Original Research Article

Pattern of dysglycemia among Nigerian adult patients with vitiligo: a 10-year retrospective study

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ABSTRACT

Background: The prevalence of dysglycemia among adult patients with vitiligo is higher than the general population. However, data is scarce in our region regarding this hypothesis. This study was to define the pattern of dysglycemia in adult Nigerian patients with vitiligo.

Methods: The study was conducted retrospectively among vitiligo patients who visited the University of Port Harcourt Teaching Hospital between 1st January 2007 and 31st December 2016. The laboratory characteristics of this patients were evaluated. Data collected irrespective of vitiligo variant were age, sex, and fasting plasma glucose concentrations. Shapiro-Wilk test, descriptive statistics, chi-square test, Fisher’s exact test, and two-sample t-test were used for analysis. The level of $p < 0.05$ was considered statistically significant.

Results: There were a total of 160 vitiligo patients consisting of 55 (34.4%) males and 105 (65.6%) females. The overall patients mean age was $35.6\pm.9$ years (range $19 -61$ years). The mean fasting plasma glucose concentration was $5.3\pm.1$mmol/l. Impaired fasting plasma glucose was detected in 41(26.6%) subjects with female preponderance (female 61.0% versus male 39.0%). Diabetes mellitus was documented in 6 (3.8%) subjects with no sex difference (female 50% versus male 50%).

Conclusions: Dysglycemia is frequent in vitiligo patients. Screening for dysglycemia should be incorporated into the management protocol of patients with vitiligo.

Keywords: Age, Dysglycemia, Nigeria, Sex, Vitiligo

INTRODUCTION

Vitiligo is a noncontagious dermatologic disorder presenting with whitish skin and mucous membrane appearance secondary to the loss of viable pigment-producing melanocytes.1 It has global distribution of about 2% with no racial or sex predilection.2 Some studies have suggested a more female preponderance of vitiligo cases.2,3 Its etiology has remained unknown several decades after it was first described.4,5 Several theories have been documented as possible factors in the evolution of the disorder including genetics, neuro-chemical, auto-toxicity of melanocytes and autoimmune disorders.6 The most widely plausible and accepted of these documented theories is the part of an autoimmune mechanism, evidenced by several autoimmune diseases frequent in vitiligo.5,6 Pernicious anemia, Addison’s disease, rheumatoid arthritis, alopecia areata and autoimmune diseases of the thyroid organ have all been described in association with vitiligo.7,8 Based on the autoimmune mechanisms in vitiligo, several authors have also suggested a distinct higher frequency of dysglycemia in patients with vitiligo relative to the general population.10,11 While some authors have suggested that vitiligo precedes dysglycemia, others have suggested that these
dysglycemia especially diabetes mellitus evokes vitiligo.14,17

However, most of these reports have emanated from abroad with a paucity of data in our region. On that note, this study was designed to define the pattern of dysglycemia in Nigerian vitiligo patients.

METHODS

This was a retrospective, descriptive, cross-sectional analysis of fasting plasma glucose laboratory parameters of vitiligo patients who had visited the Department of Chemical Pathology and Metabolic Medicine of the University of Port Harcourt Teaching Hospital (UPTH), Port Harcourt, Nigeria between January 2007 to December 2016. All vitiligo patients had been diagnosed in the hospital by the specialist dermatologist. Ethical approval with informed consent was not required due to the retrospective nature of the study.

Criteria for inclusion were records of fasting plasma glucose of adult vitiligo patients above 18 years (irrespective of vitiligo type) who presented for fasting plasma glucose test. Criteria for exclusion were records of patients with vitiligo who are either diagnosed or on treatment for dysglycemia, those below 18 years and those whose data are incomplete.

Fasted plasma samples were used for glucose analysis via the glucose oxidase method with reagents sourced from Randox laboratories, United Kingdom. All records from laboratory result sheets and case notes of each vitiligo patient were collected, reviewed and entered into Statistical Package for Social Sciences (SPSS) version 20.

Demographic data (Age and sex), clinical diagnosis (vitiligo), fasting plasma glucose concentrations in mmol/l were collected. A fasting plasma glucose of 3.3-6.0 mmol/l, 6.1-6.9 mmol/l, and >7.0 mmol/l were defined as diabetes mellitus unlikely, impaired fasting glucose, and diabetes mellitus likely respectively.

Statistical analysis

The collected data in SPSS version 20 were analyzed using descriptive statistics, Shapiro-Wilk test, Chi-square test, Fisher’s exact test, and two-sample t-test. A p-value of <0.05 was considered statistically significant.

RESULTS

Over the 10-year period (January 2007 to December 2017), one hundred and eighty-seven (187) healthy adult vitiligo patients presented to the Department of Chemical Pathology and Metabolic Medicine of the University of Port Harcourt Teaching Hospital (UPTH) for dysglycemia screening. Data of 160 of the total 187 vitiligo patients met the inclusion criteria. Among the 160 data that met the inclusion, were 55 (34.4%) males and 105 (65.6%) females, which was statistically significant ($X^2 = 15.625; p = 0.001$).

Age and fasting plasma glucose level concentrations were all normally distributed. The mean age of study cohort was 35±1.9 years (range 19-61 years). However, the mean age of the males was 35±1.7 years (range 19-61), while that of the females was 34±1.4 (range 19-61).

In Table 1, most vitiligo patients were in the age group 20 -39 years (n = 102; 66.7%) with female preponderance (female 69.4% versus male 30.4%).

Table 1: Age and sex distribution of the vitiligo patients.

<table>
<thead>
<tr>
<th>Age Groups (years)</th>
<th>Male n(%)</th>
<th>Female n(%)</th>
<th>Total n(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 19</td>
<td>2 (50)</td>
<td>2 (50)</td>
<td>4 (2.5)</td>
</tr>
<tr>
<td>20-39</td>
<td>31 (30.4)</td>
<td>71 (69.4)</td>
<td>102 (63.8)</td>
</tr>
<tr>
<td>40-59</td>
<td>22 (44)</td>
<td>28 (56)</td>
<td>50 (31.2)</td>
</tr>
<tr>
<td>&gt;60</td>
<td>0 (0)</td>
<td>4 (100)</td>
<td>4 (2.5)</td>
</tr>
</tbody>
</table>

Fisher’s Exact test: 163.941; p <0.001

In Table 2, there was no difference in the mean age and the mean fasting plasma glucose concentrations between the males and females. However, males had higher mean levels of fasting plasma glucose concentrations than their female counterparts.

Table 2: Mean distribution of age and fasting plasma glucose concentrations in males and females.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Overall sex</th>
<th>Males</th>
<th>Females</th>
<th>t</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>35±1.9</td>
<td>35±1.7</td>
<td>34±1.4</td>
<td>0.354</td>
<td>0.724</td>
</tr>
<tr>
<td>FPG (mmol/l)</td>
<td>5.3±1.1</td>
<td>5.4±1.5</td>
<td>5.4±1.0</td>
<td>1.587</td>
<td>0.114</td>
</tr>
</tbody>
</table>

FPG = Fasting Plasma Glucose; mmol/l = millimol per liter; t = Two-sample t test

Table 2: Description of fasting plasma glucose levels among study cohorts.

<table>
<thead>
<tr>
<th>FPG (mmol/l)</th>
<th>Male n%</th>
<th>Female n%</th>
<th>Total n%</th>
<th>x²</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.5-6.0 (DM unlikely)</td>
<td>36 (31.9%)</td>
<td>77 (68.1%)</td>
<td>113 (70.6%)</td>
<td>1.538a</td>
<td>0.431</td>
</tr>
<tr>
<td>6.1-6.9 (Impaired FPG)</td>
<td>16 (39.0%)</td>
<td>25 (61.0%)</td>
<td>41 (25.6%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥7.0 (DM likely)</td>
<td>3 (50%)</td>
<td>3 (50%)</td>
<td>6 (3.8%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

FPG = Fasting plasma glucose; DM = diabetes mellitus; X²= Chi-square test; *Fisher’s Exact test
In Table 3, impaired fasting plasma glucose was detected in 41 (26.6%) subjects with female dominance (female 61.0% versus male 39.0%). Diabetes mellitus was documented in 6 (3.8%) subjects with no sex difference (female 50% versus male 50%).

**DISCUSSION**

The etiopathogenesis of vitiligo is poorly understood. However, several theories including genetics, neurochemical, melanocytic auto-toxicity, and autoimmunity have all been proposed as possible cuprite in the evolution and progression of the disorder. The theory of autoimmunity is the more accepted as vitiligo is frequently observed in coexistence with other autoimmune diseases including Addison’s disease, thyroiditis, pernicious anemia, including dysglycemia (insulin-dependent diabetes mellitus, impaired fasting glucose, and impaired glucose tolerance) in the same patient.5,6

Impaired fasting plasma glucose (IFG) has been observed in patients with vitiligo than those without vitiligo. In a recent Iranian case-control study, the authors had reported a 25.71% (18 out of 70) prevalence rate of IFG among 70 vitiligo patients, higher than the rate observed among the control subjects without vitiligo. In this present study, the prevalence of IFG was 26.6% (41 out of 160) with a female preponderance (female 61% versus male 39%), which is in accord with the recent Iranian study.

However, the prevalence of IFG among the general adult population in our region is 1.1% as reported recently by Eijke et al, which is significantly lower than the 26.6% rate documented in this study. This indicates an increased frequency of IFG among Nigerian adult vitiligo patients than the general Nigeria population. IFG precedes overt diabetes mellitus, and patients with IFG have a 1.5 fold increased risk for developing diabetes mellitus within ten years. Some authors had surmised that without lifestyle modifications and adequate monitoring, IFG will progress to clinically diagnosed diabetes mellitus. The high frequency of IFG observed in this study, warrants screening of all vitiligo patients for dysglycemia as part of their management protocol.

Vitiligo is one of the many skin diseases that exist in diabetes mellitus as diabetes mellitus has been linked to the likelihood of evolution of vitiligo.

It is proposed that many of the biochemical derangements in diabetes mellitus evokes dysfunctions of the melanocytic cells with resultant generation of anti-melanocyte antibodies leading to the destruction of melanocytes. However, none of the vitiligo patients in this study was diabetic, but we observed a 3.8% (6 out of 160) prevalence rate of diabetes mellitus among these patients with equal sex distribution (female 50% versus male 50%). The 3.8% rate observed in this study is lower than the rate observed among Indian (16%) and Saudi Arabian (8%) vitiligo patients. The observed variations of these two studies and ours could be due to differences in study methodology and genetic backgrounds.

Since diabetes mellitus especially the insulin-dependent type is generally found in 1-7% of vitiligo patients, our rate is in accord with the literature. However, the 3.8% prevalence of diabetes mellitus observed in this study is higher than the 3.0% prevalence reported recently among the general population of adult Nigerians by Ejike et al, which indicates a higher prevalence of diabetes mellitus among Nigerian vitiligo patients.18

The mechanism of dysglycemia in vitiligo has been adduced to the influence of autoimmunity and genetics. Vitiligo patients exhibit concomitant antibodies to melanocytes and to several organ-specific tissues including pancreas resulting in dysglycemia. While genetic studies have documented a shared etiologic background of vitiligo and other autoimmune diseases such as Addison’s disease, thyroiditis, pernicious anemia, including dysglycemia.

The results and findings of this study are not representative of the general population since it is a retrospective study, carried out in a single hospital setting in South-South Nigeria. These patients were not followed up to monitor their dysglycemic status in the long run.

**CONCLUSION**

Based on the results of this retrospective study and other similar studies, it is imperative to screen all patients with vitiligo for other autoimmune diseases including dysglycemia.

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**Ethical approval: Not required**

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