IJNRD.ORG ISSN : 2456-4184
INTERNATIONAL JOURNAL OF NOVEL RESEARCH AND DEVELOPMENT (IJNRD) | IJNRD.ORG
An International Open Access, Peer-reviewed, Refereed Journal

A Review On Diabetes Mellitus Type 1 And Type 2

STUDENT NAME : ZENDE GAURI SAMPAT

GUIDE : PURANIK.A.K

PRINCIPAL : S.J. INGALE.

ZENDE. G . S , ZAREKAR. S . G , RAYKAR. S . D .

DESIGNATION : ASSISTANT PROFESSOR

DHARMARAJ SHAIKSHANIK PRATISHTHAN COLLEGE OF PHARMACY WALKI,

AHILYANAGAR , 414006.

ABSTRACT :

Diabetes mellitus is a metabolic disorder characterized by the chronic either.The chronic condition characterized by hyperglycemic defects in insulin secretion.Various methods are used to diagnose diabetes in various people.The WHO employs the oral glucose tolerance test.

The pathogenesis of selective B-cells distruction with in the type 1 diabetes mellitus. The pancreatic B-cell function the progression of pathogenesis of type 2 diabetes mellitus. An estimated 366 million individual word wide had diabetes in 2011.According to a report by the international diabetes federation. An estimated in almost around the 552 million individual word number is 2030. The gestational and other chronic hyperglycemia are the environment genetic abnormalities infections and some medications are the hallmark of diabetes mellitus.

KEYWORDS :

Diabetes mellitus , Diagnosis , Glycemic Management ,Epidemiology And Etiology, Pathogenesis And Pathophysiology

INTRODUCTION :

It is a chronic disease associated with abnormally high levals of the sugar glucose in the blood. It caused by inherited acquired deficiency in production of insulin by the pancreas.[1]

Type 1 diabetes mellitus is a chronic auto-immune disease characterized by increased blood glucose level. Which are due to the insulin deficiency of the pancreatic B-Cells.[2,3,4] The pathogenesis of type 1 diabetes mellitus has been sugested to be a continuum that can be divided into stages that related to be the detection of autoantibodies and progress to B-Cell distruction.[5] Diabetes mellitus is a chronic disease that is associated with high morbidity and mortality from its complication.[6] Diabetes mellitus is the complication risk is directly related to high blood glucose lavels.[7]

The most patients with type 2 diabetes mellitus have a combination of risk hyperlipidermia.[8] Some randomized clinical trials demonstrated the benefit of blood pressure lowering in diabetes. The grater number of medication compliance seems to be a significant barrier to the attainment of positive clinical outcome among the type 2 diabetes patient in both developed and developing countries. The more drug related problem such as adverse drug reaction ,drug interaction

,medication non-compliance no valid medical indication.[9,10,11] It is used in traditional ayurvedic practices to help managing diabetes and blood sugar levals. It belong to costaceae family and also a unique protein that has hyperglycemic properties that reduced blood glucose levels.[12]

EPIDEMIOLOGY AND ETIOLOGY :

ETIOLOGY Of DIABETES :

Both type of diabetes shear are central feature: Elevated blood sugar (Glucose) level due to absolute or relative insufficiencies of insulin a hormone produced by pancreas.

TYPE 1 DIABETES:

The primary antibodies found in 90% of type 1 diabetics are against cytoplasmic proteins. The incidence of type 1 diabetes mellitus is increasing world wide and it is estimated that nearly 90,000 children are diagnosed each year.[13] According to international diabetes federation 88% of the adult population world wide has diabetes. Type 1 diabetes mellitus represent around 10% of all cases of diabetes.[14] The majority of type 1 diabetes mellitus are individuals are diagnosed either ate around the age of 4 to 5 years.[22] The annual increase in the type 1 diabetes mellitus is incidence in children under 15 years is 3.4%.[21]

TYPE 2 DIABETES :

The type 2 diabetes is the predominant form of diabetes & accounts for at least 90% of all cases of diabetes mellitus.[15] The incidence of diabetes increase with age with most cases being diagnosed after the age of 40 years.[16] Type 2 diabetes is heterogenous disorder caused by a combination of genetic factor related to impaired insulin secretion.[17]

PATHOGENESIS AND PATHOPHYSIOLOGY :

DIABETES MELLITUS :

Hyperglycemia & both physiological & behavioral reactions are directly related. The brain detects hyperglycemia & sands a message to the pancreas & other organs to lessen its effects through nerve impulses whenever it occurs.[18]

TYPE 1 DIABETES MELLITUS :

The pathogenesis of selection B-Cell distruction with in the islet in type 1 diabetes mellitus is difficult to follow due to marked heterogeneity of the pancreatic lesions.[19] The resultant inappropriately elevated glucagons levels exacerbate the metabolic defects due to insulin deficiency. There are multiple biochemical mechanism that account for impairment of tissues response to insulin.[20]

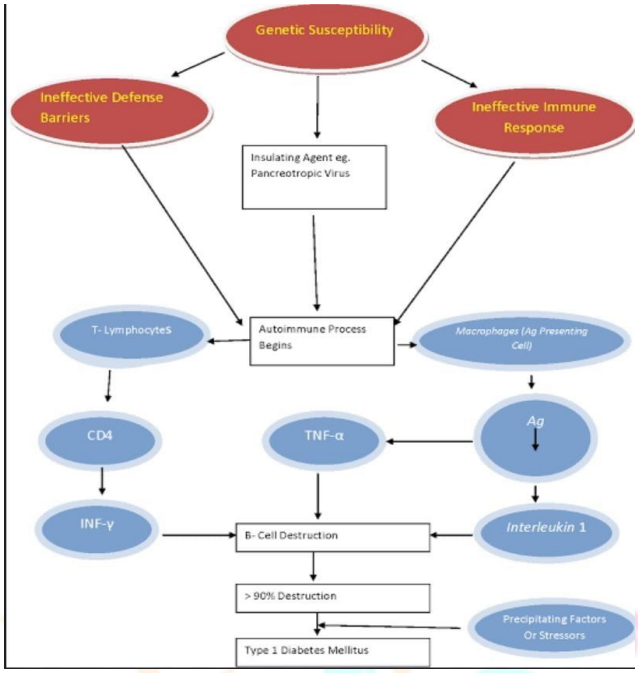

Fig : Pathogenesis of type 1 DM

TYPE 2 DIABETES MELLITUS :

A population based twin study in finland has shown a concordance rate 40% & environmental effect may be a possible reson .[23] The higher concordance rate for type 2 diabetes mellitus. The progression from impaired glucose tolerance to diabetes mellitus.[24] The level of insulin declines indicating that patient with NIDDM (NON – INSULIN DEPENDENT DIABETES MELLITUS) have increased insulin secretion. The nuclear hormone receptor super family of proteins in the etiology of type 2 diabetes. The synthesis of biochemically active compounds from vascular endothelial calls & immune cells.

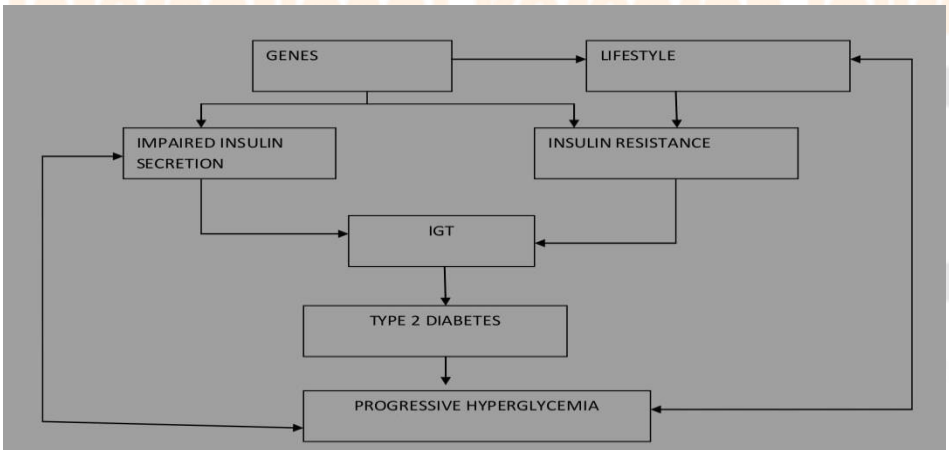

Fig : Pathogenesis of type 2 DM

DIAGNOSIS :

The patient describe in the vignette has risk factor (Obesity, Hypertension & A family of Diabetes) & should be screened.[25] About the 25% of patients with type 2 diabetes mellitus already have microvascular complications at the time of diagnosis suggesting that they have had the disease for more than 5 years at the time of diagnosis. [26,27] The identification of patients with diabetes or pre-diabetes by screening allow for earlier intervention.[28] With the potential reduction in future complication rates although randomized trials are lacking to definitively show benefit. [39]

SYMPTOMS :

Many patient with diabetes may have no symptoms especially in the early phase of the disease. The symptoms of high blood glucose level. When the blood glucose level rises about 160 to 180 mg/dl (8.9 to 10.0 mmol/l) glucose spills into the urine. Blurry vision, Increased thirst or the need to urinate, Feeling tired or ill, Recurring skin, Gum or bladder infections, Unexpected weight loss, Slow healing of cuts and bruises, Loss of feeling in the feet or tingling feet.

TREATMENT :

Dite

Exercise

Education

Weight Loss

Oral hypoglycemic therapy

In type 1 diabetes - Insulin Injection

In type 2 diabetes – Often medications by mouth & sometimes insulin or other medications by injection.

Dite, Exercise & Education are the cornerstones of treatment of diabetes . Weight loss is important for people who have overweight. Some people with type 2 diabetes & mildly elevated glucose levels can start with diet, exercise & weight loss only. However, in people with more severe glucose abnormalities or in whom lifestyle modification is not sufficient to normalize glucose, diabetes medication are required. People with type 1 diabetes (No matter their blood glucose levels) require medication when first diagnosed.[30]

BLOOD GLUCOSE LEVEL :

Fasting serum glucose(mg/dl)	Diagnosis
Below 110	Normal
Between 110 & 126	Pre-diabetes
Above 126	Diabetes
Post prandial blood sugar	Diagnosis
<140 mg/dl	Normal
140-200 mg /dl	Pre-diabetic
>200 mg /dl	Diabetic

TABLE 1 : BLOOD GLUCOSE LEVEL

LIPID PROFILE :

Result of lipid profile	Classification
LDL	
<100	Optimal
100-129	Near optimal
130-159	Borderline high
160-190	High
>190	Very high
Serum triglycerides	
<150	Optimal

150-199	Borderline high
200-499	High
>500	Very high
HDL Cholesterol	
<40	Low
>60	High

TABLE 2 : LIPID PROFILE

ORAL MEDICATION :

1) Sulfonylureas

2)Biguanides

3)Thiazolidinediones

4)Meglitinides

5)Alpha-glycosidase inhibitor

CLASSES OF ORAL HYPOGLYCEMIC AGENT :

Target insulin secretion : Sulfonylureas, Meglitinides

Target insulin resistance : Biguanides, thiazolidinediones

Target glucose absorption from intestine : Alpha-glucosides inhibitors.[31]

ORAL HYPOGLYCEMIC MEDICATION :

DRUG CLASS	DRUG NAME	BRAND NAME	MECHANISM OF ACTION
Sulfonylureas (second generation)	Glimepiride Glipizide Glyburide	Amaryl Glucotrol Duabeta Glynase Pres Tab Micronase	ncrease Insulin secretion by pancreatic beta call
Biguanides	Metformin	Glucophage	ngibit glucose production by the iver
Meglitinides	Repaglinide Nateglinide	Prandin Starlix	ncrease insulin secretion by pancreatic beta cell
Thiazolidinediones (TZDS)	Pioglitazone Rosiglitazone	Actors Acandia	ncrease glucose uptake by skeletal muscle
Alpha-glucosidase	Acarbose Miglitol	Precost Glyset	nhibit carbohydrate Absorption in the small intestine

TABLE 3 : ORAL HYPOGLYCEMIC MEDICATION

TYPES OF DIABETES COMPLICATIONS :

1) Liver damage in diabetes

2) Kidney damage in diabetes

3) Nerve damage in diabetes

4) foot problem in diabetes

CONCLUSION :

Diabetes mellitus is a chronic condition characterized by impaired glucose regulation due to either insufficient insulin production or the body's inability to use insulin effectively .

Type 1 diabetes is an autoimmune disease where the immune system attacks the insulin-producing beta cells in the pancreas. This leads to little or no insulin production, requiring lifelong insulin therapy. It is commonly diagnosed in children or young adults but can occur at any age .

Type 2 diabetes is more common and primarily results from insulin resistance, where the body's cells fail to respond effectively to insulin. Over time, insulin production may decrease. It is often associated with lifestyle factors such as obesity, poor diet, and lack of physical activity, though genetic factors also play a role . Both types can lead to severe complications if not managed properly, including cardiovascular diseases, kidney failure, nerve damage, and vision loss. While type 1 diabetes is managed through insulin therapy, type 2 diabetes can often be managed or prevented with lifestyle modifications, oral medications, and, in some cases, insulin therapy . In conclusion, early diagnosis and effective management of both types of diabetes are crucial to prevent complications and improve quality of life.

REFERENCES :

1) Shinde S, Surwade S, Sharma P. Costus IGN-5 insulin plant approach for SCID research: Its preparations as a remedy for diabetes mellitus. Int J Pharm Sci Res. 2022;13(4):1551-8. doi:10.13040/IJPSR.0975-8232.13(4).1551-58.

2) Gepts W. Pathologic anatomy of the pancreas in juvenile diabetes mellitus. Diabetes. 1965;14(6):619-33.

3) Eisenbarth GS. Type I diabetes mellitus: A chronic autoimmune disease. N Engl J Med. 1986;314(21):1360-8.

4) Atkinson MA, Eisenbarth GS, Michels AW. Type 1 diabetes. Lancet. 2014;383(9911):69-82.

5) Insel RA, et al. Staging presymptomatic type 1 diabetes: A scientific statement of JDRF, the Endocrine Society, and the American Diabetes Association. Diabetes Care. 2015;38(10):1964-74.

6) International Diabetes Federation. Diabetes Atlas. 4th ed. Brussels: International Diabetes Federation; 2010. p. 71-3.

7) American Diabetes Association. Implications of the Diabetes Control and Complications Trial. Diabetes Care. 2002;25(1):25-7.

8) Abuissa H, Bel DS, O'Keefe JH Jr. Strategies to prevent type 2 diabetes. Curr Med Res Opin. 2005;21(7):1107-14.

9) United Kingdom Prospective Diabetes Study Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet. 1998;352(9131):837-853.

10) UK Prospective Diabetes Study Group. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes: UKPDS 38. BMJ. 1998;317(7160):703-713.

11) Irons BK, Seifert CF, Horton NA. Quality of care of a pharmacist-managed diabetes service compared to usual care in an indigent clinic. Diabetes Technol Ther. 2008;10(3):220-6.

12) Hegde PK, Rao HA, Rao PN. A review on insulin plant (Costus igneus Nak). Pharmacogn Rev. 2014 Jan;8(15):67-72. doi:10.4103/0973-7847.125536. PMID: 24600198; PMCID: PMC393203.

13) Diaz-Valencia PA, Bougneres P, Valleron AJ. Global epidemiology of type 1 diabetes in young adults and adults: A systematic review. BMC Public Health. 2015;15:255.

14) International Diabetes Federation. IDF Diabetes Atlas. IDF; 2015.

Available from: http://www .diabetesatlas.org/component /attachments/?task=download&id= 116

15) Gonzalez EL, Johansson S, Wallander MA, Rodriguez LA. Trends in the prevalence and incidence of diabetes in the UK: 1996-2005. J Epidemiol Community Health. 2009;63(4):332-6.

16) Neil HA, Gatling W, Mather HM, Thompson AV, Thorogood M, Fowler GH, Hill RD, Mann Jl. The Oxford Community Diabetes Study: Evidence for an increase in the prevalence of known diabetes in Great Britain. Diabet Med. 1987;4:539-543.

17) Kaku K. Pathophysiology of type 2 diabetes and its treatment policy. JMAJ. 2010;53(1):41-6.

18) Patidar D. Pharmacology. 2nd ed. Meerut: Shree Sai Prakashan; 2011. p. 113-4.

19) AL Homsi MF, Lukic PAL. An update on the pathogenesis of diabetes mellitus. Department of Pathology & Medical Microbiology (Immunology Unit), Faculty of Medicine & Health Sciences, UAE University; 1992. Al Ain, United Arab Emirates.

20) Raju SM, Raju B. Illustrated medical biochemistry. 2nd ed. New Delhi: Jaypee Brothers Medical Publishers; 2010. p. 645.

21) EURODIAB ACE Study Group. Variation and trends in incidence of childhood diabetes in Europe. Lancet. 2000;355(9207):873-6.

22) Blood A, Hayes TM, Gamble DR. Register of children newly diagnosed with diabetes. BMJ. 1975;3(5988):580-3.

23) Kaprio J, Tuomilehto J, Koskenvuo M, Romanov K, Reunanen A, Eriksson J, Stengård J, Kesäniemi YA. Concordance for type 1 (insulin-dependent) and type 2 (non-insulin-dependent) diabetes mellitus in a population-based cohort of twins in Finland. Diabetologia. 1992;35(11):1060-7.

24) Holt GI. Diagnosis, epidemiology, and pathogenesis of diabetes mellitus: an update for psychiatrists. Br J Psychiatry. 2004;184(6):555-63.

25) Harris MI, Klein R, Welborn TA, Knuiman ML. Onset of NIDDM occurs at least 4-7 years before clinical diagnosis. Diabetes Care. 1992;15(7):815-9.

26) American Diabetes Association. Standards of medical care in diabetes-2011. Diabetes Care. 2011;34(Suppl 1):S11-61.

27) Cox EM, Edelman D. Tests for screening and diagnosis of type 2 diabetes. Clin Diabetes. 2009;27(4):132-8.

28) Cryer PE. Minireview: Glucagon in the pathogenesis of hypoglycemia and hyperglycemia in diabetes. Endocrinology. 2012;153(3):1039-48.

29) Forbes JM, Cooper ME. Mechanisms of diabetic complications. Physiol Rev. 2013;93(1):137-88.

30) MSD Manuals. Available from: https://www.msdmanuals.com

31) 1. GDC Live. Available from: https://www.gdclive.com

2. Go Online Test. Available from: https://www.goconlinetest.in