International Journal of Cerebrovascular Disease and Stroke
Ahmed HK, Int J Cerebrovasc Dis Stroke 8: 192
www.doi.org/10.29011/2688-8734.000090
www.gavinpublishers.com

Research Article

Amyloid Spells - Transient Focal Neurological Deficits in Cerebral Amyloid Angiopathy

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Citation: Ahmed HK (2025) Amyloid Spells - Transient Focal Neurological Deficits in Cerebral Amyloid Angiopathy. Int J Cerebrovasc Dis Stroke 8: 192. DOI: 10.29011/2688-8734.100192

Received Date: 23 December, 2024; **Accepted Date:** 02 January, 2025; **Published Date:** 10 January, 2025

Keywords

Intracerebral Hemorrhage (ICH), Computed Tomography (CT), Cerebral Amyloid Angiopathy (CAA), Magnetic Resonance Imaging (MRI).

Introduction

This case report describes the rare occurrence of Amyloid Spells - transient neurological deficits in a 70-year-old female, aiming to create awareness of this diagnose amongst neurologists and other clinicians nationally and internationally.

Case Presentation

Magnetic Resonance Imaging (MRI) was performed when a seventy-year-old woman was admitted for investigation of repeated, transient focal neurological deficits. During the last 3 weeks prior to the admission, she had endured 5-7 episodes of aphasia, right-sided numbness and paralysis in the upper and lower extremities, lasting for around 10 minutes each.

The stereotype attacks always initiated at the same localization on the right-hand's 2. to 4. fingers while spreading to the rest of the arm and additionally to the right side of the body during 1 minute. Hereafter, she developed paralysis and aphasia without any symptoms as headache or impact of consciousness.

Prior to the symptom debut, the patient was well-functioning, living with her spouse. Anamnestic, she indicated having memory challenges during the last 5 years but refused other cerebral symptoms. Her memory capability was not assessed clinically during the hospitalization. The patient was non-smoker and had never smoked in her life. She had not been diagnosed with diabetes, hypertension, ischemic cardiac disease or cerebrovascular disease.

Clinical Assessment

Neurological assessment was performed at admission and was normal. Blood pressure was measured to 174/84, angiopathy blood tests were inconspicuous and ECG was also assessed to be normal.

Differential diagnoses are Transient Ischaemic Attack (TIA), Epileptic or Psychogenic Non-Epileptic Seizures (PNES) and Migraine aura without the headache.

Radiological Assessment

At admission, Computed Tomography (CT) of the brain demonstrated low attenuating changes in the sub-cortical region on both sides as an expression of chronic microangiopathy and as well for her undiagnosed hypertension. CT-angiography also showed minor atherosclerotic changes in the carotid bifurcation on both side without exhibiting significant stenoses. Standard EEG showed alfa-basic rhythm with elements of irregular 2-4 Hz waves with diffuse distribution and predominance of frontal localization on both sides.

Cerebral MRI exhibited changes indicating Cerebral Amyloid Angiopathy (CAA) as accumulation of the protein Amyloid-Beta was seen in the cortical and leptomeningeal vessel walls. Transient focal neurological deficits in patients with CAA is termed «Amyloid Spells» and occurs in around 20 % of the patients [1]. These episodes are often stereotypical with wandering clinical symptoms and «Amyloid spells» can easily be confused with TIA [2].

Discussion

However, it is discussed that focal epileptic attack, changed cortical activity so-called «Cortical Spreading Depression» and local spasms happens due to accumulation of blood degradation products as etiology of transient neurological deficits in CAA [1].

Antithrombotic treatment should be avoided in patients with CAA due to increased risk of intracerebral hemorrhage (ICH). Antiepileptic treatment and migraine prophylaxis is suggested as possible therapeutical alternatives [3-5]. In this patient in hand, antiepileptic medication was initiated in form of Keppra (Levetiracetam), which had a good clinical effect on the transient neurological deficits.

Conclusion

MRI of the brain shows Cerebral Amyloid Angiopathy (CAA) with superficial siderose, cortical microbleedings and small parenchymal bleedings in the right gyrus cinguli and left temporal lobe.

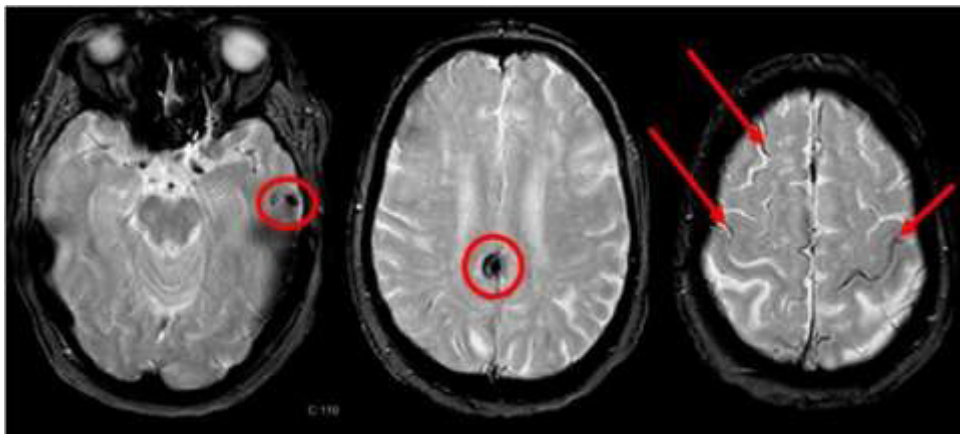

Figure 1: The T2* serie of the brain (Philips Ingenia 1,5 T, Release 5.3, TE 86,6, TR 3074, slice-thickness 5 mm, 1 mm gap) showed hypointense signal changes along the surface of the brain (see arrows) compatible with superficial siderose and small parenchymal bleedings in the right gyrus cinguli and the left temporal lobe (circles). The findings are compatible with Cerebral Amyloid Angiopathy (CAA).

Acknowledgement:

This case report constitutes original work and is not earlier published elsewhere.

Consent for Publication

Written consent was obtained from the patient prior to the publication of this case report.

Conflict of Interest

The author declares conflicting interest none to disclose.

Sources and Funding:

This work has not received any grants or funding.

References

1. Charidimou A, et al. (2012) Spectrum of transient focal neurological episodes in cerebral amyloid angiopathy: multicentre magnetic resonance imaging cohort study and meta-analysis. Stroke 43: 2324-2330.
2. Charidimou A, R Law, and DJ Werring (2012) Amyloid “spells” trouble. Lancet 380: 1620.
3. Coates R, et al. (2015) Cerebral amyloid angiopathy: amyloid spells and cortical superficial siderosis. Pract Neurol 15: 124-126.
4. Cooperman SS, AK Shah, and K Rajamani (2019) Amyloid spells. Neurol Clin Pract 9: p.e17-e18.
5. Roch JA, et al. (2005) Transient neurologic symptoms related to cerebral amyloid angiopathy: Usefulness of T2’weighted imaging. Cerebrovasc Dis 20: 412-414.