treatment options indicated that estrogen-based therapies have demonstrated efficacy in females when initiated before age 11, while the use of testosterone or high-dose estrogen in males remains less well-studied. Given the patient's advanced pubertal status (Tanner stage V) and chronological age, therapy was considered unlikely to be effective. Nevertheless, a shared decision-making process emphasizing supportive management, monitoring, and counseling was undertaken with the patient and his family.

CONCLUSION

The case highlighted the importance of early identification and timely referral for growth-reductive therapy in patients with Marfan Syndrome. Height reduction strategy has been promising, however data is limited with relatively small sample size, with inconclusive evidence on growth-suppressing therapies among male patients.

EP_A119

EXPLORING HYPERGLYCEMIA-RELATED SEIZURES: A CASE SERIES

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INTRODUCTION/BACKGROUND

Hyperglycemia-related seizures, though rare, represent a serious complication of uncontrolled diabetes, often occurring in the context of Hyperosmolar Hyperglycaemic State (HHS). We present a retrospective case series detailing the clinical characteristics, metabolic parameters and outcomes of patients admitted with hyperglycemia-related seizures.

CASE

Seven patients (4 females, 3 males) were included, with a median age of 64 years (range 16–76). All except one, a known defaulter, were on insulin therapy. Glycemic control was poor, with a median HbA1c of 9.9% (range 7.4 – 19.8). Random blood glucose levels at presentation ranged from 15 to 48.8 mmol/L. Serum sodium ranged from 120 to 150 mmol/L and serum osmolality ranged from 292.2 to 361.1 mOsm/kg. Three had HHS and one had overlapping diabetic ketoacidosis. Generalised tonic-clonic (GTC) seizures were the most common presentation (n = 6), while one had focal seizures.

Only one patient had a prior history of stroke; none had known epilepsy. Three patients required intubation for airway protection. Potential confounders included dementia (n = 2), hypertensive crisis (n = 1), liver cirrhosis (n = 2, including one with history of substance abuse) and sepsis (n = 1). Brain CT most commonly showed cerebral atrophy with small vessel disease (n = 4); two had concurrent multifocal infarct. Two EEGs were performed, showing no epileptiform changes. Most patients achieved seizure control following normalization of blood glucose. Three patients were started on antiepileptic medications, and two of these patients were discharged on the same medications. The mortality rate was high, with three deaths occurring during the study period. One of these patients developed a total anterior circulation infarct.

CONCLUSION

Our findings suggest that GTC seizures are more prevalent in patients with severe hyperglycemia. The absence of epileptiform activity on EEG supports a metabolic etiology. Early recognition, aggressive glycemic management and comprehensive post-discharge follow-up are important. These measures may improve neurological outcomes and reduce the high mortality associated with this complication.

EP_A120

PRIMARY ALDOSTERONISM IN PREGNANCY: A CASE REPORT

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INTRODUCTION/BACKGROUND

Primary aldosteronism (PA) in pregnancy is a rare and potentially severe disorder that poses significant challenges for diagnosis and treatment. Compared to essential hypertension, PA is associated with increased risks of preterm delivery, fetal growth restriction, and preeclampsia due to hypertension and hypokalemia.

CASE

We present a 34-year-old primigravida with confirmed PA of ten years duration marked by recurrent episodes of hypokalemia and hypertensive urgency. Despite the initiation of mineralocorticoid receptor antagonist (MRA) treatment, the patient's blood pressure remained poorly controlled, and the patient also had irregular follow-up. She presented at 8 weeks of gestation with uncontrolled hypertension. She required multiple antihypertensive medications with maximal doses as pregnancy progressed, including methyldopa, labetalol, and nifedipine, but BP control remained suboptimal. Imaging revealed a left adrenal nodule, leading to retroperitoneoscopic adrenal-

ectomy in the second trimester. Postoperatively, her blood pressure improved moderately, but she developed severe preeclampsia at 26 weeks, necessitating an emergency caesarean delivery and her premature infant did not survive. She remained hypertensive post-adrenalectomy and post-partum, suggesting concomitant essential hypertension.

Managing PA in pregnancy is difficult because MRAs have adverse effects in pregnancy, and other antihypertensive drugs have limited ability to lower aldosterone-mediated hypertension. This case illustrates the problems of achieving tight blood pressure control in pregnancy and consequent maternal and fetal complications. Surgical adrenalectomy may not completely alleviate hypertension during pregnancy because of ongoing vascular remodelling from chronic aldosterone excess. Compared with essential hypertension, PA in pregnancy carries a larger risk of unfavourable outcomes, including preeclampsia, IUGR and placental insufficiency due to aldosterone's direct endothelial and pro-inflammatory effects. Despite adrenalectomy, this patient still developed preeclampsia, emphasizing the persisting vascular dysfunction even after surgery.

CONCLUSION

Careful management of primary aldosteronism (PA) during pregnancy is crucial to reduce complications. Adrenalectomy may improve blood pressure control, but it does not ensure protection from adverse outcomes. Multidisciplinary care and continuous monitoring are therefore necessary.

EP_A121

PITUITARY HYPOPLASIA PRESENTING WITH HYPOPITUITARISM: A CASE REPORT

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INTRODUCTION/BACKGROUND

Hypopituitarism is a deficiency of one or more hormones secreted by the anterior or posterior pituitary gland. It is a rare condition, with a prevalence of 46 cases per 100,000 population. It can be caused by several conditions, but it is rarely caused by pituitary hypoplasia.

CASE

A 19-year-old female presented to the hospital with concerns of short stature and delayed puberty. She reported never having experienced menstruation and a lack of breast development. The patient denied headache and there was no reported history of hormonal abnormalities or previous medication use. Her intellectual abilities were noted to be well-developed.

On examination, the patient's height was 135 cm and her weight was 32 kg. Her genetic height potential was estimated to be between 142.5 and 159.5 cm. She exhibited no signs of puberty (Tanner stage I).

Laboratory results revealed: LH <0.09 mIU/mL, FSH 0.69 mIU/mL, estradiol <10 pg/mL, TSH 4.1 mIU/mL, FT4 5.52 pmol/L, IGF-1 21 ng/mL, and cortisol 1.8 µg/dL. Bone age was assessed as equivalent to a 13-year-old female, with an open epiphyseal plate. Gynecological ultrasound showed a small uterus measuring 5.47 x 2.33 cm. Brain MRI revealed pituitary hypoplasia (6.9 x 3.9 x 7.5 mm) with no other identified abnormalities.

Based on these findings, the patient was diagnosed with pituitary hypoplasia and hypopituitarism (hypogonadism, hypothyroidism, central hypothyroidism, adrenal insufficiency). Treatment was initiated with estradiol valerate 2 mg, levothyroxine 25 mcg, and hydrocortisone 20 mg. Within six months, the patient experienced menstruation and breast development.

CONCLUSION

We have treated a patient with hypopituitarism secondary to pituitary hypoplasia. We hypothesize that a genetic defect caused pituitary hypoplasia in this patient. The patient has had a positive outcome and continues to receive routine follow-up care at the hospital for hormone replacement therapy.

EP_A122

REASSESSING MEN 1 P.Ala541Thr: NON-DELETERIOUS POLYMORPHISM OR UNDERESTIMATED RISK?

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INTRODUCTION/BACKGROUND

Multiple endocrine neoplasia type 1 (MEN 1) is an autosomal dominant hereditary tumor syndrome caused by inacti-