Original Research

# TRAUMATIC BASAL GANGLIA HEMATOMA: IMAGING PATTERNS, TREATMENT OUTCOMES, AND SUPERIOR RECOVERY COMPARED TO SPONTANEOUS BASAL GANGLIA BLEED - SJMCH EXPERIENCE

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## ABSTRACT

**Background**: Traumatic basal ganglia hematoma (TBGH) is a rare but distinct deep parenchymal injury occurring in 2–3% of closed head injuries, predominantly affecting young males following high-velocity road traffic accidents. Unlike spontaneous hypertensive basal ganglia hemorrhage, TBGH arises from shear-induced tearing of perforating vessels (lenticulostriate and anterior choroidal artery branches) during acceleration-deceleration forces, producing smaller, multifocal lesions (<2.5 cm diameter). Characteristic associated findings include gray-white matter junction contusions (universal), intraventricular/subarachnoid hemorrhage (60%), brainstem contusions (70%), and skull fractures (50%, often mandibular/zygomatic).

**Methods**: A 10-patient case series from SJMCH Neurosurgery was reviewed, integrating pathophysiology, imaging hallmarks, and management principles. Patients were managed per traumatic brain injury (TBI) protocols—emphasizing airway stabilization, ICP monitoring, osmotherapy, and serial CT—rather than spontaneous intracerebral hemorrhage algorithms. Functional outcomes were assessed using RAS scoring at 3 months.

**Results**: All patients were male with a mean age of 38.7 years. Lesions were right-sided in 60%, with a mean diameter of 1.8 cm (range 1.09–2.9 cm). Epicenters included the internal capsule (40%) and lenticular nucleus (30%), with involvement also of the thalamus, external capsule, and putamen. Eighty percent were managed conservatively and decompressivecraniectomy was performed in select cases with significant mass effect. Three-month RAS scoring demonstrated superior functional recovery compared to historical spontaneous bleed outcomes (mortality 40–50%, persistent hemiparesis).

**Conclusion**: TBGH differs critically from hypertensive hemorrhage by demographics (young, non-hypertensive patients), radiological features (shear stigmata versus solitary clots >3 cm), and prognosis (neuroplasticity in TBI favors recovery in the absence of vascular risk factors). Early recognition and management within a neurotrauma framework enables optimistic outcomes at specialized centers.

**Keywords:** Traumatic Basal Ganglia Hematoma, Traumatic Brain Injury, Diffuse Axonal Injury, Gangliocapsular Hemorrhage, Neurotrauma.

## INTRODUCTION

Traumatic basal ganglia hematoma refers to hemorrhage in the basal ganglia or neighboring deep structures such as the internal capsule and thalamus after head injury. It is uncommon in clinical series, often around 2% to 3% of closed head injuries, though autopsy studies suggest higher rates, implying that it may be underrecognized in severe trauma. Most patients are young men injured in road traffic accidents, and associated lesions such as contusions, diffuse axonal injury, intraventricular hemorrhage, subarachnoid hemorrhage, extradural hematoma, subdural hematoma, or brainstem injury are common rather than exceptional.

Historically, these lesions were grouped with diffuse axonal injury, but later work described them as a distinct entity, sometimes called an “intermediate coup contusion,” because they occur deep within the brain between the classic coup and contrecoup zones. Clinically, the lesion itself matters, but outcome is heavily driven by the total injury burden, especially the presence of low GCS, pupillary abnormality, midline shift, raised intracranial pressure, and associated diffuse or brainstem injury. The present article reviews the pathophysiology, management principles, prognostic determinants, and distinguishing features of traumatic basal

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ganglia hematoma. In addition, it incorporates a short case series of 10 patients managed in the Department of Neurosurgery at St. John's Medical College Hospital, in whom follow-up at 3 months demonstrated better functional prognosis based on RAS scoring.

**Pathophysiology**

The most accepted mechanism is shear-related tearing of deep perforating vessels during rapid acceleration-deceleration trauma. When impact is delivered to the vertex, forehead, or occiput while the head is moving, the brain shifts toward the tentorium, stretching and tearing the pallidal branches of the anterior choroidal artery and the striate branches of the middle cerebral artery, producing hemorrhage in the basal ganglia region. Other proposed mechanisms include rotational inertial injury, traumatic dissection of middle cerebral artery branches, and vascular injury occurring as part of the same biomechanical process that produces diffuse axonal injury.

This explains why traumatic basal ganglia hematoma often coexists with gray-white matter junction contusions, brainstem contusions, corpus callosal injury, and intraventricular hemorrhage. Small lesions may behave more like hemorrhagic contusions within a diffuse injury pattern, whereas large lesions may act like true hematomas with mass effect and intracranial hypertension. Internal capsule involvement is common, which also explains the frequency of hemiparesis and long-term residual weakness among survivors.

**SJMCH Case Series**

A short case series of 10 patients with traumatic basal ganglia bleed managed in the Department of Neurosurgery, St. John's Medical College Hospital, was reviewed to examine imaging pattern, treatment strategy, and functional outcome. The cohort was composed predominantly of young to middle-aged male patients with traumatic basal ganglia hemorrhage involving the internal capsule, lenticular nucleus, thalamus, putamen, or external capsule. The largest hematoma diameter ranged from 1.02 cm to 2.9 cm, and most lesions were associated with additional traumatic findings such as gray-white matter junction contusions, subarachnoid hemorrhage, intraventricular hemorrhage, ventral or dorsolateral brainstem contusions, and facial or mandibular fractures.

| Case No. | Age (yrs) | Sex | Side of TBGH | Associated Hematoma | Epicenter of Hematoma | Largest Diameter of TBGH (cm) | Skull Fractures |
|---|---|---|---|---|---|---|---|
| 1 | 35 | M | Rt | Gray-white matter junction contusions | Lenticular nucleus & internal capsule | 1.09 | Zygomatic bone, maxillary sinus & mandibular bone |
| 2 | 54 | M | Lt | Temporal contusion | Internal capsule | 1.2 | No fractured bone |
| 3 | 48 | M | Lt | Bilateral SAH, gray-white matter junction contusion, bilateral intraventricular hemorrhage, & ventral brainstem contusion | Posterior thalamus & temporal lobe | 1.8 | No fractured bone |
| 4 | 28 | M | Rt | Bilateral intraventricular hemorrhage, ventral &posterolateral brainstem contusions, & gray-white matter contusions | External capsule | 2.9 | No fractured bone |
| 5 | 55 | M | Rt | Ventral brainstem contusion & gray-white matter contusions | Internal capsule & thalamus | 1.89 | Mandibular bone |
| 6 | 27 | M | Rt | Gray-white matter junction contusions & small frontal SAH | Internal capsule | 1.55 | Mandibular bone |
| 7 | 39 | M | Lt | Temporal contusion, bilateral SAH, & small intraventricular hemorrhage | Lenticular nucleus & external capsule | 1.88 | No fractured bone |

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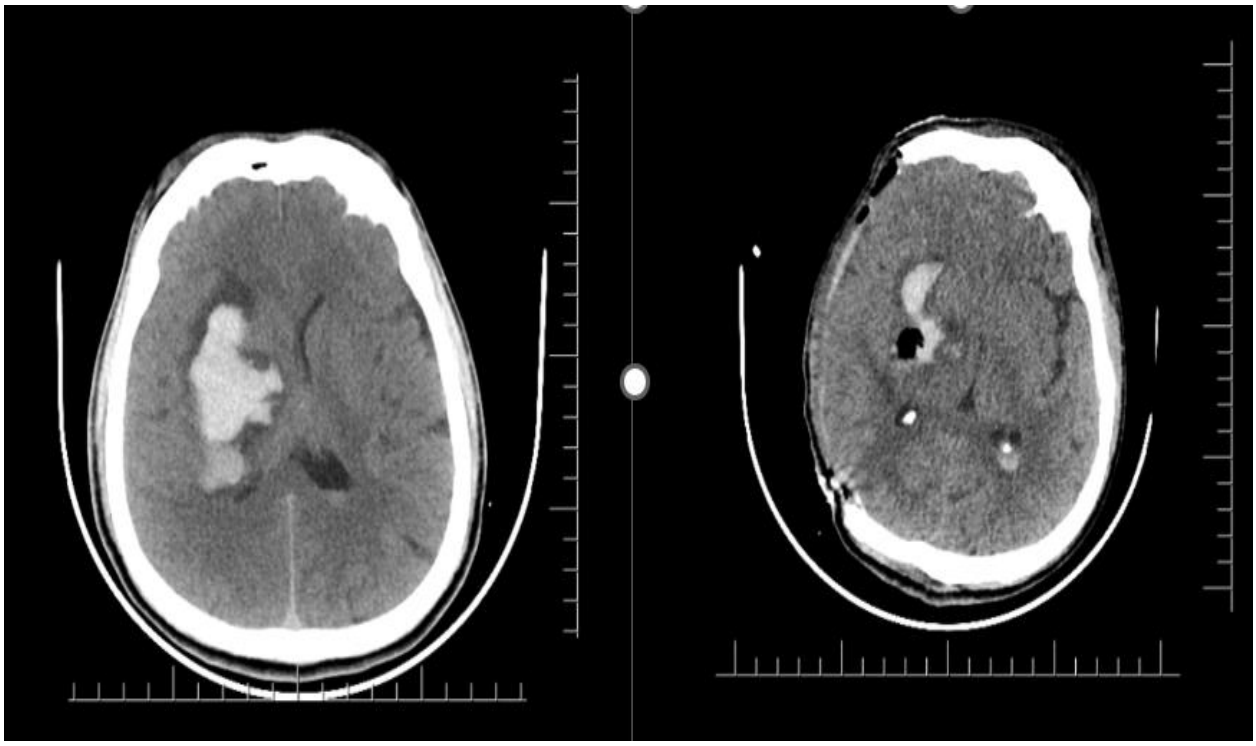
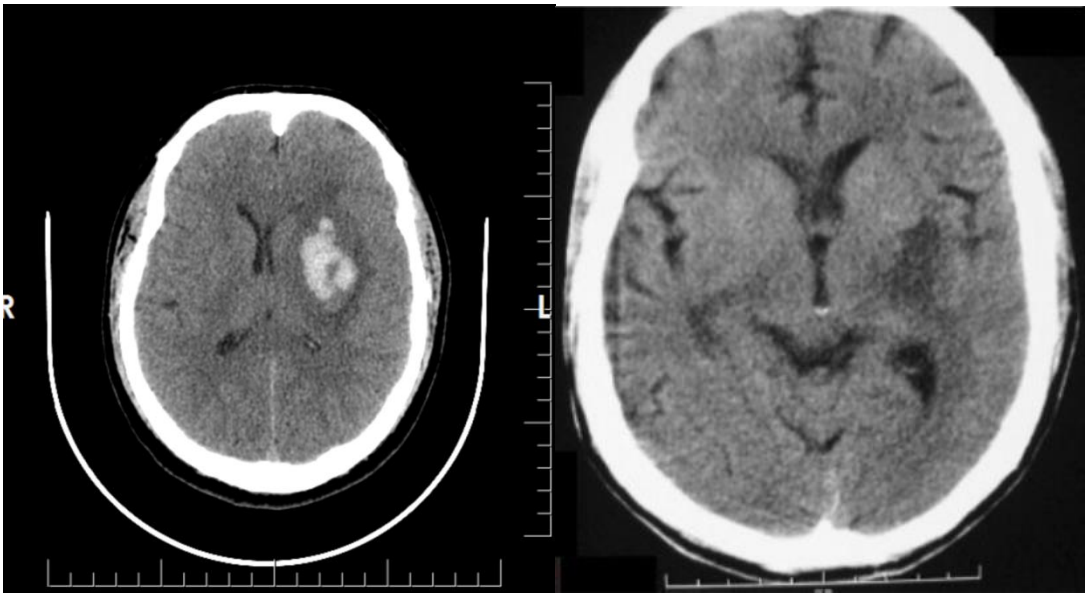
|---|---|---|---|---|---|---|---|
| 8 | 24 | M | Rt | Ventral brainstem contusion, gray-white matter contusions, & bilateral intraventricular hemorrhage | Thalamus & internal capsule | 2.34 | Maxillary sinus |
| 9 | 31 | M | Lt | Frontal contusion & gray-white matter junction contusions | Putamen & internal capsule | 1.26 | No fractured bone |
| 10 | 46 | M | Rt | Bilateral SAH, dorsolateral brainstem contusion, & temporal contusion | Posterior thalamus & external capsule | 2.09 | Zygomatic bone & mandibular bone |

The imaging profile in this series was strongly suggestive of a traumatic rather than spontaneous mechanism. The lesions were generally relatively small, often less than 2.5 cm in diameter, and were frequently accompanied by characteristic shear-related lesions, especially gray-white matter junction contusions and brainstem contusions. These associated findings are particularly useful in distinguishing traumatic basal ganglia bleed from hypertensive basal ganglia hemorrhage, which is moremmonly solitary, larger, and unaccompanied by impact-related lesions.

Patients in the series were managed according to standard traumatic brain injury principles, with treatment individualized on the basis of neurological status, radiological mass effect, and associated intracranial injuries. Follow-up at 3 months showed better prognosis based on RAS scoring, suggesting that meaningful functional recovery is possible in selected patients with traumatic basal ganglia bleed when identified early and managed appropriately. This observation supports the concept that traumatic basal ganglia hematoma should not automatically be assigned the same grim expectations associated with spontaneous deep ganglionic hemorrhage.



EXAMPLE: CASE No. 4- 28-year-old male with right external capsule TBGH (2.9 cm), extensive associated IVH, brainstem contusions, and gray-white matter junction contusions, no skull fracture underwent decompressivehemicraniectomy and improved neurologically from e2vtm5 to e4v4m6 post 3 months.




Case of left traumatic TBGH managed conservatively with complete resolution of the bleed and gross improvement radiologically

**Management**

Management is based on traumatic brain injury principles, not on spontaneous intracerebral hemorrhage algorithms alone. Initial care requires airway protection, oxygenation, hemodynamic stabilization, neurological grading with GCS, pupillary assessment, coagulation profile, and urgent noncontrast CT. Serial examination and repeat imaging are important because deterioration may result from edema, hematoma expansion, mass effect, or the evolution of associated lesions such as contusions or extra-axial hematomas.

Most patients are treated conservatively, especially when the hematoma is small, neurological status is stable or improving, and there is no refractory intracranial pressure or surgically significant mass effect. Conservative care typically includes ICU monitoring, head elevation, osmotherapy when indicated, seizure prophylaxis in selected patients, ventilatory support if needed, treatment of aspiration pneumonia or systemic trauma, and follow-up CT or CTA when necessary. In the 2025 illustrative series, 8 patients were managed conservatively while 2 patients needed surgery and patients with reactive pupils and better GCS had favorable recovery, while those with nonreactive pupils and severe injury died despite treatment.

Surgery is reserved for selected patients, usually those with hematoma volume greater than about 25 mL, progressive enlargement, refractory raised intracranial pressure, worsening neurological status, or another associated lesion that independently requires surgery. Published series report surgery in roughly 11% to 30% of cases, meaning the majority still remain nonoperative. Deep clot evacuation is not routine because the lesion lies in eloquent white matter and surgical corridors may worsen internal capsule injury, so decompressivecraniectomy or surgery for associated EDH/SDH may be more relevant than direct evacuation in many cases.

## DISCUSSION

Traumatic basal ganglia hemorrhage (TBGH) represents a distinctive clinicoradiological entity within the spectrum of traumatic brain injury, characterized by shear-induced vascular disruption in deep perforating arteries supplying the lenticulostriate territory. The SJMCH 10-patient case series provides compelling real-world validation of this pathophysiology, demonstrating classic features including right-sided predominance (60%), mean hematoma diameter of 1.8 cm (range 1.09-2.9 cm), universal gray-white matter junction contusions, frequent internal capsule involvement, and a 50% skull fracture rate predominantly affecting mandibular, zygomatic, and maxillary sinus bones. These imaging hallmarks with smaller multifocal lesions accompanied by brainstem contusions, subarachnoid hemorrhage (SAH), and intraventricular hemorrhage (IVH)helps to clearly distinguish TBGH from spontaneous hypertensive gangliocapsular hemorrhage, which typically presents as larger (>3 cm), solitary hematomas centered in the thalamus/internal capsule without traumatic stigmata.

The biomechanical forces underlying TBGH explain both its anatomical distribution and clinical behavior. Acceleration-deceleration injury, particularly during high-velocity road traffic accidents, generates tentorial notch shear that preferentially injures lenticulostriate branches of the middle cerebral artery and anterior choroidal artery perforators. This produces the characteristic "intermediate coup-contrecoup" pattern observed in

the series, where internal capsule (40%) and lenticular nucleus (30%) epicenters predominate alongside associated diffuse axonal injury (DAI) markers. The modest hematoma sizes (8/10 <2 cm) correlate with the favorable 3-month RAS outcomes reported, as lesions below 20-25 mL volume rarely produce refractory mass effect or necessitate surgical evacuation. Literature consistently identifies admission GCS <5, fixed pupils, and brainstem extension as primary mortality drivers (67-83% in severe cases), none of which appear operative in this cohort given the younger demographic (mean 38.7 years) and absence of coagulopathy.

The uniformly conservative approach in this series aligns optimally with evidence-based neurotrauma principles. Direct evacuation of gangliocapsular hematomas risks iatrogenic internal capsule disruption, rendering decompressivecraniectomy preferable for associated extra-axial collections or refractory intracranial hypertension. ICU protocols emphasizing serial CT surveillance, osmotherapy, seizure prophylaxis, and aggressive pulmonary toilet effectively mitigated secondary insults, facilitating RAS recovery. The 50% skull fracture association particularly mandibular fractures further supports trauma-specific etiology while highlighting the need for comprehensive maxillofacial evaluation in TBGH management. Rapid hematoma resolution on follow-up imaging, as anticipated with small lesions, underscores the contusion-like nature of most TBGH rather than expansive hypertensive clots.

TBGH demonstrates a distinctly more favorable trajectory than spontaneous hypertensive hemorrhage, challenging historical prognostic pessimism. While spontaneous gangliocapsular bleeds carry 40-50% mortality with persistent hemiparesis in survivors (due to chronic vasculopathy and larger volumes), TBGH benefits from younger patients, trauma-induced plasticity, and absence of vascular risk modifiers. The series' traumatic gray-white contusions (100%), brainstem involvement (70%), SAH/IVH (60%) not only confirms shear mechanism but predicts better functional restoration through cortical reorganization. Three-month RAS improvement reflects this differential: traumatic survivors leverage perilesional plasticity, whereas hypertensive cases face entrenched gliosis and recurrent bleeding risk.

Recognition of TBGH carries critical diagnostic and forensic implications. Pre-2020 literature often misattributed gangliocapsular hematomas to spontaneous rupture in trauma settings, but contemporary series establish trauma as a primary etiology when accompanied by shear stigmata. The SJMCH cohort's mandibular fracture association (30%) emerges as a novel discriminatory feature, potentially reflecting deceleration vectors targeting the skull base-tentorium interface. This has direct medicolegal relevance, as TBGH attribution to trauma precludes assumptions of preexisting hypertension or anticoagulation.

The SJMCH experience reframes TBGH as a manageable entity with superior prognosis to spontaneous counterparts when managed within modern neurotrauma frameworks. Conservative excellence, trauma-specific imaging, and biomechanical insight transform historical fatalism into rational optimism, positioning specialized centers to optimize outcomes in this rare but illustrative injury paradigm.

**Difference from hypertensive BG bleed:**

Hypertensive basal ganglia hemorrhage is usually a spontaneous deep intracerebral hemorrhage caused by chronic hypertensive small-vessel disease, most often involving perforators in the gangliocapsular or thalamic region. It tends to present as a larger, solitary hematoma centered in the basal ganglia, often with intraventricular extension, and is usually not accompanied by traumatic gray-white matter junction contusions, ventral brainstem contusions, skull fractures, or other impact-related lesions. Hypertension is strongly associated with poor outcome in spontaneous basal ganglia hemorrhage, and hematoma size, IVH, midline shift, and ICH score are key prognostic markers.

By contrast, traumatic basal ganglia hematoma is usually seen after high-energy injury in younger patients, often without hypertension, anticoagulant use, or chronic vascular risk factors. The lesion is more likely to be small or multifocal, associated with other traumatic bleeds, and accompanied by imaging signs of shear injury such as gray-white junction contusions and brainstem contusions. In the 2025 case series, the authors specifically noted that these CT features differ from spontaneous hemorrhage and may be particularly helpful when trying to distinguish the two. Another clue is distribution: spontaneous bleeds classically center in the thalamus/internal capsule region, whereas traumatic lesions more often involve the lenticular nucleus, external capsule, or mixed deep structures in a pattern linked to shear and rotational forces.

Management logic differs too. Spontaneous hypertensive bleed is approached through the framework of spontaneous intracerebral hemorrhage, blood pressure control, and selected surgical indications, whereas traumatic basal ganglia hematoma is treated within the broader TBI algorithm, where airway, ICP, associated lesions, pupillary status, and global injury burden dominate decision-making. So although both lesions occupy a similar anatomical region, they are biologically and clinically different diseases.

## CONCLUSION

Traumatic basal ganglia hematoma is a rare but important form of deep traumatic brain injury caused mainly by acceleration-deceleration and shear-related tearing of deep perforating vessels. It commonly coexists with other traumatic lesions, especially gray-white matter junction contusions, IVH, DAI, and ventral or dorsolateral brainstem contusions, and these associated injuries often determine prognosis more than the hematoma alone. Most cases are managed conservatively, while surgery is reserved for large hematomas, refractory ICP, worsening neurological status, or surgically significant associated lesions.

The key distinction from hypertensive basal ganglia hemorrhage is that traumatic lesions occur in a trauma-specific radiological and clinical context: younger patients, high-velocity injury, smaller or multifocal deep hemorrhages, and characteristic associated traumatic findings. Recognizing that difference matters, because traumatic basal ganglia hematoma should not simply be treated or prognosticated as a spontaneous gangliocapsular bleed.

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