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Review Article

DIABETES MELLITUS: AN INCISIVE REVIEW

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Abstract

Diabetes mellitus is an endocrinologicalor metabolic order with an expanding worldwide pervasiveness and rate. Diabetes mellitus (DM), or just diabetes, is a gathering of metabolic ailments in which a man has high glucose, either in light of the fact that the body does not create enough insulin, or in light of the fact that cells don't react to the insulin that is created. This high glucose creates the established side effects of polyuria (visit pee), polydipsia (expanded thirst) and polyphagia (expanded craving). Expectedly, diabetes has been separated into three Types in particular: Type 1 DM or insulin-subordinate diabetes mellitus (IDDM) in which body fails to deliver insulin, and in a matter of seconds requires the individual to infuse insulin or wear an insulin pump. This is likewise named as "adolescent diabetes". Type 2 DM or non-insulin-subordinate diabetes mellitus (NIDDM), comes about because of insulin resistance, a condition in which cells neglect to utilize insulin legitimately, with or without a flat out insulin lack. This Type was beforehand alluded to as or "grown-up onset diabetes". The third fundamental Type is gestational diabetes which happens when ladies without a past history of diabetes build up a high blood glucose level amid her pregnancy. It might go before advancement of Type 2 DM.

Key Words: Diabetes Mellitus, Types, Pathophysiology, Pathogenesis, Management.

Introduction:

Diabetes mellitus (DM) is a metabolic issue coming about from a deformity in insulin discharge, insulin activity, or both.Insulin inadequacy thus prompts unending hyperglycaemia with unsettling influences of starch, fat and protein metabolism.[1-4]Diabetes mellitus has been classified into two Types i.e. insulin subordinate diabetes mellitus (IDDM, Type I) and non-insulin subordinate diabetes mellitus (NIDDM, Type II). Type I diabetes is an immune system infection portrayed by a nearby incendiary response in and around islets that is trailed by specific pulverization of insulin discharging cells though Type II diabetes is portrayed by fringe insulin resistance and impaired insulin secretion.[5]The nearness of DM shows expanded danger of numerous inconveniences, for example, cardiovascular sicknesses, fringe vascular infections, stroke, neuropathy, renal disappointment, retinopathy, visual impairment, removals etc.[6]Diabetes is the most widely recognized endocrine issue and by the year 2010, it is assessed that more than 200 million individuals worldwide will have DM and 300 million will in this way have the infection by 2025.[7-8]The indicative criteria and the arrangement of diabetes was to start with set forward by the World Health Organization (WHO) in 1965 then by the National Diabetes Data Group (NDDG) in 1979 and this was trailed by simp lified proposals by the WHO in 1980.[9-10]These WHO recommendations were changed somewhat in 1985.The most recent suggestions have been distributed by the American Diabetes Association (ADA) in 1997 and by the WHO in 1999. Both gatherings concur on the suggestions what's more, criteria.[11-12]

Symptoms

Symptoms are comparative in both Types of diabetes yet they shift in their force. Side effects grow all the more quickly in Type 1 diabetes and more normal. The Symptoms incorporate polyurea, polydipsia, polyphagia, weight reduction, exhaustion, obscured vision, and candidiasis. Longstanding Type 1 DM patients are vulnerable to micro vascular complications; and macro vascular illness (coronary vein, heart, and fringe vascular sicknesses). Manifestations in Type 2 DM are compa-

rable however slippery in onset.

Most cases are analyzed in view of entanglements or unexpectedly. Type 2 DM carries a high danger of extensive vessel atherosclerosis usually connected with hypertension, hyperlipidemia and obesity. Most patients with Type 2 diabetes kick the bucket from cardiovascular entanglements and end stage renal disease. Geographical contrasts exist in both the greatness of these issues and their relative commitments to general morbidity and mortality.

Table no 1: Classification of Diabetes

Type 1(1a,1b)	ß-cell destruction with little or no endogenous insulin secretory capacity Autoimmune Idiopathic
Type 2	Ranges from relative insulin deficiency to disorders of insulin secretion and insulin resistance
Other specific types	Genetic defects of ß-cell function Genetic defects in insulin secretion Diseases of the exocrine pancreas Endocrinopathies Drug-induced or chemical induced Infections (congenital rubella, cytomegalovirus and others) Uncommon forms of immune mediated diabetes Other genetic syndromes sometimes associated with diabetes Gestational diabetes

Epidemiology

It is evaluated that 366 million individuals had DM in 2011; by 2030 this would have ascended to 552 million. The number of individuals with Type 2 DM is expanding in each nation with 80% of individuals with DM living in low-and center salary nations. DM brought on 4.6 million passings in 2011. It is assessed that 439 million individuals would have Type 2 DM by the year 2030.The occurrence of Type 2 DM differs significantly from one geological district to alternate as a consequence of natural and way of life hazard factors. It is anticipated that the commonness of DM in grown-ups of which Type 2 DM is getting to be noticeable will increment in the following two decades and a significant part of the expansion will happen in creating nations where the dominant part of patients are matured somewhere around 45 and 64 years.[13-14]

Pathophysiological aspects

Type 2 DM is described by insulin absence of care as a consequence of insulin resistance, declining insulin creation, and possible pancreatic beta-cell disappointment. This prompts an abatement in glucose transport into the liver, muscle cells and fat cells. There is an expansion in the breakdown of fat with hyperglycemia.[15]Type 1 diabetic patients are typically youthful (youngsters or teenagers) and when they first create side effects. There is an acquired inclination, with a 10-crease expanded frequency in first-degree relatives of a list case, and solid relationship with specific histocompatibility antigens (HLA Types). Investigations of indistinguishable twins have demonstrated that hereditarily inclined people should also be presented to a natural component, for example, viral contamination. Viral contamination may harm pancreatic B cells and uncover antigens that start a self-sustaining immune system handle. The patient gets to be obviously diabetic just when more than 90% of the B cells have been pulverized. In this Type, insulin lack weakens long haul potentiating and may prompt deficiencies in learning and memory. Type 2 diabetes is joined both by insulin resistance and by weakened insulin emission, each of which are vital in its pathogenesis. Such patients are frequently large and normally exhibit in grown-up life, the rate rising dynamically with age as B-cell work decreases. In this insulin resistance prompts both Aβ plaque arrangement and tau hyperphosphorylation. Amid hyperinsulinemia, insulin and Aβ vies for insulin degrading compound, prompting Aβ gathering and plaque arrangement. A decline in insulin receptor flagging prompts hindrance of Akt and DE phosphorylation (enactment) of GSK-3β and results in tau hyperphosphorylation.[16-17]

Pathogenesis of Type 1 diabetes mellitus

Type 1 diabetes mellitus is an endless immune system sickness connected with particular decimation of insulin-delivering pancreatic β-cells. The onset

of clinical ailment speaks to the end phase of β-cell decimation prompting Type 1 diabetes mellitus. A few elements describe Type 1 diabetes mellitus as an immune system sickness:

1. Presence of immune-competent and accessory cells in infiltrated pancreatic islets;
2. Association of susceptibility to disease with the class II (immune response) genes of the major histocompatibility complex (MHC; human leucocyte antigens HLA);
3. Presence of islet cell specific autoantibodies;
4. Alterations of T cell mediated immunoregulation, in particular in CD4+ T cell compartment;
5. The involvement of monokines and TH1 cells producing interleukins in the disease process;
6. Response to immunotherapy ;
7. Frequent occurrence of other organ specific autoimmune diseases in affected individuals or in their family members.

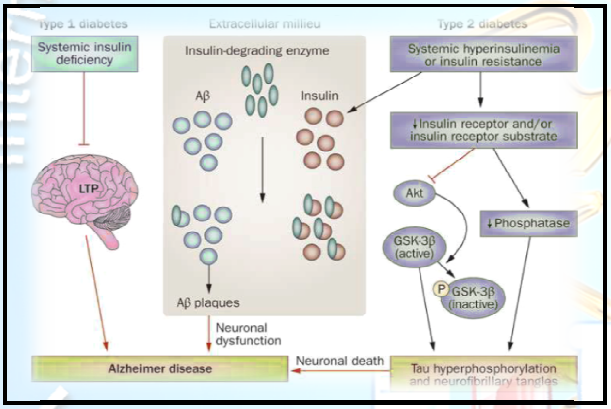

Figure no 1: Pathophysiology of Type I and Type II diabetes. Abbreviations: Aβ- Amyloid β, GSK-3β-glycogensynthase kinase 3β, LTP- long term potentiation, P- Phosphate

The pathogenesis of specific β-cell decimation inside the islet in Type 1 DM is hard to take after because of checked heterogeneity of the pancreatic injuries. At the onset of clear hyperglycemia, a blend of pseudo atrophic islets with cells delivering glycogen (a phones), somatostatin (d cells) and pancreatic poly-peptide (PP cells), typical islets, and islets containing both b-cells and invading lymphocytes and monocytes might be seen.[18]Lymphocytic invasion is discovered just in the islet containing remaining β-cells and is likely that the chronicity with which Type 1 DM creates mirrors this heterogeneity of islet injuries. As opposed to this chronicity in the characteristic history of the illness, β-cells are quickly decimated when pancreas is transplanted from indistinguishable twin benefactors into their long haul diabetic twin mates without immunosuppression. In these cases, enormous insulitis grows quickly with invading T lymphocytes showing an anamnestic immune system response. Furthermore, this perception likewise shows that the endless time course in Type 1 DM (however not in a transplanted pancreas) is an outcome of down administrative wonders partaking in immunopathology-beginning of the malady.Enactment of islet antigen - particular CD4+ T cells give off an impression of being total essential for the improvement of diabetes in every single creature model of Type 1 DM. CD4+ islet particular T-cell clones got from diabetic NOD mice, when infused into prediabetic or non-diabetes inclined mice, actuate insulitis and diabetes.[18] It was likewise reported that CD4+ T cells are adequate to prompt insulitis while CD8+ T cells add to the seriousness of the harm. These discoveries together with the proof that insulitis in unending union versus have malady may happen without CD8+ T cells propose that CD4+ T cells might be the main immunocompetent cells required in the ailment procedure. Be that as it may, it appears that one and only subset of CD4+ T cells are in charge of infection enlistment.[19]CD4+ T cell bearing alloantigen RT6 are truant in diabetes inclined BB rats and seem to shield AO rats from MLD-STZ initiated diabetes. Down-direction of diabetogenic immune system reaction by the spleen cells got from creatures treated with adjuvants could likewise be clarified by CD4+ T cell subsets interaction [20] Abnormal state of THI Type cytokines IL-2 and interferon g are found to connect then again to improve enlistment of immune system diabetes in exploratory models.[21] The TH-1 Type cells, and specifically their item IFN-g, initiate macrophages. In creature, models of Type 1 DM electron minute investigations of pancreata showed that macrophages are the principal cell Type attacking the islets.[22] In vitro thinks about and thinks about on perfused pancreas propose that Interleukin 1 (IL-1) and tumor putrefaction calculate (TNFa), two cytokines mainly created by macrophages, prompt basic changes of β-cells and concealment of their insulin discharging limit.[23] In any case, it appears that IL-1

and TNF don't contribute appreciably to the cytotoxic movement of macrophages .Interferon g is additionally an intense activator of macrophages for nitric oxide amalgamation. As of late, proof has been given demonstrating that NO synthase action is included in diabetes improvement.[24] These information showed, surprisingly, that nitric oxide might be a pathogenic figure autoimmunity and proposed a probability that another class of immunopharmacological operators, fit for adjusting nitric oxide discharge might be tried in the anticipation of Type 1 DM advancement.[22]

Pathogenesis of type 2 diabetes

Under typical physiological conditions, plasma glucose focuses are kept up inside a restricted range, regardless of wide variances in free market activity, through a firmly managed and dynamic connection between tissue affectability to insulin (particularly in liver) and insulin discharge).[25] In Type 2 diabetes these systems separate, with the outcome that the two fundamental neurotic deformities in Type 2 diabetes are disabled insulin discharge through a brokenness of the pancreatic β-cell, and weakened insulin activity through insulin resistance .[26]Type 2 diabetes mellitus has a more prominent hereditary relationship than Type 1 DM, the pathogenesis of Type 2 diabetes mellitus is portrayed by hindered insulin discharge and insulin resistance. The 100% concordance rate in indistinguishable twins is thought to be over-evaluated, because of a choice or reporting predisposition. A populace based twin study in Finland has demonstrated a concordance rate of 40%, and natural impact might be a conceivable purpose behind the higher concordance rate for Type 2 diabetes mellitus than for Type 1 diabetes mellitus [27]Type 2 diabetes mellitus influences 1 to 2% of Caucasians. However it is much higher in some ethnic gatherings, for example, Pima Indians and approaches half in South India. This demonstrates hereditary elements are more imperative than ecological elements. With the exception of development onset diabetes of the youthful (MODY), the method of legacy for Type 2 diabetes mellitus is indistinct. MODY, acquired as an autosomal predominant quality, may come about because of changes in glucokinase quality on chromosome 7p. Glucokinase is a key compound of glucose digestion system in beta cells and the liver.[28] MODY is characterized as hyperglycemia analyzed before the age of a quarter century and treatable for more than five years without insulin in situations where islet cell antibodies (ICA) are negative and HLA-DR3 and DR4 are heterozygous. MODY is uncommon in Caucasians, under 1%, and more basic in blacks and Indians, more than 10% of diabetics. Ceaseless entanglements in MODY were thought to be remarkable yet later were observed to be more regular, demonstrating its heterogeneity. Considering MODY as a different substance may disguise its relationship with particular hereditary maladies; and without a distinct hereditary marker, it ought to be dealt with as Type 1 DM.[29] ID of a drivel transformation in the glucokinase quality and its linkage with MODY was accounted for without precedent for a French family, ensnaring a change in a quality required in glucose digestion system in the pathogenesis of Type 2diabetes mellitus .[30] Later, sixteen transformations were distinguished in 18 MODY families. They included 10 transformations that brought about an amino corrosive substitution, 3 that brought about the blend of truncated protein, and 3 that influenced RNA preparing. Hyperglycemia in these families was typically mellow and started in youth, while the hyperglycemia of MODY families without glucokinase transformations generally appeared after pubescence. Subatomic hereditary studies in Type 2 diabetes mellitus, with the special case of MODY, have not been as effective as in Type 1 diabetes mellitus. Changes in the insulin quality prompt the combination and discharge of unusual quality items, prompting what are called insulinopathies.[31] The vast majority of the patients with insulinopathies have hyperinsulinemia, acquired in autosomal form, heterozygous for typical and mutant alleles, and regularly react to exogenous insulin organization.

The association of the polymorphic (hyper variable) 5' flanking locale of the human insulin quality and Type 2 diabetes mellitus is inadequate in some populace bunches, showing that it might be one of numerous components in a multifactorial illness. Indeed, even MODY patients have appeared no relationship with this district. It was said before that there is a solid relationship between HLA-DR3/4 what's more, Type 1 diabetes mellitus. It was additionally reported that such an affiliation is available with Type 2 diabetes mellitus, rendering

HLA-DR3/4 markers for beta cell annihilation in these patients.[32]pancreatic anomalies in islet secretory cells in Type 2 diabetes mellitus are noted in beta, alpha and delta cells of the islets. Absconds including insulin discharge incorporate relative lessening in basal normal, and the alpha cell mass is expanded prompting hyperglucagonemia. The islets show hyalinization and amyloid statement, containing islet amyloid polypeptide (IAPP) or amylin. This is a minor secretory peptide of the beta cells discharged alongside insulin and C-peptide, however its part inthe pathogenesis of Type 2 DM is not surely knew This amylin is thought to create insulin resistance.[33] IAAP is diminished with movement of Type 2 DM.Suggest contact between beta cells and amyloid store in Type 2 DM is noted by electron microscopy. Far from the islets in the exocrine pancreas, greasy invasion and diffuse fibrosis are apparent. Blemished islet cell capacity is the essential occasion which might be because of an immune system response creating hyperglycemia in Type 2 DM .[34] The insulin receptor quality is situated on chromosome 19 and it encodes a protein having alpha and beta subunits including the trans membrane space and the tyrosine kinase area.Changes influencing the insulin receptor quality have been distinguished and their relationship with Type 2 diabetes mellitus and Type An insulin resistance is recognized. Type 2 DM patients have a trademark bear, support truncal corpulence. Supplement piece has additionally been observed to be a hazard calculate for creating Type 2 DM, where expanded fat and diminished starch utilization have added to hyperinsulinemia of corpulence. Dietary filaments, both dissolvable and insoluble, enhance Type 2 DM. It is additionally found that straightforward sugars don't specifically bring about diabetes. Insufficiency of micronutrients, for example, chromium and copper, is observed to be a critical reason for Type 2 DM in a minority of cases. Push has likewise been thought to instigate Type 2 DM. Really, corpulence and over-accessibility of sustenance instead of push are the contributing components to Type 2 DM. Subsequently, when lasting change in dietary propensities is built up, a few people ought to be permitted to get away from the "long lasting" determination of Type 2 DM.[35]

Diagnosis

As per the America Diabetes Association (ADA), the fasting glucose focus must to be utilized in routine screening for diabetes; yet postprandial glucose, arbitrary glucose and glucose resilience test are additionally utilized for glucose assurance. For the conclusion of diabetes, no less than one measure must apply

- Symptoms of diabetes (polyurea, polydipsia, unexplained weight loss, etc.) as well as casual plasmaglucose concentration = 11.1 mmol/L (200 mg/dL).
- Fasting plasma glucose = its normal range is 70-110 mg/dl with no caloric intake for at least 8 h.

The World Health Organization (WHO) order incorporates both clinical stages (norm glycaemia,weakened glucose resilience/impeded fasting glucose (IGT/IFG), diabetes) and etiological Types of diabetes mellitus, indistinguishable to the ADA aside from that WHO bunch incorporates characterization once in the past known as gestational weakened glucose resistance (GIGT) and GDM: fasting glucose = 7.0 mmol/L (126 mg/dL) what's more, or 2-h glucose = 7.8 mmol/L (140 mg/dL) after a 75-g OGTT.[36]

Management of Diabetes Mellitus

Way of life administration is clearly the foundation of management of diabetes mellitus. It is perceived just like a fundamental piece of diabetes and cardiovascular infection counteractive action. Meta-examinations show that way of life intercessions, including diet and physical movement, prompted a 63% lessening in diabetes occurrence in those at high hazard. Way of life change programs have shown empowering change in hazard variables for diabetes; in any case, the impact on diabetes rate has not been accounted for. The dietary administration of diabetes mellitus is a supplement of way of life administration. It positively affects long haul wellbeing and personal satisfaction. Dietary administration goes for ideal metabolic control by setting up a harmony between sustenance admission, physical action, and drug to stay away from confusions. In Type 2 diabetes, the dietary target is for enhanced glycemic and lipid levels and weight reduction as proper.[37]

Insulin and oral hypoglycemic drugs

Insulin treatment should to intend to copy nature, which is astoundingly effective both in restricting postprandial hyperglycemia and avoiding hypoglycemia between meals. Site of bodyinsulin infusion is similarly critical for better and safe activity of insulin and can be given by intramuscular or intravenous route. Distinctive arrangements of insulin are accessible, for example, human insulin, meat insulin, pork insulin. Insulin treatment is no free from inconveniences and unfavorable impacts. The most critical unfavorable impact are weight pick up and hypoglycemia when improper measurements of insulin is taken and when there is confuse amongst dinners and insulin injection.[38,39] Weight increase subsequent to beginning insulin treatment for uncontrolled diabetes is an unavoidable outcome and is the aftereffect of expanded truncal fat and muscle mass. This is additionally because of decreased vitality misfortunes through glycosuria.[40] Sulphonylureas, for example, glibenclamide, glipizide and biguanides, for example, metformin, phenformin are oral hypoglycemic medications. Sulfonylureascause hypoglycemia by empowering insulin discharge from pancreatic ß-cells. They tie to sulfonylurea (SUR) receptors on the ß-cell plasma layer, bringing about conclusion of adenosine triphosphate (ATP)- delicate potassium channels, prompting depolarization of the cell layer. This thusly opens voltage gatedchannels, permitting inundation of calcium particles and ensuing discharge of preformed insulin granules. Intense organization of sulfonylureas to Type 2 DM patient's builds insulin discharge from the pancreas furthermore may facilitate increment insulin levels by diminishing hepatic leeway of the hormone. Beginning studies demonstrated that a practical pancreas was essential for the hypoglycemic activities of sulfonylureas. Biguanides, for example, metformin is antihyperglycaemic, not hypoglycemic. It doesn't bring about insulin discharge from the pancreas and does not bring about hypoglycemia, even in costly doses.[41]it has been appeared to increment fringe take-up of glucose, and to decrease hepatic glucose yield by around 20-30% at the point when given orally yet not intravenously. [42]

Newer Approaches in treatment of Diabetes Mellitus[46]

- Peroxisome proliferator Activated Receptors (PPARs)
- Glucagon like Peptide-1 Hormone
- Liver Selective Glucocorticoid Antagonists
- B3- Adrenoreceptor Agonist
- A-Lipoic Acid
- Proteins Tyrosine Phosphatase -1b
- Glycogen Synthase Kinase (GSK-3)
- AMP-Activated Protein Kinase
- Estrogen Receptors: New Players in DM
- Salsalate
- Resveratrol
- L-Arginine
- Gene Therapy
- Anakinra
- Otelixizumab

Herbal treatment of diabetes

In the most recent couple of decade's eco-accommodating, bio-accommodating, financially effective and moderately protected, plant-based have moved from the periphery to the standard with the expanded research in the field of traditional medicine. There are a few writing audits by various writers about against diabetic herbal agents. WHO has recorded 21,000 plants, which are utilized for restorative purposes the world. Among these 2500 species are in India, out of which 150 species are utilized industrially on a genuinely extensive scale. India is the biggest maker of restorative herbs and is known as the herbal garden of the world.[47]

Conclusion

The term diabetes mellitus incorporates a few different metabolic issue that all, if left untreated, result in unusually high centralization of a sugar called glucose in the blood. Diabetes mellitus Type 1 comes about at the point when the pancreas no longer creates huge measures of the hormone insulin, normally inferable from the immune system pulverization of the insulin-creating beta cells of the pancreas. Diabetes mellitus Type 2, in complexity, is currently thought to come about because of immune system assaults on the pancreas and/or insulin resistance. The

pancreas of a man with Type 2 diabetes might create ordinary or even strangely a lot of insulin. The fundamental objective of diabetes management is, beyond what many would consider possi-

ble, to reestablish sugar digestion system to an ordinary state. It is important to have an enhanced comprehension of its etiology, pathogenesis and pathophysiology to center helpful and research endeavors properly. A planned multidisciplinary approach is required that includes researchers, general wellbeing practition-ers, instructors, clinicians and diabetics, with support from government powers and nongovernmental associations to lessen the frequency of diabetes essentially.

Table no 2: Features of FDA approved oral hypoglycemic therapies in type II diabetes[43-45]

Drug	Advantages	Disadvantages	Lowers HbA1C	Most commonly reported adverse events	Failure rate
Sulfonylureas (1st &2nd generation: acetohexamide, glipizide, glyburide, glimepiride	Inexpensive, improved lipid profile by lowering Triglycerides	Weight gain, and rare but severe hypoglycemia	1.5%	Rare allergies, SIADH can be caused by first generation and disulfiram reaction with alcohol	10-15%
Short-acting insulin secretagogues (nateglinide, repaglinide) Biguanides (metformin)	Lower triglycerides, uncommon hypoglycemia Metformin is only FDA approved oral diabetic in children More or =to 10 years. Lowers TG and total cholesterol, no hypoglycemia, no weight gain	Weight gain similar to hypoglycemia Minimal effect on HDL, used as Monotherapy does not sustain HbA1C reductions.	0.6%-1.0 % 1.5 - 2.0%	Experience limited Gastrointestinal side effect (Diarrhea) minimized by XR Form. Lactic acidosis	Not reported 10-15% similar to Sulfonylureas
TZDs (pioglitazone, rosiglitazone)	Lower TG, and raises HDL, no hypoglycemia effect	Weight gain, elevated ALT levels, andedema noted.	0.5-1.5%	Gastrointestinal adverse effects atelevated dosages, rare liver failure, as we mentioned fluidretention (CI in class 3, 4 CHF)	Not known

Drug	Advantages	Disadvantages	Lowers HbA1C	Most commonly reported adverse events	Failure rate
Inhaled Insulin (Exubera)	Rapid-acting insulin, first easily inhaled insulin, reduce FPGby 23mg/dl, no refrigeration needed, no batteries, easytechnique jubbreathe	Contraindicated insmokers and inpatients who stoppedsmoking less than 6months ago , CI in COPD patients	1.4%	Respiratory adverse events mostly, PFTs should be checked, hypoglycemia, dry mouth	Not reported

Table no 3: Herbal drugs used for the treatment of diabetes[48]

Botanical name	English name	Local name	Family
Acacia Concina DC	Shikakai	Khangthur	Mimosaceae
Aloe vera Mill.	Aloe	Kunwarghandel	Liliaceae
Adhatoda vasica Nees	Vasaka	Bekkar	Acanthaceae
Alocacia indica schott	Giant Taro	Mankachu	Araceae
Anana scomosus (L) merr	Pineapple	Matikathal	Bromeliaceae
Annona reticulata L.	Bullock's heart	Atlas	Annonaceae
Antidesmaacidum Retz	Devil's tree	Nikhutenga	Euphorbiaceae
Bombox malabaricum DC	Red cotton tree	Simolu	Bombacaceae
Brassica juncea(L)Czern.	Mustard greens	Sorioh	Brassicaceae
Cassia fistula L.	Golden shower cas-sia	Sunaru	Caesalpinaceae
Cassia occidentalis Linn.	Coffee-senna	Bonoriadadol	Caesalpinaceae
Cassia sophera Linn	Senna sophera	Bonmadelua	Caesalpinaceae
Cyperus rotundus L.	Nutgrass	Deela	Cyperaceae
Dodonaea viscosa (L) Jacq.	Switch sorrel	Sanatha	Sapindaceae
Equisetum debile Roxb.	Horse Tail	Lai-utang	Equisteraceae
Ficus bengalensis L.	Banyan	Bohr	Moraceae
Ipomoea aquatica Forssk.	Water spinach	Kalmou	Convulaceae
Imperata cylindrical Beauv	Kunai grass	Ulukher	Poaceae
Hordeum vulgare L.	Barley	Jo	Poaceae
Psidium guyava Linn	Guava	Madhuri	Myrtaceae
Taraxacum officinale Weber.	Dandelion	Doddak	Asteraceae
Vigna sinensis (Burm. f.) Walp.	Cow bean	Lobia	Fabaceae
Withania coagulens (L.) Dunal	Wintercherry Chitta	Verino	Solanaceae
Zizyphus jujuba Mill	Chinee tree	Beri	Rhamnaceae

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