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Evaluation of Some Micronutrients of Hepatitis (HBV and HCV Positive) Subjects

Omolumen LE[1], Iyevhobu KO[2,*], Babatope IO[3], Aliu II[4], Ediale SE[5] and Imoren RA[1]

[1]*Department of Chemical Pathology, Faculty of Medical Laboratory Science, Ambrose Alli University, Ekpoma, Edo State, Nigeria*
[2]*Department of Public Health, National Open University of Nigeria, Uromi Community Study Centre, Uromi, Edo State, Nigeria*
[3]*Department of Haematology, Irrua Specialist Teaching Hospital, Irrua, Edo State, Nigeria*
[4]*National Laboratory Manager, Lancet Laboratories, Lagos State, Nigeria*
[5]*Federal Neuropsychiatric Hospital Benin, Edo State, Nigeria*

[*]**Corresponding author:** Iyevhobu KO, Department of Public Health, National Open University of Nigeria, Uromi Community Study Centre, Uromi, Edo State, Nigeria; E-mail: kennylamai@yahoo.com

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Abstract

Hepatitis is a condition that affects the liver and is typically brought on by viral infections, though it can also be brought on by toxic substances. The three most common viral diseases that cause hepatitis—a systemic condition that mostly affects the liver—are hepatitis A, B, and C. In developing countries, hepatitis is a common infection. It is frequently called infectious hepatitis. Vitamins and trace minerals are examples of micronutrients that must be ingested in sufficient quantities to support life and normal physiological function. Worldwide, about 2 billion people experience deficiencies, which are primarily driven on by hunger or a subpar diet. A strong immune response against viral infections needs a variety of micronutrients, yet viruses like the hepatitis B and C viruses also need them to spread infections. This study was designed as a prospective and cross-sectional study to evaluate the micronutrients (Iron, Copper, Zinc, and Magnesium) of hepatitis (HBV and HCV positive) patients in Benin City, Edo State, Nigeria. Comparisons were made between the outcomes of the biochemical markers assessed in hepatitis patients and controls. The full medical history of every subject, including their age, gender, and other significant medical information, was also obtained from their medical records. Between April 2021 and June 2021, a three-month period, this investigation was carried out. In order to ensure the veracity of the hepatitis status results, the researcher further retested both confirmed and negative cases using Hepatitis B and Hepatitis C test strips in accordance with standard laboratory procedures. A total of 180 samples was collected from sero-positive Hepatitis B and C subjects. The mean ± SD of Fe for the control group was 308.79 ± 56.75 while the test was 508.90 ± 66.65. The mean ± SD of Mg for the control group was 1.97 ± 0.25 while the test was 0.69 ± 0.28. The mean ± SD of Zn for the control group was 0.86 ± 0.13 while the test was 0.80 ± 0.16. The mean ± SD of Cu for the control group was 128.85 ± 31.31 while the test was 118.05 ± 20.36. There however for Fe and Mg there was a statistical significance $P<0.05$ when compared to the test and control group while for Zn and Cu there was no statistical significance $P>0.05$ when compared to the test and control group. The mean ± SD of Fe for the control group was 308.79 ± 56.75 while the HBV was 520.95 ± 86.62. The mean ± SD of Mg for the control group was 1.97 ± 0.25 while the HBV was 0.59 ± 0.24. The mean ± SD of Zn for the control group was 0.86 ± 0.13 while the HBV was 0.74 ± 0.18. The mean ± SD of Cu for the control group was 128.85 ± 31.31 while the HBV was 119.92 ± 23.35. There however for Fe and Mg there was a statistical significance $P<0.05$ when compared to the HBV and control group while for Zn and Cu there was no statistical significance $P>0.05$ when compared to the HBV and control group. The mean ± SD of Fe for the control group was 308.79 ± 56.75 while the HCV was 496.84 ± 35.69. The mean ± SD of Mg for the control group was 1.97 ± 0.25 while the HCV was 0.78 ± 0.28. The mean ± SD of Zn for the control group was 0.86 ± 0.13 while the HCV was 0.86 ± 0.12. The mean ± SD of Cu for the control group was 128.85 ± 31.31

while the HCV was 116.18 ± 17.16. There however for Fe and Mg there was a statistical significance P<0.05 when compared to the HCV and control group while for Zn and Cu there was no statistical significance P>0.05 when compared to the HCV and control group.

Keywords: *Hepatitis; HCV; HBsAg; HBV; Micronutrients*

Introduction

Micronutrients are vitamins and trace minerals that we acquire from food and are necessary for maintaining life and proper physiological function [1,2]. Over 2 billion individuals are affected by deficiencies, which are primarily linked to malnutrition or a poor diet [3]. Numerous micronutrients are required to trigger an efficient immune response to viral infections, but viruses like the hepatitis C virus (HCV) and hepatitis B virus (HBV) also utilise these minerals to spread infections [4]. Essential micronutrients are engaged in a variety of metabolic processes in the liver, including enzymatic activities and protein synthesis, oxidative stress and anti-oxidant defense, immunological competence, interferon therapy response controls, and modifications of viral genomes [5,6]. In addition, a number of hepatic illnesses have been linked to reactive oxygen species (ROS) that exacerbate liver diseases [7,8]. Hepatocellular carcinoma (HCC) is created as a result of the immune system's response to viral hepatitis and its accompanying oxidant generation [9]. As a result, the development of viral hepatitis is influenced by changes in micronutrients and their destructive effects against oxidative stress. Hepatitis is an illness that affects the liver and is mostly brought on by viral infections, however toxic chemicals can also cause it. Hepatitis A, B, and C are the three most frequent viral hepatitis causes. This systemic condition primarily affects the liver [10]. Hepatitis is a prevalent infection in poor nations. It is often referred to as infectious hepatitis. Food or water contamination may produce an outbreak that spreads from person to person and results in sporadic causes of the hepatitis A virus infection (HAV) [11]. Viral hepatitis is brought on by five distinct virus types. Hepatitis A, B, C, or E viruses are the most frequent culprits among these [12,13]. It is a serious global health issue that is particularly prevalent in South-East Asia and sub-Saharan Africa. It is a leading source of morbidity and mortality in these regions [14]. More than a million people die with hepatitis each year, the majority of them pass away indirectly through liver cancer or scarring [15]. When hepatitis clears up in less than six months, it is considered acute; when it does not, it is considered chronic [16]. The family Hepadnaviridae includes the double-stranded DNA virus known as the hepatitis B virus (HBV). The infection brought on by viruses, drugs, or toxic substances and defined by the presence of the hepatitis B surface antigen [17]. HBV has an incubation period of typically four months and is spread through permucosal or percutaneous exposure to infected body fluids or blood products. It is known that transmission can happen sexually, parenterally, through blood transfusions and intravenous drug misuse, horizontally between children in a family, and vertically from an infected mother to kid [18]. The family Flaviviridae includes the enveloped, single-stranded RNA virus known as the hepatitis C virus (HCV). Percutaneous or permucosal exposure to infectious blood or blood products is how HCV is transmitted. Those who frequently receive blood transfusions (such as thalassemics), engage in risky sexual behaviour, are employed in healthcare, and are transplant recipients are among the high risk groups for HCV infection. According to the above, when the hepatitis virus attacks the liver severely, acute to chronic hepatitis results. So, after the attack, there can be a change in the serum proteins. In vertebrates and several other animals, the liver is an essential organ. It contributes to the creation of metabolites required for digestion, protein synthesis, and detoxification [19]. Nearly every organ in the body depends on the liver, which is essential for living. Liver function tests (LFTs) are used to diagnose various disorders because of the liver's strategic location and multifaceted functions [20]. However, when one or more of its activities are weakened, such as by hepatitis, there is a chance that liver disease will develop. Furthermore, because the liver is crucial for protein synthesis, inflammation of the liver may potentially have an impact on serum proteins and electrophoretic patterns. A systemic illness mostly affecting the liver, viral hepatitis [21]. Important forms of both acute and chronic viral hepatitis include hepatitis B and C. According to estimates, approximately 2 billion people worldwide exhibit signs of past HBV and HCV infection; more than 350 million are chronic carriers; and hepatitis-related disorders are thought to be responsible for over one million annual deaths. Around 8% of the world's population has HBV infection, and about 5-6% of people are permanent carriers of the disease [22,23]. Therefore, the presence of a stable virus-host interaction and long-lived, non-dividing host cells almost guarantees the longevity of an infection in the absence of a strong immune response [24,25]. A significant human pathogen that causes cirrhosis, hepatocellular cancer, and acute and chronic hepatitis is the hepatitis C virus [26]. Acute hepatitis and chronic liver disease are both primarily brought on by HCV infection [27,28]. Around 686,000 HBV-infected people died worldwide in 2013 as a result of cirrhosis (317,000 deaths), liver cancer linked to hepatitis B (300,000 deaths), and acute infection (69,000 deaths) [29,30]. Hepatitis C-related fatalities

were estimated by the Global Burden of Disease research to have been 333,000 in 1990, 499,000 in 2010, and 704,000 in 2013. There are around 170 million people worldwide who have the hepatitis C virus (HCV), and there are up to 365 million people who have the hepatitis B virus (HBV) [31]. One of the main causes of liver illness in Taiwan is hepatic viral infection, with seroprevalence rates of HBV and HCV estimated to be 17.3% and 4.4%, respectively [32]. Both HBV and HCV infection have numerous extrahepatic symptoms, including hematologic, autoimmune, and dermatologic problems, in addition to liver ailments [33]. The global public health is seriously threatened by chronic viral hepatitis. The two main viruses that cause chronic hepatitis are hepatitis B virus (HBV) and hepatitis C virus (HCV), both of which are known to contribute to the development of cirrhosis and hepatocellular carcinoma (HCC) [34]. Around 257 million people worldwide had chronic HBV infection in 2015, according to estimates of the prevalence of HBV infection in the general population [35]. Around 160 million people are thought to be chronically infected with HCV, with a 2.4% incidence worldwide [36]. Understanding the serum biochemical changes will help researchers better understand how to correlate these parameters with hepatitis progression and further develop a new strategy for control and management of hepatitis in light of various reports on the increased mortality and morbidity rate of patients with hepatitis. As serum proteins and electrophoretic patterns are crucial in the diagnosis of clinical illnesses such acute and chronic inflammation, monoclonal gammopathies, nephropathy, and liver diseases, this investigation will also provide information on any notable changes in the participants with hepatitis. Few investigations, however, have examined hepatic viral infections in non-cirrhotic patients who also had osteoporosis and bone loss. We are conducting this study to assess the micronutrients (Iron, Copper, Zinc, and Magnesium) of people with hepatitis (HBV and HCV positive) in Benin City, Edo State.

Materials and Methods

Area of Study

In the Edo State city of Benin, this study was conducted. Benin City, the capital of southern Nigeria's Edo State, is estimated to have 1,147,188 residents. Located around 25 miles north of the Benin River, it is a city. It is located 200 kilometres east of Lagos on the highway. Nigeria's rubber business is centred in Benin, although processing palm nuts for oil is a significant traditional sector as well. At 6.34° North latitude, 5.63° East longitude, and 80 meters above sea level, Benin City is located [37].

Population of the Study

Population of study was determined using the formula

N= Z2pq/d2 [38]

Where N= the desired sample size (when population is greater than 10,000)

Z= is a constant given as 1.96 (or more simply at 2.0) which corresponds to the 95% confidence level.

P= Prevalence of 13.6% [39].

q= 1.0-p

d= Acceptable error (5%).

Where N= sample size, Z=1.96, p=13.6% (0.136) and d=5% (0.05)

N= 1.962 x 0.136 x 0.864/0.052

N=180.56 ≈ 181 subjects.

A minimum of One Hundred and Eighty (180) samples were obtained and used in this investigation to account for sampling error or dropouts.

One hundred and twenty (120) patients with confirmed hepatitis and sixty (60) subjects who appeared to be in good health (controls) made up the 180 participants in the study (test samples).

Research Design

In order to assess the micronutrients (Iron, Copper, Zinc, and Magnesium) of hepatitis (HBV and HCV positive) individuals in Benin City, Edo State, Nigeria, this study was organized as a prospective and cross-sectional study. Results of the biochemical parameters measured in hepatitis sufferers were compared to those in controls. Additionally, each subject's complete medical history (including age, gender, and other crucial medical data) was gathered from the patient's medical records. This study was conducted over a three-month period, from April 2021 to June 2021. Additionally, the researcher retested both confirmed and negative cases using Hepatitis B and Hepatitis C test strips in accordance with accepted laboratory practices to establish the validity of the hepatitis status results. Hepatitis and control participants are chosen and grouped for the study based on this validity. Using the proper statistical techniques, the study's overall findings were compared to the control.

Ethical Considerations

The Edo State Ministry of Health, located in Benin City, Edo State, granted ethical approval for this study. Prior to collecting samples for this investigation, patients' informed consent was also requested and acquired. The patients were fully informed of the

study's objectives and given assurances regarding the privacy of the data collected from them.

Sample Collection

Each patient had five millilitres (5ml) of venous blood drawn from the ante-cubital vein using a sterile, disposable syringe. The serum proteins, total cholesterol, and serum protein electrophoresis were promptly estimated using the blood samples that were immediately placed in plain containers. The blood was spun at 5000 rpm for 10 minutes. Using a dry, clean Pasteur pipette, the serum was separated from the red blood cells and placed into dry, clean, plain specimen containers. After that, the serum was kept at -20°C until the samples' analyses.

Sample Analysis

Qualitative Detection of Hepatitis B Surface Antigen

The method developed by Deguchi, Yamashita, and Kagita was used to determine the qualitative detection of Hepatitis B Surface Antigen [40].

Table 1: *Mean and SD of Fe, Mg, Zn and Cu of normal and Hepatitis infected subjects.*

Parameters	Control	Test	t-value	p-value
Fe	308.79 ± 56.75	508.90 ± 66.65	-13.287	0.000*
Mg	1.97 ± 0.25	0.69 ± 0.28	19.411	0.000*
Zn	0.86 ± 0.13	0.80 ± 0.16	1.775	0.081
Cu	128.85 ± 31.31	118.05 ± 20.36	33.035	0.134
*: Significant at P<0.05				

Table 2: *Mean and SD of Fe, Mg, Zn and Cu of normal and Hepatitis B infected subjects.*

Parameters	Control	HBV	t-value	p-value
Fe	308.79 ± 56.75	520.95 ± 86.62	-10.037	0.000*
Mg	1.97 ± 0.25	0.59 ± 0.24	19.074	0.000*
Zn	0.86 ± 0.13	0.74 ± 0.18	2.827	0.007*
Cu	128.85 ± 31.31	119.92 ± 23.35	1.120	0.269
*: Significant at P<0.05				

Table 3: *Mean and SD of Fe, Mg, Zn and Cu of normal and Hepatitis C infected subjects.*

Parameters	Control	HCV	t-value	p-value
Fe	308.79 ± 56.75	496.84 ± 35.69	-13.740	0.000*
Mg	1.97 ± 0.25	0.78 ± 0.28	15.188	0.000*
Zn	0.86 ± 0.13	0.86 ± 0.12	0.000	1.000
Cu	128.85 ± 31.31	116.18 ± 17.16	1.738	0.091
*: Significant at P<0.05				

Table 4: *ANOVA analysis of Fe, Mg, Zn and Cu of normal and Hepatitis C infected subjects.*

Parameters	Control	HBV	HCV	t-value	p-value
Fe	308.79 ± 56.75	520.95 ± 86.62	496.84 ± 35.69	80.970	0.000*
Mg	1.97 ± 0.25	0.59 ± 0.24	0.78 ± 0.28	194.746	0.000*
Zn	0.86 ± 0.13	0.74 ± 0.18	0.86 ± 0.12	6.003	0.004*
Cu	128.85 ± 31.31	119.92 ± 23.35	116.18 ± 17.16	1.676	0.195
*: Significant at P<0.05					

Procedure

Before opening the pouch, it was brought to room temperature. The test Strip was taken out of the sealed pouch as soon as feasible and used. Each specimen's test strip as well as the control strip were identified. Until the absorbance occurred without going above the maximum line (MAX) on the test strip, hold the test strip vertically in the sample with the arrows pointing toward the specimen. On a flat, non-absorbent surface, test strips were laid out. Until the red line appeared, the timer was begun (s). At 15 minutes, the verdict was announced. Before reading the results and after 20 minutes, it was made sure that the background was clear. The emergence of two coloured bands—the control band and the test band—confirmed positive results, while the appearance of one purplish red band in the control zone indicated a negative result. When neither the control nor the test bands appeared, the test was deemed invalid**.**

Qualitative Detection of Hepatitis C Antibodies

Using Wilber's approach, the quality of the Hepatitis C antibody detection was determined [41].

Procedure

Before opening the pouch, it was brought to room temperature. The test Strip was taken out of the sealed pouch as soon as feasible and used. Each specimen's and the control's test strip were recognized. Until the absorbance occurred without going above the maximum line (MAX) on the test strip, hold the test strip vertically in the sample with the arrows pointing toward the specimen. On a flat, non-absorbent surface, test strips were laid out. Until the red line appeared, the timer was begun (s). At 15 minutes, the verdict was announced. Prior to reading the results, the context was made clear, and the results were not to be analysed for 20 minutes. The emergence of two coloured bands—the control band and the test band—confirmed positive results, while the appearance of one purplish red band in the control zone indicated a negative result. When neither the control nor the test bands appeared, the test was deemed invalid.

Serum Iron (µg/dL)

According to Arinola & Akiibinu's instructions, the amounts of serum ferritin were measured using an atomic absorption spectrophotometer (AAS) [42].

Procedure

The Analyst 400 atomic absorption spectrophotometer was used to measure the serum trace elements via atomic absorption (AAS). The gas (Oxyacetylene) and air pump were turned on at the same time. Software operations were used to program the instrument with the parameters and requirements for each of the trace elements to be studied. The energy lamp was adjusted to 65, the wavelength to 248, 33 nm, the slit to 2.7/1.8, and the current to 15 mA for the study of iron/ferrtin.

Estimation of Zinc

According to Kaneko's direct approach, the amounts of zinc must be measured using an atomic absorption spectrophotometer (AAS) [43].

Preparation of Sample

After the samples were thawed, 0.1N HCl was added to them in a 1:4 dilution to completely breakdown the proteins that the trace elements were bound to. After that, it was aspirated into the AAS for examination.

Analytical method

The Analyst 400 atomic absorption spectrophotometer was used to test the serum's trace components (AAS). Oxyacetylene gas and the air pump were turned on. Using software manipulations, the instrument's parameters and requirements for each of the trace elements to be studied were configured.

The lamp's energy was set to 30, the wavelength to 213.8 nm, the number of slits to 4, and the current to 15 mA for the examination of zinc. The instrument was then blanked with distilled water before the flame was kindled. The instrument's probe was then put into each sample (diluted 1:4). The computer presented the samples and the readings. The outcomes were shown in mg/l. The value was multiplied by the dilution factor and then by 100 to convert to g/dl. The measurement is expressed in g/dl.

Calculation

$$\text{Zinc (µg/dL)} = \frac{\text{Test absorbance x Concentration of Standard}}{\text{Standard absorbance}}$$

Reference range

Serum Zinc: 70 – 150 µg/dL [44]

Serum Copper (µg/dL)

The Diethyldithiocarbamate technique of Eden & Green, 1940; Ventura & King, 1951 was used to assess the serum copper level.

Procedure

i. 1 mL of 100 mmol/L HCl was added to 3 ml of serum and heated until the solution turned cloudy.
ii. Allow to cool for 10 minutes before adding 1.5 mL of 6 mmol/L HCl.
iii. A little over 3 mL of 200 g/L TCA was added, and the mixture was centrifuged to separate the supernatant after standing for a little while.
iv. After washing the precipitate with about 3 mL of 50 g/L TCA, the supernatant liquids were mixed.
v. Then, 1 mL of sodium diethyldithiocarbamate, 2 mL of ammonia, and 1 mL of sodium pyrophosphate were added.
vi. To extract copper, shake the combination of 5 mL amyl alcohol and ether for 2 minutes.
vii. The organic layer was removed, and anhydrous sodium sulphate was used to dry the surface.
viii. A violet filter was used to read the absorbance (440 nm).

Serum Magnesium (µg/dL)

In Practical Clinical Biochemistry, the diethyldithiocarbamate method of Eden and Green (1940) and Ventura and King (1951) was used to assess the magnesium levels in serum [45].

Procedure

i. 1 mL of 100 mmol/L HCl was added to 3 ml of serum and heated until the solution turned cloudy.

ii. Allow to cool for 10 minutes before adding 1.5 mL of 6 mmol/L HCl.
iii. A little over 3 mL of 200 g/L TCA was added, and the mixture was centrifuged to separate the supernatant after standing for a little while.
iv. After washing the precipitate with about 3 mL of 50 g/L TCA, the supernatant liquids were mixed.
v. Then, 1 mL of sodium diethyldithiocarbamate, 2 mL of ammonia, and 1 mL of sodium pyrophosphate were added.
vi. To extract copper, shake the combination of 5 mL amyl alcohol and ether for 2 minutes.
vii. The organic layer was eliminated, and anhydrous sodium sulphate was used to dry the surface.
viii. A violet filter was used to read the absorbance (440 nm).

Statistical Analysis

The collected results' mean and standard deviation were computed. The study was conducted using SPSS program version 21 and ANOVA (LSD). In this investigation, values with p 0.05 were deemed statistically significant.

Results

Shows the Mean and SD of Fe, Mg, Zn and Cu of normal and Hepatitis infected subjects

From individuals with seropositive hepatitis B and c, 180 samples were taken. The test group had a mean and SD of 508.90 66.65 while the control group's was 308.79 56.75. The test had a Mg mean and SD of 0.69 while the control group's was 1.97 and 0.25. Zn's mean SD for the control group was 0.86 0.13 whereas it was 0.80 0.16 for the test group. The control group's mean and standard deviation for Cu was 128.85 31.31, whereas the test group's was 118.05 20.36. In contrast, there was no statistical significance P>0.05 for Zn and Cu when compared to the test and control group, but there was for Fe and Mg when compared to the test and control group (Table 1).

Shows Mean and SD of Fe, Mg, Zn and Cu of normal and Hepatitis B infected subjects

The HBV was 520.95 86.62 while the mean SD of Fe for the control group was 308.79 56.75. While the HBV was 0.59 0.24, the mean SD of Mg for the control group was 1.97 0.25. While HBV was 0.74 0.18, the mean SD of Zn for the control group was 0.86 0.13. In the control group, the mean SD of Cu was 128.85 31.31 while in the HBV group, it was 119.92 23.35. However, there was no statistical significance P>0.05 for Zn and Cu when compared to the HBV and control group, whereas there was for Fe and Mg when compared to the HBV and control group (Table 2).

Shows Mean and SD of Fe, Mg, Zn and Cu of normal and Hepatitis C infected subjects

The HCV group's mean SD of Fe was 496.84 35.69, whereas the control group's was 308.79 56.75. For the control group, the mean SD of Mg was 1.97 0.25, while for the HCV, it was 0.78 0.28. Zn's mean SD for the control group was 0.86 0.13, while it was 0.86 0.12 for the HCV group. Cu mean SD for the control group was 128.85 31.31 while it was 116.18 17.16 for the HCV group. However, there was no statistical significance P>0.05 for Zn and Cu when compared to the HCV and control group, whereas there was for Fe and Mg when compared to the HCV and control group (Table 3).

ANOVA analysis of Fe, Mg, Zn and Cu of normal and Hepatitis C infected subjects

The HBV was 520.95 86.62, the HCV was 496.84 35.69, and the mean SD of Fe for the control group was 308.79 56.75. For the control group, the mean SD of Mg was 1.97 0.25, HBV was 0.59 0.24, and HCV was 0.78 0.28. The control group's mean and standard deviation for Zn was 0.86 0.13, for HBV it was 0.74 0.18, and for HCV it was 0.86 0.12. The mean and standard deviation (SD) of Cu for the control group were 128.85 31.31, 119.92 23.35 for HBV, and 116.18 17.16 for HCV. When Zn and Cu were compared throughout the table, there was no statistical significance P>0.05, but when Fe and Mg were, there was a statistical significance P0.05 (Table 4).

Discussion

The findings of this study indicate that the HBV was 520.95 86.62, the HCV was 496.84 35.69, and the mean SD of Fe for the control group was 308.79 56.75. For the control group, the mean SD of Mg was 1.97 0.25, HBV was 0.59 0.24, and HCV was 0.78 0.28. The control group's mean and standard deviation for Zn was 0.86 0.13, for HBV it was 0.74 0.18, and for HCV it was 0.86 0.12. The mean and standard deviation (SD) of Cu for the control group were 128.85 31.31, 119.92 23.35 for HBV, and 116.18 17.16 for HCV. However, when compared throughout the table, there was no statistical significance P>0.05 for Cu, although there was for Fe, Mg, and Zn. Additionally, it was discovered that Fe, Mg, and Zn had significant (P<0.05) increases, whereas Mg and Zn had decreases. This result supported the findings of which found that Zn is involved in the activation of PPAR-, a regulator of lipid homeostasis. Zn might take involvement in PPAR-'s ability to bind DNA. Therefore, Zn shortage may lead to a decrease in PPAR- activity, which in turn may promote lipid peroxidation and, ultimately, worsen hepatic steatosis [46]. According to the criteria for hepatic steatosis for patients with HCV-related CLD, observed that the serum Zn levels of patients steadily declined as their hepatic steatosis progressed from a mild status to a severe status [47]. The outcome for Fe was consistent with the findings of who reported that when transgenic mice

expressing the HCV polyprotein were fed an excessive Fe diet, the unfold protein response was activated, leading to the development of hepatic steatosis [48]. Therefore, his research proved that in HCV-related individuals, blood ferritin levels increased in proportion to the severity of hepatic steatosis. NAFLD sufferers frequently had lower serum Cu levels [49]. As no discernible alterations were found, this was also in contrast to our study. Cu availability is probably going to lead to fatter build-up in the liver. Therefore, decreased Cu bioavailability may impact lipid metabolism and contribute to the emergence of NAFLD.

Conclusion

In conclusion, with the exception of Cu, HBV and HCV considerably altered Fe, Zn, and Mg. However, there is still much to learn about this phenomenon. More research on the impact of HBV and HCV on micronutrients is needed because it will enlighten the medical community. Additionally, those who have HBV or HCV should always follow a healthy diet.

Conflict of Interest

The authors say they have no competing interests. The paper's writing and content are solely the authors' responsibility.

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References

1. Bailey RL, West KP, Black RE. The epidemiology of global micronutrient deficiencies. Annals of Nutritional Metabolism. 2015; 66: 22-33.
2. McMillan DC, Maguire D, Talwar D. Relationship between nutritional status and the systemic inflammatory response: Micronutrients. Proceedings Nutritional Society. 2019; 78: 56-67.
3. Howson CP, Kennedy ET, Horwitz A. Prevention of Micronutrient Deficiencies: Tools for Policymakers and Public Health Workers; Committee on Micronutrient Deficiencies, Board on International Health, Food and Nutrition Board: Washington, DC, USA. 1998.
4. Rashed MN. The role of trace elements on hepatitis virus infections: A review. J of Trace Elements Med Bio. 2011; 25: 181-187.
5. Bhaskaram P. Micronutrient malnutrition, infection, and immunity: an overview. Nutritiont Rev. 2002; 60: S40-S45.
6. Ozcelik D, Ozaras R, Gurel Z, Uzun H, Aydin S. Copper mediated oxidative stress in rat liver. Bio Trace Elements Res. 2003; 96: 209-215.
7. Toubi E, Kessel A, Goldstein L, Slobodin G, Sabo E, Shmuel Z, et al. Enhanced peripheral T-cell apoptosis in chronic hepatitis C virus infection: association with liver disease severity. J Hepatology. 2001 35, 774-780.
8. Loguercio C, Federico A. Oxidative stress in viral and alcoholic hepatitis. Free Radical in Bio Med.2003; 34:1-10.
9. Jain SK, Pemberton PW, Smith A, McMahon RF, Burrows PC, Aboutwerat A, et al. Oxidative stress in chronic hepatitis C: not just a feature of late stage disease. J Hepatology. 2012; 36: 805-811
10. Bain V, Ma G. Acute viral hepatitis. In: First principle of Gastroenterology. McGraw Hill, New York .2004; 479-491.
11. Wang JT, Lee CZ, Chen PJ, Wang TH, Chen DS. Transfusion transmitted HBV infection in an endemic area: the necessity of more sensitive screening for HBV carriers. Transfusion. 2012; 42: 1592-1599.
12. World Health Organization."Hepatitis". World Health Organization. Archived from the original. 2013.
13. Salahuddin M, Syed SA, Manzoor H, Mahenaz A, Farhana T. Recent Spectrum of Acute Viral Hepatitis in Children: An Experience in a Tertiary Centre of Bangladesh. Adv Res Gastroentero Hepatol. 2017; 6: 555686.
14. Fattovich G. Natural history of hepatitis B. British J Hepatology. 2013; 39: 50-58.
15. Wang Z, Chen Y, Xie S, Lv H. Changing Epidemiological Characteristics of Hepatitis A in Zhejiang Province, China: Increased Susceptibility in Adults. 2016; 11: e0153804.
16. Bernal W, Wendon J. Acute Liver Failure". New England J Med. 2013; 369: 2525-2534.
17. Aziz MA, Seema B, Ahmed S, Bikha R, Devrajani SA, Memon G, et al. Metabolic Investigation in Hepatitis B Patients with or Without Diabetes mellitus to determine the Impact of Anti-Oxidant Activity. World Applied Sci J. 2017; 15: 765-771.
18. Satsangi S, Dhiman RK. Combating the wrath of viral hepatitis in India. Indian J Med Res. 2016; 144:1-5.
19. Fernandez-Checa JC, Kaplowitz N. Hepatic mitochondrial glutathione: Transport and role in disease and toxicity. Toxicology Applied Pharmacology. 2015; 204: 263-273.
20. Nsirim, N. Liver structure, function and physiology, chemical biochemistry for student of pathology. Longman press. 2016; 57-67.

21. Duc DD. The epidemiology of hepatitis virus in Vietnam. J Practice Med. 2017; 339:1-3.
22. Porth CM. Pathophysiology. Concepts of altered health states. 6th edition, Lippincott Williams & Wilkins. Philadelphia. 2012; 643.
23. Inlin H, Zhihua L, Fan G. Epidemiology and Prevention of Hepatitis B Virus Infection. Int J Med Sci. 2013; 1: 50.
24. Shiina S, Fujino H, Kawabe T, Tagawa K, Unuma T, Yoneyama M, et al. Relationship of HBsAg, "subtypes with HBeAg'anti-HBe status and chronic liver disease" II. Evaluation of epidemiological factors and suspected risk factors of liver dysfunction. American J Gastroenterology. 2011; 86: 872.
25. Mukaddes E. Oxidative, Stress and Benefits of Antioxidant Agents in Acute and Chronic Hepatitis. Hepatitis Monthly J. 2012; 12: 160.
26. Spada E, Abbate I, Sicurezza E, Mariano A, Parla V, Rinnone S, et al. Molecular epidemiology of a hepatitis C virus outbreak in a hemodialysis unit in Italy. J Med Vir. 2008; 80: 216-217.
27. Imran M, Waheed Y, Manzoor S, Bilal M, Ashraf W, Ali M, et al. Interaction of Hepatitis C virus proteins with pattern recognition receptors. Vir J. 2012; 9: 126.
28. Sarker NR, Saha SK, Ghosh DK, Adhikary A, Mridha A. Seropositivity of viral markers in icteric children. Bang Med J. 2014; 43: 26-29.
29. Jobayer M, Chowdhury SS, Shamsuzzaman SM, Islam MS. Prevalence of hepatitis Bvirus, hepatitis C virus and HIV in overseas job seekers of Bangladesh with the possible routes of transmission. Mymensingh Med J. 2016; 25: 530-535.
30. Alam S, Azam G, Mustafa G, Alam M, Ahmed N. Past, present and future of hepatitis B and fatty liver in Bangladesh. Gastroenterology Hepatology Open access. 2017; 6: 1-7.
31. Jha V, Prasad N. CKD and Infectious Diseases in Asia Pacific: Challenges and Opportunities. American Journal of Kidney Diseases: the Official J National Kidney Foundation. 2016; 68: 148–160.
32. Chen CH. Estimation of seroprevalence of hepatitis B virus and hepatitis C virus in Taiwan from a large-scale survey of free hepatitis screening participants. J Formos Med Association. 2007; 106: 148-155.
33. Pyrsopoulos NT, Reddy KR. Extrahepatic manifestations of chronic viral hepatitis. Current Gastroenterology Reports. 2011; 3: 71-78.
34. Perz JF, Armstrong GL, Farrington LA, Hutin YJ, Bell BP. The contributions of hepatitis b virus and hepatitis c virus infections to cirrhosis and primary liver cancer worldwide. J Hepatology. 2006 45: 529-538.
35. World Health Organization, Global Hepatitis Report, Geneva. 2017.
36. Lavanchy D. Evolving epidemiology of hepatitis c virus. Cli Microbiology Inf. 2011; 17: 107-115.
37. World Gazetteer (2007): Population of Cities, news, divisions.
38. Araoye MO. Sample Size Determination in Research Methodology with Statistics for Health and Social Sciences. Nathadex Publishers, Ilorin. 2004; 115-121.
39. Musa BM, Bussell S, Borodo MM, Samaila AA, Femi OL. Prevalence of hepatitis B virus infection in Nigeria. 2000-2013: A systematic review and meta-analysis. Niger J Clin Pract. 2015; 18: 163-172.
40. Deguchi M, Yamashita N, Kagita M. Quantitation of hepatitis B surface antigen by an automated Chemiluminscent microparticle immunoassay. J Vir Methods. 2004; 115: 217.
41. Wilber JC. Development and use of laboratory tests for hepatitis C infection: A review. J Cli Immunoassay. 1993; 16: 204.
42. Arinola OG, Akiibinu MO. The levels of antioxidants and some trace metals in Nigerians that are occupationally exposed to chemicals. Indian J Occup Environ Med. 2006; 10: 65-68.
43. Kaneko JJ. Clinical Biochemistry of Animal. 4th Edn. Academic Press Inc. 1999; 932.
44. Pennell C, Painter J, Cope Y, Jane LS. Appendix. Tietz Textbook of Clinical Chemistry, 2nd edition, edited by Carl A. Burtis and Edward R. Ashwood. 1994; 2183.
45. Practical Clinical Biochemistry. Iron, Copper and Zinc. 5th edition Volume I, edited by Harold Varley, Alan H. Gowenlock and Maurice Bell. William Heinemann Medical Books Ltd. 1980; 941-942
46. Mikhail TH, Nicola WG, Ibrahim KH, Salama SH, Emam M. Abnormal zinc and copper metabolism in hepatic steatosis. Boll. Chim. Farm.1996; 135: 591–597.
47. Bao B, Prasad AS, Beck FW, Fitzgerald JT, Snell D, Bao GW, et al. Zinc decreases C-reactive protein, lipid peroxidation, and inflammatory cytokines in elderly subjects: a potential implication of zinc as an atheroprotective agent. Am J Clin Nutr. 2010; 91: 1634-1641.
48. Nishina S, Korenaga M, Hidaka I, Shinozaki A, Sakai A, Gondo T, et al. Hepatitis C virus protein and iron overload induce hepatic steatosis through the unfolded protein response in mice. Liver Int. 2010; 30: 683-692.
49. Aigner E, Strasser M, Haufe H, Sonnweber T, Hohla F, Stadlmayr A, et al. A role for low hepatic copper concentrations in nonalcoholic Fatty liver disease. Am J Gastroenterol. 2010; 105: 1978–1985.