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Case Report

Octreotide in Sulfonylurea Poisoning

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ABSTRACT

Sulfonylurea overdose/toxicity can cause hypoglycemia which may be because of accidental consumption or suicidal attempt. Sulfonylurea causes hypoglycemia by increasing endogenous insulin release. Here we present how octreotide helps in improving blood sugar levels in refractory hypoglycemia.

Key words: Sulfonylurea, hypoglycemia, octreotide.

INTRODUCTION

Sulfonylureas are used to treat hyperglycemia in type 2 diabetes mellitus. They can be used if blood sugars are not controlled with diet alone or with other oral antihyperglycemic agents (OHAs). They promote euglycemia, but can cause hypoglycemia if patient does not eat adequately after taking the drug or if clearance is impaired because of renal insufficiency. When it is ingested by non-diabetics, it is known to cause hypoglycemia. [1] Sulfonylurea poisoning can produce sustained and profound hypoglycemia refractory to IV dextrose.

CASE REPORT

A 45yr old male non-diabetic presented to casualty with ingestion of 10-12 tablets of metformin 500mg+glimepride 2mg. On admission GRBS was 48mg/dl and vitals were stable. Despite administration of bolus doses of 25% dextrose and infusion of 25%dextrose and 5%dextrose, the patient showed no improvement in hypoglycaemic episodes and demonstrated refractory hypoglycemia. Octreotide 50microgm was given subcutaneously 8hours apart. [2] After the first dose of octreotide blood sugar level rose from 46mg/dl to 122mg/dl in 1hr and later on resulted in reduction of hypoglycemic episodes and reduced need for dextrose administration.

DISCUSSION

Sulfonylureas are absorbed from GI tract. Its absorption can be reduced by food and hyperglycemia. In plasma, sulfonylurea is 90-99% protein bound, mostly albumin. Action of Sulfonylurea is to increase insulin release from pancreas. They bind to a high affinity Sulfonylurea receptor associated with beta cell inward rectifier ATP-sensitive potassium channel. Binding of Sulfonylurea inhibits efflux of potassium through channel and results in depolarisation. Preformed insulin is released into the circulation when depolarisation opens voltage gated calcium channel and leads to calcium influx. [3]
Hypoglycemia is known to occur in sulfonylurea overdose. Patients of hypoglycemia present with headache, giddiness, slurred speech, confusion, lethargy, nausea, convulsion and coma.

Octreotide is a synthetic peptide analogue of somatostatin, binds to G protein coupled somatostatin-2 receptors in pancreatic beta cells which results in decreased calcium influx and inhibition of insulin secretion. Octreotide markedly inhibits insulin release, increases serum glucose concentration, reduces dextrose requirement and prevents recurrent hypoglycemia.

The complication associated with treatment with intravenous dextrose in sulfonylurea induced hypoglycaemia is recurrent hypoglycaemia. Recurrent hypoglycaemia occurs because dextrose administration results in hyperglycemia which in turn potentiates insulin release from the pancreas. Re-administration of dextrose perpetuates this cycle, which causes high dextrose requirements and leads to frequent monitoring of blood glucose levels. [4]

The patient received IV dextrose infusion before and after octreotide treatment. Octreotide 50microgm subcutaneously was administered followed by additional doses. Octreotide significantly increased serum glucose concentration, decreased dextrose requirement and decreased recurrent hypoglycaemic episodes. During octreotide treatment, IV dextrose infusion was gradually tapered off.

CONCLUSION

Octreotide can be used for Sulfonylurea poisoning in refractory hypoglycemic patients not responding to standard dextrose administration.

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