

Chronic lymphocytic thyroiditis (Hashimoto thyroiditis).

by Lester D. R. Thompson, MD

Chronic lymphocytic thyroiditis, eponymically referred to as *Hashimoto thyroiditis* (named after Hakarum Hashimoto, who first described the disease in 1912), is an autoimmune thyroiditis that results from autoantibodies to thyroid-specific antigens. There is a poorly understood interaction between susceptibility genes and environmental triggers, resulting in antimicrosomal and antithyroglobulin antibodies. Many patients may have another simultaneous autoimmune disease (such as Sjögren syndrome, diabetes mellitus, or myasthenia gravis), while there is some overlap with Graves disease (diffuse hyperplasia). Hypothyroidism may also result from destruction of the thyroid epithelium by inflammatory cells.

Approximately 5% of the general population has hypothyroidism, with Hashimoto thyroiditis the most common cause. The disease develops over a wide age range, increasing in frequency with advanced age. Women are affected disproportionately, with a female-to-male ratio of 10:1, although women are not affected as disproportionately when the fibrous variant is present. Patients come to clinical attention with symptoms of hypothyroidism, laboratory evidence of decreased thyroid function, identification of thyroid antibodies, or thyroid gland enlargement, most often bilateral.

Laboratory findings include decreased thyroxine (T4), possible decreased triiodothyronine (T3), and circulating antibodies to a variety of thyroid antigens (thyroglobulin, thyroid peroxidase [microsomal antigen], colloid antigen, and thyroid hormones).

Appropriate management requires lifelong thyroid hormone replacement therapy, resulting in decreased thyroid antibody levels, although complications of therapy may be seen. Surgery is employed in patients unresponsive to medical therapy. Patients with Hashimoto thyroiditis have a significantly increased risk of developing lymphoma, so long-term follow-up is required.

Macroscopically, there is symmetrical thyroid gland enlargement with a firm consistency, showing bulging lobules above the cut surface separated by fibrous tissue. The thyroid gland is usually not adherent to surrounding structures. Histologically, the whole gland is affected, although it may be unevenly involved. There is an infiltrate of chronic inflammatory cells, often forming germinal centers (figure 1). The immediately adjacent thyroid follicular epithelium shows oncocytic cytoplasm with nuclear enlargement and open nuclear chromatin. Nucleoli are often prominent (figure 2). Islands of squamous metaplasia may be seen in the background, along with isolated lymphoepithelial cysts.

Figure 1. An intermediate power demonstrates several germinal centers surrounded by lymphocytes and plasma cells. The thyroid follicular epithelium has oncocytic (oxyphilic) cytoplasm.

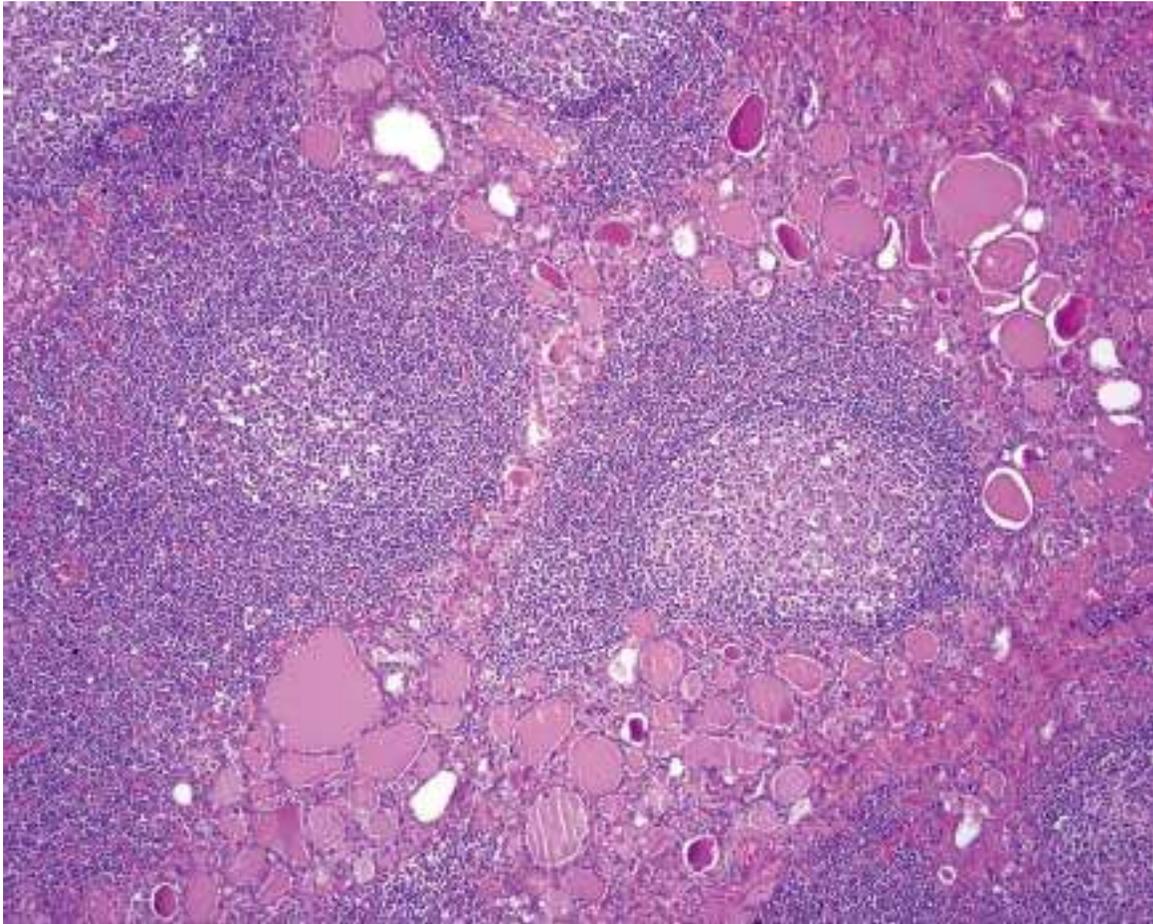
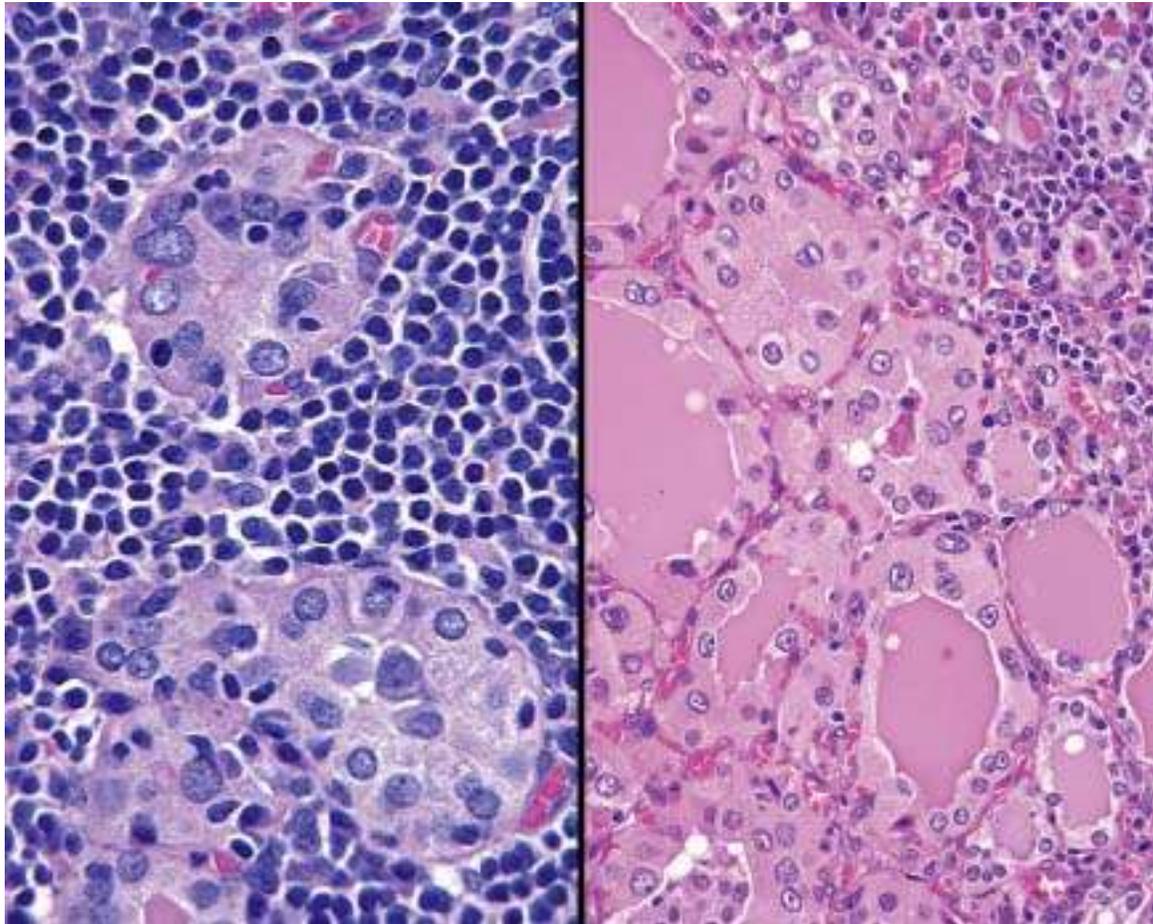


Figure 2. Left: Two thyroid epithelial follicles lack any colloid; they are completely surrounded by lymphocytes and they have granular cytoplasm. **Right:** There is an oncocytic appearance to the cytoplasm of the follicular epithelium. The nuclei are enlarged, with vesicular chromatin and prominent but small nucleoli. An inflammatory infiltrate is noted in the upper right.



With time, a dense fibrosis will be deposited (in extreme, referred to as the *fibrosing variant*), which results in increased nodularity of the gland. This fibrosis does not extend into the perithyroidal tissue and is not associated with vasculitis. Although unnecessary for the diagnosis, the lymphoid population will show appropriate compartmentalization of B- and T-cell markers without light chain (κ and λ) restriction. It is important to exclude fibrous thyroiditis (Riedel disease) and lymphoma, especially as there is an increased risk of developing lymphoma with increased disease duration. It is possible to have a concurrent follicular neoplasm (papillary or follicular carcinoma), although there is no change in prognosis or outcome for a carcinoma in the setting of concurrent chronic lymphocytic thyroiditis.

Suggested reading

1. Caturegli P, Kimura H, Rocchi P, Rose NR. Autoimmune thyroid diseases. *Curr Opin Rheumatol* 2007;19 (1): 44-8.
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3. Shih ML, Lee JA, Hsieh CB, et al. Thyroidectomy for Hashimoto's thyroiditis: Complications and associated cancers. *Thyroid*. 2008;18 (7): 729-34.

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