

Advanced MRI in cerebral small vessel disease

GIGA CRC in vivo imaging
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1. Scientific context

- **Introduction to cerebral small vessel disease**

Cerebral small vessel disease (CSVD) is a group of cerebrovascular pathologies affecting the brain's **small caliber vessels**, characterized by clinical, radiological, and neuropathological specificities.



1. Scientific context

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Cerebral small vessel disease (CSVD) is a group of cerebrovascular pathologies affecting the brain's **small caliber vessels**, characterized by clinical, radiological, and neuropathological specificities.

2 main etiologies :

- **Arteriolosclerosis**
- **Cerebral amyloid angiopathy**



1. Scientific context

- **Introduction to cerebral small vessel disease**

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2 main etiologies :

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- **Cerebral amyloid angiopathy**

Other etiologies :

- Genetic small vessel angiopathy
- Inflammation and immune-mediated small vessel diseases
- Venous collagenosis



1. Scientific context

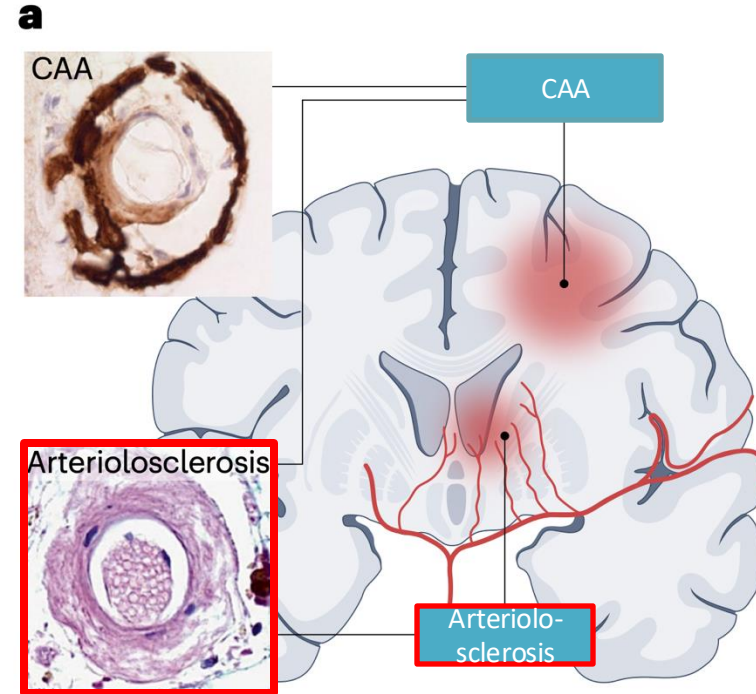
- **Epidemiology**

- A highly prevalent pathology, particularly among the elderly :
 - 25% of people over 80 years of age had > 1 silent stroke
 - The prevalence of white matter hyperintensities (WMH) increases from 5% at 50 years of age to nearly 100% at 90 years of age
- CSVD contributes to about 20% of strokes (including 25% of ischemic strokes) and 45% of dementias.
- **Risk factors :**
 - Mainly hypertension and diabetes
 - Age
 - Others common vascular risk factors : Smoking, dyslipidemia, obesity, cardiovascular disease, alcohol intake, chronic kidney disease



1. Scientific context

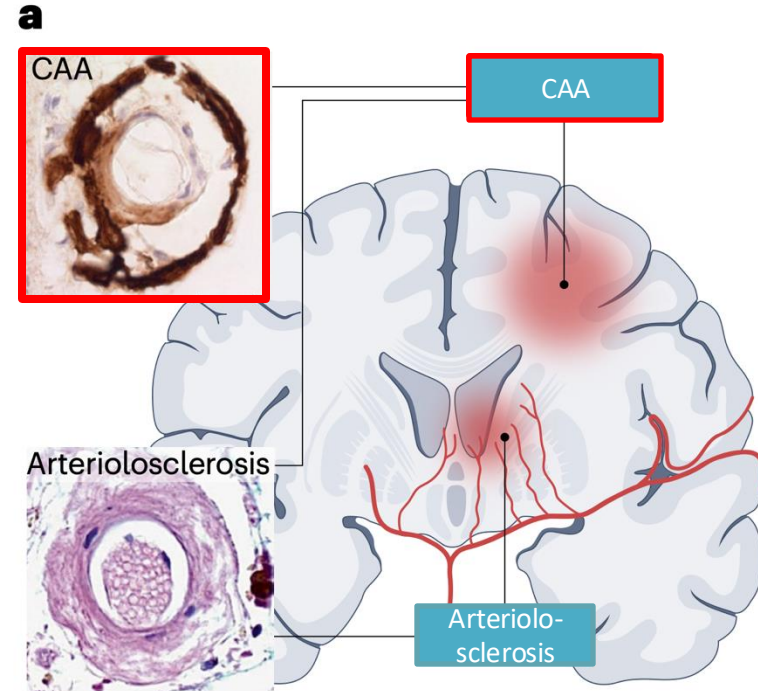
- **Neuropathology**
- Arteriolosclerosis : arteriolar fibrinoid necrosis, lipohyalinosis, microatheroma, or microaneurysms
 - ✓ Deep perforating arteries are mainly affected.





1. Scientific context

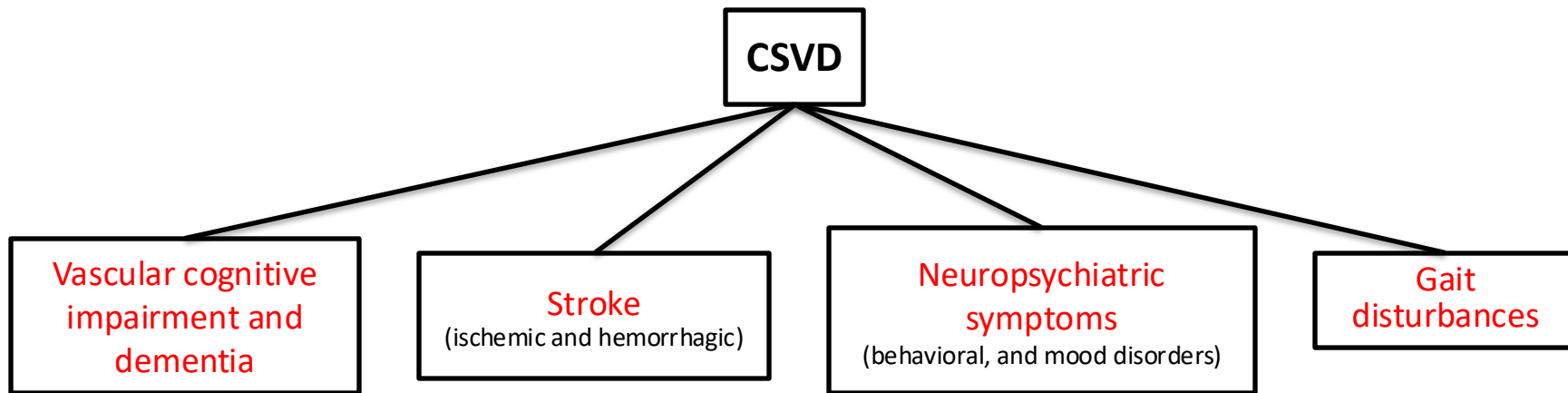
- **Neuropathology**
- CAA : abnormal deposition of amyloid- β ($A\beta$) within the vessel walls results in gradual vessel wall degeneration and, ultimately, breach of integrity/rupture leading to asymptomatic microhemorrhages (cerebral microbleeds) or lobar intraparenchymal hemorrhage.
 - ✓ Leptomeningeal and cortical vessels are primarily affected.





1. Scientific context

- **Clinical manifestations**





1. Scientific context

- CSVD features on neuroimaging

Standards for Reporting Vascular Changes on Neuroimaging 2 (STRIVE- 2) categorized and standardized the diverse **features of SVD** that are visible on structural MRI (2023).



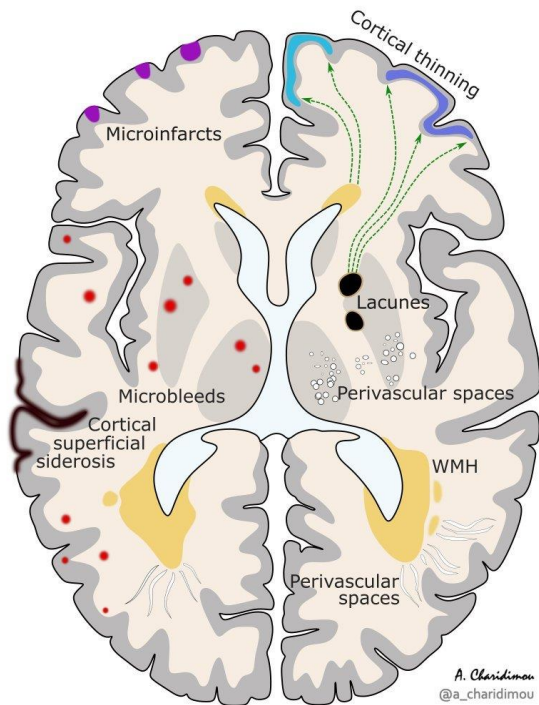
Neuroimaging standards for research into small vessel disease—advances since 2013

Marco Duering, Geert Jan Biessels, Amy Brodtmann, Christopher Chen, Charlotte Cordonnier, Frank-Erik de Leeuw, Stéphanie Debette, Richard Frayne, Eric Jouvent, Natalia S Rost, Annemieke ter Telgte, Rustam Al-Shahi Salman, Walter H Backes, Hee-Joon Bae, Rosalind Brown, Hugues Chabriat, Alberto De Luca, Charles deCarli, Anna Dewenter, Fergus N Doubal, Michael Ewers, Thalia S Field, Aravind Ganesh, Steven Greenberg, Karl G Helmer, Saima Hilal, Angela C C Jochems, Hanna Jokinen, Hugo Kuijf, Bonnie Y K Lam, Jessica Lebenberg, Bradley J MacIntosh, Pauline Maillard, Vincent C T Mok, Leonardo Pantoni, Salvatore Rudilosso, Claudia L Satizabal, Markus D Schirmer, Reinhold Schmidt, Colin Smith, Julie Staals, Michael J Thrippleton, Susanne J van Veluw, Prashanthi Vemuri, Yilong Wang, David Werring, Marialuisa Zedde, Rufus O Akinyemi, Oscar H Del Brutto, Hugh S Markus, Yi-Cheng Zhu, Eric E Smith*, Martin Dichgans*, Joanna M Wardlaw**



1. Scientific context

- CSVD neuroimaging features**



Lesion	Size (mm)	MRI Features
Lacune	3-15	Acute—diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR)/T2: hyperintense; T1: hypointense; GRE/SWI: isointense Chronic—DWI, FLAIR, and T1: hypointense; T2: hyperintense; gradient recalled echo (GRE)/ susceptibility-weighted imaging (SWI): isointense
Recent small subcortical infarcts	≤20	DWI and FLAIR/T2: hyperintense; T1: hypointense; GRE/SWI: isointense
White matter hyperintensities	Variable	DWI and GRE/SWI: isointense; FLAIR/T2: hyperintense; T1: hypointense
Enlarged perivascular spaces	≤2	DWI and GRE/SWI: isointense; FLAIR and T1: hypointense; T2: hyperintense
Cerebral microbleeds	≤10	DWI, FLAIR, T2, and T1: isointense; GRE/SWI: hypointense
Cortical superficial siderosis	Variable	DWI, FLAIR, T2, and T1: isointense; GRE/SWI: hypointense
Cortical microinfarcts	0.2-3	DWI and FLAIR/T2: hyperintense; T1: hypointense; GRE/SWI: isointense

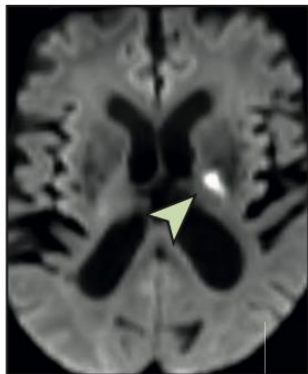


1. Scientific context

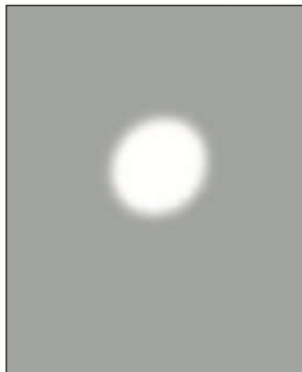
- Recent small subcortical infarct

Neuroimaging evidence of a **recent infarction in the territory of one perforating arteriole**, with imaging features and **clinical symptoms** consistent with a lesion (occurring within the previous few—ie, around 3—weeks).

Recent small
subcortical infarct



DWI



Usual
diameter

≤ 20 mm

DWI

↑

FLAIR

↑

T2-weighted

↑

T1-weighted

↓

T2*-weighted

↔



1. Scientific context

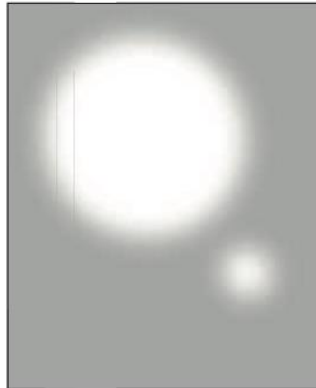
- White matter hyperintensity (of presumed vascular origin)

*Signal abnormality of variable size in the white matter that is **hyperintense on T2-weighted images**, such as fluid-attenuated inversion recovery (FLAIR), without cavitation (signal different from CSF).*

White matter
hyperintensity



FLAIR



Usual
diameter

DWI

FLAIR

T2-weighted

T1-weighted

T2*-weighted

Variable

↔

↑

↑

↔ (↓)

↑

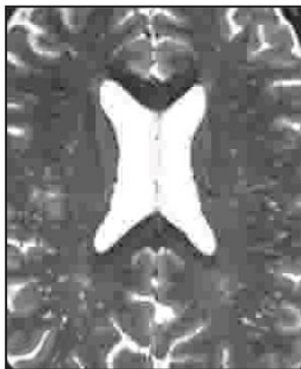


1. Scientific context

- Perivascular space

*Fluid-filled space, which follows the typical course of a vessel penetrating the brain through grey or white matter; has **signal intensity similar to CSF on all sequences**; has a round, ovoid, or linear shape (depending on the slice direction) with a diameter commonly not exceeding **2 mm** when imaged perpendicular to the course of the vessel*

Perivascular space



T2-weighted



T1-weighted



Usual
diameter

≤ 2 mm

DWI

$\leftrightarrow$

FLAIR

$\downarrow/\leftrightarrow$

T2-weighted

$\uparrow$

T1-weighted

$\downarrow$

T2*-weighted

$\leftrightarrow$

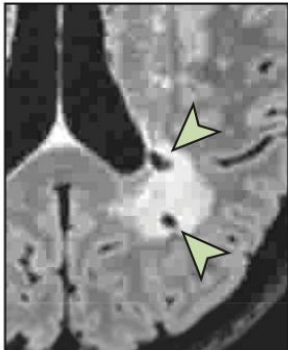


1. Scientific context

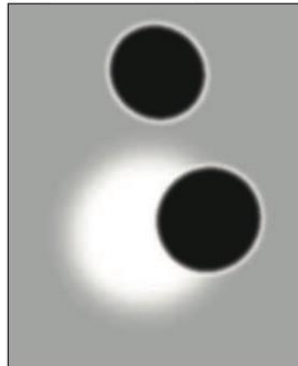
- **Lacune (of presumed vascular origin)**

*Round or ovoid, subcortical, **fluid filled** (similar signal to CSF) **cavity up to 15 mm** in diameter that is likely to be **end tissue damage** from several causes (recent small subcortical infarct, small subcortical haemorrhage, or end-stage cavitation in a white matter hyperintensity).*

Lacune



FLAIR



Usual diameter	3–15 mm
DWI	↔ (↓)
FLAIR	↓
T2-weighted	↑
T1-weighted	↓
T2*-weighted	↔

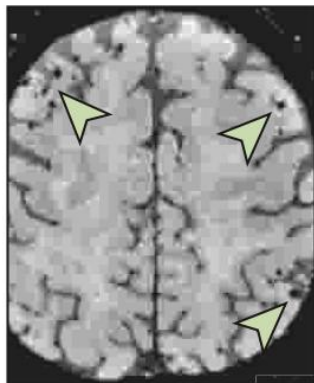


1. Scientific context

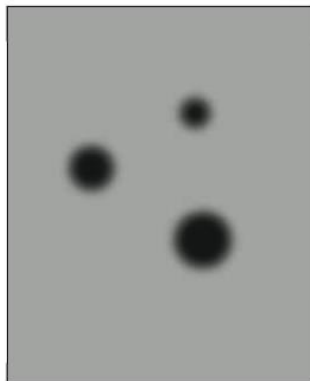
- **Cerebral microbleed**

*Small (usually 2–5 mm or sometimes 10 mm in size) areas of **signal void with associated blooming artifact** on T2* or other MRI sequences sensitive to **susceptibility effects**.*

Cerebral microbleed



T2*-weighted and SWI



Usual diameter	≤10 mm
DWI	↔
FLAIR	↔
T2-weighted	↔
T1-weighted	↔
T2*-weighted	↓↓

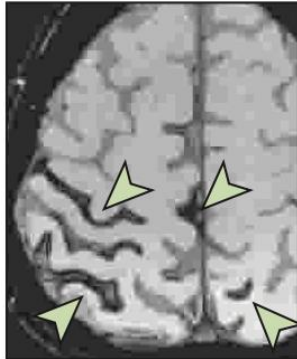


1. Scientific context

- **Cortical superficial siderosis**

Thin areas of hypointensity on T2, or other **MRI sequences sensitive to susceptibility effects**, in or overlying the **superficial cortex**, which can be confined to one gyrus and adjacent sulci, or occasionally more widespread affecting several brain regions.*

Cortical superficial siderosis



T2*-weighted and SWI



Usual
diameter
DWI
FLAIR
T2-weighted
T1-weighted
T2*-weighted

Variable

↔

↔

↔

↔

↓↓

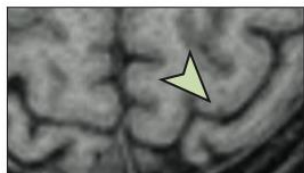
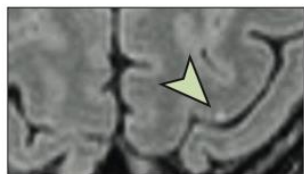


1. Scientific context

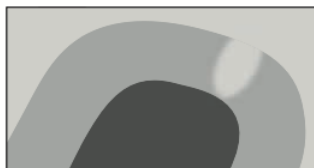
- **Cortical cerebral microinfarct**

Small lesions appearing hypointense on T1-weighted, hyperintense on T2-weighted or FLAIR, and isointense on T2-weighted MRI, operationally defined to be **strictly cortical** and with an upper size limit of **4 mm**.*

Cortical cerebral
microinfarct



T2-weighted



T1-weighted



Usual
diameter
DWI
FLAIR
T2-weighted
T1-weighted
T2*-weighted

<4 mm

↔/↑ (acute)

↑/↓

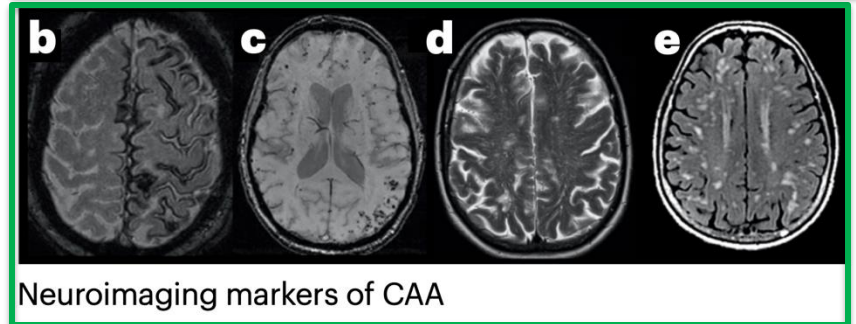
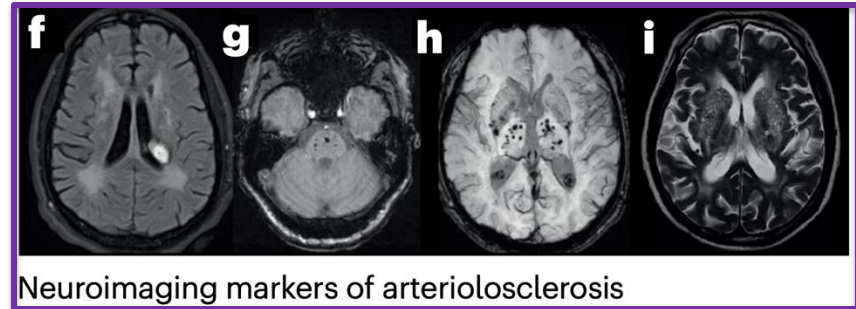
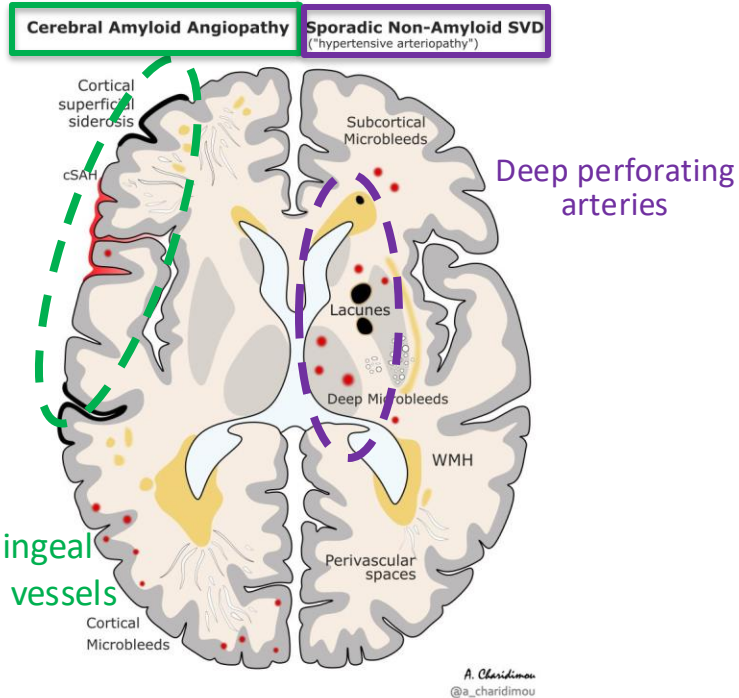
↑

↓

↔

1. Scientific context

- Arteriolosclerosis versus cerebral amyloid angiopathy





1. Scientific context

- **Boston criteria**

Boston criteria version 2.0 for sporadic cerebral amyloid angiopathy can be used to defining CSVD as **definite CAA** (with supporting pathology), **probable CAA** (with and without supporting pathology) and **possible CAA**.

The Boston criteria version 2.0 for cerebral amyloid angiopathy: a multicentre, retrospective, MRI-neuropathology diagnostic accuracy study

Andreas Charidimou, Gregoire Boulouis, Matthew P Frosch, Jean-Claude Baron, Marco Pasi, Jean Francois Albuchoer, Gargi Banerjee, Carmen Barbato, Fabrice Bonneville, Sebastian Brandner, Lionel Calviere, François Caparros, Barbara Casolla, Charlotte Cordonnier, Marie-Bernadette Delisle, Vincent Deramecourt, Martin Dichgans, Elif Gokcal, Jochen Herms, Mar Hernandez-Guillamon, Hans Rolf Jäger, Zane Jaunmuktane, Jennifer Linn, Sergi Martinez-Ramirez, Elena Martínez-Sáez, Christian Mawrin, Joan Montaner, Solene Moulin, Jean-Marc Olivot, Fabrizio Piazza, Laurent Puy, Nicolas Raposo, Mark A Rodrigues, Sigrun Roeber, Jose Rafael Romero, Neshika Samarasekera, Julie A Schneider, Stefanie Schreiber, Frank Schreiber, Corentin Schwall, Colin Smith, Levente Szalardy, Pascale Varlet, Alain Viguier, Joanna M Wardlaw, Andrew Warren, Frank A Wollenweber, Marialuisa Zedde, Mark A van Buchem, M Edip Gurol, Anand Viswanathan, Rustam Al-Shahi Salman, Eric E Smith, David J Werring, Steven M Greenberg

White matter lesions (T2-weighted images)

- ◆ Severe perivascular spaces (>20 in one hemisphere) in the centrum semiovale
- ◆ White matter hyperintensities (>10 in both hemispheres) in a multispot pattern

Lobar hemorrhagic lesions

- ◆ Intracerebral hemorrhage
- ◆ Cerebral microbleeds (susceptibility- or T2*-weighted images)
- ◆ Superficial siderosis (susceptibility- or T2*-weighted images)
- ◆ Convexity subarachnoid hemorrhage

^a Data from Charidimou A, et al, Lancet Neurol. ⁶⁴



1. Scientific context

- **Quantitative MRI in CSVD**



1. Scientific context

- **Quantitative MRI in CSVD**
 - R1 mapping
 - Advanced susceptibility imaging (R2* mapping or quantitative susceptibility mapping)
 - Magnetization transfer imaging
 - Advanced diffusion imaging



Neuroimaging standards for research into small vessel disease—advances since 2013

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1. Scientific context

- **7 Tesla MRI in CSVD**

- 7T MRI in CSVD has the potential to **improve visualization of the parenchymal lesions** :
 - ✓ Perivascular spaces : 7T MRI **increases the detection** of PvS and allows them to be visualized in greater detail than at 3T
 - ✓ CMB : 7T has been shown to be **more sensitive for detecting microbleeds** when compared to 1.5T
There is evidence to suggest that it is also superior to 3T
 - ✓ CMI : 7T MRI **can visualize cortical CMIs** that are often undetectable with conventional MRI
However, it detects only relatively large ones and likely **underestimates the true CMI burden**.
 - ✓ WMH : **7T MRI is comparable to 3T** in the depiction of WMHs and their distribution using T2-FLAIR



1. Scientific context

- **Diffusion imaging in CSVD : Diffusion tensor imaging (DTI) and neurite orientation dispersion and density imaging (NODDI)**
 - Show alteration in the **normal appearing white matter** (NAWM)
 - Multi-shell diffusion imaging like **NODDI** proves to be especially effective in identifying **subtle alterations in NAWM**
 - Advanced metrics from NODDI (*and DKI*) demonstrated strongest association with **cognitive deficits**



2. Questions, hypothesis, and experimental design

- **Hypothesis and questions**
 - High-resolution quantitative MRI at ultra-high field to reveal the spectrum of brain microstructure alteration within CSVD lesions and normal appearing brain tissue.



2. Questions, hypothesis, and experimental design

- **Primary endpoints**

- Quantify microstructural alterations in **normal-appearing grey matter (NAGM)** and **white matter (NAWM)** in comparison to age-matched controls.



2. Questions, hypothesis, and experimental design

- **Primary endpoints**

- Quantify microstructural alterations in **normal-appearing grey matter (NAGM)** and **white matter (NAWM)** in comparison to age-matched controls.
- Characterize the microstructure of **WMH** (deep and periventricular), **LPVO**, **CMB**, **CSS**.



2. Questions, hypothesis, and experimental design

- **Secondary endpoints**

- Assessing the **effect of age** and **CSVD** on size and burden of **PVS**



2. Questions, hypothesis, and experimental design

- **Secondary endpoints**

- Assessing the **effect of age** and **CSVD** on size and burden of **PVS**
- Relating microstructural and volumetric lesional load to **cognitive and motor performance**



2. Questions, hypothesis, and experimental design

■ Secondary endpoints

- Assessing the **effect of age** and **CSVD** on size and burden of **PVS**
- Relating microstructural and volumetric lesional load to **cognitive and motor performance**
- Manually **detecting CMIs** and establishing their typical **microstructural signature**



2. Questions, hypothesis, and experimental design

■ Secondary endpoints

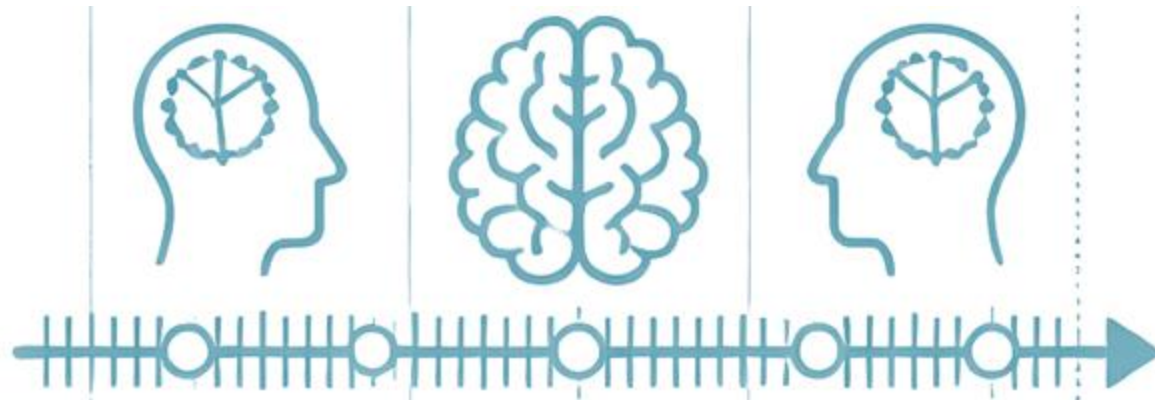
- Assessing the **effect of age** and **CSVD** on size and burden of **PVS**
- Relating microstructural and volumetric lesional load to **cognitive and motor performance**
- Manually **detecting CMIs** and establishing their typical **microstructural signature**
- Establishing the impact of white matter lesions on the **microstructure of the upstream cortex** (cortex connected by afferent or efferent fibers passing through the lesion) and **downstream fiber tracts** (such as the corticospinal tract).



2. Questions, hypothesis, and experimental design

- **Study design**

This study follows a **prospective cross-sectional single time-point observational design without specific therapeutic intervention.**





3. Data acquisition & subjects

- **Participants**

1. **Adult patients** admitted or followed at the CHU Liège, whose routine MRI scan shows **signs of CSVD** (based on STRIVE-2).
 - **Early stages** (e.g., non-confluent WMH) to **severe stages** (e.g., confluent WMH with LPVO and CMB).
2. A sample of **healthy controls** whose routine MRI scan will **not show any sign suggesting CSVD**.
 - In a **1:3 proportion** relative to the patient population



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2. A sample of **healthy controls** whose routine MRI scan will **not show any sign suggesting CSVD**.
 - In a **1:3 proportion** relative to the patient population

The goal is to scan approximately **100** participants.



3. Data acquisition & subjects

- **Exclusion criteria**
 - **Other major cause of stroke** (intra-/extra-cranial large vessel disease, cardioembolic source of stroke)
 - **CSVD due to another cause** (Genetic SVD)
 - **Recent disabling stroke** (3 months)
 - **History of major brain pathology** (head trauma, neoplasm +/- brain radiotherapy, central nervous system inflammatory/infectious disease, neurodegenerative disease, neurosurgical intervention)
 - **7T MRI scan contraindication**



3. Data acquisition & subjects

- **Medical consultation**
 - **Demographics and medical history** : sex, age, education level, hypertension, diabetes, dyslipidemia, OSAS, smoking habits, vascular events, and ongoing treatments
 - **Family history** of stroke and/or cognitive impairment



3. Data acquisition & subjects

- **Medical consultation**
 - **Demographics and medical history** : sex, age, education level, hypertension, diabetes, dyslipidemia, OSAS, smoking habits, vascular events, and ongoing treatments
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 - **Clinical assessment** : NIHSS, mRS, MOCA, grip strength, 25-feet walking test, 9-hole peg test



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 - **Family history** of stroke and/or cognitive impairment
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 - **Characteristics of CSVD on conventional MRI**



3. Data acquisition & subjects

- **Data acquisition**



3. Data acquisition & subjects

- **Data acquisition**
 - **Multiparameter mapping protocol (MPM)** : three co-localized 3D multi-echo fast low angle shot (FLASH) acquisitions at $600\ \mu\text{m}^3$ resolution.
 - **MT saturation (MTsat), $R2^*$, $R1$, PD and QSM quantitative** maps will be estimated using hMRI toolbox
 - Neurite orientation dispersion and density imaging (**NODDI**)



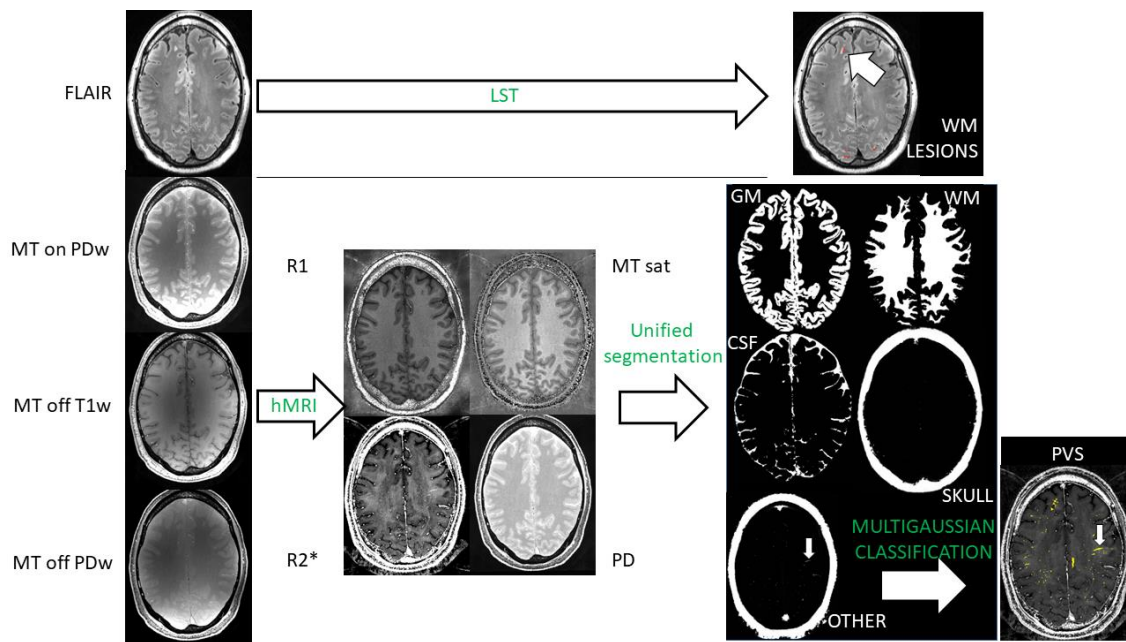
3. Data acquisition & subjects

- **Data acquisition**
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 - **MT saturation (MTsat), $R2^*$, $R1$, PD and QSM quantitative** maps will be estimated using hMRI toolbox
 - Neurite orientation dispersion and density imaging (**NODDI**)
 - **FLAIR**
 - TOF ? DIR ? MP2RAGE ?
 - Estimate duration 60 min



4. Data processing & inference

- Segmentation





4. Data processing & inference

- **Segmentation**

1. An automatic segmentation based on FLAIR can delineate **WMH**

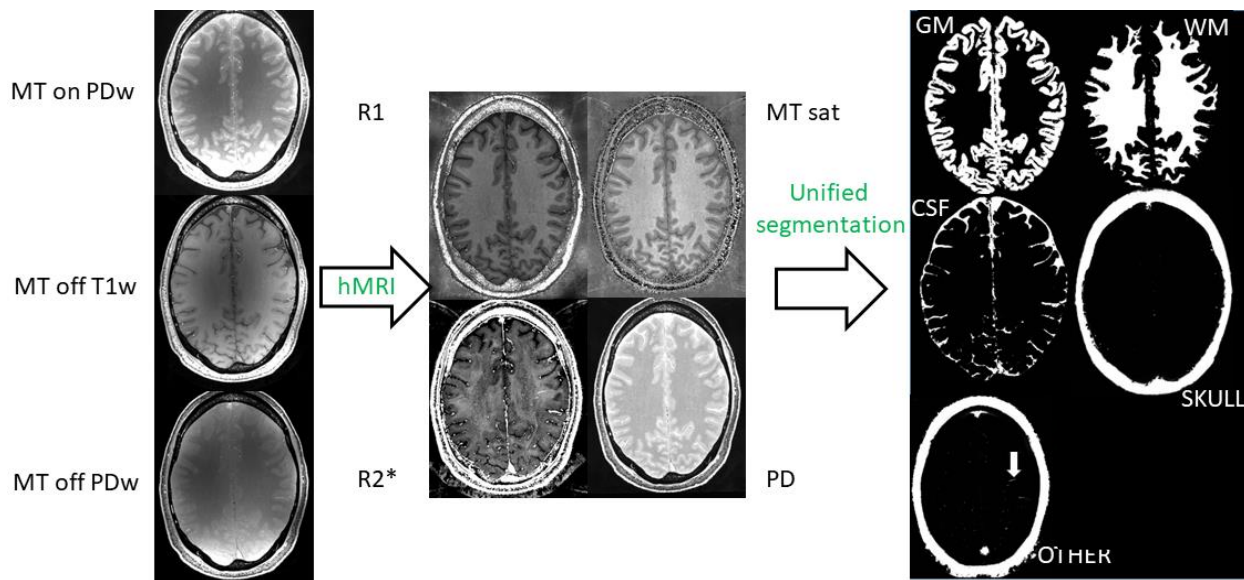




4. Data processing & inference

- **Segmentation**

2. Quantitative maps are segmented using the ‘unified segmentation’ to provide the distinction between **WM**, **GM**, **CSF**, **SKULL** and **Other**.

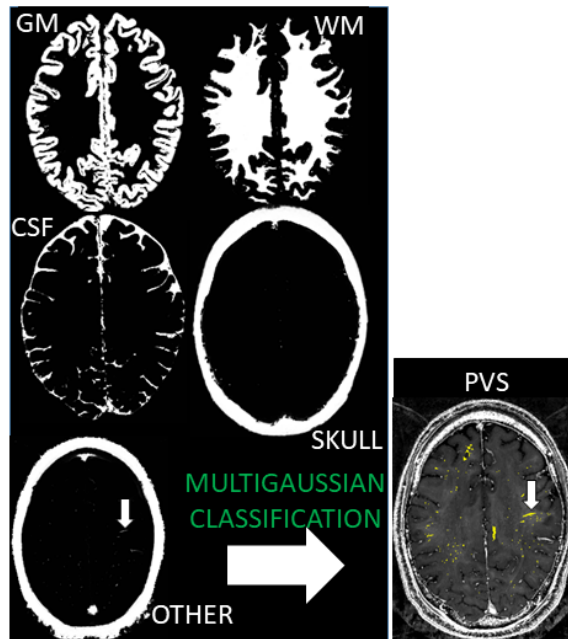




4. Data processing & inference

- **Segmentation**

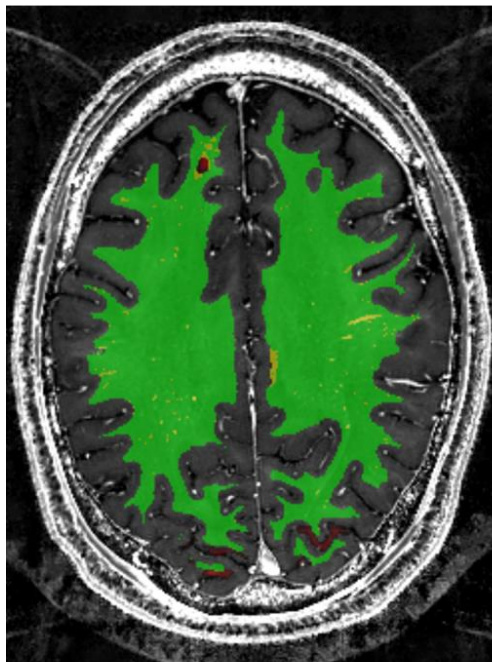
3. An automatic segmentation based on 'other' can delineate **PVS**





4. Data processing & inference

- **Segmentation**





4. Data processing & inference

- **Cortical microinfarcts**
 - Careful manual detection **3 independent and blind experts**
 - These cortical lesions will be excluded of the **cortical voxels of the NAGM**



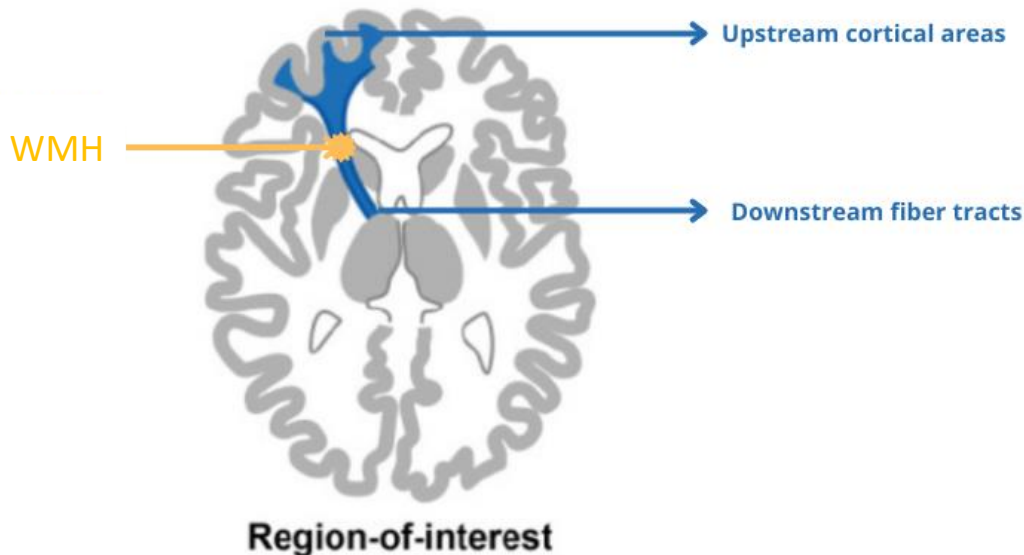
4. Data processing & inference

- **Segmentation**
 - These various segmentation steps will allow to **define several tissue classes** (NAWM, NAGM, WMH, LPVO, CMB, CSS, CMI) from **which median quantitative values of R1, R2*, PD, MT, QSM, NODDI will be extracted.**



4. Data processing & inference

- **Tract reconstruction**
 - **Diffusion** data will allow **tract reconstruction** and the identification of fiber tracts interrupted by **SVD lesions**, thereby the **upstream cortical areas** and **downstream fiber tracts**.





4. Data processing & inference

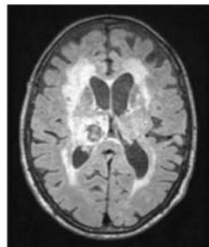
- **3 groups**

- Group 1 : **Healthy controls**



4. Data processing & inference

- **3 groups**
 - Group 1 : **Healthy controls**
 - Group 2 : **Arteriolosclerosis**
 - ✓ MRI features of arteriolosclerosis





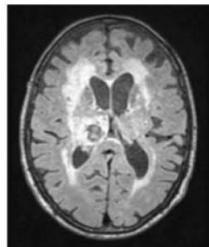
4. Data processing & inference

- **3 groups**

- Group 1 : **Healthy controls**

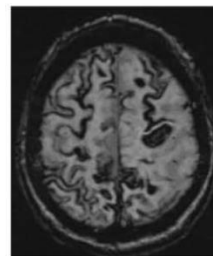
- Group 2 : **Arteriolosclerosis**

- ✓ MRI features of arteriolosclerosis



- Group 3 : **Cerebral amyloid angiopathy**

- ✓ Responding to Boston criteria 2.0. for probable CAA





4. Data processing & inference

- **Statistics**
 - **Generalized linear mixed models**, with participant intercept and slope as random variables, and age, sex, education as fixed effects factors, will be designed to extract the group effect, and probed the questions detailed above
 - Similar models can accommodate clinical scores to search for the **clinical correlates of brain microstructural changes**.
 - We reasoned that scanning **100 participants** would provide us with ~90 useful data



5. Expected results and interpretation

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 - We expect an **additive effect of age and CSVD on PVS size and burden.**



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- Microstructural alterations will correlate with **motor or cognitive performance**.
- We will establish the typical **microstructural signature of CMIs**
- **Cortex connected by afferent or efferent fibers** interrupted by CSVD lesions will show signs of **decreased myelination** (decreased $R2^*$, MTsat, neurite orientation). A similar observation should be observed in **downstream fiber tracts**, due to Wallerian degeneration.



6. Project management

- **Team:**
 - **Promoter:** Christine Bastin
 - **Co-Promoter:** Pierre Maquet
 - **Justin Baudru:**
 - MRI scan (qualification is ongoing)
 - Recruitment and medical consultations
- **Collaboration with the neurovascular team of CHU Liège :** J. LY, G. Dasnoy and F. Delvoye



7. Project administration

- **A few legal and administrative points**
 - **Funding Source:** Queen Elisabeth Medical Foundation
 - **Ethical Approval:** CHU Liège Ethical Committee

Thank you for your attention!