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The Kernohan-Woltman Notch Phenomenon in Chronic Subdural Hemorrhage

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Introduction

The Kernohan-Woltman notch phenomenon is caused by compression of the contralateral crus cerebri by intracranial lesions, causing a side-to-side mass effect. This leads to paradoxical motor deficits ipsilateral to the lesion due to damage to the contralateral corticospinal tract in the cerebral peduncle (Figure 1). Case reports in rehabilitation medicine are scarce. This report describes the diagnostic findings, clinical manifestations, and rehabilitation outcomes of this phenomenon secondary to chronic subdural hemorrhage.

Case

A 66-year-old male, presented to the emergency department with altered mental status. On physical examination, his Glasgow Coma Scale (GCS) score was 4, and Manual Muscle Test (MMT) revealed trace strength in both upper and lower extremities. Bilateral pupils were dilated and unresponsive. A brain CT scan showed chronic left subdural hemorrhage (SDH) with midline shift to right side and transtentorial left uncal herniation (Figure 2). Emergency craniectomy and hematoma removal were performed.

Four weeks later, he was transferred to the department of rehabilitation medicine for intensive rehabilitation. A brain MRI at that time showed T1 hypointensity, T2 hyperintensity, and T2 FLAIR hyperintensity in the right cerebral peduncle of the midbrain (Figure 3). Physical examination revealed a GCS score of 11, left-sided hemiplegia, left-sided peripheral facial palsy, left-sided ptosis and sluggish pupillary response, dysphagia. MMT showed trace strength in the left upper and lower extremities, and fair on the right side. The Berg Balance Scale (BBS), Mini-Mental State Examination (MMSE) scores were 0. He was receiving L-tube feeding and Videofluoroscopic swallowing study (VFSS) revealed reduced swallowing reflex and aspiration (Penetration-Aspiration Scale [PAS] score 8) due to decreased laryngeal elevation. He was discharged to an Inpatient Rehabilitation Facility (IRF) 8 weeks after onset for intensive rehabilitation programs.

Sixteen weeks after onset, follow up brain MRI showed decreased signal intensity in the right cerebral peduncle (Figure 3). Physical examination revealed a GCS score of 15. MMT showed fair strength in the left upper and lower extremities, and good strength on the right. BBS and MMSE scores were 23. However, the patient continued to exhibit left-sided peripheral facial palsy, left-sided ptosis and sluggish pupillary response, dysphagia. He remained on L-tube feeding, and a follow-up VFSS still showed reduced swallowing reflex and aspiration (PAS score 8).

Conclusion

The Kernohan-Woltman notch phenomenon is a rare condition, rarely reported in rehabilitation medicine. This case report emphasizes the importance of recognizing the phenomenon in patients with paradoxical ipsilateral hemiplegia and cranial nerve deficits, including oculomotor nerve injury. We emphasize the necessity of accurate diagnosis, comprehensive evaluation, and targeted rehabilitation management in this case.

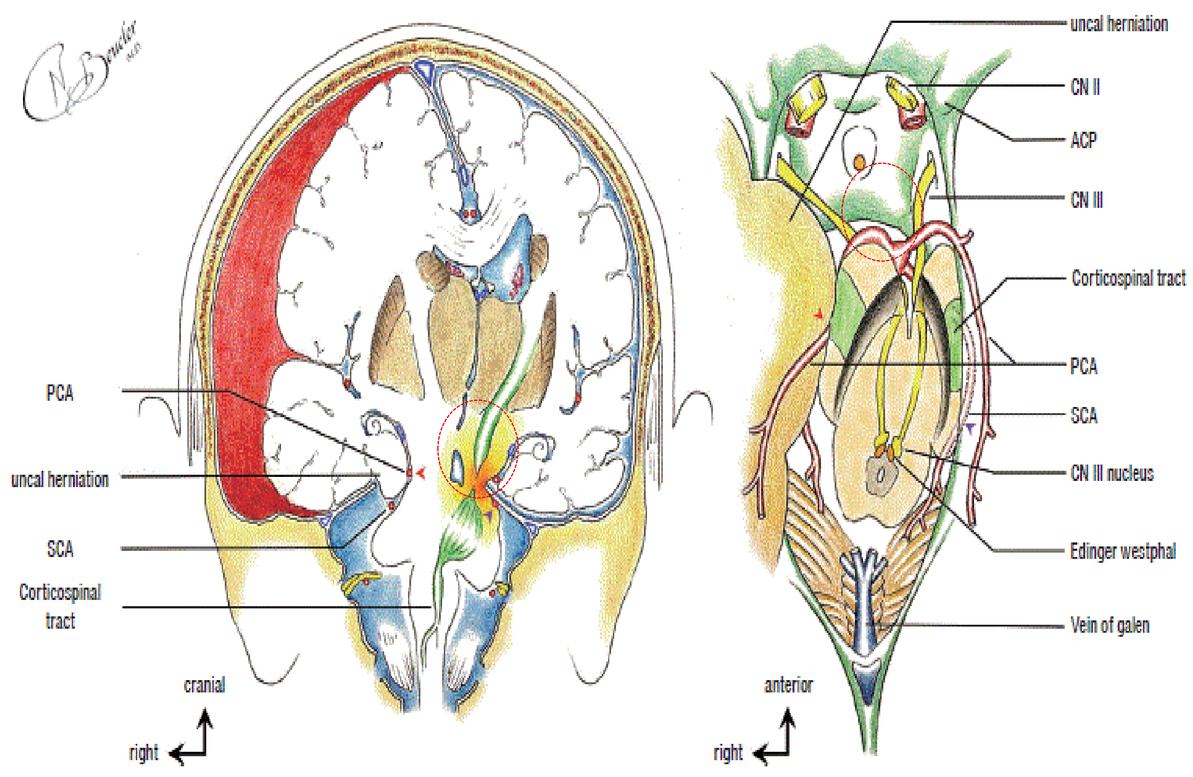

Figure 1. Anatomic image of Kernohan-Woltman notch phenomenon caused by a right-sided acute subdural hematoma in coronal (left image) and axial view (right image). Temporal uncal herniation with marked brainstem displacement results in interruption of the contralateral corticospinal tract (green) due to direct compression of the contralateral cerebral peduncle against the free edge of the cerebellar tentorium (left image, red circle). In axial view, ipsilateral CN III is stretched by the brain stem displacement (right image, red circle). CN III: third cranial nerve, oculomotor nerve.

Reference: Nathan Beucler et al., "The Kernohan-Woltman Notch Phenomenon: A systematic Review of Clinical and Radiologic Presentation, Surgical Management, and Functional Prognosis", Journal of The Korean Neurosurgical Society, 65(5), 652-664, 2022.

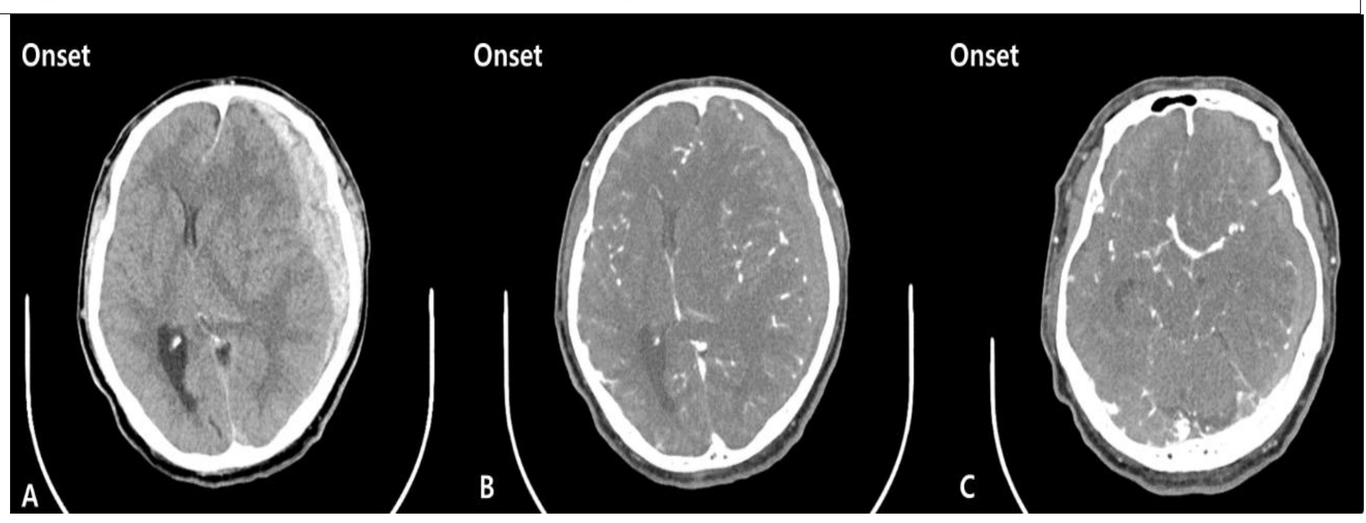

Figure 2. Pre-operative brain CT scans. (A,B,C) showed a chronic left-sided subdural hematoma with severe midline shift.

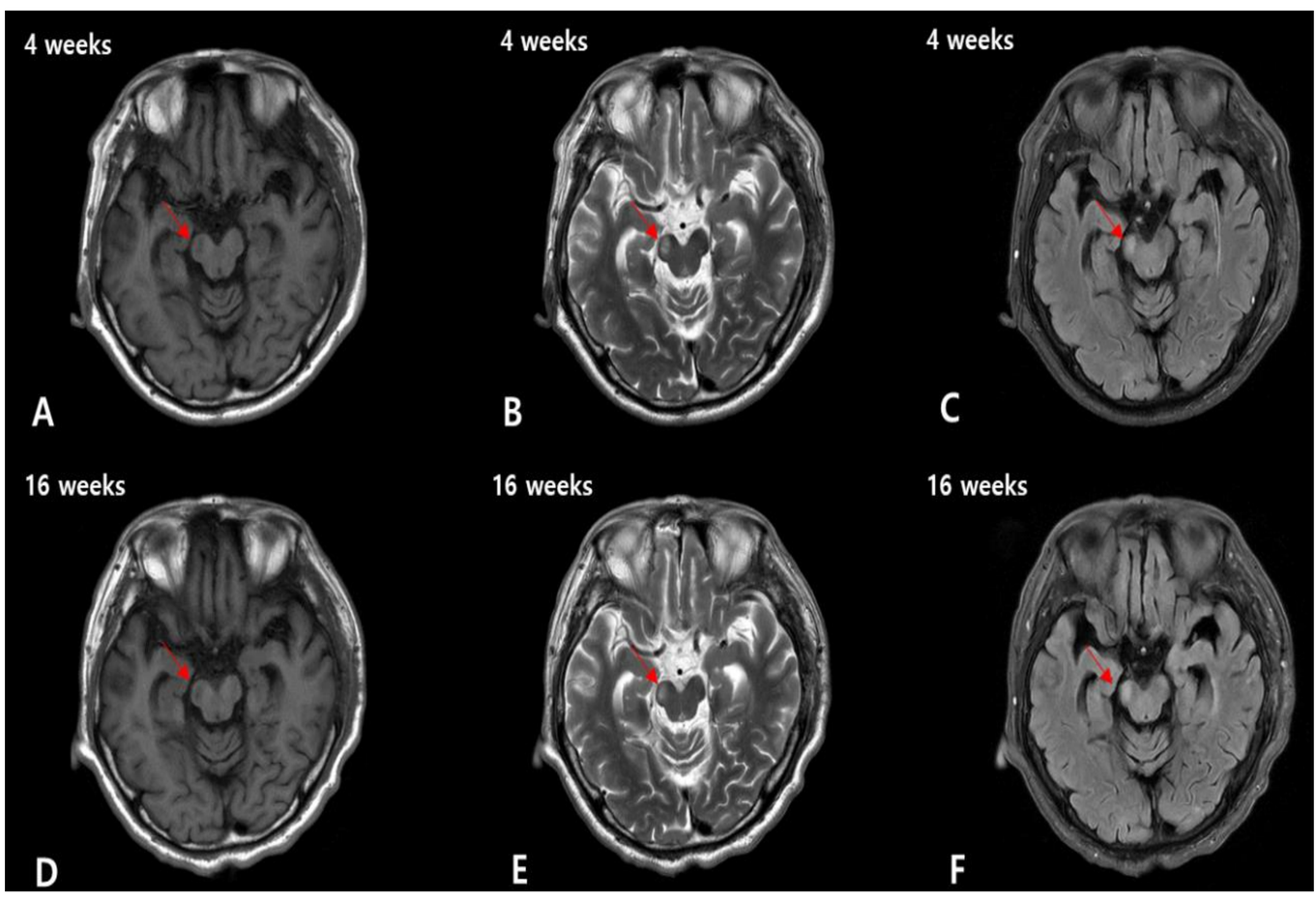

Figure 3. Post-operative brain MRI. (A,B,C) showed 4 weeks after onset. (A) T1-weighted image, (B) T2-weighted image, (C) T2-FLAIR image. (A) T1 and (B, C) T2, T2-FLAIR image showed abnormal signal intensity in the anterolateral portion of the right cerebral peduncle facing the tentorial notch. (D,E,F) showed 16 weeks after onset. (D) T1-weighted image, (E) T2-weighted image, (F) T2-FLAIR image. (D,E,F) showed decreased signal abnormality in the right cerebral peduncle compared with (A,B,C) previous image