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Correlation between the Diabetic Marker (Hba1c) and the Anemia Marker (Hba2) In Type 2 Diabetes

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Background

The hemoglobin HbA1C level is widely used to monitor diabetes mellitus patients. The aim of this study was to determine the prevalence of the effect of iron deficiency anemia on the glycated hemoglobin level in diabetic patients.

Keywords

Correlation. Type 2 diabetes - Glycated Hemoglobin HbA1c - Anemia marker HBA2 - Sex and age.Background

Introduction

Iron deficiency (ID) and iron deficiency anemia (IDA) are prevalent sorts of nutritional deficiency. Globally, 50% of anemia is attributed to iron deficiency. Reduced iron stores are linked to increased glycation of hemoglobin A1C (HbA1c). Additionally, the prevalence of IDA is considerably significant in patients with type 2 DM especially those with nephropathy. The clinical relevance of the effect of iron deficiency on glucose metabolism remains not clear. The links between glucose, anemia and HbA1c are complex and not yet fully elucidated. Diabetes can contribute to anemia through reducing absorption of iron, gastrointestinal bleeding and thru diabetic complications that cause anemia.

Studying the effect of ID and IDA on glucose metabolism in experimental animals and in human subjects revealed some important consequences of both on glucose levels, HbA1c and insulin secretion. Additionally, a number of the possible mechanisms that mediate these effects are investigated.

The present review focuses on the present knowledge on the various effects of IDA on glucose metabolism in normal and diabetic patients.

Some studies examined the hepatic expression of genes involved in maintenance of glucose homeostasis during ID. These studies have shown that dietary intervention(s) tend to elicit biologically meaningful, transcriptional responses. The ID rats in each group showed significant alterations within the expression of genes representative of glucose metabolism.

Distinguished changes in organic phenomenon include those genes related to metabolic pathways including both glycolysis and gluconeogenesis.

The significant increase within the glucokinase (Gck) expression is probably going thanks to the relative increase in circulating insulin levels observed within the ID groups, as insulin may be a known inducer of hepatic Gck mRNA expression. Increased expression of Gck could potentially be vital as ID animals are shown to possess an increased reliance on glucose as a metabolic substrate, and Gck is in a position to rapidly increase the speed of glucose phosphorylation within the liver in response to the elevations in blood sugar levels. Furthermore, as Gck catalyzes the primary step in hepatic glucose utilization it can contribute multiple pathways including glycogen synthesis, glycolysis, and de novo lipogenesis which could explain the improved glucose utilization and hyperlipidemia reported in response to dietary ID.

Previous observations suggest that alterations in metabolic organic phenomenon are indicative of an impaired hepatic insulin response wherein ID animals exhibited a sort of mixed insulin resistance. Chronic hyperinsulinemia may contribute to a mixture of hepatic insulin resistance during which the insulin-dependent activation of lipogenic organic phenomenon remains intact, but gluconeogenic organic phenomenon is inadequately repressed. during this model of mixed insulin resistance, insulin acts through the mammalian target of rapamycin complex 1 to activate lipogenesis via a sterol regulatory element (SRE) -binding protein)-1c-dependent increase in lipogenic organic phenomenon , whereas insulin-induced phosphorylation of the transcription factor forkhead box protein O1 is diminished such gluconeogenic organic phenomenon remains inappropriately active. Thus, mixed insulin resistance remains a candidate mechanism explaining the relative hyperglycemia and hyperlipidemia reported in ID animals.

Diabetes is the single most common cause of end-stage renal disease and therefore the most common cause of renal anemia. In addition, anemia may be more common in diabetes and develop earlier than in patients with renal impairment from other causes. The predominance of damage to renal interstitium, systemic inflammation, and autonomic neuropathy has all been suggested as contributors to anemia in diabetic nephropathy (DN). Like many pathophysiological changes of DN, dysfunction may be apparent before demonstrable changes in the glomerular filtration rate (GFR).

It is unproven whether anemia directly contributes to the acceleration of complications in DN or to the progression of diabetic renal disease. However, patients with diabetes may be more vulnerable to the effects of anemia because many also have significant cardiovascular disease and hypoxia-induced organ damage. In addition, a number of studies have suggested that Hb levels may be linked to the risk of cardiovascular events, hospitalization, and mortality. Against this, there is no conclusive evidence that correcting anemia significantly improves outcomes in patients with failing renal function, apart from quality of life.

Possible Causes of Anemia

Usually, it happens because you don't have enough red blood cells. That can make you more likely to get certain diabetes complications, like eye and nerve damage. And it can worsen kidney, heart, and artery disease, which are more common in people with diabetes.

Diabetes often leads to kidney damage, and failing kidneys can cause anemia. Healthy kidneys know when your body needs new red blood cells. They release a hormone called erythropoietin (EPO), which signals your bone marrow to make more. Damaged kidneys don't send out enough EPO to keep up with your needs.

Often, people don't realize they have kidney disease until it's very far along. But if you test positive for anemia, it can be an early sign of a problem with your kidneys.

People with diabetes are more likely to have inflamed blood vessels. This

can keep bone marrow from getting the signal they need to make more red blood cells.

Methods

Study was conducted on 200 patients. Each patient provided a blood sample for hemoglobin. We rated the serum HbA1C/HBA2 levels using the capillary electrophoresis technique in liquid phase.

Results

Our results showed that in 200 diabetic and non-diabetic patients. (60%) was normal while (40%) was T2D, in the same group of T2D (41.25%) presented iron deficiency anemia or HBA2 rate less than (2.2%). With a female predominance of (69.69%) compared to men (30.31%), the age group between (40-50) years revealed (45.45%) of the cases followed by the (<50years) with (36.36%), and the least affect was (30-40years) by (18.18%),

Conclusion

We found a moderately positive correlation between HbA1c and HbA2 levels in patients with diabetes mellitus. The iron deficiency anemia raises HbA1c levels in diabetics. They need to integrate the treatment of iron deficiency anemia before improving the overall care of diabetic patients.