Graves' Disease after Unilateral Riedel's Thyroiditis

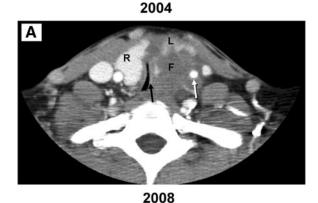
Bryan McIver, Mitra M. Fatourechi, Ian D. Hay, and Vahab Fatourechi

Division of Endocrinology, Diabetes, Metabolism, and Nutrition (B.M., I.D.H., V.F.) and Department of Internal Medicine (M.M.F.), Mayo Clinic, Rochester, Minnesota 55905

Riedel's thyroiditis, also termed "invasive fibrous thyroiditis," is the rarest form of autoimmune thyroiditis and can be associated with systemic fibrotic processes, Hashimoto thyroiditis (1, 2), and Graves' disease (3). A case of hyperthyroidism followed by spontaneous hypothyroidism and Riedel's thyroiditis has been reported (4). Two previously reported patients from our institution had invasive fibrous thyroiditis that evolved from antecedent Graves' disease, as indicated by thyroid dysfunction, bilateral ophthalmopathy, and positive TSH receptor-stimulating autoantibodies (5). We present a new case.

In 2004, a 26-yr-old woman presented with a mass in the left thyroid lobe, pain, and respiratory distress. Thyroid function was normal. A tracheostomy and biopsy showed Riedel's thyroiditis. Therapy with corticosteroids and tamoxifen stabilized the disease. After 1 yr, the tracheostomy was closed. In 2008, her TSH level was less than 0.01 mIU/liter, her free T₄ was 4.5 ng/dl (58 pmol/liter; normal, 0.8-1.8 ng/dl or 10-23 pmol/ liter), and her serum was strongly positive for TSH receptor antibodies (52%; normal, <16%). The diagnosis of Graves' hyperthyroidism was made. Her 24-h iodine-131 uptake was 33% (upper limit of normal, 29%). She was treated with 19.7 mCi iodine-131. Computed tomography of the neck, performed in 2004 and 2008, showed left lobe thyroid enlargement and compression of the trachea (Fig. 1). Ultrasonography of the thyroid in 2008 (Fig. 2) showed hypervascularity in the areas not affected by Riedel's thyroiditis; the absence of vascularity in the hypoechoic areas of the left lobe corresponded to Riedel's involvement, as shown by computed tomography.

ISSN Print 0021-972X ISSN Online 1945-7197
Printed in U.S.A.
Copyright © 2010 by The Endocrine Society
doi: 10.1210/jc.2009-2609 Received December 8, 2009. Accepted April 16, 2010.



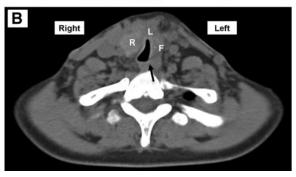


FIG. 1. Computed tomographic images of the neck. A, Image taken with contrast (2004). Note partial involvement of the left lobe with fibrosis (F), extrathyroidal extension, and significant tracheal narrowing (*black arrow*) and engulfment of the common carotid artery (*white arrow*). Also note the uninvolved part of left lobe (L). The right lobe of the thyroid (R) is completely uninvolved. B, Image taken without contrast (2008). Note the stability and partial resolution of the process in the left lobe. (F denotes partially resolved left lobe fibrosis; L, uninvolved part of the left thyroid lobe; R, right lobe. The *black arrow* shows improved tracheal narrowing.)

The present case is of interest because Graves' disease developed after 3 yr of stability of Riedel's thyroiditis. In this case, the fibrotic process with extra thyroidal extension was unilateral. Graves' hyperthyroidism developed in the uninvolved areas of the thyroid. This case and similar reported cases support the notion of autoimmune

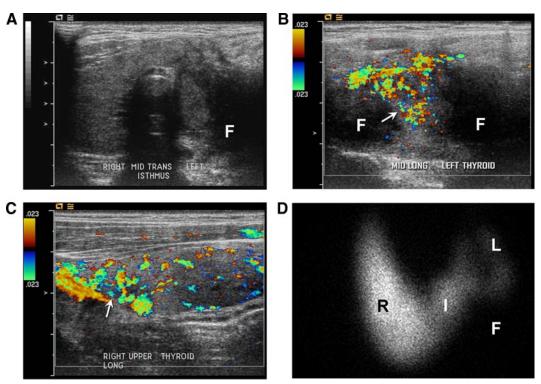


FIG. 2. Ultrasound of thyroid and isotopic scan of the thyroid (2008). A, Hypoechoic area in the left lobe corresponds to fibrotic involvement (F). B, Longitudinal sonographic image of the left lobe shows hypervascularity (by color Doppler) in the left thyroid lobe (arrow) in areas not involved by fibrotic process and hypoechoic avascular fibrotic areas (F). C, Longitudinal view of the right lobe shows diffuse hypervascularity of the right lobe (arrow), typical of Graves' hyperthyroidism. D, Iodine-131 thyroid isotopic imaging with diffuse uptake in the right lobe and similar uptake in the areas of the left lobe not involved by Riedel's thyroiditis. F denotes fibrotic Riedel area (in the left lobe); I, isthmus; L, left lobe; R, right lobe.

mechanisms having a role in the pathogenesis of Riedel's thyroiditis.

Acknowledgments

Address all correspondence and requests for reprints to: Vahab Fatourechi, M.D., Division of Endocrinology, Diabetes, Metabolism, and Nutrition, Mayo Clinic, 200 First Street SW, Rochester, Minnesota 55905. E-mail: fatourechi.vahab@ mayo.edu.

Current address for M.M.F.: Department of Medicine, University of California Irvine, Irvine, California.

Disclosure Summary: The authors have no conflicts of interest to disclose.

References

- 1. Baloch ZW, Saberi M, Livolsi VA 1998 Simultaneous involvement of thyroid by Riedel's [correction of Reidel's] disease and fibrosing Hashimoto's thyroiditis: a case report. Thyroid [Erratum (1998) 8:736] 8:337-341
- 2. Ross DS, Daniels GH 1992 Riedel's thyroiditis associated with Hashimoto's thyroiditis. J Endocrinol Invest 15:479
- 3. Armigliato M, Paolini R, Bianchini E, Monesi G, Zamboni S, D'Andrea E 2002 Hashimoto's thyroiditis and Graves' disease associated with retroperitoneal fibrosis. Thyroid 12:829-831
- 4. Yasmeen T, Khan S, Patel SG, Reeves WA, Gonsch FA, de Bustros A, Kaplan EL 2002 Clinical case seminar: Riedel's thyroiditis: report of a case complicated by spontaneous hypoparathyroidism, recurrent laryngeal nerve injury, and Horner's syndrome. J Clin Endocrinol Metab 87:3543-3547
- 5. Heufelder AE, Hay ID 1994 Evidence for autoimmune mechanisms in the evolution of invasive fibrous thyroiditis (Riedel's struma). Clin Investig 72:788-793