ISSN 2475-5451

Research Article

International Journal of Diabetes & Metabolic Disorders

Hypothyroid Myopathy: A Commonly Missed Complication of Hypothyroidism

Christian de Souza*, Shay Hasan, Adam Hoffmann and Babajide Adio

Internal Medicine Residency, MercyOne North Iowa Medical Center, Mason City, IA

***Corresponding Author**
Christian de Souza, Internal Medicine Residency, MercyOne North Iowa Medical Center, Mason City, IA

Submitted: 2023, June 28; **Accepted**: 2023, July 29; **Published**: 2023, Aug 28

Citation: Souza, C. D., Hasan, S., Hoffmann, A., Adio, B. (2023). Hypothyroid Myopathy: A Commonly Missed Complication of Hypothyroidism. *Int J Diabetes Metab Disord, 8*(3), 431-433.

Abstract

Muscle disease is a known complication of hyperthyroidism as well as hypothyroidism. The muscle disease is associated with elevated creatine kinase and pain. This the case of a 63-year-old female who presented to the ED from her cancer therapy with elevated creatine kinase of 2,000 IU/L (International units/Liter). She reports muscle pain in her shoulders and hips for the past 3 days. Patient was recently diagnosed with hypothyroidism and has started on levothyroxine 75 mcg. Her Thyroid Stimulating Hormone (TSH) stays elevated at 65 IU (international units). Other autoimmune causes of elevated creatine kinase were excluded via testing, leaving hypothyroid myopathy as the only possible probable cause of this patient elevated creatine kinase. Muscle pain and creatine kinase should resolve with proper levothyroxine dose and compliance to therapy when patient becomes euthyroid. Unfortunately, the patient passed away from complications related to fall.

1. Introduction

In adults with thyroid disease, muscle disease is a common observation however not well taught, as it is present in 79% of cases [1]. Elevated creatinine kinase is the most common preliminary lab findings of muscle disease, sensitive but not as specific. This case report presents the case of a woman with elevated creatinine kinase, the work up included that led to the diagnosis of hypothyroid myopathy and the discussion of such case correlates to the clinical and academic medicine.

2. Case Presentation

Patient is a 63 year old female with past medical history significant for type 2 diabetes mellitus, Chronic obstructive pulmonary disease, osteoarthritis and recurrent small cell lung cancer on cycle 4 Lurbinectedin alkylating therapy who was admitted to the Internal medicine teaching service for elevated creatine kinase (CK) >2,000 IU/ on her lab work when she presented to her cancer follow up appointment. The patient initially reported generalized muscle pain especially on the legs bilaterally. Pain is described as cramping and has been worse the past 3 days. She does have chronic tingling of her face and right hand which have remained unchanged and for which she is being followed by neurology. She does report feeling weaker for 1 week after Lurbinectedin therapy before regaining her energy. Last therapy was 3 weeks ago. She does ambulate with a walker, feels unsteady without a walker but denies any recent falls. She is sedentary. She denies any fevers or recent infections. She experienced lightheadedness with position changes which is not a new symptom for her. She is transitioning to a new PCP as her former physician is retiring. She is on Synthroid which has been recently increased and is also on statin therapy. Initial differential included medications induced, autoimmune or viral myopathy.

2. 1 Examination and Investigations

Initial work up included holding statin, obtaining liver function tests, troponin, TSH and myoglobin levels and initiation of IV fluids. Myoglobin and TSH levels were elevated respectively at 1,144 Nanogram per milliliter and 65 mIU/L (milli international units per liter). Creatinine kinase was repeated to confirm elevated levels recorded prior to admission.

Lab results on admission pointed less towards rhabdomyolysis with mildly elevated creatine kinase, no evidence of acute kidney injury and low potassium. Despite holding statin, creatine kinase continued to rise, making for a lesser argument for statin induced myopathy as a possible diagnosis. Creatinine kinase peaked at 3,140IU/L (international Units per Liter) the following day after admission before starting to trend down after administration of 2 Liters intravenous fluids bolus and initiation of maintenance fluid at a rate of 150 cc/hr. Intravenous fluids were initiated as an approach for therapy for an early presentation of rhabdomyolysis with mildly elevated creatinine kinase and no obvious signs of acute kidney injury. Rhabdomyolysis was ruled out as a diagnosis after urine myoglobin obtained later, came back negative. Even

though rhabdomyolysis and statin induced myopathy was ruled out as possible diagnoses, creatinine kinase remained elevated at 1,500 IU/L despite fluid resuscitation.

Other differential diagnoses included idiopathic inflammatory myopathies such as polymyositis, dermatomyositis, inclusion body myositis or immune mediated necrotizing myopathy, viral etiology or hypothyroid myopathy. On admission, the patient had concerns for bronchitis. Chest X-ray and procalcitonin level were unremarkable for any sign of bacterial infection. Respiratory panel including adenovirus, multiple coronavirus variants, human Metapneumovirus. Rhinovirus, influenza A and B, parainfluenza and respiratory syncytial virus were negative, thus ruling out a viral etiology.

Even though pain was mostly on lower extremities, she did report aching in her upper extremities and presence of a rash on her right arm. Physical exam did not show any cutaneous manifestations such as heliotrope rash or Gottron's papules which could be sufficient to confirm the diagnosis of dermatomyositis in the setting of lack of alternative diagnosis [2]. However, an autoimmune workup consisting of antinuclear antibodies (ANA), aldolase and Anti Jo was performed. Anti-Jo 1 test which if positive is consistent with polymyositis and suggestive of an increased risk of pulmonary involvement [3]. With fibrosis especially in this setting with her history of lung cancer came back negative. However, a negative Anti-Jo 1 test along with normal C reactive protein (CRP), and Erythrocyte sedimentation rate (ESR) does not exclude polymyositis [4]. The diagnosis of dermatomyositis and polymyositis are further eliminated with the negative ANA and Magnetic Resonance imaging (MRI) results not revealing any signs of myositis but showed some bone metastasis which do not explain the elevated creatine kinase. It is understood that myositis requires muscle biopsy for definitive diagnosis [3]. That can be done later an outpatient basis. Based on the results of the different tests conducted ruling out the above differential diagnosis, the likely diagnosis for this patient presentation is hypothyroid myopathy. She has an elevated TSH of 65 mIU/L (milli international units per liter). Patient was recently diagnosed with hypothyroidism and has been started on Levothyroxine. Levothyroxine dose has been recently increased likely due to dose not being therapeutic. Patient was discharged on her home dose of Levothyroxine 75 mcg daily with a reminder of the importance of taking medication daily.
She was scheduled to follow up with oncology for Mets discovered incidentally on MRI. Patient progress was followed. Patient was readmitted a month later after suffering a fall from ground level without any fracture. Cancer has however progressed; patient oxygen requirements have increased, and she decided to go on comfort care and passed away on 10/29/2021.

3. Discussion

Hypothyroid myopathy is a common muscle disease associated with untreated hypothyroidism, affecting about 79 percent of patients, mostly women [4]. It presents with nonspecific symptoms including nonspecific muscle stiffness or cramps, diffuse myalgias, fatigue and muscle weakness. The pathophysiology is not clear; however, a plausible explanation correlates the deficiency in thyroxine with oxidative imbalance leading to muscle injury [5]. This explanation is supported by the signs of symptoms discussed above and the most common lab findings of elevated creatine kinase present in 57-90% of cases. The elevated creatine kinase is not as specific and does not also correlate to the severity of the disease as patients can develop myopathy years later despite elevated creatine kinase. It is an often-missed diagnosis for 2 main reasons: failure to inquire about musculoskeletal symptoms and symptoms being labeled as fatigue. The differential diagnosis includes myositis in all its forms (polymyositis, Inclusion body and dermatomyositis), Amyotrophic lateral sclerosis, poliomyelitis, Rhabdomyolysis, and acid maltase deficiency [5].

It should be high on a differential diagnosis for any hypothyroidism patients as presence of hypothyroidism is necessary. In comparison to the other myopathies, muscle biopsy is needed. Measurement of Thyroid Stimulating hormones (TSH) and thyroxine (T4) along with elevated creatine kinase will suffice to prove a diagnosis [6]. Patients do not require referral to an endocrinologist as primary care physicians (PCP) are well equipped to manage them. However, an interprofessional team will offer better success as patients will need physical therapy and frequent monitoring of TSH and T4 levels [7]. Treatment consists of thyroid replacement therapy. Creatine kinase tends to normalize within a few weeks most often prior to TSH levels. Musculoskeletal symptoms can linger for 6-12 months [7].

For the patient in this case, there are possible confounding factors such as radiation and use of immunological therapy, which both can cause elevated creatine kinase. However, in radiation induced elevated creatine kinase occurred within two to thirty years after radiation and tend to be localized in the areas irradiated [8]. As opposed to being generalized as in our case. Immunological therapy has extremely low incidence of myopathy, which is often related to autoimmune which was ruled out through our lab results. Base of all our findings, chart reviews and lab results, the best and most valid cause of the elevated creatine kinase is related to the patient's hypothyroidism and lead us to hypothyroid myopathy especially in the setting of her uncontrolled hypothyroidism supported by the recent increase of her levothyroxine. The fact that the TSH remained elevated and even higher than when we saw her does not give support to our case. TSH level being elevated in disease is well documented and could be applied to our patient as she remained extremely sick. Our case has not been proven 100% as follow up was impossible as our patient was deceased a month later. The case was not proven all the way as our patient was deceased within a month

4. Conclusion

Hypothyroid myopathy often presents with nonspecific symptoms which may include a wide array of differential diagnosis. Elevated creatinine kinase levels occur in 57-90% of patients presenting with hypothyroid myopathy. In this case report, we presented a 63-year-

old female presenting with proximal muscle myalgias and elevated CK> 2000 and an elevated TSH of 65 mIU/L. Autoimmune workup was negative for inflammatory idiopathic myopathies. Respiratory panel was also negative for infection related causes of elevated creatinine kinase. EMG and muscle biopsy were not used in this case report. treatment consists of managing the hypothyroidism with appropriate thyroid hormone replacement and restoration of euthyroid function. Creatine kinase is expected to fall with thyroid hormone replacement within a few weeks, but symptoms can take up to 6 months to resolve with complete resolution within a year of appropriate treatment.

References

1. Sindoni, A., Rodolico, C., Pappalardo, M. A., Portaro, S., & Benvenga, S. (2016). Hypothyroid myopathy: a peculiar clinical presentation of thyroid failure. Review of the literature. Reviews in Endocrine and Metabolic Disorders, 17, 499-519.
2. Solorzano, G. E., & Phillips, L. H. (2011). Inclusion body myositis: diagnosis, pathogenesis, and treatment options. Rheumatic Disease Clinics, 37(2), 173-183.
3. Castro, C., & Gourley, M. (2012). Diagnosis and treatment of inflammatory myopathy: issues and management. Therapeutic advances in musculoskeletal disease, 4(2), 111-120.
4. Rodolico, C., Bonanno, C., Pugliese, A., Nicocia, G., Benvenga, S., & Toscano, A. (2020). Endocrine myopathies: clinical and histopathological features of the major forms. Acta Myologica, 39(3), 130.
5. Fariduddin, M. M., & Bansal, N. (2022). Hypothyroid Myopathy. 2021 Dec 19. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing.
6. Vignesh, G., Balachandran, K., Kamalanathan, S., & Hamide, A. (2013). Myoedema: A clinical pointer to hypothyroid myopathy. Indian Journal of Endocrinology and Metabolism, 17(2), 352.
7. Mangaraj, S., & Sethy, G. (2014). Hoffman's syndrome–A rare facet of hypothyroid myopathy. Journal of Neurosciences in Rural Practice, 5(04), 447-448.
8. Zuckner, J. (1994). Drug-related myopathies. Rheumatic Disease Clinics of North America, 20(4),1017-1032.