Benign MRI findings and their pathologic mimics

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Summary

Otherwise benign MRI findings (so-called “incidentalomas”) are a frequent cause of worry on the part of both clinician and patient. Knowledge of the imaging characteristics of incidentalomas, their (typically slight) pathologic significance, and how to distinguish these from less benign pathologic mimics can help avoid unnecessary worry, expense, and workup.

Neuroimaging is replete with findings that initially raise concerns, but are ultimately inconsequential. Most of the time these incidentalomas are not related to the symptoms, but this may be hard to prove to the caring physician and patient. We describe some of the more common incidental findings with little pathologic significance and provide hints on how to distinguish these from pathologic mimics. Limited complications of some of the more common incidentalomas (e.g., dilated Virchow-Robin spaces, arachnoid cysts, developmental venous anomalies) are discussed briefly.

Virchow-Robin spaces

Virchow-Robin spaces (VRS) are perivascular CSF-filled gaps that serve as an extralymphatic drainage system of the brain. Unusually large or prominent VRS can lead to misdiagnoses. Most commonly, large VRS are located in regions of the brain with penetrating arteries, e.g., the lenticulostriate and cortical medullary arteries. On MRI, VRS are smoothly contoured, linear, or ovoid spaces with isointense signal to CSF on all sequences scattered throughout the centrum semiovale, basal ganglia, and, more rarely, the brainstem (figure 1, A–D).¹ Benign VRS are visualizable in almost everyone over 65 years old with one-third having VRS over 3 mm in diameter. The larger VRS tend to be in the anterior perforant substance region or posterior centrum semiovale where the branches from the cerebral arteries penetrate into the brain.

Lacunae may be difficult to distinguish from VRS. Lacunes tend to be wedge-shaped or slit-like and larger than VRS.² VRS are isointense to CSF on inversion recovery sequences (e.g., fluid-attenuated inversion recovery), whereas lacunes tend to have some high signal indicative of gliosis. Multiple dilated VRS in the basal ganglia (l'état criblé or cribiform state) is a rare finding associated with chronic hypertension (figure 1, E and F).

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Figure 1 Dilated Virchow-Robin spaces on MRI

Coronal T1 (A), T2 (B), fluid-attenuated inversion recovery (FLAIR) (C), and axial T2 (D) sequences with normal multiple Virchow-Robin spaces (VRS) in the posterior periventricular region surrounding the cortical medullary arteries bilaterally. Note that these perivascular spaces follow CSF signal intensities. The large perivascular space (arrows in A and C) measured 3.1 mm in diameter. Also present on these images is elevated T2 and FLAIR signal and reduced T1 signal in the posterior periventricular white matter (double arrowhead in A and C), most commonly another benign finding, sometimes correlated with prematurity in young adults. (E, F) T2 (E) and FLAIR (F) demonstrate the abnormal cribriform state with many small VRS throughout the basal ganglia. On the FLAIR image, note the difference between VRS (CSF-low signal on FLAIR: arrows) and several lacunes (double arrowheads) with high signal on the FLAIR image. On the T2 image, VRS and lacunes can have a similar appearance.

Developmental venous anomalies

Developmental venous anomalies (DVAs, a.k.a. venous angiomas) are dilated deep tissue veins that converge around a dilated subependymal or cortical draining vein and are present on 0.5%–2.6% of MRIs.³ Most are due to local retention of the embryonic

Figure 2 Developmental venous anomalies

(A, B) T1 axial with and without contrast reveals a left frontal white matter developmental venous anomaly (DVA) (arrows). (C, D) Sequential coronal T1 images with contrast demonstrate right cerebellar DVA with large draining vein (arrows). (E, F) Coronal T1 with contrast reveals a left inferior insular region DVA in cross-section (white round lesion: dashed arrow) with associated cavernous malformation (double arrowhead) best seen on T2 image as round lesion with heterogeneous hyperintense core with surrounding hypointense (hemosiderin) rim.

venous drainage system. MRI reveals a round (cross-section) or longitudinal vascular structure with a signal void with a surrounding gadolinium-enhancing “caput medusa”—a stellate pattern of medullary venules around the draining vein (figure 2, A–D). The surrounding brain is typically normal, but rarely can have atrophy or white matter abnormalities.

DVAs by themselves are almost always benign, but up to one-fifth are associated with a cavernous malformation⁴ (figure 2, E–F). If the cavernous malformation is resected, it is usually important to preserve the DVA, as it is often essential for venous drainage and removal can cause catastrophic venous infarctions. Venous thrombosis of the collector vein resulting in either hemorrhagic or nonhemorrhagic infarcts has been reported, but is rare.⁵ In contrast to the well-established seizure potential of arteriovenous

Figure 3 Arachnoid cysts

(A, B) T1 sagittal and T2 axial view of arachnoid cyst in pineal region with slight mass effect on quadrigeminal plate (oblique arrows). Note the mild asymmetry of the anterior horns of the lateral ventricles (vertical dashed arrows in B). (C, D) T2 MRI and CT demonstrate incidental left middle cranial fossa arachnoid cyst with mild mass effect (oblique arrows). (E, F) T2 axial and T1 coronal MRI with bilateral middle cranial fossa arachnoid cysts in a 1-year-old patient with glutaric aciduria type 1 (horizontal double arrowheads).

malformations and cavernous angiomas, nonhemorrhagic DVAs are unlikely to contribute to epilepsy.⁶

When a DVA is identified (presumably as an incidental MRI finding), a rational approach is to check for an associated cavernous malformation with susceptibility-weighted imaging or T2* (T2–gradient-recalled echo) on the highest field strength MRI available. If no cavernous malformation is present, no follow-up imaging is typically required for the DVA.

Arachnoid cysts

Arachnoid cysts account for 1% of all intracranial masses.⁷ These cystic lesions, located in the margins of CSF spaces, are predominantly congenital (although occasionally arising following trauma, hemorrhage, or inflammation) (figure 3, A–D). Ninety percent are located

Differentiating normal transitional zones from pathologic T2 peritrigonal hyperintensities such as those caused by a leukodystrophy, demyelinating disease, or leukomalacia from ischemic injury can be problematic.

supratentorially, with over half in the middle cranial fossa. Imaging reveals a well-delineated CSF-isointense pocket, typically with mass effect on the underlying brain, which can make differentiation from pathologic CSF-filled lesions (e.g., colloid cysts, cystic tumors) challenging.

Most arachnoid cysts are asymptomatic and incidental. On occasion, the mass effect is associated with headache, seizures, cognitive impairment, and focal neurologic deficits (e.g., paralyzed upward gaze due to pineal region location). Rarely, arachnoid cysts develop adjacent subdural hemorrhage or intracystic hemorrhage either following minor trauma or spontaneously as a result of the tenuous intracystic bridging veins, which lack parenchymal support.

Although quite rare, bilateral anterior middle cranial fossa arachnoid cysts are nearly always pathognomonic for glutaric aciduria type 1 (figure 3, E and F), a severe genetic disorder which typically presents with macrocephaly, hypotonia, dystonia, dyskinesias, or seizures in infants and young children.

Prophylactic intervention of an asymptomatic arachnoid cyst is not indicated. Treatment of symptomatic cysts can be with shunting, aspiration, endoscopic fenestration, or marsupialization. Intervention for nonacute symptoms must be carefully weighed against the common complications of cyst recurrence (25%), or more rarely hemorrhage, seizure, or subdural hygroma.⁸

Peritrigonal T2 hyperintensities

Zones of posterior white matter T2 hyperintensity are often an incidental finding in patients in their 30s and younger. These are due to the imaging characteristics of transitional zones—the last myelinated regions of the brain (figure 1, A–D, double arrowheads). Normal transitional zones tend to have only slightly higher intensity than the surrounding white matter; hazy, indistinct borders; and no local atrophy.⁹

Asymmetric ventricles

Ventricular asymmetry is common, occurring on up to 40% of MRIs (figure 3B).¹⁰ Although striking asymmetry can raise the question of an intraventricular cyst or unilateral hydrocephalus, in the absence of intraventricular–periventricular structural abnormalities the risk of pathology is slight.

Unidentified bright objects

Punctate high-signal T2 lesions in the subcortical/periventricular white matter (unidentified bright objects [UBOs]) are frequently present on MRIs and increase in number with age, hypertension, and vascular risk factors. UBOs are caused by a mix of etiologies with typically slight pathologic significance. Most UBOs are along the continuum of vascular disease, and separating normal from abnormal is a matter of degree. A useful maxim is, “one UBO per decade after 40 is normal.” A history of cancer, immunodeficiency, or suspicion of demyelinating disease may warrant repetition of the MRI with contrast to evaluate for malignant etiologies.

CONCLUSION

With MRI now entrenched in clinical practice, it is incumbent upon the practitioner to understand when an “incidentaloma” is “normal” and spare the patient the anxiety and potential morbidity of further evaluation.

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