A 28-year-old nurse comes to the office at the insistence of her husband. Her main complaints consist of fatigue, edginess and trouble sleeping. These symptoms started about two months ago, about a year after the birth of her last child. On further questioning, she has also noticed palpitations, increased frequency of bowel habits, intolerance to the heat and a lighter menses flow. She denies any discomfort in the neck, eye changes, difficulty swallowing and any exposure to iodine containing drugs.

When examined, there is no evidence of Graves’ orbitopathy. The thyroid is about 15 g in size and of normal texture. Her pulse rate is 100 per minute with a BP of 130/68 mmHg. The palms are moist and there is a fine tremor to the fingers of the out-stretched hands. Pelvic examination was unremarkable. See Table 1 for a summary of the patient’s laboratory workup.

What’s your diagnosis?

Table 1

Lab workup

The patient’s lab workup shows the following results:

- Free T4: 28.9 pmol/L (10-22)
- Free T3: 8.2 pmol/L (3.5-6.5)
- Thyroid-stimulating hormone (TSH): 0.01 mIU/L (0.5-5.0)
- Anti-thyroglobulin antibodies: negative
- Anti-thyroperoxidase antibodies: negative
- Radioactive iodine uptake: 1% (6-26)
- Technetium scan: “thyroid not well-visualized because of low radioactive iodine uptake.”
What’s Your Diagnosis?

Answer: Low Radioactive Iodine Hyperthyroidism

The radioactive iodine uptake is one of the more important tests in sorting out the etiology of hyperthyroidism. This patient presents with low radioactive form of hyperthyroidism. Several important forms of thyrotoxicosis can present in this fashion.

Subacute thyroiditis and its variants are common causes of low radioactive iodine uptake forms of hyperthyroidism. This type of hyperthyroidism results from an inflammatory process, possibly triggered by a virus or immune changes, interfering with the uptake of iodine by the thyrocytes.

Classical subacute thyroiditis is unlikely in the absence of pain over the thyroid gland. Postpartum thyroiditis may be considered, but this usually occurs within six months of delivery and, given the timing of the patient’s last pregnancy, this would also be unlikely. Silent subacute thyroiditis is a possibility, however. Struma ovarii is a very rare teratoma of the ovary sometimes containing thyroid tissue in such abundant amounts that hyperthyroidism ensues. The thyroid tissue in the pelvis will produce enough thyroid hormone to turn off the thyroid-stimulating hormone (TSH) and the thyroid gland will lack any stimulation, producing a low radioactive iodine uptake. In this case the absence of any masses noted on the pelvis examination make this diagnosis unlikely.

Another rare cause of low radioactive iodine uptake hyperthyroidism is metastatic follicular cancer of the thyroid. Most follicular thyroid cancers, even when metastases are present, are not associated with hyperthyroidism. In rare cases, however, the metastases may produce sufficient thyroid hormone in sufficient amounts to turn off TSH. Consequently, the thyroid gland will lack any stimulation, producing a low radioactive iodine uptake, analogous to the situation with struma ovarii, mentioned above.
Iodine, especially when given to individuals living in an iodine-deficient area, may induce hyperthyroidism associated with a low radioactive uptake; this is often referred to as iod-Basedow’s disease. Common triggers for iod-Basedow’s disease include the cardiac drug amiodarone and certain radio-contrast dyes that contain abundant amounts of iodine.

Any patient with a high radioactive iodine uptake form of hyperthyroidism (i.e., Graves’ disease or a toxic nodule) would convert to a low radioactive iodine form of hyperthyroidism if given enough exogenous iodine.

Finally, given that the patient is a health professional, factitious hyperthyroidism has to be considered. In this situation, exogenous thyroid hormone will lower TSH resulting in a low radioactive iodine uptake.

The other significant laboratory test was a serum thyroglobulin level. The result was 1.7 pmol/L (2-56: the normal range for serum thyroglobulin). Thyroglobulin is a large molecular weight peptide produced by the thyroid gland. It is a useful marker in following patients with a history of well-differentiated thyroid cancer once their thyroid glands have been obliterated. Thyroglobulin is often produced in excessive amounts in almost all forms of hyperthyroidism, with the exception of factitious hyperthyroidism. In this case the excessive, exogenous thyroid hormone suppresses TSH, which in turn stops endogenous thyroid hormone and thyroglobulin production from the thyroid gland.

What happened?

When presented with the above findings, the patient admitted taking levothyroxine (or l-thyroxine) at a dose of 0.2 mg per day. She got the prescription from her close friend and hoped the medication would give her more energy and help her deal with the stress of carrying on her career and looking after her family.

A frank discussion with the patient and her husband took place. Mention was made that excessive amounts of thyroid hormone in a euthyroid individual will not result in more energy. In fact, it would likely have the opposite effect, not to mention possible long-term effects on bones and the heart. It was also suggested that a more equitable sharing of domestic responsibilities between the patient and her husband might help alleviate her stress and fatigue. Dx.