DOI: https://doi.org/10.53350/pjmhs221641201

ORIGINAL ARTICLE

Prevalence of Viral Hepatitis and its Variable Outcome Among Diabetic Patients

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ABSTRACT

Objective: Evaluation of the clinical and biochemical characteristics among diabetes and non-diabetic individuals suffering from acute viral hepatitis is the focus of this particular research.

Study Design: Prospective/Observational study

Place and Duration: Social Security Teaching Hospital Ferozepur Road Lahore**.** April, 2021 to Sep, 2021

Methods: This research includes 140 individuals of acute viral hepatitis. Cases in the study ranged in age from 20 to 72 years old. Following signed and informed permission, demographic information, such as a patient's BMI (Body Mass Index), dwelling address, and educational status, were gathered. Patients were divided into two categories. 70 non-diabetic patients were included in group A and 70 diabetic patients with acute viral hepatitis were in group B. In both groups, smoking history was caculated. Both groups of patients had their clinical and biochemical indicators monitored throughout their hospitalization. Analysis was performed by using SPSS 24.0.

Results: In current study males were significantly higher in numbers than females with p value 0.005. In both groups, hepatitis E was the most prevalent cause, followed by B and A. There were considerably greater mean blood bilirubin levels in diabetics than in nondiabetics, as well as lower ALT and albumin levels. Diabetic patients spent an average of 22.5±7.45 days in the hospital compared to 11.4±6.45 days for non-diabetic patients, with a p value of 0.03. Group B had a death rate of 4 (5.7%), due to liver failure, whereas group A had no mortality.

Conclusion: In the present study, we found that diabetes patients with acute viral hepatitis had lower levels of ALT, higher levels of bilirubin, a larger risk of liver failure, and longer hospital admissions than non-diabetic patients with the same condition. Except this death rate in diabetic patients was 5.7%.

Keywords: Clinical Outcomes, Diabetes, Mortality, Acute Viral Hepatitis

INTRODUCTION

Worldwide, viral hepatitis with long-term consequences is a serious hazard to public health. HBV and HCV are the major causes of cirrhosis and hepatocellular carcinoma, both of which have a growing mortality and burden of illness in emerging countries[1]. Patients with persistent HCV infection may be more likely to develop diabetes as a consequence of end-stage liver disease since the liver plays an important role in glucose metabolism. As another significant public health issue, determining whether chronic viral hepatitis is linked to an elevated risk of diabetes before the onset of end-stage liver disease is very essential.[2]

The link between HBs Ag positivity and diabetes incidence has only been explored in a few longitudinal studies[3,4], none of which indicated a significant correlation. Although the number of participants in these trials was modest and they had various degrees of HBV-related liver illness, they found no correlation between positive HBsAg serology and diabetes. A study published in the Journal of Clinical Endocrinology and Metabolism found a link between HCV antibodies (HCV Ab) and diabetes risk, despite the fact that many of these studies were not prospective, had small sample sizes, or included patients with liver disease that can increase the risk of diabetes. Hepatitis C virus (HCV) is an RNA virus of the Flaviviridae family that is transmitted via the blood. [5-10]

The prevalence of hepatitis C (HCV) fluctuates depending on the population and other variables. HCV may be transmitted by blood transfusions, intravenous drug use, inadequate sanitation of medical equipment such as dental tools, tattoos, and medical injections, among other prevalent routes. It may also be passed from mother to child, however this is a less usual occurrence. A person may be infected with various genotypes of the HCV, although this is very uncommon.[11] A patient's genotype and the degree of liver damage influence his or her treatment options. Between two weeks and six months is the incubation time for the HCV virus to take hold. Many people have no symptoms at all. Symptoms that are seen in the acute stage include: high or low fever, reduced appetite, tiredness, nausea, vomiting, joint pain, dark or grey urine, and jaundice. Since acute HCV infection is frequently asymptomatic, early detection of the disease is very rare. Anti-HCV antibodies in the blood are the primary method for diagnosing HCV, however a nucleic acid test for the RNA of HCV is also used to confirm chronic HCV infection. Chronic hepatitis C (HCV) patients have an increased risk of developing type II diabetes mellitus (DM) compared to healthy individuals (13-33 percent). Cirrhosis of the liver was associated with a greater incidence of type 2 diabetes in comparison to chronic hepatitis [12]. Diabetes mellitus (DM) is a long-term metabolic condition characterized by unstable glucose homeostasis.[13]

A person with diabetes has either type 1, which is hereditary, or type 2, which happens when the pancreas is unable to generate enough insulin and the body is unable to properly react to insulin's actions.[14] Adipose tissue, the liver, and muscles all benefit from insulin's ability to facilitate glucose absorption. Glycogen storage is aided by insulin because it inhibits hepatic glucose production while also increasing peripheral glucose absorption. Insulin resistance (IR) is defined by a decreased ability of certain tissues to respond to normal insulin levels. Blood glucose levels rise as a result of DM, causing damage throughout the body. The symptoms of DM might be apparent, subtle, or even completely missing from time to time. A wide range of health consequences, including diabetes-related retinopathy, neuropathy, heart failure, and foot disease, may be linked to DM. Metformin is the medicine of choice for treating patients with persistent HCV infection who have diabetes mellitus (DM). [15]

For this study, researchers compared the aetiological pattern, clinical and biochemical picture, and the outcome of acute viral hepatitis in diabetics and non-diabetics.

MATERIAL AND METHODS

This prospective study was conducted at Social Security Teaching Hospital Ferozepur Road Lahore and comprised of 140 patients.

After receiving written permission from the patient and receiving informed consent from the patient, a patient's demographic information was gathered. This information included the patient's age, gender, BMI (Body Mass Index), domicile, and education status. In addition, patients who had a recent history of acute hepatitis, a history of severe alcohol use, likely ischemic hepatitis, or who were pregnant were not allowed to participate in the trial.

The ages of the participants ranged from 20 to 72 years. Following the patient's admission, a semi-structured questionnaire was utilised to perform a bedside interview. This questionnaire included questions on the patient's medical history, physical examination, and the findings of any pertinent investigations. Serum samples from each patient revealed the presence of HBs Ag, IgM anti-HBc, IgM anti-HIV, and IgM anti-HAV. It was decided to employ the CMIA chemiluminescence immunoassay, as well as IgM anti-HBc and IgM anti-HAV, to determine the presence of HBs Ag. Testing for anti-HEV antibodies using IgM antibodies has now been completed. We monitored all of them for at least a year, if not longer, or until they had recovered completely, whichever happened first. Every patient had an upper gastrointestinal endoscopy done as a preventative measure.' Additional abdominal ultrasounds were performed on all patients as well. To establish the extent of fibrosis in the patient's liver, a fibroscan was also done.

Patients categorized in two groups. 70 non-diabetic patients were included in group A and 70 diabetic patients with acute viral hepatitis were in group B. The smoking histories and the underlying causes of both groups were investigated. During their time in the hospital, patients in both groups had their clinical and biochemical characteristics compared and contrasted. All of the data was analyzed using SPSS version 24.0.

RESULTS

In group A males were 42 (60%) and in group B 45 (64.3%) were males. Mean age of the patients in group A was 35.15±9.88 years and had mean BMI 23.11±16.45 kg/m^2 while in diabetic group mean age was 40.9±12.63 years with mean BMI 26.7±5.72 kg/m^2. In group A 30 (42.9%) cases were from rural areas and in group B 29 (41.4%) patients were from rural areas. Majority of the patients were married and were educated among both groups.

Table-1: Information about the enrolled cases

Variables	Non-Diabetic	Diabetic
Mean Age (years)	35.15±9.88	40.9±12.63
Mean BMI (kg/m^2)	23.11±16.45	26.7±5.72
Gender		
Male	42 (60%)	45 (64.3%)
Female	28 (40%)	25 (35.7%)
Residency		
Rural	30 (42.9%)	29 (41.4%)
Urban	40 (57.1%)	41 (58.6%)
Marital Status		
Yes	38 (54.3%)	43 (61.4%)
No	32 (45.7%)	27 (38.6%)
Literacy		
Yes	47 (67.1%)	44 (62.9%)
No	23 (32.9%)	26 (37.1%)

We found that hepatitis E was the most common etiology among both groups found in 42 (60%) in group A and 52 (74.3%) in group B followed by hepatitis B in 15 (21.4%) cases of group A and 11 (15.7%) patients in group B. Hepatitis A found in 13 (18.6%) cases of group A and 7 (10%) patients of group B. (table 2)

Table-2: Comparison of etiology among both groups

Variables	Non-Diabetic	Diabetic
Hepatitis		
E	42 (60%)	52 (74.3%)
B	15 (21.4%)	11 (15.7%)
A	13 (18.6%)	7 (10%)
Total	70 (100)	70 (100)

There were considerably greater mean blood bilirubin levels 18.325±4.342 mg/dl in diabetics than in nondiabetics 8.637±9.249, as well as lower ALT 836.10±43.6 U/L and albumin levels 602.16±25.19 U/L in diabetic group as compared to non-diabetic group 1241.10±176.8 U/L and 1054.39±311.9 U/L .(table 3)

Table-3: Examination of the similarities and differences in biochemical characteristics between the two groups

Variables	Non-Diabetic	Diabetic	P value
Biochemical Parameters			
Bilirubin (mg/dl)	8.637±9.249	18.325±4.342	0.03
ALT (U/L)	1241.10±176.8	836.10±43.6	0.04
Albumin (mg/dl)	1054.39±311.9	602.16±25.19	0.02

Diabetic patients spent an average of 22.5±7.45 days in the hospital compared to 11.4±6.45 days for non-diabetic patients, with a p value of 0.03. Group B had a death rate of 4 (5.7%), due to liver failure, whereas group A had no mortality.(table 3)

Table-3: Comparison of hospital stay and mortality among both groups

Variables	Group A	Group B
Hospital stay (days)	11.4±6.45	22.5±7.45
Deaths		
Yes	0	4 (5.7%),
No	70 (100%)	66 (94.3)

DISCUSSION

The illness known as acute viral hepatitis (AVH) is often associated with complete spontaneous clinical, biochemical, and viral recovery within four to six weeks after the time it first manifests itself. Even while AVH was formerly believed to pose no health risks, new evidence gleaned through expanded study and thorough observation of people living with the condition suggests that this may not be the case. In one percent to five percent of patients diagnosed with AVH, additional complications such as acute liver failure (ALF), subacute hepatic failure (SHF), or a prolonged icteric phase have been discovered. It is recognised that some viral etiologies and host characteristics, such as the patient's immunological status, age, and the presence of an underlying chronic liver disease, play a part in the complicated natural course that the illness takes. [16]

This research includes 140 individuals of acute viral hepatitis. Cases in the study ranged in age from 20 to 72 years old. Males were significantly higher in numbers than females with p value 0.005. Our research produced results that were similar to those of earlier studies.[17,18] Mean age of the patients in group A was 35.15±9.88 years and had mean BMI 23.11±16.45 kg/m^2 while in diabetic group mean age was 40.9±12.63 years with mean BMI 26.7±5.72 kg/m^2. In group A 30 (42.9%) cases were from rural areas and in group B 29 (41.4%) patients were from rural areas. Majority of the patients were married and were educated among both groups.[17] The prevalence of diabetes was shown to be greater than expected in patients had HBsAg (-), but not in patients of HBsAg (+)in several investigations. [18,19] HBV infection was not related with diabetes or glucose intolerance in a 10-year prospective analysis confined to people without cirrhosis, although this investigation was constrained by a small sample size.[20] The large sample size, meticulous laboratory procedures, and capacity to account for many baseline factors, including the existence of fatty liver disease, enabled us to discover a strong link between HBsAg and diabetes in both cross-sectional and incident analyses.

We found that hepatitis E was the most common etiology among both groups found in 42 (60%) in group A and 52 (74.3%) in group B followed by hepatitis B in 15 (21.4%) cases of group A and 11 (15.7%) patients in group B. Hepatitis A found in 13 (18.6%) cases of group A and 7 (10%) patients of group B. [21] Our results that HCV Ab (+) and diabetes go hand in hand are consistent with the vast majority of past studies on the subject. For diabetes, a meta-analysis reported an OR of 1.68 comparing those with and without HCV infection. [22] Chronic HBV or HCV infection

and elevated liver enzyme levels were highly associated to diabetes in one research, even though there were no differences between the two infections in the analysis. [23] Cirrhosis and age 65 or older are risk factors for diabetes in patients with chronic HCV infection, according to an Italian research. [24] It's no surprise that illnesses of the liver, including liver cirrhosis and hepatocellular carcinoma, may disrupt glucose homeostasis, given the liver's critical involvement in glucose metabolism. [25]However, it is unknown what processes are at play when viral hepatitis without cirrhosis causes glucose metabolism to become disrupted. Chronic HBV or HCV infection and high levels of liver enzymes were shown to be associated with an increased risk of diabetes in one research, with no differences between the two infections in terms of the degree of liver fibrosis seen. [26] Chronic hepatitis C virus (HCV) patients' diabetes was solely connected with cirrhosis and advanced age, according to research by an Italian study. [27]

Alterations in glucose metabolism in patients with hepatitis C (HCV) have been linked to other processes. In experiments, HCV has been shown to directly interfere with insulin signalling in infected hepatocytes. Which may be driven by genotype-specific mechanisms. HCV may also affect glucose metabolism indirectly by developing peripheral insulin resistance in the muscles of chronic HCV patients.[28,29] Chronic HBV infection has been linked to the development of type 2 diabetes in several studies, however the role of HBV in the development of diabetes remains unclear. Understanding how HBV and HCV patients' glucose metabolism is affected requires more study. [30]

There were considerably greater mean blood bilirubin levels 18.325±4.342 mg/dl in diabetics than in nondiabetics 8.637±9.249, as well as lower ALT 836.10±43.6 U/L and albumin levels 602.16±25.19 U/L in diabetic group as compared to non-diabetic group 1241.10±176.8 U/L and 1054.39±311.9 U/L Diabetic patients spent an average of 22.5±7.45 days in the hospital compared to 11.4±6.45 days for non-diabetic patients, with a p value of 0.03. Group B had a death rate of 4 (5.7%), due to liver failure, whereas group A had no mortality. Tests on the liver and liver biopsies revealed more severe liver damage in diabetes individuals with HCV. Diabetes may not be linked to HCV infection itself, but the chronic inflammation in the liver after infection may. A dysfunction of the -cells is caused by early chronic HCV infection, although diabetes does not emerge until cirrhosis has taken hold. [31]

CONCLUSION

In the present study, we found that diabetes patients with AVH had lower levels of ALT, higher levels of bilirubin, a larger risk of liver failure, and longer hospital admissions than non-diabetic patients with the same condition. Except this death rate in diabetic patients was 5.7%.

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