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ORIGINAL ARTICLE

Diagnostic Value of Contrast-Enhanced Vessel Wall Imaging in the Evaluation of Various Intracranial Non-Vascular Pathologies: A Single Center Experience

Deniz Aliş[1], Düzgün Yıldırım[2], Amalya Zeynalova[2], Filiz Tüzüner[3], Mustafa Emir Tavşanlı[4], Mustafa Seçkin[4], Elvan Cevizci Akkılıç[5], Murat Hamit Aytar[6], Deniz Esin Tekcan Şanlı[7]

[1]Department of Radiology, Acibadem Maslak Hospital, Istanbul, Türkiye
[2]Department of Radiology, Acibadem Taksim Hospital, Istanbul, Türkiye
[3]Department of Neurology, Acibadem Taksim Hospital, Istanbul, Türkiye
[4]Department of Anesthesiology, Acibadem Taksim Hospital, Istanbul, Türkiye
[5]Department of Neurology, Acibadem Kozyatagi Hospital, Istanbul, Türkiye
[6]Department of Radiology, Acibadem Kozyatagi Hospital, Istanbul, Türkiye
[7]Department of Neurosurgery, Acibadem Kozyatagi Hospital, Istanbul, Türkiye

Abstract

Introduction: Vessel Wall Imaging (VWI) is a relatively novel magnetic resonance imaging (MRI) technique primarily aimed at diagnosing vascular pathologies. In this study, we aimed to evaluate the diagnostic value of contrast-enhanced (CE) VWI in identifying various intracranial non-vascular pathologies.

Methods: This retrospective study was approved by our institutional ethics committee with approval number 2022-05/17 on March 11, 2022. We retrospectively evaluated cranial CE-MRI, including the VWI sequence, of 189 patients (76 females and 113 males) who were referred to our radiology department for brain imaging for various reasons. MRI examinations were performed using a 3 Tesla unit. A single observer evaluated anonymized cranial MRI images without CE-VWI in addition to the relevant clinical information in a random order. The same observer interpreted the CE-VWI with relevant clinical information six weeks later. The findings, which could only be visualized on VWI in the second session, were noted.

Results: In 10 patients of our study cohort (5.3%), VWI demonstrated pathological signal alterations or contrast enhancement (e.g., post-status frontal lobe pial enhancement in a patient with autoimmune epilepsy, contrast enhancement in the hippocampus in a diffusion-negative hyper-acute ischemic stroke patient, and optic disc enhancement in a patient with intracranial hypertension) that apparently reflected underlying clinical disorders, which otherwise could not be visualized on conventional MRI.

Discussion and Conclusion: CE-VWI might serve as a valuable adjunct for the diagnosis of various parenchymal or meningeal intracranial diseases, yet further, more comprehensive studies are needed to reveal the true potential of VWI.

Keywords: FLAIR; Intracranial Parenchymal Diseases; MRI; Vessel Wall Imaging.

Correspondence: Amalya Zeynalova, M.D. Department of Radiology, Acibadem Taksim Hospital, Istanbul, Türkiye
Phone: +90 553 988 38 44 **E-mail:** dr.amaliyazeynalova@yahoo.com
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Intracranial applications of vessel wall imaging (VWI) have received considerable attention in the current decade, and VWI is increasingly being used in the diagnosis of various intracranial vascular pathologies, including atherosclerosis, vasculitis, and aneurysms[1,2]. Apart from the traditional imaging modalities of the intracranial arterial system, such as digital subtraction angiography (DSA), computed tomography angiography (CTA), or magnetic resonance angiography (MRA), VWI could demonstrate the pathologies of the vessel wall and entail a detailed characterization of the detected lesion by suppressing signals from the flowing blood in the vessel lumen[3,4]. It has been well shown that VWI could accurately detect intracranial arterial pathologies at a very early stage and prevent further complications of the diseases by allowing physicians to perform prompt treatments. To date, many different VWI protocols have been identified[3,4]. Despite the fact that the physics principles of VWI could be discussed in pages, it could be briefly explained that VWI mainly suppresses the signals coming from mobile tissue components such as flowing blood while enhancing the signals from static tissues such as brain parenchyma.

Contrast-enhanced (CE) T1-weighted (T1W) spin-echo (SE) imaging is the primary technique of choice when assessing various intracranial pathologies. However, several studies have reported that lesions might be obscured or mimicked due to potential artifacts in the CE-T1W sequence[5]. Furthermore, several other contrast-enhanced methods, including CE fluid attenuation inversion recovery (FLAIR) sequence, have been shown to have better lesion-to-background and grey-to-white matter contrast-to-noise ratios; thus, they are superior in delineating and characterizing intracranial lesions, particularly by suppressing background and cerebrospinal fluid (CSF)[5-7]. To our knowledge, no study has evaluated the ability of VWI to diagnose intracranial pathologies other than vascular diseases.

Herein, we assessed cranial CE-MR examinations, including CE-VWI, of patients admitted to our radiology department for various reasons to evaluate the potential diagnostic value of CE-VWI in detecting intracranial pathologies.

Materials and Methods

The local ethics committee approved this retrospective study. We evaluated cranial contrast-enhanced MRI (CE-MRI), including the CE-VWI sequence, of 192 patients (76 females and 116 males). Among these patients, 3 were excluded due to inadequate image quality of the VWI. The mean age of the patients was 47.3 years (age range, 22-74 years). All MRI examinations were performed using a 3 Tesla unit equipped with a 32-channel head coil (3T Magnetom Skyra, Siemens AG Healthcare, Erlangen, Germany). Our cranial CE-MRI protocol included pre-contrast T1-weighted 3D magnetization-prepared rapid gradient echo (MPRAGE), axial T2-weighted (T2W), coronal T2W, axial T2W FLAIR, susceptibility-weighted imaging (SWI), diffusion-weighted imaging (DWI), time-of-flight (TOF), and post-contrast (intravenous administration of 0.1 mmol/kg gadolinium at a flow rate of 2 mL/s) T1W 3D MPRAGE sequences. Following our standard protocol, CE-VWI sequences were obtained six to eight minutes after contrast injection, following the post-contrast T1W 3D MP-RAGE sequence. The parameters for the CE-VWI were: TR=600 ms, TE=20 ms, turbo factor (TF): 28, Black Blood, Interleaved, turbo spin-echo (TSE), Fat-Sat. The VWI sequence was obtained using isotropic voxels (0.5 mm) with a scan duration of approximately 8-9 minutes, covering the main cerebral arteries of the circle of Willis. Additional CSF suppressing methods were not utilized. We completed our protocol with delayed CE-FLAIR sequences, given their ability to demonstrate subtle pathological signals by enhancing subtle contrast enhancements in the late phase. The parameters for the late T2 FLAIR sequence were: TR=9000 ms, TE=124 ms, Time to inversion: 2492 ms. All other sequences were obtained using the manufacturer's standard cranial MRI protocols.

Initially, a single observer (with more than 15 years of neuroradiology experience) evaluated anonymized cranial MRI images with relevant clinical information in a random order. Pathological findings were noted. Six weeks after the initial interpretation session, CE-VWI was added to the first data set, and the observer re-evaluated the same anonymized cranial MRI images. Initially, the observer evaluated only CE-VWI. If the observer identified any pathological contrast enhancement on CE-VWI, then they cross-checked TOF images to elucidate whether the contrast-enhanced area was consistent with the intracranial arteries, especially where the arteries pass through the dura mater, as these areas might show contrast enhancement in healthy subjects. All pathological findings were again noted. Afterwards, a second reader (with 5 years of neuroradiology experience) evaluated

all the findings from the first and second session, and findings that could only be visualized on CE-VWI in the second session were noted.

Results

Finally, cranial MRI images of 189 patients were reviewed, and in 10 patients, we detected findings that could not be detected on conventional CE-MRI but were visualized on CE-VWI. These findings included an additional metastatic focus in the cerebellar vermis in a lung cancer patient, post-status frontal lobe pial enhancement in a patient with autoimmune epilepsy, postoperative residual glossopharyngeal schwannoma, oculomotor nerve enhancement in a patient with Miller-Fisher syndrome, contrast enhancement in the right hippocampus in a patient with hyper-acute ischemic stroke with normal early diffusion MRI findings, a previously undetected pituitary gland micro-adenoma, enhancement in labyrinths in a case with posttraumatic vestibular syndrome, and optic disc enhancement in a patient with intracranial hypertension (Table 1). In 4 of these 10 patients (40%), cases 3, 8, 6, and 10, delayed CE-FLAIR did not show any pathological contrast enhancement in the relevant area, while in the other 6 patients (60%), CE-FLAIR images also depicted the pathology. Figures 1-3 show the VWI and conventional MRI findings of cases with numbers 1, 2, and 9.

Table 1. Clinical information and CE-VWI findings of the cases in which additional findings were identified on CE-VWI.

Cases	Age/ Gender	Clinical diagnosis	Findings that could be only detected in VWI	Confidential information
1	78/M	Lung carcinoma-Brain metastases	Enhanced small junctional nodule in the left frontal lobe.	Negative MRI 2 years ago
2	17/M	Autoimmune Epilepsy	Pial enhancements in the left frontal lobe.	No epileptic episode was observed after immune suppressive treatment, and CE-WVI findings were dramatically regressed.
3	37/M	Glossopharyngeal neuralgia	Post-operative residual lesion in the right juguler foramen.	The residual tumor was also detected in contrast enhanced sequences after resolution of post operative inflammation
4	37/M	MTHFR gene mutation	Parenchymal enhancement in various localizations.	Fist, Findings were evaluated for potential underlying vasculitis; however, clinical and laboratory findings were found negative. Hence, gene analysis was conducted then the final diagnosis was established.
5	35/M	Lyme disease	Parenchymal and oculomotor nerve enhancement.	Positive Lyme test.
6	62/F	Miller Fisher syndrome	Brain stem and 3rd cranial nerve enhancement.	Patient was well responded to the treatment of the syndrome after diagnosis.
7	51/M	Parotid gland abscess	VWI demonstrated detailed the close relation of the abscess and the facial nerve; therefore, aided to determine a precise entry point with 3D mapping for drainage.	Drainage was performed from the inferior part without any complication.
8	29/M	Pituitary adenoma	Microadenoma located on the right side of the pituitary gland.	Presence of galactorrhea and increased prolactin levels.
9	61/M	Acute infarct	The right-sided MCA infarct, negative DWI findings but enhancement in the right MCA zone in WVI.	Diffusion restriction was observed in repeated DWI after following hours
10	33/M	Vestibular contusion.	Vestibular and labyrinths enhancement in the right side.	Positive clinical test and complete response to treatment in 3 weeks.

* contrast-enhanced vessel wall imaging (CE-CWI), diffusion-weighted imaging (DWI), methylenetetrahydrofolate reductase (MTHFR), middle cerebral artery (MCA), magnetic resonance imaging (MRI)

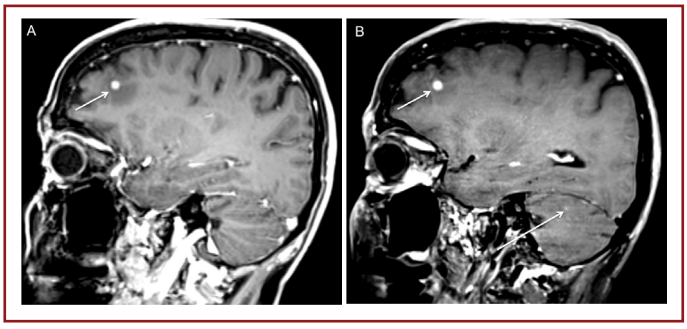

Figure 1. A 78-year-old male patient with known lung cancer (case number 1). **(a)** The metastatic lesion with abundant contrast enhancement is seen on post-contrast T1W 3D MP-RAGE sequence in the left frontal lobe (short arrow). **(b)** CE-VWI of the same patient demonstrates another metastatic focus with a smaller diameter in the cerebellar vermis (arrow), in addition to the lesion in the left frontal lobe (short arrow).

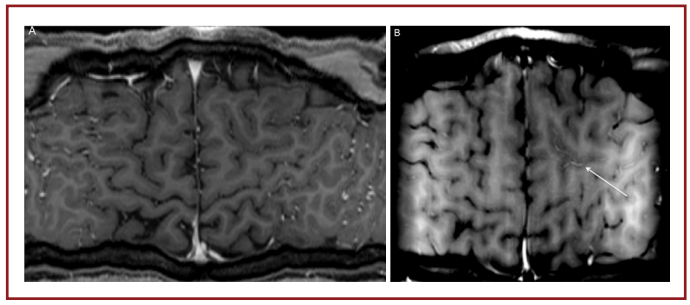

Figure 2. A 17-year-old male (case number 2) with a history of epileptic episodes. **(a)** Post-status CE T1W is normal, while **(b)** VWI sequence reveals pial enhancement in the frontal lobe (arrows).

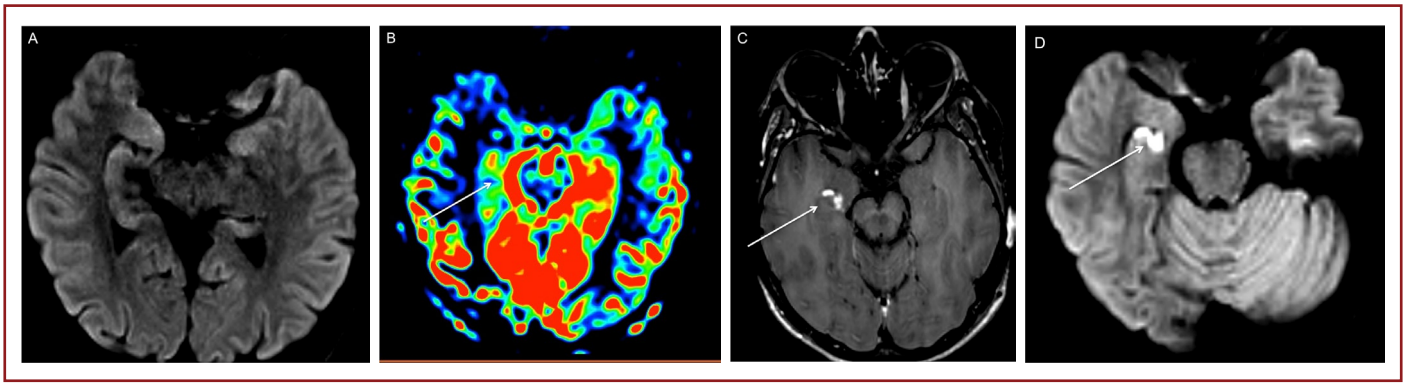

Figure 3. A 61-year-old male presented with rapidly onset short-term memory loss. Initial DWI is negative **(a)**, yet arterial spin labeling image clearly depicts reduced perfusion in the right hippocampus (arrow) **(b)**. CE-VW image of the same patient shows prominent contrast enhancement in the right hippocampal area (arrow) **(c)**. Further DWI of the patient after 12 hours of the initial examination shows diffusion restriction (arrow) **(d)**.

Discussion

In our study, we were able to substantially suppress the background signals, such as those from flowing blood and CSF, with the parameters we utilized on CE-VWI. Consequently, we identified non-vessel intracranial pathological lesions in 5.3% of the patients by CE-VWI, which otherwise could not be detected. In our study, all of the additional pathological contrast enhancements on VWI were also visualized and confirmed on delayed CE-FLAIR images. However, the observer first identified pathological contrast enhancement on CE-VWI and then searched for the pathological signal alterations at the same area on CE-FLAIR images, which eventually might have caused a bias for the accuracy rate of the delayed CE-FLAIR images in detecting the pathologies. On the other hand, the reader subjectively reflected that the contrast enhancement on the CE-VWI was more prominent in nearly all patients. However, given the design of the study, it was not possible to compare the diagnostic ability of the two methods; furthermore, it was also beyond the scope of the present work.

We would like to briefly discuss potential theoretical superiorities of two techniques over each other. FLAIR images are heavily T2W images characterized by long TR and TE with an inversion time for suppressing CSF signals. Given the T2W nature of the technique, most pathological intracranial lesions show hyperintense signal on null FLAIR images. Thus, the observer could not determine whether the high signal intensity occurs due to contrast enhancement; therefore, a delayed CE-FLAIR sequence needs to be performed with both pre-contrast scans[8-10]. On the other hand, CE-VWI does not have this disadvantage, except in cases of hemorrhage, which shows high signal on null VWI, and high signal intensity on CE-VWI almost always reflects pathological

contrast enhancement[3]. VWI is also superior to FLAIR sequences owing to its ability to demonstrate potential vascular pathologies[1-4]. Finally, and also one of the most significant disadvantages of the delayed CE-FLAIR sequence compared to CE-VWI, is the potential presence of hyperintense artifacts caused by CSF pulsations, which might substantially limit the diagnostic value of the sequence, especially in the brainstem[11]. Contrarily, the nature of VWI allows the sequence to adequately suppress CSF pulsations[11-13]. Moreover, in cases where very slow CSF flow might not be adequately suppressed by default on VWI, adding inversion sequences for CSF or using delay alternating with nutation for tailored excitation (DANTE) preparation pulses could substantially suppress signals due to CSF pulsation[11-13].

The relatively long acquisition time of the CE-VWI sequence, which ranges from 7 to 8 minutes depending on the size of the examined area, is the main disadvantage of CE-VWI compared to the CE-FLAIR sequence. However, we suggest that integrating VWI with other CE-MRI sequences could compensate for the long acquisition time of the VWI sequence. Overall, despite many researchers investigating the potential role of CE-FLAIR sequences, especially in comparative studies of CE-FLAIR and CE-T1W sequences, the results have been inconclusive among studies[14-16], and the CE-FLAIR sequence is not widely used in current daily clinical practice for the diagnosis of parenchymal or meningeal pathologies, often being used for research purposes[17]. To our knowledge, no study has investigated the value of CE-VWI for the diagnosis of non-vascular intracranial pathologies; therefore, it is not possible to compare our results.

In the current and following paragraphs, we will briefly discuss several cases in which VWI played a substantial role in the diagnosis and prognosis of the patients. However, we underline that all cases to be discussed below are comprehensive, and thoroughly discussing all their clinical and radiological findings is beyond the scope of the present study. In the patient with primary lung adenocarcinoma, we identified a 3 cm solitary metastasis in the left frontoinsular region using images from our standard cranial MRI protocol. However, we identified another metastatic focus, which was 5 mm in size and located in the cerebellar vermis using CE-VWI. We suggest that the abundant suppression of background parenchyma by VWI allowed the identification of the contrast-enhanced focus in the cerebellar vermis. Detecting the second metastatic lesion would have an enormous impact on the management of the patient[18,19].

In the patient with the provisional diagnosis of autoimmune epilepsy, which was established by clinical findings in addition to epileptic waves on the left frontal lobe on electroencephalography (EEG), the conventional MRI dataset of the patient was interpreted as normal. However, we identified slight contrast enhancement through the left frontal convexity pia-arachnoid meningeal interface on CE-VWI, which was concordant with the EEG and clinical findings. The patient responded well to treatment, and the findings on CE-VWI also regressed entirely. This case, in which the contrast enhancement was subtle (as shown in fig. 2), contrasts with cases where abundant confluent contrast enhancement was detected in the brainstem or cerebral hemispheres in a patient with methylenetetrahydrofolate reductase (MTHFR) mutation (case 4), a patient with Lyme disease presented with increased autoantibodies (case 5), and a patient with Miller-Fisher syndrome having oculomotor nerve involvement (case 6). These contrast enhancements were also confirmed on CE-FLAIR sequences, yet were slightly less prominent compared to CE-VWI. Furthermore, contrast enhancements in the cranial nerves were more prominent in the patient with Lyme disease on CE-VWI sequences compared to CE-FLAIR.

The phenomenon of diffusion-negative stroke has been described in the literature[20,21]. In our study cohort, we had a patient whose MRI examination was performed within the very early hours of acute ischemia, resulting in negative findings on DWI. However, VWI showed contrast enhancement in the right anterior peri-insular area, which is supplied by the right MCA. In this patient, repeated DWI after several hours showed diffusion restriction in the same area. We suggest that in a patient whose clinical presentation is suggestive of acute ischemia though MRI findings are negative, VWI could provide valuable information for prompt diagnosis and management.

Several limitations of this study need to be acknowledged. First and foremost, our study was designed as a descriptive case-control study; thus, we did not compare the diagnostic ability of VWI with other well-established cranial MRI sequences, and we only evaluated the presence of additional findings in VWI. Therefore, we did not have any data on whether VWI also showed all the pathological findings identified in other sequences. Second, we randomly evaluated cranial MRI examinations of patients admitted to our radiology department for various reasons; thus, we were not able to assess the diagnostic ability of VWI in identifying pathological changes in specific diseases. We believe that assessing the

potential role of the sequence by comparing its diagnostic ability with other well-known cranial MRI sequences in further studies focused solely on aforementioned diseases or other non-vascular intracranial diseases with large patient populations could be noteworthy. Finally, the same reader performed all the interpretations; thus, we were not able to assess interobserver variability. Moreover, despite anonymization of the patients, the observer was aware of the clinical information given the design of the study, which might have caused a bias while assessing patients' MR images.

Conclusion

In conclusion, to our knowledge, our study is the first one to use VWI in assessing non-vascular intracranial pathologies. Overall, in the present work, VWI offered a substantial aid that enabled the reader in identifying distinct pathological contrast-enhancements likely reflective of the underlying pathological process of the relevant disease. We highlight that VWI seems to be a promising adjunct for the diagnosis of various parenchymal or meningeal intracranial diseases yet further, more comprehensive studies are needed to confirm our findings and to reveal the true value of VWI.

Ethics Committee Approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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