Dear editor

I am writing in response to the recently published study on the dynamic changes in abdominal fat area parameters, laboratory parameters, and anthropometric parameters in patients with hyperthyroidism during 12 months of drug therapy.1 The study provides valuable insights into the effects of hyperthyroidism treatment on various biochemical markers and abdominal fat distribution. However, I would like to address some critical concerns regarding the interpretation and implications of the study’s findings.

The study observed an increase in visceral fat area (VFA) and subcutaneous fat area (SFA) after treatment, with thyroid hormones (THs) returning to normal levels and blood lipid levels also increasing but remaining within the normal reference range. The authors suggest that these changes may contribute to the increased risk of metabolic disorders and cardiovascular disease (CVD) in patients with hyperthyroidism even after treatment. While the results are noteworthy, drawing a direct causal relationship between the treatment-induced changes in fat distribution, lipid profile, and increased CVD risk requires cautious interpretation.

Firstly, the study design—a single-center, single-group repeated measures approach limits the ability to establish causality. Without a control group or a randomized controlled trial design, it is difficult to ascertain whether the observed changes are a direct consequence of the treatment or other confounding factors. The authors themselves acknowledge this limitation, suggesting the need for multicenter studies and longer-term follow-ups.

Secondly, the follow-up period of 12 months, while sufficient to observe some changes, may not capture the long-term effects of hyperthyroidism treatment on abdominal fat and lipid profiles. Longer follow-up periods could provide more comprehensive insights into whether these changes persist, diminish, or worsen over time, thereby offering a clearer picture of the long-term health implications for patients.

Additionally, it’s worth noting that in the studies by Franklyn et al and Hall et al, which were cited in this study and showed increased mortality risk in treated hyperthyroid patients, the individuals had undergone radioactive iodine (RAI) therapy. Some of them subsequently developed hypothyroidism and required T4 replacement therapy.2,3 This raises the possibility that the continued elevated mortality risk may be related to RAI itself or the resultant hypothyroidism.2 In fact, in a later prospective study by Franklyn et al, no increase in mortality was observed in patients receiving T4 replacement post-RAI.4 Given the well-established link between hypothyroidism and cardiovascular disease,5,6 it would have been beneficial for the authors to mention the prevalence of subclinical hypothyroidism or hypothyroidism in their patient population post-treatment.

Thank you for considering these points. I hope this contributes to a more nuanced understanding of the study’s implications.
Disclosure
The author reports no conflicts of interest in this communication.

References