"High-Resolution MR Intracranial Vessel Wall Imaging - State of the art"

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Authors: C. Tolman; The Hague/NL
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Learning objectives

- Introduce High Resolution Magnetic Resonance Intracranial Wall Vessel Imaging (HR MR IVWI).
- Illustrate the value of IVWI complementary to CTA, MRA and DSA with a case series from our institute.
- Recognize specific imaging characteristics of atherosclerosis, vasculitis and reversible cerebral vasoconstriction syndrome (RCVS) and differentiate amongst these.
- Evaluate different "black blood" MRI techniques to optimize the imaging protocol for IVWI.
- Inform about the newest 2D and 3D HR MR IVWI techniques in literature, discuss limitations and propose future developments.
Background

Intracranial Vessel Wall Imaging (IVWI) is based on the analysis of the vascular wall by subtraction of the signal of blood in the arteries. This technique has been used for many years in cardiac MRI, to evaluate the temporal artery in giant cell arteritis and to calculate the composition and risk profile of atherosclerotic plaques in carotid arteries (the presence of a fibrous cap, intraplaque hemorrhage, neovascularity and ulceration).

IVWI complements traditional luminal imaging techniques CTA, MRA and DSA; these are limited by nonspecific angiographic findings since a number of vasculopathies can present with similar luminal patterns, such as vasculitis, reversible cerebral vasoconstriction syndrome (RCVS), intracranial atherosclerotic disease (ICAD), vasospasm, infection, and radiation-related vasculopathy. Furthermore, DSA has a low sensitivity for small-vessel vasculitis DSA (30%) and CTA and MRA have a low detection rate for non-stenotic pathology, such as positive remodeling in atherosclerosis. Moreover, biopsy for vasculitis is invasive and carries high-risk complications. HR-MR IVWI improves the existing diagnostic tools for differentiating intracranial arteriopathies, especially non-stenotic and small vessel vasculopathy.

Commonly used black-blood techniques are Turbo Spin Echo and Inversion Recovery. Furthermore, high resolution 3D techniques are evolving every day (eg. VRFA, MSDE, DANTE). In our hospital, we use the "poor man's" black blood technique Turbo Spin Echo (TSE), which is not that fancy, but with a few more sequences you will be able to give your clinician that piece of extra information to lead to the correct diagnosis. Our "vessel wall imaging" protocol consists of T2 and T2 TIRM series, proton density, diffusion weighted imaging and a SWI or haem series. We perform a MR-angiography Time of flight (TOF) with MIP/MPR reconstructions. The extra Turbo Spin Echo Black-blood series are T1 weighted images with fat saturation, both pre- and post intravenous gadolineum contrast.

In short something about the TSE technique (Fig.1): Turbo spin echo pulse sequences have inherent blood nulling properties, even without black blood preparation pulses. In order to contribute signal to the image, a given spin must be subjected to a 90° excitation and 180° refocusing pulse. Mobile spins such as blood may move out of plane in between these two slice selective pulses and therefore will not experience the 180° refocusing pulse and generate a spin-echo. This "washout effect" leads fast moving spins to appear black in the final image. Despite the inherent blood suppression of TSE sequences, black blood preparation pulses are used to ensure complete and homogenous blood nulling, especially for slow moving or in-plane flow.
Fig. 1: Background theory of Turbo Spin Echo.

Findings and procedure details

IVWI can differentiate atherosclerosis, vasculitis and reversible cerebral vasoconstriction syndrome (RCVS) based on features such as wall thickening (concentric vs. eccentric, smooth vs. irregular), distribution (proximal arteries vs. medium- small vessels), wall enhancement (degree and homogeneity) and follow -up (progressive vs. reversible). This is illustrated by a case series from our institute. Other vasculopathies are dissection and Moya Moya disease; however both entities have a very specific appearance on CTA and DSA, and we found IVWI less helpful in the practical faily radiological workup. Therefore, we will not discuss them here.

Case 1

The first case was a 50-year old woman who presented in the ER with an acute leftsided hemiparesis and dysarthria. A non-enhanced CT scan of the head (Fig.2) showed recent ischemia in the right caudate nucleus and old ischemia in the left basal ganglia. Subsequent CTA (Fig.2)demonstrated less enhancing M2 segments of the medial cerebral artery on the right, suspect for occlusion (although no acute stop was observed). Laboratory results and liquor were normal. Patient went for Diagnostic Subtraction Angiography (DSA, Fig.2), which showed luxury perfusion and narrowing of the frontal segments of the right medial cerebral artery. No thrombus was seen and no thrombectomy was performed. Differential diagnosis at the time was Reversible Cerebral Vasoconstriction Syndrome (RCVS) or vasculitis. During admission, patient improved in speech and also in her loss of strength. MRI confirmed recent ischemia in the right lentiform nucleus and thalamus and old infarcts in the left caudate and lentiform nucleus (Fig.3). Also, we performed black-blood vessel-wall imaging: There was intense enhancement after contrast on T1 images of short segments of proximal medial and anterior artery on both sides, compared to the non-enhanced black-blood images (Fig.3). Ultrasound of the heart, laboratory results (AN(C)A) were negative. Finally, serology and liquor turned out positive for Borrelia IgG and the diagnosis vasculitis / neuroborreliosis could be set.

Case 2

The second case was a 72-year old man with a left hemiparesis since one day. His CT showed a multi-infarct brain (not showed here) and his CT-angiography demonstrated multiple stenoses and dilatations in the medial cerebral artery and basilar artery, unchanged since 2011 (Fig.4). Differential diagnosis was atherosclerosis. We performed MRI with extra black-blood vessel wall imaging series. There was no diffusion restriction and MR-angiography TOF showed irregular calibre changes. There was no enhancement on black-blood imaging after IV-contrast, nor intraplaque bright T1 hemorrhage. The diagnosis was diffuse atherosclerosis without any recent ischemia or acute occlusions.
**Atherosclerosis**

The specific imaging characteristics of atherosclerosis on IVWI are depicted in figure 5. The shape of the wall in atherosclerosis is eccentric (positive remodeling) and irregular. Any artery can be involved, but mostly the larger vessels are involved: The distal internal carotid artery and proximal medial and anterior (and posterior) cerebral artery are affected most. The signal intensity of the vessel wall is highly variable, dependent on the components of the vessel wall: A lipid core is iso-intens on T1WI and hypo-intens on T2WI, a fibrous cap gives a plaque an iso T1 and T2 appearance, intraplaque hemorrhage is hyperintens on T1WI and hard plaques containing many course calcifications are mainly dark on T1 and T2WI. Enhancement is variable and often heterogeneous considering its heterogeneous contents. Symptomatic plaques demonstrate a higher proportion of contrast enhancement than asymptomatic plaques. On follow-up there is no resolution of vessel wall abnormalities; On the contrary, atherosclerosis is a continuous, progressive process.

**Vasculitis**

Another example of intracranial vasculitis from the literature (Fig.6) demonstrates smooth, circumferential or concentric, intense enhancement of the right distal internal carotid artery and proximal medial artery (M1 segment), correlated with an (aspecific) stenosis on MRA TOF. After treatment with steroids and appropriate antibiotics the patient improved clinically and so did the imaging findings. After 12 months no stenosis nor pathological enhancement could be seen anymore (Fig.6).

In case of multiple ischemic strokes with different ages in different vascular territories, think of vasculitis. The specific imaging characteristics of vasculitis on IVWI are depicted in figure 7. By definition, medium-sized arteries are located distal to the bifurcation of the middle and anterior cerebral artery. IVWI directly visualizes wall inflammation and edema of medium- to small sized intracranial arteries by showing vessel wall thickening and multifocal homogeneous, smooth, intense, concentric enhancement of the vessel wall. This pattern is in contradistinction to that seen in intracranial atherosclerosis, in which there is eccentric, expansive wall involvement and more heterogeneous enhancement. Usually, the findings on MRI resolve by treatment with immunosuppressives and antibiotics; therefore IVWI can track treatment response on follow-up MRI scans. Moreover, it may guide surgeons to biopsy in the areas of active disease on MRI. In future, IVWI may even limit the need for invasive biopsy.

**RCVS**

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Reversible cerebral vasoconstriction syndrome (RCVS) is the second arteriopathy that, like vasculitis, involves mainly the medium- to small sized vessels. The RCVS syndrome describes a cluster of symptoms (headache, neurological deficits, luminal narrowing and spontaneous reversal), caused by non-inflammatory vasospasms. The exact pathophysiology remains unclear. Although considered a benign self-limiting entity, possible complications of RCVS include infarction, edema and hemorrhage. In an example from literature, a middle-aged patient presented with acute severe "thunderclap" headache with no neurological deficits. The differential diagnosis was an aneurysmal subarachnoid hemorrhage or sinus thrombosis. IVWI showed mild wall thickening and enhancement, with a uniform, diffuse distribution, involving bilateral A1, M1, P2 and PCOM segments (Fig.8). The involved areas showed luminal narrowing on the MR angiography. Diagnostic work-up for vasculitis was negative. On follow-up MRI after 3 months the stenoses and wall thickening vanished with some minimal residual wall enhancement of the M1 segments (Fig.8).

RCVS and intracranial vasculitis share both clinical and radiological features. However, radiological findings in RCVS are much milder; a pattern of diffuse, uniform and continuous wall involvement is seen, with minimal concentric thickening and with or without mild enhancement throughout the entire wall of the diseased vessels (Fig.9). Shortening of smooth muscle cells that results in increased wall thickness and luminal stenosis may explain the wall thickening observed on HR-MR IVWI. Leakage of contrast through the BBB or edema and subsequent enhancement are probable explanations for the weak enhancement seen on MRI. In RCVS, there is near uniform resolution of imaging findings within 3 months of onset, while vasculitis restores in about 7-17 months. Finally, RCVS has a higher rate of resolution compared to vasculitis (88.9% vs. 33.3%); in vasculitis, multifocal areas of enhancement diminish, but stenoses often persist.
Images for this section:

**Fig. 2:** CT, CTA and DSA of case 1.

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**Fig. 3:** MRI, MRA Time of flight (TOF) and black blood vessel wall imaging (VWI) of case 1.

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**Fig. 4:** case 2: MR-angiography Time of Flight and additional Black blood pre- and post-contrast T1 weighted images.

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<th>Atherosclerosis</th>
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**Fig. 5:** Table with characteristics of VWI, highlighted for atherosclerosis.
Fig. 6: Example of vessel wall imaging for intracranial vasculitis.

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**Fig. 7:** Table with characteristics of VWI, highlighted for vasculitis.

Fig. 8: Example of vessel wall imaging for reversible vasoconstriction syndrome (RCVS).

**Fig. 9:** Table with characteristics of VWI, highlighted for reversible vasoconstriction syndrome.

Conclusion

Intracranial vessel wall imaging (IVWI) is an emerging technique that focuses on intracranial arteriopathies, and depicts vessel wall abnormalities, in addition to the traditional luminal techniques. It is a welcome addition to CTA, MRA and DSA to differentiate a.o. between atherosclerosis, vasculitis and RCVS. Our HR MRI protocol includes four different scans: black blood T1-weighted pre- and post IV contrast series, T2-weighted and proton density-weighted MRI, as well as three-dimensional turbo spin echo imaging techniques with multiplanar reconstruction. Despite current limitations such as some overlapping radiological findings, the question arises: Can high resolution MR IVWI replace invasive techniques such as DSA and biopsy in the future?
References