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CASE REPORT

Hypothyroidism presented with dysarthria and generalized weakness: a case report and review of literature

Hipotiroidismo presentado con disartria y debilidad generalizada: informe de un caso y revisión de la literatura

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Abstract

Generalized muscle weakness is a common complaint in the emergency department. The presence of concomitant dysarthria often triggers the suspicion of an acute stroke. However, none of these symptoms are specific to stroke and up to 30% of dysarthric patients ended up having another disease. Endocrine disturbance is a well-known mimicker of stroke and should always be considered in patients presenting with generalized weakness. It was estimated that over 70% of patients with hypothyroidism had complaints suggestive of muscle dysfunction. Although bulbar muscle involvement is rare, a few case reports have described undiagnosed hypothyroidism presented with dysarthria and generalized weakness. Here we described a 64-year-old female admitted for acute weakness in bilateral upper and lower extremities causing difficult ambulation and slurred speech. On physical examinations, the muscle power was 3/5 in all four limbs, and dysarthria was appreciated. She was initially treated for an acute stroke until she was found to have a significantly elevated serum creatinine kinase (CK) level and serum thyroid-stimulating hormone (TSH). Her CK was 977 U/L, TSH 289, free T4 <0.07, and T3 0.3. She also had dyslipidemia. Repeat history and medication reconciliation revealed poor compliance to Synthroid treatment for Hashimoto thyroiditis. Her muscle power and speech drastically improved after intravenous levothyroxine of 100mcg daily. Hypothyroid myopathy typically affects the type II fast-twitching muscle, leading to retarded muscle contractions. CK is elevated in about 60-90% of the patients and does not correlate to the severity of muscle involvement. Although mostly progressive, cases with an acute presentation of generalized weakness have been reported due to superimposed metabolic or electrolyte imbalance. In addition, dysarthria can be attributed to bulbar myopathy and/or glucosaminoglycan deposition. A review of literature identified two cases of hypothyroidism presented with dysarthria. The other two patients were diagnosed only after the routine thyroid function test came back positive. It appears that hypothyroidism is an underrated cause of dysarthria and that associated symptoms are frequently missed in the initial history due to low physician suspicion. Therefore, the initial assessment for these patients should include relevant history, physical examination, and a TSH level. Treatment is thyroid hormone replacement therapy. Prompt recovery of muscle weakness and dysarthria is expected, typically within 2 months.

Key words: Hypothyroidism, dysarthria, weakness.

Resumen

La debilidad muscular generalizada es una queja frecuente en los servicios de urgencias. La presencia de disartria concomitante suele hacer sospechar un ictus agudo. Sin embargo, ninguno de estos síntomas es específico del ictus y hasta el 30% de los pacientes con disartria acaban teniendo otra enfermedad. Las alteraciones endocrinas son un mimetismo bien conocido del ictus y siempre deben tenerse en cuenta en los pacientes que presentan debilidad generalizada. Se calcula que más del 70% de los pacientes con hipotiroidismo presentan quejas que sugieren una disfunción muscular. Aunque la afectación de los músculos bulbares es rara, algunos informes de casos han descrito hipotiroidismo no diagnosticado que se presenta con disartria y debilidad generalizada. Aquí describimos a una mujer de 64 años que ingresó por una debilidad aguda en las extremidades superiores e inferiores bilaterales que le dificultaba la deambulación y la dificultad para hablar. En la exploración física, la potencia muscular era de 3/5 en las cuatro extremidades y se apreciaba disartria. Inicialmente fue tratada por un accidente cerebrovascular agudo, hasta que se descubrió que tenía un nivel de creatinina quinasa (CK) sérica y de hormona estimulante de la tiroides (TSH) significativamente elevados. Su CK era de 977 U/L, TSH 289, T4 libre <0,07 y T3 0,3. También tenía dislipidemia. La repetición de la historia y la conciliación de la medicación revelaron un mal cumplimiento del tratamiento con Synthroid para la tiroiditis de Hashimoto. Su fuerza muscular y su habla mejoraron drásticamente tras la administración de levotiroxina intravenosa de 100mcg diarios. La miopatía hipotiroidea suele afectar al músculo de contracción rápida tipo II, lo que provoca un retraso en las contracciones musculares. La CK está elevada en aproximadamente el 60-90% de los pacientes y no se correlaciona con la gravedad de la afectación muscular. Aunque la mayoría de las veces es progresiva, se han descrito casos con una presentación aguda de debilidad generalizada debido a un desequilibrio metabólico o electrolítico superpuesto. Además, la disartria puede atribuirse a la miopatía bulbar y/o al depósito de glucosaminoglicano. Una revisión de la literatura identificó dos casos de hipotiroidismo presentados con disartria. Los otros dos pacientes fueron diagnosticados sólo después de que la prueba rutinaria de la función tiroidea resultara positiva. Parece que el hipotiroidismo es una causa infravalorada de disartria y que los síntomas asociados se pasan por alto con frecuencia en la historia inicial debido a la baja sospecha del médico. Por lo tanto, la evaluación inicial de estos pacientes debe incluir la historia pertinente, la exploración física y un nivel de TSH. El tratamiento es la terapia de sustitución de la hormona tiroidea. Se espera una pronta recuperación de la debilidad muscular y la disartria, normalmente en dos meses.

Palabras clave: Hipotiroidismo, disartria, debilidad.

Introduction

Generalized muscle weakness is a common complaint in the emergency department (ED) with inexhaustible differential diagnoses. Patient history and careful neurological examinations are crucial as some etiologies may be life-threatening[1]. Dysphagia and dysarthria indicate possible brain-stem stroke, but any causes affecting the bulbar muscles can appear similarly. The non-specific nature of weakness was demonstrated in a prospective observational study involving 79 consecutive patients presenting with generalized weakness. The final diagnosis encompassed 14 distinct international classification of disease-10 codes and endocrine, nutritional, and metabolic diseases were the second most frequent cause (14%)[2]. However, the list of endocrine, nutritional, and metabolic disturbance is equally heterogeneous, including hypothyroidism, hypoglycemia, adrenal insufficiency, and periodic paralysis due to electrolyte imbalance[1]. Hypothyroidism results from deficiencies in the thyroid hormones, thyroxine (T4) and triiodothyronine (T3). It is estimated that 3-10% of the world population has clinical or subclinical hypothyroidism[3]. Patients may complain about fatigue, cold intolerance, constipation, irregular menses, and weight gain, reflecting a generalized slowing of metabolic processes[4]. On physical examinations, dry skin, coarse hair, generalized non-pitting edema, macroglossia, and bradycardia may be appreciated[3,4].Diagnosis is made through laboratory data showing a high serum thyroid-stimulating hormone (TSH) concentration and low serum free T4 level. In addition, hyponatremia, dyslipidemia, and hyperuricemia are not uncommon[3]. Hypothyroid myopathy occurs in 30-80% of hypothyroid patients as weakness, spasm, pain, or stiffness in the muscles[5]. Serum creatine kinase is often elevated[6]. Atrophy of the fast-twitching muscles and hypertrophy of the slow-twitching muscles due to glucosaminoglycan deposition in muscle fibers may be observed on physical examinations[7]. Although rare, hypothyroidism presenting with generalized muscle weakness and dysarthria is possible. Here we presented a patient with undertreated hypothyroidism who endorsed progressive generalized weakness and slurred speech. The stroke workup was negative and the initial history disclosed symptoms pertinent to hypothyroidism. Intravenous levothyroxine was commenced and her muscle strength improved drastically. The clinical presentations, differential diagnosis, pathogenesis, and management of hypothyroid myopathy were summarized in the discussion.

Case report

A 64-year-old female presented to our ED due to acute weakness in bilateral upper and lower extremities causing difficult ambulation and slurred speech. Her past medical history was remarkable for Hashimoto thyroiditis, dyslipidemia, asthma, osteopenia, and primary hyperparathyroidism. On examination, the temperature was 97.7°F, the blood pressure 205/102 mmHg, the heart rate 51 beats per minute, and the oxygen saturation 99% while the patient was breathing ambient air. A review of systems was notable for progressive weakness, lethargy, and psychomotor retardation for one month. She was prescribed Synthroid 125 mcg daily but had had varying TSH readings from <0.015 to 9.77 due to poor compliance. Her last dose of Synthroid was one week prior to admission. On evaluation, the patient was alert and oriented. She had mild periorbital swelling and a thyroid goiter. Her muscle bulk was compatible with her age and there was no non-pitting edema or induration in the legs. Strength in both arms and legs was assessed as 3/5. She had no focal neurological deficits and deep tendon reflexes were normal. Computed tomography and magnetic resonance imaging of the brain revealed no acute ischemic or hemorrhagic change. Electrocardiography showed regular sinus rhythm with no acute ST changes. Routine thyroid function tests reported a TSH of 289, free T4 <0.07, and T3 0.3. The lipid panel revealed a cholesterol level of 420, low-density lipoprotein 287, high-density lipoprotein 81, and triglyceride 312. The complete metabolic panel was normal except for an elevated creatine kinase (CK) at 977 U/L. Under high suspicion for hypothyroid myopathy, intravenous levothyroxine 100 mcg daily was commenced. Her muscle power improved to 3/5 in both upper and lower extremities on the second day of hospitalization and fully recovered after three days of levothyroxine treatment. Her thyroid function normalized and her subjective symptoms improved. She was discharged on Tirosint 125 mcg daily.

Discussion

Myopathy in hypothyroid patients is not fully understood. T4 deficiency reduces mitochondrial electron transport chain efficiency, which causes selective atrophy of the fast-twitching (type IIb) muscle and the clinical presentation of retarded muscle contractions[7]. At the same time, compensatory hypertrophy and glycosaminoglycan deposition results in prolonged oxidative damage and eventually rhabdomyolysis[7]. Although a TSH plus free T4 level suffices for the diagnosis of hypothyroid myopathy, their value does not always correlate with the severity of muscle weakness[7]. CK is a non-specific marker widely used to diagnose muscle injury. Causes of an elevated CK include inflammatory myopathies, dystrophinopathies, rhabdomyolysis, and certain medications[9]. CK is elevated in about 60-90% of hypothyroid patients even years before clinically overt muscle diseases[6]. In contrast, CK usually remains normal in other endocrine myopathies such as hyperthyroidism, hyperparathyroidism, and

Cushing's syndrome[9]. Electromyography can be helpful but is neither sensitive nor specific[7]. Typical findings include short-duration, polyphasic, and low-amplitude motor unit action potentials[10]. Hoffmann syndrome is a subtype of hypothyroid myopathy characterized by proximal muscle weakness and pseudohypertrophy of the calf muscles[10]. Muscle biopsy is not usually needed but will show type II fiber atrophy, type I fiber hypertrophy, mitochondrial inclusions, glycogen accumulation, disorganized myofibrils, focal necrosis, and minimal inflammatory infiltrates[7].

Besides muscle damage, hypothyroidism rarely presents with periodic paralysis due to disturbance in potassium ion metabolism. T4 normally increases the number and efficiency of the hydrogen-potassium ATP pump on the distal convoluted tubules[11]. Therefore, hypothyroidism predisposes patients to hypokalemia when there is a concomitant cause of renal potassium wasting. However, periodic paralysis is more commonly seen in patients with hyperthyroidism due to intracellular potassium shift and 14 cases associated with hypothyroidism had been reported[11].

Another interesting finding in our case was dysarthria. Stroke remains the most important differential diagnosis for acute dysarthria but up to 30% of these patients ended up having another disease[12,13]. A review of literature identified only two cases of bulbar muscle involvement in hypothyroid patients. The first case was a 43-year-old man presented to the ED with dizziness, blurred vision, and slurred speech for one day, suspected to have a brain-stem infarction. The patient had sleep apnea but no history of thyroid disorder[14]. The second case was a 39-year-old female complaining about dysarthria for 6 months. She had sleep apnea but her past medical history was otherwise unremarkable[15]. Both cases were diagnosed after the routine thyroid function tests came back positive and the second history revealed long-standing hypothyroid symptoms that were initially neither reported nor asked for. Dysarthria attributed to hypothyroidism may be caused by myopathy in the articulating muscles, edematous swelling of laryngeal and hypopharyngeal structures, and macroglossia from glucosaminoglycan deposition[14,15]. We were able to identify hypothyroid myopathy at the initial evaluation given the patient's positive medical history. However, dysarthria could also be the presenting symptom in patients with no known thyroid disease, as in the two previous cases. Physicians should maintain a high vigilance when other features such as fatigue, cold intolerance, weight gain, sleep apnea, and dry skin are noted.

Prompt recovery of muscle weakness and dysarthria is expected, typically within 2 months, after adequate hormone replacement therapy.

Conclusion

Hypothyroidism should be considered in patients presenting with generalized muscle weakness with or without dysarthria, an elevated CK level, and a negative stroke work-up. A focused history and thyroid function tests usually confirm the diagnosis. Rapid recovery with thyroid hormone replacement is expected.

Competing interests

The authors have no competing interests to declare that are relevant to the content of this article.

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