

Deliberate sulphonylurea poisoning mimicking hyperinsulinaemia of infancy

Louise Owen, Matthew Ellis, Julian Shield

Abstract

A 6 month old child presenting with seizures was found to be hypoglycaemic secondary to hyperinsulinism. A family history of type II diabetes prompted estimation of sulphonylurea in the baby's blood, which was found to be high. A multidisciplinary case conference concluded that the sulphonylurea ingestion was likely to be the result of Munchausen syndrome by proxy. When investigating hypoglycaemia of infancy this possibility should be considered.

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Case report

A 6 month old child presented to the accident and emergency department following a generalised seizure. The patient was apyrexial, poorly perfused, and responding only to pain. There was no report of choking, apnoea, or cyanosis, and no significant past medical history. On arrival at hospital the baby was resuscitated and underwent a septic screen, which was negative, and a urine toxicology screen which did not detect benzodiazepines, amphetamines, cocaine, methadone, or opiates. Blood glucose was 6.0 mmol/l (2.6-7.0 mmol/l); examination was unremarkable. The child remained unresponsive for five hours after admission, before returning to normal consciousness. No further seizure activity was reported.

Five days later the child presented again with a history of a seizure. On arrival the child was conscious and alert, but capillary blood glucose was 1.3 mmol/l. Plasma ammonia and lactate were normal, urinary ketones were negative. Dextrose 10% was administered, providing more than 10 mg/kg/min glucose. Despite this, the blood glucose repeatedly fell below 2.5 mmol/l, and further dextrose boluses were needed over the next 24 hours.

An initial insulin level was 428 pmol/l, inappropriately high for the original blood glucose of 1.3 mmol/l, and C peptide was 5.396 nmol/l, suggesting endogenous hyperinsulinism. A full metabolic screen, prolonged fast, glucose tolerance test, and computed tomography of the pancreas revealed no abnormalities. The child had no further hypoglycaemic events.

Questioning on the ward the day after admission revealed that the maternal grandmother and great grandmother had type II diabetes mellitus, controlled with oral hypoglycaemic agents. Sulphonylurea concentrations were therefore requested on the blood samples

from the second admission and were detected at a level of 806 µg/l. The assay was unable to identify the precise type of sulphonylurea. Unfortunately lack of stored blood from the first admission precluded a retrospective assay.

A multidisciplinary case conference attended by representatives from social services, police, and health workers (community paediatrician and health visitors) revealed complex problems in coping mechanisms, as well as a history of violence, and physical and sexual abuse, in several family members. It was disclosed that the mother's first child had been removed from her care, aged 3, owing to emotional, physical, and sexual abuse. Both the mother and her then partner were named as perpetrators. The case conference convened on the present occasion recommended foster care pending further police investigation of presumed Munchausen syndrome by proxy.

Discussion

The differential diagnosis in cases of infant hypoglycaemia includes endocrine deficiencies, inborn errors of metabolism, and hyperinsulinism. However, sulphonylurea ingestion is not commonly considered. Accidental sulphonylurea ingestion is seen in young children,¹ and cases of factitious hypoglycaemia have been reported, following intentional sulphonylurea ingestion by adults.² However, deliberate administration of oral hypoglycaemic agents has not previously been described in infancy. The sulphonylurea level of 806 µg/l is extremely high; the usual therapeutic range in adults is 5-200 µg/l.

There have been cases of prolonged neurological sequelae after sulphonylurea ingestion,³ despite correction of the blood glucose, with hemiparesis, agitation, and confusion persisting for up to 48 hours post ingestion. This raises the possibility that the patient's first admission might also have been owing to sulphonylurea ingestion. Her blood glucose on admission was normal but her neurological status remained depressed for several hours.

The standard investigations for hypoglycaemia in infancy do not include an estimation of sulphonylurea concentrations. The initial finding in this child appeared consistent with endogenous hyperinsulinaemia. When investigating hypoglycaemia of infancy we suggest that a history of potentially available sources of oral hypoglycaemic agents should be elicited from carers. If such a history is obtained, estimation of sulphonylurea concentrations should be undertaken.

Bristol Children's
Hospital, Bristol, UK
L Owen
M Ellis
J Shield

Correspondence to:
Dr M Ellis, Institute of Child
Health, St Michael's Hill,
Bristol BS2 8BJ, UK
email: M.Ellis@Bristol.ac.uk

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May 2000 issue

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