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-