Convexity subarachnoid hemorrhage

Convexity subarachnoid hemorrhage (cSAH), defined as intrasulcal bleeding restricted to hemispheric convexities, has several etiologies: reversible cerebral vasoconstriction syndrome, cerebral amyloid angiopathy, and internal carotid artery stenosis or occlusion [1].

MRI scans of 130 consecutive patients meeting modified Boston criteria for probable CAA were analysed for cortical superficial siderosis (focal, ≤3 sulci; disseminated, ≥4 sulci), and key small vessel disease markers. We compared clinical, imaging and cortical superficial siderosis topographical mapping data between subjects with versus without acute cSAH, using multivariable logistic regression.

Charidimou et al., included 33 patients with probable CAA presenting with acute cSAH and 97 without cSAH at presentation. Patients with acute cSAH were more commonly presenting with transient focal neurological episodes (76% vs 34%; p<0.0001) compared with patients with CAA without cSAH. Patients with acute cSAH were also more often clinically presenting with transient focal neurological episodes compared with cortical superficial siderosis-positive, but cSAH-negative subjects with CAA (76% vs 30%; p<0.0001). Cortical superficial siderosis prevalence (but no other CAA severity markers) was higher among patients with cSAH versus those without, especially disseminated cortical superficial siderosis (49% vs 19%; p<0.0001). In multivariable logistic regression, cortical superficial siderosis burden (OR 5.53; 95% CI 2.82 to 10.8, p<0.0001) and transient focal neurological episodes (OR 11.7; 95% CI 2.70 to 50.6, p=0.001) were independently associated with acute cSAH.

This probable CAA cohort provides additional evidence for distinct disease phenotypes, determined by the presence of cSAH and cortical superficial siderosis [2].

Chertcoff et al., retrospectively analyzed all cases of convexity subarachnoid hemorrhage admitted to our hospital between January 2012 and April 2017. Demographic features, clinical characteristics, complementary investigations, etiology and mortality were assessed. Twenty patients (65% females) were identified. Mean age: 53 years (range, 15-86 years).

Symptoms on admission: headache (65%), sensory and/or motor symptoms (50%) and seizures (35%). Commonest causes: cerebral vein thrombosis (20%), reversible cerebral vasoconstriction syndrome (20%) and cerebral amyloid angiopathy (20%). Two patients died.

Convexity subarachnoid hemorrhage may be related to a wide spectrum of etiologies. In our patients, an increased prevalence of cerebral vein thrombosis was observed. Mortality was low and not related to the bleeding itself [3].

Fukuma et al. retrospectively investigated patients admitted to the hospital between 2005 and 2013 with ischemic stroke or transient ischemic attack caused by cerebral artery dissection. Cerebral artery dissection was diagnosed by cervical or cerebral magnetic resonance imaging (MRI) or computed tomography (CT) showing a wall hematoma. CT angiography, ultrasonography, or intra-arterial digital-subtraction angiography detected cerebral artery dissection if a double lumen, string sign, intimal

flap, or dissecting aneurysm was observed at a nonbifurcation site.

They used CT or MRI to detect cSAH, which was defined as blood collection restricted to one or few cerebral sulci without extending to the basal cisterns, ventricles, or Sylvian and interhemispheric fissures. Demographic, neuroimaging, treatment, and prognostic data were collected.

In total, 82 patients were diagnosed with ischemic stroke caused by cerebral artery dissection. The following arteries were affected: the ICA (9 patients), anterior cerebral artery (ACA; 12 patients), middle cerebral artery (MCA; 12 patients), vertebral artery (37 patients), basilar artery (5 patients), posterior cerebral artery (2 patients), and posterior inferior cerebellar artery (4 patients). In addition, 1 patient presented with simultaneous dissection in both the vertebral and internal carotid arteries, and 6 patients (7%) presented with cSAH (3 men and 3 women, age 39-67 years). The MCA was dissected in four cases and the ACA in two cases, with cSAH frequencies of 33 (4 of 12) and 17% (2 of 12), respectively, in those vessels. Artery dissection in the vertebrobasilar artery system was not responsible for cSAH (0 of 48). In all the MCA dissection cases, cSAH occurred in the arterial border zone between the ACA and MCA territories. Although 2 patients showed early reperfusion with temporary cSAH enlargement, cSAH was self-limiting. Antithrombotic treatment did not complicate the clinical course when used in 4 patients during acute or subacute phases. All patients achieved a 3-month poststroke modified Rankin Scale of 0-2 [4)].

Case reports

Bandeo et al., presented a 26-year-old female patient with a diagnosis of UC taking Adalimumab. She received her last doses the same day she was admitted to our hospital for an acute severe UC exacerbation. Steroids were added to the treatment. Five days after admission she presented a thunderclap headache with photophobia, nausea, and vomiting. An MRI was performed showing left frontal convexity subarachnoid hemorrhage and hyperintense lesions on T2-weighted and FLAIR sequences located in both occipital lobes, left cerebellar hemisphere, and brainstem. Digital angiography was unremarkable. Adalimumab was discontinued but persisted on treatment with steroids. The patient evolved with complete resolution of her symptoms and was discharged with a normal neurological exam. Two months later, she was asymptomatic and her MRI revealed superficial siderosis secondary to cSAH with resolution of white matter hyperintensities. Convexity subarachnoid hemorrhage in our patient could be secondary to PRES or to RCVS. Analogous MRI findings can be observed in both syndromes, along with similar clinical and angiographic findings. This suggests that both conditions may reflect different manifestations of the same pathology, in which vascular tone and endothelial dysfunction play a major role. To our knowledge, this is the first report of a patient with severe UC and convexity subarachnoid hemorrhage associated with Adalimumab [5)].

References

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