

The Kernohan-Woltman Syndrome:

Implications in neurorehabilitation

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Patients who are admitted to a neurorehabilitation ward generally have a definitive diagnosis and prognosis, which optimises their multidisciplinary journey back into the community. When there is diagnostic difficulty, such planning becomes a challenge for the care provider and patient. We present a patient who was initially unconscious, and who later developed the false localising sign of having a right sided hemiplegia, despite radiological evidence of right sided subdural haematoma, with pontine and medullary involvement on computed tomography (CT). This in turn affected the process of goal setting from a multidisciplinary point of view. With further magnetic resonance imaging (MRI) we diagnosed Kernohan-Woltman Syndrome...

Admission

A 46-year-old ambidextrous alcohol-dependent male was found collapsed on the floor in his home and was brought into hospital by ambulance. He was known to have collapsed the day prior to admission. Initially, he had a Glasgow Coma Score (GCS) of 6 (Eyes 1, Verbal 1, and Motor 4). Pupils were equal in size and shape (2mm in diameter) and reactive to light. His creatine kinase was 3850 IU/L, correlating with the patient having been on the floor for some time. Other than alcohol dependence and hepatitis C, he had no other significant past medical history. There were no clotting abnormalities. A CT scan of the brain revealed an extensive fronto-parieto-temporal subdural haematoma extending along the tentorium. Imaging revealed temporal lobe contusions and surrounding oedema. (see Figure 1.) There were no fractures. There was subarachnoid blood with widespread sulcal effacement, effacement of the ipsilateral lateral ventricle and the third ventricle, with a 1.7cm midline shift. There was associated right uncus displacement consistent with herniation. A decompressive craniectomy was done on the same day of admission and post evacuation CT head confirmed evacuation of the right acute subdural haematoma, complete resolution of the right uncus herniation, and an improvement of the midline shift and sulcal effacement.

Postoperatively, his GCS eventually improved to 11 (E3 V2 M6), despite an initially slow recovery which was complicated by aspiration pneumonia. On transfer to the rehabilitation unit, he maintained a GCS of 15 and general systemic examination was unremarkable. There were no peripheral stigmata of liver disease. He had a right-sided mydriasis (right pupil 5mm, left pupil 3mm), a right sided facial droop with sparing of the forehead,



Figure 1: Axial CT head demonstrates an acute right hemispheric and tentorial subdural haematoma with right temporal lobe haemorrhagic contusion. There is marked mass effect with midline shift to the left and associated obstruction of the left lateral ventricle. There is effacement of the basal cisterns due to transtentorial herniation and there is associated impingement on the midbrain.

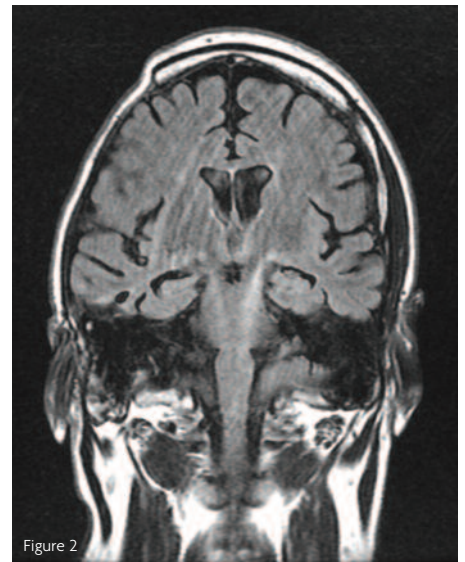


Figure 2: Coronal FLAIR MR image at follow up demonstrates a right sided craniectomy and right temporal encephalomalacic changes. There is signal abnormality within the contralateral midbrain adjacent to the tentorial hiatus.

and deviation of his uvula to the left. He had a significant oro-pharyngeal dysphagia contributed by weakness in the right facial, tongue and laryngeal muscles. He had a marked articulatory dysarthria, however with no expressive or receptive dysphasia. There was no visual field defect nor any nys-

tagmus. He had fasciculations in the right gastronemius.

There was a sustained right ankle clonus. His dense right sided hemiplegia persisted. Light touch, temperature, and pain sensation were intact on the left side, but markedly reduced in the right upper and proximal lower limbs. Proprioception however was preserved throughout. Reflexes were brisk in the lower limbs. The Babinski reflex was positive on the right. There were no signs of cerebellar involvement. In terms of his cognition, he required support in problem solving, had impulsivity, and lacked the ability of initiation and completion of multistep tasks.

Conventionally, there would be a contradiction between the site of the original lesion (right sided haematoma) and the residual right sided hemiplegia. A repeat CT head performed to identify the cause of the ipsilateral hemiplegia was performed, but this still revealed no obvious correlation between the radiological and clinical findings. On discharge from the Neurorehabilitation Unit, the diagnostic challenge was explained to the family and the patient. On further investigation with an MRI scan of the brain 3 months later, it was revealed that this patient had demonstrated a false localising sign.

MRI revealed increased T2 signal involving the left cerebral peduncle, extending inferiorly into the basis pontis and the medullary pyramid, along the descending corticospinal tracts. This correlates with the transtentorial herniation and compression of the brainstem against the contralateral tentorial edge, or known as the Kernohan's notch. These findings were consistent with Wallerian degeneration secondary to compressive injury, and they do correlate with the patient's clinical and radiological findings. (see Figure 2.)

Discussion

Conventionally, a cortical lesion is predicted to cause a contralateral hemiparesis, however in this case the patient presented with an ipsilateral hemiplegia, and pons and medullary signs, contrary to what a CT scan would typically reveal.

Kernohan-Woltman Syndrome, which was reported by Kernohan et al. in 1929,¹ describes an ipsilateral hemiplegia secondary to transtentorial herniation. It has been associated in some cases with an oculomotor palsy.²⁴ In this patient however, we had pontine and medullary signs with an ipsilateral hemiplegia. The presence of pontine and medullary signs have not been reported in the past to our knowledge thus making diagnosis even more difficult. The clinical picture was further complicated by global cognitive deterioration after this trauma which was possibly a consequence of the delay in his presentation. This diagnostic and prognostic challenge was difficult to overcome, especially when explaining to the patient and family in multidisciplinary meetings. This in turn affected the process of goal setting from a multidisciplinary point of view. In the future, it may be indicated that a patient with a false localising sign has an MRI head scan in order to diagnose Kernohan-Woltman Syndrome.

Conclusions

In a subdural haematoma with ipsilateral hemiplegia and pupillary dilatation with reduced consciousness and uncal herniation on CT, Kernohan-Woltman Syndrome should be suspected. Also, it is paramount in the neurorehabilitation setting that a diagnostic MRI is performed to confirm this. Moreover, pontomedullary involvement was demonstrated in Kernohan-Woltman Syndrome, which has not been reported before to our knowledge. ♦

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