KRESSER INSTITUTE
FOR FUNCTIONAL AND EVOLUTIONARY MEDICINE

Nutrition: Thyroid Disorders – Part 1

Hey, everybody, in this presentation we're going to talk about nutrition for thyroid disorders. More than 20 million Americans have thyroid disease. Sixty percent of people who have a thyroid condition are unaware that they have it. One in eight women will develop a thyroid disorder in their lifetime. Levothyroxine, or Synthroid, is now the number one selling prescription drug with 21.5 million prescriptions sold per month. This is more prescriptions than Crestor, which is a statin drug, or Nexium, which is a proton pump inhibitor. Up to 10 percent of women over 60 have clinical or subclinical hypothyroidism.

Every cell in the body has receptors for thyroid hormone. Thyroid hormone impacts the brain, the gastrointestinal tract, cardiovascular system, bone metabolism, red blood cell metabolism, gallbladder and liver, steroid hormone production, glucose, protein and lipid metabolism, and body temperature regulation, just to name a few systems. The standard of care for thyroid disorders in conventional and even in alternative medicine, however, is hopelessly inadequate. The basic treatment is just thyroid hormone replacement; a common alternative treatment might be iodine, and there's rarely any investigation into what might be causing or contributing to the thyroid disorder in the first place. And unfortunately, iodine, while it can be helpful in some cases, may be contraindicated in other cases, and thyroid hormone medication, while it can be helpful and even necessary, doesn't often address the underlying cause of the problem.

At least 90 percent of people with thyroid disorders, according to some studies, are producing antibodies to thyroid tissue. Autoimmune hypothyroidism is called Hashimoto's disease or Hashimoto's thyroiditis, and hyperthyroidism with an autoimmune component is known as Grave's disease. Most cases of hyperthyroidism are also autoimmune. Hashimoto's is the most common autoimmune disorder in the US; it affects 7 to 8 percent of the American population, and conventional medicine does not really offer any treatment for Hashimoto's other than thyroid hormone replacement. And though in some cases of autoimmune disease, like inflammatory bowel disease or rheumatoid arthritis, conventional medicine might use immunosuppressive drugs. In the case of Hashimoto's, it's generally thought that the complications and potential side effects of these immunosuppressive drugs outweigh the benefit in Hashimoto's, so they're rarely used. The standard of care is just to wait until the immune system has destroyed enough thyroid tissue to warrant hormone replacement. Other symptoms, like depression or insulin resistance that might develop as a result of Hashimoto's, would just be medicated as well. They might prescribe an antidepressant or a drug like metformin to deal with the insulin resistance. It's important to understand that Hashimoto's is a polyendocrine autoimmune pattern, so this means that it's not uncommon for Hashimoto's patients to have antibodies to other tissues like transglutaminase or cerebellum or intrinsic factor, glutamic acid decarboxylase, and antibodies produced to these other tissues can cause other concurrent autoimmune diseases, like, for example, celiac disease in the case of transglutaminase, or neurological disorders in the case of cerebellum antibodies, pernicious anemia in the case of intrinsic factor antibodies, and then anxiety and/or late onset type one diabetes in the case of glutamic acid decarboxylase antibodies.

The symptoms of hypothyroidism are diverse, as you would predict since every cell in the body has receptors for thyroid hormone. They include mental slowing, difficulty concentrating and brain fog, depression, weight gain, constipation, dry skin, hair loss, cold intolerance or cold hands and feet, hoarse voice, irregular menstruation, muscle stiffness and pain, fatigue, high cholesterol, and dry skin, brittle nails, dry hair, and hair loss. Those are probably the most predominant symptoms, but it's only a partial list, and as I said, if you understand that thyroid hormone affects just about every cell in the body, you'll see a really wide range of symptoms in your patients with hypothyroidism.

Symptoms of hyperthyroidism are kind of the mirror image of the hypothyroid symptoms; it'd be excess sweating, excess hunger, heat intolerance, tachycardia, arrhythmia, abnormal heart rate or rhythm, irregular light menstruation, nervousness or anxiety, exophthalmos, which is a protrusion of the eyes, insomnia, irritability, weight loss, diarrhea, muscle weakness, tremor, and hair loss.

When thinking about hypothyroidism, the conventional interpretation is you just measure TSH, thyroid-stimulating hormone, and if it's high, that's hypothyroidism, and if it's normal, it's not, and oftentimes that's about as deep as the investigation goes. But there are several patterns of thyroid dysfunction that are important to be aware of that may not show up on the standard lab tests, especially if you're just looking at thyroid-stimulating hormone. So, one is hypothyroidism that's caused by a pituitary dysfunction. So this can be a result of elevated cortisol from chronic stress or blood sugar imbalances, insulin resistance, pregnancy, or active infection. And what can happen there is the signal that's sent from the pituitary to the thyroid gland is inadequate to produce enough thyroid hormone, so instead of getting a high TSH, you might have a low, low-normal TSH but the thyroid hormone output will be low as a result, and so you can either see normal TSH with low thyroid hormones or low-normal or even low TSH with low thyroid hormones, and that's not a typical presentation, and it will often be overlooked. Another pattern is underconversion of T4 to T3, so most of what's produced in the thyroid gland is T4, and that T4 has to be converted into the more active form of thyroid hormone, which is T3, before the body can use it. And that conversion is reduced or blocked by things like inflammation, elevated cortisol levels or deficiencies of certain nutrients, also by gut problems. Now in some cases, the underconversion of T4 to T3 will be significant enough to lead to an increase in TSH that will show up on a lab test, but in other cases it might be subclinical, in the sense that TSH won't actually increase, or at least it won't go above the standard lab reference range, and these patients will often be missed. Another pattern would be elevated thyroid-binding globulin. So thyroid-binding globulin, or TBG, is the protein that transports thyroid hormone throughout the blood, and if TBG levels are too high, that will cause low levels of free or unbound thyroid hormone, which is the form of the thyroid hormone that's actually available to activate cellular transcription. So free thyroid hormone is what does the job of thyroid hormone in the body, and elevated thyroid-binding globulin levels can be caused by high estrogens, which is often associated with birth control pills or hormone replacement therapy in older women, and the excess estrogen has to be cleared from the body in that case, and again, this will often not show up on a standard blood thyroid panel that's just looking at TSH and even T4 and T3. The flipside of that is decreased thyroid-binding globulin, so that can lead to too much free thyroid hormone, and that could cause hyperthyroidism in the short term, but in the long term

what happens there is in an effort to protect itself, the body makes these cells resistant to the effect of thyroid hormone because there's too much of it floating around in the blood, so it can either downregulate thyroid hormone receptor sensitivity or downregulate the expression of thyroid hormone receptors, and then the patient ends up with hypothyroid symptoms, and this can be caused by high testosterone in women, especially like you'd see in PCOS and insulin resistance, and the treatment there would be to restore insulin sensitivity and balance androgenic hormones. And then the last pattern is thyroid resistance, so we just kind of talked about this in the context of the last pattern. This is when there may not be an issue with the production of thyroid hormone, but more an issue in the sensitivity of cells to thyroid hormone, and this doesn't necessarily show up on a lab test either. So this should give you some idea of the complexity here and some of the underlying factors that you have to consider when a patient has a thyroid issue. It goes far beyond just looking at their TSH and often beyond looking at even their TSH and T4 and T3.

Dietary recommendations for hypothyroidism, Hashimoto's, and then Grave's disease, hyperthyroidism, depend on the etiology of the condition. Different patterns of thyroid dysfunction require different focus for treatment, including diet and lifestyle recommendations. So, autoimmune thyroid disorders versus non-autoimmune mediated thyroid disorders would have different etiology; in one case it's the immune system attacking the thyroid, in the other case it might be a nutrient deficiency like selenium or iodine or zinc; and thus you'll have different diet and lifestyle factors that are required to address each of those particular problems.