Original

Epidemiology of Leg Ulcers in Sickle Cell Disease Patients- A Multicenter Study

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ABSTRACT

Background: Sickle cell leg ulcers contribute to significant morbidity in patients with sickle cell disease (SCD). Nigeria has the highest burden of SCD with a significant proportion of affected patients having chronic leg ulcers. This study aimed to determine the sociodemographic, clinical and haematological characteristics of SCD patients with leg ulcers in Benin City, Nigeria.

Methodology: This was a multicenter based cross-sectional study conducted at the University of Benin Teaching hospital (UBTH) and the sickle center, Benin City between June 2023 and November 2023 among SCD patients. Eighty-eight persons were recruited in this study, comprising of 33 SCD patients with leg ulcers, 33 SCD patients and 22 genotype AA individuals. Data were analysed using the Statistical Package for Social Sciences (SPSS) version 23.

Results: The mean age (SD) of SCD patients with leg ulcers, SCD controls and HbAA controls were 29±6.6yrs, 29.3±5.9yrs and 29.9±6.7yrs respectively. The differences in mean age across the study groups were not statistically significant (p=0.932). Nineteen (57.6%) individuals with sickle cell leg ulcers were females and 14 (42.4%) were males. There was no statistically significant difference in the sex distribution (p=0.521).

Conclusion: The prevalence of leg ulcers in SCD reduced with increasing age. Most leg ulcers occurred on the right leg at the medial malleolus and full blood count showed significantly elevated platelet count in patients with chronic leg ulcers compared to controls.

Keywords: Sickle cell, Epidemiology, leg ulcers, Anaemia

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INTRODUCTION

Sickle cell disease (SCD) is one of the most common genetic diseases worldwide and it has its highest prevalence in the Middle East, Mediterranean regions, Southeast Asia, and sub-Saharan Africa which accounts for about 80% of cases[1,2] It is now considered to be a disorder of global importance both clinically and economically[3] A World Health Organization (WHO) report estimated that around 2% of newborns in Nigeria were affected by sickle cell anaemia; giving a total of 150,000 affected children born every year in Nigeria alone.[4] Another study reports that SCD affects about 2 to 3% of the Nigeria population of more than 200 million[1] Nwogoh et al. in Benin City, South-South Nigeria reported a SCD prevalence of 2.39% and a carrier rate of about 23%.[5]

The aetiology of leg ulcers in SCD is complex and multifactorial but not well understood, hence attempts at prevention have been disappointing. Management of sickle cell leg ulcers is often protracted, requiring a long stay in the hospital, thereby worsening both the economic and social well-being of persons with sickle cell leg ulcers.

The clinical manifestations of SCD are numerous and diverse. Among them, leg ulcers are relatively common and can be disabling. The prevalence of leg ulceration varies, being low before age 10 years, commoner in males and patients with low steady state haemoglobin levels.[6] Its geographical distribution is also variable, affecting 75% of HbSS patients in Jamaica but only 8–10% of North American patients.[6] Bazuaye et al. reported a prevalence rate of 9.6% and 22.4% for current ulcers and previous ulcers, respectively in Benin City.[7] They commonly arise near the medial or lateral malleolus and may be single or multiple.[6]

Leg ulcers are the most common cutaneous manifestation of SCD. The health status of individuals living with chronic leg ulcers is not only affected by clinical manifestations such as chronic pain but also the societal stigmatization and rejection. Umeh et al. observed that SCD patients with leg ulcers expressed that they experienced social isolation, intense and frequent ulcer pain, and difficulty in physical function.[8]

Hence this study aimed to determine some sociodemographic factors, clinical and haematological parameters associated with leg ulcers in SCD patients in Benin City, Nigeria; with the hope of profound better understanding of the disease, preventing modifiable risk factors, improving the quality of life of affected patients, disease prevention and achieve healthy stable state.

Materials and Methods

Study Design: A cross-sectional study was carried out

Study Area: The study was conducted at the University of Benin Teaching Hospital (UBTH), Benin City, Nigeria. Respondents were recruited from the Department of Haematology and Plastic and Burns Department both of UBTH and the Sickle cell centre Benin City. UBTH is a federal government owned hospital with over 800 bed spaces, located in Egor local government area, Benin City. The sickle cell centre is a dedicated health facility with over 9 bed spaces established by the Edo State Government for the care of patients with SCD. It is located in Oredo local government area, Benin City, Nigeria.

Study Duration: The study was terminated when the estimated sample size was realized. It was carried out within a period of 6 months (June 2023-November 2023).

Participants and Sampling Technique: Consecutive sampling technique was used for this study. The study participants were individuals with SCD aged 18 -45 years:

Group I: This consisted of SCD patients with leg ulcers aged between 18 and 45 years (both old and new).

Group II: This consisted of SCD patients in steady state without leg ulcers. They were matched for age and sex with those in group 1.

Sample size estimation: Using the formula $n = \frac{z^2\,pq}{d^2}$ to determine the mean sample size

z =1.96

p = 0.015

p=prevalence rate;(using a minimum prevalence of 1.5%)

q =1-p =0.985

d= degree of error =0.05

n =minimum sample size = 23

Minimum calculated sample size is 23; however, 33 sickle cell patients with leg ulcer were recruited into the study.

Study Instrument: After obtaining consent, a designed non-validated study proforma was used to collect demography of study participants, medical history, drug history and history of complications. Thereafter, blood samples were collected for complete blood count and clotting profile.

Sample Collection: Six milliliters (mls) of venous blood was drawn aseptically from the antecubital vein of each subject with minimal stasis. Four and a half (4.5mls) of

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whole blood for coagulation test (Prothrombin time [PT] and activated partial thromboplastin time [APTT]) was dispensed into a sample bottle containing 0.5mls of 0.109M sodium citrate (3.2%). This was to obtain a blood: citrate ratio of 9:1. The sample was mixed by gentle inversion at least six times to ensure adequate mixing of the anticoagulant with the blood. The sample was transported in an ice pack to maintain viability from point of collection to the laboratory within two hours. The sample was centrifuged at room temperature at a speed of 2000 gravities (g) for 10mins to obtain platelet poor plasma. The plasma was carefully removed to prevent cell lysis with a plastic pipette into a plane bottle. The PT and APTT assays were analyzed immediately. The remaining volume of whole blood was dispensed into commercially prepared ethylene di-amine tetra-acetic acid (EDTA) bottle for full blood count. The sample was mixed gently but thoroughly to prevent cell lysis and ensure anticoagulation. The EDTA sample was analysed immediately. All specimens were labelled with personally generated identification numbers and recorded in the datasheet.

Test Procedures: Basic Haematological Parameters: Full blood count includes haematocrit, haemoglobin concentration, total white cell count and platelet count was obtained from the EDTA sample, using automated blood cell counter (Sysmex Haematology Autoanalyser model KN21). The basic principles underlying these techniques are electronic impedance and light scatter. This was done in the main haematology laboratory, UBTH. Blood sample is aspirated and proportioned, then diluted to a pre-set ratio and labelled with a proprietary fluorescence marker that binds specifically to nucleic acids. Next the sample is transported into the flow cell. The sample is illuminated by a semiconductor laser beam, which can separate the cells using different signals. The intensity of the forward scatter indicates the cell volume. The side scatter provides information about the internal cell structure and its content, such as nucleus and granules. The side fluorescence indicates the amount of nucleic acids present in the cell.

Haemoglobin Electrophoresis: The haemoglobin phenotypes of both subjects and healthy participants were confirmed using haemoglobin electrophoresis.

Determination of coagulation tests: Prothrombin time and activated partial thromboplastin time test was carried out for study group. This was done in the haematology Laboratory in UBTH Benin.

Data Analysis: Data obtained was analysed using Statistical Package for the social sciences (SPSS) version 23. Continuous variables (age, PT and APTT) were tested for normality. Normally distributed variables (age, WBC count and differentials, platelet count, HCT, PT, APTT) were summarized as mean, standard deviation and ranges while skewed variables (p-selectin and VWF levels) were summarized as median and interquartile ranges. Comparison of mean between the groups for normally distributed continuous variables was done with the student t-test while Mann Whitney U test was used to compare differences in Median.

A total of eighty-eight individuals participated in this study, comprising 33 SCD patients with sickle cell leg ulcers, 33 SCD controls (without leg ulcers) and 22 HbAA controls. The age range of the SCD individuals with sickle cell leg ulcers (SCLU) was 20 – 45 years with a mean (SD) of 29.7±6.6yrs. The SCD controls had a mean (SD)age of 29.3±5.9yrs and HbAA controls 29.9 ± 6.7yrs. The differences in mean age across the study groups were not statistically significant (p=0.932). The peak age range of SCD SCLU was 25 – 29yrs. Nineteen (57.6%) individuals with SCA SCLU were females and 14 (42.4%) were males. The differences in the sex distribution between the study population was not statistically significant (p = 0.521) (Table 1). Majority (18, 54.5%) of the SCA SCLU population had secondary level education and 12 (36.4%) had tertiary level education.

The median duration from onset of SCLU was 2.0yrs with an interquartile range of 1.0 – 2.0 years. In 22 (66.7%) of SCD SCLU individuals, the leg ulcers were localized to the right foot, in 7 (21.2%) to the left and in 4 (12.1%), both legs were affected. (Table 2). The most prevalent complaint in individuals with SCD SCLU was pain (13, 39.4%), followed by swollen feet (3, 9.1%), discharge (1, 3.0%) and paresthesia (1, 3.0%). Fourteen (42.4%) patients with SCLU had stage II ulcers and 19 (57.6%) had stage III ulcers.

Twenty-three (69.7%) SCLU individuals reported having less than three Vaso-occlusive events per annum. Two (6.1%) reported three or more hospital admissions per year and 18 (54.5%) were transfused more than three months previously. (Table 3)

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Results

Table 1: Sociodemographic characteristics of the study population

	SCD SCLU n = 33	**SCD Control** n = 33	**HbAA Control** n = 22	χ^2	**P-value**
Age group (in years)					
20 – 24	6 (18.2)	6 (18.2)	4 (18.2)		
25 – 29	15 (45.5)	15 (45.5)	8 (36.4)		
30 – 34	5 (15.2)	6 (18.2)	5 (22.7)	0.731	0.999
35 – 39	4 (12.1)	3 (9.1)	3 (13.6)		
≥40	3 (9.1)	3 (9.1)	2 (9.1)		
Mean ± SD	29.7±6.6	29.3±5.9	29.9 ± 6.7	0.071	0.932
Range	20 – 45	21 – 45	20 – 44		
Age at SCD diagnosis (in years)					
Median	4.0	4.0			0.553
IQR	2.0 – 6.5	2.0-6.0			
Sex					
Male	14 (42.4)	13 (39.4)	12 (54.5)	1.305	0.521
Female	19 (57.6)	20 (60.6)	10 (45.5)		
Education					
Primary	3 (9.1)	3(9.1)		0.167	0.920
Secondary	18 (54.5)	19(57.6)			
Tertiary	12 (36.4)	11(33.3)			

Key: SCD-Sickle cell disease, SCLU: Sickle cell leg ulcer, HbAA: Haemoglobin AA

Table 2: History of sickle cell leg ulcer

	SCD SCLU n = 33
Leg ulcer	
Duration from onset (in years)	
Median	2.0
IQR	1.0 – 2.0
Distribution	
Right	22 (66.7)
Left	7 (21.2)
Both	4 (12.1)
Ulcer Symptoms	
Pain	13 (39.4)
Swelling	3 (9.1)
Discharge	1 (3.0)
Tingling sensation	1 (3.0)
Stage of ulcer	
II	14 (42.4)
III	19 (57.6)

Key: SCD-Sickle cell disease, SCLU: Sickle cell leg ulcer

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Table 3: Sickle cell disease severity indices

Disease severity indices	SCD SCLU n = 33
Frequency of VOC/year	
<3	23 (69.7)
≥3	10 (30.3)
Number of admissions/years	
<3	31 (93.9)
≥3	2 (6.1)
Last Transfusions	
<3months	15 (45.5)
≥3months	18 (54.5)

Key: SCD-Sickle cell disease, SCLU: Sickle cell leg ulcer

Table 4: Comparison of Haematological parameters between SCLU patients and SCA controls

Haematological Parameters	SCA SCLU (n = 33) Mean ± SD	SCA Control (n = 33) Mean ± SD	P-value
WBC (x10^9/l)	12.9±7.3	9.7±3.6	0.027
ANC (x10^9/l)	7.1±5.2	5.2±2.2	0.058
ALC (x10^9/l)	4.8±2.2	3.6±1.3	0.009
Monocytes (x10^9/l)	0.7±0.4	0.8±1.1	0.573
Haematocrit (%)	24.8±4.5	26.5±4.8	0.148
Platelet count (x 10^9/l)	296.8±138.7	272.5±82.7	0.391
Stable HCT (%)	24.0±4.0	24.4±2.7	0.648
Prothrombin time(s)	16.6±1.2	16.5±1.7	0.869
APTT(s)	45.0±3.4	40.9±4.4	0.001

Key: SCD-Sickle cell disease, SCLU: Sickle cell leg ulcer, APTT: Activated partial thromboplastin time, ANC: Absolute neutrophil count, ALC: Absolute lymphocyte count

The mean (SD) white blood cell count in the SCA SCLU group was higher than that of the SCA controls (12.9±7.3 x 10^9/L versus 9.7±3.6 x 10^9/L, p = 0.027) . Similarly, the mean absolute neutrophils count (7.1±5.2 x 10^9/L vs. 5.2±2.2 x 10^9/L, p = 0.058) and mean absolute lymphocyte counts (4.8±2.2 x 109/L vs. 3.6±1.3 x 109/L, p = 0.009) were higher in the SCA SCLU group (Table 4). The mean monocyte count was however lower in the SCA SCLU group than in the SCA control group (0.7±0.4 x10^9 /L vs 0.8±1.1 x 10^9 /L) but the difference in mean was not statistically significant (p=0.573).

The mean haematocrit in the SCA SCLU group was 24.8±4.5% and the SCA controls 26.5±4.8 % and the mean difference was not statistically significant (P=0.148). The mean platelet count was higher in the SCA SCLU group (296.8±138.7x10^9 /L vs 272.5±82.7x10^9/L, p=0.391). (Table 4).

The mean prothrombin time in the SCA SCLU group was 16.6 ±1.2secs. and in the SCA control 16.5±1.7 secs. The mean difference was not statistically significant (p= 0.869). The mean Activated prothrombin time was higher in SCA SCLU compared to SCA controls (45 ± 3.4secs versus 40.9± 4.4secs) and the mean difference was statistically significant, p= 0.001. (Table 4)

DISCUSSION

This study did not notice a significant gender preference in developing leg ulcers in SCD patients. This is converse to initial findings by Bazuaye et al. in UBTH and previous studies in other centers.[7,11-13] About half of the patients with leg ulcers in this study were between ages 25-29 years. This finding was similar to studies done

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in by Bazuaye et al. and Idaewor et al.[7,14] In- lieu to these studies, the index study noted a decrease in the incidence of leg ulcers with increasing age. This could be explained by the fact that this age group is very active and thus prone to injuries. The proportion of SCD CLU patients who attained tertiary level of education was similar to SCD controls HbAA controls. This is suggestive that affectation with CLU was not a negative predictor to maximal educational attainment.

The major site affected by ulcers in this study was the skin around the medial malleoli. This is consistent with earlier reports by Durosinmi et al.[11] This finding is also in concordance with that of Hassan et al. in northern Nigeria and Santos et al. in Brazil.[15,16] The distribution of less subcutaneous fat, thin skin and decreased blood flow to the region may underlie the increased risk of ulceration observed in the anatomic site.[15]

The right lower limb was the most frequently affected. This is contrary to reports by Bazuaye et al., Idaewor et al. and Akinyanju et al. who noted dominance of the left.[7,13,14] Expectedly, the right limb is most dominant in majority of the population, hence it is most actively engaged in activities which thus increases the risk of injury.

The major presenting complaint from participants with leg ulcers in this study was pain at the ulcer site. This was consistent with findings from a study by Halabi-Tawil et al. among French respondents.[17] In their study, they reported that ninety percent of patients with leg ulcers needed analgesics generally while about 20% needed opioids to relieve severe pains. In a similar study involving ninety-eight patients with sickle cell leg ulcers, all patients required painkillers and 32% took opioids.[17] Moreover, a longitudinal observational study on 450 subjects with chronic wounds of different causes showed that opioid use was associated with a reduced likelihood of their chronic wounds healing.[18]

Majority of the patients in this study had less than three episodes of vasoocclusive crisis (69.7%) and hospital admissions (93.9%) per year. This contrasts with expectations, that with increased level of inflammatory markers, endothelium disruption and reduced steady state PCV patients should experience more crises that would warrant more hospital admission.[19] The reason for this is not clear but could be linked with the management option of using chronic blood transfusion for sickle cell patients with leg ulcers. This management option allows for proper perfusion of tissues and improved steady HCT concentration.

The mean WBC and its differentials were significantly higher in respondents with chronic leg ulcers than those with stable disease. This is similar to the studies by Nolan et al., Hassan et al. and Babalola et al. who reported the same trend in the United States of America (USA), Zaria and Ibadan respectively.[10,20,21] The difference in the level of WBC between the two groups could reflect an increased haematopoiesis associated with haemolysis and/or due to microbial colonization or infection of ulcers.[20]

The mean haematocrit (HCT) of leg ulcer patients in this study was lower than those of the control (SCA) group, however this was not statistically significant. This was consistent with similar studies by Babalola and Hassan et al. and Bazuaye et al.[10,21] As reported in these studies, the index study also noted a lower stable HCT level of the sickle cell leg ulcer population in comparison to the stable SCD group.[7,10,21] Chronic haemolysis is one of the major causes of reduced steady state haemoglobin in non-leg ulcer controls. Studies have shown that sickle cell anaemia patients with a low steady-state HCT are at increased risk of developing chronic leg ulcers and the HCT is a marker of the severity of haemolysis.[20]

The sickle cell leg ulcer group had higher number of platelets than the control (SCD) group. This finding was similar to studies by Cummings, Babalola and Hassan et al. in their different works.[10,21,22] Hypercoagulability contributed by elevated platelet count may have a possible role in the causation of leg ulcer formation as it contributes to skin ischemia, resulting in friability and ulceration.[23] Sickle cell ulcers are characterized partly by an increase in clotting ability as a result of increased platelets, hypercoagulability and a measured increase in clotting factors at the wound itself.[23] Babalola et al. in their study noted that platelet was positively correlated with wound size.[10] Some authors, including Wirth et al, assert that an efficient decrease in the concentration of platelets may be therapeutic in the management of ulcers.[24]

CONCLUSION

The prevalence of leg ulcers in SCD reduced with increasing age. There was no association between level of education and leg ulcers in SCD. Most leg ulcers occurred on the right leg on the medial malleolus and platelet count was significantly higher in patients with chronic leg ulcers compared those in steady state.

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Declaration

Conflict of Interest: There was no conflict of interest between the authours in this study

REFERENCES

1. Adewoyin AS. Management of sickle cell disease: a review for physician education in Nigeria (sub-Saharan Africa). Anemia. 2015; 2015(1):791498.
2. Vos T, Allen C, Arora M, Barber R. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. The Lancet. 2016; 388 (10053): 1545-1602.
3. Serjeant GR. The natural history of sickle cell disease. Cold Spring Harb Perspect Med. 2013;3(10): a011783.
4. World Health Assembly, 59. (2006). Sickle-cell anaemia: report by the Secretariat. World Health Organization. Available from https://iris.who.int/handle/10665/20890
5. Nwogoh B, Adewoyin AS, Iheanacho OE, and Bazuaye GN. Prevalence of haemoglobin variants in Benin City, Nigeria. Annals of Biomed. Sci. 2012; 11(2): 60–64.
6. Minniti CP, Eckman J, Sebastiani P. Leg ulcers in sickle cell disease. AJH 2010; 85(10): 831-833.
7. Bazuaye GN, Nwannadi AI, Olayemi EE. Leg Ulcers in Adult sickle cell disease patients in Benin City, Nigeria. GJMS 2010; 8(2):190–194.
8. Umeh NI, Ajegba B, Buscetta AJ, Abdallah KE. The psychosocial impact of leg ulcers in patients with sickle cell disease: I don't want them to know my little secret. PLoS One. 2017; 12(10):e0186270.
9. Habibi A, Maryse E, Emmanuelle B, Maria D, Pagona F, Ersi V. Leg ulcers in sickle cell disease patients undergoing hydroxyurea therapy:insights from two large cohort studies. Blood. 2023;142(1):2500
10. Babalola OA, et al. Haematological indices of sickle cell patients with chronic leg ulcers on compression therapy. Afr J Lab Med. 2020; 9(1):1037.
11. Durosinmi MA, Gevao SM, Esan GI. Chronic leg ulcers in sickle cell disease: experience in Ibadan, Nigeria. Afr. J.Med.Sci. 1991; 20(1): 11-14.
12. Koshy M, Entsuah R, Koranda A. Leg Ulcers in Patients with Sickle Cell Disease. Blood.1989;74(4):1403-1408.
13. Akinyanju O, Akinsede I. Leg ulceration in sickle cell disease in Nigeria. Trop Geogr Med. 1979;31(1):87-91.
14. Idaewor PO, Enosolease MC, Momoh MI. Leg ulceration in a population of Nigerian patients with sickle cell anaemia – twenty years' experience. Int Med Biomed Res.2002;1(1):18 -21.
15. Hassan A, Gayus DL, Abdulrasheed I, Umar MA, Ismail DL, Babadoko AA. Chronic leg ulcers in sickle cell disease patients in Zaria, Nigeria. Arch Int Surg. 2014; 4(3): 141–145.
16. Santos EDC, Santana PVB, Jesus LLS, Melo GIV, Yahouédéhou SCMA, Guarda CCD, Santiago RP. Leg Ulcers in Sickle Cell Disease: A Multifactorial Analysis Highlights the Hemolytic Profile. Hematol Rep. 2023;15(1):119-129.
17. M. Halabi-Tawil F, Lionnet R..Girot C, Bachmeyer PP, Levy S. Sickle cell leg ulcers: a frequently disabling commplication and a marker of severity. Br J Dermatol. 2008; 158(2): 339-344.
18. Hanmugam VK, Couch KS, McNish S, Amdur RL. Relationship between opioid treatment and rate of healing in chronic wounds. WRR. 2017;25(1):120–130.
19. Urhie OO, Awodu O, Okuonghae ME, Dirisu MI, Awotiku OO, Ibhayehor JO. Evaluation of Vonwilliebrand factor levels in sickle cell disease patient with leg ulcer in South-South Nigeria. World journal of pharmaceutical research 2026; 15(5): 803-816
20. Nwagha T.U, Nweke M & Ezigbo E.D. Contributions of von Willebrand factor to clinical severity of sickle cell disease: a systematic review and metanalysis. Hematology. 2022; 27(1):860-866.
21. Omer NE, Satti MH, Mohammed AO. Plasma level of von Willebrand factor: An indicator of severity in sickle cell disease. Sudan J.med.sci. 2009;4(2).
22. Monfort JB, Senet P. Leg Ulcers in Sickle-Cell Disease: Treatment Update. Adv Wound Care (New Rochelle).2020; 9(6):348-356.
23. Cumming V, King L, Fraser R, Serjeant G, Reid M. Venous incompetence, poverty and lactate dehydrogenase in Jamaica are important predictors of leg ulceration in sickle cell anaemia. Br J Haematol. 2008;142(1):119–125.
24. Madu AJ, Ubesie A, Madu KA, Okwor B, Anigbo C. Evaluation of clinical and laboratory correlates of sickle leg ulcers. WRR. 2013; 21(6) :808-812.

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