An Unusual Combination of Posttraumatic Ipsilateral Basal Ganglia Infarction with Contralateral Hemorrhage: A Rare Case Report and Review of Literature

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Abstract

Posttraumatic ipsilateral basal ganglia infarction with contralateral hemorrhage (IBGICH) is an extremely rare neuropathologic entity. Hematomas or infarction of the basal ganglia in head injury have long been recognized but their mechanism has not been revealed clearly. It is presumed to be secondary to rupture and/or vasospasm, followed by thrombosis of the lenticulostriate and/or anterior choroidal artery. This happens by shearing stress as a result of acceleration or deceleration torques. Outcome of traumatic basal ganglia hemorrhage (TBGH) appears favorable unless it is large, associated with coagulation disorders or other intracranial injuries such as diffuse axonal injury, cerebral contusion etc. We present a rare case of a 32-year-old man with traumatic IBGICH (mirror-image). Patient was managed conservatively and discharged with residual paresis and aphasia. Mechanism of trauma, clinical features, management, and most importantly the medicolegal aspect of the TBGH is discussed.

Keywords
- traumatic
- basal ganglia
- infarction
- hemorrhage
- medico-legal

Introduction

Ipsilateral basal ganglia infarction with contralateral hemorrhage (IBGICH) is an extremely unusual outcome of closed head injury. Traumatic basal ganglia hematoma (TBGH) is a rare neuropathologic entity, defined as a hemorrhagic lesion located in the basal ganglia or neighboring structures such as the internal capsule and thalamus.[1,2] Its incidence is approximately 3% after a closed head injury.[3] However, autopsy series indicate a higher incidence ranging between 10 to 12%.[1,3] Traumatic basal ganglia infarction (TBGI) is a still rarer neuropathologic entity and mostly restricted to the pediatric population.[4]

Though the mechanism is poorly understood, most of the literature point toward the shearing strain in lenticulostriate or anterior choroidal vessels, caused by the acceleration/deceleration forces. They result in micro tears and/or vasospasm in the vessels, resulting in hemorrhage or infarct, respectively.[1,4] Both TBGH and TBGI can occur as an isolated lesion or associated with other intracranial injuries such as diffuse axonal injury, cerebral contusion, and subdural or epidural hematoma.[2,5,6] Isolated TBGI and TBGH have a better prognosis.[3] The importance of the medicolegal aspect of TBGH/TBGI/IBGICH cannot be undermined, where often it is a daunting task to prove/disprove "trauma" as the cause of the catastrophe.

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Case Description

A 32-year-old man was received in the emergency department, 12 hours after being hit by a truck. The patient was under the influence of alcohol while driving his bike. He had external injuries more prominent on the right side of body and head and neck.

On examination, he was unconscious and needed to be intubated, owing to compromised airway. There was no history of aural/nasal bleed, seizure, or cerebrospinal fluid leak. The patient was not a known case of hypertension or diabetes mellitus. The blood pressure was normal on admission. The Glasgow coma scale (GCS) score on admission was 7/15 (E2V2M3). Pupils were 2 mm bilaterally, sluggishly reacting to light. There was paresis in all four limbs.

The noncontrast computerized tomography (CT) scan of the brain revealed a hypodense lesion at the right lentiform nucleus suggesting ipsilateral basal ganglia infarction with contralateral gangliocapsular hyperdense lesion suggesting left basal ganglia hematoma of volume around 2 mL (►**Fig. 1A)**. CT angiography done after 10 days did not reveal any abnormality (►**Fig. 1B)**. Magnetic resonance imaging (MRI) with MR angiography was not possible due to an implant in situ in his right femur because of a fracture 3 months back. The laboratory work-up were within normal ranges, including complete blood cell counts, bleeding time, prothrombin time, activated partial thromboplastin time, liver function tests, and blood glucose level. Protein C (77%), protein S (69%), and D-dimer (221 ng/mL) levels were normal. Antinuclear antibody (0.22), and anti-Ds DNA (0.01) were negative.

Additional risk factors for vascular infarction including anticardiolipin IgM and IgG, antiphospholipid IgM and IgG were also found to be negative. His T3, T4, and thyroid-stimulating hormone levels were also normal.

Blood pressure of the patient had been within normal range throughout a 4-week hospitalization period. In search for an etiology of bilateral hemorrhage with infarction of the basal ganglia, we could not find anything, except the head trauma, despite full diagnostic work-up. Our final diagnosis was traumatic ipsilateral basal ganglia infarction with contralateral hemorrhage.

Patient was managed conservatively in the intensive care unit (ICU) with ventilator support. He remained comatose in the neuro ICU for 3 weeks and required nasogastric feeding and tracheostomy. Noncontrast CT done after 35 days showed almost complete resolution of the hematoma (►**Fig. 1C**). Supportive care was continued and the patient was weaned off the ventilator.

He developed spontaneous eye opening and was able to maintain eye contact with the caregivers. On follow-up visit at 3 months, the patient had regained consciousness and was obeying verbal commands. He was left with dysphasia and spastic quadriparesis with power of grade 3/5 in right limbs and 4+ /5 in left limbs.

Discussion

TBGH is rare and its incidence is higher in autopsy series, which ranges from 10 to 12%,[1,3] which suggests that most of these patients succumb to their illness, even before they are diagnosed. The risk factors for TBGH are coagulation abnormality, diffuse axonal injury, intraventricular hemorrhage, contusion, and extra-axial hematomas. These patients are quite vulnerable to the worst outcome, mainly due to the associated brain injuries.[3]

The mechanism of TBGH is still not clear. Regarding mechanism of TBGH, Mosberg and Lindenberg demonstrated traumatic tear of a pallidal branch of anterior choroidal artery as the source of pallidal hematoma in an autopsy case.[7] 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, 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

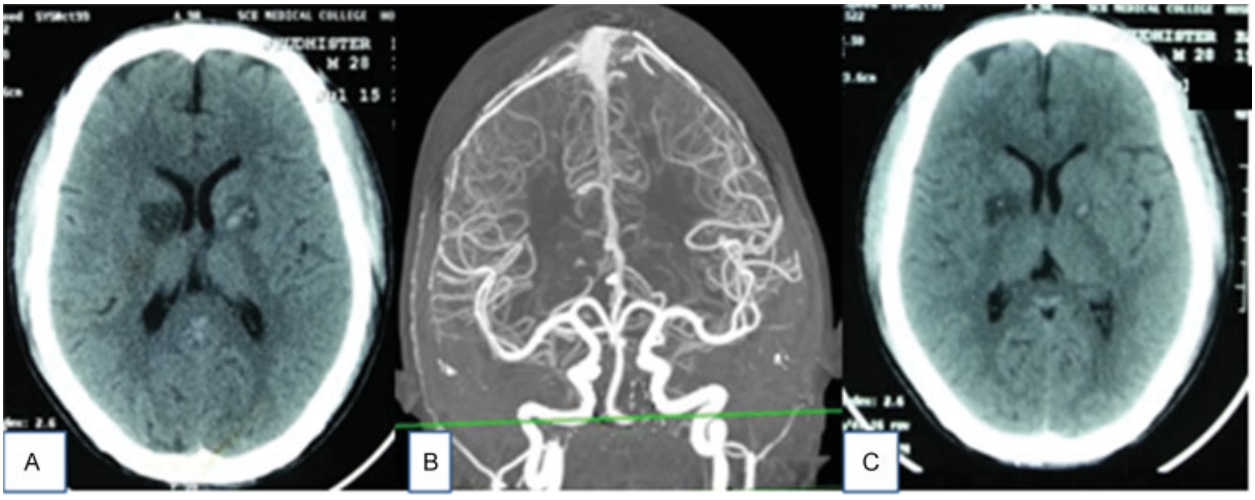

Fig. 1 (**A**) Noncontrast computed tomography scan of the brain showing right basal ganglia infarct with left basal ganglia hematoma. (**B**) Computed tomography angiography showing normal vascular anatomy with no abnormality. (**C**) Follow-up noncontrast computed tomography scan showing resolving left basal ganglia contusion with right basal ganglia infarct.

hemorrhages in the basal ganglia region.[1,8] Also this shearing force can cause vasospasm or intimal trauma and subsequent thrombosis in lenticulostriate branch or anterior choroidal artery, causing infarction in basal ganglia.[9,10]

In addition, basal ganglia are a region predisposed to hypertensive hemorrhage, when head injury and hypertension are simultaneously present. Despite thorough evaluations for etiology, it may still be difficult to reach a definitive conclusion. For this reason, differential diagnosis of traumatic or spontaneous basal ganglia hematomas can be a crucial medicolegal issue.[11] Therefore, a very careful evaluation of past medical records as well as physical examination and different imaging modalities, are mandatory for correct diagnosis and proper management.

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 basal ganglia hemorrhage except the head injury, as evidenced by the full diagnostic work-up which ruled out causes of intracerebral hemorrhage, such as arteriovenous malformation, aneurysm, vasculitis, or coagulopathy.

TBGH are dynamic lesions and tend to expand in volume during acute posttraumatic period, hence there is importance of serial CT scanning.[8] Treatment options for TBGH include conservative, open surgery, CT-guided stereotactic or ultrasound-guided aspiration.[2,12,13] The outcome of most surgically treated patients is poor.[5] Boto et al[2] recommend surgical evacuation for all lesions having a volume greater than 25 mL. In the series by Kumar et al[14] all the patients were managed conservatively (average volume 13.2 mL). In the case of bilateral TBGH by Jang et al[11] the patient had a GCS of 15 and hence was conservatively managed.

The absolute indications of surgery are: (1) volume of lesion $>$ 25 mL, (2) increasing size of hematoma, and (3) deterioration of neurological status of the patient. In addition, medical and supportive treatment should be instituted which includesintracranial pressure monitoring, elective ventilation, and close monitoring in ICU.[3]

The good prognostic factors in TBGH are (1) younger age ($<$60 years), (2) small lesions ($<$2 cm), (3) GCS $>$ 9 at the time of admission, and (4) isolated lesion.[15] 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 associated cerebral hemorrhage.[3,6]

It is amply mystifying how a particular injury can cause two lesions on the opposite ends of a vascular ailment spectrum. The answer is not straight forward, as this is a very unusual occurrence.

The clues may lie with the hypothesis that the vasospasm/tear in the vessel wall ipsilaterally causes the TBGI; it may well push the extra blood volume into the contralateral vessel lumen. This results in transiently raised pressure in the contralateral vessel wall; which when exceeds its compliance, may give way to result in TBGH. At the end we get IBGICH, in the same patient after one particular traumatic episode.

There are only few case reports on TBGH/TBGI—two bilateral TBGH cases (Yanaka et al[10]) and one case by Jang et al.[11] As per our extensive database review of PubMed, this might be the first reported case of IBGICH in published literature.

Conclusion

Traumatic IBGICH is a very rare entity. As per our extensive database review of PubMed, this might be the first reported case in published literature. Proper medical management and timely intervention may prolong the life expectancy of these patients. Finally, the medicolegal aspects of IBGICH make these cases interesting as well as conflicting, which make the intricacies of diagnosis and management of this rare entity extremely important.

Conflict of Interests
None.

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