A Case of Acute Insulin Poisoning

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ABSTRACT

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Clinical presentation of massive overdose of any of the various oral antidiabetic medications available or the various forms of commercial insulin available either accidental or as a result of suicidal intent is possible in the emergency department. The prompt recognition of the condition, knowledge about the various metabolic derangements possible, providing correct management, avoiding rebound hypoglycemia and providing proper monitoring for a prolonged period are major challenges to be overcome. This case report describes a patient who presented with massive overdose of glibenclamide, metformin and insulin and was managed efficiently. The various options in management are discussed.

Keywords: Overdose of Glibenclamide, Metformin and Insulin, Suicidal Intent, Emergency Admission with Antidiabetic Medication Overdose

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CASE STUDY

A 65 year old lady was presented at 4 am in casualty as she was found unconscious in bed. Initially thought of minor episode of hypoglycaemia and was taken to local hospital by relatives. Patient did not receive adequate glucose at the hospital where she was initially taken.

On examination at admission, she was unconscious, but arousable after stimuli. She answered question and confirmed intake of Insulin, and Glucored (Glibenclamide + Metformin) which she had revealed after partial recovery by glucose. She had taken massive doses of Insulin self-administered for suicidal purpose about 1860 units of Humalog Mix and consumed 20 tablets of Glucored 6 hours prior to arrival.

Patient was a known case of chronic diabetes, insulin resistance state and was on high dose insulin regimen.

Laboratory Tests

Random Blood Glucose levels were persistently low for 24 hours.

Serum insulin	764 mmol
C-Peptide	2.4mmol
HbA1c	8.2%

Renal Function Tests (RFT) and Thyroid Function Tests (TFT) were within normal limits. ECG showed nonspecific ST-T changes.

DIAGNOSIS

Patient was diagnosed with Acute Insulin Poisoning leading to Severe Hypoglycaemic Crisis

MANAGEMENT

Patient was treated with continuous glucose infusion, Octreotide Acetate, Potassium Chloride (KCL), NaHCO3 till Serum Insulin measurement came down to 24.5 mmol which required four days of treatment.

DISCUSSION

To assess pre-mortem hypoglycaemic state at the time of dying, it is necessary to establish post mortal concentrations of glucose, lactate and potassium in vitreous humor. Since the advent of insulin for medical treatment, only two cases with diagnosis of suicidal insulin overdose were confirmed at the Institute of Forensic Medicine in Belgrade.

A meticulous search in the English literature reveals that hardly any fatal cases of combined insulin and

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Glibenclamide and Metformin overdose have been reported, with almost no cases from India, thus making this case report relevant and unique.

Heald et al[1] reported most commercial insulin assays fail to detect recombinant insulin analogues. The final diagnosis of death in hypoglycaemic or diabetic coma should always be done as a synopsis of anamnestic response, morphology, biochemical (glucose, lactate, HBA1c, ketone bodies, insulin, and C-peptide) and toxicological findings. High glucose levels in vitreous humor (more than 13 mmol/L, 234 mg/dL) or combined values of glucose and lactate in vitreous humor or in cerebrospinal fluid over threshold values of 23.7 mmol/L, (427 mg/dL) and 23.4 mmol/L, (422 mg/dL) is diagnostic.

Uchida et al[2] reported acute pulmonary oedema caused by hypoglycaemia due to insulin overdose and Berbel et al[3] reported transient cerebral oedema, involving hippocampus and basal ganglia since hypoglycaemia may induce blood-brain-barrier permeability and subsequently brain oedema. The molar ratio of insulin to C-peptide is an aid to the diagnosis of hypoglycaemia due to surreptitious (or inadvertent) insulin administration. After beta-cell stimulation by carbohydrate or other secretagogues, insulin and C-peptide are secreted into the portal vein in a 1:1 molar ratio. ICPR in excess of 1.0 in a hypoglycemic patient argues persuasively for surreptitious or inadvertent insulin administration and against Insulinoma (or sulfonylurea ingestion) as the cause of the hypoglycaemia. Octreotide reverses hyperinsulinemia and prevents hypoglycaemia induced by sulfonylurea overdoses. The molar ratio of proinsulin to insulin was 1:6 in healthy subjects, 1:1 in Insulinoma patients and 10:1 in sulphonylurea induced hypoglycaemic patients. Diagnosis by measurement of serum C-peptide immune-reactivity and insulin-binding antibodies the simultaneous demonstration of low plasma glucose, high immune-reactive insulin and suppressed C-peptide immune-reactivity represents a triad of results pathognomonic of exogenous insulin administration. Glucagon is avoided. The use of glucagon should probably only be considered in the rare circumstance that intravenous access or dextrose are unavailable. Glucagon requires adequate hepatic glycogen stores to be effective, and these stores are likely to be quickly exhausted in patients with insulin or sulphonylurea overdose.

END NOTE

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Conflict of Interest: None declared

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