

## CASE REPORT

# A case of Hashimoto's thyroiditis following Graves' disease

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**Abstract**

Graves' disease is characterized by the presence of circulating autoantibodies that stimulate the TSH receptor, inducing hyperthyroidism and goiter. Hashimoto's thyroiditis is an autoimmune disease leading to thyroid tissue destruction by cell and antibody-mediated immune processes. The occurrence of Hashimoto's thyroiditis following Graves' disease has been rarely reported. Its pathogenesis is not clear. Herein, we report the case of a 40-year-old woman who was referred to our department for thyrotoxicosis. Laboratory tests revealed overt hyperthyroidism. Thyroid scintigraphy showed an enlarged gland with diffusely increased tracer uptake, confirming the diagnosis of Graves's disease. The patient was treated with propranolol and thiamazole. Two months later, she received radioactive iodine therapy. Three years and 9 months later, the patient presented with hypothyroidism and very high levels of thyroperoxidase antibodies consistent with the diagnosis of Hashimoto's thyroiditis. She was treated with levothyroxine. The shift from Graves' disease to Hashimoto's thyroiditis was reported in the literature. However, its pathogenesis has not been clearly elucidated.

**KEYWORDS**

Graves' disease, Hashimoto' thyroiditis, hyperthyroidism, hypothyroidism, TRAb, TSBAb

## 1 | INTRODUCTION

Graves' disease and Hashimoto's thyroiditis are both autoimmune diseases of the thyroid gland. Graves' disease is typically characterized by the presence of circulating autoantibodies that stimulate the TSH receptor (TRAb), inducing hyperthyroidism and goiter.<sup>1</sup> Hashimoto's thyroiditis is a chronic thyroiditis in which lymphocytic infiltration and thyrocyte damage may progress to hypothyroidism.<sup>2</sup> It is associated with thyroid peroxidase and thyroglobulin autoantibodies. TSH stimulation-blocking antibodies

(TSBAb) may be present in patients with Hashimoto's thyroiditis causing a blockage of the TSH action and consequently atrophy of the thyroid gland.<sup>3</sup> In the early stage of Hashimoto's thyroiditis, mild and transitory hyperthyroidism may occur as a result of thyroid cells destruction and the release of thyroid hormones into blood circulation.<sup>4</sup> The development of Hashimoto's thyroiditis following Graves' disease was rarely reported. Its pathogenesis has not been confirmed.

Herein, we report a case of Hashimoto's thyroiditis following Graves' disease treated with radioiodine.

## 2 | CASE PRESENTATION

A 40-year-old woman was referred to our department for thyrotoxicosis. Her past medical history was unremarkable, with no history of autoimmune diseases. She was not taken any drugs. There was no family history of autoimmune diseases.

She presented with weight loss, palpitations, excessive sweating, tremor, and nervousness.

On physical examination, she had a body weight of 65 kg, a body height of 1m58 corresponding to a body mass index of 26 kg/m<sup>2</sup>, a blood pressure of 120/80 mmHg, a regular heart rate of 112 beats/min; a diffusely enlarged thyroid gland, and a tremor in both hands. No signs of Graves's orbitopathy were observed.

Laboratory tests revealed overt hyperthyroidism with free thyroxine (FT4) level at 77.23 pmol/L (normal range: 9.01–19.30) and a thyroid-stimulating hormone (TSH) level of 0.001 mIU/L (normal range: 0.35–4.94). Thyroid scintigraphy showed an enlarged gland with diffusely increased tracer uptake, confirming the diagnosis of Graves's disease. Thyroid antibodies were not available.

The patient was treated with propranolol and thiamazole. Two months later, she received radioactive iodine therapy. Euthyroidism was achieved following radioiodine therapy and antithyroid drug discontinuation. Three years and nine months later, the patient presented with weight gain and asthenia. On examination, she had a normal-sized thyroid gland. Laboratory investigations revealed a TSH level of 8.787 mIU/L, a FT4 level of 12.2 pmol/L, and a thyroperoxidase antibodies level of 2208 IU/ml (normal range: <35 IU/ml) consisting with the diagnosis of Hashimoto's thyroiditis. She was treated with levothyroxine.

## 3 | DISCUSSION

Hyperthyroidism is defined by elevated levels of FT4 and/or free triiodothyronine (FT3) and a low level of serum TSH. Its etiologies include Graves' disease, toxic multinodular goiter, toxic adenoma, and thyroiditis. Hashimoto's thyroiditis can be initially expressed by a hyperthyroid phase called hashitoxicosis that results from thyroid cells destruction and the release of preformed thyroid hormones into blood circulation. It is generally a mild and transitory hyperthyroidism. Based on the severity of hyperthyroidism and the scintigraphic features, the diagnosis of Graves' disease was established in our patient.

Graves' disease represents the most common cause of hyperthyroidism. Its natural history in the absence of treatment is not well known. However, it is estimated that 60% to 70% of the patients with Graves' disease follow an

undulating course with alternating hyperthyroid and euthyroid phases, and that about 30% to 40% experience only one hyperthyroid episode.<sup>5</sup>

Treatment options for Graves' disease include antithyroid drugs, radioiodine therapy, and surgery. Our patient was initially treated with antithyroid drugs and secondary she received radioiodine therapy.

The occurrence of hypothyroidism in patients with Graves' disease may be caused by surgical treatment (thyroidectomy) or radioiodine therapy. The latter is a safe and effective treatment of hyperthyroidism. Iodine-131 is a beta-emitting radionuclide. It is taken up by the thyroid gland and incorporated into thyroid hormones.<sup>6</sup> The release of beta particles is responsible for ionizing damage and tissue necrosis, gradually leading to thyroid volume reduction and the control of the thyrotoxicosis.<sup>7</sup> Hypothyroidism following radioiodine occurs in 80%–90% of the patients, generally during the first year after treatment.<sup>6,8</sup> However, in some cases, hypothyroidism may develop several years post radioiodine therapy.<sup>9</sup> In our case, hypothyroidism manifested three years and nine months following radioiodine therapy. Thus, it may be secondary to this therapeutic method. However, the very high levels of thyroperoxidase antibodies are consistent with the diagnosis of Hashimoto's thyroiditis. The diagnosis of Hashimoto's thyroiditis relies on the clinical manifestation of hypothyroidism with high TSH levels, and the occurrence of thyroid antibodies.

About 15%–20% of patients with Graves' disease may develop spontaneous hypothyroidism due to Hashimoto's thyroiditis after antithyroid drugs discontinuation.<sup>10–12</sup> Umar et al.<sup>4</sup> reported four cases of Hashimoto's disease in patients who have been previously diagnosed with Graves' hyperthyroidism and treated with antithyroid drugs. Hashimoto's thyroiditis manifested 7 to 25 years after the treatment of Graves' disease in three cases and after a few months in one patient. On the contrary, Sukik et al.<sup>13</sup> reported a very rare case of Hashimoto's thyroiditis shifting to Graves' disease after sixteen years from the initial diagnosis. Both Graves' disease and Hashimoto's thyroiditis share autoimmunity pathogenesis and the shift from one condition to another is possible.<sup>13</sup> However, the pathogenesis of Hashimoto's thyroiditis following Graves' disease has not been confirmed. Many theories were suggested. The most plausible one is the simultaneous presence of both blocking and stimulating antibodies. The alterations in thyroid function are related to the balance in the activity of TRAb and TSBAb.<sup>14</sup> In patients with Graves' disease, it was demonstrated that antithyroid drugs may lower the TRAb level. In patients who have coexisting stimulating and blocking antibodies, decreased TRAb level may increase the action of TSBAb, which eventually causes Hashimoto's thyroiditis.<sup>15</sup>

## 4 | CONCLUSION

Both Graves' hyperthyroidism and Hashimoto's thyroiditis are thyroid autoimmune diseases.

The shift from Graves' disease to Hashimoto's thyroiditis was reported in the literature. However, its pathogenesis has not yet been confirmed. This case highlights the challenges of managing a patient with Graves' disease and spontaneously oscillating thyroid function. Therefore, a long-term follow-up of thyroid functions in patients with Graves' disease is necessary.

### AUTHOR CONTRIBUTIONS

IO involved in conception and design, acquisition and interpretation of data, manuscript creation and drafting; SS involved in manuscript creation and drafting; MC critically revised the article for important intellectual content; all authors were involved in the management of this patient and the revision of the manuscript and approved the final version.

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### CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

### DATA AVAILABILITY STATEMENT

Data sharing not applicable - no new data generated

### ETHICAL APPROVAL

Ethical approval for this case report was not required.

### CONSENT

A written informed consent was obtained from the patient for the publication of this report.

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