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Type 2 Diabetes Genetic Epidemiology in Mexican Mestizos

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Abstract

It has been postulated that altered bone quality brought on by the underlying metabolic abnormalities of type 2 diabetes (T2D) causes altered bone strength and turnover, increasing the risk of fracture in T2D patients. Studies concentrating on White men and women have largely formed the basis for current knowledge about changes in bone turnover indicators in T2D patients. Nevertheless, T2D and osteoporosis are more common in Hispanic people in the US. We looked at the relationships between bone turnover rate and glycemic control in 69 older (>50 years) Mexican American Cameron County Hispanic Cohort (CCHC) participants with T2D. The relationships between HbA1c (%), serum osteocalcin (OC), and serum sclerostin were evaluated using multivariable models. Our study discovered that Mexican American men with T2D who had worse glycemic control experienced decreased bone turnover (indicated by lower serum OC), which is consistent with published data from other racial/ethnic communities. Glycemic control and OC did not significantly correlate for the women in our study. In contrast, HbA1c was found to be positively linked with sclerostin in women; however the association was not statistically significant. We advise checking for bone loss and fracture risk in Mexican Americans with T2D, especially in those with poor glycemic control.

Keywords: Type 2 diabetes mellitus; Epidemiology; Human disorders

Introduction

A complex illness with high rates of morbidity, mortality, and recurrence all across the world is venous thromboembolism (VTE). Deep vein thrombosis, pulmonary embolism, or both clinically describe it. Age, trauma, hormonal imbalance, immobility, hypercoagulable state, thrombophilia defects, and hypertension are some of the risk factors that can contribute to the development of VTE. Also, as genetic factors are a significant part of the aetiology, research is being done to find predictive biomarkers that may aid in diagnosing the condition. Short tandem repeats (STRs), also known as microsatellites, are the primary cause of genetic variation in humans. Human coding genes contain 17% of STRs, which are widely distributed in regulatory areas that impact gene transcription and expression. As a result, STRs have been associated with disease phenotypes, particularly in neurodegenerative and neuromuscular illnesses. STRs have recently been linked to complex disorders like cancer, diabetes, and cardiovascular diseases (CVD), indicating that they have a greater phenotypic impact than many single nucleotide polymorphisms (SNPs) [1].

Five STRs are found inside the genes TH01 (human tyrosine hydroxylase), TPOX (human thyroid peroxidase), vWA (von Willebrand factor), CSF1PO (c-fms proto-oncogene for CSF-1 receptor gene), and FGA within the combined DNA index system (CODIS) (human alpha fibrinogen). Several of these have been linked to thrombotic events, including vWA, FGA, and TPOX. A glycoprotein called von Willebrand factor (vWF) is created by platelets and vascular endothelium. Moreover, vWF serves as a biomarker of CVD in clinical settings and is crucial for haemostasis. It also promotes thrombosis by encouraging platelet adhesion and aggregation. Genetic factors control the synthesis and secretion of vWA, and as of this writing, more than 19 SNPs in intron regions have been linked to vWF antigen levels. Intron 40 also contains a complex STR marker that has been linked to coagulation-related illnesses. Genetic diversity has been linked to fibrinogen levels and fibrin network topology, and fibrinogen is thought to be a risk factor for CVD. As a result, polymorphisms in FGA have been linked to both an elevated risk of VTE and a stroke [2].

Finally, multiple studies indicate that thyroid peroxidase (TPOX) activity is connected with coagulation abnormalities and vascular endothelial dysfunctions. Moreover, thyroid levels have been linked to thromboembolic potential, indicating that TPOX may be a factor in VTE. Using polymorphic markers, the quest for biomarkers linked to thrombosis risk aims to contribute to and identify the biological bases of this complicated disease. Also, the introduction of preventative medicines to delay the beginning of illness could be made possible by the use of genetic predictive diagnostic tests. Nonetheless, single nucleotide polymorphisms (SNPs) have received a lot of attention in genotype-phenotype association research, undervaluing markers of hyper variable polymorphisms. In order to uncover potential susceptibility markers, the main goal of the current study is to investigate whether there is a connection between VTE and the combined impact of many genetic variants in the vWA, FGA, and TPOX microsatellites. Three controls were used for each case in a case-control study that we conducted. Our results imply that allele's 9-TPOX, 12-TPOX, and 18-vWA may be associated with thrombosis risk in our community. This link was still present following population stratification correction, indicating that it is not a synthetic effect. Our data support the idea that using STR analysis to create markers can help identify the genetic causes of many human disorders [3].

Materials and Method

The participants were both males and women who were a part of the population-based elder (50 years) cohort nestled in the CCHC project. The CCHC study's setting, data collection method, and follow-up strategy have all been previously described in detail. Simply stated, the Cameron County Hispanic Cohort (CCHC) is a two-stage randomly selected cohort of Mexican Americans along the border between the

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United States and Mexico. Between the population that the CCHC was selected from and the majority of the Caucasian population, there are significant health disparities. Almost 4000 men and women who are 18 years or older and have been observed for more than 10 years make up the CCHC, 1400 of whom are in the elder cohort [4].

Researchers gather data on this population's demographics, past fall and fracture histories, medication usage, co-morbidities, and behavioural risk factors like smoking, physical activity, and alcohol use through an annual survey. The procedure also calls for the collection of serum samples, which are analysed for signs of various diseases, such as metabolic risk factors, as well as data on glucose metabolism, such as blood levels of HbA1c. The older cohort has been created to examine the epidemiology of ageing in Mexican Americans, a community with significant metabolic risk factors, such as T2D and obesity. In order to comprehend the epidemiology of skeletal health risk factors associated with age-related bone loss and fracture risk in this group, a bone health protocol was created in 2013. We provide data from 69 men and women who have been given a T2D diagnosis (according to American Diabetic Association diagnosis criteria 2010) [5].

The University of Texas Health Science Center at Houston's committee for protection of human subjects (CPHS) gave its approval to both the main CCHC trial and the bone protocol. The following criteria were used to weed out participants: active cancer, type 1 diabetes, known metabolic bone disease, known osteoporosis, history of osteoporosis treatment, and use of drugs that may influence bone health, such as thiazolidinedione. By self-reported history of no menstrual cycle in the previous six months, all of the women included in this study were postmenopausal [6].

Discussion

Our examination of the genetics of T2D in Mexican mestizo subjects reveals that 26 variants spread across 21 genes are linked to this disease, demonstrating that our population also exhibits significant levels of T2D heterogeneity. Alleles of specific genes are therefore involved in some people, whilst gene variations are involved in other people. Based on an examination of three genetic markers, a recent finding that T2D in Mexican mestizos is genetically homogeneous now seems unsupportable. Although if the percentage of European ancestry in the Mexican mestizo community is close to 30%, not all of the alleles that increase the risk of diabetes in Europeans are linked to T2D in our group. These disparities might be attributed to genetic make-up, variances in clinical classifications, sample size, selection and analysis standards, and environmental elements like obesity, lifestyle, and cuisine. On the other hand, studies in a number of ethnic groups have revealed a link between T2D and genes that have not yet been examined in the Mexican community. It would be crucial to analyse these genes to see if their variations are likewise linked to T2D in patients from Mexico and to learn more about the genetic epidemiology of this disease in our nation [7].

For both age groups and both sexes in our investigation, the high serum sclerostin (an biomarker of deficient bone formation) did not support the negative relationships between glycemic control and serum OC. Yet, in older Mexican American men and women, we compared bone turnover with inadequate or adequate blood glucose control. According to the lab where serum samples were evaluated, the mean sclerostin levels for men and women in our study were greater than the 50th percentile of normal ranges (for males 32-67 pmol/l and for women 22-52 pmol/l). Clinical evaluation of high risk for fracture is frequently relied on a pattern of bone turnover that is above or below the 50th percentile in the lack of unambiguous normative data in clinical practise for older persons without metabolic or bone health concerns. Considering the normal ranges, the higher than normal values discovered in our study suggest that bone formation may be low for both men and women, confirming a similar pattern of bone formation in older adults with T2D reported for people from other racial/ethnic backgrounds, despite the lack of nondiabetic controls limiting this study [8].

The addition of serum OC levels to current testing as an indicator may aid to improve screening of T2D patients for their risk of fracture because BMD frequently is deceiving for assessing fracture risk in people with T2D. We advise establishing a baseline level for markers of bone turnover at the time of screening with DXA BMD and continuous monitoring over time in association with HbA1c to track bone metabolism in older T2D Mexican Americans because an age-appropriate normal range for those subjects (>65 years) is not available.

The connection between glycemic management and bone turnover in an older Mexican American cohort has never been examined before. This work offers essential scientific knowledge concerning changes in bone metabolism in T2D patients with Hispanic backgrounds, even though our findings might not be generalizable to persons from different racial/ethnic backgrounds. Our study is limited by the small sample size, absence of nondiabetic controls, and cross-sectional data analysis [9]. None of the study subjects, however, were taking any drugs known to have an impact on bone metabolism, such as thiazolidinedione and bisphosphonates. Further research on this cohort will assist characterise the effect of T2D on fracture risk of the ageing skeleton in older Mexican American men and women by adding more men and women with and without T2D as well as more precise resorption and formation markers. Our study's substantial results in males, who had a smaller sample size than women, also raise questions about whether men's bone metabolism is altered more severely than that of women, which could mean that men with T2D are more likely to experience fragility fractures.

Having poor glycemic control increases the risk of developing numerous cardio-metabolic health issues in older Mexican American men and women. Our study adds the possibility of aberrant bone metabolism to this list of issues, and as a result, we urge screening for bone loss and fracture risk among Mexican Americans with T2D, especially in those with poor glycemic control [10].

Conclusions

This cross-sectional study's findings, which include the fact that insulin therapy may obscure the link between depression and glycemic control, demonstrate that patients with T2DM in Kuwait have a high prevalence of depression, which is linked to worse diabetes outcomes.

Conflict of Interest

None

Acknowledgement

None

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