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Prevalence and Factors Associated with Dyslipidemia in Adults with Sickle Cell Disease in Parakou (Benin)

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Abstract: Objective: Determine the prevalence and factors associated with dyslipidemia in adult subjects with sickle cell disease in Parakou in 2017. Methods: This research work was a descriptive, analytical, cross-sectional and observational study carried out from January 6 to June 6, 2017. It involved 100 adult subjects with sickle cell disease (mean age: 28.77 ± 8.73 years) in the city of Parakou (Benin). Serum lipid parameters were measured through enzymatic colorimetric methods on Mindray ® BS-120 automaton (Guangdong, China). The different types of dyslipidemia were classified according to criteria defined in the National Cholesterol Education Program Adult Treatment Panel III. Results: The overall prevalence of the different types of dyslipidemia was estimated at 82% (95% CI [75.27; 88.34]). The different types of dyslipidemia were distributed as followed: HDL hypocholesterolemia (79%; 95%IC [69.71; 86.51]), LDL hypercholesterolemia (13.0%; 95%CI [7.11; 21.20]), total hypercholesterolemia (7.0%; 95%CI [02.86; 13.89]), hypertriglyceridemia (4.0%; 95%CI [1.10; 9.93]) and mixed hyperlipidemia (1.0%; 95%CI [0.03; 5.45]). Atherogenic dyslipidemia was not found out. HDL hypocholesterolemia was significantly associated with personal history of hypertension (P = 0.029), emaciation (P = 0.023) and age above or equal to 50 years (P = 0,016). Tobacco consumption (P = 0.01) and age below 50 years (P = 0.02) were significantly associated with hypertriglyceridemia. Conclusion: The prevalence of the different types of dyslipidemia is high among adults with sickle cell disease in Parakou.

Keywords: Dyslipidemia, Hypercholesterolemia, Sickle Cell Disease, Benin

1. Introduction

Sickle cell disease is a hereditary disease due to substitution of glutamic acid at position 6 of the β-globin chain by valine, resulting in hemoglobin S synthesis [1, 2]. It is responsible for the damage of many organs with high risk of early mortality. The management of that disease has seen advances in developed countries whereas it is still causing morbidity and early mortality in developing countries [3].

In addition to hemorheological signs and symptoms that characterize homozygous sickle cell disease (Hb SS), inflammatory response, oxidative stress [4-6] and high atherogenic risk [7, 8] exacerbate prognosis for subjects with that phenotype. Subjects presenting with simple heterozygous form (Hb AS) seem to develop a tendency for coronary insufficiency [9].

There is a controversy about lipid parameter values in subjects with sickle cell disease. According to Rahimi et al. [10], subjects with sickle cell disease trait presented with increased HDL cholesterol whereas subjects with homozygous sickle cell disease had lower total cholesterol concentration compared to normal subjects (Hb AA) and subjects with sickle cell disease trait (Hb AS). For Ephraim et al. [11] and Gueye Tall et al. [12], a decline in total cholesterol and HDL cholesterol has been observed in subjects with homozygous and heterozygous sickle cell disease compared to subjects with no sickle cell disease. Magalhães Aleluia et al. [6] have reported increase in total cholesterol, HDL cholesterol and LDL cholesterol and

decline in triglycerides. Other authors had found out declined HDL cholesterol and concurrent increase in LDL cholesterol, thus indicating potential biomarkers for disease severity [13, 14].

This study aimed to determine the prevalence and factors associated with the different types of dyslipidemia among adult subjects with sickle cell disease in Parakou in 2017.

2. Materials and Methods

2.1. Ethics

This study has been approved and authorized by the Local Ethics Committee for Biomedical Research of the University of Parakou (Decision Notice No. 0001/CLERB-UP/P/SP/R/SA).

2.2. Study Target Population

This research work was a descriptive, analytical, cross-sectional and observational study conducted over six-month period from January 6 to June 6, 2017.

The study population consisted of adult subjects with sickle cell diseases (mean age: 28.77 ± 8.73 years) in the city of Parakou (Benin) admitted for hemoglobin electrophoresis at the Borgou Regional branch of the National Agency for Blood Transfusion during the study period. Sampling was complete and helped recruit a total of 100 subjects with sickle cell disease (AS, SS and SC) on the basis of hemoglobin electrophoresis with pH 8.6 on Hydragel Sebia.

The study has included subjects with sickle cell disease aged 18 years or over, in stationary phase, who gave their informed consent to participate to the survey, and living in Parakou for at least six months. It has not included pregnant women, and subjects who received blood transfusion dating back from less than three months or on drugs likely to disturb lipid metabolism.

2.3. Laboratory Tests

Blood samples (4 mL) were collected into dry and EDTA tubes by means of superficial venipuncture in the antecubital area of each study subject who has been fasting for 12 hours. The blood samples collected in dry tubes were centrifuged in 1800 g during 10 minutes and then serums were decanted. The latter were used on the same day to measure lipid parameters. The blood samples collected in EDTA tubes were used to perform hemoglobin electrophoresis. Total cholesterol, HDL cholesterol and triglycerides were measured using enzymatic colorimetric assay on Mindray ® BS-120 automaton (Guangdong, China). LDL cholesterol was calculated using the formula of Friedewald et al. [15] if triglyceridemia was below 4.00 g/L. The different types of dyslipidemia were classified according to criteria defined in the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) [16].

2.4. Data Analysis

The data were processed and analyzed using the software Epi Info7. Depending on the case, Chi-square test or Fischer's test was used to compare proportions and ratios. Search for association between types of dyslipidemia and independent variables was done using logistic regression. Significance threshold was set at 0.05.

3. Results

Table 1 shows the general characteristics of the study population.

Table 1. *General characteristics of subjects with sickle cell disease in Parakou in 2017.*

N		100
Mean age (years)		28.77 ± 8.73
Sex (n)	Male	62
	Female	38
Mean BMI (kg/m2)		22.82 ± 4.38
Mean systolic blood pressure (mmHg)		116.32 ± 11.04
Mean diastolic blood pressure (mmHg)		75.93 ± 9.50
Tobacco consumption (n)		2
Alcohol consumption (n)		41
Hb SS (n)		8
Hb AS (n)		82
Hb SC (n)		10

Variations in serum lipids are shown in Table 2.

Table 2. *Distribution by age groups of variations in serum lipid profile of subjects with sickle cell disease in Parakou in 2017 (N=100).*

	Total	**Age groups (years)**	
		≤ 50	**> 50**
Total cholesterol (n)	100	96	4
Reduced	11	11 (100.00)	0 (0.00)
Normal	82	79 (96.34)	3 (3.66)
High	7	6 (85.71)	1 (14.29)
HDL cholesterol (n)	100	96	4
Reduced	79	79 (100.00)	0 (0.00)
Normal	21	17 (80.95)	4 (19.04)
LDL cholesterol (n)	100	96	4
Reduced	28	28 (100.00)	0 (0.00)
Normal	59	56 (94.92)	3 (5.08)
High	13	12 (92.31)	1 (7.69)
Triglycerides (n)	100	96	4
Reduced	50	50 (100.00)	0 (0.00)
Normal	46	43 (93.48)	3 (6.52)
High	4	3 (75.00)	1 (25.00)

NB: The values mentioned in the Table are indicated as population size and percentage in brackets.

Prevalence of the different types of dyslipidemia

HDL hypocholesterolemia (79%) and LDL hypercholesterolemia LDL (13%) were the predominant types of dyslipidemia. Atherogenic dyslipidemia was not identified (Table 3).

Table 3. *Distribution of subjects with sickle cell disease according to type of dyslipidemia in Parakou in 2017 (N=100).*

	Population size	Frequency (%)	95% CI
Total hypercholesterolemia	7	7	[02.86; 13.89]
HDL hypocholesterolemia	79	79	[69.71; 86.51]
LDL hypercholesterolemia	13	13	[7.11; 21.20]
Hypertriglyceridemia	4	4	[1.10; 9.93]
Mixed hyperlipidemia	1	1	[0.03; 5.45]

HDL hypocholesterolemia was the most common dyslipidemia, regardless of the hemoglobin phenotype. LDL hypercholesterolemia, total hypercholesterolemia and mixed hyperlipidemia were observed in subjects with heterozygous sickle cell disease (Table 4).

Table 4. *Distribution of hemotypes according to type of dyslipidemia in subjects with sickle cell disease in Parakou in 2017 (N = 100).*

	Hemotypes			
	Total	AS, n (%)	SC, n (%)	SS, n (%)
HDL hypocholesterolemia	79	66 (80.49)	7 (70.00)	6 (75.00)
LDL hypercholesterolemia	13	13 (15.85)	0	0
Total hypercholesterolemia	7	7 (8.54)	0	0
Mixed hyperlipidemia	1	1 (1.22)	0	0
Hypertriglyceridemia	4	3 (3.66)	1 (10.00)	0
Total	100	82	10	8

Factors associated with the different types of dyslipidemia

In multivariate analysis, personal history of hypertension (P = 0.029), emaciation (P = 0.023) and age range between 51 years and more (P = 0.016) were significantly associated with HDL hypocholesterolemia (Table 5). As well, tobacco consumption (P = 0.01) and age group from 18 to 50 years (P =0.02) were significantly associated with hypertriglyceridemia (Table 5).

Table 5. *Multivariate analysis of factors associated with different types of dyslipidemia in adult persons with sickle cell disease in Parakou in 2017 (N = 100).*

	RR	P	95%CI
HDL hypocholesterolemia			
High blood pressure	0.062	0.029	[0.005; 0.74]
BMI < 18 kg/m2	0.12	0.023	[0.02; 0.74]
Age > 50 years	0.02	0.016	[0.001; 0.50]
Hypertriglyceridemia			
Age [18-50 years]	1.29	0.02	[0.73; 2.27]
Tobacco consumption	0.51	0.01	[0.12; 2.06]

RR: relative risk; CI: confidence interval; P: significant

4. Discussion

The role of lipids in inflammatory response has been suggested because of anti-inflammatory properties of HDL cholesterol [17] and pro-inflammatory properties of LDL cholesterol [18]. As subjects with sickle cell disease have a chronic inflammatory state, the study of the different types of dyslipidemia in that group may help improve knowledge about their heterogeneous clinical manifestations.

Total hypercholesterolemia was exclusively observed in AS heterozygous subjects. No subject with homozygous sickle cell disease has presented with hypercholesterolemia in this study. Several previous studies which investigated subjects with SS homozygous sickle cell disease had rather noted hypocholesterolemia [1, 13, 19]. The severity of hypocholesterolemia depends on the degree of anemia in those subjects with SS homozygous sickle cell disease [1]. Many possible explanations of that hypocholesterolemia have been proposed. Chronic hemolysis results in increased erythropoiesis which consumes the cholesterol pool responsible for hypocholesterolemia [20]. Oxidative stress may be another cause of hypocholesterolemia. Indeed, a negative correlation between plasma cholesterol and malondialdehyde (MDA) activity has been observed in subjects with sickle cell disease [5], thus indicating a reduction in the number of red blood cells or a decline in plasma lipoproteins due to hyper-metabolism in response to oxidative stress [21]. In this study, the fact that no subject with homozygous sickle cell disease has presented with hypercholesterolemia substantiate data from the literature.

In this research work, HDL hypocholesterolemia was the most common dyslipidemia. In some children with sickle cell disease in Nigeria, HDL hypocholesterolemia and declined concentration of n-3 polyunsaturated fatty acids of serum phospholipids have been observed [22]. Many hypotheses have been put forward to explain decline in HDL cholesterol among subjects with sickle cell disease. Decreased volume of red blood cells results in increased plasma volume with dilution of plasma components, including lipids and lipoproteins [13]. As far as metabolism is concerned, abnormal down regulation of cholesterol biosynthesis through reduced activity of hydroxymethylglutaryl-coenzyme

A reductase has been suggested [13]. It was also suggested a decline in the activity of lecithin cholesterol acyltransferase (LCAT), an enzyme that catalyzes the formation of cholesteryl esters from cholesterol within HDL particles [13]. The increased use of cholesterol for synthesis of erythrocyte membranes in the context of red blood cell regeneration due to chronic hemolysis and liver failures [23], has also been mentioned. That HDL hypocholesterolemia with decline in Apo A-I concentration is recognized as a risk factor for artery dysfunction both in the general population and in subjects with sickle cell disease. It is a factor that potentially contributes to pulmonary hypertension in subjects with sickle cell disease even if the long term effect is low [24]. HDL cholesterol plays an important role as prognostic marker for sickle cell disease [6]. Actually, HDL cholesterol has anti-inflammatory, antioxidant, anti-platelet aggregation, anticoagulant and profibrinolytic properties [25]. A high level of HDL cholesterol may reduce the risk for intravascular hemolysis and endothelial dysfunction [14]. The HDL hypocholesterolemia observed in subjects with sickle cell disease investigated in this study may exacerbate their prognosis, for homozygous sickle cell disease is characterized by inflammatory state and lipidoperoxidation.

LDL hypercholesterolemia, which is the second most common dyslipidemia in this study, was found out only in subjects with AS heterozygous sickle cell disease. Several authors [6, 13, 26] have reported a significant reduction of LDL cholesterolemia among subjects with homozygous sickle cell disease (Hb SS) compared to Hb AA subjects with no sickle cell disease. On the contrary, Ephraim et al. [11] did not identify any modification of LDL cholesterolemia. The role of lipids has been mentioned in the inflammatory response [17, 18]. As the subject with homozygous sickle cell disease is characterized by an inflammatory state, the study of the different types of dyslipidemia may contribute to the understanding of pathophysiological mechanisms during the disease [6].

Even though it has a low prevalence in this study, hypertriglyceridemia has been observed by Mokondjimobe et al. [17] in subjects with sickle cell disease. That hypertriglyceridemia may be due to increased production of VLDL and reduced activity of lipoprotein lipase because of oxidative stress. In fact, VLDL cholesterol is used to restore membranes altered due to oxidative stress; this is not the case of triglycerides which then get accumulated [23]. Those data from the literature which associate hypertriglyceridemia with sickle cell disease are contrary to significant decline in triglycerides reported by Shores et al. [13] among black American subjects with sickle cell disease, without pathophysiological substratum.

Unlike this study, the research work of Ephraim et al. [11] identified a significant association between hyperVLDLemia and systolic ($P = 0.01$, OR: 0.74 [CI: 0.6-093]) as well as diastolic ($P = 0.023$, OR: 1.45 [CI: 1.05-2.0]) blood pressures. For Lalanne-Mistrih et al. [2], only hypertriglyceridemia was associated with acute chest syndrome ($P<0.05$). Very few studies investigated the different types of dyslipidemia and associated factors in subjects with sick cell disease.

This study has some limitations. The small size of the sample of homozygous subjects (Hb SS) may limit the power of the statistical tests used. Data collection using a questionnaire may lead to underestimation or overestimation of data due to respondents' subjective assessments and appraisals. Moreover, there may be biases in the measurement of physical parameters. However, they were minimized in this research work since parameters were collected by only one person and the same measuring material was used for all the subjects.

5. Conclusion

The prevalence of the different types of dyslipidemia in adults with sickle cell disease in Parakou in 2017 was high, with predominance of HDL hypocholesterolemia, LDL hypercholesterolemia and total hypercholesterolemia. There is no atherogenic dyslipidemia in the study population. Emaciation, excessive consumption of tobacco, age and personal history of hypertension are variably associated with different types of dyslipidemia.

This study points out that subjects with sickle cell disease are exposed to the risk of developing atheromatous plaque, in addition to hemorheological consequences related to sickle cell disease. Preventive measures should be implemented in order to limit those abnormalities of the lipid profile.

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Conflicts of Interest Statement

The authors certify that they have NO affiliation with organizations or entities with interest of any kind (political, ideological, religious, financial or non-financial) in the subject matter or materials discussed in this manuscript.

Author's Contribution

All authors contributed to conception and design, manuscript preparation, read and approved the final manuscript.

References

[1] Zorca S, Freeman L, Hildesheim M, Allen D, Remaley AT, TaylorJG, Kato GJ (2010) Lipid levels in sickle-cell disease associated with haemolytic severity, vascular dysfunction and pulmonary hypertension. Br J Haematol 149 (3): 436–445. doi: 10.1111/j.1365-2141.2010.08109.x.

[2] Lalanne-Mistrih ML, Connes P, Lamarre Y, Lemonne N, Hardy-Dessources MD, Tarer V, Etienne-Julan M, Mougenel D, Tressières B, Marc Romana M (2018) Lipid profiles in French West Indies sickle cell disease cohorts, and their general Population. Lipids in Health and Disease 17: 38. https://doi.org/10.1186/s12944-018-0689-5.

[3] Agrawal S, Tikariha BP, Khodiar PK (2013) Serum Lipid Profile In Sickle Cell Disease Patients In Raipur District, Chhattisgarh. IJBAP 2 (1): 132-135.

[4] Alsultan AI, Seif MA, Amin TT, Naboli M, Alsuliman AM (2010) Relationship between oxidative stress, ferritin and insulin resistance in sickle cell disease. Eur Rev Med Pharmacol Sci 14: 527-538.

[5] Yesim OE, Suna S, Selma U, Hilal O, Nuriman O (2011) Hypocholesterolemia is associated negatively with hemolysate lipid peroxidation in sickle cell anemia patients. Clin Exp Med 11: 195-198. doi: 10.1007/s10238-010-0124-3.

[6] Magalhães Aleluia M, Conceição da Guarda C, Pereira Santiago R, Teresa Cristina Cardoso Fonseca TC, Idalina Neves F, Quinto de Souza R, Alves Farias L, Araújo Pimenta F, Magalhães Fiuza L, Nogueira Pitanga T, Dutra Ferreira JR, Vitória Adorno E, Veloso Cerqueira BA, de Souza Gonçalves M (2017) Association of classical markers and establishment of the dyslipidemic subphenotype of sickle cell anemia. Lipids in Health and Disease 16: 74. doi: 10.1186/s12944-017-0454-1.

[7] Monnet PD, Kane F, Konan-Waidhet D, Akpona S, Kora J, Diafouka F, Sess D, Sangare A, Yapo AE (1996) Evaluation of atherogenic risk in homozygous sickle cell disease: study of lipid and apolipoprotein AI and B plasma levels. Bull Soc Pathol Exot 89 (4): 278-281.

[8] Monde AA, Kouane-Koutouna A, Tiahou GG, Camara CM, Yapo AA, Djessou SP, Sess ED (2010) Profil lipidoprotéique, isotopique et risque athérogène dans la drépanocytose en Côte d'Ivoire. Med Nucl 34: 17-21. doi: 10.1016/j.mednuc.2010.07.015.

[9] Ould Amar AK, Gibert AP, Darmon O, Besse P, Cenac A, Césaire R (1999) Hémoglobinopathies hétérozygotes AS et risque coronaire. Archives des maladies du cœur et des vaisseaux 92: 1727-1732. French.

[10] Rahimi Z, Merat A, Haghshenass M, Madani H, Rezaei M, Nagel RL (2006) Plasma lipids in Iranians with sickle cell disease: Hypocholesterolemia in sickle cell anemia and increase of HDL-cholesterol in sickle cell trait. Clinica Chimica Acta 365: 217 – 220. doi: 10.1016/j.cca.2005.08.022.

[11] Ephraim RKD, Adu P, Ake E, Agbodzakey H, Adoba P, Cudjoe O, Agoni C (2016) Normal Non-HDL Cholesterol, Low Total Cholesterol, and HDL Cholesterol Levels in Sickle Cell Disease Patients in the Steady State: A Case-Control Study of Tema Metropolis. Journal of Lipids. Article ID 7650530, 5 pages. http://dx.doi.org/10.1155/2016/7650530

[12] Gueye Tall F, Ndour EHM, Cissé F, Gueye PM, Ndiaye Diallo R, Diatta A, Lopez Sall P, Cissé A (2014) Perturbations de paramètres lipidiques au cours de la drépanocytose. Rev. CAMES SANTE 2 (2): 35-41. French.

[13] Shores J, Peterson J, Vander Jagt D, Glew RH (2003) Reduced cholesterol levels in African-American adults with sickle cell disease. J Natl Med Assoc 95: 813–817.

[14] Seixas MO, Rocha LC, Carvalho MB, Menezes JF, Lyra IM, Nascimento VM, Couto RD, Atta AM, Reis MG, Goncalves MS (2010) Levels of high-density lipoprotein cholesterol (HDL-C) among children with steady-state sickle cell disease. Lipids Health Dis 9: 91. doi: 10.1186/1476-511X-9-91.

[15] Friedewald WT, Levy RI, Fredrickson DS (1972) Estimation of concentration of lowdensity lipoprotein cholesterol in plasma without use of ultracentrifuge. Clin Chem 18 (6): 499-502.

[16] National Cholesterol Education Program (2002) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. Circulation 106 (25): 3143-421.

[17] McMahon M, Grossman J, FitzGerald J, Dahlin-Lee E, Wallace DJ, Thong BY, Badsha H, Kalunian K, Charles C, Navab M, Fogelman AM, Hahn BH (2006) Proinflammatory high density lipoprotein as a biomarker for atherosclerosis in patients with systemic lupus erythematosus and rheumatoid arthritis. Arthritis Rheum 54 (8): 2541–2549. doi: 10.1002/art.21976.

[18] Mineo C, Deguchi H, Griffin JH, Shaul PW (2006) Endothelial and antithrombotic actions of HDL. Circ Res 98: 1352–1364.

[19] Akinlade KS, Adewale CO, Rahamon SK, Fasola FA, Olaniyi JA, Atere AD (2014) Defective lipid metabolism in sickle cell anaemia subjects in vaso-occlusive crisis. Niger Med J 55 (5): 428-431. doi: 10.4103/0300-1652.140388.

[20] Shalev H, Kapelushnik J, Moser A, Knobler H, Tamary H (2007) Hypocholesterolemia in chronic anemias with increased erythropoietic activity. Am J Hematol 82: 199-202.

[21] Er Oztas Y (2012) Hypocholesterolemia in Sickle Cell Anemia: What is the Reason behind it? Biochem & Pharmacol 1: e110. doi: 10.4172/2167-0501.1000e110µ.

[22] Erasmus RT, Olukoga AO, Ojuawo O (1990) Plasma lipids and lipoproteins in Nigeria children with sickle cell anemia. Ann Trop Paediatr 10: 421-423.

[23] El Hazmi MAF, Warsey AS, Al-Swailem A, Al-Swailem A, Bahakim H (1995) Red cell genetic disorders and plasma lipids. J Trop Paediatr 41: 202–205.

[24] Yuditskaya S, Tumblin A, Hoehn GT, Wang G, Drake SK, Xu X, Ying S, Chi AH, Remaley AT, Shen RF, Munson PJ, Suffredini AF, Kato GJ (2009) Proteomic identification of altered apolipoprotein patterns in pulmonary hypertension and vasculopathy of sickle cell disease. Blood 113: 1122–1128.

[25] Nofer JR, Kehrel B, Fobker M, Levkau B, Assmann G, von Eckardstein A (2002) HDL and arteriosclerosis: beyond reverse cholesterol transport. Atherosclerosis 161: 1–16.

[26] Bhatkulkar P, Khare R, Meshram AW, Dhok A (2015) Status of Oxidative Stress and Lipid Profile in Patients of Sickle Cell Anemia. IJHSR 5 (3): 189-193.

[27] Mokondjimobe É, Longo-Mbenza B, Ovono-Abessolo F, Gombet T, Guie G, Ngou-Milama E, Heni JP (2012) Évaluation du profil lipoprotéique et du risque athérogène chez les drépanocytaires homozygotes et hétérozygotes de Brazzaville. Ann Biol Clin 70 (2): 183-188. doi: 10.1684/abc.2012.0687 French.