Teaching NeuroImages: Spindle coma following cerebral herniation and pontine infarction

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A 67-year-old woman presented with bilateral subdural hematomas. After hematoma evacuation, she was unresponsive with extensor posturing and preserved brainstem reflexes. MRI (figure 1) revealed extensive posterior circulation infarction including the mid-pons, thought secondary to herniation and vascular compression. EEG (figure 2) revealed an unreactive background resembling normal N2 sleep (including spindles, K-complexes, and vertex waves) for >24 hours, diagnostic of spindle coma.¹ Conditions implicated in spindle coma tend to disrupt the ascending arousal system at the brainstem and hypothalamic level, but spare the thalamus.^{1,2} Underlying etiology, extent of injury, and EEG reactivity provide more prognostic insight than spindle coma alone.^{1,2}

Figure 1 Brain MRI, mid pontine region, axial view

Brain MRI demonstrates extensive acute infarction of bilateral occipital lobes, bilateral posterior temporal lobes, and mid pons (arrows) on diffusion-weighted imaging (A) and adjusted diffuse coefficient imaging (B).

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Figure 2 EEG and quantitative EEG spectrogram

EEG (A) in longitudinal bipolar montage is without change after sternal rub (arrow), but normal N2 sleep graphoelements (B) are demonstrated: sleep spindles (red arrows), K-complex (blue arrow), and vertex wave (green arrow). Two hours of quantitative EEG rhythmicity and power spectrogram (C) do not show the frequency variability of normal sleep cycling as seen in a typical non-comatose patient (D).

Appendix Authors

Name	Location	Contribution
Stefanie P. Cappucci, MD	Beth Israel Deaconess Medical Center, Boston, MA	Designed and conceptualized case report, drafted and revised manuscript
Kyle C. Rossi, MD	Beth Israel Deaconess Medical Center, Boston, MA	Designed and conceptualized case report, revised manuscript
Daniel M. Goldenholz, MD, PhD	Beth Israel Deaconess Medical Center, Boston, MA	Revised and edited the manuscript

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