EP_A081

ADRENAL SUPPRESSION DURING BIOLOGIC-FACILITATED STEROID TAPERING IN SEVERE ASTHMA: A REAL-WORLD TERTIARY-CENTRE EXPERIENCE

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INTRODUCTION
A significant proportion of severe asthma patients require oral corticosteroids (OCS) to maintain symptom control. Prolonged OCS predisposes patients to hypothalamic-pituitary-adrenal axis suppression which can be life-threatening. Biologic therapies are effective steroid-sparing options suitable for select severe asthma patients. We evaluated steroid tapering outcomes and adrenal function during biologic therapy in a tertiary severe asthma clinic.

METHODOLOGY
We retrospectively analyzed our severe asthma clinic cohort. Patients were included if they attended the clinic in 2025, had severe asthma requiring ≥5 mg maintenance prednisolone daily (or equivalent), initiated biologic therapy, and underwent adrenal function assessment with morning serum cortisol and/or Short Synacthen Test (SST) during steroid tapering between 2024 and 2025. Patients who were still weaning maintenance steroids were excluded.

RESULTS
A total of 43 patients were included. Median age was 66 years (interquartile range [IQR] 60–74), and 65% were male. Mepolizumab was the most used (17 patients), followed by benralizumab (15), tezepelumab (6), dupilumab (4), and omalizumab (1). Median baseline prednisolone dose was 10 mg (IQR 5–10). Median lowest dose achieved was 3 mg (IQR 0–3), sustained for at least 4 weeks in 90.7% of patients. Median reduction in prednisolone dose was 5 mg (IQR 5–7.25). In all, 42% of patients successfully discontinued maintenance steroids. Adrenal function was assessed using morning serum cortisol (n = 37) and/or SST (n = 28). Evidence of adrenal insufficiency was identified in 19 patients (44%), including 15 patients with failed SST and four with low morning cortisol suggestive of adrenal suppression. Morning serum cortisol was indeterminate in four patients. A total of 17 patients were referred to endocrinology. During steroid tapering, 11 asthma-related hospital admissions and eight emergency department presentations occurred, involving 10 patients.

CONCLUSION
Biologics facilitate the reduction of the OCS burden in severe asthma. Tertiary adrenal suppression is common during steroid tapering and requires prompt biochemical assessment and endocrinology referral. Structured monitoring of adrenal function is essential during biologic-facilitated steroid tapering in routine clinical practice.

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METASTATIC EXTRA-OVARIAN STEROID CELL TUMOR PRESENTING WITH HYPERANDROGENISM AND TRANSAMINITIS POST OOPHORECTOMY

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INTRODUCTION
Steroid cell tumors are rare sex cord-stromal tumors, accounting for <0.1% of ovarian neoplasms. Extraovarian steroid cell tumors are exceptionally rare, often androgen-secreting, and pose significant diagnostic challenges. Early recognition is essential to prevent prolonged morbidity from hyperandrogenism.

CASE
A 60-year-old female, 15 years after total hysterectomy and bilateral salpingo-oophorectomy for a large ovarian mass with massive ascites, presented with deranged liver function tests on routine follow-up. Ultrasonography and computed tomography imaging revealed multiple hypervascular lesions in the liver, retroperitoneum, and peritoneum, suggestive of metastatic disease, with normal-appearing adrenal glands. Biopsy of a liver lesion demonstrated a metastatic neoplasm with morphology and immunoprofile favoring a steroid cell tumor. However, metastasis from the adrenal cortex or an ovarian primary could not be excluded.

Given the prior bilateral oophorectomy, metastatic adreno-cortical carcinoma was initially suspected, prompting endocrine evaluation. Further history revealed a 1-year history of progressive virilization, including increased facial hair and frontal balding. Hormonal studies demonstrated elevated testosterone (12.2 mmol/L and reference range 0.1–1.42) and dehydroepiandrosterone sulfate (15.9 µmol/L and reference range 0.510–5.560) and the adrenocorticotropic hormone level of 11.7 pmol/L (reference range 1.60–13.9) with suppressed gonadotrophins. Additional workup for catecholamine, cortisol, and aldosterone excess was unremarkable. The

discordance between androgen excess and normal adrenal imaging, despite absent ovarian tissue, suggested an extra-adrenal androgen-secreting steroid cell tumor. A second histopathology review and multidisciplinary discussion with radiology, gynecologic oncology, and pathology teams were undertaken. As the disease was deemed inoperable, repeat retroperitoneal lesion biopsy confirmed metastatic steroid cell tumor and guided palliative chemotherapy. She was subsequently referred to gynecologic oncology for systemic chemotherapy.

CONCLUSION

Extra-adrenal steroid cell tumors, though rare, should be considered in patients with hyperandrogenism long after bilateral oophorectomy, especially when adrenal imaging is normal. Multidisciplinary evaluation and repeat biopsy are often crucial for establishing the diagnosis and guiding treatment.

EP_A083

SEVERE HYPERTRIGLYCERIDEMIA-INDUCED ACUTE PANCREATITIS IN PREGNANCY: A CASE REPORT AND REVIEW OF MANAGEMENT

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INTRODUCTION

Severe hypertriglyceridemia during pregnancy is a rare yet potentially life-threatening disorder. It is frequently linked to acute pancreatitis and poses considerable risks for both maternal and fetal morbidity.

CASE

We report a 35-year-old G4P3 female at 33 + 6 weeks gestation with underlying type 2 diabetes mellitus who presented with acute abdominal pain, nausea, vomiting, and poor oral intake, initially mistaken for preterm labor and sepsis. Further evaluation revealed profound hypertriglyceridemia (triglycerides 79.1 mmol/L) with markedly elevated total cholesterol (>32 mmol/L) and raised serum amylase, consistent with hypertriglyceridemia-induced acute pancreatitis. She was managed in a multidisciplinary setting with intravenous insulin infusion, dextrose supplementation, aggressive fluid resuscitation, and empiric antibiotics. Due to clinical deterioration and ongoing risk of maternal complications, she underwent emergency lower segment caesarean section and exploratory laparotomy at 34 weeks of gestation. Intraoperative findings included inflammatory intra-abdominal fluid and a bulky pancreas, supporting the diagnosis. Rapid biochemical improvement was observed following treatment, with triglyceride levels decreasing by approximately 70% within 24 hours (79.1–24.0 mmol/L) and achieving >90% reduction over 10 days. Postpartum management included initiation of omega-3 fatty acids, fibrate, and statin therapy, resulting in sustained lipid control. Ophthalmologic examination demonstrated lipemia retinalis, further confirming severe dyslipidemia.

CONCLUSION

This case highlights the importance of early recognition of severe hypertriglyceridemia in pregnancy, particularly in patients with metabolic risk factors such as diabetes. Prompt initiation of insulin therapy can achieve rapid triglyceride reduction and may obviate the need for plasmapheresis in selected cases. Delivery should be considered in the setting of maternal instability. Long-term lipid management postpartum is essential for preventing recurrence and reducing future metabolic risk. A multidisciplinary approach remains critical in optimizing both maternal and fetal outcomes in this rare but serious condition.

EP_A084

SEVERE PREMATURE CORONARY ARTERY DISEASE IN HOMOZYGOUS FAMILIAL HYPERCHOLESTEROLEMIA WITH MARKED LIPID REDUCTION AFTER INCLISIRAN THERAPY

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INTRODUCTION

Homozygous familial hypercholesterolemia (HoFH) is a rare inherited disorder characterized by markedly elevated low-density lipoprotein cholesterol (LDL-C) from birth, childhood xanthomas, and accelerated atherosclerotic cardiovascular disease. Achieving LDL-C targets remains difficult despite statins, ezetimibe, and lipoprotein apheresis, especially in patients with treatment interruption, poor adherence, or limited access to specialized lipid services. We report a female with genetically confirmed HoFH who developed severe premature coronary artery disease and showed marked lipid reduction after inclisiran therapy.

CASE

A 28-year-old Malay female with LDL receptor mutation-confirmed HoFH, diagnosed at age 7, had a strong family history of premature cardiovascular death. She underwent biweekly lipoprotein apheresis from ages 8 to 15 years, but later defaulted on follow-up. During pregnancy in 2021, weekly then biweekly apheresis was reintroduced for severe hypercholesterolemia, but she disengaged postpartum. In 2025, she re-presented with exertional