(MS) in patients presenting with both non-enhancing and enhancing brain lesions, cranial nerve enhancement and spinal cord lesions.
CASE REPORT
A 37-year-old woman was hospitalized because of rapidly progressive neurological symptoms. Clinical history and examination revealed blurred vision, left facial numbness, left-sided hearing loss, dysesthesia in both legs, as well as gait ataxia, and fatic disturbances. Previous medical history included systemic sarcoidosis with panuveitis and vasculitis.
Brain MR imaging demonstrated multiple T2 and FLAIR hyperintense lesions in the periventricular and juxtacortical white matter. Non-enhancing, homogeneous and ringlike enhancing lesions as well as open-ring enhancing lesions were observed.
Cranial nerve enhancement of the left trigeminal nerve, including the intrapontine trajectory, was noticed. More subtle enhancement of the right trigeminal nerve, left facial nerve, optic nerve/chiasma and pituitary stalk enhancement was seen. Leptomeningeal enhancement was absent.
MR angiography was normal. Spinal MR imaging displayed gadolinium-enhancing high-signal intensity T2-lesions within the cervical and thoracic spinal cord. High-resolution computed tomography (HRCT) of the chest was negative.
Relevant laboratory findings included normal angiotensin converting enzyme (ACE) levels, unique oligoclonal IgG banding in the CSF and serum.
Repeat MR imaging of the brain after 3 and 5 weeks demonstrated a combined waxing and waning pattern of the lesions.
Because of the rapid progressive clinical deterrioration of the patient, brain biopsy was performed. Pathology revealed lymphocytic inflammatory changes in the white matter and leptomeninges. Myelin staining showed white matter demyelination. Final diagnosis of MS was established.
DISCUSSION
MS and neurosarcoidosis (NS) share MR imaging features: both non-enhancing and enhancing parenchymal lesions, cranial nerve enhancement and spinal cord involvement. Open-ring enhancement after gadolinium injection has been described in the literature as being very specific for demyelinating lesions.
CONCLUSION
Open-ring enhancement of white matter lesions is in our experience a key finding allowing to differentiate between the MR pattern of NS and MS, prompting the diagnosis of the latter, which was confirmed by the pathological findings.

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ANTI-NEUTROPHIL CYTOPLASMIC ANTIBODIES (ANCA)-ASSOCIATED VASCULITIS: MRI AND MRA FINDINGS

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PURPOSE
To describe a rare case of brain involvement in anti-neutrophil cytoplasmic antibodies (ANCA)-associated vasculitis.
METHODS
Multiplanar and multiecho MRI imaging of the brain was performed emphasizing the relative T1, T2 and DWIADC signal characteristics. TOF MRA of the intracranial arteries was obtained. Clinical data were retrieved from the patient's records.
RESULTS
A 61-year-old lady developed fever, widespread aches and pains, acral ischemia of the feet leading to multiple digit amputations, hemoptysis, and impaired consciousness. Laboratory tests showed raised inflammatory markers and positive cytoplasmic ANCA. 24-hour urinary protein was raised at 700 mg. Infective and autoimmune serology was otherwise negative. Chest CT showed bilateral, ground glass attenuation (GGA) consistent with pulmonary hemorrhage. Brain MRI disclosed restricted diffusion of the left hemisphere, consistent with subacute ischemic lesions, while MRA revealed multiple stenoses of the distal branches of the left middle cerebral artery.
ANCA-associated vasculitis was diagnosed and aggressive immunosuppressive therapy started with gradual clinical improvement which was confirmed by signal intensity alterations resolution.
CONCLUSIONS
Brain involvement is very rare in ANCA-associated vasculitis. MRI and MRA are very helpful in the differential diagnosis and in monitoring response to therapy.

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APLASIA OF THE INTERNAL CAROTID ARTERY: MRA FINDINGS

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PURPOSE
Aplasia and hypoplasia of the internal carotid artery (ICA) are rare congenital anomalies, mostly incidentally found. Symptomatic cases, usually present with acute ischemia. A number of intracranial vascular anomalies, such as aneurysms of the circle of Willis and abnormal collateral arteries are commonly associated findings. This report describes a young individual with left ICA agenesis, whose unusual presenting symptom was a mild discontinuous headache.
METHODS
A 8-year old boy was admitted in our hospital complaining of intermittent headaches. Brain magnetic resonance imaging and angiography (MRI and MRA), were performed which demonstrated absence of the left ICA.
RESULTS
The left ICA was found to be missing. The left middle cerebral artery arose from the basic artery with normal flow. The right posterior communicating artery was visualized, while both anterior cerebral arteries (A2 branches) arose from the right A1. Both vertebral arteries exhibited normal flow.
CONCLUSIONS
ICA aplasia/hypoplasia is most commonly incidentally discovered as no particular symptom suggests its presence. A daily, intermittent headache of few hours duration is uncommon. The