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Embolic stroke: a rare but probably real cause of aneurysmal-like subarachnoid hemorrhage

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Abstract

**Background:** Ischemic stroke is being increasingly recognized as a possible cause of spontaneous isolated convexity subarachnoid hemorrhage (SAH). However, it is a much less established etiology of cisternal, aneurysmal-like SAH. Only 3 case reports of concomitant cisternal SAH and perforator infarcts exist in the literature, raising the possibility of perforating artery rupture as a potential mechanism. In contrast, embolic stroke is not recognized as an etiology of aneurysmal-like SAH.

**Case Description:** We present 2 patients with embolic cerebral infarctions mimicking intracranial aneurysm rupture, in whom diagnosis was confirmed by DWI MRI after cerebral angiography failed to reveal an underlying vascular lesion. Extracranial atherosclerosis was identified as the source of emboli in each case. One patient was started on antiplatelet therapy, while the other underwent surgical revascularization. Both patients had a favorable hospital course, with no recurrent hemorrhage or ischemia.

**Conclusions:** Based on these observations, we propose that embolic stroke should be included in the differential diagnosis of angiogram-negative SAH and that brain MRI and vascular imaging of the neck should be part of the routine workup of this relatively common entity.
Introduction

Nearly 15% of all cases of spontaneous subarachnoid hemorrhage (SAH) are non-aneurysmal, and, in a third of those cases, another vascular, tumoral, or systemic etiology is ultimately identified.\(^1\) Recently, ischemic stroke has been recognized as a possible cause of spontaneous isolated convexity SAH.\(^2\)\(^7\) However, it remains a much less established etiology of cisternal SAH and is seldom considered in the differential diagnosis of intracranial aneurysm rupture. Only 3 previous case reports of concomitant cisternal SAH and perforator infarcts exist in the literature, which have been postulated to result from rupture of small perforating arteries.\(^8\)\(^9\) In contrast, embolic stroke is not a recognized etiology of aneurysmal-like SAH. We report 2 unique cases of embolic cerebral infarction mimicking intracranial aneurysm rupture.
Case Reports

Patient 1

A 64-year old woman, hypertensive and heavy smoker, presented with a sudden-onset worst headache of her life, but no other accompanying neurological symptoms. Neurological exam was unremarkable. Head CT revealed diffuse and symmetric cisternal SAH (figure 1A). She was classified WFNS grade 1, Fischer grade 3. CTA of the head was unremarkable. CTA of the neck demonstrated high-grade stenosis of the proximal right subclavian artery, secondary to a calcified atherosclerotic plaque distal to its origin from the brachiocephalic trunk (figure 1B). In addition, a calcified ulcerated plaque of the right internal carotid artery (ICA) origin was identified (figure 1C). Six-vessel cerebral angiography was performed the following day and repeated 1 week later, but showed no underlying vascular lesions (figure 1D-F). However, a subclavian steal phenomenon was observed on the right side, resulting in complete reversal of flow in the right vertebral artery (figures 1G,H). MRI of the brain performed on SAH day 10 showed no evidence of underlying vascular or tumoral lesions, but identified multiple small foci of diffusion restriction on DWI, within the distal right middle cerebral artery territory and bilateral cingulate gyri (figures 1I-P). Stroke Neurology consultation was obtained, and an echocardiogram revealed no intracardiac embolic sources. It was concluded that the patient has had right ICA territory infarcts, secondary to arterio-arterial emboli arising from the right ICA plaque. She was started on oral aspirin and had an uneventful hospital course, with no evidence of recurrent hemorrhage or delayed cerebral ischemia. She remained neurologically intact and was discharged home on SAH day 14.
Patient 2

A 66-year old woman, hypertensive and smoker, presented with acute-onset right periorbital pain, photophobia, and an impression of seeing "floaters" in the right eye. Neurological exam was unremarkable. Head CT revealed focal SAH in the anterior interhemispheric fissure, concerning for an anterior cerebral artery aneurysm (figures 2A,B). She was classified WFNS grade 1, Fischer grade 3. Four-vessel cerebral angiography was performed the following day, revealing high-grade (90%) focal atherosclerotic stenosis of the proximal left ICA, just distal to its origin (figure 2C). However, no intracranial vascular abnormalities were identified (figures 2D-F). The left ICA was not selectively catheterized, and only common carotid arterial injections were performed. Six hours later, the patient exhibited sudden-onset aphasia, after turning her head to the side. Brain MRI was urgently performed, revealing multiple foci of restricted diffusion in the left middle cerebral artery territory on DWI, suggestive of embolic infarcts originating from the left ICA plaque (figures 2G-L). The patient was started on oral aspirin and an emergent left carotid endarterectomy was performed the following morning. The patient had an uneventful post-operative course with resolution of her symptoms. She was discharged home 2 days later, neurologically intact. An echocardiogram was performed prior to discharge, and was normal.
Discussion

In 85% of cases, spontaneous SAH results from rupture of an intracranial aneurysm. Another 5% are caused by less common entities, including cerebral vascular malformations, intracranial dissections, cerebral venous thrombosis, moyamoya disease, cerebral vasculitides, amyloid angiopathy, tumors, vascular lesions in the spinal cord, coagulopathy, and central nervous system stimulant (cocaine, amphetamines, etc) abuse. Finally, the etiology of roughly 10% of SAH cases remains unknown, despite a complete workup. Recently, cerebral ischemia secondary to extracranial or intracranial atherosclerotic disease has been identified as a relatively common cause of spontaneous isolated convexity SAH. It has been postulated that cerebral ischemia leads to dilation of compensatory leptomeningeal collateral vessels and that rupture of these fragile pial vessels may lead to convexity SAH, similarly to the mechanism proposed for moyamoya disease. In contrast, cerebral ischemia is a much less established etiology for aneurysmal-like (i.e. cisternal) SAH. In fact, only 3 previous case reports described the occurrence of cisternal SAH concomitantly with a perforator stroke in the brainstem or basal ganglia. In both papers, SAH was thought to result from the rupture of a small perforating artery, hence the concomitant infarction. In fact, rupture of a small pial artery as a cause of SAH had been previously demonstrated in an autopsy report. Conversely, embolic stroke is not recognized as a potential etiology of aneurysmal-like SAH. Therefore, this small case series is the first study to suggest a causal relationship between embolic stroke and cisternal SAH. The possibility of ischemic stroke should be kept in mind in older patients with multiple cardiovascular risk factors, particularly heavy smokers and those with known atherosclerotic disease, presenting with angiogram-negative cisternal SAH. In fact, both patients in this report
were in their 60’s, were heavy smokers, and had hypertension. We propose a similar mechanism to that of hemorrhagic transformation following embolic infarction, namely ischemic vessel wall damage leading to blood extravasation once spontaneous recanalization occurs. If emboli travel distally in the cerebral vasculature, resulting in occlusion of superficial pial arteries, then spontaneous recanalization could lead to isolated cisternal SAH, without associated intraparenchymal hemorrhage.

The major limitation of this report is that, in both patients, brain MRI was performed after cerebral catheter angiography. Thus, it could be argued that the observed areas of restricted diffusion on DWI MRI could correspond to embolic events sustained during angiography, rather than present at admission and causally related to SAH. In fact, the rate of DWI MRI changes following cerebral angiography has been reported to be as high as 11%. While this scenario cannot be completely ruled out in patient 1, it would be extremely unlikely in patient 2. In fact, MRI in this patient was triggered by symptoms of cerebral ischemia, which started abruptly 6 hours following completion of the angiogram. Thus, the delayed onset of cerebral ischemia argues against a procedure-related event. Moreover, the left ICA was not selectively catheterized during the procedure. Therefore, the risk of intraprocedural embolization from the left ICA plaque is likely to be very low.
Conclusions

Embolic stroke is a rare but probably real cause of cisternal SAH, and should be included in the differential diagnosis of angiogram-negative SAH, particularly in older patients, those with multiple cardiovascular risk factors, and those with known atherosclerotic disease. In this setting, brain MRI and vascular imaging of the neck can provide invaluable information, and should thus be incorporated in the routine workup of patients with angiogram-negative SAH. Specifically, our findings suggest that initial CTA workup in SAH patients should probably include the neck as well as the head. Likewise, when cerebral catheter angiography is performed, routine imaging of the common carotid bifurcations is advisable. Furthermore, when vascular imaging fails to reveal an underlying lesion, an MRI of the brain with DWI sequence should be performed to rule out other less common etiologies, particularly ischemic stroke.
References


Figure Legends

Figure 1 – Patient 1: A, Head CT demonstrates a diffuse, symmetric, aneurysmal pattern of SAH. B and C, CTA of the neck reveals a calcified atherosclerotic plaque, causing high-grade stenosis of the proximal right subclavian artery (B, arrow), and a calcified ulcerated plaque at the origin of the right ICA (C). D-H, Cerebral angiography shows no evidence of underlying aneurysms or vascular malformations (D-F), but presence of a right subclavian steal phenomenon, with reversal of flow in the right vertebral artery (G and H). Two Lower Rows, DWI MRI of the brain reveals multiple small foci of diffusion restriction in the distal right MCA territory and bilateral cingulate gyri.

Figure 2 – Patient 2: A and B, Head CT shows focal SAH in the anterior interhemispheric fissure, concerning for an anterior cerebral artery aneurysm. C-F, Cerebral angiography demonstrates focal high-grade atherosclerotic stenosis of the proximal left ICA (2C), but no evidence of intracranial aneurysms or vascular malformations (2D-F). Two Lower Rows, DWI MRI of the brain reveals multiple foci of restricted diffusion in the left MCA territory.
Abbreviations

SAH  Subarachnoid Hemorrhage
CT   Computerized Tomography
CTA  Computerized Tomography Angiography
DWI  Diffusion-weighted Imaging
ICA  Internal Carotid Artery
MCA  Middle Cerebral Artery
MRI  Magnetic resonance Imaging
WFNS World Federation of Neurosurgical Societies
Highlights

. Embolic stroke can mimic intracranial aneurysm rupture

. Proposed mechanism: extravasation from superficial pial artery after recanalization

. Suspect in older patients with known atherosclerotic disease or risk factors

. Brain MRI and vascular imaging of neck are essential after angiogram-negative SAH