Letter to the Editor

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From a favorable laboratory profile to RAI-related outcomes: extending the evidence for LT3-assisted withdrawal

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Dear Editor,

We read with great interest the recent article by Tüfekçi et al.[1] entitled "LT3-treatment protocol of short-term hypothyroidism induction in patients with differentiated thyroid carcinoma; a safer strategy with lower risk of laboratory abnormalities compared to 'no treatment' protocol". The authors should be congratulated for addressing an important clinical issue regarding preparation for radioactive iodine (RAI) therapy in patients with differentiated thyroid carcinoma (DTC). Their findings suggest that temporary LT3 administration before withdrawal may attenuate several laboratory abnormalities associated with hypothyroidism while preserving adequate TSH stimulation.

However, we believe that the clinical implications of these findings would be strengthened by evaluating RAI-related outcomes rather than laboratory parameters alone. Current international guidelines emphasize that the success of RAI preparation should ultimately be assessed using clinically meaningful endpoints, including post-therapy whole-body scintigraphy, stimulated thyroglobulin (Tg), anti-thyroglobulin antibody dynamics, ablation success, and dynamic response-to-therapy assessment, rather than biochemical alterations during thyroid hormone withdrawal alone.[2-5]

Although the laboratory profile observed in the LT3-treated group appears favorable, it remains unclear whether these biochemical improvements translate into improved RAI efficacy or long-term oncological outcomes. A protocol that minimizes short-term metabolic disturbances would have greater clinical relevance if it also resulted in comparable or superior remnant ablation rates, lower stimulated Tg levels, improved response-to-therapy classification, or reduced disease persistence during follow-up.[2-8]

Future prospective studies incorporating these clinically relevant endpoints may therefore better define the true clinical value of LT3-assisted withdrawal. Such investigations would determine whether the observed laboratory advantages are accompanied by equivalent oncological effectiveness and could facilitate broader implementation of this preparation strategy in selected patients with differentiated thyroid carcinoma.[3-8]

Overall, we congratulate the authors for their valuable contribution and believe that extending future investigations toward clinically meaningful RAI outcomes will further clarify the role of LT3-assisted withdrawal in contemporary differentiated thyroid cancer management.

ETHICAL DECLARATIONS

Peer Review Process

This letter was externally peer-reviewed.

Conflict of Interest

The author declare no conflicts of interest.

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Author Contributions

The author is solely responsible for the entirety of conception, execution, analysis, and writing of the manuscript.

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Letter to the Editor

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Author reply "'LT3-treatment' protocol of short-term hypothyroidism induction in patients with differentiated thyroid carcinoma: a safer strategy with lower risk of laboratory abnormalities compared to 'no treatment' protocol"

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Dear Editor,

We sincerely thank the authors for their thoughtful comments regarding our study, "LT3-treatment" protocol of short-term hypothyroidism induction in patients with differentiated thyroid carcinoma: a safer strategy with lower risk of laboratory abnormalities compared to "no treatment" protocol. We appreciate their important emphasis on the distinction between achieving adequate TSH stimulation and demonstrating the therapeutic effectiveness of radioactive iodine (RAI) therapy.

Our study was primarily designed to evaluate the short-term biochemical profile and tolerability of LT3-assisted withdrawal compared with the "no treatment" protocol during preparation for RAI therapy. We found that LT3-assisted withdrawal was associated with a more favorable laboratory profile while maintaining adequate TSH stimulation. We agree, however, that adequate TSH elevation should be considered a surrogate marker of appropriate preparation and does not, by itself, establish the efficacy of subsequent RAI therapy.

As the authors correctly point out, clinically meaningful RAI-related outcomes, including post-therapy imaging findings, longitudinal thyroglobulin and anti-thyroglobulin antibody levels, ablation success, and subsequent biochemical or structural response, would provide more direct evidence regarding the oncological implications of LT3-assisted withdrawal. These outcomes were not collected as part of our study and therefore cannot be addressed directly within the present analysis.

Accordingly, our findings should not be interpreted as demonstrating oncological equivalence or superiority of LT3-assisted withdrawal with respect to RAI outcomes. Rather, they provide evidence supporting the short-term biochemical feasibility and improved laboratory tolerability of this approach. Whether these advantages can be achieved without compromising the effectiveness of RAI therapy remains an important question.

We agree that future prospective comparative studies should incorporate standardized RAI-related and oncological endpoints alongside measures of biochemical tolerability. Such studies would help determine whether the more favorable laboratory profile associated with LT3-assisted withdrawal translates into comparable ablation outcomes and long-term biochemical and structural responses.

We thank the authors for highlighting this important aspect of LT3-assisted withdrawal. Their comments appropriately broaden the clinical perspective of our findings and identify an important direction for future research.

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