Vessel wall MR imaging in neuroradiology

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Abstract
Vessel wall MR imaging (VW-MRI) has been introduced into clinical practice and applied to a variety of diseases, and its usefulness has been reported. High-resolution VW-MRI is essential in the diagnostic workup and provides more information than other routine MR imaging protocols. VW-MRI is useful in assessing lesion location, morphology, and severity. Additional information, such as vessel wall enhancement, which is useful in the differential diagnosis of atherosclerotic disease and vasculitis could be assessed by this special imaging technique. This review describes the VW-MRI technique and its clinical applications in arterial disease, venous disease, vasculitis, and leptomeningeal disease.

Keywords Vessel wall MR imaging · 3D MRI · Vessel wall enhancement · Magnetization transfer · MR angiography · Delay alternating with nutation for tailored excitation

List of symbols

| Symbol | Description |
|--------|-------------|
| TOAST  | Trial of Org 10 172 in Acute Stroke Treatment |
| VW-MRI | Vessel wall MR imaging |
| CE     | Contrast enhanced |
| TOF    | Time-of-flight |
| MRA    | MR angiography |
| CTA    | CT angiography |
| FSE    | Fast spin echo |
| VFA    | Variable flip angle refocusing pulse |
| MPR    | Multiplanar reconstruction |
| CSF    | Cerebrospinal fluid |
| MSDE   | Motion-sensitized driven equilibrium |
| DANTE  | Delay Alternating with Nutation for Tailored Excitation |
| Mt     | Magnetization transfer |
| SNR    | Signal-to-noise ratio |
| IPH    | Intraplaque hemorrhage |
| LRNC   | Lipid-rich necrotic core |
| CAD    | Cervical artery dissection |
| ICAD   | Internal carotid artery dissection |
| VAD    | Vertebral artery dissection |
| SAH    | Subarachnoid hemorrhage |
| RCVS   | Reversible cerebral vasoconstriction syndrome |
| MCA    | Middle cerebral artery |
| CNN    | Convolutional neural network |
| SWI    | Susceptibility-weighted imaging |
| 3D     | Three-dimensional |
| DSA    | Digital subtraction angiography |
| PACNS  | Primary angiitis of the central nervous system |
| GCA    | Giant cell arteritis |
| FDG-PET| Fluorodeoxyglucose positron emission tomography |
| A-AION | Arteritic anterior ischemic optic neuropathy |
| HIV    | Human immunodeficiency virus |
| COVID-19| Coronavirus disease 2019 |
| GRE    | Gradient echo |

Introduction
Ischemic stroke is the common neurologic diseases and following five broad subtypes has been classified to represent most clinical scenarios in the Trial of Org 10 172 in Acute
Stroke Treatment (TOAST) system: large artery atherosclerosis, small artery occlusion, cardioembolism, other demonstrated cause, and undetermined cause [1, 2]. Intracranial atherosclerosis is one of major causes of ischemic stroke, and vessel wall MR imaging (VW-MRI) is suitable for evaluation of intracranial atherosclerosis compared with other intraluminal imaging such as transcranial Doppler, time-of-flight (TOF) MR angiography (MRA), contrast enhanced (CE) CT angiography (CTA), and digital subtraction angiography (DSA). For these reasons, high-resolution VW-MRI has been gaining interest for detailed visualization of intracranial vessel walls. The subtype classification to determine the causes of stroke is important in clinical practices [1, 2]. The evidence of extracranial or intracranial disease supports large artery atherosclerosis [3, 4], and negative results on VW-MRI are also important for the subtype determination of cardioembolisms and undetermined causes.

VW-MRI has been introduced in clinical practices, applied to various diseases, and its usefulness has been reported. The purpose of this paper was to review imaging findings of VW-MRI.

**Techniques of VW-MRI**

VW-MRI has been used mainly to evaluate vulnerable plaque in extracranial carotid artery with 2D imaging such as double inversion recovery previously [5]. The drawbacks of 2D imaging are low slice-selective resolution compared with in-plane resolution and partial volume effect. Technical development has provided various options for plaque imaging such as 3D fast spin echo (FSE) imaging with variable flip angle refocusing pulse (VFA). Pseudo-steady state can be achieved by prospectively controlled signal decay at the beginning of the echo train; thus, constant signal intensity is maintained [6]. Black blood effect is brought about since phase dispersion arises from the intravoxel blood flow velocity variation as well as the uncompensated first-order gradient moment during each echo readout [7]. Further phase dispersion occurs due to stimulated echoes introduced by the low-flip-angle refocusing pulses [8, 9].

3D MR imaging with high spatial resolution and improved anatomic coverage became possible by optimized and efficient k-space trajectories with sampling in both in-plane and through-slab phase-encode directions [10]. Higher acceleration in phase and partition direction can be applied for 3D FSE with various techniques including compressed sensing [11, 12]; thus, wide coverage and scan time reduction become possible. Availability of multiplanar reconstruction (MPR) for suitable visualization of vessel wall is another advantage of 3D imaging, and both vessel segment focused VW-MRI and whole brain VW-MRI have been performed recently [13].

VW-MRI techniques rely on blood flow to achieve blood-signal suppression. Incomplete signal suppression in the periphery of the lumen can mimic vessel wall thickening and/or wall enhancement. Recirculating or slow flow within an aneurysm, low velocity flow in a dilated artery, and retrograde filling of a branch artery via leptomeningeal collaterals may result in incomplete signal suppression [14, 15]. Therefore, further intravoxel dephasing effect can be brought about by additional preparation pulses and followings have been developed to achieve better black blood effect as well as cerebrospinal fluid (CSF) suppression: Diffusion-sensitizing gradient preparation (motion-sensitized driven equilibrium, MSDE) [7, 16], T2-prepared inversion recovery [17], a flip-down radiofrequency pulse module [18], and delay alternating with nutation for tailored excitation (DANTE) [19, 20]. Although many articles using VW-MRI with MSDE have been reported, DANTE has been reported to produce better signal-to-noise ratio (SNR) compared with MSDE [20].

DANTE pulse is a continuous irradiation of short duration hard pulse with small flip angle, and it has been used for frequency selective excitation in NMR spectroscopy [21] and cardiac tagging [22]. Recently DANTE pulse is used for VW-MRI with the advantage of signal suppression of both blood flow and CSF signal [20, 23–26]. CSF is known to affect vessel walls [27]. DANTE pulse is also used for intravascular signal suppression of arterial spin labeling [28], detection of brain metastasis [29], and neuromelanin-sensitive MRI due to certain magnetization transfer (MT) effect [30]. With acceleration of parallel imaging, high-resolution vessel wall imaging using DANTE pulse can be available in clinically feasible scan time [30, 31].

**Deep learning application for VW-MRI**

Deep learning technique has been introduced for acute ischemic stroke and plaque identification. Hyperdense middle cerebral artery (MCA) sign on CT is a well-known sign of acute embolism, and nowadays deep learning-assisted identification has been introduced [32]. Moreover, convolutional neural network (CNN)-based domain adaptive lesion classification could locate target arteries and distinguish carotid atherosclerotic lesions [33].

Deep learning can be used for improvement of SNR, and CNN improved overall image quality for high-resolution VW-MRI [34].
Clinical application of VW-MRI—arterial diseases

Carotid artery plaque

Rupture of vulnerable plaque is known to be an important cause of stroke rather than the luminal stenosis [35]. Chronic inflammation occurs with atherosclerosis, which is associated with the accumulation of lipids in the vessel wall and the formation of fibrous tissue [36]. Characteristics of carotid artery plaques such as intraplaque hemorrhage (IPH), a large lipid-rich necrotic core (LRNC), and a thin or ruptured fibrous cap (TRFC) are associated with cerebrovascular symptoms [37]. It is also reported that enlargement of an atherosclerotic artery with outward plaque growth or expansive remodeling might be an important indicator of high-risk plaque [38, 39].

VW-MRI is used to assess the morphology and characteristics of carotid artery plaques [5, 40, 41]. 2D spin echo T1WI technique used to be applied for intraplaque components of carotid artery plaque [40]; however, recent MRI studies reported usefulness of 3D MRI in detection of hyperintensity plaque representing IPH, low intensity plaque representing LRNC, and very low intensity representing calcification [42] (Fig. 1).

Acute ICA occlusion

Acute ICA occlusion usually causes severe long-term consequences [43]. There is no gold standard for differentiating acute from chronic carotid occlusion. Acute extracranial ICA occlusions were certainly defined when the estimated time of occlusion was within 7 days prior to CTA [44]. A ring of contrast enhancement in the carotid wall surrounding a hypodense thrombus in the lumen may help differentiate acute from chronic carotid artery occlusion [44]. On ultrasound examination, a mass arising from the ICA that fills the lumen and oscillates with the cardiac cycle is called an "oscillating thrombus" and is considered a specific finding of acute embolic internal carotid occlusive disease [45–47] (Fig. 2).

Chronic ICA occlusion

Chronic ICA occlusion (ICAO) is usually formed based on progressive atherosclerosis at the bifurcation of the carotid artery. Both symptomatic and asymptomatic chronic ICAO patients are at high risk for stroke [48]. As the occlusion duration gets longer, the thrombus gradually becomes fibrotic or calcified, and the occluded segments of ICA undergo atrophy. The atherosclerotic lesion typically develops from proximal part. The efficacy of carotid endarterectomy (CEA) has been established in symptomatic patients with moderate and greater stenosis [49].

VW-MRI showed that the cervical and petrous segments of ICA are commonly involved in patients with symptomatic and asymptomatic chronic carotid artery occlusion [50]. VW-MRI also revealed that atrophic ICA lead to decreased ipsilateral-to-contralateral diameter ratios at the cervical and petrous segments of ICA, which reduced endovascular recanalization success [48]. In 9 of 13 patients with symptomatic chronic carotid artery occlusion, VW-MRI showed contrast enhancement of the thrombus [50].

Diagnostic utility of VW-MRI in stroke

VW-MRI has provided supplemental information to luminal imaging [51], but diagnostic utility of VW-MRI in the work-up of ischemic stroke has also been reported. Kesav et al. reported that VW-MRI reclassified etiology and influenced diagnostic evaluation in cases originally

Fig. 1 A 75-year-old male with right ICA stenosis. a TOF-MRA shows stenosis of the proximal part of right ICA (arrow). b Axial MPR section of DANTE T1-SPACE shows hyperintensity plaque at the right ICA (arrow). c Coronal MPR section of DANTE T1-SPACE shows hyperintensity plaque at the right ICA (arrow)
classified as “undetermined” etiologies and large (intracranial) artery atherosclerosis [52]. Song et al. reported that VW-MRI changed etiologic classification, resulting in a higher percentage of cases reclassified as intracranial atherosclerotic disease [51]. VW-MRI can significantly improve diagnostic differentiation of intracranial vascular disorders compared with luminal imaging alone [51, 53, 54].

Specifically, vessel wall enhancement is important in diagnostic differentiation. Vessel wall enhancement may be associated with the culprit plaque in acute ischemic stroke. VW-MRI revealed vessel wall enhancement in 28 of 48 patients with acute ischemic stroke or transient ischemia attack [55]. Hyperintense plaques and plaque surface irregularity may predict A-to-A embolic infarction [56]. Meta-analysis of VW-MRI showed plaques were detected in about half of acute ischemic stroke patients with non-stenotic intracranial MRA [57]. Intracranial high-risk plaque with zero or mild stenosis is associated with ischemic stroke and unfavorable outcome [57]. VW-MRI detected peri-thrombus vascular hyperintensity sign, tubular or tortuous hyperintensity surrounding a filling defect (intravascular thrombus), in 49% of acute ischemic stroke patients [58].

The presence and intensity of vessel wall enhancement has been reported to help differentiate reversible cerebral vasoconstriction syndrome (RCVS) from vasculitis and atherosclerosis [54]. Vessel wall enhancement is associated with both acute and future stroke in patients with cerebral amyloid angiopathy [59].

**Dissection**

Cervical artery dissection (CAD) affects the cervical portion of the internal carotid artery (ICAD), the vertebral artery (VAD), or both. Cervical pain is often seen in VAD, and headache is seen in ICAD and VAD. The incidence of ICAD is estimated to be slightly higher than that of VAD [60]. CAD is a major cause of ischemic stroke in the young, and intramural hematoma detection significantly contributed to acute ischemic stroke pathogenesis in patients with suspected CAD [61, 62]. ICA dissection occurs more often in the intracranial segment than in the cervical segment of the carotid artery [63].

Intracranial artery dissection, which is most common in Asia, accounted for up to 67–78% of all cervicocephalic artery dissections [43] (Fig. 3). Intracranial artery...
dissections often affect the posterior circulation, especially at the intradural portion, more frequently than the anterior circulation [64, 65]. Intracranial cerebral artery dissection of the anterior circulation was reported to be in relation with cortical subarachnoid hemorrhage (SAH) [66]. Intradural arteries are characterized by a well-developed internal elastic lamina, a paucity of elastic fibers in the media, little adventitial tissue, and no external elastic lamina [67], which may result in weaker supporting tissues than cervical arteries and may be associated with SAH [68]. (Fig. 4).

On VW-MRI, intramural hematoma is iso-intensity during acute phase of CAD, subsequently become hyperintensity a few days after the onset until about 2 months or later. Follow-up imaging is necessary for CAD, and intramural hematoma usually heal within 3–6 months.

**Carotid web**

A carotid web is a thin intraluminal filling defect along the posterior wall of the carotid bulb observed on CTA or DSA. Carotid web may contribute to recurrent ipsilateral ischemic stroke in patients with no other determined stroke risks [69]. During a 12-month period, ipsilateral carotid webs were identified prospectively in 7 patients with acute ischemic stroke at the single institute [69]. In another series, carotid artery webs were found in 2 of 132 symptomatic patients with suspected stroke and in 7 of 312 asymptomatic patients [70]. Pathological analysis for carotid web showed marked fibroelastic thickening of the intima. Although the incidence of carotid web is low, and CTA is the better tool for detection of carotid web, VW-MRI may depict the presence of carotid web (Fig. 5). Neuroradiologists should check the abnormalities of carotid bulb, especially in patients with recurrent ipsilateral stroke.

**Aneurysm**

MRA is suitable for serial follow-up of aneurysms and recent progress of compressed sensing technique enables high resolution MRA with wide coverage in clinical routines [71, 72].

Although MRA or CTA can be easily performed and they provide intraluminal characteristics, VW-MRI provides the characteristics of aneurysmal wall. Wall thickening with enhancement was associated with unstable (ruptured, symptomatic, or undergoing morphological modification) intracranial aneurysms [73]. Several mechanisms including inflammatory response, vasa vasorum, atherosclerosis, and intramural hematoma may cause aneurysmal wall enhancement. Whether aneurysmal wall enhancement on VW-MRI represents inflammatory process or not has not been answered yet [74]. Wall
enhancement might imply fragility of the aneurysm wall which leads to remodeling, thinning, and daughter sac formation [75]. In terms of the size of aneurysms, wall enhancement was noted on all large aneurysms (≥ 7 mm) and 67% (20/30) of the small aneurysms (< 7 mm) [76].

**Angiogram-negative SAH**

Angiogram-negative SAH comprises approximately 15% of SAH case in which no causative vascular abnormality was found on angiography [77]. Angiogram-negative SAH can be classified into two subgroups: one is perimesencephalic SAH in which distribution of SAH is observed at the perimesencephalic region with low risk of recurrent hemorrhage and excellent clinical outcome. The other is non-perimesencephalic diffuse SAH in which angiogram-negative SAH may develop hydrocephalus, vasospasm, rebleeding, which results in poor clinical outcome due to the presence of undetected vascular abnormalities [78].

Recent retrospective study showed VW-MRI revealed that abnormal findings such as dissection and blood blister-like aneurysm in 14 out of 17 patients with diffuse non-aneurysmal SAH [23].

**AVM**

Brain AVM is an abnormal connection between arteries and veins existing in the brain parenchyma without intervening capillary beds. The transition between artery and vein is called as a nidus. The risk of hemorrhage is associated with deep venous drainage, and deep and infratentorial brain location [79], and demographically children and females [80]. TOF-MRA with wide coverage like a whole brain MRA is necessary to evaluate feeders and nidus of AVM [81]. CE-MRA or MR-DSA is also performed for evaluation of brain MRA [82].

VW-MRI is also considered important for evaluation of thrombus formation in nidus and the rupture risk of AVM. Comparison of VW-MRI and histopathology findings in a ruptured AVM revealed luminal thrombus in the vessel wall, fibrin deposition inside and outside the vessel, and inflammatory cell infiltration [83]. VW-MRI demonstrates nidal enhancement and perivascular enhancement adjacent to the nidus even in unruptured AVM [84]. Although enhancement on VW-MRI may represent remodeled vessel wall without active inflammation as well as true persistent inflammatory changes, further longitudinal studies are required [84].
Moyamoya disease

3 T MRA clearly visualizes moyamoya vessels with the advantage of T1 elongation and MT effect [85]. Intraluminal flow is often evaluated with TOF-MRA in moyamoya disease, and recent technical improvement such as compressed sensing enables us to perform TOF MRA with wide coverage [86]. Although initial studies for a small number of cases reported lack or weak contrast enhancement of vessel wall [87, 88], recent studies showed a high frequency of ICA and MCA wall enhancement [89]. Another study revealed negative remodeling of the vessels in moyamoya patients [90]. However, most previous studies have focused on differentiating moyamoya disease from atherosclerotic disease, and only a limited number of studies have focused on the relationship between VW-MRI and the disease activity [91]. Consequently, the clinical usefulness of VW-MRI for moyamoya disease is not established yet.

Reversible cerebral vasoconstriction syndrome (RCVS)

RCVS is characterized by severe headaches, with or without other acute neurological symptoms, and diffuse segmental constriction of cerebral arteries that resolves spontaneously within 3 months [92]. A thunderclap headache is a severe pain that peaks within seconds and usually recurs for one to two weeks [92, 93]. Patients typically report at least one trigger such as sexual activity, straining during defecation, stressful or emotional situations, physical exertion, coughing, sneezing, urination, bathing, showering, swimming, laughing, sudden bending down, postpartum state, pre-eclampsia, recreational drugs, vasoactive substances, and antidepressants [92, 94]. Imaging abnormalities include cortical SAH, cerebral infarction, intracerebral hemorrhage, and reversible brain edema [92, 94]. Diagnostic criteria include the demonstration of segmental vasoconstriction. It is worth noting that even in the presence of hemorrhage or cerebral edema, initial angiogram may be normal if the examination is performed early and vasoconstriction can be difficult to detect in very distal branches [95, 96]. Follow-up examination should be performed several days later for detection of vasoconstriction if RCVS is clinically suspected. VW-MRI of RCVS shows minimal to no enhancement and minimal wall thickening [54]. VW-MRI may help distinguish RCVS from vasculitis and intracranial atherosclerosis.

Clinical application of VW-MRI—venous disease

Venous structures

Venous structures are usually evaluated with susceptibility-weighted imaging (SWI). SWI is a high-spatial-resolution three-dimensional (3D) gradient echo MR technique that exploits the magnetic susceptibility differences. SWI shows deoxyhemoglobin inside the veins due to its paramagnetic property [97]. SWI visualizes hypointense venous structures in acute large arterial infarction probably due to increase of deoxyhemoglobin and dilatation of veins [98]. VW-MRI is also useful for evaluation of venous structures. The positive findings of venous thrombus used to be non-filling of venous sinus or cerebral vein on CE-CT or CE-MRI. On the other hand, VW-MRI can show the thrombus as evident high signal intensity even in the subacute stage of venous thrombosis without administration of contrast media [99] (Fig. 6).
Clinical application of VW-MRI—vasculitis

CNS vasculitis

Adult primary angiitis of the central nervous system (PACNS) is a heterogeneous disease although secondary CNS vasculitis is ruled out with complete work-up for malignancies, cardiopathy, systemic vasculitis, and connective tissue disorders. Most of PACNS shows multi-territorial, bilateral, distal acute stroke lesions with small to medium artery distribution, and a predominant carotid artery distribution [100]. Hemorrhagic infarctions and parenchymal hemorrhages were also frequently found [100]. Occasionally PACNS showed tumor-like appearance characterized with mainly small-sized vessel disease mimicking primary CNS lymphoma; however, global outcomes are good under appropriate treatment [101]. Tumor-like PACNS can be seen in younger patients compared with the other PACNS and accompanies seizure, and more enhancement on CE MRI [101].

VW-MRI revealed a concentric contrast enhancement of arterial walls, localized in multiple vascular territories in patient with PACNS [102, 103]. According to the systematic review of CNS vasculitis, features of VW-MRI for vasculitis affecting the intracranial and extracranial arteries included vessel wall enhancement (89%), vessel wall thickening (72%), vessel wall edema (10%), or perivascular enhancement (16%) [104].

Giant cell arteritis (GCA)

GCA or temporal arteritis is the most common idiopathic large vessel vasculitis as well as Takayasu arteritis. Patients are usually greater than 50 years of age, and it mainly affects the thoracic and abdominal aorta, and its primary branches. The etiology and pathogenesis of GCA are still unknown. Classic cranial manifestations consist of headache, scalp tenderness, jaw claudication, and vision loss. Vision loss occurs in approximately 20% of patients with GCA and immediate diagnosis and early initiation of intravenous high-dose corticosteroid therapy are required [105]. Stroke or transient ischemic attack occurs in 1.5–7% of patients with GCA and are caused by stenosis or occlusion of the extradural vertebral or carotid arteries [105, 106]. GCA tends to affect arteries with elastic tissue in their wall, whereas intradural arteries contain little or no elastic tissue. Inflammatory cells enter the vessel wall through vasa vasorum which is less in intradural arteries. These are thought to be the reasons why intracranial lesions of GCA are rare. Temporal artery biopsy remains the gold standard for diagnosis of GCA.

3D VW-MRI increased diagnostic accuracy of GCA compared with 2D VW-MRI [107]. 18F fluorodeoxyglucose positron emission tomography (FDG-PET)/CT is also useful for the diagnosis, therapy response assessment, and prognosis of GCA [108]. Arteritic anterior ischemic optic neuropathy (A-AION) is the most common cause for permanent vision impairment in patients with GCA [105]. A-AION is caused by arteritic ischemia of the anterior part of the optic nerve secondary to inflammatory occlusion of the posterior ciliary arteries. VW-MRI revealed a strong and blurry contrast enhancement aside the optic nerve and the adjacent orbital fat following the course of the posterior ciliary arteries [109].

Other vasculitis and inflammatory diseases

VW-MRI can depict inflammatory changes in a wide range of secondary vasculitis, including radiation-induced and those associated with infectious disease such as the human immunodeficiency virus (HIV), syphilis [110], herpes [111], and varicella zoster (Fig. 7). Preliminary findings obtained with VW-MRI also suggested a possible inflammatory
mechanism underlying a percentage of cryptogenic stroke in coronavirus disease 2019 (COVID-19) patients [112]. VW-MRI can identify inflamed intracranial vessels, enabling precise localization of biopsy targets [113]. MR findings are important in the management of infectious diseases [114], and VW-MRI may add values in diagnostic workups.

Clinical application of VW-MRI—leptomeningeal diseases

Leptomeningeal diseases

Image sequences used for VW-MRI are spin echo-based pulse sequences that have relatively lower signal intensity in white matter, in part due to MT effects [115]. In addition, intravascular signal suppression facilitates detection of microscopic metastases and leptomeningeal carcinomatosis [116].

3D CE VW-MRI showed a higher sensitivity than CE gradient echo MRI in detection of leptomeningeal carcinomatosis [117] (Fig. 8).

Conclusion

VW-MRI has been applied in clinical practices not only in evaluation of vulnerable plaques, but various kinds of cerebrovascular diseases, vasculitis, and other diseases. High-resolution 3D VW-MRI with good SNR has been available with or without additional preparation pulses. VW-MRI in addition to the routine MR imaging protocols may lead to better diagnostic workup when necessary.

Fig. 7 A 60-year-old male with right facial nerve palsy and neuralgia. CE DANTE T1-SPACE (a, c) and CE GRE T1WI (b, d) are shown. He was diagnosed as Ramsay-Hunt syndrome, and CSF analysis demonstrated varicella zoster meningitis. (a, c) CE DANTE T1-SPACE showed enhancement of right facial nerve and geniculate ganglion (arrows). (b, d) CE GRE T1WI showed less pronounced enhancement in the facial nerve (arrows). Note that non-specific mild enhancement is often seen in geniculate ganglion, but the enhancement is more pronounced in this case.

Fig. 8 A 10-year-old girl with CNS dissemination of acute lymphocytic leukemia. CE DANTE T1-SPACE showed enhancement of bilateral vestibular nerves (white arrows), abducens nerves (white arrowheads) a, trigeminal nerves (black arrows) b, and oculomotor nerves (white double arrows) c. CE DANTE T1-SPACE showed leptomeningeal enhancement at the left occipital lobe (black arrowheads) (a, b).
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