

CASE REPORT

A Case of Multiple System Atrophy (MSA) – First Presentation in a Psychiatry Service

Elena Alina ROSCA¹, Andra Livia BRONESCU¹, Ovidiu Iulian TUDOR²

Abstract

A 67 years old female developed cognitive deficits, depression and anxiety as first symptoms of multiple system atrophy (MSA). In the course of an year autonomic failure, parkinsonism and cerebellar ataxia also developed. The case is particular because of the somatic symptoms that were initially categorized as psychogenic, the presence of confusional and vertigo episodes that were recurring but not permanent.

Keywords: cognitive deficits, depression, anxiety, multiple system atrophy, psychiatry.

¹"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

²"Regina Maria" Hospital, Bucharest, Romania

Corresponding author:

Elena Alina ROSCA, „Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania.

INTRODUCTION

Multiple system atrophy (MSA) is a rare progressive neurodegenerative disease with adult onset caused by cell loss, gliosis, glial cytoplasmic inclusions in striato-nigral olivopontocerebellar and autonomic systems. It is characterized clinically by parkinsonism, cerebellar ataxia, autonomic failure and corticospinal signs with poor or no response to levodopa ¹. Female gender, longer disease duration, and more serious disease severity represent common predictors for depression and anxiety found in MSA ².

CASE PRESENTATION

We present a case of a female patient (67 years old) without psychiatric history, who was observed over the course of 6 years (2015–2021). She reported no alcohol, tobacco or drug abuse and no family history of psychiatric disorders.

September 2015: Patient was admitted to our hospital for depressed mood, mild attention and memory deficits, difficulties maintaining sleep, fluctuating appetite, paresthesia, fatigue and recurrent episodes of tremor, sweating, dizziness, nausea and vomiting.

Blood analysis was performed with the following results: blood count and leukocyte formula in normal ranges, normal thyroid function, glucose in normal range, normal ionogram, liver and renal function normal except a mild increase of TGP (39 U/L) and no inflammatory markers identified. Abdominal ultrasound showed postcholecystectomy dilatation of the bile duct.

MRI scan showed millimetric white matter hyperintensity lesions on T2 and fluid attenuated inversion recovery (FLAIR) in fronto-parietal cortex (demyelinizations, gliosis), no diffusion restrictions, mild supratentorial atrophy more emphasized in bilateral fronto-parietal cortex with secondary deepening of fronto-parietal sulci and interhemispherical fissure, deepening of cerebral sulci. MRI results were interpreted as most probably having a microangiopathic cause. EEG was normal.

Patient was diagnosed with moderate depressive episode, mild cognitive impairment and unspecified polyneuropathy and was treated with a 10 day-cure of intravenous nootropic agent Cerebrolysin and a long-term treatment with tianeptine 37,5 mg/day and gabapentine 600 mg/day was initiated.

December 2015: Patient returned for the persistence of neurocognitive deficits and depressed mood and new symptoms: increased anxiety, irritability, anhedonia, thought of hopelessness and worsening of the neurovegetative and gastrointestinal symptoms. She reported partial compliance to the psychiatric treatment. We decided to reinstate the same psychiatric treatment together with low doses of lorazepam (1–2 mg/day) and continue investigating possible somatic comorbidities.

February 2016: Patient was admitted for follow-up. Depressive symptomatology worsened, patient showed marked psycho-motor inhibition with sad facies, slow movements and hypophonia. Pathological crying was reported: „I have frequent episodes that last a few seconds when I feel my head going soft, dizziness, nausea, I start to cry and shake.”. Head CT scan showed moderate cerebral and cerebellar atrophy. We maintained the nootropic treatment and decided to switch the antidepressant medication to Escitalopram 10 mg/day.

March 2016: Patient underwent an internal medicine examination after 10 days of digestive intolerance, nausea, vomiting and epigastric pain. Blood tests excluded liver or pancreatic disease, blood count and ionogram were normal. Abdominal CT scan: Postcholecystectomy syndrome. Upper endoscopy: Erythematous gastritis. Final diagnosis was psychogenic vomiting.

April 2016: Recurring episodes of dizziness and pathological crying became more frequent and intense and associated confusion. Vertigo was more intense in orthostatic position and during left-right head movements. Diagnosis: Recurring nonspecified vestibular disorder.

June 2016: Patient was admitted for follow-up. Psychological tests showed: MMSE=22/30, ADL=6/6, IADL=6/8, GDS Reisberg stage=4, W-M questionnaire with pathological scores for depression, anxiety, hypochondria and instability. Confusional and vertigo episodes worsened.

Blood pressure was 110/60 mmHg in clinostatism and 80/50 mmHg in orthostatism. Ambulatory blood pressure test results: mean diurnal BP=102/61 mmHg, mean nocturnal BP=82/46 mmHg. Cervical Doppler ultrasound: carotidian atheromatosis with no hemodynamic consequences. Neurological examination showed: no coordination disorder, no motor or sensitivity deficits, positive Romberg test, bilateral hand tremor, positive bilateral Noica sign, normal osteo-tendinous reflexes, abnormal posture reflexes. Diagnosis: Parkinson-like syndrome in observation. Orthostatic

and nocturnal hypotension. Carotidian atheromatosis.

Patient showed no response to Escitalopram therefore we switched to Venlafaxine 37.5 mg/day. Treatment with Midodrinum 5 mg/day and Vincaminum 60 mg/day was initiated.

September 2016: All medical data were gathered and the diagnosis of multiple system atrophy was formulated. Levodopum 250 mg/Carbidopum 25 mg and Risperidone 0.5 mg were added to the patient treatment.

March 2020: Patient returned for follow-up after 3,5 years of slowly progressive disease and showed: no ability to walk, FMS scale 4/5 superior loins, 2/5 inferior loins, ADL=2/6, IADL=0/8, urinary and fecal incontinence, dysmetria. Psychiatric examination showed temporal and spatial disorientation, severe attention and memory deficits (MMSE=9/30), bradypsyhia, bradylalia, depressed mood, visual and auditive hallucinations, episodes of psycho-motor agitation., GAFS=10/100. Diagnosis of dementia was formulated and treatment with Rivastigmine was initiated. The disease progressed to exitus by the beginning of 2021.

CONCLUSION

Even though it is a rare disease, MSA should be considered when excluding medical causes of depression and anxiety. Dementia is not considered a common characteristic of MSA but attention and memory deficits were also found in this case.

Compliance with ethics requirements:

The authors declare no conflict of interest regarding this article. The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from all the patients included in the study.

References

1. Gilman S, Wenning GK, Low PA, Brooks DJ, Mathias CJ, Trojanowski JQ, Wood NW, Co- losimo C, Dürr A, Fowler CJ, Kaufmann H, Klockgether T, Lees A, Poewe W, Quinn N, Revesz T, Robertson D, Sandroni P, Seppi K, Vidailhet M: Second consensus statement on the diagnosis of multiple system atrophy. *Neurology* 2008;71:670–676.
2. Zhang, L.-Y., Cao, B., Zou, Y.-T., Wei, Q.-Q., Ou, R.-W., Zhao, B., ... Shang, H.-F. (2017). Depression and anxiety in multiple system atrophy. *Acta Neurologica Scandinavica*, 137(1), 33–37. doi:10.1111/ane.12804