Thyroiditis

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Pierpaolo Trimboli and Luca Giovanella

Abstract

Thyroiditis is an inflammation of the thyroid gland that has several etiologies and can be associated with normal, elevated, or depressed thyroid function, often with evolution from one condition to another. Basing on clinical presentation, different subtypes of thyroiditis can be divided into those associated with thyroid pain and tenderness, and those that are painless. The most frequent painful form is subacute thyroiditis, while the most frequent painless form is autoimmune thyroiditis (i.e., Hashimoto's disease). In this chapter, some principles of differential diagnosis, therapy, and follow-up of thyroiditis are briefly discussed. Furthermore, some typical images of thyroiditis are provided.

Keywords

Subacute thyroiditis • Autoimmune thyroiditis • Hashimoto's disease • Inflammation • Thyroiditis • Thyroid

P. Trimboli, MD
Section of Endocrinology and Diabetology, Ospedale Israelitico, Rome, Italy

Department of Nuclear Medicine, Thyroid and PET/CT Centre, Oncology Institute of Southern Switzerland, Via Ospedale 12, Bellinzona and Lugano CH-6500, Switzerland

Prof. Dr. med. L. Giovanella, MD, PhD (✉)
Department of Nuclear Medicine, Thyroid and PET/CT Centre, Oncology Institute of Southern Switzerland, Via Ospedale 12, Bellinzona and Lugano CH-6500, Switzerland
e-mail: luca.giovanella@eoc.ch

8.1 Introduction

Thyroiditis is an inflammation of the thyroid gland that has several etiologies and can be associated with normal, elevated, or depressed thyroid function, often with evolution from one condition to another. Although there is considerable overlap, different subtypes of thyroiditis can be divided into those associated with thyroid pain and tenderness, and those that are painless.

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8.2 Painful Thyroiditis

8.2.1 Subacute Thyroiditis

Also named subacute granulomatous thyroiditis, giant cell thyroiditis, or de Quervain's thyroiditis, subacute thyroiditis is attributed to a viral infection being the most common cause of thyroid pain. It affects more women than men, and most often occurs at 40–50 years of age. Clinical presentation includes a tender, diffuse goiter and neck pain that often radiates up to the ear associated to myalgia, pharyngitis, low-grade fever, and fatigue [1]. C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are typically increased. Thyroid follicular cell damage leads to a transient unregulated release of large amounts of thyroid hormones into the circulation. This process usually is transient (i.e., 3–6 weeks), and patients usually return to euthyroidism within 3–12 months. However, in 10–15 % of patients, hypothyroidism persists, requiring long-term levothyroxine therapy [1]. Treatment for subacute granulomatous thyroiditis is firstly based on nonsteroidal anti-inflammatory drugs (NSAIDs). If no improvement occurs within 1 week, prednisone may be given in a dosage of 40–60 mg daily tapered to discontinuation over 4–6 weeks [1–3]. Beta-blockers may be given to treat symptoms of thyrotoxicosis.

8.2.2 Radiation-Induced Thyroiditis

Approximately 1 % of patients who have radioactive iodine for hyperthyroidism develop radiation thyroiditis between 5 and 10 days after the procedure. Clinical presentation resembles subacute thyroiditis. A brief course of NSAIDs or, rarely, prednisone in dosages of 40–60 mg per day may be used to alleviate pain; a beta-blocker often is required to block the peripheral effects of the thyroid hormone [1].

8.2.3 Suppurative Thyroiditis

This is an extremely rare form of thyroiditis caused by bacterial, fungal, or parasitic infection of the thyroid. Infection usually spreads to the thyroid from the adjacent structures directly or through the blood or lymphatic system or from a distant focus. Suppurative thyroiditis commonly present with acute unilateral anterior neck pain and erythema of the skin overlying an exquisitely tender thyroid. ESR is elevated, and the white blood cell count generally shows a marked increase with a left shift. Fine-needle aspiration of the lesion with Gram stain and culture is the most useful diagnostic test. Parenteral antibiotics should be given, and surgical drainage may also be required [1, 2].

8.3 Painless Thyroiditis

8.3.1 Autoimmune Thyroiditis

Also known as chronic lymphocytic thyroiditis or Hashimoto's thyroiditis, autoimmune thyroiditis (AIT) is a T-cell mediated autoimmune disorder in which thyroid-specific autoantibodies are produced [1, 2, 4]. This thyroid-specific pathway affects thyroid structure, and thyroid gland damage leads over time to an impaired gland function (i.e., hypothyroidism) [3]. The course of the disease varies, and there are no specific symptoms of AIT until hypothyroidism is achieved [5]. High circulating levels of antithyroid peroxidase antibodies (TPOAb) are a hallmark of AIT [1]. The indications for treatment of Hashimoto's disease with levothyroxine are goiter or clinical hypothyroidism [2].

8.3.2 Painless Sporadic Thyroiditis and Postpartum Thyroiditis

Painless sporadic thyroiditis (also known as subacute lymphocytic thyroiditis or silent sporadic thyroiditis) and postpartum thyroiditis are similar, but the former occurs in the absence of pregnancy. They appear to be autoimmune in origin as the thyroid contains a lymphocytic infiltrate and TPOAb are generally positive [3]. Most patients present (2–6 months after delivery in postpartum type) with a small, nontender goiter in the absence of pain. Hyperthyroidism is frequently asymptomatic and may be followed

by transient hypothyroidism in about 25 % of patients. Many patients finally develop permanent hypothyroidism 2–10 years after the first episode [1, 2].

8.3.3 Drug-Induced Thyroiditis

Amiodarone, interferon-alfa, interleukin-2, and lithium may cause a destructive thyroiditis with hyperthyroidism or hypothyroidism, low radioiodine uptake, and variable presence of TPOAb. Treatment is similar to that of subacute granulomatous or lymphocytic thyroiditis. The thyroid abnormalities usually resolve with discontinuation of the responsible drug.

8.3.4 Riedel's Thyroiditis

Riedel's thyroiditis (also known as fibrous thyroiditis) is a rare condition characterized by an extensive fibrotic process of unknown etiology involving the thyroid and adjacent structures. It may be associated with a diffuse fibrotic process affecting multiple tissues (idiopathic multifocal fibrosclerosis). Patients present with a rock-hard, fixed, painless goiter, often accompanied by symptoms of esophageal or tracheal compression [6].

8.4 Diagnosis and Follow-Up of Thyroiditis

Laboratory test (thyroid function, CRP, ESR, blood cell count), thyroid ultrasound (Figs. 8.1, 8.2, 8.3, 8.4, and 8.5), and thyroid scintigraphy (Figs. 8.6 and 8.7) are employed to differentiate thyroiditis providing the basis for a specific treatment. Overt or subclinical hyperthyroidism is generally found in painful and painless destructive thyroiditis, while thyroid function varies in autoimmune and Riedel's thyroiditis. Positive TPOAb are detected in autoimmune, painless, and postpartum thyroiditis. CRP and ESR are increased in subacute and suppurative thyroiditis. White blood cells are typically increased in suppurative thyroiditis. Ultrasound pattern is aspecific showing a diffusely reduced echogenicity and heterogeneity of thyroid echotexture with diffuse pseudonodular pattern. Moderate to increased thyroid vascularity frequently occurs even if a reduction in vascular signal is detected in subacute, painless, and drug-induced thyroiditis. Thyroid scan with ^{123}I or ^{99m}Tc-pertechnetate demonstrates homogeneously reduced uptake in destructive thyroiditis, while large variability is reported in AIT. Sometimes thyroiditis can be incidentally detected as diffuse fluorodeoxyglucose (FDG) uptake by positron emission tomography/computed tomography (Fig. 8.8).

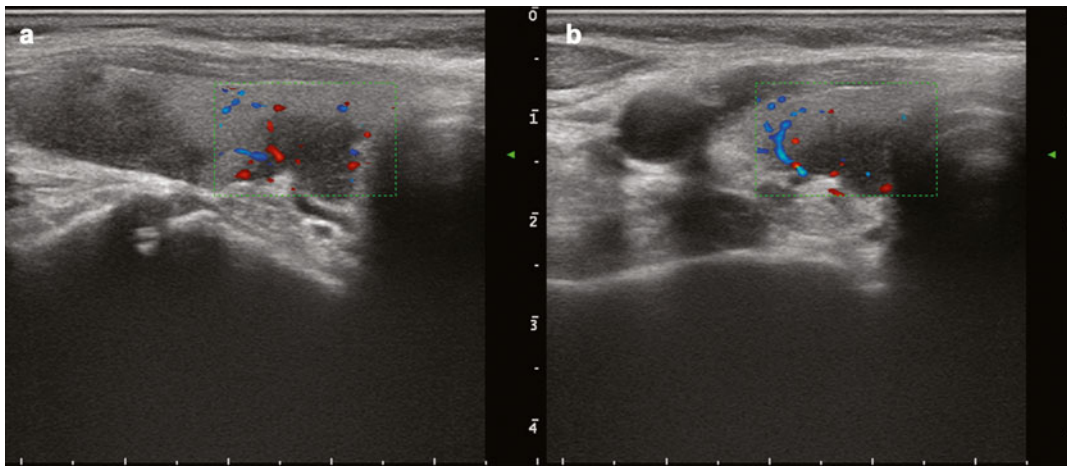

Fig. 8.1 Ultrasound presentation of a subacute de Quervain thyroiditis. Two hypoechoic areas with undefined margins and vascular spots are present at the superior and inferior thirds of the right thyroid lobe (**a**, longitudinal scan; **b**, transversal scan). Mild-moderate pain is detectable at physical examination and ultrasound evaluation

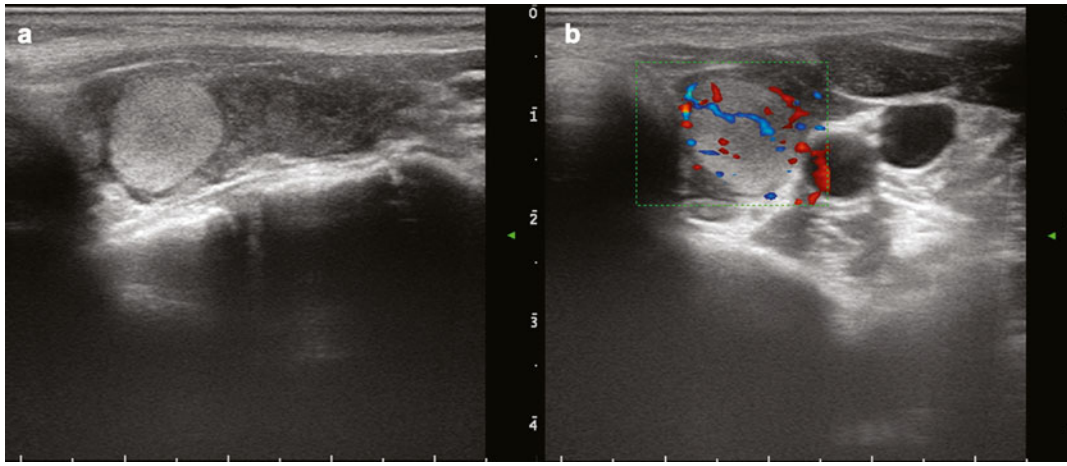

Fig. 8.2 Ultrasound presentation of a case of Hashimoto thyroiditis with a well defined hyperechoic area. The gland has an extended hypoechogenicity as a sign of tissue destruction by Hashimoto disease. At the base of left lobe there is a normoechoic (hyperechoic with respect to the surrounding tissue) nodular area (**a**) with normal vascularization (**b**) of about 1 cm. This area is not a "true" nodule but represents a part of the gland not involved by Hashimoto autoimmune pathways

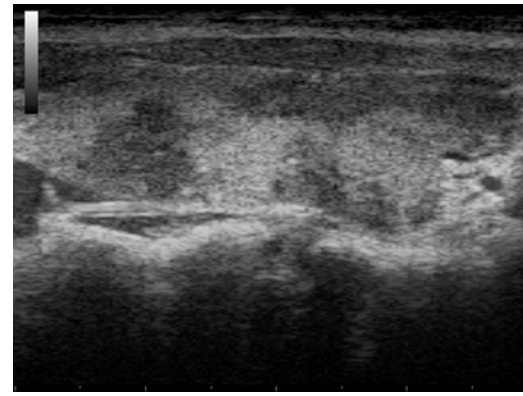

Fig. 8.3 Longitudinal ultrasonography scan of right thyroid lobe with Hashimoto's thyroiditis. The structure is inhomogeneously damaged, the majority of the tissue appears hypoechoic due to the damage by autoimmune pathway. Normoechoic areas represent the normal (normofunctioning) tissue

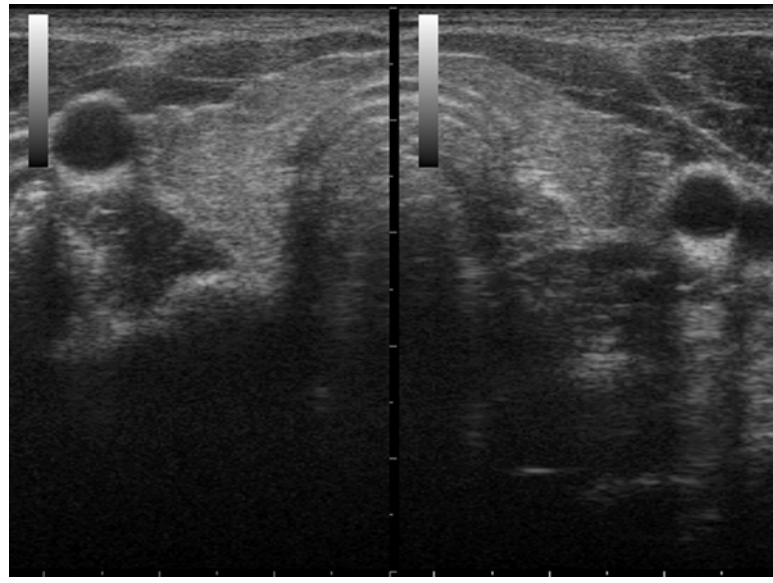

Fig. 8.4 Transverse ultrasonography scan of thyroid gland with thyroiditis. Thyroid volume is reduced, margins are irregular, echostructure is slightly inhomogeneous, echogenicity is poor. This thyroid US aspect frequently correlates with hypofunction (hypothyroidism)

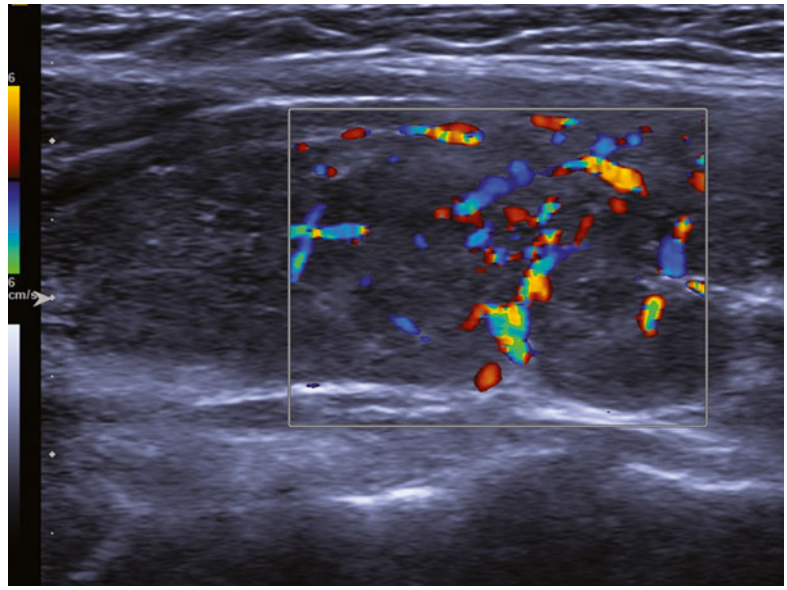

Fig. 8.5 Doppler US demonstrating moderate increased thyroid vascularity in a case of autoimmune thyroiditis

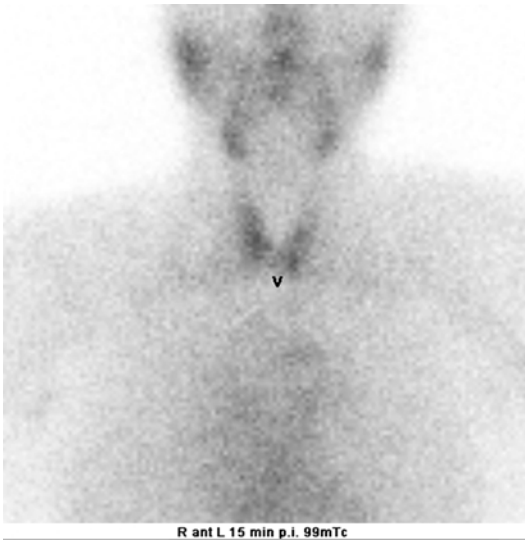

Fig. 8.6 Thyroid scintigraphy with ^{99m}Tc showing global reduced tracer uptake in a patient with autoimmune thyroiditis

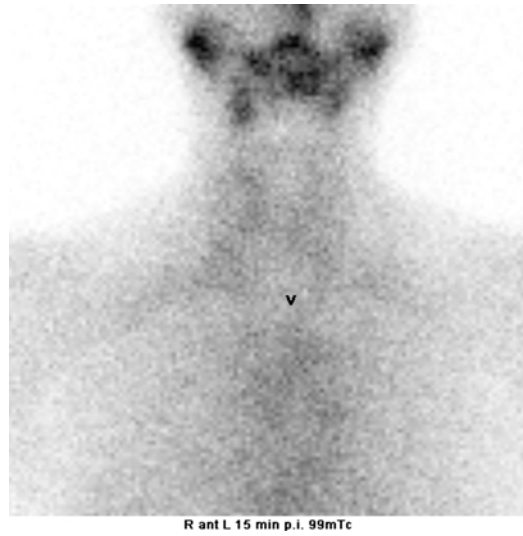

Fig. 8.7 Thyroid scintigraphy with ^{99m}Tc showing absent tracer uptake in a patient with subacute thyroiditis

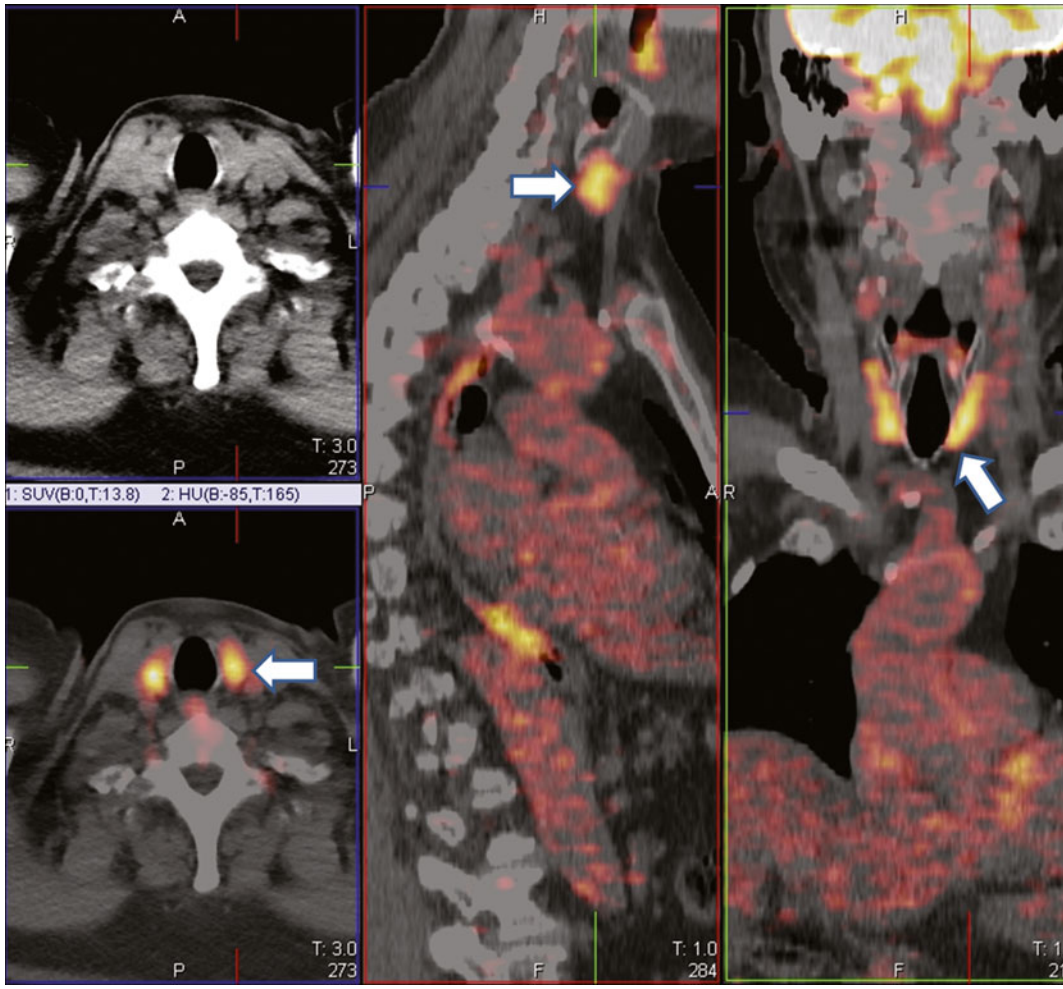

Fig. 8.8 Thyroiditis incidentally detected by fluorine-18 fluorodeoxyglucose positron emission tomography/computed tomography showing diffuse tracer uptake in the thyroid gland (*arrows*)

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