

DR. BERNSTEIN'S DIABETES SOLUTION

A COMPLETE GUIDE TO ACHIEVING NORMAL BLOOD SUGARS

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The Difficult Case of Meyer K.

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In 1997, Meyer K. came to me as a slim 82-year-old retired widower with type I diabetes since the age of 51. Because of diminished cognition, unsteady gait, reduced vision and other problems caused by his long history of very high blood sugars, he was cared for by a younger gentleman. His care giver did not speak English and was not skilled in elementary arithmetic. He measured the insulin, and Meyer gave himself the injections.

The pair were brought to me by Meyer's children because of his bouts of severe hypoglycemia with loss of consciousness and his hospitalizations for diabetic ketoacidosis.

At his first visit, Meyer's HbA1c was 10.4%. This corresponded to an average blood sugar of approximately 320 mg/dl over the prior four months.

Meyer admitted to feeling depressed for several years and had been suffering from chronic diarrhea for three years. This condition would frequently attack without enough warning for him to reach the toilet, and he did not respond to the ordinary anti-diarrhea medications that he took several times a day. Because of this condition, he rarely left home for fear of an "accident." he also complained of frequent belching during and after meals.

A Plethora of Problems

Physical examination revealed a number of long-term diabetic complications. Meyer suffered from cataracts, periodontal disease, missing teeth, peripheral sensory neuropathy, postural hypertension, severely impaired short-term memory, glaucoma, double vision in all directions of gaze, severe sympathetic neuropathy in his feet, diminished pulsatile circulation in his left ankle, the classic "claw" shape of "diabetic feet" and weak/wasted muscles in the hands and feet. Most important was severe impairment of heart rate variation during deep breathing. In nondiabetics his age, the vagus nerve slows the heart rate by about 25 percent from inhaling to exhaling. Meyer's only slowed by two percent.

Questions:

1. What could be causing Meyer's frequent belching and poor BG control?
2. What is the ideal treatment regimen for Meyer considering his obstacles to treatment?

Solution #1:

Since the vagus nerve is also the major mediator of digestion, and in particular stomach emptying, it was clear that Meyer must also be suffering from gastroparesis, a weakness or paralysis of the muscles of the stomach. This extreme neuropathy of his vagus nerve probably explained his frequent belching.

Meyer's gastroparesis predicted that it would be nearly impossible to normalize his blood sugars because gastroparesis renders stomach emptying and digestion of meals unpredictable. You never know what amount of a meal will eventually be converted to blood sugar, so you never know when and how much insulin would be needed to cover a given meal on a given day. As a result, many gastroparetics who take insulin will experience dangerously low blood sugars after a meal fails to leave the stomach, only to experience very high blood sugars eight, twelve or twenty hours later, after the stomach finally empties and the insulin taken for the meal is no longer working. One could easily cope with this if it happened the same way every day, but it doesn't. For further information on diagnosing and coping with this condition, see chapter 21 of my book, *Dr. Bernstein's Diabetes Solution*, Little, Brown, 1997.

Solution #2:

The treatment plan for Meyer included a number of important elements. First, we had to deal with the disabling diarrhea that worried him more than any of his other problems. This was easy.

I gave him a low dose clonidine skin patch, the standard treatment for diabetic diarrhea. He was instructed to change the patch weekly. It immediately reduced the frequency and severity of episodes (explosions) so we soon increased the dose to an intermediate strength patch. This worked almost perfectly, and after eight months of improved sugars, the diarrhea was gone. Initially, his blood pressure was normal, and although clonidine is marketed as a powerful antihypertensive agent, he did not experience any symptomatic worsening of his prior blood pressure drops when standing. In my experience, this is usually the case when clonidine skin patches are used.

Simultaneously, while addressing the diarrhea, we had to work on improving Meyer's blood sugars. This would not be an easy job because of the gastroparesis. The first step was to negotiate a meal plan wherein foods would be soft and easily chewed but every low in carbohydrate.

These foods included whole milk unflavored yogurt sweetened with stevia and flavored with liquid baking extracts (chocolate, vanilla, banana, etc.), eggs, fish, chopped meat and chopped, cooked, low-carbohydrate vegetables. Carbohydrate total six grams for breakfast and 12 grams for lunch and inner.

The low carbohydrate diet was essential to eliminating Meyer's episodes of severe hypoglycemia as it allows for lower doses of injected insulin—more in keeping with the levels non-diabetic produce.

Next an insulin regimen had to be devised. The plan was to initially under-estimate insulin needs, but to correct blood sugar levels promptly so that ketoacidosis could no longer occur. Then insulin doses could be increased slowly based upon blood sugar records.

Our insulin regimen for type I diabetes emulates the manner and amounts of insulin production in non-diabetics. People without diabetes make small amounts of insulin all day long, even when fasting, to prevent the liver from converting bodily protein (like muscles) to glucose. This is called basal insulin production.

They also make small boluses of insulin while meals are being digested to facilitate delivery of glucose derived from food into tissues where it is used for energy or to build fat.

We inject long acting insulin (usually ultralente) on waking and at bedtime to emulate basal insulin production. We inject rapid acting insulin (regular or lispro) from about 15 minutes (for lispro) to about 40 minutes (for regular) before meals to emulate the boluses made by non-diabetics. Ideally the rapid insulin should begin to lower blood sugar at the same time as the meal starts to raise blood sugar. With the low carbohydrate diet and small doses of rapid acting insulin, the net result is usually little or no change in blood sugar before, during or after a meal. This regimen works elegantly if you don't have gastroparesis. It obviously had to be modified for Meyer.

I put Meyer on our standard insulin regimen but with his meal-time boluses of regular insulin given at the end of the meal rather than the usual 40 minutes before eating. Gastroparesis can cause a feeling of fullness early in a meal. This, combined with the fact that Meyer is an especially slow eater, meant that Meyer was often unable to finish a given meal.

With this insulin regimen, he could, on these occasions, reduce his post-meal regular insulin dose accordingly. In order to prevent hypoglycemia from occurring on those random occasions when meals are especially slow in emptying, I kept his mealtime insulin doses below those usually required by non-gastroparetics to cover similar meals.

This means that when a meal eventually finishes emptying, his BGs can increase considerably. To correct such increases, I asked him to check BGs before each meal and at bedtime. Elevations about 115 mg/dl would be brought down to 100 mg/dl with an injection of lispro. Based on his weight, one-quarter unit of lispro lowers his BGs by 15 mg/dl. Indeed this calculation turned out to be valid. Note: I never prescribe Lispro to cover meals for people with gastroparesis. It just works too fast for the slow digestion.

I also had Meyer cover levels below 100 mg/dl with calibrated amounts of oral glucose tolerance test solution.

Since neither Meyer nor his care giver could be relied upon to perform the usual calculations, I gave them a table of doses of glucose (teaspoons or tablespoons) and lispro for every possible BG between 0 and 300 mg/dl (in five mg/dl increments).

After several months of clonidine skin patches and improved blood sugars Meyer's diarrhea stopped. At this point, I prescribed one tablespoon of cisapride suspension about an hour before

meals in the hopes of easing his gastroparesis. Since this medication can cause diarrhea in people without gastroparesis, I elected to wait until his diarrhea was gone. The effectiveness of this product in people with severe gastroparesis is uncertain. In any event, Meyer no longer complains of belching and discontinued his habit of chewing antacid tablets every hour or so. Meyer was then started on 4.5 units of Ultralente insulin on waking, five units of regular insulin after breakfast, 3.25 units of regular after lunch, six units of regular after dinner, and 8.75 units of ultralente at bedtime. The high bedtime dose is to cover that part of his dinner that leaves his stomach while he is sleeping. This is necessary even though his bedtime is five hours after he finishes dinner. His care giver measures out small doses of lispro insulin, according to his table, to bring elevated BGs down to a target of 100 mg/dl.

Summary:

Meyer's bouts of severe hypoglycemia are a thing of the past—not one episode since the new regimen. His most recent HbA1c was 6.6%, corresponding to a four-month average blood sugar of 164 mg/dl.

He no longer feels depressed and his gait and strength have improved. His short-term memory, measured by repeating six digits in reverse order, went from only one digit at his second visit to all six digits currently.

Twenty-four hour urine glucose levels declined from 52 gm to one gm. His kidney function, measured by creatinine clearance, improved by 20 percent from 52 to 71 ml/min. This reflected a 20 percent drop in serum creatinine. Finally, his serum fibrinogen level, a major risk factor for heart disease, stroke, retinopathy and kidney disease, dropped from 396 to a normal level of 272. Meyer is no longer chronically tired and now goes out for shopping, club meetings, social affairs and so on. His children no longer dread the specter of ongoing emergencies.

I've heard a number of physicians comment that efforts to tightly control blood sugars would be wasted on the elderly. What do you think?

Addendum to the Case of Meyer K.

At his last visit in February 1999, Meyer, now 85 years of age, was alert and enjoying his new dog. His former ghostly pallor was replaced by a natural pink tone. His diabetic diarrhea is totally gone, and he no longer needs to be treated with clonidine. His frequent belching has resolved. His heart rate variation on deep breathing (R-R Interval Study), a quantitative test for autonomic neuropathy has improved nearly sixfold from 2 percent to 11 percent. Normal for his age is about 25 percent. If such improvement continues, his gastroparesis should be totally cured in another two to three years. His 24-hour urine glucose excretion dropped from 52 grams (about 17 packets of sugar) to 0.1 grams, which is normal.

Meyer's HbA1c has dropped from 10.4% to 5.6%. This is the approximate equivalent to a reduction in four-month blood sugar average from 316 mg/dl to 124 mg/dl. In spite of this, he has not had even one of the frequent hypoglycemic episodes that had plagued his life for many years.

About the Author:

Dr. Bernstein has had type I diabetes for 52 years and is in better health than most non-diabetics his age. He invented self-monitoring of blood sugar in 1969 and multi-dose intensive insulin therapy in 1970. Because he was unable to convince the diabetes medical establishment that BGs could and should be normalized, he gave up his business career and entered medical school in 1979 at the age of 45. In 1998, he was granted a US Patent for a new approach to the treatment of obesity. Dr. Bernstein limits his medical practice in Mamaroneck, New York, to the treatment of diabetes and obesity. Most of his patients come from around the country and overseas for intensive training in methods for normalizing blood sugars. They are also given a physical exam and laboratory tests designed to expose early diabetic complications. Customized meal plans are negotiated with each patient and medications prescribed accordingly. After training, patients fax their BGs to Dr. Bernstein and he fine tunes their diet, medications and exercise over the telephone. Dr. Bernstein can be reached at (914) 698-7500. Dr. Bernstein's latest book *Dr. Bernstein's Diabetes Solution* was published by Little, Brown & Company in 1997.