Hyperglycaemia induced by paracetamol

Sir,
Hypoglycaemia is a well-recognised feature of paracetamol overdose, after the development of fulminant hepatic failure, but we are aware of only one previous description of hyperglycaemia and that was in a patient with a family history of diabetes mellitus.¹

Over recent months we have seen five patients, all male, aged 17 to 40, who have had random blood sugars of 7.2–12.8 mmol/l (mean 9.1 mmol/l) at the time of their initial paracetamol level, taken 6–12 h after the overdose (paracetamol levels of 50–184 mg/l, mean 147 mg/l). Hepatic failure, but with full recovery, developed in two patients. None were known to have diabetes, although two had a family history of non-insulin-dependent diabetes mellitus.

Paracetamol has previously been shown to interfere in glucose measurements,² although YSI have modified their method and this is now less of a problem. However, none of the instruments used in the emergency laboratories in the Leicester area employ this method, and we investigated the spiking of sera with known glucose levels with paracetamol to see if the glucose results altered. Over a paracetamol concentration range of 10–400 mg/l, there was no change; thus paracetamol does not interfere with glucose measurements.

It would thus appear that hyperglycaemia is a feature of a significant paracetamol overdose over the first 12 hours. Whether this is due to release of stress hormones or a direct effect of either paracetamol or its metabolites on the pancreas or liver is unclear.

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1 Thomas JS, Prescott LF. Liver damage and impaired glucose tolerance after paracetamol overdosage. *BMJ* 1966; 2: 506–7.
2 Fleetwood JA, Robinson SMA. Paracetamol interference in glucose measurement by YSI analyser. *BMJ* 1981; 283: 438–9.

Metabolic effects of paracetamol overdose

Hepatotoxicity

- raised aspartate amino-transferase/alanine amino-transferase
- raised lactate dehydrogenase
- raised bilirubin
- disordered clotting
- hypoglycaemia
- hyperammonaemia

Hypokalaemia

Renal damage

- acute tubular necrosis
- proteinuria
- haematuria

Dementia, myopathy, and idiopathic hypoparathyroidism

Sir
Idiopathic hypoparathyroidism (IH) is a rare disease characterised by deficient secretion of parathyroid hormone (PTH) without a defined cause (box 1). Some patients with this disorder also develop adrenal insufficiency or other endocrine deficiencies, pernicious anaemia, vitiligo, chronic mucocutaneous candidiasis, or other syndromes.¹ We report a patient with IH who presented with dementia and myopathy.

A 61-year-old man was admitted because of uncontrollable seizures. His past medical history was irrelevant except for epilepsy diagnosed at the age of 30, and a progressive decline in mental functions in the last two years, to the point that he was unable to perform everyday tasks. On physical examination he was in apparent good health but apathetic and incoherent, he had no dysmorphic features, and his heart, lungs, abdomen, extremities, and neurologic examination were normal. Every five or ten minutes he suffered brief generalised tonicoclonic convulsive attacks, without complete loss of consciousness or urinary incontinence, that responded poorly to anticonvulsant therapy.

Blood analysis revealed a moderate leucocytosis with left deviation, calcium 5.5 mg/dl (albumin 3.7 g/dl), phosphate 4.8 mg/dl, intact parathyroid hormone 1.2 pg/ml, creatine kinase 2220 U/l, alanine aminotransferase 190 U/l, aspartate aminotransferase 121 U/l and lactic dehydrogenase 435 U/l. An electrocardiogram showed a prolonged QT interval. Radiographs of the chest were normal. A computed tomographic scan of the brain disclosed calcifications, particularly in the basal ganglia.

When blood calcium levels were received, an intravenous calcium infusion was instituted and the convulsive attacks ceased. Oral calcium and vitamin D were then initiated. In the following weeks his mental functions moderately improved and he began to perform some daily activities by himself.

Symptoms of IH usually begin in childhood, but sometimes appear later. Epilepsy was probably the first manifestation of IH in our patient, but his illness was not diagnosed until the age of 61, when dementia, probably related to brain calcifications,² had developed. IH is one of the few causes of reversible dementia, but other cases³ and ours show that adequate treatment may not produce complete recovery of mental functions. This emphasises the importance of an early diagnosis.

As in other cases of IH,^{4–6} serum muscle enzymes were increased in our patient, probably as a consequence of rhabdomyolysis secondary to hypocalcemia and/or recurrent seizures.^{6,7}

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Idiopathic hypoparathyroidism: features

- deficient secretion of parathyroid hormone
- sporadic or familial basis
- hypocalcaemia and hypophosphataemia
- increased neuromuscular excitability, extrapyramidal syndromes, irritability and other mental changes, dental abnormalities, cataracts
- raised intracranial pressure
- extraosseous calcifications, particularly in brain
- prolongation of QT interval

Box 1

Learning/summary points

- idiopathic hypoparathyroidism is one of the few causes of reversible dementia
- adequate treatment may not produce complete recovery of mental functions. Early diagnosis is therefore essential

Box 2

1 Aurbach GD, Marx SJ, Spiegel AM. Parathyroid hormone, calcitonin, and the calciferols. In: Wilson JD, Foster DW, eds. *Williams textbook of endocrinology*. Philadelphia: W.B. Saunders Company, 1992; pp. 1397–476.
2 Nicolai A, Lazzarino LG. Dementia syndrome in patients with postsurgical hypoparathyroidism and extensive brain calcifications. *Eur Neurol* 1994; 34: 230–5.
3 Friedman JH, Chiucchini I, Tucci JR. Idiopathic hypoparathyroidism with extensive brain calcification and persistent neurologic dysfunction. *Neurology* 1987; 37: 307–9.
4 Battistella PA, Pozzan GB, Rigon F, Zancan L, Zacchello F. Autoimmune hypoparathyroidism and hyper-CK-emia. *Brain Dev* 1991; 13: 61.
5 Ishikawa T, Inagaki H, Kanayama M, Manzai T. Hypocalcemic hyper-CK-emia in hypoparathyroidism. *Brain Dev* 1990; 12: 249–52.
6 Akmal M. Rhabdomyolysis in a patient with hypocalcemia due to hypoparathyroidism. *Am J Nephrol* 1993; 13: 61–3.
7 Gabow PA, Kaehny WD, Kelleher SP. The spectrum of rhabdomyolysis. *Medicine* (Baltimore) 1982; 61: 141–52.