Spontaneous convexial subarachnoid haemorrhage: a case series with different etiologic diagnoses

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Abstract

Objective: Non-traumatic acute convexity subarachnoid haemorrhage (cSAH) is a rare condition caused by various vascular-nonvascular pathologies. The aim of this study was to evaluate the etiologic background of patients with cSAH.

Methods: For this purpose, the files of the patients with cSAH were retrospectively reviewed. The demographic data, clinical presentation, complementary investigations, etiology, and outcome of five patients with cSAH due to amyloid angiopathy and cerebral venous thrombosis were recorded.

Results: There were five patients with cSAH; 4 were women. The most common clinical presentation was focal neurologic signs, followed by acute headache and seizures. Detailed investigations revealed different causes of cSAH as follows: amyloid angiopathy (n=4) and cerebral vein thrombosis (CVT) (n=1). The patient with etiologic diagnoses of CVT was younger than 40 years, and the other patients with amyloid angiopathy were older than 65 years. The haemorrhagic lesion was bilateral in two cases with CVT and amyloid angiopathy. The outcome was favourable in four patients; one patient (case 4), who presented with a higher National Institutes of Health Stroke Scale died.

Conclusion: The etiologic diagnosis of cSAH is essential for determining treatment. Complete diagnostic examination, including parenchymal and vascular imaging should be performed.

Keywords: Amyloid angiopathy, convexial subarachnoid hemorrhage, MR, subarachnoid hemorrhage, venous thrombosis

INTRODUCTION

Non-traumatic subarachnoid haemorrhage (SAH) is mostly due to aneurysm rupture at suprasellar and basal cisterns. There are some locations that suggest other etiologies of SAH. Subarachnoid haemorrhage localised to one or several sulci of the cerebral convexity or sylvian fissure without evidence of blood in the basal cisterns or elsewhere is called convexal SAH (cSAH). It is a relatively rare condition caused by various vascular and nonvascular pathologies. Cerebral venous thrombosis (CVT), vascular malformations, reversible cerebral vasoconstriction syndrome (RCVS), vasculitis, infectious diseases, coagulation disorders, Moyamoya disease, severe atherosclerotic carotid disease, posterior reversible encephalopathy syndrome, cerebral amyloid angiopathy (CAA), and post-carotid endarterectomy may present with cSAH (1–17). The aim of this study was to evaluate the etiologic background of our patients who presented with cSAH.

METHODS

The files of patients who were admitted with cSAH to the stroke unit in the last three years were retrospectively evaluated. Convexal SAH was defined as SAH localised to one or several sulci of cerebral convexity or sylvian fissure without evidence of blood in the basal cisterns or elsewhere.

The demographic data, clinical presentation, the National Institute of Health Stroke Scale (NIHSS) score at admission, complementary investigations, laboratory tests for hypercoagulability and vasculitis, etiologic diagnosis, and
outcome of each patient were recorded. Radiologic investigations including non-enhanced cranial computed tomography (CT) (Asteion; Toshiba, Tokyo Japan), CT angiography (CTA) (Aquillon; Toshiba, Tokyo, Japan), CT venography (CTV) (Aquillon; Toshiba, Tokyo, Japan), brain magnetic resonance imaging (MRI) (Signa; GE, Milwaukee, WI, Verio; Siemens, Erlangen, Germany), magnetic resonance venography (MRV) (Signa; GE, Milwaukee, WI, Verio; Siemens, Erlangen, Germany), magnetic resonance angiography (MRA) (Signa; GE, Milwaukee, WI, Verio; Siemens, Erlangen, Germany), and digital subtraction angiography (DSA) (Multistar Plus/T.O.P.; Siemens, Erlangen, Germany) images were evaluated retrospectively.

Microhaemorrhagic foci were identified according to the criteria proposed by Greenberg et al.: black round or ovoid lesions on T2*-weighted MRI; smaller than 5 mm with blooming effect on T2*-weighted MRI and devoid of signal hyperintensity on T1-weighted or T2-weighted sequences; at least half of the lesion surrounded by brain parenchyma; distinct from potential mimics such as iron or calcium deposits; vessel flow void, and clinical history excluding traumatic diffuse axonal injury (18). Critical hemosiderosis was defined as linear low signals of the cerebral cortex on gradient-echo (GRE)-weighted or susceptibility-weighted images (SWI).

We conducted our retrospective study in compliance with the Declaration of Helsinki. Verbal consent was obtained from the patients.

RESULTS
There were five patients with cSAH; 4 were women. The mean age was 67.4±19.4 (range 30-83) years. One patient was younger than 40 years. Four patients were older than 65 years. The demographic, clinical, and imaging features and etiologic diagnoses of the patients are presented in the Table 1. Laboratory tests for hypercoagulability and vasculitis were normal in all patients.

The most common clinical presentation was focal neurologic signs, followed by acute headache and seizures. Four patients presented with focal neurologic symptoms including hemiparesis/quadriparesis, hemihypoesthesia, and aphasia. Two patients had acute headaches, but only one (case 2) experienced a thunderclap headache. Seizure was present in only one patient (case 4). NIHSS scores were consistent with minor neurologic deficits in four patients.

The diagnosis of cSAH was confirmed with cranial CT and/or MRI in all patients. Four of the five patients were evaluated with at least one of the following vascular imaging studies, including CTA, MRA, MRV, CTV, and DSA. Detailed investigations revealed different causes of cSAH as follows: amyloid angiopathy (n=4) and CVT (n=1). The patient with etiologic diagnoses of CVT was younger than 40 years, and the other patients with amyloid angiopathy were older than 65 years. The haemorrhagic lesion was bilateral in two cases with CVT and amyloid angiopathy. The outcome was favourable in four patients; one patient (case 4), who presented with a higher NIHSS score, died.

Table 1. The demographic, clinical, and imaging features, and etiologic diagnoses of the patients

<table>
<thead>
<tr>
<th>Case No</th>
<th>Age (year)/sex</th>
<th>Clinical symptoms</th>
<th>NIHSSS</th>
<th>Location of SAH on CT on MRI</th>
<th>Radiologic findings</th>
<th>Diagnostic examination</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>67/F</td>
<td>Hemihypoesthesia and hemiparesis in left side</td>
<td>4</td>
<td>Right central sulcus</td>
<td>-cSAH -Resolution after 6 months and superficial hemosiderosis</td>
<td>Cranial CT, MRI, CTA, MRV, DSA</td>
<td>Probable amyloid angiopathy</td>
</tr>
<tr>
<td>2</td>
<td>76/F</td>
<td>Headache (thunderclap)</td>
<td>0</td>
<td>Right parietooccipital sulcus</td>
<td>-cSAH -DSA: normal -SWI: no microhaemorrhagic foci</td>
<td>Cranial CT, MRI, DSA</td>
<td>Amyloid angiopathy</td>
</tr>
<tr>
<td>3</td>
<td>30/F</td>
<td>Headache, hemiparesis in the left side, seizure</td>
<td>2</td>
<td>Right central, postcentral, left central, precentral sulcus</td>
<td>-cSAH -Hyperdense Trolard veins at CT -Hyperdense focus at the SSS junction with the Trolard veins</td>
<td>Cranial CT, MRI, MRV, DSA</td>
<td>Cerebral venous thrombosis</td>
</tr>
<tr>
<td>4</td>
<td>83/F</td>
<td>Sensorimotor aphasia, quadriparesis</td>
<td>13</td>
<td>Right post central, precentral, superior frontal sulcus, left lateral sulcus</td>
<td>-cSAH -Microhaemorrhagic foci at the cerebral and cerebellar hemispheres</td>
<td>MRI</td>
<td>Amyloid angiopathy</td>
</tr>
<tr>
<td>5</td>
<td>81/M</td>
<td>Hemihypoesthesia in the left side</td>
<td>2</td>
<td>Right central, precentral and postcentral sulci</td>
<td>-cSAH</td>
<td>Cranial CT, CTA, MRI, MRV</td>
<td>Amyloid Angiopathy</td>
</tr>
</tbody>
</table>

DSA: digital subtraction angiography; CT: computed tomography; CTA: computed tomography angiography; MRI: magnetic resonance imaging; MRV: magnetic resonance venography; SSS: superior sagittal sinus; cSAH: convexity subarachnoid haemorrhage; SWI: susceptibility-weighted imaging
Our first case was a 67-year-old female who presented with acute hemihypoesthesia and hemiparesis on the left side. She had cSAH at the right central sulcus on initial cranial CT and MRI examinations (Figure 1a). The MRI examination six months later showed that cSAH was resolved and there was hypointensity on T2-weighted and SWI at the superficial cortical layers along the central sulcus corresponding to cortical hemosiderosis (Figure 1b). MRV, CTA, and DSA were normal in this patient.

Our second case was a 76-year-old female who presented with a thunderclap headache. Cranial CT and MRI examinations revealed cSAH at the right parieto-occipital sulcus (Figure 2). DSA of the cranial vessels was normal. There were no microhaemorrhagic foci on SWI.

**Figure 1.** a, b. Initial (a) and 6 months later (b) axial SWI images show subarachnoid hemorrhage at the right central sulcus (a) resolved and there was cortical hemosiderosis (b) SWI: susceptibility-weighted imaging

**Figure 2.** Sagittal SWI image shows subarachnoid hemorrhage at the right parietooccipital sulcus SWI: susceptibility-weighted imaging

**Figure 3.** Axial CT show hyperdensity of both Trolard veins (cord sign) CT: computed tomography
Our third case was a 30-year-old female who presented with acute headache, hemiparesis, and generalized tonic-clonic seizure. On cranial CT, both Trolard veins were hyperdense (cord sign) and there was a hyperdense focus at the superior sagital sinus (SSS) at the junction with the Trolard veins (Figure 3). In the initial CT examination, cSAH had not been observed. Fluid attenuated inversion recovery (FLAIR) images performed after four hours showed bilateral high intensity in the right central, right post-central, left central, and left precentral sulci at the cerebral convexity (Figure 4). Both Trolard veins were hypointense (cord sign) on GRE images (Figure 5). Time-of-flight MRV examination revealed non-visualisation of bilateral Trolard veins and focal discontinuity of SSS at the draining point of bilateral Trolard veins (Figure 6). Digital subtraction angiography excluded the possibility of aneurysm and showed non-opacification of bilateral Trolard veins.

Our fourth case was an 83-year-old female who presented with acute sensorimotor aphasia and quadriparesis. Magnetic resonance imaging showed cSAH at right post-central, precentral, superior frontal sulci, and left lateral sulci (Figure 7a, b). She had microhaemorrhagic foci at the cerebral and cerebellar hemispheres with posterior cortical and cerebellar dominance (Figure 8).

Figure 4. Axial FLAIR image show bilateral high intensity in the right central, right post-central left central, left precentral sulci at the cerebral convexity
FLAIR: fluid attenuated inversion recovery

Figure 5. Axial GRE image show hypointensity of Both Trolard veins (cord sign)
GRE: gradient-echo

Figure 6. Time-of-flight (TOF) MRV examination revealed non-visualisation of bilateral Trolard veins and focal discontinuity of SSS at the draining point of bilateral Trolard veins
MRV: magnetic resonance venography; SSS: superior sagittal sinuse
Our fifth case was an 81-year-old male who presented with hemihypoesthesia on the left side. He had cSAH at the right central, precentral, and post-central sulci on cranial CT and MRI (Figure 9). Magnetic resonance venography, and CTA were unremarkable in this patient.

**Figure 7. a, b.** Axial GRE MR image (a) show subarachnoid hemorrhage at right superior frontal sulcus and post-central sulcus. Sagital T1-weighted MR image (b) show subarachnoid hemorrhage at right pre-central and post-central sulci.
GRE: gradient-echo; MR: magnetic resonance

**Figure 8.** Axial GRE MR image show multiple microhemorrhagic foci at the cerebellar hemispheres.
GRE: gradient-echo; MR: magnetic resonance

**Figure 9.** Axial GRE MR image show cSAH at the right central sulcus.
GRE: gradient-echo; MR: magnetic resonance; cSAH: convexity subarachnoid haemorrhage.
DISCUSSION

Although cranial CT is the first-line examination in patients who are suspected of having SAH, cSAH can also be seen on MR images. FLAIR imaging is more sensitive than CT for the detection of both acute and subacute SAH (19). Anatomic distribution of hemorrhage affects the diagnostic value of MRI techniques (19). FLAIR is sensitive for superficial/convexity SAH; however, SWI is better for visualisation of centrally-located haemorrhages (19). Cranial CT was diagnostic in four out of five patients who underwent CT in this series, mainly because they were admitted in the acute stage of the disease. In one patient (case 3), CT showed hyperdense foci suggestive of cerebral sinus thrombosis and displayed the underlying mechanism of cSAH. Convexal SAH was determined with FLAIR images in this patient. Thus, a negative CT examination does not exclude cSAH and should be always followed by FLAIR and SWI sequences of MRI, especially in the patients who are admitted with typical symptoms of cSAH. The contribution of MRI to etiologic diagnosis was most remarkable in the patients with CAA.

The central sulcus is usually involved in cSAH (20). Patients with SAH at the central sulcus may be symptomatic because of the highly functional status of the central gyrus (20). Two of our patients had unilateral and one patient had bilateral cSAH at the central sulcus, and all patients had focal neurologic signs such as hemiparesis, hemihypoesthesia and/or aphasia.

Kumar suggested two main etiologies for cSAH: CAA in patients older than 60 years and RCVS in the younger group (21). The older patients (aged over 60 years) in our case group had CAA. However, the younger patient had CVT. In our opinion, for younger patients, CVT as well as RCVS should always be kept in mind as a possibility.

Cerebral venous thrombosis is responsible for 1-2% of strokes in young adults (13). For rapid treatment, radiologic diagnosis is very important to assess patients’ clinical conditions. Patients with CVT are relatively younger (19). There is female dominance. Our patient with CVT was also young and female. Hypercoagulable states, pregnancy, postpartum period, oral contraceptive use, history of recent surgery, personal or family history of deep venous thrombosis, smoking, rheumatoid arthritis, antiphospholipid syndrome, and systemic lupus erythematosus are some of the risk factors for venous thrombosis. Our patient was in the post-partum period. Consistent with the literature, our patient had cSAH located at the convexity of the brain (16). When reporting CT images of patients with consistent headache and acute neurologic symptoms, radiologists should look for hyperdense thrombosed veins on CT (due to increased attenuation of thrombotic material) and hypointensity on GRE images (due to the magnetic susceptibility of deoxyhemoglobin), which is called “cord sign”. In our case, cord sign was evident on the CT before the time period that cSAH could be seen on MRI.

Fluid attenuation inversion recovery or SWI images are more sensitive for detecting acute or subacute cSAH; patients with cord sign and without visible cSAH on a CT should undergo MRI to look for cSAH and parenchymal ischemic or hemorrhagic lesions due to CVT. MRV or CTV should be also performed in young patients with cSAH to exclude CVT. Convexal SAH is usually located just near the thrombosed veins, as it was in our case, but it should be kept in mind that it may also be present in remote areas of the thrombosed sinuses. A comprehensive diagnosis can be made with contrast-enhanced or non-enhanced MRV. One of the proposed pathophysiologic explanations of SAH that develops in patients with CVT is the extension of dural sinus thrombosis into the superficial veins, which causes localised venous hypertension of the valveless, thin cortical veins that eventually rupture into the subarachnoid space (14). This pathophysiologic explanation may be acceptable for our patient because there was focal discontinuity of the SSS at the draining point of bilateral Trolard veins (Figure 6). The other possible explanation was rupture of parenchymal haemorrhagic infarct into the subarachnoid space (14). Our patient had cSAH without parenchymal haemorrhage or infarction. The final explanation is the presence of local inflammatory response, which increases vascular permeability caused by CVT (13).

We reported cSAH in four patients aged over 65 years without a history of head trauma (cases 1, 2, 4, and 5). Convexal SAH without a history of head trauma in an elderly patient is suggestive of CAA (22). Amyloids may be deposited in cortical and/or leptomeningeal vessels. As a result, primary bleeding may occur in the cerebral cortex, and secondary bleeding may occur in the subarachnoid space as a consequence of rupture of the meningeal vessels (23).

One of our patients (case 4) was an 83-year-old female who had cSAH at the right post central, precentral, superior frontal sulcus, and left lateral sulcus. She also had microbleeds in GRE images with a posterior cortical dominance, which we accepted as probable CAA according to the Boston criteria. The Boston criteria were developed to noninvasively designate “possible” and “probable” CAA for patients at or above the age of 55 years who have cortical, subcortical or lobar microhaemorrhages on imaging (24). In contrast to hypertensive microbleeds, which is also frequent in this age group, microhaemorrhages related to CAA spare basal ganglia, deep white matter, and the brain stem, and show posterior cortical dominance (22).

One of our patients (case 1) was a 67-year-old woman with cSAH upon initial examination and superficial hemosiderosis in the follow-up. It has been shown with animal models that superficial hemosiderosis is a late result of repeated bleeding in the subarachnoid space caused by deposition of hemosiderin in the subpial layer of the brain (25). We ob-
served this in our patient. At the follow-up examination after 6 months, cSAH was resolved, but superficial hemosiderosis at the superficial cortical layers along the sulcus (Figure 1b) had appeared, corresponding with focal cortical hemosiderosis. Two patients aged >70 years in our case series had cSAH, one in the right parietooccipital sulcus (case 2), and the other one in the right central, precentral and postcentral sulci (case 5). These three patients (cases 1, 2, and 5) had no cortical, subcortical or lobar haemorrhages, but a few studies have found that cSAH or superficial siderosis as a result of cSAH can be the only MRI finding in patients with histologically proven CAA (23, 26). It is now accepted that cSAH can be an early sign of CAA and progress to lobar hematoma (27).

When evaluating older patients with cSAH and transient ischemic attack-like symptoms, taking CAA into account has a great clinical impact on patient management, especially considering the risks of haemorrhagic complications due to antiplatelet and anticoagulation therapy (28). Therefore, elderly patients with cSAH should be investigated for other findings of CAA, such as lobar, cortical, and subcortical haemorrhage, microbleeds, and superficial siderosis, which can be seen with MRI, especially on GRE and SWIs.

Our study has some limitations such as the small sample size and especially the retrospective design.

The outcome of cSAH depends on the underlying etiology, and poor outcomes should be expected (27). In young patients, CVT or RCVS, which can be assessed with venography or angiography examinations, should be considered in the differential diagnosis of cSAH without head trauma. In older patients, radiologists should look for other signs of CAA, such as cortical or subcortical microbleeds and superficial hemosiderosis.

**Ethics Committee Approval:** Authors declared that the research was conducted according to the principles of the World Medical Association Declaration of Helsinki “Ethical Principles for Medical Research Involving Human Subjects”, (amended in October 2013).

**Informed Consent:** Verbal informed consent was obtained from patients who participated in this study.

**Peer-review:** Externally peer-reviewed.


**Conflict of Interest:** The authors have no conflicts of interest to declare.

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**REFERENCES**


28. Linn J. Central sulcus focal subarachnoid hemorrhage in the elderly: cerebral amyloid angiopathy is the most frequent cause. AJNR Am J Neuroradiol 2011; 32: E161. [CrossRef]