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CASE REPORT

# Anesthetic management of a case of insulinoma

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## ABSTRACT

Insulinomas are an uncommon cause for recurrent hypoglycemia. Surgical resection is the definitive treatment. We present a case of an 87 year old female patient diagnosed with insulinoma who had history of recurrent hypoglycemic attacks and neurological deficits due to seizure induced hypoxic episodes. Laparoscopic enucleation of the tumor was undertaken. Perioperative management of blood sugar in these patients is of utmost importance. As anesthesiologists our target is to prevent severe hypoglycemic episodes which may lead to permanent neurological damage and severe hyperglycemia post procedure which has to be managed by titrating the dose of insulin and dextrose.

Key words: Insulinoma; Hypoglycemia; Laparoscopy; Neuroendocrine tumors

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## INTRODUCTION

Insulinomas are rare neuroendocrine tumors with a yearly incidence of 1 in 250,000.[1] However, insulinomas are the most common pancreatic endocrine tumors. The most common clinical manifestation is recurrent attacks of hypoglycemia with or without neuroglycopenic symptoms. Surgical resection has always been the treatment of choice since its first detection back in 1924.[2] Inoperable tumors, or patients who are not surgical candidates, may be managed on diazoxide or somatostatin analogues like octreotide.[3,4] The main challenge in the anaesthetic management of an insulinoma is to maintain optimum glucose levels and prevent wide swings in blood glucose perioperatively.

## CASE REPORT

An 87 year old female patient presented with recurrent episodes of loss of consciousness and seizures associated with hypoglycemia since 2 months. A diagnosis of insulinoma was suspected, which was confirmed with low fasting sugar and elevated insulin and C-peptide levels. A CT scan of the abdomen revealed a hypervascular lesion, measuring 11 × 10 mm in the distal pancreas suggestive of an insulinoma. She was then posted for laparoscopic enucleation of the tumor.

On preoperative examination, she was found to have an altered sensorium with a low Glasgow Coma Scale score of 9/15. She was also a known hypertensive on regular medications. Her vital signs were within normal limits. Auscultation of the chest revealed bilateral crepitations. Rest of the systemic examination was normal. Her ECG and echocardiogram suggested left ventricular hypertrophy. A CT scan of the brain revealed ischemic changes in bilateral periventricular white matter (hypoxic damage) with age related atrophy. In view of her present comorbidities, a written informed consent was taken after explaining the possible need for postoperative mechanical ventilation.

Gastric tube feeds were withheld six hours prior to surgery. She was started on 10% dextrose solution at 75 ml/h with 2 hourly monitoring of blood sugar. On shifting her to the operating room, a right subclavian central venous line and a radial arterial line were

secured. Electrocardiogram, pulse oximeter, capnometer and invasive blood pressure monitoring were set up. Preoxygenation was done for 3 min and premedication was done with midazolam 1 mg. A rapid sequence induction was done with 80 mg of propofol and succinylcholine 75 mg. Intubation was done with an oral cuffed endotracheal tube sized 7.5 mm. Fentanyl 100 µg was administered. She was maintained with atracurium and $O_2$: $N_2O$ mixture and 1 % sevoflurane.

Blood sugar was recorded once every 30 min until tumor was reached, and every10 min once tumor manipulation was started. Blood sugar ranged between 90 to 350 mg/dl intraoperatively. Intravenous fluids - 10% dextrose or normal saline were titrated according to blood sugar. The surgery was completed in 90 min and she was shifted to the intensive care unit with the endotracheal tube in situ. She was extubated two hours later when she was fully awake.

Postoperatively, blood sugar was monitored every 30 min for the first 4 h and every 2 h thereafter. Blood sugar ranged between 55-320 mg/dl on the first postoperative day. Insulin therapy was begun if random blood sugar value was above 250 mg/dl. One hypoglycemic episode occurred following insulin therapy. She was administered dextrose/normal saline solution at 100 ml/h and 5% dextrose was given following the episode of hypoglycemia. Somatostatin analogue – octreotide was started immediately after the surgery and then every 8 h.

Her sensorium improved significantly and after one week she was advised to take feeds orally. Until then her sugar ranged between 120-200 mg, while on dextrose/normal saline solution at 100 ml/h. No further episode of hypoglycemia was noted, and she was discharged after normalization of blood sugar.

## DISCUSSION

Insulinoma is an adenoma of beta cells of islets of Langerhans and the most common cause of endogenous hyperinsulinism. The median age of presentation is approximately 47 years with a mild female preponderance.[5,6] The tumors are generally small, solitary and benign (90%). Malignant insulinomas may be associated with multiple endocrine neoplasia type-1 (MEN -1) syndrome.

Whipple first described the pathognomonic triad of symptoms in 1938, e.g. neuroglycopenic symptoms, documented hypoglycemia (random blood sugar < 60 mg/dl) and relief of symptoms following glucose administration.[7,9] The clinical features include headache, dizziness, seizures, amnesia, confusion due to neuroglycopenia and anxiety, tremors, sweating and palpitations due to adrenergic response to hypoglycemia.

The current treatment of choice is laparoscopic resection of the tumor. The main focus of anesthetic management is to prevent hypoglycemia during tumor resection and rebound hyperglycemia after resection. Rebound hyperglycemia, which is usually transient, is expected due to increased levels of anti-insulin hormones like growth factor, glucagon and corticosteroids following tumor resection.[8]

The preoperative examination must include a complete neurological evaluation and all neurologic damage that has occurred due to the hypoglycemic episodes must be documented. Intraoperatively, the signs of hypoglycemia may be masked under the effect of anesthesia. Hence, frequent blood sugar monitoring is extremely vital. Blood sugar monitoring must continue in the postoperative period until normalization of sugar which may take several hours to days. Hypoglycemia in the postoperative period must make one consider the possibility of residual tumor or multiple insulinomas. Hyperglycemia following surgery may warrant insulin therapy. The use of an artificial pancreas has been advocated but its use is limited by its cost.

## CONCLUSION

Although surgical resection of insulinomas is the definitive treatment, meticulous management of perioperative sugar plays a key role in preventing permanent neurological damage and overall outcome of the patient. We reinforce the need for frequent glucose monitoring and prompt administration of sufficient glucose or insulin therapy as required perioperatively.

Conflict of interest: Nil

Author contribution: NS-Concept, conducted the case; HU-Manuscript writing; PV- Conduct the case

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