Imaging in Endocrinology

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with website

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About the Companion Website

This book is accompanied by a companion website:
www.wiley.com\go\Pozzilli\endocrinemetabolicdisease

The website includes:

- Powerpoints of all figures from the book for downloading

Preface

No medical discipline requires such a precise phenotypic classification and careful consideration of the "image" as does endocrinology. Indeed, it is from "observation" that the endocrinologist extrapolates the elements upon which he or she bases clinical reasoning in the identification of a medical condition.

This atlas aims to be a valuable guide in endocrine diagnosis – suitable for both specialists and physicians in training, as well as physicians in other disciplines with an interest in endocrine disorders. Using the image as a unifying theme, we address the most salient themes of the science of endocrinology – including thyroid, pituitary, adrenal, endocrine pancreas, bone and mineral metabolism, and gonads. Each section provides iconographic support for the pathologies examined.

The universal character of this atlas guarantees a high standard of quality. The work was carried out in both Italy and the USA and has the added benefit of combining the rigor and scientific integrity belonging to the cradle of modern endocrinology (Rome) and the clinical resources of a major quaternary endocrine referral center (Mayo Clinic).

We would like to express our gratitude to all those who collaborated on this project. Their passion and enthusiasm toward the completion of this work has been exceptional. Without their hard work and dedication, this publication would have not seen the light.

We would also like to thank Wiley and its editors, who have demonstrated, once again, the high level of professionalism and special attention to detail needed to successfully bring to fruition this type of publication. We hope you enjoy consulting the Atlas.

Paolo Pozzilli, Andrea Lenzi, Bart L Clarke
and William F Young Jr
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Contents

1 Thyroid

Hashimoto's thyroiditis (chronic autoimmune thyroiditis)

Definition and epidemiology

Hashimoto's thyroiditis (HT), or chronic lymphocytic thyroiditis, is an autoimmune disease in which the thyroid gland is attacked by a variety of cell and antibody-mediated immune processes. The name "Hashimoto's thyroiditis" is derived from the 1912 original report by Hashimoto describing patients with both goiter and intense lymphocytic infiltration of the thyroid (Figs 1.1 & 1.2) as "struma lymphomatosa."

Hashimoto's thyroiditis is the most common cause of primary hypothyroidism in iodine-sufficient areas of the world; it is among the most common causes of nonendemic goiter. On average 1.0–1.5/1000 people suffer from this disease. It occurs far more often in women than in men (incidence of 10 : 1 to 20 : 1, respectively), and it is most prevalent between 45 and 65 years of age. Occurrence in children is also uncommon, especially in populations where iodine is not a dietary scarcity.

Etiology and pathogenesis

Autoantibodies may be present against thyroid peroxidase, thyroglobulin, and thyroid-stimulating hormone (TSH) receptors, although a small percentage of patients may have none of these antibodies present. Antibody-dependent cell-mediated cytotoxicity is a substantial factor behind the apoptotic fallout of HT. Activation of cytotoxic T lymphocytes ($CD8^+$ T cells) in response to cell-mediated immune response affected by helper T lymphocytes ($CD4^+$ T cells) is central to thyrocyte destruction. Recruitment of macrophages is another effect of helper T-lymphocyte activation, with Th1-axis lymphocytes producing inflammatory cytokines within the thyroid tissue to further macrophage activation and migration into the thyroid gland for a direct effect. Infection, stress, sex steroids, pregnancy, iodine intake, and radiation exposure are known possible precipitating factors for HT. Fetal microchimerism within the maternal thyroid is also a possibility.

Signs and symptoms

Hashimoto's thyroiditis very often results in hypothyroidism with bouts of hyperthyroidism. Symptoms of HT include weight gain, depression, mania, sensitivity to heat and cold, paresthesia, fatigue, panic attacks, bradycardia, tachycardia, high cholesterol, reactive hypoglycemia, constipation, migraine, muscle weakness, cramps, memory loss, infertility, hair loss, and myxedematous psychosis.

Diagnosis

Laboratory findings

Laboratory tests for HT include:

- Antithyroid peroxidase antibodies (TPOAbs) and thyroglobulin antibodies (TgAbs)
- TSH, free thyroxine (FT_4)
- Total cholesterol, high density lipoprotein (HDL), and triglycerides

Imaging tests

Imaging tests for HT include:

- Neck ultrasound (Fig. 1.3)
- Computed tomography (CT) scan (rare)
- $^{99m}TcO_4$ thyroid scintigraphy (Fig. 1.4)

Treatment

In patients with primary hypothyroidism, the main treatment is levothyroxine.

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Illustrations (Figs 1.1–1.4)

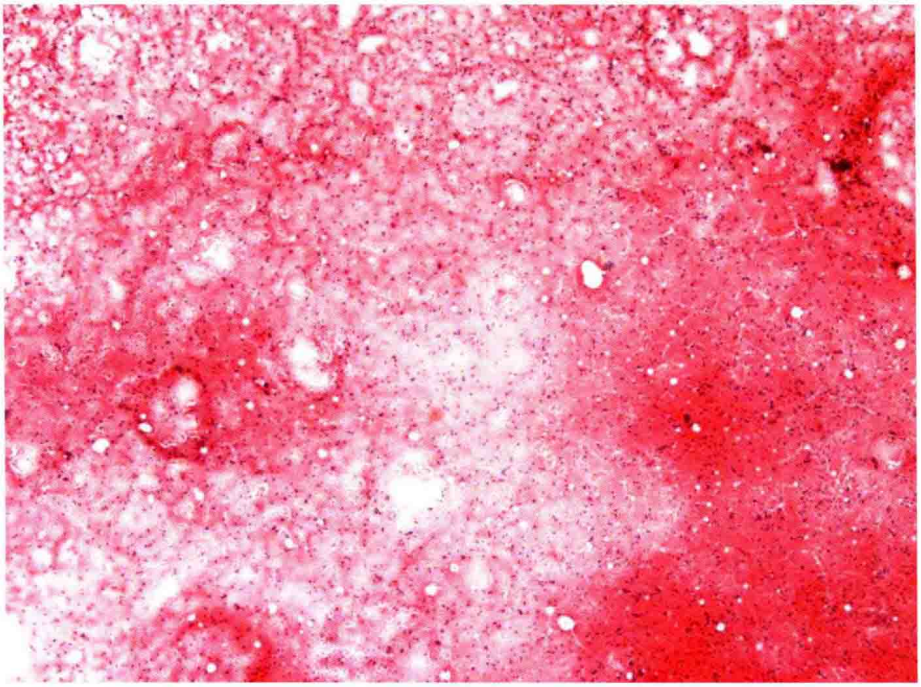


Figure 1.1 Cytology of thyroiditis. This figure shows rare and normal thyrocytes associated with numerous lymphocytes (Papanicolau, 10 ×).

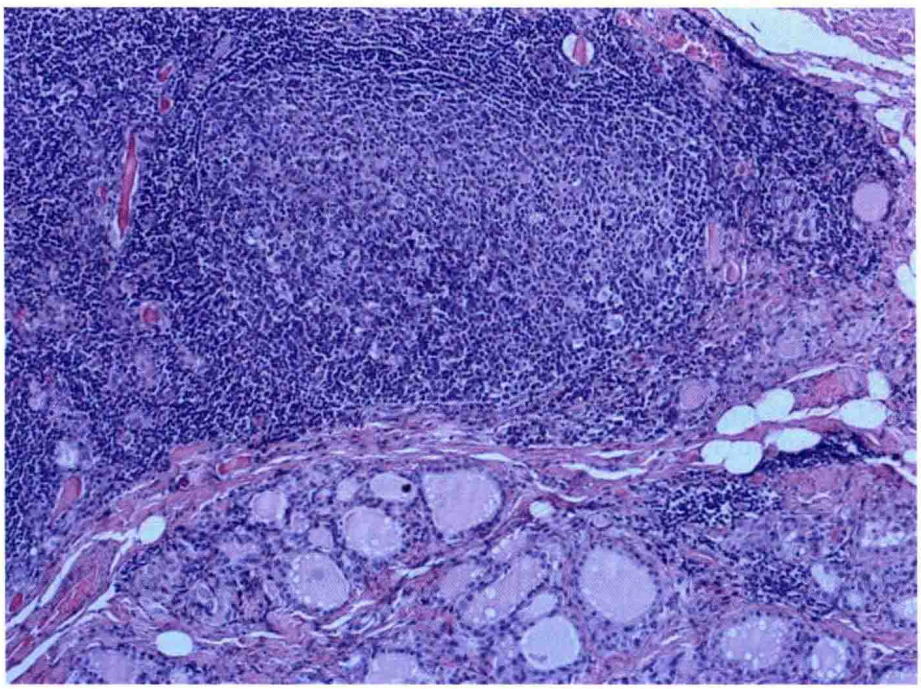
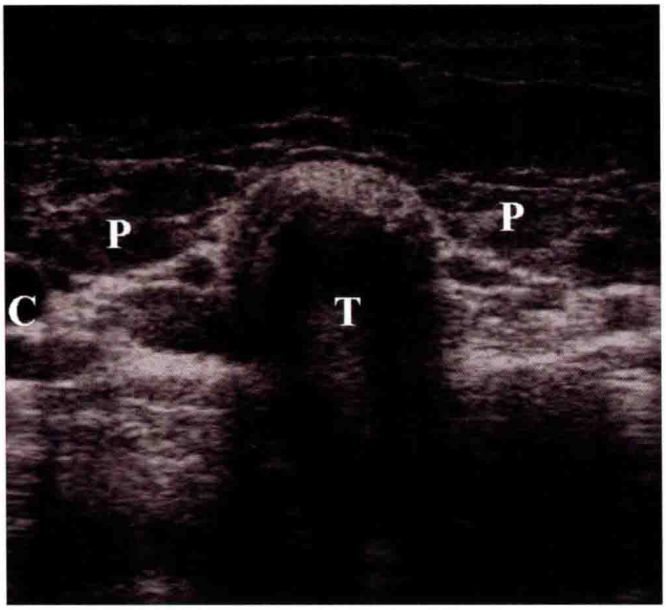


Figure 1.2 Histology of thyroiditis. Hashimoto thyroiditis is characterized by Hürthle cells associated with follicular lymphoid structures (HE, 10 ×).



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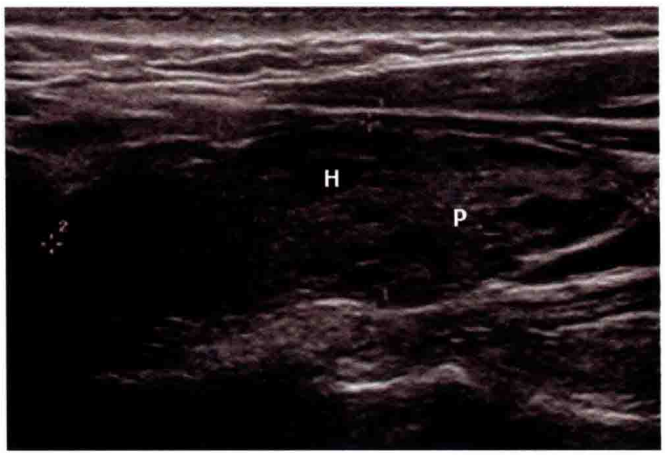
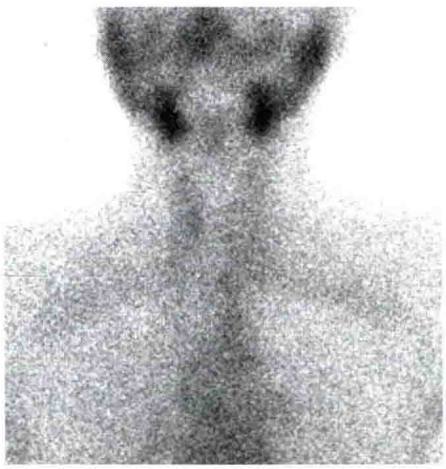


(b)

Figure 1.3 A 46-year-old woman with a recent episode of cervical tenderness and a familiar history of thyroid disease. The patient complained of fatigue and reported a weight gain of about 10 kg in the last 2 months. (a) Thyroid ultrasound – cross section. This ultrasound shows a thyroid with a slight increase in volume, globular shape, and homogeneous structure, and less echogenic than normal. (b) Thyroid ultrasound – longitudinal section. This ultrasound shows diffuse patchy hypoechoic lesions throughout the gland. This sonographic appearance is called a "leopard skin" pattern and is seen in lymphocytic infiltration of the thyroid in Hashimoto's thyroiditis. The hypoechoic lesions within the thyroid are areas of lymphocytic infiltration of the thyroid tissue. C, carotid artery; H, hypoechoic lesions; P, thyroid parenchyma; T, trachea.



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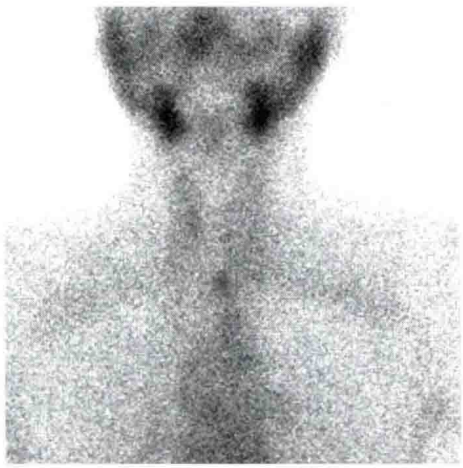

(b)

Figure 1.4 The same patient as in Fig. 1.3: $^{99m}TcO_4$ thyroid scintigraphy with iodine uptake curve. Iodine uptake was 2% at 4 hours (a) and 2% at 24 hours (b). The scan showed no uptake in the thyroid bed. The free triiodothyronine (FT_3) and free thyroxine (FT_4) levels were low with elevated thyroid stimulating hormone (TSH) and antibodies against thyroperoxidase (TPOAb) values. The patient started levothyroxine treatment.