Advanced MR image analysis in sporadic and Dutch-type hereditary Cerebral Amyloid Angiopathy

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Chapter 7 | Hyperintense lesions suspect for acute cerebral microbleeds on ultra-high field T1 weighted 7 Tesla MRI in patients with cerebral amyloid angiopathy

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Abstract

Objective

The aims of this study are to describe T1 hyperintense lesions on 7T MRI in patients with hereditary Dutch-type (D-CAA) and sporadic cerebral amyloid angiopathy (sCAA) and their detection rate on 7T and 3T MRI.

Methods

We included D-CAA mutation carriers and patients with sCAA who participated between 2018 – 2023 in our natural history studies in D-CAA (AURORA) and sCAA (FOCAS). We assessed T1-weighted 7T MRI for small (<10mm) hyperintense lesions and T1-weighted 3T MRI for corresponding lesions. We classified them as possible acute (3-7 days after onset) CMBs when exhibiting characteristics of CMBs on the T2*-weighted gradient echo sequence (GRE) 7T and/or susceptibility weighted imaging (SWI) 3T MRI. Follow-up T1-weighted 7T and 3T MRI and T2*-weighted GRE 7T and/or SWI 3T MRI were assessed to confirm the hemorrhagic origin of 7T T1 hyperintense lesions found at baseline.

Results

Baseline 7T MRI and 3T MRI were available in 112 participants. We found 16 T1 hyperintense lesions on 7T in 12 participants (11%). Of the 16 lesions, 10 (63%) were subsequently identified on T1-weighted 3T MRI. In patients with available follow-up imaging, 11 out of 15 lesions (73%) were classified as acute CMBs based on 7T and/or 3T follow-up imaging.

Conclusion

7T T1 hyperintense lesions were identified in approximately one in ten patients in our cohort. Acute CMBs were more often identified on 7T MRI in comparison to 3T MRI. This highlights the additional value of ultra-high field MRI in the assessment of CAA-related MRI markers.

Introduction

Cerebral Amyloid Angiopathy (CAA) is a common cause of lobar intracerebral hemorrhage (ICH) and vascular dementia in older adults.[2, 208] Among the MRI markers associated with CAA, lobar cerebral microbleeds (CMBs) are well-known.[209-213] CMBs occur due to amyloid deposition in cortical and leptomeningeal vessels, leading to decreased vessel wall integrity and rupture.[213, 214] Unlike ICH, CMBs usually do not cause acute symptoms and often go unnoticed at the time of occurrence.

Ultra-high-field 7 Tesla Magnetic Resonance Imaging (7T MRI) enables improved visualization of intracerebral abnormalities compared to lower field strengths.[215] Several novel imaging markers associated with CAA have been described on 7T MRI, including intragyral hemorrhage and the striped cortex sign.[216] In a patient with CAA, we observed a small (< 10 mm) hyperintense lesion on T1-weighted 7T MRI. On susceptibility-weighted imaging (SWI) from a 3 Tesla (3T) scan acquired the same day, the lesion showed characteristics of a CMB (i.e. a hypointense lesion showing a blooming effect). Notably, the lesion was not visible on the 3T T1-weighted sequence.

Based on the hyperintensity on T1-weighted sequence and blooming hypointensity on T2*-weighted sequence, we interpreted this lesion as an acute CMB of approximately 3-7 days old (acute to early subacute stage).[217, 218] A similar lesion has been described previously, showing hyperintensity on both 1.5T T1-weighted and T2 weighted fluid-attenuated inversion recovery (FLAIR) imaging, which was estimated to be 7-28 days old (late subacute stage).[219] These observations raise the question how frequently such T1 hyperintense lesions occur in the context of CAA and whether 7T MRI improves their detection compared to 3T MRI. Novel MRI markers, such as acute CMBs, may offer insights into the timing and mechanisms of CAA-related microvascular injury and might in the future help in risk stratification and clinical management.

The aims of this study are therefore twofold: 1) to describe the characteristics of T1 hyperintense lesions on 7T MRI and frequency in our cross-sectional sample of patients with hereditary Dutch-type (D-)CAA and sporadic CAA (sCAA) and 2) to compare the detection rate of these lesions between 7T and 3T MRI.

Material and methods

Patient Selection

We included D-CAA mutation carriers and patients with sCAA who participated between 2018 – 2023 in our natural history studies on disease progression and biomarkers in D-CAA (AURORA) and sCAA (FOCAS).[181] Participants underwent yearly routine follow-up with overall health questionnaires, physical examination, as well as 7T and 3T MRI. From these studies we selected participants in whom a 7T MRI as well as a 3T MRI were performed at baseline and were acquired on the same day.

All D-CAA participants were recruited through the (outpatient) clinic of the Leiden University Medical Center (LUMC). D-CAA is one of few hereditary subtypes of CAA in the world. D-CAA is caused by inheritance of the autosomal dominant point mutation at codon 693 of the amyloid precursor protein (APP) gene that results in amyloid-β deposition in the cortical and

leptomeningeal vessels of the brain.[179, 220] Biochemical, pathological, and radiological disease characteristics are similar between D-CAA and sCAA, although in D-CAA the disease progresses more quickly, and presents at a younger age when the influences of age related comorbidities and vascular risk factors are still limited.[88] Persons with D-CAA were included when they were ≥18 years of age and had a DNA proven APP mutation. D-CAA mutation carriers were classified as pre-ICH when they did not have a history of symptomatic ICH. Participants with sCAA were included from the (outpatient) clinic of the LUMC and diagnosed by an experienced vascular neurologist and neuroradiologist based on the modified Boston criteria for probable CAA.[221]

The AURORA and FOCAS studies were approved by the Medical Ethical Committee of the Leiden University Medical Center and written informed consent was obtained from all study participants before enrollment.

MRI Analysis

MRI protocols for 3T and 7T have been described previously and are shown in Supplementary Material 1.[181] Both 7T and 3T MRI scans were independently reviewed by two raters (MRS and SV), who were blinded to all clinical data. Firstly, we assessed T1-weighted images on 7T for small (< 10 mm) hyperintense lesions. Secondly, we assessed the T1-weighted 3T imaging for corresponding lesions. We classified them as possible acute (3-7 days after onset) CMBs when exhibiting characteristics of CMBs (i.e. blooming effect) on the T2*-weighted gradient echo sequence (GRE) 7T and/or SWI 3T MRI.[210, 217, 218] Discrepancies between the two raters were discussed with a neuroradiologist with over 20 years of experience in the field (MAAvW) to reach a consensus rating. For explorative analysis, baseline MRI markers, such as CMBs, cortical superficial siderosis (cSS), white matter hyperintensities (WMHs) and enlarged perivascular spaces (EPVS) were assessed by trained scorers according to the STRIVE criteria.[222]

Follow-up analysis

To further verify the hemorrhagic origin of lesions found at baseline, all participants with 7T T1 hyperintense lesions were subjected to follow-up analysis of 7T and 3T MRI scans. The follow-up time between baseline and follow-up scans was recorded. Lesion characteristics were examined on the available follow-up T1-weighted 7T and 3T MRI and T2*-weighted GRE 7T and/or SWI 3T MRI, depending on available follow-up scans.

Statistical analysis

We analyzed demographic characteristics of D-CAA and sCAA populations with descriptive statistics. The first part of the analysis was to exploratively analyze differences in baseline ICH and 3T CAA-related MRI markers, such as the number of CMBs, presence of cSS, presence of WMHs and severity of EPVS (0= no EPVS, 1= 0-10 EPVS, 2=10-20 EPVS, 3= 20-40 EPVS, 4= >40 EPVS). Therefore, we reported these markers for participants in whom 7T T1 hyperintensities were found, as well as for participants in whom no 7T T1 hyperintensities were found. We reported the proportion of participants with 7T T1 hyperintensities and the number of 7T T1 hyperintensities including 95% confidence intervals (CI), as well as the proportion of participants with T1 hyperintensities and the number of T1 hyperintensities also visible on 3T. We reported the follow-up characteristics of lesions, as well as a total proportion of

lesions verified to be of hemorrhagic origin at follow-up. In addition, we calculated a Cohen's kappa to assess interrater agreement on T1 hyperintensities for both 7T and 3T MRI after the two experienced raters analyzed the lesions. Proportions were compared using the Chi-squared (χ^2) test. Continuous variables were compared using the Student's t-test. All data were analyzed with IBM SPSS Statistics version 25 (IBM Corp., Armonk, NY, USA).

Results

In total 112 participants underwent both a 7T MRI as well as a 3T MRI at baseline; 29 pre-ICH D-CAA mutation carriers (mean age 41 years, 66% women), 28 D-CAA mutation carriers with ICH (mean age 59 years, 46% women) and 55 participants with sCAA (mean age 71 years, 40% women) Thirty-two (58%, 95% CI: 20-38%) of the participants with sCAA had a history of ICH, 24 (22%, 95% CI: 14-30%) presented with TFNEs and three (6%, 95% CI: 1-10%) with cognitive decline (Table 1). Specific reasons for exclusion are outlined in Figure 1.

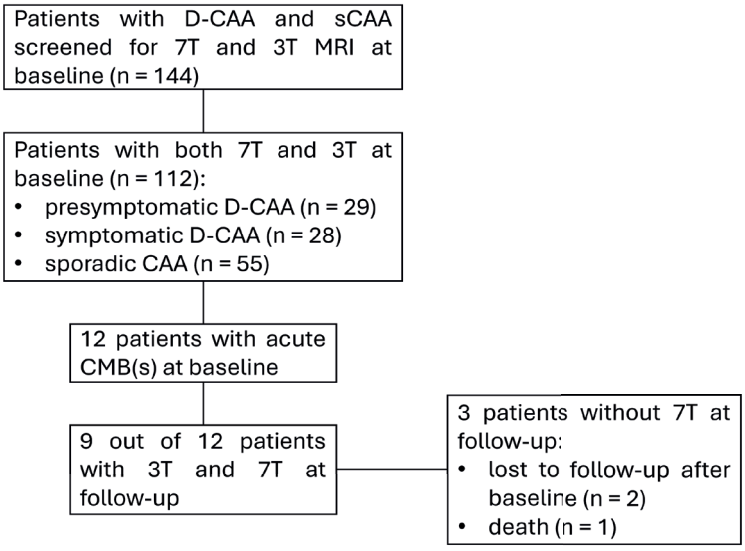

Figure 1. Flow chart of patient inclusion.

Abbreviations. D-CAA; Dutch-type Cerebral Amyloid Angiopathy. sCAA; sporadic Cerebral Amyloid Angiopathy, 7T; 7 Tesla. 3T; 3 Tesla. CMBs; cerebral microbleeds.

Table 1. Baseline characteristics and the presence of 7T and 3T T1 hyperintensities

	Pre-ICH D-CAA mutation carriers (n=29)	D-CAA mutation carriers with ICH (n=28)	Patients with sporadic CAA (n=55)
Mean age (range, SD) [y]	41 (26-68, 10)	59 (47-74, 7)	71 (58-86, 6)
Women (%)	19 (66)	13 (46)	22 (40)
Hypertension (%)	4 (14)	9 (32)	27 (49)
History of symptomatic ICH (%)	0	28 (100)	32 (58)
Number of patients with 7T T1 hyperintensities (%)	1 (3)	6 (21)	5 (9)
Number of total 7T T1 hyperintensities	1	9*	6
Number of patients with 3T T1 hyperintensities (%)	1 (3)	5 (18)	2 (4)
Number of total 3T T1 hyperintensities	1	7**	2

* Four acute CMBs in one patient. ** Three acute CMBs in one patient; in the same patient as *

Abbreviations. 7T; 7 Tesla. 3T; 3 Tesla. CMBs; cerebral microbleeds. D-CAA; Dutch-type Cerebral Amyloid Angiopathy. ICH; Intracerebral hemorrhage. IQR; interquartile range. TFNE; Transient focal neurological episode(s). SD; standard deviation.

We found 16 T1 hyperintense lesions on 7T in 12 participants (11%, 95% CI: 5-12%); one pre-ICH D-CAA mutation-carrier with only pre-existing white matter hyperintensities, six D-CAA mutation-carriers with ICH and five participants with sCAA. (Figure 2 and 3). Of the 16 lesions, 10 (63%, 95% CI: 35-84%) were subsequently identified on T1-weighted 3T MRI (Table 1). The Cohen's kappa between the two raters was 0.25 (95% CI: -0.03 – 0.54) for 7T T1 hyperintensities and 0.33 (95% CI: -0.04 – 0.70) for 3T T1 hyperintensities (Supplementary Material 2).

7 Tesla and 3 Tesla MRI imaging of an acute CMB (confirmed on follow-up imaging) in a patient with sporadic CAA. A) 7 Tesla T1-weighted image, showing an acute CMB (red arrow). In addition, an acute intracerebral hemorrhage (> 10 mm) is visible in left occipital lobe. B) 3 Tesla T1-weighted image, showing the corresponding acute CMB C) 7 Tesla T2*-weighted gradient echo image, showing the acute CMB as a hypointensity with blooming effect D) 3 Tesla SWI image also showing a hypointensity with blooming effect. (CMB; cerebral microbleed. SWI; susceptibility-weighted imaging. GRE; gradient echo.)

Participants with 7T T1 hyperintensities at baseline more often had a history of symptomatic ICH (83% vs. 50% in participants without T1 hyperintensities, 95% CI: 10-56%, p=0.031). In an explorative analysis of other CAA-related MRI markers, there was no substantial difference in number of CMBs (median 268 vs. 143, 95% CI: -284 – 34, p=0.123), presence of cSS (67% vs. 49%, 95% CI: -11-46%, p=0.247), presence of WMH (100% vs. 94%, 95% CI: 1.4 – 11%, p=0.383), and severity of EPVS (median score 4 vs. 4, p=0.638) between participants with and without 7T T1 hyperintensities (Table 2).

Follow-up 7T scans were available in 9 of the 12 participants and follow-up 3T scans in all 12 participants with one or more hyperintense lesion(s). 7T T2*-weighted GRE imaging was available for 8 out of 12 participants, and 3T SWI imaging in all 12 participants. A total of 12 T1 lesions on 7T and all 15 lesions on 3T at follow-up were examined. The median time between scans for 7T was 24 months (95% CI: 22-29 months) and 12 months (95% CI: 11-16 months) for 3T (Figure 1). For the specific available scan sequences and follow-up time per participant, see Supplementary Material 3. On follow-up imaging, one lesion was hypointense and showed blooming effect typical of hemorrhage on 7T T2*-weighted GRE and 3T SWI; 10 lesions were hypointense and showed blooming effect typical of hemorrhage on 3T SWI only (no 7T-GRE available for 3 lesions; 3 lesions invisible on 7T T2* GRE; 7T GRE uninterpretable for 4 lesions). In total, 11 out of 15 lesions (73%, 95% CI: 45-92%) were classified as acute CMBs based on 7T and/or 3T follow-up imaging. For an overview of all lesion characteristics, see Supplementary Material 3.

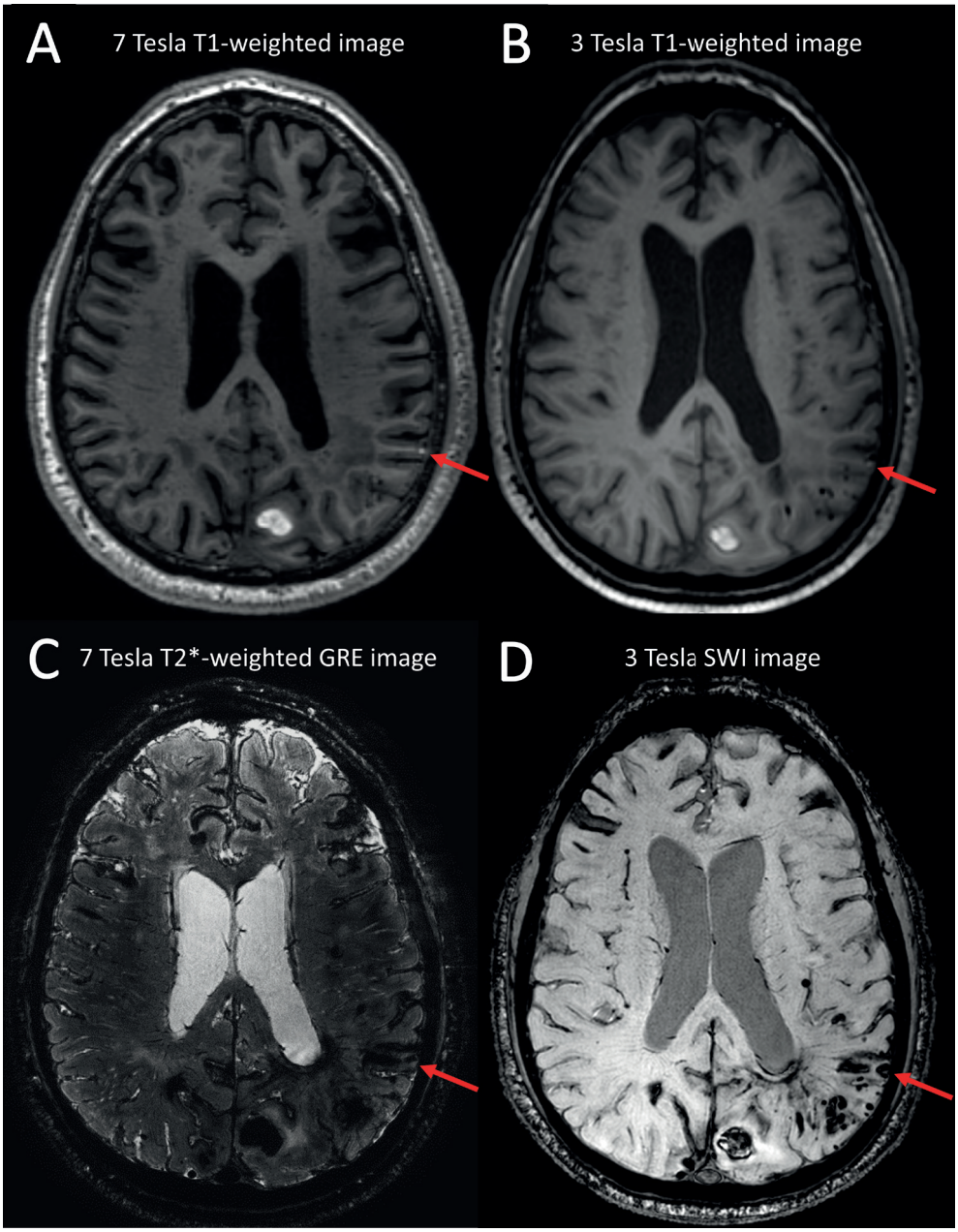

Figure 2. 7 Tesla and 3 Tesla MRI imaging of an acute CMB (confirmed on follow-up imaging) in a patient with sporadic CAA. A) 7 Tesla T1-weighted image, showing an acute CMB (red arrow). In addition, an acute intracerebral hemorrhage (> 10 mm) is visible in left occipital lobe. B) 3 Tesla T1-weighted image, showing the corresponding acute CMB C) 7 Tesla T2*-weighted gradient echo image, showing the acute CMB as a hypointensity with blooming effect D) 3 Tesla SWI image also showing a hypointensity with blooming effect.

Abbreviations. CMB; cerebral microbleed. SWI; susceptibility-weighted imaging. GRE; gradient echo.)

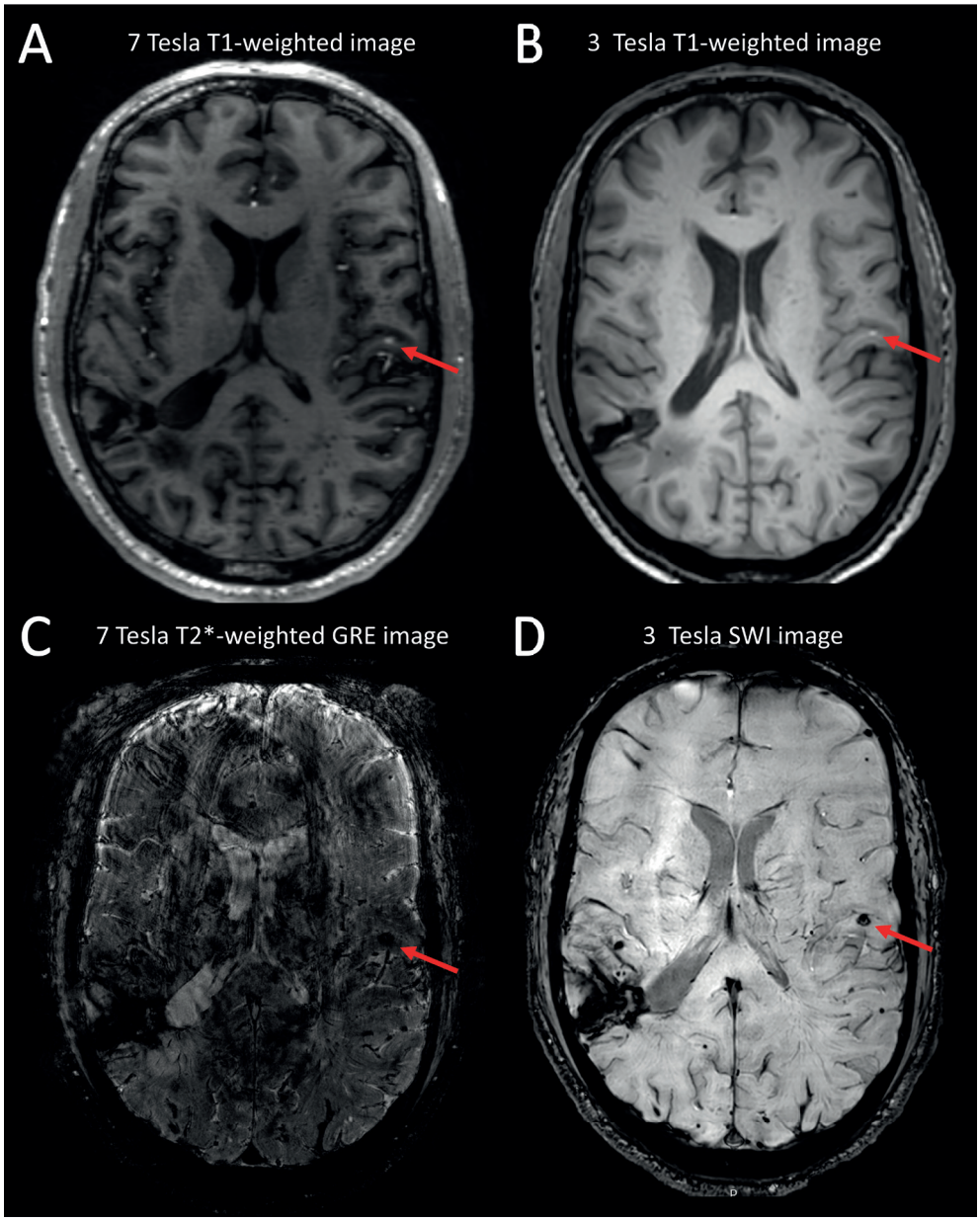

Figure 3. 7 Tesla and 3 Tesla MRI imaging of an acute CMB (confirmed on follow-up imaging) in a D-CAA mutation-carrier with ICH. A chronic ICH is visible in the right hemisphere. A) 7T T1-weighted image showing an acute CMB (red arrow), B) 3 Tesla T1-weighted image, showing the corresponding acute CMB C) 7T T2*-weighted gradient echo image, showing a hypointensity; note that image quality was poor, but blooming effect could still be appreciated D) 3T SWI image showing a hypointensity with blooming effect.

Abbreviations. CMB; cerebral microbleed. SWI; susceptibility-weighted imaging. GRE; gradient echo.

Table 2. 3T MRI characteristics of patients with and without 7T T1 hyperintensities at baseline

	With 7T T1 hyperintensities (N=12)				Without 7T T1 hyperintensities (N=100)			
	Pre-ICH D-CAA mutation carriers (N=1)	D-CAA mutation carriers with ICH (N=6)	Patients with sCAA (N=5)	Total	Pre-ICH D-CAA mutation carriers (N=28)	D-CAA mutation carriers with ICH (N=22)	Patients with sCAA (N=50)	Total
Number of total CMBs on 3T, median (IQR)	0 (0)	302 (266)	280 (433)	268 (132)	45 (3)	112 (362)	127 (117)	143 (271)
Presence of cSS on 3T (%)	0 (0)	6 (100)	2 (40)	8 (67)	3 (11)	15 (68)	30 (60)	48 (49)
Presence of WMHs (%)	1 (100)	6 (100)	5 (100)	12 (100)	22 (79)	22 (100)	50 (100)	94 (94)
Severity score of EPVS, median (IQR)	4 (4)	3.5 (2)	4 (0)	4 (1)	3 (2)	4 (0)	4 (1)	4 (1)

Note. EPVS severity score: 0 = no EPVS, 1 = 0-10, 2=10-20, 3=20-40, 4 = >40 EPVS.

Abbreviations. 7T; 7 Tesla. ICH; Intracerebral hemorrhage. D-CAA; Dutch-type Cerebral Amyloid Angiopathy. sCAA; sporadic Cerebral Amyloid Angiopathy. CMBs; cerebral microbleeds. 3T; 3 Tesla. IQR; interquartile range. cSS; cortical superficial siderosis. WMH; white matter hyperintensities. EPVS; enlarged perivascular spaces.

Discussion

In our study, T1 hyperintense lesions on 7T MRI suggestive of acute CMBs were detected in 11% of participants in our cohort of patients with D-CAA and sCAA. These lesions appeared almost twice as frequently on 7T MRI as compared to 3T MRI. Of the 15 identified lesions at follow-up, 11 were classified as acute CMBs; for four lesions, this could not be confirmed with certainty.

To our knowledge, this is the first study to systematically describe T1 hyperintense lesions on 7T MRI suggestive of acute CMBs in a large cohort of both hereditary Dutch-type CAA (D-CAA) and sporadic CAA (sCAA). Our findings are in line with prior research demonstrating the superior sensitivity of 7T MRI for detecting CMBs as compared to 3T MRI..[216, 223-228] This research advances the understanding of CAA by identifying imaging markers suggestive of acute or early subacute cerebral microbleeds (CMBs), indicating periods of active vascular injury. This supports the concept of CAA as a dynamic disease marked by episodic vessel leakage rather than a static process.[180, 220, 229] These findings primarily contribute to the conceptual understanding of CAA. In future studies, they may help refine approaches to risk stratification and trial design by informing hypotheses on disease timing, mechanisms, or potential subgroups of interest. Awareness of cerebral microbleeds may assist clinicians in managing patients on anticoagulation therapy or those at increased risk of intracerebral hemorrhage.

Hemorrhagic markers are central to the diagnosis of CAA under the modified Boston criteria.[230] However, the sensitivity of the criteria remains limited particularly for patients without ICH who present with TFNEs or cognitive decline.[230] Detecting acute hemorrhagic markers, such as acute CMBs, could enhance early diagnosis in atypical cases without overt hemorrhagic symptoms and may hold future value guiding patient risk stratification. Therefore, improving imaging detection methods for novel markers has the potential to improve clinical care and outcomes. However, clinical utility of 7T MRI is constrained by limited availability and is therefore mainly confined to research settings. Lesions suspected to represent acute CMBs were also detectable on 3T MRI, though less frequently. Our protocol did not include a direct comparison with 1.5T MRI, which remains the clinical standard in many centres. This limits the ability to directly relate our findings to routine clinical practice. Importantly, the primary aim of this study was to gain pathophysiological insights rather than to establish immediate clinical applicability. Future studies including 1.5T MRI will be essential to clarify the added value of higher field strengths for detecting CAA-related markers such as acute CMBs. The impact of this study on clinical practice remains limited until comparative data with 1.5 Tesla MRI becomes available.

A major strength of our study is the well-characterized cohort of patients with CAA, uniquely including patients with Dutch-type hereditary CAA. Moreover, both 3T and 7T MRI were performed on the same day and the same protocol was scanned in all participants. However, several limitations should be noted. First, not all participants were able or willing to undergo 7T MRI, which may have led to selection bias toward participants in better clinical condition. Second, the interrater agreement for identifying acute CMBs, particularly on 7T MRI, was poor. The lower-than-expected agreement may be explained by the high prevalence of negative ratings, which artificially inflate percentage agreement but deflate Cohen's Kappa, as seen in our results. Other contributing factors to disagreement may include lesion size, contrast-to-noise ratio and variability in anatomical location. Although, in our study, consensus was

reached between raters in discrepant cases with help of an experienced third rater, the low interrater agreement highlights the need for experience and development of structured assessment protocols. Standardization would be essential for validating T1 hyperintense lesions in a research and clinical setting.

Third, we relied on blooming artifacts on 7T GRE and 3T SWI sequences to support a hemorrhagic origin, while no histopathological analysis was performed to confirm. While these MRI artifacts typically indicate a hemorrhagic origin, they can also result from calcium deposits, vessel flow voids or volume artifacts from bone.[212] Moreover, prior studies have shown that CMBs observed on MRI are histopathologically heterogeneous.[231, 232] cortical CMBs seen on MRI may represent acute or chronic microhemorrhages, vasculopathies, or hemorrhagic microinfarcts. Also, involved vessels are not always visible on histopathological analysis, complicating histopathological interpretation.[233] Lastly, our interpretation of these lesions as acute (3-7 days; acute to early subacute stage) was based on transiently increased signal intensity on T1-weighted MRI and hypointense signal on T2*-weighted imaging, consistent with previous literature, but other sequences such as T2-weighted FLAIR imaging were not assessed. In previous literature describing acute CMBs in CAA, T2-weighted FLAIR imaging also showed hyperintensity.[219] Based on our assessment, our lesions could have occurred earlier, but no later than the previous report. However, exact timing of these lesions at baseline remains uncertain. Assessing the timing of such lesions might be important for future research.

Conclusion

We describe 7T T1 hyperintense lesions suspect for acute CMBs present in our cohort of patients with D-CAA and sCAA. These lesions were visible in approximately one in ten patients in our cohort. The increased identification of acute CMBs on 7T MRI in comparison to 3T MRI highlights the additional value of ultra-high field MRI in the assessment of CAA-related MRI markers. Future research is necessary to investigate their relationship with other CAA-related markers detectable on 7T and 3T MRI, as well as their impact on clinical outcomes.

Supplemental materials to Chapter 7

Supplementary Material 1. MRI protocols

3 Tesla MRI protocol

MRI was performed on a whole-body 3.0 Tesla MRI scanner (Philips Achieva, Best, the Netherlands) with a standard 32-channel head coil in all D-CAA and sCAA participants. Three-dimensional T1 weighted images were acquired with the following parameters: repetition time(TR)/ echo time (TE): 9.7/4.6ms, flip angle 7°, 130 slices with no interslice gap and a field of view (FOV) of 217 x 172 x 156 mm with a voxel size of 1.2 x 1.2 x 1.2 mm, resulting in a scan duration of 2:48 min. Susceptibility weighted images (SWI) were acquired using the following parameters: TR/TE: 31/7.2ms, flip angle 17°, 130 slices and an FOV of 230 x 190 x 130 mm with a voxel size of 0.6 x 0.6 x 1 mm resulting in a scan duration of 3:31 min.

7 Tesla MRI protocol

For all participants MRI was performed on a whole-body human 7 Tesla-MR-system (Philips, Best, The Netherlands) using a quadrature transmit and 32-channel receive head coil (Nova Medical, Wilmington, MA, USA) in Leiden, The Netherlands. All participants were scanned using a 3D T1-weighted scan TR/TE: 4.3/1.9 ms with a flip angle of 7°, 193 slices with no interslice gap, a FOV of 246 x 246 x 174 with a voxel size of 0.9 x 0.9 x 0.9 mm. A 2D flow-compensated transverse T2*-weighed gradient echo sequence was acquired using the following parameters: TR/TE: 1851/25 ms, flip angle 60°, slice thickness 1.0 mm with a 0.1 mm interslice gap, 92 slices and a FOV of 240 x 180 x 100 mm with an in-plan matrix size of 1000 x 751, resulting in an in-plane spatial resolution of 0.24 x 0.24 mm, multiband factor 2.

Supplementary Material 2. Cohen's Kappa calculation

Figure 1. the Cohen's Kappa for T1 hyperintensities on 7T MRI.

		Scorer 1		
		No	Yes	Total
Scorer 2	No	96	7	103
	Yes	6	3	9
	Total	102	10	112

Figure 2. the Cohen's Kappa for T1 hyperintensities on 3T MRI.

		Scorer 1		
		No	Yes	Total
Scorer 2	No	103	3	106
	Yes	4	2	6
	Total	107	5	112

Supplementary Material 3. Patient and imaging characteristics of participants with 7T acute CMBs at baseline

Participant	Age, y*	Sex	Hypertension	Diagnosis	Baseline 7T MRI characteristics	CMB location on 7T MRI	Basline 3T MRI characteristics	Follow-up time 7 MRI, months	Follow-up 7T MRI characteristics	Follow-up time 3T MRI, months	Follow-up 3T MRI characteristics
1	40-50	F	No	Pre-ICH D-CAA	Hyperintensity visible, on GRE hypointensity with blooming effect	Medial of posterior horn lateral ventricle	Also hyperintense on 3T, on SWI hypointensity with blooming effect	24	no GRE available	12	Hypointense lesion with blooming effect on SWI
2	70-80	F	No	D-CAA with ICH	Hyperintensity visible, location not visible on GRE	Medulla oblongata	Not visible on 3T or 3T SWI	NA	No 7T follow-up	12	Invisible on SWI
3	60-70	F	No	D-CAA with ICH	Hyperintensity visible, on GRE hypointensity with blooming effect	Temporal	Also hyperintense on 3T, on SWI hypointensity with blooming effect	NA	No 7T follow-up	18	Hypointense lesion with blooming effect on SWI
4	60-70	F	Yes	D-CAA with ICH	4 hyperintensities visible, on GRE two hypointensities with blooming effect, two not interpretable due to hemorrhage	Occipital	3 out of 4 also hyperintense on 3T, on SWI 3 hypointensities with blooming effect	24	4 hypointense lesions, GRE blooming uninterpretable	13	3 hypointense lesions with blooming effect on SWI
5	50-60	M	No	D-CAA with ICH	Hyperintensity visible, on GRE hypointensity with blooming effect	Occipital	Also hyperintense on 3T, on SWI hypointensity with blooming effect	22	Hypointense lesion, no blooming effect on GRE	12	Hypointense lesion with blooming effect on SWI
6	60-70	M	No	D-CAA with ICH	Hyperintensity visible, GRE difficult to interpret, some blooming effect visible	Brain stem	Not visible on 3T or 3T SWI	25	GRE not interpretable	12	Invisible on SWI

7	60-70	F	Yes	D-CAA with ICH	Hyperintensity visible, on GRE hypointensity with blooming effect	Centrum semi-ovale	Also hyperintense on 3T, on SWI hypointensity with blooming effect	24	Hypointense lesion with blooming effect on GRE	12	Hypointense lesion with blooming effect on SWI
8	60-70	M	Yes	sCAA	Hyperintensity visible, GRE difficult to interpret, some blooming effect visible	Cerebellum	Not visible on 3T or 3T SWI	24	GRE uninterpretable	24	SWI shows hypointense lesion without blooming effect
9	60-70	F	Yes	sCAA	Hyperintensity visible, on GRE hypointensity with blooming effect	Parietal	Not visible on 3T or 3T SWI	38	GRE uninterpretable	12	Hypointense lesion with blooming effect
10	70-80	M	Yes	sCAA	2 hyperintensities visible, on GRE one small and one big hypointensity with blooming effect	Parietal	Also hyperintense on 3T, on SWI hyperintensity with hypointense ring, some blooming effect visible	24	2 lesions invisible on GRE	12	2 hypointense lesion with blooming effect
11	70-80	M	Yes	sCAA	Hyperintensity visible, GRE difficult to interpret, some blooming effect visible	Pons	Not visible on 3T or 3T SWI	9	GRE uninterpretable	11	Invisible on SWI
12	70-80	F	No	sCAA	Hyperintensity visible, GRE macrobleed visible with blooming effect	Occipital	Also hyperintense on 3T, on SWI hypointensity with blooming effect	NA	No 7T follow-up	10	Hypointense lesion with blooming effect

*Researchers are aware of exact age in years of participants; age is categorized for the purpose of anonymity.

Abbreviations. F; female. M; male. CMBs; cerebral microbleeds. D-CAA; Dutch-type Cerebral Amyloid Angiopathy. sCAA; sporadic Cerebral Amyloid Angiopathy.