

Possible Expression of Viral Seromarkers and Inflammatory Responses in Patients with Decreased Plasma Superoxide Dismutase

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Journal of Experimental Research

September 2020, Vol 8 No 3

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ABSTRACT

Expression of viral seromarkers indicate viral infection or immunity while plasma cytokines and Superoxide Dismutase (SOD) are biomarkers of pro/anti-inflammatory responses and anti-oxidative bioactivities respectively. This work was designed to determine possible expression of viral seromarkers and inflammatory responses in patients with decreased plasma Superoxide dismutase. Sixty-three (63) patients (Male-33; Female-30) aged 27-71 years, with decreased SOD, normal blood glucose who were negative to AFB test and Giemsa thick blood film technique for *Plasmodium* were studied as test subjects. Whereas seventy individuals (Male-35; Female-35; Age- 27 – 71 years) with normal SOD, normal blood glucose, and who were negative to AFB test, Giemsa thick blood film technique for *Plasmodium* and also did not express seromarkers of HIV, HBV and HCV were studied as control subjects. Identification of *Plasmodium* and AFB and estimation of blood glucose, SOD, plasma TNF α and IL-10 were all done by standard operative procedures. The results in patients with decreased plasma Superoxide dismutase showed a frequency of viral seromarkers of 11.1% (7) Anti-HCV; 20.6% (13) Anti-HBe; 20.6% (13) HBeAg; 20.6% (13) HBsAg; 3.2% (2) HIVAg(p24)/Ab; 0% (0) Anti-HCV + HIVAg/Ab; 3.2% (2) HBsAg + HBeAg + HIVAg/Ab and 20.6% (13) HBeAg + HBsAg + anti-HBe. Seromarker of hepatitis B virus was found to be more prevalent while 3.2% (2) of the patients expressed HBsAg + HBeAg + HIVAg/Ab. The frequency of viral seromarkers of hepatitis B virus were more than the seromarkers of hepatitis C virus and Human Immunodeficiency Virus while seromarkers of both HBV and HCV were more than those of HIV. Only coinfection of HIV and HBV [3.2% (2)] was found in the patients expressed as anti-HBe, HBsAg + HBeAg + HIVAg/Ab. There was also a significant increase in plasma TNF α and a significant decrease in IL-10 in patients who expressed seromarkers of HBV, HCV and HIV which was more pronounced in patients who expressed HIV seromarkers and also in those who co-expressed HIV and HBV seromarkers ($p < 0.05$). There was a significant decrease in the plasma SOD in patients who expressed Anti-HCV, anti-HBe, HBeAg, HBsAg, HIVAg, Ab, HBsAg+HBeAg+HIV and HBeAg+HBsAg+ anti-HBe compared with the results obtained in the control ($p < 0.05$). Patients with decreased SOD expressed viral seromarkers including significant inflammatory response indicated by an increase in TNF α and decreased IL-10 which generally signify evidence of immune or inflammatory responses as well as evidence of active viral infection. Infection Prevention and Control (IPC) is therefore essential to prevent decrease in blood Superoxide Dismutase (SOD) which is an important antioxidant that protects cells from oxidative damage.

Keywords: Viral seromarkers, TNF α , IL-10, Superoxide dismutase

INTRODUCTION

Superoxide dismutase is an important antioxidant that protects cell exposed to reactive oxygen from cellular damage (McCord and Fridovich, 1988; Hayyan et al. 2016). It is an enzyme involved in the dismutation/partitioning of superoxide (O_2^-) radical into either ordinary molecular oxygen (O_2) or hydrogen peroxide (H_2O_2). Superoxide is produced from the metabolism of oxygen and, if not controlled can cause cell damage. Hydrogen peroxide which can also cause cellular damage. It can be degraded by other

enzymes such as catalase (McCord and Fridovich, 1988; Hayyan et al. 2016).

Superoxide dismutase acts as antioxidant to protect cell damage by converting the reactive oxygen (O_2^-) species to less damaging species. In other words Superoxide dismutase dismutates superoxide anion radical (O_2^-) to O_2 and hydrogen peroxide (H_2O_2) to prevent cell damage (McCord and Fridovich, 1988; Hayyan et al. 2016). Cytokines are substances that play important roles in pro and anti-inflammatory immune responses (Cannon, 2000; Locksley et al. 2001). They are involved in humoral and cell

mediated immune responses (Swardfager et al. 2010). Tumor Necrosis factor- α (TNF- α) is a pro-inflammatory cytokine that regulates immune cells (Cannon, 2000; Locksley et al. 2001).

This cytokine is an endogenous pyrogen that can bring about fever, apoptotic cell death, cachexia and inflammation. It can also inhibit tumorigenesis and viral replication (Cannon, 2000; Locksley et al. 2001). Tumor Necrosis factor - α (TNF- α) can act on macrophages to stimulate phagocytosis and liver to generate acute phase response (Cannon, 2000; Locksley et al. 2001). Interleukin 10 (IL-10) or human cytokine synthesis inhibitory factor (CSIF) (Brennan et al. 2008; Mosser and Zhang, 2008) on the other hand, is an anti-inflammatory cytokine that can inhibit the synthesis of pro-inflammatory cytokines such as IFN- γ , IL-2, IL-3, TNF α and GM-CSF. The cytokine (IL-10) regulates the enzyme responsible for the conversion of TNF- α (Brennan et al. 2008; Mosser and Zhang, 2008).

Viral Seromarkers are expressed or characterized in the serum or plasma as a result of viral infection (Lee et al. 2010; Olowe et al. 2016; Prabina et al. 2019). Viral infection stimulates immune response leading to the production of antibody specific to the invading virus (antigen) (Lee et al. 2010; Olowe et al. 2016; Prabina et al. 2019). This immune product can which are always in the serum are detected or characterized as viral seromarkers to indicate evidence of viral infection (Lee et al. 2010; Olowe et al. 2016; Prabina et al. 2019). They are expressed in the serum or plasma after seroconversion (Lee et al. 2010; Olowe et al. 2016; Prabina et al. 2019). Viral seromarkers include anti-HCV for Hepatitis C virus infection, HBsAg for Hepatitis B virus infection, anti-HBe indication of HBV infection and clearance of HBeAg; HBeAg indicating replication and active infection of HBV and anti-HIV/P24 antigen indicating HIV infection (Lee et al. 2010; Olowe et al. 2016; Prabina et al. 2019).

This study was designed to determine viral seromarkers (HIVp24 antigen and antibodies to HIV1/HIV2, anti-HCV, HBsAg, HBeAg, and anti-HBe) and inflammatory responses in patients with decreased plasma Superoxide dismutase.

MATERIALS AND METHODS

Study area

This work was carried out in Owo, Ondo State –Nigeria. Owo is a major city in Ondo State-Nigeria. Owo was the capital of a Yoruba city-state between 1400 and 1600 AD. Owo hosts Federal Medical Centre, Achievers University, Rufus Giwa Polytechnic, Secondary and Primary Schools. It is the headquarters of Owo local government area in Owo/Ose Federal constituency of Nigeria.

Study Population

Sixty three (63) patients (Male-33; Female-30); aged: 27-71 years, with decreased plasma super oxide dismutase (SOD) who were negative to Acid Fast Bacilli and Thick-Giemsa Film blood technique (for the detection of *Plasmodium*) were recruited as test volunteers while Seventy (70) volunteers (Male-35; Female-35); aged 27-71 years, with normal super oxide dismutase (SOD) who were negative to Acid Fast Bacilli and Thick-Giemsa Film blood technique (for the detection of *Plasmodium*) were studied as control subjects.

The study was approved by Ethical and Research committee of Federal Medical Centre, Owo -Nigeria (FMC/AUOS//19/0199) and informed consent of each of the participants was also obtained.

Inclusion Criteria:

Subjects were included in the study if they were:

Volunteers with decreased plasma SOD and normal blood glucose were included as test subjects or volunteers with normal SOD and blood glucose were included as control subjects

Exclusion criteria:

Subjects were excluded from the study if they were positive to Acid Fast Bacilli and Thick-Giemsa Film blood technique (for the detection of *Plasmodium*) with abnormal blood glucose level were excluded from the study. Malaria and tuberculosis are more prevalent in the area according to the reports of Dada et al.(2016) that overall prevalence percentage of malaria was found to be high in Ondo State Hospital Akure (20.7%). Adeniyi et al. (2017) reported that the most prevalent five respiratory

diseases were tuberculosis (TB), pneumonias, chronic obstructive pulmonary diseases (COPD), asthma and lung cancer (53%, 21.1%, 13.7%, 8.4% and 1.4% respectively).

Specimen Collection and preparation:

Five milliliters of venous blood was obtained from each of the subjects into lithium heparinized bottles. The Plasma was extracted for biochemical and ELISA assays while the whole blood was used for the identification of *Plasmodium*. Sputum samples was obtained from each of the subjects consecutively for three days for Ziehl Neelsen staining.

Analytical Methods:

Superoxide Dismutase (S.O.D) Activity was determined using Abcam kit (ab65354).

HIVp24 antigen and antibodies to HIV-1 (groups M and O) and HIV-2 in human serum

These were determined in the subjects using Bio-Rad Genscreen™ ULTRA HIV Ag-Ab qualitative enzyme immunoassay kit (72278 883666) designed for the detection of HIV p24 antigen and antibodies to HIV-1 and HIV-2 in human serum/plasma by Enzyme Linked Immunosorbent Assay(ELISA). Manufacturer's instructions were strictly followed and applied.

Detection of Anti-HCV

Anti-HCV was determined in the subjects using Bio-Rad Monolisa™ Anti-HCV PLUS Version 3 screening kit(72315 883663) designed for the detection of anti-HCV antibodies (Hepatitis C Virus) in human plasma or serum by ELISA technique. Manufacturer's instructions were strictly followed and applied.

Detection of HBsAg, HBeAg, HBeAb by ELISA

Hepatitis B surface antigen (HBsAg) was determined by a one step MONOLISA AgHBs PLUS enzyme immunoassay technique of the sandwich type (72408 883665_2013_12) for the detection of HBsAg in serum or plasma using the reagent kit of BIO –RAD Raymond Poincare, Marnes La Coquette.

HBeAg and anti-HBe tests were done by immunoassay technique based on ELISA sera

principle for the detection of Hepatitis B e antigen(HBeAg) and antibody(Anti-HBe) in human plasma and sera using the reagent kit (0318) of: DIA. PRO Diagnostic BioprobesSrlVia Columella, Milano – Italy. Manufacturer's instructions were strictly followed and applied.

Measurement of Blood glucose and cytokines concentrations

Blood glucose was determined in the subjects by glucose oxidase method using the reagent kit of RANDOX (GL364). Manufacturer's instructions were strictly followed and applied.

TNF- α level was determined with ELISA method using Human ABCAM ELISA Kit (ab181421) whereas IL-10 was also assayed by ELISA method using Human IL-10 ELISA Kit (ab46034) strictly following manufacturers instructions.

Detection of Acid Fast Bacilli (AFB) in sputum and Identification of *Plasmodium* in blood

Acid Fast Bacilli test and Identification of *Plasmodium* using sputum and blood samples respectively were carried out by the method described by Cheesbrough, (2006).

Data analysis

The results obtained was subjected to statistical analysis to determine mean, standard deviation student 'T' test and probability values at 0.05 level of significance using SPSS IBM20.0. The frequency of viral seromarkers, mean \pm standard deviation of the biochemical and immunological parameters, 'T'and probability values and level of significance at 0.05 were presented under results in tables and figure.

RESULTS

The frequency of viral seromarkers obtained in this study include 11.1%(7) Anti-HCV; 20.6%(13) Anti-HBe ; 20.6%(13) HBeAg ; 20.6%(13) HBsAg ; 3.2% (2) HIVag(p24)/Ab ; 0%(0) Anti-HCV + HIVag/Ab ; 3.2% (2) HBsAg + HBeAg + HIVag/Ab and 20.6%(13) HBeAg + HBsAg + anti-HBe. Seromarker of hepatitis B virus was found to be more prevalent. 3.2% (2) of the

patients expressed HBsAg + HBeAg + HIVAg/Ab (Table 1; Figure 1).

Table 1: Frequency of viral seromarkers, mean and standard deviation of plasma TNF-α, IL-10 SOD and glucose obtained in the patients.

Viral Seromarkers	Frequency	TNF-α (pg/ml)	IL-10 (pg/ml)	Plasma Glucose (mg/dl)	Plasma SOD, (U/mL)	AFB test	Plasmodium test
Anti-HCV	11.1 %(7)	4.3 ± 0.1	2.7 ± 0.1	101 ± 3.0	53 ± 1.0	Negative	Negative
Anti-HBe	20.6%(13)	4.0 ± 0.2	2.9 ± 0.2	98 ± 2.0	55 ± 2.0	Negative	Negative
HBeAg	20.6%(13)	4.5 ± 0.1	2.5 ± 0.1	99 ± 3.0	55 ± 1.0	Negative	Negative
HBsAg	20.6%(13)	4.3 ± 0.1	2.7 ± 0.2	98 ± 2.0	54 ± 1.0	Negative	Negative
HIVAg(p24)/Ab	3.2% (2)	5.3 ± 0.1	2.2 ± 0.15	100 ± 2.0	52 ± 2.0	Negative	Negative
Anti-HCV + HIVAg/Ab	0	0	0	0	0	-	-
HBeAg + HBeAg + HIVAg/Ab	3.2% (2)	7.3 ± 0.2	2.0 ± 0.1	98 ± 2.1	51 ± 1.0	Negative	Negative
HBeAg + HBsAg + anti - HBe	20.6%(13)	4.3 ± 0.1	2.7 ± 0.1	99 ± 1.5	55 ± 1.5	Negative	Negative
Control	70	2.3 ± 0.1	4.7 ± 0.1	99 ± 2.0	98 ± 1.3	Negative	Negative

Note: Anti-HCV - antibody to HCV
 Anti-HBe – Envelope Antibody to HBV
 HBeAg- Envelope Antigen to HBV
 HBsAg- HBV surface antigen

HIVAg(p24)/Ab- HIV antigen and antibody
 TNF-α (pg/ml)- Tumor necrosis factor alpha
 IL-10 (pg/ml)- Interleukin 10

The results obtained in patients with decreased plasma Superoxide dismutase showed that the frequency of viral seromarkers of hepatitis B virus were more than the seromarkers of hepatitis C virus and Human Immunodeficiency Virus while seromarkers of both HBV and HCV were more than those of HIV. Only coinfection of HIV and HBV (3.2% (2)) was found in the patients expressed as anti-HBe, HBsAg + HBeAg + HIVAg/Ab (Table 1; Figure 1). There was no significant difference in the plasma value of TNF-α (pg/ml) and IL-10 (pg/ml) in the results obtained in patients who

expressed HBeAg + HBsAg + anti-HBe and Anti-HCV (p>0.05; Table 1,2; Figure 1)

There was a significantly higher plasma value of TNF-α (pg/ml) in patients who expressed HIVAg(p24)/Ab than in patients who expressed HBeAg + HBsAg + anti-HBe (p<0.05; Table 1, 2; Figure 2). However there was no significant difference in the plasma value of IL-10 (pg/ml) in patients who expressed HIVAg(p24)/Ab and HBeAg + HBsAg + anti-HBe (p>0.05; Table 1,2; Figure 1).

Table 2 : Comparative analysis of TNF- α and IL-10 obtained in the patients

	HBeAg + HBsAg + anti-HBe Vs. Anti-HCV	HBeAg + HBsAg + anti-HBe Vs. HIVAg(p24)/Ab	HBeAg + HBsAg + anti-HBe Vs. HBsAg + HBeAg + HIVAg/Ab	HIVAg(p24)/Ab Vs. Anti-HCV
TNF-α (pg/ml)	T-value 0	-7.07107.	-13.41641.	7.07107.
	P-value 0.5	0.00971*	0.002755*.	0.00971*
IL-10 (pg/ml)	T-value 0	2.23607.	4.94975.	-2.23607.
	P-value 0.5	0.077423.	0.019238*	0.077423.

Note: *Significant (p<0.05)

There was a significantly higher TNF- α and lower plasma IL-10 in patients who expressed HBsAg + HBeAg + HIVAg/Ab than those who

expressed HBeAg + HBsAg + anti-HBe (p<0.05; Table 1,2; Figure 1).

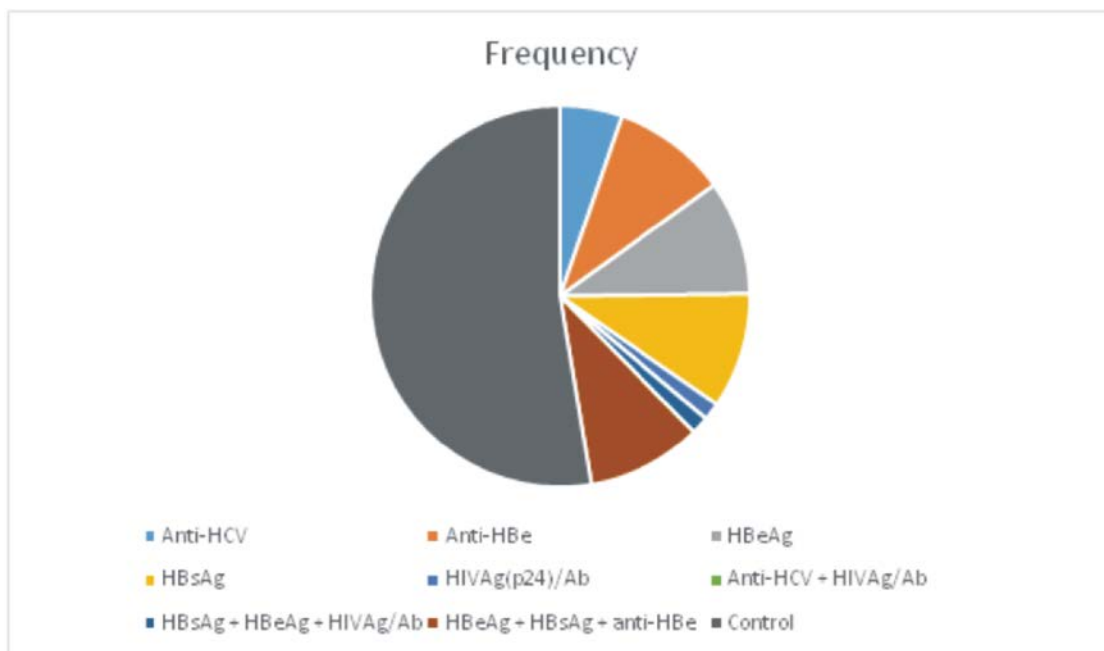


Figure 1: Comparative description of viral seromarkers, plasma TNF- α and IL-10 obtained in the patients and control

There was a significantly higher TNF- α in patients who expressed HIVAg(p24)/Ab than those who expressed Anti-HCV in their plasma (p<0.05; Table 1,2; Figure 2). However there was no significant difference in the plasma value of IL-10 (pg/ml) in patients who expressed HIVAg(p24)/Ab and Anti-HCV (p>0.05; Table 1,2; Figure 2).

There was a significantly higher TNF- α and lower plasma IL-10 in patients who expressed HBeAg + HBsAg + anti-HBe, HIVAg(p24)/Ab, HBsAg + HBeAg +

HIVAg/Ab, Anti-HCV than the Control subjects (p>0.05; Table 1, 3; Figure 1).

There was a significantly higher TNF- α and lower plasma IL-10 in patients who expressed HBeAg + HBsAg + anti-HBe, HIVAg(p24)/Ab, HBsAg + HBeAg + HIVAg/Ab, Anti-HCV than the Control subjects (p<0.05; Table 1,2; Figure 2) except that no significant difference was obtained in plasma IL-10 in patients with HBeAg + HBsAg + anti-HBe and Control (p>0.05; Table 1,3; Figure 1).

Table 3: Comparative analysis of TNF- α and IL-10 obtained in the patients and control

	HBeAg + HBsAg + anti-HBe Vs. Control	HIVag(p24)/Ab Vs. Control	HBsAg + HIVAg/Ab Vs. control	Anti-HCVVs. Control
TNF-α (pg/ml)	T-value 14.14214. P-value 0.002481*	21.2132. 0.001107*.	22.36068. 0.000997*	14.14214. 0.002481*
IL-10 (pg/ml)	T-value 2.23607. P-value 0.077423.	-11.18034 0.003953*.	-19.53874. 0.001305*	-14.14214. 0.002481*

There was a significant increase in plasma TNF α and a significant decrease in IL-10 in patients who expressed seromarkers of HBV, HCV and HIV which was more pronounced in patients who expressed HIV seromarkers and also in those who co-expressed HIV and HBV seromarkers (p<0.05). There was a significant

decrease in the plasma SOD in patients who expressed Anti-HCV, anti-HBe, HBeAg, HBsAg, HIVAg-Ab, HBsAg + HBeAg + HIV and HBeAg + HBsAg + anti-HBe compared with the results obtained in the control (p<0.05; Table 1,3, 4; Figure 1)

Table 4: Comparative analysis of plasma SOD obtained in test and controls

	Anti-HCV Vs Control	Anti-HBe Vs Control	HBeAg Vs Control	HBsAg Vs Control	HBeAg-Ab Vs Control	HBeAg-HBeAg+IHV Vs Control	HBeAg-HBsAg+antiHBe Vs Control
Plasma SOD, (U/mL)	T-value -31.82 P-value 0.0005***	-19.23 0.001**	-38.91 0.0003***	-31.11 0.0005***	-20.57 0.001**	-33.23 0.0005***	-19.23 0.001**

Highly Significant at p<0.05; *More Highly Significant at p<0.05

DISCUSSION

The frequency of viral seromarkers obtained in this study include 11.1%(7) Anti-HCV; 20.6%(13) Anti-HBe; 20.6%(13) HBeAg ; 20.6%(13) HBsAg; 3.2% (2) HIVag(p24)/Ab; 0%(0) Anti-HCV + HIVAg/Ab; 3.2% (2) HBsAg + HBeAg + HIVAg/Ab and 20.6%(13) HBeAg + HBsAg + anti-HBe. Seromarker of hepatitis B virus was found to be more prevalent. 3.2% (2) of the patients expressed HBsAg + HBeAg + HIVAg/Ab.

The results obtained in patients with decreased plasma Superoxide dismutase showed that the frequency of viral seromarkers of hepatitis B virus were more than the seromarkers of hepatitis C virus and Human Immunodeficiency Virus while seromarkers of

both HBV and HCV were more than those of HIV. Only coinfection of HIV and HBV (3.2% (2)) was found in the patients expressed as anti-HBe, HBsAg + HBeAg + HIVAg/Ab. There was a significant increase in plasma TNF α and a significant decrease in IL-10 in patients who expressed seromarkers of HBV, HCV and HIV which was more pronounced in patients who expressed HIV seromarkers and also in those who co-expressed HIV and HBV seromarkers.

Expression of viral seromarker is an indication of active viral infection or immunity. It may also indicate viral replication acute or chronic viral infections. Expression of more than one viral seromarkers is an indication of coinfection with others virus (Hosein et al. 1991; Mel et al. 2005; Adeniyi et

al. 2017).

In hepatitis B virus infection HBsAg is produced in the liver in excess by the virus and secreted into the blood, where it serves as a marker for active infection and infectivity (Mel et al. 2005). Expression of HBeAg in the plasma of the patients can be associated with high levels of HBV replication, greater infectivity and an increased risk of hepatic fibrosis (Mel et al. 2005). Presence of anti-HBe in plasma or serum indicates HBV infection, clearance or decreased plasma level of HBeAg in the serum, but this may not alter the sequelae of chronic infection (Adeniyi et al. 2017). Generally, hepatitis B e-antigen (HBeAg) is expressed in the serum of patients with a new acute infection which is associated with higher HBV DNA levels an indication of increased infectiousness.

Antibody to HCV (Anti-HCV) in serum is an indication of HCV infection. The antibody is produced upon HCV infection and appear in the serum after seroconversion (Hosein et al. 1991). Antibody to HIV is a non-neutralizing/non-protective antibody expressed in HIV infection and expressed in the serum after seroconversion. HIVp24 antigen is more reliable antigen for the diagnosis of HIV infection. Presence of HIVp24 antigen and anti-HIV indicates HIV infection. HIVp24 antigen is detectable earlier than antibodies to HIV (Bystryak and Chitrangada, 2016).

Expression of viral seromarkers in patients with decreased SOD can be associated with the fact that viral infection can bring about oxidative stress in the affected individuals (Schwarz, 1996; Romá-Mateo et al. 2005; Segal, 2005; Halliwell, 2007; Valko et al. 2007; Hwang, 2013; Joseph et al. 2015). Oxidative stress is an imbalance between the systemic manifestation of reactive oxygen species and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage (Schwarz, 1996; Romá-Mateo et al. 2005; Segal, 2005; Halliwell, 2007; Valko et al. 2007; Hwang, 2013; Joseph et al. 2015). This is due to the production of toxic peroxides and free radicals that can damage cell, and its components. The damage is usually caused by reactive oxygen species (ROS), e.g. O₂⁻ (superoxide radical), OH (hydroxyl radical)

and H₂O₂ (hydrogen peroxide) (Schwarz, 1996; Romá-Mateo et al. 2005; Segal, 2005; Halliwell, 2007; Valko et al. 2007; Hwang, 2013; Joseph et al. 2015). However, reactive oxygen species are also beneficial, as they are used by the immune system to attack and kill pathogens (Schwarz, 1996; Romá-Mateo et al. 2005; Segal, 2005; Halliwell, 2007; Valko et al. 2007; Hwang, 2013; Joseph et al. 2015).

Decreased plasma level of SOD in the patients can be explained by the activities of antioxidants like SOD to inhibit oxidation which is a chemical reaction that can produce free radicals that can damage the cells of patients (Kalra et al. 1994; Ezimah et al. 2005). There was a significant decrease in the plasma SOD in patients who expressed Anti-HCV, anti-HBe, HBeAg, HBsAg, HIVAg-Ab, HBsAg+HBeAg+HIV and HBeAg+HBsAg+anti-HBe compared with the results obtained in the control. Excessive utilization of SOD as antioxidant against oxidation induced by viral infection may be responsible for the reduction in the plasma level of SOD. In another way, low plasma SOD an antioxidant that protects the body against free radical toxicity may be responsible for the patients' susceptibility to viral infections (Schwarz 1996; Valko et al. 2007) as indicated by the expression of Anti-HCV; Anti-HBe; HBeAg; HBsAg; HIVAg(p24)/Ab; HBsAg + HBeAg + HIVAg/Ab and HBeAg + HBsAg + anti-HBe. in this study. Low SOD is also a form of immune response in viral infection (Kalra et al. 1994; Beck, 2001; Ezimah et al. 2005).

Increased TNF α and reduced plasma IL-10 in patients who expressed viral seromarkers in this study indicates pro and anti-inflammatory responses (Beck, 2001; Seo and Robert, 2002; Kim and Solomon, 2010; Shi et al. 2013). Increased TNF α can be due to its pro-inflammatory bioactivities in viral infection leading to the excessive production to act on liver for acute phase response, phagocytosis and for inhibition of viral replication (Beck, 2001; Seo and Robert, 2002; Kim and Solomon, 2010; Shi et al. 2013).

Reduced plasma IL-10 in this work can be due to the anti-inflammatory activities of the cytokine as IL-10 can inhibit pro-inflammatory cytokines such as TNF α . IL-10 has been reported to suppress cytokine et al.

secretion, antigen presentation and CD4⁺ T cell activation (de Waal et al.1991; Aste-Amezaga et al.1998; Akdis et al. 2000; Joss et al. 2000).

CONCLUSION

Patients with decreased SOD expressed viral seromarkers including significant inflammatory response indicated by an increase in TNF α and decreased IL-10. There was also a higher frequency of viral seromarkers of hepatitis B virus than the seromarkers of hepatitis C virus and Human Immunodeficiency Virus while seromarkers of both HBV and HCV were more than those of HIV. Only coinfection of HIV and HBV (3.2% (2)) was found in the patients expressed as anti-HBe, HBsAg + HBeAg + HIVAg/Ab. Effective Infection Prevention and Control (IPC) is therefore essential to prevent decrease in blood Superoxide Dismutase (SOD) which is an important antioxidant that protects cells from oxidative damage.

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