Growth Hormone Replacement in a Hypopituitary Insulin Dependent Woman

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INTRODUCTION

Whilst hypoglycaemia is a common manifestation in children with GH deficiency, this is seldom a clinical problem in GH deficient adults. However, when GH deficiency occurs in insulin dependent diabetic adults, hypoglycaemic attacks are more frequent, prolonged and life threatening /1,2/. Spontaneous resolution of retinopathy in a woman who infarcted her pituitary gland led to pituitary ablation as a form of treatment /3/. In spite of GH being implicated in the pathogenesis of diabetic retinopathy, GH replacement therapy was undertaken in FG because the quality of her life became unacceptable.

CLINICAL HISTORY

FG was born in 1954 and at the age of 7 years presented with insulin dependent diabetes. She was treated with a controlled carbohydrate diet and a single daily dose of a mixture of soluble and protamine zinc insulin. Diabetic control was poorly documented but was probably indifferent since her adult height is only 1.48 metres. In 1975, aged 21 years, 14 years after the onset of her diabetes, she sustained a left vitreous haemorrhage during her final university examinations. A major retinal detachment had occurred so that no further treatment was possible and visual acuity in her left eye has remained 6/60. The right eye demonstrated proliferative retinopathy and over the subsequent two years 2379 laser burns were applied to her retina. Since 1977 she has retained 6/9 vision in her right eye. In 1977 FG was introduced to home glucose monitoring and her own blood glucose results finally convinced her that she could not achieve adequate glycaemic control on a single daily injection of insulin. In 1980 HbA1 measurements were introduced into our clinic, allowing an independent estimate of her blood glucose control. Her first and second pregnancies in 1981 and 1983 were relatively uneventful. In 1986 at 31 weeks gestation she developed a severe headache; a subarachnoid haemorrhage was suspected but the cerebrospinal fluid contained no excess red or white cells. She remained nauseous with a very poor appetite requiring much smaller insulin doses. An elective Caesarian section under epidural anaesthesia was carried out at 33 weeks gestation. She failed to lactate, felt lethargic and required much smaller doses of insulin. Six weeks post partum she was investigated for hypopituitarism.

INVESTIGATIONS

Table 1 demonstrates that FG had both low basal plasma cortisol values and low cortisol secretion rate. She had no prolactin or TSH response to TRH, her gonadotrophins were low and failed to rise following GnRH. A long synacthen test demonstrated an adrenal cortical response at 240 minutes. Investigation of GH secretion demonstrated no GH response, either to hypoglycaemia or GHRH. To our surprise her free T_4 and free T_3 were initially within the normal range and her T_4 was transiently raised. Five weeks later her thyroid hormones were in the hypothyroid range; see Table 2.

Antepartum pituitary infarction had been confirmed. Infarction of a pituitary tumour or lymphocytic hypophysitis were excluded as computerised axial tomography demonstrated a