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CASE REPORT

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Acute arthritis revealing Hashimoto's thyroiditis

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Abstract

Rheumatic manifestations can reveal hypothyroidism, such as arthritis and non-specific musculoskeletal symptoms. We report herein the case of an acute polyarthritis revealing Hashimoto's thyroiditis (HT). Hormone replacement therapy leads to the resolution of arthritis related to HT, suggesting the role of thyroid hormone in the pathogenesis of arthritis.

KEYWORDS

arthritis, autoimmune thyroiditis, Hashimoto disease, thyroiditis

1 | INTRODUCTION

Hashimoto's thyroiditis (HT) is autoimmune thyroid disease and one of the common causes of primary hypothyroidism.[1] It can be responsible or associated with several rheumatic manifestations.[2]

Rheumatic manifestations, such as arthritis and non-specific musculoskeletal symptoms, can reveal hypothyroidism. These manifestations can be related to thyroid dysfunction, connective tissue diseases associated with HT, or thyroid autoimmunity.[3]

We report the case of an acute polyarthritis revealing primary hypothyroidism caused by HT.

We emphasize clinical, biological, and management of this condition.

2 | CASE PRESENTATION

A 34-year-old woman with no medical history presented to our department with a 5-day history of fatigue and inflammatory joint pain affecting the small joints of the left hand, wrist, right elbow, and ankle. She had myalgia without sicca symptoms. The patient had no family history of thyroid, rheumatic, or autoimmune disease. No recent history of viral infection or childbirth was reported.

Physical examination showed a normal blood pressure of 110/60 mmHg, synovitis of the left wrist and metacarpophalangeal joints, right elbow, and ankle. No goiter was found.

Laboratory findings revealed a high C-reactive protein level (28 mg/L, Normal value [N] < 6), normocytic anemia (hemoglobin: 7.7 g/dl, N ≥ 12 g/dl, mean corpuscular volume: 96.5 femtoliter, N: 80–100), high Lactate dehydrogenase (LDH) level (280 IU/L, N: 91–260), and high Creatine-phosphokinase (CPK) level (485 IU/L, N: 22–269). Liver and renal tests were within the normal range.

Anti-nuclear antibodies (ANA), anti-SSA, SSB, rheumatoid factor, and anti-citrullinated protein antibodies (ACPA) were negative.

C3 and C4 complement levels were normal. Thyroxine (T4) level was low (2.8 pmol/L, N: 7.9–14.4), and thyroid-stimulating hormone (TSH) level was 7 times the upper limits of normal (39 μIU/L, N: 0.34–5.6). The thyroid peroxidase antibodies were positive (670 IU/ml, N: <35 IU/ml), whereas anti-thyroglobulin antibodies were negative.

Radiographs of hands, feet, and pelvis were without abnormalities. Musculoskeletal ultrasound showed synovial

thickening of the left wrist and the right elbow with hyperemia in power Doppler imaging. There was also synovial thickening of the left metacarpophalangeal joints, right ankle, and tenosynovitis of the posterior tibial and fibular tendons. The chest radiograph was normal. Thyroid ultrasound showed inhomogeneous and hypoechogenic thyroid parenchyma with a pseudo-nodular appearance without starry sky aspect nor giraffe sign. However, ultrasonography of the salivary glands was without abnormalities.

The diagnosis of primary hypothyroidism related to Hashimoto thyroiditis was made. There were not enough criteria to make the diagnosis of Sjogren's syndrome.

A thyroid hormone replacement therapy was started at 25 μg daily and increased progressively to reach 150 μg daily.

After 3 months of treatment, the TSH level had become within the normal range, and polyarthritis had disappeared. The TSH level remained stable, and the patient did not develop a recurrence of the polyarthritis after 20 months of follow-up.

Consent from the patient for publication of this case study was obtained.

3 | DISCUSSION

We report a case of acute polyarthritis revealing HT. HT is the most frequent auto-immune endocrine disorder[4] and the most common etiology of hypothyroidism.[1] It results from a diffuse lymphocytic infiltration of the thyroid.

The diagnosis of HT relies on clinical features which vary from subclinical hypothyroidism to typical myxedema, positivity of serum thyroid antibodies (mainly thyroperoxidase [90%] and thyroglobulin), and reduced echogenicity on thyroid ultrasonography.[5]

HT can be responsible for various rheumatic manifestations due to thyroid dysfunction. These manifestations often reveal this disease.

It is also closely associated with several autoimmune diseases, which can be themselves responsible for articular and muscular manifestations, such as Sjögren's syndrome, rheumatoid arthritis, systemic lupus erythematosus, or scleroderma.[6–9] Therefore, it is necessary to rule out these diagnoses before attributing articular manifestations to HT.

In our case, apart from arthritis, there were no clinical nor immunological features for the diagnosis of systemic lupus erythematosus, Sjögren's syndrome, or rheumatoid arthritis. Moreover, thyroid hormone replacement therapy led to a complete resolution of polyarthritis.

Musculoskeletal symptoms related to hypothyroidism[10] occur in 25 to 79%of cases. Muscular signs may include pain, cramps, and weakness. The muscle enzyme levels (CK, myoglobin, and lactate dehydrogenase)[10] can be increased. The CK levels correlated with TSH levels,[11,12] and hypothyroid myopathy symptoms usually resolve after thyroxin supplementation.[11,13]

Polyarthralgia is the most frequent rheumatic symptom occurring during hypothyroidism.[3] The classic clinical presentation of hypothyroid arthropathy is less frequent; it is characterized by synovial thickening and joint effusions typically affecting the knees, metacarpophalangeal, proximal interphalangeal, and metatarsophalangeal joints. Synovial fluid can be non-inflammatory with highly increased viscosity.[14–17]

Other rheumatic manifestations were also reported in patients with hypothyroidism, such as frozen shoulder,[18] chondrocalcinosis,[19] and carpal tunnel syndrome.[20] The incidence of osteoarthritis seemed to be increased in patients with hypothyroidism.[21]

Rheumatological manifestations in patients with hypothyroidism result from hormonal dysfunction[17] and deposition of mucopolysaccharides, notably hyaluronic acid in articular and peri-articular structures.[22] Indeed, the excess of hyaluronic acid in patients with primary hypothyroidism is due to the stimulation of hyaluronic acid synthesis by the increased TSH levels and the inhibition of hyaluronic acid degradation caused by thyroxine deficiency.[17,23–26]

The role of TSH in the pathogenesis of arthropathy was also highlighted because normalization of TSH levels under thyroid hormone substitution therapy leads to improvement and complete resolution of articular symptoms.[14,15,17,27–29]

However, other studies showed that rheumatic manifestations may occur in patients with chronic lymphocytic thyroiditis, even in those with TSH levels within normal ranges.[3,30,31,25,28] Besides, Le Riche et al.[32] reported cases of inflammatory polyarthritis (joint stiffness, tenderness, and effusion) in patients with HT with no improvement of arthritis under thyroid replacement. These findings suggest that rheumatological manifestations can be related to either inflammatory or serological features of HT.[32]

4 | CONCLUSION

The diagnosis of hypothyroidism should be considered in patients with articular impairment, even without classic signs of hypothyroidism.

Thyroid replacement can lead to alleviation or complete resolution of hypothyroid arthropathy.

AUTHOR CONTRIBUTIONS

Dr. Takwa Mehmli: Roles/Writing - original draft. Dr. Maroua Slouma: Methodology and Writing - review & editing. Dr. Rim Dhahri: data curation and formal analysis. Dr. Siwar Ben Dhia: data curation. Dr. Imen Gharsallah:

visualization. Dr. Leila Metoui: conceptualization. Dr. Bassem Louzir: validation.

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CONFLICT OF INTEREST

None.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

CONSENT

Written informed consent was obtained from the patient for publication of the clinical information and any accompanying images.

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REFERENCES

1. Mincer DL, Jialal I. Hashimoto thyroiditis. In: *StatPearls [Internet]*. Treasure Island, FL: StatPearls Publishing; 2021. http://www.ncbi.nlm.nih.gov/books/NBK459262/. PMID: 29083758.
2. Tagoe CE, Zezon A, Khattri S. Rheumatic manifestations of autoimmune thyroid disease: the other autoimmune disease. *J Rheumatol*. 2012;39(6):1125-1129.
3. Punzi L, Betterle C. Chronic autoimmune thyroiditis and rheumatic manifestations. *Joint Bone Spine*. 2004;71(4):275-283.
4. Golden SH, Robinson KA, Saldanha I, Anton B, Ladenson PW. Prevalence and incidence of endocrine and metabolic disorders in the United States: a comprehensive review. *J Clin Endocrinol Metab*. 2009;94(6):1853-1878.
5. Caturegli P, De Remigis A, Rose NR. Hashimoto thyroiditis: clinical and diagnostic criteria. *Autoimmun Rev*. 2014;13(4–5):391-397.
6. Buchanan WW. The relationship of Hashimoto's thyroiditis to rheumatoid arthritis. *Geriatrics*. 1965;20(11):941-948.
7. Karsh J, Pavlidis N, Weintraub BD, Moutsopoulos HM. Thyroid disease in Sjögren's syndrome. *Arthritis Rheum*. 1980;23(11):1326-1329.
8. Gordon MB, Klein I, Dekker A, Rodnan GP, Medsger TA. Thyroid disease in progressive systemic sclerosis: increased frequency of glandular fibrosis and hypothyroidism. *Ann Intern Med*. 1981;95(4):431-435.
9. Scofield RH. Autoimmune thyroid disease in systemic lupus erythematosus and Sjögren's syndrome. *Clin Exp Rheumatol*. 1996;14(3):321-330.
10. Anwar S, Gibofsky A. Musculoskeletal manifestations of thyroid disease. *Rheum Dis Clin North Am*. 2010;36(4):637-646.
11. Duyff RF, Van den Bosch J, Laman DM, van Loon BJ, Linssen WH. Neuromuscular findings in thyroid dysfunction: a prospective clinical and electrodiagnostic study. *J Neurol Neurosurg Psychiatry*. 2000;68(6):750-755.
12. Cruz MW, Tendrich M, Vaisman M, Novis SA. Electroneuromyography and neuromuscular findings in 16 primary hypothyroidism patients. *Arq Neuropsiquiatr*. 1996;54(1):12-18.
13. Mastaglia FL, Ojeda VJ, Sarnat HB, Kakulas BA. Myopathies associated with hypothyroidism: a review based upon 13 cases. *Aust N Z J Med*. 1988;18(6):799-806.
14. Dorwart BB, Schumacher HR. Joint effusions, chondrocalcinosis and other rheumatic manifestations in hypothyroidism. *Am J Med*. 1975;59(6):780-790.
15. Gillan MM, Scofield RH, Harley JB. Hashimoto's thyroiditis presenting as bilateral knee arthropathy. *J Okla State Med Assoc*. 2002;95(5):323-325.
16. Bland JH, Frymoyer JW, Newberg AH, Revers R, Norman RJ. Rheumatic syndromes in endocrine disease. *Semin Arthritis Rheum*. 1979;9(1):23-65.
17. Bland JH, Frymoyer JW. Rheumatic syndromes of myxedema. *N Engl J Med*. 1970;282(21):1171-1174.
18. Cohen C, Tortato S, Silva OBS, Leal MF, Ejnisman B, Faloppa F. Association between frozen shoulder and thyroid diseases: strengthening the evidences. *Rev Bras Ortop*. 2020;55(4):483-489.
19. Job-Deslandre C, Menkes CJ, Guinot M, Luton JP. Does hypothyroidism increase the prevalence of chondrocalcinosis? *Rheumatology*. 1993;32(3):197-198.
20. Frymoyer JW, Bland J. Carpal-tunnel syndrome in patients with myxedematous arthropathy. *JBJS*. 1973;55(1):78-82.
21. Becker KL, Ferguson RH, McConahey WM. The connective-tissue diseases and symptoms associated with Hashimoto's thyroiditis. *N Engl J Med*. 1963;268(6):277-280.
22. Naeye RL. Capillary and venous lesions in myxedema. *Lab Investig J Tech Methods Pathol*. 1963;12:465-470.
23. Asboe-Hansen G. Endocrine control of connective tissue. *Am J Med*. 1959;26(3):470-484.
24. Asboe-Hansen G, Iversen K. Influence of thyrotrophic hormone on connective tissue; pathogenetic significance of mucopolysaccharides in experimental exophthalmos. *Acta Endocrinol*. 1951;8(1):90-96.
25. Pinchera A, Pinchera MG, Stanbury JB. Thyrotropin and long-acting thyroid stimulator assays in thyroid disease. *J Clin Endocrinol Metab*. 1965;25:189-208.
26. Schiller S, Slover GA, Dorfman A. Effect of the thyroid gland on metabolism of acid mucopolysaccharides in skin. *Biochim Biophys Acta*. 1962;58(1):27-33.
27. Zoubeidi H, Daoud F, Rachdi I, et al. Severe hypothyroidism presenting as arthropathy of the fingers: a new case report. *MOJ Orthop Rheumatol*. 2019;11(6):208-210.
28. Punzi L, Sfriso P, Pianon M, et al. Clinical manifestations and outcome of polyarthralgia associated with chronic lymphocytic thyroiditis. *Semin Arthritis Rheum*. 2002;32(1):51-55.
29. Longato L, Tarocco RP, Anania A, Corino D, Quaranta L, Perotti L. Arthropathies and thyroid diseases. *Minerva Endocrinol*. 2004;29(2):55-62.

30. Tagoe CE, Zezon A, Khattri S, Castellanos P. Rheumatic manifestations of euthyroid, anti-thyroid antibody-positive patients. *Rheumatol Int*. 2013;33(7):1745-1752.
31. Golding DN. Rheumatism and the thyroid. *J R Soc Med*. 1993;86(3):130-132.
32. LeRiche NG, Bell DA. Hashimoto's thyroiditis and polyarthritis: a possible subset of seronegative polyarthritis. *Ann Rheum Dis*. 1984;43(4):594-598.

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