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## Case report

# Traumatic brain injury presenting with bilateral basal ganglia hemorrhage



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## ABSTRACT

**Context:** Traumatic basal ganglia hemorrhage (TBGH) is a rare presentation of traumatic brain injury. Bilateral lesions are even rarer. Only twelve similar cases were previously published.

**Case report:** We report the case of a patient with bilateral TBGH. He was managed conservatively. Long-term follow-up disclosed a cognitive dysfunctions attributed to associated diffuse axonal injury. Acceleration and deceleration forces may have torn pallidum arterial branches determining hemorrhage.

**Conclusion:** Bilateral TBGH is an uncommon presentation of traumatic brain injury. Associated diffuse axonal injury worsens the outcome.

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## 1. Introduction

Traumatic brain injury (TBI) may present with a wide variety of lesion types, but basal ganglia hemorrhages are uncommon. They are founded in about 3% of patients referred to neurosurgeons [1], though its prevalence at autopsies series may be up to 10% [2].

Bilateral traumatic basal ganglia hemorrhage (TBGH) is extremely rare. We report the case of a patient with bilateral

TBGH. We also provide a literature review with all previously published similar cases and discuss the pathophysiologic theories and outcome possibilities.

## 2. Case report

A 57-year-old man was referred to our hospital with TBI. He was beat in the head during a strife and he was under alcohol effect. The patient was admitted with a GCS score of 8, with an

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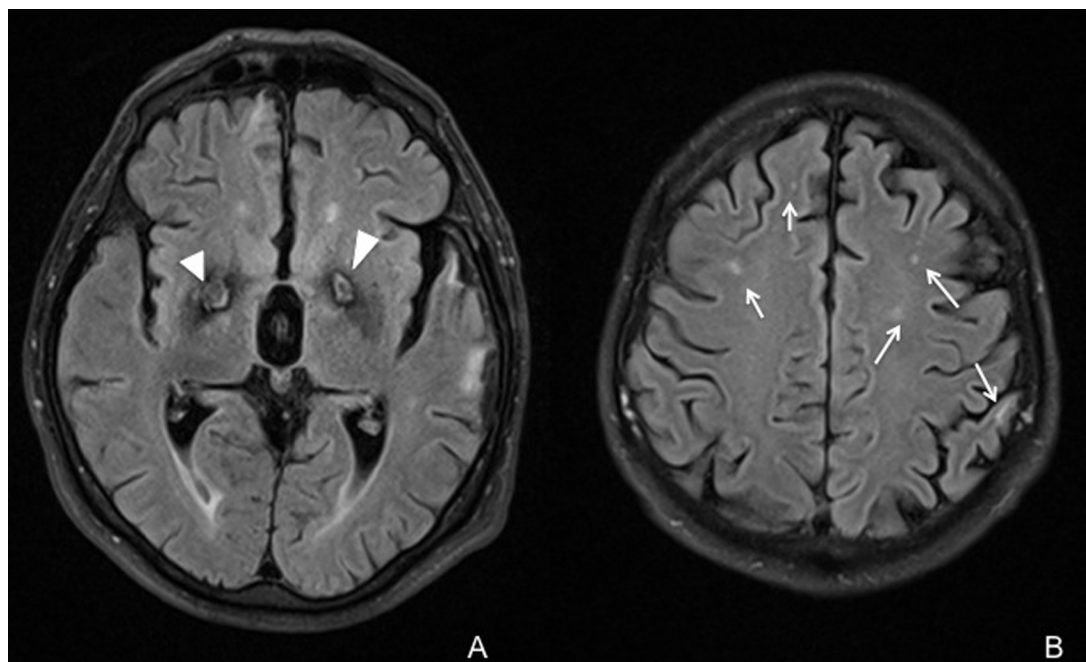
**Fig. 1** – CT image shows the basal ganglia hematomas (\*), traumatic subarachnoid hemorrhage (small arrow), temporal hemorrhagic contusions (arrow head) and intraventricular hemorrhage (large arrow). The patient also had a parietal fracture (not shown).

endotracheal tube and mechanic ventilation. General clinic parameters were normal. Pupils were miotic due to sedative drugs administered. Neither abnormal reflexes nor asymmetrical motor responses were observed. Computerized

tomography (CT) scan showed a hemorrhage within the lentiform nucleus bilaterally, without midline shift (Fig. 1). Other TBI features were present, such as hemorrhagic contusion in the left temporal lobe, subarachnoid hemorrhage in the right frontal convexity, a mild ventricular hemorrhage within the occipital horns, and a linear left parietal fracture. At the right, the hematoma had approximately 4.7 ml and, at the left, 1 ml. The patient was managed conservatively at intensive care unit (ICU). Serial CT scans showed no enlargement of the hemorrhages. By the 8th day post-admission the patient showed recovery of the consciousness and was taken off mechanical ventilation. Extubation was unremarkable. On the 12th day he was discharged from ICU. By this time he had no asymmetrical motor deficits. The patient was discharged after 17 days from admission with a cognitive impairment. A MRI performed one year after the trauma showed glial scars within the basal ganglia and diffuse foci of hypersignal on the brain parenchyma (Fig. 2). At 18-months follow up, an experienced neurosurgeon performed a full neurocognitive assessment. The patient showed memory deficits for fixation, consolidation and recovery of information, as well as temporal disorientation and dyscalculia.

### 3. Discussion

Spontaneous basal ganglia hemorrhage is a common presentation of cerebrovascular disease related to systemic hypertension. It tends to initiate in the region of thalamus and internal capsule and then grow. On the other hand TBGH is an uncommon feature among head injured patients and, in these cases, it tends to initiate in the zone of lenticular nucleus and external capsule [3].



**Fig. 2** – MRI performed one year after the trauma. (A) Glial scars within basal ganglia (arrowheads). (B) Diffuse hypersignal foci (arrows).

We performed a literature search for similar cases in Medline (via Pubmed), Cochrane, Embase and Lilacs databases. Using the terms: bilateral, traumatic and basal ganglia, we found 20 different articles. Articles were excluded when not related to blunt head trauma and when related to other traumatic lesions. Five articles were selected. Full publications references were reviewed to find other articles. A total of 10 articles were included, accounting for 12 case reports. Table 1 summarizes the cases.

In the series of 37 patients with unilateral TBGH from Boto et al., traffic accidents were responsible for the majority of their cases. This suggests that intracranial phenomena are responsible for TBGH [3]. From the 11 previously reported cases of bilateral TBGH, 7 were due to traffic accidents. Falls from height accounted for 3 cases. Aygün et al. reported a curious case of bilateral TBGH after rocket explosion [8]. Our case is singular as the trauma mechanism was a punch on the head. Though a fight context was present in a case reported by Jang et al., they blamed the trauma on the fall [5].

Pathophysiology mechanisms for the development of TBGH are uncertain, but some theories have been proposed. Mosberg and Lindenberg were the first to postulate that branches from the anterior choroidal artery may be torn during the trauma [14]. A biomechanical analysis proposes that an impact to the vertex, the forehead or the occipital area creates a force line that passes through the tentorium. This way, the brain is shifted to the tentorial notch in such a fashion that pallidum branches of the anterior choroidal artery are stretched and torn due to acceleration and deceleration forces [1–3]. Jang et al. objectively demonstrated that a strong impact to the occiput produced a fracture exactly on the midline. The direction of the impact was parallel to the tentorium and corresponded to the hemorrhagic site within the basal ganglia [5]. Fujioka et al. proposed that a traumatic injury to middle cerebral artery could also be responsible for hemorrhages

within the putamen [15]. Finally, Jain et al. hypothesized that for their patient some degree of asymptomatic derangement of the microvasculature might have predisposed to the hematoma formation [11].

Clinical presentation depends on the severity of the trauma. Most of the reviewed cases presented with coma. Headache was a common symptom among patients with mild TBI. On CT exams, TBGH may be accompanied by any other traumatic lesion [10]. Adams et al. considered 2 cm in the greatest diameter as the cutoff to define small or large lesions [2]. In addition small hemorrhagic lesions seem to be more associated to diffuse axonal injury (DAI) [11]. Actually the proposed pathophysiology mechanisms for TBGH have similarities to the mechanisms responsible for DAI. Acceleration and deceleration forces with rotational movement in the coronal plane may stretch and tear both vessels and axons.

Treatment must be based on the protocols for the management of TBI and intracranial hematomas. Some aspects have to be taken into account: neurological status, mass effect, intracranial pressure (ICP) and response to medical means of controlling ICP [9]. ICP monitoring may be guaranteed like in other severe TBI lesions [12]. Patient with a TBGH which volume is higher than 25 ml, with enlarging volume on serial CT scans or patients with refractory raised intracranial pressure despite optimized clinical measures have worse outcomes [11]. This points to the need for a more aggressive management of these patients – that is surgical hematoma drainage.

Prognosis of patients with TBGH is highly dependent on the presence of associated injuries, mainly DAI [5,6]. Poor prognosis is common among patients with advanced age (60 years or more), with abnormal pupillary reaction or impaired oculoccephalic and motor response, and with associated intraventricular or brainstem hemorrhage [16]. That means prognosis is poor in severely injured patients with very low

**Table 1 – Summary of bilateral TBGH reported in the literature.**

Author/year	Age/sex	Trauma mechanism	Clinical presentation	Management	Outcome
Yanaka et al., 1991 [4]	17yr, M	Traffic accident	Comatose, right hemiparesis	Stereotactic aspiration	NA
	75yr, M	Fall from height	Headache, nausea	Conservative	Good recovery
Jang et al., 2007 [5]	50yr, M	Fall during fight	Headache	Conservative	Good recovery
Kumar et al., 2009 [6]	35yr, F	Traffic accident	GCS 4, left hemiparesis	Conservative	Moderate disability
Kushal et al., 2011 [7]	42yr, M	Traffic accident	GCS 5	Conservative	Limbs paresis, mainly to the left
Aygün et al., 2012 [8]	35yr, M	Rocket explosion	Comatose	Conservative	Good recovery
Bhargava et al., 2012 [9]	25yr, M	Traffic accident	GCS 4, right hemiparesis	Conservative	Dysphasia, right hemiparesis
	50yr, M	Traffic accident	GCS 4	Conservative	NA
Jain et al., 2013 [10]	38yr, M	Fall from height	Asymptomatic	Conservative	Good recovery
Gupta et al., 2014 [11]	50yr, F	Traffic accident	GCS 7	Conservative	Left hemiparesis
Calderon-Miranda et al., 2014 [12]	28yr, M	Traffic accident	GCS 6	Conservative <sup>a</sup>	Death
Pandey et al., 2014 [13]	37yr, M	Traffic accident	GCS 6	Decompressive craniectomy	GCS 7
Present case	57yr, M	Fight	GCS 8	Conservative	Cognitive impairment

NA: outcome was not clearly stated.

<sup>a</sup> In this case it was performed a craniotomy to drain an epidural hematoma, but the TBGH were not managed surgically.

GCS score on presentation. Outcome is better when TBGH is an isolated finding [17]. In our review of bilateral TBGH there was one death but no publication reported severely disabled patients. Our patient developed a cognitive impairment that may be accounted to some degree of axonal injury, which is favored by the diffuse hypersignal foci on the brain parenchyma. Additionally, his mechanism of trauma may have been responsible for the occurrence of TBGH and DAI.

In conclusion bilateral TBGH are very rare. Through the extensive literature searches, only 12 cases of bilateral TBGH were found. In the presented case the mechanism of trauma was somehow different from the previously published similar cases. We highlighted the theories for the pathophysiology of TBGH and we reinforce the need for diagnosing associated DAI, which plays a key role on the prognosis of TBI with TBGH.

### Conflict of interest

None declared.

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### Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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