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Review Article

Recent Developments in the Treatment and Prevention of Type Two Diabetes and Its Risk Factors

Smita Patel*

Department of clinical psychology, Department of Pharmacology and Toxicology, Canada

*Corresponding Author's E-mail: SmitaPtel65@gmail.com

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Abstract

A dangerous and prevalent chronic disease, type 2 diabetes is caused by a complicated interaction between genes, environment, and additional risk factors like obesity and sedentary behaviour. With high rates of diabetes-related morbidity and death, type 2 diabetes and its consequences are a significant global public health issue that affect practically all populations in both developed and developing nations. Type 2 diabetes is becoming more and more common, and both developing nations and populations that are being "westernised" or modernised have high prevalence rates. It is necessary to develop new effective therapy strategies and suitable prevention measures for the control of type 2 diabetes due to the multiplicity of risk factors for the disease, the delayed diagnosis until micro- and macrovascular complications appear, life-threatening complications, failure of the current therapies, and the financial costs associated with the treatment of this illness. The epidemiology of type 2 diabetes, the roles of genes, lifestyle, and other variables leading to the sharp rise in type 2 diabetes incidence are all summarised here. The main objectives are to present novel therapeutic approaches and financially sensible type 2 diabetes intervention trials.

Keywords: Genetic factor, intervention trial, Lifestyle, Treatment, Type 2 diabetes

INTRODUCTION

A dysfunction in the body's ability to control and utilise sugar as fuel results in type 2 diabetes. This sugar is also known as glucose. There is too much sugar flowing in the blood as a result of this chronic illness. Over time, cardiovascular, neurological, and immune system issues might result from excessive blood sugar levels (Adhikari M., et al 2011). Two issues predominate in type 2 diabetes. Insulin, a hormone that controls how quickly sugar enters cells, is not produced by the pancreas in sufficient amounts. Additionally, cells absorb less sugar and have a weak insulin response. Although type 1 and type 2 diabetes can start in childhood and adulthood, respectively, type 2 diabetes used to be classified as adult-onset diabetes (Arentz M., et al 2012). Older persons are more likely to have type 2. But more young individuals are developing type 2 diabetes as a result of the rise in the number of obese children.

Type 2 diabetes does not have a treatment (Babu S., et al 2009). Exercise, a healthy diet, and weight loss can all help with disease management. Diabetes medicines or insulin therapy may be advised if diet and exercise are insufficient to regulate blood sugar (Banfield S., et al 2012). Type 2 diabetes mellitus is caused by a combination of genetic, environmental, and metabolic risk factors (Bates M., et al 2012). The most at-risk people have a history of diabetes mellitus in their families, are older, obese, and inactive (Bhargava A., et al 2013). Minority communities are also more susceptible, not only due of genetics and family history but also because of poor dietary and exercise habits that have developed as a result of adaptation to the American environment (Bhaskaram P., et al 2002). Children of women who previously had gestational diabetes are more likely to develop type 2 diabetes mellitus (Black GF., et al 2002). A person is more likely to acquire type 2 diabetes and poor glucose tolerance if they have insulin resistance.

Many of the same risk factors that are present in type 2 diabetics are also present in people with insulin resistance. These include polycystic ovarian syndrome, hypertension, prothrombin condition, hyperuricemia, glucose intolerance, hyperinsulinemia, and thermogenic Dyslipidemia. Currently, type 2 diabetes mellitus prevention and retardation strategies focus on altering environmental risk factors, such as lowering obesity rates and encouraging physical exercise (Chintu C., et al 1993). With the aim of reducing microvascular and macrovascular consequences, screening, early identification, and treatment in high-risk populations will be encouraged by knowledge of the risk factors for type 2 diabetes (Co DO., et al 2006). This review article examines how type 2 diabetes is currently thought of by scientists as a complicated condition that affects many other organs in addition to the pancreas and the insulin system, including the liver, kidneys, stomach, muscle, fat cells, and even the brain. Understanding the intricate pathophysiology of type 2 diabetes helps primary care physicians treat patients with this prevalent and dangerous condition more effectively.

DISCUSSION

Your pancreas produces the hormone insulin, which functions as a key to allow blood sugar to enter your body's cells for use as fuel. Insulin resistance, which occurs when cells don't react to insulin as they should in people with type 2 diabetes. To try and elicit a response from cells, your pancreas produces more insulin. Your blood sugar eventually rises as a result of your pancreas' inability to keep up, which can lead to type 2 diabetes and prediabetes. High blood sugar harms the body and increases the risk of renal disease, heart disease, and other major health issues. Both type 2 diabetes in children and childhood obesity are on the rise. Over 75% of kids with type 2 diabetes also have a close family who is affected by the disease. But it's not usually because they are connected; it could possibly be because they have certain tendencies in common that make them more vulnerable. By creating a strategy for the entire family, parents can aid in the prevention or delay of type 2 diabetes:

- consuming more water and less sweetened beverages
- eating more vegetables and fruit
- improving the health of favourite foods
- enhancing the fun of physical exercise

CONCLUSION

T2DM and its associated problems place a significant burden on health around the globe, and there are currently no viable treatments available. The interplay of genetic and environmental risk is the primary cause of the epidemic of diabetes. The disorders can also be attributed to a variety of other causes. While the majority of antidiabetic medications have demonstrated positive outcomes when used singly or in combination, they are also linked to side effects such weight gain, hypoglycemia, gastrointestinal problems, or cardiovascular disease. Finding the proper treatment is now one of the main goals in the fight against T2DM, which is on the rise. Numerous treatment plans, including the use of SGLT2 inhibitors, DPP-4 inhibitors, and GPR40 agonists, have been established to far. Above all, stem cell educator therapy provided a safe and very effective means of creating novel therapeutic approaches for the treatment of T2DM. Additional research should concentrate on the following topics the precise mechanism causing T2DM and its complications; successful intervention trials and prevention measures to prevent the occurrence of this disease; earlier diagnosis for earlier treatment; and novel drugs with more beneficial effects and fewer adverse effects, in order to treat this disease, enhance quality of life, and extend lifespan. According to research, T2DM causes visible immune system changes, particularly in adipose tissue, pancreatic islets, the liver, the vasculature, and circulating leukocytes 151. These changes include altered levels of some cytokines and chemokines, changes in the number and activation state of various leukocyte populations, as well as an increase in apoptosis and tissue fibrosis. These modifications show that inflammation is a key player in the development of T2DM and associated consequences. In the treatment of T2DM patients, salicylates and interleukin-1 antagonists serve as illustrative medications with immunomodulatory effects that can lower blood glucose levels and diminish the severity and prevalence of related problems 152, 153. Clinical investigations in phase III have recently been ongoing. Another innovative and potentially successful method for treating T2DM patients is antioxidant therapy, which may significantly reduce the chance of acquiring diabetes and associated complications. For the treatment of oxidative stress in T2DM patients, a variety of antioxidants, including vitamins, supplements, active ingredients derived from plants, and medications having antioxidant activities, have been employed. The best supplements against oxidative stress and its side effects are vitamin C, vitamin E, and betacarotene 76. For instance, vitamin C can increase insulin action and lower fasting plasma insulin and HbA1c levels, and beta-carotene may lower oxidised LDL 156. T2DM 76 can be treated with the help of plants that contain compounds with antioxidant qualities such monoterpenes, cinnamic acids, coumarins, flavonoids, triterpenes, phenylpropanoids, triterpenes, tannins, and lignin. Drugs s with antioxidant qualities, such as carvedilol and -lipoic acid, also have antioxidant benefits in T2DM. According to statistics, roughly 50% of diabetics go misdiagnosed, and 20–30% of patients typically have difficulties before receiving a diagnosis 157. Thus, the urgent need for an alternative screening method for the earlier diagnosis of T2DM. Currently, a number of risk assessment tools have been created for the prediction of T2DM based on self-reported, biochemical measurements, or genetic markers. These tools are more useful and practical than traditional blood glucose screening tests because they allow interventions to be used on people with impaired glucose tolerance to delay the onset of T2DM. C-reactive protein, liver enzymes, and other new biochemical indicators are a few examples. In the European Prospective Investigation into Cancer and Nutrition-Potsdam Study, liver enzymes with blood lipid concentrations can clearly improve prediction beyond the noninvasive parameters and measures of glycemia 170 but C-reactive protein has not demonstrated additional prognostic information beyond the extended prediction model. White blood cell count can also help predict outcomes, according to a risk score from Taiwan, despite the low precision of the calculated score.

REFERENCES

- Adhikari M, Jeena P, Bobat R, Archary M, Naidoo K, et al (2011). HIV-associated tuberculosis in the newborn and young infant. Int J Pediatr. 354208.
- Arentz M, Pavlinac P, Kimerling ME, Horne DJ, Falzon D, et al (2012). Use of anti-retroviral therapy in tuberculosis patients on second-line anti-TB regimens: A systematic review. PloS ONE. 8: e47370.
- Babu S, Bhat SQ, Kumar NP, Anuradha R, Kumaran P, et al (2009). Attenuation of toll-like receptor expression and function in latent tuberculosis by coexistent filarial infection with restoration following antifilarial chemotherapy. PLoS Negl Trop Dis. 3: e489.
- Banfield S, Pascoe E, Thambiran A, Siafarikas A, Burgner D, et al (2012). Factors associated with the performance of a bloodbased interferon-γ release assay in diagnosing tuberculosis. PloS ONE. 7: e38556.

- Bates M, O'Grady J, Mwaba P, Chilukutu L, Mzyece J, et al (2012). Evaluation of the burden of unsuspected pulmonary tuberculosis and co-morbidity with non-communicable diseases in sputum producing adult inpatients. PloS ONE. 7: e40774.
- Bhargava A, Chatterjee M, Jain Y, Chatterjee B, Kataria A, et al (2013). Nutritional status of adult patients with pulmonary tuberculosis in rural central India and its association with mortality. PloS ONE. 8: e77979.
- 7. Bhaskaram P (2002). Micronutrient malnutrition, infection, and immunity: An overview. Nutr Rev. 60: S40-S45.
- Black GF, Weir RE, Floyd S, Bliss L, Warndorff DK, et al (2002). BCG-induced increase in interferon-γ response to mycobacterial antigens and efficacy of BCG vaccination in Malawi and the UK: Two randomised controlled studies. Lancet. 359: 1393-1401.
- Chintu C, Bhat G, Luo C, Raviglione M, Diwan V, et al (1993). Seroprevalence of human immunodeficiency virus type 1 infection in Zambian children with tuberculosis. Pediatr Infect Dis J. 12: 499-504.
- Co DO, Hogan LH, Karman J, Heninger E, Vang S, et al (2006). Interactions between T cells responding to concurrent mycobacterial and influenza infections. J Immunol. 177: 8456-8465.