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Recurrent Transient Ischemic Attacks Revealing Cerebral Amyloid Angiopathy: A Comprehensive Case

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Abstract

This case report investigates the manifestation of cerebral amyloid angiopathy (CAA) through recurrent Transient Ischemic Attacks (TIAs) in an 82-year-old patient. Despite initial diagnostic complexities, cerebral angiography-MRI revealed features indicative of CAA. Symptomatic treatment resulted in improvement, but the patient later developed a fatal hematoma. The discussion navigates the intricate therapeutic landscape of repetitive TIAs in the elderly with cardiovascular risk factors, emphasizing the pivotal role of cerebral MRI and meticulous bleeding risk management. The conclusion stresses the importance of incorporating SWI sequences, specifically when suspecting a cardioembolic TIA, as a diagnostic measure to explore and exclude CAA in the differential diagnosis. This case report provides valuable insights into these challenges, highlighting the need to consider CAA in relevant cases.

Keywords

Cerebral Amyloid Angiopathy, Transient Ischemic Attacks, Recurrent Hemiparesis, Susceptibility-Weighted Imaging, Cardioembolic Origin, Bleeding Risk Management, Differential Diagnosis

1. Introduction

Cerebral amyloid angiopathy [CAA] is a vascular pathology characterized by the deposition of Aß-amyloid peptides in the walls of cortical and leptomeningeal vessels, leading to small vessel damage [1] [2] [3] [4] [5]. The accumulation of

amyloid protein in cerebral vascular walls increases the risks of both ischemic and hemorrhagic events [1]. The prevalence of CAA increases with age, and it is often associated with conditions such as hypertension. Understanding the significance of CAA is crucial as it presents challenges in diagnosis and management, especially when manifesting through symptoms like Transient Ischemic Attacks (TIAs) [1] [3].

This case report details the case of an 82-year-old patient experiencing repetitive Transient Ischemic Attacks (TIAs) as an initial manifestation of CAA.

2. Case Report

An 82-year-old hypertensive patient was admitted with recurrent left hemiparesis episodes, predominantly brachio-facial, spontaneously regressing within 30 minutes. Despite an unremarkable cerebral angiography-MRI, vascular-like nonspecific hypersignals associated with leukoencephalopathy were observed. With a presentation of recurrent TIAs and an ABCD2 score (a tool used to predict short-term risk of stroke in patients with TIAs) < 4, dual antiplatelet therapy (aspirin and clopidogrel) was initiated. However, daily symptom recurrence indicated a potential cardioembolic origin. Subsequently, a cerebral angiography-MRI with SWI sequences was performed to look for a new ischemic event. The MRI revealed features suggestive of cerebral amyloid angiopathy, including subarachnoid sulcal hemorrhage, microbleeds, and superficial cortical siderosis (**Figure 1**). Cerebral MRI showed T2 FLAIR hypersignal lesions predominantly in the semiovale center (**Figure 2**). The diagnosis of probable cerebral amyloid angiopathy was made following Boston criteria. Symptomatic treatment, including correction of cardiovascular risk factors and hypertension control, resulted in an improvement, reducing the frequency of daily TIAs.

Four months later, the patient developed left hemiplegia, aphasia, agitation, and altered consciousness. Cerebral AngioMRI revealed a large right fronto-temporo-insular hematoma causing mass effect (**Figure 3**). The patient was admitted to the intensive care unit and passed away ten days later.

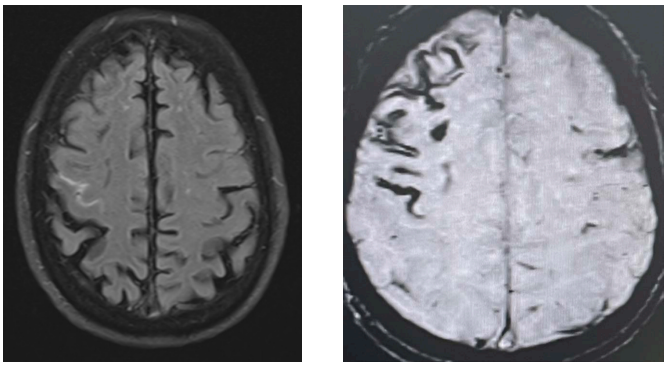

Figure 1. Cerebral MRI with SWI sequences reveals distinctive features, showcasing subarachnoid sulcal hemorrhage, microbleeds, and superficial cortical siderosis (depicted in the right figure). Additionally, the left figure displays a sulcal hyperintense T2 signal associated with the subarachnoid sulcal hemorrhage.

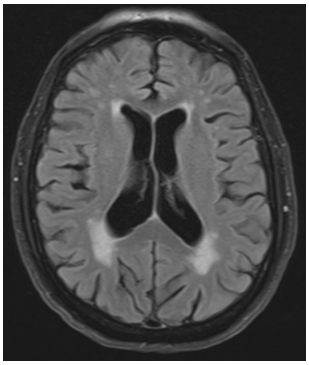

Figure 2. T2 FLAIR hypersignal lesions related to leukoencephalopathy.

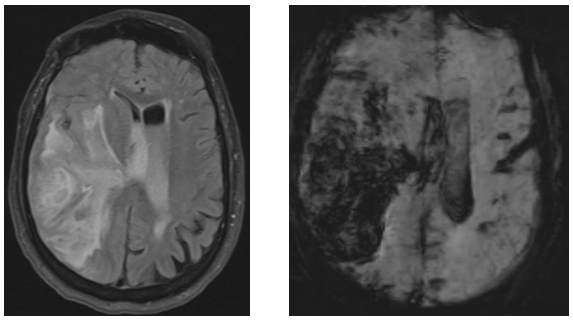

Figure 3. Cerebral AngioMRI disclosed a sizable hematoma in the right fronto-temporo-insular region, inducing a noticeable mass effect. The left picture illustrates the hematoma on the T2 FLAIR sequence, while the right picture captures the hematoma on the SWI sequence.

3. Discussion

Repetitive Transient Ischemic Attacks (TIAs) are a therapeutic challenge, especially in the elderly with cardiovascular risk factors [1] [3]. Cerebral amyloid angiopathy [CAA] should be considered in the presence of a stuttering focal neurological deficit of brutal onset. The initial step is to establish the indication of thrombolysis. In cases of sudden-onset focal neurological deficits, cerebral MRI helps identify possible ischemic signs on diffusion-weighted imaging (stroke) or their absence (TIA). The T2* sequence is valuable for excluding microbleeds, cerebral amyloid angiopathy, or hematoma [1] [2]. Managing bleeding risk is essential, especially when anticoagulation is required, notably in cardioembolic TIAs [3]. Antiplatelet agents and anticoagulants are contraindicated in CAA [3].

Patients with CAA revealed by repetitive TIAs and superficial cortical siderosis have a higher risk of large lobar hematomas [1] [2] [3], as observed in our patient. The treatment of cerebral amyloid angiopathy is primarily symptomatic, focusing on managing cardiovascular risk factors [1] [2] [3]. Controlling hypertension is a critical element in these patients, especially to reduce the risk of hemorrhagic transformation. The target blood pressure in this patient population is <120/80 mmHg [3]. In our patient, hypertensive peaks were directly correlated with recurrent deficits (lasting approximately 15 minutes). Blood pressure control helped reduce the number of daily TIAs.

Superficial cortical siderosis can cause transient focal neurological deficits [2] [3]. The pathophysiology of these events is controversial, but the most plausible hypothesis is focal cortical depolarization that can generalize. Clinicians may suggest antiepileptic treatments such as sodium valproate, lamotrigine, or topiramate [3].

4. Conclusion

In cases of repetitive TIAs suggesting a cardioembolic origin, obtaining an SWI sequence is crucial to raise awareness of the possibility of cerebral amyloid angiopathy. This article provides some insights into the challenges posed by repetitive TIAs and underscores the significance of considering cerebral amyloid angiopathy in the differential diagnosis.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Charidimou, A., Boulouis, G., Gurol, M.E., Ayata, C., Bacskai, B.J., Frosch, M.P., *et al.* (2017) Emerging Concepts in Sporadic Cerebral Amyloid Angiopathy. *Brain*, **140**, 1829-1850. https://doi.org/10.1093/brain/awx047

[2] DeSimone, C.V., Graff-Radford, J., El-Harasis, M.A., Rabinstein, A.A., Asirvatham, S.J. and Holmes, D.R. (2017) Cerebral Amyloid Angiopathy: Diagnosis, Clinical Implications, and Management Strategies in Atrial Fibrillation. *Journal of the American College of Cardiology*, **70**, 1173-1182. https://doi.org/10.1016/j.jacc.2017.07.724

[3] Kozberg, M.G., Perosa, V., Gurol, M.E. and van Veluw, S.J. (2021) A Practical Approach to the Management of Cerebral Amyloid Angiopathy. *International Journal of Stroke*, **16**, 356-369. https://doi.org/10.1177/1747493020974464

[4] Salam, S., Anandarajah, M., Al-Bachari, S., Pal, P., Sussman, J. and Hamdalla, H. (2017) Relapsing Cerebral Amyloid Angiopathy-Related Inflammation: The Wax and The Wane. *Practical Neurology*, **17**, 392-395. https://doi.org/10.1136/practneurol-2017-001599

[5] Rempe, T., Sollero, C.E.V., Rodriguez, E., Viswanathan, V.T., Carlson, A., Rees, J., *et al.* (2020) Corticosteroids Lead to Short-Term Improvement in Cerebral Amyloid Angiopathy-Related Inflammation. *Journal of Neuroimmunology*, **348**, 577377. https://doi.org/10.1016/j.jneuroim.2020.577377