A Pattern Approach to Focal White Matter Hyperintensities on Magnetic Resonance Imaging

Sangam Kanekar, MD[a,b,*], Puneet Devgun, DO[a]

KEYWORDS

• Focal white matter hyperintensity • Multiple sclerosis • Acute disseminated encephalomyelitis • CNS vasculitis • Lyme • Sarcoid

KEY POINTS

- Evaluation of focal white matter hyperintensities on magnetic resonance (MR) imaging in any age group is always challenging.
- It is important to have a specific imaging approach, including age, pattern of distribution, signal characteristics on various sequences, enhancement pattern, and other ancillary findings, to infer to a correct cause for white matter hyperintensities.
- Normal MR imaging almost always excludes intracranial vasculitis. However, there are no pathognomonic MR imaging findings in vasculitis.
- Asymptomatic (silent) lacunar infarcts are at least 5 times more common than symptomatic infarcts.
- The risk of dementia and severity of cognitive impairment is preferentially associated with periventricular white matter lesions, whereas mood disorders are more likely seen with deep white matter lesions.

INTRODUCTION

Evaluation of focal white matter hyperintensities (WMH) on magnetic resonance (MR) imaging in any age group is always challenging because the cause of these hyperintensities may vary from infectious, inflammatory, neoplastic, or demyelinating findings to nonspecific findings related to aging and other systemic conditions (**Box 1**, **Table 1**). Most clinicians look to the imager for a specific diagnosis or to limit the differential diagnosis so that an appropriate test may be advised to confirm the cause or underlying disease process. Without an appropriate clinical history and findings, these nonspecific WMH can be challenging to differentiate. An understanding of the clinical presentation, pathophysiology, and associated imaging findings can allow the radiologist to limit the differential. It is important to have a specific imaging approach, including age, pattern of distribution, signal characteristics on various sequences, enhancement pattern, and other ancillary findings, to infer a correct cause for these hyperintensities. Many times in clinical practice it may not be able to characterize these hyperintensities, and in such cases discussion with the clinicians with appropriate follow-up may be the best

[a] Department of Radiology, Penn State Milton S. Hershey Medical Center and College of Medicine, The Pennsylvania State University, 500 University Drive, Hershey, PA 17033, USA; [b] Department of Neurology, Penn State Milton S. Hershey Medical Center and College of Medicine, The Pennsylvania State University, 500 University Drive, Hershey, PA 17033, USA

* Corresponding author. Department of Radiology, College of Medicine, Penn State Milton S. Hershey Medical Center and College of Medicine, The Pennsylvania State University, 500 University Drive, Hershey, PA 17033.
E-mail address: skanekar@hmc.psu.edu

Radiol Clin N Am 52 (2014) 241–261
http://dx.doi.org/10.1016/j.rcl.2013.11.010
0033-8389/14/$ – see front matter

Box 1
Differential diagnosis of focal white matter hyperintensities on T2-weighted imaging

1. Virchow-Robin spaces
2. Migrainous ischemia
3. Multiple sclerosis
4. Acute disseminated encephalomyelitis
5. Central nervous system (CNS) vasculitis
   - **Primary vasculitis**
   - a. Giant-cell arteritis
   - b. Primary angiitis of the CNS
   - c. Takayasu disease
   - d. Polyarteritis nodosa
   - e. Kawasaki disease
   - f. Churg-Strauss syndrome
   - g. Wegener granulomatosis
   - **Secondary vasculitis**
   - h. Collagen vascular diseases
   - i. Systemic lupus erythematosus
   - j. Scleroderma
   - k. Rheumatoid arthritis
   - l. Sjögren syndrome
   - m. Mixed connective tissue disease
   - n. Behçet disease
   - o. Infection
   - p. Illicit drugs
   - q. Malignancy
   - r. Other systemic conditions
6. Cerebrovascular disease
   - a. Lacunar infarcts
   - b. Watershed infarctions
7. CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy)
8. Sarcoidosis
9. Lyme disease
10. Progressive multifocal leukoencephalopathy
11. Age-related changes
12. Effects of radiation therapy or drugs
13. Metastatic disease
14. Inherited white matter diseases
15. CNS lymphoma

solution. The purpose of this article is to provide a pattern approach to differentiate various common and a few uncommon diseases presenting as focal WMH.

PERIVASCULAR SPACES OR VIRCHOW-ROBIN SPACES

Perivascular spaces (PVS) or Virchow-Robin spaces (VRS) are pial-lined, fluid-filled structures surrounding penetrating arteries and arterioles. These spaces are seen most commonly along the path of lenticulostriate arteries entering the basal ganglia, or along the perforating medullary arteries entering the cortical gray matter. Other areas where prominent PVS can be seen include the subinsular region, dentate nuclei, and cerebellum. The exact etiology of these PVS has yet to be delineated. Multiple hypotheses have been suggested, including spiral elongation of the penetrating blood vessels, increased cerebrospinal fluid (CSF) pulsations, sequelae of ex vacuo phenomenon, abnormality of arterial wall permeability, and accumulation of brain interstitial fluid between the vessel and pia or interpial space.[1]

PVS become prominent and dilated with the age of the patient. Prominence of PVS in older patients is thought to be due to 2 main reasons. First, VRS are a direct extension of the subarachnoid space, and aging is associated with enlargement of ventricles and sulci, resulting in prominence of the subarachnoid space. Second, atherosclerotic changes, particularly in hypertensive patients, result in unfolding and tortuosity of the vessels, leading to prominence of PVS.

On MR imaging PVS appear as round to oval, smoothly demarcated fluid-filled cysts, typically less than 5 mm in diameter, and often occur in clusters.[1] PVS are isointense to CSF on all pulse sequences including fluid-attenuated inversion recovery (FLAIR), and demonstrate no enhancement after contrast administration (**Fig. 1**). PVS do not cause focal mass effect or restriction on diffusion-weighted (DW) images. On axial images they are typically seen around the lateral portion of the anterior commissure. Although most show normal signal intensity in the adjacent brain; 25% may have a small rim of slightly increased signal intensity. VRS in the midbrain surrounding the branches of the collicular and accessory collicular arteries are slightly hyperintense to CSF on FLAIR images.

One of the common clinical challenges is to differentiate PVS from lacunar infarction. Location, morphology, and signal intensity tend to differentiate these 2 conditions. Dilated PVS usually are isointense to CSF on all pulse sequences,