Camptocormia and Antipsychotic Medications

Rashmi Subhedar, MD; Malathi L. Perugula, MD; Kaushal Shah, MD, MPH; Zeeshan Mansuri, MD, MPH; and Shailesh Jain, MD, MPH

Camptocormia, or "bent spine syndrome" (Greek *Kamptein* meaning "to bend" and *Kormos* meaning "trunk"), refers to an abnormal posture with marked flexion of the thoracolumbar spine that increases during walking and resolves in the supine position.[1] This last feature is a characteristic differential sign.[2] It is an often-overlooked adverse effect of antipsychotics.[2] Camptocormia can be a part of Parkinson disease (prevalence: 3%–18%) or associated with various neuromuscular disorders.[3] Camptocormia can be fatal due to falls, dyspnea, dysphagia, and deep venous thrombosis.[4] Diagnosing and treating this condition early is essential to prevent any severe consequences. Due to the paucity of literature, we present this rare concept with an updated literature review.

Literature Review

Our literature review (Table 1) shows that camptocormia is a neuromuscular condition and a dystonia spectrum of extrapyramidal syndrome (EPS) resulting from antipsychotic medications.[1–7] Dystonic reactions in camptocormia affect the paraspinal and abdominal muscle groups, causing disfigurement. Other complications comprise symptoms worsening on standing and remitting when lying down. Complications include pain, arthritis (if it persists), breathing difficulties, sleep impairment, and difficulty sitting or walking. The condition may arise acutely or chronically. The early signs and symptoms are back pain and tenderness in the paraspinal muscles.[1–7] The most implicated antipsychotics associated with camptocormia are risperidone and olanzapine.[1–7] Removal of olanzapine and adding clozapine or quetiapine in addition to electroconvulsive therapy are the suggested interventions to address camptocormia.[1–7] Evidence points to a disorder in the basal ganglia, with many lines of evidence suggesting the

Table 1.
Literature on Camptocormia and Antipsychotic Medications

Author	Age, y/sex	Causative antipsychotic	Psychiatric/ medical history	Presenting symptom	Duration of development of camptocormia	Diagnostic method	Intervention	Outcome
Robert et al[1]	73/ female	Olanzapine	Chronic delusional disorder	Back pain	Unspecified	Tenderness in paraspinal muscles with necrosis and edema on the MRI	Olanzapine discontinued	Symptoms resolved
González-Pablos et al[2]	34/ male	Olanzapine	Schizophrenia	Trunk stoop up to 45° sometimes up to 90°, Simian-like arm movement, and back pain	18 mo later	None reported	Removal of olanzapine and addition of clozapine, lorazepam, and biperiden	Significant improvement in camptocormia and overall functioning
Mashima et al[4]	30/ female	Risperidone	Schizophrenia	Mild scoliosis in 30s and 40s and camptocormia at the age of 52 y	Chronic	Hypertrophy of the rectus abdominis and lateral abdominal muscles on abdominal CT	Switched to quetiapine	Dystonia, hypertrophy, and creatine kinase levels all resolved
Mehta et al[6]	67/ female	Levosulpiride	Dyspepsia	Altered speech, forward bending of the spine, and bradykinesia	Acute	Clinical examination and imaging	Levosulpiride discontinued and levodopa/carbidopa started	Significant improvement in camptocormia after 2 mo of levodopa
Vela et al[7]	52/ female	Olanzapine	Melancholic depression with psychotic features and agitation	Spine bent to 90°, neck extended, and arms dropped loosely	Over time, unspecified	Physical examination and imaging	Olanzapine withdrawn and ECT given	Abnormalities resolved

Abbreviations: CT = computed tomography, ECT = electroconvulsive therapy, MRI = magnetic resonance imaging.

involvement of the dopaminergic system.[5] All antipsychotics modulate dopamine, and most can affect the acetylcholine systems.[5] Both systems have been implicated in the development of EPS through the nigrostriatal dopaminergic pathway. These drugs block dopamine nigrostriatal tracts along with a relative increase in cholinergic activity.[5]

Conclusion

It is crucial for clinicians to carefully monitor patients who are undergoing antipsychotic treatment and quickly identify and manage this condition to improve treatment outcomes and patients' quality of life. More research is required to fully understand the mechanisms and risk factors of this adverse drug reaction.

References

1. Robert F, Koenig M, Robert A, et al. Acute camptocormia induced by olanzapine: a case report. *J Med Case Reports*. 2010;4:192.
2. González-Pablos E, Valles-de la Calle JM, Iglesias-Santa Polonia F, et al. A case report of camptocormia coinciding with olanzapine use. *J Clin Psychopharmacol*. 2016;36(2):183–184.
3. Srivanitchapoom P, Hallett M. Camptocormia in Parkinson's disease: definition, epidemiology, pathogenesis and treatment modalities. *J Neurol Neurosurg Psychiatry*. 2016;87(1):75–85.
4. Mashima Y, Uchida H, Kinoshita S, et al. Camptocormia secondary to antipsychotic-associated dystonia of the rectus abdominis muscles detected by abdominal computed tomography: a case report. *J Clin Psychopharmacol*. 2021;1(5):608–610.
5. Karimi M, Perlmutter JS. The role of dopamine and dopaminergic pathways in dystonia: insights from neuroimaging. *Tremor Other Hyperkinet Mov (N Y)*. 2015;5:280.
6. Mehta S, Kumar R, Lal V. An unusual cause of camptocormia. *Tremor Other Hyperkinet Mov (N Y)*. 2019;9:608.
7. Vela L, Jiménez Morón D, Sánchez C, et al. Camptocormia induced by atypical antipsychotics and resolved by electroconvulsive therapy. *Movement Disord*. 2006;21(11):1977–1980.

Article Information

Published Online: May 23, 2024. https://doi.org/10.4088/PCC.23br03678

Prim Care Companion CNS Disord 2024;26(3): 23br03678

Submitted: August 23, 2023; accepted October 19, 2023.

To Cite: Subhedar R, Perugula ML, Shah K, et al. Camptocormia and antipsychotic medications. *Prim Care Companion CNS Disord*. 2024;26(3): 23br03678.

Author Affiliations: Department of Psychiatry, Bergen New Bridge Medical Centre, Paramus, New Jersey (Subhedar); Department of Psychiatry, Texas Tech University Health Science Center at Odessa/Permian Basin Odessa, Midland, Texas (Perugula, Jain); Department of Psychiatry, Wake Forest University School of Medicine, Winston-Salem, North Carolina (Shah); Department of Psychiatry, Yale University School of Medicine, New Haven, Connecticut (Mansuri).

Corresponding Author: Rashmi Subhedar, MD, Department of Psychiatry, Bergen New Bridge Medical Centre, 8 Danielle Lane, Monroe Township, Paramus, NJ 08831 (drrashmiphadke@gmail.com).

Author Information: Drs Mansuri and Jain share equal credit for the senior authorship.

Relevant Financial Relationships: None.

Funding/Support: None.