



Acute-onset rapidly progressive peripheral polyneuropathy in a 12-year-old girl due to Hashimoto thyroiditis: traps in the diagnosis



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Introduction and purpose

- Thyroid dysfunction may cause a wide range of neurological disorders in children and adolescents. Hypothyroidism is associated with peripheral nerve demyelination. However, minimal data are available in children.
- *Our purpose is to describe a 12-year-old girl newly diagnosed with Hashimoto thyroiditis suffering acute polyneuropathy.*

Case report

History

- A 12-year-old girl with newly diagnosed HT started on levothyroxine (LT4) was admitted because of a 2-week history of left upper limb weakness and paresthesia.
- Her past history was uneventful except for possible cow milk allergy. According to the family history, the patient's mother was diagnosed with HT and systematic erythematosus lupus, maternal grandmother and aunt with HT and paternal grandmother was known to have single kidney with lithiasis.

Physical and laboratory evaluation

- She had diminished touch sensation and motor weakness on left upper limb and bilateral reduction of knee reflexes.
- Cerebrospinal fluid (CSF) examination revealed slightly elevated albumin (82 mg/dl) while electrophysiological study was indicative of acute sensory polyneuropathy.

Initial diagnosis

- Based on the above findings, the patient received intravenous immunoglobulin treatment with a diagnosis of *Guillain-Barré syndrome*.

Course

- The following days clinical deterioration was recorded; she had wide based standing, her gait was slightly ataxic and she developed acute onset moderately severe, continuous, burning pain affecting her left foot.
- Meanwhile, thyroid function evaluation questioned the diagnosis. Thyroid stimulating hormone (TSH) was suppressed (0.667 μ IU/ml) while free thyroxine (fT4) was elevated (1.66 ng/dl) with high titers of antithyroid antibodies.
- Further detailed history revealed signs and symptoms of hyperthyroidism (weight loss, tremor, anxiety, sleeplessness, palpitations, diarrhea).
- Based on negative thyroid stimulating immunoglobulin (TSI) a diagnosis of *iatrogenic hyperthyroidism* was made. LT4 was discontinued and beta-blocker was prescribed because of severe tachycardia and hypertension leading to gradual clinical and biochemical improvement.
- Despite the normalization of fT4 and TSH, further deterioration of weight loss and neuropathic pain alongside with amenorrhea and complaints of nausea and dysphagia necessitated further evaluation of sensory polyneuropathy including and *anorexia nervosa*.

Investigation

- Potential causes of acute sensory polyneuropathy (vitamin deficiency, metabolic, toxic, infectious, inflammatory, autoimmune, paraneoplastic, inherited) were excluded.
- The only pathological laboratory findings (mildly increased aminotransferases, extremely elevated ferritin and low sex steroid and gonadotropin serum levels) could be attributed to possible anorexia nervosa.
- However, psychiatric evaluation considered the potential of anorexia nervosa as not very likely.
- On evaluation, salivary gland dysfunction was recorded, expected in HT patients. Furthermore, upper gastrointestinal tract endoscopy revealed gastritis. The above findings could explain dysphagia.

Treatment

- She started physiotherapy and was treated initially with B6 and B12 vitamins and magnesium without significant clinical or electrophysiological improvement. On discharge, she was also prescribed pregabalin for neuropathic pain and lansoprazole for gastritis. Beta-blocker was gradually discontinued.

Follow-up

- On 6-month follow-up is euthyroid, while signs and symptoms of hyperthyroidism have resolved. She gained the lost body weight and her menstrual started to normalize. Glove and stocking distribution neuropathy has improved significantly. She continues physiotherapy.

Final diagnosis

- Based on this clinical course, HT can be considered the cause of neuropathy explaining also the slightly elevated CSF albumin and salivary gland dysfunction leading to dysphagia and body weight loss.
- Hypothyroidism is associated with decreased nerve conduction velocity. In adults, polyneuropathy with the classical glove-and-stocking distribution is frequent. Deep tendon reflexes show a delayed relaxation phase.

Conclusion

- This case experiencing the whole spectrum of thyroid dysfunction, moving from hypothyroidism to hyperthyroidism and finally to euthyroidism underlies the necessity of thyroid function evaluation in children with acute polyneuropathy. Symptoms of neuropathy may precede the diagnosis of hypothyroidism and persist despite normalization of thyroid hormone levels.

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