

Kernohan-Woltman Notch Phenomenon—Case Report

Fenômeno do entalhe de Kernohan-Woltman—relato de caso

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Abstract

Keywords

- ▶ cerebral peduncle
- ▶ head injury
- ▶ Kernohan-Woltman notch phenomenon
- ▶ transtentorial herniation

Resumo

Palavras-chave

- ▶ pedúnculo cerebral
- ▶ traumatismo cranioencefálico
- ▶ fenômeno do entalhe de Kernohan-Woltman
- ▶ herniação transtentorial

The Kernohan-Woltman notch phenomenon is a paradoxical neurological manifestation consisting of a motor deficit ipsilateral to a primary brain injury. It has been observed in patients with brain tumors and with supratentorial hematomas. It is considered a false localizing neurological sign. Magnetic resonance imaging (MRI) scan has been the test of choice. The recognition of this phenomenon is important to prevent a surgical procedure on the opposite side of the lesion. The present case report describes a case of chronic subdural hematoma with a probable finding of the Kernohan-Woltman phenomenon, and it discusses its pathophysiology, imaging findings, treatment, and prognosis.

O fenômeno do entalhe de Kernohan-Woltman é uma manifestação neurológica paradoxal que consiste em um déficit motor homolateral à lesão cerebral primária. Este fenômeno tem sido observado em casos de neoplasia cerebral e de hematomas supratentoriais, e é considerado um sinal de falsa localização neurológica. Ressonância magnética tem sido o exame de escolha. É importante o reconhecimento deste fenômeno para evitar um procedimento cirúrgico no lado oposto à lesão. O presente relato de caso apresenta um caso de hematoma subdural crônico, com provável achado do fenômeno de Kernohan-Woltman, e discute sua fisiopatologia, seu achado de imagem, seu tratamento e prognóstico.

Introduction

The Kernohan-Woltman phenomenon or Kernohan-Woltman notch signal is a hemiparesis ipsilateral to a supratentorial brain injury^{1–5} in a brain tumor patient. The ipsilateral hemiparesis is secondary to a transtentorial herniation due to a midline deviation at the level of the midbrain, resulting in the compression of the contralateral cerebellar tentorium pyramidal fibers.² This leads to partial or total damage of these pyramidal fibers as they course

the spinal cord to innervate the other side of the body. Injuries in these fibers cause a hemiparesis ipsilateral to the brain lesion, constituting a false localizing neurological sign.^{2,5,7}

The present case report describes a probable case of Kernohan-Woltman phenomenon diagnosed through neurological examination and computed tomography (CT) scanning in a patient with a chronic subdural hematoma, as well as the physiopathology, diagnosis and prognosis of the condition.

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Case Report

A male patient, J.J.B., 59 years old, rural worker, presented with a history of mild head trauma 40 days before. The patient evolved with a moderate headache that was relieved with simple painkillers. For 72 hours, the patient had been presenting with a decrease in the level of consciousness, as well as decreased movements on the left side of the body and generalized seizures. The physical examination determined that the patient was in a regular general condition. The patient had systemic arterial hypertension (SAH), with no proper diet, but with sporadic use of specific medication. His blood pressure was 170×110 mm Hg. The neurological examination detected a decreased level of consciousness. At admission, the electrocardiogram (EKG) score was 10. The patient presented with left hemiparesis with crural predominance. The left pupil could not be viewed (**►Fig. 1**), and the right pupil measured 3.0 mm and presented little reaction to light stimuli. A CT of the head with no contrast medium revealed the absence of the left eye, as well as the presence of a double-density chronic subdural hematoma located in the left frontoparietal region with mass effect, compressing the ipsilateral brainstem, obliterating the homolateral ventricle and deviating midline structures (**►Figs. 2 and 3**). The patient was submitted to a left posterior parietal trepanopuncture and to drainage of the hematoma. Forty-eight hours after the surgical procedure, the patient was awake, with EKG = 13 and improvement of the left-sided muscle strength deficit. The seizures were controlled with hydantoin. The patient was discharged at the 6th postoperative day, with orientation for outpatient return.

Discussion

Hemiparesis ipsilateral to a brain injury is called Kernohan-Woltman⁶ notch sign, and it is considered a false localizing neurological sign. The Kernohan-Woltman phenomenon was initially described by Kernohan et al in 1928, in a necropsy examination of a brain tumor patient with a notched brain peduncle from a contralateral herniation.⁸ The condition has been associated with brain tumors and supratentorial-located hematomas.⁵



Fig. 1 Absence of the left eye (enophthalmos).

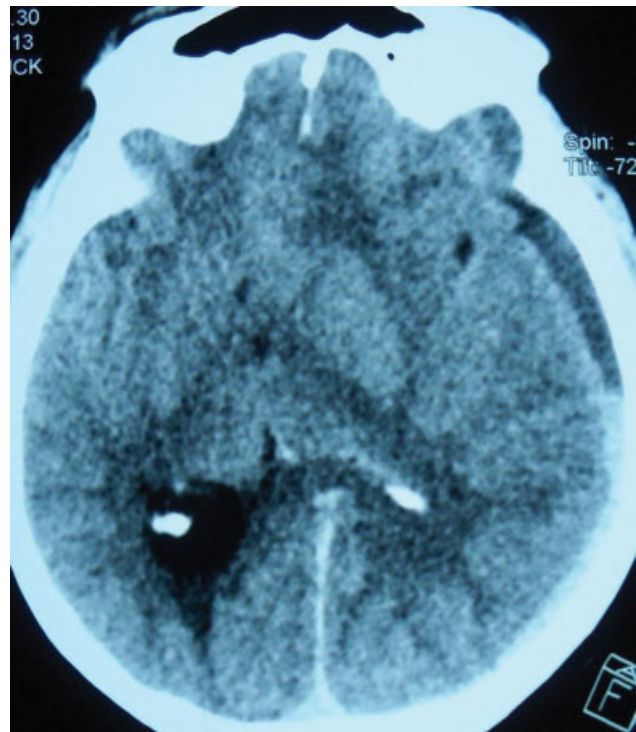


Fig. 2 Axial head computed tomography without contrast medium showing a mass effect, double-density chronic subdural hematoma located in the left cerebral hemisphere.



Fig. 3 Axial head computed tomography without contrast medium showing a chronic subdural hematoma in the left cerebral hemisphere and obliteration of the homolateral ventricle with obliteration of the interpeduncular cistern.

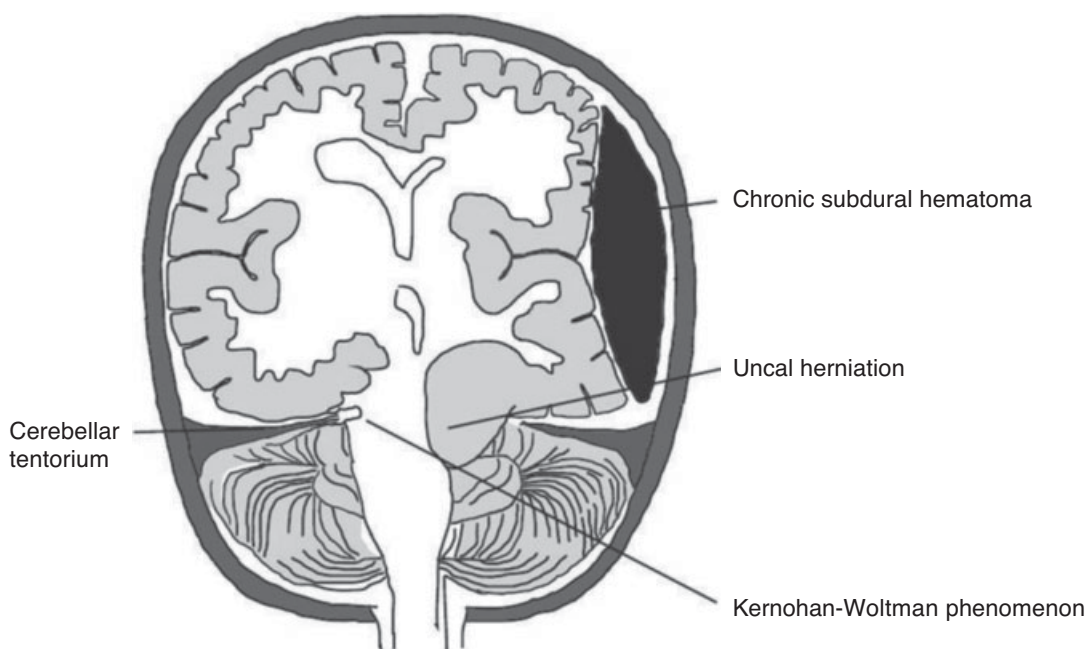


Fig. 4 Schematic representation of the Kernohan-Woltman notch phenomenon.

Transtentorial herniation is a neurological phenomenon well described in the medical literature, and it is associated with a supratentorial expansive lesion. This herniation causes direct neural compression and indirect effects of vascular compromise, leading to obstructive hydrocephalus and herniation of the strangled tissue.¹

Uncal herniation is a common subtype of transtentorial herniation, in which the most internal part of the temporal lobe, the uncus, can be compressed against the tentorium, forming a recess at the crus cerebri known as Kernohan-Woltman notch and compressing the brainstem, mainly the midbrain.^{2,5,8} With time, the integrity of the crus cerebri (the frontal aspect of the cerebral peduncle) and of the corticospinal descending tract is compromised, causing contralateral motor deficit associated with a decreased level of consciousness.

The Kernohan-Woltman notch signal is a rare condition in which the peduncle contralateral to a supratentorial expansive lesion is forced against the end of the tentorium, leading to hemiparesis ipsilateral to the lesion (→ **Fig. 4**). The rigid edge of the tentorium can transect the brain peduncle, particularly the fibers protruding to the leg. The lesion can occur in the absence of an uncal herniation, probably due to a rapid acceleration-deacceleration leading to a backlash injury of the cerebral peduncle.¹ Therefore, the hemiparesis ipsilateral to a supratentorial expansive lesion is known as Kernohan-Woltman phenomenon. This phenomenon is considered a false localizing neurological sign.

The diagnosis of the Kernohan-Woltman phenomenon is based on neurological examination and on neuroimaging findings.^{2,8} The neurological condition is characterized by mydriasis and hemiparesis, and it may be associated with a decreased level of consciousness.⁴

A CT scan can demonstrate the effect of an expansive mass lesion compressing the brainstem, while the identification of

an uncal herniation is more difficult.⁵ A resonance magnetic imaging (MRI) exam has better definition in multiple planes and better resolution to evaluate the brain stem, being able to reveal a deformity or injury in the cerebral peduncle resulting from a transtentorial herniation.⁸⁻¹⁰ The Kernohan-Woltman phenomenon has been identified in coronal T2-weighted and fluid attenuation inversion recovery (FLAIR) sequences, and it tends to present a peripheral triangular morphology.^{1,8} According to Moon et al,¹¹ the lesion is a small, hypointense signal in T1-weighted images, with a hyperintense signal in T2-weighted images, in the anterolateral region of the midbrain.

In the present case, due to the clinical conditions of the patient in the emergency room, only one CT scan was performed, but the neurological picture was consistent with the Kernohan-Woltman notch signal.

Patients who present with the Kernohan-Woltman notch phenomenon are surgically treated; however, in severe cases, hyperventilation, administration of osmotic diuretics, and general care can be performed, not always with a good prognosis.

The Kernohan-Woltman notch signal has been considered a rare finding. Magnetic resonance imaging scans have been used to confirm the diagnosis. The present case serves as a warning for emergency physicians to be aware of its severity and of the fact that it may not always be possible to identify this phenomenon in CT scans as a false localizing neurological sign in order to avoid an incorrect surgical approach.

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