Original Research Article

Comparative study of HbA1c level among iron deficiency diabetic patients with non-iron-deficiency diabetic patients among Karaikal population

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Abstract

Introduction: Iron deficiency anemia is the most common form of anaemia in India. Haemoglobin A1c (HbA1c) is used in diabetic patients as an index of glycemic control reflecting glucose levels of the previous 3 months. Glycosylated hemoglobin (HbA1c) is used as a gold standard for monitoring glycemic control and as a predictor of diabetic complications. Conditions that affect erythrocyte turnover affect HbA1c concentration. Although many forms of anemia are associated with lowering of HbA1c, iron deficiency anemia tends to increase HbA1c.

Aim of the study: To compare the HbA1c level and hemogram levels among iron deficiency diabetic patients with non-iron-deficiency diabetic patients.

Materials and methods: 25 Non-iron deficiency diabetic patients (controls) and 25 iron deficiency age matched diabetic patients (cases) with good glycemic control were enrolled in the study. Absolute HbA1c levels were measured in both the patients. The levels of hemoglobin, mean corpuscular hemoglobin (MCH), hematocrit, mean corpuscular volume (MCV), mean corpuscular hemoglobin concentration (MCHC), were measured by an automated counter these values were compared with those in the control population.

Results: The mean baseline HbA1c level in iron deficiency diabetic patients (8.60) was significantly higher than that of controls (4.48) however, after 3 months of iron deficiency treatment, a significant decline from 8.60 to 6.5.

Conclusion: Our study proved that iron deficiency anemia has a straight forward correlation with...
HbA1c level in diabetic patients. Moreover, with correction of iron deficiency in the anemic diabetic patients and HbA1c levels decrease.

**Key words**
Iron deficiency anemia, Hemoglobin A1c, HbA1c, Glycated hemoglobin.

**Introduction**
Iron deficiency anemia is the most common form of anemia in India. Many studies revealed a relationship between iron deficiency anemia and HbA1c levels and attempted to explain the alteration in HbA1c levels in iron deficiency anemia on the basis of both modifications to the structure of hemoglobin levels of HbA1c in old and new red blood cells [1]. According to the recent American Diabetes Association Guidelines, HbA1c levels should be maintained below 7% in all diabetic patients in order to prevent the development of microvascular complications [2]. Also as per the recent recommendations by the IDF and AACE, the optimum level at which HbA1c should be maintained in diabetics has been brought down to the target of 6.5%. HbA1c levels are not affected by blood glucose alone. They are also altered in hemolytic anemias, Hemoglobinopathies, acute and chronic blood loss, pregnancy and uremia, Vitamin B12, folate and iron deficiency anemia have also been shown to affect HbA1c levels. Throughout the circulatory life of the red blood cell glycol hemoglobin is formed continuously by addition of glucose to the N-terminal of the hemoglobin beta-chain. This process is non-enzymatic and reflects the average exposure of hemoglobin to glucose over a period of nearly 2-3 months. Glycohemoglobin has been defined as the fast fraction hemoglobin (HbA1a, A1c) which elute first during column chromatography with cation exchange resin [3]. HbA1c levels are not affected by blood glucose levels alone. Any condition that shortens the life span of erythrocytes is likely to decrease HbA1c level. They are acute or chronic blood loss, sickle cell anemia, thalassemias, hemolytic anemia, aplastic anemia, splenectomy, pregnancy, chronic kidney diseases, vitamin-B12, and folate deficiency anemia. Some studies show that HbA1c levels are increased in iron deficiency anemia and attempted to explain on the basis of both modifications to the structure of hemoglobin and levels of HbA1c in old and new red blood cells [4].

**Materials and methods**
The cases were selected from A Private Set Up Clinic at Karaikal District Between (2016-2017). A blood sample was obtained from 50 subjects (both men and women) aged between 30-50 Yrs. among them 30 were women and 20 men. The patients were selected based on their hemoglobin levels (Hb< 12gm/dl, MCV <80 fl and MCH<26 pg/dl) and on their peripheral blood smears (mostly microcytic hypochromic). Patients with a history of acute or chronic blood loss, hemolytic anemia, hemoglobinopathies, kidney diseases, pregnancy, chronic alcohol ingestion were excluded. The levels of hemoglobin, mean corpuscular hemoglobin (MCH), hematocrit, mean corpuscular volume (MCV), mean corpuscular hemoglobin concentration (MCHC), platelet count, total leucocyte count (TLC) and differential leucocytes count (DLC) were measured by an automated counter. Peripheral blood smear examinations were performed to define the anemia type. Those with predominantly microcytic indices (MCV <80 fl), hypochromic indices (MCH < 26 pg/cell) were considered to have iron deficiency anemia. HbA1C levels were measured by using the glycol hemoglobin ion exchange resin method kit Quo-Lab® A1c [5, 6].

**Results**
Fasting blood glucose level was found to more in iron deficiency diabetic patient when compared to non-iron-deficiency diabetic patients.98±8.42 and 84±8.6 of p Value > 0.05. Post prandial blood glucose was found to more in iron deficiency
diabetic patient when compared to non-iron-deficiency diabetic patients 113±6.4 and 93±5.82 p value> 0.05. The Mean Hemoglobin level was found to more in the non-iron diabetic patient when compared to iron deficiency diabetic patients 7.74±1.98 and 13.80±0.54 p value< 0.01. The mean hematocrit level was found to more in the iron-diabetic patient when compared to iron deficiency diabetic patients 21.99±5.42 and 33.57±1.92 p value< 0.01. Mean corpuscular volume was found to be more in the non-iron diabetic patient when compared to iron deficiency diabetic 63.40±10.66 and 87.20±6.76 p value< 0.01. The mean corpuscular hemoglobin was found to be more in the iron diabetic patient when compared to iron deficiency diabetic patients 18.66±4.46 and 31.01±2.40 p value < 0.01. The mean HBA1C levels in anemic patients where was found to be more when compared non-iron-deficiency diabetic patients 8.91±0.47 and 6.83±0.39 p< 0.01 (Table – 1).

**Table – 1:** Comparison of the HbA1c level and hemogram parameters level among iron deficiency diabetic patients and non-iron-deficiency diabetic patients.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>IDA DM patients (n=25) Mean + SD</th>
<th>Non - IDA DM patients (n=25) Mean + SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Years)</td>
<td>32.6±8.97</td>
<td>42.85±13.57</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>FBS (mg/dl)</td>
<td>98±8.42</td>
<td>84±8.6</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>PPBS (mg/dl)</td>
<td>113±6.4</td>
<td>93±5.82</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Hb (gm/dl)</td>
<td>7.74±1.98</td>
<td>13.80±0.54</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>HCT (%)</td>
<td>21.99±5.42</td>
<td>33.57±1.92</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>63.40±10.66</td>
<td>87.20±6.76</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>MCH (pg/cell)</td>
<td>18.66±4.46</td>
<td>31.01±2.40</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>HbA1C (%)</td>
<td>8.91±0.47</td>
<td>6.83±0.39</td>
<td>&lt; 0.01</td>
</tr>
</tbody>
</table>

**Discussion**

Iron deficiency anemia is the most common form of anemia. HbA1c is glycated hemoglobin that can be used to assess the glycemic status of the diabetic patient for the previous 3 months. Besides blood sugar, other conditions such as hemolytic Anemias, hemoglobinopathies, acute and chronic blood loss, pregnancy, and uremia have been shown to affect HbA1c levels. Recently, researchers have become interested in studying HbA1c levels in more commonly encountered anemias like iron deficiency anemia [7]. Certain studies have been done which show that HbA1c levels are affected in hemolytic anemias. In one of these studies done by Horton and Huisman showed that HbA1c is decreased due to the reason that the life span of the RBC's is reduced. So, from these studies, it became evident that HbA1c should be taken as a measure of glycemic control only if such disorders are ruled out [8]. However, interest further arose as to what happens to HbA1c levels in more commonly encountered anemias like iron deficiency anemia. Brooks, et al. showed that HbA1c levels were higher in patients with iron deficiency anemia at baseline and decreased on treatment. The reason speculated by them was that the quaternary structure of hemoglobin gets altered and that, glycation of beta globin chain occurs more readily in the relative absence of iron. Sluiter, et al. later gave a different reason to explain the findings of Brooks et al. Among the 50 patients studied, 30 were female, suggesting that iron deficiency anemia is more common in women [9]. As expected, the mean hemoglobin and mean serum ferritin levels increased in anemia patients over 2 months of iron treatment. None of our patients had nonresponsive iron-deficiency anemia [10]. Our observation of increased HbA1c levels at baseline and its subsequent fall on iron supplementation was in accordance with most of the studies done in the past. There are a number of variable explanations available to explain these findings. This signifies
that as the level of hemoglobin drops with increasing severity of iron deficiency in anemic subjects, at the same time HbA1c levels increase correspondingly. Moreover, with correction of iron deficiency in the anemic subjects, the HbA1c levels decline to near normal values.

**Conclusion**

Our analysis suggests that iron deficiency anemia is unlikely to be a major concern in diagnosing diabetes using concentration of HbA1c according to the American Diabetes Association (ADA) guideline. Our study proved that iron deficiency anemia has a straight forward correlation with HbA1c levels in diabetic patients. Moreover, with correction of iron deficiency in the anemic diabetic patient HbA1c levels decrease. The cause of iron deficiency is mainly nutritional. Further studies in large number sample size are needed to confirm our findings [11, 12].

**References**