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## Kernohan's notch phenomenon in chronic subdural hematoma: MRI findings

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### Abstract

We report two cases of Kernohan's notch phenomenon secondary to chronic subdural hematoma detected by MRI. In the first case, the patient was drowsy with an oculomotor palsy and a hemiparesis ipsilateral to the chronic subdural hematoma. MRI in the post-operative period showed no abnormal signal or deformity of the crus cerebri. The neurological signs immediately resolved after trephination. In the second case, the patient was admitted with progressive decrease in their level of consciousness and ipsilateral hemiparesis with the chronic subdural hematoma. MRI on admission revealed an abnormal signal in the contralateral crus cerebri against the chronic subdural hematoma. After surgery, the mental state gradually recovered to normal with some degree of residual hemiparesis. In patients with chronic subdural hematoma, a compressive deformity of the crus cerebri, without abnormal signal on MRI, may predict a better neurological recovery in patients with Kernohan's notch phenomenon.

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**Keywords:** Chronic subdural hematoma; Ipsilateral hemiparesis; Kernohan's notch; Magnetic resonance imaging

### 1. Introduction

Kernohan's notch phenomenon, compression of the contralateral crus cerebri against the edge of the tentorium that is due to a supratentorial mass, produces ipsilateral hemiparesis or hemiplegia; it is usually observed in patients with severe head injury or advanced brain tumor.<sup>1</sup> Recently, radiological evidence for structural damage of the cerebral peduncle contralateral to a supratentorial lesion has been demonstrated on MRI in patients with acute traumatic brain injury.

To the best of our knowledge, only two patients with Kernohan's notch phenomenon associated with chronic subdural hematoma have been demonstrated on MRI in the literature.<sup>2,3</sup> We report two further cases of Kernohan's notch phenomenon in patients with chronic subdural hematoma on MRI. We also discuss possible mechanisms and the clinico-radiological relationship of this phenomenon with a review of the literature.

### 2. Case reports

#### 2.1. Patient 1

A 56-year-old woman was admitted to our hospital with deterioration of mental state for 2 days. Five days prior to admission, she complained of a headache of increasing severity. Physical examination on admission demonstrated a Glasgow Coma Scale (GCS) score of 11, an unresponsive dilated right pupil and a right hemiparesis (Grade III/V). A CT scan on admission revealed a right chronic subdural hematoma and severe midline shift to the left. There was obliteration of the basal cisterns and displacement of the brain stem (Fig. 1). An emergency right trephination was performed to remove the subdural hematoma, which was a typical chronic hematoma with an outer membrane. After the operation, the abnormal neurological signs, including deterioration of mental state, right-sided hemiparesis and unresponsive dilated right pupil immediately returned to normal. MRI obtained 7 days after the operation demonstrated that the size of the hematoma had markedly diminished. There was no abnormal signal change or deformity of the crus cerebri contralateral to the hematoma (Fig. 2).

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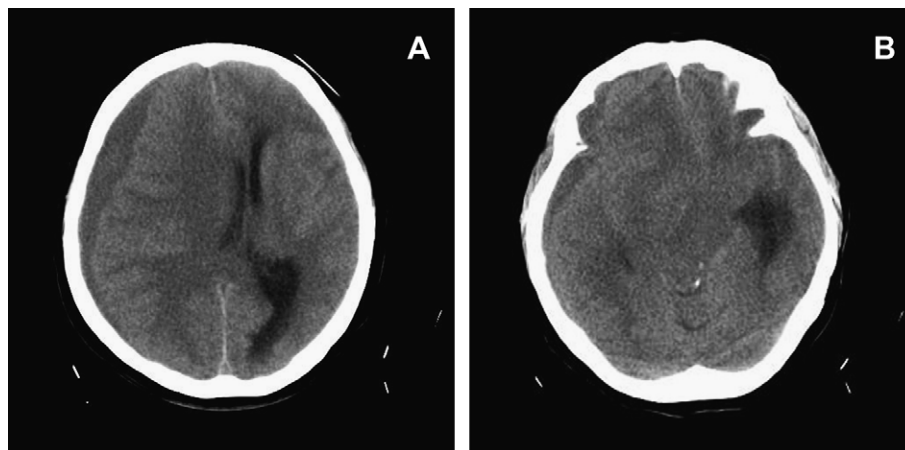


Fig. 1. Pre-operative CT scans in Patient 1 (A,B) show a chronic right-sided subdural hematoma with severe midline shift.

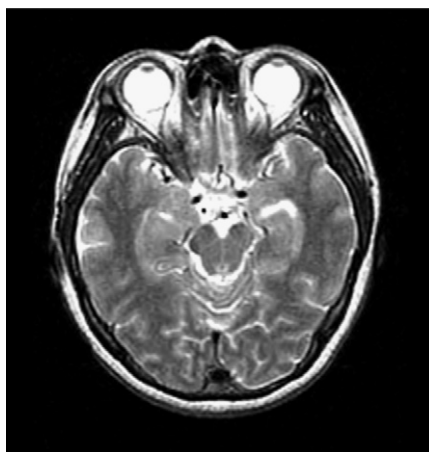


Fig. 2. Post-operative T<sub>2</sub>-weighted axial MRI in Patient 1 demonstrating the completely removed hematoma without abnormality of the cerebral peduncle.

### 2.2. Patient 2

A 70-year-old man was admitted to our hospital with a progressive decrease in his level of consciousness and exacerbation of abnormal verbal responses. Five years before this

admission, he underwent a craniotomy with clipping of aneurysm after a subarachnoid hemorrhage that originated from a right middle cerebral artery aneurysm. Physical examination revealed a GCS score of 9 and a left hemiparesis (Grade III/V). MRI revealed a left chronic subdural hematoma and mild midline shift to the right. There was a small round region in the right crus cerebri with low signal intensity on T<sub>1</sub>-weighted images, and high signal intensity on T<sub>2</sub>-weighted images (Fig. 3). An emergency left trephination was performed to remove the hematoma, which was a typical chronic subdural hematoma with an outer membrane. After the operation, his mental state gradually recovered to normal with some remaining hemiparesis (Grade III/V).

### 3. Discussion

Compression of the crus cerebri against the free edge of the tentorium caused by a contralateral mass was first described at the Mayo Clinic by Kernohan and Woltman in 1929.<sup>4</sup> Gross and microscopic evaluation of 276 brains with primary brain tumors revealed 34 cases (12%) with compression of the contralateral cerebral peduncle against the free edge of the tentorium. This led to the visible notching of the anterolateral

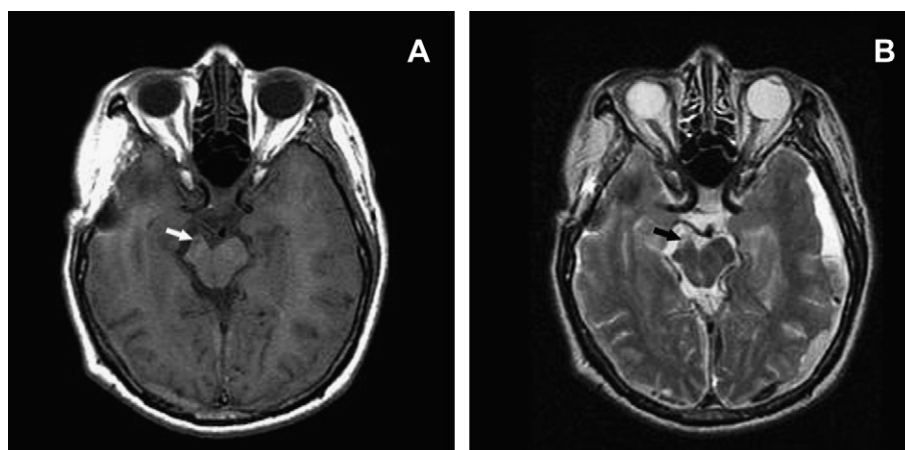


Fig. 3. Pre-operative MRI in Patient 2. (A) T<sub>1</sub> and (B) T<sub>2</sub>-weighted axial MRI showing Kernohan's notch with signal abnormality in the anterolateral portion of the right cerebral peduncle (arrow).

cerebral peduncle with resulting tissue destruction and the false lateralizing sign of hemiparesis ipsilateral to the mass lesion, due to the interruption of the descending corticospinal motor fibers in the contralateral cerebral peduncle. Although most commonly seen in this setting, several studies have also reported patients with traumatic head injury or spontaneous intracerebral accident with definitive evidence of Kernohan's notch on MRI. The majority of reported cases have been related to acute space-occupying lesions such as a subdural hematoma,<sup>5–10</sup> epidural hematoma,<sup>11,12</sup> depressed skull fracture<sup>13</sup> or spontaneous intracerebral hematoma.<sup>1,14,15</sup> Only two patients with chronic subdural hematoma<sup>2,3</sup> have been reported with MRI.

Along with traumatic injury, other possible causes of ipsilateral hemiparesis include a primary brain stem lesion, a contralateral contusion, pressure necrosis secondary to elevated intracranial pressure, or arterial infarction.<sup>16,17</sup> Especially in the setting of acute traumatic injury, it is important to distinguish Kernohan's notch phenomenon from direct brain stem injury. Although the CT scan is the gold-standard diagnostic method in evaluating head trauma, it is not effective in detecting small lesions in the brain stem. Primary traumatic brain stem lesions typically occur posterolaterally in the midbrain or upper pons on MRI and tend to produce more profound and prolonged impairment in the level of consciousness.<sup>16,17</sup> A lesion associated with Kernohan's notch phenomenon, in an acute trauma patient, represents a small round area, hypointense on T<sub>1</sub>- and hyperintense on T<sub>2</sub>-weighted images, in the anterolateral midbrain region, corresponding to the cerebral peduncle.<sup>5–13</sup> In chronic subdural hematomas, MRI reveals similar results.<sup>2</sup> However, Itoyama et al.<sup>3</sup> reported a compressed deformity of the crus cerebri without signal change that closely paralleled neurological recovery in chronic subdural hematoma. Interestingly, in our Patient 1 there was no signal change of the crus cerebri on post-operative MRI, with marked reduction in the size of the hematoma. In Patient 2, MRI revealed signal changes and deformity of the crus cerebri against the contralateral hematoma.

Neurological examination for patients with Kernohan's notch phenomenon usually reveals severe impairment of mental state, ipsilateral motor weakness and third nerve palsy. These neurological deficits may resolve after surgery, but some degree of deficit, especially motor weakness, generally remains. A reported patient with signal changes of the crus cerebri on MRI was in a deeply comatose mental state, with ipsilateral motor weakness and bilateral pupil abnormality. This patient recovered gradually with persistent hemiparesis.<sup>2</sup> The clinico-radiological course of this reported case is similar to that of our Patient 2. By contrast, a reported patient showing only compressive deformity of the crus cerebri without signal change demonstrated complete recovery after surgery.<sup>3</sup> Unfortunately, we were not able to show the serial changes of the crus cerebri in our Patient 1 because MRI before surgery was not available. However, considering the fact that there was clinically obvious Kernohan's notch phenomenon without definitive signal change on

post-operative MRI, the mechanism of Patient 1 may have been related to a simple compressive deformity of the crus cerebri. Similar to a previously reported case,<sup>3</sup> neurological symptoms in Patient 1 recovered completely after surgery.

The mechanism underlying Kernohan's notch phenomenon is not completely understood. Although considerable individual anatomic variation in the tentorial notch has been reported,<sup>18</sup> there is little evidence to support that a narrow notch predisposes to Kernohan's notch phenomenon. The vector of force exerted by the space-occupying lesion is likely to be more important. Given the chronic nature of the mass effect, horizontal shift by a lesion situated laterally has a greater likelihood of shifting the brain stem across the midline as demonstrated by Patient 1.<sup>19,20</sup>

Kernohan's notch phenomenon with permanent tissue damage in the crus cerebri, shown by signal changes on MRI, predicts persistent motor deficit. However, with the gradual onset of a chronic subdural hematoma, Kernohan's notch phenomenon may develop with only transient compression of the crus cerebri. In this situation recovery is more complete than in those patients with signal changes in the crus cerebri identified by MRI.

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## Spinal angioliipoma with acute subarachnoid hemorrhage

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### Abstract

Angioliipoma is a rare tumor of the spine commonly presenting with compressive myelopathy. We report a spinal angioliipoma in a 14-year-old patient with acute spinal subarachnoid hemorrhage (SAH). To our knowledge this is the first reported case of a spinal angioliipoma presenting with SAH, associated with post-subclavian coarctation with diffuse hypoplasia of the descending aorta. This association of coarctation of aorta, aortic hypoplasia and spinal angioliipoma has also not been reported previously.

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**Keywords:** Spinal angioliipoma; Subarachnoid hemorrhage; Coarctation of aorta; Aortic hypoplasia

### 1. Introduction

Spinal angioliipoma is a rare tumor of the spinal axis. It usually presents with features of compressive myelopathy.<sup>1</sup> Subarachnoid hemorrhage (SAH) from spinal angioliipoma has not previously been reported.

### 2. Case report

A 14-year-old boy presented with acute-onset back and nuchal pain, with opisthotonic posturing and bifrontal headache of 6 days' duration. He had mild non-progressive limping gait for the past 8 years. He had also undergone surgical correction of coarctation of aorta 2 years previously. On examination he was febrile, his blood pressure was 174/100 mmHg in the upper limbs, 190/100 mmHg in the lower limbs and he had grade II hypertensive retinopathy changes. There was severe neck rigidity, paraspinal muscle spasm and opisthotonic posturing (Fig. 1A,B). His left leg

was shorter than the right by 2 cm, with associated diffuse wasting and flexion deformity at the ankle (Fig. 1A). Asymmetric pyramidal signs (left > right) and subtle sensory impairment over the left lower limb were present. Cardiac evaluation revealed mild cardiomegaly. Blood investigations including biochemistry, renal and liver function tests and coagulation profile were normal. Lumbar puncture was performed based on the clinical suspicion of acute meningitis after a CT head scan, which was normal.

Cerebrospinal fluid (CSF) was hemorrhagic with glucose 7 mg/dL (blood glucose 105 mg/dL), protein 2590 mg/dL and 300 white cells/mm<sup>3</sup> (polymorphs 70%, lymphocytes 30%). CSF was assumed to be a 'traumatic tap' although the three samples were equally hemorrhagic. The patient was treated for pyogenic meningitis. CSF gram stain, acid-fast bacilli (AFB) and fungal stains were negative and there was no growth on bacterial and fungal cultures. A repeat CSF study was performed 5 days later, as the patient did not show adequate response to treatment. The second tap revealed xanthochromia with 400 white cells/mm<sup>3</sup> (polymorphs 2%, lymphocytes 92%), glucose 28 mg/dL (blood glucose 115 mg/dL) and protein 312 mg/dL. At this

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