PARADIGM SHIFT: from transient ischemic attacks to transient bleeding attacks. Cortical Subarachnoid Hemorrhage related Transient Focal Neurological Episodes and emergency pitfalls.

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Background

Transient Focal Neurological Episodes (TNFEs) as clinical manifestation of cerebral amyloid angiopathy (CAA) may be mistaken for transient ischemic attacks (TIAs) especially in the emergency room (ER). The spectrum of "suspected" CAA (sCAA) relies mostly on blood sensitive imaging biomarkers including cortical subarachnoid hemorrhage, (c-SAH), cortical superficial siderosis (CSS) and cerebral microbleeds (CBMs).

Case description

We present a 67 years old man, admitted to the ER for paresthesia starting at in his left hand, spreading up his left arm into the left side of his face over less than 10 minutes. Neurological examination was unremarkable. Acute brain CT scan was unremarkable. The patient was discharged after anti-PLT therapy with aspirin 300 mg daily was started. Although his medical history included several cardiovascular risk factors (hypertension, dyslipidemia, bilateral carotid endoarterectomy and ischemic cardiomyopathy) he wasn't on anti-PLT therapy previously. The same night and the day after he experienced two further identical episodes and was readmitted to the ER. A repeated brain CT scan showed c-SAH of the right central sulcus. Brain MRA was unremarkable. Brain SWI-MRI showed disseminated CSS, a finding consistent with CAA. Anti PLT was discontinued. Antiepileptic treatment was initiated.

Discussion

TNFEs may be recurrent, stereotyped, brief (usually <30 min) with a wide spectrum of clinical features. They are common in CAA, being found in about 14% of patients with probable or possible CAA. TFNEs may be caused by CSS, cSAH, or lobar CMBs. The prevalence of TFNEs among patients with sCAA is significantly higher in those with CSS/cSAH and anatomically related to cSAH. Other possible TNFEs-mechanisms include cortical spreading depression or focal seizure activity. The differential diagnosis of TNFEs include TIA, seizure and migraine. Repeated CT scan revealed acute c-SAH. cSAH is common in subjects with sCAA and may cause a significantly higher risk of future cerebral bleeding events (ICH or cSAH) regardless of the severity of the baseline CMBs burden. Acute Brain CT may fail to show the CAA related hemorrhagic abnormalities. SWI-MRI should at least be planned early for elderly patients with atypical TIA presentations.

Suggested Readings

•Charidimou A, Baron JC, Werring DJ. Transient focal neurological episodes, cerebral amyloid angiopathy, and intracerebral hemorrhage risk: looking beyond TIAs. _et_al-2013-International_Journal_of_Stroke. Int J Stroke. 2013 Feb;8(2):105-8.

•Ni J, Auriel E, Jindal J, Ayres A, Schwab KM, Martinez-Ramirez S, Gurol EM, Greenberg SM, Viswanathan A. The characteristics of superficial siderosis and convexity subarachnoid hemorrhage and clinical relevance in suspected cerebral amyloid angiopathy. Cerebrovasc Dis. 2015;39(5-6):278-86.

•Calviere L, Cuvinciuc V, Raposo N, Faury A, Cognard C, Larrue V et al. Acute Convexity Subarachnoid Hemorrhage Related to Cerebral Amyloid Angiopathy: Clinicoradiological Features and Outcome. J Stroke Cerebrovasc Dis. 2016 Feb 25. pii: S1052-3057(15)00621-7.

Conclusions

Any decision on acute treatment with anti-PLT or anticoagulant drugs for presumed TIA should take into account the possibility that transient symptoms may be related to an underlying CAA (CAA-related TFNEs) which is likely to increase the risk of drug-related cerebral bleeding. SWI-MRI may be reasonable and should be performed as soon as possible in this clinical setting.

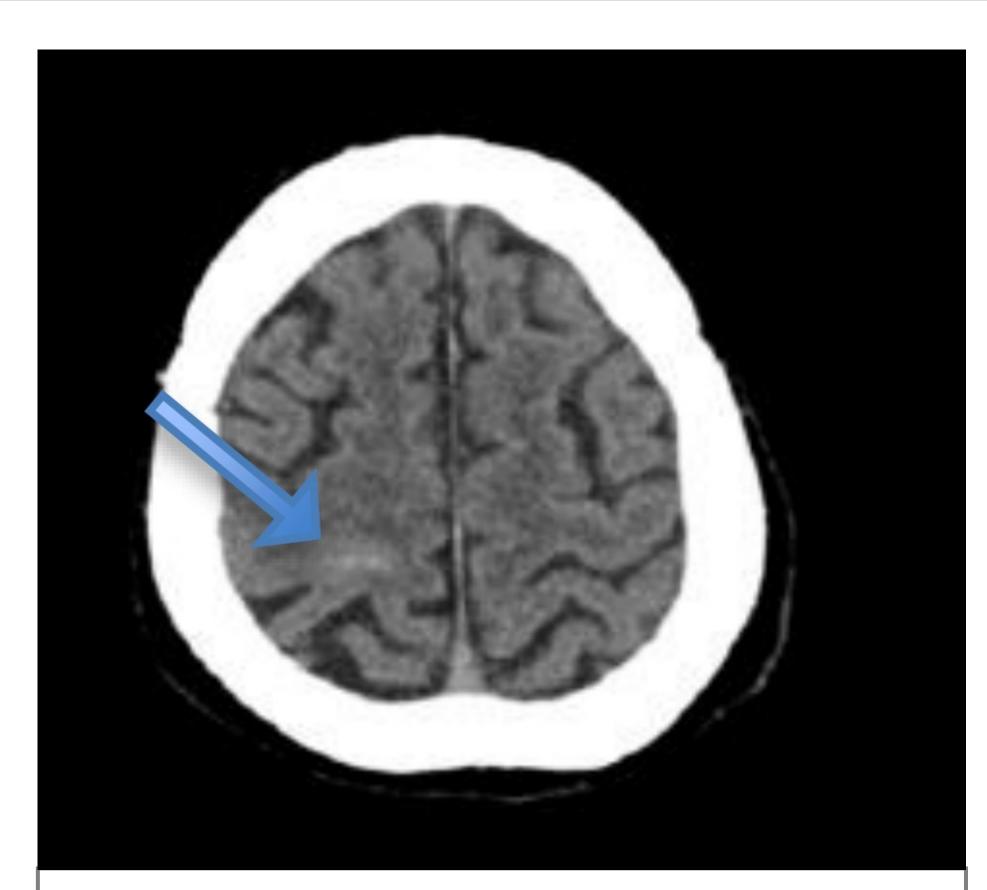


Figure 1.
Repeated CT scan showing focal cortical sub-arachnoid hemorrhage in the right central sulcus.

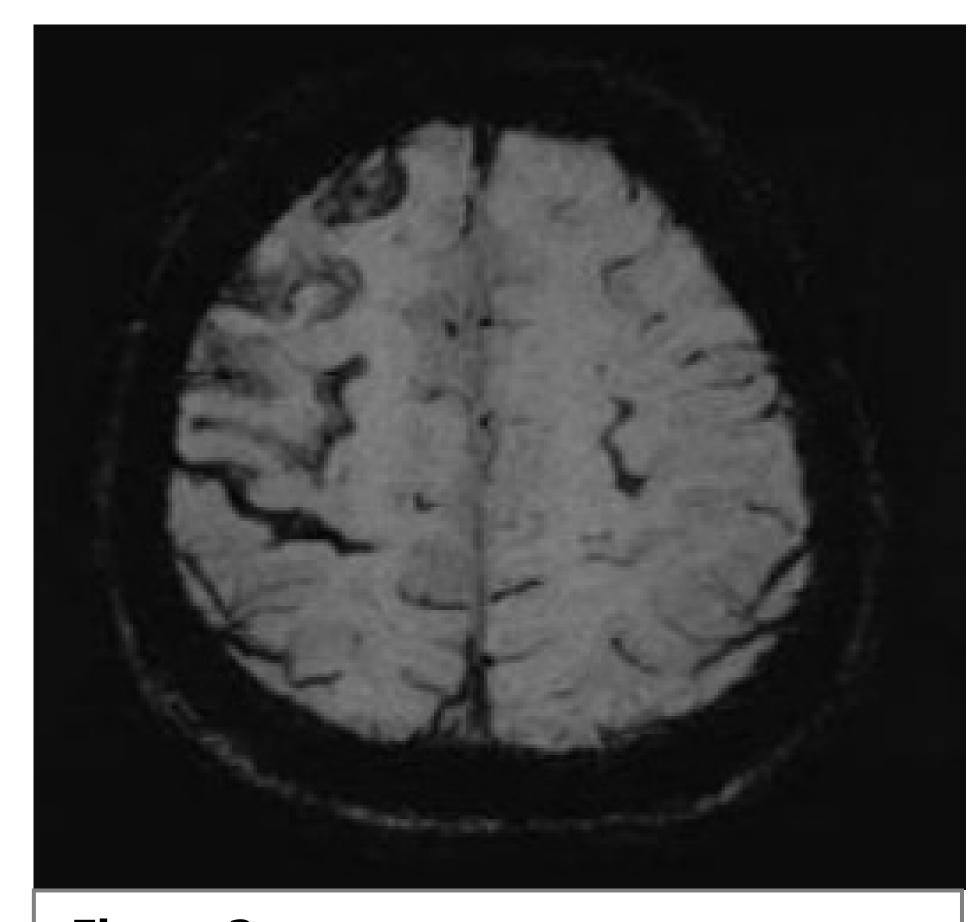


Figure 2.
SWI-MRI showing disseminated superficial cortical siderosis consistent with CAA.

