

The onset of systemic lupus erythematosus and thyroid dysfunction following Graves' disease – A case report and literature review

Yuanyuan Zhanga¹, Xiaoyan Xiaoa¹, Qiufa Haoa¹, Xianhua Lia¹, Jianmin Renb², Zhao Hu¹

¹Qilu Hospital of Shandong University, Department of Nephrology, Jinan, China;

²Qilu Hospital of Shandong University, Department of Endocrinology, Jinan, China

SUMMARY

Introduction Graves' disease is a multifactorial autoimmune thyroid disease, with the presence of typical circulating autoantibodies that can activate the thyroid hormone receptors, resulting in hyperthyroidism, goiter, and ophthalmopathy. Systemic lupus erythematosus is a multi-systemic autoimmune disease that involves almost all the organs of the human body and is characterized by autoantibodies formation. Several studies have reported that autoimmune thyroid and rheumatic disorders can present an unusual relationship.

Case Outline We report a case of a middle-aged woman who presented with systemic lupus erythematosus one year after being diagnosed with Graves' disease. Prednisone and cyclophosphamide were administered to control the development of systemic lupus erythematosus. Furthermore, a percutaneous thyroid biopsy was performed for further confirmation of Graves' disease. Methimazole instead of propylthiouracil was added into the therapeutic scheme. A month later, the patient's clinical manifestation and laboratory tests got significant improvement, except that new thyroid dysfunction appeared opposite to the original one. The administration of anti-thyroid drug was discontinued. With a period of decreased administration of prednisone, the patient's thyroid function gradually got back to normal levels without any levothyroxine replacement.

Conclusion In conclusion, the clinical use of prednisone and antithyroid drugs may result in instability of the hypothalamus–pituitary–thyroid axis, and thyroid function should be carefully monitored in such patients.

Keywords: Graves' disease; systemic lupus erythematosus; thyroid dysfunction

INTRODUCTION

Graves' disease (GD) is a multifactorial autoimmune thyroid disease (AITD), with the presence of typical circulating autoantibodies which could activate the thyroid hormone receptor, resulting in hyperthyroidism, goiter, and ophthalmopathy [1]. Systemic lupus erythematosus (SLE), as a multi-organ autoimmune disorder, is characterized by a loss of self-tolerance and organ dysfunction. The autoantibodies are mistakenly directed to attack healthy tissue [2, 3]. The association between thyroid disease and SLE was first mentioned in 1961 by White et al. [4] and Hijmans et al. [5]. Moreover, thyroid disorders appear to be more frequent in SLE patients [6]. Several studies have reported that a pathogenic association of AITD with SLE may exist in a wide range of variability [7, 8, 9]. The mechanisms by which AITD may be linked to systemic autoimmune diseases have not been fully clarified yet; however, alterations of common pathways are suggested by shared genetic variants affecting autoantigen presentation and regulation of the immune response [8].

Herein, we report a case of a diagnosis of GD and SLE, followed by the development of a new thyroid dysfunction after prednisone and cyclophosphamide (CTX) treatment. We

also reviewed the medical literature on thyroid problems induced by the glucocorticoids usage.

CASE REPORT

A 48-year-old woman who complained of palpitation, hyperhidrosis, for one year and edema of lower extremities for two months was admitted to Qilu Hospital of Shandong University in March 2015. The woman complained of weakness, dysphoria, chest tightness and wheezes after exercise, irregular menstruation for one year, and weight loss of 5 kg in two months. She was diagnosed with GD, hypertension, and coronary heart disease in the local hospital. She received propylthiouracil (PTU), metoprolol, valsartan, and indapamide for treatment and the symptoms were soon relieved. However, two months later, edema of lower extremities, alopecia, skin erythema, irregular fever, and foam urine started presenting themselves and the patient was referred to Qilu Hospital of Shandong University for further treatment.

On physical examination, her temperature was 36.2°, pulse was 101 beats/min. and regular, respiratory rate was 23 breaths/min., and blood pressure was 161/89 mmHg. The physi-

Correspondence to:

Zhao HU
Department of Nephrology
Qilu Hospital, Shandong
University
107 Wenhua Xi Road, Jinan
250012
Shandong Province
China
h212040813@163.com

cal examination revealed poor spirit, angular face, and erythematous patches on her neck. Diffuse goiter and axillary lymph nodes were palpated. No tremor or vascular murmur could be found in the thyroid. Increased breath sounds were listened to on both sides of the chest. The abdomen was remarkable for splenomegaly. There was marked edema of lower extremities.

After the admission on March 25, 2015, the patient underwent a series of laboratory tests and imaging examinations. The laboratory data are listed in Table 1. Chest and abdomen computed tomography revealed focal fibrosis and hydrothorax of double lung, hydropericardium, cholecystitis, hydrocholecystitis, pancreatitis, splenomegaly, and the mediastinal lymph node enlargement. Doppler sonography of the thyroid showed multiple thyroid nodules (goiter with adenoma).

The patient was diagnosed as GD; SLE, lupus nephritis; hypertension (Grade 2; extremely high risk group); and coronary heart disease. The disease activity index of SLE (SLEDAI) was 14. Percutaneous renal biopsy was performed. The histological findings were membranoproliferative glomerulonephritis along with "full-house" deposits by immunofluorescence staining, which was considered lupus nephritis (WHO IV-Ga, Figure 1). The electron microscope further showed the deposits were electron-dense and diffusely located in the subendothelial, subcutaneous, and mesangial area consistent with lupus nephritis (WHO IV-Ga, Figure 2). The patient was treated with prednisone 1 mg/kg/day along with CTX 0.6 g/m² per month. Fine-needle aspiration biopsy (FNAB) of the thyroid was performed. FNAB showed cellular smear with similar features to hyperplastic nodule (Figure 3). PTU was discontinued and methimazole (10 mg bid) was added in case of PTU-induced lupus.

After two weeks, the patient was alleviated with disappeared erythematous patches, fever and relieved edema. Urinalyses revealed white blood cell count (WBC) 30 p/ul, red blood cell count (RBC) 444.3 p/μL, proteinuria/24h 11.29 g. Blood routine showed hemoglobin (HGB) 128 g/L (115–150). Hepatic and renal function demonstrated serum creatinine 75 μmol/L (53–97), albumin 22.1 g/L (40–55). Her treatment was continued according to the original plan. A month later, the laboratory analysis revealed urine WBC 10.56 p/μL, RBC 24.42 p/μL, proteinuria/24h 5.43 g; HGB 112 g/L (115–150); serum albumin 18.3 g/L (40–55). Thyrotropin (TSH) 0.01 μIU/ml (0.35–4.94), free triiodothyronine (FT3) 1.54 pmol/L (2.63–5.70), and free thyroxine (FT4) 5.15 pmol/L (9.01–19.05) were all below the normal level. Methimazole was discontinued. She continued to receive prednisone 1 mg/kg/day plus CTX 0.6 g/m² again. Two months later, her clinical manifestation and laboratory index improved significantly. Laboratory test results included proteinuria/24h 3.35g; serum albumin 24.6 g/L (40–55) and antinuclear antibodies (ANAs) positive for (1:160). TSH 0.211 μIU/ml (0.35–4.94), FT3 1.54 pmol/L (2.63–5.70) and FT4 5.15 pmol/L (9.01–19.05), were still below the normal level. Amounts of prednisone were reduced by 10% of the original amount every two weeks and she continued to receive CTX 0.6 g/m². After three months, laboratory data showed proteinuria/24h 0.95 g, HGB 112

Table 1. Laboratory data on the patient's first admission

Parameter	Value	
Blood routine	WBC (×10 ⁹ /L)	5.13
	RBC (×10 ¹² /L)	3.24
	Hb (g/L)	90
	HCT (%)	25.1
	Platelet (×10 ⁹ /L)	146
Urine	pH	5.0
	Glucose	-
	Occult blood	3+
	RBC (p/μl)	46.2
	WBC (p/μl)	58.2
	Ketone body	-
	ACR (g/g)	8.01
Urine renal tubular function	κ/λ (0.75–4)	1.56
	Albumin (mg/L)	1960
	β ₂ -MG (mg/L)	0.22
	α ₁ -MG (mg/L)	166
Hepatitis and tumor marker	IgG (mg/L) (0–14)	159
	Hepatitis B surface antigen	-
	Hepatitis C antibody	-
Biochemistry	Tumor marker	-
	Total protein (g/L)	35.5
	Albumin (g/L)	15.6
	ALT (IU/L)	8.0
	AST (IU/L)	17.0
	γ-GTP (IU/L)	19.0
	LDL-C (mmol/L)	2.95
	TG (mmol/L)	1.41
	HDL-C (mmol/L)	1.21
	BUN (mmol/L)	11.72
	Cre (μmol/L)	70
	eGFR (mg/min/1.73m ²)	88.2
	UA (μmol/L)	590
	K (mmol/L)	4.29
	Cl (mmol/L)	108
Na (mmol/L)	136	
Ca (mmol/L)	1.68	
Thyroid-related	TSH (μIU/ml)	<0.003
	FT ₃ (pmol/L)	8.39
	FT ₄ (pmol/L)	15.12
	TPO-Ab (IU/ml)	650.81
	TG-Ab (IU/ml)	245.95
	TR-Ab (μIU/ml) (0–1.22)	5.66
	Rheumatic-related	ANAs
dsDNA (IU/ml) (<100)		82.97
c-ANCA (U/ml) (<5)		4.0
p-ANCA (U/ml) (<5)		0.53
GBM-Ab		-
ACL-IgG (U/ml) (<10)		2.49
ACL-IgM (U/ml) (<10)		2.84
SSA-Ab		-
SSB-Ab		-
Sm-Ab		-
Jo-1-Ab		-
Scl-70-Ab		-
nRNP-/Sm-Ab		-
AMA (<1:20)		-
GPI (mg/L) (<0.2)		0.12
ACPA (Ru/ml) (<25)		4.15
Serum C3 (g/L)		0.42
Serum C4 (g/L)		0.094
IgG (g/L)	7.12	
IgA (g/L)	3.25	
IgM (g/L)	0.80	
Type of T cells	CD3+ cells (%)	73.4
	CD3+CD4+/CD3+CD8+	0.84
	CD3+CD4+ cells (%)	32.28
	CD3+CD8+ cells (%)	38.54

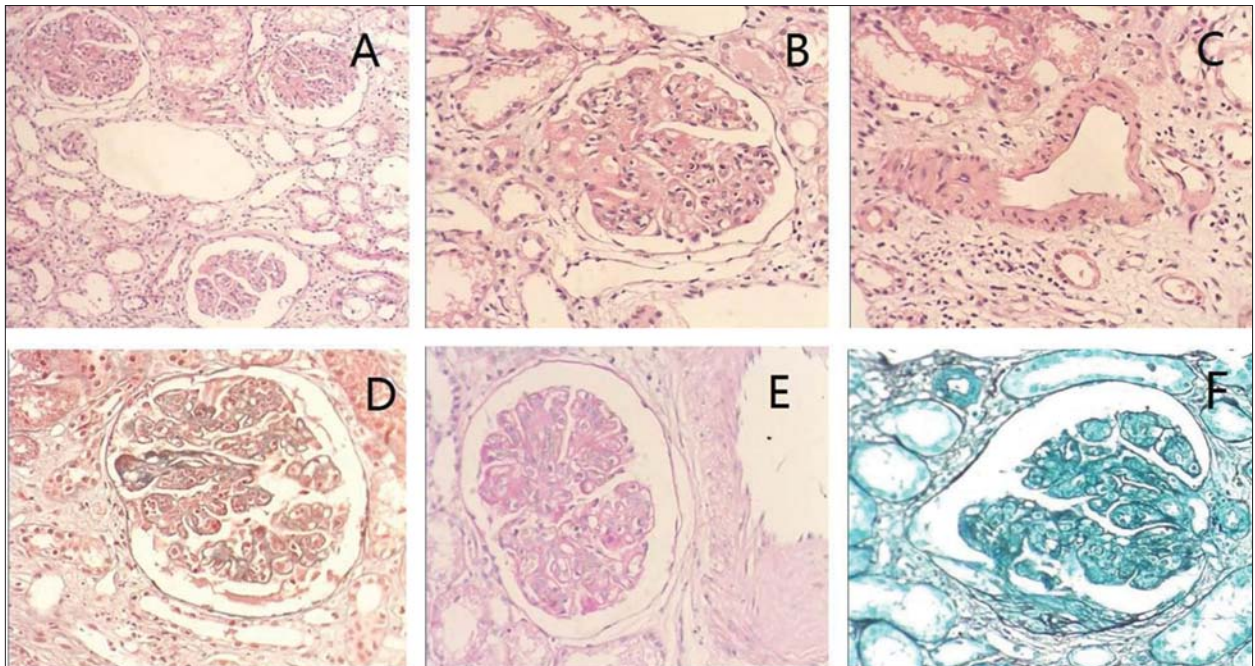


Figure 1. Histopathology of renal sections with HE (hematoxylin-eosin), Masson, PAS (periodic acid-Schiff staining), and PASM (periodic acid silver methenamine) staining; A: HE staining (×100); B: HE staining (×400); C: HE staining of tubulointerstitial (×400); D: Masson staining (×400); E: PAS staining (×400); F: PASM staining (×400)

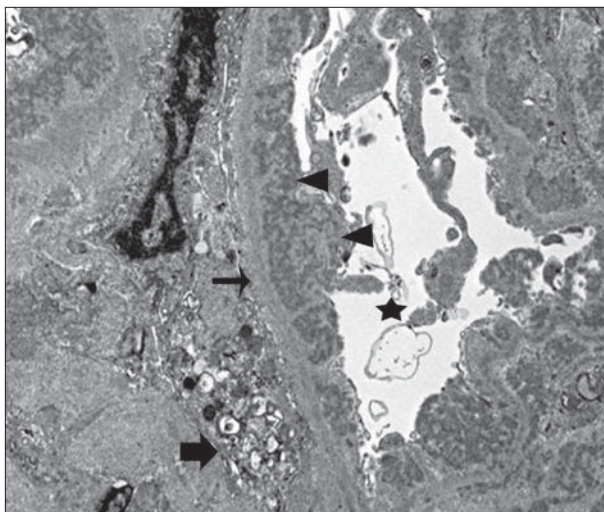


Figure 2. Renal electron microscopy (magnification ×6,000): glomerular basement membrane (thin arrow), podocyte (thick arrow), electron deposits (arrowheads), vessel lumen (star)

g/L (115–150), serum albumin 30.8 g/L (40–55), ANAs <:80 (<1:80), anti-double stranded DNA antibody (anti-dsDNA) 3.97 U/ml (0–100), TSH 0.684 μIU/ml (0.35–4.94), FT3 1.56 pmol/L (2.63–5.70), FT4 6.38 pmol/L (9.01–19.05), which all showed significant improvement. On the follow-up four months later, kidney and thyroid function were essentially back to normal range, and proteinuria was approximately 0.18 g / 24 h. The patient's thyroid function from the hospitalization to the last follow-up is shown in Graph 1. Changes of serum albumin and urine protein of the previous seven months are shown in Graph 2.

DISCUSSION

Several studies have revealed a conceivable relationship between thyroid disease and SLE [7, 8, 9]. Both GD and SLE are multi-systemic autoimmune disorders sharing common genetic basis. The strongest association for both

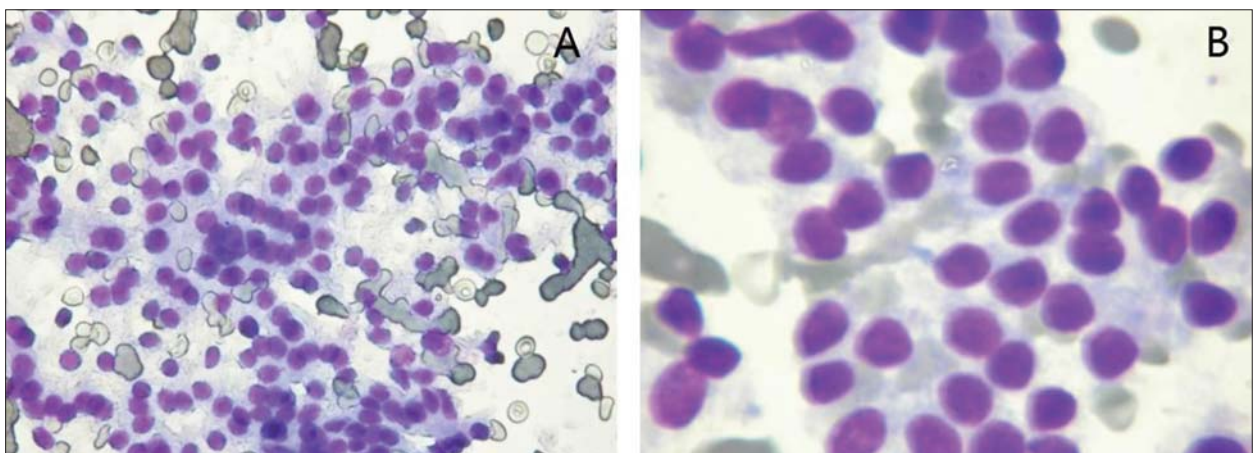
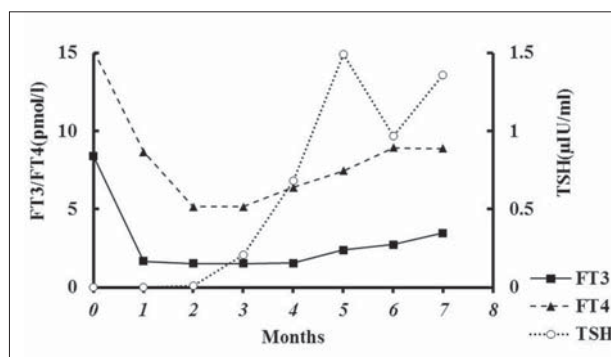
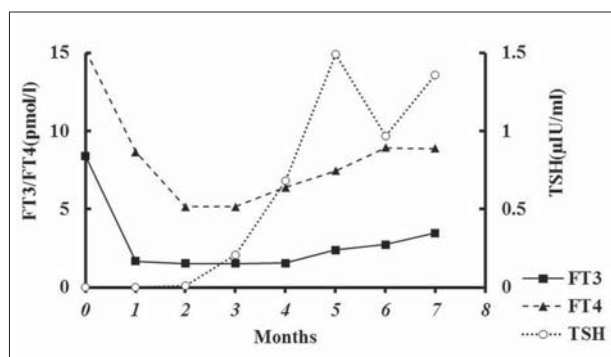


Figure 3. Thyroid tissue fine-needle aspiration biopsy with HE staining; A: magnification ×40; B: magnification ×100



Graph 1. Patient's thyroid function change from hospitalization in March 2015 to the last follow-up

FT3 – free triiodothyronine; FT4 – free thyroxine; TSH – thyrotropin



Graph 2. Corresponding change in serum albumin and urine protein from March 2015 to the last follow-up

GD and SLE are HLA haplotypes including HLA-B8, HLA-DR3 and inositol 1,4,5-triphosphate receptor type 3 (ITPR3) [8, 10]. Sex hormones have been considered to be responsible for susceptibility to autoimmune disease through modulation of Th1/Th2 response. Estrogens appear to promote autoimmune disease with a type 2 cytokine profile, such as in GD and SLE [11]. Therefore, our case with both GD and SLE is not accidental.

Drug-induced lupus (DIL) related to PTU is not rare, therefore correct diagnosis of idiopathic or drug induced lupus is very important for patients treated with PTU. DIL is characterized as musculoskeletal (joint and muscle) pain, serositis and constitutional manifestations such as fever, fatigue, and loss of appetite [12]. Laboratory findings of DIL, specifically serum positivity for ANAs and antihistone antibodies, along with negative anti-Smith antibodies, anti-dsDNA and normal complement profile are common. Classic mucocutaneous signs including malar erythema, discoid lesions, hair loss, and oral ulcers are also common in DIL. However, renal or neurologic manifestations are not usually involved in it. The course of DIL is usually benign and remission over several weeks after discontinuation of the inducing drug is usually seen. There is less chance of DIL in this middle-aged woman with PTU for one year, considering unmatched clinical and serologic manifestations.

In addition, GD can present with similar manifestations of SLE due to overlapping clinical and laboratory criteria. The differentiation between these two diseases requires

careful laboratory evaluation. Diagnosis of SLE should be suspected if patients are diagnosed with GD or have received PTU with characteristic symptoms of SLE and positive ANA. Patients with GD who display SLE but not enough for a diagnosis of SLE should be closely followed up for avoiding misdiagnoses and mistreatments.

Glucocorticoids commonly used for the treatment of SLE can inhibit the secretion of serum TSH, and reduce the thyroid hormone by several mechanisms. There are numerous targets of drug interaction on the pathways of thyroid hormone at different phases of synthesis, secretion, and transport in the circulation and metabolism [13]. Being the single best marker of thyroid function, serum TSH values are usually low or normal in patients with hyperthyroidism, but they are usually high due to the biologically inactive TSH secretion [13, 14]. Some experts have reported that high-dose glucocorticoids suppress the secretion of TSH in hypothyroid patients and normal subjects [15]. Glucocorticoids may suppress the secretion of TSH depending on protein kinase C [16]. Recently, high doses of glucocorticoids have been found to play a role in decreasing the level of TRH mRNA in human hypothalamus, which may illustrate the mechanism by which lower TSH levels are secreted from the pituitary gland [17]. In addition, glucocorticoids can impair peripheral 5'-deiodination of T4 and lower serum thyroid binding globulin (TBG) level [18]; therefore, serum concentrations of TT4, FT4, and TT3 might decrease.

Glucocorticoids are commonly used for treatment of SLE due to its immunosuppressive effects, but their use can lead to thyroid dysfunction, especially in patients receiving antithyroid agents. We reviewed the literature of patients with thyroid dysfunction induced by use of various glucocorticoids and related data is shown in Table 2. Most of the patients were female with mean age of 43 years. It is believed that thyroid dysfunction induced by glucocorticoid cessation may be the rebound of immune activity [19, 20, 21]. Bartalena et al. [22] reported that Graves' hyperthyroidism and ophthalmopathy occur during chronic low-dose glucocorticoid therapy. They suggested that a high dose of glucocorticoids for the treatment of severe Graves' ophthalmopathy might indeed suppress the disease as well. However, the low dose use might aggravate the disease [22]. Glucocorticoids can lower serum potassium and induce thyroid periodic paralysis by several mechanisms such as an increased Na⁺/K⁺-ATPase pool in skeletal muscles, steroid-induced hyperinsulinemia, and hyperglycemia [23]. Interestingly, we observed the patient developed a new onset of thyroid dysfunction in which FT3, FT4, and TSH are all below the normal level and may be called "central hypothyroidism" after receiving glucocorticoid and CTX in our case. To our knowledge, there are no reported cases of developing "central hypothyroidism" with simultaneous treatment with glucocorticoids and antithyroid agents. The possibility of "central hypothyroidism" in our case could be speculated upon as follows: glucocorticoid can decrease secretion of TSH and lower serum TSH level through direct effects on TRH in the hypothalamus [18]. Glucocorticoid and methimazole

Table 2. Thyroid dysfunction induced by the use of glucocorticoids

Reference	Maruyama et al. [19]	Nagai et al. [20]	Morita et al. [21]	Bartalena et al. [22]	Wongprasert et al. [23]
Age (years)	51	42	53	38	23
Sex	F	F	F	F	M
Primary disease	RA, CLT	Uveitis	AR	PV	CIDP, GD
Inducing factors	Dexamethasone cessation	Prednisone cessation	Celestamine cessation	Oral prednisone	Pulse methylprednisolone
Manifestations	Thyrotoxicosis	Goiter	Hand tremor	Thyrotoxicosis	Paralysis
Pathology	CLT	HT	None	None	None
Diagnosis	CLT	HT	PT	GD	TPP
Treatment	None	None	Thiamazole	Radioiodine	Potassium
Recovery time (months)	2	2	14	4	Several hours
Follow-up time (months)	9	3	18	Not given	Not given
Outcome	Hypothyroidism	Euthyroid	Euthyroid	Euthyroid	Euthyroid

F – female; M – male; RA – rheumatic arthritis; CLT – chronic lymphocytic thyroiditis; AR – allergic rhinitis; PV – pemphigus vulgaris; CIDP – chronic inflammatory demyelinating polyneuropathy; GD – Graves' disease; HT – Hashimoto's thyroiditis; PT – painless thyroiditis; TPP – thyrotoxic periodic paralysis

decreased the level of thyroid hormone at the beginning of treatment; however, thyroid-stimulating hormone level does not immediately increase because of glucocorticoid inhibitory effect on hypothalamus. Therefore, reduced use of anti-thyroid drugs should be considered in patients who developed GD with other diseases received both glucocorticoid and anti-thyroid drugs to avoid instability of the hypothalamus–pituitary–thyroid axis. We didn't administer levothyroxine replacement since the patient had suffered from GD before. Her thyroid function improved markedly along with prednisone reduction as expected. As the pattern of thyroid dysfunction may actually reflect a sick euthyroid state that may normalize after hypercortisolism is resolved, we recommend withholding thyroid hormone replacement under this condition.

In summary, SLE and Graves' disease are systemic autoimmune diseases sharing common genetic basis and similar clinical manifestations. The clinical use of prednisone and antithyroid drugs may result in instability of

the hypothalamus-pituitary-thyroid axis and the thyroid function should be carefully monitored in such patients

ACKNOWLEDGEMENTS

We thank Yan Liu and Junhui Zhen (Qilu hospital of Shandong University) for pathology analysis.

NOTE

The patient's written consent was obtained according to the Declaration of Helsinki.

Yuanyuan Zhang and Xiaoyan Xiao contributed equally to this work.

The study was supported by General Financial Grant from China Postdoctoral Science Foundation (No. 2015M572048).

REFERENCES

- Menconi F, Maccocci C, Marino M. Diagnosis and classification of Graves' disease. *Autoimmun Rev.* 2014; 13(4-5):398–402. [DOI: 10.1016/j.autrev.2014.01.013] [PMID: 24424182]
- Ghodke-Puranik Y, Niewold TB. Immunogenetics of systemic lupus erythematosus: A comprehensive review. *J Autoimmun.* 2015; 64:125–36. [DOI: 10.1016/j.jaut.2015.08.004] [PMID: 26324017]
- Tsokos GC. Systemic lupus erythematosus. *N Engl J Med.* 2011; 365(22):2110–21. [DOI: 10.1056/NEJMr1100359] [PMID: 22129255]
- White RG, Bass BH, Williams E. Lymphadenoid goitre and the syndrome of systemic lupus erythematosus. *Lancet.* 1961; 1(7173):368–73. [DOI: 10.1016/S0140-6736(61)91537-9] [PMID: 13784857]
- Hijmans W, Doniach D, Roitt IM, Holborow EJ. Serological overlap between lupus erythematosus, rheumatoid arthritis, and thyroid auto-immune disease. *Br Med J.* 1961; 2(5257):909–14. [PMID: 13714204]
- Kumar K, Kole AK, Karmakar PS, Ghosh A. The spectrum of thyroid disorders in systemic lupus erythematosus. *Rheumatol Int.* 2012; 32(1):73–8. [DOI: 10.1007/s00296-010-1556-5] [PMID: 20658291]
- Antonelli A, Fallahi P, Mosca M, Ferrari SM, Ruffilli I, Corti A, et al. Prevalence of thyroid dysfunctions in systemic lupus erythematosus. *Metabolism.* 2010; 59(6):896–900. [DOI: 10.1016/j.metabol.2009.10.010] [PMID: 20005534]
- Bourji K, Gatto M, Cozzi F, Doria A, Punzi L. Rheumatic and autoimmune thyroid disorders: a causal or casual relationship? *Autoimmun Rev.* 2015; 14(1):57–63. [DOI: 10.1016/j.autrev.2014.10.007] [PMID: 25315745]
- Lin WY, Chang CL, Fu LS, Lin CH, Lin HK. Systemic lupus erythematosus and thyroid disease: A 10-year study. *J Microbiol Immunol Infect.* 2015; 48(6):676–83. [DOI: 10.1016/j.jmii.2014.03.004] [PMID: 24874431]
- Oishi T, Iida A, Otsubo S, Kamatani Y, Usami M, Takei T, et al. A functional SNP in the NKX2.5-binding site of ITPR3 promoter is associated with susceptibility to systemic lupus erythematosus in Japanese population. *J Hum Genet.* 2008; 53(2):151–62. [DOI: 10.1007/s10038-007-0233-3] [PMID: 18219441]
- Fairweather D, Frischno-Kiss S, Rose NR. Sex differences in autoimmune disease from a pathological perspective. *Am J Pathol.* 2008; 173(3):600–9. [DOI: 10.2353/ajpath.2008.071008] [PMID: 18688037]
- Pretel M, Marques L, Espana A. Drug-induced lupus erythematosus. *Actas Dermosifiliogr.* 2014; 105(1):18–30. [DOI: 10.1016/j.ad.2012.09.007] [PMID: 23164669]
- Surks MI, Sievert R. Drugs and thyroid function. *N Engl J Med.* 1995; 21;333(25):1688–94. [DOI: 10.1056/NEJM199512213332507] [PMID: 7477223]

14. Horimoto M, Nishikawa M, Ishihara T, Yoshikawa N, Yoshimura M, Inada M. Bioactivity of thyrotropin (TSH) in patients with central hypothyroidism: comparison between in vivo 3,5,3'-triiodothyronine response to TSH and in vitro bioactivity of TSH. *J Clin Endocrinol Metab.* 1995; 80(4):1124–8. [DOI: 10.1210/jcem.80.4.7714080] [PMID: 7714080]
15. Wilber JF, Utiger RD. The effect of glucocorticoids on thyrotropin secretion. *J Clin Invest.* 1969; 48(11):2096–103. [DOI: 10.1172/JCI106176] [PMID: 4980930]
16. John CD, Christian HC, Morris JF, Flower RJ, Solito E, Buckingham JC. Kinase-dependent regulation of the secretion of thyrotrophin and luteinizing hormone by glucocorticoids and annexin 1 peptides. *J Neuroendocrinol.* 2003; 15(10):946–57. [PMID: 12969239]
17. Alkemade A, Unmehopa UA, Wiersinga WM, Swaab DF, Fliers E. Glucocorticoids decrease thyrotropin-releasing hormone messenger ribonucleic acid expression in the paraventricular nucleus of the human hypothalamus. *J Clin Endocrinol Metab.* 2005; 90(1):323–7. [DOI: 10.1210/jc.2004-1430] [PMID: 15509645]
18. Haugen BR. Drugs that suppress TSH or cause central hypothyroidism. *Best Pract Res Clin Endocrinol Metab.* 2009; 23(6):793–800. [DOI: 10.1016/j.beem.2009.08.003] [PMID: 19942154]
19. Maruyama H, Kato M, Mizuno O, Kataoka K, Matsuki S. Transient thyrotoxicosis occurred after cessation of steroid therapy in a patient with autoimmune thyroiditis and rheumatoid arthritis. *Endocrinol Jpn.* 1982; 29(5):583–8. [DOI: http://doi.org/10.1507/endocrj1954.29.583] [PMID: 6897816]
20. Nagai Y, Ieki Y, Ohsawa K, Kobayashi K. Simultaneously found transient hypothyroidism due to Hashimoto's thyroiditis, autoimmune hepatitis and isolated ACTH deficiency after cessation of glucocorticoid administration. *Endocr J.* 1997; 44(3):453–8. [DOI: 10.1507/endocrj.44.453] [PMID: 9279524]
21. Morita S, Ueda Y, Yokoyama N. Painless thyroiditis induced by the cessation of betamethasone. *Intern Med.* 2001; 40(8):744–6. [DOI: 10.2169/internalmedicine.40.744] [PMID: 11518115]
22. Bartalena L, Bogazzi F, Chiovato L, Tanda ML, Martino E. Graves' hyperthyroidism and ophthalmopathy associated with pemphigus vulgaris: onset of thyroid autoimmune disease during chronic low-dose glucocorticoid therapy. *J Endocrinol Invest.* 1997; 20(3):155–7. [DOI: 10.1007/BF03346895] [PMID: 9186823]
23. Wongraoprasert S, Buranasupkajorn P, Sridama V, Snaboon T. Thyrotoxic periodic paralysis induced by pulse methylprednisolone. *Intern Med.* 2007; 46(17):1431–3. [DOI: 10.2169/internalmedicine.46.0044] [PMID: 17827844]

Јављање системског еритемског лупуса и дисфункције штитасте жлезде током Грејвс–Базедовљеве болести – приказ болесника и преглед литературе

Јуанјуан Џанга¹, Сјаојан Сјаоа¹, Ћуфа Хаоа¹, Сјанхуа Лиа¹, Ћенмин Женб², Џао Ху¹

¹Болница „Ћилу“ Универзитета „Шандонг“, Одељење за нефрологију, Ћинан, Кина;

²Болница „Ћилу“ Универзитета „Шандонг“, Одељење за ендокринологију, Ћинан, Кина

КРАТАК САДРЖАЈ

Увод Грејвс–Базедовљева болест је мултифакторна аутоимуна болест штитасте жлезде, уз присуство типичних циркулишућих аутоантитела која могу активирати рецептор хормона штитасте жлезде, што резултира хипертироидизмом, гушавошћу и офталмопатијом. Системски еритемски лупус је мултисистемска аутоимуна болест која утиче на скоро све органе људског тела, а коју карактерише формирање антитела. У неколико студија је наведено да аутоимуни тироидни и реуматски поремећаји могу успоставити необичан однос.

Приказ болесника Приказујемо случај средовечне жене којој се јавио системски еритемски лупус годину дана пошто јој је успостављена дијагноза Грејвс–Базедовљеве болести. Преписани су јој преднисон и циклофосфамид како би се ограничио развој системског еритемског лупуса. Уз то је извршена перкутана биопсија штитасте жлезде за по-

тврду дијагнозу Грејвс–Базедовљеве болести. У терапију је уведен метимазол уместо пропилтиоурацила. Месец дана касније клиничка слика и лабораторијски налази значајно су се побољшали, с тим што се нова дисфункција штитасте жлезде јавила као супротност првобитној дисфункцији. Прекинута је примена анти tiroидних лекова. Уз смањену примену преднисона функција штитасте жлезде болеснице постепено се вратила на нормални ниво без увођења левотироксина.

Закључак Клиничка примена преднисона и анти tiroидних лекова може резултирати нестабилношћу осе хипоталамус – хипофиза – штитаста жлезда, те би код таквих болесника требало пажљиво пратити функцију штитасте жлезде.

Кључне речи: Грејвс–Базедовљева болест; системски еритемски лупус; дисфункција штитасте жлезде

Примљен • Received: 21/12/2015

Ревизија • Revision: 27/04/2016

Прихваћен • Accepted: 15/06/2016