Nontraumatic Convexity Subarachnoid Hemorrhage: Different Etiologies and Outcomes

Ruth Geraldes, MD, MSc, Paulo R. Sousa, MD, Ana C. Fonseca, MD, MSc, Filipa Falcão, MD, Patrícia Canhão, MD, PhD, and Teresa Pinho e Melo, MD

Background: Nontraumatic convexity subarachnoid hemorrhage (cSAH) is a rarely reported condition with multiple etiologies. We report the clinical presentation, imaging findings, etiologies, and long-term outcomes of a case series of cSAH. *Methods:* We retrospectively analyzed consecutive cases of cSAH, admitted at a Stroke Unit of a tertiary hospital (January 2006 to March 2012). Recorded variables were demographics, clinical presentation, complementary investigation, etiology, and outcome. *Results:* We included 15 patients (9 men, median age of 65 years), 7% of the 210 nontraumatic SAH patients in this period. The most common clinical manifestation was a focal neurologic deficit. Predominant location of the cSAH was frontal. In 5 cases, there was a clinical significant internal carotid artery (ICA) atheromatous stenosis, ipsilateral to cSAH. Two patients had a possible cerebral amyloid angiopathy (CAA) at presentation. There were 2 cases of reversible cerebral vasoconstriction syndrome, 1 cerebral venous thrombosis, 2 dural fistulae, and 3 undetermined. Short-term outcomes were good in most patients. At follow-up (24.3 months), 2 of the patients with undetermined etiology had a lobar hematoma conferring a severe disability, and the diagnosis of CAA was made. There were no other relevant events or added disability in the other patients. *Conclusions:* Significant ICA atherosclerotic stenosis was the most frequent cause of cSAH in our series, reinforcing that cSAH should prompt vascular imagiological evaluation including cervical vessels. Outcomes in cSAH seem to be related to etiology. Patients with undetermined etiology should be followed up because cSAH may be the first manifestation of CAA. **Key Words:** Convexity subarachnoid hemorrhage—atherosclerotic carotid artery stenosis or occlusion—hyperdense sulcus—cortical subarachnoid haemorrhage—acute ischemic stroke—amyloid angiopathy.

Background

Nontraumatic convexity subarachnoid hemorrhage (cSAH) is a rarely reported condition in which the hemorrhage is located in one or a few cortical sulci of the brain, without spread into the sylvian fissure, interhemispheric fissure, basal cisterns, or the ventricles. The absence of blood in the basal cisterns and fissures differentiates this subtype of subarachnoid hemorrhage (SAH) from aneurysmal and nonaneurysmal perimesencephalic SAH. The largest reported cSAH series has 34 cases.[1] cSAH has been associated with multiple vascular causes, such as arteriovenous malformations, dural fistulae, cavernomas, cerebral venous thrombosis (CVT), reversible cerebral vasoconstriction syndrome (RCVS), moyamoya disease, arterial dissection/stenosis, and nonvascular causes, such as brain tumors and abscesses, coagulation disorders, cerebral amyloid angiopathy (CAA), and others.[1-9] A stepwise diagnostic complementary evaluation of patients has been previously proposed.[2,5] Patients with

From the Stroke Unit, Department of Neurosciences (Neurology), Hospital de Santa Maria, Lisbon, Portugal.

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Address correspondence to Ruth Geraldes, MD, MSc, Department of Neurology, Hospital de Santa Maria, Av. Prof. Egas Moniz, 1649-035 Lisboa, Portugal. E-mail: ruth.geraldes@gmail.com.

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Table 1. *Overview of demographic, risk factors, clinical, laboratory, and imaging findings, etiology, treatments, and outcome of patients*

No.	Age, sex	Presenting signs and symptoms	Brain CT/MRI FLAIR DWI, T2*	AngioMRI/DSA	CVUS and TCD/other exams
1	50, Male	Transient L central facial palsy; dysarthria	R PosC cSAH, R MCA acute and nonrecent ischemia, no microbleeds	ND	Atheromatous stenosis R ICA (85%)
2	67, Female	L facial and brachial paresis, hypesthesia headache	R FP cSAH, R MCA acute and nonrecent ischemia, no microbleeds	R ICA stenosis/ND	Atheromatous stenosis R ICA (95%)
3	59, Male	L hemiparesis	R C cSAH, R MCA acute ischemia, no microbleeds	R ICA occlusion	R ICA occlusion, L ICA stenosis (50%)
4	83, Male	Transient R brachial monoparesis, dysarthria	L P cSAH, L ACA/MCA watershed nonrecent ischemia, no microbleeds	Stenosis of left ICA at carotidal bifurcation	L ICA (carotidal bifurcation) L MCA, ACA stenosis
5	44, Female	Dysarthria, paraphasia	L PreC cSAH, L MCA acute ischemia, no microbleeds	L ICA occlusion/ND	L ICA atheromatous occlusion, R PCA stenosis
6	52, Female	Acute headache	L TO cSAH, no microbleeds	ACA stenosis	Normal
7	59, Female	Acute headache	L P cSAH, bilateral O acute ischemia, no microbleeds	Multiple intracranial stenosis	Bilateral stenosis MCA, L ACA, and PCA
8	73, Male	L hemihypalgesia, L crural ataxia	R TO cSAH, R TO abnormal enhancing voids	R parasagittal dural fistula	Normal
9	65, Male	Headache, transient L arm paresthesia > paresis	R F cSAH/ND	SLS CVT R dural pial fistula	Normal
10	41, Female	Headache, L hemiparesis	R FP cSAH, SLS CVT	SLS CVT	Normal
11	79, M	3 Transient, L brachial paresthesia	R C cSAH, leukoaraiosis, multiple microbleeds, no hemosid.	ND	Nonsignificant atheromatosis
12	68, Male	Aphasia, generalized seizures	L P cSAH leukoaraiosis, 2 microbleeds, no hemosid.	Normal	Normal
13	84, Female	Several episodes L brachial paresthesia monoparesis	R FP cSAH, no microbleeds	Normal	N/EEG: independent slow frontotemporal activity
14	65, Male	Several episodes, R paresthesia and R hemiparesis	L C cSAH/L C cSAH, and R F cSAH, leukoaraiosis, no microbleeds	Normal	Nonsignificant atheromatosis
15	73, Male	Several episodes, R arm paresthesia > paresis	L C cSAH, leukoaraiosis, no microbleeds	Normal	Atheromatous stenosis L ICA (55%), R ICA stenosis (30%)

Abbreviations: ACA, anterior cerebral artery; ACA, anterior communicant artery; AF, atrial fibrillation; CAA, cerebral amyloid angiopathy; cSAH, convexity subarachnoid hemorrhage; C, central; CSF, cerebrospinal fluid; CVT, cortical venous thrombosis; CVUS, cervical vessel ultrasonography; DSA, digital subtraction angiography; DWI, diffusion-weighted imaging; EEG, electroencephalography; F, frontal; FLAIR, fluid-attenuated inversion recovery; FP, frontoparietal; Hemosid., hemosiderin deposits; ICA, internal carotid artery; L, left; LAA, large artery atherosclerosis; MCA, middle cerebral artery; MRI, magnetic resonance imaging; mR1, modified Rankin scale score at discharge; mR2, modified Rankin scale score at follow-up; ND, not done; O, occipital; P, parietal; PCA, posterior cerebral artery; PosC, postcentral sulcus; PreC, precentral sulcus; PT, parietotemporal; R, right; RBC, red blood cells; RCVS, reversible cerebral vasoconstriction syndrome; SLS, superior longitudinal sinus; TCD, transcranial Doppler; TIA, transient ischemic attack; TO, temporo-occipital, Und., undetermined.

cSAH may have a poor outcome.[10,11] In this study, we propose to identify the clinical presentation, imaging findings, and etiologies in patients with cSAH, using a stepwise diagnostic protocol that includes cervical vessel imaging and to analyze long-term outcomes in each etiologic category.

Methods

We conducted a review of consecutive cSAH cases, admitted to the stroke unit of a tertiary hospital, from January 2006 to March 2012. Our Institution Ethical Board reviewed the study, and no consent was found necessary for the retrospective study. cSAH was defined as hyperdensity exclusively in a cortical sulci on brain computed tomography (CT) and/or a hyperintensity on fluid-attenuated inversion recovery (FLAIR) and T2*-weighted sequences hypointensity. Initial noninvasive complementary evaluation included brain magnetic resonance imaging (MRI) with FLAIR and T2* sequences (except if contraindication), angioMRI, cervical vessel ultrasonography (US), and transcranial Doppler (TCD). Other diagnostic procedures were performed if this initial investigation was inconclusive. A normal digital subtraction angiography (DSA) was required in cases classified as "undetermined cause" because CT angiography could not reliably exclude dural fistulae. Three stroke neurologists reviewed all images and classified cSAH etiologies. After