

Kernohan-Woltman Notch Phenomenon Related to Chronic Subdural Hematoma: A Case Report

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Abstract

Kernohan-Woltman notch phenomenon defines the neurological picture caused by a compressive injury of the cerebral peduncles at the tentorial edge due to the contralateral supratentorial lesion and leads to ipsilateral hemiparesis or hemiplegia and mydriasis. We have represented this case of Kernohan-Woltman notch phenomenon that developed secondary to chronic subdural hematoma in a 88-year-old patient as a very rarely reported case. We conclude that advanced brain atrophy contributes to the formation of Kernohan-Woltman notch phenomenon resulted from chronic subdural hematoma. Additionally, investigation of cerebral peduncles via magnetic resonance imaging is important in determination of the clinical picture and evaluation of the response to the treatment in such cases.

Keywords: Kernohan-Woltman notch phenomenon; Chronic subdural hematoma; Cerebral peduncle; Tentorial edge

Introduction

Kernohan-Woltman notch phenomenon (KWNP) is defined

as compression of the cerebral peduncle against the tentorial edge caused by the displacement of the brain tissue at the ipsilateral side of the paradoxically supratentorial-localized lesion such that it may lead to hemiparesis or hemiplegia and mydriasis [1]. This phenomenon is usually reported secondary to brain tumours and severe head injuries [2]. The development of KWNP secondary to chronic subdural hematomas has been rarely reported. Although, there are various etiological factors, compression of cerebral peduncles at the tentorial edge is the essential mechanism in development of this phenomenon. We have discussed this case of KWNP secondary to chronic subdural hematoma (CSH) with its clinical and radiological findings in the view of literature.

A 88-year-old male patient, that was operated for right-sided chronic subdural hematoma 3 months ago, has reappeared our clinic with complaints of headache and weakness on his right-side. Glasgow Coma Score was 14 in his neurological examination. The case had dysphasia, mydriasis on his right-pupil and right hemiparesis. Brain Computed Tomography (CT) showed recurrence of right frontoparietal CSH and an evident shift to left side of brain (Fig. 1). When compared with right side of basilar cisterns and cerebral peduncle, Magnetic Resonance Imaging (MRI) revealed an

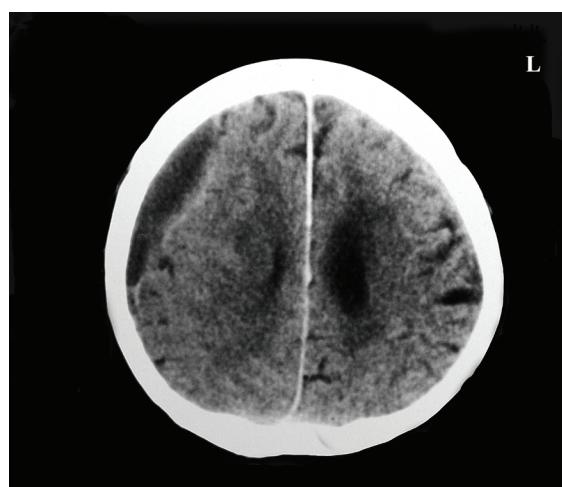


Figure 1. Brain CT showing CSH at right frontoparietal and an evident shift to left side of brain.

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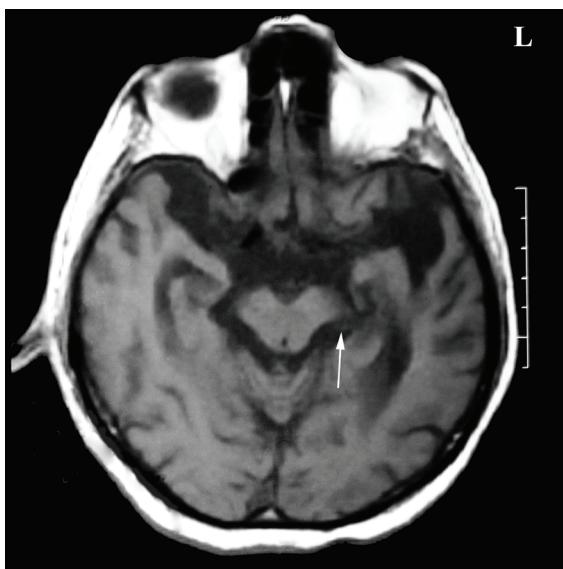


Figure 2. MRI showing compression and a narrowing of the left cerebral peduncle at the tentorial edge when compared with right side.

obliteration in the basilar cisterns and a narrowed distance between left cerebral peduncle and tentorial edge at the left side secondary to subdural hematoma (Fig. 2). Additionally, MRI showed no different pathology such as cerebral ischemic attack that may be associated with present neurological picture. For treatment of CSH, burr hole defects of previous operation were widened to remove hematoma and a drain was placed into the subdural distance. The neurological picture of the patient was completely recovered in the early postoperative period.

Discussion

Kernohan and Waltman have described at 1929 that any supratentorial-localized mass may compress the contralateral cerebral peduncles at the tentorial edge [3]. It has been reported that injury of corticospinal motor neurons in the contralateral cerebral peduncles may cause the ipsilateralisation in the course of KWNP [4]. As though, there is no comprehensive clinical trial concerned with the incidence of KWNP, nevertheless, there is limited number of case of KWNP secondary to CSH [4-6].

Chronic subdural hematoma is a frequent situation following head traumas in usually old patients. Traumas easily cause injury of bridging-venous structures of the brain in the old patients because of the decreased volume of brain tissue. A progressive hematoma may develop even following a mild head trauma. The brain atrophy of the old people also causes easier displacements of the central brain structures due to any space-occupying lesion at the supratentorial distance (hematoma, tumour etc). Our case had remarkable

brain atrophy depending on his age. The displacement of the mid-line structures was monitored. When the mechanism of Kernohan-Woltman notch phenomenon was considered, we conclude that remarkable brain atrophy of our case was a facilitating factor in development of KWNP secondary to wide subdural hematoma.

The clinical evaluation is the gold standard in diagnosis of Kernohan-Woltman notch phenomenon. Although, presence of lateralization findings and the lesion at the same-side and non-existence of another accompanying pathology that may lead to KWNP are efficient for diagnosis, it is critically important to investigate the signal changes through the cerebral peduncles and compression of basilar cisterns by MRI. Moon et al have reported that there is a correlation between the signal changes through cerebral peduncles and recovery of neurological deficits occurring in the course of KWNP and that cases without any signal change showed a much faster and stronger recovery [4]. Itoyama et al have evaluated the cerebral peduncles in case with CSH pre- and post-operatively and stated that hemiparesis of the patient recovered secondary to the improvement of the deformity in the cerebral peduncles during the postoperative period [5]. In our case, no signal change was monitored although a compression was encountered in the cistern surrounding the peduncle by MRI. The completely recovery of neurological picture of our case in the early postoperative period supported the reports of Moon et al [4].

Conclusion

CSH may rarely cause KWNP in the cases with advanced age and remarkable brain atrophy. The investigation of cerebral peduncles via preoperative MRI would provide an important contribution in the definition of the neurological deficits and evaluation of the treatment outcomes in such cases.

References

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