Letter to the Editor

Acute Exacerbation of Chronic Thyroiditis with Painless Thyroid

To the Editor;

Destruction-induced thyrotoxicosis includes various painful diseases such as subacute granulomatous thyroiditis, acute exacerbation of chronic thyroiditis and acute supplicative thyroiditis. In contrast, the clinically painless form of destruction-induced thyrotoxicosis is almost exclusively restricted to painless or silent thyroiditis. We report what we believe to be a case of acute exacerbation of chronic thyroiditis with painless thyroid.

A 50-year old woman first noticed her goiter 5 years ago and was hospitalized for high fever (~40°C). On admission, extremely hard goiter without tenderness was noted. Erythrocyte sedimentation rate and C-reactive protein (CRP) were 156 mm/h and 31.6 mg/dl (normal range <0.6 mg/dl), respectively. Thyroid function tests showed an increased FT$_3$ (5.97 pg/ml; normal range 2.1–4.2) and FT$_4$ levels (3.30 ng/dl; normal range 1.0–1.8), and TSH level was suppressed to 0.092 g10$^9$ IU/ml (normal range: 0.47–4.33). Both anti-thyroid peroxidase (anti-TPO) antibody (116 U/ml; normal range <0.3) and anti-thyroglobulin antibody levels (762 U/ml; normal range <0.3) were extremely high but TSH receptor antibody was negative (6.8%; normal range <15%). Ultrasonography revealed nodular configuration of the thyroid with heterogeneous internal texture and no ill-defined hypoechoic area. Scintigraphy with technecium-99 m revealed low uptake in the thyroid bed. From the first hospital day, 30 mg of prednisolone was administered orally. On day 5 after admission, her fever subsided to normal range and the thyroid gland was markedly softened and reduced. On day 19 after admission CRP was 0.4 mg/dl. One month after admission, FT$_3$, FT$_4$ and TSH were 2.24 pg/ml, 0.94 ng/dl and 2.72 µIU/ml, respectively. Anti-TPO antibody was still over 50 U/ml; however anti-thyroglobulin antibody levels were significantly decreased to 43.8 U/ml.

Acute exacerbation of chronic thyroiditis is generally characterized by abrupt swelling and tenderness in the thyroid associated with systemic inflammatory reactions including fever, increased CRP, and high erythrocyte sedimentation rate [1]. The clinical and laboratory findings in these patients are most compatible with subacute thyroiditis (SAT). However, Shigemasa et al. reported that antithyroglobulin (TGHA) and antimicrosome (MCHA) antibodies were extremely high in the patients with painful chronic thyroiditis, whereas TGHA was absent in all and MCHA was present in low titer in only 1 of the 11 SAT patients in their study [2]. The immunological examinations as well as ultrasonographical study revealed that the present case was not likely SAT. To our knowledge, only one case of acute exacerbation of chronic thyroiditis has ever been reported to be nontender thyroid [3]. In that case, the patient’s anti-TPO antibody (0.7–1.5 U/ml; normal range <0.3) and anti-thyroglobulin antibody levels (35–64%; normal range <10) remained largely unchanged throughout the clinical course. Our patient showed abrupt swelling of thyroid (within 3 days) and extremely high titer of anti-TPO antibody and anti-thyroglobulin antibody levels which were decreased rapidly after improvement of inflammation using steroid hormone, suggesting that acute exacerbation of chronic thyroiditis was the most probable diagnosis for this case although the cytological examination for definite diagnosis could not be performed because of the patient’s refusal. Zimmerman et al. reported that the degree of thyroidal enlargement did not necessary correlate with the degree of tenderness [4]. In chronic thyroiditis, various histological forms are reported [2, 5] and our case could possibly be one of the variant forms of chronic thyroiditis with tender thyroid. In summary, in such a cases, careful observation including anti-thyroglobulin antibody levels is necessary to avoid misdiagnosis as infection or other thyroid diseases, particularly SAT, when thyroid biopsy is not available.

References

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