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Original Research Article

Foetal Haemoglobin in Sickle Cell Anaemia in Federal Medical Centre, Owerri, Nigeria

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Abstract

The foetal haemoglobin levels of adult suffering from sickle cell anaemia were deterimed in Owerri between September and October 2012. Also determined were the foetal haemoglobin levels of normal adults belonging to the genotype AA who served as control. Both foetal haemoglobin level were determined using standard haematological methods.The results obtained showed a statistical significant difference in the mean of the levels of haemoglobin F in patients (2.48±1.06) compared to controls (1.27±0.40) (p<0.05).The foetal haemoglobin levels of males with sickle cell anaemia were compared with those of their females' counterparts.The mean levels of haemoglobin F for male with sickle cell anaemia (2.94±1.14) compared to that of females with sickle cell anaemia (2.12±0.78) which showed statistical difference (p <0.05).

Keywords

F-cell
Foetal haemoglobin
Haemoglobin
Sickle cell anaemia

Introduction

Haemoglobin is found in the red blood cells of every human being, child or adult. It is necessary because haemoglobin binds and carries oxygen from the lungs to other tissues within the body. A developing baby has foetal haemoglobin while an adult,in most cases,carries adult haemoglobin. During the entire nine months of foetal formation, three different haemoglobins are produced, with one switching off as the next one beings. All three types contain the same haem molecules with an iron atom in the centre but they have different globins (Cox et al., 1999). Foetal haemoglobin (haemoglobin F) is the predominant form of haemoglobin expressed in a developing foetus. Haemoglobin F appaers a few months post conception and exists for a few months post-birth. Foetal red blood cells have a greater affinity for oxygen than adult haemoglobin (Omoti, 2005). However, at thirty five weeks, 90% of the foetal haemoglobin starts to phase out, leaving the third type, adult haemoglobin (haemoglobin A) to begin the production. Sickle cell disease (SCD) is an inherited disorder of erythrocytes,characterised by life-long anaemia and

recurrent painful episodes (Mcgra-Hill, 2002). It is a heritable disease for which no cure has yet been found(Okochi and Okpuzor, 2005). The molecular basis of the prototypical genetic disease sickle cell anaemia (SCA) is that valine is substituted for glutamic acid in the sixth position of the haemoglobin chain (Fasola et al., 2007).

Sickle haemoglobin (HbS) gelation studies showed that HbF did not interact in HbS, it was reported that compoud heterozygotes for sickle cell trait hereditary persistence of HbF (HPFH) were clinically normal despite having a very high HbS concentration (Steinberg and Rodgers, 2001). HbF is the most powerful modulator of the clinical and haematologic features of sickle cell anaemia (SCA).

To protect against various complications of disease,differnt concentrations of HbF were postulated to be required, although any increament in HbF had a beneficial effect on mortality (Platt et al., 2004). The major objectives of the study are given hereunder.

- To determine the foetal haemoglobin level in people living with sickle cell anaemia.
- To compare the foetal haemoglobin level in people living with sickle cell anaemia with foetal haemoglobin level in normal adult.
- To compare foetal haemoglobin level in both male and female with sickle cell anaemia.

Materials and methods

Study area

The study was conducted at federal medical centre, Owerri, Nigeria.

Study population and sample size

Fifty subjects were enrolled into this study.This included 30 sickle cell patients in steady state attending Federal Medical Centre, Owerri out patient clinic who were consecutively recruited into the study after an informed consent,they comprised of 17 females and 13 males. Also, 20 HbAA adults include 11 females and 9 males were enrolled into the study as control.

Collection of samples

5ml of venous blood was colected by clean venepuncture from each subjects via the ante-cubital vein into EDTA anticoagulated containers.4ml of the blood was used to prepare haemolsysate for HbF estimation and 1ml used for haemoglobin electrophoresis using cellulose acetate at 8.6 to confirm HbAA and HbSS status.

Statistical analysis

The results were presented as mean ±standard deviation(SD).The results were analysed using student t-test set at $p<0.05$.

Results

Foetal haemoglobin, a heritable trait in adults accounting for substantial phenotypic diversity of sickle cell diseasewas estiamted. The mean haemoglobin F (HbF) level expected in HbAA is 1.7% (Ochei and Kolhautkar, 2008) but for the study control was 1.27% which is still within the range because HbF declines after birth (Olivieri and Weatherall, 2000). Earlier study obtained a mean foetal haemoglobin level in HbSS people as 6.4±4.0% (Enoselease et al., 2005) and 5.16±0.4% (Olaniyi et al., 2010) But the mean HbF of patients of 2.48±1.065 in this study is lower than earlier values (Table 1).

Table 1. Percentage of HbF in the sickle cell anaemia patients.

Subjects	Mean±SD	Level of significance
HbSS(30)	2.48±1.06	$p<0.05$
HbAA(20)	1.27±0.40	

Table 2. Percentage of HbF based on gender of the subjects.

Subjects	Mean±SD	Level of significance
Female	2.12±0.78	$p<0.05$
Male	2.94	

HbF is significantly higher than that of HbAA beciase of genetic abnormalities of haemoglobin production of haematopoietic stress. In eukaryotic cells various factors determine whether a gene is expressed or not. Celular mechanisms to detremine which proteins are produced can act at any step in this process (Olivieri and Weatherall, 2000). In adults, rapid regeneration or expansion of the bone marrow results in an increased production of F-cells suggesting that the kinetics of erythropid regeneration determine whether a cell become F-cell (Steinberg and Rodgers, 2001). Althogh,the mean of HbF in male with sickle cell anaemia of 2.94% was slighlty higher than that of females (2.12%) ($p<0.05$) (Table 2). Another local study found a statistically higher leve of HbF in males than in females (Kotila et al., 2000).

Conclusion

Although all patients with sickle cell anaemia (SCA) have exactly the same molecular defects there is considerable clinical variation,ranging from death in ealry childhood to a normal life span with few complication due to influence of genetic modifiers of sickle cell anaemia.Therefore,patients with increased levels of HbF tend to have a relatively mild clinical course because HbF reduces the red cell This highlights the need to determine HbF with HbA_2 in assisting to differentiate HbSS, HbS-beta-thalassaemia and HbS-HPFH.

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