“PAEDIATRIC ACUTE ABDOMEN

A PROSPECTIVE STUDY”

By

Dr.BHARAT.S

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IN

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Under the guidance of

Dr.B.M.NYAMANNAWAR M.Ch

ASSOCIATE PROFESSOR

DEPARTMENT OF SURGERY

B.L.D.E UNIVERSITY'SSHRI B.M.PATIL MEDICAL COLLEGE,

HOSPITAL &RESEARCH CENTRE,

BIJAPUR, KARNATAKA.

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Date:

Place: Bijapur

Dr.BHARAT.S.
Post Graduate
Department of General Surgery,
B.L.D.E.U's Shri B. M. Patil Medical College, Hospital &Research Centre,
Bijapur.

B.L.D.E UNIVERSITY'S

SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL&

RESEARCH CENTRE, BIJAPUR.

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Date:
Place: Bijapur

Dr.B.M.NYAMANNAWAR
Associate Professor,
Department of General Surgery,
B.L.D.E.U's Shri B. M. Patil Medical College, Hospital &Research Centre,
Bijapur.

B.L.D.E UNIVERSITY'S

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Date:
Place: Bijapur

Dr.TEJASWINI VALLABHA
Professor and HOD,
Department of General Surgery,
B.L.D.E.U.'s Shri B. M. Patil Medical College, Hospital &Research Centre,
Bijapur.

B.L.D.E UNIVERSITY'S

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Date:
Place: Bijapur

Dr.M.S.BIRADAR
Principal
B.L.D.E.U.'s Shri B. M. Patil Medical College, Hospital & Research Centre, Bijapur.

B.L.D.E UNIVERSITY'S

SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL &

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Date:
Place: Bijapur

Dr.BHARAT.S.
Post Graduate
Department of General Surgery,
B.L.D.E.U's Shri B. M. Patil Medical College, Hospital &Research Centre,
Bijapur.

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Date: **Dr.BHARAT.S.**

Place: Bijapur.

LIST OF ABBREVIATIONS

Ca^{2+}	Calcium
CBC	Complete Blood Counts
CECT	Contrast Enhanced Computed Tomography
Cl^{-}	Chloride
cm	Centimetre
CRP	C Reactive Protein
DLC	Differential Leucocyte Count
GI	Gastro Intestinal
H^{+}	Hydrogen
K^{+}	Potassium
m^2	Square Meter
mm	Millimetre
MR	Magnetic Resonance
Na^{+}	Sodium
NEC	Necrotizing Entero Colitis
NSAP	Non-Specific Abdominal Pain
n	Number
SMA	Superior Mesenteric Artery
TLC	Total Leucocyte Count
US	Ultrasound
USG	Ultrasonography

ABSTRACT

BACKGROUND AND OBJECTIVES

Abdominal pain is a common complaint in children which necessitates medical or surgical consultation. Children form a special subgroup of population in whom special attention to the subtle symptoms and signs is essential for early diagnosis and management. While medical causes of acute abdomen can be managed conservatively, it is essential to identify children who need immediate surgical intervention to prevent morbidity and mortality. The underlying aetiologies for acute abdomen in children vary with regard to age, nourishment, socioeconomic status and geographical location. In this study we analysed the clinical spectrum of paediatric acute abdomen and identified the common aetiologies in different paediatric age groups in Bijapur.

METHODS

131 children less than 14 years of age who presented with acute abdomen were included in this study. Their clinical profile, investigation, common diagnosis and management were analysed.

RESULTS AND OBSERVATIONS

69% (n=90) of the children required operative management. The most common aetiology necessitating surgery was Uncomplicated Acute Appendicitis accounting for 40% of all cases of acute abdomen with a mean age of 11.3 years. Intestinal obstruction (10 %) was commonly seen in children below 5 years of age which required emergency surgery. Appendicular perforation (n=6) was the commonest cause of generalized peritonitis and accounted for 4.5% of all cases. Urolithiasis accounted for 10% of paediatric acute abdomen requiring surgery.

31 %(n=41) of the children were treated conservatively and the most common diagnosis was Non-Specific Abdominal Pain (71%) followed by Mesenteric Lymphadenitis (24%).

CONCLUSION

This study showed that the aetiologies of paediatric acute abdomen vary depending on the age. Acute abdomen was most common between 9 to 14 years of age and in the male child. Unlike other studies we report a higher incidence of urolithiasis as a cause of surgical acute abdomen in children which probably reflect the role of geographical location and ethnicity on aetiologies of paediatric acute abdomen.

KEY WORDS

Paediatric Acute Abdomen, Acute Appendicitis, Urolithiasis

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INTRODUCTION

Abdominal pain is a common complaint in children which necessitates medical or surgical consultation. There is often a diagnostic dilemma in children with acute pain abdomen; this can be attributed to the wide spectrum of aetiologies and the fact that children have a poor ability to accurately express themselves.[1]

Children often present with excessive crying and irritability, seldom localise the sight of pain and anxious parents tend to exaggerate the degree of severity. In this context a multidisciplinary team including paediatricians, physicians and surgeons are often required to make an accurate timely diagnosis which can expedite management, reduce morbidity and ensure recovery. A good history, thorough clinical examination and appropriate investigations help distinguish between medical and surgical paediatric acute abdomen.

The term *acute abdomen* refers to signs and symptoms of abdominal pain and tenderness, a clinical presentation that often requires emergency surgery.[2] The term acute abdominal pain generally refers to previously undiagnosed pain that arises suddenly and is of less than 7 days (usually less than 48 hours) duration.[3]

It is loosely defined as pain in or over the abdominal cavity experienced for anything between few hours and a few weeks. The time interval is necessarily blurred but majority of patients present between six and eight hours after developing pain.

Zachary Cope in 1921 wrote that 'The majority of severe abdominal pain which ensues in patients who have previously been fairly well and which last long as six hours are caused by conditions of surgical importance.[4]

The diagnoses associated with an acute abdomen vary according to age and gender. The aetiologies of acute abdomen may also vary depending on the geographical location, level of urbanization and socioeconomic status of the children.

There are no studies till date detailing the various aetiologies and modes of presentation of paediatric acute abdomen in a semi urban location such as Bijapur. This study aims to analyse the clinical spectrum of paediatric acute abdomen and to identify the common aetiologies in different paediatric age groups in Bijapur.

AIMS AND OBJECTIVES

The study was undertaken with the following objectives.

1. To identify the common non-traumatic surgical causes of paediatric acute abdomen.
2. To analyse the clinical spectrum of paediatric acute abdomen.

REVIEW OF LITERATURE

Pain abdomen is a common chief complaint in children with approximately 10% of all children attending the emergency room having a complaint referable to the abdomen.[5] A large proportion of children assessed in the hospital with abdominal pain may leave with no definitive diagnosis or a diagnosis of Non Specific Abdominal Pain.[6] The challenge is to identify and treat the majority of children with self-limiting but benign conditions conservatively, while the child with an uncommon but potentially life-threatening cause of pain may need emergency surgery.

Many diseases, some of which are not surgical or even intra-abdominal, can produce acute abdominal pain and tenderness.[7] The majority of surgical diseases associated with an acute abdomen result from infection, inflammation, obstruction, ischemia, or perforation. The timing of intervention is often the single most important factor in determining the outcome.

In children different conditions of the gastrointestinal tract present with similar clinical features and accurate diagnosis by clinical methods is often a very challenging task. Repeated examination at hourly intervals usually solves the problem. Children should be subjected to minimal investigations and non-invasive investigations are better tolerated by the child with severe pain abdomen.

Acute abdomen can be defined as severe, persistent abdominal pain of sudden onset that is likely to require surgical intervention to treat its cause.

Pathophysiology

Clinically, abdominal pain falls into 3 categories:

1. Visceral (splanchnic pain)
2. Parietal (somatic pain)
3. Referred pain

Visceral pain occurs when noxious stimuli affect a visceral organ, such as the stomach or intestines. Tension, stretching and ischemia stimulate visceral pain fibres. Tissue congestion and inflammation tend to sensitize nerve endings and lower the threshold for stimuli. Visceral pain fibres are bilateral and unmyelinated and enter the spinal cord at multiple levels, thus visceral pain is usually dull, poorly localised and felt in the midline. Pain from foregut structures cause periumbilical pain, and hind gut structures cause lower abdominal pain.

Parietal pain arises from noxious stimulation of the parietal peritoneum. Pain resulting from ischemia, inflammation, or stretching of the parietal peritoneum is transmitted through myelinated afferent fibres to specific dorsal root ganglia on the same side and at the same dermatome as the origin of the pain. Parietal pain usually is sharp, intense, discrete and localized and coughing or movement can aggravate it.

Referred pain has many of the characteristics of parietal pain but is felt in remote areas supplied by the same dermatome as the diseased organ. It results from shared central pathways for afferent neurons from different sites. A classic example is a patient with pneumonia or empyema who presents with abdominal pain because the T9 dermatome distribution is shared by the lung and the abdomen.[5]

Table 1 below lists a few common organs which when inflamed or injured cause referred pain at specific locations.

Table 1 : Locations of Referred pain

Right Shoulder

- Liver
- Gallbladder
- Right hemi diaphragm

Left Shoulder

- Heart
- Tail of pancreas
- Spleen
- Left hemi diaphragm

Scrotum and Testicles

- Ureter

ANATOMY

The Abdominal cavity:

The Abdominal cavity is an extensive space which extends upwards, deep to the costal margins into the concavity of the diaphragm and projects downwards and backwards into the bony pelvis as the pelvic cavity. A considerable part of the abdominal cavity is overlapped by the thoracic cage above and the bony pelvis below.

Boundaries

Superiorly	The diaphragm extending to the 5th intercostal space
Posteriorly	The lumbar vertebrae, quadratus lumborum and transverse abdominis muscles
Anterolaterally	The muscles of abdominal wall (transverse abdominis, internal and external oblique).
Inferiorly	Pelvic brim

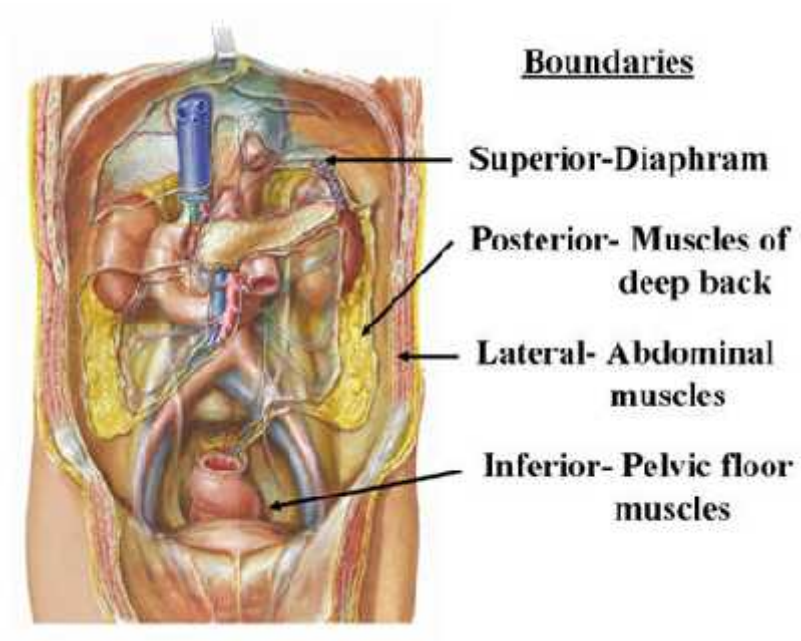


Fig 1: Boundaries of the Abdominal Cavity

Regions of abdomen:

The abdomen is divided into nine regions by two vertical and two horizontal lines. The vertical lines are drawn from midclavicular point to the mid inguinal point on the same side. Horizontal lines are the transpyloric line a horizontal line passing through midpoint between xiphisternum and umbilicus, and the transtubercular line a horizontal line passing through the tubercles of the iliac crest.

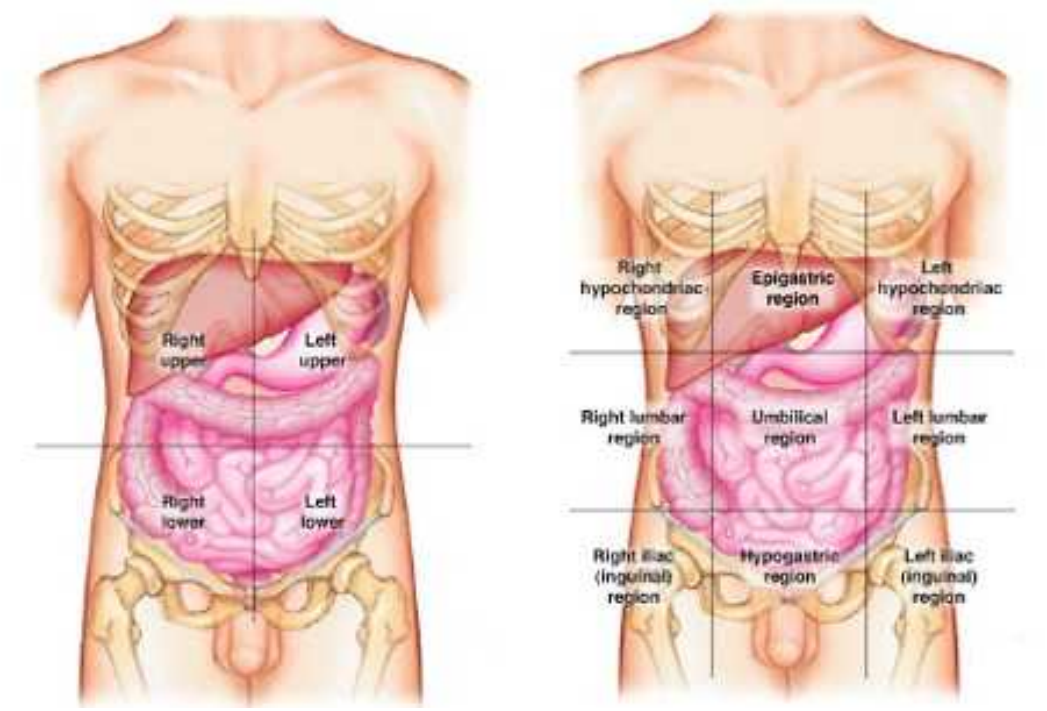


Fig 2: Quadrants and Regions of the Abdomen

The Abdominal Wall [10]

The abdominal wall is an anatomically complex, layered structure with segmentally derived blood supply and innervation. The layers of the abdominal wall include the following:

Skin

The dermis contains collagen fibres that tend to be horizontal, forming the creases of the skin. These are called Langer's lines or Kraissl's lines. Abdominal incisions taken along these natural skin creases tend to heal more cosmetically.

Superficial fascia

It is formed by the connective tissue that is not aponeurosis, tendon or ligament. There are two layers. *Camper's fascia* or the fatty layer, first of the two layers which is found throughout the abdominal wall. Second the *Scarpa's fascia* or the Membranous layer, found in the lower third of the anterior abdominal wall. It has a restrictive location.

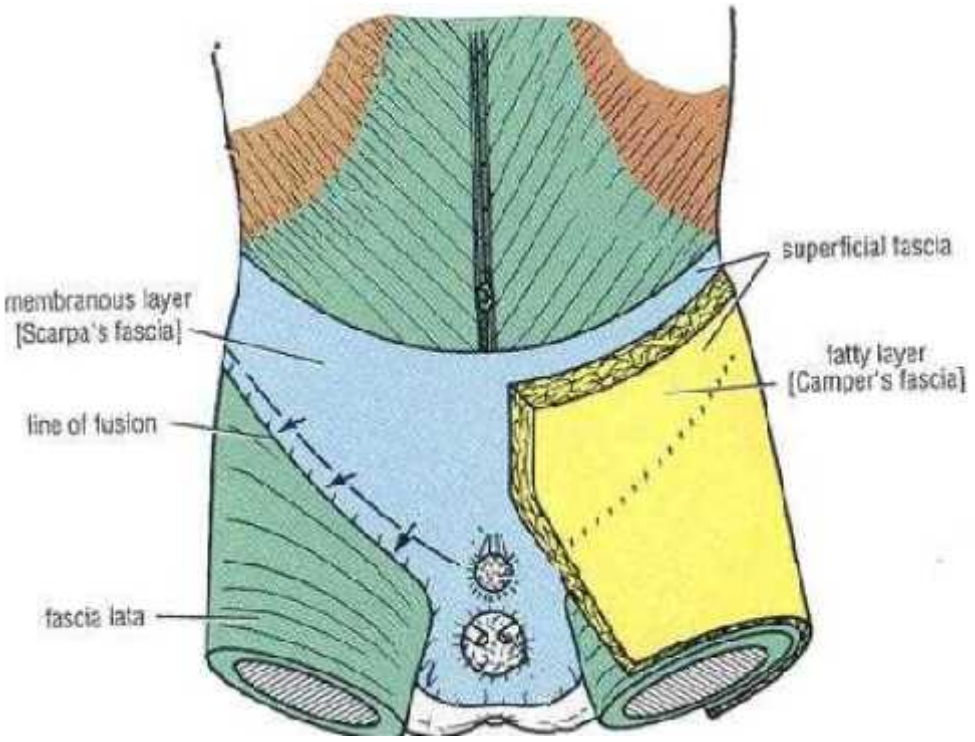


Fig 3: Superficial Fascia of Abdominal Wall

Table 2 The Anterior Abdominal Wall Muscles and Their Aponeurosis

Sl.No	Muscle	Origin	Insertion	Nerve Supply	Action
1.	External Oblique	Lower 8 ribs	Xiphoid process, linea alba, pubic crest, pubic tubercle, iliac crest	Lower 6 thoracic nerves and iliohypogastric and ilioinguinal nerves(l1)	Supports abdominal contents, assists in flexing and rotation of trunk, raises intrabdominal pressure
2.	Internal oblique	Lumbar fascia, iliac crest, lateral two thirds of inguinal ligament	Lower three ribs and costal cartilages, xiphoid process, linea alba, symphysis pubis	Lower 6 thoracic nerves and iliohypogastric and ilioinguinal nerves(l1)	Supports abdominal contents, assists in flexing and rotation of trunk, raises intrabdominal pressure
3.	Transversus abdominis	Lower 6 costal cartilages, lumbar fascia, iliac crest, and lateral third of inguinal ligament.	Xiphoid process linea alba, symphysis pubis	Lower 6 thoracic nerves and iliohypogastric and ilioinguinal nerves(l1)	Compresses abdominal contents
4.	Rectus abdominis	Symphysis pubis And pubic crest	5th and 6th costal cartilages and xiphoid process	Lower 6 thoracic nerves	Compresses abdominal contents and flexes vertebral column, accessory muscles of expiration
5.	Pyramidalis (if present)	Anterior surface of pubis	Linea alba	12th thoracic nerve	Tenses the Linea alba

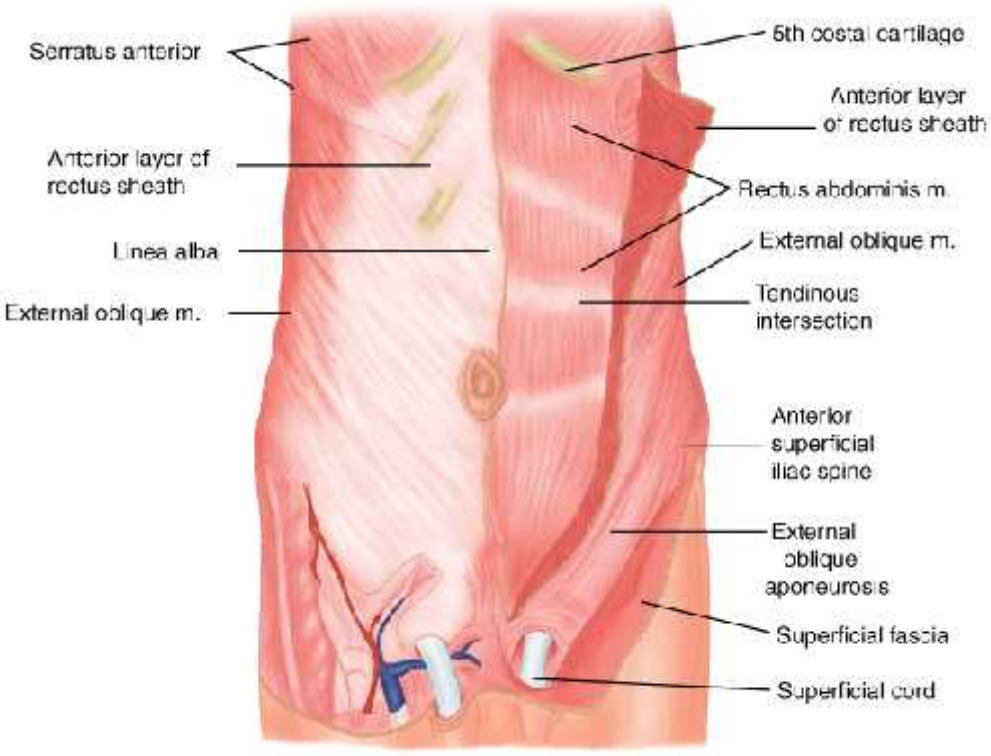


Fig 4: Muscles of the Abdominal Wall [8]

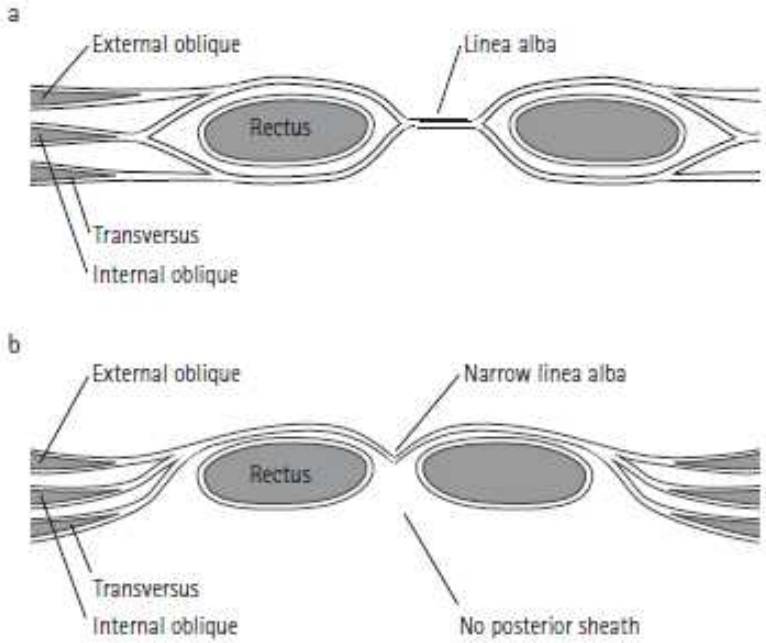


Fig 5: Formation of Rectus Sheath (a) Above the Arcuate line (b) Below the Arcuate line [9]

Arterial Supply and Venous Drainage of the Anterior Abdominal Wall

The superficial tissues of the lower anterolateral abdominal wall are supplied by three branches of the femoral artery. These branches from lateral to medial are the superficial circumflex iliac artery, the superficial epigastric artery and the superficial external pudendal artery. Branches of these arteries travel toward the umbilicus in the subcutaneous connective tissues. All three arteries have anastomosis with the deep arteries.

The deep arteries lie between the Transversus abdominis and the internal oblique muscle. These are the posterior intercostal arteries, the anterior branch of the subcostal artery, the anterior branches of the four lumbar arteries and the deep circumflex iliac artery.

The rectus sheath is supplied by two arteries. The superior epigastric artery arises from the internal thoracic artery. The inferior epigastric artery arises from the external iliac artery, just above the inguinal ligament.

To avoid injury to the major vessels in abdominal operative laparoscopic procedures, laterally situated trocars should be placed at least 8cm from the midline and at least 5cm above the pubic bone.

The venous drainage is through the veins that accompany the arteries.

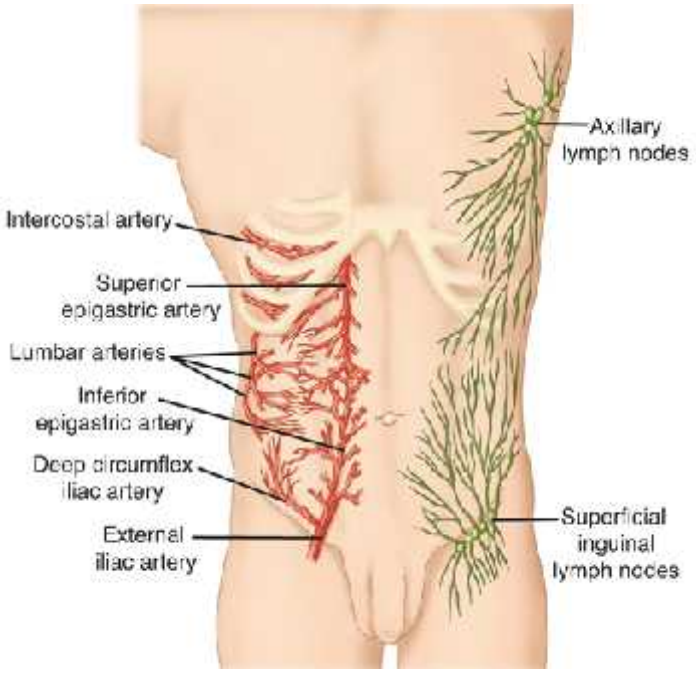


Fig 6: Arteries of the Abdominal Wall [8]

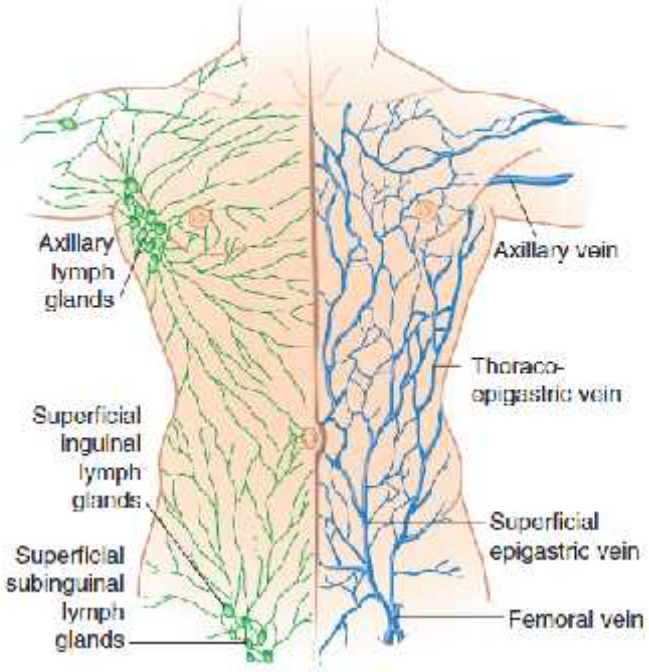


Fig 7: Veins of the Abdominal Wall [2]

Nerve supply of Anterior Abdominal Wall

Anterior abdominal wall is supplied by the ventral rami of T7-T12 and L1. The Iliohypogastric nerve directly superior to the ilioinguinal nerve innervates the suprapubic area. The Ilioinguinal nerve traverses the inguinal canal, with the spermatic cord (male) or round ligament (female) to supply the scrotum (or labia majora) and the medical aspect of thigh. Both iliohypogastric and ilioinguinal nerve may come off as a single nerve and branch later.

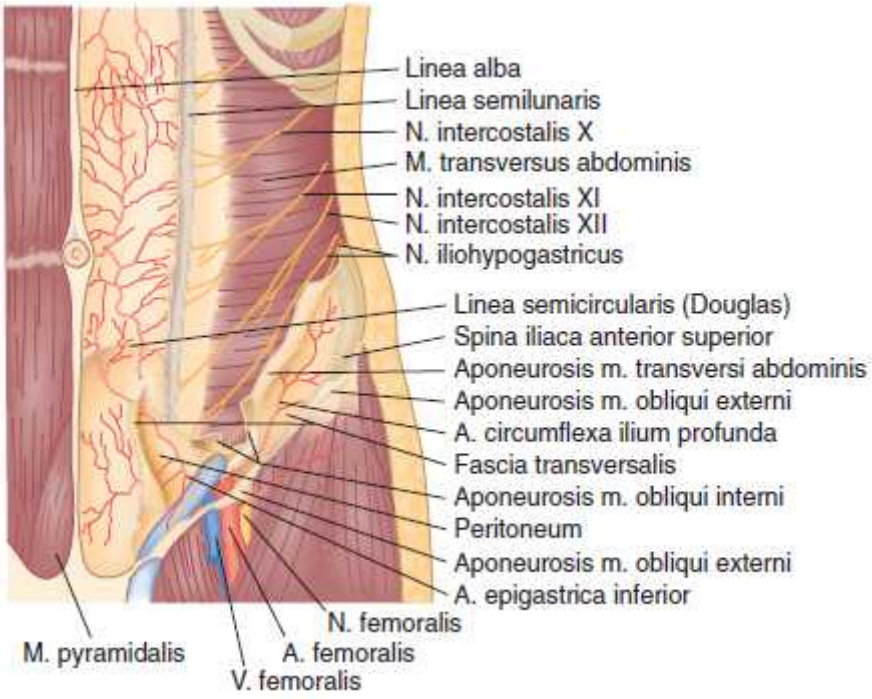


Fig 8: Nerves of the Abdominal Wall [2]

Peritoneum and Peritoneal cavity[2]

The peritoneum consists of a single sheet of simple squamous epithelium of mesodermal origin, termed *mesothelium,* lying on a thin connective tissue stroma. The surface area is 1.0 to 1.7 m^2 approximately that of the total body surface area. In males, the peritoneal cavity is sealed, whereas in females it is open to the exterior through the ostia of the fallopian tubes. The peritoneal membrane is divided into parietal and visceral components. [2]

The parietal peritoneum covers the anterior, lateral, and posterior abdominal wall surfaces and the inferior surface of the diaphragm and the pelvis. It is pain sensitive because of somatic innervations and pain is well localized. It is loosely attached to the walls by extra peritoneal connective tissue and can therefore be easily stripped.[11]

The visceral peritoneum covers most of the surface of the intraperitoneal organs (i.e., stomach, jejunum, ileum, transverse colon, liver, spleen) and the anterior aspect of the retroperitoneal organs (i.e., duodenum, left and right colon, pancreas, kidneys, adrenal glands).It is firmly adherent to the underlying organ and cannot be stripped. Blood and nerve supply are same as those of underlying viscera, thus due to its autonomic innervation it is relatively pain insensitive and produces vague dull aching pain when inflamed.[11]

The peritoneal cavity is a potential space and contains only a few millilitres of fluid. The peritoneal cavity consists of a main region termed the Greater sac and the other lesser sac or omental Bursa. It is divided into pelvic and abdominal portions. The abdominal portion is divided into supracolic and infracolic compartment by transverse colon and mesocolon. The infracolic compartment is divided into right and left by mesentery.

The Right infracolic and left infracolic is divided into external and internal paracolic gutters by ascending and descending colon respectively. Supracolic compartment is below the diaphragm and above transverse colon and mesocolon. The liver, gallbladder, stomach, first part of the duodenum and spleen lie in this space. [12]

There are 11 ligaments and mesenteries which divide the peritoneal cavity into the above mentioned compartments. These include the coronary, gastrohepatic, hepatoduodenal, falciform, gastrocolic, duodenocolic, gastrosplenic, splenorenal, and phrenicocolic ligaments and the transverse mesocolon and small bowel mesentery.

These structures partition the abdomen into nine potential spaces—right and left subphrenic, subhepatic, supramesenteric and inframesenteric, right and left paracolic gutters, pelvis, and lesser space. These ligaments, mesenteries, and peritoneal spaces direct the circulation of fluid in the peritoneal cavity and thus may be useful in predicting the route of spread of infectious and malignant diseases.

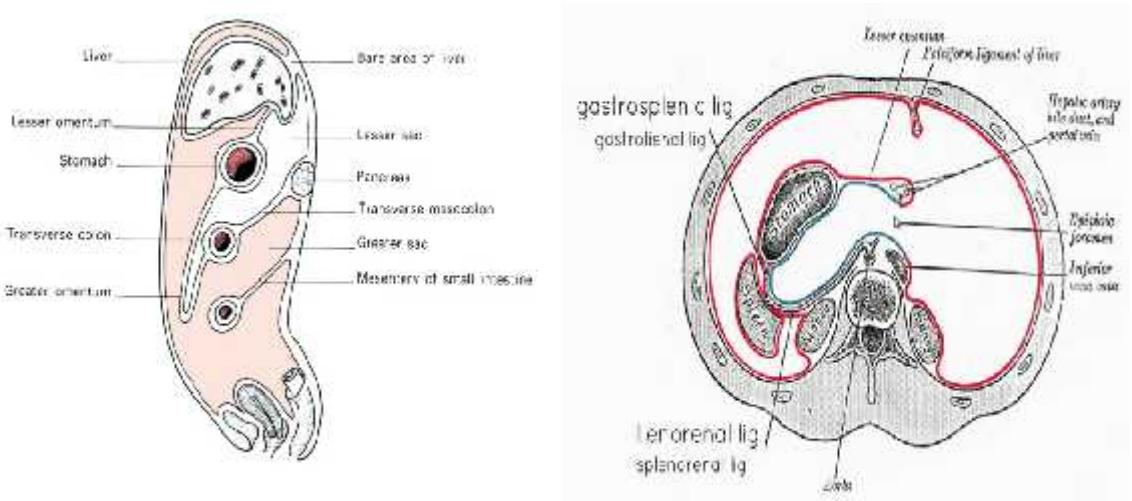


Fig 9: Peritoneal Reflections and Compartments [13]

Development and Anatomy of Intestine [14]

During embryonic period, the embryonic disc folds on itself and yolk sac becomes enclosed within the embryo and a cord lined by endoderm is formed, which leads to formation of future gut. The part, proximal to the communication forms foregut. The part caudal to the communication forms hindgut. And intervening part forms midgut.

The yolk sac communication is progressively narrowed and now termed as definitive yolk sac, the narrowed connecting channel is called vitello-intestinal duct (vitelline duct) which usually disappears.

As the head and tail folds are forming, similar folds on the lateral side are formed, these are called lateral folds which enclose the embryo all around by ectoderm except at the region of vitello- intestinal duct passage, which leads to a circular aperture called umbilical opening.

While gut is being formed, a midline artery the dorsal aorta is formed just dorsal to the gut which gives series of branches to the gut. Those running in the midgut run right up to the yolk sac, which is called vitelline artery. Only three of these arteries persist, one for each portion of the gut.

- Foregut - Coeliac artery
- Mid gut - Superior mesenteric artery
- Hind gut - Inferior mesenteric artery

As the narrowing of the communication between the primitive gut and yolk sac occurs, the midgut forms loop with mesentery through which the midgut artery runs. The part proximal to the artery is called pre arterial and caudal to it is called post arterial segment. A bud arises from the post arterial segment near the apex of the loop, which is known as caecal bud, which appears at 5th week. Rotation of the gut begins round about this period.

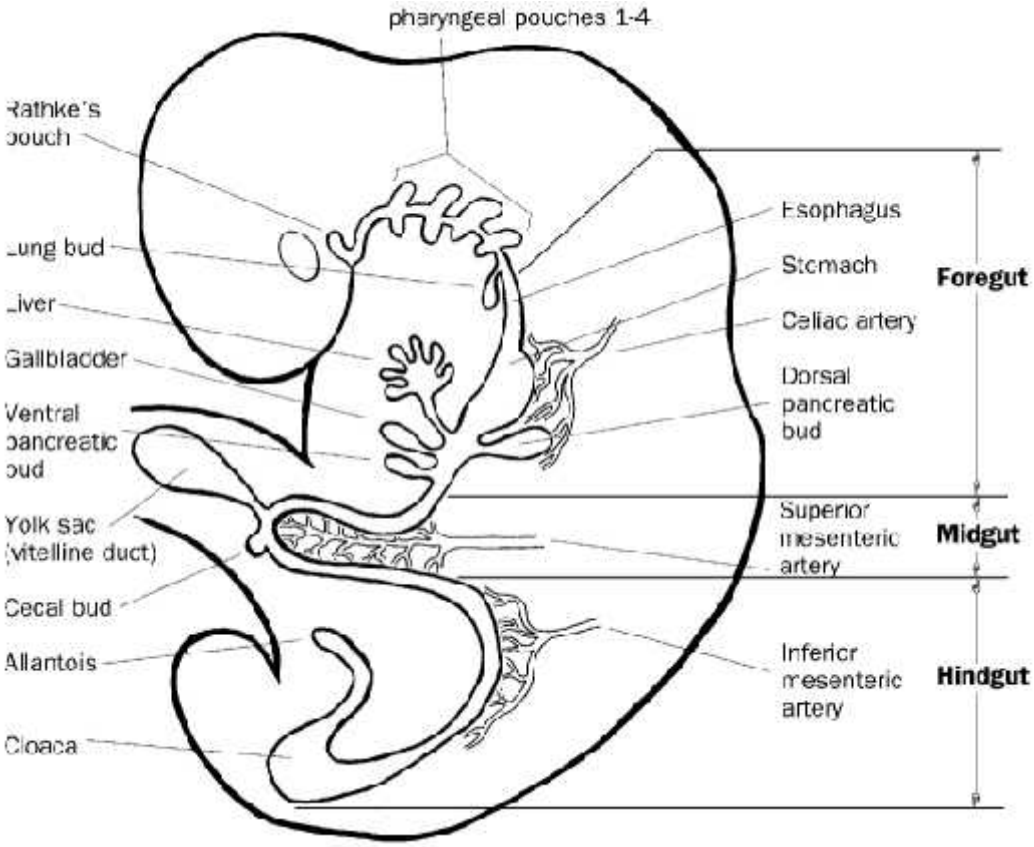


Fig 10: Embryology of Gut

First stage of rotation (5 to 10 weeks of intra uterine life)

This stage of rotation is due to rapid development of the liver cleft. The first, stage of rotation is complete when the mid gut loop has rotated though 900 in an anti clock wise direction.

Second stage of rotation (10th week to 11th week)

By the end of 10th week, Mid gut loop returns to the abdominal cavity from the umbilical hernia (physiological).The returning of hernia into the coelomic cavity takes place in an order, the pre arterial segment returns first progressively followed by the remaining part and caecum being the last. As the bowel reduces into the coelomic cavity the small bowel (pre arterial segment) enters the abdomen to the right of artery and descending colon comes to the left flank. As the caecum and right half of the colon reduces passes upwards and to the right completing another 90^0 rotation. Subsequent growth and elongation of the colon pushes the caecum to the right loin so that the transverse colon coming in front of the artery, thus making the mid gut loop rotation 270^0 and completes the second stage.

Third stage of rotation (11th week and shortly after birth)

During this stage the caecum descends further reaching the right iliac fossa. The mesentery of small intestine becomes adherent to posterior abdominal wall. The post arterial mesentery of the transverse colon persists as transverse mesocolon. The mesentery of caecum, ascending colon, hepatic flexure and descending colon completely obliterated by fusion with posterior abdominal wall except the mesentery of the pelvic colon, which persists as pelvic mesocolon. So in third stage of rotation fixation of the gut to the posterior abdominal wall takes place.

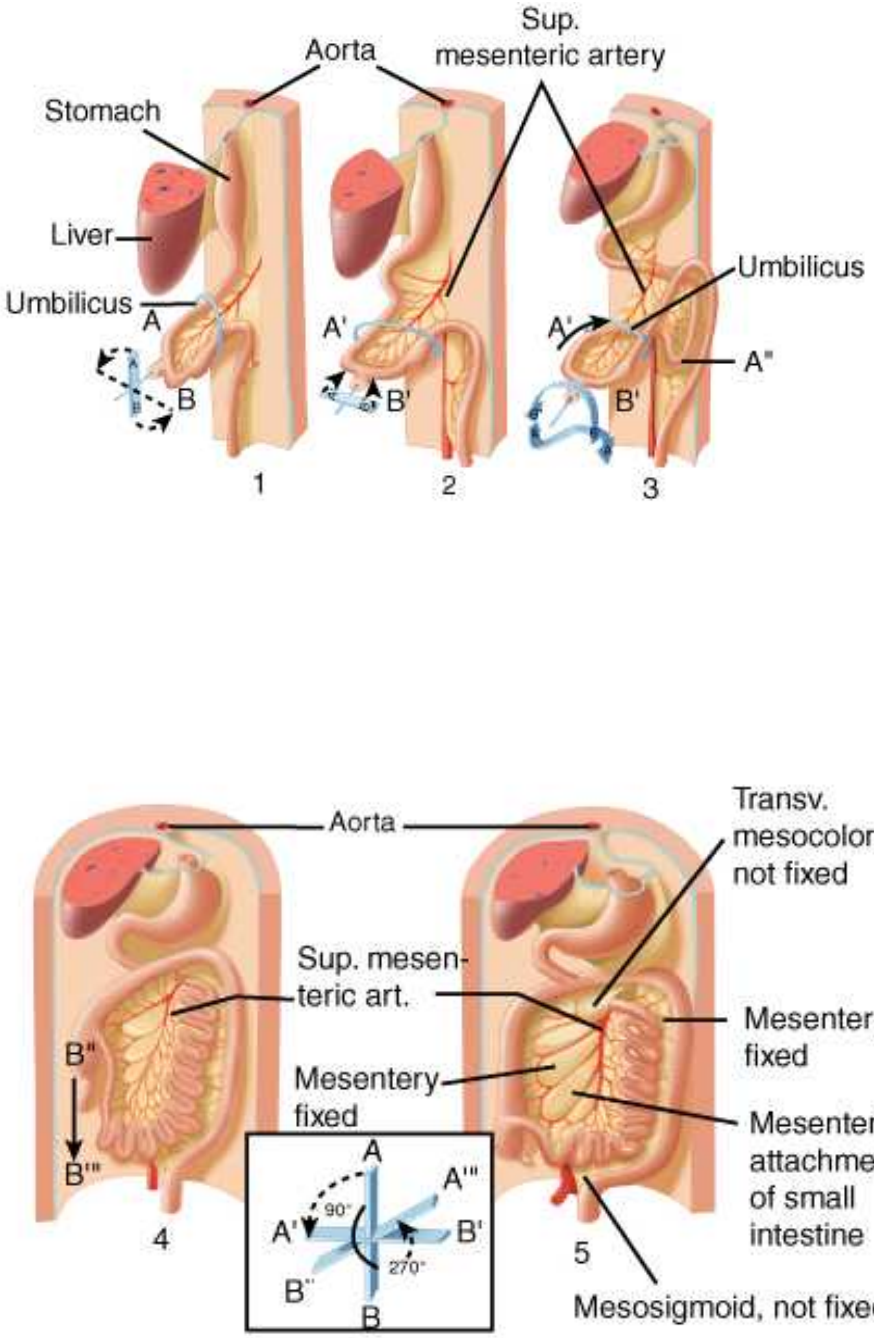


Fig 11: Rotation of Gut [15]

Anatomic relationships of intestinal mesentery to the retroperitoneum after completion of intestinal rotation during fetal development.

The Stomach

The stomach is a capacious saccular organ – the most dilated part of the G.I. tract connected superiorly with the inferior end of the oesophagus and inferiorly with the first portion of the duodenum.

The Duodenum

The duodenum is 25 cm long and is mostly retroperitoneal and fixed. It forms the shape of C with its concavity to the left; it is described in four parts. The first part is about 5 cm in length and runs upwards, backwards and to the right from the pylorus. It is mobile in the first half and becomes retroperitoneal. The second part is about 8 cm in length and extends downwards and gives an opening to the ampulla of Vater through which the pancreatic and bile juices enter intestine. The third is the horizontal portion and the fourth is the ascending portion and continues as the jejunum.

The Jejunum

Jejunum means empty - The name is derived from its empty state after death. It includes $2/5^{th}$ length of the small intestine, after duodenum. It commences from the duodenum on the left side of the second lumbar vertebra and terminates in the ileum.

The Ileum

Ileum means to twist; it derives its name from its numerous coils or convolutions in the remaining $3/5^{th}$ of the small intestine, occupying chiefly umbilical, hypogastric and right iliac regions and terminates into caecum.

The Mesentery

Mesentery of the small bowel is broad fan shaped fold of peritoneum, which suspends the coils of jejunum and ileum from the posterior abdominal wall.

Large Intestine

Extends from the ileo caecal junction to anus, 1.5 meters, long and divided into caecum, ascending colon, transverse colon, descending colon, sigmoid (pelvic) colon and rectum.

Gall bladder

The gallbladder stores, and concentrates the bile secreted by the liver. It is globular or pear-shaped viscous with a capacity of about 60 millilitres, and consists of three parts-fundus, body and neck. It lies in the gallbladder fossa on the visceral surface of the right lobe of the liver, adjacent to the quadrate lobe.

Biliary Tract

The extrahepatic biliary tract consists of the three hepatic ducts (right, left and common), the gallbladder and cystic duct, and the bile duct. The right and left hepatic ducts, exit from the liver and join to form the common hepatic duct, near the right end of the porta hepatis. The common hepatic duct is soon joined on its right side at an acute angle by the cystic duct from the gallbladder, to form the bile duct. When the liver retracted at operation the ducts are seen to descend below the liver, but at rest they lie in loose contact with porta hepatis.

Pancreas

The pancreas is a composite gland having exocrine acini which discharge the secretions into the duodenum to assist digestion, and groups of endocrine cells, the islets of Langerhans, whose role is in carbohydrate metabolism. Its length is about 15cm. The gland lies somewhat obliquely, sloping from the head upwards towards the tail behind the peritoneum of the posterior abdominal wall.

The head, the broadest part of the pancreas, is moulded to the C-shaped concavity of the duodenum, at the level of L.2 vertebra. The main blood supply is from the splenic artery, which supplies the neck, body and tail. One large branch is named the arteria pancreatica magna. The head is supplied by the superior and the inferior pancreaticoduodenal arteries. Venous return is by numerous small veins into the splenic vein and, in the case of the head, by the superior pancreaticoduodenal vein into the portal vein and by the inferior pancreaticoduodenal vein into the superior mesenteric vein.

Paediatric Acute Abdomen

There are many disorders which give rise to acute abdominal pain in children.

Table 3 : Common causes of acute abdominal pain in children

1. **Gastrointestinal causes**
   - Gastroenteritis
   - Appendicitis
   - Nonspecific abdominal pain
   - Mesenteric lymphadenitis
   - Constipation
   - Abdominal trauma
   - Intestinal obstruction
   - Peritonitis
   - Food poisoning
   - Peptic ulcer
   - Meckel's diverticulitis
   - IBD
   - Lactose intolerance
2. **Liver, spleen and biliary tract disorders**
   - Hepatitis
   - Cholecystitis
3. **Genitourinary causes**
   - UTI
   - Hematocolpos
   - Urinary calculi
   - Dysmenorrhoea
4. **Metabolic disorders**
   - Diabetic ketoacidosis
   - Hypoglycaemia
   - Porphyria
5. **Haematologic disorders**
   - Sickle cell anaemia
   - Henoch – Schonlein purpura
   - Haemolytic ureamic syndrome
6. **Pulmonary causes**
   - Pneumonia
   - Diaphragmatic pleurisy
7. **Miscellaneous**
   - Infantile colic
   - Functional pain
   - Angioneurotic Oedema

With thorough physical examination and relevant history it is essential to differentiate the potentially life threatening surgical emergencies. Table 4 lists the differences in the presentation of some of the commonest surgical acute abdomens.

Table 4: Distinguishing Features of Acute Gastrointestinal Tract Pain in Children[17]

DISEASE	ONSET	LOCATION	REFERRAL	QUALITY	COMMENTS
Pancreatitis	Acute	Epigastric, left upper quadrant	Back	Constant, sharp, boring	Nausea, emesis, tenderness
Intestinal obstruction	Acute or gradual	Periumbilical–lower abdomen	Back	Alternating cramping (colic) and painless periods	Distention, obstipation, emesis, increased bowel sounds
Appendicitis	Acute	Periumbilical, then localized to lower right quadrant; generalized with peritonitis	Back or pelvis if retrocecal	Sharp, steady	Anorexia, nausea, emesis, local tenderness, fever with peritonitis
Intussusception	Acute	Periumbilical–lower abdomen	None	Cramping, with painless periods	Hematochezia, knees in pulled-up position
Urolithiasis	Acute, sudden	Back (unilateral)	Groin	Sharp, intermittent, cramping	Hematuria
Urinary tract infection	Acute, sudden	Back	Bladder	Dull to sharp	Fever, costochondral tenderness, dysuria, urinary frequency

APPENDICITIS

Appendicitis most commonly affects children age 10–19, with an overall incidence of approximately 20 cases per 10,000 population annually.[18] Among those under age 20, infants age 0–4 have the lowest incidence of appendicitis (2 cases per 10,000 annually), but up to two-thirds will present with perforation.[19]

Perforation is common because infants often present later in their disease course and because of the difficulty in obtaining an accurate history. The diagnosis is further complicated by diseases of childhood that can mimic appendicitis. Mesenteric adenitis, an inflammation of the mesenteric lymph nodes secondary to upper respiratory tract infection, can present with fever and right lower quadrant pain. Streptococcal pharyngitis and bacterial meningitis can also present with fever, nausea, and abdominal pain. These diagnoses should be considered when evaluating children for suspected appendicitis.[20]

In children with an equivocal history and physical examination, CT has been shown to be highly accurate in diagnosing appendicitis. Garcia Pena and associates compared ultrasonography and rectal contrast CT in 139 children with suspected appendicitis and found CT to be more sensitive (97% for CT, 44% for ultrasound) and more specific (94% for CT, 93% for ultrasound).[21]

Gross Anatomy of the Appendix

In addition to the variable location of the tip of the appendix, its relationship to surrounding structures is protean. The appendix may lie across the psoas muscle or over the pelvic brim, resting on the pelvic fascia that over lies the obturator internus muscle. These positions account for the physical findings of pain on extension of the hip (psoas sign) or pain with flexion and internal rotation of the thigh (obturator sign).

Additional variations in appendiceal position result from the abnormalities of midgut rotation and because the attachment of the appendix to the base of the cecum is a constant, anomalies such as malrotation can lead to a left-sided appendix. The size and shape of the appendix also vary. It may be funnel shaped or cylindrical with a uniform calibre. The length can range, from 0.3 to 33 cm, with appendices of males tending to be slightly longer than those of females. The diameter is typically less than or equal to 6 mm, and thus a measurement larger than that is one of the ultrasound criteria for diagnosis of appendicitis.[22]

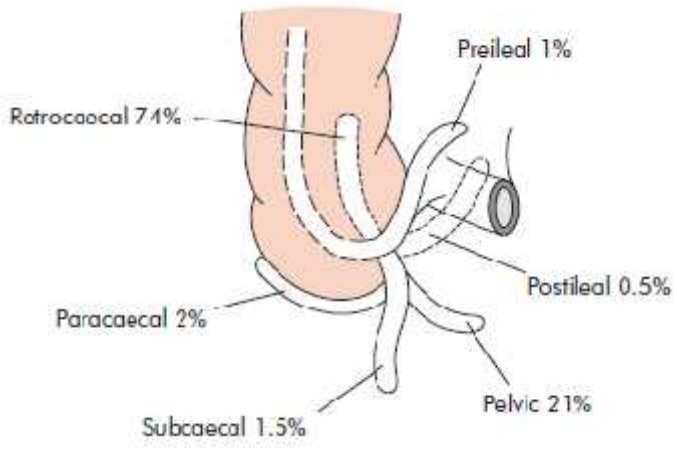


Fig 12: Positions of the tip of Appendix [23]

The arterial supply to the appendix is from the ileocolic artery, a branch of the superior mesenteric artery. One of four terminal branches of the ileocolic artery, the appendiceal artery passes posterior to the terminal ileum and gives off multiple short, straight branches to the appendix. The retroileal course of the appendiceal artery may predispose it to kinking with resultant ischemia and inflammation of the appendix.

The venous drainage of the appendix is via the superior mesenteric vein to the portal vein, which accounts for the occasional findings of pyelophlebitis or liver abscess following appendicitis.[13]

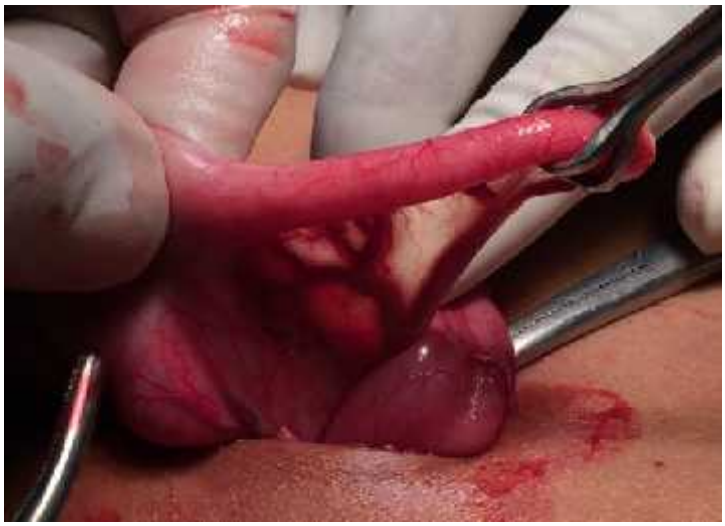


Fig 13: Intraoperative Photograph showing mesoappendix and appendicular vessels

The appendix is innervated by branches of the splanchnic nerves that arise from the lower thoracic ganglia. Typically, the T10 ganglion is the one via which painful stimuli from the appendix are conducted to the dorsal nerve root, along the spinothalamic tract to the brain. Because the umbilical region of the abdominal wall develops from the same dermatome as the appendix, appendicular pain initially localizes to the periumbilical region.[13]

The appendix has four layers. In both the mucosa and submucosa, germinal follicles and lymphoid pulp are prominent in infants and children. The lymphoid tissue gradually atrophies with age. Whether this lymphoid tissue predisposes children to appendicitis by luminal obstruction during periods of inflammation is unproved.

Pathophysiology of Appendicitis

The pathologic sequences ending in appendicitis is the obstruction of the proximal lumen. Fecoliths are the most frequent example of obstructing appendicular lesions and are present in approximately 30%-50% of appendicitis patients.[24]

Other obstructing lesions may include lymphoid hyperplasia, foreign bodies, parasite infestation or conditions that cause increased colonic pressure and decreased mobility such as Hirschsprung's disease or meconium ileus.[25]

Enteric bacteria are the most common organisms associated with appendicitis. In the patients with perforation, Escherichia coli, Enterococcus, Bacteroides and Pseudomonas are the species most frequently isolated from the above. Parasitic infestations with Enterobius or Ascaris have also been reported in association with appendicitis.

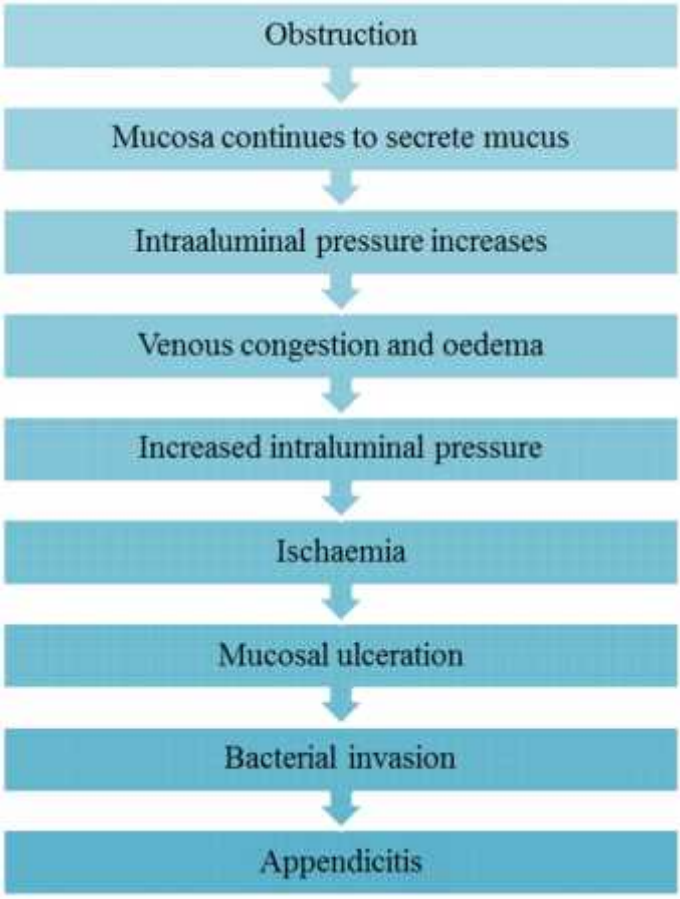


Fig 14: Pathogenesis of Appendicitis

Children with appendicitis classically present with visceral, vague, poorly localized periumbilical pain, within 6-48 hours. The pain becomes parietal as the overlying peritoneum becomes inflamed; the pain then becomes well localized and constant in right iliac fossa.

The incidence of perforation in infants younger than 1year of age is almost 100%, and although it decreases with age, it is still 50% at 5 years of age, the mortality rate in this age group remains as high as 5%. [25]

In prospective observational study conducted by Shakya KN et al, they concluded that appendicitis is the most common surgical condition in children who present with abdominal pain.[26] Pujari AA et al, in a retrospective study on acute gastrointestinal emergencies requiring surgery concluded that the most common surgical acute abdomen in children was acute appendicitis followed by intestinal obstruction.[27]

MESENTERIC LYMPHADENITIS

Mesenteric lymphadenitis is an inflammatory condition of the mesenteric lymph nodes that can present with acute or chronic abdominal pain. The nodes are usually in the right lower quadrant and hence mesenteric lymphadenitis sometimes mimics appendicitis. [28]

Aetiology of mesenteric lymphadenitis includes viral and bacterial gastroenteritis such as Yersinia enterocolitica, Group A streptococcal pharyngitis, inflammatory bowel disease and lymphoma. Viral infection is the most common cause. In 1926, Wilensky and Hahn classified mesenteric lymphadenitis into four groups:

I. Simple mesenteric lymphadenitis
II. Suppurative mesenteric lymphadenitis
III. Tuberculous mesenteric lymphadenitis
IV. Terminal stage of mesenteric lymphadenitis (calcification). [28]

Mesenteric lymphadenitis is diagnosed by an ultrasound that shows abdominal lymph nodes greater than 10mm. The presence of enlarged lymph nodes on diagnostic imaging does not by itself exclude a diagnosis of appendicitis; it is necessary to demonstrate a normal appendix as well.[29]

A study done by Vignault F et al on 70 children with clinically suspected acute appendicitis, 16% had a final diagnosis of mesenteric lymphadenitis established by ultrasound, clinical course or surgery.

NON SPECIFIC ABDOMINAL PAIN (NSAP)[30-39]

Non Specific Abdominal pain is a common complaint in children worldwide which leads to substantial school absenteeism. It is childhood abdominal pain which is not associated with organic disease. Little is known about childhood NSAP, in family practice, in population and school based studies, the prevalence of NSAP ranges from 4% to 10%. The impression is that family physicians consider NSAP in children to be a benign disorder needing little more than reassurance of parent and child.

The above view, however, contradicts the presence of psychological comorbidity and other nonspecific somatic symptoms, and the high prevalence of prolonged symptoms found in children referred to secondary and tertiary care. In specialist care, childhood NSAP is considered to be a complex and time consuming problem, and parents of children with NSAP are found to be hard to reassure.

A study was done by Holland A and Gollow IJ at Princess Margaret Hospital for children, Perth over a 3 year period on 1313 children admitted with acute abdominal pain. 54% were discharged without surgical intervention out of which 70% of them were diagnosed to have NSAP. [40]

INTESTINAL OBSTRUCTION

Intestinal obstruction is defined as the failure of the motor functioning of the intestinal tract. It is probably the most common reason for emergency surgery in infants and young children.[41] Depending on the degree of obstruction to the flow of intestinal contents, Obstruction may be partial or complete and be further characterized as simple or strangulated, depending on the integrity of the blood supply to the involved bowel.

Aetiology

Intestinal obstruction may either be due to an actual physical barrier known as mechanical or dynamic obstruction, or due to a functional failure of unidirectional purposeful progressive intestinal transit called paralytic ileus or adynamic obstruction.[42] The causes of Intestinal Obstruction in children are listed below.[43]

A. Mechanical obstruction of the lumen / Dynamic obstruction

I. Obstruction of the lumen (Intraluminal)

- Intussusception
- Impactions- bezoar, roundworms, foreign body, faeces, polyp, tumour.

II. Intrinsic lesions of the bowel (Intramural)

- Congenital- atresia/stenosis, duplication, Meckel's diverticulum
- Inflammatory - tubercular, regional enteritis, NEC
- Traumatic, e.g. duodenal hematoma
- Neoplastic - tumour, malignant stricture
- Iatrogenic- anastomotic stricture, radiation stricture.

III. Extrinsic lesions of the bowel

- Adhesions (postoperative, post infectious)
- Bands - congenital, inflammatory
- Hernia- inguinal, femoral, umbilical, incisional and internal hernia
- Extrinsic mass - abscess, hematoma, annular pancreas, anomalous vessel
- Volvulus - gastric, midgut, colonic.

B. Inadequate propulsive activity / Adynamic obstruction

I. Neuromuscular defect - megacolon, paralytic ileus

II. Vascular occlusion - arterial, venous.

PATHOPHYSIOLOGY OF INTESTINAL OBSTRUCTION [44-48]

Simple Mechanical Intestinal Obstruction

In simple mechanical obstruction, the blood supply of the gut is intact. An obstructed gut dilates proximal to the obstruction due to accumulation of large volumes of fluid and gas, and this presents with abdominal distension. Initially the peristaltic activity of the dilating gut increases to overcome the obstruction, causing rushes of hyper peristaltic bowel sounds, or high pitched tinkling sounds, or both. Later, as ileus develops, the obstructed gut becomes silent. Intestinal distension causes reflex vomiting. Water and electrolyte absorption is impaired and intestinal secretion is increased in the obstructed bowel. Inadequate fluid intake combined with the loss of fluid, by repeated vomiting and into the lumen of the obstructed gut, depletes the extracellular fluid, so that the child becomes dehydrated, hypovolemic, acidotic and may go into shock.

Proximal obstruction, causing relatively greater vomiting and less intestinal distension, results in the losses of water, Na+, CI-, H+ and K+, producing dehydration with hypochloremia, hypokalemia and metabolic alkalosis. In distal obstruction with relatively greater intestinal distension, the serum electrolyte abnormalities are usually less dramatic.

Intestinal distension also leads to increased intra-abdominal pressure, decreased venous return from the lower limbs and splinting of the diaphragm with impairment of ventilation. During intestinal obstruction, whatever the cause, there is stasis and rapid proliferation of intestinal bacteria, which leads to the small intestinal contents becoming feculent. Bacterial translocation in the distended, obstructed intestine may be responsible for the systemic infections and septic consequences associated with intestinal obstruction.

Strangulation Obstruction

This occurs when the blood supply to the obstructed gut becomes impaired, which may be due to significantly increased intraluminal pressure, or due to a closed loop obstruction.

A closed loop obstruction refers to a mechanical obstruction in which both the afferent and efferent segments of the involved bowel are occluded, and hence, children with this condition are at high risk of developing strangulation, necrosis, and perforation.

Pressure necrosis can develop if unyielding adhesive bands or hernial rings hold the obstructed distending gut. The deformity or twisting of the mesentery, as in volvulus or intussusception, can cause mesenteric vascular occlusion, which is a very dangerous condition and demands early treatment before gangrene of the bowel rises.

Mesenteric vascular occlusion alone may give rise to gangrene without mechanical obstruction. In addition to the effects of strangulation, the child has all the ill effects of simple obstruction.

The loss of blood and plasma in strangulation obstruction is greater in predominant venous vascular obstruction than in predominant arterial vascular obstruction, and it can lead to shock in an already dehydrated child.

Besides the loss of blood and plasma, another important factor in strangulation obstruction is the toxic material in the lumen of the strangulated bowel, which is formed due to the presence of bacteria and necrotic tissue. The ischemic gut mucosa readily permits the transudation of bacteria and toxins prior to perforation, producing systemic effects.

As the strangulated gut becomes gangrenous, it may perforate into the peritoneal cavity, causing generalised peritonitis and septic shock. If it perforates into hernia sac, the transudate containing lethal toxins and bacteria is more localized.

DIAGNOSIS OF INTESTINAL OBSTRUCTION

Intestinal obstruction should be suspected in any child presenting with abdominal pain of acute onset, vomiting, absolute constipation (obstipation) and abdominal distension with or without abdominal tenderness.

Abdominal pain is typically colicky, occurring in paroxysms at 4-5 minute intervals in proximal obstruction and less frequently in distal obstruction. As the bowel distends, its motility is inhibited and the colicky pain may subside.

Closed loop obstructions are associated with sudden onset of severe unremitting abdominal pain, out of proportion to the physical findings.

In high intestinal obstructions like those in the duodenum or upper jejunum, the vomiting appears almost simultaneously with pain, even before the onset of any significant distension and it may be constant, frequent and profuse. In contrast, in low small bowel obstructions, vomiting occurs a few hours after the onset of pain by which time there is significant abdominal distension with or without visible peristalsis.

Physical examination should confirm the diagnosis, determine the level and degree of obstruction, exclude peritonitis, and estimate physiologic compromise and the influence of inter-current illness. The most common systemic manifestations of intestinal obstruction are related to hypervolemia (eg. tachycardia, tachypnea, altered mental status, oliguria, and hypotension).

The abdomen is usually distended and hyper resonant and visible peristalsis may be present. The distension is central in small bowel obstruction and peripheral in large bowel obstruction. Peristaltic activity is more likely to be seen in children with chronic sub-acute intestinal obstruction as in tubercular involvement, or Hirschsprung's disease.

Presence of a visible hernia or a previous laparotomy scar should be noted. Abdominal tenderness is unusual in uncomplicated obstruction. However, localized tenderness, rebound tenderness and guarding suggest peritonitis and strangulation.

Palpation may reveal distended bowel loops of bowel, a hernia, or a mass (intussusception, faeces, or tumour being the most common). The classic sausage shaped lump, hardening under the palpating finger synchronously with the bout of pain, is that of an intussusception. A “doughy” feel of the abdomen on palpation may suggest that the obstruction is probably due to abdominal tuberculosis.

On auscultation, the bowel sounds in intestinal obstruction are usually frequent, high-pitched, tinkling or musical, but in prolonged obstruction the bowel sounds disappear as intestinal motility decreases. In contrast, the abdomen is silent in diffuse peritonitis or in obstruction due to paralytic ileus.

INVESTIGATION

Investigations are usually done to confirm the clinical suspicion and assess the general condition of the child. The laboratory tests include haemoglobin estimation with a complete blood count serum Na+, Cl+, K + and creatinine, blood urea and urine analysis.

Radiologic Investigations

Radiographs usually confirm the clinical diagnosis and define more accurately the site of obstruction. Four X-ray views of the abdomen, including an upright chest, upright abdomen, supine abdomen and a left lateral decubitus view, are advisable in all patients with intestinal obstruction. The abdominal X-rays may reveal abnormally large quantities of gas in the bowel. In supine views, the distended small bowel is identified by the presence of valvulae conniventes whereas, the presence of haustral markings indicate colonic distension.

The pattern of air-fluid levels in the upright views, in conjunction with the clinical history can help differentiate between a proximal small bowel obstruction or a distal intestinal obstruction. In complete mechanical small bowel obstruction, there is usually no gas visible distal to the level of the obstruction or in the pelvis, a "cut-off" sign which is almost pathognomonic of the condition. The presence of significant amounts of colonic gas should raise the suspicion of a large bowel rather than a small bowel obstruction.

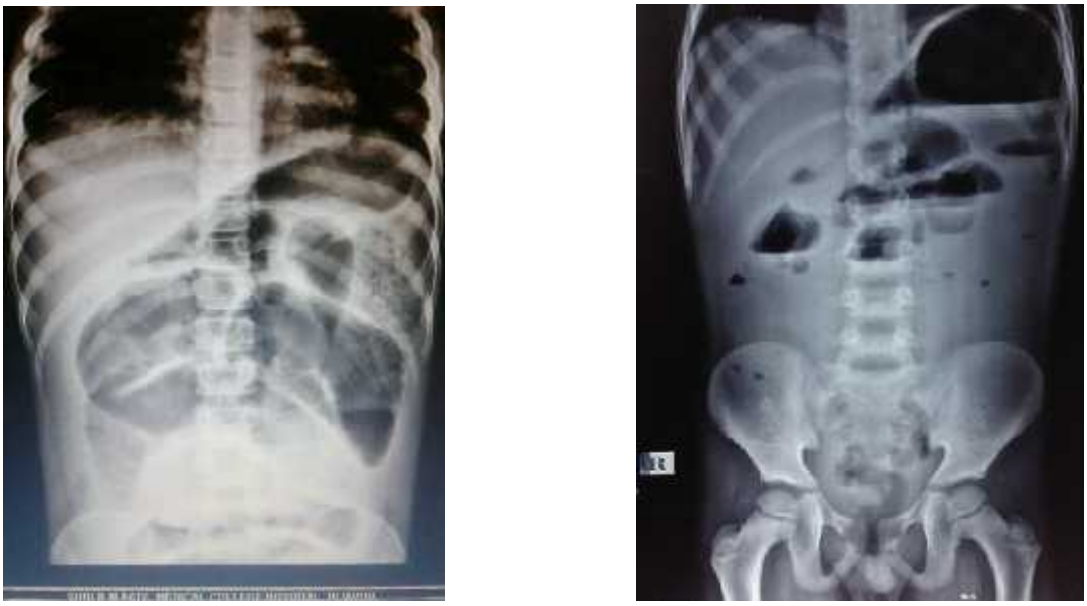


Fig 15: Erect X Ray Abdomen – Showing Multiple Air Fluid Levels

Abdominal ultrasonography has been shown to be useful in the diagnosis of small bowel and colonic obstruction and can help in determining the location and cause of obstruction. It can delineate the mass of an intussusception, which is characteristically seen as a “target lesion".

Okada et al have analyzed hemodynamics in the superior mesenteric artery using pulsed Doppler sonography and have found it useful in differentiating strangulating obstruction from simple obstruction.

CT is sensitive for diagnosing complete obstruction of the small bowel and for determining the location and cause of obstruction. On CT, the criteria for small bowel obstruction included a discrepancy in calibre between the proximal dilated and more distal collapsed loops, or generalized small – bowel dilatation (>2.5 cm) in the presence of a collapsed colon.

Treatment of Intestinal Obstruction

The overlapping sequence of events in the management of children with intestinal obstruction should be investigation, resuscitation and operation. Active preoperative management is needed to stabilize the child hemodynamically so as to minimize the intraoperative morbidity and mortality.

This involves establishing an intravenous line and replacing fluid and electrolyte deficits and continuing losses; ensuring adequate urine output; and typing and cross-matching blood, if surgery is anticipated. Withholding of all oral intake and intestinal decompression are other important supportive measures in these patients.

Surgery is usually necessary in most patients with suspected complete small bowel obstruction.

The presence of fever, tachycardia, localized tenderness and leucocytosis indicates that surgery is mandatory in these patients, including those with no history of previous abdominal surgery and those with incarcerated external hernias.

Conservative or non-operative therapy can be attempted for 6-12 hours in early simple mechanical obstructions, especially partial or early postoperative obstructions (within 3 weeks of a previous laparotomy), and in-children with recurrent small bowel obstructions.

Operative Treatment

All children with intestinal obstruction undergo surgery under general anaesthesia, administered with an endotracheal tube in place.

Surgery involves relieving the obstruction with resection of gangrenous bowel if present. Lysis of adhesions or division of band(s) relieves obstruction due to adhesions or bands.

Laparotomy, enterotomy and removal of foreign body, bezoar and roundworms, is the treatment in obstruction of small bowel due to these causes. If technically possible, the distended bowel should be emptied of its luminal contents, so as to improve the blood supply of the bowel and facilitate abdominal closure.

INTUSSUSCEPTION

Intussusception is the invagination of the intestine into an adjoining intestinal lumen; it is among the most common causes of acute abdominal pain in children younger than 5 years of age. It is a disease primarily of infants and toddlers, although Intussusception can occur in utero, in neonates and adults. 80% to 90% of Intussusception occurs in children between 3 months and 3 years of age.

Aetiology:

The most common cause of Intussusception is indeterminate and termed Idiopathic. In a number of patients, particularly those younger than 3 years of age, there may be enlargement of Payer's patch. These children may have a viral prodromal illness 5 to 10 days earlier.

In 2% to 12% of all paediatric cases, the Intussusception has an anatomically identifiable lead point. Children with Intussusception secondary to surgical lead points usually require operative treatment because the intussusceptum is rarely reduced by pressure reduction. The frequency of lead points as the cause of Intussusception increases with age, in which the prevalence of lead points complicating Intussusception has been supported to be as high as 57%.

Meckel's diverticulum is the most common anatomic lead point identified in children. Intussusception is a feature of the gastrointestinal problems associated with cystic fibrosis and Henoch-schonlein purpura. Together, these medical processes account for 3% to 5% of cases of Intussusception.

Pathogenesis:

The pathogenesis of Intussusception has been explained to be an inhomogeneity of longitudinal forces along the intestinal wall. In the resting state, normal prospective forces meet a certain resistance at any point. This stable equilibrium can be disrupted when a portion of the intestine does not appropriately promulgate peristaltic waves. Small perturbations provided by contraction of the circular muscle perpendicular to the rise of longitudinal tension results in a kink in the abnormal portion of the intestine, creating a rotary force (torque).

Distortion may continue, infolding the area inhomogeneity and eventually capturing the circumference of the small intestine. This invaginated intestine then acts as the apex of the intussusception.

Pathology:

The intussusception is composed of the internal layer, the returning middle layer and the outer ensheathing layer. The entering and returning layers, including adjacent mesentery, are referred to as the intussusceptum and include any surgical lead point. The receiving or ensheathing layer is referred to as intussuscepiens.

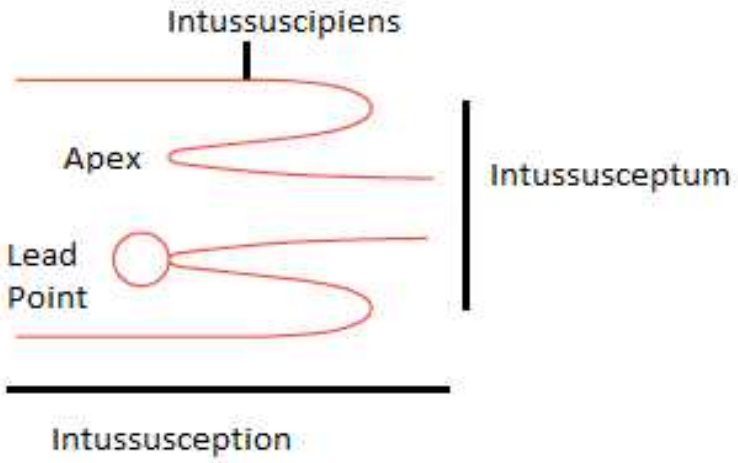


Fig 16: Mechanism and nomenclature of intussusception

With normal peristalsis, the length of the intussusceptions increases, and the cycle of venous congestion, lymphatic obstruction and eventual arterial compromise is initiated. The vascular supply at the apex of the intussusceptions is the most compromised, and the mucosa of the apex experiences secondary sloughing.

The combination of mucous discharge from the bowel and vascular sloughing results in a currant jelly stool.

Radiographic evaluation shows patterns of small bowel obstruction with absence of gas in the colon. The most predictive finding of the disease is the presence of a right upper quadrant soft tissue density, found in 25% to 60% of cases. The absence of bowel gas in the right lower quadrant (Dance's sign) may be identified in the patient with intussusceptions. Other radiographic findings include reduced amount of gas in the jejunum, lateralization of the ileum into the right iliac fossa, indiscernible caecal shadow, and reduced amount of faeces in the colon. Even using these radiographic indicators, 50% to 66% of children who undergo a diagnostic enema for suspected intussusceptions do not have the disease. [49]

Diagnosis of intussusceptions is established by the ultrasonographic demonstration of 'target sign' on transverse views and 'pseudokidney' sign on longitudinal views of the intussusceptions.

Treatment

Often with a clinically suggestive story and nonspecific findings on plain abdominal radiographs, contrast enemas are performed for diagnosis and therapy. Only in children who have clinical peritonitis or radiographic evidence of perforation, attempt at pressure reduction absolutely contraindicated.

Reduction can be done in the following ways

1. Hydrostatic reduction
2. Pneumatic reduction
3. Operative reduction

Hydrostatic reduction[50]

Hydrostatic reduction was proposed by Hirschsprung in 1876 for the treatment of intussusceptions. The use of contrast enemas allow direct visualization of the reduction under fluoroscopic control and is reported to be successful in 65% to 70% of cases. In the hands of an experienced paediatric radiologist, the ratio of successful reduction approach 85%.[43]

A radiographic "rule of threes" applies to hydrostatic reduction in intussusception:

1. The barium contrast column should be no greater than 3ft above the table (100cm)
2. Each attempt should persist until reduction of the intussusceptum fails to progress for a period for 3 to 5 minutes
3. Maximum of 3 attempts should be made

Because most intussusceptions are reduced within the first two attempts, successful hydrostatic reduction after three attempts is unlikely.[42] If two attempts at radiographic reduction are not successful, operation is warranted.

Ultrasound guided reduction

Ultrasound guided reduction of intussusceptions can be performed.

Pneumatic reduction

Pneumatic reduction of intussusceptions has been used extensively in china and is now readily accepted in North America and Western Europe

Operative reduction

In children who have clinical evidence of peritonitis or radiographic evidence of perforation, surgery is indicated after initiating fluid resuscitation and starting broad spectrum antibiotic.

Successful manual reduction can be expected in about 90% of paediatric patients, even if surgical lead points are present. These lead points if identified should be removed and a primary enteroenterostomy should be performed. True irreducibility of the intussusception suggests that gangrenous intestine is present. Even in those instances, a primary resection and anastomosis is appropriate unless the child is so seriously ill that resection and exteriorization are clinically indicated to expedite closure and lessen the anaesthetic risk.

Relevant Studies

Pujari AA et al[27] conducted a retrospective study to examine the aetiologies of acute abdominal emergencies in children. 100 children below the age of 12 years were studied at Indira Gandhi Medical College and hospital, Nagpur. All patients were operated on an emergency basis and the intraoperative findings were correlated with the clinical findings. Acute abdominal emergencies in the paediatric age group were heterogenous, with a myriad of aetiological factors. There were 100 children, 69 (69%) were males and 31 (31%) were females (M: F = 2.22:1). Mean age of presentation was 7.09 years. Seventy-three (73%) patients were greater than 3 years of age. The largest group in this study was acute appendicitis (58%). It was followed by intestinal obstruction (32%). Perforation peritonitis accounted for 7% of the cases. Other causes were primary peritonitis (2%), gastric volvulus (1%) and necrotising enterocolitis without perforation (1%). One patient had features of intestinal obstruction due to Meckel's diverticulum, having band and intraoperative finding of inflammed appendix. Majority of the acute appendicitis (46.55%) occurred in the age group of 10-12 years. The most common symptom of acute appendicitis was abdominal pain and the most common sign was tenderness at McBurney's point. The rate of appendicular perforation was 13.79%.

Tseng YC et al[1] conducted a retrospective study between 2005 and 2007 at Changhua christian hospital Changhua, Taiwan and studied 400 patients who were having acute pain abdomen and divided them into traumatic and non-traumatic groups. In the non-traumatic group (n=335), the most common etiology in infants was incarcerated inguinal hernia (14/31, 45.1%), followed by intussusception (13/31, 41.9%), while acute appendicitis was the major cause in children older than 1 year (68.7%).

In the traumatic group (n=65), the major cause of acute abdomen was traffic accidents (76.9%). In both groups, bowel loop dilation and local ileus were the two most common findings demonstrated by plain film X-rays. The aetiology of acute abdomen varied depending on the age of the patient. Acute appendicitis was the most common cause of acute abdomen in children older than 1 year of age, followed by traumatic injury. Abdominal CT scanning was a useful diagnostic imaging modality in patients with both traumatic and non-traumatic abdominal pain.

Shakya KN et al[26] conducted a prospective study to find out the causes of abdominal pain and their frequency among Nepali children. Subjects included children with abdominal pain who presented at the emergency room and paediatric outpatient department of Kathmandu Medical College during January 2006-December 2007. They were all clinically evaluated and investigated to find out the causes and frequency of their abdominal pain. The outcomes were tabulated and analyzed for interpretation. Of 444 patients attended, 356 completed investigations and came for follow up. Cause of pain abdomen was apparent in 117 (32.9%) only. 91.5% were medical causes, comprising predominantly of diarrheal diseases (28.3%), infantile colic (9.4%), urinary tract infection (7.7%), and acid peptic disease (6.8%). 8.5% of the cases were related to surgical conditions, which needed operative management. Secondary or extra-abdominal causes were found in 20 cases (17.1%). Pneumonia (n=2), functional (n=5), vulvovaginitis (n=2) and infantile colic (n=11) were predominant causes.

Holland et al[40] conducted a quality audit of the case records of 1313 children admitted with acute abdominal pain over a three year period under the care of paediatric surgeons at the Princess Margaret Hospital for Children, Perth. Fifty-four per cent (n = 714) of the patients were discharged without surgical intervention.

In this group the most frequent (70%, n = 503) diagnosis was non-specific abdominal pain (NSAP). Of those children having surgery, 74% (n = 443) had appendicitis proven on histopathology; the remaining appendices (n = 134) were reported as normal and no other surgical cause for the patients symptoms were identified. Only 3.7% (n = 22) of children having surgery had another surgical cause for their pain. Of this group, 11 had adnexal pathology, eight had complications of a Meckel's diverticulum and three had torsion of the omentum. There were no deaths in this series, and 39 patients (3%) had wound infections. Based on these results, they concluded that only 35% of children referred to a surgeon with abdominal pain will actually require surgical intervention, although as a consequence of concern over clinical status an additional 10% will have a laparotomy with normal findings.

Abubakar et al[51] retrospectively studied the factors affecting outcome of emergency paediatric abdominal surgery in children between 0-15 years, admitted to University of Ilorin Teaching Hospital, Nigeria between January 1994 and December 2001. 251 children undergoing emergency surgery were studied. Intestinal obstruction was the most common overall cause of acute abdomen 34.7%. Typhoid perforations accounted for 28.3% and were the most common surgical emergency above 1 year of age. Appendicitis accounted for 21.9%

Chana RS[52] prospectively studied the Role of ultrasonography in the evaluation of children with acute abdomen in the emergency set-up in children presenting to the surgical emergency section of Jawaharlal Nehru Medical College Aligarh. During a period from September 2001 to October 2003, 75 patients (<15 years) who presented with acute abdomen were studied. There were 49 males (65.33%) and 26 females (44.67%), their mean age was 6.5 years (age range 6 days-13 years) and 7.9 years (age range 9 months-15 years) respectively.

Acute abdominal pain was the most common symptom (88.6%) followed by fever (56%) and vomiting (50.67%). Tenderness of abdomen (90.67%) was the commonest sign followed by abdominal distension (72%) and rigidity/ guarding (56%) of abdomen. Of the 75 patients, 45 patients were correctly diagnosed by the clinical examination alone (60%). Similarly when ultrasound was used as the only diagnostic modality, 50 patients (66.6%) were correctly diagnosed. When clinical examination was combined with radiography, diagnosis was established in 48 patients (64%). However, when clinical evaluation, radiography abdomen and ultrasound abdomen all were combined; then diagnosis was established in 74 patients (98.67%). Intestinal obstruction was the most common diagnosis with 30.67% followed by intrabdominal abscess 25.33%. Peritonitis and Acute Appendicitis were seen in 17.33% and 16% respectively. Mesenteric lymphadenopathy was seen in 4% and renal colic was noted in 2.67%.

Erkan T et al[53] conducted a prospective study to analyse the Clinical spectrum of acute abdominal pain in Turkish paediatric patients. Children aged between 2 and 16 years who presented to the emergency department of Cerrahpasa Medical School, Istanbul University between July 2001 and August 2002 with acute abdominal pain were enrolled in this study. The number of children referred to the emergency department was 7442, with 399 (5.4%) of these having acute abdominal pain. The mean age of the study population was 6.9±3.5 years, and 201 of the patients were male. The five most prevalent diagnoses were: (i) upper respiratory tract infection and/or complicated with otitis media or sinusitis (23.7%); (ii) abdominal pain with uncertain etiology (15.4%); (iii) gastroenteritis (15.4%); (iv) constipation (9.4%); and (v) urinary tract infection (8%). The most common associated symptoms were decreased appetite, fever and emesis.

Because of follow-up deficiency the progress of 28 patients was not obtained. Eighty-two children were referred to the department of paediatric surgery, but only 17 of 82 (20.7%) required surgical intervention (15 of these 17 for appendicitis). Eleven patients returned within 10 days for re-evaluation, but the initial diagnosis was not changed. The complaints of 57 patients with uncertain aetiology were resolved within 2 days. They concluded that an acute complaint of abdominal pain was usually attributed to a self-limited disease; however, the percentage of surgical aetiology is not negligible.

Clinical Evaluation of Paediatric Acute Abdomen [54-55]

In evaluating children with abdominal pain, a thorough history is required to identify the most likely cause. An initial evaluation of the history is followed by a physical examination and frequent reassessment to judge the progress of the disease.

Age of onset:

Numerous Studies have shown that age is a key factor in the evaluation of abdominal pain. Certain diseases are more common in certain age groups of children.

Abdominal Pain:

Children who do not verbalise typically present with late symptoms of disease. In these children the initial 24 hour history of vague nausea, periumbilical pain may be unreported or go unnoticed. Any child with pain that localizes to the right lower quadrant should be suspected of having appendicitis.

Enquiry into the location, timing of onset, character, severity, duration and radiation of pain are all important points and must be viewed in the context of the child's age.

History of trauma

A history of recent trauma may indicate the cause of pain. Trauma may go unnoticed in children and hence a thorough physical examination for abrasions and contusions is essential to rule out the possibility of trauma.

Precipitating or relieving factors:

Parietal pain is aggravated by movement, relief of pain after a bowel movement suggests a colonic source and relief after vomiting suggests a source in the more proximal bowel.[48]

Associated symptoms:

In acute surgical abdomen, pain generally precedes vomiting and the reverse is true in medical condition. Any child presenting with bilious vomiting should be presumed to have a bowel obstruction. Diarrhoea often is associated with gastroenteritis or food poisoning, but it can also occur with other conditions. Bloody diarrhoea is much more suggestive of inflammatory bowel disease or infectious enterocolitis.

The classic “currant jelly stool” is often seen in patients with intussusception. Failure to pass flatus / faeces suggests intestinal obstruction. Urinary frequency, dysuria, urgency and malodorous urine suggest a urinary tract infection.

Cough, shortness of breath and chest pain point to a thoracic source. Joint pain, rash and smoke – coloured urine suggest Henoch- schonlein Purpura.

Past Health:

All previous hospitalizations or significant illness such as sickle cell anaemia and porphyria should be noted. A history of surgery not only can eliminate certain diagnosis but also can increase the risk of other, such as intestinal obstruction from adhesions. A history similar pain may suggest a recurrent problem.

Drug history:

A detailed drug history is important, because certain drugs may cause abdominal pain. Drugs like: Erythromycin Salicylates Lead poisoning Venoms.

Family history:

A family history of sickle cell anaemia or cystic fibrosis may indicate that diagnosis. The patient’s ethnic background is important because sickle cell anaemia is most common in blacks of African origin and in India children from northern states.

Physical examination

General appearance:

In general, children with visceral pain tend to writhe during waves of peristalsis, while children with peritonitis, remain, quite still and resist movement. The hydration of the child should also be assessed.

Vital signs:

Fever indicates an underlying infection or inflammation. High fever with chills is typical of pyelonephritis and pneumonia. Tachycardia and hypotension suggest hypovolemia. Hypertension may be associated with Henoch-schonlein purpura or haemolytic uremic syndrome. Kussmaul's respiration indicates diabetic ketoacidosis.

Table 5: Paediatric Vital Signs[56]

Pediatric normal vital signs

	0–3 mo	3–6 mo	6–12 mo	1–3 y	3–6 y	6–12 y	>12 y
Heart rate (beats/min)	100–150	90–120	80–120	70–110	65–110	60–95	55–85
Respiratory rate (breaths/min)	35–55	30–45	25–40	20–30	20–25	14–22	12–18
Systolic blood pressure (mm Hg)	65–85	70–90	80–100	90–105	95–110	100–120	110–135
Diastolic blood pressure (mm Hg)	45–55	50–65	55–65	55–70	60–75	60–75	65–85

Abdominal examination

The abdominal examination should begin with visualization of obvious abnormalities, such as distension, bruising, or masses. The breathing pattern should be observed, and the patient should be asked to distend the abdomen and then flatten it.

After the child is asked to indicate, with one finger, the area of maximal tenderness the abdomen should be gently palpated moving toward (but not palpating) that area. The physician should examine for Rovsing's sign (when pressure on the left lower quadrant distends the column of colonic gas, causing pain in the right lower quadrant at the site of appendicular inflammation).

Then gently assess muscle rigidity. Gentle percussion best elicits rebound tenderness. Deeper palpation is necessary to discover masses and organomegaly

Rectal and pelvic examination

These examinations should be used when significant information is sought or expected.

Associated signs

Jaundice suggests haemolytic or liver disease. Pallor and jaundice point to sickle cell crisis. A positive iliopsoas test (passive extension of the right hip and flexion of the right thigh against resistance) or obturator test (rotation of the right flexed hip) suggests an inflamed retrocaecal appendix, a ruptured appendix or an iliopsoas abscess. Purpura and arthritis suggest HSP.

INVESTIGATIONS

Specific Investigations often help in reaching an accurate diagnosis. Laboratory investigations should be tailored. Non-invasive investigations are better tolerated by the younger child. While ordering blood investigations it is a helpful practice to draw sufficient samples of blood to facilitate all investigations in a single attempt rather than multiple blood sampling.

A complete blood profile is often the first basic investigation sought. A leucocytosis with neutrophilia points to an acute infective or inflammatory pathology. A low haemoglobin and platelet count suggest blood loss or an underlying hematologic disorder. Urine analysis can help identify urinary tract pathology, such as infection / stones.

Acute phase proteins are plasma proteins, mostly synthesized in the liver whose plasma concentrations may increase several hundred fold as part of the response to inflammatory stimuli. Three of the best known examples of these protein are C-reactive protein (CRP), fibrinogen and serum amyloidal A protein (SAA). Synthesis of these molecules by hepatocytes is up regulated by cytokines, especially IL-6 (for CRP and fibrinogen) and IL – 1 or TNF (for SAA). Many acute phase proteins, such as CRP and SAA bind to microbial cell walls, and they may act as opsonins and fix complements. They also bind chromatin, possibly aiding in the clearing of necrotic cell nuclei.

During the acute phase response, serum amyloid A protein replaces apolipoprotein A, a component of high density lipoprotein particles. This may alter the targeting of high density lipoproteins from liver cells to macrophages, which can utilize these particles as a source of energy producing lipids.

The rise in fibrinogen causes erythrocytes to form rouleaux that sediment more rapidly at unit gravity than do individual erythrocytes. This is the basis for measuring the erythrocyte sedimentation rate as a simple test for the systemic inflammatory response, caused by any number of stimuli including Lipopolysaccharides.

Most studies on the value of CRP in patients with an acute abdomen have focused only on acute appendicitis. Eriksson et al [57] studied the value of repetitive CRP and white all count in patients already considered for Appendicectomy. They found that a normal value for both tests should be an indication to defer surgery.

Ultrasonography is one of the most helpful non-invasive investigations to diagnose the paediatric acute abdomen. Ultrasonography, unlike CT or fluoroscopy, does not use ionising radiation. Ultrasound can be performed in any imaging plane which is advantageous when evaluating such structures as the pylorus and appendix, which may not be fixed in their orientation, small children with abdominal pain often are not able to lie down quietly for a CT or MR image without the use of sedation. US, however, is able to obtain diagnostic images in non-sedated children. It is also cost effective, being less expensive than CT or MR imaging. Real time ultrasonography can be performed in the radiology department or at the bedside in the emergency department. [52]

Plain abdominal radiographs are most useful when intestinal obstruction or perforation of a hollow viscous in the abdomen is a concern. Multiple air fluid levels indicate intestinal obstruction while air under the domes of diaphragm is often sought in suspected perforations.

Chest radiographs may help rule out pneumonia. Abdominal Films also reveal renal, ureteric and bladder calculi.

A CECT Abdomen and Pelvis may be considered when the above investigations have failed to demonstrate the exact nature of the disease and when the clinical condition is not amenable to conservative management. Pancreatitis, Ureteric Calculi, Intussusception and Appendicitis are sometimes better picked up by the CECT scan.

Other investigations including stool for occult blood, stool for ova and cysts, electrolytes, renal functions, liver function tests and coagulation profiles may be required for select cases.

Treatment

Severe abdominal pain should be relieved as soon as possible. Parental anxiety should not be discounted and is often of significance even if the child does not appear especially unwell. Treatment should be directed at the underlying cause. In many patients, the key to diagnosis is repeated physical examination by the same physician over an extended time.

Indications for surgical intervention in children with acute abdominal pain:

a) Severe or increasing abdominal pain with progressive signs of deterioration

b) Bile stained or feculent vomitus

c) Involuntary abdominal guarding / rigidity

d) Rebound abdominal tenderness

e) Marked abdominal distension

f) Signs of acute fluid or blood loss into the abdomen

g) Significant abdominal trauma

h) Abdominal pain without an obvious aetiology

i) Suspected surgical cause for the pain

Non Specific Abdominal Pain and Mesenteric Lymphadenitis are managed conservatively. Some Surgeons advocate a diagnostic laparoscopy and Appendicectomy in children with multiple attacks of acute mesenteric lymphadenitis. Analgesics and broad spectrum antibiotics are the main stay in these conditions with attention to fluid and electrolytes.

Acute Appendicitis, Intestinal Obstruction, Peritonitis require surgery in addition to analgesics, antibiotics and fluid management. Laparoscopy is advantages in children as it reduces the post-operative pain, and facilitates earlier return to work. Urological interventions are required in children with diagnosed urolithiasis.

The clinical evaluation, investigations and Management of a child with acute abdomen ideally should all happen simultaneously to facilitate early interventions. Parental counselling is as essential as the above as parental anxiety often creates confusion to the severity of diagnosis.

MATERIALS AND METHODS

Source of Data

Children less than 14 years of age (excluding neonates), who presented with acute pain abdomen and were admitted in Shri. B.M.Patil Medical College, Hospital and Research Center Bijapur, between October 2012 to May 2014 were included in the study. The following criteria were included in selection of cases.

Inclusion Criteria

Children less than 14 years (excluding neonates) presenting to the hospital with symptoms of acute pain abdomen, the severity of which necessitated hospital admission.

Exclusion Criteria

1. Neonates
2. Children above 14 years of age
3. Children presenting with chronic pain abdomen
4. Traumatic pain abdomen
5. Children with proven medical cause of abdominal pain
6. Children with previous history of abdominal surgery

SAMPLING

Study Duration - October 2012 to May 2014 (1 year 8 months)

Study Design - Prospective Study

Study Type - Observational and Descriptive

Sample Size - 131

Based on the above inclusion and exclusion criteria a total of 131 children were included in the study. All children were thoroughly examined and relevant investigations performed preoperatively. A Complete Blood Count, Urine examination, Erect X ray Abdomen and USG Abdomen was done for all children. Other specific investigations such as Stool routine with stool for occult blood and ova cysts, CRP, Liver function tests, CECT Abdomen, Chest X Ray, Electrolyte and Renal profile were done when required.

Consent was obtained for documenting all clinical data and inclusion into the study. All clinical data were recorded as per a standardized proforma. Children were either treated conservatively or operatively. Details regarding, intraoperative findings, postoperative complications and total duration of hospital stay were recorded.

Children with a diagnosis of acute appendicitis underwent open or laparoscopic Appendicectomy while those with intestinal obstruction or peritonitis were taken for exploratory laparotomy and proceed. In certain children an initial observation and resuscitation followed by operative intervention was followed, such as in pancreatitis, ovarian torsion and sub - acute intestinal obstruction. Urological consultation and interventions were required in children with urolithiasis presenting as acute abdominal pain. Further histopathological reports were collected when available. The children were grouped into the following groups to study the common aetiologies and spectrum of paediatric acute abdomen.

1. Less than 2 years
2. 2 to 5 years
3. 5 to 9 years
4. 9 to 14 years

Statistical - Analysis

A master chart was tabulated including relevant investigations, diagnosis and treatment. The following statistical tools were used to tabulate and compare the results.

1. Diagrammatic presentation
2. Mean ± SD
3. 't' Test
4. χ^2 Test

OBSERVATIONS AND CLINICAL PHOTOGRAPHS

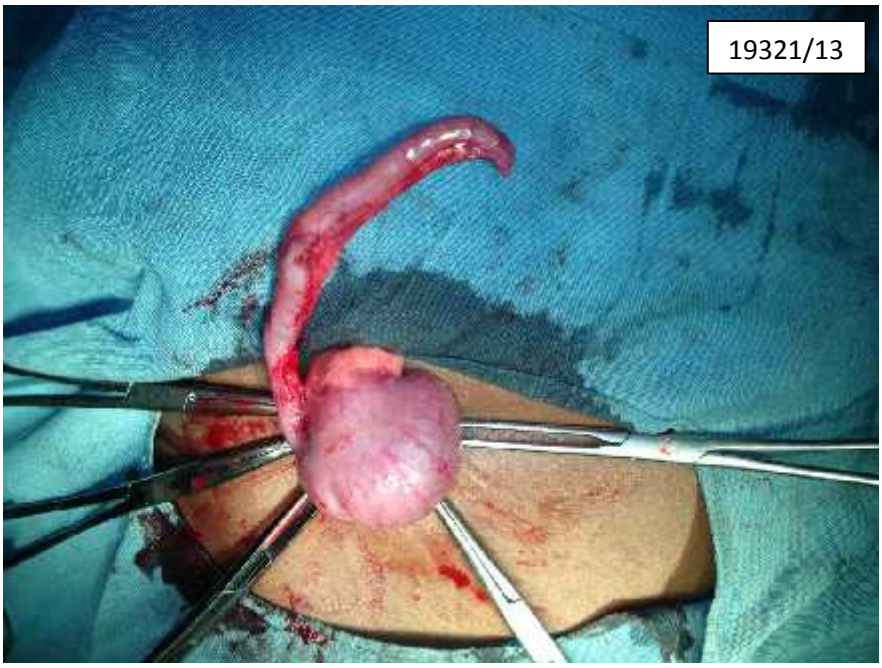


PHOTO 1: ACUTE APPENDICITIS

PHOTO 2: MESENTERIC LYMPHADENOPATHY

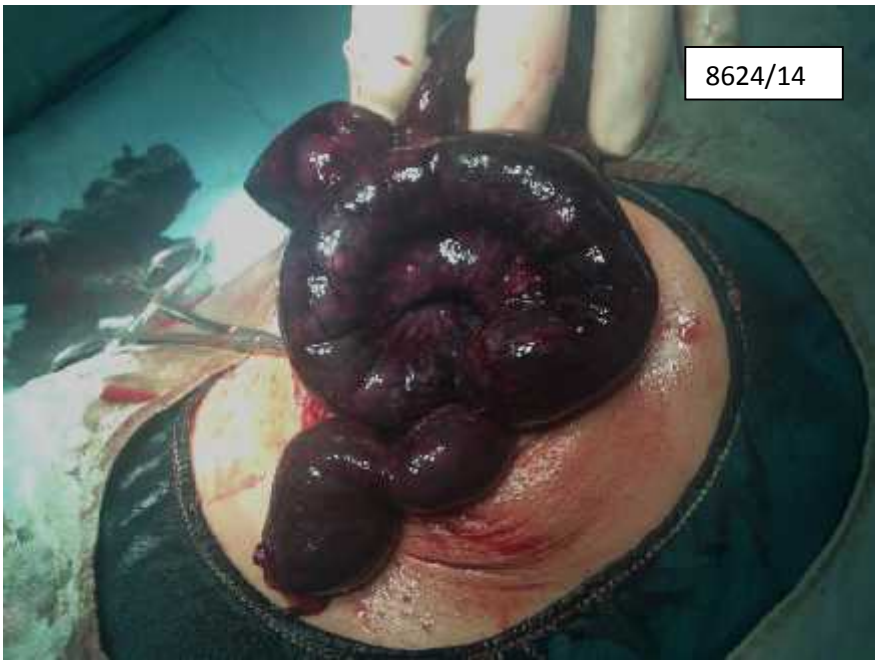
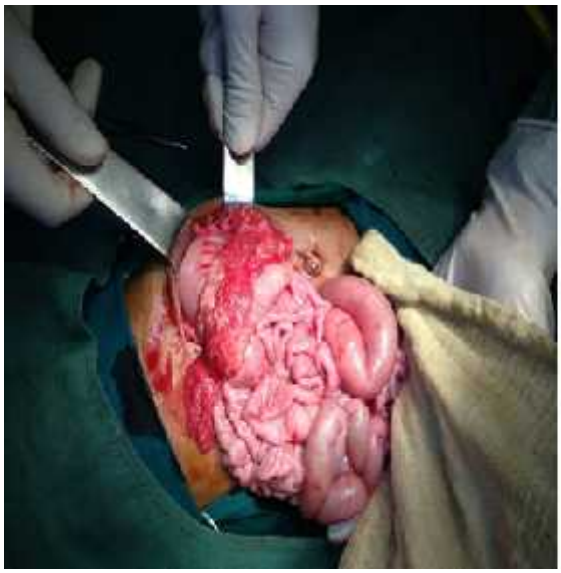


PHOTO 3: INTESTINAL GANGRENE

PHOTO 4: OBSTRUCTED RIGHT INGUINAL HERNIA



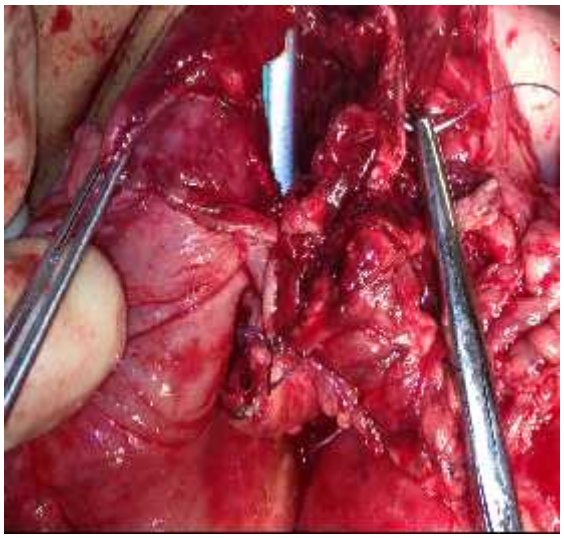
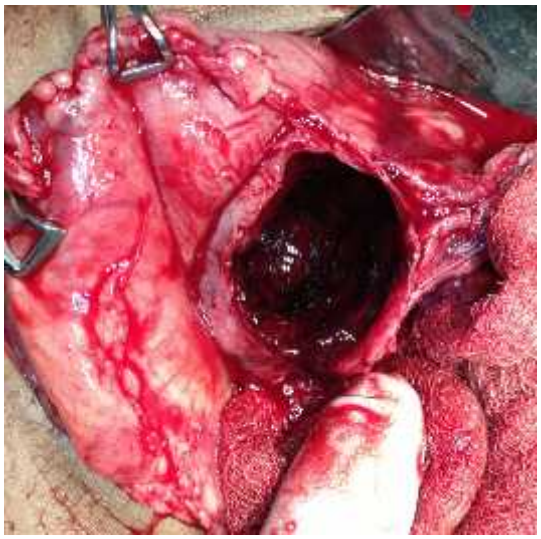




PHOTO 5 (a)-(d): HAEMORRHAGIC PANCREATIC CYST

EXPLORATORY LAPAROTOMY AND EXTERNAL DRAINAGE

PERFORMED

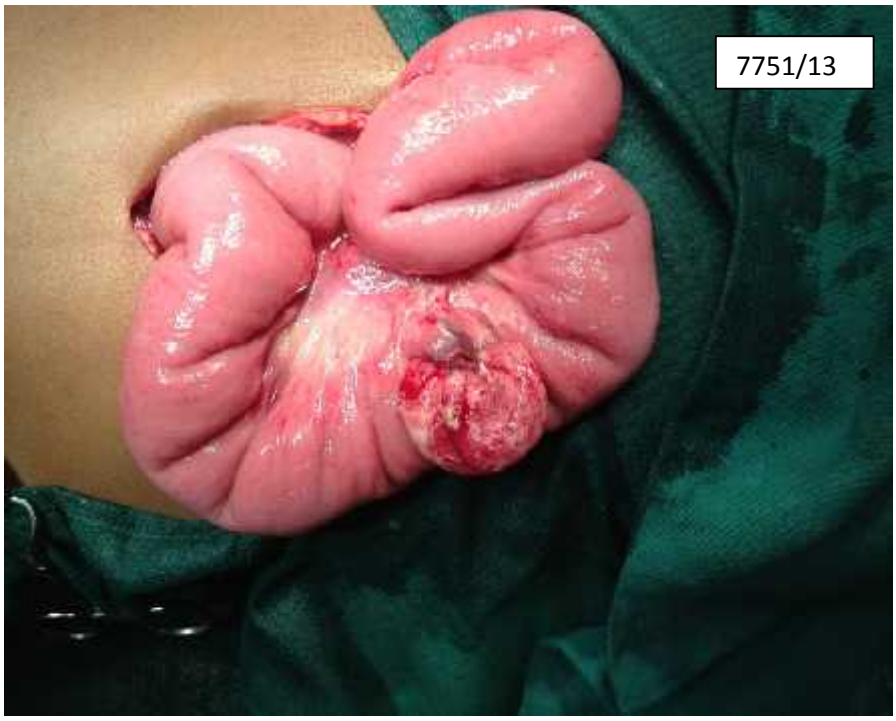


PHOTO 6: MECKEL'S DIVERTICULUM – PERFORATED

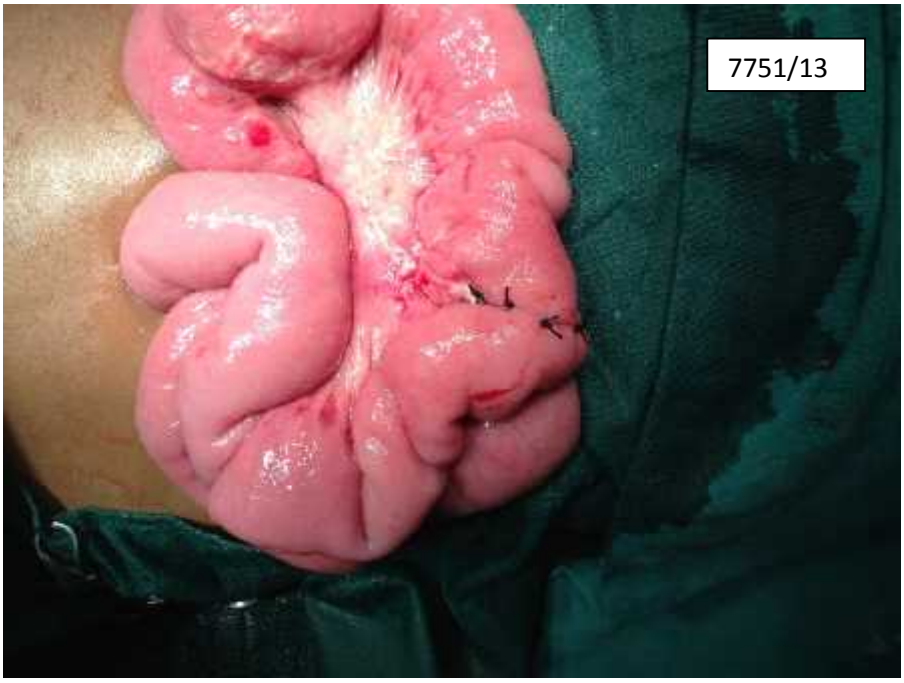


PHOTO 7: MECKEL'S DIVERTICULUM – WEDGE RESECTION

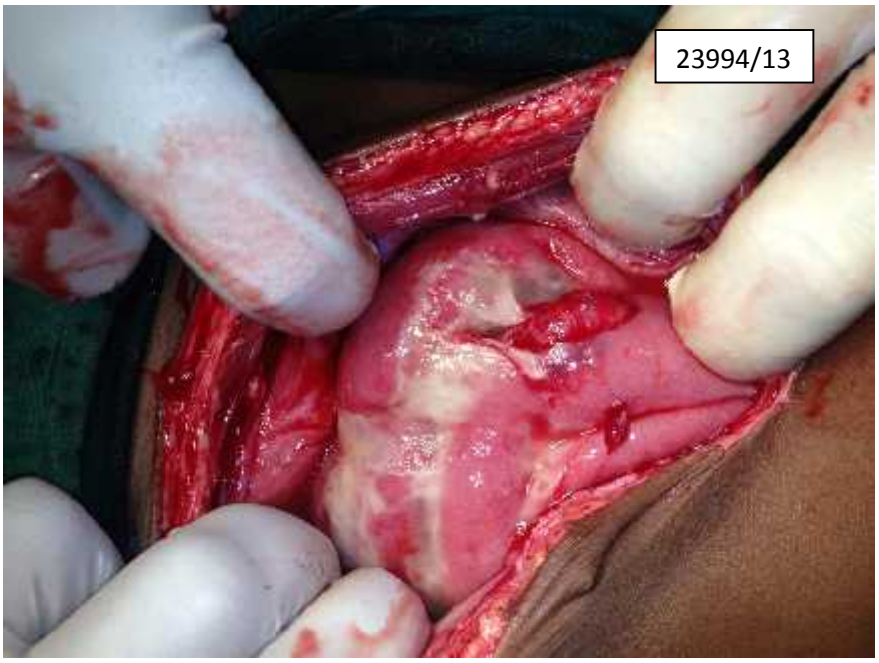


PHOTO 8: PERITONEAL FLAKES – PERFORATED APPENDICITIS

PHOTO 9: PERFORATED APPENDIX

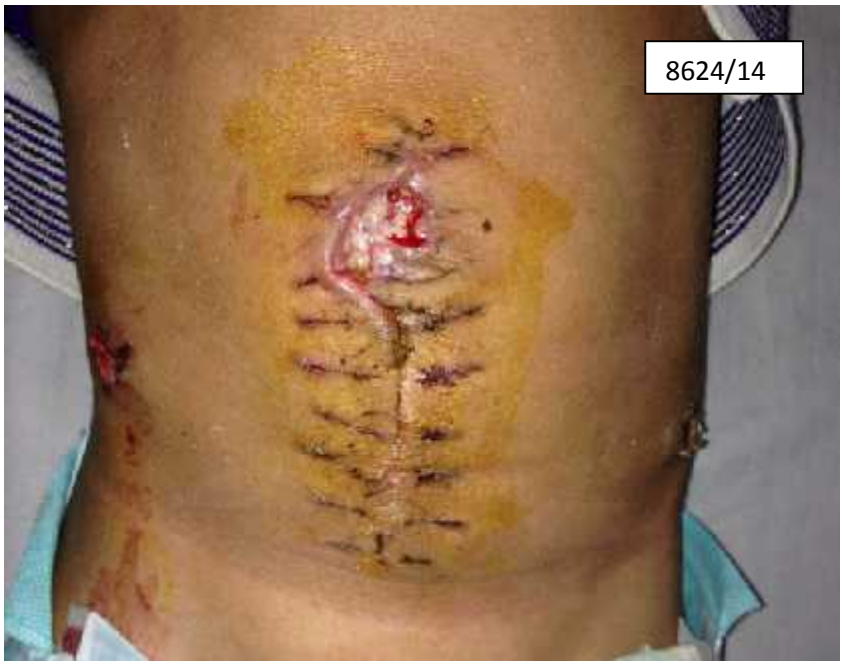


PHOTO 10: SURGICAL SITE INFECTION FOLLOWING LAPAROTOMY FOR PERITONITIS

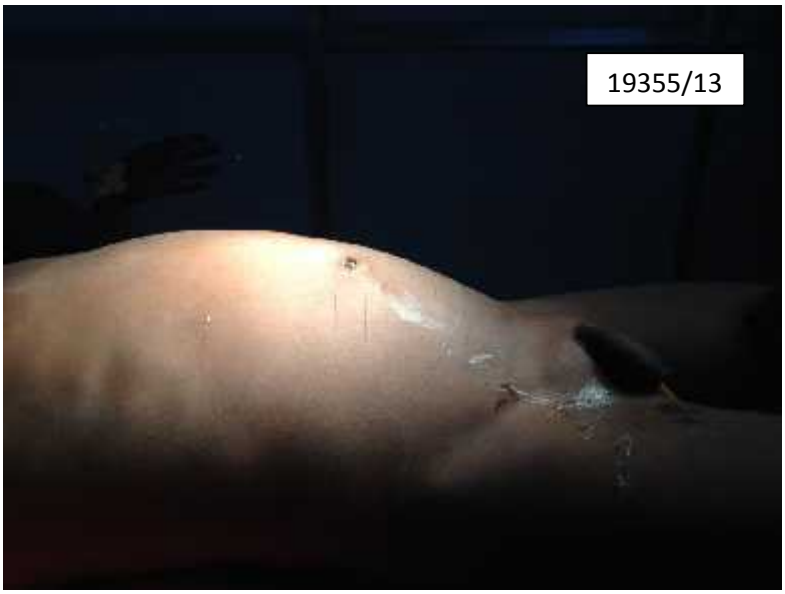


PHOTO 11: ABDOMINAL DISTENSION – INTESTINAL OBSTRUCTION

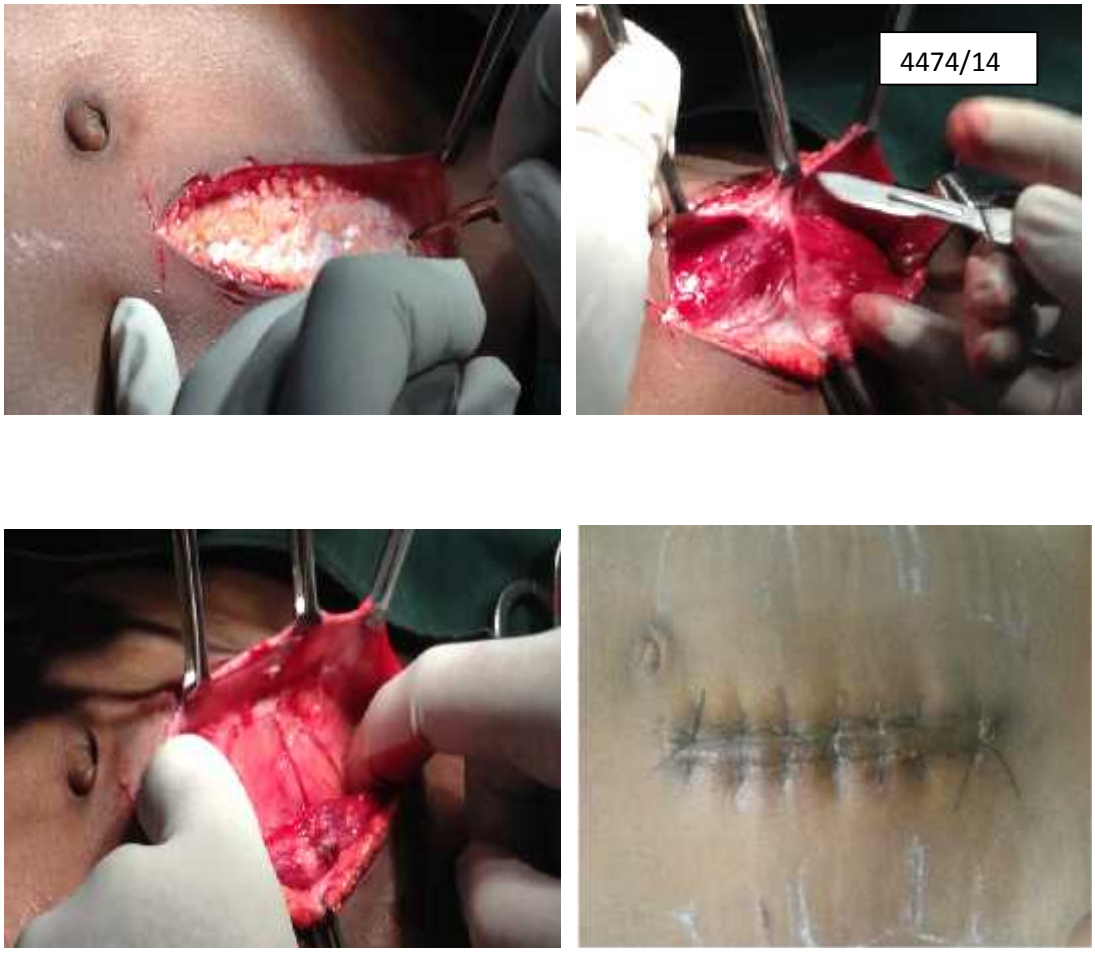


Photo12(a)-(d): Right lower paramedian incision for appendicular perforation

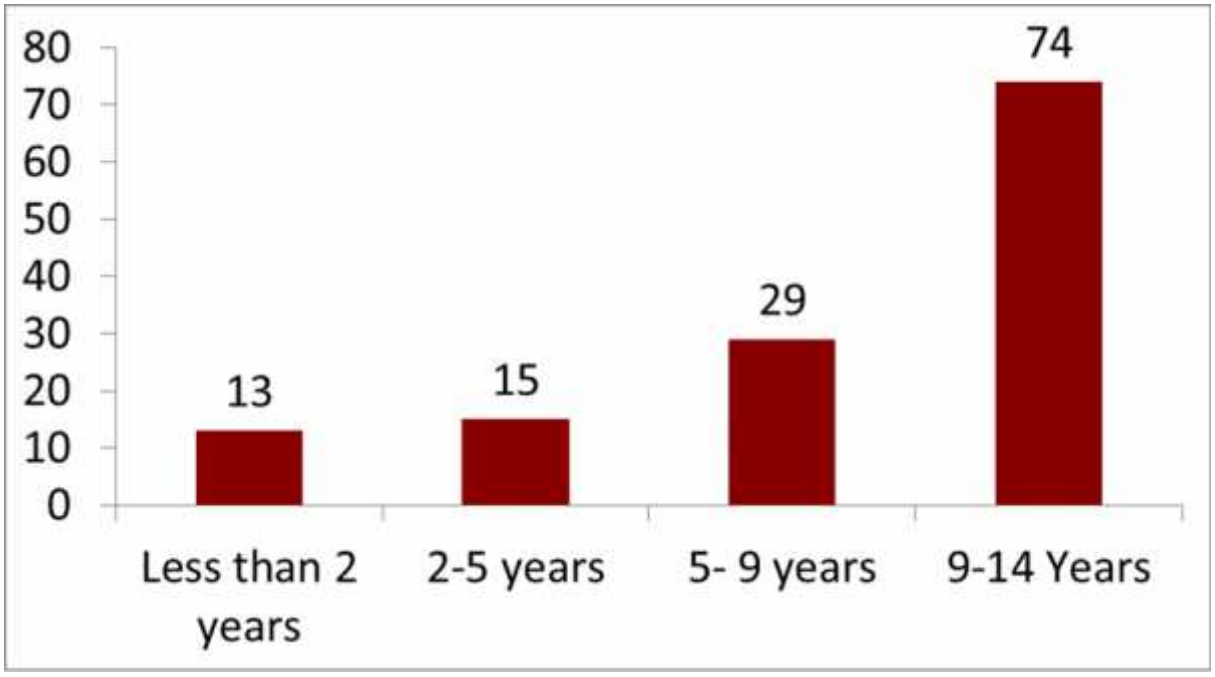
RESULTS

A total of 131children were admitted with the chief complaint of acute abdominal pain in the study institute between October 2012 and May 2014.

Table 6: Age distribution of paediatric acute abdomen

Age in years	Frequency	Percent
<2	13	9.9
2-5	15	11.5
5-9	29	22.1
9-14	74	56.5
Total	131	100.0

Graph 1: Age distribution of paediatric acute abdomen

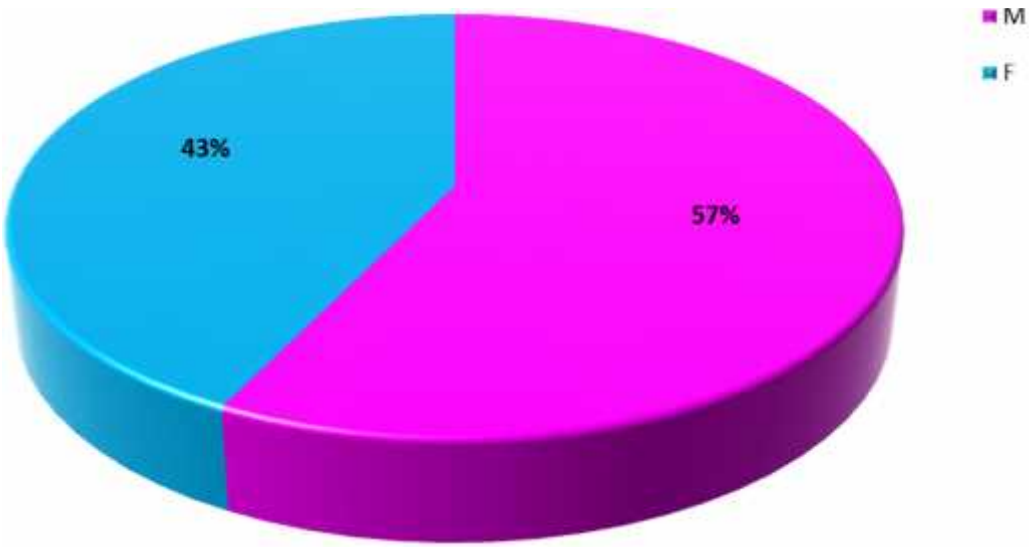


It was observed that acute abdominal pain was most common between 9 to 14 years accounting for 56.5% of the cases

Table 7: Sex distribution of patients

Sex	Frequency	Percent
Male	75	57.3
Female	56	42.7
Total	131	100.0

Graph 2: Sex distribution of patients

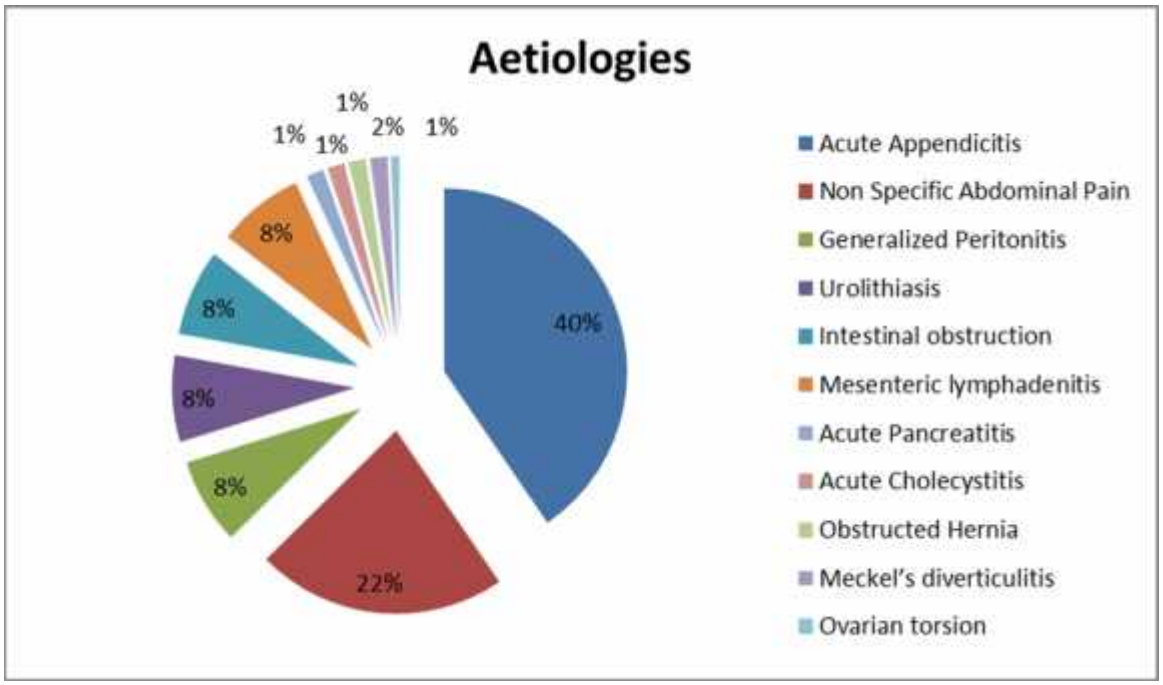


It was observed that there was an overall higher incidence of acute pain abdomen in male children.

Table 8: Aetiological Spectrum of Acute Abdomen

Sl.No	Diagnosis	Frequency
1	Acute Appendicitis	53
2	Non Specific Abdominal Pain	29
3	Generalized Peritonitis	10
4	Urolithiasis	10
5	Intestinal obstruction	10
6	Mesenteric lymphadenitis	10
7	Acute Pancreatitis	02
8	Acute Cholecystitis	02
9	Obstructed Hernia	02
10	Meckel's diverticulitis	02
11	Ovarian torsion	01
	Total	131

Graph 3: Aetiological Spectrum of Acute Abdomen

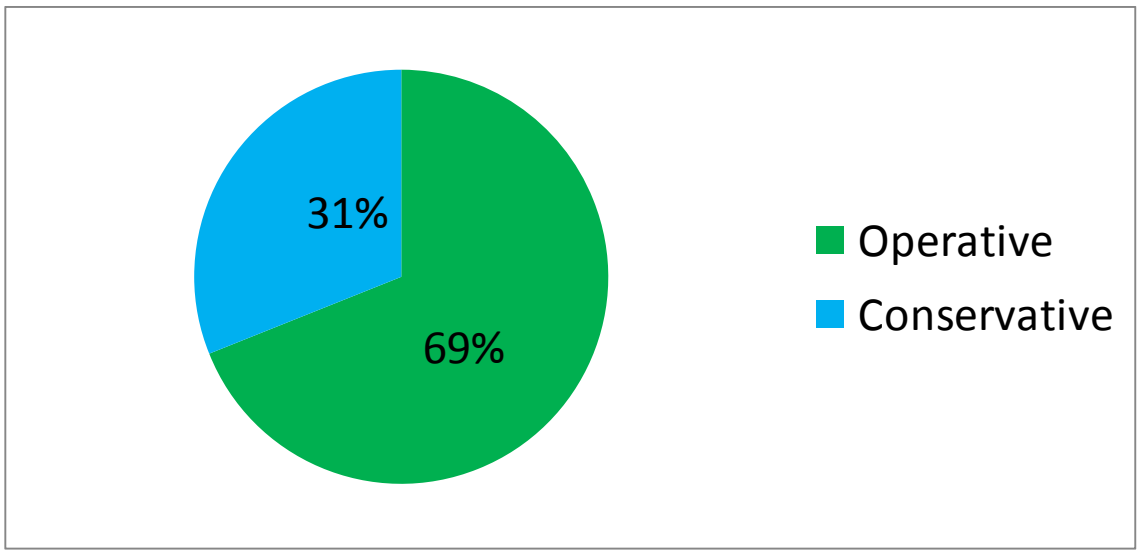


Acute Appendicitis was the commonest cause of acute abdomen accounting to 40% of all diagnosis

Table 9: Treatment of Patients

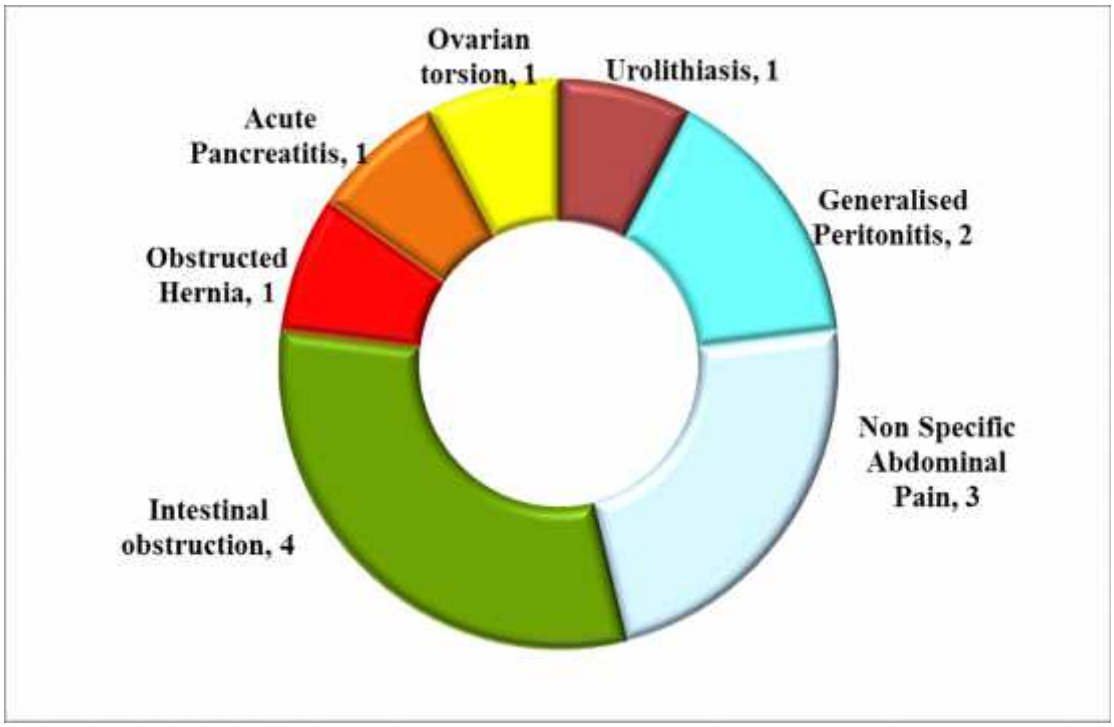
Management	Frequency	Percent
Operative	90	69
Conservative	41	31
Total	131	100.0

Graph 4: Treatment of Patients



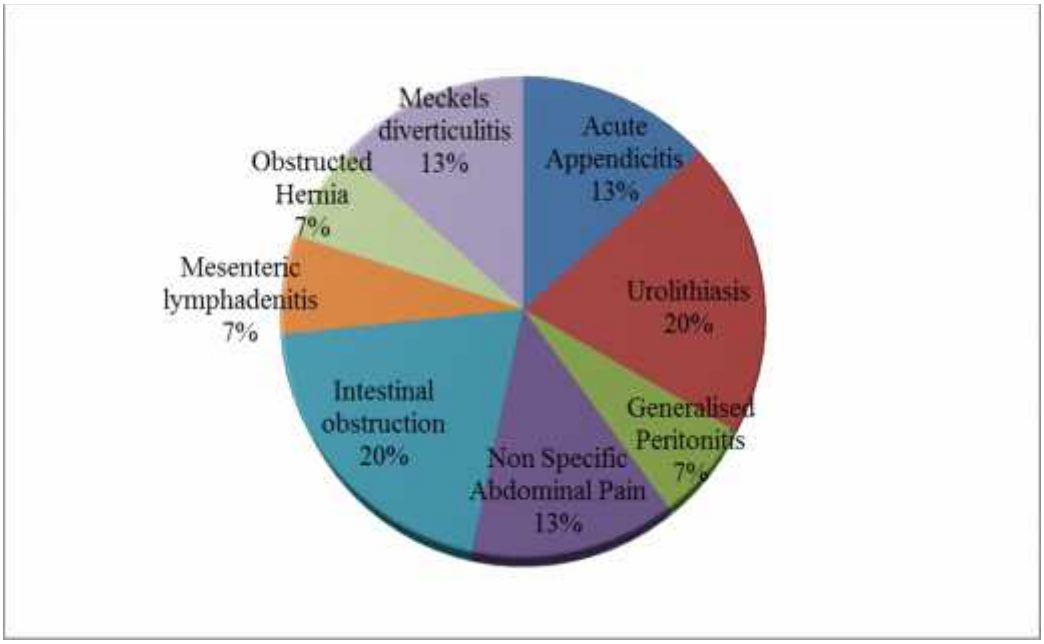
Majority of the children (69%) who presented to our institute with acute abdominal pain underwent surgical intervention.

Graph 5: Aetiology in less than 2 years of age



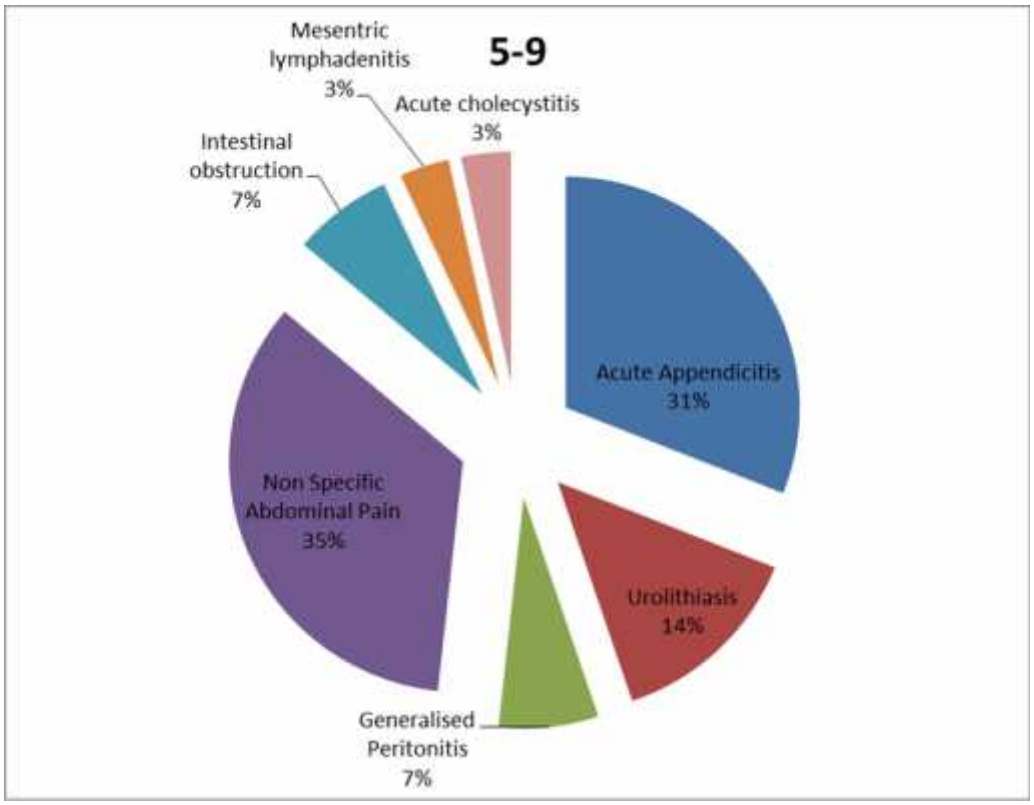
Intestinal Obstruction is the commonest cause of acute abdomen in children less than 2 years of age accounting for 31 % of the cases in this age group. The cause for intestinal obstruction was equally shared by intussusception (Ileocolic variety) and adhesions and bands. It is followed by non-specific abdominal pain accounting for 23 %. There were no cases of Acute Appendicitis and Mesenteric lymphadenopathy diagnosed in this group of children. However, among the two cases that underwent exploratory laparotomy for generalized peritonitis one child had appendicular perforation while the other had perforation of a choledochal cyst.

Graph 6: Aetiology between 2 and 5 years of age



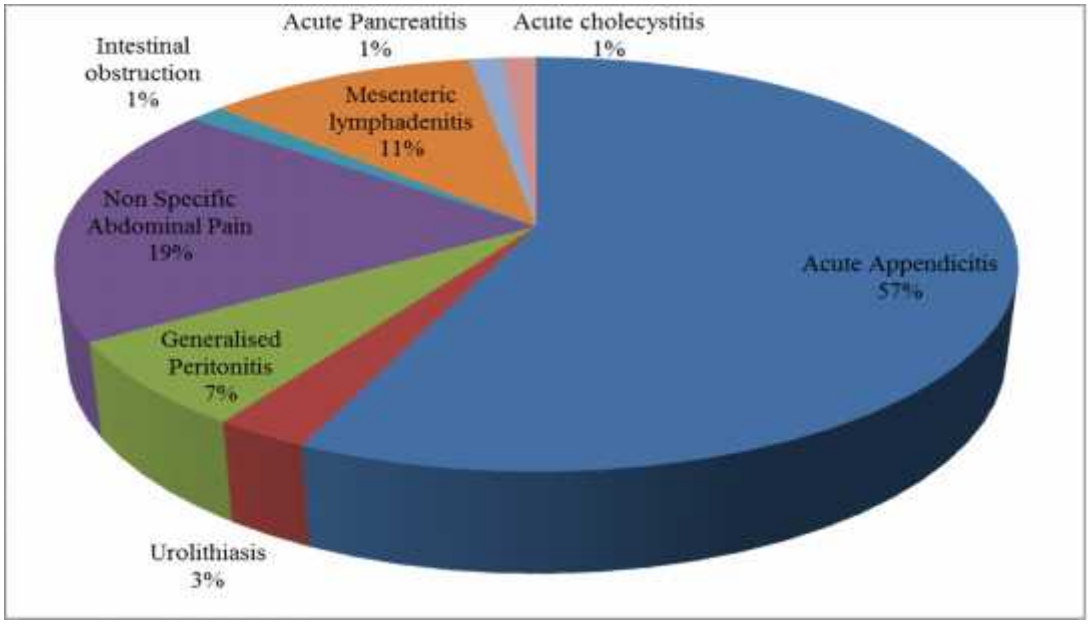
Meckel's diverticulitis was specifically seen only in this age group of children. 2 children out of the 131 accounting for an overall 1.5% of acute abdomen had Meckel's diverticulitis. Intestinal obstruction and urolithiasis were the commonest conditions seen in this age group.

Graph 7: Aetiology between 5 and 9 years of age



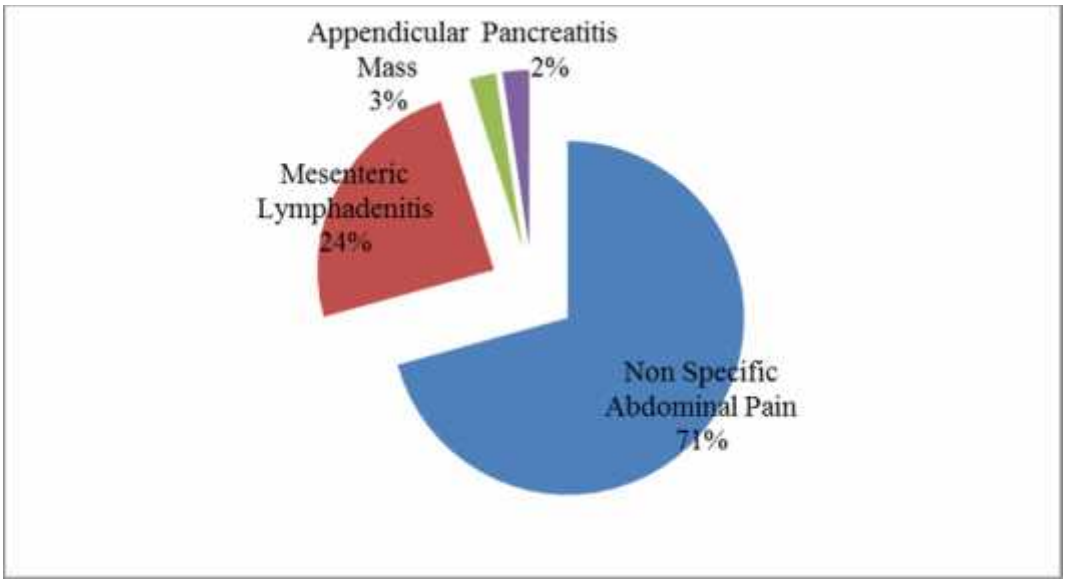
Acute appendicitis and non-specific pain abdomen were the commonest aetiologies in this age group.

Graph 8: Aetiology between 9 and 14 years of age

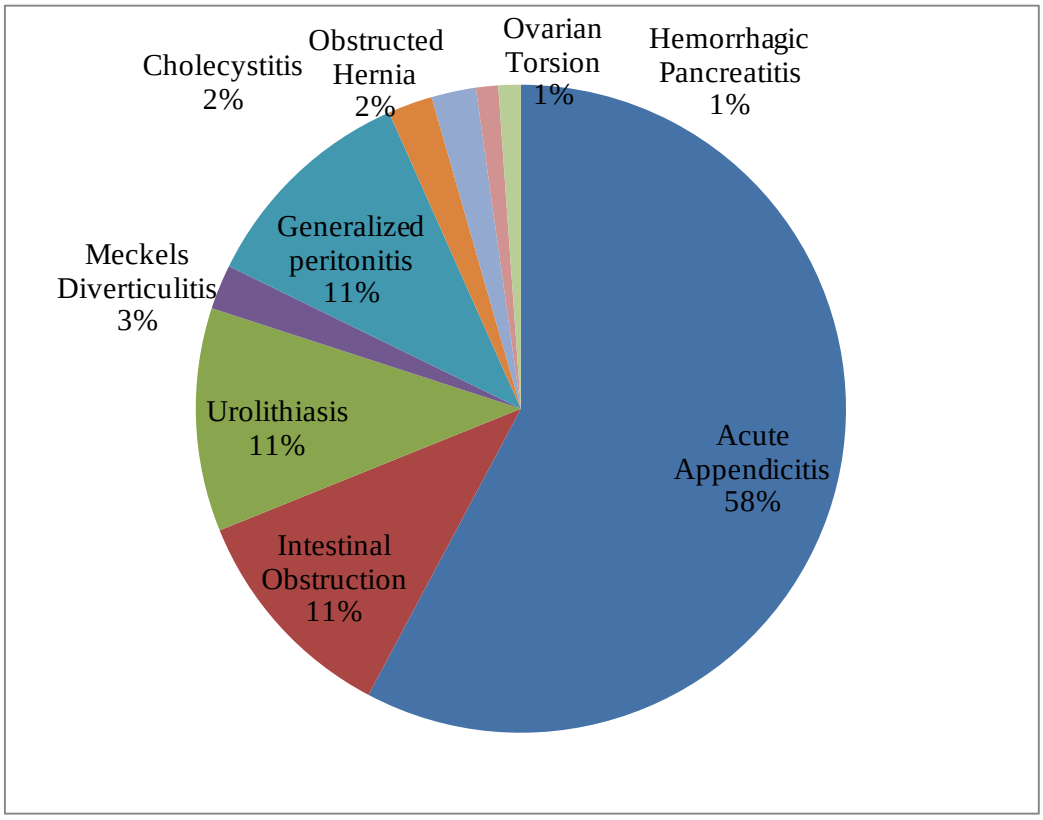


Acute appendicitis and Mesenteric Lymphadenitis are both commonly seen in this group of children. Non-specific abdominal pain is also very common in this group. Intestinal obstruction is less common in this group of children.

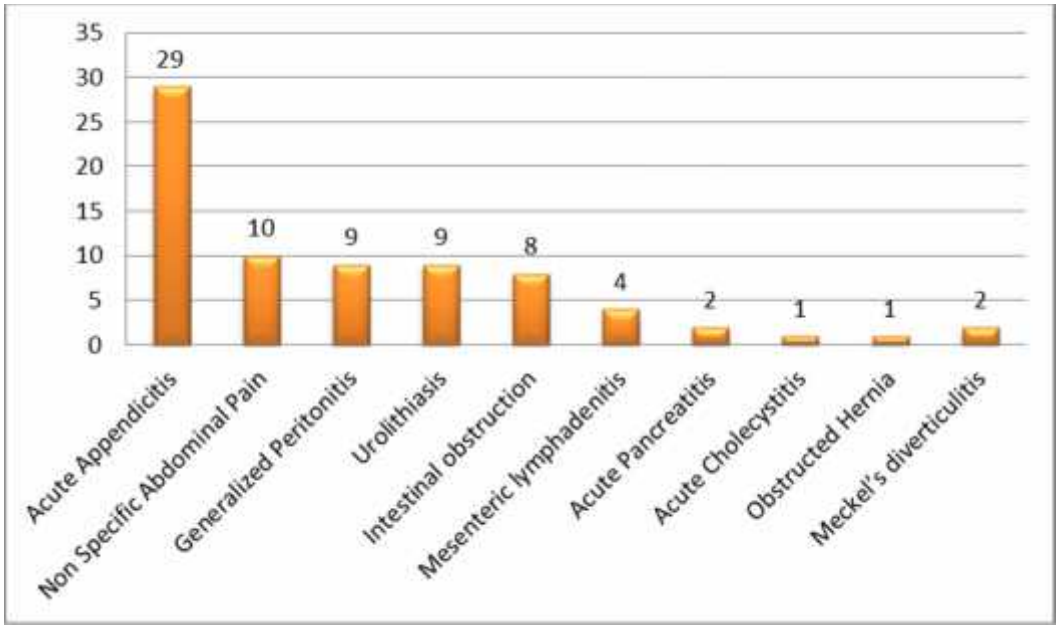
Graph 9: Aetiology of Conservative management



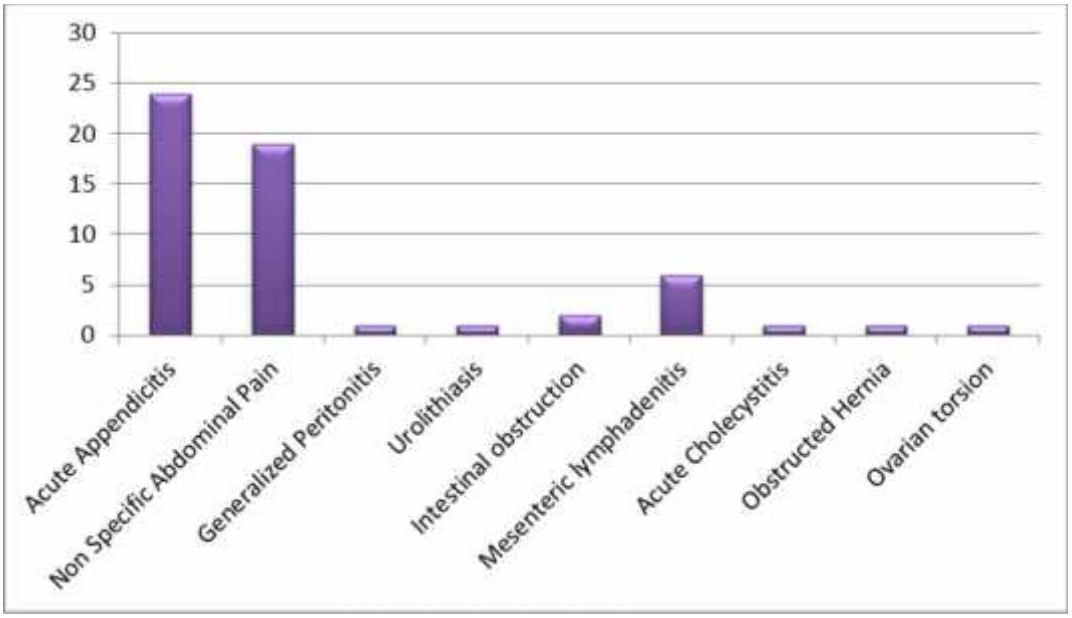
Graph 10: Aetiology of Operative management



Graph 11: Diagnosis in Males



Graph 12: Diagnosis in Females



Graphs 11 and 12 show that while acute appendicitis was seen in both males and females, Nonspecific Abdominal pain and Mesenteric Lymphadenitis had a more female preponderance and Urolithiasis and Peritonitis were seen more in males.

Table 10: Various presentations of Acute Appendicitis

Presentation	frequency	%
Uncomplicated Acute Appendicitis	50	84.7
Acute Appendicitis with omental torsion	01	1.7
Appendicular Mass	01	1.7
Appendicular Perforation with peritonitis	06	10.2
Appendicular Abscess	01	1.7
Total	59	100.0

Graph 13: Presentation of Acute Appendicitis

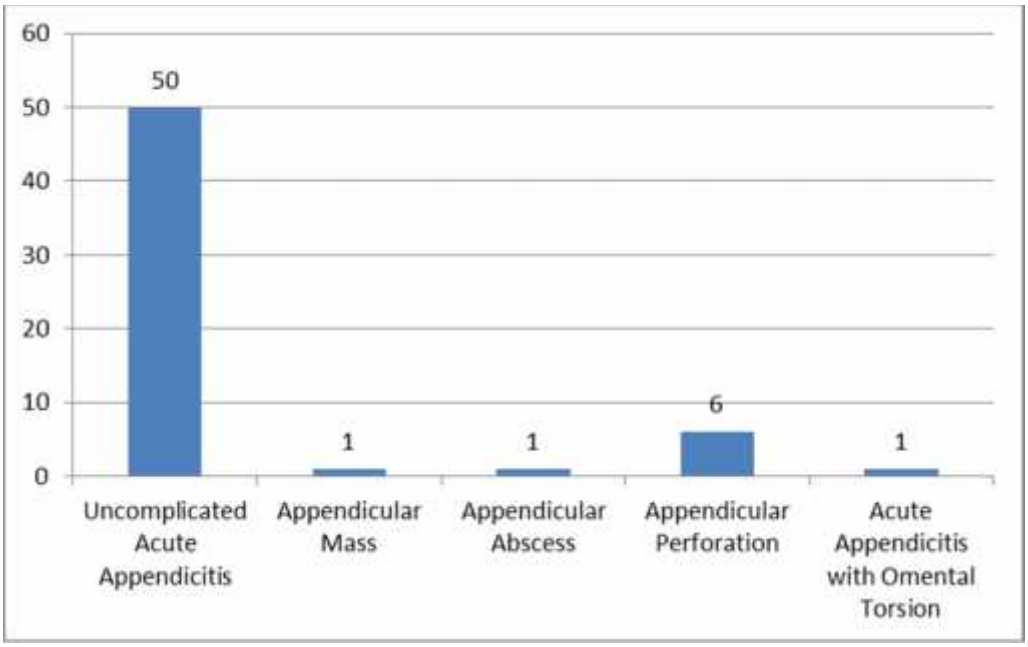
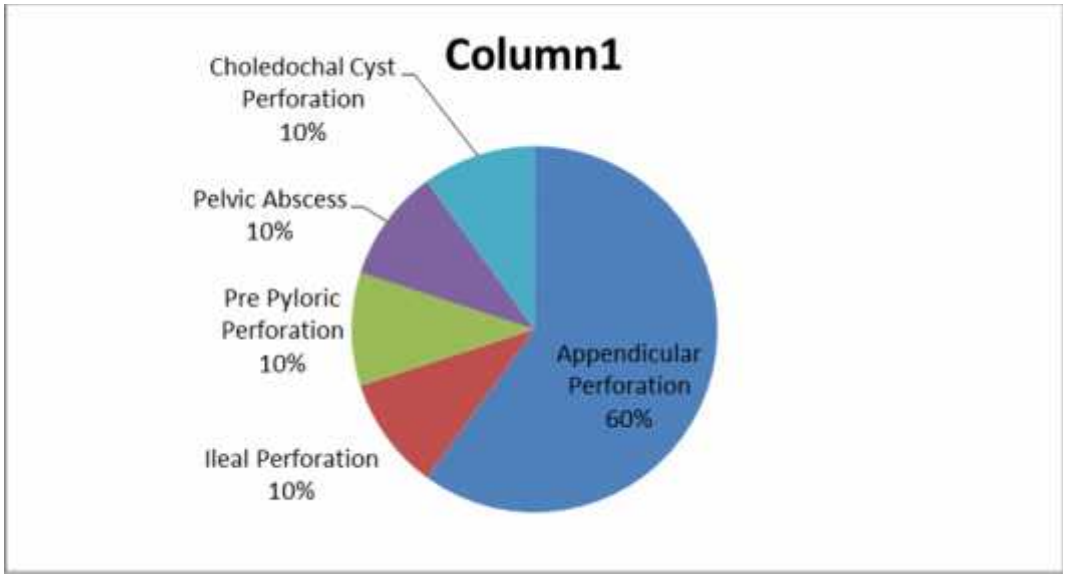


Table 11: Aetiology of Generalized Peritonitis

Causes	Frequency	%
Appendicular Perforation	06	60
Ileal Perforation	01	10
Prepyloric Perforation	01	10
Pelvic abscess	01	10
Choledochal Cyst Perforation	01	10
Total	10	100.0

Graph 14: Aetiology of Generalized Peritonitis

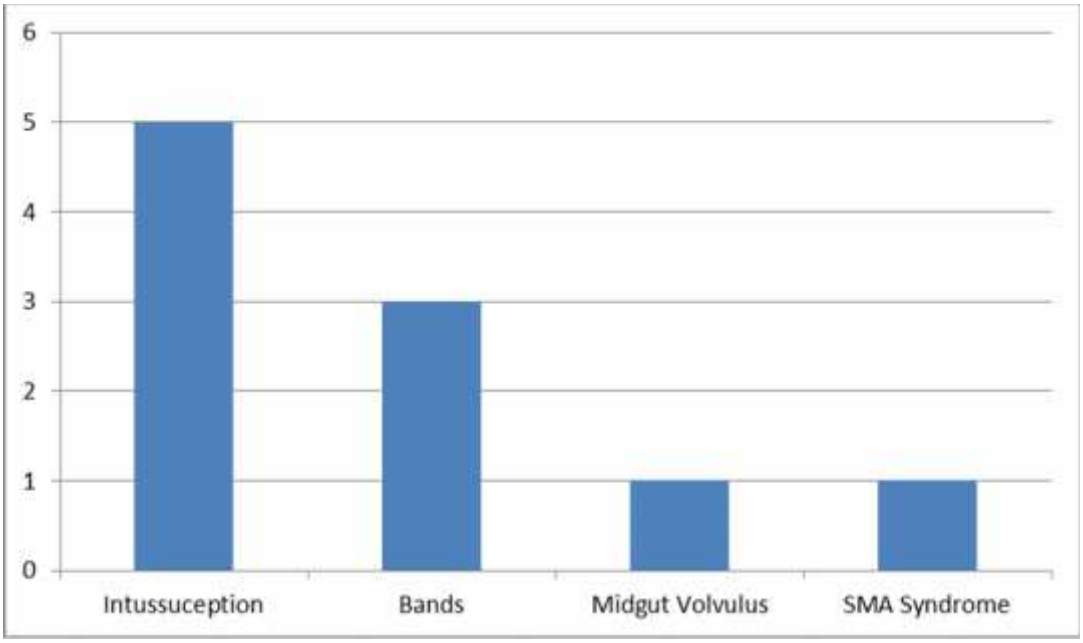


The commonest cause of generalized peritonitis in this study was perforative peritonitis secondary to appendicular perforation.

Table 12: Causes of Intestinal Obstruction.

Causes	Frequency	%
Intussusception	05	50.0
Bands and Adhesions	03	30.0
Midgut Volvulus	01	10.0
SMA Syndrome	01	10.0
Total	10	100.0

Graph 15: Aetiology of Intestinal Obstruction



The commonest cause of intestinal obstruction in children is Intussusception followed by congenital bands and adhesions.

Table 13 : Correlation between Neutrophilia and Operative Management.

Count	Treatment			
	Operative Management	Conservative Management	Total	P-value
32-45	5	7	12	
45-75	35	27	62	
75-96	50	7	57	<0.0001
Total	90	41	131	

Table 14: Correlation between Leucocytosis & Neutrophilia with operative Management.

	DLC (N)				
	N	Mean	Std. Deviation	Std. Error Mean	P-value
Conservative Management	41	59.8537	13.58043	2.12091	<0.0001
Operative Management	90	74.1778	14.63149	1.54229	
	TLC				
Conservative Management	41	7867.3902	2704.16863	422.32019	<0.0001
Operative Management	90	13157.4222	4173.71275	439.94795	

It was observed that elevated neutrophils in the differential leucocyte count and leucocytosis had statistically significant co relations with operative management.

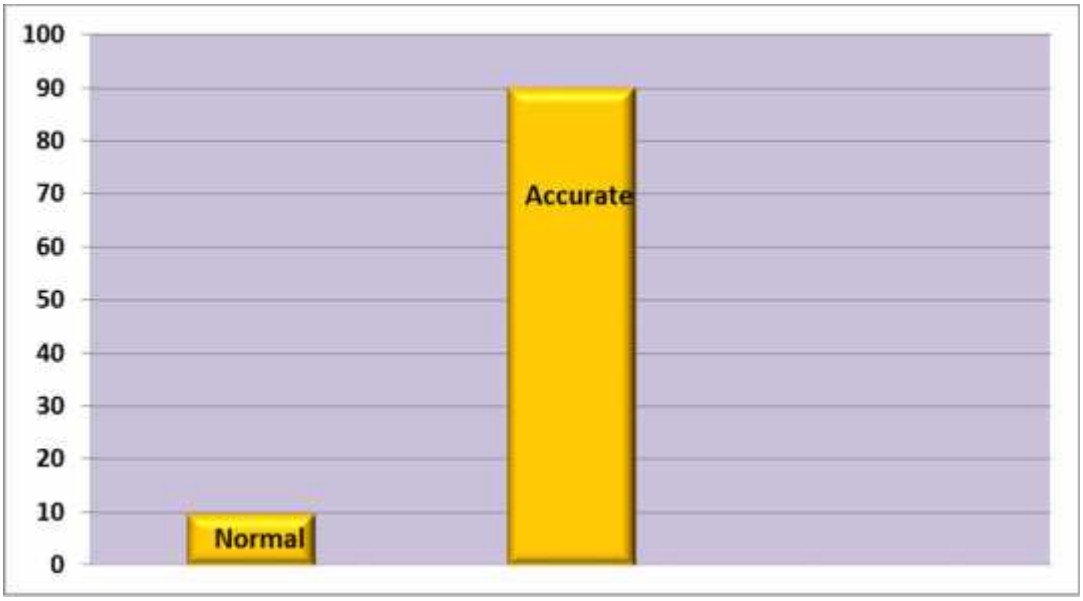
Table 15: Role of USG in diagnosis

	Disease				
Test	Present	n	Absent	n	Total
Positive	True Positive	a= 78	False Positive	b= 0	a + b = 78
Negative	False Negative	c= 12	True Negative	d= 31	c + d = 43
Total		a + c = 90		b + d = 31	

Sensitivity	$\frac{a}{a+c}$	= 86.67 %	95% CI: 77.86 % to 92.91 %
Specificity	$\frac{d}{b+d}$	= 100.00 %	95% CI: 88.58 % to 100.00 %
Positive Likelihood Ratio	$\frac{\text{Sensitivity}}{100 - \text{Specificity}}$		
Negative Likelihood Ratio	$\frac{100 - \text{Sensitivity}}{\text{Specificity}}$	= 0.13	95% CI: 0.08 to 0.23
Disease prevalence	$\frac{a+c}{a+b+c+d}$	= 74.38 % (*)	95% CI: 65.65 % to 81.88 %
Positive Predictive Value	$\frac{a}{a+b}$	= 100.00 % (*)	95% CI: 95.33 % to 100.00 %
Negative Predictive Value	$\frac{d}{c+d}$	= 72.09 % (*)	95% CI: 56.33 % to 84.65 %

In our study USG abdomen had a specificity of 100% and a sensitivity of 86.67% in diagnosing acute abdomen. It was normal in children with non-specific abdominal pain where no organic pathology was identifiable. No false positives were observed with USG.

Graph 16: X-ray finding in intestinal obstruction



Erect X Ray Abdomen was accurate in diagnosis 9 out of 10 cases of intestinal obstruction.

Table 16: Distribution of patients according Duration Of Hospital Stay

DOH	Frequency	Percent
2-8	102	77.9
8-14	22	16.8
14-20	4	3.1
20-26	3	2.3
Total	131	100.0

Most Children with acute abdomen had a hospital stay between 2 – 8 days.

Table 17: Duration of Hospital Stay for various conditions

Diagnosis	N	Mean	Median	Std. Deviation
Acute Appendicitis	53	6.0000	6.000	1.69842
Urolithiasis	10	5.4000	5.000	2.45855
Generalized Peritonitis	10	12.3000	11.00	4.08384
Non Specific Abdominal Pain	29	3.5517	3.000	2.02813
Intestinal obstruction	10	12.4000	11.00	5.71936
Mesenteric lymphadenitis	10	3.6000	3.000	1.07497
Acute Pancreatitis	2	18.0000	23.00	11.31371
Acute Cholecystitis	2	11.0000	11.00	.00000
Obstructed Hernia	2	6.5000	6.500	2.12132
Meckel's diverticulitis	2	11.0000	11.00	1.41421
Ovarian torsion	1	18.0000	--	.

Table 18: Duration of stay in relation to various treatment modalities

Treatment	N	Mean	Median	Std. Deviation
Open Appendicectomy	43	6.000	6.000	1.63299
Lap Appendicectomy	11	5.000	5.000	1.34164
Conservative	42	3.9048	3.000	2.23944
Urological Intervention	10	5.4000	5.000	2.45855
Laparotomy	24	13.1667	11.000	5.18079
Cholecystectomy	2	11.0000	12.500	0.00000
Herniorraphy	2	6.5000	13.000	2.12132

DISCUSSION

All children less than 14 years of age (excluding neonates), who presented to us with non-traumatic acute abdomen were analysed. A total of 131 children were included in the study.

Symptomology

Acute abdomen almost always presents with severe pain abdomen in children, other associated symptoms frequently included distension of abdomen, vomiting, obstipation and fever. Children with Non-Specific pain abdomen and Mesenteric Lymphadenitis also presented with complaints of easy fatigability and upper respiratory tract infections.

Age Distribution

Among the 131 children who presented to us, the highest incidence of acute abdomen was observed in the children above 9 years of age accounting for 56.5% of all admissions followed by children between 5 and 9 years of age accounting for 22.1% of admissions. The least incidence was in children less than 2 years of age with an incidence of 9.9%. These findings are consistent with other studies which have reported that surgical acute abdomen is commoner in the older child, such as Sansi et al and Pillai CKP et al who reported a 36.36% incidence in children above 10years.

Sex Distribution

Our study reports a slight male preponderance in the incidence of acute abdomen. 57.3% of acute abdomen was seen in males while female children accounted for 42.7% of all cases. Shakya et al reported a male incidence of 62.53%.[26]

Aetiology

The commonest cause of surgical acute abdomen in our study was acute appendicitis with a total of 53 children (40 % of the cases) being diagnosed and treated for appendicitis. Non Specific Abdominal Pain was the next common overall aetiology with an incidence of 22%. Intestinal Obstruction, Peritonitis, Mesenteric Lymphadenitis and Urolithiasis were observed in 8 % of the children.

It is interesting to note the high incidence of Urolithiasis in children of Bijapur presenting with acute abdomen. 10 children presented with symptoms and signs of urolithiasis and required surgical intervention. It accounts for a significantly higher incidence when compared to other studies. Shakya et al have reported urinary calculi in only 3 children out of the 444 children they studied.[26] Most other studies have not observed urinary calculi as a significant cause of pain abdomen.

69% of the children with acute abdomen required surgical intervention while 31% were managed conservatively. This is in close comparison to the study by Holland et al [40] in which 46 % required surgery. However this is in contrast to studies by Shakya et al[26] and Tülay Erkan et al [53.] These studies have reported surgical intervention in less than 1.2 to 10.7 % of paediatric acute abdomen. This can be partly explained based on the referral patterns in Bijapur to our institute. Most cases of acute abdomen with no identifiable surgical cause are managed conservatively by a large section of family physicians and paediatricians and seldom referred for surgical consultation, while cases prompting an aggressive management are referred to us for surgical intervention.

Table 19: Common Aaetiologies according to age group

Age group	Conservative	Operative
< 2 years	Non Specific Abdominal Pain (23 %)	Intestinal Obstruction (30%)
2 – 5 years	Non Specific Abdominal Pain (13 %)	Intestinal Obstruction (20%) and Urolithiasis (20%)
5 – 9 years	Non Specific Abdominal Pain (35 %)	Acute Appendicitis (31 %)
9 – 14 years	Non Specific Abdominal Pain (19 %) and Mesenteric Lymphadenitis (11%)	Acute Appendicitis (57 %)

Acute Appendicitis

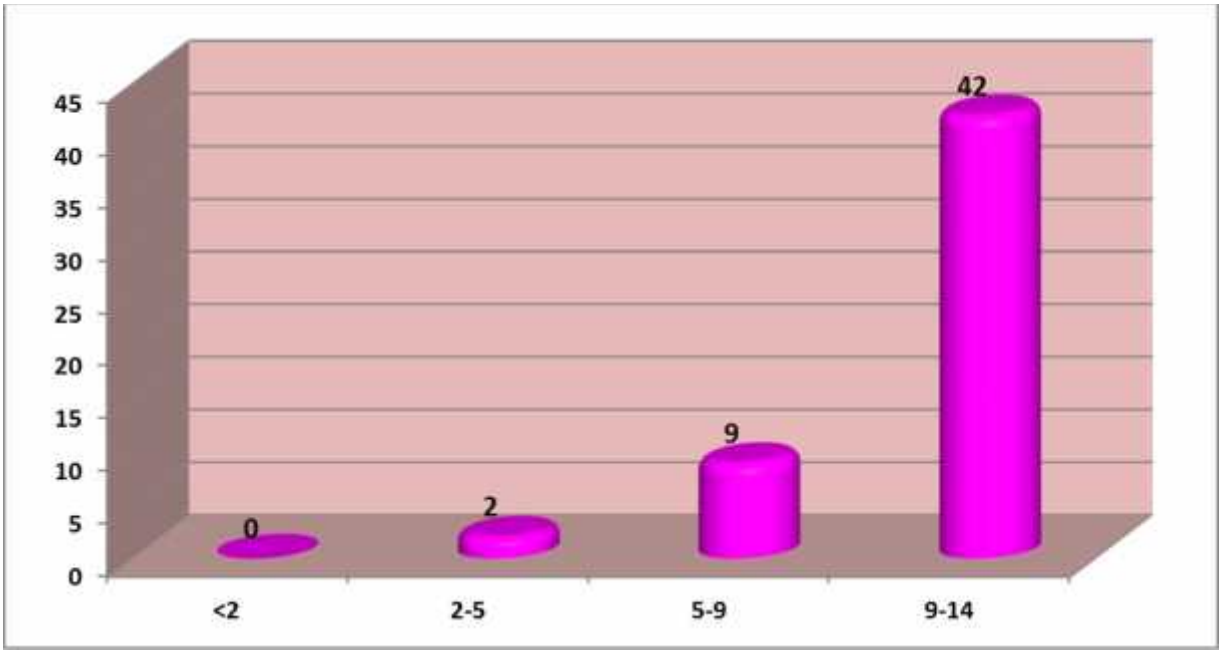
The pathophysiology of appendicitis in children is different from that of adults because of the changing anatomic location and susceptibility of the appendix throughout childhood.

Appendicitis is infrequent in infancy because of their funnel-shaped appendix, soft diet, recumbent posture, and infrequent gastrointestinal and upper respiratory infections. This is consistent with our study as we have not reported acute appendicitis in children less than 2 years of age.

After 2 years of age, the appendix becomes more susceptible to appendicitis as it assumes the adult shape. With increasing age of the child lymphoid follicle hyperplasia and size of the appendix gradually increase and peak in adolescence, which represents the highest incidence of appendicitis. [58]

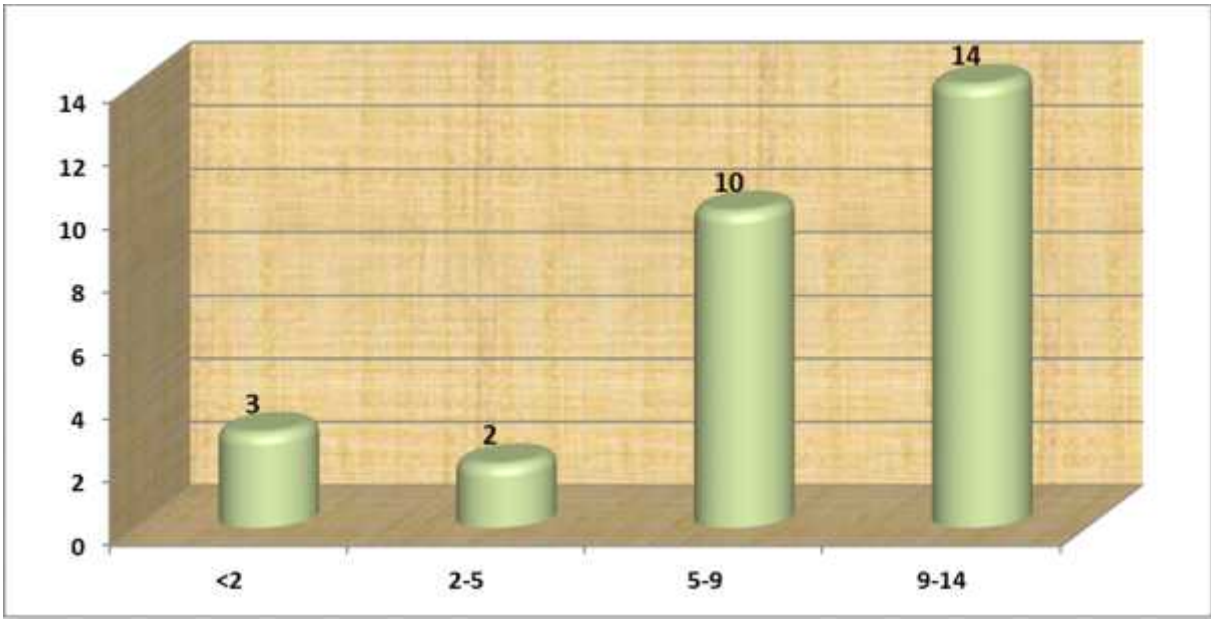
Likewise in our study the highest incidence of acute appendicitis was seen in children between 9 and 14 years. Our findings concur with those of Pujari AA et al [27] conducted at Nagpur Indira Gandhi medical College .In their study the largest group was acute appendicitis (58%) which is very similar to our results. This further reiterates the referral patterns of acute abdomen cases to teaching institutes and medical colleges in comparison with data from private clinics and practitioners.

Graph 17: Age wise distribution of Acute Appendicitis



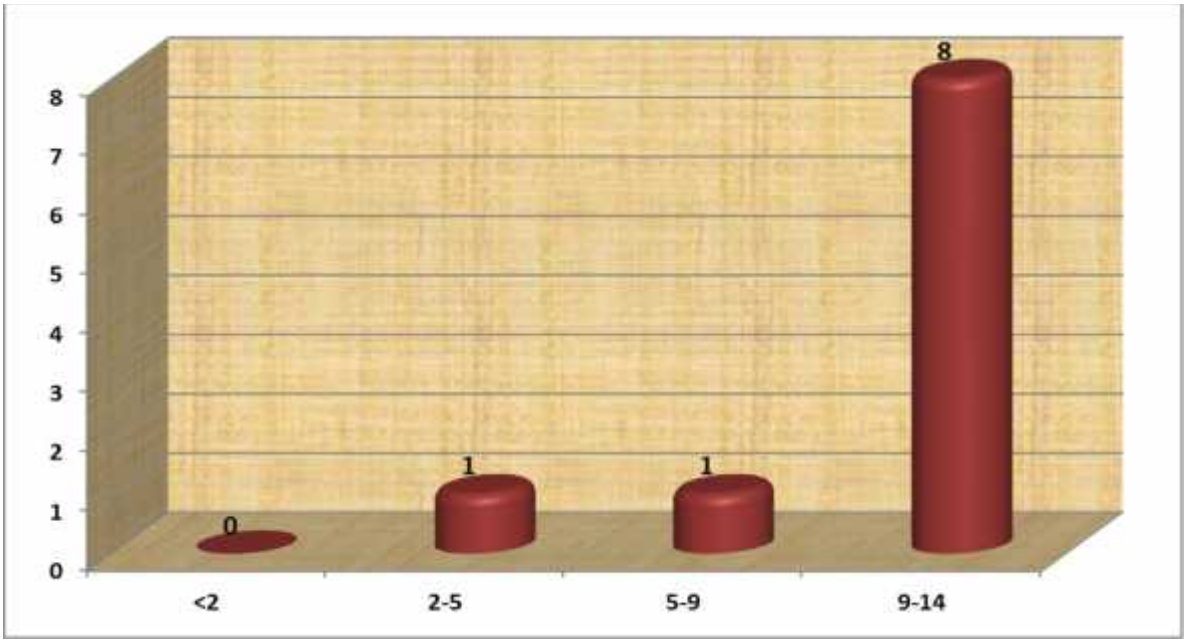
In our study the highest incidence of uncomplicated acute appendicitis was seen between 9 and 14 years of age. The mean age at diagnosis was 11.3 years. There was a slight male preponderance (n=29) compared to females (n = 24). All children with acute appendicitis were treated with either Laparoscopic or open Appendicectomy.

Graph 18: Age wise distribution of Non-Specific Abdominal Pain



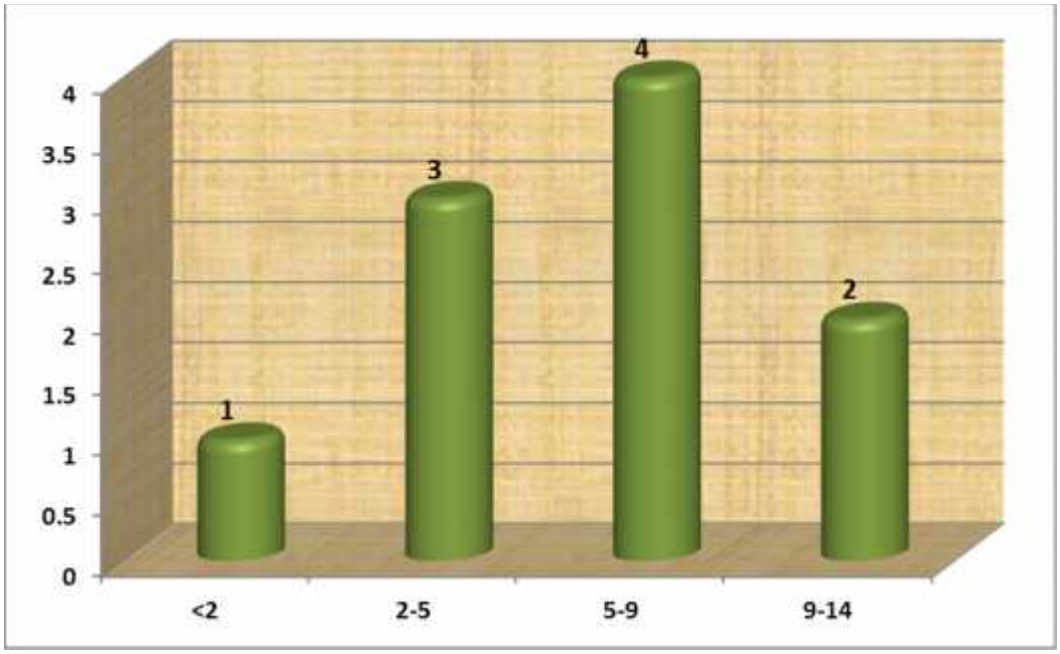
Non-specific abdominal pain was the most common cause of pain abdomen managed conservatively without any operative intervention in all the age groups. The highest incidence was seen between 9 and 14 years of age and was almost twice as more common in the girl child. F: M (1.9:1). These findings concur with the study by Holland et al [40]. In their study a total of Fifty-four per cent (n = 714) of the patients were discharged without surgical intervention; in this group the most frequent (70%, n = 503) diagnosis was non-specific abdominal pain (NSAP). Children with NSAP were treated conservatively. Mean age of presentation was 8.8 years.

Graph 19: Age wise distribution of Mesenteric Lymphadenitis



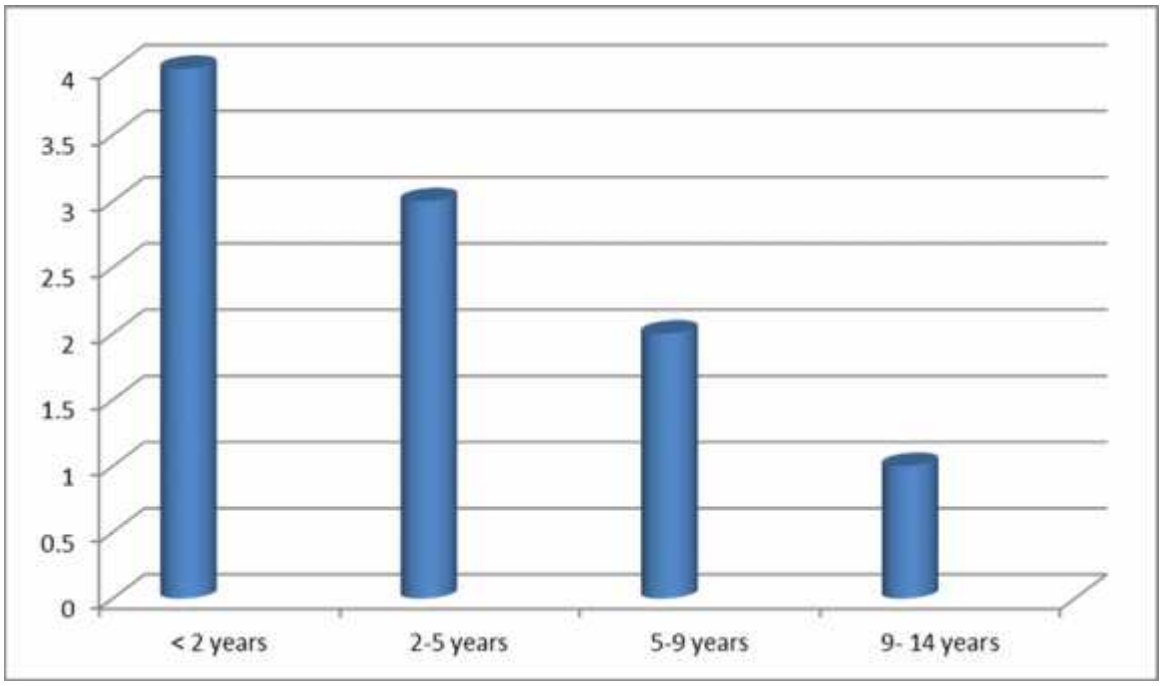
Mesenteric Lymphadenitis was more common among female children (n= 6) compared to males (n = 4). The mean age of presentation was 11.3 years similar to that of appendicitis. The mean age of presentation of mesenteric lymphadenitis and acute appendicitis is same, probably indicative of a common underlying pathogenesis. While most authors favour a conservative management for mesenteric lymphadenitis with analgesics and antibiotics some advocate a diagnostic laparoscopy and proceed with Appendicectomy if the appendix appears to be inflamed. Recurrent and catarrhal appendicitis are frequently associated with mesenteric lymphadenopathy.

Graph 20: Age wise distribution of Urolithiasis



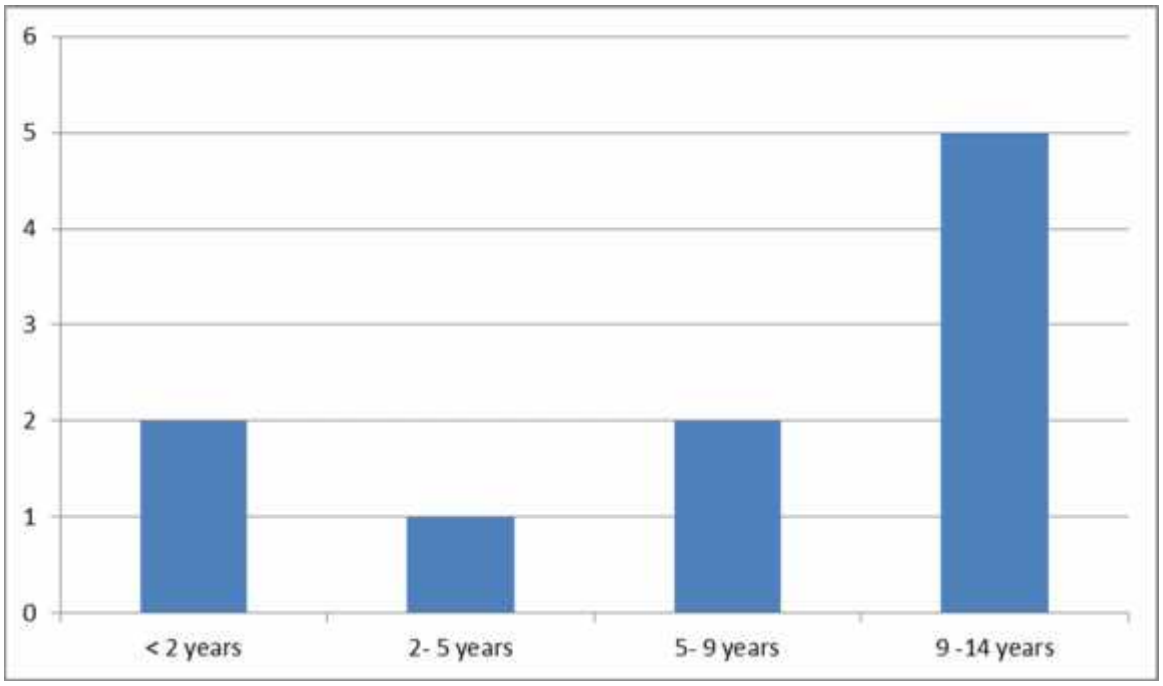
In our study a total of 10 children were diagnosed with urolithiasis and required urological intervention. Five children had vesical calculi and all five of them were boys. The remaining five were diagnosed with right renal and ureteric calculi. The Male to female ratio for urolithiasis in children is 9: 1 in our study. This aetiology is unique to our study and further large scale studies are required to estimate the true incidence of urolithiasis in Bijapur and the incidence in children in comparison with adults.

Graph 21: Age wise distribution of Intestinal Obstruction



Intestinal Obstruction was the commonest aetiology of acute abdomen in children less than 2 years of age. 70% of intestinal obstruction occurred in children less than 5 years of age. The commonest cause of obstruction was intussusception. All children underwent exploratory laparotomy. One child with gangrenous bowel underwent resection anastomosis. These findings concur with studies by Pujari AA et al who reported that intussusception was the most common aetiology for intestinal obstruction, accounting for 7% cases of acute abdominal emergencies in their study [27]. The mean age of presentation of intestinal obstruction in our study was 4.35 years.

Graph 22: Age wise distribution of generalized peritonitis



A total of 10 children (8%) presented with generalized peritonitis. On exploratory laparotomy 6 out of these 10 were noted to have appendicular perforation. The incidence of appendicular perforation is about 4.5% of all cases of acute abdomen in children and accounts for 60% cases of generalized peritonitis. Ileal perforation, choledochal cyst perforation and pelvic abscess were other causes of peritonitis. Perforation peritonitis accounted for 7% of the cases in the study by Pujari et al[27]. Our findings are consistent with their study.

Other Aetiologies.

Two children were diagnosed to have Meckel's diverticulitis and underwent laparotomy and wedge resection of the diverticulum. One child had ovarian torsion and underwent oophorectomy for the same. It has been reported to account for up to 2.7% of all cases of paediatric abdominal pain.[1] Two children had acute pancreatitis one was managed conservatively while other child underwent necrosectomy and drainage of the haemorrhagic pancreatic cyst. Two Children had Obstructed Hernia one umbilical and one Inguinal, which were the cause for acute abdominal pain. We also noted two cases of Acute Cholecystitis. One child had omental torsion in addition to acute appendicitis and underwent partial omentectomy.

These less frequent causes of abdominal pain must be remembered and kept in mind while evaluating a child with abdominal pain. In comparison with other studies [26, 27, 40] in our study we did not note any case of primary peritonitis, torsion testis or gastric volvulus. These diagnoses must also be borne in mind.

Investigations

The most helpful investigations which are cost effective, convenient and provide a high yield in diagnosis include a CBC with leucocytosis and neutrophilia indicating a need for operative intervention. USG abdomen had a specificity of 100% and a sensitivity of 86.67% in diagnosing acute abdomen. However it must be remembered that USG is operator dependant. Erect X ray was useful in detecting both obstruction and urolithiasis. These are simple and cost effective tools to confirm our diagnosis. In our study we did not routinely resort to the use of CT Abdomen and other higher investigations. In our study a thorough clinical evaluation and the above simple investigations helped confirm the diagnosis in more than 90 % of the cases.

Duration of Stay

77.9% of children had a hospital stay between 2 to 8 days. Those with NSAP and Mesenteric Lymphadenitis had shorter hospital stay. Children who underwent Laparoscopic Appendicectomy had shorter stay compared to those undergoing open Appendicectomy. Children who underwent exploratory laparotomies had longer duration of stay with a mean of 11 days. Children with acute pancreatitis had an even longer mean duration of stay 23 days.

SUMMARY

The clinical spectrum of 131 children, less than 14 years of age, who presented with symptoms and signs of acute abdomen, was studied. There were different aetiologies in different age groups. The following observations were made.

- 69% of children with acute abdomen required operative management.
- 31% of children were managed conservatively.
- Paediatric acute abdomen is more common in the male child
- The most common age group affected is children between 9 and 14 years
- Acute Appendicitis 40% is the most common surgical emergency.
- Non-Specific Abdominal Pain 29% is the second most common cause of acute abdomen.
- Generalized Peritonitis, Intestinal obstruction, Urolithiasis and Mesenteric Lymphadenitis each account for 8 % of all paediatric acute abdomen.
- Non Specific Abdominal Pain and Mesenteric Lymphadenitis are more common in female children.
- Urolithiasis is more common in male children.
- Appendicular perforation 4.5% is the commonest cause of generalized peritonitis in children.
- Intussusception 3.8% is the most common cause of intestinal obstruction in children.
- Acute Pancreatitis, Acute Cholecystitis, Meckel's diverticulitis, Obstructed Hernia and Ovarian torsion can also occur in children and must be remembered as significant causes of acute abdomen.

- Leucocytosis and Neutrophilia are the most easily available markers of need for surgical intervention when viewed in conjunction with a thorough clinical examination.
- USG and Erect X Ray abdomen are cost effective, non-invasive methods to confirm the cause of paediatric acute abdomen.
- Ultrasonography has 100% specificity and 86.67% sensitivity in diagnosing acute abdomen.
- There was no mortality in our study.

CONCLUSION

Children are a special subgroup of population in whom the clinical symptoms and signs of acute abdomen are subtle. Repeated clinical examination continues to be the cornerstone in diagnosing and managing the child with acute abdomen. A complete blood profile, urine routine, Erect X ray abdomen and Ultrasonography of the abdomen are adjunctive investigations helpful in diagnosing many conditions and also help rule out organic pathology.

Regardless of the aetiology of paediatric acute abdomen, there is a twofold need for a compassionate and calm approach. The first aim is to rapidly identify the surgical causes of acute abdomen and institute appropriate management. Second and equally important is to reduce parental anxiety. A well counselled and relaxed parent can provide more valuable history and thus help in the management of Paediatric Acute Abdomen.

Non Specific Abdominal pain and Mesenteric Lymphadenitis were common conditions in children and were successfully managed with antibiotics and analgesics. Acute appendicitis and Appendicular perforations were the most common surgical emergencies in children of the older age while intestinal obstruction due to intussusception or bands was common in the younger child. We also noted that urolithiasis is common among the children of Bijapur and is an important cause of paediatric acute abdomen.

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ANNEXURE-I

Institutional Ethical Committee
BIJAPUR-586 103.
OUTWARD
No. 0466/12
Date 18/10/12
BLDE U's Shri B.M. Patil Medical College

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 18-10-12 *at* 3-30pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title Paediatric acute abdomen
a prospective study
—X—X—X—

Name of P.G. student Dr Bharat S.
Surgery

Name of Guide/Co-investigator Dr B.M. Nyamannavar
Associate prof Surgery

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

ANNEXURE-II

CONSENT FORM

TITLE OF THE PROJECT	:	PAEDIATRIC ACUTE ABDOMEN – A PROSPECTIVE STUDY
GUIDE	**:**	Dr. B.M.NYAMANNAWAR
P.G. STUDENT	**:**	Dr. BHARAT. S.

PURPOSE OF RESEARCH:

I have been informed that this study is conducted to identify the causes of acute abdominal pain in children. My child has also been given free choice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received my child will be asked series of questions by the investigator. My child may be asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

RISK AND DISCOMFORTS

I understand that my child may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS

I understand that my child's participation in the study will help to identify the causes of acute abdomen in the paediatric population.

CONFIDENTIALITY

I understand that the medical information produced by this study will become a part of hospital records and will be subject to confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION

I understand that I may ask more questions about the study at anytime to Dr. Bharat S at the department of general surgery who will be available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION

I understand that my child's participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. Bharat S may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability.

_____ _____

Dr. BHARAT S Date

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that **Dr. BHARAT. S** has explained to me the purpose of research, the study procedure, that my child will undergo and the possible discomforts as well as benefits that my child may experience in my own language. I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give consent for my child to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to signature) Date

ANNEXURE-III

CASE PROFORMA

1) Informant	:	CASE NO	:
2) Name	:	IP NO	:
3) Age/sex	:	DOA	:
4) Religion		DOS	:
5) Residence	:	DOD	:

6) Chief Complaints :

Site of pain At onset

At present

Radiation

Aggravating Factors

Movement/ coughing/ respiration/ food/ other / none

Relieving factors

Lying still/ vomiting / antacids/ food/ other/ none

Progression of pain

Better/ same / worse

Duration of pain

Type of pain Intermittent / steady / colicky

Associated symptoms

Nausea	y/n	Vomiting	y/n	Anorexia	y/n
Indigestion	y/n	Jaundice	y/n		

Bowels Normal/Constipation/ Diarrhoea /Blood/Mucus

Micturition Normal/Frequency/Dysuria/Dark/Haematuria

7) Past History - Number of similar attacks in the past

8) Treatment history - Any surgery/ Systemic illness

9) Personal History - Diet/ Appetite /Sleep/ Habits

10) Family History -

11) General Physical Examination –

Vitals: Pulse Rate/ Temperature/ Blood Pressure/Respiratory rate

Built and Nourishment/ Weight

Pallor/Icterus/Cyanosis/Clubbing/Pedal Edema/Generalized lymphadenopathy

12) Per Abdomen examination.

Distension : yes / no

Abdominal Girth :

Tenderness / Rebound tenderness

Guarding /Rigidity

Mass /Intestinal movements /Bowel sounds

Rectal / vaginal tenderness

13) Other systemic examination

- Respiratory system.
- Cardiovascular system.
- Central nervous system.

14) INVESTIGATIONS:

- Complete blood count
- Urine routine
- Erect x ray
- USG
- Other Investigations

15) Final Diagnosis

16) Surgical intervention

17) Intraoperative findings

18) Post-operative status

19) Histopathological findings

20) Condition at discharge

21) Total duration of post-operative stay

22) Time duration between diagnosis and surgical intervention

KEY TO MASTER CHART

Ac.Ap	Acute Appendicitis
Ac.Chol	Acute Cholecystitis
Ac.Pan	Acute Pancreatitis
Adl	Adhesiolysis
AFL	Air Fluid Level
Ap	Appendicular
B/L	Bilateral
Cal	Calculus
CC	Choledochal Cyst
CJ	Choledochojejunostomy
CONS	Conservative
D	Days
DJ	DudenoJejunostomy
DJS	Double J Stenting
DLC (N)	Differential Leucocyte Count (Neutrophils)
DoH	Duration of Hospital
E.X Ray	Erect X Ray Abdomen
Exp Lap	Exploratory Laparotomy
F	Female
GP	Generalized Peritonitis
Int	Intussusception
IO	Intestinal Obstruction
IP No	In Patient Number
Lap A	Laparoscopic Appendicectomy
M	Male

MD	Meckel's Diverticulitis
MGV	Midgut Volvulus
ML	Mesenteric Lymphadenitis
N	Normal
NED	Necrosectomy with External Drainage
NSAP	Non- Specific Abdominal Pain
O A	Open Appendicectomy
O Cy	Open Cystolithotomy
OIH	Obstructed Inguinal Hernia
Oop	Oophorectomy
OR	Operative Reduction
OUH	Obstructed Umbilical Hernia
PCNL	Per Cutaneous Nephro Lithotomy
PL	Peritoneal Lavage
PO	Partial Omentectomy
PP	Pre Pyloric
PR	Primary Repair
PUJ	Pelvi Ureteric Junction
R	Right
RA	Resection Anastomosis
SMA	Superior Mesenteric Artery Syndrome
T.L.C	Total Leucocyte Count
URS	Uretero Reno Scopy
USG	Ultrasonography
V Cal	Vesical Calculus
WR	Wedge Resection

MASTER CHART

Sl.No	Name	Age	Sex	IP No	T.L.C	DLC (N)	E.X Ray	USG	DoH Stay	Diagnosis	Treatment
1	Abhilash	13	M	19321/13	12500	88	N	Accurate	7 D	Ac.Ap	O A
2	Abhishek	14	M	16426/14	11700	92	N	Accurate	5 D	Ac.Ap + Omental Torsion	Lap A+ PO
3	Abhishek	11	M	16059/14	15250	83	Localized Ileus	Accurate	10D	Ac.Ap + Ap Mass	CONS
4	Abishek M Yendigiri	5	M	13605/14	10500	78	R Renal cal	Accurate	4 D	R PUJ Cal	R URS+ DJS
5	Aditya	2	M	19094/13	8800	72	N	Accurate	6 D	GP-Ap Perforation	Exp Lap + A + PL
6	Aduba	5	F	6698/14	5600	42	N	N	3 D	NSAP	CONS
7	Ahmed Kalar	1	M	3118/14	16800	76	N	Accurate	8 D	IO-Int	Exp Lap + OR
8	Aisha	11 Months	F	18961/13	20000	46	N	Inaccurate	18 D	R Ovarian Torsion	Exp Lap + Oop
9	Aishwarya	12	F	29140/12	13000	72	N	Accurate	6 D	Ac.Ap	O A
10	Akash	13	M	22830/14	4830	70	N	Accurate	4 D	Ac.Ap	O A
11	Akkamma Ashok	12	F	5902/14	6825	65	N	N	3 D	NSAP	CONS
12	Akshata	14	F	17363/13	9600	48	N	Accurate	4 D	ML	CONS
13	Akshatha Shantappa	13	F	4259/14	11400	53	B/L Renal Cal	Accurate	6 D	R Renal Cal	R URS+ DJS
14	Amit Nanasaheb	10	M	1686/14	9790	62	N	Accurate	3 D	ML	CONS
15	Anil Ramesh Chavan	3	M	21993/12	13000	50	V cal	Accurate	11 D	V Cal	O Cy
16	Apporva	9	F	20740/14	5600	65	N	N	5 D	NSAP	CONS
17	Arabaj	11	M	7907/14	12300	88	N	Accurate	6 D	Ac.Ap	O A
18	Arsubai	11	F	14362/13	11500	96	N	Accurate	5 D	Ac.Ap	O A
19	Arun	7	M	11264/14	8050	56	N	N	2 D	NSAP	CONS
20	Arun Kumar	10	M	28398/12	10250	52	N	Accurate	9 D	Ac.Ap	O A
21	Aruna	7	F	13513/14	9800	49	N	Accurate	3 D	ML	CONS
22	Asif Attar	12	M	22309/12	10100	54	N	Accurate	7 D	Ac.Ap	O A
23	Bablu Gouri	9	M	19355/13	8100	91	AFL	Inaccurate	14 D	IO-Bands	Exp Lap + Adl
24	Balangouda	14	M	8382/12	8400	52	N	Accurate	3 D	ML	CONS
25	Basavaraj	3	M	7751/13	13000	74	AFL	Inaccurate	12 D	MD	WR
26	Basavaraj	5	M	17841/13	11500	82	N	Accurate	3 D	Ac.Ap	O A
27	Bhagyalakshmi	7	F	6980/14	6500	45	N	N	3 D	NSAP	CONS
28	Bhagyashree	9	F	3512/13	12800	66	N	Accurate	6 D	Ac.Ap	O A
29	Bhagyashree	8	F	7494/14	5500	32	N	N	8 D	NSAP	CONS
30	Bhagyashree	11	F	13340/14	5020	88	N	N	3 D	NSAP	CONS
31	Bharathi	10	F	2463/14	14250	89	N	Accurate	5 D	Ac.Ap	O A
32	Bhimanna	8	M	11619/14	12560	72	V Cal	Accurate	3 D	V Cal	O Cy

33	Bhuvaneshwari	12	F	21727/14	10950	76	N	Inaccurate	5 D	Ac.Ap	Lap A
34	Danemma	3 Months	F	1093/14	9630	56	N	N	3 D	NSAP	CONS
35	Darshan	2	M	22941/13	11500	90	AFL	Accurate	11 D	IO-Bands	Exp Lap + Adl
36	Darshan	8 Months	M	13789/14	13540	80	AFL	Accurate	5 D	IO-Int	Exp Lap + OR
37	Dundappa	9	M	44/14	10900	67	V Cal	Accurate	3 D	V Cal	Lithotripsy
38	Gouramma	11	M	26606/12	11900	90	N	Inaccurate	6 D	Ac.Ap	Lap A
39	Jyothi	12	F	17780/13	18500	92	N	Inaccurate	4 D	Ac.Ap	O A
40	Jyothi	13	F	8410/14	4680	44	N	N	6 D	NSAP	CONS
41	Kamaleshi	8	F	4110/14	8750	46	N	N	2 D	NSAP	CONS
42	Karthik Rathod	3	M	1044/14	7780	70	V Cal	Accurate	6 D	V Cal	O Cy
43	Kavitha Jadhav	14	F	16298/13	7354	66	N	Accurate	5 D	Ac.Ap	O A
44	Kiran	3 Months	M	2804/14	8900	85	N	N	2 D	NSAP	CONS
45	Lakkanna Sanganna	10	M	19136/12	8900	43	N	Accurate	7 D	Ac.Ap	O A
46	Lakshmi	11	F	16159/14	11630	87	N	Accurate	5 D	Ac.Ap	Lap A
47	LAKSHMI	14	F	2753/14	17420	77	N	Accurate	6 D	Ac.Ap	O A
48	Lakshmi	10	F	23558/14	8400	70	N	N	5 D	NSAP	CONS
49	Mahantappa	12	M	5302/12	11670	87	N	Accurate	5 D	Ac.Ap	O A
50	Malliksab	9	M	20381/14	12300	92	R Renal Cal	Accurate	7 D	R R Cal	R PCNL
51	Manju Waddar	8	M	5025/13	11500	73	N	Accurate	6 D	Ac.Ap	O A
52	Manjunath	3	M	20958/12	11700	77	AFL	Accurate	18 D	IO-Int	Exp Lap + RA
53	MANJUNATH	14	M	2862/14	9810	37	N	Accurate	5 D	Ac.Ap	O A
54	Manoj	14	M	5594/13	11580	81	N	Accurate	7 D	Ac.Ap	O A
55	Megha	5	F	10404/14	5670	56	N	N	3 D	NSAP	CONS
56	Mohahamadshashida	10	M	13002/14	4560	45	N	N	2 D	NSAP	CONS
57	Mohamad Faizan	3	M	18844/12	9000	34	N	Accurate	10 D	M D	WR
58	Moshin	3	M	8624/14	28000	78	AFL	Inaccurate	22 D	IO - MGV	Exp Lap + RA
59	Moulasab Narabenchi	12	M	15895/12	22000	84	N	Accurate	11 D	GP-Pelvic Abscess	Exp Lap + PL
60	Mubarath	1.5	M	12824/14	19000	76	AFL	Accurate	10 D	IO-Bands	Exp Lap + Adl
61	Neelamma Walikar	9	F	20339/12	6900	60	AFL	Inaccurate	19 D	IO-SMA	Exp Lap + Adl + DJ
62	Ninganna	12	M	29003/12	19900	90	N	Accurate	8 D	Ac.Ap	O A
63	Nivedita	11	F	21896/14	6500	68	N	N	3 D	NSAP	CONS
64	PALLAVI	6	F	12125/13	11800	74	N	Accurate	6 D	Ac.Ap	O A
65	Pallavi	12	F	21456/14	10560	88	N	Accurate	5 D	Ac.Ap	Lap A
66	Panchami	13	F	1205/13	9840	52	N	Accurate	3 D	ML	CONS

67	Pavithra	9	F	19824/14	19560	96	N	Accurate	6 D	Ac.Ap	Lap A
68	Poornima	8	F	3981/14	16330	77	N	Accurate	11 D	Ac.Chol	O C
69	Prashanth	11	M	21051/13	13564	76	N	Accurate	10 D	Ac.Ap	O A
70	Prashanth Panidraswami	12	M	25736/12	11000	72	AFL	Accurate	8 D	Ac.Ap + Ap Abscess	Exp Lap + A + PL
71	Priyanka Masali	14	F	10558/14	16000	54	N	Accurate	8 D	Ac.Ap	Lap A
72	Radhika	9	F	27130/13	8450	55	N	Accurate	6 D	Ac.Ap	O A
73	Raghavendra	13	M	6998/13	7860	78	N	Accurate	5 D	ML	CONS
74	Rajashri	2	F	8971/14	11560	77	N	Accurate	5 D	OUH	Herniorraphy
75	RAVI	13	M	5547/14	12630	76	N	Accurate	5 D	Ac.Ap	O A
76	Ravi Mareppa	10	M	8705/12	7300	76	N	Accurate	11 D	Ac.Chol	OC
77	Ravi Navi	13	M	20031/12	8400	57	N	Accurate	6 D	Ac.Ap	O A
78	Rekha	10	F	19333/14	7420	62	N	N	3 D	NSAP	CONS
79	Riya	9	F	3481/14	17250	88	N	Accurate	6 D	Ac.Ap	O A
80	Roshab	6 Months	M	5607/14	5690	54	N	N	5 D	NSAP	CONS
81	Sachin	2	M	12613/14	14000	58	V Cal	Accurate	5 D	V Cal	PCCL
82	Sadhashiv	14	M	22873/14	10235	60	N	N	3 D	NSAP	CONS
83	Sagar	14	M	20963/14	4560	77	N	N	2 D	NSAP	CONS
84	Sagar M Salukeri	12	M	24016/12	10300	78	N	Accurate	6 D	Ac.Ap	O A
85	Saheblal	12	M	1011/14	9750	62	N	N	3 D	NSAP	CONS
86	Sakshi	6	F	24262/14	5600	47	N	N	2 D	NSAP	CONS
87	Sandya	11	F	7109/14	8425	60	N	N	2 D	NSAP	CONS
88	Santosh	8	M	25154/12	15600	58	AFL	Inaccurate	11 D	GP-Ileal Perforation	Exp Lap + PR + PL
89	Santosh	13	M	11561/14	9700	58	R Renal Cal	Accurate	6 D	R PUJ Cal	Right Pyeloplasty
90	Savidha	4	F	13367/14	7500	78	N	Accurate	3 D	ML	CONS
91	Savitha	13	F	11933/13	6850	77	N	Accurate	5 D	ML	CONS
92	Shabana	13	F	12610/14	11500	45	N	Accurate	4 D	Ac.Ap	Lap A
93	Sharat Rathod	7	M	5368/14	8900	70	R Renal Cal	Accurate	3 D	R R Cal	R URS + DJS
94	Shilpa	12	F	5065/14	8700	75	N	N	3 D	NSAP	CONS
95	Shivasharan	12	M	19868/12	12560	80	N	Accurate	6 D	Ac.Ap	O A
96	Shivayya Shrishail	13	M	5166/14	9640	71	N	Accurate	5 D	ML	CONS
97	Shobha	6	F	20639/12	12500	57	N	Accurate	5 D	Ac.Ap	O A
98	Shreedevi	3	F	15560/14	13470	82	AFL	Accurate	6 D	IO-Int	Exp Lap + OR
99	Shrishail	14	M	30422/12	11000	75	N	Accurate	6 D	Ac.Ap	O A
100	Shubam	5	M	18977/13	8750	65	N	Accurate	8 D	OIH	Herniorraphy

101	Shubam H	1	M	18640/12	25450	90	AFL	Inaccurate	21 D	GP-CC Perforation	Exp Lap + CJ
102	Sidamma Irrappa	10	F	16329/12	17600	73	AFL	Accurate	9 D	GP-Ap Perforation	Exp Lap + A + PL
103	Siddamma	9	F	22245/12	11200	78	AFL	Accurate	8 D	Ac.Ap	O A
104	Siddappa	2	M	23025/13	16270	40	AFL	Inaccurate	26 D	Ac.Pan	Exp Lap + NED
105	Siddu Pujari	13	M	23994/13	14800	95	AFL	Accurate	13 D	GP-Ap Perforation	Exp Lap + A + PL
106	Sneha Mallikarjun	3	F	21225/12	11800	75	AFL	Accurate	6 D	Ac.Ap	O A
107	Soniya	14	F	30232/12	14600	85	N	Accurate	3 D	Ac.Ap	Lap A
108	Soubhagya	13	F	4497/14	10560	88	N	Accurate	6 D	Ac.Ap	O A
109	Soumya	10	F	18774/14	8750	50	N	N	2 D	NSAP	CONS
110	Spurthi	8	F	5472/13	16500	65	N	Inaccurate	11 D	Ac.Ap	O A
111	Suchitra	10	F	8462/14	9870	90	N	Accurate	5 D	Ac.Ap	O A
112	Sudeep	12	M	10993/13	15600	72	N	Accurate	8 D	Ac.Ap	O A
113	Sunil Bijapur	11	M	30464/13	17850	78	N	Accurate	8 D	Ac.Ap	O A
114	Suresh	13	M	6369/13	12680	76	N	Accurate	5 D	Ac.Ap	O A
115	SUSHMA	14	F	13402/13	13470	74	N	Accurate	6 D	Ac.Ap	O A
116	Thanushri	9	F	5982/14	5740	68	N	N	2 D	NSAP	CONS
117	TOPANNA	13	M	2274/13	11700	77	N	Accurate	4 D	Ac.Ap	O A
118	Ujawala	9	F	14517/14	4890	47	N	N	5 D	NSAP	CONS
119	Varun Lakshman	14	M	13951/14	15000	42	AFL	Accurate	10 D	Ac.Pan	CONS
120	Vedhant	9	M	16622/14	7800	62	N	N	4 D	NSAP	CONS
121	VEERESH	12	M	9442/14	16300	82	N	Accurate	5 D	Ac.Ap	O A
122	Vijay Msk	11	M	22718/14	12850	88	AFL	Accurate	11 D	IO-Int	Exp Lap + OR
123	Vijay Singh	7	M	1280/13	11400	73	LI	Accurate	13 D	GP-Ap Perforation	Exp Lap + A + PL
124	Vijaya Somanath Chavan	12	M	4474/14	26500	92	AFL	Accurate	13 D	GP-Ap Perforation	Exp Lap + A + PL
125	Vijayakka	12	F	7957/12	14700	45	N	Accurate	2 D	ML	CONS
126	Vikas	14	M	15920/14	6500	70	N	Accurate	2 D	NSAP	CONS
127	Vinay	8	M	3496/14	4088	60	N	Accurate	3 D	NSAP	CONS
128	Vinod	14	M	10004/14	14550	82	N	Accurate	4 D	Ac.Ap	Lap A
129	Vishwanath Iranna	5	M	21561/12	16900	96	AFL	Accurate	10 D	GP-Ap Perforation	Exp Lap + A + PL
130	Vivek	14	M	10004/14	11450	58	N	Accurate	4 D	Ac.Ap	Lap A
131	Yalappa	14	M	23356/13	18960	89	AFL	Accurate	16 D	GP-PP Perforation	Exp Lap + PR + PL