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Teaching NeuroImage: Unilateral Temporal Lobe Hypoperfusion: A Pathogenic Mechanism in Transient Global Amnesia?

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Contributions:

Eva Tallon: Drafting/revision of the manuscript for content, including medical writing for content

Shane Hanratty: Major role in the acquisition of data

Karl Boyle: Major role in the acquisition of data; Study concept or design

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A 51-year-old man developed sudden onset anterograde amnesia several hours following a typical migraine attack. He had no medical history or vascular risk factors other than a migraine disorder since early adulthood. There were no deficits in other cognitive domains and no loss of personal identity. Symptoms resolved within 24 hours. CT brain and angiogram during the episode were normal, however CT perfusion imaging (Figure, A) performed three hours after symptom onset revealed an area of focal left temporal hypoperfusion. MRI-brain (Figure, B and C) performed 48 hours later did not show any corresponding areas of ischaemic change or punctate DWI lesions as previously described in 69% of cases with highest sensitivity at 12-24 hours (1). Transient global amnesia is associated with migraine and migraine in turn has an association with vascular pathology (2). This case underlines that transient temporal hypoperfusion may play an important role in the pathogenesis of transient global amnesia.

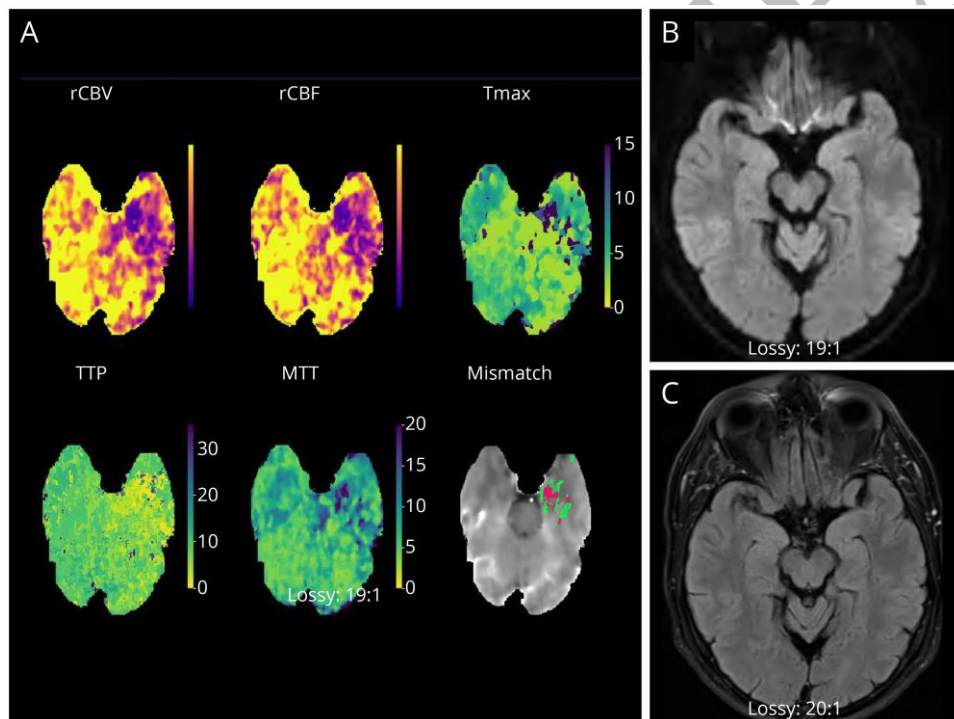
WNL-2023-000339_slides --- <http://links.lww.com/WNL/C954>

Reference:

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Figure CT perfusion and MRI brain:

CT-perfusion 3-hours post symptom onset (A; rCBV/rCBF-relative cerebral blood volume/flow; Tmax-time to maximum; TTP-time to peak; MTT-mean transit time) demonstrating left medial temporal lobe hypoperfusion. Diffusion weighted (B) and T2-FLAIR (C) MRI-brain sequence images at 48-hours post symptom onset demonstrating no evidence of infarct in the medial temporal lobe.



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