Case Report

Insulin poisoning with suicidal intent

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ABSTRACT

We report a 27-year-old paramedical lady with no known comorbidities, who presented with rapid-onset coma with hypoglycemia (plasma glucose at admission was 35 mg/dL). Clinical alertness suspected and confirmed the diagnosis of exogenous insulin administration probably with suicidal intent. During the course of her ICU stay, she developed bradycardia and hypotension which required ionotropic support. She remained in coma for 90 hours. A total of 470 g of dextrose was infused until she regained consciousness. No other complications of insulin overdose were observed during her stay in the hospital. Recovery was complete without any residual neurological deficits. Insulin administration should be kept in differential diagnosis when any case presents with coma and hypoglycemia, especially in paramedical personnel.

Key words: Hypoglycemic coma, insulin, suicide

Introduction

Insulin is essential for survival in type 1 diabetes mellitus and insulin-requiring type 2 diabetes mellitus patients. The same insulin if taken in overdose in these patients or in non-diabetics can lead to hypoglycemic coma which can have varied outcome from complete reversal to death. Since the introduction of insulin therapy in 1921, diabetics have used insulin overdose as a mode of suicide.[1] Insulin poisoning is also used as a mode of suicide in non-diabetics, especially medical and paramedical personnel and relatives of diabetic patients.[2] We report a case of a paramedical personnel injecting insulin with suicidal intent and discuss the various issues in management.

Case Report

A 27-year-old paramedical personnel without any comorbidities, working as an assistant in the operation theater, was found to be drowsy and drenched in sweat with bradycardia (34 beats/min) and hypotension (80/50 mm of Hg). She was immediately shifted to ICU. She was pale and there was no cyanosis, icterus, clubbing, lymphadenopathy, or any evidence of external injury. Temperature was 99.0°F, with a respiratory rate of 20/min and cold peripheries. Pupils were bilateral 3 mm, reactive to light, and oculocephalic reflex was preserved. Deep tendon reflexes were brisk and plantars were flexor. Meningeal signs were absent. Her systemic examination was unremarkable. An electrocardiogram showed sinus bradycardia. Atropine was given intravenously and normal saline infusion started. Blood pressure remained low which prompted initiation of norepinephrine drip. Capillary blood glucose (CBG) was 35 mg/dL, hence 50 mL of 50% dextrose bolus was given and 5% dextrose infusion started. Her neurological status started deteriorating and she rapidly lapsed into coma, 90 minutes from her initial presentation. At this stage, pupils were bilateral 2 mm and nonreactive, with loss of occulocephalic reflex and dysconjugate deviation of eye. She continued to have bradycardia and hypotension. Repeat CBG was 32 mg/dL and bolus of 50 mL 50% dextrose was repeated.

No history could be gathered regarding the preceding events. At this stage, in addition to malaria, encephalitis, cerebrovascular accident, exogenous insulin administration was considered as another staff detected one empty vial

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of insulin. Blood samples were drawn for glucose, insulin, and c-peptide. Patient had an episode of generalized tonic clonic seizure which was treated with intravenous lorazepam 4 mg. Again a bolus of 50 mL of 50% dextrose was repeated and 10% dextrose infusion started. There was no recurrence of seizure. Patient was transferred to our tertiary care center.

Magnetic resonance imaging (MRI) of the brain was normal. Cerebrospinal fluid analysis was normal except for hypoglycorrhizia (CSF glucose 10 mg/dL and plasma glucose 15 mg/dL). Her other parameters were as follows: Hemoglobin 12.4 g/dL, total leukocyte count 7400 cells, normal differential count, normal urine examination, urine pregnancy test negative, blood urea nitrogen 12 mg/dL, serum creatinine 0.9 mg/dL, sodium 136 mEq/L, potassium 3.9 mEq/L, phosphorous 1.7 mg/dL, magnesium 2.1 mg/dL, calcium 9.1 mg/dL, total bilirubin 0.8 mg/dL, serum glutamic oxaloacetic transaminase (SGOT) 33 IU/L, serum glutamic pyruvate transaminase (SGPT) 31 IU/L, alkaline phosphatase 122 IU/L, total protein 6.9 g/dL, and albumin 4.1 g/dL. Arterial blood gas showed a pH of 7.39, bicarbonate of 22 mmol/L, and pCO_2 of 39 mmHg.

CBG was measured hourly and dextrose infusion rate adjusted to maintain blood glucose around 200 mg/dL. While on dextrose infusion, there were at least two instances when CBG was less than 50 mg/dL, which were managed with 50% dextrose boluses. After 24 hours, Ryle's tube feeding was started with mixed meal preparation. Patient remained comatose for 36 hours after which her sensorium fluctuated. Pulse rate normalized and the ionotropic support was gradually tapered and stopped. Sensorium started to improve gradually and she regained full consciousness with no neurological deficit 90 hours after the initial presentation. Serum electrolytes were measured serially and were within normal range. The graphical representation of blood glucose over 90 hours is depicted in Figure 1. She was diagnosed as a case of depression on psychiatric evaluation and started on antidepressant.

Serum insulin was 402 μIU/mL (for a random sample <112 μIU/mL) and c-peptide was <0.3 ng/mL (1.1–4.4 ng/mL). These results confirmed the diagnosis of exogenous insulin administration as a cause for hypoglycemia in this patient.

Discussion

Hypoglycemia is clinically defined as blood glucose levels low enough to cause symptoms and signs. Biochemically, it is defined as plasma glucose less than 55 mg/dL in healthy adults.[3] However, in diabetics, blood glucose of

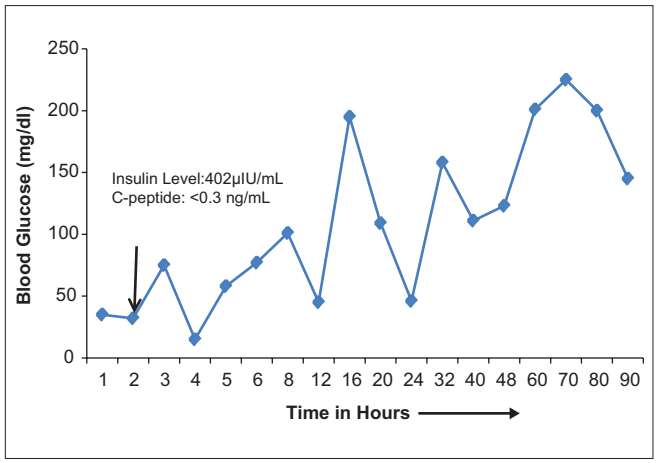

Figure 1: Chart depicting blood glucose values over 90 hours the patient took to recover

less than 70 mg/dL is considered to be hypoglycemia.[3] Confirmation of hypoglycemia is by Whipple's triad – 1) signs and/or symptoms consistent with hypoglycemia, 2) low plasma glucose, and 3) resolution of signs and/or symptoms after rising of plasma glucose. However, if irreversible damage to brain has occurred, the third criterion would be unlikely to be fulfilled.

Hypoglycemia can have varied presentation. Its clinical diagnosis is mainly based on symptomatology. These have been divided as neurogenic and neuroglyopenic. The neuroglyopenic symptoms range from subtle behavioral abnormalities to loss of consciousness and seizures. Hypoglycemia can also present with hypotension and bradycardia as it was in this patient.[4] Diaphoresis with bradycardia and hypotension drives the clinician to search for a cardiovascular cause and hypoglycemia is not thought of until blood glucose is measured.

Hypoglycemia in a non-diabetic can be due to either exogenous or endogenous hyperinsulinemia, in addition to drugs, tumors, critical illness, and hormone deficiencies. Exogenous hyperinsulinemia due to surreptitious use of insulin is diagnosed when during hypoglycemia, insulin level is elevated (at least 3 μIU/mL) with low c-peptide (less than 0.6 ng/mL).[2] Consumption of sulfonylurea was excluded in our patient as there was no increase in c-peptide. Sulfonylurea produces endogenous hyperinsulinemia which leads to elevated c-peptide and insulin as c-peptide is co-secreted in equimolar concentration with insulin from pancreatic β cells.

Exogenous insulin administration as a mode of suicide may be more common in medical and paramedical personnel.[2] In one study, out of the 25 patients managed for insulin overdose, five were non-diabetic health care

professionals.[5] As our patient did not give history probably due to administrative reason, homicidal and accidental administration of insulin is unlikely. Hence, suicidal intent was strongly suspected. It is also more common in patients with psychiatric disorder and in relatives of diabetics.

All types of insulin have been used for suicidal intention, including the short- and long-acting insulins.[6-8] When long-acting insulin is taken, there can be delayed effects. Short-acting insulins can also produce delayed effects. This is explained on the basis of depot effect. Significant reduction in local blood flow results by compression of tissue at injection site, when large quantity of insulin is injected. Delayed effects can also be seen in the presence of renal or hepatic dysfunction. In diabetics, lipoatrophy at the injection site or circulating antibodies against insulin can produce delayed effects.

CSF analysis in hypoglycemia shows low glucose levels.[9] Equilibration of glucose between plasma and CSF takes about 2 hours. CSF glucose reflects plasma glucose from a few hours earlier. But this can be misleading sometimes, especially in septic encephalomeningitis.

High doses of insulin can lead to dyselectrolytemia.[6] Insulin excess leads to salt and water retention and resultant dilutional hyponatremia. There can be intracellular shift of potassium and phosphorous, leading to hypokalemia and hypophosphatemia. Our patient had hypophosphatemia at initial evaluation which spontaneously corrected. Acute pulmonary edema can complicate insulin overdose due to sympathetic activation[10] and hepatic steatosis has also been reported with suicidal insulin toxicity.[11]

Management of hypoglycemia is with dextrose. As the plasma insulin levels increase and reach a level of 50–60 μU/mL, the hepatic glucose output is completely suppressed and glucose needs to be given exogenously. Most patients require dextrose infusions for prolonged period. Whenever an episode of hypoglycemia occurs, it can be treated with boluses of 50% dextrose and at other periods with 5 or 10% dextrose solutions. The average requirement of glucose till full recovery can be anywhere between 160 and 1100 g and the duration of treatment might vary from 12 to 62 hours.[5] Our patient required 470 g of dextrose which was given over a period of 90 hours. If there are no contraindications, Ryle's tube feeding with a mixed meal should be initiated. Dextrose infusion can itself be a cause for excessive insulin secretion especially in non-diabetics and lead to recurrent hypoglycemia. Excision of subcutaneous fat at the injection site has been shown to drastically reduce the dextrose infusion rates.[12]

Conclusion

We have successfully diagnosed and treated a young patient, who presented with coma and hypoglycemia due to exogenously administered insulin with probable suicidal intent. Insulin administration should be kept in differential diagnosis when any case presents with coma and hypoglycemia, especially in paramedical personnel.

References

1. Beardwood JT. A case of attempted suicide with insulin. JAMA 1934;102:765-6.
2. Hawton K, Clements A, Simkin S, Malmberg M. Doctors who kill themselves: A study of methods used for suicide. Q J Med 2000;93:351-7.
3. Cryer PE, Axelrod L, Grossman AB, Heller SR, Montori VM, Seaquist ER, *et al*. Evaluation and Management of Adult Hypoglycemic Disorders: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab 2009;94:709-28.
4. Auer RN, Hall P, Ingvar M, Siesjo BK. Hypotension as a complication of hypoglycemia leads to enhanced energy failure but no increase in neuronal necrosis. Stroke 1986;17:442-9.
5. Mégarbane B, Deye N, Bloch V, Sonneville R, Collet C, Launay JM, *et al*. Intentional overdose with insulin: Prognostic factors and toxicokinetic/toxicodynamic profiles. Crit Care 2007;11:R115.
6. Thewjitcharoen Y, Lekpittaya N, Himathongkam T. Attempted suicide by massive insulin injection: A case report and review of literature. J Med Assoc Thai 2008;91:1920-4.
7. Wong OF, Tsui KL, Kam CK. A case of acute insulin poisoning. Hong Kong J Emerg Med 2006;13:232-4.
8. Tofade TS, Liles EA. Intentional overdose with insulin glargine and insulin aspart. Pharmacotherapy 2004;24:1412-8.
9. Seehusen DA, Reeves MM, Fomin DA. Cerebrospinal fluid analysis. Am Fam Physician 2006;68:1103-8.
10. Ortega E, Wagner A, Caixas A, Barcons M, Corcoy R. Hypoglycemia and pulmonary edema: A forgotten association. Diabetes Care 2000;23:1023-4.
11. Jolliet P, Leverve X, Pichard C. Acute hepatic steatosis complicating massive insulin overdose and excessive glucose administration. Intensive Care Med 2001;27:313-6.
12. Campbell IW, Ratcliffe JG. Suicidal insulin overdose managed by excision of insulin injection site. Br Med J 1982;285:408-9.

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