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Management of Diabetes Mellitus, Risk Factors and Complications

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ABSTRACT

Chronic progressive metabolic problem and chronic hyperglycemia caused by a dysregulation of protein, lipid, and carbohydrate metabolism are two symptoms of diabetes mellitus, a complicated condition. Verapamil belongs to a group of calcium channel blockers that are not dihydropyridines. It works by preventing calcium from entering beta cells' cytoplasm, preventing the second phase of insulin release driven by glucose, as well as sulfonylurea and glucagon. Nephropathy, neuropathy, and retinopathy are examples of microvascular consequences of diabetes mellitus (cardiovascular and cerebrovascular disease). In both kinds of diabetes mellitus, higher urine albumin excretion (proteinuria) or decreased kidney glomerular filtration rate are symptoms of diabetic nephropathy, a microvascular consequence. By lowering patients' blood sugar levels and reducing their risk of cardiovascular disease, diabetes mellitus therapy aims to reduce mortality, delay the onset of disease complications, and slow the disease's progression. Metformin increases peripheral glucose utilization, liver, muscle, and adipose tissue sensitivity to insulin, inhibits gluconeogenesis, and reduces glucose absorption from the gastrointestinal system.

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

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INTRODUCTION

Chronic progressive metabolic problem and chronic hyperglycemia, which result from carbohydrate, lipid, and protein metabolism dysregulation, are two symptoms of diabetes mellitus, a complicated condition. Over 463 million people worldwide suffer from diabetes mellitus, a serious health problem [1]. The two most prevalent forms of diabetes mellitus are covered in turn below. The rapid pace of beta cell failure that characterizes type 1 diabetes mellitus can be traced to an absolute lack of insulin hormone secretion. Type 1 diabetes mellitus frequently manifests itself in pediatric patients, while it may also affect adults [2]. Type 2 diabetes mellitus is more frequently caused by peripheral insulin resistance, relative insulin hormone deficiency, or insufficient insulin secretion by pancreatic beta cells. The resistance to insulin then results in elevated plasma levels of free fatty acids and proinflammatory cytokines, which ultimately reduces glucose transport into muscle cells, increases hepatic glucose production, and speeds up fat breakdown [3]. Insulin resistance in skeletal muscle, the liver, and adipose tissue is the primary cause of type 2 diabetes mellitus and eventually hastens the death and failure of pancreatic beta cells [4].

RISK ELEMENTS FOR TYPE 2 DIABETES

The commonly recognized risk factors for diabetes mellitus include family history, obesity, race/ethnicity, age increment greater than or equal to forty years, prior notable fasting glucose impairment or impairment of glucose tolerance, hypertension, hyperlipidemia, and history of gestational diabetes mellitus [1]. The following sections each cover a different medicine that can cause diabetes [5]. Thiazide diuretics result in hypokalemia, which eventually impairs insulin production as a result of potassium depletion. Thiazides may also stop proinsulin from converting to insulin. Because of their increased free fatty acid mobilization, thiazide diuretics may also hasten the development of insulin resistance. Verapamil belongs to a group of calcium channel blockers that are not dihydropyridines; it inhibits calcium uptake into beta cells'

cytosol, preventing the second phase of insulin release driven by glucose, as well as sulfonylurea and glucagon. Through weight growth, beta adrenoceptor blockers can partially counteract the development of insulin resistance, and beta blockers also stop the release of insulin from pancreatic beta cells.

By itself, human immunodeficiency virus protease inhibitors produce peripheral lipodystrophy, hyperlipidemia, and insulin resistance in the muscles. They also stop proinsulin from being converted to insulin. HIV protease inhibitors may bind to proteins that control lipid metabolism, leading to an increase in circulating fatty acids that may disrupt insulin signaling and compete with intermediates of the glucose cycle. By raising hepatic glucose output, beta adrenoceptor agonists can trigger insulin secretion, which then leads to hyperglycemia. By inhibiting ATP-sensitive potassium channels, fluoroquinolones stop the release of insulin. Because of increased free fatty acid mobilization, niacin increases skeletal muscle resistance to insulin. Several implantable hormonal contraceptives may be to blame for the changes in carbohydrate metabolism, including impaired glucose tolerance and increased insulin resistance. Oral contraceptive pills and estrogen replacement therapy can lower insulin sensitivity in women without diabetes taking some contraceptives. Through post-receptor pathways, glucocorticoids can increase the production of glucose and decrease the sensitivity of peripheral and hepatic tissues to insulin. The use of atypical antipsychotics, particularly clozapine and olanzapine, alters receptor binding properties, increasing insulin resistance whereas conventional antipsychotic drugs can occasionally cause hyperglycemia. Phenothiazines stop the pancreatic beta cells from releasing insulin.

COMPLICATIONS FROM TYPE 2 DIABETES

The most prevalent risk factors for the development of diabetes mellitus complications are gender, long duration of diabetes mellitus, poor and inadequate glycemic control, negative attitude toward diabetes mellitus, poor treatment adherence, and low illness knowledge [6]. Chronic hyperglycemia can lead to serious consequences as nephropathy, retinopathy, neuropathy, and cardiovascular disorders. Chronic exposure to elevated blood sugar levels can cause complications of diabetes mellitus by impairing insulin metabolism and biological macromolecules such carbs, lipids, proteins, and nucleic acids. The most significant cause of morbidity and mortality in the world today is diabetes mellitus complications [7]. Nephropathy, neuropathy, and retinopathy are examples of microvascular consequences of diabetes mellitus (cardiovascular and cerebrovascular disease). In both kinds of diabetes mellitus, higher urine albumin excretion (proteinuria) or decreased kidney glomerular filtration rate are symptoms of diabetic nephropathy, a microvascular consequence [8].

Indicated by a non-inflammatory deterioration of the structure and function of peripheral somatic or autonomic nerves brought on by metabolic-vascular pathology, diabetic neuropathy is a microvascular consequence [9]. One of the most severe microvascular consequences caused by persistently high blood sugar levels, diabetic retinopathy is almost always present in the middle and late stages of both kinds of diabetes mellitus. Blindness, vitreous hemorrhage, and severe visual impairment are all potential outcomes of diabetic retinopathy [10]. Hyperglycemia and disturbed lipid metabolism can also lead to macrovascular problems. Heart failure has become a significant and aggravating clinical and public health issue. It can be a serious macrovascular consequence. A higher risk of coronary heart disease is linked to diabetes mellitus. The leading cause of illness and mortality worldwide is coronary heart disease [11]. Diabetes mellitus increases the risk of heart disease, hypertension, and stroke because diabetics experience an earlier onset of atherosclerosis than non-diabetics. Diabetes mellitus patients frequently develop diabetic foot ulcers on the soles of their feet as a result of peripheral neuropathy or peripheral artery disease on all skin layers, necrosis, or inflammation [12].

Control of Type 2 Diabetes

By lowering the patient's blood sugar and reducing their risk of cardiovascular disease, diabetes mellitus therapy aims to reduce mortality, delay the onset of disease complications, and slow the disease's progression.

Diet and exercise, oral antidiabetic drugs, and insulin treatment make up the three mainstays of diabetes mellitus management [13].

PHARMACEUTICAL-FREE MANAGEMENT

Correcting any associated blood lipid abnormalities, enabling adequate glycemic control with blood glucose levels, guaranteeing weight control, and meeting nutritional needs are the goals of dietary management of diabetes mellitus. All people with diabetes mellitus should receive medical nutritional treatment, and crucial therapy is advised for those who have the disease. To reach and maintain a healthy body weight, people with type 1 diabetes should concentrate on managing their insulin administration through a balanced diet. In order to promote weight loss and prevent complications, type 2 diabetes mellitus typically requires calorie restriction [14]. In most people, physical activity, especially aerobic exercise, improves glycemic management and insulin sensitivity, lowers cardiovascular risk factors, aids in weight loss, and improves overall wellbeing. At least 150 minutes per week of low intensity exercise are required to meet physical activity goals [15].

MEDICATIONS MANAGEMENT

Oral hypoglycemic drugs fall into four categories: biguanides, which inhibit gluconeogenesis in the liver and contain metformin; insulin secretagogues, which increase insulin production and contain sulfonylureas; insulin

sensitizers, which improve the sensitivity of peripheral tissues to insulin; and alpha glucosidase inhibitors, which contain acarbose and miglitol. Metformin increases peripheral glucose utilization, liver, muscle, and adipose tissue sensitivity to insulin, inhibits gluconeogenesis, and reduces glucose absorption from the gastrointestinal tract [13]. With multiple mechanisms of action, such as improving peripheral glucose uptake and usage in muscle and fat, thiazolidine can lower skeletal muscle insulin resistance and maintain pancreatic beta cell function, ultimately lowering liver glucose release. Sulfonylurea, including glipizide, glyburide (glipalamide, glimepiride), function by promoting insulin production from the pancreatic insulin-releasing beta cells and reducing insulin resistance in peripheral target tissues by a small amount (muscle, fat).

Their binding results in the inhibition of ATP dependent potassium channel channels, which changes the resting potential of the cell, causing calcium influx and stimulating insulin release [17]. Their receptor is a component of the ATP dependent potassium channel in the pancreatic beta cells. As sulfonylureas, meglitinide analogues act as an insulin secretagogue. Meglitinide analogues differ structurally from sulfonylureas, but they share a lot of the same mechanisms of action (they control ATP-dependent potassium channels in pancreatic beta cells) as sulfonylureas because they stimulate the release of insulin from pancreatic beta cells by binding to a different binding site on the "sulfonylurea receptor" [18]. Acarbose and miglitol are components of alpha glucosidase inhibitors. Inhibitors of intestinal -glucosidase enzymes slow down the rate at which carbohydrates are digested, giving rise to the option of lowering postprandial. Alpha glucosidase inhibitors work by inhibiting alpha glucosidase, an enzyme that sticks more complex carbohydrates into sugars and limits the digestion and absorption of carbohydrates [19]. This enzyme is present in brush border cells of the small intestine. By keeping the plasma glucose value in an ideal range throughout the day, insulin promotes glucose homeostasis. Insulin aids in the movement of blood sugar into the cells of the body, where it is converted into energy. Before eating, regular insulin can be administered to prevent the postprandial rise in blood sugar. Insulin with ultra-fast action starts working four to seven minutes after injection and lasts for about three hours. Short-acting insulin begins to circulate throughout the body in thirty minutes, peaks after two to three hours, and remains active for three to six hours. The Latin word "lentos," which means slow or sluggish in English, is where the term "lente" originates. By adding protamine to the insulin formulation, the rate of NPH insulin absorption can be reduced. Basal insulin content is provided by long-acting insulins. In patients with insulin deficiency, basal insulins prevent hepatic gluconeogenesis to prevent rising blood sugar levels during the fasting state. Basal insulins also prevent ketogenesis, helping individuals with type 1 diabetes [20,21].

CONCLUSION

Chronic progressive metabolic problem and chronic hyperglycemia caused by a dysregulation of protein, lipid, and carbohydrate metabolism are two symptoms of diabetes mellitus, a complicated condition. HIV protease inhibitors may bind to proteins that control lipid metabolism, leading to an increase in circulating fatty acids that may disrupt insulin signaling and compete with intermediates of the glucose cycle. Chronic hyperglycemia can lead to serious consequences as nephropathy, retinopathy, neuropathy, and cardiovascular disorders. Diet and exercise, oral antidiabetic drugs, and insulin therapy make up the three mainstays of diabetes mellitus management. With multiple mechanisms of action, such as improving peripheral glucose uptake and usage in muscle and fat, thiazolidine can lower skeletal muscle insulin resistance and maintain pancreatic beta cell function, ultimately lowering liver glucose release.

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