Overview on Diabetes Mellitus

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Abstract

Diabetes mellitus is a group of diverse illnesses that often show hyperglycemia and glucose intolerance via insulin shortage, insulin impairment or both (Sicree et al., 2006). These difficulties occur due to disruptions in regulation systems controlling the storage and movement of metabolic fuels, including carbohydrate, lipid and protein catabolism and anabolism, induced by poor insulin production, insulin activity or both (Shillitoe, 1988; Votey and Peters, 2004). With more than 62 million diabetics already diagnosed in India, the situation of a potential pandemic is approaching fast.

1. Introduction

Many variables affect the prevalence of the disease across a nation and it is essential to understand these elements to facilitate change in health situations. So what are the current factors affecting diabetes in India that exacerbate the problem? Diabetes mellitus is categorized by etiology and clinical presence. Diabetes mellitus is thus divided into four sorts or classes: type 1 diabetes, type 2 diabetes, gestational diabetes, and other subtypes (Sicree et al., 2006). Although type 1 diabetes has been shown to be the most common form of diabetes in younger age groups in most industrialized nations, it constitutes a modest percentage of the overall burden of diabetes in a community. Type 1 diabetes in both industrialized and developing countries is becoming more common. In addition, a tendency in youngsters to type 1 diabetes is predicted at an earlier age (Sicree et al., 2006).

Diabetes mellitus and milder forms of glucose intolerance, including blood glucose tolerance, currently impact almost every locality in the world, and epidemiological research has shown that diabetes is expected to continue to grow worldwide in the lack of effective prevention and control programs (WHO, 1994). It is projected that approximately 285 million people aged 20 to 79 would have diabetes worldwide in 2010, with around 70 percent residing in developing nations. The number is expected to grow to about 438 million by 2030. Moreover, by 2030, the number of people living with IGT is projected to reach 472 million or 8.4 percent of the adult population (Sicree et al., 2006).

Diabetes is complicated in India, including genetic and environmental factors, such as obesity associated with increasing living standards, ongoing urban migration, and lifestyles changes. No country-wide or multi-centric study on diabetes prevalence and its effects has been done despite India's high incidence of diabetes. The research carried out are also likely to be mistaken since the diversity of the Indian people in culture, ethnic origin and the socioeconomic position may result in the extrapolation of regional findings to the whole country.

Obesity is a major risk factor for diabetes, although there has been a minimal study on this risk factor in India. Although the incidence of overweight and obesity is lower in India, the prevalence of diabetes is higher than in Western nations, implying that diabetes may afflict Indians with a far lower body mass index (BMI) than Europeans. As a result, Indians who are little and have a lower BMI may be in the same risk as fats. Indians are also genetically predisposed to coronary artery disease due to dyslipidemia and low levels of high-density lipoproteins, which contribute to Indians being more likely to develop diabetes.
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problems in their younger years (20-40 years) than Caucasians (> 50 years), implying that diabetes should be properly tested and managed in India regardless of patient age.

Because of the disease's long duration, an increase in individuals with early-onset diabetes is also responsible for the development of different diabetic complications. Despite this, data on the incidence of diabetes complications in India is limited.

A recent international study has shown that neuropathy was the most prevalent (24.5%), followed by cardiovascular issues (23.6%), renal difficulties (21.1%), retinopathy (16.6%), and foot ulcers (16.6% per cent). Diabetes control in persons suffering from a protracted disease (9.95.5 years) is most prevalent in neuropathy (24.6%), with cardiovascular consequences (23.6%), renal (21.1%), retinopathy (16.6%) and foot ulcers (5.5 per cent). These findings agree with previous South Indian population investigations, but more information from other parts of India is needed to establish if the issue rate patterns vary throughout the nation. In diabetic populations in India, poor glycemic gynaecological control is a cause of micro- and macrovascular diabetes-related changes and a risk factor for additional illnesses, including diabetic myonecrosis and diabetic muscular infarction. Case Plasmodium falciparum infection has increased in diabetic patients, and the combination of the two diseases limits treatment options and increases morbidity, mortality, and financial costs in a resource-constrained country like India.

Diabetes treatment in India is fraught with difficulties. Whereas HbA1c is the standard test start and intensification globally, it is not yet available for a substantial percentage of the Indian population. Furthermore, both the physician and patient communities lack "clinical inertia" for insulin therapy. The most frequent concerns are the complexities of the insulin system, concerns about weight gain, hypoglycemic episodes, and the fear of insulin pricking. Inadequacies in Indian guidelines are also to blame for wide disparities in treatment preferences across the country; simple and practical insulin guidelines that primary health care professionals can incorporate into routine clinical practice are urgently needed to facilitate insulin treatment and initiate treatment in all parts of the country. Suitable government measures and coordinated efforts by all societal stakeholders are needed to reduce the disease burden caused by diabetes in India.

2. Discussion of previous works

Clinicians may assist develop early detection and diabetes testing, diabetes prevention, self-management and diabetes therapy in line with local standards. The screening and diagnosis of prediabetes (especially in pregnant women, 26 children, and those with BMIs of less than 25) may improve society's results. 27 Continuing education programs for general practitioners may provide the "clinical inertia" required to adhere to the program and can be a key step to reach glycemic objectives and reduce the effects of the illness. Aggressive treatment measures such as early initiation of insulin, optimal doses of oral hypoglycemic medicines, and appropriate changes in the lifestyle may have long-term effects on disease management.

Government policies may contribute to implementing recommendations on diabetes management, support Community initiatives to increase public awareness on diabetes risk reduction and ensure that all community members have access to medicine and diagnostic services.

Governments and organizations across the globe have made great health results for their people by intervening in diabetes management. There are several successful US diabetes preventive and management initiatives supported by the government and the business sector. Similarly, the Australian government conducts programs such as the National Health Priority Areas Initiative, which focuses on chronic illnesses such as diabetes and focused and continuous treatment. To provide patient education, the National Health Service conducts several patient education and research studies, such as the DAFNE study and the Diabetes Education & Self-Management for Ongoing & New Diagnosed (DESMOND) study. An expert group has been established in the United Arab Emirates to offer suggestions for diabetes treatment and public awareness programs. This has had a beneficial health impact and may have halted the country's rising diabetes rate. 34 Similar initiatives and services at the grassroots level are needed in India to decrease the pandemic of new-age diabetes.

2.1 Mellitus and Other Lifer Regulation Classification Category

The conditions at the time of diagnosis greatly affect the assignment of a person to a type of diabetes, and many diabetics are not appropriately grouped. For example, those with gestational mellitus (GDM) diabetes may be diagnosed with type 2 diabetes and will remain hyperglycemic following birth. A diabetic person, on the other hand, may develop diabetes via high dosages of exogenous steroids.

The patient becomes normoglycemic if glucocorticoids are discontinued, but the patient may develop diabetes many years later with repeated pancreatitis episodes. Another example is a person who gets diabetes years later who is treated with thiazides. Since thiazides seldom lead to significant self-evident hyperglycemia, these persons may develop diabetes type 2, which is worsened by the medication. Therefore, the cause of and treatment of hyperglycemia is more essential than the diabetes type for the doctor and the patient.
Diabetes Type 1 (death of beta cells, resulting in total insulin shortage)

2.2 Immune system-caused diabetes.

This kind of diabetes, which only concerns 5–10% of people with diabetes and has been formerly known as insulin-independent diabetes, diabetes type 1 diabetes or juvenile-onset diabetes, is responsible for cell-based autoimmune damage in cells. The use of _ characterizes immunological destruction-cells for island autoantibodies, insulin autoantibodies, glutamic acid autoanticord (GAD65), and autoanticords for tyrosine-phosphatases IA-2 and IA-2_. 85–90 percent of these autoanticords are present when hyperglycemia is first diagnosed. There are also substantial HLA associations with the illness, including DQA and DQB gene connections and their impact on DRB genes. The HLA-DR/DQ alleles may be protective or predisposing.

Idiopathic diabetes is a kind of diabetes that is unobtrusive. There are no recognized causes for different types of type 1 diabetes. Some individuals are vulnerable to chronic insulinoplasia and ketoacidosis yet have no evidence of autoimmunity. Although this group includes just a small percentage of people of type 1, most of them are African or Asian. Episodic ketoacidosis occurs in people with this kind of diabetes and they have different insulin levels between episodes. This kind of diabetes is mainly hereditary, immune-free and not related to HLA. In those afflicted, insulin replacement treatment may be necessary.

While the reasons are unknown, there are no autoimmune injuries to cells, and there are no additional causes of diabetes listed above or below. Most individuals with such diabetes are fat and obesity promotes insulin resistance. A larger percentage of body fat may be distributed mostly in the belly in patients who do not correspond to the conventional definition of obesity. This kind of diabetes is uncommon for ketoacidosis and is generally related to stress from other diseases, such as infection. Hyperglycemia is typically neglected over many years due to sluggish development and an often inability to detect any traditional diabetes symptoms in the early stages. Macrovascular and microvascular problems are nevertheless more likely to develop. Insulin levels
may seem normal or raised for those with such diabetes, but if normal, an increase in blood glucose levels is expected to generate even greater insulin levels in these diabetic people. As a result, insulin production is impeded and insulin resistance cannot be compensated for. Insulin resistance may improve with weight loss and/or medication hyperglycemia, although rarely. This kind of diabetes is more likely to develop with age, obesity and lack of physical exercise. The prevalence varies according to race and ethnicity in women with preceding GDM and women with high blood pressure or dyslipidemia. It is linked with a significant genetic predisposition more frequently than the autoimmune variant of type 1 diabetes. The genetics of this diabetes are, on the other hand, complicated and uncertain.

2.3 Mellitus Diagnostic Criteria For Diabetes
The factors for assessing whether or not someone has diabetes are listed in Table 2. Diabetes may be diagnosed in three ways and one of three methods mentioned in Table 2 must each be confirmed on the next day in the absence of unequivocal hyperglycemia. At present time, hemoglobin A1c (A1C) is not recommended for diagnosis of diabetes. GDM is a kind of pancreatic diabetes. Carpenter and Coustan's criteria are used for abnormal glucose tolerance in pregnancy (3). Carpenter/Coustan Diagnostic criteria and the 75-g 2-h OGTT diagnosis were approved by the Fourth International Workshop on Gestational Diabetes Mellitus of the American Diabetes Association in March 1997. The following table outlines these requirements.

2.4 Gestational diabetes diagnosis
For a positive diagnosis, two or more of the venous plasma concentrations must be reached or surpassed. The test should take place in the morning after an overnight fast of eight to 14 hours, at least three days of unrestricted eating and unrestricted physical activities (150 g carbohydrates/day). The individual should stay sitting and not smoking throughout the test.

On the first prenatal appointment, a risk assessment for GDM should be carried out. Women who have a significant risk of GDM (severe obesity, GDM personal history, glycosuria, or a significant family history of diabetes) should have their glucose levels checked as soon as practicable. Between 24 and 28 weeks of pregnancy, they should be checked if GDM free has been confirmed in the initial screening. Medium risk women from 24 to 28 weeks of pregnancy should be evaluated. The diagnosis of people with diabetes is validated by fasting plasma glucose levels at _126 mg/dl (7.0 mmol/l) or by casual plasma glucose levels of _200 mg/dl (11.1 mmol/l). If there is no obvious evidence of hyperglycemia the following day, the diagnosis must be confirmed. If the diagnosis is verified, the need for a glucose challenge is removed without this degree of hyperglycemia.

In women with medium or high-risk properties, one of two techniques should be employed to evaluate GDM. One stage process. Perform an OGTT diagnosis without plasma or serum glucose levels first. The one-step approach may be economical for high-risk people or communities (e.g., some Native-American groups).

A two-step process
Measuring plasma or blood glucose levels for women above a GCT glucose threshold 1 hour after 50 g oral glucose load (GCT) and OGTT diagnose. GCT Diagnosis of Glucose _80 percent of women with GDM are found to be _140 mg/dl (7.8 mmol/l) using the two-stage method and improve the yield to 90 percent by a cut of 130 m g/dl (7.2 mmol/l). The diagnosis of GDM is based on OGTT in every situation. The following table includes diagnostic criteria for the 100-g OGTT derived from the earlier studies of O'Sullivan and Mahan(4), and amended by Carpenter and Coustan (3). A load of 75 grams of glucose and the results of the test may also be used for diagnosis. The quicking, 1 and 2 h glucose threshold values (Table 2 below, while not as valid as the 100-g OGTT are provided;

2.5 Mellitus Etiology Diabetes
Diabetes mellitus has a variety of unknown causes. The etiology of diabetes mellitus is now widely recognized as complicated, including both genetic and environmental components.

2.6 Mellitus and Genetic Factors of Diabetes
Can Run in Families with Diabetes. According to ancient Hindu doctors, diabetes may be handed down from generation to generation. 25 to 50 per cent of all diabetics have a favorable family history. 5 Pincus and White6 investigated the prevalence in the family of diabetic patients with diabetes mellitus to establish whether the illness was really inherited. In close relatives (parents and siblings) with diabetes, 10 to 30 percent of diabetes was reported when more sensitive indicators were used, such as oral and intravenous glucose tolerance tests and glucose tolerance tests for cortisone-induced disease. One to six percent of relatives of non-diabetic people have diabetes. As a result, close relatives with diabetes have an increased risk of both clinical and impaired glucose tolerance.
2.7 Mellitus Diabetes Ethnic and Enviable Factors
2.7.1 Environmental and Ethnic Factors Type I
In etiology and results, both types I and type II diabetes were associated with environmental factors. Diabetes type I prevalence and incidence vary substantially depending on ethnicity and location. 18 The Nordic countries have the most reported cases of diabetes type I and Finland (35/100,000/year) have the greatest incidence among children aged 0–14 years. Asia is the lowest (0.5–1.3 per 100,000 per year). Low rates have also been reported in Africa and Latin America. Differences in genetic and environmental factors may be attributed to the significant inequalities in type I diabetes. According to much research, the prevalence and clinical features of diabetes mellitus vary greatly. This difference across ethnic groups may be due to a mix of hereditary and environmental factors. It may also indicate that genetic differences exist among ethnic groups. There seems to be a connection between excessive consumption and the prevalence of diabetes. Diabetes has increased substantially among certain groups in Israel due to their migration to Israel, and subsequent dietary changes, such as Kurdish and Yemeni Jews. Environmental factors thus have a role in ethnic changes in the incidence of diabetes mellitus. However, the clinical phenomenology of diabetes in ethnic groups differs significantly, suggesting an inherited component. The annual rate of child type I diabetes in France between 1988 and 1995 was similar to that of Nordic countries. Although the annual rates of childhood type I diabetes are similar, the overall incidence of type I diabetes in French children is still lower. This shows that environmental factors affect the development of type 1 diabetes significantly.

2.8 Symptoms and indications of diabetes
Many people miss the signs and symptoms of diabetes due to the continuing disease. People may not believe this is a severe problem, since, unlike many other diseases, the effects of hyperglycemia may not occur quickly. Several people may not realize that many years before symptoms appear, harm may begin. This is problematic because early symptom detection may help treat the illness quickly and prevent vascular problems.

Diabetes warning signals and typical symptoms In its early stages, because of the quiet nature of Type 2 diabetes, it is important that individuals be aware of their warning signals.

Polyuria, polydypsies and polyphagia, which have a quick start of acute hyperglyceremia, and type 2 diabetes with very high levels of hyperglycemia, are frequent signs of diabetes in both type 1. Only type 1 and type 2 diabetes that has not been diagnosed for a long time produce substantial weight loss. Undiagnosed diabetes may also cause tiredness, restlessness and physical discomfort. Loss of weight not explained. Moderate or gradual symptoms may be missed.

2.9 Diabetic and complementary pathophysiology
The real cost of diabetes in both financial and human suffering lies in the many repercussions of the illness. There are three kinds of difficulties, each with its unique growth process, while everyone shares some aspects. There is one factor in common with all the problems: excessive blood glucose levels. The three types of complications include macrovascular, microvascular and neurological problems.

2.10 Macrovascular system complications
The major blood arteries in the heart, brain, and legs are referred to as macrovascular. The coronary arteries and the legs are the most common sites of macrovascular disease. The majority of people with diabetes are affected by coronary artery atherosclerosis, which is the leading cause of diabetes mortality. 10 The disease may strike these people considerably younger than the average population, and women are not immune. The process behind the development of macrovascular disease in people without diabetes is the same, except for the speed and lack of women’s protection. It is unclear how this connects to dangerously high blood glucose levels. Cholesterol, smoking, sedentary lifestyle, obesity, hypertension, and other risk factors are all recognized to increase the probability and severity of coronary artery disease. High blood glucose levels are well recognized to interfere with the coagulation process. High levels of glucose may promote thrombosis and delay fibrin clearance, two key cardiac thrombosis variables, via a variety of negative effects on platelets and fibrin. Increased blood glucose levels may hasten the formation of atherosclerotic plaques through processes similar to those described below for microvascular disease. While the exact process is unknown, blood glucose levels in diabetics must be managed in order to prevent serious blood vessel damage. Blood glucose levels are also essential for better outcomes after acute cardiac events and surgery. Recent research shows that keeping blood glucose levels below 150 mg/ml throughout the post-myocardial or coronary artery bypass grafting phase reduces morbidity and death by almost half. IV insulin regimens should be part of the therapeutic plan in all coronary treatment units and critical operational care units.

2.11 Complications with microcirculation and the nervous system
The etiology of small vessel illnesses is more frequent in individuals with diabetes mellitus than that of large vessel diseases. Microvascular disease affects the body and causes a variety of symptoms. The eyes and kidneys are undoubtedly the most impacted organs. Diabetic retinopathy is the leading cause of adult blindness, whereas diabetic nephropathy affects more than
half of dialysis patients or recipients of kidney transplants. Microvascular disease may be caused by three different mechanisms: polyol, enzyme glycosylation, and non-enzyme glycosylation. The core of all vascular damage processes is a distinguishing characteristic of vascular and nerve tissues. Without insulin, these tissues are allowed to enter the cells. Most tissue cells need insulin to absorb glucose, however, glucose may be absorbed in vascular and nerve tissue. As a result, glucose in the vascular endothelium and nerve cells is identical to glucose in the plasma. If the glucose level in these cells becomes too high, the glucose removal mechanism must be triggered. These disposal techniques are essential, but they are unpleasant. This has the potential to harm blood vessels and nerves, as well as the organs that serve them. To prevent serious damage, blood glucose levels must be maintained within the normal range most of the time. Inadequate insulin and an overabundance of glucose cause glycosylation of the enzyme. Glucose is introduced into the cells, causing a series of chemical reactions that produce energy, CO2, and water through the cancer cycle. The phosphorylation of carbon 6 glucose is the initial step. To activate glucose, the G-6-P hexokinase enzyme, which is sensitive to insulin catalysts, is needed. In the presence of insulin shortages, the cell glucose exceeds, but it cannot enter a normal glycolytic process and must be transferred as it accumulates in other metabolic pathways. Enzyme glycosylation is one of these methods since the process needs two enzymes. When glucose cannot be converted to G-6-P, the non-insulin-dependent hexokinase and phosphorylate1 glucose enzymes are activated (G-1-P). In the absence of insulin, another enzyme, glycosyl transferase, is activated. This enzyme binds G-1-P to certain amino acids in the protein chains of the blood vessel's cell membrane.

The basement membrane comprises protein rings surrounding the blood artery, similar to onion peel rings. Water, oxygen, glucose, amino acids, and other tiny molecules will be permitted to enter the tissue via the blood artery wall but larger blood molecules such as serum albumin and other proteins will be retained. This is the function of tiny, slit membrane pores, which enable small molecules to flow while preventing large molecules from passing through. Proteins are retained on the membrane in addition to slit pores, rejecting the proteins through an electrical weight in the blood artery. By glycosylation of the basement membrane protein, the basement membrane membrane is stretched and the slit holes are enlarged. Because the electrical charge is transferred when the glucose binds to the protein, the membrane becomes electrically neutral. This electrically neutral barrier lacks blood proteins, as shown by extended slit holes and protein leakage. The presence of protein in the urine is the first sign of diabetic nephropathy. Both fluorescein leakage into the retina and peripheral album leakage are caused by the same process. The thickening of the membrane cells may also decrease blood channel diameter, limit blood flow, and increase intravascular pressure.

2.12 Treatment / Management
Diabetes physiology and therapy are complex, and a range of therapies are needed for effective disease control. In diabetes treatment, diabetic education and patient involvement are important. Patients who can manage their food, exercise regularly (more than 150 minutes a week) and check their levels of glucose independently can get better outcomes. [28] For the remainder of one’s life, treatment may be necessary to prevent complications. Glucose levels should be maintained between 90 and 130 mg/dL, and HbA1c should not exceed 7 percent. While it is important to regulate glucose, overly strong therapy may lead to hypoglycemia that has severe or fatal implications.

Because the T1DM is mainly due to insulin failure, daily injections or an insulin pump are the cornerstone of therapy. Diet and exercise, in particular initially, maybe adequate T2DM treatment. Other therapies may increase the sensitivity to insulin or decrease blood sugar levels. Secretion of pancreatic insulin should be enhanced. Certain particular inhibitors include: biguanides, meglitinides, alaphaglucosides, thiazolidinedion, agonists, glucagonlike-peptide-1, dipeptidyl-peptidase inhibitors (DPP-4), amylinonime transporter and SGLT-2 (sodium-glucose) Metformin is the first-line diabetes medication to decrease both basal and postprandial plasma glucose levels. T2DM patients, particularly those with poor glucose control, may require insulin therapy in the late stage of the illness. Bariatric surgery may be performed in extreme obesity to normalize glucose levels. For individuals who have not responded to prior treatments and have several comorbidities. It is suggested. [29] Both GLP-1 agonists, liraglutide and semaglutide were related to improved cardiovascular results. Empagliflozin and canagliflozin inhibitors SGLT-2 have also been shown to improve cardiovascular outcomes and offer potential rehabilitation and prevention of heart failure.

3. Conclusion
Microvascular problems are a feared diabetes result that needs regular tests. Regular retinal diabetes examinations for diabetic retinopathy should be performed by trained medical professionals. A neurological examination and a monophilament test may be used to identify individuals at risk of amputation with neuropathy. Clinicians may also urge patients to conduct foot examinations for lesions that may otherwise be missed because of neuropathy. Diabetes, low-dose tricyclic antidepressants, duloxetine, anti-invulsants, topical capsaiicine, pain medicines may require neuropathic pain. Early diabetic renal alterations such as albuminuria may be identified above 30 mg/g of creatinine in combination with the estimated GFR from the urine microalbumin test. The angiotensin converting enzymes (AECs) inhibitors and receptors have antiproteinuric effects that make them the drugs to delay the development of macroalbuminuria in people with type 1 and type 2 diabetes.
In order to treat diabetic peripheral neuropathy, FDA has authorized Pregabaline and Duloxetine. Furthermore, anti-convulsants and tricyclic antidepressants are utilized to relieve pain in diabetic neuropathy with varied effectiveness levels. Regular blood pressure screening, aimed at 130 mmHg, is also recommended for diabetes by systolic blood pressure and diastolic blood pressure of 85 mmHg.

Angiotensin converting enzyme inhibitors, angiotensin receptor blockers, diuretics, beta-blockers and/or calcium channel blockers are often used to treat high blood pressure diabetes. The American Diabetes Association (ADA) advises monitoring diabetics for LDL-C cholesterol levels below 100 mg/dL and 70 mg/dL without cardiovascular disease (CVD) (ASCVD). Statins are the first-line diabetes therapy. Low-dose aspirin may be useful for individuals at high risk for diabetes, the American Diabetes Association says, but aspirin does not help reduce cardiovascular events in patients with diabetes.

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References

[1] Townsend, T. (2000). A decade of diabetes research and development. International Journal of Diabetes and Metabolism, 8, 88-92.
[2] Amos, A. F., McCarty, D. J., & Zimmet, P. (1997). The rising global burden of diabetes and its complications: estimates and projections to the year 2010. Diabetic medicine, 14(S5), S7-S85.
[3] Zimmet, P. (2003). The burden of type 2 diabetes: are we doing enough?. Diabetes & metabolism, 29(S5), S7-S85.
[4] LL, F. (1957). Diabetes mellitus in the texts of old Hindu medicine (Charaka, Susruta, Vagbhata). The American journal of gastroenterology, 27(1), 76-95.
[5] Emery, A. E., & Rimon, D. L. (Eds.). (1990). Principles and practice of medical genetics (Vol. 2). Churchill Livingstone.
[6] Pincus, G., & White, P. (1933). On the inheritance of diabetes mellitus I An analysis of 675 family histories. American Journal of the Medical Sciences, 186, 1-14.
[7] Trevisan, R., Vedovato, M., & Tiengo, A. (1998). The epidemiology of diabetes mellitus. Nephrology, dialysis, transplantation: official publication of the European Dialysis and Transplant Association-European Renal Association, 13(suppl 8), 2-5.
[8] Harris, R. L. (2008). Neurocognitive implications of diabetes on dementia as measured by an extensive neuropsychological battery. University of North Texas.
[9] Rotter, J. I., Rimon, D. L., & Samloff, I. M. (1978). Genetic heterogeneity in diabetes mellitus and peptic ulcer. Genetic epidemiology, 381-414.
[10] Rimon, D. L. (1969). Ethnic variability in glucose tolerance and insulin secretion. Archives of internal medicine, 124(6), 695-700.
[11] Sharp, P. S., Mohan, V., Levy, J. C., Mather, H. M., & Kohner, E. M. (1987). Insulin resistance in patients of Asian Indian and European origin with non-insulin dependent diabetes. Hormone and metabolic research, 19(02), 84-85.
[12] Scheen, A. J., & Lefèbvre, P. J. (1996). Pathophysiology of type 2 diabetes mellitus. In Oral Antidiabetes (pp. 7-42). Springer, Berlin, Heidelberg.
[13] Berntorp, K., Lindgärde, F., & Malmquist, J. (1984). High and low insulin responders: relations to oral glucose tolerance, insulin secretion and physical fitness. Acta Medica Scandinavica, 216(1), 111-117.
[14] O'Dea, K., Traianedes, K., Hopper, J. L., & Larkins, R. G. (1988). Impaired glucose tolerance, hyperinsulinemia, and hypertriglyceridemia in Australian aborigines from the desert. Diabetes Care, 17(1), 23-29.