



## **Complete Blood Count (CBC) Status among Hypertensive Subjects in Isiala Mbanjo, Imo State, South East, Nigeria**

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### **Authors' contribution**

*This work was carried out in collaboration between all authors. Authors MUE, AAN, JNE and MCO designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors CNE and PIU managed the analysis of the study. Authors PCU, DCE and MIO managed the literature. All authors read and approved the final manuscript.*

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### **ABSTRACT**

**Aim:** The Complete Blood Count (CBC) among hypertensive subjects in Isiala Mbanjo, Imo State, Nigeria West Africa were studied.

**Methodology:** The American College of Cardiology/American Heart Association (2017) current definition of hypertension was used to ascertain, diagnoses and assigned eighty (80) hypertensive

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subjects after three different consecutive blood pressure check into tests groups. Other blood pressure measures such as pulse pressure and mean arterial pressure were calculated appropriately. Venous blood samples were collected with 5mls syringe and immediately emptied into EDTA container for complete blood count analysis.

**Results:** The results showed increase in RBC count, HB, PCV and decrease in MCV among hypertensive subjects compared with normotensive subjects. It was statistically insignificant in all age groups. No changes were seen in MCHC and MCH. The results also, showed increase in PLTS count. The increase in PLTS count was statistically significant at  $P < 0.05$  and  $< 0.001$  among hypertensive age groups 36-65yrs and  $> 66$ yrs respectively. Increase in PLTS count among 20-35yr age group was not significant. There were increase in WBC count among hypertensive subjects compared with normotensive subjects in all age groups. It was statistically significant at  $P < 0.05$  and  $< 0.001$  among hypertensive age groups 36-50yrs and  $> 66$ yrs respectively. Neutrophil was increased in all age groups and was statistically significant at  $P < 0.05$  except 20-35yr age group. No changes were seen on lymphocytes, monocytes, eosinophils and basophils.

**Conclusion:** Haematological parameters investigated could predict possible cardiovascular abnormalities such as hypertension.

*Keywords: Hypertension; normotension; complete blood count (CBC).*

## 1. INTRODUCTION

Blood pressure is the pressure of the blood within blood vessels. It is produced primarily by the contraction and relaxation of heart muscle [1]. Its measurement is recorded by systolic pressure- measured after the heart contracts and diastolic pressure- measured before the heart contracts. [1] Normal blood pressure or normotension according to New guideline from the American Heart Association and the American College of Cardiology is a systolic blood pressure of 120-129mmHg and diastolic blood pressure of 70-80mmHg [2]. Hypertension is a condition in which the blood vessels have persistently raised pressure (systolic  $> 130$ mmHg and diastolic  $> 80$ mmHg), putting them under increased stress [2,3].

Hypertension is the leading cause of preventable death and disability worldwide and is a major global risk factor for Cardiovascular Diseases (CVD) [4]. The prevalence of hypertension has increased, especially in low-and middle-income countries. Estimates suggest that 31.1% of adults (1.39billion) worldwide will have hypertension in 2020 [5]. The prevalence of hypertension among adults was higher in low-and middle-income countries (28.5%, 1.04billion people) than in high-income countries (28.5%, 349 million people)

The World health organisation reports that the [5]. Prevalence of hypertension is highest in African region in 46% of adults aged 25years and above whereas the lowest prevalence at 35% is found in the Americans [6]. In most countries in

sub-Saharan African, hypertension and its complications are reported to be significant contributors to morbidity and mortality [7,8]. In Nigeria, hypertension has been reported to be number one risk factor for stroke, cardiomyopathy, ischemic heart disease and renal complications [7].

Hypertension and its complications are responsible for about 25% of emergency medical admissions in urban hospitals in Nigeria and is the commonest clinical diagnosis in elderly Nigerians, senior executives and army recruits [7]. There were about 20million cases of hypertension in Nigeria in the year 2010. This is projected to rise to 39.1million cases by the year 2030 [9].

Several studies have reported the impact of hypertension on haematological parameters such as haematocrit, haemoglobin, red blood cell count, white blood cell count and platelet count [10,11,12]. Impaired haematological parameters may strongly indicate hypertensive end-organ damage specifically kidney [10,11,12].

Hypertension is strongly associated with functional and structural abnormalities to organs that involve in haematopoiesis [13,14,15] and blood viscosity is increased in most hypertensive patients [16]. Development of hypertension is accompanied by reduction in deformability and increase in size, number and aggregability of red blood cells which may worsen the microcirculation and enhance an end-organ damage [17,18].

Also, several studies have reported a positive association between hypertension and elevated white blood cell counts among the number of population types [19-23]. These reports suggest that inflammation could possibly play a key role in the development of cardiovascular disease [24]. Hypertensive leucocytosis has been described among Indians and Caucasians; increased leukocyte counts have been found to be related to the development of hypertension with increased risk in persons with the highest leukocyte [25,26]. Tatsukawa et al. [19] reported that significant association exists between increased neutrophils count and the incidence of hypertension among Japanese women and concludes that neutrophils are the major white blood cell component contributing to the increased incidence. Eziuzo et al. [27] in their study on comparative assessment of some WBC and platelet parameters among normotensive and hypertensive subjects in Port Harcourt, Nigeria reported significant higher leukocytes, platelet counts, mean platelet volume and lower percentage of lymphocyte.

The increasing high prevalence of hypertension in our country and the reported haematological parameters as a possible risk factor among hypertensive participants in our environment are relatively scarce. Previous studies in South-East Nigeria worked not on complete blood count, not age group-related haematological parameters; hence this present study, attempts a comparative assessment on complete blood count of hypertensive and normotensive subjects. This could provide insight into the possible role these parameters play in the prognosis of hypertension in our environment.

## 2. MATERIALS AND METHODS

This study was done at Melendu Hospital Isiala Mbanu, Imo State, Nigeria. The complete blood count such as haemoglobin (Hb), Pack cell volume (PCV), White blood cell (Wbc) count and its differentials, red blood cell (Rbc) count, Mean corpuscular haemoglobin concentration (MCHC), Mean corpuscular haemoglobin (MCH), Mean corpuscular volume (MCV) and Platelets (PLTS) were evaluated at Haematology Department Imo State Teaching Hospital, Imo State.

### 2.1 Comparative Analysis

The American college of cardiology/American heart association, 2017 current definition of hypertension was used to assign a diagnosis of

hypertension in 80 patients who presented to the Medical and General Out Patient Departments with symptoms and signs of hypertension together with some that are asymptomatic. Hypertension was characterized with repeated measurement of > 130 mmHg Systolic blood pressure (SBP) or > 80 mmHg Diastolic blood pressure (DBP).

The inclusion criteria were subjects (n=80), seen by physician and diagnosed as hypertensives based on American College of Cardiology/American Heart Association, 2017 current definition of hypertension; those who came for regular follow up and are currently on antihypertensive medications. Some are newly diagnosed and yet to commence oral antihypertensive medications. In addition to that, 47 normotensive and apparently healthy subjects participated as control.

Exclusion criteria were subjects diagnosed with complications of hypertension such as ischemic and haemorrhagic stroke, congestive cardiac failure, cardiac asthma, arrhythmias, renal failure and other co-morbidity diseases like DM, malnutrition, inflammatory, infectious diseases, dyslipidaemia and malignant diseases.

A total of one hundred and twenty-seven (127) adult male and female human subjects participated. They were divided into hypertensive (80) and normotensive (47) subjects. The hypertensive subjects are those who came for regular follow up and are currently on antihypertensive medications and those newly diagnosed but yet to commence antihypertensive medications. Venous blood samples were collected with 5mls syringe and immediately emptied into EDTA container for haematological analysis.

### 2.2 Laboratory Analysis

Haematological parameters analyzed include: Red blood cell count, pack cell volume, haemoglobin concentration, mean corpuscular haemoglobin concentration, mean corpuscular haemoglobin, mean corpuscular volume, platelets, white blood cell count and its differentials. These parameters were analysed using modified automated Wallace H. Coulter. [28]

### 2.3 Data Analysis

Data generated from this study was entered into Excel sheet, coded and analyzed using

Statistical Package for Social Sciences (SPSS) version 25. One- way ANOVA was used to analyze the data.

**3. RESULTS**

Tables 1-13 shows comparison of hematological parameters between hypertensive and normotensive subjects. There was an increase in RBC, HB, PCV and decrease in MCV among hypertensive subjects compared with normotensive subjects. It was statistically insignificant in all age groups. No changes were seen in MCHC and MCH. The increase in PLTS count was statistically significant at P<0.05 and

<0.001 among hypertensive age groups 36-65yrs and >66yrs respectively. Increase in PLTS count among 20-35yr age group was not significant.

There was increase in WBC count among hypertensive subjects compared with normotensive subjects in all age groups. It was only statistically significant at P<0.05 and <0.001 among hypertensive age groups 36-50yrs and >66 yrs respectively. Neutrophil was increased in all age groups and was statistically significant at P<0.05 except 20-35yr age group. No changes were seen on lymphocytes, monocytes, eosinophils and basophils.

**Table 1. Comparison of blood pressure (mmHG) in age- related hypertensive subjects**

S/N	Age groups	Normotensive (mmHg)		Hypertensive (mmHg)		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	28.11±4.54	4	29.25±4.35	9	0.681
2	36-50	42.09±3.59	16	46.38±3.69	11	0.006*
3	51-65	56.76±3.63	34	59.88±4.70	17	0.020*
4	66>	71.20±3.12	26	72.00±5.13	10	0.649

*Mean±SD. \*statistically significant(p<0.05) at the age groups of 36-50yrs and 51-65yrs respectively*

**Table 2. Comparison of haematological parameters (RBC ml/ul) between normotensive and hypertensive subjects**

S/N	Age groups	Normotensive subjects		Hypertensive subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	4.40±0.73	9	5.61±0.95	4	0.065
2	36-50	3.61±0.88	11	4.27±0.81	16	0.055
3	51-65	4.11±0.72	17	4.29±0.74	34	0.067
4	66>	3.81±0.66	10	4.41±0.66	26	0.060

*Mean±SD Not statistically significant (p>0.05) at all the age groups of 20-66 yrs and above respectively*

**Table 3. Comparison of haematological parameters (Hbg/l) between normotensive and hypertensive subjects**

S/N	Age groups	Normotensive subjects		Hypertensive subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	12.30±1.57	9	14.38±1.15	4	0.091
2	36-50	10.69±2.21	11	11.79±2.17	16	0.061
3	51-65	11.85±1.47	17	12.32±1.96	34	0.074
4	66>	10.36±1.93	10	13.10±5.97	26	0.066

*Mean±SD Not statistically significant (p>0.05) at all the age groups of 20-66 yrs and above respectively*

**Table 4. Comparison of haematological parameters (PCVg/l) between normotensive and hypertensive subjects**

S/N	Age groups	Normotensive subjects		Hypertensive subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	36.90±4.72	9	43.15±3.42	4	0.087
2	36-50	32.09±6.65	11	35.35±6.49	16	0.063
3	51-65	35.55±4.42	17	36.98±5.83	34	0.069
4	66>	31.09±5.79	10	36.20±5.35	26	0.065

*Mean±SD Not statistically significant (p>0.05) at all the age groups of 20-66 yrs and above respectively*

**Table 5. Comparison of haematological parameters (MCHCg/dl) between normotensive and hypertensive subjects**

S/N	Age groups	Normotensive subjects		Hypertensive subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	33.02±1.36	9	34.26±0.90	4	0.128
2	36-50	31.84±1.92	11	32.96±2.03	16	0.162
3	51-65	33.52±3.17	17	33.31±1.70	34	0.765
4	66>	31.69±1.71	10	32.85±1.70	26	0.074

*Mean±SD Not statistically significant (p>0.05) at all the age groups of 20-66 yrs and above respectively*

**Table 6. Comparison of haematological parameters (MCH pg) between normotensive and hypertensive subjects**

S/N	Age Groups	Normotensive subjects		Hypertensive Subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	27.27±1.50	9	27.74±1.21	4	0.599
2	36-50	26.42±1.83	11	27.70±2.30	16	0.135
3	51-65	27.28±1.38	17	27.80±1.30	34	0.196
4	66>	26.16±1.80	10	27.33±1.73	26	0.081

*Mean±SD Not statistically significant (p>0.05) at all the age groups of 20-66 yrs and above respectively*

**Table 7. Comparison of haematological parameters (MCV fl) between normotensive and hypertensive subjects**

S/N	Age Groups	Normotensive subjects		Hypertensive Subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	80.56±2.31	9	82.16±2.25	4	0.085
2	36-50	78.97±2.33	11	81.25±4.18	16	0.073
3	51-65	80.84±2.75	17	81.49±2.65	34	0.074
4	66>	79.49±2.03	10	81.25±3.13	26	0.063

*Mean±SD Not statistically significant (p>0.05) at all the age groups of 20-66 yrs and above respectively*

**Table 8. Comparison of haematological parameters (PLT ml/ul) between normotensive and hypertensive subjects**

S/N	Age Groups	Normotensive subjects		Hypertensive Subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	331.33±86.73	9	314.50±117.86	4	0.067
2	36-50	362.45±118.87	11	299.69±134.55	16	0.05*
3	51-65	337.94±104.71	17	336.76±132.12	34	0.05*
4	66>	338.30±102.87	10	327.46±132.12	26	0.02*

*Mean±SD. \*statistically significant (p<0.05) at the age groups of 36-50yrs and 51-65yrs respectively*

**Table 9. Comparison of haematological parameters (WBC ml/ul) between normotensive and hypertensive subjects**

S/N	Age Groups	Normotensive subjects		Hypertensive Subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	4.78±1.78	9	5.12±2.54	4	0.77
2	36-50	5.05±3.19	11	7.06±7.68	16	0.05*
3	51-65	4.68±1.52	17	4.94±1.96	34	0.061
4	66>	6.69±2.96	10	4.06±1.50	26	0.001*

*Mean±SD. \*statistically significant (p<0.05) at the age groups of 36-50yrs and 51-65yrs respectively*

**Table 10. Comparison of haematological parameters (NEUTROPHILS) between normotensive and hypertensive subjects**

S/N	Age Groups	Normotensive subjects		Hypertensive Subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	49.55±7.24	9	46.08±3.94	4	0.393
2	36-50	46.66±8.40	11	50.21±7.38	16	0.05*
3	51-65	49.49±9.95	17	52.53±9.11	34	0.05*
4	66>	55.23±7.52	10	50.81±7.93	26	0.03*

Mean±SD. \*statistically significant (p<0.05) at the age groups of 36-50yrs and 51-65yrs respectively

**Table 11. Comparison of haematological parameters (Lymphocytes) between normotensive and hypertensive subjects**

S/N	Age Groups	Normotensive subjects		Hypertensive Subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	38.92±8.35	9	41.45±3.40	4	0.577
2	36-50	42.53±8.18	11	38.58±7.22	16	0.197
3	51-65	39.72±9.58	17	36.12±9.00	34	0.193
4	66>	34.52±7.22	10	38.04±8.55	26	0.257

Mean±SD Not statistically significant (p>0.05) at all the age groups of 20-66 yrs and above respectively

**Table 12. Comparison of haematological parameters (Monocytes) between normotensive and hypertensive subjects**

S/N	Age Groups	Normotensive subjects		Hypertensive Subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	9.94±1.95	9	10.51±1.29	4	0.608
2	36-50	9.58±1.94	11	10.15±1.77	16	0.434
3	51-65	9.50±1.30	17	9.71±1.49	34	0.629
4	66>	8.89±1.89	10	9.88±1.66	26	0.155

Mean±SD Not statistically significant (p>0.05) at all the age groups of 20-66 yrs and above respectively

**Table 13. Comparison of haematological parameters (EOSINOPHILS (EOSN)) between normotensive and hypertensive subjects**

S/N	Age Groups	Normotensive subjects		Hypertensive Subjects		P-Value
		(Mean±SD)	N	(Mean±SD)	N	
1	20-35	1.58±1.22	9	1.96±0.08	4	0.561
2	36-50	1.24±0.76	11	1.06±0.85	16	0.592
3	51-65	1.29±0.77	17	1.65±0.85	34	0.156
4	66>	1.30±0.82	10	1.27±0.78	26	0.910

Mean±SD Not statistically significant (p>0.05) at all the age groups of 20-66 yrs and above respectively

#### 4. DISCUSSION

Hypertension is a salient killer; a leading cause of preventable death and disability worldwide with a major global risk factor for CVD [4]. It is a major risk factor for stroke, cardiomyopathy, ischemic heart disease and renal complications in Nigeria [7]. Hypertension is associated with physiological and structural changes in blood vessels and organs involve in blood formation [17].

This study evaluated complete blood count status among hypertensive subjects and

compared it with normotensive subjects in Owerri, Imo State. In this study, the prevalence of hypertension is highest in age groups 30-50 yrs followed by 51-65yrs. Several studies in both developed and developing countries have consistently shown a positive relationship between age and blood pressure [29,30,31]. Aging increases stiffness of arteries in the vasculature and endothelial atherosclerotic changes. Blood pressure has an increasingly positive association with arterial stiffness as age increases [32]. The results of epidemiological studies have revealed the relation of age with arterial stiffness in patients with hypertension, as

age advance, so do the prevalence of hypertension and arterial stiffness [33,34].

There is increase in WBC count with predominant neutrophilia among hypertensive subjects when compared with the normotensive subjects in this study. This collaborate with the following studies [19,20-27]. Also, platelet count was high and significant among hypertensive subjects when compared with the normotensive subjects in this study. This is in agreement with studies [27,35,36]. Links between inflammation, platelets and hypertension have been suggested in the past with various inflammatory markers including high sensitivity c-reactive protein, interleukin-6 and white blood cell count have been studied and found to be associated with hypertension and its complication [37,38,39]. In early stages of hypertension, there is autoregulatory changes in vascular resistance through the autocrine/paracrine system occur in response to the production of endogenous vasoconstrictors (catecholamines) or endogenous vasodilators(nitric oxide) [40]. In untreated and poorly managed hypertension, the sustained increase in blood pressure overwhelms the autoregulation of the endothelial control of vascular tone, leading to mechanical vascular wall stress with subsequent endothelial damage and vascular permeability [40]. This permeability leads to the leakage of plasma into the vascular wall, resulting in increased activation of platelets, initiation of the coagulation cascade and recruitment of inflammatory mediators [41,42,43]. High blood pressure causes endothelial damage via shear stress; inflammation which may also contribute to increasing microvascular capillary resistance, initiation of platelet aggregation and increased catecholamine levels [19,26,36]. Results obtained in this study showed increase in platelet count that was statistically significant among the hypertensive subjects when compared with the normotensive subjects. Platelets secrete and express a large number of substances that are crucial mediators of coagulation, thrombosis, atherosclerosis and inflammation [44,45]. Platelets from hypertensive patients show increased sensitivity to agonist and have high intracellular free calcium ion concentration [46]. Intracellular events and platelet aggregatory responses may be linked to common regulatory pathways. Vasoactive ligands, such as angiotensin II bind to specific G protein-coupled membrane-bound receptors, resulting in phospholipase C activation and consequent phosphoinositide hydrolysis. These intracellular

responses may lead to increased cytosolic calcium concentrations [46]. These reports clearly implicate platelet in the possible pathogenesis of inflammation, atherogenesis and possibly hypertension. Elevated WBC counts might cause a chronic inflammation that alters endothelial function affecting nitric oxide and prostacyclin production and consequently, a loss of vasodilator, antithrombotic and antiatherogenic properties of the vascular endothelium [47]. On the other hand, increased adherence of stimulated leukocytes to vascular endothelium might cause capillary leucocytosis and subsequent increased vascular resistance; hence a raised WBC count may therefore indicate increased catecholamine or enhanced sympathetic nervous system activity, thus causing an increased in blood pressure and eventually resulting in sustained hypertension. [26]. Also, hypertension is characterized by increased peripheral vascular resistance to blood flow in arteries. These resistant arteries eventually undergo vascular remodelling such as increased media width and reduced lumen size which leads to structural and functional changes in the endothelium [48]. These changes are often apparent in the early stages of hypertension in part due to systemic inflammation but progresses in untreated and poorly controlled hypertension [49].

There was an increase in RBC, PCV and HB among hypertensive subjects when compared with the normotensive subjects in this study but it was statistically insignificant. Studies done by [17,18,35] demonstrated significant increase in RBC and HB. Hypertension is strongly associated with functional and structural abnormalities to organs that involve in haematopoiesis [13,14,15] and blood viscosity is increased in most hypertensive patients [16]. Development of hypertension is accompanied by reduction in deformability and increase in size, number and aggregability of red blood cells which may worsen the microcirculation and enhance an end-organ damage [17,18]. During hypertension, there is alteration in rheological, mechanical and biochemical characteristics of erythrocytes. Also, there is increase in blood viscosity, decrease in RBC deformability, formation of RBC rouleaux and RBC aggregates. These hemorheological determinants can favour an increase of peripheral resistances and arterial blood pressure, causing or worsening hypertension. It also decreased oxygen transport to tissue and reduced peripheral perfusion [50]. Hypertensive erythrocyte membrane

abnormalities are also based on the mechanical stress accompanying vasoconstriction derived from vascular endothelial dysfunction [51]. Impaired erythrocyte passage in the microvasculature causes mechanical damage to the stagnant erythrocyte membrane, resulting to insufficient oxygen delivery and tissue hypoxia, thus leading to further vasoconstriction and blood pressure elevation [52]. This vicious rheologic cycle may underlies hypertensive progression and complications. Oonishi T et al. [53] demonstrated and confirmed through filtration technique that mechanical impairment of human erythrocyte deformability in vitro is reversible. Therefore, deformability is mostly linked to temporary blood pressure elevation and inadequate blood control cannot reverse the impaired deformability leading to more mechanical damage to erythrocyte, reduction in HB getting to tissues and decreased oxygen supply to tissue, thus leading to vicious rheologic cycle. Target organ damage in the hypertensive patients is promoted by oxidant stress and procoagulant, proinflammatory effects mediated partly by the angiotensin type 1 receptor via the toll-like receptor [54]. These harmful effects cause marked increase in erythrocyte deformability, reduction in HB getting to tissues and impaired microcirculation accelerates hypertensive complications. Increase in HB seen among hypertensive subjects is as a result of increase in erythrocyte count which in turn increases blood pressure through imposing additional load on cardiovascular system. HB a major cytosolic protein in erythrocytes through limiting the nitric oxide availability for vascular smooth muscle cells induced vasoconstriction which explains the positive association between elevated level of HB with hypertension.

MCV was decreased in hypertensive subjects compared with normotensive subjects in this study. The decrease in MCV was not statistically significant. This is consistent with studies done by [55,56] whereas [57,58] showed no relationship between MCV and hypertension. Decrease in MCV might be adaptive mechanism to decrease RBC induced hypertension and viscosity without compromising blood flow.

## 5. CONCLUSION

In conclusion, the haematological parameters investigated in this study showed increase in WBC count, RBC count, HB, PLTS and decrease in MCV among hypertensive subjects. This provides an insight regarding the relationship

between haematological parameters with risk of hypertension in a representative sample of adults Imo State which have a high prevalence of hypertension and CVD. Therefore, routine haematological investigation is recommended for hypertensive patients for early detection and effective management of hypertension and its complications.

## CONSENT

A total of one hundred and twenty-seven (127) male and female aged 25years and above were recruited in this studied having sought and obtained their consent.

## ETHICAL APPROVAL

Approval for this study was granted by the Ethical Committee of the Federal Medical centre Owerri, Imo State (Ref no.34/380). Informed consent was obtained from the subjects before the collection of blood sample.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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