Article published online: 2022-09-22

CASE REPORT

# Bilateral large traumatic hemorrhage of the basal ganglion

Nityanand Pandey, Ashok Mahapatra[1], Pankaj Kumar Singh

Departments of Neurosurgery, All India Institute of Medical Sciences, New Delhi, India [1]All India Institute of Medical Sciences, Bhubhaneshwar, Orissa, India

## ABSTRACT

Traumatic bilateral basal ganglia bleed is extremely rare. It is defined as a hemorrhagic lesion located in the basal ganglia or neighboring structures such as the internal capsule and the thalamus. This report describes a 37-year-old man who had large bilateral basal ganglia hemorrhage (BGH) with subdural hematoma and traumatic subarachnoid hemorrhage. With regards to an etiology of bilateral hemorrhage of the basal ganglia, we could not disclose any possible cause except head injury in spite of full diagnostic work-up. Our final diagnosis was bilateral traumatic BGH (TBGH). The pathomechanism of such injuries is still not clear and it is proposed to be due to shear injury to the lenticulostriate and choroidal arteries. Rather than any features of the TBGH itself, duration of coma and/or associated temporal herniation predicted slower recovery and worse outcome. Bilateral TBGH is an extremely rare entity, compatible with a favorable recovery, if not associated with damage to other cortical and subcortical structures and occurring in isolation. TBGH can be considered as a marker of poor outcome rather than its cause. The BGHs seem to be hemorrhagic contusions resulting from a shearing injury, due to high velocity impact.

**Key words:** Basal ganglia, bilateral, hemorrhage, poor outcome, trauma

## Introduction

Traumatic basal ganglia haemorrhage (TBGH), is a rare entity and reported in only 3% of closed head injuries.[1] It is defined as a hemorrhagic lesion located in the basal ganglia or neighboring structures, such as the internal capsule and the thalamus.[2,3] It can be classified as "large," if it is more than 2 cm in diameter or as "small" if it measures <2 cm in diameter.[2] The prognosis in patients of head injury with concomitant basal ganglia bleed is dismal, especially in bilateral cases.[2,4,5] We briefly summarized our uncommon case of bilateral TBGH and discussed its possible mechanisms.

| Access this article online | |
|---|---|
| Quick Response Code: | Website: www.asianjns.org |
| | DOI: 10.4103/1793-5482.146644 |

**Address for correspondence:**
Dr. Pankaj Kumar Singh, Department of Neurosurgery,
Room No. 720, CN Center, All India Institute of Medical Sciences,
Ansari Nagar, New Delhi - 110 029, India.
E-mail: drpankajsingh11@gmail.com

## Case Report

A 37-year-old man received in the Emergency Department after being hit by a car while crossing the road. On examination, he was unconscious and needed to be intubated due to compromised airway. There was no available history of hypertension or diabetes mellitus. The blood pressure was normal on admission. The Glasgow coma scale (GCS) score on admission was 6. The noncontrast computerized tomography (CT) scan of the head showed left frontotemporal acute subdural hematoma with significant midline shift, left side traumatic subarachnoid hemorrhage (TSAH) and bilateral BGH with punctuate hemorrhages in the corpus callosum [Figure 1]. Magnetic resonance imaging (MRI and magnetic resonance angiography [MRA]) done after 9 days show late sub-acute intra parenchymal hematoma involving the bilateral capsuloganglioni region with surrounding edema without abnormal enhancement [Figure 2]. MRA did not reveal any vascular abnormalities [Figure 3]. The laboratory results were within normal ranges, including complete blood cell counts, bleeding time, prothrombin time, activated partial thromboplastin time, liver function tests, and blood glucose level. With regards to an etiology of bilateral hemorrhage of the basal ganglia, we could not find out any possible cause, except head injury despite full diagnostic work-up. Our final diagnosis was bilateral traumatic hemorrhage of the basal ganglia. Left frontotemporoparietal decompressive craniectomy was done

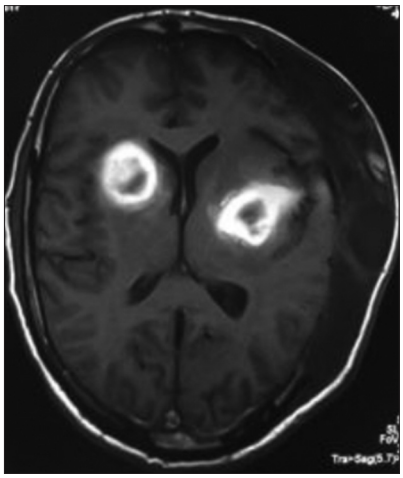

**Figure 1:** Noncontrast computerized tomography brain reveals bilateral basal ganglia hemorrhage with left frontotemporoparietal acute subdural hemorrhage and traumatic subarachnoid hemorrhage

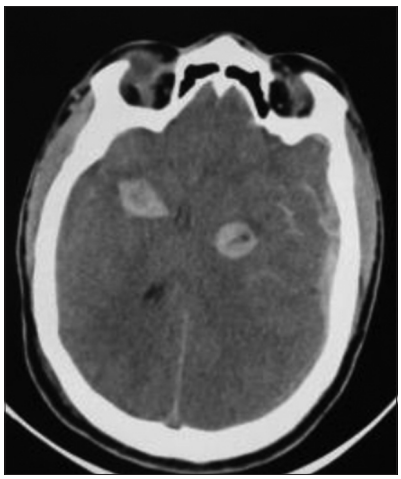

**Figure 2:** T1-weighted image 9 days after injury showing late sub-acute bilateral basal ganglia hematoma with postoperative changes

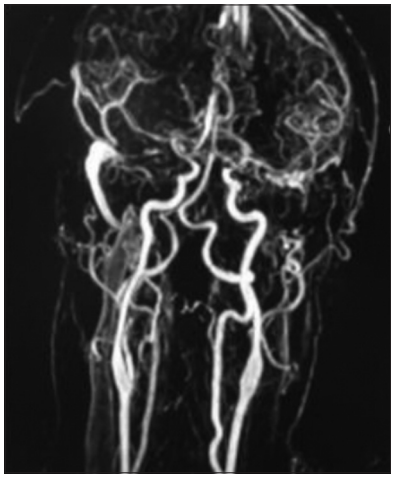

**Figure 3:** Magnetic resonance angiography showing no abnormal vascular lesion

in the emergency. The patient was on ventilatory support postoperatively and remained comatose. He was in the neuro Intensive Care Unit for 2 weeks and also required tracheostomy. At discharge, 4 weeks after injury his GCS was E4VtrM2.

## Discussion

Traumatic BGH is rare and its incidence is higher in autopsy series, which ranges from 10% to 12%.[1,2] The patients with TBGH have increased incidence of coagulation abnormalities, diffuse axonal injury, intraventricular hemorrhages, contusions, and extra axial hematomas. These patients are more prone to poorer outcome mainly due to the associated brain injuries.

There are only three case reports (four cases), two bilateral TBGH cases by Yanaka *et al.*,[6] and one case each by Jang and Jwa[7] and Kaushal and Kataria.[8] The mechanism of TBGH is still not clear. It is most likely caused by the shearing injury of a lenticulostriate or an anterior choroidal artery as a result of acceleration or deceleration forces brought about by a high velocity injury. When the strong impact is applied to the vertex, forehead, or occipital area and directed toward the tentorium, there would be a shift of the brain through the tentorial notch with stretching and tearing of vessels by shearing forces, resulting in hemorrhages in the basal ganglia region.[2,9]

As the lenticular region is prone to nontraumatic bleed, it is often difficult to rule out the exact cause in patients with trauma and basal ganglia bleed. However, in the present patient, there was no available medical history of hypertension or diabetes mellitus. His blood pressure had been within normal ranges during the hospital stay. We could not find any other cause of bilateral BGH except head injury as evidenced by the full diagnostic work-up studies that did not show other causes of intracerebral hemorrhages such as arteriovenous malformation, aneurysm, vasculitis and coagulopathy. Noncontrast brain CT revealed large hemorrhages in both the basal ganglia, acute subdural hemorrhage in the left frontotemporal region and TSAH in the left side. Hence, bilateral TBGH was made as a final diagnosis. The hemorrhages of our patient were classified as large hemorrhages because they measured more than 2 cm in diameter, as described in the literature.[2]

Contusions are bruises of the neural parenchyma as a result of head injury and their pathomechanism is poorly understood. Because deeply located hemorrhages such as the basal ganglia develop in the parenchyma between coup and contrecoup contusions, they have been considered as intermediary contusions and probably result from shear injury of the vessels in the basal ganglia region.

Various surgical options have been used for these patients including open surgery, CT guided stereotactic aspiration and ultrasonography guided aspiration. Except few reports, most of them experienced poor outcome in surgically treated

patients. Rather than any features of the TBGH itself, duration of coma and/or associated temporal herniation predicted slower recovery and worse outcome. The hemorrhage itself determines clinical signs related to particular subcortical structures involved and the side of the lesion. Overall cognitive impairment and speed and quality of recovery are more related to associate cerebral damage.[1]

## Conclusion

Traumatic BGH is rarely reported, compatible with a favorable recovery if not associated with damage to other cortical and subcortical structures and present in isolation. Bilateral TBGH can be considered as a marker of poor outcome. The BGHs seem to be hemorrhagic contusions as a result of a shearing injury due to high velocity impact.

## References


1. Katz DI, Alexander MP, Seliger GM, Bellas DN. Traumatic basal ganglia hemorrhage: Clinicopathologic features and outcome. Neurology 1989;39:897-904.
2. Adams JH, Doyle D, Graham DI, Lawrence AE, McLellan DR. Deep intracerebral (basal ganglia) haematomas in fatal non-missile head injury in man. J Neurol Neurosurg Psychiatry 1986;49:1039-43.
3. Boto GR, Lobato RD, Rivas JJ, Gomez PA, de la Lama A, Lagares A. Basal ganglia hematomas in severely head injured patients: Clinicoradiological analysis of 37 cases. J Neurosurg 2001;94:224-32.
4. Kim SH, Song SH, Youm JY, Kim Y. Clinical analysis of the traumatic basal ganglia hemorrhage. J Korean Neurosurg Soc 1996;25**:**1223-9.
5. Lee JP, Wang AD. Post-traumatic basal ganglia hemorrhage: Analysis of 52 patients with emphasis on the final outcome. J Trauma 1991;31:376-80.
6. Yanaka K, Egashira T, Maki Y, Takano S, Okazaki M, Matsumaru Y, *et al.* Bilateral traumatic hemorrhage in the basal ganglia: Report of two cases. No Shinkei Geka 1991;19:369-73.
7. Jang KJ, Jwa CS. Bilateral traumatic hemorrhage of the basal ganglia. J Korean Neurosurg Soc 2007;41:4, 272.
8. Kaushal R, Kataria R. Traumatic bilateral (mirror image) basal ganglia bleed. Indian J Neurotrauma (IJNT) 2011;8:109-10.
9. Graham D. Neuropathology of head injury. In: Harayan PK, Wilberger JE, Povlishock JT, editors. Neurotrauma. New York: McGraw-Hill; 1996. p. 43-59.


**How to cite this article:** Pandey N, Mahapatra A, Singh PK. Bilateral large traumatic hemorrhage of the basal ganglion. Asian J Neurosurg 2014;9:240.

**Source of Support:** Nil, **Conflict of Interest:** None declared.