Case report

Complete callosal disconnection after closed head injury

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Received 1 July 1994; revised 5 September 1994; accepted 5 September 1994

Abstract

We report a patient with complete callosal disconnection syndrome after severe closed head injury. MRI showed a complete destruction of the corpus callosum throughout its length. Although traumatic callosal lesions are a frequent finding in autopsy studies, as well as in some recent MRI studies, clinical signs of callosal disconnection are rarely observed after head injuries and a complete disconnection syndrome has not been reported yet. This case study and the review of other reported cases suggest that posttraumatic callosal disconnection is probably often overlooked. Our patient had also a severe memory disorder that might be partly related to the bilateral involvement of the fornix, as this structure is closely adjacent to the posterior corpus callosum, and was also shown by MRI to be very probably damaged.

Keywords: Corpus callosum; Callosal disconnection; Fornix; Amnesia; Head trauma

1. Introduction

Traumatic lesions of the corpus callosum in closed head injury have been observed for many years at autopsy or as a result of radiological studies [1–6]. However, interhemispheric disconnection syndrome is rarely observed, and the literature contains only 8 such cases, with anatomical documentation of focal callosal injury in only 6 of them [7–14]. This has prompted us to report the findings of a patient who presented a total callosal disconnection syndrome of posttraumatic origin, with a destruction of the corpus callosum throughout its length as shown by MRI.

2. Case report

In 1973, at the age of 29, J.P.C., a right-handed, French-speaking man, a locksmith by trade with a normal school education, without any previous illness, suffered a road accident. He was immediately rendered unconscious and was admitted to the neurosurgery department in deep coma and with signs of decerebration. Radiographs showed no skull fracture, and an unremarkable bilateral carotid arteriography ruled out an expansive intracranial lesion. EEG showed diffuse delta bradyrhythmia. A Spitz-Nulsen ventriculocardiographic shunt was performed at the end of the second month because gamma-cisternography showed communicating hydrocephalus. After 4 weeks of deep coma, the patient remained in a vegetative state which lasted 5 months. Slowly the patient improved; he began to speak and recovered limited motor activity. 8 months after admission he showed signs of severe residual tetraspasticity and global intellectual impairment and was transferred to an institution. Because of his persistent severe memory disturbance, he was forced to remain in a nursing home for all the following years.

At the age of 49, the patient was hospitalized in our neurology department for worsening gait and balance difficulties. On neurological examination, there was an unsteady gait with widened base, mild bilateral, right predominant spasticity with right Babinski sign, but normal strength and deep tendon reflexes. Slight cerebellar dysynergia of all four limbs and a dystonic right lateral
bending posture of the head were present. There was no grasping. Sensation was normal for touch, pain and vibration. Cranial nerve examination showed left hyposmia, limitation of vertical gaze, absent convergence and irregular ocular pursuit.

The results of the neuropsychological examination are presented in the following 3 sections.

2.1. Standard neuropsychological evaluation

During several sessions of 1.5 h, the patient appeared to be alert and cooperative, but with passive and infantile attitudes. He seemed unaffected by his disabilities and failures during tests, and, when asked, reported indifferent feelings.

He was disoriented in time (date, year or season) and was unable to correctly tell his age. Spontaneous speech was sparse, with monotone dysprosody, and slowed by a moderate dysarthria with both pseudobulbar nasality and slight cerebellar scanning. Oral language was otherwise normal, grammatically correct and informative. Boston Naming Test showed 23/34 correct responses and 4 semantic paraphasias (moderate impairment), repetition, spelling, and auditory-verbal comprehension were normal. Automatic series enumeration (e.g. months) is flawless, but not in reverse order. Category and literal word list generation was insufficient (moderate and severe impairment, respectively). Reading aloud was slow, but normal, as was reading comprehension. Oral calculation was slow, but correct for basic operations. Buccofacial praxis was normal. Right–left distinction and pointing to the examiner’s or his own body parts were correct. Visual-spatial abilities were slightly impaired for discrimination of superimposed line drawings (7/12) and incomplete figure recognition (0/4) whereas colour naming and matching, recognition of examiners’ faces among a multiple choice of photographs, and identification and location of landmarks on blank geographic maps, were preserved. There was no spatial neglect on line crossing and cancellation tests, but moderate right bias on line bisection (mean 2 cm, with right hand). Left hemiextinction was noted on visual, auditory and tactile double stimulations. Luria gesture and graphic alternating sequences (with right hand), as well as automatic response inhibition for a Go/No-Go condition and the Stroop color test failed. The score on the Mini-Mental State Examination was poor (18/30).

2.2. Specific tests of interhemispheric disconnection

**Tactile naming**

When blindfolded, the patient correctly identified 8 of 10 common objects when placed in his right hand (RH). However, when placed in his left hand (LH) all responses except for one (rubber) were totally unrelated to the object, without approximation, description of shape or material, or a definition by the object’s use, but characterized by persevering errors (4 objects incorrectly called ‘rubber’ and 4 others ‘pencil’ or ‘pen’) and by a strong influence of previously seen surrounding objects.

Retrieval from a choice of three objects was successful by palpation as by pointing for 4 of 5 of the objects placed in RH, but failed for all those placed in LH, possibly because of interference due to the patient’s untrained misnaming. Left astereognosis or tactile agnosia seemed excluded by the rapid, skilful and appropriate manipulation by the fingers, a careful and lengthy palpation of significant details (e.g., comb’s teeth, key’s indentation) strikingly contrasting with anoma. Furthermore, when the patient was asked to demonstrate the use of an object held in LH, while blindfolded and without naming it, behaviour was correct for 2 of the 5 trials (e.g. scissors), not understandable for one, and in the other trials he infringed given instructions, immediately incorrectly naming a held object and then demonstrating a misuse induced by the incorrect naming.

The patient’s RH fingers were correctly named when touched by the examiner; touched fingers of LH were moved but could not be named.

**Tactile lecture**

Identification of five letters written on the skin of the palm was possible only once in LH, although incorrectly named (‘a cross’ for the letter ‘X’), and 4 times in RH (X was similarly called ‘a cross’).

**Tactile comparison**

With the patient blindfolded, two similar objects simultaneously placed one in each hand were reported without hesitation as being entirely different in all of 3 trials. The object which was held in RH was manipulated and named immediately and always first, whilst that held in LH was palpated and (wrongly) named after a delay. A single object held in both hands at the same time was reported as two distinct ones in all 3 trials: once as similar (‘two pens’), and twice as entirely different.

**Alien hand sign**

With the patient blindfolded, the examiner’s hand was correctly recognized when placed in the patient’s RH, but when it was placed in his LH, the patient described it is a rubber. The patient’s own LH when placed in his RH was identified as belonging to the examiner or to a third party, and his own RH when placed in his LH was identified as the examiner’s thumb.

Intermanual conflict, involuntary anarchic hand movements or strangeness of feeling in one limb were not observed, nor were they reported by the patient.

**Writing**

Spontaneous or dictated writing of RH showed some stroke repetitions and spatial disorganisation, with slop-
ing and telescoping lines (Table 1). With LH, spontaneous writing could not be obtained and writing on dictation was slow and illegible, and proceeded from left to right and not in a mirror fashion (Table 1). LH writing on a blackboard was no better. RH wrote correctly in 8 of 10 dictated letters, the two errors (‘m’ for ‘n’, ‘p’ for ‘b’) being rectified after repetition, and LH in only 3 of 10, some letters being recognizable but erroneous (Table 1).

Writing with mobile anagram letters of dictated words, whilst flawless with RH, failed with LH, even though the patient’s oral spelling of the words was correct at the same time: NAVIRE (meaning boat) was written RAN Vie; MAISON (meaning house) MA- SOn; and CHEVAL (meaning horse) VHCELA. However much time he was allowed and even when all letters were correctly selected, their order always remained erroneous. LH copying of anagram letters was better, since after a first approximation (NAE VIR) the patient rectified this and wrote the correct word (NAVIRE).

Calculation of written arithmetic operations failed with both hands, because of computational errors with RH and agraphia for numbers of LH.

**Ideomotor praxis**

With RH, 5 of 6 symbolic content (intransitive) gestures and 5 of 6 object’s use pantomiming (transitive) gestures were correctly executed on verbal command, body part as object being the only error among the latter. LH showed only one correct movement for each of the same 6 transitive and 6 intransitive gestures series (military salute and hair combing, respectively). With LH, the patient touched his nose with his index finger for ‘thumbing one’s nose’ and ‘making a kiss’, drummed his fingers for ‘waving good-bye’, moved his hand in front his face for ‘silencing one’ and persevered with a military salute for ‘making a threatening fist’. With the same hand, when showing ‘use of a hammer’, he repeatedly opened and closed his fist, for ‘using a saw’, he hit the table with his palm, and for ‘using a toothbrush’ and ‘drinking a glass of wine’, he wagged his index finger in front of his mouth.

Actual use of objects was flawless with both hands under free vision (actual use of objects while blindfolded has been described in Tactile Naming, as wrong naming induced LH errors).

Imitation of finger postures demonstrated by the examiner in front of the patient was prompt and flawless with RH for 4 of 5 trials, the LH succeeding in none of the same 5 trials.

Only one of four demonstrated meaningless bimanual gestures could be reproduced easily, and two more were reproduced after imitation to rectify; LH was always hesitant before adopting correct position.

There was no diagnostic apraxia.

**Constructional praxis**

Copying of simple geometric forms (e.g. square, circle) was unaltered with either hand. Copying of more complex figures (e.g. cube, house, bicycle) was impaired with both hands but, although faster with RH, was clearly better with LH (Table 2) and qualitatively distinguishable from RH performance by the presence of perspective elements for cube drawing, more details for house and bicycle drawing, as well as a global, non-piecemeal approach and initial contour outlining. Stick-construction of a bidimensional representation of a cube was correct with LH, but not with RH (Table 2).

In Kohs’ block design test the 4-element models were not correctly reproduced with either hand, but again LH seemed to perform slightly better (Table 2).

Interestingly, when the patient was free to perform the same task with both hands simultaneously, he was no more successful, but as he struggled to no avail with rectifications, he used LH almost exclusively.

**Somesthetic transfers and related tests**

While out of vision, specific posture passively impressed by the examiner on one of the patient’s hand, either right or left, was replicated in the opposite hand only once in 5 trials in each direction. When one of his fingers was touched by the examiner, the patient was consistently unable to indicate, by moving it, the corre-

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**Table 1**

Writing on dictation. Samples of the patient’s productions are shown

<table>
<thead>
<tr>
<th>Dictated letters:</th>
<th>Right hand</th>
<th>Left hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>F-Y-T-P-U</td>
<td>![Image]</td>
<td>![Image]</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dictated words:</th>
<th>Right hand</th>
<th>Left hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>IL FAIT BEAU</td>
<td>![Image]</td>
<td>![Image]</td>
</tr>
</tbody>
</table>
sponding one on the opposite side. Both tasks were flawless when performed with open eyes.

Examination of topesthesia revealed that, when touched by the examiner, a distal point on opposite upper limb was correctly indicated only 1 of 4 times by the patient’s RH and not even once by his LH.

Bimanual coordination was also impaired. Regularly alternating rhythm of both hands and reciprocal coordination (e.g. opening one hand while closing the other in turn) both failed. Bimanual manipulation of tools out of visual control proved impossible for tasks such as putting a folded paper in an envelope in spite of a 3-min

| Table 2 |
| Constructional praxis. Times for the patient’s productions are indicated in seconds |

<table>
<thead>
<tr>
<th>Right hand</th>
<th>Left hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Copy of drawings</td>
<td></td>
</tr>
<tr>
<td>![Image of cube and square drawn by right hand]</td>
<td>![Image of cube and square drawn by left hand]</td>
</tr>
<tr>
<td>![Image of car drawn by right hand]</td>
<td>![Image of car drawn by left hand]</td>
</tr>
<tr>
<td>![Image of bicycle drawn by right hand]</td>
<td>![Image of bicycle drawn by left hand]</td>
</tr>
<tr>
<td>Stick-construction of a cube</td>
<td></td>
</tr>
<tr>
<td>![Image of cube constructed from sticks by right hand]</td>
<td>![Image of cube constructed from sticks by left hand]</td>
</tr>
<tr>
<td>Kohs’ block design copy</td>
<td></td>
</tr>
<tr>
<td>![Image of block design drawn by right hand]</td>
<td>![Image of block design drawn by left hand]</td>
</tr>
<tr>
<td>![Image of block design drawn by right hand]</td>
<td>![Image of block design drawn by left hand]</td>
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<td>![Image of block design drawn by right hand]</td>
<td>![Image of block design drawn by left hand]</td>
</tr>
<tr>
<td>![Image of block design drawn by right hand]</td>
<td>![Image of block design drawn by left hand]</td>
</tr>
</tbody>
</table>
trial duration (successful in 30 sec with open eyes) and was possible only with hesitation in four other interlocking tasks (e.g. passing a pencil through a ring).

Bilateral crossed visuomotor ataxia was also noted.

Tachistoscopic naming and reading

With the patient's visual fixation kept on a central luminous locus, a series of visual stimuli were briefly (150 msec) flashed one by one on either right, left or both half-fields of a screen. For right visual half-field projections, colors of shapes and drawings were named with 75% accuracy, 80% of letters could be read, 25% of monosyllabic or bisyllabic words could be read or, in a further 35%, only their beginning could be read or spelled. Finally, from a choice of two displayed figures, the patient could point to the one corresponding to a projected word in 100% of trials with his RH.

For left visual half-field projections, 25% of colors (not significantly different from chance since only four different colors were used) and none of the drawings were named correctly, and none of the letters and none of the words could be read, or even spelled. A corresponding figure could not be pointed to with LH for any of the projected words. Most often, the patient denied left-sided stimuli occurrence.

Dichotic listening

Tape-recorded and earphone-presented monosyllabic and bisyllabic words were repeated out loud in normal range for right (8/10) and left (9/10) monaural listening, but dichotic listening showed total left ear extinction (0/30 for left ear presented words; for right ear presented words, 18/30 were correct and 5 others were phonetically related). The patient denied having heard any sound on the left side.

Using neither LH, nor RH, left ear performance was improved in a similar task where the patient had to point with RH or LH to one of two displayed figures which corresponded to one of the heard words.

2.3. Assessment of memory

Immediate memory was normal with verbal and visuospatial span in the normal ranges (6 and 5, respectively, 50th centile). Acquisition of new information was severely impaired. After a few minutes, the patient recalled only 3 of a 10-word list and none of three hidden objects. Recognition in multiple choice condition was no better. Recall for events that occurred before his accident was also severely impaired, including both autobiographical and public events. For example, he could remember his wife, but not his two children, born 8 and 5 years earlier before his accident. Memory of major political or public events was equally poor for the same period.

3. Results

3.1. Magnetic resonance imaging findings

Serial T1 and T2-weighted axial and sagittal MR images revealed a severe and T2-hyperintense atrophy of the entire corpus callosum, from rostrum to splenium inclusive. The corpus callosum was almost absent on sagittal views (Fig. 1A). Anterior commissure could not be reliably distinguished. There were no associated malformations suggestive of agenesis [15,16]. The fornix was likely to be also extensively destroyed, at least from its posterior pillars (crura fornici) to its body (corpus fornici) (Fig. 1B and C). Medial temporal lobe structures, especially at the hippocampal level, appeared to be intact (Fig. 1D).

Besides this, there was a diffuse cortical and subcortical atrophy, marked ventricular enlargement with the ventriculocardiac shunting catheter’s inlet orifice in the right lateral ventricle’s body, T2-hyperintense white matter and contusion scars in both frontal lobes, brain stem atrophy without visible focal lesion, and small chronic subdural hematomas around both cerebral hemispheres. The cerebellum was normal.

4. Discussion

Twenty years after a severe closed head injury (CHI), our patient showed a frontal-predominant intellectual impairment in association with dense anterograde-retrograde amnesia and a typical complete callosal disconnection syndrome (CDS), including tactile anoma, agraphic esthesia, agraphia and ideomotor apraxia of the left hand, right-predominant constructional apraxia, left ear total suppression in dichotic listening, left hemislexia and visual hemianomia, bilateral crossed visuomotor ataxia, and loss of bimanual coordination and somesthesia transfers [8,17–20]. To our knowledge, this is the first reported case of a complete CDS of post-traumatic origin, which has been demonstrated both clinically and anatomically. Only 8 other cases of post-traumatic CDS have been described in the literature [7–14], but all were partial CDS with focal damage of the CC, and not all of them were anatomically confirmed.

Only the patient described by Schott et al. [7] in 1969 had signs of a similar extensive CDS (left tactile anoma, agraphia and ideomotor apraxia, bilateral constructional apraxia, left tactile and visual hemiextinction, crossed visuomotor ataxia) 3 months after a CHI and coma lasting a few days, but there was no anatomical confirmation of the suspected callosal damage. Again, without anatomical confirmation, Brion and Jedynak [8] observed an isolated left tactile anoma in a patient who also had a Korsakoff syndrome after CHI. The first anatomo-clinical correlation was done by Rubens et al.
[9] in a patient with left unilateral agraphia and apraxia after CHI, who had a severe callosal degeneration with gliosis and hemosiderin deposits at autopsy.

A few cases with MRI confirmation have been reported in recent years. Three had lesions in the CC body or posterior portion and frontal contusions [10–12], one with left ideomotor and constructional apraxia, left agraphia, impaired somesthetic transfers, left ear verbal extinction and left visual half-field alexia [10], one with left ideomotor apraxia without agraphia, but with right constructional apraxia, and impairment of left tactile naming and of somesthetic transfers [11], another with unilateral left ideational apraxia (i.e. failure in accurate use of common objects with left but not right hand) [12]. Still another patient was mentioned incidentally in the study by Levin et al. [13] of dichotic listening after CHI, who had left tactile anomaia and impaired somesthetic transfers, but no dichotic listening abnormality (pa-
Patient M.H., figure 3 in ref. 13). Previously, Warren and Alexander [14] had localized the interhemispheric auditory pathways in reporting a posttraumatic hemorrhagic lesion in the posterior third of the CC body, shown by CT, in association with bifrontal contusions, which caused an almost complete left ear suppression in dichotic listening, and also impaired crossed matching of hand postures. Most of all these patients had had a severe CHI followed by rather prolonged coma.

The small number of reported cases with a post-traumatic CDS is most likely due to the fact that such signs are not systematically looked for after CHI. Only Levin et al. [13] investigated prospective dichotic verbal listening in a series of 69 patients 3 days to 3 months after CHI. They found an increased right ear advantage directly related to the severity of the CHI [13]. However, MRI showed focal lesions of the CC in only 3 of those patients and dichotic listening impaired performance was not correlated with other tests of interhemispheric disconnection (i.e. left hand tactile naming and writing, somesthesia transfer), but was rather related to deep cerebral lesions interfering with auditory pathways [13]. Nevertheless, errors in left tactile naming and writing, as well as in somesthesia transfer, were more frequent in patients with severe CHI than in patients with mild CHI and in controls [13].

From the anatomical standpoint, callosal lesions have been known and well described for many years [1-4; unpublished data]. They may be diffuse, at the axonal level, or focal, single or multiple, necrotic or hemorrhagic or both, and their mechanisms are varied [3,21]. Focal macroscopic lesions are frequent and encountered in 16-40% of autopsies after fatal CHI [1,4,22,23]. Most are produced by torsion or shearing strains at the time of head trauma and are associated with diffuse axonal injury of cerebral white matter [1-4,21]. Several recent neuroradiological studies using MRI [5,6,24] confirmed a high incidence of traumatic callosal damage, seen in 22-49% of CHI in the acute stage [5,24] and 24-47% in the remote stage [5,6], but missed with CT in 25% to 100% of the cases [5,6,25]. Focal macroscopic lesions tend to occur mainly in the posterior part of the CC (body and splenium) and their size is usually limited [1,3,21]. A destruction of the CC throughout its length, as it is the case in our patient, was present in only 18% of autopsy cases investigated by Lindenberg et al. [1].

Considering all observed disconnection signs, an extensive lesion could also be suspected for the patient of Schott et al. [7], but anatomical or radiological confirmation is lacking. All the other reported cases of posttraumatic CDS we have reviewed above had a limited focal lesion, most being in the body or in the splenium [10,11,13,14].

A further point of interest is the associated lesion of the fornix that is suspected by our patient’s MRI. Neuropathological [2-4] and neuroradiological [5,26] studies showed a frequent extension of callosal traumatic damage towards adjacent structures, such as the pillars of the fornix and the interventricular septum pellucidum. This feature might be involved in our patient’s severe amnesic syndrome, as well as in the major amnesic disorders which are also frequently noted in other reported cases with a posttraumatic CDS [8-11,14]. Persistent and significant memory disturbances after surgical callosotomy have been related to concurrent incidental section of the fornix [27], and similarly, the presence of an amnesic syndrome in splenial tumors has been underscored and ascribed to the involvement of the fornix, which is closely applied to the posterior part of the CC [28]. Moreover, isolated damage to the fornix can produce a definite memory impairment, which is imputed to the interruption of hippocampal-diencephalic pathways [29,30]. Memory disorders are frequent after severe CHI, but remain unexplained. Because MRI showed that our patient’s medial temporal lobe structures are intact, we believe that complete destruction of the fornix might play a crucial role in his profound amnesia. Of course, bilateral frontal, as well as diffuse axonal lesions might also contribute to its severity.

Considering the high frequency of CHI, we suspect that post-traumatic CDS is not uncommon, but often goes undetected because signs of interhemispheric disconnection are overlooked if not searched for by specific tests [8,17,18]. Damage to the CC is a frequent neuropathological and radiological finding, and CDS belongs to the constellation of neurobehavioural sequela of CHI. Although it seems to be most often partial and restricted to the posterior corpus callosum, leading to varying disconnection signs, it may also sometimes be complete, as is the case in our patient. Furthermore, because of possible concomitant damage to the adjacent fornix, a particularly severe amnesia could frequently occur in association with post-traumatic CDS.

Acknowledgement

We thank Cendrine Hirt for collaboration in the neuropsychological evaluation of the patient.

References


[21] Reference deleted.


