Beyond the thyroid: Myxedema coma unmasking pulmonary arterial hypertension

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Case presentation:
A 55-year-old male with history of Hashimoto's disease and pre-diabetes presented with lower extremity swelling and pain for 3 days. The patient presented with concomitant lower extremity cellulitis and signs of CHF, with BNP 5600 at arrival. He was severely hypothyroid with TSH 14.7 and T4 undetectable and had progressively worsening mentation, suggesting myxedema coma. The patient was treated with levothyroxine and stress dose steroids. Bedside ultrasound demonstrated sings of fluid overload including appreciable B lines. TTE showed EF >55% , severely dilated IVC, and RV dysfunction. Patient underwent RHC demonstrating severely elevated pulmonary arterial pressure (mean 57 mmHg), normal PCWP, and elevated PVR. Patient was diagnosed with pulmonary arterial hypertension and treatment with sildenafil and ambrisentan was initiated in conjunction with aggressive diuresis. During hospitalization, the patient's thyroid function improved, and he was discharged on levothyroxine.

Discussion:
Pulmonary arterial hypertension (PAH) is a progressive, severe disease defined by elevated pulmonary artery pressures, normal left heart filling pressures, and elevated pulmonary vascular resistance. Several studies have demonstrated a higher prevalence of thyroid disease in patients with PAH than in the general population. In 2025, an *IJC Cardiology Heart & Vasculature* study addressed pulmonary hypertension in hypothyroidism, concluding that hypothyroidism is causally associated with PAH. One prospective cohort found a 49% prevalence of autoimmune thyroid disease in PAH, suggesting overlapping immunologic etiologies. New research demonstrates that preexisting hypothyroidism exacerbates pulmonary hypertension (PH) by precipitating inflammation and oxidative stress pathway activation in animal models. Comorbid thyroid disease may be a prognostic factor in PAH, suggesting that treatment of underlying thyroid disease may play a role in approaching PAH.

This case highlights the multifactorial etiology of PAH and demonstrates that hypothyroidism may exacerbate or unmask cardiopulmonary conditions. It underscores the possibility of thyroid disease as a contributing and treatable component of PAH. This complex presentation of PH in the setting of cardiac dysfunction, hypothyroidism, hypervolemia, and OSA also demonstrates the diagnostic challenge of categorizing pulmonary hypertension and directing treatment.

Conclusion:
The interplay between hypothyroidism and pulmonary arterial hypertension has not been fully elucidated. As recently as 2025, the causal association between thyroid dysfunction and PAH is unknown. This case suggests that exacerbation of thyroid disease, in this case, myxedema coma, may unmask or worsen pulmonary arterial hypertension. Clinicians should consider screening for thyroid abnormalities in patients with PAH and consider hypothyroidism a factor in PAH disease progression. In addition, this case underscores the importance of clearly categorizing PH to ensure guideline-directed treatment decisions.

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