

Case Report

Transient ischemic attack as initial presentation of convexal subarachnoid hemorrhage: a case report

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Abstract: A 51-year-old man presented a 20-minute episode of slurred speech and numbness in the left arm, and then recovered entirely. The past medical history indicated a similar attack two weeks ago which also recovered after 20 minutes. The neurological examination was otherwise unremarkable. A magnetic resonance imaging scan indicated that there had high signal of T1 and low signal of T2 in the right precentral sulcus. And there had some high signal in fluid attenuated inversion recovery (FLAIR). The susceptibility-weighted imaging (SWI) showed that magnetic sensitive signal in frontal and parietal sulcus. A computed tomography scan revealed a high-density lesion restricted to the right precentral sulcus. The diagnosis was convexal subarachnoid hemorrhage. The patient was discharged from hospital with the high-density lesion in right precentral sulcus disappeared in a repeated CT scan.

Keywords: Transient ischemic attack, convexal subarachnoid hemorrhage, susceptibility weighted imaging

Introduction

Nontraumatic convexal subarachnoid hemorrhage (cSAH) is a rare and not-yet well-characterized condition. According to a single-institution study, its prevalence among in-hospital SAH patients is as few as 6.3% [1]. cSAH may exhibit itself with a variety of distinct clinical presentations such as headache, somnolence and confusion, transient neurological impairments or seizures [1, 2]. Consequently, various symptoms stem from distinct etiologies such as cerebral amyloid angiopathy (CAA), reversible cerebral vasoconstriction syndrome (RCVS) and cerebral venous thrombosis may result in misdiagnosis in different clinical settings [3, 4]. Here we report a case of cSAH present with transient ischemic attack (TIA)-like symptoms, and propose a cerebrovascular disease spectrum for transient focal neurologic episodes (TFNEs).

Case description

A 51-year-old man presented a 20-minute episode of slurred speech and numbness in the

left arm. The patient recovered entirely from the attack after twenty minutes. The past medical history indicated a cerebrovascular event resulted from subarachnoid hemorrhage in the absence of aneurysm, verified by digital subtraction angiography (DSA). The patient has had clinical events due to "cerebral infarction" three times, which caused the sequela of paralysis on left limbs. The patient had hypertension for 30 years with oral medication amlodipine, and diabetes for 1 year without medical care. The neurological examination was otherwise unremarkable except for paresis on left extremities. The laboratory examination, electrocardiogram, electroencephalogram, Doppler echocardiography, transcranial Doppler, ultrasound of digestive system and urinary system was all unremarkable. A computed tomography (CT) scan at presentation revealed a high-density lesion restricted to the right precentral sulcus (**Figure 1A**), while disappeared on repeated CT scan performed 19 days later (**Figure 1D**). A magnetic resonance imaging (MRI) scan, including T1-weighted, T2-weighted, fluid attenuated inversion recovery (FLAIR) and susceptibility-weight-

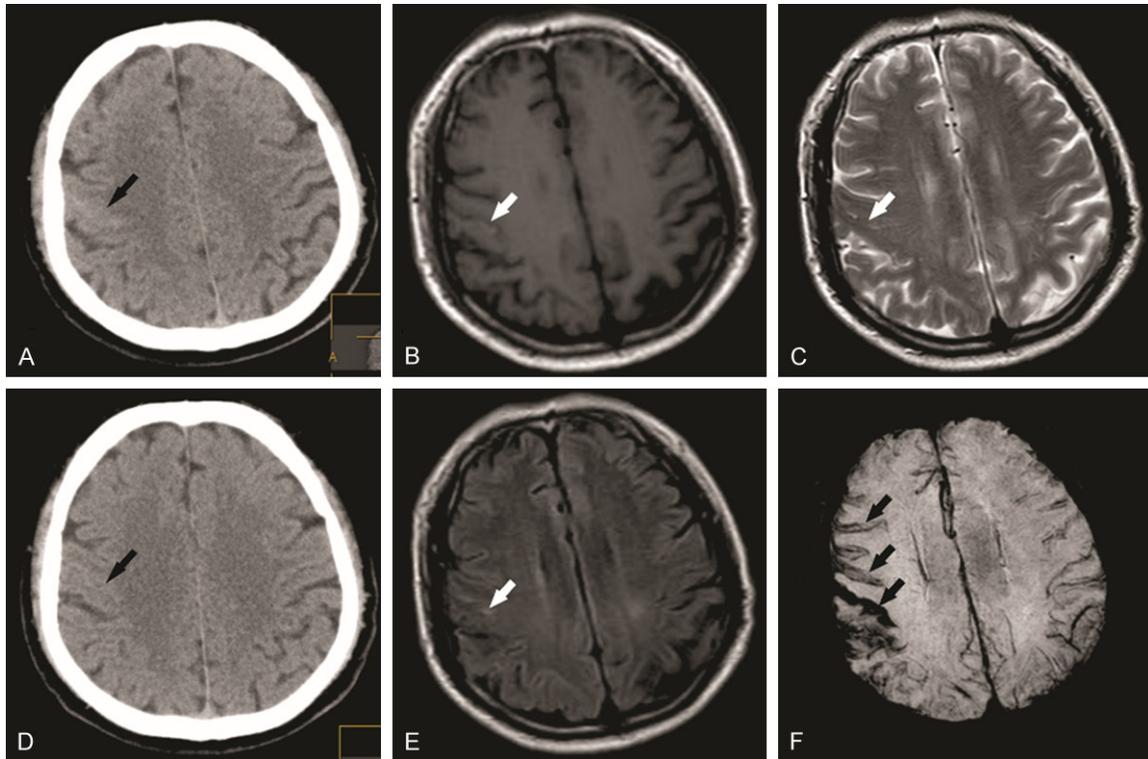


Figure 1. Initial CT scan shows subtle hyperdense signal in the precentral sulcus, which resolved in the follow-up scan (black arrows; A, D). MRI shows hyperintense signal in T1-weighted and FLAIR (white arrows; B, E), and hypointense signal in T2-weighted sequence (white arrow; C). Susceptibility-weighted imaging shows apparent magnetic susceptibility effect (black arrows; F).

ed imaging (SWI) (**Figure 1B, 1C, 1E and 1F**), confirmed convexal subarachnoid hemorrhage in multiple anterior temporal sulcus. The patient was discharged from hospital 14 days later, with the high-density lesion in right precentral sulcus disappeared in a repeated CT scan.

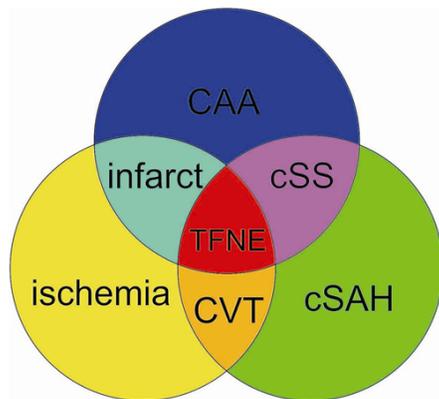
Discussion

Non-traumatic cSAH is an atypical manifestation of subarachnoid hemorrhage (SAH), in which the bleeding is restricted to sulci of the brain without any involvement of the adjacent cerebral parenchyma, ipsilateral gyri, fissures, cisterns and ventricles. cSAH is also called cortical SAH or sulcal SAH [1]. Kumar et al. analyzed 389 cases of spontaneous SAH, only 29 cases of which meet the diagnostic criteria for non-traumatic cSAH [1].

The clinical manifestation of cSAH at the onset varies significantly. A retrospective study conducted by Kumar et al. revealed that 62% of the cases presented with headache, whereas the

majority (54%) of patients over 60-year-old were with TFNEs [1]. Moreover, 69% of the patients over 60-years-old showed CAA through imaging studies, such as leukoaraiosis, cerebral microbleeds and deposits of hemosiderin. Another observational study included 24 cases confirmed that 45% of over 60-years-old patients presented with transient ischemic attack (TIA)-like symptoms, while only 42% cases present with headache were observed in this single-center study [5]. The discrepancy between the two studies may be attributed to distinct age compositions, i.e., the retrospective study contained 55% of patients whose ages were under 60 [1], while only 17% of the participants in the observational study were in such population [5]. Other possible etiologies of cSAH include posterior reversible encephalopathy syndrome, cerebral venous (sinus) thrombosis, hypertensive cerebral small vessel disease, idiopathic thrombocytopenia purpura, cavernous hemangiomas, cerebral vasculitis, brain abscesses and the use of anticoagulants [4, 6-9].

Convexal subarachnoid hemorrhage present with TIA



TFNE: transient focal neurological episodes
CAA: cerebral amyloid angiopathy
cSS: cortical superficial siderosis
cSAH: convexal subarachnoid hemorrhage
CVT: cortical venous thrombosis

Figure 2. Spectrum of diseases present with transient focal neurologic episodes (TFNEs).

The differentiation between cSAH and CAA is noteworthy when they initially present with TFNEs. A multicenter cohort study included 172 patients with CAA showed that 14.5% of patients with CAA manifested with TFNE initially [10]. Meanwhile, 54% of these patients demonstrated cSAH or deposits of hemosiderin, which are most common exhibitions throughout neuroimaging modalities in this study. CAA patients present with TFNE are more likely to exhibit cSAH or deposits of hemosiderin in relative to those without TFNE. The relevance between TFNE and CAA [11-13], as well as the overlap between the two entities, was confirmed by a number of studies [14, 15]. The patient in this case did not bear history on cerebral parenchymal hemorrhage or microbleeds in neuroimaging, therefore it cannot be diagnosed as CAA in terms of Boston criteria.

Multi-modality imaging of MRI is instrumental for differentiating TIA, cSAH and CAA. About one third TIA patients show restricted diffusion in diffusion weight imaging (DWI) [16]. Thanks to the advances of modern medical imaging, the contemporary concept of TIA is transforming from the duration of clinical neurological dysfunctions to histopathological changes, which may be partially reflected based on imaging findings [17, 18]. In fact, the new definition of TIA proposed by American Stroke Association/American Heart Association has already recommended imaging examinations, particularly

DWI to identify the necrosis of brain tissue, emphasizing TIA's definition in the absence of tissue necrosis [19]. Moreover, t2-weighted gradient-echo sequence or SWI are usually utilized to detect subacute, chronic cerebral bleeding or microbleeding. In this regard, recent evidence suggests that SWI may be more sensitive than gradient-echo T2*WI [20-22].

We analyzed the possible primary etiology of the patient in this case. Arteriovenous malformation, Moyamoya disease, aortic dissection, vasculitis, intracranial tumors and brain abscess were excluded according to the clinical features, laboratory examinations and imaging findings including MRI, SWI, magnetic resonance angiography, magnetic resonance venography (MRV). CAA was also excluded as stated above. The differential diagnosis of isolated cortical venous thrombosis (iCVT) was initially considered. The patient did not have risk factors of hypercoagulability resulted from dehydration, medication or fever, and common symptoms of iCVT such as headache, nausea, vomiting and convulsion. Meanwhile, D-dimer was normal after repeat examinations. Furthermore, the sulcal bleeding of cSAH is often with rough boundary and heterogeneous signal in irregular shape, which is coined as "triangle sign" in SWI [23]. On the contrary, signals of sulcal veins are usually smooth and homogeneous in SWI (Figure 1). The MRI and MRV of the patient showed there was no intravenous thromboembolism, with further evidenced by repeated CT scans displaying the sulcal signal changed from high-density to iso-density.

TFNE due to cerebrovascular etiology is usually diagnosed as TIA, while ischemic cerebrovascular spectrum may include cerebral infarction, TIA and venous thrombosis etc. On the other hand, cSAH may be secondary to cerebral venous sinus thrombosis [7, 24] or iCVT [25] which may resemble cSAH in neuroimaging findings [23]. In addition, CAA patients present with TFNE are more likely to exhibit cSAH or deposits of hemosiderin compared to those without. Recurrent cSAH may herald relapsing lobar hemorrhage, indicative of a CAA-derived etiology [26].

In conclusion, TFNE due to various etiologies may exhibit similar clinical manifestations, but represents a number of distinct diseases (Figure 2). Classical TIA due to ischemic causes

Convexal subarachnoid hemorrhage present with TIA

accounts for only a part of the spectrum. Prudent considerations combining medical history, symptoms and other examinations should be carried out when uncovering etiologies behind TFNEs.

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Disclosure of conflict of interest

None.

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