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Reversible Bilateral Basal Ganglia Lesions Due to Multifactorial Toxic-Metabolic Disorders

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Abstract **Case Report**

Bilateral basal ganglia lesions can arise from a diverse range of causes, such as metabolic imbalances, toxic exposure, degenerative conditions, vascular events, inflammatory processes, infections, and tumors. We present a case involving a 66-year-old male patient who was admitted to the hospital following acute changes in behavior and psychomotor slowing. His medical history was notable for Parkinson's disease, with a recent adjustment in L-Dopa dosage, as well as diabetes mellitus and arterial hypertension. Upon initial evaluation, the patient was hypertensive, drowsy, disoriented in both time and space, exhibited dysarthria, and displayed global bradykinesia. Diagnostic investigations revealed a brain CT scan that showed bilateral, symmetrical hypodensities in the lenticulo-caudate region without midline shift or evidence of cerebral herniation. The cerebrospinal fluid (CSF) analysis found 15 cells/µL with no other abnormalities, while laboratory tests highlighted hypernatremia (175 mEq/L), elevated creatinine (3.5 mg/dL), hyperglycemia (<300 mg/dL), along with slightly increased C-reactive protein and anticardiolipin antibodies, and thrombocytopenia (107,000/uL). Following treatment adjustments—including the correction of metabolic imbalances and discontinuation of L-Dopa—subsequent brain imaging showed improvement in the lesions, and the patient's condition returned to baseline. The basal ganglia play a complex role in brain function, relying heavily on glucose and oxygen, which makes them particularly sensitive to metabolic disturbances. In this case, symmetrical lesions in the basal ganglia resulted in acute mental status changes, likely linked to hyperglycemia, acute kidney injury, hypertension, and potential exposure to toxic substances, including medication and environmental toxins. Full clinical recovery, alongside negative follow-up investigations and regression of the lesions, supports the proposed diagnosis.

Keywords: Acute Confusional State, Toxic Exposures (L-Dopa), type 2 Diabetes Mellitus, Acute Kidney Injury, Bilateral and Symmetrical Lenticulo-Caudate Hypodensities.

INTRODUCTION

Bilateral basal ganglia lesions are a rare syndrome that can emerge in various disorders, including systemic metabolic derangements, toxic exposures, neoplasms, as well as degenerative, vascular, inflammatory, and infectious conditions. This case report discusses the relationship between the toxic-metabolic disturbances present in our patient, their symptoms, and the changes observed in imaging studies. Despite the known importance of metabolic and vascular factors in pathophysiology, their precise roles remain unclear.

OBSERVATION

A 66-year-old man, with a previously independent lifestyle, was admitted to the neurology department with an acute confusional state and psychomotor slowing, both of which had appeared on the day of admission. His medical history included Parkinson's disease (with a recent increase in L-Dopa dosage), diabetes mellitus, and arterial hypertension. There was no history of alcohol use, recent travel, or animal bites. Furthermore, no prior episodes of similar symptoms were recorded. Upon admission, the patient was found to be hypertensive (systolic blood pressure = 180 mmHg), drowsy, and had difficulty maintaining attention, along with disorientation to time and place. He exhibited moderate dysarthria, global bradykinesia, and increased limb muscle tone. There were no signs of involuntary movements, focal neurological deficits, or fever. Blood work revealed significant hypernatremia (175 mEq/L), acute kidney injury (creatinine up to 3.5 mg/dL), and elevated blood glucose (consistently <300 mg/dL), all of which were subsequently managed.

Additionally, the patient had an elevated C-reactive protein (20.9 mg/L) and mild thrombocytopenia (107,000/uL). A brain CT scan showed bilateral, symmetric hypodensities in the lenticulo-caudate region without mass effect or midline shift. Additionally, there was evidence of diffuse corticosubcortical atrophy, with cortical sulci widening and mild ventricular enlargement.

After addressing the patient's metabolic imbalances and eliminating the suspected toxic influences, follow-up brain CT imaging showed the same lesions but with more heterogeneous characteristics, suggesting resolution. The patient returned to his previous neurological baseline.

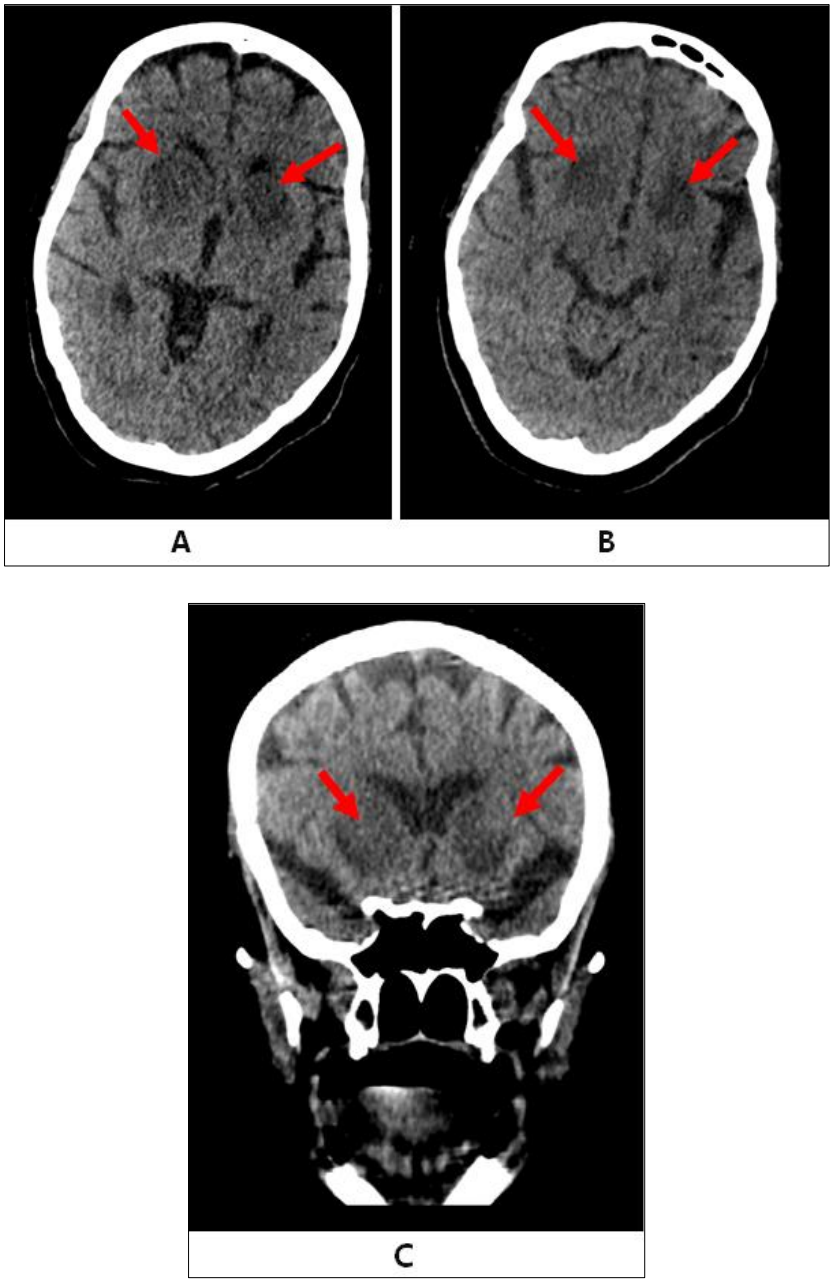

Fig. 1: Cerebral CT scan without contrast injection in axial (A and B) and coronal (C) sections, showing bilateral, symmetrical lenticulo-caudate hypodensities with no mass effect on medial structures and no sign of cerebral involvement.

DISCUSSION

We present a rare case involving symmetrical lesions in the basal ganglia, where the patient exhibited an acute onset of altered mental state and behavioral disturbances. These symptoms were linked to hyperglycemia, acute kidney injury, hypertension, and high-dose treatments (L-Dopa) alongside possible exposure to other chemical substances. Both the clinical symptoms and the lesions resolved after correcting the metabolic abnormalities and eliminating the toxic exposure. This mirrors outcomes seen in other cases of acute bilateral basal ganglia lesions. The complete

recovery of the patient, with a negative work-up and regression of the lesions, further supports the diagnosis. Certain toxic and metabolic conditions affecting the central nervous system display specific patterns that help suggest the underlying cause. These patterns often involve symmetrical areas such as the basal ganglia, thalami, cortical gray matter, periventricular white matter, corticospinal tracts, corpus callosum, parieto-occipital regions with vasogenic edema, or the central pons. In contrast, asymmetrical or focal lesions in the basal ganglia are more frequently associated with infections or neoplastic processes. Common causes of bilateral basal ganglia lesions include carbon monoxide poisoning, hypoxia, exposure to toxins, metabolic imbalances, vasculitis, and encephalitis. However, the diagnosis can be challenging, often requiring careful correlation of clinical signs, imaging findings, and laboratory results. In this case, partial reversibility of the lesions was observed. As previously reported, the extent of reversibility depends on the duration and severity of the exposure. Though this syndrome is uncommon, it is important to emphasize that metabolic disorders and exposure to certain toxic agents can lead to significant damage to the central nervous system, particularly the basal ganglia. Due to their high metabolic demands and reliance on glucose and oxygen, these structures are especially vulnerable to systemic disruptions. Early recognition and treatment of these conditions are critical, as proper management can lead to the reversal of damage.

CONCLUSION

Metabolic disturbances and exposure to specific toxins can significantly impact the central nervous system, particularly the basal ganglia. Due to their high metabolic demands and dependence on glucose and oxygen, the basal ganglia are especially susceptible to metabolic imbalances and various systemic or generalized disease conditions. Recognizing these vulnerabilities is crucial, as prompt treatment and management of these disorders can lead to the reversal of the resulting damage.

BIBLIOGRAPHY

- Ari, Ş., Çaça, İ., & Kayabaşi, H. (2007). Bilateral complete optic atrophy and hemorrhagic infarction of the putamen caused by methanol intoxication. *Annals of Ophthalmology*, *39*, 249-252.
- Bekiesinska-Figatowska, M., Mierzewska, H., & Jurkiewicz, E. (2013). Basal ganglia lesions in children and adults. *European journal of radiology*, *82*(5), 837-849.
- Bhatia, R., Kumar, M., Garg, A., & Nanda, A. (2008). Putaminal necrosis due to methanol toxicity. *Practical neurology*, *8*(6), 386-387.
- Chalela, J. A., Wolf, R. L., Maldjian, J. A., & Kasner, S. E. (2001). MRI identification of early white matter injury in anoxic–ischemic encephalopathy. *Neurology*, *56*(4), 481-485.
- Chattopadhyay, S., & Chandra, P. (2007). Putaminal necrosis. *New England Journal of Medicine*, *356*(22), e23.
- de Oliveira, A. M., Paulino, M. V., Vieira, A. P., McKinney, A. M., da Rocha, A. J., Dos Santos, G. T., ... & Lucato, L. T. (2019). Imaging patterns of toxic and metabolic brain disorders. *Radiographics*, *39*(6), 1672-1695.
- Dujardin, M., Peeters, E., Ernst, C., & Stadnik, T. (2006). Bilateral putaminal necrosis due to methanol abuse. *JBR BTR*, *89*(6), 316.
- Glazer, M., & Dross, P. (1993). Necrosis of the putamen caused by methanol intoxication: MR findings. *AJR. American journal of roentgenology*, *160*(5), 1105-1106.
- Kamei, S., Takasu, T., Mori, N., Yoshihashi, K., & Shikata, E. (1996). Serial imaging of bilateral striatal necrosis associated with acidaemia in adults. *Neuroradiology*, *38*, 437-440.
- Lang, C. J. (2000). The use of neuroimaging techniques for clinical detection of neurotoxicity: a review. *Neurotoxicology*, *21*(5), 847-855.
- McKinney, A. M., Kieffer, S. A., Paylor, R. T., SantaCruz, K. S., Kendi, A., & Lucato, L. (2009). Acute toxic leukoencephalopathy: potential for reversibility clinically and on MRI with diffusion-weighted and FLAIR imaging. *American Journal of Roentgenology*, *193*(1), 192-206.
- McLean, D. R., Jacobs, H., & Mielke, B. W. (1980). Methanol poisoning: a clinical and pathological study. *Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society*, *8*(2), 161-167.
- Sefidbakht, S., Rasekhi, A. R., Kamali, K., Borhani Haghighi, A., Salooti, A., Meshksar, A., ... & Nabavizadeh, S. A. (2007). Methanol poisoning: acute MR and CT findings in nine patients. *Neuroradiology*, *49*, 427-435.
- Wang, H. C., & Cheng, S. J. (2003). The syndrome of acute bilateral basal ganglia lesions in diabetic uremic patients. *Journal of neurology*, *250*, 948-955.