THE ROLE OF HEPATITIS C INFECTION IN A COHORT OF PATIENTS WITH LIVER DISEASE

Candan TUNCER, M.D., Seren ÖZENİRLER, M.D., Hüseyin ALKIM, M.D.,
Selahattin ÜNAL, M.D., Uğur KANDİLCİ, M.D.

Gazi University, Faculty of Medicine, Department of Gastroenterology and Hepatology, Ankara, Turkey
Gazi Medical Journal 6 : 109-112, 1995

SUMMARY : *The hepatitis C virus (anti-HCV) antibodies were investigated in 73 patients with chronic liver disease. The state of the hepatitis B virus (HBV) infection was also evaluated. Patients with chronic liver disease was distributed as follows : Cirrhosis 54, chronic active hepatitis 14, chronic persistent hepatitis 5.*

Nineteen patients who had pathology excluding chronic liver disease formed the control group. When the serologic testing for hepatitis B virus was taken into consideration, 50.68 % of HBV infection in chronic liver disease was encountered and 13.69 % of the patients had anti - HCV positivity. Multiple virus infection with HBV and HCV was found in 15.06 % of the patients. Fifteen cases (20.54 %), 14 of which had cirrhosis, did not reveal positive serologic test. In the control group HBV infection was observed in 36.84 % and no anti - HCV was detected. There was a history of 9.3 % of blood transfusion and / or operation in the patients with cirrhosis caused by HBV and, 75 % in the patients with cirrhosis caused by HCV. Moreover, there was blood transfusion in 28.5 % patients with cirrhosis who did not have positive serologic testing.

We concluded that HBV and HCV infection plays an important role in chronic liver disease. Anti - HCV prevalence in our patients who had blood transfusion history is lower than reported in the literature. In our country, other factors may play an important role in transmission of HCV.

Key Words : *Chronic Liver Disease, Anti-HCV Antibodies.*

INTRODUCTION

At the beginning of 1970s as blood donors were screened for hepatitis B, it was discovered that another agent was responsible for the majority of hepatitis cases caused by transfusion (9). In 1974, it was found that this was not hepatitis A and the disease was named as post-tranfusion non-A, non B hepatitis (8). The agent which was spread by parenteral transmission was later named as C virus (HCV). Fifty percent of non-A, non-B hepatitis caused by HCV, becomes chronic and in 25 % of the patients cirrhosis develops (2, 14). Patients undergoing transfusion of blood and blood products, hemodialysis patients, those who use intravenous drugs and undergoing tattooing are under a high risk (1, 3). In urine and semen samples, anti-HCV antibodies and HCV RNA could be detected (11). It can penetrate from the cracks and open injuries on the skin. Sexual and perinatal transmission are also considered (4, 12).

Many studies showed that HCV has an important role in the etiology of chronic liver disease

(CLD). In our country, where the viral agent is the most important cause in CLD, it is thought that HCV, like HBV, is an important threat as HCV antibodies are found.

This study was planned in order to investigate the anti-HCV prevalence in CLD, which was diagnosed and observed in our department and to scrutinize the state of the HBV infection again.

MATERIALS AND METHODS

The study was carried out with 73 patients who had CLD that was confirmed by means of clinical, biochemical, ultrasonografical, endoscopic and histopathologic observations. Fifty five of the patients were male, 18 of them were female and the average of age was 47.7. The distribution of CLD is as follows; cirrhosis 54, chronic active hepatitis (CAH) 14, and chronic persistent hepatitis (CPH) 5. Nineteen patients formed the control group. In this group, there were 13 males and 6 females and the average age was 50.3. Controls were composed of following patients : Diabetes mellitus 2, metastatic liver cancer 2, vena porta thrombosis 5, Gilbert 1, seconder sclerosing cholangitis 1, polycthemia vera 1, choledocolithiasis 1, pancreatitis 1, pancreatic cancer 1, liver steatosis 2, toxic hepatitis 2. Serum AST, ALT, akaline phosphatase, GGT, bilirubin values of the patients were measured by means of routine biochemical methods. Mean ALT levels were 56.35 IU in cirrhosis, 56.6 IU in CPH, 107.28 IU in CAH. The serologic markers HCV antibodies (anti HCV) were tested with second - generation ELİSA (ABBOTT laboratories).

RESULTS

The serologic viral markers and the prevalence of HBV and HCV infections are shown in table 1 and 2. When the other serologic markers of HBSAg and of HBV were taken into consideration, it was found that there was 50.68 % of HBV infection in CLD. 13.69 % of patients had positive anti-HCV antibodies. 15.06 % of the patients with CLD, had HBV+HCV together. 15 cases (20.54 %), 14 of which had cirrhosis, did not have positive serologic markers for HCV. Seven of these patients were alcoholic cirrhosis and eight of them didn't have positive serologic marker for HCV or alcohol abuse history. In the control group HBV infection was found in 36.84 % of the patients and they were negative for anti HCV antibodies.

DISCUSSION

In this study, all the patients with CLD who had liver cirrhosis (LC), CAH and CPH had the prevalence of HBV infection higher than HCV infection. In a study reported from Taiwan, patients with cirrhosis had 74.5 % of HBs Ag and 27.4 % of anti-HCV antibodies.

In our cases with LC, when 31.4 % of HBsAg and the other markers of HBV are taken into consideration, HBV infection is 46.2 % and anti-HCV is positive in 14.8 %. Still in the same study, while patients without liver disease who formed the control group had 16.6 % of HBsAg and 10.5 % of anti-HCV antibodies, in our control group HBsAg was 5.2 %, HBV infection was positive in 36.84 % and there weren't anti-HCV antibodies. In the control group, the state of HBsAg and of anti-HCV is compatible with the normal population.

The prevalence of HCV antibody in healthy population in different geographical areas are between 0.1 % and 5.2 % (6, 10). In another study, no anti-HCV antibodies in healthcare professionals were found (15). In our control group, the reason why HBV infection is high may be related to the group that formed. Some of the patients with CLD had

Serologic Testing	Cirrhosis (n=54)		CAH* (n=14)		CPH** (n=5)	
	n	%	n	%	n	%
HBsAg	17	31.4	7	50	3	60
anti HBc+ anti HBe	8	14.8	1	7.1	1	20
anti HCV	8	14.8	2	14.2	-	-
HBsAg+anti HCV	2	3.7	1	7.1	1	20
anti HBc+anti HCV	5	9.2	2	14.2	-	-
(-)	14	25.9	1	7.1	-	-

* CAH : chronic active hepatitis

** CPH : chronic persistent hepatitis

Table 1 : Serologic testing for anti HCV antibodies in CLD (Cirrhosis, CAH, CPH).

	HBV		HCV		HBV+HCV	
	n	%	n	%	n	%
Cirrhosis	25	46.2	8	14.8	7	12.9
CAH*	8	57.1	2	14.2	3	21.4
CPH**	4	80	-	-	1	20

* CAH : chronic active hepatitis
** CPH : chronic persistent hepatitis

Table 2 : HBV and HCV infection in CLD.

HBV and HCV infections together (15.06 %).

Dolar et al from Turkey reported anti-HCV antibodies in 26.92 % of the patients with, CLD, they found HBV and HCV together in 10.71 % of the patients (5).

We did not find any positive viral serologic marker in 25.9 % (15/54) of our patients who had LC and in 7.1 % (1/14) who had CLD. Liver disease in 7 of them was caused by chronic alcohol drinking. There was blood transfusion history in 2 of 8 patients who did not have positive serologic markers and who did not drink alcohol. Although anti-HCV antibodies was negative in these patients, it can be said that patients with cirrhosis caused by HCV may increase in case that HCV-RNA is examined.

In our study, there was 9.3 % of blood transfusion and/or operation in patients with cirrhosis caused by HBV and 75 % in patients with cirrhosis caused by HCV. There was anti-HCV in 8 of 12 patients (66.6 %) who had blood transfusion history. This seems to be lower than the proportion (76-89 %) informed in literature (7, 13). However, it is thought that blood transfusion and operations have an important role in transmision of HCV infection and in the devolopment of CLD.

According to our findings, HBV and HCV infection has an important role in the devolopment of CLD. In our country, it can be said that other factors excluding transfusion may play an important role in the etiology of CLD caused by HCV.

Since there is no vaccine for hepatitis C, it is necessary to screen blood donors regularly in order to prevent transmission of the infection and to make advanced observations widespread in order to bring up the existence of the infection in a reliable way.

Correspondence to : Dr.Candan TUNCER
Gazi Üniversitesi Tıp Fakültesi
Gastroenteroloji Anabilim Dalı
Beşevler
06500 ANKARA - TÜRKİYE
Phone : 312 - 214 10 00 / 5819

REFERENCES

1. Abildgaard N, Petersłund NA : Hepatitis C virus transmitted by tattooing needle (letter). Lancet 1991; 338 : 460.
2. Bruix J, Calvet X, Costa J, et al : Prevalence of antibodies to hepatitis C virus in spanish patients with hepatocellular carcinoma and hepatic cirrhosis. Lancet ii 1989; 1004-1006.
3. Calabrese G, Vagelli G, Guaschino R, Gonella M : Transmission of anti-HCV within the household of hemodialysis patients (letter). Lancet 1991; 338 : 1466.
4. Degos F, Maisonneuve P, Thiers V, et al : Neonatal transmission of HCV from mother with chronic hepatitis (letter). Lancet 1991; 338 : 758.
5. Dolar E, Acar Y, Ateş KB, et al : Kronik karaciğer hastalıklarında ve bazı risk gruplarında hepatit C virus antikor prevalansı. Gastroenteroloji 1992; 3 : 643-646.
6. El-Zayadi A, Selim O, Rafik M, El-Haddad S : Prevalence of hepatitis C virus among non-A, non-B related chronic liver disease in Egypt (letter). J Hepatol 1992; 14 : 416-417.
7. Esteban JI, Gonzales A, Hernandez JM, et al : Evaluation of antibodies to hepatitis C virus in a study of transfusion associated hepatitis. N Engl J Med 1990; 323 : 1107-1112.
8. Feinstone SM, Kapikian AZ, Purcell RH, et al : Transfusion - associated hepatitis without serological evidence of hepatitis type A or B. N Eng J Med 1975; 292 : 767-770.
9. Goldfield M, Bill J, Black H : Hepatitis associated with the transfusion of HBAg negative blood. In : Uyas GN, Perkins HA, Schmid R : eds. Hepatitis and Blood Transfusion. New York, Grune and Stratton 1972; pp. 353-361.
10. Klein RS, Freeman K, Taylor PE, Stevens CE : Occupational risk for hepatitis C virus infection among New York City dentists. Lancet 1991; 338 : 1539-1542.
11. Morris AJ, Rassam SW, Brown D, Dusheiko GM : Detection of HCV RNA in body fluids of patients with chronic hepatitis C infection. Hepatology 1992; 16 : 570.
12. Nishiguchi S, Kuroki T, Fukuda K, et al : Familial clustering of HCV (letter). Lancet 1992; 339 : 1486.

13. Roggendorf M, Deinhardt F, Rasshofer R, et al : Antibodies to hepatitis C virus (letter). Lancet ii : 1989; 324-325.
14. Tsai JF, Chang WY, Jeng JE, et al : Hepatitis B and C virus infection as risk factors for liver cirrhosis and cirrhotic hepatocellular carcinoma : a case - control study. Liver 1994; 14 : 98-102.
15. Turgut H, Turhanoğlu M, Aydın K, et al : Değişik gruplarda anti-HCV seropozitifliği, Gastroenteroloji 1992; 3 : 640-642.