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Calcium–Phosphorus Metabolism in Patients with Sickle Cell Disease: A Study from Yaoundé, Cameroon

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Abstract

Background

Calcium-phosphorus disorders are an underestimated source of morbidity in sickle cell patients in sub-Saharan Africa. This study aimed to evaluate the biological parameters of calcium-phosphorus metabolism in sickle cell patients treated in Yaoundé, Cameroon.

Objective: To evaluate the biological parameters of calcium-phosphorus metabolism in homozygous sickle cell patients monitored in Yaoundé.

Methodology: We conducted a cross-sectional analytical study from November 2023 to August 2024 among patients monitored in Yaoundé. We measured and confirmed the haemoglobin phenotype and measured parathyroid hormone (unfractionated) and total serum vitamin D, calcium, phosphorus and serum albumin. Associations between variables were sought using linear regression and odds ratio (OR) calculations with a 95% confidence interval (95% CI). The significance threshold was set at 5%, and a p-value of less than 0.05 was therefore considered statistically significant.

Results: Of the 86 participants, the median age was 16 years [9;20], 46% were aged between 15 and 29, 65% (n=54) were male, with a male-to-female ratio of 1.2. The majority (66, 76.74%) had high ALP and hyperphosphataemia, i.e. 61 (70.9%) with moderate vitamin D deficiency in 73% (n=63). We found that being under 16 years of age was significantly associated (p=0.026) with a 72% reduction in the risk of hypercalcaemia. No factors were associated with hypocalcaemia, hyperphosphataemia, hypophosphataemia or vitamin D deficiency. A statistically significant positive correlation was found between serum phosphorus levels and bone remodelling (p = 0.00; Rho = 0.48) and a significant negative correlation between age and bone remodelling (p = 0.01; Rho = -0.26).

Conclusion: This preliminary study shows that subjects with homozygous sickle cell disease had hyperphosphataemia, hypovitaminosis D and elevated alkaline phosphatase levels, the early detection of which could reduce the frequency and severity of complications.

morbidity and mortality. This study illustrates the need for regular monitoring of these biochemical parameters in individuals with sickle cell disease.

Background

Sickle cell disease, first described by Herrick in 1910, is the most common genetic disorder in the world [1]. It is a major global public health issue due to its high morbidity and mortality rates worldwide [1]. It results from a single mutation on chromosome 11, a change from glutamic acid to valine, leading to the replacement of beta globin chains (also known as A chains) by S chains with polymerisation capabilities [2]. In various situations such as hypoxia, acidosis, dehydration and inflammation, this polymerisation causes red blood cells to deform (sickling), leading to venular vaso-occlusion and haemolytic anaemia [3]. Haemolytic processes, vaso-occlusive processes, disruption of the activity of certain hormones

(calcitonin and parathyroid hormone), vitamin D, and enzymes (alkaline phosphatase) involved in bone metabolism during the disease are the factors responsible for the calcium-phosphorus metabolism disorders reported in individuals with homozygous sickle cell disease (HbSS) [4]. In sub-Saharan Africa, there is little data available describing variations in the biochemical parameters of calcium-phosphorus metabolism in individuals living with homozygous sickle cell disease [5]. To our knowledge, there is none in our context. Hence the interest of this study, which aimed to evaluate the biological parameters of calcium-phosphorus metabolism in homozygous sickle cell patients monitored in Yaoundé, with a view to improving the assessment and management of sickle cell patients in our context.

METHODOLOGY

Study setting

This was a cross-sectional, analytical study conducted across three healthcare facilities in Yaoundé (Cameroon) over a period of ten months from November 2023 to August 2024. Participants were recruited from the haematology unit of Yaoundé Central Hospital (HCY) and the paediatric outpatient unit of the Chantal BIYA Foundation Mother and Child Centre (CME/FCB). The University Teaching Hospital of Yaounde (UTHY) played a dual role, serving as a recruitment site for participants in the outpatient haematology unit and as a location for all biochemical analyses carried out in the institution's biochemistry laboratory.

Study population

The study population consisted of homozygous sickle cell patients residing in Yaoundé and actively followed up at the designated study sites. Inclusion criteria for this study included participants aged at least 12 months who provided free and informed consent, with parental consent for minors. Exclusion criteria included individuals with a heterozygous sickle cell genotype (AS, SC, S-β thalassaemia), those with pre-existing chronic kidney or liver disease, and those with haemolysed or lipaemic blood samples.

Sampling

To determine the minimum sample size, we used Cochran's formula. This calculation determined a minimum sample size of 60 participants. Recruitment was carried out using consecutive sampling, enrolling all eligible participants encountered during the study period.

Materials and data collection : We were interested in the following data: sociodemographic parameters (age, gender, level of education), clinical parameters (age at the time of sickle cell diagnosis, background treatments, prophylaxis, history of chronic vascular complications and comorbidities) and biological parameters (serum albumin, serum calcium, serum 25-hydroxyvitamin D, intact parathyroid hormone, serum phosphorus, haemoglobin electrophoresis and quantification, and complete blood count). Indicators of disease severity included the frequency of sickle cell crises per year, the number of hospitalisations per year, and the number of blood transfusions per year.

Statistical analysis : The data were initially entered and coded using CSPro version 6.0, then exported to IBM SPSS Statistics version 21.0 for statistical analysis. Continuous quantitative variables were summarised by their mean and standard deviation, while discrete or non-normally distributed variables were described using medians and interquartile ranges (IQR: 25th to 75th percentiles). The Kolmogorov-Smirnov test was used to assess the normality of the data distribution. Categorical variables were presented as frequencies and corresponding proportions. Factors associated with hypovitaminosis D were studied using Fisher's exact test, followed by multivariate logistic regression analysis to adjust for potential confounding variables. The strength and direction of associations were quantified using odds ratios with 95% confidence intervals. Median comparisons of vitamin D, serum calcium, serum phosphorus, alkaline phosphatase (ALP), and parathyroid hormone (PTH) levels between groups were performed using the Mann-Whitney U test. Spearman's rank correlation coefficient was used to assess the impact of vitamin D, serum calcium, serum phosphorus, ALP, and PTH concentrations on the risk of calcium-phosphorus disorders. The results are presented with the corresponding beta coefficients and statistical significance levels (p-value < 0.05).

Ethics

This research was conducted in accordance with the ethical principles set out in the 1964 Declaration of Helsinki, revised in October 2013. The necessary authorisations were obtained from the relevant institutions. Informed consent was obtained from all participants. All data collected during the study were kept strictly confidential and used exclusively for scientific research purposes.

RESULTS

We had a total of 86 participants, comprising 45 (35%) women and 54 (65%) men, giving a male-to-female ratio of 1.2. The age of participants ranged from 1 to 52 years, with a median age of 16 years (interquartile range: 9–20 years). The cohort had a bimodal age distribution, with 35% of participants belonging to the paediatric age group (1–14 years) and 46% were adolescents/young adults (aged 15–29). Patients had secondary education (42, or 48.8%), primary education (21, or 24.4%), higher education (15, or 17.4%) or no education (8, or 9.3%).

Clinical data

The only comorbidity reported among participants was diabetes, affecting 2 (2.3%). The median age of participants at the time of sickle cell diagnosis was 4 years [2;6]. The circumstances in which sickle cell disease was discovered were hand-foot syndrome 54 (62.8%), severe anaemia 17 (19.8%), recurrent bone pain 5 (5.8%), recurrent infections 5 (5.8%), jaundice with dark urine 3 (3.5%), and screening 2 (2.3%). At least twice a year, patients had at least two crises and were hospitalised (70%). Complications included leg ulcers 4 (4.7%), stroke 2 (2.3%) and osteonecrosis of the hip 2 (2.3%). Treatment consisted of folic acid 54 (62.8%), folic acid + hydroxyurea 32 (37.2%)

Biological data

In terms of calcium-phosphorus metabolism disorders, the majority (66, or 76.74%) of patients had elevated ALP, and hyperphosphataemia was found in 61 (70.9%) of patients (Table 1). There was a statistically significant association (p = 0.026) between patient age under 16 years and the presence of hypercalcaemia. An odds ratio of 0.28 indicates that age under 16 years is associated with a 72% reduction in the risk of hypercalcaemia compared to participants aged 16 years or older.

Two other factors could be associated with hypercalcaemia: high PTH levels and vitamin D deficiency. Although this association is not statistically significant (p = 0.19), it suggests that participants with high PTH levels have a risk of developing hypercalcaemia (82% and 58% respectively) (Table 2). No factors were associated with hypocalcaemia (Table 3), hyperphosphataemia (Table 4) or hypophosphataemia (Table 5). Vitamin D was found in normal amounts (4%), in excess (1%), in moderate deficiency (73%), insufficient (16%) and severe deficiency (6%). No factors were associated with vitamin D deficiency (Table 6). With regard to phosphocalcic disorders affecting bone remodelling through ALP, a statistically significant correlation was found between serum phosphorus levels and bone remodelling (p = 0.00; Rho = 0.48) (Table 7). This positive correlation suggests an increase in phosphorus levels in parallel with those of PAL (Fig. 1). The same was true for age and bone remodelling; however, this was a negative correlation, suggesting a decrease in bone remodelling with increasing patient age (p = 0.01; Rho = -0.26) (Fig. 2).

Table 1
Distribution of calcium and phosphorus disorders (N = 86).

Variables	**Number (n)**	**Percentage (%)**
Corrected calcium level		
Hypercalcemia	17	19,8
Hypocalcemia	6	7
Phosphorus		
Hyperphosphoremia	**61**	**70,9**
Hypophosphoremia	2	2,3
ALP		
High	66	76,74
PTH		
High	1	1,2
Low	15	17,4

Table 2
Factors associated to hypercalcemia(N = 86).

Variables		Hypercalcemia		OR [IC 95%]	P value
		Yes (n = 17)	**No (n = 69)**		
Age (years)					
	< 16	10(33,3)	20(66,7)	0,28[0,09;0,86]	**0,026**
	≥ 16	7(12,5)	49(87,5)	1	
Gender					
	Masculine	10(21,3)	37(78,7)	0,80[0,27 ; 2,37]	0,78
	Feminine	7(17,9)	32(82,1)	1	
Annual number of crises					
	≥ 2	13(19,1)	55(80,9)	0,82[0,23 ; 2,93]	0,74
	< 2	4(22,2)	14(77,8)	1	
Annual number of hospitalisations					
	≥ 2	9(17,6)	42(82,4)	0,72[0,25 ; 2,10]	0,37
	< 2	8(22,9)	27(77,1)	1	
Annual number of blood received					
	≥ 2	0(0)	0(0)	-	-
	< 2	17(19,8)	69(80,2)	1	
Underweight / Denutrition					
	Yes	8(13,8)	19(67,9)	0,33[0,11 ; 1,00]	0,80
	No	9(32,1)	50(86,2)	1	
Severe anaemia					
	Yes	17(19,8)	69(80,2)	-	-
	No	0(0)	0(0)	1	
Hypophosphatemia					

Variables		Hypercalcemia		OR [IC 95%]	P value
		Yes (n = 17)	No (n = 69)		
	Yes	0(0)	2(100)	0,79[0,71 ; 0 ,88]	1
	No	17(20,2)	67(79,8)	1	
Hyperphosphatemia					
	Yes	12(19,7)	49(80,3)	0,98[0,30 ; 3,14]	1
	No	5(20)	20(80)	1	
High PTH					
	Yes	1(100)	0(0)	0,18[0,12 ; 0,29]	0,19
	No	16(18,8)	69(81,2)	1	
vitamine D deficiency					
	yes	11(16,4)	56(83,6)	0,42[0,13 ; 1,36]	0,19
	No	6(31,6)	13(68,4)	1	

Table 3
Factors associated with hypocalcemia(N = 86).

Variables	Hypocalcemia		OR [IC 95%]	P value
	Yes (n = 6)	No (n = 80)		
Age (years)				
< 16	0(0)	30 (100)	1,12 [1,02 ; 1,22]	0,08
≥ 16	6(10,7)	50 (89,3)	1	
Gender				
Masculine	3(6,4)	44 (93,6)	1,22 [0,23 ; 6,4]	1
Feminine	3(7,7)	36 (92,3)	1	
Annual number of crises				
≥ 2	4(5,9)	64(94,1)	0,5[0,08 ; 2,97]	0,60
< 2	2(11,1)	16(88,9)	1	
Annual number of hospitalisation				
≥ 2	3(5,9)	48(94,1)	0,66[0,12 ; 3,51]	0,68
< 2	3(8,6)	32(91,4)	1	
Annual number of blood received				
≥ 2	0(0)	0(0)	-	-
< 2	6(7)	80(93,0)	1	
Underweight/denutrition				
Yes	4(6,9)	54(93,1)	0,96[0,16 ; 5,60]	1
No	2(7,1)	26(92,9)	1	
Severe Anaemia				
Yes	6(7,0)	80(93 ,0)	-	-
No	0(0)	0(0)	1	
Hypophosphatemia				
Yes	0(0)	2(100)	0,92[0,87 ; 0,98]	1
No	6(7,1)	78(92,9)	1	
Hyperphosphatemia				
Yes	4(6,6)	57(93,4)	0,8[0,13 ; 4,71]	1

Variables	Hypocalcemia		OR [IC 95%]	P value
	Yes (n = 6)	**No (n = 80)**		
No	2(8,0)	23(92,0)	1	
High PTH				
Yes	0(0)	1(100)	0,92[0,87 ; 0,98]	1
No	6(7,1)	79(92,9)	1	
Vitamine D deficiency				
Yes	5(7,5)	62(92,5)	1,45[0,15 ; 13,23]	1
No	1(5,3)	18(94,7)	1	

Table 4
Factors associated with hyperphosphatemia (N = 86)

Variables	Hyperphosphatemia		OR [IC 95%]	P Value
	Yes (n = 61)	**No (n = 25)**		
Age (Years)				
< 16	22(73,3)	8(26,7)	0,83[0,31 ; 2,24]	0,80
≥ 16	39(69,6)	17(30,4)	1	
Gender				
Masculine	37(78,7)	10(21,3)	0,43[0,16 ; 1,11]	0,09
Féminine	24(61,5)	15(38,5)	1	
Phenotype				
Annual number of crises				
≥ 2	50(73,5)	18(26,5)	1,76[0,59 ; 5,2]	0,38
< 2	11(61,1)	7(38,9)	1	
Annual number of hospitalisation				
≥ 2	34(66,7)	17(33,3)	0,59[0,22 ; 1,58]	0,34
< 2	27(77,1)	8(22,9)	1	
Annual number of blood received				
≥ 2	0(0)	0(0)	-	-
< 2	61(70,9)	25(29,1)	1	
Underweight/ denutrition				
Yes	42(72,4)	16(27,6)	1,24[0,46 ; 3,31]	0,80
No	19(67,9)	9(32,1)	1	
Severe anaemia				
Yes	61(70,9)	25(29,1)	-	-
No	0(0)	0(0)	1	
Hypocalcemia				
Yes	4(66,7)	2(33,3)	0,05[0,13 ; 4,71]	1
No	57(71,3)	23(28,7)	1	
Hypercalcemia				

Variables	Hyperphosphatemia		OR [IC 95%]	P Value
	Yes (n = 61)	**No (n = 25)**		
Yes	12(70,6)	5(29,4)	0,98[0,30 ; 3,14]	1
No	49(71,0)	20(29,0)	1	
High PTH				
Yes	1(100)	0(0)	0,70[0,61 ; 0,81]	1
No	60(70,9)	25(29,4)	1	
vitamine D deficiency				
Yes	49(73,1)	18(26,9)	1,58[0,54 ; 4,66]	0,40
No	12(63,2)	7(36,8)	1	

Table 5
Factors associated with l'hypophosphatemia (N = 86).

Variables	Hypophosphatemia		OR [IC 95%]	P Value
	Yes (n = 2)	**No (n = 84)**		
Age (years)				
< 16	1(3,33)	29(96,7)	0,52[0,032 ; 8,74]	1
≥ 16	1(1 ; 8)	55(98,2)	1	
Gender				
Masculine	0(0)	47(100)	1,05[0,98 ; 1,13]	0,20
Féminine	2(5,1)	37(94,9)	1	
Annual number of crises				
≥ 2	2(2,9)	66(97,1)	1,03[0,98 ; 1,07]	1
< 2	0(0)	18(100)	1	
Annual number of hospitalisation				
≥ 2	1(2)	50(98,0)	0,68[0,04 ; 11,24]	1
< 2	1(1,9)	34(97,1)	1	
Annual number of blood received				
≥ 2	0(0)	0(0)	-	-
< 2	2(2,3)	84(97,7)	1	
Underweight/denutrition				
Yes	2(3,4)	56(96,6)	1,03[0,98;1,08]	1
No	0(0)	28(100)	1	
Severe anaemia				
Yes	2(2,3)	84(97,7)	-	-
No	0(0)	0(0)	1	
Hypocalcemia				
Yes	0(0)	6(100)	0,97[0,93 ; 1,01]	1
No	2(2,5)	78(97,5)	1	
Hypercalcemia				
Yes	0(0)	17(100)	0,97[0,93 ; 1,01	1

Variables	Hypophosphatemia		OR [IC 95%]	P Value
	Yes (n = 2)	**No (n = 84)**		
No	2(2,9)	67(97,1)	1	
High PTH				
Yes	0(0)	1(100)	0,97[0,94 ; 1,00]	1
No	2(2,4)	83(97,6)	1	
vitamine D deficiency				
Yes	1(1,5)	66(98,5)	0 ,27[0,01 ; 4,57]	0 ,39
No	1(5,3)	18(94,7)	1	

Table 6
Factors associated to vitamine D deficiency (N = 86).

Variables	vitamine D deficiency		OR [IC 95%]	P Value
	Yes (n = 63)	No (n = 23)		
Age (years)				
< 16	26(86,7)	4(13,3)	0,42[0,12 ; 1,40]	0,18
≥ 16	41(73,2)	15(26,8)	1	
Gender				
Masculine	38(80,9)	9(19,1)	0,68[0,24 ; 1,90]	0,60
Féminine	29(74,4)	10(25,6)	1	
Annual number of crises				
≥ 2	55(80,9)	13(19,1)	2,11[0,66 ; 6,69]	0,21
< 2	12(66,7)	6(33,3)	1	
Annual number of hospitalisation				
≥ 2	43(84,3)	8(15,7)	2,46[0,87 ; 6,96]	0,11
< 2	24(68,6)	11(31,4)	1	
Annual number of blood received				
≥ 2	0(0)	0(0)	-	-
< 2	67(77 ,9)	19(22 ,1)	1	
Underweight/denutrition				
Yes	47(81,0)	11(19,0)	1,7[0,59 ; 4,88]	0,40
No	20(71,4)	8(28,6)	1	
Severe anaemia				
Yes	67(77,9)	19(22,1)	-	-
No	0(0)	0(0)	1	
Hypocalcemia				
Yes	5(83,3)	1(16,7)	1,45[0,15 ; 13,23]	1
No	62(77,5)	18(22,5)	1	
Hypercalcemia				

Variables	vitamine D deficiency		OR [IC 95%]	P Value
	Yes (n = 63)	No (n = 23)		
Age (years)				
Yes	11(64,7)	6(35,3)	0,42[0,13 ; 1,36]	0,19
No	56(81,2)	13(18,8)	1	
Hypophophoremia				
Yes	1(50)	1(50)	0,27[0,01 ; 4,57]	0,39
No	66(78,6)	18(21,4)	1	
Hyperphosphatemia				
Yes	49(80,3)	12(19,7)		
No	18(72,0)	7(28,0)	1	
High PTH				
Yes	1(100)	0(0)	0,77[0,69;0,87]	1
No	66(77,6)	19(22,4)	1	

Table 7
Study of calcium and phosphorus disorder in bone remodelling through ALP (N = 86).

ALP		
	Rho	**P Value**
Calcium	-0,06	0,54
Phosphorus		**0,00**
PTH	-0,75	0,49
Vit D	0,08	0,43
Haemoglobine count	0,13	0,23
Age	-0,26	**0,01**
BMI	-0,20	0,05

DISCUSSION

The overall objective of this study was to evaluate the biological parameters of calcium and phosphorus metabolism in homozygous sickle cell patients monitored in Yaoundé. Our study has several limitations

and difficulties: the heterogeneous definition of bone disease; we did not use a densitometer to clearly assess bone mineralisation defects; renal function was not assessed; the reduced quantity of reagents and their cost due to the fact that our financial resources came from ourselves. We did not receive any external financial support.

The patients ranged in age from 1 to 52 years, with a median age of 16 years. The most represented age group was [15; 30 [years (46%). These data show that the paediatric population, particularly adolescents, are the most affected by sickle cell disease. This is consistent with studies by Wastnedge et al. in 2018 and Egesa et al. in 2022 [6, 7]. This high prevalence among the young population could be explained by adults' reluctance to seek medical advice or expose themselves due to the frustrations, stigma and prejudice associated with this condition [8].

In terms of clinical data, the median age of diagnosis of sickle cell disease was 4 years [2.75; 6.00]. This result is comparable to that of Chemegni et al. in 2018 in Cameroon, who found that sickle cell disease was diagnosed and managed at an early age in Cameroon due to the existence of facilities and centres dedicated to the management and follow-up of sickle cell patients [9].

As for calcium-phosphorus metabolism disorders, the majority (66, or 76.74%) of patients had high ALP, and hyperphosphataemia was found in 61 (70.9%) of them. The hyperphosphataemia data are similar to those found by Batte et al. in 2023 in Nigeria, which suggested that children with sickle cell anaemia had hyperphosphataemia involving impaired renal reabsorption [10]. This is all the more true given that our participants commonly had low 25(OH) vitamin D levels. The role of the kidneys in phosphate metabolism in sickle cell disease needs to be studied further in order to elucidate the correlations observed in this study. Furthermore, given that normal parathyroid hormone levels were most commonly found in our participants, one possible reason for hyperphosphataemia could be related to the release of phosphate by red blood cells in chronic haemolytic conditions.

Alkaline phosphatase was elevated in our study. These results suggest insidious bone damage or damage associated with growth in our participants, most of whom were adolescents of growing age.

Vitamin D deficiency was found in the majority of cases (63% (73%)). These results are consistent with those of Mekone et al. in 2021 in Cameroon and AlJama et al. in 2018 in Saudi Arabia, who found a rate of 82% vitamin D deficiency [11, 12]. This could be explained by the fact that children spend most of their time fully covered by clothing and mostly at home (away from the sun). This deficiency can be attributed to factors such as the inhibitory effect on vitamin D synthesis and activity of high levels of fibroblast growth factor 23, potentially stimulated by hyperphosphataemia, as well as limited sun exposure due to cultural practices that emphasise covering the body and minimising sun exposure [13].

We found a statistically significant association ($p = 0.026$) between the age of participants (under 16 years) and the presence of hypercalcaemia, with an odds ratio of 0.28 indicating that being under 16 years of age was associated with a 72% reduction in the risk of hypercalcaemia compared to participants aged 16 years or older, probably related to the frequency of crises in childhood. Indeed, in

the critical phase, abnormalities in the red blood cell membrane with increased calcium permeability and calcium accumulation in the red blood cells of patients with sickle cell disease have been reported, and it has been suggested that there may be an alteration in the calcium-binding properties of the plasma membrane of sickle cell red blood cells, with a stronger binding affinity for sickle cells than for normal red blood cells, thus contributing to calcium imbalance. [4, 14, 15]

However, we also found no association between age under 16 and a low risk of hypocalcaemia (p = 0.08), phosphorus disorder (p = 0.09) and vitamin D deficiency (p = 0.18).

Among the biomarkers of calcium and phosphorus disorders, a statistically significant correlation was found between serum phosphorus levels and bone remodelling (p = 0.00; Rho = 0.48). This positive correlation suggests an increase in phosphorus levels with ALP. Indeed, alkaline phosphatase (ALP) produced by osteoblasts, located in their membrane and released into the bloodstream, is involved in the bone formation and mineralisation process by hydrolysing phosphate esters on the surface of osteoblasts, in order to obtain the high phosphate concentration necessary for bone mineralisation [16].

The same was true for age and bone remodelling, however this is a negative correlation suggesting a decrease in bone remodelling with increasing patient age (p = 0.01; Rho = -0.26). This could be explained by bone growth, and another theory suggests that overweight people force their skeleton to increase bone volume to support their weight. Without advocating the merits of obesity, it is therefore important to avoid being underweight, which predisposes people to bone remodelling disorders. [17]

CONCLUSION

This study is the first to investigate the biological parameters of calcium-phosphorus metabolism in a cohort of patients with homozygous sickle cell disease (HbSS) followed up in designated hospitals in Yaoundé. Hyperphosphataemia and elevated alkaline phosphatase levels were the most common calcium-phosphorus abnormalities. A significant proportion of participants had moderate vitamin D deficiency. Young age (≤ 16 years) was identified as a protective factor against hypercalcaemia. A significant positive correlation was observed between serum phosphorus levels and alkaline phosphatase levels, while a negative correlation was observed between age and alkaline phosphatase levels. These findings suggest that calcium-phosphorus metabolism is significantly impaired in patients with HbSS in Yaoundé. Furthermore, the study highlights the importance of age as a factor influencing certain aspects of calcium-phosphorus metabolism, particularly in relation to hypercalcaemia. These results underscore the need for comprehensive assessments of bone health and mineral metabolism in patients with HbSS, with a particular focus on vitamin D status and addressing potential contributing factors such as hyperphosphataemia.

Declarations

Author contributions

Kévin Brice Kollo Nzima, Baldagaï Ndeva, Jan René Nkeck, Annick Ndoumba Mintya: Designed the study, collected and analyzed data, and wrote the manuscript. Joachim Tchikapa, Moïse Henri Julien Nko'o, Manuela Audrey Eko, Antoine Kandeda Kavaye: Reviewed and edited the manuscript. Vicky Jocelyne Ama Moor: Supervised the research and critically reviewed the manuscript.

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Declarations Ethics approval : This study was approved by the Ethics Committee of Faculty of Medecine and Biomedical Sciences of University of Yaounde I (N°100/UYI/FMSB/VDRC/CSD), all patients provided written informed consent, and the study adhered to the principles outlined in the Declaration of Helsinki.

Consent for publication : Not applicable.

Competing interests : The authors declare no competing interests.

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Figures

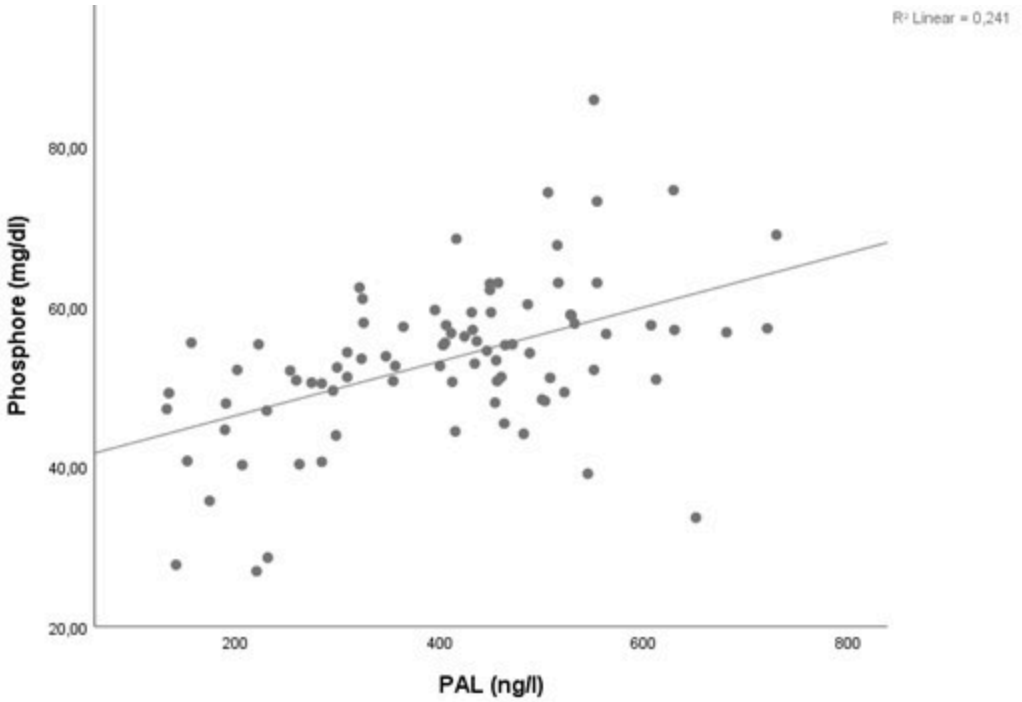


Figure 1

Correlation between ALP concentrations and phosphate concentrations.

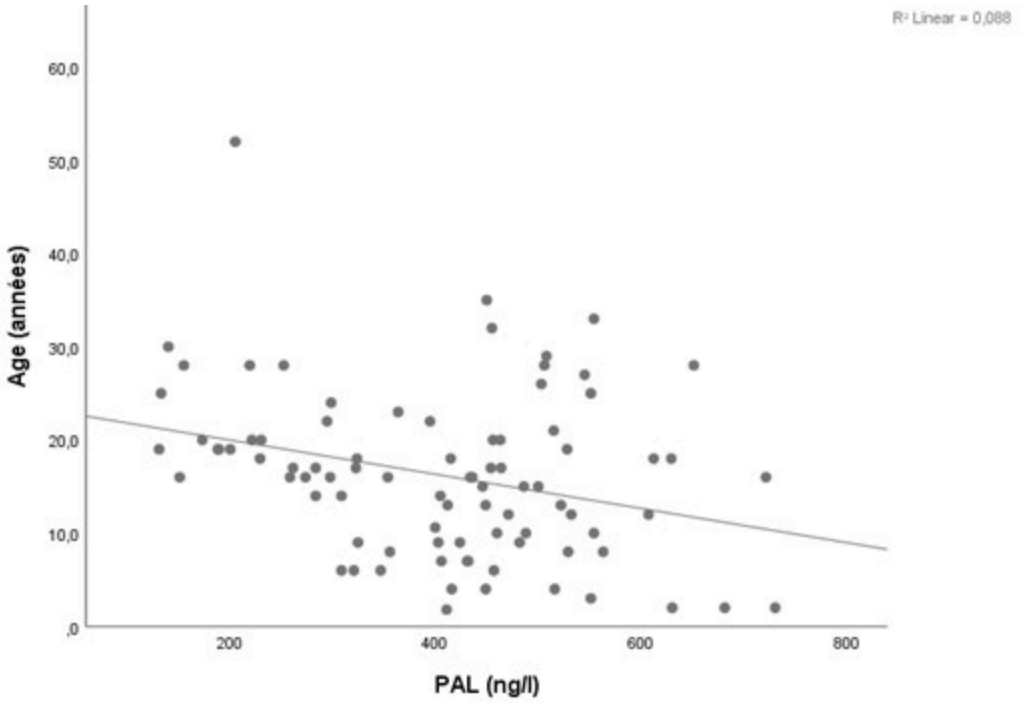


Figure 2

Correlation between ALP concentrations and ages.