False Localizing Sign: Kernohan’s Syndrome Due to Right Temporal Meningioma

Sinal Falso Localizatório: Síndrome de Kernohan Causada por Meningioma Temporal Direito

ABSTRACT
The Kernohan syndrome results from the compression of the cerebral peduncle against the tentorium cerebelli. This phenomenon represents a relevant clinical sign of transtentorial herniation due to an ipsilateral expansive lesion. We reported a case of a 50-year-old woman with a right temporal meningioma who developed a false localizing hemiparesis, which improved after microsurgical resection. This case emphasizes the mechanism and rarity of this pathology.

Key words: Kernohan syndrome; Meningioma; False localizing sign; Case report

INTRODUCTION
Neurological signs are described as false localizing when they reflect deficits distant from the expected anatomical region of injury, making diagnosis a challenge¹. In 1929, Kernohan and Woltman, in an autopsy study, described a case of a 47-year-old man with frontotemporal brain tumor who presented with left hemiparesis². Such a finding came to be known as Kernohan’s syndrome.

CASE REPORT
A 50-year-old female patient presented with a history of right progressive hemiparesis, and brachial predominance for about 6 months. In addition, she reported a constant headache, with periods of exacerbation associated with emetic episodes. Upon neurological examination, she presented grade 4 strength in the right upper limb and 4+ in the right lower limb according to the modified scale of the Medical Research Council (mMRC) and discrete right hemibody hyperreflexia, with no other signs of pyramidal release. No other abnormalities.

The patient was submitted to magnetic resonance imaging (MRI) of the brain that showed a cystic-solid extra axial lesion in the right temporal fossa with intense enhancement in the contrasted phases with significant perilesional edema and important deviation of midline structures, compressing the midbrain on the contralateral border of the tentorium (Fig. 1, Fig. 2, Fig. 3). Based on radiological findings and considering that the deficits were ipsilateral to the lesion, the presence of the Kernohan-Woltman phenomenon was verified.
A right pterional craniotomy and complete resection of the lesion were performed by microsurgical technique. To avoid excessive parenchyma retraction and preservation of venous structures, tumor debulking and subsequent resection were performed. In the immediate postoperative period the patient evolved with a significant improvement of hemiparesis. At the postoperative CT, the presence of residual lesion was not evidenced. Histopathological study of the lesion showed a meningothelial meningioma. On the 4th postoperative day the patient was discharged home on phenytoin 100mg every 8 hours. The clinical and radiological follow-up of 6 months demonstrated complete resolution of the disease.
Discussion

Ipsilateral hemiparesis – whether accompanied by ipsilateral mydriasis or not - related to supratentorial brain pathology is a rare presentation described in 1929 by Kernohan and Woltman, and since then has been known as Kernohan syndrome. Postulated mechanism of this phenomenon is the compression of the cerebral peduncle on the free border of the contralateral tentorium caused by a hernia of the medial part of the temporal lobe (uncus). Although the phenomenon has been described by Kernohan and Woltman as a tumor causing a lesion on the cerebral peduncle, it has not been considered the most common etiology of the syndrome. This phenomenon is mainly associated with injuries of traumatic etiology such as epidural hematomas and subdural collections. Furthermore, as a rare etiology, it was found an astrocytic tumor, a reabsorption bone syndrome and an arachnoid cyst. Gassel, investigating the presentation of false localized signs, analyzed 250 meningiomas and found that 3.6 percent of the patients had ipsilateral pyramidal signs. However, little has been described to conclude that these cases manifested as Kernohan syndrome. In our review, we did not find other cases of meningioma related to the Kernohan phenomenon.

The exact mechanism of how compression to the cerebral peduncle causes the motor deficit is not fully known. However, Carrasco et al., based on MRI and computed tomography (CT) studies, reported that direct compression of the cerebral peduncle on the tentorium would lead to a degeneration of the myelinated fibers of the corticospinal tract, which would provoke the motor signs and symptoms of the syndrome. The ipsilateral mydriasis, on the other hand, would occur as a direct result of the compression of the uncus on the oculomotor nerve, which is easily explained by the anatomical arrangement of its fibers, considering that the parasympathetic fascicle, regulator of the pupil sphincter muscle, is more superficially disposed in relation to the motor fibers.

Authors have suggested that some people would be more susceptible to develop the syndrome due to anatomical factors. Adler and Milhorat, in an autopsy study, analyzed the morphology of the tentorium of 100 individuals and found anatomical differences in the diameter of the notch of the tentorium that varied from 24.5 to 39.0 mm. Based on this data, it is possible to speculate that people with smaller diameter of this structure would tend to herniate, as well as to generate compression of the contralateral cerebral peduncle more easily when compared to those with larger diameter.

Because this syndrome manifests itself as a false localization sign, it is very important that in the initial evaluation the exclusion of other pathologies that could be present mainly with ipsilateral hemiparesis, such as contralateral cortical lesion and spinal cord injury, should be performed.

Zhang et al., in a review study, analyzed 39 case reports of Kernohan–Woltman notch phenomenon and founded that 30.8 percent achieved total deficit resolution, 35.9 percent were still improving their deficit, 10.6 percent remained with some residual deficit and 23.1 percent did not have the outcome reported. As the great majority of related cases are due to traumatic injuries, the factors that lead to a worse prognosis are probably the same as those of the lesions. Advanced age, low Glasgow Coma Scale (GCS) scores, pupillary abnormalities, midline deviation > 5 mm and obliteration of the basal cisterns.

Conclusion

In our review, we found that Kernohan’s syndrome is a very rare phenomenon with predominantly traumatic etiology. With regard to cases of neoplastic origin, we found only one report, which was an astrocytoma. False localizing signs, certainly, have challenged many experts a few decades ago. However, in the current era (of neuroimaging), prior knowledge about the etiology, as well as the pathophysiology of these signs are not only facilitators, but also fundamental for a correct diagnosis.

References


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Os autores referem que não existem conflitos de interesse. Relato de caso organizado conforme CARE guideline (consensus-based clinical case reporting)