# A 15-year journey to total physical disability; a case of Astasia-abasia

Frank Mulindwa[1,2*], Maheen Irshad[1], Kanza Ahmed[1], Rabia Malik[1], Ali Javed[1], Hadia Waheed[1], Yasir Ahmed[1]

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## ABSTRACT

**Background:** The diagnosis of functional movement disorders, including Astasia-abasia has always been a challenge even with seasoned clinicians due to a tendency to border on caution to avoid missing potentially life-threatening structural neurological disorders, leading to delays in diagnosis. Chronicity of symptoms, a guarded prognosis, and eventual physical conditioning pose treatment challenges.

**Case Presentation:** We present a case of a 69-year-old Caucasian man with past medical history of essential hypertension, well controlled type II diabetes mellitus, hyperlipidemia and generalized anxiety disorder who was evaluated in three hospital systems for what he described as "difficulty standing in one position" and later gait disturbances that was incongruent with findings on neurological examination and well as imaging, nerve conduction studies and electromyography. We describe his clinical course to becoming wheelchair bound, eventually having multiple hospital admissions due to life-threatening complications of immobility, including decubitus ulcers, sepsis, and rhabdomyolysis leading to acute kidney injury.

**Conclusion:** The presented case of Astasia-abasia highlights diagnostic challenges involving repetition of diagnostic tests as patients move from one hospital system to another, as well as unpredictability of treatment response and the potential for severe physical deconditioning leading to medical complications of immobility.

**Keywords:** Functional movement disorders, Astasia-abasia, physical disability.

**Type of Article:** CASE REPORT **Speciality:** Neurology

**Correspondence to:** Frank Mulindwa
*Department of Internal Medicine, United Health Services Hospitals, Wilson Hospital Johnson City, New York, USA and Makerere University Infectious Diseases Institute, Kampala, Uganda.
**Email:** mulindwafrank93@gmail.com
*Full list of author information is available at the end of the article.*

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## Background

Astasia (inability to stand) abasia (inability to walk) belong to a group of conditions known as functional movement disorders (FMDs), a subtype of functional neurological symptom disorder also known as conversion disorder [1]. The diagnosis of FMDs, including Astasia-abasia is always a challenge as clinicians border on caution to avoid missing potentially life-threatening structural neurological disorders, leading to delays in diagnosis. Early recognition of these disorders is pivotal to having favorable outcomes, as multidisciplinary care can be initiated early, preventing both physical and psychological disability.

## Case Presentation

A 69-year-old Caucasian man with a past medical history of essential hypertension, well-controlled type II diabetes mellitus, hyperlipidemia, and generalized anxiety disorder presented to a tertiary hospital following 5 years of symptoms that he described as "I cannot hold myself up while standing in a stationary position". His home medication included: amlodipine, ezetimibe, folic acid, metformin, and metoprolol. He reported no family history of neurological disorders or any auto-immune conditions.

He reported the symptoms were absent when he was walking or running and was able to go to the gym, running up to 5 miles an hour on a treadmill. The symptoms started over days to weeks. He stated that he had to keep shuffling his feet to maintain balance. He had been evaluated at symptom onset (5 years earlier) at another tertiary hospital, where a non-contrasted magnetic resonance imaging (MRI) of the brain had been performed and found to be normal. His medical records also showed that he had had cerebrospinal fluid studies and an electromyogram that were quoted as much as specific laboratory reports were not available for reference. His long-term prescription of atorvastatin was withheld at that hospital due to suspicion for possible early-stage statin-induced myopathy with normal serum creatine kinase levels, but still he had not improved. Two years prior to the current visit (3 years after symptom onset), he had been involved in a motor

accident where his vehicle was rear-ended in a shopping mall parking lot and got what he described as a "whiplash injury". He did not lose consciousness, had neck pain, and was "foggy" for a few minutes after. He reported that his previous symptoms worsened after the accident that he had trouble supporting himself in a sitting position. He reported no associated headaches, no back pain, no loss of sensation, no loss of bowel or bladder control, no constitutional symptoms, no changes in vision, no muscle pain, no loss of muscle mass, no skin changes, and no memory changes.

He appeared well nourished, in no apparent distress. Central nervous system exam: pupils were equal and reactive to light. Cranial nerves 2-12 were intact. He had normal muscle bulk, no fasciculations, power was 5/5 in all flexor and extensor muscle groups in all limbs, and he had normal tone. Sensation to light touch, vibration, and crude touch was normal. Biceps, triceps, brachioradialis, and knee jerk reflexes were 2+ (normal), ankle jerk reflex 1+. He had down-going plantar reflexes (normal). Finger to nose test was normal with a slight tremor at the end, had an intact heel to shin test. When sitting unsupported, the patient swayed and wanted to lean back, becoming short of breath.

Gait: When standing still, he would become short of breath and asked to take short steps around. Once he started walking, his gait was normal, and was no longer short of breath. He was able to walk heel to toe, as well as able to rise from a sitting position without arm support.

A repeat non-contrasted brain MRI was normal. A non-contrasted MRI of the lumbar spine showed a slightly asymmetric bulge on the right L4-L5 without nerve root compression, which could not explain his symptoms. Electromyography (EMG) and nerve conduction studies were also normal. Laboratory studies, including vitamin B12 levels, thyroid-stimulating hormone, anti-nuclear antibodies, rapid plasma reagin, full hemogram, renal and liver function tests, were normal. A lumbar puncture was done with normal opening pressure. Cerebrospinal fluid (CSF) studies done were all within normal limits, including absence of oligoclonal bands and a negative CSF paraneoplastic panel.

The clinical impression after evaluation was suspicious for a psychogenic cause of the symptoms and very unlikely due to a structural neurogenic or myopathic cause. He was advised to restart atorvastatin, start physiotherapy, and continue anxiety treatment with as needed lorazepam, which he already was using.

Four years later, in another hospital system (9 years from symptom onset), he was re-evaluated with similar symptoms, and a lumbar spinal MRI was repeated, which turned out normal. A diagnosis of Astasia-abasia was made in that institution, and he was started on sertraline in addition to lorazepam, physiotherapy, occupational therapy, as well as serial psychiatric evaluations.

### *Progress over time*

He was agreeable to the diagnosis and was very compliant with medical reviews with fluctuating severity of symptoms over different clinical visits. Six years after the establishment of the diagnosis (15 years from symptom onset), the patient reported a diminished response to medication and physiotherapy (Table 1). He became wheelchair bound first in a manual wheelchair and later

***Table 1.*** *Timeline of the patient's clinical course.*

| TIMELINE | EVENT | ACTIONS |
|---|---|---|
| Symptom onset<br>(Five years before presenting to our hospital) | -Subacute onset of motor neurological symptoms<br>-He was initially evaluated at another tertiary hospital | Initial tests including MRI brain, CSF studies and EMG evaluation were reported normal |
| Three years after symptom onset<br>(2 years prior to presentation to our hospital) | -He was involved in a minor road traffic accident<br>-He noted worsening symptoms after the accident | |
| Five years after symptom onset<br>(presentation to our hospital) | -He presented to seek a second opinion | Repeat work up was within normal limits including:<br>MRI brain and total spine, EMG, CSF studies including an autoimmune panel, syphilis, vitamin B12 studies) |
| Nine years after symptom onset<br>(4 years after initial evaluation at our hospital) | -He sought care at another tertiary hospital | Most of the above laboratory and imaging tests were repeated, again with negative results.<br>A diagnosis of Astasia-abasia was made and treatment commenced |
| Fifteen years after symptom onset<br>(10 years after initial evaluation at our hospital) | -He reported a diminished response from ongoing multi-disciplinary therapies<br><br>-He became wheelchair bound<br><br>-Multiple hospital admissions with: decubitus ulcers, muscle disuse rhabdomyolysis with acute kidney injury, lactic acidosis | |

in an electric wheelchair because he reported reduced arm strength to propel himself. Objective evaluation over the years remained normal with normal cranial nerves, normal sensory system, normal motor system, coordination with gait and stance findings fluctuating and appearing abnormal purposefully.

Over the course of disease, the patient was evaluated in three tertiary hospital systems, had up to 10 MRI evaluations (multiple brain and spine MRIs), multiple electromyograms, as well as multiple nerve conduction studies and lumbar punctures for CSF analysis.

Within 6 months of becoming wheelchair bound, he was admitted twice with complications of immobility including; a grade III decubitus ulcer, psoas abscess, sepsis, muscle disuse rhabdomyolysis with acute kidney injury, and lactic acidosis.

## Discussion

We present a case of a FMD, Astasia-abasia' highlighting diagnostic challenges, a tendency for over investigation, and eventual potentially life-threatening complications from the resultant physical deconditioning.

Astasia (inability to stand) abasia (inability to walk) belong to a group of conditions known as FMDs, a subtype of functional neurological symptom disorder also known as conversion disorder [1]. Patients have gait and positional symptoms with variable clinical signs that are incongruent with known neurological pathology. Median age of presentation is 40 years, but there are case reports of children presenting as early as 6 years and in older adults, and they are more common in females accounting to up to 60%-90% [2]. There is no clear-cut etiological factor, but it is believed that a constellation of bio-psychosocial factors is involved, with predisposing factors being; younger age, female sex, presence of other neurological conditions/health conditions, difficulties in interpersonal relationships, and psychiatric illnesses. Precipitating factors may include physical injuries, surgery emotional events, and the condition can be perpetuated by the presence of factors including untreated anxiety, litigation, physical disability benefits, psychosocial stress, and physical deconditioning [3,4]. The diagnosis involves taking a comprehensive history, performing a thorough neurological physical examination, including blood/CSF tests/EMG, nerve conduction studies, as well as neuroimaging to rule out alternative possible causes.

The diagnosis of FMDs is always a challenge. Even with suggestive history and examination findings, seasoned clinicians border on caution to avoid missing potentially life-threatening structural neurological disorders, leading to delays in diagnosis[5-7]. The patient we present had symptoms for 9 years before a definitive diagnosis was established, much as on prior reviews, FMDs were suspected. Age alone should not be used to exclude FMDs. The age of onset of the patient's symptoms was 64 years, largely above the reported average age of symptom onset, which could have downplayed clinical suspicion. This is not uncommon though. In a reported retrospective cohort of 117 patients with FMDs in India, 18 were above 60 years at symptom onset [4,8]. The delay in diagnosis led to delays in targeted multi-disciplinary treatment, a well-documented contributor to poor prognosis [9,10].

Variable treatment success rates have been reported, all anchored around having a multi-disciplinary approach involving physical therapy, occupational therapy, cognitive behavioral therapy, neuropsychiatry assessments, and neurology inputs. Patients should be educated about their diagnosis, as having insight improves treatment adherence. Early psychiatry referrals not only help in the establishment of the diagnosis but also in the treatment of comorbid conditions, which may include anxiety, depression, post-traumatic stress, personality traits, and atypical presentations of psychosis [11,-13]. The patient we present largely received the required therapies from multiple disciplines once the diagnosis was made. However, the pitfall was a late diagnosis, which may have been a limitation to treatment success.

Prognosis for FMDs is usually guarded, with many patients facing long-term disability, underlining the importance of clinicians to realize that the absence of a structural lesion does not mean the problem will eventually resolve on its own [14].

## Conclusion

The presented case of Astasia-abasia highlights diagnostic challenges involving repetition of diagnostic tests as patients move from one hospital system to another. We also highlight the unpredictability of treatment response and the potential for severe physical deconditioning leading to severe life-threatening medical complications of immobility. Early diagnosis is imperative to prevent irreversible physical disability as well as mitigate the costs involved in unnecessary investigations.

### What's new?

The authors present a case report of an atypical presentation of a functional movement disorder, Astasia-abasia, spanning 15 years, highlighting life-threatening complications at the end of the disease spectrum.

### List of Abbreviations

| | |
|---|---|
| CSF | cerebral spinal fluid |
| FMD | functional movement disorders |
| MRI | magnetic resonance imaging |

### Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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None.

## Consent for publication

Written informed consent for this publication was obtained from the patient.

## Ethical approval

Ethical approval was granted by the United Health Sciences Institutional Review Board: ID-13499.

## Author details

Frank Mulindwa[1,2], Maheen Irshad[1], Kanza Ahmed[1], Rabia Malik[1], Ali Javed[1], Hadia Waheed[1], Yasir Ahmed[1]

1. Department of Internal Medicine, United Health Services Hospitals, Wilson Hospital Johnson City, New York, USA
2. Makerere University Infectious Diseases Institute, Kampala, Uganda

## References

1. Psychiatry.org - DSM. [Online] [cited 2024 Apr 04]. Available from: https://www.psychiatry.org/psychiatrists/practice/dsm
2. Factor SA, Podskalny GD, Molho ES. Psychogenic movement disorders: frequency, clinical profile, and characteristics. J Neurol Neurosurg Psychiatry. 1995 Oct;59(4):406–12. https://doi.org/10.1136/jnnp.59.4.406
3. Defazio G, Pastore A, Pellicciari R, Pierri G, Gigante AF, Fabio G, et al. Personality disorders and somatization in functional and organic movement disorders. Psychiatry Res. 2017 Nov;257:227–9. https://doi.org/10.1016/j.psychres.2017.07.068
4. Batla A, Stamelou M, Edwards MJ, Pareés I, Saifee TA, Fox Z, et al. Functional movement disorders are not uncommon in the elderly. Mov Disord. 2013 Apr;28(4):540–3. https://doi.org/10.1002/mds.25350
5. Morgante F, Edwards MJ, Espay AJ. Psychogenic movement disorders. Continuum (Minneap Minn). 2013 Oct;19(5 Movement Disorders):1383–96. https://doi.org/10.1212/01.CON.0000436160.41071.79
6. Gupta A, Lang AE. Psychogenic movement disorders. Curr Opin Neurol. 2009 Aug;22(4):430–6. https://doi.org/10.1097/WCO.0b013e32832dc169
7. van der Salm SM, van Rootselaar AF, Cath DC, de Haan RJ, Koelman JH, Tijssen MA. Clinical decision-making in functional and hyperkinetic movement disorders. Neurology. 2017 Jan;88(2):118–23. https://doi.org/10.1212/WNL.0000000000003479
8. Chouksey A, Pandey S. Functional movement disorders in elderly. Tremor Other Hyperkinet Mov (N Y). 2019 Jul;9:1–6. https://doi.org/10.5334/tohm.463
9. Perjoc RS, Roza E, Vladacenco OA, Teleanu DM, Neacsu R, Teleanu RI. Functional neurological disorder-old problem new perspective. Int J Environ Res Public Health. 2023 Jan;20(2):1099. https://doi.org/10.3390/ijerph20021099
10. LaFaver K, LaFrance WC, Price ME, Rosen PB, Rapaport M. Treatment of functional neurological disorder: current state, future directions, and a research agenda. CNS Spectr. 2020 Dec;26(6):1–7. https://doi.org/10.1017/S1092852920002138
11. McCormack R, Moriarty J, Mellers JD, Shotbolt P, Pastena R, Landes N, et al. Specialist inpatient treatment for severe motor conversion disorder: a retrospective comparative study. J Neurol Neurosurg Psychiatry. 2014 Aug;85(8):895–900. https://doi.org/10.1136/jnnp-2013-305716
12. Schrag A, Trimble M, Quinn N, Bhatia K. The syndrome of fixed dystonia: an evaluation of 103 patients. Brain. 2004 Oct;127(Pt 10):2360–72. https://doi.org/10.1093/brain/awh262
13. Jacob AE, Kaelin DL, Roach AR, Ziegler CH, LaFaver K. Motor retraining (MoRe) for functional movement disorders: outcomes from a 1-week multidisciplinary rehabilitation program. PM R. 2018 Nov;10(11):1164–72. https://doi.org/10.1016/j.pmrj.2018.05.011
14. Gelauff J, Stone J, Edwards M, Carson A. The prognosis of functional (psychogenic) motor symptoms: a systematic review. J Neurol Neurosurg Psychiatry. 2014 Feb;85(2):220–6. https://doi.org/10.1136/jnnp-2013-305321

## Summary of case

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|---|---|---|
| 1 | Patient (gender, age) | 69 years/ male |
| 2 | Final diagnosis | Astasia-abasia |
| 3 | Symptoms | Difficulty in standing in one position |
| 4 | Medications | Anxiolytics |
| 5 | Clinical procedure | Physiotherapy, psychotherapy |
| 6 | Specialty | Neurology |