Open Journal of Medical Images and Case Reports
ISSN: 3067-1078 | Volume 3, Issue 2, 2026
www.aytinpublications.com

Case Report

Neuroimaging Findings of Basal Ganglia Hyperintensities in the Setting of Polysubstance Abuse

Hajdi Gorica[1], Pavllo Djamandi[2], Emili Mara[2], Gentian Vyshka[3]*

Service of Neurology, Trauma University Hospital Center of Tirana, Albania.
Division of Neurosciences, University Hospital Center 'Mother Teresa', Tirana, Albania.
Biomedical and Experimental Department, Faculty of Medicine, University of Medicine in Tirana, Albania.

*Corresponding Author: Gentian Vyshka, Biomedical and Experimental Department, Faculty of Medicine, University of Medicine in Tirana, Albania.

Abstract

Cocaine and heroin-related basal ganglia lesions are a rare reported complication of isolated cocaine or heroin abuse. We report the case of a 40-year old man with a history of chronic cocaine and heroin abuse who presented with dysarthria and right-sided weakness. Neuroimaging demonstrated bilateral symmetrical basal ganglia hyperintensities, which showed diffusion restriction, an initial diagnosis of subacute ischemia or toxic-hypoxic lesions of the basal ganglia were suspected. During the course of several days, the patient's neurological deficits improved significantly.

Keywords: Substance Abuse, Neuroimaging, Cocaine, Heroin, Basal Ganglia Lesions.

Article Information

Received: 03 August 2026

Accepted: 14 August 2026

Published: 16 August 2026

Cite this article as:

Hajdi Gorica, Pavllo Djamandi, Emili Mara *et al.* Neuroimaging Findings of Basal Ganglia Hyperintensities in the Setting of Polysubstance Abuse. Open Journal of Medical Images and Case Reports. 2026; 3(2): 01-03

https://doi.org/10.71123/3067-1078.030201

Introduction

The basal ganglia are highly metabolically active structures that are particularly susceptible to hypoxic, metabolic, toxic and vascular insults. Cocaine has been associated with both ischemic and hemorrhagic stroke through many mechanisms such as vasospasm, endothelial dysfunction, inducing platelet aggregation and thrombus formation (2). It is particularly challenging to distinguish between vascular and metabolic injury of basal ganglia in the setting of polysubstance use.

Case Description

A 40-year-old man with a history of cocaine and heroin abuse and hepatitis B carrier presented to the emergency department with a 12-hour history of slurred speech and right-handed weakness. On first evaluation, the patient was alert, oriented to person, time, place, dysarthric, pupils were symmetrical and reactive to light, no CN palsies were noted, right hemiparesis with an evaluated motor strength of 2/5 MRC right arm and 1-2/5 right leg, asymmetry of the DRT more pronounced on the right, extensor plantar response on the right. The lab work revealed a very high serum creatine kinase level of 5951U/l, with normal level of CK-MB, Troponin I, urea, creatinine; serum potassium, sodium and calcium level. The urine toxicology screening was positive for cocaine and heroin. CK levels decreased to 2123 U/L after 24 hours and 592 U/l after 48 hours. Non-contrast CT of the head showed bilateral symmetrical basal ganglia hypodensities; no abnormalities were noted on CTA of the supraaortic vessels. (**Figure 1**)

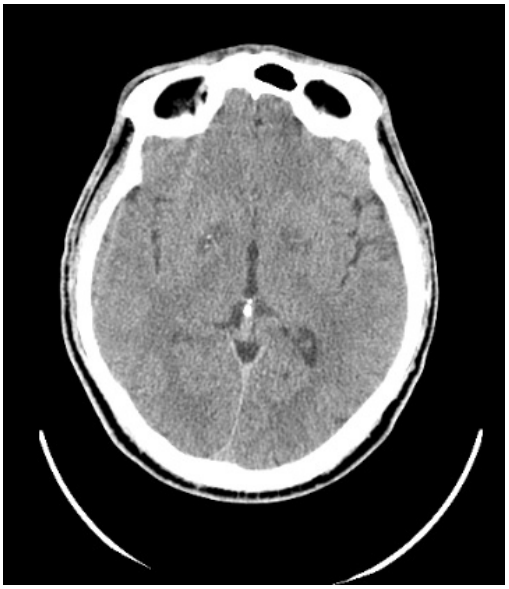

Figure 1. *Brain computerized tomography demonstrating bilateral symmetrical basal ganglia hypodensities.*

Magnetic Resonance Imaging of the head revealed bilateral symmetrical basal ganglia lesions, which appeared hyperintense on T2 and Fluid Attenuated Inversion Recovery Imaging with diffusion restriction of Diffusion Weighted Imaging. (**Figure 2**)

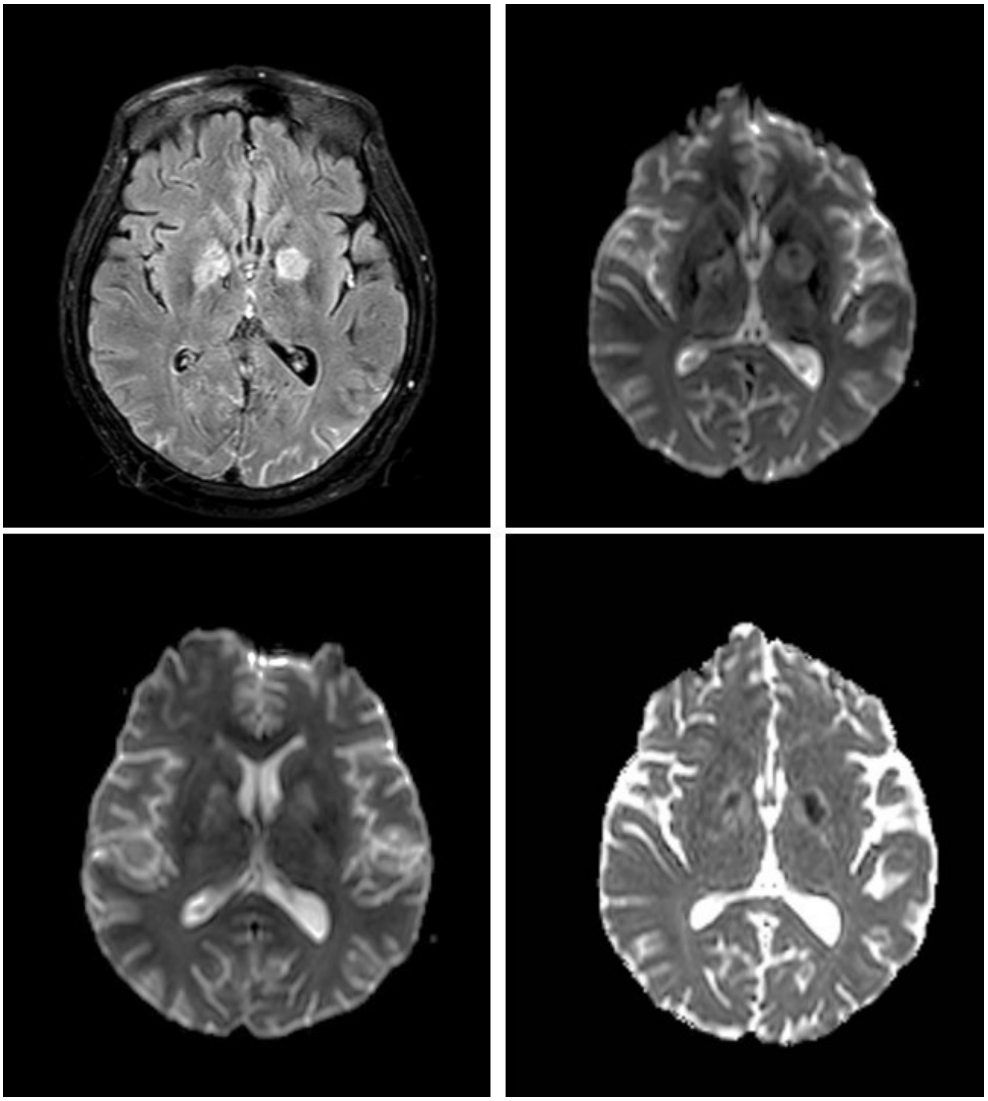

Figure 2. *Magnetic Resonance Imaging of the head revealed bilateral hyperintense symmetrical basal ganglia lesion on Fluid Attenuated Inversion Recovery Imaging (upper left inset); with diffusion restriction of Diffusion Weighted Imaging (upper right inset; lower insets).*

Based on these findings, a toxic-metabolic or subacute ischemic basal ganglia injury was considered. The patient was treated with IV fluids and electrolytes as needed, and during the first days of hospital stay, his neurological status improved significantly; the weakness of the right extremities improved to a slight drift in Mingazzini I and II.

Discussion

Symmetrical basal ganglia lesions have a broad differential including hypoxic-ischemic, toxic injury, metabolic disorder, infectious causes or mitochondrial disease (1, 3). Cocaine is known to cause stroke via vasospasm, endothelial dysfunction, and thrombosis, whereas heroin may cause hypoxic or toxic injury (2). In our case, the severe CK elevation indicated acute rhabdomyolysis, likely related to substance use and less likely to prolonged immobilization. Normal renal function and rapid CK decline supported a transient toxic muscle injury rather than systemic metabolic failure. The coexistence of rhabdomyolysis, bilateral basal ganglia diffusion restriction, the improvement of the neurological deficits, and rapid decline of serum CK level favored a diagnosis of toxic-hypoxic mechanism. This case underscores the importance of an integrated approach to decision making, integrating clinical, biochemical and neuroimaging findings when evaluating basal ganglia lesions in substance users.

References

1. Mätzler W, Nägele T, Gasser T, Krüger R. Acute parkinsonism with corresponding lesions in the basal ganglia after heroin abuse. Neurology. 2007 Feb 6; 68(6):414. Doi: 10.1212/01.wnl.0000250230.86581.ea. PMID: 17283314.

2. Rendon LF, Malta S, Leung J, Badenes R, Nozari A, Bilotta F. Cocaine and Ischemic or Hemorrhagic Stroke: A Systematic Review and Meta-Analysis of Clinical Evidence. J Clin Med. 2023 Aug 10; 12(16):5207. Doi: 10.3390/jcm12165207. PMID: 37629248; PMCID: PMC10455873.

3. Trejos M, Nittala S, Nedd K. Cocaine-Induced Bilateral Basal Ganglia Infarction in a Patient With ST-Elevation Myocardial Infarction: A Case Report. Cureus. 2025 Mar 24; 17(3):e81099. Doi: 10.7759/cureus.81099. PMID: 40271306; PMCID: PMC12017778.