The article in this journal entitled ‘Sub-clinical Hypothyroidism: A Determinant of Polycystic Ovary Syndrome’ by Ghosh S et al. [1] is very interesting. The authors’ attempt to explain etiological aspects of polycystic ovary syndrome (PCOS) is much appreciated.

I have a few comments to make. The first concerns how the authors selected patients with subclinical hypothyroidism who did not present any health problems requiring medical attention. Further, the diagnosis of subclinical hypothyroidism is usually based on normal free thyroid hormone levels with elevated thyrotropin in the absence of clinical features [2]. The authors, however, have described low total thyroxine (T4) with marginally elevated thyroid-stimulating hormone (TSH) in patients with subclinical hypothyroidism, which seems to be something of a discrepancy.

The second comment relates to the hypo-thyroid PCOS cases. I would like to ask whether the raised levels of TSH that were found could have been due to crossreactivity of the TSH assay (if not done immunoradiometrically) with raised gonadotropin in PCOS cases. This seems plausible, because I do not see an association between frank or overt hypothyroidism and PCOS in our clinical settings, and it is a known fact that subclinical hypothyroidism may eventually become overt at a reported rate of 5% a year [3].

The authors’ extrapolation of the view of Yen [4] to explain the differential effect of hypothyroidism on gonadotropin release was quite far from thinking from practice. Subclinical hypothyroidism, reported to be a determinant of PCOS, is an early event in the development of overt hypothyroidism but only a few subjects go on to develop PCOS or PCOS-like conditions.

References


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