The Value of Using Echocardiography in Patients of Advanced Liver Disease with Cardio Pulmonary Complications

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ABSTRACT:

BACKGROUND:

Patients with chronic liver disease are liable to get cardio – pulmonary complications, one of these complications is development of pulmonary hypertension ranging from mild to it's sever form – this complication could give some abnormal findings in chest x-ray, electrocardiography but more prominently by echocardiography.

OBJECTIVES:

To clarify the benefit of using the echocardiogram is detecting pulmonary hypertension prior to the use of invasive methods (catheterization) in patients with advanced liver disease.

METHODS:

A total of 50 patients with chronic liver diseases (cirrhosis, chronic active hepatitis) their age range is (25-70 years) (mean are 37.07 years), the fifty patients were free from any cardiac or respiratory diseases.The study extending from December 2007-August 2008. All patients went through full history and routine blood test: including complete blood picture, fasting blood sugar, blood urea & screatinne, lipid profile, liver function tests, clinical examination, and investigations included routine blood test, chest x-ray, and electrocardiography, echocardiography (Transthoracie and transoesphageal). The child-Pugh score used for assessing the severity and prognosis of chronic liver disease and it classified into three groups (A, B, C) used in this study.

RESULTS:

Fifteen patients from the 50 cases (30%) only proved to have cardio-pulmonary changes (i.e. pulmonary hypertension right ventricular hypertrophy and dilatation with the mean value of ≥ 25mm Hg at rest or ≥ 30mm Hg during exertion) those patients were having fatigue in (70%) of them while dysponea, chest pain, cyanosis, syncope were detected in 30%, 10%, 1%, 2% respectively in those cases of pulmonary hypertension with chronic liver disease.

CONCLUSION:

The use of non invasive methods especially echocardiography were helpful in detecting the presence of pulmonary hypertension in patients with chronic liver disease.

KEYWORDS: echo, chronic liver disease

INTRODUCTION:

Pulmonary hypertension is the sustained elevation of mean pulmonary artery pressure (≥ 25mm Hg at rest or ≥ 30 during exertion) it is well recognized that there is a common association between liver and other systems (i.e. cardiac, pulmonary) in the form of hepato pulmonary association[1,2] pulmonary hypertension in one of these complications[3]. This disorder related to many factors as the development of recurrent thrombo-embolic events via porto-systemic shunts [2]-Also the liver factor. Another explanation for the development of pulmonary hypertension in chronic liver disease is the raised level of antinuclear antibodies in some families which raise the point of the presence of the associated dominant gene as factor for the development of pulmonary hypertension [6].

PATIENTS AND METHODS:

Fifty patients with chronic liver disease with suggestive evidence of secondary pulmonary hypertension were included in this study after excluding primary cardiac and pulmonary disorders. A complete history with full clinical examination have been done followed by complete list of blood tests including liver function tests, lipid profile, Fasting blood sugar, Burea and serum-creatinine. Chest x-ray, electrocardiography and echocardiography. The criteria for abnormal chest x-ray in this study were (cardiac enlargement, enlargement of pulmonary conus, pruning of

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peripheral blood vessels and increased interstitial lung markings with pleural effusion. The criteria for electrocardiography were (p-pulmonale, p-mitrale, right axis deviation, right ventricular dilation or hypertrophy and right bundle branch block. On studying the echocardiography the machine used was (HP 4500 echocardiography machine) and the parameters measured were (dilated left and main pulmonary artery, pulmonary regurgitation, right ventricular hypertrophy, right ventricular dilatation, tricuspid regurgitation and others if present using the continuous wave Doppler study (CW). The pulmonary artery systolic pressure measured by putting the cursor at the direction of the flow of blood at the Tricuspid regurgitation which represent the pressure difference between right ventricle and right atrium (normally is around 12mm Hg) Also exclude any underlying connective tissue disease serological tests like ANA anti DNA, anticardiolipin have been don for all patients.

RESULTS:

From the 50 cases of chronic liver disease (age range 25-70 yrs)were examined at the echocardiography unit only 15 patients (30%) were having (i.e. pulmonary hypertension) these 50 cases of chronic liver disease were catogerized into 40 cases 80% of cases as liver cirrhosis, 5cases (10%) as chronic active hepatitis (type B virus), 3 cases 6% chronic active hepatitis (type C Virus), 1 case 2% as auto immune hepatitis, 1 case 2% toxic hepatitis as shown in Table 1.

The 15 cases (30%) from the total number were having features suggesting pulmonary hypertension and distributed in table 2 according to the type of liver diseases as shown below.

Also these 15 cases (30%) are distributed according to the child –pugh staging as shown in Table3. The radiological findings in those cases of suggestives evidence of pulmonary hypertension were 4cases (8%) showed the presence of dilated pulmonary conus and another for 4cases (8%) showed right ventricular hypertrophy and dilatation an in table 4.

The presence of tricuspid regurgitation in 15 cases (30%) is significant with the presence of other echo findings which exclude the possibility of normal finding as show in table 5.

A comparison done between the echocardiography changes and radiological changes in these cases of hepato – pulmonary changes as shown in table 6. These tables indicate that the results of using echocardiographic changes are better than depending on radiological changes.

It has been showen in this study that of 5 cases (33%) from the cases were having dilated portal vein (i.e. > 13mm, Grey's Anatomy) as in shown in table 7. This table indicates that about ⅓ of cases have dilated portal rein which is an important point in support to the presence of pulmonary hypertension. Regarding the ECG changes in those patients with pulmonary hypertension secondary to chronic liver disease only 4 cases from the 15 cases showing signs of partial right bundle branch block (i.e. 8% from the 30% cases) have some ECG changes. These 4 cases were seen in those cases whom having right ventricular hypertrophy and dilatation group.

Table 1: Type of the disease/number I percentage of this study.

Type of Disease	number	%
liver cirrhosis	40	80%
chronic active hepatin B	5	10%
chronic active hepaitis C	3	6%
Auto immune hepatitis	1	2%
toxic hepatitis	1	2%
Total	50	100%

Table 2: Distribution of pulmonary hypertension according to the type of liver disease.

type of liver disease	Number of cases	% from the total
liver cirrhosis	12	24%
chronic active hepatitis B hepatitis	1	2 %
chronic persistent (C) hepatitis	2	4%
Total	15	32%

Table 3: Classification of chronic liver disease with pulmonary hypertension according to the child-Pugh staging.

Type of liver disease	stage A	stage B	stage C
live cirrhosis	2(4%)	3(6%)	7(14%)
chronic B hepatitis	1(2%)	0	0
Chronic c hepatitis	0	1(2%)	1(2%)

Table 4: The Radiological changes in those cases of hepatio – pulmonary disorders .

radiological changes	numbers cases	% from the Total
dilated pulmonary conus	4	8%
Right ventricular Hypertrophy and dilatation.	4	8%

Table 5: Echocardiography findings in cases with pulmonary hypertension.

Echocardiographic changes	Number and % of cases
dilated pulmonary conus (Lt main pulmonary, main pulmonary artery)	6 (12 %)
Tricuspid regurgitation	15 (30%)
Right ventricular hypertrophy and dilation	15 (30%)

Table 6: Comparison of echocardiogrphic and radiological changes in cases hepato pulmonary disorder

Pathological changes	Echocardiographic changes	Radiological changes
Dilated pulmonary conus	6 (12%)	4 (8%)
Right ventricular hypertrophy and Dilation	15 (30%)	4 (8%)

Table 7: Showing the prevalence dilated portal vein in pulmonary hypertension cases.

Number of cases	% of cases
5 Cases > 13 mm	33 %
10 Cases< 13 mm	66 %

DISCUSSION:

It has been found in this study that there is variation in patients symptoms in comparison with other studies. The main symptoms were fatigue 40% of the cases, syncope (25%) of case and by dysponea (10%) of cases. This does not fit with other studies in which dysponea was commoner than the others in correlation with the child pugh staging. It has been shown that cases whom having pulmonary hypertension were mainly are stage C (6) of the cases rather than in A and B.

This result in agreement with the study done by Waffaa AH et al[8] and Schen KP[3] et al and Alizadeh AH[10] Probably because of the severity advanced disease our studies showed the liver cirrhosis is the commonest presentation of pulmonary hypertension in comparison to hepatitis B or C which does not fit with the study done by Anand AC et al [11] who documented that hepatitis B is the commonest causes of cirrhosis and not fit with the study of waffaa Ah et al [8] which showed that hepatitis C is the commonest cause of liver cirrhosis. Regarding the echocardiographic changes of right ventricular hypertrophy and dilation with tricuspid regurgitation was seen in all 15 cases with measurement of gradient across tricuspid valve in all fifteen cases (100%) which support the presence of pulmonary hypertension making the result of this study fit with kuopc et al[12] study in which they showed that echocardiographic changes were seen in 90% of the cases of evidence of pulmonary hypertension, the echocardiographic changes in this study was very high in comparison to the x-ray changes and electro cardiographic changes (100% , 8 % and 4%) respectively . The x-ray changes was lower from the figure reported by Alizadeh AH study[10] by 82% this showed that echocardiography is the best parameter between others to diagnose pulmonary hypertension and to overcome the discrepancy of the results of and to overcome the discrepancy of the results of these studies.

CONCLUSION:

This study showed that echocardiography is the best non invasive method to diagnose pulmonary hypertension especially in places where catheterization in not available. Also from the other Aspect of this problem it is not practical to do cathctrization study to all patients with chronic liver disease.

REFERENCES:

1. Knowka Mj. pulmonary manifestation of chronic liver disease, schiffs, Disease of the liver, 8th Ed., Philadelphia: Williams and Wilkins. 1999, 489-502.
2. Yuan JX, Rubin LJ. Pathogenesis of pulmonary arterial hypertension: the need for multiple hits. *Circulation*. 2005; 111, 534-538.
3. Barst RJ, et al. Diagnosis and differential assessment of pulmonary arterial hypertension, *Am. J. cardiology*. 2004; 43, 405-475.
4. Ger D et al. pulmonary hypertension, the Washington manual of medical therapcutics 32nd Ed., 2007,245 -251.
5. Langeben D, Dupais j, et al. pulmonary endothelium -1- clearance in human pulmonary arterial hypertension. *Chest.* 2005; 128, 604-616.
6. Newman JH, et al. Genetic basis of pulmonary arterial Hypertension, current understanding and future *Am. J. Cardiology.* 2004; 43, 335-395.
7. Bonderman D, Fleischmann D, Prokop M, Klepetko W, Lang IM. Compression of the left main coronary artery by the pulmonary trunk in pulmonary hypertension. *Circulation*. 2002; 105, 256.
8. Waffaa Ah, Gadallah, et al. Acardiopulmonary diagnostic in liver cirrhosis. *Arab Journal of gastrocnterology* . 2007; 8, 20-23.
9. Schen kp, et al. prognostic significance of the hepato-pulmonary syndrome in patients with cirrhosis. *Gastroenterology.* 2003; 125, 1042-1052.
10. Alizadeh AH, et al. clinical feature of hepatopulmonary in cirrhotic patient's world. *Gastroenterology*. 2005, 1954-1956.
11. Anand AC, et al. hepatopulmonary syndrome, pervalance and clinical profile. *Indian Gastroenterology*. 2001; 20, 24-27.
12. Kupc, et al. Distinctive clinical feature of porto-pulmonary hypertension. Chest. 1997; 112, 980.