ORIGINAL RESEARCH ARTICLE

Role of Ultrasonography in Diagnosis of Liver Cirrhosis and its Complications

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A B S T R A C T

Introduction: Ultrasonography has a major role to play in screening, diagnosis and management of patients with focal and diffuse liver disease. This study was undertaken to ascertain the role of ultrasonography in diagnosis of liver cirrhosis and its complications.

Material and methods: The present study was conducted on 114 cases, suspected to be of liver cirrhosis clinically and by laboratory data during study period. These included cases attending on out-patient department basis and also those admitted in wards. The data collection was done by using semi-structured questionnaire and clinical examination. Clinically suspected cases of liver cirrhosis were screened for ultrasonography features.

Result: Maximum number of cases of liver cirrhosis occurred in age group of 11 – 60 years, that is 84 out of 100 (84%), males outnumbered the females. Majority of cases were of alcoholic cirrhosis 56% followed by post necrotic cirrhosis 37%. Raised echogenecity, surface nodularity and course echopattern were frequently encountered signs. Ascites and gall bladder complications were most commonly seen.

Conclusion: Ultrasound is a quick, noninvasive, easy, repeatable & effective method of diagnosing liver cirrhosis and its complications. The sensitivity of ultrasonography as a diagnostic tool is high and approaches to that of other investigations.

Keywords: Ultrasound, Liver Cirrhosis, Alcohol Consumption, Echogenecity, Surface Nodularity, Coarse Echopattern

INTRODUCTION

Liver is the largest single organ in the body and also largest reticuloendothelial unit in itself. In old days, clinical examination of patient was the only means of liver study. Then came intravenous cholangiography. This was an indirect method of assessing liver lesions as radiographic data was not very clear. Surgeons had laproscopy and laprotomies as ways to directly approach liver pathologies.

Further diagnostic approaches like liver scintigraphy, splenoportography and arteriography helped to reach at a conclusive diagnosis. As these methods were not cost effective and also being invasive they could not become mainstay in diagnosis of liver disease.

For the past two decades liver imaging has progressed by leaps and bounds. Modalities like ultrasonography, CT scan, MRI, SPECT have changed the very concept of liver disease and their management. Ultrasound had the advantage of being real time, economical, easily available, repeatable and quick. Also it depend very little on liver function. Now with the widespread use of ultrasound most of structures of the body have become crystal clear in the eyes of physicians and surgeons. Liver, is no exception to this fact. Information of this splendid organ can now be achieved at low cost, little risk and most importantly in a non – invasive way with use of ultrasound. Patient preparation is required to a minimum, pt co-operation is also of meager need and this procedure can be done in emergency wards; as a bedside investigation.

Cirrhosis is the last stage of chronic liver disease. It is associated with possible side effects, such as gastrointestinal changes, eczema, liver disease, and the development of hepatocellular carcinoma (HCC), which requires careful medical attention.[1-3] Ultrasound has emerged as an important tool in diagnosis of liver cirrhosis and its complications. It has also helped in management of patients suffering from this disease. Guided procedures like FNAC of a suspected malignant lesion or paracentesis can be done with aid of sonography.

Cirrhosis is a generic term used to describe chronic liver disease involving diffuse parenchymal necrosis, active formation of connective tissue leading to fibrosis and nodular regeneration of liver, resulting in disorganization of hepatic lobular and vascular architecture.[4] All the rivers of pathologies that flood liver lead and meet at one common sea of fate; that is cirrhosis.

Detection of liver edge beneath costal margin does not always indicate hepatomegaly. Detection of large masses by physical

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examination alone is not possible in most of the cases. Symptoms of liver disease in early stages are nonspecific i.e. anorexia, malaise, dyspepsia and clinical signs of liver disease may not emerge until liver failure has reached an advanced stage. Biochemical tests can be used to quantitate impaired liver function but are of limited value in narrowing the differential diagnosis of disease.[5] Hence ultrasonography has a major role to play in screening, diagnosis and management of patients with focal and diffuse liver disease.

This study was undertaken to ascertain the role of ultrasonography in diagnosis of liver cirrhosis and its complications.

MATERIAL AND METHODS

The present study was conducted on 114 cases, suspected to be of liver cirrhosis clinically and by laboratory data during study period. These included cases attending on out-patient department basis and also those admitted in wards.

Out of these 114 cases, 9 cases were of alcoholic hepatitis and 5 cases were of post necrotic hepatitis. Hence total of 14 patients were excluded. This made the list of study cases of liver cirrhosis 100.

Data collection procedure

The data collection was done by using semi-structured questionnaire and clinical examination. Clinically suspected cases of liver cirrhosis were screened for ultrasonography features like, Liver surface irregularity, changes in hepatic architecture – coarse echopattern, hepatic parenchymal echogenecity, Hepatic morphology – liver size, ratio of transverse diameter of caudate lobe to that of rt. lobe (C/RL/>/= 0.65 as baseline value), signs of portal hypertension – diameter of portal vein, loss of caliber variation of splenic & superior mesenteric vein, collateral between portal and systemic circulation, splenomegaly, ascites, hepatocellular carcinoma etc

RESULTS

Maximum number of cases of liver cirrhosis occurred in age group of 11 – 60 years, that is 84 out of 100 (84%). Youngest patient was 8 year old and oldest was 86 year old. The study included 70 male patients of liver cirrhosis and 30 female patients.(table-1)

The shows that majority of cases were of alcoholic cirrhosis 56% followed by post necrotic cirrhosis 37%. Other causes of cirrhosis that were found were: 2 cases of cardiac cirrhosis and 5 cases were cryptogenic, making it 7%. Alcoholic to post necrotic ratio in males was 3.85:1 and in females was 0.17:1. The overall sensitivity of USG in detecting cirrhosis was 87.7%.(Table2)

Raised echogenecity, surface nodularity and course echopattern were frequently encountered signs.

Increase caudate to rt. lobe ratio and loss of normal triphasic waveform were seen in 57% and 56.9% respectively.

Shruken liver was seen in 47% cases.(Table 3)

Portal hypertension was found in 33 out of 100 patients. Splenomegaly with dilated splenic radicles, porto – systemic collaterals and dilated splenic vein were frequently found signs of portal hypertension.

Loss of respiratory caliber variation in splenic vein and

Characteristics	Number	Percentage
Age		
0 – 10	4	4
11 – 20	13	13
21 – 30	17	17
31 – 40	20	20
41 – 50	20	20
51 – 60	13	13
61 – 70	9	9
>70 years	3	3
Gender		
Male	70	70
Female	30	30

Table-1: Basic characteristics

Type of cirrhosis	Male	Female	Total
Alcoholic	54	2	56
Post – necrotic	14	23	37
Others	2	5	7

Table–2: Etiological factors in cases of liver cirrhosis

Features	Number	Percentage
Liver size		
Increased	18	18
Decreased	47	47
Normal	35	35
Surface nodularity		
Present	67	67
Absent	33	33
Echogenecity		
Raised	70	70
Normal	30	30
Echopattern		
Coarse	68	68
Fine & homogeneous	32	32
Caudate to rt. lobe ratio C/RL		
Normal <0.65	43	43
0.66 – 0.75	37	37
0.76 – 0.85	17	17
>0.85	3	3
Hepatic venous wave form (n=85)		
Monophasic	17	20
Dampened	28	36.9
Triphasic (normal)	40	43.1

Table–3: Ultrasonographic features of liver cirrhosis

Features	Number	Percentage
Portal vein diameter >1.3 cm	15	45.45%
Splenomegaly with dilated splenic vein radicles	29	87.87%
Dilated splenic vein	25	75.75%
Loss of mild respiratory variation of superior mesenteric and splenic vein	24	72.72%
Porto – systemic collaterals	27	81.81

Table–4: Ultrasound features of portal by hypertension

Complications	Number	Percentage
Portal hypertension	33	33
Ascites	64	64
Splenomegaly	37	37
Hepato – cellular carcinoma	8	8
Portal vein thrombosis	9	9
Gall Bladder complications	62	62

Table–5: Complications of liver cirrhosis by USG

superior mesenteric vein was seen in 72% cases. Portal vein dilatation was seen in 45% cases.(Table4)

In our study, ascites and gall bladder complications were most commonly seen. Splenomegaly and portal hypertension had incidence of 37% & 33% respectively.(Table5)

Gall bladder wall thickening was seen in 60% of patients, while gallstones were found in 22% cases. Gall bladder varices were seen in 2% of cases and these had associated portal vein thrombosis.

DISCUSSION

Age (years)

In present study, liver cirrhosis is prevalent in age group of 8-86 yrs. Maximum number of cases were seen in age group of 11-60 yrs (84%) which is in accordance to other studies. The youngest patient was 8 yrs old and was of post – necrotic cirrhosis. Older age groups were seen to have alcoholic and post – necrotic cirrhosis. 2 patients in middle age group had cardiac cirrhosis.

Leiber C S in 1978 showed alcoholic liver disease to be common in age group of 21 – 65 yrs.[6] Walewska et al showed chronic hepatitis was common in age group of 18-71 yr.[7]

Gender

In the present study, males outnumbered the females, ratio being 2.33:1. The greater number of male patients is due to more consumption of alcohol by males. Similar findings also reported by previous studies.

Etiological factors in cases of liver cirrhosis

The commonest etiological factors of cirrhosis in our study was alcoholism, followed by post necrotic cirrhosis, they accounted for 56% and 37% cases respectively. 2% cases were of cardiac cirrhosis due to constrictive pericarditis. These patients also had other features of congestive cardiac failure like pleural effusion, pericardial effusion etc. 5% cases were of cryptogenic type. According to previous studies alcoholic cirrhosis accounts for 60-70% cases, post necrotic 10% and other causes account for rest i.e. 20-30%.

In our study ration of alcoholic to post necrotic cirrhosis was 1.5:1. Alcoholic to post necrotic cirrhosis ration in males was 3.85:1 and in females it was 0.17:1. This was due to more prevalence of alcohol addiction in males. Females showed chronic hepatitis to be common etiological factor.

Ultrasound features of liver cirrhosis

Increased parenchymal echogenecity of liver occurs in number of conditions, alcoholic steatosis, hepatitis, cirrhosis, being foremost causes. Fatty liver is condition in which triglyceride content is more than 5% of liver weight. This is due to increased delivery of fatty acids as there is increased synthesis and less oxidation. Alcohol, diabetes, obesity, pregnancy, Reye's syndrome, drugs (steroids) etc. malnutrition are some of its causative factors. Alcohol causes fatty change in liver and necrosis due to free radical injury, cytotoxic effect of aldehyde, increased activation of enzymes. As a reaction to necrosis, fibrosis results and as it affects entire liver leads to homogeneous & diffuse pattern. Many fibrotic – nonfibrotic and fat-nonfat interfaces lead to increased echogenecity i.e. seen in liver cirrhosis.[8]

Holmes one of the first to study liver by ultrasound, reported increased echogenecity in cirrhosis.[9]

Dewbury KC et al studied echopattern of liver in 67 cases of cirrhosis were found as 65% Bright liver (increased echogenecity) and 35% Normal echogenecity.[10]

In our study, raised echogenecity was found in 70% cases and normal echogenecity was seen in 30% cases. This is due to high number of alcoholic cirrhosis in our study.

In one case feature of raised echogenecity and coarse echopattern was found but on HPR it proved to be alcoholic hepatitis. The findings of raised echogenecity in cirrhosis in our study correlate with study carried out by Dewbury et al and Holmes et al.[9,10]

Surface nodularity of liver can be seen in conditions like cirrhosis malignancies, metastasis etc. Detection of nodularity depends on size of nodules, frequency of transducer and presence or absence of ascites. Ascites renders detection of nodularity to be more effective as it provides an echofree background.

Normal liver surface is seen as a hyper echoic line due to Glisson's capsule which is less than 1 mm thick. In micro nodular cirrhosis, this line is interrupted by fine nodules.[8]

Knobby contour of liver is seen in macro nodular cirrhosis.

Richard P. Bonniaud et al criterion of irregular hepatic outline to diagnose cirrhosis in 58 out of 72 patients. Sensitivity was 80.5% & specificity was 78.5%[11]

Free man et al studied irregularity of liver surface in cirrhotics and found knobby contour of liver.[12]

Alessandro Di Lelio et al studied liver surface irregularity with high frequency probe. Their study had sensitivity of 88% and specificity of 95%.[13]

Simonovsky V. et al studied 100 pts. of liver cirrhosis with 7.5 MHz probe. Sensitivity was 91.1% and specificity was 93.2%.[14]

In present study, surface nodularity was found in 67% cases while it was absent in 33% cases.

Alessandro Di Lelio et al diagnosed liver cirrhosis in 4 out of 7 patients. Recently Fukuda H. et al in Japan evaluated coarse echopattern in cirrhosis. They analysed the coarseness using neural network and devised coarse score. They found strong correlation between coarseness and liver cirrhosis.[13]

In our study coarse echopattern was found in 68 cases i.e. 68%.

Caudate to rt. lobe ration transverse diameter = / >0.65 is useful indication of cirrhosis. Caudate lobe has dual blood supply both from hepatic artery & portal vein. In cirrhosis due to fibrosis there is stenosis of branches of hepatic artery and portal vein branches. Hence the resistance to flow is increased. But as veins of caudate lobe have short course and they flow is increased. But as veins are distorted to lesser

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extent. Hence the ischemic insults of cirrhosis affect caudate lobe to a lesser extent.

Harbin et al used ratio of tansverse diameter of caudate lobe to rt. lobe to separate cirrhotics from non – cirrhotics. Their study had sensitivity of 84% and specificity of 100%.[15] Seitz JF et al diagnosed liver cirrhosis using criterion of caudate lobe enlargement with sensitivity of 73.46%.[16] Giorgion A et al studied cirrhosis with same criteria with sensitivity of 43% and specificity of 100%.[17] Hess CF et al studied role of C/RL ratio in liver cirrhosis and found positive results with sensitivity of 73.3%.[18]

In our study caudate lobe enlargement and C/RL ratio of >0.65 was found in 57% cases.

In cirrhosis due to fibrosis and cell necrosis there is gross volume reduction of liver. This leads to shrinkage of rt. lobe and left lobe and quadrate lobe. Lafortune M et al studied changes of quadreate lobe in liver cirrhosis. In controls the mean diameter was 4.3 cm $\pm$ 8 mm. In cirrhosis mean diameter was 2.8 cm $\pm$ 9 mm. They proposed it as useful adjunct sign in cirrhosis.[19]

Richard Bonniaud et al diagnosed cirrhosis in 58 out of 72 patients. Hepatomegaly was one of the useful criteria.[11]

Alessandro Di Lelio et al found that hepatomegaly was seen in 63% cirrhotics and 67% non – cirrhotics.[13]

In our present study, decreased liver size was seen in 47% cases using the standard method of measuring liver size in midhepatic line. Hepatomegaly was seen in 18% cases and normal liver size was seen in 35% cases. Hepatomegaly can also be caused by alcoholic steatosis, hepatitis, storage disorders etc.

Ultrasound features of portal hypertension

Portal vein diameter is normally less than 1.3 cm. It is increased in portal hypertension. In a study carried out by Bolondi L. et al portal vein diameter of >1.3 cm was alone used as criterion for diagnosing portal hypertension. It had sensitivity of 41.8%.[20]

According to Wienreb et al cirrhotic patients had portal vein diameter of 1.2 cm.[21] Iber FL et al showed portal vein diameter >1.3 cm to be 75% sensitive & 100% specific for portal hypertension.[22]

Vilgrain V et al showed portal vein diameter to be 40% sensitivity in diagnosing portal hypertension. In this study, portal diameter >1.3 cm was found in 15 out of 33 cases of portal hypertension i.e. 45.45% cases of portal hypertension. Portal vein diameter was found to revert back to normal after opening of large porto systemic collaterals.[23]

Splenomegaly occurs due to backpressure effect in portal hypertension. In gross splenomegaly, splenic radicles are also dilated.

Bolondi L et al showed splenomegaly to be present m 91.3% cases of portal hypertension.[20]

Martin – Herrera L et al in Spain studied prognostic usefulness of signs of portal hypertension. Splenomegaly with or with out portal vein dilatation formed a group with higher mortality rate.[24]

In our study splenomegaly was seen in 29 out of 33 cases of portal hypertension i.e. 87.87% which is comparable with previous studies.

With deep inspiration or valsalva maneuver, intra abdominal pressure increases and portal venous system distends. Diameter of splenic or superior mesenteric vein increases by 50-100%. In portal hypertension these veins are already maximally distended and also pressure changes are very minimally transmitted through fibrous, less compliant liver. Hence in portal hypertension due to cirrhosis the mild respiratory variation in caliber of splenic and superior mesenteric vein is lost.[25]

Bolondi L et al showed this finding in 78.5% and 88.4% of cases.[26]

In our study this finding was seen in 72.72% of cases of portal hypertension. This correlates with findings in previous study. Dilated splenic vein of > 1.2 cm is useful sign of portal hypertension. This occurs due to congestive effects of portal hypertension. Bolondi L et al showed this sign to be 79.7% sensitive and 100% specific is his study of 75 cases of portal hypertension[26] Author showed these findings of portal hypertension to have sensitivity of 79.7%.[20]

In our study it was found in 75.75% of cases. This is probably due to early cases of portal hypertension seen in our study.

Collaterals which form between portal and systemic circulation are effective means of decompressing high pressure portal system. Vilgrain E et al showed collaterals in 80% of cases with portal hypertension.[23]

Subramanyan B R et al showed collaterals in 88% cases of portal hypertension.[27]

In our study, collaterals were seen in 27 out of 33 cases of portal hypertension i.e. 81.81%. This correlates with previous studies. Many patients had multiple collaterals at the same time.

Complications of liver cirrhosis

Cirrhosis causes ascites by many mechanisms, that is hypo-proteinemia, colloid osmotic pressure, obstruction to lymphatic drainage etc. Minimal amount of fluid, 10-15 ml can be detected by ultrasonography in Morrison's pouch in supine position or in pouch of Douglas in erect posture. Fluid causes separation of bowel coils and freely floating coils give lollipop appearance.[8]

Trey C, Trey G et al showed ascites to be most common complication of cirrhosis.[28]

Our study correlates with these findings. In our study ascites was seen in 64% patients with liver cirrhosis.

Hepatocellular carcinoma is known complication of cirrhosis due to fibrosis and regenerating nodules.

Tremolda F et al found incidence of hepatocellular carcinoma to be 9.4% in cirrhotic livers.[29]

Male preponderance of hepatocellular carcinoma was proposed by Williams R et al. In our study incidence of hepatocellular carcinoma was 8% which was comparable with previous studies. Post – sinusoidal intrahepatic type of portal hypertension is seen in liver cirrhosis. Incidence of portal hypertension in our study was 33%.[30]

Bolondi L et al showed splenomegaly to be present in 91.3% cases of portal hypertension.[26] In our study incidence of splenomegaly was 87.87% which is comparable with previous study.

Gall bladder findings in cirrhotic liver include thickening of

gall bladder wall, gall stones & gall bladder varices.

Poor food intake, alcohol consumption result in spasm of sphincter of Oddi and edema of papilla of vater. Hypersplenism and hemolysis all contribute to formation of gall stones.[31] They are mostly pigment stones.

Acalouschi M et al found incidence of gall stones in cirrhotices to be 29.2.32 In our study incidence of gall stones is 22% which is comparable to previous study.

Gall bladder wall thickening due to hypoproteinemia and ascites is seen in cirrhosis.

Huang VS, Lee SD et al found thickened gall bladder wall in liver cirrhosis with ascites.[33]

Bronga A, Ferrara R et al studied 16 cirrhotic patients and 16 control cases. They found gall bladder wall thickening in 100% cases.[34]

In our study out of 64 cirrhotics with ascites, gall bladder wall thickening was seen in 60% cases i.e. 93.7% cases. This is comparable with previous studies. Gall bladder varices is seen in 2 cases. In both patients portal vein thrombosis was associated finding.

CONCLUSIONS

Ultrasound is a quick, noninvasive, easy, repeatable & effective method of diagnosing liver cirrhosis and its complications. The sensitivity of ultrasonography as a diagnostic tool is high and approaches to that of other investigations. Hence ultrasound would be continued to be one of the leading investigations in screening and diagnosing liver cirrhosis and its complications.

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