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Unusual Widening of Virchow-Robin Spaces: MR Appearance

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Summary: MR in two patients with unusual widening of the Virchow-Robin spaces showed multiple cystic foci up to 2 cm in diameter along the perforating medullary arteries in the cerebral white matter, mainly in one cerebral hemisphere. These areas were of the same signal intensity as cerebrospinal fluid on all pulse sequences. In one patient, the cystic foci in the white matter were biopsied and histologically confirmed to be large Virchow-Robin spaces.

Index terms: Virchow-Robin spaces; Brain, magnetic resonance

It is generally accepted that free communication exists between the perivascular and sub-arachnoid spaces (1). However, the results of electron microscopy and tracer studies have shown that the subarachnoid space does not communicate directly with the perivascular spaces of the cerebral cortex (2). Although the perivascular space has been the exclusive domain of anatomists and pathologists, it has become an important area of study to the radiologist in the magnetic resonance (MR) era (3). Occasionally, it is difficult to differentiate the perivascular spaces from lacunar infarcts in the basal ganglia (3). When widening of the perivascular spaces is observed in the white matter, the differentiation is especially difficult. In this article, we present two patients with unusual widening of the perivascular spaces studied with MR imaging.

Case Reports

Patient 1

A 50-year-old hypertensive man had a ringing in his left ear. Neurologic examination was normal except for left sensory neural hearing disturbance.

A computed tomography (CT) scan showed an enhancing tumor in the left cerebellopontine angle cistern

and multiple cystic areas in the cerebral white matter (Fig 1A). MR imaging, performed with 0.5-T unit, also showed multiple cystic foci located in the cerebral white matter (Fig 1B–E) especially on the left. Most did not reveal mass effect. A few large foci showed subtle mass effect. They were located along the perforating medullary arteries in the white matter (Fig 2A). Their signal intensity was similar to that of cerebrospinal fluid (CSF) on T1- (gradient-echo, 560/10/2 [repetition time/echo-time/excitations], 90° flip angle), proton density- (spin-echo, 3000/35/1) and T2- (spin-echo 3000/90/1) weighted images. A few small foci of CSF signal on all pulse sequences on both sides of the anterior commissure also were demonstrated.

The laboratory findings were normal. Serologic test results for parasites were negative. A diagnosis of left acoustic neurinoma was made. The multiple cystic foci were diagnosed as large perivascular spaces. However, because a few large cysts showed subtle mass effect, a biopsy at the time of craniotomy also was performed. The tumor in the left cerebellopontine angle cistern was histologically confirmed as an acoustic neurinoma. The cystic areas were histologically confirmed large perivascular spaces (Fig 2B). Follow-up MR images 3 years after discharge did not show any change in these cystic foci (Fig 3).

Patient 2

A 67-year-old normotensive woman became dizzy when she sat up and vomited several times. Thereafter, she had several similar episodes. Neurologic examination did not reveal any abnormalities. Her condition was diagnosed as positional vertigo.

A CT scan showed multiple cystic areas in the cerebral white matter. The lesions were bilateral but predominantly in the right cerebral hemisphere. They did not show any mass effect. MR imaging, performed with 0.5-T unit, showed multiple well-demarcated cystic foci chiefly in the right cerebral white matter (Fig 4A–D). A few also were detected in the right putamen. Their signal intensity was similar to that of CSF in T1- (gradient-echo 300/14/2, flip

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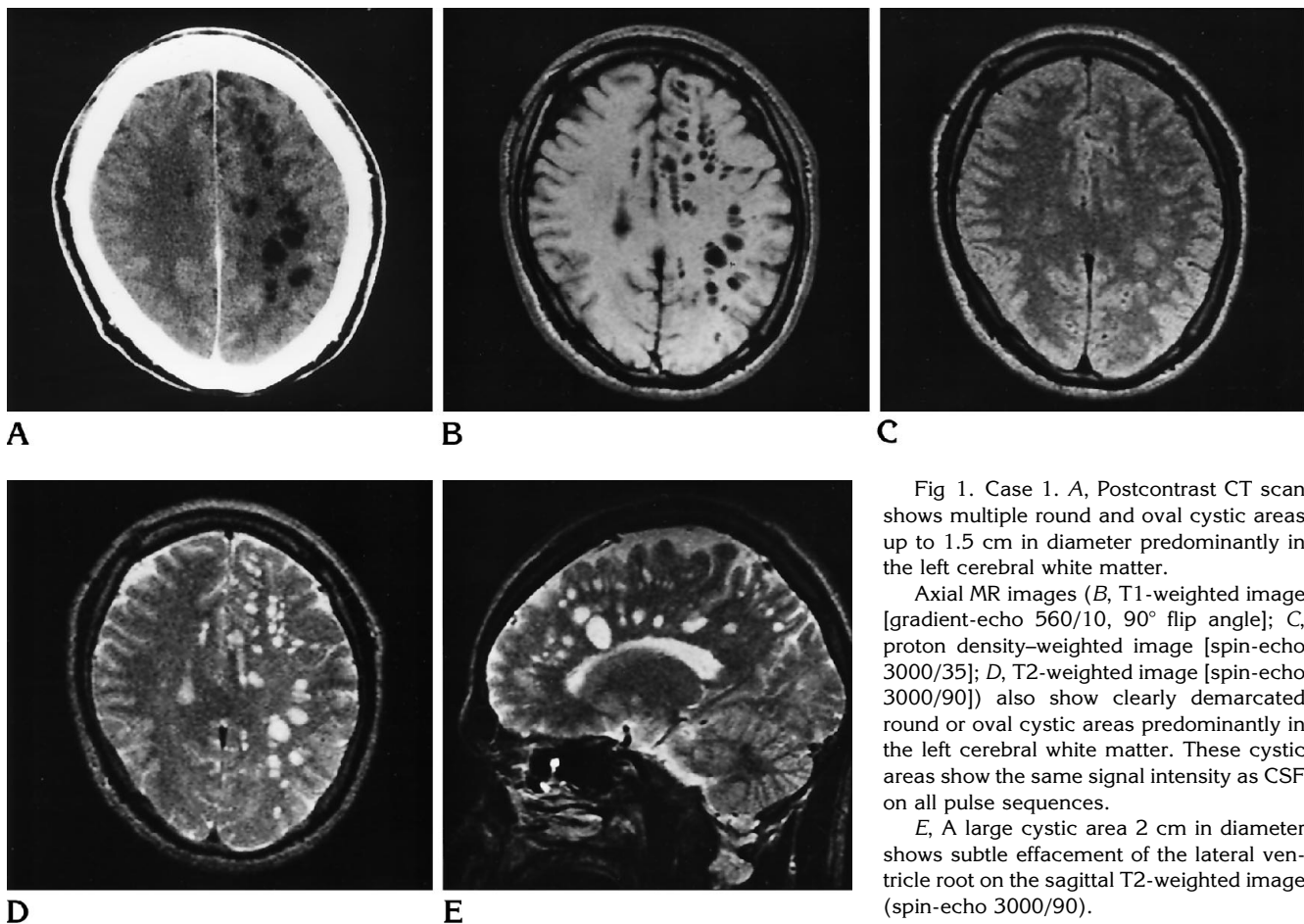


Fig 1. Case 1. A, Postcontrast CT scan shows multiple round and oval cystic areas up to 1.5 cm in diameter predominantly in the left cerebral white matter.

Axial MR images (B, T1-weighted image [gradient-echo 560/10, 90° flip angle]; C, proton density-weighted image [spin-echo 3000/35]; D, T2-weighted image [spin-echo 3000/90]) also show clearly demarcated round or oval cystic areas predominantly in the left cerebral white matter. These cystic areas show the same signal intensity as CSF on all pulse sequences.

E, A large cystic area 2 cm in diameter shows subtle effacement of the lateral ventricle root on the sagittal T2-weighted image (spin-echo 3000/90).

angle 90°), proton density- (spin-echo 2000/30/1), and T2- (spin-echo 2000/120/1) weighted images. They were located along the perforating medullary arteries in the white matter (Fig 2A).

The patient was admitted for therapy of the positional vertigo and further investigation of the multiple cystic areas. The laboratory findings were normal. The serologic test results for parasites were negative. During hospitalization, the vertigo improved with medication. The cystic foci were diagnosed as large perivascular spaces. Follow-up MR images 2 years after discharge did not show any interval change of the multiple cystic areas (Fig 4E).

Discussion

Punctate and patchy foci of abnormal hyperintensity in the cerebral white matter on T2-weighted images occur in more than 30% of the neurologically healthy elderly population (4–6). These signal hyperintensities initially were called “unidentified bright objects” (7). Histopathological study and MR correlation has clarified that these are perivascular spaces and are often demonstrated on high-field MR scans

(8–17). It is important to differentiate these perivascular spaces from pathologic lesions.

Jungreis et al found hyperintensities attributable to the perivascular spaces in 59 of 100 patients on T2-weighted images (14). Heier et al (3) found large perivascular spaces in MR scans of 314 (38%) of 816 patients. Perivascular spaces larger than 2 mm in diameter were found in 67 (8%) of 816 patients. They divided them into two types according to their location. The first lie along the path of the lenticulostriate arteries as they enter the basal ganglia through the anterior perforated substance. The second lie along the path of the perforating medullary arteries as they enter the cortical gray matter over the high convexities and extend into white matter. MR frequently shows the former and rarely the latter. Whereas Jungreis et al reported that the lenticulostriate perivascular spaces did not correlate with advancing age (14), Heier et al concluded that the larger perivascular spaces were another phenomenon of the aging brain

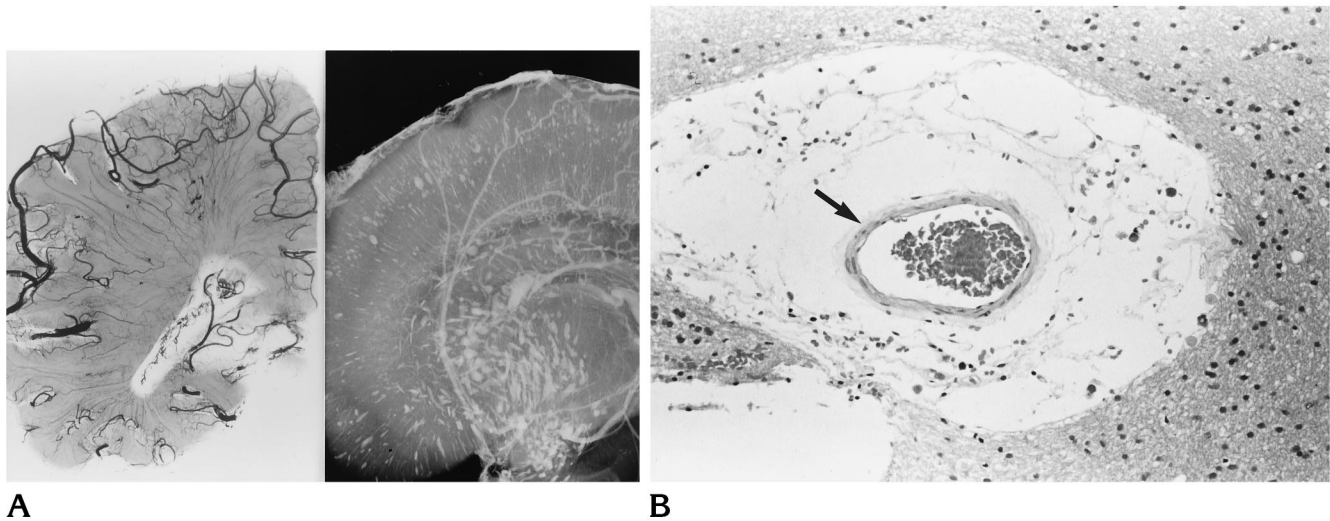


Fig 2. Case 1. A, Coronal microangiogram (*left*) through the trigone of lateral ventricle obtained from a cadaver's brain and sagittal microangiogram (*right*) through the near midline obtained from the other cadaver's brain. Small amounts of contrast medium leak out from the medullary arteries into the perivascular spaces. Multiple cystic areas observed on MR imaging (Figs 1E and 4D) conform to the path of the penetrating medullary arteries on these microangiograms.

B, Histologic section obtained from right medial frontal lobe shows widening of a Virchow-Robin space (hemotoxylin and eosin, $\times 100$). An arrow indicates the arteriole in the center of the dilated Virchow-Robin space.

(3). High convexity and larger perivascular spaces had the strongest correlations with age.

On MR imaging, perivascular spaces are defined as follows: (a) are round, oval, or curvilinear with a well-defined, smooth margin; (b) are isointense relative to CSF in the subarachnoid space on all pulse sequences; (c) conform to the path of penetrating arteries; and (d) have no

mass effect. The lenticulostriate spaces are found in the lower third of the basal ganglia (14). The MR findings in our patients most closely resembled the above findings. However, a few large perivascular spaces in patient 1 showed subtle mass effect.

Poirier et al proposed the neuropathologic classification of cerebral lacunae caused by various pathologic processes and divided cerebral lacunae into three categories (18, 19). Type I lacunae are old, small cerebral infarcts. Type II lacunae are old, small hemorrhages. Type III lacunae are caused by dilatation of the perivascular spaces. They also divided Type III lacunae into four varieties on the basis of their number and volume. Type IIIa lacunae are numerous and very small cavities. Type IIIb lacunae have the same size, location, and number as Type I lacunae. Type IIIc lacunae correspond to single large perivascular dilatations. Type IIId lacunae present as space-occupying lesions (18, 19). According to their classifications, the expanding, large perivascular spaces in patient 1 correspond to Type IIId lacunae, although we think that the term *lacunae* should be limited to the areas of tissue loss or tissue damage caused by infarction. Moreover, there are also some reports in which large perivascular spaces showed mass effect (20–23).

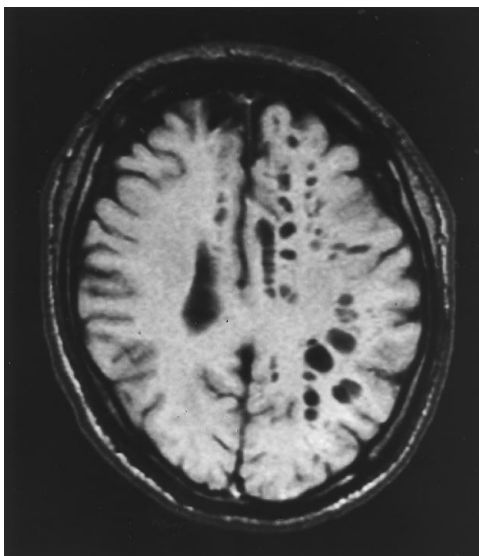


Fig 3. Case 1. Follow-up axial T1-weighted image (gradient-echo 560/10) 3 years after discharge does not show any interval change in the cystic foci.

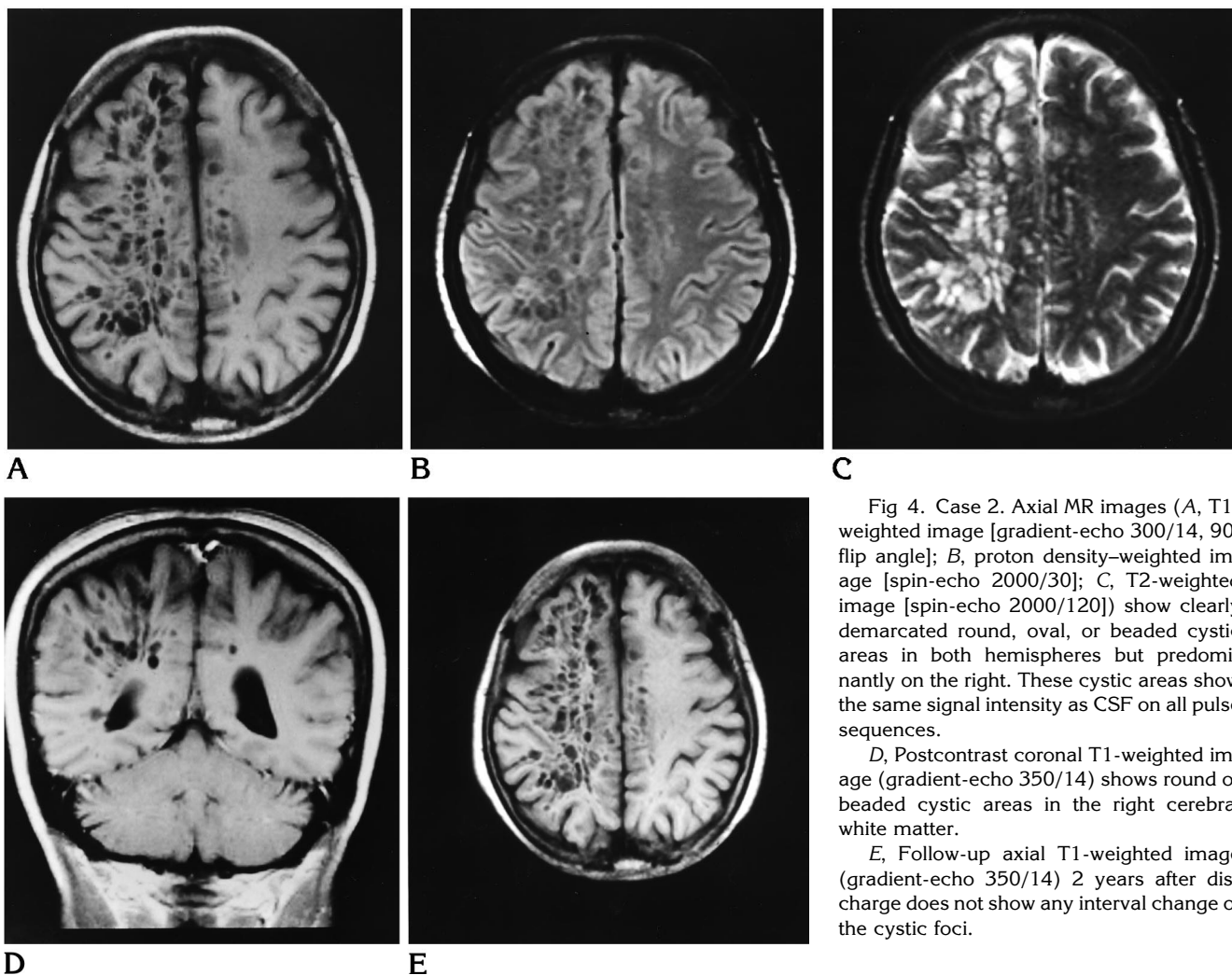


Fig 4. Case 2. Axial MR images (A, T1-weighted image [gradient-echo 300/14, 90° flip angle]; B, proton density-weighted image [spin-echo 2000/30]; C, T2-weighted image [spin-echo 2000/120]) show clearly demarcated round, oval, or beaded cystic areas in both hemispheres but predominantly on the right. These cystic areas show the same signal intensity as CSF on all pulse sequences.

D, Postcontrast coronal T1-weighted image (gradient-echo 350/14) shows round or beaded cystic areas in the right cerebral white matter.

E, Follow-up axial T1-weighted image (gradient-echo 350/14) 2 years after discharge does not show any interval change of the cystic foci.

The pathogenesis of large perivascular spaces still remains to be elucidated. Several mechanisms such as increased CSF pulsations, an ex vacuo phenomenon, vascular ectasia, or an abnormality of arterial wall permeability may be related to their formation (16, 21, 24). If large perivascular spaces are the result of CSF pulsation or vascular ectasia, one might expect them to be seen more frequently in the elderly. Heier et al pointed out a strong correlation between the large perivascular spaces and age (3). Moreover, there are some reports supporting the hypothesis that an abnormality of arterial wall permeability causes the dilatation of the perivascular space (20, 21, 23). Multiple factors mentioned above may be etiologically related to the large perivascular spaces.

The differential diagnosis of perivascular spaces is that of CSF-intensity lesions. A variety

of cysts (ependymal cyst, neuroepithelial cyst, and arachnoid cyst), ventricular diverticula, cystic infarction, and mucopolysaccharidosis should be included. Most cysts usually border the ventricle or subarachnoid space (9). The reports of pure intraparenchymal cysts of non-neoplastic origin are rare (25, 26). The content of such cysts may be not isointense with CSF on all pulse sequences, and their location will not conform to the course of medullary arteries. In our cases, the multiple cystic lesions are not related to the ventricles and/or subarachnoid spaces. Therefore, ventricular diverticula and brain cysts can be excluded. Chronic infarct with cystic change is known to be isointense relative to CSF (8). However, if all our cystic lesions are cystic infarcts, clinical symptoms and neurologic findings should be present. Patients with mucopolysaccharidosis also may

have dilated perivascular spaces (27). However, these diseases can be easily excluded by the clinical findings.

Our two cases showed the unilaterality of the disorder. However, we have no explanation for its cause. In conclusion, when multiple cystic foci are seen that conform to the course of the penetrating arteries in the white matter, with CSF signal on all pulse sequences in patients without neurologic deficit, one should consider dilatation of the perivascular spaces high on the list of possible diagnoses.

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