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Case report

Effects of apomorphine, spinal rTMS, and BoNT on camptocormia: an exploratory wearable sensor-based analysis in a patient with MSA

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ABSTRACT

Multiple System Atrophy (MSA) is a progressive neurodegenerative disorder characterized by autonomic dysfunction, parkinsonism, and cerebellar signs. Among its motor manifestations, camptocormia—defined as an abnormal forward flexion of the trunk—often emerges early in the disease course and significantly compromises patients' quality of life. Aside from physical therapy, the treatment of camptocormia remains particularly challenging, especially in MSA, where surgical approaches such as deep brain stimulation and spinal cord stimulation are generally contraindicated. This underscores the urgent need for effective, non-invasive therapeutic strategies. We report a case of MSA presenting with early-onset and severe camptocormia, who underwent sequential treatment with three non-invasive interventions: subcutaneous apomorphine injection, repetitive trans-spinal magnetic stimulation (rTMS), and botulinum neurotoxin (BoNT) injections targeting the abdominal muscles. We aimed to evaluate and compare the efficacy of each intervention using standardized clinical rating scales, instrumented gait analysis, and static posturography. All three interventions resulted in partial improvements, though with varying degrees of efficacy. Subcutaneous apomorphine injection improved walking velocity and step length but was limited by adverse effects (nausea and hypotension), which precluded the use of continuous subcutaneous infusion as a long-term therapeutic option. Spinal rTMS enhanced performance on the Timed Up and Go (TUG) test, particularly during the return phase, but had no significant effect on posture-related measures. BoNT treatment yielded the most substantial improvements in both walking velocity and step length. Moreover, it was the only intervention to improve the posture item of the Unified Multiple System Atrophy Rating Scale (UMSARS) part II. This case underscores the importance of an integrated therapeutic approach in managing camptocormia, emphasizing the role of neurophysiological and biomechanical assessments in identifying biomarkers of therapeutic response and evaluating the efficacy of treatments such as BoNT and neuromodulation.

1. Introduction

Camptocormia is defined as a marked forward flexion of the trunk (>45°) in the sagittal plane, which typically worsens during standing or walking and completely resolves in the supine position (Umapathi et al., 2002). Several conditions may lead to camptocormia, including central nervous system (CNS) and peripheral nervous system (PNS) disorders. Among the CNS disorders associated with camptocormia, Parkinson's Disease (PD) and Multiple System Atrophy (MSA) are the most frequently reported (Finsterer and Strobl, 2010). Although PD and MSA

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can sometimes be difficult to distinguish, camptocormia in PD generally manifests 5–10 years after disease onset and is predominantly observed in advanced stages. Conversely, when camptocormia appears early in patients with parkinsonism, a diagnosis of MSA should be strongly considered (Finsterer and Strobl, 2010; Sławek et al., 2006).

MSA is a progressive neurodegenerative disorder characterized by autonomic dysfunction, parkinsonism, and cerebellar signs. There are currently no disease-modifying therapies available for MSA, and treatment is primarily based on symptomatic and supportive care. Among the motor manifestations, camptocormia represents a therapeutic challenge, with first-line management based on physiotherapy and orthotic support (Perez-Lloret et al., 2015). Further therapeutic approaches can be divided into non-pharmacological, pharmacological, and surgical strategies. Surgical options include spinal cord stimulation (SCS) and deep brain stimulation (DBS), which are used in PD but are generally considered contraindicated in MSA (Agari and Date, 2012; Sako et al., 2009). Among non-pharmacological strategies, trans-spinal non-invasive repetitive magnetic stimulation (rTMS) has been shown to produce immediate relief of camptocormia in patients with PD (Arii et al., 2014; Mitsui et al., 2022). Additionally, botulinum neurotoxin (BoNT) injections in the abdominal muscles and continuous subcutaneous apomorphine infusions have been proposed as alternative strategies (Srivanitchapoom and Hallett, 2016; Mensikova et al., 2015). Preliminary results in PD with camptocormia suggest the need for further investigation of these non-pharmacological treatments, particularly in patients with MSA, for whom surgical interventions are not recommended.

We describe a patient with MSA and early-onset camptocormia who underwent a wearable sensor-based comparative assessment of posture and gait following apomorphine testing, spinal rTMS, and BoNT treatment.

2. Case report

A 67-year-old man presented to our outpatient clinic with hypophonia and unsteady gait since few months. Symptoms of REM sleep behavior disorder had been present several years prior to the onset of motor symptoms. During the following six months, right bradykinesia and cogwheel rigidity appeared. Laboratory test and magnetic resonance imaging with volumetric brainstem measurements of the brain were unremarkable. He was diagnosed with PD and levodopa (LD) 300 mg/die was initiated with improvement in motor symptoms.

Three years after clinical onset, the patient developed a markedly stooped posture characterized by forward flexion of the trunk, which worsened during walking and completely resolved in the recumbent position. The forward flexion of the trunk—diagnosed as camptocormia—was not influenced by LD timing or dosage and was accompanied by abdominal tension, gait disturbances, and postural instability. Furthermore, the patient's speech became severely dysphonic, the response to LD gradually diminished, and severe autonomic features—including urinary urge incontinence and neurogenic orthostatic hypotension—emerged. The previous diagnosis of PD was therefore questioned, prompting further assessments. Dopamine transporter single-photon emission computed tomography showed dopaminergic denervation involving both putamina. Polysomnography confirmed the presence of REM sleep without atonia. Autonomic function testing, including cardiovascular reflex assessment (beat-to-beat heart rate and blood pressure monitoring using Finapres® NOVA) sympathetic skin response and evaluation (Dantec Keypoint G4), revealed severe sympathetic (vasopressor and sudomotor) and parasympathetic (cardiovagal) hypofunction (De Marinis et al., 2000). ^{123}I-metaiodobenzylguanidine myocardial scintigraphy demonstrated reduced cardiac uptake, a feature more typical of PD, though reported in up to 30% of patients with MSA (Nagayama et al., 2010). Due to the presence of more than two red flags, PD was excluded according to the MDS Clinical Diagnostic Criteria for PD, and a diagnosis of clinically probable MSA was established in accordance with the MDS Diagnostic Criteria for MSA (Postuma et al., 2015; Wenning et al., 2022).

At that time, camptocormia was the most disabling symptom and was unresponsive to LD. Consequently, apomorphine testing, spinal rTMS, and BoNT treatment were performed sequentially and compared to establish the most effective therapeutic strategy. The patient was assessed before and after each therapeutic intervention in OFF state using the Unified Multiple System Atrophy Rating Scale (UMSARS) part II, inertial sensor–based Timed Up and Go (TUG) test, inertial sensor–based 10-Meter Walk Test (10MWT), and static posturography (Table 1).

Apomorphine testing was conducted on the same day as the baseline assessment, using a single 3 mg subcutaneous injection. The treatment resulted in improvements in walking velocity and step length compared to baseline. No changes were observed in the posture item of the UMSARS Part II. Moreover, apomorphine testing was associated with nausea and hypotension, thereby precluding continuous subcutaneous infusion via an external pump as a therapeutic option.

Spinal rTMS was initiated the day after the baseline assessment and administered at 08:30 a.m. twice weekly (Mondays and Thursdays) for 4 weeks. rTMS was delivered using a MagPro® stimulator (Medtronic Inc., USA) connected to a butterfly coil (MC-B70; MagVenture). The coil, oriented to the vertical axis, was centered over the spinous process of T12-L1, therefore targeting the lumbar spinal cord enlargement, with the patient seated and the upper limbs relaxed. The stimulation was administered at 5 Hz in 10 trains of 1-s duration, with 10-s inter-train intervals (50 total stimuli) (Mitsui et al., 2022). The patient was assessed before and at the end of the final stimulation session. The treatment was well tolerated, and no side effects were reported. An improvement in TUG test performance, particularly during the return walking phase, was noted. No changes were observed in the posture item of the UMSARS Part II, despite the patient reporting a subjective improvement in camptocormia.

Treatment with BoNT was administered one month after the baseline assessment. Surface electromyography (sEMG) of the rectus abdominis, obliquus externus abdominis, and lumbar paravertebral muscles was

Table 1
UMSARS Part II, TUG, 10MWT, and static posturography scores at baseline and after apomorphine, spinal rTMS, and BoNT treatments. UMSARS, Unified Multiple System Atrophy Rating Scale; TUG, Timed Up and Go; 10MWT, 10-Meter Walk Test; R, right; L, left; EO, eyes open; EC, eyes closed.

	Baseline	Apomorphine	Spinal rTMS	BoNT treatment
UMSARS part II - Total	14	14	14	13
UMSARS part II - Posture	3	3	3	2
UMSARS part II - Body sway	1	1	1	1
UMSARS part II – Gait	1	1	1	1
TUG	13.16 s	13.74 s	11.33 s	13.42 s
Sit-to-stand phase	1,95 s	1,92 s	1,84 s	2,08 s
Forward walking phase	2,99 s	3,02 s	2,94 s	2,69 s
Mid-turn phase	1,86 s	2,85 s	1,85 s	2,18 s
Return walking phase	3,99 s	3,07 s	2,58 s	4,06 s
Final turn phase	1,63 s	1,45 s	1,27 s	1,63 s
Stand-to-sit phase	0,74 s	1,43 s	0,85 s	0,78 s
10MWT	1,01m/s	1,07 m/s	0,94 m/s	1,11 m/s
Step length R	1,21 m	1, 34 m	1,18 m	1,42 m
Step length L	1,23 m	1,35 m	1,17 m	1,43 m
AP sway EO	18 mm	18 mm	19 mm	22 mm
AP sway EC	24 mm	28 mm	25 mm	32 mm
ML sway EO	4 mm	7 mm	12 mm	9 mm
ML sway EC	6 mm	10 mm	16 mm	14 mm
Sway area EO	68 mm^2	128 mm^2	231 mm^2	199 mm^2
Sway area EC	143 mm^2	267 mm^2	313 mm^2	371 mm^2

performed in supine, sitting, and standing positions, both with and without the patient's back supported against a wall (sensory trick) (Fig. 1). Treatment was performed according to sEMG findings, using OnabotulinumtoxinA (100 U/mL, diluited with sterile 0.9% sodium chloride), and injected using an EMG-guided technique with a 30-gauge, 0.31 × 25 mm needle. A total dose of 160 U was administered, with 30 U injected into each rectus abdominis muscle at two sites per side (15 U per site), and 50 U into each obliquus externus abdominis muscle at three sites per side (15 U at two sites and 20 U at one site). The patient was evaluated one month after treatment, two months following the baseline assessment. BoNT treatment resulted in the greatest improvement in walking velocity and step length among all therapeutic approaches and was the only treatment to improve the posture item of the UMSARS Part II. The injections were well tolerated, with no reported side effects. According to the patient, BoNT treatment was the most effective and preferred intervention.

3. Discussion

Camptocormia is an axial abnormal posture that occurs in both PD and MSA (Sławek et al., 2006; Srivanitchapoom and Hallett, 2016). The standardized Red Flag Checklist (RFCL) developed by the European MSA Study Group (EMSA-SG) identified camptocormia in 32.1% of MSA patients, compared to 5.9% in PD, highlighting its relevance as a clinical feature suggestive of MSA (Köllensperger et al., 2008). When present in PD, camptocormia typically emerges after a prolonged disease course and is often associated with more severe cases and advanced H&Y stages; however, rare instances of camptocormia occurring in the early stages of the disease have also been reported (Lepoutre et al., 2006). In our case, camptocormia appeared early in the disease course, supporting the diagnosis of MSA, together with the other clinical red flags.

The pathogenesis of camptocormia in PD and MSA is not yet fully understood. Both central and peripheral mechanisms have been suggested, and four pathogenetic mechanisms have been described: (1) a manifestation of disease progression; (2) a form of axial dystonia; (3) a consequence of paraspinal myopathy; and (4) an adverse effect of dopaminergic therapy (Srivanitchapoom and Hallett, 2016). It is likely that these mechanisms are differentially involved in camptocormia in PD and MSA, with disease progression representing the predominant mechanism in PD, and dystonia representing the one in MSA. Therefore, the efficacy of various therapeutic interventions for camptocormia in PD and MSA appears to depend on the predominant underlying pathogenetic mechanism.

It is widely recognized that postural deformities can contribute to the development of gait impairments, postural instability, and functional disability. Gait parameters such as step length and stride length have been associated with camptocormia and have been proposed as markers of disease severity, as they correlate with reduced walking velocity, impaired balance, and overall gait deterioration (Geroin et al., 2015; Tramonti et al., 2017). In our study, we evaluated the effects of apomorphine, rTMS, and BoNT on camptocormia, gait dynamics, and postural control using a wearable sensor for quantitative movement analysis.

Apomorphine is a potent and fast-acting dopamine agonist that targets both D1 and D2 receptor subtypes (Mensikova et al., 2015). While its affinity for D2 receptors accounts for the beneficial effects on motor complications in advanced PD, emerging evidence suggests that sustained stimulation of ventrolateral striatal D1 receptors may explain the observed improvements in 10MWT performance and step length in our patient (Mensikova et al., 2015).

Spinal rTMS is a non-invasive neuromodulation technique that has been shown to produce immediate but short-lasting effects on camptocormia in patients with PD (Arii et al., 2014). In accordance with a recent study, we observed an improvement in the TUG test following a single trial of rTMS administered twice a week for four weeks (Mitsui et al., 2022). The underlying mechanism of rTMS-related improvement remains unclear; however, proposed explanations include afferent sensory blockade, descending motor pathway activation, corticostriatal modulation, or efferent stimulation (Arii et al., 2014; Mitsui et al., 2022).

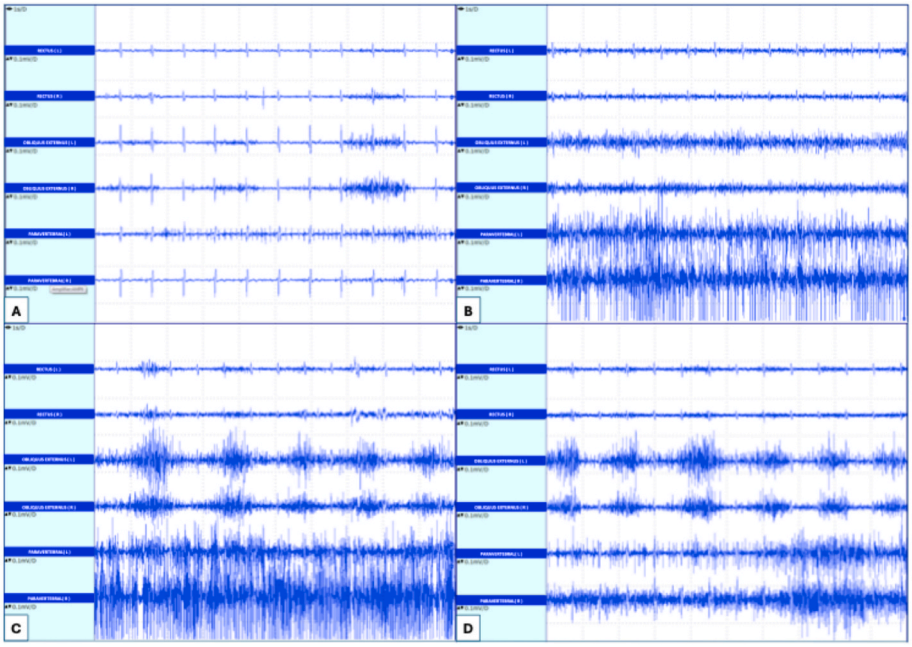

Fig. 1. Surface electromyography of the rectus abdominis, obliquus externus abdominis, and lumbar paravertebral muscles. **(A)** No involuntary muscle activity was observed, aside from mild activity in the left paravertebral muscles, with the patient in the supine position. **(B-C)** High-voltage tonic activity in the paravertebral muscles, along with an oscillatory rhytmic burst activity in the external oblique muscles during the expiratory phase, was observed with the patient in both sitting and standing positions. Minimal activity of the rectus abdominis muscles was noted. **(D)** Tonic activity of lower voltage in the paravertebral muscles, along with an oscillatory rhytmic burst activity in the external oblique muscles during the expiratory phase, was observed with the patient standing and the back supported against the wall (sensory trick).

BoNT is considered the treatment of choice in focal dystonia due to its ability to reduce muscle overactivity (Albanese et al., 2006). Given the role of dystonia in the pathogenesis of camptocormia, BoNT has been evaluated in a limited number of clinical studies, demonstrating limited and variable responses despite the use of different injection targets, dosing regimens, and administration techniques (Bertram et al., 2015). In our patient, sEMG identified the primary muscles involved, and BoNT was administered under EMG guidance, leading to an improvement of the posture item of the UMSARS Part II, as well as in walking velocity and step length, after one month. Nevertheless, despite the patient being evaluated one month after the final rTMS session, a long-term effect of rTMS contributing to the observed clinical improvements cannot be excluded. Lastly, none of the therapeutic approaches led to improvements in sway parameters, underscoring the complex pathophysiology of postural imbalance in MSA and further supporting the use of postural sway as a potential marker of disease progression (Panyakaew et al., 2019).

4. Conclusion

Camptocormia remains a complex and poorly understood condition, with limited evidence supporting effective treatments. Physiotherapy combined with orthoses is considered the first line approach, but provides only modest improvement. Other non-invasive strategies, such as spinal rTMS and BoNT treatment, are promising, particularly in patients with MSA, where postural deformities often arise early and surgical interventions are generally not recommended. Further studies combining rehabilitative strategies with BoNT and rTMS are warranted to investigate their combined effects on camptocormia. In addition, neurophysiological investigations are needed to identify potential responders and to improve our understanding of the underlying pathophysiological mechanisms.

Informed consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Data access, responsibility, and analysis

The Corresponding Author had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Author disclosures

The authors report no disclosures relevant to the study.

Ethical approval

Ethical approval is not required for this study in accordance with local guidelines.

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CRediT authorship contribution statement

D. De Monte: Conceptualization, Data curation, Formal analysis, Investigation, Supervision, Writing – original draft. **G.M. Squintani:** Conceptualization, Data curation, Investigation, Supervision. **G. Ermanis:** Visualization, Writing – original draft. **F. Lavezzi:** Data curation, Investigation. **S. Flumignan:** Data curation, Investigation. **G. Valiante:** Data curation, Investigation. **B.H. Ercole:** Data curation, Investigation. **E. Belgrado:** Supervision. **A. Bernardini:** Supervision. **Y. Tereshko:** Formal analysis, Investigation, Supervision. **M. Valente:** Supervision. **S. Rossi:** Formal analysis, Investigation, Supervision, Validation. **C. Lettieri:** Conceptualization, Data curation, Formal analysis, Supervision, Validation, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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