Section: Medicine

Case Report

HYPOGLYCEMIA : TWO DIFFERENT PRESENTATIONS AND COMPLETE RESOLUTION

Saifa M Latheef[1], Shubham Gupta[2], Pankaj Sood[2], Yashpal Singh[3]

[1]*Associate Professor and Clinical HOD, Department of Emergency Medicine, School of Medical Sciences and Research, Sharda hospital, Greater Noida, Uttar Pradesh, India*
[2]*PGY-3, Department of Emergency Medicine, School of Medical Sciences and Research, Sharda hospital, Greater Noida, Uttar Pradesh, India*
[3]*Professor, Deputy Commandant Army Hospital, R&R Delhi, India*
[4]*SMO, Department of Emergency Medicine, School of Medical Sciences and Research, Sharda hospital, Greater Noida, Uttar Pradesh, India*

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Corresponding Author:
Dr. Niraj Kumar,
Department of Medicine, School of Medical Sciences and Research, Sharda hospital, Greater Noida, Uttar Pradesh, India.
Email: mniraj31@yahoo.com

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ABSTRACT

Abstract

Background: Insulinomas are the most common functioning pancreatic endocrine tumors and present with recurrent episodes of hypoglycemia. Symptoms may be neuroglycopenic (altered sensorium, confusion, seizures) or adrenergic (weakness, sweating), which makes the diagnosis clinically challenging.

Case Presentation: We report the clinical profile and management of two otherwise healthy Indian Army soldiers (aged 35 and 28 years) who presented in the fasting state with severe neurological manifestations: recurrent unconsciousness (Case 1) and generalized tonic-clonic seizures (Case 2). Both patients had a positive Whipple's triad. During hypoglycemia, random blood glucose levels were 37 mg/dL and 45 mg/dL, with inappropriately raised insulin (2.8 and 2.4 µIU/mL) and C-peptide (1.09 and 2.08 ng/mL) levels. The sulfonylurea screen was negative, and the glucagon stimulation test was positive, confirming hyperinsulinemic hypoglycemia. Endoscopic ultrasound (EUS) and FNAC identified a neuroendocrine tumor (insulinoma) in the pancreatic head (10 mm) in Case 1 and in the distal pancreatic body (14 × 9 × 13.5 mm) in Case 2. Case 1 underwent a pylorus-preserving Whipple's procedure, and Case 2 underwent tumor enucleation. Both patients experienced complete resolution of hypoglycemia.

Conclusion: A high index of suspicion for hyperinsulinemic hypoglycemia (insulinoma) should be maintained in healthy individuals presenting with unexplained neurological symptoms and fasting hypoglycemia. EUS is an excellent tool for precise preoperative localization, and surgical excision is the definitive, curative treatment.

Keywords: Hypoglycemia; Insulinoma; Neuroendocrine tumor; Endoscopic ultrasound; Whipple's triad; Pancreas.

INTRODUCTION

Insulinomas are the most common functioning pancreatic endocrine tumors, with an estimated incidence of 3 to 10 cases per million people per year.[1] The great majority (about 90 to 95%) are benign, a minority are malignant, and roughly 4 to 8% occur in association with multiple endocrine neoplasia type 1 (MEN1).[2] These tumors secrete insulin autonomously, leading to recurrent episodes of fasting hypoglycemia.

The diagnosis rests on Whipple's triad: symptoms of hypoglycemia, a documented low plasma glucose, and relief of symptoms once glucose is corrected.[5] The symptoms can be neuroglycopenic (diplopia, blurred vision, confusion, altered sensorium, seizures, coma), adrenergic (weakness, warmth, sweating, hunger, tremor, palpitations, anxiety), or a combination of both.[6] Because these features are nonspecific and often intermittent, insulinoma is easily mistaken for a primary neurological, psychiatric, or epileptic disorder, and the diagnosis

may be delayed for months or years. Repeated hypoglycemia can also blunt the adrenergic warning symptoms (hypoglycemia unawareness), so that neuroglycopenic features become the main presentation.

Once hyperinsulinemia hypoglycemia is confirmed biochemically, the next challenge is finding the tumor. These lesions are often small, and preoperative localization succeeds in only about 60% of patients.[3] Non-invasive imaging such as ultrasound, MRI, and multiphase CT has variable sensitivity, particularly for tumors smaller than 1 cm, whereas endoscopic ultrasound (EUS) has a reported sensitivity of 40 to 90% and adds value when combined with CT.[4] Surgical removal is the treatment of choice, and the operation depends on the number and position of the tumor.[6]

We report two otherwise healthy young Indian Army soldiers who presented with different neurological manifestations of fasting hypoglycemia: recurrent unconsciousness in one and a generalized tonic-clonic seizure in the other. Both were eventually diagnosed with a pancreatic insulinoma, localized by EUS and confirmed by FNAC, and both were cured by surgery. These cases highlight the need to measure blood glucose in any young, healthy person with unexplained neurological symptoms, and the role of EUS in localizing small insulinomas.

Case 1:

35 years old male without any known comorbidity became symptomatic first time in Jan 2019 when he felt dizziness while doing his daily activity in afternoon. The dizziness lasted for 10 minutes and was relieved taking some food. Few days after the first incidence he was found unconscious at his work place. He was treated with Intravenous dextrose and improved immediately after infusion. He had 8 episodes of similar incidence and it occurred in fasting state. He also gave h/o blurring of vision and slurring of speech. There was no history of tongue bite, altered sensorium, headache, fever, weight loss, diarrhea, dysuria, shortness breath or chest pain.

There was no significant past medical history. He is an Indian army soldier and used to do strenuous physical activity. There was no h/o alcohol consumption, smoking or any use of illicit drugs.

No other family history of Autoimmune disease, Coronary artery disease, Bronchial asthma or any other chronic disease.

In emergency department on examination his temperature was 99 f, Blood Pressure 126/70 mm Hg, Heart rate 96 per minute, Respiratory rate 16 breath per minute and Oxygen saturation was 98% at room air.

He was drowsy at the time of presentation in Emergency department. His random blood sugar was 37 mg/dl by point of care glucometer and was confirmed later by plasma venous blood glucose. His complete blood count, liver function test, Renal function test, Serum electrolytes and Arterial blood gas analysis was normal. There no positive finding in toxicological screen. His Thyroid function, Prolactin and Serum Cortisol level were normal.

He was given 25% Dextrose through intravenous line. Patient improved and became fully alert after 10 minutes.

Case 2: 28 years old male without any significant past history presented to emergency department with h/o Generalized tonic clonic movements of both upper and lower limbs at his work station. There was no history of tongue bite and Urinary incontinence during the episode. He regained consciousness within few minutes. There was no residual neurological deficit after the episode. On enquiring about the previous similar episodes, he informed there was no similar episodes in the past but had episodes of drowsiness for last six months.

He also informed that symptoms were more in early morning when he was empty stomach and improved with food intake. There was no h/o alcohol consumption, sleep deprivation, fever with altered sensorium or trauma.

There was no significant past history. He is an Indian army soldier with good record of physical activity. He is non-vegetarian and has no addictions.

No h/o Diabetes, Bronchial Asthma, hypo or hyperthyroidism or any h/o Autoimmune disorder in the family

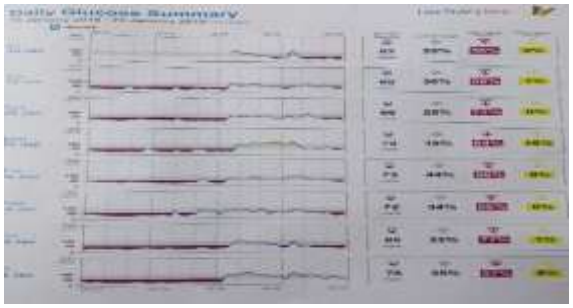
Initial evaluation in emergency department showed Temp 98.6of, Blood Pressure 140/70 mm Hg, Heart Rate 106/min, Respiratory Rate 16/min and saturation at room air 98%. Systemic examination was normal.

His Random blood sugar was 42 mg/dl at the time of presentation by point of care glucometer and was confirmed later by plasma venous blood glucose . His complete blood count, LFT, KFT, Serum electrolytes ABG and toxic screen were normal. He received 25% Dextrose and reported improvement of symptoms in few minutes. His Thyroid function test , Prolactin and Serum Cortisol level were normal.

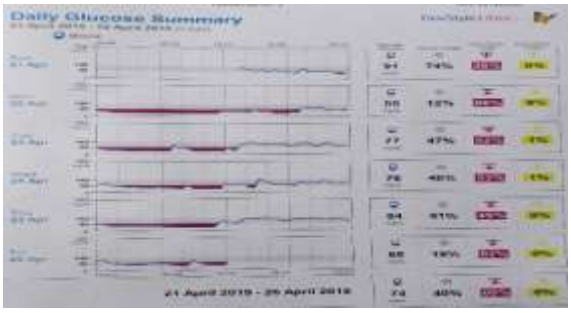
Initial finding of Hypoglycemia with improvement with correction with intravenous glucose confirmed diagnosis of Hypoglycemia (Whipple's triad)5. Simultaneously both cases were also evaluated for toxic ingestion, Neurological abnormality, infection and end stage renal disease. There was no abnormality in above evaluation. Now the challenge was to determine the cause of Hypoglycaemia in two otherwise healthy individuals with neurological manifestation. On subsequent follow up both the patients showed absence of adrenergic symptoms of Hypoglycemia (Hypoglycemia unawareness).

Both were placed on continuous glucose monitoring sensors which showed persistently low sugar values.

Patient 1



Patient 2

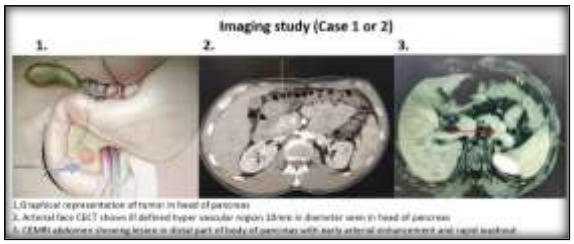


The clinical evaluation in both the patients was suggestive of fasting hypoglycemia due to a hyperinsulinemic state. The patients were planned for evaluation Hyper-insulinemic Hypoglycemia. Critical insulin level showed raised value for both patients (given in table below) confirming the diagnosis Hyper-insulinemic Hypoglycemia which was further confirmed by Glucagon stimulation test.

Investigation

Features	Case 1	Case 2
HB/TLC/PLT Gm per dl/per cmm/per cmm	15.3/5200/170000	15.2/5400/169000
Urea/Creatinine(mg/dl)	21/0.8	24/0.9
HbA1C(%)	4.8	4.6
Basal Cortisol/Post ACTH Microgm per dl	14.9/25.8	13.89/28.9
Free T3/T4/TSH Pg per ng/microgm perml/ng perml	1.32/9.34/2.15	3.28/1.24/1.44
Critical sample monitoring RBS/Insulin/C peptide (mgperdl/microgm per ml/ng perml)	37/2.8/1.09	45/2.4/2.08
Response to Inj 1mg Glucagon	RBS increased from 43 to 100 mg/dl	RBS increased from 45 to 105 mg/dl
Screen for Sulfonyl urea	Negative	Negative

The next step in the work up was to localize the tumor. In first case USG and CECT abdomen was normal. Triple phase CT showed ill defined transiently enhancing lesion in head of pancreas (10mm in diameter) which was confirmed with endoscopic ultrasonography. Endoscopic USG confirmed the lesion localized to head of pancreas. FNAC of this lesion confirmed the diagnosis of Neuroendocrine tumor (Insulinoma). In the second case his CEMRI abdomen showed 14x09x13.5 mm lesion in distal part of pancreas with early arterial enhancement. Endoscopic USG localized the lesion to body of pancreas. FNAC confirmed the diagnosis.





Treatment

After biochemical confirmation both were managed with frequent meals along with Inj Octreotide 100 Mcg SC BD. The improvement of blood glucose with above measures was marginal. The Surgical consultation was taken and planned for surgical removal of tumor.

In the first case tumor was located at head of the Pancreas and underwent pylorus preserving whipple's procedure. In Case 2 tumor was removed through enucleation. Post procedure both were observed for Hypo/Hyperglycemia. Post surgery Both patients had uneventful recovery. There was no recurrence of hypoglycemia. Case 1 developed pancreatic exocrine insufficiency and was put on replacement for the same. Case 2 remained euglycemic with no evidence of endocrine or exocrine insufficiency.

Insulinoma

- Insulinomas are most common pancreatic endocrine tumor with incidence of 3-10 case per million people per year.1
- Most of them (Approx 90-95%) are benign.10 % lesions are malignant, 4-8 % associated with MEN type 1 related insulinoma.2
- They are insulin secreting tumors present with recurrent episodes of Hypoglycemia. Diagnosis based on clinic presentation Hypoglycemia and rapid improvement with correction of Hypoglycemia (Whipple's triad). Symptoms may be Neuroglycopenic (Diplopia, Blurred vision, Confusion, Alerted sensorium, Seizure, Coma) or/and adrenergic (Weakness, Warmth, Sweating, Hunger, Tremor, Palpitations, Anxiety)6.
- Biochemical diagnosis required increased plasma insulin level (> 5 lu/ml), increased C peptide level (> 0.75 pg/ml) during a supervised fast. Measurement of insulin level at low plasma sugar (<45 mg/dl) is called critical sample test6.
- Patient should also be evaluated for MEN1 to rule out Hyperprolactinemia, Hyperparathyroidism, Hypergastrinemia.
- Imaging studies are required after biochemical confirmation. Preoperative localization is possible only in 60%.3
- Non-invasive imaging technique is helpful when tumor size > 1cm. transabdominal USG (9-66%), MRI (40-70%), Multiphase helical CT (50-80).4
- Invasive imaging technique Pancreatic arteriography (25-50%), Intra-arterial calcium stimulation (80-94%), Endoscopic USG (40-90%), Intraoperative USG (83-93%)4

Endoscopic USG can detect insulinoma in the pancreas and yield improved in combination with CT scan. Localization during surgery with ultrasound has diagnostic and therapeutic advantage.[1-3]

Surgical removal of tumor is treatment of choice. Choice of surgery depends on number and position of tumor. For solitary adenoma enucleation is curative with high recurrence rate in patients with MEN1. Difficult location, multiple numbers and malignancy required extensive surgery.[4-6]

Medical management has role during confirmation of diagnosis and preparation of surgery. Frequent small meals, diazoxide (inhibit insulin secretion), Octreotide has been found useful. Continuous Glucagon infusion has been tried in few cases and had short term symptomatic effect.

DISCUSSION

Insulinomas are rare but significant neuroendocrine tumors. Diagnosis relies heavily on establishing Whipple's triad and demonstrating inappropriately elevated plasma insulin (>3-5 µIU/mL) and C-peptide levels during a supervised fasting state or spontaneous hypoglycemic episode. In our patients, the insulin levels (2.8 and 2.4 µIU/mL) were modest but inappropriately detectable and not suppressed at glucose values of 37 and 45 mg/dL. Accompanied by raised C-peptide, a glucagon-induced rise in glucose, and a negative sulfonylurea screen, this pattern conclusively indicated endogenous hyperinsulinism.

Both of our patients were young, fit soldiers whose insulinomas presented with severe neuroglycopenic features (unconsciousness and seizures). Repeated hypoglycemia is known to blunt the autonomic response, causing patients to progress directly to neuroglycopenic features without adrenergic warning signs (hypoglycemia unawareness). This likely explains the severe presentations in two otherwise physically fit individuals.

Localization remains a primary challenge once hyperinsulinemic hypoglycemia is confirmed biochemically. While non-invasive imaging techniques are helpful, EUS provides superior spatial resolution, particularly for tumors smaller than 2 cm. In our report, EUS effectively confirmed the site and facilitated FNAC, highlighting its crucial combined role in localization and tissue confirmation.

Surgical removal is the definitive treatment, with the approach tailored to the tumor's size, number, and location. Enucleation is often curative for solitary peripheral adenomas (as seen in Case 2), while deeper lesions or those in the pancreatic head closely related to the main pancreatic duct may necessitate a pancreaticoduodenectomy (Whipple's procedure), as performed in Case 1. Medical management—including frequent small meals, diazoxide, and octreotide—serves as a vital bridge to surgery but is rarely sufficient for long-term resolution, mirroring the marginal benefit observed in our patients.

CONCLUSION

Insulinoma should be strongly suspected in healthy individuals who present with unexplained neurological symptoms or seizures in the fasting state. Early bedside glucose measurement, critical sample testing during hypoglycemia, and a systematic approach to localization allow for prompt diagnosis. Endoscopic ultrasound (EUS) combined with FNAC is an excellent tool for precise preoperative localization and tissue diagnosis. Tailored surgical excision remains the definitive and curative treatment for insulinomas.

Learning Points

- Measure blood glucose in any young, healthy person with unexplained loss of consciousness or seizure.
- Hypoglycemia unawareness can make neuroglycopenic features the only presentation of insulinoma.
- EUS with FNAC is valuable for localizing and confirming small insulinomas.

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