ejpmr, 2018,5(11), 81-86

SJIF Impact Factor 4.897

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Research Article

ISSN 2394-3211

EJPMR

HEPATITIS B AND HUMAN IMMUNODEFICIENCY VIRUS (HIV) CO-INFECTION IN PORT HARCOURT METROPOLIS, NIGERIA: A COMPARISON OF THEIR RENAL INDICES ASSESSMENT

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Article Received on 02/09/2018 **Article Revised on 24/09/2018** **Article Accepted on 15/10/2018**

ABSTRACT

HIV and HBV co-infection has emerged over last two decades as a serious public health challenge. This study was done to determine the renal function indicators in subjects who are human immunodeficiency (HIV) and Hepatitis B surface antigen (HBsAg) positive. One hundred (100) subjects made up of Twenty five (25) each for HIV positive, Hepatitis B surface antigen positive and HIV-Hepatitis Virus (HBV) co-infected and HIV and HBsAg negative individuals (Controls) had their sodium, Potassium, Chloride, bicarbonate, Urea and creatinine determined using VITROS auto analyser 250/350/950/5, 1 FS and 4600 Chemistry Systems and the VITROS 5600 Integrated System manufactured by Ortho-Clinical Diagnostics, Inc. The results showed that there was significant difference ($P<0.05$) in sodium, Potassium, Chloride, bicarbonate concentrations of HIV positive, HBV positive and HIV-HBV co-infected patients when compared to their respective controls while there was no difference in their Urea and Creatinine concentrations. The study has shown that HIV and HBsAg positive and HIV-HBV co-infection cause electrolyte imbalance. Therefore, there is need for adequate awareness among health care providers and the public on the effect of the spread of these viral infections, the consequence of their co-infection and their modes of prevention.

KEYWORDS: HIV, Hepatitis B, Infection, Renal Function, Port Harcourt.

1.0 INTRODUCTION

Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV) co-infection has emerged over last two decades to pose challenges to hepatologists, internists and infectious disease specialists globally. With both sharing similar modes of transmission and with the advent of highly effective antiretroviral therapy leading to longer survival of HIV positive patients, HBV positivity has become common in HIV positive patients (Ayodele, *et al*., 2016). In fact, of 33.3 million HIV positive patients worldwide (in 2009) while Nigeria has the second largest HIV epidemic in the world due to the size of Nigeria's population (meaning that, 3.2 million people were living with HIV in 2016 alone)(UNAIDS, 2017), which Aaron et al., (2017) attributed to social, attitudinal and cultural misinformation. Although HIV prevalence among adults is remarkably small (2.9%) compared with other sub-Saharan African nations such as South Africa (18.9%) and Zambia (12.4%),(UNAIDS, 2017) with recent facts showing that 1.6 million women are living with HIV compared to 1.4 million men. Women and girls also experienced higher infection rates, accounting for 53% of new infections in 2016 which according to Aaron et al., (2017) could be due to poverty and culture imbalance disfavouring the female gender. It is much more worrisome to note that, Rivers State has topped the new chart of HIV/AIDS prevalent states in Nigeria for 2017 with 15.2 percent according to Nigerhealthblog.com and http://www.nairaland.com/nghealth. Taraba state is on the second place on the chart with 10 percent, while Kaduna State was placed third with 9.2 percent. Other states present on this list include Nasarawa, FCT, AkwaIbom, Sokoto, Oyo and Benue States. Similarly, 4 million HIV patients had co-infectionwith HBV (Alter, 2006; Konopnicki*et al.*, 2005). Nigeria for example accommodates. Nigeria is categorized as an hyper-endemic region for HBV infection by (Ugwuja and Ugwu, 2010), at the same time, Port Harcourt city is noted to have a proportion of HBV-HIV co-infections (Frank- Peterside and Neenwi, 2010; Frank-Peterside and Ayodele, 2016). This simple data speaks volumeof the magnitude of the problem globally and in Port Harcourt. With, HIV taking pandemic scales in some part of the world, HIV and HBV co-infection is also taking significant proportion. In HBV endemic regions of

Africa and Asia, the majority of HBV infection is vertically transmitted at birth or before the age of five years (Modi and Feld, 2007). On the other hand in western countries, majority of the infection are acquired by sexual transmission or by intravenous route (Konopnicki*et al*., 2005; Soriano*et al.,* 2010). These differences in modes of transmission are clinically important regarding preventive methodology and with increased coverage of HBV vaccination; then, we can expect a decrease in new HBV infection, especially among young infants.

HIV and HBV virus both influence each other's natural history and it has significant clinical and therapeutic importance. HIV accelerates HBV associated liver disease, especially when CD4 count is low and HIV RNA load is high. HIV has a strong tropism for hepatic stellate cells through chemokine receptors (CXCR4 and CXCR5) and exerts direct cytopathic effects on liver6 (Babu*et al.,* 2009). Furthermore, HIV also triggers a pro-inflammatory cascade in liver leading to myofibroblastic differentiation, which may enhance fibrosis and cirrhosis in alreadydamaged liver(Tuyama*et al*., 2010). HIV infection promotes higher rates of chronicity after an acute exposure, greater levels of HBV replication in chronic carriers and lower levels of HBeAg or HBsAgsero-conversion. Recently, detection of HBV deoxyribonucleic acid (DNA) in HBV-HIV co-infected individuals have also been advocated by Ayodele, *et al*., (2016) as part of routine diagnosis for possible early detection or discouraged disease progression. Over all, the two major complications of HBV infection, namely cirrhosis and hepatocellular carcinoma are more prevalent in HIV positive patients (Martin-Carbonero*et al.,* 2011; Clifford *et al*., 2008). In fact, in western countries, liver related complications have become a leading cause of death in HIV positive persons and HBV is a major contributor. Therefore, studies like this in this part of the world would be very useful as it will assist to form a necessary database for statistical health forecast, planning and policy formulation. This study was conducted to determine renal function of HBVand HIV positive subjects using sodium, potassium, chloride, bicarbonate, urea and creatinine as indicators.

MATERIALS AND METHOD

Study Area

This study was carried out in Port Harcourt, the capital of Rivers State, with rich crude oil resources. The two centers where the subjects were recruited are spread to cover two distinct parts of the metropolitan city and they are reference points in the healthcare for a large majority of the populace.

Subjects

One hundred (100) subjects made up of Twenty five (25) each for HIV positive, HBV positive and HIV- HBV co-infected positive while the control was made up of twenty five (25) HIV and HBV negative subjects.

Ethical Approval

Ethical approval was gotten from the University of Port Harcourt Teaching Hospital, Port Harcourt, and Obio Cottage Hospital, Rumuobiakani, Port Harcourt from where the subjects in the study were recruited.

Consent

Written consent was obtained from all subjects after proper explanation and understanding of the procedure to be undertakenin the research and consenting individuals were recruited to be part of this study.

Sample Collection and Processing

All consenting subjects who were between $\geq 20 \leq 60$ years were recruited into the study. Blood sample equivalent to 5mls was collected aseptically from consented subjects, centrifuged at 3,500 revolutions per min (rpm) to obtain serum. This was used to screen for HIV and HbsAgusing determine HIV kits and HbsAgAbbot kits respectivelyand biochemical analyses for renal function markers which was carried out for each study group using VITROS auto analyser 250/350/950/5,1 FS , 4600 Chemistry Systems and the VITROS 5600 Integrated System manufactured by Ortho-Clinical Diagnostics, Inc.

Principles of the Analytical Procedures

The VITROS Cl^-Slide is a multilayered, analytical element coated on a polyester support that uses direct potentiometry (Siggard-Anderson 1986) for measurement of chloride ions. The slide consists of two ion-selective electrodes, each containing a protective layer, a silver layer and a silver chloride layer coated on a polyester support. The protective layer inhibits interference from normal levels of bromide and uric acid.The VITROS Na^+: The slide consists of two ion-selective electrodes, each containing methyl monensin (an ionophore for sodium), a reference layer, and a silver layer and a silver chloride layer coated on a polyester support. A drop of patient sample and a drop of VITROS Reference Fluid on separate halves of the slide results in migration of both fluids toward the center of the paper bridge. The VITROS BUN/UREA Slide is a multilayered, analytical element coated on a polyester support. The VITROS K^+Slide is a multilayered, analytical element coated on a polyester support that uses direct potentiometry for measurement of ionic potassium. The slide consists of two ion-selective electrodes, each containing valinomycin (an ionophore for potassium), a reference layer, and a silver chloride layer coated on a polyester supportA drop of patient sample and a drop of VITROS Reference Fluid on separate halves of the slide results in migration of both fluids toward the center of the paper bridge. A stable liquid junction is formed connecting the reference electrode to the sample indicator electrode. Each electrode produces an electrical potential in response to the activity of chloride ions applied to it. The potential difference poised between the two electrodes is proportional to the chloride concentration in the sample. The VITROS ECO_2Slide is a multilayered, analytical element coated on a polyester

support. A drop of patient sample is deposited on the slide and is evenly distributed by the spreading layer to the underlying layers. The high pH in the spreading layer ensures that essentially all CO_2 in the sample is in the bicarbonate form. Bicarbonate diffuses to the gel layer and is used to carboxylate phosphoenolpyruvate in the presence of phosphoenolpyruvate carboxylase to form oxalacetate and inorganic phosphate. The final reaction involves the malate dehydrogenase–catalyzed oxidation of NADH and reduction of oxalacetate to produce NAD^+ and malate.The slide is incubated at 37°C. The concentration of CO_2 in the sample is determined by measuring the absorbance of the unreacted NADH by reflectance spectrophotometry.

RESULT

The result of the study showed significant difference (P<0.05) in mean concentrations of sodium (Mmol/l), Potassium (Mmol/l), Chloride (Mmol/l) and bicarbonate (Mmol/l) in control, HBV-HIV Coinfection, HIV Positive and HBV Positive while there was no significant different (P>0.05) of urea(Mmol/l) and creatinine (Umol/l) in control, HBV-HIV Coinfection, HIV Positive and HBV Positive as shown table 1.

There was significant difference (P<0.05) in mean concentrations of sodium (Mmol/l), Potassium (Mmol/l), Chloride (Mmol/l) and bicarbonate (Mmol/l) in male control and female control compared with male HBV-HIV Coinfection, female HBV-HIV Coinfection, Male HIV Positive, Female HIV Positive, Male HBV positive and female HBV Positive while there was no significant different (P>0.05) of urea(Mmol/l) and creatinine (Umol/l) in male HBV-HIV Coinfection, female HBV-HIV Coinfection, Male HIV Positive, Female HIV Positive, Male HBV positive and female HBV Positive as shown table 1.

Table. 1: Gender Evaluation of Renal Function Indicesin Hepatitis B and HIV Positive Subjects.

Group	Gender	Sodium (Mmol/l)	Potassium (Mmol/l)	Chloride (Mmol/l)	Bicarbonate (Mmol/l)	Urea (Mmol/l)	Creatinine (umol/l)
Control	Male (a)	144.96±1.33	4.31±0.11	110.09±0.77	23.18±0.63	3.81±0.37	87.81±3.42
	Female(b)	137.52±1.26	4.39±0.11	107.21±1.10	20.57±0.69	3.14+0.12	64.5±2.59
HBV-HIV Coinfection	male(c)	126.48±1.51	3.73±0.10	91.33±1.31	11.91±1.33	5.25±2.46	100.83±20.6
	Female(d)	129.40±1.55	4.04±0.17	93.92±2.27	12.31±0.93	4.34±0.83	98.46±35.79
HIV Positive	Male(e)	142±1.60	4.97±0.48	106.25±2.95	14.50±0.86	4.25±0.99	89.25±22.70
	Female(f)	142.91±0.99	4.81±0.24	106.90±0.99	13.14±079	3.13±0.29	59.91±3.97
HBV Positive	male(g)	144.05±2.19	4.88±0.24	105.43±1.22	19.43±1.64	4.77±0.39	77.64±1.34
	Female(h)	138.33±1.79	4.61±0.21	104.36±2.29	17.36±2.21	6.22±2.61	89.09±23.66
	F	19.360	3.751	19.354	11.553	0.785	0.927
	P	0.000	0.001	0.000	0.000	0.602	0.489

There was significant difference (P<0.05) in mean concentrations of sodium (Mmol/l), Potassium (Mmol/l), Chloride (Mmol/l) and bicarbonate (Mmol/l) in control at age groups (years) 20-30, 31-40, 41-50 and 51-60 compared with HIV-HBV Coinfection, at age groups 20-30, 31-40, 41-50 and 51-60, HIV Positive at age groups 20-30, 31-40, 41-50 and 51-60 and HBV Positive at age groups 20-30, 31-40, 41-50 and 51-60 while there was no significant different (P>0.05) of urea(Mmol/l) and creatinine (Umol/l) in in control at age groups 20-30, 31-40, 41-50 and 51-60 compared with HIV-HBV Coinfection, at age groups 20-30, 31-40, 41-50 and 51-60, HIV Positive at age groups 20-30, 31-40, 41-50 and 51-60 and HBV Positive at age groups 20-30, 31-40, 41-50 and 51-60 as shown table 2.

Table. 2: Renal Function Parameters in Different age groups in Hepatitis B and HIV positive individuals.

Group	Age group	SODIUM (Mmol/l)	POTASSIUM (Mmol/l)	CHLORIDE (Mmol/l)	BICARBONATE (Mmol/l)	UREA (Mmol/l)	CREATININE (umol/l)
Control	20-30	142.39±2.00671	4.3714±.07781	108.57±1.52530	21.7143±.80812	3.0429±.28440	69.7143±5.62611
	31-40	136.73±1.35010	4.2778±.11759	106.11±1.20698	22.555±.70929	3.4556±.28921	74.4444±5.65713
	41-50	143.47±2.59612	4.4714±.22223	110.71±.91844	21.571±1.17224	3.8000±.48008	78.8571±6.54446
	51-60	144.15±1.65000	4.2500±.25000	111.00±.00000	18.5000±3.50000	3.5000±.40000	79.5000±6.50000
HBV-HIV Co-infection	20-30	130.74±1.11834	4.0000±.17627	94.1250±1.72624	14.6250±.80039	3.3375±.14260	69.8750±5.23531
	31-40	127.84±1.43440	3.8333±.12019	92.0833±2.01682	11.3333±1.09637	3.8583±.96809	108.75±38.41759
	41-50	124.00±3.53030	3.8800±.37868	91.8000±4.17612	10.0000±2.23607	9.3000±5.74326	125.20±49.33295
	51-60						
HIV Positive	20-30	140.65±2.65000	5.7500±.45000	107.50±.50000	12.5000±.50000	3.1000±1.20000	57.0000±4.00000
	31-40	144.05±1.58575	4.9727±.43923	108.00±1.57826	13.5455±1.06484	2.7000±.33494	57.1818±7.03086
	41-50	142.03±1.1661	4.6111±.21631	104.67±1.16667	13.0000±1.37437	3.9667±.55202	75.5556±10.06798
	51-60	141.87±2.14968	4.3667±.12019	108.33±3.75648	14.3333±1.20185	3.7333±.96148	64.0000±5.50757
HBV Positive	20-30	145.43±2.79154	5.1714±.39864	104.86±1.53419	20.4286±2.41875	4.8143±.57214	79.8571±1.99319
	31-40	137.40±3.15527	4.4125±.20392	102.38±3.34844	13.7500±2.09378	7.1375±3.59300	93.3750±32.97236
	41-50	142.11±1.79663	4.7600±.22910	107.10±.60461	21.0000±1.80739	4.4500±.42798	76.1000±1.78543
	51-60						
	F	10.102	2.637	10.955	8.365	1.052	.0.637
	P	0.000	0.004	0.000	0.000	0.412	0.817

DISCUSSION

The result of the study has shown that there was significant difference in sodium, potassium, chloride and bicarbonate of HIV Positive, Hepatitis B virus positive and HIV-Hepatitis B Virus co-infection compared with their respective controls. With dramatic improvements in survival and disease progression in the era of combination antiretroviral therapy (ART), complications such as kidney, liver, and cardiac disease have largely replaced opportunistic infections as the leading causes of mortality among people living with HIV (Selik*et al*., 2002). Patients with HIV are at risk for both acute kidney injury (AKI) and chronic kidney disease (CKD) (Lucas et al., 2014), secondary to medication nephrotoxicity, HIV-associated nephropathy (HIVAN) (Gardenswartz*et al*., 1984,Pardo*et al*., 1984,Rao*et al*., 1984,D'Agati and, Appel 1998) immune complex kidney diseases (D'Agati and, Appel 1998 Casanova *et al*., 1995, Kimmel *et al*., 1993;Balow 2005; Kimmel *et al*., 1992) and, less commonly, kidney disease in the setting of thrombotic microangiopathy (Boccia*et al*., 1984,Bachmeyer*et al*., 1995). In addition, the aging cohort of HIV-positive patients may be at increased risk for kidney disease related to hepatitis B or C virus co-infection (D'Agati and Appel 1998;Stokes *et al*., 1997; Lai *et al*., 1991) and comorbid or treatment-related diabetes and hypertension.

The result of the study also showed that there was significant difference in sodium, potassium, chloride and bicarbonate of male and female subjects who were HIV positive alone, Hepatitis B virus positive and HIV- HBV co-infection positive compared with their respective controls.This is similar to studies by Ando *et al*., (2012), Rasch*et al*.,(2013), Islam *et al*., (2012), Roe *et al*.,(2008).Other predictors of AKI included low CD4 count, high viral load, and co-infection with hepatitis C virus (HCV). Hyper-kalaemia is a common feature of renal tubular acidosis type 4 which results from a defect in hydrogen and potassium secretion in the distal tubules rather than from aldosterone deficiency (Battle *et al*., 1981). This could explain the reason for the increased serum potassium level in hepatitis B carriers. Acute tubular necrosis involves damage to the renal tubules and can be a complication in individuals with hepatitis B infection as a result of diminished reabsorption of potassium by the tubules (Gines& Arroyo, 1999). Lai & Lai (1991) reported a diminution in glomerular filtration capacity as a renal complication of hepatitis B viral infection. Also, the DNA of the hepatitis B virus was found in the nearby renal tubules, where urine is concentrated, which suggests that the virus replicates in the tubules of the kidney.

CONCLUSION

This study has shown that HIV and HBsAg positive and HIV-HBV co-infection cause electrolyte imbalance. Therefore, there is need for adequate awareness among health care providers and the public on the effect of the spread of these viral infections, the consequence of their co-infection and their modes of prevention.One of the major barriers to accessing HIV prevention programmes for men who have sex with men are laws that prohibit their activities. For example, same-sex relations in Nigeria are criminalised with 14 years imprisonment. This is not only limiting access for HIV prevention programming for this community, but causing nationwide stigma and discrimination against people based on their sexual orientation. The feminisation of Nigeria's HIV epidemic is in part due to the gender inequality that is embedded in its society, culture and law.

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