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Case Report

Graves Disease Following Subacute Thyroiditis in a Chinese Man

Souad Al-Bacha, MD, Sharon Wu Lahiri, MD *

Department of Internal Medicine, Division of Endocrinology, Diabetes, Bone and Mineral Disorders, Henry Ford Health System, Detroit, Michigan

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ABSTRACT

Background/Objective: The development of Graves disease (GD) after subacute thyroiditis (SAT) is rare, with approximately 31 reported cases, of which only 5 occurred in men. We describe a case of GD diagnosed based on newly elevated thyroid-stimulating immunoglobulin (TSI) and thyroid-stimulating hormone (TSH) receptor autoantibody (TRAb) levels after SAT.

Case Report: A 32-year-old Chinese man presented with right anterior neck pain, swelling, sore throat, cough, and fever. He had a diffuse tender goiter but no proptosis, lid lag, or stare. His TSH level was 0.03 mIU/mL (normal range [NR] 0.45-5.33 mIU/mL), serum free thyroxine (FT4) level was 2.40 ng/dL (NR 0.61-1.44 ng/dL), total triiodothyronine (TT3) level was 113 ng/dL (NR 87-178 ng/dL), TSI level was <0.10 IU/L (NR < 0.10 IU/L), and erythrocyte sedimentation rate was 21 mm/h (NR < 15 mm/h). After 7 weeks of prednisone, the symptoms resolved, FT4 level was 0.95 ng/dL, and TT3 level was 91 ng/dL. At 11 weeks after SAT onset, the TSH level was <0.01 mIU/mL, TT3 level was 257 ng/dL, FT4 level was 3.03 ng/dL, TSI level was 1.94 IU/L, then 3.42 IU/L 2 weeks later, TRAb level was 8.72 IU/L (NR < 2 IU/L), and erythrocyte sedimentation rate was 4 mm/h. After 1 month of methimazole, the FT4 level was 1.32 ng/dL and TT3 level was 110 ng/dL. Genetic testing revealed human leukocyte antigen-B35 and DRB1*15:01 positivity.

Discussion: GD after SAT is thought to be due to the activation of thyroid autoimmunity induced by SAT in genetically susceptible individuals.

Conclusions: This case illustrates the induction of thyroid autoimmunity after SAT, resulting in GD, supporting TSI and/or TRAb testing if hyperthyroidism recurs. The presence of HLA alleles associated with SAT and GD suggests a genetic contribution to the development of thyroid autoimmunity.

Introduction

Subacute thyroiditis (SAT) is a self-limited inflammation of the thyroid, typically followed by the return of normal thyroid function. Persistent hypothyroidism is rare, but recurrent SAT can occur in up to 20% of cases.[1] Graves disease (GD) after SAT is rare, with only approximately 31 cases described in the literature, and has been reported to occur in <1% of patients presenting with GD or SAT in a Japanese study.[2-15] Thyroid antigen release in patients with SAT is thought to trigger thyroid autoimmunity and GD.[2] Certain human leukocyte antigen (HLA) genes are associated with susceptibility to either GD or SAT alone, but there are inadequate data regarding associations between specific HLA alleles and the risk of GD after SAT.[1,16-18] Herein, we report a 32-year-old Chinese man who developed elevated thyroid-stimulating immunoglobulin (TSI) and thyroid-stimulating hormone (TSH) receptor autoantibody (TRAb) levels after SAT and was found to be positive for HLA-B35 and DRB1*15:01.

Case Report

An otherwise healthy 32-year-old Chinese man presented virtually to his primary care physician on January 19, 2021, with about a 7-day history of right anterior neck pain and swelling, sore throat, cough, fatigue, and a temperature of 37.5 °C. He reported no recent travel, exposure to ill individuals, or SARS-CoV-2 vaccination. He reported no past medical or surgical history or family history of autoimmune disease or cancer. His mother had thyroid

Abbreviations: ESR, erythrocyte sedimentation rate; FT4, free thyroxine; GD, Graves disease; NR, normal range; RAIU, radioactive iodine uptake; SAT, subacute thyroiditis; TPO Ab, thyroid peroxidase antibody; TSH, thyroid-stimulating hormone; TSI, thyroid-stimulating immunoglobulin; TRAb, TSH receptor autoantibody; TT3, total triiodothyronine.

* Address correspondence to Dr Sharon Wu Lahiri, Department of Internal Medicine, Division of Endocrinology, Diabetes, Bone and Mineral Disorders, Henry Ford Health System, New Center One, 3031 W. Grand Blvd, 8th Floor, Detroit, MI 48202.

E-mail address: slahiri1@hfhs.org (S.W. Lahiri).

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nodules but never required thyroid medication. He did not take any medications. Testing for SARS-CoV-2 RNA yielded a negative result, and a 7-day course of amoxicillin was prescribed. He returned virtually on January 26, 2021, because of worsening anterior neck pain, cough, chest tightness, and a fever (39 °C). Chest x-ray revealed no radiographic evidence of acute cardiopulmonary process. Laboratory testing (day 1) revealed a TSH level of 0.03 mIU/mL (normal range [NR] 0.45-5.33 mIU/mL), serum free thyroxine (FT4) level of 2.40 ng/dL (NR 0.61-1.44 ng/dL), serum free triiodothyronine level of 3.9 pg/mL (NR 2.5-4.4 pg/mL), total triiodothyronine (TT3) level of 113 ng/dL (NR 87-178 ng/dL), TSI level of <0.10 IU/L (NR < 0.10 IU/L), and thyroid peroxidase antibody (TPO Ab) level of 3 IU/mL (NR < 9 IU/mL). Computed tomography of the neck showed a diffusely enlarged and heterogeneous-appearing thyroid gland. Thyroid ultrasound demonstrated a diffuse mild increase in vascularity, as determined using color flow Doppler, and estimated the thyroid size to be 50 mL in volume.

He presented in-person to the endocrinology department on February 1, 2021, with the same symptoms and a 13-pound weight loss. On physical examination, his heart rate was 123 beats/min, thyroid was diffusely enlarged and mildly tender bilaterally, and skin was warm and slightly moist. He had no proptosis, chemosis, periorbital edema, lid lag, or stare. Prednisone at 30 mg daily, propranolol 20 mg three times daily, and ibuprofen 200 to 400 mg every 6 to 8 hours were initiated. Testing (day 7) showed a FT4 level of 4.33 ng/dL and TT3 level of 227 ng/dL. Methimazole at 10 mg daily was taken from day 7 to 13 until testing (day 14) revealed a C-reactive protein level of 4.3 mg/dL (NR < 0.5 mg/dL), erythrocyte sedimentation rate (ESR) of 21 mm/h (NR < 15 mm/h), and repeat TSI level of <0.10 IU/L. The laboratory findings are shown in the Figure.

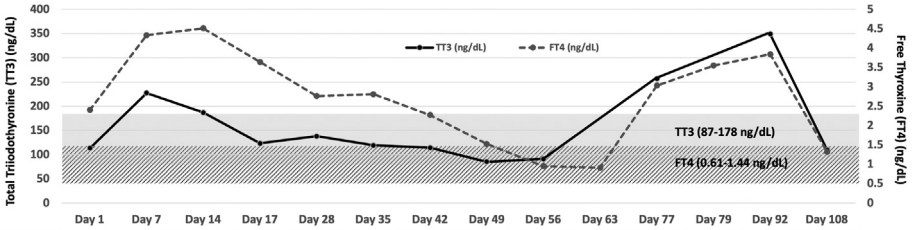
Prednisone was taken for 7 weeks. On day 56, the FT4 level was 0.95 ng/dL, the TT3 level was 91 ng/dL, and he reported no symptoms. On day 77, testing revealed a TSH level of <0.01 mIU/mL, TT3 level of 257 ng/dL, and FT4 level of 3.03 ng/dL. He reported no neck pain or fever but noted occasional palpitations and mild heat intolerance. Prednisone was restarted for 2 days, before the treatment was changed to methimazole (day 79). The TSI level was 1.94 IU/L, increasing to 3.42 IU/L 2 weeks later. The TPO Ab level was 805 IU/mL and TRAb level was 8.72 IU/L (NR < 2 IU/L). The C-reactive protein level and ESR were 0.1 mg/dL and 4 mm/h, respectively. The 24-hour radioactive iodine uptake (RAIU) was 66.2% (NR 10%-30%), and thyroid scan showed diffuse homogeneous radiotracer uptake throughout the thyroid and pyramidal lobe. Ultrasound estimated the thyroid size to be 34 mL in volume, with an increase in vascularity—as determined using color Doppler imaging—similar to a prior study. His symptoms resolved, FT4 level was 1.32 ng/dL, and TT3 level was 110 ng/dL 1 month after starting methimazole.

HLA testing revealed HLA-B35 and -DRB1*15:01 allele positivity. Testing for additional HLA variants (HLA-B*46:01, -B*18:01, -DPB1*05:01, DQB1*05:02, -DQB1*06:04, -DRB1*01, -DRB1*03:01, -DRB1*14:03, and -DRB1*16:02) yielded negative results.

Discussion

The diagnoses of SAT and GD in our patient were based on previously published criteria.[1,11,19] The criteria for the diagnosis of SAT include elevated ESR and either painful and tender goiter or ultrasound findings consistent with SAT in addition to 1 or 2 additional criteria (depressed RAIU, transient hyperthyroidism, fine aspiration needle biopsy findings consistent with SAT, and absent or low titers of thyroid autoantibodies).[1,11] Our patient had a painful tender goiter, elevated ESR, transient hyperthyroidism, and absent thyroid autoantibodies, fulfilling the criteria for SAT.[11]

GD is typically diagnosed based on the classic findings of moderate-to-severe hyperthyroidism, the presence of a symmetrically enlarged thyroid gland, and recent onset of orbitopathy.[19] If this constellation of signs is absent, TRAb measurement, RAIU determination, or ultrasonography to measure thyroidal blood flow is recommended.[19] Either TRAb or TSI measurement are the recommended initial test for GD because of their excellent sensitivity (96%-97%) and specificity (99%), cost effectiveness, and timely return of results.[19] In our patient, GD diagnosis was based on the elevated TSI and TRAb levels and supported by the elevated RAIU and absent ESR. The IMMULITE 2000 TSI assay (Siemens Healthcare, Llanberis, UK) was used for all TSI measurements in this case. This is an automated chemiluminescent immunoassay that uses recombinant human TSH receptors to selectively detect stimulating TRAbs.[20] Receiver-operating characteristic analysis of this assay found that a threshold of 0.55 IU/L resulted in a sensitivity of 98.6% and specificity of 98.5%.[20] The Kronus TRAb enzyme-linked immunosorbent assay kit (Star, Idaho) was used for the semiquantitative determination of the TRAb level in this patient. The clinical sensitivity and specificity of this assay was 85% and 100%, respectively.



	Day 1	Day 7	Day 14	Day 17	Day 28	Day 35	Day 42	Day 49	Day 56	Day 63	Day 77	Day 79	Day 92	Day 108
TSH (mIU/mL)	0.03	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.02	0.1	<0.01	<0.01	<0.01	<0.01
TSI (<0.10 IU/L)	<0.10			<0.10							1.94		3.42	
TPO Ab (<9 IU/mL)	3												805	
TRAb (<2 IU/L)													8.72	
CRP (<0.5 mg/dL)			4.3		4.8						0.1			
ESR (<15 mm/hr)				21	17								4	
Prednisone (5 to 30 mg/day)		X	X	X	X	X	X	X			X			
Methimazole (10-15 mg/day)		X									X	X	X	X

Fig. Laboratory data and treatment over time. Normal reference ranges are in parentheses. *CRP* = C-reactive protein; *ESR* = erythrocyte sedimentation rate; *FT4* = free thyroxine; *TPO Ab* = thyroid peroxidase antibody; *TRAb* = TSH receptor autoantibody; *TSH* = thyroid-stimulating hormone; *TSI* = thyroid-stimulating immunoglobulin; *TT3* = total triiodothyronine.

We identified 14 publications reporting 31 cases of GD after SAT (Table).[2-15] Demographics, features used to diagnose SAT and GD, including antibody status, and genetic test results were compared. The criteria for SAT, including tender goiter and either elevated ESR and/or low RAIU, were documented in 29 of 31 cases (93.5%)[2-14]; histologic findings of SAT were reported in 4 of the 31 cases (13%).[4,10,15] GD diagnosis was supported by elevated RAIU in 26 of the 31 cases (84%), elevated TSI or TRAb level in 21 of the 31 cases (68%), and both in about 16 of the 31 cases (52%).[2,5-15] Two cases documented a change in the TRAb or TSI level from being absent at the onset of SAT to being elevated when GD was diagnosed.[8,12] Elevated TSI and/or TRAb levels after SAT, especially if prior levels are noted to be absent, as in our patient, provide support for thyroid damage and autoimmune activation, previously described as a cause of GD after SAT.[8] Elevated TRAb levels and GD have been reported to develop via a similar mechanism after radioactive iodine treatment for toxic nodular goiter.[8]

Genetic factors likely also play a role in GD after SAT. Bartalena et al[8] questioned why GD after SAT is so rare when thyroid damage and the release of thyroid autoantigens is considered a common feature in patients with SAT. They postulated that genetic susceptibility to thyroid autoimmunity, specifically positivity for HLA alleles associated with GD and SAT, exists for GD to occur after SAT.[8] Genetic factors, especially certain HLA alleles, have been estimated to be responsible for 79% of the liability of the development of GD.[17] Specific HLA alleles associated with GD have been reported in different ethnic populations: HLA-C*07, -DQA1*05:01, -DQB1*03:01, -DR B1*03,-DRB1*08 (White); HLA-B*35:01,-B*46:01, -DRB1*14:03, -DQB1*06:04, -DPB1*05:01 (Japanese); HLA-A*02:07, -A*11:01/02, -B*46:01, -C*01:02, -DPA1*02:02, -DPB1*05:01, -DQA1*02:01, -DQB1*02:01, -DQB1*05:02, -DRB1*07:01, -DRB1*15:01, and -DRB1*16:02 (Chinese).[16,17] HLA-B35 positivity has been reported in up to 70% of patients with SAT and 40% of SAT cases specifically in Chinese patients.[1,18] HLA-B*18:01, -DRB1*01, and -C*04:01 are also associated with SAT.[1] Our patient was positive for HLA-B35 and -DRB1*15:01 alleles, which are associated with SAT and GD, respectively. He was negative for additional HLA variants associated with GD (HLA-B*46:01, -DPB1*05:01, -DQB1*05:02, -DQB1*06:04, -DRB1*03:01, -DRB1*14:03, -and DRB1*16:02) and SAT (HLA-B*18:01 and -DRB1*01).[1,16,17] HLA testing was performed in 8 of the

Table
Graves Disease Following Subacute Thyroiditis: Summary of Case Report Data From the Literature. A Modification of the Table From Nakano et al[2]

Case	Author, year, country	Sex	Age (y)	Race	SAT to GD (mo)	Elevated ESR		24-h RAIU		Thyroid-directed auto-Ab		HLA typing
						SAT	GD	SAT	GD	SAT	GD	
1	Sheets,[3] 1955, USA[a]	F	37	White	7	Yes	…	12%	80%	…	…	No
2	Perloff,[4] 1956, USA[b]	M	19	White	0.5	Yes	…	…	…	…	…	No
3	…	F	43	Black	2	…	…	…	…	…	…	No
4	…	F	32	White	4	Yes	…	42%	84%	…	…	No
5	…	F	47	White	6	Yes	…	19%	45%	…	…	No
6	…	F	53	White	8		…	7%	35%	…	…	No
7	Werner,[5] 1979, USA	F	50	…	5	Yes	…	10%	56%	−TPOAb, −TGAb	−TPOAb, −TGAb, +TSI,	No
8	Wartofsky and Schaaf,[6] 1987, USA[c]	F	34	Black	3	…	…	6.3%	86.4%	+TPOAb, −TGAb	+ TSI	Yes[k]
9	…	F	34	White	8	Yes	…	13.4%	39%	−TPOAb, −TGAb	…	No
10	Fukata et al,[7] 1992, Japan	F	45	…	84	Yes	…	1.3%	62.7%	−TPOAb, −TGAb	+TPOAb, +TSI, +TBII, -TGAb	Yes[l]
11	…	F	60	…	96	Yes	…	1.2%	52.3%	−TPOAb, −TGAb	+TSI, +TBII	Yes[m]
12	Bartalena et al,[8] 1996, Italy	F	57	White	4	Yes	No	<1%	80%	−TPOAb, −TGAb, −TRAb	+ TPOAb, +TGAb, +TRAb	Yes[n]
13	Bennedbaek et al,[9] 1996, Denmark	F	49	…	6	Yes	…	No uptake[d]	Increased uptake[d]	…	+TPOAb, +TRAb	Yes[o]
14	Grunenberger et al,[10] 1998, France[e]	M	46	White	…	No	No	…	Increased uptake[d]	…	+TRAb	Yes[p]
15-20	Iitaka et al,[11] 1998, Japan[f]	1M/5F	50 ± 9	…	5 to 48	Yes	…	1%; 8%	28%; 53%	…	+TBII in 5 of 6, + TSI in 3 of 6, +TRAb in 1 of 6	Yes in 1 case[q]
21	Iitaka et al,[12] 2001, Japan	M	45	…	18	Yes	…	3.1%	53%	−TPOAb, −TGAb, +TBII, −TSI	+TSI, −TBII	No
22	Wang and Renedo,[13] 2004, USA	F	56	…	12	…	…	2%	55%	…	−TPOAb, −TGAb, −TRAb	No
23	Nakano et al,[2] 2011, Japan	M	43	…	5	Yes	No	…	65%	…	+TRAb	No
24	…	F	44	…	2	Yes	No	…	72%	…	+TRAb, +TSI	No
25	…	F	49	…	6	Yes	No	…	61.2%	…	+TRAb	No
26	…	F	40	…	5	Yes	No			…	+TRAb	No
27	…	F	31	…	6	Yes	No	…	49.7%	+TRAb, −TPOAb, −TGAb[g]	+TSI, +TRAb, −TPOAb, −TGAb	No
28	…	F	59	…	8	Yes	No	…	40.8%	−TPOAb, +TGAb, +TRAb, +TSI[h]	+TRAb	No
29	…	F	66	…	1	Yes	No	…	59.4%	−TPOAb, +TRAb[i]	+TRAb, +TSI	No

(continued on next page)

Table (*continued*)

Case	Author, year, country	Sex	Age (y)	Race	SAT to GD (mo)	Elevated ESR		24-h RAIU		Thyroid-directed auto-Ab		HLA typing
						SAT	GD	SAT	GD	SAT	GD	
30	Dow et al,[14] 2014, USA	F	79	…	2 to 2.5	Yes	No	…	…	…	+TPOAb, +TSI	No
31	Hallengren et al,[15] 2015, Sweden[j]	F	43	…	142	…	…	…	…	…	+TRAb	Yes[r]

Abbreviations: Ab = antibodies; GD = Graves disease; ESR = erythrocyte sedimentation rate; HLA = human leukocyte antigen; RAIU = radioactive iodine uptake; SAT = subacute thyroiditis; TBII = thyrotropin-binding inhibitory immunoglobulin; TGAb = thyroglobulin antibody; TPOAb = thyroid peroxidase antibody; TRAb = TSH receptor autoantibody; TSI = thyroid-stimulating immunoglobulin; USA = United States of America.

[a] This patient received 2 courses of x-ray therapy for thyroiditis, which might have contributed to thyroid damage and the activation of GD.

[b] In 2 of the 5 cases (a 19-year-old man and a 43-year-old woman), SAT diagnosis was based on a painful and tender thyroid (both of the cases) and elevated ESR in case 2. A presumptive diagnosis of GD was made when hyperthyroidism persisted and worsened, leading to the decision to remove the thyroid in both of the cases. However, surgical pathology revealed SAT in both of the cases.

[c] The first case (a 34-year-old Black woman) had GD in remission with propylthiouracil 6 years before developing SAT and then subsequent GD. The development of GD might have been due, in part, to the relapse of pre-existing autoimmune thyroid disease rather than fully related to preceding SAT.

[d] Technetium pertechnetate was taken up by the thyroid in these cases.

[e] This patient had GD in remission with antithyroid medication. The subsequent relapse of GD 5 years later was managed surgically. TRAb was present at the initial and subsequent presentation with GD. The diagnosis of SAT was based only on a histologic examination, which revealed remnants of colloid surrounded by rings of histiocytes and giant cells scattered over fibrotic and inflammatory parenchyma in the right thyroid lobe.

[f] Iitaka et al described 38 patients with SAT who developed TRAb positivity.[11] The patients were divided into groups based on when the antibodies became present and by thyroid function. The subset of patients who had persistent hyperthyroidism was categorized in a different group compared with those in whom hyperthyroidism developed after complete recovery from SAT because the 2 groups were considered to have different underlying immunologic mechanisms. Only the 6 patients who developed hyperthyroidism after complete resolution of SAT were included here.

[g] The TRAb level was checked not at the onset of SAT, but at the time of resolution of SAT, 50 days after the onset of SAT.

[h] The TRAb and TSI levels were checked not at the onset of SAT, but at the time of resolution of the first episode of SAT, 5 months after the onset of SAT.

[i] There was overlap in the diagnosis of SAT and GD, with GD being diagnosed 20 days after SAT diagnosis and hyperthyroidism persisting despite prednisone. Simultaneous SAT and GD is a possible explanation for the presence of TRAb during SAT diagnosis.

[j] The diagnosis of SAT in this patient was verified using fine-needle aspiration and a cytologic examination.

[k] HLA typing demonstrated the presence of A28, 30; B35; Cw4, DR 4, DQ 3, DRw 52, 53.

[l] HLA typing demonstrated the presence of A24; B35, BW46; CW11; DRW8.

[m] HLA typing demonstrated the presence of A11, A26; BW54; BW67; CW1, CW7; DRW15 (DR2), DR4, DRW53.

[n] HLA typing demonstrated the presence of A24, A29; B35, B44; DR3, DR11, DR52, DQ2, DQ7.

[o] HLA typing demonstrated the presence of A1, A24; B8, B35; DR-B1*03,11; DR52.

[p] HLA typing demonstrated the presence of A2, A26; B18, B27; DR3, DR52.

[q] HLA typing demonstrated the presence of B35.

[r] HLA typing demonstrated the presence of HLA-B*35 and -DRB1*03.

31 reported cases (26%) of GD after SAT.[6-11,15] Of these 8 patients, 6 were positive for HLA-B35 and 4 were positive for HLA alleles associated with GD (HLA-BW46, -DRB1*03, and -DR3).[6-9,11,15-17] Currently, there are inadequate data to determine the association between certain HLA profiles and the risk of GD after SAT, and genetic testing is not recommended as part of the evaluation of hyperthyroidism[19]; however, more HLA testing in these rare cases may reveal specific correlations.

With regard to demographics, 26 of the 31 reported cases (84%) were women over the age of 40 years, similar to what has been seen in cases in which GD or SAT occurs alone.[1-9,11,13-15] Race was not documented in all the cases, but reports originated from many different countries. We did not find documentation of the Chinese race in any of the reported cases or any reports originating from China. To our knowledge, ours is the first reported case of GD after SAT in a Chinese man.

Conclusion

This case of SAT followed by GD in a Chinese man with HLA positivity for HLA-B35 and -DRB1*15:01 highlights the utility of checking the TSI and/or TRAb levels to diagnose GD in cases of recurrent hyperthyroidism after SAT. Elevated TSI and/or TRAb levels after SAT support autoimmune thyroid activation by SAT as a mechanism of GD development. Genetic factors, specifically the presence of HLA alleles associated with GD or SAT, may increase susceptibility to the development of thyroid autoimmunity after SAT; however, because of the rarity of these cases and inadequate HLA data, a correlation between this risk and specific HLA alleles cannot be made. This case adds to the few reported cases of this rare occurrence in men, provides support for the use of TSI and/or TRAb testing to detect the autoimmune activation of the thyroid and GD after SAT, and adds to the literature on HLA alleles detected in these rare cases.

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Author Contributions

S.A. researched the data, contributed to writing the manuscript, and reviewed the manuscript. S.W.L. researched the data, wrote the manuscript, and reviewed and edited the manuscript. S.W.L. is the guarantor of this work and, as such, had full access to all the sections of the manuscript and references and takes responsibility for the integrity and accuracy of this paper.

Disclosure

The authors have no multiplicity of interest to disclose.

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