At 24 + 4 weeks, TFTs showed persistently low FT4 (5.84 pmol/L) and TSH (0.022 mIU/L). Differential diagnoses included central hypothyroidism or assay interference. Tests were repeated using different platforms. Both the Beckman system (FT4: 5.73 pmol/L, TSH: 0.086 mIU/L) and Roche system (FT4: 8.83 pmol/L [12.0–22.0], TSH: 0.11 mIU/L [0.27–4.20]) confirmed low values, ruling out assay interference. Following endocrinology consultation and given her asymptomatic status, IH was considered most likely, and a plan for close monitoring was initiated.

Serial follow-up demonstrated biochemical evolution. By 31 + 6 weeks, TFTs showed a low TSH (0.753 mIU/L) with an FT4 level (5.86 pmol/L) within the normal reference range, a pattern typical of IH in pregnancy. Repeat TFTs 6 weeks postpartum showed both TSH and FT4 within normal range.

CONCLUSION

This case illustrates a rare and unexpected biochemical progression of IH in pregnancy, initially presenting with a pattern indistinguishable from central hypothyroidism. It underscores that in an asymptomatic, fertile patient, IH can manifest with an atypical pattern early in gestation. The findings suggest that vigilant serial monitoring, rather than immediate extensive pituitary workup, may be a prudent initial approach in such scenarios, as the biochemical profile can normalize toward the classic IH pattern as pregnancy advances.

EP_A173

WHEN TSH SUPPRESSION BECOMES HARMFUL: THYROXINE OVER-REPLACEMENT DRIVING CARDIOVASCULAR DECOMPENSATION IN ADVANCED HEART FAILURE

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INTRODUCTION

Thyroid stimulating hormone (TSH) suppression following differentiated thyroid carcinoma is widely recommended to reduce recurrence risk. However, this strategy assumes cardiovascular tolerance to supraphysiologic thyroid hormone exposure. In patients with advanced structural heart disease, this assumption may fail, exposing a critical limitation of guideline-directed TSH suppression.

CASE

We report a 71-year-old male with end-stage renal failure on hemodialysis and severe ischemic cardiomyopathy (ejection fraction 23%) who presented with acute decompensation characterized by dyspnea, rapid atrial fibrillation, and non–ST-elevation myocardial infarction. He had a history of papillary thyroid carcinoma treated with total thyroidectomy and radioactive iodine over 20 years prior and was maintained on levothyroxine 200 mcg daily for TSH suppression. Despite biochemically euthyroid indices (TSH 1.8 mIU/L, free thyroxine 4 17 pmol/L), he developed recurrent arrhythmia with heart failure decompensation.

This case highlights a dissociation between biochemical euthyroidism and tissue-level thyrotoxicity in a structurally compromised myocardium. Papillary thyroid carcinoma after definitive therapy typically follows an indolent course with low short-term mortality. In contrast, in severe left ventricular dysfunction, excess thyroid hormone increases adrenergic sensitivity and myocardial oxygen demand, precipitating arrhythmia and ischemia. This risk is amplified in end-stage renal disease, where altered hormone handling renders biochemical indices less reliable.

CONCLUSION

Biochemical euthyroidism does not equate to physiological safety. In patients with advanced cardiovascular disease, thyroid hormone therapy should be titrated to cardiovascular tolerance rather than oncologic targets alone, and routine TSH suppression may be inappropriate.

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SEVERE BIOCHEMICAL THYROTOXICOSIS WITHOUT CLINICAL HYPERTHYROIDISM IN ESRF FOLLOWING PARATHYROIDECTOMY: A DIAGNOSTIC AND THERAPEUTIC PITFALL

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INTRODUCTION

Thyrotoxicosis following neck surgery is typically attributed to transient destructive thyroiditis from follicular disruption. In end-stage renal failure (ESRF), however, altered thyroid hormone kinetics, including reduced protein binding, impaired peripheral metabolism, and decreased clearance, can distort biochemical interpretation.