

Abnormal brain CT perfusion in a patient with a migraine with aura episode: Case report.



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Clinical case

A 30 year old male patient, without cardiovascular risk factors or migraine history, presented to the emergency department with a 90 minutes negative visual defect compatible with right homonymous hemianopia with progressive improvement and bifrontal headache. At admission, no other neurological deficits were found. He was immediately studied with a non-enhanced computed tomography (NECT) and CT angiography (CTA) with no pathological findings. CT perfusion (CTP) showed a prolonged Time-to-Maximum (Tmax) in both posterior cerebral arteries territory as a sign of hypoperfusion. As symptoms were potentially disabling and ischaemic stroke was suspected, treatment with alteplase was administered. Patient persisted with residual visual deficit for at least 24 hours. A magnetic resonance imaging scan was performed at that point, with no evidence of acute ischemic images. Diagnosis of transient ischemic attack versus debut of probable migraine with prolonged aura (MA) was initially made. At 10 month follow-up, he presented a new episode of visual aura with positive symptoms, confirming the diagnosis of MA.

Images

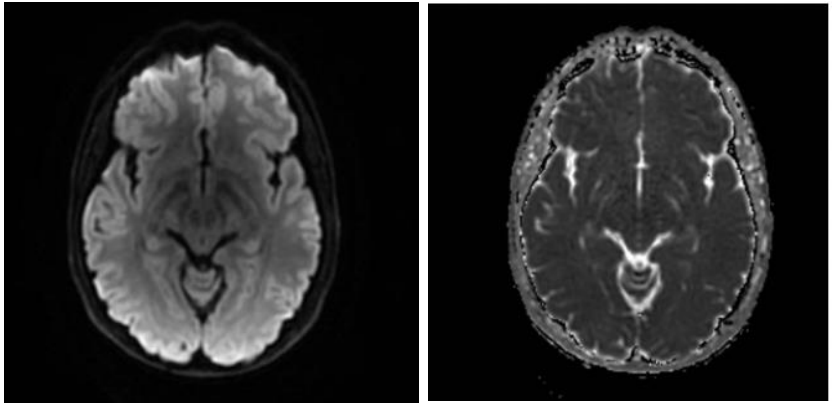
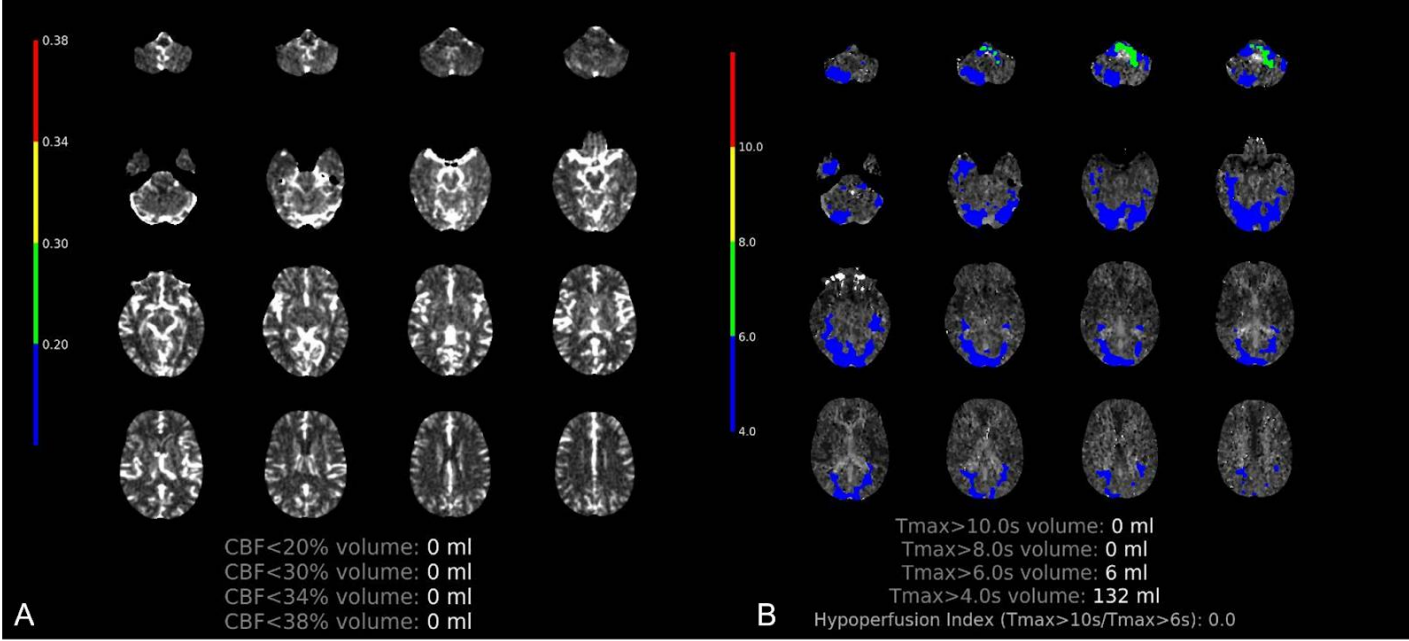


Image 2: Brain MRI performed after 24 hs of symptoms showed no restrictive images in diffusion sequences

Conclusion

Reduced cerebral blood flow (CBF) and cerebral blood volume are the most commonly reported findings on perfusion studies in the aura phase of migraine. These perfusion alterations are followed in frequency by prolonged mean transit time (MTT), time to peak (TTP) or Tmax; encompassing more than one vascular territory with an occipital predominance as seen in our patient, even with normal CBF and CBV. Perfusion imaging studies in stroke patients show a more severe increase of MTT, TTP or Tmax, reaching values that are atypical in MA; these findings should raise a suspicion of critical hypoperfusion, as seen in acute ischaemic stroke.

Image 1: Brain CTP showed no alterations on CBF (A) and prolonged max (B) in both occipital lobes, not reaching the critical hypoperfusion threshold (Tmax > 6 seconds).