

Pubertal Development of Adolescents with Homozygous Sickle Cell Disease in Brazzaville

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Abstract

Introduction: Pubertal development represents a physical, biological, and psychological process resulting in the acquisition of reproductive capacity. Among the factors inhibiting pubertal development are chronic diseases, including sickle cell disease, which is a public health problem. The objective of this work was to report the prevalence of pubertal development abnormalities and to identify the associated factors that delay pubertal development in adolescents with sickle cell disease. **Materials and Methods:** This was a multicentric cross-sectional analytical study conducted from April 1 to October 30, 2022, at the National Reference Center for Sickle Cell Disease and the University Hospital of Brazzaville. It included adolescents with sickle cell disease aged 10 to 19 years. The study was based on the evaluation of secondary sexual characteristics using Tanner's classification. The variables studied were socio-demographic, clinical, paraclinical, and therapeutic. Data processing and analysis were performed using SPSS version 20. The significance threshold was set at 5%. **Results:** Among the 347 adolescents included, 56.5% had normal puberty, 42.6% had delayed puberty, and 0.9% had hypogonadism. Associated factors were irregular follow-up, malnutrition, more than 5 hospitalizations per year, low baseline hemoglobin levels, hypochromic microcytic anemia, and a high percentage of hemoglobin S. **Conclusion:** The prevalence of delayed puberty is high among adolescents with sickle cell disease in Brazzaville. Improved follow-up and intensified treatment for children with sickle cell disease appear necessary.

Keywords

Pubertal Development, Adolescent, Homozygous Sickle Cell Disease, Brazzaville

1. Introduction

Pubertal development represents a physical, biological, and psychological process resulting in the acquisition of reproductive capacity [1] [2]. It is regulated by activating and inhibiting factors, among which chronic diseases, including sickle cell disease—the world’s leading hemoglobinopathy and a public health issue—are included [3]. Certain factors affecting pubertal development in adolescents with homozygous sickle cell disease, such as inadequate nutrition and deficiencies in essential micronutrients, are avoidable [4]-[7]. Sickle cell disease leads to endocrine complications in adolescence, such as growth delay and delayed puberty, which are common reasons for consultation [8]-[13].

The prevalence of delayed puberty in adolescents with sickle cell disease varies by region: 8% to 50% in Europe and 15% in Latin America [10] [11] [14]. African studies confirm these trends, with prevalence rates of 25% in Egypt and 75% in Nigeria [15] [16]. In Congo, studies report prevalence rates of delayed puberty ranging from 28.7% to 53.7% in children with sickle cell disease, thus constituting a significant health concern [17] [18].

The objective of this work was to report the prevalence of pubertal development abnormalities and to identify the associated factors that delay pubertal development in adolescents with sickle cell disease in Brazzaville.

2. Materials and Methods

This was a multicentric cross-sectional analytical study conducted from April 1 to October 30, 2022, in Brazzaville (the political capital of the Republic of Congo), specifically at the Antoinette Sassou Nguesso National Reference Center for Sickle Cell Disease (CNRDr-ASN), the Pediatric General Unit (PGE), and the Mother and Child Consultation Unit (CME) at the University Hospital of Brazzaville. The study included, after obtaining consent from the parent/legal guardian (for minors) and/or the adolescent (for those of legal age), all adolescents with sickle cell disease aged 10 to 19 years with at least one complete blood count in their medical records. The study was based on the evaluation of secondary sexual characteristics using Tanner’s classification and complete blood count analysis. Adolescents with other known chronic conditions (congenital heart disease, respiratory failure, HIV/AIDS infection, endocrine disorders) that could impact growth, as well as female adolescents with homozygous sickle cell disease who were pregnant, were excluded. The study variables included sociodemographic factors (age in years, gender, educational level, social status [orphaned or not], place of residence, follow-up center, birth order, diet [number of meals per day]); clinical factors (age at diagnosis, circumstances of disease discovery, number of hospitalizations per year, number of blood transfusions since diagnosis, status of acute and chronic complications, splenectomy, nutritional status as evaluated by WHO 2006 standards, age of onset of secondary sexual characteristics [thelarche, menarche, pubarche, spermarche, gynecomastia], pubertal stage according to Tanner’s Classifica-

tion, sexual activity, and lifestyle); paraclinical factors (electrophoretic profile [hemoglobins S, A, F], hematometric parameters [baseline hemoglobin level; mean corpuscular volume, white blood cell and platelet count]); and therapeutic factors (follow-up, prophylactic treatment, vaccination coverage, and basic treatment). Variables related to parents (age; number of dependent children, number of other children with sickle cell disease, nationality, marital status, socioeconomic status, and level of education) were also studied.

In this study, delayed puberty was defined in boys as the absence of testicular enlargement (<4 mL or length <25 mm) beyond the age of 14 years, and in girls as the absence of breast development at age 13 years or the absence of menstruation (primary amenorrhea) at age 15 years. An adolescent was considered prepubescent if they had no secondary sexual characteristics at that age, depending on their sex.

Data collected on a pre-established standardized survey form were entered into Microsoft Excel version 2013. Data processing and analysis were performed using SPSS version 20. Central tendency parameters (mean, median, mode) were used to describe quantitative variables. For univariate analysis, a significance threshold of 5% was used to identify potential risk factors. Logistic regression was used for multivariate analysis to eliminate confounding factors. Authorization was obtained from the Health Sciences Research Ethics Committee (CERSSA) under number 0027-18/MESRSIT/DGRST/CERSSA/-22, along with approval from the heads of the selected centers.

3. Results

3.1. Sociodemographic Characteristics

The average age of the adolescents was 15.1 ± 0.7 years (range: 10 to 19 years); there were 182 boys (52.4%) and 165 girls (47.6%), with a sex ratio of 1.1. In 210 cases (60.5%), the adolescents had a secondary level of education, while thirteen adolescents (3.7%) were not attending school. Fifty-six adolescents (16.1%) were orphans (**Figure 1**).

Their diet was varied in 346 cases (99.7%); however, 140 of them (40.6%) consumed fewer than 3 meals per day. The average age of the fathers was 48.07 ± 2.6 years (range: 29 to 72 years), and that of the mothers was 41.02 ± 2.1 years (range: 15 to 62 years). Three hundred twenty-three (93.1%) of the parents were Congolese nationals. Other nationalities included Senegal (1.1%), Mali (2.6%), the Democratic Republic of the Congo (2.9%), and Cameroon (0.3%). One hundred eighty-eight (54.2%) of the adolescents' parents were married, 126 (36.3%) were single, and 33 (9.5%) were part of a blended family. One hundred four adolescents (30%) came from families of low socioeconomic status.

3.2. Clinical Aspects

The average age of the adolescents at the time of diagnosis was 5.25 ± 0.6 years (range: 8 months to 15 years) (see **Table 1**).

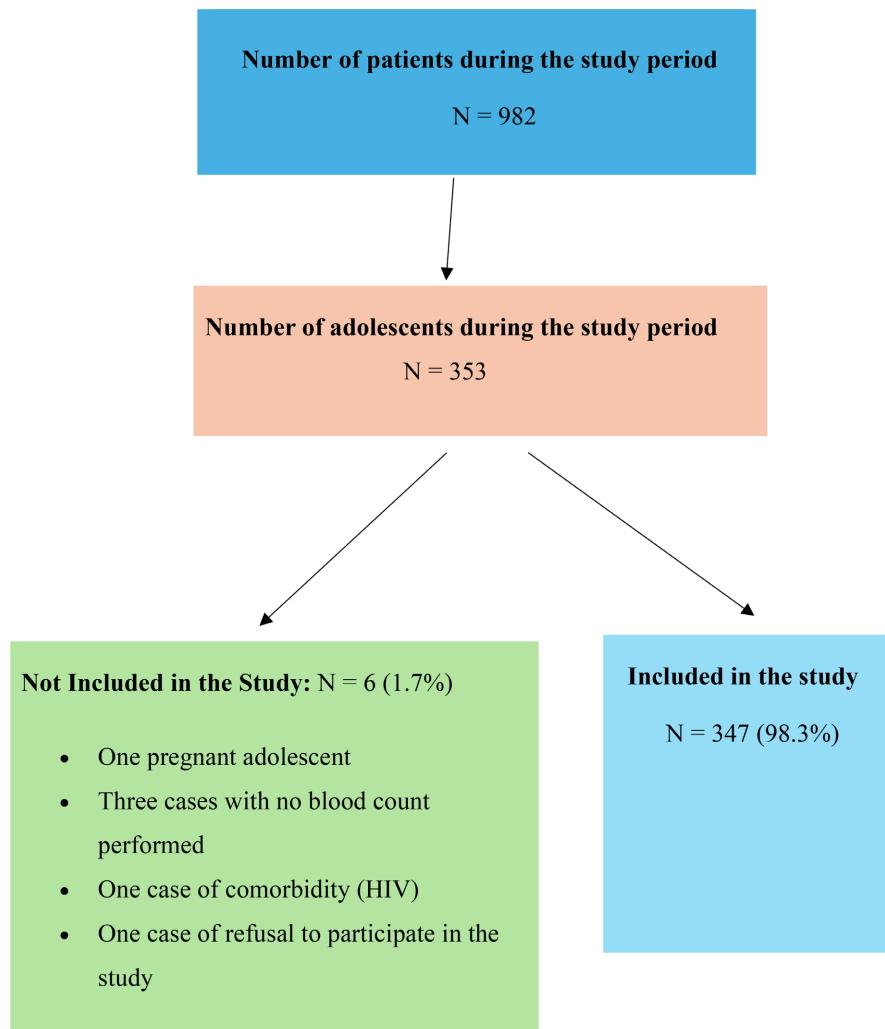


Figure 1. Selection flow diagram.

Table 1. Distribution of adolescents by age at diagnosis.

Age (years)	Number (n)	Percentage (%)
≤1	125	36
]1 - 5]	142	40.9
]5 - 10]	61	17.6
>10	19	5.5
Total	347	100

Three hundred twenty-six adolescents (93.9%) had previously received blood transfusions. The average number of blood transfusions was 3.7 ± 0.2 (range: 1 to 18 transfusions).

Table 2 shows the distribution of adolescents according to the circumstances of disease discovery.

Table 2. Distribution of adolescents by circumstances of discovery.

	n	%
Circumstance of Discovery		
Hand-foot syndrome	118	34
Vaso-occlusive crisis (VOC)	95	27.4
Anemic crisis	80	23
Screening	14	4
Anemic crisis + VOC	12	3.5
Anemic crisis + hand-foot syndrome	2	0.6
Others*	26	7.5
Total	347	100

One hundred twelve (32.3%) adolescents had severe emaciation.

For girls, the average age of thelarche (breast development) was 13.09 ± 1.2 years (range: 10 to 16 years); the average age of menarche (first menstruation) was 15.95 ± 1.1 years (range: 13 to 18 years). During the study period, 117 (33.7%) girls had already experienced menarche.

For boys, the average age of spermarche (first ejaculation) was 15.76 ± 1.3 years (range: 13 to 18 years); 46 boys (13.3%) had already experienced their first ejaculation. In both genders, the average age of pubarche (appearance of pubic hair) was 13.91 ± 1.1 years (range: 10 to 18 years).

In our study, 196 adolescents (56.5%) had normal pubertal development for their age. Delayed pubertal development was observed in 151 adolescents, representing 43.5% (see **Figure 2**).

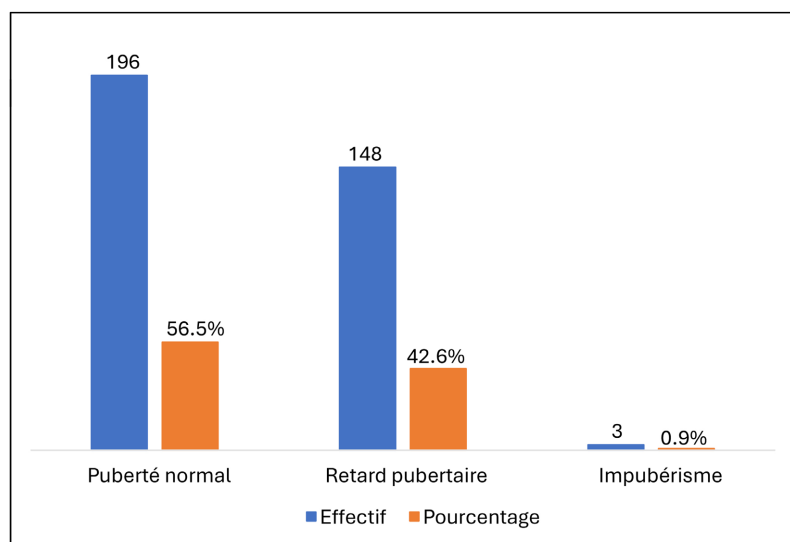


Figure 2. Distribution of adolescents by pubertal development.

3.3. Paraclinical Aspects

The average baseline hemoglobin level was 7.61 ± 0.5 g/dl (range: 5.8 to 10.3 g/dl). Two hundred one (57.9%) adolescents had a baseline hemoglobin level below 8 g/dl. Hemoglobin electrophoresis was performed on 203 adolescents (58.5%), with an average hemoglobin S percentage of $90.98\% \pm 7.4\%$ (range: 76% to 94%). All cases exhibited anemia, classified as normocytic normochromic in 178 cases (51.3%), microcytic hypochromic in 145 cases (41.8%), and macrocytic in 24 cases (6.9%).

3.4. Therapeutic Aspects

Follow-up was regular in 239 cases (68.9%). Folic acid was administered to 337 adolescents (97.1%). Vaccine coverage rates were as follows: 35.7% for typhoid, 58.2% for pneumococcus, 25.1% for meningococcus, and 21% for influenza. Thirty-three adolescents (9.5%) received intensified treatment with hydroxyurea. Two adolescents (0.6%) underwent exchange transfusion, indicated for ischemic stroke.

3.5 Risk Factors for Delayed Puberty

Table 3 lists the different risk factors for delayed puberty identified after logistic regression.

Table 3. Risk factors for delayed puberty.

Risk Factor	n (%)	OR	95% CI	p-value
Irregular follow-up	108 (31.1)	2.1	[1.1 - 4.01]	0.024
Nutrition (meals < 3/day)	140 (40.3)	1.8	[1.1 - 3.1]	0.023
Hospitalizations (>5/year)	67 (19.3)	2.9	[1.2 - 7.3]	0.017
Baseline Hb level (<8 g/dl)	201 (57.9)	8.2	[1.6 - 40.6]	0.009
Hb S percentage (>80%)	183 (52.7)	4.9	[1.6 - 156]	0.037
Microcytic hypochromic anemia	145 (41.8)	5.5	[1.5 - 21]	0.012

4. Commentary and Discussion

The study on pubertal development in children with sickle cell disease in Brazzaville aimed to report the prevalence of pubertal development abnormalities and identify the factors influencing pubertal development. However, it encountered several limitations. The first limitation was related to data sources, as some adolescents did not have their health follow-up documents; when available, many records were incomplete, leading to declarative data. The second limitation was the lack of funding for biological tests, such as hormonal profiles, which could have provided information on hormonal dynamics during puberty in adolescents with homozygous sickle cell disease. As a result, puberty diagnosis was purely clinical,

based on Tanner's classification. Additionally, the cross-sectional nature of the study only provides a snapshot. A longitudinal study would be more suitable to determine the dynamics of puberty in adolescents with sickle cell disease, as puberty is a process.

Despite these limitations, the study's location at the only sickle cell reference center in Brazzaville and the largest pediatric department in the national health system in Congo, combined with the sample size, supports that these results reflect the sickle cell population in Brazzaville.

The prevalence of pubertal development abnormalities among children living with sickle cell disease in Brazzaville is high: 43.5%, of which 42.6% are cases of delayed puberty. This rate is lower than that reported by Mabiala Babela (53.7%) in the same hospital [18] [19].

Improved follow-up and treatment, including increased use of hydroxyurea over recent years in Congo, may explain the improvement in pubertal development among adolescents with sickle cell disease. However, efforts are needed to further reduce these rates to levels similar to those reported by Soliman in Egypt (25%) and Özen in Turkey (8%): 8% [10] [16].

Delayed puberty affected 21.5% of boys and 21.1% of girls, whereas previous Congolese studies reported prevalences of 30.2% and 42% in boys and girls, respectively [17] [20].

Hypogonadism, observed in only 0.9% of this study's sample and exclusively among boys, contrasts with higher rates reported by other authors [15] [16] [20]. Nonetheless, the gender pattern remains consistent; males are often more likely to experience hypogonadism than females.

The late onset of puberty at 13.09 years and menarche at 15.9 years in girls are consistent with those described in the literature. In fact, it has been reported that adolescents with sickle cell disease experience a delay in the average age of puberty onset of approximately two years compared to their peers without sickle cell disease [17] [21]-[23]. Factors associated with this delay include: irregular follow-up, fewer than three meals per day, more than five hospitalizations per year, a baseline hemoglobin level below 8 g/dL, a hemoglobin S percentage above 80, and the presence of microcytic hypochromic anemia. Irregular monitoring of adolescents (31.1% in this study) is common in Africa, as found in the studies by Minto'o Rogombé in Gabon (29%), Boiro in Senegal (44.1%), Diakité in Mali (71.1%), and Mbika Cardorelle in Congo (85%) [24]-[27]. In sub-Saharan Africa, poor monitoring is often linked to the low socioeconomic status of parents, their level of education, lack of socio-health education, and socio-cultural and religious beliefs. According to them, the use of traditional treatments is a culturally acceptable, affordable, and accessible alternative. Sangho in Mali and Minto'o Rogombé in Gabon report respective prevalences of 15% and 26.7% of children whose parents reported having used traditional treatments for sickle cell disease [24] [28]. Undernourishment is a factor hindering the growth and development of Congolese adolescents, as evidenced by 40.6% of the population in this study. According to

the Demographic and Health Survey (EDS Congo 2012), 23% of Congolese households have inadequate food consumption, preventing them from leading healthy and active lives [29]. In its 2018 annual report, the United Nations Children's Fund (UNICEF) reports that 149 million children worldwide suffer from stunted growth, nearly 50 million suffer from wasting, and more than 340 million suffer from essential micronutrient deficiencies, also known as "hidden hunger" [30]. Thus, undernutrition associated with sickle cell disease, a hypermetabolic condition, hinders organ maturation and therefore pubertal development. The high number of hospitalizations per year is, in this study as in that of M'Pembaloufoua, a factor associated with delayed puberty [20]. A high frequency of hospitalizations observed in adolescents with sickle cell disease in Brazzaville often reflects the severe form of the disease. These hospitalizations are caused by recurrent vaso-occlusive crises (VOC) and frequent deglobulization crises leading to anorexia and psychological distress, with a consequent impact on growth and pubertal development. Severe chronic anemia has a negative impact on pubertal development because it is responsible for intense erythropoiesis, excessive oxygen consumption by the myocardium, and energy expenditure at rest by vital organs, thus causing tissue malnutrition. Adolescents with hemoglobin S levels above 80% (52.7%) had a fivefold increased risk of delayed puberty compared to their peers with hemoglobin S levels below 80% (p-value < 0.037). The presence of hemoglobin S reduces the affinity of oxygen for red blood cells, causing chronic tissue hypoxia, which is responsible for malnutrition in the body and explains delayed puberty.

5. Conclusion

The prevalence of delayed puberty among children with sickle cell disease in Brazzaville is high, with secondary sexual characteristics delayed by an average of two years compared to the general population. Factors associated with delayed puberty in our context include irregular follow-up, fewer than three meals per day, more than five hospitalizations per year, a baseline hemoglobin level below 8 g/dl, an Hb S percentage above 80%, and microcytic hypochromic anemia. Health education, optimized care, and intensified treatment for children with sickle cell disease are essential tools for improving pubertal development in these patients.

Conflicts of Interest

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

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