Serial Neuroimaging of a Patient with Minor Stroke due to Isolated Cortical Vein Thrombosis and Convexal Subarachnoid Hemorrhage

Pei-Ya Chen [1], Kun-Eng Lim [2,3], Shinn-Kuang Lin [1,3]

Abstract-

Purpose: Convexal subarachnoid hemorrhage (cSAH) comprises less than 5% of cases of nontraumatic SAH and frequently presents as a focal and transient neurological deficits that mimics transient ischemic attack (TIA). Isolated cortical vein thrombosis (ICVT) is rare and accounts for only 6.3% of cerebral venous thrombosis. We present a case of minor stroke due to cSAH secondary to ICVT, and alos put emphasis on the chronological change of those serial imagings.

Case Report: An 87-year-old man presented with episodes of numbness and dropping of his left arm, which had lasted for three days. Brain computed tomography disclosed a cSAH in the right fronto-parietal region. Brain magnetic resonance (MR) study showed a cSAH in the right fronto-parietal sulci. Focal swelling of the right frontal cortex with an intraluminal filling defect in the right cortical vein and venous congestion were observed using post-contrast T1-weighted images, suggesting partial thrombosis with recanalization of the cortical vein but a patent superior sagittal sinus. Diffuse linear superficial cortical hemosiderosis (SCH) was detected in the right anterior frontal cortex, right fronto-parietal cortex and left high frontal cortex. He spontaneously recovered from his minor neurological deficits within two weeks. A follow-up MR study three weeks later found a hyperintense cord sign indicating a cSAH in the right high central sulcus on fluid-attenuated inversion recovery and T2-weighted images. A further follow-up MR study two months later showed gradual shrinkage of the cSAH with persistent diffuse SCH.

Conclusion: This case report clearly showed chronological change of brain MRI and head CT findings. MR studies help in recognizing the occurrence of acute and chronic cSAHs, and ICVT.

Key Words: convexal subarachnoid hemorrhage, isolated cortical vein thrombosis, superficial cortical hemosiderosis, cerebral amyloid angiopathy

Acta Neurol Taiwan 2016;25:104-110

INTRODUCTION

Transient ischemic attack (TIA) is defined as a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without an acute infarction[(1)]. The differential diagnosis of TIA is

From the [1]Stroke Center and Department of Neurology, [2]Medical Imaging, Taipei Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Taiwan, [3]School of Medicine, Tzu Chi University, Hualien, Taiwan.

Received May 6, 2016. Revised June 14, 2016.
Accepted July 26, 2016.

Correspondence to: Shinn-Kuang Lin, MD. Department of Neurology, Taipei Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Taiwan. No. 289, Jian Guo Road, 231, Xindian District, New Taipei City, Taiwan.
E-mail: jy0428@totalbb.net.tw, sk2022@tzuchi.com.tw

challenging. Symptoms involving transient disturbance of motor or sensory functions are usually considered a TIA-mimic syndrome. Pathological conditions which can cause TIA-mimic syndrome include migraine, seizure, syncope, amyloid spells, and anxiety disorders[2]. Overall, 85% of patients with nontraumatic subarachnoid hemorrhages (SAH) result from a saccular aneurysm rupture, while 10% of them result from a benign perimesencephalic SAH[3]. Convexal SAH (cSAH) comprises less than 5% of nontraumatic SAH and frequently presents as a focal, transient neurological deficit that mimics TIA[3-5]. Most cSAH in patients under 60 years old results from reversible vasoconstriction syndrome whereas cerebral amyloid angiopathy is the most common cause in patients over 60[6]. Cerebral venous thrombosis represents < 1% of cerebral strokes[7,8]. Isolated cortical vein thrombosis (ICVT) accounts for only 6.3% of overall cerebral venous thrombosis[7]. Here we present a rare case of minor stroke due to cSAH secondary to ICVT.

CASE REPORT

An 87-year-old man without hypertension, diabetes, or any history of head injury presented with intermittent numbness and fluctuating weakness in his left arm for three days. There was no associated dizziness, headache, or slurred speech. On examination, he was alert with a normal blood pressure (140/64 mmHg), a normal body temperature (36.3˚C) and a regular heart beat (68 bpm). The neck was supple. A neurological examination did not find any cranial dysfunction or an abnormal deep tendon reflex except for a decrease in pin-prick sensation and the presenc of mild weakness (Medical Research Council 4/5) affecting his left arm. An emergency brain computed tomography (CT) scan found a focal cSAH in the right fronto-parietal region (Fig 1). After admission, further brain magnetic resonance (MR) imaging showed a cSAH in the right fronto-parietal brain sulci and brain surface (Fig 2). Focal swelling of the right frontal cortex with an intraluminal filling defect in the right cortical vein and venous congestion were observed in post-contrast T1-weighted images (T1WI), suggesting partial thrombosis with recanalization of the fronto-parietal cortical vein but a patent superior sagittal sinus. MR angiography did not show any abnormal arterial lesion. Diffuse linear superficial cortical hemosiderosis (SCH) was detected in the right anterior frontal cortex, right fronto-parietal cortex and left high frontal cortex. The results of laboratory examinations including fasting glucose, lipid profile, hemogram, prothrombin time, activated partial thromboplastin time, erythrocyte sedimentation rate, fibrinogen, and protein C and protein S were normal. Based on the relatively minor neurological deficit and the patient's age, we chose conservative observation of his cSAH and isolated cortical vein thrombosis (ICVT). The muscle power of his left arm had recovered completely one week later and the impairment in pin-prick sensation in his left arm had subsided two weeks later.

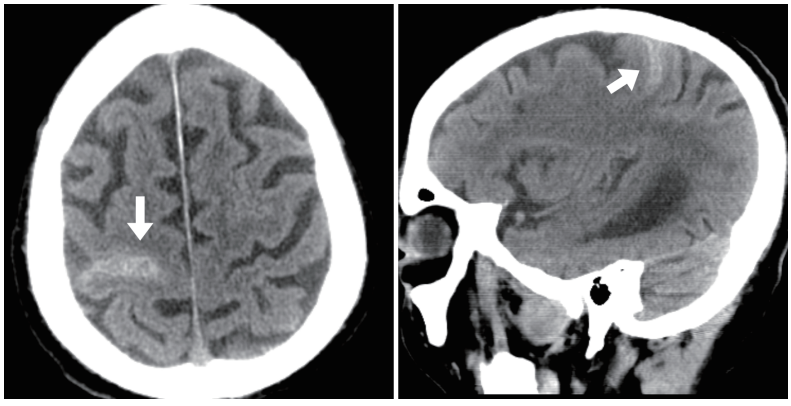

Figure 1. Brain computed tomography (CT) shows a focal SAH in the right fronto-parietal region (arrows).

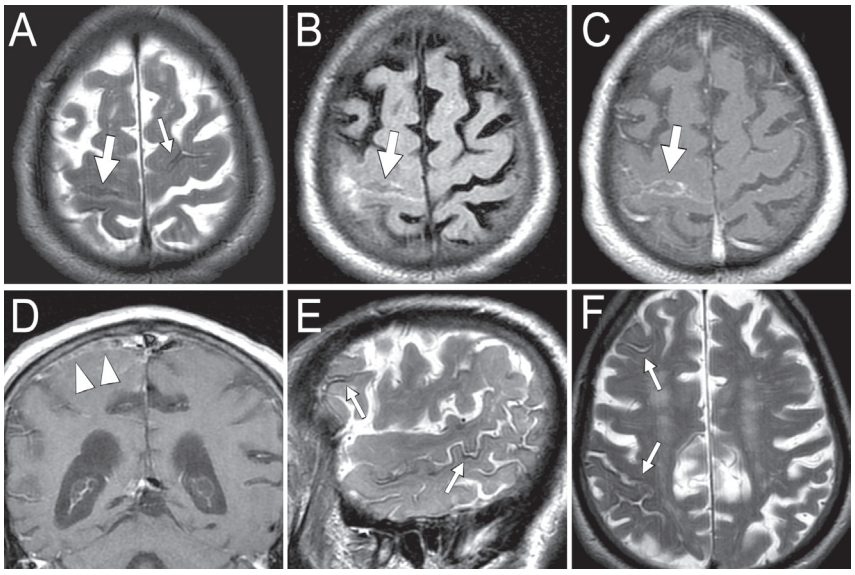

Figure 2. Brain magnetic resonance imaging (MRI) demonstrates isointense acute blood clot accumulation in the right central sulcus (large arrows) on T2-weighted images (T2WI) (A), fluid-attenuated inversion recovery (FLAIR) (B), and enhanced T1-weighted images (T1WI) (C). The coronal view on enhanced T1WI discloses focal swelling of the right frontal cortex with an intraluminal filling defect and venous congestion in the right cortical vein (arrowheads) with a patent superior sagittal sinus (D). Axial (A, F) and sagittal T2WI images (E) show diffuse linear superficial siderosis in the right anterior frontal cortex, right fronto-parietal cortex and left high frontal cortex (small arrows).

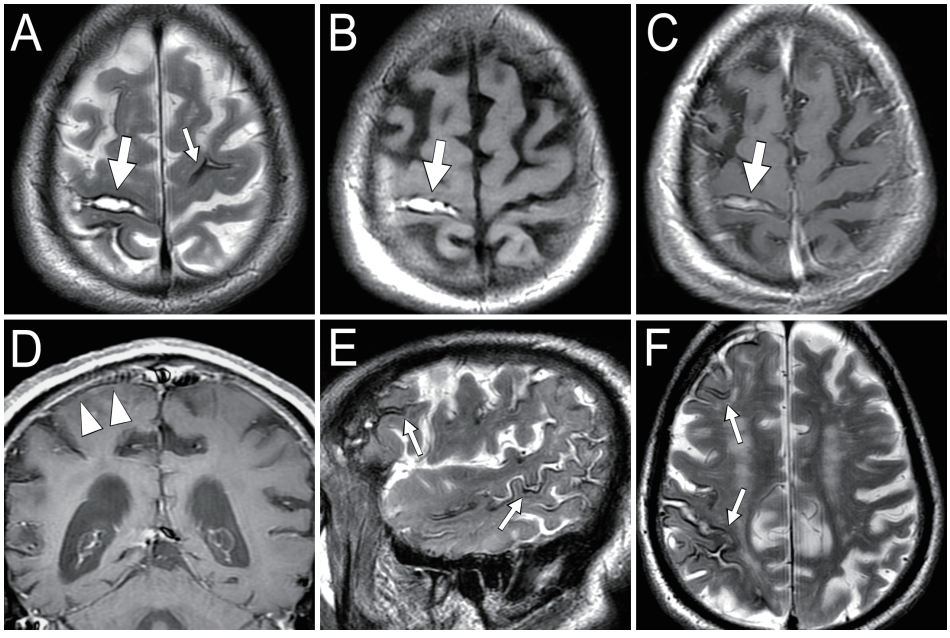

Figure 3. Follow-up brain MRI three weeks later shows a prominent hyperintense cord sign, indicating a subarachnoid hemorrhage (large arrows) in the right central sulcus on T2WI (A), FLAIR (B), and enhanced T1WI (C). The coronal view on enhanced T1WI demonstrates improvement in the intraluminal filling defect in the right cortical vein (arrowheads) and focal swelling of the right frontal cortex (D). Axial (A, F) and sagittal T2WI images (E) disclose persistent diffuse linear superficial siderosis in the right anterior frontal cortex, right fronto-parietal cortex and left high frontal cortex (small arrows).

A follow-up MR study three weeks later found a prominent hyperintense cord sign, indicating cSAH in the right high central sulcus on fluid-attenuated inversion recovery (FLAIR) and T2-weighted images (T2WI) but less swelling in the right high frontal cortex on coronal T1WI. Diffuse linear SCH was still present in same areas detected previously (Fig 3). A follow-up MR study two months after the initial CT showed no more edematous changes in the right high frontal cortex and gradual shrinkage of the cSAH in the right high central sulcus with persistent diffuse linear SCH. MR venography disclosed partial thrombosis affecting the inferior end of the superior sagittal sinus. No weakness or numbness in the left arm was observed at the six month follow-up.

DISCUSSION

This patient had ICVT presenting as cSAH with minor sensorimotor stroke. cSAH was found affecting the corresponding cerebral cortex contralateral to the symptomatic limb when a brain CT study was carried out. ICVT causing cSAH with adjacent brain swelling and concomitant discrete SCH, which is indicative of old cSAHs were disclosed by a MR study. No exact cause of the ICVT was found after laboratory studies. Although his minor neurological deficits recovered spontaneously, the follow-up MR studies showed persistent SCH.

Spontaneous nonaneurysmal cSAH is an uncommon type of cerebrovascular disease. It is characterized by blood collections in one or several adjacent sulci with the absence of blood at the base of the brain or elsewhere[9]. Unlike aneurysmal SAH, headache is not often observed. Most reported clinical presentations consist of one or recurrent brief attacks of sensory and/or motor dysfunction mimicking TIAs[5,9,10]. The mechanism could be a cortical spreading depression underlying migraine with aura triggered by the subarachnoid blood[3,11]. Although a mechanism involving focal epileptic seizure has been proposed, no epileptiform activity has been detected using electroencephalograms in the previous studies [12]. The etiologies of cSAH include cerebral amyloid angiopathy (CAA), reversible cerebral vasoconstriction syndrome, posterior reversible encephalopathy syndrome, arteriovenous malformations or fistulas, cerebral venous thrombosis, vasculitides, mycotic aneurysm, severe atherosclerotic carotid disease, and coagulation disorders [3,13]. According to a review by Kumar et al, the most probable cause of cSAH among patients older than 60 years is CAA, while reversible cerebral vasoconstriction syndrome is the most common etiology among patients younger than 60 years[6]. Cortical vein thrombosis is a rare cause of cSAH. In three recent large patient cohort studies of cSAH published by Kumar et al (29 patients) [6], Beitzke et al (24 patients) [9], and Renou et al (30 patients) [3], cortical venous thrombosis was observed in only two of the 83 patients.

cSAHs are mostly located in the central, precentral, or postcentral sulcus contralateral to the clinical symptoms [5]. The incidence of cSCHs may be underestimated because it often occurs without clinical symptoms in silent areas of the cortex. The diagnosis of cSAH is made by brain CT when there is a linear hyperdense cord sign along the cerebral convexity. In Ertl et al's review, initial unenhanced CT scans were able to detect cSAH in 95% of patients[4]. However, a small amount of blood in the subarachnoid space might be invisible on CT scans several days after hemorrhage. MR provides better sensitivity than CT when detecting cSAH, especially when FLAIR, gradient recalled echo T2 (GRE T2) or susceptibility-weighted (SWI) MR sequences are used. Extravasated fresh blood is generally isointense relative to cerebrospinal fluid (CSF) on T2WI. Nevertheless, because of serum protein, fresh blood sometimes can appear of high signal intensity which can be masked by CSF[14]). FLAIR sequences suppress the CSF signal and allow the signal from globin and its breakdown products to be seen[15]. Acute and subacute cSAH is best seen on a FLAIR sequence as a hyperintense signal. However, subarachnoid FLAIR hyperintensity can also be observed in meningitis, leptomeningeal metastasis, status epilepticus, and when intravenous iodinated or gadolinium contrast material is used[13]. GRE T2 is sensitive to hemosiderin and can detect the minimal changes in the magnetic field, and as a result this improves the detection rate small lesions, particularly microbleeds[16]. cSAH is displayed as a hypointense signal on GRE T2 sequences[3,4]. However, GRE images do not allow differentiation between acute hemorrhage and residual posthemorrhagic hemosiderin deposits[4].

ICVT is the thrombosis of one or more cerebral cortical veins without occlusion of the major dural venous

sinuses or deep cerebral veins[8]. ICVT is rare and has only been described in case reports and small patient series[8,17-20]. Common symptoms of ICVT are seizure, headache, motor weakness and sensory disturbance[20]. ICVT causing cSAH is extremely rare. In Kitamura et al's review, cSAH was observed in only 15.7% (8/51) of patients with ICVT[20]. The diagnosis of ICVT is difficult owing to the small diameter of the veins and anatomical variations. Brain CT of ICVT often demonstrates intraparenchymal hemorrhage or nonspecific focal edema [8]. Brain MR studies, particularly the SWI and GRE T2 sequences, are the most important methods for further documentation of ICVT. Direct signs of thrombi in veins and indirect signs of venous stasis or slow collateral flow are the main findings when MR study are carried out[7]. During the acute stage, a hyperintense cord sign or dot sign of a thrombus might be visible on T1WI (deoxyhemoglobin) but these are hypointense using T2WI, GRE T2, SWI and FLAIR. During the subacute stage, the thrombus becomes highly intense on both T1WI, T2WI, and FLAIR (methemoglobin), but hypointense on GRE T2 and SWI. The signal intensity in a chronic thrombus is isointense on T1WI and isointense or hyperintense on T2WI, but persistently hypointense on GRE T2 and SWI[7,17,21]. Swelling of adjacent gyral due to venous congestion may be visible on T2WI and FLAIR and might help to differentiate ICVT with cSAH from CAA with cSAH.

SCH is defined as linear residues of blood in the superficial layers of the cerebral cortex as opposed to punctuate subcortical microhemorrhages[22]. It is caused by hemorrhage into the subarachnoid space with hemosiderin deposition. It is best displayed by MR T2WI and appears as bilinear "tracklike" low intensity hemosiderin with a middle hyperintense normal-appearing CSF space [22,23]. CAA typically presents with lobar intraceberal macrohemorrhage or microbleeds. SCH due to occult cSAH has been reported to be a common finding in patients with CAA. Inclusion of SCH as a criterion has resulted in the diagnostic upgrading of patients with CAA without lowering the specificity of the Boston criteria [11,22,24]. The MR study in this case report showed bilateral asymptomatic SCH, similar to CAA. However, we could not find other common picture of microbleeds that indicated CAA based on the MR study. The acute cSAH was displayed as an isointense lesion in the right central sulcus on T2WI (Fig 2). The presence of cSAH reduces the direct sign of a hypointense thrombus in the cortical vein on T2WI. The diagnosis of ICVT was based on the initial MR study which showed an intraluminal filling defect and venous congestion in the right cortical vein on enhanced T1WI, together with adjacent gyral swelling.

Table1. Neuroimaging findings for convexal subarachnoid hemorrhage and isolated cortical vein thrombosis

	Blood in convexal subarachnoid hemorrhage			Thrombus in isolated cortical vein thrombosis		
	Acute	Subacute	Chronic	Acute*	Subacute	Chronic
CT	High density	Invisible	Invisible	Invisible or high density	Invisible	Invisible
MRI						
T1WI	Isointensity or hyperintensity	Isointensity or hyperintensity		Isointensity or hyperintense cord/ dot sign	Hyperintensity	Isointensity
T2WI	Isointensity or hyperintensity (masked by CSF)	Hyperintensity	SCH	Hypointensity	Hyperintensity	Isointensity or hyperintensity
FLAIR	Hyperintensity	Hyperintensity	Isointensity	Hypointensity	Hyperintensity	Isointensity or hyperintensity
GRE T2	Hypointensity	Hypointensity	SCH	Hypointensity	Hypointensity	Hypointensity
SWI	Hypointensity	Hypointensity		Hypointensity	Hypointensity	Hypointensity

CT, computed tomography; MRI, magnetic resonance imaging; T1WI, T1-weighted image; T2WI, T2-weighted image; FLAIR, fluid-attenuated inversion recovery; GRE T2, gradient recalled echo T2; SWI, susceptibility-weighted image; CSF, cerebrospinal fluid; SCH, superficial cortical hemosiderosis

* Indirect sign in CT: intraparenchymal hemorrhage or nonspecific edema; indirect sign in MRI: venous stasis or collateral slow flow

cSAH was clearly demonstrated as a hyperintense cord sign on T2WI and FLAIR images three weeks later. The asymptomatic bilateral SCH found on T2WI implied previous occult SAHs from recurrent ICVT. Table 1 summarizes the reported neuroimaging findings of cSAH and ICVT using brain CT and MRI. The American Heart Association/American Stroke Association Stroke Council recommends MRI as a superior diagnostic tool compared with CT for managing TIA[1]. Digital subtraction angiography diagnosis of cortical vein thrombosis relies on indirect signs, such as delayed local venous drainage, lack of opacification of the corresponding brain region, or missing cortical veins, and these have been replaced by the use of MR studies as well.

Treatment and outcomes of cSAH depend on the underlying etiology. In Kitamura et al's review, 82.5% of patients with ICVT received anticoagulants and their outcomes were not different from those of patients without anticoagulant therapy[20]. Although the outcomes of patients with ICVT are generally favorable, the outcomes of patients with cSAH from ICVT are not well understood due to the rarity of the disease.

In conclusion, TIA-mimic syndrome deserves to be surveyed more carefully and more intensively in order to identify the various possible underlying etiologies. MR studies, particularly the SWI and GRE T2 sequences, provides more information than brain CT studies and these help with the recognition of uncommon acute symptomatic and chronic silent cSAHs, as well as pinpointing a rare cause of cSAH, namely ICVT.

ACKNOWLEDGEMENT

The study was supported by grants from Taipei Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation (TCRD-TPE-103-RT-16).

REFERENCES

1. Easton JD, Saver JL, Albers GW, Alberts MJ, Chaturvedi S, Feldmann E, Hatsukami TS, Higashida RT, Johnston SC, Kidwell CS, Lutsep HL, Miller E, Sacco RL. Definition and evaluation of transient ischemic attack: a scientific statement for healthcare professionals from the American Heart Association/ American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists. Stroke 2009; 40:2276–2293.
2. Nadarajan V, Perry RJ, Johnson J, Werring DJ. Transient ischaemic attacks: mimics and chameleons. Pract Neurol 2014;14:23–31.
3. Renou P, Tourdias T, Fleury O, Debruxelles S, Rouanet F, Sibon I. Atraumatic nonaneurysmal sulcal subarachnoid hemorrhages: a diagnostic workup based on a case series. Cerebrovasc Dis 2012;34:147-152.
4. Ertl L, Morhard D, Deckert-Schmitz M, Linn J, Schulte-Altedorneburg G. Focal subarachnoid haemorrhage mimicking transient ischaemic attack--do we really need MRI in the acute stage? BMC Neurol 2014;14:80.
5. Apoil M, Cogez J, Dubuc L, Bataille M, de la Sayette V, Touzé E, Viader F. Focal cortical subarachnoid hemorrhage revealed by recurrent paresthesias: a clinico-radiological syndromestrongly associated with cerebral amyloid angiopathy. Cerebrovasc Dis 2013;36:139-144.
6. Kumar S, Goddeau RP Jr, Selim MH, Thomas A, Schlaug G, Alhazzani A, Searls DE, Caplan LR. Atraumatic convexal subarachnoid hemorrhage: clinical presentation, imaging patterns, and etiologies. Neurology 2010;74:893-899.
7. Battal B, Sari S, Hamcan S, Akgun V. Susceptibility-weighted imaging in the diagnosis of isolated cortical vein thrombosis. Eur Neurol 2014;71:57-58.
8. Singh R, Cope WP, Zhou Z, De Witt ME, Boockvar JA, Tsiouris AJ. Isolated cortical vein thrombosis: case series. J Neurosurg 2015;123:427-433.
9. Beitzke M, Gattringer T, Enzinger C, Wagner G, Niederkorn K, Fazekas F. Clinical presentation, etiology, and long-term prognosis in patients with nontraumatic convexal subarachnoid hemorrhage. Stroke 2011;42:3055-3060.
10. Field DK, Kleinig TJ. Aura attacks from acute convexity subarachnoid haemorrhage not due to cerebral amyloid angiopathy. Cephalalgia 2011;31:368-

371.
11. Brunot S, Osseby GV, Rouaud O, Kazemi A, Ricolfi F, Couvreur G, Catteau A, Hervieu M, Moreau T, Giroud M, Béjot Y. Transient Ischaemic Attack Mimics Revealing Focal Subarachnoid Haemorrhage. Cerebrovasc Dis 2010;30:597-601.
12. Izenberg A, Aviv RI, Demaerschalk BM, Dodick DW, Hopyan J, Black SE, Gladstone DJ. Crescendo Transient Aura Attacks: A Transient Ischemic Attack Mimic Caused by Focal Subarachnoid Hemorrhage. Stroke 2009;40:3725-3729.
13. Cuvinciuc V, Viguier A, Calviere L, Raposo N, Larrue V, Cognard C, Bonneville F. Isolated acute nontraumatic cortical subarachnoid hemorrhage. AJNR Am J Neuroradiol 2010;31:1355-1362.
14. Rumboldt Z, Kalousek M, Castillo M. Hyperacute Subarachnoid Hemorrhage on T2-Weighted MR Images. AJNR Am J Neuroradiol. 2003;24:472-475.
15. Mitchell P, Wilkinson ID, Hoggard N, Paley MN, Jellinek DA, Powell T, Romanowski C, Hodgson T, Griffiths PD. Detection of subarachnoid haemorrhage with magnetic resonance imaging. J Neurol Neurosurg Psychiatry 2001;70:205-211.
16. Tang MY, Chen TW, Zhang XM, Huang XH. GRE T2*-weighted MRI: principles and clinical applications. Biomed Res Int 2014;2014:312142.
17. Boukobza M, Crassard I, Bousser MG, Chabriat H. MR imaging features of isolated cortical vein thrombosis: diagnosis and follow-up. AJNR Am J Neuroradiol 2009;30:344-348.
18. Morris JG, Fisher M, Carandang RA. Cortical vein thrombosis as a mimic for isolated cortical subarachnoid hemorrhage and transient ischemic attack. Case Rep Neurol 2010;2:63-68.
19. Rathakrishnan R, Sharma VK, Luen TH, Chan BP. The clinico-radiological spectrum of isolated cortical vein thrombosis. J Clin Neurosci 2011;18:1408-1411.
20. Kitamura Y, Hara K, Tsunematsu K. Isolated superficial sylvian vein thrombosis with long cord sign: case report and review of the literature. Neurol Med Chir (Tokyo) 2014;54:253-259.
21. Linn J, Michl S, Katja B, Pfefferkorn T, Wiesmann M, Hartz S, Dichgans M, Brückmann H. Cortical vein thrombosis: the diagnostic value of different imaging modalities. Neuroradiology 2010;52:899-911.
22. Linn J, Herms J, Dichgans M, Brückmann H, Fesl G, Freilinger T, Wiesmann M. Subarachnoid hemosiderosis and superficial cortical hemosiderosis in cerebral amyloid angiopathy. AJNR Am J Neuroradiol 2008;29:184-186.
23. Kumar N, Cohen-Gadol AA, Wright RA, Miller GM, Piepgras DG, Ahlskog JE. Superficial siderosis. Neurology 2006;66:1144-1152.
24. Linn J, Halpin A, Demaerel P, Ruhland J, Giese AD, Dichgans M, van Buchem MA, Bruckmann H, Greenberg SM. Prevalence of superficial siderosis in patients with cerebral amyloid angiopathy. Neurology 2010;74:1346-1350.