"EVALUATION OF C-REACTIVE PROTEIN LEVEL AND TOTAL LEUCOCYTE COUNT IN ACUTE APPENDICITIS"

By

Dr.PRASHANT.K.DHANNUR

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SURGERY

IN

GENERAL SURGERY

Under the guidance of

Dr. P.N. SREERAMULU
Professor

DEPARTMENT OF GENERAL SURGERY,
SRI DEVARAJ URS MEDICAL COLLEGE & RESEARCH,
TAMAKA, KOLAR-563101

APRIL 2016

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation/thesis entitled **"EVALUATION OF C-REACTIVE PROTEIN LEVEL AND TOTAL LEUCOCYTE COUNT IN ACUTE APPENDICITIS"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.P.N.SREERAMULU** Professor, Department of General Surgery, Sri Devaraj Urs Medical College, Tamaka, Kolar.

Date:

Place: Kolar **Dr.PRASHANT K. DHANNUR**

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"EVALUATION OF C-REACTIVE PROTEIN LEVEL AND TOTAL LEUCOCYTE COUNT IN ACUTE APPENDICITIS"** is a bonafide research work done by **Dr.PRASHANT.K.DHANNUR** in partial fulfillment of the requirement for the Degree of MASTER OF SURGERY in **GENERAL SURGERY.**

Date:

Place: Kolar

SIGNATURE OF THE GUIDE

Dr.P.N.SREERAMULU,

Professor,

Department Of General Surgery,

Sri Devaraj Urs Medical College

Tamaka, Kolar.

ENDORSEMENT BY THE HOD,

PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled ***"EVALUATION OF C-REACTIVE PROTEIN LEVEL AND TOTAL LEUCOCYTE COUNT IN ACUTE APPENDICITIS"*** *is a bonafide research work done by* ***Dr.PRASHANT .K. DHANNUR*** *under the guidance of* ***Dr. P.N.SREERAMULU****, Professor, Department Of General Surgery.*

Dr.K MOHAN KUMAR MS
Professor & HOD,
Department of General Surgery,
Sri Devaraj Urs Medical College,
Tamaka, Kolar

Date:
Place: Kolar

Dr. B.G RANGANANTH MD
Principal,
Sri Devaraj Urs Medical College,
Tamaka, Kolar

Date:
Place: Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTER, TAMAKA, KOLAR, KARNATAKA

ETHICS COMMITTEE CERTIFICATE

This is to certify that the Ethics committee of Sri Devaraj Urs Medical College & Research Center, Tamaka, Kolar has unanimously approved

Dr.PRASHANT.K.DHANNUR

Post-Graduate student in the subject of

GENERAL SURGERY *at Sri Devaraj Urs Medical College, Kolar*

to take up the Dissertation work entitled

"EVALUATION OF C-REACTIVE PROTEIN LEVEL AND TOTAL LEUCOCYTE COUNT IN ACUTE APPENDICITIS"

to be submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA,

Date:

Place: Kolar

Member Secretary

Sri Devaraj Urs Medical College &

Tamaka, Kolar–563101

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

COPY RIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka shall have the rights to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purpose.

Date:

Place: Kolar **Dr. PRASHANT.K.DHANNUR**

ACKNOWLEDGEMENT

I am highly indebted to my guide ***Dr. P. N. SREERAMULU,*** *M.S., Professor, Department of General Surgery, Sri Devaraj Urs Medical College, Tamaka, Kolar, who guided me in bringing out this work with his thought provoking ideas and constant encouragement.*

It gives me immense pleasure to express my gratitude and sincere thanks to ***Dr.K.MOHANKUMAR,*** *M.S., Professor and H.O.D., Department of General Surgery, Sri Devaraj Urs Medical College, Tamaka, Kolar, who took deep interest and gave constant support by encouraging in moulding this work.*

I also acknowledge my debt to ***Dr. A. BHASKARAN****, M.S.,* ***Dr. K. KRISHNA PRASAD,*** *D.N.B,* ***DR.SHASHIREKHA.C.A*** *M.S, Department of General Surgery, Sri Devaraj Urs Medical College, Tamaka, Kolar, who gave me moral support and guidance by correcting me at every step.*

I express my sincere thanks to all my teachers and Professors of Department of General Surgery, Sri Devaraj Urs Medical College, Tamaka, Kolar.

I remain thankful to all my assistant professors for their support and encouragement. My special thanks to ***Dr.Srinivas, Dr.Girish Gowda, Dr.Rakesh*** *for their timely support.*

My heartful thanks to ***Dr.Sunil Bage, DrPreeti and Dr.Shilpa Ciryam*** *for their invaluable support in completing my dissertation work.*

I acknowledge my sincere thanks to all my co-P.G.'s ***Dr.Vivekananda, Dr.Naveen, Dr.Akarsh, Dr.Chandan, Dr.Munireddy*** *and all my juniors for their unconditional help and support at every step throughout my study.*

I am much thankful to my parents ***Dr.K.B.DHANNUR*** *and* ***Mrs. KASTHURI*** *for their love, blessings and invaluable help. I thank all my sisters,* ***Dr.GEETHA.P, Dr.LATHA, Mrs.SHEELA.V*** *for believing in me when I found it difficult to believe in myself and his support.*

Last, but not the least, I thank the ***Almighty*** *and* ***my patients*** *for providing me the opportunity to carry out my study.*

Dr. PRASHANT.K.DHANNUR

LIST OF ABBREVIATIONS

CT	Computer Tomography
HPE	Histopathological Examination
PID	Pelvic Inflammatory Disease
RLQ	Right Lower Quadrant
WBC	White Blood Count
CRP	C-Reactive Protein
USG	Ultrasonography
AA	Acute Appendicitis
IA	Inflammed Appendix
PA	Perforated Appendix
GA	Gangrenous Appendix
AA	Acute Appendicitis
CA	Chronic Appendicitis
RA	Recurrent Appendicitis

ABSTRACT

BACKGROUND AND OBJECTIVES

Acute appendicitis is one of the most common surgical emergency and is usually a clinical diagnosis. Many patients may present with typical history and examination findings. However 30.0% of the cases have atypical clinical presentation and it remains a diagnostic dilemma.

Recently, C Reactive Protein (CRP) along total leukocyte count is considered as the one of the indictor of acute appendicitis. It is not disease specific but it offers valuable diagnostic information about the presence of acute infection with concomitant evaluation of patient history and physical examination.

Thus, this study is an attempt to evaluate the significance of total leukocyte count and CRP in diagnosing acute appendicitis where no other obvious diagnosis of concern is being considered

MATERIALS AND METHODS

This study was performed on 114 patients who have been clinically diagnosed to have Acute Appendicitis and who were posted for emergency appendicectomy in General Surgery Department of RL Jalappa Hospital attached to Sri Devaraj Urs Medical College, Tamaka, Kolar during the period from January 2014 to June 2015. CRP, Total leucocyte count was done in all cases. After obtaining consent, patients were operated and the appendectomy specimen was sent for histopathological examination. The HP report was considered as the final diagnosis.

RESULTS: Out of 114 patients clinically diagnosed to have acute appendicitis, male predominance was seen with most common presenting age group of 21-30 years. Out of 67 subjects who had leukocytosis, 71.6% turned out to be acute appendicitis and out of 87 patients of acute appendicitis (confirmed by HPE), only 64 (73.56%) cases were positive for CRP. But both raised TLC and CRP was observed in 63 (55.26%) cases, which was extremely significant.

CONCLUSION: Serum CRP value when interpreted in combination with clinical findings and leucocyte count, was found to be significant in diagnosing acute appendicitis.

KEYWORDS: Acute appendicitis, C-reactive protein, Total leukocyte count

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

LIST OF GRAPHS

LIST OF PHOTOGRAPHS

INTRODUCTION

Acute appendicitis is one of the most common surgical emergency and is usually a clinical diagnosis. Many patients may present with typical history and examination findings. However 30.0% of the cases have atypical clinical presentation and it remains a diagnostic dilemma even for the senior surgeons , even in the presence of multiple diagnostic tool, and this can lead to its complications like appendicular abscess, appendicular perforation, peritonitis.[1,2,3]

Western literatures report that 6% of population have risk of suffering from appendicitis during their lifetime.[4] The overall mortality rate for appendicitis has decreased from about 26% to less than 1% because of antibiotics and early surgical intervention, but in elderly it is approximately 5 to 15%.The morbidity due to appendicular perforation ranges from 17% to 40%. The perforation rate is higher in both elderly and children.[5]

Diagnosis of acute appendicitis is established by surgeon's clinical impression depending on presenting history, clinical examination and relevant laboratory investigations. A typical presentation is not so common as many inflammatory and non-inflammatory conditions mimic appendicitis. A misdiagnosis might result in negative exploration, while delayed diagnosis results in complications like appendicular perforation and abscess. Therefore surgeons are more inclined to operate, when diagnosis is probable rather than wait until it is certain.

Recently, C Reactive Protein (CRP) is considered as the one of the indictor of

acute appendicitis. It is one of the acute phase reactant protein that may rise in concentration during acute phase response to inflammation. It is not disease specific marker but it offers valuable diagnostic information about the presence of acute infection with concomitant evaluation of patient history and physical examination.

Thus, this study is an attempt to evaluate the significance of total leukocyte count and CRP in diagnosing acute appendicitis where no other obvious diagnosis of concern is being considered.

OBJECTIVES

1. To individually correlate CRP and total leukocyte count with histopathology report in case of acute appendicitis
2. To evaluate the efficacy of combining both CRP and TLC in acute appendicitis.
3. To interpret how these investigations can be used effectively to improve the diagnosis and management of acute appendicitis.

REVIEW OF LITERATURE

EVOLUTION OF APPENDICITIS[6]

The disease appendicitis has been known since centuries. It seemed reasonable to believe that the presence of the appendix was well known when the pyramids were built, because all the viscera were removed from the body during the process of mummification and placed in four separate Coptic jars.

Aretaeus, of Cappadocia, in 30 A.D., is reported to have described accurately an abscess of the appendix in which the patient recovered after simple incision and drainage.

Leonardo Da vinci clearly depicted the appendix in anatomic drawings which was made in 1492 but was not published until the 18th century. Berengario da Carpi, in 1524, gave the first written account of the appendix which has been preserved. In 1543, the Fleming, Andreas Vesalius, professor of anatomy at Padua, accurately described and illustrated the normal appendix, with its relationship to other organs, in the magnificent "De fabrica humani corporis."

Verheye, in 1710, coined the term "appendix vermiformis" as a marginal heading in one of his writings. Santorini, in 1742, described the various positions of the appendix in the adult and illustrated several fecal concretions and worms found in his specimens. He concluded that the appendix served as a "nest" for worms of the gastro-intestinal tract.

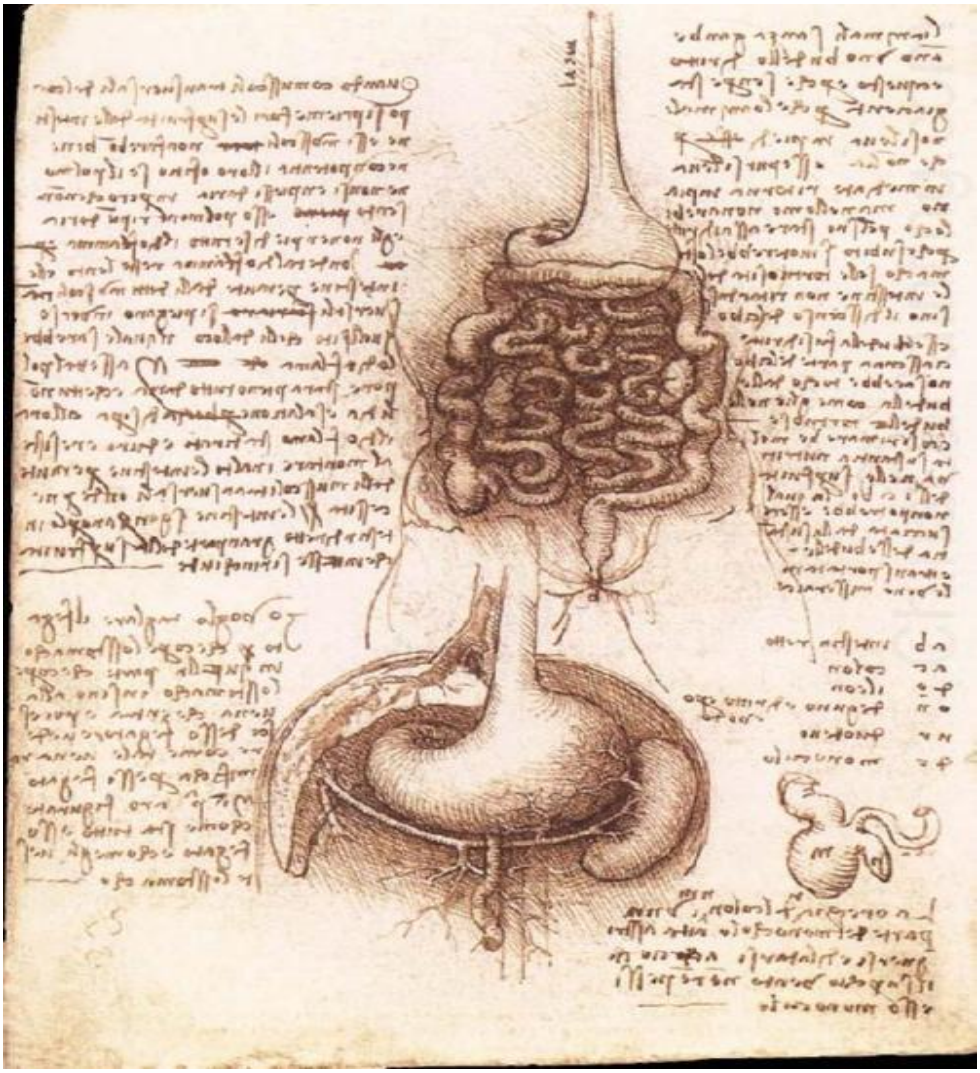


Figure 1: An early depiction of the Appendix by Leonardo da vinici

Lorenz Heister, professor of surgery at Helmstadt discovered a case of appendicitis in 1711, when he was called to dissect the body of a criminal who had been executed. When he was about to demonstrate the situation of the great guts, he found the vermiform process of the caecum black and adhering closer to the peritoneum than usual".[7]

The first modern operation for an abscess of the appendix was done by a French surgeon, Mestivier, in 1759. He had originally pointed out that pathologic processes in the appendix would cause death.

In 1827, Melier, a French physician, presented a classic description of appendicitis

and stated that it could cause primarily the lesions found in disease in the right lower quadrant of the abdomen. However, Dupuytren, the leading French surgeon of his day, together with his pupils, Husson, Dance and Meniere, disagreed with Melier's statements. About 1830, Goldbeck and his German school advanced the belief that the primary disease in the ileocaecal region lay in the caccum and not in the appendix. Thus, they advanced the terms "perityphlitis," "epityphlitis," and "endotyphlitis," in the hope that they were clarifying the situation. In reality, their efforts led to confusion and to a further retardation of the accurate clinical conceptions of appendiceal disease.

In 1846, Volz, another German, showed conclusively in forty post-mortem examinations of cases of appendicitis that the inflammation of the caecum was always secondary to that of the appendix. However, his work was not widely accepted by his fellow countrymen at that time.

In 1848, Hancock, of London, operated on a patient with acute appendicitis before the formation of abscess; a fecolith was found obstructing the lumen. In 1886, Hall was the first American surgeon to remove a gangrenous appendix found accidentally in a strangulated inguinal hernia. In the same year, Fitz published the first 100 cases of successful drainage of appendicular abscesses that covered the period from 1848 to 1886 that he was able to find in the literature of this country. From this work he showed, for the first time in a clear and decisive manner, that all inflammatory processes in the right lower quadrant of the abdomen should be considered, until proved otherwise, as originating in the appendix. He coined the term "appendicitis."

In 1899 Charles McBurney of New York illustrated that "exact locality of the maximum tenderness, when one examines with the finger tips in adults, is one-half to two inches inside the right anterior spinous process of the ileum on the line drawn to the umbilicus. The accuracy of this sign (McBurney's point) I have demonstrated in every case operated upon by me since I first made the observation".[7] This point corresponds to the base of the appendix and therefore does not move with the tip.

In 1893, Ribbert, of Germany, was the first to advance the belief that the appendix normally obliterated its lumen physiologically from the tip proximally toward the base. In 1894, Fowler is said to be the first American to publish a book on appendicitis; he reported 200 successfully performed operations. He stated that appendicitis is the most common cause of disease in the right lower quadrant in men.

EVOLUTION OF APPENDICECTOMY

According to Richardson RG in "The Surgeons Tale", the first appendicectomy was performed in 1726, at St. Georges Hospital, London, by Claudius Amyand. The patient, a boy, had hernia and a faecal fistula. Richardson reported that, "When he opened the scrotum he found the appendix in the unusual position and that appendix was perforated by a pin which was removed and then he dealt with the hernia and fistula".[8]

Hancock in London successfully drained an appendix abscess in a female patient aged 30 years that was in her eight month of pregnancy in 1848. After incising the peritoneum, fluid was drained and he made no search for the appendix.[7] Willard Parker, an American surgeon, started draining appendiceal abscesses since1867.

He did not remove the appendix and his technique is still used but the appendix is removed later on.[7]

Lawson Tait, the great English surgeon, was the first to remove an acutely inflamed appendix.[9] He thought that his patient had a general peritonitis resulting from rupture of caecum or appendix. However, when he opened the abdomen he found "a large abscess which extended deeply down towards the brim of the pelvis lying bare was the vermiform appendix which was black and discolored and gangrenous". The patient made a perfect recovery following appendicectomy and drainage of abscess.[7]

Abraham Groves performed the first elective appendicectomy in Canada in 1883. His patient was a twelve-year old boy. The appendix was removed and the stump was cauterized with a heat probe heated over the flame of a lamp.

Early operation for appendicitis was widely promulgated by surgeons like John Deaver (1855-1931), Charles McBurney (1845-1913) and Murphy of Chicago.[10] In 1894 McBurney described incision for appendectomy, but McArthur LL, who had already used this incision in more than 60 cases,[7] for almost a long time. Later McBurney gave Mcarthur the credit for using the incision first, but despite this, it is still known as the McBurney's incision.

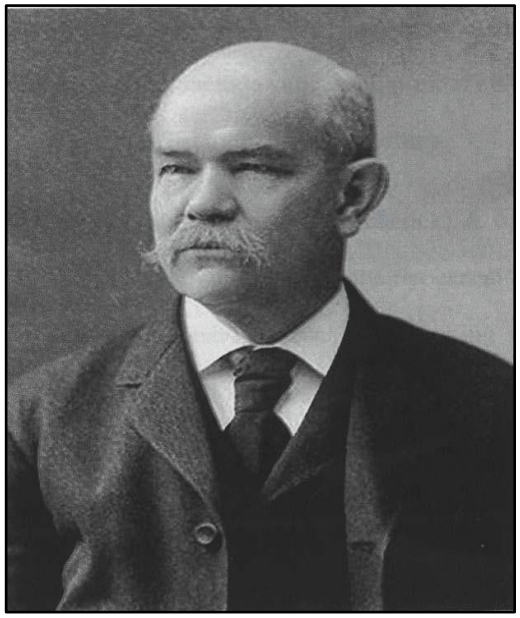


Figure 2: Reginald Fitz, coined the term Appendicitis in 1886

Figure 3: Claudius Amyand

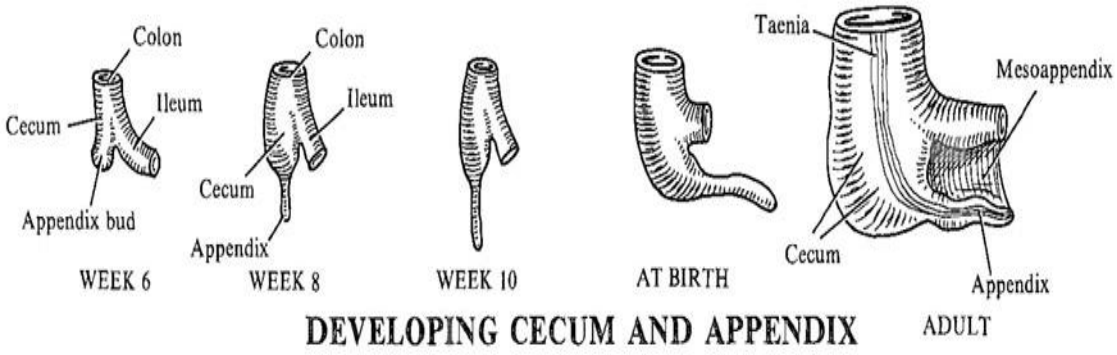


Figure 4

EMBRYOLOGY OF APPENDIX

Appendix develops as an underdeveloped distal end of the caecum in the 6th week of intra-uterine life. It develops from the post arterial segment of the midgut, along with caecum, ascending colon and the right 2/3rd of the transverse colon.

Initially a bud called the caecal bud arises from the post arterial segment very near to the apex of the loop. The proximal part of bud grows rapidly to form the caecum but the distal part remains narrow and forms the appendix[11]. The vermiform appendix becomes visible in the eighth week of gestation (length of the fetus 10-12 cm).

Subsequently, the lateral wall of the caecum grows much more rapidly than the medial wall, thus the point of attachment of appendix comes to lie on the posteromedial aspect of the caecum.[12]

Initially the caecum lies just below the liver and the ascending colon cannot be demarcated. Gradually the caecum descends to the right iliac fossa and the ascending, transverse and descending colon becomes distinct.

In the final stage, the duodenum, ascending colon and the descending colon becomes retroperitoneal by the fusion of their mesenteries to the posterior abdominal wall. But the small intestine, transverse colon, sigmoid colon and appendix mesenteries remain free.

CONGENITAL VARIATIONS

Congenital absence

Robinson JO (1952) reported 68 cases of congenital absence of appendix.

Duplication and Triplication

In 1968, Tinkler reported on operating on a triple appendix in a Chinese male child aged 12 months with other congenital anomalies.

Wall Bridge (1962) classified duplication of appendix as:

Type A: Distal duplication with a common base (i.e., bifid appendix).

Type B: A single caecum with 2 complete separate appendices, further divided into:

Type B-1: Bud like 2 appendices symmetrically placed on either side of the ileo- caecal valve.

Type B-2: 'Taenia colic' type - one appendix from the usual site, the other from the caecum above the lining of taenia at varying distance from the first.

Type C: A double caecum each bears an appendix.

The classification by Waugh differentiates only three types:

Type 1-Appendix with two separate luminae and a common appendiceal muscular wall.

Type 2-Two completely separated appendices originating from the cecum.

Type 3-A normal-shaped and normal-positioned appendix combined with a hypoplastic one, with possible atypical origin.

Variations in position

- Due to incomplete downward descent of the caecum, the appendix may remain in sub-hepatic position.
- Due to overgrowth of ascending colon, appendix may sometimes descend down to a pelvic position along with the caecum.
- Due to incomplete or non-rotation of the midgut loop, appendix may assume a position on the left side of the abdomen, associated with transposition of other viscera.
- Caecum may have a long mesentery and be mobile; because of its mobility, appendix may assume a variable position in the abdomen.

ANATOMY[10,13,14]

Vermiform appendix is described as a narrow, worm-like tube arising from the posteromedial caecal wall. It constantly arises from the site at which the 3 taenia coli converge. The 3 taenia coli merge into a complete longitudinal muscle layer over the appendix. The anterior taenia is usually distinct and traceable to the appendix, offering a guide to it.

Appendix varies from 2-20 cm in length, the average being about 9 cm. It is longer in children and atrophies or diminishes after mid adult life. The diameter is about 5 mm. The lumen is quite narrow and get obliterated after mid-adult life. The canal of vermiform process is small, extends throughout the whole length of tube, and communicates with the caecum by an orifice, which is placed below and behind the ileocaecal opening. It is sometimes guarded by a semi lunar valve formed by a fold of mucous membrane known as valve of Gerlach, but this is by no means constant.

POSITIONS[15,16]

Treves describes the following anatomical types comparing the appendix with the face of the clock.

- 11'O clock **paracolic** (lies on the sulcus in lateral aspect of the caecum).
- 12'O clock **retroceacal** (lies behind the caecum and may even be totally or partially retrocaecal).
- 1'O clock **pre-ileal**
- 2'O clock **post-ileal**

- 3'O clock **promontoric** (the tip of appendix points towards promontory of sacrum).
- 4'O clock **pelvic** (appendix dips into the pelvis)
- 6'O clock **subcaecal or mid inguinal**

Wakeley, in the year 1933 in an analysis of 10000 cases at post-mortem examination gave the location of appendix as follows:[17]

Retrocaecal and retrocolic	: 65.28%
Pelvic	: 31.01%
Subcaecal	: 2.26%
Preileal	: 1.0%
Postileal	: 0.4%

MESENTERY OF APPENDIX

The appendix has a complete peritoneal investment and a small mesentery. This fold is derived from left leaf of peritoneum and is a continuity of the mesentery. It is triangular in shape and is attached along the whole length of the appendix.

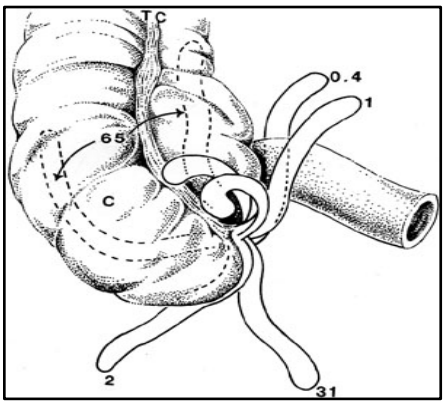


Figure 5: The diagram illustrates the positions, the appendix may occupy in relation to the cecum and ileum, with frequencies of occurrence (%)

BLOOD SUPPLY

Arterial

Main appendicular artery is a branch of the lower division of the ileo-colic, runs behind the terminal ileum and enters the mesoappendix a little away from the base of the appendix. Here it gives off a recurrent branch which anastamoses at the base of the appendix, with a posterior caecal artery branch.

The terminal part of the main artery lies on the wall of the appendix and may get thrombosed in appendicitis resulting in distal gangrene or necrosis. Variations are considerable. In nearly 50% of the cases there is an accessory appendicular artery, a branch of posterior caecal artery (artery of Sheshachalam).

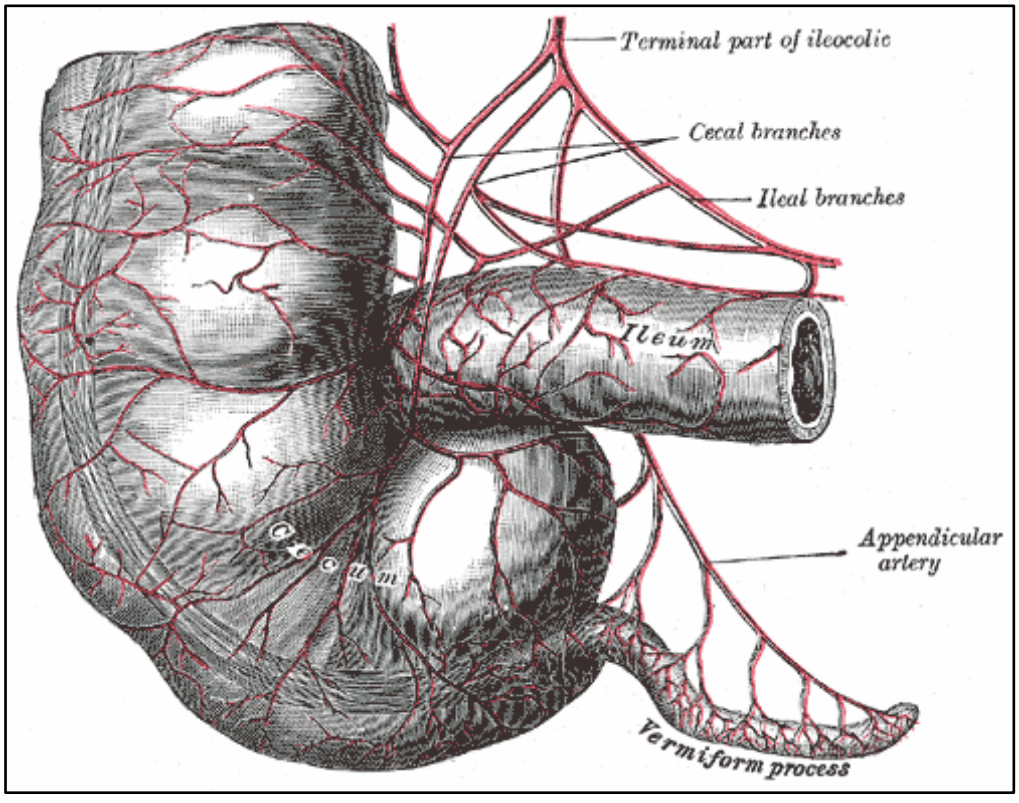


Figure 6: Blood supply of Appendix

Venous

Appendicular vein is a tributary to the ileo-colic vein, which in turn drains into the portal system.

Lymphatic

Through the muscle wall the lymphatics drain into nodes in the meso appendix. These drain into the paracolic nodes lying along the ileo-colic artery and then into the superior mesenteric group.

Nerve Supply

Sympathetic : Coeliac and superior mesenteric ganglia (T11, T12)

Parasympathetic : Vagus

Both these nerves form a plexus around the artery supplying the appendix.

SURFACE MARKING

The base of the appendix corresponds to the McBurney's point. It is formed by the junction of the lateral $1/3^{rd}$ and medial $2/3^{rd}$ of the line joining the umbilicus and the right anterior superior iliac spine. It is merely a surgical approximation and variations are considerably common.

LUMEN OF THE APPENDIX

Canal is small and opens into the caecum by an orifice lying just below and behind the ileocaecal opening. The orifice is guarded by a semi lunar mucosal fold forming a valve. The lumen of appendix is lined by columnar cells with few crypts .At the base of crypts, special cells called .Kultschitzsky cells lie, which can give rise to carcinoid tumors, and that can lead to appendicitis.

HISTOLOGY[18,19,20,21]

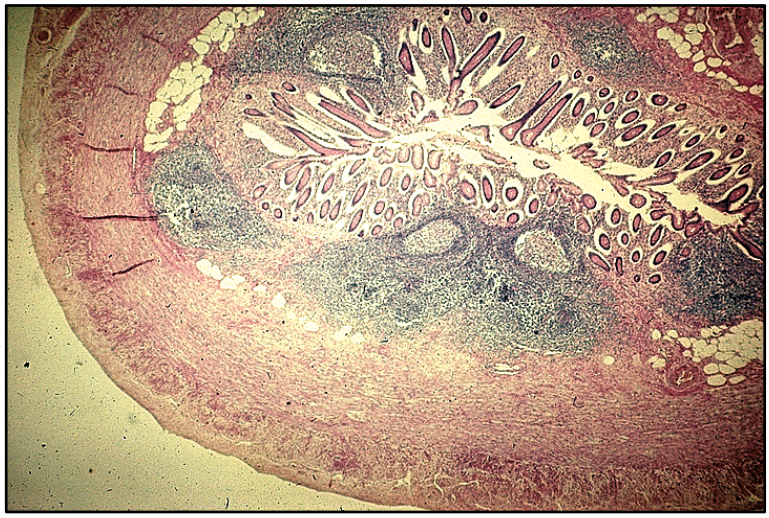


Figure 7: Normal histology of appendix

The structure of the appendix includes 4 layers i.e, serosa, muscularis mucosa, sub-mucosa, mucosa .

Serosa: is a complete investment except along the mesenteric attachment and there is a subserosal layer of connective tissue.

Muscularis layer: longitudinal muscle fibers form a complete uniformly thick layer, except over a few small areas where both muscular layers are deficient leaving serosa and submucosa in contact. At the base the longitudinal muscle thickens to form the rudimentary taeniae. The circular muscle fibers form a thicker layer separated by connective tissue.

Submucosa: contains many lymphoid masses, causing the mucosa to bulge into the lumen, narrowing it irregularly. This profusion of lymphoid tissue has promoted the description of 'Abdominal Tonsil' for appendix.

Mucosa: is covered by columnar epithelium and attenuated antigen transporting M-cells

Glands are few, penetrating deeply into lymphoid tissue. Lymphoid tissue in the lamina propria contains many plasma cells with lymphocytes, eosinophils, mast cells, macrophages embedded in fibro-cellular reticulum.

Function of Appendix

Appendix was previously viewed as a vestigial organ, but now it is well recognized as an immunologic organ, which actively participates in secretion of immunoglobulins, particularly IgA.

Appendix is an integral part of gut-associated lymphoid tissue (GALT), but its function is not essential.

AETIOPATHOGENESIS[10,22]

Etiological factors can be divided into 2 types, predisposing and exciting factors.

I. Predisposing factors

1. **Age**: Commonest in the 2nd and 3rd decades. Rare in infancy and old age. In infancy the lumen of the appendix is fairly large and in old age, the appendix often undergoes involution.
2. **Sex**: Males are more commonly affected than females. Before puberty, M:F ratio is 1:1, after puberty, the same is 2:1 up to 25 years.
3. **Race and Diet**: The disease is common in highly civilized countries and certain communities, but rare in remote rural districts and among primitive people. Natives of rural areas who live on a diet abundant in cellulose are immune to the disease, but when they adopt the diet of civilization, they lose that immunity. The severe gangrenous type of appendicitis is confined to meat eating people. But this cannot be the whole explanation because acute appendicitis occurs even

in life-long vegetarians and in babies on breastfeeds. Denis P Burkitt after extensive research, concluded that undue refining of dietary carbohydrate is the most important causative factor.

4. **Social status**: It is more common in the upper and middle class than in the lower classes.
5. **Familial susceptibility**: This is unusual but generally accepted fact as hereditary abnormality in the position of the organ is noticed, which predisposes it to infection. Such as the whole family having a long retroceacal appendix with comparatively poor blood supply.
6. **Seasonal**: More common in winter.
7. **Abuse of purgatives**: They cause violent peristaltic waves. This results in perforation of the inflamed appendix. This occurs particularly in case of castor oil taken for stomach ache.
8. **Obstructive agents**
    - Faecoliths: begins to form with entrapment of a bit of vegetable fiber in the lumen of the appendix stimulating secretion and deposition of calcium rich mucus. The mucus subsequently becomes inspissated around the fiber. Eventually concretions reach a diameter of approximately 1 cm at which point, if not expelled, they may obstruct the lumen and cause appendicitis.
    - Worms: Ascariasis, Enterobius vermicularis, taenia, etc. They can injure the appendicular mucosa and occasionally block its lumen.
    - Swelling of the abundant lymphoid tissue.
    - Contraction of a sphincter-like mechanism at the base of appendix.

- Fibrous contracture of the proximal end from previous attacks.
- Kinking of the appendix by a band or a fold.
- Distal obstruction of colon: acute appendicitis can result from an obstructive carcinoma, usually of the right colon in the elderly.
- Foreign bodies: small fragments of bone, metal, seeds, pins, etc., can cause damage and incite inflammation.
- Barium contrast agent.
- Carcinoid tumor.

PATHOLOGY[19,20,21,22]

Acute appendicitis is thought to arise from infection superimposed on luminal obstruction.

Pathology

It is of great importance to recognise two types of acute appendicitis:

a. Non-obstructive (catarrhal) acute appendicitis: Catarrhal appendicitis is initially a mucosal and submucosal inflammation. Externally; the appendix may be quite normal, or hyperemic in early stages. However the mucosa wall is thickened edematous and reddened. Later it becomes studded with dark brown hemorrhagic infarcts, patches of green gangrene, or small ulcers. Eventually the appendix becomes swollen and turgid and the serosa becomes roughened coated with fibrinous exudates. In these cases the lumen of appendix is patent and these cases rarely progress to gangrene. However the lymphoid hyperplasia may lead to obstruction of the lumen and proceed to gangrene. Furthermore, if the episode of catarrhal appendicitis resolves, adhesion formation and kinking of the appendix may lead to a final episode of acute obstructive appendicitis or recurrent appendicitis.

b. Obstructive acute appendicitis

The lumen of the appendix may be obstructed by fecolith, hyperplasia of submucus lymphoid follicles, stricture, tumor, or any pathological condition. Once obstruction occurs, continuous mucus secretion and inflammatory exudation increases intraluminal pressure which in turn obstructs lymphatic drainage. Oedema and mucosal ulceration develops due to pressure atrophy followed by bacterial translocation to the submucosa. Resolution occurs at this point either spontaneously

or in response to antibiotic therapy. If this condition progresses, further distention of the appendix may cause venous obstruction and ischemic of the appendix wall. With ischemia, bacterial invasion occurs through the muscularis propria and submucosa, producing acute appendicitis. The inflammation of wall leads to thrombosis of vessels as the appendicular artery is an end artery. Finally ischemic necrosis of the appendicular wall produces gangrenous appendicitis, with free bacterial contamination of the peritoneal cavity, which further causes the greater omentum and loops of small bowel to become adherent to the inflamed appendix, resulting in an appendicular mass or appendicular abscess.[14]

Obstructive appendix is thus the dangerous type, since the appendix becomes closed loop of bowel containing fecal material.

Microbiology

Cultures from inflamed appendices usually reveal that the infection is mixed and there is hardly a pyogenic organism, which has not been isolated from such specimens. The most common organisms are a mixture of *Escherichia coli* (85%), *entercocci* (30%), nonhemolytic *streptococci*, anaerobic *streptococci*, together with *clostridium welchii* (30%) and bacteroides. In most instances, the infecting organisms are normal inhabitants of the lumen of the appendix. The foul odour of exudates associated with perforated appendix is caused by anaerobic streptococci or anaerobic bacilli and not by Escherichia coli.

The most frequent site of perforation is along the antimesenteric border, usually near the tip, as the Appendicular artery is subserosal at this point and more prone to be involved in the inflammatory process and become thrombosed. After

perforation a localized abscess may form in the right iliac fossa or the pelvis, or diffuse peritonitis may ensue. Whether the peritonitis remains localized or becomes generalized depends on many factors, including age of the patient, the virulence of the invading bacteria, the rate at which he inflammatory condition has progressed within the appendix and the position of the appendix.[14] It is usually stated that the poorer localization of the infection occurs in infants because the omentum of the child is filmy and less able to form a protective sheath around the inflamed appendix. A more likely explanation is that delays in diagnosis is more prone to occur in infants. Similar delay occurs in the management of elderly persons. Gangrenous appendix is most dangerous than the catarrhal type of appendicitis. An appendix situated in the retrocaecal position is more likely to form a local abscess than one in the preilieal or subcaecal position.[23]

The consequences of a perforated appendix are potentially severe in women of childbearing age. The relative risk of infertility is increased three to five times in a female patient with a history of a ruptured appendix.[9]

Morphology

Initially the only sign is dilated vessels on the serosal aspect of the appendix. The distal segment then dilates and may contain purulent material. As inflammation spreads through the wall, patchy purulent exudates appear on the serosal surface. The appendix later becomes soft violaceous and haemorrhagic with developing necrosis culminating in gangrene and perforation.

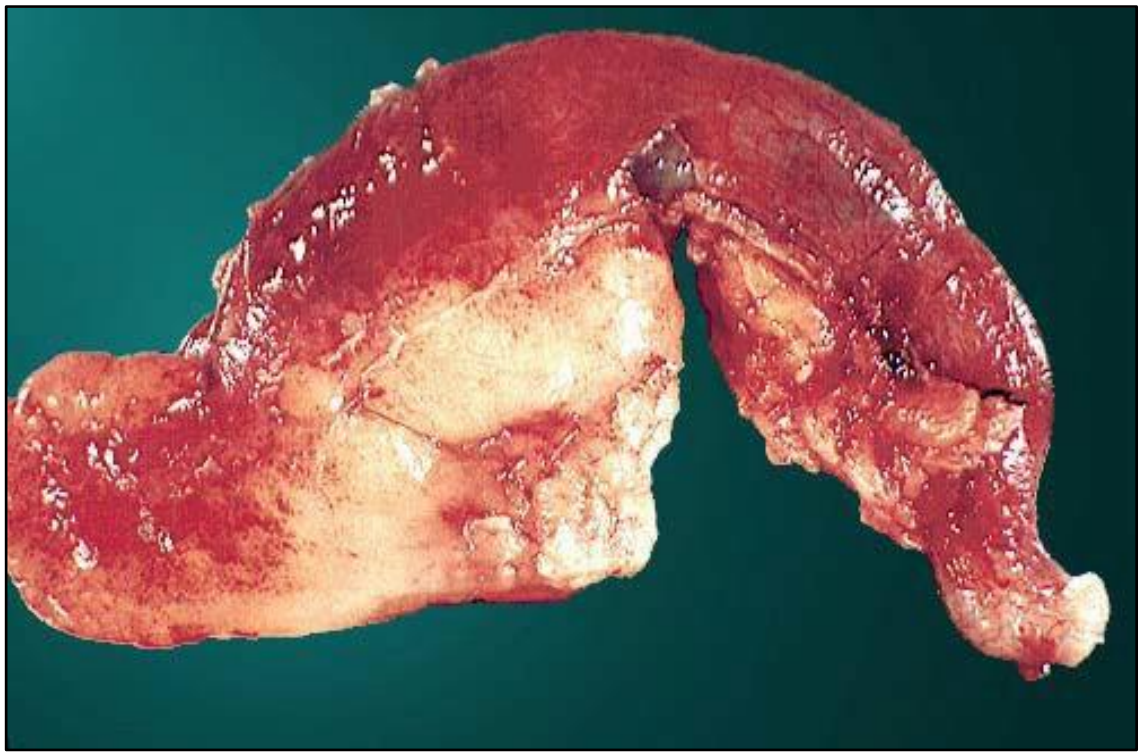


Figure 8: Gross specimen of acute appendix

MICROSCOPY

The histologic criterion for the diagnosis is neutrophilic infiltration of the muscularis. In the early stages, there is only scanty transmural infiltrate of neutrophils and a modest perivascular neutrophilic infiltration. The inflammatory reaction transforms the normal glistening serosa into a dull, red, granular membrane. This transformation signifies early acute appendicitis for an operating surgeon. At a late stage, a prominent neutrophilic exudate generates a fibrinopurulent reaction in the serosa. With the worsening of the inflammatory process, abscess formation occurs within the wall along with ulcerations and foci of suppurative necrosis of the mucosa. This stage constitutes acute suppurative appendicitis. In the later stages it further leads to large areas of haemorrhagic ulceration of the mucosa and green black gangrenous necrosis.

The necrosis extends through the wall into the serosa creating acute gangrenous appendicitis, which is quickly followed by rupture, causing suppurative peritonitis.

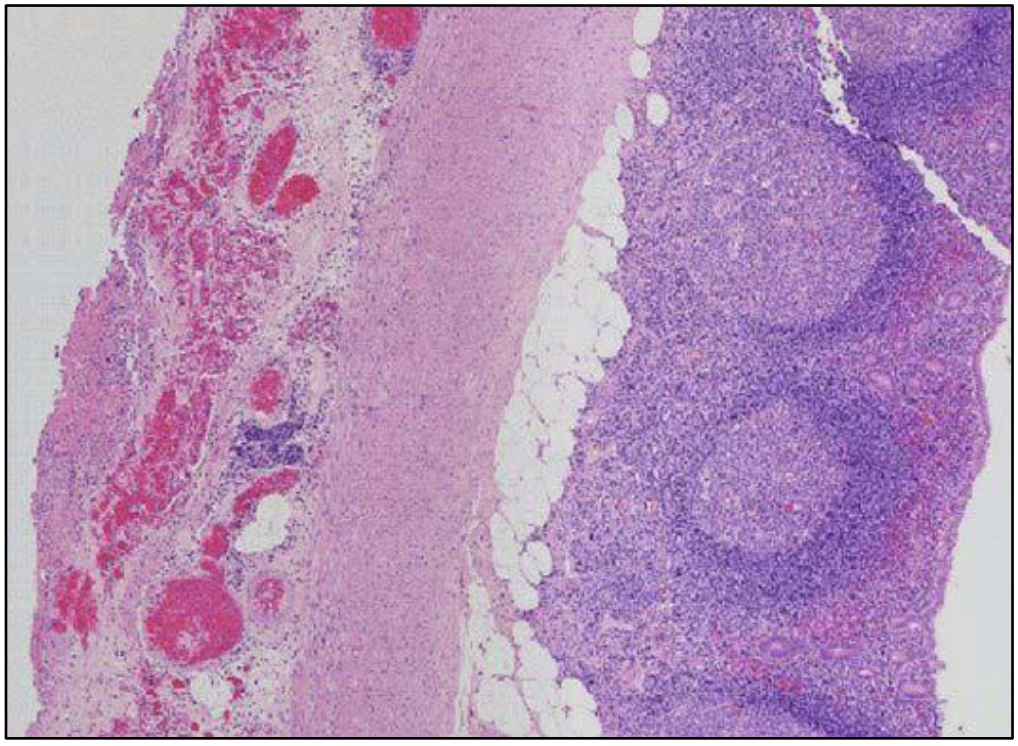


Figure 9: Microscopic picture of acute appendicitis showing neutrophilic infiltration

Chronic appendicitis: Chronic appendicitis is a controversial entity often referred to as a grumbling appendix. A small shrunken appendix with fibrous obliteration of lumen and atrophy of the lymphoid tissue might suggest previous attacks of inflammation. However, such findings are as common in symptomless population as well. Histology reveals lymphocytes and plasma cells throughout the wall, in the absence of neutrophilic polymorphs. It is a rare finding.

CLINICAL FEATURES

The clinical features progress in the following manner.

1. Anorexia
2. Mild to moderate, poorly localized mid-abdominal pain.
3. Nausea and vomiting.
4. Pain migrating to right lower quadrant (RLQ)
5. Localized tenderness or guarding over the appendix.
6. Muscular rigidity or peritoneal signs in the RLQ.

Murphy's syndrome is charecterised by sequence of pain, vomiting and fever. "If vomiting occurs before pain abdomen then the diagnosis of acute appendicitis is questionable and a peaceful night is assured to the surgeon".[24]

Murphy states that, "The symptoms occur almost without exception in the above order, and if the order varies I always question the diagnosis." This dictum is usually true with occasional exceptions.

1. Anorexia: Many authors have stated anorexia to be essential in the diagnosis of appendicitis. Return of appetite is not a reliable sign in excluding the diagnosis of acute appendicitis.

2. Abdominal pain: Pain is the chief symptom of acute appendicitis and typically follows anorexia. When the pain begins suddenly in the right lower quadrant (RLQ) and is severe, with signs of rebound pain at the onset, it is unlikely that appendicitis is the cause. Classically, the initial pain is centered over mid abdomen. Over a variable period of time, that can be as little as 4 to 6 hours to as long as 24 to 48 hours, the pain migrates to the RLQ and remains there. This shift in pain occurs

when the inflammation has spread to the serosal coat of the appendix and local peritonitis is present. Knowledge of the most common anatomic positions of the appendix will lead to recognition in alterations of pain pattern and location.

3. Nausea and vomiting: Distension and obstruction of any luminal structure will produce nausea and vomiting. Nausea and vomiting are thought to occur less commonly with retrocaecal and retroileal appendicitis. Vomiting may be more prominent in children.

4. Constipation or diarrhoea: As appendicitis progresses, an ileus may develop leading to constipation or atleast decreased frequency of the normal bowel habit. Diarrhoea in contradiction is not a common component, except in patients with missed appendicitis and postileal appendix. Diarrhea may be more common in children than in older patients.[25]

5. Urinary symptoms: Dysuria can occur in pelvic appendicitis. An inflamed appendix if in contact with the bladder will cause frequency of micturition, alterations of pain pattern and location.

6. Pyrexia: Fever is usually only 99° F to 100° F. Fever in excess of 102° F is often, but not always associated with perforated appendicitis. Usually, it is associated with tachycardia.

Peritoneal signs

a. Pointing test: When patient is asked to point the site of pain, it usually corresponds with the site of localized tenderness in McBurney's point. This point is at the junction of lateral third with medial two thirds of the spino-umbilical line (McBurney's sign).[26]

b. Rovsing's sign: Palpation in the left iliac fossa may produce pain in the right iliac fossa.[18]

c. Dunphy's sign: When the patient coughs vigorously and holds his or her RLQ or refuses to cough because of pain, RLQ peritonitis is suspected.

d. Blumberg's sign (Release sign): Rebound tenderness in the RLQ suggests localised peritonitis.[27]

e. Psoas test: A retrocaecal appendix lies on psoas major muscle. Inflammation of this causes irritation of psoas major, which is concerned, with flexion of hip joint.

f. Cope's obturator test: Flexion and internal rotation of hip in a patient with pelvic appendicitis initiates pain as the appendix lies over obturator internus muscle.

g. Baldwin's sign: A hand is placed over the right flank and patient is asked to raise the right lower limb with knee extended. In retrocaecal appendicitis, this initiates pain.

h. Ligat's sign: Hyperaesthesia in Sherren's triangle (formed by lines joining the umbilicus, right anterior superior iliac spine and symphysis pubis) is an occasional but inconstant accompanient of gangrenous appendicitis.[27]

Clinical outcome of acute appendicitis

1. Resolution
2. Gangrenous appendicitis
3. Perforation leading to generalized peritonitis
4. Appendicular mass or abscess formation
5. Fibrosis

COMPLICATIONS

Perforation of appendix is the most common and most serious complication leading to generalised peritonitis[28] and localized abscess formation. Abscesses may perforate into rectum or vagina. In women, the end-result of perforation of the appendix can be tubal adhesions and infertility.[29] Fistula may occur between the appendix and bladder or elsewhere in the gastrointestinal tract. Occasionally, vessels in mesoappendix can become infected and thrombosed. The thrombosis may propagate into larger vessels and may predispose to hepatic abscess.

SPECIAL FEATURES

A. According to positions

1. Retrocaecal appendicitis: Rigidity is often absent in retrocaecal appendicitis. Psoas test and Rovsing's sign are positive. This may produce flank or back pain.

2. Pelvic appendicitis: Irritation of the bladder (strangury) and the rectum (passage of mucus per anum and tenesmus)[9] can be present. Presence of Rovsing's sign and psoas test confirms the diagnosis. Rectal examination shows tenderness. Pain may be present in suprapubic region.

3. Post ileal: Although this is rare, it account for some of the cases of missed appendix. Pain may not shift. Diarrhoea is a feature, with marked retching. Tenderness is ill defined. May also produce testicular pain.

4. Maldescended (subhepatic): Tenderness is in the subhepatic region. It is sometimes mistaken for acute cholecystitis.

B. According to age:

1. Appendicitis in Children: Appendicitis is rare before 2 years of age because of relatively wide lumen of the appendix. Between 2-11 years the incidence rises. Mortality and morbidity in preschool children with appendicitis is high, because

(a) the child can't give history

(b) Child is brought to the hospital late by parents

(c) Omentum is under developed in children so diffuse peritonitis develops early.

In the Elderly: Gangrenous changes and perforation occurs five times as often in the older age group. This is because of poorer localization of the infection and diminished blood supply of the appendix, allowing rapid progression of the disease. Elderly patient with lax abdominal wall or obesity with gangrenous appendix may have little evidence of it, and the clinical picture may simulate subacute obstruction of intestine. Coincidental medical conditions produce a much higher mortality for acute appendicitis in the elderly.

Most elderly people are less likely to complain of pain than younger people and also acute appendicitis is not suspected most of the times.

Appendicitis in Pregnancy[30]

Pregnant women are neither more nor less prone to appendicitis than a nonpregnant young female. But the diagnosis is undoubtedly more difficult in pregnant women.

1) In the first trimester it is confused for ruptured ectopic pregnancy. The nausea and vomiting may be thought to be physiologic morning sickness, consequently delaying accurate diagnosis.

2) In the second trimester uterus enlarges and appendix is pushed upward and more laterally. Thus, the pain, tenderness and guarding are situated in the mid or upper abdomen, which may lead to confusion with pyelitis or cholecystitis.

3) In later stages of pregnancy because of stretched abdominal muscles, detection of guarding or rigidity becomes difficult.

In general, there is a counterclockwise rotation, with the tip of the appendix being displaced cephalad. A useful sign may be to roll the patient on her left side. If the pain shifts, it is more likely to be appendicitis.

The best rule is to treat the patient as if she were not pregnant. Once perforation occurs, labour may ensue, resulting in prematurity or fetal demise. Peritonitis leads to increased foetal loss, which has been quoted to be 35% to 70%.

Babler's statement from the early 1900s is apt today: *"The mortality of appendicitis complicating pregnancy is the mortality of delay"*.

DIFFERENTIAL DIAGNOSIS

Although acute appendicitis is the most common abdominal emergency, the diagnosis at times can be extremely difficult. There are a number of common conditions that it is wise to consider carefully and, if possible exclude. The differential diagnosis differs in patients of different ages. In women, additional differential diagnosis is involvement of the genital tract.

Table 1: Differential diagnosis for acute appendicitis

CHILDREN	ADULTS	ADULT FEMALE	ELDERLY
Gastro enteritis	Regional enteritis	Mittleschmertz	Diverticulitis
Mesenteric adenitis	Ureteric colic	Pelvic inflammatory disease	Intestinal obstruction
Meckel's diverticulitis	Perforated peptic ulcer	Pyelonephritis	Colonic carcinoma
Intussusception	Torsion testis	Ectopic pregnancy	Torsion appendix
Henoch Schonlein purpura	Pancreatitis	Torsion/rupture of ovarian cyst	Mesenteric infarction
Lobar pneumonia	Rectus sheath haematoma	Endometriosis	Leaking aortic Aneurysm

INVESTIGATIONS IN ACUTE APPENDICITIS

WBC Count

Some authors stress a polymorphic leukocytosis as an important feature for diagnosing acute appendicitis. The leukocyte count is raised above 12000 cells/mm^6 in three fourths of patients with acute appendicitis. In a study of 493 patients with acute appendicitis, Pieper and associates in 1982 noted that 66.7% had a leukocyte count of 11,000 or more and 5.5% had a raised count of more than 20,000.[1]

A considerable overlap exists between the TLC and neutrophil count of healthy individuals and those with acute appendicitis. Interpretation of these counts together is more significant than either count alone.

It is clear that 80-85% patients with acute appendicitis will have a total WBC count of over 10,000/cu mm.[15,16] Neutrophilia of > 75% will occur in 78% patients.[12] When TLC and neutrophil count are taken together, less than 4% patients with acute

appendicitis will have normal values. However, TLC is raised in 20-70% of patients with other causes of acute right iliac fossa pain. Leukocytosis increases with the duration of the disease process, but even a perforated appendix may present with a normal TLC. Of note is the observation of some that if TLC is repeated after a few hours, it tends to remain high in those with acute appendicitis but tends to fall in those without.

Andersson et al[12] reported that the WBC and neutrophils count had higher power in discriminating for advanced appendicitis than for all appendicitis. Appendicitis was unlikely at lowest level of the WBC and neutrophils count and rate (LR0.16-0.28 at WBC count <8000/cmm, neutrophils count<7000/cmm or rate <70%) and likely at the highest WBC count. However, Coleman C et al reported that WBC is a poor predictor of the severity of the disease.[11]

The white cell and neutrophil count are especially sensitive in children and elderly patients. Doraiswamy pointed out that the combination of a raised leukocyte count and neutrophilia is useful in the diagnosis of acute appendicitis in children.[31] He found that in 225 children with acute appendicitis, 96% had neutrophilia and 42% had a raised leukocyte count.

Vermeulen et al after evaluating 221 adult patients admitted with right lower abdominal pain have concluded that the white cell count did not significantly influence surgical decision-making in cases of suspected acute appendicits.[32] Coleman et al. reported that WBC is a poor predictor of the severity of the disease.[11] With appendicitis the white cell count has been variously reported as being either reliable or unreliable. Thus a raised white cell count, although highly sensitive for acute appendicitis, is rendered almost useless due to its low specificity and has little diagnostic value.

Where the white cell count is at variance with the clinical features, the latter should take precedence. The only value of white cell count would seem to be to prompt observation rather than operation in a patient who has equivocal features of appendicitis together with a normal count.

Urine examination

The presence of hematuria or pus cells in the urine does not rule out appendicitis. Irritation of ureter or urinary bladder by the inflamed pelvic or retrocecal appendix may cause microscopic hematuria or pyuria.[8,22]

The incidence of urinary findings was more in patients over 40 years of age. Urine examination is not helpful in the diagnosis or exclusion of appendicitis .Thus urine analysis is not a diagnostic test in patients with symptoms of either appendicitis or urinary tract infection.

Radiography

Plain films of abdomen in supine and erect position are of value in differential diagnosis of acute abdominal pain. However, they are nonspecific.

Brookers and Killen[33] have described a number of radiological signs in patients with acute appendicitis:

- Fluid level localized to the caecum and to the terminal ileum.
- Localized ileus, with gas in the caecum, ascending colon or terminal ileum.
- Increased soft tissue density in the right lower quadrant.
- Blurring of the flank strip, the radiolucent line produced by fat between the peritoneum and transverse abdominals.
- A faecolith in the right iliac fossa.

- Blurring of psoas shadow on the right side.
- A gas filled appendix.
- Free peritoneal gas
- Deformity of caecal gas shadow due to an adjacent inflammatory mass

They reviewed the X-rays of 200 patients undergoing laparotomy for acute appendicitis without knowing the diagnosis. 80% of patients with acute appendicitis had one or more of these signs positive. However 37% of patients who had normal appendix had similar X-rays findings.

Gas under the diaphragm is rare in perforated appendix and it is seen in 1-2% of cases only. Saebo reported three examples of pneumoperitoneum associated with a perforated appendix.[34]

Although there is no radiologic sign that is pathognomonic, there are certain signs that may point towards the diagnosis of acute appendicitis. None of these signs are specific to acute appendicitis and may be seen in patients with other pathology in the right iliac fossa and sometimes in normal subjects.

Furthermore, irradiation hazards, especially in women of reproductive age group and in children, as well as the cost of overloading radiology departments make this investigation of low yields.

BARIUM ENEMA STUDY

A single contrast study is performed on an unprepared bowel. Radiologic signs of acute appendicitis after barium enema are:

- Persistent non-visualization of appendix (5-10% normal appendices cannot be visualized).

- Partial visualization
- Pressure effects on the caecum.
- Irritability of the caecum or ileum as seen by fluoroscopy.

A further advantage is that barium enema can diagnose other diseases which may be confused with acute appendicitis[34] e.g. to exclude Crohn's disease, colon cancer, ischemic colitis which mimics appendicitis.

Its disadvantage lies in its relatively high incidence of technical failure and its radiation hazard. It can also lead to perforation. Better investigations are available than barium enema. So it is not done routinely.

Ultrasound in Diagnosis of Acute Appendicitis[35,36,37]

Objectives are:

1. To identify the patient with acute appendicitis
2. And the ones without acute appendicitis, to identify an alternative explanation for their right lower quadrant pain. There is well recognized overlap of symptomatology of appendicitis with a variety of other gastrointestinal conditions like acute typhilis, acute mesentric adenitis, variations of Crohn's disease, right sided diverticulitis.

In women, this list is expanded to include acute gynaecological conditions. Bendeck et al. found that women in particular benefited from preoperative imaging, by having a statistically significant lower rate of negative appedicectomy than in women who did not undergo preoperative imaging.

Technique

The graded compression technique for ultrasound examination of the appendix was

described by Julien Pnylaert in 1986.[38,39] Using a probe of at least 7MHz over the point of maximum tenderness in the right iliac fossa, pressure is gradually increased over the area in order to displace the bowel loops and fat. This reduces the artifacts from bowel contents and reduces the distance from the transducer to appendix. The compression should be applied gently and slowly to avoid pain. The direct visualization of the inflamed appendix is the sonographic hallmark of appendicitis.

The appendix may then be seen lying over the psoas muscle. In women a full bladder allows better examination of the uterus and ovaries; however it impedes examination of the appendicular region, thus bladder should be emptied after uterus and ovaries are examined.

Anatomy:

Identifiable structures of right iliac fossa are the filled caecum, the terminal ileum, a few mesenteric lymph nodes and a variable amount of intra- abdominal fat. The normal appendix cannot be usually identified however other investigations have reported seeing normal appendices on a sonogram. The normal appendix is mobile compressible with wall thickness of less than or equal to 3MM

Posterior manual compression technique:

Recently Lee et al.[40] described graded compression technique with adjuvant use of posterior manual compression technique for the sonographic diagnosis of acute appendicitis. With graded compression sonography alone, they achieved visualization of vermiform appendix in 485 (85%) of 570 patients. After the adjuvant use of this technique, the vermiform appendix was found in an addition 57 of 85 patients, with number identifiable increasing to 542 (95%) of 570 patients.

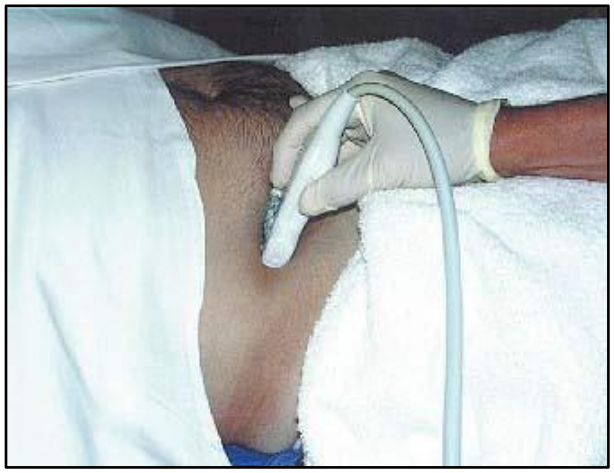


Figure 10: Linear ray ultrasound probe using compression technique
It is used to examine the RLQ of the abdomen. The examiner's left hand may be placed posteriorly to the patient's flank to ensure adequate compression.

Classical ultrasound features in acute appendicitis

MOST SENSITIVE SIGN

A) Blind-ending tubular structure at the point of tenderness

1) Non compressible, sausage like concentrically layered and other end attached to caecal pole with a gut signature
2) Diameter 7 mm or greater
3) No peristalsis

Sensitivity and specificity being a diameter of 6 mm or greater (sensitivity, 98%; specificity, 98%), lack of compressibility (sensitivity, 96%; specificity, 98%), and inflammatory fat changes (sensitivity, 91%; specificity, 76%).[41]

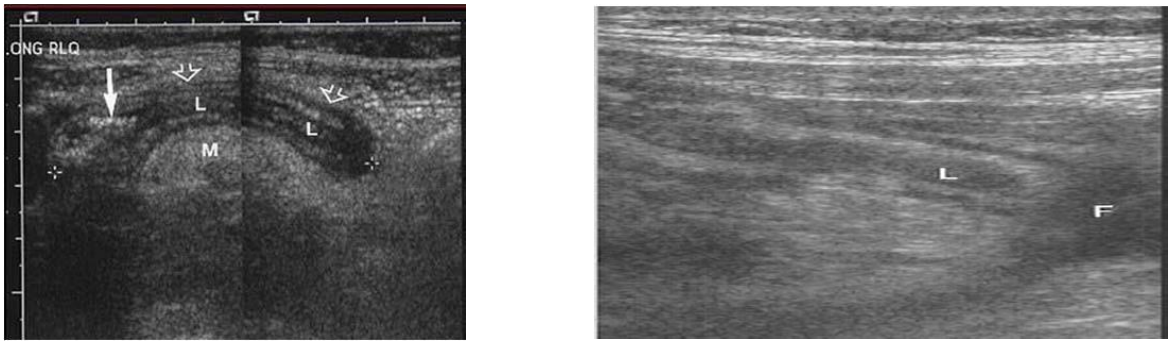


Figure 11: Ultrasound of the RLQ of the abdomen showing blind-ended tubular structure (open arrows) corresponding to acutely inflamed appendix. Also shown is the distended lumen [L], the echogenic surrounding mesentery [M], and the echogenic structure with acoustic shadow (arrow) at the base of the appendix corresponding to an appendolith.

B) Appendicolith obstructing the lumen (30% of cases) causing acoustic shadow.

C) High echogenicity non-compressible surrounding inflamed fat, large mesoappendix.

D) Surrounding fluid or abscess, non-compressible strongly reflective masses surrounding the appendix indicates extension of the omentum inflammation to the peri-appendicular fatty tissue.

E) Oedema of caecal pole. (This indicates extension of the inflammation to the peri appendiceal tissue).

F) An irregular, asymmetrical contour and loss of the layered structure indicates imminent perforation.

G) In half of the patients with appendicitis enlarged mesenteric lymph nodes can be demonstrated. During compression the nodes are found out lateral to IVC and right iliac vessels.

H) Oedema of the caecal pole

Color Doppler ultrasonography in acute appendicitis:

- Increased conspicuity (increase in size and number) of vessels in and around the appendix (hyperemia)
- Decreased resistance in arterial waveforms
- Continuous / pulsatile venous flow

Patriquin et al have demonstrated that acute appendicitis is characterized by inflammatory hypervascularity which is seen as an increased number of colour signals and higher diastolic doppler shifts as compared with those found in normal persons.[42] No doppler shifts are identified in areas of appendicular ischemia.

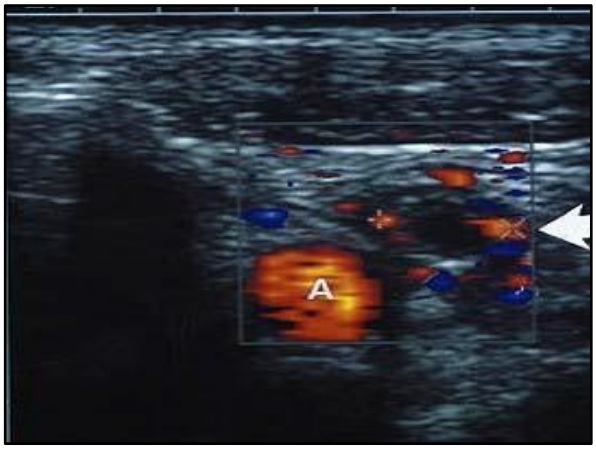


Figure 12: Transverse color – flow ultrasound of the RLQ of the abdomen demonstrates increased vascularity (arrow) in a fluid-filled structure corresponding to acute appendicitis. External iliac artery [A] is identified.

CT SCAN ABDOMEN IN ACUTE APPENDICITIS

High resolution, helical computer tomography also has been used to diagnose appendicitis. On CT scan, the inflamed appendix appears dilated and the wall is thickened. There is usually evidence of inflammation, with a dirty fat, thickened mesoappendix and even an obvious phlegmon. Fecolith can be visualized. An important suggestive abnormality is the arrow head sign which is caused by thickening of the cecum, this funnels the contrast towards the orifice of the inflamed appendix. CT scanning is an excellent technique for identifying other inflammatory processes masquerading as appendicitis.

A number of studies have proved the improvement in diagnostic accuracy with liberal use of CT scanning in the workup of suspected appendicitis. CT lowered the rate of negative appendicectomies from 19 to 12 % in one study and the incidence of negative appendicectomies in women from 24 to 5% in another.[8] Sensitivity is 96% and the specificity is 98-99%.

Initial studies evaluated sequential (non helical) CT in the diagnosis of appendicitis. In 1993, Malone evaluated 211 patients with non enhanced, sequential CT and reported a sensitivity of 87% and a specificity of 97%. The addition of IV and oral contrast agent increases sensitivity to 96-98% but increases cost to approximately $900. Sequential CT with oral and IV contrast enhancement is highly accurate but time consuming and expensive; it is best used for equivocal presentations when helical CT is not available.

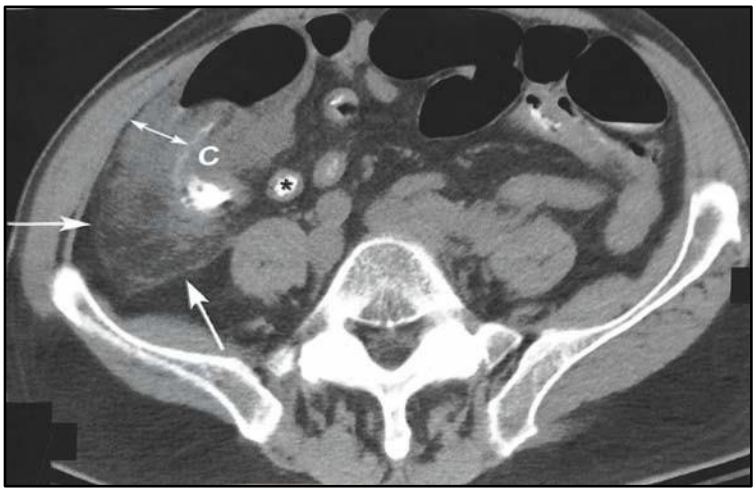


Figure 13: Contrast CT- Showing inflamed Appendix arrows (single headed) pointing to Abscess

Advantages of CT scanning include its superior sensitivity and accuracy compared with those of other imaging techniques, ready availability, non-invasiveness, and potential to reveal alternative diagnosis.

Disadvantages of CT scanning are radiation exposure, potential for anaphylactic reaction if intravenous (IV) contrast agent used, lengthy acquisition time if oral contrast is used, expensive and patient discomfort if rectal contrast is used, also it cannot be used in pregnancy.

Magnetic Resonance Imaging (MRI)

Although it is firmly established as the imaging modality of choice for the central nervous system and musculoskeletal system, MRI evaluation of the acute abdomen has not enjoyed similar widespread use. Incesu et al in a study on 60 patients have found that MR imaging is superior to sonography in revealing suspected acute appendicitis.[43] They have concluded that MR imaging can be used after sub- optimal or non-diagnostic sonography in suspected cases.

Radioactive isotope imaging

Patient's leukocytes can be labelled or tagged with a radioactive isotope. After reinjection, these leucocytes are detected on scanning an inflamed appendix. Technetium-99m and Indium-III have been used. Sensitivity is 83 to 89 percent and specificity is 92 to 100 percent.[44] The method was shown to be unreliable in diagnosing appendicitis in women. So it may need to be supplemented with an ultrasound scan to exclude gynecological disease. Limitations being not widely available and expensive.

Diagnostic Laparoscopy

Although directly visualising the appendix might be a valid method of determining acute appendicitis, patients with a normal appendix would be exposed to the risks and costs of general anaesthesia and diagnostic laparoscopy.[45] For this reason, it is not preferred as a diagnostic tool. During diagnostic laparoscopy for suspected acute appendicitis, therapeutic procedures can also be done, also if no other pathology is identified, the appendix is removed regardless of gross appearance.

Scoring System

- Despite advances in other diagnostic modalities, appendicitis remains a diagnosis based primarily on history and physical examination. In order to reduce the negative appendectomy rates various scoring systems have been developed for supporting the diagnosis of acute appendicitis.[33,46]

Alvarado score[47] is a 10-point scoring system was based on sophisticated statistical analysis of symptoms, signs and laboratory data on 305 patients admitted to Nazareth Hospital in Philadelphia from 1975 to 1976. Studies have shown that Alvarado score has diagnostic accuracy of around 88%.

The Alvarado score was modified by Kalan et al.[48] by excluding one laboratory finding – shift to left of neutrophil maturation i.e., score 1, as this is not routinely available and therefore, patients were scored out of 9 instead of 10.

Table:2 Interpretation of the Modified Alvarado score

Characteristic	Score
M=migration of pain to the RLQ	1
A=anorexia	1
N=nausea and vomiting	1
T=tenderness in RLQ	2
R=rebound pain	1
E=elevated temperature	1
L=leukocytosis	2
Total	9

Score 1-4: Acute Appendicitis very unlikely, keep for observation.
Score 5-6: Acute Appendicitis may be present, regular observation.
Score 7-9: Acute Appendicitis probable, operate.

In his original paper, Alvarado recommended an operation for all patients with a score of 7 or more and observation for patients with scores of 5 or 6. Alvarado included eight predictive factors. A high score was found to be an easy and satisfactory aid to early diagnosis of acute appendicitis in children and men, but had a high false-positive rate in women.[47]

C- Reactive Protein

C-reactive protein (CRP) has been a measure of acute phase reactions to inflammation for the last 15 years. Recently improved highly sensitive and standardized quantitative assays in serum and cerebrospinal fluid, have allowed a re-evaluation of its potential as a diagnostic laboratory.

C-reactive protein is the first protein to be discovered which behaves as an acute phase reactant. It has been named for its calcium-dependent interaction with the somatic C-polysaccharide of pneumococci.

The discovery of C-reactive protein was reported in 1930 by Tillet and Francis.[49] They were investigating serological reactions in pneumonia with various extracts of pneumococci and observed that a non-type-specific somatic polysaccharide fraction, which they designated fraction C, was precipitated by the sera of acutely ill patients. After the crisis, the capacity of the patients sera to precipitate C-polysaccharide rapidly disappeared, and the C-reactive material was not found in the sera from normal healthy individuals.

Lofstrom G (1944) independently described a non-specific capsular-swelling reaction of some strains of pneumococci when mixed with acute-phase sera and subsequently showed that the substance responsible was C-reactive protein. He detected C-reactive protein in non-infectious as well as infectious conditions – and the acute-phase reaction, in which the concentration of certain plasma proteins increase, is now recognized as a general and non-specific response to most forms of infective and non-infective inflammatory processes, cellular and /or tissue necrosis and malignant neoplasia.

Structure of C-reactive protein[50]

CRP is a cyclic pentameric protein composed of five non-covalently bound, identical 23.5 kDa subunits, arranged in a doughnut-shaped polymer. The main function of this pentamer is related to the ability to bind biologically significant ligands in vivo. The human C-reactive protein molecule (Molecular weight – 1,05,500 Da) is composed of five identical nonglycosylated polypeptide subunits (each of mass 23027 Da), with each subunit containing 206 amino acid residues. The promoters are non-covalently associated in an annular configuration with cyclic pentameric symmetry.

Each promoter has the characteristic 'lectin fold', composed of a two- layered □-sheet with flattened jelly roll topology. The ligand binding site, composed of loops with two calcium ions bound 4 A□□apart by protein side-chains, is located on the concave face. The other face carries a single □-helix. The pentraxin family is named for its electron micrographic appearance from the Greek penta (five) ragos (berries).

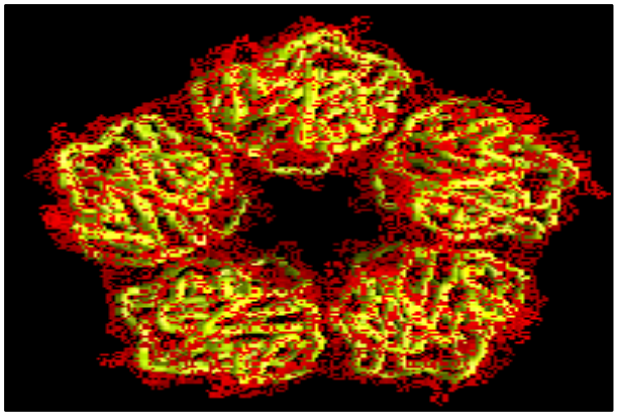


Figure 13: Human C-reactive protein 3D structure

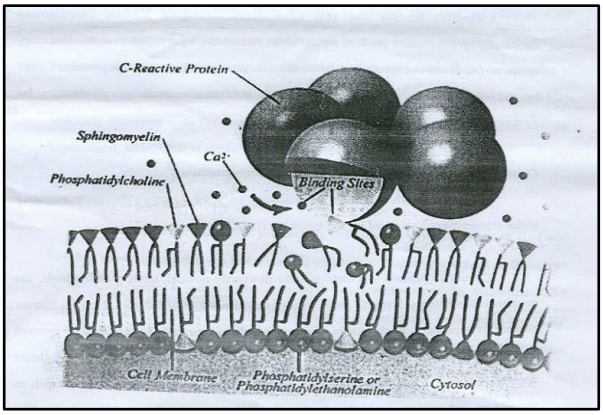


Figure 14: Pentameric structure of CRP

Functional properties:[51,52,53]

C-reactive protein has calcium interaction with the somatic C-polysaccharide of pneumococci, wherein it recognizes phosphocholine residues. It also binds to other substances which contain phosphocoline, including phospholipids, some plasma lipoproteins and plasma membranes of damaged or apoptotic, but not intact cells. In addition, C-reactive protein binds specifically to small nuclear ribonucleoprotein particles when they are exposed in dead or damaged cells.

The function of CRP is related to its role in the innate immune system. Similar to immunoglobulin IgG, it activates complement, binds to Fc receptors and acts as an

opsonin for various pathogens. Interaction of CRP with Fc receptors leads to the generation of proinflammatory cytokines that enhance inflammatory response. Unlike IgG, which specifically recognizes distinct antigenic epitopes, CRP recognizes altered self and foreign molecules based on pattern recognition. Thus, CRP is thought to act as a surveillance molecule for altered self and certain pathogens. This recognition provides an early defense and leads to a proinflammatory signal and activation of the humoral, adaptive immune system.

CRP binds to molecular groups found on a wide variety of bacteria and act as an opsonin. A number of functions have been ascribed to CRP, including initiation of opsonization and phagocytosis and activation of complement, neutrophils, monocytes and macrophages.

C-reactive protein can also have tissue damaging effects. Complement activation by C-reactive protein exacerbates ischemic injury, the pro-inflammatory actions of C-reactive protein and its binding to phospholipids and lipoproteins may be pro-atherogenic. Also its capacity to stimulate tissue factor production by macrophages may be pro-atherogenic.

C-reactive protein synthesis and its serum concentration

Plasma C-reactive protein is produced only by hepatocytes, predominantly under transcriptional control by the cytokine IL-6.[51,52]

In man, the only CRP gene coding sequence is found on Chromosome 1. CRP is synthesized by the liver. Trace amounts of mRNA for CRP have been found in other cells, it is not known of the importance of locally produced CRP. Synthesis of CRP and other acute phase proteins by hepatocytes is modulated by cytokines. Interleukins 1b and

6 and tumour necrosis factor are the most important regulators of CRP synthesis. After stimulation with IL-6, IL-1b, TNF and INF, the hepatocytes receive signals to start transcription of DNA coding for CRP.

CRP begins to rise in bacterial infections within 4-6 hours, peaks at 36-50 hours, closely parallels acute response with 4-7 hour half-life, and normalizes 3-7 days after the stimulus is withdrawn.

C-reactive protein is a trace protein in overtly normal, healthy individuals, the median value being 0.8 mg/L, with an interquartile range of 0.3 to 1.7mg/L. Ninety percent of apparently healthy subjects have levels of less than 3 mg/L and 99 percent less than 10 mg/L. The values increase with age, with median CRP level approximately doubled with age, from □1mg/L in the youngest decade to □2mg/L in the oldest and tend to be higher in females. Serum levels are lower in healthy newborns, but reaches adult levels within a few days.

Following an acute-phase stimulus, C-reactive protein values may increase from less than 50□g/L to more than 500mg/L,that is 10,000-fold. De-novo hepatic synthesis starts very rapidly after a single stimulus, serum concentrations rising above 5mg/L by about 6 hours and peaking around 45 hours. The plasma half-life of C-reactive protein is about 19 hours and is constant under all conditions of health an disease, so that the sole determinant of circulating CRP concentration is the synthesis rate, which thus directly reflects the intensity of the pathological process stimulating CRP production. When the stimulus for increased production completely ceases, the circulating CRP concentration falls rapidly at almost the rate of plasma CRP clearance.

Because CRP levels are stable over long period of time, are not affected by food intake and demonstrate almost no circadian variation, there is no need to obtain fasting samples for CRP measurement.

The only physical condition which seriously interferes with the capacity to intercept CRP levels is serious hepatocellular impairment, since CRP is synthesized exclusively in the liver. Other factors known to effect CRP are smoking, obesity, patients on HRT and oral contraceptive pills. Aspirin and Statin therapy is known to reduce CRP levels in the serum probably explaining their direct anti-inflammatory effects.

All acute inflammatory processes (infectious and non-infectious) and certain malignant conditions result in rise in serum CRP as a non-specific phenomenon. CRP production is a non-specific response to disease and it can never, on its own, be used as a diagnostic test. However, if CRP results are interpreted in the light of full clinical information on the patient, then it can provide exceptionally useful information. On further serial measurements, important information about the resolution or continuation of the inflammation process can be obtained.

Routine clinical uses of CRP measurement

- ***Screening test for organic disease***
- ***Assessment of disease activity in inflammatory conditions:***

1. Juvenile chronic (rheumatoid) arthritis, Rheumatoid arthritis, Ankylosing spondylitis, Reiter's disease, Psoriatic arthropathy
2. Vasculitides - Behcet's syndrome, Wegner's granulomatosis, Polyarteritis Nodosa, Polymyalgia rheumatica

3. Crohn's disease, Rheumatic fever, Familial Mediterranean Fever, Acute Pancreatitis

- ***Diagnosis and management of infections***: Bacterial endocarditis, Neonatal speticaemia and meningitis, Systemic Iupus erythematosus, leukemia and its treatment, operative complications including infection and thromboembolism.
- ***Differential diagnosis/classification of inflammatory disease***: Serum lupus erythematosus Vs rheumatoid arthritis, Crohn's vs ulcerative colitis, Predictor of cardiovascular event.
- ***Detection and management of intercurrent infection***: CRP levels are elevated in bacterial and protozoal infections, neonatal sepsis.[54]

LABORATORY METHODS OF MEASURING CRP:

Specimen collection and preparation

1. The serum or whole blood specimen should be collected under standard laboratory conditions.
2. Patient samples perform best when tested immediately after collection. The blood specimen must be tested within 24 hours. If the serum sample cannot be tested within 24 hours, it must be frozen until the test can be performed. Allow sample to reach room temperature before proceeding.

3. Sodium azide can be added as a preservative up to 0.1% without affecting results.

- **Latex Agglutination Assay**

Traditional methods for measuring CRP include precipitation and agglutination assays. The latex agglutination assay is a qualitative test with a detection limit of

approximately 10 mg/litre (normal upper limit). Because CRP levels can increase so rapidly and dramatically, the latex agglutination assay is subject to false-negative reactions due to a prozone-type phenomenon in which all of the antibody combining sites on the latex particles are bound to an excess of CRP, so no cross-linking (agglutination) can occur. Consequently, the qualitative tests should be performed on several dilutions of serum to avoid negative reactions. If several dilutions are formed, the latex agglutination method can easily be converted to a semi-quantitative assay so distinctions can be made between levels of positivity (e.g. less than 50 mg/litre and more than 150 mg/litre). Such semi-quantitative distinctions would be very useful to the clinician trying to distinguish between bacterial (high CRP levels) and viral infections (normal to slightly elevated CRP).

- **Immunoassays**

Highly specific antibodies to CRP permit the development of rapid, specific, and very sensitive assays for this protein. These newer immunoassays include laser nephelometry (the most popular method), RIA, and enzyme immunoassays and have created a renewed interest in CRP testing in a variety of clinical settings. Recently, instrument manufacturers have developed assay systems that allow random access assays for CRP to be performed virtually on demand with 10 to 20 minutes turn- around-time.

- **Ultra-sensitive or High-sensitivity (hs) CRP Assay**

An ultra-sensitive immunoturbidimetric assay has been developed for CRP. The new assay measures the increased turbidity resulting from antibody-antigen complexes formed when sample and antibody reagent is mixed. The assay has sensitivity of 0.1 mg/L. The ready-to-use liquid reagents can be placed directly on a chemistry

analyzer and will yield precise results in minutes.

- **Factors that affect results**

As in all serological tests, haemolytic, lipemic or turbid sera may cause incorrect results and should not be used. Drugs that may cause false-positive results include oral contraceptives. Drugs that may cause false-negative results due to suppression of inflammation include NSAIDs, steroids and salicylates. The presence of intrauterine device may cause inflammation, which produces a positive test. Overnight refrigeration of the sample may produce a false-positive result.

- **Expected Values**

It is recommended that each laboratory establish its own normal range based on patient population. However, based on published literature, healthy individuals are expected to have CRP values as follows:

- Neonatal serum: 0.01 to 0.35 µg/mL
- Adult serum: 0.07 to 8.00 µg/mL

MANAGEMENT[14,10,22]

"The earlier the operation, the lower the mortality"

— Murphy JB

Early diagnosis and prompt surgical treatment are still most important principles in dealing with acute appendicitis and this applies to patients of all age groups.

PRE-OPERATIVE

A few hours and not more than 6 hrs is set aside for pre-operative workup. Clinical examination, laboratory investigations and radiological examination is followed by the below measures:

- Patient is kept nil orally
- Parenteral fluid therapy to maintain fluid and electrolyte balance,
- Analgesics to relieve pain and anxiety,
- Shaving and preparing parts
- Pre-anesthetic evaluation

OPERATIVE PROCEDURE

ANAESTHESIA

General or spinal anaesthesia can be administered.

INCISION

Experience should enable the surgeon to determine with a fair degree of accuracy before operation, the position and pathological changes in the appendix and hence choose an appropriate incision.

a) Grid-iron incision: This was first described by McArthur though it is

popularly known as McBurney's incision. The incision is made at the centre of the the McBurney's point i.e, right angles to a line joining lateral 1/3rd and medial 2/3rd from the right anterior superior iliac spine to the umbilicus. The external oblique is incised along the line of the incision. The fibers of internal oblique and transversus abdominis are separated, and after suitable retraction, peritoneum opened. This incision is said to be associated with the lowest complication rate. It can be converted into a Flower- Weir incision, by extending inward through the rectus.

b) Rutherford-Morrison incision: It is an oblique muscle-cutting incision with its lower end at McBurney's point and extending obliquely upwards and laterally as necessary. All layers are divided in the same line. This incision is useful if the appendix is para or retrocaecal and fixed.

c) Lanz's incision: This is a small transverse incision put 1 inch medial and above the anterior superior iliac spine and extending up to the lateral border of the rectus sheath. Thereafter, the muscles are split as in grid-iron incision. The method has a definite cosmetic value, but extending the incision if necessary, proves difficult.[53]

d) Battle's incision: Battle, in 1895, described an incision of variable length in the right semilunar line. This involves the rectus medially. The inferior epigastric vessels are easily avoided, but the vertical peritoneal incision is limited to about 2½ inches and damage to the segmental nerves is to be avoided.

e) Right Lower Paramedian incision: It is a vertical incision lying parallel to and 1.25 to 2.25 cm to the right of the midline. It commences 2.5 cm below the level of the umbilicus and ends just above the pubis. The anterior rectus sheath is incised in the

line of the incision and the rectus muscle retracted laterally. Transversalis fascia and peritoneum are incised together, the peritoneal cavity being opened through the length of the incision, taking care not to injure the bladder inferiorly.

Advantages: It gives good access to the pelvic organs in the female and if necessary, it can be readily extended upwards, to deal with a perforated duodenal ulcer or other unexpected intra-abdominal pathology.

Disadvantage: The organ is often comparatively inaccessible to this approach.

REMOVAL OF APPENDIX

After opening the abdomen, the wall along with the peritoneum is lifted up. After removing pus or serous exudate with a sucker, a pack is inserted to the wound on the medial side. Using a swab, the caecum is withdrawn. A finger may be inserted into the wound on the medial side to aid delivery of the appendix. Once the appendix has been delivered, the caecum is grasped by the assistant. A tissue holding forceps is applied around the appendix. The base of the mesoappendix is clamped with a hemostat, tied and severed.

Appendix now completely freed and is crushed near its junction with the caecum in a hemostat, which will be removed and reapplied just distal to the crushed portion. A catgut ligature is tied around the crushed portion close to the caecum and an atraumatic catgut purse-string suture is applied to the caecum about 1.25 cm from the base. Stitch passes through the muscle coat especially picking up the taenia coli. It is left untied until the appendix has been amputated with a scalpel below the hemostat. The stump is invaginated while purse-string tied, thus burying the appendix stump. Haemostasis shold be confirmed and abdomen closed in layers.

LAPAROSCOPIC APPENDECTOMY[55]

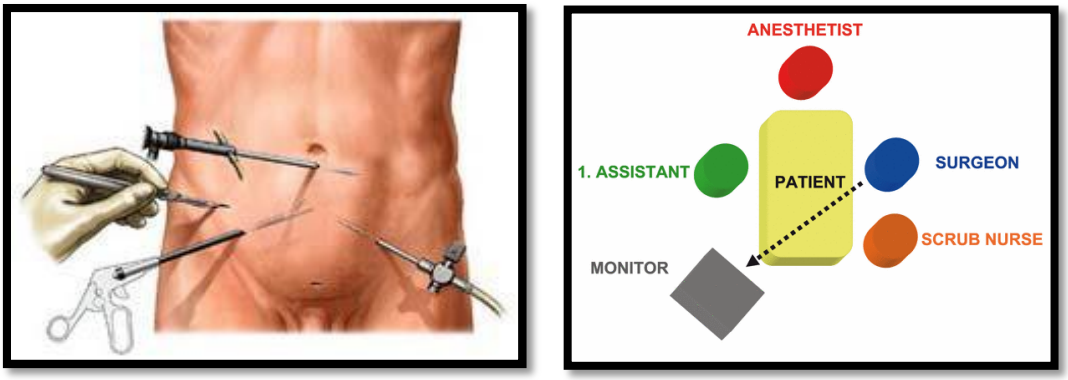


Figure 16

The surgeon typically stands on the left of the patient, and the assistant stands on the right. The anesthesiologist and the anesthesia equipment are placed at the patient's head, and the video monitor and instrument table are placed at the feet.

Although some variations are possible, 3 ports are placed during the procedure with three trocars placed in triangular formation. The optical trocar is generally a 10/11mm trocar placed in the periumbilical position. Two operating trocars are placed ideally at a minimum of 8 to 10cm from one another. One operating trocar (5 or 10/11mm) is placed in the right iliac fossa and another operating trocar (5 or 10/11mm) is placed in left iliac fossa position. Two of them have a fixed position (i.e., periumbilical and left iliac fossa). The third trocar placed may vary greatly depending on the patient's anatomy either in in the right iliac fossa or suprapubic region .

According to the preferences of the surgeon, a short umbilical incision is made to allow the placement of a Hasson cannula or Veress needle that is secured with 2 absorbable sutures.

Pneumoperitoneum (10-14 mm Hg) is established and maintained by

insufflating carbon dioxide. Through the access, a laparoscope is inserted to view the entire abdomen cavity.

The procedure begins with an exploration to confirm the diagnosis of acute appendicitis. If acute appendicitis is confirmed, any adhesions between the appendix and the peritoneal wall are divided to expose the appendix from its tip to its base. The appendix is grasped and retracted upward to expose the mesoappendix. The mesoappendix is divided using a dissector inserted. Then, a linear Endostapler, Endoclip, or suture ligature is passed to ligate the mesoappendix. The mesoappendix is transected using a scissor or electrocautery. To avoid perforation of the appendix and iatrogenic peritonitis, the tip of the appendix should not be grasped.

The appendix may now be transected with a linear Endostapler, or, alternately, the base of the appendix may be suture ligated in a similar manner to that in an open procedure. The appendix is now free and may be removed through the periumbilical trocar using a laparoscopic pouch to prevent wound contamination. Peritoneal irrigation is performed with antibiotic or saline solution. Completely aspirate the irrigant. The trocar sites are then removed and the pneumoperitoneum is reduced.

The fascial layers at the trocar sites are closed with absorbable suture, while the cutaneous incisions are closed with interrupted subcuticular sutures or sterile adhesive strips.

POST-OPERATIVE MANAGEMENT

- Oral feeds are withheld till the bowel sounds return and flatus is passed.
- IV fluids and electrolytes are given till oral feeds are allowed.
- Broad spectrum antibiotics are given to cover against mixed intestinal flora, till culture report of the peritoneal exudate is obtained.
- TPR chart is maintained.
- Analgesics and sedatives.
- Drain if placed in the peritoneal cavity, is removed by 24-48 hrs.
- Sutures removed by 7-10 days.

COMPLICATIONS OF APPENDICECTOMY[14]

Most of the complications are not peculiar to appendicectomy, but occur with any abdominal surgery.

Early Complications

1) Haemmorhage

2) Diffuse peritonitis

3) Pulmonary complications

4) Neurogenic or adhesive ileus

5) Retention of urine

Intermediate Complications

1. Secondary or residual abscess
    - Pelvic
    - Paraceacal
    - Perinephric
    - Subdiaphragmatic

2. Wound infection: the commonest, especially in a complicated appendicitis
3. Pyelophlebitis
4. Femoral or Iliac vein thrombosis
5. Parotitis
6. Persistent sinus or fistula
7. Rupture of caecal wall

Late Complications

1. Incisional hernia
2. Right sided indirect inguinal hernia
3. Intestinal obstruction

The treatment of complications should be done as and when required, by early recognition and skillful surgical intervention or conservatively as required.

PROGNOSIS

Simple appendectomy in uncomplicated acute appendicitis still carries a mortality rate approaching 0.2%. Regardless of the phase of the disease, the overall mortality of the primary appendectomy is appreciably under 1%. The average hospital stay approximates 3 days for Simple appendectomy. But complications of gangrene and perforation extend the average stay to 7 days.

Anesthesia, age, infirmity and associated disease influence the outcome with respect to both morbidity and mortality.

Improved surgical techniques, antimicrobials, nasogastric intubation and decompression, pre and post operative fluid and electrolyte replacement, and the application of supportive aids in recovery and intensive care units have contributed appreciably to the reduction in morbidity and mortality from the complications inherent in

delayed diagnosis.

CRP, WBC AND DIFFERENTIAL COUNT IN ACUTE APPENDICITIS

Gurleyik et al.[63] compared serum CRP study of 108 patients suspected of having appendicitis on clinical grounds. The false-negative rate of CRP was 3% and the false-positive rate was 11%. CRP levels were true (positive or negative) in the remaining 103 patients. On the other hand, the diagnosis depending on surgeon's clinical impression was true in 90 patients and false in 18 patients. This difference was statistically significant (p = 0.0035). The sensitivity, specificity, and accuracy of serum CRP measurements were calculated as 93.5, 80, and 91 percent, respectively. They recommend CRP measurement as a routine laboratory test in patients with suspected diagnosis of acute appendicitis as it supports surgeons clinical diagnosis.

Gronroos JM, Gronroos P[55] in a retrospective study studied the preoperative leucocyte counts and Creactive protein (CRP) values in three groups of patients operated on for a clinical suspicion of acute appendicitis. They concluded that acute appendicitis is very unlikely when both the leucocyte count and CRP value are normal.

Shakhatreh HS[57] in a prospective study involving 98 patients. The diagnosis of acute appendicitis was histopathologically confirmed in 89 patients (91%), while 9 normal appendixes (9%) were removed. CRP levels were true (positively or negatively) in 93 patients and it was false positive in one patient (11%) and false negative in 4 patients (4%). On the other hand, the clinical diagnosis was correct in 89 cases (91%) and false in 9 cases (9%), the difference is statistically significant (p value = 0.009).This study conclude that CRP is very helpful in the diagnosis of acute appendicitis, but it doesn't replace the clinical skills of general surgeons.

Asfar S et al.[56] conducted a double blind trial in 78 patients to study the impact of a normal (rather than raised) serum C-reactive protein in reducing the rate of negative explorations. White blood count (WBC), CRP and the histopathology findings were correlated. In patients with histopathologically proven acute appendicitis both the WBC count and serum CRP level were significantly raised ($p=0.025$ and $p<0.000$ respectively). Serum CRP level was normal in 13 out of 15 negative explorations (normal appendix on histopathology). The specificity and sensitivity of serum CRP was 86.6% and 93.6%, respectively. They concluded that a normal pre-operative serum CRP measurement in patients with suspected acute appendicitis is most likely associated with a normal appendix. Deferring surgery in this group of patients would probably reduce the rate of unnecessary appendicectomies.

In a study by Svend Dueholm et al.,[59] the diagnostic value of C-reactive protein (CRP), total white blood cell (WBC) count, total neutrophil count, and neutrophil differential count were evaluated in a prospective blinded study of 204 patients submitted with the tentative diagnosis of acute appendicitis. WBC count demonstrated the best sensitivity (83 percent) and predictive value of a negative result (88 percent). Combining the tests by an "or" rule enhanced the sensitivity to 100 percent. It was concluded that both single tests and combined tests are of limited value in predicting acute appendicitis. However, the triple test combination proved a predictive value of a negative result at 100 percent (95 percent confidence limits 92 to 100 percent), indicating that acute appendicitis is unlikely when these tests are simultaneously negative. Therefore, the triple test is recommended as a help in reducing the significant rate of negative laparotomies in patients suspected of having acute appendicitis.

Yang et al.[58] studied the role of leukocyte count, neutrophil percentage and C-reactive protein in the diagnosis of acute appendicitis in the elderly and concluded that patients with normal results in all these tests were highly unlikely to have acute appendicitis and should be evaluated with extra caution before surgery.

Oosterhuis WP, Zwinderman AH et al.[61] conducted a study in 209 patients, to find out if the C reactive protein concentration is of any value in the diagnosis of acute appendicitis, either alone or in combination with other laboratory test. It concluded that C reactive protein concentration of 6 mg/1 alone had a sensitivity of 87% and a specificity of 50%, measurement of the C reactive protein concentration can increase the accuracy in the diagnosis of acute appendicitis.

Khan MN et al.[70] carried out a study to find out the specificity and sensitivity of white cell count (WCC) and C-Reactive Protein (CRP) in diagnosing appendicitis in 259 patients presenting with right iliac fossa pain. A total of 259 patients were included in this study and out of them 37 had a normal appendix giving an over all negative appendicectomy rate of 14.3%. Out of these 11 were male and 26 were female, male to female ratio being 1:2.3. The sensitivity and specificity of WBC in this study was 83% and 62.1 % and that for CRP was 75.6% and 83.7 %. : Both the inflammatory markers i.e. WBC and C-reactive protein can be helpful in the diagnosis, when measured together as this increases their positive predictive value

Erikson et al. (1994)[69] measured serum CRP level and WBC count every four hours in a cohort of 227 patients with suspected acute appendicitis, and reported that it was unusual to find a normal CRP level after 8 hours of observation in the presence of acute appendicitis. If these test results are normal, the surgeon should preferably refrain from

operating and consider other differential diagnosis. The positive and negative predictive values (96.7% and 76.5%, respectively) of serum CRP was reported in the current study.

In a study conducted by Eugene Albu et al.[71] published in journal of diseases of colon and rectum that serum C-reactive protein was measured in 56 patients hospitalized with a suspected diagnosis of acute appendicitis. Based on these determinations, it is concluded that an increase in C-reactive protein levels to more than 2.5 mg/dl is not a definite indicator of acute appendicitis. However, if the C- reactive protein level in blood drawn 12 hours after the onset of symptoms is less than 2.5 mg/ dl, acute appendicitis can be excluded.

Thimsen et al.[65] studied 70 suspected cases of acute appendicitis and concluded that a normal CRP value in a patient presenting with symptoms for more than 12 hours, does not have acute appendicitis and can be followed in an outpatient setting.

Al Saigh,[66] after measuring serum C-reactive protein quantitatively before surgery in 189 patients undergoing appendicectomy assessed that CRP had a high specificity (76.3%) but had a sensitivity of only 39.7%.

Davies et al.[67] conducted a study on 60 patients with right iliac fossa pain, CRP and full blood counts were performed. In his study, 94% of patients had raised CRP with acute appendicitis and 83% of patients had negative CRP results with negative appendictomies.

Marchand et al.,[68] studied 106 patients admitted to the emergency room with a tentative diagnosis of acute appendicitis and who subsequently underwent appendectomy. They concluded that the cytochemically determined neutrophil count, when greater than the upper limit of the reference interval of either 75% or 7.88x 109/L, and the total white

blood count greater than the upper limit of reference interval of 10.5x109/L were single best tests for diagnosis of acute appendicitis with the highest sensitivities of all tests examined (81-84%). The manual differential count and C-reactive protein showed significantly lower sensitivities. They also suggested that the combination of these tests has 100% sensitivity and 50% specificity in diagnosis of acute appendicitis.

Verma et al.[32] measured C-reactive protein in 42 cases admitted to a general hospital with suspected acute appendicitis. Thirty five were operated and thirty one of these with raised CRP had an inflamed appendix. Four cases with normal CRP value had scarred appendix (healed appendicitis) which was confirmed by biopsy reports. These four cases also had normal white blood cell count and ESR.

Mikaelsson, Arnbjornsson[70] studied the clinical usefulness of preoperative determination of C-reactive protein (CRP) in patients with suspected acute appendicitis in 156 patients undergoing appendectomy. CRP values were found to increase with an advancing stage of the appendicitis found at operation and the length of the preoperative phase of illness.

A multivariate analysis by Oosterhuis et al.[61] showed that serial CRP measurement can improve the accuracy of diagnosing acute appendicitis. Other reports did not support this view.

According to David Berchley,[78] when used individually, both the absolute and categorical WBC and NC distinguish normal appendices from acute appendicitis, though they do not distinguish uncomplicated from complicated appendicitis. Neither do they individually predict abscess when used as absolute or categorical variables. CRP has no definite value for predicting acute appendicitis in either its absolute or categorical forms,

though a significantly elevated level is strongly suggestive of abscess. In terms of excluding appendicitis in the patient, the inflammatory markers were less effective. Normal values for WCC, NC and CRP excluded appendicitis. Laboratory tests of the white cell count, neutrophil count and C-reactive protein are more effective in supporting a clinical diagnosis of acute appendicitis in patients with typical clinical features than in excluding the diagnosis.

In a meta analysis of 22 articles by Hallan S, Asberg,[60] the aim of the study was to review the literature on the accuracy of C-reactive protein (CRP) in diagnosing acute appendicitis. The sensitivity ranged from 0.40 to 0.99, and the specificity from 0.27 to 0.90. The cut-off values for a positive test varied from 5 to 25 mg/l. The diagnostic accuracy of CRP tended to be a little inferior to that of total leukocyte count (13 studies).CRP is a test of medium accuracy in diagnosing acute appendicitis. However, definitive conclusions on the clinical usefulness of the test could not be drawn.

MATERIALS AND METHODS

SOURCE OF DATA:

This study was performed on 114 patients who have been clinically diagnosed to have Acute Appendicitis and who were posted for emergency appendicectomy in General Surgery Department of RL Jalappa Hospital attached to Sri Devaraj Urs Medical College, Tamaka, Kolar during the period from January 2014 to June 2015

METHOD OF COLLECTING DATA:

Sample size: Minimum of 60 study subjects of Acute Appendicitis

Sampling method: Simple random sampling

Inclusion criteria

All patients clinically diagnosed to have acute appendicitis and subjected to appendicectomy.

Exclusion criteria

1) Concomitant conditions where CRP or Leukocyte Count is elevated

eg. Rheumatoid arthritis, SLE, glomerular nephritis, gout, inflammatory bowel disease

2) Patient with appendicular mass, abscess or generalised peritonitis.

Clinical diagnosis of acute appendicitis was done in the Department of Surgery, based on symptoms of pain, migration, nausea and vomiting, anorexia, fever and signs of peritoneal inflammation like right iliac fossa tenderness, rebound tenderness and guarding. Once acute appendicitis was suspected, patient was subjected to routine investigations as per the hospital protocol. Urine microscopy was performed in all cases. Elderly patients were subjected to further investigations as part of pre-anaesthetic work up

including X-ray chest, ECG etc.

CRP, Total leucocyte count was done in all cases. WBC count of more than 10,000 cells/mm^3 was considered as Leukocytosis. Ultrasonography of abdomen was done in most of the cases to confirm diagnosis and rule out other causes of pain abdomen. CRP more than 6 mg/dl was considered to be positive. No special preparation of the patient was required prior to sample collection by approved techniques. When there was delay, the sample was stored at 2-8^{0}C. Maximum period of storage was 72 hours. Patients with strong suspicion of acute appendicitis were advised emergency appendicectomy. After obtaining consent, patients were operated and the appendicectomy specimen was sent for histopathological examination. The HP report was considered as the final diagnosis.

The patients were meticulously monitored in the post-operative period for any complications. All patients were followed up in the outpatient department for a period of two months. The case study was done as per a detailed proforma which is shown in the annexure. The hospital ethical committee clearance was obtained prior to undertaking the study.

Statistical Analysis

Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. The following assumptions on data is made, Assumptions: 1.Dependent variables should be normally distributed, 2.Samples drawn from the population should be random, Cases of the samples should be independent

Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups.

Statistical software: The Statistical software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1 ,Systat 12.0 and R environment ver.2.11.1 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc**.**

RESULTS

The present study was performed on 114 patients, clinically diagnosed to have acute appendicitis and who were posted for emergency appendicectomy in General Surgery Department of RL Jalappa Hospital attached to Sri Devaraj Urs Medical College, Tamaka, Kolar during the period from January 2014 to June 2015.

Apart from the routine investigations all the cases were subjected specifically to the following two investigation i.e. W.B.C. count and CRP, to evaluate their role in accurately diagnosing a case of acute appendicitis. All the cases were subjected for histopathological examination which was considered as gold standard to confirm the diagnosis. The following observations were made in the study.

AGE DISTRIBUTION

The age of the patients ranged from 8-65 years, with a mean age of 23.96 ± 9.56years.

N	Minimum	Maximum	Mean	Std. Deviation
114	8	65	23.96	9.564

Table 3: Age distribution

Age in years	No. of patients	%
<10	3	2.6
10-20	41	36.0
21-30	44	38.6
31-40	21	18.4
>40	5	4.4
Total	114	100.0

Out of 114 cases, most common presenting age group is 21-30 years- 44 cases (38.6%), followed by age group of 10-20 years- 41 cases (36%), and age group of 31-40 years- 21 cases (18.4%). The least number of patients were seen in the age group <10 years- 5cases (4.4%).

Graph 1: Age distribution

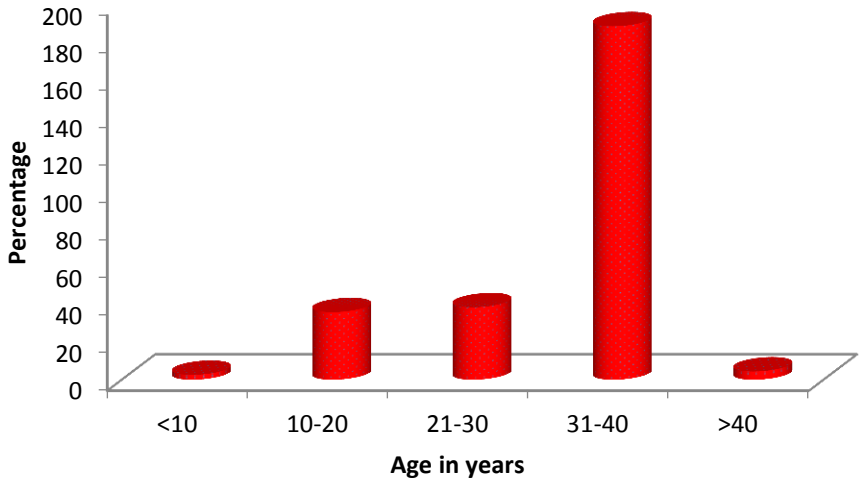


Table 4: Gender distribution

Gender	No. of patients	%
Female	48	42.1
Male	66	57.9
Total	114	100.0

In the present study, out of 114 cases, 48(42.1%) patients were females and the remaining 66 (57.9%) were males. The male to female ratio in the present study is approximately 1.3:1.

Graph 2: Gender distribution

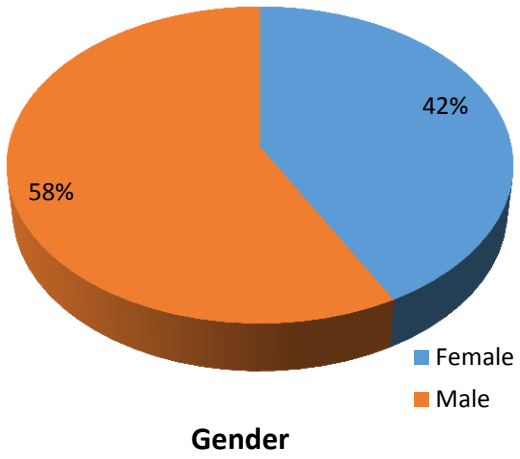


Table 5: Gender distribution in relation to age.

Age in years	Gender		Total
	Female	Male	
<10	2(4.2%)	1(1.5%)	3(2.6%)
10-20	18(37.5%)	23(34.8%)	41(36%)
21-30	16(33.3%)	28(42.4%)	44(38.6%)
31-40	11(22.9%)	10(15.2%)	21(18.4%)
>40	1(2.1%)	4(6.1%)	5(4.4%)
Total	48(100%)	66(100%)	114(100%)

In males, most common age group of presentation of acute appendicitis was between 21-30 years (42.4%), followed by the age group 10-20 years (34.8%).In females, most common age group was between 10-20 years (37.5%), followed by 21-30 years (33.3%).

Graph 3: Gender distribution in relation to age

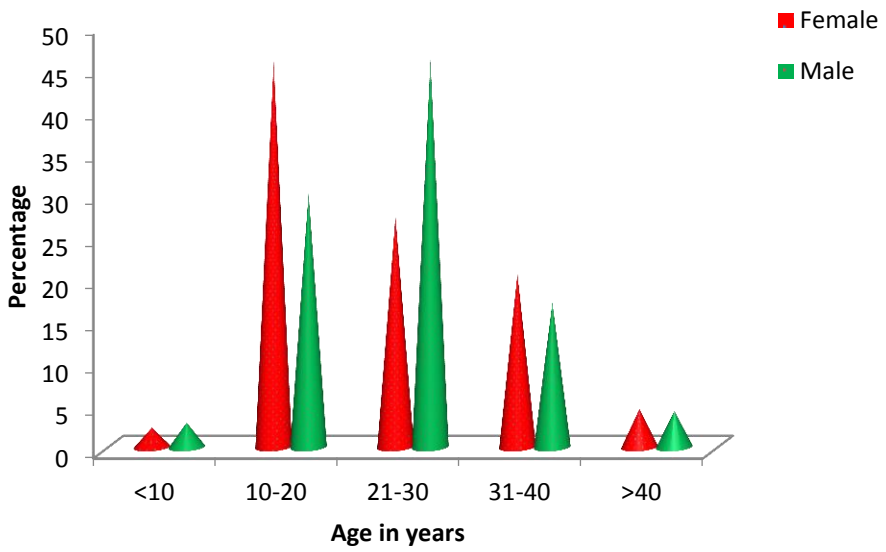


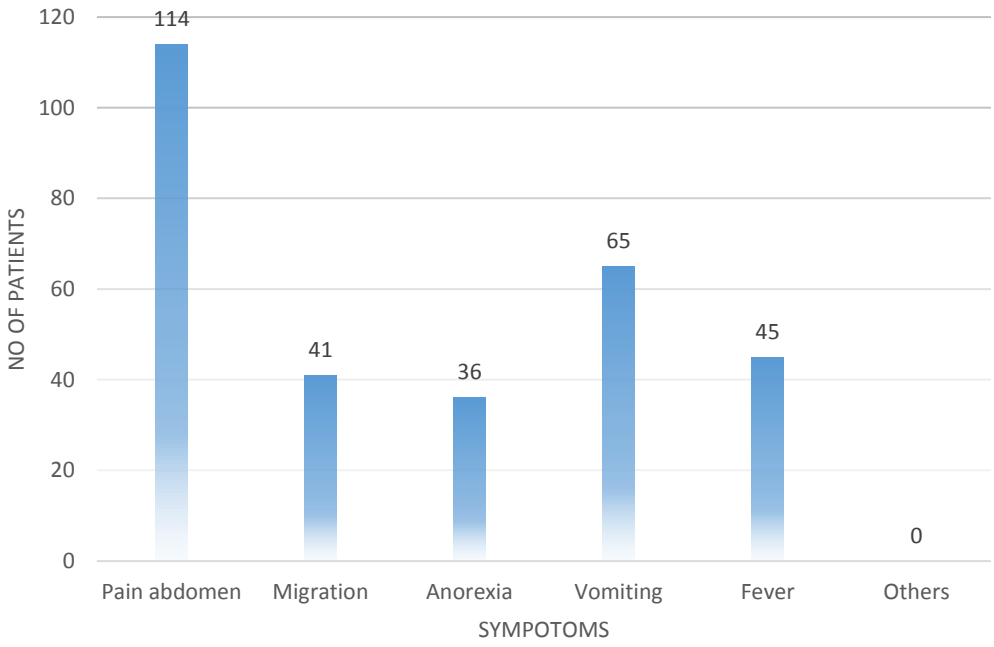
Table 6: Distribution of symptoms and signs

Symptoms	No. Of patients (n=114)	%	Signs	No. of patients (n=114)	%
Pain abdomen	114	100.0	RIF Tenderness	114	100.0
Migration of pain to RIF	41	36%	Rebound tenderness	37	32.45
Anorexia	36	31.57	Guarding	10	8.7
Vomiting	65	57.01	Rovsing's sign	25	21.9
Fever	45	39.46	Psoas sign	0	0.0
Others	0	0.0	Temp (>>99^0F - FB)	44	38.6
-	-	-	Tachycardia>90	16	14.1

Pain abdomen was the presenting complaint in all the cases in our study. 41 (36%) of them had migration of pain to the right iliac fossa. The next common symptom was vomiting in 65 (57.01%) subjects followed by fever in 45 (39.5%) subjects and anorexia in 36(31.6%) subjects.

Among clinical signs, right iliac fossa tenderness was present in all cases (100%), rebound tenderness was present in 37(32.45%) cases, guarding was present in 10(8.8%) of cases, which reflects severity of inflammation. Other peritoneal signs like Rovsing sign was elicited in 25(21.9%) cases.

Graph 4: Distribution of symptoms



Graph 5: Distribution of signs

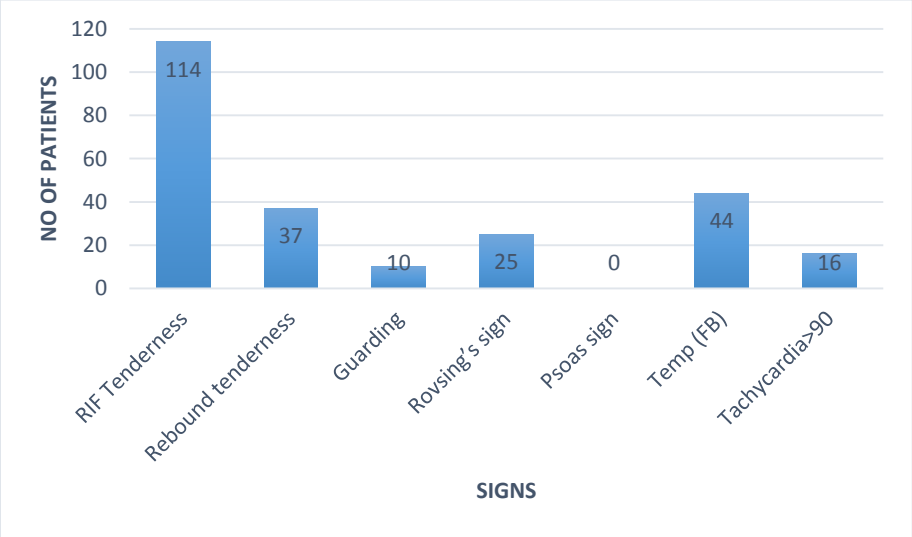
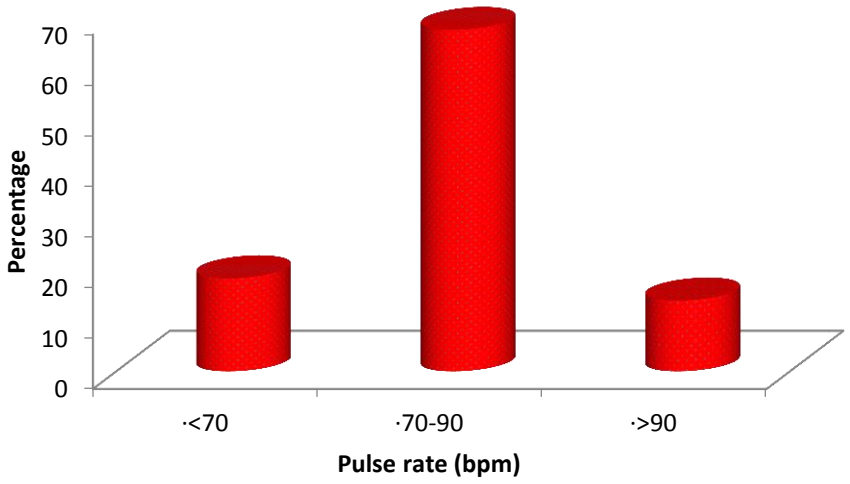


Table 7: Pulse rate and temperature

	Total (n=114)
Pulse rate (bpm)	
• <70	21(18.4%)
• 70-90	77(67.5%)
• >90	16(14%)
Temp >99^0F	
• AF	70(61.4%)
• FB	44(38.6%)

Out of 114 patients, only 16 (14%) patients shown to have tachycardia i,e >90bpm with majority of the patients(67.5%) having pulse rate between 70-90 bpm. 44 patients (38.6%) were febrile.

Graph 6: Pulse rate



Graph 7: Temperature

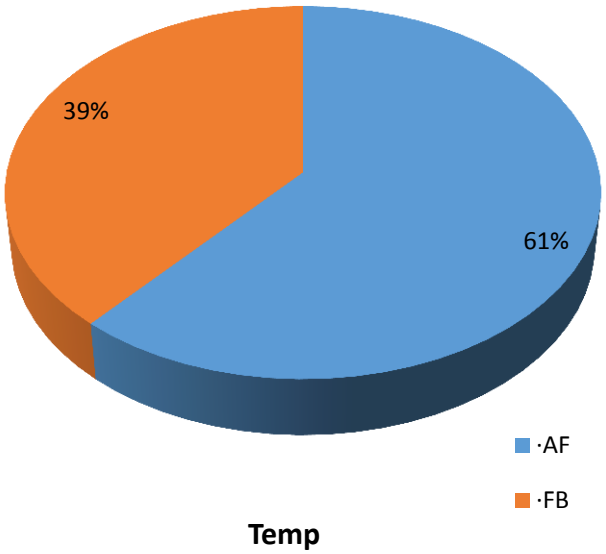


Table 8: TLC distribution

TLC	No. of patients	%
<10	47	41.2
>10	67	58.8
Total	114	100.0

Out of 114 cases, 58.8 % of the cases had leukocytosis with more than 10,000 T/cumm.

Graph 8: TLC distribution

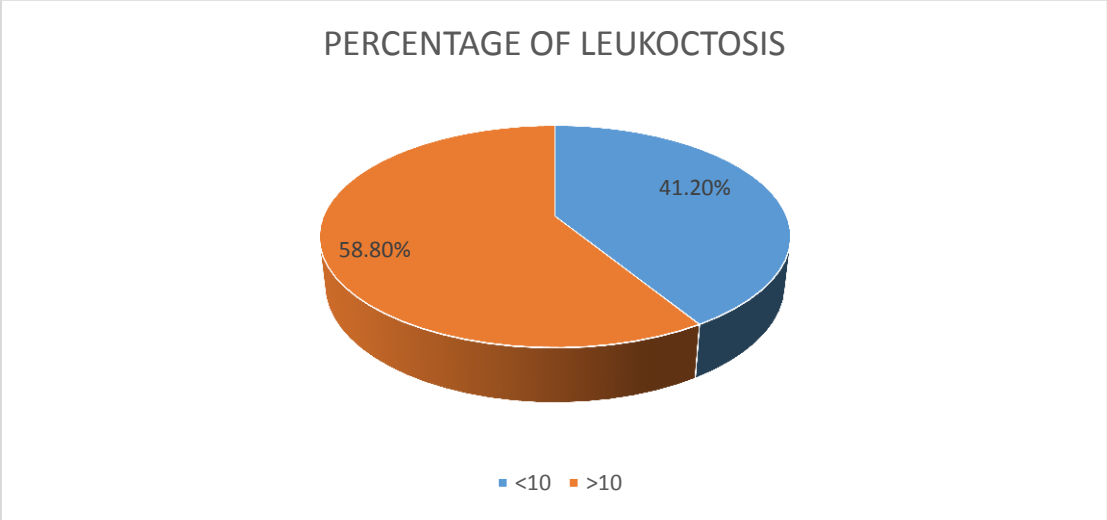
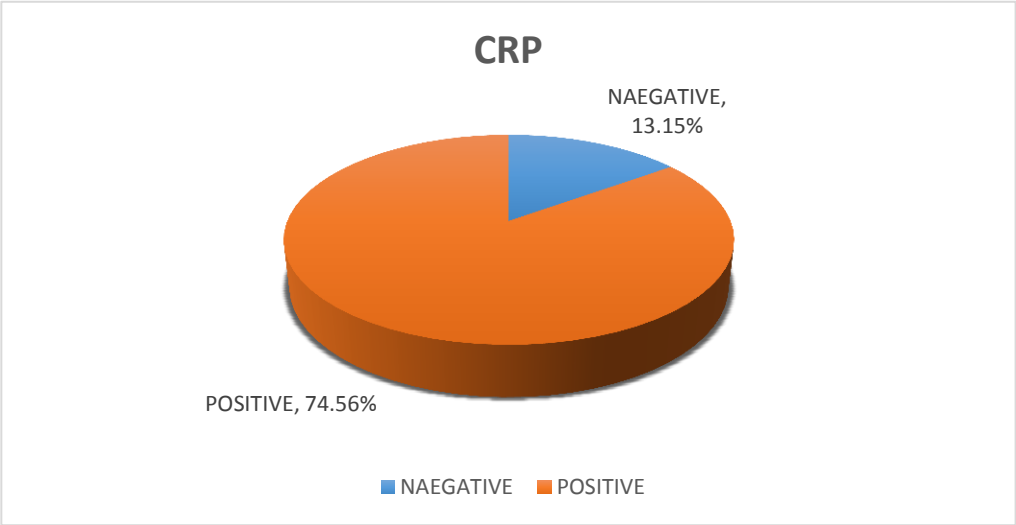


Table 9: CRP distribution

CRP	Total
Negative	15(13.15%)
Positive	85(74.56%)
Total	114(100%)

Graph 9: CRP distribution



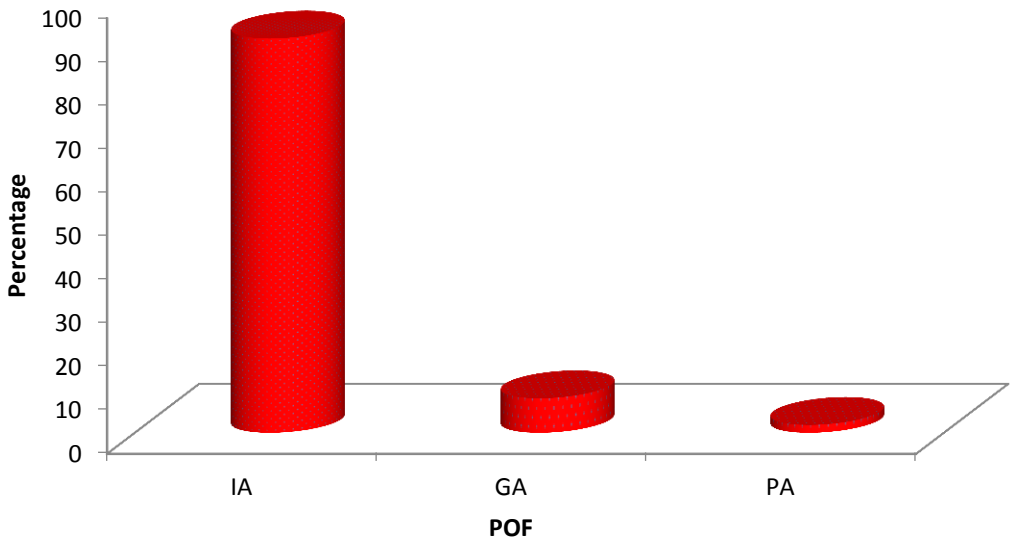
Out of 114 patients clinically diagnosed to have acute appendicitis, CRP was positive in 74.56% of the cases.

Table 10: Distribution of cases as per Intra-Operative Findings (IOF)

IOF	Total
IA	103(90.4%)
GA	9(7.9%)
PA	2(1.8%)
Total	114(100%)

Out of 114 subjects, intraoperatively 90.4% of the appendix were inflamed, 7.9% were gangrenous, and 1.8% were perforated.

Graph 10: Intraoperative findings



DISTRIBUTION OF CASES AS PER HISTOPATHOLOGICAL REPORT

Table 11: HPR distribution in patients studied

HPR	Total
AA	87(76.3%)
CA	11(9.6%)
GA	10(8.8%)
RA	4(3.5%)
PA	2(1.8%)
Total	114(100%)

In the present study (76.3 %) cases were histopathologicaly found to be acute appendicitis, 9.6% cases were turned out to be chronic appendicitis, 8.8% cases were gangrenous, 3.5% cases were ruptured and 1.8% cases were perforated. So there were 16 cases of complicated appendicitis.

Graph 11: HPR distribution

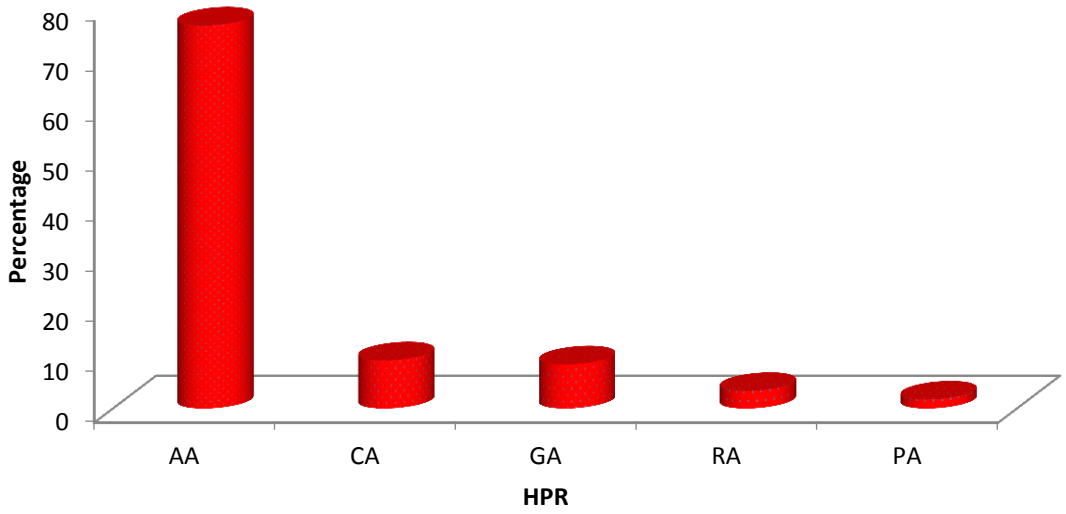


Table 12: TLC levels in relation to Intra-Op Findings

TLC	POF			Total
	IA	GA	PA	
<10	47(45.6%)	0(0%)	0(0%)	47(41.2%)
>10	56(54.4%)	9(100%)	2(100%)	67(58.8%)
Total	103(100%)	9(100%)	2(100%)	114(100%)

P<0.001**, Significant, Fisher Exact test

Out of 103 inflamed appendix, 54.4% of the cases have had leukocytosis, whereas all the cases of gangrenous and perforated appendix were observed to have an elevated leukocyte count which was **statistically significant**.

Graph 12: TLC levels in relation to intraoperative findings

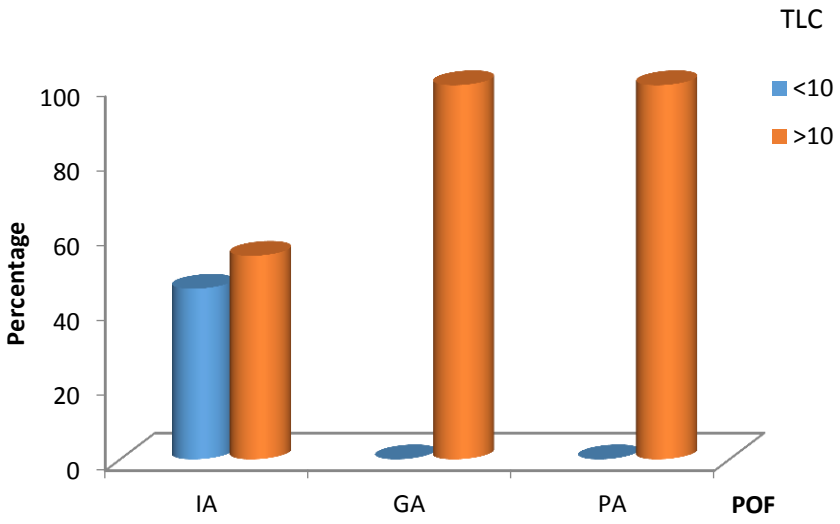


Table 13: CRP in relation with Intra-op findings:

CRP(mg/dl)	IOF			Total
	GA	IA	PA	
N	1	28	0	29
P	8	75	2	85
Total	9	103	2	114

Out of 114 patients, 85 (74.56%) patients had CRP positive. Among these, 75 (88.23%) patients were found to have inflamed appendix intraoperatively, 8 (9.4%) patients had gangrenous appendix and 2(2.3%) patients had perforated appendix.

Graph 13: CRP in relation with Intra-op findings:

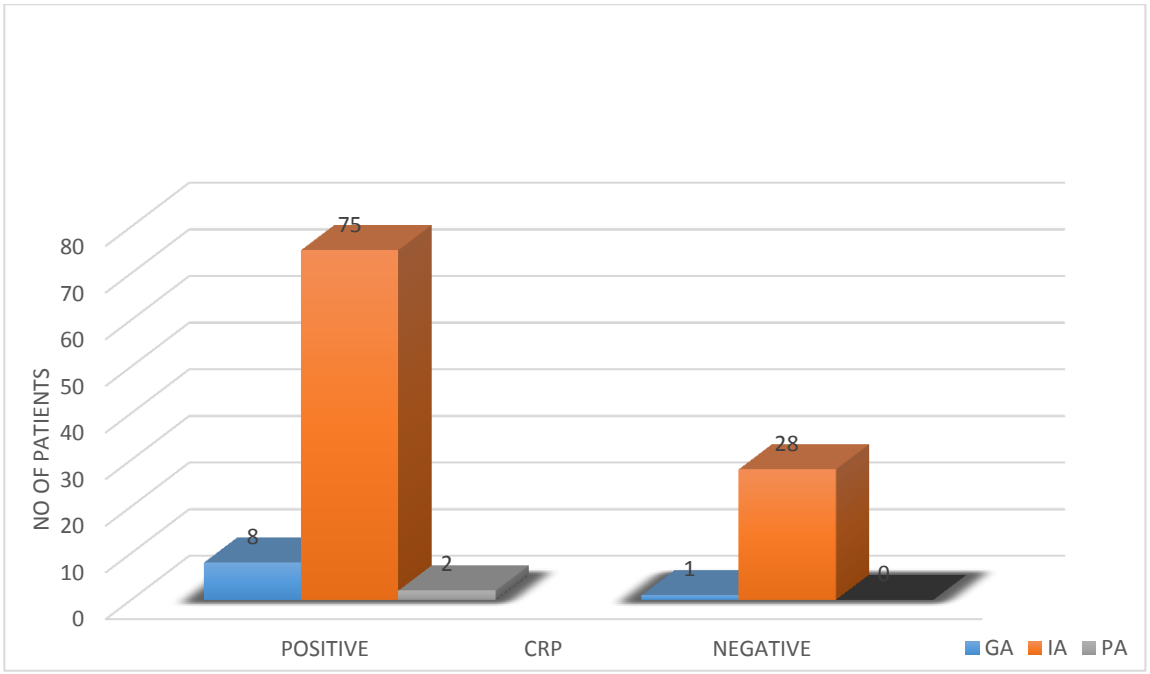


Table 14: HPR findings in relation to TLC levels

HPR	TLC		Total
	<10	>10	
AA	39(83%)	48(71.6%)	87(76.3%)
CA	5(10.6%)	6(9%)	11(9.6%)
GA	0(0%)	10(14.9%)	10(8.8%)
PA	0(0%)	2(3%)	2(1.8%)
RA	3(6.4%)	1(1.5%)	4(3.5%)
Total	47(100%)	67(100%)	114(100%)

P=0.008**, Significant, Fisher Exact test

Out of 67 subjects who had leukocytosis, 71.6% turned out to be acute appendicitis, 9% of the cases were chronic appendicitis, 14.9% were gangrenous, 3% were perforated and 1.5% were recurrent appendicitis on histopathological examination which is **statistically significant.**

Graph 14: HPR findings in relation to TLC levels

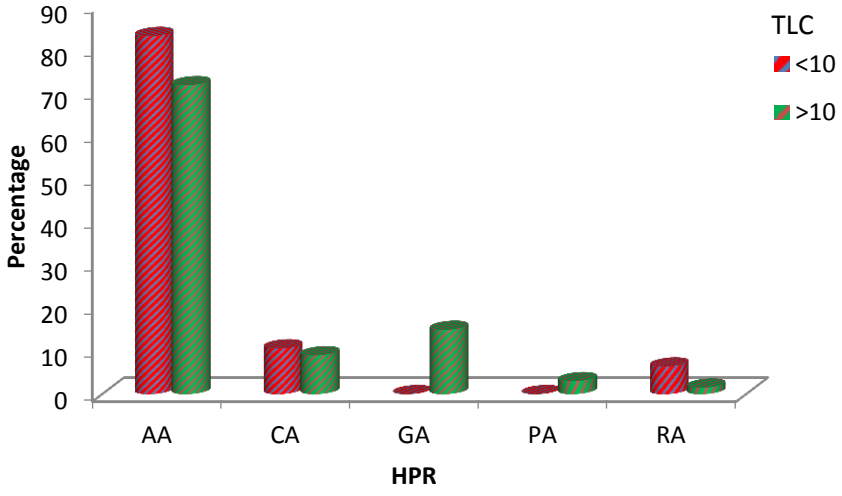


Table 15: CRP findings in relation to HPR findings

CRP	HPR					Total
	AA	CA	GA	PA	RA	
Positive	64(73.56%)	7(63.63%)	9(90%)	2(100%)	3(75%)	85(74.5%)
Negative	23(26.44%)	4(36.36%)	1(10%)	0	1(25%)	29(25.4%)
Total	87(100%)	11(100%)	10(100%)	2(100%)	4(100%)	114(100%)

P=0.656, Not significant, Fisher Exact test

Out of 87 patients of acute appendicitis (confirmed by HPE), only 64 (73.56%) cases were positive for CRP, rest 23 (26.44%) cases patients had normal CRP. Whereas 7(63.63%) cases of chronic appendicitis, 9(90%) cases of gangrenous appendicitis, 2(100%) cases of perforated appendix and 3(75%) of recurrent appendicitis were positive for CRP, which is not statistically significant.

Graph 15: CRP findings in relation to HPR findings

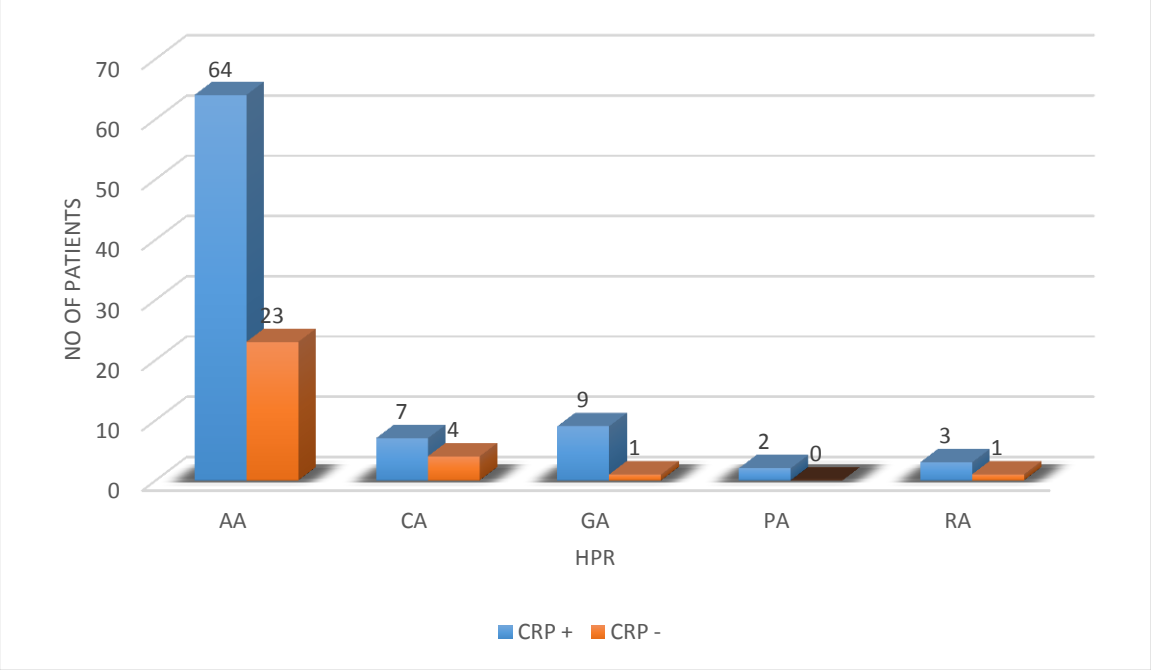


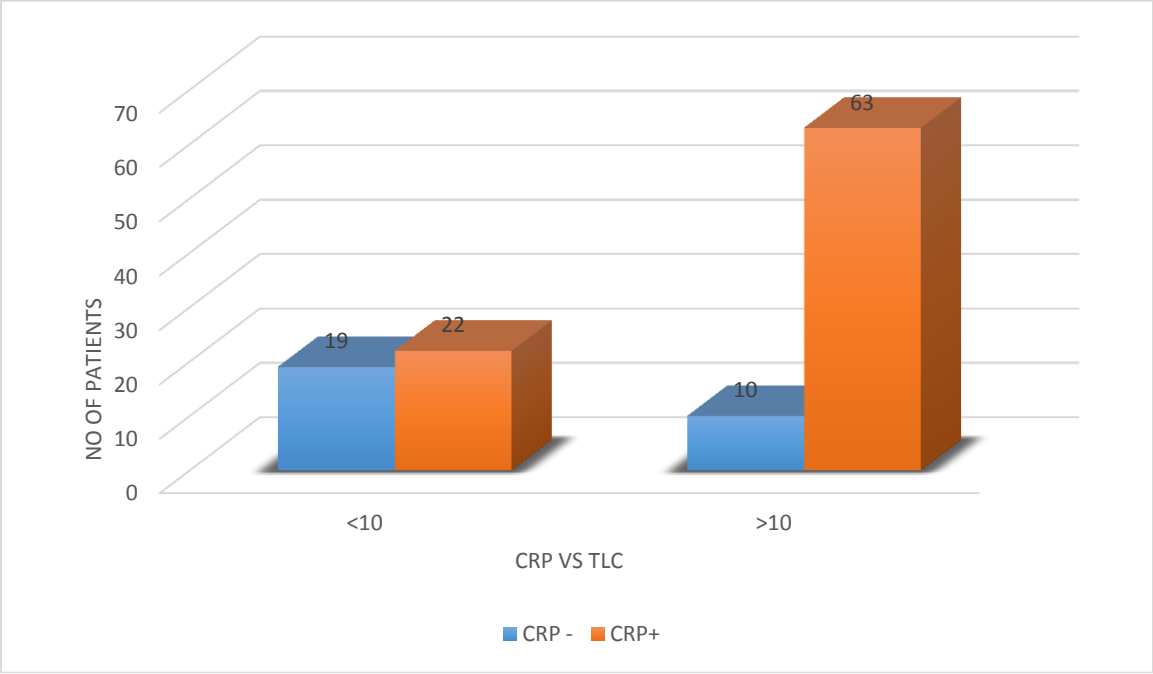
Table 16: CRP in relation to TLC

TLC	CRP(mg/dl)		TOTAL
	NEGATIVE	POSITIVE	
<10	19	22	41
>10	10	63	73
TOTAL	29	85	114

P=0.00, **extremely significant**, Chi-Square test

Among 114 cases clinically diagnosed to have acute appendicitis, both raised TLC and CRP was observed in 63 (55.26%) cases, which was **extremely significant**. Whereas only 19,2% of CRP positive cases did not have leukocytosis and 8.7% of cases with raised TLC were not positive for CRP.

Graph 16: CRP in relation to TLC



DISCUSSION

The present study was done to correlate CRP and TLC with histopathological reports in case of acute appendicitis and to evaluate the efficacy of combined CRP and TLC in diagnosing acute appendicitis.

The study was conducted in Department of General Surgery, R.L.Jalappa Hospital And Research Centre, attached to Sri Devaraj Urs Medical College, Tamaka, Kolar, from period of January 2014 to June 2015 on 114 patients who have been clinically diagnosed of acute appendicitis.

The age of the patients ranged from 8 – 65 years, with a mean age of 23.96 +/- 9.56 years. The most common presenting age group in our study was 21-30 years (38.6%) (Table 3). In a study by CS Agrawal et al, age of the patients ranged from 6 years to 60 years. Maximum number of patients i.e 86 (59.2%) were from the age group of 11-30 years.[73] Appendicitis is common in the age group of 20 - 29 years and <20 years in this study. Appendicitis reaches its peak incidence in the teens and early 20's.

Out of 114 patients, 66 (57.9%) were males and 48 (42.1%) were females (Table 4). So male predominance is seen in the present study. Gender distribution in relation to age was also studied. In males, most common age group of presentation of acute appendicitis was between 21-30 years (42.4%), followed by the age group 10-20 years (34.8%). In females, most common age group was between 10-20 years (37.5%), followed by 21-30 years (33.3%) (Table 5). In the study by Mostafa D et al, it was observed that, out of 426 cases,255 patients (59.9%)

were male and 171 (40.1%) were female. So male predominance was seen in this study also supporting the present study.

Table 6 shows the distribution of symptoms and signs in patients diagnosed with acute appendicitis. In our study, pain abdomen was the presenting complaint in all the patients. The next common symptom was vomiting followed by fever and anorexia. The most common clinical sign was right iliac fossa tenderness which was present in all the cases. In 70% of the cases the clinical presentation is typical and there is no difficulty in making a diagnosis. The remaining 30% have atypical clinical presentation which make the diagnosis difficult.

Many prospective studies have demonstrated that the accuracy of preoperative clinical diagnosis lies in the range of 70-78%. Thus, giving a negative appendicectomy rate around 20.0-25.0% on average.[74]A negative appendicectomy ranging from 10.0-44.0 has been considered acceptable by various authors. In our study, there were no negative laparotomies as clinical diagnosis was found to be correct in all the cases.

In our study, the total leucocyte count was > 10,000 cells / cumm in 67 (58.8%) patients (Table 8). Various studies evaluating TLC in diagnosis of acute appendicitis have variable results. 80–85% patients with acute appendicitis will have TLC count of more than 10,000/cmm.[75]A raised TLC is regarded as sensitive test for diagnosis of acute appendicitis but is not diagnostic because of its lower specificity.[68]The diagnostic value of TLC is increased when combined with neutrophilia and C-reactive proteins.

C – Reactive protein was found to be positive in 85 (74.56%) patients (Table 9). Intraoperative findings were noted as shown in table 10. Out of 114 subjects, intraoperatively 90.4% of the appendix were inflamed, 7.9% were gangrenous, and 1.8% were perforated.

After the operation, appendicectomy specimen was sent for histopathological examination. The histopathology report was considered as the final diagnosis. In the present study (76.3 %) cases were histopathologicaly found to be acute appendicitis, 9.6% cases were chronic appendicitis, 8.8% cases were gangrenous, 3.5% cases were recurrent and 1.8% cases were perforated.

Correlation of total leucocyte count with intraoperative findings is shown in table 12. Out of 114 cases, 67 (58.8%) cases had leukocytosis. Among these, 56 cases had inflamed appendix, 9 cases had gangrenous appendix and 2 had perforated appendix which was statistically significant (p=<0.001).

Marchand et al concluded in their study that WBC > 10.5 x 109/L was one of the single best test for diagnosis of acute appendicitis with highest sensitivities amongst all the tests examined (81-84%).[68]

According to study done by JM Goonroos et al WBC was the test of choice in diagnosing uncomplicated acute appendicitis, however it is a poor predictor of protracted inflammation.[55] This is supported in study by David and Berchley et al. The WBC count when done individually distinguishes normal appendix from uncomplicated acute appendicitis.[76] But does not distinguish uncomplicated from complicated appendicitis. Coleman C et al reported that WBC is a poor predictor of severity of disease.[10] Vermenum et al after evaluating 221 patients concluded that WBC count did not significantly influence the surgical decision making.[31]

Table 13 shows C- reactive protein levels in relation to the intraoperative findings. Out of 114 patients, 85 (74.56%) patients had CRP positive. Among these, 75 (88.23%) patients were found to have inflamed appendix intraoperatively, 8 (9.4%) patients had gangrenous appendix and 2(2.3%) patients had perforated appendix which was not statistically significant.

Many reports have investigated the value of CRP in improving the diagnostic accuracy of acute appendicitis with conflicting results. A multivariate analysis by Oosterhuis et al[61] showed that serial CRP measurement can improve the accuracy of diagnosing acute appendicitis. Other reports did not support this view. In addition, a meta-analysis of 22 published articles concluded that CRP is a test of medium accuracy in diagnosing acute appendicitis.

The correlation between histopathology of the appendix with TLC is shown in table 14. Leukocytosis (> 10,000 / cumm) was found in in 67 cases while the count was within normal limit in 47 cases of histopathological proven appendicitis cases. Out of 67 subjects who had leukocytosis, 71.6% turned out to be acute appendicitis, 9% of the cases were chronic appendicitis, 14.9% were gangrenous, 3% were perforated and 1.5% were recurrent appendicitis on histopathological examination which is statistically significant (p=0.008).

In a study by Hyder et al, it was observed that out of 100 cases, 81 cases had histopathological features of acute appendicitis, out of which 62 cases had leukocytosis of > 10,000.[77] In another study by Mostafa D et al, it was noticed that 214 cases had acute appendicitis, 102 cases were chronic appendicitis, 36 were gangrenous and 25 were perforated appendicitis.[78] In another study by CS Agarwal et al, 81 patients were histologically found to have acute appendicitis with leukocytosis of > 11,000 per cumm.[73] In all these studies it was noted that TLC was

a significant variable in diagnosing acute appendicitis. Sengupta A et al in their study on 98 patients found that TLC was raised to > 11,000 in 85 cases with significant p value of 0.012.[79]

Table 15 shows correlation of CRP with HPR findings. Out of 87 patients of acute appendicitis (confirmed by HPE), only 64 (73.56%) cases were positive for CRP, rest 23 (26.44%) patients had normal CRP. Whereas 7(63.63%) cases of chronic appendicitis, 9(90%) cases of gangrenous appendicitis, 2(100%) cases of perforated appendix and 3(75%) of recurrent appendicitis were positive for CRP, which is not statistically significant.

In a study by CS Agarwal et al, Appendicitis was diagnosed histopathologically in 103 cases. Among these CRP was raised in 77 cases and was normal in 26 cases.[73] Davies et al. conducted a study on 60 patients with right iliac fossa pain, CRP and full blood counts were performed and found that 94% of patients had raised CRP with acute appendicitis and 83% of patients had negative CRP results with negative appendictomies.[67]

Verma et al, measured C-reactive protein in 42 cases admitted to a general hospital with suspected acute appendicitis. Thirty five were operated and thirty one of these with raised CRP had an inflamed appendix. Four cases with normal CRP value had scarred appendix (healed appendicitis) which was confirmed by biopsy reports. These four cases also had normal white blood cell count and ESR.[80]

Gurleyik et al.compared serum CRP study of 108 patients suspected of having appendicitis on clinical grounds. The diagnosis depending on surgeon's clinical impression was true in 90 patients and false in 18 patients. This difference was statistically significant (p = 0.0035). They recommend CRP measurement as a routine laboratory test in patients with suspected diagnosis of

acute appendicitis.[63]

CRP in relation to TLC is shown in table 16. Among 114 cases clinically diagnosed to have acute appendicitis, raise in both TLC and CRP was observed in 63 (55.26%) cases, which was extremely significant. Whereas only 19.2% of CRP positive cases did not have leukocytosis and 8.7% of cases with raised TLC were not positive for CRP.

CRP levels were not statistically significant in diagnosing acute appendicitis when considered individually. Whereas TLC was found to be statistically significant with p= 0.008. However, when both CRP levels and TLC were considered, the results were found to be extremely significant with p= 0.00.

Gronroos JM, Gronroos P in a retrospective study studied the preoperative leucocyte counts and C-Reactive protein (CRP) values in three groups of patients operated on for a clinical suspicion of acute appendicitis. They concluded that acute appendicitis is very unlikely when both the leucocyte count and CRP value are normal.[55]

Asfar S et al.[56] conducted a double blind trial in 78 patients to study the impact of a normal (rather than raised) serum C-reactive protein in reducing the rate of negative explorations. White blood count (WBC), CRP and the histopathology findings were correlated. In patients with histopathologically proven acute appendicitis both the WBC count and serum CRP level were significantly raised ($p=0.025$ and $p<0.000$ respectively). Serum CRP level was normal in 13 out of 15 negative explorations (normal appendix on histopathology). The specificity and sensitivity of serum CRP was 86.6% and 93.6%, respectively. They concluded that a normal pre-operative serum CRP measurement in patients with

suspected acute appendicitis is most likely associated with a normal appendix. Deferring surgery in this group of patients would probably reduce the rate of unnecessary appendicectomies.

Erikson et al. (1994)[69] measured serum CRP level and WBC count every four hours in a cohort of 227 patients with suspected acute appendicitis, and reported that it was unusual to find a normal CRP level after 8 hours of observation in the presence of acute appendicitis. If these test results are normal, the surgeon should preferably refrain from operating and consider other differential diagnosis.

All the above studies recommend that CRP and TLC measurement as a routine laboratory test in patients with suspected cases of acute appendicitis as it supports surgeons clinical diagnosis and minimizes negative appendicectomy.

CONCLUSION

Out of 114 patients clinically diagnosed to have acute appendicitis, male predominance was seen with most common presenting age group of 21-30 years.

Clinical diagnosis was found to be correct in all the cases and hence there were no negative laparotomies for acute appendicitis in our study emphasizing the importance of clinical diagnosis.

Leukocytosis was found to be significant in diagnosing acute appendicitis whereas CRP was insignificant in our study. However, combining CRP and TLC the results were found to be extremely significant.

Thus, it should be stressed that serum CRP estimation does not replace clinical diagnosis, but is useful adjunct in diagnosis of acute appendicitis. Clinical diagnosis is crucial in ruling out alternate diagnosis and other conditions. Thus serum CRP value should be interpreted in combination with clinical findings and leucocyte count.

SUMMARY

The present study was conducted on 114 patients who have been clinically diagnosed to have acute appendicitis and posted for emergency appendicectomy in General Surgery Department, R.L.Jalappa Hospital And Research Centre, attached to Sri Devaraj Urs Medical College, Tamaka, Kolar, from period of January 2014 to June 2015. The aim of the study was to correlate CRP and Total leukocyte count with histopathology report in case of acute appendicitis and to evaluate the efficacy of combining both CRP and TLC in acute appendicitis. All the patients were subjected to histopathological examination which was taken to be the gold standard.

Out of 114 patients clinically diagnosed to have acute appendicitis, male predominance was seen with most common presenting age group of 21-30 years.

Clinical diagnosis was found to be correct in all the cases and hence there were no negative laparotomies for acute appendicitis in our study emphasizing the importance of clinical diagnosis.

Out of 67 subjects who had leukocytosis, 71.6% turned out to be acute appendicitis, 9% of the cases were chronic appendicitis, 14.9% were gangrenous, 3% were perforated and 1.5% were recurrent appendicitis on histopathological examination which is statistically significant with p value of 0.008.

Out of 87 patients of acute appendicitis, only 64 (73.56%) cases, 7(63.63%) cases of chronic appendicitis, 9(90%) cases of gangrenous appendicitis, 2(100%) cases of perforated appendix and 3(75%) of recurrent appendicitis were positive for CRP, which is not statistically significant.

Both raised TLC and CRP was observed in 63 (55.26%) cases, which was extremely significant. Whereas only 19.2% of CRP positive cases did not have leukocytosis and 8.7% of cases with raised TLC were not positive for CRP.

Thus, it should be stressed that serum CRP estimation does not replace clinical diagnosis, but is useful adjunct in diagnosis of acute appendicitis. Clinical diagnosis is crucial in ruling out alternate diagnosis and other conditions. Thus serum CRP value should be interpreted in combination with clinical findings and leucocyte count.

BIBLIOGRAPHY

1. Harold E, Nathanson LK. Appendix and Appendectomy. 10th ed. In: Maingot's Abdominal operation, Zinner MJ, Schwartz SI. Harold E, eds. Connecticut: A Simon and Schuster Company; 1997. 2:1192,1193,1200,1202.

2. Brown SP. Acute appendicitis. 13th ed. In: Ellis BW, Brown SP, editors. Hamilton Bailey's Emergency Surgery. New York: Arnold; 2000. pp. 399-400.

3. John H, Neff U, Kelemen M. Appendicitis diagnosis today: clinical and ultrasonic deductions. World J Surg 1993; 17:243 -9.

4. Balsano N. Cayten CG. Surgical Emergencies of the abdomen. Emerg Med Clinc North Am. 1990:8:399-410.

5. Lewis FR. Hocroft JW. Boey J. Dumphy E. Appendicitis: A critical review of the diagnosis and treatment in 1000 cases. Arch Surg. 1975; 110:677-84.

6. Collins DC. Historic phases of appendicitis, fellow in surgery, the mayo foundation from the section on pathologic anatomy of the mayo clinic.

6 Zuidema: Shackelford's Surgery of the Alimentary Tract, 5th ed.

7 Bernard M Jaffe and David H.Berger ; The appendix; In: Schwartz's principles of surgery; F.Charles Brunicardi, MD, FAS (Ed); 9th edition; Mc Graw Hill; 2010; p 1073-90.

8 Pieper R, Kager L et at. Obstruction of the appendix vermiformis causing acute appendicitis. An experimental study in the rabbit. Acta Chir Scand. 1982;148:63.

9 John Maa, MD, Kimberly S, Kirkwood, MD. The Appendix. In: Sabiston text book of surgery: Courtney M. Townsend (Ed); 18th ed; Saunders; 2008. I:1333-46.

10 Coleman C, Thompson J, Bennion R, Schmit P. White Blood Cell count is a poor predictor of severity of disease in diagnosis of appendicitis. Am Surg 1998;64(10):983-5.

11 Bolton JP, Craven ER, Croft Rj, Menzies-Grow N. Am assessment of the white cell count in the management of suspected acute appendicitis. Br J surg 1975; 62:906-8.

12 Gray's anatomy-The Anatomical Basis of Clinical practice Susan Standring (Ed); 39th ed; Elsevier Churchill Livingstone; 2008; p 1366-7.

13 DouglasS.smink/DavidI.soybel; Appendix and appendectomy; In Maingot's abdominal operations; Michel J, Zinner (Ed); 11th ed; McGraw-Hill; 2007; pp. 589-608.

14 Swasso RD, Hanna EA, Moore DL. leukocytic and neutrophilic counts in the acute appendicitis. Am j surj 1970; 120:563-6.

15 English DC. allen W, coppala ED, Sher A.Excessive dependence on the leucocytosis clue in diagnosing appendicitis. Am j surg 1977; 43:399-402.

16 Wakeley, CPG. The position of the vermiform appendix as ascertained by an analysis of 10, 000 cases. J Anat Physiol 1933;67:277-83.

17 Lally KP, Cox CS Jr, Andrassy RJ. Appendix. In: Townsend CM, Beauchamt RD, Evers BM, Mattox KL, editors. Sabistion text book of surgery. 16th edn. Philadelphia: WB Saunders company; 2001. p 918, 919, 920.

18 Robbins's and Cotran Pathologic basis of diseases; Kumar V; Abbas AK; Fausto N (Eds); 8th ed; Elsevier; 2010; 826-8.

19 Juan Rosai; Appendix; In: Ackerman's surgical pathology; Juan Rosai (Ed); 9th ed; Mosby; 2005; VOL I p 757-60.

20 Sternbergs diagnostic pathology ; Stacey E mills (ed) 4th edition ;Lippincott, Williams and wilkins ; 2004 SEC viii (VOL 2) ; p 1521-4.

21 P.RonanO'Connell; The vermiform appendix; In: Bailey andlove's short practice of surgery; ChristopherJ.K.Bulstrode, Norman S. Williams, P.RonanO'Connell (Ed); 25th ed; 2008 Hodder Arnold; p 1204-17

22 Wangensteen OH, Bowera WF. Significance of the obstructive factor in the genesis of acute appendicitis. An experimental study. Arch Surg 1937;34:496.

23 Wilkie DPD. Acute Appendicitis and acute Appendicular obstruction. BMJ. 1914;2:959.

24 Horwitz JR, Gursoy MF, Jaksic T et al. Importance of diarrhea as presenting symptom of appendicitis in very young children. Am J Sur 1997;173:80-2.

25 Allan Clain. Hamilton Bailey's demonstration of physical signs in clinical surgery. 16th ed. 1980;288-94.

26 William Silen. Appendicitis: Silen Cope's early diagnosis of the acute abdomen.Oxford University Press. 20th ed. 2000:6;65-81.

27 Fitz RH. Perforating inflammation of vermiform appendix, with special reference to its early diagnosis and treatment. Trans Assoc Phys 1986;1:107-43.

28 Mueller BA, Daling JR, Moore DE, et al. Appendectomy and the risk of tubal infertility. N Engl J Med 1986;315:1506-8.

29 Mourad J, Elliott JP, Erickson L et al. Appendicitis in pregnancy: new information that contradicts long-held clinical beliefs. Am J Obstet Gynecol. 2000;182:1027-9.

30 Doraiswamy NV. Leucocyte counts in the diagnosis and prognosis of acute appendicitis in children. Br J Surg 1979;66:782.

31 Vermeulen B, Morabia A, Unger PP. Influence of white cell count on surgical decision making in patients with abdominal pain in the right lower quadrant. Eur J Surg1995;161:483-6.

32 Brooks DW, Killen DA. Roentgenographic findings in acute appendicitis. Surgery.1965;57:377.

33 Jona JZ, BelinRP, Selke AC. Barium enema as a diagnostic aid in children with right lower quadrant pain J A M A 1981; 245;505-648.

34 Stephanie R Wilson; Gastrointestinal system in; Diagnostic ultrasound Carol Rumack M.D, Stephanie R Wilson, J William Chorboneau M.D (ed) 3rd edition Mosby 2005 VOL .I

35 J.B.C.M Puylaert. The Appendix;Abdomen and General Ultrasound ;David Cosgrove, Hyltone Meire, Keith Drewbury (Ed) 1st edition ;Churchill livingstone 1993 VOLII ;p777.

36 Clive A Barrtan ;GIT Large bowel;Diagnostic Radiology A T extbook Of Medical Imaging ;R.G Granger, D.J Allison (Ed) 3rd edition ;Mosby; VOL II SEC IV 1042 chapter 50.

37 Puylaert JB, Rutgers PH, Lalisang RI, et al. A prospective study of ultrasonography in the diagnosis of appendicitis. N Engl J Med. 1987;317:666-9.

38 Puylaert JB. Acute appendicitis: US evaluation using graded compression. Radiology. 1986;158:355-60.

39 HS Fung, S Lau, JCW Siu, CM Chan, SCH Chan; Audit of Ultrasonography for Diagnosis of Acute Appendicitis Retrospective Study; J HK Coll Radiol. 2008;11:108-11.

40 Kessler N, Cyteval C, Gallix B, et al. Appendicitis: evaluation of sensitivity, specificity, and predictive values of US, Doppler US, and laboratory findings. Radiology. 2004;230:472-8.

41 Patriquin HB, Garcier JM, Lafortune M et al. Appendicitis in children and young adults. Doppler sonographic- pathologic correlation. AJR Am J Roentgenol 1996;166:629-33.

42 Incesu L, Coskun A, Selcuk M.B et al. MR imaging and sonographic correlation. AJR Am J Roentgenol 1997;168:669-74.

43 Kipper SL, Rypins EB, Evans DG et al. Neutrophil-specific 99mTc- labeled anti-CD15 monoclonal antibody imaging for diagnosis of equivocal appendicitis. J Nucl Med 2000;41:449-55.

44 Paterson-Brown S, Thompson JN, et al. Which patients with suspected appendicitis should undergo laparoscopy? Br Med J 1988;296-363.

45 Owens TD. Williams H. Stiff G. Jenkinson L.R. Evaluation of the Alvarado score in acute appendicitis. J.R. Soc Med 1992:85:87-9.

46 Alvarado A. A practical score for the early diagnosis of acute appendicitis. Ann Emerg Med 1986;15:557-64.

47 Kalan M., Rich AJ., Talbot D., Cunliffe WJ.: Evaluation of the modified Alvarado score in the diagnosis of acute appendicitis: a prospective study. Ann R. Coll. Surg. Engl 1994; 76:418-9.

48 Tillet W, Francis T. Serological reactions in pneumonia with nonprotein somatic fraction of pnuemococcus. 1930;52:561.

49 Shrive AK, Cheetham GM, Holden D, Myles DA, Turnell WG, Volanakis JE, Pepys MB, Bloomer AC, Greenhough TJ. Three dimensional structure of human C-reactive protein. Nat Struct Biol 1996;3:346-54.

50 Gewurz H, Mold C, Siegel J, Fiedel B.C-reactive protein and the acute phase response. Adv Intern Med. 1982;27:345-72.

51 Stein MP, Edberg JC. C-reactive protein binding to Fcgamma RIIa on human monocytes and neutrophils is allele-specific. J Clin Invest 2000 Feb; 105(3):369-76.

52 Gershov D, Kim S. C-Reactive protein binds to apoptotic cells, protects the cells from assembly of the terminal complement components, and sustains an antiinflammatory innate immune response: implications for systemic autoimmunity. J Exp Med. 2000 Nov 6;192(9):1353-64.

53 Erkasap S, Ates E. Diagnostic value of interleukin-6 and C-reactive protein in acute appendicitis. Swiss Surg. 2000;6(4):169-72.

54 Duff SE, Dixon AR. Laparoscopic appendicectomy: safe and useful for training. Ann R Coll Surg Engl 2000; 82:388-91.

55 Gronroos M and Gronroos P. leucocyte count and C- reactive protein in the diagnosis of acute appendicitis.BJS 1999; 86: 501-4.

56 Asfar S, Safar H, Khoursheed M, Dashti H, Al-Bader A. Would measurement of Creactive protein reduce the rate of negative exploration for acute appendicitis? J R Coll Surg Edinb Feb 2000; 45: 21-4.

57 Shakhatresh HS. The accuracy of C-reactive protein in the diagnosis of acute appendicitis compared with that of clinical diagnosis. Med Arh 2000;54(2):109-10

58 Pruekprasert P, Maipang T, Geater A, Apakupakul N, Ksuntigij P. Accuracy in diagnosis of acute appendicitis by comparing serum C-reactive protein

measurement, Alvarado score and clinical impression of surgeons. J Med Assoc Thai 2004 Mar;87(3): 296-303.

59 Dueholm S. Bagi P. Bud M. Laboratory aid in the diagnosis of acute appendicitis. A blinded, prospective trial concerning diagnostic value of leukocyte count neutrophil differential count, and C- reactive protein. Dis colon rectum 1989 Oct; 32(10):855-9.

60 Hallan S, Asberg A. The accuracy of C-reactive protein in diagnosing acute appendicitis - a meta-analysis. Scand J Clin Lab Invest 1997; 57: 373-80.

61 Oosterhuis WP, Zwinderman AH, Teeuwen M, van Andel G, Oldenziel H, Kerkhoff JF et al. C reactive protein in the diagnosis of acute appendicitics. Eur J Surg 1993 Feb;1599(2):115-9.

62 Shryl-Chryl Chen, MD, Kai-Mo Chen, MD, Shih-Ming Wang MD. Abdominal Sonography screening of clinically diagnosed or suspected appendicitis before surgery. World Journal of Surgery 1998;22:419-51.

63 Gurleyik E, Gurleyik G. Unalmiser S. Accuracy of serum of C- reactive protein measurements in diagnosis of acute appendicitis compared with surgeon's clinical impression. Dis Colon Rectum 1995 Dec;38 (12):1270-4.

64 Birchley D. Patients with Clinical Acute Appendicitis Should have Pre-operative Full Blood Count and C-Reactive Protein Assays. Ann R Coll Surg Engl 2006 Jan;88(1)27-32.

65 Thimsen DA, Tong GK, Gruen DA, Tong GK, Gruenberg JC. Prospective evaluation of C- reactive protein in patients suspected to have acute appendicitis. Am Surg 1989 Jul; 55(7):466-8.

66 Eriksson S. Granstrom L. Carlstrom A. The diagnostic value of repetitive preoperative analyses of C-reactive protein and total leucocyte count in patients with suspected acute appendicitis. Scand J Gastroenterol 1994 Dec; 29 (2):1145-9.

67 Davies AH. Bernau F, Salisbury A, souter RG. C- reactive protein in right iliac fossa pain. JR Coll Surg Edinb 1991 Aug; 36(4):242-4.

68 Marchand A. Van Lente F, Galen RS. The assessment of laboratory tests in the diagnosis of acute appendicitis. Am J Clin Pathol 1983 Sep; 80(3):369-74.

69 Eriksson S. Granstrom L. Olander B, Wretlind B. Sensitivity of interleukin – 6 and CReactive protein concentration in the diagnosis of acute appendicitis. Eur J Surg 1995 Jan; 161(1):41-5.

70 Khan MN, Davie E, Irshad K. The role of white cell count and c-reactive protein in the diagnosis of acute appendicitis.

71 Albu E, Miller B.M. Choi Y. Lakhanpal S, Murthy RN, Gerst PH. Diagnostic value of C-reactive protein in acute appendicitis. Dis Colon Rectum 1994; 37:49-51.

72 Agrawal CS, Adhikari S, Kumar M. Role of serum C- reactive protein and leukocyte count in the diagnosis of acute appendicitis in Nepalese population. Nepal Med Coll J 2008; 10(1): 11-15.

73 Schwartz SI. Appendix. In: Schwartz S I Ed principles of surgery. 6 th ed New York: McGraw Hill; 1994: 1307-18.

74 Bener A, Suwaidi MH, Ghazawi IE. Diagnosis of appendicitis. Can J Rural Med 2002;7: 26–9 15.

75 De Carvalho BR, Diogo FA, Fernandes C. Barra CB. Leukocyte count, C-reactive protein, alpha-1 acid glycoprotein and erythrocytes sedimentation rate in acute appendicitis. Arch Gastroenterol 2003; 40:25–30.

76 Birchley D. Patients with Clinical Acute Appendicitis Should have Pre-operative Full Blood Count and C- Reactive Protein Assays. Ann R Coll Surg Engl 2006 Jan; 88(1)27-32.

77 Kamran H, Naveed D, Nazir A. Role of total leukocyte count in diagnosis of acute appendicitis. J Ayub Med Coll Abbottabad. 2008;20:70-71.

78 Dahmardehei M, Khazaei A, Vahab M. Diagnostic value of leukocytosis, ESR and CRP in patients with suspected acute appendicitis. Zahedan J Res Med Sci; 2013:15:59-63

79 Sengupta A, Bax G, Brown SP. White cell count and C-reactive protein measurement in patients with possible appendicitis. Ann R Coll Surg Engl 2009; 91: 113–115.

80 Amrita Verma, Mehta FS, Vyas KC, Sharma VP, Dhruv AK. C- Reactive protein in acute appendicitis. Indian Journal of Surg July-Aug 1995: 273-5.

ANNEXURE I

PROFORMA

NAME: D.O.A:

AGE: D.O.O:

SEX: D.O.D:

OCCUPATION:

ADDRESS:

CHIEF COMPLAINTS:

1) Pain

2) Vomiting/ nausea

3) Fever 4) Diarrhoea/ constipation

5) Distension of abdomen 6) Other complaints

HISTORY OF PRESENTING ILLNESS

1) PAIN: Duration / Time and mode of onset / Site of pain: (RIF/ Epigastric/ Periumbilical/ Diffuse)/ Shifting of pain /. Migration or radiation of pain / Character of pain /Aggravating factors / Relieving factors

2) VOMITING: Duration / Its relation with pain/ Frequency and quantity / Character/ Colour and nature of vomitus

3) FEVER: Mild/ Moderate/ Severe, Continuous/ Intermittent/ Remittent associated with chills and rigors

4) ANOREXIA

5) BOWELS: Diarrhoea / Constipation /Tenesmus

6) MICTURITION: Associated with increased Painful/ burning / Frequency / Quantity / Colour (hematuria)

7) OTHER COMPLAINTS:

PAST HISTORY

1) History of similar attacks, Duration, Treatment taken
2) History of previous surgeries or past medical history
3) History suggestive of Hypertension/ Diabetes/ Tuberculosis

PERSONAL HISTORY

Diet: Vegetarian/ Mixed Habits: Smoking/ Alcohol/ Tobacco Bowel and Bladder habits Sleep

MENSTRUAL HISTORY Age of menarche, menstrual cycles (regular /irregular / h/o passing clots /dysmenorrhoea), L. M. P / Vaginal discharge

Marital status, obstetric history

FAMILY HISTORY

Similar illness in other family members

GENERAL PHYSICAL EXAMINATION

1. General survey
2. Body build and nourishment
3. Appearance and. Attitude: Restless/ Quiet
4. Dehydration: Mild/ Moderate/ Severe/ Nil
5. Anaemia/ Jaundice/ Clubbing/ Cyanosis/ Lymphadenopathy/ Pedal oedema
6. Pulse
7. Temperature
8. Respiratory rate
9. Blood pressure

LOCAL EXAMINATION

ABDOMEN:

1. INSPECTION

Contour / Position of umbilicus / Movements with respiration / Any operative scar / Visible swelling Flanks / Spine / External genitalia / Hernial orifices

2.PALPATION

Local rise of temperature / tenderness on superficial palpation / Hyperaesthesia at Sherren's triangle / Tenderness at McBurney's point / guarding / rigidity/ rebound tenderness / Rovsing's sign / Cope's psoas test / Baldwin's test / Palpable mass / Hernial orifices / External genitalia / Liver/ Spleen/ Kidney

3.PERCUSSION

Percussion note: Resonant/ Dull/ Tympanic, Shifting dullness/ Fluid thrill, Liver dullness

Renal angles: Dull/ Resonant

4.AUSCULTATION

Bowel sounds

RECTAL EXAMINATION

VAGINAL EXAMINATION

SYSTEMIC EXAMINATION

- Cardiovascular system
- Respiratory system
- Central nervous system

INVESTIGATIONS

1. Blood: Hb%
2. BT
3. CT
4. ESR
5. Blood group and RH type
6. Urine: Albumin/ Sugar/ Microscopy
7. Plain X-ray erect abdomen
8. Chest X-ray
   A) Total leucocyte count
   B) Differential leucocyte count
   C) C-Reactive protein
   D) Ultrasonography abdomen and pelvis – features suggestive of Acute appendicitis / alternative diagnosis / inconclusive

DIAGNOSIS

MANAGEMENT

SURGERY

Type of Anaesthesia / Type of incision

OPERATIVE FINDINGS

- Position/length / thickness of appendix:
- Inflamed / not inflamed:
- Fecolith present / absent:

- Gangrene of appendix:
- Perforation of appendix:
- Associated peritonitis, abscess:
    - Other findings:

HISTOPATHOLOGY

REPORT

Inflamed appendix / Gangrenous appendix / Normal appendix / Perforated appendix

FINAL DIAGNOSIS

Acute appendicitis

Other diagnosis

POST-OPERATIVE PERIOD:

Eventful / Uneventful:

ANNEXURE II

INFORMED CONSENT FORM

"EVALUATION OF C-REACTIVE PROTEIN LEVEL AND TOTAL LEUCOCYTE COUNT IN ACUTE APPENDICITIS"

I, the undersigned, agree to participate in this study and authorize the collection and disclosure of my personal information as outlined in this consent form.

I understand the purpose of this study, the confidential nature of the information that will be collected and disclosed during the study. The information collected will be used only for research.

I also understand the need of required investigations (CRP and TLC) for the purpose of carrying out this study.

I have had the opportunity to ask questions regarding the various aspects of this study and my questions have been answered to my satisfaction.

I understand that I remain free to withdraw from this study at any time and this will not change my future care.

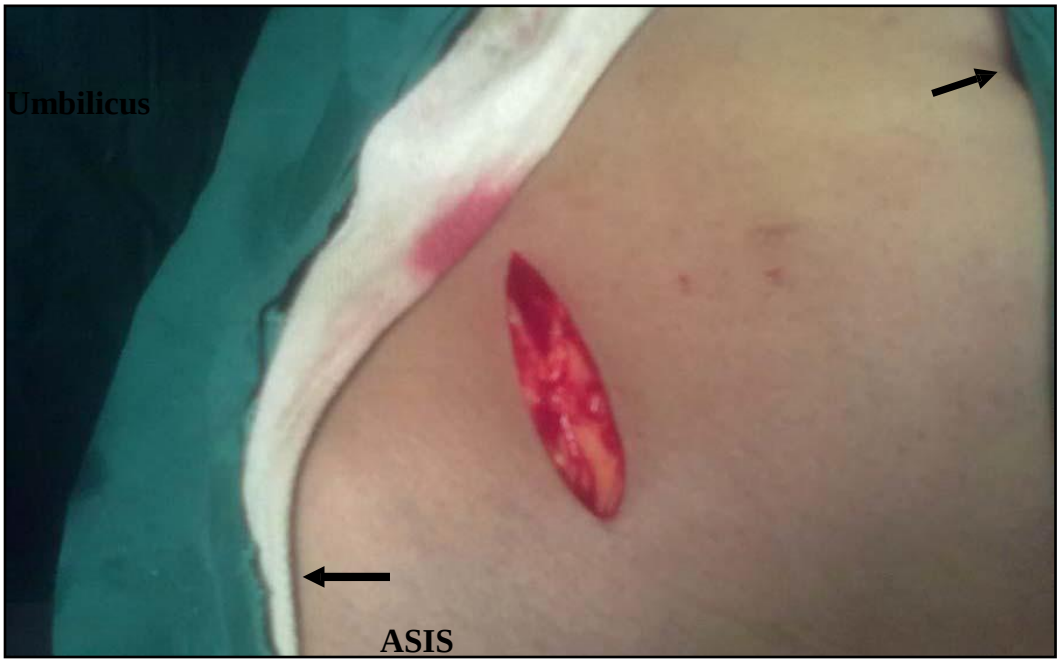
Participation in this study does not involve any extra cost to me.

Subject's name and signature /thumb impression Date:

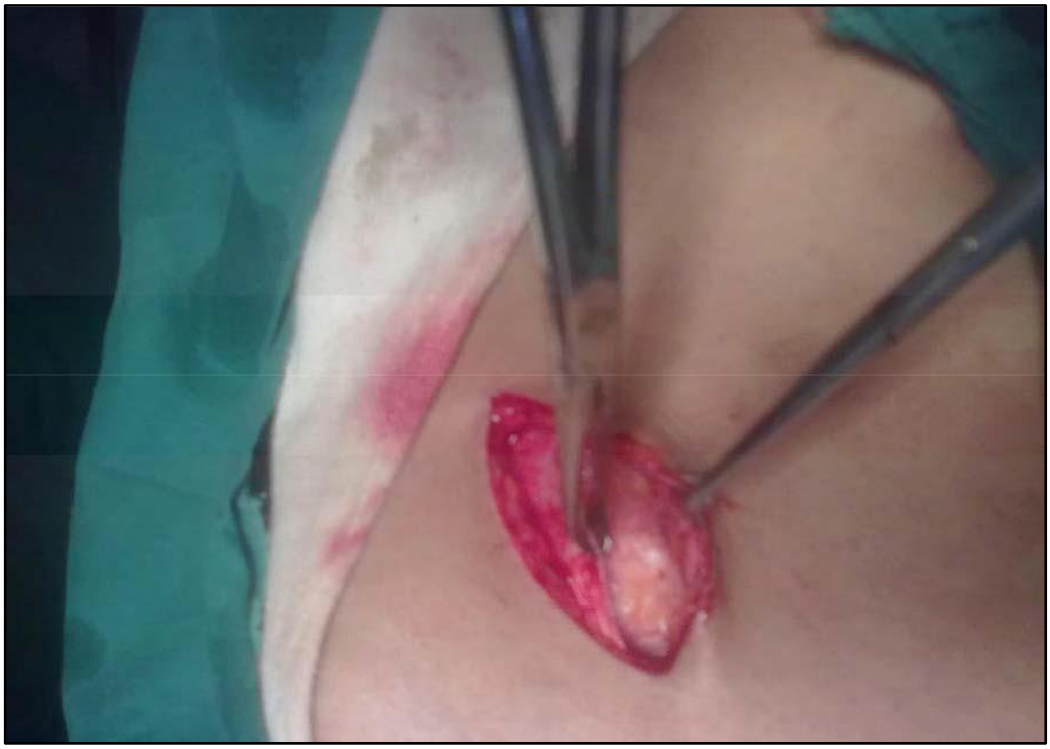
Name and signature of witness Date:

Name and signature of person obtaining consent Date:

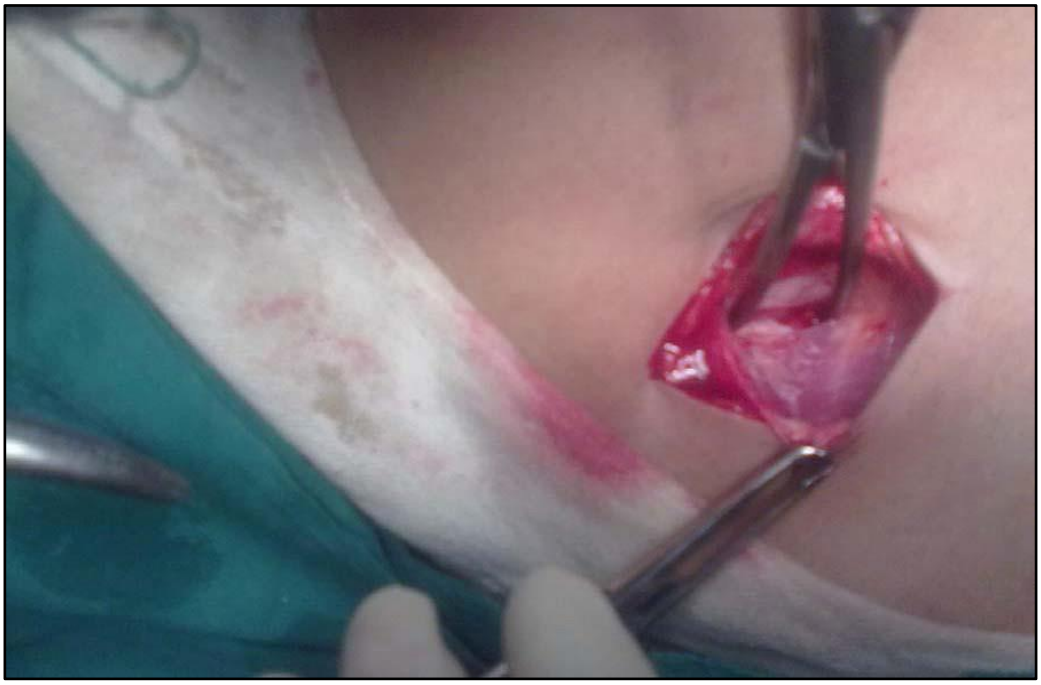
ANNEXURE III
PHOTOGRAPHS



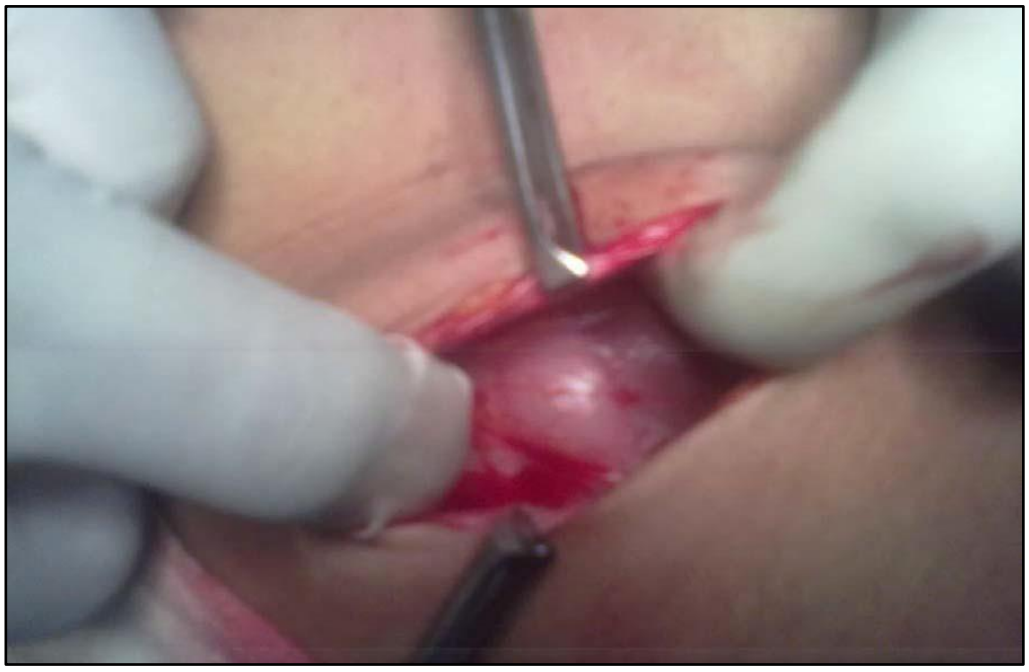
Picture 1: McBurney's incision taken on the skin



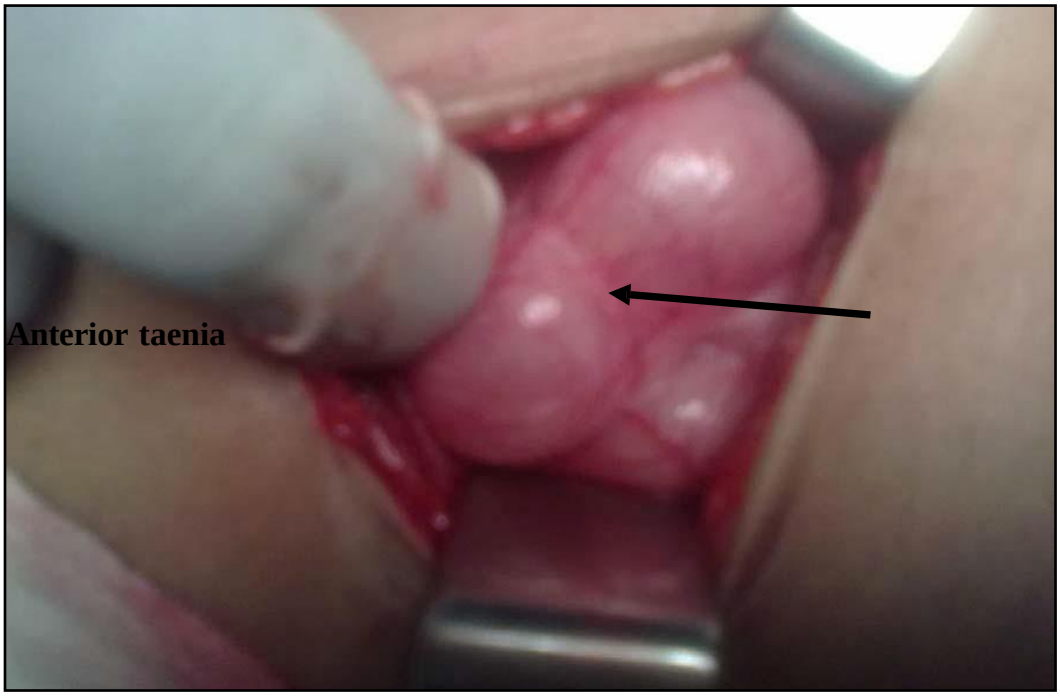
Picture 2: External oblique cut



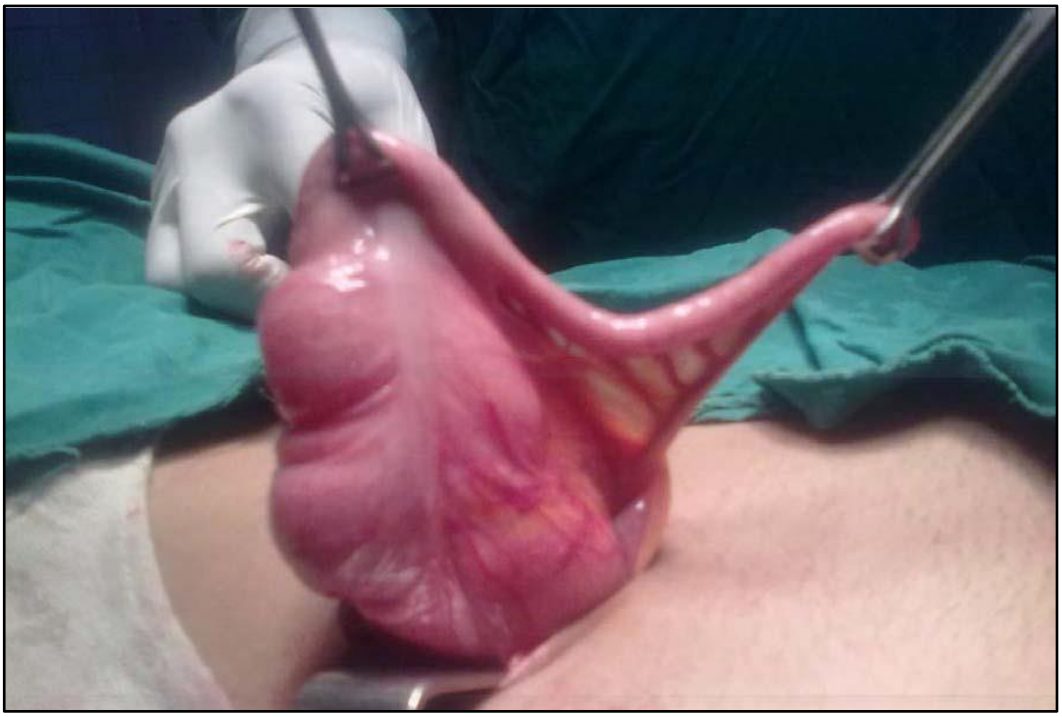
Picture 3: Internal oblique and transverse abdominis split



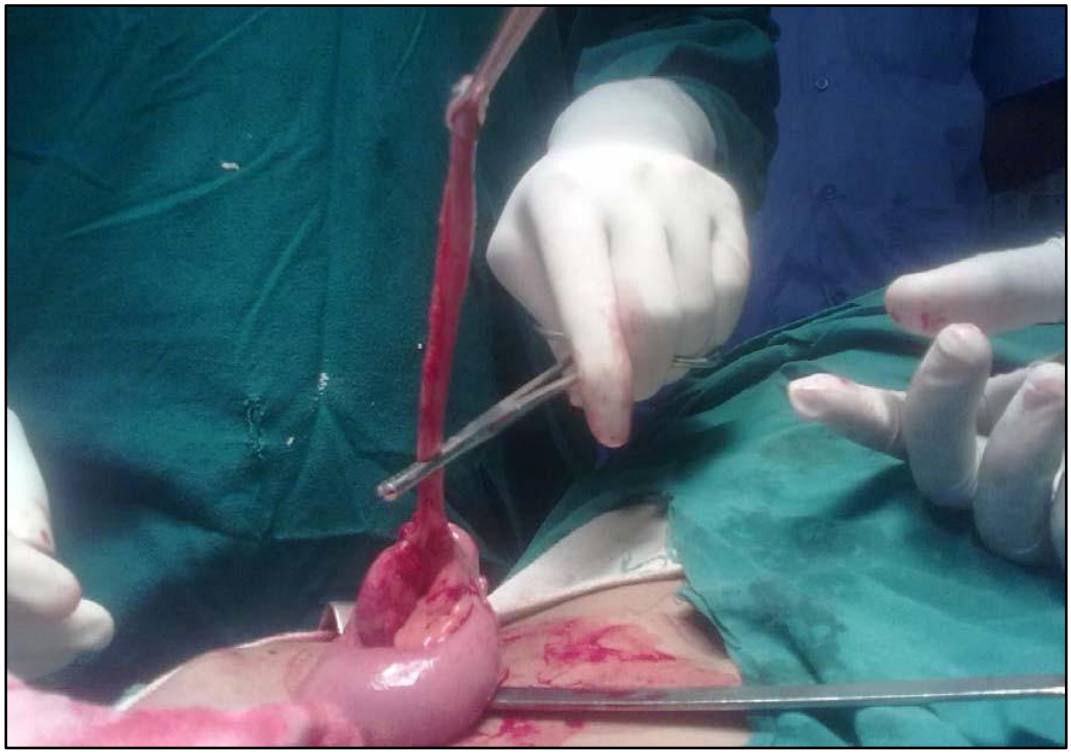
Picture 4: Peritoneum opened



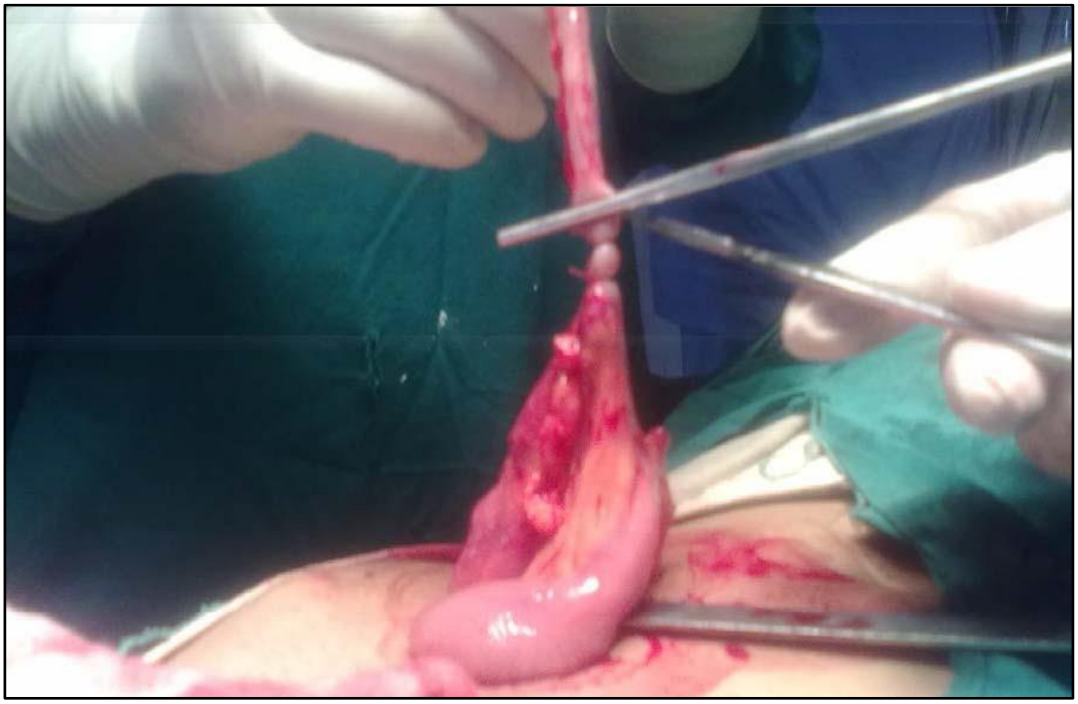
Picture 5: Caecum with anterior taenia



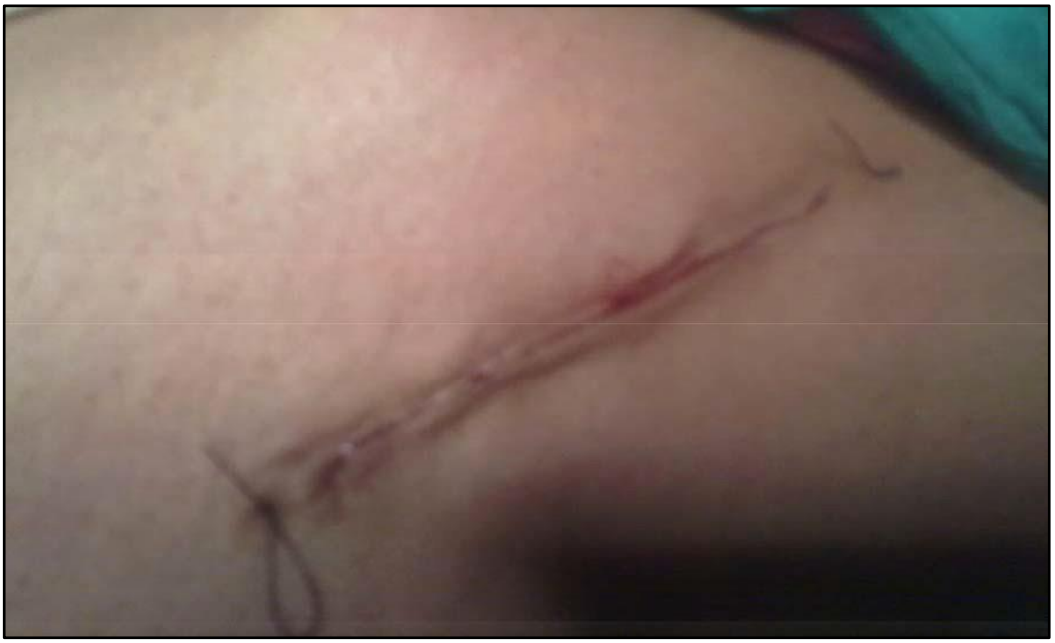
Picture 6: Appendix with mesoappendix



Picture 7: After ligation of mesoappendix, base of appendix, crushed, clamped, ligated and cut



Picture 8: Appendicectomy done



Picture 9: Abdomen incision closed in layers

ANNEXURE IV

KEY TO MASTER CHART:

M- MALE

F- FEMALE

ABD. PAIN – ABDOMINAL PAIN

PR – PULSE RATE

TEMP – TEMPERATURE

AF – AFEBRILE

FB - FEBRILE

ROV SIGN – ROVSING'S SIGN

TLC – TOTAL LEUCOCYTE COUNT

IOF - INTRAOPERATIE FINDINGS

CRP – C REACTIVE PROTEIN

HPR – HISTOPATHOLOGY REPORT

A – ABSENT

P- PRESENT

N - NEGATIVE

IA – INFLAMMED APPENDIX

PA –PERFORATED APPENDIX

GA – GANGRENOUS APPENDIX

AA –ACUTE APPENDICITIS

CA –CHRONIC APPENDICITIS

RA- RECURRENT APPENDICITIS

SL/NO	HOSP.NO	SEX	GE(YRS	ABD. PAIN	MIGRATION	ANOREXIA	VOMITING	FEVER	TENDERNESS	REBOUND TENDERNESS	GUARDING	PR	TEMP	ROV SIGN	OTHERS	TLC(T/cumm)	IOF	CRP(mg/dl)	HPR
1	14930	M	20	P	P	P	P	P	P	P	P	90	FB	P	A	17.3	GA	P	GA
2	968429	F	23	P	A	P	A	A	P	A	A	88	AF	A	A	10.9	IA	P	AA
3	968412	F	32	P	P	P	P	P	P	P	A	68	FB	P	A	15.4	IA	P	AA
4	969098	M	16	P	A	A	P	A	P	A	A	77	AF	A	A	12	IA	P	AA
5	969789	M	22	P	A	A	P	A	P	A	A	71	AF	A	A	10.9	IA	P	RA
6	969818	F	18	P	A	A	A	A	P	A	A	85	AF	A	A	5.8	IA	N	AA
7	971391	F	24	P	P	P	P	P	P	P	A	75	FB	P	A	16	IA	P	AA
8	973949	M	41	P	A	A	P	P	P	A	A	90	FB	A	A	15	IA	P	CA
9	974756	F	22	P	P	A	A	A	P	A	A	73	AF	A	A	8	IA	P	AA
10	977729	M	33	P	A	P	P	P	P	P	A	89	FB	A	A	12.1	IA	P	AA
11	977802	F	17	P	A	A	A	A	P	A	A	92	AF	A	A	4.89	IA	N	CA
12	978014	M	19	P	A	A	A	A	P	A	A	84	AF	A	A	4.3	IA	N	AA
13	980360	M	23	P	A	P	P	P	P	A	A	81	FB	A	A	11.6	IA	P	AA
14	980396	M	29	P	A	A	A	A	P	P	A	74	AF	A	A	10.7	IA	P	AA
15	982085	F	12	P	A	P	A	P	P	P	A	76	FB	P	A	18.2	IA	P	AA
16	982862	F	36	P	P	P	P	P	P	P	P	98	FB	P	A	26.8	GA	P	GA
17	983583	M	26	P	P	P	P	P	P	P	A	92	FB	A	A	14.4	IA	P	AA
18	983920	F	24	P	P	A	A	A	P	A	A	75	AF	A	A	12	IA	P	AA
19	984904	F	14	P	P	P	P	p	P	A	A	71	FB	A	A	12.5	IA	N	AA
20	987496	F	35	P	A	A	A	A	P	A	A	84	AF	A	A	9.5	IA	N	AA
21	988610	M	29	P	A	P	P	P	P	P	P	83	FB	P	A	16.1	IA	P	AA
22	989255	M	22	P	A	A	A	A	P	A	A	80	AF	A	A	7.8	IA	P	RA
23	991252	M	25	P	P	A	A	A	P	A	A	74	AF	A	A	7.4	IA	P	AA
24	992655	M	11	P	A	P	P	P	P	A	A	79	FB	A	A	12.5	IA	P	AA
25	993198	F	27	P	P	A	A	P	P	A	A	87	FB	A	A	8.2	IA	N	AA
26	993731	M	30	P	A	A	A	A	P	A	A	64	AF	A	A	14.7	IA	P	AA
27	995809	M	28	P	P	A	P	P	P	A	A	74	FB	A	A	8.9	IA	N	CA
28	997288	F	22	P	A	A	A	A	P	A	A	69	AF	A	A	7.1	IA	N	AA
29	997751	F	29	P	P	A	P	P	P	A	A	81	FB	P	A	14.7	IA	P	AA
30	998188	M	32	P	A	P	A	P	P	A	A	91	FB	P	A	16.2	IA	P	AA
31	998886	F	34	P	A	A	A	A	P	A	A	68	AF	A	A	8.7	IA	P	AA
32	998894	F	21	P	A	A	P	A	P	P	A	79	AF	A	A	9.1	IA	P	AA
33	1001613	F	13	P	A	A	P	A	P	A	A	78	AF	A	A	7.4	IA	N	RA
34	1001626	F	19	P	P	A	A	P	P	A	A	67	FB	A	A	8.6	IA	N	AA
35	1001321	F	24	P	P	A	A	A	P	A	A	77	AF	A	A	10.2	IA	P	AA
36	1010290	M	28	P	A	A	A	A	P	P	A	84	AF	A	A	7	IA	N	AA
37	1011452	M	32	P	P	P	P	P	P	A	P	97	FB	A	A	18.1	GA	P	GA
38	1012917	M	13	P	A	A	P	A	P	A	A	74	AF	A	A	10.7	IA	P	AA
39	1014709	M	24	P	P	P	P	P	P	P	P	94	FB	P	A	20.1	GA	P	GA
40	1014905	M	16	P	A	A	P	P	P	A	A	89	FB	A	A	13.4	IA	P	AA
41	1018480	M	11	P	A	P	A	P	P	P	A	87	FB	A	A	12.2	IA	P	AA
42	1018660	M	10	P	A	P	A	A	P	A	A	92	AF	A	A	11.8	IA	N	CA
43	2567	M	22	P	P	P	P	P	P	P	A	88	AF	A	A	12	IA	P	AA
44	13175	M	28	P	P	A	P	P	P	P	A	89	FB	P	A	19	IA	P	CA
45	1462	M	34	P	A	A	A	A	P	A	A	74	AF	A	A	8	IA	P	AA
46	1482	M	20	P	A	A	A	A	P	A	A	80	AF	A	A	6.3	IA	P	AA
47	12617	F	8	P	A	A	A	A	P	A	A	64	AF	A	A	8.8	IA	P	AA
48	13417	M	19	P	A	P	A	A	P	A	A	81	AF	P	A	8.3	IA	N	AA
49	1767	F	23	P	P	A	A	P	P	P	A	94	FB	A	A	12.2	IA	P	AA
50	13758	M	31	P	P	A	p	P	P	P	A	101	FB	P	A	18.1	IA	P	AA
51	16510	M	27	P	A	P	P	A	P	A	A	64	AF	A	A	10.6	IA	P	GA
52	19547	F	16	P	P	A	P	P	P	P	A	59	FB	P	A	12.2	IA	P	AA
53	517	M	25	P	A	A	A	A	P	A	A	71	AF	A	A	8.7	IA	P	AA
54	538	M	36	P	A	A	P	P	P	A	A	74	AF	A	A	7.3	IA	P	AA
55	571	F	10	P	A	A	A	A	P	P	A	82	AF	A	A	10.8	IA	N	AA
56	2045	M	39	P	P	P	P	P	P	P	P	71	FB	P	A	18	GA	P	GA
57	29428	F	12	P	A	A	A	A	P	A	A	79	AF	A	A	7	IA	P	AA
58	63064	M	14	P	A	A	P	A	P	A	A	67	AF	A	A	9.8	IA	N	AA
59	67358	M	14	P	A	A	P	A	P	A	A	83	AF	A	A	10.2	IA	P	AA
60	68336	M	24	P	A	P	P	P	P	A	A	71	FB	A	A	11.6	IA	N	AA
61	68934	M	16	P	A	A	A	A	P	A	A	68	AF	A	A	9.4	IA	P	AA
62	73535	M	23	P	A	A	P	A	P	A	A	88	AF	A	A	9.8	IA	P	AA
63	76625	M	36	P	P	P	A	P	P	P	A	89	FB	A	A	13.3	IA	P	AA
64	75697	M	13	P	A	A	P	A	P	A	A	67	AF	A	A	5.02	IA	N	AA
65	76398	F	18	P	A	A	A	P	P	A	A	75	FB	A	A	5	IA	P	AA
66	77631	F	13	P	P	P	P	A	P	P	A	84	AF	A	A	14	IA	N	AA
67	78715	F	9	P	P	P	P	P	P	P	A	93	FB	A	A	15.4	PA	P	GA
68	79146	F	15	P	A	P	P	P	P	A	A	91	FB	P	A	15.5	IA	P	AA
69	79583	F	13	P	P	A	P	P	P	A	A	63	AF	A	A	12	IA	P	AA
70	80320	F	65	P	A	A	A	A	P	A	A	76	AF	A	A	11	IA	P	AA
71	82054	M	20	P	P	P	P	P	P	P	A	89	FB	P	A	17.2	IA	P	CA
72	82063	M	20	P	P	A	P	P	P	A	A	95	FB	P	A	15.7	IA	P	AA
73	83826	M	9	P	A	A	A	A	P	A	A	71	AF	A	A	8.5	IA	N	AA
74	85788	M	11	P	P	P	P	P	P	P	A	68	FB	A	A	21	IA	P	AA
75	87062	M	43	P	A	A	P	A	P	A	A	73	AF	P	A	7.7	IA	P	AA
76	87508	M	37	P	P	P	P	P	P	A	A	65	FB	A	A	16.6	IA	P	AA
77	90239	M	23	P	P	P	P	P	P	P	A	80	AF	A	A	14.9	GA	P	PA
78	92705	M	45	P	A	A	A	A	P	P	A	90	AF	P	A	14.2	IA	P	AA
79	93648	F	16	P	A	A	A	A	P	A	A	71	AF	A	A	6.1	IA	N	AA
80	93688	F	23	P	A	A	P	A	P	A	A	75	AF	A	A	6.9	IA	P	AA
81	98157	M	13	P	P	A	A	A	P	A	A	65	AF	A	A	10	IA	P	AA
82	98359	M	19	P	P	A	P	A	P	A	A	76	FB	A	A	16.8	IA	P	AA
83	99911	F	40	P	A	P	P	P	P	A	A	93	FB	P	A	19.4	GA	P	GA
84	99969	F	28	P	P	A	P	A	P	A	A	73	AF	A	A	5.8	IA	N	AA
85	100425	M	12	P	P	P	A	P	P	P	P	101	FB	P	A	22.6	GA	P	GA
86	101947	F	38	P	A	A	P	A	P	A	A	66	FB	A	A	13	IA	N	AA
87	1019596	M	20	P	A	A	P	A	P	P	A	87	AF	A	A	9.3	IA	P	AA
88	1019971	M	28	P	A	A	A	A	P	A	A	81	AF	A	A	10.6	IA	P	AA
89	1020039	F	40	P	A	A	P	A	P	A	A	77	AF	A	A	10.9	IA	P	AA
90	1020136	M	32	P	A	A	A	A	P	P	A	73	AF	A	A	9.6	IA	N	AA
91	1020297	M	26	P	A	A	P	A	P	A	A	84	AF	A	A	8.4	IA	N	AA
92	102031	F	12	P	A	A	P	A	P	A	A	89	AF	A	A	8.3	IA	P	AA
93	5465	M	23	P	A	A	A	A	P	A	A	61	AF	A	A	6.8	IA	P	AA
94	20726	F	30	P	P	A	P	A	P	P	A	91	AF	A	A	9.9	IA	P	RA
95	31217	M	13	P	A	A	P	A	P	A	A	79	AF	A	A	8.2	IA	P	AA
96	37645	M	27	P	A	A	P	A	P	A	A	90	FB	A	A	9.2	IA	P	AA
97	108690	M	28	P	A	A	A	A	P	A	A	62	AF	A	A	7.1	IA	P	CA
98	37773	F	30	P	A	A	A	A	P	A	A	75	AF	A	A	7.7	IA	N	CA
99	41004	F	23	P	P	P	P	A	P	P	P	102	FB	A	A	27	IA	P	AA
100	42470	F	35	P	P	A	P	P	P	A	A	87	AF	A	A	15.6	IA	P	AA
101	171195	F	20	P	P	A	P	P	P	P	P	70	FB	P	A	17.3	PA	P	PA
102	108380	M	23	P	A	A	A	A	P	A	A	86	AF	A	A	10.9	IA	P	AA
103	151695	F	32	P	A	A	P	A	P	P	A	64	FB	P	A	15.4	IA	P	AA
104	152049	F	16	P	A	A	P	A	P	A	A	75	AF	A	A	12	IA	P	CA
105	146726	M	22	P	A	A	A	A	P	A	A	71	AF	A	A	10.9	IA	P	AA
106	172719	F	38	P	A	A	A	A	P	A	A	84	AF	A	A	5.8	IA	N	AA
107	77707	M	24	P	P	P	P	A	P	P	P	72	AF	A	A	16	IA	P	AA
108	158412	M	41	P	A	A	P	A	P	A	A	67	AF	A	A	15	GA	N	GA
109	172334	M	22	P	A	A	A	A	P	A	A	81	AF	A	A	8	IA	P	CA
110	158019	F	33	P	P	P	P	P	P	A	A	90	AF	P	A	12.1	IA	P	AA
111	152041	M	17	P	A	A	A	A	P	A	A	80	AF	A	A	4.89	IA	N	AA
112	168791	F	19	P	A	A	A	P	P	P	A	74	FB	A	A	4.3	IA	N	AA
113	176302	F	23	P	A	P	P	A	P	A	A	78	AF	P	A	11.6	IA	P	CA
114	151219	M	29	P	A	A	P	A	P	A	A	68	AF	A	A	10.7	IA	P	AA