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7T MRI as a powerful tool to detect small and medium size vessel CNS vasculitis

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ABSTRACT

Imaging can help to diagnose CNS vasculitis. Yet so far, no imaging studies of CNS vasculitis at 7T are available. We share our experience of vessel wall imaging (VWI) at 7T in patients with suspected vasculitis. All included patients (n=45) underwent a clinically approved 7T MRI comprising high resolution arterial TOF angiography as well as high resolution VWI with T1SPACE and T1SE acquired pre- and post-contrast. 23 patients showed negative and 22 patients positive VWI at 7T. Ten out of 22 7T VWI positive cases were suggestive of vasculitis with 9 patients showing VWI of large and medium size vessels and one patient VWI of small vessels. Small vessel vasculitis was only depicted with 7T VWI, but not 3T VWI. Our work demonstrates that diagnosing CNS vasculitis, especially small vessel vasculitis, is feasible at 7T and highlights the potential of high field VWI encouraging further studies in this field.

ABBREVIATIONS: FS= fat-saturated, SNR= signal-to-noise ratio, VWI= vessel wall imaging

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INTRODUCTION

CNS vasculitis is a relatively rare but serious and potentially life threatening condition [1, 2]. The diagnostic process of CNS vasculitis is usually challenging, as there are currently no specific biochemical, immunological, serological, or imaging techniques available [3]. A biopsy of the leptomeninges or cortex remains the gold standard, yet it is an invasive method associated with relevant morbidity and it is prone to sampling error and thus, prone to false negative results [4, 5]. DSA has strongly varying sensitivity, decreasing significantly in small vessels, emphasizing that a normal angiogram therefore does not exclude primary CNS vasculitis [6-8]. Vessel wall imaging (VWI) is a useful adjunct technique to visualize vascular changes indicative of vasculitis that differ for example from intracranial atherosclerotic plaques and other causes of intracranial arterial narrowing [1, 9]. Current vessel wall visualization techniques at 3T are limited by its special resolution and signal-to-noise ratio (SNR) limits and do not resolve smaller intracranial vessels. Common pitfalls are caused by slow flow, dural enhancement, veins and vasa vasorum mimicking arterial wall thickening or enhancement [9] . So far VWI for the assessment of vasculitis has mainly been performed at 3T [9] and explored at lower field strengths [10]. While VWI at 7T is widely explored, especially in atherosclerotic disease and aneurysms, imaging studies specifically dedicated to vasculitis at 7T are currently not available [11]. The aim of this work note is to share our experience of vessel wall imaging at 7T in patients with suspected vasculitis and highlight the potential of this technique at higher field strength.

MATERIAL AND METHODS

We retrospectively screened the health records of our tertiary care institution to identify patients examined at 7T MRI with clinical suspicion of any CNS vasculitis. This retrospective analysis was approved by the local ethic committee (2020-02902). As far as applicable to this brief report, the STROBE guidelines were followed. Patients with suspicion of any form of CNS vasculitis were included (i.e. involvement of large vessels, small vessels or mixed pattern, suspected or confirmed neurovascular pathologies). Cases with isolated leptomeningeal enhancement only were classified as negative for vessel wall enhancement.

All patients were scanned on a clinically approved 7 T whole-body MRI scanner (MAGNETOM Terra, clinical mode, Siemens Healthcare, Erlangen, Germany) equipped with a 1-channel transmit and 32-channel receive head coil (Nova Medical, Wilmington, MA, USA) and on a 3 T scanner (MAGNETOM Prisma, Siemens Healthcare) with a 32-channel head coil in the same session at the translational imaging center (TIC). This retrospective analysis was approved by the local ethics committee. All patients were informed about the MRI-related risks before the examinations as a part of the routine procedure and signed a general consent form.

VWI at 7T comprised axial arterial time-of-flight angiography $0.14 \times 0.14 \times 0.25$ mm3 (interpolated, acquired: $0.36 \times 0.28 \times 0.50$ mm3) with whole brain coverage, axial T1 sampling perfection with application optimized contrast using different flip angle evolution (SPACE) isovoxel $0.50 \times 0.50 \times 0.50$ mm3 with TR/TE = 1200/13 ms; variable flip angle; TF of 40 and total acquisition time 9 minutes and 16 seconds as well as T1 SE of a selected region of interest with 0.20 x 0.20 x 1.0 mm3 and TR/TE=700/13 ms; flip angles 71°/135°; FOV 195 x 240 x 16, two averages and total acquisition time of 7 minutes and 45 seconds. The remaining sequences comprised: 3D MP2RAGE, susceptibility-weighted imaging (SWI) and T2 turbo spin-echo (T2 TSE) sequences as previously described [12].

The 3 T MRI followed a standardized (standard product sequences) vasculitis contrast-enhanced protocol including ToF angiography $0.50 \times 0.50 \times 0.50$ mm3, vessel wall imaging axial T1 SPACE $0.50 \times 0.50 \times 0.50$ mm3 (extrapolated) pre-contrast and post-contrast, TR/TE = 600/12 ms, 2D axial T2 fat-saturated (FS) darkblood post-contrast 2D axial T1 FS darkblood and 2D axial T1 SE 2.0 mm slice thickness, TR/TE=500/13 ms, flip angle 70°, FOV 159 x 159 x 19.8 as well as dynamic susceptibility contrast (DSC) perfusion if not available from recent examinations. Macrocyclic i.v. contrast agent (Gadobutrol 1 mmol/ml, 1 ml/10kg body weight, 5 ml/sec; Gadovist Bayer AG Switzerland) was administered. Exemplary T1 SPACE and T1 SE images at 3T and 7T are illustrated in the Supplemental Figure 1.

We extracted the referral information, radiological reports, final clinical diagnosis and follow-up information if available. VWI was defined as suggestive of vasculitis with the presence of circumferential contrast enhancement in multiple foci on multiple slices with or without abnormalities on ToF-angiography. The findings itself were regarded independent of different etiologies of vasculitis, however the distribution pattern of vessel involvement was reported. All examinations were interpreted in the clinical routine by two experienced clinical readers (5 and 10 years of 7T MRI experience). No additional image reading was performed.

RESULTS

Forty-five patients (45, 20 males and 25 females) were examined between November 2020 and July 2023. Median age was 54.8 (mean 53.6, range 17.8-83.3). All examinations were performed without interruption or side effects. All cases referred for imaging at TIC were ambiguous, based on standard diagnostic workup for vasculitis including 1.5T or 3T MRI with or without prior VWI (Supplemental Figure 4). 21 patients had acute or subacute infarcts, 5 patients presented with hemorrhages and 19 with other pathologies (chronic stroke, leptomeningeal enhancement, white matter lesions). 21 out of these patients underwent prior vessel wall imaging in the routine clinical setting, of which 10 had vessel wall enhancement. Specifically, the cohort includes cases with an equivocal clinical picture, discrepancies between imaging sessions, diverging multimodal evidence or mismatch between the clinical picture and cerebrovascular findings (Supplemental Table 1).

All examinations at TIC were performed in the following order: 7T examination without contrast agent, 3T examination with sequences pre and post application of i.v. contrast and 7T examination with contrast agent. The mean time between contrast application and acquisition of T1 SPACE at 7T was 20 minutes (mean 18 minutes, min. 6 and max. 54 minutes; the delay was equally distributed among the vessel wall imaging negative and positive cases).

According to 7T radiological reports, all examinations showed diagnostic quality. Twenty-three (23, 51%) out of 45 cases were reported as negative, including one previously VWI positive case in clinical routine at 3T (Supplemental Figure 2) based on negative VWI sequences on 7T MRI with subsequent exclusion of vasculitis. Twenty-two (22, 49%) out of 45 cases were reported with positive VWI findings. 10 out of those 22 cases were suggestive of vasculitis, 9 affecting large and mid-size arteries (Figure

1) and one exclusively affecting perforating arteries, that are considered to be small vessels, as their diameter is smaller than 300µm (Figure 2, Supplemental Figure 3).

The remaining 12 cases were reported with arteriosclerosis (7), non-vasculitic small vessel disease (2), dolichoectasia (1), local inflammatory process not related to vasculitis (1) and persisting imaging changes after inflammation (1).

DISCUSSION

In this report, we highlight the potential of 7T imaging to support the diagnosis of CNS vasculitis, as it overcomes several limitations encountered at lower field strengths.

In contrast to 3T, 7T has a higher SNR and imaging resolution and is capable to sharply resolve the vessel wall and its lumen.[13] We used a whole brain 3D T1 SPACE sequence and a 2D T1 TSE sequence, covering the brain area adapted to the clinical question. As illustrated in this brief report vasculitic vessel wall enhancement can be better delineated at 7T with both sequences compared to 3T. T1 TSE achieved a higher in-plane resolution allowing to slightly better characterize a particular vessel segment. However, the whole brain coverage of T1 SPACE is advantageous, as vasculitis often affects several vessel segments.[14] Also, the possibility to assess the whole vessel wall circumference with 3D isotropic data of the T1 SPACE improves analysis. Importantly, smaller vessels, such as perforating arteries, are robustly visualized at 7T with the whole brain T1 SPACE. This is of particular significance, as other techniques such as DSA are only moderately sensitive in detecting VWI of small vessels.

Thus, VWI at 7T may help to depict especially deep small vessel involvement in e.g. autoimmune connective tissue disorders, that are prone to cerebrovascular events compared to the overall population. To date, there are only a few published case reports about vessel wall imaging in patients with rheumatic diseases. For example, a published case report of CNS vasculitis in a systemic lupus erythematosus patient who initially presented with stroke, showed circular VWI of the distal ICA and the M1 segment, consistent with vasculitis.[15] Another case report of a Sjögren's patient with stroke showed circular VWI in the ICA, M1 and the A1 segment, also consistent with CNS vasculitis.[16] In the Sjögren's patient in our cohort only perforating vessels were affected, but no medium sized vessels. As detection of vessel wall enhancement of perforating vessels is only feasible at very high field strength, we hypothesize that CNS vasculitis is likely underdiagnosed in patients with rheumatic diseases, and merits further investigations.

Also for disease monitoring 7T VWI may add value, as it allows a better visualization of the vessel wall and consequently treatment effects. Importantly, as evident in our patient collective, the presented imaging technique is feasible in clinical routine and can help to solve ambiguous cases, with the increasing availability of FDA approved and CE certified 7T MR scanners.

Our work has limitations, as the time of contrast application and the acquisition of vessel wall imaging sequences is non-standardized, not established at 7T (in contrast to lower field strength [17]) and varied between individuals but to a similar degree in VWI positive and negative cases in this study. Nevertheless, quantitative analysis of vessel wall enhancement was not feasible due to different contrast delay time and a small patient collective. Properties of contrast enhancement as well as optimal delay for vessel wall imaging at 7T including direct application at 7T need to be further studied; they have been examined to some extent, however for other applications but not specifically for CNS vasculitis. [18, 19] Potentially, delayed imaging could lead to a decrease of false positive findings reducing specificity. On the other hand, sensitivity could increase, as contrast agent has more time to accumulate within the vessel wall. In the field of CNS vasculitis specifically, a prospective head-to-head comparison of 7T vs. 3T would be necessary to prove a benefit of the higher field strength. Furthermore, advantages and pitfalls of T1 SPACE and T1 SE at 7T should be examined with T1 SPACE providing whole brain coverage and T1 SE providing superior SNR in a selected field of view. Finally, subgroups of patients with CNS vasculitis benefiting most from 7T must be identified. In summary, higher resolution VWI at 7T is feasible, addresses several limitations encountered at lower field strength and shows the potential to improve detection of CNS vasculitis, including involvement of small vessels, encouraging further studies in this field.

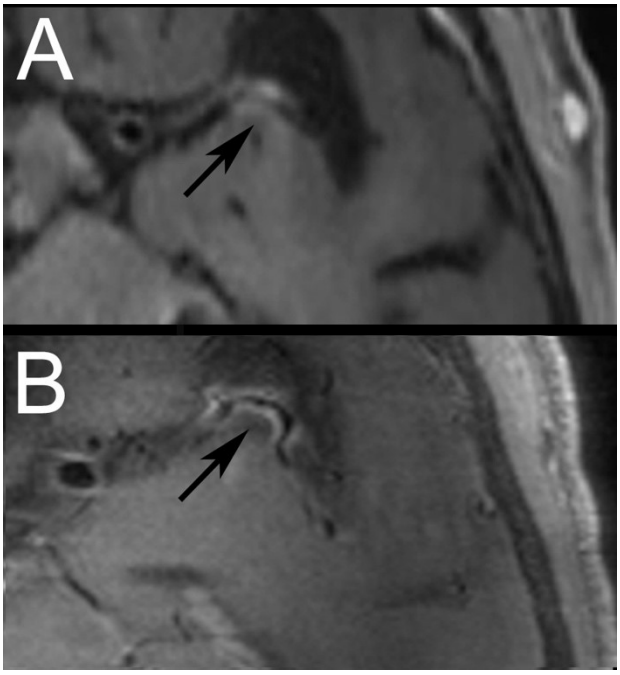

FIG 1. Medium to large vessel vasculitis. Representative T1 SPACE images of a patient later diagnosed with CNS vasculitis due to borrelia burgdorferi infection are presented at admission at 3T (A) and 7T (B). VWI at 3T and 7T show enhancement of the proximal M2 segment. The thickened, enhancing vessel wall is more clearly delineated at 7T compared to 3T.

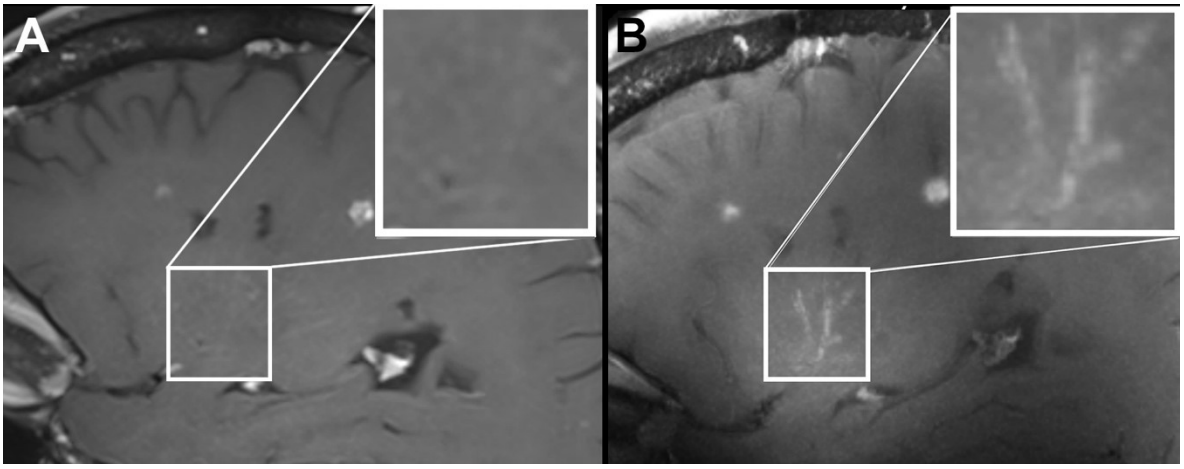

FIG 2. Detection of small vessel vasculitis at 7T with VWI in a patient with Sjögren's disease and subacute stroke. Representative T1 SPACE images of a patient later diagnosed with CNS vasculitis due to borrelia burgdorferi infection are presented at admission at 3T (A) and 7T (B). VWI at 3T and 7T show enhancement of the proximal M2 segment. The thickened, enhancing vessel wall is more clearly delineated at 7T compared to 3T.

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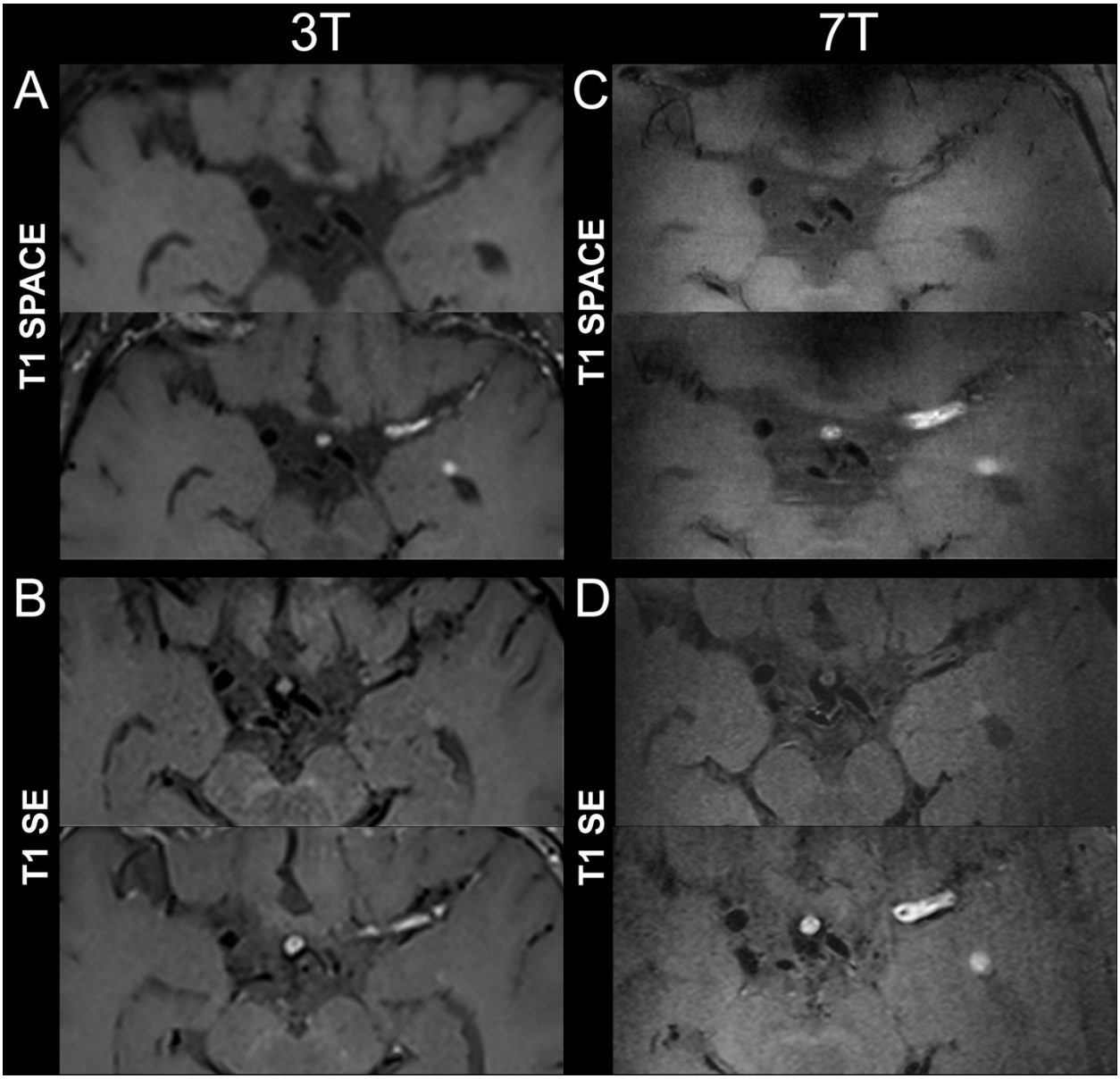
SUPPLEMENTAL FILES

Supplemental Table 1. Indications for 7T MRI as documented in the electronic health record.

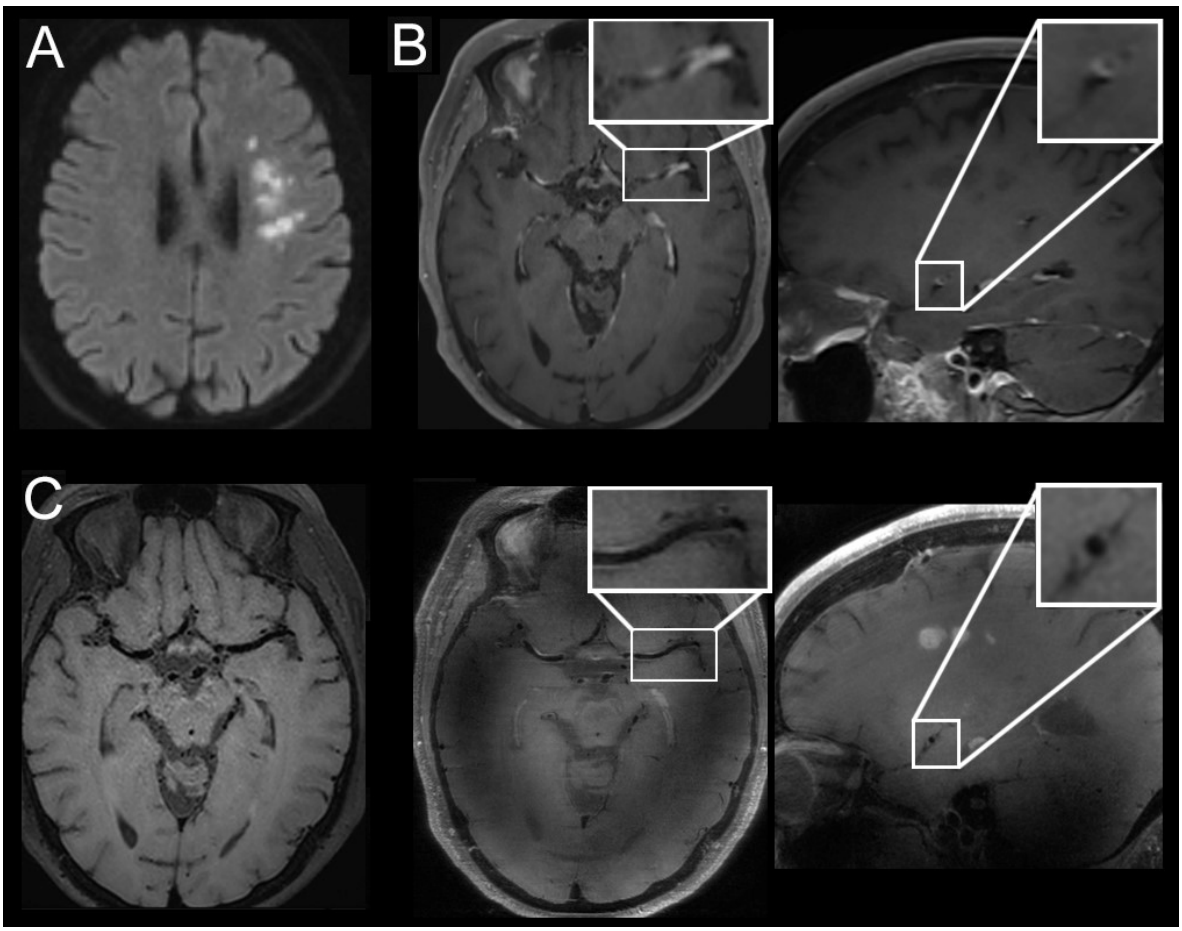
No.	Sex	Age	Indication
1	f	55	Acute onset left-sided cerebellar syndrome, multiple ischemic lesions of varying age, DSA negative. Vasculitis?
2	f	50	Known SLE. Multiple ischemic lesions and subarachnoid hemorrhages of unknown etiology. Multiple vessel irregularities but no vessel wall enhancement in standard imaging. Vasculitis?
3	f	47	Clinically suspected vasculitis, vessel wall irregularities at 3T MRI. Vasculitis?
4	f	21	White matter lesions of unknown etiology, recurrent headaches. Vasculitis?
5	f	29	Recurrent headaches, vessel wall abnormalities at 3T MRI of unknown etiology. Vasculitis?
6	m	83	Ischemic lesions of unknown origin, headache and confusion episodes, cerebral microbleeds on SWI, possible vessel wall enhancement at 3T MRI. Vasculitis?
7	f	55	HSV-1 infection in medical history, chronic headaches, neurological deficits attributed to cranial nerves, cognitive decline. Encephalitis? Vasculitis?
8	m	59	Multiple ischemic lesions of varying age, suspicion of lymphoma or vasculitis on 3T. Vasculitis?
9	m	40	TIA, headaches, abnormal CSF findings, suspected dissection of the left ICA on standard MRI. Vasculitis?
10	m	52	Multiple ischemic lesions in right MCA territory, suspected arteriosclerotic disease. Vasculitis?
11	m	44	White matter lesions, repetitive brain hemorrhages, cerebral microbleeds. Differentiation vasculitis vs. CAA/CAA-related inflammation
12	f	61	Headaches and cognitive decline. Confirmed rheumatoid arthritis. Clinically suspected CAA-related inflammation. Differentiation vasculitis vs. CAA/CAA-related inflammation
13	f	55	Ischemic stroke of unknown etiology, multiple white mater lesions, cerebral microbleeds. Vasculitis?
14	m	50	Ischemic stroke, at 3T suspected vessel irregularities and vessel wall enhancement suspicious of vasculitis, no signs of vasculitis in blood and CSF analysis. Vasculitis?
15	m	52	Multiple cortical and subcortical ischemic lesions of unknown etiology. Suspected neoplastic disease of urinary tract. Vasculitis?
16	m	67	Pachymeningitis of unknown origin with perineuritis of the optic nerves, suspected IgG4-associated disease. Signs of vasculitis?
17	f	63	Multiple white matter lesions of unknown etiology. Vasculitis?
18	f	59	Ischemic stroke in left MCA-territory, suspected vasculitis at 3T MRI, CSF examination without abnormalities. Vasculitis?
19	f	83	Intracerebral hemorrhage, clinically suspected vasculitis. Vasculitis?
20	m	18	Acute stroke with confirmed COVID infection. Vasculitis?
21	m	68	Ischemic strokes, severe white matter lesions of unknown etiology, cerebral miccrobleeds. Vasculitis?
22	f	68	Multiple ischemic lesions of different age. Vasculitis?
23	m	26	ICA and MCA stenosis, C. burnetii infection. Vasculitis?
24	f	47	White matter lesions, clinically suspected Sjögren-Syndrome. Vasculitis?
25	m	46	Multiple ischemic lesions of unknown etiology. Differentiation Vasculitis, reversible cerebral vasoconstriction syndrome, embolic stroke
26	f	42	White matter lesions of unknown etiology, known arteriosclerotic disease. Differentiation vasculitis, atherosclerosis, antiphosphoslipid syndrome
27	m	55	Recurrent ischemic strokes in left MCA territory, fluctuating symptoms with cognitive deficits and aphasia. Vasculitis?
28	m	60	Unknown inflammatory disease of the CNS, corticosteroid-responsive, fluctuating symptoms. Vasculitis?
29	m	56	Suspected sarcoidosis, CNS manifestations, Vasculitis?
30	f	54	Confirmed giant cell arteritis. New intracranial stenoses of the ICA and MCA on standard MRI. Vasculitis?
31	m	29	Encephalopathy with seizure, confirmed COVID. Influenza B and Influenza A encephalopathy in the medical history.

			Vasculitis?
32	m	52	Confirmed Sjögren-Syndrome. CNS vasculitis?
33	f	33	Suspected varizella zoster virus (VZV) vasculitis. Differentiation primary CNS vasculitis vs VZV vasculitis
34	f	54	White matter lesions of unknown origin. Vasculitis?
35	f	69	Vessel wall enhancement at 3 MRI. Differentiation vasculitis vs. arteriosclerotic disease
36	m	62	Suspected primary angiitis of the CNS. Confirmation of MRI findings, localization of biopsy site
37	m	60	Spinocerebellar syndrome of unknown etiology. Vasculitis?
38	f	56	White matter lesions of unknown etiology, multiple sclerosis, small vessel disease, vasculitis
39	f	50	Encephalitis of unknown origin, clinically suspected autoimmune etiology. Vasculitis?
40	m	68	Recurrent ischemic strokes of unknown etiology. Vasculitis?
41	f	53	White matter lesions and cognitive symptoms. Differentiation primary angiitis of the CNS vs CADASIL
42	f	65	White matter lesions of unknown etiology clinically suspected vasculitis. Vasculitis?
43	f	78	Clinically suspected eosinophilic cANCA-associated vasculitis. Vasculitis?
44	f	58	Atypical intracranial hemorrhage of unknown etiology. Differentiation reversible cerebral vasoconstriction syndrome vs. vasculitis
45	f	63	Confusion. Corpus callosum lesion. Vasculitis?

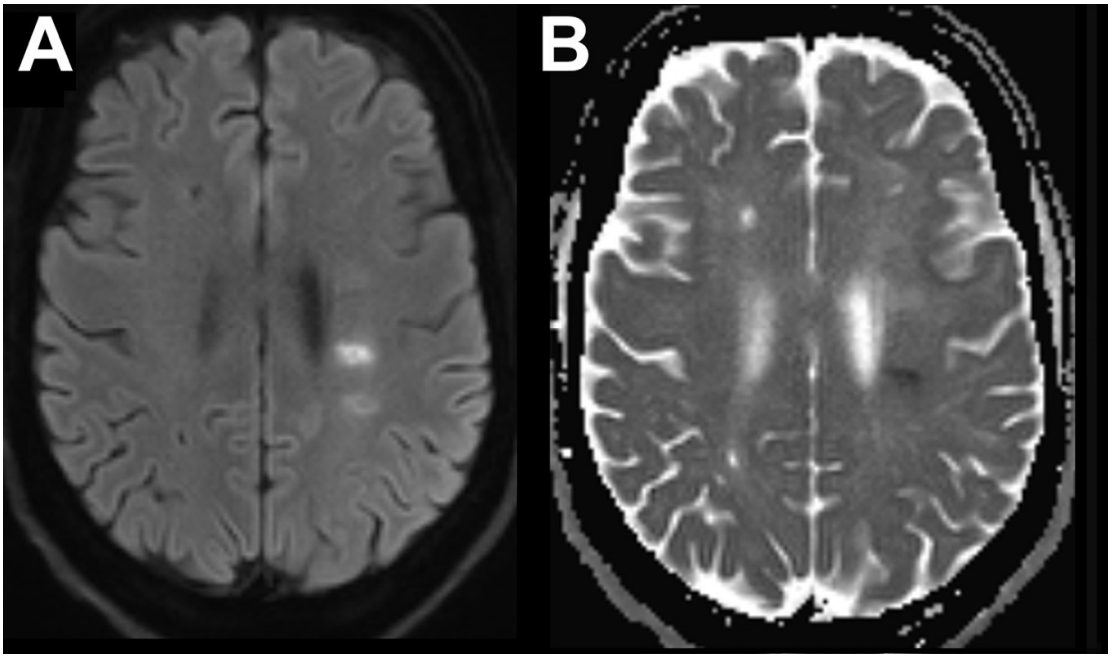
SUPPLEMENTAL FIGURES



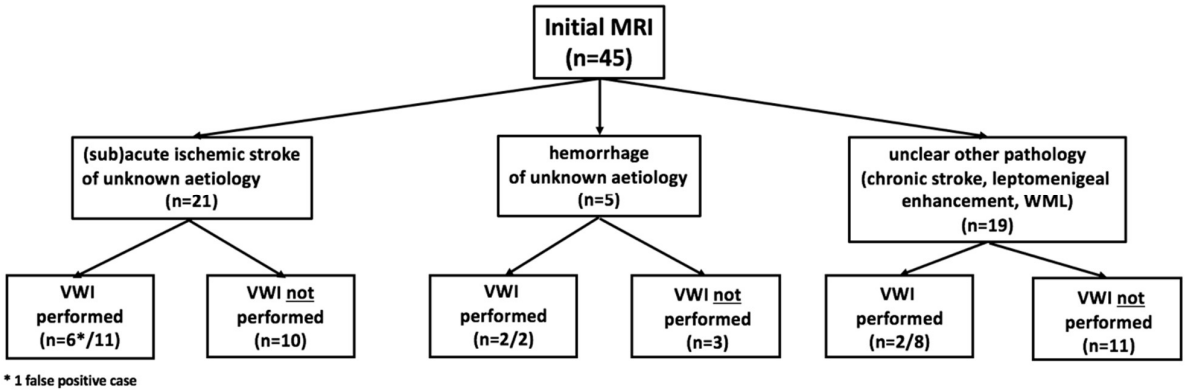
Supplemental Figure 1: Vessel wall imaging sequences at high and ultra-high field. Representative T1 SPACE (A) and T1 SE (B) images at 3T without (upper image) and with contrast agent (lower image); the vessel wall is less clearly delineated compared to T1 SPACE (C) and T1 SE (D) images at 7T without (upper image) and with contrast agent (lower image). The residual vessel lumen is visualized at 7T with both sequences. Signal to noise is higher, if the T1 SE sequence is used. In contrast to T1 SE sequence, full head coverage can be obtained with the T1 SPACE sequence. In this patient, with recurrent ischemic strokes in the left MCA territory, fluctuating cognitive deficits with aphasia, dysarthria and right hemi-symptomatic, a primary CNS vasculitis with the involvement of left terminal ICA and proximal MCA (shown) was diagnosed



Supplemental Figure 2: False positive vessel wall enhancement at 3T. A patient with watershed infarcts in the left corona radiata was examined at 3T to assess a possible vasculitis (A, DWI b1000 image). Circumferential left MCA vessel wall enhancement was evident at 3T VWI (B, contrast-enhanced T1 SPACE, MCA magnified in right upper corner). In contrast to 3T, at 7T no vessel wall thickening on native T1 SPACE images was evident (C), nor contrast enhancement along the MCA (C, T1 SPACE without and with contrast (axial and sagittal slice), MCA magnified in right upper corner). Clinical follow-up confirmed the absence of vasculitis. This case illustrates that venous Gd enhancement can be reduced at higher field strength as T2* relaxation time for blood is decreased at 7T compared to 3T, leading mainly to a faster T2* signal decay at 7T. An increased signal to noise ratio as well as delayed imaging after contrast administration at 7T (in this patient 20 minutes) may also contribute to the observed finding.



Supplemental Figure 3: Infarct pattern corresponding to the Sjögren's disease patient in Figure 2. Acute and subacute infarcts are visible in the right corona radiata on the b1000 image (A) and ADC map (B) at 3T. Old lacunes are seen in the fontal white matter of both hemispheres.



Supplemental Figure 4. Flow chart of included patients illustrating why suspicion of vasculitis arose and how many patients underwent prior vessel wall imaging (VWI). VWI at 3T MRI suspected VW enhancement in 6 out of 11 patients. Out of these 6 patients one patient was later diagnosed VW enhancement negative at 7T MRI (marked as * on the bottom of left box = 1 false positive case out of 6 cases, images are shown in supplemental figure 2).