I read with great interest the paper by Nolte et al. (European Journal of Endocrinology 1997 137 459–466) as well as the commentaries by Mooser & Hobbs (European Journal of Endocrinology 1997 137 450–452) on the influence of human growth hormone (hGH) treatment on serum lipoprotein(a) (Lp(a)). These authors do not seem to be aware of other papers published recently on the same topic by both the Swedish group (1) as well as ours (2–4), wherein the Swedish group treated adult patients with hGH for short periods, and we treated both adult growth hormone deficiency (GHD) patients and children for a period of at least 1 year.

We also reported the effect of the rise of endogenous hGH by the administration of GH-releasing substances on serum Lp(a) (4).

Both the exogenous as well as the stimulated endogenous hGH elevated significantly serum Lp(a) levels in some patients even over the accepted risk limit of 300 mg/l. This was especially true in children with chronic renal failure (3).

In contradistinction, our group demonstrated in other studies that insulin-like growth factor-I (IGF-I) is a very potent agent in the reduction of serum Lp(a) even when the basal levels are not high (5). This was also confirmed in adults by the Swedish investigators (1).

In our young adult patients retreated with GH, the reduction in serum adiposity as measured by skinfold thickness was transitory and lasted for only 6–9 months (6). The same was true for the IGF-I treatment of adult patients with Laron syndrome (7).

Our opinion is that Lp(a) should be monitored not only in adult patients undergoing GH replacement treatment, but also children, and in addition in any subject receiving ‘non-conventional’ hGH treatment (8). Those identified as being able to profit from GH, but having or developing serum Lp(a) levels of 300 mg/l or more, should be considered for IGF-I treatment.

References

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