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## **RESEARCH ARTICLE**

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# CLINICAL CASE OF MULTISYSTEM ATROPHY WITH PARKINSONISM

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## **Abstract**

Multiple system atrophy (MSA) is a steadily progressive neurodegenerative disease involving the pyramidal system, cerebellum, and autonomic nervous systemx[1]. MSA is an  $\alpha$ -synucleinopathy with specific glioneuronal degeneration involving striatonigral, olivopontocerebellar, and autonomic nervous systems but also other parts of the central and peripheral nervous systems[3]. Clinical manifestations include hypotension, urinary retention, constipation, ataxia, parkinsonism, and postural instability. Multiple system atrophy affects both men and women equally, the first symptoms usually appear after age 53. The etiology of this pathology is unknown, but is associated with the accumulation of bodies containing alpha-synuclein in different parts of the brain[1]. There are two types of MSA. The first type occurs with a predominance of cerebellar dysfunctions: ataxia, postural instability. In the clinical setting of the second type of MSA, symptoms of parkinsonism predominate, such as muscle rigidity, bradykinesia, postural disturbances, non-resting tremor and dysarthria. Also, both types exhibit autonomic disorders. The prognosis for this pathology is unfavorable; after the first symptoms appear, life expectancy is 9-10 years.

**Keywords** Multiple system atrophy (MSA), ataxia, parkinsonism, arterial hypotension, alpha-synuclein, orthostatic hypotension.

## **INTRODUCTION**

At the moment, diagnosis and treatment are limited. The diagnosis of multiple system atrophy is made on the basis of clinical data from a combination of signs of autonomic failure and parkinsonism or cerebellar disorders, as well as MRI data. There is no specific treatment for MSA; only supportive therapy is provided [1]. The use of a combination of antiparkinsonian drugs-levadopa and carbidopa, may be ineffective or provide only minor benefit. None of the available treatments significantly slows the aggressive course of MSA[2]. There is a need to consider new methods to

maintain the quality of life of a patient with MSA at an optimal level, prevent complications and increase the life expectancy of patients.

Purpose of the study. To describe a clinical case of Multiple System Atrophy with a predominance of parkinsonian symptoms in old age.

## RESEARCH OBJECTIVES

- 1. Analyze the theoretical concepts of Multiple System Atrophy
- 2. Carry out a theoretical analysis of symptoms, methods of diagnosis and treatment of patients

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with MSA.

Materials and methods. Patient G., 62 years old, was admitted with complaints of slow movements, decreased strength in the arms and legs, a drop in blood pressure, dizziness, instability when walking, and intermittent tremor in the arms. The above symptoms have been bothering him for 2 years. Life history: without any developmental features.

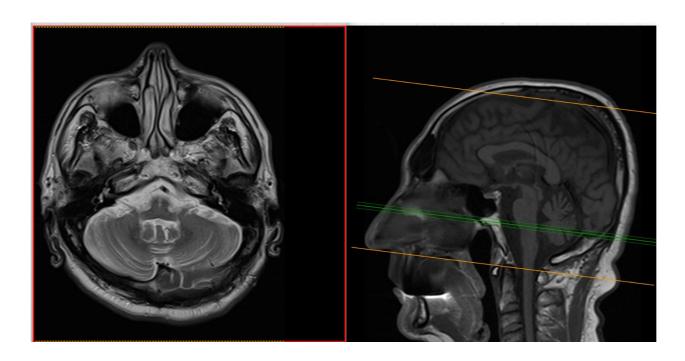
During the examination, neurological examination: the patient is conscious, oriented in time and place. Blood pressure = 90/60 mm Hg. The sensitive area is unchanged. Eye movement is complete. There are no vertical seconds. The pupils are the same D=S. Nystagmus is absent. There are no face zone triggers. The face is symmetrical. There is no smoothness of the nasolabial triangle. The tongue occupies a central position, the uvula in the center. There are no swallowing disorders. The pharyngeal reflex is evoked. Hearing is not impaired. Muscle tone is reduced. Tendon reflexes: evoked from the

arms and legs. Muscle strength is weakened. Static coordination tests are unsatisfactory. Romberg test is positive. Hyperkinetic syndrome - no. Scoliosis of the spinal column - no. Movement in the limbs is limited. There are no meningeal symptoms.

Bradykinesia, muscle rigidity, increasing after functional tests. During the examination, Levadopa 125 mg was given. Functional tests were performed 45 minutes after taking the tablet. Based on complaints, medical history, and initial examination, a preliminary diagnosis was made: Parkinson's disease. Multiple system atrophy (questionable). At the outpatient stage, an MRI of the brain was prescribed.

## **RESULTS**

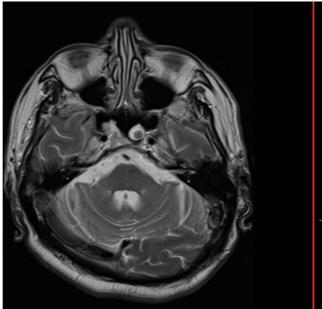
The patient's condition did not improve while taking Levadopa 125 mg, blood pressure = 80/40 mmHg, and when performing functional tests, rigidity in the limbs did not decrease.

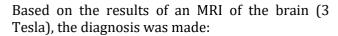


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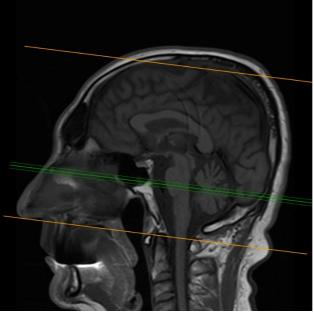
Multiple system atrophy; there are signs of atrophy in the pons, midbrain and cerebellum in T1 mode.

Diagnostic criteria for making this diagnosis are: atrophic changes in the putamen, middle cerebellar peduncles, pons and cerebellum. In addition, MSA is characterized by the "cross" sign, often described in degenerative spinocerebellar ataxias, hyperintensity of the dorsolateral edge of the putamen in combination with hyperintensity of the "vertical" signal from the suture [4]. Laboratory markers of this disease are: an increase in the level of total  $\alpha$ -synuclein and homocysteine, a decrease in the level of coenzyme Q10 and uric acid [5].

Antihypotensive treatment was prescribed after consultation with a cardiologist. Antiparkinsonian drugs were not prescribed due to their ineffectiveness.

## **CONCLUSION**

This clinical example shows the features of the course, difficulties of diagnosis and treatment of Multiple system atrophy in an elderly patient. Taking into account the high risk of complications in such patients and the similarity of clinical and instrumental signs, it was necessary to conduct a



differential diagnosis with the typical form of Parkinson's disease, dementia with Lewy bodies, true autonomic failure, autonomic neuropathies, progressive supranuclear palsy, multiple cerebral infarctions and drug-induced parkinsonism.

In the last few years, due to research and technology development, a number of promising imaging and laboratory markers have been identified for the comprehensive diagnosis of MSA, including at the initial stages of the neurodegenerative process.

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