Evolution of Hypoxic–Ischemic Injury

Ruchira M. Jha, MD¹, Aaron L. Berkowitz, MD, PhD¹, and Joshua P. Klein, MD, PhD¹

The Neurohospitalist
3(1) 46
© The Author(s) 2013
Reprints and permission:
sagepub.com/journalsPermissions.nav
DOI: 10.1177/1941874412467807
http://nhos.sagepub.com

Figure 1. Axial diffusion weighted (A), apparent diffusion coefficient (B), and fluid-attenuated inversion recovery (C) magnetic resonance images demonstrate diffuse cytotoxic edema. Axial (D, E) and coronal (F) computed tomography reveal widespread parenchymal encephalomalacic atrophy.

A 21-year-old man had pulseless electrical activity (PEA) arrest after cocaine overdose. Resuscitation initiated 40 minutes after the arrest led to a perfusing rhythm after 20 minutes. He remained comatose. Hypothermia was not instituted. Magnetic resonance imaging of the brain revealed symmetric reduced diffusivity and abnormal T2 hyperintensity in the cortex and deep gray matter structures, stigmata of hypoxic–ischemic injury portending poor prognosis (Figure 1A–C).¹ After 28 months in a persistent vegetative state, he was reevaluated for tremulous activity, ultimately found to be rigors caused by urosepsis. Computed tomography of the head at that time demonstrated the dramatic evolution of his severe hypoxic–ischemic injury, a rarely seen phenomenon (Figure 1D–F).

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Reference

1. Wijman CA, Mlynash M, Caulfield AF, et al. Prognostic value of brain diffusion weighted imaging after cardiac arrest. *Ann Neurology*. 2009;65(4):394–402.

¹ Department of Neurology, Brigham and Women's Hospital, Boston, MA, USA

Corresponding Author:

Ruchira M. Jha, Brigham and Women's Hospital, 75 Francis Street, Boston, MA, USA
Email: rjha@partners.org