Early-filling cerebral vein

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A 46-year-old man underwent biopsy of a cerebral lesion suspected to be a primary neoplasm. Cranial computed tomography demonstrated an ill-defined, low-density area at the left frontotemporal junction. Cerebral angiography disclosed an early filling vein, with displacement of vessels in the left frontal area. The biopsied lesion, however, was found to be a cerebral infarction. Many physicians may be unaware of abnormalities that were commonly noted on cerebral angiography prior to newer techniques of neuroimaging.

Recent advances in neuroimaging have changed patient evaluation drastically. In some cases, neuroimaging has completely replaced older diagnostic modalities. Improved visualization of pathologic conditions assists patient care both directly and indirectly, by reducing the morbidity and mortality associated with invasive neuroradiologic studies. However, increased reliance upon new techniques is concomitant with decreased awareness of the significance of abnormalities demonstrated on conventional testing.

In reviewing previous clinical records, we found an interesting case of cerebrovascular disease in which unfamiliarity with the angiographic finding of an early-filling vein (EFV) obscured clinical judgment and led to the misdiagnosis of stroke.

Report of case

A 46-year-old, right-handed man in good health awoke unable to speak, write, or read and was brought to a local hospital. Computed tomography (CT) and EEG results were normal.

Three days later, the patient was transferred to our institution. Neurologic evaluation was normal, except for fluent, nonrepetitive aphasia, with occasional para-

phasias and word-finding difficulties (but good comprehension). Thromboembolic infarction was thought to be the etiology of the patient's symptoms.

Repeat CT demonstrated a low-density, nonenhancing area in the left frontotemporal area (Fig 1). Two-dimensional echodardiography had normal findings. Cerebral angiography, which was performed five days later, showed no evidence of carotid disease, but it defined an area of contrast pooling, an early-filling vein in the left frontal area, and slight mass effect upon adjacent vessels (Fig 2).

The possibility of a primary CNS neoplasm presenting as a stroke was considered, and biopsy of the lesion was performed. An abnormal dilated cortical vein was visualized, and the surrounding tissue appeared hemosiderin-stained without mass effect. Histopathology was consistent with infarction. Postoperatively, the patient had no added neurologic deficits.

Follow-up evaluation a year later revealed no clinical signs of neurologic deterioration. Only mild word finding difficulties persisted. At that time, CT showed a stable, nonexpanding hypodense lesion, which was compatible with a diagnosis of ischemic infarction.

Discussion

The appearance of a vein within the first three seconds of intracerebral contrast circulation, or the visualization of an abnormal sequence of venous filling, is referred to as an EFV.^{1,2} Attributed to an increase in local cerebral blood flow, EFV is an expected finding in vascular malformations and neoplasms. Its occurrence in cerebral infarction (occasionally disappearing with hyperventilation³) also suggests that it can represent a transient disturbance of cerebral metabolism in stroke, as well as in other cerebral disorders. (Table 1.⁴)

A review of the angiographic features described in cerebral infarction reveals three sequential stages, although any one or all may be seen in any patient.⁵ The first is local vasodilation and a focal increase in the rate of circulation, or "luxury per-

Fig 1. CT scan without contrast demonstrates somewhat ill-defined low density in the left frontal area.

fusion.”⁶ This is believed to be caused by local tissue acidosis. These changes result in local increase in blood flow and decrease in normal circulation time.⁷⁻¹⁰ The angiographic correlate is a vascular blush or the appearance of EFVs, or both.⁸⁻¹¹

The second stage is a focal slowing of circulation, during which a delay in emptying of an arterial branch or prolongation of local arteriovenous circulation time occurs.^{7,8} The last stage is a generalized prolongation of arteriovenous circulation time. This may be more extensive than the area of infarction and can involve the entire hemisphere.⁹

The time course of each angiographic stage is variable. The first can occur immediately, but it generally is thought to represent a transit event that lasts one to two weeks.¹¹ The durations of the remaining two stages are more variable; they are influenced by the completeness of infarction, collateral blood flow, size of infarction, and central regulation of cerebral blood flow, and they possibly may be progressive because of continuing atherosclerosis.^{5,12,13}

The frequency of superficial EFVs in cerebral infarction is estimated to be as high as 40%,¹⁴ while deep EFVs occur in approximately 30% of cases.¹¹ Despite attempts to improve its detection and speci-

Fig 2. Lateral view of left internal carotid angiogram in mid-to-late-arterial phase shows an early-filling cortical vein with contrast pooling and minimal mass effect.

TABLE 1. ETIOLOGY OF CEREBRAL EFV. ⁴

Abscess
Angioma
Arteriovenous malformation
Cerebral infarction with "luxury perfusion" (atherothrombosis, cerebral arteritis, or vasospasm secondary to subarachnoid hemorrhage)
Cerebral trauma
Degenerative CNS disease
Meningoencephalitis
Neoplasm, or brain compression resulting from another mass
"Normal" variant
Status epilepticus
Toxic encephalopathy

ficity, the findings of EFV in only a percentage of cerebral infarction patients suggests that focal hyperemia plays a role in only a select group.^{15,16} Because the EFV is an angiographic abnormality related to changes in cerebrovasculature, methods that evaluate cerebral blood flow and metabolism might correlate with its pathophysiology.¹⁷

Comment

EFV is a well-recognized angiographic finding in cerebral infarction. However, familiarity with it has decreased as other neuroimaging techniques have proliferated. In the case presented here, the finding of an EFV on angiography led to cerebral biopsy and the diagnosis of infarction. In retrospect,

re-evaluating the patient with CT one or two weeks later likely would have made the diagnosis more obvious.

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