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Managing Type 2 Diabetes in the 21st Century: From Oral Agents to Injectables



Audience	Dietitians and Nutrition: Diabetes
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Hours	2.00 contact hours
Level	Level II
Passing Grade	70%
Test Retries	Unlimited

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After completion of this course the participant should be able to:

1. Describe the pathophysiology of type 2 diabetes.
2. Explain the mechanism of action of sulfonylureas, meglitinides, biguanides, alpha-glucosidase inhibitors, thiazolidinediones, dipeptidylpeptidase-4 (DPP-4) inhibitors, and incretin mimetics.
3. Explain two differences between DPP-4 inhibitors and incretin mimetics.
4. Describe the clinical use of sulfonylureas, meglitinides, biguanides, alpha-glucosidase inhibitors, thiazolidinediones, DPP-4 inhibitors and incretin mimetics.
5. Describe two indicators for the use of insulin.
6. Differentiate insulin preparations based upon type, onset of action, peak and clinical use.

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Course Sample:

Introduction

Forty years ago the only ways to treat diabetes were the now obsolete forms of insulin from cows and pigs, and sulfonylureas that stimulate insulin secretion from the beta cells of the pancreas. Both of these therapies caused dangerous hypoglycemic reactions and excessive weight gain. But the past twenty years have been marked with the rapid development of new and more effective treatments for diabetes. We now have oral agents that target the specific metabolic abnormalities of type 2 diabetes – insulin resistance, insulin deficiency and hepatic glucose over production. Patients are benefiting from improved forms of insulin, a range of oral medications to control blood glucose and reduce the need for insulin, and new drugs that may not only control blood glucose, but also strengthen the activity of patients' own insulin producing beta cells¹.

It is well documented in the scientific literature that much of the morbidity and mortality associated with diabetes can be prevented or delayed with tight control of blood glucose levels – HbA1c less than 7. The medications of the 21st century will enable practitioners to do just that – prevent and delay the morbidity and mortality of diabetes.

Pathophysiology of type 2 diabetes

Type 2 diabetes previously called "non insulin dependent diabetes" or "adult onset diabetes" is caused by three metabolic defects: insulin resistance, decreased insulin secretion and an increase in glucose production by the liver – all of which result in chronic hyperglycemia.

Insulin regulates blood glucose levels by controlling glucose uptake by muscle, fat, and liver cells. In insulin resistance these cells are insensitive or resistant to the action of insulin resulting in decreased glucose uptake by the muscle and fat cells, increased fat breakdown (lipolysis) and increased hepatic glucose production (glycogenolysis). In type 2 diabetes insulin resistance usually predates the development of chronic hyperglycemia² and can occur decades before the onset of overt diabetes. Early in the disease process the beta cells of the pancreas are able to compensate and keep glucose levels within a normal range by increasing circulating insulin. Over time the beta cells will gradually lose the ability to compensate leading first to impaired fasting glucose (IFG), and ultimately to overt diabetes³.

IFG, also known as pre-diabetes is characterized by a fasting blood glucose level which is 100 to 125 milligrams per deciliter (mg/dl) after an overnight fast. This level is higher than normal but not high enough to be classified as diabetes. Studies have shown that progression to diabetes among those with pre-diabetes is not inevitable. Weight loss, increased physical activity and in some cases pharmacologic intervention can prevent or delay diabetes onset and return blood glucose levels to normal⁴.

The progression from IFG to overt diabetes is characterized by worsened insulin resistance, eventual failure and destruction of the beta cells and increased glucose production by the liver. Understanding the progression of type 2 diabetes is essential in successful management of type 2 diabetes. Today we have several different classes of diabetes medications that are effective at different stages of the disease. Primary care providers must formulate treatment regimens that reflect the pathological differences between these defects. The optimal pharmacologic intervention at the right time will improve blood glucose levels and help to prevent the morbidity and mortality associated with diabetes³.

Oral Antihyperglycemic Agents

Understanding the progression of type 2 diabetes from insulin resistance to mild postprandial hyperglycemia to overt diabetes is essential in successful management of type 2 diabetes in the 21st century. Different classes of antidiabetes medications have been found effective at different stages of the disease. There are now six classes of oral diabetes medications- sulfonylureas, thiazolidinediones, biquanides, alpha-glucosidase inhibitors, meglitinides, and dipeptidyl peptidase-4 (DPP-4) inhibitors.

Sulfonylureas:

Sulfonylureas also called insulin secretagogues, lower blood glucose levels by increasing insulin secretion from the beta cells of the pancreas, an effect dependent upon functioning beta cells in the pancreas⁵. In 1958 the first generation of sulfonylureas came to market – actohexamide, chlorpropamide, tolazamide and tolbutamide. With the exception of chlorpropamide these drugs are rarely used today⁶ and have been replaced by second generation sulfonylureas – glipizide, glyburide, glimepiride. Second generation secretagogues are more potent, provide more predictable results with fewer adverse effects and more convenient dosing. Common adverse effects that can be seen with this class of medication include hypoglycemia, gastrointestinal disturbances, rash, and weight gain⁷. Most sulfonylureas are metabolized by the liver and cleared by the kidney; therefore they should be used cautiously in patients with hepatic or renal impairment. Sulfonylureas are approved for use as monotherapy and in combination with most other oral drug classes and insulin; they are not approved for use in combination with glinides. Dosing of sulfonylureas varies by agent but maximum glucose lowering effect usually plateaus at half maximum recommended dose. Predominate impact of sulfonylureas is on fasting blood glucose and reduces HbA1c levels by 1% to 2%⁵.

The first generation:

- Chlorpropamide
 1. Diabinese® is made by Pfizer . It was FDA approved in October 1958⁹.
 2. Administered orally in a single 250 mg daily dose. Older patients should be started on smaller amounts of Diabinese, in the range of 100 to 125 mg daily.
 3. Should be given in the morning with food. High doses of nicotinic acid greater than 3 gm can cause elevated glucose .
 4. Gastrointestinal disturbances are the most common adverse reactions; nausea, diarrhea, vomiting, anorexia and hunger.
 5. Diabinese is contraindicated in patients with type 1 diabetes, diabetic ketoacidosis and hypersensitivity to any component of Diabinese⁸.

The second generation:

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