ULTRASONOGRAPHIC FINDINGS IN OBSTRUCTIVE JAUNDICE: THE ABILITY OF ULTRASOUND TO ACCURATELY DETERMINE THE SITE AND CAUSE OF OBSTRUCTION.

DISSERTATION

TO BE SUBMITTED IN PART FULFILMENT FOR THE DEGREE OF MASTER OF MEDICINE IN DIAGNOSTIC IMAGING AND RADIATION MEDICINE, UNIVERSITY OF NAIROBI

BY

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DECLARATION

I, Dr.Ngoseywe Kennedy declare that the work contained herein is my original idea and has not been presented at any other place to the best of my knowledge.

Signature.....Date.....15/12/08

APPROVAL BY SUPERVISOR

This research has been submitted with my approval as university supervisor.

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TABLE OF ABBREVIATIONS

ACE---------- ANGIOTENSIN CONVERTING ENZYME

AIC---------- AUTOIMMUNE CHOLANGITIS

AIDS---------- AQUIRED IMMUNE DEFICIENCY SYNDROME

AIH---------- ACUTE IMMUNE HEPATITIS

AMA---------- ANTIMICHONDRIAL ANTIBODIES

ANA---------- ANTINUCLEAR ANTIBODIES

BRIC---------- BENIGN RECCURENT INTRAHEPATIC CHOLANGITIS

CD---------- CLUSTER OF DIFFERENTIATION

CHD---------- COMMON HEPATIC DUCT

CBD---------- COMMON BILE DUCT

CTD---------- CONNECTVE TISSUE DISORDERS

CT---------- COMPUTED TOMOGRAPHY

DC---------- DIRECT CHOLANGIOGRAPHY

ERCP---------- ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY

FUO ---------- FEVER OF UKNOWN ORIGIN

GGT----------GAMMA GLUTAMYL TRANSFERASE

GH---------- GRANULOMATOUS DISEASE

GVHD----------GRAFT VERSUS HOST DISEASE

HLA---------- HUMAN LEUCOCYTE ANTIGEN

ICAMS---------- INTERCELLULAR ADHENSION MOLECULES

IL---------- INTERLEUKIN

KG---------- KILOGRAM

NSAID ----------NONSTEROIDAL ANTIINFLAMATORY DRUGS

PBC---------- PRIMARY BILIARY CIRHOSIS

PSC---------- PRIMARY SCLEROSING CHOLANGITIS

PTC---------- PERCUTANEUOS TRANSHEPATIC CHOLANGIOGRAPHY

TNF----------TUMOR NECROSIS FACTOR

TPN ----------TOTAL PARENTAL NUTRITION

UDCA---------- URSODEOXYCHOLIC ACID

UDP ----------URIDINE DIPHOSPHATE

U/S---------- ULTRASOUND

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ABSTRACT

Introduction

The ability of ultrasound to accurately distinguish obstructive jaundice from nonobstructive jaundice has made sonography the accepted screening procedure in the jaundiced patient. Determination of the anatomic site of obstruction and its cause is critical in the management of the jaundiced patient, whether traditional surgical therapies are contemplated or newer "nonsurgical therapies like" radiologic methods are instituted. Choosing the correct therapy and obtaining the best results from that therapy usually require a precise knowledge of anatomic detail and the nature and extent of the disease. The high accuracy and relative safety of direct cholangiography and newer imaging methods such as magnetic resonance cholangiopancreatography have set a high standard against which sonography must compete.

Objectives

The main objective was to evaluate the capacity of ultrasound in determining the site and cause of obstructive jaundice.

Study design

A descriptive prospective study.

Setting

Kenyatta National Hospital and University Of Nairobi radiology departments.

Subjects

All patients with obstructive jaundice who had undergone U/S examination and another diagnostic imaging test /surgery with which the results of U/S were compared.

Methods

A total of 40 patients with evidence of intrahepatic or extra hepatic obstruction were entered prospectively into the study from June 2007 to April 2008.

Results

The site of obstruction was **predicted in 26 patients** (sensitivity of 65% and specificity of 77%), **but** was indeterminate in 35% because of the inability to visualize the complete biliary tract. The cause of obstruction was correctly predicted **in 31 patients** (sensitivity of 78% and specificity of 72%) and was **indeterminate in 22%** ($p<0.05$).

Conclusion

Ultrasonography was accurate in differentiating obstructive from non obstructive jaundice but was non specific in assigning a definite cause of obstruction and in predicting the site of obstruction.

INTRODUCTION AND LITERATURE REVIEW

INTRODUCTION

Jaundice and pain are the most common presenting complaints in patients with hepatobiliary disease. Acute biliary tract diseases cause significant morbidity and mortality, and about 2% of all admissions to Hospital are for hepatobiliany diseases. Acute pancreatitis (0.54% of all admissions) and acute cholecystitis (0.48%) are the leading indications for hospitalization. Radiologic examinations play a major role in the diagnosis of many of these conditions (1).

The prevalence of jaundice varies with age and sex, newborns and older adults are most often affected. In the United Kingdom, the prevalence of jaundice is 3% and most of these subjects have Gilbert's syndrome (2).

The causes of jaundice also vary with age. Approximately 20 percent of term newborns develop jaundice in the first week of life, primarily because of immaturity of the hepatic conjugation process. Congenital abnormalities, hemolytic or bilirubin uptake disorders, and conjugation defects are also responsible for jaundice in infancy or childhood. Viral hepatitis A is the most frequent cause of jaundice among school-age children. Common duct stones, alcoholic liver disease and neoplastic jaundice occur in middle-aged and older patients (2).

In men jaundice is most likely to be due to cirrhosis, chronic hepatitis B, hepatoma, pancreatic cancer or sclerosing cholangitis (2). In contrast, women tend to have higher rates of common duct stones, primary biliary cirrhosis and carcinoma of the gallbladder (2).

Worldwide cholelithiasis or presence of gallstones is the most common cause of jaundice.Billiary tract tumors are uncommon but serious causes of posthepatic jaundice.Cholangiocarcinoma accounts for about 25% of hepatobilliary cancers and is associated with approximately 50% survival (2).

Ultrasonography is the imaging modality of first choice for evaluating obstructive jaundice. The diagnostic accuracy of U/S in differentiating obstructive from non obstructive jaundice is estimated to be high in the order of about 90 %(1).

A study by Harvey.Neiman and Richard Minter in 1977(3), in which a comparison of diagnostic accuracy of U/S and cholangiography found that recognition of billiary tract caliber by U/S was accomplished in 86% of cases and was most accurate in 89% of subjects with dilated intrahepatic ducts.

In the same study, the degree of confidence was also assessed and in 71% of cases the diagnosis was reported as definite. The etiology of the obstruction was suggested in 73% of the cases as compared to 95% by cholangiography.They concluded that billiary duct dilatation can be confidently recognized by U/S.

A study by C.A Muhletaler, Gerlock A J(4) in which U/S was compared to PTC in the diagnosis of obstructive jaundice found that U/S was not very accurate in diagnosing obstructive jaundice in the absence of dilated ducts. A total of 29 subjects were studied and obstructive jaundice with non dilated bile ducts was identified by PTC in nine of 29 jaundiced patients in whom the etiology of the jaundice was not clearly established by clinical or laboratory means and no dilated ducts were seen at sonography.

P.L Cooperberg, D Li (5) studied the accuracy of CHD size by U/S in the evaluation of extrabiliary obstruction. The authors evaluated the accuracy of using 4mm internal diameter as the upper limit of normal in evaluation of obstruction. Of 98 subjects with jaundice whose duct exceeded 4mm on U/S, obstruction was proved in 84. Fourteen patients had no radiogical or pathological evidence of obstruction, but 7 had undergone cholecystectomy and clinical evidence suggested another 6 had passed a stone before or after the ultrasound study. Of 72 patients with a duct greater than or equal to 4 mm, only one had obstruction; a mass blocked the right and left hepatic ducts. The sensitivity of the test was 99%, the specificity 87%. They thus concluded that if the common hepatic duct is more than 4mm in diameter on ultrasound, extra hepatic biliary obstruction is probably present.

C.Zemel, A B Zajko (6) studied retrospectively the role of sonography and transhepatic cholangoigraphy in the diagnosis of biliary complications after liver transplantation in 41 subjects. Abnormalities included bile duct stricture (26 cases), occluded internal biliary stent (six cases), common duct redundancy with resultant functional biliary obstruction (three cases), bile leak (three cases), choledocholithiasis (two cases), and an abscess in a cystic duct remnant (one case). Sonography was abnormal in 22 of the 41 cases (sensitivity, 54%). Bile duct dilatation was the positive sonographic finding in 19 (86%) of the 22 abnormal examinations. In the remaining 19 patients, sonography was normal. The authors concluded that sonography was not a reliable test for the early detection of biliary abnormalities after liver transplantation.

S.E Mitchell, A Clark (7) compared ultrasonography and computed tomography (CT) scan in the diagnosis of choledocholithiasis. Sonography correctly diagnosed nine of 49 patients with choledocholithiasis, a sensitivity rate of 18%. The accuracy rate for sonography was 19%; there were five false-positive examinations. CT correctly identified common duct stones in 26 of 30 patients, a sensitivity rate of 87%. The accuracy rate was 84%; there was

one false positive. The conclusion was that sonography is limited in its ability to image calculi in the distal common bile duct. CT is effective for imaging common duct stones and is superior to sonography for diagnosing this cause of biliary obstruction.

A study done in Italy by Renato Costi MD, Leopoldo Sarli (8) on the role of U/S assessment of the size and number of gallstones could identify patients at increased risk of having asymptomatic CBD stones. Ultrasonographic data for 300 consecutive patients undergoing laparoscopic cholecystectomy were analyzed. Patients were divided into a group in which multiple small (≤5 mm) or multiple variably sized (both ≤5 and >5 mm) gallbladder stones were present ("positive" stones) and a group with multiple large (>5 mm) or single gallbladder stones, considered "negative." The ultrasonographic description was compared with surgical findings; finally, the prevalence of asymptomatic common bile duct stones in the 2 groups was compared. Ultrasonographic classification of gallbladder stones was confirmed at surgery in 285 cases (95%). Asymptomatic common bile duct stones were diagnosed in 9.5% of patients with an ultrasonographic diagnosis of positive gallbladder stones and in only 2.3% of patients with a diagnosis of negative gallbladder stones ($P <$ .05). The conclusion was that ultrasonography is able to accurately show gallbladder stones; the appearance of multiple small and variably sized gallbladder stones represent a risk factor for synchronous asymptomatic common bile duct stones.

A study by RN Gibson, E Young (9) in which various modalities were compared for their capability to demonstrate the level and cause of obstruction. The level of obstruction was correctly indicated by US in 95% of patients and by CT in 90%, and the cause was correctly indicated by US in 88%, by CT in 63%, and by Direct cholangiography (DC) in 89%. In predicting tumor resectability, US was correct in 71% of patients, compared with 42% for CT, 58% for DC, and 25% for cholangiography. US therefore appear to be the single most useful modality in the evaluation bile duct obstruction.

Regarding the Kenyan situation, little data is available on the accuracy of U/S in the diagnosis of the cause and site of obstruction.

Wambugu M N, Okoth F A (10) carried out a prospective study at Kenyatta National Hospital between June 1987 and September 1988 to look at some aspects of obstructive jaundice in patients above 12 years of age. Screening for cases was done by use of abdominal U/S.A total of 20 cases (11females & 9 males) were diagnosed. The authors found that carcinoma of the head of the pancreas accounted for 55% of cases of obstruction, followed by gallstones 10%, hepatocellular carcinoma 10% and gall bladder tumor 10%.In this study the accuracy of U/S in determining the site of the obstruction was not assessed and also the age distribution of the various causes of obstruction was not determined.

In his dissertation for master of medicine in diagnostic radiology in 1988, Byarugaba S.D found that ultrasound had an accuracy of 98% in the detection of biliary cholelithiasis (11). Stephen.p.Honickman and Peter.R.Mueller carried out a prospective study of the ability of U/S to accurately determine the site and etiology of biliary obstruction in62 patients. The site of obstruction was predicted in 27% of patients and was indeterminate in 73%.The cause was correctly predicted in 23% of patients and was indeterminate in 76%.The authors concluded that U/S was not accurate in predicting the site and cause of obstruction[12].

Anatomy of the hepatobilary system

BILIARY TRACT

Although biliary anatomy follows a stereotypical template, variations in the anatomy of the ducts, gallbladder and hepatic arteries are very common; indeed, it is difficult to find an individual patient who has a completely orthodox hepatobiliary system. Knowledge of these variations is no mere academic pursuit: it has important implications for the investigation, diagnosis and treatment of disease in the biliary system.

An understanding of orthodox anatomy is also important. Failure to recognize that a segmental bile duct remains unopacified on contrast studies, for instance, may allow a significant lesion in that segment to remain undisclosed or give a false impression of the site or extent of a lesion.

The intrahepatic components of bile duct, hepatic artery and portal vein (portal triad) run together and are arranged on the basis of the segmental anatomy of the liver. The extrahepatic components of these structures are more loosely associated (13).

The intrahepatic ducts are not in a fixed relation to the portal veins within the portal triad. They may be anterior, posterior to the vein or tortuous about it. The extrahepatic biliary tract consists of: Right, left and common hepatic ducts, Gallbladder, Cystic duct and the Common bile duct (14).

Biliary terminology:

Proximal- the portion of the biliary tree in relative proximity to the liver

Distal -the caudal end closer to the bowel

Branching order -the level of division of the bile ducts starting from the common hepatic ducts e.g. first order branches are the right and left hepatic ducts, second order branches and their respective divisions (secondary biliary radicals)

Central -proximity to the porta hepatis

Peripheral –are higher order branches of the intrahepatic biliary tree extending into the hepatic parenchyma.

Bile drains from the canalicular and ductular network of the acini.(14).

Portal triad is composed of branches of;

1. Portal vein.
2. Hepatic artery
3. Bile duct.

Drainage is from the smallest interlobular bile ducts

↓

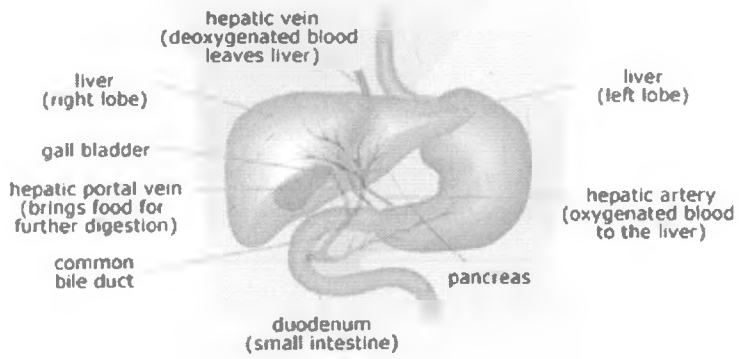
Septal bile ducts

↓

Right and left Hepatic ducts

Caudate lobe has its own separate vascular and biliary apparatus(13).

Figure 1; Anatomy of hepatobiliary tract



Extra hepatic ducts

The right and left main hepatic ducts fuse at the hilum anterior to the bifurcation of the portal vein to form the common hepatic duct (runs caudally in the free edge of the lesser omentum).CHD 4mm.

The main bile duct is divided by the cystic duct insertion into

-common hepatic duct

-common bile duct

The cystic duct

Joins the CHD in its supraduodenal segment in **80%.** It may however extend inferiorly to join it at its retroduodenal or retropancreatic segment.

The common bile duct

Passes inferiorly posterior to the first part of the duodenum and the pancreatic head within the hepatoduodenal ligament. it lies anterior to the portal vein and to the right of the hepatic artery. Terminates in a short common channel with the main pancreatic duct within the wall of the duodenum the 'hepatopancreatic ampulla of vater'.The ampulla and the ends of the

two ducts are each surrounded by sphincteric muscle forming ampullary sphincter of Oddi.The ampulla itself opens into the posteromedial wall of the second portion of the duodenum at the major duodenal papilla 10cm from the pylorus. The dimensions of CBD: length 8-10cm, width 5-6mm- in adults (14).

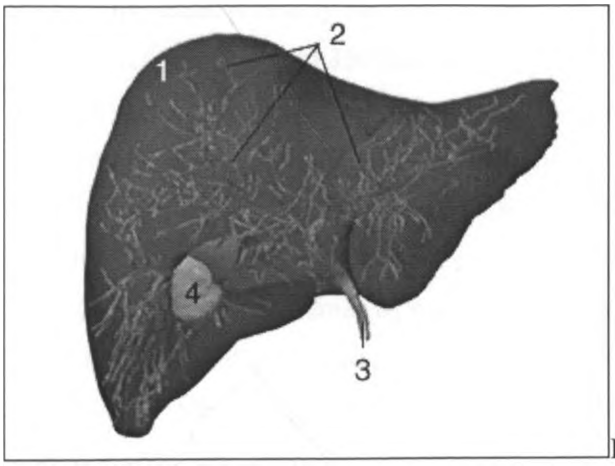


Figure 2; Intra and extrahepatic ducts

1-Liver

2-Intrahepatic bile ducts

3-CBD

4-Gall bladder

GALLBLADDER

Function: stores and concentrates bile secreted by the liver.

Structure: It is globular and pear shaped with a capacity of *30-60ml.It is* fibromuscular with minimal muscular tissue on histology. The Mucosa is simple columnar epithelium.

It is situated in gallbladder fossa on the inferior surface of the liver. Its fundus projects at the inferior margin of the liver touching the parietal peritoneum at the tip of the 9th costal cartilage. It consists of the following parts:

- fundus -most anterior &inferior portion
- body -in contact with the first part of the duodenum.
- neck-continues into the cystic duct.

Spiral valves of heister -thin mucosal folds found in the neck and part of the cystic duct. A mucosal fold in the body give a honeycomb appearance.Hartmanns pouch is a small diverticulum of the neck where it joins the cystic duct. Is pathological and a common site of impaction of gallstones

HEPATOBILLIARY PATHOLOGY

Jaundice

Jaundice (icterus) is yellow pigmentation of tissues and body fluids due to elevated serum bilirubin. Bilirubin is formed from breakdown of the heme ring of hemoglobin molecules and hemoproteins, primarily the cytochromes. The average daily production of total bilirubin in adults is 250 to 350 Mg (16).

Jaundice may be brought to clinical attention by a darkening of urine, or a yellow discoloration of the skin or sclera.Scleral pigmentation is attributed to richness of this tissue in **elastin**, which has a special affinity for bilirubin (16).

Bilirubin occurs in unconjugated and conjugated forms. Unconjugated bilirubin, the direct breakdown product of heme, is water-insoluble at physiologic pH and is measured as indirect bilirubin. Conjugated bilirubin is produced in hepatocytes by esterification of unconjugated bilirubin with glucuronic acid. This process is catalyzed by microsomal uridine diphosphate glucuronyl transferase (UDP-glucuronyl transferase). Conjugation of bilirubin confers water solubility and is measured as direct bilirubin. Normally, total serum bilirubin ranges from 0.3 to 1.2 mg per dL (6 to 20 (micro) mol per L); with conjugated bilirubin accounting for less than 15 percent. The relative proportions of conjugated and unconjugated bilirubin are important in establishing the etiology of jaundice (16)

CLASSIFICATION

Jaundice is usually divided into obstructive (cholestatic) and non obstructive jaundice.

CONJUGATED JAUNDICE

Conjugated hyperbilirubinemia due to any form of hepatobiliary disease is essentially the result of impairment in bile formation and/or bile flow, a condition known as cholestasis [16]. Cholestatic jaundice is often accompanied by a broad spectrum of laboratory, clinical, and histological abnormalities.

Laboratory abnormalities include increased serum levels of alkaline phosphatase and gamma-glutamyltransferase (GGT), and variable elevation of bilirubin, serum copper, ceruloplasmin, cholesterol, lipoprotein X, and serum bile acids, as well as of prothrombin time, which is corrected by vitamin K supplementation, there is minimal or no elevation of aminotransferases. Clinically, pruritus, fatigue, xanthomas, back pain from osteoporosis, pale stools, or even steatorrhea may be present, with evidence of fat-soluble vitamin deficiency.

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Histologically, conjugated hyperbilirubinemia is characterized by bile plugs (bilirubinostasis), feathery degeneration of hepatocytes (cholatestasis), small-bile-duct destruction, pericholangitis, portal edema, bile lakes and infarcts (typically with extra hepatic obstruction), and finally, biliary cirrhosis [16].

MECHANISM OF CHOLESTASIS

Bile formation originates in hepatocytes with the uptake and production of organic anions, bilirubin, and bile salts through diverse cellular transporters that may be either sodium-dependent or independent [17]. Bile salts taken up at the sinusoidal surface of the hepatocytes are generally conjugated to increase their water solubility and subsequently are excreted into the biliary tree at the apical (canalicular) surface. Secretion is achieved via the combined process of Na+ coupled, carrier mediated, or vesicular-transport systems [17].

Multiple factors contribute to the impairment of bile flow: Endotoxins are potent stimuli for activating cytokine production from macrophages [18,19] and have acute cholestatic effects on hepatic bile production [19]. Endotoxins and several proinflammatory cytokines [tumor necrosis factor (TNF)alpha, interleukin (IL)-1, and IL-6 downregulate hepatic transport mechanisms that determine bile acid-dependent bile flow, affecting both bile acid uptake and canalicular secretion[20,21]. These proinflammatory cytokines also promote the expression of MHC class II molecules on target cells, thereby enhancing target antigen presentation [22]. Proinflammatory cytokines activate neutrophils and T and B cells, increase the expression of intercellular adhesion molecules (ICAMs), and may promote tissue damage by direct action. It is proposed that these portal tract inflammatory events can contribute to the downregulation of hepatocellular bile salt transport, and hence aggravate cholestasis.

Unfortunately, there are few cases of cholestatic jaundice in which the specific cellular defect has been identified. For most cholestatic process, multiple defects may act in concert to produce disease. (22).

EVALUATION OF THE PATIENT WITH CHOLESTATIC JAUNDICE

The first question to be resolved is whether the cholestasis results from intrahepatic or extrahepatic disease process, bearing in mind that several intrahepatic causes of cholestatic jaundice can mimic extrahepatic obstruction to varying degree [23, 26]. Comprehensive clinical evaluation comprising the history, physical examination, and basic laboratory tests and the additional information provided by ultrasonography (US) or computed tomography (CT) are highly successful in making this important distinction.

Clinically important clues to extrahepatic obstructions include abdominal pain, a palpable gallbladder or upper abdominal mass, evidence of cholangitis, and a history of previous biliary surgery. Clinical clues to intrahepatic cholestasis include pruritus, as in primary biliary cirrhosis (PBC) and primary sclerosing cholangitis (PSC) patients [24]. Pruritus may be prominent in alcoholic hepatitis and has been reported in about 10% of patients with acute viral hepatitis [24]..

The patient should be asked about risk factors, including alcohol intake, medications, sexual contact, drug abuse, needle punctures, and travel history. The family history is of value in benign recurrent intrahepatic cholestasis (BRIC). Details regarding the onset of jaundice and its duration, whether intermittent or progressive, as well as its associated symptoms like darkening of the urine, acholic stools, arthralgia, rash, weight loss, fever, chills, and pain in the right upper quadrant should be obtained[15].

Physical examination should involve careful observation of stigmata of chronic liver disease, xanthelasma, clubbing, and lymphadenopathy. Hepatomegaly is usual in alcoholic liver disease, primary or secondary hepatic neoplasm, infiltrative disease, and primary biliary cirrhosis (PBC)[25]. Marked splenomegaly suggests cirrhosis with portal hypertension or lymphoproliferative disease [28, 29].

Laboratory work-up for cholestatic jaundice should include complete blood count with differential, urea, creatinine, electrolytes, and a liver panel including alkalinephosphatase, GGT, aminotransferases, albumin, bilirubin, and prothrombin time. Immunological markers such as AMA, ANA, ASMA, ANCA, and immunoglobulin and serological markers for viral hepatitis are helpful. Serum alpha-fetoprotein, carcinoembryonic antigen, and CA19.9 may be increased in patients with malignancies [30].

Clinical evaluation is quite sensitive, but has a positive predictive value of only about 75%; that is, about 25% of patients with suspected obstruction actually have hepatocellular disease [26].

U/S and CT have comparable sensitivity (85%-96%) in detecting dilatation of the intrahepatic and extrahepatic biliary tree in patients with proven obstruction .US is widely recommended as the first-line imaging procedure in the evaluation of cholestatic jaundice. Although gallbladder stones are readily detected by US, common bile duct stones may be missed in 60% of patients because of the interference caused by intestinal gas. Obesity may also lead to an unsatisfactory study. Moreover, with the exception of mass lesion in the head of the pancreas, US usually does not identify the type of obstruction (15).

CT is more likely to yield information regarding the level of the obstruction, localizing this in 90% of patients [15,31]. CT is also a reasonable first choice in patients with lymphoma, in whom it may provide information regarding retroperitoneal lymph node involvement [32]. Although a negative US or CT may represent a logical stopping point in the diagnostic work-up of a patient in whom obstruction is not strongly suspected on clinical grounds, a negative study should not dissuade the clinician from further evaluation of a patient in whom obstruction is considered highly likely.

In patients in whom the clinical suspicion of biliary obstruction is supported by CT or U/S, direct visualization of the biliary tree with percutaneous transhepatic cholangiography (PTC) or endoscopic retrograde cholangiography (ERCP) is appropriate and necessary. PTC and ERCP have in common 99% sensitivity and specificity for the diagnosis of biliary obstruction, and both are capable of demonstrating the site and the nature of the obstruction in more than 90% of patients [33]. Both also provide therapeutic interventions including removal of stones, dilatation of strictures, and the placement of stents across obstructing lesions, as well as the placement of biliary drainage catheters [34].

ERCP is the procedure of choice in suspected ampullary or duodenal lesions, in pancreatic carcinoma and when gallstone obstruction is suspected, in which case sphincterectomy and stone extraction can be implemented.

Palliative stenting of neoplastic obstruction and temporary stenting of certain types of traumatic lesions of the common bile duct are frequently accomplished with ERCP [34]. ERCP is also a logical first procedure in patients with suspected PSC and in patients who have undergone cholecystectomy in whom jaundice is suspected on the basis of choledocholithiasis, since US is often unhelpful in this setting, as the stone is likely to be missed and ductal dilatation may be absent[35].

PTC is often preferred when an obstructing lesion high in the biliary tree is anticipated, as it will permit visualization of the proximal extent of the lesion and enable immediate biliary drainage of obstructed intrahepatic ducts. PTC is also preferred in patients with previous gastrointestinal surgery like Billroth II gastrectomy [35].

PTC is usually contraindicated in patients with marked ascites and coagulopathy.

In some instances, both PTC and ERCP may be used together in a combined therapeutic approach from above and below to maneuver guide wires and stents across a difficult obstruction. Some times hepatobiliary scintigraphy, which is of established value in the diagnosis of acute cholecystitis, may help in evaluating biliary leaks and congenital malformations [35].

Recently, endoscopic CT and magnetic resonance cholangiography have been found to be very helpful in the diagnosis of biliary obstruction, especially in the setting of liver transplantation [38,39]. A negative study obtained by ERCP or PTC represents a reasonable endpoint to the work-up of obstruction in the jaundiced patient.

Liver biopsy may be appropriate at this time point. Minor complications of a cutting needle biopsy, such as prolonged right upper quadrant pain, occur in up to 6% of cases[40].Major complications such as clinically significant intra-abdominal bleeding are uncommon, and mortality (almost always from hemorrhage) is approximately 0.01%[41,42]. Cholestasis per se does not appear to increase the risk of a major complication.

Percutaneous liver biopsy is contraindicated in patients with a significant coagulopathy or substantial ascites; in these instances, performance of a transjugular liver biopsy [43] or not performing a biopsy at all are alternatives. Weighing against these negative considerations are the potential benefits of obtaining histological information. Liver biopsy may be of great value in differentiating hepatocellular cholestasis from obstructive cholestasis [44].Unfortunately, differentiating drug-induced cholestatic hepatitis from other causes cannot be performed histologically.

A chief question in a patient with cholestatic jaundice is whether there is significant underlying chronic liver disease [45] or an infiltrative process, particularly granulomatous disease, lymphoma, or metastatic carcinoma [46].

Portal tract neutrophilic infiltrates seen in liver biopsy are a common accompaniment of biliary obstruction, ascending cholangitis, outright sepsis, cholangiolytic drug reactions, and hyperalimentation [47].

Finally, biopsy of the liver is particularly helpful in differentiating the cholestatic picture of alcoholic hepatitis from that of cholangitis [48].

DIFFERENTIAL DIAGNOSIS

Cholestatic liver disease can be broadly categorized as extra-or intrahepatic. The extrahepatic component is best approached anatomically. The intrahepatic component comprises intrinsic disease, infiltrative disease, systemic disease, and space-occupying lesions.

EXTRAHEPATIC CAUSES OF CHOLESTAT I C JAUNDICE

Among the extrahepatic causes of chronic cholestasis, secondary sclerosing cholangitis due to choledocholithiasis or biliary surgery is probably the most common. This is usually related to a single stricture of the common hepatic duct or common bile duct. Other causes of extrahepatic cholestasis are listed in Table 1.

Table 1: Differential diagnosis of cholestasis and hyperbilirubinemia (cholestatic jaundice)

CBD dilated; best diagnostic approach is anatomical

AMPULLA OF VATER
Stones, carcinoma of pancreas, chronic pancreatitis, ampullary neoplasm, diverticulum, pancreatic cyst, abscess of pancreas, sphincter of Oddi dysfunction
COMMON BILE DUCT
Benign traumatic stricture, stones, choledochal cyst, cholangiocarcinoma, parasites, hemobilia, extrahepatic atresia
GALLBLADDER
Carcinoma of gallbladder
Portal nodes
Cholangicarcinoma, lymphoma, metastatic carcinoma, cavernous portal vein

CHOLEDOCHOLITHIASIS

Although gallstones produce jaundice by impaction in the common bile duct, acute cholecystitis is associated with mild jaundice in up to 20% of patients. This is attributed to edema of the common duct (Mirizzi syndrome) or to direct involvement of the porta hepatis by inflammation [49]. Common duct stones retained after cholecystectomy may produce jaundice in the immediate postoperative period or even several years after Cholecystectomy. Acute gallstone obstruction is often associated with pain from biliary colic or from acute pancreatitis resulting from ampullary obstruction. Sudden impaction of a stone in the common duct may be associated with a rapid rise in aminotransferases 20-50 above normal, followed by an equally rapid decline within 72 hours [50]. Cholangitis is relatively common in patients with choledocholithiasis and manifests as fever with chills, abdominal pain, and jaundice, a syndrome known as Charcot's triad, although jaundice may be absent in one third of patients with cholangitis[51].

BENIGN STRICTURES OF THE BILE DUCTS

Benign biliary stricture in adults following previous surgery and biliary atresia in the pediatric population are the two most common type of strictures. PSC may produce multiple or diffuse strictures that are not associated with proximal ductal dilatation [52]. In patients with chronic alcoholic pancreatitis, a long stricture may develop in the intrapancreatic portion of the common duct, leading initially to cholestasis and eventually

to secondary biliary cirrhosis [53].Ampullary stenosis may result in patients with acquired immunodeficiency syndrome (AIDS) [54] or from the trauma of passing a stone. Cholangitis is frequent in patients with benign biliary obstruction, in contrast to its relative infrequency in the framework of malignant obstruction [30].

NEOPLASTIC OBSTRUCTION

Pancreatic carcinoma is the commonest neoplasm producing obstructive jaundice. Other tumors include cholangiocarcinoma, ampullary tumors, and carcinoma of the gallbladder [30, 55]. Abdominal pain radiating into the back, along with loss of appetite and weight loss, may be present, but jaundice may also develop without pain (usually progressive and deep jaundice). Cholangiocarcinoma may obstruct the biliary system at any level, and the clinical presentation is similar to pancreatic cancer [56]. Cholangiocarcinoma of the extrahepatic bile ducts may be growing into the lumen. The sclerosing variant of cholangiocarcinoma, which frequently arises at the confluence of the right and left hepatic ducts (Klatskin's tumor), may be difficult to distinguish from PSC both radiologically and on biopsy. This tumor infiltrates early into the wall of the bile duct, where it elicits a markedly sclerotic response [57].

Tumors producing complete obstruction of the common bile duct may be accompanied by marked, palpable dilatation of the gallbladder (Courvoisier's law). Ampullary tumors may produce intermittent jaundice because of sloughing of the tumor and partial relief of the block. Metastatic cancer may obstruct the bile duct, as may lymphoma [58]. Hepatocellular carcinoma may uncommonly rupture into the biliary system and give rise to tumor emboli that lodge in and obstruct the common duct [59]. The extrahepatic ducts may be compressed by adjacent tumor, by peribiliary lymph node infiltrated by lymphoma, or by metastatic carcinoma of breast [58]. Direct infiltration of the ducts by lymphoma may also lead to obstruction [60].

UNCOMMON CAUSES OF OBSTRUCTIVE JAUNDICE

Choledochal cyst may first manifest as obstructive jaundice after 17 years of age [61]. A duodenal diverticulum is a rare cause of biliary obstruction.Hemobilia, mostly a result of hepatic trauma, including invasive procedures or neoplasm, presents with the triad of biliary colic, jaundice and gastrointestinal bleeding [62]

Invasion of the common bile duct with Ascaris or with liver flukes of the Fasciola, Clonorchis, or Opisthorchis genera may produce cholangitis [63]. Secondary sclerosing Cholangitis due to opportunistic infection of immunodeficient patients has become increasingly common since the advent of AIDS. Cryptosporidium

Parvum, cytomegalovirus (CMV), and Microsporidia are the organisms most frequently found [64].

JUSTIFICATION/RATIONALE

Ultrasonography is a cheap, readily available and non invasive imaging modality for diagnosing various hepatobiliary pathology and without risk of radiation.

To the best of my knowledge, no study had been carried out locally to determine the capacity of ultrasound in determining the site and cause of obstructive jaundice.

This study may form a foundation upon which future research on hepatobiliary diseases will be based on.

RESERCH QUESTION

Is ultrasonographic examination reliable in determining the site and etiology of biliary obstruction?

OBJECTIVES

Broad objective

The main objective of this study was to determine the ability of ultrasonographic examination to accurately determine the site and etiology of biliary obstruction.

SPECIFIC OBJECTIVES

To determine the pattern of ultrasonographic findings in obstructive jaundice.

To evaluate the regional distribution in the biliary tree of the various causes of obstructive jaundice.

To determine the age and sex distribution and the relative frequency of the lesions

Causing obstructive jaundice

ETHICAL CONSIDERATION

There were a number of ethical considerations made while undertaking the study.

The patients' names were not used in the study in order to maintain confidentiality.

Before commencement of this study, the proposal was submitted to the ethical committee of KNH for approval.

Signed consent was required before recruiting patients into the study.

The results of this study will be delivered to the KNH ethical committee to assist them form a database for future study and reference and to facilitate any possible improvement in patient management.

The information obtained from this study was treated with confidentiality and results of the study used for academic and clinical improvement purposes only.

MATERIALS AND METHODOLOGY

STUDY AREA

This study was conducted at the radiology department of KenyattaNatonal Hospital and University of Nairobi.

STUDY POPULATION

The study population consisted of all patients with a clinical suspicion of obstructive jaundice. Patients were mainly recruited from the surgical (liver) clinic at KNH. A total of 40 patients (23 males, 17 females, mean age 60yrs) with sonographic evidence of biliary obstruction were entered into the study from June 2007 to April 2008.

The patients had to undertake another diagnostic test or surgery in which the ultrasound results were compared with.

STUDY DESIGN

This was a descriptive prospective study.

SAMPLE SIZE

The sample size was determined using the statistical formula below;

At confidence interval of 95% and a margin error of 5% and prevalence rate of hepatobiliary diseases at 2%, the sample size was calculated by the formula:

$$N = (1.96/m)^2 p(1-p)$$

Where p=proportion of prevalence
N=sample size
M=proportion of margin of error

Using this formula the sample size (n) was 30 subjects.

The actual sample size was 40 patients, done to increase precision and power of study.

SAMPLING METHOD

All ultrasound examination of consenting patients were studied consecutively for the period extending from June 2007 to April 2008 and who met the inclusion criteria outlined below were included in the study.

INCLUSION AND EXCLUSION CRITERIA

Inclusion criteria

Patients with a clinical suspicion of obstructive jaundice referred to KNH and DDR UON who consented to the study and had a second diagnostic imaging study or underwent surgery.

Exclusion criteria

Patients with obstructive jaundice but no second test to confirm the U/S findings.

Patients who did not consent.

Patients with non obstructive jaundice.

STUDY LIMITATIONS

Some subjects opted out of the study to receive further treatment at a different facility after being recruited into the study and this resulted in breakdown of follow up.

Some patients were also unable to undergo further investigation or surgical exploration to determine the proof of the cause of jaundice in which ultrasound results had to be compared with.

In some cases there was a long lag period between the time of undergoing ultrasound examination and the time other modalities were undertaken to determine cause and site of obstruction. This may have resulted in discrepancy between ultrasonographic findings and surgical findings due to progress of pathology during this lag period.

EXAMINATION PROCEDURE

Patients with sonographic evidence of intrahepatic or exrahepatic biliary obstruction were entered prospectively into the study from June 2007 to April 2008.

Standard criteria below was used to distinguish intrahepatic from extrahepatic obstruction.Intrahepatic ducts were identified by "double tracking" or "multiple tubes" in the liver.Extrahepatic obstruction was diagnosed when the common hepatic ducts were more than 4 mm in diameter and common bile duct was more than 8mm in diameter. Ultrasonography was performed in the two departments utilizing the following machines:

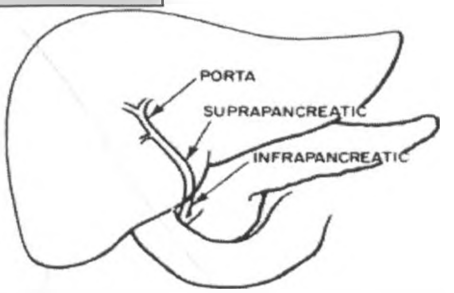
- General Electric (LOGIQ 3)-UON
- Philips ultrasound HD 11 (2006)-KNH.

Real time transducer of 3.5-5 MHZ with colour Doppler facilities was used.

Ultrasound examination was carried out with the patient in supine position. Liquid gel was smeared on the abdomen and the liver and the biliary system scanned in Sagittal, transverse and oblique planes. The patients had to have starved for at least 6 hours before undergoing ultrasound examination. In some cases liquid water was given during scanning time to assist in outlining the pancrease.There is no examination which was done on emergency situation. The images were mainly stored in hard copies. The intrahepatic ducts were identified and any dilatation noted.Colour Doppler was used to differentiate the ducts from hepatic vessels. The extrahepatic ducts were examined from the porta hepatis up to the ampullar and their diameter noted.

The extrahepatic biliary system was divided into three regions: the portahepatis, the suprapancreatic common bile duct and the intrapancreatic or ampullary part of the common bile duct (**Fig 12**) and the site of obstruction was determined from one of these sites.

FIGURE 12



Schematic drawing of the three anatomic divisions of the extrahepatic common bile duct.

The patient's history and laboratory investigations and sonographic findings were recorded. The principal investigator carried out the ultrasound examinations on patients with clinical diagnosis of obstructive jaundice. The caliber of the biliary ducts was measured to determine the presence of dilatation.

A diagnosis of extrahepatic dilatation was made if the CHD was more than 4mm in diameter and if the CBD was more than 8mm in diameter.

The cause of the obstruction and the site of the obstruction were also evaluated by US. The examination was repeated by consultant radiologist and the same parameters as above measured and the two results compared to come to a final radiological diagnosis. The proof of the cause and site of obstruction was determined by other modalities such as PTC, MRCP, CT SCAN, percutaneuos biopsy or surgical exploration. The results of these tests were interpreted by the radiologist who carried out the examination independent of sonographic findings to avoid bias.

A definitive diagnosis of the cause of obstruction was made when the clinical, laboratory, and radiographic evidence overwhelmingly favored a particular conclusion. Specifically, obstruction secondary to choledocholithiasis was prospectively diagnosed only when an echogenic focus in the common bile duct demonstrated accompanying

Shadowing. A mass in the pancreas was considered to be malignant when other ancillary features signifying malignancy were present such as liver metastasis or lymphadenopathy.

DATA AND RESULT PRESENTATION.

Statistical package for social scientist (SPSS version 17) method was used in data processing.

Fourty two patients were recruited into the study. Two patients were unable to undergo further examinations or surgical exploration and were not entered into the study.

Fourty patients (23 males, 17 females, mean age 60yrs) with sonographic evidence of intrahepatic or extrahepatic biliary obstruction were entered prospectively into the study from June 2007 to April 2008.

A total of 33 patients underwent surgical exploration and two had percutaneuos biopsies. The remaining five patients were followed up in surgical clinic with repeat radiologic examination for at least four months before a clinical diagnosis was confirmed. CT scan was carried out on 25 patients.

Proof of the site of obstruction was either by surgery (33 patients), cholangiography (two patients) and MRCP (five patients). Proof of etiology was either by surgery (33 patients), biopsy (two patients), or clinical follow-up with repeat examinations (five patients).

TABLE I and figure 13 summarizes the final confirmed diagnoses at surgery in the 40 patients.

TABLE 1.Pathologic diagnosis

	NUMBER OF PATIENTS	male	female
NEOPLASM: Ca pancrease-----23case cholangiocarcinoma-4cases Lymphoma--------1 case	28	17	11
CHOLEDOCHOLITHIASIS	4	1	3
BENIGN STRICTURE	3	2	1
PANCREATIC INFLAMATORY DISEASE	3	2	1
CHOLEDOCHOMEGALLY	2	1	1
TOTAL	40	23	17

Twenty-three patients had carcinoma of the pancreas, four patients had cholangiocarcinoma, one had lymphoma, three had Pancreatitis, and 4 had common duct stones. In 2 cases the common bile duct was enlarged and gallstones with acute and/or chronic cholecystitis were found at surgery. The bile ducts of these patients were 8 mm to 1.2 cm in diameter. In none of these patients was a specific cause for bile duct dilatation found. The presumptive diagnosis was a passed common duct stone. Finally, there were three cases of benign strictures involving the common duct or biliary enteric anastomosis Malignant lesions were the main cause of obstructive jaundice in the sampled patients. The majority of the cases were due to carcinoma of the pancrease with 23 cases 82% or (57%of the total), followed by cholangiocarcinoma with 4cases-(14% or 10% 0f the total) and lymphoma with 1case contributing to 4%or 3 % of the total number.

The sex distribution of the neoplastic condition is as shown in the table below:

Neoplasm	Total	male	female
Ca pancreas	23	14	9
Cholangiocarcinoma	4	2	2
Lymphoma	1	1	0
Total	28	17	11

TABLE II and figure 14 summarizes the results of the prospective assessment of the 40 cases with respect to site of obstruction on ultrasound.

TABLE 2.Site of obstruction

SITE OF OBSTRUCTION	DETERMINATIOM	
	Correct	Incorrect
Porta hepatis	6	1
Suprapancreatic CBD	2	2
Infrapancreatic CBD	12	3

N=40 Site of obstruction was indefinite in 14/40(35%) of patients

Of the 40 cases, the site of obstruction was not sonographically determined in14 cases (35%). This was a common problem due to either overlying gas or the inability to pinpoint the level of the obstruction even when the common bile duct was seen. The site of obstruction was prospectively predicted by u/s in 26 cases (65%).Of the 26 predicted cases we were correct in 20 (77%),or 50% of the total number of cases and incorrect in 6cases. None of the three sites appeared to be more easily determined than the others. In those cases in which the site was indeterminate or incorrect, scanning did not persistently miss any one particular area.

TABLE III and figure 15 shows the prospective assessment of the cause of obstruction in the 40 cases on ultrasound.

TABLE 3.CAUSE OF OBSTRUCTION

	DETERMINATION	
CAUSE	**CORRECT**	**INCORRECT**
TUMOUR	25	1
STONE	2	1
PANCREATIC INLAMATORY STRICTURE	1	0
BENIGN SRICTURE	1	0

N=40 The cause of obstruction was indefinite in9/40 patients

In 9 cases (22%) the cause of obstruction could not be determined by ultrasound. Of the remaining 31 patients (78%), the cause was correctly determined in 29(72%) of the total cases analysed. In this group there were twenty two cases of pancreatic carcinomas(75.8%),one case of lymphoma(3.4%), two cases of cholangiocarcinoma(7%), two cases of bile duct stone(7%), one case of pancreatic inflammatory mass(3.4%),and one case of postoperative biliary stricture(3.4%).

In two cases (the double error) a shadowing focus within the common bile duct, which was thought to be caused by a stone, actually resulted from a surgical clip and the other which was thought to be carcinoma of the head of pancreas was tuberculous inflammatory mass.

The ability to assign a correct cause to the 27 noncalculous lesions was entirely dependent on the availability of ancillary data, *i.e.* the identification of liver metastasis in14 cases, the history of malignancy in 5 cases, a second mass, i.e lymphadenopathy in 6 cases, a sonographic appearance of a calcified pancreas suggesting chronic Pancreatitis in one, and a history of biliary enteric anastomosis suggesting the presence of a stricture in one case.

Table 4 shows the cause of obstruction in cases which were defined as indeterminate.

Dilated but nonobstructed CBD	2 cases
Non visualized CBD stones	2 cases
Benign biliary enteric stricture	2 cases
Pancreatic masses	3 cases
Total number	9 cases

The causes of obstruction in the 9 patients defined as indeterminate were nonvisualized common duct stones in 2 patients(these were also missed on CT scan), dilated but nonobstructed common ducts (presumed passed stone) in 2 patients, benign biliary enteric strictures in 2 patients, visualized pancreatic masses without features that distinguished between cancer and Pancreatitis in 3 patients.

Twenty-six proven pancreatic masses caused biliary obstruction; 23 were primary pancreatic tumors and three resulted from Pancreatitis in which one case was Tuberculous Pancreatitis. Fourteen patients had a proven normal pancreas. All of these patients had biliary dilatation from either choledocholithiasis or biliary stricture (benign &malignant). Only 2 common bile duct stone and one common bile duct stricture were prospectively and correctly diagnosed. The pancreas could not be seen in 2 patients due to overlying gas.

Of 12cases in which the pancreas was considered normal on sonography, the sonographic findings were proved correct in 12 cases (100%).

All 26 cases called abnormal by sonography proved to be abnormal both at CT scan and surgery. There were23 patients with carcinomas and three with inflammatory masses. Of the two patients in whom the pancreas was not seen, one had benign disease and one had malignant disease. These were demonstrated at CT scan and surgery.

Table 5 and figure 16 shows the age and sex distribution of patients with obstructive jaundice

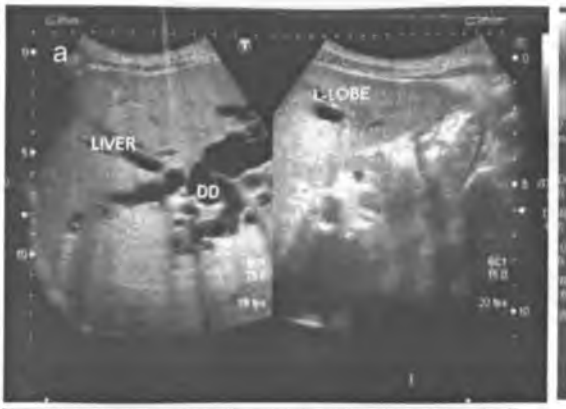
Age distribution(in years)	Total number of cases	Male	female
0-10	0	0	0
11-20	0	0	0
21-30	2	1	1
31-40	3	3	0
41-50	7	4	3
51-60	5	2	3
61-70	12	7	5
71-80	8	5	3
>81	3	1	2
TOTAL	40	23	17

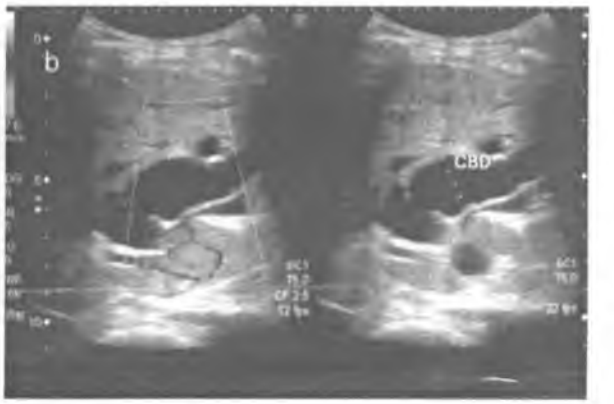
MALE=57% **FEMALE=43%**

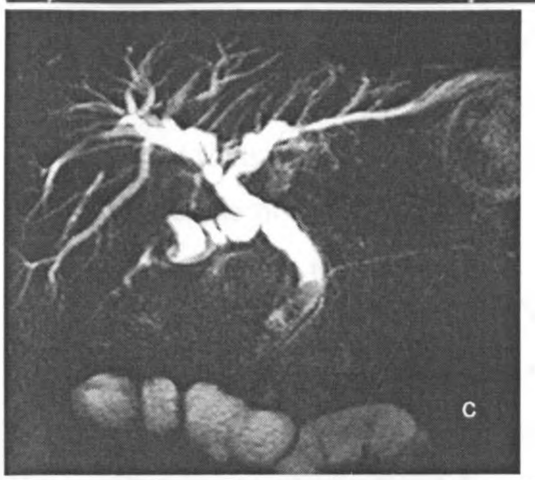
The 40 patients evaluated in this study consisted of 23 males (57%) and 17 females (43%) giving a male to female ratio of 1.3:1.The age range varied from 23 years to 94 years. Both the youngest and the oldest patients were females. The youngest patient had cholangiocarcinoma and the oldest had carcinoma of the head of the pancreas.

Majority of the patients were in the 61-70 year group with a total of 12 patients (30%of total), seven were males and five were females.

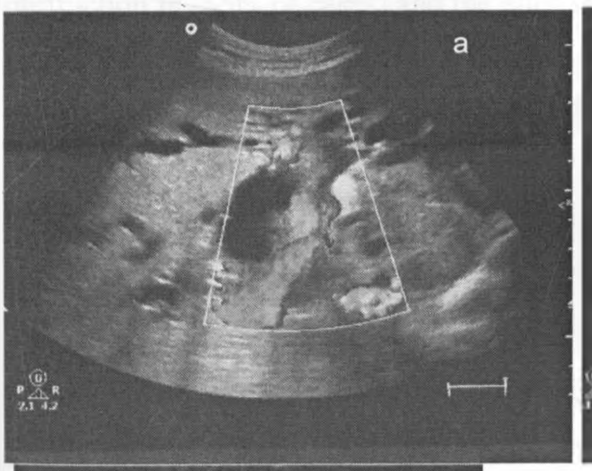
The mean age of the patients who were recruited into this study was 59.5 years.

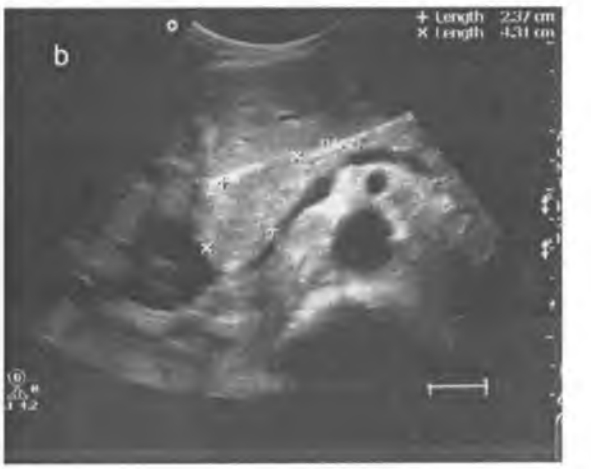


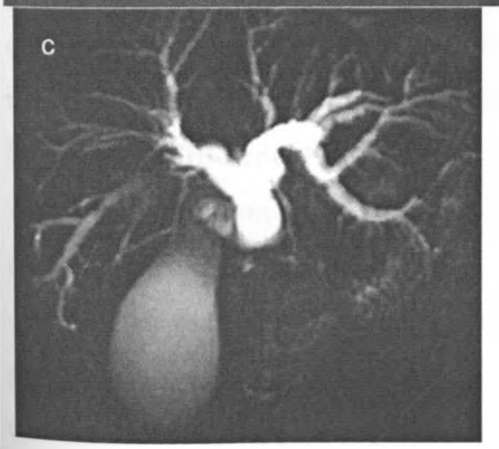




66-year-old woman with jaundice and fever. a&b.Transverse and Sagittal sonogram demonstrates dilated intrahepatic and common duct *(CD).No cause or site of obstruction could be seen.* In this
Case the site and cause of obstruction were "indeterminate."
c. MRCP demonstrates low signal filling defects in distal CBD which is consistent with final diagnosis of choledocholithiasis found at surgery







71-year-old man with jaundice.
a. Sagittal sonogram demonstrates typical findings of biliary dilatation but not the site or cause of obstruction (b) Transverse sonogram demonstrate mass in head of pancreas which is consistent with carcinoma of pancreas.
(c). MRCP demonstrates obstruction in the suprapancreatic portion of the common duct, which is consistent with the final diagnosis of carcinoma of the pancreas

DISCUSSION

The expanding spectrum of therapeutic options for the jaundiced patient has made it necessary for the radiologist to do more than simply discriminate between obstructive and nonobstructive jaundice. Correct choices among therapeutic options usually rest upon a precise assessment of etiology, location, and extent of disease.

In the literature the accuracy of U/S in predicting the site and cause of obstructive jaundice is variable. The results of previous studies indicate that sonography compares well with other imaging tools such as direct cholangiography and MRCP in accurately determining the site and cause of biliary obstruction [3].Other few studies show that U/S is not very accurate in predicting the cause and site of biliary obstruction as shown by studies by C.A.Muhletaler et al [4] and Stephen.P.Honickman et al [12]. Many authors imply that no further diagnostic procedures are necessary prior to the institution of therapy. The results of this study do differ with this thesis to some extend.The success in determining the site of obstruction in (65% of cases) and the etiology of obstruction in (78% of cases) is less than that of 85% to95% success rates reported by others (9). I believe this discrepancy stems from the stringent criteria we applied in defining the site and cause of obstruction and the lack of such criteria in prior studies.

This discrepancy is mainly due to different criteria and stringent measures which are applied in determining the cause and site of obstruction.

The failure to determine the site of obstruction in 35% of the patients was primarily due to the inability to visualize the common duct in its entirety. One must see the dilated common duct terminate at a specific point or merge into a normal-sized common duct to accurately determine the site of obstruction. Thus even in cases in which the pancreas was visualized, its exact relationship to the obstructive duct could not always be seen.

Because the differential diagnosis for an obstructing lesion, as well as therapeutic options, varies depending on the location of obstruction, in this study, the CBD was divided into three distinct areas (portal hepatis, suprapancreatic CBD and infrapancreatic CBD). For example, disease at the porta hepatic is almost always malignant and often treated by percutaneous drainage rather than surgery. Suprapancreatic obstruction usually indicates carcinoma. Similarly, distal common duct disease may be benign or malignant and may be

diagnosed and even treated by endoscopic retrograde examination and papillotomy. One may argue that separation of the site of obstruction into "high in the porta" or "low near the pancreas" is adequate for patient management. The value of more definitive information may vary with the referring clinician. However, accurate determination of the exact point of the obstruction, as well as its cause, is helpful and may change the approach to therapy from surgery to percutaneous drainage or endoscopy.

The two cases in this series of "dilated nonobstructed" biliary systems illustrate the drawbacks of ultrasonography. This subgroup of patients was indistinguishable on sonography from the subgroup with dilated "obstructed systems" from impacted gallstones or ampullary tumor. Whether these two cases resulted from recent passage of a stone, dislodging of a stone intra-operatively, ampullary stenosis, or prior episodes of obstruction secondary to stone passage can never be known.

Distinguishing the acutely obstructed system from the dilated nonobstructed system could be of critical importance in avoiding sugery in a patient whose ability to tolerate surgery is marginal.

The reasons for failure to pinpoint a specific cause in 22% of cases were more complicated than simple technical problems such as nonvisualization of the obstructing mass. The inability to determine the cause of obstruction when a mass was present was not usually due to technical problems. The primary problem, then, is the nonspecificity of a focally enlarged hypoechoic pancreas. It has to be decided whether to call all pancreatic masses as carcinoma. The distinction between malignant obstruction and pancreatitis can be made only if ancillary data, for example evidence for a liver metastasis, are present. The distinction between a focal mass and a diffusely enlarged pancreas has been shown not to be an adequate distinguishing feature of pancreatitis and carcinoma of the pancreas (9).

When the pancrease was confidently reported as abnormal, as it were in 26of 40

Patients, the pancreas was proved to be abnormal by MRI, CT scan or surgery. On the other hand, the pancreas was proved normal in 12/12 predicted cases.. Thus despite the nonspecificity of ultrasound in determining the etiology of the lesion, it was sensitive enough to identify the abnormal pancreas, as has been described in other studies. U/S was correct in identifying normal and abnormal pancrease.

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The limitations of ultrasound must be weighed against other modalities that offer more specific information about the site and cause of obstruction.

Similarly encouraging results have been reported for MRCP. Reinhold C, Bret PM et al [65] recently reported exciting results using MRCP in determining the site and cause of biliary obstruction with a high degree of accuracy. This degree of accuracy compares favorably with that seen with direct cholangiography, and during MRCPexamination, there is added benefit of tumor staging by delineating the extent of disease.

CONCLUSION

The results of this study demonstrate that ultrasound is an accurate imaging modality in differentiating obstructive from non obstructive jaundice as has been reported by others. Some studies have indicated it to have an accuracy of 95-99 %(9).

When it comes to detecting the cause of obstruction, ultrasound has the ability to identify the abnormality but is non specific in assigning a definite diagnosis to the abnormality.

Ultrasound can not be solely be relied upon to determine the specific site of obstruction as shown by the results of this study. More accurate imaging modalities such as MRCP or PTC have to be employed to accurately pinpoint the site of obstruction in most cases.

Ultrsound is accurate in identifying the pathology but it is not specific in correctly predicting the site of obstruction.

Nevertheless ultrasound has a big role to play when it comes to investigating patients with obstructive jaundice.

RECOMMENDATION

The results of this study demonstrate that the use of ultrasound should be encouraged as the first screening tool in patients who present with obstructive jaundice. Indeed, these findings support previous data depicting the accuracy of ultrasound in separating obstructed from nonobstructed biliary systems and its ability to detect pancreatic masses.

Nonetheless, it is also important to know the limitations of any "screening" modality in the work up of a jaundiced patient.

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APPENDIX A: QUESTIONNAIRE

QUESTIONNAIRE (DATA COLLECTION FORM)

1) Patients number

2) Age

3) Sex

4) Previous history of surgery **Yes** **No**

5) History and duration of jaundice

(b) Liver disease

6) Presenting Symptom Present Absent

- jaundice
- abd pain
- fever/chills

APPENDIX A: QUESTIONNAIRE

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6) Presenting Symptom Present Absent

- jaundice
- abd pain
- fever/chills

(7)U/S scan findings

a) cause of obstruction

b) Site of obstruction

8) Surgical findings

9) Percutaneous biopsy findings

10) ERCP findings

11) PTC findings

12) Any other imaging finding,

APPENDIX B: BUDGETARY JUSTIFICATION

BUDGET

No.	Requirement	Cost (Kshs)
1	Stationary, photocopying, typing	18,000
2	Secretarial services	5,000
3	Data analysis	15,000
4	Printing and scanning documents	16,000
6	Binding	5,500
7	Data collection	14,000
8	Transport	6,000
9	Contingency	20,000
	TOTAL	**99,500**

The above expenses were be met by the researcher.

The contingency allocation provided was to cater for any unforeseen expenditure

APPENDIX C:CONSENT

CONSENT FORM

My name is Dr. Ngoseywe Kennedy, a Master of Medicine student at the Department of Diagnostic Radiology, University of Nairobi. I am doing a study on Hepatobiliary disease and the accuracy of ultrasound in diagnosing these diseases, and would wish to recruit you to participate. The information you will give and the examination findings will be handled with utmost confidentiality.

Your name will not be included at all, but your treatment number will be used.

I have been given the opportunity to ask questions concerning the study and such questions have been answered to my satisfaction.

I understand that I am not obliged to participate, and that I may at any time during the course of the study revoke the consent for the study without any prejudice.

If you accept to participate, please sign below:

Sign

Date

Number

I certify that the patient has understood the nature of the study and consented to fully participate.

Dr Ngoseywe Kennedy

Signature

KIBALI CHA KUHUSIKA KATIKA UTAFITI.

Jina langu ni Daktari Ngoseywe Kennedy, mwanafunzi katika chuo cha udakitari, Chuo Kikuu cha Nairobi. Ninafanya utafiti kuhusu magonjwa ya maini na mishipa yake" nikilinganisha uwezo wa picha ya ultrasound kuweza kuyatambua magojwa hayo, na ningependa kukuchagua kama mmoja wapo wa watakao husika katika utafiti huu.

Habari utakayotoa au ile itakayopatikana kukuhusu, itakuwa siri na kutumika tu katika utafiti huu. Jina lako halitajumlishwa, bali ile nambari ya matibabu ndiyo tu itakayo tumika.

Nimepewa nafasi ya kukuuliza maswali ambayo yanahusu utafiti huu , ambayo yamejibiwa kikamilivu.

Naelewa ya kwamba sio lazima nihusike katika huu utafiti, na pia naweza kubadili nia yangu kuhusu kuendelea kushiriki.

Asante sana kwa ushirika wako.

Kama unakubali kushiriki, tafadhali weka sahihi yako hapa chini:

Sahihi

Tarehe

Nambari

Ninathibitisha ya kwamba muhusika ameelewa na kukubali kushiriki kwa utafiti huu.

Daktari Ngoseywe Kennedy

Sahihi

Tarehe