Hashimoto’s disease

Hashimoto’s Disease was first described by a Japanese doctor in 1912. Hashimoto’s disease is a description often levelled at anyone with hypothyroidism, in much the same way as Graves’ Disease is with hyperthyroidism. The thing to understand about Hashimoto’s disease is that it is basically a variant of hypothyroidism, caused specifically by autoimmunity; the thyroid gets infiltrated by white cells and slowly loses its function; it usually enlarges but may not, and may start with an over-active phase before becoming under-active.

In fact, both Graves’ disease and Hashimoto’s disease are forms of autoimmune thyroiditis, where the body’s defence mechanisms overact or respond dysfunctionally, and cause damage to target tissues.

Hashimoto’s disease, stress and pregnancy

Hashimoto’s disease is relatively common and has several ways of presenting itself. Both Graves’ and Hashimoto’s disease can be triggered off by stress factors and also pregnancy. There is a genetic predisposition for both conditions, and both may involve the infiltration by white cells (lymphocytic infiltration), increased immunoglobulins within the gland, and increased blood levels of thyroid antibodies. Each can change into the other, although more commonly it is the over-active thyroid resulting from immune dysfunction that will revert to the under-active phase.

Hashimoto’s disease should more properly be called chronic immune thyroiditis and can be detected chemically by the presence of antibodies, the rise in TSH, and low blood levels of thyroid hormones. Where the TSH rises, but the thyroid hormone levels remain in range, the condition can be called subclinical hypothyroidism. It is worth noting that the antibody levels may diminish as hypothyroidism becomes established, or where treatment with thyroxine is given.

Hashimoto’s disease and blood tests

It frequently happens that Hashimoto’s disease is missed because of undue reliance on blood tests. Usually, the T4 is low, although it may not be out of range. So too, maybe the T3. This will suggest poor thyroid hormone output. While in the normal course of events the TSH normally rises in this situation, it may actually be normal, or even low.

The reason for this is a down-grading of the hypothalamic/pituitary axis due to the state of hypometabolism that the low thyroid function induces. (This also applies to adrenal function which can also be damaged by autoimmune disease.)

The hypothalamus responds poorly to the low thyroid blood levels, and may not produce a normal level of Thyrotrophin Releasing Hormone (TRH). In addition, the pituitary, also affected adversely by the state of hypo-metabolism, may not properly respond to the TRH, and not, therefore, produce a normal level of TSH.

The thyroid itself, being damaged by the white cell infiltration and with damaged TSH receptors,
may well be less responsive to TSH anyway. So there is a chain reaction of failure, beginning with
the hypothalamus and extending to the pituitary. The antibody test is usually pretty conclusive and
any level of thyroid antibodies will suggest an autoimmune process at work; although as noted
above, as the damage becomes chronic the levels may lessen.

**How Hashimoto’s disease presents**

Clinically, there is a range of features to be found in patients with autoimmune thyroiditis. Firstly,
there may be nothing to show for it at all, and it is noticed during general blood “work up” for
something else. Sooner or later though, hypothyroidism becomes clearly evident. Of the various
ways it shows itself, we can pick out two.

- **Goitrous Autoimmune Thyroiditis** – here the progressive infiltration of white cells enlarges the
thyroid, and this is the version Hashimoto first described. In addition to the white cells taking
over the glandular tissue, the gland itself becomes a mass of fibrous tissue, with the follicular
cells (where the thyroid hormone is made) disappearing. The gland itself, of course, becomes
enlarged into a goitre. Sometimes the fibrous tissue takes over completely. It has been found
also that an increase in dietary iodine has a tendency to worsen autoimmune thyroiditis.

- **Atrophic Autoimmune Thyroiditis** – this is the most common form and results in the thyroid
gland shrinking with progressive loss of tissue. The antibodies block the TSH receptors in the
thyroid and on the basis that what you don’t use, you lose, the glandular tissue shrinks.

While Hashimoto’s disease is simply a common cause of hypothyroidism, the term should not be
used for hypothyroid conditions unless antibodies are present although the treatment remains the
same as for any hypothyroid state.

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