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Association between 8-oxoguanine glycosylase 1 polymorphism and some markers of liver detoxification in people living with human immunodeficiency virus

Abiodun Mathias Emokpae[1], Akinlawon Adetiloye Adepeju[2]

[1]Department of Medical Laboratory Sciences, College of Medical Sciences, University of Benin, Benin City, Nigeria
[2]Department of Chemical Pathology, UNIOSUN Teaching Hospital, Osogbo, Nigeria

Abstract

Introduction: Single nucleotide polymorphisms in 8-oxoguanine glycosylase 1 (*OGG1*) gene play protective role against 8-hydroxy-2-deoxyguanosine (8-OHdG) mutation, and may influence individual's response to treatment in human immunodeficiency virus (HIV) infection. The current study determined the impact of *OGG1* polymorphisms on some markers of liver function and antioxidants among people living with HIV (PLWH).

Material and methods: This cross-sectional study was conducted among 200 HIV-positive individuals, attending outpatient clinic dedicated to PLWH of Ladoke Akintola University of Technology Teaching Hospital (LAUTECH), Osogbo, Nigeria, and 100 HIV-negative subjects as controls. A structured questionnaire was administered to collect relevant medical and socio-demographic data. Multistage random technique was employed in enrolling subjects for the study. Serum glucose, total protein, albumin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), vitamin E, and glutathione reductase (GR), were determined using spectrophotometric and enzyme-linked immunosorbent assay methods. Data were analyzed with Student's *t*-test, χ^2 test, analysis of variance, and Pearson's correlation coefficient.

Results: ALT ($p < 0.002$) and AST ($p < 0.001$) were significantly higher, while GR ($p < 0.008$) and vitamin E ($p < 0.01$) were significantly lower among patients with OGG1 genotype CC than those with genotypes GG + CC. ALT ($p < 0.033$) was significantly higher, and GR ($p < 0.001$) was significantly higher among subjects with viral load below 1,000 copies than those with viral load above 1,000 copies.

Conclusions: The findings of this study suggest that single nucleotide polymorphism in *OGG1* gene may play significant role in individual's response to antiretroviral drugs and disease progression, and patients with genotype CC are likely to response poorly compared with those with GG and CC genotypes.

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Key words: 8-oxoguanine, HIV infection, liver, humans.

Address for correspondence: Akinlawon Adetiloye Adepeju, Department of Chemical Pathology, Uniosun Teaching Hospital, Osogbo, Nigeria, e-mail: akinla2001@gmail.com

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Introduction

Human immunodeficiency virus (HIV) is one of the greatest health crisis of today's world. HIV is a lentivirus that infects and by various mechanisms, kills vital cells of human immune system, such as T helper cells, macrophages, and dendritic cells, thereby, causing immunodeficiency [1]. Despite progress in therapeutic control, viral mutations continue to accumulate in the peripheral blood compartment over time, indicating either low level reactivation or replication [2].

Detoxification is a set of physiological and psychological processes, which assist the body in identifying, neutralizing, and eliminating harmful chemicals, metabolic waste, habits, and patterns. The liver is the human body's most essential organ for toxins' elimination, carried out throughout the day continuously, and it is responsible for processing a wide range of chemicals from the digestive system and rest of the body. It differentiate various substances, some of which are very toxic and others beneficial. The liver plays an important role in removing reactive oxygen species (ROS) by breaking down chemicals, which may produce ROS and antioxidant enzymes [3]. The liver is excellent in determining what should be preserved and what removed. It is like a massive chemical factory that creates molecules, eliminates harmful ones, and distributes particles throughout the body for usage, storage, or excretion. To fulfil its duty in eliminating harmful items, it employs phase 1 and phase 2 routes. Phase 1, as the component that breaks the particles down and transfers raw materials to phase 2, creates new compounds by combining molecules, called "conjugation". In phase 1, toxic chemicals and metals (from food, water, and air) are converted into less hazardous compounds by a series of chemical processes, which occur when a P-450 enzyme is activated. The conjugation route is the second phase in the process of waste removal, accomplished by dissolving fat-soluble harmful chemicals and converting the poison into water-soluble compounds. They are then drained out of the body through bodily fluids, such as bile or urine. These systems differ greatly amongst people, and are influenced by environment, lifestyle, and genetics. Poor detoxification was linked to illnesses, such as cancer, Parkinson's disease, fibromyalgia, and long-term immunological dysfunction syndrome. Data on these enzyme systems and how the body controls them, showed that the capacity to effectively remove and detoxify xenobiotics may have an impact on the above-mentioned long-term health issues.

A study reported that different persons and antiretroviral therapy (ART) combinations demonstrates various probabilities of developing liver damage [4]. A study conducted in Ethiopia indicated that 20.1% of highly active ART (HAART) patients and 22% of non-HAART participants had abnormal liver enzymes. Also, patients who were on HAART had a higher average level of alanine aminotransferase (ALT) than those not on HAART [5]. However, these findings are not consistent, as some did not observe the same magnitude or frequency of ART adverse effects. The reason for this difference may be due to geographical and genetic variations. Furthermore, the severity of HIV infection and individual susceptibility are usually influenced by a number of factors, including genetic variations and liver detoxification. It is generally accepted that viral replication is involved in the progression of HIV to fully developed AIDS. This occurs when HIV enters into the host cell and causes significant damage to cellular DNA that must be repaired immediately for the virus to replicate successfully. The aim of this research was to determine the impact of *OGG1* polymorphisms on some markers of liver function and antioxidants among HIV-infected patients.

Material and methods

Study area

The current study was conducted at Ladoke Akintola University of Technology Teaching Hospital (LAUTECH) Osogbo, Osun State, Nigeria.

Study design and population

This was a cross-sectional study with a total of 200 HIV-positive subjects (100 newly diagnosed patients and 100 on HAART) attending the outpatient clinic dedicated to people living with HIV (PLWH) at LAUTECH Teaching Hospital in Osogbo (newly named, UNIOSUN Teaching Hospital), and 100 HIV-negative subjects as controls. A structured questionnaire was administered to collect relevant information. Multi-stage random technique was employed in enrolling subjects for the research. All participants were informed about the investigation, and their consent was obtained.

Inclusion and exclusion criteria

HIV-positive male and female subjects on HAART, those who were not on HAART (naïve), and HIV-negative subjects as controls, were included in the study. Patients with history of dyslipidemia, diabetics, hypertension, and drug addiction, were excluded from the study.

Ethical approval

Ethical approval was obtained from the Ethical Review Committee of LAUTECH Teaching Hospital, Osogbo (approval No: LTH/EC/2020/01/444.). All participants provided informed consent to participate in the study.

Clinical and anthropometric measurement

Clinical and anthropometric measurements of each participant, including sex, age, body mass, height, and parameters of special interest, such as medical records, known systemic diseases, and treatment regimen, were collected using a standard questionnaire.

Sample size

With a prevalence of 1.4% reported by NAIIS [6], the minimum sample size was determined using a formula described by Araoye and Margaret [7]. Even though the minimum sample size was 21, 200 HIV-infected subjects were eventually recruited for the study.

Blood collection and laboratory analysis

Venous blood sample was collected from cubital fossa using 21-gauge needle and syringe, and 4 ml was dispensed into a plain bottle (non-anticoagulant bottle). The blood was allowed to clot and centrifuged at 1,200 rpm for 5 minutes to separate serum from cells. The serum sample was stored at –20°C for maximum three weeks before analysis. Then, 1 ml of the sample was mixed with DNA/RNA shield using a Zymo Quick-RNA Miniprep Kit (Zymo Research, USA). Blood samples for DNA extraction and restriction fragment length polymorphism (RFLP) analysis were stored at room temperature until further use.

OGG1 Ser326Cys polymorphism [8]

DNA was extracted from whole blood stabilized with DNA/RNA shield with a Zymo Quick-RNA Miniprep Kit (Zymo Research, USA), according to the manufacturer. Extracted DNA was quantified with UV spectrophotometer (purity and quantity of the extracted DNA were assessed by measuring OD values), and standardized to 10 ng/µl. Polymerase chain reaction (PCR) restriction fragment length polymorphism (RFLP) was used to determine *OGG1* SNP genotypes. A 234 bp PCR product was amplified using 6 pmol of primers (Inqaba Biotechnical Industries Ply Ltd.); *OGG1* forward: 5′-CCCAACCCCAGTGGATTCTCATTGC-3′, *OGG1* reverse: 5′-GTGCCCCATCTAGCCTTGCGGCCC-TT-3′). PCR amplification was carried out in 30 µl reaction volumes in separate PCR tubes. The reaction mixture contained 10 ng genomic DNA, 1× PCR buffer, 1.25 mM $MgCl_2$ 200 µM dNTP mix, 0.5 U Taq DNA polymerase, and DNase RNase nuclease-free water. Subsequently, the reaction mixture was subjected to 94°C for 5 minutes as initial denaturation, followed by 30 cycles at 94°C for 1 min, 55°C for 30 sec, and 72°C for 1 min, while the final extension step was carried out at 72°C for 5 minutes. Target gene segment was successfully amplified from *OGG1*, while the PCR product was resolved on 1.5% agarose gel after electrophoresis. Amplicons were visualized using Image J software. The PCR amplicon underwent restriction endonuclease digestion to determine the presence of polymorphic restriction site. The products were digested for 12 hours (37°C) using Fnu4HI restriction enzyme (Thermo Fisher Scientific) into two fragments, and the fragments were separated on 2% agarose gel containing ethidium bromide. Three possible genotypes were defined based on three distinct banding patterns observed through ultraviolet spectrophotometer: only 200 bp fragments were assigned as Ser/ Ser (CC) genotype, both 100 bp and 200 bp fragments were assigned as Ser/Cys (CG) genotype, while only 100 bp fragments were assigned as Cys/Cys (GG) genotype. The restriction fragments were visualized using Image J software.

ALT determination [9]

ALT catalyzes the transfer of amino group between l-alanine and l-glutamate; the corresponding α-keto acids in this process are α-ketoglutarate and pyruvate. Pyruvate is converted to red-brown hydrazone after reaction with 2.4-dinitrophenylhydrazine. The absorbance of the color produced was measured at 505 nm.

Aspartate aminotransferase determination [9]

Aspartate and 2-ketoglutarate catalyzes the transfer of amino group from aspartate to ketoglutarate, forming oxaloacetate reacts with 2.4-dinitrophenylhydrazine, which in alkaline pH are red-brown. The absorbance of the color produced was measured at 505 nm.

Serum albumin determination [10]

Albumin binds specifically to bromocresol green under an acidic condition to produce a blue-green color that was read at 630 nm. The intensity of the color is directly proportional to the concentration of albumin.

Total protein determination [11]

Cupric ions in an alkaline medium interact with protein peptide bonds, resulting in the formation of a colored complex.

Vitamin E [12]

The reaction is based on a reduction of ferric to ferrous ions by α-tocopherol, which then form a red-colored complex with 2,2'-dipyridyl that was read at 520 nm. Values were calculated as mg/dl.

Glutathione reductase [13]

The assay is based on the oxidation of NADPH to NADP+ catalyzed by a limiting concentration of glutathione reductase (GR). One GR activity unit is defined as the amount of enzyme catalyzing the reduction of one micromole of GSSG per minute at 7.6 pH and 25°C. One molecule of NADPH is consumed for each molecule of reduced GSSG. Therefore, the reduction of GSSG is determined indirectly by the measurement of NADPH consumption, as demonstrated by a decrease in absorbance at 340 nm (A_{340}) as a function of time.

Statistical analysis

Data were analyzed with Statistical Package for Social Science (SPSS) version 21 software. Simple descriptive statistical analysis was utilized to obtain percentages, mean, and standard deviation. Independent Student's *t*-test and analysis of variance (ANOVA) were employed to compare the means, while Pearson's correlation coefficient was used to correlate the measured variables between groups. *P*-value < 0.05 was considered statistically significant.

Results

A total of 300 subjects were included in the study: 123 males and 177 females, with a mean age of 41.24 ± 7.73 years. One hundred and forty-four samples were used for genotyping.

Table 1 shows demographic and clinical characteristics of study participants. There were no significant differences between genotypes CC and GG + CG in age, and diastolic and systolic blood pressures. The most frequently prescribed nucleoside reverse transcriptase inhibitors (NRTI) backbone was lamivudine-zidovudine used by 53.1% of the subjects. Tenofovir-lamivudine, abacavir-lamivudine, and didanosine-lamivudine were utilized by the remaining patients. The study participants were divided into three categories, including newly diagnosed (HIV-positive HAART-naïve), patients on HAART, and HIV-negative control subjects. There was no significant difference among all groups in terms of age, height, body mass index (BMI), and blood pressure distribution.

Table 2 shows that serum ALT ($p < 0.003$), aspartate aminotransferase (AST) ($p < 0.002$), albumin ($p < 0.001$), and total protein ($p < 0.001$), were significantly lower among HIV-positive subjects on HAART than HIV-positive HAART-naïve subjects and HIV-negative controls. Serum GR and vitamin E levels were significantly lower ($p < 0.001$) among HIV-positive HAART-naïve subjects compared with HIV-positive on HAART and control participants.

Table 3 indicates the correlation between measured liver function parameters and viral load among HIV-positive subjects. Serum ALT ($r = 0.453$, $p < 0.001$) and AST ($r = 0.300$, $p < 0.002$) correlated positively with viral load, while total protein and albumin did not significantly correlate with viral load.

Table 4 illustrates the comparison of measured parameters between HIV-positive subjects on HAART with viral load suppressed below 1,000 copies and those with viral load above 1,000 copies. Serum ALT activity was significantly lower ($p < 0.033$), while serum GR ($p < 0.001$) was significantly higher among patients with viral load suppressed below 1,000 copies compared with those whose viral load was above 1,000 copies. However, no significant differences were observed for AST, albumin, protein, and vitamin E ($p > 0.05$).

Table 5 demonstrates the stratification of markers of liver function and antioxidants according to *OGG1* SNPs among HIV-positive cases. Serum ALT ($p < 0.002$) and AST ($p < 0.001$) were significantly higher in patients with genotype CC than those with genotypes GG + GC. Moreover, GR

Table 1. Socio-demographic characteristics of study participants

Parameters	HIV-positive subjects on HAART (*n* = 100)	HIV-positive HAART-naïve subjects (*n* = 100)	HIV-negative control subjects (*n* = 100)	*p*-value
Systolic (mmHg)	113.70 ± 21.07	113.10 ± 17.51	111.80 ± 13.13	0.736
Diastolic (mmHg)	74.40 ± 13.73	73.10 ± 12.12	73.60 ± 9.48	0.739
Height (m)	1.61 ± 0.07	1.62 ± 0.09	1.62 ± 0.07	0.130
Weight (kg)	63.65 ± 11.31	59.84 ± 10.25	61.40 ± 11.41	0.050
BMI (kg/m^2)	23.79 ± 3.86	23.10 ± 4.23	23.64 ± 4.60	0.483

Data presented as mean ± standard deviation (SD).
HAART – HIV-positive subjects on antiretroviral drugs, naïve – HIV-positive subjects not on antiretroviral drugs

Table 2. Parameters of study subjects

Parameters	HAART (*n* = 100)	Naïve (*n* = 100)	Control (*n* = 100)	*F*-value	*p*-value
ALT (U/l)	8.71 ± 11.68	9.46 ± 10.08	3.32 ± 2.49	6.199	0.003
AST (U/l)	11.60 ± 13.14	13.11 ± 13.72	5.22 ± 3.01	6.416	0.002
Albumin (g/l)	38.61 ± 4.54	40.67 ± 3.56	42.02 ± 2.36	10.578	0.001
Protein (g/l)	83.33 ± 8.34	88.38 ± 8.64	66.93 ± 7.44	86.799	0.001
GR (mU/ml)	1.31 ± 0.50	0.49 ± 0.46	2.14 ± 0.70	130.248	0.001
Vitamin E (mg/dl)	9.09 ± 2.60	8.58 ± 2.61	31.27 ± 12.03	153.385	0.001

ALT – alanine aminotransferase, AST – aspartate aminotransferase, GR – glutathione reductase

($p < 0.008$) and vitamin E ($p < 0.01$) presented significantly lower levels among patients with genotype CC compared with those with genotypes GG + GC. No significant difference was observed for albumin ($p > 0.05$).

Discussion

HIV remains a persistent public health concern in sub-Saharan Africa. The World Health Organization (WHO) reported that more than 35 million patients died due to HIV infection in 2017. Sub-Saharan Africa is the most HIV-affected region globally, with an estimated 25.6 million PLWH. There has been a repeated call for the end of HIV pandemic. In 2014, the Joint United Nations Program on HIV/AIDS (UNAIDS) launched the 90-90-90 target to diagnose 90% of all HIV-positive persons, providing ART for 90% of those diagnosed, and achieving viral suppression in 90% of those treated by 2020. A new 95-95-95 target was set to be achieved by the end of 2030 [14]. Available evidence indicates that early placement of patients on treatment and achievement of viral load suppression reduce mortality and HIV transmission, and improve quality of life. However, while the access to HAART improved tremendously, virologic failure (when antiretroviral therapy fails to suppress and sustain in an infected person's viral load to less than 1,000 copies/ml) remains a common problem. Some authors showed that several factors are associated with virologic failures [15]. Previous studies highlighted numerous aspects, which may be linked to viral suppression, such as WHO clinical stage 4, sub-optimal adherence, poor tolerability, and drug-resistance [16]. However, there is limited information on the impact of *OGG1* SNPs in this regard.

Liver abnormalities can be related to multiple factors, such as co-infection with hepatitis viruses, genetics, opportunistic infections, etc. Moreover, abnormalities can result from direct inflammation in hepatocytes caused by the virus. Mechanisms, by which HIV causes hepatic damage, can occur through apoptosis and mitochondrial dysfunction. The level of liver cell injury is usually assessed by measuring the plasma of transaminase enzyme activities. It is obvious that the liver is the site of synthesis of thousands of enzymes; if there is any insult on the liver, these enzymes are released into the plasma, resulting in an increased activity. Serum ALT and AST are the most sensitive indicators of liver cell injury and used for the diagnosis of acute hepatocellular disease. The observed significantly higher ALT and AST activities might be due to both the viral infection and side effects of HAART. Findings from this study also revealed that severe form of liver enzyme elevation was not observed in any of the groups. However, different studies reported that HAART resulted in different degrees of liver enzyme elevation or hepatotoxicity (such as mild, moderate, and severe). Differences observed in this regard might be due to a number of factors, such as co-infection with hepatitis virus, alcohol ingestion, drugs' regimen, duration of treatment, presence

Table 3. Correlation between measured liver function test parameters and viral load among people living with HIV

Correlation	*r*-value	*p*-value
Viral load/ALT	0.453	0.001
Viral load/AST	0.300	0.001
Viral load/Albumin	0.020	0.845
Viral load/Protein	–0.098	0.333
ALT/AST	0.856	0.001
ALT/Protein	–0.222	0.027

ALT – alanine aminotransferase, AST – aspartate aminotransferase

Table 4. Liver detoxification and severity of HIV infection among HIV-positive patients on HAART

Parameters	Viral load		*p*-value
	< 1,000 (*n* = 85)	> 1,000 (*n* = 15)	
ALT (U/l)	7.39 ± 7.01	13.00 ± 17.38	0.033
AST (U/l)	10.62 ± 9.64	16.36 ± 17.41	0.067
Albumin (g/l)	38.67 ± 4.64	39.87 ± 4.36	0.355
Protein(g/l)	83.47 ± 8.26	83.87 ± 8.39	0.865
GR (mU/ml)	1.58 ± 0.34	0.63 ± 0.12	< 0.001
Vitamin E (mg/dl)	9.59 ± 2.50	8.39 ± 2.41	0.088

ALT – alanine aminotransferase, AST – aspartate aminotransferase, GR – glutathione reductase

Table 5. Stratification of some markers of liver function and antioxidants according to *OGG1* SNPs among HIV-positive subjects

Parameters	GG + CG genotypes (*n* = 45)	CC genotype (*n* = 54)	*p*-value
ALT (U/l)	5.01 ± 7.19	9.84 ± 11.04	0.002
AST (U/l)	6.24 ± 5.87	14.51 ± 14.75	0.001
Albumin (g/l)	40.37 ± 4.02	40.41 ± 3.71	0.950
GR (mU/ml)	1.46 ± .89	1.10 ± .70	0.008
Vitamin E (mg/dl)	18.50 ± 13.80	13.05 ± 10.40	0.010

Values are expressed in means ± standard deviation.
ALT – alanine aminotransferase, AST – aspartate aminotransferase, GR – glutathione reductase, GG + CG – Cys326Cys + Cys326Ser, CC – Ser326Ser

or absence of comorbid conditions, geographic condition, and genetic polymorphisms. The subjects of this study were neither smokers nor occupationally more exposed to pollutants than the controls. Socio-economic factors may be co-founders in this instance. The results of the current study are in line with Sulkowski *et al.* [17], who reported that HAART was associated with the elevation of liver enzyme activities. Contrary to our finding, Quaye *et al.* [18] showed a significantly lower GR activity among HIV-positive patients compared with sero-negative controls. GR is responsible for converting oxidized glutathione into the reduced form and, therefore, low GR activities will lead to elevated oxidized glutathione levels, which may result in an increased oxidative stress and abnormal liver detoxification processes [19]. Vitamin E appears to be among the first-line defenders against peroxidation of polyunsaturated fatty acids (PUFAs) in cellular and sub-cellular membrane phospholipids. It is a sacrificial antioxidant that can donate hydrogen atoms, and it is localized in membranes and lipoproteins, where it can interrupt the radical chain reaction of lipid peroxidation. Plasma level is a reliable indicator of antioxidant status, because it reflects bio-availability as well as increased utilization in counter lipid peroxidation. Additionally, in the present study, ALT activity was significantly higher among patients with severe infection compared with those with suppression of viral load, while a significantly lower level was noted in GR activity among the same group. The liver plays an important role in detoxification of ROS through metabolism of compounds, which potentially generate ROS. Although HAART was observed to influence liver damage [20], the significantly higher levels of ALT and lower levels of GR in HAART-naïve HIV-positive patients as compared with those on HAART in this study, suggest that viral replication rather than HAART may be the underlining cause of the observed higher activities of the enzymes. This finding is in line with a previous research [18].

In the current study, significantly higher serum ALT ($p < 0.002$) and AST ($p < 0.001$) activities as well as significantly lower GR ($p < 0.008$) and vitamin E ($p < 0.01$) were observed among individuals with genotype CC than in those with genotypes GG + GC. Even though evidence is lacking on the effect of SNPs on detoxification in HIV-positive patients, some authors reported that SNPs within *OGG1* was observed to predispose HIV-infected South African women to adverse effects of obesity. Subjects with the wild type (CC) genotype presented an increased susceptibility to HIV-associated low-birth weight and pulmonary tuberculosis [8]. Toxic insults in individuals may be dependent on their genetic sensitivity to HIV infection or other environmental toxicants. The genes of DNA repair pathway have been shown to be highly polymorphic, thus, affecting the structure and function of proteins. This polymorphism may affect an individual's response to external insults [8]. Such changes down-regulate the S326C *OGG1* catalytic action and its susceptibility in forming dimerization and disulfide bond in an oxidizing environment [21]. These might be responsible for the higher activity of AST and ALT as well as lower GR and vitamin E concentration. Subsequently, these 54 out of 99 (54.5%) HIV-positive individuals with CC genotype may be at increased risk of oxidative pathologies. A high death rate of 4.2% was reported among cancer patients with CC genotype compared with Ser326Cys carriers [14], since cells homozygous for Cys variants exhibit an increased genetic instability and declined in vivo 8-oxoguanine glycosylase repair rates.

Oxidative stress plays significant role in the progression and development of diseases, partly via the induction of DNA damage. The DNA damage caused by the generation of free radical under physiologic conditions is the highly mutagenic 8-dihydrodeoxyguanosine (8-oxoG), the persistence of which can lead to SNPs. A correlation between the levels of 8-oxoG DNA glycosylase activity in lymphocytes and cancer predisposition was demonstrated [23].

Conclusions

Findings from this study revealed that 54 out of 99 (54.5%) HIV-positive subjects on HAART presented CC genotype, while 45 out of 99 (45.5%) had GG + CG genotypes. Serum ALT and AST were significantly higher, while GR and vitamin E were significantly lower among patients with genotype CC than those with genotypes GG + GC. The changes also correlated with viral load. This may suggest that oxidative stress may be higher among individuals with genotype Ser/Ser (CC) than in those with genotypes Cys/Cys (GG) + Cys/Ser (GC).

Disclosures

1. Institutional review board statement: This research obtained ethics approval issued by the Ethical Review Committee of LAUTECH Teaching Hospital, Osogbo (approval number: LTH/EC/2020/01/444).
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3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

References

1. Ivanov AV, Valuev-Elliston VT, Ivanova ON, Kochetkov SN, Starodubova ES, Bartosch B, Isaguliants MG. Oxidative stress during HIV infection: mechanisms and consequences. Oxid Med Cell Longev 2016; 2016: 8910396. DOI: 10.1155/2016/8910396.
2. Dampier W, Nonnemacher MR, Mell J, Earl J, Ehrlich GD, Pirrone V, et al. HJIV-1 genetic variation resulting in the development of new quasispecies continues to be encountered in the peripheral blood of well-suppressed patients. PLoS One 2016; 11: e155382. DOI: 10.1371/journal.pone.0155382.
3. Lee CY, Park KS, Park HG. A fluorescent G-quadruplex probe for the assay of base excision repair enzyme activity. Chem Commun (Camb) 2015; 51: 13744-13747.
4. Dietlein F, Thelen L, Reinhardt HC. Cancer-specific defects in DNA repair pathways as targets for personalized therapeutic approaches. Trends Genet 2014; 30: 326-339.
5. Shiferaw MB, Tulu KT, Zegeye AM, Wubante AA. Liver enzymes abnormalities among highly active antiretroviral theraphy expe-

rienced and HAART naïve HIV-1 infected patients at Debre Tabor Hospital, North West Ethiopia: a comparative cross-sectional study. AIDS Res Treat 2016; 2016: 1985452. DOI: 10.1155/2016/1985452.
6. NAIIS. Nigeria HIV/AIDS indicator and impact survey. 2019.
7. Araoye MO. Sample size determination. Research methodology with statistics for health and social sciences. Ilorin: Nathadex Publishers; 2004, p. 115-118.
8. Anderson SM, Nandoo RN, Ramkaran P, Asharam K, Muttoo S, Chuturgoon AA. OGG1 Ser326Cys polymorphism, HIV, obesity and air pollution exposure influence adverse birth outcomes susceptibility within South African women. Reprod Toxicol 2018; 79: 8-15.
9. Reitman S, Frankel S. A colorimetric method for the determination of serum glutamic oxalacetic and glutamic pyruvic transaminases. Am J Clin Pathol 1957; 28: 56-63.
10. Harding JR, Keyser JW. Bromocresol green as a reagent for serum albumin. Proc Assoc Clin Biochem 1968; 5: 51.
11. Hicham T, IIyas E, Tarik H, Noureddine B, Omar B, Rachid F, Kingsley GR. The direct biuret method for the determination of serum proteins as applied to photoelectric and visual colorimetry. J Lab Clin Med 1942; 27: 840-845.
12. Baker H, Frank O. Clinical vitaminology. Methods and interpretation. Interscience Publishers; 1968.
13. Worthington DJ, Rosemeyer MA. Glutathione reductase from human erythrocytes: catalytic properties and aggregation. Eur J Biochem 1976; 67: 231-238.
14. Bain LE, Nkoke C, Noublap N. UNAIDS 90-90-90 targets to end the AIDS epidemics by 2020 are not realistic: comment on "Can the UNAIDS 90-90-90 target be achieved? A systematic analysis of national HIV treatment cascades". BMJ Glob Health 2017; 2: e000227. DOI: 10.1136/bmjgh-2016-000227.
15. Hicham T, Ilyas E, Tarik H, Noureddine B, Omar B, Rachid F, et al. Risk factors associated with unsuppressed viral load in HIV-1 infected patients at the first antiretroviral therapy in Morocco. Int J Mycobacteriol 2019; 8: 113-117.
16. Monkgomotsi JM, Leabaneng T, Prisca KT, Kaelo KS, Sikhulile M, Axel M, et al. Association of CYP2B6 genetic variation with efavirenz and nevirapine drug resistance in HIV-1 patients from Botswana. Pharmacogenomics Pers Med 2021; 14: 335-347.
17. Sulkowski MS, Thomas DL, Moore RD, Brinkley SC, Torbenson MS, Higgins YM, et al. Relationship of liver disease stage and antiviral therapy with liver-related events and death in adults co-infected with HIV/HCV. JAMA 2012; 308: 370-378.
18. Quaye O, Kuleape JA, Bonney EY, Puplampu P, Tagoe EA. Imbalance of antioxidant enzymes activities and trace elements levels in Ghanaian HIV-infected patients. PLoS One 2019; 14: e0220181. DOI: 10.1371/journal.pone.0220181.
19. Rajopadhye S, Mukherjee S, Chowdhary A. Oxidative stress in HIV/AIDS patients in Mumbai, India. J Immunol Virol 2015; 1: 53-59.
20. Mataranyika PA, Kibuule D, Kalemeera F, Kaura H, Godman B, Rennie WT. Liver enzyme elevations in a cohort of HIV/AIDS patients on first-line antiretroviral therapy in Namibia: findings and implications. Alexandria Journal of Medicine 2017; 54: 49-56.
21. Simonellia V, Camerinib S, Mazzeia F, Van Loon B, Allione A, D'Erricoa M, et al. Genotype–phenotype analysis of S326C OGG1 polymorphism: a risk factor for oxidative pathologies. Free Radic Biol Med 2013; 63: 401-409.
22. Corella D, Coltell O, Macian F, Ordovas JM. Advances in understanding the molecular basis of the mediterranean diet effect. Ann Rev Food Sci Technol 2018; 9: 227-249.
23. Bravard A, Vacher M, Moritz E, Vaslin L, Hall J, Epe B, Radicella PJ. Oxidation status of human OGG1-S326C polymorphic variant determines cellular DNA repair capacity. Cancer Res 2009; 69: 3642-3649.