improvement, and she underwent biopsy of the lesion which revealed chronic granulomatous inflammation, confirming the diagnosis of IGM. She was also found to have hyperprolactinemia at 125.9 ng/ml secondary to a microprolactinoma and was started on cabergoline. Two weeks post treatment, the prolactin level normalized with resolution of galactorrhoea.

CONCLUSION

Hyperprolactinemia is one of the predisposing factors for the development of IGM by increasing inflammation of the breast tissue. When evaluating for IGM, serum prolactin should always be measured to exclude elevated prolactin levels. The cause of hyperprolactinemia should be further investigated and addressed, and treatment with dopamine receptor agonist could reduce recurrence of IGM.

EP_A058

FASTING AND POSTPRANDIAL HYPOGLYCEMIA IN AN ADOLESCENT PRESENTING WITH ENDOGENOUS HYPERINSULINEMIC HYPOGLYCEMIA LIKELY INSULINOMA: A CASE REPORT

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Nashriq Khan Adam Khan and Noor Rafhati Adyani Abdullah
Endocrinology Unit, Department of Internal Medicine, Hospital Sultanah Bahiyah, Kedah, Malaysia

INTRODUCTION/BACKGROUND

Hypoglycaemia is an uncommon clinical problem in individuals without diabetes mellitus. It is a clinical syndrome with various causes in which low plasma glucose leads to hypoglycaemic symptoms and signs that resolve when the plasma glucose is raised. The diagnosis of a true hypoglycaemic disorder requires fulfilment of these specific criteria (Whipple's triad). Once the presence of a hypoglycaemic disorder is verified, a detailed clinical history often suggests a specific underlying cause. Insulinoma is a type of functional neuroendocrine tumour characterized by hypersecretion of insulin, causing hypoglycaemia, characteristically fasting hypoglycaemia. We describe a patient with insulinoma with both fasting and postprandial hypoglycaemia.

CASE

A pre-morbidly healthy 12-year-old male presented with recurrent hypoglycaemia for 3 months. He experienced a severe episode of hypoglycaemia manifesting as seizure with blood glucose of 1.4 mmol/L. Symptoms resolved after glucose administration. He developed progressive weight gain over the course of 4 years. His BP was 120/72, PR 98, BMI 30 with absence of acanthosis nigricans. Blood investigation during clinic visit revealed asymptomatic hypoglycaemia mediated by endogenous hyperinsulinemia, with random blood glucose of 1.7 mmol/L (<3 mmol/L), serum insulin 364.8 pmol/L (>20 pmol/L) and C-peptide 1495 pmol/L (>200 pmol/L). Mixed meal test confirmed fasting hypoglycaemia, with RBS of 2.5 mmol/L, insulin 300 pmol/L, C-peptide of 1145pmol/L. At 120 minutes (postprandial) following the test, RBS was 2.3 mmol/L, insulin 557 pmol/L, C-Peptide 2282 pmol/L. Prolonged supervised fasting test revealed hypoglycaemia after 4 hours with RBS 2 mmol/L, insulin 286.8 pmol/L and C-peptide 1459 pmol/L. Beta-Hydroxybutrate remained suppressed at 0.1 mmol/L following fasting. Sulphonylureas screening was negative. HbA1c was 4.2% and serum Ca (corrected) was 2.22 mmol. CT pancreatic protocol revealed a hypervascular lesion (1.9 x 2.0 x 2.9cm) at the pancreatic head. Diazoxide was initiated to prevent hypoglycaemia and it was well tolerated. The patient is planned for surgical resection of the pancreatic lesion which is likely to be an insulinoma.

CONCLUSION

Recurrent hypoglycaemia requires careful and comprehensive assessment to diagnose a patient. Around 20% of insulinoma patients have both fasting and postprandial hypoglycaemia needing clinical suspicion and prompt assessment to improve outcomes for these patients.

EP_A059

LONG-STANDING ACROMEGALY WITH PERSISTENT DISEASE RESPONSIVE TO PASIREOTIDE: A CASE REPORT

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Eileen Tan,[1] Chee Keong See,[1] Saiful Shahrizal Shudim,[1] Ilham Ismail,[1] Nurbadriah Jasmiad,[1] Zhe Lan Wong,[1] Subashini Rajoo,[2] Mohamad Badrulnizam Long Bidin,[2] Xin-Yi Ooi[2]
[1]*Hospital Sultan Haji Ahmad Shah Temerloh, Malaysia*
[2]*Hospital Kuala Lumpur, Malaysia*

INTRODUCTION/BACKGROUND

In acromegaly patients, chronic hypersecretion of growth hormone from pituitary adenoma results in significant morbidity and mortality. Achieving biochemical control can be challenging, requiring a combination of pituitary surgery, radiotherapy and medical therapy. Pasireotide, a new multireceptor-targeted somatostatin receptor ligand, has a broader binding profile and an increased affinity for SSTR1, 2, 3, and 5 that has demonstrated superiority compared to Octreotide LAR.