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. Hormone replacement therapy leads to the resolution of arthritis related to Hashimoto's thyroiditis, suggesting the role of thyroid hormone in the pathogenesis of arthritis.

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Authors' contributions

- Dr. Takwa MEHMLI: Roles/Writing - original draft

- Dr. Maroua SLOUMA: Methodology and Writing - review & editing

- Dr. Rim DHAHRI: data curation and formal analysis

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- Dr. Imen GHARSALLAH: visualization

- Dr. Leila METOUI: conceptualization

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DECLARATION

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Abstract **(50 words)*:***

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. Hormone replacement therapy leads to the resolution of arthritis related to Hashimoto's thyroiditis, suggesting the role of thyroid hormone in the pathogenesis of arthritis.

Structured abstract:

Introduction:

Rheumatic manifestations such as arthritis and non-specific musculoskeletal symptoms can reveal hypothyroidism.

We report herein the case of an acute asymmetric polyarthritis revealing Hashimoto's thyroiditis.

Case presentation:

A 34-year-old woman presented to our department with acute seronegative polyarthritis.

She had high thyroid-stimulating hormone levels, low thyroxine levels, and positive thyroid peroxidase antibodies. Ultrasonography revealed a thyroiditis aspect.

The diagnosis of Hashimoto's thyroiditis was made.

There were no clinical nor immunological features for the diagnosis of Sjögren's syndrome, rheumatoid arthritis, systemic lupus erythematosus.

After three months of thyroid hormone replacement therapy, the thyroid-stimulating hormone level had become within the normal range, and polyarthritis had resolved.

Conclusion:

We highlight the link between thyroid disorders and rheumatic manifestations. The diagnosis of hypothyroidism should be considered in patients with rheumatic complaints. Hormone replacement therapy leads to the resolution of arthritis related to Hashimoto's thyroiditis, suggesting the role of thyroid hormone in the pathogenesis of arthritis.

Key clinical message:

Hypothyroidism should be considered in polyarthritis patients.

Rheumatic manifestations can be due to thyroid dysfunction or autoimmunity, or associated connective tissue diseases.

Thyroid hormone replacement therapy leads to arthritis resolution.

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

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 Hashimoto's thyroiditis.

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

Case presentation:

A 34-year-old woman with no medical history presented to our department with a five-day history of arthritis affecting the small joints of the left hand, wrist, right elbow, and ankle. She had myalgia without sicca symptoms.

Physical examination showed a normal blood pressure of 110/60 mmHg, synovitis of the left wrist and metacarpophalangeal joints, right elbow, and ankle.

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 high (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 pelvic 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. 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 25µg daily and increased progressively to reach 150 µg daily.

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

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

Discussion:

We report a case of acute polyarthritis revealing Hashimoto's thyroiditis. Hashimoto's thyroiditis 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 Hashimoto's thyroiditis 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].

Hashimoto's thyroiditis can be responsible for various rhematic 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][7][8][9]. Therefore, it is necessary to rule out these diagnoses before attributing articular manifestations to Hashimoto's thyroiditis.

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][15][16][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 [23][24][25][26][17].

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 [27][14][28][15][17][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, LeRiche 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 Hashimoto's thyroiditis [32].

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.

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