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Arterial blood pressure indices among young adults with sickle cell anaemia in Nigeria

Abstract

Background: Sickle cell disease (SCD) is a hereditary chronic haemolytic disorder characterised by repeated vaso-occlusion and chronic haemolysis resulting into chronic complications such as pulmonary arterial hypertension (PAH) and early mortality.

Aim: This study is aimed at assessing the arterial blood pressure indices and the clinical implications in individuals with SCA compared with normal Haemoglobin AA among young adult individuals in Nigeria.

Subjects and Methods: A total of 132 young adults (18 – 35 years) participated in the study. They were classified into two groups; 69 cases (participants with Hb SS) and 63 age and sex matched controls (participants with Hb AA) which were purposively selected after haemoglobin typing using electrophoresis method. Blood pressure (BP) measurement was done three times after five minute rest using a validated digital sphygmomanometer (Lumiscope) of appropriate cuff size attached to the arm. The average of the last two measurements was calculated and used as the systolic and diastolic blood pressure. Pulse Pressure (PP), Mean Arterial Pressure (MAP), Rate Pressure Product (RPP) was calculated using standard formulae. Data were analyzed using descriptive and inferential statistics and alpha value was set as < 0.05.

Results: The mean SBP [110.62 ± 10.55 vs 114.94 ± 9.49 (t=-2.297, p=0.023)], DBP [66.20 ± 7.06 vs 77.06 ± 8.44 (t=-6.956, p<0.0001)] and MAP [81.01 ± 8.21 vs 89.69 ± 7.92 (t=-5.774, p<0.0001)] were significantly lower while the PP [44.42 ± 8.60 vs 37.88 ± 8.14 (t=4.187 ± p<0.0001)] was significantly higher among the participants with SCA.

Conclusion: This study showed that SCA is associated with significantly lower SBP, DBP, and MAP with a significantly higher PP when compared with age and sex matched young adults with normal haemoglobin type.

Keywords: Sickle cell anaemia, blood pressure indices and young adults

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Introduction

Sickle cell anaemia (SCA) is a multisystemic disorder that resulted from a single gene mutation which is characterized by chronic haemolytic anaemia, vaso-occlusion, vasculopathy, organ failure/ dysfunction and early mortality.[1-3] The incidence of sickle cell disease is estimated to be between 300,000 and 400,000 newborn each year globally, the majority in sub-Saharan Africa especially Nigeria.[4] Chronic haemolytic anaemia that occurs in SCA is known to cause increased cardiac output as a result of an increase in stroke volume. Sustained increase in blood volume or cardiac output is expected to increase blood pressure unless peripheral resistance falls, as indicated in the Hagen-Poiseuille equation.[4,5] In SCA, endothelin levels and metabolites of nitric oxide (NO) are increased, which suggest increased catabolism and possible depletion of NO.[6,7] The release of erythrocyte arginase during haemolysis will limit the availability of arginine to NO synthase, contributing to a deficiency of NO.[8] This reduction in serum NO is expected to cause increase in blood pressure because of the vasodilatory effect of NO but previous studies have shown that the arterial blood pressure is lower among sickle cell individuals when compared with age and sex-matched individuals with normal haemoglobin type (Hb AA).[9]

The lower blood pressure among people with SCA have been attributed to salt losing sickle cell nephropathy, alteration in circulating levels of catecholamine, renin, aldosterone, and prostaglandin, or changes in the sensitivity of receptors to these agent.[10,11] The reduced blood pressure of steady state SCA was also due to natriuresis and hyposthenuria as a result of impairments in renal handling of sodium and potassium.[12] Also, because of frequency of hypoxia/ ischaemia, more fluid is retained via the rennin-angiotensin-aldosterone mechanism but vasopressor effect of angiotensin II on blood vessels in order to increase blood pressure, being a potent vasodilator is not effective in SCA individuals.[13] Other reasons suggested for these observations but not with general scientific acceptance are lower BP and has been intrinsically linked to low weights of the patients while a direct vascular control mechanism has also been suggested - since SCA subjects exhibit decreased pressor response to angiotensin II but not to norepinephrine.[14,15]

Mean arterial pressure (MAP) is the average arterial pressure in one cardiac cycle, systole and diastole. MAP is influenced by cardiac output and systemic vascular resistance, each of which is under the influence of several variables.[16] MAP regulation is at the cellular level through a complex interplay between the cardiovascular, renal and autonomic nervous systems. The cardiovascular system determines the MAP through cardiac output and systemic vascular resistance. Cardiac output is regulated on the level of intravascular volume, preload, afterload, myocardial contractility, heart rate, and conduction velocity. Systemic vascular resistance regulation is via vasoconstriction and dilation. The renal system affects MAP via the renin-angiotensin-aldosterone system; this is a cascade that ends in the release of aldosterone, which increases sodium reabsorption in the distal convoluted tubules of the kidneys and ultimately increases plasma volume. The autonomic nervous system plays a role in

regulating MAP via baroreceptors located in the carotid sinus and aortic arch.[17] The autonomic nervous system can affect both cardiac output and systemic vascular resistance to maintain MAP in the ideal range. The mean arterial pressure was reported to be significantly lower among young adults with sickle cell when compared with age and sex-matched individuals with Hb AA.[18] This may explain the end-organ manifestations like ischaemia and infarction that occurs in some organs like brain due to the inability to perfuse the cerebral tissues because of persistently reduction in MAP.[19] Few studies assessing the arterial blood pressure indices and the implications had been conducted in this environment despite the highest population of individuals with SCA, hence this study. The aim of the present study is to assess the arterial blood pressure indices and the clinical implications in individuals with SCA compared with normal Hb AA among young adult individuals in Nigeria.

Methodology

We studied 69 SCA individuals in steady state and 63 individuals with Hb AA who were age and sex-matched within the age range of 18 – 35 years. Fifty-eight percent (58%) of the SCA participants were female while 42% (29) of the Hb AA were male. The participants were students of a Nigeria tertiary institution recruited between February 2020 and November 2021. The steady state was defined as absence of crisis or any symptoms attributable to acute illness over the preceding four weeks without blood transfusion in the last three months. None of the female participants was pregnant. The participants were all informed about the research and consents were obtained before participating. Ethical clearance was obtained from the Ethics and Research Committee of the Obafemi Awolowo University, Ile-Ife, Nigeria. The SCA participants were confirmed Hb SS recruited at a tertiary health center during their routine sickle cell clinic. The Hb type was reconfirmed through the haemoglobin electrophoresis. This was carried using EDTA blood sample kept at an alkaline pH of 8.6, using the method described in the Helena BioSciences procedure by Monica Ceesbrough, 2008.[20] Blood pressure (BP) measurement was done by an indirect method using a validated digital sphygmomanometer (Lumiscope) of appropriate cuff size attached to the arm. The BP measurements were taken three times in a sitting position after five minutes of rest. The average of the last two measurements was calculated and used as the systolic and diastolic blood pressure. Pulse Pressure (PP) was calculated using the formula PP = SBP – DBP. Mean Arterial Pressure (MAP) was calculated using the formula MAP = DBP + 1/3(PP). Rate Pressure Product (RPP) was calculated as the product of SBP and heart rate. Results were presented as mean ± standard deviation (SD). The student t-test was used to determine the significant difference between two groups. Data was analyzed using SPSS version 20 software.

Results

A total of one hundred and thirty nine (139) young adults within the age range of 18 – 35 years participated in this study. The mean ages ± SD in years of the two groups (SCA vs HbAA) were (21.70 ± 3.40 vs 22.10±3.45 t= -0.636, p=0.526). The mean SBP (110.62 ± 10.55 vs 114.94 ± 9.49 t= -2.297p= 0.023), DBP (66.20 ± 7.06 vs 77.06 ± 8.44 t= -6.956 p= <0.0001) and MAP (81.01 ± 8.21 vs 89.69 ± 7.92 t= 5.774 p= <0.0001) of the two groups showed a significantly lower DBP, SBP and MAP in the SCA compared to the controls while PP was significantly higher in SCA individuals than in the controls (44.42 ± 8.60 vs 37.88 ± 8.14 t= 4.187 p=<0.0001). RPP and PR showed no statistical difference among two groups as shown in table 1. The DBP and MAP were significantly higher in the male and female controls than in the SCA group while the PP was significantly higher in the female SCA cases than in the controls as shown in table 2 and 3. No significant difference was seen in PP among the male participants in the two groups. When we investigated the sex differences in the BP indices among the controls, we found a significantly higher PP and SBP in the males and a higher PR in the females. This as shown in table 4. This was similarly done among the SCA participants and we found a significantly higher PR and a lower PP in the female participants as shown in table 5.

Table 1 Effect of sickle cell anaemia on indices of blood pressure

	Hb SS (n=69)	Hb AA (n=63)	t	p-value
Age	21.70 ± 3.40	22.10 ± 3.45	-0.636	0.526
SBP	110.62 ± 10.55	114.94 ± 9.49	-2.297	0.023*
DBP	66.20 ± 7.06	77.06 ± 8.44	-6.956	<0.0001*
PR	76.74 ± 11.23	73.07 ± 8.60	1.935	0.055
PP	44.42 ± 8.60	37.88 ± 8.14	4.187	<0.0001*
RPP	8510.05 ± 1631.73	8369.73 ± 946.38	0.545	0.587
MAP	81.01 ± 8.21	89.69 ± 7.92	-5.774	<0.0001*

*- Significant (p-value < 0.05), SBP, systolic blood pressure; DBP, diastolic blood pressure; PR, pulse rate; PP, pulse pressure; RPP, rate pressure product; MAP, mean arterial pressure.

Table 2 Effect of sickle cell anaemia on indices of blood pressure among male participants

	Hb SS (n=29)	Hb AA (n=28)	T	p-value
Age	22.41 ± 3.67	22.92 ± 3.65	0.507	0.615
SBP	112.97 ± 10.81	118.56 ± 9.73	1.985	0.052
DBP	65.05 ± 7.42	75.20 ± 9.34	4.448	<0.0001*
PR	72.41 ± 11.28	70.52 ± 6.33	0.744	0.460
PP	47.91 ± 9.73	43.36 ± 6.47	1.990	0.052
RPP	8248.60 ± 1914.60	8369.42 ± 1067.12	0.280	0.781
MAP	81.02 ± 7.39	89.65 ± 8.97	3.878	<0.0001*

*- Significant (p-value < 0.05), SBP, systolic blood pressure; DBP, diastolic blood pressure; PR, pulse rate; PP, pulse pressure; RPP, rate pressure product; MAP, mean arterial pressure.

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Table 3 Effect of sickle cell anaemia on indices of blood pressure among female participants

	Hb SS (n=40)	Hb AA (n=35)	T	p-value
Age	21.18 ± 3.13	21.28 ± 3.10	0.132	0.895
SBP	108.93 ± 10.15	111.32 ± 7.85	1.006	0.318
DBP	67.04 ± 9.02	78.92 ± 7.13	5.583	<0.0001*
PR	79.88 ± 10.22	75.62 ± 9.82	1.655	0.103
PP	41.89 ± 6.74	32.40 ± 5.55	5.896	<0.0001*
RPP	8699.60 ± 1386.70	8370.04 ± 830. 56	1.072	0.288
MAP	81.00 ± 8.85	89.72 ± 6.90	4.188	<0.0001*

*- Significant (p-value < 0.05), SBP, systolic blood pressure; DBP, diastolic blood pressure; PR, pulse rate; PP, pulse pressure; RPP, rate pressure product; MAP, mean arterial pressure.

Table 4 Sex differences in blood pressure indices among young adults with normal haemoglobin

	Male (n=28)	Female (n=35)	T	p-value
Age	22.92 ± 3.651	21.28 ± 3.10	1.712	0.093
SBP	118.56 ± 9.73	111.32 ± 7.85	2.895	0.006*
DBP	75.20 ± 9.34	78.92 ± 7.13	1.583	0.120
PR	70.52 ± 6.33	75.62 ± 9.86	2.176	0.035*
PP	43.36 ± 6.47	32.40 ± 5.55	6.431	<0.0001*
RPP	8369.42 ± 1067.12	8370.04 ± 830.56	0.002	0.998
MAP	89.65 ± 8.97	89.72 ± 6.90	0.029	0.977

*- Significant (p-value < 0.05), SBP, systolic blood pressure; DBP, diastolic blood pressure; PR, pulse rate; PP, pulse pressure; RPP, rate pressure product; MAP, mean arterial pressure.

Table 5 Sex differences in blood pressure indices among young adults with sickle cell anaemia

	Male (n=29)	Female (n=40)	T	p-value
Age	22.41 ± 3.67	21.18 ± 3.13	1.509	0.136
SBP	112.97 ± 10.81	108.93 ± 10.15	1.588	0.117
DBP	65.05 ± 7.42	67.04 ± 9.02	0.971	0.335
PR	72.41 ± 11.28	79.88 ± 10.22	2.866	0.006*
PP	47.41 ± 9.73	41.89 ± 6.74	3.042	0.003*
RPP	8248.60 ± 1914.60	8699.60 ± 1386.70	1.136	0.260
MAP	81.02 ± 7.39	81.00 ± 8.85	0.008	0.994

*- Significant (p-value < 0.05), SBP, systolic blood pressure; DBP, diastolic blood pressure; PR, pulse rate; PP, pulse pressure; RPP, rate pressure product; MAP, mean arterial pressure.

Discussion

In this study, the mean SBP, DBP and MAP were significantly lower while pulse pressure was significantly higher in SCA individuals than control. These findings were comparable with earlier studies of Pikilidou et al.[21] and Oguanobi et al.[22] The MAP and DBP were significantly lower in male participants with SCA when compared with male controls. In females, the DBP and MAP were significantly lower while the PP is significantly higher in SCA individuals when compared with their female control. These findings have been attributed to systemic vasodilation mediated by the cardiovascular autonomic dysfunction as well as renal tubular defect that causes the increased sodium and water excretion among SCA individuals.[23] Other factors attributed to this findings were compensatory mechanism to overcome the detrimental effects of vasoocclusive crisis, disease salt-losing sickle cell nephropathy, lower peripheral resistance, low body mass index, zinc deficiency which resulted in alteration of circulating levels of catecholamine, renin, aldosterone and prostaglandin or changes in the sensitivity of receptors to these agents.[24-26] The significant increase PP among individuals with sickle cell anaemia was projected to be as a result of the haemodynamic effect of chronic anaemia is a predictor cardiac morbidity as seen in diabetic and hypertensive individuals.[27-29] The increased pulse pressure has been shown to predict cardiac morbidity in studies carried out among diabetic and hypertensive individuals.[27,28] Among control, the sex difference showed that the mean SBP and PP are significantly higher in males while PR is significantly higher in females. The PP is significantly lower while PR is significantly higher in females when compared with their SCA male counterparts. The RPP and DBP are higher in both SCA and control female though not significant when compared with their male counterparts. The reason for increased SBP in males has been attributed to androgen which was supported by the report of Reckelhoff et al.[30] that androgen receptor blockage reduces the blood pressure in males and administration of testosterone to ovariectomized females increases the blood pressure and modifies the pressure natriuresis. The significant difference in HR was largely accounted for by the size of the heart which is smaller in females than males.[31] This may be due to the fact that the smaller female heart, pumping less blood with each beat and needs to beat at a faster rate to meet their body demand. Furthermore, women have a different intrinsic rhythmicity to the pacemaker of their hearts, which causes them to beat faster.[32] Ramaekers et al.[33] suggested that sympathetic activity is lower in females than males which provide an explanation for the protection against cardiovascular disease observed in females.

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In conclusion, our findings support previous reports of relatively lower arterial blood pressure and mean arterial pressure with high pulse pressure in individuals with sickle cell anaemia in steady state when compared with age and sex matched young adults with normal haemoglobin type. Wide PP has been found to be an independent risk factor for increased mortality, development of proteinuria and chronic kidney disease, and adverse clinical outcome. It is hereby recommended that PP should routinely be calculated in SCA during clinic visits rather than focusing on just the SBP and DBP. Interventions such as low dose thiazide diuretics which has been found to lower PP can be introduced as routine medication for the care of sickle cell patients. With these findings a different blood pressure classification for individuals with sickle cell anaemia need to be developed.

Declarations

Ethics approval and consent to participate

Ethical approval was obtained from the Ethics and Research Committee of Obafemi Awolowo University Ile-Ife, Nigeria. The participants were all informed about the research and written consents were obtained**.** The procedures used in this study adhere to the tenets of the Declaration of Helsinki.

Consent for publication

Written informed consent was obtained from each participants.

Conflicts of interest

The authors declare that they have no competing interests**.**

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Availability of data and materials

All data generated or analyzed in this study are available with the authors and will be made available on request.

Authors' Contribution

AMA, AIO, OO and BRA contributed to the design of this research study, AMA, AIO and OO conducted the research, AMA, AIO, OO and BRA analysed the data and wrote the manuscript. All authors critically reviewed and edited the final manuscript. All authors read and approved the final manuscript.

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