fine-needle aspiration, the constellation of a palpable goiter, characteristic ultrasonographic findings, negative autoimmunity, and active multisystem disease strongly supported a diagnosis of direct histiocytic infiltration of the thyroid gland. She was initiated on appropriate glucocorticoid coverage and subsequent levothyroxine replacement, alongside systemic intravenous cytarabine.

CONCLUSION

This case highlights a rare, mixed endocrine profile in multisystem LCH, demonstrating that pituitary and direct end-organ infiltration can coexist. Hypothyroidism in LCH patients with established central diabetes insipidus should not be reflexively assumed to be central in origin. A comprehensive diagnostic workup, including autoantibody screening, ultrasound, and ideally histopathological confirmation, is essential to accurately identify primary endocrine failure and guide appropriate clinical management in these complex cases.

EP_A150

IMMUNE-MEDIATED PANCYTOPENIA ASSOCIATED WITH GRAVES' DISEASE MIMICKING EVANS SYNDROME AND CARBIMAZOLE-INDUCED AGRANULOCYTOSIS

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INTRODUCTION

Autoimmune thyroid disease is frequently associated with other immune-mediated disorders; however, clinically significant pancytopenia is rare. In patients with Graves' disease receiving antithyroid therapy, leukopenia raises concern for drug-induced agranulocytosis, a rare but potentially life-threatening complication characterized by severe neutropenia requiring immediate drug withdrawal. The coexistence of hemolytic anemia and thrombocytopenia may instead suggest Evans syndrome, defined by autoimmune hemolytic anemia with immune thrombocytopenia, with or without neutropenia. Importantly, uncontrolled thyrotoxicosis itself may cause immune-mediated cytopenias, creating a diagnostic challenge.

CASE

We report a 55-year-old female with thyroid receptor antibody-positive Graves' disease who presented with jaundice and pancytopenia while receiving carbimazole therapy. Laboratory evaluation demonstrated anemia with reticulocytosis and a positive direct antiglobulin test, thrombocytopenia and leukopenia. Complement testing revealed reduced C3 with normal C4, consistent with immune-mediated hemolysis. Peripheral blood film showed no blast cells or marrow infiltration, and autoimmune screening, including antinuclear antibodies and anti–double stranded DNA, was negative.

The coexistence of Coombs-positive hemolysis and thrombocytopenia initially raised suspicion for Evans syndrome, while leukopenia during carbimazole therapy prompted concern for drug-induced agranulocytosis. However, neutropenia was not severe, and the absence of marrow infiltration or systemic autoimmune disease made alternative causes of pancytopenia less likely. Importantly, blood counts progressively improved following the optimization of thyroid control despite continuation of carbimazole at a reduced dose, without the use of immunosuppressive therapy. This clinical course supported the interpretation of thyrotoxicosis-associated immune cytopenia rather than primary Evans syndrome or carbimazole-induced agranulocytosis.

CONCLUSION

This case highlights thyrotoxicosis-associated immune cytopenia as an important mimic of Evans syndrome and carbimazole-related hematological toxicity. Recognizing this entity is essential to avoid unnecessary discontinuation of antithyroid therapy or inappropriate immunosuppressive treatment.

EP_A151

COLD SPOT WITHIN A HOT NODULE: THYROID STORM FROM TOXIC ADENOMA REVEALING RARE HURTHLE CELL ADENOMA

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INTRODUCTION

Hurthle cell adenoma is a rare benign thyroid neoplasm that can only be diagnosed through histopathological examination. Hurthle cell neoplasm typically presents as nonfunctioning cold nodule on thyroid scintigraphy. We report a rare case of Hurthle cell adenoma presenting with thyroid storm, with unusual findings of "cold" within "hot" thyroid nodule on scintigraphy.

CASE

A 73-year-old male with hypertension, chronic kidney disease, coronary artery disease, and Parkinson's disease presented to the emergency department with fever and diarrhea. His temperature was 38.4°C, heart rate 106 bpm, and blood pressure 138/75 mmHg, with atrial fibrillation and signs of heart failure. The Burch-Wartofsky score was 50, consistent with thyroid storm.

Laboratory tests revealed free thyroxine 4 37.8 pmol/L (NR 11.5–22.7), free thyroxine 3 5.6 pmol/L (NR 3.5–6.5), and thyroid-stimulating hormone <0.01 mIU/L (NR 0.55–4.78). Thyroid autoantibodies, including anti-thyroid peroxidase, anti-thyroglobulin, and thyroid-stimulating immunoglobulins, were negative (<0.10 IU/L). The thyroid storm was precipitated by invasive *Klebsiella* syndrome with endophthalmitis and lung and liver abscess. He was treated with Lugol's iodine, corticosteroid, antibiotics, and carbimazole.

Ultrasound thyroid revealed a mixed cystic-solid nodule in the left thyroid lobe, measuring 2.3 × 3.3 × 4.3 cm (TI-RADS category 3). Technetium-99m thyroid scintigraphy demonstrated a hyperfunctioning left thyroid nodule with a focal intranodular cold spot measuring 5.0 × 3.7 cm. Fine needle aspiration cytology of the nodule was benign follicular cells. Following stabilization with anti-thyroid treatment, he underwent left hemithyroidectomy. Histopathology examination revealed a Hurthle cell adenoma without capsular or vascular invasion.

Postoperatively, he remained clinically euthyroid. Surveillance ultrasound performed 8 months later showed a normal right thyroid lobe, and lifelong surveillance was planned.

CONCLUSION

This case illustrates a rare and unusual presentation of thyroid storm caused by a toxic Hurthle cell adenoma containing an intranodular cold spot on scintigraphy. To our knowledge, only one similar case has been reported in the literature, and our case is the first to present with thyroid storm.

EP_A152

ACUTE THYROID PAIN IN PREGNANCY: PAINFUL HASHIMOTO'S THYROIDITIS VERSUS SUBACUTE THYROIDITIS— A CASE REPORT

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INTRODUCTION

Painful Hashimoto's thyroiditis is a rare and atypical form of autoimmune thyroid disease characterized by thyroid pain and tenderness. It can closely resemble subacute thyroiditis and must be distinguished from other causes of thyroid pain, including hemorrhage into a cyst and thyroid abscess. This distinction is particularly important in pregnancy, where radionuclide imaging is not recommended.

CASE

A 27-year-old pregnant female (G4P3) at 24 weeks' gestation, with Southeast Asian ovalocytosis and diet-controlled gestational diabetes, was diagnosed with primary hypothyroidism at 14 weeks following evaluation of a painless goiter. Initial tests showed markedly elevated thyroid-stimulating hormone (>150 mIU/L), low free thyroxine (3.5 pmol/L), and positive anti-thyroid peroxidase antibodies (319 IU/mL), consistent with Hashimoto's thyroiditis. Levothyroxine therapy achieved biochemical euthyroidism.

At 24 weeks, she developed acute right-sided anterior neck pain radiating to the ear, with dysphagia, odynophagia, and fever. Examination revealed a tender thyroid without lymphadenopathy. Ultrasound demonstrated diffuse enlargement with bilateral ill-defined hypoechoic avascular areas. Inflammatory markers showed markedly elevated C-reactive protein (184 mg/L) with a normal erythrocyte sedimentation rate (16 mm/h), while thyroid function remained within target range. Imaging and clinical findings made abscess and hemorrhage unlikely.

The presence of pre-existing autoimmune thyroid disease, antibody positivity, and hypothyroidism before symptom onset supported painful Hashimoto's thyroiditis over subacute thyroiditis. This case highlights that normal thyroid function tests do not necessarily indicate disease remission, as inflammatory activity may persist independently of hormone levels.