Letter to the Editor/ Editöre Mektup
doi: 10.5606/phhb.dergisi.2024.24

OPEN ACCESS

Etiology of chorea: Stroke, drug-induced, or something more?

Kore etiyolojisi: İnme mi, ilaca bağlı mı, yoksa daha fazlası mı?

Tuğçe Mengi[1], Yeşim Beckmann[2]

[1]*Department of Neurology, Demokrasi University, Buca Seyfi Demirsoy Training and Research Hospital, İzmir, Türkiye*
[2]*Department of Neurology, Katip Çelebi University Atatürk Training and Research Hospital, İzmir, Türkiye*

Hyperkinetic movement disorders are important phenomenon in the intensive care unit and are associated with diverse etiologies and therapeutic implications. Herein, we reported generalized chorea in a critical care patient with ischemic stroke.

An 88-year-old female patient was admitted in the neurological inpatient ward with diagnosis of acute ischemic stroke. The National Institutes of Health Stroke Scale score was 10. The electrocardiogram showed atrial fibrillation. Cranial magnetic resonance imaging revealed acute ischemic stroke involving the right mesial temporal lobe and thalamus. The patient's level of consciousness decreased. Therefore, the patient was intubated with midazolam and rocuronium. The treatment that included enoxaparin, amlodipine, pantoprazole, and continuous infusion of midazolam were administered. During follow-up, intravenous flumazenil was used in increments of 0.25 mg/min (a total dose of 1 mg) for extubation. After the administration of flumazenil, the patient partially regained consciousness. The neurointensivist who examined the patient observed abnormal movements in all extremities after administration of flumazenil. Those involuntary, nearly continuous, abrupt, rapid, jerky, nonrhythmic, rotatory, and generalized movements involved distal parts of limbs more than the proximal ones. The movements were considered to be chorea and had a less prominent ballistic component as well.

The causes of chorea are many and heterogeneous. They include infectious, immunologic, genetic, metabolic, nutritional, hypoxic-ischemic, and cerebrovascular reasons.[1] Laboratory workup were normal except for mild, stable anemia. Control cranial magnetic resonance imaging revealed restricted diffusion in the right posterior cerebral artery territory compatible with acute infarction. Generalized chorea caused by unilateral lesion has been rarely reported.[2] Drug-induced

Correspondence: / İletişim adresi: Tuğçe Mengi, MD. Demokrasi Üniversitesi, Buca Seyfi Demirsoy Eğitim ve Araştırma Hastanesi, Nöroloji Kliniği, 35390 Buca, İzmir, Türkiye.
E-mail *(e-posta):* tugceangin@gmail.com

Received / *Geliş tarihi:* August 04, 2024 *Accepted* / Kabul tarihi: July 02, 2025 Published online / *Online yayın tarihi:* 13 Ağustos 2025

This study was presented as an oral presentation at the 11th International Medicine and Health Sciences Researches Congress, December 24-25, 2022, Ankara, Türkiye.

Citation:
Mengi T, Beckmann Y. Etiology of chorea: Stroke, drug-induced, or something more? Parkinson Hast Harek Boz Derg 2024;27(1):17-18. doi: 10.5606/phhb.dergisi.2024.24.

chorea is the most common form of acute-onset chorea in adults.[1] It has been reported with the use of levodopa, apomorphine, antipsychotics, antiepileptics, oral contraceptives, antihistamines, tricyclic antidepressants, and selective serotonin reuptake inhibitors. In addition, drug withdrawal may rarely cause chorea.[3] There are only a few case reports of chorea secondary to cessation of benzodiazepines or administration of flumazenil.[3,4] Benzodiazepines facilitate the inhibitory effect of gamma-aminobutyric acid (GABA) at the presynaptic junction. With prolonged facilitation of GABA inhibition, compensatory increases in the activity of excitatory dopaminergic pathways in the striatum may occur. As long as GABA neuroinhibition is increased, the GABA and dopaminergic activities are balanced. Withdrawing benzodiazepines leads to an acute decrease in GABA-mediated inhibition and hence a functional excess of dopaminergic activity, which has been implicated as a cause of chorea.[3] Flumazenil is being used to reverse the adverse pharmacological effects of benzodiazepine and may antagonize the GABA-benzodiazepine receptor complex or may induce dopamine hypersensitivity.[4] Therefore, we speculate that the mechanism by which chorea occurs after discontinuation of the benzodiazepine and administration of flumazenil is due to an imbalance between the GABAergic and dopaminergic pathways.

Multiple factors may lower the threshold for the induction of movement disorders such as chorea and ballism.[1] We hypothesize that the presence of stroke in this case has lowered the threshold for the induction of chorea by flumazenil. Given the temporal association between chorea and flumazenil, flumazenil may be considered one of the causal agents.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Has contributed to and conceptualized the clinical management of the patient, she interpreted the data regarding movement disorders, stroke and drugs, wrote the manuscript: T.M.; Revised and edited the manuscript: Y.B. All authors read and approved the final manuscript.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article

REFERENCES

1. Delaruelle Z, Honoré PJ, Santens P. Adult-onset Sydenham's chorea or drug-induced movement disorder? A case report. Acta Neurol Belg 2016;116:399-400. doi: 10.1007/s13760-015-0537-6.
2. Chang JH, Seo WK, Park MH, Lee JM, Kwon DY, Koh SB. Generalized chorea induced by an unilateral anterior cerebral artery territorial infarction. J Mov Disord 2009;2:37-9. doi: 10.14802/jmd.09009.
3. Patrick SJ, Snelling LK, Ment LR. Infantile chorea following abrupt withdrawal of diazepam and pentobarbital therapy. J Toxicol Clin Toxicol 1993;31:127-32. doi: 10.3109/15563659309000381.
4. Kim JS, Ko SB, Choi YB, Lee KS. Flumazenil-induced ballism. J Korean Med Sci 2003;18:299-300. doi: 10.3346/jkms.2003.18.2.299.