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REVIEW ARTICLE

Kernohan–Woltman notch phenomenon: a review article

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ABSTRACT

Kernohan–Woltman notch phenomenon (KWNP) is a false localising sign which may still cause diagnostic confusion. It was first described by Kernohan and Woltman in 1929, through post-mortem studies on 297 patients following cases of false localisation. They proposed that raised intracranial pressure causes uncal herniation, which can compress the contralateral cerebral peduncle against the tough tentorium, manifesting as hemiparesis ipsilateral to the primary brain lesion. A number of case reports have been written since the original description of this phenomenon, primarily secondary to intracranial bleeds, and little has been written about long-term outcome of patients who develop KWNP. We performed a literature search of all published cases of KWNP, and reviewed its clinical, pathophysiological, imaging and neurophysiological characteristics. Furthermore, we summarise the long-term outcomes of these patients as described by case reports, with the aim to improve understanding of rehabilitation potential. Thirty-eight cases were found through a PubMed search. We also included a case from our own Trust, making the total number of cases in the analysis 39. Thirty-six cases were secondary to intracranial bleeds (22 of which were traumatic), the remainder were due to an arachnoid cyst, high grade glioma, and reabsorption bone syndrome. Additional clinical manifestations to hemibrachiocrucial syndrome included third nerve palsy, mydriasis of the contralateral and ipsilateral pupils, facial nerve palsies, and parkinsonism. Twenty-six (67%) patients had improvement in motor function of varying degrees, with twelve (31%) patients attaining complete motor recovery or independence. More studies on long-term outcome of patients who develop KWNP are needed to understand rehabilitation potential.

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Kernohan; transtentorial herniation; false localisation; corticospinal tract; neurorehabilitation

Introduction

Kernohan–Woltman notch phenomenon (KWNP) is a rare false localising sign, first described by Kernohan and Woltman in 1929,¹ through post-mortem studies on 297 patients, following several clinical cases of false localisation of brain tumours. They proposed that increased intracranial mass effect exerts force on the contralateral cerebral peduncle, causing it to compress against the contralateral tentorial edge, manifesting as hemiparesis ipsilateral to the primary brain lesion. Since this original paper, descriptions of KWNP have primarily been case reports, and little is published about patients' long-term outcome. We reviewed the clinical, pathological and radiological features of KWNP, and summarised all published case reports of the phenomenon with the aim to further understand rehabilitation potential.

Methods and data collection

A Pubmed search for the following terms was conducted. 'Kernohan phenomenon', 'Kernohan's phenomenon', 'Kernohan's notch', and 'Kernohan–Woltman notch phenomenon'. A total of 35 case reports were found, describing 38 patients, from 1990 to 2015. These included single, multiple case reports, and NeuroImages. We also included a recent patient from our own hospital who developed KWNP secondary to a spontaneous acute subdural and intracerebral haematoma in the data analysis, bringing the total number of patients included to 39. In each case, we recorded information on patient demographics, mechanism of

brain injury, clinical features, timing of MRI and its features, supplementary neuroimaging or neurophysiological studies, and patient outcomes if it was described. The majority of patients displaying KWNP are secondary to traumatic brain injury (TBI), and we further compared outcome of our patient cohort with that described in the literature.

Results

Clinical features

In Kernohan and Woltman's original paper, they provided anecdotal cases of misdiagnoses due to this false localising sign. One case was a large left frontotemporal tumour producing left pyramidal tract clinical features, leading to a diagnosis of right brain tumour. Another case also described irradiation of a right frontal tumour to treat left hemiplegia, and necropsy discovered a left frontal lobe tumour. They subsequently performed necropsy on 276 brain tumour patients, discovering 36 that had demonstrated 'notching' of the contralateral incisura cruris. Six other specimens with notching were found secondary to a chronic subdural haematoma, and five brain abscesses, totalling the number to 40. Retrospective collection of clinical details found that 24 of these patients (all brain tumours) had ipsilateral pyramidal signs (either hyperreflexia or positive Babinski reflex).

Since Kernohan's original paper, cases have been published to further illustrate this pathophysiological phenomenon.

Clinical case example

Our case is a good illustrative example of the clinical course of patients described in the literature who develop KWNP secondary to an intracranial bleed. She was a 56-year-old lady who presented with an acute drop in consciousness and seizures whilst being treated as an inpatient for her recently diagnosed multiple myeloma. Prior to intubation, she was flexing to pain, and had a fixed and dilated left pupil. Initial CT head showed a left-sided acute subdural haematoma and a temporal intracerebral haematoma (Figure 1). She has a past medical history of multiple myeloma (with good response to therapy), acute kidney injury and posterior reversible encephalopathy syndrome. She was on aspirin, clopidogrel and heparin. After administration of hypertonic agents and reversal of heparin, she underwent a craniotomy and evacuation of the subdural and intracerebral haematoma. A few days post-operatively, she was noted to have a left-sided hemiparesis. Cervical spine pathology and stroke were excluded, and MRI brain showed signal changes in the contralateral cerebral peduncle (Figure 2). She was transferred to the rehabilitation ward and had a residual left upper limb weakness six months after the initial bleed.

Literature search

Table 1 summarises all case reports published between 1991 and 2015. Thirty-nine cases were found, including our patient. The table outlines the patient demographics, mechanism of injury, clinical severity, neuroimaging and neurophysiology findings, and comments on long-term outcome. Patients are grouped into the mechanisms of injury and arranged in ascending order of age.

The majority of patients developed KWNP secondary to TBI (22 patients). Other cases that involved an acute deterioration in consciousness included spontaneous acute subdural haematomas, that were secondary to aneurysm,² spinal tap,³ AVM.^{4,5} Six patients had an insidious onset of hemiparesis, ranging from 2 months to 2 years. Aetiologies of cases with insidious onsets were three chronic subdural haematomas,^{6–8} an reabsorption bone syndrome,⁹ a high grade glioma,¹⁰ and arachnoid cyst.¹¹

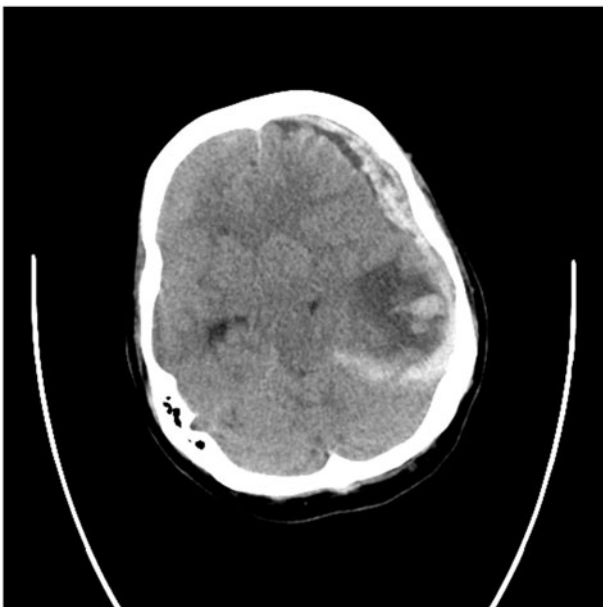


Figure 1. CT scan showing left acute subdural haematoma and temporal intracerebral haematoma.

Pyramidal tract and ocular features

The characteristic finding in KWNP is a hemibrachioocular syndrome which is ipsilateral to the primary lesion, and this feature is seen through all 39 cases presented. In addition to this, 25 cases explicitly described an ipsilateral mydriasis at presentation.^{2–5,8,12–30} These were all in the context of intracranial haemorrhage. Five of these cases also had motor involvement of the ipsilateral oculomotor nerve, leading to a divergent gaze and partial or complete ptosis.^{12,13,22,24,27} One case described a contralateral mydriasis, which was attributed to a transient lesion affecting the Edinger–Westphal Nucleus.²⁵ Two patients also had an ipsilateral upper motoneuron facial nerve palsy, suggesting involvement of corticobulbar fibres in the cerebral peduncle.^{14,24}

Extrapyramidal features

Another case describes Parkinsonism secondary to compression of the midbrain against contralateral tentorium.¹⁸ This patient had suffered a traumatic left ASDH, and subsequently developed left-sided Parkinsonism as soon as he regained motor skills. Interestingly, he did not have any pyramidal signs. However, MRI did demonstrate high T2 signal traversing the right cerebral peduncle and substantia nigra.

Pathophysiology

KWNP classically occurs when a lesion causes compression of the contralateral cerebral peduncle against the tentorium, resulting in disruption of the corticospinal tract fibres above the medullary decussation (Figure 3). This ultimately results in a hemiparesis ipsilateral to the side of the primary lesion. The uncus, parahippocampal gyrus and hippocampus are structures closely related anatomically to the incisura. Posterior cerebral artery and superior cerebellar artery are the vascular structures coursing along this space, and the first six cranial nerves are also closely related. All of these structures have the potential to be involved in herniation

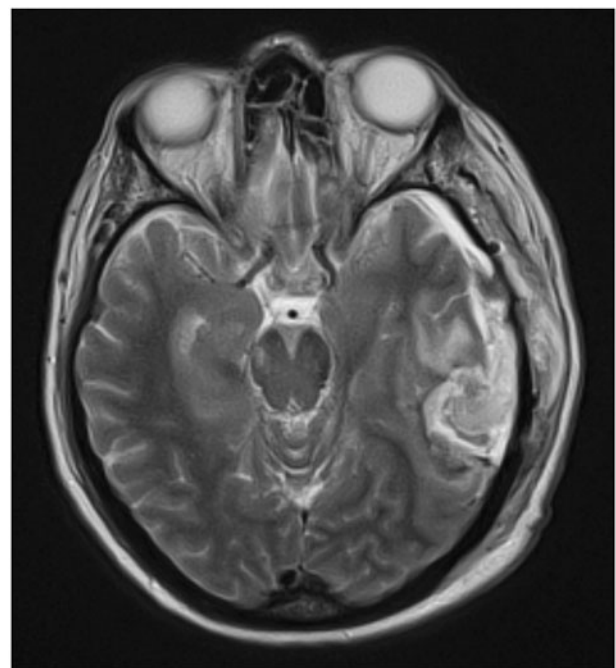


Figure 2. MRI brain shows a hyperintense lesion at the right cerebral peduncle.

Table 1. Details of all reported cases of KWNP. Motor outcome is classified into resolved, improving, residual weakness and unknown.

Author	Year	Age	Sex	Mechanism of injury	Initial GCS	Pupils	Pathology	Surgery	Time of MRI	MRI findings	Other investigations	Outcome description	Outcome
Traumatic brain injury (n = 22)													
Zafonte ¹²	1997	16	Male	Road traffic accident	GCS 3		Traumatic left ASDH	Emergency craniotomy	2 weeks post-operatively	<i>Right cerebral peduncle:</i> T1: hypointense		Independent with ambulation	Improving
Namura ¹³	1997	16	Female	Road traffic accident	GCS 5 (E1 V1 M3)	Right fixed and dilated pupil, partial oculomotor palsy	Traumatic right ASDH	Decompressive craniectomy	1 month post-operative	<i>Left cerebral peduncle:</i> T1: hypointense T2: hyperintense. Also T2 hyperintense in thalamus and occipital areas		1 year: returned to former life	Resolved
Cohen ¹⁴	1990	19	Male	Road traffic accident	Somnolent, de-cerebrate posturing	Fixed and dilated ipsilateral pupil	Traumatic left EDH	Exploratory burr holes, then proceeded to craniotomy	1 week post-operative	<i>Right cerebral peduncle:</i> T2: hyperintense		Walk independently, but mild residual weakness of face and arm at 2 weeks post-operatively	Improving
Uesugi ¹⁵	2010	19	Male	Boxing head injury	GCS 7	Left pupil sluggish	Traumatic left ASDH	Craniotomy	7 days post-operative	<i>Right cerebral peduncle:</i> T2: hyperintense	DWI: Restricted diffusion at lesion. Repeat at 6 weeks: FA increased in the affected peduncle, but no difference between ADC and control	Unknown	Unknown
Binder ¹⁶	2004	21	Male	Road traffic accident	GCS 6 (E1 V1 M4)	Left dilated pupil	Left depressed temporal bone fracture, with left contusions and 4 mm midline shift	Ventriculostomy, and insertion of ICP monitor	MRI right peduncle T2 prolongation up to internal capsule	<i>Right cerebral peduncle:</i> T2: hyperintense up to internal capsule	<i>Transcranial magnetic stimulation:</i> increase in voltage threshold and reduction in MEP waveform	Had tracheostomy, then went to rehabilitation unit	Unknown
Mansilla-Fernández ¹⁷	2015	24	Male	Attacked	GCS 4 (E1 V2 M1)	Left dilated and sluggish pupil	Traumatic left ASDH	Craniotomy	45 days after injury, and a repeat MRI 2 months later	<i>Right cerebral peduncle:</i> Ischaemic changes	FA was reduced in the right at the mesencephalon. Tractography performed 2/12 after initial MRI (45 days, therefore overall, 3.5 months post injury), when he had LUL 5/5, LLL 4+/5	Left limbs 4+/5 at 2 months	Improving
Jang ³⁵	2013	25	Female	Assault	GCS 10		Traumatic left ASDH with frontal ICH	Craniotomy	10 days after injury	<i>Right cerebral peduncle:</i> T1: hypointense T2: hyperintense	DTI (8 weeks): Reduced FA and increased ADC at right peduncle and pons	15 months: ambulating independently, can grasp and release small objects	Resolved
Evans ¹⁸	2004	27	Male	Feel backwards on stairs	GCS 3	Fixed and dilated pupils	Traumatic left ASDH, parieto-occipital fracture	Left craniotomy	9 months after injury	<i>Right cerebral peduncle:</i> T2: hyperintense, involving right substantia nigra	DAT scan 5 years later: bilateral asymmetrical reduction in striatal uptake, putamen preserved	Unified Parkinson's Disease Rating Scale 10 (range 0–199, 0 = no disability, 199 = total disability)	Resolved
Kole ¹⁹	2001	29	Male	Boxing match	GCS 3	Right fixed and dilated pupil	Traumatic right ASDH	Emergency craniotomy	Uncertain	<i>Left cerebral peduncle:</i> T2: hyperintense		Independent with daily living	Resolved
Gimenez-Pando ²⁰	2004	34	Male	Fall	GCS 4 (E1 V1 M2)	Left dilated pupil	Left EDH, with left-sided cranial vault fracture	Craniotomy	13 days. Hyperintense on T2 and FLAIR, isointense on T1	<i>Right cerebral peduncle:</i> T1: isointense T2: hyperintense FLAIR: isointense		Recovered almost completely	Improving
Clement ²⁴	1996	36	Male	Blunt head trauma	Not given	Dilated right pupil	Left EDH	Emergency craniotomy	3 months post-operative	<i>Right cerebral peduncle:</i> T2: hyperintense		Unknown	Unknown
Schedler ²¹	2002	38	Male	Struck on head in a fight	Sleepy	Divergent eyes	Traumatic right ASDH	Emergency craniotomy				Independent ambulation, returned to baseline cognition	Resolved
Wolf ²²	1994	39	Male	Struck on head by golf club	Somnolent	Divergent eyes	Traumatic left ASDH	Right craniotomy, then left craniotomy weeks later				Unknown	Unknown

(continued)

Table 1. Continued

Author	Year	Age	Sex	Mechanism of injury	Initial GCS	Pupils	Pathology	Surgery	Time of MRI	MRI findings	Other investigations	Outcome description	Outcome
Mejia Kattah ²³	2014	40	Male	Found in street with signs of head injury	GCS 4 (E1 V1 M2)	Right dilated pupil	Right EDH	Craniotomy	3 days after injury	Left cerebral peduncle: Ischaemia of left peduncle and internal capsule (hyperintense on T2)		Residual right hemiparesis despite intensive therapy	Residual weakness
Zafonte ¹²	1997	42	Male	Assault with brick	GCS 4	Right oculomotor nerve palsy	Traumatic right ASDH	Emergency craniotomy	2 weeks post-operative	Left cerebral peduncle: T1: hypointense		6 weeks: Independent with ambulation	Improving
Hamada ³⁶	2000	52	Male	Slipped on ice	GCS 4 (E1 V1 M2)	Partial right oculomotor nerve palsy	Traumatic left ASDH	Emergency craniotomy	5 days post-operative	Right cerebral peduncle: T2: hyperintense		Unknown	Unknown
Codd ²⁴	2013	52	Female	Fell 1.5 m	Somnolent, but rousable		Traumatic right ASDH	Craniotomy	MRI 3/12: T2 hyperintensity	Left cerebral peduncle: T2: hyperintense	DWI: Restricted diffusion in left cerebral peduncle	Complete resolution of neurology function in 3 months	Resolved
Jang ³⁵	2013	67	Male	Assault	GCS 3		Traumatic right ASDH	Craniotomy	5 days post-operatively	Left cerebral peduncle: Shifted towards tentorium, but no signal change	DWI (4 weeks): Reduced FA of left peduncle and anterior horns. No change at 9 month DTI scan	15 months: ambulate with quad-cane under supervision, cannot grasp and release freely	Residual weakness
Panikath ³³	2013	69	Male	Recurrent falls, Alzheimer's disease	Coma		Right subacute subdural haematoma	Craniotomy	Post-operatively	Compression of left mid-brain with hyperintensity		Unknown	Unknown
Mastroradi ²⁵	1999	73	Female	Slipped on stairs	GCS 4 (E1 V1 M2)	Left dilated pupil	Traumatic right ASDH	Emergency craniotomy	2 weeks post-operative	Left cerebral peduncle: T1: hypointense T2: hyperintense		Unknown	Unknown
Carrasco ²⁶	2009	81	Female	Minor head injury, on anti-coagulants	GCS 7	Right dilated pupil	Traumatic right ASDH	Craniotomy	4 week 1 peduncle hyper on T2	Left cerebral peduncle: T2: hyperintense	Gradient echo MRI: linear hypointense signal at lower border of peduncular lesion	Moderate improvement at 3 months	Improving
Eesa ³⁷	2010			Head injury			Left sided contusion	Conservative management		Right cerebral peduncle: T2: hyperintense	DWI: Restricted diffusion at the lesion	Partial recovery of motor function	Residual weakness
Spontaneous ASDH (n = 8)													
Fujimoto ⁴	2004	23	Female	Rupture of left occipital AVM	GCS 4 (E1 V1 M2)	Left dilated pupil. Both pupils unreactive to light	Left occipital ICH and ASDH	Emergency craniotomy to evacuate ASDH. 2 days later, craniotomy to remove ICH and AVM			SSEP: Voltage of left median nerve stimulation/right scalp recording, was less than right nerve stimulation/left scalp recording	No significant neurological deficit 1 month later	Resolved
Jones ²⁷	1991	25	Male		Confused	Right pupil-sparging oculomotor nerve palsy	Spontaneous right ASDH	Craniotomy	Post-operative	Left cerebral peduncle: T1: hypointense T2: hyperintense		Mild right-sided weakness at 11 days post-operatively	Improving
Dibble ⁵	2015	31	Female	Left parietal arterio-venous malformation (Spetzler-Martin 2)	Obtunded	Fixed and dilated left pupil	Spontaneous L ASDH	Decompressive craniectomy	Post-operative	Right cerebral peduncle: T2: hyperintense		4+/5 in left limb power, smooth unassisted ambulation	Resolved
Openshaw ³	2005	32	Female	Lumbar puncture for infection of intrathecal reduced metho-trexate	Abrupt onset of headache and sensorium reduced	Left dilated pupil	Left ASDH	Left craniotomy	1 week after injury	Right cerebral peduncle: T2: hyperintense		Hemiparesis persists 32 months later	Residual weakness
Iwama ²	1992	39	Male	Rupture of left anterior cerebral artery aneurysm	Deep coma	Fixed and dilated pupils	Spontaneous left ASDH	Craniotomy and aneurysm clipping	3 months post-operative	Right cerebral peduncle: T1: hypointense T2: hyperintense		Unknown	Unknown
Example clinical case	2015	56	Female		GCS 7 (E1 V2 M4)	Fixed and dilated left pupil	Spontaneous left ASDH and ICH	Craniotomy	7 days post-operative	Right cerebral peduncle: T2: hyperintense		Residual right upper limb weakness at 6 months	Improving
Simonin ²⁸	2014	69	Male	Vaquez disease	GCS 3	Left fixed and dilated pupil	Spontaneous left ASDH	Craniotomy	T1 hypo, T2 hyper	Right cerebral peduncle: T1: hypointense T2: hyperintense		6 months after injury: discrete residual left brachioradial hemisyndrome	Improving

(continued)

Table 1. Continued

Author	Year	Age	Sex	Mechanism of injury	Initial GCS	Pupils	Pathology	Surgery	Time of MRI	MRI findings	Other investigations	Outcome description	Outcome
Yoo ³⁸	2008	78	Male		4 limb paralysis, R more severe		R acute subdural, unknown origin (basal cisterns effaced)		6 weeks post-operatively	Left cerebral peduncle: T2: hyperintense	DTI: FA of left cerebral peduncle and medulla in patient was lower than controls, when compared to right side. Tractography showed interruptions at left mid-brain and medulla. TMS: MEP from right APB showed lower amplitude, prolonged latency and high excitatory threshold	Improving	Improving
Chronic subdural haematoma (n = 6)													
Çabalır ³⁹	2014	43	Male		GCS 13 (E3 V4 M6)	Right dilated pupil	Right CSDH	Burr hole	Post-operative period	Left cerebral peduncle: T2: hyperintense		Gradual recovery 3 months post-operatively, mRS 4	Improving
Bhatoe ⁶	2005	56	Male	Fell off bike 2 weeks earlier	GCS 11		Traumatic left CSDH	Burr hole	Post-operative period	Right cerebral peduncle: T2: hyperintense		Gradual recovery in 3 months	Improving
Moon ³⁰	2006	56	Female		GCS 11	Fixed and dilated right pupil	Right CSDH	Burr hole	1 week after surgery	No abnormalities at cerebral peduncle		All neurological symptoms immediately resolved post-operatively	Resolved
Itoyama ⁷	1995	69			GCS 9		Left CSDH	Burr hole	9 days post-operatively	Right cerebral peduncle: Deformity, no signal change		Left hemiparesis improved significantly post-operatively	Improving
Moon ³⁰	2006	70	Male		GCS 9		Left CSDH	Burr hole	Pre-operative	Right cerebral peduncle: T1: hypointense		Some remaining hemiparesis post-operatively	Improving
Albayrak ⁸	2012	88	Male		GCS 14	Right dilated pupil	Right CSDH	Burr holes	Pre-operative MRI	T2: hyperintense		Complete recovery in early post-operative period	Resolved
Other Primary Pathologies													
Other primary pathologies (n = 3)													
Carrasco-Moro ⁹	2014	18	Male	Sunken flap syndrome			Sunken flap syndrome	Bilateral decompressive craniectomy, Bone was replaced in 2 weeks time	5 months after injury, when ipsilateral weakness presented	Compression of left cerebral peduncle against tentorium	TMS: confirmed KWNP	Complete recovery of motor function in first follow up month	Resolved
Oster ¹¹	2007	55			GCS 15		Right frontal arachnoid cyst	Marsupialise into sylvian fissure	Pre-operative, and 7 months post-operative	Left cerebral peduncle: Residual notching and gliosis 7 months post-operative		Asymptomatic after 1 year	Resolved
Velasquez ¹⁰	2015	55	Female				Left temporal and insula high grade astrocytoma	Brain biopsy		Right cerebral peduncle: T1: hypointense		Unknown	Unknown

ASDH: acute subdural haematoma; CSDH: chronic subdural haematoma; EDH: extradural haematoma; ICH: intracerebral haematoma; DAT: dopamine transporter SPECT scan; DTI: diffusion tensor imaging; DWI: diffusion weighted imaging; TMS: transcranial magnetic stimulation.

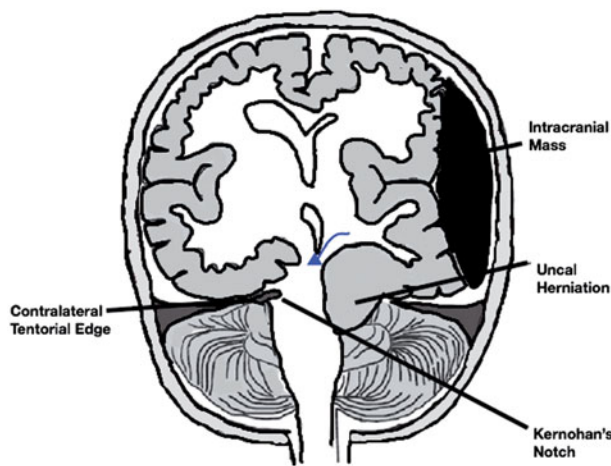


Figure 3. Schematic diagram of transtentorial herniation causing compression of the contralateral cerebral peduncle against the tentorium cerebelli.

syndromes, and some of which have been described in the literature.¹

Kernohan originally described KWNP in the context of advanced brain tumours, and the pathophysiology to be of a mechanical nature. Staining for myelin and axons showed localised destruction of the myelin, sometimes extending down to the substantia nigra. However, in his series, few cases had descending axon involvement despite the myelin degeneration.

There have been subsequent questions on predispositions to developing transtentorial herniation and Kernohan's phenomenon. Adler and Milhorat³¹ performed an autopsy study on 100 people to classify anatomical variations in the tentorial notch, and classified maximum notch width into narrow (24.5–27.0 mm), typical (27.1–31.9 mm) and wide (32.0–39.0 mm) categories. It was proposed that narrow notch width patients may be more predisposed to developing transtentorial herniation, and subsequent KWNP. However, there is currently little evidence to support or refute this hypothesis.²⁶ There is also speculation that involvement of the uncrossed component of the corticospinal tract (~25%) by the primary lesion is the cause of ipsilateral hemiparesis, and that anatomical variations in the uncrossed fibres could determine susceptibility of patients to developing KWNP.³² It is likely that a combination of the factors including rate of mass effect, degree of lateral brainstem displacement and width of the tentorial incisura can contribute to the development of KWNP.^{13,30,31}

Neuroimaging

CT scans are helpful to demonstrate transtentorial herniation and deformation of the midbrain,³³ which in the presence of ipsilateral hemiparesis should raise suspicions of KWNP. Whilst CT is useful in the acute setting, these findings are not specific to KWNP, and conventional MRI was used on 36 cases at varying points in time ranging from initial presentation of the hemiparesis to three months later.³⁴ MRI study findings were not included in three cases.^{4,21,22}

Conventional MRI demonstrates notching and midbrain deformity in keeping with CT findings. However, in the majority of cases where MRI was used (X/Y), conventional MRI also demonstrates a T2 hyperintense and T1 hypo to isointense region in the contralateral cerebral peduncle (Figure 2). These T1 and T2 changes were seen even when MRI was not performed at initial presentation, and even after treatment of initial mass effect.^{2,3,6,9,11–15,17–20,23–29,33–38}

Moon et al.³⁰ postulated that a lack of MRI signal change may correlate with a better functional prognosis in the context of CSDH, based on two patients in their case review, and two other cases in the literature.^{7,8} However, in our literature review, signal change does not necessarily correlate with better prognosis. Eight^{5,11,13,18,19,21,24,35} out of 29 cases with MRI signal changes had resolved motor deficits, three had poor motor recovery,^{3,23,37} and one³⁵ out of the five cases which did not have MRI signal changes had a poor outcome. The long-term persistence of T2 and T1 changes on MRI is not known – only one report describes that the T2 MRI signal changes resolve with rehabilitation.¹¹

Specialised MRI techniques used in addition to conventional brain MRI can demonstrate further findings in KWNP. Diffusion weighted imaging MRI findings are not consistent; whilst Uesugi et al. demonstrated low apparent diffusion coefficient (ADC) in keeping with restricted diffusion within the contralateral cerebral peduncle, this finding was not observed in other cases.^{35,38} With susceptibility weighted MRI, Carrasco et al. reported hypointense signal on a patient who suffered KWNP secondary to an ASDH, thought to be attributed to haemosiderin deposition.²⁶

Recent case reports have attempted to use MRI diffusion tensor imaging (DTI) and diffusion tensor tractography (DTT) to diagnose KWNP and predict better functional outcome. Jang et al.³⁵ showed reduced fractional anisotropy in two patients' contralateral cerebral peduncles, one of which did not have T1/T2 changes on conventional MRI. Only one of the two patients had interrupted tractography, who made a worse recovery than the patient who had intact tractography. However a separate study by Yoo et al.³⁸ demonstrated slow but good recovery in a patient who had interruptions to the corticospinal tract in the midbrain and medulla on tractography.

Neurophysiological results

Neurophysiological studies conducted include somatosensory evoked potentials (SSEP), and three cases of transcranial magnetic stimulation (TMS) motor evoked potentials.^{9,16,38} Fujimoto et al.⁴ used SSEP alongside clinical features to diagnose KWNP, noting a reduced SSEP voltage over the contralateral cerebral hemisphere when the ipsilateral median nerve was stimulated, compared to contralateral nerve stimulation and ipsilateral hemisphere recording. Binder et al.¹⁶ conducted TMS on a 21-year-old male who suffered a depressed fracture and intracranial contusions, to help further characterise his ipsilateral hemiparesis. The patient also had a characteristic hyperintense T2 lesion extending from the right cerebral peduncle to the right internal capsule. The unsurprising result was a marked increase in voltage threshold and reduction in complexity of the MEP waveform on the hemiparetic side. Similarly, Yoo et al.³⁸ also showed a higher voltage threshold, lower MEP amplitude and prolonged latency in the hemiparetic side.

Long-term outcome

There has been little formal description of long-term functional outcome for patients who develop KWNP. This is possibly due to the rarity of the condition, as well as the complex nature of associated primary pathologies. Most cases of KWNP described here are secondary to severe TBI. Factors influencing outcomes for TBI patients are well described.

From our cohort of patients, 67% (26) showed some degree of improvement in motor function. 31% (12) had an outcome description indicating independence or complete resolution of

neurological deficits (Table 1, marked as 'Resolved'). 10% (4) had residual weakness up to 32 months post-injury. These included a 40-year-old patient sustaining an EDH,²³ a 67-year old with a traumatic ASDH,³⁵ conservative management of a cerebral contusion,³⁷ and an ASDH secondary to lumbar puncture for injecting intrathecal methotrexate for a patient with acute lymphoid leukaemia.³

In the group of TBI patients (22 in total), 77% (17) were showing improvement in motor function, with 27% (6) having resolved neurology. 32% (7) of TBI cases did not describe the patient's outcome, and 23% (9) cases out of all those reviewed did not mention patient's outcome in the report.

Discussion

Kernohan's phenomenon is a rare and interesting complication secondary to intracranial mass effect. Since this first description, there have been many reports describing clinical and radiological features of this phenomenon. Collating all of the cases and findings, investigation results appear to support the initial findings by Kernohan and Woltman. Compressive effects of the tentorium on the crus can damage myelin, leading to prolonged latency and higher voltage thresholds observed in neurophysiology studies. Petchial haemorrhages may be seen through gradient echo MRI, and cytotoxic oedema could also be present. When the compressive effect of the tentorium extends to involve the substantia nigra, Parkinsonism may become a clinical manifestation of KWNP.

Although originally described to primarily be a pyramidal tract disorder, the involvement of other midbrain structures close to the cerebral peduncle can manifest KWNP in a spectrum of neurological disorders. Examples given in case reports include involvement of the Edinger–Westphal nucleus and the nigrostriatal pathway. The nature of false localisation, in the instance of KWNP, still causes diagnostic difficulty in the clinical setting, despite the advances in neuroimaging. These include operating on the wrong side of the haematoma,²² requiring neuropsychological testing to help localise the lesion,³⁴ and a case of tetraparesis leading to a misdiagnosis of consciousness.²⁸ It is also important in the initial stage to exclude other primary pathologies (for example contralateral cortical lesions, spinal cord injuries), and brainstem lesions that may appear similar on imaging to KWNP. These include duret haemorrhages, petchial haemorrhages, and spontaneous hypertensive bleeds.³⁰

Little has been published on the long-term outcomes for patients who develop KWNP. The majority of patients who develop KWNP are moderate to severe TBI patients, the outcome of which is well documented. Factors that are associated with worse mortality and overall function include advanced age, low initial GCS, involvement of pupillary reflexes, >5 mm midline shift,³⁹ obliteration of basal cisterns, and secondary insults.⁴⁰ The pathophysiology of KWNP in the context of TBI involves most of the above, implying that patients who develop KWNP inherently have poor prognostic factors.

Most TBI studies examine outcome in the form of mortality or Glasgow Outcome Scale. One study of severe TBI patients examined motor outcomes, showing percentage of patients with normal arm and leg power at six months post-injury was 82% and 81%, respectively.⁴⁰ This subsequently increased to 87% and 91% respectively at two years post-injury. This study included patients with initial GCS <9, regardless of primary pathology. Compared to patients from our literature review that matched the initial GCS criteria as the above study, 24% (5 out of 21) of patients with KWNP were described to have reached normal motor power, or

were independent, ranging from 3 months to 15 months. 76% (16 out of 21) were showing improvements in motor function within the post-operative period. The seemingly higher percentage of patients with normal motor power in Walker's study compared to our cohort with KWNP may imply that patients with direct disruption of the corticospinal tract take longer to rehabilitate. However, without knowing what proportion of the KWNP will reach their premorbid upper and lower limb power, as well as whether it matches the other inclusion criteria of Walker's study, it is difficult to directly compare motor outcome of the two groups.

Similarly, the majority of TBI outcome studies use Glasgow Outcome Scale, which includes cognitive and psychiatric assessments in order to elucidate the patient's ability to carry out activities of daily living and re-integrate into society. Most of the KWNP case reports only address rehabilitation of motor deficits, therefore a direct comparison in GOS of TBI patients with develop KWNP with TBI outcomes in the literature is difficult.

The incidence of KWNP secondary to TBI is also unknown. Patients described in these case reports have all survived the initial brain injury, despite the poor prognostic indicators that are associated with the pathophysiology of KWNP. All of the affected patients in this review had been sufficiently weaned off sedation to note an ipsilateral hemiparesis, which warranted further investigations that usually included MRI scans. It is unknown what proportion of patients with poorer outcomes also had KWNP, and therefore there is an inherent publication bias through these case reports. Similarly, previous studies on TBI outcome do not stratify KWNP as a complication, and it is a possibility that within the IMPACT or CRASH databases, there were patients who have had KWNP, which may have influenced outcome. Consideration of disruptions to the corticospinal tract due to KWNP affecting functional outcome should be taken for future TBI studies.

Despite the uncertainties about overall functional outcome in patients who develop KWNP, the results from our literature review show that the motor deficit is reversible in the majority of patients who survive the primary injury. In addition to the known factors that affect TBI outcome, it is likely that in the context of KWNP, other factors such as time to decompression, tentorial notch width, radiological and neurophysiology features can predict motor recovery.

Conclusion

Kernohan's phenomenon is a rare false localising sign, which can lead to diagnostic and clinical confusion. We have reviewed the proposed pathophysiology, neuroimaging features associated with this phenomenon, and demonstrated that whilst functional outcome is determined by factors influencing the primary pathology, motor function can be regained in most cases. Patients affected by KWNP should be counselled on the rehabilitation potential of the affected limbs, however, more data into the reversibility of KWNP can help to further understand rehabilitation potential.

Disclosure statement

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this article.

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