“HYPERBILIRUBINEMIA- A NEW DIAGNOSTIC MARKER IN ACUTE APPENDICITIS AND APPENDICULAR PERFORATION.”

By

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Dissertation submitted to

THE BLDE UNIVERSITY BIJAPUR, KARNATAKA

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY

In

GENERAL SURGERY

Under the guidance of

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ACKNOWLEDGMENT

अखण्डमण्डलाकारंव्याप्तंयेनचराचरम्।

तत्पदंदर्शितंयेनतस्मैश्रीगुरवेनमः

Salutations to that respected Guru who showed us the place of the one who pervades the vast universe with all its movable and immovable things.

It is the most appropriate that I begin by expressing my gratitude to the Almighty for having blessed me to pursue Postgraduate study in General Surgery. I dedicate this study to the God.

I also dedicate this dissertation to my mother and father for their unconditional love, extra care and for providing the strength and time for my studies.I sincerely thank my grandfather **Shri A.B Patil** who wished me to join this college and also i thank my uncle, aunt, brothers , sisters and my family for their unconditional support in all my deeds.

It gives me immense pleasure to express my deep gratitude, respect and sincere thanks to my esteemed guide **Dr. VIJAYA PATIL** M.S., Professor in Department of General Surgery, BLDEUS Shri B M Patil medical college Hospital and Research centre Bijapur. Her insight, high caliber and personal qualities have been profoundly inspirational to me, not only for this study but for the whole of my post graduation and shall continue to be so in the future. I thank her for his expertise in guidance and preparation of this dissertation. I also thank her for sharing her knowledge, especially during our PG teaching program. Thank you mam for everything.

It is with great respect, I acknowledge **Dr.Tejaswini Vallabha** M.S., Professor and Head of Department of General Surgery, BLDEUS Shri B. M. Patil medical college hospital and research centre, Bijapur for her continuous support and his constant effort in the upliftment for our academics.

My special thanks to My Professors **DR. ARAVIND PATIL, DR B.B. METAN, DR. M.B PATIL, DR.M.S.KOTENNAVAR** who continuously stood as the inspiration to study and shared their knowledge and guided me for the academics.

I am grateful to my Associate Professors of Surgery **Dr.Basavaraj Narasanagi, Dr. Hemanth Kumar, Dr. Girish Kulloli, Dr.Ramakanth Baloorkar, Dr. B.P. Kattimani** for sharing their knowledge and guiding me throughout the course.

I am grateful to my Assistant Professors **Dr. Prasad Sasnur Dr.Vikram Sindagikar, Dr.Deepak Chavan, Dr. Ravi Pattar**, **Dr. Y.D.Badiger,, Dr. Basavaraj Badadal and Dr. Dayanand Biradar, Dr. Sanjay Namdar,** and my Senior Residents, **Dr. Santosh Patil, Dr. Ravindra Nidoni, Dr. Prasanna Kamble** for their advice and help.

I am thankful and grateful to **Dr.M S Biradar,** Principal of BLDEU's Shri B.M. Patil Medical College Hospital and Research Centre for permitting me to utilize the hospital resources during my study period.

I am thankful to my seniors **batch 2010 and 2011 and all my juniors of batch 2013 and 2014 for their valuable help and advice.**

I thank my fellow post graduates **Dr. Harshavardhan Biradar, Dr. Bharat S. Dr. Sachin Kadlewad Dr. Rakshit Aggarwal, Dr. Sunil Kumar** &

Dr. Aniketan.K.V, for their companionship, help and valuable advice throughout these three years.

I express my thanks to Mr.S.B.Madagi , Mr.Yadrami, Mrs.Vijaya Statisticians, for his services in preparing my dissertation.

I also thank Mr. Ashok Palke, Mr. Prakash, Mr.subhash Madagond for their valuable help throughout the course.

Lastly would like to thank nursing staff and the patients who cooperated and helped for my study and i also thank PREETI NET ZONE for the digital work.

Dr. RAVI .A. ICHALAKARANJI

LIST OF ABBREVIATIONS USED

ALP	-	Alkaline phosphatase
ALT	-	Alanine transaminase
AST	-	Aspartate transaminase
ATP	-	Adenosine triphosphate
cm	-	Centimeter(s)
CRP	-	C-reactive protein
CT	-	Computed tomography
dL	-	Deciliter(s)
DLC	-	Differential leukocyte count
E. Coli	-	Escherichia coli
ELISA	-	Enzyme linked immunosorbent assay
G	-	Gram(s)
HbsAg	-	Hepatitis B surface antigen
IL-6	-	Interleukin-6
LFT	-	Liver function tests
mg	-	Milligram(s)
mL	-	Milliliter(s)
mm	-	Millimeter(s)
n	-	Total number
NPV	-	Negative predictive value
OR	-	Odds ratio
PPV	-	Positive predictive value
SB	-	Serum bilirubin
SGOT	-	Serum glutamic oxaloacetic transaminase
SGPT	-	Serum glutamic pyruvic transaminase
Sr.	-	Serum
TLC	-	Total leukocyte count
TNF	-	Tumor necrosis factor
TSB	-	Total serum bilirubin
USG	-	Ultrasonography
WBC	-	White blood cells

ABSTRACT

Background and Objectives

Acute appendicitis is the most common abdominal emergency encountered in general surgery.In most of the cases,the diagnosis can be made clinically by assessing the symptoms and physical findings and confirmed by laboratory tests and ultrasonography.

However,diagnosis is difficult sometimes even after all these tests and in such doubtful cases either the diagnosis is missed or patients normal appendix is operated on,leading to increase in mortality and morbidity.

The present study was undertaken to assess relationship between hyperbilirubinemia and acute appendicitis and to evaluate its credibility as a diagnostic marker for acute appendicitis and also, to see whether elevated bilirubin levels have a predictivepotential for the diagnosis of appendiceal perforation.

Methodology

A cross sectional study was conducted in the Department of Surgery, BLDEU'S Shri B M Patil Medical college Hospital and Research centre Bijapur during the period of October 2012 to May 2014 A total of 250 patients with clinical diagnosis of acute appendicitis or appendiceal perforation were studied. The serum bilirubin and liver function tests were carried out in all the patients.

Results:

In the present study of the 250 patients enrolled for the study, 149 patients (60%) were males while the remaining 101 patients (40%) were females.The mean age in our study population (250 patients) was 31.45 ± 13.85 years. Hyperbilirubinemia (> 1.0 mg/dL) in our study was found in 125 patients (50%) of all

the 250 patients (n=250) enrolled in the study, while 125 patients (50%) had normal bilirubin levels (≤ 1.0 mg/dL).

Sensitivity and Specificity of bilirubin in predicting acute appendicitis and appendiceal perforation diagnosis was 53.2% and95% respectively. Similarly Positive predictive value and Negative predicative value of bilirubin in predicting acute appendicitis and appendiceal perforation diagnosis was 99.2% and 8.26% respectively.

The Odd's ratio was calculated to be 0.045%.

Keywords

Acute Appendicitis; Appendiceal perforation; Hyperbilirubinemia; Serum Bilirubin

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INTRODUCTION

Acute appendicitis is the most common abdominal emergency encountered in general surgery. The diagnosis of appendicitis can be difficult, occasionally taxing the skills of even the most experienced surgeon.Addiss and associates[1] estimated the incidence of acute appendicitis in the United States population to be 11 cases per 10,000 populations annually. The disease is slightly more common in males, with a male: female ratio of 1.4:1. In a lifetime, 8.6% of males and 6.7% of females can be expected to develop acute appendicitis.Young age is a risk factor, as nearly 70% of patients with acute appendicitis are less than 30 years of age. The highest incidence of appendicitis in males is in the 10- to 14-year-old age group (27.6 cases per 10,000 population), while the highest female incidence is in the 15- to 19-year-old age group (20.5 cases per 10,000 population). Patients at extremes of age are more likely to develop perforated appendicitis. Overall, perforation was present in 19.2% of cases of acute appendicitis.

This number was significantly higher, however, in patients under 5 and over 65 years of age. Although less common in people over 65 years old, acute appendicitis in the elderly progresses to perforation more than 50% of the time.[1]In most of the cases, the diagnosis can be made clinically by assessing the symptoms and physical findings and confirmed by laboratory tests and ultrasonography.However, diagnosis is difficult sometimes even after all these tests and in such doubtful cases either the diagnosis is missed or patients normal appendix is operated on, leading to increase in mortality and morbidity.[2]No reliably specific marker for acute appendicitis has been identified till now. A raised white cell count is not specific for appendicitis and although C-reactive protein is commonly used in the assessment of suspected appendicitis, its specificity varies markedly between studies and may only

significantly raise once appendiceal perforation takes place.[3]Cases presenting with non-specific abdominal pain and acute appendicitis are extremely common in general surgery, accounting for about 75% of admissions due to acute abdominal complaints. Also, the rate of negative appendectomies in these cases is about 30%, leading to increased morbidity and risk of incisional hernia.Whereas delayed diagnosis and treatment of patients with acute appendicitis may lead to several complications that are potentially life threatening, such as perforation, peritonitis, sepsis, small bowel obstruction, urinary retention and abdominal abscess formation.Recently, elevation in serum bilirubin was reported, but the importance of the raised total has not been stressed in acute appendicitis and appendiceal perforation.

The endotoxin of Escherichia coli has been shown in vivo to affect physiological bile flow, which led to the theory that hyperbilirubinemia may possess inferential potential in the preoperative early diagnosis of appendix perforation[4]Elevated Serum bilirubin level will help in the early and accurate diagnosis of acute appendicitis and in predicting its serious complications, most importantly the perforation.

Thus the need for the study is to conclude whether the serum bilirubin can be considered as a new laboratory marker to aid in the diagnosis of acute appendicitis and if so, does it have the predictive capacity to warn us about Appendicular perforation.

OBJECTIVES

1. To study the relationship between hyperbilirubinemia and acute appendicitis and to evaluate its credibility as a diagnostic marker for acute appendicitis.
2. To evaluate whether elevated Bilirubin levels have a predictive potential for the diagnosis of Appendicular perforation.

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE:

The first descriptions of the appendix date to the sixteenth century.[5–7] Although first sketched in the anatomic notebooks of Leonardo da Vinci around 1500, the appendix was not formally described until 1524 by da Capri[8] and 1543 by Vesalius.[9]In 1554 the French physician Jean Fernel (1497-1558) reported the first case of perforative appendicitis at autopsy.10

A classical post-mortem description is owed to Lorenz Heister (1683-1758), professor of medicine and also a practising surgeon at the universities ofAltdorf-Nürnberg and Helmstedt in Germany (1712). Heister was the first tostudy the pathology of appendicitis (1711).11

The 19th century pathological concept is based on the notion´perityphilitis´, that is inflammation of the cecum (typhlon, blind). The cecum rather than the appendix was considered as the site of the disease; this is easilyexplained by advanced stages of inflammation which were observed in autopsies.Surgery for appendicitis

The first appendicectomy was performed at St. George's Hospital,London, in 1736 by Claudius Amyand, a surgeon at St. George's Hospital inLondon and Sergeant Surgeon to Queen Ann, King George I, and King GeorgeII. The acutely inflamed appendix, perforated by a pin, and surrounding omentumwas removed through a scrotal wound while dealing with a faecal fistula in achronic scrotal hernia. The patient was 11-year-old boy and patient recovered.12The first published account of appendicectomy for appendicitis was by

Krönlein in 1886. However, the patient died two days postoperatively.Fergus, in Canada, performed the first elective appendicectomy in1883.13Charles McBurney(1845-1913) was one of the surgeons pioneering thediagnostics and

operative treatment of appendicitis. McBurney’s classic report onearly operative interference in cases of appendicitis was presented before theNew York Surgical Society in 1889. In it he described the area of greatestabdominal pain in this disease process, now known as McBurney’s point.Five years later in 1894, he set forth in another paper the incision that heused in cases of appendicitis, now called McBurney’sincision.However, McBurney later credited McArthur with first describing thisincision.14

The US surgeon John Benjamin Murphy introduced and popularized earlyremoval of the appendix in all cases of suspected appendicitis. In 1904 hedescribed the triad of pain in abdomen, vomiting and fever, which remains asound basis for diagnosis even today.15

Dawbarn suggested the use of a purse string suture, placed around thebase of the appendix. In 1889, Senn first drew attention to the risks of ligatureslipping off the appendix stump with subsequent peritoneal contamination.On 13 September 1983 the gynaecologist Professor Kurt Semmperformed the world's first laparoscopic appendicectomy at the University ofKielin Germany.16

EMBRYOLOGY

Embryologically, the appendix and cecum develop as outpouchings of the caudal limb of the midgut loop in the sixth week of human development. By the fifth month, the appendix elongates into its vermiform shape. At birth, the appendix is located at the tip of the cecum, but due to unequal elongation of the lateral wall of the cecum, the adult appendix typically originates from the posteromedial wall of the cecum, caudal to the ileocecal valve.

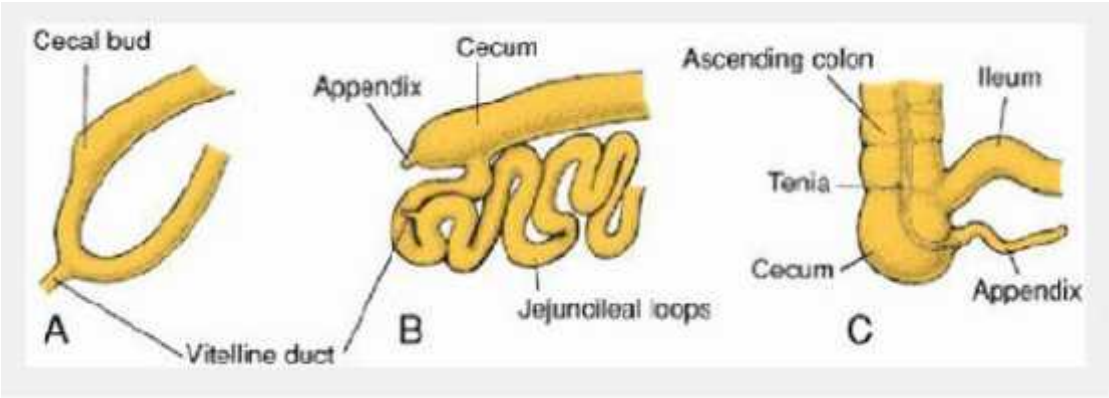


FIG 1.Successive stages in development of the caecum and appendix.
A. 7 weeks. B. 8 weeks. C. Newborn.

CONGENITAL ABNORMALITIES:

Congenital abnormalities[32] of the appendix are:

1. Congenital absence
2. Duplication or triplication
3. Variation in positions
4. Congenital diverticulum / band of appendix.

1. Congenital absence:

Robinson (1952) in reporting a case of congenital absence of the appendix was able to collect only 68 other examples, a figure sufficiently indicative of the greater rarity of this condition.

2. Duplication / Triplication of Appendix:

It is extremely rare anomaly reviewed by Khanna, fewer than 100 cases have been reported.

Wall bride (1962) classified duplication into three types-

Type A- Partial duplication of single caecum

Type B- Single caecum with two completely separate appendices. This is further subdivided into-

B1-„Bird like appendix" because of its resemblance to the normal arrangement in birds where there are two appendices symmetrically placed on either side of the ileocaecal valve.

B2- One appendix arises from the usual site on the Caecum, with another rudimentary appendix arising from caecum along the line of one of the taenia coli.

TYPE C- There are two caeci each bearing one appendix.

Tincker described an unique case of a triple appendix, associated with a double penis and ectopiavesicae.

3. Variation in position:

Due to the developmental changes in caecum, midgut loop and caecal mesentery the following different variations may be seen.

Incomplete downward descent of Caecum may cause appendix in subhepatic position. Over growth of the ascending colon may cause appendix down to pelvic position with Caecum.

Incomplete or non-rotation or the midgut loop may cause the appendix on the left side of the abdomen. It may be associated with transposition of the viscera.

Caecum may have a mesentery and be mobile. Because of its mobility appendix may take variable positions in abdomen.

Congenital diverticulum / band of appendix:

Congenital diverticulum differs from acquired one, by having a muscular coat in its wall. Some diverticulae originate from the vitellointestinalduct and caecum develops at the point of attachment of the duct. In such cases the diverticulum is attached to the umbilicus by a fibrous band. Apart from the band, a ring may be found upto the umbilicus called the „appendiculo ovarian ligament" .

ANATOMY

The appendix averages 9 cm in length,[17] with its outside diameter ranging from 3–8 mm and its lumen ranging from 1–3 mm. The base of the appendix is consistently found by following the teniae coli of the colon to their confluence at the base of the cecum. The appendiceal tip, however, can vary significantly in location. Sir Frederick described the various positions of the appendix comparing the position with the face of a clock[18].

- 11 O clock(0.2%)- Para colic (lies in the sulcus on the lateral aspect of the caecum).
- 12 O clock(65.28%)- Retrocaecal (lies behind the caecum and may be totally or partially retroperitoneal)
- 1 O clock(1%)- Pre-ileal
- 2 O clock(0.2%)- Post ileal
- 3 O clock(0.05%)- Promonteric (the tip of the organ points towards the promontory of the sacrum).
- 4 O clock(31.01%)- Pelvic (Appendix dips into the pelvis).
- 6 O clock(2.26%)- Subcaecal or midinguinal or mid Poupart

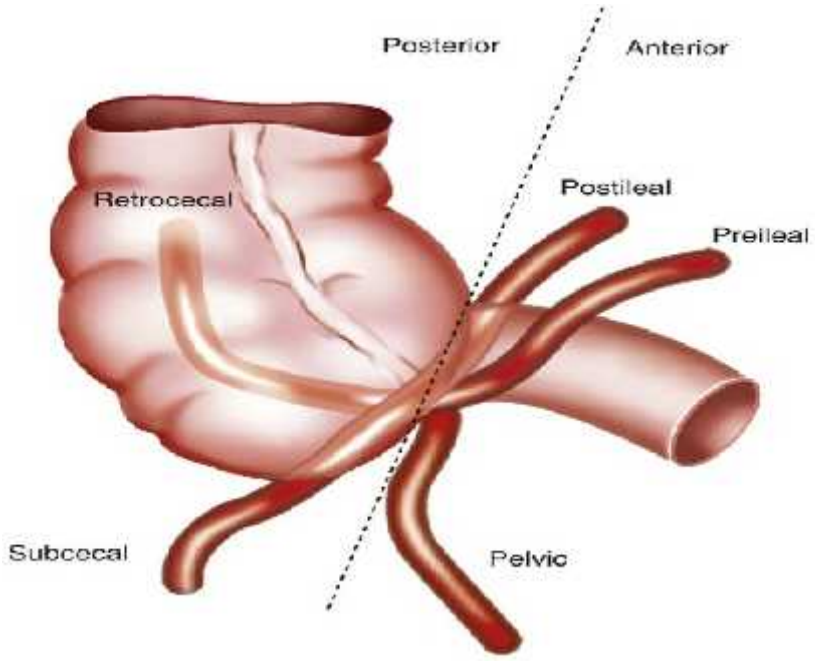


FIG 2.Various Position of Appendix.

Vascular Supply

Is byAppendiceal artery, a branch from the lower division of theileocolic artery, runs behind the terminal ileum and enters the mesoappendix ashort distance from the appendiceal base. Here it gives off a recurrent branch,which anastomoses at the base of the appendix with a branch of the posteriorcaecal artery.

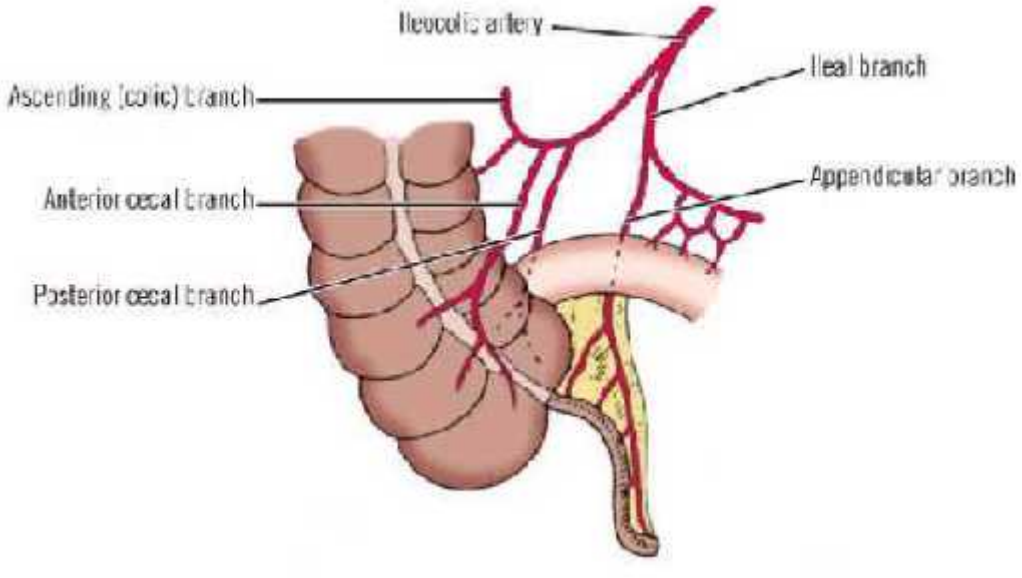


FIG. 3 Blood supply for appendix

Appendiceal Veins:

The appendix is drained via one or more appendiceal veins into theposterior caecal or ileocolic vein and thence into the superior mesenteric vein.

Lymphatic drainage:

Lymphatic vessels in the appendix are numerous: there is abundantlymphoid tissue in its walls. From the body and apex of the appendix 8 to 15vessels ascend in the mesoappendix, and are occasionally interrupted by one ormore nodes. They unite to form three or four larger vessels which run into thelymphatic vessels draining the ascending colon, and end in the inferior andsuperior nodes of the ileocolic chain.

Innervation

The appendix and overlying visceral peritoneum are innervated by sympathetic and parasympathetic nerves from the superior mesenteric plexus.Visceral afferent fibres carrying sensation of distension and pressure mediate thesymptoms of pain felt during the initial stages of appendiceal inflammation. Inkeeping with other

structures derived from the midgut, these sensations arepoorly localized initially, and referred to the central (periumbilical) region of theabdomen.

Mesoappendix

The mesentery of the appendix is a triangular fold of peritoneum aroundthe vermiform appendix. It is attached to the posterior surface of the lower end ofthe mesentery of the small intestine close to the ileocaecal junction. It usuallyreaches the tip of the appendix but sometimes fails to reach the distal third, inwhich case a vestigial low peritoneal ridge containing fat is present over the distalthird. It encloses the blood vessels, nerves and lymph vessels of the vermiformappendix, and usually contains a lymph node.

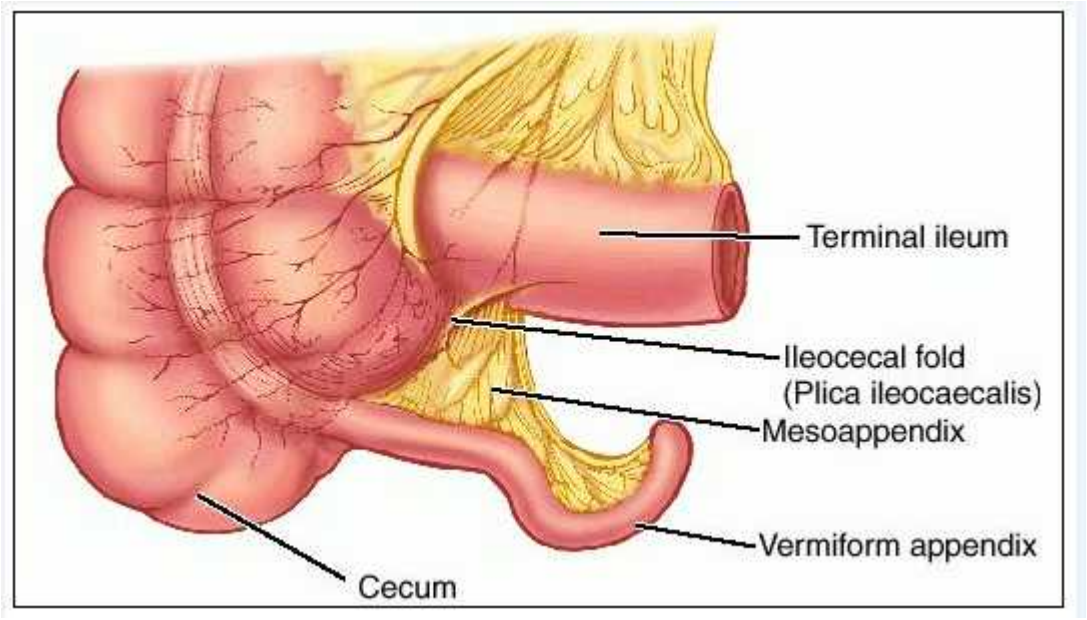


FIG 4.Mesoappendix

Mucosa

The mucosa is covered by a columnar epithelium , and M cells are presentin the epithelium that overlies the mucosal lymphoid tissue. Glands (crypts) arefewer in number and thus less densely packed. They penetrate deep into thelymphoid tissue of the mucosal lamina propria.

Sub-Mucosa

The submucosa typically contains many large lymphoid aggregates thatextend from the mucosa and obscures the muscularis mucosae layer: consequentlythis becomes discontinuous. These aggregates also cause the mucosa to bulgeinto the lumen of the appendix, so that it narrows irregularly . They are absent atbirth but accumulate over the first 10 years of life to become a prominent feature.The submucosal lymphoid tissue frequently exhibits germinal centres within itsfollicles, indicative of B-cell activation, as it is in secondary lymphoid tissueelsewhere. In adults, the normal layered structure of the appendix is lost and thelymphoid follicles atrophy and are replaced by collagenous tissue. In the elderly,the appendix may be filled with fibrous scar tissue.

MuscularisExterna

The muscularisexterna has outer longitudinal and inner circular layers ofsmooth muscle. The longitudinal fibres form a continuous layer but, with theexception of the uniform outer muscle layer of most of the appendix,macroscopically these are aggregated as longitudinal bands or taeniae coli. At thebase of the appendix, the longitudinal muscle thickens to form rudimentarytaeniae that are continuous with those of the caecum and colon. Between thetaeniae coli the longitudinal layer is much thinner, less than half the circular layer in thickness.

Serosa

The serosa forms a complete covering, except along the mesentericattachment. The longitudinal muscular fibres form a complete layer of uniformthickness, except over a few small areas where both muscular layers are deficient,leaving the serosa and submucosa in contact.

FUNCTIONS OF THE APPENDIX

The human vermiform appendix is usually referred to as a vestigial organ with no known function. On the contrary currently available evidences suggest that the appendix is highly specialized part of alimentary tract.

Postulated functions of the appendix[19]:

1. Exocrine: There have been suggestions that the appendix in human has an exocrine function, assisting in digestion of plant foods. However the 2 ml of clear fluid secreted containing mucin, amylase and proteolytic enzymes per day in low concentrations cannot have any effect on food stuffs in the caecum and food stuffs wouldn‟t ideally enter the appendix for processing.
2. Endocrine: The neuroendocrine cells and their secretory products in the appendix have not shown to have any selective endocrine functions.
3. Neuromuscular: It has been suggested that, the appendix may be the pacemaker for synchronized contraction and emptying that side of the bowel.
4. Lymphoid: The amount of the lymphoid tissue in the appendix is equal to that in the ascending, transverse and descending colon. There is a relative increases in IgM, IgA and IgG containing lymphocytes in the lamina propria of the appendix.

Stowens claims that the appendix is not a vestigial organ but has the same function as the thymus and possible function as a mammalian equivalent of the bursa of fabricus has been suggested.

Pathophysiology

Wangensteen extensively studied the structure and function of the appendix and the role of obstruction in appendicitis.[20,21]Based on anatomic studies, he postulated that mucosal folds and a sphincterlike orientation of muscle fibers at the appendiceal orifice make the appendix susceptible to obstruction. He proposed the following sequence of events to explain appendicitis:

1. closed loop obstruction is caused by a fecalith and swelling of the mucosal and submucosal lymphoid tissue at the base of the appendix;
2. intraluminal pressure rises as the appendiceal mucosa secretes fluid against the fixed obstruction;
3. increased pressure in the appendiceal wall exceeds capillary pressure and causes mucosal ischemia; and
4. luminal bacterial overgrowth and translocation of bacteria across the appendiceal wall result in inflammation, edema, and ultimately necrosis. If the appendix is not removed, perforation can ensue.

Although appendiceal obstruction is widely accepted as the primary cause of appendicitis, evidence suggests that this may be only one of many possible etiologies. First, some patients with a fecolith have a histologically normal appendix.[22,23,24] Moreover, the majority of patients with appendicitis show no evidence for a faecolith. Arnbjornsson and Bengmark[25] studied at laparotomy the appendixes of patients with suspected appendicitis. They found the intraluminal pressure of the appendix prior to removal to be elevated in only 8 of 27 patients with non-perforated appendicitis. They found no signs of obstruction in the remaining patients with non-perforated appendicitis, as well as all patients with a normal appendix. Taken together, these studies imply that obstruction is but one of the possible etiologies of acute appendicitis.

Bacteriology

The principal organisms seen in the normal appendix, in acute appendicitis, and in perforated appendicitis are *Escherichia coli* and *Bacteroides fragilis.*[26–29.]Appendicitis is a polymicrobial infection, with some series reporting the culture of up to 14 different organisms in patients with perforation.[26]

Aerobic and Facultative	Anaerobic
Gram-negative bacilli	Gram-negative bacilli
Escherichia coli	*Bacteroides fragilis*
Pseudomonas aeruginosa	Other *Bacteroides* species
Klebsiella species	*Fusobacterium* species
Gram-positive cocci	Gram-positive cocci
Streptococcus anginosus	*Peptostreptococcus* species
Other *Streptococcus* species	Gram-positive bacilli
Enterococcus species	*Clostridium* species

Pathology:

At the earliest stages, only a scanty neutrophilic exudate may be found throughout the mucosa, submucosa, and muscularispropria. Subserosal vessels are congested, and often there is a modest perivascular neutrophilic infiltrate. The inflammatory reaction transforms the normal glistening serosa into a dull, granular, red membrane; this transformation signifies early acute appendicitis for the operating surgeon. At a later stage, a prominent neutrophilic exudate generates a fibrinopurulent reaction over the serosa.

As the inflammatory process worsens, there is abscess formation within the wall, along with ulcerations and foci of suppurative necrosis in the mucosa. This state constitutes acute suppurative appendicitis.

Further vascular compromise leads to large areas of hemorrhagic green ulceration of the mucosa and green-black gangrenous necrosis through the wall, extending to the serosa, creating acute gangrenous appendicitis, which is quickly followed by rupture and suppurative peritonitis.

The histological criterion for the diagnosis of acute appendicitis is neutrophilic infiltration of the muscularispropria. Usually, neutrophils and ulcerations are also present within the mucosa. Since drainage of an exudate into the appendix from alimentary tract infection may also induce a mucosalneutrophilic infiltrate, evidence of muscular wall inflammation is requisite for the diagnosis.

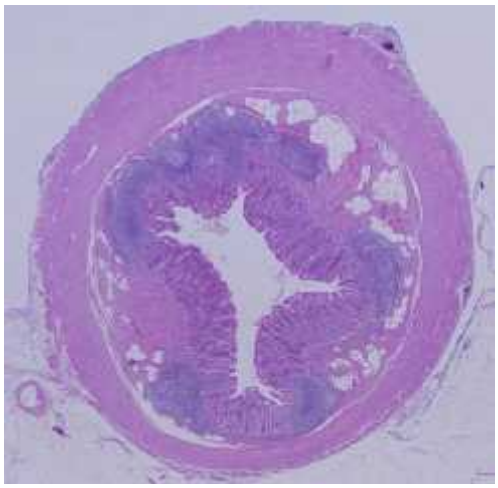


FIG 5 histology of normal appendix **FIG 6 histology of acute appendicitis**

Clinical Presentation:

The classic presentation of acute appendicitis begins with crampy, intermittent abdominal pain, thought to be due to obstruction of the appendicular lumen. The pain may be either periumbilical or diffuse and difficult to localize. This is typically followed shortly thereafter with nausea; vomiting may or may not be present. If nausea and vomiting precede the pain, patients are likely to have another cause for their abdominal pain, such as gastroenteritis. Classically, the pain migrates to the right lower quadrant as transmural inflammation of the appendix leads to inflammation of the peritoneal lining of the right lower abdomen. This usually occurs within 12–24 hours of the onset of symptoms. The character of the pain also changes from dull and colicky to sharp and constant. Movement or Valsalvamaneuver often worsens this pain, so that the patient typically desires to lie still; some patients describe pain with every bump in the car or ambulance ride to the hospital. Patients may report low-grade fever up to 101°F (38.3°C). Higher temperatures and shaking chills should again alert the surgeon to other diagnoses, including appendicular perforation or non-

appendicular sources. When questioned, patients who have appendicitis commonly report anorexia; appendicitis is unlikely in those with a normal appetite.

Perforated Appendicitis:

When acute appendicitis has progressed to appendicular perforation, other symptoms may be present. Patients will often complain of two or more days of abdominal pain, but their duration of symptoms may be shorter, as previously discussed. The pain usually localizes to the right lower quadrant if the perforation has been walled off by surrounding intra-abdominal structures including the omentum, but it may be diffuse if generalized peritonitis ensues. The pain may be so severe that patients do not remember the antecedent colicky pain. Patients with perforation often have rigors and high fevers to 102°F (38.9°C) or above. A history of poor oral intake and dehydration may also be present.

Diagnosis

History and Physical Examination:

Many patients with acute appendicitis do not have a classic history. Because the differential diagnosis of appendicitis is extensive, patients should be queried about certain symptoms that may suggest an alternative diagnosis. Surgeons must also remember that a previous appendectomy does not definitively exclude the diagnosis of appendicitis, as "stump appendicitis" (appendicitis in the remaining appendiceal stump after appendectomy), although rare, has been described.[30]

On inspection, patients look mildly ill and may have slightly elevated temperature and pulse. They often lie still to avoid the peritoneal irritation caused by movement. The surgeon should systematically examine the entire abdomen, starting in the left upper quadrant away from the patient's described pain. Maximal tenderness is typically in the right lower quadrant, at or near McBurney's point, located one-third

of the way from the anterior superior iliac spine to the umbilicus. This tenderness is often associated with localized muscle rigidity and signs of peritoneal inflammation, including rebound, shake, or tap tenderness. Right lower quadrant tenderness is the most consistent of all signs of acute appendicitis;[31,32] its presence should always raise the specter of appendicitis, even in the absence of other signs and symptoms. Because of the various anatomic locations of the appendix, however, it is possible for the tenderness to be in the right flank or right upper quadrant, the suprapubic region, or the left lower quadrant. Patients with a retrocecal or pelvic appendix may have no abdominal tenderness whatsoever. In such cases, rectal examination can be helpful to elicit right-sided pelvic tenderness

Physical examination:

Various signs:

1. *The pointing sign:* The patient is then asked to point to where the pain began and where it moved.
2. *Rovsing's sign:* Pain in the right lower quadrant on palpation of the left lower quadrant, is further evidence of localized peritoneal inflammation in the right lower quadrant
3. *Psoas sign:* Pain with flexion of the leg at the right hip, can be seen with aretrocecal appendix due to inflammation adjacent to the psoas muscle.
4. *The obturator sign:* Pain with rotating the flexed right thigh internally,indicates inflammation adjacent to the obturator muscle in the pelvis.

Laboratory Studies:

Laboratory studies can be helpful in the diagnosis of appendicitis, but no single test is definitive.

White Blood Cell Count (WBC):

A White Blood Cell count (WBC) is perhaps the most useful laboratorytest. The white blood cell count is elevated with more than 75% neutrophils inmost patients. A completely normal leukocyte count and differential is found inabout 10% of patients with acute appendicitis. A high white blood cell count(>20,000/mL) suggests complicated appendicitis with either gangrene orperforation.[33]

The clinician must remember, however, that the WBC count can benormal in patients with acute appendicitis, particularly in early cases. Serial WBCmeasurements improve the diagnostic accuracy, with a rising value over timecommonly seen in patients with appendicitis.[34]

C-reactive protein:

C-reactive protein (CRP) is an acute-phase reactant synthesized by theliver in response to infection or inflammation and rapidly increases within thefirst 12 hours. CRP has been reported to be useful in the diagnosis ofappendicitis; however, it lacks specificity and cannot be used to distinguishbetween sites of infection. CRP levels of greater than 1 mg/dl are commonlyreported in patients with appendicitis, but very high levels of CRP in patientswith appendicitis indicate gangrenous evolution of the disease, especially if it isassociated with leukocytosis and neutrophilia.However, CRP normalization is known to occur 12 hours after onset ofsymptoms. Several prospective studies have shown that in adults who have hadsymptoms for longer than 24 hours, a normal CRP level has a negative predictivevalue of 97-100% for appendicitis.[35-37]Multiple studies have been done evaluating the sensitivity of CRP levelalone for the diagnosis of appendicitis in patients selected to undergo appendicectomy. Gurleyik et al noted a CRP sensitivity of 96.6% in 87 of 90patients with histologically proven disease.[38]

Urine analysis

Urinalysis is performed to diagnose other potential causes for abdominalpain, specifically urinary tract infection and ureteral stone. Significant hematuriawith colicky abdominal pain suggests ureterolithiasis, and testing directed at thisdiagnosis is indicated. A urinary tract infection, on the other hand, is notuncommon in patients with appendicitis. Its presence does not exclude thediagnosis of acute appendicitis, but it should be identified and treated. Althoughpyuria suggests urinary tract infection, it is not uncommon for the urinalysis in apatient with appendicitis to show a few white blood cells solely due to inflammation of the ureter by the adjacent appendix. In certain patient populations, other laboratory tests are indicated. In women of childbearing age, the urine human chorionic gonadotropin should be checked to alert the clinician to the possibility of ectopic or concurrent pregnancy. Ectopic pregnancy is another cause of right lower quadrant pain that demands emergent diagnosis and treatment.

Imaging Studies

The potential imaging modalities for diagnosis of acute appendicitis include plain radiographs, ultrasound, and computed tomography.

Plain radiographs

Prior to the wide-spread use of modern imaging techniques, plain abdominal films were often obtained in patients with abdominal pain, and a right lower quadrant faecolith (or appendicolith) was considered pathognomonic for acute appendicitis.[39]A calcified appendicolith is visible on plain films in only10% to 15% of patients with acute appendicitis. Studies show that faecoliths arenot pathognomonic for appendicitis, as some patients with abdominal pain and faecolith have a normal

appendix. In addition, faecoliths are not common enough in patients with appendicitis to be used as a reliable sign.

As a result, plain abdominal radiographs are neither helpful nor cost effective and are not recommended for the diagnosis of acute appendicitis.

Plain abdominal films may be useful for the detection of ureteral calculi, small bowel obstruction, or perforated ulcer, but such conditions are rarely confused withappendicitis.[40]

Ultrasonography (USG)

Among patients with abdominal pain, *Abdominal ultrasonography* has a sensitivity of about 85% and a specificity of more than 90% for the diagnosis of acute appendicitis.[41]

Sonographic findings consistent with acute appendicitis include:

1. Appendix of seven mm or more in antero-posterior diameter,
2. A thick-walled, non- compressible luminal structure seen in cross sectionreferred to as a *target lesion.*
3. Increased echogenicity of the surrounding fat signifying inflammation, or
4. Presence of an appendicolith
5. In more advanced cases, peri-appendiceal fluid or a mass may be found. Ultrasonography has the advantages of being a noninvasive modality requiring no patient preparation that also avoids exposure to ionizing radiation. For these reasons, it is commonly used in children and in pregnant patients with equivocal clinical findings suggestive of acute appendicitis. Disadvantage of ultrasonography is that it is highly operator-dependent, and it is frequently unable to visualize the normal appendix.[42]

Pelvic ultrasound can be especially useful in excluding pelvic pathology,such as tubo-ovarian abscess or ovarian torsion, that may mimic acuteappendicitis.[43]

Computed tomography

Computed tomography (CT) is commonly used in the evaluation of adult patients with suspected acute appendicitis, especially so in the elderly. CT benefits has a high diagnostic accuracy for appendicitis,[44]and visualization and diagnosis of many of the other causes of abdominal pain that can be confused with appendicitis. Improved imaging techniques, including the use of 5-mm sections, have resulted in increased accuracy of CT scanning,[45]which has a sensitivity of about90% and a specificity of 80% to 90% for the diagnosis of acute appendicitis among patients with abdominal pain. Controversy remains as to the importance of intravenous, oral gastrointestinal, and rectal contrast in improving diagnostic accuracy. In general, CT findings of appendicitis increase with the severity of the disease. Classic findings include a distended appendix greater than seven mm in diameter and circumferential wall thickening, which may give the appearance of a halo or target. As inflammation progresses, one may see periappendiceal fat stranding, edema, peritoneal fluid, phlegmon, or a periappendiceal abscess. CT detects appendicoliths in about 50% of patients with appendicitis and also in asmall percentage of people without appendicitis. Among patients with abdominalpain, the positive predictive value of the finding of an appendicolith on CT remains high at about 75%.

In prospective studies, CT demonstrated a sensitivity of 0.94 and a specificity of 0.95.CT thus has a high negative predictive value, making it particularly useful in excluding appendicitis in patients for whom the diagnosis is in doubt. Appendicitis is highly unlikely if enteric contrast fills the lumen of the appendix and no surrounding inflammation is present. The clinician must remember, however, that a CT performed

early in the course of appendicitis might not show the typical radiographic findings. The rational approach is – the selective use of CT scanning.

Laparoscopy

Although most patients with appendicitis will be accurately diagnosed based on history, physical exam, laboratory studies, and if necessary, imaging techniques, there are a small number in whom the diagnosis remains elusive. For these patients, diagnostic laparoscopy can provide both a direct examination of the appendix and a survey of the abdominal cavity for other possible causes of pain. Laparoscopy can serve as both a diagnostic and therapeutic maneuver for patients with acute abdominal pain and suspected acute appendicitis. Laparoscopy is probably most useful in the evaluation of females with lower abdominal complaints, because appendicectomy is performed on a normal appendix in as many as 30 to 40% of these patients. Differentiating acute gynecologic pathology from acute appendicitis can be effectively accomplished using the laparoscope.[47]

Barium enema studies

In the past, barium enema examination was used to diagnose appendicitis However in the era of ultrasonography and CT scanning, barium enema study has absolutely no role in the diagnosis of acute appendicitis.

Scoring Systems

A number of clinical and laboratory-based scoring systems have been devised to assist diagnosis. The most widely used is the Alvarado score. A score of seven or more is strongly predictive of acute appendicitis.

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

Liver Function Tests

Importance of hyperbilirubinemia or elevated Serum Bilirubin (serumbilirubin) and its association in acute appendicitis has being postulated recently. It is hypothesized that an association exists between hyperbilirubinemia and acute appenditics and its complications such as appendicular perforation.[48]

Bilirubin

Bilirubin (a tetrapyrrole, formerly referred to as hematoidin) is the endproduct of the metabolic degradation of haem, prosthetic group of haemoglobin,myoglobin, the cytochrome P450s and various other haemo-proteins.[49]The serumlevel of bilirubin represents the balance between production and excretion(destruction) of this breakdown product. Laboratory evaluation of serum bilirubin allows detection in two forms

1. Indirect or Unconjugated bilirubin (i.e. before hepatic metabolism)
2. Direct or Conjugated (i.e. after hepatic metabolism)[50]

Since bilirubin is potentially toxic waste product, hepatic handling is designed to eliminate it from the body via biliary tract. There are various steps involved in this process namely; hepatocellular uptake, intracellular binding, conjugation and excretion.49Modern analytical methods document that normal plasma contains virtually no bilirubin conjugate. The 10 to 20% of the bilirubin in normal plasma that gives rise prompt (Diazo) reaction is an artifact of kinetic of the Van Den Berg reaction which with along various modifications is the method most commonly used to quantitate bilirubin in clinical laboratories. Indeed, when direct reacting fraction is less than 15% of total bilirubin at virtually any total bilirubin concentration, the bilirubin in the sample can beconsidered as essentially all unconjugated.[51]

Conjugated bilirubin (mono- and di-glucronide) is excreted across canalicular plasma membrane into the canaliculus by an ATP dependant transport process mediated by a canalicular membrane protein called multi-drug resistantassociated-protein-2. The canalicular transport mechanism of excretion of bilirubin conjugate is very sensitive to injury. Accordingly, in hepatocellular disease, as well as with either cholestasis or mechanical obstruction to the bileduct, bilirubin conjugates within the hepatocytes, prevented from taking their normal pathway into the canaliculi and down the bile duct, may reflux into bloodstream, resulting in mixed or less often a truly conjugated hyperbilirubinemia.[51]

Hyperbilirubinemia occurs either due to cholestatic, hepatocellular or haemolytic diseases. Cholestatic and hepatocellular hyperbilirubinemia are associated with a rise in liver enzymes. In these cases the bilirubin is predominantly conjugated

in type (mixed type). An isolated rise in serum bilirubin (without enzyme elevation) may be familial or due to hemolysis.

Cholestasis is the failure of normal bile to reach duodenum. This may be due to pathology anywhere between the hepatocyte and ampulla of Vater. Intrahepatic cholestasis includes those conditions where there is no demonstrable obstructionto major bile duct. The causes are drugs, hormones, primary billiary cirrhosis andsepsis.[52]Sepsis reaches to the liver by various routes but one of the commonest routes is through portal vein from the gastro-intestinal tract. Any inflammatory condition may cause transmigration/translocation of bacteria; its toxin orcytokines may cause suppression of hepatocellular function and reduced excretion of bile from billiary canaliculi.[53]

Pathophysiology behind the elevation of Serum bilirubin in acute appendicitis:

Both increased bilirubin production and alteration in bilirubin clearance can lead to bilirubin accumulation and may be involved in the hyperbilirubinaemia observed in patients with appendix perforation.

It has frequently been demonstrated that several bacterial infections accompanying hepatic dysfunction, to the extent that anomalies in bile flow and bile acid production arise as a result.

These patients together with those who have extrahepatic bacterial infections demonstrated cholestasis, induced by nitric oxide and a proinflammatory cytokine via detrimental hepatocellular & bile duct formation

In addition, the most common bacterial species cultured from the appendix walls of patients with acute appendicitis are *Escherichia coli* and *Bacteroides fragilis*, two species that inhibit microcirculation and cause sinusoidal damage as shown in rat liver model.

Lipopolysaccharides associated with *Escherichia coli* can affect hepatocyte uptake and bile acid secretion and further research involving a rat liver model has shown that Escherichia coli cause a dose dependent cholestasis disorder.

In addition, *Escherichia coli* infection leads to regular hemolysis of erythrocytes, increased bilirubin load and, perhaps, the development of hyperbilirubinaemia may be a consequence of this mechanism.[54]

Literature review

1) A retrospective study conducted in department of general & visceral surgery, academic teaching hospital of the Ruhr University, Bochum,Germany found elevated bilirubin in all patients in the range of 0.1-4.3mg/dl, while patients with appendiceal perforation had bilirubin in the range of 4.0-4.3 mg/dl.
2) In a study conducted by Department of Surgery, Nepalgunj Teaching Hospital, Nepalgunj, Nepal, found elevated Total serum bilirubin in 87% of cases. The mean of elevated serum bilirubin was 2.26mg/dl and in patients with gangrenous or perforated appendix; elevation of TSB was found to be much higher. The specificity, sensitivity was 100%, 82.07% respectively with positive predictive value of 100% and negative predictive value of 17.3%.
3) In a retrospective study done in USC Medical Centre, Los Angeles found elevated Bilirubin levels in 38% of cases and patients with Gangrene/perforation were significantly more likely to have hyperbilirubinemia than those with Acute Appendicitis. The odds of appendiceal perforation are three times (odds ratio 2.6) for patients with hyperbilirubinemia compared to those with normal bilirubin levels.

4) In a retrospective study done in St Luke's Hospital, Kilkenny, Ireland and South Tipperary Hospital, Clonmel, Ireland found hyperbilirubinemia had a specificity of 88% and a positive predictive value of 91% for acute appendicitis. The specificity for perforation or gangrene was 70%.
5) In a study done inPostgraduate Institute of Medical Education and Research, Dr Ram ManoharLohia Hospital (Dr RMLH and PGIMER), New Delhi, India by PorasChaudhary, Ajay Kumar, NeerajSaxena, Upendra C. Biswal found Total serum bilirubin including both direct and indirect was found to be significantly increased in case of acute suppurative appendicitis. Serum bilirubin was much higher (P <0.000) in cases of gangrenous/perforated appendicitis.
6) In a study done in Department of Surgery D, Herlev Hospital, University of Copenhagen, Herlev, Denmark by J. Burcharth, H. C. Pommergaard, J. Rosenberg, I. Gögenur found Bilirubin was significantly higher in patients with appendiceal perforation compared with patients with appendicitis without perforation. Elevated serum bilirubin had a sensitivity ranging from 0.38 to 0.77 and a specificity ranging from 0.70 to 0.87 in predicting appendiceal perforation.
7) In A prospective study of 70 cases evaluating the role of hyperbilirubinemia in acute inflammation of appendix done by NitinWasnik, Vijay P Agrawal, T. Dihare, Kunal R and Jitendra.YDepartment of General Surgery, NKP Salve Institute of Medical Sciences and LataMangeshkar Hospital, Nagpur, India foundOut of 70 cases, 41 were males and 29 were females. Their age ranged from 12 years to 60 years with average of 26.9 years. Duration of symptoms ranged from

1 day to maximum 8 days. All the cases diagnosed as acute appendicitis clinically. Per operatively, all cases had inflamed appendix. Among 70 cases, SB was raised in 42 (60%) cases where as 28 (40%) cases had normal SB level. It ranged from 1.2 mg/dL to 4.0 mg/dL. The average level of SB was 1.648 mg/dL.

8) A study done by Young Ran Hong, Chul-Woon Chung, Jong Woo Kim, Chang Il Kwon,[1] DaeHoAhn, Sung Won Kwon, and Seong Ki Kim in KOREA found Hyperbilirubinemia is a statistically significant diagnostic marker for acute appendicitis and the likelihood of perforation.

9) A study done by Mohammad Vaziri, AbdolrezaPazouki, Zeinab Tamannaie, Farshid Maghsoudloo, Mohadeseh Pishgahroudsari, Shahla Chaichian Minimally Invasive Surgery Research Center, Tehran University of Medical Sciences, Tehran, Iran found Eighty patients who underwent open appendectomy including 70% men and 30% women with a mean age of 34±11 years in Group I (perforated appendicitis) and 47.5% women and 52.5% men with a mean age of 33±14 in Group II (simple appendicitis) were included in this study. The mean bilirubin levels were higher for patients with perforated acute appendicitis compared to those with a non-perforated simple appendicitis (1.04±05 mg/dl vs 0.7±0.1 mg/dl) and this difference is highly significant ($p<0.01$).

METHODOLOGY

SOURCE OF DATA:

- All patients admitted with clinical diagnosis of acute appendicitis or appendicular perforation under general surgery in BLDE UNIVERSITY'S SHRI B M PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, BIJAPUR were taken as subjects for this study.
- After taking the proposed informed consent data were collected using the questionnaire / proforma.
- The primary data for this study were the blood investigations of the patients.

METHOD OF COLLECTION OF DATA:

The following tests were carried out for patients diagnosed as acute appendicitis or appendicular perforation under general surgery and admitted to BLDE UNIVERSITY'S SHRI B.M.PATIL MEDICAL COLLEGE,HOSPITAL AND RESEARCH CENTRE. BIJAPUR:

1. Complete blood count,
2. Serum bilirubin (total &direct bilirubin),
3. C-reactive protein,
4. Liver enzymes (SGPT, SGOT, ALP),
5. Seropositivity for HbsAg and HCV,
6. Ultrasonography of abdomen and pelvis.

INCLUSION CRITERIA:

- All patients diagnosed as acute appendicitis clinically on admission.
- All patients diagnosed as appendicular perforation clinically on admission.

  For both these groups, only patients with histopathological report suggestive of appendicitis would be included

EXCLUSION CRITERIA:

- All patients documented to have a past history of Jaundice or liver disease.
- Chronic alcoholism (i.e. intake of alcohol of >40gm/day for men and >20 gm/day in women for 10 years).
- All patients with hemolytic diseases.
- All patients with acquired or congenital biliary diseases.
- All patients who are HbsAg& HCV positive.
- All patients with bilirubin metabolism syndromes like Gilbert's syndrome, Criggler Najjar syndrome, Rotor syndrome, Dubin Johnson syndrome.
- Patients on Hepatotoxic drugs.

RESEARCH HYPOTHESIS:

Hyperbilrubinemia, as a new biochemical marker in acute appendicitis and also it has role in predicting Appendicular perforation.

SAMPLING:

Study period from: October 2012 to May 2014

All the patients admitted during this period, who fulfilled the inclusion criteria, were included in this study.

Study design: Cross – sectional study

Sample size: 250

Estimation of sample size :

Following formula to be used to estimate the sample size for the findings of bilirubin levels in acute appendicitis.

$$n = \frac{z^2_{\alpha/2} pq}{e^2}$$

$$= \frac{1.96^2 * 0.576 * 0.424}{0.06126^2}$$

$$= 250$$

here e – the permissible error (value of estimator – value of parameter, e =0.06126)

$z_{\alpha/2}$ - be the critical value of z distribution at 5% level of significance.

p –be the incidences of acute appendicitis (p = 0.576 with reference to the article by International journal of collaborative research on Internal medicine and public health vol. 1 No. 5 (July 2009).

$q = 1 - p$

Procedure

Ethical clearance for the study was obtained from Institutional Ethics Committee BLDE UNIVERSITY'S SHRI B M PATIL MEDICAL COLLEGE, HOSPITALAND RESEARCH CENTREBIJAPUR. Based on the selection criteria patients admitted with clinical diagnosis of acute appendicitis orappendiceal perforation under Department of Surgery, BLDE UNIVERSITY'S SHRI B M PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE BIJAPUR during the study period were screened for eligibility. The eligible patients were briefed about the nature of the study and a written informed consent was obtained from the consented patients. Thorough history was taken and clinical examination was done for all patients and findings were recorded on predesigned and pretested proforma. These investigations are required as routine before taking any patient for **appendicectomy:**

1. Complete blood count.
2. Urine Routine
3. Serum bilirubin (total &direct bilirubin).
4. C-reactive protein
5. Liver enzymes (SGPT, SGOT, ALP).
6. Seropositivity for HbsAg and HCV.
7. Ultrasonography of abdomen and pelvis.
8. Serum creatinine

Reference Range of Serum Bilirubin and Liver Enzymes

Test		Normal Range
Serum Bilirubin	Total	0.3 - 1.0 mg/dL
	Direct	0.1 – 0.4 mg/dL
	Indirect	0.1- 0.6mg/dL
Liver Enzymes	SGPT	0 – 35 U/L
	SGOT	0 – 35 U/L
	ALP	30 – 120 U/L

STATISTICAL ANALYSIS

The data obtained was tabulated on Microsoft excel spreadsheet and analysed as below.

- Patients with clinical diagnosis of acute appendicitis having hyperbilirubinemia were expressed in percentage as

$$= \frac{\text{Patients with clinical diagnosis of acute appendicitis with elevated Serum bilirubin level}}{\text{All patients with clinical diagnosis of acute appendicitis}}$$

- Mean of the level of elevation of Serum bilirubin was calculated for patients with clinical diagnosis of acute appendicitis.
- Patients with clinical diagnosis of appendicular perforation having hyperbilirubinemia were expressed in percentage as;

$$= \frac{\text{Patients with clinical diagnosis of appendicular perforation with elevated Serum bilirubin}}{\text{All patients with clinical diagnosis of appendicular perforation}}$$

- Mean of the level of elevation of serum bilirubin were calculated for patients with clinical diagnosis of appendicular perforation.
- A hypothesis was made based on the observation of the level of the two means.
- Also, sensitivity, specificity, positive predictive value.

Sensitivity : $\frac{a}{a+c} \times 100$

Specificity = $\frac{d}{b+d} \times 100$

Positive predictive value : $\frac{a}{a+b} \times 100$

Negative predictive value : $\frac{d}{c+d} \times 100$

Odds ratio : $\frac{ad}{bc}$

RESULTS

The present one year cross sectional study was conducted in the Department of Surgery, BLDE UNIVERSITY'S SHRI B M PATIL MEDICAL COLLEGE AND RESEARCH CENTRE BIJAPUR during the period of October 2012 to May 2014.

A total of 250 patients with clinical diagnosis of acute appendicitis or appendiceal perforation were enrolled in the study and studied.

Table 1 Sex Distribution

Sex	Number	Percentage
Male	149	59.06%
Female	101	40.04%
Total	250	100%

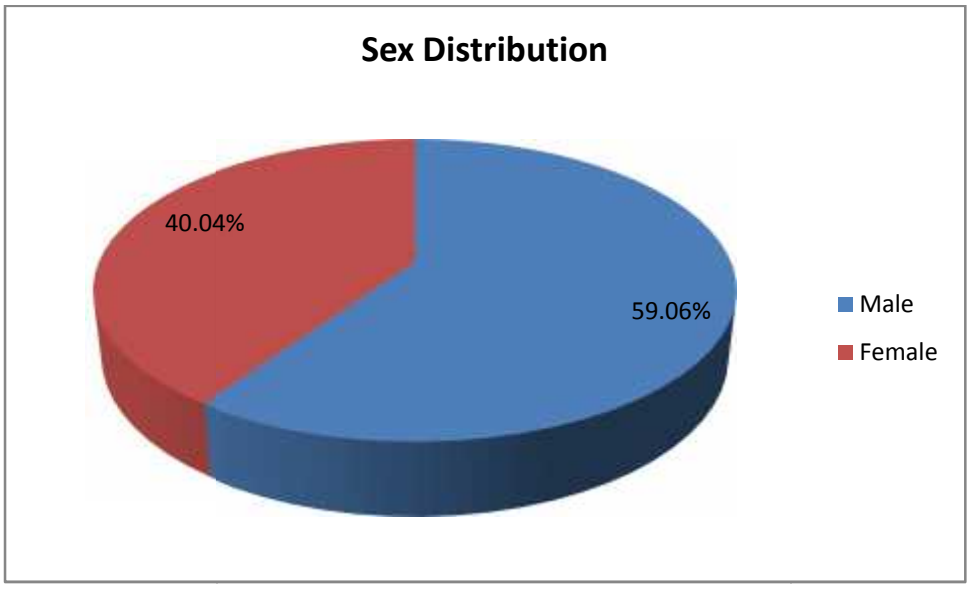


Table 2 (A)Age Distribution

Age	Male	Female	Total
1 – 10	4	2	6
10 – 20	25	17	42
20 – 30	53	34	87
30 – 40	28	20	48
40 – 50	17	15	32
50 – 60	17	11	28
60 – 70	5	2	7
Total	149	101	250

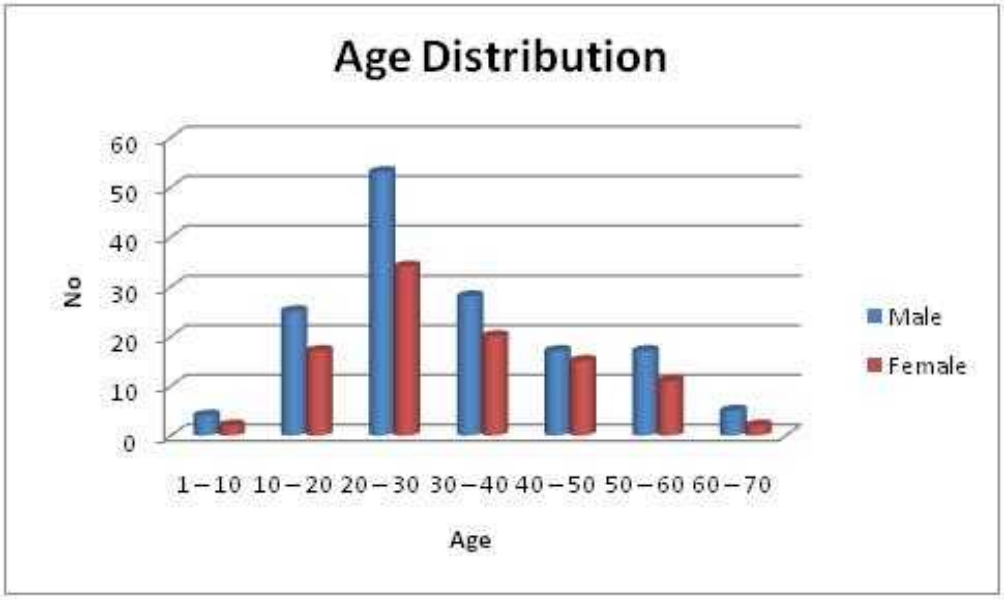


Table 2 (B)AgeDistribution

Sex	Mean	SD
Male	31.59	14.05
Female	31.24	13.62
Overall	31.45	13.85

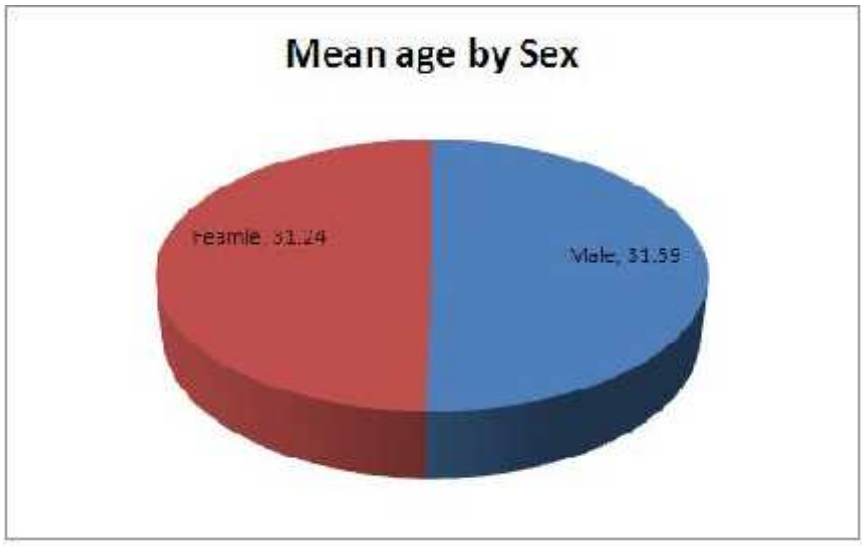
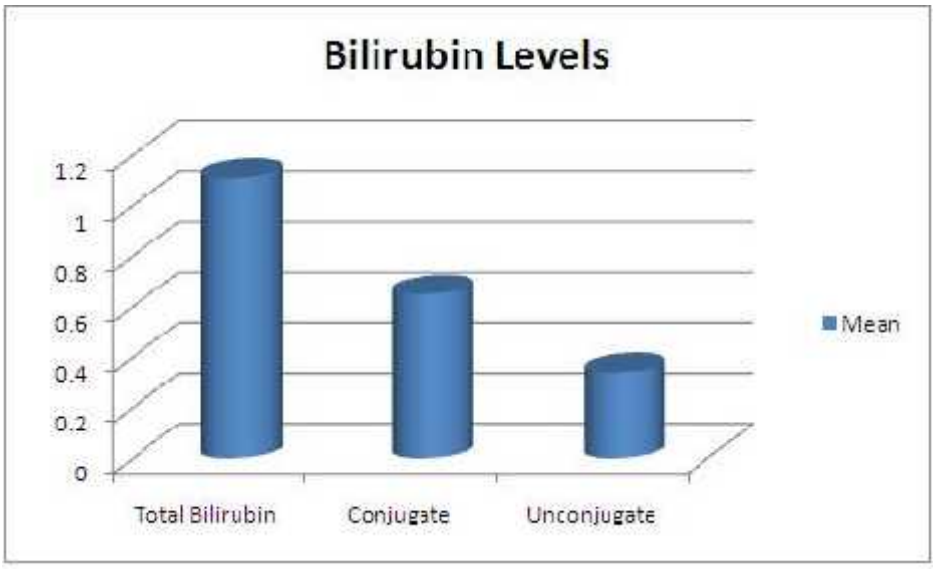


Table 3 Liver Function Tests

Parameters	Mean	SD
Total Bilirubin	1.11	0.45
Conjugate	0.65	0.23
Unconjugate	0.34	0.12
SGOT	25.36	7.03
SGPT	20.52	4.85
ALP	36.94	10.96



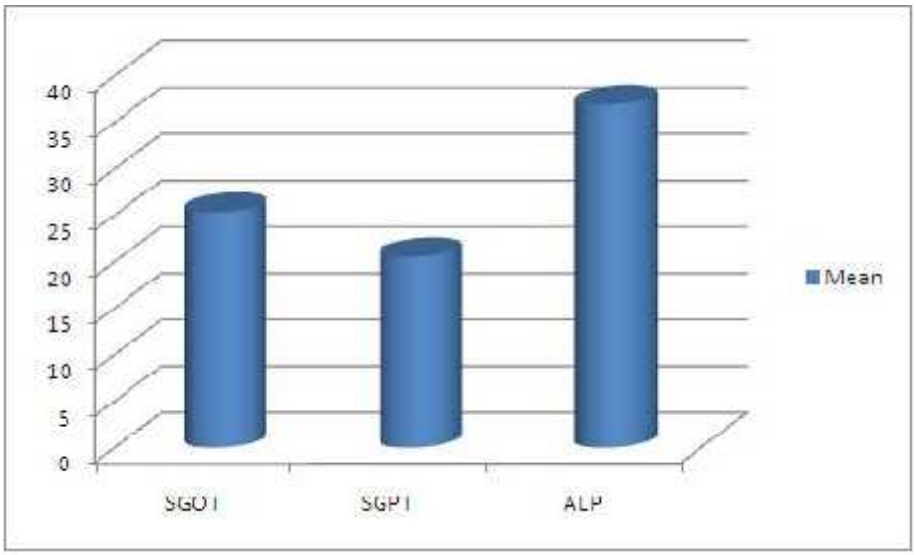


Table 4 Total Bilirubin levels in all patients (N=250)

Total Bilirubin	Number	Percentage	Clinical		Intraoperative		Ultrasound	
			AA	AP	AA	AP	AA	Q
< = 1.0	125	50%	125	0	125	0	60	65
> 1.0	125	50%	108	17	105	20	113	12
Total	250	100%						

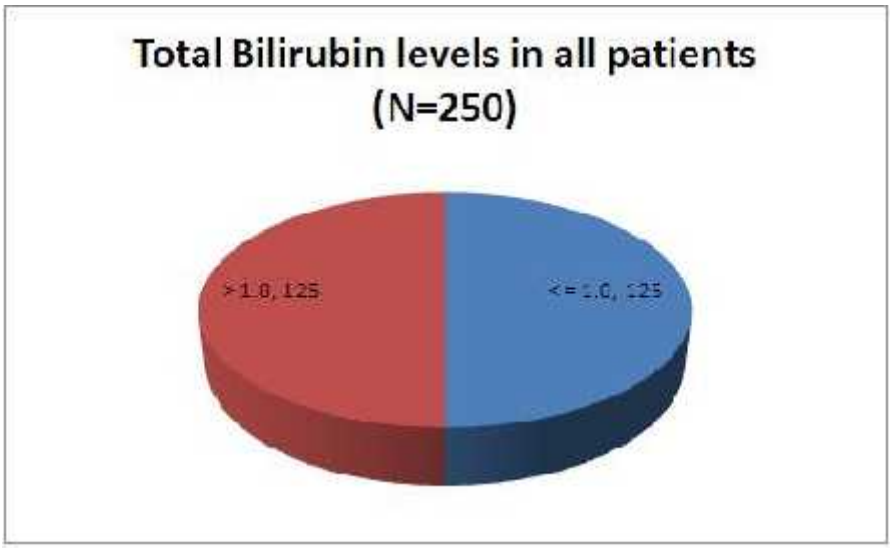


Table 5.No.of patients with raised conjugated and unconjugated bilirubin.

Total bilirubin >1.0	125
Conjugated bilirubin >0.4	125
Unconjugated bilirubin >0.6	35

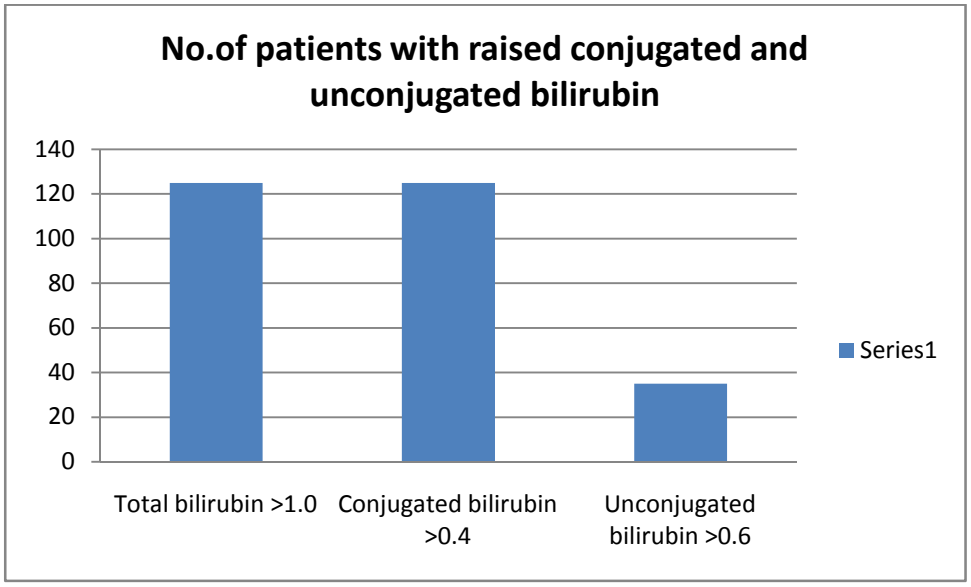


Table 6 (A) Bilirubin levels in patients with acute appendicitis diagnosis (Clinical)

Total Bilirubin	Number	Percentage
< = 1.0	125	53.6%
> 1.0	108	46.4%
Total	233	100%

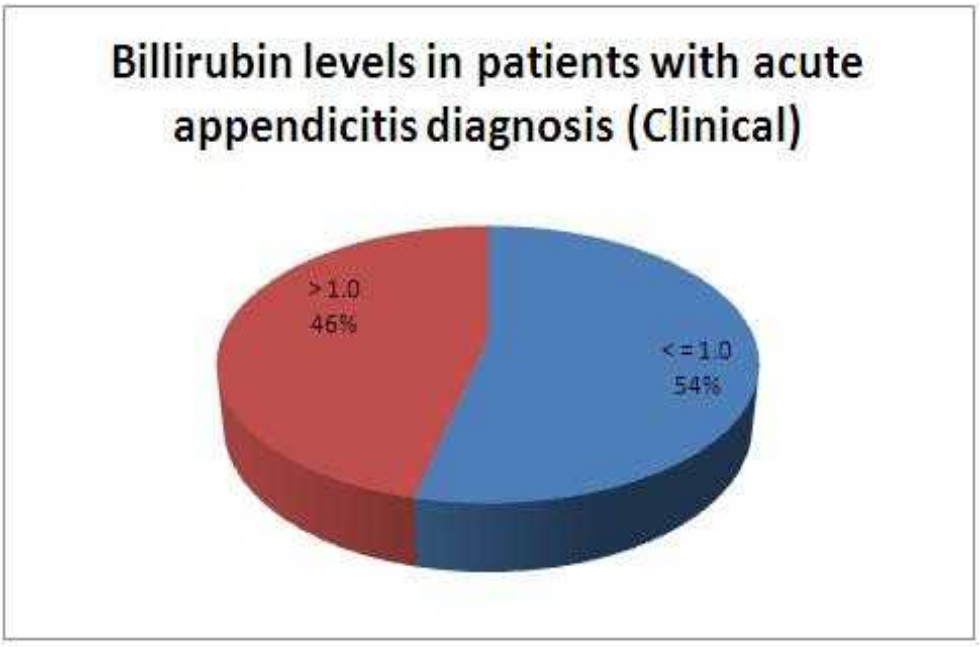


Table 6 (B) Bilirubin levels in patients with acute appendicitis diagnosis (Intraoperative)

Total Bilirubin	Number	Percentage
< = 1.0	125	54.4%
> 1.0	105	45.6%
Total	230	100%

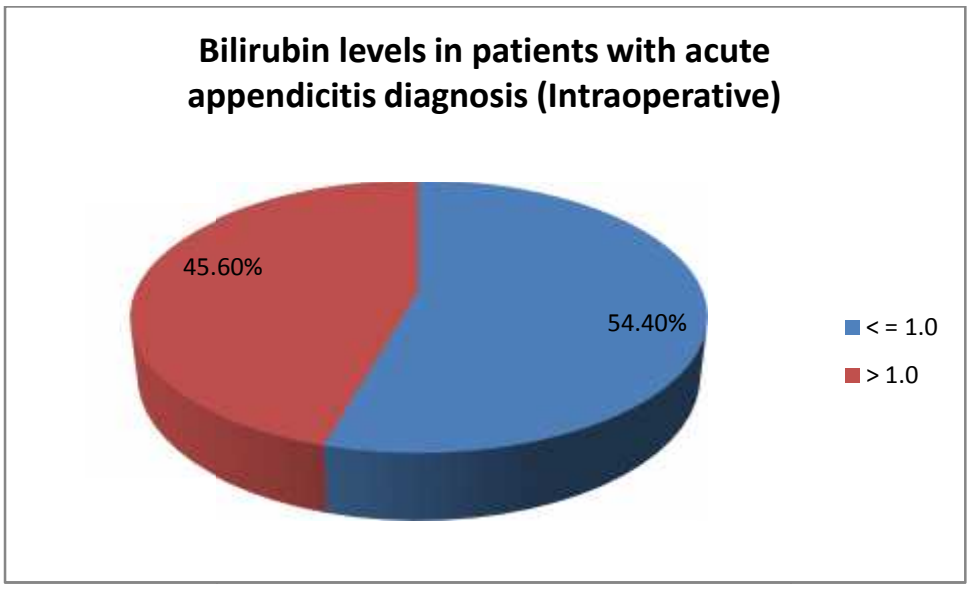


Table7 (A) Bilirubin levels in patients with appendicular perforation diagnosis (Clinical)

Total Bilirubin	Number	Percentage
< = 1.0	0	0%
> 1.0	17	100%
Total	17	100%

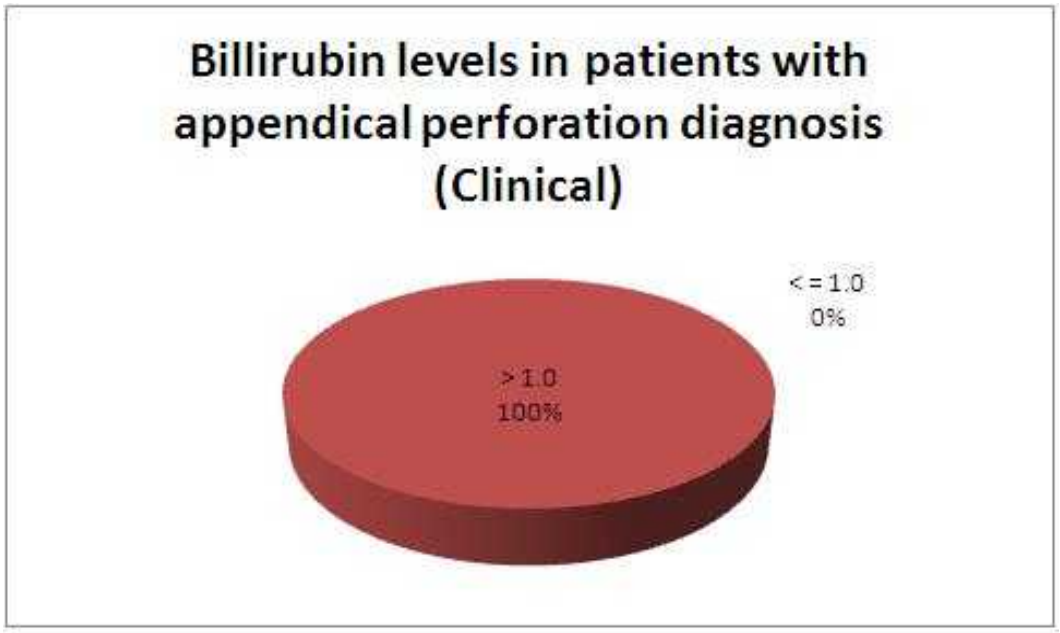


Table7 (B) Bilirubin levels in patients with appendicular perforation diagnosis (Intraoperative)

Total Bilirubin	Number	Percentage
< = 1.0	0	0%
> 1.0	20	100%
Total	20	100%

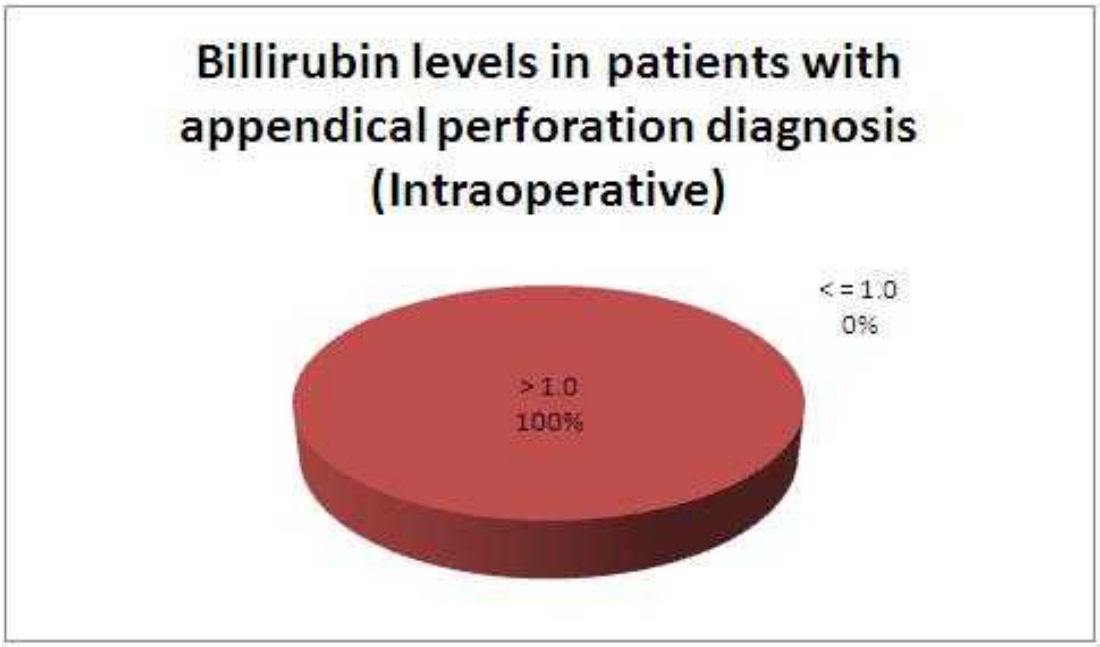


Table 8 Total Leukocyte count (TLC)

TLC Count	Number	Percentage
< = 11000	177	71%
> 11000	73	29%
Total	250	100%

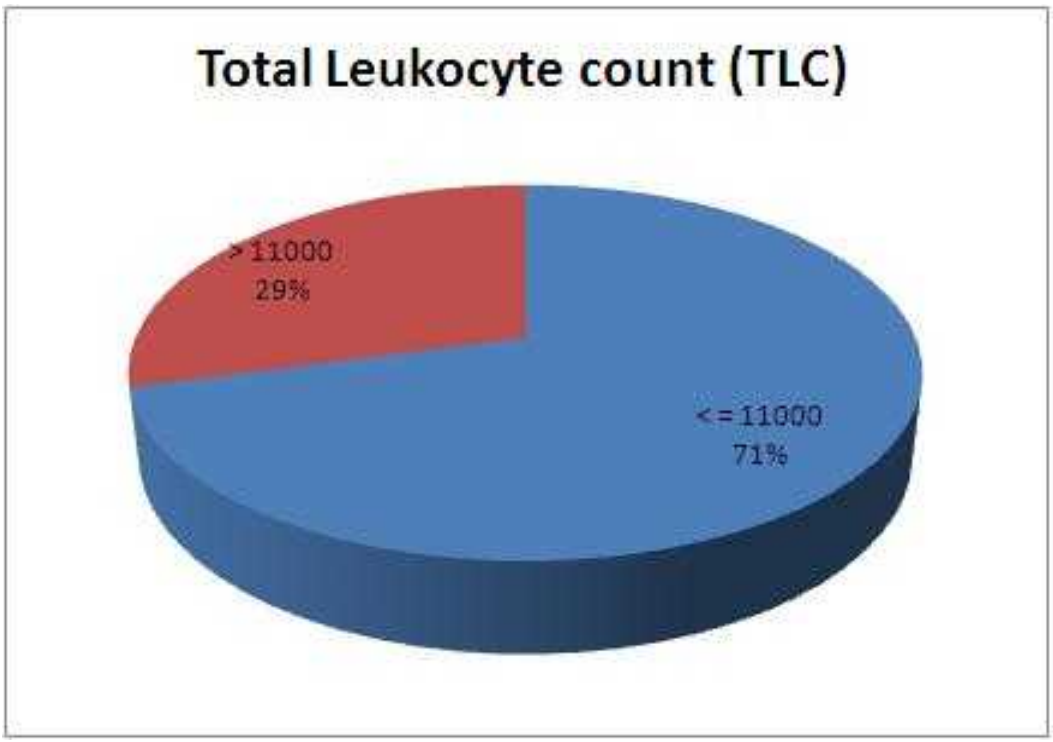


Table 9 Differential Leukocyte Count (DLC)

DLC	Mean	SD
Total Count	10036.84	2343.95
Neutrophils	82.65	3.11
Lymphocytes	10.92	2.49
Monocytes	3.74	0.92
Eosinophils	2.73	0.74

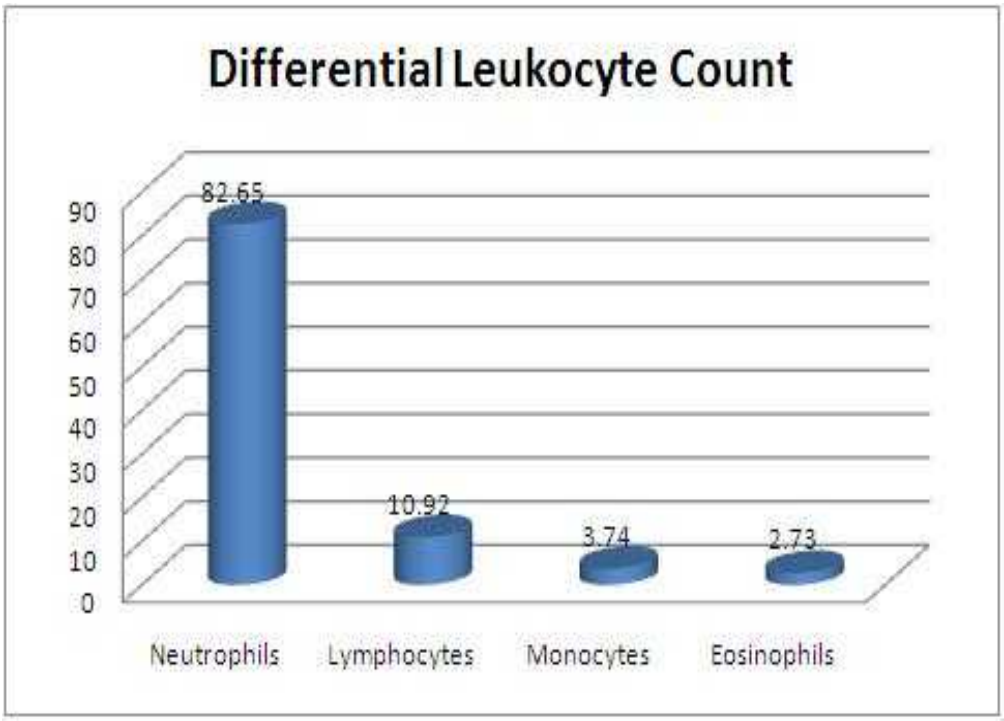


Table 10 Pre-operative Diagnosis (Clinical)

Pre-operative Diagnosis	Mean	Percentage
Acute Appendicitis	233	93.2%
Appendicular Perforation	17	6.8%
Total	250	100%

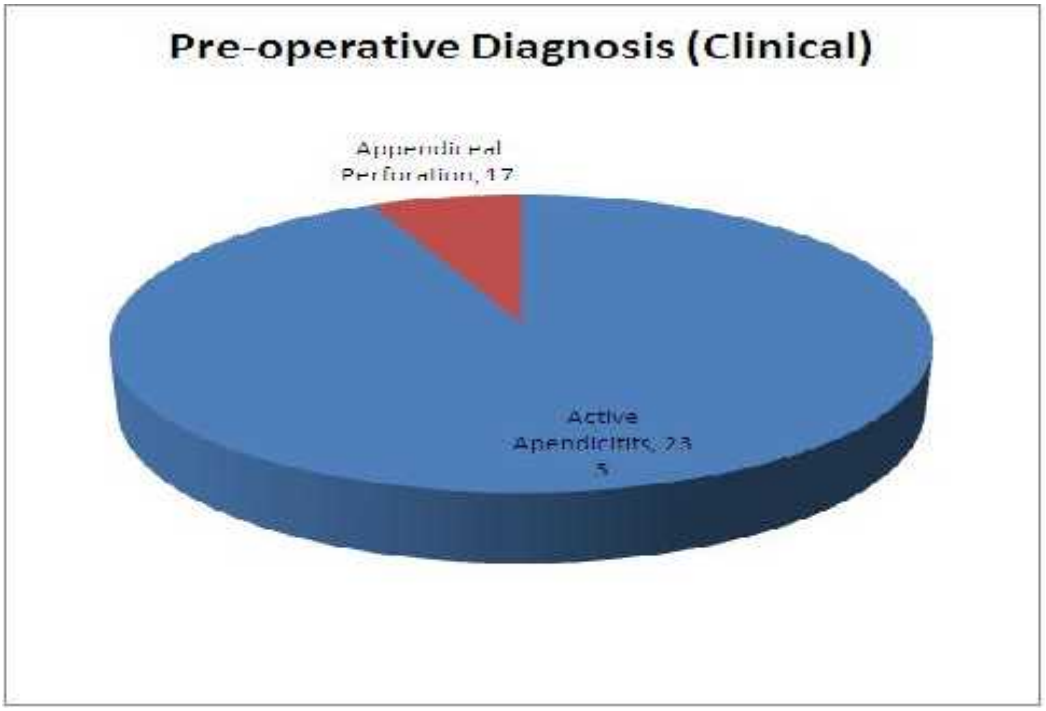


Table 11 Ultrasonographic Findings

Findings	Number	Percentage
Normal/Q	77	30.8%
Acute Appendicitis	173	69.2%
Appendicular Perforation	00	0%
Total	250	100%

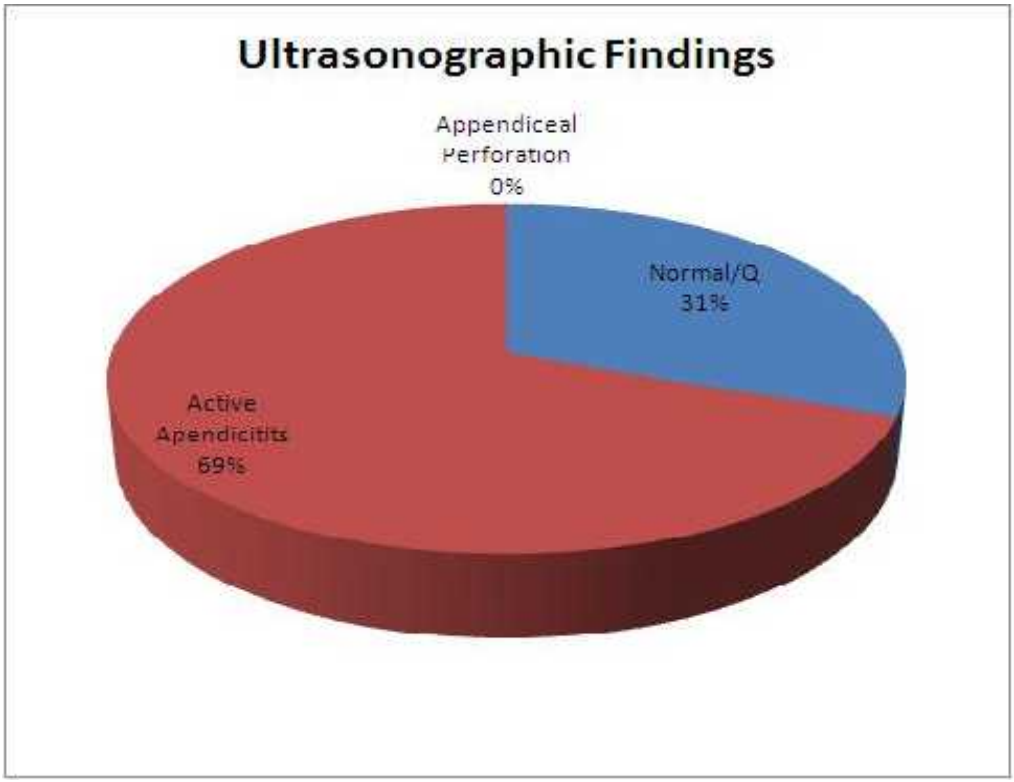


Table 12 Post-operative diagnosis (Intraoperative)

Diagnosis	Number	Percentage
Acute Appendicitis	230	92%
Appendicular Perforation	20	8%
Total	250	100%

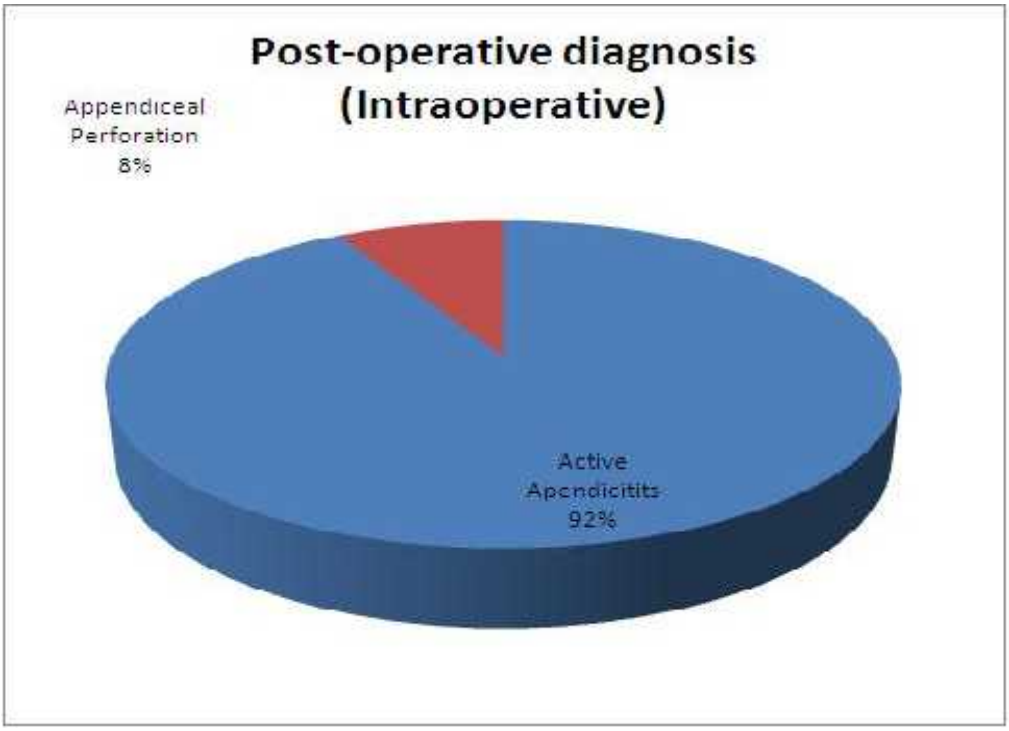


Table 13 Comparison of mean serum bilirubin levels in patients with acute appendicitis and appendicular perforation (Intraoperative)

Bilirubin Levels	Acute appendicitis (N=230)		Appendicular perforation (N=20)		Z-value	P-value	Conclusion
	Mean	SD	Mean	SD			
Total Bilirubin	1.02	0.33	2.16	0.32	15.6	< 0.0001	Significant
Conjugate	0.66	0.26	1.27	0.31	9.38	< 0.0001	Significant
Unconjugate	0.36	0.12	0.87	0.22	11.33	< 0.0001	Significant

Table 14 Comparison of mean serum bilirubin levels in patients with acute appendicitis and appendical perforation (Clinical)

Bilirubin Levels	Acute appendicitis (N=233)		Appendicular perforation (N=17)		Z-value	P-value	Conclusion
	Mean	SD	Mean	SD			
Total Bilirubin	1.03	0.34	2.22	0.30	16.1	< 0.0001	Significant
Conjugate	0.66	0.26	1.31	0.31	10.02	< 0.0001	Significant
Unconjugate	0.36	0.13	0.85	0.24	11.24	< 0.0001	Significant

Table 15 Correlation of acute appendicitis and appendicular perforation with total serum bilirubin levels(Clinical)

Serum Bilirubin Levels	Acute appendicitis	appendicularperforation	Chi Square	P-value	Conclusion
	Number	Number			
< = 1.0	125	0	14.20	0.001	Significant i.e. there is high correlation/association between serum bilirubin levels and status of appendicitis
> 1.0	108	17			
Total	233	17			

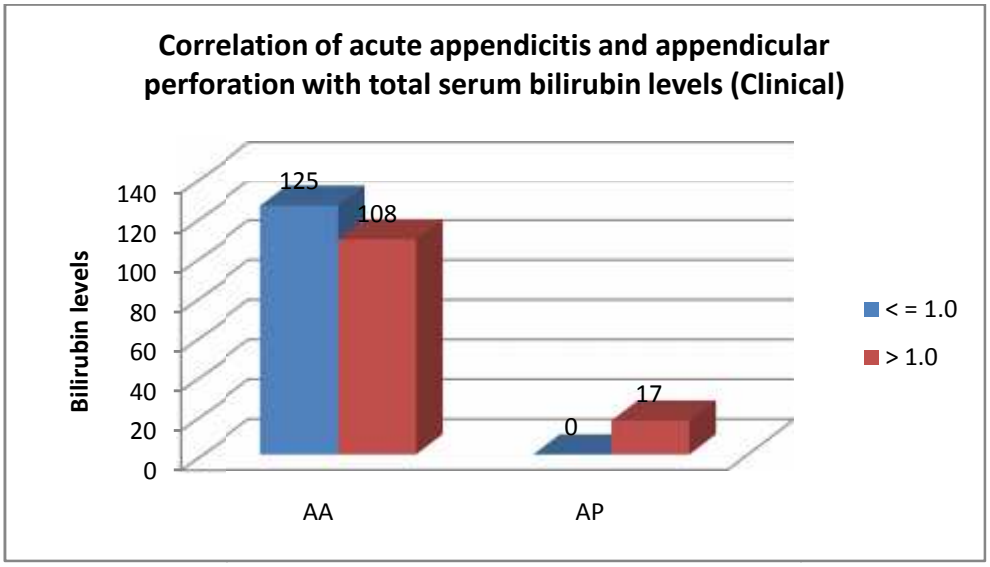


Table 16 Correlation of acute appendicitis and appendicular perforation with total serum bilirubin levels (Intraoperative)(HPR)

Serum Bilirubin Levels	Acute appendicitis Number	Appendicular perforation Number	Chi Square	P-value	Conclusion
< = 1.0	125	0	19.6	0.001	Significant i.e. there is high correlation/association between serum bilirubin levels and status of appendicitis
> 1.0	105	20			
Total	230	20			

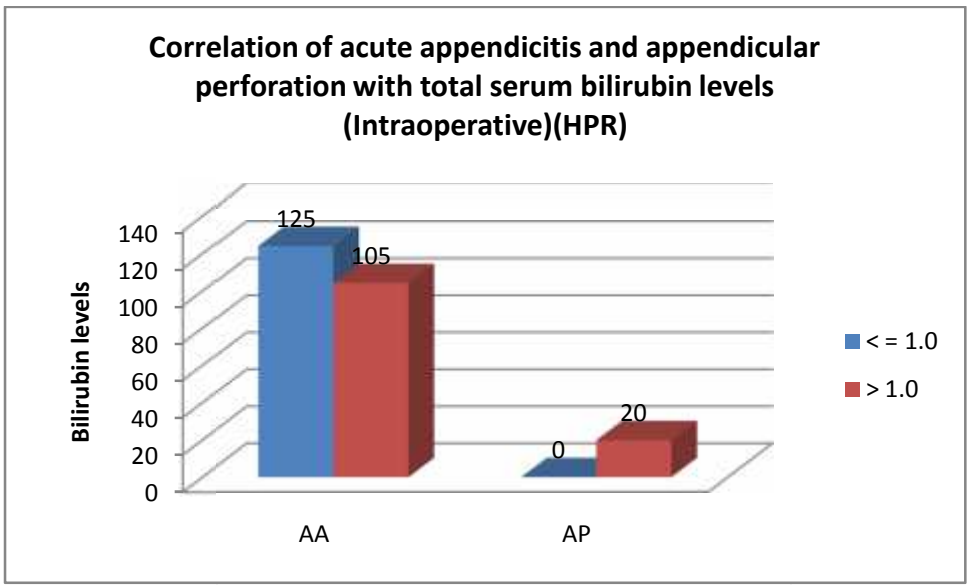
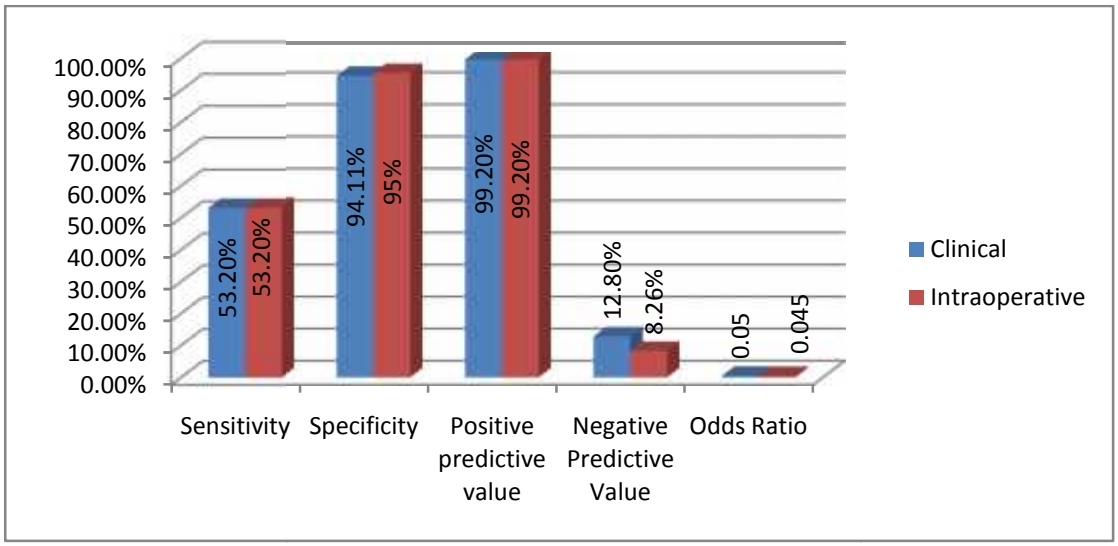


Table 17.Sensitivity, specificity, positive predictive value, negative predictive value, odds ratio.

Measures	Clinical	Intraoperative
Sensitivity	53.2%	53.2%
Specificity	94.11%	95%
Positive predictive value	99.2%`	99.2%
Negative Predictive Value	12.8%	8.26.%
Odds Ratio	0.05	0.045



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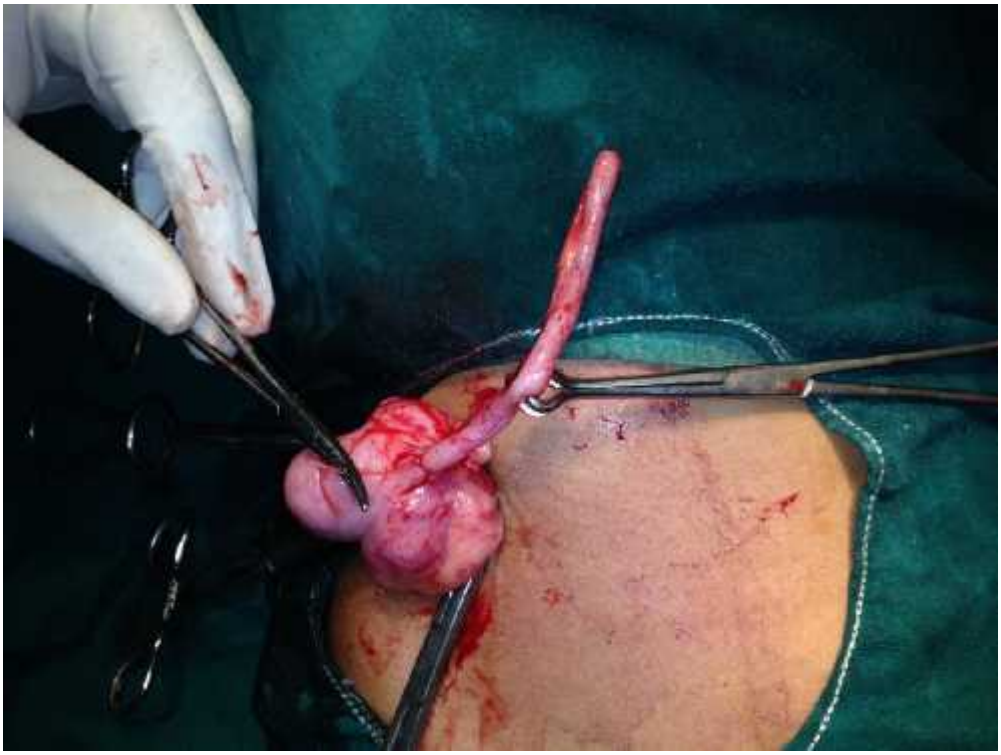
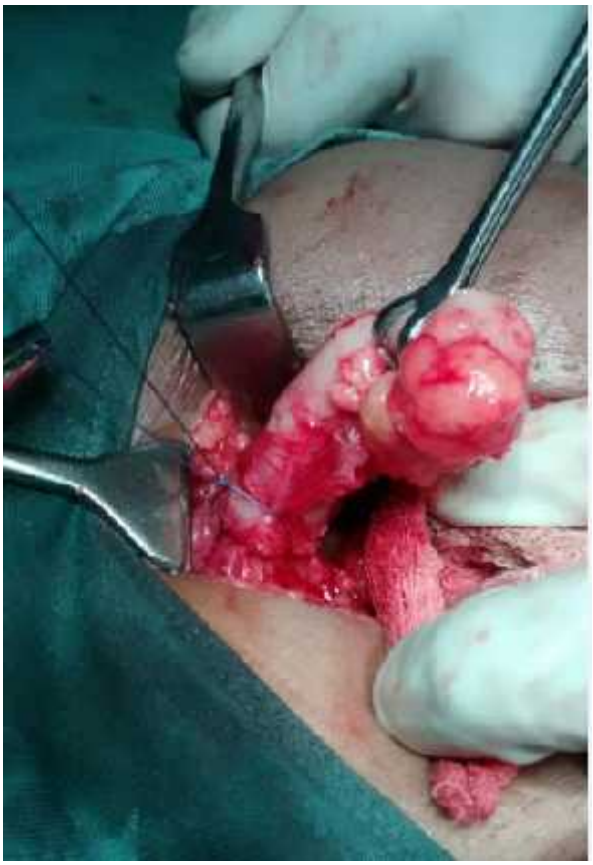
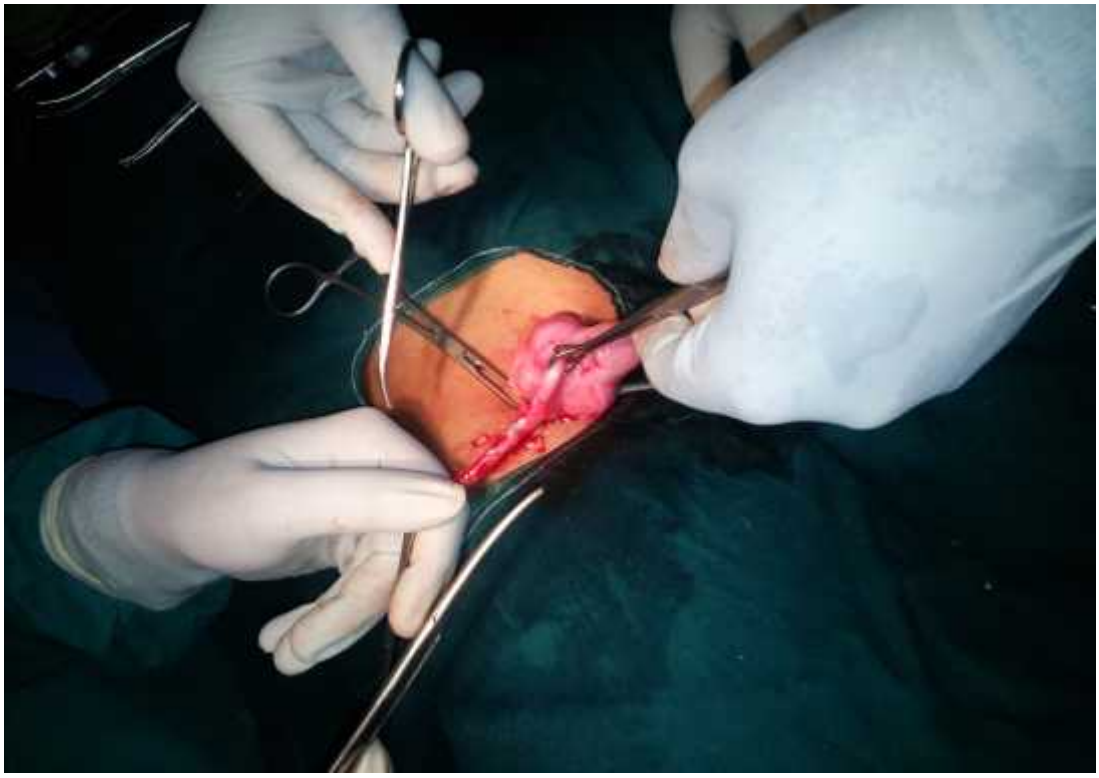


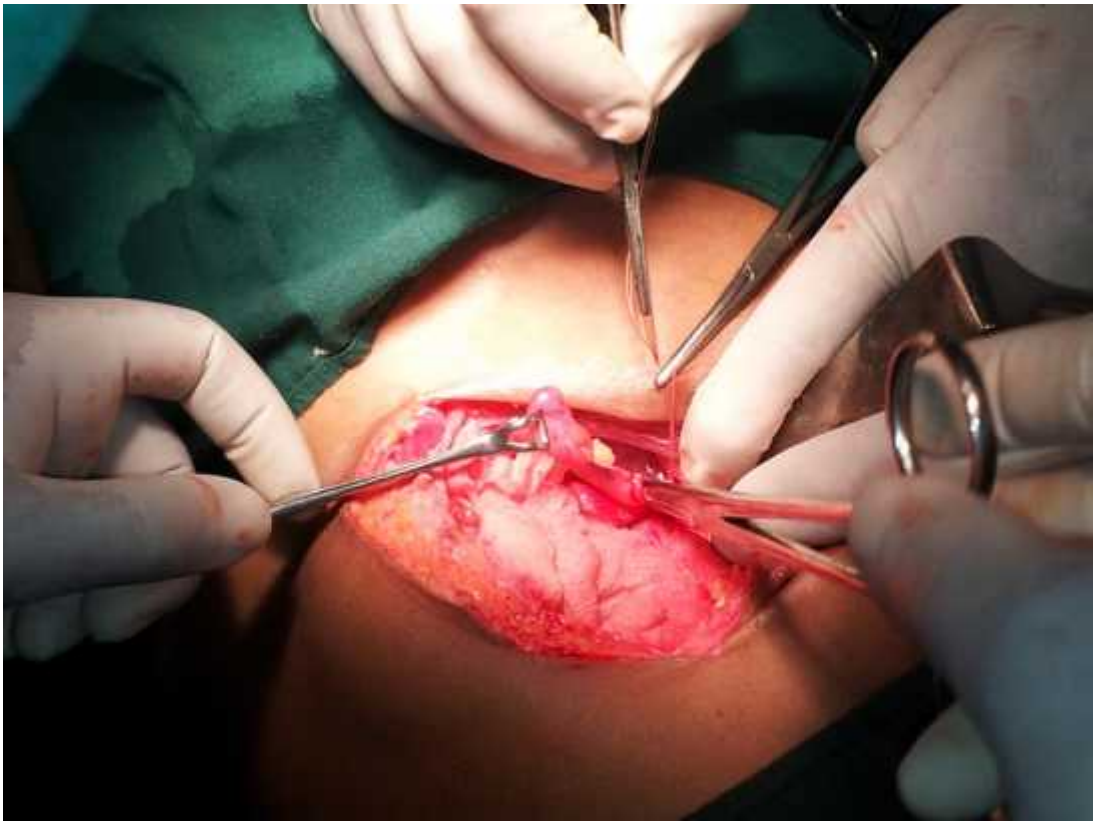
Photograph.1. Acute appendicitis



Photograph 2.Appendiceal perforation



Photograph.3. Acute appendicitis



Photograph 4.Appendiceal perforation

DISCUSSION

Acute appendicitis is the most common cause of 'acute abdomen' in young adults. Appendicectomy is the most frequently performed urgent abdominal operation and is often the first major procedure performed by a surgeon in training. About 8% of people in Western countries have appendicitis at some time in their lifetime.[55]

The peak incidence of acute appendicitis is in the second and third decade of life. It is relatively rare in infants, and becomes increasingly common in childhood and early adult life. The incidence of appendicitis is equal in males and females before puberty. In teenagers and young adults, the male – female ratio increases to 3:2 at age. The lifetime rate of appendicectomy is 12% for men and 25% for women, with approximately 7% of all people undergoing appendectomy for acute appendicitis during their lifetime.[56,57]Obstruction of the lumen is believed to the major cause of acute appendicitis.Faecoliths are the usual cause of obstruction. Less- common causes are hypertrophy of lymphoid tissue, tumors, intestinal parasites.[58]Thebacteriology of normal appendix is similar to that of normal colon. The principal organism seen in normal appendix, in acute appendicitis, and in perforated appendicitis are *Escherichia Coli* and *Bacteroides fragilis*. However a wide varietyof both the diagnosis of acute appendicitis is essentially clinical; however, a decision to operate based on clinical suspicion alone can lead to the removal of normal appendix in 15 to 30% of cases. The premise that it is better to remove a normal appendix than to delay diagnosis does not stand up to close scrutiny, particularly in the elderly. Hence, the diagnosis of Appendicitis still remains a dilemma in spite of the advances in various laboratory and radiological investigations.A new tool to help in the diagnosis of acute appendicitis would thus be welcome.

Serum Bilirubin level elevation will help in the accuracy of clinical diagnosis of acute appendicitis and more importantly help in foreseeing and preventing impeding complications of acute appendicitis.

This study was taken up with this thought – that is it possible to add serum bilirubin as a new laboratory marker to aid in the diagnosis of acute appendicitis and if so, does it have the credibility to help us foresee an impending complication of acute appendicitis? Importance of hyperbilirubinemia and its association in acute appendicitis has being postulated recently. There are only a few case reports in the available literature that describe the finding of hyperbilirubinemia in patients of acuteappendicitis.[59]It is hypothesized that an association exists between hyperbilirubinemia and acute appenditics and its complications.[59]The present study was undertaken to study the relationship between hyperbilirubinemia and acute appendicitis and to evaluate its credibility as a diagnostic marker for acute appendicitis and also, to evaluate whether elevated bilirubin levels have a predictive potential for the diagnosis of appendiceal perforation.

This study was conducted in the Department of General Surgery, BLDE UNIVERSITY'S SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, BIJAPUR over aperiod from October 2012 to May2014 on 250 patients with clinical diagnosis of Acute appendicitis and Appendiceal perforation.

In the present study of the 250 patients enrolled for the study, 149 patients (60%) were males while the remaining 101 patients (40%) were females.The mean age in our study population (250 patients) was 31.45 ± 13.85 years. This is consistent with the quoted incidence of Appendicitis in the literature where it is most frequently seen in patients in their second through fourth decades of life.The average age group

in females31.24±13.62 years was slightly higher than males 31.59±14.05years. Hyperbilirubinemia (> 1.0 mg/dL) in our study was found in 125 patients(50%) of all the 250 patients (n=250) enrolled in the study, while 125patients(50%) had normal bilirubin levels (≤ 1.0 mg/dL). Estrada et al had found hyperbilirubinemia in 59 (38%) of 157 patients studied with acute appendicitis.

The mean total serum bilirubin of all 250 patients was 1.11 ± 0.45 mg/dL(range, 0.66 – 1.56 mg/dL), which was above the normal range (≤ 1.0 mg/dL) considered for the study, hence indicating the occurrence of hyperbilirubinemia. The mean of Direct bilirubin was 0.65±0.23 mg/dL (range, 0.42-0.88 mg/dL) while that of Indirect bilirubin was 0.34±0.12 mg/dL (range, 0.22 – 0.46 mg/dL). Our finding was consistent with hyperbilirubinemia found in a study conducted by Khan S,15who found average level of serum bilirubin in his study population to be 2.38 mg/dL. All patients were found to have SGOT and SGPT within the normal range, thus excluding any associated liver pathology (Exclusion criteria). The mean SGOT and SGPT were 25.36± 7.03 U/L (range, 18.33-32.39U/L) and 20.52± 4.85U/L (range, 15.67-25.37 U/L). The mean ALP values were 36.94 ± 10.96 U/L (range, 25.98 - 47.9U/L).

In our study population of 250 patients, 233 patients (93.2.%) were diagnosed as acute appendicitis pre-operatively while 17 patients (6.8%) were diagnosed with Appendiceal perforation. The diagnosis was confirmed post-operatively by histopathological reports (HPR) and those differing from the pre-operative diagnosis were excluded from the study.

Amongst the patients diagnosed with Acute appendicitis pre-operatively (n=233), 108 patients (46.4%) were found to have elevated bilirubin (>1.0 mg/dL) while only 125 patients (53.6%) had normal bilirubin levels (≤1.0 mg/dL). In patients diagnosed with Appendicular perforation (n=17), 17 patients (100%) had bilirubin

elevated (>1.0 mg/dL). Thus, Hyperbilirubinemia was found in most of the patients diagnosed with acute appendicitis (46.4%) or appendiceal perforation (100%).

The total leukocyte count was found elevated in just 177 patients (71%) of the total 250 patients. The mean of TLC count in all patients was 10036.84±2343.95/mm3 (range, 7692- 12380.79/mm3), in which the highest percentage constituted Neutrophils with 82.65% followed by 10.92% by Lymphocytes.

On Ultrasonography, 173patients (69.2%) were diagnosed as acute appendicits while 77 patients (30.8%) were reported as normal ultrasonographic findings. None however were diagnosed as Appendiceal perforation on ultrasonography. Ultrasonography per-se was not helpful as a useful investigation for appendicitis or appendiceal perforation in our study as none of the USG findings reported Appendiceal perforation, hence belief that the diagnosis of appendicitis still remains essentially clinical, still hold true. The mean bilirubin levels in patients diagnosed with acute appendicitis was 1.02 ± 0.33 mg/dL (range, 0.69– 1.35 mg/dL) while in patients diagnosed with Appendicular perforation was 2.16±0.32 mg/dL (range, 1.84–2.48 mg/dL). Hence, we see that patients with appendicular perforation had nearly two times more levels of bilirubin as compared to that of acute appendicitis. So we infer that, patients with features suggestive of appendicitis with two times their normal range of bilirubin, are more susceptible of having appendicular perforation than those with normal or slightly elevated total serum bilirubin. Sand et al in his study found the mean bilirubin levels in patients with appendicular perforation to be significantly higher than those with a non- perforatedappendicitis.

The Direct bilirubin and indirect bilirubin in patients diagnosed with acute appendicitis was 0.66±0.26mg/dL and 0.36±0.12 mg/dL respectively.Similarly, direct

bilirubin and indirect bilirubin in patients diagnosed with Appendicular perforation was 1.27±0.31mg/dL and 0.87±0.22 mg/dLrespectively. And in our study the conjugated bilirubin is raised in 125(100%) patients among 125 patients of raised total bilirubin and unconjugated bilirubin is raised in 35(28%) among 125 patients of raised total bilirubin.

The Sensitivity, Specificity, Positive predictive value, Negative predictive value and Odds ratio was calculated from a 2x2 table.Sensitivity and Specificity of bilirubin in predicting acute appendicitis and appendicular perforation diagnosis was 53.2% and95%respectively. Similarly Positive predictive value and Negative predicative value of bilirubin in predicting acute appendicitis and appendicular perforation diagnosis was 99.2% and 8.26% respectively.

The Odds ratio was calculated to be 0.045%. The sensitivity in our study was more than that by Sand et al in which, he found the sensitivity and specificity in his study of hyperbilirubinemia for predicting appendiceal perforation to be 70% and 86.0% respectively.

CONCLUSION

Finding of the present study suggest;

- Serum bilirubin levels appears to be a promising new laboratory marker for diagnosing acute appendicitis, however diagnosis of appendicitis remains essentially still - clinical. Its levels come out to be a credible *aid* in diagnosis of acute appendicitis and would be helpful investigation in decision making.
- Patients with clinical signs and symptoms of appendicitis and with hyperbilirubinemia three times the normal range should be identified as having a higher probability of appendicular perforation suggesting, serum bilirubin levels have a predictive potential for the diagnosis of appendicular perforation.

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ANNEXURE-I

B.L.D.E. UNIVERSITY

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act,1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

IEC Ref No, 39|2013

January 23rd, 2013

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on 23rd January 2013 *at* 11.A.M *to scrutinize the Synopsis / Research projects of Postgraduate student / Undergraduate student / Faculty members of this University / college from ethical clearance point of view. After scrutiny the following original / corrected & revised version synopsis of the Thesis / Research project has been accorded Ethical Clearance.*

Title **"Hyperbilirubinemia- A New Diagnostic Marker in Acute Appendicitis and Appendicular Peforation."**

Name of PA.D./ P. G. / U. G. Student / Faculty member **Dr. Ravi A. Ichalakaranji**

Name of Guide **Dr. Vijaya Patil**

Dr. Sharada Metgud
Chairperson, I.E.C
BLDE University,
BIJAPUR – 586 103

Mr.G.V.Kulkarni
Secretary, I.E.C
BLDE University,
BIJAPUR – 586 103.

Following documents were placed before Ethical Committee for Scrutinization:

- Copy of Synopsis / Research project
- Copy of informed consent form
- Any other relevant document's

Smt. Bangaramma Sajjan Campus, Sholapur Road, Bijapur – 586103, Karnataka, India.
University: Phone: +918352-262770, Fax: +918352-263303, Website: www.bldeuniversity.org, E-mail: office@bldeuniversity.org
College: Phone: +918352-262770, Fax: +918352-263019, Website: www.bldea.org, E-mail: bmpmc1@yahoo.co.in

ANNEXURE-II

CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR – 586103, KARNATAKA

TITLE OF THE PROJECT:	**HYPERBILIRUBINEMIA- A NEW DIAGNOSTIC MARKER IN ACUTE APPENDICITIS AND APPENDICULAR PERFORATION.**
PRINCIPAL INVESTIGATOR:	**Dr. RAVI A. ICHALAKARANJI** (PG) Department of General Surgery Email: ravi.ichalkaranji@gmail.com
PG GUIDE:	**Dr.VIJAYA PATIL**$_{M.S.}$ Professor of Surgery B.L.D.E. University's Shri B.M. Patil Medical College & Research Centre, Sholapur Road, BIJAPUR - 586103

PURPOSE OF RESEARCH:

I have been informed that this study will analyse the effectiveness of hyperbiluribinemia in the evaluation of acute appendicitis and its role in prediction of appendicular perforation.

I have been explained about the reason for doing this study and selecting me/my ward as a subject for this study. I have also been given free choice for either being included or not in the study.

PROCEDURE:

I have been explained that depending upon the group allocated to me/my ward,I'll/my ward will be subjected to certain blood investigations like bilirubin levels, total leucocyte count and urine investigations, and USG.

RISKS AND DISCOMFORTS:

I understand that I/my ward may experience some complications during drawing blood for investigations like injection site infection, bleeding etc, and I understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

I understand that my/my wards participation in this study will help to analyse the effectiveness of hyperbilirubinemia in diagnosis of acute appendicitis and its role in early prediction of appendicular perforation.

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may askmore questions about the study at any time. Dr. Ravi Ichalakaranji will be availableto answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me.

And that a copy of this consent form will be given to me for keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that Dr. Ravi Ichalakaranji will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: Dr.VijayaPatil (Guide) Dr. Ravi I (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Ravi Ichalakaranjihas explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

ANNEXURE – III

PROFORMA OF CASE TAKING

1) Name: CASE NO:

2) Age: IP NO:

3) Sex: DOA:

4) Religion: DOS:

5) Occupation: DOD:

6) Residence:

7) CHIEF COMPLAINTS:

8) HISTORY OF PRESENTING ILLNESS:

9) PAST HISTORY:

- Diabetes mellitus
- Hypertension
- History of any drug intake
- Renal disease
- Jaundice

10) FAMILY HISTORY:

11) GENERAL PHYSICAL EXAMINATION:

Pallor: present/absent

Icterus: present/absent

Clubbing:	present/absent
Generalized Lymphadenopathy:	present/absent
Build:	Poor/Middle /Well
Nourishment:	Poor / Middle / Well

12) VITALS

PR:

BP:

RR:

Temp:

Weight:

13) OTHER SYSTEMIC EXAMINATION:

- Per Abdomen examination
- Respiratory System
- Cardiovascular System
- Central Nervous System

14) INVESTIGATION:

BLOOD: Hb :

URINE: Albumin:

TC : Sugar:

DC: Microscopy:

ESR: BT, CT:

SERUM BILIRUBIN(Total):

CONJUGATED :

UNCONJUGATED:

C-REACTIVE PROTEIN:

BLOOD UREA:

SERUM CREATININE:

RBS:

SGOT: SGPT: ALP:

USG ABDOMEN:

16) FINAL DIAGNOSIS:

SL.NO	NAME	AGE	SEX	IP NO	TOTAL BILIRUBIN	CONJ BILIRUBIN	UNCONJ BILIRUBIN	CRP	TOTAL COUNT	N	E	M	B	L	SGOT	SGPT	ALP	CLINICAL D	ULTRASOUND	INTRAOPERATIVE D
1	VINOD	23	M	12473	3.2	2	1.2	POSITIVE	13450	92	2	3	0	3	32	31	53	AP	AA	AP
2	SUKANYA	22	F	16569	2.6	1.6	0.6	POSITIVE	14520	92	2	2	0	4	20	32	32	AP	Q	AP
3	MALLIKARJUN	35	M	2741	2.4	1.8	0.6	POSITIVE	13000	91	2	2	0	5	35	13	76	AP	Q	AP
4	SHANKAR	42	M	15826	2.4	1.8	0.6	POSITIVE	14320	91	2	2	0	5	20	9	34	AP	AA	AP
5	NAGANAGOUDA	16	M	2856	2.2	1	1.2	POSITIVE	12370	90	3	2	0	5	35	18	40	AP	Q	AP
6	VINOD	17	M	2869	2.2	1.1	1.1	POSITIVE	13400	91	2	3	0	5	30	21	41	AP	AA	AP
7	KANKABAI	32	F	9427	2.2	1.2	1	POSITIVE	15670	90	2	3	0	5	36	26	30	AP	AA	AP
8	RENUKA	17	F	16825	2.2	1.2	1	POSITIVE	14000	91	2	3	0	5	34	16	33	AP	AA	AP
9	MANOJ	38	M	26828	2.2	1	1.2	POSITIVE	14500	91	2	3	0	4	29	21	35	AP	Q	AP
10	SHARADA	45	F	8493	2.1	0.9	1.2	POSITIVE	13560	91	2	3	0	5	22	13	33	AP	AA	AP
11	REKHA	28	F	9444	2.1	1.4	0.7	POSITIVE	17640	91	2	3	0	5	39	19	38	AP	AA	AP
12	SIDDU	28	M	12117	2.1	1.2	0.9	POSITIVE	14560	90	3	2	0	6	34	22	31	AP	Q	AP
13	KITAPPA	56	M	5575	2	1.2	0.8	POSITIVE	13240	90	2	3	0	5	20	24	34	AP	Q	AP
14	NAGESH	20	M	5913	2	1.2	0.8	POSITIVE	13250	90	2	2	0	6	28	22	41	AP	AA	AP
15	RAJESH	18	M	8989	2	1.1	0.9	POSITIVE	13800	91	2	2	0	5	33	18	36	AP	Q	AP
16	SHANKARAPPA	29	M	12075	2	1.2	0.8	POSITIVE	14560	90	2	2	0	6	36	25	48	AP	Q	AP
17	CHANDRAKANT	29	M	13347	2	1.5	0.5	POSITIVE	11230	91	2	3	0	4	30	28	33	AP	Q	AP
18	SUNIL	16	M	7394	1.8	1	0.8	POSITIVE	11250	83	4	3	0	10	14	23	63	AA	Q	AP
19	SANPANNA	35	M	8179	1.8	0.9	0.9	POSITIVE	13450	88	2	3	0	7	29	15	31	AA	AA	AP

20	LAKAPPA	50	M	8235	1.8	1.1	0.7	POSITIVE	13000	86	3	2	0	9	24	18	33	AA	Q	AP
21	DEVARAJ	46	M	8851	1.8	1.4	0.4	POSITIVE	12730	85	3	4	0	9	27	20	22	AA	AA	AA
22	RAKESH	32	M	11173	1.8	1.4	0.4	POSITIVE	12240	88	2	4	0	6	25	11	42	AA	AA	AA
23	MAHESH	31	M	11985	1.8	1	0.8	POSITIVE	13450	88	2	3	0	7	23	15	42	AA	AA	AA
24	MADHURI	23	F	13194	1.8	1.2	0.6	POSITIVE	12670	86	3	2	0	9	28	12	43	AA	AA	AA
25	RUKMINI	55	F	14676	1.8	1.1	0.7	POSITIVE	11000	88	2	3	0	7	30	16	67	AA	AA	AA
26	SANGAPPA	58	M	14936	1.8	1.2	0.6	POSITIVE	14560	87	2	3	0	8	32	22	51	AA	AA	AA
27	TANVEER	48	M	15189	1.8	1.2	0.6	POSITIVE	13440	88	2	3	0	7	24	28	57	AA	AA	AA
28	MAHESH	36	M	15865	1.8	1.6	0.2	POSITIVE	13420	86	2	4	0	8	26	19	44	AA	AA	AA
29	PRABHAVATI	38	F	9267	1.7	1.1	0.6	POSITIVE	12450	85	2	3	0	11	24	19	60	AA	AA	AA
30	BAGAPPA	45	M	4972	1.6	1.1	0.5	POSITIVE	15680	84	4	4	0	8	39	34	45	AA	AA	AA
31	NAGAMMA	48	F	5195	1.6	1	0.6	POSITIVE	12450	83	3	3	0	11	22	13	54	AA	AA	AA
32	RAJU	25	M	6835	1.6	1	0.6	POSITIVE	12000	84	2	4	0	10	28	18	59	AA	AA	AA
33	KALMESH	27	M	10964	1.6	1.1	0.5	POSITIVE	15670	82	3	4	0	11	30	24	35	AA	AA	AA
34	GURUBAI	52	F	11168	1.6	1.2	0.4	POSITIVE	12340	84	2	5	0	9	23	20	40	AA	AA	AA
35	DEEPA	28	F	13924	1.6	1	0.6	POSITIVE	10000	84	2	3	0	11	32	25	45	AA	AA	AA
36	SANGAMESH	18	M	535	1.5	1	0.5	POSITIVE	11450	85	2	4	0	9	33	15	37	AA	AA	AA
37	HARISH	42	M	4304	1.5	1	0.5	POSITIVE	14563	84	2	4	0	10	35	32	30	AA	AA	AA
38	GANGABAI	57	F	4841	1.5	1.1	0.4	POSITIVE	12564	85	3	3	0	9	21	24	40	AA	AA	AA
39	KENCHAPPA	21	M	5085	1.5	1.3	0.2	POSITIVE	13240	83	2	3	0	12	20	18	44	AA	AA	AA
40	PRASANNA	65	M	8395	1.5	1	0.5	POSITIVE	12130	83	2	3	0	12	19	15	38	AA	AA	AA
41	DARIYAPA	60	M	11744	1.5	1.1	0.4	POSITIVE	15670	84	3	3	0	10	32	28	32	AA	AA	AA
42	REVATHI	46	F	16374	1.5	0.9	0.6	POSITIVE	13450	84	3	4	0	9	43	7	46	AA	AA	AA
43	LAXMI	22	F	17871	1.5	1	0.5	POSITIVE	14560	84	3	3	0	10	32	18	70	AA	AA	AA
44	SHANTAPPA	50	M	21966	1.5	1	0.5	POSITIVE	11400	85	2	4	0	9	35	22	32	AA	AA	AA
45	GANGA	35	F	2905	1.4	0.9	0.5	POSITIVE	9860	81	2	5	0	12	34	22	40	AA	AA	AA

46	SUNANDA	25	F	3890	1.4	0.9	0.5	POSITIVE	9980	84	3	3	0	10	28	24	44	AA	AA	AA
47	GANGABAI	65	F	4341	1.4	1	0.4	POSITIVE	8970	82	3	5	0	10	32	28	41	AA	AA	AA
48	SOUBHAGYA	13	F	4497	1.4	0.9	0.5	POSITIVE	8230	85	2	3	0	10	20	23	49	AA	AA	AA
49	RAJENDRA	46	M	4865	1.4	0.9	0.5	POSITIVE	8430	85	2	3	0	10	20	24	35	AA	AA	AA
50	VIDYA	40	F	6624	1.4	0.8	0.6	POSITIVE	9832	84	2	4	0	10	25	28	38	AA	AA	AA
51	RAMESH	35	M	7386	1.4	0.9	0.5	POSITIVE	9930	86	2	4	0	8	34	22	34	AA	AA	AA
52	MALLAPPA	50	M	8075	1.4	0.8	0.5	POSITIVE	9430	83	2	3	0	12	22	12	68	AA	AA	AA
53	KALAVATHI	45	F	8265	1.4	1	0.4	POSITIVE	9902	84	3	3	0	10	32	22	44	AA	AA	AA
54	NEELAMMA	25	F	10277	1.4	0.9	0.5	POSITIVE	7820	83	2	3	0	12	29	24	38	AA	AA	AA
55	PRIYANKA	14	F	10558	1.4	1	0.4	POSITIVE	7780	84	2	4	0	10	25	16	31	AA	AA	AA
56	PRAKASH	24	M	10646	1.4	1	0.4	POSITIVE	11000	84	2	3	0	11	40	19	61	AA	AA	AA
57	RAJANI	36	F	10883	1.4	1.1	0.3	POSITIVE	11260	83	3	3	0	11	27	8	54	AA	AA	AA
58	MALLIKARJUN	29	M	11729	1.4	1	0.4	POSITIVE	12340	83	3	4	0	10	34	16	50	AA	AA	AA
59	CHIDANAND	41	M	13706	1.4	1	0.4	POSITIVE	12320	84	2	5	0	9	16	20	42	AA	AA	AA
60	SUNDAR	30	M	15869	1.4	1	0.4	POSITIVE	14500	85	2	3	0	10	24	21	34	AA	AA	AA
61	RUKMABAI	40	F	16222	1.4	0.8	0.6	POSITIVE	12670	83	2	4	0	11	34	9	31	AA	AA	AA
62	AKASH	13	M	22830	1.4	1	0.4	POSITIVE	10400	82	2	1	2	13	20	22	32	AA	AA	AA
63	YELAPPA	59	M	3999	1.3	0.9	0.4	POSITIVE	8970	85	2	3	0	10	18	21	39	AA	AA	AA
64	BASAVARAJ	29	M	4711	1.3	0.8	0.5	POSITIVE	7910	82	2	4	0	12	15	20	45	AA	AA	AA
65	MUKTABAI	50	F	5041	1.3	0.8	0.5	NEGATIVE	8970	84	2	5	0	9	22	12	55	AA	AA	AA
66	LAKKAPPA	65	M	5225	1.3	0.9	0.4	NEGATIVE	8610	83	2	5	0	10	24	21	40	AA	AA	AA
67	RAJKUMAR	40	M	5440	1.3	0.7	0.6	POSITIVE	9870	85	2	4	0	9	23	19	44	AA	Q	AA
68	RAKESH	37	M	5863	1.3	1.1	0.2	POSITIVE	9810	83	2	4	0	11	20	10	38	AA	Q	AA
69	GANGAMMA	45	F	6576	1.3	0.7	0.6	NEGATIVE	9970	82	3	4	0	11	31	22	30	AA	AA	AA
70	SAVITHA	20	F	8202	1.3	0.9	0.4	NEGATIVE	8960	84	3	3	0	10	19	8	67	AA	AA	AA
71	MAHESH	25	M	8428	1.3	0.8	0.5	POSITIVE	8890	84	3	3	0	10	28	20	65	AA	AA	AA

72	YAMNUR	56	M	11967	1.3	1	0.3	POSITIVE	8070	83	2	4	0	11	25	24	47	AA	AA	AA
73	SUSHMA	14	F	13402	1.3	0.8	0.5	POSITIVE	9610	84	2	4	0	10	25	19	32	AA	AA	AA
74	DEVANAND	34	M	14096	1.3	1	0.3	POSITIVE	8950	82	2	4	0	12	27	22	30	AA	AA	AA
75	GEETHA	46	F	14126	1.3	0.8	0.5	POSITIVE	9860	84	3	3	0	10	32	20	28	AA	Q	AA
76	BEERESH	35	M	14583	1.3	0.8	0.4	POSITIVE	9960	85	2	5	0	7	33	35	40	AA	AA	AA
77	SANJEEV	28	M	15022	1.3	0.7	0.6	POSITIVE	8950	82	3	4	0	11	20	34	32	AA	AA	AA
78	NAZIRSAAB	25	M	15938	1.3	0.8	0.5	NEGATIVE	9060	84	2	3	0	11	15	16	48	AA	AA	AA
79	SAVITA	25	F	22781	1.3	0.9	0.4	POSITIVE	9800	78	2	3	1	15	25	17	30	AA	AA	AA
80	DEVANNA	60	M	3588	1.2	0.8	0.4	POSITIVE	9450	83	3	5	0	9	23	18	32	AA	AA	AA
81	SUSHILA	34	F	4157	1.2	0.8	0.4	POSITIVE	9470	84	2	4	0	10	25	12	34	AA	AA	AA
82	RAMESH	24	M	6370	1.2	0.6	0.6	NEGATIVE	9960	83	2	4	0	11	12	23	58	AA	AA	AA
83	SHANTABAI	50	F	6914	1.2	0.8	0.4	POSITIVE	8960	83	2	3	0	12	15	14	34	AA	AA	AA
84	SANGEETHA	22	F	7214	1.2	1	0.2	POSITIVE	9970	91	3	3	0	13	28	21	53	AA	AA	AA
85	VINOD	14	M	10004	1.2	0.8	0.4	POSITIVE	9850	83	3	4	0	10	17	15	39	AA	Q	AA
86	ANANDKUMAR	21	M	10830	1.2	0.8	0.4	POSITIVE	6780	91	3	3	0	3	23	26	30	AA	AA	AA
87	BHARATHI	35	F	11194	1.2	0.9	0.3	POSITIVE	8850	84	3	2	0	11	12	15	46	AA	AA	AA
88	ANUSUYA	51	F	11994	1.2	1	0.2	POSITIVE	10600	82	3	3	0	12	20	20	44	AA	AA	AA
89	YEMANNAPA	58	M	12345	1.2	0.8	0.4	POSITIVE	8920	80	4	5	0	11	23	21	31	AA	AA	AA
90	SHABANA	13	F	12610	1.2	0.9	0.3	POSITIVE	9850	83	3	3	0	11	28	11	31	AA	Q	AA
91	LAXMI	35	F	12892	1.2	0.8	0.4	POSITIVE	9810	81	3	4	0	13	20	13	56	AA	Q	AA
92	MANJULA	24	F	12941	1.2	0.8	0.4	POSITIVE	8960	83	2	4	0	11	20	22	56	AA	AA	AA
93	RADHA	30	F	14018	1.2	1	0.2	POSITIVE	7810	82	4	4	0	10	20	17	53	AA	AA	AA
94	TULAJARAM	52	M	14423	1.2	0.8	0.4	POSITIVE	8900	84	3	3	0	10	20	23	63	AA	AA	AA
95	GANGABAI	59	F	15848	1.2	0.8	0.4	POSITIVE	9850	83	2	4	0	11	28	30	32	AA	AA	AA
96	ANAND	24	M	16439	1.2	0.9	0.3	POSITIVE	8970	88	4	2	1	5	34	20	34	AA	AA	AA
97	KITTAPA	50	M	16879	1.2	0.8	0.4	POSITIVE	7890	81	2	4	0	13	29	24	44	AA	AA	AA

98	SUNIL	18	M	18016	1.2	0.8	0.4	POSITIVE	9810	84	2	3	0	11	29	28	32	AA	Q	AA
99	RANI	38	F	24285	1.2	0.8	0.4	POSITIVE	9910	81	4	3	0	17	26	30	40	AA	Q	AA
100	JYOTHI	20	F	24319	1.2	0.8	0.4	POSITIVE	7850	82	3	4	0	13	25	20	35	AA	AA	AA
101	AISHA	23	F	2915	1.1	0.8	0.3	POSITIVE	9800	83	2	2	1	12	38	25	40	AA	Q	AA
102	SAVITA	30	F	3805	1.1	0.8	0.3	POSITIVE	9980	82	3	3	0	12	15	21	33	AA	AA	AA
103	RAJESH	42	M	4225	1.1	0.7	0.4	POSITIVE	10500	83	2	4	0	11	20	18	36	AA	AA	AA
104	LAXMAN	38	M	5224	1.1	0.8	0.3	NEGATIVE	7000	80	3	4	0	13	24	12	34	AA	AA	AA
105	BOURAMMA	25	F	5270	1.1	0.7	0.4	POSITIVE	9956	82	3	5	0	10	34	29	30	AA	Q	AA
106	MANJULA	24	F	6621	1.1	0.8	0.3	POSITIVE	8790	80	3	3	0	14	10	32	54	AA	AA	AA
107	SATISH	23	M	6825	1.1	0.8	0.3	POSITIVE	11000	82	2	3	0	13	33	17	31	AA	AA	AA
108	GANGADHAR	43	M	7359	1.1	0.6	0.5	POSITIVE	13420	81	3	4	0	12	22	20	32	AA	AA	AA
109	NEELABAI	42	F	7392	1.1	0.8	0.3	POSITIVE	12430	82	3	3	0	12	23	27	65	AA	AA	AA
110	BASAVARAJ	56	M	8376	1.1	0.8	0.3	POSITIVE	14250	81	4	4	0	11	28	30	35	AA	AA	AA
111	GIRIAPPA	46	M	10877	1.1	0.6	0.5	POSITIVE	12376	80	4	4	0	12	25	20	34	AA	AA	AA
112	ROOPA	45	F	11247	1.1	0.8	0.3	POSITIVE	13422	82	3	3	0	12	20	19	42	AA	AA	AA
113	PALLAVI	6	F	12125	1.1	0.6	0.5	POSITIVE	13245	83	2	3	0	12	35	27	31	AA	AA	AA
114	BASAVARAJ	47	M	12203	1.1	0.9	0.2	POSITIVE	11890	83	3	5	0	9	33	25	30	AA	AA	AA
115	REKHA	19	F	12571	1.1	0.7	0.4	POSITIVE	13456	82	3	4	0	11	30	15	32	AA	AA	AA
116	KASAPPA	46	M	12810	1.1	0.8	0.3	POSITIVE	12120	83	3	3	0	11	15	23	36	AA	AA	AA
117	BHARATHI	40	F	13228	1.1	0.8	0.4	POSITIVE	8890	81	4	4	0	11	29	34	35	AA	AA	AA
118	SHANTABAI	26	F	14240	1.1	0.6	0.5	POSITIVE	10450	81	3	4	0	12	26	9	34	AA	AA	AA
119	SHRIKANT	28	M	15412	1.1	0.8	0.3	POSITIVE	10100	84	3	3	0	10	18	28	45	AA	AA	AA
120	NAMRATHA	24	F	17088	1.1	0.7	0.4	POSITIVE	10560	82	3	4	0	11	34	18	48	AA	AA	AA
121	NAGAMMA	55	F	17837	1.1	0.6	0.5	POSITIVE	9850	81	4	3	0	12	32	25	45	AA	Q	AA
122	MUKTESH	25	M	22911	1.1	0.7	0.4	POSITIVE	9780	80	1	3	1	15	22	30	32	AA	AA	AA
123	MOHIT	35	M	23505	1.1	0.8	0.3	NEGATIVE	9940	83	2	4	1	10	30	20	52	AA	Q	AA

124	MEGHABAI	70	F	409	1	0.5	0.5	POSITIVE	7890	83	3	4	0	11	22	18	38	AA	Q	AA
125	TOPANNA	13	M	2274	1	0.7	0.3	POSITIVE	9680	82	3	3	0	12	32	12	44	AA	AA	AA
126	MANJUNATH	14	M	2862	1	0.5	0.5	POSITIVE	9980	81	3	4	0	12	31	25	58	AA	AA	AA
127	ASHOK	25	M	3152	1	0.8	0.2	POSITIVE	9840	86	4	3	0	7	34	20	30	AA	Q	AA
128	SHIVSARAN	34	M	3436	1	0.7	0.3	POSITIVE	8960	83	2	4	0	11	32	14	56	AA	AA	AA
129	RIYA	9	F	3481	1	0.8	0.2	POSITIVE	8890	81	2	3	0	14	22	8	43	AA	AA	AA
130	ABHILASH	46	M	4648	1	0.6	0.4	POSITIVE	8560	82	2	4	0	12	28	24	30	AA	AA	AA
131	KEERTHI	16	F	5151	1	0.5	0.5	POSITIVE	9800	82	3	3	1	12	25	25	31	AA	Q	AA
132	SHIVANAND	33	M	5689	1	0.5	0.5	POSITIVE	9980	81	4	4	0	11	18	12	34	AA	AA	AA
133	CHANAMMA	57	F	6448	1	0.8	0.2	NEGATIVE	9560	83	4	4	0	11	32	26	32	AA	AA	AA
134	MANJULA	20	F	6857	1	0.5	0.5	POSITIVE	7860	81	4	3	0	12	21	23	55	AA	AA	AA
135	RAGHAVENDRA	19	M	6998	1	0.5	0.5	NEGATIVE	8960	83	2	4	0	11	19	11	28	AA	AA	AA
136	BABU	35	M	7120	1	0.5	0.5	NEGATIVE	8870	83	3	4	0	10	20	22	34	AA	Q	AA
137	GEETHA	24	F	10230	1	0.6	0.4	NEGATIVE	6780	80	4	5	0	11	25	28	30	AA	AA	AA
138	SHIVARAJ	28	M	10656	1	0.6	0.4	POSITIVE	7760	81	3	4	0	12	39	20	32	AA	Q	AA
139	SAYAWWA	55	F	10661	1	0.7	0.3	POSITIVE	9870	82	3	4	0	11	18	15	34	AA	Q	AA
140	SAROJA	16	F	12903	1	0.7	0.3	POSITIVE	9970	81	3	4	0	12	20	12	34	AA	AA	AA
141	SAJANNA	58	M	14426	1	0.8	0.2	NEGATIVE	8560	83	3	3	0	11	26	13	39	AA	AA	AA
142	GANGABAI	46	F	14528	1	0.6	0.4	POSITIVE	8950	81	4	5	0	10	18	22	42	AA	AA	AA
143	ABHISHEK	14	M	16426	1	0.3	0.7	POSITIVE	9760	80	3	4	1	11	31	25	35	AA	Q	AA
144	ANAND	23	M	20772	1	0.5	0.5	POSITIVE	7450	90	2	2	0	6	34	25	38	AA	AA	AA
145	SANGANAGOUDA	22	M	21915	1	0.5	0.5	POSITIVE	8750	80	1	2	0	17	34	25	50	AA	AA	AA
146	YELAPPA	27	M	21969	1	0.5	0.5	POSITIVE	8695	81	3	3	0	13	30	26	53	AA	Q	AA
147	MAMTA	32	F	23661	1	0.5	0.5	NEGATIVE	8850	82	2	4	0	12	31	26	39	AA	AA	AA
148	DANAMMA	25	F	2798	0.9	0.6	0.3	POSITIVE	8960	82	3	4	0	11	12	15	35	AA	Q	AA
149	NINGAWWA	40	F	3264	0.9	0.5	0.4	POSITIVE	7800	80	2	5	0	13	33	22	42	AA	AA	AA

150	YATHISH	23	M	3464	0.9	0.5	0.4	NEGATIVE	8770	81	3	4	0	11	25	22	63	AA	AA	AA
151	LAXMAN	32	M	5336	0.9	0.5	0.4	POSITIVE	9560	81	2	4	0	13	26	15	30	AA	AA	AA
152	HARSHA	33	M	6002	0.9	0.5	0.4	NEGATIVE	8900	80	2	5	0	13	24	30	35	AA	AA	AA
153	HANAMANTGOUDA	39	M	7580	0.9	0.5	0.4	POSITIVE	10900	80	3	4	0	13	21	20	31	AA	Q	AA
154	REVATI	35	F	9082	0.9	0.6	0.3	POSITIVE	12320	81	2	4	0	13	20	18	47	AA	AA	AA
155	DEVAMMA	50	F	9624	0.9	0.6	0.3	NEGATIVE	10070	81	4	4	0	11	20	24	30	AA	AA	AA
156	HANAMANTH	29	M	10090	0.9	0.5	0.4	POSITIVE	11320	80	2	4	0	14	20	22	34	AA	Q	AA
157	GIRISH	30	M	12585	0.9	0.7	0.2	NEGATIVE	11450	81	5	5	0	9	20	22	30	AA	AA	AA
158	JAYASHREE	34	F	13240	0.9	0.6	0.3	POSITIVE	12340	80	4	5	0	11	20	22	35	AA	Q	AA
159	BHIMARAYA	28	M	13358	0.9	0.5	0.4	POSITIVE	10670	81	4	3	0	12	15	18	36	AA	AA	AA
160	PRIYANKA	28	F	14696	0.9	0.6	0.3	NEGATIVE	9870	80	3	5	0	12	16	14	38	AA	Q	AA
161	SHRIDEVI	25	F	14892	0.9	0.5	0.4	NEGATIVE	8970	80	2	3	0	15	26	27	65	AA	AA	AA
162	PREM KUMAR	18	M	19598	0.9	0.5	0.4	POSITIVE	7960	80	2	4	0	14	32	10	32	AA	AA	AA
163	MADIWALLAPPA	28	M	22137	0.9	0.5	0.4	NEGATIVE	7760	80	4	5	0	11	34	23	34	AA	Q	AA
164	AWAMMA	25	F	22470	0.9	0.5	0.4	POSITIVE	8750	80	3	4	0	13	29	32	35	AA	Q	AA
165	SHIVANAND	20	M	2609	0.8	0.3	0.5	NEGATIVE	7890	80	3	4	0	13	36	22	33	AA	AA	AA
166	LAKSHMI	14	F	2753	0.8	0.5	0.3	POSITIVE	7760	80	3	5	0	12	27	28	34	AA	Q	AA
167	VILAS	25	M	2909	0.8	0.5	0.3	POSITIVE	6750	80	3	3	0	14	11	10	46	AA	Q	AA
168	SHIVARAJ	35	M	3108	0.8	0.6	0.2	POSITIVE	5680	80	2	4	0	14	19	14	32	AA	Q	AA
169	SHIVANAND	24	M	3226	0.8	0.5	0.3	POSITIVE	12000	80	3	4	0	13	34	22	34	AA	Q	AA
170	RAVIKUMAR	28	M	3353	0.8	0.4	0.4	NEGATIVE	10500	81	3	3	1	12	20	14	38	AA	AA	AA
171	SHANTABAI	35	F	4095	0.8	0.5	0.3	POSITIVE	11670	80	3	4	0	13	12	22	57	AA	AA	AA
172	AWAMMA	45	F	4297	0.8	0.6	0.4	NEGATIVE	12360	81	4	5	0	10	24	25	30	AA	AA	AA
173	JANABAI	19	F	5079	0.8	0.4	0.4	NEGATIVE	10080	80	3	3	0	14	19	21	32	AA	Q	AA
174	MANOJ	14	M	5554	0.8	0.4	0.3	POSITIVE	11000	80	3	5	0	12	34	16	34	AA	Q	AA
175	RENUKA	25	F	6524	0.8	0.5	0.3	NEGATIVE	11000	80	3	5	0	12	14	22	41	AA	AA	AA

176	SOORAJ	23	M	6680	0.8	0.5	0.3	POSITIVE	12350	80	2	5	0	13	34	28	43	AA	AA	AA
177	DANAPPA	49	M	7628	0.8	0.5	0.3	NEGATIVE	11340	80	3	5	0	12	24	20	31	AA	Q	AA
178	RAMAYYA	48	M	9093	0.8	0.6	0.2	NEGATIVE	11270	80	4	4	0	12	24	17	36	AA	AA	AA
179	ASHWINI	35	F	9230	0.8	0.4	0.4	NEGATIVE	6750	85	2	4	0	9	25	20	24	AA	AA	AA
180	SHARANAWWA	34	F	10439	0.8	0.5	0.3	NEGATIVE	8790	80	2	4	0	14	15	10	43	AA	Q	AA
181	KALPANA	20	F	11425	0.8	0.6	0.2	POSITIVE	8890	80	2	4	0	14	22	26	37	AA	AA	AA
182	ANIL	20	M	11954	0.8	0.5	0.3	POSITIVE	9870	86	4	2	0	8	12	25	34	AA	Q	AA
183	SURESH	35	M	12547	0.8	0.6	0.2	NEGATIVE	6780	80	3	5	0	12	32	15	36	AA	Q	AA
184	VILAS	26	M	13732	0.8	0.6	0.2	NEGATIVE	11340	80	3	5	0	12	28	21	38	AA	AA	AA
185	SHARAN	55	M	13895	0.8	0.4	0.4	NEGATIVE	9000	80	2	5	0	13	20	11	33	AA	Q	AA
186	BHAGAPPA	55	M	15166	0.8	0.6	0.2	POSITIVE	9860	80	3	5	0	12	26	30	35	AA	Q	AA
187	DEEPAK	20	M	15321	0.8	0.6	0.2	POSITIVE	6780	80	3	4	0	13	32	28	30	AA	AA	AA
188	SANGAMESH	24	M	16405	0.8	0.5	0.3	POSITIVE	7600	81	2	4	0	13	34	23	48	AA	AA	AA
189	AMBAWWA	46	F	21296	0.8	0.4	0.4	POSITIVE	8905	80	3	3	0	14	32	20	32	AA	AA	AA
190	VISHWANATH	5	M	21561	0.8	0.5	0.3	POSITIVE	5460	80	2	4	0	14	37	26	58	AA	Q	AA
191	JETINGARAYA	17	M	22125	0.8	0.5	0.3	POSITIVE	6780	82	3	5	0	10	24	20	38	AA	AA	AA
192	MAHANANDA	18	M	22486	0.8	0.5	0.3	POSITIVE	7890	80	2	5	0	13	40	22	44	AA	AA	AA
193	DEEPAK	24	M	23745	0.8	0.5	0.3	POSITIVE	5670	80	3	5	0	12	20	18	54	AA	Q	AA
194	MAHADEV	25	M	26527	0.8	0.5	0.3	POSITIVE	6680	80	3	4	0	13	19	21	40	AA	Q	AA
195	REVANASIDDA	35	M	30140	0.8	0.5	0.3	NEGATIVE	5600	80	2	5	0	12	32	16	34	AA	Q	AA
196	TEJASWINI	16	F	4501	0.7	0.4	0.3	NEGATIVE	8900	80	3	5	0	12	33	23	45	AA	AA	AA
197	CHANAMMA	35	F	5360	0.7	0.4	0.3	POSITIVE	9600	80	4	5	0	11	26	20	32	AA	AA	AA
198	RAVI	13	M	5547	0.7	0.4	0.3	POSITIVE	10560	80	2	5	0	13	22	21	56	AA	Q	AA
199	SAVITRI	15	F	5602	0.7	0.4	0.3	NEGATIVE	7860	80	4	4	0	12	16	24	75	AA	AA	AA
200	ASHWINI	25	F	5755	0.7	0.4	0.3	POSITIVE	8600	80	3	2	0	15	17	20	45	AA	Q	AA
201	BHAVYA	28	F	5860	0.7	0.4	0.3	NEGATIVE	5800	80	3	5	0	12	24	25	30	AA	Q	AA

202	SAVITA	23	F	6226	0.7	0.4	0.3	NEGATIVE	9800	80	2	3	1	14	23	22	42	AA	AA	AA
203	RAMANAGOUDA	22	M	10232	0.7	0.4	0.3	POSITIVE	8760	80	3	3	0	14	18	19	30	AA	Q	AA
204	VEERESH	18	M	12490	0.7	0.5	0.2	NEGATIVE	11800	81	3	4	0	12	30	27	55	AA	Q	AA
205	SHRIKANT	20	M	12703	0.7	0.5	0.2	NEGATIVE	5690	80	2	5	0	13	24	22	34	AA	Q	AA
206	MADIWALLAPPA	20	M	14988	0.7	0.4	0.3	POSITIVE	7680	80	3	5	0	12	10	24	38	AA	AA	AA
207	CHANDRAM	55	M	14992	0.7	0.4	0.3	POSITIVE	8965	80	4	3	0	13	25	20	41	AA	AA	AA
208	UMAR	19	M	15018	0.7	0.4	0.3	POSITIVE	9800	80	3	5	0	12	41	21	51	AA	Q	AA
209	RAKSHITHA	22	F	15313	0.7	0.5	0.2	NEGATIVE	9000	80	2	5	0	13	18	18	49	AA	Q	AA
210	SHANTABAI	17	F	15995	0.7	0.4	0.3	NEGATIVE	11897	81	3	4	0	12	20	12	53	AA	AA	AA
211	PRADEEP	18	M	18741	0.7	0.4	0.3	POSITIVE	12340	80	4	4	0	12	25	18	66	AA	AA	AA
212	ADITYA	20	M	19094	0.7	0.4	0.3	POSITIVE	11700	85	3	4	0	8	15	26	34	AA	AA	AA
213	ABHILASH	13	M	19321	0.7	0.4	0.3	POSITIVE	10320	81	3	5	0	11	33	20	43	AA	AA	AA
214	DANAMMA	22	F	20139	0.7	0.4	0.3	NEGATIVE	11300	81	4	5	0	10	28	18	32	AA	AA	AA
215	SWATHI	18	F	26082	0.7	0.4	0.3	NEGATIVE	8870	80	3	3	0	14	28	21	38	AA	Q	AA
216	PREETI	18	F	27292	0.7	0.4	0.3	NEGATIVE	5670	80	3	5	0	12	33	17	39	AA	Q	AA
217	APPASAHEB	34	M	29032	0.7	0.4	0.3	NEGATIVE	8970	80	4	4	0	12	28	35	40	AA	Q	AA
218	VINAYAK	25	M	30013	0.7	0.3	0.4	NEGATIVE	9850	81	4	4	0	11	11	18	76	AA	Q	AA
219	SOMU	22	M	1683	0.6	0.4	0.2	NEGATIVE	9460	80	3	4	0	13	20	32	38	AA	AA	AA
220	RENUKA	35	F	2587	0.6	0.3	0.3	POSITIVE	5895	80	4	3	0	13	30	15	34	AA	AA	AA
221	HIRAGAPPA	23	M	2896	0.6	0.3	0.3	NEGATIVE	6890	80	3	5	0	12	27	20	39	AA	Q	AA
222	HENA	24	F	5345	0.6	0.3	0.3	NEGATIVE	6870	81	2	4	0	13	26	24	30	AA	AA	AA
223	SIDDAPPA	45	M	6270	0.6	0.3	0.3	NEGATIVE	9980	80	4	3	0	13	11	9	44	AA	Q	AA
224	SIDDAPPA	35	M	10002	0.6	0.4	0.2	NEGATIVE	8580	80	2	5	0	13	20	6	45	AA	AA	AA
225	RAMESH	29	M	11161	0.6	0.4	0.2	NEGATIVE	5890	80	3	3	0	14	20	11	62	AA	Q	AA
226	RAJU	24	M	13189	0.6	0.4	0.2	POSITIVE	6700	80	3	5	0	12	23	6	34	AA	Q	AA
227	KAVITA	14	F	16299	0.6	0.3	0.3	NEGATIVE	6608	80	3	4	0	13	15	22	32	AA	Q	AA

228	MAHESH	35	M	19854	0.6	0.6	0.2	NEGATIVE	10500	80	2	4	0	14	32	14	48	AA	AA	AA
229	JYOTHI	22	F	20961	0.6	0.4	0.2	NEGATIVE	11000	80	3	4	0	13	30	28	32	AA	Q	AA
230	SAIDU	25	M	24797	0.6	0.3	0.3	NEGATIVE	10230	80	2	5	1	12	20	22	36	AA	AA	AA
231	IRANNA	19	M	29067	0.6	0.3	0.3	NEGATIVE	11600	81	4	4	0	11	27	24	34	AA	AA	AA
232	SHIVAYOGEPPA	65	M	429	0.5	0.3	0.2	POSITIVE	9600	80	3	5	0	12	32	12	37	AA	Q	AA
233	RAJU	16	M	2871	0.5	0.3	0.2	POSITIVE	9670	80	3	5	0	12	23	9	43	AA	Q	AA
234	MANJU	8	M	5025	0.5	0.3	0.2	POSITIVE	5890	81	3	3	0	13	25	22	55	AA	Q	AA
235	SUMITRA	34	F	6490	0.5	0.3	0.3	NEGATIVE	6754	80	4	3	0	13	23	27	31	AA	Q	AA
236	SURESH	28	M	6986	0.5	0.3	0.3	POSITIVE	5700	80	3	3	0	14	43	21	58	AA	Q	AA
237	VEERESH	12	M	9442	0.5	0.3	0.2	POSITIVE	5230	80	4	5	0	11	15	28	65	AA	Q	AA
238	CHANDRASHEKAR	30	M	4586	0.5	0.3	0.2	POSITIVE	8600	81	4	5	0	10	20	18	33	AA	Q	AA
239	SAHIDABANU	13	F	12610	0.5	0.3	0.2	NEGATIVE	9870	80	4	5	0	11	15	18	66	AA	Q	AA
240	DAVALATRAY	38	M	12828	0.5	0.3	0.2	NEGATIVE	7890	80	3	5	0	12	20	22	30	AA	Q	AA
241	TUKARAM	27	M	14870	0.5	0.3	0.2	NEGATIVE	7600	80	2	5	0	13	20	24	34	AA	Q	AA
242	BOURAMMA	58	F	16291	0.5	0.3	0.2	NEGATIVE	5800	83	2	3	0	12	30	34	39	AA	Q	AA
243	ASHOK	24	M	17272	0.5	0.3	0.2	POSITIVE	9500	84	2	5	0	9	10	28	30	AA	Q	AA
244	BALAJI	9	M	19355	0.5	0.3	0.2	POSITIVE	7860	80	4	5	1	10	29	30	40	AA	Q	AA
245	NAGESH	22	M	20319	0.5	0.3	0.2	NEGATIVE	6780	80	3	4	0	13	28	15	43	AA	Q	AA
246	SANTOSH	15	M	22498	0.5	0.3	0.2	POSITIVE	8760	80	3	5	0	12	30	8	35	AA	AA	AA
247	PREMA	25	F	24735	0.5	0.3	0.2	NEGATIVE	8690	82	3	4	0	11	20	38	20	AA	Q	AA
248	REVANASIDDA	35	M	30148	0.5	0.3	0.2	NEGATIVE	9500	80	3	4	0	13	14	9	35	AA	Q	AA
249	ROOPA	20	F	24196	1.1	0.6	0.5	POSITIVE	10560	83	2	2	1	12	20	21	30	AA	AA	AA
250	MAHENDRA	50	M	24972	1.2	0.9	0.3	POSITIVE	10400	85	2	1	0	12	23	21	34	AA	AA	AA