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AN ATYPICAL PRESENTATION OF MYASTHENIA GRAVIS: A CASE REPORT

Neurology

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ABSTRACT

Neuromuscular connections are damaged in myasthenia gravis (MG), an autoimmune disorder that falls on a range. Females are more likely to be affected by this uncommon condition. Dysphagia, dysphonia, and fatigable fluctuating diplopia or ptosis are the signs of the customary show. In spite of its unique case, this issue might affect any arrangement of skeletal muscles, remembering those for the neck and proximal appendages. We know of no one who has experienced isolated neck weakness as MG symptoms. The female patient, who was 81 years old, came in with a slight soreness and weakness in her neck that had gotten worse throughout the day. The examination only showed a decrease in motor strength in the cervical muscles. Except for marginally expanded creatine phosphokinase (CPK) levels (350 U/l) and against acetylcholine receptor antibodies (restricting Stomach muscle 12.04 nmol/L, hindering Abdominal muscle 52% while changing Stomach muscle 84%), any remaining imaging and lab tests were inside typical reaches. The patient received pyridostigmine (Mestinon) 60 mg QID, which quickly and significantly alleviated her neck weakness. Following two years of treatment, the patient's condition has not worsened. Atypical symptoms of MG may sometimes manifest in older women, but the condition is more often seen in middle-aged female populations. To minimize investigational expenses and patient suffering, clinicians should be on the lookout for myasthenia symptoms such as fatigable muscular weakness.

KEYWORDS

neck, weakness, atypical, elderly

INTRODUCTION

Myasthenia gravis (MG) is a common autoimmune disorder in middle-aged women [1]. The myoneural intersection and the post-synaptic layer are harmed by supplement obsession in this condition, which is intervened by a sort II immunizer reaction. The antibodies target nicotinic acetylcholine receptors that are post-synaptic. The inability of action potentials to go across neurons causes neuromuscular weakness in the absence of stiffness [2]. In a classic model, anticholinergic autoantibodies attack the muscles outside of the eye, causing the muscular fatigability to fluctuate and most often leading to ptosis and bilateral diplopia, which tend to be more severe in the evenings. It accounts for almost 50% of the instances [1]. Typically seen in older men, bulging muscles that cause difficulty swallowing and speaking have not been reported as the first symptom [2].

Presentation Of The Problem

Our patient is an 81-year-old woman with a long list of health issues, including right hip degenerative joint degeneration, diabetes, and high blood pressure. She complained of a two-week period of neck weakness, which she characterized as an unsteadiness in holding her head up straight. It started without any prior illness, injury, or major stressor. A sudden onset of neck weakness has been going on, and it's just getting worse today. I've been experiencing slight discomfort, and I can't seem to find anything that's making it worse or better. The patient's inability to fully extend her neck was noted by the nursing home staff. There were no signs of regurgitation,diplopia, ptosis, photophobia, dysphagia, dysarthria, dysphonia, chills, fever, or difficulty breathing. With the exception of hypothyroidism in the mother, there is no notable history of autoimmune illnesses in the family.

Extensive testing showed that the muscles in the neck were only 3/5 strong, but that the muscles in the arms and legs were perfectly OK. There were no positive signals from Spurling and Lhermitte. There was no evidence of a sensory impairment. There was a lack of Babinski signal and symmetrical deep tendon reflexes throughout. Degenerative joint disease, compression fracture of the spine, dystrophy of the muscles, neuromuscular illness, and paraneoplastic process were all possibilities at this stage in the diagnostic procedure. The primary blood tests returned typical. The patient had a cervical spine X-ray in addition to a metabolic panel and blood tests for inflammatory markers. The X-ray showed no abnormalities, and the full metabolic profile and complete blood count (CBC) showed no signs of illness or inflammation. With the exception of inflammatory conditions affecting the muscles, liver function tests (LFTs) revealed normal levels of AST, ALT, and ALP. As for autoimmune markers, they were also normal. The erythrocyte sedimentation rate (ESR) was 15 mm/hr, and serum creatine phosphokinase (CPK) levels were somewhat high at 350 U/l (compared to the normal 22-198 U/l).

The patient returned fourteen days after the fact, this time whining of demolishing neck shortcoming and an expansion in the troubles she had in keeping her neck unmoving. A chest X-ray revealed no abnormalities, including the absence of thymoma. These results are in accordance with a diagnosis of myasthenia gravis: Anti-acetylcholine receptor bindingantibody was 12.04 nmol/L (normally 0.40 nmol/L), blocking antibody was 52% (normally 26%), and modulating antibody was 84% (normally 45%). Based on the clinical picture and laboratory results, we decided to treat myasthenia with cholinergic agonists rather than electromyography. Metforminon (pyridostigmine) 60 mg once day was started on the patient. Three days later, we told her to come back. Once she started taking pyridostigmine, her symptoms were much better. The progression of the illness was mild. Since then, the patient's weakness has not returned, and she is stable on the medication. There have been no adverse effects from the treatment. The patient was able to go back to her previous way of life without having any problems with her vision, swallowing, speech, or gait.

Subject For Debate

Although myasthenia gravis is the most common neuromuscular junction disorder, the case described above is a very unusual symptom of a condition that is even rarer. Unexplained bulbar or neck shortcoming in the old, particularly without trademark fluctuating shortcoming, ought to raise a high clinical list of doubt for myasthenia, as found in this occurrence. Myasthenic weakness is produced by damaged post-synaptic acetylcholine receptors, which hinder the propagation of action potentials and prevent muscles from depolarizing. There are only around 10–20 new cases of MG per million people each year [3]. The sickness' bimodal conveyance is best displayed in the second and third many years of life for ladies and the fourth and eighth many years of life for men [4].In its classic form, it manifests as fatigue-induced, intermittent weakening of the skeletal muscles, most often affecting the muscles that move the eyes and, to a lesser degree, the muscles that chew food. Myasthenia may quickly develop to respiratory failure and manifests itself in older men as dysphagia, according to some findings [2]. Any group of muscles, including those in the neck and proximal limbs, may be affected in addition to the usual ones.

In older people, there have been reports of significant delays or frequent misdiagnoses [5].The anticipated time for diagnosis was 4.5 months for those older than 60 years, compared to 2.5 months for those younger than that age group [5]. Osteoporotic vertebral fractures, metastases, degenerative joint disease, disk herniation, and neck weakness in older women are all possible causes. Senile people may be undiagnosed for myasthenia because of the wide variety of conditions that may induce neuromuscular symptoms, such as TIAs or strokes, Horner syndrome, motor neuron disease, neuropathies, and Parkinson's disease [6].

For patients with stable myasthenia gravis, indirect-acting anticholinesterases like pyridostigmine and neostigmine have long been the standard of care [7]. The target survival rate in the elderly may be achieved immediately with extensive treatment with anticholinesterases and steroids [8]. Although thymectomy may help younger people with their symptoms, it is generally not recommended for older patients, particularly those over the age of 60 [6]. Treatments have generally been well-received by elderly patients [7].

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CONCLUSIONS

No instances of myasthenia characterized by weakness in the neck alone have been documented in the community of older women. Misdiagnosis, which may cause further problems, is more common in the elderly. In conclusion, the lack of characteristic symptoms in this instance demonstrates that myasthenia should be considered a potential in older individuals with neck weakness. In order to effectively treat myasthenia, a prompt diagnosis is crucial.

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