# High-Resolution Vessel Wall MRI as a Complementary Investigation for CNS Tuberculosis

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Tuberculous meningitis (TBM) has been associated with vascular arterial damage, caused by infiltrative, proliferative, and necrotizing pathology.[1] Autoimmune mechanisms have also been suspected in these cases since they can appear even during effective treatment. The recent development of a new magnetic resonance imaging (MRI) sequence – the high-resolution vessel wall imaging (HR-VWI) – has allowed submillimeter MRI evaluation of arterial walls[2–4] and can contribute to the diagnosis of this disease.

The diagnosis of tuberculosis of the central nervous system can be difficult. Cerebrospinal fluid (CSF) acid-fast smears and culture have low sensitivity, and CSF nucleic acid amplification tests are highly specific but also lack sensitivity.[5] Some studies have demonstrated poor specificity of adenosine deaminase (ADA) for TBM diagnosis in certain populations, particularly in HIV-infected adults with concurrent infections or cerebral lymphomas.[5] Treatment is recommended upon a clinical, preliminary CSF test and imaging suspicion.

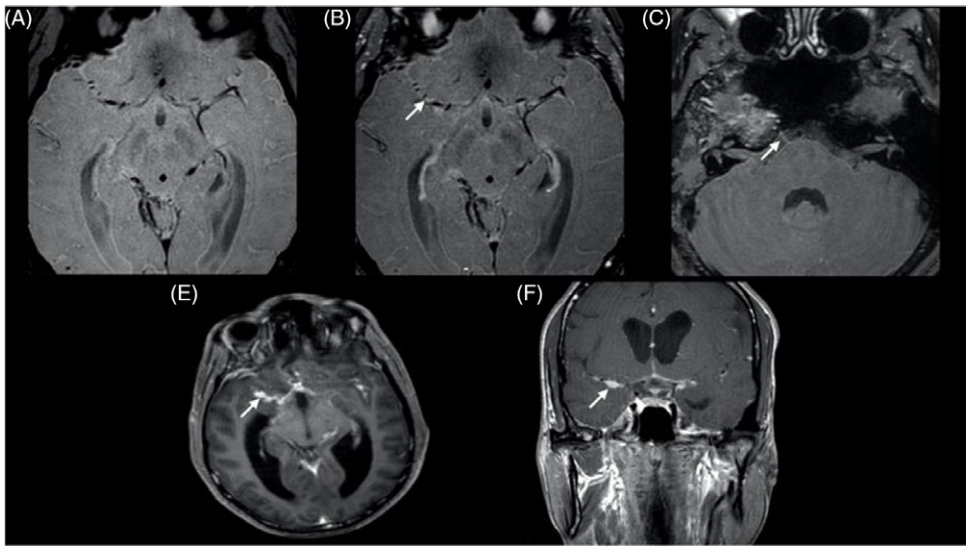


***Figure 1:*** *Axial HR-VWI (Achieva 3.0T; Philips Healthcare, the Netherlands; RT, 600 ms; ET, 29 ms; SPAIR; reconstruction matrix, 320; FoV, 120 mm; voxel size 0.8 × 0.8 × 0.8 mm$^3$; blood suppression, VISTA) in A (without contrast), B and C (post-contrast) showing wall enhancement of the middle cerebral arteries (arrow in B) and also enhancement of the VI right cranial nerve (arrow in C). D (axial) and E (coronal) T1-weighted image post-contrast in D (axial) and E (coronal) depicting basal meningeal enhancement and also leptomeningeal thickening and enhancement along Sylvian fissures (arrows).*

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We report a case of confirmed TBM, with clinical and radiological presentations and a good outcome after treatment.

A 33-year-old man presented with a history of acute headache, mental confusion, and significant weight loss. The emergency room evaluation showed no motor deficits or other significant neurologic examination findings. He had not been adherent to the treatment for AIDS in the last 7 years. Pulmonary tuberculosis was also diagnosed recently, but he had quit treatment. HIV viral load was log 6.03 copies/ml, CD4 count 33 cells/ml, and sputum TB Rt-PCR was positive. Initial brain MRI showed diffuse leptomeningeal enhancement of the basal cisterns and the Sylvian fissure, hydrocephalus, and multiple cranial nerve enhancement. Complementary HR-VWI showed enhancement of the walls of right middle cerebral artery, consistent with vasculitis and cranial nerve enhancement (Figure 1). The patient was treated with Rifabutin, Isoniazid, Ethambutol, Pyrazinamide, and Prednisone as well as highly active antiretroviral therapy. The patient was discharged 1 month after admission with no significant neurological deficits. The current viral load is 1.7 copies/ml and CD4 count is 401 cells/ml.

TBM is a common manifestation of intracranial tuberculosis. Ischemic lesions secondary to arterial involvement and cranial neuropathies[1] are common complications of basal TBM involvement adding morbidity and mortality. The angiographic pattern already described consists of narrowing of arteries at the base of the brain and narrowed small or medium-sized arteries.[1] HR-VWI complements luminal imaging and can differentiate many potential causes of luminal narrowing.[2–4] Vessel wall enhancement usually corresponds to expected patterns of vessel wall inflammation and/or increased *vasa vasorum* density in distinct vascular pathologies[2] and can also detect cranial nerve enhancement,[4] as illustrated in this report. MRI with HR-VWI is a relevant tool to demonstrate TBM arteritis and can identify vessel wall enhancement without changes in the vessel caliber.

## Disclosures

The authors have no disclosures related to this work.

## Statement of Authorship

LMF, VMJ – manuscript writing and editing and image selection. MCR – manuscript review. FR – manuscript writing, manuscript review, and image selection.

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