Pathophysiology of Stroke in the Contralateral Posterior Cerebral Artery Distribution from a Tentorial Herniation

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Dear Editor,

Since the original description of ipsilateral occipital-lobe infarction from a tentorial herniation in 1920,¹ pathologic and neuroradiologic studies have suggested posterior cerebral artery (PCA) impingement against the leading edge of the tentorial incisura as the underlying mechanism. However, the rare cases of isolated contralateral PCA infarction have not been explained. The case reported here illustrates the underlying pathophysiologic mechanism of this unusual phenomenon.

An 81-year-old otherwise healthy woman with history of atrial fibrillation and taking rivaroxaban slipped and fell on the floor. Three days later she was found in an unresponsive state. The examination in the emergency room revealed a stuporous woman with sluggish pupils. An admission CT scan on day 1 disclosed a right subdural hematoma with marked midline shift. Following emergent surgical evacuation, 1 day later she was able to show two fingers with her right hand on command and had mid-sized briskly reactive pupils. A visual-field assessment was unreliable due to lethargy and eyelid-opening apraxia. A follow-up CT scan on day 3 revealed a left occipital infarction (Fig. 1C) with resolution of the midline shift. She subsequently became septic and ventilator-dependent. She was placed on comfort care and expired 2 weeks following the subdural evacuation.

Ipsilateral pupillary dilatation from oculomotor nerve compression (cranial nerve III) is a cardinal clinical sign of evolving rostral-caudal temporal-lobe transtentorial herniation. Similarly, the PCA, which originates at the tip of the basilar artery rounding the midbrain just above the origin of the oculomotor nerve, may be impinged upon, resulting in occipital-lobe infarction.

Keane² reported seven cases of bilateral visual-field blindness in patients who survived tentorial herniation, and speculated that caudal descent and distortion of the brainstem could have been responsible for the contralateral PCA impinging on the tentorial edge. Sato et al.³ noted occipital infarction from a tentorial herniation based on CT scans in 9 of 100 consecutive patients. An ipsilateral occipital infarction was revealed in seven of these nine cases, whereas contralateral occipital infarction was present in only two. Those authors did not speculate on the mechanism underlying this rare phenomenon.

The admission CT in our case revealed a severe leftward midline shift from the right subdural hematoma (Fig. 1A). The upper brainstem was being pulled to the left resulting in a widened ipsilateral prepontine cistern (arrowhead in Fig. 1B). A consequence of this midline shift is distortion of the interventricular foramen that impedes the contralateral CSF outflow. An enlarged left lateral ventricle (arrow in Fig. 1B) independently worsened the compartment pressure via an “accordion” effect. The combined forces associated with the midline shift and left ventricular enlargement result in the descent of the left medial temporal lobe causing the PCA to impinge on the rigid tentorial edge.

An isolated contralateral PCA infarction secondary to herniation requires a predominant

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Fig. 1. Axial brain CT slices: (A and B) on admission and (C) 2 days later following subdural evacuation. A: A large right subdural hematoma with a marked midline shift and a relative left ventriculomegaly. B: Widened ipsilateral prepontine cistern (arrowhead) with leftward rostral brainstem shift, with a dilated left temporal horn (arrow). C: Left occipital-lobe infarction with residual tentorial subdural hematoma. The leftward brainstem shift and left temporal horn enlargement have both improved.

horizontal vector shift from an ipsilateral expanding mass. The interaction between an ipsilateral rostrocaudal descent and horizontal contralateral midline shift probably determines whether an ipsilateral, contralateral, or bilateral PCA infarction occurs.

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