focal neurological dysfunction and pseudo-bulbar palsy.[4] This last aspect as the CT-pathological ones related to the sequence demyelination and gliosis — white matter loss and hydrocephalus, is in agreement with the slow progression of the disease (10 years and more). The clinical CT-pathological diagnosis of Binswanger's disease considered above does not apply to the cases presently by the Authors and to the subcortical alterations they found, these latter rightly attributed by the same to terminal episodes of hypoxic-ischaemic damage in patients with multi-infarctual cortical alterations. The Authors reflections on the opportunity of not considering Binswanger's disease as a separate entity from M.I.D. do not therefore appear to be conclusive. On the other hand the studies above reported[1,3,4] suggest a possible separation, among cerebro-vascular patients, of those cases with clinical-CT-pathological features of localized or diffuse, acute or chronic, subcortical ischaemia due to sclero-jalinotic alterations of the small cerebral arteries. The classical pathological pictures of "état lacunaire", "état criblè" and Binswanger's disease, often overlapping, could then be valued in this nosological context.

Domenico Inzitari, M.D.
Mario Mascalchi, M.D.
Department of Neurology
University of Florence
V.le Morgagni 85, Florence, Italy

References

1. Goto K, Ishii N, Fukasawa H. Diffuse white-matter disease in the geriatric population. Radiology 1981; 141: 687-695.
2. Binswanger O. Die Abgrenzung der allgemeinen progressiven Paralyse. Berl Klin Wochenschr 1984; 31: 1103-1105, 1137-1139, 1180-1186.
3. Caplan LR and Schoene WC. Clinical features of subcortical arteriosclerotic encephalopathy (Binswanger disease). Neurology 1978; 28: 1206-1215.
4. Kinkel WR, Jacobs L, Polachin I et al. Subcortical arteriosclerotic encephalopathy (Binswanger's disease) computed tomographic, nuclear magnetic resonance and clinical correlations. Arch Neurol 1985; 42: 951-959.

REPLY

To the Editor

We thank Drs. Inzitari and Mascalchi for reminding us of the "in vivo" evaluation of the subcortical white matter alterations in Binswanger's Disease. According to Binswanger[1] and other authors, this disease is characterized: clinically, by progressive deterioration of intellect, often associated with a variety of focal symptoms and signs in clinical plateaux or even with improvement; pathologically by large patches of demyelination of cerebral white matter with sparing of the arcuate fibres and also frank infarcts; and etiologically, by arteriosclerosis of the cerebral arteries. Binswanger was a pathologist who first described this disease by gross appearance without microscopic investigation. Later reports with postmortem examination produced many controversies: some cases without dementia[2,3] and some without arteriosclerosis of the cerebral arteries[4,5] were described.

CT findings are, after all, shadows due to various sorts of lesions, such as oedema, softening, demyelination, etc., the true nature of which can only be determined by pathological investigation or long follow-up with CT study. The hypodense area around a cerebral haemorrhage is generally thought to be due to oedema by many CT specialists, but in our study, seven of twelve haemorrhages surrounded by a ring of hypodensity on CT were later proved to be necrosis of different ages histologically.[6] Laizou et al[7] performed clinical and radiological investigations of subcortical arteriosclerotic encephalopathy with CT and cited two cases showing a low density area in the white matter which subsequently subsided, so that the hypodense areas could not be the basis of subcortical arteriosclerotic encephalopathy.

One must not neglect the anatomy of the arteries of the cerebral white matter which are supplied by long and short medullary branches centripetally and ventricular branches centrifugally, without anastomoses.[8] Changes in these arteries do not produce large patches of demyelination with sparing of the arcuate fibres; this pattern of involvement is caused only by obstruction of venous drainage.

Our denial of the existence of this disease requires further confirmation to be conclusive. We plan more work to support our contention, and hope that Drs. Inzitari and Mascalchi also will continue their follow-up studies and perhaps secure pathological evidence proving it to be a true disease.

Ke-wei Huang,
Division of Neuropathology
Chinese PLA General Hospital and Military
Post-graduate Medical School
Beijing, People's Republic of China

References

1. Binswanger O. Die Abgrenzung de allgemeinen progressiven paralyse. Berl Klin Wochenschr 1894; 31: 1103-1105, 1137-1139, 1180-1186.
2. Olszewski J. The myth of Binswanger Disease (Discussion). J Neuropath Exp Neurol 1961; 20: 312.
3. De Reuck J, Crevits L, De Coster W et al. Pathogenesis of Binswanger chronic progressive subcortical encephalopathy. Neurology 1980; 30: 820-928.
4. Poppe W and Tenustedt A. Ein Beitrag Zur Encephalopathia subcorticalis Binswanger. Psychiat Neurol 1963; 145: 27-35.
5. Orlando R and Orlando YJC. Contribución al conocimiento de las leucocefaloses tipo Binswanger. Comptes-Rendues Ve Congr Neur Intern lisbonne 1953; Lisboa 1954; 4: 60-74.
6. Huang KW, Wang LN, Cao DQ. Histopathologic correlation of CT findings in cerebral hemorrhage and infarct. Chin Med J 1984; 97: 483-492.
7. Loizou LA, Jefferson SM, Thomas SW. Subcortical arteriosclerotic encephalopathy (Binswanger type) and cortical infarct in a young normotensive patient. J Neurol Neurosurg Psychiat 1982; 45: 409-417.
8. De Reuck J. The human periventricular arterial blood supply and the anatomy of cerebral infarctions. Eur Neurol 1971; 5: 321-334.

https://doi.org/10.1017/S0317167100036131 Published online by Cambridge University Press