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-

vating mutations in the MEN 1 tumor suppressor gene. Germline mutations in MEN 1 show high penetrance and account for approximately 70–80% of diagnosed MEN 1 cases. Several polymorphisms have also been identified within the MEN 1 gene region, with at least 12 benign variants reported in the general population. While these variants are typically considered non-pathogenic, the c.1621A>G variant has been reported in some studies as potentially contributing to a low-penetrance MEN 1 phenotype in certain carriers.

**CASE**

We report a case of a 56-year-old female who presented with a 3-month history of painless jaundice and anorexia. She had no personal or family history of malignancy or endocrine disorders. Investigations revealed cholestatic jaundice (bilirubin 89 µmol/L), and hypercalcemia (2.88 mmol/L). Imaging showed a solitary 1.7 cm enhancing pancreatic head lesion. Biochemical workup indicated primary hyperparathyroidism (intact-PTH 22.9 pmol/L [normal range; 1.96 – 8.49], calcium/creatinine clearance ratio 0.04). She underwent Whipple's procedure, and histopathology confirmed a 2.1 cm grade 1 pancreatic neuroendocrine tumor (T2N0). Gallium-68 PET/CT showed no distant disease but identified a right lower thyroid lobe focus, suggestive of a parathyroid adenoma. Pituitary MRI was unremarkable.

She met the 2 hallmark features for MEN 1; primary hyperparathyroidism and a pancreatic neuroendocrine tumor, although her presentation occurred later than is typical for MEN 1 cases. The whole exome sequencing showed no pathogenic MEN 1 mutation but detected a c.1621A>G variant that is classified as non-deleterious polymorphism. Interestingly, pathogenic variants in TP53 and BRCA1 were identified without phenotypic expression to date.

**CONCLUSION**

This case raises questions about the possible pathogenic role of the MEN 1 c.1621A>G variant, especially considering previous reports linking it to low-penetrance MEN 1. Its coexistence with mutations in TP53 and BRCA1 further suggests potential gene-gene interactions or modifier effects, warranting further investigation.

## EP_A123

### METASTATIC POORLY DIFFERENTIATED THYROID CANCER: A CASE REPORT

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**INTRODUCTION/BACKGROUND**

Poorly differentiated thyroid carcinoma (PDTC) is an aggressive subtype of thyroid cancer, representing 0.23%–2.6% of cases.1 Due to its rarity, the role of thyroglobulin (Tg) monitoring and the effectiveness of radioactive iodine (RAI) ablation have not been clearly defined. Elevated Tg levels in PDTC are associated with higher recurrence suggesting prognostic significance. While RAI avidity is variable, 25% of PDTC cases maintain the ability to uptake iodine. In such cases, RAI ablation significantly improves survival after thyroidectomy.

**CASE**

A 51-year-old female presented to a private hospital with a one-year history of neck swelling in April 2023. Initial blood investigations, including thyroid function tests were normal and she was advised that no surgical intervention was necessary. There was progressive enlargement of the neck, and by January 2024, she developed airway compression. CT scan showed a large multinodular goiter, an ill-defined hypodense mass in the left thyroid lobe and pulmonary nodules measuring 0.5–2 cm suggesting metastases. She underwent total thyroidectomy and histopathology confirmed PDTC with lymphovascular spread (pT3aNx, high risk).

She was referred to Endocrinology post-thyroidectomy and was started on TSH suppression therapy and given RAI ablation (150 mCi) in April 2024. Baseline stimulated Tg was >500 ng/mL with negative anti-Tg antibodies.

**CONCLUSION**

This is the first case of PDTC in our center. A multidisciplinary team was important in management. Our case highlights the prognostic role of Tg, the need for more evidence on the efficacy of each treatment modality and the importance of a standardized treatment algorithms for PDTC.