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A STUDY OF 31 PATIENTS WITH LIVER CIRRHOSIS IN BASRAH

Abbas A. Mansour* & Saad S. Hamadi@

*FICMS, Specialist Physician, Basrah Military Hospital. @FICMS, Assistant Professor, Basrah College of Medicine, Basrah; IRAQ.

Summary

This is a prospective follow up study designed to assess liver cirrhosis in Basrah. All cases included in the study were proven to have cirrhosis by biopsy. The total number of patients was 31, 25 males and 6 females; mean age 41.1 years. The commonest cause was hepatitis B virus (HBV) in 19.3% followed by hepatitis C virus (HCV) in 16.1% and 35.4% cryptogenic. We have 19.3% mortality, 29% compensated cirrhosis and 54.8% were child's class A, the commonest cause of liver cirrhosis was viral infection. One third of cirrhosis is cryptogenic.

Introduction

Liver cirrhosis is the irreversible results of chronic liver diseases[1]. Liver cirrhosis and its related complication continue to represent world wide health care burden[2].

In order to study liver cirrhosis in Basrah, we conducted this study. The aim was to know the causes, clinical presentations, complication and treatment of cirrhosis in Basrah.

Patients and Methods

This was prospective study for 31 patients with liver cirrhosis in Basrah started from January 1994-February 2001, all of the patients having clinical evidence of portal hypertension with esophageal varices and splenomegaly. The patients were attending the military and Basrah General Hospital. Liver cirrhosis was diagnosed by needle biopsy using Menghini procedure according to the WHO recommendations[3].

Hepatitis B virus (HBV) infection was diagnosed on the base of HBs Ag positive in the serum.

Hepatitis C virus infection (HCV) was diagnosed on the base of positive anti HCV antibodies detected by third generation ELISA in the serum. Recombinant immunoblot assay (RIBA) to confirm ELISA positive cases was not available, as is the PCR for detection of HCV RNA.

Alcoholic liver disease was diagnosed on the base of history of drinking more

Correspondence to:
Dr. Abbas A. Mansour
P.O. Box 142 Basrah; IRAQ
e-mail: a.a.m.b@uruklink...et

than 16 unites (160 grams) of alcohol daily for at least 5 years, with negative viral markers and autoantibodies[4].

Wilson's disease was diagnosed on the bases of Kaiser fleischer ring and low serum careuloplasmine.

Case of autoimmune hepatitis was diagnosed according to the Criteria of the International Autoimmune hepatitis Group for the diagnosis of autoimmune hepatitis[5].

Cryptogenic cirrhosis was diagnosed by exclusion of above causes with negative results of the available autoantibodies.

Patients were considered to have compensated cirrhosis, if they were a symptomatic, and discovered during routine clinical examination with normal biochemical tests, or mild increase in the transaminases.

Results

The total number of patients was 31. Duration of follow up range 1-6 years. Table I, showing the age and sex distribution. Table II, showing the causes of cirrhosis in our patients, who were HBV in 19.3 %, HCV in 16.1%, alcohol in 12.9% and 35.4% cryptogenic.

Age (y) mean 41.1 (range 12-70)			
Range (yr)	**No.**	**M**	**F**
12-29	11	7	4
30-50	9	8	1
51-65	10	9	1
>65	1	1	
total	31	25	6

Table I. Age and sex distribution.

Tables III, showing presentations, complications and mortality. Some have more than one presentation and complications, others have frequent attacks of same complications. The mortality rate was 19.3%, and we had 54.8% in Child's class A.

Cause	**No.**
Hepatitis B virus	6(19.3%)
Hepatitis C virus	5(16.1 %)
Cryptogenic	11(35.4%)
Alcoholic	4(12.9%)
Autoimmune	1(3.2%)
Wilson disease	2(6.4%)
Cardiac cirrhosis	1(3.2%)
Hepatitis C virus and alcohol	2(6.4%)

Table II. Causes of cirrhosis

Presentations and complications No. (%)
Jaundice 9(29%)
Ascites 13(41.9%)
Encephalopathy 6(19.3%)
Portal hypertensive gastropathy 13(41.9%)
GIT hemorrhage 8(25.8%)-6(19.3%) variceal
2(6.4%) from the stomach
Cyanosis 2
Spontaneous peritonitis 2
Hepatorenal syndrome 2
Hepatic pleural effusion 1(right sided)
Sever hyponatremia 2
Septicemia 1
Glomerulonephritis 1
Acquired porphyria cutanea tarda (alcoholic) 1.
Death 6(mortality rate 19.3%)
-Encephalopathy 4(1 with hematemesis, 1 with hepatorenal syndrome)
-Hepatocellular carcinoma(HCC) 1
-Bulbar palsy 1
Compensated cirrhosis 9(29%)
Child's class
Class A 17(54.8%), Class B 7(22.5%), class C 7(22.5)

Table III. Presentations, complications and Child's class.

Table IV, showing the associated diseases. While in Table V, we can see the treatment modality for our patients. Which was for ascites, salt restriction with diuretics, repeated paracentesis with, or with out albumin, or all of these modalities. Esophageal varices were treated with sclerotherapy for secondary prevention, but no primary prevention procedure done. β- blocker was used in secondary prevention in 5 patients and in primary prevention in 2.Only propranolol was used.

We have two patients in the cryptogenic group, one male and the other female were ANA positive, and they showed good response to steroid. The only one definite autoimmune cirrhosis was 16 years old female responds to steroid.

Disease	No.
Diabetes mellitus	3
Hypertension	3
Scleroderma	1
Pulmonary TB with TB spine	1
Staghorn renal stone	1
Congestive heart failure(CHF)	1

Table IV. Associated disease

Treatment modality	No.
Salt restriction with Diuretic	6
Paracentesis with or with out albumin	7
Sclerotherapy (All were secondary prevention)	5
Corticosteroid	3
-blocker (2 primary prevention,5 secondary prevention)	7
Interferon alfa-2b (1HBV,1HCV)	2
Ribavirin 1 (HCV)	
D-pencillamine 1	1

Table V. Lines of treatment

One female patient in her 40s was known case of scleroderma, with anti-mitochondrial antibody negative, and the liver biopsy was showing no bile duct injury, so considered as cryptogenic.

We had two patients with Wilson disease. Both were males .One presented with neuropsychiatric features (predominantly extrapyramidal signs) at the age of 17 years that was progressive followed by bulbar palsy and death due to aspiration pneumonia after five years of follow up. No D-pencillamine was given because it was not available. Liver biopsy showed liver cirrhosis. but he was lacking the florid stigmata of chronic liver disease. The second one was presented with pure liver disease with no neurological dysfunction at age of 25 years. He was treated with D-pencillamine that caused dramatic improvement.

One patient developed glomerulonephritis (Recurrent hematuria, dysmorphic RBC with cast), but no kidney biopsy was done for him.

Cryptogenic cirrhosis was seen in 11 patients.

We have one patient with Child's class C conceived but we terminated the pregnancy in her 6th month of gestation for the seek of the life of patient, and another one Child's class A, she delivered full term normal baby.

Discussion

The commonest cause of cirrhosis in our patients was HBV followed by HCV. The detection of hepatitis C Antibodies were by third generation ELISA. Recombinant immunoblot assay (RIBA) to confirm ELISA positive case was not available. HCV RNA detected by PCR was not available for detection of HCV. Therefore, we may miss some case of HCV infection.

HCV is the most common cause of post transfusion and community acquired non-A, non-B hepatitis, and cryptogenic cirrhosis worldwide[6].

Moreover, HCV is the commonest cause of cirrhosis in Italy and Japan, with cirrhosis that frequently a symptomatic followed by HBV[7,8].

Two of our patients having alcohol abuse with HCV. HCV is likely to play an important role in the pathogenesis of liver cirrhosis in alcoholism and their effect is synergetic, and alcohol misuse may enhance the replication of HCV[9,10,11].

The cirrhosis in HCV infection will take 2-4 decades to appear, so we are expecting to see more chronic liver diseases in future due to large number of blood transfusion in the Iran-Iraq war in 1980s[12,13].

We should admit that the high number of cryptogenic cirrhosis in our patients, may be due to lack (at some times) of availability of autoantibodies like

antimitochondrial, antismooth muscles antibodies, antinuclear antibodies and anti liver /kidney microsomal type 1 anti bodies, that's why we have 2 cryptogenic cirrhosis respond to steroid. Some of our patients may be variant forms of autoimmune hepatitis like over lap syndrome or outlier syndrome[5,14-16].

We have one patient with massive right-sided hepatic hydrothorax. Hepatic hydrothorax occurs in 1-5% of cirrhotic patients[17].

The mortality rate in our study was 19.3%, and the commonest cause of death was encephalopathy, while world-wide most patients with cirrhosis die of HCC now[18,19].

Variceal bleeding seen in our study in 19.3%. One third of cirrhotic will experience variceal bleeding over 10 years of follow up[20]. Of whom 2/3 will die. The most reliable predictor of bleeding from varices is their size, the larger the size the higher the risk of bleeding. Patients with large varices should be given propranolol as primary prevention. Carvidelol now a day is superior to propranolol in preventing variceal bleeding[21].

In patients with high risk esophageal varices, endoscopic ligation of varices is safe and more effective than propranolol for the primary prevention of variceal bleeding[22].

Portal hypertensive (congestive) gastropathy was seen in 41.9% of our patients. The prevalence of congestive gastropathy in cirrhotic patients is between 30%-70%[23]. Its more in those with large varices and severe liver disease. Of GIT hemorrhage in cirrhotic, 10-20% are caused by this condition, while in our study 6.4% bleed from gastropathy[2].

β-blocker are the treatment of choice. We under used antiviral and interferon in our study because of shortage of drugs, but again their use in cirrhosis stage is too late to be useful for both HBV and HCV infection[24,25].

We had 2 patients conceived. Pregnancy is feasible in cirrhotic patients regardless the Child's class, but there is a risk of prematurity and dysmaturity[26]. Bleeding from varices can be treated with sclerotherapy during pregnancy.

Conclusion

The commonest cause of cirrhosis in our study was HBV followed by HCV. We have a lot of cryptogenic case due to shortage of investigations.

Recommendations

We should:

1- Start screening for HCV in the community as a whole.
2- Detect chronic liver diseases in early stage.
3- Make Interferon and antiviral drugs available to be used when indicated.
4- Start primary prevention with β-blocker and/ or sclerotherapy for high-risk esophageal varices.
5- Have Further studies with better facilities to detect the exact causes of cryptogenic cirrhosis.
6- Stress on the importance of screening all blood before transfusion.

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