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Corpus callosum lesions after closed head injury in children: MRI, clinical features and outcome

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Summary. Thirty-four children who sustained moderate to severe closed head injury underwent magnetic resonance imaging (MRI). Eight (24%) had MRI evidence of corpus callosum injury, most commonly within the posterior body and splenium. In contradistinction to reports in adults, there was no definite relationship between callosal injury and lower initial Glasgow Coma Scale scores, nor was there a significantly higher incidence of primary brain-stem lesions, diffuse axonal shear injury or intraventricular hemorrhage. In none of these 8 children did the initial admission computed tomography show evidence of callosal injury. Callosal injuries on MRI are not necessarily a poor prognostic finding, the majority of the 8 children showing good functional recovery.

Key words: Brain – Magnetic resonance imaging – Trauma – Corpus callosum

It is well documented that traumatic corpus callosum lesions are common at autopsy in fatal head injuries. Several neuropathologic reports of diffuse axonal injury (DAI) in adults sustaining closed head injury (CHI) have documented the vulnerability of the corpus callosum [1–6]. Lindenberg et al. [5] found that lesions of the corpus callosum were present in 16% of fatal CHI, typically with unilateral extension rather than limited to the midline. Of 45 neuropathologically confirmed cases of DAI studied by Adams et al. [1], all had the triad of focal lesions in the corpus callosum and the dorsolateral quadrants of the rostral brain stem, and microscopic evidence of diffuse damage to axons. Although this "triad" was present in only 15 of 34 cases of DAI described by Blumbergs et al. [4], callosal lesions were present in more than two-thirds of the series. Corpus callosum lesions were present in 69% of primates subjected to angular acceleration in various directions by Gennarelli et al. [7]. The presence of focal lesions in the corpus callosum was related to the severity of DAI.

In contrast to these converging neuropathologic studies, there is a paucity of magnetic resonance imaging (MRI) data pertaining to corpus callosum injury. In a recent MRI study [8] corpus callosum lesions were present in 47% of CHI patients who were heterogeneous as regards age, type, severity and chronicity of injury. Callosal lesions were present in 31 of 63 patients (49.2%) studied in the acute phase of injury and in 6 of 15 (40%) studied several years after head trauma; consistent with neuropathologic reports, Gentry et al. [8] found the splenium to be most frequently involved (95% of callosal injuries) and lesions were present in the body or genu in 51% of the cases with callosal injury. Callosal injury was associated with a significantly higher incidence of lesions of subcortical white matter, the basal ganglia and brain stem [8]. Callosal lesions were also associated with more severe CHI as reflected by lower Glasgow Coma Scale (GCS) scores [9].

In this paper we report the MRI and clinical features of corpus callosum lesions in head-injured children. In view of previous neuropathologic and MRI studies [1–6], we postulated that corpus callosum injury in children would probably be associated with lesions in subcortical white matter, basal ganglia and brain stem as well as with a lower GCS score and poorer outcome.

Subjects and methods

Prospective MRI studies of 34 head-injured children were reviewed to assess the frequency of corpus callosum injury. All studies were performed after obtaining informed consent from the parents and the investigation was approved by the institutional review board.

Criteria for selection of children who sustained moderate to severe CHI included: (1) a postresuscitation GCS score of 13 or below and/or acute brain lesions detected by computed tomography (CT) (this latter criterion is incompatible with a mild CHI); (2) no history of neuropsychiatric disorder (e.g. epilepsy, mental retardation) prior to the CHI; (3) no evidence of child abuse or other cause of repeated head trauma; (4) age 15 years or below at time of injury and at least 6 years at time of MRI. Confining the study to school-aged children facilitated a standard assessment of outcome as reflected by academic progress and psychosocial function. All children were recruited according to the above criteria rather than

Table 1. Clinical and demographic features of patients with and without corpus callosum lesions

Case No.	GCS	Duration of coma (days)	Age at injury (years)	Interval (days)	Age at MRI (years)	Sex	Mechanism	Complications	GOS
Corpus callosum lesion									
1	3.0	19.0	7.1	889	9.3	M	MVA		MD
2	3.0	21.0	5.8	901	8.3	M	Ped/MVA		MD
3	4.0	6.0	13.1	87	13.3	M	MVA		GR
4	6.0	5.0	15.9	94	16.2	M	MVA		GR
5	7.0	5.0	5.3	848	7.6	M	Ped/MVA	ICP	GR
6	8.0	3.0	2.6	1849	7.6	F	MVA		GR
7	9.0	2.0	2.6	1437	6.6	M	Falling object		GR
8	11.0	0.08	5.7	128	6.0	M	Ped/MVA		GR
X	6.4	7.0	7.3	779	9.4				
SD	2.9	7.9	8.3	653	3.6				
Without corpus callosum lesion									
9	3	17.0	5.2	726	6.8	M	MVA	ICP	GR
10	3	13.0	4.9	1199	8.3	M	MVA		MD
11	3	23.0	7.9	141	8.3	M	Falling object		MD
12	6	14.0	4.9	1654	9.5	M	MVA	ICP	MD
13	6	6.0	6.5	114	6.8	M	Ped/MVA		GR
14	6	0.125	13.6	442	14.8	M	Ped/MVA		GR
15	6	15.0	4.4	1218	7.7	M	Ped/MVA		MD
16	7	1.0	12.2	1434	16.1	F	MVA		GR
17	7	7.0	7.2	106	7.5	M	MVA		MD
18	7	16.0	5.5	1077	8.5	M	MVA		GR
19	7	13.0	4.8	1370	8.5	F	MVA		MD
20	7	14.0	4.8	428	6.0	F	Ped/MVA		MD
21	7	12.0	12.6	264	13.3	F	Ped/MVA		MD
22	7	3.0	14.0	116	14.3	F	MVA		GR
23	7	4.0	14.3	76	14.5	M	Ped/MVA	SZ	GR
24	8	0.125	4.5	1044	7.4	M	MVA		GR
25	8	4.0	1.7	1869	6.8	M	Ped/MVA	SZ	GR
26	9	15.0	7.9	919	10.4	M	MVA		MD
27	11.0	0.054	7.1	958	9.8	F	MVA		GR
28	12.0	N/A	10.7	118	11.0	M	MVA	ICP	MD
29	12.0	0.210	12.2	923	14.7	M	Bike		GR
30	12.0	0.0	7.7	103	7.9	M	Sports		GR
31	14.0	0.125	11.7	78	11.9	M	Falling object		GR
32	15.0	0.0	7.4	302	8.2	F	Sports		GR
33	15.0	0.0	8.1	723	10.1	M	Falling		GR
34	15.0	0.0	2.9	1522	7.1	F	MVA		GR
mean	8.5	7.1	7.9	727.8	9.9				
SD	3.7	7.3	3.6	571.8	3.0				

GCS, Glasgow Coma Scale score; GR, good recovery; GOS, Glasgow Outcome Scale; ICP, raised intracranial pressure; MD, moderate disability; MVA, motor verhicle accident; Ped, pedestrian; SZ, seizures

by clinical referral. Children hospitalized on the neurosurgery service were selected according to the criteria; we also recruited consecutively admitted children during their initial hospitalization for CHI and scheduled their MRI at approximately 3 months after injury. Past experience indicated that unsedated, severely injured children were often unable to co-operate for MRI before this time. This postinjury interval for MR was consistent with our focus on correlating long-term outcome with concurrent MRI findings. Overall outcome of head injury was evaluated by the Glasgow Outcome Scale (GOS) [10]. Table 1 displays the demographic and clinical features of the patients, grouped according to the presence of a corpus callosum lesion and their lowest postresuscitation GCS score.

MRI was performed using a 0.5 T system. T1-weighted (600/30/2) 5-mm midsagittal images were obtained; T2-weighted (3000/30/120/1) and T1-weighted (300/20/2) coronal images were also obtained, using a 7.5-mm slice thickness and a 1.5-mm gap. A gradient echo sequence was not obtained because of the longer scanning time and increased likelihood of movement artifact.

Results

Injury to the corpus callosum was detected in 8 (24%) of the 34 head-injured children (Table 1). Five had lesions confined to the splenium/posterior body junction, 1 involving both the body and the splenium, 1 confined to the splenium, and 1 involved the genu/rostrum (Figs. 1–4).

It is seen in Table 1 that the presence of callosal damage was unrelated to the age at injury, $F\ (1,32) = 0.14$, $P < 0.71$ or to the interval since injury $F\ (1,32) = 0.05$, $P < 0.83$. Approximately half the children in each group were passengers in motor vehicle accidents and about a quarter were pedestrians injured by motor vehicles. Severity of impaired consciousness, as measured by the lowest postresuscitation GCS score, was comparable in the children with callosal lesions and the other children studied after CHI, $F\ (1,32) = 2.16$, $P < 0.15$. Duration of im-

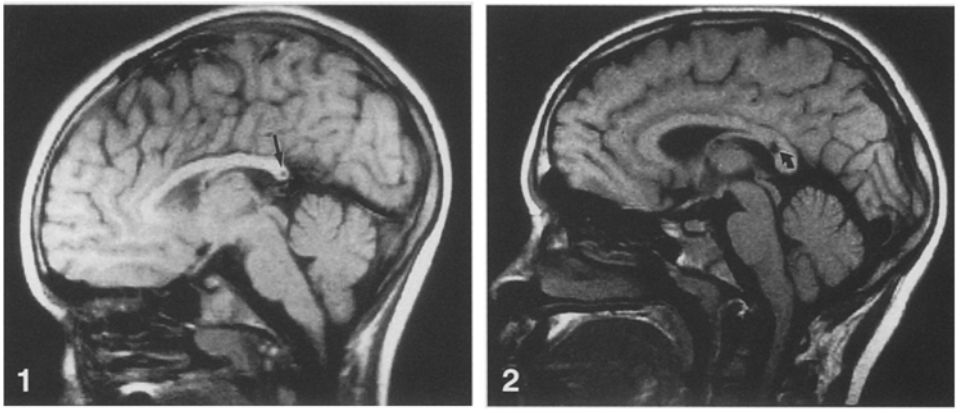

Fig. 1. Case 6. Sagittal T1-weighted (600/30) MRI shows a small area of decreased signal *(arrow)* within the splenium of the corpus callosum

Fig. 2. Case 3. Sagittal T1-weighted (600/30) MRI showing focal encephalomalacia *(arrow)* at the junction of the splenium and posterior body of the corpus callosum

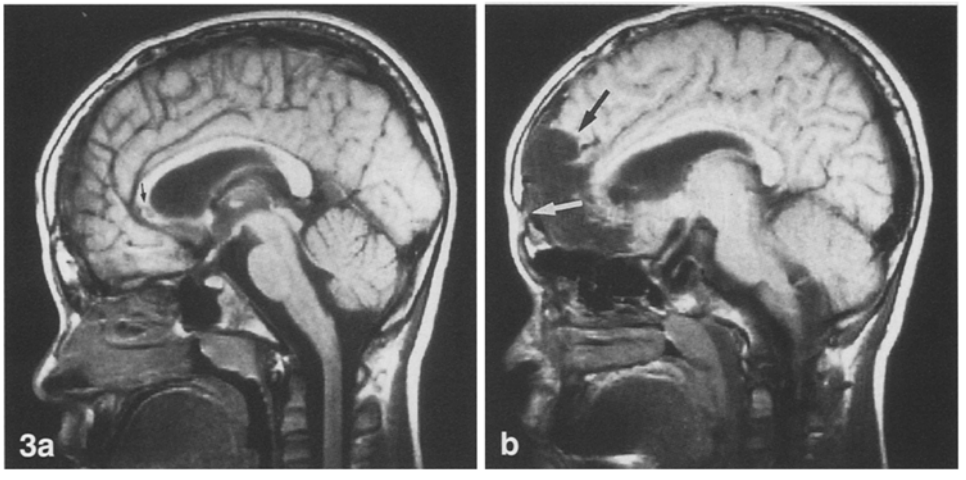

Fig. 3 a, b. Case 2.
a Sagittal T1-weighted image (600/30) showing post-traumatic encephalomalacia *(arrow)* within the genu/rostrum of the corpus callosum.
b Parasagittal T1-weighted image (600/30) to the left of midline demonstrates severe frontal post-traumatic encephalomalacia *(black arrow)*. In addition, a defect is noted within the frontal bone representing the site of a diastatic skull fracture *(white arrow)*

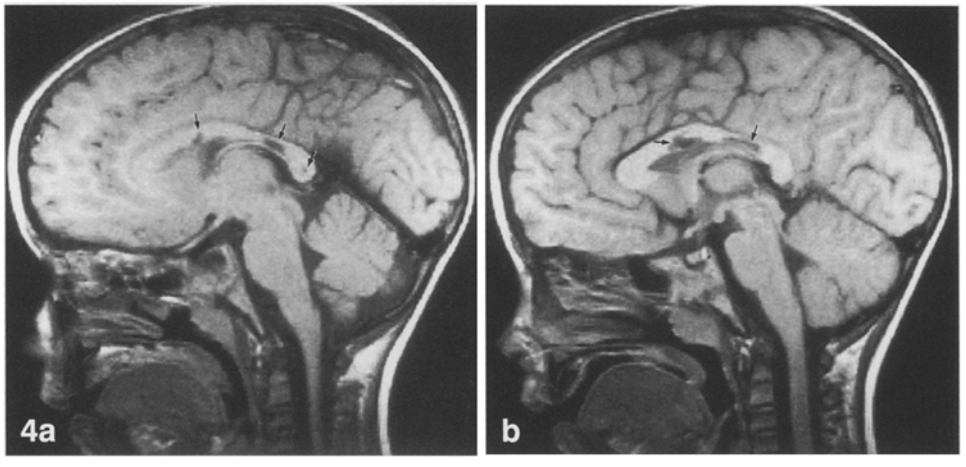

Fig. 4 a, b. Case 8. Adjacent sagittal T1-weighted images (600/30) showing extensive post-traumatic encephalomalacia *(arrows)* involving the splenium and mid and posterior body of the corpus callosum

paired consciousness did not differ between the callosal-injured and other CHI groups, $F\,(1,31) = 0.03$, $P < 0.86$. Two children with callosal lesions and 1 child without callosal injury had acute extra-axial hematomas which were evacuated surgically. Of 7 children who underwent surgical repair of a depressed skull fracture, only 1 had a callosal lesion. The most common complication of CHI was intracranial hypertension which occurred in 1 child with and 3 children without callosal lesions.

The GOS category at the time of MRI is listed for each patient in Table 1. A good recovery denotes return to regular school curriculum without onset of academic problems or behavioral disturbance, whereas moderate disability indicates problems in academic and/or behavioral domains following injury. Six of the callosal lesion group (75%) and 16 children without callosal lesions (62%) made a good recovery.

Associated MRI features

In view of the postulated relationship between corpus callosum injury and the severity of DAI, we studied the presence of lesions in the brain stem and deep cerebral white matter [1]. As shown in Table 2, white matter lesions were present in 4 of the 8 children (50%) with cor-

Table 2. Relationship of corpus callosum injury to other types of intracranial injury +

Corpus callosum Injury																	
Case No.	Frontal			Temporal			Parietal			Occipital			Corpus callosum				
	G	W	G/W	G	W	G/W	G	W	G/W	G	W	G/W	AB	PB	S	Extracerebral
1	+				+										+	Hygroma
2		+	+										+			
3														+		
4		+													+	Hygroma
5			+		+										+	
6		+						+							+	
7	+														+	
8														+	+	
·/· of group																
	25	38	25	13	0	13	0	13	0	0	0	0	13	25	75	25
Without corpus callosum injury														BG	IVH	
9	+	+													+	
10			+													
11			+											+		Hygroma
12	+															
13	+	+		+											+	
14						+										
15		+													+	
16																
17		+														
18		+	+					+								
19																
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21		+														
22																
23																
24																
25																
26		+				+			+							
27						+										
28																
29																Temporal hygroma
30																
31																
32							+									
33		+														Parietal hygroma
34	+															
·/· of group																
	15	31	12	4	0	12	4	4	4	0	0	0	4	12	12	

AB, anterior aspect or anterior body; BG, basal ganglia; G, grey matter; IVH, intraventricular hemorrhage; PB, posterior body; S, splenium. W, white matter

pus callosum injury and 9 of the 26 patients (35%) without corpus callosum lesions, a nonsignificant difference according to Fisher's Exact Test. Cortical lesions were found in 3 patients with callosal injury (38%) as compared with 5 children who were spared callosal injury (19%), a difference which was also not significant. Lesions overlapping gray and white matter were present in 2 children with callosal injury (29%). Table 2 indicates that the frontal lobes were the most common site of parenchymal lesions in both groups of patients. If corpus callosal lesions are an indication of structural alterations progressing to deeper areas of the brain, then an association with deep gray lesions would be predicted [11]. Contrary to expectation, midline lesions were not associated with callosal lesions. The 1 child with of a basal ganglia lesion and 2 with intraventricular hemorrhage were in the group without callosal injury. Two patients with (25%) and 3 children without callosal injury (12%) had extracerebral lesions.

Discussion

Callosal injury has previously been regarded as a poor prognostic sign on CT due to the fact that in order to visualize this injury there had to be a hemorrhagic component implying a major traumatic force. With MRI, however, far more subtle, nonhemorrhagic injuries can be detected, accounting for the lack of associated findings within the brain stem or intraventricular hemorrhage, and thereby not necessarily implying a poor prognosis. MRI, with its multiplanar capabilities, is far more sensitive than CT to callosal lesions. It is also superior for delineation of injuries to the anterior commissure, fornix and septum

pellucidum. Although CT depicts foci of hemorrhage well, the majority of callosal injuries are nonhemorrhagic, CT therefore being insensitive to them [8]. In our series the 5-mm sagittal T1-weighted (600/30/2) sequence best demonstrated chronic callosal injury as focal areas of decreased signal similar to that of cerebrospinal fluid, representing focal encephalomalacia, giving increased signal on T2-weighted coronal images. Although our correlation of long-term outcome of injury with MRI findings necessitated a postinjury scanning interval of at least 3 months, we acknowledge that MRI sooner after injury might have revealed additional lesions.

Lesions of the corpus callosum were identified by Strich [6] as a prominent macroscopic feature of DAI, a finding confirmed by later neuropathologic studies [1–4] and in the experimental model of subhuman primate injury [7]. Lindenberg et al. [5], who found callosal lesions in 16% of consecutive fatal cases of CHI, described three subgroups according to the extent and area of the callosum involved. Lesions confined to the splenium characterized the largest subgroup (about 40%), a pattern which was corroborated in an MRI study of CHI in a series comprising predominantly adults [8]. Our findings in children also indicate the vulnerability of the splenium and posterior body of the corpus callosum. These lesions appear to be related to DAI rather than secondary to raised intracranial pressure.

Although we found callosal lesions in a lower percentage of children (24%) surviving CHI than the series (47% of series) reported by Gentry et al. [8], several factors including age-related differences in the pathophysiologic features of injury might explain this disparity. Apart from an overall difference in the proportion of CHI patients with corpus callosum lesions in the present study as compared to the previous report, we were unable to demonstrate a significant association between callosal injury and deep lesions in the cerebral white matter, basal ganglia or brain stem, and intraventribular hemorrhage: they occurred with comparable frequency in the callosal and noncallosal groups. We recognize, however, that the longer interval from injury to MRI in our study could have resulted in resolution of associated lesions.

Consistent with neuropathologic studies of CHI in adults [1–3], our MRI findings show that the frontal lobes are particularly vulnerable to damage. Comparison of the groups with and without callosal lesions disclosed no significant differences in severity (according to GCS scores), chronicity or the mechanism of injury. However, 7 of the 8 children who had callosal lesions were occupants of or struck by motor vehicles. Although these mechanisms of injury are consistent with DAI by angular rotation of the brain [1], they also predominated in children with callosal lesions. Pending replication, our findings raise the possibility that the pattern of DAI in the child's brain differs from that found in adults. Distinct features of the shearing injury in the young brain may produce more variable MRI findings than in adults. Why these injuries involve predominantly the splenium or junction of the posterior body and splenium of the corpus callosum is thought to be related to shear strains at the junction of the corpus callosum with the septum pellucidum and fornix when the massive cerebral hemispheres are displaced. The rigid falx, broader posteriorly, prevents the cerebral hemispheres moving across the midline, further straining the connecting corpus callosum. Anteriorly the falx is shorter and allows transient displacement across the midline [8].

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