Severe Diabetes with Remission

Report of a Case

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The purpose of this report is to describe another instance of diabetes with remission which bears a remarkable similarity to a case[1] recently reported by Harwood. Both were young adults who were not known to have diabetes prior to the abrupt onset of symptomatic diabetic ketoacidosis and both remitted fully. In addition to this, we should like to record an interesting feature which may be helpful in diagnosis, utilizing intravenous tolbutamide as a diagnostic test for diabetes. Since Harwood's review of the literature, which did not show any other case of remission after severe acidosis in adults, Peck, Kirtley and Peck[2] have reported such an instance. Because of the great rarity of apparent cure or complete remission it was deemed worth-while to report the following case.

CASE REPORT

The patient, a nineteen-year-old white airman, had been apparently in good health until the middle of May 1957. At that time he was stationed at Lowry Air Force Base, Colorado. In April 1957, he observed that he had started to become noticeably thirsty and was losing weight. Early in May, he developed profound symptoms of diabetes: increasing polydipsia, polyuria and polyphagia. On May 27, 1957, he reported for examination and his urine contained 4-plus sugar, 4-plus acetone and 4-plus diacetic acid. CO_2—combining power of the blood was 14 volumes per cent; a blood glucose was 510 mg. per 100 ml.; peripheral blood count and chest film were normal.

There was no family history of diabetes. The patient had entered the service Dec. 26, 1956, and at that time had no evidence of any disease. His physical findings were within normal limits except for apathy and drowsiness. He responded to regular insulin, and after two days was placed on 30 units of NPH and a restricted diet 180 CHO, 95 fat and 70 protein. Later, because of increasing glycosuria, the dose of NPH insulin was increased to 65 units daily. The fasting and two-hour postprandial blood sugar returned to normal in several weeks and gradually the dose was reduced to 45 units of NPH daily when he seemed fairly stable and experienced no hypoglycemic reactions. After one month he was transferred to Wright-Patterson Air Force Base Hospital for Physical Evaluation Board action. At the time of admission to the second hospital he appeared in excellent control with a fasting blood sugar of 86 mg. per cent. Never under any conditions at our hospital did he have a fasting blood sugar over 95 mg. per cent while on insulin and diet 180 CHO, 95 fat and 70 protein. Because of low blood-sugar values and because he at times felt somewhat shaky, insulin was gradually withdrawn over a period of three weeks. Twenty-four-hour urine collections were negative for glucose at this time.

Several weeks after management on diet alone, a glucose tolerance test was done; this was within normal limits (figure 2, Number 1); two tolbutamide[4] tolerance tests revealed a relatively flat response (figure 1, Numbers 1 and 2). According to Unger[4] the blood glucose in a normal individual should drop 40 to 50 mg. per cent within the first twenty to forty minutes after intravenous tolbutamide.[4] This was confirmed in a control subject. Two glucose tolerance tests were done after the patient had been on a regular diet for several weeks (figure 2). It is interesting to note that in this period his glucose tolerance decreased. The patient at no time spilled any detectable sugar in his urine. These tests were carried out in a period of six weeks after admission to the Wright-Patterson Air Force Base Hospital, and at no time during these tests did the patient receive insulin. Tests for thyroid, adrenal and pituitary function were within normal limits (table 1).

After the patient was discharged from the hospital, it was

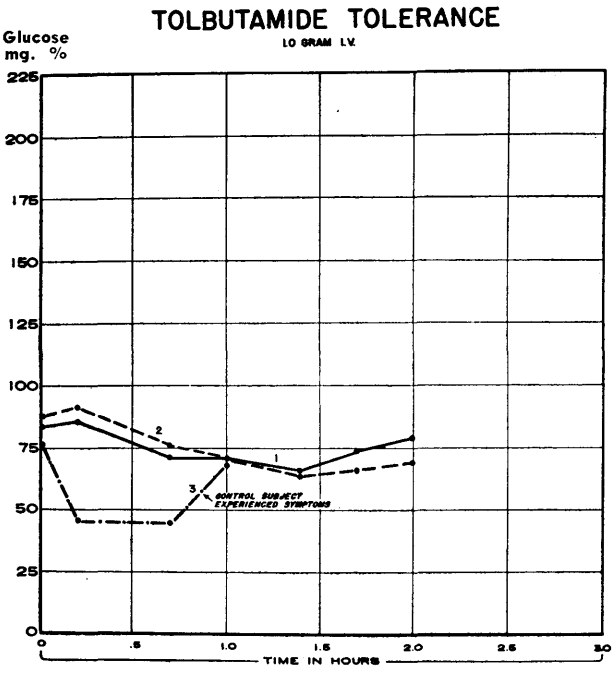

FIGURE 1

From the Department of Internal Medicine, United States Air Force Hospital, Wright-Patterson Air Force Base, Dayton, Ohio.

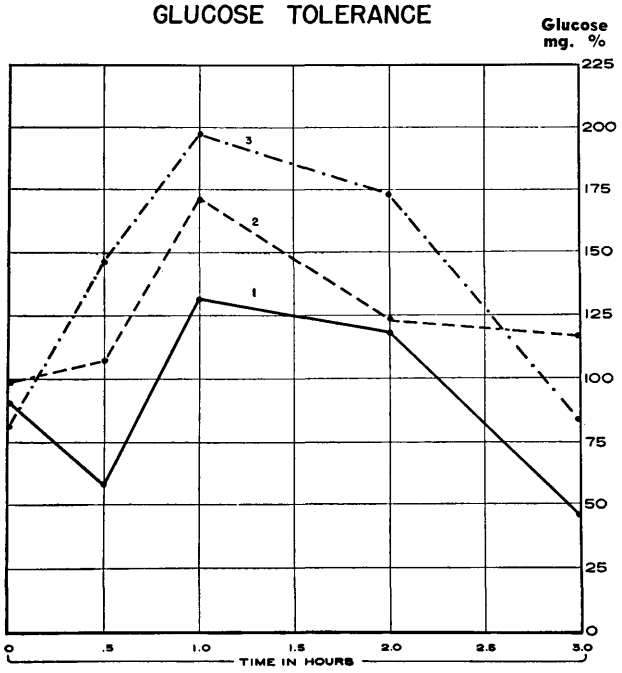

FIGURE 2

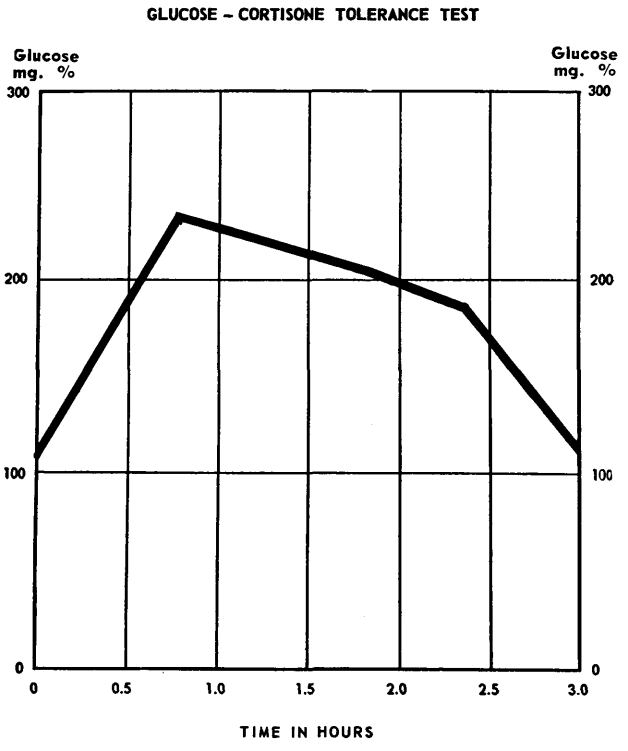

FIGURE 3

TABLE 1

Laboratory values

Protein bound iodine = 8.0 μg.
B.M.R. = (—6)
Cholesterol = 188 mg. per cent
A/G = 1.1 = 3.36/3.00, T.P. = 6.36

		Globulins per cent	Albumin per cent
Protein electrophoresis	Alpha one	3.8	52
	Alpha two	9.6	
	Beta	9.6	
	Gamma	25.0	

17-ketosteroids = 17.4 mg./24 hr.

suggested that a Fajans-Conn glucose-cortisone tolerance test be performed. The data appear in figure 3. It is obvious that according to the criteria of Fajans et al.[5] that this patient is a potential diabetic.

DISCUSSION

Joslin,[3] in his discussion of remissions, curability of diabetics and criteria for cures, feels that the tendency for diabetes is inborn and must remain so for life. In our case, there was no family history and no evidence for any precipitating factor for the onset of the diabetic ketoacidosis. Since we had a patient who was in remission as evidenced by the oral glucose tolerance, we thought it would be interesting to give him intravenous tolbutamide and see what the shape of his tolerance curve indicated. Unger by this means had been able to separate those diabetic subjects who have normal fasting blood sugars but have "diabetic" responses to the oral glucose test, from normal subjects because the latter have a sharp fall of 40 to 50 mg. per cent in blood glucose while the diabetics did not fall significantly. This decline was observed within ten to forty minutes. Our patient on two separate occasions had a flat curve. His glucose tolerance was normal when the first curve was done, but had decreased when the second curve was performed. This indicated that, even though the patient had an apparent remission, he is still a diabetic.

The use of an intravenous tolbutamide tolerance test has important implications in that it may help predict subjects who will develop diabetes in the future.

Several months after our patient had left the hospital, he developed lobar pneumonia and was ill seven days. During and after this infection there was no glycosuria and a fasting blood sugar obtained at the height of his disease was 95 mg. per 100 ml. Insofar as the stress of an infection usually aggravates existing diabetes we felt that this episode was significant in demonstrating the state of remission. Possibly if he continues for five years in such a manner the case could then be called a cure.

The Fajans-Conn provocative cortisone test and the tolbutamide tolerance test are both positive in this case and indicate that this patient still is a potential diabetic. Two fasting blood sugars determined under conditions without therapy or special diet one and one half years after the onset of diabetic ketoacidosis were 107 and 83 mg. per cent.

In the light of these tests most will agree with Joslin's view:

"The more I see of diabetes, the more strongly I think we should speak of remissions rather than of cures."

SUMMARY

1. A case of diabetic ketoacidosis with apparent remission has been described in a young male.

2. Severe infection did not bring on the symptoms of diabetes in the patient several months after he had an apparent remission from the diabetic acidosis.

3. Both the provocative cortisone and intravenous tolbutamide tolerance tests were positive for latent diabetes long after the patient was in a state of clinical remission.

SUMMARIO IN INTERLINGUA

Reporto De Un Caso De Remission in Diabete Sever

1. Es describite un caso de ceto-acidosis diabetic con remission apparente in un juvene masculo.

2. Un infection de grados sever non evocava le symptomas de diabete plure menses post le remission ab le acidosis diabetic.

3. Le test provocatori de cortisona e le test del tolerantia pro tolbutamido intravenose esseva positive pro diabete latente un longe tempore post que le patiente habeva attingite un stato de remission clinic.

ACKNOWLEDGMENT

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The delineation of the complete genetic and biochemical pattern of man that I have envisaged involves, of course, many difficulties; but it also offers many challenges and promises important rewards. To give just one example, it would be of the utmost value in the elucidation of the basic mechanisms underlying heart disease to be able to formulate the metabolic interrelationships that must exist between the lipids and the purines. The relative levels of uric acid, cholesterol, and phospholipids have been shown to be related to the development of atherosclerosis and coronary artery disease.[1] Although the metabolism of each of these substances may be independently controlled by the activities of known genes, as in gout, xanthomatosis, and the lipidoses, respectively, some basic, underlying, genetic mechanism must surely exist which enzymatically conditions their interacting biochemical activities. A step in this direction may have been taken with the recent demonstration that familial amyloidosis represents an inherited aberrancy in lipopotein metabolism.[2]

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