Effect of growth hormone on fatty liver in panhypopituitarism

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Abstract
A 17 year old boy was admitted because of short stature and hepatomegaly. He was diagnosed with panhypopituitarism and fatty liver. The fatty liver improved, not with hydrocortisone or levothyroxine treatment, but with growth hormone administration. The fatty liver in this patient was attributable to a growth hormone deficient state. (Arch Dis Child 1997;76:537–538)

Keywords: fatty liver; growth hormone; growth hormone deficiency; hypopituitarism

It has become evident that besides its growth promoting action, growth hormone has several metabolic actions on adipose tissue. Chronic exposure to growth hormone induces a decrease in lipogenesis and an increase in lipolysis. As the lipolytic effect of growth hormone is more pronounced in the abdominal fat mass, patients with growth hormone deficiency usually present with truncal-type obesity. However, the massive deposition of fat in the liver, that is fatty liver, has not been reported in growth hormone deficient patients. We report a patient with panhypopituitarism who suffered from fatty liver that improved with growth hormone treatment.

Case report
A 17 year old boy was admitted to our hospital because of short stature and hepatomegaly. He was the product of a normal full term pregnancy with a breech presentation; his birth weight was 2690 g. At the age of 13 years he was diagnosed with tricuspid regurgitation, and a tricuspid valve repair operation was performed. At the same time he was noted to have hepatomegaly and hypothyroidism (serum triiodothyronine 1.31 nmol/l, serum thyroxine 37 nmol/l). A thyrotrophin releasing hormone (TRH) stimulation test demonstrated a delayed and prolonged rise in thyroid stimulating hormone, indicating hypothalamic TRH deficiency. Administration of gonadotrophin releasing hormone showed no gonadotrophin response. His plasma cortisol was below the detection level, and insulin induced hypoglycaemia failed to increase the plasma cortisol concentration. A growth hormone provocation test using insulin, clonidine, and growth hormone releasing factor also failed to increase the serum growth hormone concentration. His serum insulin-like growth factor (IGF)–I concentration was 2.9 nmol/l. Based on these findings we made a diagnosis of panhypopituitarism. Magnetic resonance imaging of the brain did not show any abnormal findings.

Hydrocortisone (0.3 mg/kg) replacement therapy was begun, and the dose of levothyrox-
Aspartate aminotransferase (U/l) 94 (11-32)*
Alanine aminotransferase (U/l) 86 (6-39)*
γ-Glutamyltranspeptidase (U/l) 107 (3-40)*
Lactate dehydrogenase (U/l) 393 (236-455)*
IGF-I (nmol/l) 2.9 (37.4-82.0)*

*Normal range for the patient’s age.

Discussion

Our case is particularly interesting as the fatty liver improved not with hydrocortisone or levothyroxine treatment, but with the administration of growth hormone. This fact strongly suggests that the fatty liver in this patient was attributable to the growth hormone deficient state. Although numerous metabolic and nutritional disorders have been known to cause fatty liver, the fatty liver associated with growth hormone deficiency has not been reported. As adipocytes lack functional IGF receptors, the lipolytic effect is a direct consequence of growth hormone rather than mediated through IGF-I given the evidence that IGF-I treatment in children with Laron’s syndrome produces lipolytic effects.