Glycosylated Hemoglobin in Pregnancy

Glycosylated hemoglobin (HbA1c) concentrations may be affected by pregnancy, but reports on the direction of the change have not been consistent (1–5). A group of 167 normoglycemic Pima Indian women aged 15–44 years, were each seen three times over a median of 2.7 years (25th, 75th centiles 1.6 and 3.4 years, respectively) to assess differences in HbA1c and 2-h postload plasma glucose (2HPG) measured when the women were pregnant and nonpregnant (3rd trimester) examination and from nonpregnant examination to the next nonpregnant examination were evaluated. On average, HbA1c was significantly (P < 0.001) lower during pregnancy by 0.5%, regardless of the order of measurement (Fig. 1). 2HPG was also lower during pregnancy by 0.14 mmol/L, but the difference was not significant (P = 0.34), and HbA1c remained significantly (P < 0.001) lower during pregnancy even when controlled for 2HPG. The correlation between HbA1c and 2HPG was 0.67 (P < 0.001) when the women were pregnant and 0.78 (P < 0.001) when they were not.

These findings show that HbA1c concentrations are lower when a woman is pregnant than when she is not despite the fact that the plasma glucose concentration is not significantly different. Regardless of the reasons for the reduction in HbA1c during pregnancy, normality in pregnancy cannot be judged by nonpregnant standards.

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References


Subclinical Brain Dysfunction in Patients With Diabetic Ketoacidosis

Subclinical brain dysfunction has been reported to occur in most conscious adults during therapy for diabetic ketoacidosis (1). It may be an unpredictable but potentially deleterious complication of diabetic ketoacidosis in neurologically symptom-free or mildly symptomatic patients (2–5). Cranial computed tomography scans, measurement of cerebrospinal fluid pressure, and echoencephalography have shown a high frequency of alteration in brain volume, compatible with mild brain swelling during diabetic ketoacidosis (3–5). We evaluated the diagnostic value of sensory evoked potentials (SEPs) in the detection of subclinical brain dysfunction during diabetic ketoacidosis.

Five patients (1 male and 4 female patients aged 20–66 years) with diabetic
ketoacidosis were studied after admission to the intensive care unit. Diabetic ketoacidosis was defined as hyperglycemia (serum glucose, >300 mg/dl [16.7 mmol/l]), metabolic acidosis (arterial pH, <7.25; serum bicarbonate, <15 mmol/l), and ketosis (presence of urine ketones). On admission, laboratory data were as follows: blood glucose, 41.4 ± 18.1 mmol/l; pH, 7.1 ± 0.1; serum bicarbonate, 6.9 ± 2.5 mmol/l; and serum osmolality, 349 ± 40 mOsm/l. All patients received conventional fluid and low-dose insulin therapy. Regular crystalline insulin was given as an intravenous bolus of 20 IU. The insulin therapy following was given as a continuous intravenous infusion at a rate of 3-6 IU/h, adjusted to each patient’s response. Fluid therapy was started with normal saline solution at a rate of 1,000 ml/h for the first hour, followed at a rate of 200–500 ml/h according to central venous pressure. If blood glucose was <250 mg/dl (14 mmol/l), 5% glucose solution was added to the intravenous replacement fluid. Patients did not receive sedatives at the time of study. Short- and long-latency SEPs were recorded according to standard methods 2 h after the onset of ketoacidosis treatment and 7 days after the normalization of the metabolic disorder, respectively (6). At the time of study, all patients were neurologically asymptomatic (Glasgow Coma Scale Score 15) without deterioration of consciousness (except slight drowsiness) or any clinical signs of increased cerebral pressure.

Two hours after the start of therapy, cervical N13 to cortical N20 interpeak latency of SEPs (5.8 ± 0.3 ms) was significantly prolonged in all five patients, compared with age-matched healthy subjects (5.3 ± 0.2 ms, P < 0.05). The N20 peak latency (20.8 ± 1.9 ms) of short-latency SEPs and the N35 peak latency (40 ± 6 ms) and the N70 peak latency (102 ± 13 ms) of long-latency SEPs yielded a significant delay in all five patients, compared with age-matched control subjects (N20: 18.6 ± 1.3 ms, P < 0.05; N35: 34 ± 2 ms, P < 0.05; N70: 76 ± 4 ms, P < 0.01). N13–N20 interpeak latency (5.1 ± 0.4 ms), N20 (19 ± 1.0), N35 (34 ± 4 ms), and N70 peak latency (70 ± 4 ms) normalized in all five patients 7 days after recovery from diabetic ketoacidosis. The amplitudes of the N20, N35, and N70 peaks showed no difference between the first measurement and the group of control subjects nor between the first and the second SEP recording. All values are expressed as means ± SD. Data were statistically analyzed by Student’s t test for paired and unpaired data.

Our study demonstrates that both the early and the late cortical components of SEP peak latencies are significantly prolonged in all five neurological asymptomatic patients with severe diabetic ketoacidosis. Since SEPs were significantly prolonged in all five patients, our findings indicate that subclinical brain dysfunction may be a common but transient complication in patients with diabetic ketoacidosis without any obvious clinical manifestation. Our results indicate that SEPs provide additional information in the cerebral monitoring of patients during the treatment of diabetic ketoacidosis.

Vaccines and the Appearance of Islet Cell Antibodies in Offspring of Diabetic Parents

Results from the BABY-DIAB Study

Controversy exists about the influence of mumps infection and vaccination on the development of type I diabetes (IDDM). Hyoty et al. (1) observed a plateau in the rising incidence of type I diabetes after the introduction of a nationwide mumps-measles-rubella (MMR) vaccination in Finland. They suggested that the elimination of the natural mumps virus by vaccination had decreased the risk for IDDM. In their study, the protective effect of MMR vaccination was combined with a concomitant decrease in mumps antibody levels in diabetic children (1). On the other hand, a direct causal relationship between vaccination and onset of diabetes was published by Helmkie et al. (2) from Germany. Helmkie et al. (2) described seven children who developed diabetes shortly after an active mumps-measles vaccination. Furthermore, they reported induction of islet cell antibodies (ICAs) in 21 of 127 nondiabetic children in association with mumps. One of these 21 ICA+ children had developed clinical IDDM on follow-up. Besides mumps there is a recent ongoing debate whether bacille Calmette-Guérin (BCG) vaccines may influence islet cell autoimmunity in human and animal diabetes (3,4).

We therefore evaluated the appearance of islet-associated autoantibodies (insulin autoantibodies [IAA]; glutamic acid decarboxylase antibodies [GADA]; tyrosine phosphatase IA-2 antibodies [IA-2A]) in association with vaccination in 280 nondiabetic children of IDDM parents (232 of IDDM mothers and 48 of IDDM fathers). Prospectively, all children were followed from birth up to the age of 2 years or more, with regular venous blood sampling in the BABY-DIAB study (design of BABY-DIAB (5,6)). Antibody positivity was defined by the 99th percentile of antibody levels in age-matched control sera.

Of all 280 children, 29 (10.4%) were found to be positive for at least one of the four antibodies tested (14 of 29 had more...