High radio-isotope uptakes in patients with hypothyroidism

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Summary

Hypothyroidism is usually associated with a low radio-isotope uptake by the thyroid gland. We report 8 cases of Hashimoto's thyroiditis with clinical and biochemical hypothyroidism and with borderline high or overtly increased technetium-99m pertechnetate and/or iodine-131 uptakes.

Hashimoto's disease is a relatively common disorder, the highest incidence being in middle-aged women. A diffuse goitre is a typical feature. An auto-immune process is the major abnormality in the pathogenesis of the disease, borne out by the presence of an intense lymphocytic infiltration of the gland, the presence of high antibody titres directed against several thyroid-related antigens and the frequent coexistence of other presumed autoimmune disorders. Early on in the disease radio-isotope uptake by the thyroid gland may be increased. At this stage the patients are usually euthyroid but may be mildly thyrotoxic with elevated thyroxine levels.1 With time, the uptake and the serum thyroxine concentration decline as hypothyroidism supervenes. Hashimoto's disease is thus associated with raised, normal or diminished radio-isotope uptakes. We report and discuss the combination of overt clinical and biochemical hypothyroidism and increased isotope uptakes.

Patients and methods

The 8 patients were all referred for investigation of hypothyroidism and/or goitre. Clinical assessment included documentation of relevant symptoms and physical signs. Routine thyroid function tests were performed including tri-iodothyronine (T₃) resin uptake (T₃U), total serum thyroxine (TT₄), free thyroxine index (FTI), and serum thyroid-stimulating hormone (TSH) concentration. Antimicrosomal and antithyroglobulin antibody titres were assayed by routine methods. Thyroidal uptake of technetium-99m pertechnetate (⁹⁹ᵐTc) was assessed after 20 minutes and/or uptake of radioactive iodine (¹³¹I) after 24 hours.

Results

Clinical data, thyroid function, antibody titres and isotope uptakes are shown in Table 1. All the patients studied were female; 2 were children, 3 were young adults and the remaining 3 elderly. Seven patients presented at the onset with the classic clinical features of hypothyroidism, and 6 had small-to-moderate, firm, asymmetrical goitres. All 8 patients had strongly positive antimicrosomal antibody titres, while positive antithyroglobulin titres were evident in 5. All 8 had significantly elevated serum TSH concentrations (mean 78.5 μIU/ml). Five patients showed subnormal TT₄ and FTI levels while 3 had levels in the low normal range. In 5 of the 6 patients who received ⁹⁹ᵐTc, the uptake measured was in the supranormal range while in 1 case it was in the high normal range. Three patients underwent 24-hour ¹³¹I uptake measurements; in 1 patient this was clearly increased (as was the ⁹⁹ᵐTc uptake) and in 2 patients the uptakes were in the high normal range.

Discussion

A clinical diagnosis of thyroid failure due to Hashimoto's disease was made in all 8 patients. All were clinically and biochemically hypothyroid and all had strongly positive microsomal antibody titres. In Hashimoto's disease thyroid isotope uptake may be high, normal or low and in the hypothyroid state the uptake is typically decreased,2,3 although uptake may be increased in the presence of a subnormal secretion of thyroxine.4 In all patients thyroid isotope uptake was in the supranormal to high normal range, despite the presence of both clinical and biochemical thyroid failure (5 patients) and compensated biochemical euthyroidism (3 patients).5 All our patients had significantly raised serum TSH concentrations. Technetium is not organically bound in the human thyroid gland,6 and the early thyroid uptake assessment primarily reflects the iodide-trapping mechanism of the gland,7 which is TSH-dependent. The raised ⁹⁹ᵐTc uptake in our patients may thus reflect a normal or supranormal iodide-concentrating mechanism, despite the presence of hypothyroidism and low thyroxine levels. The hypothyroidism of Hashimoto's disease is in part attributable to the marked derangement in intrathyroid iodine utilization8-11 and it is suggested that the iodide-binding organisation process is more susceptible to auto-immune damage than is the iodide-trapping concentrating mechanism. Thus the raised isotopic uptake which may be evident in Hashimoto's thyroiditis demonstrates an intact TSH-dependent iodide-trapping mechanism in an otherwise hypothyroid patient whereas a primary defect of impaired iodide organisation may be confirmed by an abnormal perchlorate discharge.12 It is therefore concluded that an elevated isotope uptake by the thyroid is compatible with the hypothyroidism of Hashimoto's disease.

REFERENCES


### Table 1: Clinical Data, Antibody Titres, Thyroid Function and Radio-isotope Uptake

<table>
<thead>
<tr>
<th>Antibodies</th>
<th>Classic clinical hypothyroidism</th>
<th>Goitre</th>
<th>FT4 (nm/l)</th>
<th>T3 (µg/dl)</th>
<th>T4 (%)</th>
<th>TSH (µIU/ml)</th>
<th>Thryoglobulin</th>
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<tr>
<td>Microsomal</td>
<td>Positive</td>
<td>+</td>
<td>13</td>
<td>43.0</td>
<td>30.0</td>
<td>&gt; 400</td>
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<tr>
<td>Goitre</td>
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<td>7</td>
<td>25.0</td>
<td>30.0</td>
<td>100</td>
<td>+</td>
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<tr>
<td>C/F</td>
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<td>48.0</td>
<td>35.0</td>
<td>10.0</td>
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<td>28</td>
<td>78.0</td>
<td>NA</td>
<td>17-60</td>
<td>++</td>
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</table>

**FT4** = Free Thyroxine, **TSH** = Thyroid Stimulating Hormone.