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Case Report

Subacute thyroiditis presenting with thyrotoxicosis and a non-suppressed TSH: a two-case report

HN Sarker[1*], Anower Hosen[2]

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*Corresponding author

SUMMERY

Subacute thyroiditis (SAT), or de Quervain thyroiditis, is a self-limiting inflammatory disorder of the thyroid that typically presents with anterior neck pain, thyroid tenderness, systemic inflammatory features, and transient thyrotoxicosis. The thyrotoxic phase usually results from destructive release of preformed thyroid hormones rather than increased thyroid hormone synthesis. A suppressed serum thyroid-stimulating hormone (TSH) concentration is therefore expected in most patients during the thyrotoxic phase.We report two patients with clinically typical painful subacute thyroiditis who presented with biochemical elevation of free thyroxine (FT4) but a reportedly normal TSH concentration. Both patients had prominent adrenergic symptoms, unilateral thyroid tenderness, raised erythrocyte sedimentation rate (ESR), and sinus tachycardia. They were treated with propranolol and prednisolone, with marked clinical improvement and subsequent biochemical recovery. These cases highlight the importance of integrating the clinical phenotype with thyroid-function testing and considering assay-related, physiological, and analytical explanations when the TSH concentration appears discordant with the FT4 concentration.

Keywords: *Subacute thyroiditis, thyrotoxicosis, non-suppressed TSH*

1. *Professor (ex), Medicine, Sher-e-Bangla Medical College, Barishal, Bangladesh (ORCID: 0000-0001-6523-9395)*
2. *Principal, Professor and HOD, Medicine, Sher-e-Bangla Medical College, Barishal, Bangladesh*

INTRODUCTION

Subacute thyroiditis is the most common painful inflammatory disorder of the thyroid. It is generally self-limiting and is characterised by thyroid pain and tenderness, systemic inflammation, and a transient thyrotoxic phase caused by destruction of thyroid follicles and release of preformed thyroid hormones into the circulation [1,2]. The classical laboratory pattern during the thyrotoxic phase is an increased serum FT4 and/or FT3 concentration accompanied by suppression of TSH, often with a markedly raised ESR or C-reactive protein concentration [1,3].

The clinical course generally progresses through a thyrotoxic phase, followed by a euthyroid phase and, in some patients, a transient hypothyroid phase before recovery [1,4]. Because the thyroid gland is releasing stored hormone rather than actively synthesising excessive hormone, radioactive iodine uptake is characteristically low during the thyrotoxic phase [1,5].

We report two patients with a characteristic clinical presentation of subacute thyroiditis but an apparently non-suppressed TSH despite an increased FT4 concentration. The cases illustrate an important diagnostic dilemma in interpreting discordant thyroid-function tests.

Case 1

A 45-year-old man presented with a 10-day history of excessive sweating, palpitation, restlessness and mild pain on the right side of his neck. He had no documented previous thyroid disease. On examination, he appeared anxious, with a pulse rate of 110 beats per min and blood pressure of 135/80 mm Hg. Fine tremor of the hands was present. The right lobe of the thyroid was palpable and distinctly tender.

His complete blood count was normal, but the ESR was markedly increased at 90 mm in the first hour. Electrocardiography showed sinus tachycardia. Thyroid-function testing showed an increased FT4 concentration, whereas the reported TSH concentration was within the laboratory reference range.

The combination of recent-onset thyrotoxic symptoms, unilateral thyroid tenderness, markedly increased ESR and an elevated FT4 concentration led to a clinical diagnosis of subacute thyroiditis. He was treated with propranolol for symptomatic control and prednisolone for the inflammatory thyroid disease. At review 10 days later, his palpitation, sweating and restlessness had substantially improved, and the thyroid tenderness had resolved clinically.

Case 2

A 30-year-old woman presented with a 5-day history of sweating and palpitation associated with pain on the left side of the neck, particularly during swallowing. She had no significant abnormality on general examination apart from tachycardia. Her pulse rate was 120 beats per min and blood pressure was 140/70 mm Hg. Fine tremor of the hands was present, and the left thyroid lobe was palpable and tender.

Her complete blood count was normal, while the ESR was 60 mm in the first hour. Electrocardiography demonstrated sinus tachycardia. FT4 was increased, although the TSH concentration was reported as normal.

A diagnosis of subacute thyroiditis was made on the basis of the characteristic painful thyroid enlargement, inflammatory response and transient thyrotoxic presentation. She received propranolol and prednisolone. At 14-day follow-up, she had substantial clinical improvement, with resolution of the palpitation and sweating and improvement in the thyroid tenderness. Follow-up thyroid-function testing showed biochemical improvement.

DISCUSSION

These two cases share several characteristic features of subacute thyroiditis: acute or subacute onset of anterior neck pain, unilateral thyroid tenderness, symptoms of thyrotoxicosis, sinus tachycardia and a substantially increased ESR. The combination is clinically highly suggestive of painful subacute thyroiditis [1,2].

The pathophysiology of SAT differs fundamentally from that of Graves' disease. In SAT, inflammatory destruction of thyroid follicles causes leakage of preformed T4 and T3 into the circulation. Consequently, the initial thyrotoxic phase represents **destructive thyrotoxicosis**

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rather than increased thyroid hormone synthesis [1,3]. This distinction has practical therapeutic implications because antithyroid drugs such as carbimazole or methimazole generally have little role in the thyrotoxic phase of SAT.

A markedly elevated ESR is one of the characteristic laboratory abnormalities in painful subacute thyroiditis. ESR is frequently greater than 50 mm/h and may exceed 100 mm/h in severe disease [1,6]. Both patients in this report had substantial elevation of ESR—90 mm/h in the first patient and 60 mm/h in the second—supporting the diagnosis in the appropriate clinical context.

The apparent discordance between the FT4 and TSH results in these cases deserves particular attention. In the classical thyrotoxic phase of SAT, TSH is expected to be suppressed because circulating thyroid hormone exerts negative feedback on the hypothalamic-pituitary axis [1,5]. Previous physiological studies have demonstrated simultaneous elevation of T4/T3 and suppression of TSH during the acute phase of subacute thyroiditis [7]. Thus, a genuinely normal TSH in the presence of raised FT4 is atypical and should not simply be regarded as a characteristic laboratory feature of SAT.

Several possibilities should therefore be considered when this pattern is encountered. First, the timing of testing might have influenced the results. TSH does not change instantaneously with alterations in circulating thyroid hormone concentrations, and the degree of suppression may vary according to the phase and duration of illness. Second, analytical interference can produce apparently discordant thyroid-function results. Biotin use, heterophile antibodies and other assay-related factors can occasionally interfere with immunoassays and should be considered when biochemical results do not fit the clinical picture. Third, the FT4 result should ideally be confirmed using an alternative analytical platform or an appropriate reference method when the discrepancy is substantial or persistent.

The differential diagnosis of thyrotoxicosis with a non-suppressed TSH is broader than SAT alone. Persistent elevation or inappropriate normality of TSH despite elevated FT4 should prompt consideration of conditions such as assay interference, resistance to thyroid hormone, or, much less commonly, a TSH-secreting pituitary adenoma. However, the presence of a painful and tender thyroid together with a markedly elevated ESR and spontaneous improvement is strongly supportive of an inflammatory destructive process.

Thyroid ultrasonography with colour Doppler and, where clinically appropriate, measurement of radioactive iodine uptake can provide additional diagnostic information. SAT typically produces heterogeneous hypoechoic areas with reduced vascularity on Doppler examination, while radioactive iodine uptake is characteristically low during the thyrotoxic phase [1,5,6]. Measurement of TSH-receptor antibodies may also help distinguish SAT from Graves' disease when the diagnosis is uncertain.

Treatment in SAT is primarily directed towards controlling pain, inflammation and symptoms of thyrotoxicosis. β-blockers such as propranolol can provide effective relief from palpitation, tremor and other adrenergic symptoms [1]. NSAIDs are generally appropriate for mild disease, whereas corticosteroids are commonly used when symptoms are moderate to severe or inadequately controlled with NSAIDs [1,2]. Evidence from comparative studies supports the effectiveness of glucocorticoids in achieving rapid symptomatic relief in appropriately selected patients [2,8].

The rapid clinical improvement observed in both patients following prednisolone and propranolol is compatible with the expected response in SAT. Nevertheless, corticosteroid therapy should generally be tapered according to clinical response rather than stopped abruptly after a very short course, because recurrence can occur during or after treatment [2].

An important limitation of these cases is the absence of several potentially useful investigations, including baseline and follow-up T3, repeat TSH measurements, CRP, thyroid autoantibodies, thyroid ultrasonography with Doppler, and radioactive iodine uptake. Therefore, the diagnosis rests principally on the clinical phenotype, inflammatory markers, thyroid-function abnormalities and subsequent clinical course.

CONCLUSION

These two cases illustrate a clinically typical presentation of subacute thyroiditis with an unusual thyroid-function pattern: raised FT4 with a reportedly non-suppressed TSH. Painful thyroid enlargement, marked elevation of ESR and transient thyrotoxic symptoms remain important clinical clues to SAT. However, a non-suppressed TSH should prompt confirmation of the thyroid-function results and consideration of assay interference and other causes of discordant thyroid-function tests rather than being automatically attributed to subacute thyroiditis. Recognition of destructive thyrotoxicosis is important because treatment is primarily supportive, with β-blockade for adrenergic symptoms and anti-inflammatory therapy for thyroid pain and inflammation.

Contributors

The clinical cases were evaluated, interpreted and managed by the treating clinical team. The manuscript was prepared from the clinical information provided and the relevant published literature.

Declaration of interests

No competing interests were declared.

REFERENCES

1. Pearce EN, Farwell AP, Braverman LE. Thyroiditis. **N Engl J Med** 2003;349:1286–1295.
2. Ray I, D'Souza B, Sarker P, et al. Management of subacute thyroiditis: a systematic review of current treatment protocols. **Int J Gen Med** 2022;15:6425–6439.
3. Hennessey JV. Subacute thyroiditis. **Endotext**. South Dartmouth, MA: MDText.com. The thyrotoxic phase is characterised by release of preformed thyroid hormone and typically increased T4/T3 with suppressed TSH.
4. Pearce EN, Bogazzi F, Martino E, et al. The natural history of subacute thyroiditis. **J Clin Endocrinol Metab** 2006;91:2100–2105.
5. Bahn Chair RS, Burch HB, Cooper DS, et al. Hyperthyroidism and other causes of thyrotoxicosis: management guidelines of the American Thyroid Association and American Association of Clinical Endocrinologists. **Thyroid** 2011;21:593–646.
6. Stasiak M, Lewiński A. New aspects in the pathogenesis and management of subacute thyroiditis. **Front Endocrinol (Lausanne)** 2021;12:646970. The characteristic inflammatory response and low thyroid blood flow during the thyrotoxic phase are described in this review.
7. Larsen PR, Ingbar SH. The thyroid gland. In: Williams Textbook of Endocrinology. Philadelphia: Elsevier.
8. Yuan A, Wu J, et al. Comparison of treatment outcome between glucocorticoids and non-steroidal anti-inflammatory drugs in subacute thyroiditis patients: a systematic review and meta-analysis. **BMC Endocr Disord** 2024.