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Subacute thyroiditis: a rare presentation in pregnancy

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A 35-year-old pregnant woman, in the 12th week of gestation, was admitted to our outpatient clinic with a complaint of neck pain that had been ongoing for 2 months. She had no known history of thyroid disease or thyroid medication use and had no history of fever or recent upper respiratory tract infection. Her blood pressure was 100/70 mm Hg on physical examination, and her heart rate was 82/min. The thyroid gland was tender and enlarged on neck palpation. Laboratory examination revealed thyrotoxicosis with a thyroid-stimulating hormone (TSH): 0.001 mIU/ml (reference range, 0.34–5.60 mIU/ml), free thyroxine (fT4): 29.34 pmol/L (reference range, 7.71–16.09 pmol/L), and free triiodothyronine (fT3): 7.90 pmol/L (reference range, 3.98–6.94 pmol/L). Antithyroid autoantibodies were negative [thyroid peroxidase antibodies (anti-TPO): < 1 IU/mL (reference range, 0–9 IU/mL), thyroglobulin antibodies (anti-Tg): < 1 IU/ml (reference range, 0–4 IU/mL), TSH receptor antibodies (TRAB): 0.3 IU/L (reference range, 0–1.58 IU/L)]. White blood cell (WBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) values indicating inflammation were high [WBC: $13.10^3/\mu L$, neutrophil/lymphocyte/monocyte %: 80.6/12.3/6.5%, ESR: 45 mm/h (reference range, 0–15 mm/h), CRP: 62.7 mg/L (reference range, 0–3.5 mg/L])] (Tab. 1). Heterogeneous hypoechoic areas and pseudo nodule formations were observed in both thyroid lobes in thyroid ultrasonography (US). Doppler US showed decreased blood flow in the thyroid parenchyma.

Since clinical, laboratory, and imaging findings were compatible with subacute thyroiditis (SAT), so nonsteroidal anti-inflammatory treatment (NSAIDs) (ibuprofen 1200 mg) was initiated. Two weeks after the initiation of treatment, the patient's complaints significantly decreased, and the treatment was continued for 5 weeks. At the 18th week of pregnancy, TSH: 9.81 mIU/mL, fT4: 5.66 pmol/L, fT3: 2.61 pmol/L, and hypothyroidism developed. Levothyroxine (50 mcg/day) was started, and the dose was adjusted according to the results of thyroid function tests. The patient gave birth to a healthy baby at term. No adverse outcome was observed during pregnancy or after delivery. Control thyroid US performed one year after admission showed that the thyroid parenchyma was homogeneous, and the thyroid gland volume was significantly reduced. At the three-year follow-up, permanent hypothyroidism was confirmed, the patient's current levothyroxine requirement was 100 mcg/day, and further follow-up was planned at 6-month intervals.

Recognition of thyroid dysfunction during pregnancy is very important for maternal and foetal health. Depending on the severity, hyperthyroidism and hypothyroidism can lead to many complications such as spontaneous abortion, stillbirth, premature birth, low birth weight, congenital anomalies, neurocognitive deficits, maternal hypertension, heart failure, and preeclampsia. Overt hyperthyroidism is a rare condition seen in 0.1–0.4% of all pregnancies [1].

SAT is a transient, self-limiting, and inflammatory thyroid disease. It usually presents with transient hyperthyroidism. Neck pain and tenderness are the most common symptoms which may be accompanied by fever and thyrotoxicosis symptoms (palpitations, sweating, irritability, etc.) like non-specific complaints during pregnancy. Inflammatory markers increase in the early stages of the disease. Although the aetiology is unknown, it is thought to be caused by previous viral infections. On ultrasonography (US) imaging of the SAT, unilateral or bilateral enlargement and hypoechoic and heterogeneous areas are typical. Doppler US shows decreased blood flow in the affected parenchyma. Depending on the destruction of the thyroid tissue after SAT, transient or permanent hypothyroidism may de-

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Table 1. *Laboratory findings of the patient during pregnancy and after delivery*

	TSH [mIU/mL]	fT4 [pmol/L]	fT3 [pmol/L]	Anti-Tg [IU/mL]	Anti-TPO [IU/mL]	TRAB [IU/L]	ESR [mm/h]	CRP [mg/L]	WBC [10^3 μL]	PLT [10^3 μL]	Treatment
Reference range	0.34–5.60	7.71–16.09	3.98–6.94	0–4	0–9	0–1.58	0–15	0–3,5	4.8–10.8	130–400	
12th week of gestation	0.001	29.34	7.90	< 1	< 1	0.3	45	62.7	13	417	Ibuprofen 2 × 600 mg
13th week	0.003	25.75	6.89					29.9	10.3	459	Ibuprofen 2 × 600 mg
16th week	0.003	13.55	4.27					12.8	11.3	421	İbuprofen discontinued
18th week	9.81	5.66	2.61					5.77	11.8	429	LT4 50 mcg
22nd week	5.58	8.39	4.14								LT4 100 mcg
30th week	1.22	11.01	4.63								LT4 100 mcg
38th week	2.02	9.09	4.23								LT4 100 mcg
Postpartum 2nd month	1.17	11.91	4.51	3.9	< 1						LT4 100 mcg
Postpartum 1st year	1.73	18.10	4.72								LT4 100 (5 d) 75 mcg (2 d)
Postpartum 2nd year	1.75	18.4	4.64								LT4 100 (5 d) 75 mcg (2 d)
Postpartum 3rd year	2.08	16.22	4.26				15		8.4	440	LT4 100 (5 d) 75 mcg (2 d)

TSH — thyroid-stimulating hormone; fT4 — free thyroxine; fT3 — free triiodothyronine; anti-Tg — thyroglobulin antibodies; anti-TPO — thyroid peroxidase antibodies; TRAB — TSH receptor antibodies; WBC — white blood cell; ESR — erythrocyte sedimentation rate; CRP — C-reactive protein; PLT — platelets; LT4 — levothyroxine

velop. Inflammatory markers and US findings improve after the active phase of the disease [2].

Treatment of SAT is symptom relief with anti-inflammatory drugs and monitoring of thyroid function tests. Antithyroid drugs have no place in treatment. Based on the clinician's experience, NSAIDs are administered for mildly symptomatic patients and corticosteroids for those with moderate to severe complaints [3].

SAT is extremely rare in pregnancy and usually occurs in the first trimester or early second trimester. To our knowledge, 8 cases of SAT occurring during pregnancy have been reported to date [4]. The first case was reported by Hiraiwa et al. in 2006 [5]. The literature reported that 4 cases were treated with corticosteroids, one was treated with paracetamol and the others were followed up without treatment. Although levothyroxine treatment was initiated in 6 of these cases, permanent hypothyroidism was not reported. There was no information about postnatal thyroid replacement requirements in 3 of the cases [4]. The current case is the first in the literature in which permanent hypothyroidism developed after SAT in pregnancy.

In conclusion, SAT, which is a rare cause of hyperthyroidism in pregnancy, should be kept in mind in terms of maternal and foetal health due to its different treatment approaches and potential to develop hypothyroidism.

Ethics statement

The research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Data were collected retrospectively.

Author contributions

N.G, — data collection, article writing, revision.

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Conflict of interest

The author declares no conflict of interest.

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