IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)
An International Open Access, Peer-reviewed, Refereed Journal

A Comprehensive Review Of Diabetes Mellitus And Clinical & Non-Clinical Management

[1]Kriti Sood, [2]Vandana Pathak, [3]Nishi Jain, [4]Sagar Kumar*
[1]Associate Professor, [2]Lecturer, [3]Assistant Professor, [4]Assistant Professor
[1]Guru Ramdas Khalsa Science and Technology Pharmacy, Jabalpur, Madhya Pradesh, India
[2]Sir Aatma Ram Institute of Pharmacy and Technology, Jabalpur, Madhya Pradesh, India - 482020
[3]Lakshmi Narain College of Pharmacy, Kalchuri, Nagar Raisen Road, Bhopal, Madhya Pradesh, India - 462022
[4]Shri Ram College of Pharmacy, Parikrama Marg, Muzaffarnagar, Uttar Pradesh – 251001

Abstract: Diabetes mellitus stands as one of the most prevalent non-communicable maladies globally. India is confronted with an array of challenges in the management of diabetes, encompassing a mounting incidence of this ailment across urban and rural domains, a prevailing dearth of public awareness regarding the disorder, inadequate medical infrastructure, elevated treatment costs, suboptimal glycemic control, and a surge in the occurrence of diabetic complications. Subcutaneous injections serve as a modality for administering insulin therapy in diabetes management, allowing for up to four administrations daily. Nonetheless, the long-term employment of insulin therapy is beset by detriments to patient outcomes due to challenges associated with patient adherence and the intrusive nature of its delivery mechanism. While type 1 diabetes is observing an upward trajectory, the focal point of the diabetes epidemic is primarily anchored in type 2 diabetes mellitus, which represents over 90% of all diabetes cases. Type 2 diabetes, a perilous and pervading chronic ailment, emerges from a multifaceted interplay of genetic predisposition, environmental factors, and supplementary risk elements, including obesity and sedentary lifestyle behaviors.

Index Terms – Diabetes mellitus; Classification; Diagnosis; Cause; Treatment.

1. Introduction

Diabetes mellitus, a chronic metabolic disorder affecting the metabolism of proteins, lipids, and carbohydrates, is characterized by an intrinsic deficiency or suboptimal secretion of insulin, resulting in compromised carbohydrate (glucose) utilization [1]. Foremost among its diagnostic attributes is an inadequate insulin secretory response, which can stem from either an insufficient supply of insulin or, less commonly, impaired insulin functionality (insulin resistance). Termed colloquially as "sugar," diabetes mellitus (DM) constitutes a prevalent endocrine malady. The International Diabetes Federation (IDF) reports a current diabetic population of 40.9 million in India, with projections indicating a surge to 69.9 million by 2025 [2].

Pancreatic function involves the secretion of two pivotal hormones, insulin and glucagon. These hormones emanate from distinct cellular populations within the islets of Langerhans: insulin is produced by beta (ß) cells, whereas glucagon is secreted by alpha cells within the same organelles. The orchestrated action of insulin is characterized by its facilitation of glycogenesis and glucose transportation to critical metabolic sites encompassing muscles, liver, and adipose tissue. Consequently, insulin exerts a glucose-lowering effect. Conversely, the regulatory role of alpha cells is manifested in the generation of glucagon, which orchestrates blood glucose management by fostering glycogenolysis, thus elevating blood glucose levels. Notably, certain physiological components, such as erythrocytes and neural tissue, exhibit a distinctive glucose utilization pattern wherein insulin is not requisite for glucose uptake and utilization [3].

In conjunction with an elevated susceptibility to obesity, metabolic derangements, and cardiovascular pathologies, it is noteworthy that neonates, subsequent to parturition, also manifest an augmented predisposition to malignancies. The etiological spectrum of diabetes mellitus predominantly comprises type II diabetes, accounting for a substantial 80% to 90% of total cases. Geographical variance further imparts heterogeneity to the clinical trajectory, encompassing both the gravity of the conditions and the composite burden of morbidity and mortality. Furthermore, a discernibly marginal reduction in mortality risk is observed among individuals with diabetes who engage in modest physical activity in comparison to their sedentary counterparts. It is now well-established that a distinct genetic underpinning is pivotal for the manifestation of such propensities. Evidently, the escalating prevalence of diabetes and other noncommunicable ailments in the WHO African Region states emerges as a formidable impediment to health progress and the advancement of economies [4]. For illustrative content, please refer to Figure 1.

Diabetes is typified by aberrant insulin production or secretion, as exemplified in Type 1 diabetes mellitus (IDDM) and pancreatic duct stenosis. Alternatively, it may arise from the emergence of insulin resistance or irregular insulin synthesis, exemplified in Type 2 diabetes (NIDDM) and certain secondary diabetic states

2. CLASSIFICATION OF DIABETES MELLITUS

The initial delineation of diabetes mellitus categories was introduced by the World Health Organization (WHO) in 1980, subsequently revised in 1985. Central to our discourse is the primary or idiopathic variant of diabetes mellitus, which claims pre-eminence due to its ubiquity and clinical significance. This primary variant necessitates clear demarcation from secondary diabetes mellitus, a realm encompassing instances of hyperglycaemia where the pathogenic trajectory involves well-defined causative factors. Etiological factors encompass the incitement of pancreatic islet destruction through inflammatory pancreatic disorders, surgical interventions, neoplasms, specific pharmacological agents, iron accumulation (hemochromatosis), and targeted acquired or genetic endocrinopathies [5].

The taxonomy of diabetes mellitus encompasses a spectrum of hyperglycaemic manifestations, in tandem with clinical phases and etiological subtypes. Categorizing an individual's diabetes type often hinges upon the prevailing conditions that precipitated their diagnosis, a nuance that frequently eludes facile classification for many affected individuals. Hyperglycaemia is discerned as an overarching feature transcending various malady, of which primary diabetes mellitus is but one [6].

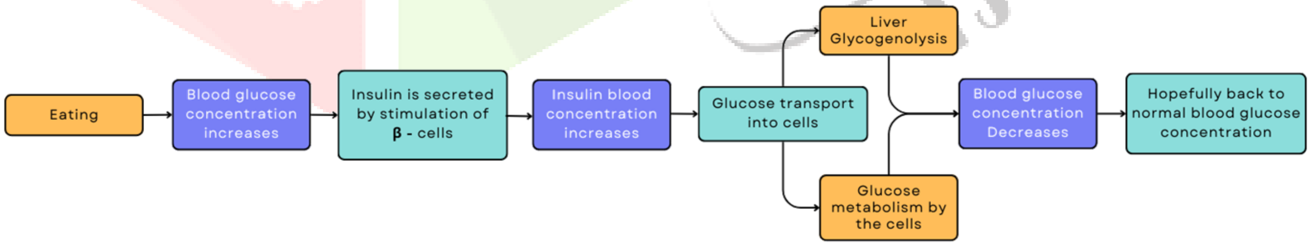

Fig 1: Glucose Metabolism of dietary product

The contemporary classification schema of diabetes mellitus (DM) comprises distinct stages delineating varying degrees of hyperglycaemia across individual patients, encompassing diverse pathological trajectories that may culminate in the manifestation of diabetes mellitus.

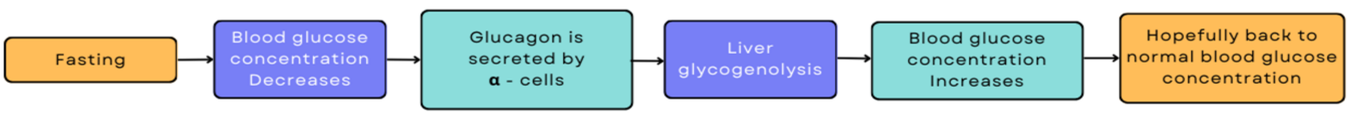

Fig 2: Nominal response towards fasting

The contemporary nomenclatural framework that delineates four discrete categories of diabetes mellitus supersedes the archaic classifications of noninsulin-dependent diabetes mellitus (NIDDM) and insulin-dependent diabetes mellitus (IDDM), originally introduced by the WHO in 80’s. This evolution in terminology was documented by the WHO Expert Committee in 1999 [7]. Subsequently, these designations were incorporated into the ICD-10 in 1992 and the International Nomenclature of Diseases in 1991. Consequently, the current taxonomy of diabetes mellitus is structured in the following manner:

1. Insulin Dependent Diabetes Mellitus (IDDM or Type 1 DM)

Formerly categorized under the terms "ketosis-prone" or "juvenile-onset" diabetes, this specific manifestation of diabetes mellitus has undergone reclassification as autoimmune diabetes. Individuals affected by this subtype might concurrently seek medical attention for concomitant autoimmune disorders, including but not limited to Hashimoto's thyroiditis, Graves' disease, and Addison's disease. The nomenclature of IDDM, more commonly recognized as type I diabetes, predominantly affects paediatric and young adult populations. Its onset is frequently abrupt and potentially fatal. The pathophysiological underpinnings of type 1 diabetes involve the presence of antibodies targeting insulin, islet cells, or glutamic acid decarboxylase, signifying the autoimmune processes that culminate in β-cell demise [8]. Diabetes type 1 arises from the demise of beta cells, resulting in an absolute insulin deficiency. The pace of β-cell deterioration exhibits significant interindividual variability, manifesting as either rapid or gradual degeneration. The consequential loss of pancreatic ß-islet cells leads to pronounced insulin insufficiency or absence, necessitating exogenous insulin administration. A notable observation is that upon the initial identification of fasting diabetic hyperglycaemia, 85-90% of individuals afflicted with Type 1 diabetes mellitus exhibit immunological markers of damage, including autoantibodies targeting glutamic acid decarboxylase (GAD), islet cell autoantibodies, and/or autoantibodies against insulin. Despite a prevailing consensus regarding an autoimmune etiology involving beta islet cell-targeting autoantibodies, the precise etiological origins of diabetes mellitus remain elusive [9].

2. Non-Insulin Dependent Diabetes Mellitus (NIDDM or Type 2 DM)

NIDDM, synonymous with adult-onset diabetes, is characterized by the interplay of insulin resistance and progressive deterioration in insulin secretion mechanisms, leading to compromised insulin functionality (American Diabetes Association, 2014). Impaired insulin action is a hallmark of this diabetic subtype. Notably, the primary drivers of morbidity and mortality in diabetes arise from chronic vascular, renal, ocular, and neural complications. The etiological underpinnings of type 2 diabetes are intricate, encompassing multifaceted factors such as obesity, sedentary lifestyle, advancing age (predominantly affecting middle-aged and elderly individuals), and genetic predisposition (Ross and Wilson 2010). Consequently, this cohort is predisposed to a heightened vulnerability to both macrovascular and microvascular complications [10].

3. Gestational Diabetes Mellitus (GDM)

GDM denotes the emergence of glucose intolerance, either newly manifesting or detected, within the context of pregnancy. The terminology encompasses women who encounter the onset of Type 1 DM during gestation and those who unveil latent, asymptomatic Type 2 DM during this period. GDM is a provisional descriptor for pregnancy-associated hyperglycaemia that lacks definitive diabetic classification. It is pertinent to note that offspring born to mothers afflicted with gestational diabetes mellitus (GDM) face an escalated susceptibility to obesity and eventual onset of type 2 diabetes in their later years. This predisposition is linked to the ramifications of intrauterine exposure to hyperglycaemic conditions. Importantly, gestational diabetes mellitus may materialize during gestation and potentially resolve postpartum [11].

4. Other Specific Type (Monogenic Types)

Hepatocyte nuclear factor (HNF)-1a, a pivotal hepatic transcription factor, undergoes mutational perturbations located on chromosome 12, characterizing the most prevalent manifestation of monogenic diabetes forms. Previously designated as beta cell genetic deficiencies, these anomalies culminate in aberrations of pancreatic function. The occurrence of hyperglycaemia at an early age, often preceding 25 years, is a shared attribute among diverse diabetes subtypes. Individuals afflicted with exocrine pancreatic pathologies like pancreatitis or cystic fibrosis, those grappling with endocrine disorders such as acromegaly, and those encountering pancreatic impairment elicited by pharmacological agents, chemical exposures, or infections, are encompassed within the scope of maturity-onset diabetes of the young (MODY) or youth-onset diabetes, often correlated with insufficiencies in insulin action [12]. Select pharmacotherapies find utility in contexts such as organ transplantation or concurrent HIV/AIDS management. A subset of familial cases evinces hereditary defects impairing proinsulin conversion into insulin, inherited in an autosomal dominant manner, albeit representing a minority fraction, constituting less than 10% of diabetes mellitus occurrences.

SOME COMMON SIGN AND SYMPTOMS

Cells afflicted with diabetes mellitus encounter a state of compromised metabolic capacity that leads to impaired glucose utilization, culminating in a state akin to nutritional deprivation. Over the long term, the ramifications encompass a gradual evolution of retinopathy, precipitating the risk of vision impairment and even blindness, nephropathy, an entity fraught with the potential for renal insufficiency, and neuropathy, underpinning the heightened susceptibility to foot ulcers, Charcot joints, and various neuropathic and sexual dysfunction manifestations. Evidently, diabetes mellitus engenders an escalated predisposition to diverse morbidities [13].

Numerous manifestations ensue due to distinct mechanistic pathways:

i. The orchestration of glucose synthesis from amino acids and endogenous proteins begets muscle wasting, tissue degeneration, and a concurrent surge in circulating glucose levels.
ii. Catabolic processes involving adipose tissue are triggered, precipitating the release of stored energy and an excessive accumulation of ketone bodies [14].

DIABETES MELLITUS: ETIOLOGY

The Greek term "aetiologia" serves as the etymological precursor for the nomenclature "aetiology." Consequently, aetiology pertains to the scientific discipline dedicated to elucidating the underlying causative factors and origins of diseases. This encompassing domain encompasses various aspects, notably including:

1. The etiology attributed to the juvenile-onset (insulin-dependent) subtype presently leans towards an autoimmune etiological framework.
2. Viral agents, such as coxsackie-B, are recognized as potential contributors to the etiology of diabetes.
3. Pertinent studies have established a morphological remodelling of islet-cell architecture in response to viral infections such as mumps and rubella.
4. The role of genetics in the diabetes pathogenesis is a topic of ongoing debate. It is plausible that an individual's genetic predisposition could render their pancreas more susceptible to the aforementioned viral influences due to inherent hereditary attributes [15].

Causes of Diabetes Mellitus

Disturbances or anomalies within the gluco-receptor function of ß cells may engender altered responses to elevated glucose concentrations or precipitate a relative deficiency in ß cells. In either scenario, a resultant diminution in insulin secretion ensues, potentially culminating in ß cell insufficiency. Furthermore, the ramifications of hyperglycaemia exert direct influences on neuronal metabolism, intertwining with the proposition of primary involvement in the etiopathogenesis of microvascular maladies, thus giving rise to the concept of neural hypoxia.

1. A reduction in the quantity of insulin receptors coupled with the phenomenon of 'downregulation' in insulin receptors leads to diminished sensitivity of peripheral tissues to insulin. Concurrently, instances of abdominal obesity, dyslipidaemia, and hyperinsulinemia coexist even in the presence of normoglycemia. This convergence contributes to a discernible insulin resistance, particularly evident within the liver, muscles, and adipose tissue. The manifestation of angiopathy has been linked to the context of hyperinsulinemia.
2. An excess of hyperglycaemic hormones such as glucagon, along with lipid accumulation, engenders a relative insufficiency of insulin, thereby casting ß cells in a state of insufficient response. This dynamic is underpinned by two theoretical constructs, elucidating the perturbations in nitric oxide metabolism that subsequently evoke alterations in perineural blood perfusion, culminating in nerve impairments.

3. The spectrum of less common diabetes mellitus types, denoted as type 3, encompasses entities such as "maturity onset diabetes of young" (MODY), diverse endocrinopathies, cases stemming from pancreatectomy, and GDM, all traceable to specific genetic aberrations.
4. The intricate milieu of diabetes mellitus etiology may emanate from a perturbed receptor equilibrium. Specialized receptor examples encompass beta3 (ß3) adrenergic receptor, peroxisome proliferator-activated receptor (PPAR), glucagon-like peptide-1 (GLP-1) receptor, and select enzymes such as dipeptidyl peptidase IV and glycosidase enzyme.
5. Ongoing research endeavours in diabetic neuropathy are prominently centred around key themes including protein kinase C, the polyol pathway, advanced glycation-end products, and oxidative stress, [16].

DIAGNOSIS OF DIABETES MELLITUS

It is imperative not to predicate the diagnosis of diabetes in an asymptomatic individual solely on the basis of an isolated aberrant blood glucose measurement. The attending clinician must exercise meticulous caution, ensuring unequivocal confirmation of diabetes status due to the profound and enduring implications for the patient's well-being. A comprehensive array of diagnostic modalities, encompassing urine glucose assessment, blood glucose levels, glucose tolerance testing, determination of renal glucose threshold, assessment of impaired glucose tolerance, evaluation of elevated glucose tolerance, identification of renal glycosuria, profiling of glucose resistance curves, cortisone-stimulated glucose resistance evaluation, intravenous glucose resistance examination, and oral glucose resistance testing, collectively serve as diagnostic tools within the ambit of diabetes mellitus assessment.

CLINICAL AND NON-CLINICAL MANAGEMENT OF DIABETES MELLITUS

Aside from the imperative of eradicating the instigating factor, the therapeutic approach encompasses the administration of elevated dosages of regular insulin. Following successful intervention, the requisite for insulin subsides to baseline levels. The objectives intrinsic to the management of diabetes mellitus encompass the following facets:

1. Endeavouring to restore the anomalous metabolic milieu of the diabetic patient to a state of normalcy, while concurrently upholding considerations of comfort and safety.
2. Mitigating or decelerating the progression of both immediate and enduring perils associated with the ailment.
3. Equipping the patient with the needed motivation, essential knowledge and resources essential to autonomously undertake enlightened self-care practices [17].

A. DIABETES MELLITUS: TYPES OF THERAPY

1. Stem cell therapy

Monocytes and macrophages are believed to exert a significant influence within the context of chronic inflammatory conditions and insulin resistance among individuals with T2 DM, as suggested by scientific research. A leading-edge therapeutic approach, termed stem cell educator treatment, endeavours to mitigate or ameliorate immunological dysfunctions. This method entails the extraction of blood from the patient's circulatory system using a closed-loop system, Following the segregation of lymphocytes from the entire blood sample, these lymphocytes are subjected to co-cultivation alongside adherent cord blood-derived multipotent stem cells (CB-SCs). Subsequently, the educated lymphocytes are reintroduced into the patient's circulatory system, with a deliberate exclusion of the reintroduction of the CB-SCs.

2. Antioxidant therapy

In the context of managing oxidative stress within individuals afflicted with type 2 diabetes mellitus (T2DM), an array of antioxidants has been utilized, encompassing vitamins, supplementary agents, bioactive constituents sourced from botanical origins, and pharmaceuticals endowed with antioxidant attributes.

Notable among these are vitamin C, vitamin E, and beta-carotene, which stand as efficacious compounds in countering oxidative stress and its associated repercussions. The integration of antioxidants plays a pivotal role in mitigating the susceptibility to DM onset and its ensuing complications.

3. Anti-inflammatory treatment

The alterations observed underscore the pivotal role of inflammation in the progression of type 2 diabetes mellitus (T2DM) and its attendant sequelae. Tissues such as adipose tissue, hepatic tissue, circulating leukocytes, the vasculature, and pancreatic islets manifest discernible susceptibility to the impacts of T2DM, as are the numbers and states of activation of various leukocyte populations, enhanced apoptosis, and tissue fibrosis. Immunomodulatory medications are offered.

B. DIETARY MANAGEMENT

Both individuals with diabetes and those without the condition should adhere to appropriate dietary management, which encompasses ensuring an optimal caloric intake that meets the required energy needs, given below:

- Emphasize complex carbohydrates with low glycaemic index (GI) to mitigate postprandial hyperglycaemia.
- Distribute carbohydrate intake across meals to optimize glycaemic control throughout the day.
- Monitor carbohydrate intake to avoid abrupt glucose spikes.
- Advocate a diet rich in dietary fibre from sources such as whole grains, legumes, vegetables, and fruits.
- Fiber aids in glycaemic control by slowing carbohydrate absorption and promoting satiety.
- Encourage consumption of unsaturated fats, encompassing both monounsaturated and polyunsaturated fat sources.
- Omega-3 fatty acids from sources like fatty fish have potential benefits for cardiovascular health in diabetes.
- opt for lean protein sources, including poultry, fish, legumes, and low-fat dairy products.
- Adequate protein intake supports muscle preservation and satiety.
- Promote portion awareness to prevent overconsumption and stabilize blood glucose levels.
- Utilize methods like plate method or carbohydrate counting for effective portion control.
- Encourage regular meal timings to prevent erratic blood glucose fluctuations.
- Consider balanced spacing of meals and snacks to avoid prolonged periods of fasting.
- Limit added sugars and sugary beverages in the diet.
- Explore artificial sweeteners cautiously, considering their impact on glycaemic response.
- Advocate moderate alcohol consumption, if desired, with consideration of its potential effects on blood glucose and interactions with medications.
- Stress the importance of staying hydrated, as dehydration can affect blood glucose levels.
- Tailor dietary recommendations to individual needs, preferences, and cultural considerations.
- Collaborate with registered dietitians to develop personalized meal plans.
- Educate individuals about interpreting nutritional labels to make informed food choices.
- Regularly monitor blood glucose levels to gauge the impact of dietary choices.
- Address weight management, if necessary, as achieving and maintaining a healthy weight can enhance glycaemic control.
- Emphasize collaboration among healthcare professionals, including physicians, dietitians, and diabetes educators, for comprehensive dietary management.
- Provide education about the role of nutrition in diabetes management to empower individuals to make informed dietary decisions.

- Promote dietary changes that are sustainable over the long term to ensure ongoing diabetes management.

C. NEWER INSULIN DELIVERY DEVICES

Newer insulin delivery devices signify a paradigm shift in diabetes management, offering enhanced precision, flexibility, and patient-centred care. As these technologies continue to evolve, clinicians and researchers must collaborate to maximize their potential for improving glycaemic control, reducing complications, and increasing the overall QoL for individuals living with diabetes mellitus.

1. **Insulin Pumps:**
   Insulin pumps represent a sophisticated wearable technology that delivers a continuous subcutaneous infusion of rapid-acting insulin. These devices mimic physiological insulin release by providing basal rates along with bolus doses during meals. Advanced features include integrated continuous glucose monitoring (CGM), automated insulin suspension during hypoglycaemia, and customizable insulin delivery profiles. Insulin pumps offer tighter glycaemic control, reduced hypoglycaemic events, and improved quality of life for eligible patients.

2. **Closed-Loop Systems:**
   Emerging as a groundbreaking innovation, closed-loop systems combine insulin pumps with real-time CGM data and sophisticated algorithms to automate insulin delivery based on individual glucose levels. Also known as artificial pancreas systems, these devices offer precise glucose regulation, minimizing hyperglycaemia and hypoglycaemia without constant manual intervention. Closed-loop systems are transforming diabetes management, particularly for individuals with type 1 diabetes, by maintaining glucose within target ranges effortlessly.

3. **Inhaled Insulin:**
   Inhaled insulin devices provide an alternative route of insulin delivery, enabling rapid absorption through the pulmonary system. These devices offer a needle-free option for mealtime insulin dosing, appealing to individuals with needle phobia. Inhaled insulin devices demonstrate rapid onset of action and flexibility in dosing, though proper inhalation technique and patient education are crucial for optimal outcomes.

4. **Implantable Insulin Delivery**:
   Implantable insulin delivery devices are a burgeoning area of research, aiming to provide sustained insulin release within the body. Encapsulated cell technologies and bioengineered devices hold the potential to achieve prolonged glycaemic control without external interventions. Although still in experimental stages, implantable insulin delivery systems present an exciting avenue for future diabetes therapy.

D. ORAL HYPOGLYCAEMIC OR ANTIDIABETIC AGENTS

Oral hypoglycaemic agents, also known as antidiabetic agents, constitute a cornerstone in the management of diabetes mellitus. These pharmaceutical agents play a pivotal role in regulating blood glucose levels by various mechanisms. Sulfonylureas exert their physiological effect by eliciting the secretion of insulin from pancreatic β-cells, thereby facilitating an augmented uptake of glucose. Metformin curbs hepatic gluconeogenesis, concurrently enhancing peripheral insulin sensitivity. Thiazolidinediones exert their effects by improving insulin responsiveness in adipose, muscle, and liver tissues. SGLT-2 inhibitors and DPP-4 inhibitors, modulate incretin hormones and renal glucose reabsorption, respectively.

The utilization of glucagon-like peptide-1 (GLP-1) receptor agonists further contribute to glycaemic control by promoting insulin secretion and suppressing glucagon release. As pivotal components of comprehensive diabetes management, these agents are integrated into treatment protocols based on individual patient profiles, disease progression, and potential side effects. While offering effective blood glucose regulation, it is crucial to judiciously select and monitor these agents to achieve optimal therapeutic outcomes while mitigating potential risks, aligning with the personalized approach to diabetes care that is becoming increasingly central in contemporary medical practice. [18].

Importance of Oral Hypoglycaemic Agents

DM can be construed as a contemporary malady exerting substantial impact on morbidity, mortality, and demographic susceptibilities. Cushing's syndrome is precipitated by recurrent glucocorticoid exposure, manifesting a spectrum of clinical manifestations encompassing central obesity, proximal muscular debility, hirsutism, neurophysiological perturbations, macrovascular sequelae, autonomic neuropathies, gastrointestinal disturbances, dental afflictions, and related anomalies.

CONCLUSION

In summation, diabetes mellitus, especially type 2 DM, has appeared as a prominent non-communicable ailment with global implications. India, in particular, confronts a constellation of challenges in efficaciously combating this escalating epidemic. The nation grapples with escalating diabetes prevalence across urban and rural landscapes, compounded by deficient public awareness, inadequate medical infrastructure, elevated treatment expenses, suboptimal glycaemic management, and a surging tide of diabetic complications.

While subcutaneous insulin administration exhibits efficacy, its sustained utilization is marred by adherence complexities and the intrusive nature of the intervention. Furthermore, the ascendant incidence of type 1 DM exacerbates the burden; however, type 2 DM overwhelmingly dominates the disease landscape, propelling the epidemic's momentum. This intricate malady is a product of a multifaceted interplay between genetic predisposition, environmental determinants, and additional risk facets such as obesity and sedentary comportment.

Addressing India's diabetes scourge necessitates a holistic paradigm, enjoining public health awareness initiatives, augmented medical accessibility, economically viable therapies, and lifestyle interventions. Collaborative engagement among researchers, healthcare stakeholders, policy formulators, and society at large is imperative for propagating prevention, early detection, and optimal diabetes control, thereby ameliorating the weight of this formidable and pervasive chronic affliction. Through a concerted, multifaceted endeavour, India can strive for improved patient outcomes and a healthier trajectory for its populace, surmounting the diabetes challenge in stride

REFERENCES

[1] Zimmet PZ, (1991). Kelly West Lecture-challenges in diabetes epidemiology - from West to the rest: Diabetes Care 15(2),232-52. https://doi.org/10.2337/diacare.15.2.232

[2] Kumar CR, (1992) Basic Pathology, Prism PVT. Limited Bangalore, 5th edition, 569-587.

[3] Mukherjee PS, Ghosh S, Mukhopadhyay P, Das K, Das DK, Sarkar P, Bhattacharya D, Mazumdar S, Chatterjee K, (2020). A diabetes perception study among rural and urban individuals of West Bengal, India: are we ready for the pandemic?: International Journal of Diabetes in Developing Countries, 40,612-618.

[4] Chia YC, Ching SM, Chew BN, Devaraj NK, Siti Suhaila MY, Tay CL, Kang PS, Verna Lee KM, Kong SZ, Teoh SW, Nurjasmine AJ, (2019). May measurement month 2017 blood pressure screening: findings from Malaysia - SouthEast Asia and Australasia: European Heart Journal Supplements, 21,77-79.

[5] Atkinson MA, Eisenbarth GS, (2001) Type 1 diabetes new perspectives on disease pathogenesis and treatment: Lancet, 358,221-229.

[6] Geldsetzer P, Manne-Goehler J, Theilmann M, Davies JI, Awasthi A,Vollmer S, (2018). Diabetes and hypertension in India: a nationally representative study of 1.3 million adults: JAMA Internal Medicine, 178(3),363-372.

[7] Bansal V, Kalita J, Misra U (2006). Diabetic neuropathy: Journal of Postgraduate Medicine, 82(964),95-100.

[8] Betterle C, Zanette F, Pedini B, Presotto F, Rapp LB, Monsciotti CM (1983). Clinical and subclinical organ– specific autoimmune manifestations in type 1 (insulin– dependent) diabetic patients and their first-degreerelatives: Diabetologia, 26,431-36.

[9] Verge CF, Gianani R, Kawasaki E, Yu L, Pietropaolo M, Jackson RA (1996). Predicting type I diabetes in first– degree relatives using a combination of insulin, GAD, and ICA512bdc/IA-2autoantibodies: Diabetes, 45,926-33.

[10] Zimmet PZ, Tuomi T, Mackay R, Rowley MJ, Knowles W, Cohen M (1994). Latent autoimmune diabetes mellitus in adults (LADA): the role of antibodies to glutamic acid decarboxylase in diagnosis and prediction of insulin dependency: Diabetic Medicine 11,299-303.

[11] DeFronzo RA, Bonadonna RC, Ferrannini E, Zimmet P (1997). Pathogenesis of NIDDM, International Textbook of Diabetes Mellitus, 635-712.

[12] American Diabetes Association. (2014, April 27). Diagnosis and classification of diabetes mellitus.

[13] Mooy JM, Grootenhuis PA, de Vries H, Valkenburg HA, Bouter LM, Kostense PJ (1995). Prevalence and determinants of glucose intolerance in a Dutch population: Diabetes Care, 18,1270-1273.

[14] Lillioja S, Mott DM, Spraul M, Ferraro R, Foley JE, Ravussin E (1993). Insulin resistance and insulin secretory dysfunction as precursors of non–insulin–dependent diabetes: The New England Journal of Medicine, 329,1988-1992.

[15] Harris MI, (1993). Undiagnosed NIDDM, clinical and public health issues: Diabetes Care, 16,642-52,

[16] Jun SK, Yoon YW, (2002). A new look at viruses in Type 1 diabetes: Diabetes/Metabolism Research and Reviews, 19,8-31.

[17] Boney CM, Verma A, Tucker R, Vohr BR (2005). Metabolic syndrome in childhood: association with birth weight, maternal obesity, and gestational diabetes mellitus: Paediatrics, 45(4),115-118.