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BMH Med. J. 2018;5(4):108-110 **Case Report**

Rhabdomyolysis Due To Hashimoto's Thyroiditis Aggravated By Asthma Exacerbation

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Abstract

Hashimoto's thyroiditis is the most common form of primary hypothyroidism. Patients may present with muscular manifestations like weakness, stiffness, pain and elevated muscle enzymes. Rhabdomyolysis has been reported in severe asthma. This is a case of a middle aged male, who presented with acute exacerbation of asthma accompanied by severe muscle aches and pain, and was found to have rhabdomyolysis. Following treatment for asthma, his symptoms of dyspnoea and muscle aches decreased, but rhabdomyolysis was persistent. On further evaluation, he was found to have Hashimoto's thyroiditis. He was managed with intravenous fluids and levothyroxine along with nebulisations and intravenous steroids, following which he improved.

Key words: Hashimoto's thyroiditis, hypothyroidism, rhabdomyolysis, creatine kinase, asthma

Case presentation

A middle aged male presented with wheezing and breathlessness since morning. He is a known asthmatic on inhalers (formoterol and budesonide). He has also been experiencing muscle aches for the past 4 months; with exaggeration of symptoms during asthma attacks. There were no other significant or associated symptoms.

On examination, he was conscious, oriented and afebrile. He was overweight with BMI of 27 Kg/m^2. His pulse rate was 60/minute, blood pressure 140/90 mmHg and respiratory rate 30/ minute with saturation 90% (in room air). His respiratory system revealed bilateral diffuse rhonchi. Neurologically, he had sluggish deep tendon reflexes but no muscle weakness. Other general and systemic examinations were normal.

His complete hemogram showed eosinophilia (total counts 9000 cells/cmm with neutrophils 58%, lymphocytes 30%, eosinophils 12%) with normal haemoglobin and platelet counts. Absolute eosinophil count and serum IgE levels were elevated (1080 cells/cmm and 990 IU/ml respectively). Peripheral smear also revealed eosinophilia. Renal functions were deranged with urea 44 mg/dL (10-40) and creatinine 3.81 mg/dL (0.9-1.3). His liver functions, electrolytes, calcium and HbA1c were

normal. His fasting lipid profile showed dyslipidemia with LDL 197 (< 130), HDL 30mg/dL (40-60), VLDL 36mg/dL (6-38) and triglycerides 285mg/dL (< 150). Low voltage complexes were seen on ECG. Echocardiography and chest X-ray were normal. His CK and LDH levels were elevated, 1055 U/L (24-190) and 339 U/L (114-240) respectively. Urine examination was normal, with no myoglobinuria or haematuria. Troponin I and ANA profile were negative. His thyroid profile was suggestive of Hashimoto's thyroiditis, with TSH 66.59 μIU/ml (0.40-4.20), T3 34.6 ng/dL (40-181) and T4 2.44 μg/dL (4.60-10.50), and positive antithyroid peroxidase antibody of 176 U/ml (< 35). Ultrasound of neck revealed features suggestive of thyroiditis.

He was managed with salbutamol, budesonide and ipratropium bromide nebulisations, along with montelukast and intravenous methyl prednisolone (40 mg q8h). He had two episodes of fever following admission, and was started on oral cefuroxime (250 mg twice daily) empirically. Oral levothyroxine was given at 125 mcg once daily, along with aggressive intravenous fluid administration.
By day 6 of admission, his creatinine levels started normalizing; and he was discharged on day 10 with a normal renal functions. Since the patient had improved symptomatically, electromyography and muscle biopsy were not done. His discharge medications comprised of levothyroxine 125 mcg (once daily), montelukast 10 mg (once daily), acebrophylline 100 mg (twice daily), and formoterol-budesonide inhaler. On follow-up after 2 weeks, the patient was asymptomatic. His blood investigations showed a normal thyroid profile, CK levels and renal functions.

Discussion

Rhabdomyolysis can manifest as myalgia, limb weakness and swelling along with passing of dark coloured urine in the absence of haematuria. Myoglobinuria may be present. The condition is characterised by muscle necrosis and release of intracellular muscular components like myoglobin, creatine kinase (CK), lactate dehydrogenase (LDH), aldolase, aspartate aminotransferase, and potassium into the circulation. Rhabdomyolysis can have a wide range of presentation; from asymptomatic elevations in serum muscle enzymes to acute kidney injury. Trauma is the most common aetiology; and non-traumatic causes include seizures, endocrine disorders, heat exposure, electrolyte imbalance, infections and heavy exercise.

Hashimoto's thyroiditis (HT) is a form of primary hypothyroidism, wherein the gland is destroyed by the immune system. Proximal muscle weakness, pain or cramps are some of the musculoskeletal symptoms associated with hypothyroidism. Alcoholism, diabetes, liver and renal disease, and old age are some of the risk factors associated with hypothyroid myopathy. Rhabdomyolysis as a result of hypothyroidism is a rare scenario; and can be precipitated by trauma, exercise, alcohol and drugs like statins[1].

Various hypotheses like induction of insulin-resistant state, impaired mitochondrial oxidative metabolism, decreased muscle carnitine levels andautoimmune mechanism have been proposed with regard to rhabdomyolysis in hypothyroidism [2,3]. Thyroxine deficiency can lead to abnormal glycogenolysis, mitochondrial oxidative metabolism and triglyceride turnover; which in turn can result in impaired muscle function by causing a transition of fast-twitching type 2 muscle fibres to slow-twitching type 1 fibres, low myosin ATPase activity and low adenosine triphosphate turnover in the skeletal muscles [4].

Rhabdomyolysis secondary to status asthmaticus was first described by Chugh et al. in 1978; following which only a handful of cases have been reported [5]. Severe respiratory muscle exertion can cause mechanical and thermal muscle injury and ATP depletion. Generalized hypoxemia secondary to status asthmaticus can result in muscular ischemia and injury. Metabolic acidosis following hypercapnia can add on to the situation. The combination of respiratory and metabolic acidosis may aggravate the risk of corticosteroid-induced myopathy. Electrolyte imbalances are seen in status asthmaticus; and of these hypokalemia is the most notorious, as it can depolarize the muscle

membrane, inhibit the production and storage of glycogen in myocytes, and even limit vasodilatation in the muscle microvasculature during exercise. Asthma can be triggered by lung infections. These infections can also precipitate rhabdomyolysis by direct tissue damage, effect of toxins and inflammatory mediators, or associated fever, acidosis and dehydration. Drugs like glucocorticoids, theophylline and beta 2-Adrenergic agonists can cause myopathy, but rarely rhabdomyolysis. Antibiotics like levofloxacin and clarithromycin, which are used during lung infection induced asthma exacerbations, may induce rhabdomyolysis.

The patient being discussed had symptoms of muscle aches initially, which were exaggerated during asthma attacks. Following treatment with glucocorticoids and nebulisation, his respiratory symptoms improved, but his muscle aches showed only mild decrease. He was found to have rhabdomyolysis, which was initially thought to be due to asthma exacerbation; but further evaluation revealed an underlying Hashimoto's thyroiditis. Rhabdomyolysis secondary to hypothyroidism is a rare scenario.

Conclusion

Rhabdomyolysis has been observed in Hashimoto's thyroiditis and during severe asthma attacks. Since both hypothyroidism and asthma can precipitate rhabdomyolysis, this case highlights the importance of assessing the thyroid status of all asthmatic patients presenting with muscle aches.

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