Neuroradiology (1998) 40: 27–31

M. Adachi
T. Hosoya
T. Haku
K. Yamaguchi

Dilated Virchow-Robin spaces: MRI pathological study

Received: 14 March 1997
Accepted: 2 May 1997

M. Adachi (✉) · T. Hosoya · T. Haku · K. Yamaguchi
Department of Radiology,
Yamagata University School of Medicine
2-2-2 Iidanishi Yamagata, 990-23 Japan
Tel. +81-236-33-1122; fax +81-236-28-5389

Abstract We performed a histopathological study of two human brains to look at dilated Virchow-Robin (V-R) spaces in the anterior perforated substance and putamen. We measured the diameter of 74 arteries in 54 dilated V-R spaces. In 28 patients without neurological deficits we ascertained the characteristic location of dilated V-R spaces in the anterior perforated substance and basal ganglia on MRI, measuring the distance from 64 foci of cerebrospinal fluid signal intensity to the centre of the mamillary body on 1 mm thick images.
In the histopathological study, the mean diameter of the arteries was 39.0 ± 36.0 μm. Dilatation of the V-R space was observed from the end of the indentation of the pial membrane towards the brain surface along the perforating artery. In the MR images, the mean distance from the dilated V-R space to the mamillary body was 10.0 ± 4.5 mm. The V-R space was confined to a fixed level in the lower part of the basal ganglia, and not found near the brain surface.

Key words Brain, anatomy · Magnetic resonance imaging,

Introduction

We often see foci of cerebrospinal fluid (CSF) signal intensity in the basal ganglia on MRI in patients without neurological deficits. In general, such foci, found in the anterior perforated substance and basal ganglia in all age groups, are considered to be enlarged Virchow-Robin (V-R) spaces. However, the characteristics of the dilated V-R space and the mechanism of its dilatation are not well understood. To ascertain the location of the dilated V-R space histopathologically and to explore the mechanism of its dilatation, we looked at these foci using pathological examination and MRI.

Materials and methods

Pathological study

We used tissue blocks obtained from two human brains in which dilated V-R spaces were observed. These brains were those of a woman aged 67 years who had had a subarachnoid haemorrhage, and of a man aged 61 years who had had heart failure. There were six 5-mm-thick tissue blocks (four from the woman and two from the man), which were cut in the coronal plane. They were fixed with 10 % formaldehyde solution and embedded in paraffin. We obtained 10 contiguous sections from each block (for a total of 60 sections). Sections 7 μm in thickness were stained with haematoxylin and eosin for histological examination. On photomicrographs, we measured the diameter of a total of 74 arteries without sclerotic changes, within 54 dilated V-R spaces.

MRI study

We looked at 64 foci of CSF signal intensity in the anterior perforated substance and putamen of 13 men and 15 women ranging in age from 26 to 70 years (mean 58.0 ± 12.1 years). These patients

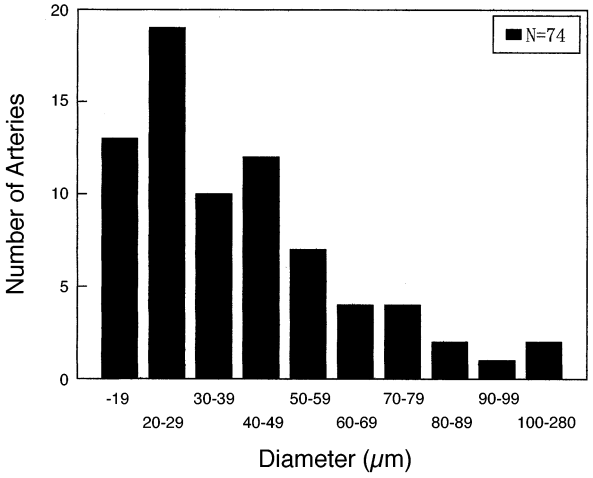


Fig. 1 Diameter of arteries within dilated Virchow-Robin spaces

had no neurological deficits but had sought treatment for headache or vertigo. We looked at well-demarcated foci with CSF signal intensity of which the smallest diameter was more than 2 mm but less than 5 mm. Using a 1.5-T superconductive system, we obtained images, using three-dimensional Fourier transformation-spoiled gradient-recalled acquisition in the steady state (3DFT-SPGR) with the following parameters: field of view 16–18 cm, relaxation time 26 ms, echo time 4.5 ms, flip angle 35°, matrix 256 × 192 × 120, slab thickness 120 mm, one excitation. We measured the distance from the centre of the mamillary body to the centre of each focus, using a 1-mm-thick axial image parallel to the plane including the anterior and posterior commissures.

Results

In the pathological study, the widest portion of the V-R space included small-diameter arteries: mean ($n = 74$) 39.0 ± 36.0 μm (Fig. 1). The cavity of the dilated V-R space was covered by a pial membrane we took to be the inner pial layer (Fig. 2). The dilatation of the V-R space extended from the end of the pial membrane, with an indentation towards the brain surface (Fig. 3). No pathological changes were observed in the parenchyma surrounding the dilated V-R space, and no sclerotic changes in the arteries within it.

In the imaging study, the majority of the well-demarcated foci were in the anterior perforated substance the lower part of the putamen (Fig. 4). The mean distance from the centre of the 64 foci to the centre of the mamillary body was 10.0 ± 4.5 mm (Fig. 5). Although such foci were often present at a fixed level deep to the surface of the brain, none were seen near the surface.

Discussion

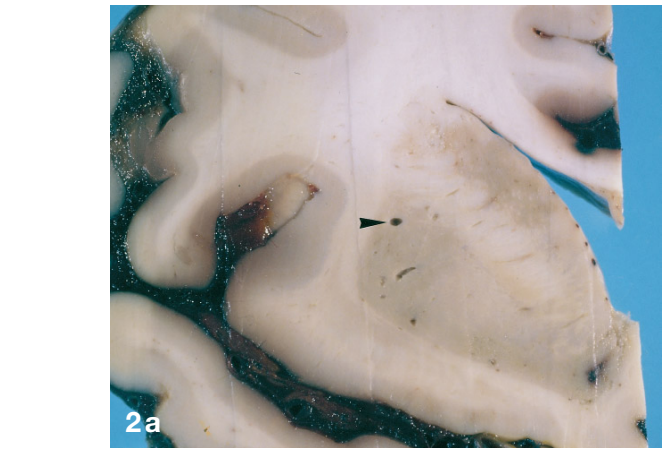
Although the term Virchow-Robin space refers to the extension of the subarachnoid space accompanying a vessel penetrating the cerebral cortex, in the recent anatomical literature, within the brain parenchyma the small arteries are surrounded by the inner and outer pial layers which are invaginated to the level of the capillaries. Deeper in the brain, the outer layer is connected with the inner at the arterioles, and these pial layers form the pial space as an enclosed space [1]. In the subarachnoid space, the vessels are covered with the arachnoid membrane, but the latter does not follow the vessels on their intracerebral course.

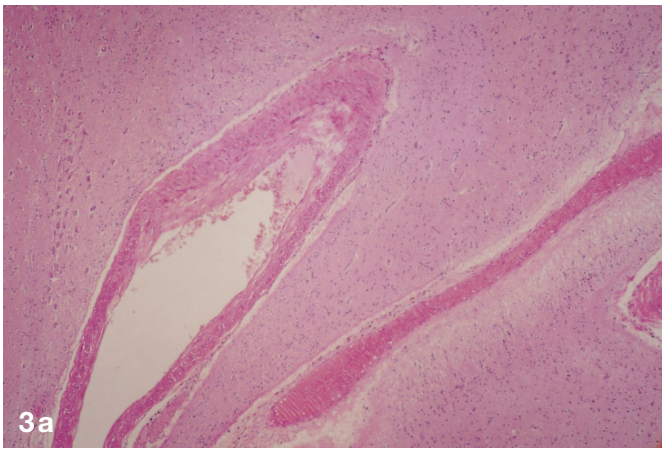
In our pathological study, we found that the greater part of the dilated V-R spaces included small arteries. The mean diameter of these arteries was about 40 μm, while that of proximal portion of a lenticulostriate artery was almost 300 μm. The dilatation of the V-R space was observed to extend from the end of the indentation of the pial membrane at the arteriole towards the brain surface. The pial layer constituted the wall of the dilated V-R space. This membrane we thought to be the inner pial layer, because there was no membrane between it and the glia limitans. We therefore suggest that the full length of the V-R space does not dilate uniformly; rather, it probably begins to dilate at the end of the pial membrane accompanying the intracerebral vessels.

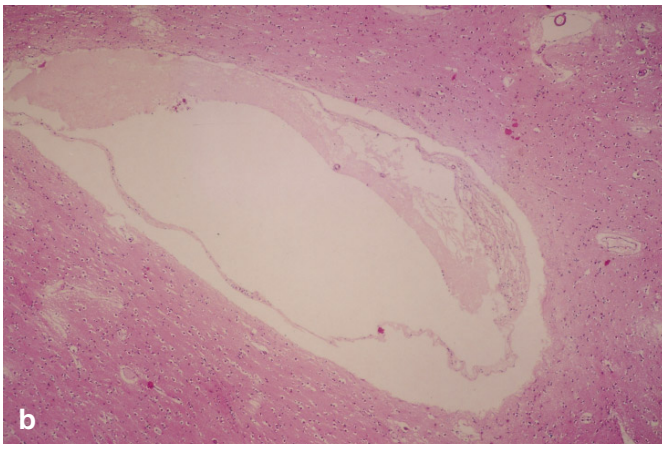
According to the recent literature dealing with large V-R spaces, the foci in the inferior third of the anterior perforated substance and basal ganglia are invariably perivascular spaces around branches of lenticulostriate arteries. However, in the upper two thirds, cavities in brain specimens a usually found to be lacunar infarcts. These tend to give high signal or to have high-signal edges on proton-density images and tend to be relatively large usually 5 mm or more in diameter [2]. In other descriptions, MRI routinely demonstrated small foci of CSF signal intensity, a large V-R space, on either side of the anterior commissure at the level of the inferior third of the basal ganglia [3].

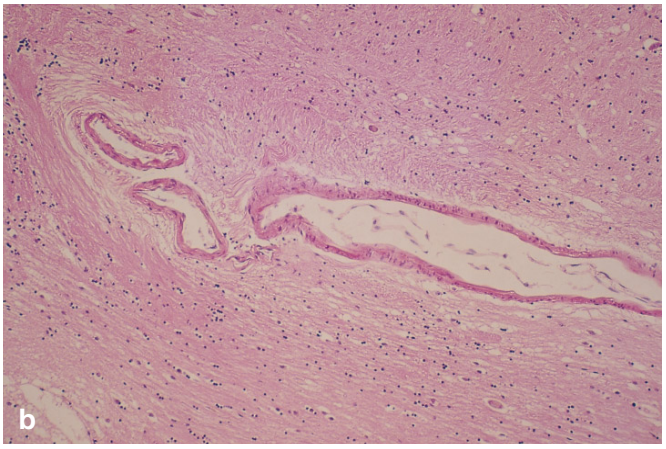
The greater part of the foci with CSF signal intensity were in the lower part of the putamen and the anterior perforated substance, in our images, and we found none near the brain surface. The centre of the large V-R spaces was invariably confined to a fixed level in the basal ganglia, at least 4 mm from the mamillary body. These findings corroborate the characteristic histopathological tendency of the V-R space to dilate from the end of the pial membrane accompanying the intracerebral vessels.

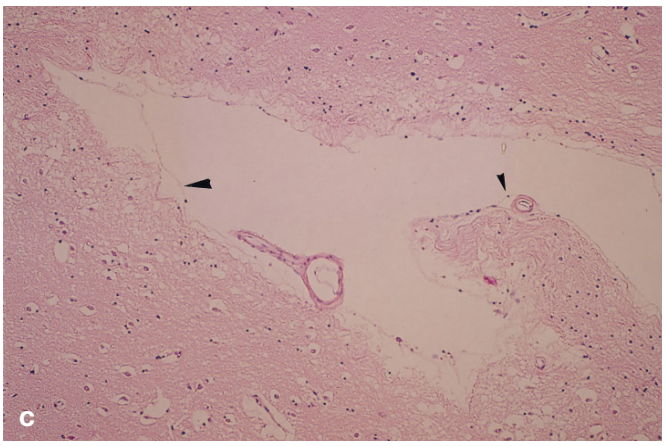
It is not clear why the pial space, as an enclosed space, should dilate. Heier et al. [3] reported that age (an index of atrophy), hypertension, dementia and incidental white-matter lesions were significantly associated with large V-R spaces [3]. However, in their multiple logistic regression analysis, only age remained significant, and large V-R spaces were interpreted as possibly a natural phenomenon in the ageing brain [3]. Braffman et al. [4] did not observe any pathological











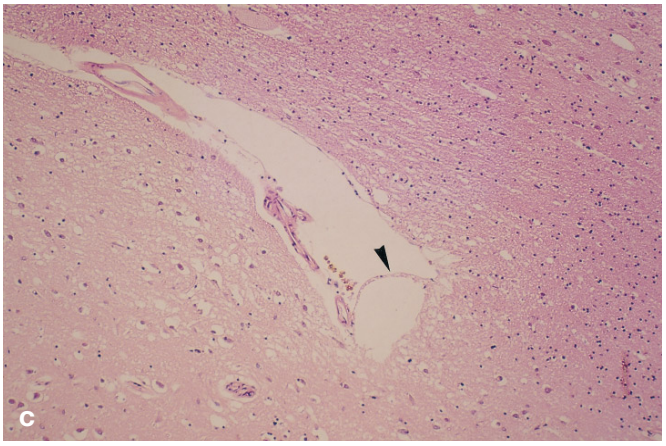


Fig. 2 **a** Coronal section of the brain of a woman aged 67 years demonstrates a dilated Virchow-Robin space in the right putamen *(arrowhead).* **b** A photomicrograph demonstrates this space covered by the pia. It contains small arteries about 10 μm in diameter (haematoxylin and eosin; original magnification × 40). **c** Photomicrograph of another dilated Virchow-Robin space. This is covered by a single cell layer *(large arrowhead),* thought to be the inner pial layer. Another cell layer *(small arrowhead),* close to the small artery, is thought to be the outer pial layer. No ischaemic change is seen in the surrounding parenchyma (haematoxylin and eosin; original magnification × 100)

Fig. 3 **a** Photomicrograph of the proximal part of a lenticulostriate artery 250 μm in diameter does not demonstrate dilatation of the Virchow-Robin space (haematoxylin and eosin; original magnification × 40). No arteriosclerotic changes are observed. **b** The Virchow-Robin space is not dilated around an artery 100 μm in diameter (original magnification × 100). **c** Photomicrograph showing dilatation of the Virchow-Robin space around an arteriole. A single cell layer (thought to be the inner pial layer) covering the cavity arises at the arteries about 10 μm in diameter *(arrowhead)* (original magnification × 100)

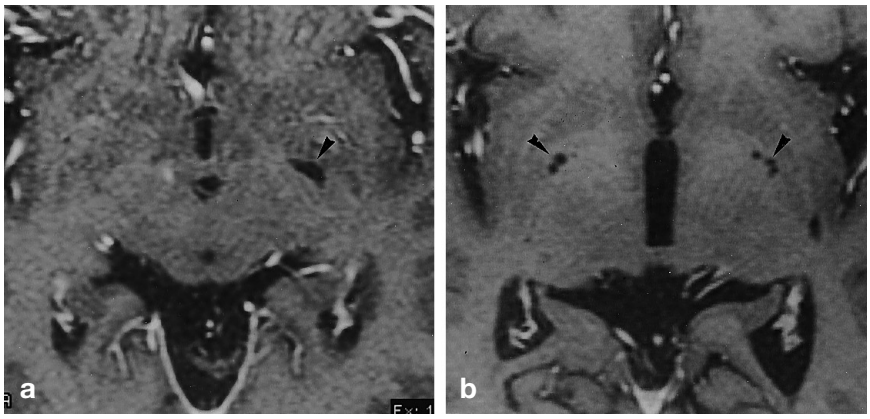


Fig. 4 **a** Axial T1-weighted image of a woman aged 65 years. A well-demarcated focus of CSF signal intensity *(arrowhead)* is seen in the lower part of the left putamen. This wedge-shaped area has no mass effect. **b** Axial T1-weighted image in a man aged 61 years. Several small foci *(arrowheads)* are seen on both sides in the lower putamen; they are round and well-demarcated

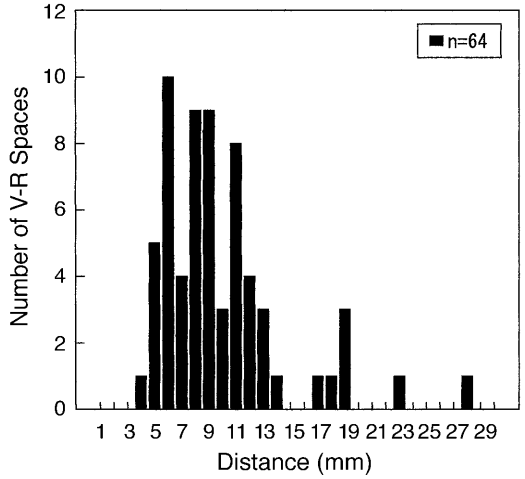


Fig. 5 Distance from the centre of the large Virchow-Robin (V-R) space to the mamillary body

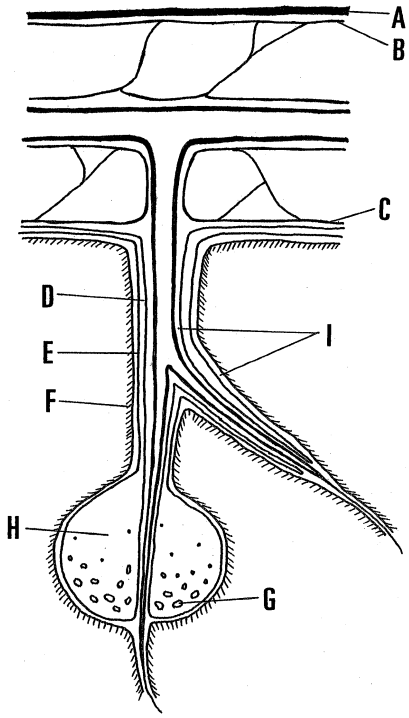


Fig. 6 Diagram of the relationships of the pial membrane and the dilated Virchow-Robin space. *A* dura mater *B* outer *C* inner arachnoid layers *D* outer *E* inner pial layer *F* glia limitans *G* fenestrae *H* pial space *I* intercellular compartment

changes in the parenchyma surrounding the dilated perivascular spaces, and Jungreis et al. [2] suggested that dilated V-R spaces are indeed benign normal variants. The loosening of the adventitia of the blood vessels from the surrounding brain tissue was attributed by Hughes [5] to spiral pro elongation of small intracerebral arteries under the pulsation of elevated blood pressure [5]. Awad et al. [6] reported that vascular ectasia and dilated perivascular spaces reflect skrinkage or atrophy of the brain parenchyma around blood vessels and result in an extensive network of tunnels filled with extracellular water.

Spiral elongation of blood vessels and brain atrophy could cause the V-R space to dilate. However, if these factors were the main causes of dilatation, the full length of the network of the space should dilate uniformly. Our histopathological and MRI studies suggest that some other factor may play a part. Zhang et al. [7] found that the pial membrane surrounding the metarteriole was fenestrated. On the other hand, with regard to drainage of the interstitial fluid in the brain, Krisch et al. [1] reported that with intracerebral injection of horseradish peroxidase, the intercellular compartment (the subpial space, between the vessel and the outer pial layer) was continuously labelled; they suggested that interstitial fluid drained through the intercellular compartment. Bradbury et al. [8] also noted that the perivascular space was the drainage route of interstitial fluid.

We speculate that the interstitial fluid may gradually leak from the intercellular compartment to the pial space around the metarteriole through the fenestrae in the brain parenchyma (Fig. 6). Therefore, the Virchow-Robin space would be likely to dilate around a small artery, and the dilatation to be observed within a fixed level in the basal ganglia on MRI.

References

1. Krisch B, Leonhardt H, Oksche A (1984) Compartments and perivascular arrangement of the meninges covering the cerebral cortex of the rat. Cell Tissue Res 238: 459–474
2. Jungreis CA, Kanal E, Hirsch WL, Martinez A, Moossy J (1988) Normal perivascular spaces mimicking lacunar infarction: MR imaging. Radiology 169: 101–104
3. Heier LA, Bauer CJ, Schwartz L, Zimmerman RD, Morgello S, Deck MD (1989) Large Virchow-Robin spaces: MR-clinical correlation. AJNR 10: 929–936
4. Braffman BH, Zimmerman RA, Trojanowski JQ, Gonatas NK, Hickey WF, Schlaepfer WW (1988) Brain MR: pathologic correlation with gross histopathology. 1. Lacunar infarction and Virchow-Robin spaces. AJNR 9: 621–628
5. Hughes W (1965) Origin of lacunes. Lancet ii: 19–21
6. Awad IA, Johnson PC, Spetzler RF, Hodak JA (1986) Incidental subcortical lesions identified on magnetic resonance imaging in the elderly. II. Postmorten pathological correlations. Stroke 17: 1090–1097
7. Zhang ET, Inman CBE, Weller RO (1990) Interrelationships of the pia mater and the perivascular (Virchow-Robin) spaces in the human cerebrum. J Anat 170: 111–123
8. Bradbury MWB, Cserr HF, Westrop RJ (1981) Drainage of cerebral interstitial fluid into deep cervical lymph of the rabbit. Am J Physiol 240: 239–336