World Journal of Pharmaceutical and Life Sciences
WJPLS
www.wjpls.org
SJIF Impact Factor: 7.409

DIABETES MELLITUS IN YOUNGER INDIVIDUALS

Dr. G. Nagaraju[1*], G. Sirisha[1], M. Meghana[2], T. Divya[3], M. Veneela[4], G. Swathi[5] and K. Madhupriya[6]

[1]Principal of Dhanvanthari Institute of Pharmaceutical Sciences, Sujthanagar, Kothagudem.
[1]M. Pharm Associate Professor of Dhanvanthari Institute of Pharmaceutical Sciences, Sujthanagar, Kothagudem.
[2,3,4,5,6]Pharmacy IV Year Students of Dhanvanthari Institute of Pharmaceutical Sciences, Sujthanagar, Kothagudem.

***Corresponding Author: Dr. G. Nagaraju**
Principal of Dhanvanthari Institute of Pharmaceutical Sciences, Sujathanagar, Kothagudem.

Article Received on 07/02/2025 **Article Revised on 27/02/2025** **Article Accepted on 17/03/2025**

ABSTRACT
Diabetes mellitus is a heterogeneous group of disorders characterized by hyperglycemia due to an absolute or relative deficit in insulin production or action. The chronic hyperglycemia of diabetes mellitus is associated with end organ damage, dysfunction, and failure, including the retina, kidney, nervous system, heart, and blood vessels The chronic metabolic disorder diabetes mellitus is a fast-growing global problem with huge social, health, and economic consequences. It is estimated that in 2010 there were globally 285 million people (approximately 6.4% of the adult population) suffering from this disease. The International Diabetes Federation (IDF) estimated an overall prevalence of diabetes mellitus to be 366 million in 2011, and predicted a rise to 552 million by 2030The origin and etiology of DM can vary greatly but always include defects in either insulin secretion or response or in both at some point in the course of disease.

KEYWORDS: Hyperglycemia, Chronic, Economical consequences, Etiology, Dysfunction.

INTRODUCTION

Diabetes mellitus, disorder of macro molecule metabolism characterized by impaired ability of the body to supply or answer endocrine and thereby maintain correct levels of sugar (glucose) within the blood.[1] malady may be a chronic disease that happens once the duct gland is not any longer able to build endocrine, or once the body cannot observe use of the endocrine it produces. Endocrine may be a endocrine created by the duct gland that acts sort of key to let aldohexose from the food we have a tendency to eat pass from the blood stream into the cells within the body to supply energy. All macromolecule foods square measure countermined into aldohexose within the blood. Endocrine helps aldohexose get into the cells. This chapter introduces the types of diabetes and diabetic complications such as impairment of immune system, periodontal disease, retinopathy, nephropathy, somatic and autonomic neuropathy, cardiovascular diseases and diabetic foot. Also included are the current management and treatments, and emerging therapies.

SIGNS AND SYMPTOMS

- Increased thirst (polydipsia)and dry mouth
- Frequent urination
- Fatigue
- Blurred vision
- Unexplained weight loss
- Numbness or tingling in hands and feet
- Slow heating source or cuts
- Extreme hunger
- Frequent skin or vaginal yeast infections

CAUSES

Too much glucose circulating in your bloodstream causes diabetes, regardless of the type. However, the reason why your blood glucose levels are high differs depending on the type of diabetes.

Insulin resistance: Insulin resistance happens when cells in your muscles, fat and liver don't respond as they should to insulin. Several factors and conditions contribute to varying degrees of insulin resistance, including obesity, lack of physical activity, diet, hormonal imbalances, genetics and certain medications

Autoimmune disease: Type 1 diabetes and LADA happen when your immune system attacks the insulin-producing cells in your pancreas.

Hormonal imbalances: During pregnancy, the placenta releases hormones that cause insulin resistance. You may develop gestational diabetes if your pancreas can't produce enough insulin to overcome the insulin

resistance. Other hormone-related conditions like acromegaly and Cushing syndrome can also cause Type 2 diabetes.

Pancreatic damage: Physical damage to your pancreas — from a condition, surgery or injury — can impact its ability to make insulin.

RISK FACTORS

T2DM risk factors include a complex combination of genetic, metabolic and environmental factors that interact with one another contributing to its prevalence. Although individual predisposition to T2DM due to non-modifiable risk factors (ethnicity and family history/genetic predisposition) has a strong genetic basis, evidence from epidemiological studies suggests that many cases of T2DM can be prevented by improving the main modifiable risk factors (obesity, low physical activity and an unhealthy diet).[2][3]

Ethnicity and Family History/Genetic Predisposition

Globally, the incidence and prevalence of T2DM are found to vary widely depending on ethnicity and geographical region with Japanese, Hispanics and Native Americans having the highest risks.[4][5][6] It has been shown higher incidence rates in Asians compared with a White American population[7],[8], and white population in the UK,[9], where the highest risk is among the black population.[10] Whilst no clear reasons have been found, contributing factors such as modern lifestyle factors (which promote obesity), socioeconomic and direct genetic propensity or gene environmental interactions have been postulated.

Obesity, Low Physical Activity and Unhealthy Diet

Obesity (body-mass index [BMI]≥30 kg/m2) is the strongest risk factor for T2DM[11],[12] and is associated with metabolic abnormalities resulting in IR.[13] There exists an inverse linear relationship between BMI and the age at diagnosis of T2DM.[14] The exact mechanisms by which obesity induces T2DM and IR remain to be elucidated; however, numerous factors have shown a significant role in the development of this pathological process, which involves both cell-autonomous mechanisms and inter-organ communications.

A sedentary lifestyle is another risk factor for T2DM as shown by the Women's Health Study and in the Kuipio Ischemic Heart Disease Risk Factor Study, which showed a reduction of 34% and 56% reduction of developing T2DM in participants walking 2–3 h a week or at least 40 min a week, respectively.[15][16]

PATHOPHYSIOLOGY

Regarding the pathophysiology of the disease, a malfunctioning of the feedback loops between insulin action and insulin secretion results in abnormally high glucose levels in blood.[17] In the case of β-cell dysfunction, insulin secretion is reduced, limiting the body's capacity to maintain physiological glucose levels. On the other hand, IR contributes to increased glucose production in the liver and decreased glucose uptake both in the muscle, liver and adipose tissue. Even if both processes take place early in the pathogenesis and contribute to the development of the disease, β-cell dysfunction is usually more severe than IR. However, when both β-cell dysfunction and IR are present, hyperglycemia is amplified leading to the progression of T2DM.[18][19]

DIAGNOSIS

Fasting blood glucose test: For this test, you don't eat or drink anything except water (fast) for at least eight hours before the test. As food can greatly affect blood sugar, this test allows your provider to see your baseline blood sugar.

Random blood glucose test: "Random" means that you can get this test at any time, regardless of if you've fasted.

A1c: This test, also called HbA1C or glycated hemoglobin test, provides your average blood glucose level over the past two to three months.

To screen for and diagnose gestational diabetes, providers order an oral glucose tolerance test.

Type of test

Fasting blood glucose test
In-range (mg/dL) Less than 100
Prediabetes(mg/dL) 100 to 125
Diabetes (mg/dL) 126 or higher.
Random blood glucose test
In-range (mg/dL) N/A
Prediabetes(mg/dL) N/A
Diabetes (mg/dL) 200or higher
$A1_C$ test
In-range (mg/dL) Less than 5.7%
Prediabetes(mg/dL) 5.7 to 6.4%
Diabetes (mg/dL) 6.5 % or higher

The following test results typically indicate if you don't have diabetes, have prediabetes or have diabetes. These values may vary slightly. In addition, healthcare providers rely on more than one test to diagnose diabetes.

TREATMENT

Insulin and oral hypoglycemic drugs

Insulin therapy should aim to mimic nature, which is remarkably successful both in limiting postprandial hyperglycemia and preventing hypoglycemia between meals[20] of administration of insulin injection is equally important for better and safe action of insulin and can be given by intramuscular or intravenous route. Different preparations of insulin are available such as human insulin, beef insulin, pork insulin.

Insulin therapy is no free from complications and adverse effects. The most important adverse effect are weight gain and hypoglycemia when inappropriate dose of insulin is taken and when there is mismatch between meals and insulin injection.[21, 22]

Weight gain after starting insulin therapy for uncontrolled diabetes is an inevitable consequence and is the result of increased truncal fat and muscle bulk. This is also due to reduced energy losses through glycosuria.[23,24]

Sulphonyl ureas such as glibenclamide

glipizide and biguanides such as metformin, phenformin are oral hypoglycemic drugs. Sulfonylureas cause hypoglycemia by stimulating insulin release from pancreatic ß-cells. They bind to sulfonylurea (SUR) receptors on the ß-cell plasma membrane, causing closure of adenosine triphosphate (ATP)-sensitive potassium channels, leading to depolarization of the cell membrane. This in turn opens voltage gated channels, allowing influx of calcium ions and subsequent secretion of preformed insulin granules.

Acute administration of sulfonylureas to type 2 DM patient's increases insulin release from the pancreas and also may further increase insulin levels by reducing hepatic clearance of the hormone. Initial studies showed that a functional pancreas was necessary for the hypoglycemic actions of sulfonylureas[25]

Biguanides

Such as metformin is antihyperglycemic, not hypoglycemic.[26] It does not cause insulin release from the pancreas and does not cause hypoglycemia, even in large doses.[27] It has been shown to increase peripheral uptake of glucose, and to reduce hepatic glucose output by approximately 20-30% when given orally but not intravenously.

PREVENTION

Type 1 and type 1.5 diabetes are not preventable because they are caused by an issue with the immune system. Some causes of type 2 diabetes, such as your genes or age, aren't under your control either. Yet many other diabetes risk factors are manageable. Most diabetes prevention strategies involve making simple adjustments to your diet and fitness routine. If you've received a diagnosis of prediabetes, here are a few things you can do to delay or prevent type 2 diabetes:

- Get at least 150 minutes per week of aerobic exercises like walking or cycling. Cut saturated and trans fats, along with refined carbohydrates, out of your diet.
- Eat more fruits, vegetables, and whole grains.
- Eat smaller portions.
- Try to lose 5% to 7%Trusted Source of your body weight if you have overweight or obesity.

CONCULSION

Diabetes is very critical and serious complication in today's life. The lifestyle and day today circumstances are playing major role in occurring this type of serious complications. In this review we get some idea regarding diabetes mellitus.

REFERENCE

1. Diabetes mellitus MEDICAL DISORDER WRITTEN BY: The Editors of Encyclopedia Britannica See Article History.
2. Hu F.B., Manson J.E., Stampfer M.J., Colditz G., Liu S., Solomon C.G., Willett W.C. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. N. Engl. J. Med., 2001; 345: 790–797. doi: 10.1056/NEJMoa010492. [DOI] [PubMed] [Google Scholar]
3. Schellenberg E.S., Dryden D.M., Vandermeer B., Ha C., Korownyk C. Lifestyle interventions for patients with and at risk for type 2 diabetes: A systematic review and meta-analysis. Ann. Intern. Med., 2013; 159: 543–551. doi: 10.7326/0003-4819-159-8-201310150-00007. [DOI] [PubMed] [Google Scholar]
4. Chan J.C., Cheung C.K., Swaminathan R., Nicholls M.G., Cockram C.S. Obesity, albuminuria and hypertension among Hong Kong Chinese with non-insulin-dependent diabetes mellitus (NIDDM) Postgrad. Med. J., 1993; 69: 204–210. doi: 10.1136/pgmj.69.809.204. [DOI] [PMC free article] [PubMed] [Google Scholar]
5. Dabelea D., DeGroat J., Sorrel man C., Glass M., Percy C.A., Avery C., Hu D., D'Agostino R.B., Jr., Beyer J., Imperatore G., et al. Search for Diabetes in Navajo youth: Prevalence, incidence, and clinical characteristics: The Search for Diabetes in Youth Study. Diabetes Care, 2009; 32(Suppl. 2): S141–S147. doi: 10.2337/dc09-S206. [DOI] [PMC free article] [PubMed] [Google Scholar]
6. Liu L.L., Yi J.P., Beyer J., Mayer-Davis E.J., Dolan L.M., Dabelea D.M., Lawrence J.M., Rodriguez B.L., Marco vina S.M., Waitz Felder B.E., et al. Type 1 and Type 2 diabetes in Asian and Pacific Islander U.S. youth: The SEARCH for Diabetes in Youth Study. Diabetes Care, 2009; 32(Suppl. 2): S133–S140. doi: 10.2337/dc09-S205. [DOI] [PMC free article] [PubMed] [Google Schola
7. Karter A.J., Schillinger D., Adams A.S., Moffet H.H., Liu J., Adler N.E., Kanaya A.M. Elevated rates of diabetes in Pacific Islanders and Asian subgroups: The Diabetes Study of Northern California (DISTANCE) Diabetes Care, 2013; 36: 574–579. doi: 10.2337/dc12-0722. [DOI] [PMC free article] [PubMed] [Google Scholar)
8. Sattar N., Gill J.M. Type 2 diabetes in migrant south Asians: Mechanisms, mitigation, and management. Lancet Diabetes Endocrinol., 2015; 3: 1004–1016. doi: 10.1016/S2213-8587(15)00326-5. [DOI] [PubMed] [Google Scholar

9. McKeigue P.M., Shah B., Marmot M.G. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. Lancet., 1991; 337: 382–386. doi: 10.1016/0140-6736(91)91164-P. [DOI] [PubMed] [Google Scholar)
10. Haines L., Wan K.C., Lynn R., Barrett T.G., Shield J.P. Rising incidence of type 2 diabetes in children in the U.K. Diabetes Care, 2007; 30: 1097–1101. doi: 10.2337/dc06-1813. [DOI] [PubMed] [Google Scholar]
11. Fuchsberger C., Flannick J., Teslovich T.M., Mahajan A., Agarwala V., Gaulton K.J., Ma C., Fontanillas P., Moutsianas L., McCarthy D.J., et al. The genetic architecture of type 2 diabetes. Nature, 2016; 536: 41–47. doi: 10.1038/nature18642. [DOI] [PMC free article] [PubMed] [Google Scholar]
12. McCarthy M.I. Genomics, type 2 diabetes, and obesity. N. Engl. J. Med. 2010; 363: 2339–2350. Doi: 10.1056/NEJMra0906948. [DOI] [PubMed] [Google Scholar]
13. Dimas A.S., Lagou V., Barker A., Knowles J.W., Magi R., Hivert M.F., Benozzo A., Rybin D., Jackson A.U., Stringham H.M., et al. Impact of type 2 diabetes susceptibility variants on quantitative glycemic traits reveals mechanistic heterogeneity. Diabetes, 2014; 63: 2158–2171. Doi: 10.2337/db13-0949. [DOI] [PMC free article] [PubMed] [Google Scholar]
14. Flannick J., Florez J.C. Type 2 diabetes: Genetic data sharing to advance complex disease research. Nat. Rev. Genet., 2016; 17: 535–549. Doi: 10.1038/nrg.2016.56. [DOI] [PubMed] [Google Scholar]
15. Franks P.W., Pearson E., Florez J.C. Gene-environment and gene-treatment interactions in type 2 diabetes: Progress, pitfalls, and prospects. Diabetes Care, 2013; 36: 1413–1421. Doi: 10.2337/dc12-2211. [DOI] [PMC free article] [PubMed] [Google Scholar]
16. Bellou V., Belbasis L., Tzoulaki I., Evangelou E. Risk factors for type 2 diabetes mellitus: An exposure-wide umbrella review of meta-analyses. Plops ONE, 2018; 13: e0194127. Doi: 10.1371/journal.pone.0194127. [DOI] [PMC free article] [PubMed] [Google Scholar]
17. Carey V.J., Walters E.E., Colditz G.A., Solomon C.G., Willett W.C., Rosner B.A., Speizer F.E., Manson J.E. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women. The Nurses' Health Study. Am. J. Epidemiol., 1997; 145: 614–619. Doi: 10.1093/oxfordjournals.aje.a009158. [DOI] [PubMed] [Google Scholar]
18. Sinha R., Dufour S., Petersen K.F., Lebon V., Enoksson S., Ma Y.Z., Savoye M., Rothman D.L., Shulman G.I., Caprio S. Assessment of skeletal muscle triglyceride content by (1)H nuclear magnetic resonance spectroscopy in lean and obese adolescents: Relationships to insulin sensitivity, total body fat, and central adiposity. Diabetes, 2002; 51: 1022–1027. Doi: 10.2337/diabetes.51.4.1022. [DOI] [PubMed] [Google Scholar]
19. Hillier T.A., Pedulla K.L. Complications in young adults with early-onset type 2 diabetes: Losing the relative protection of youth. Diabetes Care, 2003; 26: 2999–3005. Doi: 10.2337/diacare.26.11.2999. [DOI] [PubMed] [Google Scholar]
20. Weinstein A.R., Sesso H.D., Lee I.M., Cook N.R., Manson J.E., Buring J.E., Gaziano J.M. Relationship of physical activity vs body mass index with type 2 diabetes in women. JAMA. 2004; 292: 1188–1194. Doi: 10.1001/jama.292.10.1188. [DOI] [PubMed] [Google Scholar]
21. Lynch J., Helmrich S.P., Lakka T.A., Kaplan G.A., Cohen R.D., Salonen R., Salonen J.T. Moderately intense physical activities and high levels of cardiorespiratory fitness reduce the risk of non-insulin-dependent diabetes mellitus in middle-aged men.
22. Arch. Intern. Med., 1996; 156: 1307–1314. Doi: 10.1001/archinte.1996.00440110073010. [DOI] [PubMed] [Google Scholar]
23. Stumvoll M., Goldstein B.J., van Haaften T.W. Type 2 diabetes: Principles of pathogenesis and therapy. Lancet, 2005; 365: 1333–1346. Doi: 10.1016/S0140-6736(05)61032-X. [DOI] [PubMed] [Google Scholar]
24. Cerf M.E. Beta cell dysfunction and insulin resistance. Front. Endocrinol. (Lausanne) 2013; 4: 37. Doi: 10.3389/fendo.2013.00037. [DOI] [PMC free article] [PubMed] [Google Scholar]
25. Zheng Y., Ley S.H., Hu F.B. Global a etiology and epidemiology of type 2 diabetes mellitus and its complications. Nat. Rev. Endocrinol., 2018; 14: 88–98. Doi: 10.1038/nrendo.2017.151. [DOI] [PubMed] [Google Scholar]