

## Case Study Responses

### **Expert Opinion provided by Derek LeRoith, MD, PhD**

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Note: Readers are encouraged to visit [www.InsulinJournal.com](http://www.InsulinJournal.com) to review the details of a Case Study published in the April 2007 issue of *Insulin*.

This was the case of a 26-year-old black woman with type 2 diabetes mellitus (DM). She had presented for further treatment after being told by her primary care physician that her DM was poorly controlled.

**Question 1.** Which of the following therapies would be inappropriate for treating this patient's DM?

**Answer:** c. Rosiglitazone.

This patient's major medical conditions included obesity, edema, hypertension, and postprandial hyperglycemia. All drugs except thiazolidinediones would be appropriate. The thiazolidinediones (such as rosiglitazone) act by increasing insulin sensitivity. Although this improves fasting glucose levels, it fails to adequately treat postprandial hyperglycemia and can cause fluid retention, which is a problem for this patient. A high-fiber diet delays the absorption of glucose and slows gastric emptying, thereby allowing better timing between insulin release and the peak blood glucose after meals. Sitagliptin, a member of the dipeptidyl peptidase-IV class of drugs, inhibits the enzyme that degrades incretin hormones (eg, glucagon-like peptide-1 [GLP-1], glucose-dependent insulinotropic polypeptide). This has a significant effect on lowering postprandial glucose levels. Exenatide, a GLP-1 analogue, also delays gastric emptying and alters islet cell function by decreasing glucagon secretion and raising insulin levels.

**Question 2.** Which of the scenarios below would *not* classically present with postprandial BG levels similar to this patient's levels?

**Answer:** d. A 49-year-old man with sarcoidosis.

Sarcoidosis is not typically associated with postprandial hyperglycemia. Due to changes in stress hormone levels (cortisol), negative emotions (anxiety, sadness, and fear) raise postprandial glucose levels (by increasing insulin resistance and decreasing insulin secretion) and could certainly be expected in a young woman with long-standing anxiety. Hyperthyroidism enhances the rate of gastric emptying, thus causing postprandial hyperglycemia. A history of gastrointestinal surgery or gastroparesis can cause erratic blood glucose levels (especially postprandially with hyperglycemia) due to changes in food absorption.

**Question 3.** Given this patient's cardiac risk profile, what change in medication would you most likely make at this visit?

**Answer:** a. Discontinue atorvastatin. Start rosuvastatin and titrate to LDL goals. Add omega-3 fatty-acid supplementation.

The patient's lipid profile with atorvastatin 10 mg was nowhere near the ideal goal of a low-density lipoprotein (LDL) level <70 mg/dL. Her triglyceride levels were also high. Add to these findings her elevated C-reactive protein level (which increases her myocardial risk), and this patient has a very high-risk profile for future cardiovascular events. Rosuvastatin has been shown to reduce LDL better than doubling atorvastatin and is the best option listed for lowering the patient's LDL to goal level. Adding omega-3 fatty-acid supplements will address the triglyceride issue and should raise her high-density lipoprotein levels as well, thereby significantly reducing her cardiac risk. Fenofibrate would lower triglycerides, and a high-fiber diet might modestly help lower LDL, but a satisfactory reduction of LDL from 151 mg/dL is unlikely. Niacin would lower triglycerides sufficiently well, but LDL levels would still not be at goal by adding this therapy alone. Although not mentioned, the patient would also benefit from use of an angiotensin-converting enzyme inhibitor, which would lower her blood pressure and preserve renal function.

**Question 4:** In reference to Question 1, what other treatment options might you offer this patient to control her BG levels, and why?

**Answer:** The rapid-acting insulins—insulin lispro, insulin aspart, or insulin glulisine—would be good options. These may be taken within 15 minutes before meals to maximize patient flexibility. Regular insulin can also be used, but this should be taken 0.5 to 1 hour before the meal to time the insulin peak with the glycemic rise. Other acceptable therapies for treating postprandial hyperglycemia include repaglinide and nateglinide, which act by stimulating the pancreas to produce insulin based on the rise in postprandial glucose levels.

Some agents would not be appropriate options. Because acarbose has been tried with poor results, miglitol would likely have the same effect and is not a good alternative. The longer-acting insulins (neutral protamine Hagedorn, insulin detemir, or insulin glargine) would not adequately address the patient's postprandial hyperglycemia.

Readers are invited to consider a new Case Study (see page 144) and submit responses to [www.InsulinJournal.com](http://www.InsulinJournal.com) before the deadline.