International Journal of Public Health and Epidemiology ISSN 2326-7291 Vol. 10 (3), pp. 009, August, 2021.
Available online at www.internationalscholarsjournals.com © International Scholars Journals

Review Article

A review on epidemiology, diagnosis, management and treatment of diabetes mellitus

Kamal Singh Bani[1*], Khushboo Bhardwaj[2] and Stuti Tripathi[3]

[1]Department of Pharmaceutics, B.B.S College of Pharmacy, Utter Pradesh, India.

[2]Department of Pharmaceutics, Dr K.N. Modi Institute of Pharmaceutical Education and Research, Utter Pradesh, India.

[3]Department of Pharmaceutics, Sardar Patel College of Pharmacy, Utter Pradesh, India.

Accepted 05 August, 2021

Diabetes mellitus (DM), a condition caused by metabolic abnormalities, is the most common worldwide. Sometimes known as diabetes, in which a person's blood sugar levels are abnormally high, either because the body does not make enough insulin or because the insulin that is produced does not reach the cells. Polyuria (frequent urine), polydipsia (increased thirst), and polyphagia are all common indicators of elevated blood sugar (increased hunger). Type 1 diabetes, also known as insulin-dependent diabetes mellitus (IDDM), is characterized by the body's inability to manufacture insulin, necessitating the use of insulin injections or the use of an insulin pump. It's also known as "juvenile diabetes." Insulin resistance, also known as non-insulin-dependent diabetes mellitus (NIDDM), is a disease in which cells fail to utilize insulin properly, either with or without an absolute insulin deficit. This condition was originally known as "adult-onset diabetes." The third major kind is gestational diabetes, which happens when women who have never had diabetes have a high blood glucose level during pregnancy. It may occur before the onset of type 2 diabetes. Insulin and oral hypoglycemic medications are currently available medicines for the treatment of diabetes mellitus. These medications work by raising insulin production from the pancreas or decreasing plasma glucose levels by enhancing glucose absorption and decreasing gluconeogenesis. These present medications, on the other hand, do not restore normal glucose homeostasis for a longer period of time and are not without side effects such as hypoglycemia, kidney illness, GIT difficulties, hepatotoxicity, heart risk concerns, insulinoma, and they must be used for the rest of one's life. Various herbal medications have also been shown to be successful in the treatment of diabetes due to their beneficial ingredients. As a result, the current analysis attempts to concentrate on the physiological elements of diabetes, its consequences, management goals, and synthetic and herbal diabetes treatment.

Key words: Diabetes mellitus (DM), types of DM, diagnosis, management, synthetic treatment, herbal treatment.

INTRODUCTION

Diabetes Mellitus (DM) is the most common endocrine illness, affecting over 100 million individuals globally (6 percent population). It is caused by a lack of or inadequate insulin synthesis by the pancreas, which causes in an increase or reduction in blood glucose concentrations. It has been discovered to harm a variety of biological systems, including blood vessels, eyes, kidneys, heart, and nerves (Sachin et al., 2009). Insulin-dependent diabetes mellitus (IDDM, Type I) and non-insulin-dependent diabetes mellitus (NIDM, Type II) are the two kinds of diabetes mellitus (NIDDM, Type II). Type I diabetes is an autoimmune illness marked by a localized inflammatory response in and around islets, followed by the selective death of insulin-secreting cells, whereas Type II diabetes is marked by periphery inflammation (Bastaki, 2005). Many consequences are associated with diabetes, including cardiovascular disease, peripheral vascular disease, stroke, neuropathy, renal failure, retinopathy, blindness, and amputations. Medicinal herbs and their bioactive ingredients can be utilised to treat diabetes mellitus all over the world, particularly in regions where access to traditional anti-DM drugs is limited. (Mohammad et al., 2009). Drugs are primarily used to prolong life and relieve symptoms. Secondary goals include preventing long-term diabetic problems and increasing longevity by removing various risk factors. Insulin replacement therapy is the mainstay for people with type 1 diabetes, but diet and lifestyle changes are the cornerstone for type 2 diabetes

*Corresponding author. Kamal Singh Bani, Tel: +918287801503,
E-mail: kamalbani94@gmail.com.

treatments and control (Nandakumar et al., 2007). For the treatment of diabetes, hypoglycemic drugs such as biguanides and sulfonylureas are available. However, none of these drugs are optimal because of their harsh side effects, and continuous usage might lead to a reduction in responsiveness (Dixit et al., 1985). The biggest drawback of currently available medications is that they must be used for the rest of one's life and cause adverse effects (Eshrat et al., 2003). Various experimental approaches for screening plant anti-diabetic properties are also available (Grover N et al., 2011). As a result, the current study aims to learn more about diabetes mellitus, its clinical manifestations, epidemiological data, consequences, and contemporary diabetes treatment options.

Diabetes mellitus (DM) is likely one of humanity's oldest ailments. It was first written over 3000 years ago in an Egyptian text (Ahmed, 2002). The distinction between type 1 and type 2 diabetes mellitus was determined in 1936 (Defronzo, 2010). In 1988, type 2 diabetes was initially identified as a component of metabolic syndrome. (Chiniwala et al., 2011). Type 2 diabetes (also known as non-insulin dependent diabetes) is the most common type of diabetes and is characterized by hyperglycemia, insulin resistance, and insulin insufficiency. (Collier CA, 2006). Type 2 diabetes is caused by a combination of genetic, environmental, and behavioral risk factors (Dixit VP et al., 1985). People with type 2 diabetes are more susceptible to a variety of short- and long-term problems, which can lead to death. Because of the prevalence of type 2 diabetes, its insidious start, and late identification, especially in resource-poor developing countries like Africa, individuals with type 2 diabetes have a higher risk of morbidity and mortality (Eshrat, 2003).

EPIDEMIOLOGY

The use of epidemiology in the study of diabetes mellitus has yielded useful information on the disease's natural history, prevalence, incidence, morbidity, and mortality in many communities around the world. Identification of the disease's aetiology and potential preventive measures that may be used to stop or delay the spread of this disease that has reached epidemic proportions in both developed and developing countries (Fuhlendorff et al., 1998). Unfortunately, gains in outcomes for individual diabetic individuals have not translated into equal benefits in public health. The global prevalence of diabetes has continued to rise at an alarming rate. As of 2011, an estimated 366 million individuals worldwide had diabetes, with type 2 accounting for around 90% of cases (Grover et al., 2011). Every country is seeing an increase in the number of persons with type 2 diabetes, with 80 percent of those affected living in low- and middle-income countries. A review of the literature revealed that there are few statistics on the prevalence of type 2 diabetes in Africa as a whole. According to statistical trends in Africa, there has been a tremendous increase in frequency in both rural and urban settings, affecting both genders equally (Grinstein et al., 2003). According to a 2008 World Fact Book study, the prevalence of diabetes mellitus in Africa was 3.2 percent, with 40,895 people (2.0 percent) in Ethiopia (González et al., 2009).

Although T2DM is commonly diagnosed in adults, it has become much more common in children over the last two decades. T2DM now accounts for 8-45 percent of all new instances of diabetes recorded in children and adolescents, depending on the demographic investigated (Hu et al., 2001). T2DM is more common in girls than boys in the pediatric population, just as it is more common in women than men (Nandakumar et al., 2007). T2DM often occurs between the ages of 12 and 16 years, coinciding with puberty, when a physiologic condition of insulin resistance develops. T2DM develops only if poor beta-cell function is combined with other risk factors (such as obesity) in this physiologic state (Kawamori et al., 2009).

Type 2 diabetes mellitus (DM) is a chronic metabolic disorder in which prevalence has been increasing steadily all over the world. Type 2 diabetes is the predominant form of diabetes and accounts for at least 90 percent of all cases of diabetes mellitus8. The rise in prevalence is predicted to be much greater in developing than in developed countries (69 versus 20 percent).This rise in type 2 diabetes is intrinsically connected to genetics and changes to a Western lifestyle (heavy food, low physical activity) (Kim, 2008). Obesity is becoming a global epidemic, affecting not just adults but also children, teenagers, and young adults (Kaprio et al., 1992). Obesity has been linked to around 55 percent of type 2 diabetes occurrences. Between the 1960s and the 2000s, an increase in childhood obesity is thought to have contributed to an increase in type 2 diabetes in children and adolescents (Kaku, 2010). Type 2 diabetes is a complex disease caused by a mix of variables, including:

1. Genetic factors linked to insulin resistance and decreased insulin production.

2. Environmental factors such as obesity, overeating, inactivity, and stress, as well as aging (Lang et al., 2008).

Studies in monozygotic twins reveal that genetics plays a larger role in the genesis of T2DM, with concordance rates for T2DM approaching 100%. Although the genetic contribution is uncertain, multiple genes are implicated. Insulin receptor, GLUT, and Glycogen Synthase are just a few of the vulnerable genes that have been studied. Genes on chromosomes 1q, 12q, and 20q that is susceptible. Strong candidate genes for type 2 DM genetic vulnerability include the GLUT2 gene, which is expressed in liver and pancreatic beta cells, and the GLUT4 gene, which is expressed in skeletal muscle and adipocytes (Mueckler, 1990).

In 2011, 366 million individuals were expected to have diabetes; by 2030, that number will have increased to 552 million. Every country is seeing an increase in the number of persons with type 2 diabetes, with 80 percent of those affected living in low- and middle-income countries. In 2011, DM claimed the lives of 4.6 million people. By 2030, it is anticipated that 439 million individuals would have type 2 diabetes (Mohammad et al., 2009). Because of environmental and lifestyle risk factors, the incidence of type 2 diabetes varies greatly from one geographical region to the next (Patlak, 2002).

A review of the literature revealed that there are few statistics on the prevalence of type 2 diabetes in Africa as a whole. Studies looking at data patterns in Africa show a huge increase in incidence in both rural and urban areas, affecting

both men and women equally (Rosenbloom et al., 1999).

Type 2 diabetes appears to comprise the bulk of the DM burden in Africa, with type 1 diabetes accounting for fewer than 10% of DM cases. According to a 2011 report from the Centers for Disease Control and Prevention (CDC), diabetes affected approximately 25.8 million people in the United States (7.8% of the population) in 2010, with 90 percent to 95 percent of them having type 2 diabetes (Ripsin et al., 2009).

The prevalence of diabetes in adults, particularly type 2 diabetes, is expected to rise over the next two decades, with much of the increase occurring in developing countries, where the majority of patients are between the ages of 45 and 64 (Scheen, 2005). In developing countries, the latter is expected to equal or possibly exceed the former, resulting in a twofold burden as a result of the current trend of transitioning from communicable to non-communicable diseases (Shorr et al., 1996).

Lifestyle, genetics, and medical conditions

Type 2 diabetes is caused mostly by genetics and lifestyle choices (Type 2 diabetes in children and adolescents, 2000). A number of lifestyle factors have been linked to the development of type 2 diabetes. Physical inactivity, a sedentary lifestyle, cigarette smoking, and excessive alcohol use are all examples (Van et al., 1997). Obesity has been linked to around 55 percent of type 2 diabetes occurrences (Wild et al., 2004). Between the 1960s and the 2000s, an increase in childhood obesity is thought to have contributed to an increase in type 2 diabetes in children and adolescents (Willi et al., 2007). Toxins in the environment may be playing a role in the recent rise in type 2 diabetes rates. A component of various plastics, has been discovered to have a modest positive connection with the occurrence of type 2 DM in the urine (Yki-Järvinen, 2004).

Management

By altering one's lifestyle and diet. Studies have shown that a combination of maintaining a body mass index of 25 kg/m2, eating a diet high in fiber and unsaturated fat and a diet low in saturated and trans-fats and glycemic index, regular exercise, quitting smoking, and moderate alcohol consumption can significantly reduce the incidence of type 2 diabetes. It is suggested that the majority of type 2 diabetes can be avoided by changing one's lifestyle. Patients with type 2 diabetes should have their nutrition evaluated by a medical professional, and lifestyle suggestions should be matched to their physical and functional abilities.

PHARMACOLOGICAL AGENTS

Biguanides

Metformin is a biguanide that inhibits hepatic glucose synthesis, increases insulin sensitivity, promotes glucose uptake by phosphorylating GLUT-enhancer factor, increases fatty acid oxidation, and lowers glucose absorption from the gastrointestinal system (Yoon et al., 2006). Research published in 2008 shows further mechanism of action of metformin as activation of AMP-activated protein kinase, an enzyme that plays a role in the expression of hepatic gluconeogenic genes (Yach et al., 2004). Due to the concern of development of lactic acidosis, metformin should be used with caution in elderly diabetic individuals with renal impairment. It has a low incidence of hypoglycemia compared to sulfonylureas (Zimmet, 1992).

Sulfonylureas

These are generally well tolerated, but because they stimulate endogenous insulin secretion, they can cause hypoglycemia. Sulfonylureas are associated with a 36 percent increased risk of hypoglycemia in elderly patients with diabetes compared to younger patients. Glyburide is associated with higher rates of hypoglycemia than glipizide.

Meglitinides

Repaglinide and nateglinide are non-sulfonylurea secretagogues that stimulate insulin release by acting on the ATP-dependent K-channel in pancreatic beta cells, similar to sulfonylurea, however the binding location is different. Because meglitinides have a quick onset and a brief duration of action (4-6 hours), they reduce the risk of hypoglycemia. Meglitinides are used to control blood glucose levels after meals. Pre-prandial administration gives you more freedom if you miss a meal without risking hypoglycemia. Because repaglinide is mostly metabolized in the liver and excreted in very small amounts through the kidneys, dose adjustments are not required in patients with renal insufficiency, with the exception of those with end-stage repaglinide.

Thiazolidinediones

Thiazolidinedione is an insulin sensitizer that binds to the peroxisome proliferator-activated gamma transcription factor. They are the first medications to address the fundamental problem of insulin resistance in type 2 diabetes patients, whose class now mostly consists of pioglitazone, following the FDA's recent recommendation to restrict the use of rosiglitazone due to elevated cardio-vaginal risk. Pioglitazone does not cause hypoglycemia and can be administered in cases of renal impairment, making it well tolerated in the elderly. However, because of concerns about peripheral edoema, fluid retention, and fracture risk in women, its use in older persons with DM may be limited. Pioglitazone is contraindicated in elderly patients with congestive heart failure and should be avoided.

Alpha-glucosidase inhibitors

Although Acarbose, Voglibose, and Miglitol have not been widely used to treat type 2 diabetes, they are likely to be safe and effective. These medications work well for postprandial hyperglycemia and should be avoided in patients with severe renal impairment.Due to the high rates of side effects such as diarrhoea and flatulence, their use is usually restricted. The newest of the medications, Voglibose, has been proven in a study to enhance glucose tolerance in terms of delayed disease development and the number of patients who reach normoglycemia.

Incretin-based therapies

Glucagon-like peptide 1 (GLP-1) analogues are the cornerstone of incretin-based therapy, which aim to address this hitherto overlooked aspect of DM pathogenesis, resulting in long-term glycemic management and weight loss. In people with type 2 diabetes, they can be used as a monotherapy, as an adjuvant to diet and exercise, or in combination with oral

hypoglycemic medications. Exenatide, an incretin mimicker, and Liraglutide are two examples.

With the administration of GLP-1 treatments, there is no danger of hypoglycemia (unless combined with insulin secretagogues). Furthermore, new research reveals that incretin-based medicines may benefit inflammation, cardiovascular and hepatic health, sleep, and the central nervous system.

Dipeptidyl-peptidase IV inhibitors

DPP IV inhibitors inhibit dipeptidyl peptidase-4 (DPP-4), a ubiquitous enzyme that rapidly inactivates both GLP-1 and GIP, resulting in increased active levels of both hormones and improved islet function and glycemic control in type 2 diabetes. DPP-4 inhibitors are a novel family of anti-diabetic medications that are comparable to current treatments in terms of efficacy. They work as a monotherapy in people who can't control their diabetes with diet and exercise, and as an add-on therapy with metformin, thiazolidinediones, and insulin. DPP-4 inhibitors are well tolerated, have a low risk of hypoglycemia, and have no effect on weight. They are, nevertheless, relatively costly. It is yet unknown how long the effect on glycemic control and beta-cell morphology and function will last.

Insulin

Insulin can be used alone or in combination with oral hypoglycemic medications to control blood sugar levels. If some beta cell function remains, augmentation therapy with basal insulin can help. If beta cell fatigue occurs, basal-bolus insulin replacement is required. In cases of glucose poisoning, replacement therapy is required, which should mirror the normal release of insulin by the pancreas' beta cells. Insulin is available in four different types of injectables: rapid acting, short acting, intermediate acting, and long acting. When compared to short-acting versions, long-acting forms are less likely to cause hypoglycemia.

Insulin analogues

Insulin therapy's capacity to replicate normal physiologic insulin production was restricted. Traditional intermediate- and long-acting insulins (NPH insulin, lente insulin, and ultra-lente insulin) have irregular absorption and activity peaks, which can cause hypoglycemia. The new insulin analogues' pharmacokinetic characteristics differ from those of normal insulins, and their onset and duration of action range from short to lengthy. Insulin lispro and insulin aspart, both rapid-acting insulin analogues, and insulin glargine, a long-acting insulin analogue, are currently available.

Pathophysiology of type 2 diabetes mellitus (niddm)

The new insulin analogues' pharmacokinetic characteristics differ from those of normal insulins, and their onset and duration of action range from short to lengthy. Insulin lispro and insulin aspart, both rapid-acting insulin analogues, and insulin glargine, a long-acting insulin analogue, are currently available.

• Individuals who have a normal glucose tolerance.

• Diabetes caused by chemicals (called impaired glucose tolerance).

• Diabetes with modest fasting hyperglycemia (fasting plasma glucose 140 mg/dl).

• Diabetes mellitus with overt fasting hyperglycemia (fasting plasma glucose > 140 mg/dl).

Despite having the highest amounts of plasma insulin, people with poor glucose tolerance exhibit hyperglycemia, showing that they are insulin resistant. The level of insulin drops as impaired glucose tolerance progresses to diabetes mellitus, showing that people with NIDDM have decreased insulin secretion. In the average NIDDM patient, insulin resistance and insulin insufficiency are prevalent. Although insulin resistance is the primary cause of NIDDM, some researchers believe that insulin shortage is the fundamental reason because even moderate insulin resistance isn't enough to develop NIDDM. Both abnormalities are present in the majority of patients with the common form of NIDDM.A member of the nuclear hormone receptor superfamily of proteins has been implicated in the aetiology of type 2 diabetes, according to new research. Thiazolidinedione medications are a relatively recent family of pharmaceuticals used to boost the body's insulin sensitivity. These chemicals bind to the peroxisome proliferators-activated receptor g (PPAR-) and modify its function. PPAR- is a transcription factor that interacts to another transcription factor called the retinoid x receptor when activated (RXR). When these two proteins bind together, a group of genes is triggered. PPAR- is a crucial regulator of adipocyte differentiation that can cause fibroblasts or other undifferentiated cells to differentiate into mature fat cells. PPAR- is also involved in the creation of physiologically active chemicals by immune cells and vascular endothelial cells. Although there is no cure for the disease, therapy options include lifestyle changes, obesity management, oral hypoglycemic medications, and insulin sensitizers such as metformin, a biguanide that decreases insulin resistance and is still the first-line medicine for obese people. Non-sulfonylurea secretagogues, thiazolidinediones, alpha glucosidase inhibitors, and insulin are all useful medicines. New drugs, such as glucagon-like peptide 1 analogues, have been introduced as a result of recent study into the pathophysiology of type 2 diabetes:inhibitors of the sodium-glucose co-transporter-2, dipeptidyl peptidase-IVinhibitors, and 11-ß-hydroxysteroiddehydrogenase-1, insulin-releasing-glucokinase activators and pancreatic-G-protein-coupled fatty-acid-receptor agonists,glucagon-receptor antagonists, quick-release bromocriptine and metabolic inhibitors of hepatic glucose output. Inhaled insulin was approved for usage in 2006, but it was taken off the market due to low demand.

Complications

As the condition advances, tissue or vascular damage occurs, resulting in serious diabetes consequences include retinopathy, neuropathy, nephropathy, cardiovascular problems, and ulceration. Patients with type 1 diabetes for a long time are at risk for microvascular problems as well as macrovascular disease (coronary artery, heart, and peripheral vascular disease). Large artery atherosclerosis is a common complication of type 2 diabetes, which is also linked to hypertension, hyperlipidemia, and obesity. Cardiovascular problems and end-stage renal disease are the leading causes of death in people with type 2 diabetes.

Screening and diagnosis

Diabetic mellitus (DM) screening and diagnosis tests are widely available. Because the screening test is the same as the one used to diagnose diabetes, a positive screen is the same as a diagnosis of pre-diabetes or diabetes mellitus. Although around 25% of patients with type 2 diabetes have microvascular problems at the time of diagnosis, implying that they have had the disease for longer. It is still based on the American Diabetic Association (ADA) guidelines of 1997 or the World Health Organization (WHO) National Diabetic Group criteria of 2006, which are for a single raised glucose reading with symptoms (polyuria, polydipsia, polyphagia, and weight loss), otherwise raised values on two occasions, of either fasting plasma glucose (FPG) 7.0 mmol/L (126 mg/dL) or with an oral glucose tolerance test (OGTT), 2 hr after the oral dose a plasma glucose ≥11.1 mmol/L (200 mg/dL).

The FPG is the focus of the 1997 ADA recommendations for diagnosing diabetes, but the OGTT is the focus of the WHO. For determining blood sugar control over time, glycated hemoglobin (HbA1c) and fructosamine are still relevant. Practicing physicians, on the other hand, usually use additional measures in addition to those indicated. In July 2009, the International Expert Committee (IEC) proposed that a HbA1c level of 6.5 percent be added to the diagnostic criteria for diabetes. This group recommended that the term "pre-diabetes" be phased out in favor of a range of HbA1c levels between 6.0 and 6.5 percent to identify persons at high risk of developing diabetes.

There is no precise HbA1c threshold at which normalcy ends and DM begins, just as there is no definite threshold for glucose-based diagnostics. The IEC chose a cut-off point for DM diagnosis that emphasizes specificity, claiming that this balances the stigma and cost of incorrectly diagnosing people as diabetic with the limited clinical repercussions of delaying diagnosis in a patient with an HbA1c level <6.5%.

Alternative therapies to diabetes mellitus

Many efficient alternative remedies for treating diabetes mellitus have been developed, mainly in India. These treatments are highly effective and have no negative side effects. Yoga, acupuncture, hydrotherapy, and medicinal plants are examples of alternative therapies that are growing increasingly popular.

Yoga: Yoga comes from the Sanskrit term 'Yuj,' which means 'union of the body, breath, and mind.' Through an enzymatic process, stretching the abdomen during yoga exercise stimulates pancreatic cell regeneration and increases glucose utilisation and metabolism in peripheral tissues, liver, and adipose tissues. During yoga, the blood supply to the muscles is improved, and the muscles are relaxed, resulting in higher glucose uptake and a reduction in blood glucose levels. Yoga increases the activity of hepatic lipase and lipoprotein lipase at the cellular level, affecting lipid metabolism and increasing triglyceride storage in adipose tissue while decreasing blood triglyceride level. The sensitivity of the pancreatic -Cells to glucose has improved as a result of the various Yoga poses, resulting in increased insulin secretion. Fasting blood sugar (FBS), serum total cholesterol, low-density lipoproteins (LDL), very low-density lipoproteins (VLDL), and total triglycerides were dramatically reduced, while HDL-C was significantly increased, according to interventional research on yoga.

Acupuncture: Acupuncture is most well-known as a treatment for chronic pain. It has, however, been employed in the treatment of diabetes and its consequences in recent years. Acupuncture stimulates the pancreas, causing it to produce more insulin, increase the number of receptors on target cells, and speed up the use of glucose, reducing blood glucose levels. Although acupuncture has shown to be useful in the treatment of diabetes, its mechanism of action is yet uncertain.

Hydrotherapy: Because some people with type 2 diabetes are unable to exercise due to diabetic problems, hot-tub therapy is indicated to improve blood supply to the skeletal muscles. In diabetic individuals, 30 minutes of hot tub therapy reduced body weight, mean plasma glucose level, and mean glycosylated hemoglobin level, according to a study. When recommending hot tub therapy for diabetic patients, extra caution should be taken to guarantee optimum water sanitation and temperature.

Medicinal plants: Several medicinal plants have been found as having antidiabetic properties, according to Ayurveda. The majority of herbal remedies derived from these therapeutic plants are said to have few or no negative effects. Herbal plants have been utilized to treat diabetes mellitus since antiquity. The following are some of the most prevalent and helpful antidiabetic herbal herbs from India: Allium sativum (Garlic), Ghrita kumara(Aloe vera), Azadirachta indica (Neem), Benincasahispida (Ash Gourd), Caesalpinia bonducella (Fever Nut), Hibiscus rosa-sinesis (Gurhal), Jatropha curcas (Purging Nut), Mangifera indica (Mango), Momordica charantia (karela), Morus alba (Mulberry), Mucuna pruriens (Kiwach), Ocimum sanctum (Tulsi), Citrullus colocynthis (Bitter Apple) Coccinia indica (Ivy Gourd), Punica granatum (Anar), Syzygiumcumini (Jamun), Tinospora cordifolia (Giloy), and Trigonella foenum-graecum (Methi), Ficus benghalenesis(Banyan Tree), Acacia arabica (Babul), Aegle marmelose (Bael), Agrimonia eupatoria (Church steeples), Allium cepa (Onion), Gymnemasylvestre (Gurmar), Pterocarpus marsupium (bisasar). Evidence suggests that the active compounds of medicinal plants are also used in the development of modern allopathic drugs used to treat diabetes mellitus. For example, Metformin, the first-line conventional medication, was created from the guanidine-rich medicinal herb Galega officinalis. Because herbal medicine is inexpensive and has few or no negative effects in poor nations, 70-95 percent of the population has explored it for primary health care. The World Health Organization's (WHO, 2013) estimate on herbal medicine also supports the notion that 80% of the world's population still uses herbs and other conventional medicines to meet their primary health-care needs. Many diabetics in India are reliant on natural medications. Despite their widespread acceptance, the number of standardized herbal medications is limited due to a lack of regulatory standards and implementation protocols. Despite the fact that over 1000 plants have been employed in antidiabetic herbal formulations, only around 100 have been scientifically approved, and no single official herbal medicine has been developed for large-scale use to date. It's primarily due to a lack of standardization processes in place prior to drug development. Aside from that, herbal medicine is regarded as a one-of-a-kind alternative therapy for dialysis.

Herbal treatment of diabetes

With growing study in the field of traditional medicine, eco-friendly, bio-friendly, cost-effective, and reasonably safe plant-based medications have moved from the margins to the mainstream in the last few decades. There are multiple literature reviews on anti-diabetic herbal compounds by various authors, but the most useful is the review of Atta-ar-Rahman, who has recorded over 300 plant species recognized for their hypoglycemic activities. The plants in this review were categorized based on their botanical name, country of origin, parts used, and active agent nature. Momordica charantia is one of these plants (Family: Cucurbitaceae). The World Health Organization (WHO) has compiled a list of 21,000 plants used for therapeutic purposes around the world. India has 2500 species, with 150 of them being used economically on a considerable basis. India is the world's largest producer of medicinal herbs and is known as the "botanical garden."

Complications of diabetes

Diabetes mellitus causes a slew of consequences, including metabolic abnormalities, increased oxidative stress, and cardiovascular and renal illness. Diabetes problems are becoming more common among poor urban slum dwellers, middle-class people, and even rural residents. This is related to an increase in physical inactivity, nutritional changes, and stress among the population. Unfortunately, delayed therapy may raise the risk of complications in poor diabetic people. According to a study, those who engage in less physical activity are more likely to develop metabolic syndrome and hypertension. According to the same study, those who engage in light-intensity physical activity have a higher risk of developing coronary artery disease than those who do not. Both macrovascular and microvascular problems are the leading cause of morbidity and mortality in diabetes patients. Studies in India, such as The Chennai Urban Population Study and The Chennai Urban Rural Epidemiology Study (CURES), have provided valuable information on diabetic complications. The prevalence of coronary artery disease among diabetic participants, according to that study, was greater compared to the subjects with normal glucose tolerance. It was also noted that subclinical atherosclerosis measured by intimal medial thickness was high in diabetic subjects at every age. The prevalence of diabetic retinopathy was studied by CURES Eye study is the largest population-based data in India showed that the overall prevalence was 17.6 percent. A population-based study in Indians was reported that the prevalence of nephropathy was 2.2 percent and microalbuminuria was 26.9 percent. Overall, Asian Indians appear to have more cardiovascular problems. According to a recent research of the Chennai metropolitan population, total death rates are 18.9 per 1000 individuals per year, about three times higher in diabetics than in non-diabetic subjects (5.3 per 1000 person-year). As a result, the hazard ratio for diabetes-related all-cause mortality was shown to be greater. The study also found that mortality in diabetic patients owing to cardiovascular (52.9%) and renal disease (23.5%) disease was higher than mortality in nondiabetic persons due to cardiovascular (24.2%) and renal disease (6.1%) disease. Obesity, glucose intolerance, and dyslipidemia were found to be associated with migration from India's villages to cities, according to a study.

Management of diabetes mellitus

The cornerstone of diabetes mellitus management appears to be lifestyle management. It is well acknowledged as an important component of diabetes and cardiovascular disease prevention. According to meta-analyses, lifestyle interventions such as diet and physical activity reduced diabetes incidence by 63 percent in people at high risk. Although lifestyle modification programs have shown to alter diabetes risk variables, no data on their impact on diabetes incidence has been published. Diabetes mellitus nutritional management is a supplement to lifestyle management. It is beneficial to one's long-term health and quality of life. To avoid difficulties, dietary management seeks to achieve optimal metabolic regulation by maintaining a balance between food intake, physical activity, and medicine. Improved glycemic and lipid levels, as well as weight loss, are the dietary goals for people with type 2 diabetes.

Despite the relevance of lifestyle changes in diabetes treatment, most diabetics cannot ignore the importance of medication in achieving goal glucose levels. Various oral hypoglycemics have been used to help diabetics maintain blood glucose levels at the required threshold through various processes. Sulfonylureas and non-sulfonylurea secretagogues induce normoglycemia by increasing endogenous insulin secretion; alpha-glucosidase inhibitors delay intestinal carbohydrate absorption; and thiazolidinediones (TZDs) maintain normoglycemia by improving insulin sensitivity, primarily by increasing peripheral glucose disposal and suppressing hepatic glucose production. Metformin acts by reducing hepatic gluconeogenesis while also improving glucose mobilization and excretion in the peripheral tissues. Injections of synthetic insulin are also used to treat type 1 diabetes. Despite the availability of numerous effective oral hypoglycemic medications for the treatment of type 2 diabetes, 5 to 10% of diabetics have secondary failure, according to this bottleneck can be avoided if practitioners are aware of the limits of several currently used medicines. Secondary failure occurs when beta cell activity deteriorates, medication adherence is poor, weight gain, less exercise, dietary changes, or illness occurs. Hypoglycemic medications have a number of disadvantages, including the fact that they are costly and might cause side effects in patients. Plant-based therapies have been proven to be quite effective in the treatment of diabetes mellitus. It's worth noting that there's a new tendency in the world to resort to phyto-drugs to prevent the side effects of traditional hypoglycemics. Many plant species, including diabetes mellitus, have been utilized to treat life-threatening conditions. According to a research conducted by the World Health Organization (WHO), 80 percent of the world's population relies completely on medicinal plants for their main health care needs.

To date, the list of anti-diabetic medicinal plants is expanding at a brisk pace, especially on the African continent. Perhaps the economic condition in Africa has prompted African diabetics to seek out less expensive treatment and management choices. Because of this overreliance on antidiabetic medicinal plants, scientists are likely to conduct bioassays on these plants in order to discover new hypoglycemia medicinal plants. Some medicinal plant extracts have been shown to have anti-diabetic

properties in human and animal models of type 2 diabetes. More extensive research on antidiabetic plants, on the other hand, is required to alleviate concerns about in vivo safety and efficacy.

What does the future hold?

Glucose control (fasting and postprandial) and hemoglobin A reduction are currently the mainstays of type 2 diabetes treatment (1c). The consensus is that diabetes treatment should focus on slowing disease progression. Treatment methods are designed to target the disease's identified pathogenetic abnormalities. Treatment and/or management techniques have recently been focused on the development of new therapeutic options that are more effective in preserving normoglycemia in type 2 diabetics and provide long-term glucose control.

The realization that diabetes is a "metabolic curse" should pique one's interest in learning more about the biochemical and molecular causes of this metabolic illness. This knowledge will help researchers develop more effective diabetes control strategies. More effective synthetic insulin with quick actions, ability to traverse all bodily compartments, less side effects, and longer durations of activity are needed in this area. Oral hypoglycemic medications, which are reportedly plagued with side effects, must be tailored to counteract these drawbacks. To achieve the stated goal of decreasing the glycemic index in diabetics, lifestyle management must be optimized.

The difficulties of diabetes mellitus will undoubtedly be addressed by gene therapy. The cloning of the insulin gene prompted a groundbreaking gene therapy approach to diabetes mellitus. The technique was predicated on the idea that by utilizing an appropriate promoter and insulin gene construct, non-insulin generating cells might be made to produce insulin. It was anticipated that these surrogate cells could help diabetics restore their insulin production. The human genome has been unraveled thanks to advances in molecular biology. This milestone can be used to define the insulin gene in preparation for its eventual usage in diabetes care. Current advancements in molecular biology can help solve the immunological problems that underpin gene therapy. Regardless of these issues, it is critical to remember that the benefits of gene therapy for diabetes outweigh the disadvantages and current benefits when compared to traditional treatment before this strategy can achieve universal acceptance in the medical community.

CONCLUSION

Diabetes mellitus refers to a group of metabolic illnesses that all result in an excessively high concentration of a sugar called glucose in the blood if left untreated. Type 1 diabetes develops when the pancreas stops producing large amounts of the hormone insulin, mainly due to autoimmune damage of the pancreas' insulin-producing beta cells. Type 2 diabetes, on the other hand, is currently assumed to be caused by autoimmune attacks on the pancreas and/or insulin resistance. A person with type 2 diabetes may have a pancreas that produces normal or even abnormally excessive levels of insulin. The basic goal of diabetes management is to return carbohydrate metabolism to normal as much as feasible. Individuals with an extreme insulin deficit require insulin replacement therapy, which is administered via injections or pills. Insulin resistance, on the other hand, can be treated with dietary changes and exercise. Other objectives of diabetes management include preventing or treating the numerous problems that might arise from the disease and its treatment. Type 2 diabetes is a metabolic condition that can be avoided by changing one's lifestyle, controlling one's nutrition, and controlling overweight and obesity. The public's education is still crucial in combating this rising disease. Despite fresh insights into the disease's pathogenesis, there is no cure in sight. Individuals with type 2 diabetes should have their management adjusted to improve their quality of life. Diabetes can become a patient's companion if blood sugar levels are kept under control, allowing him or her to live life to the fullest. This study serves as a brief introduction to diabetes, which is a rising health-care concern that affects people's health, the health-care system, and the global economy. Patients with diabetes will be seen by health professionals at the hospital. It is their responsibility to be well-informed about diabetes and the most recent treatment options. New diabetes medications are being developed, and these vital functions have a significant impact on the prevention and control of the condition, hence improving the patient's quality of life.

REFERENCES

1. Arora Sachin, Ojha Shreesh Kumar, Vohora Divya (2009). Characterisation of Streptozotocin induced diabetes mellitus in Swiss Albino mice. Glo J of Pharmacol. 3: 81-84.
2. Ahmed AM (2002). History of diabetes mellitus. Saudi Med J. 23: 373-378.
3. Azevedo M, Alla S (2008). Diabetes in sub-saharan Africa: kenya, mali, mozambique, Nigeria, South Africa and zambia. Int J Diabetes Dev Ctries. 28: 101-108.
4. Barlow SE, Expert committee (2007). Expert committee recommendations regarding the prevention, assessment and treatment of childhood and adolescent overweight and obesity: Summary report. Paediatrics. 120: S164-S192.
5. Bastaki S (2005). Review Diabetes mellitus and its treatment. Int J Diabetes & Metabolism. 13: 111-134.
6. Boffetta P, Mclerran D, Chan Y, Manami I, Sinha R, Gupta PC (2011). Body mass index and diabetes mellitus in Asia. A cross sectional pooled analysis of 900,000 individuals in the Asia cohort consortium.
7. Blicklé JF (2006). Meglitinide analogues: a review of clinical data focused on recent trials. Diabetes Metab. 32: 113-120.
8. Coniff RF, Shapiro JA, Seaton TB, Bray GA (1995). Multicenter, placebo-controlled trial comparing acarbose (BAY g 5421) with placebo, tolbutamide, and tolbutamide-plus-acarbose in non-insulin-dependent diabetes mellitus. Am J Med. 98: 443-451.
9. Chen L, Magliano DJ, Zimmet PZ (2011). The worldwide epidemiology of type 2 diabetes mellitus: present and future perspectives. Nature reviews endocrinology.

10. Chiniwala N, Jabbour S. Management of diabetes mellitus in the elderly. Curr Opin Endocrinol Diabetes Obes. 18: 148-152.

11. Collier CA, Bruce CR, Smith AC, Lopaschuk G, Dyck DJ (2006). Metformin counters the insulin-induced suppression of fatty acid oxidation and stimulation of triacyglycerol storage in rodents skeletal muscle. Am J Physiol Endocrinol Metab. 219: 182-189.

12. Dixit VP, Joshi S (1985). Antiatherosclerotic effects of alfalfa and injection in Chicks: A Biochemical evaluation. Ind J of physiol & pharmacol. 29: 47-50.

13. Department of Health and Human Services. Centres for Disease Control and Prevention, 2011. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the United States, 2011.

14. Defronzo RA (2010). Current issues in the treatment of type 2 diabetes. Overview of newer agents: where treatment is going. Am J Med. 123: S38-48.

15. Eshrat M. Halim (2003). Effect of Coccinia indica (L.) and Abroma augusta (L) on glycemia, lipid profile and on indicators of end organ damage in streptozotocin induced diabetic rats. Ind J of Clin Biochem. 18: 54-63.

16. Fuhlendorff J, Rorsman P, Kofod H, Brand CL, Rolin B, MacKay P, Shymko R, et al. (1998). Stimulation of insulin release by repaglinide and glibenclamide involves both common and distinct processes. Diabetes. 47: 345-351.

17. Grover N, Bafna PA, and Rana AC (2011). Diabetes and methods to induce experimental diabetes. Inter J of Pharm and Biolo Scie. 1: 414-419.

18. Grinstein G, Muzumdar R, Aponte L, Vuguin P, Saenger P, DiMartino-Nardi Joan (2003). Presentation and 5-year follow-up of type 2 diabetes mellitus in African-American and Caribbean-Hispanic adolescents. Horm Res. 60: 121-126.

19. 19. González EL, Johansson S, Wallander MA and Rodríguez LA (2009). Trends in the prevalence and incidence of diabetes in the UK:1996 – 2005. J. Epidemiol. Community Health. 63: 332 336.

20. 20. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, Willett WC (2001). Diet, lifestyle, and the risk of type 2diabetes mellitus in women. N Engl J Med. 345: 790-797.

21. Jothivel Nandakumar, Ponnusamy Sethumathi Pudhupalayam, Appachi Malini, Singavarel Sengottuvelu, Rasilingam Duraisamy, Deivasigamani Karthikeyan, Thangavel Sivakumar (2007). Antidiabetic activities of methanol leaf extract of Costus pictus D. Don in alloxan-induced diabetic rats. J of Health Sci. 53: 655-663.

22. Kawamori R, Tajima N, Iwamoto Y, Kashiwagi A, Shimamoto K, Kaku K (2009). Voglibose for prevention of type 2 diabetes mellitus: a randomised, double-blind trial in Japanese individuals with impaired glucose tolerance. Lancet. 373: 1607-1614.

23. Kim YD, Park KG, Lee YS, Park YY, Kim DK, Nedumaran B (2008). Metformin inhibits hepatic gluconeogenesis through AMP-activated protein kinase-dependent regulation of the orphan nuclear receptor SHP. Diabetes. 57:306-314.

24. Kaprio J, Tuomilehto J, Koskenvuo M, Romanov K, Reunanen A, Eriksson J, Stengård J, et al. (1992). Concordance for Type 1 (insulin dependent) and Type 2 (non-insulindependent) diabetes mellitus in population based cohort of twins in Finland. Diabetologia. 35: 1060- 1067.

25. Kaku k (2010). Pathophysiology of type 2 diabetes and its treatment policy. JMAJ. 53: 41-4.

26. Lang IA, Galloway TS, Scarlett A, Henley WE, Depledge M, Wallace RB, Melzer David (2008). Association of urinary bisphenol A concentration with medical disorders and laboratory abnormalities in adults. JAMA. 300: 1303-1310.

27. Mueckler M (1990). Family of glucosetransporter genes: implications for glucose homeostasis and diabetes. Diabetes. 39: 6-11.

28. Mbanya JC. “The burden of type 2 diabetes mellitus in the African diaspora.”

29. Mohammad Ismail, Mohammad Yaheya (2009). Clinical evaluation of antidiabetic activity of Trigonella seeds and Aegle marmelos Leaves. Worl Appl Scien J. 7: 1231-1234.

30. Prevalence of overweight and obesity among adults with diagnosed Diabetes United States, 1988-1994 and 1999-2000”Centers for Disease Control and Prevention (CDC) (November 2004) MMWR. Morbidity and Mortality Weekly Report. 53: 1066-1068.

31. Patlak M (2002). New weapons to combat an ancient disease: treating diabetes. FASEB J 16: 1853e.

32. Rosenbloom AL, Joe JR, Young RS, Winter We (1999). Emerging epidemic of type 2 diabetes in youth. Diabetes Care. 22: 345-354.

33. Ripsin CM, Kang H, Urban RJ (2009). Management of blood glucose in type 2 diabetes mellitus. Am Fam Physician. 79: 29-36.

34. Scheen AJ (2005). Drug interactions of clinical importance with antihyperglycaemic agents: an update. Drug Saf. 28: 601-631.

35. Shorr RI, Ray WA, Daugherty JR, Griffin MR (1996). Individual sulfonylureas and serious hypoglycemia in older people. J Am Geriatr Soc. 44: 751-755.

36. Type 2 diabetes in children and adolescents (2000). American Diabetes Association.” Pediatrics. 105: 671-680.

37. Van Staa T, Abenhaim L, Monette J (1997). Rates of hypoglycemia in users of sulfonylureas. J Clin Epidemiol. 50: 735-741.

38. Wild S, Roglic G, Green A, Sicree R, King H (2004). “Global prevalence of diabetes: estimate for the year 2000 and projections for 2030. Diabetes Care. 127: 1047-1053.

39. Willi C, Bodenmann P, Ghali WA, Faris PD, Cornuz J (2007). Active smoking and the risk of type 2 diabetes: a systematic review and meta-analysis. JAMA. 298: 2654-2664.

40. Yki-Järvinen H (2004). Thiazolidinediones. N Engl J Med. 351: 1106-1118.

41. Yoon KH, Lee JH, Kim JW, Cho JH, Choi YH, Ko SH (2006). Epidemic obesity and type 2 diabetes in Asia. Lancet. 68:1681-1688.

42. Yach D, Hawkes C, Gould CL, Hofman KJ (2004). The global burden of chronic diseases: overcoming impediments to prevention and control. JAMA. 291:2616-2622.

43. Zimmet PZ (1992). Kelly West Lecture 1991. Challenges in diabetes epidemiology--from West to the rest. Diabetes Care. 15: 232-252.