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IRON HOMEOSTASIS IN THE SUBJECTS WITH IMPAIRED GLUCOSE TOLERANCE

Laboratory Medicine

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ABSTRACT

Diabetes is common. Globally, diabetes has increased. Changes in iron metabolism markers, such as transferrin and ferritin, may induce or exacerbate type 2 diabetes. Several iron metabolism indicators independently predict reduced glucose tolerance, type 2 diabetes, and chronic glycemic characteristics. Ferritin and transferrin levels have increased with IGT and T2DM prevalence. Rising ferritin levels are likely due to increased iron reserves, but the association between transferrin and diabetes seems unrelated to iron storage, inflammation, or lipid metabolism. Thus, iron storage and metabolism may cause IGM and T2DM. However, disregard reverse causality. Hyperferritinemia is vital in Type 2 diabetes and other insulin-resistant diseases because too much iron causes poor outcomes, insulin resistance, and disease progression. Serum ferritin with type 2 diabetes graph. High ferritin levels may affect islet cell function, insulin production, and other risk factors for type 2 diabetes. Insulin influences serum ferritin transcription and cell iron absorption. Insulin production diminishes, and peripheral tissues get less iron when there is too much iron. Iron excess causes extreme oxidative stress, disrupting several biological processes. Serum transferrin and type 2 diabetes are little studied. The glomeruli cannot filter the large, negatively charged serum transferrin molecule. Thus, urine transferrin levels may indicate diabetic nephropathy development. Iron levels should be examined in Type 2 diabetics since too much might aggravate the illness.

KEYWORDS

Diabetes, iron homeostasis, ferritin, transferrin, oxidative stress

INTRODUCTION

Diabetes is a prevalent metabolic disease. Recently, diabetes has become more common around the world [1]. The rate of type 2 diabetes (T2DM) has risen dramatically in Indian adults over 30 years old. Diabetes has become one of the most critical public health issues in the last few years. Iron, one of the trace minerals, is essential for the growth and differentiation of living cells. It also plays a role in electron transfer between cells, which affects DNA synthesis.

In addition, iron may interact with oxygen, transport it to other bodily areas, and play a role in several vital metabolic processes [2]. As a powerful oxidant, iron may accelerate the development of many reactive oxygen radicals that affect the signal transduction process of islet -cells, altering the quantity of Insulin produced and how glucose is used. Iron also plays an essential function in mitochondria. This can help make adenosine triphosphate and change how much Insulin is released, which can cause glucose metabolism problems.

For all mammalian organisms to live, they need much iron. You can change its redox state, which is essential for oxygen transport, enzymes that make DNA and the citric acid cycle, and enzymes that make oxygen [3]. However, this also makes too much iron terrible because it causes the body to make reactive oxygen species (ROS), which can cause much damage to the body's organs. Iron reserves are strictly managed by a network of molecules that includes the liver, the stomach, the erythropoietic bone marrow, and the reticuloendothelial system to supply the body's iron demands and prevent iron toxicity [4]. People who are overweight, have insulin resistance, or have the nonalcoholic fatty liver disease often have iron alterations. Numerous studies have connected iron shortage (particularly in morbidly obese individuals) and iron excess (dysmetabolic iron overload syndrome) to obesity-related disorders.

Changes in iron metabolism markers, such as transferrin and ferritin, may induce or exacerbate type 2 diabetes [5]. However, the findings on the association between iron metabolism and type 2 diabetes did not hold for all sexes and ethnicities. All racial/ethnic groupings had significantly higher ferritin concentrations among women with diabetes than those without diabetes. However, men with diabetes had lower ferritin concentrations than men without diabetes. As iron is a potent promoter for creating free radicals, an increase in oxidative stress is believed to be responsible for iron-induced IR. However, there is no actual evidence to support this notion. People with Type 2 diabetes and obesity have increased oxidative stress, which leads to Insulin-resistant fat cells. This reduces their ability to use Insulin [6].

MATERIALS AND METHODS

A comparative cross-sectional study was conducted on patients with a prior diagnosis of type 2 diabetes mellitus who were undergoing treatment at the diabetic clinic located at the Sevana clinic in Kozhikode, Kerala. The investigation covers the period from December 2020 to May 2022. Patients were deemed eligible for this study if they were between the ages of 35 and 65, willing to participate, and had no life-threatening diseases (such as liver disease, renal disease, endocrine disease, or cancer). Each test subject was matched with an individual of the same age and gender. As a foundation for selecting control subjects, the first studies sought individuals between the ages of 35 and 65 with no clinical symptoms of severe problems.

The demographic information, baseline values of the lipid profile, and biochemical markers were all collected and recorded on the Datasheet. Each participant will have 5 milliliters of fasting venous blood drawn from them. The blood sample is divided into two portions: one for the analysis of whole blood and the other for the analysis of serum. A patient who had been fasting was given a blood sample under aseptic circumstances to separate the serum. The material was put in a tube containing a clot activator. The serum is then divided in half, with one component utilized for baseline analysis and another stored at -20 degrees Celsius for specific research. On the same day, investigations of the baseline are conducted.

Blood glucose was estimated by the hexokinase method. HbA1c estimated by ion-exchange high-performance liquid chromatography

RESULTS AND DISCUSSION

To evaluate iron homeostasis, serum concentrations of transferrin, ferritin, and TIBC are measured. The statistical significance of the data was determined using SPSS version 20. There was statistical significance at the 0.05 level or below. The data is averaged, and standard deviations are shown and tested statistically. Body mass index (BMI) rose significantly in the experimental group compared to the control group. The test group had substantially greater fasting blood sugar, postprandial blood sugar, and hemoglobin A1c levels than the control group. The levels of ferritin and TIBC are much higher in the test group, while the transferrin levels are much lower in the control group. Researchers have shown a correlation between higher ferritin levels and type 2 diabetes and early anomalies in glucose metabolism, the subject of most previous studies on iron status and T2DM. Consistent with these findings, and after controlling for several potential confounders, our results on ferritin show that the estimated strength of the association is very similar between subjects with T2DM and is more potent in newly diagnosed diabetic cases than in known diabetic cases, suggesting that ferritin, or instead increased body iron, may play a role in the early pathogenesis of T2DM. Similarly, our study found increased ferritin levels linked to rising fasting hyperglycemia. There needs to be more focus on transferrin. A decrease in ferritin levels is correlated with an increase in transferrin production, suggesting a negative relationship between the two. However, we found a strong link between high transferrin levels and

type 2 diabetes.BMI of the test population increased in the test population compared to the control and is found to be highly significant.

Glycaemic parameters, namely FBS, PPBS, and HbA1c, showed significant elevation in the test population compared to the control.Ferritin and TIBC are significantly elevated, while Transferrin levels significantly decreased in the test population compared to the control population. Iron is one of the trace elements that the body needs. The body has 3–5 g of iron in it. The body controls how much iron it takes in primarily by absorbing it. It does not work right when the body does not get enough or too much iron. The more iron you have, the more likely you are to get type 2 diabetes because it damages pancreatic -cells and makes Insulin less effective. Most of the body's iron is stored in a protein called ferritin, which is crucial for maintaining iron homeostasis. An elevated ferritin level may be a sensitive sign of an excessive iron burden in the body caused by the damage of non-stored iron organs. Ferritin levels in the blood are higher in people with type 2 diabetes. Diabetic risk increases in both Western and Asian populations with elevated ferritin levels. A higher serum ferritin level was associated with an increased likelihood of developing type 2 diabetes in the whole sample. This could be because the average blood ferritin levels of the patients in these three studies were extremely high.

Since too much iron is associated with poor outcomes, insulin resistance, and accelerated disease progression, hyperferritinemia is crucial in people with Type 2 diabetes and other insulin-resistant illnesses. One possible graph depicting the connection between serum ferritin and type 2 diabetes High ferritin levels in the blood may, for example, contribute to the development of type 2 diabetes by influencing the activity of islet cells, the body's ability to produce Insulin, and other risk factors for the disease. In addition, Insulin facilitates iron uptake by cells and regulates serum ferritin transcription. When too much iron is already in the body, insulin synthesis decreases, and peripheral tissues get less of the iron they need. Extreme oxidative stress is produced by an iron overload, which disrupts the regular operation of many body systems. The association between serum transferrin and type 2 diabetes has been the subject of little research. The serum transferrin molecule is too big and has a negative charge to be adequately filtered by the glomeruli. So, checking the levels of urine transferrin might be a great technique to gauge the rate of progression of diabetic nephropathy. Patients with Type 2 diabetes should have their iron levels checked because too much iron can worsen the disease. Ferritin, serum iron, and transferrin saturation should all be checked.

A functional relationship between iron and glucose metabolism has been found primarily in people with metabolic diseases, such as diabetes. For example, when glucose is made in the liver, it makes hepcidin, which stops cells from getting rid of iron. This leads to cell iron overload and low iron levels in the blood. Cellular iron overload, on the other hand, regulates insulin production in pancreatic -cells and is thought to impact glucose metabolism in diabetic patients significantly. This interaction is likely to happen in other diseases, like cardiovascular diseases.

Declaration of interest

The authors state that they have no conflict of interest that may be seen as impairing the research's objectivity.

CONCLUSIONS

In conclusion, we have shown that several indices of iron metabolism are independently associated with impaired glucose tolerance (IGT), type 2 diabetes, and chronic glycemic features. The increasing prevalence of IGT and T2DM has been frequently associated with elevated ferritin and transferrin levels. While rising ferritin levels are presumably the result of increased iron stores, there seems to be a mechanism unrelated to iron storage, inflammation, or lipid metabolism mediating the link between transferrin and diabetes. Therefore, the etiology of IGM and T2DM may be linked to iron stores in the body and the accompanying metabolic processes. On the other hand, the possibility of reverse causality must be ignored.

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