MR and CT Imaging, Part II

Differential diagnostic evaluation of white matter disorders

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Approach to imaging

I. Pattern recognition

II. Acute vs. chronic abnormalities

III. Longitudinal changes
I. Pattern recognition

Intra-cortical and deep gray matter lesions

1) Difficult to detect due to reduced contrast between normal and affected GM (compared to WM)
2) Majority of lesions occur adjacent to cortical veins
3) Intra-cortical demyelination occurs as much as WM demyelination in SP MS, and more than WM demyelination in PP MS.
4) GM lesions likely contribute independently to clinical disability

History / lab red flags

a. onset age < 10 or > 50
b. clinicoradiographic mismatch
c. hearing loss
d. progressive or acute onset
e. seizures at onset
f. simultaneous bilateral vision loss
g. complete transverse myelitis
h. elevated ESR
i. CSF protein > 100 and/or CSF WBC > 50
j. h/o rheum, autoimmune, or psych diagnoses
I. Pattern recognition

### History / lab red flags

- Age-related white matter changes
- Acute disseminated encephalomyelitis
- Behcet’s disease
- Bacterial infections (syphilis, Lyme disease)
- Central nervous system atrophy, subcortical
  - Atrophy, and leucodystrophy. KCD-MS
- Cervical spine/soft or denervation
- HIV infection
- Human T-lymphotrophic virus I / II
- Ischemic optic neuropathy (arteritic and nonarteritic)
- Leukodystrophies (e.g., adrenoleukodystrophy, metachromatic leukodystrophy)
- Neoplasms (e.g., lymphoma, glioma, meningioma)
- Trigeminal
- Systemic lupus erythematosus, antiphospholipid antibody
- Stroke and ischemic cerebralvascular disease and spinal cord infarction
- Unidentified bright objects
- Vascular malformations
- Vascularity (primary CNS or other)
- Vitamin B12 deficiency

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### Imaging red flags

- hypercellularity
- hemorrhage
- infarction
- necrosis
- unusual lesion appearance
- unusual enhancement pattern
- hypervascularity
- infiltration/membrane turnover

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### Neuromyelitis optica

Longitudinally extensive central cord lesions
Bilateral or sequential optic neuritis is more common than in MS
Sarcoidosis
CSF normal, no OCBs, serum ACE elevated
5-15% involvement of CNS

Behçet disease
CSF lymphocytic pleocytosis, no OCBs
Relapsing course, brainstem lesions are common

Cobalamin deficiency
Seen in pernicious anemia (anti-parietal cell Abs),
and after gastric bypass. Copper deficiency and
nitrous oxide exposure can produce similar lesions.

Lupus
+ANA, +dsDNA, elevated APLA IgM, and 3 OCBs
Primary CNS vasculitis
Lymphocytic pleocytosis, ESR nl, biopsy negative; relapsing course; can see enhancing lesions

CADASIL
Notch-3 gene mutation. +FH of early strokes

Anaplastic astrocytoma (multicentric)
Hypercellularity, hypervascularity, and necrosis are atypical for MS

Intravascular lymphoma
Small vessel vasculitis, CSF lymphocytic pleocytosis, angiogram negative. Skin and kidneys involvement.
II. Acute vs. chronic abnormalities

Re-identify all previously seen lesions (T1 & T2)

Identify any new lesions (T1 & T2)

Identify acute demyelination (T1-post & DWI)

Identify non-lesional volumetric changes (T1 & T2)

Reporting: interval or acute demyelination
II. Acute vs. chronic abnormalities

T1-post                      T2-FLAIR

II. Acute vs. chronic abnormalities

Radiographics 2006;26:S173

III. Longitudinal changes

Meier DS and Guttmann CR
III. Longitudinal changes

Can longitudinal MRIs predict clinical phenotype and future disability? Therapeutic response?

Parenchymal volume loss (absolute, rate of change)
- gray matter fraction
- white matter fraction
- cervical spinal cord

White and gray matter lesion burden
- T2 hyperintense lesions
- T1 hypointense lesions

Bakshi R et al, Lancet Neurol 2008;7:615

Quantifying atrophy in MS

Klein JP et al, AJNR 2011;32:1138
Quantifying atrophy in MS

Opposing pathological processes impact CNS volume in MS

- volume loss due to neuronal/axonal degeneration/gliosis
- volume gain due to inflammation/edema

Spot measurements of volume may be uninterpretable with respect to progression, disease activity, or prognosis.

Longitudinally, there is clearly accelerated volume loss in patients with all forms of MS compared to controls.
III. Longitudinal changes

MRI-based continuous scale as a marker of MS disease severity, the “MRDSS”

- T2 lesion volume (T2LV)
- T1 lesion volume (T1LV)
- Brain parenchymal fraction (BPF)
- T1:T2 lesion volume (assessment of lesion severity)

Distinguishes RR from SP phenotypes

DTI tractography of the descending corticospinal tracks

Three cases of pediatric infiltrating glioma
Summary

I. Pattern recognition

II. Acute vs. chronic abnormalities

III. Longitudinal changes