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http://jept.ir
doi:10.34172/jept.1789

Journal of Emergency Practice and Trauma
J Emerg Pract Trauma 2025;11(1):65-67

Case Report

Traumatic Basal Ganglia Hemorrhage Following Motor Vehicle Accident: A Case Report

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Received: August 10, 2025
Accepted: November 28, 2025
ePublished: June 7, 2026

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Citation: Ertuğrul B, Akçin İ. Traumatic basal ganglia hemorrhage following motor vehicle accident: a case report. J Emerg Pract Trauma 2025;11(1):65-67. doi:10.34172/jept.1789

Abstract

Introduction: Traumatic basal ganglia hemorrhage is a rare phenomenon compared with spontaneous hypertensive intracerebral hemorrhage. We aimed to present a rare case of traumatic basal ganglia hemorrhage following a motor vehicle accident and discuss its clinical and radiological features.

Case Presentation: A 59-year-old woman was admitted to the emergency department after a motor vehicle accident with confusion and left-sided weakness. Her Glasgow Coma Scale score was 12. Cranial computed tomography revealed a right basal ganglia hematoma measuring approximately 26 × 20 mm without midline shift or intraventricular hemorrhage. The patient was managed conservatively with antiedema treatment and close neurological follow-up. Follow-up imaging demonstrated gradual hematoma resolution, and the patient recovered without neurological deficit.

Conclusion: Traumatic basal ganglia hemorrhage is an uncommon form of traumatic intracerebral hemorrhage. Careful differentiation from spontaneous hypertensive hemorrhage is essential. Conservative management may provide favorable outcomes in patients without significant mass effect or neurological deterioration.

Keywords: Basal ganglia hemorrhage, Traumatic brain injury, Intracerebral hemorrhage, Motor vehicle accident

Introduction

Intracerebral hemorrhage is a common form of cerebrovascular disease, and the basal ganglia are the most frequent location of spontaneous nontraumatic hemorrhage, most commonly associated with hypertension (1). In contrast, traumatic basal ganglia hemorrhage (TBGH) is a relatively rare phenomenon and accounts for approximately 2%–3% of traumatic brain injuries diagnosed radiologically, although higher incidences have been reported in autopsy series (2,3). TBGH is defined as hemorrhage involving the basal ganglia and adjacent structures such as the internal capsule or thalamus following head trauma.

The exact pathophysiology of traumatic basal ganglia hemorrhage remains unclear. It has been suggested that acceleration-deceleration forces and shear strain may lead to injury of the lenticulostriate or anterior choroidal arteries, resulting in hemorrhage within deep brain structures (3-11). These lesions are frequently associated with severe traumatic brain injury and may coexist with diffuse axonal injury, cerebral contusions, or other intracranial pathologies. Recent reports have demonstrated that isolated traumatic basal ganglia hemorrhage without accompanying intracranial lesions is extremely rare (10-14).

The prognosis of TBGH varies according to hematoma size, associated intracranial injuries, Glasgow Coma Scale score at admission, and the presence of mass effect or intraventricular extension. While some patients require surgical intervention, some patients with isolated lesions and stable neurological findings may be successfully managed conservatively (12-15).

Differentiating traumatic basal ganglia hemorrhage from spontaneous hypertensive hemorrhage is clinically and forensically important, particularly in patients presenting after motor vehicle accidents or blunt head trauma. In this report, we present a rare case of isolated traumatic basal ganglia hemorrhage following a motor vehicle accident and discuss its possible mechanisms, radiological characteristics, and management strategies.

Case Presentation

A 59-year-old female patient was admitted to the emergency department following a motor vehicle accident. On admission, the patient had confusion, epistaxis, and multiple superficial abrasions on the face and extremities. No major extracranial injury was identified. The patient had no history of hypertension, diabetes mellitus, anticoagulant or antiplatelet drug use, alcohol abuse, or bleeding disorder.

On neurological examination, she was confused, her eyes opened in response to painful stimuli, and she was spontaneously mobile. Her Glasgow Coma Scale score was 12. Bilateral pupillary light reflexes were intact, and motor examination demonstrated 4/5 muscle strength in the left upper extremity.

Laboratory investigations including complete blood count, biochemical parameters, and coagulation profile were within normal ranges.

Cranial computed tomography demonstrated a hyperdense hematoma located in the right basal ganglia region involving the caudate nucleus, measuring approximately 26×20 mm. No intraventricular hemorrhage, hydrocephalus, midline shift, or significant mass effect was observed. No additional intracranial traumatic lesion such as epidural hematoma, subdural hematoma, or cerebral contusion was identified (Figure 1).

The patient was admitted to the intensive care unit for close neurological monitoring. Since the patient had moderate traumatic brain injury and focal neurological deficit, antiedema treatment with mannitol and furosemide was initiated to reduce intracranial pressure. Prophylactic phenytoin treatment was administered to reduce the risk of early post-traumatic seizures.

Follow-up cranial CT performed 8 hours post-admission demonstrated no hematoma expansion. Conservative treatment was continued. During hospitalization, the patient showed gradual neurological improvement, and no additional neurological deficit developed. On the fourth day of hospitalization, the patient was transferred to the neurosurgery ward. Follow-up CT obtained on the ninth hospital day demonstrated marked hematoma resorption. The patient was discharged without neurological deficit.

Discussion

Traumatic basal ganglia hemorrhage is an uncommon form of traumatic intracerebral hemorrhage and is generally associated with high-energy trauma. Although spontaneous basal ganglia hemorrhage is most commonly related to hypertension, traumatic lesions may occur secondary to acceleration-deceleration injury causing shear stress on small penetrating arteries such as the lenticulostriate or anterior choroidal arteries (3,5,6,10,11).

Traumatic basal ganglia hemorrhages are rare and account for approximately 2%–3% of traumatic brain injuries diagnosed radiologically (2,3). Isolated lesions without accompanying intracranial pathologies such as epidural hematoma, subdural hematoma, subarachnoid hemorrhage, cerebral contusion, or diffuse axonal injury are considerably rarer and have mainly been reported as isolated case reports in recent literature (10-14). In the present case, no additional traumatic intracranial lesion was identified, supporting the diagnosis of isolated traumatic basal ganglia hemorrhage.

The exact mechanism of traumatic basal ganglia hemorrhage remains unclear. Previous autopsy studies have demonstrated rupture of pallidal branches of the anterior choroidal artery as a possible source of hemorrhage (7). In addition, injury to branches of the middle cerebral artery has also been proposed as a contributing mechanism (8,9).

Radiological differentiation between traumatic and spontaneous hemorrhage may sometimes be difficult. However, the temporal relationship with trauma, absence of hypertension history, lack of anticoagulant use, and stable radiological course supported traumatic etiology in our patient.

The prognosis of traumatic basal ganglia hemorrhage depends on hematoma size, associated intracranial injuries, neurological findings at admission, and development of mass effect. Hemorrhages larger than 2 cm are generally considered large lesions (2,4). Since the hematoma size in our patient was approximately 26×20 mm, the lesion could be considered borderline large.

Management strategies depend on neurological status, hematoma size, and radiological progression. Conservative treatment and close radiological follow-up may provide favorable outcomes in some patients who show no significant mass effect or neurological deterioration (12-15). In our patient, surgical intervention was not required

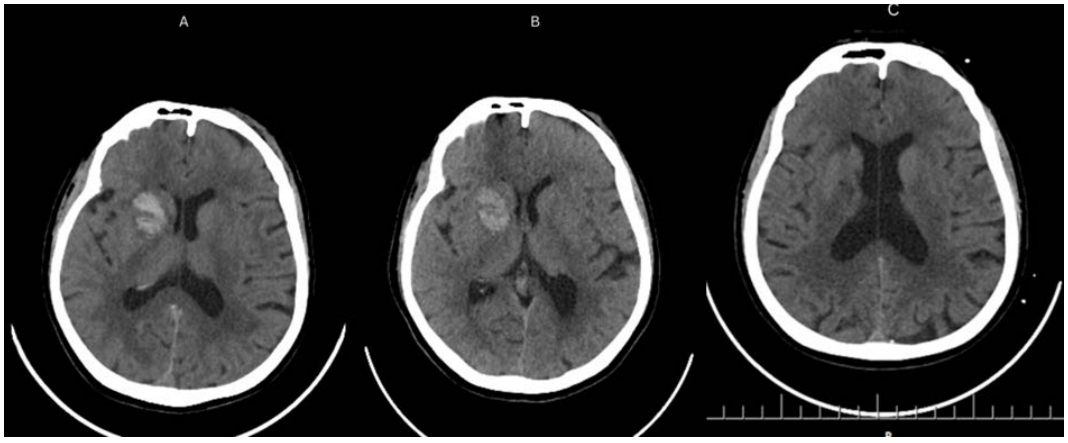

Figure 1. Serial cranial computed tomography images of the patient
(A) Admission CT scan demonstrating right basal ganglia hemorrhage
(B) Follow-up CT scan obtained 8 hours after hospitalization showing stable hematoma size without expansion
(C) Follow-up CT scan obtained on the ninth hospital day demonstrating marked hematoma resorption

because neurological findings improved during follow-up, no hematoma enlargement was observed on serial CT imaging, and significant hematoma resorption developed with conservative management.

Conclusion

Traumatic basal ganglia hemorrhage is a rare form of traumatic intracerebral hemorrhage and should be differentiated carefully from spontaneous hypertensive hemorrhage. Clinical history, radiological findings, and associated traumatic lesions are important for accurate diagnosis. Isolated traumatic basal ganglia hemorrhages without significant mass effect may have favorable outcomes with conservative management and close neurological follow-up.

Authors' Contribution

Conceptualization: Bilal Ertuğrul
Data curation: İsmail Akçin
Formal analysis: Bilal Ertuğrul; İsmail Akçin
Funding acquisition: None
Investigation: Bilal Ertuğrul
Methodology: Bilal Ertuğrul
Project administration: Bilal Ertuğrul
Resources: Bilal Ertuğrul; İsmail Akçin
Software: None
Supervision: İsmail Akçin
Validation: İsmail Akçin
Visualization: İsmail Akçin
Writing–original draft: Bilal Ertuğrul
Writing–review & editing: Bilal Ertuğrul; İsmail Akçin

Competing Interests

None.

Ethical Approval

Ethical approval was waived because this study was a single case report. Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Funding

None.

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