Post-partum thyroiditis can be painful

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Summary: A patient with post-partum thyroiditis is described. She was a 22 year old with a negative family history of autoimmune thyroid disease who was noted to have a high titre of antithyroid microsomal antibody during pregnancy. She developed mild hyperthyroidism 8 weeks post-partum but at 12 weeks she had a mildly painful enlarged thyroid gland. At 20 weeks post-partum she had severe thyroidal pain with dysphagia. The thyroid was exquisitely tender to palpation. She was treated with L-thyroxine and the pain resolved within 4 weeks. This is the first report documenting pain in the thyroid as a feature of post-partum thyroiditis.

Introduction

Post-partum thyroiditis (PPT) characterized by transient asymptomatic hyperthyroidism followed by symptomatic hypothyroidism occurs in up to 16% of normal women.¹ It is associated with the presence of antimicrosomal thyroid antibodies and abnormal thyroid function tests. Clinically the patient may develop a goitre but, unlike subacute thyroiditis, pain in the thyroid has not been previously described. We describe, for the first time, a patient with PPT with associated pain in the thyroid who was being followed post-partum in the Caerphilly Pregnancy Study.

Case report

A 22 year old Welsh woman, para 1 + 0, a moderate smoker, delivered a normal female infant in December 1987. Both her parents had undergone thyroidectomy in the past for non-toxic goitre but there was no family history of autoimmune thyroid disease. At delivery she had a microsomal antibody (mic Ab) level of 3890 U/ml (normal less than 525 U/ml) and a thyroglobulin antibody level of 2080 U/ml (normal less than 250 U/ml) as measured by ELISA. Free thyroxine (FT4), free triiodothyronine (FT3), and thyroid stimulating hormone (TSH) were measured by Amerlex assays (Amersham International plc). Normal ranges are 8–26 pmol/l, 3–9 pmol/l and less than 5 mU/l respectively. Her subsequent course during the next 10 months is shown in Figure 1.

Figure 1 Thyroid tests at delivery and post-partum. Changes in FT3 (normal range: 3–9 pmol/l), FT4 (normal range 8–26 pmol/l), TSH (normal range < 5 mU/l) and mic Ab (normal range < 525 U/ml) from delivery to 40 weeks post-partum. —▲— FT3; —●— FT4; —○— TSH; —□— micAb. *¹²³I uptake 4 hours: 35%, 48 hours: 42%.

At 4 weeks post-partum, she was clinically and biochemically euthyroid with a moderate, firm goitre. One month later, she was clinically euthyroid but biochemically there was evidence of mild thyrotoxicosis; however, there was no change in goitre size.

At 12 weeks post-partum, she felt depressed and tired and became very forgetful. She had a mildly painful thyroid but no clinical abnormality was

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found by her general practitioner. However, her TSH was elevated at 11.2 mU/l. Thyroidal ^{123}I uptakes were 35% at 4 hours and 42% at 48 hours (normal) but PB ^{123}I was elevated at 1.01% dose per litre of plasma (normal less than 0.4%), consistent with a diagnosis of thyroiditis.

Five months after delivery, she complained of dysphagia for fluids and solids. She had severe pain in the neck radiating to the jaw and there was a 2 kg weight loss over a 2-week period. On examination, she was afebrile but had a firm moderate goitre which was exquisitely tender to palpation. There was no lymphadenopathy or evidence of throat infection. ESR was 40 mm/h, adenovirus titre 1/128, total white count $5.4 \times 10^9/\text{l}$. She refused a fine needle biopsy of the thyroid and treatment with L-thyroxine 0.1 mg was commenced.

Four weeks later she felt much better, the thyroidal pain improving after a few days and being substantially relieved within 2 weeks of starting thyroxine. There was no dysphagia and she had regained her weight. There was a slight reduction in goitre size but a dramatic improvement in tenderness of the thyroid on palpation. The adenovirus titre was 1 in 16.

At 46 weeks post-partum she was asymptomatic with a marked reduction of goitre size. L-Thyroxine was continued for a further 3 months.

Discussion

Dysphagia and a painful thyroid have not previously been reported in PPT. It is unlikely that this patient had subacute thyroiditis as the clinical

features are inconsistent with such a diagnosis. She presented with a painful thyroid when the TSH level was elevated whereas in subacute thyroiditis the pain is usually associated with frank hyperthyroidism.² The patient's ^{123}I uptakes were normal at the time of the pain in her thyroid unlike the typical suppressed uptake in subacute thyroiditis. The presence of viral antibody may merely indicate an anamnestic response to thyroid inflammation³ or be just a coincidence. The presence of thyroid antibodies at delivery which became progressively elevated reaching a maximum at the peak of the TSH level strongly suggests that this condition is autoimmune in origin.

The cause of the painful thyroid in this case is not known but it may have been due to capsular stretching by rapid enlargement of the already enlarged gland under the influence of TSH. Although there was no discernible change in thyroid size, the presence of dysphagia may indicate that there was posterior enlargement of the gland which would not have been detected clinically. Measurement of thyroid volume by ultrasound would have been of value.

While treating this case we have observed two other patients with PPT associated with pain in the thyroid gland. Thus the occurrence of a painful thyroid in the post-partum period may be a presenting feature of PPT but subacute thyroiditis should of course be excluded. These cases stress the similarity with Hashimoto's disease which may also rarely present with a painful thyroid.⁴ Treatment with thyroxine is indicated for amelioration of pain and maintenance of euthyroidism.

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