Secondary Enuresis Associated With Hyperthyroidism

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Anecdotal reports of individual patients who manifest unique symptoms of a relatively common disease can illustrate or even amplify understanding of an important body function. This report describes a case that demonstrates such a linkage between thyrotoxicosis and bladder control. Enuresis, both primary or secondary (an individual who had been reliably dry for at least a year and then starts wetting again), is one of the most common disorders encountered in children. The possible causes for this disorder range from social and psychiatric disturbances to urologic or neurologic disorders.[1-3] Hyperthyroidism, though, does not appear in the differential diagnosis of enuresis. This case represents a unique instance of secondary enuresis associated with hyperthyroidism. The lack of previous recognition of such a conceivable common manifestation of autonomic dysfunction in thyrotoxicosis is surprising. Routine inquiry seeking such an association could be productive, especially in younger patients.

CASE REPORT

A 17-year-old male student presented to Loyola University Medical Center (LUMC) for evaluation of secondary enuresis, which he stated had occurred almost nightly for the preceding few months. On further questioning, he admitted to excessive nervousness, weight loss, heat intolerance, polydipsia, and polyuria (1,800 to 2,600 mL/d). The patient was first seen at LUMC 18 months earlier for a routine school physical examination. At this time he had none of the above complaints and a normal physical examination. The past medical and family histories were unremarkable.

His physical examination revealed a nervous thin man with a blood pressure of 140/90 mmHg, a sinus tachycardia of 110 beats per minute, and a temperature of 37 °C. A diffusely enlarged, nontender, soft thyroid gland was noted. No bruit was perceived over the gland, and there was no evidence of lid lag, exophthalmos, muscle weakness, or tremor. The neurologic examination was normal. The clinical suspicion of hyperthyroidism was confirmed by a plasma triiodothyronine (T_3) of 494 ng/dL (normal 60 to 180 ng/dL); thyroxine (T_4), 18.2 mg/dL (normal 4 to 11 mg/dL); and triiodothyronine (T_3) resin uptake, 36 percent (normal 25 to 35 percent). The urinalysis on multiple occasions was negative for blood, acetone, protein, and glucose, and the urine sediment was benign. The specific gravity of the first morning void was consistently over 1.020. Results of the rest of his routine laboratory tests were unremarkable. An intravenous pyelogram demonstrated two normal kidneys and normal bladder emptying. Further evaluation, including multiple negative urine cultures, failed to demonstrate any other explanation other than hyperthyroidism for the polyuria and enuresis.

The patient was started on methimazole, 10 mg three times a day for one week followed by 10 mg once a day, and propranalol, 10 mg three times a day. After three weeks of treatment, the patient stated that his enuresis had stopped, and he no longer noted polyuria, polydipsia, or heat intolerance. Over the next four weeks the patient appeared to be euthyroid and stated he had had only two episodes of enuresis. The patient inappropriately discontinued his medication and presented to the clinic three weeks later with recurrence of his original complaints including the enuresis. Additionally, he was noted to have rapid atrial fibrillation. The patient was then admitted to the hospital and treated with propranalol and radioactive iodine. Subsequently, the patient became euthyroid, and once again his enuresis disappeared. Some months later, after entering college, his thyrotoxicosis clinically recurred. Once again it was associated with enuresis as an apparent sequela. After appropriate documentation, he was given a second course of radioactive iodine therapy and subsequently has remained symptom free.

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DISCUSSION

The patient presented with secondary enuresis as one of the major initiating symptoms of his hyperthyroidism. With successful control of his thyroid disease, his enuresis ceased, only to return with each of two subsequent recurrences of his thyrotoxicosis. Following definitive therapy with radioactive iodine, the patient became euthyroid permanently and has had no subsequent episodes of enuresis.

This instance of reported secondary enuresis associated with hyperthyroidism is unique. Walter et al,[4] however, described two patients with primary enuresis associated with thyrotoxicosis, which similarly disappeared following rendering of the patients euthyroid.

Despite suggestions of Bradley[5] and Mendez Bauer et al[6] of an effect of thyroid hormone on bladder function, two cases of Walter et al[4] and the present case are surprisingly the only cases of enuresis associated with hyperthyroidism reported. In fact, hyperthyroidism is not even mentioned as a possible etiologic factor in reviews of the pathogenesis and management of enuresis or in reviews on hyperthyroidism. This lack of association may be because patients are embarrassed by this behavior and do not complain about it.

There are at least two possible explanations for the enuresis. The psychological stress, emotional lability, and nervousness that accompany hyperthyroidism may be enough to lead to bladder irritability and disruption of learned bladder control. Kolvin[2] has previously noted that there could be a psychogenic cause or at least a contributing factor in as many as 50 percent of patients with secondary enuresis. Excessive thyroid hormone may be augmenting tissue sensitivity to adrenergic activity, thereby inducing altered "functional bladder" capacities and tone, in turn leading to enuresis. Hannappel[7] has recently reviewed the effects of the adrenergic nervous system on the upper and lower urinary tract. He points out the importance of both α-adrenergic and β-adrenergic receptors in the bladder dome and body, as well as in the bladder neck and urethra, in the control of micturition. Thus, enuresis may be just another manifestation, such as palpitations and sweating, of an augmented adrenergic nervous system seen with hyperthyroidism. This explanation would account for the improvement of the patient's symptoms, including the enuresis, in response to propanalol therapy as well as explain the recurrence of enuresis with the inadvertent discontinuation of the propranalol prior to rendering the patient euthyroid. Enuresis is a reasonably expected sequela of thyrotoxicosis, and a specific history of enuresis might profitably be elicited in such patients, if indeed the association is valid.

References

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