medication. In this case of an acute overdose in an adult, cerebral and respiratory depressions reversible with naloxone were the primary manifestations, consistent with an opioid overdose.

The half-life of tramadol has been reported at 5.1 to 5.8 hours and that of the active M1 metabolite has been reported to be 6.7 to 9 hours.[1,5] These are significantly longer than the half-life of naloxone, thus requiring repeated doses of the opioid antagonist. In this case, a continuous intravenous infusion of naloxone was effective and ultimately discontinued 16 hours after presentation. We did not check a serum level of tramadol. Although such an assay is available, it was believed that treating the symptoms of a narcotic overdose clinically was of primary importance and would not be affected by a serum level. Calculating dosage from the total number of pills missing at the scene, we estimated a total ingestion of 5.5 grams.

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3,4-METHYLENEDIOXYMETHAMPHETAMINE (MDMA, OR "ECSTASY") AND ASSOCIATED HYPOGLYCEMIA

To the Editor:—Recreational use of 3,4-methylenedioxymethamphetamine (MDMA) is common. Both related toxic effects and their treatment (which may include sedating agents) may be associated with altered consciousness. We report a case of severe MDMA toxicity associated with profound early hypoglycemia, which might have passed undetected with potentially serious results.

A 25-year-old woman had taken 3 tablets of MDMA and collapsed 4 hours later. Witnesses reported a grand-mal convulsion. On arrival at hospital she was agitated, tachycardic (155 beats/min), and febrile (rectal temperature 41.9°C). Her blood pressure was 120/40 mm Hg, and her blood sugar level was 7.2 mmol/L. She was cooled with ice-packs and given 3 L cooled intravenous saline, 10 mg rectal diazepam, and 50 mg intravenous dantrolene. Twenty minutes later her temperature was 40.4°C and an additional 30 mg dantrolene was administered. At 90 minutes, her temperature was 38.5°C and her blood sugar level was 4.3 mmol/L. Agitation increased and a chlormethiazole infusion was initiated.

At 6 hours, routine BM Styx dipstick assessment of blood sugar read "low." A blood drawn at that time showed her blood sugar level to have been 0.6 mmol/L. She was administered 50 mL 50% dextrose solution intravenously, with an immediate improvement in conscious level. Over the subsequent day, her BM Styx dipped to 3.8 mmol/L at 17 hours but otherwise remained >4.0 mmol/L.

Over subsequent days, she required support for progressive coagulopathy and rhabdomyolysis, although thyroid and liver enzyme levels (other than aspartate transaminase) remained normal.

Acute hypoglycemia has not previously been reported as an adverse effect of MDMA toxicity. Although hepatotoxicity in association with MDMA is becoming increasingly recognized, its usual time course makes it an unlikely cause of hypoglycemia here.[1,2] In addition, the fact that blood sugar levels were sustained without the need for continued supplementation suggests that acute hepatic failure was not the sole cause. Prolonged status epilepticus can rarely be associated with hypoglycemia, especially in patients with reduced hepatic glycogen stores.[3] However, neither of these conditions occurred in our patient. Neither rhabdomyolysis or hyperpyrexia, nor the administration of dantrolene or chlormethiazole, has been previously associated with hypoglycemia. Indeed hyperpyrexia is more commonly associated with hyperglycemia.[4]

Interestingly, a case remarkably similar to ours has been reported in association with halothane exposure.[5] In this case, too, malignant hyperpyrexia was treated with dantrolene, and rhabdomyolysis associated with an early profound hypoglycemia (0.2 mmol/L) developed. The authors reviewed the related literature but were unable to identify the cause of the hypoglycemia. It is interesting to note the presence of pyrexia, rhabdomyolysis, and dantrolene treatment in both cases.

Questions remain about causality. Is the hypoglycemia related to one of the above in isolation, all in combination, or directly to MDMA/halothane toxicity via another route? We note that salicylate poisoning may be associated with both pyrexia and hypoglycemia, possibly through an uncoupling of oxidative phosphorylation. Might a common mechanism be at work in this case?

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