VISUAL HALLUCINATIONS: A NON-UNIQUE SYMPTOM

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INTRODUCTION Different mechanisms involved in migraine pathophysiology, especially in migraine with aura (MA), might contribute to the development of ischemic brain injury, such as impaired cerebral autoregulation, cortical spreading depolarization (CSD), embolism and endothelial dysfunction.

CASE REPORT A 75-year-old man was affected by MA since the age of 30, experiencing monthly episodes of scintillating scotomas, rarely followed by headache. In March 2025, due to an acute myocardial infarction, he underwent coronary angioplasty. During hospitalization, he reported the sudden onset of a peculiar visual disturbance, described critically by the patient, characterized by palinopsia, scintillating scotomas, and visual hallucinations consisting of moving figures and grotesque black masks on a red background, accompanied by typical migraine. Because of the persistence and unusual nature of the disturbance, the patient underwent a baseline electroencephalogram, which was normal, and a brain MRI, which revealed a small acute ischemic lesion in the right occipital cortical region (Fig. 1), with normal representation of the main intracranial arterial afferents. Considering the history of MA, treatment with Lamotrigine was recommended, gradually increased up to 50 mg/day, resulting in rapid disappearance of symptoms and complete resolution of the clinical picture after approximately two weeks.

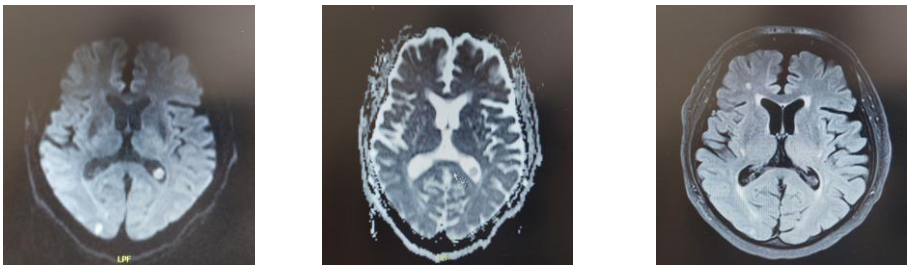

Fig. 1. Brain MRI: ischemic lesion in the right occipital cortical region on DWI (left), ADC (center), and FLAIR (right) sequences.

DISCUSSION AND CONCLUSIONS Patients with a history of MA can underestimate focal stroke symptoms because of their similarity to aura manifestations, making diagnosis challenging. The clinical presentation of our patient did not meet the criteria for either a “migrainous infarction,” characterized by a typical aura lasting more than 60 minutes, or for “status migrainosus,” which is instead defined by a long-lasting headache (>72 hours) preceded by an aura of typical duration. In the pathogenesis of our patient’s ischemic stroke, in addition to vascular risk factors such as arterial hypertension and dyslipidemia, the possible role of MA and the recent coronary revascularization procedure cannot be overlooked. Cerebral vulnerability induced by migraine, mainly through mechanisms such as cortical spreading depolarization, endothelial dysfunction and a prothrombotic state, may act synergistically with the hemodynamic and embolic stress caused by the aforementioned procedure, together with the use of vasodilator drugs, in facilitating the onset of a cerebral ischemic lesion. This suggests the importance of including also nontraditional risk factors (e.g., migraine) in the management of vascular patients, as exemplified by the QRISK3 model.

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