### ABSTRACT

The effect of sickle cell disease (SCD) on total white blood cell (WBC) count, differential white blood cell count and packed cell volume (PCV) estimations were assayed on 64 known sickle cell disease patients attending sickle cell clinic at University of Nigeria Teaching Hospital (UNTH) Enugu. These three parameters were also assayed in 20 normal subjects that served as control. Sickle cell disease patients were grouped into four states namely steady state, vaso-occlusive crisis state, and steady state with bacterial infection and vaso-occlusive crisis state with bacterial infection. The mean PCV results of 22.70 ± 4.35%, 17.30 ± 2.99%, 19.27 ± 1.48% 20.40 ± 2.91% and 49.05 ± 2.31% were recorded in SCD patients in steady state, vaso-occlusive crisis state, steady state with bacterial infection, vaso-occlusive crisis state with bacterial infection and control subjects respectively. The mean PCV of patients in the 4 different states of SCD significantly decreased (P<0.05) when compared with control subjects. The mean PCV of SCD patients in steady state increased significantly (P<0.05) compared with those in vaso-occlusive crisis state. There was no significant difference between the mean PCV (P>0.05) of patients in steady state with bacterial infection and those in vaso-occlusive crisis state with bacterial infection. The mean total WBC count results of 15.14 ± 4.33 x 10^9/L, 26.34 ± 8.86 x 10^9/L, 17.87 ± 3.08 x 10^9/L, 19.50 ± 4.46 x 10^9/L, and 7.92 ± 2.53 x 10^9/L were recorded in SCD patients in steady state, vaso-occlusive crisis state, steady state with bacterial infection, vaso-occlusive crisis state with bacterial infection and control subject respectively. The mean total WBC count of patients in the 4 different states of SCD significantly increased (P<0.05) when compared with the control subjects. The mean total WBC count of SCD patients in steady state decreased significantly (P< 0.10) when compared with those in vaso-occlusive crisis state but that in steady state with bacterial infection decreased significantly (P>0.05) when compared with those in vaso-occlusive crisis state with bacterial infection. The mean neutrophil count results of 50.60 ± 13.68%, 61.30±19.29%, 59.67±2.27%, 70.10±8.88%. and 43.40± 2.67% were recorded in SCD patients in steady state, vaso-occlusive crisis state, steady state with bacterial infection, vaso-occlusive crisis state with bacterial infection and control subjects respectively. The mean neutrophil count of patients in the 4 different states of SCD significantly increased (P<0.05) when compared with the control subjects. The mean neutrophil count of SCD patients in steady state decreased significantly (P < 0.10) when compared with those in vaso-occlusive crisis state but that in steady state with bacterial infection decreased significantly (P<0.05) compared with that in vaso-occlusive crisis state with bacterial infection. The mean lymphocyte count results of 45.80± 15.55%, 34.20± 18.31%, 37.80± 2.51%, 24.11± 8.71% and 54.20± 2.96% were recorded in SCD patients in steady state, vaso-occlusive crisis state, steady state with bacterial infection, vaso-occlusive crisis state with bacterial infection and control subjects respectively. The mean lymphocyte count of patients in the 4 different states of SCD significantly decreased (P<0.05) when compared to control subjects. The mean lymphocyte count of SCD patients in steady state increased significantly (P<0.05) when compared with those in vaso-occlusive crisis state but that in steady state with bacterial infection increased significantly (P<0.05) compared with that in vaso-occlusive crisis state with bacterial infection. There was no much difference in the eosinophil and monocyte count in the 4 different states of SCD patients when compared with each other and with the control subjects. Out of the 64 bacterial cultures done, 37.5% yielded significant growth and the organisms isolated include Staphylococcus aureus, Klebsiella aerogenes. Escherichia coli, Pseudomonas aeruginosa and Streptococcus pyogenes with Staphylococcus aureus being the most predominant.

**KEYWORDS:** leucocyte count, children, sickle cell anaemia, steady state, vaso-occlusive crisis state, bacterial infection.
INTRODUCTION
Sickle cell disease as defined by Britton (1969) is a chronic haemolytic anaemia occurring almost exclusively in Negroes. The disease is peculiar to Africans and people of African origin it can also be defined as a condition in which an individual has inherited two abnormal Hb genes at least one of which is HbS and the resulting symptomatology or Pathology is attributable to the sickling phenomenon. Other abnormal haemoglobins e.g. Hbs D Punjab, O Arab etc may also co-polymerize with HbS to produce sickle cell disease (Azubuike and Nkanginieme, 1999).

Sickle cell anaemia is the homozygous form of the disease in which the individual inherits double dose of the abnormal gene that codes for haemoglobin -S (Hb-S). The substitution of valine for glutamic acid in the sixth position of the beta chain of the Hb molecule is the cause. This disease rarely manifests before the age of 6 months due to the predominance of Hb-F at birth, but is always fatal at the age of 30 years (Britton, 1969). The effect of this substitution produces different physicochemical changes in the haemoglobin molecules. It also changes the behaviour of haemoglobin molecules which tend to polymerize on deoxygenation, dehydration and decrease in pH. As a consequence, a red blood cell becomes less pliable and some become deformed into the characteristic sickle shape.

Sickle cell crisis refers to the onset of acute symptoms generally due to sudden in vivo sickling in a person who is a sickler. It is a sharp turn or definite change in the course of the disease with the development of new symptoms and signs. There are four main types of crisis namely vaso-occlusive crisis, haemolytic crisis, sequestration syndrome and aplastic crisis. The vaso-occlusive crisis is the most common type and also the only painful one. The basic cause of such a crisis is obstruction of blood flow by tangled mass of sickled cells and immeasurable degree of vasospasm. Vaso-occlusive crisis as defined by (Azubuike and Nkanginieme, 1999) is referred to as pain or thrombotic crisis. It is caused by occlusion of blood vessels by sickled cells, resulting in pain from ischaemic tissue injury. It can affect any part of the body but it is particularly common over the long bones, abdomen, chest and the back. The pain usually lasts for 4 -6 days but may persist for weeks. Precipitating factors include physical exertion, fever, dehydration, exposure to extremes of weather and emotional disturbance (Kaine, 1983).

The steady state is defined as that period when the patient is free of crisis or other acute problems. Bacterial infection is a major cause of morbidity and mortality in patients with sickle cell anaemia. Patients with sickle cell anaemia are at greatly increased risk to pneumococcal infection especially meningitis but also to bacteremia. Organisms responsible include Streptococcus pneumonia. Streptococcus pyogenes, Staphylococcasaeus. Escherichia coli, Klebsiella aero genes. Pseudomonas aeruginosa, and Haemophilus influenzae and Salmonella species. Bacterial infections also precipitate sickle vaso-occlusive crises (Charache et al., 1989).

There appears to be a clear role for leucocytes in exacerbating many ischaemic destructive processes and this is likely to include the tissue damage that occurs in sickle cell vaso-occlusive crisis (Embry et al., 1994).

Leucocytosis and a shift to the left support a diagnosis of bacterial infection in patients with haemoglobinopathy. Leucocytosis is also common in steady state (Diggs, 1932). White blood cell count also increases during the painful crisis occasionally stimulating a leukaernoid reaction (Diggs, 1965; Buchanan and Glader, 1978). In the presence of bacterial infection the leucocytosis is more marked and associated with an increase in band neutrophil. The increase in leucocyte count in children with sickle cell anaemia may be related to decreased margination or increased bone marrow myeloid activity (Buchanan and Glader, 1978). Determination of leucocyte count helps to detect infection since sickle cell disease exposes patients to infection. There is no known work on leucocyte count in relation to sickle cell disease in this part of the world.

AIMS AND OBJECTIVES OF THIS STUDY
1. To investigate and compare leucocyte count in sickle cell patients both in steady state and vaso-occlusive crisis state.
2. To investigate and compare leucocyte count in sickle cell patient with bacterial infection both in steady state and vaso-occlusive crisis state.
3. To compare findings with that of homozygous HbA individuals.

MATERIALS AND METHODS
SUBJECTS AND SAMPLES
Samples were collected from sickle cell disease patients attending the sickle cell clinic at University of Nigeria Teaching Hospital Enugu. A total of 64 samples (23 female, 41 male.) were used in this work with confirmed Hb genotype. Their age ranges from 1-18 years. The sickle cell disease patients were grouped into four categories namely those in steady state, vaso-occlusive crisis state, steady state with bacterial infection and vaso-occlusive crisis state with bacterial infection.

Blood samples were collected from 30 steady state (10 male and 20 female) 10 vaso-occlusive crisis (5 male and 5 female), 15 steady state with bacterial infection (9 male and 6 female) and 9 vaso-occlusive crisis state with bacterial infection (7 male and 2 female). Twenty primary and secondary school students with confirmed Hb genotype AA were used as control. All blood samples were collected by venepuncture with sterile disposable needles and syringes into an EDTA anti coagulated bottle.
Urine samples, sputum samples, wound and ear swabs were also collected from all patients in steady and vaso-occlusive crisis states, those without significant white cells in their wet preparation were excluded for culture.

TOTAL WHITE CELL COUNT (TURKS METHOD CITED IN DACIE AND LEWIS (1994))
PRINCIPLE Blood was diluted in Turks solution containing 1% glacial acetic acid tinged with gentian violet, the glacial acetic acid lyses all the red cells while the gentian violet stains the white blood cells making it easy to be identified for counting.

METHOD
A 1 in 20 dilution of blood was made by adding 0.02ml of anti-coagulated blood to 0.38ml of Turks solution in a test tube. The suspension was mixed on a mechanical mixer for 10 minutes. The unproved Neubauer chamber was charged and filled with the suspension using a stout glass capillary. Prior to counting, the chamber was allowed to stand for about one minute to allow the white cells to settle on the chamber. The cells were counted using the x 40 objective and x 10 eyepiece of the microscope. All the white cells in the four corner squares were counted and multiplied by 50 which is a factor previously determined from first principle calculation and this will give the WBC value per cubic millimeter.

DIFFERENTIAL WHITE CELL COUNT (METHOD CITED IN BAKER AND SILVERTON (1985))
A thin blood film was made on a glass slide using a spreader and the blood film was allowed to air dry. The film was fixed with leishman stain for 2 minutes after which the stain was double diluted with buffer solution (pH 6.8) and stained for 10 minutes.

It was then washed and differentiated with buffer solution until it is salmon pink colour. It was then drained and dried in the air. The stained slide was then observed with oil immersion objective of the microscope and 100 cells were counted and the result expressed as a percentage.

HAEMOGLOBIN GENOTYPE (METHOD CITED IN BAKER AND SILVERTON (1985))
PRINCIPLE At alkaline pH (8.6) haemoglobin is a negatively charged protein and will migrate towards the anode in an electric field. Most structural variants of haemoglobin will separate due to surface charge differences thus allowing identification of abnormal forms.

METHOD Cellulose acetate strips were soaked in the barbitone buffer. The strips were then blotted and then placed across the bridges of the electrophoresis tank. The strips were secured using the wicks of whatman No. 1 filter paper soaked in buffer. The haemolysate (lysed blood sample) was applied near the cathode bridge using fine stick. Electrophoresis was carried out at 200V. Separation was completed within 20 minutes and the strips were read. A known control sample was electrophoresed with each batch.

PACKED CELL VOLUME (HAEMATOCRIT) (METHOD CITED IN DACIE AND LEWIS (1994))
PRINCIPLE A column of whole blood is centrifuged at 10,000rpm for 5 mins in a microhaematocrit centrifuge; complete packing of red cells is achieved while the leucocytes and plasma are suspended above the column.

METHOD
Well mixed EDTA anti coagulated blood was allowed to enter a 75mm capillary tube by capillarity leaving at least 15mm unfilled. The tube was sealed in the pilot light of a bunsen burner combined with rotation. It was then centrifuged in a micro haematocrit centrifuge at 10,000 rpm for 5 minutes and the haematocrit read off from the Hawksley micro haematocrit reader. The result was expressed as a percentage of the column of blood.

CULTURAL METHODS (METHOD CITED IN SHANSON (1999))
The urine samples were mixed by inverting the containers several times; a sterile platinum wire loop of internal diameter 0.003mm was used. The wire which was sterilized by flaming was dipped into the urine vertically and inoculated into the CLED medium and on blood agar medium. The sputum samples, wound swabs and ear swabs were also cultured on the same media. The plates were incubated aerobically at 37°C for 24 hours. The urine samples were centrifuged at 3000 revolutions per minute for 5 minutes and the sediments were examined microscopically to detect and count the white blood cell, red blood cells and casts. Gram stain was carried out on the sputum samples and ear swabs for the presence of pus cells (white cells). After 24hours incubation, the CLED and blood agar media were read for cultures that have pure isolates of significant growth and the identification test and sensitivity test were carried out as well.

RESULTS
A total of 64 samples were included in the investigation. The subjects were between the ages of 1 year and 18 years. Thirty (46.88%) out of the 64 sickle cell disease patients were on steady state, 10 (15.63%) were on vaso-occlusive crisis, 24 (37.5%) had bacterial infection with 15(23.44%) on steady state and 9(14.06%) on vaso-occlusive crisis state.

The normal Hb AA individuals had normal PCV estimation with a mean of 40.05± 2.31%. Steady state SCD patients had a mean PCV of 22.70± 4.35%, those in vaso-occlusive crisis, state had a mean PCV of 17.30± 2.99%, those in vaso-occlusive steady state with bacterial infection had a mean PCV of 19.27± 1.48% and those in vaso-occlusive crisis state with bacterial infection had mean PCV of 20.40±2.91%.
The normal Hb AA individual had normal total white cell count with a mean of 7.92±2.53 x 10^9/L, steady state SCD patients had a mean total WBC count of 15.14±4.33 x 10^9/L, those vaso-occlusive in crisis state had a mean total WBC count of 26.34±8.86 x 10^9/L, those in steady state with bacterial infection and vaso-occlusive crisis state with bacterial infection had a mean white cell count of 17.87 ± 3.08 x 10^9/L and 19.50 ± 4.46 x 10^9/L respectively.

The normal Hb AA individual had normal neutrophil count of 43.40± 2.67%, patients in steady state had a mean neutrophil count of 50.60± 13.68%, vaso-occlusive crisis state had a mean neutrophil count of 61.30 ± 19.29% and those in steady state with bacterial infection and vaso-occlusive crisis state with bacterial infection had a mean neutrophil count of 59.67± 2.27% and 70.10± 8.88% respectively.

The normal Hb AA individual had normal lymphocyte count of 54.20± 2.96%, steady state had a mean lymphocyte count of 45.80± 15.55%, vaso-occlusive crisis state had a mean lymphocyte count of 34.20± 18.31% and those in steady state with bacterial infection as well as those in vaso-occlusive crisis state with bacterial infection had a mean lymphocyte count of 37.80± 2.51% and 24.11± 8.71% respectively.

There was no much difference in the eosinophil and monocyte count in the different state of SCD patients when compared with that of control subjects. Blood picture of SCD patients showed moderate to marked hypochromasia, anisocytosis, poikilocytosis, nucleated red cells and sickle shaped cells. There is also presence of target cells reflecting the absence of normal splenic function. Blood film of normal subjects showed normochromic and normocytic red cells. The culture proven bacterial infections occurred in 24 children (15 steady state) and (9 vaso-occlusive crisis state). The infections included episodes of bacteremia, some of which were associated with a focus of infection including pneumonia, osteomyelitis, urinary tract infection and boils. Out of the organisms isolated from the samples, the predominant organisms were Staphylococcus aureus, KlebsieUa aero genes and Escherichia coli.

These results were compared in Tables I, II, III and IV. Table I shows the summary of the data obtained for all the parameters assayed to investigate leucocyte count of SCD patients in steady state and in control individuals. All the parameters showed a significant difference (P<0.05) when compared with that of control except the eosinophil that was not significantly different from the control (P>0.05).

Table II shows the summary of the data obtained for all the parameters assayed to investigate leucocyte count of SCD patients in vaso-occlusive crisis state and in control individuals. All the parameters showed a significant difference (P<0.05) from the control.

Table III respresents and compares the results of SCD patients in steady state and those in vaso-occlusive crisis state. There was significant difference in all the parameters assayed (P<0.10) except in monocyte count where there was no significant difference (P>0.10).

Table IV represents and compares the results of patients in steady state with bacterial infection and vaso-occlusive crisis state with bacterial infection. There was no significant change (P>0.05) in the PCV, total white cell count and monocyte count but there was significant difference in the neutrophil, lymphocyte and eosinophil count (P< 0.05). Table V represents the frequency of organism isolated.

Figure III is the diagrammatic representation of total white cell count of SCD patients in vaso-occlusive crisis state, vaso occlusive crisis with bacterial infection, steady with bacterial infection and steady state. Figure IV is the diagrammatic representation of neutrophil count of SCD patients in vaso-occlusive crisis with bacterial infection, vaso-occlusive crisis state, steady state with bacterial infection and steady state. Figure V is the diagrammatic representation of lymphocyte count of SCD patients in steady state, steady with bacterial infection, vaso-occlusive crisis state and vaso-occlusive crisis with bacterial infection. Figure VI is the diagrammatic representation of PCV estimation of SCD patients in steady state, vaso-occlusive crisis state with bacterial infection, steady state with bacterial infection and vaso-occlusive crisis state. Fig VII is the diagrammatic representation of the frequency of microorganisms isolated.

<table>
<thead>
<tr>
<th>subjects</th>
<th>PCV %</th>
<th>Total WBC*10^9/L</th>
<th>Neutrophil %</th>
<th>Lymphocyte %</th>
<th>Eosinophil %</th>
<th>Monocyte %</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCD in steady late n=30</td>
<td>22.70± 4.35(SD)</td>
<td>15.14± 4.33(SD)</td>
<td>50.60± 13.68(SD)</td>
<td>45.80± 15.55(SD)</td>
<td>1.63±1.28 (SD)</td>
<td>2.10±1.85 (SD)</td>
</tr>
<tr>
<td>Control n=20</td>
<td>40.05± 2.31(SD)</td>
<td>7.92± 2.53(SD)</td>
<td>43.40± 2.62(SD)</td>
<td>54.20±1.96(SD)</td>
<td>1.25±0.54 (SD)</td>
<td>1.10±0.70 (SD)</td>
</tr>
<tr>
<td>Remark</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
<td>P&gt;0.05</td>
<td>P&lt;0.05</td>
</tr>
</tbody>
</table>
Table II: Values of PCV, total white cell count and differential white cell count of SCD patients in vaso-occlusive crisis state and control subjects.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>PCV %</th>
<th>Total x 10^9</th>
<th>Neutrophil</th>
<th>Lymphocyte %</th>
<th>Eosinophil %</th>
<th>Monocyte %</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCD in crisis state n=10</td>
<td>17.30±2.99(SD) 26.34±8.86(SD)</td>
<td>61.30±19.29(SD) 34.20±18.31(SD)</td>
<td>2.50±1.85(SD)</td>
<td>1.70±0.53(SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control n=20</td>
<td>40.05±2.31(SD) 7.92±2.53(SD)</td>
<td>43.40±2.67(SD) 54.20±2.96(SD)</td>
<td>1.25±0.54(SD)</td>
<td>1.10±0.7(SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Remark</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
</tr>
</tbody>
</table>

Table III: Values of PCV, total white cell count and differential white cell count of SCD patients in steady and vaso-occlusive crisis states.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>PCV %</th>
<th>Total x 10^9</th>
<th>Neutrophil</th>
<th>Lymphocyte %</th>
<th>Eosinophil %</th>
<th>Monocyte %</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCD in steady state n=30</td>
<td>22.70±4.35(SD) 15.14±4.33(SD)</td>
<td>50.60±13.68(SD) 45.80±5.55(SD)</td>
<td>1.63±1.28(SD)</td>
<td>2.10±1.85(SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCD in crisis state n=10</td>
<td>17.30±2.99(SD) 26.34±8.86(SD)</td>
<td>61.30±19.29(SD) 34.20±18.31(SD)</td>
<td>2.50±1.85(SD)</td>
<td>1.70±0.53(SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Remark</td>
<td>P&lt;0.10</td>
<td>P&lt;0.10</td>
<td>P&lt;0.10</td>
<td>P&lt;0.10</td>
<td>P&lt;0.10</td>
<td>P&gt;0.10</td>
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</tbody>
</table>

Table IV: Values of PCV, total white cell count and differential white cell count of SCD patients in steady state with bacterial infection and vaso-occlusive crisis state with bacterial infection.

<table>
<thead>
<tr>
<th>Subjects</th>
<th>PCV %</th>
<th>Total x 10^9</th>
<th>Neutrophil</th>
<th>Lymphocyte %</th>
<th>Eosinophil %</th>
<th>Monocyte %</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCD in steady state with bacterial infection n=15</td>
<td>19.27±1.48(SD) 17.87±3.08(SD)</td>
<td>59.67±2.27(SD) 37.80±2.51(SD)</td>
<td>1.33±0.87(SD)</td>
<td>1.20±0.0(SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCD in crisis state with infection n=9</td>
<td>20.40±2.91(SD) 19.50±4.46(SD)</td>
<td>70.10±8.88(SD) 24.11±8.71(SD)</td>
<td>2.33±1.33(SD)</td>
<td>1.30±1(SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Remark</td>
<td>P&gt;0.05</td>
<td>P&gt;0.05</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
<td>P&lt;0.05</td>
</tr>
</tbody>
</table>

Table V: Frequency of Micro-Organisms Isolated Micro-Organisms Isolated Frequency.

<table>
<thead>
<tr>
<th>Micro-Organism</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staphylococcus aureus</td>
<td>7</td>
</tr>
<tr>
<td>Klebsiella aerogenes</td>
<td>6</td>
</tr>
<tr>
<td>Escherichia coli</td>
<td>5</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>3</td>
</tr>
<tr>
<td>Streptococcus pyogenes</td>
<td>3</td>
</tr>
</tbody>
</table>

DISCUSSION
The results obtained from the assay of PCV, total white cell count and differential white cell count of thirty patients in steady state were compared with that of ten SCD patients in crisis state and also with that twenty normal Hb AA individuals; also the result of fifteen SCD patients in steady state with bacterial infection was compared with that of nine SCD patients in vasocclusive crisis state with bacterial infection.

The normal Hb AA individuals had mean PCV of 40.05±2.31% and this is similar to the value given by Dacie and Lewis (1994), a value of 41+5% was established. Mean PCV values of 22.70±4.35% and 17.30±2.99% were observed in steady and in crisis states respectively. These findings agree with the observation made by Kaine (1982) in Nigerian children who observed mean PCV values of 25.30% and 20.20% in steady and in crisis states respectively. The mean PCV values of SCD patients in steady and in vaso-occlusive crisis states were significantly different (P<0.05) from normal control subjects.

Patients in SCD steady state had a mean total leucocyte count of 15.14±3.33x10^9/L, while those in vaso-occlusive crisis had theirs as 26.34±8.86 x 10^9/L. These findings are similar to those of Kaine (ibid) who observed 13.70x10^9/L and 30.50 x 10^9/L for steady and vaso occlusive crisis states respectively. This finding also agrees with observation made by Serjeant (1992) and Karayalcin (1975) that total white cell bands in steady state, painful vaso-occlusive crisis and bacterial infection in both steady and crisis states.

Patients in SCD, steady state had a mean lymphocyte count of 45.80±15.55%, those in vaso-occlusive crisis

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states had theirs as 34.20±18.31% and those in steady state with bacterial infection and vaso-occlusive crisis states with bacterial infections had a main lymphocyte count of 37.80±2.51% and 24.11±8.71% respectively. Normal control subjects had mean lymphocyte count of 54.20±2.96%. There is lymphocytosis in normal subjects when compared with patients in different states of SCD. Other workers made similar report on lymphocytosis in normal subjects. These include the report made by (De Gruchy, 1978 Thompson and Proctor, 1984) they attributed this lymphocytosis to increase demand for antibody production in children.

There was no much difference in eosinophil and monocyte count in the different states of SCD when compared to control. There was no available literature found comparing eosinophil and monocyte count in SCD patients with the control.

The predominant organisms isolated were Staphylococcus aureus, Klebsiella aero genes and Escherichia coli. This result disagrees with the observation made by Charache et al., (1989) who observed that Streptococcus pneumonia. Haemophilus influenzae and Streptococcus pyogones were the predominant cause of bacterial infection in SCD patients. Embury et al. (1994) also observed that Streptococcus pneumonia. Neisseria meningitidis and Staphylococcus aureus were the predominant cause of bacterial infection in SCD patients.

The reason for leucocytosis in children with sickle cell anaemia is unclear but may be related to decreased margination or increased bone marrow myeloid activity (Buchanan and Glader, 1978).

Finally, the results suggest that a simple test like the total and differential white cell count may be useful for helping to identify bacterial infection in the febrile child with sickle cell disease. Bacterial infection should be checked as it is the commonest cause of the early morbidity and mortality in SCD children. I also suggest that simple urinalysis which may be followed up with culture should be included as part of routine check up for SCD patients when bacterial growth is present, also treatment with the right antibiotics should proceed immediately.

REFERENCES