# Case lesson 37/2025

## Brain amyloidosis mimicking metastases, a challenge

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**Introduction:** Cerebral amyloid angiopathy (CAA) is a cerebrovascular disorder characterized by deposition of β-amyloid in the walls of small- to medium-sized cortical and leptomeningeal vessels. In elderly patients, it frequently manifests with lobar intracerebral hemorrhage, microbleeds, cortical superficial siderosis, or cognitive impairment in elderly[1]. Less common, CAA and its related entities can resemble as tumor-like or mass-like lesions mimicking neoplastic disease, creating significant diagnostic uncertainty. Primary brain amyloidoma, a rare focal amyloid deposition, represents another diagnostic challenge as it can be radiologically indistinguishable from metastases or gliomas. Differentiating between metastatic tumors and amyloid-related lesions is essential, given their different management approaches[2].

**Case report:** A 71-year-old man with a history of stage I hypertension and dyslipidemia presented to ER with severe headache, disoriented and progressive left lower limb paresthesia. He was not on blood thinners treatment. First brain MRI at 24 hours revealed a right pre-Rolandic hyperintense lesion in T2-weighted image, suggestive an old hematoma and a left temporal acute hemorrhage (Figure 1). He was conservatively treated for the diagnosis of intraparenchymal hematoma, and discharged one week later without further complications. A second MRI one month after showed a partial absorption of pre-Rolandic lesion and the left Temporal lesion hyperintense in T2 with ring-enhancing contrast in T1-gad, raising suspicion for a brain metastasis (Figure 2). A total body CT scan (thorax, abdomen, pelvis) showed no evidence of a primary tumor or extracranial metastases. There was no history of malignancy. Several weeks later he presented again to ER with left hemiparesis with a motor force of 2/5. Brain MRI demonstrated a new cortico-subcortical lesion in the right temporo-parietal lobe corresponding to an acute hemorrhage in SWI sequence (Figure 3). Brain hemorrhagic metastasis was the most consistent diagnosis. His local neurologist sent him to consultation for a second opinion. Reviewing his MRIs, cortical superficial siderosis was noted along the parietal

sulci. Edema was modest compared to typical metastatic lesions. Given these MRI features, his comorbidities and the negative systemic evaluation, the diagnosis was revised to probable CAA, rather than metastatic disease and another control MRI one month later was recommended and showed the left temporal lesion almost disappeared (Figure 4), and definitive amyloidosis was the diagnosis. The patient was managed conservatively, with emphasis on vascular risk factor control and close neurocognitive monitoring. He was discharged with a slight improvement of the motor deficit and in three months control MRI.

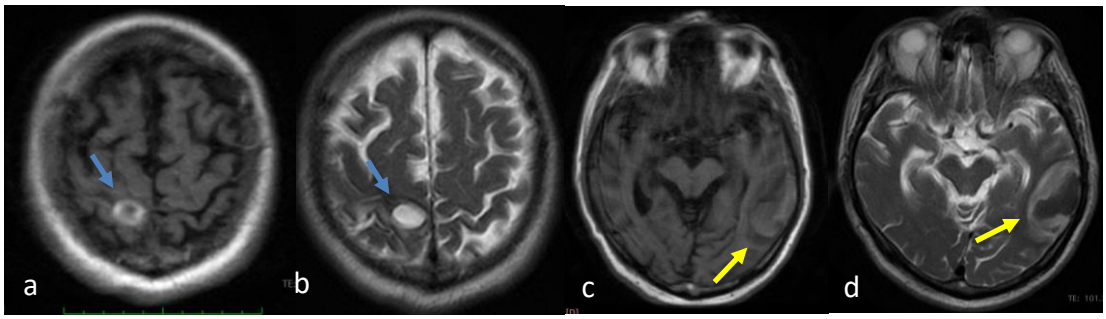


***Figure 1.*** First MRI: a right pre-Rolandic lesion (blue arrow), hypointense with hyperintense ring in T1-weighted sequence (a); hyperintense in T2-weigted sequence (b); and a left temporal lesion (yellow arrow) iso to hyperintense in T1- weighted sequence (c); and hypointense in T2-weighted sequence (d)

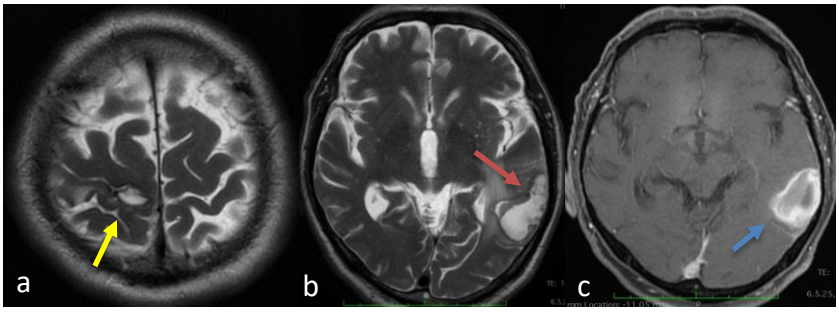


***Figure 2.*** Second MRI: a partial absorption of right pre-Rolandic lesion (a); the left Temporal lesion hyperintense in T2 (b); with ring-enhancing contrast in T1-gad (c)

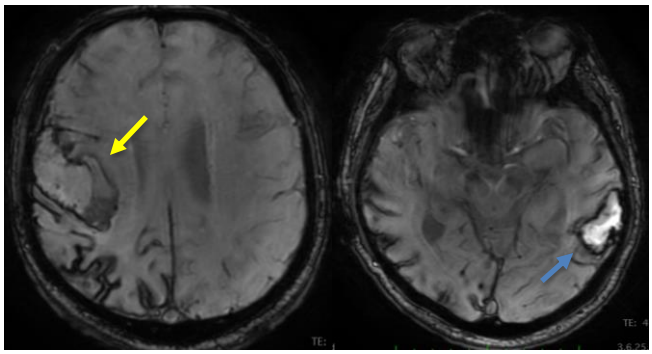

***Figure 3.*** Brain MRI demonstrated a new cortico-subcortical lesion in the right temporo-parietal lobe corresponding to an acute hemorrhage in SWI sequence (yellow arrow) and cortical siderosis in the left temporal lobe (blue arrow)

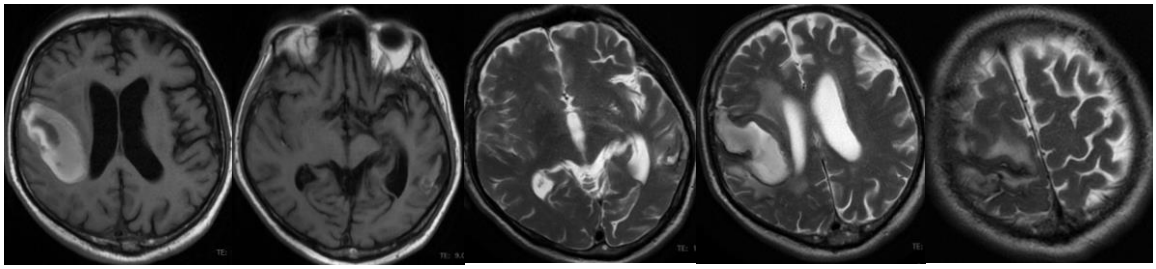

***Figure 4.*** fourth MRI: the right pre-Rolandic lesion is no longer seen, and there is almost total absorption of the left temporal lesion

**Discussion:** This case illustrates the diagnostic challenge of distinguishing between CAA and metastatic brain disease. The clinical course of metastases usually evolves mass effect and focal deficits, making it more difficult to differentiate from CAA related intraparenchymal hemorrhages. Both may appear as enhancing cortico-subcortical lesions in elderly patients. However, imaging clues such as hemorrhagic markers can guide diagnosis. CAA shows microbleeds, cortical superficial siderosis, and hemosiderin deposits on GRE/SWI sequences, while metastases are hemorrhagic only in certain tumor types (e.g., melanoma, renal carcinoma). In addition, CAA lesions favor lobar regions, whereas metastases often localize to the gray-white matter junction and may have leptomeningeal dissemination. A systemic workup is also important, if negative, it argues against metastasis and supports amyloid pathology if there is suspicion evidence on MRI findings[3].

Primary brain amyloidoma, although rare, further complicates the differential diagnosis. It represents localized amyloid deposition that can mimic both neoplastic and degenerative processes, as highlighted in case literature[4] and in our case lesson 30/2025[5]. Careful correlation

of neuroimaging, systemic evaluation, and clinical presentation is mandatory to avoid misdiagnosis and not indicated aggressive treatment such as biopsy and surgery. Only tumor markers in circulating exosomes such as miR-124-3p significantly decreased in brain metastases may be helpful on differential diagnosis according to recent studies[6].

**Conclusion:** In elderly patients with brain lesions suggestive of metastases but no evidence of systemic malignancy, CAA and other amyloid-related pathologies must be considered. Susceptibility-weighted MRI findings, including microbleeds and cortical superficial siderosis, are essential diagnostic clues. Recognizing these features can prevent misdiagnostic oncologic treatment and avoid antithrombotic or anticoagulant medications.

*Acknowledgements: We thank Dr. Kjanda Elpenoria for the collaboration.*

*References*

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