

Recurrent Transient Neurological Deficit Due to Intracerebral Steal Phenomenon in Association with a Developmental Venous Anomaly

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We report a symptomatic developmental venous anomaly (DVA) not causing parenchymal abnormality to provide a pathophysiologic clue in patients with recurrent transient neurologic deficit. A 32-year-old male presented with recurrent transient motor aphasia and headache in the left fronto-temporal region for three years. The symptoms usually lasted for one hour. Brain computed tomography (CT) angiography and magnetic resonance imaging using gradient recalled echo showed a prominent penetrating vein at the left frontal periventricular region. Brain CT perfusion imaging performed during the symptoms revealed increased perfusion in the corresponding area with relatively decreased perfusion in the left fronto-temporal cortices. Digital subtraction angiography revealed collecting venous blood from the left septal and thalamostriate veins draining into the left cavernous sinus without early arteriovenous shunting. In this patient, an inciting incident might have led to imbalance of the venous flow surrounding the DVA, causing venous hypertension and the intracerebral steal phenomenon in the surrounding area. The relatively hypoperfused cortical area adjacent to the DVA could be considered the cause of the transient motor aphasia, while venous hypertension could be the cause of the headache.

Key Words: Developmental venous anomaly—Transient ischemic attack—Intracerebral steal phenomenon—Asymptomatic vascular malformation
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Introduction

Developmental venous anomaly (DVA) is a form of intracranial vascular malformation with an incidence of 2.6% in a brain autopsy study.^{1,2} Usually presenting as a benign form draining normal brain parenchyma, DVA

rarely requires surgical intervention, unless it is symptomatic.^{1,2}

We present a symptomatic DVA not causing parenchymal abnormality to provide a pathophysiologic clue for patients with recurrent transient neurologic deficit.

Case report

A 32-year-old non-smoking male with no underlying medical history presented with recurrent transient motor aphasia and throbbing headache in the left fronto-temporal region for three years. The symptoms usually lasted for one hour. Upon admission, no focal neurologic deficit was found.

Brain computed tomography (CT) angiography (Fig. 1A) and magnetic resonance imaging using gradient recalled echo (Fig. 1B) showed a prominent penetrating vein at the left frontal periventricular region. Brain CT perfusion imaging (Fig. 1C) performed in the presence of the symptoms revealed

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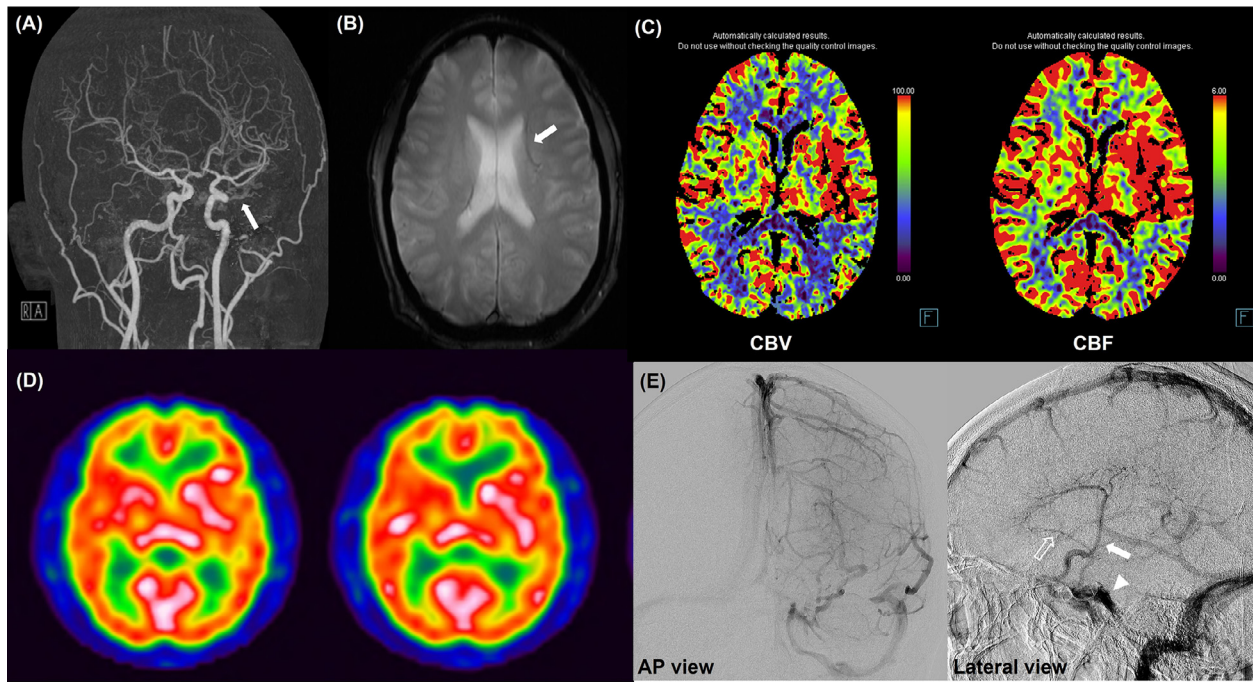


Fig. 1. Computed tomography (CT) angiography (A) and magnetic resonance imaging using gradient recalled echo (B) show developmental venous anomaly (arrows). CT perfusion images (C) reveal increased cerebral blood volume and cerebral blood flow in the left frontal area but relatively decreased flow in the left fronto-temporal cortices, also observed as hypoperfusion in brain single-photon emission computed tomography (D). Digital subtraction angiography (E) presents a venous anomaly draining from the left septal vein (hollow arrow) and thalamostriate vein (white arrow) to the cavernous sinus (arrow head).

increased perfusion in the corresponding area with relatively decreased perfusion in the left fronto-temporal cortices, which also was found on brain single-photon emission computed tomography (Fig. 1D). Digital subtraction angiography (Fig. 1E) revealed collecting venous blood from the left septal and thalamostriate veins draining into the left cavernous sinus without early arteriovenous shunting. Further treatment is being withheld as the patient has since remained asymptomatic.

Discussion

The following possible pathomechanisms of symptomatic DVA have been proposed: (i) mechanical compression of the cerebrospinal fluid from a dilated collecting vein; (ii) increased/decreased outflow causing venous hypertension or thrombosis; and (iii) idiopathic.³

In the deep cerebral venous system, the thalamostriate, septal, and choroid veins are the tributary veins draining into the intracerebral vein.^{1,4} In the present case, the left septal and thalamostriate veins drained into the left cavernous sinus instead of the intracerebral vein, and an inciting incident might have led to imbalance of the venous flow surrounding the DVA, causing venous hypertension and the intracerebral steal phenomenon in the surrounding area.^{1,5} The cortical area adjacent to the DVA, which was relatively hypoperfused, could be considered as the cause of the transient motor aphasia. The

throbbing headache in the left fronto-temporal region could have been brought from the venous hypertension induced by the imbalance of the drain system. The headache has been differentiated from the complicated migraine from his previous history without migraine and relatively increased perfusion in the area of the venous hypertension instead of hypoperfusion induced by a vasoconstriction followed by downstream ischemia in the complicated migraine.⁶

Symptomatic DVA, consisting 1% of patients of DVA detected, is usually presented in different ways, such as ischemic infarction, hemorrhage, headache, ataxia, or seizure.^{7,9,10} Only two cases of transient neurologic deficit without parenchymal involvement associated with DVA have been reported, which is extremely rare, even considering the rarity found in the symptomatic DVA itself presented as hemorrhage in studies ranges from 0.22 to 0.68%.⁷⁻⁹ An intracerebral steal phenomenon associated with an intracranial DVA should be considered in patients with recurrent transient ischemic attacks.

CRedit authorship contribution statement

Hyeyoung Jung: Data curation, Formal analysis, Investigation. **Jae Young An:** Conceptualization, Project administration, Investigation, Supervision, Validation, Writing - review editing.

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