testosterone level of 5.74 nmol/L (NR: <1.67). Other blood investigations were normal. Further investigation for the hyperandrogenism from possible androgen-producing tumour was postponed till post-delivery. However, during the 6-month follow-up post-delivery, she had regularised menses and reduced facial hair, which minimised regular shaving. Her repeat testosterone level taken 4 months post-delivery was 0.49 nmol/L.

CONCLUSION

Elevated testosterone during pregnancy is a normal physiological response vital for maintaining pregnancy and initiation of parturition. It is caused by the increased production and reduced clearance of testosterone. Excess testosterone during pregnancy does not cause clinical hyperandrogenism as a result of increased SHBG, which binds the androgens, and placental aromatase, which converts excess testosterone to estradiol. However, PCOS can result in a diminished protective effect of the placenta aromatase, resulting in clinical hyperandrogenism during pregnancy. Our patient had a pre-pregnancy PCOS diagnosis, which worsened her hyperandrogenism intrapartum. This condition was similar to a few published case reports. The resolution of PCOS symptoms post-delivery can transiently happen due to the stabilisation of hormones postpartum; unfortunately, PCOS symptoms may recur later on.

EP_A061

WHEN THE THYROID AND STOMACH COLLIDE: APS TYPE 3B BEHIND CARDIAC SYMPTOMS

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INTRODUCTION/BACKGROUND

Autoimmune Polyglandular Syndromes (APS) are a group of disorders characterised by the simultaneous or sequential occurrence of multiple autoimmune-mediated diseases affecting endocrine glands. Pernicious anaemia is commonly part of this broader spectrum of autoimmune conditions.

CASE

We report the case of a 65-year-old male with a seven-year history of megaloblastic anaemia treated with cyanocobalamin, who presented with severe anaemia-induced non-ST-elevation myocardial infarction (NSTEMI) that manifested as chest pain, reduced exercise tolerance and profound fatigue. Initial investigations revealed pancytopenia, with a haemoglobin level of 5.0 g/dL, elevated mean corpuscular volume (142.2 fL), platelet count of 26 × 10^9/L, white cell count of 0.9 × 10^9/L, and significantly elevated troponin I levels (initially 2069 ng/L and rising to over 25,000 ng/L). Iron studies showed low serum iron (9.3 µmol/L), marginally elevated ferritin (325.3 ng/mL) and reduced total iron-binding capacity (40.84 µmol/L). Vitamin assays confirmed severe vitamin B12 deficiency (59 pmol/L) with elevated folate (49.2 nmol/L). Given the profound B12 deficiency, immunological testing revealed the presence of anti-parietal cell antibodies and elevated intrinsic factor IgG, which confirmed the diagnosis of pernicious anaemia. Given the clinical features suggestive of hypothyroidism, thyroid function testing was performed, revealing a free T4 of 4.3 pmol/L, TSH of 108.96 mIU/L and anti-thyroid peroxidase antibodies >600 IU/mL, consistent with Hashimoto's thyroiditis. Levothyroxine and cyanocobalamin replacement therapy were initiated subsequently. These findings led to a diagnosis of APS type 3b, characterised by the coexistence of pernicious anaemia and Hashimoto's thyroiditis.

CONCLUSION

Hashimoto's thyroiditis (HT) and autoimmune gastritis (AIG) often coexist. Studies have shown that HT is present in 10–40% of patients with gastric disorders, and about 40% of those with AIG also have HT. This case emphasizes the need to screen for coexisting autoimmune conditions.

EP_A062

OVARIAN OVERDRIVE: FUNCTIONING GONADOTROPH ADENOMA LEADING TO SPONTANEOUS OVARIAN HYPERSTIMULATION

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INTRODUCTION/BACKGROUND

Functioning gonadotroph adenomas (FGAs) are rare pituitary tumours characterised by the hypersecretion of biologically active gonadotrophs. We report a case of a 22-year-old Malay female diagnosed with FGA with ovarian hyperstimulation syndrome (OHSS), highlighting her clinical presentation, management, and post-operative outcomes.

CASE

The patient first presented at age 19 with acute abdominal pain and irregular menstruation. An abdominal ultrasound