# Cerebral Amyloid Angiopathy

**Joseph Tarsia, MD**

**Vascular Neurology**

Cerebral Amyloid Angiopathy Clinic

Ochsner
The Debra H. and Robert J. Patrick Neuroscience Institute

# Financial Disclosures

- None

# Objectives

- **CAA Pathophysiological Overview**
- **5 Diagnostic and Clinical pearls**
- **Ochsner CAA Multi-Disciplinary Clinic**

# What is **Cerebral Amyloid Angiopathy**?

1. Dietmar Rudolf Thal, Estifanos Ghebremedhin, Mario Orantes, Otmar D. Wiestler, Vascular Pathology in Alzheimer Disease: Correlation of Cerebral Amyloid Angiopathy and Arteriosclerosis/Lipohyalinosis with Cognitive Decline, *Journal of Neuropathology & Experimental Neurology*, Volume 62, Issue 12, December 2003, Pages 1287–1301
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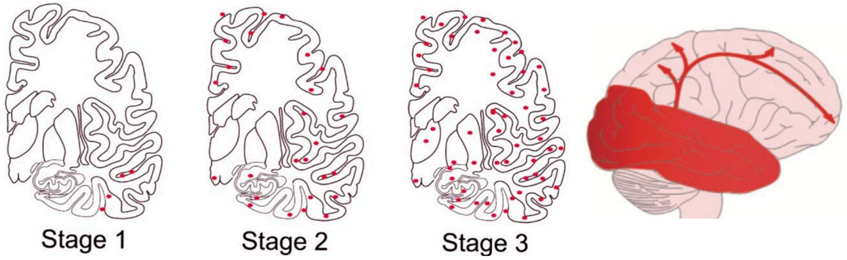


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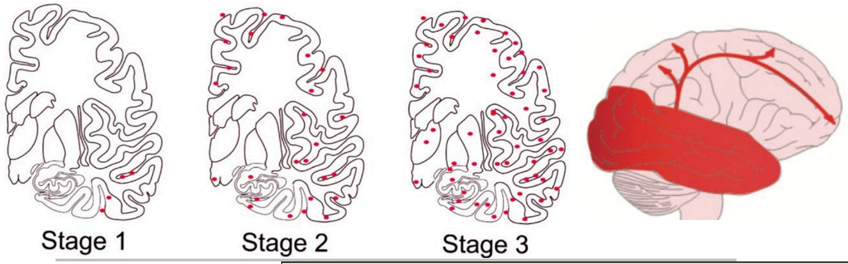


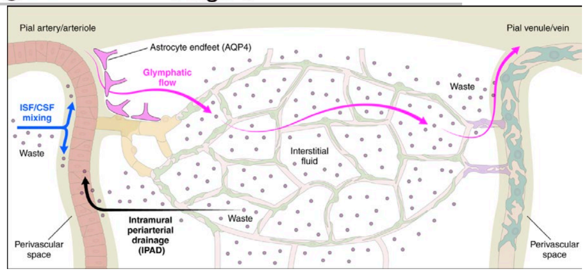



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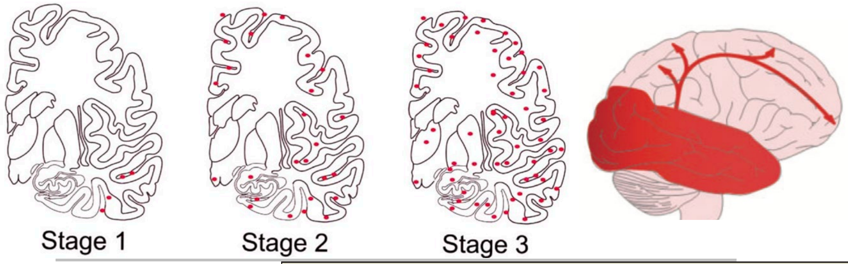


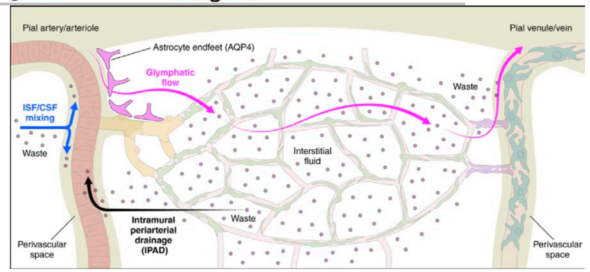



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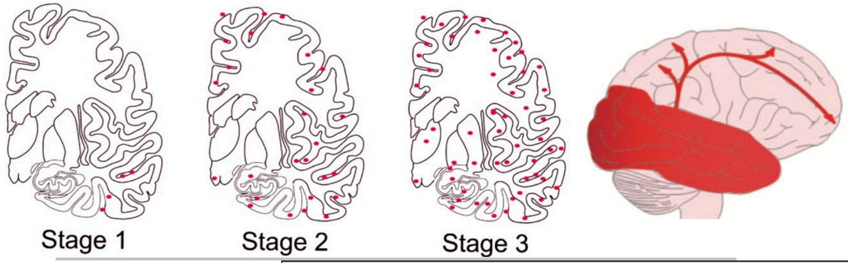
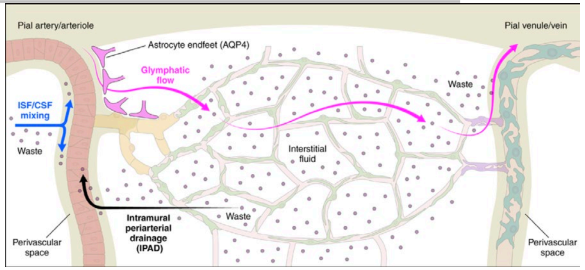
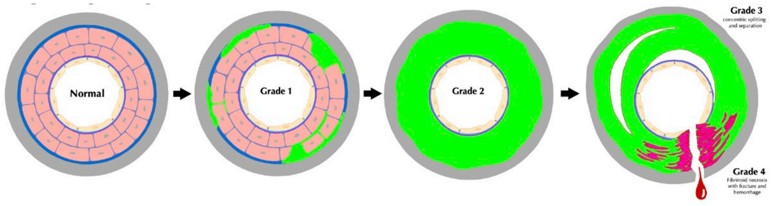
| **-opathy** | Progressive smooth muscle cell loss with neurovascular decoupling & vessel wall remodeling |





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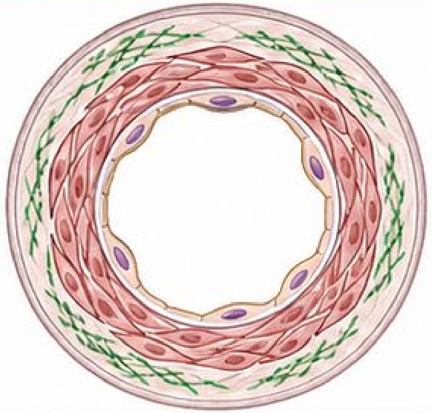
# CAA Pathophysiologic Progression: From Amyloid Deposition to Brain Injury

## Stage 1
### Initial Vascular Amyloid Deposition



**PATHOLOGY** Initial Aβ deposition in vascular basement membranes; smooth muscle cells preserved; vessel wall intact

**BIOMARKER**

( CSF Aβ40 and Aβ42 (earliest detectable change)

**IMAGING**

No overt MRI findings

**CLINICAL** Subclinical / preclinical phase — asymptomatic

~30–20 yrs before first symptomatic hemorrhage

*Timeline derived primarily from Dutch-type hereditary CAA biomarker data; individual sporadic CAA trajectories may vary.*


1. Sellimi A, Schwartze J, Humphries F, Panteleienko L, Mallon D, Banerjee G, et al. Contemporary perspectives in cerebral amyloid angiopathy. Expert Rev Neurother. 2025:1–24. doi: 10.1080/14737175.2025.2526113.
2. Koemans EA, Chhatwal JP, van Veluw SJ, van Etten ES, van Osch MJP, van Walderveen MAA, et al. Progression of cerebral amyloid angiopathy: a pathophysiological framework. The Lancet Neurology. 2023;22(7):632–42. doi: 10.1016/s1474-4422(23)00114-x.
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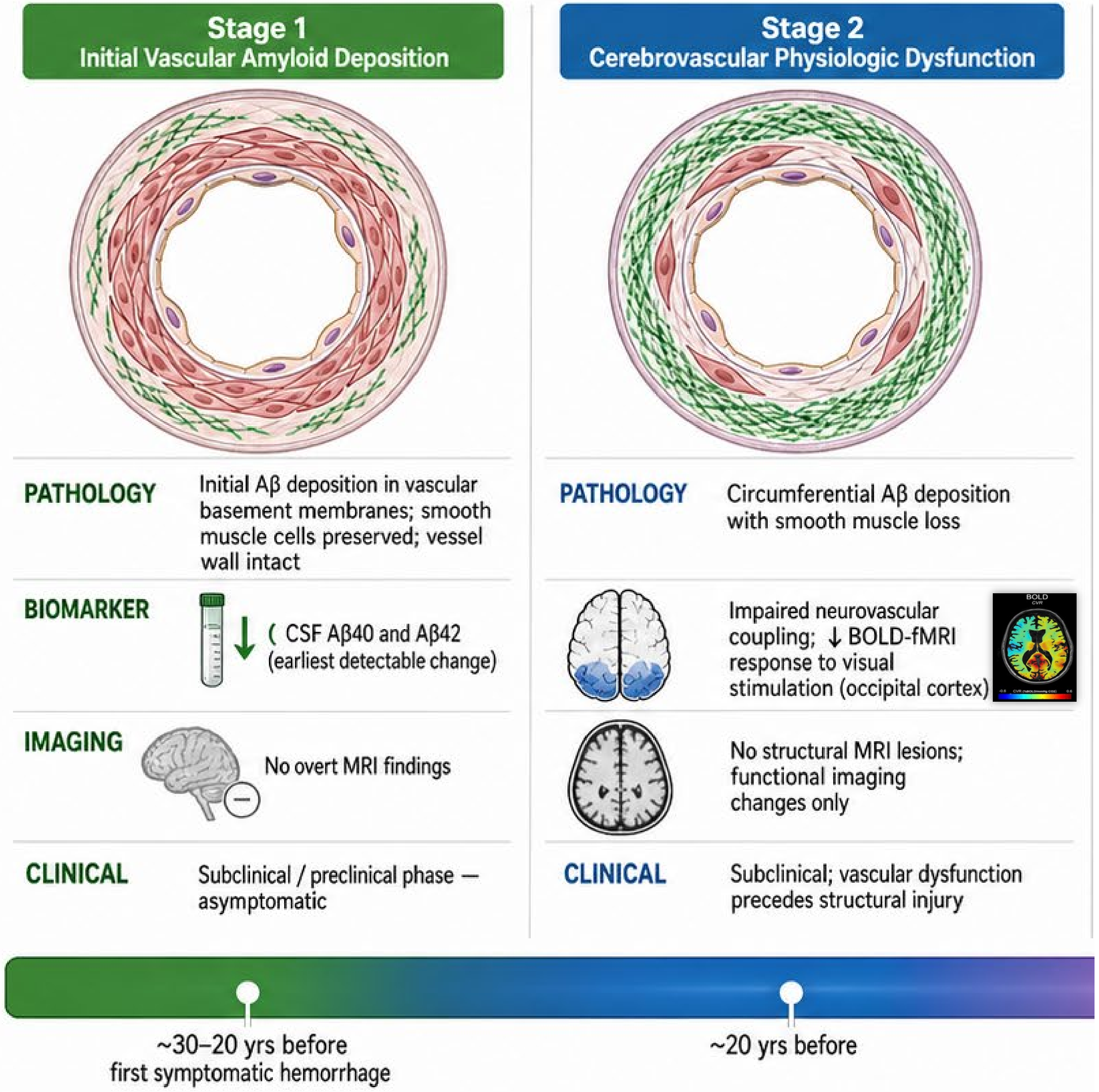


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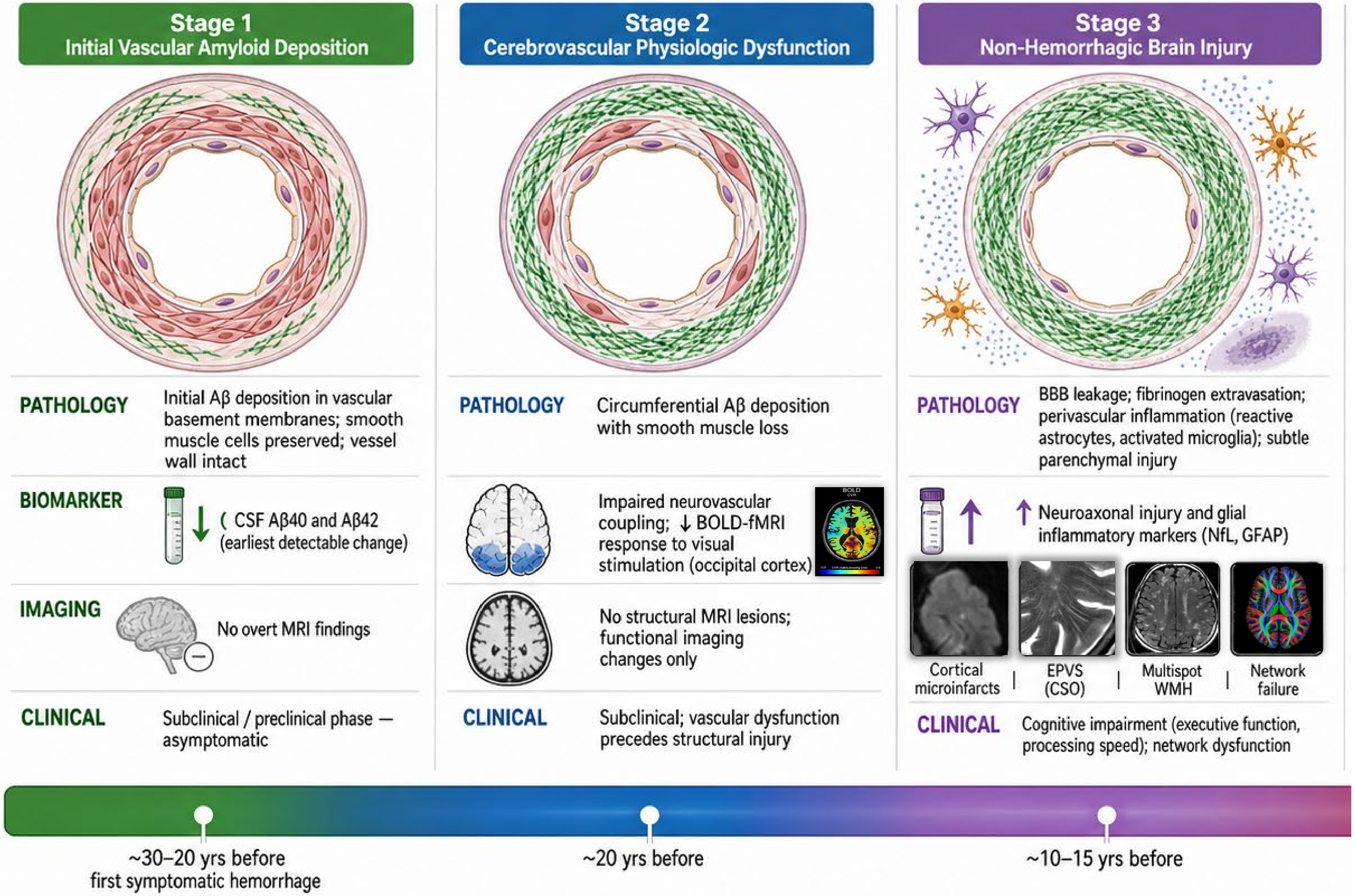


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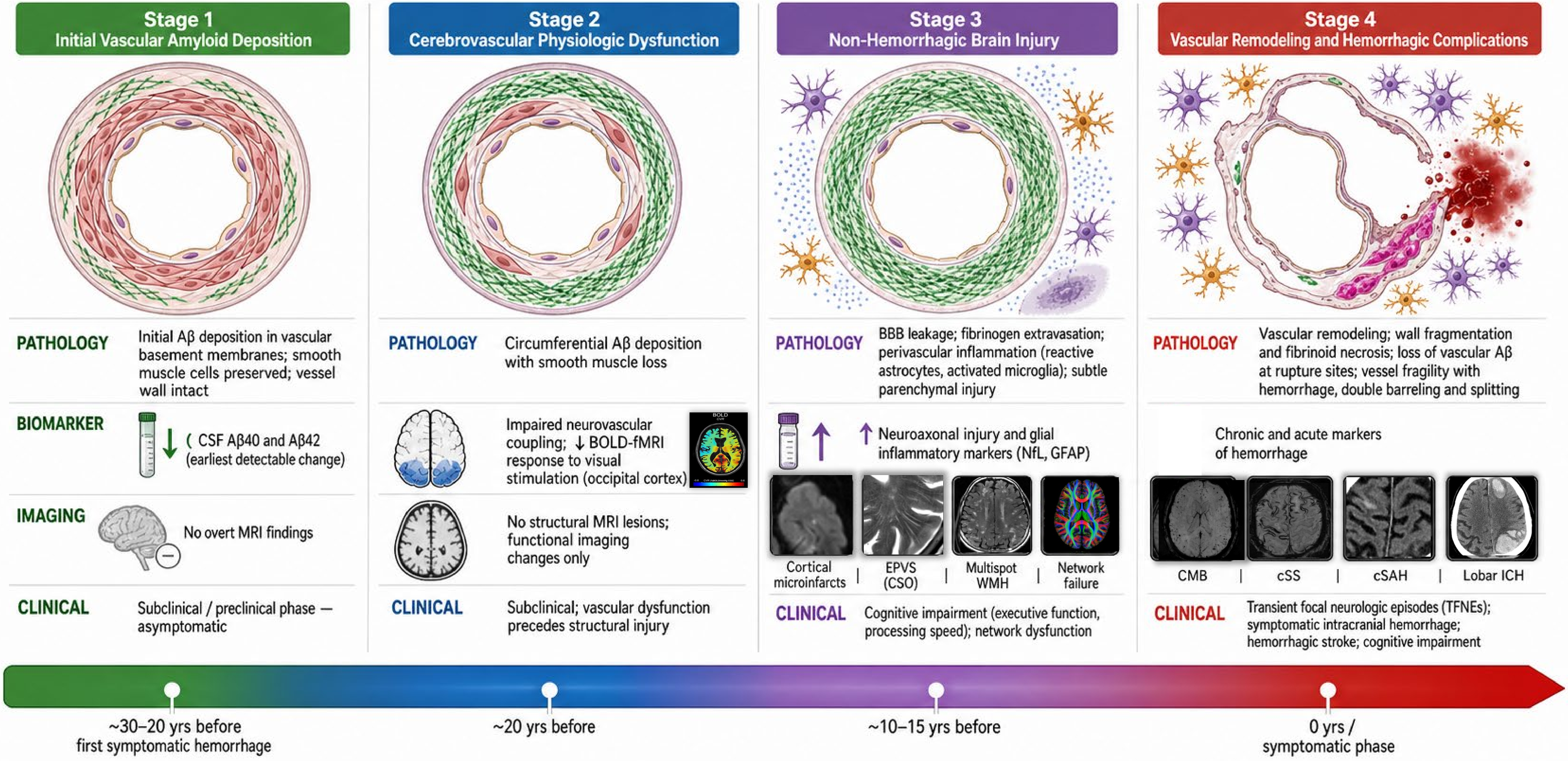


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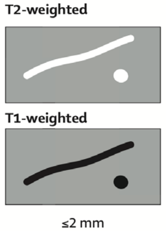
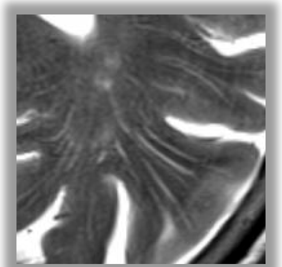
# Cast of Characters

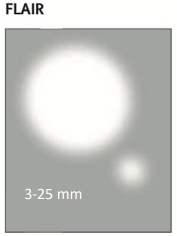
## Non-Hemorrhagic




### Severe Enlarged Perivascular Spaces

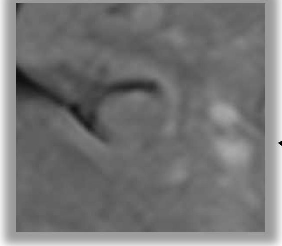
The subcortical spaces around blood vessels become enlarged and visible (>20/hemisphere)

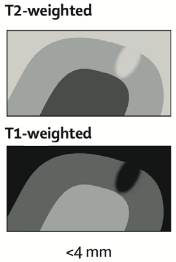





### WMH Multi-Spot Pattern

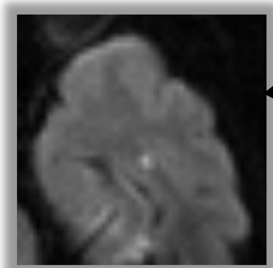
Subcortical ovoid or round areas representing BBB dysfunction or ischemic change (> 10)





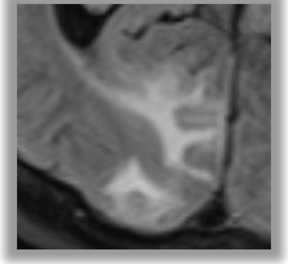

### Cortical Micro-Infarcts

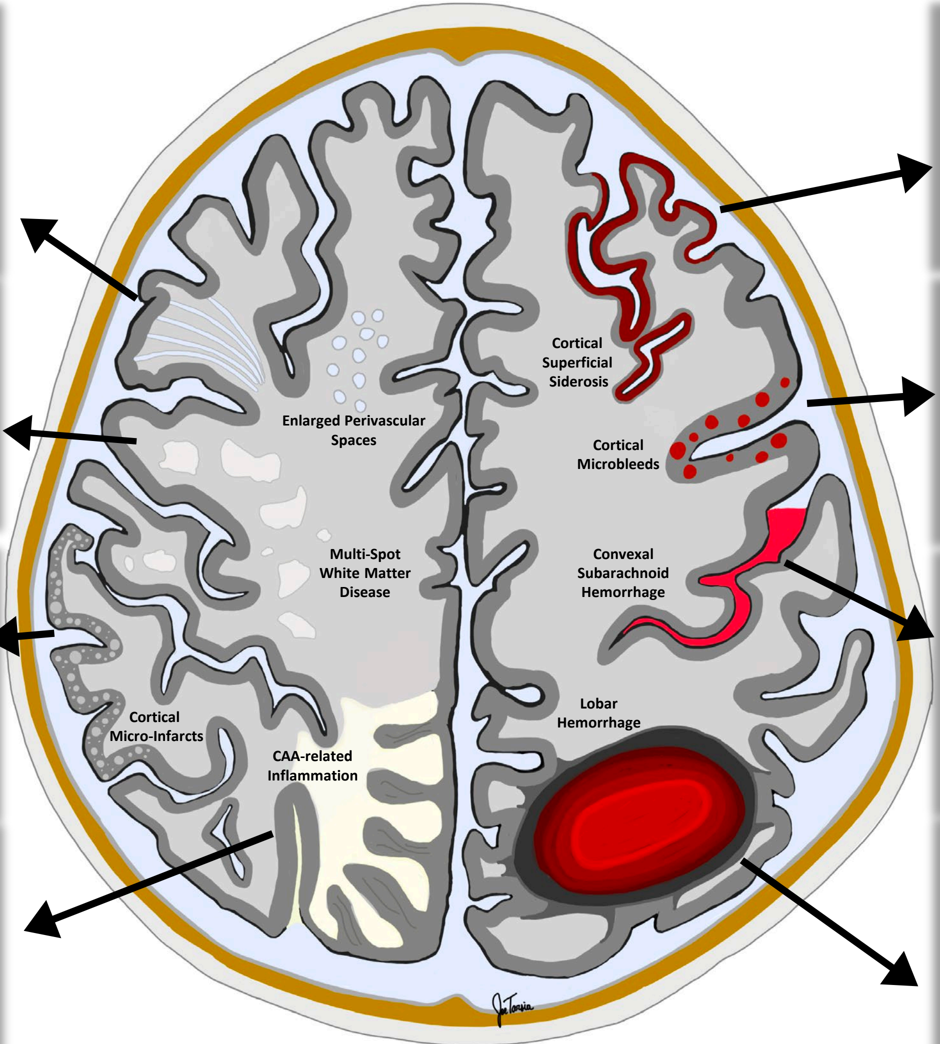
More evident on higher magnet strength. Microscopy is best assessment



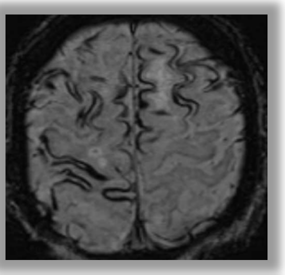
### CAA-ri (related inflammation)

Vasogenic, asymmetric lesions extending to cortex. Also, sulcal effusion, gyral edema leptomeningeal enhancement possible



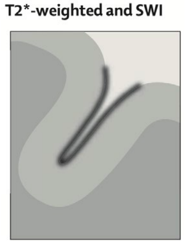


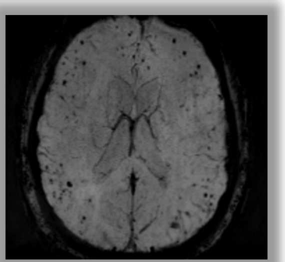

## Hemorrhagic



### Cortical Superficial Siderosis

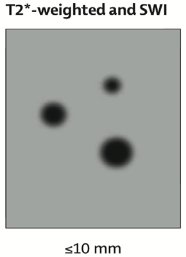
Hemosiderin staining along pial/subpial surface, likely chronic subarachnoid blood

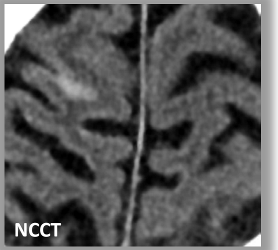


### Cortical Cerebral Microbleeds

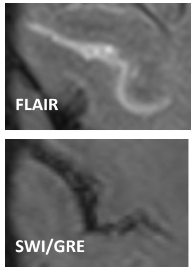
Areas of hemosiderin extravasation from cortical arterioles/capillaries




### Convexal SAH

Acute hemorrhagic lesions within sulci at surface of brain




### Lobar Hemorrhage

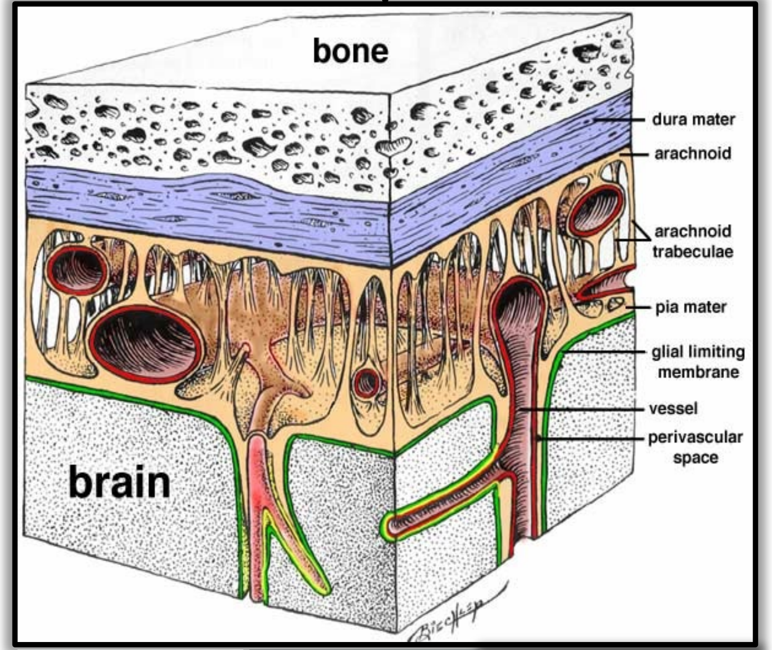
Large (> 10 mm) brain hemorrhage, often but not always symptomatic

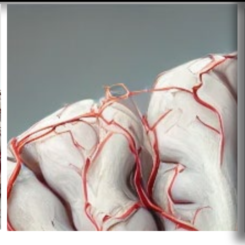

1. Duering M, Biessels GJ, Brodtmann A, Chen C, Cordonnier C, de Leeuw FE, et al. Neuroimaging standards for research into small vessel disease-advances since 2013. Lancet Neurol. 2023;22(7):602-18. Epub 20230523. doi: 10.1016/S1474-4422(23)00131-X. PubMed PMID: 37236211.

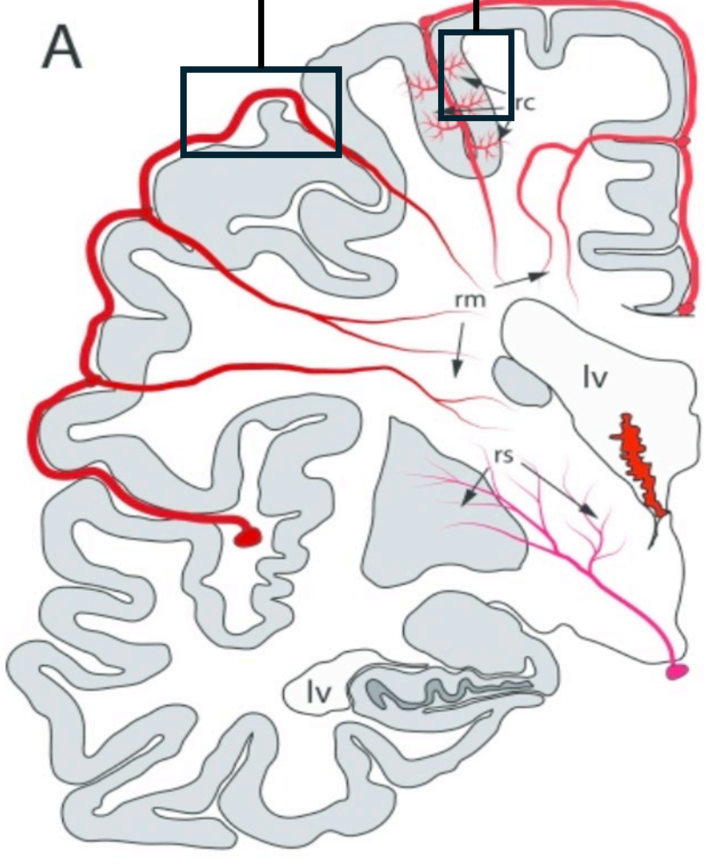
# Leptomeningeal (Pial)
## Arteries & Arterioles

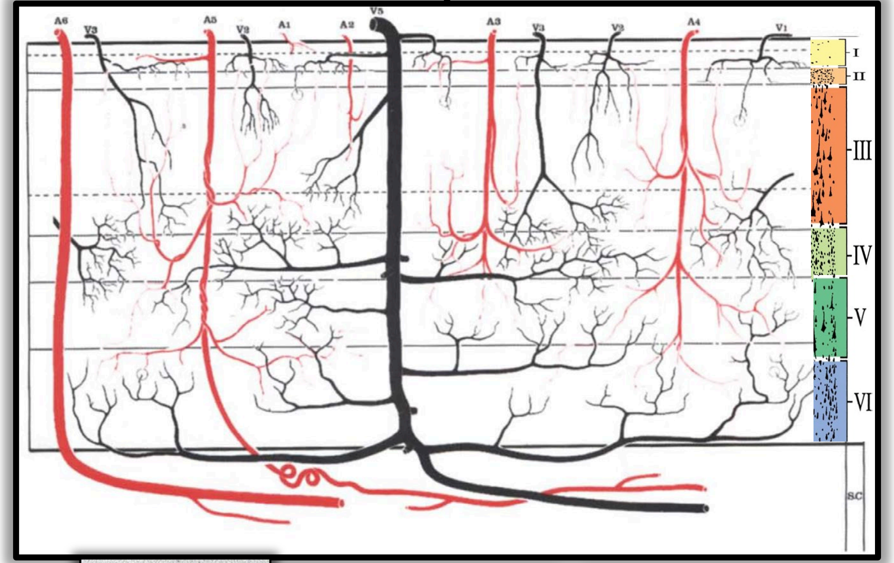
# &

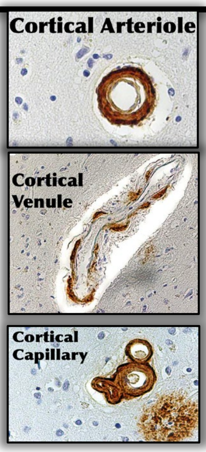
# Cortical
## Arterioles, Capillaries, Venules

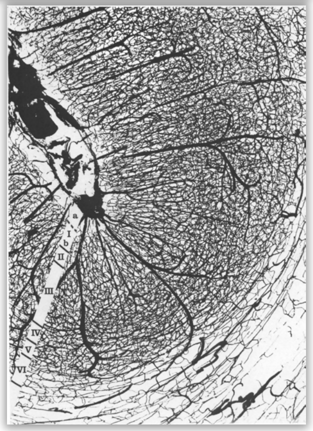
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## Arteries & Arterioles

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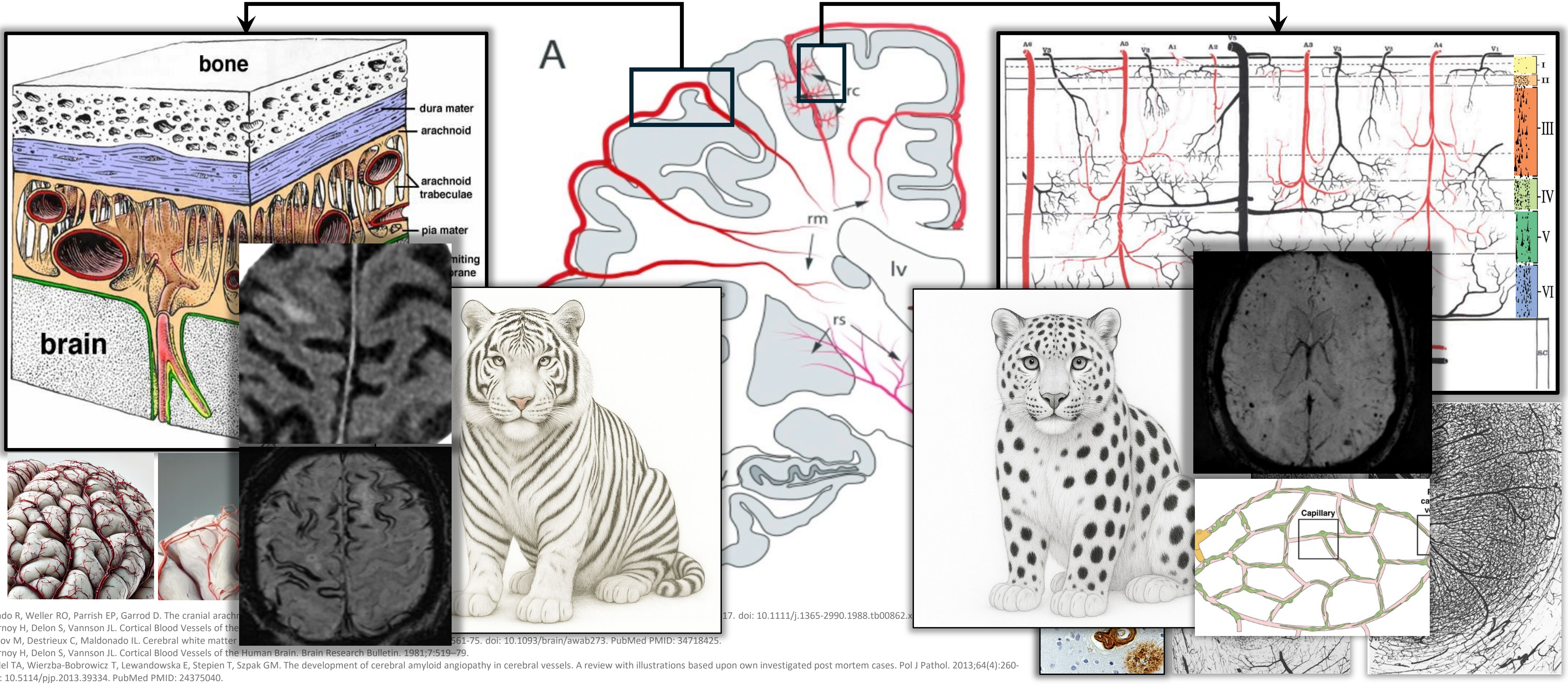
# Cortical
## Arterioles, Capillaries, Venules





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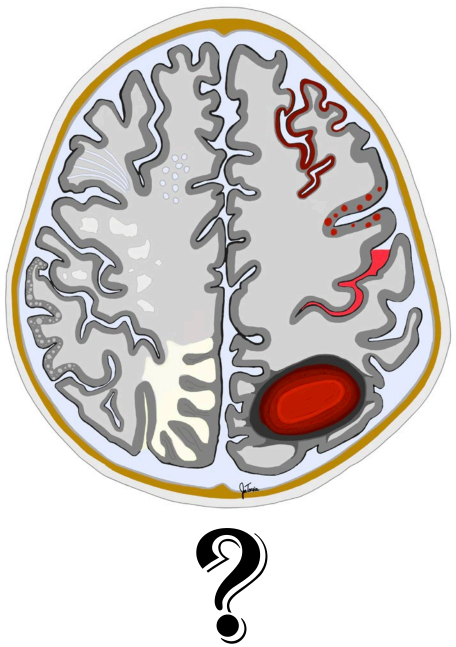
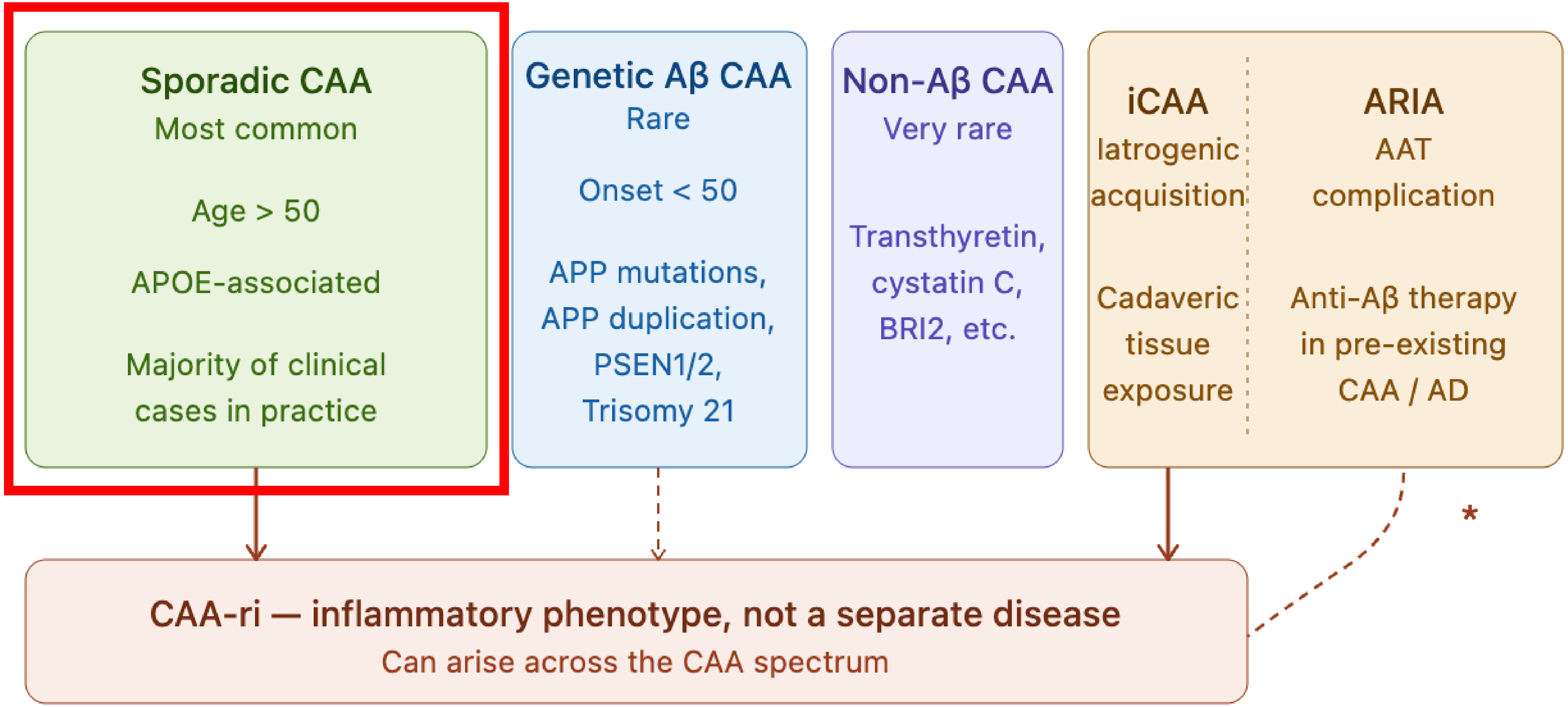
# 5 Key Points

1. **Poses Significant Diagnostic Challenges**: Often not considered in DDx for years. **Misdiagnosis** (e.g. TFNE/Amyloid Spell for TIA or seizures) occurs often, leading to unnecessary or harmful diagnostics & interventions. Additionally, due to substantial **mimic** burden, overdiagnosis occurs.
2. **Major Cause of Symptomatic Brain Hemorrhage (Stroke) in Older Adults**: It is the leading etiology of lobar **ICH** and convexal **SAH** in those over 60, with the **highest recurrence rate** among hemorrhagic causes. *Atraumatic **SDH** may also be associated with CAA.*
3. **Contributes to Cognitive Impairment & Dementia**: CAA independently drives **vascular cognitive impairment** via chronic pathological changes, sudden strategic impairments from stroke. Additionally, CAA frequently coexists with **Alzheimer's** pathology.
4. **Common and Underrecognized:** CAA is highly **prevalent** with aging — not a rare or exotic diagnosis. True prevalence is difficult to quantify, with clues coming from specific populations, and depends heavily on definition *(Pathology vs. MRI vs. ICD-10 codes)*.
5. **Prognosis & Risk is Spectrum-Based**: Both hemorrhagic and cognitive risks are **heterogeneous** and patient-specific — this is not a "one-size-fits-all" disease. CAA also increases the **risk of ischemic stroke**, which in many cases can outweigh hemorrhagic risk. Discussions with patients hinge upon this critical, **individualized approach** to diagnostic workup and risk management plans.

# Encountering CAA In Clinical Practice





# How do we make a **diagnosis** of CAA?

**Definitive Diagnosis: Brain biopsy** = seeing the disease under a microscope

**OR**

## Probability (Best Estimate) Diagnosis Based on Criteria (non-invasive):

In the absence of a biopsy, the diagnosis of CAA remains probabilistic. We rely on clinical criteria, imaging patterns, supporting data from labs or history, and careful exclusion of mimics.

### Clinical Presentation

- Cognitive Impairment or Dementia

and / or

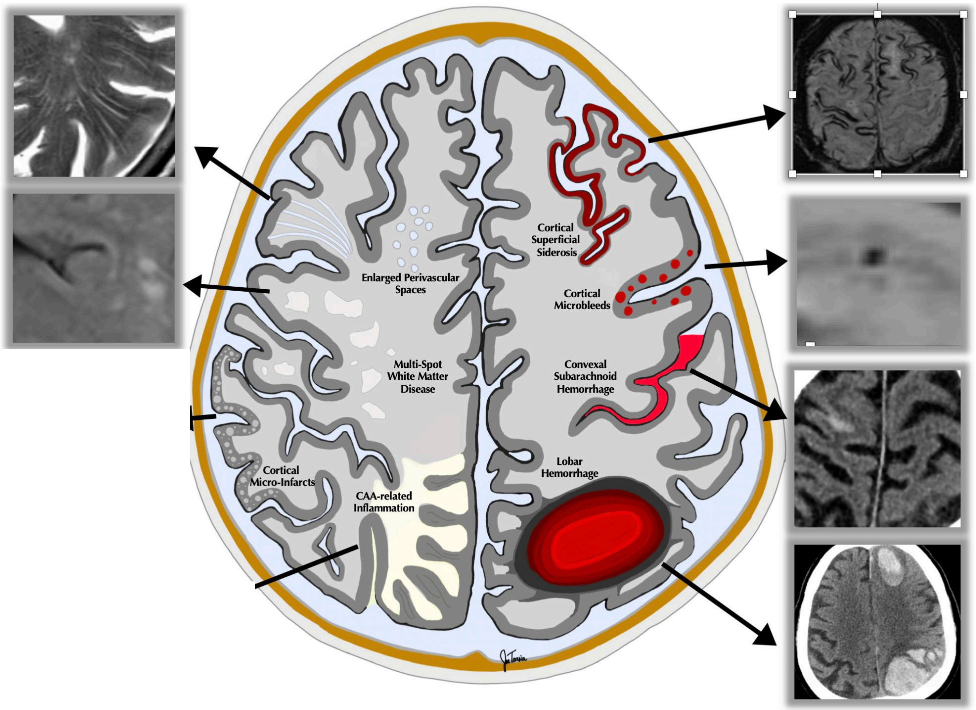
- Spontaneous Lobar ICH or Convexal SAH

and / or

- Transient Focal Neurological Episodes (TFNE)
  Common cause of ED visits as mimic for TIA in elderly

+

### Brain Imaging




+/-

In some cases, supplementary evidence from other sources can provide greater clarity regarding the presence of CAA:

- Medical history
- Further blood tests for amyloid disease (**blood biomarkers**)
- Cerebrospinal fluid (**CSF**) analysis for amyloid biomarkers
- Additional imaging techniques capable of detecting amyloid disease (**Amyloid PET**)

# Boston Criteria v2.0

## Diagnostic Criteria

| ☐ Probable CAA with Supporting Pathology | ☐ Probable CAA | ☐ Possible CAA |
|---|---|---|
| **Clinically presenting with**<br>☐ spontaneous lobar intracerebral hemorrhage<br>☐ transient focal neurological episodes<br>☐ cognitive impairment or dementia<br><br>**Pathological tissue (evacuated hematoma or cortical biopsy)** demonstrating:<br>• Some degree of CAA in specimen<br>• Absence of other diagnostic lesion | **Age ≥ 50**<br><br>**Clinically presenting with**<br>☐ spontaneous lobar intracerebral hemorrhage (ICH)<br>☐ convexity subarachnoid hemorrhage<br>☐ transient focal neurological episodes<br>☐ cognitive impairment or dementia<br><br>**≥ 2 of the following strictly lobar/superficial hemorrhagic lesions on T2*-GRE/SWI:**<br>☐ lobar ICH (acute, subacute, chronic)<br>☐ lobar cerebral microbleeds<br>☐ foci of cortical superficial siderosis<br>☐ foci of convexal subarachnoid hemorrhage<br><br>**OR**<br><br>**One lobar hemorrhagic lesion:**<br>☐ lobar ICH (acute, subacute, chronic)<br>☐ lobar cerebral microbleeds<br>☐ foci of cortical superficial siderosis<br>☐ foci of convexal subarachnoid hemorrhage<br>**Plus one white matter feature:**<br>☐ severe perivascular spaces in the centrum semiovale<br>☐ white matter hyperintensities in a multispot pattern<br><br>• *Absence of any deep hemorrhagic lesions (ie, intracerebral hemorrhage or cerebral microbleeds) on T2*-weighted MRI*<br>• *Absence of other cause of hemorrhagic lesions*<br>• *Hemorrhagic lesion in cerebellum not counted as either lobar or deep hemorrhagic lesion* | **Age ≥ 50**<br><br>**Clinically presenting with**<br>☐ spontaneous lobar intracerebral hemorrhage<br>☐ convexity subarachnoid hemorrhage<br>☐ transient focal neurological episodes<br>☐ cognitive impairment or dementia<br><br>**Only 1 of the following strictly lobar/superficial hemorrhagic lesion on T2*-GRE/SWI:**<br>☐ lobar ICH (acute, subacute, chronic)<br>☐ lobar cerebral microbleeds<br>☐ foci of cortical superficial siderosis<br>☐ foci of convexal subarachnoid hemorrhage<br><br>**OR**<br><br>**Only one white matter feature:**<br>☐ severe perivascular spaces in the centrum semiovale<br>☐ white matter hyperintensities in a multispot pattern<br><br>• *Absence of any deep hemorrhagic lesions (ie, intracerebral hemorrhage or cerebral microbleeds) on T2*-weighted MRI*<br>• *Absence of other cause of hemorrhagic lesions*<br>• *Hemorrhagic lesion in cerebellum not counted as either lobar or deep hemorrhagic lesion* |


1. Charidimou A, Boulouis G, Frosch MP, Baron J-C, Pasi M, Albucher JF, et al. The Boston criteria version 2.0 for cerebral amyloid angiopathy: a multicentre, retrospective, MRI–neuropathology diagnostic accuracy study. The Lancet Neurology. 2022;21(8):714-25. doi: 10.1016/s1474-4422(22)00208-3.
2. Greenberg SM, van Veluw SJ. Cerebral Amyloid Angiopathy. Stroke. 2024. Epub 20240125. doi: 10.1161/STROKEAHA.124.044293. PubMed PMID: 38269538.
3. Zanon Zotin MC, Makkinejad N, Schneider JA, Arfanakis K, Charidimou A, Greenberg SM, et al. Sensitivity and Specificity of the Boston Criteria Version 2.0 for the Diagnosis of Cerebral Amyloid Angiopathy in a Community-Based Sample. Neurology. 2024;102(1). doi: 10.1212/wnl.0000000000207940.
4. Switzer AR, Charidimou A, McCarter S, Vemuri P, Nguyen AT, Przybelski SA, et al. Boston Criteria v2.0 for Cerebral Amyloid Angiopathy Without Hemorrhage. Neurology. 2024;102(10). doi: 10.1212/wnl.0000000000209386.
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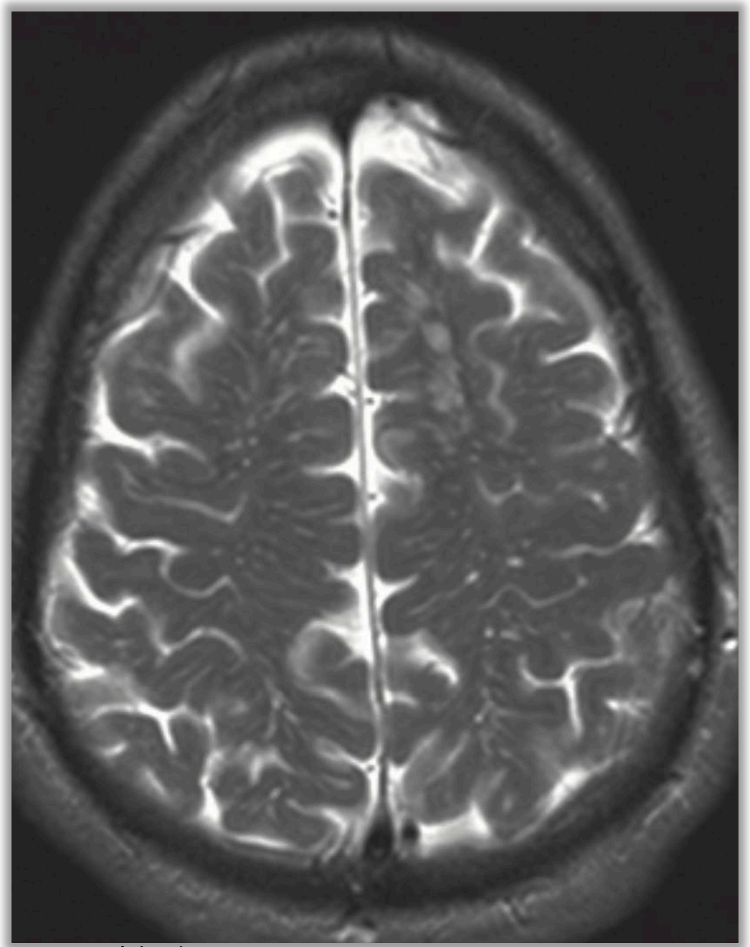
# Boston Criteria v2.0

## Non-Hemorrhagic Features

### Severe EPVS-CSO*

> 20 visible PVS in the centrum semi-ovale of 1 hemisphere



* higher impact on sensitivity

**Probable CAA**

**Age ≥ 50**

**Clinically presenting with**
- spontaneous lobar intracerebral hemorrhage (ICH)
- convexity subarachnoid hemorrhage
- transient focal neurological episodes
- cognitive impairment or dementia

**≥ 2 of the following strictly lobar/superficial hemorrhagic lesions on T2*-GRE/SWI:**
- lobar ICH (acute, subacute, chronic)
- lobar cerebral microbleeds
- foci of cortical superficial siderosis
- foci of convexal subarachnoid hemorrhage

**OR**

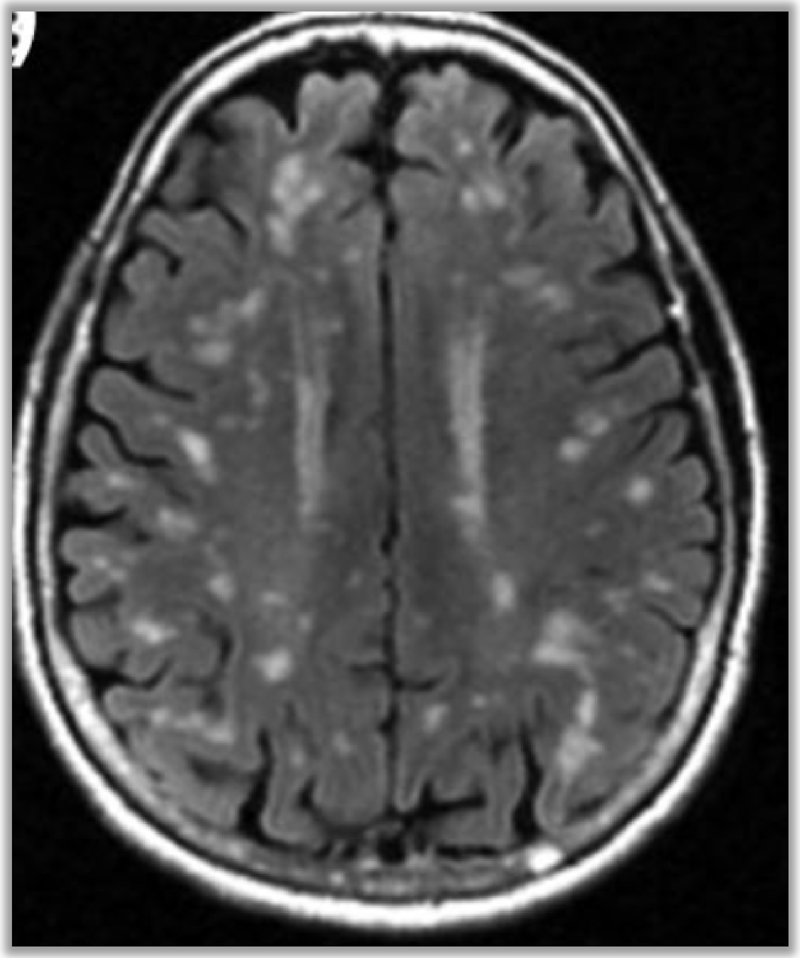
**One lobar hemorrhagic lesion:**
- lobar ICH (acute, subacute, chronic)
- lobar cerebral microbleeds
- foci of cortical superficial siderosis
- foci of convexal subarachnoid hemorrhage

**Plus one white matter feature:**
- severe perivascular spaces in the centrum semiovale
- white matter hyperintensities in a multispot pattern

- *Absence of any deep hemorrhagic lesions (ie, intracerebral hemorrhage or cerebral microbleeds) on T2*-weighted MRI*
- *Absence of other cause of hemorrhagic lesions*
- *Hemorrhagic lesion in cerebellum not counted as either lobar or deep hemorrhagic lesion*

### WMH-Multi-Spot Pattern

>10 small (3-25 mm), juxtacortical/subcortical, round/ovoid hyperintense lesions on MRI FLAIR




1. Greenberg SM, van Veluw SJ. Cerebral Amyloid Angiopathy. Stroke. 2024. Epub 20240125. doi: 10.1161/STROKEAHA.124.044293. PubMed PMID: 38269538.
2. Charidimou A, Boulouis G, Frosch MP, Baron J-C, Pasi M, Albucher JF, et al. The Boston criteria version 2.0 for cerebral amyloid angiopathy: a multicentre, retrospective, MRI–neuropathology diagnostic accuracy study. The Lancet Neurology. 2022;21(8):714-25. doi: 10.1016/s1474-4422(22)00208-3.
3. Cordonnier C, Klijn C, Smith EE, Al-Shahi Salman R, Chwalisz BK, van Etten E, et al. Diagnosis and management of cerebral amyloid angiopathy: a scientific statement from the International CAA Association and the World Stroke Organization. Int J Stroke. 2025:17474930251365861. doi: 10.1177/17474930251365861. PubMed PMID: 40721902.

# Boston Criteria v2.0

## Context is Critical

### Symptomatic Disease

**Sensitivity = 74.5%** [65.4–82.7]

**Specificity = 95.0%** [83.1–99.4]

- AUC = 0.798 [0.741–0.854]

**Sp = 92.9%** in lobar ICH presentations

**Sp = 96.2%** in other presentations

In **asymptomatic** patients:

Community Dwelling (ROSMAP Cohort) n= 134] [3]

**Sensitivity = 38.8%** (+12% c/w 1.5)

**Specificity = 83.5%** (-5% c/w 1.5)

In **elderly asymptomatic & cognitive**

Mayo Clinic Study of Aging and ADRC (n=54)[4]

**Sensitivity = 29 %**

**Specificity = 65 %**

**Probable CAA**

**Age ≥ 50**

**Clinically presenting with**
- ☐ spontaneous lobar intracerebral hemorrhage (ICH)
- ☐ convexity subarachnoid hemorrhage
- ☐ transient focal neurological episodes
- ☐ cognitive impairment or dementia

**≥ 2 of the following strictly lobar/superficial hemorrhagic lesions on T2*-GRE/SWI:**
- ☐ lobar ICH (acute, subacute, chronic)
- ☐ lobar cerebral microbleeds
- ☐ foci of cortical superficial siderosis
- ☐ foci of convexal subarachnoid hemorrhage

**OR**

**One lobar hemorrhagic lesion:**
- ☐ lobar ICH (acute, subacute, chronic)
- ☐ lobar cerebral microbleeds
- ☐ foci of cortical superficial siderosis
- ☐ foci of convexal subarachnoid hemorrhage

**Plus one white matter feature:**
- ☐ severe perivascular spaces in the centrum semiovale
- ☐ white matter hyperintensities in a multispot pattern

- *Absence of any deep hemorrhagic lesions (ie, intracerebral hemorrhage or cerebral microbleeds) on T2*-weighted MRI*
- *Absence of other cause of hemorrhagic lesions*
- *Hemorrhagic lesion in cerebellum not counted as either lobar or deep hemorrhagic lesion*

| | Probable vs non-probable CAA* | Probable and possible vs no CAA |
|---|---|---|
| **All patients with brain autopsy (n=150; Boston criteria v1.5)** | | |
| Sensitivity | 64·5% (54·9–73·4) | 75·5% (66·3–83·2) |
| Specificity | 95·0% (83·1–99·4) | 87·5% (73·2–95·8) |
| AUC | 0·798 (0·741–0·854) | 0·815 (0·749–0·881) |
| PPV | 97·3% (90·5–99·7) | 94·3% (87·2–98·1) |
| NPV | 49·4% (37·8–61) | 56·5% (43·3–69) |
| **All patients with brain autopsy (n=150; Boston criteria v2.0)** | | |
| Sensitivity | 74·5% (65·4–82·4) | 88·2% (80·6–93·6) |
| Specificity | 95·0% (83·1–99·4) | 70·0% (53·5–83·4) |
| AUC | 0·848 (0·794–0·901) | 0·791 (0·713–0·869) |
| PPV | 97·6% (91·7–99·7) | 89% (81·6–94·2) |
| NPV | 57·6% (44·8–69·7) | 68·3% (51·9–81·9) |
| **Patients with brain autopsy who presented with intracerebral haemorrhage (n=75; Boston criteria v2.0)** | | |
| Sensitivity | 90·2% (79·8–96·3) | 91·8% (81·9–97·3) |
| Specificity | 92·9% (66·1–99·8) | 71·4% (41·9–91·6) |
| AUC | 0·915 (0·836–0·995) | 0·816 (0·689–0·944) |
| PPV | 98·2% (90·4–100) | 93·3% (83·8–98·2) |
| NPV | 68·4% (43·4–87·4) | 66·7% (38·4–88·2) |
| **Patients with brain autopsy and presentations other than intracerebral haemorrhage (n=75; Boston criteria v2.0)** | | |
| Sensitivity | 55·1% (40·2–69·3) | 83·7% (70·3–92·7) |
| Specificity | 96·2% (80·4–99·9) | 69·2% (48·2–85·7) |
| AUC | 0·756 (0·676–0·836) | 0·765 (0·66–0·869) |
| PPV | 96·4% (81·7–99·9) | 83·7% (70·3–92·7) |
| NPV | 53·2% (38·1–67·9) | 69·2% (48·2–85·7) |
| **Patients with brain autopsy and T2*-GRE (n=127; Boston criteria v2.0)** | | |
| Sensitivity | 72·6% (62·5–81·3) | 87·4% (79–93·3) |
| Specificity | 93·8% (79·2–99·2) | 68·8% (50–83·9) |
| AUC | 0·832 (0·77–0·894) | 0·781 (0·692–0·869) |
| PPV | 97·2% (90·1–99·7) | 89·2% (81·1–94·7) |
| NPV | 53·6% (39·7–67) | 64·7% (46·5–80·3) |
| **Patients with brain autopsy and SWI (n=23; Boston criteria v2.0)** | | |
| Sensitivity | 86·7% (59·5–98·3) | 93·3% (68·1–99·8) |
| Specificity | 100% (63·1–100) | 75% (34·9–96·8) |
| AUC | 0·933 (0·844–1) | 0·842 (0·668–1) |
| PPV | 100% (75·3–100) | 87·5% (61·7–98·4) |
| NPV | 80% (44·4–97·5) | 85·7% (42·1–99·6) |

Prespecified subgroup analyses of the Boston criteria (v.15 and v2.0) within the whole cohort. CAA=cerebral amyloid angiopathy. GRE=gradient-recalled echo. SWI=susceptibility-weighted imaging. AUC=area under the receiver operating characteristic curve. PPV=positive predictive value. NPV=negative predictive value. *Non-probable CAA refers to patients not fulfilling criteria for probable CAA.

**Table 4: Diagnostic performance of Boston criteria v2.0 in prespecified subsets of all individuals with available autopsy**


1. Charidimou A, Boulouis G, Frosch MP, Baron J-C, Pasi M, Albucher JF, et al. The Boston criteria version 2.0 for cerebral amyloid angiopathy: a multicentre, retrospective, MRI–neuropathology diagnostic accuracy study. The Lancet Neurology. 2022;21(8):714-25. doi: 10.1016/s1474-4422(22)00208-3.
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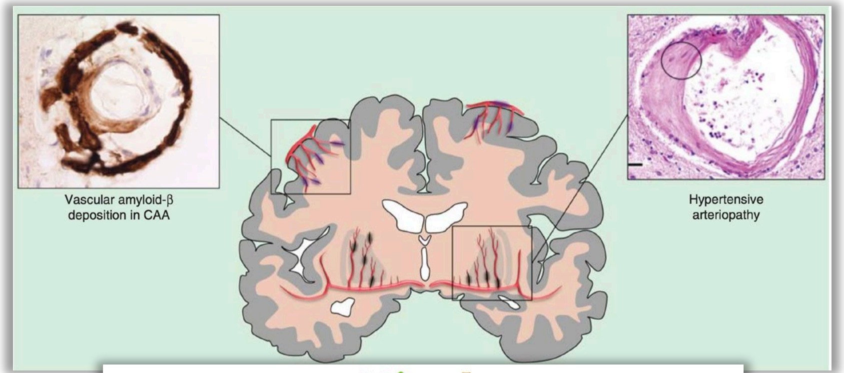
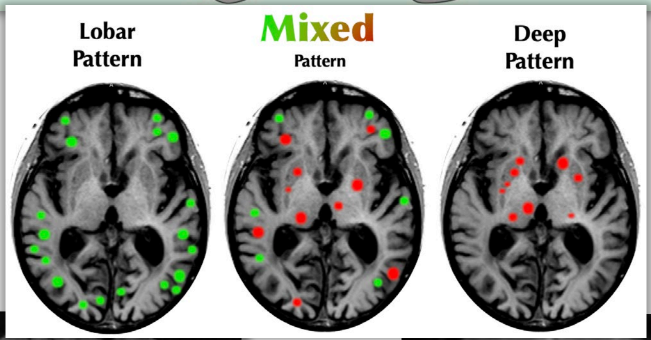
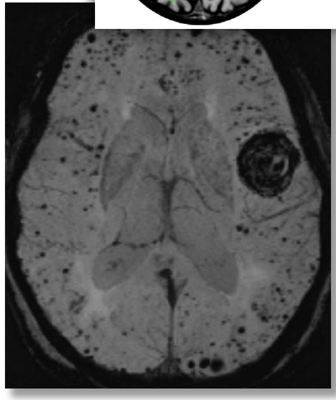
6. Sellimi A, Schwartze J, Humphries F, Panteleienko L, Mallon D, Banerjee G, et al. Contemporary perspectives in cerebral amyloid angiopathy. Expert Rev Neurother. 2025:1–24. doi: 10.1080/14737175.2025.2526113.
7. Cordonnier C, Klijn C, Smith EE, Al-Shahi Salman R, Chwalisz BK, van Etten E, et al. Diagnosis and management of cerebral amyloid angiopathy: a scientific statement from the International CAA Association and the World Stroke Organization. Int J Stroke. 2025:17474930251365861. Epub 20250728. doi: 10.1177/17474930251365861. PubMed PMID: 40721902.

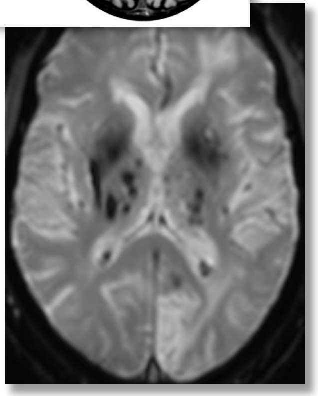
# Boston Criteria v2.0

## Mixed Pattern Disease





### Probable CAA

**Age ≥ 50**

**Clinically presenting with**

- ☐ spontaneous lobar intracerebral hemorrhage (ICH)
- ☐ convexity subarachnoid hemorrhage
- ☐ transient focal neurological episodes
- ☐ cognitive impairment or dementia

**≥ 2 of the following strictly lobar/superficial hemorrhagic lesions on T2*-GRE/SWI:**

- ☐ lobar ICH (acute, subacute, chronic)
- ☐ lobar cerebral microbleeds
- ☐ foci of cortical superficial siderosis
- ☐ foci of convexal subarachnoid hemorrhage

**OR**

**One lobar hemorrhagic lesion:**

- ☐ lobar ICH (acute, subacute, chronic)
- ☐ lobar cerebral microbleeds
- ☐ foci of cortical superficial siderosis
- ☐ foci of convexal subarachnoid hemorrhage

**Plus one white matter feature:**

- ☐ severe perivascular spaces in the centrum semiovale
- ☐ white matter hyperintensities in a multispot pattern

- *Absence of any deep hemorrhagic lesions (ie, intracerebral hemorrhage or cerebral microbleeds) on T2*-weighted MRI*
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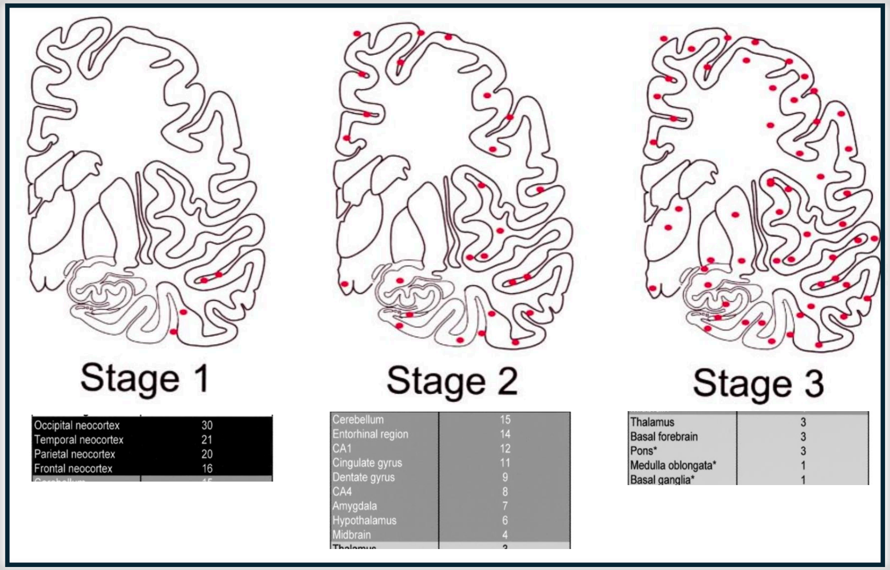
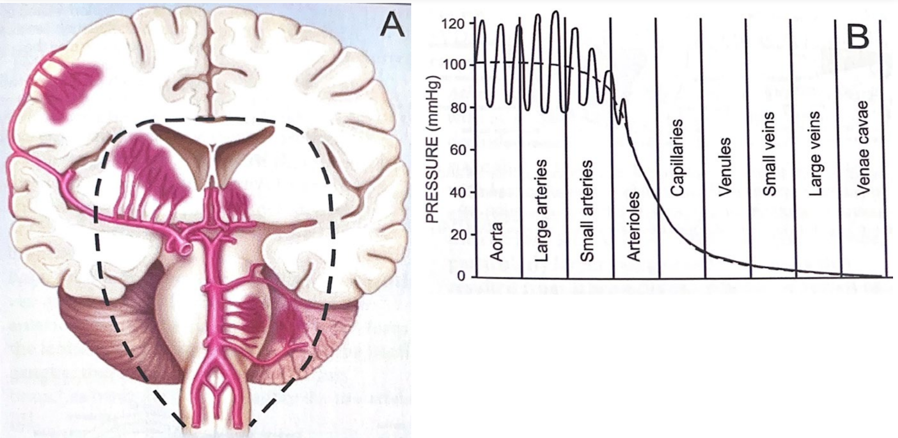


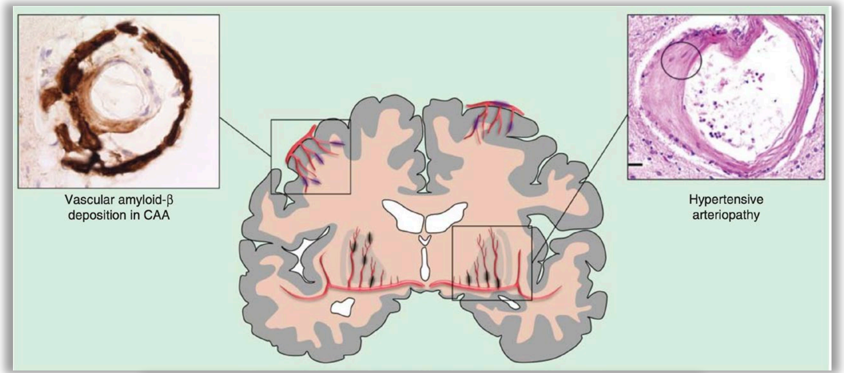



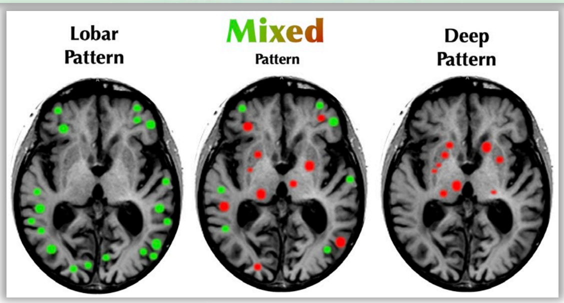


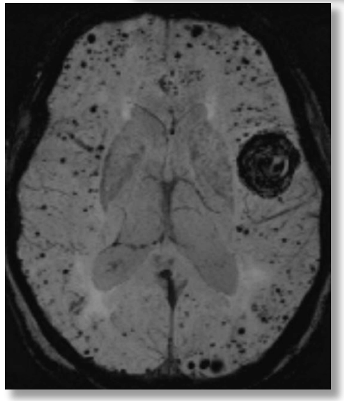
1. Thal DR, Ghebremedhin E, Orantes M, Wiestler OD. Vascular Pathology in Alzheimer Disease: Correlation of Cerebral Amyloid Angiopathy and Arteriosclerosis/Lipohyalinosis with Cognitive Decline. *Journal of Neuropathology & Experimental Neurology*. 2003;62(12):1287–1301. doi:10.1093/jnen/62.12.1287
2. 1. Wilson D, Charidimou A, Werring DJ. Advances in understanding spontaneous intracerebral hemorrhage: insights from neuroimaging. *Expert Rev Neurother*. Jun 2014;14(6):661–78. doi:10.1586/14737175.2014.918506

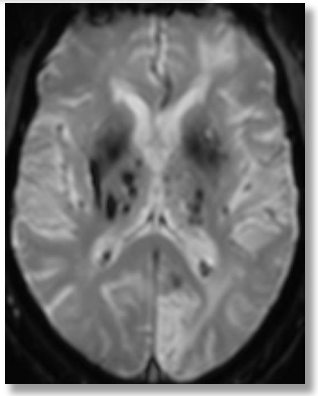
# Boston Criteria v2.0

## Mixed Pattern Disease





### Probable CAA

**Age ≥ 50**

**Clinically presenting with**
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**OR**

**One lobar hemorrhagic lesion:**
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- *Absence of other cause of hemorrhagic lesions*
- *Hemorrhagic lesion in cerebellum not counted as either lobar or deep hemorrhagic lesion*

## Favors CAA

- Presence of **Cortical Superficial Siderosis**
- Ratio Lobar:Deep **≥ 5:1**


1. Thal DR, Ghebremedhin E, Orantes M, Wiestler OD. Vascular Pathology in Alzheimer Disease: Correlation of Cerebral Amyloid Angiopathy and Arteriosclerosis/Lipohyalinosis with Cognitive Decline. *Journal of Neuropathology & Experimental Neurology*. 2003;62(12):1287–1301. doi:10.1093/jnen/62.12.1287
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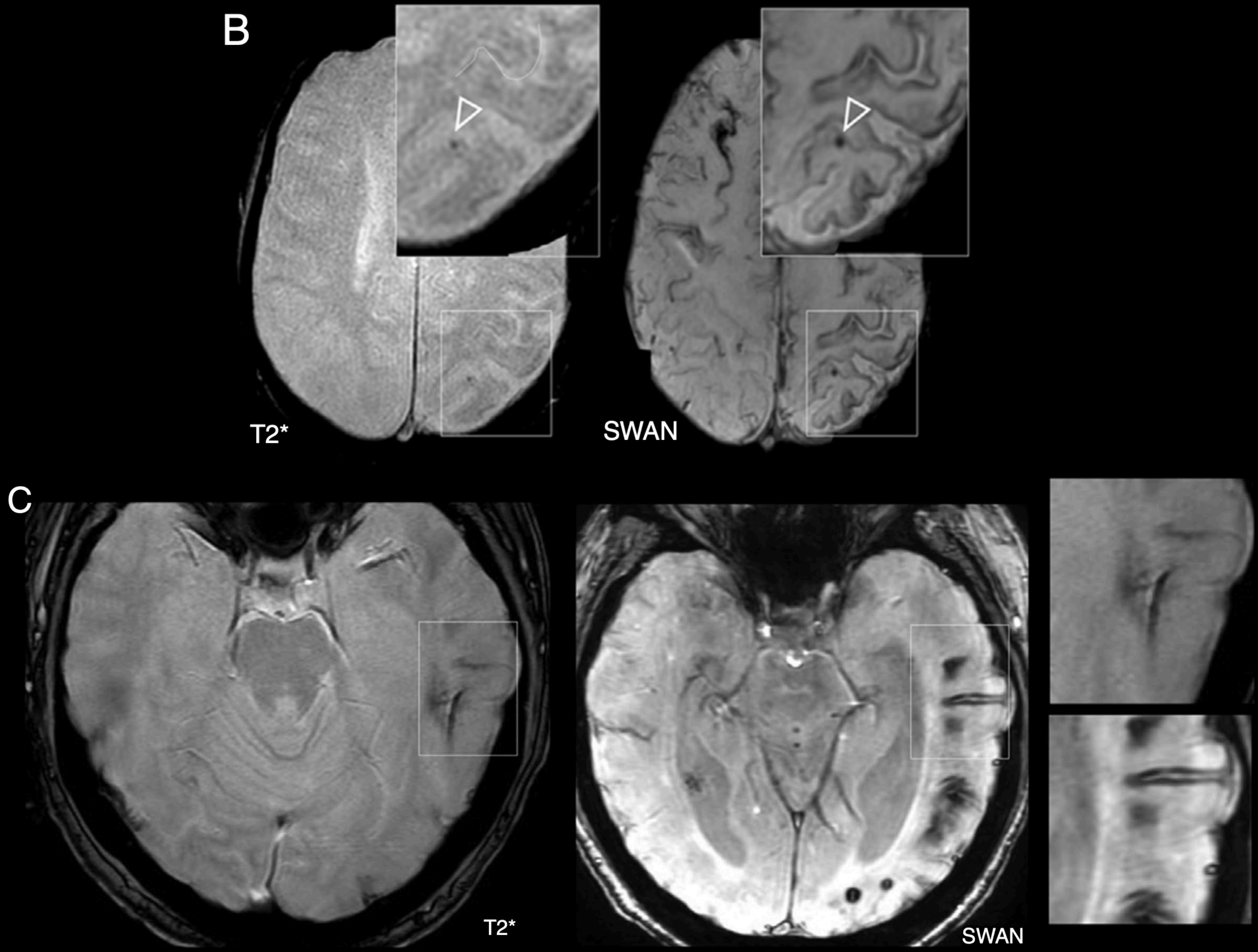
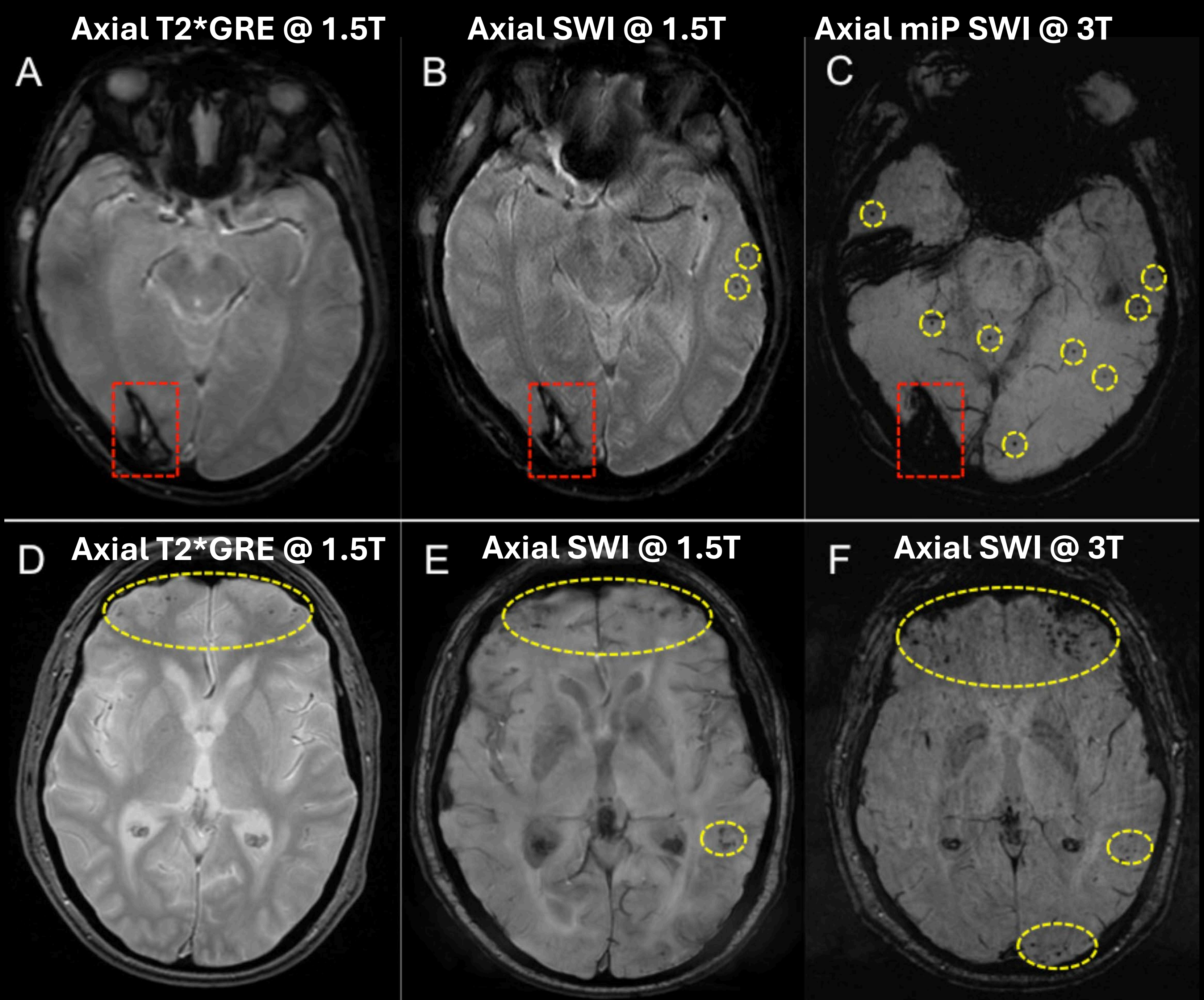
4. Das AS, Gokcal E, Biffi A, Regenhardt RW, Pasi M, Abramovitz Fouks A, et al. Mechanistic Implications of Cortical Superficial Siderosis in Patients With Mixed Location Intracerebral Hemorrhage and Cerebral Microbleeds. Neurology. 2023;101(6):e636–e44. Epub 20230608. doi: 10.1212/WNL.0000000000207476. PubMed PMID: 37290968;

# SWI vs. GRE/T2*/heme



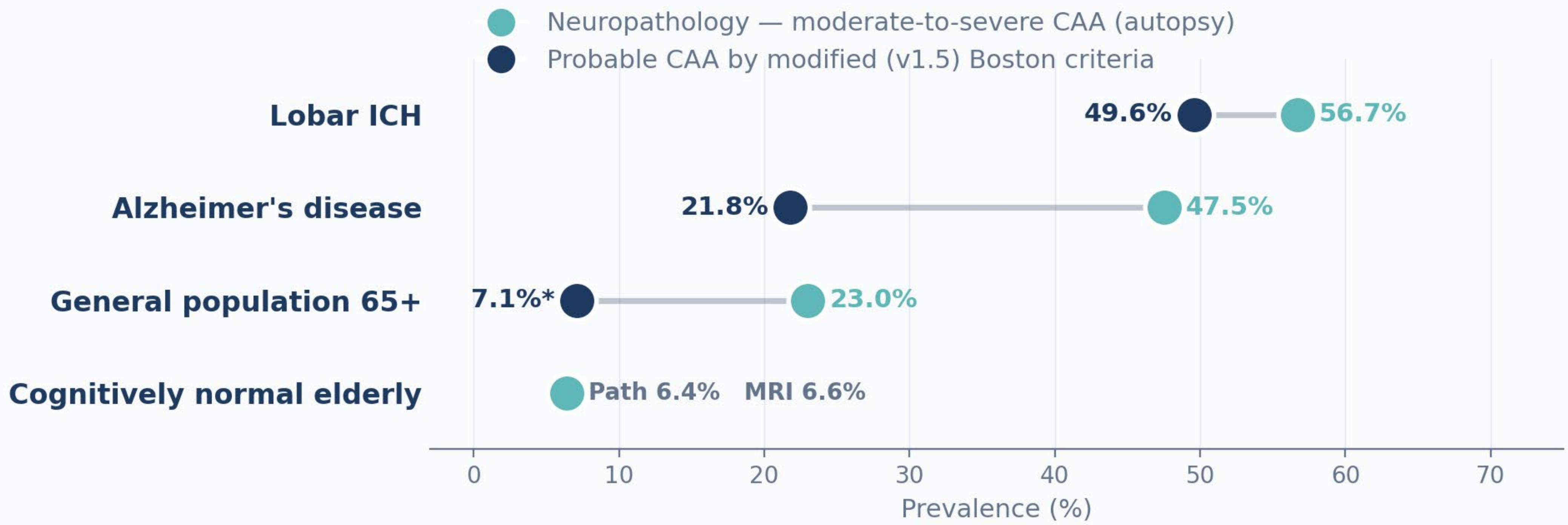

1. Boulouis G. Revue neurologique. 2017;173(9):554–561.




1. Puy L, Pasi M, Rodrigues M, van Veluw SJ, Tsivgoulis G, Shoamanesh A, et al. Cerebral microbleeds: from depiction to interpretation. Journal of Neurology, Neurosurgery & Psychiatry. 2021;92(6):598-607. doi: 10.1136/jnnp-2020-323951.

# Prevalence By Detection Tool

*MRI reveals only the tip of the iceberg*




**Key insight:** Across every population, pathology finds substantially more CAA than MRI can detect — because MRI markers capture only hemorrhagic, late-stage disease. Including mild CAA (not just moderate-to-severe) would widen the gap further.

**Strictly lobar cerebral microbleeds on MRI (used as proxy for CAA in non-ICH populations — Boston criteria not validated outside ICH cohorts in this review)*

*Jäkel L, De Kort AM, Klijn CJM, Schreuder FHBM, Verbeek MM. Prevalence of cerebral amyloid angiopathy: A systematic review and meta-analysis. Alzheimer's & Dementia 2022;18(1):10–28. doi:10.1002/alz.12366 | 170 studies, 73,000 subjects*

# Edinburgh Diagnostic CT Criteria

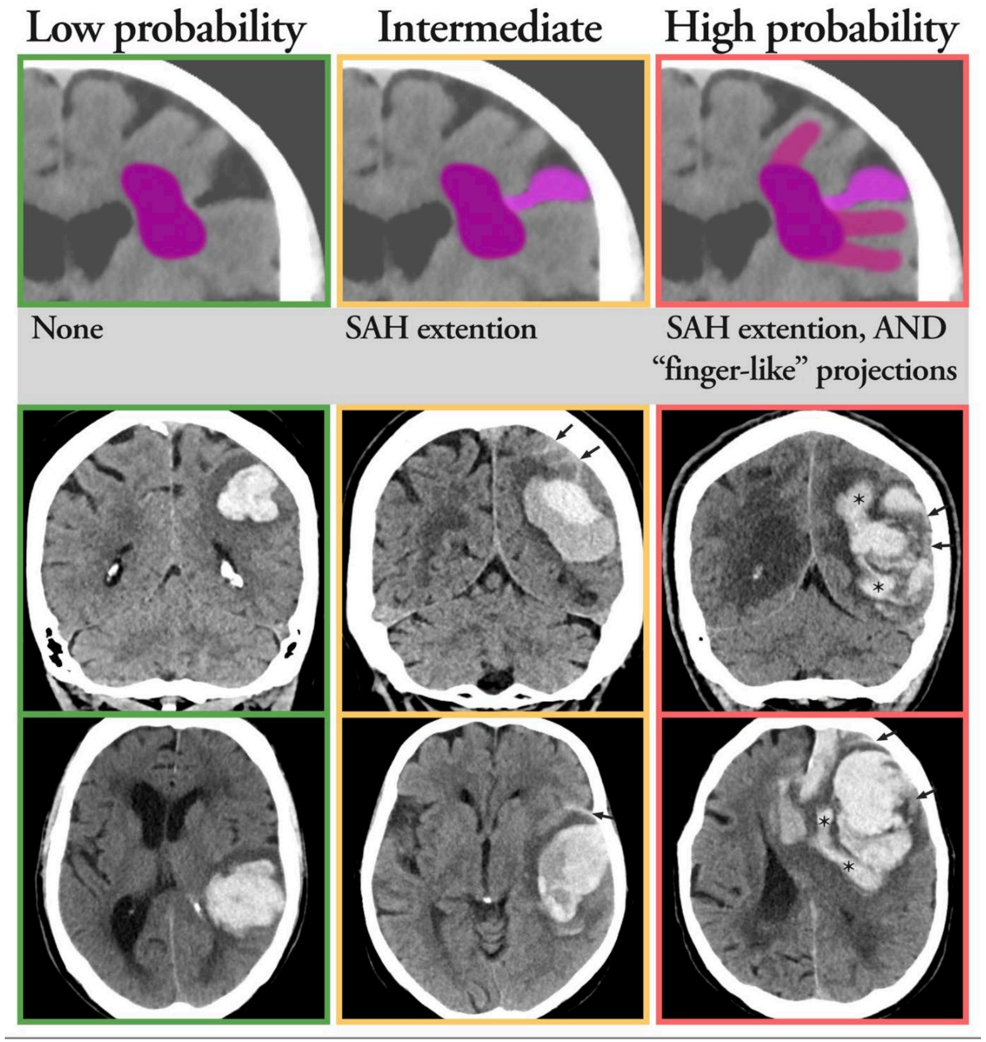
- ☐ SAH associated with the IPH
- ☐ Finger-like projections of the IPH
- ☐ Possession of ≥1 APOE e4 alleles

**2 of 3 present** = high-probability → **(“rule-in”)** diagnosis (Sp ~100%)

**0 of 3 present** = low-probability → **(“rule-out”)** diagnosis (Se ~100%)

**Simplified Criteria** = omit APOE genotype

Se 81%, Sp 87%





1. Cordonnier C, Klijn C, Smith EE, Al-Shahi Salman R, Chwalisz BK, van Etten E, et al. Diagnosis and management of cerebral amyloid angiopathy: a scientific statement from the International CAA Association and the World Stroke Organization. Int J Stroke. 2025:17474930251365861. Epub 20250728. doi: 10.1177/17474930251365861. PubMed PMID: 40721902.
2. Sellimi A, Schwartze J, Humphries F, Panteleienko L, Mallon D, Banerjee G, et al. Contemporary perspectives in cerebral amyloid angiopathy. Expert Rev Neurother. 2025:1–24. doi: 10.1080/14737175.2025.2526113.
3. Rodrigues MA, Seiffge D, Samarasekera N, Moullaali TJ, Wardlaw JM, Schreiber S, et al. Association between the Edinburgh CT and genetic diagnostic criteria for cerebral amyloid angiopathy-associated lobar intracerebral haemorrhage and recurrent intracerebral haemorrhage: an individual patient data meta-analysis. The Lancet Neurology. 2025;24(10):828–39. doi: 10.1016/s1474-4422(25)00285-6.
4. Smith EE. Refining risk stratification in cerebral amyloid angiopathy: the role of the Edinburgh criteria. Lancet Neurol. 2025;24(10):803–5. doi: 10.1016/S1474-4422(25)00321-7. PubMed PMID: 40975088.
5. Sembill JA, Knott M, Xu M, Roeder SS, Hagen M, Sprugel MI, et al. Simplified Edinburgh CT Criteria for Identification of Lobar Intracerebral Hemorrhage Associated With Cerebral Amyloid Angiopathy. Neurology. 2022;98(20):e1997–e2004. Epub 20220321. doi: 10.1212/WNL.0000000000200261. PubMed PMID: 35314501.
6. Van Etten E, Kaushik K, van Zwet E, Voigt S, van Walderveen M, van Buchem M, et al. Sensitivity of the Edinburgh Criteria for Lobar Intracerebral Hemorrhage in Hereditary Cerebral Amyloid Angiopathy. Stroke. 2020;51:3608–12. doi: 10.1161/STROKEAHA.120.031264Downloaded
7. Rodrigues MA, Samarasekera N, Lerpiniere C, Humphreys C, McCarron MO, White PM, et al. The Edinburgh CT and genetic diagnostic criteria for lobar intracerebral haemorrhage associated with cerebral amyloid angiopathy: model development and diagnostic test accuracy study. Lancet Neurol. 2018;17(3):232–40. Epub 20180110. doi: 10.1016/S1474-4422(18)30006-1. PubMed PMID: 29331631;.
8. Hillal A, Apostolaki-Hansson T, Ramgren B, Hansen B, Norrving B, Wasselius J, et al. The probability of cerebral amyloid angiopathy according to the Simplified Edinburgh CT criteria in a large, unselected lobar intracerebral hemorrhage population. Neuroradiology. 2025. Epub 20250212. doi: 10.1007/s00234-025-03555-8. PubMed PMID: 39937267.

# Transient Focal Neurological Episodes (TFNEs)

## aka "Amyloid Spells"

- **Recurrent** (days → weeks), **stereotyped** episodes with focal neurological symptoms, typically **< 30 min**
- Symptomatology (often mixed, often **evolve/spread**):
  - *"Positive" → Aura-like spreading/migratory paresthesias[2], visual (distorted vision, flickering or flashing lights, zig-zags), limb jerking*
  - *"Negative" → TIA-like loss of function (unilateral motor/sensory, vision loss, aphasia, dysarthria)*
- Felt to be a result of cortical spreading depolarizations (CSDs) within a sulcus affected by an **acute cSAH or chronic cSS lesions**
- Avoid unnecessary anti-thrombotics! (i.e. TIA misdiagnosis)– *adjacent (esp. SAH) cortical **DWI+ lesions** may be present !*
- High risk of lobar ICH and increased mortality[3] *(esp. if motor Sx or antiplatelet prescribed)*

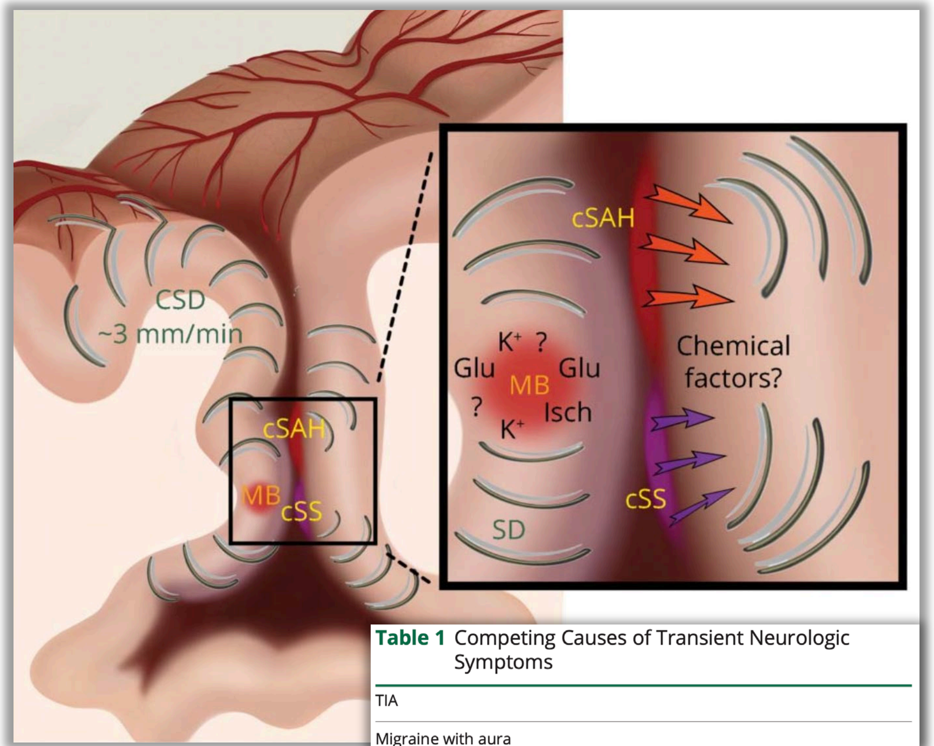
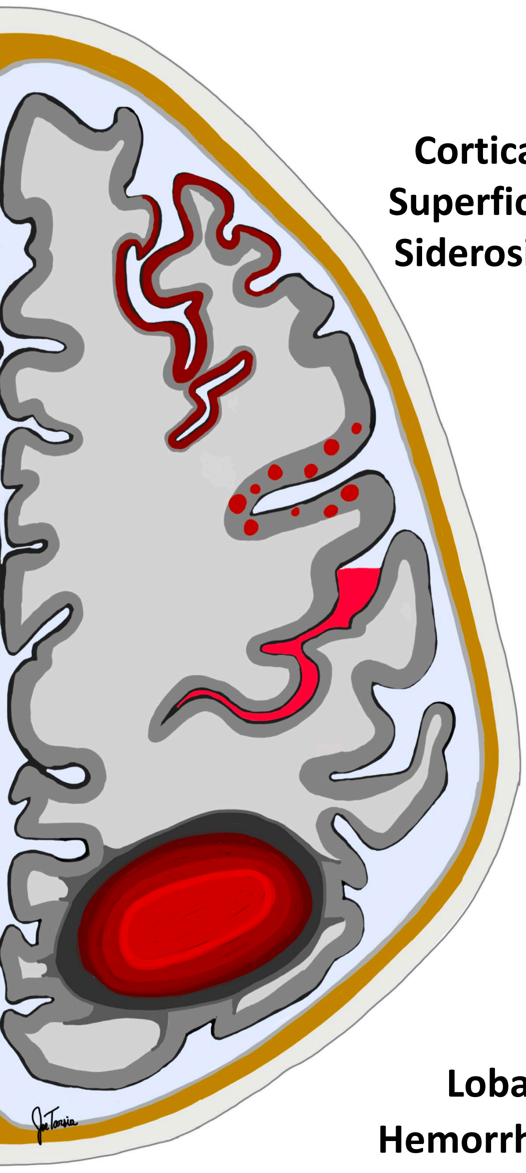



**Table 1** Competing Causes of Transient Neurologic Symptoms

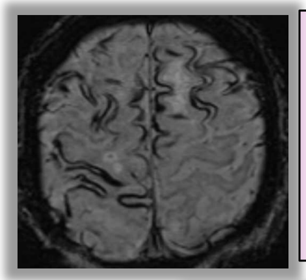
| |
|---|
| TIA |
| Migraine with aura |
| Focal seizure |
| Structural lesions (e.g., tumor, vascular malformation, subdural hematoma) |
| Metabolic abnormalities (e.g., hypoglycemia, hyponatremia) |
| Syncope or presyncope |
| Functional neurologic disorder |


1. Theodorou A, Chondrogianni M, Bakola E, et al. Cortical Superficial Siderosis and Transient Focal Neurological Episode Preceding Lobar Hemorrhage in Cerebral Amyloid Angiopathy. *Stroke*. Feb 2023;54(2):e48–e51. doi:10.1161/STROKEAHA.122.041395
2. Smith EE, Charidimou A, Ayata C, Werring DJ, Greenberg SM. Cerebral Amyloid Angiopathy-Related Transient Focal Neurologic Episodes. *Neurology*. Aug 3 2021;97(5):231–238. doi:10.1212/WNL.0000000000012234
3. Sanchez-Caro JM, de Lorenzo Martinez de Ubago I, de Celis Ruiz E, et al. Transient Focal Neurological Events in Cerebral Amyloid Angiopathy and the Long-term Risk of Intracerebral Hemorrhage and Death: A Systematic Review and Meta-analysis. *JAMA Neurol*. Jan 1 2022;79(1):38–47. doi:10.1001/jamaneurol.2021.3989
4. Khurram A, Kleinig T, Leyden J. Clinical Associations and Causes of Convexity Subarachnoid Hemorrhage. Stroke. 2014;45(4):1151–3. doi: 10.1161/strokeaha.113.004298.
5. Charidimou A, Peeters A, Fox Z, Gregoire SM, Vandermeeren Y, Laloux P, et al. Spectrum of transient focal neurological episodes in cerebral amyloid angiopathy: multicentre magnetic resonance imaging cohort study and meta-analysis. Stroke. 2012;43(9):2324–30. Epub 20120712. doi: 10.1161/STROKEAHA.112.657759. PubMed PMID: 22798323.

# Are You Sure it's a CAA lesion??



## Cortical Superficial Siderosis[7,8]

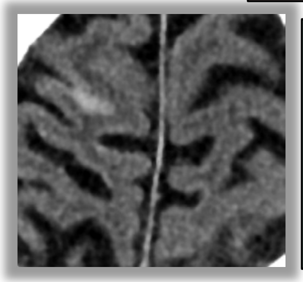


- Classical SS (Dural defects or tears → posterior fossa)
- Localized (trauma, AVM/AVF, tumor, craniospinal surgery)
- Cortical veins (deoxyHgb)
- Thrombosed vessels
- Hemorrhagic transformation of cortical infarcts
- Cortical laminar necrosis
- Prior lobar ICH (esp. if SAH or Intraventricular Ext)
- Other cSAH source
- Sturge Weber

## Cortical Lobar Microbleeds

- Post-Bypass (Valve, CABG, etc)
- Catheter-based structural heart interventions (LAAO, TAVR, MVR) > PCI
- Other SVD (HTN Arteriopathy, CADASIL, etc)
- Infective endocarditis
- Cavernomatosis
- Hemorrhagic metastases
- Atrial myxoma
- DAI/severe head trauma
- Neurocystercercosis
- AHLE
- Fat/Ca++/Air emboli
- Delayed post-hypoxic leukoencephalopathy
- Critical Illness (CICM)
- Radiation-induced vasculopathy
- HLH (Hemophagocytic Lymphohystiocytosis)
- Intravascular Lymphoma

## Convexal Subarachnoid Hemorrhage[5]



- Cortical vein thrombosis
- CVST
- Infective Endocarditis
- Arterial Stenosis
- PRES/RCVS

## Lobar Hemorrhage[1]

- HTN
- CVST
- Endocarditis
- RCVS
- Hemorrhagic PRES
- Hemorrhagic Transformation of Infarct
- CNS Angiitis
- Reperfusion/hyperperfusion
- Tumor/Mets
- AVM / Aneurysm / Dural AVF / Cavernoma
- Severe coagulopathy


1. Tartarin H, Morotti A, Van Etten ES, Hausman-Kedem M, Charidimou A, Jouvent E, et al. Uncommon Causes of Nontraumatic Intracerebral Hemorrhage. Stroke. 2024;55(5):1416-27
2. Khurram A, Kleinig T, Leyden J. Clinical Associations and Causes of Convexity Subarachnoid Hemorrhage. Stroke. 2014;45(4):1151-3.
3. Kumar S, Goddeau RP, Selim MH, Thomas A, Schlaug G, Alhazzani A, et al. Atraumatic convexal subarachnoid hemorrhage. Neurology. 2010;74(11):893-9.
4. Werring DJ, Banerjee G. Convexity subarachnoid haemorrhage. The Lancet. 2023;401(10372):193-4.
5. Zedde M, Grisendi I, Assenza F, Napoli M, Moratti C, Pavone C, et al. Spontaneous Non-Aneurysmal Convexity Subarachnoid Hemorrhage: A Scoping Review of Different Etiologies beyond Cerebral Amyloid Angiopathy. Journal of Clinical Medicine. 2024;13(15):4382. doi: 10.3390/jcm13154382.
6. Charidimou A, Perosa V, Frosch MP, Scherlek AA, Greenberg SM, van Veluw SJ. Neuropathological correlates of cortical superficial siderosis in cerebral amyloid angiopathy. Brain. 2020;143(11):3343-51. doi: 10.1093/brain/awaa266. PubMed PMID: 32935842;
7. Kumar N. Superficial Siderosis: A Clinical Review. Ann Neurol. 2021;89(6):1068-79. Epub 20210428. doi: 10.1002/ana.26083. PubMed PMID: 33860558.
8. Charidimou A, Linn J, Vernooij MW, Opherk C, Akoudad S, Baron JC, et al. Cortical superficial siderosis: detection and clinical significance in cerebral amyloid angiopathy and related conditions. Brain. 2015;138(Pt 8):2126-39. Epub 20150626. doi: 10.1093/brain/awv162. PubMed PMID: 26115675.
9. Zanon Zotin MC, Makkinejad N, Schneider JA, Arfanakis K, Charidimou A, Greenberg SM, et al. Sensitivity and Specificity of the Boston Criteria Version 2.0 for the Diagnosis of Cerebral Amyloid Angiopathy in a Community-Based Sample. Neurology. 2024;102(1). doi: 10.1212/wnl.0000000000207940.
10. Cipriano, Lorenzo et al. 'Systematic Review on the Role of Lobar Cerebral Microbleeds in Cognition'. 1 Jan. 2022 : 1025 – 1035

# 5 Key Points

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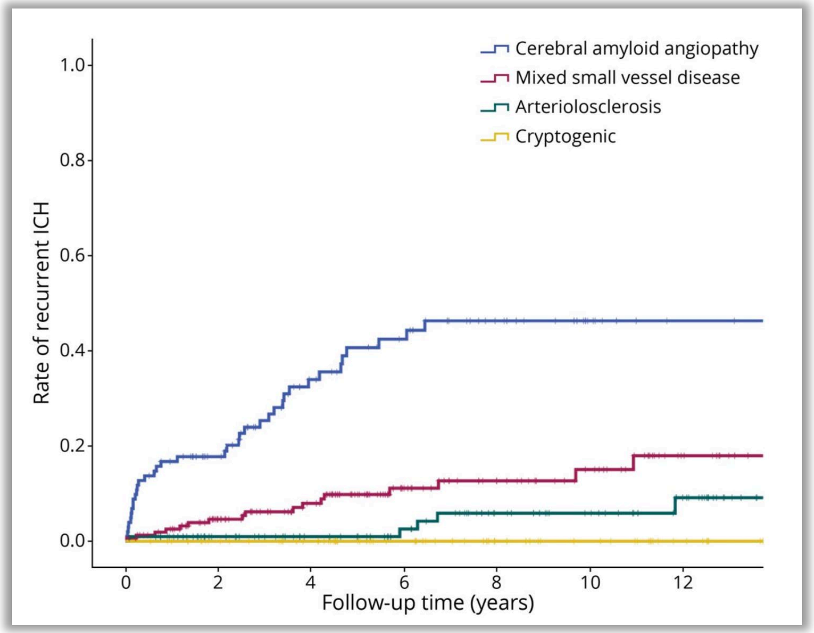
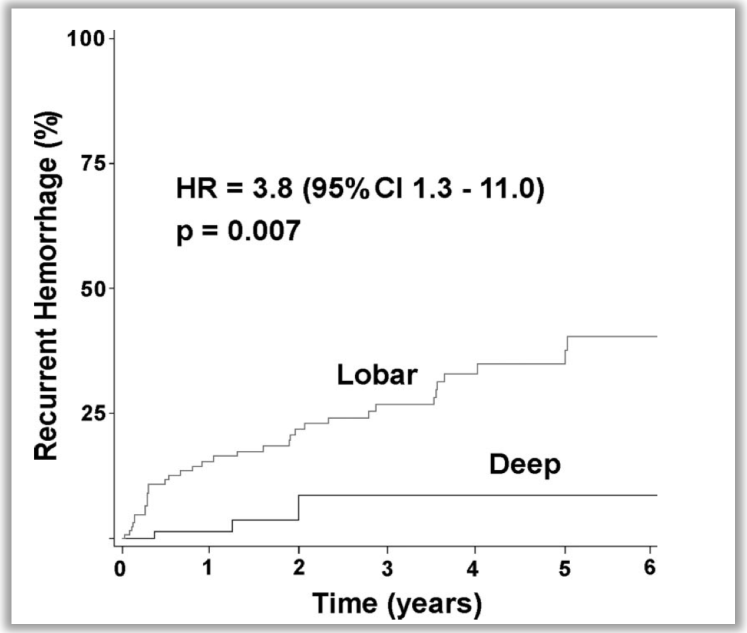
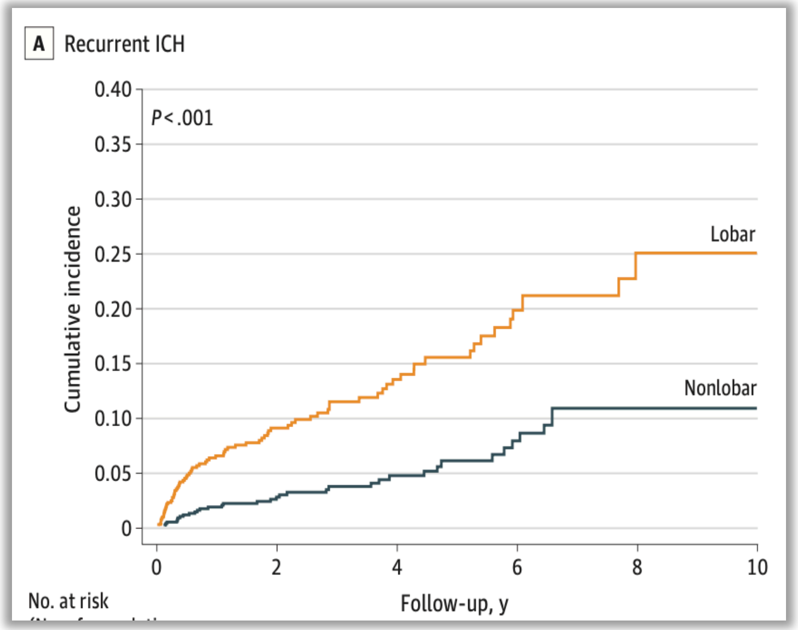
# Risk of **Recurrent** Lobar ICH

**≈ 8 % per year**[4]



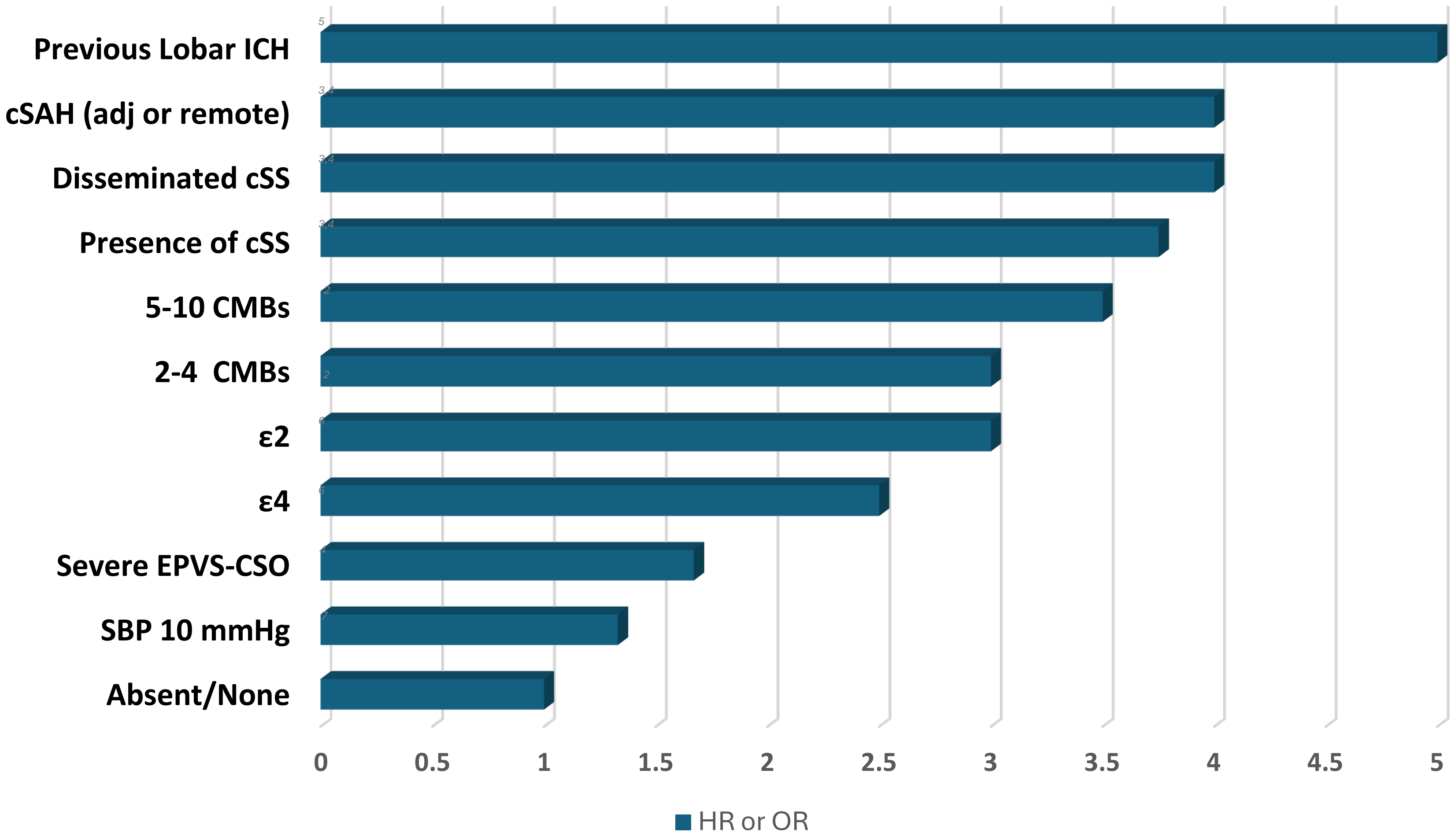

1. Fandler-Hofler S, Obergottsberger L, Ambler G, Eppinger S, Wunsch G, Kneihsl M, et al. Association of the Presence and Pattern of MRI Markers of Cerebral Small Vessel Disease With Recurrent Intracerebral Hemorrhage. Neurology. 2023 Aug 22;101(8):e794-e804. PubMed PMID: 37349111. PMCID: PMC10449438. Epub 20230622.
2. Viswanathan A, Rakich SM, Engel C, Snider R, Rosand J, Greenberg SM, et al. Antiplatelet use after intracerebral hemorrhage. Neurology. 2006;66:206-9.
3. Boe NJ, Hald SM, Jensen MM, Kristensen LMB, Bojsen JA, Elhakim MT, et al. Major Cardiovascular Events After Spontaneous Intracerebral Hemorrhage by Hematoma Location. JAMA Network Open. 2023;6(4).
4. Greenberg SM, van Veluw SJ. Cerebral Amyloid Angiopathy. Stroke. 2024 Jan 25. PubMed PMID: 38269538. Epub 20240125.

# Risk of **Recurrent** Lobar ICH

**Symptomatic Intracerebral Hemorrhage**

+

## High Risk Features




1. Greenberg SM, van Veluw SJ. Cerebral Amyloid Angiopathy. Stroke. 2024 Jan 25. PubMed PMID: 38269538. Epub 20240125.
2. Charidimou A, Imaizumi T, Moulin S, Biffi A, Samarasekera N, Yakushiji Y, et al. Brain hemorrhage recurrence, small vessel disease type, and cerebral microbleeds A meta-analysis. Neurology. 2017;89:820-9.
3. Fandler-Hofler S, Obergottsberger L, Ambler G, Eppinger S, Wunsch G, Kneihsl M, et al. Association of the Presence and Pattern of MRI Markers of Cerebral Small Vessel Disease With Recurrent Intracerebral Hemorrhage. Neurology. 2023 Aug 22;101(8):e794-e804. PubMed PMID: 37349111
4. Jia X, Bo M, Zhao H, Xu J, Pan L, Lu Z. Risk factors for recurrent cerebral amyloid angiopathy-related intracerebral hemorrhage. Front Neurol. 2023;14:1265693. PubMed PMID: 38020625. PMCID: PMC10661374. Epub 20231107.
5. Biffi A, Halpin A, Towfighi A, Gilson A, Busl K, Rost N, et al. Aspirin and recurrent intracerebral hemorrhage in cerebral amyloid angiopathy. Neurology. 2010;75(8):693-8.
6. O'Donnell HC, Rosand J, Knudsen KA, Furie KL, Segal AZ, Chiu RI, et al. Apolipoprotein E Genotype and the Risk of Recurrent Lobar Intracerebral Hemorrhage. New England Journal of Medicine. 2000;342(4):240-5.
7. Biffi A, Anderson CD, Battey TWK, Ayres AM, Greenberg SM, Viswanathan A, et al. Association Between Blood Pressure Control and Risk of Recurrent Intracerebral Hemorrhage. JAMA. 2015;314(9):904.

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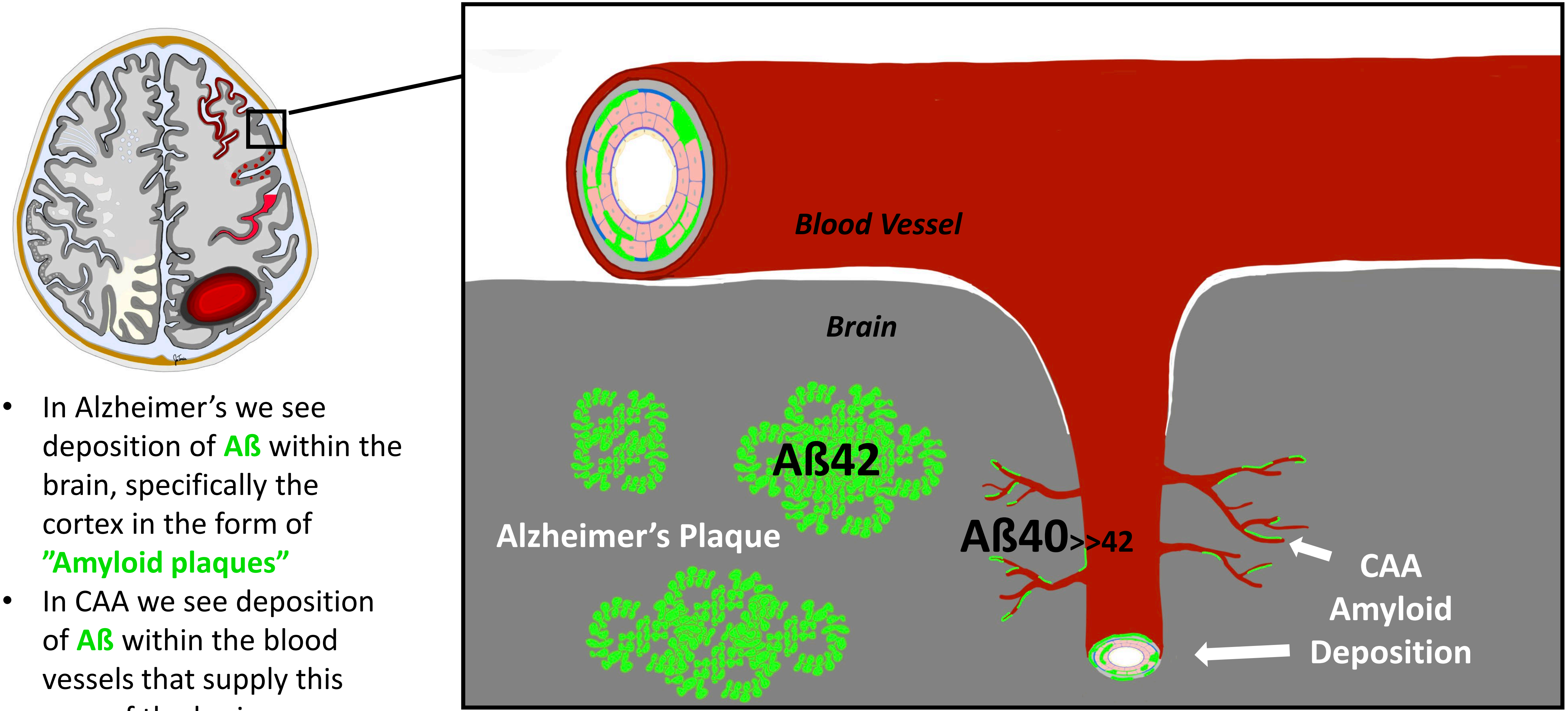
# How is CAA different from Alzheimer's?

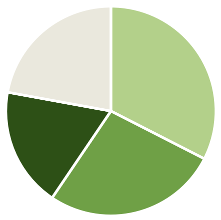



- In Alzheimer's we see deposition of **Aß** within the brain, specifically the cortex in the form of **"Amyloid plaques"**
- In CAA we see deposition of **Aß** within the blood vessels that supply this area of the brain

# CAA and AD: bidirectional overlap

NACC autopsy series, n = 3,976 (Brenowitz 2015)

### Brains with frequent neuritic plaques

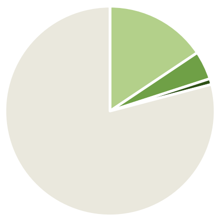


**~80%**

have any-severity CAA

*(n = 3,130)*

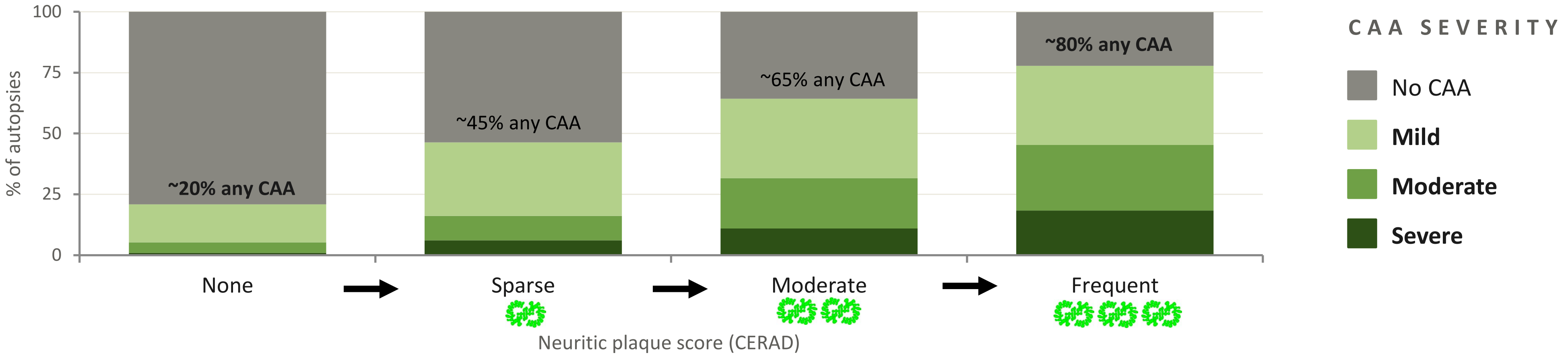
### Brains with no neuritic plaques



**~20%**

have any-severity CAA

*(n = 846)*




*Brenowitz WD, Nelson PT, Besser LM, Heller KB, Kukull WA. Neurobiol Aging. 2015;36(10):2702–2708. PMID: 26239176*

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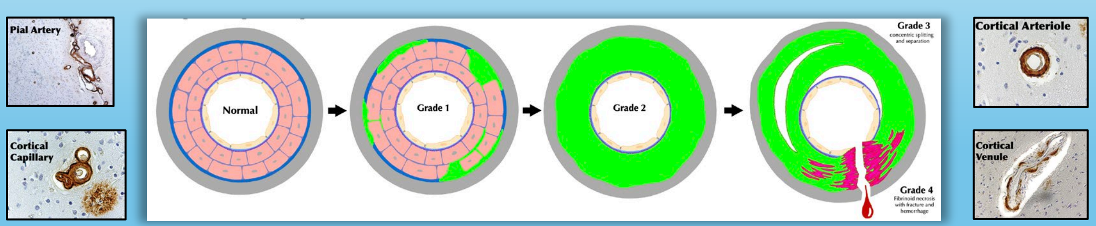
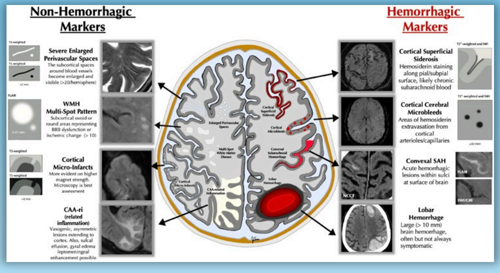
# Detection Method & Definition Matter




ICD-10

11 per 10,000


1. Greenberg SM, Vonsattel JP. Diagnosis of cerebral amyloid angiopathy. Sensitivity and specificity of cortical biopsy. Stroke. 1997 Jul;28(7):1418-22. PubMed PMID: 9227694.
2. Jäkel L, De Kort AM, Klijn CJM, Schreuder FHBM, Verbeek MM. Prevalence of cerebral amyloid angiopathy: A systematic review and meta-analysis. Alzheimer's & Dementia. 2021;18(1):10-28.
3. Bruce SS, Zhang C, Liberman AL, Merkler AE, Navi BB, Chiang GC, et al. Prevalence of Cerebral Amyloid Angiopathy and Associated Risk of Subsequent Ischemic and Hemorrhagic Stroke and Mortality in a Nationwide Cohort. Ann Neurol. 2025. Epub 20250501. doi: 10.1002/ana.27253. PubMed PMID: 40309957.

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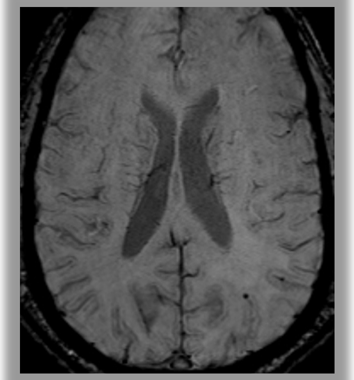
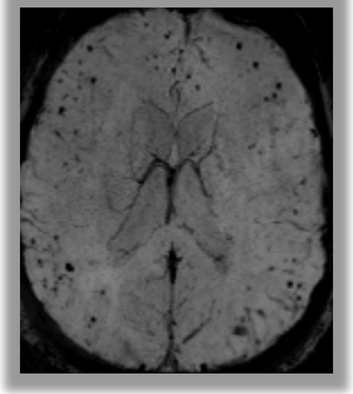
# Annual Risk of Future Symptomatic Hemorrhage

**Single/Few CMBs**



< 1%[7]

**Many CMBs**

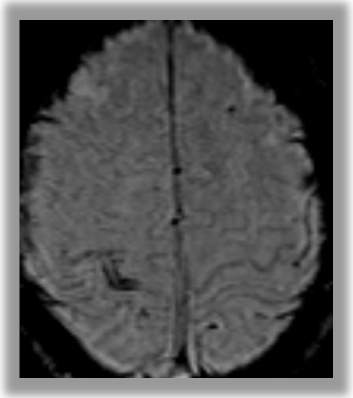


3-5%[6]

**Lobar ICH**

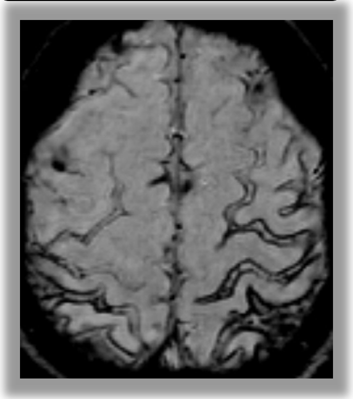
7-9%[5]

**Focal cSS**



9%[2]

**Disseminated cSS**



12.5%[2]
++ if progression[8-9]
++ multifocality[10]

**Convexal SAH**

10-13%[3-4]

**Mildly Elevated**

**Moderately Elevated**

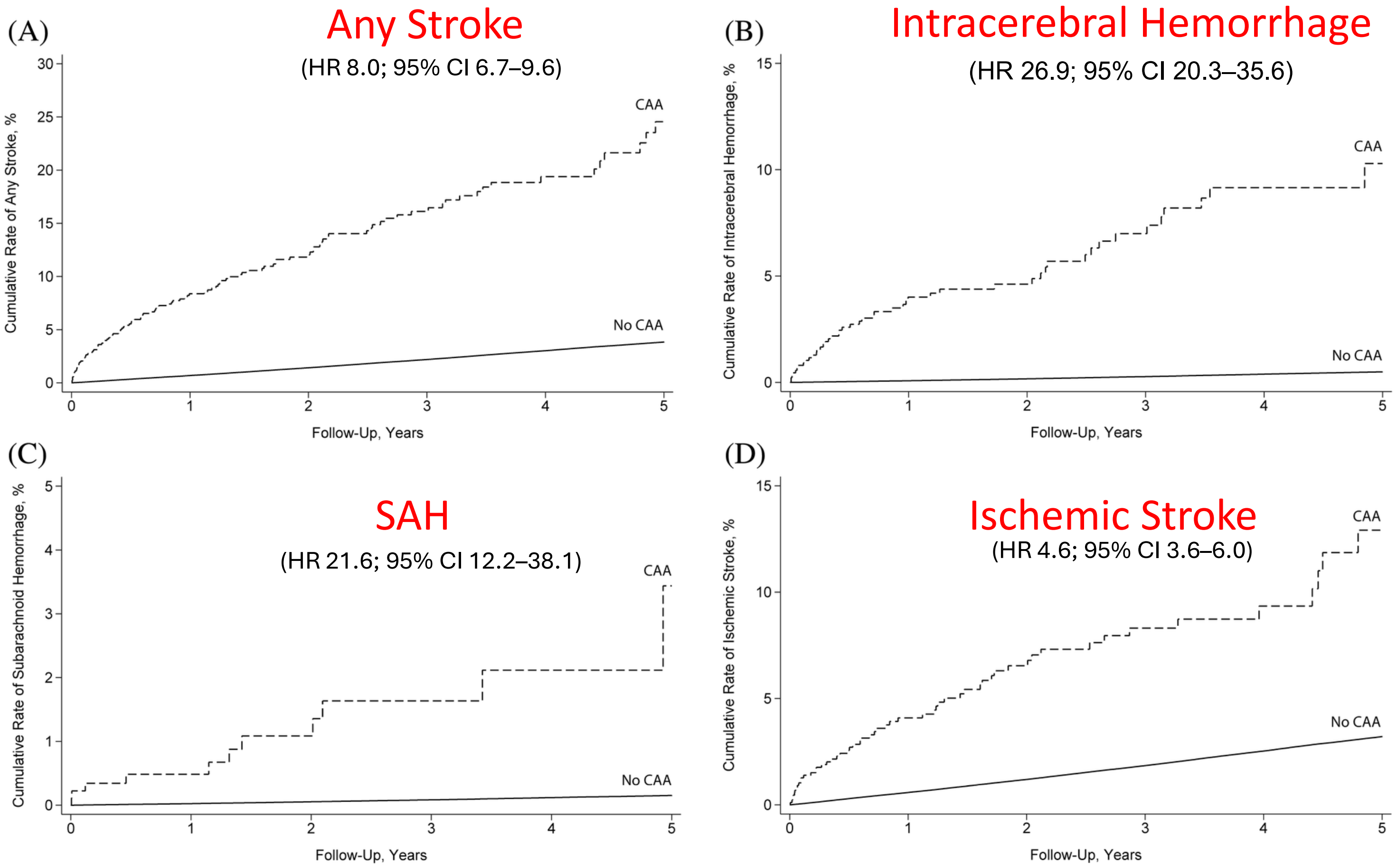
**Severely Elevated**


1. Kozberg MG, Perosa V, Gurol ME, van Veluw SJ. A practical approach to the management of cerebral amyloid angiopathy. Int J Stroke. 2021 Jun;16(4):356-69. PubMed PMID: 33252026. PMCID: PMC9097498.
2. Charidimou A, Boulouis G, Greenberg SM, Viswanathan A. Cortical superficial siderosis and bleeding risk in cerebral amyloid angiopathy: A meta-analysis. Neurology. 2019 Dec 10;93(24):e2192-e202. PubMed PMID: 31732564. PMCID: PMC6937489.
3. Calviere L, Viguier A, Patsoura S, Rousseau V, Albucher JF, Planton M, et al. Risk of Intracerebral Hemorrhage and Mortality After Convexity Subarachnoid Hemorrhage in Cerebral Amyloid Angiopathy. Stroke. 2019 Sep;50(9):2562-4.PMID: 31337297.
4. Hostettler IC, Wilson D, Fiebelkorn CA, Aum D, Ameriso SF, Eberbach F, et al. Risk of intracranial haemorrhage and ischaemic stroke after convexity subarachnoid haemorrhage in cerebral amyloid angiopathy: international individual patient data pooled analysis. Journal of Neurology. 2021;269:1427.
5. Greenberg SM, van Veluw SJ. Cerebral Amyloid Angiopathy. Stroke. 2024 Jan 25. PubMed PMID: 38269538. Epub 20240125.
6. van Etten ES, Auriel E, Haley KE, Ayres AM, Vashkevich A, Schwab KM, et al. Incidence of Symptomatic Hemorrhage in Patients With Lobar Microbleeds. Stroke. 2014;45(8):2280-5.
7. Charidimou A, Shams S, Romero JR, Ding J, Veltkamp R, Horstmann S, et al. Clinical significance of cerebral microbleeds on MRI: A comprehensive meta-analysis of risk of intracerebral hemorrhage, ischemic stroke, mortality, and dementia in cohort studies (v1). International Journal of Stroke. 2018;13(5):454-68.
8. Pongpitakmetha T, Fotiadis P, Pasi M, Boulouis G, Xiong L, Warren AD, et al. Cortical superficial siderosis progression in cerebral amyloid angiopathy: Prospective MRI study. Neurology. 2020 Apr 28;94(17):e1853-e65. PubMed PMID: 32284360. PMCID: PMC7274850
9. Charidimou A, Boulouis G, Xiong L, Pasi M, Roongpiboonsopit D, Ayres A, et al. Cortical Superficial Siderosis Evolution. Stroke. 2019 Apr;50(4):954-62. PubMed PMID: 30869563. PMCID: PMC6433512.
10. Charidimou A, Boulouis G, Roongpiboonsopit D, Auriel E, Pasi M, Haley KE, et al. Cortical superficial siderosis multifocality in cerebral amyloid angiopathy. Neurology. 2017;89:2128-35.

# Prevalence of Cerebral Amyloid Angiopathy and Associated Risk of Subsequent Ischemic and Hemorrhagic Stroke and Mortality in a Nationwide Cohort

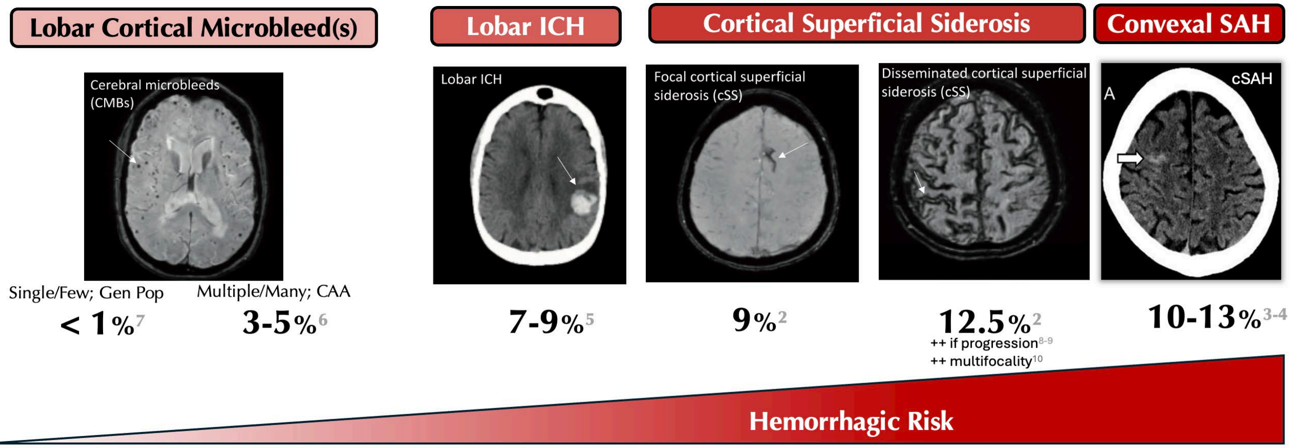
Samuel S. Bruce, MD, MA,[1] Cenai Zhang, MS,[1] Ava L. Liberman, MD,[1]
Alexander E. Merkler, MD, MS,[1] Babak B. Navi, MD, MS,[1] Gloria C. Chiang, MD,[2]
Costantino Iadecola, MD,[1] Hooman Kamel, MD, MS,[1†] and
Santosh B. Murthy, MD, MPH[1†]

**Retrospective cohort study using inpatient and outpatient claims from 2008 to 2022 from a 5% national sample of Medicare beneficiaries**
***(Total N= 1,920,312, CAA N= 2161)***

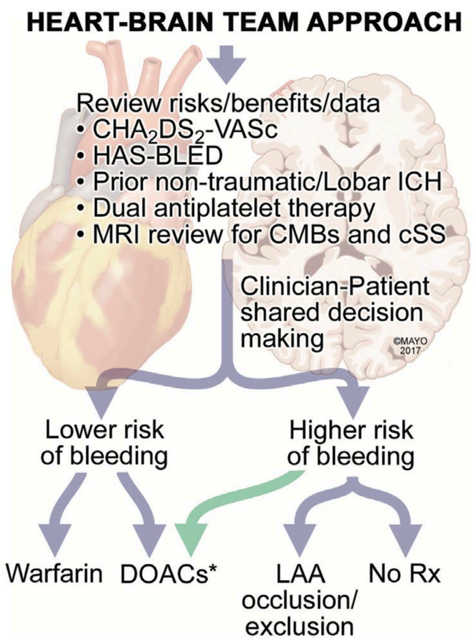




1. Bruce SS, Zhang C, Liberman AL, Merkler AE, Navi BB, Chiang GC, et al. Prevalence of Cerebral Amyloid Angiopathy and Associated Risk of Subsequent Ischemic and Hemorrhagic Stroke and Mortality in a Nationwide Cohort. Ann Neurol. 2025. Epub 20250501. doi: 10.1002/ana.27253. PubMed PMID: 40309957.

# Cardiovascular Management Summary

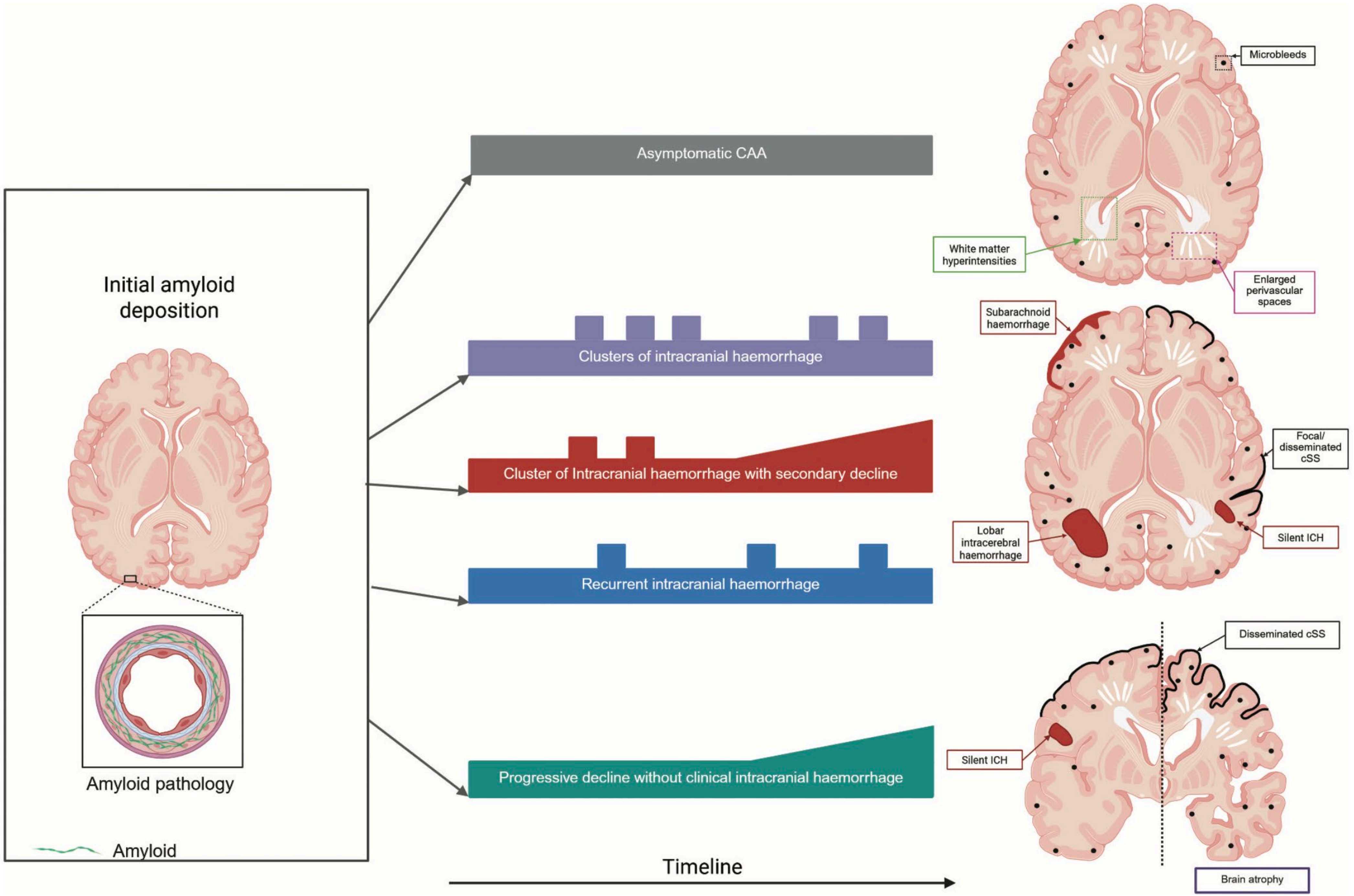



| | | |
|---|---|---|
| **HTN** | **Goal is CONTROL – Consistent monitoring, affordable regimen, provider & patient accountability**<br><br>SBP **< 130/80 mmHg**<br>*Consider < 120/80 mmHg in those who can tolerate (evidence limited)* | |
| **Anti-Hyperlipidemic** | Statin impact appears low and likely ok when indicated for AHA/ASA Guidelines<br>Hydrophilic statin preference: rosuvastatin and pravastatin | Despite lack of direct evidence, strong consideration should be made for alternatives to statins (PCSK9i, **ezetimibe***, inclisiran, **Nexlizet**) especially if high risk / amplifying features are present |
| **Antiplatelet** | **Monotherapy** antiplatelet when indicated in secondary prevention<br><br>Minimize use of long-term dual-antiplatelet | **Avoid dual antiplatelet therapy** unless post-stent – limit duration<br><br>*Post-AIS DAPT should limit to 7-10 days* |
| **Anticoagulation** | **Avoid use of VKA** unless clearly indicated by evidence (Mechanical Valve, Valvular A.fib, Antiphospholipid Antibody Syndrome, etc) | **Avoid use / limit duration of DOAC when possible**:<br>- A.fib: Consider Left Atrial Appendage Closure vs. reduced dose DOAC<br>- VTE: Consider IVC filter in select cases |





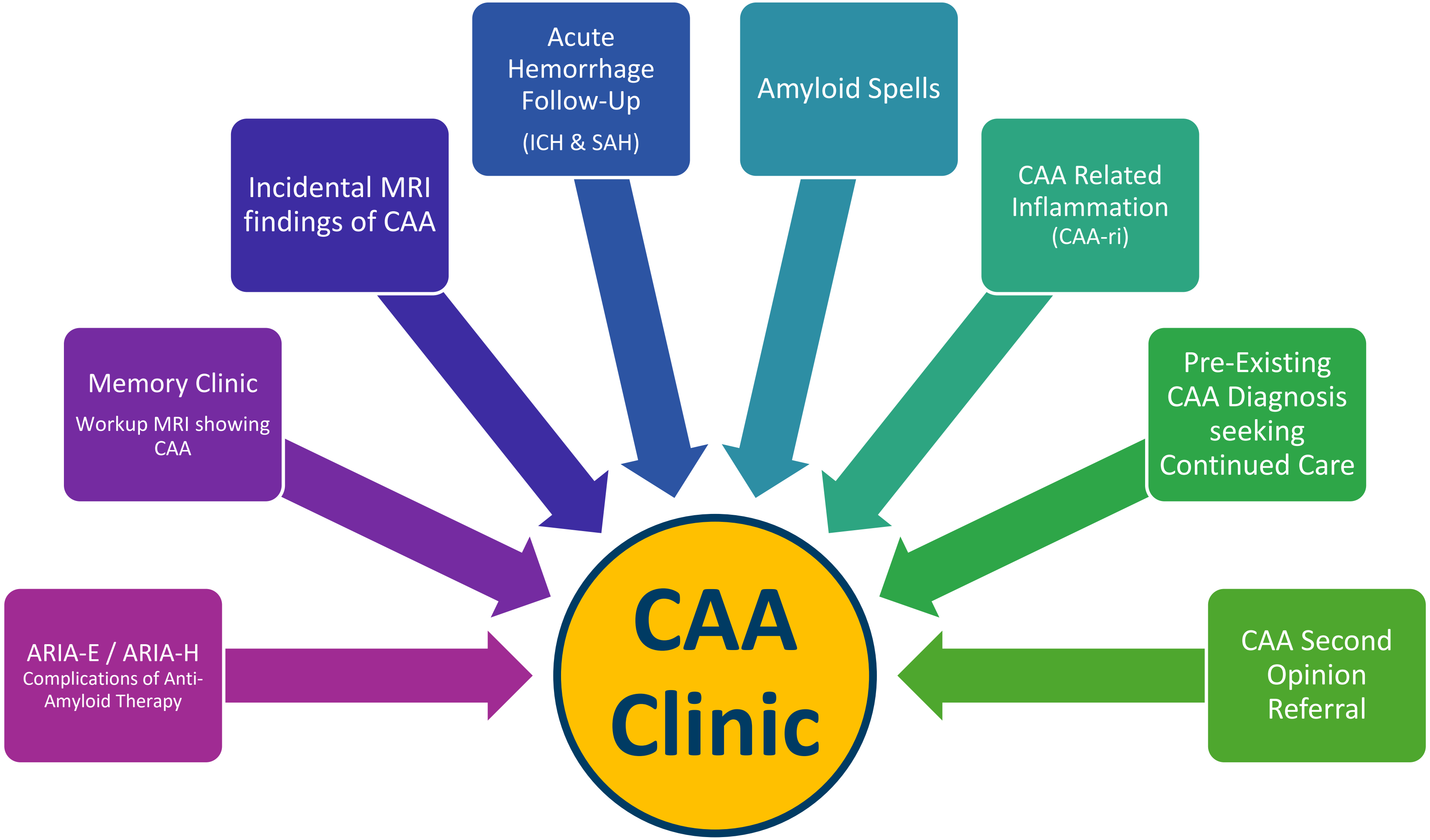
1. Charidimou A, Smith EE. Cardiovascular Management in Asymptomatic (Silent) Cerebral Microbleeds and Suspected Cerebral Amyloid Angiopathy. Stroke. 2024 Apr;55(4):1101-12. PubMed PMID: 38465605. Epub 20240311.
2. DeSimone CV, Graff-Radford J, El-Harasis MA, Rabinstein AA, Asirvatham SJ, Holmes DR, Jr. Cerebral Amyloid Angiopathy: Diagnosis, Clinical Implications, and Management Strategies in Atrial Fibrillation. J Am Coll Cardiol. 2017;70(9):1173-82. doi: 10.1016/j.jacc.2017.07.724. PubMed PMID: 28838368

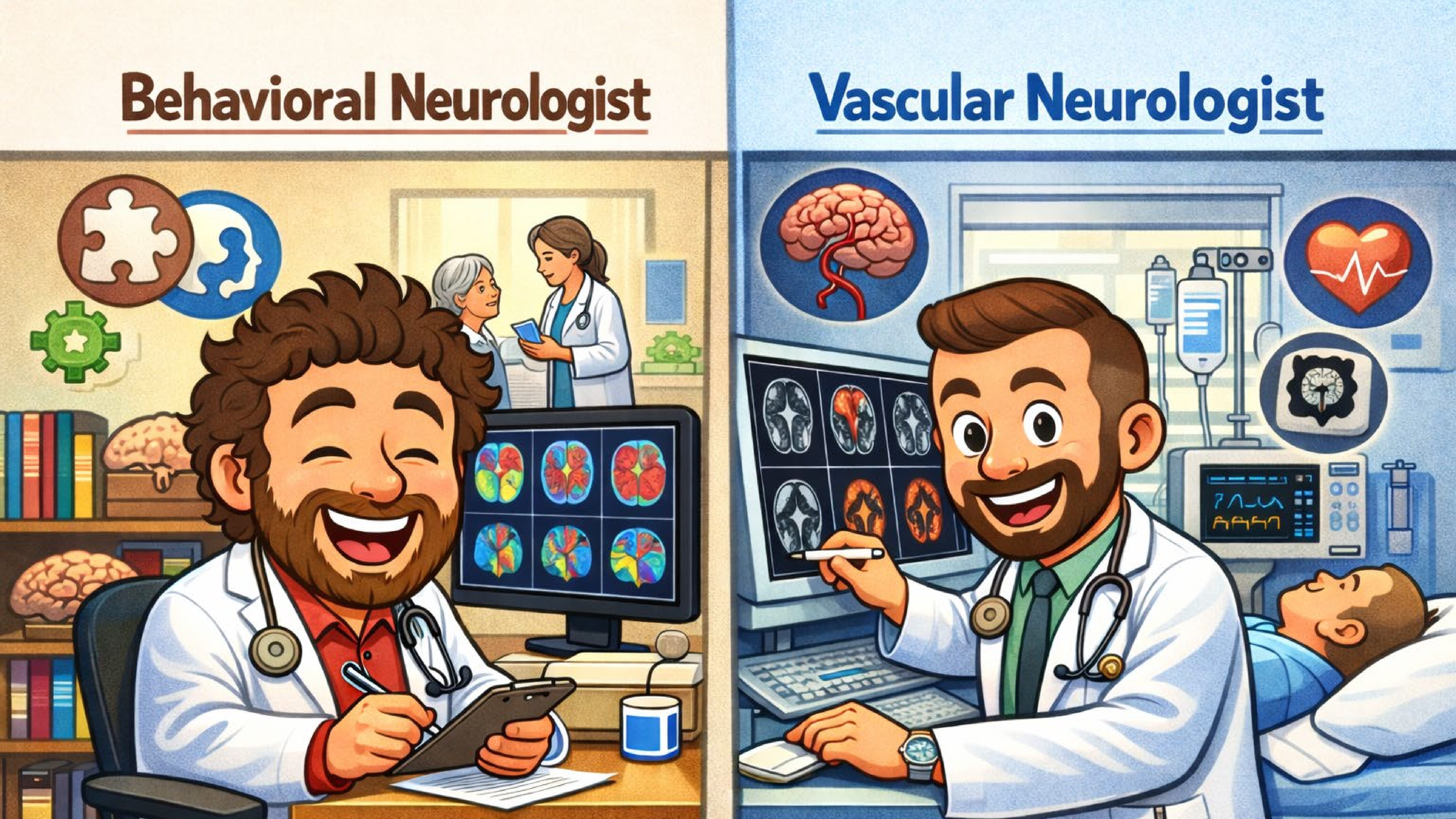



1. Sellimi A, Schwartze J, Humphries F, Panteleienko L, Mallon D, Banerjee G, et al. Contemporary perspectives in cerebral amyloid angiopathy. Expert Rev Neurother. 2025:1–24. doi: 10.1080/14737175.2025.2526113.

# Cerebral Amyloid Angiopathy Clinic

Ochsner
The Debra H. and Robert J. Patrick Neuroscience Institute




Behavioral Neurologist
Vascular Neurologist

# Cerebral Amyloid Angiopathy Clinic

**Ochsner**
The Debra H. and Robert J. Patrick Neuroscience Institute

**Neurocognitive History & Exam** (~60 min)

**Vascular Neurology Assessment** (~60 min)
CAA Vascular Risk Factor Profiling

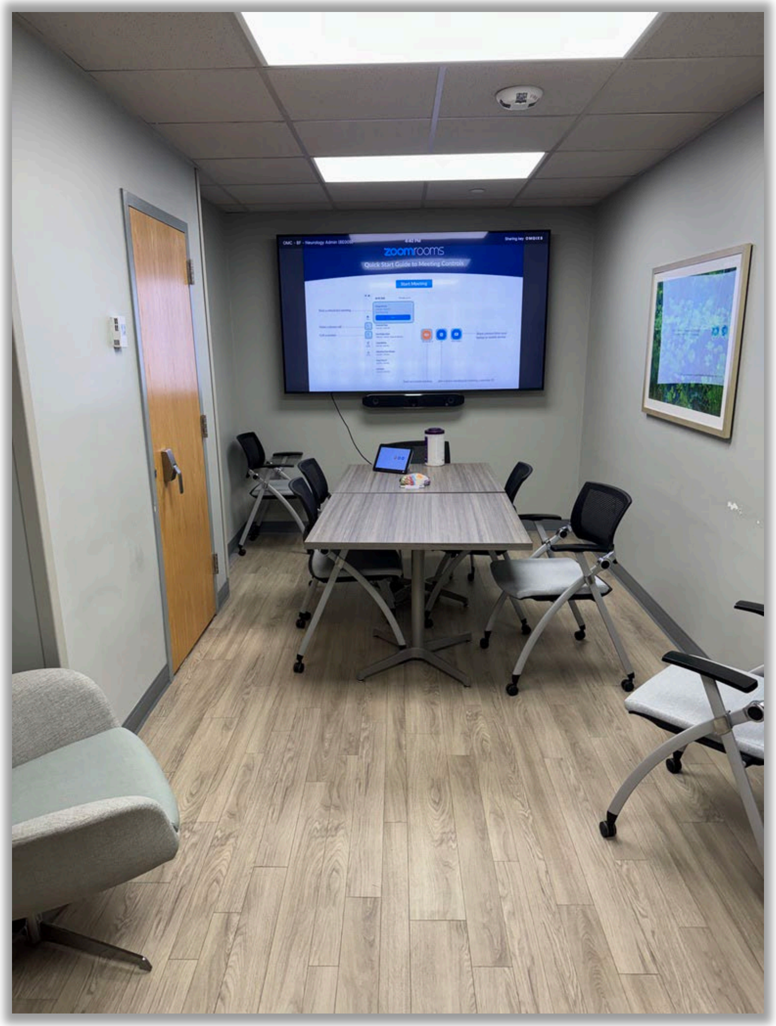
**Neurocognitive Testing** (~60 min)

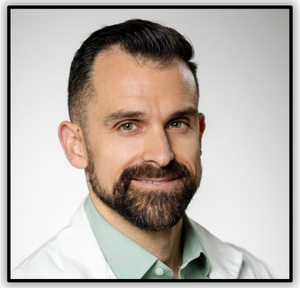
**Team-Based Case Discussion**

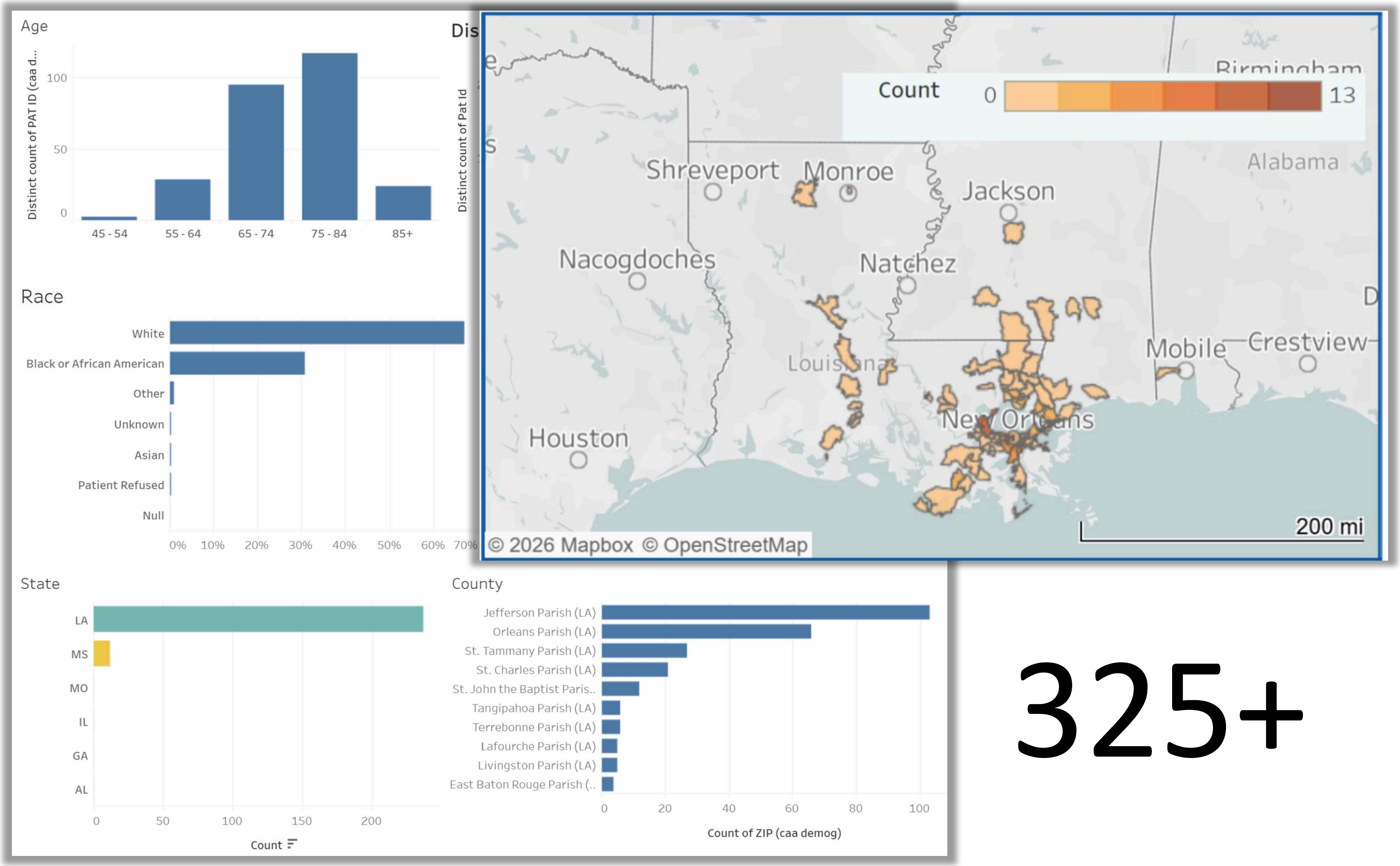
## Patient & Family Debrief Meeting

**Meeting as a group with all CAA providers in our conference room to review and summarize the details specific to your case and discuss the next steps**

(Prognosis, Imaging Review, Management Plan)






Age
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0
45 - 54
55 - 64
65 - 74
75 - 84
85+
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Other
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Patient Refused
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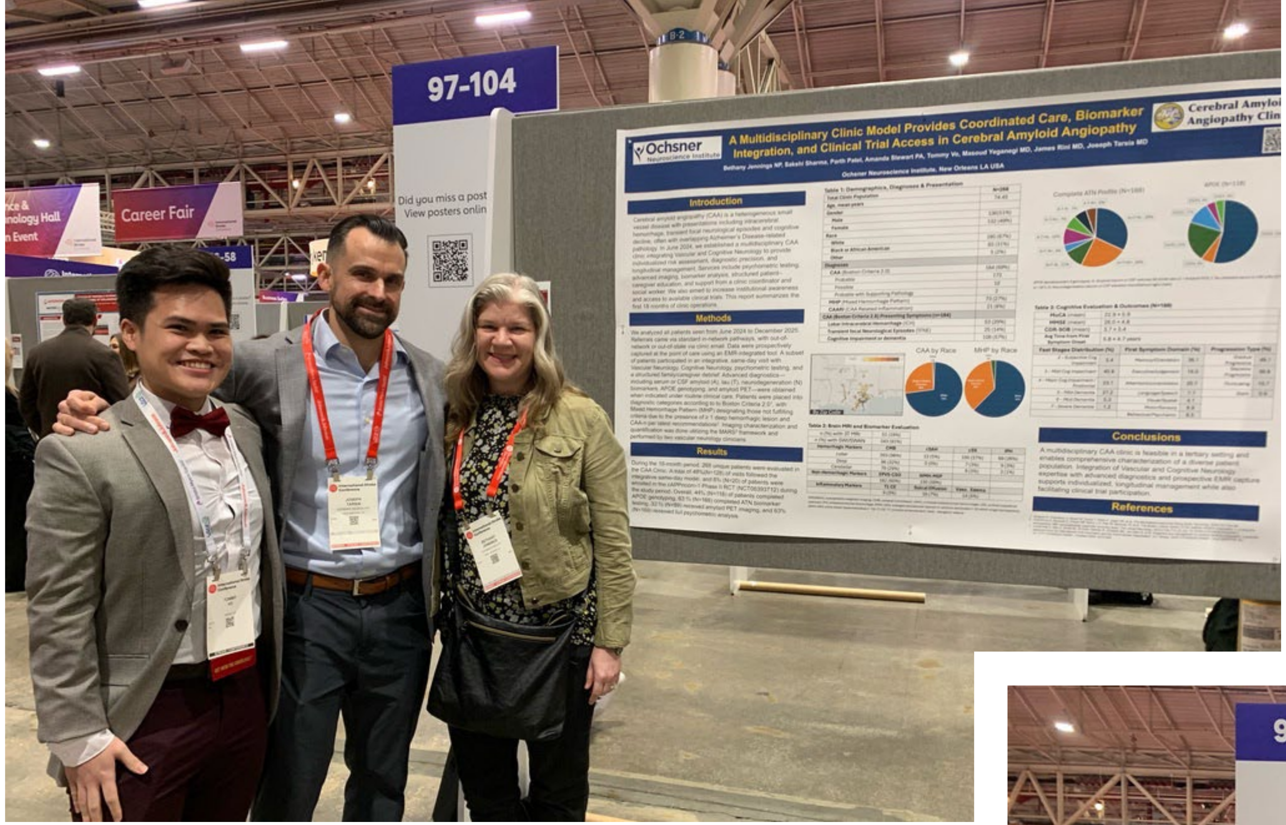


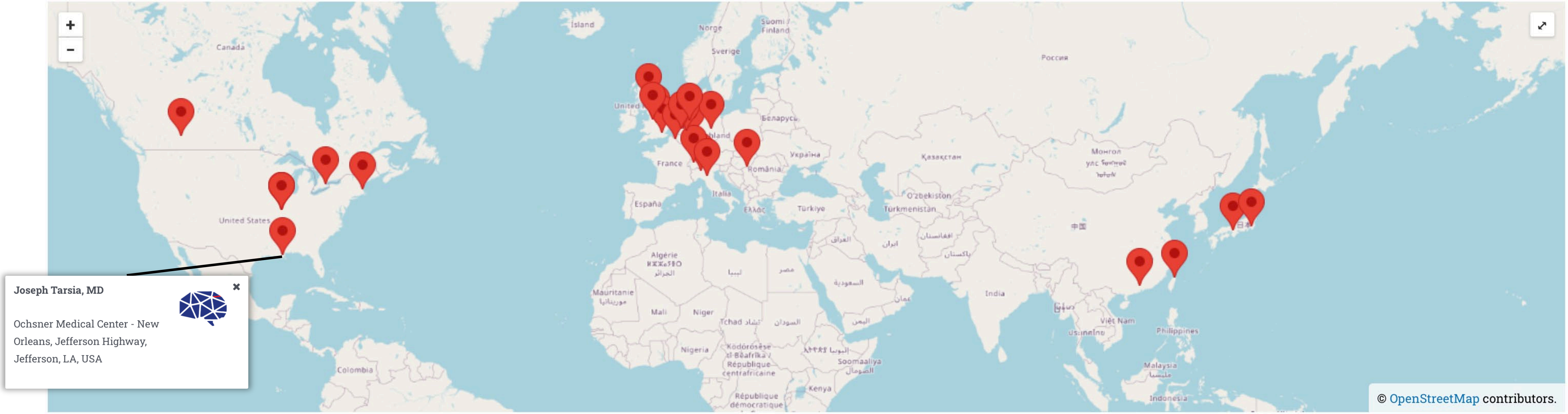


# International Stroke Conference 2026

INTERNATIONAL
# caa
ASSOCIATION

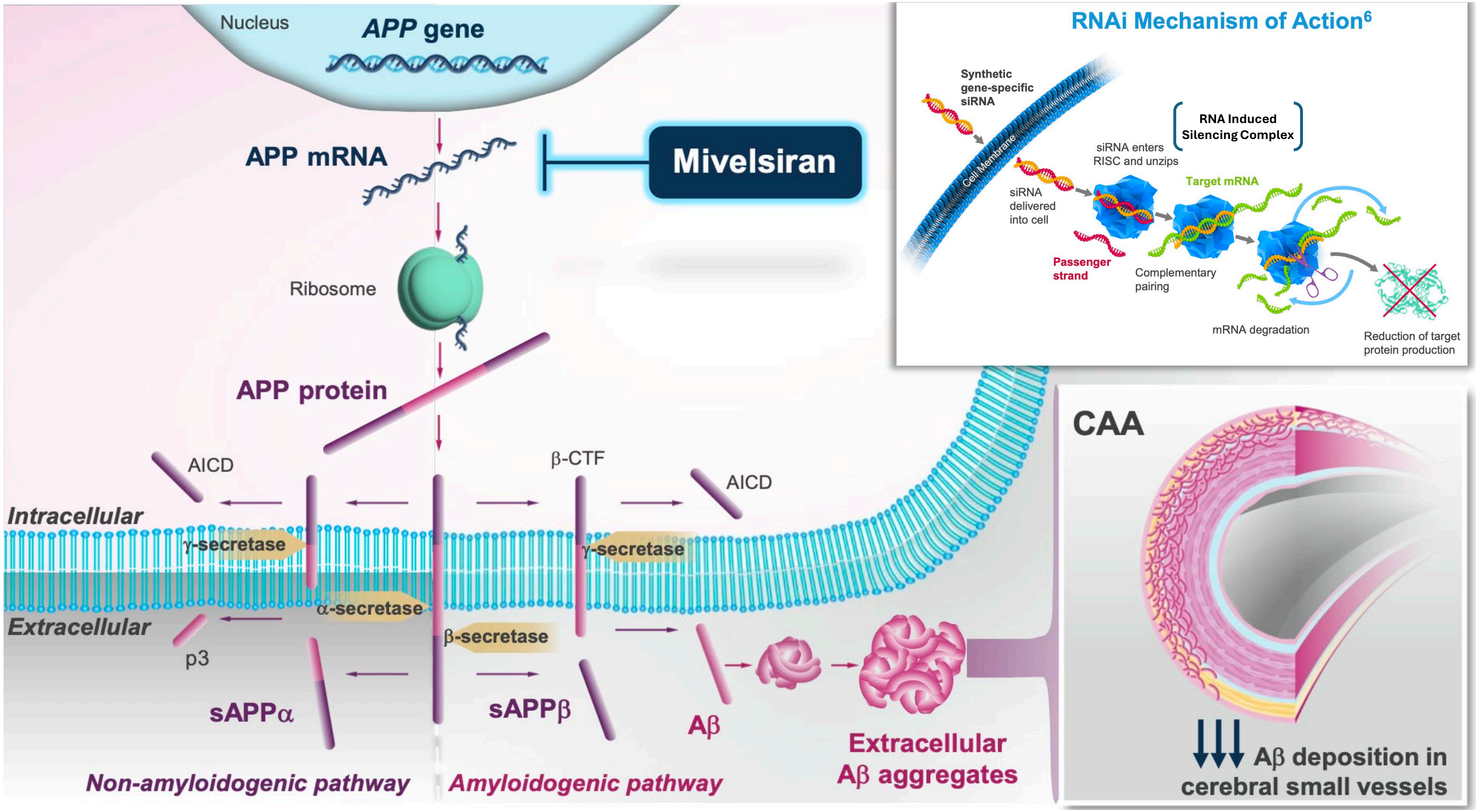


## Find a Doctor



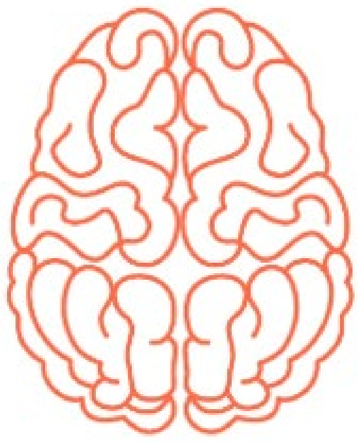
# cAPPricorn-1: A Phase 2 Study of Mivelsiran in CAA

## Multiple-Dose Study Evaluating Efficacy, Safety, and Pharmacodynamics





1. Greenberg, et al. *Nat Rev Neurol.* 2020;16(1):30-42. 2. DeSimone CV, et al. *J Am Coll Cardiol.* 2017. 3. O'Brien RJ, et al. *Annu Rev Neurosci.* 2011. 4. Gatti L, et al. *Int J Mol Sci.* 2020 May; 21(10): 3435.
6. Jadhav V et al. Nature Biotechnol. 2024;42:394–405.



cAPPricorn-1

# A Phase 2 Trial of ALN-APP in Patients With Cerebral Amyloid Angiopathy

ENROLLING

Alnylam

The purpose of the study is to evaluate the effect of ALN-APP on measures of CAA disease progression and to characterize the safety, tolerability, and pharmacodynamics (PD) of ALN-APP in adult patients with sporadic CAA (sCAA) and Dutch-type CAA (D-CAA). The study will be conducted over 2 periods: a 24-month double-blind treatment period and an optional 18-month open-label extension (OLE) period. The estimated duration of study participation, inclusive of screening, treatment, and additional safety follow-up, is up to 50 months.

## As of today, 22 patients enrolled at Ochsner which is 15% of the total trial population

## (61 international sites)

**#1**

cAPPricorn-1 Enrollment Leaderboard

May 2025

| Current Rank | Site | Country | Randomized Last Month | Total Randomized | Previous Chart Position |
|---|---|---|---|---|---|
| 1 | Site 1020 (Dr. Joseph Tarsia) Ochsner Health System PARENT | United States | 3 | 12 | 1 |
| 2 +2 ▲ | Site 1202 (Dr. Richard Bergeron) Recherches Neuro-Hippocampe Inc., d/b/a Ottawa Memory Clinic | Canada | 2 | 4 | 4 |
| NEW 2 | Site 1015 (Dr. Mariel Kozberg) Massachusetts General Hospital | United States | 2 | 2 | |
| NEW 4 | Site 1013 (Dr. Scott Kasner) Hospital of the University of Pennsylvania | United States | 1 | 3 | |
| 4 -3 ▼ | Site 5201 (Prof. Giovanni Frisoni) Hôpitaux Universitaires de Genève - HUG-Centre de la mémoire, Bâtiment A1 - Morier | Switzerland | 1 | 3 | 1 |
| NEW 4 | Site 1025 (Dr. Shawn Kile) Sutter Institute for Medical Research | United States | 1 | 2 | |
| NEW 4 | Site 1010 (Dr. Eugene Scharf) Mayo Clinic | United States | 1 | 1 | |
| NEW 4 | Site 1029 (Dr. Sheila Baez Torres) K2 Medical Research, | United | 1 | 1 | |

# Thank You

Email: CAA@Ochsner.org

Cerebral Amyloid Angiopathy Clinic

Ochsner Neuroscience Institute