Honeycomb-like appearance in the brain of a former boxer with hyperhomocysteinemia, severe carotid disease and hemorrhagic stroke

Dear editor,

Brain perivascular spaces (PVSs) or Virchow-Robin spaces are pial-lined, interstitial fluid-filled structures surrounding the arteries entering brain parenchyma.1 Enlarged PVSs are radiological manifestation of posttraumatic encephalopathy2 and a common biomarker of small vessel disease (SVD).3 Exuberant forms of PVSs are rarely reported.

A 55-year-old male truck driver, retired professional boxer with a history of severe untreated hypertension, obesity, brainstem lacunar stroke, dyslipidemia, heavy smoking, right sided severe internal carotid stenosis (SICS) (90–99%) and hyperhomocysteinemia was brought to the emergency room because of sudden onset of right hemiparesis followed by focal complex hemiconvulsion. On neurological examination the

**Figure 1.** Brain CT showing severe leukoaraiosis and exuberant enlarged perivascular spaces lesions in the hemispheric/subcortical cerebral white matter predominately affecting the right side and left ganglionic hemorrhage with ventricular dissection (a) to (e) as also the radiological findings present at first stroke, 3 years earlier (f).
patient was 10 in Glasgow coma score, showed slight right spastic hemiparesis and bilateral pyramidal signs. Aside from severe hypertension (170/110 mmHg) and obesity (body mass index of 35.5) the general examination was unremarkable. The brain computed tomography (brain CT) revealed the presence of left sided ganglionic hypertensive hematoma with dissection to the lateral ventricles, severe predominantly right sided leukoaraiosis and marked dilated PVSs resembling a “honeycomb” (Figure 1(a) to (e)). The previous brain CT from 3 years ago showed that the aforementioned SVD markers were already present (Figure 1(f)).

Despite aggressive medical management, the patient died after 2 days. At the first stroke, the patient was discharged without finishing investigation including brain magnetic resonance imaging (brain MRI) and was lost to follow-up. The basic diagnostic workup (hemogram, urinalysis, renal /hepatic function, echocardiogram) and the extensive blood investigation was negative, with exception of several right sided SICS and high homocysteine levels (≥50 μmol/L).

Homocysteine is implicated in acceleration and development of a generalized small-vessel disease. Therefore, it is reasonable to consider that the coincidental combination of untreated traditional vascular risk factors, hyperhomocysteinemia and previous exposure to brain traumatic sport may have contributed to the severe expression of SVD. The right side predominance of SVD can be explained by the presence of ipsilateral SICS causing chronic hypoperfusion and further enlargement of PVSs. PVSs can rarely enlarge causing symptoms and therefore be mistaken by diseases such as multicystic tumors or infectious diseases. However, these alternative diagnoses are highly improbable in our patient because of the absence of systemic manifestations as also the relative stability of the brain CT performed with 3-year interval. Furthermore, with exception of microbleeds, which we cannot rule out by brain CT, our patient showed all signs of small vessel disease with deep ganglionic hypertensive hemorrhage, leukoaraiosis, lacunar stroke and severe dilated PVSs with honeycomb appearance.

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