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Delayed Hemiplegia and Posterior Cerebral Artery Infarction in Kernohan-Woltman Notch Phenomenon: Case Report and Literature Review

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ABSTRACT

Background: Kernohan-Woltman Notch Phenomenon (KWNP) causes ipsilateral mydriasis and paradoxical ipsilateral hemiparesis or hemiplegia. Delayed ipsilateral hemiplegia and infarction of the posterior cerebral artery (PCA) are rare manifestations of KWNP. **Case summary:** A 42-year-old man presented with loss of consciousness after an accident. His GCS was 10/15. He was anisocoric with left-sided mydriasis and had hypertonia and hyperreflexia in the left lower and upper limbs with normal tone and reflexes in the right limbs. A computerized tomography (CT) scan showed an acute left subdural hematoma with midline shift. After craniotomy, he developed hemiplegia (Medical Research Council [MRC] grade 0/5) on his left upper and lower limbs. He also developed an "inability to see" from his left eye. He was discharged 4 weeks later with a GCS of 15/15 and power of MRC grade 5/5 on his right and left upper and lower limbs. **Investigations**: A pre-operative CT scan revealed an acute left subdural hematoma and midline shift. A post-operative CT scan revealed a contralateral (right) PCA infarct. All laboratory tests conducted were normal. **Outcome/Interpretation:** The patient had an acute left subdural hematoma with midline shift. The subdural hematoma also caused KWNP with delayed hemiplegia and contralateral (right) PCA infarction, which caused visual changes. **Conclusion**: KWNP may cause delayed hemiplegia. This presentation may also be accompanied by infarction of the contralateral PCA, causing visual changes. Recognizing this presentation is crucial in diagnostic formulation for hemiplegia and visual changes occurring after decompressive craniotomy. MRI may be useful in diagnosing KWNP and differentiating it from a primary brainstem hemorrhage.

Keywords: Kernohan-Woltman Notch Phenomenon, acute subdural hematoma, PCA infarction, case report.

INTRODUCTION

Kernohan-Woltman Notch Phenomenon (KWNP) refers to the occurrence of an intracranial lesion, which causes significant side-to-side mass effect; specifically, the herniation of the uncus through the tentorial notch of the tentorium cerebelli (1). The herniated uncus compresses the contralateral crus

cerebri against the free edge of the tentorial notch, leading to paradoxical hemiparesis or hemiplegia that is ipsilateral to the lesion, as shown in Figure 1 (1). The hemiparesis or hemiplegia is paradoxical as it occurs ipsilateral to the lesion, compared to classical uncal herniation, which causes contralateral hemiparesis, as the ipsilateral crus cerebri is directly compressed by the herniated uncus. However, KWNP and classical uncal herniation have a similarity, as they both cause compression of the ipsilateral oculomotor nerve at the level of the superior colliculus. This leads to ipsilateral mydriasis. Thus, KWNP causes paradoxical ipsilateral hemiparesis or hemiplegia and ipsilateral mydriasis.

There are numerous documented variations of KWNP presentation. For instance, the phenomenon may cause patients to present with a decerebrating posture or even a forehead-sparing facial droop, on top of the ipsilateral mydriasis and hemiparesis/hemiplegia (2,3). Knowledge of these varying presentations is important as the incidence of traumatic brain injuries (TBIs) is on the rise, and prompt diagnosis and treatment of these patients is vital. Globally, approximately 69 million individuals experience a TBI annually, with the highest prevalence occurring in low- and middle-income countries (4). In Kenya, the use of motorcycles as a means of public transport has increased by more than 33% in the past 15 years, leading to increased accidents and TBIs due to excessive speeding, overloading, riding under the influence of alcohol, and poor quality of roads in the country (5). Meningiomas and gliomas, which are the most common brain tumors among adults in Kenya, can also cause side-to-side mass effect, leading to KWNP (6). Thus, due to the high risk of lesions that may cause KWNP in the country, documenting novel presentations and developing an approach to diagnosis and treatment is vital.

The case report aims to describe a possible variation of KWNP, which presents with delayed hemiplegia and contralateral infarction of the PCA, causing visual changes. The case report also aims to describe the utility of Magnetic Resonance Imaging (MRI) in patients who have had traumatic brain injury (TBI) to diagnose KWNP and distinguish it from primary brainstem hemorrhage.

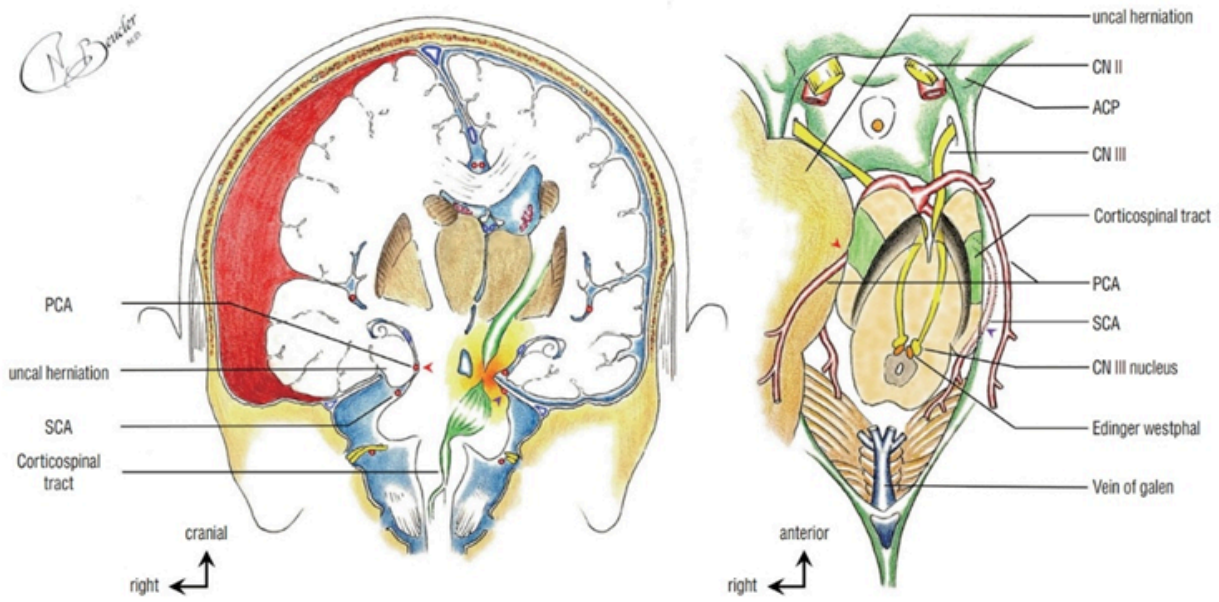

**Figure 1: An artistic view of Kernohan-Woltman Notch Phenomenon caused by a right-sided acute subdural hematoma in coronal and axial illustration. Reproduced from Beucler et al., Journal of Korean Neurosurgery Society 65:652-664, 2022, under CC BY-NC 4.0* (1).

CASE PRESENTATION

Patient information:

A 42-year-old male presented to the Accident and Emergency Unit (A&E) at Kenyatta National Hospital (KNH) following a road traffic accident. He presented with loss of consciousness for an unknown period of time. His Glasgow Coma Scale (GCS) was 10/15 (E2, V3, M5). He is a known tobacco smoker for the past 17 years (4.25 pack years). History of otorrhea, rhinorrhea, vomiting, and convulsions were also unknown; however, these symptoms were not experienced while he was admitted at the A&E unit.

Clinical findings:

The patient was unconscious on presentation. A primary survey revealed patent airways with no secretions, a clear C-spine, a respiratory rate of 15 breaths per minute, SpO2 of 96% on room air and vesicular breath sounds with good and equal chest rise bilaterally. His blood pressure was 116/76 mmHg and his heart rate was 46 beats per minute. His peripheries were warm with strong pulses, he was acyanotic and had no pallor. His temperature was 36.3 degrees Celsius. Most of his secondary survey was unknown, excluding his involvement in a road traffic accident.

A head-to-toe examination revealed superficial scalp abrasions, forehead and occipital scalp swellings with no lacerations. He had no lacerations or bruises on his neck. There were no lacerations, bruising or crepitus noted on chest examination. There was no step-off deformity, laceration or abrasion on his back. The abdomen was soft, non-tender without lacerations or abrasions. His pelvis was stable. There were minor abrasions on his upper and lower limbs, with no obvious deformity or swelling noted.

He was anisocoric, with a left sided mydriasis. The right pupil was reactive to light, while the left was sluggishly reactive. The remainder of the cranial nerve examination was unremarkable. The motor examination revealed patellar, Achilles, biceps and triceps hyperreflexia in the left upper and lower limb, with normal tone and reflexes on the right side. The left upper and lower limbs were also hypertonic. There was no hemiparesis or hemiplegia at this point. A comprehensive cerebellar examination was limited by the patient's lack of consciousness. The cardiovascular, respiratory and musculoskeletal examinations were unremarkable, except for the bradycardia described above.

Investigations:

During examination, blood samples were drawn for a full blood count (FBC), random blood sugar, arterial blood gas (ABG), urea, electrolytes and creatinine (UEC), international normalized ratio (INR) and grouping and cross matching (GXM). The results for these tests were normal, and the patient's blood type was successfully identified. An urgent non-contrast computerized tomography (CT) scan was also done, revealing an acute left subdural hematoma with midline shift, a depressed temporal skull fracture, an intracerebral contusion and cerebral edema, causing compression and effacement of the lateral ventricles and basal cisterns (Figures 2 and 3).

Diagnosis: Acute left subdural hematoma with midline shift, a biparietal skull fracture and intraparenchymal contusions secondary to traumatic brain injury.

Management:

The decision to operate was made based on the presence of a subdural hematoma, which caused midline shift, and the presence of a depressed skull fracture. Under general anesthesia and with the patient in supine position, a decompressive craniotomy was done to evacuate the subdural hematoma 4 hours after presentation. A subgaleal drain was left in situ.

Outcome and follow-up:

After the surgery, the patient was admitted to the Intensive Care Unit (ICU). Sedation was maintained with Dexmedetomidine, with laboratory tests such as UECs monitored daily. He was also kept on neuroprotective measures, with neuromonitoring done using the head injury chart. The plan was to maintain normocarbia (PC02 – 35-40mmHg), normotension (mean arterial pressure greater than 30, systolic blood pressure greater than 110 mmHg), normoglycemia (6-10 mmol/L), normothermia and SpO2 at 94% or higher. His fluid input and output were also monitored strictly. The subgaleal drain was monitored in the ICU, and removed 24 hours after the surgery as it drained less than 30 milliliters (mL) of blood. A post-operative CT scan was also done 24 hours after the surgery, showing the

evacuated hematoma (Figure 4). It was interesting that an infarct had developed in the PCA territory contralateral to the evacuated hematoma on the post-operative CT scan (Figure 5).

Examination on day 1 of admission in the ICU revealed that the mydriasis and anisocoria reported before surgery had resolved. He had also developed left sided hemiplegia, with power of 0/5 on the Medical Research Council (MRC) scale on the left upper and lower limbs. The right upper and lower limbs had a power of 5/5 on the same scale. The GCS at this point was 3T/15. On day 2, GCS improved to 8T/15, which was the same on day 3. On day 4, GCS improved to 10T/15. Progressive weaning to continuous positive airway pressure (CPAP) was started, with simultaneous weaning off sedation. On day 6, he was extubated and placed on a non-rebreather mask (NRM). He gained consciousness on the same day, with a GCS of 12/15. On day 7, his GCS improved to 13/15 and he was de-escalated to ward care. He was discharged to the ward on day 8, with a GCS of 13/15, and persistent left sided hemiplegia of the upper and lower limbs.

His stay in the ward was unremarkable, as he was fed via nasogastric tube (NGT) before soon tolerating oral feeding (day 10). His fluid intake and output were continuously monitored, and laboratory tests (ABG, FBC, UEC and blood sugar) were all normal. His GCS improved to 15/15 on day 13 of admission. On day 13, he also complained of an "inability to see" from his left eye, which also persisted. The hemiplegia gradually improved, with power of MRC grade 2/5 on both left upper and lower limbs reported on day 15, MRC grade 3/5 on day 18 and MRC grade 4/5 on day 23. He was discharged 4 weeks after surgery with a power of MRC grade 5/5 on the right and left upper and lower limbs. He was discharged for perimetry, and the plan was to follow up on his condition two weeks later.

He was discharged on Levetiracetam 500mg BID for 7 days, paracetamol 1 g QID, and esomeprazole 40 mg OD. He was instructed to take the medication as prescribed and report back to the hospital if he developed any danger signs, including seizures, severe headaches, projectile vomiting, or discharge from the incision site. He was also instructed to return to the clinic 2 weeks after discharge having done perimetry to continue his care. On the first clinic visit, he still had adequate muscle power (MRC 5/5 bilaterally) but had not gone for perimetry due to financial constraints.

Ethics:

Written informed consent was obtained from the patient for the publication of this case report and accompanying images. All identifying information such as the patient's name and initials have been omitted to protect patient confidentiality.

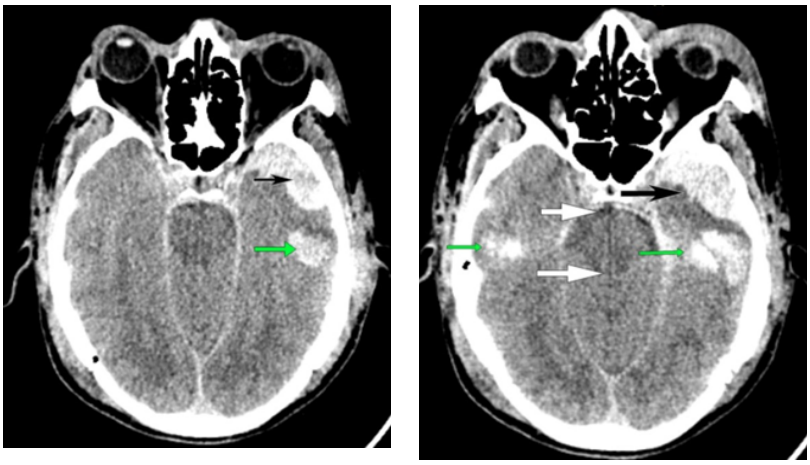

Figure 2: A preoperative brain window CT scan. The image shows an acute left subdural hematoma (black arrows), acute intraparenchymal contusions (green arrows), and cerebral oedema effacing the interpeduncular and quadrigeminal cisterns (white arrows).

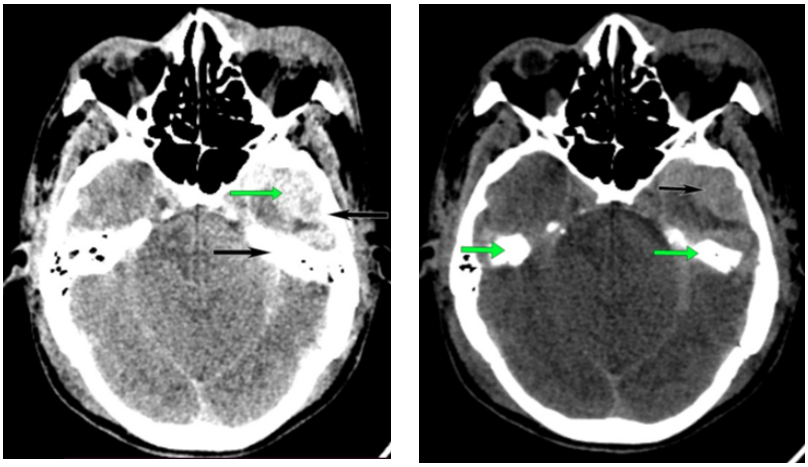

Figure 3: An axial view of the pre-operative CT scan, brain window (a) and a subdural window CT scan (b). Figure (a) shows the acute left subdural hematoma (green arrow) and the temporal bone (black arrows). Figure (b) shows the subdural hematoma (black arrow) and the temporal bone (green arrows). The subdural window was obtained using a window length of 80 Hounsfield Units, and a window width of 150 Hounsfield Units.

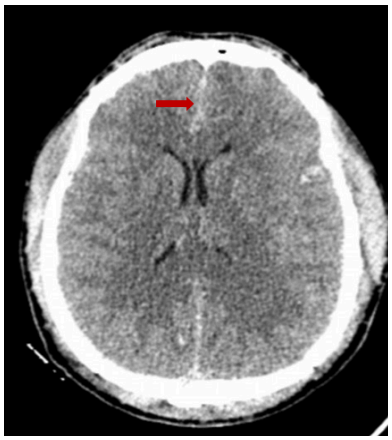

Figure 4: An axial view of the pre-operative brain window CT scan. The image shows deviation of the falx cerebri towards the right, indicative of a midline shift (red arrow). The image also shows compression and effacement of the lateral ventricles, with the left lateral ventricle appearing more effaced than the right.

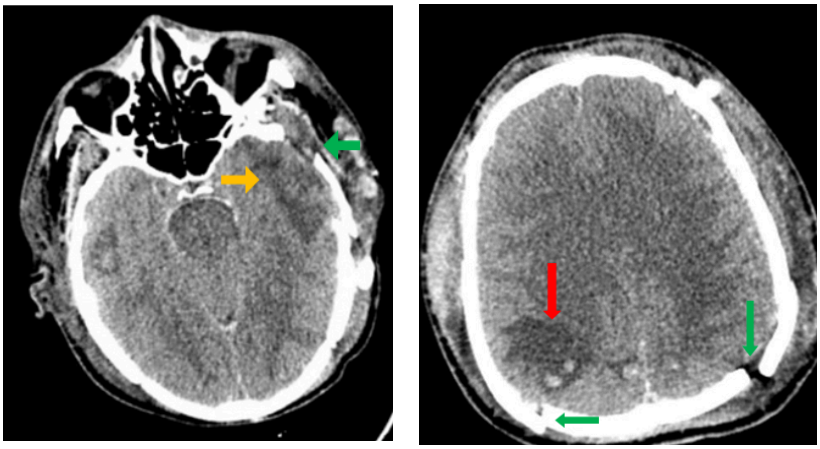

Figure 5: (**a**) An axial view of the post-operative brain window CT scan. The image shows a resolved subdural hematoma and the craniotomy performed on the patient's left temporal bone (yellow arrow). (**b**) The image shows a right-sided, wedge-shaped lesion visible in the occipital lobe, consistent with the features of a PCA infarct. The image also shows the biparietal skull fracture (green arrows).

DISCUSSION

This case report presents a patient who had an acute left subdural hematoma, a biparietal skull fracture and intraparenchymal contusions secondary to traumatic brain injury. On examination, the patient had anisocoria with left-sided mydriasis, a sluggish light response on the left pupil (right pupil was reactive), hyperreflexia and hypertonia in the left upper and lower limbs with normal reflexes and tone in the right upper and lower limbs. After decompressive craniotomy and elevation of the fracture, the patient developed hemiplegia (MRC scale 0/5) on his left upper and lower limbs, which resolved completely (MRC scale 5/5 for both limbs) when he was discharged 4 weeks later. He also developed an "inability to see" from his left eye which persisted on follow-up 2 weeks after discharge.

The anisocoria with left-sided mydriasis and sluggish response to light on the left side are characteristic findings in KWNP, due to compression of the oculomotor nerve on the side ipsilateral to the hematoma as it emerges at the level of the superior colliculus between the superior cerebellar and posterior cerebral arteries (SCA and PCA). However, the paradoxical hemiplegia occurred after the hematoma was evacuated, representing a possible variation of KWNP presentation where hemiplegia is delayed. This variation has been recorded in the cases presented by Eschanola et al. and Namura (7,8). Recognition of this variation is crucial in clinical practice, as it contributes to diagnostic formulation for patients who develop hemiplegia and visual changes after decompressive craniotomy for subdural hematomas.

A PCA infarct located on the right occipital lobe (contralateral to the hematoma) was found in our patient's post-operative CT scan. This infarct's presence is in tandem with the literature, which states that KWNP does not occur solely due to compression of the contralateral crus cerebri, as ischemic events secondary to compression or distortion of the perforating arteries arising from the PCA and the SCA also contribute (9). The PCA ipsilateral to the hematoma may be compressed between the herniated temporal lobe medially, and the tentorial incisura laterally, while the PCA contralateral to the hematoma may be compressed between the displaced brainstem medially and the tentorial incisura laterally (8). The latter was the likely mechanism behind PCA compression in our case. Contralateral PCA compression is rare, and infarction of the ipsilateral PCA or both arteries is more common (8). The case by Namura et al. was the only reviewed case that featured a patient with KWNP and a contralateral PCA infarct confirmed on MRI. Similar to our patient, Namura et al.'s case also featured visual field defects, with a diagnosis of ipsilateral homonymous hemianopsia (8). While perimetry was not done for our patient, his "inability to see" from his left eye was likely due to left homonymous hemianopsia secondary to compression of the right PCA. Interestingly, our case was the only case we found on review featuring both delayed hemiplegia and a contralateral PCA infarct leading to vision changes.

MRI may be essential for the diagnosis of KWNP and exclusion of differential diagnoses. Non-contrast CT scans are performed for patients with suspected head injuries as they are fast and easily accessible. However, Kernohan's notch is very small and is located in the cerebral peduncle, which is very close to the skull base, making it difficult for CT scans to depict (10). Indeed, Kernohan's notch was not depicted on our patient's pre-op and post-op CT scans. As shown in figure 6, MRI images reveal a hyperintense, rounded lesion on T2-weighted images contralateral to the hematoma, indicating compression of the cerebral peduncle (8,11). The lesion appears hypointense or isointense on T1-weighted images (2). This lesion represents a degradation of myelinized fibers in the corticospinal tract caused by compression (12). These findings aid not only in the diagnosis of KWNP, but in the differentiation of KWNP from primary brainstem injury, which may also occur after TBI. This differentiation is important, as primary brainstem injuries produce more profound and prolonged impairments in the level of consciousness. Primary brainstem hemorrhages like Duret hemorrhages occur in the midline of the lower mesencephalon and upper pons, allowing for their differentiation from KWNP, whose lesions occur in the cerebral peduncles (12). A limitation of MRIs for such cases is that they are expensive. The MRI's cost was also the reason why our patient did not have an MRI scan done.

The lessons learned from the case include the presence of a possible variation in the presentation of KWNP, where the hemiplegia is delayed. Therefore, recognizing this variant presentation allows for broadening of the

post-operative differential diagnosis for hemiplegia. Ischemic events also contribute to KWNP's pathophysiology, including compression of either the ipsilateral PCA, the contralateral PCA, or both arteries. Also, MRI can be useful in differentiating KWNP from primary brainstem hemorrhages, and can be used to confirm KWNP diagnosis, as Kernohan's notch is not visible on CT scan. This case also highlights barriers to patient care in our setting. High cost prevented the patient from getting an MRI and perimetry. Indeed, policy changes are necessary to bridge the gap in healthcare access in our setting.

The limitations of the study include a lack of MRI, which would have confirmed the diagnosis, and a lack of perimetry, which would have confirmed the ipsilateral homonymous hemianopsia. There is need for more MRI-led research on KWNP to accurately describe the variations in the phenomenon's presentation, and the factors causing these variations, such as anatomical differences in the width of the tentorial notch.

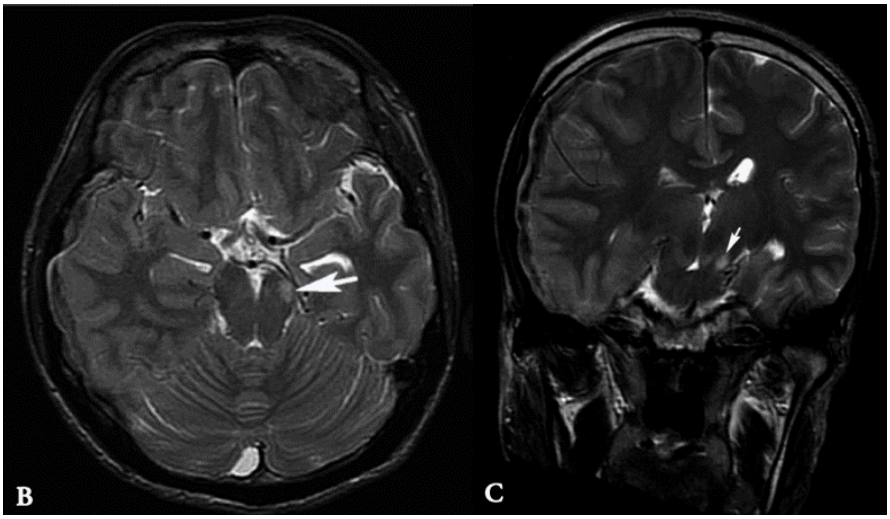

Figure 6: The image on the left shows a small area that is isointense (white arrow) on T1-weighted imaging (B) and hyperintense on T2-weighted images (C) in the left cerebral peduncle. Reproduced with permission from Chen et al., Hong Kong Journal of Emergency Medicine, 21 (2), 116-119, 2014, under the All Rights Reserved Copyright Agreement (13).

CONCLUSION

In conclusion, delayed hemiplegia is a variation of KWNP presentation. Recognizing the occurrence of delayed hemiplegia broadens the diagnostic formulation for patients who develop hemiplegia after decompressive craniotomy. KWNP also causes ischemic events secondary to compression of the PCA and SCA. Compression of the PCA contralateral to the hematoma is also rare, and may cause visual changes in ipsilateral vision as demonstrated in this case report. The case presented above discusses a rare occurrence where both delayed hemiplegia and contralateral PCA infarction were present. MRI may be essential in the diagnosis of KWNP and distinction of this condition from other possible diagnoses, such as primary brainstem haemorrhages like Duret hemorrhage.

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Author contributions: LGG, WLN and MSM: Conceptualization, authorship of the original draft, review and editing, and image preparation. KRS: Conceptualization, supervising author, review and editing.

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