Pakistan Journal of Neurological Sciences (PJNS)

Volume 12 | Issue 3 Article 8

9-2017

Kernohan–woltman notch phenomenon:Case report and Review of literature

Fareed Ahmed
Aga Khan University Hospital, Karachi, Pakistan

Muhammad Shahzad Shamim
Aga Khan University Hospital, Karachi, Pakistan

Sidra Asad Ali
Aga Khan University, Karachi,

Kernohan–Woltman Notch Phenomenon: Case Report and Review of Literature

Fareed Ahmed[1], Muhammad Shahzad Shamim[1], Sidra Asad Ali,
[1]Department of Emergency Medicine, Neurosurgery and Pathology, Aga Khan University Hospital, Karachi, Pakistan

Corresponding to: *Muhammad Shahzad Shamim, E-mail: shahzad.shamim@aku.edu*

Date of submission: *February 27, 2017* ***Date of revision:*** *May 12, 2017* ***Date of acceptance:*** *June 06, 2017*

ABSTRACT:

BACKGROUND:
Subdural hematoma (SDH) is usually secondary to mechanical trauma. Spontaneous subdural hematoma is still rare with unclear pathophysiology. The classic findings of subdural hematoma is sudden drop in mental status, an ipsilateralmydriasis with contralateral hemiparesis. However SDH can sometimes compress the contralaterl corticospinal tract resulting in ipsilateral motor weakness, this phenomenon is known as Kernohan-Woltman Notch Phenomenon.

CASE DESCRIPTION:
We describe here a case of young lady presented to emergency department with brief history of drowsiness followed by right sided weakness. On arrival her Glasgow Coma Scale was 9/15, asymmetrical pupils with right sided weakness. Urgent CT scan was done and Neurosurgery was involved. CT scan showed right sided acute on chronic subdural hematoma. She underwent urgent mini-craniotomy and evacuation of hematoma. Post operatively she recovered well with full recovery of hemiplegia. In our case there was no history of any mechanical trauma and her findings were consistent with KWNP.

CONCLUSION:
It is critical to recognize this false-localizing examination finding, particularly in the emergency setting. Emergency physician should also keep high suspicion of SDH even in the absence of any trauma. There should be no delay for neuro-imaging and every patient should have early CT scan and neurosurgery involvement for prompt management and better outcome.

KEY WORDS:
Non traumatic subdural hematoma (SDH), Kernohan-woltman Notch Phenomenon (KWNP), Emergency department (ED), Computed Topography (CT) scan.

INTRODUCTION:

The classic clinical findings of uncal herniation involve ipsilateral pupil dilatation and contra lateral hemiparesis.[6]However, compression of the contralateral corticospinal tract in the cerebral peduncle against the tentorium notch, secondary to any supratentorial space occupying lesion can sometime leads to an ipsilateral motor deficit, a phenomenon described by Kernohan and Woltman.] In(Kernohan and Woltman, 1929)demonstrated this phenomenon through postmortem examination of patients with hemiparesis ipsilateral to a brain tumor andlabelled it Kernohan–Woltman Notch Phenomenon (KWNP).] In cases of brain tumors, acute subdural hematomas (SDH), and extradural hematomas with midline shift there has been reported localizing neurological sign due to KWNP.]

We describe a case of spontaneous acute on chronic subdural hematoma with unusual presentation of ipsilateral weakness in a young woman.

CASE REPORT:

A 42 year-old lady, with no prior co-morbids, presented to ER with a two-days history of progressive drowsiness. There was no history of trauma or fall. Past medical and surgical history was unremarkable and patient was on no medications. On examination, she was hemodynamically stable, drowsy but responsive to verbal stimuli, with spontaneous semi-purposeful movements of left arm and leg even against resistance (MRC Grade 4), although over the right side, she showed complete

hemiplegia (MRC Grade 0). Her Glasgow Coma Score was 9(E2, V2, M5). She had a ptosis on right side, right pupil was of 5mm, and was non reactive. Left pupil was of 3mm, and was also non reactive. Both planters were up going.

She was urgently moved for CT scan brain which showed a large subdural hematoma of mixed attenuation (representing blood of variable ages), along the right cerebral convexity, with maximum width measuring 16 mm and causing significant mass effect over the brain parenchyma, resulting in effacement of right lateral ventricle and midline shift of 12 mm towards the left side. (Fig 1-2) No fracture was identifiable. Her blood work-up including coagulation profile was normal.With a diagnosis of acute on chronic subdural hematoma, Neurosurgery was taken onboard and the patient was rushed for an emergency mini-craniotomy and evacuation of hematoma.Her post-operative course was unremarkable and she made complete recovery of hemiplegia in the next 2-days. She was discharged on post-operative day 3 with GCS 15 and no cognitive deficits, although even at three weeks follow up, she still had partial weakness of third cranial nerve, causing ptosis and diplopia.

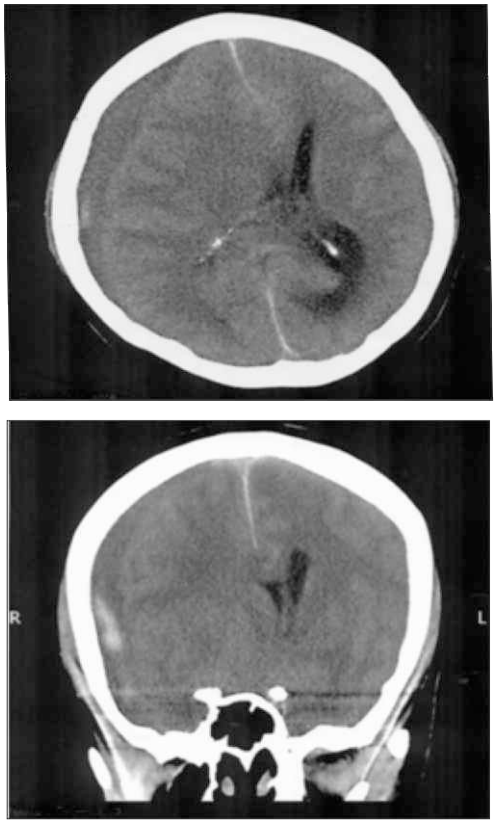

Figure 1a & 1b:
CT scan brain plain axial view and coronal view respectively showing right sidedcrescent shaped acute on chronic SDH with midline shift.

DISCUSSION

To understand the pathophysiology of SDH, it is important to understand the anatomy of bridging veins. SDH result from bleeding of the subdural portion of bridging veins, which is more fragile than the subarachnoid portion of the vein. In trauma, antero-posterior acceleration or deceleration of the head can cause traction of the bridging veins, which rupture at this weak point in the subarachnoid space. Cerebral atrophy, from ageing or alcoholism, accentuates the degree of traction on these bridging veins.[1]

In our case there was no history of any trauma or any fall. In the absence of trauma, SDH might result from sudden increase in intravenous pressure. This can happen for example during coughing or defecation when forcible exhalation against a closed glottis (Valsalva maneuver), [2], but can also happen when someone blow into high resistance instruments, like saxophone.[1]

The classic neurologic presentation of acute SDH is the drop in mental status, an ipsilateral pupillary mydriasis, contralateral (sometimes bilateral) hemiparesis, and posturing.[5] In contrast, when an intracranial mass lesion produces an ipsilateral hemiparesis this represent false-localizing Kernohan-waltman notch phenomenon. Our patient also had ipsilateral motor weakness with asymmetric pupils on presentation, which was consistent with KWNP.

In contrast to the classic ipsilateral brainstem compression by medial temporal lobe herniation from an ipsilateral compressive lesion, the Kernohan-Woltman notch phenomenon occurs when brain displacement results in compression of the contralateral crus cerebri by the tentorial edge. This finding was originally described in postmortem studies of patients with compressive brain tumors by Kernohan and Woltman at the Mayo Clinic in 1929.[3]

Previously MRI was used to demonstrated the Kernohan's notch phenomenon, which shows a rounded lesion found in the cerebral peduncle, which is hyperintense on T2-weighted images that's correlate to KWNP.[4] However some authors believe that the pathophysiology of KWNP involves the mechanism of cytotoxic edema for which diffusion-weighted imaging (DWI) could be more helpful in the initial assessment of KWNP.[8] Nonetheless MRI is detailed and more time consuming study as compare to CT scan, but there are case reports where CT scan have been used to support the diagnosis of KWNP.[7]In our case we also did CT scan of our patient and come up with diagnosis of KWNP.

CONCLUSION:

In our case prompt evacuation of SDH resulted in

complete reversal of KWNP with complete recovery of cognitive function. It is critical to recognize this false-localizing examination finding, particularly in the emergency setting. Emergency physician should also keep high suspicion of SDH even in the absence of any trauma. There should be no delay for neuro-imaging and every patient should have early CT scan and neurosurgery involvement for prompt management and better outcome.

FINANCIAL SUPPORT AND SPONSORSHIP:
Nil.

CONFLICTS OF INTEREST:
There are no conflicts of interest.

REFERENCE:

1. Brennan, P. M., Fuller, E., Shanmuganathan, M., Keston, P., &Fouyas, I. (2011). Spontaneous subdural haematoma in a healthy young male. BMJ case reports, 2011, bcr0120113694.

2. Haykowsky, M. J., Eves, N. D., Warburton, D. E., & Findlay, M. J. (2003). Resistance exercise, the Valsalva maneuver, and cerebrovascular transmural pressure. Medicine and science in sports and exercise, 35(1), 65-68.

3. KERNOHAN, J. W., & WOLTMAN, H. W. (1929). Incisura of the crus due to contralateral brain tumor. Archives of Neurology & Psychiatry, 21(2), 274-287.

4. Mastronardi, L., Puzzilli, F., Ruggeri, A., &Guiducci, A. (1999). Magnetic resonance imaging findings of Kernohan–Woltman notch in acute subdural hematoma. Clinical neurology and neurosurgery, 101(2), 122-124.

5. McKenna, C., Fellus, J., & Barrett, A. M. (2009). False localizing signs in traumatic brain injury. Brain injury, 23(7-8), 597-601.

6. Moon, K. S., Lee, J. K., Joo, S. P., Kim, T. S., Jung, S., Kim, J. H., ... & Kang, S. S. (2007). Kernohan's notch phenomenon in chronic subdural hematoma: MRI findings. Journal of Clinical Neuroscience, 14(10), 989-992.

7. Panikkath, R., Panikkath, D., Lim, S. Y., & Nugent, K. (2013). Kernohan's Notch: A Forgotten Cause of Hemiplegia—CT Scans Are Useful in This Diagnosis. Case reports in medicine, 2013.

8. Uesugi, S., Suehiro, E., Nakayama, H., & Suzuki, M. (2010). Diffusion-weighted magnetic resonance imaging in a case of Kernohan's notch phenomenon. Actaneurochirurgica, 152(10), 1809-1810.

9. Zafonte, R. D., & Lee, C. Y. (1997). Kernohan-Woltman notch phenomenon: an unusual cause of ipsilateral motor deficit. Archives of physical medicine and rehabilitation, 78(5), 543-545.

Conflict of interest: Author declares no conflict of interest.
Funding disclosure: Nil

Author's contribution:
Fareed Ahmed; concept, data collection, data analysis, manuscript writing, manuscript review
Muhammad Shahzad Shamim; data collection, data analysis, manuscript writing, manuscript review
Sidra Asad Ali; data analysis, manuscript writing, manuscript review