

CASE REPORT

Kernohan's notch and misdiagnosis of disorders of consciousness

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SUMMARY

A 69-year-old man presented with a sudden headache followed by unconsciousness. There was no head injury. The Glasgow Coma Scale (GCS) score was 3/15 and there was a left mydriasis, unreactive to light. The CT-scan showed a left acute subdural haematoma causing a remarkable mass effect. A supratentorial hemispheric craniotomy was performed. Nevertheless, after several weeks at the intensive care unit (ICU), the patient was still unresponsive to external stimuli and did not show any motor activity. A comfort care attitude was decided on with the family and the patient was extubated. However, a few days later, the patient subsequently showed a surprisingly favourable course, with improved wakefulness. Indeed, the GCS score improved, and the treatment plan was modified so that the patient benefited from rehabilitation. The MRI showed a right cerebral peduncle lesion, consistent with a Kernohan–Woltman notch phenomenon (KWNP). Six months later, the patient was able to walk and live quite normally.

BACKGROUND

Classically, in uncal herniation, clinical features include ipsilateral pupil mydriasis and contralateral hemiparesis.¹

However, a supratentorial space-occupying lesion can compress the contralateral corticospinal tract in the cerebral peduncle against the tentorium notch, causing a motor deficit ipsilaterally. Kernohan and Woltman described this phenomenon in 1929.² They performed a postmortem examination of subjects with hemiparesis ipsilateral to a brain tumour. They thought that a hemispheric mass lesion could compress the contralateral crus cerebri against the tentorial notch. This is now called the Kernohan–Woltman notch phenomenon (KWNP).³

KWNP is a cause of false localising neurological signs, creating a neurological deficit ipsilaterally to the lesion, and defying the corticospinal decussation principles.⁴

We describe a case of unilateral acute subdural haematoma, in which both ipsilateral and contralateral corticospinal tracts were affected. Despite early surgery, the patient did not show any sign of consciousness postoperatively. Nor did he exhibit any motor response. This contributed to a misdiagnosis of the real state of consciousness and rehabilitation potential.

CASE PRESENTATION

A 69-year-old healthy man presented with a sudden onset of headache followed by

unconsciousness. There was no head injury. The Glasgow Coma Scale (GCS) score was 3/15 and there was a left anisocoria, unreactive to light. The patient was intubated and transferred to the hospital by helicopter. The CT scan performed on admission ([figure 1](#)) showed a left acute subdural haematoma causing a remarkable mass effect. The CT-angiogram was negative for vascular abnormalities. A supratentorial hemispheric craniotomy was performed and an intraparenchymal intracranial pressure (ICP) monitor was inserted.

Postoperatively, the patient was transferred to the intensive care unit (ICU). The CT-scan performed 24 h after surgery was satisfactory, and ICP values were in the 15–20 mm Hg range. After 10 days at the ICU, there was still no contact with the patient, who was intubated with spontaneous respiration. Eyes were open but there was neither eye movement nor fixation. There was a skew deviation, anisocoria (left reactive mydriasis), slightly diminished left corneal reflex and blinking on noise. He was grimacing on pain stimulation, but no motor activity was present. The Coma Recovery Scale-R score was at 6/23, corresponding to a minimal conscious state minus (MCS–). An EEG was performed, showing reactive waveforms, without signs of epilepsy.

Since there was no any improvement on neurological examination, a comfort care attitude was decided on with the family and the patient was extubated.

However, a few days after making this decision, the patient subsequently showed a surprisingly favourable course, with improved wakefulness. Indeed, the GCS improved, and the neurological examination showed a left hemiplegia and a left oculomotor nerve palsy. Given the improvement, the treatment plan was modified so that the patient benefited from rehabilitation. The MRI showed a right cerebral peduncle lesion ([figure 2](#)), T1-hypointense and T2-hyperintense, which was not previously visible on CT scans.

Vaquez's disease was diagnosed during the hospital stay and provided a possible explanation of the intracerebral haemorrhage without a history of trauma.

After several weeks, the clinical status of the patient improved further, and 6 months after hospital discharge, he presented only with a discrete residual left brachioradial hemisyndrome.

OUTCOME AND FOLLOW-UP

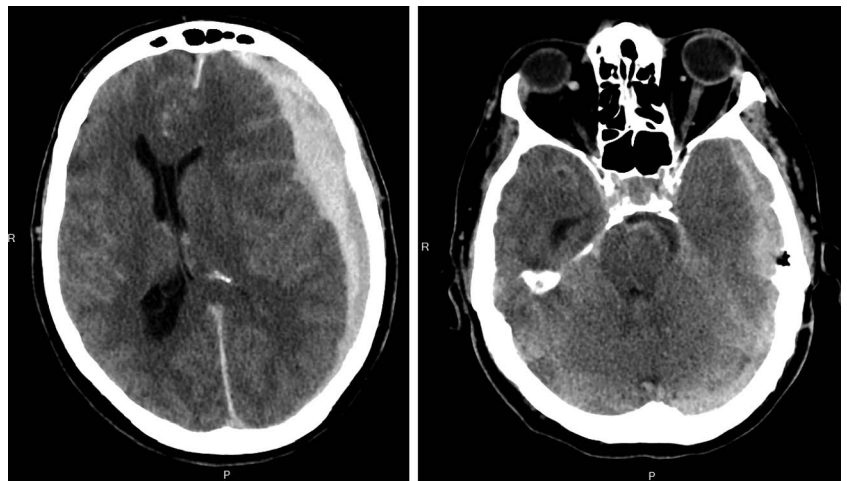
The patient went to neuro-rehabilitation for several weeks, and 6 months after hospital discharge, he



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Figure 1 Early brain CT-scan shows a left acute subdural haematoma, with significant mass effect, and transtentorial herniation.



presented only with a discrete residual left brachiofacial hemiparesis.

DISCUSSION

Acute subdural haematoma remains a condition with an extremely poor prognosis and a high mortality rate.

An acute subdural haematoma can produce a mass effect, leading to ischaemic damage of the contralateral corticospinal tract in the cerebral peduncle, mainly by occlusion of the perforating arteries following compression against the tentorial edge.¹⁻¹²

The outcome of patients with KWNP remains poorly studied. In our case, the large left acute subdural haematoma produced an uncal herniation, compressing the contralateral crus cerebri against the tentorial edge. This created a lesion of the right corticospinal tract and a left third cranial nerve deficit. Moreover, the patient presented also with a right hemiparesis, due to the left acute subdural haematoma. Overall, 1 week after surgery, the patient exhibited severe tetraparesis but did not show any sign of wakefulness. This clinical picture was misdiagnosed as a disorder of consciousness and discussion with the family concerning the withdrawal of care took place. Fortunately, the MRI performed 10 days after surgery showed a radiological correlation to Kernohan's notch phenomenon.

Since the first description of MRI findings associated with KWNP, most reported MRI correlate of KWNP is a rounded lesion found in the cerebral peduncle, hyperintense on T2-weighted images.^{1 3 4 13-16} In most cases, MRI was

performed between one and several weeks after initial presentation, and adequate timing for MRI assessment is not clearly defined.¹⁷⁻²⁸ However, some authors believe that KWNP involves the mechanism of cytotoxic oedema.¹⁶ Therefore, diffusion-weighted imaging (DWI) could be useful in early assessment of KWNP.

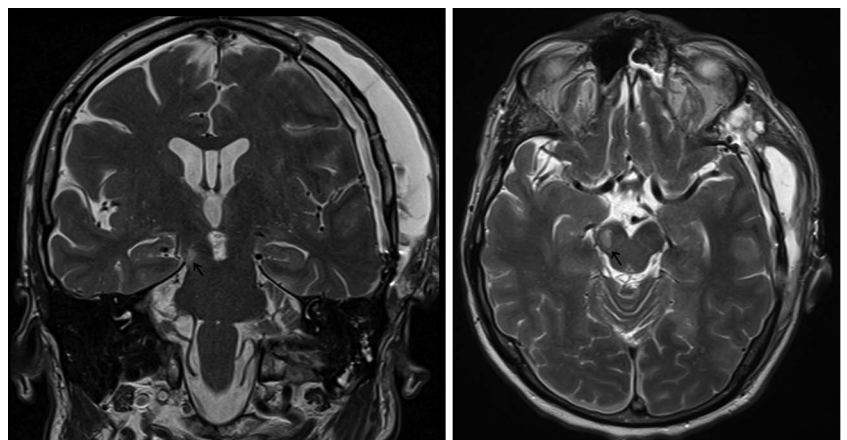
The importance of distinguishing patients showing signs of consciousness (minimally conscious state, MCS) from those who do not (vegetative state, VS)⁶ led to standardised methods designed to assess disorders of consciousness.

Clinically, evaluation of disorders of consciousness relies on observing behaviour and drawing inferences about the underlying state of consciousness. Thus, detection of behavioural signs of consciousness is subject to interobserver variability and depends on unpredictable fluctuations in arousal, sensorimotor impairment, cognitive and language deficits, and sedating medications.⁷ Scores that are being used for the clinical assessment and prognosis, like the Coma Recovery Scale revised (CRS-R), are highly dependent on motor response, and could contribute to a misdiagnosis of disorders of consciousness in patients with bilateral corticospinal tract lesions.

Diagnostic errors in classifying patients in MCS as being in a VS have been reported to range from 30% to 40%.⁷ It can have adverse consequences for clinical treatment and may lead to premature termination of therapy and missed clinical opportunities to promote rehabilitation.

Neuroimaging and electrophysiological procedures are part of the clinical assessment of disorders of consciousness, although

Figure 2 T2-weighted MRI shows a hyperintense area (arrow) involving the middle portion of the right cerebral peduncle: Kernohan's notch phenomenon.



they do not provide sufficient evidence to be included in formal diagnostic criteria yet.

The outcome of patients with KWNP has been poorly studied. However, patients with traumatic brain injury of similar severity to patients with KWNP present with persisting impairments months and even years after injury.⁸ However, our patient did not have any traumatic injury, and Vaquez's disease was diagnosed during his hospital stay. The absence of trauma could contribute to the unusual, quickly improving outcome presented by our patient.

Learning points

- ▶ Kernohan–Woltman notch phenomenon (KWNP) is a cause of false localising neurological signs.
- ▶ KWNP is caused by a supratentorial mass exerting pressure on the contralateral cerebral peduncle against the tentorial notch.
- ▶ KWNP can contribute to a misdiagnosis of disorders of consciousness in patients presenting with bilateral corticospinal tract lesions.
- ▶ Brain MRI is the most sensitive imaging test for KWNP. The timing of the procedure is not clearly defined; however, it should be performed when there are discrepancies between the CT-scan and clinical findings.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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