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Blood innate immune factors as indicators of cardiac function in COVID-19 patients

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ABSTRACT

Pre-existing cardiovascular diseases (CVDs) aggravate the severity of COVID-19 illness. Also, COVID-19 patients present clinical symptoms of CVDs, but clear physiological causes of this association have not been fully elucidated. The present study explained COVID-CVD association by determining the status of plasma innate humoral immune factors [nitric oxide (NO), myeloperoxidase (MPO), hs-CRP, lactate dehydrogenase (LDH), interferon-gamma (IFN-γ) and interleukin 4 (IL-4)] and innate immune cell (total white blood cell count and percentages of neutrophil, monocyte and eosinophil) in SARS-CoV 2 infected patients at admission and at discharge using spectrophotometry, ELISA and haematology autoanalyser as appropriate. A total of 85 newly admitted COVID-19 patients who attended the Infectious Disease Centre, Ibadan, Nigeria were followed up till discharge in this study. SARS-CoV-2 confirmatory testing was done with RT-PCR molecular test as described by World Health Organisation. The mean WBC count and neutrophil percentage were significantly reduced, while monocyte percentage was significantly increased in COVID-19 patients at admission compared with COVID-19 patients at discharge. The mean plasma nitric oxide, hs C-RP, IFN-γ and IL-4 were significantly increased, while MPO was significantly reduced in COVID-19 patients at admission compared with COVID-19 patients at discharge ($p \leq 0.05$). The reduction of cardiac function inflammation factors (monocyte percentage, plasma levels of nitric oxide, hs C-RP, MPO and IFN-γ) in COVID-19 patients at discharge might predict future non-occurrence of CVDs in treated Nigerian COVID-19 patients.

Keywords: *Heart diseases, SARS-CoV-2 infection, Inflammation, Innate immunity.*

Introduction

Although COVID-19 is primarily a respiratory disease, it has been associated with many cardiac complications such as ischemic heart disease, heart failure, arrhythmias, and myocarditis [1, 2]. The United States Department of Veterans Affairs database demonstrated the occurrence of cerebrovascular disorders, arrhythmia, ischemic heart disease, heart failure, and thrombotic disorders after COVID-19 infection [3]. The exact mechanisms of SARS-CoV-2 involvement in cardiac disorders is unclear, but probably includes SARS-CoV-2 attachment to ubiquitous ACE-2 receptors, impairment of physiologic reserve, inducement of endothelial dysfunction, raising cytokine storm and direct heart damage. The spike proteins facilitate entry of SARS-CoV-2 into host cells by binding to angiotensin-converting enzyme 2 (ACE-2) receptors, which are also present on cardiac cells [4]. As SARS-CoV-2 replicates, it enters cardiac cells to cause direct injury [1-3]. It was reported that pre-existing cardiovascular disease activates chronic inflammation pathways, which synergises COVID-19-induced inflammation [5]. The overwhelming systemic inflammation (the cytokine storm) during SARS-CoV-2 infection results in increased oxygen demand, hypoxia, coagulopathy, and arrhythmias [1]. This is of great concern in COVID-19 patients with underlying cardiovascular disease, who may have little physiologic reserve for this stress.

Pre-existing inflammation and superimposed endothelial dysfunction accelerate coronary plaque instability and cardiovascular complications in patients with COVID-19 illness [5]. Another study demonstrated that interruption in lipid synthesis due to SARS-CoV-2 replication contributed to COVID-19-related cardiopulmonary pathology. Higher levels of di- and tri-glycerides plus free fatty acids were identified in the severe COVID-19 patients [6]. Furthermore, studies reported increased viral replication in cells having excessive intracellular lipid accumulation [7]. Patients infected with SARS-CoV had dysregulated levels of serum free fatty acids and altered lipid profile, even 12 years after recovery from the disease [8]. COVID-19 patients experienced pro-inflammatory cytokine storm, oxidative stress and hypoxia [9]. All these phenomena are common to patients with CVDs [10].

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Different heart diseases, including coronary heart disease (CHD), atherosclerotic heart disease and CHF, are associated with increased levels of proinflammatory cytokines such as interferon-γ (IFN-γ), IL-1β, IL-6 and TNF-α [10]. These cytokines contribute to the formation of atherosclerotic plaque. A high number of white blood cells and their subtypes (for example, neutrophils, monocytes, lymphocytes, and eosinophils) are associated with coronary heart disease, peripheral arterial disease, and stroke [11]. Myeloperoxidase produced by leucocytes plays a critical role in innate immunity, promotes atherosclerosis, contributes to the instability of atherosclerotic plaques, impairs endothelial function and contributes to oxidative stress. Furthermore, MPO utilises nitric oxide as a physiological substrate, potentially leading to endothelial dysfunction [12]. Nitric oxide (NO) plays an important and diverse signalling role in the cardiovascular system, regulates vascular tone, endothelial function, myocardial function and thrombosis [13].

Oxidative stress, multi-organ damage, cytokine storm and haematological imbalance are features of COVID-19 [9]. Having observed that these physiological outcomes of COVID-19 are risk factors for CVDs, it is therefore pertinent to determine certain immune-based blood parameters relating to cardiac functions in COVID-19 patients. The outcome will assist to predict occurrence of CVDs post-COVID-19 or elucidate the basis of severe COVID-19 episode in people with pre-existing heart diseases before SARS-COV-2 infection.

MATERIALS AND METHODS

Before the commencement of the study, the study was approved (UI/EC/20/0283) by the University of Ibadan/University College Hospital (UI/UCH) Joint Ethics Review Committee. Also, informed consent was adequately obtained. A total of 85 confirmed cases of COVID-19 were recruited from an Infectious Disease Centre, Olodo, Ibadan, Nigeria, in this longitudinal study. Ten (10) ml of blood sample was collected in lithium heparin bottle from each patient at admission and at discharge after confirmation of the absence of SARS-CoV-2 virus in the nasopharyngeal swabs. Two (2) ml whole blood from this 10 ml blood was analysed for haematological parameters (total White Blood Cell, monocyte, neutrophil, and eosinophil) using haematology autoanalyser (Sysmex XN-450). The participants did not have hypertension, diabetes mellitus, sickle cell anaemia, cardiovascular disease, cerebrovascular disease, cancer, chronic renal disease or inflammatory conditions. Plasma was removed from the remaining 8ml whole blood by centrifugation at 1500 ×g for 10 minutes. Myeloperoxidase (MPO) activity and NO were determined spectrophotometrically, while LDH, IFN-γ and IL-4 were measured using ELISA as described by the manufacturer (Fortress Diagnostics Ltd, UK). Data obtained were presented as mean and Standard deviation. Independent Student t-test was used to compare the mean values. The p-value ≤ 0.05 was considered as statistically significant.

RESULTS

As shown in table 1, the mean WBC count and neutrophil percentage were significantly reduced while the mean monocyte percentage was significantly increased in COVID-19 patients at admission compared with COVID-19 patients at discharge. In table 2, the mean plasma nitric oxide, hs C-RP, IFN-γ and IL-4 were significantly increased while MPO was significantly reduced in COVID-19 patients at admission compared with COVID-19 patients at discharge.

Also from the same Table, the mean LDH activity in COVID-19 patients at admission was non-significantly higher compared with the level in COVID-19 at discharge.

Table 1: Cellular innate immune parameters in COVID-19 patients at admission and at discharge

Variables	COVID-19 patients at admission (n = 85)	COVID-19 patients at discharge (n = 85)	P-value
WBC ($\times 10^9$/L)	5.19 ± 1.79	6.17 ± 2.29	0.001*
Monocyte (%)	9.98 ± 3.66	7.77 ± 2.91	0.045*
Neutrophil (%)	39.33 ± 16.41	45.72 ± 15.13	0.043*
Eosinophil (%)	3.58 ± 3.18	3.65 ± 2.67	0.852

**Significant at p ≤ 0.05, WBC = White blood cells*

Table 2: Comparison of mean levels of innate humoral immune factors COVID-19 patients at admission and COVID-19 patients on discharge

Variables	COVID-19 patients at admission (n=85)	COVID-19 patients at discharge (n=85)	P-values
hs C-RP*(mg/L)*	33.76±0.50	2.19±0.44	0.010*
NO*(μg/dL)*	129.70±7.30	100.08±6.00	0.029*
MPO*(U/mL)*	1.07±0.10	2.46±0.19	0.015*
LDH (U/L)	296.15 ± 66.16	289.99± 100.78	0.549
IFN-γ (pg/L)	0.180±0.20	0.149±0.18	0.038*
IL-4 (pg/L)	0.412±0.16	0.220±0.10	0.044*

**Significant at p ≤ 0.05*

DISCUSSION

Many studies have shown that in hospitalised patients with COVID-19, pre-existing cardiovascular disease or cardiac injury predicts higher mortality [1-5]. According to the United States Center for Disease Control and Prevention (CDC), cardiovascular diseases such as heart failure, coronary artery disease, and cardiomyopathies confer a higher risk for severe COVID-19 disease [14]. A meta-analysis in 2020 reported that patients with chronic heart disease are more likely to have acute cardiovascular events when infected with SARS-CoV-2 [15]. Apart from pre-existing CVD leading to severe COVID-19, cardiovascular complications, as evidenced by acute cardiac injury, new-onset systolic heart failure, pericardial effusion, and acute myocarditis, have been reported in hospitalised COVID-19 patients [1-3]. Common cardiac complications of COVID-19 include acute coronary syndrome and arrhythmias [2, 4]. These tend to occur in older patients with known cardiovascular risk factors such as hypertension, diabetes mellitus, and coronary artery disease. Myocardial infarction has been noted in up to 1.3% of all COVID-19 patients [3].

Multiple mechanisms have been suggested as the cause of cardiac damage; however, SARS-CoV-2-based mechanisms have not been clearly elucidated thus far. However, the following possible causes were proposed during viral infection, viz: cytokine-mediated damage, microvascular thrombi, and/or direct cardiomyocyte injury due to viral invasion of the myocardium [16]. Autopsy evaluations have revealed small vessel endothelial damage and vasculitis (neutrophilic) as the cause of multisystem inflammatory syndrome after a COVID-19 infection in one case report [17]. Other post-mortem studies revealed similar findings with endothelitis in several organs and evidence of inflammatory cell death [18]. These studies also noted the presence of viral bodies in affected organs and host inflammatory response leading to endothelial damage [18]. These previous reports support our present findings of higher inflammatory factors (mean counts of monocytes, mean plasma nitric oxide, LDH, hs C-RP and IFN-γ) in COVID-19 patients at admission compared with COVID-19 patients at discharge. Inflammation plays a significant role in the etiology of myocardial infarction (MI), hypertension, angina pectoris (AP),

and ischemic heart disease (IHD) or coronary artery disease (CAD) [19]. Besides, several studies have reported that elevated level of CRP is strongly associated with the risk of CAD, initiates CHD, facilitates inflammatory incidence of myocardial cells, and also an independent predictor of MI, peripheral arterial disease, stroke and other cardiac events [20]. It has been reported that CRP binds to ligands exposed on SARS-CoV-2 to activate Complement pathways, eventually exacerbating tissue damage and leading to more severe disease. Significantly raised levels of hs CRP were found in COVID-19 patients at diagnosis compared with COVID-19 patients at discharge. The implication of this is that cardiovascular effect is imminent if COVID-19 patients are not properly managed.

The coronary heart disease risk associated with a high white blood cell count is comparable to those of other inflammatory markers, including C-reactive protein [21]. A growing body of evidence supports the usefulness of elevated WBC or subtypes (neutrophils, monocytes, lymphocytes, and eosinophils) as an indicator of inflammation and CHD atherosclerosis [22]. These cells are also useful in predicting recurrent events and death in patients who already have CHD [23]. Multiple studies have shown that a high WBC count is associated with increased mortality rates in patients who present with unstable angina pectoris, acute coronary syndromes (ACS), and acute myocardial infarction (MI), and in patients who undergo percutaneous coronary interventions (PCIs) or coronary artery bypass grafting (CABG) [22-25]. Moreover, elevated total WBC, eosinophil, neutrophil and monocyte counts coupled with reduced lymphocytes, are associated with CHD, MI, cerebral ischemia and stroke [26]. The present study found reduced neutrophils and WBC in COVID-19 patients at admission, thus supporting a low likelihood of CVD in these patients, unless poorly managed. In this study, monocyte percentage was significantly lower in COVID-19 patients at discharge compared with at admission. This could be attributed to increased death, or reduced formation and redistribution from circulation to infected organs.

The widespread presence of LDH in cells and its iso-enzymatic tissue specificity have made LDH a significant diagnostic parameter in myocardial infarction, hemolytic anemia and various types of myopathies [26]. Factors of haemolysis and inflammation have been reported in COVID-19 patients [27-29] suggesting that raised LDH in COVID-19 may contribute to CVDs in COVID-19 patients if poorly managed. In the present study, mean LDH level was not significantly higher in COVID-19 patients at admission compared with COVID-19 at discharge. The hypothesis is that haemolysis, cell damage and inflammation were still on-going in COVID-19 at discharge.

Different heart diseases, including coronary heart disease (CHD), atherosclerotic heart disease and CHF, are associated with increased levels of proinflammatory cytokines such as interferon-γ (IFN-γ) [27], which was found to be raised in COVID-19 patients at diagnosis. This is an indication that CVD may not likely occur in well treated COVID-19 patients. NO plays an important role in the protection against the onset and progression of cardiovascular disease. The underlying pathology for most cardiovascular diseases is atherosclerosis, which is in turn associated with endothelial dysfunctional. The cardioprotective roles of NO include regulation of blood pressure and vascular tone, inhibition of platelet aggregation and leukocyte adhesion, and prevention smooth muscle cell proliferation.

Disturbances in NO bioavailability leads to a loss of the cardio-protective actions and in some case may even increase disease progression [13]. We reported significantly higher level of NO in COVID-19 patients at diagnosis compared with at discharge indicated potentially reduced CVD in treated COVID-19 patients. Myeloperoxidase (MPO), an heme peroxidase that is primarily expressed by neutrophils has the capacity to generate several reactive species for antimicrobial activity. However, increased MPO level in circulation was associated with conditions of increased oxidative stress and inflammation [28]. Multiple molecular mechanisms have been proposed to explain the association between MPO and CVD. MPO related CVD factors are plasma lipoproteins, NO system, endothelium, matrix proteinases and atherosclerotic plaque [12, 13]. Given MPO's emerging role as a potential biomarker or therapeutic target for CVD [29] and the increased activities of MPO in COVID at discharge compared with diagnosis, it is therefore pertinent to concluded that reduced MPO at diagnosis reduces the risk of CVD in newly diagnosed COVID-19 patients but CVD might arise in poorly managed COVID-19 patients.

Conclusions

Reduced monocytes, NO, hs CRP, IFN-gamma in COVID 19 patients at discharge suggested non-occurrence of CVDs in treated COVID-19 patients. These innate immune factors are inexpensive, easy to do and widely available parameters, however, traditional cardiac biomarkers such as troponin and B-type natriuretic peptide (BNP), electrocardiogram and echocardiography should be considered in further studies to confirm CVD-COVID-19 relationship.

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