Case Report
Bilateral basal ganglia bleed in traumatic brain injury: A case report

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Abstract
Intracerebral haemorrhage can be classified into traumatic and non-traumatic. Traumatic Basal Ganglia Haemorrhage (TBGH) has been reported in 2.4-3\% cases of all closed head injuries whereas the incidence is higher in postmortem studies (9.8\%), nevertheless, a bilateral TBGH is an extremely rare entity. According to our search through literature, only 12 case reports of bilateral TBGH have been published previously. A simple bilateral TBGH is rarely seen without any other lesions as it is usually associated with skull fractures, haemorrhages or brainstem injuries, making its incidence more during autopsies.

We present a 30-year old male patient who had a traumatic brain injury (TBI) secondary to Road Traffic Accident (RTA) with GCS of 12/15, having no other co-morbid. CT-scan revealed bilateral basal ganglia bleed and the patient improved on conservative management only.
Keywords: Traumatic Basal Ganglia Haemorrhage (TBGH); Bilateral; Trauma Brain Injury (TBI); Road Traffic Accident (RTA).

Introduction
Traumatic Basal Ganglia Hemorrhage (TBGH) is defined as a hemorrhagic lesion located in the basal ganglia or neighboring structures such as internal capsule or thalamus. Although the true mechanism of the hemorrhage is yet unknown, however the acceleration or deceleration forces during the trauma cause a shear strain on the lenticulostriate or anterior choroid vessels. If the hemorrhagic lesion is greater than 2cm in diameter, it is categorized as a large hemorrhage whereas if the hemorrhagic lesion measures less than 2cm it is categorized as a small hemorrhage. We present a case of bilateral TBGH caused by traumatic brain injury (TBI) following a road traffic accident (RTA).

Case Report
A 30-year old non-helmeted male motorcyclist presented in Shaheed Mohtarma Benazir Bhutto Trauma Center ER 30 minutes after his bike lost its balance due to a speed breaker, resulted in a fall. He presented on 10th December 2018 with altered level of consciousness and irritability secondary to RTA. There was no history of significant illness or substance abuse. Family history was negative for any bleeding disorders, hypertension (HTN) or diabetes mellitus (DM), and he was on no medications prior to RTA. On arrival, his vitals were 90/mins HR, B/P was 230/118, Glasgow coma scale (GCS) was 12/15 with a localized pain in the right hand, eye opening on command and confused speech (E3, M5 and V4), pupils were BERL with 3mm diameter bilaterally; while RBS was 132 mg/dl. He also had a lacerated wound on his left forearm. A minor abrasion on the face along with a 4cm laceration on his scalp at vertex was also noted. Clotting profile was within normal range. An ECG of the patient revealed sinus tachycardia. CT scan
of the brain showed bilateral basal ganglia bleed with 42 ml on the right side and 2.5 ml on the left side.

The patient was provided initial resuscitation and was kept under observation. Stat dose of injection labetalol was given on arrival. His B/P remained within normal limits during the rest of his hospital stay. Patient was orally allowed on liquids and then soft diet from third day of hospital admission as his GCS improved to 15. Serial CT scan imaging revealed no increase in the size of the hematoma. The patient was discharged on the 5th day of admission with powers of 5/5 in bilateral upper and lower limbs and no focal neurological deficit. He was advised a follow-up in 2 weeks but unfortunately the patient did not return for a follow-up, which is the limitation of our study.

Discussion

Basal Ganglia performs many important functions, having multiple subcortical nuclei that is supplied by perforating branches of ipsilateral middle cerebral artery (MCA). Simultaneous Bilateral Basal Ganglia Hemorrhage (SBBGH) is a rare entity with less than 30 cases reported globally (4) and mostly occurs due to hypertension. However, even lesser number of cases has been reported globally of bilateral TBGH. Kumar et al found one case of bilateral basal ganglia bleed out of 10 TBGH cases. (5) Originally TBGH was considered to be either a small, multiple or bilateral hemorrhage formed in the lentiform nucleus with external capsule, while spontaneous hemorrhages were considered large solitary masses in thalamic or internal capsule region. (6)

According to review of the literature, the culprit in TBGH was a ruptured anterior choroidal artery forming a massive hematoma in the pallidum found in an autopsy performed by Mosbergand Lindenberg of a patient with a fatal head injury (7); and as described by Maki et al a ruptured middle cerebral artery (8), but these cases contained data only of unilateral basal ganglia bleed. All the cases mentioned were confirmed to be traumatic.
There are 2 hypotheses regarding the mechanism of intracerebral hemorrhage of the basal ganglia after brain trauma: Spontaneous hemorrhage and traumatic hemorrhage. (9) According to the spontaneous hemorrhage hypothesis, it occurs when the blood pressure elevates spontaneously due to emotional or physical stress and ruptures the lenticulostriate artery forming a hematoma. Congenital malformation, long-term hypertension or diabetes is the underlying cause according to this hypothesis. Traumatic hemorrhage hypothesis blames a shearing force that causes the tear in the pallidum branch of anterior choroidal artery as shown in an autopsy case by Lindenberg. (7, 9) Acceleration and deceleration forces can tear the pallidum arterial branches resulting in hemorrhage. Damage to the above-mentioned vessels during a trauma, can be due to shearing forces, as the brain shifts due to impact through the tentorial notch because of deformity of the skull. (10) Intermediary contusions may result in the basal ganglia due to shear injury of the vessels.

Our patient’s mechanism of injury - with no suggestion to any other predisposing factor for bleeding - suggests it to be a bilateral TBGH. Also our patient has no history of hypertension or diabetes. The elevated B/P on arrival was brought under control with one stat dose of labetalol and no subsequent elevated BP readings suggested the cause to be a sequel of the trauma and due to a spontaneous bleed. Our patient had a hemorrhage larger than 2cm in diameter bilaterally and was diagnosed with a large bilateral traumatic basal ganglia hemorrhage.

Protocol for the treatment is the same as any unilateral spontaneous intracranial hematoma. Neurological status, presence of mass effect and response to other methods of controlling intracranial pressure are given special important when prescribing the treatment programmed. (11) Treatment plans can be conservative, open surgery, CT guided stereotactic or ultrasound guided aspiration. (10) Kumar et al managed bilateral TGBH conservatively (5) while Jang et al had a patient with a GCS of 15 who was also managed conservatively for bilateral TGBH. (12)
The aftermath of TBGH outcomes are varied. Bilateral TBGH patients studied by Kumar et al\(^5\) and Jang et al\(^12\) had no mortality with a Glasgow Outcome Score of 4 or 5. Poor prognosis has been observed due to various causes such as large size, coagulation disorders, DAI, other bleeders such as intraventricular or brain stem haemorrhage and/or contusions, old age as in above 60 years, abnormal pupillary response, abnormal motor response to pain, severe head injury, extraaxonal hemotoma etc. \(^3\)

Poor outcome is also seen in surgical treatment. Duration of coma and associated herniation predicts slow recovery and worse outcome. Thus, cognitive impairment, speed and quality depend on the cerebral damage. \(^13\) Poor prognosis is seen more in patients with TBGH than any other type of post-traumatic intracranial hemorrhages, especially with bilateral TBGH. \(^3\)

**Conclusion**

Bilateral TBGH is a rare entity only seen in a handful of case reports. Although the prognosis is quiet variable and depends on multiple factors, good prognosis may be seen if other cortical and subcortical structures are spared and the lesion itself is isolated. It can be managed conservatively since the removal involves approached through the internal capsule which may cause further damage or injury. We believe that bilateral basal ganglia haemorrhage in this patient was due to head trauma and patient showed good neurological outcome. So despite having high volume on one side, patients with bilateral TBGH may be considered for conservative management if there are no signs of mass effects as observed in this patient.

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Figure 1: Non-contrast computerised tomographic scan of brain (Axial view) reveals bilateral basal ganglia haemorrhage.
Figure 2: Coronal view of non-contrast computerised tomographic scan of brain on arrival reveals bilateral basal ganglia haemorrhage.