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

https://doi.org/10.15605/jafes.038.S2.76

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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

https://doi.org/10.15605/jafes.038.S2.77

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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.

CASE
We report a case of long-standing acromegaly with persistent disease despite pituitary surgery, radiotherapy and Octreotide LAR treatment being switched to pasireotide treatment.

A 63-year-old female was diagnosed with acromegaly 12 years ago with an initial pituitary tumour size of 2.6 x 2.7 x 3.8 cm. She underwent initial transsphenoidal resection of the pituitary tumour but post-operatively she still had a residual tumour of 1.0 x 0.9 x 1.4cm. She received initial medical therapy (Octreotide LAR). However, due to persistent disease and residual tumour, she was then subjected to 11 cycles of radiotherapy. Despite radiotherapy, her serum IGF-1 levels remained elevated and she also developed uncontrolled diabetes. At this juncture, she also refused further pituitary surgery and refused an MRI scan due to claustrophobia. Over the next few years, she would be maintained on Octreotide LAR 40 mg. Her IGF-1 levels would fluctuate slightly but never achieved control. Pasireotide treatment was subsequently started for the patient 1 year ago. After initiation of pasireotide, she had shown significant improvement of serum IGF-1 levels from 628.5 ng/ml to 203.4 ng/ml after 4 months of treatment. She also finally agreed to a repeat MRI pituitary which showed minimal residual tumour.

CONCLUSION
This case demonstrated the difficulty in achieving remission in an acromegaly patient despite surgery and radiotherapy. Despite long standing acromegaly disease and long duration of Octreotide LAR treatment, initiation of pasireotide has benefit to bring patient into biochemical and symptom control.

EP_A060

ATYPICAL PRESENTATION OF FUNCTIONING MALIGNANT METASTATIC PARAGANGLIOMA WITH RECURRENT MYASTHENIA CRISIS

https://doi.org/10.15605/jafes.038.S2.78

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INTRODUCTION/BACKGROUND
The crude prevalence of paraganglioma is 2 per 100,000 persons/year. Roughly 10% of paragangliomas are malignant, resulting in a rare occurrence of 90-95 cases per 400 million people. The usual symptom of paraganglioma is related to catecholamine hypersecretion. However, we report an intra-abdominal paraganglioma presenting as recurrent myasthenia crisis without symptoms of catecholamine hypersecretion.

CASE
We report a case of a 34-year-old female who presented with abdominal pain. On work-up, CT scan revealed a 20-cm intraabdominal mass. Inguinal lymph nodes biopsy revealed reactive tissue. She was then lost to follow-up. She had recurrent admission for myasthenia crisis after 6 months of initial presentation. She received regular plasma exchange during the crisis to which she responded well. Further work-up of the abdominal mass revealed a functioning abdominal paraganglioma. Throughout her hospitalization, patient was normotensive with no symptoms to suggest paroxysm.

CT of the thorax-abdomen-pelvis revealed a 27-cm intra-abdominal multilobulated mass encasing major abdominal vessels and causing mass effect to adjacent organs. There was also compression fracture with lytic lesions of L3 and L4 vertebra. No mediastinal mass was seen. 24-hr urine metanephrines (umol/day): normetanephrine 21.9 (0.0-2.13), metanephrine 200.0 (0.0-1.62), 3-methoxytyramine 251.40 (0.10–1.79). Cervical lymph nodes tissue-biopsy revealed histological features and immunohistochemistry staining in favour of paraganglioma with necrosis and high mitotic figures.

A multi-disciplinary-team discussion was done with surgical, oncology and radio-nuclear for direction of therapy. Unfortunately, debulking surgery was not feasible