

Diabetes Mellitus Factors Contributing to the Occurrence, Diagnosis and Treatment of the Disease

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Abstract: 2 diabetes is one of the foremost common endocrine clutters. There are 415 million individuals on the planet with diabetes (1 in 11 grown-ups); It is assessed that by 2040 there will be 642 million debilitated individuals. Each 6 seconds, one individual kicks the bucket from the results of diabetes (5 million passings). The Worldwide Diabetes Alliance (IDF) has pointed out components that play a part within the advancement of the infection: hereditary qualities, way of life, environment and slim down.

Keywords: diabetes mellitus type 2; Vitamin D; antibodies; plasminogen activator inhibitor 1; inflammation mediators.

The most instrument of improvement of type 2 diabetes is affront resistance and diminished affront emission. Diabetes mellitus could be a condition of persistent hyperglycemia, characterized by disabled digestion system of carbohydrates, proteins and fats. Corpulence happens due to outright or relative affront lack, resistance, expanded glucose generation and overabundance activity of hormones with the inverse impact of affront. Science has not however clearly distinguished the extra commitment.

Factors influencing the occurrence and treatment of this disease. What underlies the formation of diabetes mellitus? clear well. Therefore, any contribution that leads in this direction is valuable.

The role of vitamin D in the development of diabetes According to many authors, vitamin D plays an important role. Role in blood glucose control, but also in the relief chronic diabetic complications. Rationale for this based on the following facts: the presence of vitamin . Pancreatic beta cell D receptor (VDR), vitamin D by activating 1-alpha-hydroxylase present in these cells, the presence insulin VDR gene, which leads to an increase synthesis of insulin under the influence of vitamin D. In addition, VDRs are present in skeletal muscle cells. H that 1,25 (OH) 2D rounds up the transcription of the insulin gene. receptor that stimulates this expression and inhibits renin gene, thereby reducing hyperglycemia-induced increases renin levels and blocks renin-angiotensin activity.

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The beneficial effects of vitamin D when it comes to diabetes may be due to its anti-inflammatory effects and influence on calcium and phosphate metabolism, as well as gene regulation of the insulin receptor. Vitamin D increases the amount of calcium in cells, which leads to increased glucose levels transport in muscles. Vitamin D also regulates nuclear PPAR (peroxisome proliferator-activated receptor), which plays an important role in insulin sensitivity. Vitamin D deficiency is associated with increased inflammation. Vitamin D reduces the expression of pro-inflammatory proteins cytokines involved in the development of insulin resistance, such as interleukin (IL)-1, IL-6 and tumor necrosis factor (TNF)-alpha. Vitamin D has this effect on this cytokine. anti-apoptotic effect on pancreatic beta cells, maintaining insulin secretion from it and increasing insulin sensitivity.



Compared to the healthy population in type 2 diabetes significantly lower circulating concentrations.25(OH)D is present 4. Common risk factors for type 2 diabetes and hypovitaminosis D are obesity, age, association with black race and reduced physical activity. Probable mechanism of vitamin D participation in glucose synthesis homeostasis is the effect on beta cell and insulin dysfunction resistance due to vitamin D deficiency. Negative correlation between blood glucose levels and insulin levels to 25(OH)D level and positive correlations . 25(OH)D levels and insulin sensitivity have been tested in several animal and human studies. Some of these studies had noticed that supplementing with vitamin D may improve insulin secretion and reduce insulin resistance in type 2 diabetes.

Possible role of vitamin D in type 2 diabetes and its impact HbA1C values were also recorded. It should be noted that there are studies that do not support the above message. It seems that there is still no consensus on this matter. Vitamin D, according to some studies, plays a role in alleviating chronic diabetic complications.

Effect data vitamin D deficiency in the fight against diabetes and the incidence of complications is moderate. Ahmedi et al. 10 inches their study examined the relationship between levels 25(OH)D and microvascular complications in patients with type 2 diabetes. It was concluded that low serum levels 25(OH)D level was an independent predictor of HbA1c, diabetes neuropathy and retinopathy. In a similar study Zoppini et al. 11 found an inverse relationship between 25(OH)D levels and prevalence of microvascular diseases complications in patients with type 2 diabetes mellitus concluded that vitamin D deficiency is more common in diabetic patients with nephropathy, but not in those for retinopathy and neuropathy. It was also found that Vitamin D levels were lower in patients with severe microvascular disorders. D deficiency has been associated with microvascular abnormalities. O complications in patients with type 2 diabetes mellitus.

Diabetic nephropathy (DN) progressively reduces vitamin levels Level of D in the body due to: loss of vitamin D binding protein. With proteinuria, impaired synthesis of vitamin D in the skin and decreased activation of vitamin D in the damaged kidney. It was noted that vitamin D deficiency is higher in patients with chronic diabetes. P renal failure (CKD) than in patients without diabetes. Eat indicates that VDRs are modulators of glomerular damage. Calcitriol, an endogenous activator of VDR, reduces glomerulosclerosis. Clinically, patients with chronic renal failure treated with paricalcitol (a selective VDR activator) experienced significant reduction in proteinuria after 23 weeks of treatment, regardless of GFR, blood pressure, or angiotensin-converting enzyme (ACE) inhibition.

The benefits of therapy with VDR activators relate specifically to anti-inflammatory effect. Animal models of primary glomerulopathy . VDR activators reduce glomerular infiltration of inflammatory cells. In addition, high serum vitamin D levels in patients with chronic renal failure are associated with reduced systemic inflammation 12. Cardio- ankle -vascular index (CAVI), a parameter in the diagnosis of occlusive disease of blood vessels. This is a new parameter for determining the stiffness of the arterial wall, starting from the aorta and ending with the arteries of the joints of the lower extremities 22. The definition of this indicator is based on the dominance of beta receptors, effect of beta receptors 1 vascular stiffness receptor.

There is a change in the caliber of the blood arteries and, as a consequence, a change in the internal pressure of the blood vessel 23. This index does not depend on the height of the pressure at this point. Beta blood hardness parameters were determined. as the ratio of pulse wave speed and diastolic blood pressure. CAVI is a constant value. The administration of alpha blockers, which reduce contracture of the smooth muscles of the arterial wall, reduces the CAVI value or blood pressure to appreciate. This means that CAVI is an indicator of arteries. an indicator of the compliance or vascular function of transporting blood from the heart to the peripheral arteries.



Thiazide diuretics, ACE inhibitors, and beta-1 blockers do not reduce CAVI when they lower blood pressure.

The beta parameter of arterial narrowing is determined along the length of the main arteries. It is calculated by measuring the pulse wave velocity in the brachial artery and the arteries of the leg at the level of the ankle joint. It measures systolic and diastolic blood pressure at the brachial artery. artery. The measured values of the application are given in Eq. It then gets a new setting called Cardio-Ankle -. Vascular Index Regarding the role of PAI-1 in wound healing of inflamed tissue in type 2 ischemic diabetes, there was a significant increases early in the reaction process neutrophilic inflammation. This directly increases swelling and tissue necrosis. Therefore, at an early stage PAI-1 inhibits the action of IL-6, which reduces the concentration proteins in the inflamed area with a delay wound treatment.

Literature :

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