

Unusual Presentation of Hypothyroidism

Nathan Jacobs¹, Taysir Al Janabi², Karampal Mand²

¹Drexel University, College of Medicine, 2900 W. Queen Lane, Philadelphia, PA 19129

²WellSpan York Hospital, 1001 S George St, York, PA 17403

***Corresponding Author:** Taysir Al Janabi, WellSpan York Hospital, 1001 S George St, York, PA 17403.
Email: taysir.aljanabi@gmail.com

Abstract

Hypothyroidism is a condition frequently encountered by physicians in an outpatient or inpatient setting. Symptoms typically follow a common pattern that includes fatigue, weight gain, and cold intolerance among other symptoms. However, there can be some variation in symptoms. Here we present a case of a patient with levothyroxine non-adherence who presented with an atypical presentation of severe hypothyroidism.

Keywords: Hypothyroidism; hyperreflexia; thyroid; fasciculations

1. INTRODUCTION

The classic symptoms of hypothyroidism are fatigue, weight gain, cold intolerance, constipation, bradycardia, along with neurological presentation, such as paresthesia and delayed relaxation of deep tendon reflexes. Hormone replacement therapy with thyroxine is the key treatment in hypothyroidism. Thyroxine has a narrow-therapeutic index which requires adequate dosing, patient compliance, and regular monitoring [1]. Thyroxine non-compliance, increased demand, decreased intestinal absorption, and increased renal clearance are some of the factors affecting the therapeutic level of thyroxine. However, patient non-adherence is the most common cause of abnormal thyroid hormone level in hypothyroid individuals [1]. In this case report, we are presenting a case of a non-compliant hypothyroid patient who presented with unusual neurological symptoms of facial fasciculation and hyperreflexia.

2. CASE REPORT

The patient was a 63-year-old female with a history of hypothyroidism on levothyroxine (112 mcg) who presented to the hospital with a chief complaint of generalized weakness that had been increasing over 2-3 months. She reported generalized weakness, fatigue, difficulty ambulating, and a weight loss of between 5 and 10 pounds over a few months. She also reported localized frontal headaches.

On physical exam she had mildly decreased strength. Notably, her reflexes were 3+ throughout, and she had right lower facial fasciculations. On day one of her hospitalization, she reported taking her levothyroxine appropriately, but when questioned further on day two of hospitalization, she admitted to not taking her levothyroxine for 2-3 months prior to admission. Thyroid-stimulating hormone (TSH) on admission was elevated at 82.7 mIU/mL which increased from 12.2 mIU/mL from two months prior, and the T4 level was below 0.3 mIU/mL.

Differential diagnoses initially included neoplastic vs paraneoplastic syndrome, multiple sclerosis, cervical spinal stenosis, and levothyroxine misuse. Infectious or inflammatory etiologies were also considered but were less likely given a normal white blood cell count and normal erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). A computed tomography (CT) scan of her head with and without contrast showed no acute intracranial pathology. A neurology consult recommended brain and cervical magnetic resonance imaging (MRIs) which both came back unremarkable. A neurologist reported that hyperreflexia could be caused by her hypothyroidism secondary to medication non-compliance versus her baseline.

After the patient admitted to not taking her levothyroxine, we restarted it at an increased dose of 150 mcg daily. Over the next several

days, the patient's strength improved to some extent, and her facial fasciculations disappeared. The patient was discharged to a skilled nursing facility with plans to continue to follow with her primary care physician. Five weeks later, a follow-up TSH was within normal limits.

3. DISCUSSION

Our patient presented with several classic findings of hypothyroidism—most notably generalized weakness and fatigue which also were her most distressing symptoms. However, her symptoms of hyperreflexia and facial fasciculations are not typical of hypothyroidism. These, combined with her initial report of appropriate adherence to her levothyroxine resulted in a brief delay in diagnosis while we ruled out a number of other diagnoses. However, a mild improvement in her symptoms on discharge after several days of an increased dose of levothyroxine (as well as the exclusion of other differential diagnoses) points towards the diagnosis of an atypical, severe presentation of hypothyroidism.

Our patient's presentation illustrates the variety of presentations that hypothyroidism can cause. Hypothyroidism classically causes hyporeflexia—not hyperreflexia. Moreover, fasciculations are not generally associated with hypothyroidism. Hypothyroidism may cause significant symptoms related to neuromuscular dysfunction such as clinical features of myopathy, mononeuropathy, and sensorimotor axonal polyneuropathy which may explain the development of hyperreflexia in our patient [2]. However, not many case reports were identified regarding similar presentation. Hyperreflexia

has been reported in two hypothyroid conditions—Hashimoto encephalopathy and central hypothyroidism—which may be related to increase thyroid-releasing hormone (TRH) level which modulates synaptic function [3].

Our patient also demonstrated mild weight loss—something not unusually associated with hypothyroidism (weight gain is the more typical presentation). However, while the history was somewhat unclear, it seemed that she may have had trouble preparing food and eating due to her weakness and fatigue.

4. CONCLUSION

Hypothyroidism is a common condition that has been extensively studied and reported in the literature. However, physicians should be aware that symptoms do not always follow the classic pattern. Additionally, patients with a history of hypothyroidism should be carefully questioned about their adherence to medication, and the dose of levothyroxine should be carefully titrated to alleviate patient symptoms.

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