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## Case Report

# Unilateral cerebral herniation resulting in combined contralateral superior cerebellar artery territory infarction and mesencephalic injury: Two cases of a severe unrecognized variant of Kernohan notch phenomenon?

John D. Leever, MD\*

The University of Kansas Medical Center, Department of Radiology, 4000 Cambridge St, Kansas City, KS 66160, USA

### ARTICLE INFO

#### Article history:

Received 29 October 2019

Revised 29 November 2019

Accepted 11 December 2019

#### Keywords:

Kernohan

Notch

Transtentorial

Herniation

Cerebellar

Mesencephalic

### ABSTRACT

A case of unilateral cerebral herniation due to an acute middle cerebral artery territory infarct and a second case of unilateral cerebral herniation due to an acute subdural hematoma are presented in this article. In both instances, the unilateral cerebral herniation resulted in a combined contralateral superior cerebellar artery territory infarction and mesencephalic injury. Unilateral cerebral herniation resulting in a combined contralateral superior cerebellar artery territory infarct and mesencephalic injury is previously undescribed in the literature and likely reflects a severe unrecognized variant of Kernohan notch phenomenon.

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## Introduction

Only 2 case reports of unilateral cerebral herniation resulting in contralateral superior cerebellar artery (SCA) territorial infarction have been reported in the literature. Furthermore, this finding has never been reported in combination with Kernohan notch phenomenon (KNP), which is recognized as

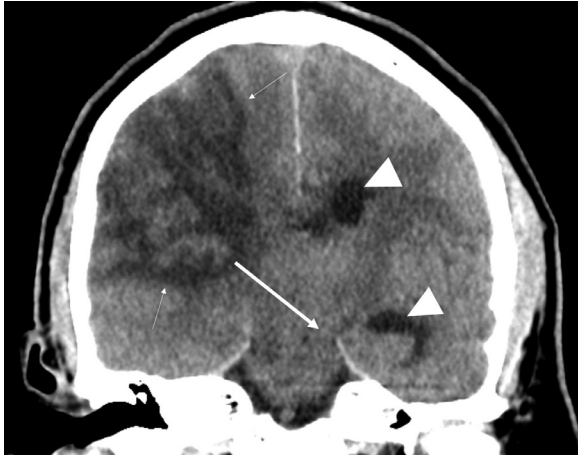
a contralateral crus cerebri compressive injury in the setting of unilateral cerebral herniation. Two cases of previously undescribed unilateral brain herniation resulting in combined contralateral SCA territorial infarction and contralateral compressive mesencephalic injury are presented in this article. Recognition of this potential complication of cerebral herniation is necessary for optimal management and patient care.

\* Corresponding author.

E-mail address: [jleever@kumc.edu](mailto:jleever@kumc.edu)

<https://doi.org/10.1016/j.radcr.2019.12.004>

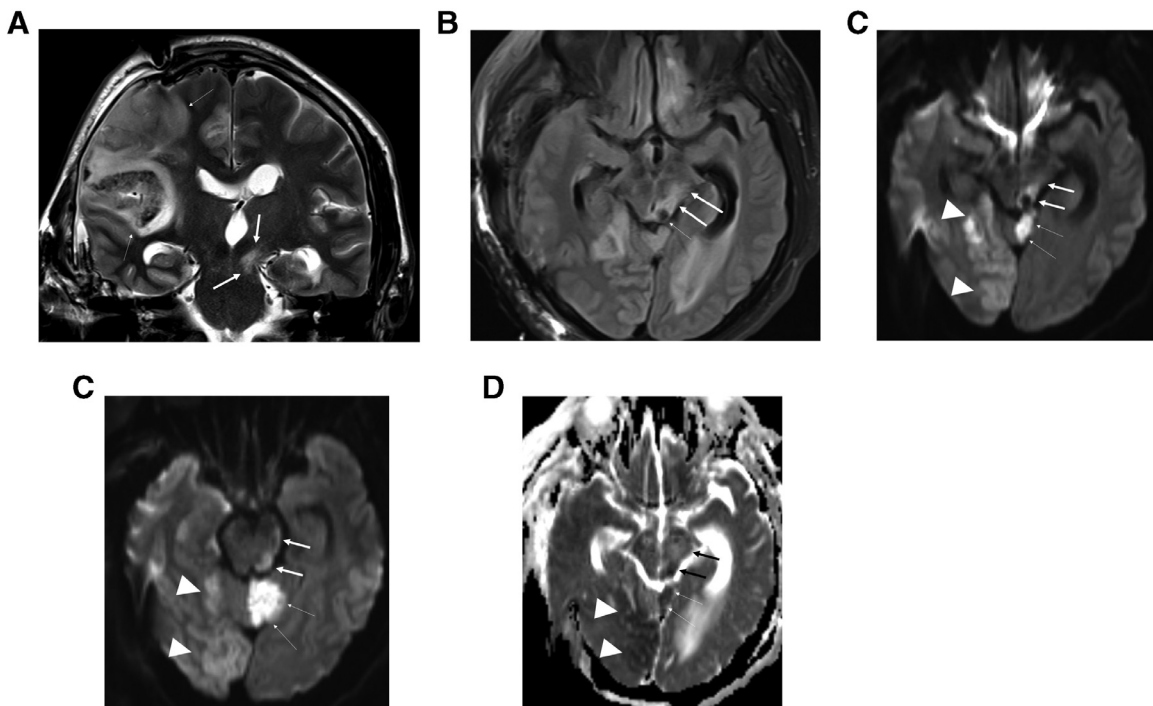
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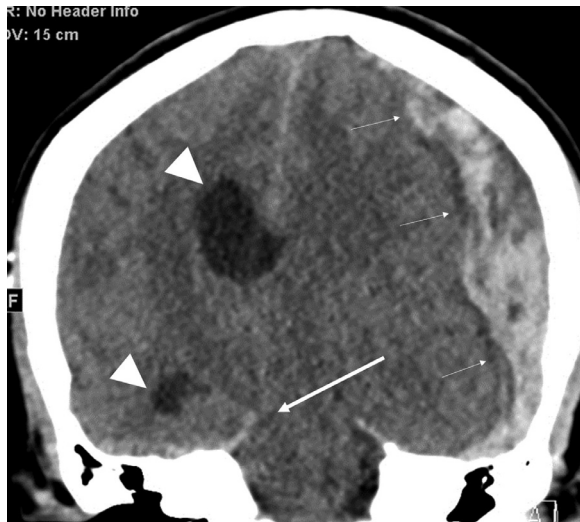
**Fig. 1 – Case 1 Noncontrast CT scan of the head with a large acute right middle cerebral artery (MCA) territory infarct (small arrows), midline shift resulting in indentation of the contralateral cerebral crus by the tentorium cerebelli (large arrow), and left sided unilateral, noncommunicating hydrocephalus (arrowheads).**

### Case 1

A 59-year old male with hypertension, diabetes, hyperlipidemia, and atrial fibrillation presented to an outside hospital with sudden onset of slurred speech, left hemiparalysis, and right gaze preference. CT head revealed a large acute right middle cerebral artery territory infarct with 3 mm of leftward midline shift. Patient was transferred to a tertiary care facility and experienced declining status requiring intubation with inability to follow commands and physical exam limited to reflexes. Repeat CT scan of the head revealed progressive leftward midline shift (up to 16 mm), indentation of the contralateral cerebral crus by the tentorium cerebelli, and left sided unilateral, noncommunicating hydrocephalus (Fig. 1). An emergent right hemicraniectomy was performed with little improvement in patient status. A subsequent MRI of the head revealed diffusion restriction and fluid attenuated inversion recovery (FLAIR) hyperintensity diffusely involving the left SCA territory, right posterior cerebral artery (PCA) territory, and within the left lateral mesencephalon (including the crus cerebri, lateral tectum, and upper tectum)



**Fig. 2 – Case 1 MRI of the head post right hemicraniectomy with evolving subacute right MCA territory infarct, new ipsilateral PCA territory infarct, and new contralateral SCA territory infarct and Kernohan notch phenomenon type mesencephalic injury. (A) Coronal T2 image demonstrating large evolving subacute right MCA territory infarct with mild petechial type hemorrhagic conversion (small arrows) and T2 hyperintense signal abnormality within the left crus cerebri and lateral tectum (big arrows) suggestive of Kernohan notch phenomenon type injury. (B) Axial FLAIR image demonstrating abnormal FLAIR hyperintense signal within the left crus cerebri, lateral tectum, and upper tectum (big arrows) and superimposed FLAIR hypointense hemorrhage within the left upper tectum suggestive of Kernohan notch phenomenon type injury. (C) Axial diffusion weighted Imaging (DWI) image demonstrating high DWI signal within the left crus cerebri, lateral tectum, and upper tectum (big arrows), right PCA territory (arrowheads), and left SCA territory (small arrows). (D) Axial DWI image demonstrating high DWI signal within the left crus cerebri, lateral tectum, and upper tectum (big arrows), right PCA territory (arrowheads), and left SCA territory (small arrows). (E) Axial apparent diffusion coefficient (ADC) image demonstrating low ADC signal, consistent with diffusion restriction, within the left lateral mesencephalon (big arrows), right PCA territory (arrowheads), and left SCA territory (small arrows). PCA, posterior cerebral artery; SCA, superior cerebellar artery.**



**Fig. 3 – Case 2 Noncontrast CT scan of the head with a large acute left supratentorial subdural hematoma (small arrows), rightward unilateral descending transtentorial cerebral herniation (large arrow), and right sided unilateral, noncommunicating hydrocephalus (arrowheads).**

(Fig. 2A-E). A tiny acute hematoma was also present within the left lateral tegmentum (Fig. 2B-C). The patient made little progress in the ensuing days and expired shortly after being switched to comfort measures only.

### Case 2

A 55-year old female with hypertension, diabetes, hyperlipidemia, chronic kidney disease, and monoclonal gammopathy of undetermined significance was found unresponsive and was emergently intubated. Initial CT scan of the head revealed a large acute left supratentorial convexity subdural hematoma with associated rightward midline shift, left unilateral descending transtentorial herniation, and a right sided unilateral, noncommunicating hydrocephalus (Fig. 3). Emergent decompressive left hemicraniectomy and subdural hematoma evacuation was performed. Patient demonstrated a limited neurological exam with minimal reflexes. A subsequent MRI of the brain demonstrated diffusion restriction and FLAIR hyperintensity diffusely involving the right SCA territory and left PCA territory (Fig. 4A-E). FLAIR hyperintensity was also demonstrated within the right lateral mesencephalon (including the crus cerebri, lateral tegmentum, and upper tectum) without associated diffusion restriction or hemorrhage (Fig. 4A-E). The patient's status and physical exam continued to decline and the patient expired shortly after being switched to comfort measures only.

### Discussion

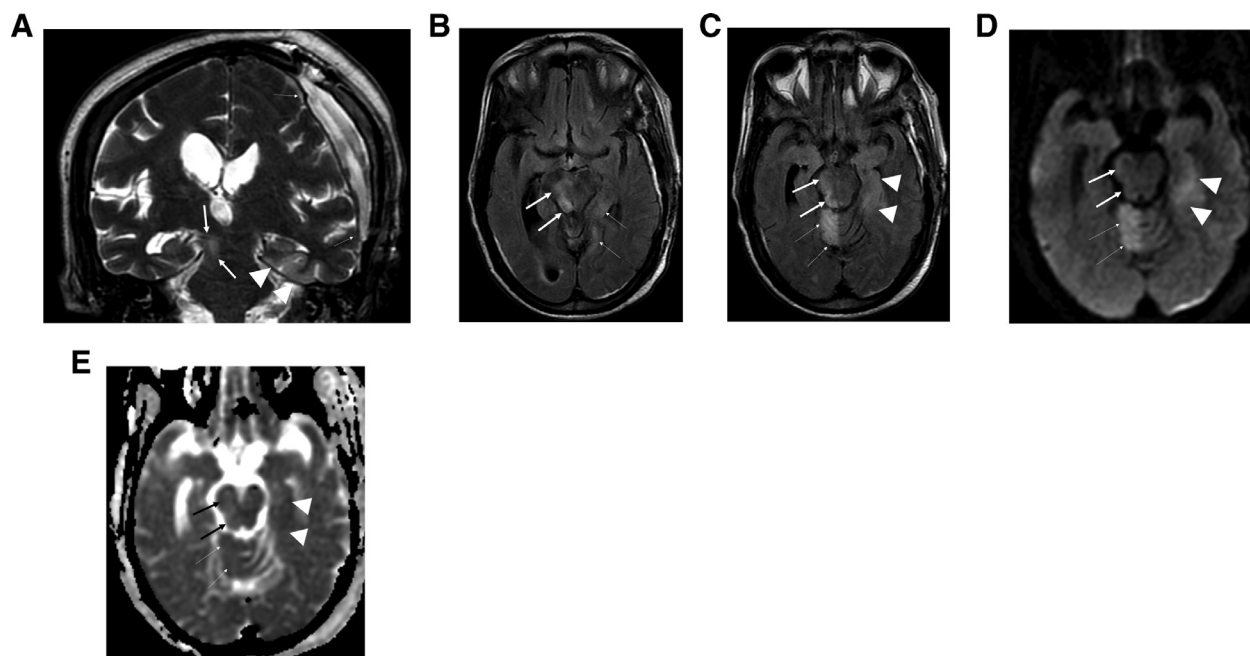
KNP and territorial vascular compromise are both well-established complications of unilateral cerebral herniation.

More than 40 cases of KNP have been reported in the literature and secondary PCA territory infarcts have been reported in up to 20% of transtentorial herniation cases [1,2]. However, unilateral cerebral herniation resulting in contralateral SCA territorial infarction has been previously reported only twice in the literature. Including the 2 cases in this article, all 4 cases of contralateral SCA territory infarct have been accompanied by an ipsilateral PCA territory infarct, however, an associated KNP type injury has never been previously described [3,4].

The original description of KNP, also sometimes referred to as Kernohan-Woltman notch phenomenon, involved a cerebral tumor presenting with a false localizing ipsilateral hemiparesis due to compression of the contralateral crus cerebri at the cerebellar tentorial free edge [5]. Magnetic resonance imaging features of KNP typically include T2 and FLAIR hyperintensity and T1 hypointensity within the contralateral crus cerebri with variable diffusion restriction or hemorrhage [1,6]. In addition to the typical crus cerebri MRI abnormalities of KNP, both of the cases presented in this article, also demonstrated FLAIR hyperintensity within the more inferolateral mesencephalic structures (such as the lateral tegmentum and upper tectum) which is not typically described in the setting of KNP. Both of the patients in this article were intubated with limited reflexes on physical exam at the time of the observed contralateral SCA territory infarct and KNP type injury. Therefore, the classic false localizing ipsilateral hemiparesis clinical presentation of both KNP and contralateral SCA territory infarct was not observed in either case.

It has been previously proposed that the mechanism of contralateral SCA territorial infarction in the setting of unilateral cerebral herniation is mechanical compression of the SCA at the tentorial free edge, similar to that of KNP [3]. The nearly identical imaging features of the acute contralateral SCA territory infarcts and the co-existent KNP type contralateral crus and mesencephalic injuries in both of these cases supports the proposed mechanism of mechanical compression of the SCA at the tentorial incisura. The differing imaging features of the contralateral crus and mesencephalic injuries in the 2 cases included cytotoxic edema and superimposed hemorrhage (case 1) and vasogenic edema (case 2). Cytotoxic and vasogenic edema and superimposed hemorrhage have all been previously described with KNP and remain consistent with the proposed mechanism of mechanical compressive injury at the tentorial incisura. Likewise, the co-existent ipsilateral PCA territory infarcts are also presumably due to mechanical compression of the PCA which is a well-established complication of cerebral herniation. Hypothetically, an oblique unilateral descending transtentorial herniation resulted in the observed ipsilateral PCA and contralateral mesencephalic and SCA mechanical compression injuries in both of these cases. The more extensive contralateral mesencephalic injury and worse clinical outcome than is typically expected for KNP and the previously undescribed co-existent contralateral SCA territory infarct in both of these cases implies a previously unrecognized more severe variant of KNP or KNP type injury.

Combined contralateral SCA territory infarction and KNP type mesencephalic injury is a previously undescribed complication of unilateral cerebral herniation. Both contralateral SCA territory infarction and KNP typically present with a false



**Fig. 4 – Case 2 MRI of the head post left craniotomy and subdural hematoma evacuation with new ipsilateral PCA territory infarct, new contralateral SCA territory infarct and Kernohan notch phenomenon type mesencephalic injury.** (A) Coronal T2 image with residual left supratentorial subdural hemorrhage (small arrows), gyral T2 hyperintensity within the mesial left temporal PCA territory (arrowheads), and T2 hyperintense signal abnormality within the right lateral mesencephalon (big arrows) suggestive of Kernohan notch phenomenon type injury. (B) Axial FLAIR image with FLAIR hyperintensity within the right crus cerebri, lateral tectum, and upper tectum (big arrows), and left mesial temporal PCA territory (small arrows). (C) Axial FLAIR image with FLAIR hyperintensity within the right crus cerebri, lateral tectum, and upper tectum (big arrows), left mesial temporal PCA territory (arrowheads), and right SCA territory (small arrows). (D) Axial DWI image demonstrating increased DWI signal within the left mesial temporal PCA territory (arrowheads) and right SCA territory (small arrows) and faintly increased DWI signal within the right crus, lateral tectum, and upper tectum (large arrows). (E) Axial ADC image demonstrating decreased ADC signal, consistent with diffusion restriction, within the left mesial temporal PCA territory (arrowheads) and right SCA territory (small arrows) and faintly increased ADC signal, consistent with increased diffusivity within the right crus, lateral tectum, and upper tectum (large arrows). PCA, posterior cerebral artery; SCA, superior cerebellar artery.

localizing ipsilateral hemiparesis and may be diagnostically challenging and seemingly paradoxical [3]. Ultimately, recognition of these potential complications of unilateral cerebral herniation is necessary for correct diagnosis and optimal management.

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