Contusional Kernohan notch phenomenon

Gregory Youngnam Chang, MD

A 30-year-old driver was involved in a car accident. Examination revealed left scalp swelling and left hemiparesis including the face. Posttraumatic Kernohan notch phenomenon typically refers to a subdural hematoma resulting in delayed ipsilateral hemiparesis.¹ Evolving uncus herniation through the tentorial incisura displaces the cerebral peduncle against the contralateral rigid tentorium, producing this false localizing sign.

In this case, imaging (figure) showed lateralized temporal lobe microhemorrhages under the site of head impact with splenic hemorrhage and a contralateral cerebral peduncular lesion suggesting recoil of the brain against the immobile falx cerebri and contralateral tentorium resulting in a contusional Kernohan notch phenomenon.

Figure Axial brain MRI slices

Diffusion-weighted image reveals a right peduncular lesion (A, arrow). Susceptibility-weighted images show contusional microhemorrhages under the site of head trauma (B, circle) and a splenic hemorrhage (C, arrowhead).

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AUTHOR CONTRIBUTIONS

Designed the concept, analyzed data, and wrote the clinical summary.

Department of Neurology, UC Davis Medical Center, Sacramento, CA.

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Correspondence to: gycchang@ucdavis.edu

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COMMENT: Bumping up Kernohan notch phenomenon

In 1929, Drs. Kernohan and Woltman described a phenomenon of hemiparesis that paradoxically localized to an ipsilateral brain tumor.¹ The mechanism involved uncus herniation from tumoral mass effect leading to brainstem displacement and compression of the contralateral cerebral peduncle against the tentorium cerebelli.

In this issue of *Neurology*[®] Clinical Practice, Dr. Chang² reports a case similar to the Kernohan notch phenomenon (KNP), but with a mechanism that is distinct from previously reported cases. Whereas classic KNP implied that the etiology is a supratentorial intraparenchymal mass or subdural hematoma leading to uncus herniation, in this case, the supratentorial injury is not exerting mass effect and a different mechanism is evoked. In this contusional KNP, it seems that the kinetic displacement of the brainstem against the tentorium directly led to midbrain edge compression injury. MRI reveals the presence of linear restricted diffusion in the contralateral midbrain. Hemorrhages in the ipsilateral splenium suggest a second site of crush injury that occurred from the kinetic central brain shift against the falx cerebri. Another less likely mechanism would be a transient uncus herniation from the supratentorial contusion that resolved by the time of imaging. More likely, uncus herniation is not a sine qua non for the diagnosis of KNP. Indeed, transtentorial herniation with a downward-lateral shift can produce the same type of injury.³ Similarly, in the absence of uncus herniation, ipsilateral mydriasis may not occur, and in fact, further brainstem shift can lead to compression of the contralateral third cranial nerve, causing contralateral mydriasis.³ Finally, ipsilateral hemiparesis similar to KNP can also occur in the absence of any type of herniation or contusion, e.g., due to cerebellar edema with brainstem displacement.⁴

KNP can thus be bumped up a notch in classification: A classic KNP, as initially described; a contusional KNP, with velocity-related recoil injury without evidence of herniation; and a modified KNP, with contralateral mydriasis.

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Heustein Sy, MD and Gregory Kapinos, MD, MS

Departments of Neurosurgery and Neurology, Hofstra Northwell School of Medicine, Manhasset, NY.

Corresponding Author: Gregory Kapinos, MD, MS, North Shore University Hospital, Dept of Neurosurgery, 300 Community Drive, Tower, 9th Floor, Manhasset, NY 11030, Ph 516-562-3020, Fax: 516-562-3631, kapiGreg@gmail.com

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