Medical Images

A 17-year-old with Goiter

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Figure 1 depicts a goiter in a 17-year-old girl presenting to the pediatric clinic for follow up of hypothyroidism. The patient presented with fatigue 3 months postpartum and was found to have an enlarged and slightly tender thyroid gland. A markedly elevated thyroid stimulating hormone (TSH) level of 110.544 uIU/mL and low free thyroxine (fT4) concentration of 0.3 ng/dL revealed hypothyroidism. Patient was started on levothyroxine 112 mcg daily. Additional testing revealed the presence of antithyroglobulin and antithyroid peroxidase (anti-TPO) antibodies. This image was taken one month after beginning treatment.

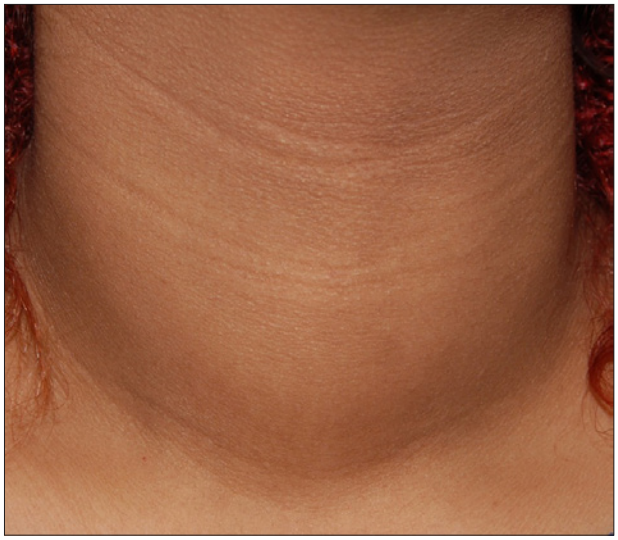

Figure 1

Autoimmune thyroid disease is common in young women. It is often asymptomatic. Positive anti-thyroglobulin antibodies and anti-TPO antibodies are common in young women (many of whom will never develop clinical thyroid disease).

As this case illustrates, the postpartum state is an interesting interval in which autoimmune thyroid disease may first present itself. The pathophysiology is related to the normal immune suppression that occurs during pregnancy to prevent rejection of the fetus and the subsequent rebound immune hyperactivity during the postpartum period. This process is thought to unmask subclinical thyroid dysfunction and aggravate autoimmune disease in at-risk women.[1] In HASHIMOTO'S DISEASE (the likely diagnosis here), patients can present with goiter and hypothyroidism, with symptoms that might be confused with postpartum depression. Patients with GRAVES' DISEASE [whose autoantibody repertoire includes thyroid-stimulating antibodies in addition to the anti-TPO and anti-thyroglobulin antibodies of Hashimoto's] can be in remission during pregnancy, only to flare after delivery of the baby. POSTPARTUM THYROIDITIS (also called SILENT THYROIDITIS due to the lack of severe pain that is seen with post-viral subacute thyroiditis) is a third variation of postpartum thyroid dysfunction. Its course is multi-phasic; autoimmune tissue destruction releases preformed thyroid hormone and results in hyperthyroidism during the first several postpartum months, often followed by a period of hypothyroidism which may be transient or permanent.[2-4] By definition, thyroid dysfunction develops within one year postpartum.[3] The symptomatology of either hyperthyroidism or hypothyroidism can be difficult to distinguish from symptoms associated with common postpartum stressors.[5] Missed diagnosis can be especially detrimental if the patient were to conceive again, as hypothyroidism is associated with an increased risk for a myriad of pregnancy complications and can result in significant morbidity in the fetus.[3,6] As such, clinicians should maintain a high index of suspicion and universal screening of thyroid function during pregnancy has been proposed.[7] Treatment of hypothyroidism entails daily levothyroxine dosed to therapeutic levels based on TSH normalization. Once a physiological replacement dose is reached, TSH should be checked annually.[8]

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