Hypoxic brain injury sparing the posterior circulation

A 37-year-old patient was admitted with anaphylactic shock, tachycardia, and hypotension. She remained comatose and developed clinical features of hypoxic brain injury (HBI). While repetitive CT scans appeared normal, MRI on day 4 showed typical signs of HBI except sparing of all hypoxia-sensitive structures of the posterior circulation, e.g., visual and cerebellar cortex, hippocampus, and thalamus (figure). This might be explicable by reflexory redistribution of blood to the posterior circulation in shock or by differential vaso-regulation of anterior and posterior circulation, which also causes posterior reversible encephalopathy syndrome to preferentially manifest posteriorly. This case emphasizes the importance of HBI lesion distribution in the deduction of underlying pathophysiologic mechanisms.

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Diffusion-weighted MRI on day 4 shows cytotoxic edema of the basal ganglia and nearly the entire cortex supplied by the anterior circulation. Parenchyma supplied by the vertebrobasilar circulation does not show any signal abnormalities.

Figure MRI showing hypoxic brain injury sparing the posterior circulation
Hypoxic brain injury sparing the posterior circulation
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