

Original Research Article

An Investigation of the Frequency of HBsAg, and Anti-HCV Including Their Effects on Some Biochemical Parameters among University Students Presenting with Malaise

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Abstract: Hepatitis B and C viruses cause viral hepatitis which could affect the function of the liver. Viral hepatitis is one of the major killer diseases associated with malaise but could be prevented. This work was designed to investigate the frequency of HBsAg, and anti-HCV including their effects on some biochemical parameters among University Students presenting with malaise. Fifty malaise University students were recruited through their Health Centre. The students were screened for anti-HCV, anti-HIV and HBsAg Immunochemical by ELIZA and Immunoblotting. Total Bile acid and Total Antioxidant were determined by spectrophotometry. Immune status was determined using CD4 count by Cyflowmetry. The results obtained showed an overall frequency of 8(16%) HBsAg seropositive, 5(10%) anti-HCV seropositive, 7(14%) HBsAg and anti-HCV seropositive, 30(60%) HBsAg or and anti-HCV seronegative students. Mono infection of hepatitis B virus and its coinfection with hepatitis C virus were more prevalent in female students than their male counterpart while hepatitis C virus infection was more prevalent in males than the female students. Total bile acid in anti-HCV seropositive students was significantly higher than in HBsAg seropositive students and in HBsAg and anti-HCV seropositive students than those who were anti-HCV seropositive also in HBsAg and anti-HCV seropositive students than HBsAg and anti-HCV seronegative students with $p < 0.05$ Total antioxidant anti-HCV seropositive students was significantly lower than in HBsAg seropositive students; also in HBsAg seropositive patients than in HBsAg and anti-HCV seronegative patients with $p < 0.05$. There was also a significantly lower plasma Total antioxidant in HBsAg and anti-HCV seropositive students than in anti-HCV seropositive students and in students with anti-HCV, HBsAg + anti-HCV seropositive than in students who were sero negative to anti-HCV and HBsAg + anti-HCV with $p < 0.05$. CD4 count was significantly lower in anti-HCV seropositive students than HBsAg seropositive students and than HBsAg + anti-HCV seronegative students; also in HBsAg + anti-HCV seropositive students than HBsAg + anti-HCV seronegative students with $p < 0.05$. Biochemical alteration on antioxidant, total bile acid and CD4 count was more in HCV and students coinfecting with HBV-HCV. The overall frequency of 8(16%) HBsAg seropositive, 5(10%) anti-HCV seropositive, 7(14%) HBsAg and anti-HCV seropositive, 30(60%) HBsAg or and anti-HCV seronegative students. More female students were mono and coinfecting with HBV and HCV. Mono-infection of HCV was more prevalent in male than female students.

Keywords: frequency, HBsAg,, anti-HCV, University Students, malaise, Total Bile Acid, Total Antioxidant, CD4

INTRODUCTION

Hepatitis, a general term referring to inflammation of the liver, may result from various causes, both infectious (i. e, viral, bacterial, fungal, and parasitic organisms) and noninfectious (e. g, alcohol, drugs, autoimmune diseases, Viral hepatitis is most commonly caused by hepatitis A virus (HAV), hepatitis B virus (HBV), and hepatitis C virus (HCV). These 3 viruses can all result in acute disease with symptoms of nausea, abdominal pain, fatigue, malaise, and jaundice. Additionally, HBV and HCV can lead to chronic infection. Patients who are chronically infected may go

on to develop cirrhosis and hepatocellular carcinoma (HCC). Furthermore, chronic hepatitis carriers remain infectious and may transmit the disease for many years. Hepatitis B virus (HBV) is transmitted through exposure to infective blood, semen, and other body fluids. HBV can be transmitted from infected mothers to infants at the time of birth or from family member to infant in early childhood. Transmission may also occur through transfusions of HBV-contaminated blood and blood products, contaminated injections during medical procedures, and through injection drug use [1-3].

HBV also poses a risk to healthcare workers who sustain accidental needle stick injuries while caring for infected-HBV patients. Safe and effective vaccines are available to prevent HBV. Hepatitis C virus (HCV) is mostly transmitted through exposure to infective blood. This may happen through transfusions of HCV-contaminated blood and blood products, contaminated injections during medical procedures, and through injection drug use. Sexual transmission is also possible, but is much less common. There is no vaccine for HCV [1-3].

Antioxidant is a molecule that inhibits the oxidation of other molecules. Oxidation is a chemical reaction that can produce free radicals, leading to chain reactions that may damage cells. Antioxidants such as thiols or ascorbic acid (vitamin C) terminate these chain reactions. The term "antioxidant" is mainly used for two different groups of substances: industrial chemicals which are added to products to prevent oxidation, and natural chemicals found in foods and body tissue which are said to have beneficial health effects [4].

To balance the oxidative state, plants and animals maintain complex systems of overlapping antioxidants, such as glutathione and enzymes (e.g., catalase and superoxide dismutase) produced internally or the dietary antioxidants, vitamin A, vitamin C and vitamin E. The antioxidant defense system has many components. A deficiency in any of these components can cause a reduction in the overall antioxidant status of an individual. Reduction in total antioxidant status has been implicated in several disease states, such as cancer and heart disease [5, 6]. Antioxidants are man-made or natural substances that may prevent or delay some types of cell damage. Antioxidants are found in many foods, including fruits and vegetables. They are also available as dietary supplements. Examples of antioxidants include; Beta-carotene; Lutein; Lycopene, Selenium, Vitamin A, Vitamin C, Vitamin E [7].

Vegetables and fruits are rich sources of antioxidants. There is good evidence that eating a diet with lots of vegetables and fruits is healthy and lowers risks of certain diseases. But it isn't clear whether this is because of the antioxidants, something else in the foods, or other factors. High-dose supplements of antioxidants may be linked to health risks in some cases. For example, high doses of beta-carotene may increase the risk of lung cancer in smokers. High doses of vitamin E may increase risks of prostate cancer and one type of stroke [8].

Bile acids are steroid acids found predominantly in the bile of mammals and other vertebrates. Different molecular forms of bile acids can be synthesized in the liver by different species. Bile acids are conjugated with taurine or glycine in the liver, forming bile salts may also interact with some medicines. Bile Acids (BA) make 67% of the total

composition of Bile. They are 24-carbon steroids generated during cholesterol metabolism. They form conjugates with either glycine or taurine to form bile salts. Five of the BAs account for more than 99% of the total population found in biofluids [9]. The average composition in healthy individuals includes conjugates of cholic, chenodeoxycholic, deoxycholic and lithocholic acids. Bile acids are critical due to their ability to solubilize lipids by forming micelles with cholesterol, and fatty acids. Their synthesis is not only critical for the removal of cholesterol from the body but they are also needed for proper uptake of dietary lipids into the small intestine [10]. The measurement of circulatory Total Bile Acids (TBA) therefore provides information about hepatic functions and liver diseases such as jaundice, and hepatocellular injury. TBA estimation can detect liver damage during early stages and permits patients to get treatment before hepatic damages become irreversible [11].

In molecular biology, CD4 (cluster of differentiation 4) is a glycoprotein found on the surface of immune cells such as T helper cells, monocytes, macrophages, and dendritic cells. CD4+ T helper cells are white blood cells that are an essential part of the human immune system. They are often referred to as CD4 cells, T-helper cells or T4 cells. CD4 is a co-receptor that assists the T cell receptor (TCR) in communicating with an antigen-presenting cell. Using its intracellular domain, CD4 amplifies the signal generated by the TCR by recruiting an enzyme, the tyrosine kinase Lck, which is essential for activating many molecular components of the signaling cascade of an activated T cell. Various types of T helper cells are thereby produced. CD4 also interacts directly with MHC class II molecules on the surface of the antigen-presenting cell using its extracellular domain. The extracellular domain adopts an immunoglobulin-like beta-sandwich with seven strands in 2 beta sheets [12-15].

During antigen presentation, both the TCR complex and CD4 are recruited to bind to different regions of the MHCII molecule ($\alpha 1/\beta 1$ and $\beta 2$, respectively). Close proximity between the TCR complex and CD4 in this situation means the Lck kinase bound to the cytoplasmic tail of CD4 is able to tyrosine-phosphorylate the Immunoreceptor tyrosine activation motifs (ITAM) present on the cytoplasmic domains of CD3. Phosphorylated ITAM motifs on CD3 recruits and activates SH2 domain-containing protein tyrosine kinases (PTK) such as Zap70 to further mediate downstream signal transduction via tyrosine phosphorylation, leading to transcription factor activation including NF- κ B and consequent T cell activation [12-15].

This work was designed to investigate of the frequency of HBsAg, and anti-HCV including their

effects on some biochemical parameters among University Students presenting with malaise.

MATERIALS AND METHODS

Study Area

The study area is Owo-Ondo State in Nigeria. Owo is a city in the Ondo state of Nigeria. Between 1400 and 1600 AD, it was the capital of a Yoruba city-state. The city has a population of 222,262, based on 2006 population census. Owo is situated in south-western Nigeria, at the southern edge of the Yoruba Hills, and at the intersection of roads from Akure, Kabba, Benin City, and Siluko. Owo is situated halfway between the towns of Ile Ife and Benin City. There are two tertiary institutions- A Polytechnic and a University in the town.

Study Population

Fifty student volunteers of Achievers University, Owo –Nigeria aged 18 to 45 years presenting with malaise were recruited through the Health Centre of the University.

Inclusion Criteria

1. Only Students who were not taking alcohol or cigarette were recruited
2. Only Students who were not on any drug medication were recruited
3. Only Students who were not HIV seropositive were recruited for the study
4. Non pregnant female students were included for the study
5. Only Non diabetic students according to their medical history were recruited for the study
6. Only non-asthmatic students were included for the study
7. Students who were unable to eat as a result of loss of appetite throughout the night before presenting at the Health Centre the following morning were recruited.

Exclusion Criteria

1. Students who were taking alcohol or cigarette were not recruited
2. Students who were on any drug medication were not included
3. Students who were HIV seropositive were not recruited for the study
4. Pregnant female students were not included for the study
5. Diabetic students according to their medical history were not recruited for the study
6. Asthmatic students were not recruited

Biological Sample

After an overnight fasting before medication, Five milliliters of venous blood were obtained from each of the students into Lithium heparinized bottles for evaluation of CD4, plasma Total Bile Acid, Total

antioxidant and determination of anti-HCV, HBsAg and anti-HIV.

Duration of Study

Six months between February and July, 2016.

Methods

Total antioxidant assay

Randox total antioxidant status kit enables assessment of the integrated antioxidant system which encompasses all biological components with antioxidant activity.

The Randox Total Antioxidant Status kit can also be used to determine the phenolic content of beverages such as wine, beer and fruit juice. Antioxidants present in red wine, tea and other beverages have been shown to give a cardio-protective effect. The compounds in beverages, which contribute to this effect, are Phenolics, of which Polymeric Phenols are the largest subgroup. Phenolics also contribute towards the taste, color, odor and preservative of the beverage.

Principle

2, 2'-Azino-di-3-ethylbenzthiazoline sulphonate is incubated with a peroxidase (metmyoglobin) and H₂O₂ to produce the radical cation. This has a relatively stable blue-green color, which is measured at 600 nm. Antioxidants in the added sample cause suppression of this color production to a degree which is proportional to their concentration.

Total Bile Acid

Principle

Two reactions are combined in this kinetic enzyme cycling method. In the first reaction bile acids are oxidised by 3- α hydroxysteroid dehydrogenase with the subsequent reduction of Thio-NAD to Thio-NADH. In the second reaction the oxidised bile acids are reduced by the same enzyme with the subsequent oxidation of NADH to NAD. The rate of formation of Thio-NADH is determined by measuring the specific absorbance change at 405nm. (Abbreviations: NADH, NAD, Thio-NADH, Thio-NAD)

Hepatitis B surface antigen (HBsAg) test

Hepatitis B surface antigen (HBsAg) test was carried out on the student volunteers by using a one step enzyme immunoassay technique of the sandwich type for the detection of HBsAg in human serum or plasma using the reagent kit of BIO –RAD Raymond Poincare, Marnes La Coquette.

Principle

MONOLISA AgHBs PLUS is a one enzyme technique of the sandwich type using three monoclonal antibodies selected for their ability to bind themselves to the various subtypes of HBsAg now recognized by World Health Organisation.

The solid phase is made up of 12 strips of 8 polystyrene wells coated with the first monoclonal antibody the two other monoclonal antibodies are bound to the peroxidase.

The assay procedure includes the following reaction step:

1. Distribution of samples into the wells of the microplates. This distribution can be visually controlled; there is a clear difference of colouration between empty well and well with sample. This distribution can also be controlled automatically by reading at 450/620 – 700 nm(optional).
2. Distribution of the conjugate into the wells: This distribution can also be visually controlled; the conjugate which is initially orange becomes red after addition into the well. It is possible to control automatically this distribution by spectrophotometric reading at 450/620–700nm [optional]. The sample deposition can also be controlled at this step of the manipulation by automatic reading at 450/620-700nm.
3. Incubation at 37°C
4. Washing and development of the enzyme activity bound solid phase by the addition of substrate.
5. Stopping development, then reading of the optical densities at 450/620 – 700nm and interpretation of the results.

HIV screening

HIV screening was carried out on the student volunteers after pre-test counseling by using the reagent kit of Abbot Laboratories Co. Ltd, Japan. The Abbot Determine HIV-1/2 is an *in vitro*, visually read qualitative immunoassay for the detection of antibodies to HIV-1 and HIV-2 in human serum plasma or whole blood. The test is intended as an aid to detect antibodies to HIV-1/HIV-2 from infected individuals. Summary and explanation of the test

AIDS [acquired Immunodeficiency Syndrome] is characterized by changes in the population of T-cell lymphocytes. In an infected individual, the virus causes depletion of helper –T cells, which leave the person susceptible to opportunistic infections and some malignancies. The virus that causes AIDS exists as two related types known as HIV-1 and HIV-2. The presence of the AIDS virus elicits the production of specific antibodies to enter to either HIV-1/HIV-2.

Biological Principle of the Procedure

Determine HIV – 1/2 is an Immunochromatographic test for the detection of antibodies to HIV-1/HIV-2. Sample is added to the sample pad. As the sample migrates through the conjugate pad, it reconstitutes and mixes with the selenium colloid – antigen conjugate. This mixture continues to migrate through the solid phase to the

immobilized recombinant antigens and synthetic peptides at the patient window site. If antibodies to HIV-1 and or HIV-2 are present in the sample, the antibodies bind to the antigen – selenium colloid and to the antigen at the patient window forming a red line at the patient window site. If antibodies to HIV-1 and HIV-2 are absent, the antigen – selenium colloid flows past the patient window and no red line is formed at the patient window site. To ensure assay validity a procedural control bar is incorporated in the assay device.

Western blot assay

The HIV confirmatory test was carried out on all the volunteers by Western blot assay, using reagent kit of Immunoetics, Inc., 27 Dryclock Avenue, Boston, USA. <http://www.immunoetics.com>

Principle

The QualicodeHIV1/2 kit is a qualitative immunoblot assay based on the Western Blot principle. The assay is performed on immunoblot membrane containing HIV-1 viral lysate protein (HLTVIII B stain) and a recombinant HIV-2 protein. To produce the membrane HIV-1 viral protein are fractionated according to molecular weight on a Polyacrylamide slab gel (PAGE) in the presence of Sodium dodecyl sulphate (SDS). The separated HIV-1 is then transferred through electrophoretic blotting from the gel to a nitrocellulose membrane two bands are directly striped on the membrane

- 1) A control band containing staphylococci protein A.
- 2) A recombinant HIV-2 specific envelope antigen.

The membrane is then cut into strips for individual sample testing. During the procedure, the strips containing HIV1/2 are reacted with the serum specimen and washed to remove unbound antibodies. Visualization of human globulin specifically bound to HIV-1 or HIV-2 proteins is performed by sequential reaction with goat anti-human immunoglobulin-alkaline phosphatase conjugate and BCIP/NBT substrate. Band positions are compared to those on the Reference Card developed using the HIV -1/2 Positive Control Serum. The intensity of the bands is monitored by comparison to the HIV-1/2 Weakly Reactive Control.

Anti – HCV test

Anti – HCV test on all the volunteers was carried out by a third generation enzyme immunoassay for the determination of antibodies to hepatitis C virus in plasma using the reagent kit of DIA.PRO, Diagnostic Bioprobes Srl, Via Columella, Milano – Italy

Principle

Microplates are coated with HCV specific antigens derived from core and ‘ns’ regions encoding

for conservative and immunodominant antigenic determinants (Core, NS3, NS4 and NS5). The solid phase is first treated with the diluted sample and HCV antibody are captured, if present, by the antigens. After washing out all other components of the sample, in the 2nd incubation bound anti - HCV are detected by the addition of anti - human immunoglobulin G & M antibody ,labeled with peroxidase . The enzyme captured on the solid phase, acting on the substrate / chromogen mixture, generates an optical signal that is proportional to the amount of anti - HCV antibodies present in the sample.

CD4 Count

CD4 Count was Carried out by Cytoflometry Using the Reagent Kit of Partec and Partec CD4 Machine

Data Analysis

The values of the parameters obtained were subjected to statistical analysis using SPSS 18.0 to determine level of significance of differences at 0.05.

Ethical Consideration

The proposal of this work was presented to the Research and Ethical committee of the Department of Medical Laboratory Science, Achievers University, Owo-Nigeria. It was reviewed and approved before the commencement of the work. Only student volunteers were studied.

RESULTS

The results obtained showed an overall frequency of 8 (16%) HBsAg seropositive, 5(10%) anti-HCV seropositive, 7(14%) HBsAg and anti-HCV seropositive, 30(60%) HBsAg or and anti-HCV seronegative students.

According to gender 5 (10%) HBsAg seropositive females, 3 (6%) BsAg seropositive males, 2 (4%) anti-HCV seropositive females, 3 (6%) anti-

HCV seropositive males, 4 (8%) HBsAg and anti-HCV seropositive females, 3 (6%) HBsAg and anti-HCV seropositive males; 16(32%) HBsAg and anti-HCV seronegative females and 14 (28%) HBsAg and anti-HCV seronegative males.

Mono infection of hepatitis B virus and its co-infection with hepatitis C virus were more prevalent in female students than their male count apart while hepatitis C virus infection was more prevalent in males than the female students.

Total bile acid in anti-HCV seropositive students was significantly higher than in HBsAg seropositive students and in HBsAg and anti-HCV seropositive students than those who were anti-HCV seropositive also in HBsAg and anti-HCV seropositive students than HBsAg and anti-HCV seronegative students with p<0.05

Total antioxidant anti-HCV seropositive students was significantly lower than in HBsAg seropositive students ; also in HBsAg seropositive patients than in HBsAg and anti-HCV seronegative patients with p<0.05.

There was also a significantly lower plasma Total antioxidant in HBsAg and anti-HCV seropositive students than in anti-HCV seropositive students and in students with anti-HCV, HBsAg + anti-HCV seropositive than in students who were sero negative to anti-HCV and HBsAg + anti-HCV with p<0.05.

CD4 count was significantly lower in anti-HCV seropositive students than HBsAg seropositive students and than HBsAg + anti-HCV seronegative students; also in HBsAg + anti-HCV seropositive students than HBsAg + anti-HCV seronegative students with p<0,05.

Table1: Frequency of HBsAg, and anti-HCV including Plasma values of Total Bile Acid and Total antioxidant

		HBsAg seropositive patients	anti-HCV seropositive patients	HBsAg and anti-HCV seropositive patients	HBsAg and anti-HCV seronegative patients
Total Bile Acid $\mu\text{mol/L}$		5.0 \pm 1.8	8.0 \pm 1.5	16 \pm 2.0	4.5 \pm 1.5
Total antioxidant μM		394 \pm 8.0	360 \pm 5.0	301 \pm 5.0	570 \pm 5.0
CD4		500 \pm 10.0	461 \pm 11	450 \pm 10.0	580 \pm 12.0
Frequency	Total	8(16%)	5(10%)	7(14%)	30(60%)
	Female	5 (10%)	2(4%)	4(8%)	16(32%)
	Male	3(6%)	3(6%)	3(6%)	14(28%)

Table 2: Comparative Analysis of CD4, Total Bile Acid and Total Antioxidant obtained in the Students

		HBsAg seropositive patients Vs anti-HCV seropositive patients	HBsAg seropositive patients Vs anti-HCV seropositive patients	HBsAg seropositive patients Vs HBsAg and anti-HCV seronegative patients	anti-HCV seropositive patients Vs HBsAg and anti-HCV seropositive patients	anti-HCV seropositive patients Vs HBsAg and anti-HCV seronegative patients	HBsAg and anti-HCV seropositive patients Vs HBsAg and anti-HCV seronegative patients
Total Bile Acid $\mu\text{mol/L}$	't'	-2.8	-4.09	0.21	-3.2	1.65	4.6
	p	0.16	0.03*	0.43	0.04*	0.12	0.02*
Total antioxidant μM	't'	3.60	9.86	-18.66	8.34	-29.70	-38.04
	p	0.03*	0.005*	0.001*	0.007*	0.0005*	0.003*
CD4/ cells/ μl	't'	2.62	3.54	-5.12	0.74	-7.31	-3.82
	p	0.06	0.04*	0.02	0.27	0.009*	0.007*

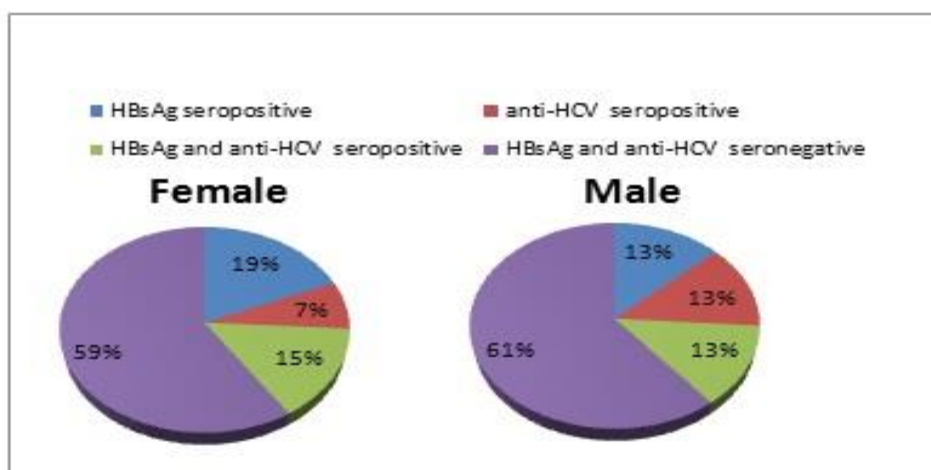


Fig-1: Prevalence of HBsAg seropositive patients, anti-HCV seropositive patients, HBsAg + anti-HCV seropositive patients and HBsAg/anti-HCV seronegative patients

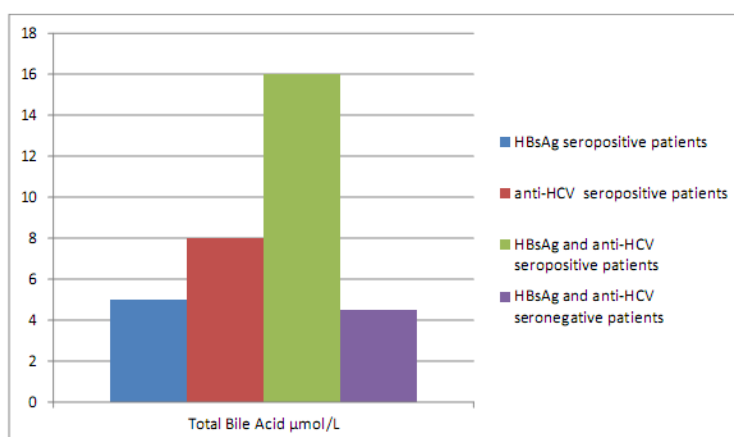


Fig-2: Total bile acid in HBsAg seropositive patients, anti-HCV seropositive patients, HBsAg + anti-HCV seropositive patients and HBsAg/anti-HCV seronegative patients

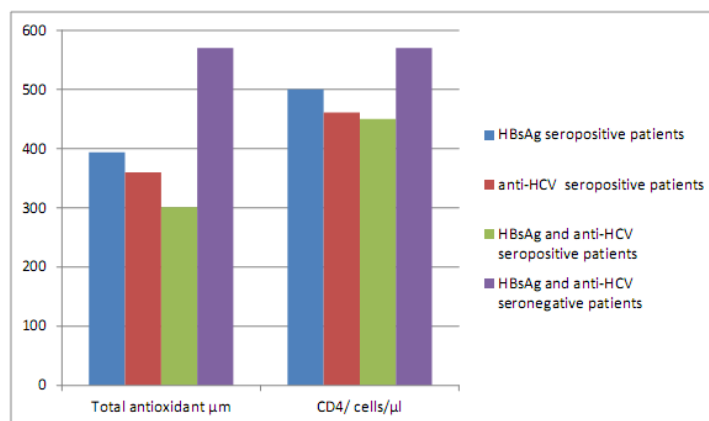


Fig-3: Total Antioxidant and CD4 in HBsAg seropositive patients, anti-HCV seropositive patients, HBsAg + anti-HCV seropositive patients and HBsAg/anti-HCV seronegative patients

DISCUSSION

Total bile acid in anti-HCV seropositive students was significantly higher than in HBsAg seropositive students and in HBsAg and anti-HCV seropositive students than those who were anti-HCV seropositive also in HBsAg and anti-HCV seropositive students than HBsAg and anti-HCV seronegative students.

The measurement of circulatory Total Bile Acids (TBA) therefore provides information about hepatic functions and liver diseases such as jaundice, and hepatocellular injury which could be associated with the findings of this study as the infecting viruses are hepatotropic viruses [11].

Total antioxidant anti-HCV seropositive students was significantly lower than in HBsAg seropositive students; also in HBsAg seropositive patients than in HBsAg and anti-HCV seronegative patients. This could be attributed to the fact that reduction in total antioxidant status has been implicated in several disease states which was found in this study disease. The HCV has also been reported to be more virulent hence the significant alteration in the infected students [5, 6].

Mono infection of hepatitis B virus and its co-infection with hepatitis C virus were more prevalent in female students than their male count apart while hepatitis C virus infection was more prevalent in males than the female students. There was a significantly lower plasma Total antioxidant in HBsAg and anti-HCV seropositive students than in anti-HCV seropositive students and in students with anti-HCV, HBsAg + anti-HCV seropositive than in students who were sero negative to anti-HCV and HBsAg + anti-HCV.

The overall prevalence of anti-HCV among the students in this study was higher than those reported by Abiodun *et al.* [14] that of the 1572 Among Undergraduates in Ogbomosho, Southwestern Nigeria, 6

tested positive, giving an overall prevalence of 0.40%. This could be as a result of the recruitment criteria that exclude those who did not have malaise as malaise has been associated with this condition. This fact also holds for HCV co-infection with HBV. In addition viruses causing hepatitis can all result in acute disease with symptoms of nausea, abdominal pain, fatigue, malaise, and jaundice. Additionally, HBV and HCV can lead to chronic infection [1-3, 15].

The prevalence of HBsAg and anti-HCV in this study was also higher than those reported by Chiekulie *et al.* [16] in a suburban University Teaching Hospital in South-East Nigeria that HBsAg positive participants were 9 (2.2%) while 3 (0.7%) were positive for HCV. No participant had triple infection of HIV/HBV/HCV. They concluded that Seroprevalence of HBV and HCV is low among HIV patients in Orlu. However there is a need for HBV and HCV testing of all HIV positive patients to reduce morbidities and mortalities from liver diseases.

CD4 count was significantly lower in anti-HCV seropositive students than HBsAg seropositive students and than HBsAg + anti-HCV seronegative students; also in HBsAg + anti-HCV seropositive students than HBsAg + anti-HCV seronegative students.

Cluster of differentiation 4 (CD4) is a glycoprotein found on the surface of immune cells such as T helper cells, monocytes, macrophages, and dendritic cells. CD4+ T helper cells are white blood cells that are an essential part of the human immune system. They are often referred to as CD4 cells, T-helper cells or T4 cells. CD4 is a co-receptor that assists the T cell receptor (TCR) in communicating with an antigen-presenting cell. This could be attributed to possible decrease in immune status as a result of HCV and its co-infection with HBV as indicated seropositive reaction to anti-HCV and HBsAg [12-14, 17].

CONCLUSION

Biochemical reduction in total antioxidant, with increased total bile acid and low CD4 count were more in students infected with HCV and students co-infected with HBV-HCV. The overall frequency of 8(16%) HBsAg seropositive, 5 (10%) anti-HCV seropositive, 7(14%) HBsAg and anti-HCV seropositive, 30 (60%) HBsAg or and anti-HCV seronegative students.

REFERENCES

1. Previsani N, Lavanchy D. World Health Organization. Hepatitis B (WHO/CDS/CSR/LYO/2002.2). 2002;. [Full Text].
2. Wasley A, Grytdal S, Gallagher K. Surveillance for acute viral hepatitis--United States, 2006. *MMWR Surveill Summ*. 2008;57(2):1-24..
3. Keeffe EB, Dieterich DT, Han SH, Jacobson IM, Martin P, Schiff ER, Tobias H. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States: 2008 update. *Clinical Gastroenterology and Hepatology*. 2008 Dec 31;6(12):1315-41.
4. Cortés-Jofré M, Rueda JR, Corsini-Muñoz G, Fonseca-Cortés C, Caraballosa M, Bonfill Cosp X. Drugs for preventing lung cancer in healthy people. *The Cochrane Library*. 2012 Oct.
5. Jiang L, Yang KH, Tian JH, Guan QL, Yao N, Cao N, Mi DH, Wu J, Ma B, Yang SH. Efficacy of antioxidant vitamins and selenium supplement in prostate cancer prevention: a meta-analysis of randomized controlled trials. *Nutrition and cancer*. 2010 Jul 23;62(6):719-27.
6. Rees K, Hartley L, Day C, Flowers N, Clarke A, Stranges S. Selenium supplementation for the primary prevention of cardiovascular disease. *The Cochrane Database of Systematic Reviews*. 2013;1(1):CD009671.
7. Abner EL, Schmitt FA, Mendiondo MS, Marcum JL, Kryscio RJ. Vitamin E and all-cause mortality: a meta-analysis. *Current Aging Science*. 2011;4(2):158-70.
8. Bjelakovic G, Nikolova D, Gluud C. Meta-regression analyses, meta-analyses, and trial sequential analyses of the effects of supplementation with beta-carotene, vitamin A, and vitamin E singly or in different combinations on all-cause mortality: do we have evidence for lack of harm?. *PLoS ONE*. 2013;8(9): e74558.
9. Chiang JY. Bile acids: regulation of synthesis. *J. Lipid Res*. 2009;50(10):1955-66.
10. Russell DW. The enzymes, regulation, and genetics of bile acid synthesis. *Annu. Rev. Biochem*. 2003;72:137-74.
11. Hofmann AF, Hagey LR, Krasowski MD. Bile salts of vertebrates: structural variation and possible evolutionary significance. *J. Lipid Res*. 2010;51(2):226-46.
12. Bernard A, Boumsell L, Hill C. Joint Report of the First International Workshop on Human Leucocyte Differentiation Antigens by the Investigators of the Participating Laboratories. In Bernard A, Boumsell L, Dausset J, Milstein C, Schlossman SF. *Leucocyte typing: human leucocyte differentiation antigens detected by monoclonal antibodies: specification, classification, nomenclature*. Berlin: Springer. 1984;45-48.
13. Isobe M, Huebner K, Maddon PJ, Littman DR, Axel R, Croce CM. The gene encoding the T-cell surface protein T4 is located on human chromosome 12. *Proc. Natl. Acad. Sci. U.S.A.* 1986;83(12):4399-402.
14. Jemilohun AC, Oyelade BO, Oiwoh SO. Prevalence of Hepatitis C virus antibody among undergraduates in Ogbomoso, Southwestern Nigeria. *African journal of infectious diseases*. 2014 May 1;8(2):40-3.
15. Brady RL, Dodson EJ, Dodson GG, Lange G, Davis SJ, Williams AF, Barclay AN. Crystal Structure of Domains 3 and 4 of Rat CD4: Relation to the NH₂-Terminal Domains. *Science-New York Then Washington-*. 1993 May 14;260:979-.
16. Diwe CK, Okwara EC, Enwere OO, Azike JE, Nwaimo NC. Sero-prevalence of hepatitis B virus and hepatitis C virus among HIV patients in a suburban University Teaching Hospital in South-East Nigeria. *Pan African Medical Journal*. 2013 Sep 10;16(1).
17. Ansari-Lari MA, Muzny DM, Lu J, Lu F, Lilley CE, Spanos S, Malley T, Gibbs RA. A gene-rich cluster between the CD4 and triosephosphate isomerase genes at human chromosome 12p13. *Genome Research*. 1996 Apr 1;6(4):314-26.