infundibulum. She underwent a biopsy of the lesion and the HPE confirmed pituitary lactotroph adenoma. She was started on cabergoline and her prolactin levels reduced significantly.

CONCLUSION

Ectopic pituitary adenomas occurring in the clivus are very rare. The main differential diagnosis to be considered is chordoma. Other than radiological imaging, additional endocrinological workup may be useful as well to establish the diagnosis. Biopsy of the lesion should be performed whenever the diagnosis is in doubt, as medical treatment with cabergoline often yields favourable outcomes in cases of ectopic prolactinoma which leads to a reduction in tumour size and prolactin level.

EP_A124

BATTLING THE UNCOMMON: A CASE REPORT ON ECTOPIC ACTH SYNDROME FROM PANCREATIC NEUROENDOCRINE TUMOUR WITH LIVER METASTASES

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INTRODUCTION/BACKGROUND

Pancreatic neuroendocrine tumours (pNETs) are rare malignancies originating from the islet cells of the pancreas, comprising only 1%-2% of pancreatic tumours. Among functional pNETs, insulinoma and gastrinoma are the most common, while ACTH-secreting tumours are very rare. Ectopic ACTH syndrome (EAS) caused by pNETs is particularly aggressive, often presenting with metastatic disease, primarily to the liver.

CASE

We present a case of a 25-year-old male who presented acutely with upper gastrointestinal bleeding. Clinical examination revealed features consistent with Cushing's syndrome, and further investigations identified an ACTH-secreting neuroendocrine pancreatic tumour with liver metastasis. The disease was complicated by extensive inferior vena cava thrombosis and concurrent thrombocytopenia, necessitating the insertion of an IVC filter. The patient underwent distal pancreatectomy, splenectomy and wedge resection of the stomach. Hypercortisolism was controlled with steroidogenesis inhibitors, including metyrapone, ketoconazole and the newer agent, osilodrostat. Post-operatively, he underwent a Ga-68 Dotatate scan which showed evidence of somatostatin-receptor avid metastatic disease in the liver and he was started on a somatostatin analogue.

pNETs typically localized in the head and body of the pancreas with an average size of 4.6 cm (2.5–7 cm) and the source of ectopic ACTH secretion may remain hidden for several years. In our case, the pancreatic lesion measured 11 cm which was larger than the average size. Despite the challenges posed by the tumour's size and complications such as IVC thrombosis, the multidisciplinary approach involving endocrinologists, surgeons and radiologists allowed for effective management. Surgical intervention was complemented by medical therapies such as metyrapone, ketoconazole, lanreotide and eventually osilodrostat to control hypercortisolism. Osilodrostat demonstrated good efficacy in reducing cortisol levels in our patient.

CONCLUSION

This case highlights the aggressive nature of EAS-pNETs and the challenges in the management, particularly when presenting with advanced metastatic disease. The use of osilodrostat represents a promising advancement in the management of EAS.

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CUSHING'S DISEASE AND THROMBOSIS: A CLINICAL PERSPECTIVE

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INTRODUCTION/BACKGROUND

Cushing's disease is the most common cause of endogenous Cushing's syndrome, which is caused by an adrenocorticotropin (ACTH) -secreting pituitary tumour. It poses a myriad of complications due to the state of excess cortisol levels, including thrombosis. It is interesting that thrombotic risk due to the hypercoagulable state in Cushing's disease is higher after pituitary surgery when cortisol levels are diminished.

We present a case of a female with recurrent Cushing's disease who developed extensive thrombotic complications post-successful transsphenoidal surgery (TSS) to highlight the importance of anticoagulation therapy in mitigating the risk of thrombosis.

CASE
A 31-year-old female with underlying diabetes mellitus, hypertension and a history of previous treatment for Cushing's disease presented with symptoms of weight gain, hirsutism and purplish striae for 2 years. MRI showed a pituitary adenoma measuring 2.8x4.4x3.3 mm. A 24-hour urinary cortisol and overnight dexamethasone suppression tests were not suppressed. She underwent endoscopic TSS. Post-surgery, her cortisol levels reduced from 419 to 57.3 nmol. Subsequently, she was found to have a saddle pulmonary embolism and extensive right lower limb deep vein thrombosis requiring pulmonary thrombectomy. Post-procedure, she was started on anticoagulants.

Hypercoagulation in Cushing's disease is due to the increase in clotting factors II, V, IX, and VIII, fast-acting plasminogen activator inhibitors and the decrease of tissue-type plasminogen. The stress post-surgery causes an abnormal Von Willebrand Factor pattern production leading to platelet aggregation and the drop in cortisol levels will trigger an inflammatory response that initiates the coagulation cascade. The elevated thrombotic risk will decrease after 3 months to a year later as the glucocorticoid effect takes time to wean off, hence requiring anticoagulation.

CONCLUSION
Recognition of thrombosis post-surgery for Cushing's disease is vital to prevent mortality and morbidity. An individualized strategy based on the degree of thrombosis is therefore essential in the management.

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HYPONATRAEMIA SECONDARY TO SIADH: COULD IT BE METHAMPHETAMINE USE?

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INTRODUCTION/BACKGROUND
Hyponatraemia can be a result of SIADH. Patients with methamphetamine use frequently present with hyponatraemia, possibly secondary to SIADH, among other complications.

We describe a patient with a history of methamphetamine use presenting with persistent hyponatraemia secondary to SIADH.

CASE
A previously well, 50-year-old male with a history of methamphetamine usage (last intake 30 years ago), presented with generalized body aches and weakness, vomiting, reduced oral intake and constipation for 1 week. On examination, his GCS was full, blood pressure was 120/72 mm Hg, heart rate was 72 and afebrile. Other systemic examinations were unremarkable. Blood parameters showed hyponatraemia with hypokalaemia (sodium 121 mmol/L, potassium 3 mmol/L). Despite 4 days of intravenous drip hydration and oral sodium chloride, his clinical condition and sodium levels did not show any improvement. His lowest sodium was 110 mmol/L, and hyponatraemic workup was consistent with SIADH (serum osmolality: 238 mOsm/kg, urine osmolality: 819 mOsm/kg and urine sodium: 187 mmol/L). Morning cortisol, thyroid function test, renal profile, Synacthen test, ACTH and tumour markers were normal. Patient was diagnosed with symptomatic hyponatraemia secondary to SIADH due to methamphetamine. Subsequently, he was started on intravenous hypertonic saline for 2 days coupled with fluid restriction of 500 mL/day. In view of imperceptible improvement of clinical symptoms and sodium level (115 mmol/L), fludrocortisone 0.1 mg tablet bid was then added. After more than 1 week of treatment, the peak serum sodium level achieved was 120 mmol/L.

Literature showed that amphetamines can be associated with serotonin-mediated hyponatraemia. This can happen as a result of SIADH or excessive water intake from hyperpyrexia following drug ingestion.

CONCLUSION
This case illustrates possibility of hyponatraemia secondary to SIADH which could be a result of methamphetamine use. Absence of urine toxicology test upon patient's presentation causes difficulty to confirm this diagnosis, and the possible duration of effect of methamphetamine-induced SIADH is yet unknown. Nevertheless, a history of recreational drug consumption should be included in the clinical evaluation of unexplained hyponatraemia.