

Dual aetiologies for hyperinsulinaemic hypoglycaemia in one patient

To the Editor—We read with interest the articles by Ching et al¹ and Ma.² We agree with Ching et al's recommendation that health care providers should be vigilant to the problem of hypoglycaemia due to inadvertent use of oral hypoglycaemic agents (OHA), and are encouraged to see the increased availability of urine toxicology services for detection of OHA in Hong Kong after the cluster of iatrogenic hypoglycaemia incidents in May 2005.³ However, we also wish to highlight the importance of not overlooking other treatable causes of hypoglycaemia, even in the presence of positive toxicology, by sharing with readers the following case.

A 24-year-old man was referred to us in September 2006 because of recurrent hypoglycaemic symptoms for 6 months. Investigations confirmed hyperinsulinaemic hypoglycaemia. His glucose level after a 12-hour fast was 1.8 mmol/L, and simultaneous insulin was 27.5 mIU/L (reference level, <6 mIU/L), C-peptide 0.97 nmol/L (reference range, 0.27–1.27 nmol/L), and beta-hydroxybutyrate 0.07 mmol/L (reference level, <0.27 mmol/L). No pancreatic mass was identified on computed tomographic scanning. Urine toxicology was positive for metabolites of gliclazide. In answer to our probing into the social history, his parents revealed that he was living with his grandparents, both of whom had diabetes mellitus and were taking gliclazide. However, he denied use of OHAs and we could identify no secondary gain to explain it. He was then transferred to the isolation ward and kept under closed circuit TV monitoring. During isolation, his urine toxicology became negative,

and he no longer had symptoms of hypoglycaemia. Yet his pre-meal haemoglucostix hovered over the brink of hypoglycaemia, between 3 and 4 mmol/L, prompting us to reconsider the differential diagnosis of insulinoma. Arterial stimulation venous sampling revealed a two-fold rise of the insulin level in the superior mesenteric artery territory. Enucleation was performed and an insulinoma confirmed on histology.

To determine whether the first urine toxicology result might have been falsely positive, the toxicology laboratory repeated the analysis of residual urine from the first urine sample to exclude an analytical error. It was again positive for gliclazide metabolites. We checked with nursing staff and could find no simultaneous urine collecting activity in the ward that could have accounted for a mix-up of samples. Urine protein electrophoresis on all his urine samples, those that had been positive for gliclazide as well as the subsequent negative ones, was also unrevealing in that they all showed normal protein excretion patterns.

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