What is Optimal Treatment of Hypothyroidism? A Matter of Clinical Common Sense

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There is general agreement that autoimmune affection is a major cause of thyroid imbalance. Hypothyroidism is conventionally defined according to biochemical variables. Patients whose values for the first-line tests TSH and free T_4 are considered, respectively, "high" and "low," are judged hypothyroid. A substantial proportion of patients have "high" TSH but "normal" free T_4; these patients are said to have "subclinical hypothyroidism" (notwithstanding clinical symptoms).

Demonstration of thyroid autoantibodies is an important manifestation of thyroid autoimmunity. In cases with a strong suspicion of hypothyroidism, the autoantibodies warrant treatment with thyroid hormone, as discussed previously in this publication.[1]

In the investigation of patients with chronic fatigue, B_{12} deficiency must be ruled out at an early stage. This condition is not infrequently concomitant with autoimmune thyroid disease. If the B_{12} deficiency is not corrected, optimal supplementation of the thyroid condition is difficult to achieve.

Clinicians seeing patients with suspected hypothyroidism, whose first-line tests fall short of attaining levels conventionally considered compatible with hypothyroidism or "subclinical hypothyroidism," are left uneasy. Could hypothyroidism really be confidently excluded on these grounds?

In the diagnostic work-up of the constantly tired patient, there is a step beyond the conventional biochemical/serological thyroid analyses. That step is a direct approach to document autoimmune assault on the thyroid. It is called "fine-needle aspiration (FNA) cytologic examination." The additional diagnostic and therapeutic yield of FNA over conventional first-line tests is considerable.[2][3] We have demonstrated autoimmune lesions of lymphocytic invasion of the gland in clinically hypothyroid patients whose presenting levels of TSH were less than 1 mU/L (and ranging up to 30 mU/L, with a median of 3.8 mU/L). The patients' clinical responses to thyroid medication were equally favourable regardless of their baseline TSH levels.

Now, that clinical hypothyroidism with cytomorphological evidence of autoimmunity has been found in patients with perfectly normal TSH levels, and that supplementation with thyroid hormone is indicated, which management protocol is optimal? Obviously, guidelines based on TSH criteria (restoration of TSH to "normal") are no longer valid. In an excellent survey, Anthony Toft[4] presents a sensible and balanced view on this topic (applicable to patients diagnosed by FNA as well). He states, "It may well not matter whether . . . therapy in patients with primary hypothyroidism results in a serum TSH concentration of less than 0.1, 1.5, or 3.7 mU/L, as long as a sense of well-being is restored."

Why do many patients on thyroid medication achieve wellness only when their TSH is low or suppressed? One explanation is that, in these patients, the TSH signals trigger and maintain autoimmune activity; and, consequently, a low TSH is a prerequisite condition for the patients to have a satisfactory quality of life.

What, then, is the criterion of optimal treatment of patients with hypothyroidism? The answer is not to perfunctorily restore the TSH to "normal." The answer is openness to patients' individual needs to restore thyroid balance—a matter of clinical common sense.

References

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