BLADDER ATONY AND ILEUS IN A MAN WITH MYXEDEMA: CASE REPORT

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Abstract
Pseudoobstruction is an uncommon manifestation of hypothyroidism. However, hypotony of the bladder in myxedema is very rare, and there were only four such cases reported previously. We present a 45-year-old man with myxedema and ileus, who was started on thyroxine therapy and was detected to have bladder hypotony on the 5th day of admission. There was no evidence of bladder outflow obstruction, and both bowel and urinary symptoms improved with thyroxine therapy. Bladder hypotony may be more prevalent in patients of hypothyroidism than previously reported; hence, it is important to have a high clinical suspicion and perform early urodynamic studies to avoid back-pressure damage to the upper tracts.

Key words: hypothyroidism, myxedema, bladder atony, pseudoobstruction, ileus.

INTRODUCTION

Thyroid hormone deficiency has profound effects on the smooth muscles of the gastrointestinal tract. The manifestations may be dysphagia, impaired gastric emptying, constipation, and ileus or pseudoobstruction (1-4). Hypotonic changes in the bladder smooth muscle in myxedema can also occur due to similar mechanisms. Surprisingly, these changes have been reported in very few cases previously (5-8). We report a case of a 45-year-old man with myxedema, ileus, and a hypotonic bladder, in whom all the symptoms responded to treatment with thyroxine therapy. To the best of our knowledge, this is only the fifth such report in English-language literature.

CASE SUMMARY

A 45-year-old male was admitted to the emergency with a 5-day history of generalized abdominal distension, obstipation, swelling over the face and neck, and

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fever. There was prior history of weight gain and cold intolerance over the past three weeks. Physical examination revealed an excessively somnolent, dull, middle-aged male with subnormal temperature (36.1 °C), and facial puffiness. The vitals were stable, and there were no diminished heart sounds. Thyroid examination was unremarkable. The abdomen was grossly distended, non-tender, with absent bowel sounds. Digital rectal examination did not reveal significant prostatomegaly or prostatic nodules. Neuromuscular examination revealed mildly diminished tendon reflexes. Serum sodium levels were low, at 121 mEq/L, and other routine hematological and biochemical blood tests were normal. Abdominal radiographs were suggestive of colonic dilatation. CT abdomen showed large bowel loops with significant dilatation mainly in the transverse and sigmoid colon, with no identifiable mechanical obstruction (Figs. 1 and 2). CT chest revealed pericardial effusion, with bilateral pleural thickening. Two-dimensional echo-cardiography confirmed mild pericardial effusion. Thyroid function tests were suggestive of severe hypothyroidism: serum T₃<0.25 mg/mL (N=0.60-1.81), serum T₄=1.10 μg/dL (N=5.60-13.70), serum TSH=36.34 μIU/mL (N=0.35-5.50). The levels of thyroid peroxidase antibody (anti-TPO) were also raised: 134.40 U/mL (N<60.00). Ultrasound neck did not reveal abnormal thyroid nodules or enlargement. A diagnosis of myxedema (possible etiology - Hashimoto’s thyroiditis) with ileus (colonic pseudoobstruction) was made, and the patient was started on conservative management with intravenous fluids, nasogastric aspiration and urinary catheterization. Oral thyroxine replacement (100μg/day) was started on the 3rd day of admission. The patient responded well, with improved alertness, decrease in facial swelling, and return of bowel sounds over 5-6 days of treatment. However, the patient did not pass urine after catheter removal on the 3rd day of admission. On the 5th day, a distended bladder up to the umbilicus was palpated, without any sensation of voiding. Catheterization was again performed, and 2.5 L of urine was drained. Subsequently, the patient did not void satisfactorily after catheter removal,

Figure 1. Coronal section of CT scan showing multiple dilated colonic loops (arrows).

Figure 2. Cross-sectional CT image also showing multiple dilated colonic loops (labeled ‘C’).
and intermittent catheterization was performed to prevent overdistension of the bladder. Uroflowmetry was done by asking the patient to void by exerting pressure on the abdominal wall, which revealed a peak flow velocity of 13 mL/s. Ultrasound of the urinary system revealed a prostate size of 24 cc, with no changes in the upper tracts. Cystometry indicated low detrusor pressures (11 cm H₂O) with increased functional bladder capacity. The patient regained partial sensation to void after about 2 weeks of treatment, and near-complete sensation at 3 weeks. A repeat thyroid profile after 3 weeks showed a significant rise in serum T₃ (0.63 ng/dL) and serum T₄ (2.80 μg/dL) levels, and a decrease in serum TSH (17.09 μIU/mL). The dose of oral thyroxine was changed to 75 μg/d, and the patient was discharged. Follow-up urodynamic studies after 2 weeks revealed a rise in detrusor pressure and a decrease in the bladder capacity. The final diagnosis was severe hypothyroidism (possible Hashimoto’s thyroiditis) with ileus and bladder atony. The patient is well, with no complaints after 2 months of treatment with no evidence of pericardial effusion.

**DISCUSSION**

The mechanisms by which thyroid hormones influence gastrointestinal motility may be the combination of a direct effect on the muscle receptors, and, an indirect effect mediated through catecholamines. The colon is often affected, leading to constipation commonly, and pseudo-obstruction in severe cases. The pathological changes which have been observed in the colon are mucosal atrophy, submucosal lymphocyte infiltration, and, deposition of a mucinous substance separating muscle fibres from the ganglia of Auerbach’s plexus (2,4,9,10). These changes are initially reversible with thyroid hormone replacement, but progressive smooth muscle atrophy may occur, which is ultimately unresponsive to treatment in late cases (10).

In the urinary tract, Evans (5) described the first case of bladder atony with myxedema in a 49-year-old woman, in 1932. The distended bladder in this patient was discovered by ‘accident’. The author attributed the bladder atony solely to thyroid insufficiency, because there was no neurological lesion, and, the urinary symptoms improved significantly with thyroid medication. In our patient, the discovery of the distended bladder was also a chance finding on the 5th day of admission. We ruled out bladder outflow obstruction by a normal prostatic evaluation (digital rectal examination, flow rates, and ultrasound). Urodynamic studies indicated low detrusor contractility, and the patient could void freely on exertion of pressure on the abdominal wall. After institution of thyroxine replacement therapy, bladder function took about three weeks to recover, and this was confirmed urodynamically by a rise in detrusor pressure.

We feel that an atonic bladder may be more prevalent in myxedema patients than previously reported. This is probably because the bladder outflow is unaffected, and the patients learn to void satisfactorily by exerting pressure on the abdomen, without perceiving any problem. Another reason that this association has
been rarely seen could be that only severe thyroid hormone deficiency may be responsible for affecting the bladder smooth muscle, as seen in our case. After thyroid replacement, bladder function was sluggish to respond. Also, severe hypothyroidism is unusual nowadays in the clinical setting. In all circumstances, recognition by the clinician is important, as late cases may not respond to thyroxine and progress to uremia. This scenario has been reported by Hansen et al. (7). Such cases need intermittent sterile catheterization to prevent urinary stasis and back-pressure, till bladder function recovers with thyroxine.

Mendez Bauer and colleagues (6) have documented the changes in the bladder tone and contractility in 3 cases of myxedema, using simple cystometric techniques available at that time. They concluded that hypothyroidism causes hypotonia and increased bladder capacity, and that these changes are reversed by thyroxine replacement. Decreased bladder tone and increased capacity were also seen in our patient; in fact, at the first catheterization, we drained almost 2.5 L of urine.

In conclusion, we would like to emphasize that bladder atony must be looked for in patients with hypothyroidism, especially severe cases. This is because thyroxine therapy alone can reverse early changes, preventing complications and improving patient comfort. The unusual features in our hypothyroid patient were the simultaneous occurrence of ileus and bladder hypotonia, which reversed completely with thyroid replacement. A prospective study in hypothyroid patients, incorporating the evaluation of bladder function, would be useful in proving this hypothesis.

References