

Case Reports

Kernohan-Woltman notch phenomenon caused by an acute subdural hematoma

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ABSTRACT

Uncal herniation through the tentorial notch is occasionally associated with false localizing ipsilateral hemiparesis, known as the Kernohan-Woltman notch phenomenon (KWNP). We report an 81-year-old female who presented with a decreased level of consciousness, a right mydriasis and an ipsilateral motor deficit caused by a large right hemispheric subdural hematoma that was immediately evacuated. The patient recovered well, although her right hemiplegia persisted. A follow-up MRI showed a residual lesion in the left cerebral peduncle, corresponding to KWNP. The presence of such a structural lesion suggests a poor prognosis for recovery from the initial motor deficit.

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1. Introduction

The Kernohan-Woltman notch phenomenon (KWNP) defines the paradoxical neurological deficits associated with a compressive injury of the cerebral peduncle against the edge of the tentorium due to the expansion of a contralateral supratentorial lesion. It is characterized by a hemiparesis ipsilateral to the mydriasis observed in patients with altered levels of consciousness.¹ The presence of false localizing neurological signs can preclude a correct topographical diagnosis of the associated pathological lesions within the central nervous system, leading to surgical treatment of the wrong site.² However, modern neuroradiological methods facilitate the recognition of the structural lesions of the nervous system as well as the pathophysiological mechanisms underlying this paradoxical phenomenon.^{3,4} We report a patient with KWNP caused by an acute subdural hemorrhage, providing CT scan and MRI evidence of the structural midbrain lesion located within the contralateral cerebral peduncle.

2. Case report

An 81-year-old female received a medical consultation after suffering an accidental minor head injury. She had been treated with anticoagulants because of a chronic atrial fibrillation. While in the emergency room, the patient suffered a sudden deterioration of her level of consciousness to the point of coma (Glasgow Coma Scale of 7/15), and showed a right mydriasis and a complete right motor deficit on the neurological examination. A CT scan demon-

strated a large right hemispheric acute subdural hemorrhage (Fig. 1). The clot was immediately evacuated through a right frontoparietotemporal craniotomy. Postoperatively, the patient had no complications. She gradually regained normal consciousness and a normal pupillary function. Nevertheless, the paradoxical right hemiplegia persisted. Postoperative CT and MRI scans were obtained after 4 weeks, and demonstrated a structural lesion within the left cerebral peduncle, probably caused by compression against the free tentorial edge (Figs. 2, 3). The motor deficit was moderately improved after 3 months.

3. Discussion

The tentorial incisura is located between the tentorial edges and communicates the supratentorial and infratentorial spaces. This area can be divided into three spaces: anterior, middle (lateral to), and posterior to the brainstem. The middle incisural space is close to the midbrain and the upper pons at the level of the pontomesencephalic sulcus. Mesial temporal lobe structures such as the uncus, the parahippocampal gyrus and the hippocampal formation, are also intimately related to the incisura. The principal vascular structures coursing along the middle incisural space are the posterior cerebral artery and the superior cerebellar artery which pass around the brainstem, parallel to the free tentorial edge. The incisura has also a close relationship with the first 6 cranial nerves.⁵

Symptoms associated with tentorial herniation may result from displacement, compression and stretching of the brainstem and cranial nerves, and/or hemorrhage and infarction caused by compression and tearing of arteries and veins coursing along the tentorial incisura. A downward shift of mesial temporal lobe structures

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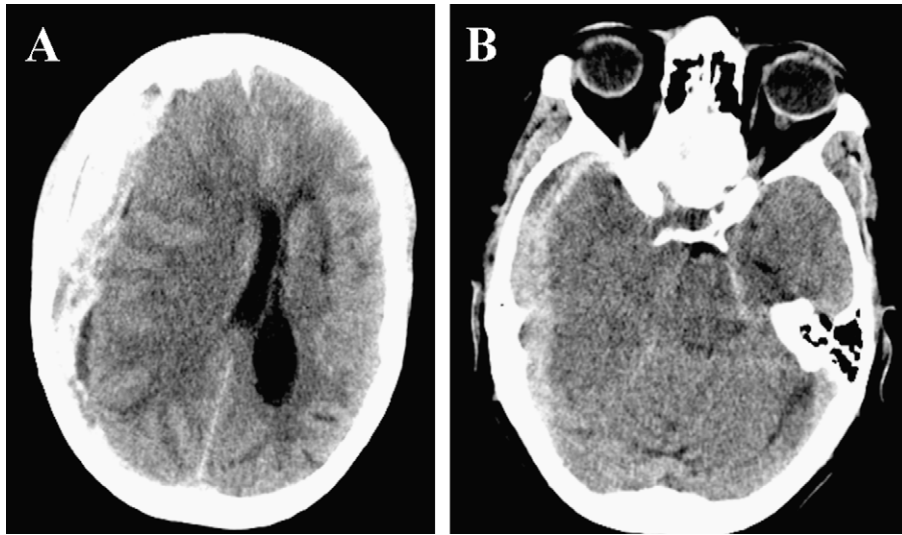


Fig. 1. Axial brain CT scans showing (A) an extensive right hemispheric acute subdural hematoma, causing a 2 cm midline shift and (B) a right uncal herniation with deformation of both cerebral peduncles and obliteration of the perimesencephalic cisterns.

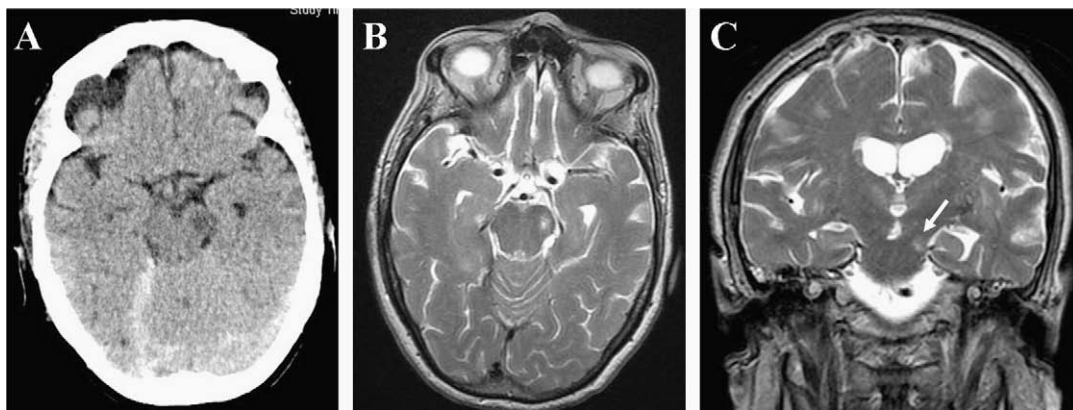


Fig. 2. (A) Axial brain CT scan showing brain re-expansion after surgical evacuation of the subdural hematoma. A well-defined rounded hypointense signal is evident within the anterolateral left midbrain. T2-weighted MRI showing a hyperintense signal in both (B) axial and (C) coronal slices (white arrow).

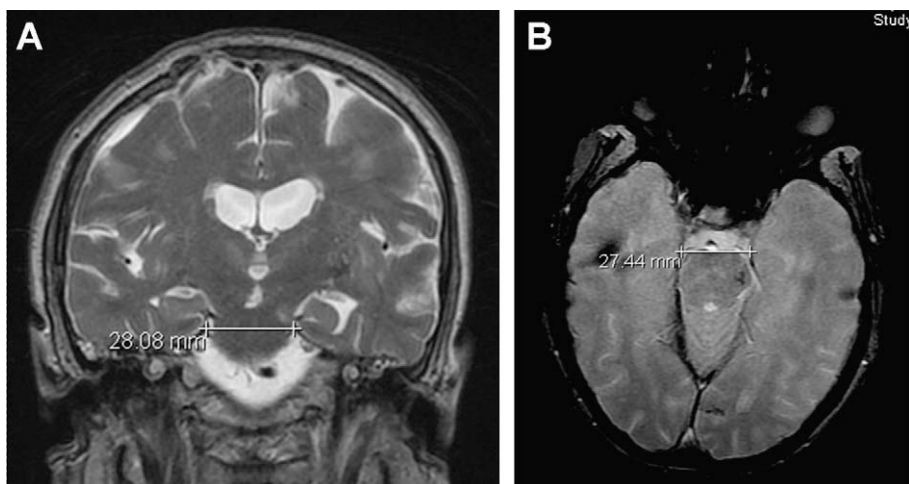


Fig. 3. Maximum notch width measurements on (A) postoperative coronal T2-weighted MRI and (B) axial T2-weighted gradient-echo MRI sections. The distance measured between the free tentorial edges was less than the mean value (29.6 mm) and near the first quartile value (27.0 mm) of Adler and Milhorat's morphometric tentorial notch analysis based on 100 human autopsies.¹⁴ A slight discrepancy of less than 1.0 mm was measured between the axial and coronal slices. In the gradient-echo MRI a linear hypointense signal can be observed at the lowest border of the peduncular lesion, indicating a paramagnetic effect probably due to iron deposition.

is typical of tentorial herniation, resulting in kinking and grooving of the uncus and parahippocampal gyrus. Compression of the ipsilateral cerebral peduncle causes contralateral pyramidal signs and, if the lateral displacement of the brainstem is severe, the contralateral cerebral peduncle may be forced against the free edge, thus producing a groove on the peduncle called a Kernohan's notch, with ipsilateral pyramidal signs.^{5,6} Hemorrhage into the brainstem as a result of tearing of arteries and veins without cerebral herniation may occur if the incisura hugs the brainstem so tightly that it prevents cerebral herniation while allowing axial displacement of the brainstem.⁵

The notion of "false localizing signs" refers to neurological signs that reflect a dysfunction that is remote from the expected anatomical site of pathology.² Collier first reported the frequency of these phenomena to be as high as 12.4% in his own series, with most occurring in patients with raised intracranial pressure due to supratentorial masses.^{7–9} However, prior to the availability of CT technology, a wide range of variation in the percentage of patients demonstrating false localizing signs was reported, ranging from 7 to 50% of patients.^{10,11} In particular, the finding of mydriasis and ipsilateral hemiparesis in patients harbouring a supratentorial expanding mass led clinicians to a topographical misdiagnosis and, in many cases, to surgical treatment of the wrong side.¹² Kernohan and Woltman first described the correlation between this paradoxical ipsilateral motor deficit and the groove deformation and/or tissue destruction observed in the contralateral cerebral peduncle.¹

Pathological mechanisms by which KWNP is produced, whether by a brisk displacement of diencephalic structures or by the pressure exerted on the brainstem by the herniated uncus, still remain unclear.¹³ Several factors, mainly the growing rate of the supratentorial mass, the degree of lateral displacement of the brainstem and the individual width of the tentorial incisura, may contribute to the development of KWNP, but there is still little evidence to support these hypotheses.^{3,14,15} The morphometric analysis of the tentorial notch variations performed by Adler and Milhorat in 100 autopsies has classified the maximum notch width in narrow (24.5–27.0 mm), typical (27.1–31.9 mm) and wide (32.0–39.0 mm) categories.¹⁴ Among the different measurements considered for the tentorial notch in this study, we believe the maximum notch width may represent a fundamental anatomical factor predisposing patients who suffer from uncal herniation to KWNP. In our patient, the maximum notch width was determined by postoperative MRI, yielding a value close to the first quartile (27.0 mm) and far from the maximum width (39.0 mm) found on necropsy specimens (Fig. 3).¹⁴ Although no definite conclusions can be drawn as to the effects of this parameter based on this observation, our finding suggests that individual anatomical variations of the tentorial incisura can influence the development of KWNP.

Neuroradiological evidence of KWNP obtained with CT and MRI technologies is scarce.^{16–18} KWNP has been mostly observed in adult patients with traumatic intracranial hematomas, but it has also been reported in both elderly and young patients.^{15,19,3} For patients with a severe traumatic brain injury, it is essential to distinguish KWNP from other possible causes of ipsilateral motor weakness such as contralateral cortical damage, arterial infarction, primary brainstem lesion and spinal cord injury.^{17,3}

Morphological changes associated with KWNP may correspond to either: (i) a reversible indentation of the cerebral peduncle caused by midbrain compression against the free tentorial edge; or (ii) irreversible tissue destruction of the adjacent corticospinal tract.^{1,20} MRI studies performed in awake patients with gradually expanding lesions usually show an indentation deformity in the contralateral cerebral peduncle as the unique morphological change associated with KWNP.^{19,3} This early mor-

phological deformation, similar to that observed in necropsy studies by Kernohan and Woltman, could theoretically be associated with a better functional motor outcome given the potential reversibility of such elastic deformation.^{1,3} However, most MRI studies investigating KWNP have been performed after the evacuation of intracranial hematomas in patients with critical head injuries.^{4,3} In these patients, the initial brainstem deformities have usually reverted. Nevertheless, a residual rounded lesion in the cerebral peduncle, that is typically hypodense on a CT scan, hypointense or isointense on T1-weighted MRI, and hyperintense on T2-weighted MRI and fluid-attenuated inversion recovery-MRI, can be observed.^{16,17} This lesion represents a degeneration of myelinated fibres within the corticospinal tract, caused by a sustained midbrain compression against the free tentorial edge, and may predict a poor functional outcome.^{3,20} In our patient, a gradient-echo MRI axial slice at the level of the pontomesencephalic union showed a hypointense linear signal located at the inferior limit of the lesion involving the corticospinal tract (Fig. 3B). To our knowledge, this is the first gradient-echo MRI performed in a patient with KWNP and is indicative of a paramagnetic effect caused by iron, probably related to hemosiderin deposition.²¹ This signal may correspond to a small peripheral rim of blood extravasation as first reported by Kernohan and Woltman, who described the presence of previous or recent petechial hemorrhages at the peripheral borders of the mechanical corticospinal lesion associated with the deepest brainstem grooves.¹

This signal must be differentiated from other brainstem hemorrhagic lesions, namely Duret hemorrhages, which can develop secondarily to a descending tentorial herniation of any cause. Duret hemorrhages usually arise in the midline of the lower mesencephalon and upper pons and are thought to be caused by the stretching and spasm of perforating brainstem arteries, and are associated with a high mortality.²² Other types of traumatic brainstem hemorrhages, such as primary shearing petechiae caused by rotational forces applied to the brainstem, are usually found in the posterolateral quadrants and not within the peduncles.²³ Spontaneous hypertensive hemorrhages are also usually located in the dorsal region at the caudal pons.²³

4. Conclusion

Mechanical lesions of the brainstem associated with KWNP are caused by a sustained notching of the nervous tissue against the tentorial edge and present a specific pathological change that must be differentiated from other brainstem lesions caused by tentorial herniation. KWNP was initially related to the final stages of supratentorial tumors but here is reported in patients harbouring supratentorial traumatic hemorrhages. CT and MRI scans can show the focal brainstem lesion that explains the presence of the false localizing hemiplegia.

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Very late thrombosis in a patient with a drug-eluting stent for intracranial atherosclerotic stenosis

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ABSTRACT

Late stent thrombosis is an infrequent, but severe, complication after implantation of a drug-eluting stent. We report one such patient, a 69-year-old man with intracranial vertebral artery stenosis. He experienced very late stent thrombosis after discontinuation of clopidogrel therapy, about 16 months after the insertion of a Taxus Stent® (Boston Scientific Corporation, Natick, MA, USA). The patient recovered well, with good clinical outcome, after the re-initiation of dual antiplatelet and anticoagulation therapy. No abnormality within the stent was identified on a repeat angiogram taken 4 weeks after treatment re-initiation.

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1. Introduction

Intracranial atherosclerotic disease is a common cause of stroke, particularly in the Asian population.¹ Preliminary observational studies have suggested that combined angioplasty and stenting may reduce the risk of stroke in patients who have symptomatic intracranial stenosis.^{2,3} In-stent restenosis is also known to be a key factor influencing the effect of intracranial stenting. Based on the successful use of the technique in cardiology, some investigators have used drug-eluting stents (DESs) for the treatment of intracranial atherosclerotic stenosis.^{4,5} Stent thrombosis is an infrequent, but severe, complication that can occur after implantation of a DES. We present a patient with a very late thrombosis after discontinuation of clopidogrel therapy, about 16 months after the insertion of a paclitaxel-eluting coronary stent (Taxus Stent®, Boston Scientific Corporation, Natick, MA, USA).

2. Case report

A 69-year-old man with a history of systemic hypertension and diabetes mellitus presented to our facility with recurrent dizziness and diplopia. MRI performed at the time of admission showed infarction of the right cerebellar hemisphere. Cerebral angiography demonstrated a 60% stenosis of the right vertebral artery (VA) (Fig. 1A) and total occlusion of the left VA. The posterior communicating arteries were not shown on digital subtraction angiography (DSA). We decided to perform percutaneous transluminal angioplasty and stenting (PTAS) to prevent further progression of stroke. Aspirin and clopidogrel were administered for 7 days before the procedure.

The PTAS procedure was performed under general anesthesia. A 6 French guiding catheter (Envoy, Endovascular Corporation, Miami, FL, USA) was positioned in the right distal VA. The stenotic segment was crossed with a Transend microwire (Boston Scientific Corporation). A Taxus stent (4.5 mm in diameter, 16 mm in length) was gently advanced over the microwire. However, the delivery of

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