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Mini Review

Risk Factors, Complications and Management of Diabetes Mellitus

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Abstract

Diabetes mellitus can be a complex disorder described by chronic progressive metabolic disorder and chronic hyperglycemia arising from dysregulation of carbohydrate, lipid and protein metabolism. Verapamil is a class of non-dihydropyridine calcium channel blockers, which prevents the second phase of glucose stimulated insulin release by inhibiting the intake of calcium into the cytosol of beta cells and also prevents sulfonylurea and glucagon-induced insulin release. The complications of diabetes mellitus are categorized as microvascular (nephropathy, neuropathy, and retinopathy) or macrovascular (cardiovascular and cerebrovascular disease). Diabetic nephropathy is a microvascular complication described by elevated urinary albumin excretion (proteinuria) or lowered kidney glomerular filtration rate in both types of diabetic mellitus. The objective of diabetes mellitus management is to inhibit the mortality and to delay the onset of disease complications and to hinder its progression by improving patients' blood sugar level and controlling the risk of cardiovascular disease. Metformin enhances liver, muscle & adipose tissues sensitivity to the insulin and elevate peripheral glucose utilization and also prevents gluconeogenesis and obviates glucose absorption from gastrointestinal tract.

Keywords: Complications; Diabetes Mellitus; Management; Risk Factors; Gastrointestinal, Cardiovascular; Nephropathy; Retinopathy; Proteinuria; Dysregulation

Introduction

Diabetes mellitus can be a complex disorder that described by chronic progressive metabolic disorder and chronic hyperglycemia arising from metabolism dysregulation of carbohydrate, lipid and protein. Diabetes mellitus is a major health issue that affects more than 463 million human beings worldwide [1]. There are two most common types of diabetes mellitus which discussed in turn below. Type 1 diabetes mellitus can be expressed by quick rate of beta cell dysfunction and results from an absolute deficiency of insulin hormone secretion. The rapid advancement of type 1 diabetes mellitus is frequently demonstrated in pediatric and perhaps also happen in adults [2]. Type 2 diabetes mellitus is more commonly resulted from peripheral insulin resistance and also relative deficiency of insulin hormone or inadequate insulin secretion by pancreatic beta cells; then the resistance of insulin causes the levels of free fatty acids and proinflammatory cytokines in plasma elevated and eventually glucose transport into muscle cells is lowered, hepatic glucose production is enhanced, and breakdown of fat is accelerated [3]. Skeletal muscle, liver and adipose tissue insulin resistance are the main cause of type 2 diabetes mellitus and eventually accelerated the destruction and failure of pancreatic beta cell [4].

Risk Factors of Diabetes Mellitus

Family history, obesity, race/ethnicity, age increment greater than or equal to forty yr, previous distinguished fasting glucose

impairment or impairment of glucose tolerance, hypertension, hyperlipidemia and history of gestational diabetes mellitus are the commonly known risk factors of diabetes mellitus [1]. Some medications cause diabetes mellitus are discussed in turn below [5]. Thiazide diuretics cause hypokalemia; then eventually cause impairment of insulin secretion secondary to potassium depletion, and thiazides may prevent the conversion of proinsulin to insulin. Thiazide diuretics may be also accelerated insulin resistance due to elevated free fatty acid mobilization. Verapamil is a class of non-dihydropyridine calcium channel blockers; which prevents the second phase of glucose stimulated insulin release by inhibiting the intake of calcium into the cytosol of beta cells; and also prevents sulfonylurea and glucagon-induced insulin release. Beta adrenoceptor blockers may antagonize induction of insulin resistance, somewhat through weight gain and beta blockers also prevent insulin release from pancreatic beta cells.

Human immunodeficiency virus protease inhibitors by itself cause peripheral lipodystrophy, hyperlipidemia and muscle resistance of insulin and also prevent conversion of proinsulin to insulin. HIV protease inhibitors may bind to proteins target, which regulate lipid metabolism, eventually cause the elevation of circulating fatty acids that could interfere with insulin signaling and compete with glucose cycle intermediates. Beta adrenoceptor agonists can stimulate insulin secretion by increasing hepatic glucose output, and finally hyperglycemia resulted from insulin secretion. Fluoroquinolones prevents the release of insulin by blocking ATP sensitive potassium channels. Niacin cause elevated skeletal muscle resistance to insulin due to accelerated free fatty acid mobilization. Oral contraceptive pills and estrogen replacement therapy can lower insulin sensitivity in women without diabetes receiving some contraceptive pills, and several implantable hormonal contraceptives may be causing the alterations in carbohydrate metabolism, including impairment of glucose tolerance and increased insulin resistance. Glucocorticoids can lower the hepatic and peripheral tissue sensitivity of insulin through post-receptor mechanisms and cause gluconeogenesis. Conventional antipsychotic medications sometimes cause hyperglycemia; however, the use of the atypical antipsychotics, especially clozapine and olanzapine, alter receptor binding characteristics, leading to enhanced insulin resistance. Phenothiazines prevents the release of insulin from pancreatic beta cells.

Complications of Diabetes Mellitus

Gender, long duration with diabetes mellitus, poor and inadequate glycemic control, negative attitude towards diabetes mellitus, poor treatment adherence and poor knowledge about the disease are the common risk factors for occurrence of diabetes mellitus complications [6]. Severe complications such as nephropathy, retinopathy, neuropathy and cardiovascular diseases resulted from chronic hyperglycemia. Complications of diabetes mellitus can be the advancement and nearly resulted by chronic exposure to high blood sugar levels by dysfunctions of insulin metabolism and biological macromolecules such as carbohydrates, lipids, proteins and nucleic acids. The complications of diabetes mellitus are quickly becoming the world's consummate important cause of morbidity and mortality [7]. The complications of diabetes mellitus are categorized as microvascular (nephropathy, neuropathy, and retinopathy) or macrovascular (cardiovascular and cerebrovascular disease). Diabetic nephropathy is a microvascular complication described by elevated urinary albumin excretion (proteinuria) or lowered kidney glomerular filtration rate in both types of diabetic mellitus [8].

Diabetic neuropathy is a microvascular complication characterized as a non-inflammatory impairment of the function and structure of peripheral somatic or autonomic nerves due to metabolic-vascular pathology [9]. Diabetic retinopathy can be one of the most serious microvascular complications, which resulted from chronic elevated blood sugar levels and nearly happen in medium and late stage of both types of diabetes mellitus. Diabetic retinopathy can eventually cause severe visual impairment, vitreous hemorrhage, and blindness [10]. Macrovascular complications can be resulted from a combination of hyperglycemia and altered lipid metabolism. Heart failure can be a major macrovascular complication emerged as an important and elevating clinical and public health problem. Diabetes mellitus is correlated with an elevated risk of coronary heart disease. Coronary heart disease is the main cause of morbidity and mortality around the world [11]. Diabetes mellitus carries an elevated risk of heart disease, hypertension and stroke due to atherosclerosis, which advances earlier in diabetics than in nondiabetics. Diabetic foot ulcer is macrovascular complications which often happen on the soles of the feet in patients with diabetes mellitus due to peripheral neuropathy or peripheral arterial disease on all skin layers, necrosis or inflammation [12].

Management of Diabetes Mellitus

The objective of diabetes mellitus management is to inhibit the mortality and to delay the onset of disease complications and to hinder its progression by improving patient's blood sugar level and controlling the risk of cardiovascular disease. There are three preponderance components of diabetes mellitus management which are diet and exercise, oral antidiabetic medications and insulin treatment [13].

Non-Pharmacological Management

The objective of dietary management of diabetes mellitus are correcting any correlated blood lipid abnormalities, permitting good glycemic control with blood glucose levels, ensuring weight control and providing nutritional requirements. Medical nutritional treatment is recommended for all individuals with diabetes mellitus and pivotal therapy for patients with diabetes mellitus. Type 1 diabetes mellitus can focus on regulating insulin administration with balanced diet to achieve and maintain healthy body weight. Type 2 diabetes mellitus usually need caloric restriction to promote weight loss and portion size and frequency or usually issues [14]. Physical activity particularly aerobic exercise ameliorates insulin sensitivity and glycemic control in the majority of individuals and reduces cardiovascular risk factors, contributes to weight loss and ameliorates well-being. Physical activity objectives involve at least 150 minutes per week of moderate intensity exercise [15].

Pharmacological Management

There are four major common groups of oral hypoglycemic medications such as biguanides which lower gluconeogenesis in the liver involving metformin, insulin secretagogues which stimulate the pancreas to secrete insulin including sulfonylureas, insulin sensitizers which ameliorate sensitivity of peripheral tissues to insulin including thiazolidinediones and Alpha glucosidase inhibitors involve acarbose and miglitol [16]. Metformin enhances liver, muscle & adipose tissues sensitivity to the insulin and elevate peripheral glucose utilization and also prevents gluconeogenesis and obviates glucose absorption from gastrointestinal tract [13]. Thiazolidine can improve glycemia by lowering the skeletal muscle insulin resistance and keeping pancreatic beta cell function with different mechanism of action such as ameliorating peripheral intake and use of glucose in muscle and fat, eventually lowering liver glucose release. Sulfonylurea such as glipizide, glyburide (glipalamide, glimepiride act by stimulating insulin secretion from the insulin releasing beta cells located in the pancreas and slightly ameliorate insulin resistance in peripheral target tissues (muscle, fat).

Their receptor is a constituent of the ATP dependent potassium channel in the pancreatic beta cells; their binding is leads to suppression of ATP dependent potassium channel channels, which alters the resting potential of the cell, leading to calcium influx and stimulate insulin releasing [17]. Meglitinide analogues are insulin secretagogue as sulfonylureas. Meglitinide analogues are structurally different from sulfonylureas; but they have relatively similar mechanism of action to that of sulfonylureas (they act by regulating ATP-dependent potassium channels in pancreatic beta cells), because they stimulate the secretion of insulin from the pancreatic beta cells via a different binding site on the "sulfonylurea receptor" [18]. Alpha glucosidase inhibitors involve acarbose and miglitol. Inhibitors of intestinal α-glucosidase enzymes degenerate the rate of carbohydrate digestion, thereby, providing an optional notation to lower postprandial. Alpha glucosidase inhibitors act on alpha glucosidase; an enzyme found in brush border cells of small intestine, sticking greater complex carbohydrates into sugars and prevents the breakdown and absorption of carbohydrates [19].

Insulin provides glucose homeostasis by thought-out the plasma glucose worth in an optimum group throughout the day. Insulin supports the transport of blood glucose into the body cells where the glucose is metabolized to produce energy. Regular insulin can be injected prior to the meal to decline the postprandial ascend in glucose levels. Ultra-fast acting insulin commences to act four to seven minutes and lasts for near three hrs. Short acting insulin achieves systemic circulation in thirty minutes, peaks after around two to three hrs. and stays active for about three to six hrs. The word Lente comes from the Latin "lentos," denotation slow, or sluggish) insulin. The absorption rate of NPH insulin can be lowered by the extension of protamine to the insulin preparation. Long-acting insulins provide basal insulin content. Basal insulins inhibit hepatic gluconeogenesis to suppress sugar levels from ascending during the fasting state in insulin-defective patients. Aid patients with type 1 diabetes, basal insulins furthermore inhibit ketogenesis [20,21].

Conclusion

Diabetes mellitus can be a complex disorder described by chronic progressive metabolic disorder and chronic hyperglycemia arising from dysregulation of carbohydrate, lipid and protein metabolism. HIV protease inhibitors may bind to proteins target, which regulate lipid metabolism, eventually cause the elevation of circulating fatty acids that could interfere with insulin signaling and compete with glucose cycle intermediates. Severe complications such as nephropathy, retinopathy, neuropathy and cardiovascular diseases resulted from chronic hyperglycemia. There are three preponderance components of diabetes mellitus management which are diet and exercise, oral antidiabetic medications and insulin treatment. Thiazolidine can improve glycemia by lowering the skeletal muscle insulin resistance and keeping pancreatic beta cell function with different mechanism of action such as ameliorating peripheral intake and use of glucose in muscle and fat, eventually lowering liver glucose release.

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