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LETTER TO THE EDITOR

Combination of acute hypertensive striatocapsular hemorrhage and mirror previous asymptomatic slit-like hemorrhage in a young patient: a new radiological clue for cerebral small vessel disease?

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Dear Editor,

Asymptomatic striatocapsular slit-like hemorrhage (SSH) has been recently described as a potential new radiological marker for hypertensive cerebral small vessel disease (cSVD) [1]. We herein report a case of a young patient with a left acute putaminal hemorrhage associated with no other cSVD marker than a contralateral asymptomatic SSH.

This 35-year-old patient, without medical history or any known cardiovascular risk factors, awakened with a right-sided hemiparesis and hemianesthesia associated with dysarthria and mild aphasia. The National Institutes of Health Stroke Scale (NIHSS) was evaluated at 10. Blood pressure was 198/119 mmHg. The non-contrast brain computed tomography revealed an hyperdense left putaminal hematoma (40 × 20 mm) together with a contralateral hypodense asymptomatic SSH (Fig. 1). CT angiography (CTA) was unremarkable. The patient received intravenous Nicardipine for the first days followed by oral anti-hypertensive therapy combining Perindopril and Amlodipine at the stroke unit.

The assessment of hypertension target organ damage revealed left ventricular hypertrophy (LVH) with positive Sokolow index and elevated left ventricular mass index of 182 g/m2 but preserved left ventricular ejection fraction (64%). Fundoscopy demonstrated hypertensive retinopathy with grade II angiosclerosis. No albuminuria was detected.

The brain magnetic resonance imaging (MRI) confirmed the acute left putaminal hematoma and the thin linear band of hemosiderin deposits within the right putamina corresponding to an SSH. Other standard markers of cSVD were absent: no white matter hyperintensities (WMH), lacunes, deep cerebral microbleeds (CMBs), recent small subcortical infarcts nor enlarged perivascular spaces were detected.

We found no evidence for a secondary origin of this hemorrhage as the brain MRI ruled out intracranial venous thrombosis or tumor, the lumbar puncture was normal, the genetic test for Fabry disease and the toxicology test were negative, and no sign of hematological disease or coagulation disorders were observed in the blood test.

Given the presence of bilateral deep brain hemorrhage and uncontrolled hypertension, with already deleterious consequences on extracerebral target organs, digital subtraction angiography was not performed as CTA and MRA already reasonably ruled out vascular malformation.

The final diagnosis was therefore a HA-related acute left putaminal hemorrhage due to untreated high blood pressure.

After five days, the patient was discharged with only a mild right facial paresis and hemihypesthesia (NIHSS of 2) and a modified Rankin Scale of 1.

The radiological key-feature was the concomitant observation of an acute putaminal hematoma on one side, together with a mirror asymptomatic SSH in the absence of other conventional markers for cSVD.

The exact physiopathology of the cSVD is still unclear, but a widely accepted hypothesis is that uncontrolled chronic hypertension, associated or not with other vascular risk factor (elderly age, diabetes mellitus…), can gradually lead to

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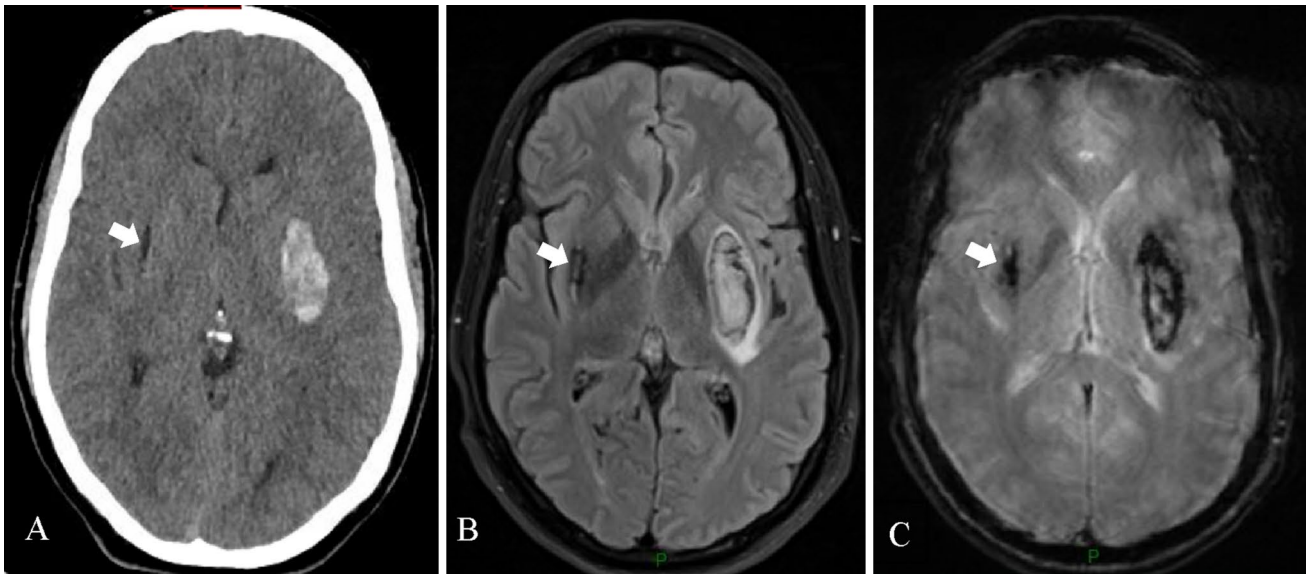

Fig. 1 NCCT and MRI of acute left-side putaminal hemorrhage and contralateral striatocapsular slit-like hemorrhage (SSH).**a** Axial transverse non-contrast brain computed tomography view showing the acute left-side putaminal hemorrhage together with a contralateral asymptomatic SSH (white arrow). **b** MR axial transverse FLAIR view in similar slice location failing to demonstrate standard other cSVD markers. **c** MR axial transverse T2*-weighted view confirming the hemorrhagic origin of the right SSH (white arrow)

increased flow velocity and pulsatility in the perforating arterioles, causing endothelial damages and blood–brain barrier leakage. This leads to endothelial dysfunction and finally to focal wall necrosis and microscopic « pseudoaneurysms», resulting in vessel wall ruptures and then deep ICH or microbleeds. [2, 3].

It is known that hypertensive arteriopathy (HA), responsible for deep perforating vessels arteriopathy, is most often associated with non-lobar ICH and CMBs, and especially in the striatocapsular area. While cerebral amyloid angiopathy is typically associated with cortico-subcortical leptomeningeal small vessels arteriopathy leading to lobar ICH and CMBs in patients over 55 years of age [2–4].

Asymptomatic striatocapsular slit-like hemorrhage, has recently been described as a potentially new radiological marker of cSVD by Tsai et al. [1]. This entity is associated with a hypointense slit-like lesion involving the lateral part of the lenticular nucleus or the external capsule, on susceptibility-weighted MRI, without associated symptomatology in the patient's clinical history.

The marker was present in 10% of their 246 patients presenting with first-ever symptomatic spontaneous HA-related ICH and was more frequently associated with LVH, albuminuria, lobar microbleeds and elevated lesion load in WMH or lacunes. The same author showed that mixed ICH/CMB locations were mainly associated with chronic hypertension, and with more severe parenchymal lesions and a higher rate of ICH recurrence. This marker is then probably reflecting a more severe hypertension, thereby requiring a more aggressive anti-hypertensive treatment.

Moreover, the asymptomatic aspect of a previous chronic cerebral hemorrhage (PCH) should probably not be considered inferior to a symptomatic one, as in a recent study of patients with acute ischemic stroke, patients with asymptomatic PCH had similar clinical and neuroimaging features to symptomatic ones [5].

Since SSH is found here in the absence of other cSVD markers, its presence alone could already be a marker of more severe hypertension and its underlying HA, even before the appearance of a significant cSVD and its other markers.

The combination of deep acute ICH with previous asymptomatic SSH should therefore strongly draw the consideration for potentially severe systemic, uncontrolled hypertension, even in the absence of usual cSVD markers at brain imaging.

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Declarations

Conflicts of Interest The authors declare that they have no conflict of interest.

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Consent to participate Not applicable.

Consent for publication Patient consent was obtained.

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