# ACUTE PSYCHOTIC DISORDER AND HYPOGLYCEMIA

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A variable array of neuroglycopenic symptoms are frequently encountered in the hypoglycemic stage, but acute psychotic disorders are quite rare. A fifty five year old female presented with an acute psychosis following oral sulfonylurea induced hypoglycemia without preceding features of adrenomedullary stimulation. This case report suggests that an acute and transient psychotic disorder may be an important neuroglycopenic feature and its early recognition protects the patient from severe hypoglycemic brain damage in a state of hypoglycemia unawareness.

Key words: neuroglycopenic symptoms, hypoglycemia, acute psychotic disorder, hypoglycemia unawareness.

#### INTRODUCTION

Absence of adrenergic symptoms during hypoglycemia is described as hypoglycemia unawareness (Gerich et al, 1991), and is often seen in diabetic patients on strict blood sugar control and in patients with diabetic autonomic neuropathy (Widom et al, 1990). To avoid severe hypoglycemia it is important to recognize the more subtle neuroglycopenic symptoms in such cases. This is an interesting case report of neuroglycopenic symptoms, which may be mistaken for functional psychiatric disorders.

### CASE SUMMARY

A fifty five year old female patient with non-insulin dependent diabetes mellitus (NIDDM) presented with a twelve day history of alteration in behavior. She had been detected to have diabetes mellitus seven years ago and was being treated with oral sulfonylurea (Glibenclamide 5 mg twice daily) which she had been taking infrequently. However, she had been on regular treatment over the last 6 months and her fasting and two hours post prandial sugar level ranged between 90-120 mg/dl and 140-160 mg/dl respectively. Twelve days prior to presentation she developed episodic altered behavior; during this period, her food intake was erratic. She had incomprehensible and incoherent speech with bizarre motor activity which was evaluated by a psychiatrist and a diagnosis of acute and transient psychotic disorder (ICD-10 Code F 23) was made. She was prescribed medication and referred to the endocrine clinic for evaluation of her diabetic status. On examination, she was conscious, non co-operative and showed bizarre motor activity. Her pulse was 90/min, regular, with normal volume. Blood pressure was 140/90 mmHg in the supine posture and 110/70 mmHg while standing. Cardiovascular, respiratory, abdominal, skin and other general examination was normal. Fundus examination showed

background diabetic retinopathy. Plantar reflexes were flexor and no localizing neurological signs could be elicited. Sensory examination could not be

performed. In view of the oral hypoglycemic drug which she continued to take along with inadequate diet, a tentative diagnosis of hypoglycemia was made. After collecting a blood sample for estimation of sugar, 50ml of 50% glucose solution was infused, following which she showed marked improvement. Her initial blood sugar value was found to be 43 mg/dl. She was managed with inj. glucagon 1 mg subcutaneously and intravenous glucose infusions. She gradually improved over a period of 7 days and on re-evaluation her mental functions were found to be normal. Peripheral sensory system examination showed loss of vibration and joint sensations in the distal part of lower extremities. Cardiovascular reflex tests revealed autonomic dysfunction viz., postural fall in systolic blood pressure of 30 mmHg (N 30), Valsalva's ratio 1.05 (N 1.21), 30:15 ratio 0.96 (N 1.1) and heart rate variation to deep breathing 12 beats/min (N 15).

Laboratory investigations during hospitalization included 25 hour urinary protein, serum creatinine and creatinine clearance and the results were 700 mg/24 hrs., 1.1 mg/dl and 90 ml/min respectively. Serum electrolytes, serum calcium and plasma cortisol were within the normal range. Once she reverted to her normal diet, diabetic status was reevaluated and she was treated with inj. crystalline insulin 6 units subcutaneously thrice daily for control of hyperglycemia.

## DISCUSSION

Hypoglycemia evokes a hierarchial response which protects against severe hypoglycemia (Mitrakou et al, 1991; Stevens et al, 1989). Absence of adreno-medullary features before neuroglycopenia (hypoglycemia unawareness) seen in patients on intensive therapy for glycemic control or having autonomic dysfunction (Slater et al, 1986; Gerich et al, 1991) may lead to irreversible brain damage.

The lack of adreno-medullary symptoms of hypoglycemia due to irregular food intake culminated in a clinical state akin to acute and transient psychotic disorder (ICD-10 Code F 23) in the present case. As no organic mental disorder or serious metabolic disturbances were noticed in this patient, psychiatric evaluation favored the possibility of an acute psychotic disorder. The present case report illustrates the importance of screening for hypoglycemia in elderly patients with acute psychotic disorders.

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