



Kernohan-Woltman Notch Phenomenon in Chronic Subdural Hematoma: An under-Diagnosed Phenomenon?

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Authors' contributions

This work was carried out in collaboration among all authors. Authors MAB and SY compiled patients' case notes from their electronic records and literature search. Author BABA did the literature search and put together the manuscript. Authors EOO and PKM did a search for relevant literature. Author KAM ensured the neurosurgical facts presented in the manuscript were accurate. Author GAR did the entire proofreading and editing of the manuscript. Author KME reported on the brain scans and compiled patients' imaging from their electronic records. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Introduction: Kernohan-Woltman notch phenomenon is a neurological picture of mydriasis and hemiparesis/ hemiplegia ipsilateral to a supratentorial mass lesion causing compression of the contralateral cerebral peduncle against the tentorial edge. The aim of this paper is to

report series of cases from a low volume centre of neurosurgical care and highlight the fact that Kernohan's notch phenomenon, although, reported to be quite rare but it's not uncommon and to also look out for this phenomenon to avoid wrong site surgeries.

Presentation of Cases: We report four cases of chronic subdural hematoma presenting with Kernohan- Woltmann notch phenomenon. The patients include: a young alcoholic who was found in a gutter after binge drinking, a middle aged man who was accidentally hit on the head with a car tire jack, an elderly female with no history of trauma, a fall nor use of anticoagulant and an elderly male, a diabetic. All four patients had emergency burr hole and drainage of subdural hematoma.

Discussion: This incidence of this phenomenon among patients with chronic subdural hematoma is rarely reported in the literature, however, a low volume centre for neurosurgical services like ours has seen five cases in a short period of time.

Conclusion: This paradoxical neurological sign is probably under-diagnosed judging from the number of cases diagnosed in a low volume center like ours.

Keywords: Kernohan-Woltman notch; Kernohan's phenomenon; false localization; transtentorial herniation; cerebral peduncle; chronic subdural hematoma; tentorial edge.

1. INTRODUCTION

Kernohan-Woltman notch phenomenon (KWNP) is a neurological picture of hemiparesis/hemiplegia and mydriasis which is ipsilateral to a supratentorial mass lesion causing compression of the contralateral cerebral peduncle against the free tentorial edge [1]. Patients with brain tumours and severe head injuries have usually been reported to have this phenomenon [2]. Kernohan's notch phenomenon resulting from chronic subdural hematoma has barely been reported.

The authors reported a single case of KWNP in a 50 year old male with chronic subdural hematoma about two years ago [3].

According Zhang et al in a review article of thirty nine cases, thirty six cases were as a result of intracranial bleeds with the rest resulting from an arachnoid cyst, a high grade glioma and a reabsorption bone syndrome [4]. Although, the landmark paper by Kernohan and Woltman was in a case of a brain tumour, subsequent cases with similar clinical presentations have been as a result of different pathologies [4]. as shown in the above review paper.

There are single case reports of Kernohan's notch phenomenon due to chronic subdural hematoma reported in the English literature. The aim of this paper is to report series of cases from a low volume centre of neurosurgical care and highlight the fact that Kernohan's notch phenomenon, although, reported to be quite rare but it's not uncommon

and to also look out for this phenomenon to avoid wrong site surgeries.

Also, a search from the English literature revealed these four cases as the highest number of Kernohan's notch phenomenon reported as a result of chronic subdural hematoma.

2. PRESENTATION OF CASES

2.1 Case One

A 33 year old male who was referred to our emergency department after been found unresponsive in his room. Patient is a chronic alcoholic who had been found intoxicated in a gutter after binge drinking two weeks prior. He complained of headache for a while until he was eventually found unresponsive and subsequently sent to the hospital.

On examination, his vitals were stable but had a Glasgow Coma Score (GCS) of 8/15; best motor score of 3, eye opening response of 4 and verbal score of 1. The right and left pupils were 5 and 3 mm in diameter respectively and reacted sluggishly to light with an associated right hemiplegia.

A pre-operative non contrast head CT scan showed a right fronto-parietal subacute subdural hematoma with a midline shift as well as brain atrophy for a 33 year old (Fig. 1). A clinical diagnosis of Kernohan's notch phenomenon was made and patient underwent an emergency burr hole with drainage of hematoma. Patient's GCS was 10/15 on 1st day post; M=5; V=1; E=4.

He was discharged home on 7th day post-op fully recovered after rehabilitation.

2.2 Case Two

A 53 year old male who was accidentally hit on the head with a car tire jack about a month earlier was brought the emergency department due to worsening neurologic condition. He had been fine after the trauma aside some headaches which he managed with analgesics. He started having reduced consciousness and weakness of the right upper and lower limbs a week prior to presentation.

On examination, his GCS was 10/15; M=5; E=3; V=2 with right hemiparesis and both pupils 3mm in diameter and a sluggish response to light.

A non-contrast head CT showed a right fronto-parietal subacute/chronic subdural hematoma with a significant midline shift (Fig. 2b). As shown in Fig. 2a, patient could not elevate the right upper and lower limbs although there was a supratentorial bleed ipsilateral to the limb weakness. Patient underwent an emergency burr hole with drainage of hematoma after a clinical diagnosis of Kernohan's notch phenomenon was made. He was discharged home on post-operative day 4 with a GCS of 15/15 and full power in all limbs.

2.3 Case Three

A 79 year old female with no comorbidity presented with a day's history of headaches, reduced consciousness, weakness of the right upper and lower limbs and difficulty in speaking. There was no history of trauma, a fall or use of anticoagulants.

On physical exam, she had a GCS of 7/15; M=3; V=2; E=2, a right hemiplegia, a right facial nerve palsy (Fig. 3b) and a right mydriasis (Fig. 3a).

A CT brain (Fig. 3c) showed a right fronto-parietal chronic subdural hematoma with a midline shift. She had an emergency burr hole with drainage of hematoma after a diagnosis of Kernohan's notch phenomenon was made. Patient's GCS on post op day 1 was 7T/15; M=5; E=1; V=1 (intubated).

A repeat CT brain (Fig. 3d) showed residual hematoma and hence was drained through the previous burr hole. Patient's GCS improved to 12/15; M=6; E=4; V=2 with significant improvement in power of the right upper and lower limbs. She was discharged on post-operative day 12 with GCS 12/15 to continue physical rehabilitation on out-patient basis.

2.4 Case Four

A 63 year old male known diabetic who was brought in with a week's history of reduced consciousness and left sided weakness. It could not be ascertained whether there was any history of fall or trauma, no usage of anticoagulants. Patient only took alcohol occasionally.

Exam showed an adult male who looked frail with a GCS of 7/15 (M=3; V=2; E=2) with a left hemiparesis (Fig. 4a) and left mydriasis (Fig. 4b).

A CT brain (Fig. 4c) showed a left fronto-temporo-parietal hematoma with a midline shift. Patient's GCS dropped to 5/15 just before an emergency burr hole and drainage of hematoma was done.



Fig. 1. Pre-operative CT scan a right fronto-parietal subacute subdural hematoma with midline shift



Fig. 2a. Patient having difficulty elevating right upper and lower limbs

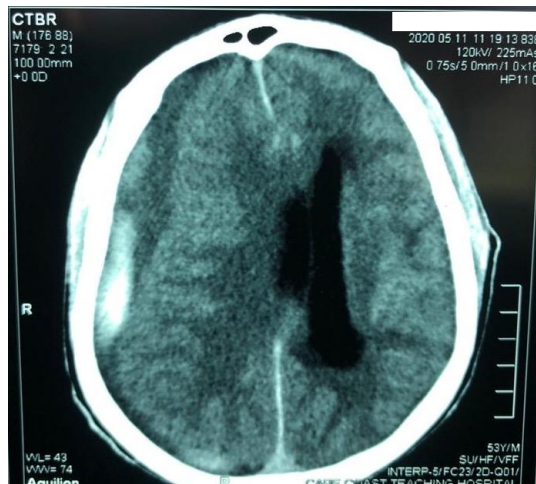


Fig. 2b. Axial CT brain with right fronto-parietal subdural hematoma and significant midline shift



Fig. 3a. Patient with right mydriasis



Fig. 3b. Patient with right facial nerve palsy

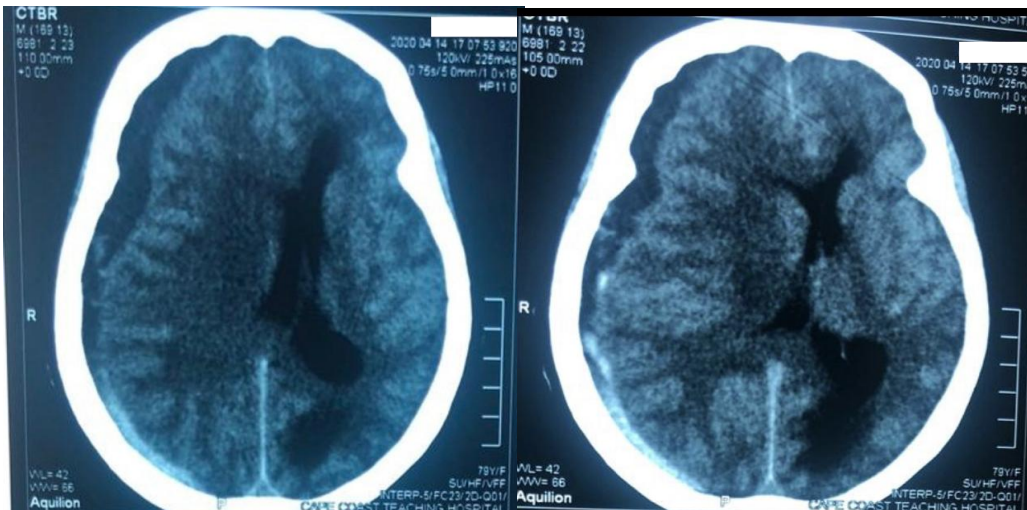


Fig. 3c. Pre-operative CT brain with fronto-parietal chronic subdural hematoma and midline shift

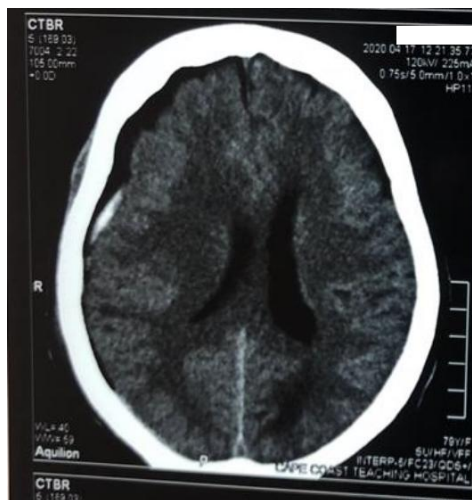


Fig. 3d. Early post op CT showing residual hematoma and midline shift



Fig. 4a. Patient with left hemiparesis (inability to elevate left lower limb)



Fig. 4b. Patient with left mydriasis



Fig. 4c. CT brain showing a left fronto-parietal hematoma with midline shift

Patient/s GCS on post-operative day one was 8/15 (M=4; V=2; E=2) but dipped again 48 hours post-op to 5/15. Chest exam showed bronchial breath sounds bilaterally and in all lung zones.

A redo drainage was done without any repeat CT brain since it would have taken more than two hours to the nearest CT center and back. About 180 millilitres of fresh blood was evacuated, however, patient passed on 12 hours later due to unavailability of any ventilatory support.

3. DISCUSSION

In 1929 Kernohan and Woltman described a phenomenon where the supratentorial mass/lesion may compress the contralateral crus cerebri against the free tentorial edge [3]. The corticospinal tracts in the contralateral crus cerebri may be injured giving rise to a false localizing sign known as Kernohan-Woltman notch phenomenon [5]. Kernohan and Woltman demonstrated this in postmortem examination of patients with hemiparesis ipsilateral to a brain tumour [5].

The corticospinal or pyramidal tracts arise from the motor cortex in the frontal lobes of the brain and descend through the internal capsules, the midbrain and subsequently the pons. At the level of the medulla majority of the fibers (about 80%) decussate. The right cerebral motor cortex thus controls movements of the left side of the body and vice versa. These tracts pass anteriorly in the crus cerebri as they course through the midbrain. Damage at the level of the midbrain thus results in ipsilateral motor weakness since these fibers at this point are yet to crossover to the contralateral side.

The incidence of chronic subdural hematomas is higher among the elderly and alcoholics after trauma to the head. Trivial trauma may still cause a subdural hematoma due to brain atrophy as well as stretching of the bridging veins. Patients who are on anticoagulants are also at increased risk. Other causes of subdural hematomas include coagulopathies, over drainage of cerebrospinal fluid shunts, ruptured intracranial aneurysms and seizure disorders. When patients present with headaches and they have any of the above risk factors there should be a high index of suspicion for a possible subdural hematoma before a focal neurologic deficit develops. The three patients described

above all had risk factors that pointed to a possible subdural hematoma vis a vis chronic alcoholism, brain atrophy, trauma to the head and being elderly.

Patients with chronic subdural hematoma tend to have a decreased blood flow to the thalamus and basal ganglia compared to other brain areas. According to Tanaka et al, a seven percent decrease in cerebral blood flow usually results in headaches while a thirty five percent decrease in cerebral blood flow leads to focal neurologic deficit such as hemiparesis in patients with chronic subdural hematoma [6]. Due to Tanaka's study it is advisable to have a low threshold for brain imaging in patients presenting with headaches and has any of the risk factors that predispose to chronic subdural hematoma. This is especially important in low resource settings where there is inadequate access to brain imaging. These patients are better off being diagnosed early before further deterioration of neurologic function.

All three patients described had ipsilateral motor weakness from a supratentorial space occupying lesion (hematoma) which had pushed the contralateral crus cerebri against the free tentorial edge. Two of the patients had uncal herniation demonstrated clinically by the presence of ipsilateral mydriasis. In compressive third-nerve palsy, the pupil becomes fixed and dilated due to paralysis of the sphincter pupillae. This is due to compression of outer parasympathetic fibres which supplies the sphincter pupillae. The typical presentation would have been an associated contralateral hemiparesis or hemiplegia. However since the contralateral cerebral peduncle with its corticospinal tracts which are yet to crossover at the pyramidal decussation has been compressed against the free tentorial edge patients presents with an ipsilateral hemiparesis or hemiplegia. The presence of hemiplegia / hemiparesis due to an ipsilateral supratentorial mass clinched the diagnosis of KWNP, a false localizing sign.

The diagnosis of KWNP can always be made after imaging and clinical examination. Binder et al however demonstrated electrophysiological changes in the corticospinal tracts in a patient with Kernohan's notch phenomenon using transcranial motor evoked potential recording [7].

Magnetic Resonance Imaging of KWNP describes a rounded lesion found in the cerebral peduncle which is hyperintense on T2 weighted images [8]. Same authors have reported a correlation between signal changes through the cerebral peduncles and recovery of neurologic deficits. Cases without any signal changes showed a much faster and stronger recovery [8]. Magnetic resonance imaging was not used in any of our patients, hence it is difficult to tell if the delayed recovery of the third case could have been due to some signal changes in her crus cerebri. Some others believe KWNP involves the mechanism of cytotoxic edema [9].

The first report of false lateralizing sign was described by Groeneveld and Schaltenbrand in a patient who had ipsilateral hemiplegia due to an intracranial lesion in 1927, thereafter, Kernohan and Woltman did an extensive examination of 276 patients [10].

To highlight this phenomenon Hadelsberg et al recently reported a series of five skull base tumours that presented with Kernohan's notch phenomenon [11].

The first patient although was quite young but had brain atrophy from chronic alcohol abuse putting him at risk of chronic subdural hematoma from any trivial trauma to the head. The delayed recovery of the third patient could have been due to her advanced age and/or signal changes in her crus cerebri. We are unable to attribute it to signal changes in her crus cerebri since no magnetic resonance imaging was obtained.

4. CONCLUSION

The incidence of Kernohan's notch phenomenon in chronic subdural hematoma may be much higher than reported in the literature. Although clinical history and thorough neurologic exam establishes the diagnosis, imaging with CT or MRI is important to avoid wrong site surgeries.

CONSENT

As per international standard or university standard, Participants' written consent has been collected and preserved by the authors.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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