Case Lessons 30-2025

Amyloidosis is not multiple cavernoma

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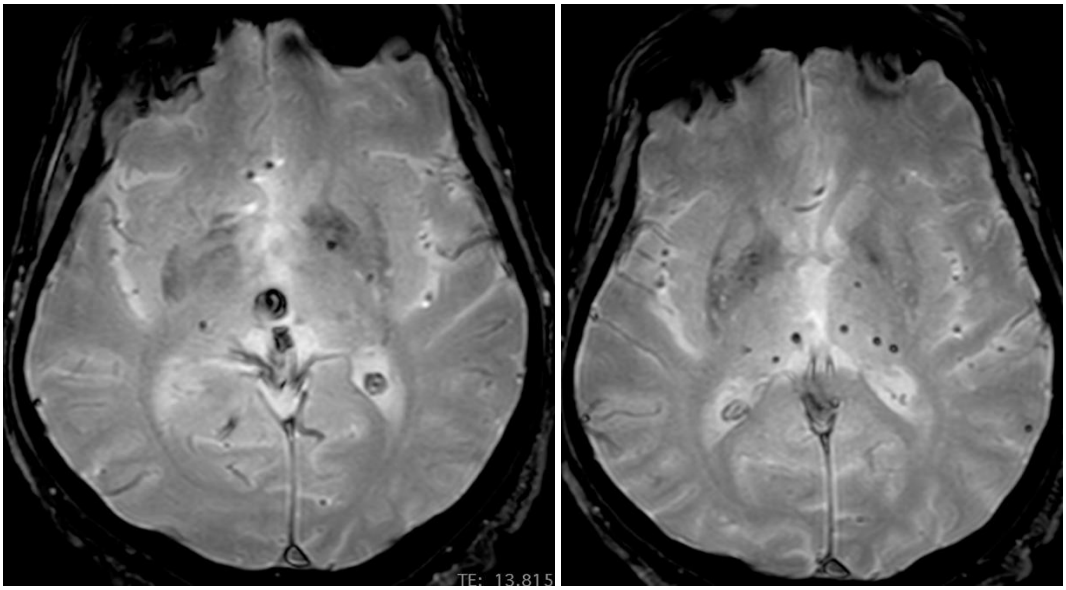
Introduction: Cerebral amyloid angiopathy (CAA) is characterized by amyloid-β deposition in the walls of small and medium-sized cerebral arteries, primarily in the cortical and leptomeningeal regions. This disrupts vascular integrity and makes vessels fragile to rupture causing smooth muscle cell loss, fibrinoid necrosis, and microaneurysm formation. As a result, patients often suffer from lobar hemorrhages, transient focal neurological episodes, and cognitive decline.

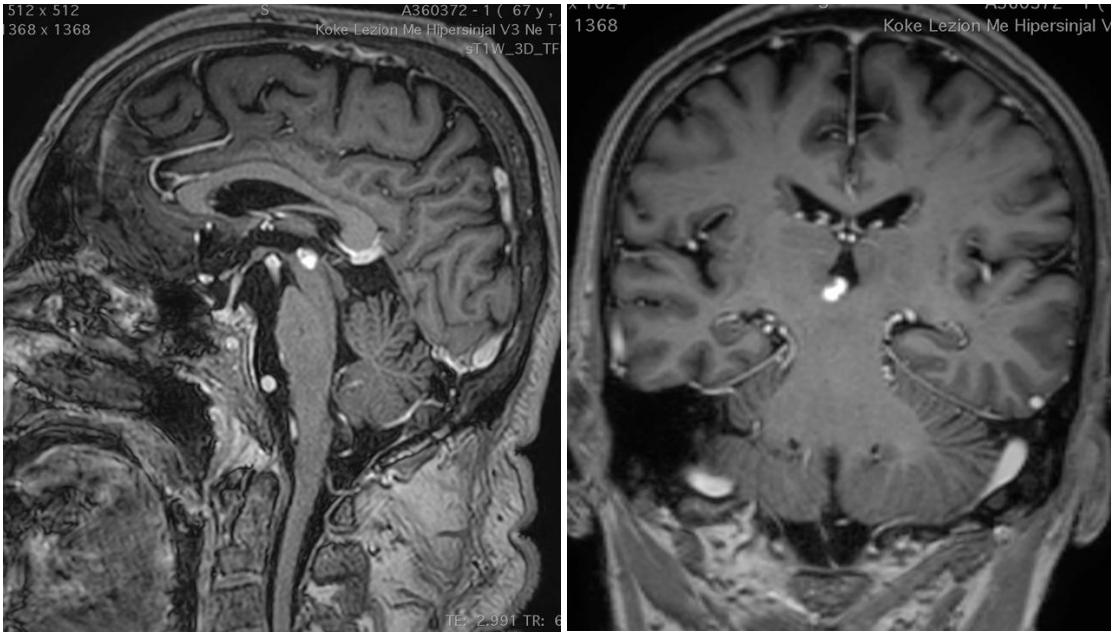
CAA and atrial fibrillation (AF) are both common age-associated conditions that can significantly impact cerebrovascular health. CAA predisposes patients to spontaneous lobar intracerebral hemorrhages (ICH), while AF is a major cause of cardioembolic ischemic stroke. The conventional approach to stroke prevention in AF includes oral anticoagulation, which is often contraindicated in patients with CAA due to increased bleeding risk [1].

Case presentation: A 68 years old male under treatment with Novel Oral Anticoagulation (NOAC) (Xarelto 20mg/day, once daily) for chronic non-valvular atrial fibrillation. He had sudden diplopia and ataxia about one month ago and was admitted in another hospital. Head CT scan showed a haemorrhagic lesion at the level of third ventricle. No vasal lesions were seen in supra-aortic Angio-CT. Brain MRI was also performed and multiple cavernomas to be followed was the diagnosis. He was discharged from the hospital with Enoxaparin 0.4UI, as recommended by the cardiologist.

The patient was revisited as outpatient in our hospital. Brain MRI showed clear signs of Cerebral Amyloid Angiopathy with multiple microhaemorrhages. Anticoagulation was immediately stopped. Under this suggestion, the patient was consulted by the cardiologist who recommended Occluding device into Left Auricle to menage the permanent atrial fibrillation instead of anticoagulants due to high risk of bleeding in the brain.



Typical Amyloidosis lesions visible on HEMO MRI sequence of the patient



The Prominent Vascular lesion on the floor of the third ventricle on T1 gad

Discussion: The prevalence of CAA increases with age and studies show that 23% of the general population may have moderate-to-severe CAA. Diagnosis relies on imaging criteria outlined in the modified Boston Criteria [2]. MRI findings such as lobar cerebral microbleeds, cortical superficial siderosis, white matter hyperintensities, and enlarged perivascular spaces are key indicators.

AF patients typically require long-term anticoagulation to prevent ischemic strokes. However, in CAA patients, especially those with prior lobar hemorrhage, anticoagulants and antithrombotic agents significantly elevate the risk of recurrent ICH [3]. Reports say that people are willing to go through 4

bleeding episodes in order to prevent one ischemic stroke [4]. For patients with high bleeding risk due to CAA, left atrial appendage occlusion (LAAO) offers a non-pharmacological alternative for stroke prevention in AF. Risk stratification tools such as CHA2DS2-VASc (Congestive heart failure, Hypertension, Age>75 years, Diabetes mellitus, Stroke/Tia/Systemic embolism, Vascular disease, Age 65-74, Sex category) and HAS-BLED (Hypertension, Abnormal renal/liver function, Stroke, Bleeding history, Labile INR, Elderly (>65 years, Drugs/alcohol concomitantly) scores may be insufficient in this subgroup. Neuroimaging markers of CAA severity should inform decisions [2].

Conclusion: CAA poses a significant challenge to the use of anticoagulation in AF patients due to the heightened risk of intracranial hemorrhage. Early diagnosis, careful neuroimaging evaluation, and consideration of alternative stroke prevention strategies such as LAAO are essential.

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3. Merella P, Cocco G, Saba L, et al. When atrial fibrillation meets cerebral amyloid angiopathy: current evidence and strategies. *J Clin Med.* 2023;12(23):7704.
4. Healey JS. Anticoagulation or antiplatelet therapy for device-detected atrial fibrillation. *N Engl J Med.* 2025;392:1749-1752.