



THE AGA KHAN UNIVERSITY

eCommons@AKU

Section of Gastroenterology

Department of Medicine

January 1996

Community acquired and post-transfusion hepatitis C "is there a difference?"

A R. Qureshi

Aga Khan University

S Hamid

Aga Khan University, saeed.hamid@aku.edu

W Jafri

Aga Khan University, wasim.jafri@aku.edu

F Ejaz

Aga Khan University

H Shah

Aga Khan University, hasnain.alishah@aku.edu

See next page for additional authors

Follow this and additional works at: https://ecommons.aku.edu/pakistan_fhs_mc_med_gastroenterol



Part of the [Gastroenterology Commons](#)

Recommended Citation

Qureshi, A. R., Hamid, S., Jafri, W., Ejaz, F., Shah, H., Abbas, Z., Abid, S., Khan, H. (1996). Community acquired and post-transfusion hepatitis C "is there a difference?". *Journal of Pakistan Medical Association*, 46(1), 9-11.

Available at: https://ecommons.aku.edu/pakistan_fhs_mc_med_gastroenterol/170

Authors

A R. Qureshi, S Hamid, W Jafri, F Ejaz, H Shah, Z Abbas, S Abid, and H Khan

Community Acquired and Post-Transfusion Hepatitis C "Is There a Difference?"

Pages with reference to book, From 9 To 11

A. R. Qureshi, S. Hamid, W. Jafri, H. Shah, Z. Abbas, S. Abid, H. Khan (Departments of Medicine, The Aga Khati University Hospital, Karachi.)

F. Ejaz (Departments of Pathology, The Aga Khati University Hospital, Karachi.)

Abstract

We analyzed 77 consecutive hepatitis C antibody positive patients to compare the history, laboratory data and histological features of community acquired (CA) and post-transfusion (PT) hepatitis C. Forty-six patients had "CA" and 31 "PT" hepatitis C. Mean age in both groups was same (45.67 vs 46 years). Male to female ratio was 2:1 in the CA group and 1:2.4 in the PT group. Mean duration between jaundice and first presentation was 8.9 years in the CA group and the mean duration between transfusion and first presentation was 9.8 years in PT group. No significant difference was found between two groups in the laboratory data. Liver biopsy was done in 32 patients (19 CA and 13 PT group). Mean histological score for disease activity was 9.3 in both groups, although more (68%) patients in the CA group had cirrhosis with chronic active hepatitis, (CAH) as compared to the PT (54%) group. Hepatitis C is an important cause of CA hepatitis. PT hepatitis C is more common in females because of increased likelihood of receiving transfusion for obstetric and gynaecological reasons. There is no significant difference in the laboratory and histological features between CA and PT hepatitis C (JPMA 46:9, 1996).

Introduction

NonA NonB hepatitis was first recognised in 1974¹. In mid 70's, it was demonstrated that 5-12% of cases developed hepatitis after transfusion of hepatitis B surface antigen negative blood. Among these patients, 90% were diagnosed to have Non A Non B hepatitis^{2,3}. The causative agent for the great majority of cases of Non A Non B hepatitis was discovered recently and named Hepatitis C virus (HCV)⁴. The subsequent development of antibody tests for HCV have made possible the easy diagnosis and the study of this infection⁵. Now hepatitis C is recognized to be a common disease with a world-wide distribution and accounts for more than 90% of post-transfusion hepatitis⁶. The current estimated incidence of acute infection among transfusion recipients is from five to ten percent⁷.

Although, initially regarded mainly as a blood-borne infection, due to the similarities with hepatitis B, a significant proportion of patients with HCV infection have no known risk factors for disease transmission⁸. This "community acquired HCV infection" is thought to differ from post-transfusion HCV (PT- HCV) in being more slowly progressive and to perhaps affect an older population⁹.

Chronicity is a common feature of both forms of hepatitis C and chronic hepatitis develops in 50% of these patients and cirrhosis in 20%⁹⁻¹¹

This study compares the features of community acquired and post transfusion hepatitis C with respect to demographic data clinical presentation laboratory data and histological findings.

Patients and Methods

Seventy-seven consecutive antibody positive patients were analyzed. They were seen either at their

outpatient visit or during their admission. A profonna filled for each patient, included demographic data, principal symptom on first presentation, histozy of blood transfusion and post transfusion jaundice and past history of jaundice. Also recorded were the risk factors for hepatitis C. like history of drug abuse, I.V injections, occupational exposure or sexual exposure to prostitutes. Laboratory data included a complete blood count, liver function test, hepatitis serology and histological features where available. Patients with a clear history of blood transfusion were included in the post-transfusion groups and those without as “community acquired hepatitis C”. Also included in this group were patients with a history of extra marital sexual contact or I.V. drug abuse.

The results are expressed in percentage incidence and rmean±SD. Chi-square tests were used to identify significant differences among the groups. P value <(105 are considered to be statistically significant.

Results

Offl patients, 46 had CA hepatitis C while 31 had post-transfusion hepatitis C. This was based on the history of transfusion in the past.

Table I. Demographic data.

| Characteristics | CA Hepatitis C (n=46) | Post-transfusion HC (n=31) |
|-----------------|--------------------------------|-------------------------------|
| Age (Years) | <i>U. + SD.</i> 45.67±12.67 | 46.00±12.77 |
| Sex (%) | | |
| Male | 30 (65.2) <i>n(%)</i> | 9 (29.03) |
| Female | 16 (34.8) | 22 (70.97) |
| Residence (%) | | |
| Urban | 35 (70.08) | 28 (90.3) |
| Rural | 11 (29.92) | 3 (9.7) |

Table 1 compares the demographic data of the two groups. A significantly higher proportion of females had post transfusion hepatitis C (p<0.005).

Appmximately 60% of patients from both groups were out patients and the remaining in-patients. The major symptoms at first presentation are listed in Table II.

Table II. First presenting symptoms.

| Presenting symptoms | CA Hep. C (%) | PT Hep. C (%) |
|--|---------------|---------------|
| General constitutional symptoms (e.g. fatigue, nausea, anorexia, lethargy etc.) | 17 (36.96) | 11 (35.48) |
| Variceal bleed | 12 (26.08) | 8 (25.80) |
| Confusion, drowsiness and coma (Hepatic encephalopathy) | 7 (15.22) | 2 (6.46) |
| Ascites/SBP | 5 (10.87) | 2 (6.46) |
| Incidental finding | 5 (10.87) | 5 (16.13) |
| Jaundice | 00 | 3 (9.67) |

SBP= Spontaneous bacterial peritonitis.

About 35% patients in both groups had general constitutional symptoms at first presentation while 10-15% in both groups were diagnosed incidentally i.e., either at surgery or they presented with some other problems and on testing were found to have an elevated aminotransferase level and later found to be HCV antibody positive.

Twenty-seven patients had a definite past history of jaundice in the "CA" group and the range of duration between jaundice to first presentation was 1-30 years and the mean duration between jaundice and presentation was 8.9 years. Forty-one percent patients had no prior history of jaundice and among these patients, 5 had a history of contact with a patient of chronic liver disease or hepatitis in the immediate family and 3 (6.52%) had a history of extra marital sexual contact. There were no drug addicts in this cohort.

Thirty-one patients had a history of blood transfusion in the past and the duration between transfusion and first presentation was 1-27 years and the mean duration was 9.8 years. Only 6 had a history of post-transfusion jaundice and all of these patients had jaundice within first 3 months of blood transfusion. A comparison of laboratory data is shown in Table III.

Table III. Laboratory data.

| Investigations | "CA" Hepatitis C | PT Hepatitis C |
|---------------------------------------|------------------|----------------|
| Haemoglobin (gm%) | 11.73±2.49 | 11.82±2.28 |
| Platelet count (x10 ⁹ E/l) | 145.30±117.02 | 146.84±99.16 |
| Bilirubin (mg%) | 2.83±3.94 | 2.46±3.69 |
| SGPT (IU/L) | 127.04±92.21 | 140.94±206.98 |
| SGOT (IU/L) | 70.70±117.82 | 80.35±88.85 |
| Alkaline phosphatase (IU/L) | 142.87±93.59 | 150.13±94.98 |
| Albumin (gm/dl) | 2.44±1.06 | 2.83±0.94 |
| Globulin (gm/dl) | 3.51±1.39 | 4.00±1.18 |
| PT (sec.) | 5.37±3.84 | 5.18±6.68 |

p value = Not significant

Minimal level of haemoglobin, platelet count and albumin and the maximum level of bilirubin, SGPT, SGOT, alkaline phosphatase, globulin and prothrombin time are analyzed for each patient. All these figures are expressed as mean±standard deviation.

Hepatitis B surface antigen and HCV antibody were done in all and both were positive in only three patients, indicating HB V/HCV co-infection. Hepatitis B core antibody IgG was done in 58 patients and was found reactive in 28 patients.

Liver biopsy was done in 32 patients. Nineteen had CA hepatitis C and 13 had PT hepatitis C. The histological features were compared and histological activity index¹² was calculated and compared in both groups. The comparison of histological features is shown in Table IV.

Table IV. Comparison of histological features.

| Histological features | CA Hep. C (%) | PT Hep. C (%) |
|----------------------------------|---------------|---------------|
| Bridging necrosis | 9 (47.37) | 8 (61.54) |
| Piecemeal necrosis | 17 (89.47) | 13 (100) |
| Fatty change | 9 (47.37) | 7 (53.84) |
| Lymphoid follicle aggregation | 14 (73.68) | 12 (92.31) |
| Fibrosis | 14 (73.68) | 7 (53.84) |
| Sinusoidal lymphocytosis | 9 (47.37) | 8 (61.54) |
| Sinusoidal hyperplasia | 9 (47.37) | 11 (84.61) |
| Bile duct proliferation | 9 (47.37) | 5 (38.46) |
| Bile duct destruction | 9 (47.37) | 8 (61.54) |
| Cholestasis | 1 (5.25) | 1 (7.69) |
| Histological diagnosis | | |
| Cirrhosis with CAH | 13 (68.40) | 7 (53.84) |
| CAH | 4 (21.10) | 6 (46.16) |
| Chronic persistent hepatitis | 1 (5.25) | 00 (-) |
| Normal | 1 (5.25) | 00 (-) |
| Mean histological activity index | 9.3 | 9.3 |

CAH = Chronic active hepatitis

Discussion

This study raises several important issues related in patients with chronic HCV infection. In majority of patients like in other studies, the mode of transmission of HCV is not clear¹³. Agents possibly implicated in this inapparent transmission of HCV are parental injections with unsterile needles as is common practice in our county, arthropod vectors like mosquitoes, sexual or household exposure etc. However, there is no conclusive proof so far for any of these factors and further studies are required in

this area.

There was no difference in the mean age of presentation between the two groups. Although a number of patients in both groups did not have a history of jaundice, the mean duration between the onset of jaundice to presentation was similar in both groups at around 9 years. Both these figures suggest that CA-HCV may not be as indolent a disease as thought to be and does not seem to have a very different natural history as compared to PT-HCV⁹. The mean duration to presentation at 9 years also suggests that in our group of patients with CA-HCV, exposure is likely to have occurred in the late 20's or early 30's and that early childhood exposure seems unlikely, given that the mean age of presentation in our patients is around 46 years.

Majority of patients with PT-HCV were females. Most of these transfusions were given for obstetric or gynaecological causes. The facilities of screening of blood for HCV are available at only few centres in our country, therefore, most transfused blood is untested for HCV.

In summary, liver disease caused by chronic HCV infection is an important problem in our setting and in the majority of these patients the route of transmission is not known. Until this is clarified, common sense precautions like the use of sterile needles and effective protection against sexual transmission are needed. Blood should be screened for HCV and the need for a transfusion should always be very carefully assessed, particularly so in places where HCV testing is not yet available.

References

1. Prince, AM., Brotman, B., Grandy, G F et al. Long incubation post-transfusion hepatitis without serological evidence of exposure to hepatitis B virus. *Lancet*, 1974;11:241-6.
2. Feinstone, SM., Kapikian, AZ., Purcell, RH. et al. Transfusion associated hepatitis not due to viral hepatitis type A or B. *N. Engl. J. Med.*, 1975;282:767.
3. Knodell, R.G., Conard, M.E., Dienstag, J.L. et al. Etiological spectrum of post-transfusion hepatitis. *Gastroenterology*, 1975;69: 1278-1285.
4. Choo, Q.L., Kuo, G, Weiner, A.J. et al. Isolation of a DNA clone derived from a blood-borne non-A, non-B viral hepatitis genome. *Science*, 1989;244:359-62.
5. Kuo, G., Choo, Q.L., Alter, H.J. et al. An assay for circulating antibodies to a major etiologic virus of human non-A Non B hepatitis. *Science*, 1989;244:362-64.
6. Dienstag, J.L. Non A, Non B hepatitis. I. Recognition, epidemiology and clinical features. *Gastroenterology*, 1983;85:439-62.
7. Alter, H.J. Transfusion associated non A non B hepatitis the first decade. In: Zuckerman AJ. ed. *Viral hepatitis and liver disease*. New York, Alan R. Liss, 1988, pp. 537-42.
8. Alter, H.J., Purcell, RH., James, W. et al. Detection of antibody to hepatitis C virus in prospectively followed transfusion recipients with acute and chronic non A, non B hepatitis. *N. Engl. J. Med.*, Nov. 30:1989;321 -22:1494-1500.
9. Alter, M.J., Margolis, H.S., Krawczynski, K. et al. The natural history of community - acquired Hepatitis C in the United States. *N. Engl. J. Med.*, 1992;327: 1899-1905.
10. Koretz, R.L., Stone, O., Mousa, M. et al. Non A Non B post transfusion hepatitis - a decade later. *Gastroenterology*, 1985;88: 1251-4.
11. Secfl LB., Buskell-Bales, Z., Wright, E.C. et al. Long-term mortality after transfusion associated non-A, non-B hepatitis. *N. Engl. J. Med.*, 1992;327:1906-11.
12. Knodell, R.G., Ishak, KG., Black, W.C. et al. Formulation and application of a numerical scoring system of assessing histological activity in asymptomatic chronic active hepatitis. *Hepatology*, 1981; 1:431-35.
13. Alter, M.J. Inapparent transmission of Hepatitis C: Footprints in the sand. *Hepatology*, 1991;14:389-91.