

“Right Brain Has Nothing Left; Left Brain Has Nothing Right”: An Interesting Complication of Posttraumatic Corpus Callosal Bleed Presenting with Split-Brain Syndrome — A Case Report and Review of Literature

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Abstract

Gross and microscopic lesions of corpus callosum and neighboring structures are common in severe closed head injury. Left limb apraxia after damage to corpus callosum has been reported in patients whose neocortical commissures were sectioned for relief of severe epilepsy and in patients affected by naturally occurring lesions either of vascular or neoplastic origin. Only few reports are found in literature to have split-brain syndrome or disconnection syndrome of traumatic origin. Here we present a patient with left-sided apraxia, agraphia, and tactile anomia with right-sided constructional disturbances after a severe head injury with corpus callosal bleed.

Keywords

- ▶ severe closed head injury
- ▶ corpus callosum
- ▶ split-brain syndrome
- ▶ left-sided apraxia
- ▶ neuropsychological tests

Left-sided apraxia was profound on verbal command, visual presentation of an object, and imitation, but not during tactile presentation or the actual use of the objects. These data are consistent with left hemisphere dominance for praxis, which is almost absolute when the retrieval of the appropriate gesture requires a semantic analysis of the stimulus.

Introduction

The corpus callosum (CC) contains nerve fibers that connect both hemispheres and make an important contribution to the interhemispheric transfer of cognitive, somatosensory, motor, executive, and visual information. Injury to CC fibers can result in impaired transfer of interhemispheric information and hence cause heterogeneous neurologic manifestations including apraxia, agraphia, alexia, tactile anomia, and visual anomia. A combination of these neurologic manifestations after injury to CC fibers is defined as callosal disconnection syndrome or split-brain syndrome (SBS).

SBS is rarely described in a traumatic scenario and is limited to few case reports only. Though a large percentage of CC hematoma is found in autopsy series of severe closed head

injury (CHI), there is sparse reporting of SBS. It may be attributed to the fact that when CHI is severe enough to damage the CC, the overall neurologic impairment renders the patients unfit for testing of signs of callosal dysfunction. In less severe CHI, the standard neuropsychological examination usually skips specific tests meant for callosal dysfunction.¹

We present here a case of traumatic CC injury with features of SBS and also discuss the pathophysiology and specific diagnostic callosal tests for SBS.

Case Description

A 75-year-old, right-handed, alcoholic male with no history of previous illness presented with CHI after a road traffic accident. The patient was received in our intensive care unit with a

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background history of posttraumatic loss of consciousness, Glasgow Coma Scale (GCS) score of E1V2M2, that is, 5/15, and bilateral sluggishly reacting pupils.

All hematological parameters were within the normal range. Visceral organ injuries with spinal cord injury were excluded. Immediate noncontrast computed tomography scan revealed corpus callosal hematoma with intraventricular hemorrhage (IVH; ►Fig. 1A, B). Patient was intubated and kept in mechanical ventilation for 2 weeks. By the third week, spontaneous eye opening with a GCS score of E4V3M4, that is, 11/15, was observed, and by the fifth week, he was completely conscious with a GCS score of E4V5M6, that is, 15/15.

During the fifth week of hospital stay, the patient was observed performing simple tasks (e.g., elevating hands above his head) flawlessly using the right limb; performance using the left limb was both slow and hesitant. He showed spontaneous and correct utilization of left limb in daily activities, but execution of gestures on imitation and command was impaired.

Magnetic resonance imaging (MRI) of brain at the end of the second week revealed subacute hematoma in the CC with perifocal edema and resolved IVH (►Fig. 1C–G). Sensory function with visual field examination was normal. There was no clinical visual extinction on double simultaneous field stimulation. Visual, somesthetic, and auditory evoked potentials were normal.

Although the patient improved, the hematoma in CC with hesitant use of left limb tempted us to search for SBS, for which the patient underwent neuropsychological evaluation for callosal dysfunction.

Neuropsychological Tests

Imitation of unimanual finger posture (e.g., forefinger and middle finger extended to represent the letter V): Patient performed it nicely with his right hand but could not do it properly with his left hand.

- Imitation of bimanual finger posture (e.g., hook both index fingers): Patient made a hook with his right index finger and left middle finger.
- Imitation of symbolic gestures (e.g., say goodbye): Here performance was correct with both limbs.
- Movement imitation test (De Renzi et al¹⁶): This test is used to assess ideomotor apraxia and consists of 24 gestures, which the examiner performs in front of the patient. Immediate reproduction was required. Patient scoring less than 62/72 was considered apraxic. Our patient was within normal range with both hands (right: 69/72; left: 67/72).
- Use of object test: The patient was requested to demonstrate 12 common objects (key, comb, hammer, spoon, knife, etc.) with both hands under following conditions:
 - Pantomime to verbal command.
 - Pantomime to visual presentation.
 - Pantomime to imitation.
 - Use of object in tactile presentation.
 - Actual use of objects.

►Table 1 illustrates the patient's response.

Constructional praxis testing (e.g., figure copying in both

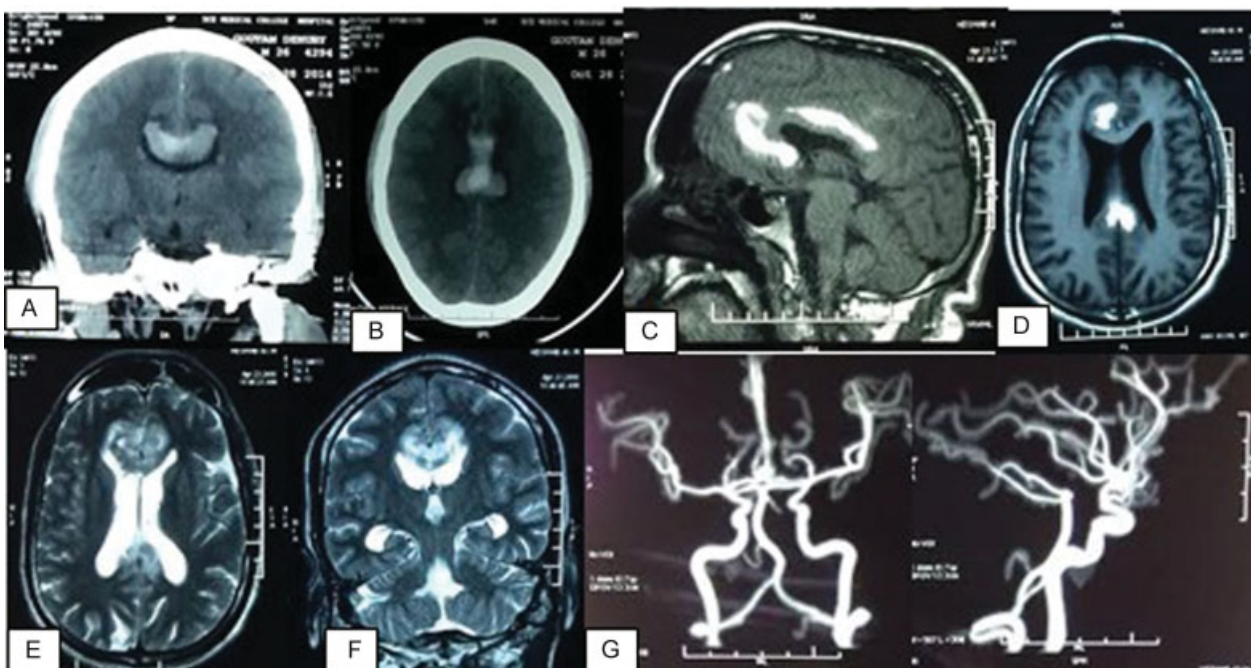


Fig. 1 (A) Coronal view of noncontrast computed tomography (CT) scan showing corpus callosal bleed with intra ventricular hematoma. (B) Axial view of CT scan showing corpus callosal bleed with intra ventricular hematoma. (C) T1 sagittal view magnetic resonance imaging (MRI) showing hematoma in the rostrum, genu, and body of corpus callosum. (D) T1 axial MRI showing corpus callosal hematoma and (E, F) showing corpus callosal hematoma in T2 MRI sequence. (G) MR angiography showing no abnormality.

Table 1 Upper extremity responses to various command stimuli

Stimulus	Verbal command		Picture of objects		Imitation		Use of real object	
	Right	Left	Right	Left	Right	Left	Right	Left
Mobile phone	Correct	No action	Correct	Rubs forehead	Correct	Scratches forehead	Correct	Correct
Scissors	Correct	Looks here and there	Correct	Open and close fist	Correct	Smiles	Correct	Correct
Ring bell	Correct	Hand over head	Correct	Wipes the table	Correct	Smiles	Correct	Correct
Cut with saw	Correct	Makes fist and stops	Correct	Makes fist	Correct	Looks here and there	Correct	Correct
Glass of water	Correct	Brushes table with palm	Correct	Keeps fist closed	Correct	Brushes the table with palm	Correct	Correct
Pound with a hammer	Correct	Makes fist and stops	Correct	Looks at the left hand	Correct	Correct	Correct	Correct
Comb hair	Correct	Looks at the left hand	Correct	Smiles only	Correct	Correct	Correct	Correct

hands): In our patient, reproduction of figure with his left hand was tremulous and hesitant but correctly reproduced. On the contrary, he was impaired in copying complex figures with his right hand (e.g., cube, Greek fret; ► **Fig. 2**).

- Stereognosis and tactile naming: Patient was requested to name 10 common objects placed in his left or right hand (while blindfolded). He made no errors with his right hand while failed to identify four objects in his left hand. When requested to select from a group of same objects he had manipulated, his performance was flawless with both hands.
- Somesthetic transfer: The patient, while blindfolded, was requested to reproduce 10 postures of left hand with his right hand. He was also asked to manipulate a three-dimensional geometric shape with his right hand. He performed flawlessly with both hands.
- Language: Patient showed no signs of aphasia or reading difficulty.
- Dictation task test: His right hand was far superior to the left in writing. He could slowly and shakily write letters and words to dictation and copy letters or words. With the left hand however he was totally unable to make more than a single, usually undecipherable letter to dictation, which didn't improve in copying.

- Intelligence: Patient's score in Raven Progressive Matrices was 35/48.
- Memory: The Wechsler Memory Scale score in our patient was found to be 97.

Discussion

Many of the signs and symptoms present with our patient can be attributed to a partial hemispheric disconnection syndrome or SBS caused by traumatic CC lesion. Our neuroradiological findings are consistent with this interpretation. MRI scan revealed involvement of rostrum, genu, middle part of body, and isthmus. Splenium seemed unaffected in the MRI scan.

Lindenberg et al reported the occurrence of callosal lesion in 16% of cases with traumatic brain injury (TBI).²

The left limb hypotonia and hypokinesia in our patient may be attributed to the involvement of supplementary motor area.³ They have also been reported in the early stage of surgical callosotomy.^{4,5}

Apraxia was mainly evident in the distal portion of limb affecting finger gestures more than hand and arm gestures, which is in agreement with reports.^{5,6} Left limb agrapahia is a closely associated finding with left limb apraxia of callosal origin, which was seen in our patient too.^{4,7,8}

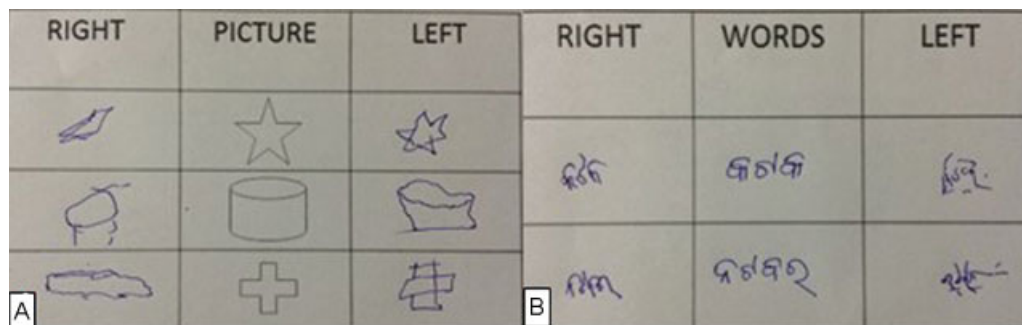


Fig. 2 (A) Patient's performance in copying figures with both hands. (B) Patient's performance in a dictation task with both hands.

The posterior callosal lesion that interrupts the transfer of information from right parieto-occipital region (known to be the seat of many perceptual-spatial abilities) to the left hemisphere results in a right limb constructional apraxia,⁹⁻¹¹ which was also found in our patient.

Our patient had impaired pantomime of verbal command, visual presentation, and imitation but not on tactile or actual presentation. Pantomiming to verbal command and visual presentation requires a semantic analysis of the stimulus to recall the visual configuration of the gesture.¹² The left hemisphere, which is strongly dominant for this kind of analysis, could not transmit information to the right hemisphere due to callosal injury resulting in poor left limb performance.^{12,13} On the contrary, pantomiming to actual and tactual presentation does not necessarily require a semantic elaboration.

Sometimes complete or partial sectioning of CC fails to produce expected symptoms. Geschwind¹¹ and Sperry et al⁴ have reviewed the factors that cause inconsistencies in the development of clinical features of callosal damage.

The absence of tactile naming deficit in the left hand in our patient may be explained by leakage of information across the nondamaged regions of CC or other commissural pathway such as anterior commissure,¹¹ control of left upper extremity by ipsilateral uncrossed motor pathways descending from the left hemisphere,¹¹ and latent language processing abilities of the right hemisphere.¹¹

It is safest to say that we do not yet have a firm knowledge of all the mechanisms that influence the extent of SBS. As the patients rarely complain about the manifestations of disconnection, the incidence of this disturbance will not be known unless specific tests of callosal function are routinely performed in posttraumatic period.

The limiting factor for our case study is the unavailability of dichotic listening test,¹⁴ tachistoscopic visual test,¹⁴ and diffusion tensor tractography¹⁵ in our center.

Conclusion

Disconnection syndrome or SBS, though common after neoplastic/vascular lesions or surgical sections of CC, can also be rarely caused by traumatic lesions of CC. The rarity of the syndrome in TBI may be attributed to the lack of search for specific tests of CC and the impaired neurologic status of the patient that makes these complicated tests unfit to be performed. Through search for specific callosal signs, along

with neuropsychological tests, our case of SBS due to traumatic corpus callosal bleed was confirmed. Regular neuropsychological assessment in the recovery period from acute TBI period along with a strong needle of suspicion will make these diagnoses of SBS more numerous and their management more easier.

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