

## Adult E-Poster

decreased to 2.44 mmol/L after six doses of calcitonin. Eventually, she delivered vaginally at 38 weeks with no complications; the neonate weighed 2.58 kg.

A 33-year-old primigravida developed hypertension at 22 weeks, progressing to preeclampsia with pulmonary oedema and blurred vision at 27 weeks, requiring emergency caesarean section. The neonate weighed 980 g; placental histology showed vascular malperfusion and retroplacental haematoma. Hypertension persisted post-delivery. Retrospectively, there was an unrecognised hypercalcaemia detected from 26 weeks gestation. Postpartum laboratory showed calcium 3.04 mmol/L, phosphate 0.67 mmol/L and intact PTH 20.3 pmol/L. Ultrasound and Tc-99m Sestamibi confirmed a left inferior parathyroid adenoma. She underwent a left parathyroidectomy, and she was immediately weaned off antihypertensives.

### CONCLUSION

PHPT in pregnancy poses significant maternal and foetal risks. Medical therapy options are limited during pregnancy. Early recognition and tailored management are crucial to minimise maternal and foetal complications.

## EP\_A092

### WHEN THE CURE BITES BACK: A CASE REPORT ON CARBIMAZOLE- INDUCED MYOSITIS

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### INTRODUCTION/BACKGROUND

Musculoskeletal complaints, including myositis, are common in thyroid disorders. However, it is a rare and non-classical side effect of antithyroid drugs.

### CASE

A 35-year-old male with Graves' disease was admitted for symptomatic thyrotoxicosis despite treatment with high-dose carbimazole, lithium and dexamethasone for direct observed therapy (DOTS). On examination, he exhibited signs of thyrotoxicosis, including tremors, sweaty palms, proptosis and a large goitre. Thyroid function tests revealed overt thyrotoxicosis (TSH <0.001 mIU/L, T4 122.5 pmol/L). He responded well to DOTS on the same treatment, with T4 decreasing to 27 pmol/L by Day 10 of admission. However, he developed severe proximal muscle aches on day 10. Laboratory investigation revealed an elevated creatinine

kinase (CK) level of 5662 IU/L. A diagnosis of carbimazole-induced myositis was made, prompting the discontinuation of carbimazole and initiation of intravenous hydration and cholestyramine while continuing lithium and steroids. The myositis improved, but there was a rebound in his thyrotoxicosis (T4 52pmol/L), prompting a trial of propylthiouracil, which led to a flare of the myositis (CK 1328 IU/L). He was planned for urgent total thyroidectomy then and was started on Lugol's iodine.

Myalgia and elevated CK levels are rare but recognised manifestations of hyperthyroidism. These can be due to direct effects of thyrotoxicosis on skeletal muscle, thyrotoxic hypokalemic periodic paralysis and drug-induced myositis by carbimazole and propylthiouracil. The pathophysiology of drug-induced myositis may be due to the abrupt decrease in circulating thyroid hormones, leading to a relative hypothyroid state in the peripheral tissues and resulting in myositis. Thioamides may also have a direct toxic effect on myocytes and trigger a local immune response at myocytes, leading to myositis.

### CONCLUSION

This case highlights myositis as a rare side effect of anti-thyroid drugs and can cause significant morbidity. Early recognition and close monitoring are essential for managing this condition.

## EP\_A093

### A RARE CASE OF HYPONATREMIA AS A LEADING SIGN OF EMPTY SELLA SYNDROME

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### INTRODUCTION/BACKGROUND

Hyponatremia is a common electrolyte disturbance that can be life-threatening. Careful evaluation of hyponatremia may reveal significant underlying conditions and may lead to the diagnosis of a rare multiple hormone deficiency, with detrimental consequences for the patient if left untreated. We present an interesting case of a patient who presented with symptoms of chronic hyponatremia, and the results of the examination led us to the discovery of partial empty sella syndrome as the aetiology.

### CASE

A 61-year-old female presented with a six-year history of general weakness, nausea, vomiting and cold intolerance.