previous study^{7 15} must be due to different actions of the vitamin D metabolites in anticonvulsant osteomalacia.

It is well documented that vitamin D₃ in the liver is hydroxylated to 25-OHD₃, which is further hydroxylated in the kidneys to 1,25-dihydroxycholecalciferol.^{19 20} Though much less is known about vitamin D₂ we assume that it follows a similar pattern. Since vitamin D₂ and vitamin D₃ act differently in anticonvulsant osteomalacia but alike in classical osteomalacia it seems that the metabolisms of vitamins D₂ and D₃ are affected differently by anticonvulsant drugs. Furthermore, since 25-OHD₃ acts like D₃ in anticonvulsant osteomalacia then a mechanism other than induction of liver enzymes must play a role; if induction of liver enzymes alone were the cause then 25-OHD₃ would have an optimum effect in anticonvulsant osteomalacia, which apparently it does not.

Possibly some "posthepatic" enzyme systems concerned with the metabolism of vitamin D are specific for the vitamin D₃ molecular configuration and sensitive to anticonvulsant drugs. On this assumption the posthepatic metabolism of vitamin D₂ would be influenced by anticonvulsant drugs only mildly—that is, treatment of anticonvulsant osteomalacia with vitamin D₂ should reflect treatment of classical osteomalacia. It is well known that vitamin D treatment causes a positive calcium balance (an increase in B.M.C.) and also that the serum calcium concentration may remain low for some time while the bone changes are repaired.²¹ For vitamin D₃ the assumption implies that the metabolism of vitamin D₃ is affected greatly by anticonvulsant drugs, possibly to the extent that an increased fractional catabolic rate at some state of metabolism is further increased by a further supply of D₃ or 25-OHD₃. The restoration of a normal serum calcium without effect on B.M.C. during treatment with D₃ or 25-OHD₃ is difficult to explain. If the biological half lives of these metabolites are greatly decreased there may be a great oscillation in the concentration of these metabolites, leading to a variable intestinal calcium absorption, which, over some time, might appear as a more constant increase in serum calcium.

Our findings differ from those in other calcium balance studies,^{1 4} but in these studies the effects of vitamin D₂, vitamin D₃, and 25-OHD₃ on anticonvulsant osteomalacia were studied in succession, which makes the evaluation of each vitamin rather difficult.

Our attempt at an explanation of our findings is pure speculation, which aims only to account for the findings. Several unanswered questions arise: what are the serum concentrations of 25-hydroxycholecalciferol (25-OHD₂) and 25-OHD₃ (the usual methods determine the sum of these two metabolites) and of

1,25 (OH)₂D₂ and 1,25 (OH)₂D₃ and possibly other metabolites? The biological half life of D₃ is decreased in anticonvulsant osteomalacia,²² but what is the biological half life of D₂ in this condition? And how does 25-OHD₂ act in anticonvulsant osteomalacia?

Clearly, vitamin D₂ and vitamin D₃, which were believed to have the same effect on osteomalacia, differ in their action when given to patients with anticonvulsant osteomalacia. Furthermore, 25-OHD₃, which was formerly the drug of choice in this condition, is no better than vitamin D₃. The different actions of vitamin D₂ and vitamin D₃ (and 25-OHD₃) may be due to the fact that vitamin D₃ is a naturally occurring vitamin while vitamin D₂ is a pharmacological product which may be less sensitive to drug-induced disturbances of specific enzyme systems.

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Disappearing Diabetes

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Summary

For five years a nurse was treated for diabetes mellitus. She was found to have tampered with her blood and urine tests and later was believed to have fabricated her condition from the outset.

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Introduction

Some diabetics, usually emotionally disturbed adolescent girls, periodically manipulate their control to gain attention or admission to hospital. In non-diabetics factitious hypoglycaemia has been described but factitious hyperglycaemia does not seem to have been reported. This paper describes a probable case of factitious diabetes mellitus.

Case Report

The patient, born in 1950, was the youngest of three sisters. In 1961, after an appendectomy, non-specific cardiac murmurs were heard.

Investigations showed nothing abnormal, however, and they later regressed. In 1966, when a nursing cadet, she was investigated for attacks of falling. She said that they had occurred since childhood, were preceded by abnormal sensations of taste, and were associated with incontinence of urine, tongue biting, and convulsions. This story was not corroborated by her mother. She also complained of poor vision, and though her visual acuity was apparently only 6/60 in each eye she read and played cards effortlessly. Four observers found different field defects on separate occasions. Two electroencephalograms were normal, and a "functional" disorder was diagnosed.

In December 1967, after starting to train as a student nurse, she presented with a four-day history of headache, nausea, thirst, and polyuria, asking if she had diabetes. Her capillary blood glucose level two hours after food was 10.7 mmol/l (192 mg/100 ml). The diagnosis was confirmed, and with insulin lente 28 IU daily and a 180-g carbohydrate diet she became asymptomatic. Repeated blood glucose determinations ranged from 4.9 to 10.8 mmol/l (88 to 194 mg/100 ml). There was no family history of diabetes; her mother had asthma, usually induced by emotional crises. The patient had never taken an oral contraceptive.

In February 1968 she complained of recurrent thirst and polyuria and was admitted to hospital ambulant but vomiting and drowsy. The venous blood glucose on admission was 4.9 mmol/l. She was put on soluble insulin twice daily, and increasing doses were given in view of thirst, polyuria, vomiting, glycosuria, ketonuria, and high capillary blood sugar levels. On one occasion 228 IU soluble insulin was given in 24 hours. Later, on 60 IU soluble insulin twice daily, random capillary blood glucose levels remained above 22.2 mmol/l (400 mg/100 ml) and included one fasting level of 77.7 mmol/l (1400 mg/100 ml).

In April 1968, when at home, she stopped her insulin because of recurrent hypoglycaemic attacks but developed no glycosuria or symptoms for two weeks. In May she resumed insulin because of the reappearance of glycosuria and three days later was readmitted with thirst and vomiting and a capillary blood glucose of 152 mmol/l (2730 mg/100 ml); repeat, 111 mmol/l (2000 mg/100 ml). She was fully ambulant, well hydrated, and not overbreathing. Venous blood urea and electrolytes were normal. Ten hours later, after 120 IU parenteral insulin, her capillary blood glucose was still over 111 mmol/l, but next morning, after a further 100 IU insulin overnight, it was 3.7 mmol/l (66 mg/100 ml). During this period she received 6.5 l of fluid, mainly intravenously, and passed over 9.5 l of urine. She was later seen by a neurologist for shaking attacks, which were thought to be hysterical.

When first referred to King's College Hospital (K.C.H.) in June 1968 as a case difficult to control the accompanying letter described her as a sensible girl who knew immediately when her blood sugar was high or ketosis had developed. She took a close interest in her diabetic control, and for seven months had recorded five times daily the results of her urine tests for glucose and ketones, which always showed synchronous fluctuations. She gave her own insulin injections as an inpatient and continued testing her own urine.

In K.C.H. in June 1968 her diabetes was easily controlled with 32 IU soluble insulin and 28 IU isophane insulin twice daily. She resented and opposed the supervision of her injections. After discharge control again became erratic and she was readmitted. A routine series of capillary blood glucose readings at 9 p.m., 7 a.m., 12 noon, and 6 p.m. were 256, 55.1, 21.4, and 1.1 mmol/l (4608, 992, 386, and 20 mg/100 ml) respectively. The next day she was seen by a psychiatrist, who thought she was well adjusted to her disease. When confronted she denied manipulating her diabetes. She was advised to give up nursing, which she eventually did after three further admissions to her first hospital for stabilization.

In March 1969 she claimed to have taken an overdose of 200 IU insulin but on treatment with oral and intravenous glucose did not become hypoglycaemic. During this admission to K.C.H. she was seen putting glucose on her ears and aspirin and acetone in her urine. She was no longer allowed to give her own injections even under supervision and remained well stabilized and virtually free from hypoglycaemia on 52 IU insulin daily in divided doses of Actrapid and semilente. She had two months of inpatient psychiatric treatment, mainly psychotherapy, and was thought to be a hysterical manipulator with denial and splitting. By July 1969 she freely admitted manipulating her diabetes.

At 14 subsequent outpatient attendances the capillary blood glucose concentrations were all between 2.9 and 6.4 mmol/l (52 and 116 mg/100 ml) on 24 to 80 IU insulin daily in divided doses. Urine showed the intermittent presence of glucose and ketones. Insulin dosage was

frequently adjusted on the basis of urine tests, blood glucose values, and reported hypoglycaemic attacks.

In October 1972 she volunteered that she had taken no insulin since December 1971 and was well apart from symptoms she attributed to hypoglycaemia. A glucose tolerance test (G.T.T.) was arranged but she failed to attend on two occasions, alleging that each time she had lost consciousness in a hypoglycaemic attack on the way to the clinic. In March 1973 she was referred to the ophthalmic department because of difficulty with vision but nothing abnormal was found. The opportunity was taken to perform an unexpected, supervised oral G.T.T. on a reluctant patient with 50 g glucose three hours after a light breakfast. The results at 0, 30, 60, 90, and 120 minutes were 4.0, 9.1, 6.1, 3.0, and 3.3 mmol/l (72, 164, 110, 54, and 60 mg/100 ml) respectively. She declined to attend for further follow-up. One year later, though living at home, she had failed to tell her mother that she no longer had diabetes. She still denied fabrication of her diabetes to her psychiatrist, who felt this to be in character were it true.

Discussion

In retrospect it is easy to suspect manipulation of blood samples and urine tests from the outset. Blood glucose at both hospitals was then routinely estimated on capillary blood samples, which are easy to fake. Her venous blood glucose, urea, and electrolytes were always normal, even when she was in apparent precoma. Her only G.T.T. result was normal and there was never any reliable biochemical evidence that she ever had diabetes mellitus. There were never any abnormal physical signs, even when capillary blood glucose concentrations were 111 mmol/l (2000 mg/100 ml). Her bizarre clinical story is most readily explained on the assumption that she fabricated everything.

She was later known to tamper with her blood and urine tests—this would explain *inter alia* the net fluid loss during treatment for precoma—but it is surprising that she never had severe hypoglycaemic attacks, because large doses (up to 100 IU) of intravenous and intramuscular insulin were given by trained staff when precoma was suspected. In non-diabetic schizophrenics, however, doses of 15 to 450 IU of insulin given subcutaneously¹ and 70 to 200 IU given intravenously² have been needed to induce hypoglycaemic coma. Presumably sufficient carbohydrate could be taken by an insulin-insensitive patient to avert hypoglycaemic coma.

There is a remote possibility that this girl had genuine diabetes mellitus with a later complete remission, though there were no predisposing factors. A faked remission is unlikely in view of the 14 consecutive normal blood glucose readings in the outpatient department before the normal G.T.T. result. The patient derived considerable advantages from inventing diabetes. She gained attention and sympathy from family, friends, and doctors and had a good excuse for successive employment failures. Her mother derived similar benefits from asthma.

Though this interesting variant of the Münchhausen syndrome must be rare and perhaps unrepeatable in these days of the autoanalyser, this case is reported because of its unlikelihood. To outwit successfully two hospitals, numerous doctors, nurses, and her family over five years, including 11 hospital admissions totalling over four months, is remarkable. Her pretence might never have been suspected had she not decided to have a prolonged remission. She was indeed well adjusted to her disease.

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