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Predictive Factors of Renal Damage during Sickle Cell Disease at the Hematology-Oncology Department of Donka University Hospital

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Abstract

Introduction: Sickle cell disease, which is the most common hereditary hemoglobinopathy in the world, attacks all body systems, particularly the kidneys. The view of this study was to investigate the predictive factors of kidney damage during sickle cell disease. **Materials and methods:** It was a retrospective, descriptive and analytical study on files of sickle cell patients hospitalized in the Hematology-Oncology Department of Donka University Hospital during a period from January 1, 2016 to December 31, 2019. Records of sickle cell patients with one or more renal abnormalities were retained. Sickle cell patients without kidney damage were also selected for a comparative study. Only patients without sickle cell disease were excluded. Results: Seventy-five (75) medical records were collected during the study period. From these cases, thirteen (13) records with kidney disease were observed, a frequency of 17%. The mean age of patients was 24.2 years for extremes of 10 and 65 years. The sex ratio was 1.6 in favor of men. The $SSFA_2$ form was the most represented with 92%. 24-hour proteinuria was measured in 13 patients between whom 6 patients (46.2%) had a proteinuria level ≤ 1 g. Eight (8) patients (61.5%) were in stage 1 of chronic kidney disease. The most common type of renal involvement was tubulo-interstitial nephropathy with 8 patients (61.5%). Bivariate analysis showed that elevated serum creatinine ($P < 0.001$), elevated serum uremia ($P < 0.001$) and the $SSFA_2$ form of the sickness ($P < 0.003$) were the main factors linked with renal damage in sickle cell patients. **Conclusion:**

After the observation of an increased serum creatinine and urea, a predominance observation of the $SSFA_2$ form, it should be possible to target patients for whom screening for kidney damage should henceforth be systematic.

Keywords

Kidney Damage, Predictive Factors, Sickle Cell Disease, Donka

1. Introduction

Sickle cell disease is an autosomal recessive genetic hemoglobin disease resulting from a point mutation in the 6th codon of the globin gene. This mutation is responsible for abnormal hemoglobin S synthesis [1]. During sickle cell disease, rheological changes are the cause of numerous structural and functional abnormalities of the kidney, the basis of acute and chronic vaso-occlusive manifestations which progressively alter renal functions [2] [3]. These abnormalities, called "sickle cell nephropathy," are defined as a set of clinical and paraclinical signs indicating damage to renal structures, caused by complications of sickle cell disease. Systematic screening is therefore necessary for early treatment. However, screening for kidney damage could be done by looking for urinary markers and estimating the glomerular filtration rate [3] [4]. Serum creatinine measurements are traditionally used in clinical practice to assess and monitor renal function by calculating its clearance by various methods [5]. More recently, the search for microalbuminuria has become of paramount importance for the early detection of renal function abnormalities [6] [7].

In Cameroon in 2017, Nke Ateba G. *et al.* [8] reported on a sample of 111 homozygous SS sickle cell patients, a rate of 10.8% of cases of proteinuria for screening by urine strip and a rate of 16.22% of cases of proteinuria for measurement on a 24-hour urine sample.

In Mali in 2009, Fongoro S. *et al.* [9] reported that sickle cell disease was a factor in renal morbidity with a prevalence of acute kidney injury (AKI) estimated at 40%.

In Côte-d'Ivoire in 2016, N'Guessan AC *et al.* [10] reported 11% of cases of chronic kidney disease (CKD) in a study on 100 adults with major sickle cell disease.

In Guinea in 2019, Kanté AS *et al.* [11] reported that the prevalence of renal failure in sickle cell patients was 32.14%.

As the renal prognosis is often poor, particularly for the homozygous form of sickle cell disease, we decided to act upstream by identifying possible predictive factors of renal damage. This will make it possible in the future to provide earlier recommendations on additional examinations to be carried out.

2. Materials and Methods

This was a retrospective descriptive and analytical study covering the files of

sickle cell patients hospitalized in the Hematology-Oncology Department of the Donka National Hospital during the period from January 1, 2016 to December 31, 2019.

The files of sickle cell patients presenting one or more renal abnormalities were retained. Also, in order to compare before concluding, we retained the files of sickle cell patients without kidney damage. Thus, only non-sickle cell patients were excluded.

The anomalies taken into account were essentially:

—Urinary biochemistry and biology: proteinuria ± hematuria ± leukocyturia;

—Plasma biochemistry and biology: hypercreatininemia ± hypocalcemia ± anemia.

Hypercreatininemia was considered whenever the creatinine clearance calculated by the MDRD (Modification of Diet in Renal Disease) formula was <60 ml/min.

Hypocalcemia was considered in all cases of serum calcium < 2 mmol/L.

Anemia was classified as severe (hemoglobin level < 6 g/dL), moderate (hemoglobin level between 6 and 9 g/dL), and mild (hemoglobin level > 9 g/dL).

We did not take into account abnormalities in the size and structure of the kidneys on imaging, nor histological abnormalities.

Concerning the probable initial nephropathy:

—Glomerular origin: was defined by a proteinuria rate ≥ 1.5 g/24h. Other signs such as edema syndrome and high blood pressure were complimentary

—Vascular origin: proteinuria between 1 g and 1.2 g/24h. High blood pressure ± left ventricular hypertrophy ± hypertensive retinopathy were additional signs

—Tubulo-interstitial origin: proteinuria ≤ 1 g/24h ± leukocyturia.

Data was collected using individual survey sheets, patient consultation and hospitalization records, as well as medical records.

We performed a frequency distribution for the qualitative variables and we calculated the mean and the standard deviation for the quantitative variables.

The information obtained was used for a purely scientific purpose and the confidentiality of each patient was respected.

Data entry, and conception of tables have been done on the 2016 office pack and the analysis was performed on Epi info software, version 7.2.4.0. The statistical test used was chi square, significant from $p < 0.05$.

3. Results

This retrospective descriptive study, carried out in the Hematology-Oncology department of Donka University Hospital, over a period of 4 years, included, according to the criteria defined in the methodology, seventy-five (75) files of sickle cell patients. Among them, thirteen (13) cases of kidney damage were observed, representing a frequency of 17.33%. The average age of patients with renal damage was 24.2 years with an extreme of 10 and 65 years. The 19 - 29 age group was the most represented with 38.46% of patients presenting kidney

damage. The sex ratio was 1.68 in favor of men. Pupils and students predominated with 54%. The $SSFA_2$ form was the most represented among those with kidney disease (92.31%).

The estimation of GFR (glomerular filtration rate) by the MDRD formula allowed us to note a predominance of stage I of chronic kidney disease with 8 patients (61.54% of the 13 renal damage patients). The most common renal involvement was tubule-interstitial nephropathy with 6 patients (46.15%). Bivariate analysis showed that elevated serum creatinine ($P < 0.001$), elevated serum urea ($P < 0.001$), and predominance of the $SSFA_2$ form of the disease ($P < 0.003$) were factors associated with renal damage in sickle cell patients.

Average age: 24.2 ± 18.91 years Extremes: 10 and 65 years

The most represented age group was that of 19 - 29 years old with 28 cases of free kidney group (37.33%) and 5 cases of kidney damage carriers (6.67%).

The mean age of our sample was 24.2 ± 18.91 years.

For patients with renal damage, according to the initial nephropathy based on the 24 h protenuria, as defined in methodology, tubulo-interstitial damage was the most common shared with 6 cases or 46.15%. It was followed by vascular (30.77%), then glomerular (23.08%).

For hemoglobin rate, all but one of our kidney patients had 6 g/dL (92.31%). The average hemoglobin rate was 5.2 ± 2.7 g/dL.

For calcemia, 8 of 13 patients had hypocalcemia (61.54%) vs 5 cases of normal calcemia.

7 patients had high uremia (53.85%) and there were 2 cases not rated, because unreadable.

According to renal function, 12 patients out of 13 had high creatininemia (92.31%) vs one case of normal.

About variables presumed to be predictive factors of renal damage, bivariate analysis found "p" not significant for sex, nor for the type of presumed nephropathy.

Furthermore "p" was significant for the $SSFA_2$ form of sickle cell disease (0.003), as well as the high creatininemia (0.001) and the high uremia (0.001).

4. Discussion

This study which was carried out in 2019 was delayed in its publication following specific events occurring in West Africa (including Guinea), as well as at the global level. These events were the Ebola virus disease (EVD) epidemic and the coronavirus disease 2019 (COVID-19) pandemic, circumstances which resulted in, among other things, the reduction of social contacts with the confinement of the population, economic difficulties, overload of activities for medical workers, etc.

Indeed, between 2014 and 2016, a large epidemic of EVD occurred in West Africa, constituting a first wave for the Republic of Guinea. A second wave will occur for this country on February 14, 2021. Guinea was hit alone until Septem-

ber 2021, the official date of declaration of the end of the EVD epidemic [12] [13].

But before the end of this second wave of EVD, the first cases of COVID-19 appeared in Guinea on March 12, 2020. This pandemic, which caused a major health crisis, appeared on December 31, 2019 in China and was declared by the World Health Organization (WHO) on January 20, 2020 as a public health emergency of global concern. The end of its severity was only officially decreed on May 5, 2023 by the director of the WHO [14] [15].

With all the details above, it is easy to understand why there was a delay in publishing this article, the investigation of which concluded in December 2019.

Furthermore, it should be noted that this study encountered difficulties and limitations. Among other things, the poor conservation of certain medical records and the insufficiency of certain data.

During this work, we identified 75 files of sickle cell patients; 13 (thirteen) among them had renal damage, so a frequency of 17.33% (**Table 1**). This result is higher than the one found by Sri LH *et al.* [16] in Washington in 2015, who reported a frequency of 11%. Renal damage in sickle cell patients, according to the literature, could be explained by papillary necrosis, renal glomerulosclerosis and microalbuminuria/proteinuria, all due to complications arising from occlusions caused by the presence of sickled red blood cells in the vasa-recta and peritubular capillaries [17] [18].

Table 1. Distribution of sickle cell patients according to age range.

Age range	Renal damage (−)	Renal damage (+)	TOTAL
]9 - 19]	21 (28%)	3 (4%)	24 (32%)
]19 - 29]	28 (37.33%)	5 (6.67%)	33 (44%)
]29 - 39]	8 (10.67%)	1 (1.33%)	9 (12%)
]39 - 49]	2 (2.67%)	2 (2.67%)	4 (5.33%)
]49 - 59]	1 (1.33%)	1 (1.33%)	2 (2.67%)
>59	2 (2.67%)	1(1.33%)	3 (4%)
TOTAL	**62 (82.67%)**	**13 (17.33%)**	**75 (100%)**

The average age of our patients was 24.2 ± 18.91 years for extremes of 10 and 65 years (**Table 1**). This result is similar to that of Fall S. *et al.* [19] in Senegal in 2010, who noted an average age of 23.5 years with extremes of 6 and 58 years. The most represented socio-professional layer was that of pupils and students. This result corroborates Fongoro S. *et al.* [9] study in Mali in 2009, which noted a predominance of pupils and students with 36.7%.

The results of urine analysis noted into our patients a predominance of proteinuria in 100% of cases, proteinuria of different levels (**Table 2**). This result is identical to Kanté AS in Guinea in 2019 [10]. The predominance of proteinuria in those studies could be explained by the fact that those samples were mainly

composed of adult patients. Indeed, according to the literature, microalbuminuria and proteinuria do not appear before the age of 7. Afterwards, their prevalence increases with the age of the patient, exceptionally for the $SSFA_2$ homozygosity field [8] [20]; for the prevention of this anomaly, some authors suggest periodically looking for proteinuria using a urine strip or measuring microalbuminuria from the age of 5, then, each time there are vaso-occlusive crises or after blood transfusions. These factors impact the results [6].

Compared to the presumed initial types of nephropathies, tubulo-interstitial nephropathy was predominant in 46.15% of cases (**Table 2**). Our result differs from that of Kanté A.S. *et al.* [10] in Guinea in 2019 who noted a predominance of glomerular nephropathy. We don't have any explination for that difference.

Table 2. Distribution of sickle cell patients with kidney damage according to the 24h urine tests.

Urine analysis	Number of cases	Percentage (%)
Proteinuria/24h:		
—Tubulo-interstitial origin	6	46.15
—Vascular origin	4	30.77
—Glomerulus origin	3	23.08
TOTAL	**13**	**100**

Concerning the hemoglobin level, all but one of our renal patients suffered from severe anemia (92.31%) and the average hemoglobin level was 5.2 ± 2.7 g/dL (**Table 3**). This result is lower than that of Thiam L. *et al.* [21] in a study carried out in 2017 with 46 children aged from 2 months to 21 years with sickle cell SS. They found that all their patients suffered from anemia with a mean hemoglobin level of 08.6 g/dL and ranges of 05.7 and 11.8 g/dL.

Anemia is multifactorial in this area, combining sickle cell disease and nephropathy. For sickle cell disease, it is explained by acute or chronic hemolysis (following autoimmune reaction, transfusion accident, malaria access, vaso-occlusive crisis), by hemorrhagic syndrome, by splenic or hepatic sequestration, by an inflammatory syndrome, and by bone marrow necrosis.

As for anemia linked to kidney disease, it frequently occurs in patients suffering from chronic kidney disease (CKD), especially in advanced stages. This anemia is multifactorial, due to erythropoietin deficiency, inhibition of erythropoiesis induced by hyperuremia, reduced lifespan of red blood cells also due to significant uremia, as well as an imbalance in iron homeostasis.

Other etiologies are possible, such as vitamin deficiencies (vitamin B_{12} and folate) [22]-[24].

For calcemia, 8 on 13 patients had hypocalcemia (61.54%) vs 5 cases of normal calcemia. We found a predominance of the $SSFA_2$ homozygous form with 82.67% (**Table 4**). This result is not far from that found by Diakité M. *et al.* [25] in Guinea in 2019, who also noted a predominance of the $SSFA_2$ form with

Table 3. Distribution of sickle cell patients with kidney damage according to biological tests.

Biological tests	Number of cases	Percentage (%)
Hemoglobin level:		
—Mild anemia (>9 g/dL)	—	—
—Moderate anemia (6 - 9 g/dL)	1	7.69
—Severe anemia (<6 g/dL)	12	92.31
Average RHb: 5.2 ± 2.7 g/dL	***Extremes***: 4.6 et 8 g/dL	
Calcemia:		
—Hypocalcemia	8	61.54
—Normocalcemia	5	38.46
Uremia:		
—High	7	53.85
—Normal	4	30.77
—Not rated	2	15.38
Creatininemia:		
—High	12	92.31
—Normal	1	7.69

Table 4. Distribution of variables presumed to be predictive factors of renal damage in sickle cell patients.

Presumed variables	Kidney damage				P. value
	Yes		No		
	N	%	N	%	
Sex:					
—Femele	5	38.46	23	37.10	1.00
—Male	8	61.54	39	62.90	0.90
Sickle cell forms:					
—SC	—	—	1	1.61	1.00
—AS	1	7.69	11	17.74	0.32
—$SSFA_2$	12	92.31	50	80.65	0.003
Creatininemia:					
—High	12	92.31	6	9.68	0.001
—Normal	1	7.69	56	90.32	0.10
Uremia:					
—High	11	84.62	10	16.13	0.001
—Normal	2	15.38	52	83.87	0.10

Continued

Probable initial nephropathy:					
—Glomerulus origin	3	23.08	19	30.65	1.00
—Vascular origin	4	30.77	12	19.35	0.15
—Tubulo-interstitial origin	6	46.15	31	50.00	1.00

77.7%. According to the literature, the homozygous form of sickle cell disease $SSFA_2$ is the most symptomatic form with rheological complications, manifesting mainly by hyperfiltration with evolution towards chronic kidney disease [26].

For serum urea, we found with renal damage patients 11 cases of high rate, or 84.62% versus 2 cases of normal serum urea, or 15.38%. Of patients without kidney damage, 10 (16.13%) had a high urea rate and 52 (83.87%) had a normal urea rate (**Table 4**). It's easy to understand a high urea rate in kidney impairment patients: toxins are not excreted due to kidney failure, depending on the level of glomerular filtration rate (GFR). For patients without kidney damage, according to the literature, the high urea rate can be explained by a diet rich in protein (which is degraded into urea by liver), or strong physical exercises (which will product much urea from muscles), or some liver diseases (by reduction of protein catabolism) [27]. For our study, the high urea rate in patients without kidney damage may be approximately explained by this last option: reduction of protein catabolism following probable liver disease. Let's remember that we are in Hematology-Oncology department, one of the main department for liver diseases.

During our study, the estimation of GFR using the MDRD formula allowed us to note that the majority of our patients were in stage I of chronic kidney disease followed by stage II. Our results are similar to those found by Kanté AS *et al.* [10] in Guinea in 2019 who reported a predominance of stage I of chronic kidney disease.

According to the literature, the homozygous form of sickle cell disease is the most symptomatic form with rheological complications targeting the kidneys, manifesting mainly through hyperfiltration and chronic kidney disease [26]. In our study, we have not had a case of hyperfiltration which is a complication appearing very early in homozygous children from the age of 2 years (by secretion of vasodilator prostaglandins, in reaction to the hemodynamic complications of sickle cell disease). However, our sample consisted of adults, adolescents and children with extremes of 10 and 65 years, all aged over 2 years. These therefore constitute fertile ground for renal complications which have had time to progress towards fibrosis lesions with chronic renal failure. This also indicates an improvement in the life expectancy of sickle cell patients, allowing time for renal complications to progress [28] [29].

Bivariate analysis showed that the $SSFA_2$ form, the elevated serum creatinine and elevated serum urea were the main factors associated with renal damage in

sickle cell patients, respectively P = 0.003, P = 0.001, P = 0.001 (Table 4). Our results are partially similar to those found by Sri LH *et al.* [16] who showed that serum elevation of creatinine with P = 0.017 and urea with P = 0.04 increased the chances of rapid decline in kidney function.

Although this study clarified the predictive capacity of these few factors for the detection of kidney damage, the smallness of the sample does not allow a definitive conclusion to be drawn. It would be more judicious to repeat the same study in the future with larger and more varied populations.

5. Conclusion

The majority of our patients were in stage I of CKD followed by stage II. Our study revealed that the high rate of the $SSFA_2$ form of sickle cell disease, and the high serum levels of creatinine and urea, constituted the main factors in predicting renal damage in this particular area. This would make it possible in the future to better target sickle cell patients for whom screening for kidney lesions will be systematic.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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