Case Report

INSULINOMA PRESENTING AS EPILEPSY AND BEHAVIORAL ABNORMALITY

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Mrs. S. aged 52 years, reported from a nearby village with the following complaints.

Generalized weakness	8 years.
Giddiness	8 years.
Convulsions	6-7years.
Abnormal Behavior	6-7years.

HISTORY

Patient was apparently alright 8 years back, when she started having generalized weakness. Patient used to feel tired even after doing minor work and felt giddy while walking. Giddiness was more in the mornings. There was no history of falling due to giddiness or associated tinnitus or vomiting.

Patient used to get recurrent tonic–clonic convulsions for the last 6-7years. There was no associated tongue bite or history of head injury. Each episode was associated with unconsciousness lasting from few minutes to a few hours. She used to regain consciousness after taking injections and intravenous (IV) fluids. She exhibited abnormal behavior in the form of forgetfulness, inability to identify the relatives and loss of orientation. This type of abnormal behavior used to appear and disappear intermittently. There was no past history of head injury, tuberculosis, diabetes mellitus or hypertension.

For the above complaints patient had consulted many doctors ranging from general practitioners, physicians, psychiatrists and neurosurgeons. She had undergone blood examination, Chest X ray, EEG, C.T. scan and ultrasound examination for the above complaints.

She had been treated with B- complex injections, IV fluids, antiepileptic and anti-psychotic drugs. She was admitted in a psychiatric hospital for 15 days for abnormal behavior. On 17th Sept. 2002, patient was brought to our hospital with history of convulsions.

CLINICAL EXAMINATION

Patient was a moderately built and poorly nourished lady. She was anemic. There was no pedal edema. Her pulse rate was 112/min; B.P:120/80mm of Hg. and her R.R was 18/min. Rest of her general examination was normal.

Examination of the central nervous system showed that she was conscious and oriented to time and space. Patient appeared dull, gloomy and inactive. Pupils: Bilaterally equal 2-3mm and reactive to light. All the cranial nerves were normal. Examination of the motor system, sensory system, cerebellar system and reflexes was normal.

Examination of other systems did not reveal any abnormality.
With this a provisional diagnosis of epilepsy with post-ictal phenomenon was made. Hysteria was considered as the differential diagnosis.

INVESTIGATIONS

1. Hb- 6gm%.
2. Total WBC Count- 8600cells/cu.mm.
3. Differential Count: Poly-86%. Lympho-12%. Eosino-02%.
4. E.S.R.: 120mm 1st hour (Westergren).
5. Urine Examination: N.A.D.
6. Random Blood Glucose: 32mg%.
7. Blood Urea: 32mg%.
8. Serum Creatinine: 0.8mg%.
9. Lipid profile: Normal.
10. E.C.G.-Sinus Tachycardia present.

In view of the severe hypoglycemia without history of intake of oral hypoglycemic agents or insulin, a possibility of insulinoma was considered and serum Insulin estimation was done.

11. Serum Insulin: 105 mU/ml. [Normal Range: 7-18 mU/ml.]

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Subsequently an abdominal ultrasound and abdominal C.T. Scan was done; both of which were normal.

TREATMENT

Patient was treated with 6 ampoules (25 ml each) of Inj. 25% dextrose and 5% dextrose infusion subsequently. She recovered consciousness within a short period, became fully oriented and there were no convulsions after treatment. Patient was later given one bottle of blood transfusion and followed up with iron and folic acid tablets for anemia.

DISCUSSION

Insulinomas are the second most common functioning islet cell tumors of the pancreas. Prevalence of insulinomas is about 1 in 10 lakhs population. They arise most frequently between 5^{th} to the 7^{th} decade of life. The classic presentation of insulinomas includes fasting hypoglycemia, symptoms of hypoglycemia and immediate relief after intravenous glucose infusion, which is known as "Whipple's triad". Symptoms related to hypoglycemia include headache, slurred speech, psychological alterations, visual disturbances, confusion and ultimately coma and death. Hypoglycemia also induces the secondary release of catecholamines leading to tremulousness, diaphoresis, pallor, palpitations, cardiac arrhythmias and behavioral irritability. Because of the episodic release of insulin, symptoms early in the course may be intermittent or occur only after prolonged fasting.

Diagnosis of insulinomas is made by demonstrating fasting hypoglycemia (F.B.S. < 50mg% in men and 45mg% in women) and normal or elevated plasma insulin levels.

Once a diagnosis of insulinomas is established, acute treatment is supportive with intravenous glucose infusion to maintain plasma glucose within normal range. Hyperglycemic agents including diazoxide, β-blockers and phenytoin can be used to support serum glucose levels, but their effects are variable and may last only for a short time Octreotide frequently inhibits insulin secretion by these tumors and may be an effective agent for acute management.

Definitive therapy is accomplished by surgical resection. Because insulinomas are usually small (most are <2 cms in diameter), only half are detected by C.T. scan. Angiography with selective venous sampling for insulin levels is about 80% sensitive and palpation at laparotomy detects 80%—90% of tumors. At surgery detectable tumors should be resected. If no detectable tumors are found, stepwise distal pancreatectomy is performed until frozen sections of resected specimens and blood glucose measurements indicate that the whole tumor has been resected.