Hypoparathyroidism as an initial presentation of systemic lupus erythematosus

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April 15, 2022

Abstract

Systemic lupus erythematosus (SLE) is an autoimmune disease and may be associated with many autoimmune conditions. hypoparathyroidism as an initial presentation of SLE is still a rare condition. In this study, we reported a young woman who presented the sign and symptoms of hypoparathyroidism.

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Title: Hypoparathyroidism as an initial presentation of systemic lupus erythematosus: a case report

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 post-surgical hypoparathyroidism. However, hypoparathyroidism as an initial presentation of SLE is still a rare condition. In this study, we reported a young woman who presented the sign and symptoms of hypoparathyroidism simultaneously with the patient's initial SLE diagnosis.

Keywords:

Systemic lupus erythematosus; autoimmune hypoparathyroidism; Hashimoto's thyroiditis

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.

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 two 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. Their 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 was 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).

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/d and hydroxychloroquine 5 mg/kg/d. 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 was the best explanation for hypoparathyroidism in this patient, given no history of surgery or irradiation in the neck, negative family history, absence of other genetic disorders, and underlying SLE disease. According to high TSH level, standard T4 and T3, and high anti-thyroid peroxidase antibody (anti-TPO), the diagnosis of sub-clinical Hashimoto's thyroiditis was also made.

Discussion

We reported a case of SLE presented with hypoparathyroidism. 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 and autoantibodies against parathyroid cells antigens. These antibodies include calcium-sensing receptors and mitochondrial antigens in the serum of patients with hypoparathyroidism(6).CaSR senses calcium concentration and stimulates PTH secretion by the parathyroid and calcium reabsorption by the renal tubules. When CaSR is destroyed, PTH secretion and calcium absorption are depleted(7).

SLE associated with hypoparathyroidism is underestimated and usually has subclinical manifestation. Hypoparathyroidism associated with SLE is extremely rare, and to the best of our knowledge, only 10 cases have been reported(8-13). 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).

Conclusion

Hypoparathyroidism can be considered part of the endocrine disorders described in SLE. However, more

research should be conducted to determine hypoparathyroidism incidence in SLE patients. This study reported a young woman who presented the sign and symptoms of hypoparathyroidism simultaneously with the patient's initial SLE diagnosis. Despite the low incidence, hypoparathyroidism has important 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. This study reminds us of the importance of considering and paying attention to the symptoms of hypocalcemia before and during the diagnosis of SLE.

Acknowledgment

We want to express our thanks to Dr. Leila Khabbazi, who helped us with the editing of the text.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflicts of interest

The authors declare that there is no conflict of interest.

Ethics

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.

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.

Consent statement

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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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 to 39

Laboratory parameters	Patient's values	Normal range
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

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