SUDAN UNIVERSITY OF SCIENCE AND TECHNOLOGY

COLLEGE OF GRADUATE STUDIES

Study of the Liver in Marasmus and Kwashior by Using Ultralsound

دراسة الكبد بواسطة الموجات فوق الصوتية لمرضى المرازمس والكواش كور

A-thesis submitted for partial fulfillment for the Requirement of MSc degree in Medical Diagnostic Ultrasound

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بسم الله الرحمن الرحيم

الآية الكريمة

قال تعالى : (وَمِنَ النَّاسِ وَالدَّوَابِّ وَالْأَنْعَامِ مُخْتَلِفٌ أَلْوَانُهُ كَذَلِكَ إِنَّمَا يَخْشَى اللَّهَ مِنْ عِبَادِهِ الْعُلَمَاءُ إِنَّ اللَّهَ عَزِيزٌ غَفُورٌ)

صدق الله العظيم

سورة فاطر [الأية :28]

Dedication

This project is dedicated:

To the Soul of My Mother

To my father

To my sisters

To Mr: Loay Salah

To my friends

ACKNOWLEDGEMENT

It is with immense gratitute that I acknowledge that support and help of my supervisor Dr. Caroline Edward Ayad.

I consider it an honor to work with her, with her encouragement guidance and support from the initial to the final level enabled me to develop an understanding of the subject . Her belief that it was, indeed possible to finish, kept me going .

My deepest gratitude to Dr. Ashraf and Dr. Hashim Hamid Elameen for Their help , patience and understanding .

This work would not have been possible unless the invaluable assistance of patient's and our colleagues in Bashier and Mohmed Elameen Hospital for pediatric so special thanks to all the staff .

I cannot Find wards to express my gratitute to my friends specially Mohmmed Mustafa, Nahied Elbkharie and Shaza Mhgoub my heart fully Thank's to them for share the credit of my work with them and Iam indebted to my many colleagues whom supported me.

Fainally I offer my regards and blessings to all of those whom supported me in any respect during the completion of the project.

Abstract

This Study Show Ultrasound findings and evaluation of liver in kwashiorkor and marasmus patients.

It was conducted at Radiology department in Ultrasound unit of Bashaier and Mohammed- Elameen hospitals for pediatrics and Lab unit of Khartoum Teaching hospital during the period from (April to September2014)

The study include a sample of 40patients which was divided in two groups- group one 27 marasmic patient's (6 female – 21 male)–group two 13 kwashiorkor patients (6 female – 7 male).

The most affected age group was from the ages between (7-61month) and their weights between (3-11 kg) both males and females.

The mild fatty liver was found in 18patients as 13 marasmic patients and 5 kowashikor.

The mid Fatty liver was found in 8pateints as 5marasmic patients and 3 of kowashikor patients and normal liver texture in 18 cases.

Liver function test results were found the presence of protein liver form (2.5 – 4.6) (1 marsmus - 11kowashikor) , (5.6 – 7.7) (15marasmas – 2 Kowashikor) and less than 7.7 (11 marasmus) .

It was found that ultrasound imaging and lab test are complementary modalities in the diagnosis of malnutrition cases for it's high ability in detecting the changes in liver texture as well as the values of liver protein.

ملخص البحث

في هذه الدراسة تم تقويم الكبد لدى الأطفال المصابين بسوء التغذية (المرازمس – الكواش – كور) .باستخدام الموجات فوق الصوتية .

أجريت هذه الدراسة في قسم الاشعة وحده الموجات فوق الصوتية بمستشفى بشائر ومستشفى محمد الأمين للأطفال ووحدة المعامل بمستشفى الخرطوم التعليمي في الفترة من (ابريل الي سبتمبر2014).

أخذت عينة عشوائية تتكون من 40 مريض قسمت الى مجموعتين الأولى تتكون من المرضي المصابين بالمرازمس وهم 27 مريض67.5% والمجموعة الثاني المصابين بالكواش-كور 32.5% وكل من المجموعتين يتراوح أعمارهم بين (7-61شهر) وأوزانهم تتراوح بين (1- 11كجم) في كل من الجنسين (ذكور – إناث) .

حيث أوضحت الدراسة أن مظهر الكبد الدهني يظهر18 حالة (المرازمس 13حاله والكواش كور 5 حالات) الكبد الأقل دهنية 8 (المرازمس 5 حالات والكواش كور3) والمظهر الطبيعي للكبد 18 حالة.

تم الحصول على هذه النتائج من خلال إجراء فحص معملي لقياس نسبة البروتين في الدم من خلال فحص وظائف الكبد بالإضافة لإجراء الموجات فوق الصوتية لقياس التغيرات في مظهر الكبد بإيضاحها وإعداد تقارير طبية لها كانت نتائج المعمل نسبة البروتين في الكبد ما بين (2.5 – 4.6) (1 مرازمس - 11 كواش كور) ، (5.6 – 7.7) (15 مرازمس 2 كواش كور) وأقل من 7.7 (11 مرازمس) .

وجد أن الموجات فوق الصوتية بالإضافة الى الفحص المعملي هما أفضل تقنيتان لدراسة التغيرات في الكبد لمرضي سوء التغذية.

Abbreviations

Item	Name
A	abdominal distention
CO	Cough
C.T	Computed tomography
Cm	centimeter
D	Diarrhea
DS	down syndrome
F	fever
Fa	father
G/dl	gram per deciliter
I	Infection
KG	kilo gram
Kwas	kwashiorkor
M	Male
Mo	month
Mot	mother
M.R.I	Magnetic Resonances Imaging
Maras	Marasmus
N.M	Nuclear Medicine
Nor	normal
P.E.M	protein energy malnutrition
S	Swelling
V	vomiting

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Chapter One

Chapter one

1-1 Introduction

Marasmus is a form of sever protein energy malnutrition characterized by energy deficiency, while kwashiorkor is an other form of sever protein energy malnutrition characterized by protein deficiency and marasmic kwashiorkor is condition in between them(Badaloo AV, Forrester T, Reid M, Jahoor F, June2006).

A liver ultrasound is a medical procedure in which sound waves are transmitted to form images that are projected in a video monitor ,allowing operator to view the inside of the body and see the pictures of the liver. The liver is responsible for filtering out waste and toxins as well as absorbing the nutrients in food there for a condition that affect its proper functioning may become fatal without treatment . ultrasound may be done in order to check for abnormalities ,such as masses or discoloration, that may indicate a liver condition, including cirrhosis or cancer. The procedure may also allow a doctor to find out the severity of condition and determine the best treatment course (Boelche, 2013).

Since it is thought to be relatively quick and non-invasive compared to other tests, u/s is often the first procedure used to diagnose a liver condition .There for this procedure used in this study .will find out and compare between marasmus and kwashiorkor liver u/s findings and related other important organs such as hepatobiliary system and spleen.

1-2 Problem of study:

Kwashiorkor and marasmus are common disease that affected children , It may cause different problems in body system including heart , spleen , kidneys and liver.

Ultra sound is non invasive tool to investigate the internal organs anatomy and pathology.

No study was done for Sudanese to investigate the effect of these disease on the liver on score high ratio of malnutrition cases .

Is malnutrition affects the liver {physiologically -pathologically} Variables:

Size of liver, echo texture, surface contour and billiary system

To enumerate the effect of malnutrition in relation to the factor causes {poverty, death of one parent or both, maternal disease, HTN, DM, anemia and infection.

Is affectance of malnutrition can affect on liver toxicity, drugs and anesthesia on operation

1-3Objectives

1-3-1General Objectives:

To study the sonographic appearance of the liver in malnutrition children and relate this with the causative factors.

1-3-2Specific Objectives:

1-To measure the liver length in different types of malnutrition.
2-To evaluate the echotexuer of the liver in patients with marasmus and kwashiorkor.

3-To correlated the ultra sound findings with the labarotary test (protein in urine_ protein in liver).
4-To corelate the ultra sound findings with clinical features
5- To describe different ultrasonic liver changes in malnutrition in relation to age, gender, weight and length.

1-4 Over view of the study

This study include five chapters, chapter one is introduction which, included, objectives and importance of study. Chapter two include back ground and literature review, Chapter three describes the material and method. Chapter four includes results presentation, finally chapter five includes the discussion, conclusion and recommendations.

Chapter Two

Chapter Two

Anatomy, physiology and pathology

2-1Anatomy of the liver

General arrangement of abdominal viscera are: liver, gall bladder ,esophagus ,stomach ,small intestine ,large intestine ,pancreas ,spleen ,kidneys ,supra renal glands and peritoneum.(Snell – clinical Anatomy Edition 7^{th}) The liver is a vital organ present in vertebrates and some other animals. It has a wide range of functions, including detoxification, protein synthesis, and production of biochemical's necessary for digestion. The liver is necessary for survival, there is currently no way to compensate for the absence of liver function in the long term, although new liver dialysis techniques can be used in the short terms(Richard S. Snell 2011).

This organ plays a major role in metabolism and has a number of functions in the body, including glycogen storage, decomposition of red blood cells, plasma protein synthesis, hormone production, and detoxification. It lies below the diaphragm in the abdominal-pelvic region of the abdomen. It produces bile, an alkaline compound which aids in digestion via the emulsification of lipids. The liver's highly specialized tissues regulate a wide variety of high-volume biochemical reactions, including the synthesis and breakdown of small and complex molecules, many of which are necessary for normal vital functions (Richard S. Snell 2011).

The liver is the largest gland in the body and has a wide variety of functions. Three of its basic functions are production and secretion of bile, which is passed into the intestinal tract involvement in many metabolic activities related to carbohydrate, fat, and protein metabolism, removing bacteria and other foreign particles that have gained entrance to the blood from the lumen of the intestine (Richard S. Snell 2011).

The liver synthesizes heparin, an anticoagulant substance, and has an important detoxicating function**.** It produces bile pigments from the hemoglobin of worn-out red blood corpuscles and secretes bile salts; these together are conveyed to the duodenum by the biliary ducts. The liver is soft and pliable and occupies the upper part of the abdominal cavity just beneath the diaphragm . The greater part of the liver is situated under cover of the right costal margin, and the right hemi diaphragm separates it from the pleura, lungs, pericardium, and heart. The liver extends to the left hemidiaphram. The convex upper surface of the liver is molded to the under surf ace of the domes of the diaphragm. The poster inferior, or visceral surface, is molded to adjacent viscera and is therefore irregular in shape; it lies in contact with the abdominal part of the esophagus, the stomach, the duodenum, the right colic flexure, the right kidney and suprarenal gland, and the gallbladder (Richard S. Snell 2011).

The liver may be divided into a large right lobe and a small left lobe by. the attachment of the peritoneum of the falciform ligament . The right lobe is further divided into a quadrate lobe and a caudate lobe by the presence of the gallbladder, the fissure for the ligamentum teres, the inferior vena cava, and the fissure for the ligamentum venos um. Experiments have shown that, in fact, the quadrate and caudate lobes are a functional part of the left lobe of

the liver. Thus, the right and left branches of the hepatic artery and portal vein, and the right and left hepatic ducts, are distributed to the right lobe and the left lobe (plus quadrate plus caudate lobes), respectively. Apparently, the two sides overlap very little. The porta hepatis, or hilum of the liver, is found on the posterior inferior surface and lies between the caudate and quadrate lobes . The upper part of the free edge of the lesser omentum is attached to its margins. In it lie the right and left hepatic ducts, the right and left branches of the hepatic artery, the portal vein, and sympathetic and parasympathetic nerve fibers. A few hepatic lymph nodes lie here; they drain the liver and gallbladder and send their efferent vessels to the celiac lymph nodes.

The liver is completely surrounded by a fibrous capsule but only partially covered by peritoneum. The liver is made up of liver lobules. The central vein of each lobule is a tributary of the hepatic veins. In the spaces between the lobules are the portal canals, which contain branches of the hepatic artery, portal vein, and a tributary of a bile duct (portal triad). The arterial and venous blood passes between the liver cells by means of sinusoids and drains into the central vein(Richard S. Snell 2011).

2-1-1 Anatomy of the Liver

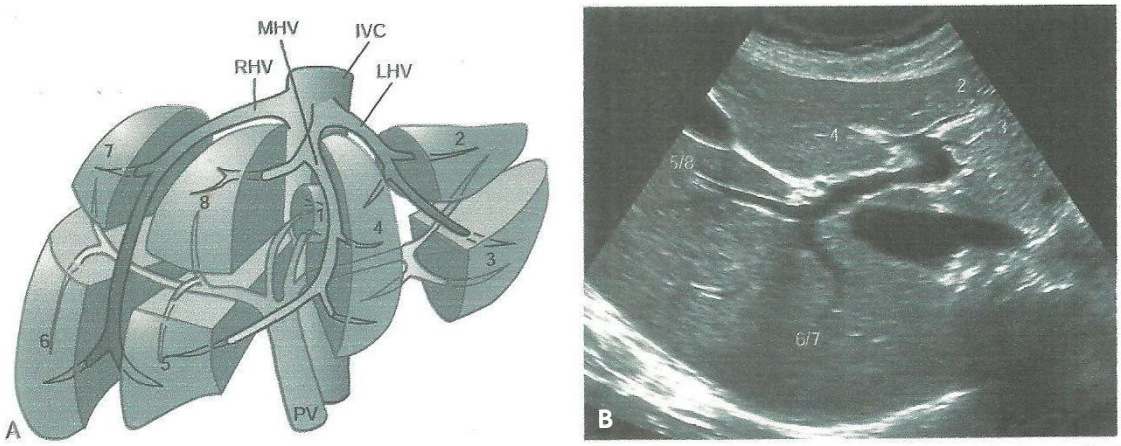


Figure 2-1 shows Couinaud's functional segmental anatomy. A, The liver is divided into nine segments. longitudinal
boundaries (right, middle, and left scissurae) are three hepatic veins. Transverse plane is defined by right main and left main portal pedicles.
Segment I, caudate lobe, is situated posteriorly. RHV, Right hepatic vein; MHV, middle hepatic vein; LHV, left hepatic vein; RPV, right
portal vein; LPV, left portal vein; GB, gallbladder. B, Corresponding sonogram shows the main portal vein with its right and left branches.

The plane through the right and left branches is the transverse separation of the liver segments. Cephalad to this level lie segments II, IVa, VII, and VIII. Caudally located are segments III, IVb, V, and VI. (Stephanie et.al;2011).

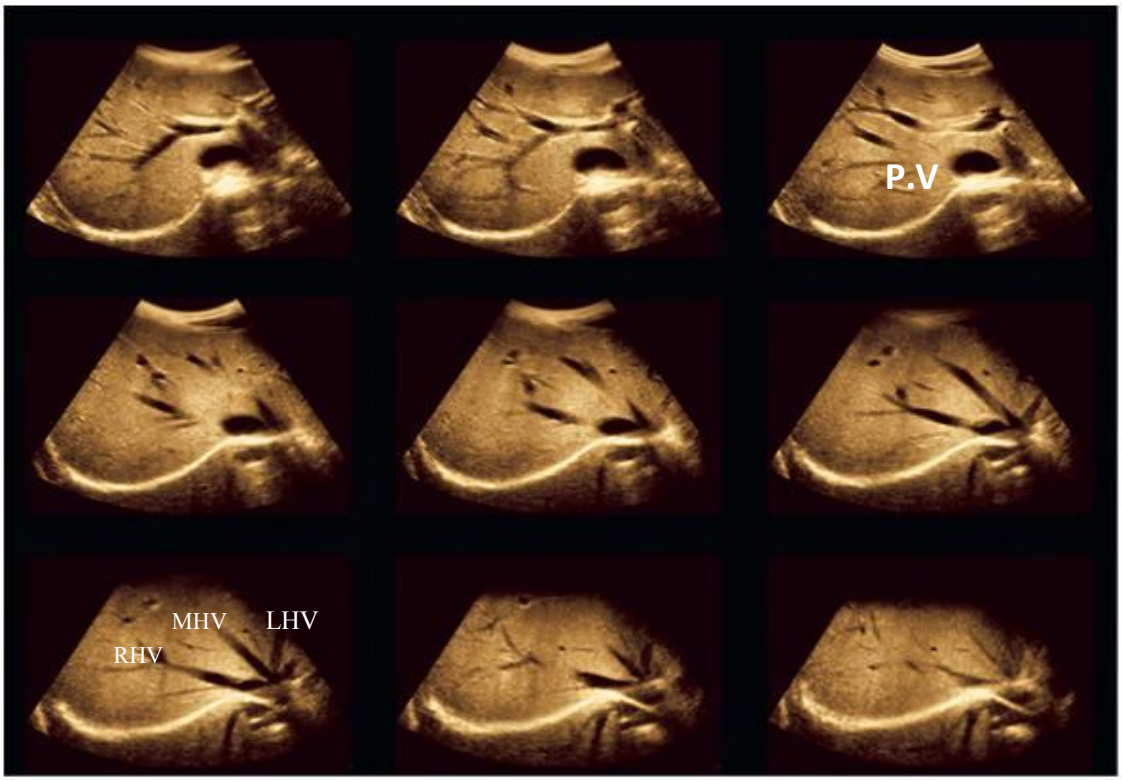


Figure 2-2. Normal liver. Liver shown in a nine-on-one format from a volumetric acquisition acquired in the axial plane, with the center point on the long axis of the portal veins (P.V) at the porta hepatis.(Stephanie et.al; 2011)

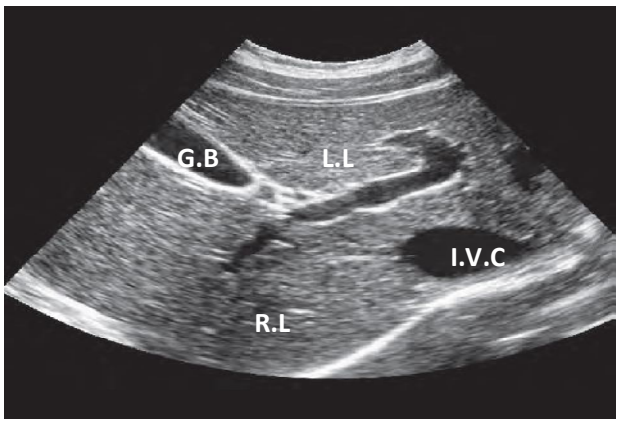


Figure2-3. Normal lobar anatomy. Right lobe of the liver (RL) can be separated from left lobe of the liver (LL) by themain lobar fissure that passes through the gallbladder fossa (GB) and inferior vena cava (IVC).(Stephanie.(Stephanie et.al; 2011).

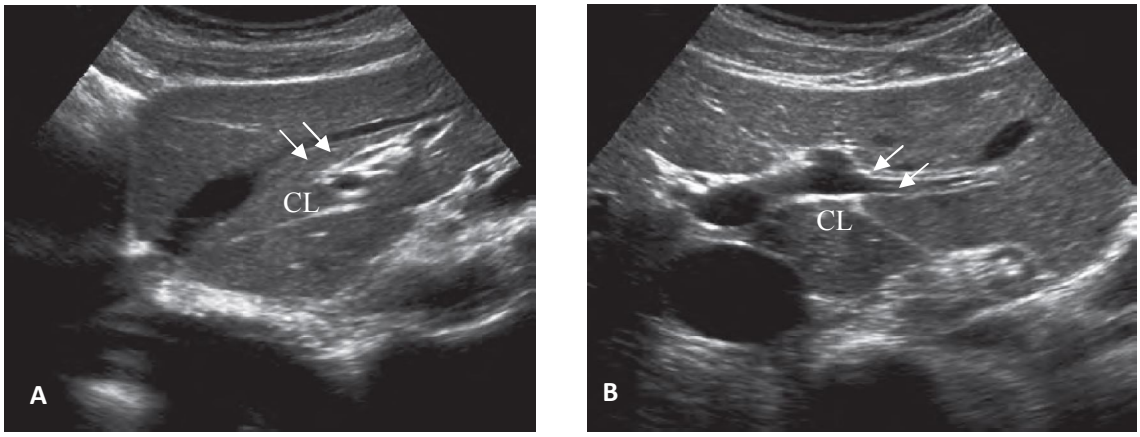


Figure 2-4. Caudate lobe. A, Sagittal view, and **B,** transverse view, show the caudate lobe (CL) separated from the left lobe by

the fissure for the ligamentum venosum (arrows) anteriorly. Posterior is the inferior vena cava *).*(Stephanie et.al; 2011)

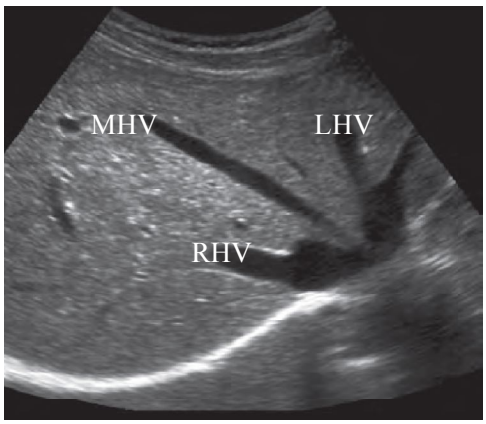


Figure 2-5. Hepatic venous anatomy. The three hepatic veins—right (RHV), middle (MHV), and left (LHV)—are interlobar and intersegmental, separating the lobes and segments.

At the level of the hepatic venous confluence with the inferior vena cava, the right hepatic vein separates the right posterior segment (segment 7) from the right anterior segment (segment 8).

The left hepatic vein separates the left medial segment from the left lateral segment. The middle hepatic vein separates the right and left lobes. As shown here, the hepatic veins are best seen on a sub costal oblique view*)*.(Stephanie et.al,2011).

2-1-2 Important relations of the liver and organs

2-1-2-1 Anteriorly: Diaphragm, right and left costal margins, right and left pleura and lower margins of both lungs, xiphoid process, and anterior abdominal wall in the sub costal angle.

2-1-2-2 Posteriorly: Diaphragm, right kidney, hepatic flexure of the colon, duodenum, gallbladder, inferior vena cava, and esophagus and fundus of the stomach. .(Anne,et.al2005).

2-1-3 Peritoneal Ligaments of Liver

The falciform ligament, which is a two-layered fold of the peritoneum, ascends from the umbilicus to the live . ft has a sickle-shaped free margin that contains the ligamentum teres, the remains of the umbilical vein. The falciform ligament passes on to the anterior and [hen the superior surfaces of the liver and then splits into two layers. The right layer forms the upper layer of the coronary ligament; the left layer form the upper layer of the left triangular ligament . The right extremity of the coronary ligament is known as the right triangular ilgament of the liver. it should be noted that the peritoneal layers forming the cronary ligament are widely separated, leaving an area of liver devoid of peritoneum. Such an area is referred to as a bare area of the liver .(Anne,et.al2005).

The ligamentum teres passes into a fissure on the visceral surface of the liver and joins the left branch of the portal vein in the porta hepatis . The ligam entum venosum**,** a fibrous band that is the remains of the ductus

venosus, is attached to the left branch of the portal vein and ascends in a fissure on the visceral surface of the liver to be attached above to the inferior vena cava . In the fetus, oxygenated blood is brought to the liver in the umbilical vein (ligamentum *teres)*. The greater proportion of the blood bypasses the liver in the ductus venosus (ligamentum venosum) and / joins the inferior vena cava. At birth, the umbilical vein and ductus venosus close and become fibrous cords. (Snell – clinical Anatomy Edition 7^{th}) The lesser omentum arises from the edges of the porta hepatis and the fissure for the ligamentum venosum and passes down to the lesser curvature of the stomach (Anne,et.al2005).

2-1-4Blood Supply of the Liver

2-1-4-1Arteries

The hepatic artery, a branch of the celiac artery, divides into right and left terminal branches that enter the porta hepatis.

2-1-4-2 Veins

The portal vein divides into right and left terminal branches that enter the porta hepatis behind the arteries. The hepatic veins (three or more) emerge from the posterior surface of the liver and drain into the inferior vena cava .(Anne,et.al2005).

2-1-5 Portal Circulation.

The blood vessels conveying blood to the liver are the hepatic artery (30%) and portal vein (70%). The hepatic artery brings oxygenated blood to the liver, and the port al vein brings venous blood rich in the products of digest ion, which have been absorbed from the gastrointestinal tract. The arterial and venous blood is conducted to the central vein of each liver lobule by the liver sinusoids. The central veins drain into the right and left hepatic veins, and - these leave the posterior surface of the liver and open directly into the inferior vena cava.(Anne,et.al2005).

2-1-6 Lymph Drainage of the liver

The liver produces a large amount of lymph—about one third to one half of all body lymph. The lymph vessels leave the liver and enter several lymph nodes in the porta hepatis The efferent vessels pass to the celiac nodes. A few vessels pass from the bare area of the liver through the diaphragm to the posterior mediastinal lymph nodes (Chummy S. et.al2005).

2-1-7 Nerve Supply of the liver

Sympathetic and parasympathetic nerves form the celiac plexus . the anterior vagal trunk gives rise to a large hepatic branch, which passes directly to the liver (Chummy S. et.al2005).

2-1-8 Bile Ducts of the Liver .

Bile is secreted by the liver cells at a constant rate of about 40 mL per hour. When digestion is not taking place, the bile is stored and concentrated in the gallbladder; later is delivered to the duodenum. The bile ducts of the liver consist of the right and left hepatic ducts, the common hepatic duct, the bile duct, the gallbladder, and the cystic duct(Chummy S. et.al2005). The smallest interlobular tributaries of the bile ducts are situated in the portal canals of the liver; they receive the bile canaliculi. The interlobular ducts join one another to form progressively larger ducts and, eventually, at the porta hepa tis, form the right and left hepatic ducts. The right hepatic duct drains the right lobe of the liver and the left duct drains the left lobe, caudate lobe, and quadrate lobe (Chummy S. et.al2005).

2-1-8-1 Hepatic Ducts

The right and left hepatic ducts emerge from the right and left lobes of the liver in the porta hepatis . After a short course, the hepatic ducts unite to form the common hepatic duct.

The common hepatic duct is about 1.5 in. (4 cm) long and descends within the free margin of the lesser omentum. It is joined on the right side by the cystic duct from the gall bladder to form the bile duct .

2-1-8-2 Common Bile Duct (CBD)

The bile duct (common bile duct) is about 3 in. (8cm) long. In the first part of its course, it lies in the right free margin of the lesser omentum in front of the opening into the lesser sac. Here, it lies in front of the right margin of the portal vein and on the right of the hepatic artery . In the second part of its course, it is situated behind the first part of the duodenum to the right of the gastro duodenal artery . In the third part of its course, it lies in a groove on the posterior surface of the head of the pancreas . Here, the bile duct comes into contact with the main pancreatic duct .
The bile duct ends below by piercing the medial wall of the second part of the duodenum about halfway down its length . It is usually joined by the main pancreatic duct, and together they open into a small ampulla in the duodenal wall, called the hepato pancreatic ampulla (ampulla of Vater). The ampulla opens into the lumen of the duodenum by means of a small papilla, the major duodenal papill& .
The terminal parts of both ducts and the ampulla are surrounded by circular muscle, known as the sphincter of the hepatopancreatic ampulla (sphincter of Oddi) . Occasional, the bile and pancreatic ducts open separately into the duodenum(Chummy S. et.al2005).

2-1-9 Gallbladder

2-1-9-1 Location and Description of Gallbladder.

The gallbladder is a pear-shaped sac lying on the under surf ace of the liver . It has a capacity of 30 to 50 ml and stores bile, which it concentrates by absorbing water. For descriptive purposes, the gallbladder is divided into the funds. body, and neck. The funds is rounded and usually projects below the inferior margin of the liver, where it comes in contact with the anterior abdominal wall at the level of the tip of the ninth right costal cartilage. The body lies in contact with the visceral surface of the liver and is directed upward, backward, and to the left. The neck becomes continuous with the

cystic duct, which turns into the lesser omentum to join the right side of the common hepatic duct, to form the bile duct(Anne et.al2005).

The peritoneum completely surrounds the funds of the gallbladder and binds the body and neck to the visceral surf ace of the liver.

2-1-9-2 Relations of Gallbaldder & organs.

Anteriorly: The anterior abdominal wall and the inferior surface of the liver Posteriorly: The transverse colon and the first and second parts of the duodenum (Anne et.al2005).

2-1-9-3Blood Supply of gallbladder.

The cystic artery, a branch of the right hepatic artery , supplies the gallbladder. The cystic vein drains directly into the portal vein. Several very small arteries and veins also run between the liver and gallbladder(Anne et.al2005).

2-1-9-4 Lymph Drainage of gallbladder

The lymph drains into a cystic lymph node situated near the neck of the gallbladder. From here, the lymph vessels pass to the hepatic nodes along the course of the hepatic artery and then to the celiac nodes(Anne et.al2005).

2-1-9- 5 Nerve Supply of gallbladder

Sympathetic and parasympathetic vagal fibers form the celiac plexus. The gallbladder contracts in response to the hormone cholecystokinin, which is produced by the mucous membrane of the duodenum on the arrival of fatty food from the stomach (Anne et.al2005).

2-1-10 Cystic Duct

The cystic duct is about 1.5 in. (3.8 cm) long and connects the neck o the gallbladder to the common hepatic duct to form the bile duct . It usually is somewhat S 3.ia)eel and descends for a variable distance in the right free margin o f the lesser omentum (Anne et.al2005).

the mucous membrane of the cystic duct is raised to form a spiral fold that is continuous with a similar fold in the neck of the gallbladder. The fold is commonly known as the spiral valve." The function of the spiral valve is to keep the lumen constantly open.

2-2Physiology

The various functions of the liver are carried out by the liver cells or hepatocytes. Currently, there is no artificial organ or device capable of emulating all the functions of the liver. Some functions can be emulated by liver dialysis, an experimental treatment for liver failure. The liver is thought to be responsible for up to 500 separate functions, usually in combination with other systems and organs (Allison grant ,et.al 2009).

2-2-1Synthesis Functions of the Liver

Further information : Proteins produced and secreted by the liver
A large part of amino acid synthesis

The liver performs several roles in carbohydrate metabolism:

Gluconeogenesis (the synthesis of glucose from certain amino acids , lactate or glycerol).

Glycogenolysis (the breakdown of glycogen into glucose).

Glycogenesis (the formation of glycogen from glucose)(muscle tissues can also do this).

The liver is responsible for the mainstay of protein metabolism, synthesis as well as degradation .

The liver also performs several roles in lipid metabolism: Cholesterol synthesis.

Lipogenesis, the production of triglycerides(fats).

A bulk of the lipoproteins are synthesized in the liver.

The liver produces coagulation factors I (fibrinogen), II (prothrombin), V,VII, IX, X and XI, as well as protein C, protein S and antithrombin.

In the first trimester fetus, the liver is the main site of red blood cell production. By the 32nd week of gestation, the bone marrow has almost completely taken over that task.

The liver produces and excretes bile (a yellowish liquid) required for emulsifying fats and help the absorption of vitamin K from the diet. Some of the bile drains directly into the duodenum, and some is stored in the gallbladder (Allison grant ,et.al 2009).

The liver also produces insulin-like growth factor 1 (IGF-1), a polypeptide protein hormone that plays an important role in childhood growth and continues to have anabolic effects in adults.

The liver is a major site of thrombopoietin production. Thrombopoietin is a glycoprotein hormone that regulates the production of platelets by the bone marrow (Allison grant ,et.al 2009).

2-2-2Breakdown Function of the Liver

The breakdown of insulin and other hormones The liver glucoronidates bilirubin, facilitating its excretion into bile.

The liver breaks down or modifies toxic substances (e.g., methylation) and most medicinal products in a process called drug metabolism. This sometimes results in toxication, when the metabolite is more toxic than its precursor. Preferably, the toxins are conjugated to avail excretion in bile or urine The liver converts ammonia to urea (urea cycle).

(supply)[citation needed] , vitamin B12 (1–3 years' supply), vitamin K, iron, and copper.

The liver is responsible for immunological effects-the reticuloend othelial system of the liver contains many immunologically active cells, acting as a

sieve for antigens carried to it via the portal system (Allison grant ,et.al 2009).

The liver produces albumin, the major osmolar component of blood serum.

The liver synthesizes angiotensinogen, a hormone that is responsible for raising the blood pressure when activated by renin, an enzyme that is released when the kidney senses low blood pressure (Allison grant ,et.al 2009).

2-2-3Function of the Gallbladder

When digestion is not taking place, the sphincter of Oddi remains closed and bile accumulates in the gallbladder. The gallbladder concentrates bile; stores bile; selectively abs orbs bile salts, keeping the bile acid; excretes cholesterol; and secretes mucus. To aid in these functions, the mucous membrane is thrown into permanent folds that unite with each other, giving the surface a honeycombed appearance. The columnar cells lining the surface also have numerous microvilli on their free surface.
Bile is delivered to the duodenum as the result of contract ion and partial emptying of the gallbladder. This mechanism is initiated by the entrance of fatty foods into the duodenum. The fat causes release of the hormone cholecystokinin from the mucous membrane of the duodenum; the hormone then enters the blood, causing the gallbladder to contract. At the same time, the smooth muscle around the distal end of the bile duct and the ampulla is relaxed, thus allowing the passage of concentrated bile into the duodenum. The bile salts in the bile are important in emulsifying the fat in the intestine and in assisting with its digestion and absorption (Anne et.al2005)

2-4 Relation of the Liver to medicine and pharmacology:

The oxidative capacity of the liver decreases with aging and therefore any medications that require oxidation (for instance, benzodiazepines) are more likely to accumulate to toxic levels.(Anne et.al2005).

However, medications with shorter half-lives, such as lorazepam and oxazepam, are preferred in most cases when benzodiazepines are required in regards to geriatric medicine.(Anne et.al2005).

2-5 Pathology

2-5-1 Mechanism of Malnutrition

In the first 24 hours following low dietary intake , the body relies for energy on breakdown of hopstic glycogen to glucose (Richard S. sneil 2011)

Hepatic glycogen stores are small and therefore gluconogenesis is soon necessary to maintain glucose level . gluconeogenesis takes place mainly from pyruvate , lactate , glycerol and amino acids especially alanine and glutamine . the majority of protein breakdown takes place in muscle . gluconogenesis for amino acids particularly from alanine in the liver and glutamine in the kidney the metabolic response to prolonged starvation differs between thin and obese individuals one of the major different concerns the energy derived from protein which determines the proportion of weight loss from lean tissues, This proportion may be up to three times smaller in obese subject than lean subjects. lipolysis the breakdown of the body's fat store also occur if is inhibited by insulin but level of this hormone falls off as starvation continues . the stored triglyceride is hydrolysable by lipase to glycerol . which is used for gluconeogenesis and also to non-esterifies fatty acids that can be used directly as a fuel or oxidized in the liver to ketene bodies as starvation continuous adaptive processes take place lost the body available protein be completely utilized . there is decrease in metabolic rate and total body energy expenditure . control neurons metablelism changes from glucose as substrate to ketene bodies which now become the main source of energy for the brain .

gluconeogenesis in the liver decrease as does Portion break down in muscle both these processes being inhibited directly by ketene bodies which are

deprival from fat most of energy of this stage comes from adipose tissue with same.(Snell – clinical Anatomy Edition 7th).

2-5-2Malnutrition:

Usually means the inappropriate intake of one or more or the nutrients essential for normal growth and development of the body

(Jamal Nassir,1963) .

2-5-3 Protein –Energy Malnutrition (PEM):

It is pathological conditions arising from coincident lack of proteins and calories ,occurring most frequently in infants and young children and commonly associated with infection .can be classified by many authors . the welcome classification is a simple and universally accepted ,depending on 2 main criteria weight loss and edema:

Table 2-1 showed (Welcome Classification Of PEM)

	Body Weight as %of Standard	Edema	Deficit in Weight for Beight
Underweight	60-80%	-	Mild
Marasmus	<60%	-	Marked
Kwashiorkor	60-80%	+	Mild
Marasmic Kwashiorkor	<60%	+	Marked
Nutritional Dwarf	<60%	-	Marked

(Jamal Nassir,1963) .

2-5-4The Under weight Child:

Characterized by failure to thrive as judged by retarded growth and development in addition , signs of infection and anemia may be present.

2-5-5 Clinical Picture of Malnutrition:

2-5-5-1. Growth failure manifested by weight loss. Slowing in linear growth (height), and delayed bone maturation.

2-5-5-2. Infection particularly gastroenteriris , pneumonia , measles ,malaria , and hookworms and schistosomiasis are more prevalent in the underweight children.

2-5-5-3. Anemia : most cases of mild PEM suffer from a moderate degree of anemia which is caused by deficiency of several factors including iron , folic acid and proteins.

2-5-5-4. Retardation of development : retarded milestones are characteristics of PEM . children may walk or talk later than usual.

2-5-5-5. Diminution of activity and restlessness are usually observed in children with PEM (Jamal Nassir,1963).

2-5- 6 Marasmus

Marasmus (or severe wasting) is form of severe PEM that may occur at any age particularly in early infancy and is characterized by severe wasting (body weight is less than 60% of the expected), loss of subcutaneous fat , gross muscle wasting and absence of edema (Jamal Nassir,1963).

2-5-7 Etiology of Marasmus:

2-5-7-1 Nutritional Marasmus:

The main cause of nutritional marasmus is an inadequate but more or less balanced diet deficient in both proteins and calories.This may occur due to:

Failure of breast feeding(This will lead to underfeeding and to replacement of breast milk by formula or by early abrupt weaning), (Inadequate amounts

of milk formula small quantities or over-dilution of the formula), as well as the use of contaminated utensils in preparing the formula causing infective diarrhea), Starvation therapy for diarrhea : if repeated and prolonged, Feeding difficulties : due to presence of mental retardation ,cerebral palsy or congenital anomalies as cleft palate and Prematurity : due to poor suckling in presence of rapid growth rate(Jamal Nassir,1963).

2-5-7-2 Secondary Marasmus:

Caused by the following diseases, Chronic severe infections :as tuberculosis ,urinary tract infection, bronchiectasis ,chronic osteomyelitis, infections produce malnutrition due to severe anorexia, Chronic diarrhea and/or vomiting, Malabsorption syndromes, Congenital malformations(GIT : pyloric stenosis , congenital biliary atresia CVS : cyanotic congenital heart disease ,Kidney : e.g. obstructive uropathy), Metabolic disorders : as galactosemia , Endocrinal diseases : hyperthyroidism , diabetes mellitus, Psychological disturbances : maternal deprivation syndrome and Malignancy as neuroblastoma (Jamal Nassir,1963).

2-5-8 Clinical Manifestations of marasmus:

2-5-8-1. Growth Failure:

Weight is less than 60% of expected for age and sex, Length and head circumference are also affected but need longer duration of malnutrition than weight.

2-5-8-2 Loss of subcutaneous fat:

it is lost in the following order From the abdominal wall leading to less of the skin elasticity, From the limbs (thighs and buttocks): the skin becomes

wrinkled and hanging into longitudinal folds and The buccinators bad of fat is the last to disappear (probably due to different chemical composition of its fat). This leads to hollowing of the cheeks, triangular face and an appearance resembling an old man face(Jamal Nassir,1963).

2-5-8-3 Marked wasting of muscles:

This together with loss of subcutaneous fat leads to Stick –like appearance of limbs, Scaphoid abdomen with marked thinning of abdominal wall. (Jamal Nassir,1963).

2-5-8-4 Psychic Changes:

Marasmic infants look anxious , irritable ,cry and sleep little however . they look less miserable than cases of Kwashiorkor . Marasmas infants are usually hungry and have good appetite . Sometimes , there is anorexia and poor feeding (especially in secondary marasmus).

2-5-8-5 Chronic diarrhea: With or without vomiting.

2-5-8-6 Intercurrent infections: Like otitis media ,bronchopneumonia , urinary tract infection are commonly present.

2-5-8-7 Associated deficiencies: of iron , vitamin **A** and **D** may be present (Jamal Nassir,1963).

2-5-9 Kwashiorkor:

Also called the wet, swollen or edematous from is associated with premature abandonment of breast feeding , which typically occurs when a younger sibling is born. So children with kwashiorkor tend to be older than those

with marsmus, Kwashiorkor may also result from an acute illness ,often gastroenteritis or anther infection. (Jamal Nassir,1963).

2-5-10 Symptoms and signs of kwashiorkor:

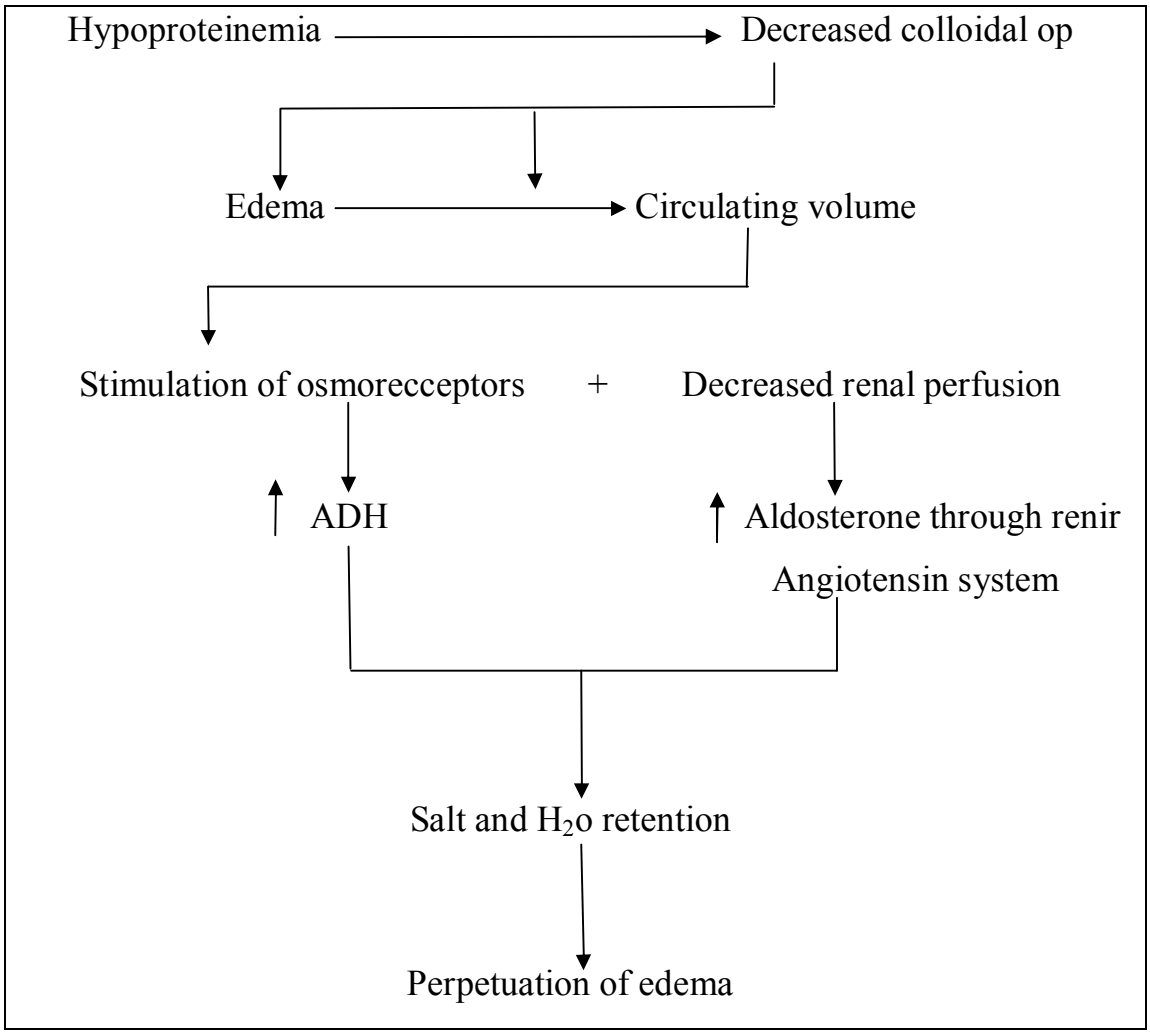
2-5-10-1. Growth Failure :

This is reflected, Weight is diminished to 60-80% of expected for age, Retared linear growth (length) This occurs mostly in long standing cases and affection is not usually to the same extent as weight, Head circumference may be also affected and Bone age may retarded (Jamal Nassir,1963).

2-5-10-2 Edema:

This is the most constant clinical manifestation of Kwashiorkor. it starts in the feet and lower parts of the legs then becomes generalized .it is usually soft and pitting , affecting more the dependent parts (back and dorsum of hands and feet).The cheeks become bulky, pale and waxy in appearance (doll-like cheeks) (Jamal Nassir,1963).

Table 2-2 demonstrate Pathogenesis of edema):



demonstrate Pathogenesis of edema (Jamal Nassir,1963).

2-5-10-3 Disturbed muscle /fat ratio:

There is a generalized muscle wasting with preservation of some subcutaneous fat . This can be demonstrated clinically by measuring the mid arm circumfrernce which is diminished in these cases .The children are often weak , hypotonic and unable it stand and walk (Jamal Nassir 1963).

2-5-10-4 Psychic Changes:

Infants with kwashiorkor have marked apathy misery and peevishness They lack interest in the surrounding they do not move. look sad never smile Their cry is weak(Jamal Nassir,1963).

2-5-11 Usual Manifestations of Kwashiorkor:

They include

2-5-11-1 Hair changes :

The hair loses its black color and becomes reddish or grayish. The cause of this dyspigmentation is obscure.

Deficiency of pantothenic acid sulfur containing amino acids (cystine and methionine) in the hair or a defect in the melanin formation may be responsible.

2-5-11-2 Gastrointestinal Manifestations :

Anorexia ,Sometimes associated with vomiting is usual in severe cases and Diarrhea is common and can be due to Infection with intestinal pathogens or parasites.Reduction of intestinal and pancreatic enzymes (e.g. amylase , lipase , trypsin…) as a result of protein deficiency .This will lead to inadequate digestion of food and passage of loose stools as a consequence, Malabsorption of nitrogen , fat, carbohydrates and minerals due to the atrophy of villi.

Disaccharidase deficiency (especially lactase) leads to a fermentative diarrhea accompanied by abnormal distention and flatulence Infection with intestinal pathogens or parasites(Jamal Nassir,1963).

Defect in conjugation of bile salts ⟶ malabsorption of lipids Infection with intestinal pathogens or parasites(Jamal Nassir,1963).

2-5-12 Occasional Manifestations of Kwashiorkor:

They include (Skin changes, Hepatomegaly, Anemia, Associated vitamin and mineral deficiencies) Infection with intestinal pathogens or parasites

2-5-12-1 Skin changes:

Dermatosis(Dermatosis of Kwashiorkor is pathognomonic and may be caused by deficiency of essential amino acids ,vitamin and Niacin as well as associated suprarenal function disturbances and zine deficiency and Sometimes petechiae may be present, particularly over the abdomen).

(Jamal Nassir,1963).

2-5-12-2 Hepatomegaly:

It is caused by fatty infiltration of the liver which is a constant pathological finding in kwashiorkor that may or may not be accompanied by hepatomegaly, increased mobilization of free fatty acids from adipose tissue to the liver, increased fatty acid synthesis from glucose, decreased oxidation of fatty acid in the liver and decreased synthesis of apolipoprteins lead to decreased release of fat from the liver. (Jamal Nassir,1963).

2-5-12-3 Anemia

It is due to multiple factors, Deficiency of protein . iron ,zinc ,copper ,folic acid and vitamins A,E,B_1,B_{12} and /or C, Infections , Associated deficiencies of vitamins ,minerals , trace elements and Poor resistance and liability to infections (Jamal Nassir,1963).

2-5-13 Complications of Kwashiorkor:

Complications of diarrhea :dehydration ,electrolyte, and acid-base disturbances , Infections and septicemia, Severe hypoglycemia, Hypothermia and Heart failure which may be iatrogenic due circulatory overload by excess IV fluids , plasma or blood transfusion .

(Jamal Nassir,1963).

Table 2-3 showed (Differentiation Between Kwashiorkor and Marasmus)

	Kwashiorkor	**Marasmus**
Etiology (primary)	Unbalanced diet (high CHO, low proteins).	Intake of diet deficient in calories and proteins nutritional disorder or secondary to diseases as chronic infections , malignancy …etc).

(Jamal Nassir,1963).

Table 2-4 showed different between kwashiorkor and marasmus in clinical manifestation:

	Clinical Manifestations	
Weight	Kwashiorkor 60-80% expected	Marasmus <60%
Edema	+ve	-ve
Subcutaneous fat	Somewhat Preserved	Loss
Muscle Wasting	+	++
Psychic changes	++	+
Hair Changes	+	+/-
Skin Changes	+	-
GIT Anorexia Diarrhea	 ++ +	 +/- +
Hepatomegaly	+	-
Anemia	+	+
Vitamins ,Minerals deficiency	+	+

(Jamal Nassir,1963).

Table 2-5 showed different between kwashiorkor and marasmus in Complication

	Complications	
Infection	Kwashiorkor ++	Marasmus +
Dehydration and electrolyte	+	+
Heart failure	+	-
Hypothermia	+	++
Hypoglycemia	+	+

Table 2-6 showed different between kwashiorkor and marsmus in investigation:

	Investigations	
Total Plasma Protein	Kwashiorkor Decreased	Marasmus Normal
Serum albumin	Decreased	Normal

(Jamal Nassir,1963).

2-5-14 Marasmic Kwashiorkor:

This is syndrome which has the characteristics of both kwashiorkor and marasmus .it main manifestations are :

2-5-14-1. Growth failure :

Body weight is less than 60% of the expected weight for age .

2-5-14-2. Edema of feet ,legs and dorsum of hands .

2-5-14-3. Loss of subcutaneous fat from the abdominal wall ,thighs ,buttocks and shoulder .

2-5-14-4. Marked wasting of muscles .

2-5-14-5. Other manifestations :psychic changes , dermatosis and hair changes may be present .

2-5-15 Management of Protein Energy Malnutrition

2-5-15-1 Mild Cases of PEM:

No need for hospitalization and Appropriate diet should be advised with sufficient amount of Calories (150 Kcal/Kg/day)+sufficient amounts of proteins (2-3g/kg/day)+vitamins and minerals Infection with intestinal pathogens or parasites (Jamal Nassir,1963) .

2-5-15-2 Severe cases of PEM (Marasmus, Kwashiorkor, Marasmic Kwashiorkor):

2-5-15-2-1Hospitalization and Investigations:

Investigations: The following investigations are helpful:(Complete blood picture(for anemia and leucocytosis), Urine analysis for pus cells and urine culture, Stool analysis for parasites as Giardia Lamblia , Stool culture in cases of diarrhea, chest x-ray for evidences of TB as enlarged lymph nods, miliary TB or for evidence of bronchopneumonia ,or empyema , tuberculin test for TB(suspected to be falsely negative) and Other investigations may be needed according to the case u/s for congenital hypertrophic pyloric stenosis ,or congenital renal abnormalities …etc

Infection with intestinal pathogens or parasites. In Kwashiorkor and marasmic Kwashiorkor :serum proteins and albumin (used as a base line follow up) (Jamal Nassir,1963).

2-5-15-2-2 Emergency Treatment :

2-5-13-2-2-1 Dehydration and electrolyte disturbances:

Rehydration is performed by oral nasogastric route I.V rehydration is used only in cases of shock or failure of oral rehydration (Jamal Nassir 1963).

Types of fluids used :

1. For oral rehydration :Low sodium ,high potassium ORS is given .

2. For I.V . rehydration .

Ringer's Lactate + glucose 5%(in a ratio 1:1)+2 ml KCI (15%)for each liter .

or Pansol + 1 ml kcl (15%)for each litre .

or Ringer's Lactate + glucose 5%+Kadalex (2:1:1) (Jamal Nassir 1963).

2-5-15-2-2-2 Infections :

The following combination can be used for 5-10 days :

Ampicillin + Chloramphenicol or

Ampicillin + Kanamycin or

Ampicillin + Gentamicin

Metronidazole can be used in anaerobic infections (e.g. Liver abscess).

Parasites should be eradicated by appropriate drugs e.g. mebendazole in the presence of clinically manifest infection , the suitable antibiotic is added to the above combination (Jamal Nassir 1963).

2-5-15-2-2-3 Hypothermia:

(rectal temperature less than 35ºC)

2-5-15-2-2-4 Hypothermia:

Can be prevented by frequent feeding (every 2 hours) and Treated in mild cases by an oral feed of glucose in water or milk.

2-5-15-2-2-5 Heart failure:

(may be manifested by failure to lose weight to in spite disappearance of edema , liver enlargement , congested neck veins , appearance of third heart sound , fine basal crepitations in lungs ,and sometimes peripheral circulatory failure), Malnourished children are sensitive to digitalis and should not be digitalized, Oxygen and other supportive measures may be required and Avoid giving unnecessary fluids I.V(Jamal Nassir 1963).

2-5-15-2-2-6 Blood transfusion:

(10 ml/kg)is given in the following conditions (Severe infections- Persistent anorexia- Loss of consciousness- Severe anemia - Unresponsive edema- Liver failure) (Jamal Nassir 1963) .

2-5-15-2-3 Dietetic Therapy and Nutritional Rehabilitation:

2-5-15-2-3-1 Starting cautious initial feeding:

Parenteral alimentation should be avoided and used only as a last resort, Feeding should be by the oral or nasogastric route, Start by little, frequent and isotonic feeds, Given from admission until return of appetite (i.e. for about 5-7 days) , It should provide 75 kcal/100 ml and <1 g of protein/ 100 ml, Give 100 ml/kg of body weight /day divided to 10 small feeds/day, A half strength humanized formula may be started with, If the child is intolerant to milk (ex: develops diarrhea due to lactose intolerance), a low lactose free formula can be used in the preparation of feeds Example of initial diet (Jamal Nassir,1963).

Composition :

Whole milk	30 ml
Sucrose	10 g
Vegetable	2ml
Electrolyte /mineral solution	2ml
Water:add to make	100 ml

(The electrolyte solution is composed of KCI +Tri K citrate + MgCI +Zine acetate and Copper sulfate) (Jamal Nassir 1963).

2-5-15-2-3-2. Subsequent diet :

Given after the first 5-7 days when child regains his appetite , Provides 100 kcal /100 ml and from 2.5-3 g of protein per 100 ml and Give at least 120-

130 ml/kg of body weight in 8 feeds (about 15 ml/kg/feed) Example of subsequent diet :

Composition:

Whole milk	85 ml
Sucrose	7.5 g
Vegetable oil	2 ml
Electrolyte/mineral solution	2 ml
Water : add to make	100 ml

2-5-15-2-3-3 High energy diet for catch-up growth:

After the tow weeks with full return of appetite , start giving semisolid feeds in addition to milk (e.g. cereals , porridge ,eggs ,cheese ,beans , minced meat ,etc.) , The child should be fed to appetite ,frequently (every 3-4 hours) and amounts are unlimited providing at least 150-220 kcal/kg and 4-6g/kg protein per day.

2-5-15-2-3-4. Supplement of micronutrients (vitamin, minerals &trace elements):

Multivitamin preparations are given provide at least double the recommended daily allowances (RDA) daily, in case of vitamin a deficiency Give a single dose of 50.000-200.000 units orally, zinc , copper and other trace elements(iodine , selenium ,etc) should also be provided and iron should NOT be given until infections are treated even if the child is anemic (Jamal Nassir,1963).

2-5-15-2-3-5 Stimulation play and emotional support:

PEM delays the mental &behavioral development, Tender , loving care , structured play and cheerful environment are as important as diet .

2-5-16 Criteria of cure:

apathy and irritability disappear early (in 3-4days), Edema increases at first (because of fluids given)then disappear in about 10 days , Skin lesions heal rapidly without local therapy in about 10 days , There is an initial weight loss then the child starts to gain weight(about 2-3 kgs in 10 weeks) , Serum albumin level of more than 3 g/dl is an evidence of initiation of cure and The child regains his normal health and vigour in about 3 months .

2-5-17 Organs affected by Malnutrition:

2-5-17-1 Brain

Malnutrition can damage the brain in several ways – according to eating Disorder information and referral center such as anorexia or bulimia have high risk of seizures

Over time can reduce number of cells that carry oxygen to your brain . Also lead to development of brain lesion (www.m.webmd.com/fatty liver).

2-5-17-2 Kidneys

Malnutrition affects kidney function, mineral and electrolyte metabolism impaired kidney function due to malnutrition may not be evident at first .

When person is not getting enough calories and protein, the kidneys cannot concentrate urine usual. the kidney filtration and flow also rate decrease (www.m.webmd.com/fatty liver).

2-5-17-3 Heart

The Risk of heart attack is greater in malnourished long term, The body may turn to it's own Muscle tissue as a source of protein and energy it heart muscles become wasted as result of malnutrition . the risk of heart attacked increase (www.m.webmd.com/fatty liver).

2-5-17-4 Lung

Impacted by Malnutrition. the respiratory muscles function can become wasted and body become generally less able to control breathing effective .the risk of lung collapse is greater as risk of lung infection(www.m.webmd.com/fatty liver).

2-5-17-5 Liver

Affected by malnutrition cause fatty liver

2-5-17-5-1 Fatty infiltration

Fatty infiltration of the liver is characterized by excessive deposition of neutral fat (triglyceride), and to a lesser extent phospholipid and cholesterol within the parenchyma cells of the liver The normal fat content of the liver varies from 5 to 7 percent by weight. In patients with fatty infiltration, fat represents 30 to 40 percent of the weight of the liver.

Alcohol consumption is the most common cause of fatty inflation of the liver in adults; malnutrition is a frequent cause in children. Other common predisposing factors of fatty infiltration of the liver include diabetes mellitus obesity , severe hepatitis_parenteral nutrition. Corticosteroid therapy starvation. jejunoleal bypass. and hyper lipidemia.' Uncommon causes are

tubes is. ulcerative colitis, excessive overeating, Reyes syndrome. glycogen storage disease. cystic fibrosis. trauma. pregnancy. halothane anesthesia, and massive tetracycline therapy.(Carlo A.et.al1993).

Fatty involvement of the liver is usually a diffuse process. At times, the fatty distribution can be non uniform, more frequently affecting the right lobe. This pa tern often occurs in patients with malnutrition. especially when it is associated with malignancy, ethanol al) se, or the exogenous ingestion of corticosteroids. The exact patho genesis of uneven or focal distribution of fatty infiltration the liver is not known, though differential blood flow to the lobes of the liver may account for these changes .(Carlo A.et.al1993).

2-5-17-5-2 Clinical Findings

The clinical features of a fatty liver depend upon the duration and severity of the condition. The patient may be symptomatic or present with hepatomegaly. right upper quadrant discomfort or pain. Tenderness. or occasionally jaundice. Liver enzymes may be normal or elevated. .(Carlo A.et.al1993).

2-5-17-5- Sonographic Characteristics

1. There is a diffuse increase in echogenicity with fine homogeneous echoes. The ratio of fibrous tissue to fat accounts for differences in echo genicty of different liver regions, although the exact etiology of the increased echogenicity is not know.

2. The liver may be mild to moderately enlarged.
3. Sound transmission through a fatty liver is very poor
4. There may' be poor delineation of the right hemi diaphragm and the

vascular structures in the liver.

5. Fatty liver has been divided into three grades according to the degree of increased echo genicity, visualization of the right hemi diaphragm and intra hepatic vessels, and through transmission of the sound beam

GRADE I **A** slight diffuse increase in fine

(Mild) echoes in the hepatic parenchyma: normal visualization of

intra hepatic vessel borders and diaphragm

GRADE II **A**

moderately diffuse increase in

(Moder- fine echoes in the hepatic pa re) renchvrna: slightly impaired vi ualization of intra hepatic vessel borders and diaphragm.

GRADE III **A** severe increase in fine echoes (Severe) in the hepatic parenchyma:

poor or no visualization of intra hepatic vessel borders. diaphragm. and posterior

6. A focal area of fatty infiltration tends to be hyper echoic and lobular with angulated and geometric or interdigitating margins separating it from the normal liver tissue and without displacement of the intra hepatic blood vessels In non uniform, fatty infiltration, the normal liver tissue is seen as hypo echoic areas within the hyper echoic fatty liver tissue. In such cases, the normal hypo echoic liver tissue may mimic malignancy or inflammation. (Carlo A.et.al1993).

2-5-17-5-4 Diagnosis of fatty liver:

Most case is discovered by chance when testing for some problem.

Blood test – measure liver foundation and lipid profile.

Imaging ultra sound and CT(Liver Biopsy) (www.m.webmd.com/fatty liver).

2-7 Total protein reference value in pediatric: (g/l)

Age	Male	Female
31 day	4.1_6.3	4.2_6.2
1-6 month	4.7_6.7	4.4_6.4
6month -1year	5.5_7	5.6_7.9
1-18 year	5.7_8	5.7_8

(text book of clinical chemistry, 1981, Norbet W,1996)

Differential Diagnosis

1. Cirrhosis
2. Hepatitis
3. Diffuse metastatic disease
4. Glycogen storage disease
5. Miliary tuberculosis. .(Carlo A.et.al1993).

2-5-17-5-5 Indications for Liver ultrasound

1. Enlarged liver/hepatomegaly.
2. Suspected liver abscess.
3. Jaundice .
4. Abdominal trauma.
5. Ascites.
6. Suspected metastases in liver.
7. Suspected liver mass.
8. Right upper abdominal pain. (Elisabetta , et.al1999)

2-5-17-5-6 Preparation for abdominal ultrasound

2-5-17-5-6-1 Preparation of the patient:

The patient should take nothing by mouth for 6-8 hours preceding the examination. If fluid is essential to prevent dehydration, only water should be given. If the symptoms are acute, proceed with the examination. I3nfants clinical condition permitting should be given nothing mouth for 3 hours preceding the examination.

In many patients, additional information can be obtained from an antero-posterior supine radiograph of the abdomen. If there is acute pain, a radiograph should also be taken with the patient erect and must include the diaphragm to exclude subphrenic air from a perforated viscus (Elisabetta , et.al1999).

2-5-17-5-6-2 Position of the patient:

The patient lies supine. Apply coupling agent liberally, first over the right upper abdomen, then over the rest of the abdomen as the examination proceeds

2-5-17-5-6-3 Choice of transducer

For adults use a 3.5 MHz transducer. For children or thin adults use a 5 MHz transducer

2-5-17-5-6-4 Setting the correct gain

The gain setting should allow the diaphragm to be clearly seen; the liver (when normal) should appear homogeneous throughout its depth. It should be possible to see clearly the normal tubular structures (the portal veins with bright edges and the hepatic veins without bright edges, Hepatic arteries and bile ducts are not seen unless dilated. Before scanning a specific area, should asked the patient to take a deep breath and hold it in patient breathes in angle transducer patient holds breath in Liver (Elisabetta , et.al1999)

2-5-17-5-6-5 Scanning technique

Scanning should be in sagittal, transverse and oblique planes, including scans through the intercostal and subcostal spaces. Scanning should be done with a slow rocking movement of the transducer in all planes to obtain the best visualization of the whole liver. It is difficult to measure accurately the overall size of the liver. In the mid-clavicular line , the longitudinal measurement form the diaphragm to the lower edge of the liver is usually less than 14cm in an adult, but there is considerable variation.

The normal liver parenchyma appears homogeneous, interrupted by the portal vein and its branches which are seen as linear tubular structures with reflective walls. The thinner hepatic veins are non-reflective. In a normal liver, it should be possible to follow the hepatic veins to their confluence with the inferior vena cava. Hepatic veins can be made to dilate when the patient performs the Valsalva manoeuvre (forced expiration against a closed mouth arid nose). The vena cava may be seen in the liver and may vary with respiration. The aorta may be identified as a pulsatile tubular structure behind and medial to the liver.

Oblique (upper) and transverse (lower) scans of the liver showing the portal and hepatic veins and the inferior vena cava .

Two longitudinal scans at slightly different angles showing the inferior vena cava, the hepatic veins and the bright (echogenic) walls of the portal veins.

The falciform ligament will be seen as a hyperechogenic structure just to the right of the midline in the transverse plane.

Transverse scan: the fissure of the ligamentum teres and the falciform ligament .

As well as the right and left lobes of the liver, it is also important to recognize the caudate lobe, limited posteriorly by the inferior vena cava and separated antero-superiorly from the left lobe of the liver by a highly

reflective line. It is limited inferiorly by the proximal left portal vein. The caudate lobe must be identified because it may be mistaken for a mass

Transverse scan: the caudate lobe of the liver and the fissure of the ligamentumvenosum.

The gallbladder and the right kidney must also be identified. The gallbladder will appear on a longitudinal scan as an echo-free, pear- shaped structure. (Elisabetta , et.al1999).

Sonographic assessment of the normal limits and percentile curves of liver, spleen and kidney dimensions in healthy Children's .

2-8 Longitudinal Length of Liver Versus Body Weight

	Length , mm				**95% CIM**	
Body weight, Kg	**Mean**	**Minimum**	**Maximum**	**SD**	**Lower Bound**	**Upper Bound**
20	105	76	164	14	103	108
30	112	84	146	13	110	114
40	116	83	149	12	114	118
50	119	83	161	15	116	121
60	123	95	165	14	120	126

2-9 Longitudinal Length of spleen Versus Body Weight

	Length , mm				**95% CIM**	
Body weight, Kg	**Mean**	**Minimum**	**Maximum**	**SD**	**Lower Bound**	**Upper Bound**
20	78	52	100	9	76	77
30	80	52	110	9	78	87
40	83	50	135	18	81	83
50	86	61	130	13	86	83
60	91	71	114	12	89	94

2-10 Longitudinal Length of Right Kidney Versus Body Weight

	Length , mm				95% CIM	
Body weight, Kg	**Mean**	**Minimum**	**Maximum**	**SD**	**Lower Bound**	**Upper Bound**
20	81	64	121	8	80	83
30	87	68	111	7	86	88
40	89	72	106	6	88	90
50	93	60	130	8	91	94
60	98	82	115	7	96	100

2-11 Longitudinal Length of Right Kidney Versus Body Weight

	Length , mm				95% CIM	
Body weight, Kg	**Mean**	**Minimum**	**Maximum**	**SD**	**Lower Bound**	**Upper Bound**
20	83	62	119	8	81	84
30	86	67	110	8	85	88
40	90	69	120	8	89	92
50	64	131	131	9	92	95
60	98	80	123	9	96	100

2-12 Correlations of Organ Dimensions With body Mass Index , Body Surface Area, Weight ,

Parameter	**Age**	**Liver**	**Spleen**	**Right kidney**	**Left Kidney**
BSA	0.820	0.351	0.341	0.582	0.500
BMI	0.268	0.278	0.277	0.277	0.301
Weight	0.678	0.388	0.390	0.552	0.511
Height	0.828	0.351	0.338	0.581	0.415
Age		0.225	0.314	0.488	0.415
Liver				0.385	0.293
Spleen				0.325	0.355
Right Kidney					0.703

2-13 Covariance and Variance of the correlation Estimates of Dimension with the body Parameters.

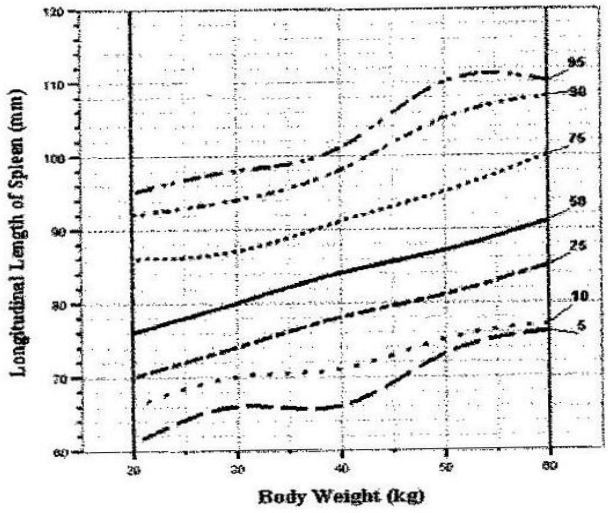
Parameter	Liver	Spleen	Right Kidney	Left Kidney
BSA	0.000	0.000	0.000	0.000
BMI	12.177	-2.707	9.034	10.351
Weight	65.814	20.057	10.069	18.514
Height	12.597	-2.151	12.927	11.033
Age	17.229	5.504	2.861	0.361

2-14 Normal Spleenic Size (length of spleen (cm)

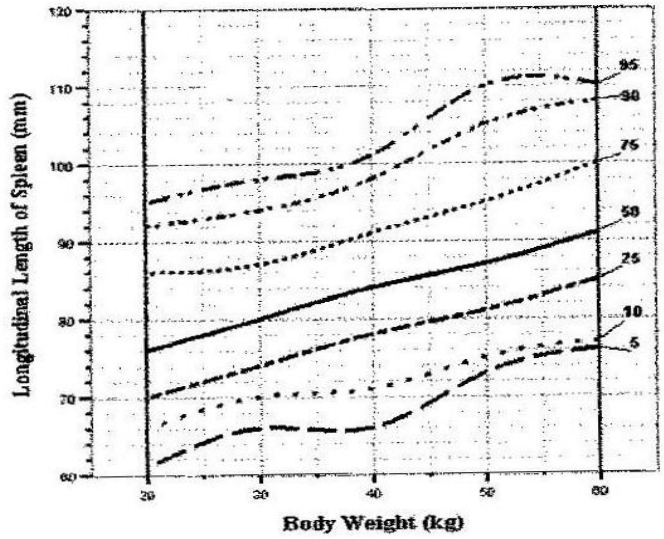
Ages	10^{th} percentile	Medium	90^{th} percentile
0-3month	3.3	4.5	5.8
3-6 month	4.9	5.3	6.4
6-12 month	5.2	6.2	6.8
1-2 year	5.4	6.9	7.5
2-4 year	6.4	7.4	8.6
4-6 year	6.9	7.8	8.8
6-8 year	7	8.2	9.6
8-10 year	7.9	9.2	10.5
10-12 year	8.6	9.9	10.9
12-15 year	8.7	10.1	11.4
Female	9	10	11.7
male	10.1	11.2	12.6

(How, why and when for pediatric,2005) .

2-6 Percentile Curves of longitudinal length of the liver versus body weight .



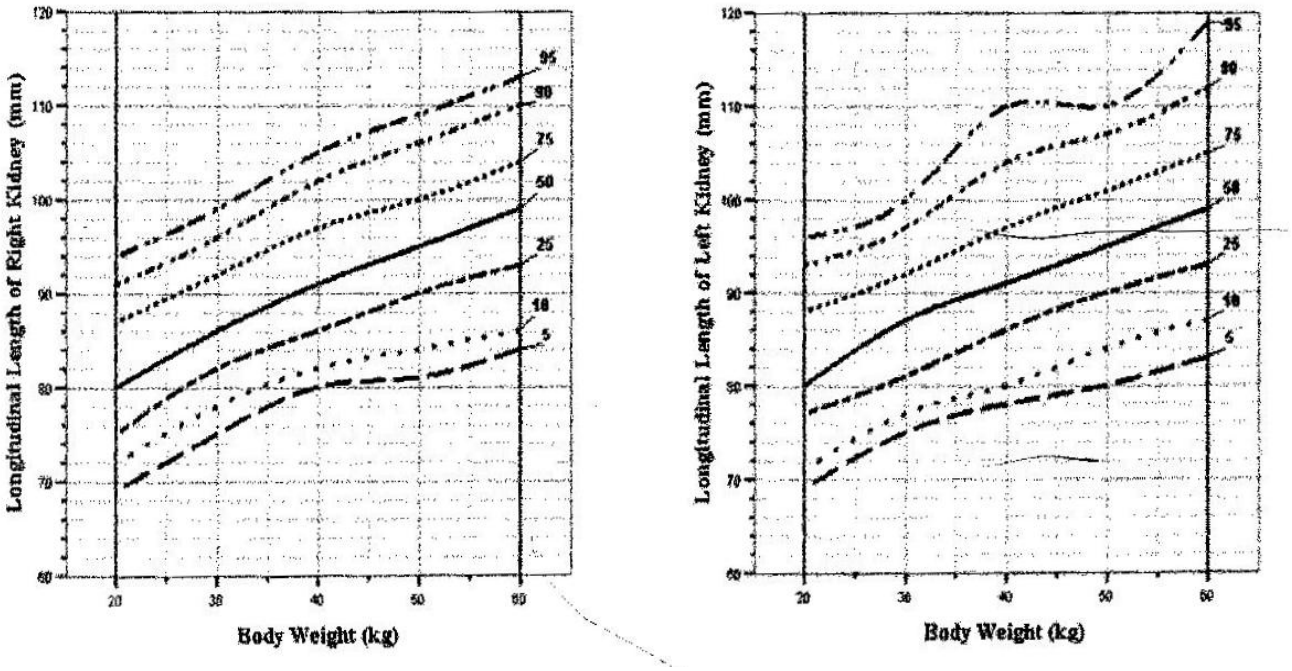
2-7 Percentile Curves of longitudinal length of the spleen versus body weight .



Ultrasound Med 2005

2-8 (A) Percentile Curves of longitudinal length of the Right kidney versus body weight . (B) Percentile Curves of Longitudinal Length of the kidney body weight .

(A) **(B)**



Ultrasound Med 2005

2- 6 Previous studies

El-hood MA,etal,2005 had studied to assess the pancreatic head size and exocrine pancreatic functions, namely serum amylase and lipase, in Protein Energy Malnutrition and subtypes and correlate any defect present with the various clinical and laboratory data of the Protein Energy Malnutrition patients with special emphasis on the affect of nutritional rehabilitation. (El-hood MA,etal were 73-467:(4)59:Apr 2005.Eur j clin Nutr).

while this study about assessment the liver (anatomy, function and ultrasound texture).

JF Doherty, etal had Studied for Children with oedematous malnutrition had significantly greater hepatic steatosis than non-oedematous children at admission and extent is not necessarily related to degree of hepatomegaly and accumulated lipid is only slowly mobilized this is study was done by JF Doherty, etal.(JF Doherty,EJ Adam,GE Griffin and MH Golden) .my study included measurement of liver length by using ultrasound and total liver protein by using liver function test.

Agostino Colli,etal,2003 was researched was about accuracy of ultrasound signs for assessment of the degree of liver fibrosis, they should that ultrasound determination of liver surface nodulatrity is an accurate method for identifying the sever liver fibrosis or cirrhosis.

(www.sld.cu/../el-ultrasonido-en-el-diag..by Acolli-2003) .

Agarwal MB,etal,1981 were measurement the affect of malnutrition on iron metabolism cases of kwashiorkor (had maximum decrease in iron binding capacity) while those with marasmus had the minimum.(www.jpg monline.com/article asp?by MB Agarwal-1981).

P kehoe,etal,2001 assessed the level of plasma corticosterone, dopamine, serotonin and metabolites in hypothalamus and hippocampus in study title (affects of prenatal protein malnutrition and neonatal stress on central nervous system responsiveness).(P/kehoe,k mallinson, bronzine-development Brain...2001,Elsevier.

Chapter Three

Materials and Methods

3-1 Type of study:

This is a descriptive and analytic study

3-2 Place and time of study:

This study was performed at Radiology department of Basheir and Mohmmed-Elameen hospital for pediatric in Khartoum state, in period from (April-2014 to September -2014).

3-3 Study sample:

This study included 40 subjects divided in to two groups marasmus group (27 patients) (6female – 21 male) and kwashiorkor (13 patients). (6 female - 7 male) study cases were selected from patient referred to ultrasound (28male and 12female).

3-4 Study variables:

The variable that were collected from each subject included ;gender(28male and 12female),ages between (7 to 61month),lengths between (70 to 120 centimeters),weights between (1to11kilograms),clinical features(fever,diarrhea,vomiting,swelling,infection,cough,down syndrom,weight loss and abdmanial distention),ultrasound finding(liver ,spleen length,gall bladder,bilary tree and liver texture),urine general test(protein in urine) and Liver function test(protein in liver).

3-5 Data collection:

The data were collected by account the number of study variables in master data table (appendix). Concern by physician and sonologist in mohammed elameen hospital for pediatrics.

3-6 Data Analysis:

The data were analysies by using SPSS program, variables using discruptive tables ,frequency ,percentage distribution tables,cross tabulation between the varibles and then all data were presentation in graph as bar graph.The degree of significant was tested using one way anova.

3-7 Materiles Used:

Ultrasound machine (ESAOTE Pie Medical Aquila-Japanese company - 3.5_5 MHZ).Thermeral ultrasound paper,ultrasound gel,printer and Medical couch.

3-8 Ultrasound technique:

Pateint lied supine on couch.applyied ultrasound gel on the area under exam,adjusted the gain, scan was done by slow rocking movement of the tranduser in Longitudinal plane,Transevers planse,Obliques,subcostal and intercostals. to visualization whole liver. Measurement the liver (at midile of claviculer line, longitudinal measurement from the diaphragm to the lower edge of the liver), evaluated GB and billiary tree, evaluted and measurement the spleen(from Inner surface to outer).Scaned other abdominal organ to area of symphsis. the results were reported and printed the image, clean the area of exam by medical cotton.

3-9 Other tools:

Medical cotton.

Meter for measure height of pt(from vertex to feet).

Weight measuring for measure pt weight.

Medical syringe and lab containers to get blood sample for Liver function test and urine general (protein level in urine which included 2 catagre normal and abnormal.

Chapter Four

Results

This Chapter Dealt with the data obtained from 40 patients affected with Marasmus and kwashiorkor ,in both genders, the patients data were collected and studied including age and weight, as well as ultra sonographic findings including liver length, spleen length ,liver texture (as normal, mild fatty, fatty liver) and gall bladder and biliary tree . The Laboratory Findings were also been evaluated including liver protein, protein urea as well as the clinical findings, all were evaluated. The clinical findings including fever, Down's syndrome, vomiting swelling, infection, weight loss, abdominal distention

Table 4.1 The Descriptive Statistics of the Sample affected with Marasmus and Kwashiorkor

Descriptive Statistics					
	N	Minimum	Maximum	Mean	Std. Deviation
Age (MO)	40	7.00	60.00	22.68	±10.34
Weight (kg)	40	3.00	10.00	6.56	±1.62
Liver Length (cm)	40	5.00	11.00	8.17	±1.55
Spleen Length (cm)	40	4.30	7.70	5.98	±1.03
Liver Protein (G/dl)	40	2.50	8.00	6.11	±1.91
Protein Urea	40	0.00	1.00	0.58	±0.50

Table 4.2The Diagnosis Classes of Marasmus and Kwashiorkor, Frequency and Percentages

Diagnosis			
		Frequency	Percentage
Class	Marasmas	27	67.5
	Kwashiorkor	13	32.5
	Total	40	100.0

Table 4.3 Clinical Findings, Frequency and Percentages

Clinical Findings			
		Frequency	Percentages%
Clinical Findings	D+V	18	45.0
	S	16	40.0
	Fever	5	12.5
	Down	1	2.5
	Total	40	100.0

Table 4.4 Age Classes Frequency and Percentages

	Month	Frequency	Percentages%
Age Classes	7-17	10	25
	18-28	20	50.0
	29-39	9	22.5
	40-50	0	0
	51-61	1	2.5
	Total	40	100.0

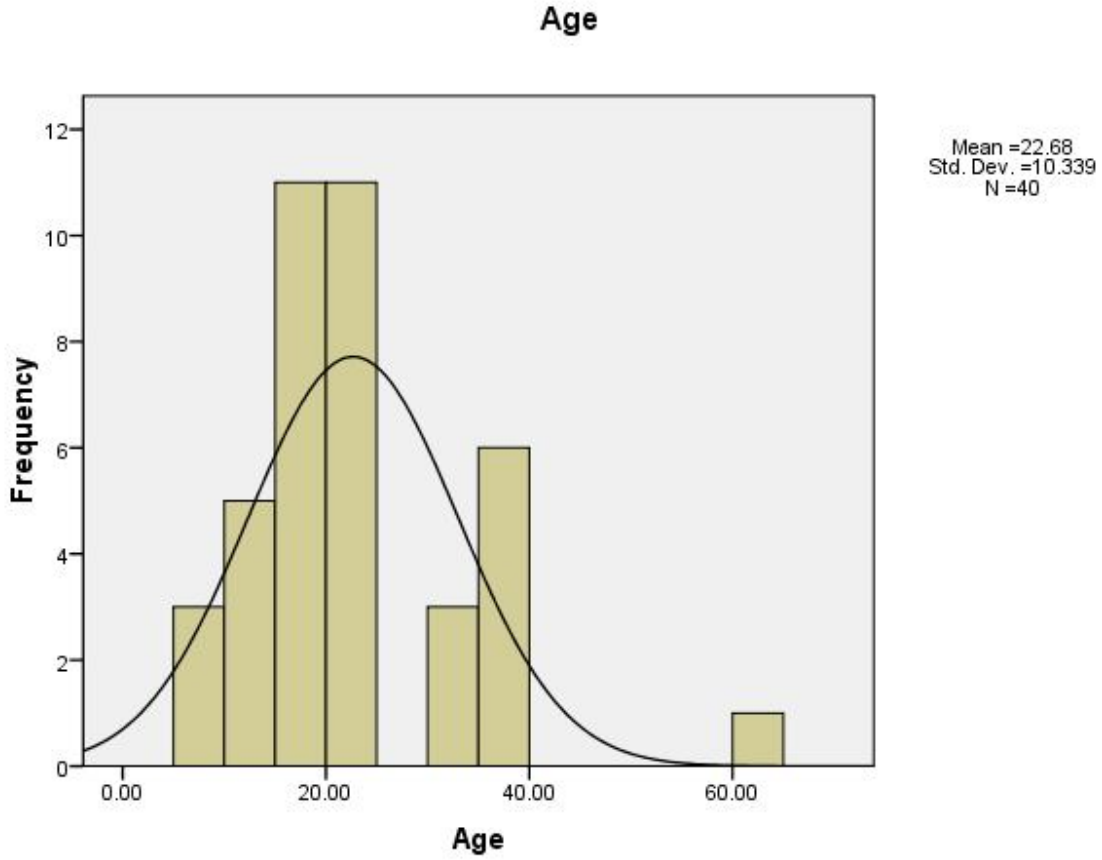


Figure 4.1 Fitted Curve of Age, Mean and Standard Deviation.

Table 4.5 Cross tabulation between Gender of the patients affected with Kwashkour and Marasmus and Diagnosis

Gender * Diagnosis Cross tabulation				
		Diagnosis		Total
		Marasmas	Kwashiorkor	
Gender	Female	6	6	12
	Male	21	7	28
Total		27	13	40

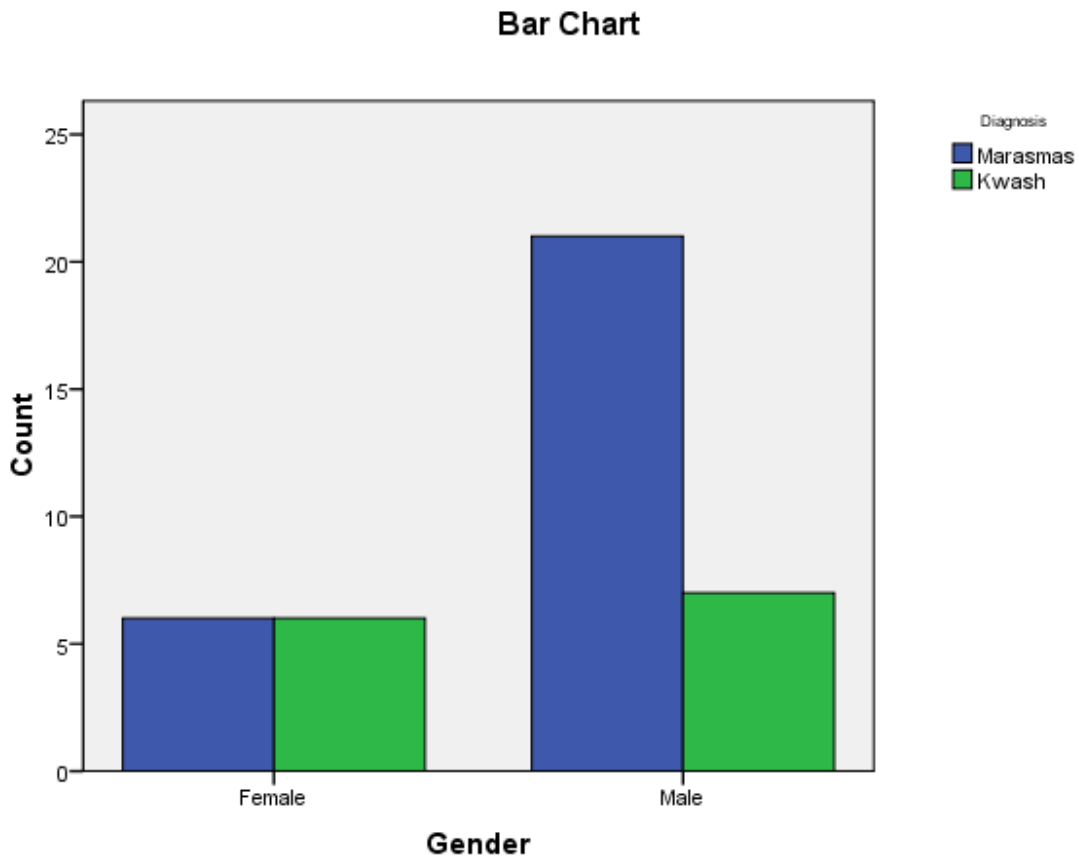


Figure 4.2 Diagnosis according to gender, frequency and percentages

Table 4.6 Cross tabulation between Ages of the patients affected with Kwashkour and Marasmus and Diagnosis

Age * Diagnosis Cross tabulation				
		Diagnosis		Total
		Marasmus	Kwashiorkor	
Age/ month	7-17	8	2	10
	18-28	12	8	20
	29-39	6	3	9
	40-50	0	0	0
	51-61	1	0	1
Total		27	13	40

Table 4.7Cross tabulation between Weight of the patients affected with Kwashkour and Marasmus and Diagnosis

Weight * Diagnosis Cross tabulation				
		Diagnosis		Total
		Marasmus	Kwashiorkor	
Weight/ Kg	3-5	6	3	9
	6-8	18	8	26
	9-11	3	2	5
Total		27	13	40

Table 4.8 Cross tabulation between Liver Length of the patients affected with Kwashiorkor and Marasmus and Diagnosis

Liver Length * Diagnosis Cross tabulation				
		Diagnosis		Total
		Marasmus	Kwashiorkor	
Liver Length (cm)	5-7	9	4	13
	8-10	18	8	26
	11-13	0	1	1
Total		27	13	40

Table 4.9 Cross tabulation between Spleen Length of the patients affected with Kwashkour and Marasmus and Diagnosis

Spleen Length * Diagnosis Cross tabulation				
		Diagnosis		Total
		Marasmas	Kwashiorkor	
Spleen Length (cm)	4.3-5.4	10	5	15
	6.4-7.5	15	7	22
	>7.5	2	1	3
Total		27	13	40

Table 4.10Cross tabulation between Liver Protiensof the patients affected with Kwashkour and Marasmus and Diagnosis

Liver Proteins * Diagnosis Cross tabulation				
		Diagnosis		Total
		Marasmas	Kwashiorkor	
Liver Proteins (G/dl)	2.5-4.6	1	11	12
	5.6-7.7	15	2	17
	>7.7	11	0	11
Total		27	13	40

Table 4.11Cross tabulation between Proteinurea of the patients affected with Kwashiorkor and Marasmus and Diagnosis

Protein Urea * Diagnosis Cross tabulation				
		Diagnosis		Total
		Marasmas	Kwashiorkor	
Proteinurea	Nil	11	6	17
	Present	16	7	23
Total		27	13	40

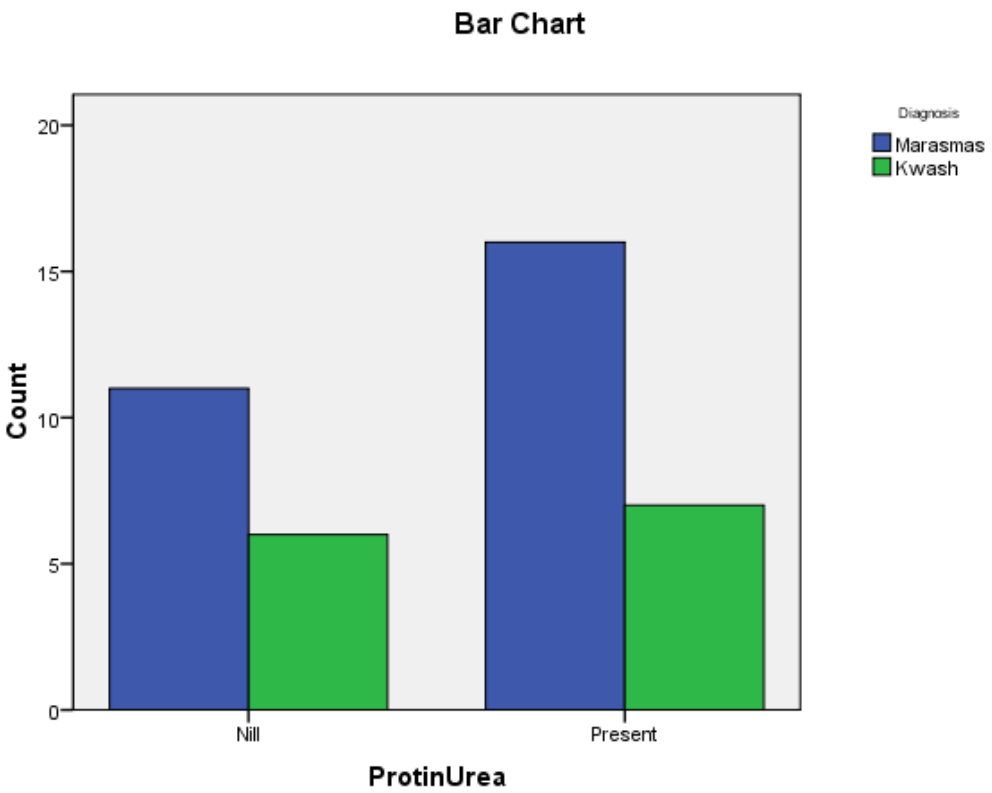


Figure 4.3 Diagnosis according to Proteins, frequency and percentages

Table 4.12 Cross tabulation between Clinical Findings of the patients affected with Kwashkour and Marasmus and Diagnosis

Clinical Findings * Diagnosis Cross tabulation				
		Diagnosis		Total
		Marasmas	Kwashiorkor	
Clinical Findings	D+V	18	0	18
	S	3	13	16
	Fever	5	0	5
	Down's Syndrome	1	0	1
Total		27	13	40

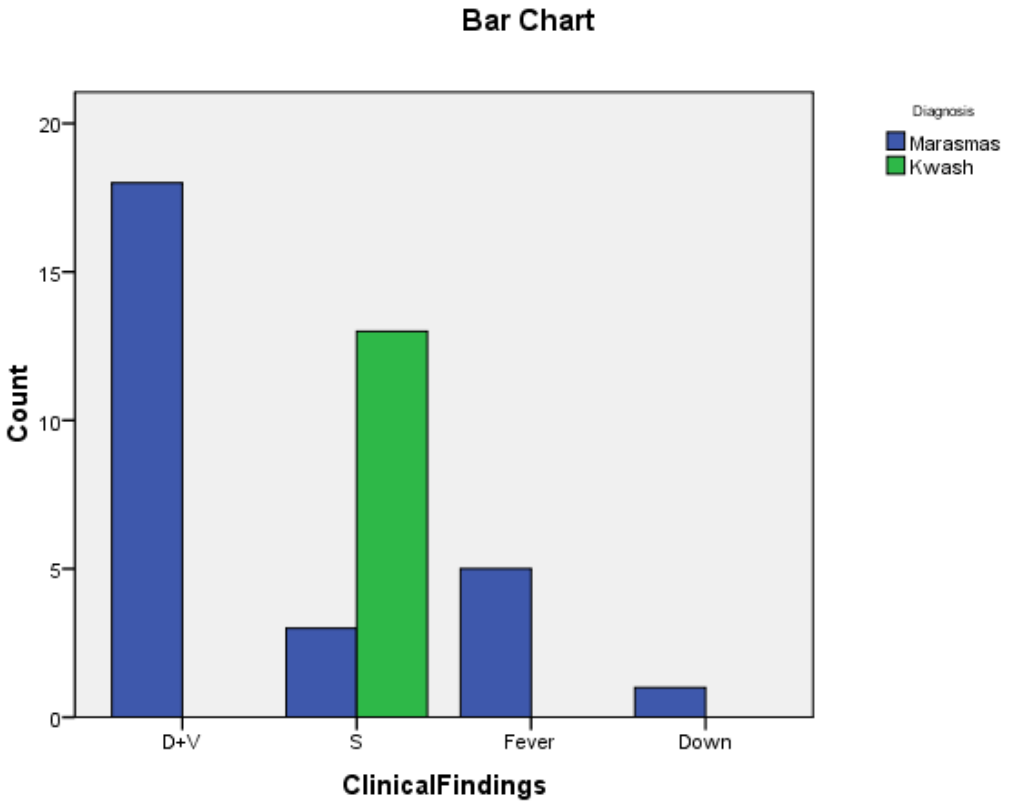


Figure 4.4 Diagnosis according to Clinical Findings, frequency and percentages

Table 4.13 Cross tabulation between Liver Texture of the patients affected with Kwashiorkor and Marasmus and Diagnosis

Liver Texture * Diagnosis Cross tabulation				
		Diagnosis		Total
		Marasmas	Kwashiorkor	
Liver Texture	Normal	13	5	18
	Mild Fatty	8	6	14
	Fatty	5	3	8
Total		26	14	40

Table 4.14 ONEWAY ANOVA table of analyses for Age, Weight, Gender, Clinical Findings, Spleen Length, Liver Length, Liver Protein, Protein Urea, Correlated with Diagnosis

ANOVA TABLE				
		Sum of Squares	Mean Square	Sig.
Age	Between Groups	.359	.359	.955
	Within Groups	4168.416	109.695	
	Total	4168.775		
Weight	Between Groups	.017	.017	.937
	Within Groups	101.802	2.679	
	Total	101.819		
Gender	Between Groups	.503	.503	.128
	Within Groups	7.897	.208	
	Total	8.400		
Clinical Findings	Between Groups	1.456	1.456	.125
	Within Groups	22.519	.593	
	Total	23.975		
Spleen Length	Between Groups	.963	.963	.349
	Within Groups	40.646	1.070	
	Total	41.610		
Liver Length	Between Groups	2.469	2.469	.317
	Within Groups	91.422	2.406	
	Total	93.891		
Liver Proteins	Between Groups	96.254	96.254	.000
	Within Groups	45.410	1.195	
	Total	141.664		
Protein Urea	Between Groups	.026	.026	.753
	Within Groups	9.749	.257	
	Total	9.775		

The Relation Is Significant At p-Value =0.05

Chapter Five

Chapter Five

Ultrasound is the method of choice for the routine of liver disease (Elsabetta 1999).

5-1- Discussion

The diagnosis of liver disease has been complicated so beside radiological investigation which guide us to some types of diagnosis a number of imaging modalities including NM,MRI,CT and Ultra sound have been used in attempt to provide apathophysiologically related.
The study found that 67.5%marsmacic and 32%kwashiorkor.

Clinical finding's in cases in the study showed that diarrihea and vomiting 45%,swealing 40%,fever12.5% and down syndrom2.5% There is no significant related to diagnosis of protein energy malnutrition but the PEM was occur commonly associated to infection and diarriahea and similar in kwashiorkor and marasmus as see in the study of Jamal Nasir,1963 .

Gender distribution showed the majority of the sample under study were 28 male(7 kwashiorkor and 11marsmaic patients) and female (6kwashikor and 6marsmaic) there is no direct relation between gender and PEM same finding as study of Jamal Nasir,1963 .

Age distribution the population divided into five age group below 8 month , between(7_17 month), between (18_28 month), between(29_39 month), between(40_50 month), between(51_61month).the most effected age group is between (18_28month) (50%) which have 12 marasmic and 8 kwashiorkor .the marasmic occur in early infancy while kwashiorkor tended older than marasmus similar to result of Jamal Nasir,1963 .

patients weights the cases in this study the weight ranged from (3-11kg) divided into groups between (3-5 kg), between(6-8kg) and between (9_11kg) .the most effected group between(6-8kg) and minimum between (9_11 kg) .

Liver length in ultrasound finding affected with kwashiorkor and marasmus divided as liver length between (5_7cm,8_16cm) and between(11_13cm) the affected group between(8_10cm) and minimum between(11_13cm) all results are within normal liver length (reference value up to 9-13 cm in pediatric patients , The hepatomagaly may be occur in kwashiorkor cases while the liver size was normal in marasmus cases similar to results in William D, 2005 study.

Also the spleen length of patient affected with kwashiorkor and marasmus were grouped between (4.3_5.4cm) ,between (6.4_7.5cm) and 7.5cm the most affectence between(6.4_7.5cm) and minimum is7.5cm. all results are within normal spleen length according formula :

$\frac{1}{3}$ x 7.5 + ages (William D 2005).

Assessed liver protein it has been found that between(2.5_4.6), and between (5.6_7.7) and >7.7.the highest value (5.6_7.7) and lowest value > 7.7as Norbet w,1996 recommended study.

Liver protein test is significant in cases of kwashiorkor the total protein was decrease while in marasmus cases total protein was with in normal range similar to result (jamal nasir,1963).

Proteinurea it has been found that nil protein urea in 17 cases (11marsmaci and 7 kwashiorkor) and 23 present cases were (16 marasmic and kwashiorkor) is insignificant value to diagnosis of PEM.

The majority of study that's affectnce of P.E.M on liver texture found there is 18 cases were normal liver texture on 13 marasmic and 5 Kwashiorkor , 14 mild fatty liver texture that 8 marasmic and 6 Kwashiorkor . 8 cases were fatty liver texture 5 marasmus and 3 kwashiorkor .

The a nova table analysis was founded that there is no significant co relation for age , weight , gender ,clinical findings , spleen length , liver length and protein urea with diagnosis of protein energy malnutrition and the relation of liver protein is significant with protein energy malnutrition. (The relation significant at P-value 0.000).

5-2-Conclusion:

The current study aimed to study the liver for marasmus and kwashiorkor patient by Ultrasound .

marasmic patient most frequently than kwashiorkor .

The result by ultrasound study under going appearance of fatty liver , mild fatty liver and normal liver texture and bilary system . the laboratory result that supporting the minimal value of protein on Liver Function test on kwashiorkor patient and marasmas patient.

Ultra sound investigation alone is mostly not suitable in order to diagnose of fatty liver in malnutrition .consequently L.F.T is essential using for evaluation protein level and fatty liver may be inadequate.

5-3 Recommendation:

Regarding the results the Ultrasound Scanning for abdominal investigate the liver, spleen and abdominal organ are essential for evaluate of organ sizes .

Lab test is also necessary for approputie diagnosis for kowash and mrasmus on results are significant.

Both investigation should be applied for all patients.

For further studies:

Advice to family planning to ensure adequate spacing of childbirth , engcourage breast feeding to last as possible and educate people to supplement the dial of their children with animal protein or vegetables protein and green vegetables .

Advice the medical field to used the ultrasound with lab investigation to diagnosis the kowash and marasmus cases.

Advice to increasing the sample size in cases of kowash and marasmus and considered the detail information about the socioeconomic statuses .

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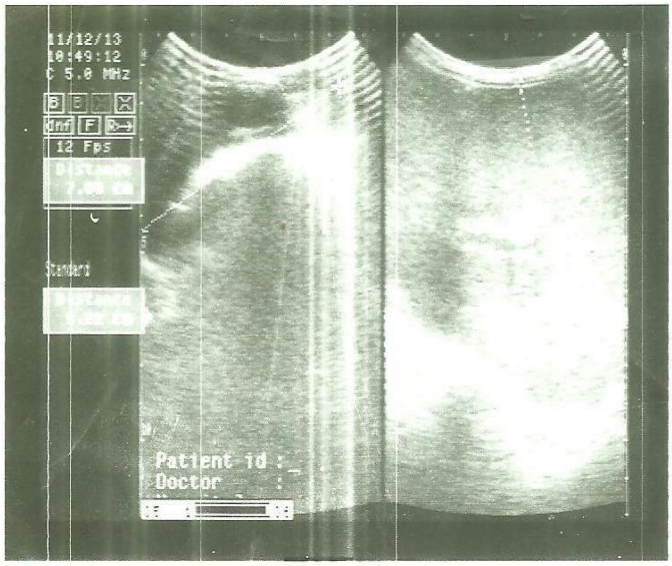
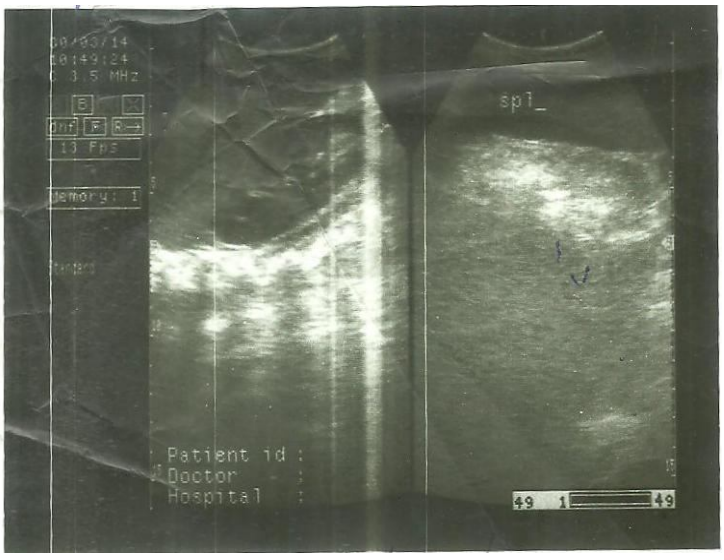
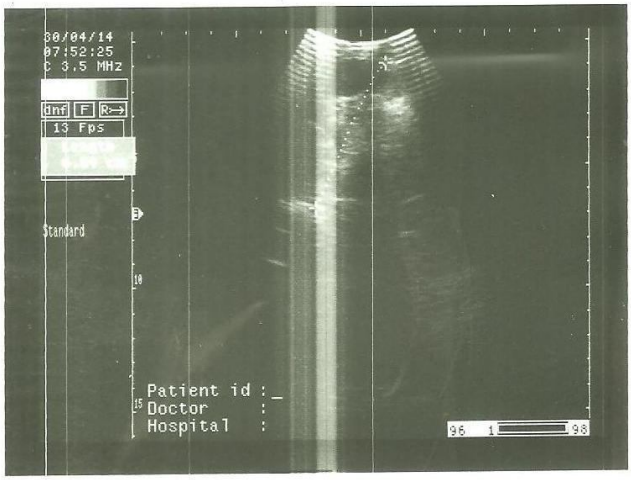
Appendices

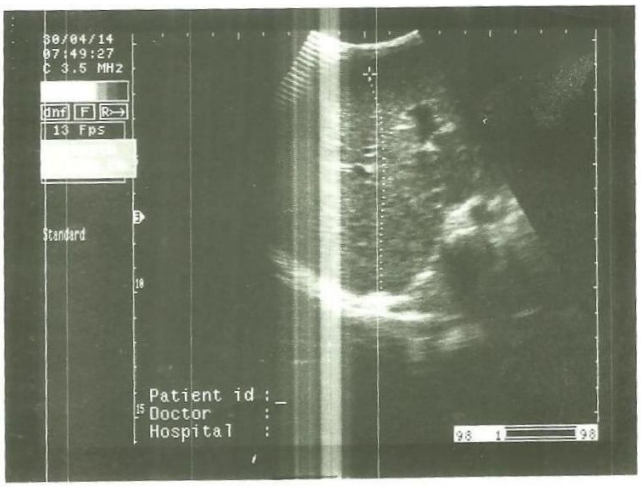


Male one years old Complain of diarrhea and vomiting after Test Diagnosis amrasmus patient, ultrasound showed that fatty liver, liver length 9.22cm and spleen length 7 cm.

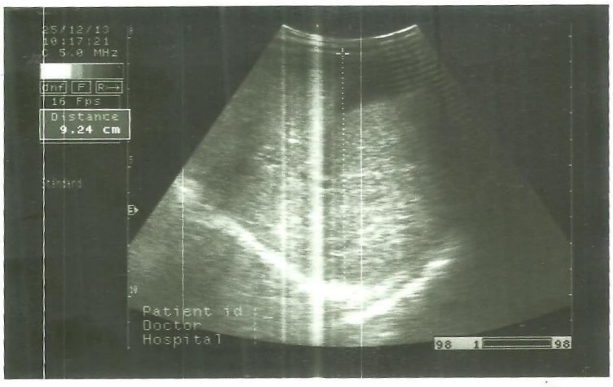


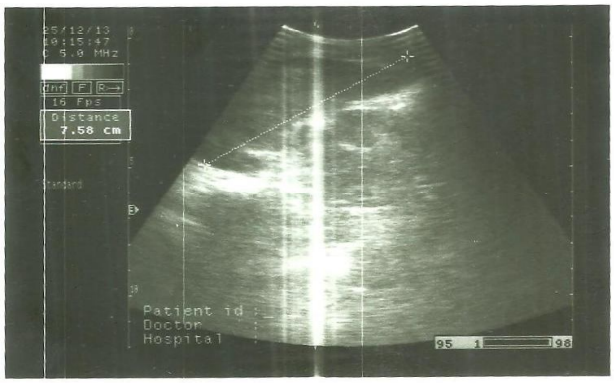
Male 30 month old known case of kwashiorkor patient , ultra sound showed normal liver texture and spleen length 7cm .

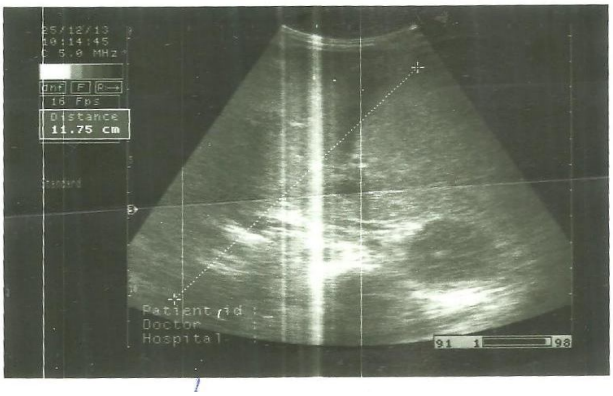




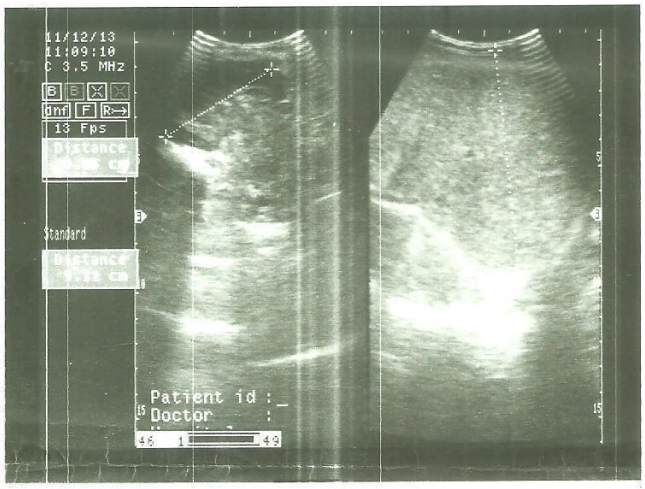
Male 3 years old known's as marasmus patient , ultra sound showed normal liver texture , liver length 9.26cm and spleen length 6.54cm .



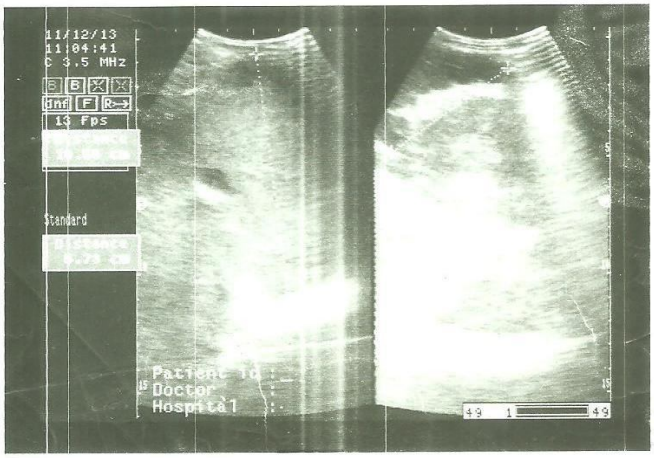




Male : 30 Month old complain of swelling Dignosis as kwashiorkor patient ultrasound showed normal liver texture , liver length 11.75cm and spleen length 7.5cm .



Female : 2 years old Dignosis complain of fever and vomiting Dignosis as marasmus patient ultrasound showed normal liver texture , liver length 9.11cm and spleen length 4.95cm .



Female 3 years old complain of vomiting and Diagnosis as Marasmus patient ultrasound showed mild Fatty liver , liver 10.58cm and spleen length 5.73 cm.

بسم الله الرحمن الرحيم

جامعـــــة السودان للعلوم والتكنولوجيا

كلية الدراسات العليا والبحث العلمي

إستمارة تقييم الحالات الدراسية للكبد لمرضى المرازمس والكواش كور

No	Gender	Age MO	Weight (KG)	Length (CM)	Life with		Father Work	Liver Size (CM)	Liver texture			GB& Tree	Spleen Size (CM)	Clinical sign	Diagnose	Lab Test	
					Mot	Fa			Normal	Mild fatty	Fatty					Protein In Urine	Protein In Liver(g/dl
1	M	36	6.3	72	√	√	free	7.5			√	nor	6	F+DS	Mars	+	6.8
2	M	18	6.7	74	√	√	free	6.8	√			nor	5.1	F+CO	Mars	Nill	8
3	M	12	6.7	70	√	√	free	9	√			nor	7.4	D+V	Mars	+	7.8
4	M	18	4	70	√	√	free	9			√	nor	7.2	V+D	Mars	Nill	7.1
5	M	7	7	78	√	√	free	6.7	√			nor	4.9	V+D	Mars	Nill	7.6
6	M	24	5.5	70	√	√	free	8.4			√	nor	5.4	S+D	Kwas	++	3
7	M	24	6	110	√	√	free	9.1		√		nor	4.5	F+S	Mars	++	7.5
8	M	36	7	120	√	√	free	9.6		√		nor	5	S+F	Kwas	Nill	3.3
9	F	24	6.5	110	√	√	free	9.1		√		nor	4.5	S+D	Kwas	Nill	4.5
10	F	9	4	72	√	√	free	5		√		nor	6.5	V+D	Mars	Nill	8
11	M	36	7.5	100	√	√	free	6.3	√			nor	6.1	V+D	Mars	Nill	7.4
12	M	18	7	75	√	√	free	7	√			nor	6.3	S+I	Kwas	+	5.5
13	M	30	7.5	95	√	√	free	6.5		√		nor	5.3	V+D	Mars	Nill	7.9
14	F	36	7.9	98	√	√	free	5		√		nor	5.7	A+D	Mars	+	7.8
15	F	18	9.3	73	√	√	free	6				nor	4.5	S+D+I	Kwas	+	4
16	M	13	6.3	71	√	√	free	6.3	√			nor	5.2	S+D	Kwas	+	3.7
17	F	24	8	82	√	√	free	6.8	√			nor	5.7	S+D	Kwas	+	3
18	M	24	8.5	74	√	√	free	9	√			nor	7.7	S+D+I	Kwas	Nill	2.5
19	F	24	7.3	77	√	√	free	9.7	√			nor	7.3	F+W	Mars	+	6.9
20	F	10	5.5	79	√	√	free	9.4			√	nor	6.3	S+I	Kwas	Nill	4
21	M	24	5	100	√	√	free	8.2			√	nor	6.3	F+S	Kwas	+F	4.5
22	M	36	7.5	110	√	√	free	9.2			√	nor	6.5	D+V	Mars	Nill	5.3
23	M	18	9	76	√	√	free	9.2			√	nor	7.6	D+V	Mars	++	7.9
24	M	16	8.7	70	√	√	free	9.2		√		nor	7.5	V+D	Mars	Nill	7.8
25	M	18	5	75	√	√	free	11		√		nor	7.5	S+D	Kwas	+	3.6
26	M	24	6.5	85	√	√	free	9		√		nor	5.7	S+D+I	Kwas	Nill	5.7
27	F	30	7	73	√	√	free	9.7		√		nor	4.3	S+D	Kwas	Nill	3.5
28	F	30	4	71	√	√	√free	9.7		√		nor	5.8	S+I	Kwas	++	2.6

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29	M	12	4	75	√	√	free	7.7		√		nor	5.2	D+V	Mars	+++	4.9
30	M	16	6.5	82	√	√	free	8.4		√		nor	5.4	D+V+I	Mars	++	7.5
31	F	24	5	74	√	√	free	9.1				nor	4.9	F+D	Mars	++	7.6
32	F	18	8	77	√	√	free	10				nor	5.7	V+D	Mars	++	8
33	M	60	10	120	√	√	free	6.8	√			nor	5.7	D+V	Mars	++	7.5
34	M	12	3	100	√	√	free	9.2			√	nor	6.9	D+V	Mars	+++	7.6
35	M	36	7	100	√	√	free	9.6		√		nor	5	D+V	Mars	++	8
36	M	18	6.5	70	√	√	free	9	√			nor	7.4	V+F+I	Mars	++	8
37	F	8	4	72	√	√	free	5	√			nor	6.5	V+D	Mars	Nill	6.6
38	M	24	8	74	√	√	free	9	√			nor	7.7	V+D	Mars	Nill	7
39	M	24	6	100	√	√	free	9.1	√			nor	4.6	F+D	Mars	Nill	6.8
40	M	18	7	75	√	√	free	6.3	√			nor	6.3	I+D+V	Mars	+	7.8

Mo = month

Mot= mother

Fa= father

Nor =normal

KG =kilo gram

Cm =cent meter

G/dl = gram per dice litter

F =fever

DS =down syndrome

D =diarrhea

V =vomiting

S =swelling

I =infection

CO =cough

W =weight loss

A =abdominal distention

M =male

Kwas =kwashiorkor

Maras =marasmus