Editorial

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Role of Oral Antidiabetic Agents in Diabetes Treatment

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Description

The main thing in treatment of type 2 diabetes includes diet, modifications of lifestyle and oral hypoglycemic therapy. The major aims are not only to reduce the weight of obese patients and to improve glycemic control but also to decrease the risk of cardiovascular diseases, improve compliance and reduce adverse effects. The specific medications used in patients with type 2 diabetes are determined by clinical judgment about the likely balance between insulin resistance and beta cell impairment. Preventing Hypoglycemia and improving compliance is attained through less frequent dosing are the other important targets.

Role of oral antidiabetic agent therapy

Oral Antidiabetic Agents (OADs) follow diet and exercise in the management in an individual with frank diabetes mellitus. While trying to use OADs in the control of diabetes mellitus, one should also try and attempt to:

- To conserve islet cell function and thereby delay subsequent insulin use
- A drop down in the prevalence of hypoglycemic episodes
- Improve patient compliance with medications (attempting to reduce the frequency of dosing)
- Consider the cost factor when the affordability of the patient comes into play.

Sulphonylureas

Sulphonylureas (SUs) augment insulin response by binding to the receptor on the extracellular domain of ATP modulated K' channels and causes its closure. This depolarizes the membrane leading to the entry of calcium through Voltage Dependent Calcium Channels (VDCC) and the resultant increase in intracellular Car concentrations lead to fusion of insulin-containing vesicles to the plasma membrane and subsequent rapid exocytosis of insulin from beta cells. In addition to this, some SUs are now known to directly activate the cAMP sensor Epac2 (exchange protein activated by cAMP 2) to induce Rap 1 signalling which can induce the exocytosis of insulin-containing vesicles. The distinct effects of various SUs appear to be because of their differential actions on Epac2/Itap 1 signalling as well as KATP channels. Kir6.2/SUR2A is present in the cardiac cell membrane (sarcolemmal) channel which closely mimics the SUR 1.

First-generation SUs like glibenclamide prevent cardioprotection by interacting with mitochondrial KATP channels and it is induced by ischemia, Incontrast second generation SUs are thought not to interfere with ischemic preconditioning.

Prevalence of hypertension in diabetes

Type 1 Diabetes: Incidence rises from 5% at 10 years to 33% at 20 years and 70% at 40 years. Increase in BP is closely related to onset of microalbuminuria and increases progressively as the renal disease progresses.

Type 2 Diabetes: 40-50% is already hypertensive at the diagnosis of diabetes. In those with hypertension, approximately 50% had hypertension even before the onset of microalbuminuria.

Pathogenesis

1. In type I diabetes, hypertension usually reflects the onset of diabetic nephropathy. Hypertension substantially increases the risk of both macrovascular and microvascular complications, including stroke, coronary artery disease and peripheral vascular disease, retinopathy and nephropathy.

2. In type 2 diabetes, hypertension is often present as part of metabolic syndrome of insulin resistance which also includes dyslipidemia and central obesity.

The mechanisms may be any one of the below.

- The accompanying insulin resistence and hyperinsulinemia causes activation of sympathetic nervous system, renal sodium retention, vascular smooth muscle cell remodeling and vasoconstriction
- Glycosuria leads to sodium retention because glucose and sodium are cotransported across the tubule by sodium glucose cotranspoters.
- Increased glycation of the vessel wall proteins and later arterial atheromatous disease leading to increased arterial thickness