Case Report

**Insulinoma-presenting as status epilepticus and hallucinations**

Singal K K¹, Singal N², Gupta I³, Sodhi S⁴, Gupta P⁵, Cheema YS⁶

**Abstract**
Insulinoma is a very rare endocrine disorder. 80% of insulinomas result from benign simple adenomas of pancreas and hypoglycemia is the commonest presenting feature. Although seizures as a consequence of insulinoma are well known, yet status epilepticus as the initial presenting feature is a rarity. Here, we report a young male in whom insulinoma masqueraded as status epilepticus.

**Keywords:** Insulinoma, Status epilepticus, hypoglycemia, hallucination

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**Introduction**
Insulinoma, the most common cause of endogenous hypoglycemia is rare in adults. Insulinoma is a neuro-endocrine tumor derived mainly from pancreatic beta-cells which secrete insulin in addition to other hormones. The symptoms of insulinoma are mainly due to hypoglycemia and about 30% of patients are initially erroneously diagnosed as seizure disorder.¹ The diagnosis of insulinoma is established by the demonstration of fasting hypoglycemia (< 50mg/dl), inappropriate plasma insulin levels (> 6microu/ml) and C-peptide levels (> 0.25nmol/l).² Seizures tend to be more common in children but permanent neurologic sequelae have been observed in about 7% adults.³

**Case Summary**
A 42 year old male, presented with 6 months history of episodic lapses in consciousness associated with incoherent speech, confusion, sweating and palpitations. The patient had been getting such episodes of unconscious spells recurrently once or twice a week. Fourteen months earlier he was referred to the psychiatric department for aggressive behaviour, visual hallucinations and paranoid delusions of 6 months duration. He was initially managed as anxiety disorder with no relief in his symptoms. Moreover, six hours prior to hospitalization, patient had an episode of unconsciousness, accompanied by abnormal body movements, up rolling of eyeballs, tongue bite and incontinence of urine. There was no history of head trauma or complaints pertaining to any other system involvement. Past medical history too was unremarkable except for selective serotonin receptor uptake inhibitors and benzodiazepine intake during last six months. On admission, patient was stuporous with a GCS of 7/15 with bilateral extensor plantar responses. The rest of the general physical and systemic examination was unremarkable. Except for plasma glucose level of 38mg/dl, his routine serum biochemistry including serum electrolytes was normal. During hospital stay, patient was getting recurrent

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1. Dr Kiran Kumar Singal, Professor (Dept. of Medicine) M.M. Medical College & Hospital, Kumarhatti, Solan (H.P.) India
2. Dr Neerja Singal, Professor (Dept. of Obs. &Gynae.) M.M. Medical College & Hospital, Kumarhatti,Solan(H.P.) India
3. Dr.Isha Gupta, Ex.Resident ( Department of Medicine) M.M. Institute of Medical Sciences & Research, Mullana (Ambala) India
4. Dr. Shankar Sodhi ,Ex Resident (Dept. of Medicine) M.M. Institute of Medical Sciences & Research, Mullana (Ambala) India
5. Dr. Parveen Gupta, Ex Professor (Dept. of Medicine) M.M. Institute of Medical Sciences & Research, Mullana (Ambala) India
6. Dr. Yuvraj Singh Cheema, Ex Resident (Dept. of Medicine) M.M. Institute of Medical Sciences & Research, Mullana (Ambala) India

**Correspondence to:** Kiran Kumar Singal, Department of Medicine, M.M. Medical College & Hospital, Kumarhatti,Solan(H.P.) India, e.mail:drkiranambala@gmail.com

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episodes of generalized tonic-clonic seizures with blood glucose levels fluctuating between 38-51mg/dl. His plasma insulin and C-peptide levels were 10.9iµu/l and 30ng/dl respectively. Plasma insulin and C-peptide levels were inappropriate for his blood glucose level of 38mg/dl and USG abdomen revealed a hypoechoic mass in pancreas measuring 9x8mm. His ECG, echocardiography, 24 hour Holter monitoring, CT head (plain) was unremarkable, CECT whole abdomen showed small hypervascular lesion in uncinate process of the pancreas, suggestive of insulinoma pancreas. With above mentioned clinical and laboratory parameters, a diagnosis of insulinoma masquerading as status epilepticus was entertained. The patient was managed conservatively and was referred to surgery department for further management.

Figure: CECT whole abdomen showing small hypervascular lesion in uncinate process of the pancreas suggestive of insulinoma pancreas

Discussion
Insulinoma is a neuro-endocrine tumor derived mainly from pancreatic -islet cells which apart from insulin, secretes gastrin, chorionicβ gonadotropin, corticotrophin, serotonin, somatostatin, glucagon and pancreatic polypeptide. The estimated incidence of insulinoma is one case per 2,50,000 patients years. Insulinoma can be sporadic or familial or a component of the MEN1(multiple endocrine neoplasia) syndrome. Insulinoma has a female preponderance with a 5% rate of malignancy. About 8% of insulinoma patients are diagnosed with multiple endocrine neoplasia type I (MEN1), a syndrome characterized by simultaneous or successive cellular proliferation in at least two endocrine organs. The more commonly affected endocrine organs include the pancreas, parathyroid and pituitary. MEN1 patients present at a younger age (median age of 25 years).In insulinoma there is dysregulated insulin release which leads to hypoglycemia. Normally increased insulin levels and hypoglycemia perse suppress insulin release. In insulinoma, suppression of insulin release by insulin and hypoglycemia is erratic and not appropriately suppressed by hypoglycemia.Clinical features of of insulinoma vary widely and are mainly due to hypoglycemia and rarely due to mass effect. Insulinomas, however, are primarily associated with neuroglycopenic symptoms, and occasionally sympatho-adrenal autonomic symptoms. Neuroglycopenic symptoms include dizziness, amnesia, confusion, personality and behavioural changes, diplopia, seizures, and in some cases stroke and coma. 20%-60% of insulinoma patients are initially misdiagnosed with neurological disorders of which 30% are diagnosed with seizure disorders. Except in late diagnosed malignant cases in which an abdominal mass and signs of metastasis may be present, physical examination is usually normal. Insulinoma can be diagnosed by demonstration of fasting hypoglycemia (< 50mg/dl), inappropriate plasma insulin and C-peptide levels (> 0.25ng/ml). The gold standard is the classic 72 hour fasting test. Hypoglycemia develops in essentially all insulinoma patients during this test, infact 75% will become symptomatic within 24 hours. 99% of insulinoma’s occur in pancreas with equal frequency in head, body and tail. Other rare sites are duodenal wall and gastrospenic omentum. CT scan and MRI of abdomen localize tumour in 50-70% cases. Real-time transabdominal high resolution ultrasonography has a 50% sensitivity, celiac arteriography and transsplenic portal venous sampling are invasive and probably not needed. Currently preoperative transabdominal ultrasonography followed by intraoperative ultrasonography is considered the most specific and sensitive approach which can detect more than 95% of tumors. Surgery is the treatment of choice for insulinoma. Diazoxide given preoperatively reduces the need for glucose supplementation and risk of hypoglycemia. In MEN1, hypercalcemia must be corrected initially by parathyroidectomy followed by tumour resection. Nonsurgical management is contemplated for recurrent and inoperable tumors. Diazoxide (3-5mg/kg/day per oral 8 hourly ) inhibits insulin release from insulinoma, is most commonly used. Phenytoin and Calcium Channel blockers are less commonly used. Octreotide 200-300 subcutaneous bid or qid and continuous
glucagon infusion too are used in selected cases. Hepatic artery embolization, chemo-embolization and chemotherapy with streptozocin and floro-uracil may be considered for malignant tumors.

**Conclusion**

To conclude, insulinoma resulting in hypoglycemia induced seizures and hallucinations should be considered in a patient of late-onset status epilepticus or refractory epilepsy especially with normal neuroimaging. It is important to mention here, that seizures in such cases cannot be adequately controlled unless underlying pancreatic tumor and hypoglycemia is taken care of.

**Ethical Approval:**

This case report was published after getting approval of the Ethics Committee of MMIMSR, Mullana (Ambala), India.

**Conflict of Interest**

No Conflict of interest has been disclosed by the authors.

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**References**