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THE NUTRI-SPEC LETTER

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From:
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Dear Doctor,

RED FLAG! RED FLAG! RED FLAG!

In taking the case history on your new patient you find that she is on hormone replacement therapy. Red Flag!

You know that the myth of estrogen's purported benefits is a cruel hoax perpetrated on unsuspecting women by the pharmaceutical industry. You know that nothing will more limit the benefits this patient will derive from your NUTRI-SPEC care than the daily assault of estrogen on her body chemistry. You must help her off it immediately.

Your next new patient is suffering from hypertension for which he takes a calcium channel blocker. Red Flag!

You know that calcium channel blockers always have a devastating (and even dangerous) impact on health. They increase your patient's chance of having a stroke, increase his chance of having a heart attack, cause extreme (even suicidal) depression, as well as loss of mental function (often interpreted as early Alzheimer's). Before you proceed with NUTRI-SPEC on this patient you must convince him to (slowly, responsibly, and safely) get off the drug.

The next patient to seek your expertise in clinical nutrition is taking Prozac for depression. Red Flag!

You know the whole story on how SSRI's are a fascinating example of pharmaceutical industry propaganda. You know that serotonin does not relieve depression -- it actually causes depression and anxiety, while

also negatively impacting health in myriad ways. Your highest priority with this patient must be to get her off the Prozac. [If she absolutely needs an anti-depressant, then suggest that she request a switch to either Sinequan or Wellbutrin. Sinequan is far superior in that it actually decreases serotonin stress in the brain. However, it is not a fashionable drug anymore so you are more likely to find a physician willing to make the switch from an SSRI to Wellbutrin, which is at least far less damaging than the SSRI's.]

Now, you are faced with another new patient, one of the millions who is …

TAKING SYNTHROID FOR THYROID INSUFFICIENCY.

Synthroid is on your list of "Red Flag" medications that are certain to be dragging your patients down. Here is the story …

Unlike estrogen, calcium channel blockers, and SSRI's, Synthroid is not inherently bad. There are actually many patients who benefit from taking Synthroid short-term, and some that even do well on that drug long-term.

In many patients, however, Synthroid is a powerful agent of destruction, which must be discontinued immediately. This letter will focus on helping you to distinguish those patients, and to come up with the best alternative.

There is one problem with Synthroid that is of relatively minor significance. You may be aware of it because it has been in the news quite a lot in the last year or so.

The company with the rights to manufacture Synthroid was bought out by another pharmaceutical company, which, for a while, was unable to obtain FDA approval to continue selling Synthroid. The problem was that the FDA had countless examples of Synthroid being manufactured with such poor quality control that the quantity in the tablet was frequently found to be nowhere near what it was supposed to be.

It all made quite a big fuss for a while, but seems to have died down to some extent. Nevertheless, you may have experienced, as I have, that many patients who were taking Synthroid for years and years and years have been switched to another form of thyroid medication because some insurance companies now refuse to pay for Synthroid.

But the fact that Synthroid has been found to be suffering from poor quality control is not your main concern.

There is a major problem with Synthroid that you must understand, then explain to your patients whenever appropriate. The nasty truth that no one wants to face is that …

IN MANY PATIENTS, SYNTHROID ACTUALLY DECREASES THYROID ACTIVITY.

How can taking thyroid hormone impair the cellular response to the thyroid gland? It seems illogical – so, to explain …

The thyroid gland produces T4 and T3 in an approximate ratio of 2.5:1. T3 is the active thyroid hormone for which every cell in the body has receptors. T4, the storage form of thyroid hormone, circulates and is converted as needed into T3 by the liver, and also to some extent by the kidney and other tissues. An insignificant amount of T4 is converted into reverse T3 (RT3), which has no biological activity, other than that it binds with the T3 receptor sites, thus blocking the action of T3.

Under normal conditions this small percentage of RT3 with respect to T3 creates no problems. However, in a stress response that involves excess cortisol output, the cortisol inhibits the conversion of T4 to T3, while simultaneously favoring the conversion of T4 to RT3. Stress of sufficient intensity or duration results in a RT3 dominance that persists even after the stress passes and the cortisol level falls back to normal. This condition is complicated by the fact that RT3 itself behaves much like cortisol in that it blocks the conversion of T4 to T3.

Thus is created a condition in which there is insufficient thyroid activity despite normal thyroid gland output (and a normal serum thyroid hormone profile).

Consider that very carefully.

The particular thyroid dysfunction you are learning about does not involve insufficient thyroid gland output of T4 and T3, but rather an inadequate peripheral conversion of T4 (the storage form of thyroid hormone) into the active T3. I know of two names by which this clinical condition has been called: Low T3 syndrome, and Reverse T3 dominance syndrome.

What does all this have to do with Synthroid?

Synthroid is pure T4, with no active hormone. The idea (hope) in prescribing it is that the T4 will fill the roll of the underactive thyroid gland, and be converted to active T3 as needed. For some patients with a primary thyroid insufficiency the T4 works beautifully:

- elevated serum TSH and low T4 return to normal
- subnormal body temperature rises to average 98.6
- a sluggish pulse rises to 72
- elevated serum cholesterol falls toward 200
- fatigue improves dramatically
- the somnolence by day and insomnia at night improves
- fluid retention decreases
- % body fat decreases
- cramping and hypertonic muscles are relieved
- deep tendon reflex recovery normalizes
- fibromyalgia eases up
- apathy or depression gives way to a new lease on life.

Some of these happy patients may need to rely on Synthroid for a lifetime; others for less than a year. In either case, these are simple thyroid insufficiencies that respond to a simple, direct solution.

Regrettably, however, few cases of thyroid insufficiency are this simple. The most common complication is that many patients with primary thyroid insufficiency (inadequate thyroid gland output) also have Reverse T3 dominance syndrome.

What happens when a case of RT3 dominance is given T4 (Synthroid)?

THE PATIENT IS DEVASTATED BY
A DOUBLE DOSE OF THYROID DEPRESSION.

First, the thyroid takes a major hit by virtue of Synthroid's extremely effective pituitary inhibition. You see, the pituitary relies on feedback from the thyroid in the form of T4 to determine its output of TSH to stimulate thyroid production. Under normal circumstances, this is a very effective feedback loop.

The problem with Synthroid is that it takes so little T4 to make the pituitary happy. In fact, the pituitary can become quite complacent in response to T4 feedback long before there is adequate thyroid function throughout the body. A satisfied pituitary sends no TSH to the thyroid; and no TSH to the thyroid means depressed T4 and T3 production by the thyroid.

What are you left with now? A patient who had low T3 to begin with, yet whose T3 insufficiency is being further exacerbated by inadequate TSH stimulation.

But Synthroid's depression of primary T3 production is only half of its 1-2 knock out punch. What do you think happens to this pharmacological dose of T4 in a patient that is already suffering from a stress-related excess conversion of T4 to T3? You guessed it – the Synthroid is not converted into T3 but into RT3, which, as you have already learned, further blocks thyroid function peripherally.

Is it any wonder you have so many patients for whom Synthroid has supposedly restored normal thyroid function, yet who have all the hypothyroid symptoms they began with, and more!

The clinical significance of Synthroid damage cannot be overstated; nor can the surprisingly high incidence of RT3 dominance. Some of the stressors that have been shown to cause low T3 syndrome or reverse T3 dominance are fasting (including repeated weight loss diets), surgery, burn trauma, alcoholism, endotoxin injection, and the clinical use of glucocorticoids including cortisone shots and prednisone therapy. The one common finding that all patients subjected to these stressors show, in addition to low T3 and elevated RT3 levels, is excess cortisol.

More on how to deal with this red flag in next month's Letter. Meanwhile – examine the list of references provided below.

In the New Year, may you experience clinical success beyond your greatest expectations.

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