CASE REPORT

Post-traumatic Occipital Cerebral Infarction following Decompressive Craniectomy for Severe TBI

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ABSTRACT

Background: Post-traumatic cerebral infarction (PTCI) is a severe consequence of secondary brain injury which is associated with poor functional recovery and high mortality. Despite advances in neurocritical care and decompressive craniectomy, outcomes remain poor.

Case Presentation: We report a 22-year-old female who sustained a severe TBI following a road traffic accident. On admission, her Glasgow Coma Scale (GCS) was 8/15 with sluggishly reactive pupils. A non-contrast CT scan revealed a left parietal-temporal acute subdural hematoma with 13 mm midline shift and multiple contusions. Emergency decompressive craniectomy was performed. Despite surgical decompression and neurocritical management, her neurological status deteriorated, and a follow-up CT scan done on postoperative day three revealed a left occipital infarction in the PCA (occipital) territory consistent with transtentorial herniation. The patient died on postoperative day eight.

Conclusion: Occipital PTCI remains a catastrophic but often under-recognized complication of TBI. Early detection through neurological monitoring and timely repeat neuroimaging, coupled with intracranial pressure and cerebral perfusion control, may mitigate risk. However, prognosis remains guarded, emphasizing the need for improved neuroprotective and vascular-targeted interventions.

INTRODUCTION

Post-traumatic cerebral infarction (PTCI) represents one of the most devastating secondary injuries following traumatic brain injury (TBI), contributing significantly to morbidity and mortality despite optimal medical and surgical management. Its incidence varies between 2–12% in clinical series and may reach 90% in postmortem studies, highlighting frequent underdiagnosis during acute care phases[1,2]. PTCI typically occurs in patients with moderate to severe TBI, and it is recognized as an independent predictor of poor neurological outcome[3]. The pathophysiology is multifactorial, including cerebral herniation, arterial dissection, traumatic vasospasm, cerebral hypoperfusion, and microvascular dysfunction[5]. Among the vascular territories, infarction within the posterior cerebral artery (PCA) distribution is most frequently

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Keywords: Traumatic brain injury; post-traumatic cerebral infarction; Posterior cerebral artery; Decompressive craniectomy; Case report.

This article is available online at: http://www.mjz.co.zm, http://ajol.info/index.php/mjz, doi: https://doi.org/10.55320/mjz.53.1.915

The Medical Journal of Zambia, ISSN 0047-651X, is published by the Zambia Medical Association

reported, often secondary to uncal or transtentorial herniation compressing the PCA against the tentorial edge[6].

Advances in neuroimaging, particularly early post-injury CT and MRI, have facilitated better identification of these infarcts; however, outcomes remain dismal. Even with decompressive craniectomy and optimized critical care, mortality rates remain as high as 40–80%, particularly in cases associated with diffuse edema and refractory intracranial hypertension[7]. Recent literature has emphasized the importance of understanding secondary ischemic cascades—including excitotoxicity, blood–brain barrier disruption, and inflammatory endothelial injury—which contribute to delayed infarction after TBI[8]. Preventive strategies are centered on maintaining adequate cerebral perfusion pressure (CPP) [50-70mmHg], early recognition of herniation syndromes, and aggressive management of intracranial hypertension[10].

Here, we present a case of a 22-year-old female who developed a left occipital infarction in the PCA territory following decompressive craniectomy for a left acute subdural hematoma, illustrating the pathophysiological interplay between traumatic herniation and secondary ischemic injury. With limitations in lack of ICP/advanced neuro-monitoring, management was based on serial clinical examination and CT image findings.

Case description

A 22-year-old previously healthy and functionally independent female, with no co-morbidities, was brought to the emergency department following a high-impact road traffic accident. Witnesses reported that she was thrown out of a moving truck after consuming alcohol. On arrival, her **airway, breathing, and circulation were stable**, with a **blood pressure of 118/74 mmHg, pulse rate of 92 beats/min**, and **oxygen saturation of 98% on room air.** Her **Glasgow Coma Scale (GCS)** score was **8/15 (E2, V1, M5),** and she exhibited **bilaterally dilated pupils (5 mm) with sluggish reaction to light**. Physical examination revealed a **swollen forehead with multiple facial abrasions**, but no signs of **basal skull fracture** (no periorbital ecchymosis, rhinorrhea, or otorrhea). Systemic examination was unremarkable with no long-bone, chest, or abdominal injuries. Past medical history was not indicative of any prior head injury or neurological illness.

Given her reduced level of consciousness, she was **intubated and placed on mechanical ventilation**. Initial **non-contrast brain computed tomography (CT)** revealed a **left parietal–temporal acute subdural hematoma, multiple intracerebral contusions, cerebral edema,** and a **midline shift of approximately 13 mm** (Figure 1A–B). There was effacement of the left lateral ventricle and compression of basal cisterns, consistent with raised intracranial pressure and impending herniation.

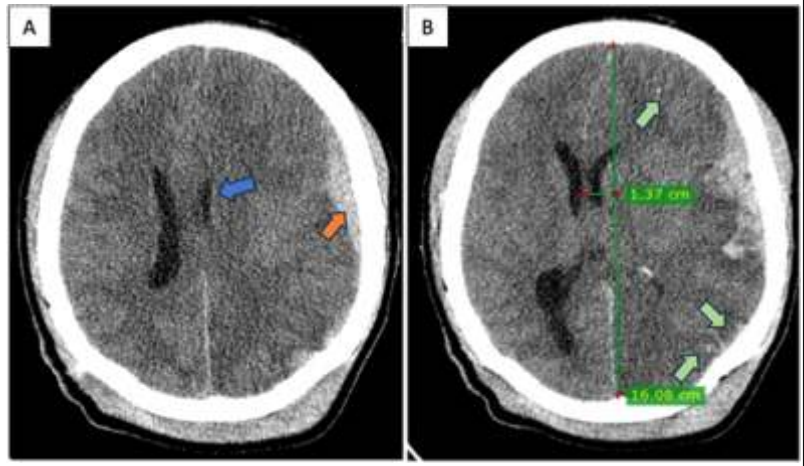

Figure 1. *Preoperative non-contrast brain CT scan.* (A) Left temporal subdural hematoma (orange arrow) with effacement of the left lateral ventricle (blue arrow) and marked cerebral edema. (B) Midline shift measuring 13.7 mm with multiple intracerebral contusions (green arrow) with no evidence of left PCA (occipital) infarction.

The patient was optimized for emergency surgery. Informed consent was obtained from the next of kin for a left decompressive craniectomy. Intravenous mannitol (0.5 g/kg) was administered before skin incision and repeated after hematoma evacuation to reduce cerebral edema. Hypertonic saline was not available. Intraoperatively, a 15 × 12 cm left fronto-temporo-parietal decompressive craniectomy was performed. Findings included a markedly swollen brain, acute subdural hematoma of approximately 50 mL, a temporal laceration with necrotic brain tissue, multiple cortical contusions, and thrombosed dural vessels. The hematoma was evacuated, necrotic tissue debrided, and the surgical field irrigated with warm saline. The dura was repaired and hitched to the temporalis fascia to minimize epidural dead space. An epidural drain was left in situ, and the bone flap was not replaced due to cerebral swelling. The scalp was closed in layers.

Postoperatively, she was admitted to the intensive care unit (ICU) for ventilatory support, intracranial pressure management and optimal control of temperature, blood glucose and PaCO2. Despite sedation, analgesia, and continued osmotic therapy, her neurological status worsened, with GCS declining to 3T/10 and bilateral fixed, dilated pupils (5 mm). She developed persistent fever, though blood cultures were negative.

On postoperative day three, a repeat non-contrast brain CT showed new left occipital hypodensity within the posterior cerebral artery (PCA) territory, consistent with ischemic infarction. There was also evidence of downward transtentorial herniation and progressive cerebral edema (Figure 2).

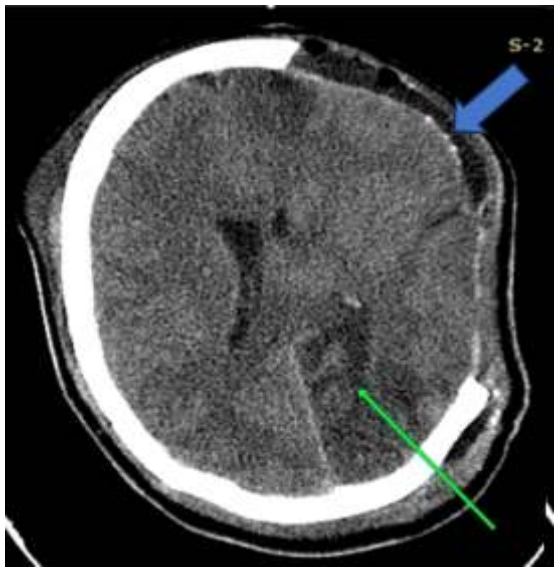

Figure 2. *Post-decompressive craniectomy CT scan* – Showing a left posterior cerebral artery territory infarction (green arrow) with brain tissue herniating through the craniectomy defect and diffuse cerebral swelling (blue arrow).

By postoperative day six, an early tracheostomy was performed in anticipation of prolonged mechanical ventilation. Despite aggressive supportive management, her neurological condition continued to deteriorate, and she succumbed on postoperative day eight.

Key event	Injury to ED	Admission to pre-OP CT	Admission to Emergency craniectomy	ICU admission	Post-OP CT	Tracheostomy	Death
Timeline	2 hours	1 hour	3 hours	POD 0	POD 3	POD 6	POD 8

Table 1.0-Timeline of key events from admission to Death

DISCUSSION

PTCI is increasingly recognized as a critical determinant of outcome in patients with moderate to severe TBI. Despite advances in neurocritical care, its pathophysiology remains complex and incompletely understood. The condition represents a final common pathway of secondary ischemic injury resulting from systemic, vascular, and intracranial mechanisms[1] (figure 3).

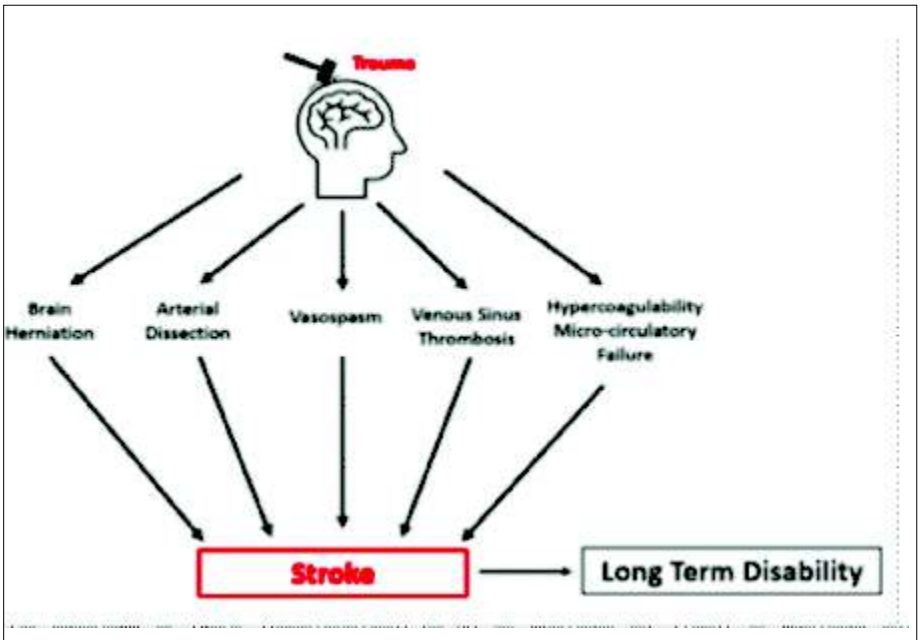

Figure 3 – Proposed mechanisms leading to post-traumatic cerebral infarction: (A) vascular compression during herniation, (B) arterial dissection, (C) vasospasm, (D) microvascular endothelial injury (1).

Note: *Adapted from: Poblete RA, Zhong C, Patel A, Kuo G, Sun PY, Xiao J, et al. post-traumatic cerebral infarction: A narrative review of pathophysiology, diagnosis, and treatment. Neurol Int. 2024;16(1):96-97*[1].

Literature review

Recent studies have identified decompressive craniectomy, low admission GCS, and mass effect lesions as independent predictors of PTCI[3,4]. Shen et al. (2025) demonstrated that patients undergoing unilateral decompressive craniectomy for severe TBI had a 13–16% risk of developing delayed infarction, predominantly in the PCA territory[4]. This aligns with our observation that, despite adequate surgical decompression, vascular compromise may persist due to tentorial distortion and autoregulatory failure. Furthermore, microvascular and endothelial dysfunction have been implicated as key contributors to secondary ischemia following TBI[8]. Serban et al. (2025) described early cerebrovascular dysautoregulation and endothelial activation as initiating factors for ischemic transformation, independent of large-vessel compression[8]. These findings underscore the need for multimodal neuromonitoring—including transcranial Doppler ultrasonography, perfusion CT, and continuous ICP–CPP coupling assessment to detect early hypoperfusion[10].

Principle findings

Our case highlights a classic presentation of PCA infarction following downward transtentorial herniation after decompressive craniectomy. In such cases, displacement of the medial temporal lobe compresses the ipsilateral PCA against the tentorial edge, compromising perfusion to the occipital cortex[2,7]. This pathomechanism is supported by radiological and autopsy studies linking uncal herniation with occipital lobe infarction ipsilateral to the lesion[5,9].

From a preventive standpoint, early recognition of herniation signs (pupillary asymmetry, decerebrate posturing, abrupt neurological decline) and immediate decompression remain crucial. However, excessive decompression can paradoxically alter cerebral hemodynamics and venous return, leading to localized hypoperfusion zones—termed the "paradoxical ischemia" phenomenon[7].

Therapeutically, recent reviews have explored endovascular and neuroprotective strategies to minimize secondary ischemia, including vasodilator therapy, hypothermia, and stem-cell-derived

extracellular vesicles targeting post-traumatic inflammation[9]. Nevertheless, clinical evidence remains limited, and outcomes are largely dictated by initial injury severity and herniation duration.

In our patient, the constellation of low GCS, massive subdural hematoma, transtentorial herniation, and postoperative PCA infarction underscores the interplay between mechanical distortion and impaired autoregulation in the genesis of PTCI. Despite optimal surgical decompression, secondary ischemia developed, culminating in fatal progression—consistent with current literature on poor outcomes in such presentations[2,3,4].

Limitations

Non-availability of ICP monitoring and routine CT perfusion or CTA pose a diagnostic challenge in early identification of such complications as cerebral infarction.

CONCLUSION

Post-traumatic cerebral infarction (PTCI), particularly within the posterior cerebral artery territory, is a devastating secondary complication of severe traumatic brain injury resulting from mechanical compression during transtentorial herniation, vascular distortion, and microvascular dysfunction. Despite timely decompressive craniectomy and optimal neurocritical care, outcomes remain poor due to irreversible ischemic cascades. Early identification of at-risk patients through vigilant monitoring, prompt neuroimaging, and meticulous control of intracranial pressure and cerebral perfusion is essential to reduce occurrence. Future research should prioritize neuroprotective strategies targeting endothelial stabilization and cerebral autoregulation to improve survival and neurological recovery.

LEARNING POINTS

1. PTCI commonly complicates severe TBI, especially following transtentorial herniation and decompressive craniectomy.
2. Early neuroimaging and vigilance for secondary ischemic injury are essential for timely diagnosis.
3. Preventive strategies focusing on ICP–CPP control and microvascular preservation may reduce risk.
4. Despite intervention, mortality remains high, warranting innovation in neuroprotective and vascular therapies.

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Declarations

Competing interests

The authors declare that they have no competing interests.

Funding

Not applicable

Authors' contributions

All authors read and approved the final manuscript.

Patient/Family perspective

Patient's guardians were aware of the extent and severity of the injury and guarded outcome as they were communicated to at every point in management.

Informed consent

Informed consent was obtained from the caregiver.