rise more than 4-6 mg per dl per day indicates perforation. The serum amylase is frequently elevated. Mild increase in serum

transaminases and alkaline phosphatase are frequent.

Use of imaging studies in the diagnosis the plain film is useful to exclude other differential diagnosis. Findings referable to the gall bladder are, air in the gall bladder, calcified stones.

Oral cholecystogram is not useful in diagnosis, because of nausea and vomiting.

Use of cholangiography was widespread 30 years ago. Ultrasonography is now the most commonly used screening test. Gallstones are detected in more than 95% of patients who have them. This titre is lower in ill patients with ileus and air distended abdomen. The findings are dilation of gall bladder, thickened walls sludge in the gall bladder pericholecystic fluid collections.

Radionuclide Scanning

A family of the labeled isotopes rapidly excreted by hepatocytes faiure to see the gall bladder. Within 60 minutes indicates cholecystitis.

CT scan

CT scans are very poor for detection of stones, but detects local edema 6 pericholecystic fluid.

Treatment

Patient is kept nil orally for 2-3days,with naso gastric aspiration to give rest to the gut. Intravenous Antibiotics are started with I.V fluids and analgesics. Temp, pulse &blood pressure charts are maintained. After 6-8 weeks, patient is asked to come for elective cholecystectomy.

Surgery

Surgery is standard treatment surgical intervention in the first 3 days prevents development of complication such as perforation gangrene, which otherwise occurs in

10%, decreases the total duration of illness and disability.

Many surgeons recommend and early cholecystectomy other indications are, patients fail to improve within 24 hours. Patients presenting with high fever or chills high WBC count generalized peritonitis palpable gall bladder suggests gangrene.

Ideal procedure is cholecystectomy. Recent procedure for ill patient is percutaneous tube cholecystostomy under ultrasonographic guidance.

Acute Acalculous Cholecystitis

The cause is unclear it is suspected that biliary stasis fasting and cyclic duct edema alteration in regional blood flow all plays a part sludge is common finding in ultrasonographic examination.

ACUTE PANCREATITIS

Etiology

Upto 75 - 85% of acute pancreatitis is associated with biliary lithosis or chronic alcohol ingestion.

Biliary lithiosis

The discussion of gallstone pancreatitis originated with the Halstead hypothesis[5,6] that obstruction of a common biliary a pancreatic channel might lead to reflex of bile into the pancreatic duct and results in pancreatitis support for this hypothesis was lacking for more than seven decades. Anatomical studies indicate however that true common channels exits only in 6% of population. Stones are impacted in the ampullae in only 1% of patients dying from pancreatitis. Ligation of pancreatic duct fails to lead pancreatitis. Normal pancreatic ductal pressures are usually found in acute pancreatitis. Bile in the pancreatitis duct at near-normal pressures does not cause pancreatitis in animals. Thus although there is an undeniable association between acute pancreatitis a passage of stones through the sphincter of oddi impaction with bile reflux is probably not a major precipitating factor in acute pancreatitis.

Alcohol: As in biliary lithosis the evidence for a relationship between alcohol ingestion and the development of pancreatitis is incontrovertible but poorly understandable Chronic ethanol abuse over a 6 to 10 years period alters pancreatic secretary activity and eventually promotes structural changes as well these changes probably predate the initial symptoms of pancreatitis.

Hyperlipidaemia

Lipomic serum is frequently found in association with and probably proceed

the development of acute pancreatitis Most strongly associated with pancreatitis Frederickson type IV or V hyperlipoproteinemia. Lipomic serum is also present in 10-20% of patients with acute alcoholic pancreatitis. The mechanism by which hypertriglyceridemia evokes pancreatitis is not known but local lipolysis with liberation of cytotoxic free fatty acids has been proposed in presence of hypertriglyceridemia the serum amylase concentration is frequently not elevated.

Obstruction

Pancreatic duct obstruction by Pancreatic duct stones, Peri-ampullary tumours, parasites such as Ascaris & clonorchis can cause pancreatitis by obstructing the Pancreatic duct.

Postoperative Pancreatitis

Commonly occurs as a result of an operation on the gland itself or an adjacent organ. It carries extremely high mortality rate of 25-40% postoperative pancreatitis occurs against a background of postoperative pain 6 nonspecific hyperamylasemia which may delay the diagnosis contribute to the high mortality rate.

Peptic Ulcer Disease

With penetration to the gland gives rise to a localized response.

Direct Physical Trauma

Common in teenage years. The diagnosis cannot be made on the basis of hypermylasemia can rarely be made on the basis of imaginary studies and usually rests on surgical exploration.

Instrumentation

The mechanism is not clear the theory that trypsinogen is activated in the presence of increased circulating calcium.

Others

Corticosteroids, thiazide diuretics, anti-metabolites sulphonamides, scorpion venom, viral illness, vascular disease.

Symtoms & Signs

Patients with acute pancreatitis typically complain of steady dull or boring mid epigastric pain. Pain is poorly localized and may radiate to back or flank. On physical examination epigastric tenderness to deep palpation is generally appreciated but abdomen is typically soft owing to the retroperitoneal location and absence of peritonitis.

Jaundice, Grey-turners sign and cullens sign may develop as the disease progresses but are seldom appreciated at admission. Tetany to hypocalcemia a metastatic fat deposits are rare.

Investigations:

Isoamylase

Serum amylase can be separated into its isoenzyme constituents salivary and pancreatic isoenzymes are more specific.

Urinary Amylase

The amylase creatinine clearance. This test is considered by some authors to be specific that a normal ratio excludes acute pancreatitis.

Serum Lipase

It is elevated in same disease in which pancreatic amylase is elevated. This test is not specific.

Trypsin and Phospholipase

Elevated levels are specific for pancreatitis significantly more elevated in polyhaemorrhagic pancreatitis.

Imaging Studies

Plain radiograph [12]

Signs of acute pancreatitis on plain abdominal films.

1. Sentinel Loop.
2. Colon Cut-off sign
3. Generalized Ileus
4. Obscure of psoasmargins
5. General haziness of ascites
6. Increased epigastric density
7. Over distended duodenum 6 jejunum
8. Displaced stomach
9. Atonic stomach
10. Diffuse pancreatic calcification peri pancreatic gas.

Ultrasound abdominal Scan

Nonspecific abnormalities of the pancreas can be identified in 30-50% of patients.

CT scan

Abnormalities can be detected in as many as 70% of unselected patients with acute pancreatitis and 100% of those with severe disease. Complications of pancreatitis including peripancreatic fluid collections, Pseudocyst, pancreatic necrosis and pancreatic abscess can be detected using this technique.

Radio Isotype Scanning

Useful to exclude cholecystitis.

Ranson's Criteria of severity for acute pancreatitis.[5]

An admission

1. Age over 55 years.

2. WBC count 16.000 per Cu mm.

3. Blood Glucose >10 mmol/lit.

4. Serum LDH > 700 units/lit.

5. SGOT > 250 sigma trankul units.

During initial 48 hours

1. Fluid sequestration >6 lit.

2. BUN increase >5 mg%.

3. Arterial Po2 < 60 mm Hg.

4. Base deficit > 4 mmol/lit.

5. Serum calcium <2 mmol/lit.

Complications of Acute Pancreatitis [6, 13]

Hypovolumia

It s the earliest most common complication. It is a result of both of actual loss of circulatory volume due to increased capillary endothelial permeability it decreased peripheral resistance, hypovolumia is refractory to fluid administration.

Acute Renal Failure

Is the second most common. Acute renal failure can develop in the absence of documented hypotension it is due to deposition of fibrin in the glomeruli possibly resulting from the activation of the coagulation cascade by trypsin, vasodilator therapy is of no benefit.

Respiratory Failure

It is due to intrapulmonary right to left shunting caused by microthrombi resulting form sub clinical DIC. Other contributing factors are diaphragmatic elevation atelectasis pulmonary infiltration pleural effusion hypoxia contributes 30% of deaths. Circulating phospholipase A17 and high concentration of fee acids damage the pulmonary surfactant.

Myocardial depression

This complication mediated by vasoactive peptides and myocardial depressant factor.

Hypocalcemia

Transient hypocalcemia develops in about 1/3 of patients with acute pancreatitis. It is due to sequestration in areas of fat necrosis and hypoalbuminemia.

Hyperglycemia

This occurs in 15-25% of patients. This results from excess circulating glucagon. Insulin levels are elevated or less than normal.

Disseminated Intravascular Coagulation

More commonly it is in subclinical form, which probably contributes to pulmonary and renal impairment.

Local Complications

Haemorrhage

Haemorrhage in association with acute pancreatitis can arise in many forms and occurs in 14% to 41% of cases.

Mechanisms of Hemorrhage in Pancreatitis

1. Coexistent peptic ulcer disease.

2. Stress gastritis.

3. Pre-existing gastro esophageal varices.

4. Diffuse mucosal hemorrhage pancreas adjacent to the inflamed

5. Intra pancreatic and peri pancreatic hemorrhage.

6. From G.I.T at the site of fistula formation.

7. Splenic vein thrombosis with formation of gastric varices.

8. Erosion of major vessels.

Phlegmon

Pancreatic phlegmon is a solid mass of indurated pancrease and adjacent retroperitoneal tissue. It is associated with fever pain prolonged leucocytosis. It usually resolves in 7-14 days.

Pancreatic Abscess

Pancreatic abscess occurs s a complication in 1-9% of admissions for acute pancreatitis. The clinical course of pancreatic abscess consists of a bowl or severe pancreatitis followed by a period of seeming resolution and the development 1 to 3 weeks later of systemic toxicity. Presenting symptoms include pain, fever, tachycardia and palpable mass.

Pseudocyst

A pseudocyst is a unilocular or multilocular collection of fluid surrounded by a fibrous capsule composed of adjacent organs visceral or parietal peritoneum and inflamed connective tissue. More commonly associated with chronic pancreatitis. Pseudocysts may develop in as many as 50% of patients with severe acute inflammation.

Fistulas

Pancreaticocutaneous fistulas or internal fistulas may develop spontaneously or in postoperative period.

Management [4, 13]

Medical Therapy

Non-operative therapy is aimed at symptomatic relief 6 treatment of systemic complications. Patient is kept nil orally with nasogastric aspiration and pain is managed with narcotic analgesics.

Shock: Dehydration is common at presentation because of nausea; vomiting and internal losses form fluid sequestration. It contributes heavily to renal failure and is dominant cause of early death due to acute pancreatitis. Thus early and vigorous I.V. fluid administration is probably the single most important therapeutic measure.

Renal Failure

The ideal is management is prevention. Diuretics should be given with great reservation and only after providing the adequacy of the intravascular volume using wedge pressures.

Ventilatory Failure

Can be insidious and blood gases should be regularly monitored standard therapy should progress to mechanical support.

Hyperglycemia

Should be treated with regular insulin and only when the blood glucose exceeds 250 mg per dl.

Nasogastric suction has been proved to be of no specific benefit. It should be reserved for relief of nausea and vomiting antibiotics should probably be reserved for the specific treatment of documented infections to avoid the promotion of opportunistic organisms.

H2 blocker to inhibit secretion release and pancreatic secretion by reduction of duodenal acidification has failed to prove beneficial

Surgical Therapy

Haemorrhagic complications other than intraparenchymal Haemorrhage,

If active arterial bleeding is found pre operative embolization is advised if bleeding from gastric varices due to splenic vein thrombosis splenectomy is advised.

Pseudocyst

Operative indications for pseudocysts arising in acute pancreatitis are bleeding rapid expansion infection and rupture.

Infected Pancreatic Necrosis

This condition demands operation by its presence because the mortality rate 100% without intervention. The principles of management include full delineation of the necrotic process. Adequate, postoperative drainage. There must be a willingness on the part of surgeon to re explore.

Fistulas of the small bowel and colon

These fistulas can be treated expectantly unless hemorrhage or sepsis supervenes.

ACUTE COLONIC DIVERTICULITIS

Incidence

It is estimated to occur in approximately 5% of the population. Uncommon before 40, the incidence increases with advancing age so that by 9th decade it is present in 2/3 of population. The sigmoid colon is involved in 90% of the cases. Right sided disease is more common in Asian countries especially Japan.

Pathology and Pathogenesis [14]

Most colonic diverticula are pseudodiverticula because they do not contain all the layers of the bowel. The combination of a relative pressure gradient from the lumen to the serosa and the presence of a defect through which the mucosa and submucosa can herniate. According to the law of Laplace the intraluminal pressure will be greatest where the lumen is narrowest and the wall is thickest. This explains increased likelihood that diverticula will develop in the sigmoid colon. It is suggested that diverticular disease is caused by a lack of fiber in the diet. It results in increased segmentation and diverticula formation right sided are usually true diverticular and the origin is commonly thought to be congenital.

Diagnosis

The signs and symptoms of acute diverticulitis are many. A history of previous episodes of diverticulitis or barium enema is helpful. The classic picture of left lower quadrant abdominal pain, low-grade fever, leucocytosis, nausea with occasional vomiting and mild abdominal distention is certainly helpful and again not always present, a palpable mass may be detected in left lower quadrant plain radiographs of the abdomen occasionally show free air within peritoneal cavity. Other more common findings noted on plain abdominal films include an ileus pattern amass effect in the

left lower quadrant or evidence of partial or complete obstruction of the colon or small bowel.

Endoscopy extremely uncomfortable it may result in perforation. Barium enema examination has been the mainstay in the diagnosis of diverticular disease but is contra indicated in the acute state. Because of the hazards of barium peritonitis if a contrast agent should be used. Certain finding that implicate diverticulitis are presence of fistula or sinus tract intramural abscess exercise compression by a pericolic mass an extaluminal collection of barium. Other investigations that are helpful are IVP.

Medical Management

Medical Management consists of bowel rest with or without nasogastric suction, IV fluids and systemic parenteral antibiotics.

The use of morphine as an analgesic should be avoided because of its association with increased intraluminal pressure if the patient does not respond to this conservative within 48 hours serious. Consideration must be given to surgical intervention. The reason for failure is development of complications.

Surgical Management

Elective resection and anastomosis is advised for uncomplicated diverticulitis.

Classification of Perforative Diverticular Disease [2, 14]

Stage I Contained pericolic abscess.

Stage II Walled-off Pelvic abscess.

Stage III - Generalized Purulent Peritonitis.

Fecal Peritonitis.

ACUTE PERITONITIS

Apart from hemorrhage, the cause of death in nearly all fatal acute abdominal cases is either secondary peritonitis or acute paralytic or mechanical obstruction of the intestines. Spreading or general peritonitis is the most common single cause of death. Most cases, unless the patient is actually moribund, sooner or later demand an opening of the abdomen. At operation, fluid may easily be obtained for examination, but even before operation it is possible to obtain some by puncturing the abdomen in the midline midway between the navel and the pubis by means of a fine-bore needle. The exudate is stained for organisms and examined microscopically.

Infective organisms may reach the peritoneum in the following ways: [4, 6]

1. Through a wound of the abdominal wall
2. Via the bloodstream
3. From the viscera within the abdomen (most common)
4. Through the diaphragm (almost never) or by lymphatic extension from the thigh (very uncommon)

The only commonly blood-borne organisms of importance are the pneumococcus, which may cause severe so-called primary peritonitis.

Organisms may reach the peritoneum from the contained viscera either by

(1) Rupture of a viscus or

(2) Escape through the diseased wall of any viscus.

In the female there is the additional path of infection via the fallopian tubes. Frequently a local abscess may form either extra- or intraperitoneally near a diseased viscus, and later this abscess may burst into the general peritoneal cavity or into an adjacent viscus.

Causes [6]

The common causes of general peritonitis are the conditions already described in previous chapters. They comprise disease or rupture of the hollow viscera and ruptured abscess of the solid viscera.

Perforation of

- Appendix vermiformis
- Gastric or duodenal ulcer
- Typhoid or tuberculous ulcer of the small intestine Dysenteric or stercoral ulcer or of a diverticulum of the colon Gallbladder or biliary ducts

Gangrene of

- Strangled coil of gut
- Intussuception
- Volvulus

Infection spreading from

- Pyosalpinx
- Infected uterus
- Pyonephrosis

Rupture of

- Liver abscess
- Splenic abscess

A less acute form of peritonitis may result from the escape of sterile bile or urine into the peritoneal cavity.

Symptoms/Signs [6]

The symptoms of peritonitis vary greatly according to the part and extent of the peritoneum involved the nature of the infective agent, and the acuteness of onset. Help in diagnosis will be obtained by regarding the symptoms as being roughly grouped into two classes, reflex and toxic:

Reflex

- Pain, Vomiting, Muscular rigidity and Anxious facial expression

Toxic

- Alteration of temperature, collapse, Distention, intestinal paresis and General toxemia

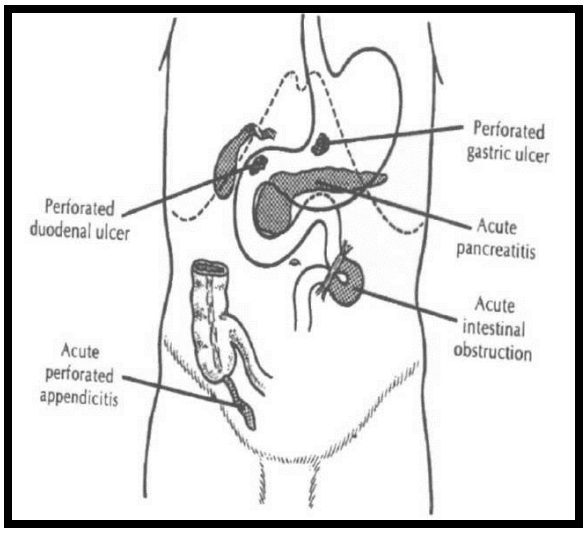


Fig 12: The more common abdominal causes of acute collapse. (In the female ruptured ectopic gestation should be added) Biliary colic and acute cholecystitis rarely cause acute collapse.

DIFFERENTIAL DIAGNOSIS

It is not difficult to diagnose a flagrant case of peritonitis, for the pain, vomiting, local tenderness, and muscular rigidity with fever sufficiently indicate the condition; but mistakes are likely to be made either because the symptoms are too

slight or because they are atypical.

The conditions that may stimulate peritonitis are as follows: [4, 8]

1. Pleuritis , 2. The colics, 3. Intestinal obstruction, 4. Internal hemorrhage, 5. Some nervous conditions (e.g., tabes, hysteria)

Perforated peptic ulcer

The signs and symptoms produced by the perforation vary according to the time that has elapsed since the rupture occurred. There are three stages in the pathological process that can usually be recognized easily, although there is no hard and fast limit between the stages. The symptoms of each stage can be enumerated.

Early (within the first two hours)

Great and generalized abdominal pain Anxious countenance /Livid or ashen appearance/ Cold extremities/ Cold, sweating face/ Subnormal temperature (95°F or 96°F) Pulse small and weak/ Shallow respiration/ Retching or vomiting (slight)/ Pain on the top of one or both shoulders.

Intermediate (two to twelve hours)

Vomiting ceases/ Abdominal pain less/ Appearance better, face regains normal color/ Temperature normal or slightly elevated/ Pulse normal/ Respiration still shallow and costal in type/ Alae nasi working slightly/ Abdominal wall very rigid, tender, and often retracted or flat Tender pelvic peritoneum/ Diminution of liver dullness/ Movable dullness in flanks (sometimes)/ Great pain on movement of the body.

Late (after twelve hours)

Vomiting more frequent but still not profuse/ Facies of late peritonitis/ Abdomen tender and distended/ Pulse rapid and small; hypovolemic shock may be present Temperature usually elevated/ Abdominal wall usually not quite so rigid/ Respiration labored and rapid.

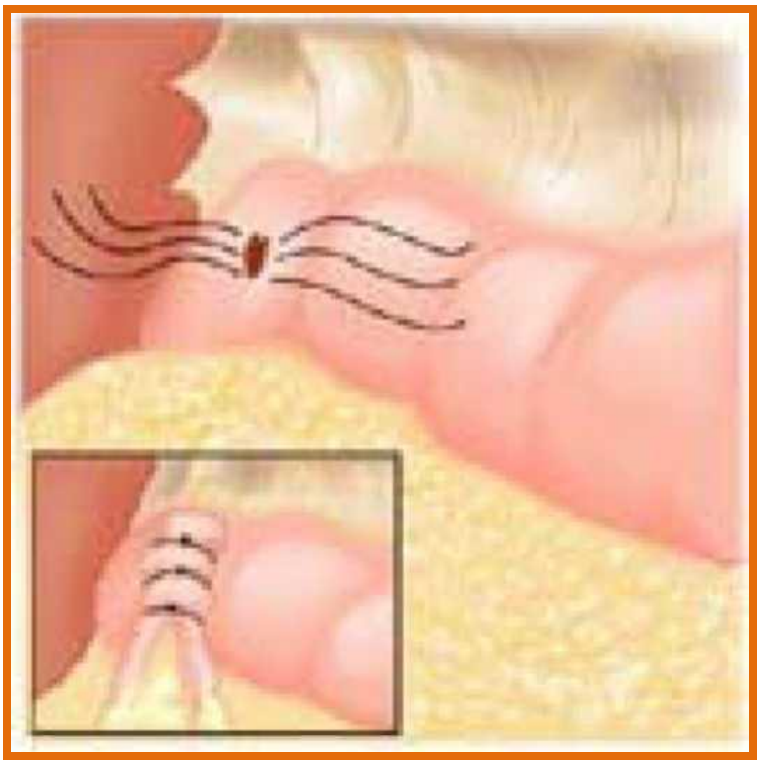
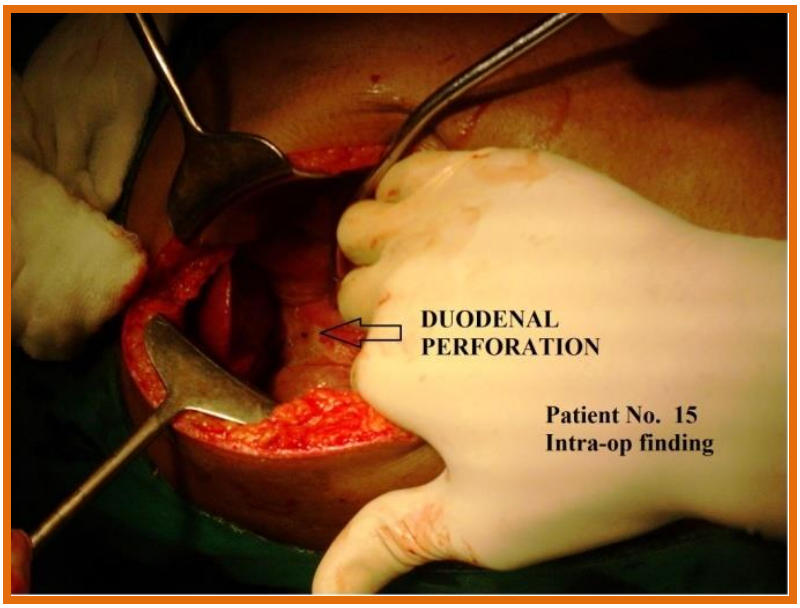


Fig 13: Line diagram showing Duodenal Perforation Closure with a pedical omental graft (Graham's patch)

DUODENAL PERFORATION GASTRIC ULCER PERFORATION



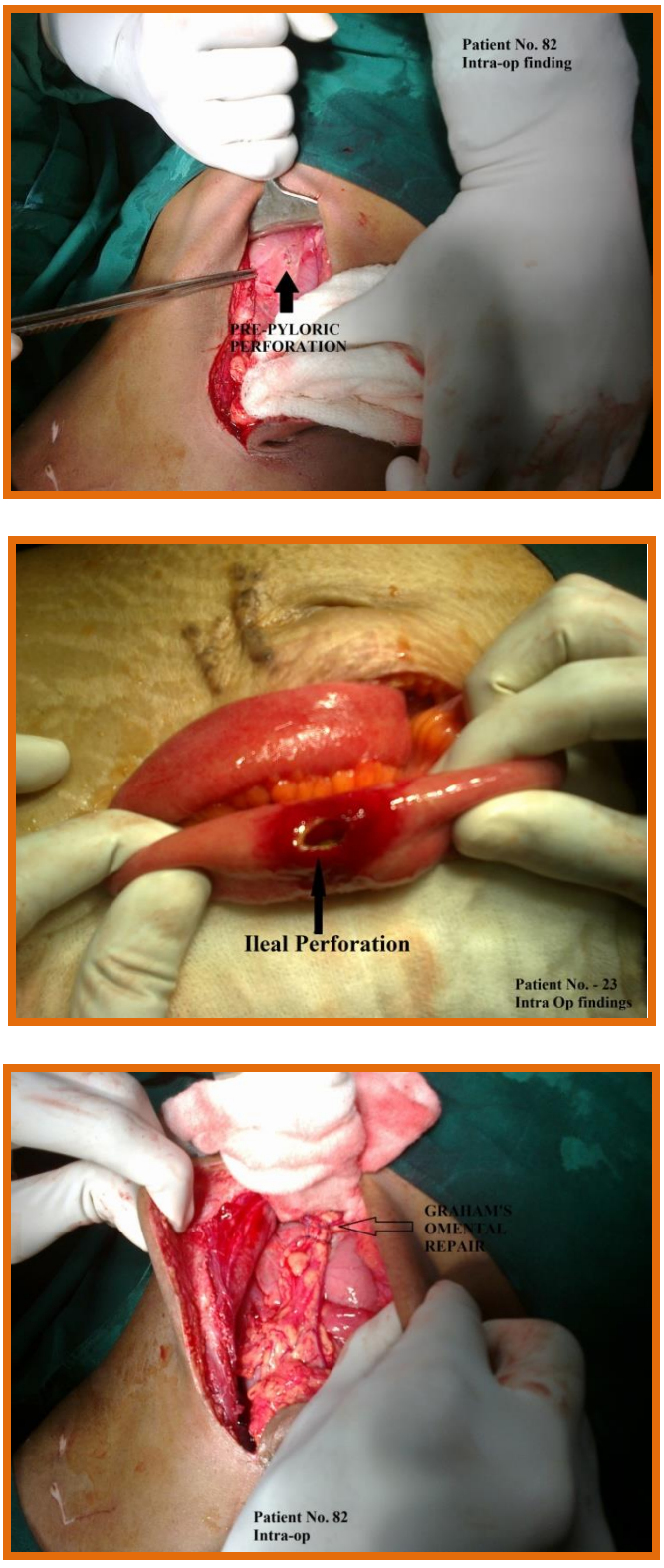


Fig 14: Different types of perforation with closure

Diagnosis [6, 12]

During the initial stage it is nearly always possible to say that there is a condition needing surgical intervention, though the exact nature of the catastrophe may be slightly doubtful. Great help is sometimes obtained from a previous history of chronic indigestion or of duodenal pain coming on about two hours after taking food. Quite a number of patients, however, give only a recent history of pain after taking food. This is more common in young people in whom acute pyloric ulcers appear to be not uncommon.

If, in a patient who has been subject to chronic indigestion, sudden collapse and very severe abdominal pain suddenly supervene, and if at the same time the abdominal wall becomes generally rigid, one is justified in suspecting perforation of an ulcer. If, in addition, the pelvic peritoneum is tender and there is resonance over the lateral aspect of the liver, the diagnosis is certain. The presence of free air radiologically confirms the diagnosis fully and no further testing need be done. In the second stage the general symptoms temporarily improve, but all the local signs remain and become still more definite, so that the careful observer should not be misled. In the third stage, there is no difficulty in diagnosing that some serious catastrophe within the abdomen has occurred.

The leakage of water-soluble contrast into the peritoneal cavity is a useful finding in doubtful cases.

Differential Diagnosis

There are four conditions, sometimes giving rise to symptoms similar to those of perforated ulcer that either does not call for operation or in which operative interference is positively contraindicated. They are:

1. Severe colic (either biliary or renal)

2. Some cases of pneumonia or pulmonary infarction 3. Gastric crises of tabes dorsalis

4. Acute pancreatitis

There are seven other conditions that are sometimes difficult to distinguish from a perforated gastric or duodenal ulcer and more commonly confused with perforated ulcer than the above three conditions.

They are

1. Acute perforative appendicitis
2. Acute intestinal obstruction
3. Ruptured ectopic gestation (in women)
4. Dissecting thoracic or ruptured abdominal aortic aneurysm
5. General or diffuse peritonitis from other causes
6. Postemetic rupture of the esophagus
7. Mesenteric embolus thrombosis

THE COLICS

True abdominal colic is always caused by the violent peristaltic contraction of one of the involuntary muscular tubes, whose normal peristalsis is quite painless. The violence of the contraction is usually produced in an effort to overcome some obstacle that prevents the passage of the normal excretion or secretion. The pain is due to the stretching or distention of the tube, and the agony produced may be as severe as any to which a human being can be subjected.

The involuntary muscular tubes that may thus cause colic are: [2, 6]

1. The stomach and intestines
2. The cystic, hepatic, and common bile ducts and the gallbladder
3. The pancreatic duct
4. The ureters
5. The uterus and fallopian tubes

The main feature of severe colic is the occurrence of acute, agonizing, spasmodic pain, which causes the patient to double up. True colic is defined as rhythmic attacks or paroxysms of pain with intermittent period of partial or complete relief. It is associated in varying degrees with the symptoms consequent to excessive stimulation of the sympathetic nervous system.

In the general diagnosis of colic the following points may help:

1. The pain usually comes in paroxysms lasting a variable time and often reflected by intermittent facial grimacing.
2. In colic, patients are usually very restless and fling themselves about as if to find some relief from the pain that grips them. A flexed position of the body may be adopted during the pain.

3. Though occasionally rigid during the paroxysms, the abdominal wall is soft between the bouts of pain. In sudden acute peritonitis, it remains rigid all the time.
4. In colic, pressure to the abdomen sometimes relieves the pain an occurrence not usual in other acute conditions.
5. In many of the colics, the distribution of pain is almost diagnostic The figure graphically depicts the periodicity and severity of the various colics.

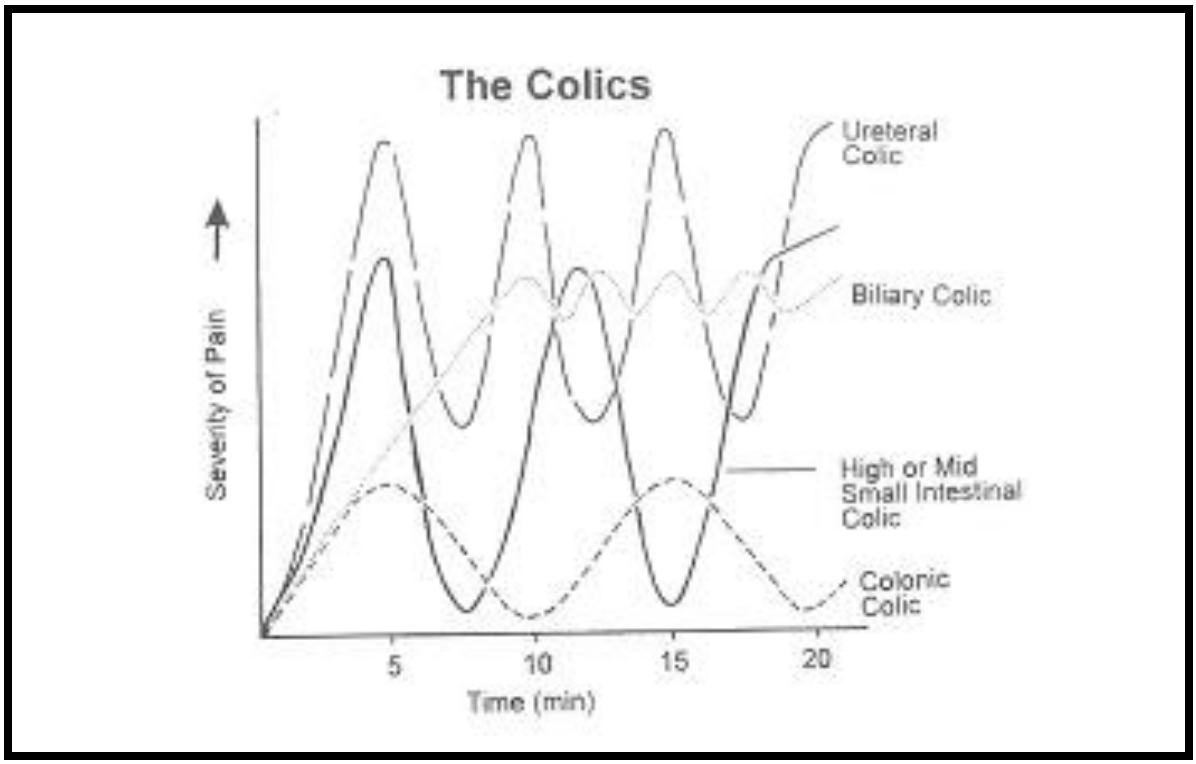


Fig 15: The Colics - A diagrammatic representation of the periodicity of the peaks of pain in the various colics. The term biliary "colic" is a misnomer because once the pain peaks; it does not have a colicky pattern.

Acute intestinal obstruction [15]

Acute obstruction of the intestine in the form of strangulated hernia was one of the first abdominal emergencies to be referred to the surgeon for treatment, while obstruction accompanied by similar symptoms, but due to internal causes that were not so obvious as an external hernial swelling, was among the latest urgent abdominal cases to be referred by the physician to the surgeon. If there is any condition in which early diagnosis and avoidance of attempts at purgation are necessary, it is intestinal obstruction.

The pathology and causation of acute intestinal obstruction are questions far too big to discuss fully in a small book. We are here concerned only with the common causes and the main types of cases that come for diagnosis. It is not always essential, in diagnosis, to know the exact cause of the obstruction, though every effort should be made to ascertain it as accurately as possible. It is useful to have knowledge of the proportion of cases due to the main pathological causes of obstruction. The list in Table 5 is a compilation of the overall causes of intestinal obstruction (both small and large bowel) taken from thirteen reported series comprising a total of 12,731 patients. The differences in the distribution of etiologies between adults and children are readily apparent. These findings do not apply to tropical regions.

When the bowel is completely obstructed, the course of the disease is inevitably fatal unless the obstruction is relieved by:

Table 6: Causes of Intestinal Obstruction[2]

Adults		Children	
Hernia	41%	Hernia	38%
Adhesions	29%	Pyloric stenosis	15%
Intussusception	12%	Ileocecal intussusception	15%
Cancer	10%	Atresias and annular pancreas	14%
Volvulus	4%	Adhesions	7%
Miscellaneous	4%	Miscellaneous	11%

1. The spontaneous rectification of the condition
2. Formation of an external fecal fistula
3. Operative interference

The third method is always advisable in unrelenting complete obstruction. So long as the obstruction is complete, exclusion of strangulation is virtually impossible. Therefore, intestinal intubation is useful and safe only in cases of partial small bowel obstruction, especially in the early postoperative period or after a recent bout of peritonitis. Until comparatively recently, the mortality after operation in cases of intestinal obstruction (excluding strangulated external hernias) was high, but during the last two decades better results have been obtained. The main desideratum is to diagnose the case early.

Intestinal obstruction may exist in a chronic or subacute form for a considerable period before an acute attack ensues. In the chronic form, the symptoms are similar in kind but different in degree from those resulting from an acute attack. Chronic obstruction, if uncorrected, usually terminates sooner or later in an acute attack.

Symptoms due to different types of obstruction [6, 15]

The features of an attack of obstruction vary according to (1) the part of gut obstructed, (2) whether or not the mesentery with the contained blood vessels is also affected, and (3) the completeness or incompleteness of the obstruction.

The part of the gut obstructed. The symptoms due to obstruction-(l) high up in the small intestine, (2) low down in the small intestine, and (3) in the large bowel can be roughly differentiated.

Obstruction high up in the small intestine

This leads to acute symptoms, vomiting comes on very early and is frequent and violent, initial pain is greater, and distention is not an early feature. The vomit is green and bilious. Such symptoms are typically seen when a large gallstone ulcerates into the duodenum. Obstruction of the duodenum by a cicatrized ulcer may sometimes be acute owing to sudden spasm and edema around the ulcer. In such cases everything taken by mouth is returned, but no feculent vomit occurs and sometimes peristalsis of the stomach may be seen. The same symptoms are seen in infants suffering from congenital hypertrophic pyloric stenosis. Distention is seen only in the epigastric region. With obstruction of the upper jejunum, the symptoms are still very acute and the vomiting begins early and is frequently repeated, while distention is not at first a noticeable feature. The farther down the jejunum the obstruction, the less acute the symptoms.

Obstruction of the lower part of the small intestine

The pain is somewhat less severe than that of obstruction higher up. Shock and pain may be present, but vomiting is a little later in onset and some time elapses before feculent vomiting occurs. Distention comes on after a few hours. In subacute cases, the ladder pattern of distention is seen, and peristalsis is often visible.

When there is obstruction in the lower ileum, considerable fluid is still absorbed from the bowel proximal to the destruction, and thus the magnitude of early volume deficits may not be great. With upper jejunal obstruction, volume deficits are large, even early after onset.

Large-bowel obstruction

Pain is much less acute, shock is comparatively insignificant (except in some cases of volvulus and intussusception), vomiting is a fairly late and infrequent symptom, while distention almost from the onset of the acute attack is the rule. An exception must be made in the case of intussusception, for in these cases distention is not an early symptom and should not be awaited, since a distended abdomen accompanying an intussusception generally means that strangulation has supervened.

Compression of the blood vessels - Strangulation of bowel

When the vessels in the mesentery of a coil of gut are compressed, first the veins and later the arteries become occluded. There is, in fact, a strangulation of the coil, and the gut soon becomes gangrenous. This strangulation of the coil of gut adds greatly to the immediate danger, for the obstruction is complete and, unless the condition is soon relieved, perforation of the bowel will occur and cause a fatal general peritonitis. This condition is commonly brought about by bands, by external or internal hernia, or by volvulus. The onset of symptoms is usually sudden, accompanied by great pain and early vomiting. Because intense symptoms usually begin early and bring the patient to the hospital soon, distention is often absent until late in the course of the illness. Collapse and severe shock are late manifestations. If the case is untreated within a short period, local peritonitis will tend to become general, and by this time the case comes to the surgeon, there may therefore be definite abdominal rigidity over the affected part of the abdomen. A mass consisting

of the closed loop is sometimes felt.

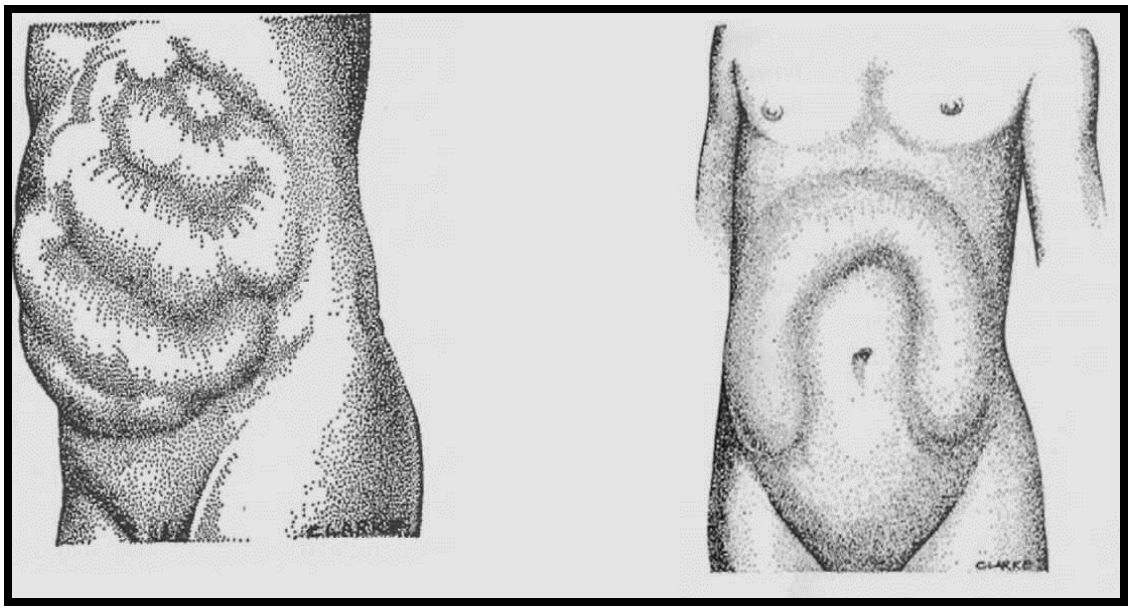


Fig 16: The ladder pattern of abdominal distension (indicating obstruction of the lower ileum)

Fig 17: The appearance of moderate distension of the large gut

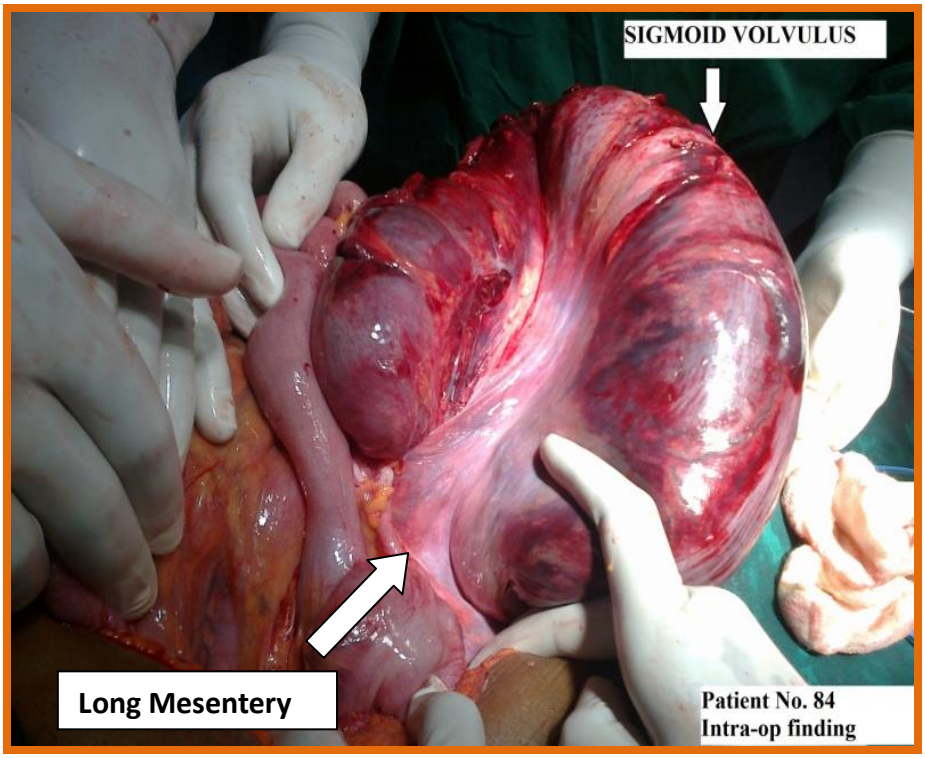


Fig 18: Sigmoid Volvulus

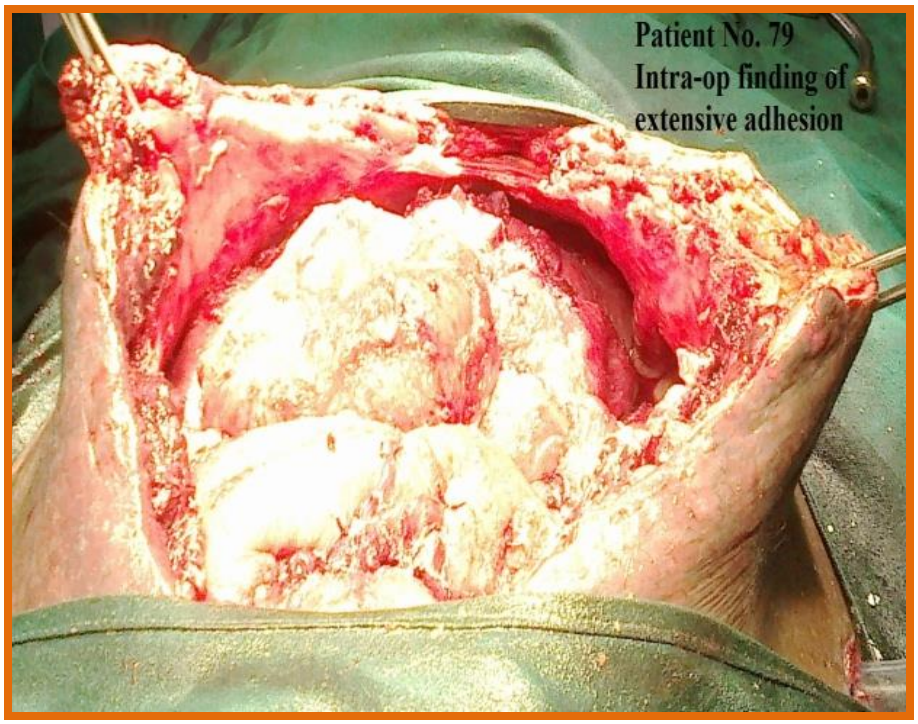


Fig 19: Post operative adhesions

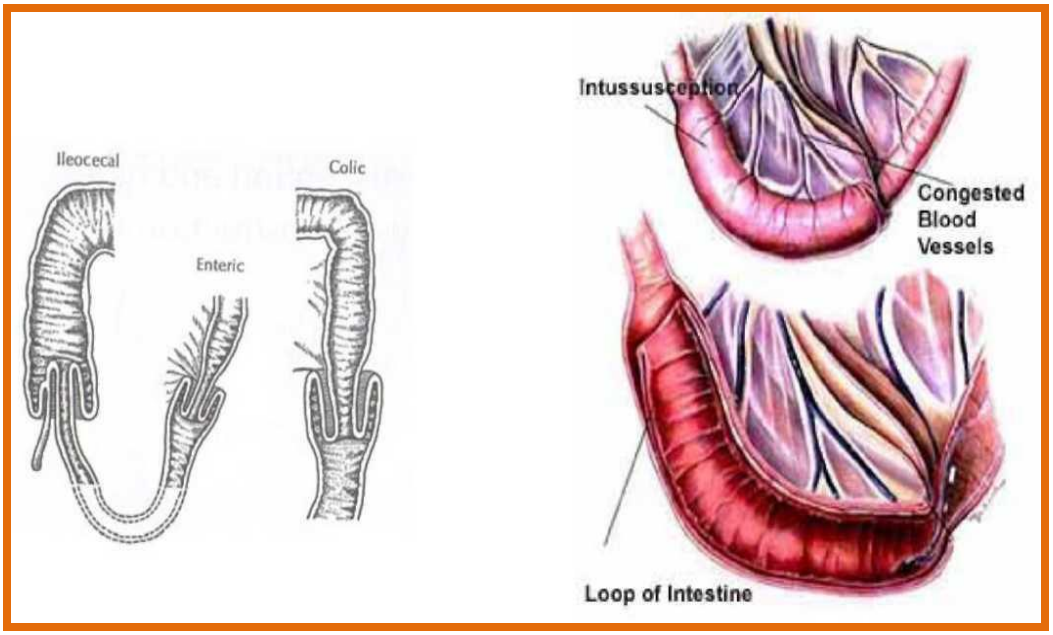


Fig 20: Types of intussusception 1) ileocecal, 2) enteric (which is termed "ileocolic" if it progresses beyond the ileocecal value), (3) colic Intussusception [4, 16]

Intussusception or invagination of the intestine is the most common abdominal emergency in children under two years. The catastrophe is all the more unexpected in that it usually attacks the most healthy-looking and well nourished babies. The condition consists in the invagination of one portion of intestine into the portion next to it. Commonly, if not invariably, the invaginated part (intussusceptum) enters the part below (intussuscipiens). Clearly, the most anatomically favorable part for such an occurrence is in the ileocaecal region, where the narrow ileum can readily enter the lax caecum, and in actual clinical experience this is the most common place for the condition to start. There are three varieties of intussusception—enteric, enterocolic, and colic. Enteric, where the small intestine alone is involved, is uncommon; colic, in which the colon alone is affected, is less rare but not very common; enterocolic is the most common variety. The enterocolic type is subdivided into Ileocaecal, in which the apex of the invaginated part is the ileocaecal valve, and ileocolic, in which a part of the gut near the end of the ileum forms the advancing apex.

Symptoms and Signs

The symptoms and signs of intussusception are usually characteristic. They comprise a few or many of the following, according to the stage at which the case is seen:

Abdominal pain/ Passage of blood and mucus per anum/ Vomiting/ An abdominal swelling/ Shock/ Visible peristalsis/ Absence of the cecum from the right iliac fossa 8. Constipation/ Tenesmus/ Distention of the abdomen/ Fever (sometimes)/ Obstruction and peritonitis (late stage)/ Rectal examination reveals blood-stained mucus on the examining finger.

Diagnosis and Treatment

A Pre-op diagnosis is essential it patients with small tube decompression. The classic findings of nonviable bowel- leucocytes tachycardia localized abdominal pain and fever.

Radiologic Studies

The use of barium studies for the diagnosis of small bowel obstruction is increasingly helpful, especially in cases where the plain films or clinical picture is non diagnostic.

It distinguishes a dynamic Ileus from mechanical bowel obstruction. In a patient with mechanical obstruction it will take 48 hours.

To reach colon, mechanical obstruction will produce dilated proximal intestine and progression of the barium to the site of obstruction in 1 hour or less. In this procedure barium is infused through a tube in to duodenum in this procedure mucosal surface can be examined thoroughly patients with early postoperative obstruction small bowel obstruction secondary to adhesions or chronic disease are less likely to progress to strangulation than those with other types of obstruction and likely to resolve with non-operative treatment failure to resolve within 48 hours are better operative.

Indications for Laparotomy [4, 16]

Physical findings

1. Involuntary guarding or rigidity
2. Severe localized tenderness
3. Progressive distention
4. Tender rectal mass with high fever or hypotention
5. Rectal bleeding with shock acidosis

6. Equivocal abdominal findings along with septicemia, bleeding, ischemia, failure of conservative treatment.

Radiological Findings

- Pneumoperitoneum
- Gross or progressive bowel distention
- Free extravasation of contrast material
- Space occupying lesion on scan with fever

  Mesentric occlusion on angiography

ACUTE ABDOMINAL SYMPTOMS DUE TO VASCULAR LESIONS

The past twenty years have seen a rapid and notable advance in the surgery of the large blood vessels, and many conditions that were formerly outside the realm of curative surgery have become amenable to such treatment.

The main conditions that need to be noticed are as follows:

1. Mesenteric arterial thrombosis or embolism
2. Non-occlusive intestinal ischemia
3. Mesenteric venous thrombosis
4. Dissecting aneurysm
5. Leakage or rupture of an abdominal aneurysm
6. Internal hemorrhage from other causes

Mesenteric arterial thrombosis or embolism [4, 17]

Mesenteric thrombosis or embolism most often affects the territory of the superior mesenteric artery, and whether the main trunk of the artery is involved proximal or distal to the middle colic artery branch will obviously influence the

course of the disease and the extent of intestinal gangrene. A detailed history indicating abdominal angina during the weeks or months before the acute episode is consistent with thrombotic occlusion of the superior mesenteric artery or its On the other hand, a history of recent endocarditis or, even more importantly, the presence of an arrhythmia such as auricular fibrillation who complains of abdominal pail suspected of having embolization to the superior mesenteric artery until proved otherwise.

Mesentric Ischaemia

Common types of mesentric ischemia

EXTENSIVE	**LIMITED**
1. Superior mesentric artery embolus.	1. Local small-bowel ischemia
2. Superior Mesentric artery Thrombus	2. Ischaemic colitis
3. Superior mesentric venous occlusion	3. Abdominal angina
4. Non-occlusive Ischemia	4. Chronic mucosal infarction.

Pathogenesis [4, 17]

Change in mucosal function becomes manifest when oxygen uptake is reduced below 50% of normal. The early responses ischemia with enterocyte. Membrane disruption and pancreatitis elastase interference with brush-border protective glycoproteins expose the underlying intracellular structures to trypsin. In the presence of hypoxia the action of trypsin converts. Xanthine oxidase proteolytic, which is abundant in villi to xanthine dehydrogenase thereby. Setting the stage for further damage by oxygen radicals during reperfusion.

Extensive Mesenteric Ischemia

Diagnosis

Role of Clinical suspicion

Clinical suspicion should arise when there is unexplained sudden abdominal pain lasting more than 2-3 hours. In a patient who might have condition associated with vascular occlusion or Ischemia - associated condition is not precisely defined especially in elderly patients experience diabetes that patients over 50 years of age with vascular or atherosclerotic heart disease chronic congestive failure arrhythmias. Previous emboli, hypotension are high risk. Other factors include hypovolemia, diuretic use, Vasoconstrictive drugs, including. Digitalis, recent Ml, treatment in a special medical unit or oxygen therapy. More recently identified factors are dialysis, decompression feeding and exercise.

Results of laboratory tests in patients with ischemia

Expected Findings

1. Significant leukocytosis
2. GIT Blood loss
3. Hemoconcentration

Limited Utility

1. Increased Serum Amylase
2. Increased Serum Phosphate
3. Increased Serum L.D.H. SGOT , Creatinine Phosphokinase alkaline phosphatase
4. Increased lactic acid
5. Base Deficits
6. Increased Serum ammonia
7. Increased lysosomal acid hydorlases

8. Hypoglycemia
9. Persistent metabolic acidosis after shock is corrected

Conditions associated with arterial occlusion

Emboli, Polycythemia vera, Thrombus, Ergot alkaloids, Ligature, Dermatomyositis, Aneurysm, Infective angiitis, Trauma, Infective angiitis, T.A.O., Infective angiitis, S.L.E., Ehlers Donlos syndrome.

Causes of venous occlusion leading to ischemia

Idiopathic, Hereditary protein's deficiency, Hypercoagulation, Polythemia Vera, Carcinoma (Pancreas, Stomach Colon), Portal hypertension, Pancreatitis Splenectomy, Inflammatory bowel disease, Oral contraception, Immunotherapy, Endoscopic Sclerotherapy

SCREENING TESTS

Ultrasound

US can be sued for follow-up after CT has shown an occlusion us has been widely used for examination of the portal system and IVC but its use in mesenteric system is less secure in CT even isolated occlusions are seen. Changes in the mesentery or bowel wall. Seen with CT. The rigid loop sign thumb printing which is well described on abdominal radiograms, abnormal gas patterns can be clearly seen with CT.

Isotope Scanning

The IV route of isotope administration permitted diagnosis of intestinal ischemia in 1hour a good advance late diagnosis was not possible

Tonometry

Tonometry, involves the indirect measurement of intramural PH to detect acidosis secondary to gut wall changes when oxygen delivery fails below tissue need.

Other methods that measure or reflect mucosal metabolic states include luminal PCO2. H2 gas clearance.

Endoscopy

Endoscopic examination of distal duodenum or proximal jejunum reveals diffusely, edematous purple-red in color necrotic and aprestic looking mucosa.

Angiography

The initial study should be an aortogram with both AP and Lt. Projections to check for aortic atherosclerosis and significant collateral flow. The absence of atherosclerosis essentially preludes the diagnosis of acute thrombosis, atherosclerotic of the superior mesentric artery is an extension of disease from the aorta thus the occlusion is at the origin and proximal 1 0 3 cm. In contrast embolic occlusion of the superior mesentric artery occurs at a branch point sparing the proximal 1 to 3 cm.

Treatment principles of extensive ischemia [4, 6]

1. Replace blood volume
2. Antibiotics
3. Support haemodynamics
4. Control of free radicals
5. Luminal agents

Arterial

a. Emboli Embollectomy Vasodilatation Preop and Postop
b. Thrombus - Bypass graft Vasodilatation -Routine second lock operation.

Venous

a. Thrombectomy if it early - Second looks operation
b. Bowel resection if it late - Second look operation

Second Look Operations

As a concept the second look operative procedure is applicable to most of these patients because the extent of resection must be limited in these cases of extensive ischemia and because confirmation of viability especially early after reperfusion is lacking.

Intraluminal therapy

Intraluminal therapy, which has a good basis in experimental work. The stimulus for an adaptive cytoproliferative mucosal response includes luminal glucose and O2. Other agents of potential value include antibiotics, dimethyloxide, perfluorocarbons, trypsin and chemotrypsin inhibitors. As with other organs subjected to reperfusion the intestines sustain additional damage that appears to be related to reactive oxygen metabolites, Therapy with free radical scavengers has been extensively evaluated in laboratory experiments.

Vasodilators

The last group of treatments that might be used for all. Patients with extensive Ischemia involve agents to produce splanchnic vasodilatation or to prevent splanchnic vasoconstriction.

Specific Measures [4, 6]

Embolectomy

Arterial emboli are treated by embolectomy unless they are very distant. The arteriotomy is closed by patch of graft Grossly infarcted bowel is removed when questionable bowel must be left a second look is done 6 to 24 hours postoperatively.

Selective intrarateriał therapy using papaverine dextran, heparin streptokinase are. tried, in mesentric ischemia.

Acute Arterial Thrombus

It carries 80% of deaths proper treatment generally is by pass grafting. The origin of the by pass is most often in the distal aorta.

Limited Mesentric Ischemia

Limited ischemia can occur in several forms limited length limited depth or limited time of which ischemic colitis is most common form.

Ischemic Colitis

Etiology: Unclear

Classification: Severe

Chronic Stricture

Mild

Location: Anywhere caecum to rectum.

Diagnosis: Mucosal ulcers hemorrhages are contrast studies.

Treatment: No peritonitis observation, Peritonitis resection, stricture elective resection.

ACUTE ABDOMINAL DISEASE WITH GENITOURINARY SYMPTOMS

RENAL COLIC

Pain of the renal colicky type may be caused by: [18]

a. Stone in the pelvis of the kidney

b. Stone in the ureter

c. Blood clot or ureteric debris in the ureter

d. Appendicitis

In renal colic, the pain starts in the loin and frequently but not always radiates to the groin or to the corresponding testicle. It may be due to anything solid or semisolid passing down the ureter. There is usually no difficulty in diagnosis, though occasionally appendicular colic or even appendicitis may cause pain of a similar nature. But in appendicitis severe enough to cause the simulation of renal colic, there will usually be persistent local muscular rigidity, which is not usually found in renal colic. The pain of renal colic is one of the most severe encountered by a patient.

Disorders of micturition

Pain or burning on urination or frequency of urination is found in nearly all cases of pyelitis and in many cases of renal colic, but it is often also noted in appendicitis and other causes of pelvic peritonitis. Examination of the urine should prove or disprove a pyelitis.

In appendicitis, pain on or frequency of urination may be due to irritation of the renal pelvis, ureter, or bladder by the inflamed appendix or contiguous peritonitis. When this symptom is accompanied by pelvic tenderness, a tender lump is felt per rectum, or a positive obturator test is obtained, the appendix will usually be found in the pelvis irritating the bladder. This set of symptoms is found whenever an inflammatory mass lies adjacent to the bladder. Thus, sigmoid diverticulitis or Crohn's

disease may cause urinary frequency, urgency, fever, and a pelvic mass. These two conditions must always be considered under these circumstances. When the same symptoms are unaccompanied by the other signs, the appendix is generally either near the kidney, where it may irritate the renal pelvis, or at the brim close to the right ureter.

In pelvic hematocele due to a ruptured ectopic gestation, there are frequently urinary symptoms. Sometimes there is retention of urine, sometimes-slight pain or frequency. In a very anemic woman with abdominal pain and urinary symptoms, it is well to think of ectopic gestation. Acute distention of the bladder may cause very severe hypogastric pain and sometimes pain in the lower lumbar region; there should be no difficulty in palpating or percussing out the distended bladder, and one must not be misled by the fact that urine may be passed by the process of overflow incontinence. In those rarer cases in which the bladder does not distend upward but rather backward and upward, it may be difficult to make out its position by percussion, but bimanual examination will determine it, and the passing of a catheter will help the diagnosis.

Abnormalities of urine

Hematuria frequently follows renal colic and may sometimes enable one to trace to its source a renal pain that was not quite typical.

Albuminuria is an exceedingly important finding, for uremic symptoms may very closely simulate intestinal obstruction, and it may only be by the discovery of albuminuria that one is put on the right track. Toxic albuminuria is found in many septic states of the abdomen, but it is usually not difficult to judge when the toxemia is severe enough to produce that symptom. In attacks of acute porphyria, the urine turns brown or reddish-brown on standing.

Testicular Pain [18]

Pain in the testicle is met with in renal colic and in a few cases of appendicitis. Ruptured aortic aneurysm with retroperitoneal hemorrhage often causes unrelenting testicular and groin pain.

Torsion of the imperfectly descended testicle may cause extreme pain in the inguinal region and may stimulate a strangulated inguinal hernia. Vomiting occurs and shock may be severe. The pain has the usual sickening character of testicular pain. The absence of the testicle from the same side of the scrotum will be noted and will make diagnosis easy.

Thrombosis or suppurative phlebitis of the veins of the spermatic cord causes pain in the inguinal region and sometimes in the iliac region of the abdomen. But the swelling and the painful area extend right down to the testicle, which becomes swollen and painful.

Retraction of the testicle is occasionally noted in cases of appendicitis. It is due to reflex contraction of the cremaster muscle.

Tenderness at the Erector-Costal Angle

Tenderness on pressure at the right erector-costal angle (i.e., the usual position for eliciting tenderness of the right kidney) is noted in many cases of appendicitis, especially when the appendix is retrocecal in position.

Renal symptoms may be caused by any retroperitoneal lesion in the region of the renal pelvis; thus a duodenal ulcer leaking posteriorly or a retroperitoneal perforation of the common bile duct may cause frequency of micturition or even slight hematuria. Pancreatitis must never be forgotten as an important cause of left flank pain and tenderness.

THE ACUTE ABDOMEN IN THE TROPICS [6]

Every surgeon intending to practice in the tropics needs to have a good knowledge of tropical medicine and must be on guard when one of the main symptoms is acute abdominal pain, for the common types of acute abdominal crises differ from those seen in temperate climes. In general, the pattern of acute abdominal disease varies with the climate and sanitary conditions and with the diet and habits of the inhabitants. Some of the acute inflammatory conditions, for example, acute appendicitis and acute cholecystitis, are less common in the tropics, particularly in the remoter regions and among the native peoples. On the other hand, there are a number of acute abdominal conditions that are common in the tropics but seldom seen elsewhere. Special mention must be made of those conditions consequent on malaria, sickle-cell anemia, amoebiasis, infestation with worms, and the results of excessive heat.

Symptoms of amebic hepatitis

There are five constant features and many inconstant symptoms to be looked for in amoebic hepatitis. The constant features are:

1. Fever
2. Enlargement of the liver
3. Pain and tenderness in the hepatic region
4. Leukocytosis
5. Rapid subsidence of the symptoms under treatment by emetine or other amoebicidal drugs

The common though inconstant features are:

1. A history of an attack of dysentery
2. Pain on top of the right shoulder or in the right iliac region

3. Jaundice

4. A rigor

5. Malaise and general lassitude

Infestation with Worms

Infestation with worms is common in many parts of the tropics where sanitary precautions are deficient or absent, particularly in the central zone of Africa, in India, and in southern China. Abdominal symptoms are frequently caused by infestation with roundworms (Ascaris), which may multiply in the jejunum until a large mass results and may cause intestinal obstruction. More rarely, one or more worms may penetrate the wall of the intestine and cause local peritonitis or an abscess. Ascaris may also obstruct the appendix or the common bile duct, with consequent cholangitis.

Filariasis is prevalent in some parts of India, and when it affects the retroperitoneal lymphatics on the right side, it may simulate appendicitis. Fever to 103° to 104°F with a rigor usually precedes pain, which generally emanates from the right iliac fossa. Nausea and vomiting usually occur. There is often mild tenderness but no guarding or rigidity. There may or may not be associated epididymo-orchitis.

Abdominal symptoms due to salt deficiency

In tropical climes, as the result of the great heat and consequent excessive sweating, there may result a state of salt deficiency. This may give rise to severe cramps that may affect the muscles of the abdominal wall and cause such great pain, collapse, rigidity, and tenderness that the observer may even diagnose the condition as one due to perforation of a peptic ulcer. A quick test of the serum electrolytes and haematocrit should suffice to make the diagnosis so that prompt treatment can be instituted. Symptoms usually subside quickly upon administration of intravenous

sodium chloride solutions.

Pyomyositis

Pyomyositis or suppurative inflammation of the muscles may affect the muscles of the abdominal wall and cavity and simulate an intraabdominal crisis. This infection, usually with staphylococcus aureus, is more common in AIDS-infected persons. If the parietal muscles are involved, there will be fever, local tenderness, and swelling of the abdominal wall, with perhaps the involvement of muscles elsewhere; absence of evidence of a deep-seated lesion should serve to discriminate. When pyomyositis affects the flat part of the iliacus muscle, it may easily simulate an appendicular abscess.

DISEASES THAT MAY SIMULATE THE ACUTE ABDOMEN

There are a number of diseases that either do not need or positively contraindicate operative interference, yet these may cause symptoms very suggestive of conditions for which operation is the best procedure. In some cases the symptoms arise from disease within the abdomen; in other instances the pain is referred to the abdomen from another part of the body (e.g., thorax: or spine).

General disease

It is not uncommon for abdominal pain and vomiting to occur at the onset of some of the specific fevers or of influenza, especially in children, but in such cases the general symptoms outweigh the local manifestations. Fever will be present at the start and the general malaise is greater, while locally there will not be found the tenderness or rigidity that suggests intra-abdominal inflammation; it is by this discrepancy that the observer will be guided.

Diabetes

Impending coma in diabetes is often accompanied by severe abdominal pain

and vomiting. There may also be some rigidity and tenderness of the abdominal wall. The way is then clear for a misdiagnosis of acute inflammation within the abdomen and, indeed, mistakes of this nature have been made.

Typhoid fever

This is sometimes accompanied by abdominal pain and local tenderness, especially in the right iliac fossa.

Malaria

In tropical climes, malaria frequently causes severe abdominal pain, but the type of fever and an examination of the blood should enable one to exclude malaria.

Tuberculous peritonitis [6]

This may cause vague abdominal pains, distention of the abdomen, and free fluid. These symptoms may occasionally give rise to the opinion that there exists some acute abdominal condition. The gradual onset of symptoms, the tumidity of the abdomen, the lack of rigidity and tenderness, and the presence of tuberculosis elsewhere in the body may be sufficient to lead to the correct diagnosis.

It must be remembered, however, that intestinal obstruction and perforative peritonitis sometimes occur in the course of tuberculous peritonitis, and these demand operative treatment. Sometimes, when the ileocecal region is extensively involved in the tuberculous process, the gut may be adherent in the iliac fossa and the simulation of appendicitis may be very close.

Food poisoning

This may give rise to abdominal pain, vomiting, and collapse. There is a serious pitfall here. Many patients who have a ruptured appendix or stomach attribute the trouble to the eating of some particular article of diet. One may therefore miss a condition needing surgical intervention, just as one may think an operation necessary

when there is no need for interference. In food poisoning, the symptoms usually follow soon after eating some article of diet suspected to be tainted. Frequently several people are simultaneously attacked. In any case, though the general symptoms are similar, the local abdominal condition is unlike that of peritonitis (no rigidity) or severe obstruction (absence of feculent vomiting), and there should usually be no difficulty in diagnosis if a careful watch is kept upon the patient.

Lead colic

Lead colic is form of colic of the small intestine that is accompanied by constipation. The pain may be extreme and collapse of the patient may be severe. During the spasms of pain, the abdominal wall may be rigid. Other signs and symptoms pointing to lead poisoning (blue line on the gums, severe constipation, local paralysis etc) may be present.

Epidemic pleurodynia

Epidemic pleurodynia or Bornholm's disease (epidemic myositis) sometimes has an onset that may cause it to be mistaken for an acute abdominal affection.

Blood diseases

Acute Porphyria

This may be accompanied by attacks of acute abdominal pain that have, on occasion, been mistaken for appendicitis or intestinal obstruction. The condition should particularly be borne in mind when the patient has been taking drugs of the sulfonamide or barbiturate groups.

Leukemia

Leukemia present as an acute abdominal emergency. The patient gave a history of prolonged indigestion, had suffered recently from irregular fever, and with an anemic appearance presented also great tenderness, rigidity, and dullness in the left

hypochondrium, so that the simulation of subphrenic abscess due to a leaking ulcer was rather close.

Attacks of severe abdominal pain accompanied by vomiting (and sometimes diarrhea) occur in some cases of pernicious anemia but seldom give rise to serious difficulty in diagnosis. We have elsewhere mentioned the acute abdominal pains that may occur during the course of sickle-cell anemia in blacks.

Hemolytic crises

It is well known that this condition is sometimes accompanied by abdominal crises in which acute abdominal pain, nausea or vomiting, fever, and tenderness on palpating the abdomen are noteworthy features. The attacks coincide with an increase in the depth of the jaundice and the intensity of the anemia. The cause of the abdominal pain during a hemolytic crisis is unknown. Since many of these patients have pigment gallstones, considerable difficulty may arise in diagnosis. The lack of typical features of biliary colic or cholecystitis together with evidence of major hemolysis and anemia usually serve to distinguish hemolytic crises.

Thoracic diseases [2, 6]

Pleurisy or pleuropneumonia

Either of these conditions may cause abdominal pain and rigidity and may be accompanied by vomiting. Whether the pleurisy results from pneumonia or a pulmonary infarct, abdominal pain is often present. A tabulation of the main differential points is given in Table.

An additional test is illustrated in Figure. If the pain is unilateral and of abdominal origin, pressure from the opposite side of the abdomen toward the affected side will cause pain, while if the pain is referred from the thorax, no pain is caused by such pressure.

Acute cardiac disease

This frequently causes symptoms referable to the abdomen. Epigastric pain and tenderness are common when the liver is congested and swollen from back pressure, but nausea and vomiting are unusual despite intense pain. Vomiting is not an infrequent symptom in myocardial infarction, and severe collapse may usher in an attack of pericarditis or accompany acute cardiac failure. Needless to say, in any case of doubt, the circulatory system must be very carefully examined. In some instances doubt remains even after one has measured the cardiac and hepatic dullness, listened to the cardiac sounds, noted carefully the character and rate of the pulse, measured the blood pressure, and observed if there is any venous pulsation. Sometimes the electrocardiogram will be of invaluable assistance in distinguishing between myocardial infarction and biliary colic. However, it may be of little help in deciding between biliary colic and angina pectoris.

Table 7: Comparative table of symptoms in acute abdominal and acute pleural or pneumonic lesions

Abdominal	Pleural or pneumonic
Previous history	
Indigestion	Common cold or "chill"
Colicky pains	Exposure to infection or previous operation
Constipation	
Diarrhea	
Onset	
Acute without fever (except pyelitis)	Acute with fever at start

Rigor unusual (except pyelitis)	Rigor common
Vomiting usual	Vomiting less common
Pain often shifts downward	Pain thoracic as well as abdominal
Examination	
Appearance	Cheeks flushed
Varies from normal to "abdominal" facies	Alae nasi working
	Sometimes herpes on lips
Skin	
Cold or clammy or normal	Skin may be hot and dry
Pulse and respiration	
No sure guide	P:R ratio lessened
Abdominal wall	
May be rigid	Less commonly rigid
Phrenic shoulder pain	
Common, but seldom below clavicle	Common, especially below clavicle
Psoas test	
Often positive	Always negative
Obturator test	
Sometimes (rarely) positive	Always negative
Testicular pain	
Sometimes present	Never present
Rectal examination	
May elicit tenderness or demonstrate lump	Negative

Examination of chest	
Frequently slight rubs in upper abdominal lesions	May be a rub or dullness or bronchial breathing, but sometimes nothing definite at onset of symptoms

Disease of the spine or spinal cord

Pain of nerve root origin

One of the most frequently overlooked causes of abdominal pain is irritation of one or more nerve roots, despite the fact that a careful history and physical examination will usually point clearly to this source. I have been asked repeatedly for consultation about patients with "puzzling abdominal pain" who come with plain films, upper and lower barium contrast meals, endoscopies, and abdominal CT scans. The causes of the nerve root irritation have included spinal osteoarthritis, spinal stenosis, and spinal cord or nerve root tumors.

Osteomyelitis

Acute osteomyelitis of the dorsal or lumbar vertebrae may cause abdominal pain and rigidity, but there will be great tenderness on pressure over the affected part of the spine that will draw attention to the origin of the pain.

In children, acute abdominal (epigastric or umbilical) pain may be consequent on Pott's disease of the spine. The absence of abdominal signs would naturally cause examination of the spine and detection of the spinal disease.

Tabes dorsalis

This condition, now rarely seen, causes severe abdominal pain in the form of gastric crises. The crises, though more common in adults, may also occur in children who are the subject of juvenile tabes. The pains may be very severe, and uncontrollable vomiting may occur. The important point to remember is that the

abdominal wall is not rigid in the intervals of the pain of a gastric crisis.

Herpes zoster

Herpes zoster affecting any of the lower dorsal spinal roots may cause pain referred to the anterior or lateral parts of the abdominal wall. When the pain precedes the vesicular eruption, it may give rise to the suspicion of intra-abdominal trouble in need of surgery. The pain is sharply delimited by the segmental distribution of the affected nerves and, unless there are other signs, the appearance of the vesicular rash will explain the nature of the pain.

Renal Disease

Serious disease of the kidneys may cause uremia, which may be accompanied by vomiting and abdominal distention. Thus intestinal obstruction may be closely simulated.

This may occur in acute nephritis, chronic nephritis, bilateral cystic disease of the kidneys, or pyonephrosis.

If, in every case of intestinal obstruction, one remembers the possibility of uremia, there should be no great difficulty in diagnosis by considering the differential points set out in Table.

Table 8: Differential diagnosis of renal disease and intestinal obstruction

Intestinal Obstruction	Uremia simulating obstruction
Vomiting tends to become feculent	Vomiting not feculent
If obstruction low down, absolute constipation of flatus and feces	Bowels may return flatus after enema
May be history of subacute attack of obstruction	May be history of some surgical or medical disease of kidneys

No renal tumors	In cystic disease, bilateral renal tumors found
Blood pressure may be normal	Blood pressure likely to be much raised

It is very seldom that the two conditions are confused when once the possibility of uremia is considered.

Other causes are: Periarteritis Nodosa, Familial mediterranean fever, Arachnidism and adrenal insufficiency.

METHODOLOGY

This is a study of non traumatic acute abdomen and management, 100 consecutive cases was done in R.L. Jalappa Hospital & Research centre attached to Sri Devaraj Urs Medical College.

Only patients with acute abdominal pain, who underwent surgery, have been included. Pain abdomen in pediatric age group, traumatic acute abdomen, Medical condition, Gynecological conditions, recurrent pain abdomen patients were excluded from study, because a correct diagnosis could be established only then.

Preoperative detailed history and thorough physical examination was done for all acute abdominal emergencies, to arrive at preoperative diagnosis.

Informed consent was taken from all patients for surgical intervention.

After admission routine investigations namely Hb%, TC, DC, BU, SC, urine examination are carried out. Relevant procedure like a plain x-ray abdomen and US abd was taken in some cases. In all the cases operative findings and postoperative diagnosis were recorded.

Postoperative follow-up was done at least 6 month period to note complications and outcome with investigations for reflex.

To arrive at conclusion, 100 cases have been studied.

RESULTS

TABLE 9

Incidence of acute abdominal conditions in 100 cases

Sl. No.	Cause of Acute Abdomen	1	2	3	Mortality
1	Perforated duodenal ulcer	37	16	19.3	2
2	Acute Appendicitis	25	23.5	8	0
3	Acute cholecystitis	14	18.6	9	0
4	Ileal perforation	8	2.8	6	0
5	Acute Intestinal Obstruction	7	10.8	21	1
6	Gastric perforation	6	8	4	0
7	Volvulus	2	1.4	0.9	0
8	Acute Mesenteric ischaemia	1	-	-	0
	Total	100			3

1) *%of own study* ***2)*** *Study conducted at University of Ghana Medical School, Accra.*

3) *Dept. of General Surgery, Government Medical College and Hospital (GMCH), Chandigarh*

100 cases of acute abdominal conditions from December 2009 to April 2011, which were operated in R.L. Jalappa Hospital & Research centre, the leading cause of acute abdomen was duodenal perforation constituting 37%.

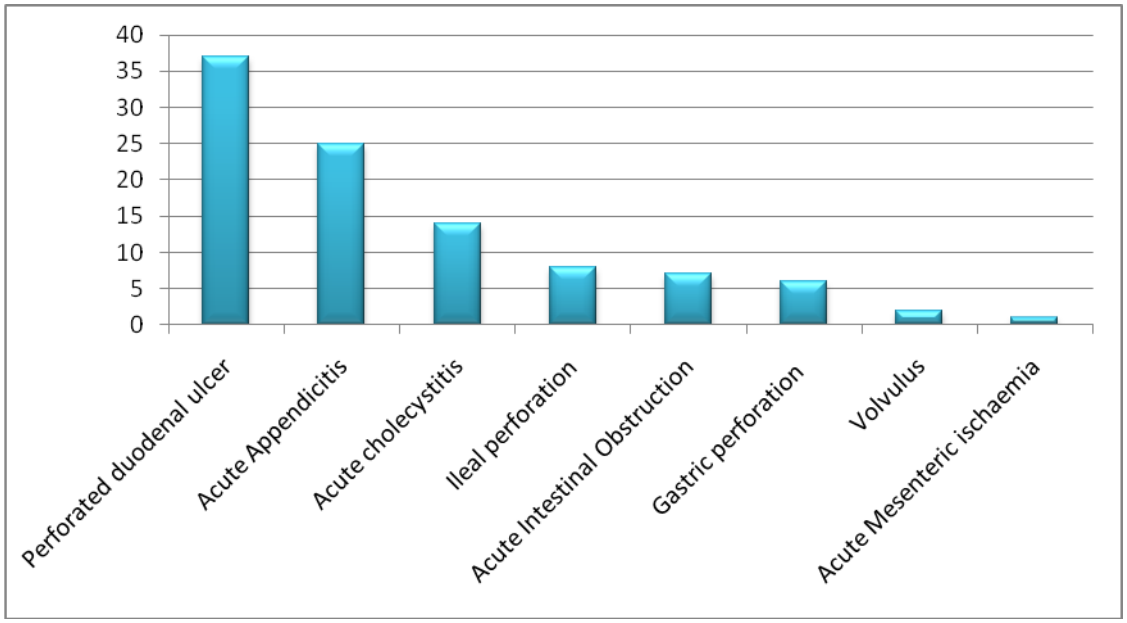


TABLE 10

Age distribution of acute abdominal cases

Age in years	Ac. App	Intestinal Obstruction	Perforation	Acute Cholecystitis	Acute Mesenteric ischemia	Total
21-30	22	1	17	-	-	40
31-40	2	2	9	3	1	17
41-50	-	-	13	3	-	16
51-60	1	1	6	5	-	13
61-70	-	3	4	3	-	10
71-80	-	2	2	-	-	4
81-90	-	-	-	-	-	-
91-100	-	-	-	-	-	-
	25	09	51	14	01	100

This table shows that majority of the patients are in the age group of 21-50 years. 14 patients were above 60 years. Youngest patient in this group was 21 years and eldest patient was 80 year old.

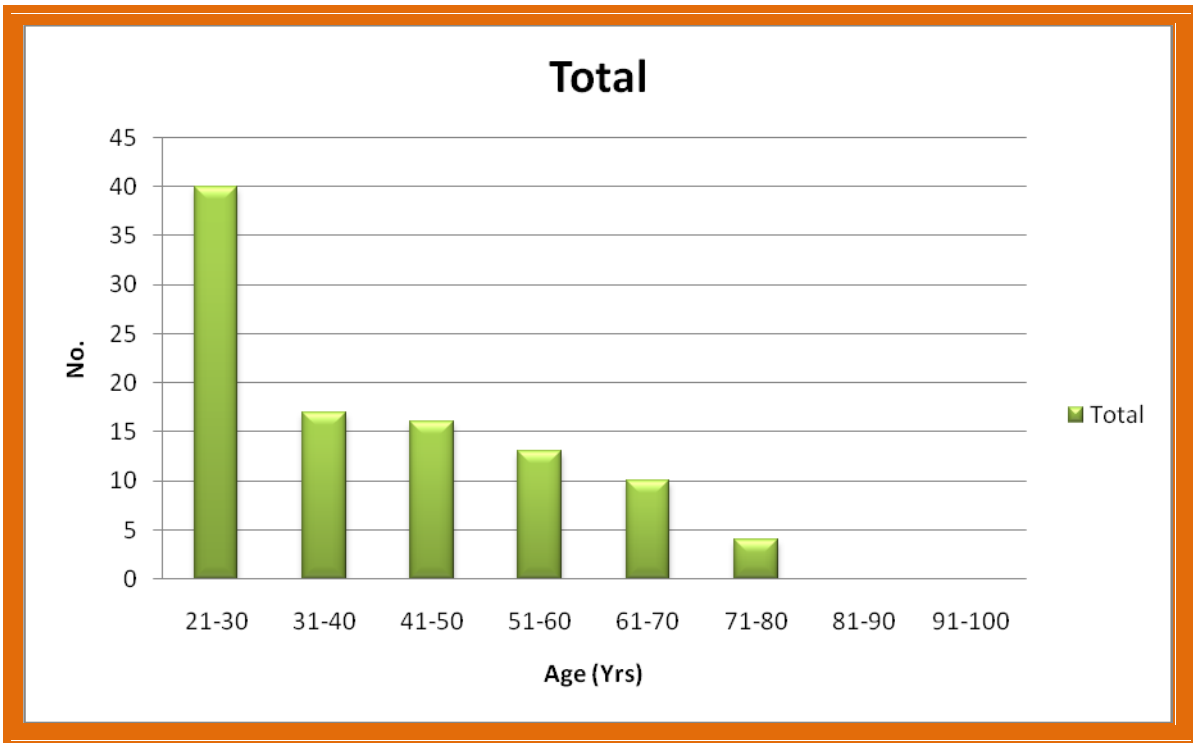


TABLE 11

Sex Incidence of acute abdomen

Disease	**Total No.**	**Male**	**%**	**Female**	**%**
Appendicitis	25	15	23.08	10	28.57
Hollow viscus perforation	51	42	64.62	9	25.71
Acute Intestinal obstruction	9	8	12.30	1	2.86
Acute Cholecystitis	14	--	--	14	40
Acute Mesenteric ischaemia	1	--	--	1	2.86
Total	**100**	**65**	**100.00**	**35**	**100.00**

Out of 100 cases the acute abdomen 65 cases were males, 35 cases were females.

Out of 51 cases of Hollow viscus perforation 42 were males, 9 were females.

25 patients diagnosed to have acute appendicitis in which 15were males and 10 females. Intestinal obstruction was found to be common in males than females (8:1).

In general, all types of acute abdomen has got preponderance in males.

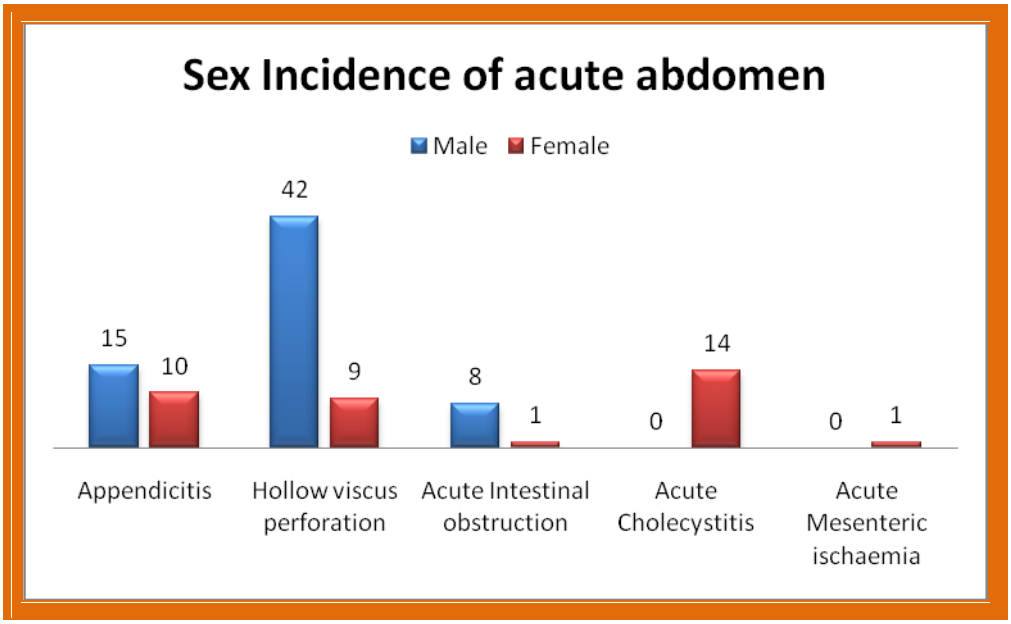


TABLE 12

Incidence of Hollow viscus perforation (Excluding Appendicular perforations)

Hollow Viscus Perforation	No. of Cases	%
Perforated duodenal ulcer	37	72
Ileal perforation	8	16
Gastric perforation	6	12
Total	51	100

Author [Ref]	Cases	Duodenal Ulcer Perforation n (%)	Gastric ulcer Perforation n (%)	Perforation of Gastric Carcinoma n (%)
Khan 2004[11]	21	16 (76.2)	5 (23.8)	0
Siu 2001[25]	121	83 (68.6)	29 (23.9)	-*
Chan 2000[17]	206	196 (95.1)	10 (4.8)	0
Dorairajan 1995[2]	80	74 (92.5)	5 (6.2)	1 (1.2)
Sugimoto 1994[60]	101	90 (89.1)	11 (10.8)	0
Wakayama 1994[18]	136	110 (80.9)	19 (13.9)	7 (5.1)
Sharma 1991[5]	47	45 (95.7)	1 (2.1)	1 (2.1)

Out of 100 patients in our study 37 (72%) had duodenal perforation, 8 (16%) patient had ileal perforation and 6 (12%) had perforated gastric ulcer.

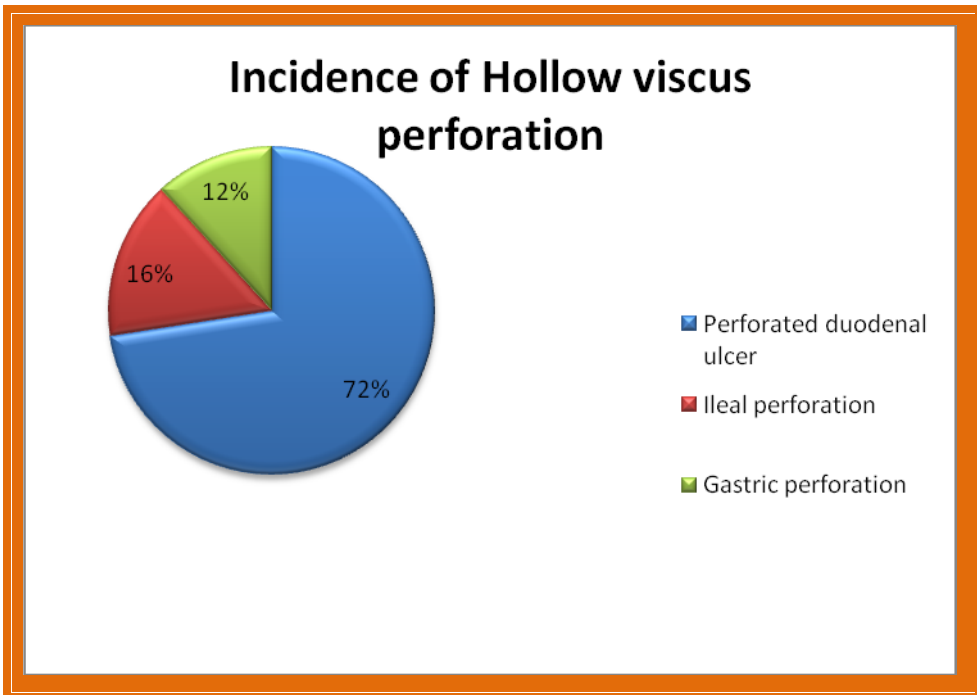


TABLE 13

Position of Appendix

Sl. No.	Position of Appendix	No. of Cases	%
1	Retro-caecal	20	80
2	Pelvic	1	4
3	Pre-ileal	1	4
4	Paracaecal	2	8
5	Subhepatic	1	4
	Total	25	100

Out of 25 cases of acute appendicitis in which position of appendix was delineated, the most common position of appendix in our study was retrocacecal, constitute 80%.

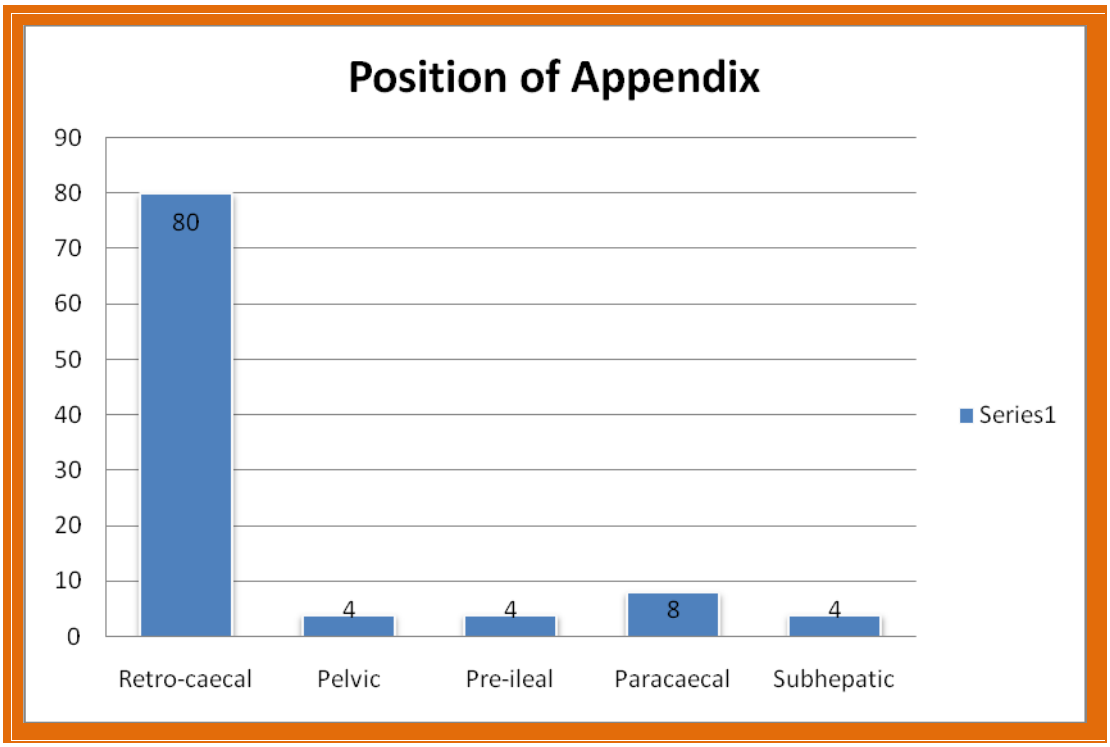
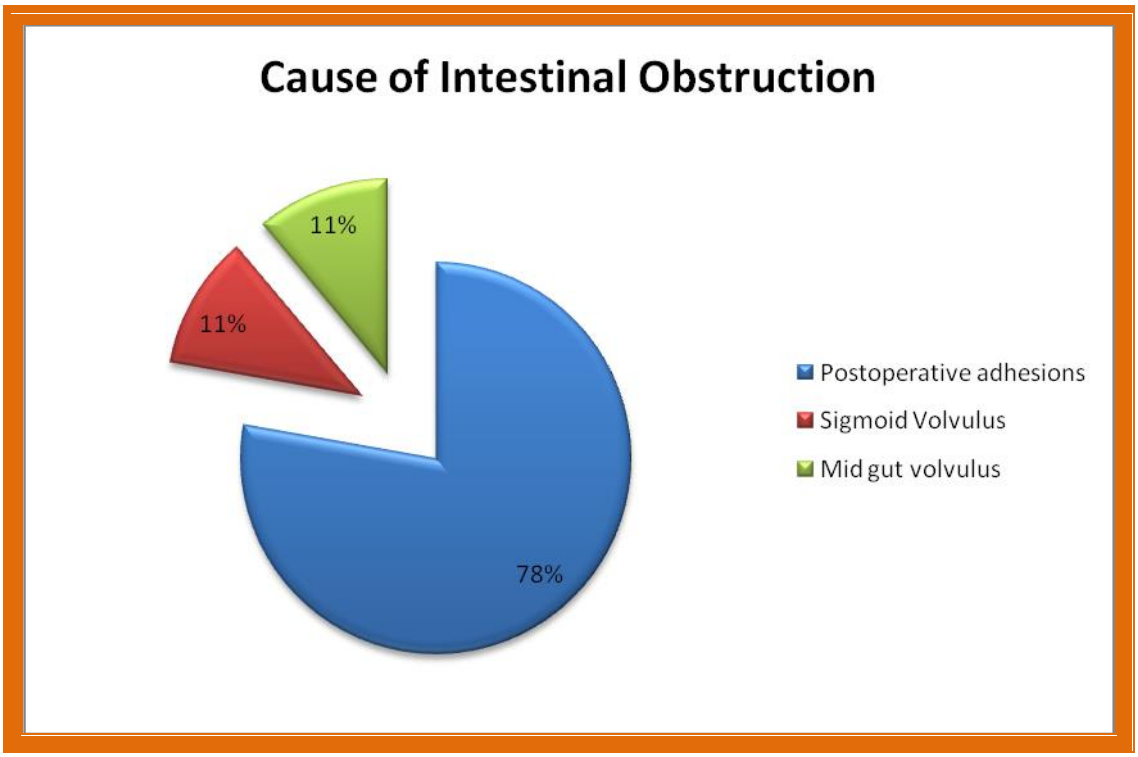


TABLE 14

Cause of Intestinal Obstruction

Sl. No.	Cause of Intestinal Obstruction	No. of Cases	%
1	Postoperative adhesions	7	77.78
2	Sigmoid Volvulus	1	11.11
3	Mid gut volvulus	1	11.11

Out of 9 cases intestinal obstruction, the commonest cause was post operative adhesions between bowel loops 7(77.78%).


Cause of Intestinal Obstruction
11%
11%
78%
Postoperative adhesions
Sigmoid Volvulus
Mid gut volvulus

DISCUSSION

Acute abdomen of non traumatic origin was included in this study, to ensure correct diagnosis. Totally 100 cases were studied.

The leading cause of acute abdomen in our study was perforated duodenal ulcer comparing with other studies shown in Table no.9, constituting 37% of cases of which exploratory laparotomy and Graham's omental patch was done in 37cases and mortality was 2 %(2 cases).

The second common cause of acute abdomen was acute appendicitis constituting 25% of cases of which mortality was nil.

The third common cause was acute cholecystitis accounting for 14% (14 cases) of cases of which mortality was nil.

Hollow viscus perforation

Hollow viscus perforation was the commonest cause of acute abdomen. Diagnosed in 50 cases: 37 were duodenal perforation, 7 ileal perforation and 6 gastric perforations. 2 cases of acute abdomen secondary to duodenal perforation expired (mortality 2%). Gastric perforations cases were secondary to analgesic and spicy food intake.

All cases of duodenal perforation and gastric perforations were treated with simple closure and pedicle omental graft (Graham's patch) and started with anti H.pyloric medications for 14 days. Patients responded well to treatment to above treatment.

All cases of ileal perforation were secondary to enteric fever, for which primary closure (two layered) was done.

Acute appendicitis

Out of total 25 cases studied 2 were gangrenous and 21 were inflamed, which were treated by appendicectomy. One case with diagnosis of appendicular mass was managed conservatively. The appendicular mass was closed without disturbing the mass. In both cases interval appendicectomy was done after 6 weeks. And for appendicular perforation exploratory laparotomy with appendicectomy was done. The commonest position of appendix seen was retrocaceal 20(80%).

Intestinal obstruction

Out of 9 cases of intestinal obstruction, 7 cases were due to post-op adhesions between bowel loops, 1 due to sigmoid volvulus, 1due to mid gut volvulus. 7 cases due to post op adhesions were taken for laparotomy and adhesiolysis was done. Sigmoid volvulus for which resection and anastamosis of sigmoid colon and fixing was done. The other cause was due to mid gut volvulus for which derotation and fixing was done.

Acute abdomen is a clinical entity, more commonly seen in males (M: F: 65:35) and patients in their 3rd decade of life, less common in extremes of age groups.

Peritonitis secondary to hollow viscus perforation was seen between the age group of 20-60 years.

Acute appendicitis was more common in second and third decade. It is uncommon after 40 years. Appendicitis was common in individuals from higher socio economic status.

Intestinal obstruction was more common in 3rd to 4th decade.

Out of 51 cases of Hollow viscus perforation 42 were males and 9 were females, 8 cases were males in intestinal obstruction 1 cases were females. Of 25 cases of acute appendicitis males were 15 and females 10.

Out of 100 cases of acute abdomen, which underwent surgery, 3 cases expired during post-operative period. Two were due to duodenal perforations; one was secondary to intestinal obstruction. 1st case (Patient No.45) of duodenal perforations was presented with shock. Following resuscitation, flank drain was kept. After stabilization patient was taken for laparotomy and duodenal perforation was closed with Graham's omental patch. Post operatively patient developed acute renal failure, dialysis was done and on 7th day patient died because of septicaemic shock. 2nd case (Patient No. 74) of duodenal perforations was presented with shock. Following resuscitation, flank drain was inserted. After stabilization patient was taken for laparotomy and duodenal perforation was closed with Graham's omental patch. Post operatively patient developed multi organ failure and expired on post op day 3. 3rd case (Patient No. 25) of intestinal obstruction was due to post operative adhesions, the distal ileal was gangrenous for which resection and ileo-ileal anastomosis was done. On the 3th postoperative day patient developed a renal failure, underwent haemodialysis once and expired on 7th post operative day due to septic shock.

CONCLUSION

- The majority of patients suffering from pain abdomen & getting investigated & admitted in the hospital are having conditions like **Non-specific abdominal pain** in which no obvious cause can be found following investigations and even laparotomy. **Acute appendicitis**, **Intestinal obstruction, Gynecological causes.**
- We have to always borne in mind, possibility of medical or non-surgical causes of pain abdomen like viral infection & diaphragmatic pleurisy, acute myocardial infarction and sickle cell anemia.
- In our study perforated peptic ulceration, predominantly duodenal has become a relatively common cause surgical emergency caused by acute ulcers, result of medical treatment with either steroids or non-steroidal anti inflammatory agents given for PUO, followed by acute appendicitis. It needs common radiological imaging like plain X-ray abdomen & Ultrasonography abdomen to confirm clinical diagnosis in our rural centre.
- The first step in diagnosing the cause of acute abdominal pain is to elicit a proper history & perform thorough clinical examination & elicit physical signs to arrive at appropriate clinical anatomical diagnosis.
- It is important to keep abreast of all developments within the general surgical field & emphasis should be given to elicit precise history & thorough investigations before subjecting patient for surgical management.

SUMMARY

The present study is a clinical study of 100 consecutive cases of acute abdomen of non-traumatic origin who underwent surgery in R.L. Jalappa Hospital & Research centre attached to Sri Devaraj Urs Institute of Medical sciences from December 2009 to May 2011, which were operated in One hundred consecutive cases of acute abdomen admitted are analyzed and presented.

Usual age incidence is in the second to fourth decade, forming majority of the total cases analyzed.

Acute abdomen is more common in males. The commonest cause of acute abdomen was hollow viscus perforation. The 2nd common cause of acute abdomen was acute appendicitis and the position was retrocaecal followed by acute calculus cholecystitis, intestinal obstruction.

In majority of cases, leucocytosis and neutrophilia was a common finding. Exploratory laparotomy with Graham's omental patch was done in 43 cases of hollow viscus perforation. Morbidity and mortality was high due to septis.

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PROFORMA

Name:

Age: **Sex:**

Address:

Occupation: **Religion:** **I.P.No.:**

DOD: **DOA:** **DOS:**

PRESENTING COMPLAINTS:

Pain Abdomen: Vomiting:

Abdominal Distension: Fever:

Constipation:

HISTORY OF PRESENTING ILLNESS:

PAST HISTORY:

PERSONAL HISTORY:

Diet: Appetite: Bowel Habits:

Bladder Habits: Sleep: Habits:

Marital status:

Menstrual History:

Obstetric History:

FAMILY HISTORY:

GENERAL EXAMINATION:

Built: Nutrition: Hydration:

Pallor: Icterus: Cyanosis:

Clubbing: Oedema: Lymphadenopathy:

VITAL SIGNS:

Temp: Pulse: BP: RR:

SYSTEMIC EXAMINATION:

ABDOMEN:

Inspection:

Palpation:

Percussion:

Auscultation:

External Genitalia:

Rectal Examination:

RS:

CVS:

CNS:

PROVISIONAL DIAGNOSIS:

INVESTIGATIONS:

Haemogram:

Bleeding Time: Clotting Time:

Urine routine:

RBS: Blood Urea: S. Creatinine:

S. Electrolytes:

ECG:

Plain Erect X-ray Abdomen:

USG Abdomen:

CT Abdomen:

PRE OPERATIVE TREATMENT:

OPERATIVE DETAILS:

POST-OPERATIVE DIAGNOSIS:

POST-OPERATIVE TREATMENT:

HISTOPATHOLOGY REPORT:

WOUND CULTURE & SESITIVITY:

FOLLOW UP:

KEYS TO MASTER CHART

A.M.A.T.	→	ACUTE MESENTERIC ARTERY THROMBOSIS
AC CAL CHOLE	→	ACUTE CALCULUS CHOLECYSTITIS
AC. APP.	→	ACUTE APPENDICITIS
APP. PERF.	→	APPENDICULAR PERFORATION
APPE	→	APPENDICECTOMY
D.B.L.	→	DILATED BOWEL LOOP
D.O.A.	→	DATE OF ADMISSION
D.S.B.L.	→	DILATED SMALL BOWEL LOOP
D.S.C.	→	DILATED SIGMOID COLON
D1 PERF	→	DUODENUM 1ST PART PERFORATION
EX. LAP.	→	EXPLORATORY LAPAROTOMY
EX. LAP. A.	→	EXPLORATORY LAPAROTOMY WITH APPENDICECTOMY
EX. LAP. P.C.	→	EXPLORATORY LAPAROTOMY PRIMARY CLOSURE
EX. LAP. R & A	→	EXPLORATORY LAPAROTOMY RESECTION AND ANASTAMOSIS
G.G.A.	→	GROUND GLASS APPEARANCE
GOP.	→	GRAHAM'S OMENTAL PATCH
HVP	→	HOLLOW VISCUS PERFORATION
INF.APP	→	INFLAMMED APPENDIX

INT. CHOLE.	→	INTERVAL CHOLECYSTECTOMY
INTS. OBST.	→	INTESTINAL OBSTRUCTION
M.A.F.L.	→	MULTIPLE AIR FLUID LEVEL
MUL. GB ST.	→	MULTIPLE GALLBLADER STONES
PERF	→	PERFORATION
PNEUMO	→	PNEUMOPERITONIUM
S.P.	→	SLUGGISH PERISTALSIS
SL. NO.	→	SERIAL NUMBER