White Matter T2-Hyperintensities

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White matter hyperintensities (WMHs)

• White matter hyperintensities (WMHs) are bright objects observed in the white matter on brain MR imaging
• The most common lesions on brain MRI
• Can be “normal” findings
• may represent pathological changes: small vessel disease (SVD), lacunar infarction, traumatic brain injury, demyelinating disease and others....
Incidental WMHs in healthy subjects

a 24-year-old female patient with headache
Incidental WMHs in healthy subjects

WMHs reported as demyelinating lesions...

a 20-year-old man with headache
Some WMHs have no specific cause, especially in young patients. Incidental WMHs without a detected cause can be extremely distressing for these patients.

The studies assessing WMHs in “healthy” individuals report marked variability in WMHs prevalence:

- Hopkins et al.: WMHs occurred in 5.3% (13 of 243) of healthy subjects ages 16-65 years
- Katzman et al.: The prevalence of WMHs was 0.5% in 1000 asymptomatic controls ages 3-83 years
- Other studies report higher rates of WMHs: 56% from age 16 to 78 years and 69% in subjects age 50 years or older
a 22-year-old female patient with migraine
Small vessel disease (SVD)

• Cerebral small vessel disease (CSVD) is the most common, chronic and progressive vascular disease.

• The changes affect arterioles, capillaries and small veins supplying the white matter and deep structures of the brain.

• Its occurrence is associated mainly with age and commonly known risk factors for vascular diseases: hypertension and diabetes mellitus.

• Other risk factors: current and former smoking, obstructive sleep apnea, chronic kidney disease, and branch atheromatous disease.
# Neuroimaging features of SVD

<table>
<thead>
<tr>
<th>Type of CSVD</th>
<th>Description</th>
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<tbody>
<tr>
<td>Recent subcortical infarcts</td>
<td>fresh, small (less than 20 mm in axial section) ischemic lesions with respect to perforating arteries, whose radiological features or clinical signs and symptoms indicate their formation in the few weeks before the test; best seen in the DWI sequence; these changes are hypointense in the T1 sequence, hyperintense in the T2 and FLAIR sequences, and isointense in the GRE-T2 sequence</td>
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<tr>
<td>Lacunae of presumed vascular origin</td>
<td>round or oval subcortical lesions 3–15 mm in diameter, filled with fluid, with cerebrospinal fluid-like signal; these lacunae correspond to history of acute cerebral infarction or bleeding from the area of vascularization of the perforating artery; the lesions are characterized by a distinctive image in the FLAIR examination; each lesion is a cavity filled with cerebrospinal fluid and surrounded by a hyperintense rim; they are isointense in the DWI sequence, hypointense in the FLAIR and T1 sequences, and hyperintense in the T2 sequence</td>
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<tr>
<td>White matter hyperintensities</td>
<td>symmetric regardless of size; hypointense in the T2, FLAIR and GRE-T2 (gradient-echo T2) sequences; isointense in DWI, and hypointense in T1</td>
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<td>Widened perivascular spaces (Virchow–Robin perivascular spaces)</td>
<td>mostly seen in basal ganglia &lt;2 mm in size; they usually accompany hyperintense lesions of the white matter and lacunar condition but not brain atrophy; the lesions are hyperintense in T2 sequences, hypointense in FLAIR and T1 sequences, and isointense in the GRE-T2 sequence</td>
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<tr>
<td>Cerebral microbleeds (CMBs)</td>
<td>small, homogeneous lesions &lt;10 mm in diameter, characterized by the ‘blooming effect’; the lesions are best seen in the gradient-echo T2 sequence (hypointense lesions); in the T2, T1 and FLAIR sequences, they are isointense; microbleeds correspond to hemosiderin-loaded macrophages that are present in the perivascular space</td>
</tr>
<tr>
<td>Brain atrophy</td>
<td>brain atrophy in the context of CSVD is considered only when the patient has not suffered a stroke or head injury</td>
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The Fazekas scale is used to simply quantify the amount of white matter T2 hyperintense lesions usually attributed to small vessel disease, although clearly not all such lesions are due to this.

Score:

Fazekas 0: None or a single punctate WMH lesion
Fazekas 1: Multiple punctate lesions
Fazekas 2: Beginning confluency of lesions (bridging)
Fazekas 3: Large confluent lesions

Fazekas 1 is considered normal in the elderly.

Fazekas 2 and 3 are pathologic < 75 years, but may be seen in normally functioning individuals.
Fazekas 3 are pathologic > 75 years
Small vessel disease (SVD)

- Fazekas 3 WMHs
- Acute lacunar infarction
- Enlarged VRSs – État criblé
- Brain atrophy

a 79-year-old female patient with chronic hypertension and cognitive deficits
Small vessel disease (SVD)

a 69-year-old female patient with hypertension and diabetes

These lesions are more often small (ie, less than 3 mm), punctate and non-ovoid, symmetric, located in the subcortical or deep gray matter (corpus callosum usually spared), would not be expected to involve the spinal cord or result in contrast-enhancement

Small vessel disease (SVD) -

a 66-year-old female patient with cognitive deficits
WMHs in migraine

a 22-year-old female patient with migraine

Do not report Fazekas 1 WMHs in the course of SVD!
WMHs in migraine

Do not report WMHs due to multiple sclerosis!

a 22-year-old female patient with migraine
Demyelinating disease

• **Multiple sclerosis (MS)** is a chronic, immune-mediated neurodegenerative disease; the most common CNS inflammatory demyelinating disorder

Other CNS inflammatory disorders:
• **Neuromyelitis Optica Spectrum Disorder (NMOSD)**
• **Myelin Oligodendrocyte Glycoprotein antibody-associated disease (MOG-AD)**
- less common but share some clinical characteristics, such as optic neuritis and myelitis, which can make a specific diagnosis challenging

2017 McDonald criteria for MS

• Dissemination in space can be demonstrated by one or more T2-hyperintense lesions* that are characteristic of MS in two or more of four areas of the CNS:
  - periventricular,
  - cortical or juxtacortical,
  - infratentorial brain regions,
  - the spinal cord

• Dissemination in time can be demonstrated by the simultaneous presence of gadolinium-enhancing and non-enhancing lesions* at any time or by a new T2-hyperintense or gadolinium-enhancing lesion on follow-up MRI, with reference to a baseline scan, irrespective of the timing of the baseline MRI

*Unlike the 2010 McDonald criteria, no distinction between symptomatic and asymptomatic MRI lesions is required.

Periventricular WMHs

Well-circumscribed ovoid lesions, adjacent to the body of the lateral ventricle
Periventricular WMHs

When the periventricular lesions extend perpendicularly into the white matter

Dawson’s fingers
Juxtacortical lesions

Isolated U-fiber lesion, juxtacortical lesion - a lesion extending along subcortical U-fibers
Juxtacortical lesions
Cortical lesions

- Cortical lesions are found in 44% patients with CIS and 70% patients with MS
- Not present in subjects with migraine, other demyelinating diseases (NMO...)

Calabrese M et al. Neurology 2012

T2 image, 3T MR
University Hospital in Wroclaw

Double Inversion Recovery (DIR)
University Hospital in Wroclaw
Cortical lesions

T2 image, 1.5 T MR
infratentorial brain regions
infratentorial brain regions

Lesions in the spinal cord

The cervical spinal cord is most frequently involved. The lesions are typically well-circumscribed in the acute stage and often involve the periphery. The lesions are usually small and short, extending up to two vertebral segments lengths (two or fewer vertebral bodies).

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Temporal regions

Temporal lobes – not commonly affected by SVD

Involvement of the corpus callosum and temporal horns is especially suggestive of MS

Involvement of the corpus callosum and temporal horns is especially suggestive of MS
The callososeptal interface is located on the inferior surface of the corpus callosum, where the septum pellucidum abuts it.
Acute/active lesions which remain ‘enhancing’ for up to 8 weeks with a majority resolving within 4 weeks. Lesions that continue to enhance beyond 8 weeks should raise suspicion for an alternate diagnosis such as sarcoidosis or malignancy.

Acute/active lesions - contrast enhancement

A punctate enhancement pattern
Tumefactive lesions
Tumefactive lesions
Clinical and radiographic spectrum of pathologically confirmed tumefactive multiple sclerosis

C. F. Luchinetti,¹ R. H. Gavrilova,¹ I. Metz,² J. E. Parisi,³ B. W. Scheithauer,³ S. Weigand,⁴ K. Thomsen,⁴ J. Mandrekar,⁴ A. Altin,² W. Brück²

[Images of MRI scans]
WMHs - Differential diagnosis

BE CAREFUL who you trust!!!
Salt and sugar are both white.
WMHs - Differential diagnosis

- Small vessel disease
- Migraine
- Multiple sclerosis
- ADEM (acute disseminated encephalomyelitis)
- NMOSD, MOG-AD
- Susac Syndrome
- CADASIL
- Vasculitis and Inflammatory: Primary CNS vasculitis, SLE, Sjogren’s Syndrome, Neurosarcoïdosis, Neuro- Behcet’s, CLIPPERS, Wegener’s, Crohn’s disease, Celiac disease
- Infectious**
- CPM (central pontine myelinolysis)
- PRES
- Wernicke
- B12 deficiency
- Mitochondrial disorders
- Adult- onset leukodystrophies
- Neoplasm

**PML, HIV, Lyme, Whipple's, neurosyphilis, cysticercosis, toxoplasmosis.

ADEM - acute disseminated encephalomyelitis

- ADEM is a **monophasic demyelinating disorder** usually affecting children and young adults, typically following a recent (1-2 weeks prior) viral infection or vaccination.

- MRI: **bilateral and asymmetric** involvement of the supratentorial and infratentorial **white and gray matter and the spinal cord** (*LETM* is the typical spinal cord manifestation).

- The thalami and basal ganglia are also frequently involved.

- **Tumefactive brain lesions** in white and gray matter, all of them at the same stage of inflammation.

- In acute stages, **enhancement is variable** - in up to 30% of patients.

*LETM* – longitudinally-extensive transverse myelitis.

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a 6-year-old girl, rubella infection 3 weeks ago
a 6-year-old girl, rubella infection 3 weeks ago

ADEM

T1+C – no contrast enhancement
Follow-up MRI after 1 month shows complete or near-complete lesion resolution.
A similar case???
ADEM???

a 26-year-old woman, viral infection 2 weeks ago
ADEM???

a 26-year-old woman, viral infection 2 weeks ago

T1+C, 1.5T MR
University Hospital in Wroclaw
Final diagnosis: MS

Follow-up MRI after 1 month: WMHs still visible
Final diagnosis: MS

Follow-up MRI after 1 month: WMHs still visible
Neuromyelitis optica spectrum disorder - NMOSD

• severe demyelinating disease caused by an autoantibody to the aquaporin-4 water channel

• the classic presentation of NMO is with the triad of optic neuritis, longitudinally extensive myelitis (LETM), and positive anti-AQP4 antibody

• the prevalence of brain lesions is variable: 24%–89% of NMOSD patients, most of these lesions remain unspecific, and clinically silent

• Furthermore, a limited number of brain lesions (approximately 16%) also satisfy the MRI criteria for MS

*LETM – longitudinally-extensive transverse myelitis

Typical brain involvement in NMOSD
Enhancement of the bilateral posterior optic nerves, chiasmatic region involvement. There is also typical enhancement of the periaqueductal region and area postrema.
NMOSD – longitudinally extensive myelitis (LETM)

a 21-year-old woman
Myelin Oligodendrocyte Glycoprotein antibody-associated disease (MOG-AD) recently proposed as a new, distinct disease entity among the demyelinating diseases

- presents with a variety of clinical phenotypes:
  - optic neuritis (most common 41-63%)
  - longitudinally extensive spinal cord lesions (30%)
  - neuromyelitis optica (6-24%)
  - encephalomyelitis (2-6%)
  - seizures - FLAIR-hyperintense lesions in anti-MOG associated encephalitis with seizures (FLAMES)
Opitic neuritis typically bilateral, involving the anterior parts, with prominently swollen edematous nerves resulting in tortuosity and papilledema; Involvement of the optic chiasm and optic tracts is uncommon.
MOG antibody-associated disease (MOG-AD)

LETM (like NMOSD) or short-segment; Central involvement is common
Conus medullaris involvement can be characteristic

*LETM – longitudinally-extensive transverse myelitis

„cloud-like enhancement”
„bright spotty lesion”

MOG antibody-associated disease (MOG-AD)

Brain lesions:
- can appear normal (75%) and/or non-specific;
- can appear similar to ADEM (large cerebral lesions);
- ‘fluffy’, that is, poorly demarcated T2-hyperintensities, in the pons and cerebellar peduncles and adjacent to the fourth ventricle

<table>
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<tr>
<th>A) Optic nerve</th>
<th>B) Spinal cord</th>
<th>C) Brain</th>
</tr>
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<tr>
<td>NMOSD-AQP4-IgG+</td>
<td>MOG-IgG+</td>
<td>Multiple Sclerosis</td>
</tr>
</tbody>
</table>

Susac syndrome

- rare small-vessel disorder (*retinocochleocerebral vasculopathy*) causes small infarcts of cochlear, retinal, and brain tissue

- It consists of a clinical triad of:
  1. acute or subacute *encephalopathy*: memory impairment, confusion, behavioral disturbances, ataxia, dysarthria, paranoid psychosis, and headaches
  2. *sensorineural hearing loss* at low and medium frequencies 1-8
  3. *branch retinal artery occlusions* leading to scotomata and vision distortion

All clinical features may not be present at the same time, and may, in fact, fluctuate over a 1-2 year period, which often leads to a delay in diagnosis.
Susac syndrome – corpus callosum involvement

Lesions typically involve the central fibers of the callosal body and splenium without abuting the callosal undersurface, the roof of the corpus callosum is also frequently involved, rather than the callososeptal interface (which is more typical of MS).

„snowballs”

A 28-year-old patient admitted to the Ophthalmology Department due to sudden visual impairment since 2 days.
Susac syndrome

Lesions can also involve the periventricular white matter, centrum semiovale, cerebellum, brainstem, and middle cerebellar peduncles.

A *string of pearls* appearance – due to punctate microinfarcts involving the internal capsule.
Susac syndrome

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a 28-year-old patient admitted to the Ophthalmology Department due to sudden visual impairment since 2 days
a 28-year-old patient admitted to the Ophthalmology Department due to sudden visual impairment since 2 days
Susac syndrome

A 23-year-old woman admitted to the Neurology Department due to extrapyramidal symptoms with suspicion of MS.

„snowballs”

„string of pearls”
CADASIL- Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy;
- microvasculopathy characterized by recurrent lacunar and subcortical white matter ischemic strokes and vascular dementia in young and middle age patients without known vascular risk factors.

Clinical presentation:
• recurrent transient ischemic attacks (TIAs) or strokes in multiple vascular territories
• migraines, may also have auras
• cognitive disorders, depression, psychosis, pseudobulbar palsy and focal neurological defects as well as seizures

• MRI: widespread confluent WMHs, in the initial course of the disease involvement of the anterior temporal lobe (86%) and external capsule (93%) are typical
a 38-year-old man

CADASIL involvement of the anterior temporal lobe - a very characteristic finding!
CADASIL

a 38-year-old man
Chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids (CLIPPERS) is a brainstem disorder characterized by perivascular histopathologic reaction with lymphocytes and macrophages in the absence of an infectious source. This autoimmune disease is a diagnostic challenge and characterized by very good response to immunosupresion therapy.

Vasculitis

**Primary Angiitis of the CNS**
- an idiopathic inflammatory vascular disease affecting the CNS with no involvement of other systems
- **MRI features are extremely variable and nonspecific:**
  - Faint or coalescent/diffuse supratentorial and infratentorial WMHs involving the superficial and deep white associated with variable hemorrhagic foci, infarcts, and acute convexity subarachnoid hemorrhage can occur in several combinations.
  - Leptomeningeal enhancement - 10-15 %
  - Lesions showing contrast enhancement – 30%

**Secondary vasculitis of the CNS**
- occurs in the context of a systemic inflammatory or infectious process
  - SLE, Sjogren’s Syndrome, Neurosarcoidosis, Neuro- Behcet's, CLIPPERS, Wegener’s, Crohn’s disease, Celiac disease
  - CNS vasculitis due to infections (Lyme, tuberculosis, neurosyphilis)
a 58-year-old woman with antiphospholipid syndrome
Typical WMHs in lupus

a 38-year-old woman with lupus
Neuroboreliosis

a 53-year-old man
Differential diagnosis
Approximately half of MS patients have at least one isolated U-fiber lesion.

Isolated U-fiber lesion, juxtacortical lesion - a lesion extending along subcortical U-fibers

WMHs in SVD do not involve the subcortical U-fibers

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lesions extending along subcortical U-fibers

CADASIL
63-year-old patient with Parkinson disease, hypertension, myocardial infarction 7 years earlier, treated with percutaneous transluminal coronary angioplasty (PTCA). In the previous MRI demyelinating lesions were reported.
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Corpus callosum involvement
Corpus callosum involvement

MS

SUSAC
100%

CADASIL
40%

Rovira A. Ateny 2014
Callososeptal interface involvement

SVD
- Small vessel disease

MS
Callososeptal interface involvement

The callososeptal interface is located on the inferior surface of the corpus callosum, where the septum pellucidum abuts it.
Any other sequence helpful in DD?
a 63-year-old patient with Parkinson disease, hypertension, myocardial infarction 7 years earlier, treated with percutaneous transluminal coronary angioplasty (PTCA). In the previous MRI demyelinating lesions were reported.
Hypertensive encephalopathy - CMBs in basal ganglia, thalamus, brainstem and cerebellum as well as subcortical white matter

a 63-year-old patient with Parkinson disease, hypertension, myocardial infarction 7 years earlier, treated with percutaneous transluminal coronary angioplasty (PTCA). In the previous MRI demyelinating lesions were reported.
Cerebral Amyloid Angiopathy (CAA)

NEUROIMAGING (MRI) CORRELATES OF CEREBRALAMYLOID ANGIOPATHY
The important MRI correlates of CAA (figures 4 and 5) include:
- Cerebral microbleeds
- White matter changes (leukoaraiosis)
- Convexity subarachnoid haemorrhage
- Cortical superficial siderosis
- Silent acute ischaemic lesions

Charidimou A et al. J Neurol Neurosurg Psychiatry 2012
CMBs typically in superficial lobar locations: most frequently in the occipital and temporal cortex followed by the parietal and frontal location. There are also hemosiderin deposits within old hematoma in the right temporal lobe. The acute hematoma – in the left temporal region.
CADASIL- Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy

T2 images

SWI
What about periventricular WMHs?
A 23-year-old man, with the diagnosis of MS (made when he was 10, after viral infection), currently treated with Copaxone. He presented mainly spinal symptoms with spastic paresis of the lower limbs and urinary incontinence as well as epileptic seizures.
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What is the most likely diagnosis?
Hypoxic-Ischemic Brain Injury: Imaging Findings from Birth to Adulthood

Benjamin Y. Huang, MD, MPH • Mauricio Castillo, MD

Global hypoxic-ischemic injury (HII) to the brain is a significant cause of mortality and severe neurologic disability. Imaging plays an important role in the diagnosis and treatment of HII, helping guide case management in the acute setting and providing valuable information about long-term prognosis. Appropriate radiologic diagnosis of HII requires familiarity with the many imaging manifestations of this injury. Factors such as brain maturity, duration and severity of insult, and type and timing of imaging studies all influence findings in HII.

Severe hypoxia-ischemia in both preterm and term neonates preferentially damages the deep gray matter, with periventricular involvement more frequently observed in the latter age group. Less profound insults result in intraventricular hemorrhages and periventricular white matter injury in preterm neonates and parasagittal watershed territory infarcts in term neonates. In the postnatal period, severe insults result in diffuse gray matter injury, with relative sparing of the periolandic cortex and the structures supplied by the posterior circulation. Profound hypoxia-ischemia in older children and adults affects the deep gray matter nuclei, cortices, hippocampi, and cerebellum. Because findings at conventional imaging may be subtle or even absent in the acute setting, particularly in neonates, magnetic resonance spectroscopy can help establish the diagnosis of HII. Promising new neuroprotective strategies designed to limit the extent of brain injury caused by hypoxia-ischemia are currently under investigation.
1. Selective brain damage

A. Acute Profound Asphyxia
   - Basal ganglia
   - Thalamus
   - Perirolandic cortex...

B. Partial Prolonged Asphyxia
   - Hypotension
   - Infection
   - Hypoglicemia
   - Watershed territories
„watershed” infarcts
End-stage PVL
(End-stage white matter injury)

Figure 11. End-stage PVL in a 9-year-old child who presented with motor and cognitive delay and seizures. The patient was born at 32 weeks gestational age. Axial fluid-attenuated inversion recovery MR images demonstrate increased signal intensity and a few tiny cysts in the immediate periventricular white matter. In b, there is enlargement of the atria of the lateral ventricles with a decrease in volume of the adjacent white matter, and the walls of the lateral ventricles have a wavy appearance.

Huang B et al. Radiographics 2008
A 27-year-old female patient presented to the Neurology Department, she had chronic headache since 2 years. In the previous MRI demyelinating lesions were reported.
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Dilated Virchow-Robin spaces
Dilated Virchow-Robin spaces

• Virchow-Robin spaces (VRSs) are perivascular areas, which play a role as drainage pathways for interstitial fluid.

• Dilated VRSs are commonly seen in normal subjects as an incidental finding and usually are asymptomatic.
Dilated Virchow-Robin spaces

- Virchow-Robin spaces (VRSs) are perivascular areas, which play a role as drainage pathways for interstitial fluid.
- Dilated VRSs are commonly seen in normal subjects as an incidental finding and usually are asymptomatic.
Giant (tumefactive) dilated Virchow-Robin spaces

- When VRSs are giant (tumefactive), they can cause mass effect and pressure symptoms, which along with bizarre configurations can lead to misdiagnosis of cystic neoplasm.
- Most commonly the giant VRSs involve the mesencephalic region as well as cerebral white matter.
- MRI: the cysts have thin walls and its internal contents show signal intensity similar to CSF, with no contrast enhancement.
- Usually there are no signal intensity alterations in the adjacent brain parenchyma. However, hyperintense foci on T2-weighted and FLAIR images may be visible in surrounding areas.

Giant mesencephalic dilated Virchow-Robin spaces

a multicystic lesion in the midbrain with extension into the third ventricle causing obstruction of the cerebral aqueduct and thus leading to obstructive hydrocephalus

a 63-year-old female patient with acute obstructive hydrocephalus, the previous MRI examination was misinterpreted as a cystic tumour

Giant (tumefactive) dilated Virchow-Robin spaces in cerebellum

T1+C
Large anterior temporal Virchow-Robin spaces: unique MR imaging features

Anthony T. Lim • Ronil V. Chandra • Nicholas M. Trost • Penelope A. McKelvie • Stephen L. Stucky

Large anterior temporal lobe VR spaces commonly demonstrate:
- perilesional T2 or FLAIR signal
- focal cortical distortion by an adjacent branch of the MCA
- smaller adjacent VR spaces
- the majority remains stable during imaging surveillance, an uncommon change in perilesional signal or size can occur

- can be misdiagnosed as a cystic tumor!
A 66-year-old female patient, after viral infection few days before, admitted due to severe headache and dizziness, on brain CT there was a suspicion of neoplastic lesion in the left temporal lobe.
• WMHs can be “normal” findings in healthy subjects
• WMHs more common in patients with migraine
• may represent pathological changes: small vessel disease (SVD), lacunar infarction, traumatic brain injury, demyelinating disease and others....
Approximately half of MS patients have at least one isolated U-fiber lesion.

Isolated U-fiber lesion, juxtacortical lesion
- a lesion extending along subcortical U-fibers

WMHs in SVD

do not involve the subcortical U-fibers

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Callososeptal interface involvement

Small vessel disease  
Susac syndrome  
MS - Callososeptal interface involvement
Susac syndrome

23-year-old woman admitted to the Neurology Department due to extrapyramidal symptoms with suspicion of MS

„snowballs”

„string of pearls”
involvement of the anterior temporal lobe and external capsule are typical!
Periventricular WMHs due to hypoxic-ischemic brain injury can be misdiagnosed as MS lesions!

Parasagittal watershed territory infarcts
Giant (tumefactive) dilated Virchow-Robin spaces can be misdiagnosed as a cystic tumor!
Thank you for your attention!