Maternal Postpartum Thyroiditis as Infant Growth Failure

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Failure to thrive is a descriptive term referring to the reduction of growth of infants from an established growth pattern curve or to a weight consistently below the third percentile. Only about 25 percent of failure to thrive cases are purely organic, the other 75 percent are due to multiple problems including nutrition and psychosocial factors.[1] Because the differential diagnosis and evaluation often focus on underlying infant illness or neglect issues, the workup can involve costly laboratory studies and lengthy hospitalizations.[2,3] The number of cases of failure to thrive that are due to maternal illness is unknown and is seldom included in the differential diagnosis. Postpartum thyroiditis is one example of a common maternal illness that can adversely affect a mother's ability to care for her child.

Thyroid disease affects 5 to 15 percent of women of reproductive age, and 5 to 9 percent of postpartum women experience thyroid dysfunction, most of which is transient.[4,5] Postpartum thyroiditis can cause hyperthyroidism or hypothyroidism and is part of a spectrum that includes other autoimmune thyroid diseases, such as Hashimoto thyroiditis.[6] This common problem is often missed because of patients' nonspecific complaints and lack of thyroid gland discomfort.[4,7,8] Of major concern in postpartum thyroiditis is the high incidence of psychiatric disturbances, including problems with cognition, memory, and affect.[9] We describe a case in which a mother's postpartum thyroid dysfunction contributed to her infant's failure to thrive. Important features of maternal postpartum thyroiditis and its potential impact on infant well-being are discussed.

Submitted, revised, 24 April 1996.

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Case Report

A male infant was born at 37 weeks' gestation by spontaneous vaginal delivery to a 32-year-old mother of 2 children after an uneventful pregnancy. Birth weight was 6 pounds, 12 ounces (3085 g). No prenatal risk factors were noted. The infant was found to be hypotonic at birth, but did well in the neonatal period and was discharged home at 3 days of age. He was readmitted at 5 days old for hyperbilirubinemia and weight loss while being breast-fed. Infectious and hematologic causes were ruled out, and the hyperbilirubinemia (bilirubin 20.7 mg/dL indirect, normal 0.1-0.9 mg/dL) resolved with phototherapy. Lactation consultation was provided by an experienced nurse lactation specialist. Evaluation included an extensive breast-feeding history, physical examination of mother and infant, and observation of breast-feeding. Diagnosis was made of lactation failure secondary to insufficient glandular development of the breast[10] based on the physical examination and the minimal amount of milk that was expressed. The infant was also believed to have a subjectively poor suck-swallow reflex.

Because the mother wished to continue breast-feeding, a management plan, including formula supplementation using a breast-feeding assistance device (supplemental nursing system; Medela, Crystal Lake, Ill), frequent visits with the lactation consultant, and close physician follow-up, was initiated. The mother at this time appeared to be very motivated, able to focus on the infant's needs, and remember fairly complex recommendations regarding use of the supplemental breast-feeding device.

After discharge the infant had very slow weight

gain without much catch-up growth while breast-feeding with supplemental formula until he attained a weight of 7 pounds, 3 ounces (3318 g) at 6 weeks of age. The mother abruptly stopped supplemental feeding without the knowledge of the physician or lactation specialist, and when seen at 8 weeks of age, the infant's weight had dropped to 6 pounds, 14 ounces (2727 g). At that time the infant was severely dehydrated and minimally responsive with poor muscle tone.

The infant was admitted to the hospital for evaluation and monitored supplemental feedings. Noteworthy laboratory findings included mildly elevated ammonia (141 µg/dL, normal 3-31 µg/dL) and increased liver function tests, all of which resolved with monitored formula supplementation. Infant thyroid function tests were normal. Cultures of blood and urine were negative. Titers obtained to rule out common in utero infections included antibody screening for toxoplasma, rubella, cytomegalovirus, and herpes simplex (TORCH). The mild elevation in ammonia also led to a metabolic evaluation, which included both urine and plasma metabolic screening tests to rule out common inborn errors of metabolism. Both the TORCH and the metabolic screening results were normal.

During this hospitalization the nursing staff noted the mother had unusual difficulty with simple instructions, such as saving diapers to be weighed. She frequently asked to have information repeated, such as when the physician could be expected, and had to be reminded where supplies were being kept. Initially this unusual behavior was perceived as being in response to the stress of being in the hospital. Upon discussion with the mother's family and her lactation consultant, however, we found the mother's concentration difficulties preceded this hospitalization and coincided with her stopping the use of her breast-feeding appliance. The mother at this time was thin and anxious; she complained of feeling very tired and admitted to having a hard time with her memory. She denied feeling sad, hopeless, or depressed.

When examined, she had a persistently elevated heart rate of 105 to 110 beats per minute. Results of initial thyroid studies obtained on the mother while the infant was hospitalized (2 months postpartum) were free thyroxine (T_4) 1.98 ng/dL (normal 0.71 to 1.85 ng/dL) and thyroid-stimulating hormone (TSH) 0.04 µU/mL (normal 0.3 to 5.0 µU/mL). Follow-up studies on the mother obtained after discharge (3 months postpartum) were consistent with hypothyroidism, with a T_4 of 2.4 µg/dL (normal 4.5 to 12.0 µg/dL) and TSH of 10.19 µg/mL (normal 0.5 to 5.1 µg/mL). Total T_4 instead of free T_4 was obtained during a follow-up visit in our outpatient clinic because it was less expensive. Antibody titers also obtained at this follow-up visit were positive for antimicrosomal antibodies at 1:400 and for antithyroglobulin antibodies at 1:640.

Thyroid replacement was prescribed for severe symptoms of impaired concentration and memory as well as generalized fatigue. Subsequently, dramatic improvement was noted in the mother's ability to care for her infant, comply with feeding regimens, and participate in his physical therapy. Lactation remained impaired, however, and the infant remained on formula. The mother continued to require T_4 replacement at 1 year postpartum. The infant was receiving physical therapy for gross motor delay. The cause of his motor problem was still being investigated but did not appear to be related to his postnatal problems. His thyroid functions have remained normal.

Discussion

Postpartum thyroiditis is an acute, painless, destructive inflammation of the maternal thyroid gland[6,7] associated with high titers of microsomal antibodies.[9,11] During the early phase, (6 weeks to 4 months postpartum) there can be a transient hyperthyroid period. The subsequent hypothyroid state occurs 2 to 6 months after delivery and generally resolves by 1 year.[4,6,7] During the hyperthyroid period in non-breast-feeding mothers, postpartum thyroiditis can be differentiated from Graves' disease by the low radioactive iodine uptake.[12] Invasive studies such as fine-needle aspiration are not indicated unless a thyroid nodule is noted on physical examination. The diagnosis during the hypothyroid period is usually based simply on low T_4 levels, with elevated TSH levels during the postpartum period associated with elevated antithyroid antibody titers. Painless thyroid enlargement might or might not occur, and symptoms are vague, so that clinicians need to be alert to thyroid dysfunction when caring for the postpartum patient.[8,12]

Psychiatric complaints are more common in

postpartum thyroiditis than in classic hypothyroidism, and there is evidence that thyroid dysfunction might be associated with an increased risk of postpartum depression.[13] In one study, increases in total complaints, carelessness, impaired concentration, and impaired memory were found during a psychiatric examination of patients with postpartum thyroiditis when compared with a euthyroid control population at 3 months postpartum. These patients' biochemical abnormalities preceded their psychiatric complaints, suggesting that screening for thyroid function abnormalities or elevated microsomal antibodies could prevent these disturbing symptoms.[9] Antimicrosomal antibody titers of 1:400 or greater at delivery or titers of 1:1600 at the first postpartum visit are predictive of serious thyroid dysfunction.[11] The cost-effectiveness of a mass screening program is unknown. Nevertheless, such a program, by preventing maternal decompensation, might also prevent failure to thrive in their infants.

The medical literature on postpartum thyroiditis focuses mainly on diagnosis and management of the mother. The well-being of the infant is rarely addressed. One small case series of 5 women followed through six pregnancies briefly mentions that the offspring had no growth or developmental problems and remained euthyroid. One breast-fed infant was mentioned.[8] A recent excellent review of thyroid problems during pregnancy mentions the consequences of thyroid diseases to the developing fetus and the risk of neonatal thyrotoxicosis in offspring of mothers with Graves' disease. Although the symptoms of postpartum thyroiditis, including impaired memory and depression, are mentioned, the effect these symptoms might have on the mother's ability to care for her infant are not discussed.[12] In the study described above, which found an association between postpartum thyroiditis and altered mental and emotional function, the risk of allowing these women to return to work outside the home was discussed, but the question of how the infant might be affected by the mother's impaired mental health was not addressed.[9]

Our initial concern, when we discovered the mother's thyrotoxicosis, was that the infant's failure to thrive was due to a direct effect of her thyroid disease on the infant before his birth. The effect of immune-mediated maternal thyroid disease on the neonate is dependent upon the type of antibody and the severity of the maternal illness. The thyroid-stimulating antibodies of Graves' disease can cross the placenta, and their action can last approximately 3 weeks. Neonatal Graves' disease, occurring in less than 1 percent of exposed infants, can cause fever, tachycardia, congestive heart failure, and failure to thrive.[4] Although less research is available on thyroid inhibitory immunoglobulins, one study suggests elevated maternal levels of inhibitory immunoglobulins might lead to a transient, self-limited neonatal hypothyroidism.[14] In contrast, while antithyroglobulin and antimicrosomal antibodies can cross the placenta, they cause little or no impact on the fetus.[4,15]

The mother in our case report was breast-feeding, and her clinical course was typical of postpartum thyroiditis; therefore, we did not order a radioactive uptake assay to rule out Graves' disease. The brief thyrotoxic period was most likely due to immune destruction rather than thyroid-stimulating antibodies. Although thyroid studies were not done during her pregnancy, the mother had no clinical evidence or history of preexisting thyroid dysfunction. The infant's initial thyroid screening test at birth and all subsequent studies have been normal. The clinical impression is that the mother's thyroid dysfunction occurred postpartum and did not directly affect the infant through placental or breast milk transfer of antithyroid antibodies or T_4.

In the case described, the infant had known risk factors for growth problems including history of mild hypotonia at birth[1,2] and suck-swallow difficulties subjectively observed by the lactation consultant, who was further concerned about the adequacy of the maternal milk supply.[9] With the support of a team of physicians, lactation specialists, and family, the mother was initially able to care for her infant and nurse with the aid of a breast-feeding appliance. Her abrupt discontinuation of the supplemental formula and uncharacteristic lack of discussion of her decision with any member of her health care team coincided with the onset of her thyroid dysfunction. The cognitive and memory impairments caused by postpartum thyroiditis, which compromised the mother's ability to care for her infant, led to the infant's failure to thrive.

Experience in our clinic suggests that while the case reported here is unusually dramatic, less se-

vere effects on infant and family well-being could be common. For example, a decline in weight percentiles from 10th to less than 5th in a 4-month-old infant led to the diagnosis of postpartum thyroiditis and severe depressive symptoms in the patient's mother. This family led an isolated existence on a Wyoming ranch and had little social support. When asked to describe her life, the mother said, "All I do is stare at the wall or scream at the baby." Another family in our clinic was more fortunate. When the mother's postpartum thyroiditis caused extreme fatigue and inability to cope with daily care of the infant and his siblings, the father was able to take extended leave from his work to help care for the family. This infant did not experience growth delay. In both of these instances of postpartum thyroiditis, T_4 therapy led to a dramatic improvement in the mother's ability to care for her infant. These examples, together with the known wide occurrence of postpartum thyroiditis and almost complete lack of information as to the effect of postpartum thyroiditis on infant well-being, suggest that further investigation of the relation between maternal thyroid dysfunction and failure to thrive is needed.

Conclusion

Postpartum thyroiditis is a common and commonly missed diagnosis that can have repercussions for both the mother and her infant. When compared with classic hypothyroidism, postpartum thyroiditis can have a predominance of psychiatric symptoms. The well-being of an infant can be affected indirectly by the emotional and cognitive difficulties experienced by many women with postpartum thyroiditis. Routine screening of postpartum women with thyroid function studies or microsomal antibody titers could decrease morbidity of both mother and infant, but further investigations of the cost effectiveness of such a program are needed. The possibility of maternal underlying illness, such as postpartum thyroiditis, should be considered during the evaluation of infants with failure to thrive.

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