

INTRODUCTION

Methamphetamine, a potent and highly addictive central nervous system stimulant, increases the release and blocks the reuptake of dopamine, norepinephrine, and serotonin. This dysregulation of neurotransmitters results in intense euphoria and sympathetic overstimulation, reinforcing its addictive nature. Chronic use of methamphetamine is closely linked to catecholamine exhaustion and vascular issues, such as hypertensive strokes, myocardial infarctions, and dissections. We present a unique case of a 57-year-old female with decades of chronic methamphetamine abuse experiencing global cerebral calcification and catecholamine depletion sequelae.

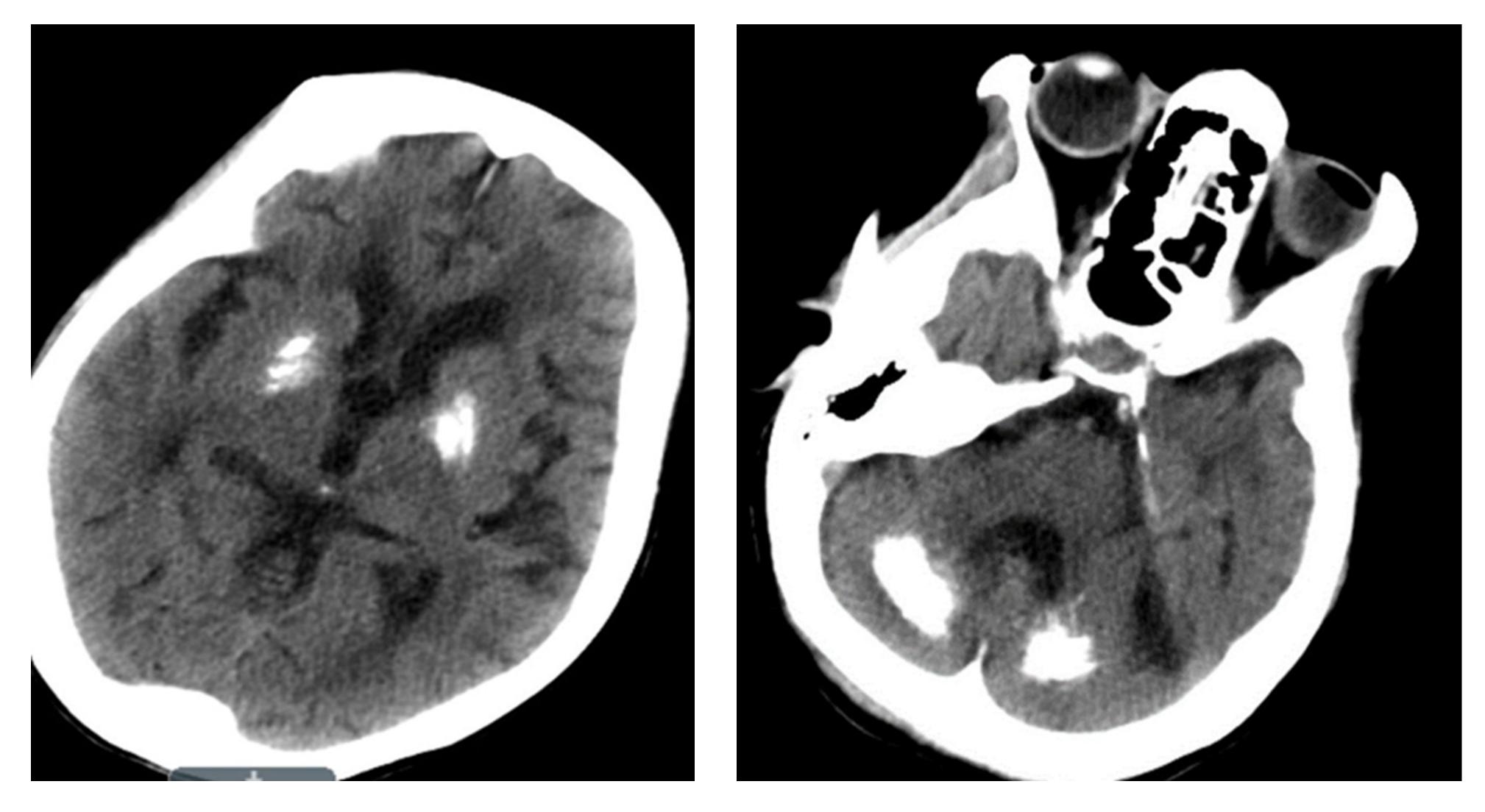
CASE PRESENTATION

- A 57-year-old woman presented obtunded after experiencing similar episodes in the past related to dehydration, stroke, or infection. Before this incident, the husband denied any seizure-like activity, fever, shortness of breath, or chest pain.
- Her initial vitals revealed a hypertensive crisis and an irregular heart rate.
- Physical examination findings were unremarkable as well as neurological exam.
- EKG, chest x-ray, and urinalysis revealed no abnormalities. Computed Tomography (CT) scan of her head showed generalized calcification of the cerebellar and basal ganglia with marked chronic periventricular white matter disease. Magnetic Resonance Imaging was completed with no acute abnormalities noted.
- After positive urine drug screen, patient disclosed a 40-year history of near daily methamphetamine abuse. Despite blood pressure control, her mental status did not improve until methylprednisolone was initiated to address catecholamine deficiency. Following steroid replacement, she showed responsiveness and improved eye-opening. She continued to improve on steroids and was discharged with a steroid taper and short-term follow-up.

"From Catecholamine Exhaustion to Cerebral Calcification: Unraveling a Decades-Long Odyssey of Methamphetamine Use" Tracy-Ann Poyser, MD¹, Collie Shaw, MD, Andrew Phillips, MD PhD, Ziad Ghneim, DO, **Courtney Hicks, MD, David Martin, PhD**

IMAGES

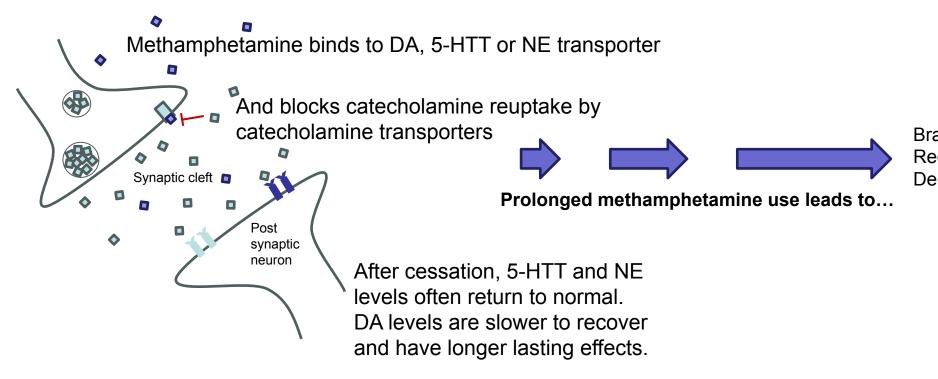




CT of the head without contrast showing cerebellar and basal ganglia calcifications

DISCUSSION

Recent studies link chronic methamphetamine use to brain calcification, stemming from oxidative stress, inflammation, and catecholamine depletion. This compromises brain function and raises the risk of cerebrovascular events, posing challenges for healthcare providers. A multidisciplinary approach, early intervention, and targeted therapies for catecholamine restoration and neuroprotection are essential. Continued research aims to alleviate the burden of methamphetamine abuse on individuals and society.



REFERENCES

Chiu VM, Schenk JO. Mechanism of action of methamphetamine within the catecholamine and serotonin areas of the central nervous system. Curr Drug Abuse Rev. 2012 Sep;5(3):227-42. doi: 10.2174/1874473711205030227. PMID: 22998621.

Rodríguez MJ, Pugliese M, Mahy N. Drug abuse, brain 2) calcification and glutamate-induced neurodegeneration. Curr Drug Abuse Rev. 2009 Jan;2(1):99-112. doi: 10.2174/1874473710902010099. PMID: 19630740.

Naidoo S, Smit D. Methamphetamine abuse: A review of 3) literature and case report in a young male. J African. 2011 Apr:66(3):124-127. doi:10.10520/EJC147292. https://hdl.handle.net/10520/EJC147292

Brain inflammation Reduced grey matte epletion of neurotransmitter

