

# Insulin overdose with recurrent severe hypoglycemia and preserved level of consciousness

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## Case presentation

Paramedics were called to the home of a 45-year-old male who had injected approximately 600 units of a mixture of NPH and regular insulin subcutaneously 3 hours earlier. In addition to the insulin, he had also taken an unknown amount of hydromorphone and alcohol. He was alert and oriented, with a Glasgow coma score of 15. His serum glucose level was 1.1 mmol/L, measured by glucometer. The attendants administered oral glucose (Glucogel®) and started an intravenous (IV) infusion of 5% dextrose (D5W) at 100 mL/h. The patient was still alert and oriented on arrival in the emergency department (ED).

His past medical history included alcoholism, IV cocaine use, depression and recently diagnosed HIV, as well as remote splenectomy and pancreatectomy following abdominal trauma. He had attempted suicide, using insulin, one month prior to the index presentation. His medications included pancrelipase preparation (Cotazym®), hydromorphone, ranitidine and insulin (45 units NPH with 25 units regular each morning, and 35 units NPH with 15 units regular each evening).

The patient's physical examination was non-contributory. Electrolytes, hemoglobin and platelet count were normal, but his leukocyte count was elevated, at  $16.0 \times 10^9/L$  with  $11.44 \times 10^9/L$  neutrophils.

Hospital policy prohibited emergency department (ED) glucometer testing (because of inadequate staff training); therefore, all serum glucose measurements were performed by the laboratory. The patient's initial serum glucose was 1.1 mmol/L. His second level, at 4 hours, was 1.3 mmol/L. Despite these levels, he remained alert and oriented.

Six hours after the patient's arrival at the ED, the attending nurse noted that he appeared diaphoretic and drowsy while he was standing outside the ED, smoking. However, he was able to walk back to his bed independently. He fell asleep, but was easily aroused and remained oriented. At this point his serum glucose was 0.2 mmol/L. The dextrose infusion was increased to D10W at 125 mL/h, and hourly glucose measurements were obtained.

The patient subsequently had 2 more episodes of laboratory hypoglycemia (2.6 mmol/L and 3.2 mmol/L). He was given 50 mL IV boluses of D50W following every hypoglycemic (<3.3 mmol/L) laboratory result.

After approximately 14 hours, his serum glucose stabilized in the normal range and a sliding scale of regular insulin was introduced. Throughout his therapy the patient remained normokalemic and did not receive potassium supplementation. Toxicology screening and free insulin levels were not performed because it was felt they would not contribute to patient management. No additional cognitive testing, beyond the Glasgow Coma Scale, was performed in the ED.

## Discussion

This patient exhibited an unusual and exceptional tolerance to hypoglycemia. On 3 occasions he was alert and oriented, with glucose levels of 1.1 mmol/L (twice) and 1.3 mmol/L. With a glucose level of 0.2 mmol/L he was drowsy but oriented.

Symptoms of hypoglycemia usually appear at glucose levels of 2.8 to 3.3 mmol/L.<sup>1</sup> Impaired brain function usually becomes apparent below 2.8 mmol/L, but subtle changes may occur at levels of 3.0–3.3 mmol/L.<sup>1–3</sup> Some individuals tolerate glucose levels below 2.0 mmol/L without cognitive changes.<sup>2–4</sup>

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Received: Nov. 4, 1999; final submission received: Jan. 15, 2000; accepted: Feb. 1, 2000  
*This article has been peer reviewed.*

During periods of flux, neurologic symptoms of hypoglycemia probably lag behind blood glucose levels. Herold<sup>4</sup> found that the maximum change in neurologic response occurred 10 to 60 minutes after nadir glucose levels were reached. In addition, recurrent hypoglycemic episodes lead to a form of tolerance, with decreased production of insulin counter-regulatory hormones and reduced sympathetic response to hypoglycemia.<sup>5</sup> This patient had attempted suicide at least once, recently, using insulin. And, based on his history of regular insulin use with poor, infrequent meals and recreational drug and alcohol abuse, he may also have had frequent episodes of hypoglycemia.

## Conclusion

This patient's exceptional hypoglycemic tolerance may be explained by genetic predisposition, acquired tolerance and delayed onset of symptoms. Clinical findings may not always reflect serum glucose levels, and "occult" hypoglycemia may be more common than we think.

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