# 1 Hashimoto's Thyroiditis

· Authors

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#### **Editors**

In: <u>De Groot LJ, Chrousos G, Dungan K, Feingold KR, Grossman A, Hershman JM, Koch C, Korbonits M, McLachlan R, New M, Purnell J, Rebar R, Singer F, Vinik A, editors.</u>

Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. 2013 Dec 20.

#### **Excerpt**

Hashimoto's thyroiditis is characterized clinically as a commonly occurring, painless, diffuse enlargement of the thyroid gland occurring predominantly in middle-aged women. The patients are often euthyroid, but hypothyroidism may develop. The thyroid parenchyma is diffusely replaced by a lymphocytic infiltrate and fibrotic reaction; frequently, lymphoid germinal follicles are visible. Attention has been focused on this process because of the demonstration of autoimmune phenomena in most patients. Persons with Hashimoto's thyroiditis have serum antibodies reacting with TG, TPO, and against an unidentified protein present in colloid. In addition, many patients have cell mediated immunity directed against thyroid antigens, demonstrable by several techniques. Cell mediated immunity is also a feature of experimental thyroiditis induced in animals by injection of thyroid antigen with adjuvants. All theories also emphasize a basic abnormality in the immune surveillance system, which in some way allows autoimmunity to develop against thyroid antigens, and as well against other tissues, including stomach, adrenal, and ovaries, in many patients with thyroiditis. We suggest that Hashimoto's thyroiditis, primary myxedema, and Graves' disease are different expressions of a basically similar autoimmune process, and that the clinical appearance reflects the spectrum of the immune response in the particular patient. This response may include cytotoxic antibodies, stimulatory antibodies, blocking antibodies, or cell mediated immunity. Thyrotoxicosis is viewed as an expression of the effect of circulating thyroid stimulatory antibodies. Hashimoto's thyroiditis is predominantly the clinical expression of cell mediated immunity leading to destruction of thyroid cells, which in its severest form produces thyroid failure and idiopathic myxedema. Copyright © 2000-2017, MDText.com, Inc.

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PMID: 25905412

# <sup>1</sup> Acute and Subacute, and Riedel's Thyroiditis

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#### **Editors**

In: <u>De Groot LJ</u>, <u>Chrousos G</u>, <u>Dungan K</u>, <u>Feingold KR</u>, <u>Grossman A</u>, <u>Hershman JM</u>, <u>Koch C</u>, <u>Korbonits M</u>, <u>McLachlan R</u>, <u>New M</u>, <u>Purnell J</u>, <u>Rebar R</u>, <u>Singer F</u>, <u>Vinik A</u>, editors.

Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. 2015 Dec 8.

#### **Excerpt**

The thyroid, like any other structure, may be the seat of an acute or chronic suppurative or non-suppurative inflammation. Various systemic infiltrative disorders may leave their mark on the thyroid gland as well as elsewhere. Infectious thyroiditis is a rare condition, usually the result of bacterial invasion of the gland. Its signs are the classic ones of inflammation: heat, pain, redness, and swelling, and special ones conditioned by local relationships, such as dysphagia and a desire to keep the head flexed on the chest in order to relax the paratracheal muscles. The treatment is that for any febrile disease, including specific antibiotic drugs if the invading organism has been identified and its sensitivity to the drug established. Otherwise, a broad-spectrum antibiotic may be used. Surgical drainage may be necessary and a search for a pyriform sinus fistula should be made, particularly in children with thyroiditis involving the left lobe. Important to differentiate from the acute bacterial infection of acute suppurative thyroiditis (AST) is subacute (granulomatous) thyroiditis (SAT) which is far more common than AST and is characterized by a more protracted course, usually involving the thyroid symmetrically. The gland is also swollen and tender, and the systemic reaction may also be severe, with fever and an elevated erythrocyte sedimentation rate. During the acute phase of the disorder, tests of thyroid function disclose a suppression of TSH, increased serum concentrations of T4, T3, and thyroglobulin while a diminished thyroidal RAIU is observed. The cause of SAT has been established in only a few instances in which a viral infection has been the initiating factor. There may be repeated recurrences of diminishing severity. Usually, but not always, the function of the thyroid is normal after the disease has subsided. Subacute thyroiditis may be treated with rest, non-steroidal anti-inflammatory drugs or aspirin, and thyroid hormone. If the disease is severe and protracted, it is usually necessary to resort to administration of glucocorticoids, but recurrence may follow their withdrawal. It is precisely the observational nature of SAT therapy combined with the use of glucocorticoids which make it so critical to definitively rule out the bacterial etiology of AST in the patient presenting with a painful thyroid. Riedel's thyroiditis is a chronic sclerosing replacement of the gland that is exceedingly rare. The process extends to adjacent structures, making any surgical intervention very difficult and potentially harmful. The exact cause of Riedel's thyroiditis remains unknown, and no specific treatment is available beyond limited resection of the thyroid gland to relieve the symptoms of tracheal or esophageal compression. The use of anti-inflammatory medical treatments has been demonstrated to have significant benefits to outcome. Sarcoidosis may involve the thyroid, and amyloid may be deposited in the gland in quantities sufficient to cause goiter. In all of these diseases it may be necessary to give the patient levothyroxine replacement therapy if the function of the gland has been impaired.

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PMID: 25905408

## **Subacute Thyroiditis**

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In: <u>De Groot LJ, Chrousos G, Dungan K, Feingold KR, Grossman A, Hershman JM, Koch C, Korbonits M, McLachlan R, New M, Purnell J, Rebar R, Singer F, Vinik A, editors.</u>

Endotext [Internet]. South Dartmouth (MA): <a href="MDText.com">MDText.com</a>, Inc.; 2000-. 2015 Apr 12.

### Excerpt

Subacute thyroiditis (SAT) is an inflammatory condition of the thyroid with characteristic presentations and clinical course. Patients with the classic. painful (DeQuervain's: Granulomatous) thyroiditis, (PFSAT) typically present with painful swelling of the thyroid. Transient vocal cord paresis may occur. At times, the pain begins and may be confined to the one lobe, but usually spreads rapidly to involve the rest of the gland ("creeping thyroiditis"). Pain may radiate to the jaw or the ears. Malaise, fatigue, myalgia and arthralgia are common. A mild to moderate fever is expected, and at times a high fever of 104°F (40.0°C) may occur. The disease process may reach its peak within 3 to 4 days and subside and disappear within a week, but more typically, onset extends over 1 to 2 weeks and continues with fluctuating intensity for 3 to 6 weeks. The thyroid gland is typically enlarged, smooth, firm and tender to palpation, sometimes exquisitely so. Approximately one-half of the patients present during the first weeks of the illness, with symptoms of thyrotoxicosis. Subsequently patients often experience hypothyroidism before returning to normal(see figure 1). This painful condition lasts for a week to a few months, usually demonstrates a very high erythrocyte sedimentation rate (ESR), elevated C- reactive protein (CRP) levels and has a tendency to recur. Copyright © 2000-2017, MDText.com, Inc.

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PMID: 25905310