The role of glucose-6-phosphate dehydrogenase deficiency in anaemia of haemodialysis patients

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Background: Chronic kidney failure or chronic kidney disease is a slowly progressive loss of renal function over a period of months or years and defined as an abnormally low glomerular filtration rate, which is usually determined indirectly by the urea and creatinine level in blood serum. CRF that leads to severe illness and requires some form of renal replacement therapy (such as dialysis) is called end-stage renal disease (ESRD). (Ruggenenti et al., 1998; Ruggenenti et al., 1999). If the kidney function dips too low, the best recommend treatment is dialysis. There are two types of dialysis: haemodialysis and peritoneal dialysis. Either of these can bide time until a possible kidney transplant. (Limido and Malberti, 2005). G6PD deficiency is an inherited condition in which the body doesn't have enough of the enzyme which helps red blood cells function normally. This deficiency can cause hemolytic anemia, usually after exposure to certain medications, foods, or even infections. Patients with both acute and chronic renal failure had significantly low G-6-PD. After adequate dialysis or renal transplantation the G-6-PD can undergo an adaptive alteration which however appears reversible. (Limido and Malberti, 2005; Carson et al., 1996). The aim of this study To determine the frequency of G6PD deficiency among Sudanese haemodialysis patients who are treated in different hospitals in Khartoum state and to evaluate the role of G6PD deficiency in anaemia of haemodialysis patients.

Methods: Sixty five patients with ESRF exposed to different periods of haemodialysis were the target population for this study. 2.5 ml of whole blood collected in ethylene di-amine tetra acetic acid (EDTA) containers. CBC using Sysmex™ Kx21n, fully automated haematological analyzer and reticulocyte count test were done. Methaemoglobin reduction test used as screening test for G6PD deficiency. Results: Results showed that 60% of patients were found to have G6PD deficiency, the mean of Hb level, PCV and RBCs count were found 10.5g/dl, 30% and 3.1x10^12 respectively, while in those without G6PD deficiency, 11.1g/dl, 32% and 3.5x10^12 respectively. Reticulocyte count was 5.9 in G6PD deficiency and 5.0 in non deficiency patients. Conclusion: This study concluded that G6PD deficiency was found in haemodialysis patients with Hb level, PCV, RBCs count lower than those without G6PD deficiency and reticulocyte count was found increased in patients with G6PD deficiency than those with normal G6PD activity.

Keywords: Methaemoglobin, haemodialysis, anaemia.

INTRODUCTION

Chronic renal disease (CKD) is defined as kidney damage with structural and/or functional abnormalities or a glomerular filtration rate (GFR) <60 mL/min/1.73 m², or both, for ≥3 months. (Chauhan et al., 1982). Anemia is an almost invariable manifestation of chronic renal failure, it appears early in the course of CKD, worsening as it
progresses. The term of anemia of chronic renal insufficiency refers to that anemia resulting directly from failure of the endocrine and excretory functions of the kidney. (Chauhan et al., 1982). It is describe as normocytic normochromic anaemia. Generally there is a 2g/dl fall in haemoglobin level for every 10mmol/l rise in blood urea . There is impaired red cell production as result of defective erythropoietin secretion. Uremic serum has also been shown to contain factors which inhibit proliferation of erythroid progenitors but, in view of the excellent response to erythropoietin in most patients, the clinical relevance of these is doubtful. Variable shorting of red cell life span occurs and in serve uremia the red show abnormalities include (spurs) and “burr” cells .Increased red cell 2, 3-diphosphoglycerate (2,3DPG) levels in response to anaemia and hyper phosphhtemia result in decrease oxygen affinity and a shift of hemoglobin oxygen dissociation curve to the right, which is augmented by uremic acidosis. The patient’s symptoms are usually mild for the degree of anaemia. (NAAC, 2006).

Other factors may complicate the anaemia of chronic renal failure. These include the anaemia of chronic disorders, iron deficiency from the blood loss during dialysis or cause by bleeding because of defective platelet function, and folate deficiency in some chronic dialysis patients. Aluminum excesses in patients on chronic dialysis also inhibit the erythropoiesis. (NAAC, 2006).

About 60% of patients with end stage renal insufficiency are treated with maintenance haemodialysis. It is an effective means of controlling most of symptoms and biochemical manifestations of renal failure, but erythropoietin secretion does not improve, and anemia continues to be major source of morbidity. (NAAC, 2006).

G6PD deficiency is the lack of glucose-6-phosphate dehydrogenase (an enzyme present in red blood cells) in the blood, which can cause a hemolytic anemia. Red blood cells carry oxygen in the body and G6PD protects these cells from natural oxygen chemicals that may build up when you have a fever or take certain medications and foods. If there are too many of these chemicals, foods, or even infections, they can destroy the red blood cells, causing hemolytic anemia. G6PD deficiency has been suggested as cause of anaemia of CRF by some researchers. (Lee et al., 1999; Adams et al., 1993; Takizawa et al., 1986).

**Objective**

The main objectives of this study are to determine the frequency of G6PD deficiency among Sudanese haemodialysis patients, to measure the CBC and reticulocyte count and to evaluate the role of G6PD deficiency in anaemia of haemodialysis patients.

**METHODS**

This is a descriptive cross sectional study conducted in Khartoum State, 65 samples from Patients with ESRF at different ages, from different tribes and sex, exposed to different periods of haemodialysis were the target population for this study. 2.5ml of whole blood collected in (EDTA) containers. CBC was done using a Sysmex™ Kx21n, fully automated haematological analyzer, reticulocyte count test was done. Methaemoglobin reduction test used as screening test for G6PD deficiency.

**RESULTS**

Blood samples were collected from 65 haemodialysis patients who were treated in different hospitals in Khartoum state, 43 male and 22 female at different ages and from different residence and tribes. All patients were screened for G6PD deficiency and CBC was performed to evaluate the role of G6PD deficiency in anaemia of haemodialysis patients.

Results of this study showed that 60% of patients were found to have G6PD deficiency and 40% were normal. 59% of patients with G6PD deficiency were male and 41% females as seen in table (1)

<table>
<thead>
<tr>
<th>Gender</th>
<th>G6PD deficiency No %</th>
<th>Non-G6PD deficiency No %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>23 35.4</td>
<td>20 30.8</td>
</tr>
<tr>
<td>Females</td>
<td>16 24.6</td>
<td>6 9.2</td>
</tr>
<tr>
<td>Total</td>
<td>39 60</td>
<td>26 40</td>
</tr>
</tbody>
</table>

Reticulocyte count was 5.9 in G6PD deficiency and 5.8 in non deficiency patients as shown in figure (1)

**DISCUSSION**

G6PD is an enzyme in the pentose phosphate pathway, a
metabolic pathway that supplies reducing energy to erythrocytes by maintaining the level of the co-enzyme NADPH which plays a role in protecting cells from potentially harmful molecules called reactive oxygen species. (Carson et al., 1996). Increased oxidative stress is now considered one of the major risk factor in CRF patients, particularly those treated by haemodialysis. Various studies have documented an increased production of reactive oxygen species in CRF patients which may be related to uremia. (Doald et al., 1988).

This study revealed that 60% of haemodialysis patients have G6PD deficiency and 40% were normal. This finding was consistent with the findings reported by Chauhan DP et al who reported that haemodialysis patients had significantly low G-6-PD. (Lucchi et al., 2005). Present study consistent with the findings of Weinstein T et al who investigated the evidence of impaired defense mechanisms against oxidative stress in haemodialysis patients and found that Plasma Glutathione reductaes was lower in patients as compared to healthy controls (P<0.005) and there was an inverse correlation between Glutathione reductaes and the degree of haemolysis. (Weinstein et al., 2000).

This study reveals that, no statistically significant difference were found in Hb level, PCV, WBCs and RBCs count between patients with G6PD deficiency and those with normal G6PD activity, 75% of patients with G6PD deficiency were treated with EPO which was found by Kanbak G et al to have a positive antioxidant effect in haemodialysis patients, so this may be the reason for the absence of difference in these parameters.

Leucocytes count was found lower in patients with G6PD deficiency but the difference was not statistically significant, recently there is no another study deals with the correlation of G6PD deficiency and each of leucocyte count and Heinz bodies in haemodialysis patients.

CONCLUSION

This study concluded that more than 50% of the study population was G6PD deficient. Patients with G-6-PD deficiency had lower Hb level, PCV, WBCs and RBCs count than patients without G6PD.
deficiency but the different was not significant. Reticulocyte count was found increased in patients with G6PD deficiency than patients with normal G6PD activity but the different was not statistically significant.

REFERENCES


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