

Painful Thyroiditis in Postpartum Period

S.O. Sakinah, MRCP*

N.A. Sharifah, DCP**

K. Yusoff, MRCP *

* Department of Medicine, Universiti Kebangsaan Malaysia, Kuala Lumpur

** Department of Pathology, Universiti Kebangsaan Malaysia, Kuala Lumpur

Summary

Destructive thyroiditis commonly occurs during the postpartum period, with a prevalence rate of 5% to 16%, and is mainly due to postpartum autoimmune thyroiditis (PPT) and, very rarely, to subacute thyroiditis. The thyroiditis is similar to Hashimoto's thyroiditis and is generally painless in nature, although cases with painful thyroiditis have been described.

We report a case of painful destructive thyroiditis occurring during the postpartum period, which was clinically and biochemically indistinguishable from the variant of painful PPT or subacute thyroiditis. Fine needle aspiration cytology showed multi-nucleated giant cells diagnostic of subacute thyroiditis.

Key words: Thyroiditis, postpartum.

Introduction

Thyroid function abnormalities occurring during the postpartum period is now recognised as a common condition. Several epidemiological studies from all over the world reported the prevalence of 2% to 16%¹. The thyroid function abnormalities observed include thyrotoxicosis, hypothyroidism or, classically, hyperthyroidism followed by hypothyroidism. More than 90% of cases with thyroid dysfunction were due to postpartum thyroiditis, characterised by low radioactive iodine uptake thyrotoxicosis followed by hypothyroidism. It is transient in nature in the majority of cases. It is autoimmune in origin, as thyroid antibodies were detected in most of the cases and pathological examination of the thyroid revealed lymphocytic infiltration¹. Classical PPT is painless in nature, similar to silent thyroiditis in patients who are not in postpartum period. However, as in Hashimoto's thyroiditis, PPT is not always painless. Cases of PPT presenting with painful thyroid have been reported², which makes it more difficult to differentiate from subacute thyroiditis.

We report a case presenting with classical painful destructive thyroiditis during the postpartum period, which was clinically and biochemically indistinguishable from a variant of painful PPT or subacute thyroiditis. However, fine needle aspiration offered a definitive diagnosis.

Case Report

A 34 year old Malay woman with mild mitral valve prolapse had forceps delivery of her first healthy infant in December 1991, under antibiotic cover. At 4 weeks postpartum, she started to experience generalised backache and increased body heat. At the same time she noticed a goitre which was tender and associated with mild pain during swallowing. She gradually developed nervousness, mild palpitations, tiredness and loss of weight. She

CASE REPORT

was seen by several practitioners who prescribed antibiotics with no improvement in her symptoms. At 20 weeks postpartum, she was seen at our unit for persistence of the above symptoms.

On examination, she was thin and her temperature was 37.5°C. There was no stigmata of bacterial endocarditis and no tremors. There was sinus tachycardia, but the reflexes were normal and there was no exophthalmos, lid lag or lid retraction. She had a moderate-sized diffuse goitre which was tender. There was no cervical lymphadenopathy and the throat and tonsils were not inflamed. There was a soft mid-systolic murmur at the apical area.

Investigations revealed no leukocytosis and differential counts were normal. Erythrocyte sedimentation rate was 20 mm per hour. The viral antibody titre by complement fixation test for measles, mumps, varicella zoster, herpes simplex and cytomegalovirus were low and there was no rise in the titre repeated 2 weeks later. The thyroid antibodies for both microsomal and thyroglobulin were negative. Repeated blood cultures were negative and echocardiographic examination showed mild mitral valve prolapse without vegetations. Serum total thyroxine (T4) was 206 nmol/l (normal range: 67-167) and thyroid stimulating hormone (TSH) was <0.5 μ IU/ml (normal range: <3.5). Radioactive iodine scan showed a diffuse low uptake. She was treated with beta-blockers and aspirin at this stage.

She was seen again at 22 weeks postpartum, where she felt much better and the goitre became less tender. The serum thyroxine was 107 nmol/l. At 28 weeks postpartum, she complained of constipation for a week, had insomnia and depression and became easily tearful. Pulse rate was 60 per min and regular. There was a diffuse goitre which was less tender. Her serum T4 was 48 nmol/l and TSH was 11.2 μ IU/ml. Her thyroid antibody was negative. Fine needle aspiration was performed and the findings are shown in Fig 1. At this stage all medication was discontinued.

Review at 36 weeks postpartum showed that she was well and asymptomatic. There was a small, firm goitre which was nontender. The serum T4 was 83 nmol/l and TSH was 3.03 μ IU/ml.

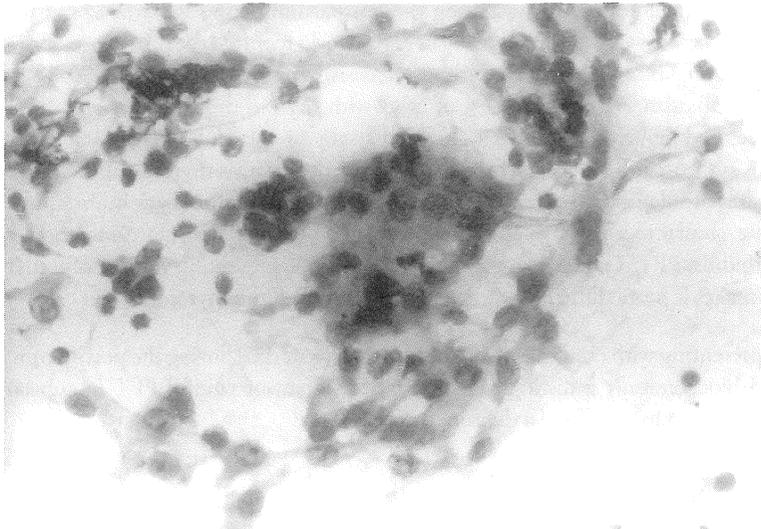


Fig 1: Smears showing 2 multi-nucleated giant cells with scattered inflammatory cells in the background. Papinicolaou stain x 400.

Discussion

This patient presented with classical features of postpartum destructive thyroiditis, namely low radioactive iodine uptake and a transient evolution of symptomatic thyroid dysfunction, i.e., thyrotoxicosis, followed by euthyroid, hypothyroid and subsequent recovery in the thyroid function.

The most common cause of this disorder during the postpartum period is PPT¹. This commonly presents with thyrotoxicosis at around 4 to 12 weeks and a later hypothyroid phase at 20 to 28 weeks postpartum. Even though it is commonly a silent thyroiditis, similar to Hashimoto's thyroiditis, cases with painful thyroid have been reported². It is possible that this patient had a variant of painful PPT, though her thyroid antibodies were negative. A high percentage of cases with PPT, particularly in those who presented with thyrotoxicosis alone, had no demonstrable thyroid antibodies in their sera¹. This probably reflects reduced sensitivity of the techniques used to measure the thyroid antibodies. Alternatively, the negative thyroid antibody in PPT cases observed in their study could be due to localised intrathyroidal antibody production, which has been observed in Hashimoto's thyroiditis. Alternatively, this patient may have had subacute thyroiditis even though it is rarely seen during this period. Unlike typical thyroiditis, this case was not associated with fever, increased sedimentation rate and leukocyte count or rise in the viral antibody titre.

Fine needle aspiration performed in this case showed the presence of multi-nucleated giant cells, which is diagnostic of subacute thyroiditis. This is in contrast to PPT, where pathological examination of thyroid usually reveals lymphocytic infiltration and absence of multi-nucleated giant cells¹.

In retrospect, when comparing the case of painful PPT described by Othman *et al*², the onset of pain during the course of thyroid dysfunction could have provided a clue in differentiating between PPT or subacute thyroiditis. In that case, the onset of painful thyroid was not during the onset of the illness (during the onset of thyrotoxicosis) but it coincided with the hypothyroid phase. It was postulated that the pain was due to capsular stretching by rapid enlargement of an already enlarged gland under the influence of TSH. In our case, the painful thyroid occurred at the onset of illness, coinciding with the thyrotoxic phase. This suggests that the inflammatory pathological processes underlying the destructive process were different in the 2 disorders.

It is interesting to note that there was a swing in mood in this patient in relation to thyroid function abnormalities. During the hypothyroid phase she became depressed and more tearful. Similar observations were noted in patients with PPT¹. Othman *et al* found that the depressive mood during the postpartum was not only common in those with abnormal thyroid function, but also in those with positive thyroid antibodies³. Both factors probably affect the mood independently, in susceptible patients, during the postpartum period.

In view of the mild and transient nature of destructive thyroiditis, treatment has been often limited to reassurance and observation. The symptomatic thyrotoxic state can be treated with beta-blockers. In the few patients who develop symptomatic hypothyroidism, it may be necessary to initiate L-thyroxine therapy.

Finally, even though PPT is the commonest thyroid disorder occurring during the postpartum period, we suggest that fine needle aspiration should be performed in cases with unusual presentation, as it provides a definitive diagnosis.

References

1. Lazarus JH and Sakinah SO. Thyroid disease in relation to pregnancy. *Clin Endocrinol* 1991;34: 91-8.
2. Othman S, Parkes AB, Richards CJ, Hall R, Lazarus JH. Postpartum thyroiditis can be painful. *Postgrad Med J* 1990;66: 130-1.
3. Othman S, Harris B, Richards C, Hall R, Lazarus JH. Postpartum thyroiditis is associated with psychiatric illness. *Progress in Thyroid Research*. Gordon A, Gross J and Henneman G (eds) 1991;189-92.