Assessment of Serum Ferritin, TIBC and Kidney Function Test in Multitransfused Thalassemic Children

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Beta thalassemia is a single gene disorder requiring regular multi-blood transfusions which causes serious side effects, as an overload of iron in the form of ferritin. We determined whether thalassemia could account for abnormal cation transport system. Traditionally serum iron, ferritin, TIBC and the % saturation, an indirect measurement of transferrin, have been used to determine iron status. The present study included 100 multitransfused thalassemic children and 35 control, in the age group of 3-10 years. Serum ferritin was evaluated by ELISA method, urea by Berthelot method, creatinine by Jaffes reaction & TIBC by Ramsay Dipyridyl method. It has been found that the serum ferritin level was significantly increased in thalassemic children as compared to control due to the regular blood transfusions. Serum kidney enzymes were also deranged. Serum ferritin, urea & creatinine levels increased and TIBC decreased. As the number of blood transfusion increases, the serum ferritin, urea & creatinine also significantly increases while TIBC decreases. Hence, it can be concluded from the present study that increased serum ferritin, urea, creatinine and decreased TIBC levels are associated with multi-transfused children.

Key words: Beta thalassemia, Kidney function test, Multi-blood transfusions.

Thalassemia major is the commonest hereditary form of transfusion dependent anemia. Thalassemia syndrome is characterized by defect in the synthesis of one or more of the globin chains that form the hemoglobin tetramer. In β-thalassemia the production of β chains is inadequate. β-thalassemia major is a single gene disorder requiring regular multi blood transfusion which causes serious side effects as an overload of iron in the form of ferritin. Excess or free iron can catalyze the formation of very toxic compounds such as OH~ radicals, through Fenton reaction which causes oxidative damage. This oxidative damage causes the pathogenesis of the membrane abnormalities observed in beta thalassemia. Iron overload may cause deposition of iron in hepatic parenchyma and other tissues like kidney and pancreas within a year of transfusion. As the iron overloading progresses the capacity of transferrin to bind and detoxify the iron may exceed and now non transferrin bound fraction of plasma iron may promote the generation of free hydroxyl radicals, propagators of oxygen related damage. The free radicals, generated can damage cellular membrane proteins and DNA resulting in wide ranging impairment in cellular function and integrity. This manifests as cirrhosis & kidney dysfunction which is most common mode of death.
Assessment of S. Ferritin level can give idea regarding starting of chelation therapy, which will reduce the concentration of Ferritin and is effective in preventing iron induced tissue injury and prolong life expectancy. Present work has been designed to assess the effect of the multitransfusion on various organs through estimation of S. Ferritin, TIBC, urea, and creatinine.

MATERIAL AND METHODS

The study was carried out at the Department of Biochemistry, with the collaboration of Department of Pediatrics. Volunteer patients diagnosed with Thalassemia Major in the clinics of the department were selected for the study. Blood was obtained from 100 morbid and 25 healthy control children. All were in the age group 3-18 years.

Inclusion criteria
- All patients were suffering from beta thalassemia major.
- All patients had received more than 15 transfusions.
- The patients who were suffering from any major disease were excluded.

The blood collected was allowed to clot and then centrifuged to obtain serum for the estimation of biochemical parameters.

Urea were estimated by Berthelot method10. Creatinine by Jaffes reaction11. TIBC by Ramsay Dipyridyl Method12 and serum Ferritin was estimated by ELISA method13.

Values have been expressed as Mean ± SD. The results were analyzed using Student’t’ test. P<0.05 was considered as significant.

Table 1. Serum values of all biochemical parameters of control and morbid groups

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Parameters</th>
<th>Control (µg/l)</th>
<th>Thalassemic (µg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Ferritin</td>
<td>79.04 ± 20.11</td>
<td>4228.92 ± 1827.40</td>
</tr>
<tr>
<td>2.</td>
<td>TIBC</td>
<td>342.34 ± 47.99</td>
<td>323.37 ± 37.0</td>
</tr>
<tr>
<td>3.</td>
<td>Urea</td>
<td>30.20 ± 8.24</td>
<td>48.20 ± 11.20</td>
</tr>
<tr>
<td>4.</td>
<td>Creatinine</td>
<td>0.65 ± 0.20</td>
<td>1.30 ± 0.30</td>
</tr>
</tbody>
</table>

Table 2. Serum values according to ferritin level in thalassemic group

<table>
<thead>
<tr>
<th>S. Ferritin Level µg/l</th>
<th>No. of patients</th>
<th>Mean ± SD TIBC [µg/dl]</th>
<th>Urea [mg/dl]</th>
<th>Creatinine [mg/dl]</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. 720 – 4000(&lt;4000)</td>
<td>48</td>
<td>324.0 ± 42.3</td>
<td>47.3 ± 10.24</td>
<td>1.1 ± 0.10</td>
</tr>
<tr>
<td>2. 4000 - 8000(&gt;4000)</td>
<td>52</td>
<td>322.7 ± 32.1</td>
<td>49.0 ± 11.24</td>
<td>1.3 ± 0.20</td>
</tr>
</tbody>
</table>

Table 3. Mean values According to No. of Blood transfusions

<table>
<thead>
<tr>
<th>No. of blood transfusions</th>
<th>No. of patients</th>
<th>Mean ± SD TIBC [µg/dl]</th>
<th>Ferritin [µg/l]</th>
<th>Urea [mg/dl]</th>
<th>Creatinine [mg/dl]</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 100</td>
<td>60</td>
<td>327.5 ± 39.4</td>
<td>2990.5 ± 109.24</td>
<td>46.28 ± 8.24</td>
<td>1.2 ± 0.10</td>
</tr>
<tr>
<td>&gt; 100</td>
<td>40</td>
<td>317.1 ± 33.0</td>
<td>6086.4 ± 856.6</td>
<td>49.10 ± 9.28</td>
<td>1.3 ± 0.20</td>
</tr>
</tbody>
</table>
RESULTS AND DISCUSSION

The study group comprised of 100 children of beta thalassemia. In present study it was found that the serum ferritin level was significantly (p<0.001) increased in thalassemic children as compared to control (Table 1), due to the regular blood transfusions. It has been previously reported by Pootrakul (1981), George (1994), Fargion (1982), Dubey (2004).

As ferritin level increased, urea & creatinine also increased and TIBC decreased (Table 2). It was also observed that as the number of blood transfusions increases, the serum ferritin concentration was also significantly (p<0.001) increased. Urea & creatinine also increases and TIBC decreases (Table 3). Ali et al. (2008) reported that, in 50 children with thalassemia had higher serum creatinine and urea values.

Mean serum value of TIBC was found to be decreased as compare to control group. It has been previously reported by CLP (1995), Finch (1982) & Tenchben (1991) that the decrease TIBC level are associated with the iron overload or thalassemic patients.

Jain (1983), (Gunn, 1972), Oliviero (1994) reported that patients with more transfusions have lower TIBC levels while urea, creatinine and ferritin level grater than the patients who had less transfusions.

CONCLUSION

It can be concluded from the present study that increased serum ferritin, urea, creatinine and decreased TIBC levels are associated with multi-transfusions in children. Thalassemia major is a serious genetic disease but its diagnosable treatable and curable. Multi-transfusions lead to iron overload in the form of ferritin which may be the cause for the development of the alteration of the various biochemical parameters. Timely intervention with chelation therapy can decrease the effect of iron over load and increase the life expectancy of the patients.

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REFERENCES