Oral cinacalcet and subcutaneous calcitonin were initiated when saline diuresis failed to lower her calcium below 4 mmol/L. Calcitonin was discontinued after 2 days due to intolerability. At the 5th hospital day, serum calcium and creatinine levels decreased to 2.77 mmol/L and 103 umol/L, respectively, with high volume intravenous saline and cinacalcet.

A multidisciplinary discussion was done and the plan was to continue oral cinacalcet and parathyroidectomy at the second trimester. Unfortunately, serial beta-hCG showed decreasing levels and transvaginal ultrasound confirmed fetal nonviability. Left inferior parathyroidectomy was then performed on the same setting at day 7 of presentation. Histopathologic examination reported giant parathyroid adenoma weighing 23 g. Her calcium level normalised and she remained normocalcemic at follow-up 5 months post-surgery.

CONCLUSION

Our case highlights the management challenges for hypercalcemia in pregnancy due to safety concerns on standard pharmacotherapy and surgery. Acute management of severe hypercalcemia in pregnancy requires timely multidisciplinary decisions to achieve the best outcome and minimise morbidity and mortality.

PA-A-40

SEVERE HYPERTRIGLYCERIDEMIA SUCCESSFULLY TREATED WITH INSULIN INFUSION: A CASE REPORT

https://doi.org/10.15605/jafes.037.S2.46

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INTRODUCTION

Patients with severe hypertriglyceridemia (HT) are at risk of developing life-threatening acute pancreatitis and cardiovascular disease. There is no standard guideline on managing severe HT in acute settings. Apheresis, heparin and insulin therapy has been utilised and reported.

CASE

We report a 42-year-old male who was referred to us from private healthcare for severe HT. He has hypertension and dyslipidaemia for 1 year and was prescribed with atorvastatin and fenofibrate for lipid management. He was only recently diagnosed with diabetes mellitus (DM) with HbA1c of 13%. There was no family history of hyperlipidemia or ischaemic heart disease. He did not have any abdominal pain or chest pain. On examination, there were multiple eruptive xanthomatas over the extensor surface of both elbows and knees and at the Achilles tendon area.

Laboratory investigations revealed severe HT with serum triglyceride (TG) of 44.6 mmol/L and total cholesterol of 19.6 mmol/L. His blood exhibited thick and milky supernatant. Serum amylase was not elevated. Liver function test was normal. In view of severe HT, the patient was admitted and variable rate insulin infusion was started. His TG decreased progressively to 22.7 mmol/L by day 2 of admission and finally to 10.7 mmol/L by day 6 of admission. He was discharged on statin and fenofibrate together with his antihyperglycaemic medications. Screening for his family members was also done. Although there was no family history, we planned for genetic study for him soon.

CONCLUSION

This case showed successful therapy for severe HT with insulin infusion. It is non-invasive and cost-effective treatment option for severe HT. Rapid reduction of high TG is important to reduce risk of pancreatitis.

PA-A-41

ACROMEGALY WITH SPONTANEOUS VENTRICULOSTOMY – A RARE PHENOMENON

https://doi.org/10.15605/jafes.037.S2.47

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INTRODUCTION

Spontaneous ventriculostomy is a unique condition that occurs in patients with chronic obstructive hydrocephalus wherein spontaneous ventricular rupture results in communication between the ventricular system and subarachnoid space. We present a case of acromegaly who presented with spontaneous ventriculostomy on magnetic resonance imaging (MRI) of the pituitary with no prior history of neurosurgical intervention.

CASE

A 66-year-old male with chronic hypertension presented with long standing bilateral peripheral vision loss, worsening over the right eye for 3 months, obstructive sleep apnea symptoms and no symptoms of pituitary apoplexy. Clinically, he had classical features of acromegaly and ophthalmological assessment confirmed bitemporal hemianopia with no optic atrophy.

Pre-operative investigations confirmed elevated IGF-1: 240 ng/ml (NV: 40.2 - 225), random growth hormone (GH): 3.7 ng/ml (NV: <3) and prolactin (PRL): 805 mIU/L (NV: 45 - 375). Thyroid and cortisol levels were normal. Pre-operative MRI of the pituitary revealed a heterogenous sellar mass with cerebrospinal fluid (CSF) fistulous connection between the floor of third ventricle and sella turcica. He underwent endoscopic chiasmopexy which revealed dense arachnoid adhesions in the sellar region with CSF gush on manipulation. No tumour was removed as a discrete adenoma could not be identified. Post-operatively, IGF-1 normalised to 184 ng/ml (40.2-225), but GH and PRL remained elevated at 4.9 ng/ml and 474 mIU/L, respectively. Failure of GH suppression following 75 g OGTT at 2.8 ng/ml denotes persistent active acromegaly. As he had mild acromegaly with prolactin co-secretion and was unable to afford somatostatin receptor ligand therapy, he was commenced on cabergoline 0.25 mg twice a week.

CONCLUSION

Preceding case reports of spontaneous ventriculostomy secondary to obstructive hydrocephalus were due to neoplastic disease or benign aqueduct stenosis. We report a case of acromegaly with prolactin co-secretion who presented with a rare finding on pituitary MRI of spontaneous ventriculostomy.

PA-A-42

NON-FUNCTIONING PITUITARY ADENOMA COMPLICATING PREGNANCY

https://doi.org/10.15605/jafes.037.S2.48

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INTRODUCTION

Pregnancies are rare in women with pituitary adenoma. We describe a patient with non-functioning pituitary adenoma who had spontaneous pregnancy. She developed progressive worsening of vision and underwent successful transsphenoidal surgery (TSS) in the second trimester.

CASE

A 35-year-old female presented with secondary amenorrhoea and galactorrhoea for 6 months. A large pituitary macroadenoma with mass effect coupled with mild increment in prolactin raised a suspicion of prolactinoma. Menstruation returned and galactorrhoea ceased following cabergoline therapy. She had a left nasal hemianopia, central hypothyroidism and hypogonadotropic hypogonadism. The other pituitary hormones were intact. Although prolactin remained suppressed for a year with cabergoline, there was no reduction in tumour size and serial perimetry showed gradual worsening of visual fields in both eyes. She was scheduled for TSS but was postponed.

She presented at 17 weeks of a spontaneous pregnancy with left eye discomfort and no other symptoms of raised intracranial pressure. MRI of the pituitary showed unchanged tumour size exhibiting mass effect with no evidence of apoplexy. Perimetry showed worsening of peripheral scotoma on the left eye with optic atrophy. Hormonal evaluation showed new onset central hypocortisolism. She underwent TSS at 19 weeks POA. Histopathology confirmed pituitary adenoma but stained negative for prolactin. Post-operatively, she developed central diabetes insipidus requiring regular desmopressin. There was no improvement in vision.

CONCLUSION

Pregnancy is a known risk factor for pituitary apoplexy. However, worsening of mass effect could be due to physiological changes of pregnancy resulting in pituitary enlargement. Surgery is recommended early prior to pregnancy especially in those with large non-functioning pituitary adenoma for optimal pregnancy outcomes. Patients who become pregnant require multidisciplinary care and TSS in the second trimester is an option. Hormonal deficiencies and cranial diabetes insipidus should be addressed promptly.