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.