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# Epidemiological, Clinical and Radiographic Profile of Thoracic Manifestations Associated with Sickle Cell Anemia in Adults at Teaching Hospital Yalgado Ouédraogo in Ouagadougou at Burkina Faso

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#### **Abstract**

**Introduction:** Sickle cell anemia is a public health priority, but do its chest manifestations always require a chest radiography? Our study aims to determine the epidemiological, clinical and radiographic profiles of the thoracic manifestations in the drepanocytic adult in the city of Ouagadougou (BURKINA FASO).

**Patients and Methods:** This was a prospective and cross-sectional study conducted from January 31, 2010 to February 01, 2011. All drepanocytic patients at least 16 years of age who presented chest signs and who carried out a chest radiography were included.

**Results**: One hundred and fourteen (114) cases were reported during the study period. The mean age was 28.40 with extremes of 16 and 56 years. The sex ratio was 0.62. The sickle cell phenotype was : AS : 10 cases (8.8%); SS : 26 cases (22.8%); SC : 74 cases (64.9%); S  $\beta$  + thalassemia : 03 cases (2.6%); S O Arabic : 01 case (0.9%). All our patients had a history of chest pain during vaso-occlusive crises. It was noted : dyspnea (36.8%), cough (9.6%) and palpitations (5.3%); fever (30.7% of cases).

Chest radiography was normal in half of our patients, the majority of radiographic lesions consisted of 31 cases (27.2%) of parenchymal lesions

**Conclusion:** The systematic carrying out of a chest radiography for thoracic manifestations in patients with sickle cell anemia allows the diagnosis of pulmonary secondary infections.

Keywords: Sickle Cell Anemia; Chest Pain; Chest Radiography; Burkina Faso

#### Introduction

Sickle cell anemia is a public health priority, but do its chest manifestations always require a chest radiography? Our study aims to determine the epidemiological, clinical and radiographic profiles of the thoracic manifestations in the drepanocytic adult in the city of Ouagadougou (BURKINA FASO).

Sickle cell anemia or hemoglobinosis S or drepanocytic anemia is an autosomal recessive disease. Hemoglobin S is the result of a mutation of the  $17^{th}$  nucleotide (Thymine - Adenine) of the  $\beta$  - globin gene causing the replacement of glutamine (negatively charged) by valine (neutral amino acid) at the level of the 6th amino acid of segment A of the chain :  $\beta$  6 glu  $\rightarrow$  val. [1-4]. The mutation results in an abnormal hemoglobin which tends to polymerize to form an elongated molecule which causes a change of

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the erytrocyte into a sickle form (sickle en Anglais, hence the name of the hemoglobin: Hb S). It is the synthesis of this abnormal hemoglobin (Hb): Hb S which is mainly responsible for all of the vaso-occlusive clinical manifestations and for chronic hemolysis with anemia of varying degrees. [1,4].

Sickle cell anemia is the most common hemoglobinopathy. Burkina Faso constitutes by its geographical location the epicenter of hemoglobinosis C on the one hand and on the other hand, belongs to the sickleemic belt of LEHMANN (which covers from the  $15^{th}$  parallel of north latitude, to the  $20^{th}$  parallel of south latitude where sickle cell frequencies are  $\geq 10\%$ ) [5]. Sickle cell anemia is therefore a real public health problem with a high prevalence of major sickle cell syndromes SS and SC [1,2,5].

Acute thoracic syndrome (occlusion of the pulmonary vessels) complicates 5 to 30% of cases of sickle cell anemia in adults [4]. It is the leading cause of death in adults, it can occur spontaneously or following surgery or a vaso-occlusive pain attack [1]. Lung damage when not diagnosed in time can lead to irreversible pulmonary fibrosis that causes respiratory failure [4]. Infectious complications (secondary to a progressive asplenia) are the main cause of death in children with sickle cell disease [4].

In our context, the standard chest radiography is the first-line examination in front of clinical signs of thoracic call.

Numerous clinical studies have been carried out on osteo-articular lesions of sickle cell anemia and on its infectious complications in children; but very few studies to our knowledge have dealt with thoracic and pulmonary complications in adult sickle cell anemia, hence the interest of our study.

The aim of our study which is to establish the epidemiological, clinical and radiographic profile of thoracic manifestations associated with sickle cell anemia in adults at Teaching Hospital YALGADO OUEDRAOGO of OUAGADOUGOU (BURKINA FASO).

#### Patients and Methods

This was a prospective and cross-sectional study conducted from January 31, 2010 to February 01, 2011. All drepanocytic patients at least 16 years of age who presented chest signs and who carried out a chest radiography were included.

This was a prospective cross-sectional study (medical emergency department and clinical hematology department of the above-mentioned University Hospital) from January 31, 2010 to February 01, 2011.

Were included:

Sickle cell patients aged at least 16 years admitted to medical emergencies for acute thoracic manifestations with lung or chest X-ray and a usable clinical record. The department of pediatrics of our hospital YALGADO OUEDRAOGO had transferred to department of clinical hematology 27 sickle cell patients that had passed the pediatric age that is to say < 16 years. The diagnosis of the drépanocytic anemia has been confirmed according to the recommendations of the french society of clinical biology [6].

Patients followed in clinical hematology with a history and / or having episodes of vaso-occlusive crises (CVO) including pulmonary, thoracic manifestations with lung or chest radiography.

Sickle cell anemia patients with uninterpretable lung or chest x-rays were not included in the study.

Operational definitions:

The following parameters were collected:

Epidemiological: age, sex, occupation

Clinics: chest pain, dyspnea, cough, palpitations, hemoptysis, functional heart murmur, crackling or bronchial rattles,

Biological: hemoglobin electrophoresis, hemogram, iron metabolism assessment (serum iron, transferrin or siderophilin and ferritinemia). When the C or S hemoglobin was detected to the electrophoresis of the hb to alkali pH, the electrophoresis of the hb to acidic pH was achieved account held of the analogs of the C Hemoglobin: HbE, HbO-Arab. The analog of HbS: Hb D-Punjab [7].

Radiological: thoracic, pleural, parenchymal, pulmonary and cardiac lesions.

#### **Operational definitions**

**Diagnostic criteria for sickle cell anemia:** All our patients benefited from hemoglobin electrophoresis at alkaline pH (pH 8.5) on cellulose acetate, from automated capillary electrophoresis to precisely measure the minor fractions of hemoglobins Hb  $A_2$  and Hb F and from a functional test with 2% metabisulfite (= EMMEL test) when hemoglobin S was found. The EMMEL test was positive when sickle cell cells were observed with a blood smear at magnification 40.

Each patient carried out a hemogram which contributed to the interpretation of the hemoglobin electrophoresis.

The iron metabolism balance was sought in the event of microcytosis (Mean Globular Volume = V.G.M. < 80 Femtolitre) and / or hypochromia (Mean Corpuscular Hemoglobin Content = T.C.M.H. <27 Picograms and / or Mean Corpuscular Concentration in Hemoglobin = C.C.M.H. <32 %) by a serum iron dosage < 12  $\mu$ mol/L, a plasma transferrin > 4 g/L and a ferritinaemia < 20  $\mu$ g/L in women and < 30  $\mu$ g/L in men. The biologic criterias for the diagnosis of the drépanocytose are described in the CERBA notebooks in "Recommandations pour la mise en oeuvre et l'interprétation de l'étude de l'hémoglobine" du laboratoire CERBA" [7].

Acute thoracic syndrome (ATS): ATS was diagnosed on the criteria of the High Authority of Health (HAS) of France: association of a new radiological pulmonary infiltrate which can appear 24 to 48 hours after one or more of following symptoms: cough, fever, acute dyspnea, sputum, chest pain or auscultatory abnormalities (crackling rattles or tubal murmur, decreased vesicular murmur) [4].

**Number of vaso-occlusive crises (VOC) in the year:** Only VOCs from the year immediately preceding our study (2009) were counted. The data were entered on a microcomputer and analyzed with the Epi info 3.5.1 software in its French version. The sociodemographic, clinical and radiological aspects have been analyzed and discussed in relation to the literature.

Proportional comparisons were made using the  $\chi^2$  test.

The degree of statistical significance (p) < 5% was retained.

#### Results

One hundred and fourteen (114) cases were reported during the study period. The mean age was 28.40 with extremes of 16 and 56 years. The sex ratio was 0.62. The sickle cell phenotype was : AS : 10 cases (8.8%); SS : 26 cases (22.8%); SC : 74 cases (64.9%); S  $\beta$  + thalassemia : 03 cases (2.6%); S O Arabic : 01 case (0.9%). All our patients had a history of chest pain during vaso-occlusive crises. It was noted : dyspnea (36.8%), cough (9.6%) and palpitations (5.3%); fever (30.7% of cases).

Chest radiography was normal in half of our patients, the majority of radiographic lesions consisted of 31 cases (27.2%) of parenchymal lesions.

#### Epidemiological and clinical characteristics of patients

Of the 144 patients, 25 had not carried out a radiography, 5 had uninterpretable radiographies. Consequently, one hundred and fourteen (114) patients were retained during the study period, representing an annual hospital prevalence of 114 cases. Our patients consisted of 70 women and 44 men, a sex ratio of 0.6. The mean age was 28.40 years with a standard deviation of 10.50. The youngest patients were 16 years old and the oldest 56 years old. The most represented age group was that of 16 to 20 years old (31 cases, or 27.2% of the patients). Pupils and students were the most affected socio-professional category (52 cases / 114). These epidemiological results that we got that are in relation with our specific objectives allow us to target the girls especially (20 to 30 years) for the therapeutic education.

Double composite heterozygotes SC (74 cases / 114) were the most represented with 64.90% of types of hemoglobinosis S as illustrated in (Table 1).

|               |       | Type of hemoglobinosis S |        |         |         | Total  |
|---------------|-------|--------------------------|--------|---------|---------|--------|
|               | AS    | SS                       | SC     | Sβ+thal | SO arab | n      |
| Medical (n)   | 1     | 8                        | 9      | 0       | 0       | 18     |
| Emergencies % | 0,9 % | 7,0 %                    | 7,9 %  | 0 %     | 0 %     | 15,8 % |
| Clinical (n)  | 9     | 18                       | 65     | 3       | 1       | 96     |
| Hematology %  | 7,9 % | 15,8 %                   | 57,0 % | 2,6 %   | 0,9 %   | 84,2 % |
| Total (n)     | 10    | 26                       | 74     | 3       | 1       | 114    |
| Frequency     |       |                          |        |         |         |        |
| Percentage    | 8,9 % | 22,8 %                   | 64,9 % | 2,6 %   | 0,9 %   | 100 %  |

n = number = number of cases; % = percentage

Table1: Distribution of the different types of hemoglobin S according to patients recruitment service

| Type of hemoglobisose S | Chest Signs |       |         |            |              |
|-------------------------|-------------|-------|---------|------------|--------------|
| Type of hemoglobisose s | Chest Pain  | Cough | Dyspnea | Hemoptysis | Palpitations |
| AS n                    | 8           | 0     | 5       | 0          | 0            |
| %                       | 7,0%        | 0%    | 4,4%    | 0%         | 0%           |
| Sβ+Thalassemia n        | 3           | 0     | 1       | 0          | 0            |
| %                       | 2,7%        | 0%    | 0,9%    | 0%         | 0%           |
| SC n                    | 74          | 8     | 24      | 0          | 4            |
| %                       | 65,0%       | 7,0%  | 22,0%   | 0%         | 3,5%         |
| S O Arab n              | 1           | 0     | 0       | 0          | 0            |
| %                       | 0,%         | 0%    | 0%      | 0%         | 0%           |
| SS n                    | 26          | 4     | 19      | 0          | 3            |
| %                       | 22,8%       | 3,5%  | 16,7%   | 0%         | 2,7%         |
| Total                   | 112         | 12    | 49      | 0          | 7            |
| Frequency %             | 98,2        | 10,5  | 43,0    | 0          | 6,1          |

n = number of cases; % = percentage

**Table 2:** Distribution of thoracic signs reported by patients during a crisis according to the type of hemoglobinosis S

Chest pain (presented by 112 patients / 114 or 98.2%) occurred mainly during vaso-occlusive crises (V.O.C.) Chest pain was the most observed functional sign for each type of hemoglobinosis as shown in Table 2. The other functional chest signs observed (cough, dyspnea, hemoptysis and palpitations) are specified in (Table 2).

One case of crackling rattles was noted in each of the following types of hemoglobinosis S: drepanocytic AS feature, drepanocytic homozygous SS, drepanocytic composite heterozygous SC.

#### Radiographic aspects

Chest radiography was normal in half of our patients. The radiographic lesions included: deformations of vertebrae in H were observed in 19 heterozygous SC patients (16.7%), 14 SS homozygotes (12.3%) and in three AS patients (2.6%); There were also two cases/114 patients (1.7%) of aseptic osteo-necrosis of the humeral heads in heterozygous SC patients, one case of aseptic osteo-necrosis of the left humeral head in one homozygous SS patient (0.9%), and one case of osteitis of the right clavicle in an SS patient (0.9%). We did not objectify costal or sternal lesions. Lesions of the pulmonary parenchyma were observed mainly in 17 SC patients (14.9%) and eight SS patients (7.0%), as specified in Table 3. A pleural effusion was observed in a heterozygous SC patient. We observed 2 cases of cardiomegaly, that is 1.7% of our study population in two SS sickle cell patients. S $\beta$ ° thalassemia, S $\beta$  + thalassemia and Arab S O did not show cardiomegaly.

In total, there were 31 cases of pleuro-parenchymal lesions (27.2%), 2 cases of cardio-mediastinal lesions (1.7%) and 42 sickle cell patients with thoracic bone lesions (36.8%), with for several radiographic images, an association of these elementary lesions.

(Table 3) shows the distribution of pulmonary parenchymal lesions according to the type of hemoglobinosis S. For interstitial involvement between heterozygotes SC and homozygotes SS: p = 0.0985.

| Electrophoresis of hemoglobin | Alveolar syndrome<br>n (%) | Interstitial syndrome<br>n (%) | Total<br>n (%) |
|-------------------------------|----------------------------|--------------------------------|----------------|
| AS                            | 0                          | 3 (2,6)                        | 3 (2,6)        |
| Sβ+                           | 0                          | 1 (0,9)                        | 1 (0,9)        |
| SC                            | 2 (1,7)                    | 15 (13,1)                      | 17 (14,8)      |
| SO Arab                       | 0                          | 1 (0,9)                        | 1 (0,9)        |
| SS                            | 1 (0,9)                    | 7 (6,1)                        | 8 (7,0)        |
| Total (%)                     | 3 (2,6)                    | 27(23,6)                       | 30 (26,2)      |

n = number of cases

Table 3: Distribution of pulmonary parenchymal lesions according to the type of hemoglobinopathy S

#### Acute chest syndrome (ACS)

No case of acute chest syndrome (ACS) was confirmed in our study.

The other nosological entities apart from the acute thoracic syndrome.

The alveolar syndrome objectified to standard radiography made it possible to guide the diagnosis of an abscess of the left lung in a heterozygote SC, in whom the culture of the liquid obtained by echo-guided puncture, highlighted a staphylococcus aureus.

Interstitial syndrome guided the diagnosis of human immunodeficiency virus (HIV) infection in a 47 years old heterozygous SC patient with bilateral pneumonia in six cases (three SS patients and three SC patients).

Pleural syndrome allowed after investigation, to evoke pulmonary tuberculosis confirmed by bacteriology in a heterozygous patient SC aged 41 years.

Chest radiography led to the conclusion of a pathology in nine cases out of 114, ie 7.90% of the cases in our series. Koffi *et al* in Côte d'Ivoire [8], found in his series, 93% of pneumonia, four cases of pulmonary tuberculosis including one case confirmed by bacteriology; he had also isolated nine different germs, including two cases of staphylococcus aureus infection.

#### Discussion

### Epidemiological and clinical characteristics of patients

The mean age in our series was 28.40 years. The mean age of our patients is explained by the fact that the majority of sickle cell patients followed in the clinical hematology department were transferred from the pediatric department of the Teaching Hospital YALGADO OUEDRAOGO after the pediatric age limit (<16 years). Furthermore, the young age of the population in developing countries could justify the predominance of this age group. The lowering of the mean age of patients which was 13.43 years in Mounkaila B *et al* series in Niger compared to our series was related to his study population (1 year to 47 years) which took into account children aged at least one year [9]. The female sex predominated with a sex ratio of 0.62 in our series. These results are

close to those of Doupa D *et al* in Dakar, Shongo M *et al* in Lubumbashi who found a sex ratio of 0.75 and 0.9 respectively [10,11]. The predominance of the female gender in the general population in Burkina Faso associated with the fact that hemoglobin electrophoresis is part of the prenatal consultation assessment could justify this female predominance in our series. Other authors do not find any predominance between the two sexes, such as Than *et al*. [12]. These differences would be related to the demographic data of each country because the transmission of the defect is autosomal therefore independent of sex [13].

Pupils and students were the most affected socio-professional category (52 cases / 114) in our study. This could be justified by the fact that the majority of our patients came from pediatrics. As a matter of fact, when they are more than 15 years old, they are transferred to the clinical hematology department of the same Teaching Hospital.

Composite SC heterozygotes were the most represented with 64.9% (74 cases / 114) of the electrophoretic profiles of sickle cell hemoglobin in our study. Our results contrast with those of Diop et al, DOUPA D et al who found a predominance of the SS homozygous form with 100% of patients [10,14]. The predominance of SC phenotypes in our study would find its answer by the fact that Burkina Faso belongs on the one hand to the LEHMANN sicklemic belt where sickle cell anemia is a real public health problem, and on the other hand, this country is part of the voltaic plateau which is the epicenter of hemoglobinosis C where the prevalence of hemoglobinosis C remains high [15]. A false heterozygote AS was observed on the electrophoresis of hemoglobin with alkaline ph on cellulose acetate; in fact this patient who often presented vaso-occlusive crises was diagnosed through an automated capillary electrophoresis: composite heterozygote  $\beta$  + thalasso-sickle cell. It is therefore necessary to perform 3 separate phenotypic tests as recommended by the nomenclature of medical biology analyzes (NABM). The performance of a single technique (High Performance Liquid Chromatography or HPLC only or electrophoresis at alkaline pH only) is not recommended and, moreover, a normal profile, whatever the system used, does not allow to eliminate a hemoglobin mutant. Thus, for example, hemoglobins S and D have the same migration in electrophoresis on cellulose acetate but are separated in other systems; in HPLC several mutants are co-eluted with Hb A, HbA2 or Hb F [6]. Another patient diagnosed with AS was confirmed homozygote SS. This AS patient had performed a hemoglobin electrophoresis six weeks after receiving a transfusion of concentrated red blood cell. Consequently, hemoglobin electrophoresis should not be performed in a patient whose transfusion of concentrated red blood cells is less than three months old [6]; the life time of red blood cells being 100 to 120 days, an electrophoresis of hemoglobin made within three months after a transfusion of concentrated red blood cells will identify the red blood cells hemoglobins phenotypes of of the recipient and the donor.

Dyspnea affected 42.9% of our sickle cell patients and was the most common symptom after chest pain. Ibidapo  $\it{et~al}$  in Nigeria found a predominance of fever (72%), followed by asthenia (59%) [16]. The lung is a target organ for sickle cell disease; due to the richness of its capillary network and its exposure to the ambient air, it is the seat of thrombosis and infection, responsible for acute and chronic manifestations [4]. Thrombosis, which causes a significant drop in the level of oxygen saturation in the blood, could explain the frequency of dyspnea. The frequency of dyspnea may also be related to how it is perceived by patients. Indeed, perceived as a sign of imminent death, dyspnea is so memorable that it is hardly forgotten in the list of functional signs encountered; but patients may tend to exaggerate its intensity. The lack of financial means did not allow an etiological assessment of five cases of dyspnea among our ten heterozygotes AS, in particular an association with an enzyme deficiency in G6PD, or a particular symptomatic form of heterozygosity. Indeed some rare mutants such as Hb S-West Indies (HBB: c. [20A> T; 70G> A]) are double mutants, that is to say that they present two mutations in cis including the mutation  $\beta$ S. (HBB: c.20A> T); currently about fifteen are described. These double mutants havent the particularity of being difficult to diagnose phenotypically because the second mutation can completely modify the physicochemical properties of Hb S. In addition, they can sometimes, like Hb S-West Indies, promote polymerization and constitute a major sickle cell syndrome even in the heterozygous state [2]. Other tropical diseases could coexist such as digestive parasitosis (hookworms and anguillulosis can cause labile infiltrates in the lung: Loeffler syndrome; amoebiasis of the lungs) and especially asthma in its atypical form.

Heart murmur was noted in 0.9% of our patients (an SS homozygote); it is the complication of chronic anemia, especially of the anemic forms of sickle cell anemia [4].

One case of crackling rattles was noted in each of the following three types of hemoglobinosis S: AS sickle cell feature, homozygous SS sickle cell, heterozygous composite SC sickle cell. Logically, there should be a predominance of crackling rattles in heterozygous composite SC sickle cells which were the most numerous and there should be fewer crackling rattles in the AS sickle cell feature who could not be affected by functional asplenia; indeed patients with major sickle cell syndromes may present a functional asplenia which makes them susceptible to infections with encapsulated germs [4]. Our results could be explained by recruitment ways: the clinical signs noted are those collected on the day of hospitalization in medical emergencies or on the day of the consultation in the clinical hematology department, crackling rattles occurring after admission to the Teaching Hospital were not recorded.

#### Radiographic aspects:

Two patients all homozygous SS or 1.7% of our sickle cell patients showed a cardiomegaly in our series. Bertrand *et al.* in Ivory Coast [17] found 32.40% of cardiac involvement; the differences with our results could be explained by the smaller size of our sample, especially in patients with SS homozygous sickle cell anemia. Indeed, the chronic anemia that characterizes homozygous SS sickle cell anemia is the source of cardiac complications [4]. The finding of cardiomegaly in our SS sickle cell patients (2 cases

/ 26) while there is no cardiomegaly in our SC sickle cell patients who were the most numerous in our series (0 cases / 74) could be justified by the fact that SS homozygous sickle cell anemia is a chronic anemic form which can be complicated by cardiomegaly [4]. At rest, the cardiac index is 1.5 times normal and increases during exercise in sickle cell anemia. This increase in the cardiac index in sickle cell anemia is not fully explained by anemia and is thought to be linked to increased tissue extraction of oxygen [4]. On the other hand, SC sickle cell anemia, S $\beta$  + thalassemia and S O arab are not anemic forms, which could explain the absence of cardiomegaly in our SC sickle cell anemia. The absence of cardiomegaly in S $\beta$  + thalassemia and S O arab in our series could also be explained by their small size of their sample. One case of osteitis of the right clavicle in an SS patient (0.9%) was observed while no case was noted in SC drepanocytic patients which are the most numerous. ; this could be explained by the fact that the clavicle is a hematopoietic bone more sensitive to chronic anemia which is the rule in homozygous sickle cell anemia.

In medical emergencies, two cases of lung parenchyma lesions and one case of pleural involvement were observed in 18 patients in this department in our series, while Jeffery *et al.* [18] in the United States found 52% of pleuro-parenchymal lesions, of which six cases of pleural involvement out of the 66 cases in their series. In addition to the smaller size of our study population (18 cases) compared to his sample (66 cases), the performance of the technical equipment used (conventional standard radiography versus digital radiography) could explain the difference with our results.

In the clinical hematology department, 30 cases of lesions of the pulmonary parenchyma were identified in 96 patients in this department. These proportions are explained by the chronicity of the lesions which result from the repetition of acute situations, thus becoming permanent [18]. Interstitial syndrome leads to the formation of pulmonary fibrosis with respiratory failure [4]. Computed tomography is more sensitive and more precise than conventional radiography; if it had been performed, it would have enabled us to determine more prevalence of interstitial syndrome and acute thoracic syndrome (ATS). Indeed in this syndrome the lesions are progressive; the initial radiography is either normal or pathological; in this case, it shows discrete infiltrates that extend quickly to the bases, with pleural involvement in 20 to 50% of the cases [4].

No case of acute thoracic syndrome (ATS) was confirmed in our study, but some cases of ATS were probably overlooked and treated as a serious bronchopulmonary infection. Indeed, the installation of the ATS is often insidious, in a sickle cell patient already hospitalized for another complication (bone vaso-occlusive crisis, pregnancy, after surgery etc ...) [4]. The poorly evaluated or poorly monitored administration of morphine, by the alveolar hypoventilation it can induce, is an etiology of ATS [4]. The signs of pulmonary appeal can be masked by the intensity of the pain of the attack, and the agitation which sometimes accompanies it; it was necessary to systematically and regularly search for them through clinical examination (respiratory frequencies, ambient air saturation and pulmonary auscultation) and repeat radiological examinations, especially when it is a conventional standard radiography. However, the radiographic examinations were not repeated in our study and the clinical signs noted are those present on admission to Teaching Hospital YALGADO OUEDRAOGO.

Thirty-six patients, or 31.60% of our drepancytic patients, had a deformation of the thoracic vertebrae while Jeffery *et al.* [18] in the United States found only 3%. The importance of the size of our sample (114 cases) compared to that of Jeffery *et al* (66 cases) on the one hand, and on the other hand, the very probable nutritional deficiencies and malabsorption of vitamin D and calcium of our patients could explain our results [19,20].

Limits: The way of data generalization: in fact, the study having been carried out in a hospital environment, it remains difficult to extrapolate these results to the whole of the general population; an analytical prospective cohort study will later permit to better document all of these aspects. The failure to perform certain biological examinations capable of detecting co-morbidity (infection with the human immunodeficiency virus: HIV, hepatitis B and C virus, etc., glucose 6 phosphate dehydrogenase deficiency: G6PD) and modify the clinical and radiological profile in particular, are all factors that can constitute a source of confusion. Indeed, our two sickle cell features that presented a cardiomegaly were not able to carry out the completeness checkup; the same is true of the three sickle cell features which presented H-shaped vertebrae; the molecular study of the globin genes would allow the precise identification of the hemoglobins of these dubious sickle cell features. The study being in hospital environment, we have a way of selection which partly explains the small size of our sample after one year of collection; in fact twenty-five (25) patients had not done an X-ray picture due to a lack of financial means generating a selection way at the socio-economic level.

#### Conclusion

The systematic carrying out of a chest radiography for thoracic manifestations in patients with sickle cell anemia allows the diagnosis of pulmonary secondary infections.

Chest pain and dyspnea dominated the scene of clinical manifestations. "H" shaped vertebral deformities and lesions of the pulmonary parenchyma (especially interstitial syndrome) were the most observed radiographic signs. Radiographic lesions were observed in 45.50% of cases and were noted even in patients with sickle cell anemia deemed to be at low risk for chronic complications. Standard radiography has contributed to the confirmation of bronchopulmonary infections during thoracic manifestations of sickle cell anemia in adults.

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# **Competing Interests**

The authors declare that they have no financial or personal relationship(s) which may have inappropriately influenced them in writing this paper.

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