

Co- Infection of HIV and HepatotropicViruses: Impact on the Biochemical and HaematologicalMarkers of Patients in Northeastern Nigeria.

ABSTRACT

Aims: Co-infection of hepatotropic viruses with human immune deficiency virus (HIV) increases the risk of progression to severe liver disease. The study was designed to assess the impact of HIV mono and co-infections on haematological and biochemical markers of the patients.

Methodology: 109 HIV patients (, aged 10-59 years, 78 males and 31 females) from tertiary health facilities in northeastern Nigeria were confirmed by western blot prospectively and consecutively. However, Hepatitis B virus (HBV) and Hepatitis C virus (HCV) antibodies were detected on the HIV positive patients by ELISA. Blood donors served as control. The study was conducted between January to October 2012.

Results: 14(12.8%) and 5 (4.6%) of the HIV patients had HBV and HCV antibodies respectively. The prevalence rate of HBV infection among males was 12.8% while females had 12.9% but lower rates of HCV were obtained in both males (5.1%) and females (3.3%). However, HIV mono-infections had higher mean baseline values for haemoglobin (Hb), CD4 and platelet counts, protein C (PC) and protein S (PS) in comparison with HIV/ HBV/HCV co-infections ($P<0.05$). In addition, Prothrombin time and partial thromboplastin time were lower in HIV mono- infection in contrast to co-infections ($P<0.05$). Similarly, Serum liver enzymes such as Aspartate transaminase (AST), Alanine transaminase (ALT), Akaline Phosphatase (ALP) and creatinine mean values were lower in HIV mono-infection compared with HIV/HBV or HIV/HCV co-infection ($P<0.05$). Total white blood cell count (Wbc), total cholesterol (Tch), Randon blood sugar (RBS) and potassium (k^+) were not significantly different ($P>0.05$) in both groups.

Conclusion: The high prevalence rate of HBV suggests the need to routinely screen HIV patients for the virus. The pathological implication of HIV co-infection with hepatotropic viruses is a pointer to increased risk of hepatic dysfunction, coagulation and metabolic disturbances which consequently increase the disease burden on the HIV patients.

KEYWORDS: HIV, Hepatitis B virus, Hepatitis C virus Co-infection, haematological and biochemical markers.

INTRODUCTION

Co-infection of human immunodeficiency Virus (HIV) with hepatotropic viruses such as Hepatitis B and Hepatitis C viruses is an emerging global public health problem. [1, 2] The three viruses are blood borne pathogens and share similar routes of transmission; [3] As deaths caused by HIV disease are declining due to the introduction of Highly Active Antiretroviral Therapy (HAART), liver disease associated with co-infection of HIV with hepatotropic viruses has become an increasing cause of morbidity and mortality [4]. The consequences of arbovirus infections in patients living in under-resourced settings are enormous with considerable implication on the progression, management and outcome of HIV disease [5, 6]. Increased risk of progression to liver cirrhosis and hepato-cellular carcinoma has been reported as a result of these co-infections [7, 4, and 8]. Previous reports have revealed that, the prevalence of HIV in Nigeria has been stabilized within the range of 4.4 and 4.1 from 2005 to 2010 [9]. Other studies had observed HIV/HBV sero-prevalence rate of 5.5% – 12.3% [10, 11, 6, 12, and 13] and HIV/HCV rates of 0.5 – 0.7% [14, 6, 12, and 13]. However, there is paucity of information on the impact of HIV/HBV/HCV co-infection on some baseline pathological markers. HIV and hepatic disease are independently known to cause a varied range of haematological changes. This is partly because of the enormous assault of these viruses on haemopoietic cells/system and the role of the liver in haemopoiesis and coagulation [15]. Also hepatotoxicity of Antiretroviral Therapy (ART) administered to AIDS patients, impact negatively on the central role of the liver in many metabolic processes. Consequently, these diseases pose a serious risk on the patient's health. [16, 17].

With this background, we set out to investigate the implications of these co-infections on some haematological and biochemical biomarkers in patients attending health care facilities in North Eastern Nigeria.

MATERIALS AND METHODS

Study area: The study was conducted in the Medical Center Ashaka Gombe State and University of Maiduguri Teaching Hospital (UMTH) Borno State. UMTH is a 560 bedded health facility designated as a Centre of Excellence for infectious disease and immunology. It provides primary, secondary, and tertiary health Services for the North Eastern part of Nigeria. It also houses a major HIV care and support Centre and a National blood transfusion Centre. Neighboring countries (Cameroun, Niger and Chad Republics) avail themselves of services provided by UMTH. Maiduguri the capital of Borno State is the largest settlement in the fringe of Sahara desert and near to the Lake Chad. It is located between longitude $11^{\circ}8^{\circ}\text{E}$ and 14°E and latitude $10^{\circ}2^{\circ}\text{N}$ and $13^{\circ}4^{\circ}\text{N}$.

Study Population: Patients and blood donors confirmed to be seropositive for human immunodeficiency Virus (HIV) infection by Western blot technique were recruited into the study from January to October 2012. Informed consent and pre-test counseling were instituted using structured questionnaire. Information concerning sexual behavior, blood transfusion, intravenous drug use and demographic data were obtained. Blood donors who were HIV, Hepatitis B virus (HBV) and Hepatitis C virus positive were recruited for the study and healthy blood donors served as controls.

Sample collection and analysis for haematological and biochemical parameters: 10 millilitres (ml) of venous blood was obtained from each participant; 2ml was dispensed into EDTA containers for Haemoglobin (Hb), white blood cell count (wbc) and platelet (PLT) count. Haematological counts were performed using automated blood analyzer sysmex Kx – 21, and its dedicated reagent kits, 4ml of blood was collected into plastic bottle containing sodium citrate (0.11molar solution) to give a final blood/citrate ratio of 9:1. Platelet poor plasma obtained by centrifuging immediately at 3000g was used for prothrombin time, partial thromboplastin (PTT) time, protein C (PC) and protein S (PS) assay through an automated coagulometer sysmex 560 series (S/N1016 Japan) using reagent acquired from Siemens

Health care diagnostics products GmbH 36041 Marburg Germany. 4ml of blood was allowed to clot in a plane blood bottle and serum separated immediately and used for Aspartate transammase (ASP) Alanine transaminase (ALT), Alkaline phosphatase (ALP), Creatinine (creat), total cholestrolal (TCh), random blood sugar (RBS) and Potassium (k+). The analysis was carried out using automated blood analyser Hitachi (Cobas C311 leums) S/N 046-02 using reagent kits manufactured by cobas Roche diagnostics GmbH-D-68298 Mannheim.

Screening for HIV, HBV, HCV

HIV screening was carried out using Immuno chromatographic kit (Chembio HIV 1 and 2 STAT-PAK). Positives samples were further confirmed by Western blotting (Qualicode™ HIV 1 and 2 kit). Similarly Hepatitis B surface antigen (HBsAg) and HCV antibodies were tested by enzymes linked immunosorbent (ELISA) assay kits (DIA PRO, Diagnostic Bioprobesthroughcolumella No 20128 Milano-Italy).

STATISTICAL ANALYSIS

Statistical package for social science (SPSS) version 17 was used and data obtained presented as mean (x) and standard deviation (SD). Statistical comparison performed using student test Chi-square (X_2) and ANOVA.

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RESULT

A total of 109 HIV positive patients were enlisted for the study. Males were 78 (72.2%) and females 31 (27.7%). The mean age of both sexes was 36 ± 20 (16 – 59) years. Age and sex matched apparently healthy subjects (blood donors: N=100) consisting of 78 (78%) males and 22 (22%) females were also recruited. These served as controls against the HIV infected individuals.

Out of 109 HIV positive patients, 14 (12.84%) were positive for HBsAg and 5 (4.6%) had HCV antibody. Table 1 represents the age distribution of HIV mono-infections, co-infections of HIV/HBV and HIV/HCV. The prevalence rate of HIV mono-infections appeared to decrease with increase in age with the least (3.7%) at ages 10-19 and 50-59years. Although a similar trend was observed in HIV/HBV co-infection, those co-infected with HCV showed no defined pattern. However, HIV/HCV co-infections were more common among those aged 40-49years compared with other age groups. There was no age related differences between or within groups ($P=0.05$).

TABLE 1: Agedistribution of patients with HIV mono-infections, HIV/HBV and HIV/HCV co-infections.

Age group	HIV –mono-infections		HIV/HBsAg		HIV/HCV	
	No tested	%positive	No tested	No positive (%)	No tested	No positive (%)
10 – 19	14	3.7	14	0 (0)	14	1 (0.9)
20 - 29	43	39.4	43	9 (8.3)	43	0 (0)

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30 – 39	42	38.5	42	5 (4.6)	42	0 (0)
40 – 49	16	14.7	16	0 (0)	16	3 (2.8)
50 – 59	4	3.7	4	0 (0)	4	1 (0.9)
Total	109	100	109	14 (12.9)	109	5 (4.6)

The gender of patients with HIV mono and co-infections is presented on table 2. A total of 14 (12.96%) of 109 HIV patients were positive for HBsAg, 10 (12.82%) males and 4 (12.90%) females. HIV/ HCV co-infection was positive for 5 (4.6%); males, 4 (5.1%) and female 1 (3.3%). No significant difference was observed among males and females with HIV mono or co-infections. The controls (blood donors) neither had HIV nor the hepatotropic virus antibodies.

Table2: Gender distribution of patients with HIV/ HBV and HIV/ HCV antibodies

Gender	No Tested	HIV positive (%)	HIV/HBV positive (%)	HIV/HCV positive (%)
HIV Patients				
Males	78	78(100)	10(12.82)	4(5.1)
Female	31	31(100)	4(12.9)	1(3.2)
Control				
Males	78	0(0)	0(0)	0(0)
Female	22	0(0)	0(0)	0(0)
	209	109 (52.1)	14 (6.7)	5 (4.6)

Table 3 reflects the haemotological parameters of HIV mono-infection, co-infected patients and the controls. Whilst Haemoglobin (Hb), CD4 counts, platelet count (PLT), Protein C (PC) and Protein S (PS) values were significantly higher ($P<0.05$), prothrombin time (PT), partial thromboplastin time (PTT) were lower compared with co-infected cases, but the values of biomarkers (Hb, CD4, PLT, PC, and PS) are much higher among the controls while PT and PTT are much lower. Nonetheless, total white cell count (wbc) showed no significant

difference between the groups. ($P>0.05$)

124 **Table 3: The haematological parameters between HIV-mono and co-infected patients**

S/No	Parameters	HIV mono-infection	HIV /HBV	HIV/HCV	Controls
1	Hb g/dl	11.29±1.89	10.02±1.19	9.060±1.02	13.7±0.91
2	Wbc ($\times 10^9/l$)	5.46±1.56	4.14±1.08	3.72±0.62	5.30±1.38
3	CD4 (cell/ μ L)	217.28±47.56	109.89±49.40	156.40±24.17	504.12±213.89
4	PLT ($\times 10^9/L$)	198.13±60.64	111.21±31.78	153.80±58.45	274.60±85.24
5	PT (Seconds)	13.00±11.98	14.70±0.97	14.30±1.20	12.4±2.14
6	PTT (Seconds)	34.88±14.99	49.64±9.06	46.40±9.12	31.90±5.74
7	PC (%Activity)	82.02±10.32	67.79±8.44	74.00±13.02	94.22±13.50
8	PS (%Activity)	80.07±10.93	68.14±14	54.060	95.71±17.06

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127 Table IV displays the mean values of some biochemical markers of the HIV mono-infections and co-infections with

128 hepatotropic virus. The mean values of Liver enzymes (aspartate transaminase, alanine transaminase and alkaline

129 phosphatase) as well as Creatinine were significantly higher ($P<0.05$) among the co-infections compared to mono-

130 infections and controls. However, the mean values for total cholesterol, random blood sugar and potassium showed no

131 significant difference ($P>0.05$) in the three groups.

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133 **TABLE IV: Biochemical parameters between HIV mono and co-infections**

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S/No	Biochemical Parameters	HIVmono- infections	HIV/HBV	HIV/HCV	Controls
1	AST (u/l)	23.66±7.57	66.93±20.74	66.80±15.64	14.7±11.56
2	ALAT (u/l)	24.89±8.14	46.00±15.63	36.60±9.29	25.0±11.56
3	ALKP (u/l)	65.30±22.37	94.36±13.608	102.80±9.71	73.30±29.63
4	CREAT (μmol/l)	59.75±26.95	89.5±24.80	91.60±17.69	40.00±31.39
5	TCH (mmol/l)	5.23±4.41	5.057±1.57	4.12±0.69	5.91±1.31
6	RBS (mmol/l)	4.70±1.64	4.60±1.18	3.78±1.25	5.0±1.96
7	K ⁺ (mmol/l)	3.84±1.30	4.27±1.01	3.20±0.61	3.44±0.76

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136 DISCUSSION

137 Globally, HIV is responsible for 38.6 million infections with Africa having about 10% of the world population
 138 carrying about 75% of HIV/AIDS burden [18]. Prevalence rates in East and Southern Africa appears to be
 139 the highest in the world with Botswana and Swaziland bearing over 35% of the African burden [19].
 140 Prevalence rate in West Africa appears to be lower with no country having a rate above 10% [20]. Since the
 141 principal routes of HIV and hepatotropic viruses such as HBV and HCV are similar, it is possible that co-
 142 infection of these viruses with HIV could occur. It has been estimated that one third of deaths in HIV patients
 143 are related to liver diseases[8]. Co-infections of HBV and HCV with HIV have been associated with reduced
 144 survival due to increased risk of progression to cirrhosis and hepato-cellular carcinoma [7]. HIV/HBV co-
 145 infection is linked most often to sexual intercourse while HIV/HCV co-infection has been more frequently
 146 associated with non-sexual parenteral route [21]. In this study, most of those studied are heterosexually
 147 active with a substantial number having multiple sex partners. This finding is in consonant with a report that,
 148 the commonest route of transmission for blood borne viruses are sexual intercourse (both heterosexual and
 149 homosexual) followed by intravenous drug use (IDU) including transfusion of blood and blood products
 150 [21,22]. None reported intravenous drug use or blood transfusion. This may be responsible for the low
 151 prevalence rate of HIV/HCV in this study. This observation is in agreement with previous reports that HCV is
 152 not efficiently transmitted by sexual routes[22, 6,13], but by percutaneous exposure to blood/products and
 153 IDU in particular [23, 12]. It has been reported that, IDU is the most efficient route for triple infection with

HIV/HBV/HCV in an Urban population [24, 25]. This may explain why none of the patients in this study presented with triple infection. However, this study recorded HIV/HBV co-infection prevalence rate of 12.8% in agreement with [14]. (2008) who reported a similar rate in South Western part of Nigeria.

Nevertheless, there is dearth of information on the implication of HIV/hepatotropic virus co-infections on baseline haematological and biochemical biomarkers in Nigeria. Nevertheless, it has been established that, HIV disease impacts adversely on haematological profile of a patient due to the enormous assault of the virus on haemopoietic cells/system in addition to hepatotoxicity and nephrotoxicity of anti-retroviral drugs (ARD) [26, 27] administered to AIDS patients. Our study has further demonstrated that co-infection with hepatotropic virus worsens the morbidity burden of HIV because Hb, CD4 count, platelet, PT, PTT, PC and PS presented values significantly dysregulated in the co-infections with hepatotropic viruses compared to HIV mono-infection. It would appear as evidenced by CD4 count in our study that the degree of immune suppression may increase with hepatotropic virus's co-infections such as HBV and HCV. This is in agreement with International Association of Providers of AIDS Care (IAPAC) [28], but differs with the reports made by Idoko et. al., [29] in the North Central region of Nigeria and Otegbayo et. al., [14] in the South West. Generally haematological profile represents an evidence of the general health status and well-being of an individual; abnormal values in co-infection conforms to increasing morbidity. Co-infection of HBV and HCV with HIV have been associated with increased risk of progression to liver disease (Cirrhosis) and are attributed to liver dysfunction which may occur due to HBV, HCV or hepatotoxicity of antiretroviral therapy (ART) [30]. The liver is central in the coagulation profile of an individual and synthesizes most of the coagulation factors that interplay in the coagulation mechanism. In addition, PC and PS which are usually synthesized by the liver are natural anticoagulants that contribute to the balancing mechanism of haemostasis by preventing wide spread clotting and occlusion of blood vessels [27, 31]. The higher values of these anticoagulant proteins in HIV mono-infections compared with the co-infections in this study could be attributed to the impact of HIV/HBV/HCV co-infections. Previous studies revealed that low CD4⁺ cell count (especially in the presence of clinical AIDS) protein S and protein C deficiency are strongly associated with two to tenfold increased risk of venous thrombosis (VTE) in HIV infections [32, 33, 34]. It could be speculated in this study that, low level of these proteins could advance the course of VTE in HIV co-infections even before the manifestation of AIDS. Liver enzymes AST, ALT ALKP levels are higher in HIV/HBV/HCV co-infection as evidenced by the mean value of these biomarkers when compared with the values in HIV mono-infection. The high values of these

biomarkers depict degenerative liver diseases in contrast to a report [12]that, liver enzymes are stable in HIV mono and co-infections.

Lower Creatinine values (a test of renal function) were also, obtained among HIV mono-infections compared to HIV/ HBV and HIV /HCV co- infections. This is in consonance with the report of Parboosinget. al.,] 35] who documented renal insufficiency with elevated creatinine levels and increased mortality in HIV/HCV co-infected patients in South Africa. Also according to IAPAC,[28], HIV Associated Nephropathy (HIVAN) is worsened by co-infection with HBV or HCV. In this study wbc count, total cholesterol, random blood sugar and potassium were all stable in the three groups as there were no significant difference between mono-infection and co-infections.

In conclusion, HBV and HCV infections are not uncommon among HIV positive patients in our environment and worsen the disease burden on HIV patients. Consideration should be given to these hepatotropicviruses during baseline work-up of HIV patients so that susceptible ones can be identified and managed properly and timely. HIV co-infections with HBV and HCV could enhance VTE due to depletion of protein S and protein C. In addition, strategies to prevent these co-infections amongst HIV infected patients should be encouraged .Such efforts will to a great extent, ameliorate the impact of the co-infection on the health status of HIV patients.

REFERENCES

1. SorianoV, Nunez M, Camino N, Garcia-Samaniego, Puoti M, Rockstroh J. Treatment of hepatitis C in HIV infected patients Hepatol Rev 2004; 1 (2):59-71.
2. Koziel MJ, Peters MG. Viral hepatitis in HIV infection. N. England J Med 2007; 356: 1445 – 1454.
3. Pseudos G (Jr), Kim JH, Suh J, Shar VL. Coinfection of hepatitis B and hepatitis C Virus in HIV infected patients in New York City USA. World JGastroenterol. 2008; 14 (43): 6689–6693
4. Weber R, Sabin CA, Friis-Moller N, Rasmussen P, EL-sajr WM, Kirk O, Dabis. Liver-related deaths in persons infected with the human immunodeficiency Virus: The DAD study Arch int Med. 2006; 166 (15) :1632-1641.
5. Nwokedi EE, Emokpae MA, Dutse AI. HIV and hepatitis B Co-infection among patients in Kano Nigeria Niger J Med. 2006; 15(3): 217-229.

- 212 6. Adewale O O, Antegi E. Hepatitis B and C Virus co-infection in Nigeria patients with HIV infection
213 the Journal of infection in developing countries . 2009; 3(5):369-375.
- 214 7. Furusayo N, Nakashima H, Kashiwagi K. Clinical outcomes of hepatitis B virus (HBV) genotype B
215 and C in Japaneses patients with chronic HBV infection Am J Trop MedHyg. 2002; 67: 151-157
- 216 8. Kumarassamy N, Vaccabhamenis, Flanigan TP, Mayer KH, Solomon S. Clinical profile of HIV in
217 India. India J Med Res 2005; 121: 377-394.
- 218 9. Federal Ministry of Health, Nigeria, 2010. Department of Public Health National AIDS/STI Control
219 programme, Technical report for National HIV sero-prevalence Sentinel Survey. Available at
220 [http://www.nigeriaaids.org/documents/2010_National%20HIV%20Sero%20Prevalence%20Sentinel%](http://www.nigeriaaids.org/documents/2010_National%20HIV%20Sero%20Prevalence%20Sentinel%20Survey.pdf)
221 [20Survey.pdf](http://www.nigeriaaids.org/documents/2010_National%20HIV%20Sero%20Prevalence%20Sentinel%20Survey.pdf). Accessed November 12 2013
- 222
- 223 10. Baba M M, Gashau W, Hassan AW. Infection of hepatitis B surface antigenaemia in Patients with
224 and without the manifestations of AIDS in Maiduguri, Nigeria. Nig Postgrad Med J 1998; 5: 125-128.
- 225 11. Egabi D Z, Bauwat E B, Audu E S, Iya D, Maudong B M. Hepatitis B surface antigen, Hepatitis C and
226 HIV antibodies in low-risk blood donor group in Nigeria. East Mediterr Health. J.2007;13(4):123-127.
- 227 12. BallahAkawuDenu, BabajideAjayi, AbubakarUsmanAbja, AbubakarAbdullahiBukar, Cecilia Akawu,
228 Ernest Ekong and Mohammed Bashir Alkali. A survey of hepatitis B and C virus prevalence in HIV
229 positive patients in a tertiary health institution in North Eastern Nigeria. International of Medicine and
230 Medical Science. 2012; 4(1): 13-18.
- 231 13. OkekeTC, Obi SN, Okezie OA, Ugwu EO, Akogu SP, et al. Coinfection with hepatitis B and C
232 viruses among HIV positive pregnant women in Enugu south east, Nigeria. Niger J Med. 2012; 21:
233 57-60
- 234 14. OtegbayoJ.A,Taiwo B O Akingbola TS. Prevalence of HBV and HCV Seropositivity in a Nigeria
235 Cohort of HIV infection ANN Hepatol 2008; 7: 152-156
- 236 15. Mehta A B., McLutye N,(1998) Haematological disorders in Liver disease. 2008; Trends in
237 experimental and clinical Medicine Forum 8:8-25
- 238 16. Alter M J. Epidemiology of Viral hepatitis and HIV co-infection. J Hepatology. 2006; 44: 56-59
- 239 17. Sulkowshi M S, Thomas DL, Charsson R E Moore D C. Hepatotoxicity associated with antiretroviral
240 therapy in adults infected with HIV and the role of HCV or HBV infection JAMA. 2000; 283: 74-80
- 241 18. UNAIDS. Report on Global AIDS Epidemic Geneva. 2004
- 242 19. UNAID. AID in Africa Three Scenarios Geneva. 2005.

- 243 20. Nasidi A, Harry T. HIV mode of transmission in: AIDS in Nigeria. Harvard University Press USA.
244 2005; 2:20-26.
- 245 21. Shermon KE, Rouster SD, Chung RT, Rajjicic N. Hepatitis C Virus Prevalence among Patients
246 infected with Human Immunodeficiency Virus across-sectional analysis of the US adult AIDS Clinical
247 trial Group Clin Infect Dis. 2002; 34: 831-837.
- 248 22. Kellerman SE, Hanson DL, McNaghten AD, Fleming PL. Prevalence of chronic hepatitis B and
249 incidence of acute hepatitis B infection in HIV. Infect Dis J. 2003; 188: 571-577
- 250 23. Wasley A, Alter MJ. Epidemiology of hepatitis C: geographic difference and temporal trends semin
251 Liver Dis. 2000; 20: 1-16
- 252 24. Maier I, Wu GY. Hepatitis C and HIV co-infection: a review. World J Gastroenterol 2002; 8:577-579.
- 253 25. Aceijas C, Abodes T. Global estimate of Prevalence of HCV infection among injecting drug users. Int.
254 J. Drug Policy. 2007; 18:352-358.
- 255 26. Spivak JL. Blood in systemic disorders. Lancet 2002; 355: 1707-1712.
- 256 27. Klein SK, Slim EJ, deKruif MD, Keller TT, ten Cate H, van Gorp ECM, Brandjes DPM. Is chronic HIV
257 infection associated with venous thrombotic disease? A systematic review. Netherlands journal of
258 medicine. 2005; 63(4):129 - 135.
- 259 28. International Association of Providers of AIDS Care (IAPAC) (2013). HIV and kidney disease. Fact
260 sheets 124, 506/507 and 651. www.aidsinfonet.org/fact/sheet. Accessed 8/11/2013.
- 261 29. Idoko J, Meloni S, Muazu M, Nimzing N, Badung B, Hawkins C. Impact of HBV infection on HIV
262 Response to Antiretroviral Therapy in Nigeria. Clin Infect Dis. 2009; 48(8): 1268-1273.
- 263 30. Rath PM, Amarapurkar DN, Borges N E, Koppikar G V, Kairo R H. Spectrum of liver disease in HIV
264 infection India J. Gastroenterol. 1997; 16:94-95.
- 265 31. Javier Carbone. Immune activation and increased prevalence of thrombosis in HIV infection. Journal
266 of AIDS. 2007; 46(3):375 – 376
- 267 32. Bibas M, Biava Gianluigi, and Antinori Andrea. HIV-Associated Venous Thromboembolism Mediterr J
268 Hematol Infect Dis. 2011;3(1): ISSN 2035-3006).
- 269 33. Katie LK, Melissa Badowshe. Risk factors for venous thromboembolism in patients with HIV -
270 infection. Pharmacotherapy. 2011; 30(12):1293 - 1302.
- 271 34. Eyal A, Vella M. HIV and venous Thrombotic event. South African Journal of surgery. 2009; 7:93 -
272 95.

35. Parboosing R, Paruk I, LallooUG. Hepatitis C virus sero-positivity in a South African cohort of HIV co-infection in ARV naïve patient is associated with renal insufficiency and increase mortality. J Med Virology. 2008; 80(9): 1530-1530.

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Declaration of originality: All authors wish to affirm that this article has not been published in any other journal

CONSENT (WHERE EVER APPLICABLE)

All authors hereby declare that informed consent and pre-test counseling were instituted using structured questionnaire. A copy of the written consent and questionnaire are available for review by the editorial office/chief editor/editorial board members of this journal."

ETHICAL APPROVAL (WHERE EVER APPLICABLE)

The ethical committee of University of Maiduguri Teaching Hospital and Ashaka Cement Medical Centre Gombe, Nigeria gave approval for the study

Suggested Reviewers

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Any other comment:

The project was self-sponsored with contributions from each of the authors. It serves as a measure of assessing severity and devastating effect of co-infections of HIV with hepatotropic viruses such as HBV and HCV. We cannot find another journal that is more suitable for the dissemination of this information than yours.