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Review

From insulin deficiency to resistance: unraveling the complexities of diabetes mellitus

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Abstract

Diabetes mellitus is a chronic metabolic disease that can be driven on by deficits in insulin secretion, action, or both. There are three types of diabetes mellitus: type 1 diabetes is brought on by the body producing insufficient amounts of insulin. Earlier names for this condition included "juvenile diabetes" and "insulin dependent diabetes mellitus" (IDDM).). It's unclear why this is happening. The main sign of type 2 diabetes is insulin resistance. It's unclear why this is happening. Type 2 diabetes begins with insulin resistance, a condition in which cells do not react to insulin as they should. The intrusive nature of insulin delivery combined with long-term use has led to issues with patient compliance, which has eventually affected patient outcomes.

Keywords: Diabetes Mellitus, Cause And Treatment, Type1 & Type2, Gestational Diabetes Mellitus.

INTRODUCTION

The most prevalent endocrine condition, diabetes mellitus (dm), affects over 100 million people globally, or 6% of the population. It is brought on by insufficient or inefficient insulin production by the pancreas, which causes blood glucose levels to rise or fall. Numerous bodily systems, including the blood vessels, eyes, kidney, heart, and nerves, are shown to be harmed by it.[1]. Insulin-dependent diabetes mellitus (IDDM, type1) and non-insulin-dependent diabetes mellitus (NIDDM, type2) are the two categories of diabetes mellitus. In contrast to type ii diabetes, which is defined by peripheral insulin resistance and decreased insulin production, type i diabetes is an autoimmune illness that is characterized by a local inflammatory response in and around islets, followed by the selective death of cells that secrete insulin.[2]. Diabetes mellitus increases the risk of numerous consequences, including peripheral vascular disease, stroke, neuropathy, renal failure, retinopathy, blindness, amputations, and cardiovascular illnesses.[3]. Endocrine is produced by the duct gland; however, it doesn't function as it should. We call this condition endocrine resistance. Understanding more about how the body uses food for energy a

process known as metabolism helps us better comprehend polygenic disease. Our body is creating million of the cell cells. The cells desire nourishment throughout a very basic type in order to produce energy. When you eat lot of food or drink when your food is converting an easy sugar known as aldohexose. Aldohexose provides the energy your body desires for daily activities [4]. An excessive amount of sugar stays in your blood if you have very little or no endocrine function or are endocrine resistant. Those with polygenic diseases have higher glucose levels than normal [5]. In 2025, expected to increase to 69.9 million [3].

Secrete the insulin hormone glucagon and pancreas. The islets of Langerhans contain beta (ß) cells that release insulin and alpha (α) cells that secrete glucagon. Insulin lowers blood glucose levels by glycogenesis and transfers glucose into the muscles, they the the liver. It and adipose tissue. Alpha (α) cells are essential for controlling blood sugar because they produce glucagon, which boosts blood glucose levels, even though erythrocytes and brain tissue lack glucose to consume glucose.by speeding up glycogenolysis. [6,7]. Along with a higher chance of obesity, cardiovascular and metabolic diseases, and cancer in the fetus's postpartum life,[8].

Eighty to ninety percent of all cases of diabetes mellitus are type ii. Geographical variation can influence total morbidity and mortality as well as the severity of the issues. [9,10]. Additionally, those with diabetes who engage in modest physical exercise have a significantly decreased risk of dying than those who do not.[11]. The word diabetes mellitus refers to a genetically diverse set of conditions, even if glucose intolerance is a sign of many other diseases. Currently, there are two idiopathic kinds of diabetes: type 1 and type 2 diabetes, also known as IDDM and NIDDM, on the one hand, and a collection of over 60 hereditary disorders on the other, where diabetes is just one symptom.[12].

Hyperglycemia is a typical symptom of a range of metabolic disorders known as diabetes mellitus. [13,14] chronic hyperglycemia harms the heart, blood vessels, kidneys, nerves, and eyes. [15]. Present time oral hypoglycemic medications have lots of side effects. As a result, alternative therapy is necessary, and it is urgent to switch to various indigenous plant and herbal compositions. [16].

Likewise, it is anticipated that t2d-related mortality will quadruple globally between 2005 and 2030.[17]. T2dm affects the 25.8 million people in the us, or 8.3% of the total population, including children and teenagers.[18]. T2d is more frequently accompanied by metabolic conditions such as dyslipidemia, hypertension, or vascular endothelial dysfunction, which may result in micro- and macrovascular consequences.[19]. While diet and exercise are important components of t2d treatment, most people need pharmaceutical intervention, which may involve glucagon-like peptide-1 agonists, sulfonylureas, metformin, or thiazolidinediones.[20].

However, weight gain, hypoglycemia, and/or subsequent failure unwanted side effects of these anti-diabetic medications may have contributed to the rise of complementary and alternative medicine.[21]. Ethnic minorities' cultural perspectives on diabetes mellitus frequently diverge, which could compromise treatment compliance. [22,23].

According to the international diabetes federation (IDF), there are around 40.9 million diabetics in INDIA overall, and by 2025, that figure is expected to increase to 69.9 million [24]. More frequently, t2d is accompanied by metabolic conditions such as dyslipidemia, hypertension, or vascular endothelial dysfunction, which may result in micro- and macrovascular problems. [25].

Classification of diabetes mellitus

In 1980, the world health organization (who) published the first accepted diabetes mellitus classification [26]. Additionally, it was changed in 1985[27]. Our presentation will center on the most prevalent and significant type of primary or idiopathic diabetes mellitus. It must be distinct from secondary diabetes mellitus, which encompasses types of hyperglycemias linked to recognizable causes, wherein inflammatory pancreatic diseases, surgery, tumors, specific medications, iron overload (hemochromatosis), and genetic endocrine or specific acquired cause the destruction of pancreatic islets.[28]. The classification includes additional types of hyperglycemia as well as the clinical stages and etiological types of diabetes mellitus.[29].

Classification of Diabetes Mellitus

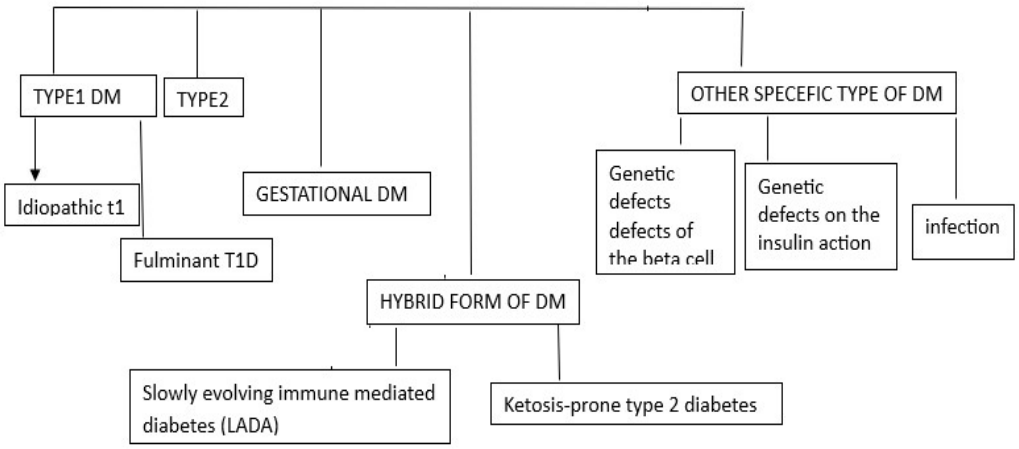

Fig: 1 Classification of diabetes mellitus

Insulin dependent diabetes mellitus (type1 IDDM)

Previously known as juvenile-onset or ketosis-prone diabetes, this form of diabetes mellitus is also referred to as autoimmune diabetes. The person may also seek treatment for further autoimmune conditions such Addison's disease, Hashimoto's thyroiditis, and graves' disease. [30]. Type one diabetes mellitus as also known as insulin-dependent diabetes mellitus (IDDM), mainly affects children and adolescents. It usually appears abruptly and can be fatal.[31]. Anti-glutamic acid decarboxylase, islet cell, or insulin antibodies are typically present in type 1 diabetes, indicating the autoimmune mechanisms that cause beta-cell death.[32]. The American diabetes association (2014) states that type 1 diabetes is caused by the death of b-cells, which typically results in complete insulin insufficiency. The pace of devastation of beta cells varies greatly; it may happen quickly for some people and slowly for others. [33].

Non-insulin dependent polygenic disorders mellitus (type2 NIDDM)

Type2 diabetes mellitus another name is ketosis resistant. In the context of insulin resistance the progressive hypoglycemic agent's secretariat lack in relation to the resistance to insulin (american a polygenic disorder association, 2014) [34]. Individuals with this type of polygenic condition frequently exhibit resistance to the effects of hypoglycemic medications.[35]. The primary sources of morbidity and mortality from polygenic disorders are the semi-permanent problems in the kidneys, blood vessels, eyes, and nerves, which can affect any kind.[36] obesity, inactivity, aging (affecting middle-aged and older adults), and genetic issues are among the main causes and predisposing factors (ross and Wilson 2010). Patients with these conditions are more likely to experience small tube and macrovascular difficulties [37,38]

Gestational diabetes mellitus (GDM)

Pregnant women with this diabetes are classified as having GDM. Class a2 is the most prevalent heritable form of monogenic disease, and women who have experienced it require insulin or other medications. The third major form, gestational diabetes, affects pregnant women who have never had diabetes mellitus before. Gdm is typically identified in the latter stages of pregnancy and frequently affects women who have never had diabetes mellitus before.[39] obesity and type 2 diabetes are more likely to occur among those with diabetes mellitus. Class a1 and class a2 are the two classes that GDM offers. It happens in roughly 4% of pregnancies [40]. In class a1, diet and exercise are in charge. Globally, gestational diabetes is expected to rise by about 18%.[41].

Other specific type of diabetes mellitus

Endocrinopathies

Many of hormones stimulate or inhibit the action of insulin. When the FBG is greater than 100 mg/dl but less than 126 mg/dl, it indicates impaired fasting blood glucose. [42]. It could include pancreatic cancer or tumors, polycystic ovarian syndrome, and other hormonal disruptions in the synthesis of insulin.

Mody

Insulin may or may not be necessary for them. Mody is an acronym for young people with maturity-onset diabetes.[43]. Mody is not insulin-dependent diabetes mellitus; rather, it is inherited diabetes mellitus caused

by a genetic mutation in an autosomal dominant gene that influences insulin synthesis or secretion. Individual diagnosis is typically made for genetically predisposed children under 25. The hepatocyte nuclear factor (hnf1-alpha) gene is responsible for roughly 70% of MODY cases.[44]. It links to a genetic flaw in the β-cells. This kind involves early-onset hyperglycemia. Clinically, they resemble type 2 diabetes.

Some Common Sign and Symptom

When cells in diabetes mellitus are unable to digest glucose normally, they essentially starve. [45]

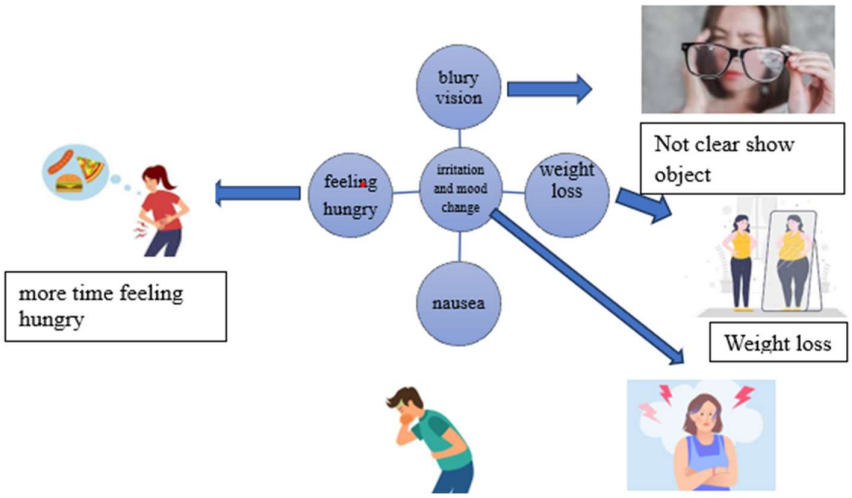

Fig 2: Sign and symptoms of Diabetes Mellitus

The long-term consequences of diabetes mellitus, which include the progressive emergence of specific complications such as retinopathy with possible visual impairment, renal disease that will lead to failure, and pathology with the potential for foot ulcers, neurologist joints, involuntary pathology, and sexual dysfunction [46]. People who have polygenic disorders are more likely to get sick. A many of symptoms were identified as a result. Gluconeogenesis from amino acids and bodily macromolecules raises blood sugar levels and causes tissue disintegration, muscular atrophy, and other problems. I cathartic energy, and destructive metabolism of body fat excessive creation of organic compound bodies [47].

Etiology of diabetes mellitus

The Greek term "aetiologia" is the root of the English word "etiology." therefore, the science of determining the causes and origins of a disease is known as etiology, and it comprises. The juvenile-onset (insulin-dependent) variant is thought to have an autoimmune origin at this time. Like coxsackie b, viruses may also contribute to the genesis of diabetes. there is debate over the genetic component of polygenic illness genesis. The exocrine gland of a person is likely more susceptible to one of the viruses due to a genetic characteristic.

Causes of diabetes mellitus

Disturbances or anomalies in the ß cell's glucose-receptor that cause them to react to elevated glucose levels or relative ß cell insufficiency. Insulin secretion is compromised in either case, which could lead to ß cell failure. [48]

Diagnosis of diabetes mellitus

A single abnormal blood sugar reading should never be used to diagnose polygenic disease in asymptomatic people. Given the extensive and protracted consequences for the individual, the practitioner should be certain that the diagnosis of polygenic disease is well validated [49]. A illness with several causes is referred to as polygenic disease. Cortisone-stressed aldohexose tolerance test, endogenous aldohexose tolerance test, oral aldohexose tolerance test, urinary organ threshold of aldohexose, decreased aldohexose tolerance, raised aldohexose tolerance, renal symptom, prolonged aldohexose tolerance curve.

Table 1: diagnosis of diabetes by OGTT

Patient status	Plasma glucose value	Diagnosis
1.fasting value	Before 110mg/dl (<6.1 nmol/l)	Normal fasting value
2.fasting value	110-126mg/dl (6.1-7.0mmol/l	Impaired fasting Glucose (ipg)**
3.fasting value	126mg/dl (7.0mmol/l	Diabetes mellitus
4. 2 hours after 75g oral glucose-load	140-200mg/dl (11.1mmol/l)	Impaired fasting Glucose (ipg)**
5. 2 hours after 75g oral glucose load	200mg/dl (11.1mmol/l)	Diabetic mellitus
6. Random value	200mg/dl (11.1mmol/l) or more	Diabetic mellitus

Tests for diagnosis of diabetes

urine testing: diabetes mellitus urine test is cheap and convenient but the diagnosis of diabetes cannot be based on urine testing alone since there may be false positive and false-negative. Urine is tested for the presence of glucose and ketones**.**

Glucosuria: benedict's qualitative test detects any reducing substance in the urine and is not specific for glucose. More sensitive and glucose specific test is dipstick method based on enzyme-coated paper strip which turn purple when dipped in urine containing glucose.

Ketonuria: test for ketone bodies in the urine are required for assessing the severity of diabetes and not for diagnosis of diabetes. However, if both glucosuria and ketonuria are present, diagnosis of diabetes is almost certain. Rothera's test and strip test are conveniently performed for detection of ketonuria.

single blood sugar estimation: for diagnosis of diabetes, blood sugar determinations are absolutely necessary. Folin-Wu method pf measurement of all reducing substances in the blood including glucose is now absolute. Currently used are o-toluidine, Somogyi-nelson and glucose oxidase method. Whole blood or plasma may be used but whole blood values are 15% lower than plasma values.

Oral glucose tolerance test: the patient who is scheduled for oral GTT is instructed to eat a high carbohydrate diet foe at least 3 days prior to the test and come after an overnight fast on the day of the test (for at least 8 hr.). A fasting blood sugar sample is first drawn then 75 gm of glucose dissolved in 300 ml of water is given. Blood and urine specimen are collected at half hourly intervals for at least 2 hr. Blood or plasma glucose content is measured and urine is tested for glucosuria to determine the approximate renal threshold for glucose. Venous whole blood concentration is 15% lower than plasma glucose value

Treatment of diabetes mellitus

High dosages of a typical hypoglycemic medication are used as part of the treatment to combat the underlying cause. Once the situation has been managed, the demand for hypoglycemic agents returns to its usual level [50]

1.improve our life style, take healthy diet, daily exercise, weight manage
2.regular blood sugar check
3. Type 1 diabetic mellitus insulin therapy

Types of therapy involved in diabetes mellitus

Stem cell therapy

Monocytes and macrophages have been implicated by researchers as major contributors to insulin resistance and chronic inflammation in individuals with type 2 diabetes [51]. A new method called stem cell educator treatment is intended to manage or even correct immunological dysfunctions. Blood from the patient is collected using a closed-loop system, lymphocytes are separated from the whole blood, adherent cord blood-

derived multi-potent stem cells (cb-scs) are co-cultured with them in trained lymphocytes in vitro, but not cb-scs, and then injected iv to the patient [52,53].

Antioxidant therapy

Numerous antioxidants, including vitamins, supplements, active ingredients derived from plants, and medications with antioxidant properties, have been utilized to treat oxidative stress in individuals with type 2 diabetes. The best vitamins to combat oxidative stress and its aftereffects are vitamin c, vitamin e, and beta-carotene. Antioxidants have a significant impact in reducing the risk of diabetes and its consequences [54].

Anti-inflammatory treatment

The alterations indicate that inflammation play a key part in the development of type 2 diabetes and its consequences. [55,56]. Changed levels of certain cytokines and chemokines, number and activation state of certain leukocyte populations, increased apoptosis, and tissue or fibrosis, particularly in adipose tissue, pancreatic the liver, circulating leukocytes in type 2 diabetes the vasculature. Immunomodulatory medications are offered [57,58].

Dietary management

Sufficient calorie content both diabetic and non-diabetic patients should follow dietary guidelines, such as: Protein, carbs, and fats should all be balanced, but in any situation, carbohydrate intake must be limited. Should resemble normal as much as possible.
consumption of food should be split up into meals of comparable sizes that are spread out regularly. Cut back on both fat and carbohydrates to lower your overall caloric consumption. .the patient needs to be counseled to maintain consistent daily eating habits.

Newer insulin delivery devices: numerous advancements have been made to attain strict glycaemia control and to make administering insulin easier and more accurate. These include insulin syringes, pen devices, insulin pumps, implanted pumps, inhaled insulin, and other insulin administration methods**.**

Oral hypoglycemic or antidiabetic agents

In 1957, phenformin, a medicinal biguanide, was manufactured alongside sulfonylureas. During further study, newer methods have produced meglitinide analogues, thiazolidinediones, α-glucosidase inhibitors, and most recently, dipeptidyl peptidase-4 (dpp-4) inhibitors [59]

Important features of oral hypoglycemic agents

One contemporary disease that has a significant influence on morbidity, morality, and the character of the afflicted person is diabetes mellitus. A common side effect of Cushing syndrome is diabetes mellitus, which is brought on by long-term exposure to glucocorticoids and manifests as a number of clinical symptoms, including central, weakness in proximal muscles, hirsutism, neuro physiological disturbance, autonomic neuropathy, macrovascular complications, digestive issues, dental issues, etc. [60]

CONCLUSION

Diabetes Mellitus, both Type 1 and Type 2, remains a significant global health challenge. Type 1 diabetes results from autoimmune destruction of insulin-producing cells, requiring lifelong insulin therapy, while Type 2 diabetes is primarily driven by insulin resistance and is closely linked to lifestyle factors such as obesity and physical inactivity. Both types, if not properly managed, can lead to severe complications, including cardiovascular diseases, neuropathy, nephropathy, and retinopathy. Early diagnosis, regular monitoring, appropriate medication, lifestyle modification, and patient education are essential strategies for effective management and prevention of disease progression. Future research is focused on developing more effective and personalized treatment strategies. Advancements in gene therapy, islet cell transplantation, and artificial pancreas technology offer hope for Type 1 diabetes. For Type 2 diabetes, novel drug targets, improved insulin sensitizers, and lifestyle-based interventions are under continuous investigation. Additionally, digital health tools like continuous glucose monitoring, mobile health apps, and artificial intelligence are expected to revolutionize diabetes care. With ongoing innovations and a better understanding of disease mechanisms, the future holds promise for improved management, prevention, and possibly even a cure for diabetes.

REFERENCES

1. Ismail, m.y., clinical evaluation of antidiabetic activity of trigonella seeds and aegle marvels leaves, worl appl skien j., 7(10): 1231-1234 (2009).

2. Arora, s., ojha, s.k., vohra, d., characterization of streptozotocin induced diabetes mellitus in swiss albino mice, glo j of pharmacal., 3(2): 81-84 (2009)
3. Joshie, n., ponnusamy, s.p., apache, m., antidiabetic activities of methanol leaf extract of costos pictus d. Don in alloxan-induced diabetic rats, j of health sci.,53(6): 655-663 (2007).
4. American diabetes association. Diabetes basics last reviewed by a cleveland clinic medical professional on 10/02/2018. 9500 euclid avenue, cleveland, ohio 44195 | 800.223.2273 | © 2020 cleveland clinic. All rights reserved.
5. Diabetes care 2018 jan; 41(supplement 1) cdc diabetes - prevalence cdc diabetes - diagnosed and undiagnosed.
6. Wassmuth r, linmark a. The genetics of susceptibility to diabetes, clinimmunol, immunopathology. 1989; 53:358 399,
7. Atkinson ma, eisenbarth gs. Type 1 diabetes new perspectives on disease pathogenesis and treatment, lancet. 2001; 358:221-229.
8.] hoit jj, tripathy bb, rao rh, yanik cs. Malnutrition and diabetes in the tropics, diabetes care. 1996; 19:1014 17,
9. Tripathy bb, samal kc. Overview and consensus statement on diabetes in tropical areas, diabetes mehtab rev. 1997; 13:63-76.
10. Bettered c, zanette f, pedini b, presto f, rapp lb, monsciotti cm et al., clinical and subclinical organ specific autoimmune manifestations in type 1 (insulin dependent) diabetic patients and their first-degreerelatives, diabetologia. 1983; 26:431-36.
11. Gupta op, joshi mh, daves sk. Prevalence of diabetes in india, adv mehtab discord. 1978; 9:147-65.
12. Rotter, j.i., vashem, c.m. and rimon, d.l. (1990) in diabetes mellitus (rifkin, h. And porte, d., eds.), pp. 378-413, elsevier, amsterdam
13. Who expert committee on diabetes mellitus: second report, world health organ. Tech. Rep. Ser., 646, pp.1-80 (1980).
14. Diabetes mellitus: report of a who study group, world health organ. Tech. Rep. Ser. 727, pp. 1-113 (1985).
15. Mayfield j.: am. Fam. Physician 58, 1355 (1998)
16. Satyanarayana t., katyayani b.m., latha h.e., et al.: phog. Mag. 2, 244 (2006).
17. World health organization, diabetes: key facts; world health organization: geneva, switzerland, 2011.
18. Centers for disease control and prevention. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the united states; u.s. department of health and human services, centers for disease control and prevention: atlanta, ga, usa, 2011.
19. Merriman, p.; bahadori, z.; azizi, f. Functional foods-based diet as a novel dietary approach for management of type 2 diabetes and its complications: a review. World j. Diabetes 2014, 5, 267–281.
20. Wang, z.; wang, j.; chan, p. Treating type 2 diabetes mellitus with traditional chinese and indian medicinal herbs. Evid. Based complement. Altern. Med. 2013, 343594, doi:10.1155/2013/343594.
21. Chang, c.l.; lin, y.; bartolome, a.p.; chen, y.c.; chiu, s.c.; yang, w.c. herbal therapies for type 2 diabetes mellitus: chemistry, biology, and potential application of selected plants and compounds. Evid. Based complement. Altern. Med. 2013, 378657, doi:10.1155/2013/378657
22. Chia lr, schlenk ea & dunbar-jacob j (2006) effect of personal and cultural beliefs on medication adherence in the elderly. Drugs aging 23, 191–202.
23. Ryan ea, pick me & marceau c (2001) use of alternative medicines in diabetes mellitus. Diabetes med 18, 242–245.
24. Bacchetta r, passerini l, gaminerie e, dai m, allan se. Defective regulatory and effector t cell functions in patients with foxp3 mutations, j clin invest. 2006; 116:1713-1722,
25. Merriman, p.; bahadori, z.; azizi, f. Functional foods-based diet as a novel dietary approach for management of type 2 diabetes and its complications: a review. World j. Diabetes 2014, 5, 267–281.
26. Verge cf, gianni r, kawasaki e, yu l, pietropaolo m, jackson ra et al., predicting type i diabetes in first degree relatives using a combination of insulin, gad, and ica512bdc/ia-2autoantibodiesdiabetes. 1996;45:926-33.
27. American diabetes association, diagnosis and classification of diabetes mellitus, diabetes care, 2014, 1.
28. Kumar cr. Basic pathology, prism pvt. Limited bangalore, 5th edition, 1992, 569-587.
29. Defronzo ra, bonadonna rc, ferrandine e, zimmet p. Pathogenesis of niddm, international textbook of diabetes mellitus. 1997, 635-712.
30. Jun sk, yoon yw. A new look at viruses in type 1 diabetes, diabetes/metabolism research and reviews. 2002; 19:8-31.
31. Wassmuth r, linmark a. The genetics of susceptibility to diabetes, clinimmunol, immunopathology. 1989; 53:358 399,
32. Who. Study group diabetes mellitus, technical report series no.727, world health organization, geneva, 1985.

33. Boney cm, verma a, tucker r, vohra br. Metabolic syndrome in childhood: association with birth weight, maternal obesity, and gestational diabetes mellitus pediatrics, 2005, 115.
34. Kumar cr. Basic pathology, prism pvt. Limited bangalore, 5th edition, 1992; 569-587
35. Lilliana s, mott dm, spraul m, ferraro r, foley je, roussin e et al., insulin resistance and insulin secretory dysfunction as precursors of non–insulin–dependent diabetes, n engl j med, 1993; 329: 1988-92.
36. Diabetes mellitus medical disorder written by: the editors of encyclopedia britannica see article history.
37. Mooy jm, goodenias pa, de vries h, valkenburg ha, bouter lm, kos tense pj et al., prevalence and determinants of glucose intolerance in a dutch population, diabetes care, 1995; 18: 1270-73.
38. Harris mi. Undiagnosed niddm, clinical and public health issues, diabetes care, 1993; 16: 642-52.
39. Who. Definition, diagnosis and classi ice tionofdiabetesmellitusanditscomplications: report of a who consultation. Part 1, diagnosis and classification of diabetes mellitus. Pages 1–59, 1999. Part- 1, document number who/ncd/ncs/99.2.
40. Médecins, c a major, m a morgan, t asrat, j s toohey, j m lien, and a t evans. Post ran dialversuspreprandialbloodglucosemonitor ing in women with gestational diabetes mellitus requiring insulin therapy. The new eng land journal of medicine, 333(19):1237–1241, 1995.
41. Rutland tyagi. Gestationaldiabetesmel lotus: diagnosisand treatment. Journalofmed ictal physiology & therapeutics, 1(3):1–3, 2017.
42. N chaudhary and n tyagi. Diabetes mellitus: anoverview. International journal of research and development in pharmacy & life science, 7(4):2278–0238, 2018.
43. Who. Definition, diagnosis and classi ice tionofdiabetesmellitusanditscomplications: report of a who consultation. Part 1, diagnosis and classification of diabetes mellitus. Pages 1–59, 1999. Part- 1, document number who/ncd/ncs/99.2.
44. N chaudhary and n tyagi. Diabetes mellitus: anoverview. International journal of research and development in pharmacy & life science, 7(4):2278–0238, 2018.
45. Ross and wilson. Anatomy and pathophysiology in health and illness, churchill livingstone elsevier, 11th edition, 2010, 227-229.
46. Wild s, rogic g, green a, scree r, king. Global prevalence of diabetes: estimates for the year 2000and projections for 2030, diabetes care, 2004; 27: 1047-53
47. Ross and wilson. Anatomy and pathophysiology in health and illness, churchill livingstone elsevier, 11th edition, 2010; 227-229.
48. Alemu s, dessie a, seid e. Insulin-requiring diabetes in rural ethiopia: should we reopen the case for malnutrition related diabetes, diabetologia. 2009; 52:1842-1845.
49. Kahiki oa, reddy mr, marzouk aa. Incidence of insulin-dependent diabetes (iddm) and non-insulin dependent diabetes (niddm) (0-34 years at onset) in benghazi, libya, diabetes res clin pact, 1996; 32: 165-173.
50. Atkinson ma, eisenbarth gs. Type 1 diabetes new perspectives on disease pathogenesis and treatment, lancet, 2001; 358: 221-229.
51. Harris mi. Undiagnosed niddm, clinical and public health issues, diabetes care. 1993; 16:642-52
52. Kahiki oa, reddy mr, marzouk aa. Incidence of insulin-dependent diabetes (iddm) and non-insulin dependent diabetes (niddm) (0-34 years at onset) in benghazi, libya, diabetes res clin pact. 1996; 32:165-173.
53. The world health report. Shaping the future, 2003.
54. Shaw j, zimmet p, de courtin m, dowse g, chiton p, grebo het al., impaired fasting glucose or impaired glucose tolerance, diabetes care. 1999; 22:399-402
55. Ramachandran a, snehalatha c, latha e, vijay v, viswanathan m. Rising prevalence of niddm in an urban population in india diabetologia1997; 40:232-237.
56. Scree r, shaw j, zimmet p. Diabetes and impaired glucose tolerance, diabetes atlas international diabetes federation, 2006, 15-103.
57. Sridhar gr, rao pv, ahuja mms. Epidemiology of diabetes and its complications in: rssdi textbook of diabetes mellitus, 2002, 95-112.
58. Who. Study group diabetes mellitus, technical report series no.727, world health organisation, geneva, 1985.
59. Verge cf, gianni r, kawasaki e, yu l, pietropaolo m, jackson ra et al., predicting type i diabetes in first–degree relatives using a combination of insulin, gad, and ica512bdc/ia-2autoantibodiesdiabetes, 1996; 45: 926-33.
60. Gupta op, joshi mh, daves sk. Prevalence of diabetes in india, adv mehtab discord. 1978; 9:147-65.