

Complete blood count among patients with sickle cell gene (S gene) visiting El-Obied hospital (Sudan)

* Rehab Omer Adam M Gibla, Abozer Elderderly, Adlan Mohammed Elnour, Mohammed Abd Elazeem Gibreel,

Ahmed Hassan Ahmed

University of El-imam El-mahadi, Faculty of Medicine and Health Sciences, Aba Island, Sudan

Abstract

The aim of this study was to identify and classify the hematological parameters among patients with sickle cell gene who frequently visit El-Obied teaching hospital for medical care. The study was conducted in El-Obied Town. Agreement was taken from Forty four (44) patients admitted in Obied hospital to be a part of the study. Blood samples were collected and analyzed for CBC and HB electrophoresis by full analyzer Sysmex and electrophoresis tank. Sickling test was carried for determination of positive or negative (S- gene). The analysis showed mean values of CBC as follows HB (7.3), PCV (24.7), MCV (81.1), MCH (24.2) and MCHC (29.7), TWBCs (17.2), RBCs (3.1), PLT count (327) and RDW (64.1) in SCA patients. In the SCT patients, the analysis showed mean values as HB (11.6), PCV (37.4), MCV (83.4), MCH (25.9) and MCHC (31.1), TWBCs (7.3), RBCs (4.5), PLT (224.8) and RDW (44.1). Electrophoresis results showed frequency of SCT as (45.5%) and SCA as (27.3%). The sickling test was found to be positive for (54.5%) and negative for (45.5%) of the study group.

Keywords: sickle cell anemia, HB electrophoresis, haemoglobin, heterozygous, sickle gene

1. Introduction

Anaemia is a medical condition in which the red blood cell count (RBC) or Hb is below the reference range^[1]. The sickle cell disease is observed in certain areas of the Sudan, ranging from 0.8% in central Sudan to 30.4% in western Sudan. The Messeria tribe as a branch of Baggara groups in Kordofan and Darfur was reported to be has the highest rate of sickle cell disease^[2]. Sickle cell anaemia (SCA) was discovered by Herrick in 1910. He described the clinical and hematological manifestations of SCA^[3]. Globally, this disease affects millions of people, particularly those who come from sub-Saharan Africa; Spanish-speaking regions (South America, Cuba, Central America); Saudi Arabia; India; and Mediterranean countries such as Turkey, Greece, and Italy^[4]. Its highest frequency was reported in tropical regions, and the Middle-East^[4]. In the United States, it affects around 72,000 people, most of them come from Africa. The disease occurs in about 1 in every 500 African-American births and 1 in every 1000 to 1400 Hispanic-American births. About 2 million Americans, or 1 in 12 African Americans, carry the sickle cell trait^[5]. Migration of substantial populations from these high prevalence areas to low prevalence countries in Europe has dramatically increased in recent decades and in some European countries sickle cell disease has now overtaken more familiar genetic conditions such as hemophilia and cystic fibrosis^[6]. Some studies found that Sickle Cell Disease (SCD) affects about 90 x 10³ Americans^[7]. The disease was also reported in one out of each five hundreds African-American births and one out of each 36 x 10³ Hispanic-American births^[7]. Most infants with SCD, born in the United States of America and some other developed countries are now identified by routine neonatal screening. Forty-four states along with the District of Columbia, Puerto Rico and the Virgin Islands currently provide universal neonatal screening for SCD^[8, 9]. In Sudan there is no special health

centers to determine the affected infants from the first time of their birth. Therefore, this study suggests, a setting up of screening programs in the main hospitals in all the states of the country. Furthermore, sickle Cell trait (SCT) was reported among about 1:12 African-Americans and 1:100 Hispanic-Americans^[10]. It was also estimated that 2.5 million Americans are heterozygous carriers for the sickle SCT^[11]. Some studies reported that about 4.2% of Saudi Arabia had SCT and 0.26% had SCD with the highest prevalence in the Eastern province where approximately 17% of the population carries SCT and 1.2% had SCD^[12]. Therefore, a screening program was started in Saudi Arabia in 2005, as a mandatory pre-marital test to decrease the incidence of SCD and thalassemia^[13]. Three quarters of SCA were reported to be in Africa. A recent WHO report estimated that around 2% of newborns in Nigeria were affected by SCA giving a total of 150,000 affected children, born every year in Nigeria alone. The carriers frequency ranges are between 10% and 40% across equatorial Africa, decreasing to 1–2% on the North African coast and <1% in South Africa. In Sudan, some studies reported SCA with high prevalence, in comparison with other Hemoglobin abnormalities. This was described in some Sudanese tribes with varying degrees eg, Misserai, Hamar, Hausa, Folani and Bargo, Elderderly and coworkers were reported Hb S with high frequency in western Sudanese ancestry^[14]. Therefore, this study is intended to identify and classify the hematological parameters related to ACD in Northern Kordofan population with S gene, taken the patients that frequently visit El-Obied teaching hospital as the case of study.

2. Materials and Methods

5mls of venous blood sample were taken from each patient and transferred to K₂-EDTA anti coagulant container. The samples collected from 44 patients were analyzed for CBC

using full analyzer sysmex. Electrophoresis tank was used for HB electrophoresis analysis. Sickling test was carried for each sample.the analysis was performed at Elobied Teaching Hospital laboratories.the obtained results were statistically analyzed using SPSS program.

3. Results and discussion

From the Fourty four screend participants 32 patients were found to be have HbAS and HbSS, 20 of these cases had the AS gene [SCT] as 45.5% and 12 had SS gene 27.3% (Table. 1). This distribution of S gene has been reported before in Sudan [15, 16]. HbAS findings in other Arabic countries were reported as follows, Saudia Arabia 2-27%, Bahrain 11.2 %, Kuwait 6 %, Jordan 0.44%, UAE 1.9% and Yemen 0.95%. The carrier state (SCT) was higher in Saudia Arabia and Bahrain. In the Afro-American population the percentage was reported as 8% heterozygous for Hb AS [17]. Some studies reported that, SCT affects 8-10% of African American and up to 25-30% of the population in West Africa [27]. Sudanese carry mixed genes of Arab and African ethnicities.

Table 1: Classification of study population

Hb variant	Frequency
SS	12 (27.3)
AS	20 (45.5)
AA	12 (27.3)
Total	44

Observations of the peripheral blood films indicated polychromasia and target cells in the majority of Hb AS cases. Macrocytic cells and thrombocytosis were also found as well as rare cases of sickle cells. These blood profiles are indication of abnormal Hb. All patients in this survey were found to have Hb S [18]. MCV and RDW were significantly higher than the normal average (p<0.05). MCV was found to be normal in most SCD cases (table 3).The analysis showed high RDW count. This may be attributed to anisocytosis, abnormal shape of RBCs and cell fragments as factors which generally lead to haemolysis [24, 25, 26].

4. Conclusion and Recommendations

As a conclusion, The results obtained by complete blood count analysis in this study showed mean values of CBC as follows HB (7.3), PCV (24.7), MCV(81.1), MCH(24.2) and MCHC (29.7), TWBCs (17.2), RBCs(3.1), PLT count (327) and RDW(64.1) in SCA patients. In the SCT patients, the analysis showed mean values as HB(11.6), PCV(37.4), MCV(83.4),MCH (25.9) and MCHC (31.1), TWBCs(7.3), RBCs(4.5), PLT(224.8) and RDW (44.1). Electrophoresis

According to the sickling test, 24 patients showed positive results and 20 patients showed negative results (Table No. 2).

Table 2: Shows Sickling test results

Sickling test result	Frequency (%)
Positive	24 (54.5)
Negative	20 (45.5)
Total	44

The obtained Hb S mean was found to be low (7.3gdl), and such result may be expected in the studied cases. The haematological profiles for patients with HbAS were summarized in (Table 3), which showed that, the PCV reduction means were not significant (p>0.05) and the RDW elevation means were also not significant (p>0.05) compared to AA mean values. The RDW mean value is not expected. The patients with HbAS in this study did not show sickle cell disease crisis. The WBC mean values were not higher than the normal range (p>0.05). This may be due less complications of Sgene among those patients with SCT [18, 19]. WBCs and PLT counts in patients with SCD were significantly higher (p<0.00) as shown by (Table No. 3).this may be explained by the increased erythropoietic turnover attributed to microorganism infection [20, 21]. Higher PLT counts may also result from BM hyperplasia due to haemolysis [22, 23].

Table 3: Complete haemograms indices in each variant of haemoglobin

Blood parameters	HbAS	Hb SS	HbAA
Hb g/dl (±SD)	11.6 (±2.4)	7.3 (±1.7)	13.9 (±0.4)
RBCS10 ¹² /L (±SD)	4.5 (±0.9)	3.1 (±0.9)	5.1 (±0.5)
WBC 10 ⁹ /L (±SD)	7.3 (±3.4)	17.2 (±9.9)	6.8 (±0.9)
MCH/pg (±SD)	25.9 (±2.1)	24.2 (±4.2)	26.4 (±0.8)
MCHC g/dl (±SD)	31.1 (±1.1)	29.7 (±2.3)	31.3 (±1.6)
PCV l/l (±SD)	37.4 (±7.4)	24.7 (±6)	43.4 (±4.2)
RDW fl (±SD)	44.1 (±6.6)	64.1 (±15.9)	41.2 (±2.9)
MCV fl (±SD)	83.4 (±6.1)	81.1 (±9.8)	87.6 (±4)
PLt 10 ⁹ /L(±SD)	224.8 (±91.9)	327.7 (±168.6)	254.5 (±27.5)

results showed frequency of SCT as (45.5%) and SCA as (27.3%).

The sickling test was found to be positive for (54.5%) and negative for (45.5%) of the study group.

As recommendations more researches may be needed to determine Sickle cell anemia (SCA) in all the states of Sudan. Educational programs about SCA may also be need as well as spesilized centers for sickle cell disease survy, diagnosis and continuos medical care. such centers may determine easily the affected newborn children.

5. References

1. Olujohungbe Howard j. The clinical case of adult patient with the sickle cell disease. Br. J hosp medicine. 2008.
2. Majid mohammed sabahelzain, hanan hamamy. ethnic disteibutionof sickle cell disease in sudan. The pan african medical journal. 2014.
3. Online Mendel an inheritance in man (TM) johns Hopkins university Baltimore, MD.MIM NO 141900.- ABrie history of sickle cell disease. Joint center for sickle cell and thalassaemia, 2001.

4. Weather all DJ, clegg GB Bull world health organ, 2001; 79(8).
5. Awasthy N. Aggarwalwaikc Qyalbc, Prasad Ms, salujaas Sharma M. Sickle cell disease, Experince of atertiary car center in anonen demic area annals of tropical medicine and public health, 2008; 1(1).
6. Roberts1, demotalembrrt M. hematological, 2007; 92(7).
7. CDC and prevention. Retrieved, 2011.
8. American academy of pediatrics sectioaqn on hematological /and cology committee on genetic healthy supervision for the children with the sickle cell disease” pediatric, 2002; 109(3):526-535.
9. Passk Alane PA, Fernhoft PM. us newborns screaming system guide lines, 2000.
10. March of Dimes R February. Retrieved 8 November 2011, 2008.
11. Cinnchin sky EP. Mahoney DH. land law SA (29 November 2011).
12. Jastaniah W. (E SCD in Saudi Arabia) Annals of Saudi medicine, 2011; 31(3):289-293.
13. Memish ZA, saeedi MY. Annals of Saudi medicine, 2011; (31):229-35.
14. WHO (PDF) Retrieved 2010, 11- 27.
15. Omer A, Ali M, Omer AH, Mustafa MD, Satir AA, Samuel AP. Incidence of G-6-PD deficiency and abnormal haemoglobins in the indigenou and immigrant tribes of the Sudan,1972.
16. Ahmed HA, Baker EA. Sickling in the Sudan. Result of surveys in Blue Nile Province. East Afr J. 1986; 63(6):395-399.
17. Boulware DS. Sickle cell anemia still a dreaded enemy. Baltimore Afro-American, 1998.
18. El-Hazmi MA, Warsy AS. A comparative study of haematological parameters in children suffering from sickle cell anaemia (SCA) from different regions of Saudi Arabia. J Trop Pediatr. 2001; 47(3):136-41.
19. de Montalembert M, Brousse V, Zahar JR. Pneumococcal prophylaxis for children with sickle cell disease in Africa. Arch Dis Child, 2008; 93(8):715-6.
20. Nolan VG, Wyszynski DF, Farrer LA, Steinberg MH. Hemolysis-associated priapism in sickle cell disease. Blood, 2005; (9):3264-3267.
21. Litos M, Sarris I, Bewley S, Seed P, Okpala I, Oteng-Ntim E. White blood cell count as a predictor of the severity of sickle cell disease during pregnancy. Eur J. 2007; 133(2):169-72.
22. Rostagno C, Prisco D, Abbate R, Poggesi L. Pulmonary hypertension associated with long-standing thrombocytosis, 1991; 99(5):1303-5.
23. Platt OS. Sickle cell anemia as an inflammatory disease. J Clin. 2000; 6(3):337-8.
24. Ballas SK, Lewis CN, Noone AM, Krasnow SH, Kamarulzaman E, Burka ER. Clinical, hematological, and biochemical features of Hb SC disease. Am J. 1982; 13(1):37-51.
25. GT El, Badawi SB. Red blood cell distribution width index in some hematologic diseases. Am J Clin Pathol. 1985; 83(2):222-226.
26. Qurtom HA, al-Saleh QA, Lubani MM, Hassanein A, Kaddoorah N, Qurtom MA, *et al.* The value of red cell distribution width in the diagnosis of anaemia in children,
27. Vector Hoffbrand Douglas, R. Higgs, David M. Keeling, Atul B. Mehta Postgraduate hematology, 5th edition, 2015.