Adult E-Poster

EP_A023

SPONTANEOUS REMISSION OF GRAVES' DISEASE FOLLOWING SYSTEMIC LUPUS ERYTHEMATOSUS TREATMENT: A CASE REPORT

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INTRODUCTION/BACKGROUND
Hyperthyroidism secondary to Graves' disease is typically managed with thionamides, radioiodine therapy, or thyroidectomy. However, spontaneous remission is uncommon, especially after prolonged thionamide therapy. This case highlights a rare instance of hyperthyroidism remission one year after treatment with steroids for systemic lupus erythematosus, despite seven years of prior thionamide use.

CASE
A 25-year-old Malay female with type 1 diabetes mellitus (T1DM) and SLE was diagnosed with Graves' disease at age 15 and treated with carbimazole for seven years. Hyperthyroidism resolved three years before her SLE diagnosis. In 2021, she was diagnosed with Class IV/V lupus nephritis and started on high-dose corticosteroids (methylprednisolone and prednisolone) with cyclophosphamide. One year after initiating steroid therapy, thyroid function tests (TFTs) remained euthyroid without antithyroid medication. Repeat TFTs confirmed continued remission.

Several mechanisms may explain the remission of Graves' disease in this case. High-dose corticosteroids suppress autoreactive B and T lymphocytes, potentially reducing thyrotropin receptor antibody (TRAb) production and facilitating remission. Additionally, corticosteroids enhance regulatory T-cell (Treg) activity, restoring immune tolerance and reducing autoimmunity. The presence of multiple autoimmune diseases suggests a broader dysregulation of immune function, thus immunosuppressive therapy for SLE may have inadvertently suppressed the pathogenic mechanisms driving Graves' disease. Lastly, long-standing autoimmunity can lead to immune exhaustion, where autoreactive immune cells become less active over time, potentially contributing to spontaneous remission.

CONCLUSION
Although corticosteroids are not a conventional treatment for hyperthyroidism, their immunomodulatory effects may inadvertently promote disease remission in select cases. This highlights the need for further research to elucidate the potential role of immunosuppressive therapy in achieving sustained remission of autoimmune hyperthyroidism.

EP_A024

AUTOIMMUNE POLYGLANDULAR SYNDROME TYPE IIIA WITH LUPUS NEPHRITIS: A CASE REPORT

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INTRODUCTION/BACKGROUND
Autoimmune polyglandular syndrome (APS) is a rare disorder characterized by multiple autoimmune endocrinopathies. The condition is driven by T-lymphocyte-mediated and autoantibody-induced destruction of various organs. APS Type III is defined by the presence of autoimmune thyroid disease in association with other autoimmune conditions, excluding adrenal insufficiency. APS Type IIIa specifically involves autoimmune thyroid disease and type 1 diabetes mellitus (T1DM). Early recognition and multidisciplinary management are crucial for optimal outcomes.

CASE
A 25-year-old Malay female with T1DM since age seven, inactive Graves' disease, and systemic lupus erythematosus (SLE) with lupus nephritis presented with loose stools (Bristol 7), vomiting, heartburn, bloating, reduced oral intake, and oliguria of eight days duration. Though she was ambulatory, she had a 2-day history of generalized muscle weakness. There was no fever or other indicators of infection. No dietary indiscretion was noted.

Type 1 diabetes mellitus was well-controlled on an insulin regimen. She had hypertension and dyslipidemia since age ten. Graves' disease resolved three years ago after carbimazole treatment. Systemic lupus erythematosus was complicated by class IV/V lupus nephritis, initially treated with corticosteroids and cyclophosphamide, then eventually shifted to mycophenolate mofetil and bisphosphonates as maintenance therapy. Renal function corresponds to CKD stage 3 (eGFR 36 mL/min/1.73 m^2).

Examination revealed central obesity, bilateral pitting edema, and striae, without overt dehydration or hyperglycemia. No anemia, acute infection, cardiac failure, or thyroid dysfunction was noted. On the basis of the presence of T1DM, autoimmune thyroid disease, and SLE, she meets APS type IIIa criteria.

CONCLUSION
This case highlights the need for heightened awareness of autoimmune polyglandular syndrome (APS), particularly APS type IIIA in patients presenting with multiple autoimmune endocrinopathies. Clinicians should maintain

a high index of suspicion for APS in individuals with a history of multiple autoimmune disorders, allowing for early screening and intervention to prevent complications. Comprehensive autoimmune surveillance and inter-disciplinary collaboration are essential for optimizing patient outcomes.

EP_A025

BILATERAL ADRENAL HISTOPLASMOSIS IN AN IMMUNOCOMPETENT ELDERLY PATIENT: A CASE REPORT

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INTRODUCTION

Histoplasmosis is a fungal infection caused by *Histoplasma capsulatum*, often acquired through inhalation of spores. Disseminated histoplasmosis with adrenal gland involvement is rare and may lead to adrenal insufficiency.

CASE

We report a case of a 70-year-old male, previously healthy, who presented with constitutional symptoms i.e. generalized lethargy, reduced appetite, and significant weight loss of 10 kilograms within 3 months. He was normotensive and no hyperpigmentation was noted. Tumor markers and viral screening were negative and HbA1c was 6.8%. There was no hyponatremia or hyperkalemia. Morning cortisol was 341 nmol/l. Computed tomography scan of the thorax, abdomen, and pelvis revealed bilateral mixed solid cystic adrenal masses and a wedge-shaped hypodense area in the spleen. Adrenal protocol of the CT showed bilateral adrenal masses measuring 5.3 x 3.7 x 3.8 cm on the right and 3.4 x 2.8 x 2.6 cm on the left. A CT-guided adrenal biopsy was performed and histopathological examination revealed highly fragmented tissue strips with huge areas of necrosis and hemorrhage composed of vague formation of epithelioid granuloma with numerous fungal spores. The fungi appearing intra-cytoplasmic in the H&E staining may suggest a histoplasmosis etiology. Synacthen test confirmed adrenal insufficiency with a peak cortisol level of 453 nmol/l. Intravenous amphotericin-B was given for two weeks followed by oral itraconazole 200 mg BD, alongside corticosteroid replacement for adrenal insufficiency. After 6 months, his condition improved significantly, and his adrenal size decreased on follow-up imaging with the largest diameter measuring 3.8 cm. We plan to complete itraconazole for a total of 12 months duration.

CONCLUSION

Bilateral adrenal histoplasmosis usually affects immunocompromised patients or those from endemic areas. This diagnosis should be taken into consideration in a patient presenting with bilateral adrenal masses. Diagnosis is often delayed due to nonspecific symptoms. Imaging, serology, and biopsy are essential for diagnosis. Prompt diagnosis and commencement of antifungal treatment are imperative to prevent adrenal crises.

EP_A026

SECRETIVE SECRETIONS, EXPLOSIVE EXCRETIONS: A RARE CASE OF VIPoma

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INTRODUCTION/BACKGROUND

Vasoactive Intestinal Peptide (VIP)-secreting neuroendocrine tumours (VIPomas) are rare, usually presenting with profuse watery diarrhea leading to severe electrolyte imbalances. Our patient's initial presentation with obstructive jaundice and portal vein thrombosis preceded the typical presentation, complicating early diagnosis.

CASE

A 34-year-old male presented in May 2024 with progressive jaundice, tea-colored urine and pale stools but no diarrhea. Investigations revealed markedly elevated direct hyperbilirubinemia (205 µmol/L), alkaline phosphatase (742 U/L) and gamma-glutamyl transferase (612 U/L), which was suggestive of biliary obstruction. CT scan identified a 4.2 cm pancreatic mass with hepatic metastases and portal vein thrombosis. EUS visualized a hypoechoic lesion at the head of the pancreas measuring 38 x 35 mm, causing upstream dilatation of the pancreatic and common bile duct. ERCP was done for sphincterectomy and stenting. Histopathology revealed a low-grade neuroendocrine neoplasm.

Six months later, the patient developed persistent watery diarrhea for two weeks, along with fatigue and weight loss. His severe hypokalemia (as low as 1.5 mmol/L) was