HAEMATOLOGICAL INDICES & ELECTROLYTE STATUS IN SICKLE CELL DISEASE
AT RURAL HOSPITAL OF CENTRAL MAHARASHTRA

Ajay W Meshram1, Priyanka A Bhatkulkar1, Ruchir Khare1, Komal Pazare2
1 Department of Biochemistry, Jawaharlal Nehru Medical College, Sawangi (M), Wardha, Maharashtra, India
2 Department of Physiology, Jawaharlal Nehru Medical College, Sawangi (M), Wardha, Maharashtra, India

Correspondence to: Ajay W Meshram (meshram.ajay66@yahoo.in)

DOI: 10.5455/ijmsph.2014.230820141 Received Date: 14.07.2013 Accepted Date: 23.08.2014

ABSTRACT
Background: Sickle cell disease is one of the most common autosomal recessive diseases in the world caused by a single nucleotide substitution (GTG - GAG) and is located at the sixth codon of the human-globin gene.
Aims & Objectives: This study was designed to determine the haematological values & electrolyte status that can be used in monitoring the status and management of sickle cell anaemia patients.
Materials and Methods: This study is an observational study done in 50 sickle cell patients in steady state, at Acharya Vinoba Bhave Rural Hospital, Sawangi Meghe, Wardha, Maharashtra, India. The complete blood counts (CBC) were analyzed using the Automated Coulter Counter & serum electrolytes estimated on ABL 800 Radiometer.
Results: Haemoglobin concentration and the MCV was significantly low (≤0.05) in sickle cell patients as compared to controls. TLC- ra-nts with SCD suffer repeated vaso-occlusive events characterized by ischemia-reperfusion injury and inflammation, in which red blood cells (RBCs) and white blood cells (WBCs) play a key role.[4] Although red cell sickling is more prominent during crisis, continuous sickling does occur at a lower rate in the steady state. Hence, a certain proportion of sickled cells are always present in the circulation of SCA patients even in steady state.[3]

It has been demonstrated that sickling is accompanied by an intra-erythrocytic loss of potassium and gain of sodium, thus creating disequilibrium in the ionic strength across the cell membrane.[4] Biochemical abnormalities have been associated with sickle cell disease. However, there is a paucity of information on the roles of these ions in the pathogenesis and management of sickle cell disease.[5]

Based on this background, this study was aimed to determine the routine haematological indices along with less commonly determined variables such as red cell indices and red cell distribution width of adult patients with sickle cell anaemia, in addition to assess electrolyte balance of the patients suffering from SCD.

Materials and Methods

50 patients, age group of 16-50 years, with diagnosed SCD, attending the outpatient clinic at the Acharya Vinoba Bhave Rural Hospital, Sawangi Meghe, Wardha, Maharashtra, were taken as subjects. All subjects studied were diagnosed as SCA based on positive sickling tests, and haemoglobin electrophoresis at a pH of 8.6 on cellulose acetate paper.

Equal number of age and sex matched sickle cell negative subjects were selected as a control group. All procedures were conducted with due consent of the subjects and institutional ethics committee approval.

Introduction

Sickle cell disease, one of the most common autosomal recessive diseases in the world, is caused by a single nucleotide substitution (GTG - GAG) at the sixth codon of the human-globin gene. This point mutation results in well-known haemolytic and vaso-occlusive complications that characterize sickle cell disease (SCD).[1]

In patients with sickle cell disease (SCD), because of the instability and insolubility of HbS, polymerization of the deoxy-HbS renders the red blood cells (RBCs) non-deformable to traverse the microcirculation. Consequently, patients with SCD suffer repeated vaso-occlusive events characterized by ischemia-reperfusion injury and inflammation, in which red blood cells (RBCs) and white blood cells (WBCs) play a key role.[4] Although red cell sickling is more prominent during crisis, continuous sickling does occur at a lower rate in the steady state. Hence, a certain proportion of sickled cells are always present in the circulation of SCA patients even in steady state.[3]

5 ml of venous blood was collected by clean venipuncture from each patient via the ante-cubital vein, using a plastic syringe with minimum stasis, into commercially prepared concentrations of sequestrene Ethylene Di-amine Tetraacetic Acid (EDTA) bottles & 2 ml in the plain bulb. After estimation of haematological parameters, the plasma & serum were separated by centrifuging the blood for 10 min.

The complete blood counts (CBC) were analyzed using the automated COULTER (Beckman Coulter). The CBC included haemoglobin (Hb) haematocrit (HCV), total white blood cell count (TWBC), platelet count, mean cell volume (MCV), mean cell haemoglobin (MCH), mean cell haemoglobin concentration (MCHC) and red cell distribution width (RDW). Serum sodium and potassium were estimated by ABL800 radiometer, by ion selective method, using indirect potentiometry.

Data was analyzed using SPSS version 16.0. The results were expressed as mean ± standard deviation and student t-test was used to compare the values of cases to controls. p value ≤ 0.05 was considered to be significant.

Results

50 patients of SCD (Group-1) and 50 age and sex matched healthy control (Group-2) were taken and all the parameters were analysed in both the group. The haematological parameters and electrolyte status of both the groups have been shown in Table-1.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group-1</th>
<th>Group-2</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb (g/dl)</td>
<td>10.3 ± 0.7</td>
<td>12.9 ± 0.34</td>
<td>≤ 0.05*</td>
</tr>
<tr>
<td>TLC (×10⁹/µl)</td>
<td>93.0 ± 13.0</td>
<td>80.0 ± 31.0</td>
<td>≥ 0.05</td>
</tr>
<tr>
<td>Platelet (×10⁹/µl)</td>
<td>233.0 ± 31.0</td>
<td>171.0 ± 23.0</td>
<td>≤ 0.05*</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>83.0 ± 3.0</td>
<td>83.0 ± 3.0</td>
<td>≤ 0.05*</td>
</tr>
<tr>
<td>MCHC (g/100ml)</td>
<td>34.0 ± 1.1</td>
<td>32.0 ± 0.7</td>
<td>≥ 0.05</td>
</tr>
<tr>
<td>RDW (%)</td>
<td>10.7 ± 0.89</td>
<td>2.1 ± 0.24</td>
<td>≤ 0.05*</td>
</tr>
<tr>
<td>Sodium (mEq/L)</td>
<td>127.0 ± 2.1</td>
<td>141.0 ± 1.21</td>
<td>≤ 0.05</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>4.7 ± 0.17</td>
<td>3.6 ± 0.17</td>
<td>≤ 0.05</td>
</tr>
</tbody>
</table>

*Values are expressed in Mean ± SD. * statistically significant.

There was a statistically significant difference in the haemoglobin concentration (P<0.05), mean cell volume (P<0.01), red cell distribution width (P<0.05) and platelet count between two groups. However TLC, MCH & MCHC were not significantly different. Serum sodium levels were significantly lowered in patients of SCD (127.0 ± 2.1 mEq/L), as compared to control group (141.0 ± 1.21 mEq/L), while serum potassium levels were significantly raised in patients of SCD (4.7 ± 0.17 mEq/L), as compared to control group (3.6 ± 0.17 mEq/L).

Discussion

In our study, the haemoglobin level was significantly low in patients of SCD. Haemoglobin concentration remained significantly low in SCA patients. The SCD patients suffer from continuous hemolysis of red cells, with a short survival rate of the erythrocytes between 12-14 days. Hence, the haemoglobin values are usually lower than normal healthy individuals.

The mean total white blood cell count (WBC) in this study were similar to other earlier studies. Studies reported rise in TLC in patients of SCD, as compared to control groups. But in our study, it was not statistically significant (>0.05). Studies indicate that neutrophils, in addition to factors such as plasma proteins, endothelial abnormalities, and sickle RBC, could have a pivotal role in the initiation and/or propagation of vaso-occlusion in SCD. WBC counts are correlated with the severity of crisis and risk of premature death. Insignificant rise in leucocyte count shows the steady state of the patients of our study.

The mean platelet count for steady state value is similar to other studies. The platelet count in patients of SCD was significantly higher than control (P≤0.05) – which may be due to loss of splenic platelet pool function in adult sickle cell patients consequent upon autosplenectomy. This result is in agreement with previous studies. Platelet aggregate formation activity is normal in SCD patients in steady state, but abnormally high in each SCD patient with vaso-occlusive crisis. It is a matter of further research that whether increased platelet aggregate formation during acute thrombotic event is primarily responsible for the initiation of the event, or is a secondary phenomenon in response to tissue injury.

In this study, the mean corpuscular volume (MCV) value was significantly lower in cases of SCD, as compared to control groups – which is similar to previous studies conducted by Omoti C E, Ahmed S G & Tripette J. In contrast, there was no significant difference in the MCH & MCHC values in present study. The pattern of haematocrit in this study revealed that our subjects suffered from mild to moderately severe anaemia.

The RDW, which is a measure of erythrocyte anisocytosis, was significantly higher in Group-1 as compared to control (P<0.05). This is in agreement with previous studies, which showed that SCD is associated with marked anisocytosis. This may be because of more
In contrast to study of Ibe E O,\textsuperscript{44} in our study, we observed that the sodium concentration was much lower in the cases, as compared to control group. It indicated hyponatremia, which was similar to the study reported by Agoreyo F O\textsuperscript{45} and Ibe E O.\textsuperscript{44} Dehydration can be one of the causes of sodium movement into the sickle cell. During crisis, the dehydration of RBCs can take place, which could be a possible cause of sodium loss from the extracellular fluid into the intracellular fluid.\textsuperscript{14,5,14-16}

In this study, along with haematological indices, the serum sodium and potassium were compared between the control group (HbAA) subjects, and the cases of SCD. It was observed that the sodium concentration was much lower in the cases, as compared to control group. It indicated hyponatremia, which was similar to the study reported by Agoreyo F O\textsuperscript{45} and Ibe E O.\textsuperscript{44} Dehydration can be one of the causes of sodium movement into the sickle cell. During crisis, the dehydration of RBCs can take place, which could be a possible cause of sodium loss from the extracellular fluid into the intracellular fluid.\textsuperscript{14,5,14-16}

However, this study is limited by the fact that we have taken patients of steady state and not of crisis state. The picture might have been slightly different if patients of steady state and sickle cell crisis both have been taken. The age group which is considered was also very broad. Other biochemical parameters which can assess oxidative and anti-oxidative properties could have also been included. Further studies including all confounding factors are required to assess the effect of these haematological findings with patients of sickle cell anaemia and its effective management.

Conclusion

Recent therapeutic approaches to SCD focus on attempt to reduce intracellular HbS polymerization by altering the haemoglobin species. So monitoring all the haematological indices has a great diagnostic & prognostic value in patients of sickle cell anaemia either in steady or crisis state. It could be inferred that sodium and potassium level maintenance is necessary in the management of the SCD patients, with a comprehensive medical care and management approach. The health status and life expectancy of these patients can be improved considerably by monitoring these parameters.

References