

CHAPTER ONE

Introduction

Introduction

1-1 Tuberculosis (T.B)

T.B remains a major global public problem. In less developed countries where population is dense and hygienic standards poor, tuberculosis remains a major fatal disease. (world journal of gastroenterology 2004). In 2011 - 8.7 million people fell ill with T.B and 1.4 million died from T.B. Over 95% of T.B death occur in low-and middle-income countries and it is among the top three of death for women aged 15-45 (WHO 2013).

There is many investigation modalities used for diagnosing T.B; conventional x-ray (chest x-ray), CT, laboratory investigations and ultrasound.

Diagnosis of abdominal tuberculosis is often difficult because clinical manifestations and results of laboratory studies are non specific. If sonographic findings are sufficiently characteristic for diagnosis; sonography would be useful (R Jain et al -1995)

Most TB can be successfully treated using combination of powerful antibiotic medicines. Antibiotic drugs fight bacteria infection by either killing the bacteria that cause the infection or preventing them from reproducing.

Tuberculosis is a bacterial disease which caused by members of Mycobacterium tuberculosis complex .Which consist of M.tuberculosis, M.bovis, M.microti M.africanum, and M.canettii. Disease caused by M.tuberculosis is referred to as human tuberculosis (HTB).pathology of T.B –university of Pretoria).Tuberculosis can attack any part of a body most frequently the lungs. The primary infection may spread through the body blood stream or lymphatic causing military tuberculosis (a highly fatal form if not adequately treated).

Mycobacterium is airborne bacteria pass from person to person through the air (Sudan medical relief.org).

About one-third of the world's population has latent T.B ,which mean people have been infected by T.B bacteria but are not yet ill with disease and can't transmit the disease .person with compromised immune systems ,such as people living with HIV ,malnutrition or diabetes or people who use tobacco have much higher risk of falling ill (WHO 2013).

In spite the fact that tuberculosis (abbreviated TB for tubercle bacilli-the name of the organism that cause TB) is a treatable and curable disease, it is currently the second most common cause of death from infectious diseases in the world. This blatant contradiction seems to invite an obvious inquiry –if doctors know how to cure TB, why it is still such a deadly problem? Like TB it self, the answer to this question is complicated, multifaceted, and extremely relevant to the current state of health around the world.

TB has existed since the beginning of the time. It is profile and history have confounded science and society throughout the ages .The medical tools now exist to make TB a disease of the past, but it still flourishes in places where poverty thrives and health care systems are inadequate and insufficient .Understanding the relationship between the physical and social aspects of the TB first requires knowledge of the disease.

TB begins when people inhale airborne M.bacterium. When the bacteria are inhaled by a healthy person, the immune system is usually strong enough to defend the body and the bacteria remain harmless producing no infection or subsequent disease. If, however, the bacteria are more powerful than the response produced by the immune system to stop the attack, infection begins and all-out disease may fallow.

TB infection usually affects the lungs, producing a kind of TB called pulmonary TB. Less frequently, TB infection moves from damaged lung –tissue through blood steam and affects other parts of the body, like the kidney, liver, spleen, pancreas, lymph node or brain. TB that does not affect the lungs is called extra-pulmonary TB, and since TB must be spread from one person to another through the air, this type of the disease is usually not contagious.

TB is generally classified as being either latent or active.

- Latent TB:

In this condition, you have a TB infection, but the bacteria remain in your body in an inactive state and cause no symptoms. Latent TB, also called inactive TB or TB infection, isn't contagious. However, it can turn into active TB, so treatment is important for the person with latent TB and to help control the spread of TB in general. An estimated one-third of the world's population has latent TB.

- Active TB :

This condition makes you sick and can spread to others. It can occur in the first few weeks after infection with the TB bacteria, or it might occur years later.

Most infections are asymptomatic and latent, but about one in ten latent infections eventually progresses to active disease.

Tuberculosis is divided into two types:

- **Pulmonary tuberculosis (TB)**

T.B infection of the lungs is known as pulmonary TB.

Symptoms include: A persistent cough of more than three weeks that brings up phlegm, breathlessness, lack of appetite and weight loss, a high temperature of 38°C (100.4°F) or above, [night sweats](#), extreme tiredness or fatigue, unexplained pain for more than three week.

- **Extra pulmonary tuberculosis (TB)**

In some cases, TB can occur outside the lungs, which is known as extra pulmonary TB.

A TB infection can affect the lymph nodes, bones and joints, the digestive system, genitourinary system, nervous system, spleen, liver and pancreas.

The symptoms of tuberculosis depend on where the infection occurs.

TB usually develops slowly. Your symptoms might not begin until months or even years after you were initially exposed to the bacteria

- *Symptoms of lymph node TB include:* persistent, painless swelling of the lymph nodes, which usually affects nodes in the neck, but swelling can occur in nodes throughout your body, Over time, the swollen nodes can release fluid through the skin.
- *Symptoms of skeletal TB include:* Bone pain, curving of the affected bone or joint, loss of movement or feeling in the affected bone or joint, weakened bone that may fracture easily.
- Symptoms of gastrointestinal TB include: abdominal pain, [diarrhoea](#), rectal bleeding.
- Symptoms of genitourinary TB include: a burning sensation when you urinate, [blood in your urine](#), a frequent urge to pass urine during the night, Groin pain.

TB Risk Factors

Generally people at high risk for developing TB disease fall into two categories:

People who have been recently infected with TB bacteria

People with medical conditions that weaken the immune system

You have a higher chance of getting TB disease if you have HIV infection, have been infected with TB bacteria in the last two years, have other [health problems](#) that make it hard for your body to fight disease, abuse alcohol or use illegal drugs, were not treated correctly for TB infection or TB disease in the past, have Leukemia , lymphoma and other cancers, have poorly controlled diabetes mellitus. (www.mayoclinic.org/diseases).

1-2 Problem of study:

Tuberculosis continues to be an important public health problem worldwide, Sudan characterized by high prevalence rate of tuberculosis and it is a major cause of morbidity and mortality in Sudan. Also lack of knowledge such as experimental information related to ‘gray-scale’ abdominal ultrasound finding of tuberculosis in patients especially Sudanese represent gap in the knowledge.

1-3 Objectives:

1-3-1 General objective

To characterize abdominal findings of tuberculosis patients using ultrasound in order to evaluate the impact of this disease on the visceral organs.

1-3-2 Specific objectives

- To evaluate the size - echo texture and masses of liver, spleen, pancreas, kidneys and any abdominal masses or abscesses.
- To find ascities and pleural effusion
- To evaluate lymph nodes and omentum thickening.

1-4 Significance of the study:

This study will give a clear picture about the impact of tuberculosis on the abdominal organs; therefore it will assist the physician in diagnosis the complications of tuberculosis in abdominal organs using ultrasound scan.

1-5 Overview of the study:

Chapter one deals with introduction which includes objectives and problem of the study. Chapter two presented scientific background includes a review of anatomy, physiology and pathology; also a review of scholarly literature ‘previous studies. Chapter three includes methods and material used to collect the data. Chapter four presented the result of the collected data, and finally chapter five gives discussion of the results, conclusion and recommendations.

CHAPTER TWO

LITERATURE REVIEW

2-1 Abdominal Tuberculosis

Tuberculosis is still common in the tropics and continues to figure in the differential diagnosis of many medical and surgical conditions.

Abdominal tuberculosis is defined as tuberculosis infection of the abdomen including gastrointestinal tract, peritoneum, omentum, mesentery and its nodes and other solid intra-abdominal organs like Liver, Spleen, Pancreas and kidney.

Mycobacterium tuberculosis, *Mycobacterium Bovis* and atypical *Mycobacteria* can cause abdominal tuberculosis. *Mycobacterium tuberculosis* is the most frequently isolated organism. Any part of the gastrointestinal tract may be affected by tuberculosis. Involvement of the upper gastrointestinal tract such as mouth, oropharynx, esophagus, duodenum are unusual. (Vimlesh and kabra 2006).

Mycobacterium tuberculosis spread to the abdomen by several routes:

- 1-The peritoneum, mesenteric nodes, and the intestines may become infected during the bacteraemic phase that may follow primary pulmonary TB in childhood.
- 2-Secondary intestinal disease can arise from swallowed sputum containing tuberculous bacilli.
- 3-*Mycobacteria* may spread from adjacent organs (fallopian tube).
- 4-Intestinal infection can result from lymphatic spread, from mesenteric lymph nodes.
- 5-*Mycobacteria* may also be get disseminated in the bile since they are sequestered and excreted from granulomas in the liver.

The common sites of involvement of the GIT are the ileocaecal region followed by the colon and jejunum. (A k agarwal 2007)

Abdominal tuberculosis is divided into gastrointestinal (enteric), peritoneal and lymph nodal form, and others each can be primary or secondary to swallowing to sputum or from lympho-hematogenous spread.

Manifestations of abdominal tuberculosis:

A. Enteric form:

Esophageal	ulcer –hematemesis- broncho esophageal fistula- stricture-dysphagia.
Gastroduodenal	ulcer- Gastric outlet obstruction- Hematemesis- Duodenal obstruction due to extrinsic- Compression- Obstructive jaundice.
Intestinal	ulcero-constrictive- Stricture- Perforation- lower GI bleeding- mal-absorption- appendicitis.
Anorectum	Bleeding- Stricture- abscess- fistula

B .Peritoneal

Wet/ ascetic type	Generalized
Fibro-adhesive type	Localized.
	Mass- Fistula.

C. Lymph node

(ileocecal, pyloro-duodenal). Mass-
Obstruction.

D. Others

Hepatobiliary	Miliary- Focal- Gallstone- Obstructive jaundice.
Spleen	Hypersplenism- Splenomegaly.
Pancreatic	Abscess- Obstructive Jaundice.

(Vimlesh Seth- S K kabra 2006.

Characteristic sonographic features of abdominal tuberculosis are mesenteric thickness of 15 mm or more, increase mesenteric echogenicity due to fat deposition and lymphatic obstruction, ascites, dilated small bowel loops, matted fixed bowel loops, omentum, inflammation and thickening. (Suraj Gupte 2007) .

2-2-1 STOMACH

The stomach is located in the upper left quadrant of the abdominal cavity, to the left of the liver and in front of the spleen. Although part of the alimentary tube, the stomach is not a tube, but rather a sac that extends from the esophagus to the small intestine. Because it is a sac, the stomach is a reservoir for food, so that digestion proceeds gradually and we do not have to eat constantly. Both mechanical and chemical digestion takes place in the stomach. The cardiac orifice is the opening of the esophagus, and the fundus is the portion above the level of this opening. The body of the stomach is the large central portion, bounded laterally by the greater curvature and medially by the lesser curvature. The pylorus is adjacent to the duodenum of the small intestine, and the pyloric sphincter surrounds the junction of the two organs. The fundus and body are mainly storage areas, whereas most digestion takes place in the pylorus. The gastric pits are the glands of the stomach and consist of several types of cells; their collective secretions are called gastric juice. Mucous cells secrete mucus, which coats the stomach lining and helps prevent erosion by the gastric juice. Chief cells secrete pepsinogen, an inactive form of the enzyme pepsin. Parietal cells produce hydrochloric acid (HCl); the external muscle layer of the stomach consists of three layers of smooth muscle: circular, longitudinal, and oblique layers. These three layers are innervated by the myenteric plexuses of the enteric nervous system. Stimulatory impulses are carried from the CNS by the vagus nerves (10th cranial) the pyloric sphincter is usually contracted when the stomach is churning food; it relaxes at intervals to permit small amounts of chyme to pass into the duodenum. This sphincter then contracts again to prevent the backup of intestinal contents into the stomach. (Valerie c .scanlon Tina sanders 2007)

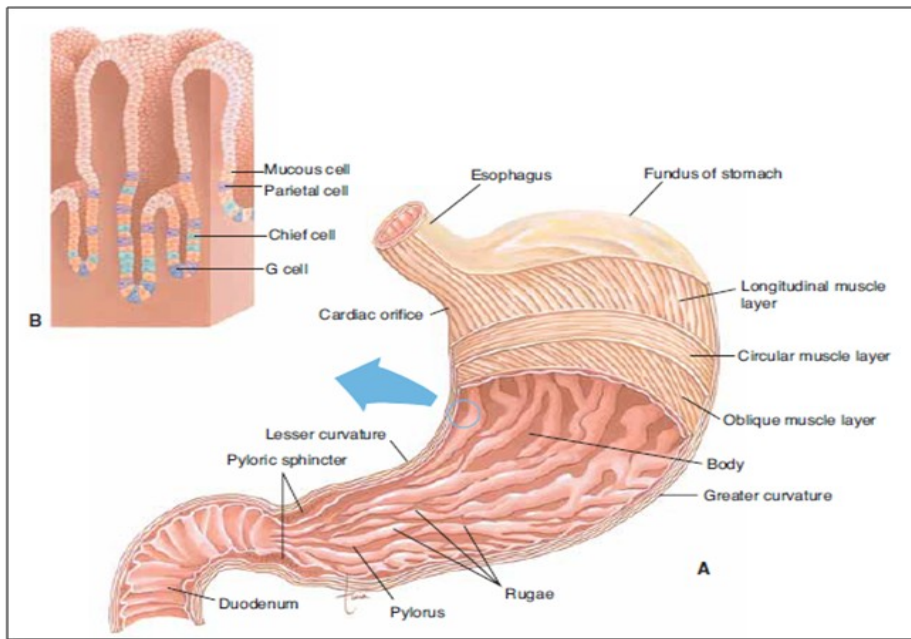


Figure 2-1 Stomach

2-2-2 Tuberculosis of Stomach

Radiologically tuberculosis of the stomach may present as shallow ulceration, agranulomatous mass or as fibrosis. The most common of these is ulceration at the pylorus, which causes gastric outlet obstruction. Tuberculosis gastric ulceration has been reported on both the greater and lesser curvatures of the stomach, usually surrounded by indurations and therefore reducing peristalsis. These ulcers are usually shallow but can be quite large. The likely mistaken radiological diagnosis will be lymphoma or carcinoma.

The tuberculosis granulomatous mass in the stomach is the least common presentation of this uncommon form of tuberculosis. (D.H. Connor I.J. Dunn 2001).

2-3-1 SMALL INTESTINE

The small intestine is about 1 inch (2.5 cm) in diameter and approximately 20 feet (6 m) long and extends from the stomach to the cecum of the large intestine. The duodenum is the first 10 inches (25 cm) of the small intestine. The common bile duct enters the duodenum at the ampulla of Vater. The jejunum is about 8 feet long, and the ileum is about 11 feet in length. In a living person, however, the small intestine is always contracted and is therefore somewhat shorter. Digestion is completed in the small intestine, and the end products of digestion are absorbed into the blood and lymph. The mucosa has simple columnar epithelium that includes cells with

microvilli and goblet cells that secrete mucus. Enteroendocrine cells secrete the hormones of the small intestine. Lymph nodules called Peyer's patches are especially abundant in the ileum to destroy absorbed pathogens. The external muscle layer has the typical circular and longitudinal smooth muscle layers that mix the chyme with digestive secretions and propel the chyme toward the colon. Stimulatory impulses to the enteric nerves of these muscle layers are carried by the vagus nerves. (Valerie c Scanlon Tina sanders 2007).

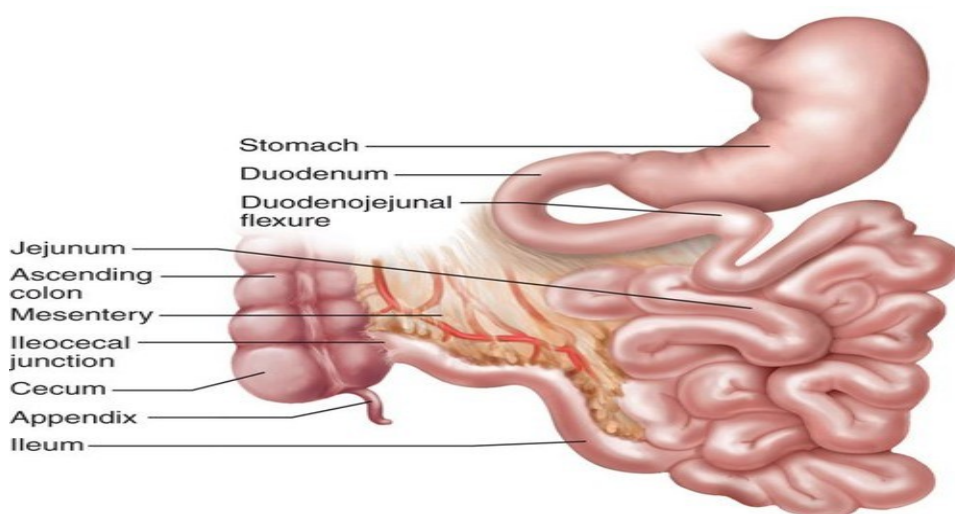


Figure 2-2 Small intestine

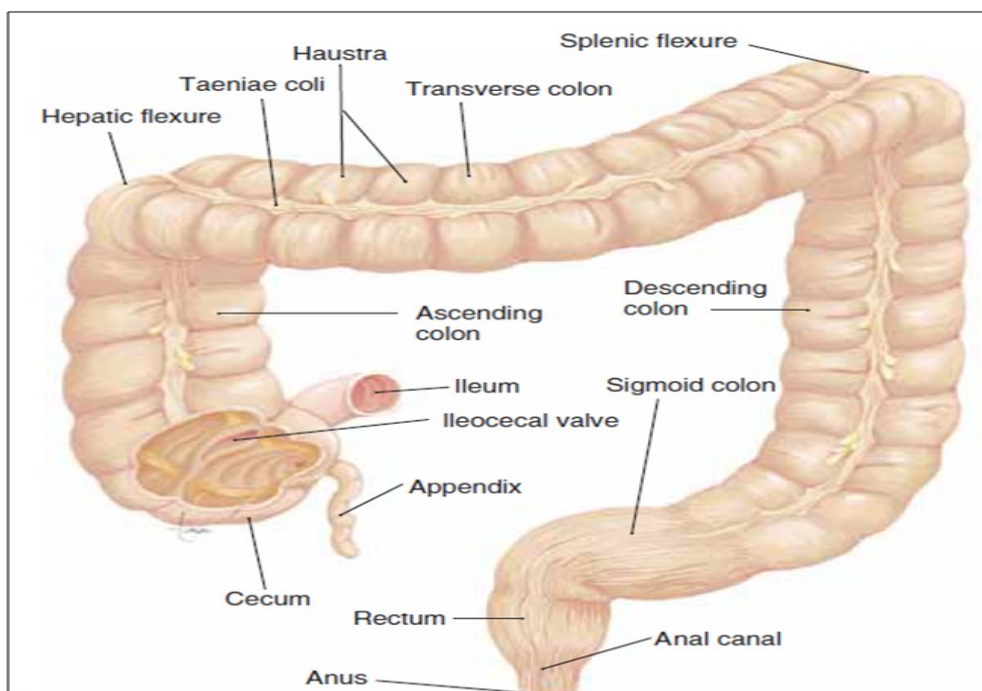
2-3-2 Tuberculosis of duodenum and small intestine

Intestinal tuberculosis is a chronic inflammation of the bowel caused by *Mycobacterium tuberculosis*, the ileo-cecal area is the most common site. The clinical presentation in most cases is of upper small bowel obstruction, but occasionally the obstruction is lower, in the ileum. There is usually a history of intermittent attacks of abdominal pain, with nausea and sometimes vomiting; when the small bowel is involved, mal-absorption and malnutrition occur more frequently. Eventually the obstruction becomes persistent. Structures are most common in the ileum but may be seen in the jejunum and even in the duodenum. Tuberculosis strictures are not as long as those of regional enteritis. Occasionally in tuberculosis there are fistulae in addition to the strictures, and the differential diagnosis then becomes impossible without histological examination. Calcified enteroliths are commonly seen within dilated loops of small bowel proximal to tuberculosis strictures, but more uncommon in any other chronic enteritis (D.H. Connor I.J. Dunn 2001).

2-4-1 LARGE INTESTINE

The large intestine, also called the colon, is approximately 2.5 inches (6.3 cm) in diameter and 5 feet (1.5 m) in length. It extends from the ileum of the small intestine to the anus, the terminal opening. The cecum is the first portion, and at its junction with the ileum is the ileocecal valve, which is not a sphincter but serves the same purpose. Attached to the cecum is the appendix, a small, dead-end tube with abundant lymphatic tissue. The appendix seems to be a vestigial organ. The remainder of the colon consists of the ascending, transverse, and descending colon, which encircle the small intestine; the sigmoid colon, which turns medially and downward; the rectum; and the anal canal. The rectum is about 6 inches

long, and the anal canal is the last inch of the colon that surrounds the anus. No digestion takes place in the colon. The only secretion of the colonic mucosa .The functions of the colon are the absorption of water, minerals, and vitamins and the elimination of indigestible material is mucus, which lubricates the passage of fecal material. The external anal sphincter is made of skeletal muscle and surrounds the internal anal sphincter if defecation must be delayed, the external sphincter may be voluntarily contracted to close the anus (Valerie c Scanlon Tina 2007).



Large intestine Figure 2-3

2-4-2 Tuberculosis of the cecum

Tuberculosis of the cecum is one of the most common types of intra-abdominal tuberculosis and in some series it accounts for up to 60 percent of all tuberculosis bowel infections .The clinical symptoms are vague .There may be alternating diarrhea and constipation , changing in bowel habit, a palpable mass , pain in the right iliac fossa. When

tuberculosis affects the cecum and distal ileum, initially there will be intense ileo-cecal spasm seen on barium studies. Later the lumen and size of the cecum are compromised as the granulomas form into a mass and undergo fibrosis. (D.H.connor –I.J.dunn 2001)

Sonography used in patient with intestinal tuberculosis to document it is classic features, i.e bowel wall thickening, hyperemia, stricture, and mesenteric lymphadenopathy. When tuberculosis peritonitis co-exist, sonography shows ascites, omentum cake, and thickening mesentery with an adherent small bowel loop: thus, ultrasonography may be used as a primary investigative tool in patient with suspected or recurrent tuberculosis.

2-4-3 Sonographic findings of intestinal tuberculosis:

The normal bowel wall is visualized as single circular, hypoechoic layer surrounding hyperechoic bowel contents, foodstuff, or feces.

The hypoechoic layer is considering being the muscular layer. The normal bowel wall is uniform with an average thickness of 2-3 mm if distended and 3-5 mm if not. When the wall thickness in the transverse section is more than 5 mm, the bowel is regarded as being pathologically thickened.

The ileo-cecal region is most commonly involved in tuberculosis. The principal sonographic features of intestinal tuberculosis are diffuse, uniform, concentric, and circumferential wall thickening of the terminal ileum, cecum and ascending colon. These wall thickenings are continuous without skip lesions. Mild wall thickening of small bowel may be overlooked, and it is difficult to distinguish individual loops when they adhere to each other. (G .Maconi –G. Bianchi 2007).

2-5-1 PERITONEUM

The membranes of the abdominal cavity are serous membranes called the peritoneum and mesentery. The peritoneum is the membrane that lines the entire abdominal wall, and the mesentery is the continuation of this membrane, folded around and covering the outer surfaces of the abdominal organs.

2-5-2 Great omentum (epiploon)

Is a large fold of visceral [peritoneum](#) that hangs down from the stomach. It extends from the greater curvature of the [stomach](#), passing in front of the small intestines and reflects on itself to ascend to the [transverse colon](#) before reaching to the posterior [abdominal wall](#). The common anatomical term "epiploic" derives from "epiploon" from the Greek "epipleein" meaning to float or sail on, since the greater omentum appears to float on the surface of the intestines. The greater omentum is the largest peritoneal fold. It consists of a double sheet of [peritoneum](#), folded on it self so that it is made up of four layers.

The two layers which descend from the greater curvature of the stomach and commencement of the [duodenum](#) pass in front of the [small intestines](#), sometimes as low down as the [pelvis](#); they then turn upon themselves, and ascend again as far as the [transverse colon](#), where they separate and enclose that part of the intestine.

These individual layers may be easily demonstrated in the young subject, but in the adult they are more or less inseparably blended.

The left border of the greater omentum is continuous with the [gastro-lineal ligament](#); its right border extends as far as the commencement of the [duodenum](#).

The greater omentum is usually thin, a [cribriform](#) present's appearance, and always contains some [adipose tissue](#), which in obese people

accumulates in considerable quantity. The greater omentum develops from the [dorsal mesentery](#) that connects the [stomach](#) to the posterior abdominal wall.. The right and left [gastroepiploic vessels](#) provide the sole blood supply to the greater omentum. Both are branches of the [celiac trunk](#). The right gastroepiploic is a branch of the [gastroduodenal artery](#), which is a branch of the [common hepatic artery](#), which is a branch of the celiac trunk. The left gastroepiploic artery is the largest branch of the [splenic artery](#), which is a branch of the celiac trunk.
(en.wikipedia.org/wiki)

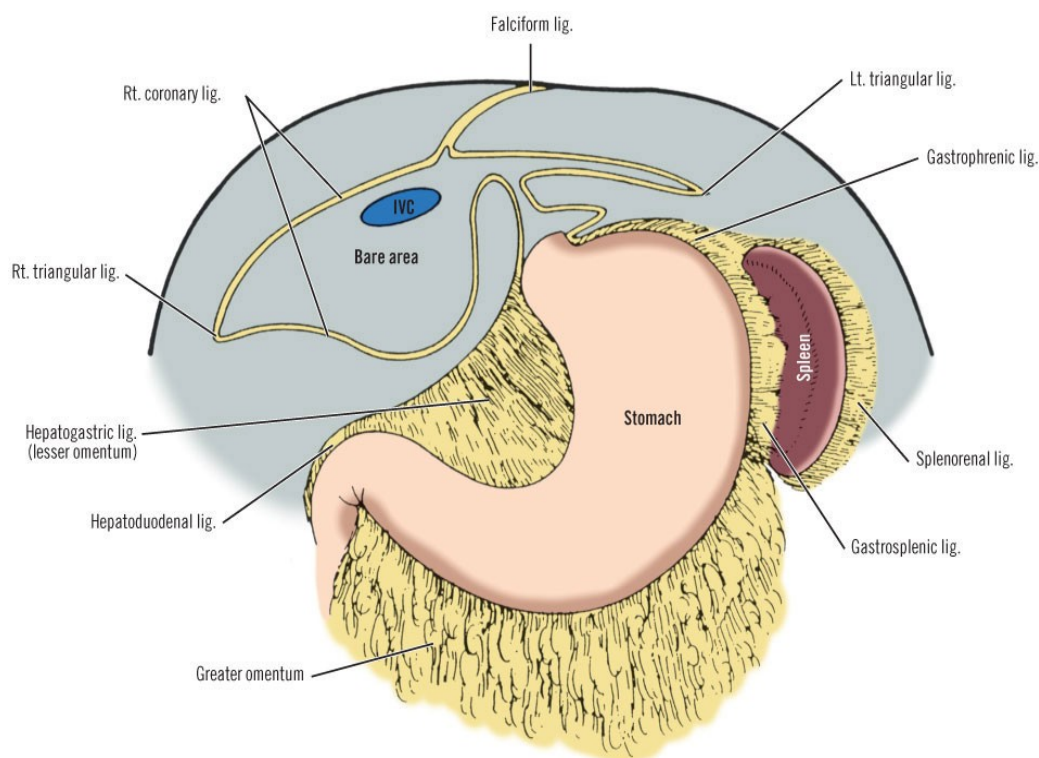


Figure 2-4
omentum

The functions of the greater omentum are:

- 1- Fat deposition.
- 2- Immune contribution.
- 3- Infection and wound isolation.

(Essential of anatomy and

physiology –Valerie c scanlon Tina sanders – F.A. davis company – 5th edition 2007 – page 82).

2-5-3 Tuberculosis of the peritoneum

Peritoneal tuberculosis (PT), a form of AT, occurs in three forms: wet type with ascites, dry type with adhesions, and fibrotic type with omental

thickening and loculated ascites. Clinically, PT is characterized by fever, abdominal pain, anorexia, weight loss, and ascites. However, none of these symptoms is specific for the disease, so it is commonly misdiagnosed, especially as carcinomatous peritonitis in the elderly. Early diagnosis of PT is of major importance in the control of the disease.

Tuberculosis peritonitis occurs at any age and in both sexes, and in some series account for 30 % of all non-pulmonary tuberculosis at for at least 20 % of all cases of ascites. In most countries about half the cases of abdominal tuberculosis will be due to peritoneal infection. In almost every case there is associated abdominal lymphadenopathy. The major clinical symptoms are abdominal distension and abdominal pain.

Vomiting and diarrhea are less common, occurring in fewer than 30% of patients.

2-5-4 Sonography of Peritoneum

Ultrasonography is the method of choice for imaging tuberculosis peritonitis. There are three imaging pattern, there may be ascites, there may be multiple caseous nodules and adhesions, or there may be a combination in which loops of bowel, omentum or mesentery have clumped together often becoming palpable and associated with ascites. When there is ascites, it can be free, localized or loculated . When free, it is either clear fluid or contains multiple thin strands, septa, or floating debris. These strands may be mobile and quite delicate or relatively thick so that adhesions occur .Fluid may be trapped between the thickened loops of bowel, producing alternating echoic and echo-free bands. On ultrasonography the density of tuberculous ascites is variable, when clear, it is a transudate in the early stage and becomes thickened later .There may be progression to an abscess, seen on ultrasound as well- defined localized fluid collections.

In the dry form of peritonitis, ultrasonography will demonstrate irregular echo-free or echo-poor, nodular or laminar thickening of the peritoneum. The nodules are poorly echogenic and occur almost anywhere on the peritoneal surface. Ultrasonography can be very useful for imaging PT. The following features may be seen, usually in combination.

(1) Intra-abdominal fluid, which may be free or loculated; and clear or complex (with debris and septae). Fluid collections in the pelvis may have thick septa and can mimic ovarian cyst.

(2) .Club sandwich or sliced bread. Sign is due to localized fluid between radially oriented bowel loops, due to local exudation from the inflamed bowel (interloop ascitis).

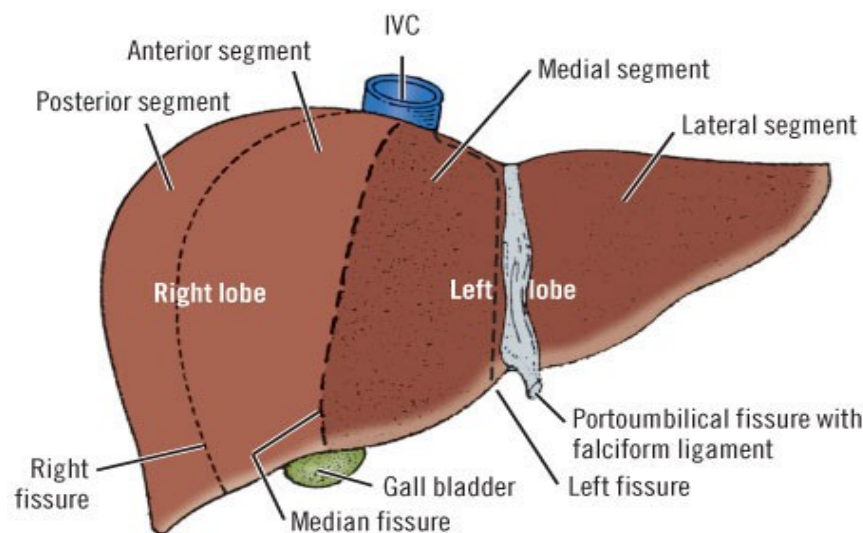
(3) Lymphadenopathy may be discrete or conglomerated (matted). The echotexture is mixed heterogeneous, in contrast to the homogeneously hypoechoic nodes of lymphoma. Small discrete anechoic areas representing zones of caseation may be seen within the nodes.

(D.H.connor I.J.dunn 2001).

2-6-1 LIVER

The liver consists of two large lobes, right and left, and fills the upper right and center of the abdominal cavity, just below the diaphragm. The structural unit of the liver is the liver lobule, a roughly hexagonal column of liver cells (hepatocytes). Between them are the portal vein. The capillaries of a lobule are sinusoids, large and very permeable vessels between the rows of liver cells. The sinusoids receive blood from both the hepatic artery and portal vein, and it is with this mixture of blood that the liver cells carry out their functions. The hepatic artery brings oxygenated blood, and the portal vein brings blood from the digestive organs and spleen. Each lobule has a central vein. The central veins of the entire lobules unite to form the hepatic veins, which take blood out of the liver to the inferior vena cava. The cells of the liver have many functions; their digestive function is the production of bile. Bile enters the small bile ducts, called bile canaliculi, on the liver cells, which unite to form larger ducts and finally merge to form the hepatic duct, which takes bile out of the liver. The hepatic duct unites with the cystic duct of the gallbladder to form the common bile duct, which takes bile to the duodenum.

Production of bile is stimulated by the hormone secretin, which is produced by the duodenum when food enters the small intestine.



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Figure 2-5 Liver

FUNCTIONS OF THE LIVER

1. Carbohydrate metabolism
2. Amino acid metabolism
3. Lipid metabolism
4. Synthesis of plasma proteins
5. Formation of bilirubin
6. Storage
7. Phagocytosis by Kupffer cells
8. Detoxification

2-6-2 GALLBLADDER

The gallbladder is a sac about 3 to 4 inches long located on the undersurface of the right lobe of the liver. Bile in the hepatic duct of the liver flows through the cystic duct into the gallbladder, which stores bile

until it is needed in the small intestine. The gallbladder also concentrates bile by absorbing water. When fatty foods enter the duodenum, the enteroendocrine cells of the duodenal mucosa secrete the hormone cholecystokinin. This hormone stimulates contraction of the smooth muscle in the wall of the gallbladder, which forces bile into the cystic duct, then into the common bile duct, and on into the duodenum. (Valerie c.scanlon Tina sanders 2007).

2-6-2 Hepatic Tuberculosis

Tuberculosis can affect the liver in many ways. Hepatic tuberculosis has been categorized as miliary, local, and biliary in the literature. The miliary form of spreading is the most common, and is thought to involve haematogenous dissemination via the hepatic artery. Some believe that hepatic tubercles are present in all cases of miliary tuberculosis. Active pulmonary tuberculosis might be present or not. Because of low oxygen tension in the liver, which is unfavorable for mycobacterial growth the local form of hepatic tuberculosis with no clinical extra-pulmonary manifestations is relatively rare. It is often found in the portal areas and may reach the liver by the portal vein. It may involve the liver diffusely or focally as space occupied nodular lesions.

Hematogenous disseminated tuberculosis can result in small tuberculous nodules in any organ. Clinically generalized hepatomegaly may be palpable when there is abdominal tuberculosis.

Liver involved in TB infection in two forms .More commonly liver is involved in TB as part of military or disseminated disease. There may not be any specific signs or symptoms related to the liver except the hepatomegaly and biopsy may show presence of granuloma. The second form, which is seen less often, is localized form of TB involving liver and

biliary ducts .Localized hepatobiliary tuberculosis occur as; (i) localized solitary or multiple nodules, tuberculoma and tuberculous hepatic abscess without bile duct obstruction; (ii) and bile ductal epithelium involvement producing inflammatory strictures with obstructive jaundice; (iii) enlarge lymph node at porta causing obstruction to the bile duct. Obstructive jaundice is more common in those who have biliary system involvement. Unlike obstructive jaundice of the biliary form, the clinical presentation of local hepatic tuberculosis has no pathognomonic feature. Abdominal pain, fever, and body weight loss are most commonly found. The clinical sign also lacks consistency. Hepatomegaly is frequently found. Biochemical studies reveal no consistent findings, although a raised ALP concentration is the most frequently noted abnormality. (YP Munjal -etal 2012).

2-6-3 Sonography of the liver tuberculosis

In addition to intra-abdominal lymphadenopathy , ultrasound may show evidence of liver involvement . Miliary lesion have been reported as causing multiple echogenic foci or even a diffusely echogenic liver. Large confluent granulomata present as echo poor or echogenic lesion 1 to 2 centimeter in diameter. Healed granulomata may calcify. In AIDS patient the picture is frequently complicated by fatty infiltration. (Barry Goldberg –Holger pettersson 1996)

Ultrasonography may reveal hypoechoic or rarely hyperechoic nodules. Most lesions of hepatic tuberculosis are small. Giant nodular lesions greater than 3 cm in diameter are distinctly rare.

There may be multiple small tuberculous granulomas in the liver, which on ultrasonography have granular echoic or hypoechoic appearance .The granulomas can become macronodular, depending on the stage of

development, some will have central more echogenic areas. If a tuberculosis abscess develops, the hypoechoic center will be surrounded by a hyperechoic rim .One or more areas of caseous necrosis may develop in some tuberculous granulomas where will be calcification causing acoustic shadowing on ultrasonography .Large tuberculosis masses are unusual . Almost always there will be marked tuberculous lymphadenopathy within the abdomen, and often in peripheral nodes also. (D.H.connor –I.J.dunn 2001).

2-7-1 PANCREAS

The pancreas is located in the upper left abdominal quadrant between the curve of the duodenum and the spleen and is about 6 inches in length. Although the pancreas is both an exocrine (digestive) gland as well as an endocrine gland The hormone-producing cells of the pancreas are called islets of Langerhans (pancreatic islets; they contain alpha cells that produce glucagon and beta cells that produce insulin. Glucagon stimulates the liver to change glycogen to glucose.

Insulin increases the transport of glucose from the blood into cells by increasing the permeability of cell membranes to glucose. The exocrine glands of the pancreas are called acini they produce enzymes that are involved in the digestion of all three types of complex food molecules.The pancreatic enzyme amylase digests starch to maltose. Lipase converts emulsified fats to fatty acids and glycerol. Trypsinogen is an inactive enzyme that is changed to active trypsin in the duodenum.

The pancreas also produces a bicarbonate juice (containing sodium bicarbonate), which is alkaline .duodenum is very acidic. (Valerie c.scanlon Tina sand 2007)..

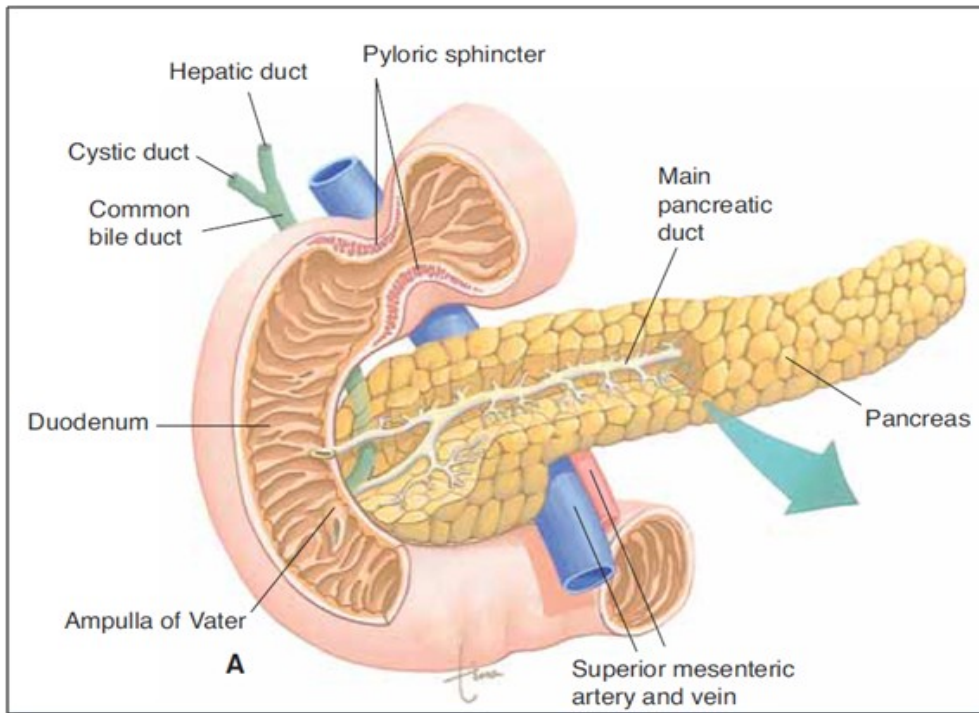


Figure 2-6 Pancreas

2-7-2 pancreatic tuberculosis

Pancreatic tuberculosis is uncommon .Pancreatic TB is most often the result of lymphatic or hematogenous dissemination or direct spread from other adjacent organs. the involvement of the pancreas by TB infection can occur as part of military or disseminated form of TB or as isolated pancreatic TB .The usual lesion in such patients is a pancreatic mass, and, therefore, symptoms depend upon the type and the site of the mass .As other sites, anorexia, malaise, low-grade fever, weight loss, night sweats are seen in majority of the patients. The specific symptoms include abdominal pain, obstructive jaundice if pancreatic head is involved. The imaging shows mass in the pancreas which is a conglomerate of lesion and peri-pancreas lymph nodes. Therefore, pancreatic TB mimics pancreatic malignancy. The other differential diagnosis includes pancreatic lymphoma, and chronic pancreatitis. (YP Munjal-et al. 2012).

2-7-3 Sonography of Pancreas tuberculosis

Sonographic appearance of pancreatic tuberculosis may be nodular form or an abscess, usually of complex echogenicity but with less surrounding pancreatitis than a pyogenic abscess. The nodular pattern is less easily recognized. Pancreatic lesions are unlikely to be seen in children .In adult they are probably going to become more common in patient with AIDS. (D.H.connor – I.J.dunn 2001)

2-8-1 SPLEEN

The spleen is located in the upper left quadrant of the abdominal cavity, just below the diaphragm, behind the stomach. The lower rib cage protects the spleen from physical trauma .In the fetus, the spleen produces red blood cells, a function assumed by the red bone marrow after birth. After birth the spleen is very much like a large lymph node, except that its functions affect the blood that flows through it rather than lymph .The functions of the spleen after birth are:

1. Contains plasma cells that produce antibodies to foreign antigens.
2. Contains fixed macrophages (RE cells) that phagocytize pathogens or other foreign material in the blood. The macrophages of the spleen also phagocytize old red blood cells and form bilirubin. By way of portal circulation, the bilirubin is sent to the liver for excretion in bile.
3. Stores platelets and destroys them when they are no longer useful.

The spleen is not considered a vital organ, because other organs compensate for its functions if the spleen must be removed. The liver and red bone marrow will remove old red blood cells and platelets from circulation .The many lymph nodes and nodules will phagocytize pathogens (as will the liver) and have lymphocytes to be activated and plasma cells to produce antibodies. Despite this redundancy, a person

without a spleen is somewhat more susceptible to certain bacterial infections such as pneumonia and meningitis. (Valerie c.scanlon Tina sanders 2007)

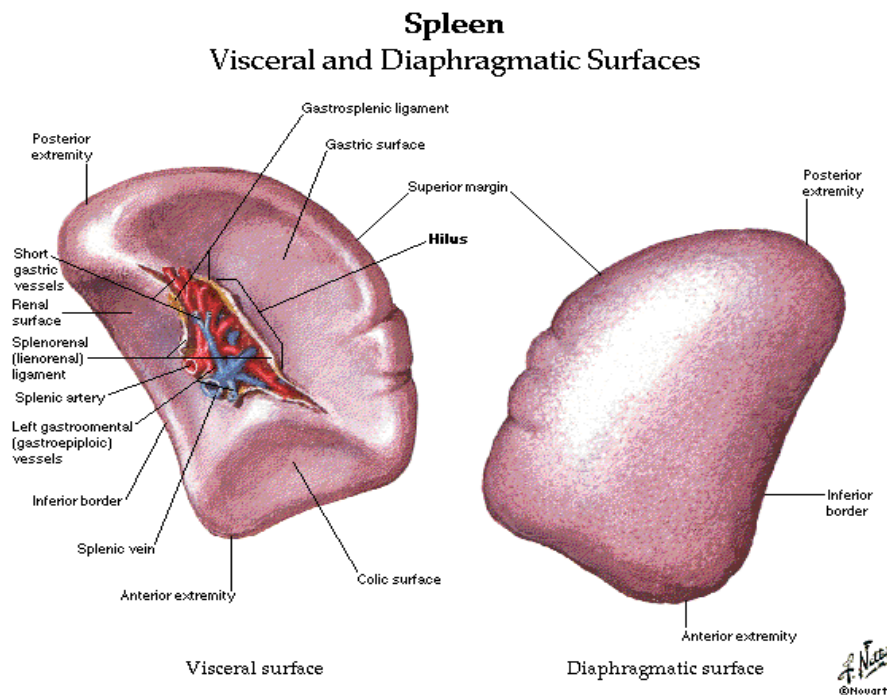


Figure 2-7 spleen

2-8-2 Splenic tuberculosis

Splenic tuberculosis is rare .spleen can be involved by TB infection in two ways mostly as a part of disseminated or miliary form of the disease and occasionally as isolated splenic TB, most of these patients are immuno-compromised; splenic TB, however, has been reported in other wise immunocompetent individuals .Most of the cases of splenic TB are recognized on imaging and sometimes as surprise on operated splenectomy specimen .In those with disseminate form of the disease ,there are enough clinical clues in other organs ; there is no specific feature in isolated splenic TB to suggest a clinical diagnosis of splenic

TB . Fever, left upper quadrant abdominal pain, weight loss ,splenomegaly and or hepatomegaly are usual features .

Splenic tuberculosis may present as splenic abscess or with hypersplenism. (API textbook of medicine.YP Munjal –etal.the association of physician of India .nine edition volume 1-2012.page 828).

In acute miliary tuberculosis, the spleen is commonly involved and the cut surface shows numerous tubercles 1 to 2 mm in diameter .The miliary tubercles are similar in size to the lymphoid follicles, but are more numerous. In the less acute form the tubercles are large and fuse together .In chronic non-miliary tuberculosis involvement of spleen is much less frequent and the number of tubercles may be small .(John w.l.fielding-Michael T.Hallisey 2005).

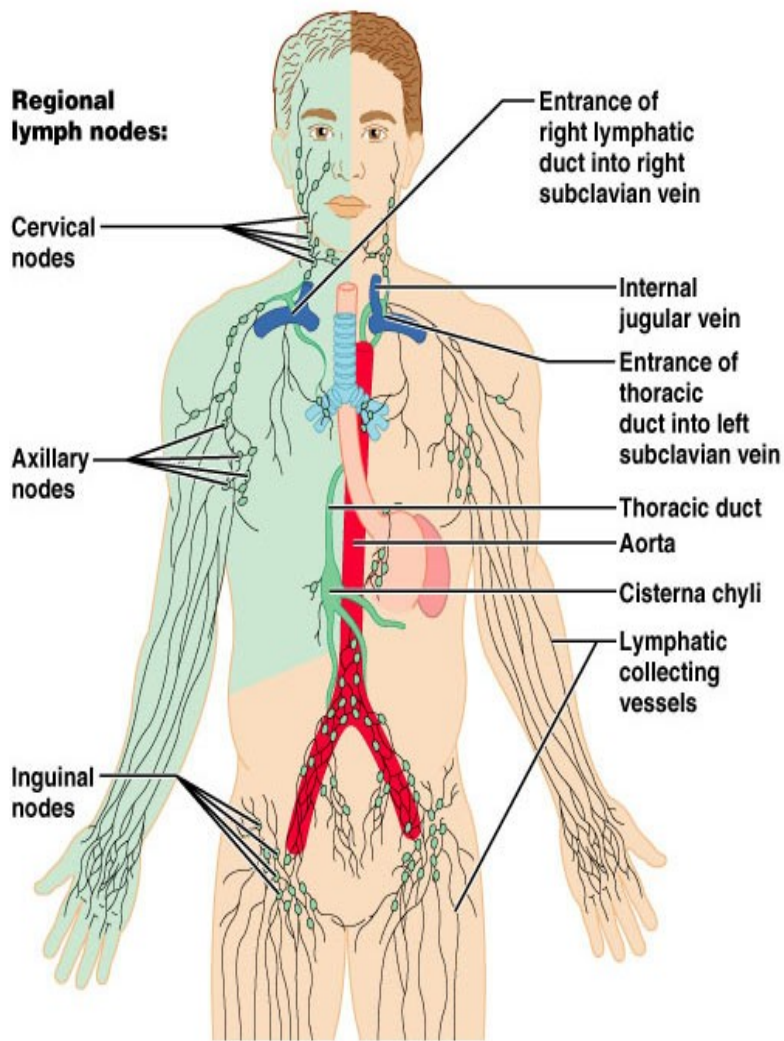
2-8-3- Sonography of Spleen tuberculosis

Splenic tuberculosis and lymphoma on ultrasonography may appear as hypodense nodular implants within the splenic parenchyma .If diffuse involvement occurs, the spleen may be enlarged .The spleen in an HIV-positive patient is highly suggestive of disseminated tuberculosis there are multiple hypoechoic lesions present .(Diane M.Kawamura1992)

Calcified granulomas are infrequent incidental findings the appearance is often striking with multiple small highly reflective foci often with posterior shadowing .Spleen may be enlarged and there may be one or multiple lesions. Splenic abscesses are uncommon .The treatment is similar as elsewhere except that splenectomy may be required in a few patients where spleen is studded with TB abscesses. (Ultrasonography .Barry Goldberg-Holger pettersson .publisher –NICER institute-Norway 1996. Page (139).

2-9-1 LYMPHATIC

The lymphatic system is responsible for returning tissue fluid to the blood and for protecting the body against foreign material. The parts of the lymphatic system are the lymph, the system of lymph vessels, and lymphatic tissue, which includes lymph nodes and nodules, the spleen, and the thymus gland. Lymph is the name for tissue fluid that enters capillaries. The system of lymph vessels begins as dead-end lymph capillaries found in most tissue spaces. Lymph capillaries unite to form larger lymph vessels, whose structure is very much like that of veins. The lymph vessels from the lower body unite in front of the lumbar vertebrae to form a vessel called the cisterna chyli, which continues upward in front of the backbone as the thoracic duct. Lymph vessels from the upper left quadrant of the body join the thoracic duct, which empties lymph into the left subclavian vein. Lymph vessels from the upper right quadrant of the body unite to form the right lymphatic duct, which empties lymph into the right subclavian vein. Lymphatic tissue consists mainly of lymphocytes in a mesh-like framework of connective tissue. Lymph nodes and nodules are masses of lymphatic tissue. Nodes and nodules differ with respect to size and location. Nodes are usually larger, 10 to 20 mm in length, and are encapsulated. Lymph nodes are found in groups along the pathways of lymph vessels, and lymph flows through these nodes on its way to the subclavian veins. There are many groups of lymph nodes along all the lymph vessels throughout the body, but three paired groups deserve mention because of their strategic locations. These are the cervical, axillaries and inguinal lymph nodes. (Valerie c .scanlon Tina 2007).



(a)

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Figure 2-8 Lymphatic system

2-9-2 Tuberculosis of abdominal lymph nodes

At least one- third of patients with tuberculous peritonitis will have lymphadenopathy, and in some countries the frequency of the lymph involvement may as high as 70%. Lymphadenopathy may occur without detectable bowel involvement. All groups of nodes within the abdomen can be infected, particularly those around the pancreas, portal region, aorta and vena cava.

2-9-3 Sonography of the lymph node

On the ultrasonography the enlarged nodes are usually hypoechoic, and some display central echogenic areas where caseation has started. Some nodes will be discrete, while others adhere together into large masses. Both CT and Ultrasonography can demonstrate nodal calcification before it can be seen on a plain radiography. It is probable that some of the abscesses within the peritoneum result from extensive caseation of lymph node. They have a very variable ultrasound and CT appearance depending on the degree of caseation, central necrosis and septation. Enlargement of the abdominal lymph nodes may cause direct pressure on various part of the gut, where may be distortion of the pyloric antrum, duodenal loop, and upper jejunum in particular. If the nodes are very large and swollen, the bowel may become adherent to them and involved in tuberculous process. Rupture of enlarged nodes into the intestine has been reported, but only happened when this adhesive process has occurred. It can present as a communicating diverticulum.

Lymphadenopathy has been used for the evaluation of lymph nodes in abdominal tuberculosis. (D.H. Connor – I.J. Dunn. S. 2001).

2-10-1 KIDNEYS

The two kidneys are located in the upper abdominal cavity on either side of the vertebral column, behind the peritoneum (retroperitoneal). The upper portions of the kidneys rest on the lower surface of the diaphragm and are enclosed and protected by the lower rib cage. The kidneys are embedded in adipose tissue that acts as a cushion and is in turn covered by a fibrous connective tissue membrane called the renal fascia, which helps hold the kidneys in place. Each kidney has an indentation called the hilum on its medial side. At the hilum, the renal artery enters the

kidney, and the renal vein and ureter emerge. The renal artery is a branch of the abdominal aorta, and the renal vein returns blood to the inferior vena cava. In a coronal or frontal section of the kidney, three areas can be distinguished. The lateral and middle areas are tissue layers, and the medial area at the hilus is a cavity. The outer tissue layer is called the renal cortex; it is made of renal corpuscles and convoluted tubules. These are parts of the nephron and are described in the next section. The inner tissue layer is the renal medulla, which is made of loops of Henle and collecting tubules (also parts of the nephron). The renal medulla consists of wedge-shaped pieces called renal pyramids. The tip of each pyramid is its apex or papilla. The third area is the renal pelvis; this is not a layer of tissues, but rather a cavity formed by the expansion of the ureter within the kidney at the hilus. Funnel shaped extensions of the renal pelvis called calyces enclose the papillae of the renal pyramids. Urine flows from the renal pyramids into the calyces, then to the renal pelvis and out into the ureter.

The nephron is the structural and functional unit of the kidney. Each kidney contains approximately 1 million nephrons. A renal corpuscle consists of a glomerulus surrounded by a Bowman's capsule. The glomerulus is a capillary network that arises from an afferent arteriole and empties into an efferent arteriole. Bowman's capsule (or glomerular capsule) is the expanded end of a renal tubule; the renal tubule continues from Bowman's capsule and consists of the following parts: proximal convoluted tubule (in the renal cortex), loop of Henle (or loop of the nephron, in the renal medulla), and distal convoluted tubule (in the renal cortex). The distal convoluted tubules from several nephrons empty into a collecting tubule. Several collecting tubules then unite to form a papillary duct that empties urine into a calyx of the renal pelvis.

(Valerie c.scanlon Tina sanders 2007).

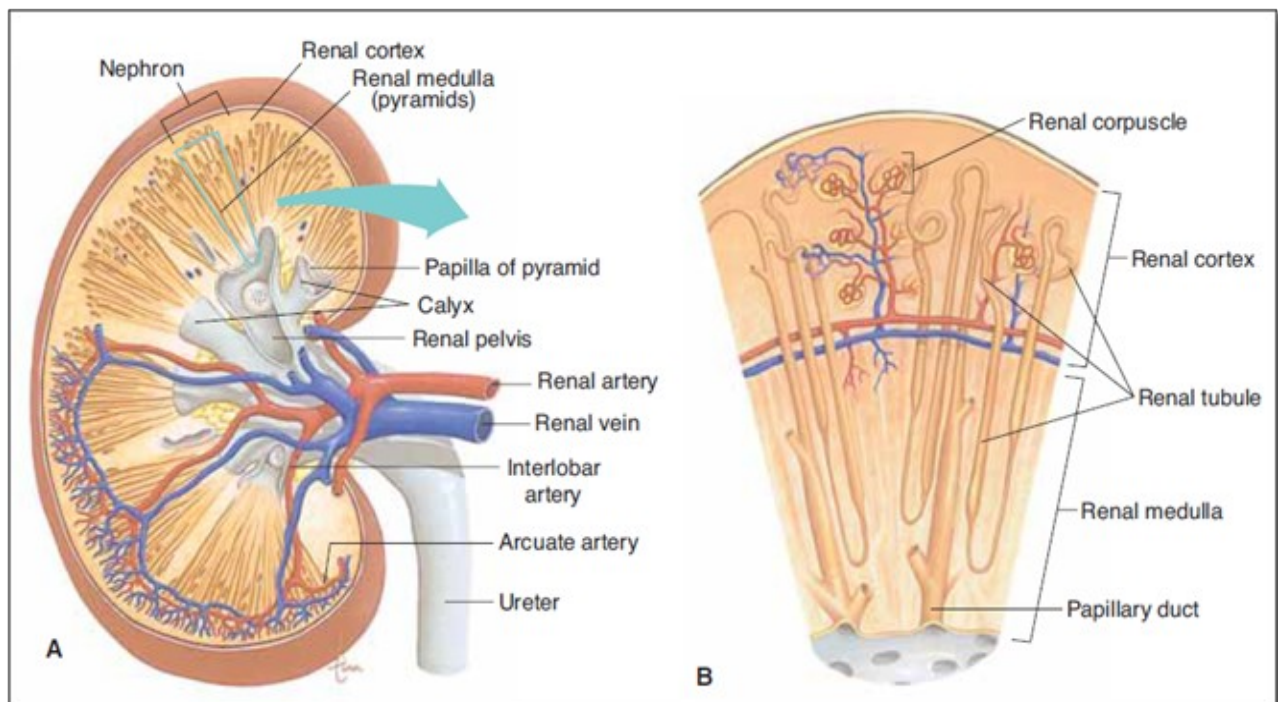


Figure 2-9 Kidney

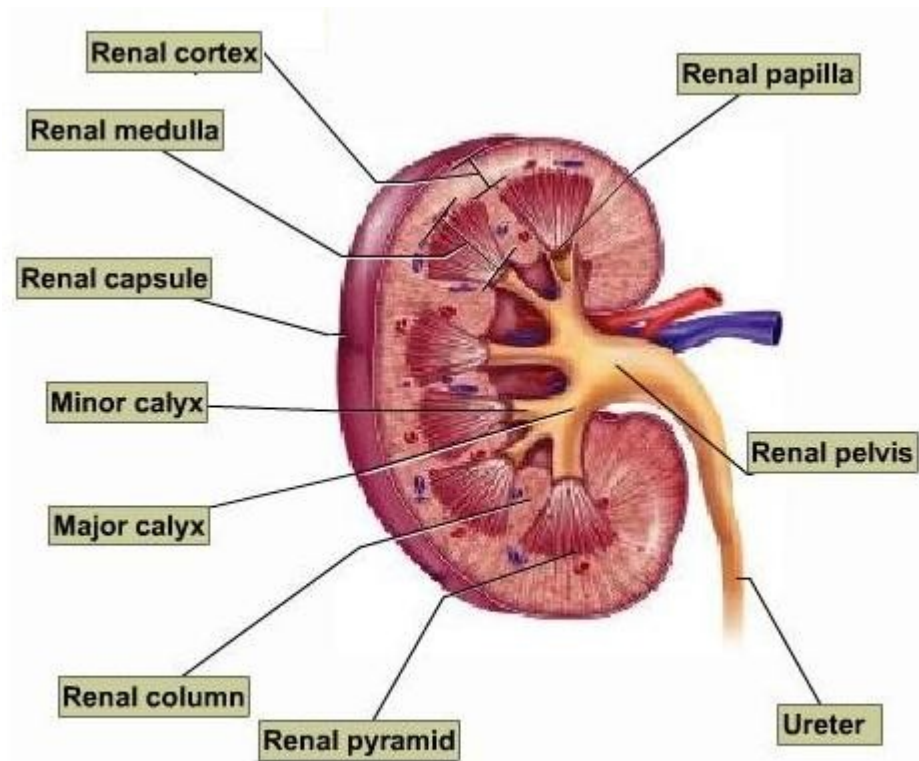


Figure 2-10 Kidney

2-10-2 Renal Tuberculosis

Renal tuberculosis accounts for approximately 15 to 20 percent of the cases of extra-pulmonary tuberculosis and is primarily a disease of young adult, with 60 percent of the patients in the age group between 20-40 years.

M.tuberculosis gains access to human body usually by inhalation, through the infection with Bovis organism may be acquired by ingestion of un-pasteurized milk. Renal tuberculosis is always preceded by primary tuberculosis focus .The primary infection frequently result in a silent bacillema .The latent focus may get aggravated years later to result in overt tuberculosis.

The kidney and possibly, the prostate are the primary sites of infection in the genitourinary system. Renal infection is by definition bilateral owing to the hematogenous spread but clinically significant involvement is usually unilateral.

Renal tuberculosis progress slowly and usually as a rule is asymptomatic till the lesion has involved the calyces or pelvis. Tuberculosis of the genital tract is uncommon before puberty. (Vimlesh. 2006).

Tuberculosis of the renal tract is usually the result of hematogenous spread of pulmonary tuberculosis and can affect the parenchyma, as well as the collecting system and ureters, in parenchymal disease the bacilli favor the high cortical blood flow, oxygen saturation and increase blood viscosity found in the efferent arterioles. If they proliferate, the infection then ruptures into the proximal tubule and the bacilli congregate in the apex of the loop of Henle in the medulla, forming medullary granulomas and abscesses which can rupture into the collecting system leading to ureteric strictures and urinary bladder fibrosis.

The early stages are not apparent on ultrasound, but parenchymal abscesses, cavities, foci of calcification, and dilated calyces become visible as the disease progresses. Increase destruction of the renal architecture is seen as the infection spreads through the kidney, segmental or generalized dilatation of the collecting system can be seen as a result of strictures and fibrosis.

Ultrasound- guided fine needle aspiration is reported to be of value in confirming the diagnosis of renal tuberculosis in patient with consistently negative urine culture for acid-fast bacilli.

Eventually the kidney becomes shrunken and fibrotic .Extensive calcification may be present; this appearance is sometimes referred to as an autonephrectomy .

A clinical feature of genitourinary tuberculosis symptoms includes;

- Dysuria.
- Renal colic.
- Macroscopic hematuria with back pain.
- Fever –anorexia-weight loss.
- Chronic renal insufficient.

(Grant M.Boxter –Paul S. sidhu 2006)

Bladder tuberculosis

When tuberculosis affects the bladder there is gradual and usually localized thickening of the bladder wall, with increasing diminution of the bladder volume. Trabeculation of the mucosa may develop. The vesicoureteric orifices are affected by this progressive fibrosis and there will then be bilateral, often asymmetrical hydroureter and hydronephrosis . Both C.T and Ultrasonography will demonstrate the irregular and bladder wall caused by tuberculous granulomas. To show this clearly, the bladder must be full .Contrast radiography or cystography is likely to demonstrate reflux up to the dilated ureters because the orifices will usually be rigid and held open.

Bladder calcification due to tuberculosis is very uncommon and when it occurs, it patchy .Spontaneous tumour calcification can occur in bladder neoplasm and need to differentiated from tuberculosis.

(D.H. connor- I.J. dunn 2001).

ultrasonographic examination, special care should be taken to differentiate between renal tuberculosis and the following renal diseases:

2-10-3 Sonography of renal tuberculosis:

- **Renal tuberculosis and the renal cyst:**

Simple renal cyst is a benign degenerative manifestation of the senescent kidney. In the ultrasound image, it shows a single or several round anechoic zones with a clear boundary. Its wall is thin and smooth with enhanced echoes at the rear wall. Behind both lateral walls, there is an inward ultrasound shadow. If the cyst develops inwards, the compression sign of the collecting system group can be seen; if the cyst develops outwards, local protrusion and malformation of the kidney will occur. Nephrectasia type renal tuberculosis results from tuberculous cavity. In the renal parenchyma, several anechoic zones in different sizes can be seen within the cysts.

There are nebulous light spot echoes. When the cold abscess is combined with calcification, within the dark zone there are some strong light masses with ultrasound shadow. In most cases, the shape of the cyst is irregular; the wall of the cyst is thickened and rough.

- **Renal tuberculosis and hydronephrosis**

Hydrops type renal tuberculosis and hydronephrosis are easily confused with each other. The differences between both lesions in ultrasound images are often subtle or minimal, and make the differentiation of both lesions difficult or impossible through ultrasound images. Sometimes ultrasonographically guided needle aspiration of the kidney is required for differentiation.

- **Renal tuberculosis and ureterostenosis:**

When the ureter is involved by the pathological developing process of renal tuberculosis, the wall of the ureter will become swollen, thickened and rough, its lumen becomes stenotic. If the patients have only mild or even no evident clinical symptoms, the examiner will often rest content with the diagnosis of ureterostenosis rather than further consider what the cause of the problem is. With the patients symptoms getting' worse and multimodality methods for diagnosis employed, the final diagnosis of renal tuberculosis was established.

- **Renal tuberculosis and non-tuberculous:**

inflammatory changes of the kidney Renal tuberculosis and non-tuberculous inflammatory changes of the kidney have manifestations in ultrasound images in some similar patients. The differentiated diagnosis between them was usually based on the clinical manifestations, laboratory examinations, IVU, RGP and CT scans.

- **Empyema type renal tuberculosis and hydronephrosis with infection:**

Both of these cases show the distended renal pelvis and calyces, within which there are anechoic zones due to poor acoustic penetration. The differentiation of both lies in that:

(a) for renal tuberculosis, the boundary between the renal pelvis and calyces is unclear. The wall of the renal pelvis is thickened and rough with enhanced echoes; within it there is a nebulous spotted hypoechoic zone. In the local area of the kidney some irregular, macula spot-like strong echoes with a weak sound shadow could be seen. For the latter,

there are only a few and scattered spotted echoes in the hydrops anechoic zone.

(b) for pyonephrosis, the laminated planes of hypoechoic light spot sediments could be seen within the anechoic zone; and for the latter, there is no such manifestation; and (c) in the former, the lesion often involves the ureter, and causes the ureter wall to thicken and become rough, with enhanced echoes; the ureter lumen develops stenosis and distension above the stenosed segment of the ureter. There are no such lesions in the latter.

- **Renal tuberculosis and kidney stones:**

In renal tuberculosis, calcification may occur in the renal cortex, the manifestation of which is strong echoic light masses in the ultrasound image. In some cases this may be accompanied with sound shadow similar to that of calculus. But in most cases the density of the strong echoes is inhomogeneous in renal tuberculosis. Part of the calcified focuses present macula patch-like appearances, irregularly distributed with an unclear boundary. The intensity of the echoes is lower than that of calculus. In renal tuberculosis, most calcified focuses are located in the renal cortex; but the kidney stone is located in the renal pelvis and calyx. Calcification is an important denotation of advanced renal tuberculosis. When associated with hydronephrosis and without renal atrophy it is easily confused with kidney stones.

- **Renal tuberculosis and the renal tumor:**

In mixed type renal tuberculosis, the kidney may present an enlarged contour with mass lump-like echoes, and can potentially be misdiagnosed as renal tumor. But the contour in most cases is irregular, and its rear echoes are lightly enhanced. For a renal tumor, it arouses a sense of

spheroid; and for a bigger tumor, its rear echoes present an attenuated appearance.

Both lesions may sometimes have similar appearances in Ultrasound images. In this series, three patients were misdiagnosed as having a renal tumor. Different clinical presentation and different laboratory examination results may help to differentiate both diseases. Final diagnosis requires bacteriological or pathological examination of the aspirated material through ultrasonographically guided needle aspiration of the kidney (R XUEFANG et al - 2007).

2-11 ASCITES

Is a gastroenterological term for an accumulation of fluid in the [Peritoneal cavity](#). The medical condition is also known as peritoneal cavity fluid, peritoneal fluid excess, hydroperitoneum or more archaically as abdominal dropsy. Mild ascites is hard to notice, but severe ascites leads to abdominal distension. Patients with ascites generally will complain of progressive abdominal heaviness and pressure as well as shortness of breath due to mechanical impingement on the diaphragm. Ascites is detected on [physical examination](#) of the abdomen by visible [bulging of the flanks](#) in the reclining patient ("flank bulging"), "[shifting dullness](#)" (difference in percussion note in the flanks that shifts when the patient is turned on the side) or in massive ascites with a "fluid thrill" or "[fluid wave](#)" (tapping or pushing on one side will generate a wave-like effect through the fluid that can be felt in the opposite side of the abdomen). Ascitic fluid can accumulate as a transudate or an exudate.

Classification:

Ascites exists in three grades:

- Grade 1: mild, only visible on ultrasound and CT
- Grade 2: detectable with flank bulging and shifting dullness
- Grade 3: directly visible, confirmed with the [fluid wave/thrill test](#)

Causes:

Causes of transudate are :

- [Cirrhosis](#) - 81% (alcoholic in 65%, viral in 10%, cryptogenic in 6%)
- [Heart failure](#)
- Hepatic venous occlusion: [Budd-Chiari syndrome](#) or [veno-occlusive disease](#)
- [Constrictive pericarditis](#)
- [Kwashiorkor](#) (childhood protein-energy malnutrition)
- [Nephrotic syndrome](#)

Causes of low exudate are :

- [Cancer](#) (primary peritoneal carcinomatosis and metastasis)
- Infection: [Tuberculosis](#) - or [Spontaneous bacterial peritonitis](#)
- [Pancreatitis](#)
- [Serositis](#)
- Hereditary [angioedema](#)

Other Rare causes:

- [Meigs.](#)
- [syndrome.](#)

- [Vasculitis](#).
- [Hypothyroidism](#).
- Renal dialysis.

(en.wikipedia.org/wiki)

2-12 PLEURAL EFFUSION

A pleural effusion is a build up of fluid between the layers of tissue that line the lungs and chest cavity.

Causes:

The body produces pleural fluid in small amounts to lubricate the surfaces of the pleura, the thin tissue that lines the chest cavity and surrounds the lungs. A pleural effusion is an abnormal, excessive collection of this fluid.

There are two different types:

- Transudative pleural effusions are caused by fluid leaking into the pleural space. This is caused by increased pressure in the blood vessels or a low blood protein count. [Congestive heart failure](#) is the most common cause.
- Exudative effusions are caused by blocked blood vessels or lymph vessels, inflammation, lung injury, and tumors.

The following tests help to confirm a diagnosis:

- [Chest CT scan](#)

- [Chest x-ray](#)
- Kidney and liver function blood tests
- [Pleural fluid analysis](#) (examining the fluid under a microscope to look for bacteria, amount of protein, and presence of cancer cells)
- [Thoracentesis](#) (a sample of fluid is removed with a needle inserted between the ribs)

Ultrasound of the chest and heart

Ultrasonography is a useful tool for physicians managing pleural diseases. It permits imaging of pleural effusion and other pleural pathology. In addition, ultrasonography has utility in the guidance of thoracentesis and various pleural interventions. Ultrasound examination of the pleura is particularly influenced by the presence of ribs and aerated lung. In bone, there is nearly complete absorption of sound waves; this yields a shadowing artifact. Ribs completely stop transmission of ultrasound and block any view of structures deep to the rib in question. Air is a powerful ultrasound reflector. As a result, most of the ultrasound wave is reflected back from the lung surface if the lung is filled with air. (Medical University of South Carolina, Charleston, SC, USA Paul H. Mayo, MDa,T, Peter Doelken, MD Clin Chest Med 27 (2006) 215 – 227.)

Ultrasound serves as a more accurate imaging tool than chest radiography for the diagnosis of pleural effusions. Ultrasound has the additional potential benefits that it can be rapidly performed, repeated over time and lacks the ionizing radiation associated with both chest radiographs and computed tomography scans. Furthermore, ultrasound can allow diagnosis of complicated pleural effusions, such as empyemas and abscesses that may be associated with a higher risk for a drainage

procedure. (International Scholarly Research Notices ISRN emergency medicine Volume 2012 . ‘<http://dx.doi.org/10.5402/2012/676524>’).

2-13 PREVIOUS STUDIES:

Uzunkoy et al. (1996) studied abdominal tuberculosis using ultrasound their study consisted of 11 patients (4 males , 7 females with mean age 39 years) diagnosed with abdominal tuberculosis in Harran university hospital between January 1996 and October 2003. Their results showed that ascites was present in all cases. tubo- ovarian mass in 5, omental thickening in 3 , enlarged lymph node (mesenteric, para-aortic) in 2 in conclusion they recommend that abdominal tuberculosis should be considered in all cases with ascites.

Tom Heller et al (2010) in South Africa study of 180 adult HIV positive patient showed sonographic signs of abdominal tuberculosis .Abdominal lymph node enlargement was the diagnostic finding in almost all cases (96.7%), hypoechoic lesions of the spleen were seen in 50% and ascites in 73.3%.

Poul Kelly (2008) in Zambia university study of 140 HIV positive patients showed that 71% had definite or probable abdominal T.B. the commonest ultrasound findings were ascites, para –aortic lymphadenopathy and hepatomegaly .

R jain et al. (1995) in India study of 56 patients with clinical features suggestive of abdominal tuberculosis using ultrasound the result showed that there are mesenteric lymph node enlargement in 19 patients and ascites in 12 patients.

Delphine (2010) in Combodia study of 200 tuberculosis patients using ultrasound the result showed that there are 29 patients have abnormal abdominal ultrasound , 19 of them with enlarged lymph node .

Malik, (2003) in India study of 66 abdominal tuberculosis patients using ultrasound (Study was done to emphasize the importance of ultrasound and ultrasound-guided fine needle aspiration biopsy in the diagnosis of abdominal tuberculosis) the result showed that Peritoneum tuberculosis was the most common form, of the wet ascetic type, ascites was clear in 19 patients and complex in 17. Tuberculosis lymphadenopathy was seen in 37 patients. There was a predilection of periportal , per pancreatic and mesenteric locations compared with the degree of retroperitoneal involvement . Calcification and hetrogenous echotexure were seen in 7 cases. Intestinal disease was seen in 14 patients. Hepatic or splenic involvement was seen as diffuse organomegaly: less commonly, focal lesions were seen.

Xuefang Rui(2005) To explore the value of B-mode ultra sonography in the diagnosis of renal tuberculosis study of 258 cases of renal tuberculosis .136 (52.7%) males and 122 females (47.3%).The ages ranged from 17 to 73 years old. Left kidney tuberculosis occurred in 120 cases, accounting for 46.5%; right kidney tuberculosis occurred in 120 cases, accounting for 46.5%; right kidney tuberculosis occurred in 118 cases (45.7%); both-sided kidney tuberculosis occurred in 20 cases. In this series of 258 cases, 152 cases were correctly diagnosed as renal tuberculosis by B-mode ultra-sonography. The coincidence rate was The remaining 106 cases at rest were separately misdiagnosed by B-mode Ultra -sonography as hydronephrosis, 27 cases; hydronephrosis with calculi, 18 cases; renal cysts, 19 cases; ureterostenosis at the upper

segment, 7 cases; kidney tumor, 3 cases; ureter tumor at the lower segment, 5 cases; non-tuberculosis inflammatory change of the kidney cases; and duplicated kidney, 1 case. Their final diagnosis was 26 obtained by multi-modality method.

S Rai (2013) study of 36 patients with documented diagnosis of abdominal TB. Abdominal X-ray signs were detected in only 5 patients (none specific for TB). An ultrasound scan of the abdomen was performed in 28 patients, with findings suggestive of TB in 9 of these patients (fibrinous strands in ascitic fluid, localized ascites, calcified lymph nodes). 11 others were found to have ascites but no specific features. A cconsistent with abdominal TB in 6 (adenopathy predominantly in the retroperitoneal and mesenteric compartments, splenomegaly, ascites, ileocaecal mass.)

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CHAPTER THREE

MATERIALS AND METHODS

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MATERIALS AND METHODS

3-1 Area of study and duration:

This study was conducted in Omdurman teaching hospital for chest & medicine (ABO ANGA), in the period From the 30 of April 2014 to 14 of July 2014.

3-2 Population of study:

The data of this study was collected from patient suffering from abdominal tuberculosis and free from other abdominal disease that does not related to TB.

3-3 Sample size and type

The data of this study collected from 50 patients chosen continently.

3-4 Design of the study

This is a descriptive study of a cross-sectional type.

3-5 Machine used:

An ultrasound machine (FUKUDA Denshi SF sonic 4200 R) is used .The transducer is 3.5MHz convex type, and ultrasound gel is applied to the transducer to prevent any attenuation or artifact.

3-6 Ultrasound techniques:

The following techniques were applied for visualization of abdominal organs:

Supine: The patient in supine position. The liver scanned in transverse, longitudinal and oblique planes including scans through intercostal and subcostal spaces multiple transverse and longitudinal scans are made with rocking the transducer. The pancreas scanned in longitudinal and transverse planes at the level of xiphoid process.



Figure 3-1 Liver -intercostal



Figure 3-2 Liver- transverse
(subcostal)



Figure 3-3 Pancreas (transverse)
(Supine)



Figure 3-4 pancreas - sagittal
(Supine)

Prone: The patient lying in prone position. This position is done when scanning the kidney.

Lt Lateral oblique: when scanning the spleen and the left kidney, the coupling agent applied over the left lower chest, multiple scans are necessary.

Rt lateral oblique: When scanning the right kidney the transducer over the right upper abdomen, the right kidney also seen in the supine position using the liver as an acoustic window.



Figure 3-5 Spleen –coronal oblique oblique

(Rt lateral decubitus)



Figure 3-6 rt kidney –coronal oblique

lt lateral decubitus)



Figure 3-7 Lt Kidney coronal oblique coronal

(Rt lateral decubitus)



Figure 3-8 rt Kidney - coronal

(Supine)

Lt Decubitus: In this position the patient is lying on his lateral left side to view the right kidney. The transducer placed in coronal oblique.

Rt decubitus: The patient lying on his right lateral side, this is good position for scanning the spleen.

3-7 Data collection:

A data collection sheet is used to collect the data and to number the patients.

3-8 Data analysis:

Finally these data were tabulated, described, represented and analyzed using SPSS.



Figure 3-9
Fukuda Denshi

CHAPTER FOUR

RESULTS

CHAPTER FOUR

RESULTS

Table 4.1 show the mean and standard deviation of the measured study variables

variable	Mean±SD
age	34.9±1 14.2
height	168.4±8.6
weight	48.3± 8.7
BMI	17.1± 2.9
T.B duration	18.9±19.9
liver size	13.9±0.8
spleen size	11.4±0.7
kidney size	10.2±0.6

Table 4-2

:Age groups frequency distribution

Frequency	Age
28	32 -17
11	48 -33
11	67 -49
50	Total

.(Total sample size is 50 .56% (28 of 50)under group (17-32
(of 50) under group (33-48). 22% (11 of 50) under group (49-67 11) 22%

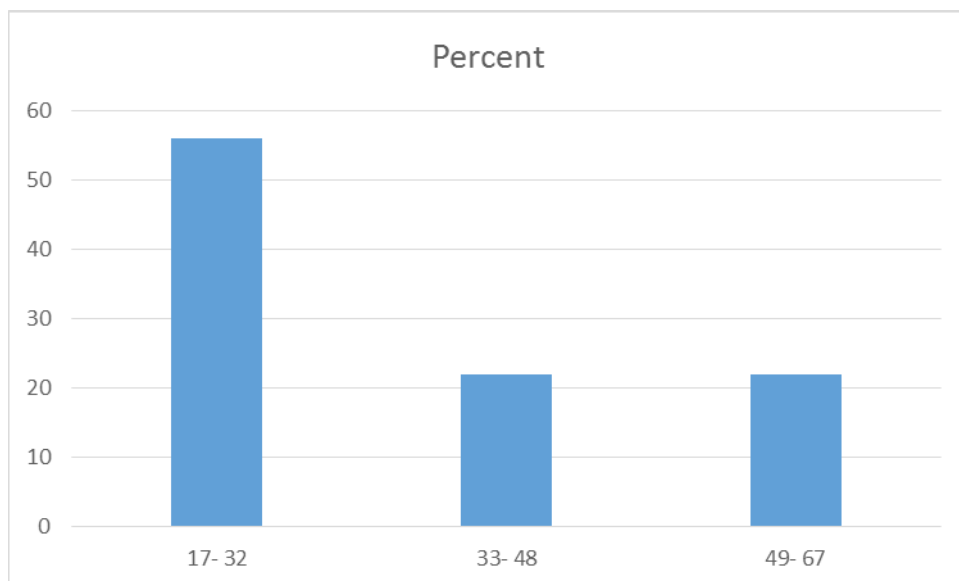


Figure 4-1

A bar graph show age group percent

Table 4.3

Residence frequency distribution

Residence	Frequency
Khartoum	40
west	4
Middle	3
east	3
Total	50

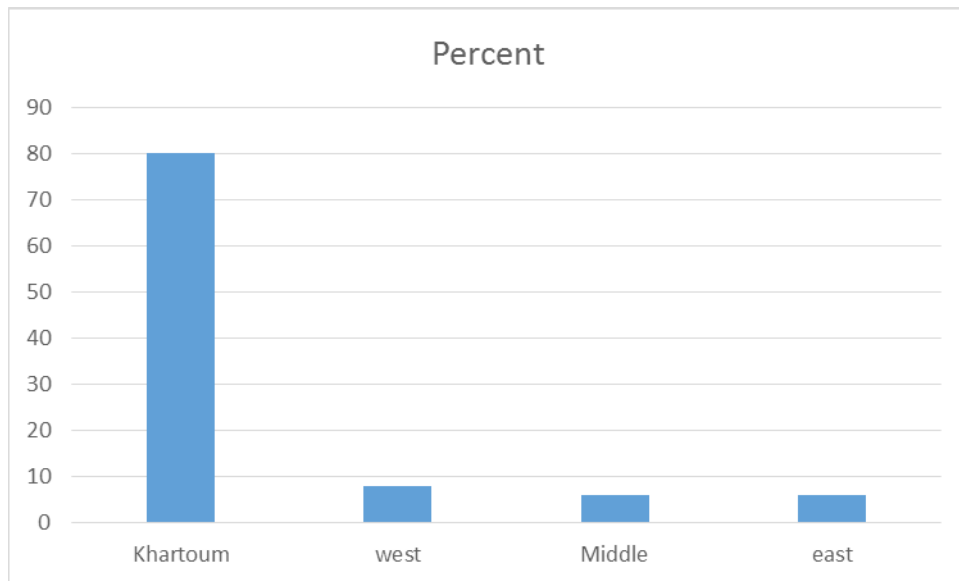


Figure 4-2

A bar show residence percent

Table 4.4

Gender frequency distribution

gender	Frequency
male	34
female	16
Total	50

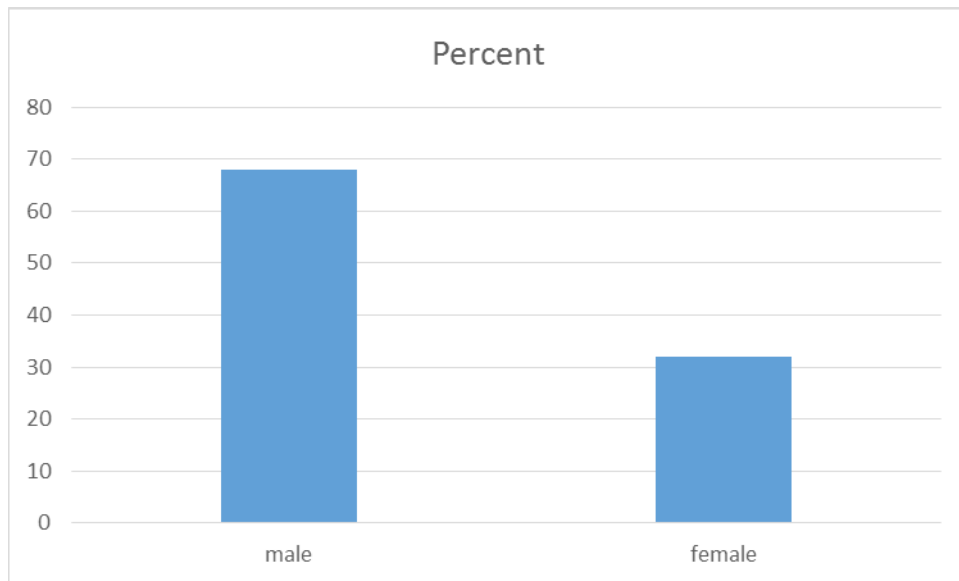


Figure 4-3

A bar show gender percent

Table 4.5

Duration frequency distribution

Duration in months	Frequency
2-35	41
36-65	6
66-96	3

Total	50

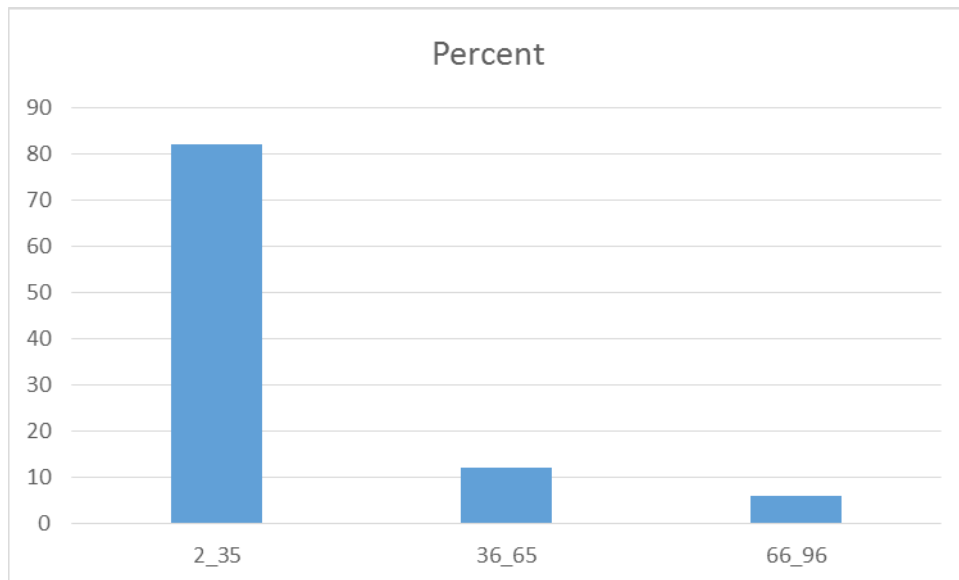


Figure 4-4

A bar show duration percent

Table 4.6

Liver size frequency distribution

liver size	Frequency
Normal	42
Abnormal	8
Total	50

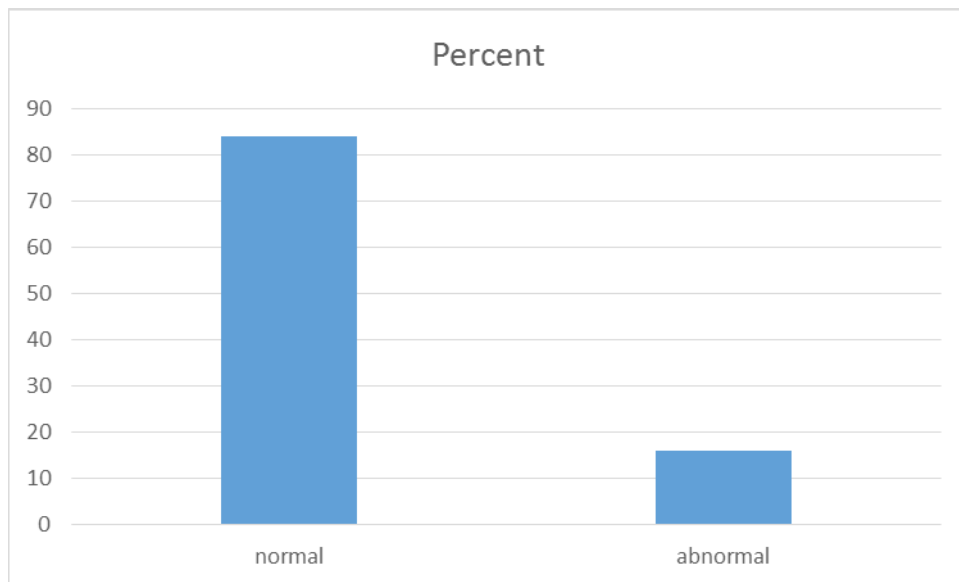


Figure 4-5

A bar show liver size percent

Table 4.7

Spleen size frequency distribution

spleen SIZE	Frequency
Normal	48

Abnormal	2
Total	50

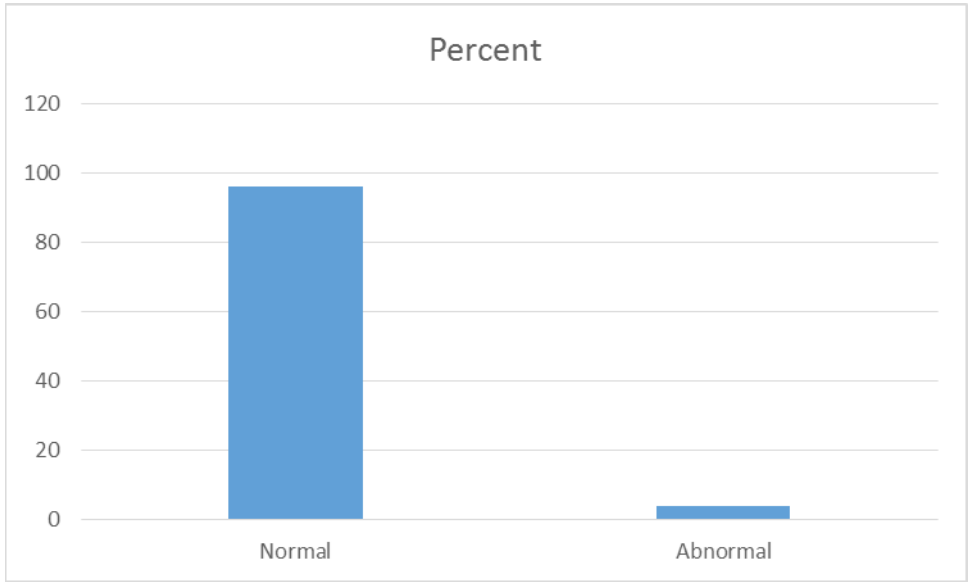


Figure 4-6
A bar show spleen size percent

Table 4.8

Pleural effusion frequency distribution

pleural effusion	Frequency	Percent
0	48	96.0

1	1	2.0
2	1	2.0
Total	50	100.0

normal 1=mild 2= moderate =0

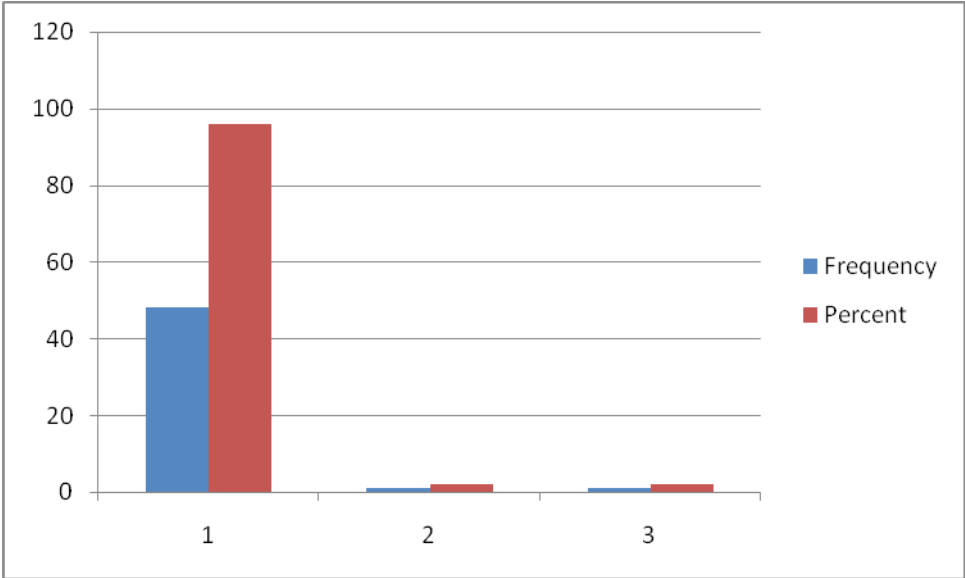
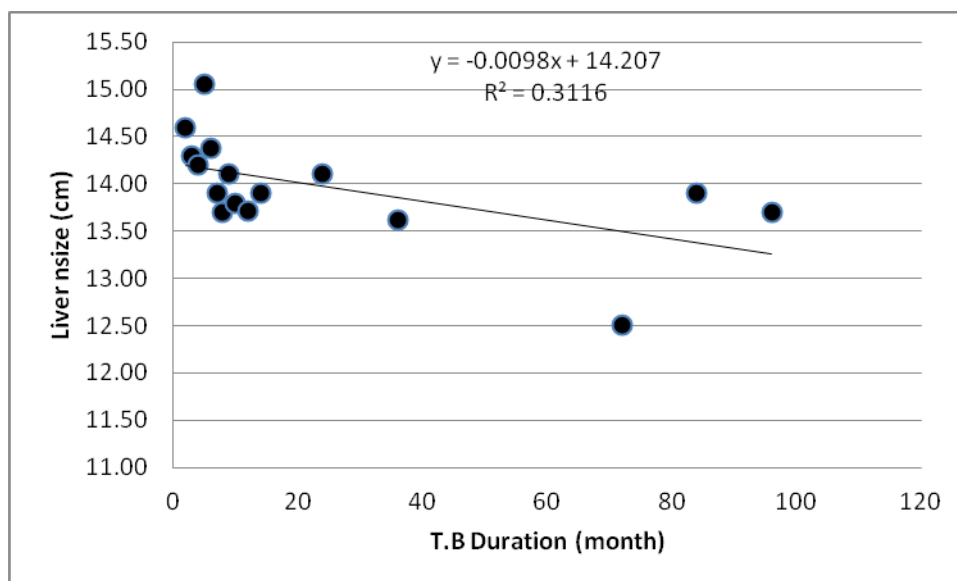


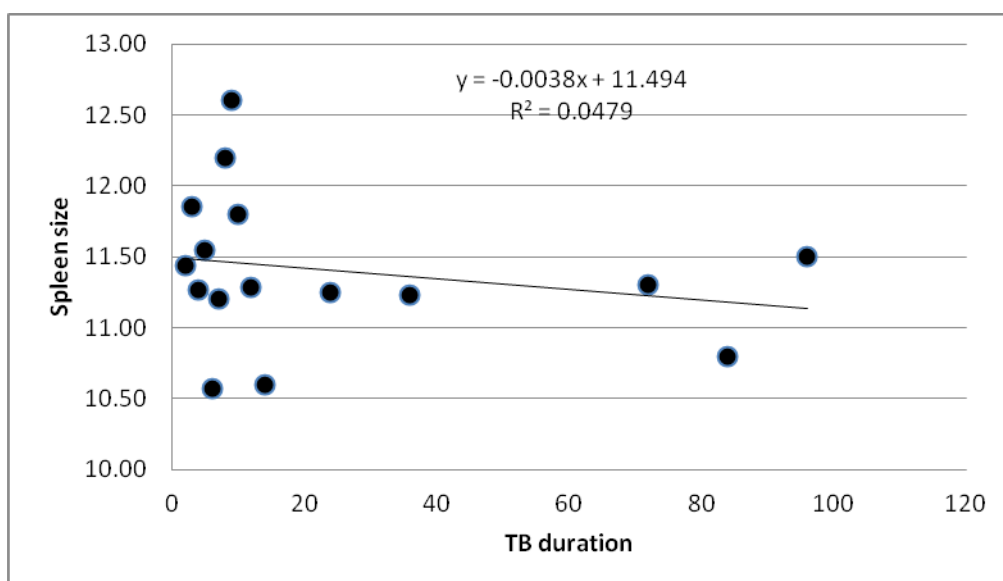
Figure 4-7

A bar show pleural effusion frequency and percent

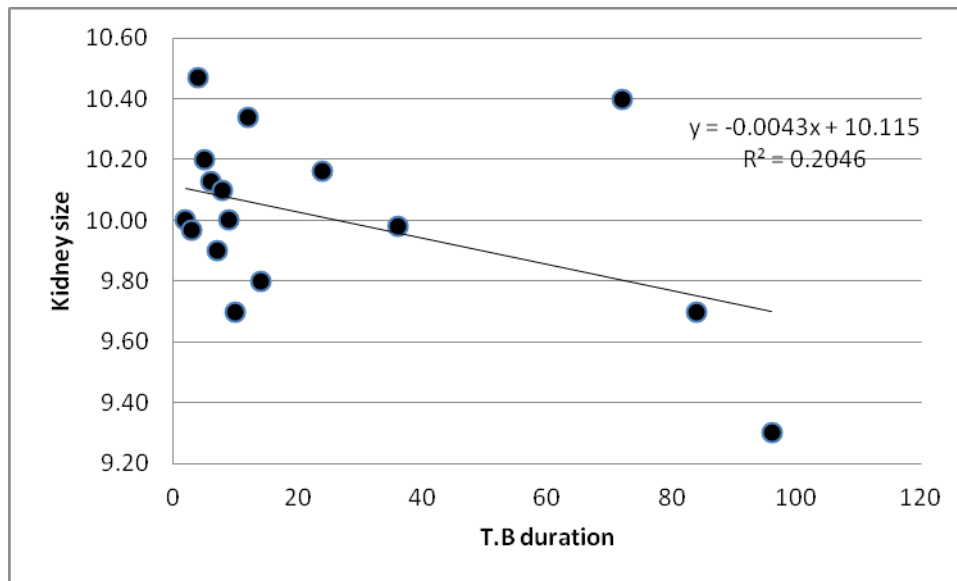
normal 2= mild 3=moderate=1



(A)



(B)



(C)

Figure 4-8

scatter plot show an inverse linear relationship of T.B duration and (A) liver size, (B) spleen size and (c) kidney size; which means the sizes of these organs decreases as the duration of T.B duration increases here .sparse correlation were noted

Table 4-9 analysis of variance table for T.B duration effects on the size of liver spleen and kidney

		Sum of Squares	df	Mean Square	F	.Sig
liver size	Between Groups	8.258	15	0.551	0.713	0.755
	Within Groups	26.270	34	0.773		
	Total	34.528	49			
spleen size	Between Groups	8.155	15	0.544	0.965	0.509
	Within Groups	19.149	34	0.563		
	Total	27.305	49			
kidney size	Between Groups	2.882	15	0.192	0.483	0.933
	Within Groups	13.516	34	0.398		
	Total	16.398	49			

Note: the result showed that there are no significant effects of T.B .duration on the organs sizes

Note: also the body characteristics ‘age, weight, height and BMI showed insignificant correlation with the liver, spleen and kidney sizes. Also in case of gender there is no significant difference between male and .female; therefore they are treated as one sample

Table 4-10 the average size of liver, kidney and spleen in respect to T.B duration

T.B duration	liver size	kidney size	spleen size
2	14.60	10.00	11.43
3	14.30	9.97	11.85
4	14.20	10.47	11.27
5	15.05	10.20	11.55
6	14.37	10.13	10.57
7	13.90	9.90	11.20
8	13.70	10.10	12.20
9	14.10	10.00	12.60
10	13.80	9.70	11.80
12	13.71	10.34	11.28
14	13.90	9.80	10.60
24	14.11	10.16	11.25
36	13.62	9.98	11.23
72	12.50	10.40	11.30
84	13.90	9.70	10.80
96	13.70	9.30	11.50

CHAPTER FIVE

Discussion, conclusion and recommendation

CHAPTER FIVE

5-1 Discussion

This study has been done in Abo Anga teaching hospital for Chest & Medicine –Omdurman in the period between 30 of April 2014 to 14 of July 2014, for 50 positive tuberculosis patients, age between 17-67 years old. 68% male (34), 32% female (16), was categorized into three age groups, 17-32 years (56%, '28'), 33-48 (22% '11') and 49 -67 (22% '11').

The result of this study showed that 4% of the patients had splenomegaly but there were no changes in the echotexture of the spleen. While concerning the liver 16% had hepatomegaly but there were no changes in echotexture of the liver, while 4% had pleural effusion.

The above result showed that the echotecture of the liver, spleen and the kidney they don't affected by the TB in respect to normal patient while increase in size were not in a few cases , therefore they will not be considered as cardinal signs of TB impact but they are expected to happen.

In respect to the scatter plot shown in Figure 4-8 which represent the relationship between the duration of tuberculosis as independent factor and the size of the liver (A), size of the spleen (B) and size of the kidney (C) as dependent factors; the general trend of the line graphs showed that in general the sizes of these organs decreases as long as the period of disease increase; it depict an inverse linear relationship. The sizes of these organs decreased by a coefficient equal to 0.01cm/month for the liver and by 0.004 cm/month for the spleen and the kidney; although the reductions were minimal but if the disease were contracted for long period a serious complications might arises.

Other abdominal organs such as pancreas and intestine there were no changes in size and echotecture also para-aortic lymph node there is no enlargement observed or any ascites detected.

5-2 conclusion

This study has been done in Abo Anga teaching hospital for chest & medicine for 50 tuberculosis patient, there age between 17 – 67 years (34 male, 16 female).

The goal of the study is to know is there any effects of tuberculosis on the abdominal organs and to assist the physician in diagnosis if there is any complications of tuberculosis in abdominal organs using ultrasound.

The result conclude that there are some patients have splenomegaly, hepatomegaly and pleural effusion, but no increase in size in others abdominal organs (pancreas, kidney), no any changes in echotexture of all abdominal organs (liver , spleen , kidney , pancreas, intestine) also no ascites or para aortic lymph node enlargement .

From the results we also conclude that when the duration of the T.B increases the size of the liver, spleen, kidney decrease.

Finally we found there were no significant relationship between the pulmonary T.B and organomegaly so no direct significant effects of pulmonary T.B in the abdominal organs but as the result of the general emaciation visceral organs start to decrease in size.

5-3 Recommendations:

At the end of the study, I recommended the following:

- All pulmonary tuberculosis patients should do abdominal u/s to exclude abdominal T.B.
- Increase the number of patients in the following studies to clarify the complication
- Ultrasound should be done before the patient take any treatment, so any effects can appear.
- Any area contaminated with TB should be checked for abdominal TB.
- Female tuberculosis showed be checked for infertility.
- Any tuberculosis patient with ascities showed be checked for aortic lymph node.

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5-5 APPENDIX

DATA SHEET

.....:Patient NO

.....:Residence

.....: Age

.....: Sex

.....:Tall

.....:Weight

.....:T.B Duration

-:Liver

☐ Normal ☐ Abnormal

-: Size ☐ Normal ☐ Increase ☐ Decrease

-:Echotexture ☐ Normal ☐ Hyper ☐ Hypo

-:Mass ☐ No ☐ Solitary ☐ Multiple

☐ Cystic ☐ Solid ☐ Complex

-:**Spleen**

☐ Normal ☐ Abnormal

-: Size ☐ Normal ☐ Increase ☐ Decrease
 -:Echotexture ☐ Normal ☐ Hyper ☐ Hypo
 -:Mass ☐ No ☐ Solitary ☐ Multiple
 ☐ Cystic ☐ Solid ☐ Complex

:Kidney

☐ Normal ☐ Abnormal

-: Size ☐ Normal ☐ Increase ☐ Decrease
 -:Echotexture ☐ Normal ☐ Hyper ☐ Hypo
 -:Mass ☐ No ☐ Solitary ☐ Multiple
 ☐ Cystic ☐ Solid ☐ Complex

:Pancreas

☐ Normal ☐ Abnormal

-: Size ☐ Normal ☐ Increase ☐ Decrease

-: Echotexture ☐ Normal ☐ Hyper ☐ Hypo

-: Mass ☐ No ☐ Solitary ☐ Multiple

☐ Cystic ☐ Solid ☐ Complex

-:Abdominal Mass

☐ Size ☐ Site ☐ Texture

-:Lymph Node

☐ Normal ☐ Abnormal

-:Omentum Thickening

☐ Normal ☐ Thick

-:Ascites

☐ NO ☐ Yes

☐ Mild ☐ Moderate ☐ Sever

-:Pleural Effusion

☐ NO ☐ Yes

☐ Mild ☐ Moderate ☐ Sever



Image 1

(years male tuberculosis patient with hepatomegaly (15.4cm 35



Image 2

(years male tuberculosis patient with splenomegaly (13.7 cm 29

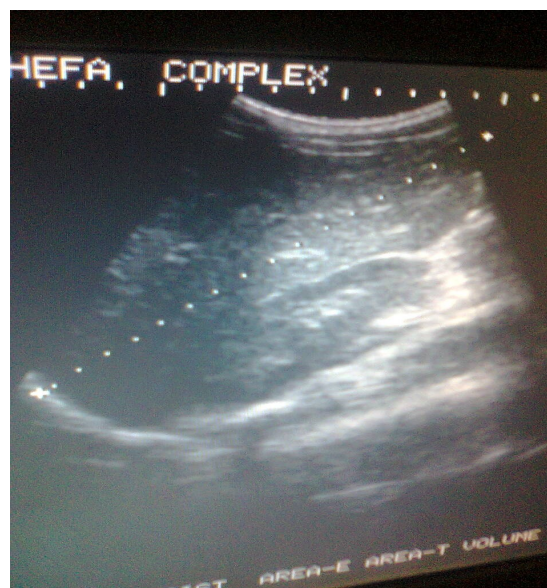


Image 3

(year female tuberculosis patient with hepatomegaly (15.3cm 22

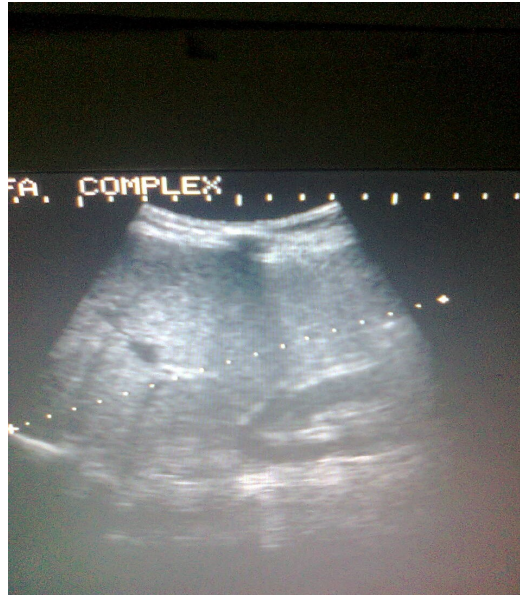


Image 4

(years male tuberculosis patient with hepatomegaly (15.cm 28



Image 5

(23years female tuberculosis patient (normal spleen 11.3



Image 6

(37years male tuberculosis patient with splenomegaly (14.1cm



Image 7

(35years female tuberculosis patient (normal spleen 12.5cm



Image 8

(years male tuberculosis patient (normal spleen 11.8 29



Image 9

25 years male tuberculosis patient (normal liver 12.2cm)



Image 10

27 years male tuberculosis patient (normal kidney 9.3cm)



Image 11

(years male tuberculosis patient (normal rt kidney 9.7cm 42



Image 12

(years female tuberculosis patient (normal spleen 11.4cm 31



Image 13

(years male tuberculosis patient(normal liver 13.3cm 27



Image 14

(years male tuberculosis patient (normal rt kidney 9.6cm 21