Case Report

Insulin-dependent diabetes mellitus in an Inuit child

Heather J. Dean, MD, FRCPC
James Carson, MD, FRCPC

The incidence of insulin-dependent diabetes mellitus (IDDM) varies by geographic location and race. It is highest among children in Sweden, Finland and Norway[1] and much lower among oriental and North American Indian children.[2] No cases have been reported until recently in an Inuit population. Since January 1986 we have been treating a 10-year-old Inuit girl with IDDM from a settlement in the Northwest Territories. The initial course was not unusual, but geographic isolation, nomadic lifestyle, preference for a high-protein and simple-carbohydrate diet, and a language barrier provided new challenges.

Case report

A 10-year-old Inuit girl presented to the local nursing station with sudden onset of vomiting after 2 to 3 weeks of polyuria and polydipsia. Diabetic acidosis was diagnosed by the nurse practitioner. After intravenous fluid therapy was started the child was flown about 1600 km to Winnipeg.

The girl had been born of a 19-year-old primigravid woman after a normal 42-week pregnancy and had weighed 4140 g. The only admission to hospital had been for tonsillectomy. A half sister of her maternal grandmother was obese and had non-insulin-dependent diabetes mellitus, Hashimoto's thyroiditis and hypothyroidism. There was no other family history of diabetes and no known consanguinity.

The patient was found to be dehydrated and to have Kussmaul's respiration. She was alert, oriented and euthyroid. Her body mass index after rehydration was 20 (normally 20 to 25) kg/m^2. The plasma glucose level was 22.1 mmol/L, the serum β-hydroxybutyrate level 1010 (normally less than 300) μmol/L, the arterial blood pH 6.99 and the blood bicarbonate level 1.5 mmol/L.

Intravenous fluid therapy was continued, and insulin was given by means of continuous intravenous infusion. The patient's metabolic status was stable within 8 hours. Subsequent treatment included a regimen of semisynthetic human insulin, an 8800-kJ meal plan and an individualized diabetic education program, which emphasized insulin administration, daily monitoring of plasma glucose levels and adjustment of diet, exercise and insulin dose to optimize daily glycemic control.

The titre of thyroid microsomal antibody (1:6400) suggested Hashimoto's thyroiditis. The serum thyroxine, triiodothyronine and thyroid stimulating hormone levels were normal. The titre of islet cell antibodies was not determined. The histocompatibility leukocyte antigen (HLA) profile was established.

Compliance with insulin administration and daily monitoring of plasma glucose levels has been satisfactory. There have been no further admissions to hospital or episodes of severe hypoglycemia or diabetic acidosis. The local school has been very supportive, and school attendance has been good. The girl's daily insulin requirement has remained at about 0.7 U/kg. Her height and weight have been in the 50th percentile for her age, and her pubertal development has been normal.

Three major problems occurred during the second and third years of management. The first was a discrepancy between the glycosylated hemoglobin level and the plasma glucose levels recorded at home. The plasma glucose levels before meals were 6 to 10 mmol/L, but the glycosylated hemoglobin level was 12% to 17%; the normal range of total glycosylated hemoglobin in our laboratory is 4% to 7% as measured with the use of affinity chromatography.[3] Hemoglobin electro-

From the departments of Paediatrics and Child Health and of Community Health Sciences, University of Manitoba, Winnipeg

Reprint requests to: Dr. Heather J. Dean, Children's Hospital, 685 William Ave., Winnipeg, Man. R3E 0Z2

phoresis was not done to rule out a hemoglobin disorder that would falsely elevate the glycosylated hemoglobin level, but such a disorder was unlikely because of the patient's normal hemoglobin level. Other situations such as uremia, alcohol abuse and pregnancy that might result in a falsely elevated total glycosylated hemoglobin level were not present. Falsely low levels of plasma glucose may have occurred at low temperatures because of the temperature dependence of the enzymatic reaction in the glucose strip reagent pad. However, this is unlikely, because the girl's home was adequately heated, and only temperatures below 18°C will give falsely low readings. Furthermore, the strips are reliable after freezing as long as they are brought to room temperature before use. The final reason to suspect false readings at home was the persistence of random or fasting plasma glucose levels greater than 10 mmol/L at the nursing station or diabetes clinic.

The second management problem was inconsistent carbohydrate intake. The family preferred a diet high in protein and low in complex carbohydrates, and the intake of simple carbohydrates was erratic. The concept of consistent carbohydrate content was difficult to teach and reinforce.

The third problem was lack of peer support and geographic isolation. A summer camping experience with Manitoba children who had diabetes did not help: the cultural and language differences seemed to be greater than the benefits of peer support. The community was fortunate to have a full-time resident physician who worked closely with the diabetes education team, the extended family, the local nurses and the school personnel to address these problems and attempt to develop alternative strategies in the home and community.

Comments

The incidence of diabetes is rare in the Inuit population. The age-adjusted prevalence among the Inuit of Alaska and Greenland is 8 to 10 per 1000, as compared with 25 per 1000 in the general population of North America.[4] Non-insulin-dependent diabetes mellitus accounts for more than 90% of these cases, and its prevalence may be increasing among the Inuit in a pattern similar to that seen among North American Indians during the past 30 years.[4] To our knowledge this is the first clinical report of IDDM in an Inuit child. One child was included in a recent study of diabetes mellitus among Alaskan natives,[4] but no clinical data were presented.

Although we did not determine the titre of islet cell or autoinsulin antibodies we believe that the IDDM in this case was due to autoimmune isletitis. The girl showed signs of concomitant thyroiditis, which supports the possibility of autoimmune disease.[5] More than 95% of people with IDDM have the HLA-DR3 or HLA-DR4 haplotype, or both. The patient we have described had the HLA-DR4 haplotype; however, 60% to 80% of Inuit people have this haplotype, as compared with 40% of other nondiabetic people.[6,7] Therefore, IDDM could not be diagnosed on the basis of her HLA-DR haplotype.

The possibility of non-Inuit genes was ruled out after the primary care physician questioned the family and community members about the girl's lineage. An environmental toxin or trigger was unlikely. The girl had not lived outside the Keewatin district and had never taken any drugs except for antibiotics. Direct viral-induced B-cell destruction probably accounts for fewer than 10% of the cases of IDDM, but in this case there was no antecedent febrile illness or symptoms immediately before or after resolution of the diabetic acidosis. Thus, an environmental cause seems unlikely.

The diagnosis of IDDM in remote Arctic settlements requires astuteness of health care providers. Equally challenging is long-term management, especially control of diet because of the seasonal variation in the type of food and a traditional diet high in protein. In addition, conventional teaching methods may not be applicable because of the cultural and language differences.

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