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CASE REPORT

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Hypoparathyroidism as one of the initial presentations of systemic lupus erythematosus

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Abstract

Systemic lupus erythematosus (SLE) is an autoimmune disease and may be associated with many autoimmune conditions. Hypoparathyroidism is a rare disease. The leading cause of hypoparathyroidism is postsurgical hypoparathyroidism. However, hypoparathyroidism as an initial presentation of SLE is still a rare condition. Here, we report a case of SLE presented with hypoparathyroidism and Hashimoto's thyroiditis.

KEYWORDS

autoimmune hypoparathyroidism, Hashimoto's thyroiditis, systemic lupus erythematosus

1 | INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune disease and may be associated with many other autoimmune diseases, including autoimmune endocrine disorders.[1] Many endocrine disorders are caused by immune-mediated endocrine gland destruction, including diabetes type 1, Graves' disease, Hashimoto's thyroiditis, Addison's disease, and hypoparathyroidism.[1–4] Here, we report a case of SLE presented with hypoparathyroidism and Hashimoto's thyroiditis.

2 | CASE PRESENTATION

A 19-year-old woman was admitted with low-grade fever and myoclonus in February 2021. She had sought medical attention 40 days before her admission because of delayed menstruation treated with dydrogesterone. After 2 days, she experienced a diffuse erythematous rash on her face and was treated with an antihistamine. However, no improvement was observed, and she developed fever and myoclonic movements. On admission, she had an erythematous rash across the nose and cheek and myoclonic movements in the tongue and lower limbs. Her body temperature was 37.8°C. Other vital signs were in the normal range. Trousseau's and Chvostek's signs were positive. She had no remarkable past medical and family history. Initial laboratory tests showed normochromic normocytic anemia, leukopenia, and hypocalcemia (Table 1). Electrocardiography revealed a prolonged QT interval. Treatment of hypocalcemia was started with calcium gluconate infusion and continued with calcium carbonate (1200 mg elemental calcium daily) and calcitriol (1 microgram daily) orally. Serial serum calcium levels during treatment were: 5.5 (0.6), 5.5 (0.7), 7.8 (0.95), 7.9 (0.91), 8 (0.95), and 8.8 (1.01) mmol/L total (ionized calcium). The tests were requested with the possibility of hypoparathyroidism secondary to autoimmune diseases (Table 1). Thyroid ultrasound showed a large heterogeneous thyroid consisting of many hypoechoic nodules (Hashimoto type).

TABLE 1 Laboratory parameters of the patient

Laboratory parameters	Patient's values	Normal range
Leukocyte count, per µl	2.5×10^3 (67% Neut, 30% Lymp)	4–10 × 103
Hemoglobin, g/dl	8.7	12.3–15.3
MCV, fl	86	80–100
Reticulocyte count, %	0.5	0.5–2.5
ESR, mm/h	37	0–30
CRP, mg/L	7	<6
SGOT, g/dl	34	8–35
SGPT, g/dl	31	8–35
Albumin, g/dl	3.5	3.4–5.4
LDH, U/L	640	140–280
BUN, mg/dl	13	7–20
Creatinine, mg/dl	0.7	0.5–1.1
Serum calcium, mg/dl	5.5	8.5–10.3
Serum ionized calcium, mg/dl	1.01	4.4–5.5
Serum magnesium, mg/dl	2.1	1.7–2.2
Serum phosphorus, mg/dl	4.2	3.4–4.5
25 OH vitamin D, ng/dl	15	30–50
iPTH, pg/ml	8	14–72
TSH, mIU/L	7.2	0.5–5
Anti-TPO, IU/ml	1000	<9
ANA, IU/ml	3.7	<0.8
Anti-dsDNA, IU/ml	5.8	<1.2
C3, mg/dl	65	80–160
C4, mg/dl	8	15–45
CH50, mg/dl	31	42–95
Lupus anticoagulant	24	20–39
Anti-cardiolipin (IgM)	4	0–15
Anti-cardiolipin (IgG)	3	0–15
Anti-beta-2-glycoproteins (IgM)	5	0–20
Anti-beta-2-glycoproteins (IgG)	7	0–20

Abbreviations: ALT, aspartate alanine transferase; ANA, antinuclear antibody; ANA, antinuclear antibody; anti-dsDNA, anti-double-stranded DNA; anti-TPO, anti-thyroid peroxidase antibody; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CRP, C- reactive Protein; ESR, erythrocyte sedimentation rate; iPTH, intact parathyroid hormone; LDH, lactic dehydrogenase; Lymph, lymphocyte; MCV, mean corpuscular volume; Neut, neutrophil; TSH, Thyroid-Stimulating Hormone; TSH, Thyroid-stimulating hormone.

Brain magnetic resonance imaging was normal. SLE was diagnosed based on the malar rash, pancytopenia, positive anti-nuclear antibody (ANA), positive anti-dsDNA, and low serum complement levels. The patient was treated with prednisolone 30 mg/day and hydroxychloroquine 5 mg/kg/day. Due to severe hypocalcemia, average phosphorus, and low parathyroid hormone (PTH), hypoparathyroidism was diagnosed as the cause of the patient's hypocalcemia. In our opinion, autoimmune damage to parathyroid glands could be considered the best explanation for hypoparathyroidism in this patient, given no history of surgery or irradiation in the neck, negative family history, or absence of other genetic factors disorders, and underlying SLE disease. According to high thyroid stimulating hormone (TSH) level, normal T4 and T3, and high anti-thyroid peroxidase antibody (anti-TPO), the diagnosis of sub-clinical Hashimoto's thyroiditis was also made.

3 | DISCUSSION

This study reported a young woman who presented the sign and symptoms of hypoparathyroidism simultaneously

with the patient's initial SLE diagnosis. Acquired hypoparathyroidism results from deficient PTH secretion following surgery, radiation or autoimmune damage to the parathyroid glands, and storage or infiltrative diseases of the parathyroid glands.[5] Postsurgical and idiopathic hypoparathyroidism are the most common causes.[5,6] An autoimmune reason for idiopathic hypoparathyroidism (IH) has been suggested because of the close association between IH and other autoimmune diseases.[6] Autoantibodies against parathyroid cells, including calium-sensing receptor (CaSR) and mitochondrial antigens, were found in the serum of patients with IH.[6] The CaSR senses calcium concentration, stimulates PTH secretion, and increases the reabsorption of Ca by the renal tubules. Destroying CaSR by these autoantibodies led to PTH secretion and Ca absorption reduction.[7]

SLE associated with hypoparathyroidism is underestimated and usually has subclinical manifestation. Hypoparathyroidism associated with SLE is extremely rare; to our knowledge, only 10 cases have been reported.[8–13] Despite the low incidence, hypoparathyroidism has significant complications and symptoms, including prolonged QT interval, which may lead to sudden death; severe hypocalcemia may lead to heart failure; long-term hyperphosphatemia may cause calcification and ossification of several vital tissues. In 80% of cases, hypoparathyroidism presented before or simultaneous with SLE. In 20% of cases, autoimmune thyroid disease co-exists with hypoparathyroidism. Thyroid autoimmunity is more common, reported in 6–60% of SLE patients. Anti-TPO antibody and Hashimoto's thyroiditis have been reported in up to 33% and 8% of patients with SLE, respectively.[1]

4 | CONCLUSION

This study reminds us to consider the possibility of autoimmune hypoparathyroidism and pay attention to the symptoms of this condition before and during the diagnosis of SLE.

AUTHOR CONTRIBUTIONS

Leyla Gadakchi: The conception and design of the report and preparing the manuscript. Ali-Asghar Ebrahimi: Drafting the article or revising it critically for important intellectual content. Vahideh Sadra: Drafting the article or revising it critically for important intellectual content. Mohammadreza Moslemi: Drafting the manuscript or revising it critically. Alireza Khabbazi: The conception and design of the report and final approval of the version to be submitted.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICAL APPROVAL

This study was performed according to the principles outlined by the World Medical Association's Declaration of Helsinki on experimentation involving human subjects, as revised in 2000, and has been approved by the ethics committee of the Tabriz University of Medical Sciences.

CONSENT

Written informed consent was obtained from the patient to publish this report and clinical images. Consent has been signed and collected by the journal s patient consent policy.

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