Restricted Diffusion in the Corpus Callosum in Various Pediatric Diseases

Poster No.: C-0529
Congress: ECR 2013
Type: Scientific Exhibit
Authors: W. K. Kim, H. S. Hong, A. L. Lee, J.-G. Cha, H. K. Lee, W. K. Bae; Bucheon/KR
Keywords: MR, Pediatric, CNS, Comparative studies, Trauma, Seizure disorders
DOI: 10.1594/ecr2013/C-0529

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method ist strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Purpose

To evaluate the reversible restricted diffusion in the corpus callosum in pediatric patients with clinical findings, and to discuss the possible pathogenesis of these lesions.

Methods and Materials

Patients

Between January 2007 and November 2011, we recruited 7 pediatric patients who revealed restricted diffusion in the corpus callosum (CC) on brain MRI. The MRI results of 7 patients (five boys and two girls; age range, 0-19 years; mean age, 8.6 years) were retrospectively reviewed with patients’ symptoms, clinical histories, medications, laboratory findings, clinical diagnoses, and prognoses.

MR Imaging and Image Analysis

All scanning was performed using a 1.5 T or 3.0 T MRI scanner. The MRI protocol included T1-weighted (T1-W) spin-echo with or without contrast agents, T2-weighted (T2-W) fast spin-echo, fluid-attenuated inversion-recovery (FLAIR), diffusion-weighted imaging (DWI), and apparent diffusion coefficient (ADC) mapping. Diffusion-weighted images were acquired at a b value of 1000 s/mm$^2$.

MRI of all these children were assessed by two experienced board-certified pediatric neuroradiologists and one resident. All images were reviewed individually. We analyzed MRI findings, such as location and shape of the CC lesions, signal abnormalities on DWI and an ADC map. Conventional T1- and T2-W images were reviewed to assess pre- and coexisting parenchymal lesions or other brain diseases. On follow-up MRI, we defined reversible restricted diffusion in the CC when the high signal intensity (SI) lesions on DWI and low SI lesions on an ADC map in the CC, had disappeared on all sequences during the follow-up study. Additionally, clinical diagnoses and outcomes were reviewed and compared to the MRI findings for the seven patients.

Results

Clinical Outcome of Patients
Table 1 lists the details of the clinical data for the seven patients. Clinical data were analyzed retrospectively, with particular attention to identifying the etiology of the lesions. The children's symptoms, which prompted MRI, included: drowsy to stuporous mentality in three of the patients (cases 1-3), seizures in two (cases 4, 7), headache with fever in one (case 5), and nausea with vomiting in another (case 6). Diseases and conditions associated with the lesions included trauma in three patients (cases 1-3), neonatal seizure (case 4), clinically suspected mild encephalopathy (case 5), MS (case 6), and seizure with subdural hygroma (case 7). Clinical outcomes were good in six patients (cases 1-6), but poor in one (case 7).

**MRI Findings**

Table 2 lists the details of MRI findings for the seven patients.

**Location and Shape**

The callosal lesions were located in both the splenium and the genu in two patients (cases 1, 2) (Fig. 1), the splenium and the body in one patient (case 3) (Fig. 2), and the splenium only in four patients (cases 4-7) (Figs. 3-6). All of the cases involved the splenium. The shape of the lesion was round-to-ovoid in four patients (cases 1, 3, 5, 6) (Figs. 1, 2, 4, 5) and linear in three patients (cases 2, 4, 7) (Figs. 3, 6).

**Signal Abnormalities**

All patients exhibited high SI lesions on DWI, while six of the seven showed a low SI lesions on the ADC map in the CC (Figs. 1-6). Only one patient (case 5) showed an iso-SI on an ADC map (Fig. 4B). The ADC value was not evaluated because all cases were evaluated retrospectively. On axial T1- or T2-W and FLAIR images, six of the seven patients showed a high SI on T2-WI or FLAIR image and a low SI on T1-WI, except for one patient (case 4), who showed no obvious signal abnormality on T2-WI (Fig. 3C).

**Other Parenchymal Lesions**

Regarding these case series, an isolated corpus callosum lesion was apparent in only one case of clinically suspected mild encephalopathy (case 5) (Fig. 4). Other parenchymal lesions were found in six cases. In the trauma cases (cases 1-3), crescentic subdural hygroma (Fig. 1C), multiple hemorrhagic foci, and contusions were observed in brain parenchyma (Fig. 2C). In the patient with neonatal seizure (case 4), crescentic subdural hemorrhage was observed along the tentorium and interhemispheric fissure (not shown). In the patient with MS (case 6), multifocal nodular high SI lesions were observed in both thalami, the right caudate nucleus, and the periventricular white matter of the left parietal lobe (Fig. 5C). In case 7, crescentic subdural hygroma was observed in both frontotemporoparietal regions (Fig. 6C). Additionally, ill-defined high SI lesions on DWI,
with subtle cortical sulcal effacement, were present in both occipital lobes (Fig. 6A), without low SI on ADC map, suggesting vasogenic edema.

**Follow-Up MRI**

The interval between the initial and follow-up MRI scans ranged from 17 days to 17 months. Upon follow-up MRI, six patients showed complete resolution of signal abnormalities in the corpus callosum (cases 1-6), whereas, in one patient, the restricted diffusion in the corpus callosum was persistent (case 7) (Fig. 6D, E).

**Images for this section:**

![Images](image_url)

**Fig. 1:** A 4-year-old boy with drowsy mentality caused by motorcar accident (case 1). Initial MRI (A-C) shows well-defined, tiny, ovoid-shaped bright SI lesions (arrows) on DWI (A) in the genu and the splenium, dark SI (arrow) on an ADC map (B) in the genu of the corpus callosum. T2-WI (C) shows high SI lesions in the genu and the splenium (arrows) and crescentic subdural hygroma in the left frontal region (arrowhead). Follow-up MRI (D-F) after 6 months shows complete resolution of signal abnormalities on all sequences.
Fig. 2: A 14-year-old girl with stuporous mentality following a motorcycle traffic accident (case 3). Initial MRI (A-C) shows multifocal nodular high SI lesions (arrow) on DWI (A) and low SI (arrow) on an ADC map (B) in the splenium and the body of the corpus callosum. FLAIR imaging (C) shows high SI lesions in the splenium and the body (arrow), with hemorrhagic contusion in the left frontotemporal cortex (arrowhead). Follow-up MRI (D-F) after 17 months shows complete resolution of signal abnormalities on all sequences.
Fig. 3: A male infant with clonic seizures (case 4). Initial MRI (A-C) shows bilateral linear high SI lesions (arrows) on DWI (A) and low SI (arrows) on an ADC map (B) in the splenium of the corpus callosum. On T2-WI (C), the signal abnormality is not obvious. Follow-up MRI (D-F) after 17 days (20 days of age), shows resolution of signal abnormalities on DWI (D) and an ADC map (E). Follow-up T2-WI (F) also shows no signal abnormality.
Fig. 4: A 14-year-old boy with headache and fever (case 5). Initial MRI (A-C) shows ovoid high SI lesion (arrow) on DWI (A) and iso-SI (arrow) on an ADC map (B) in the central portion of the splenium. T2-WI (C) shows well-defined, ovoid, high SI (arrow) in the splenium of the corpus callosum. Follow-up MRI (D-F), after 1 month, shows resolution of signal abnormalities on all sequences.
Fig. 5: A 19-year-old man with a history of multiple sclerosis (case 6) experienced nausea and vomiting. Initial MRI (A-C) shows focal, nodular, bright SI lesion (arrow) on DWI (A) and dark SI (arrow) on an ADC map (B) in the right lateral aspect of the splenium. T2-WI (C) shows high SI lesion in the splenium (white arrow) and other multifocal nodular high SI lesions in both thalami, right caudate nucleus, and periventricular white matter of the left parietal lobe (black arrows), suggesting multiple sclerosis. Follow-up MRI (D-F), after 17 months, shows resolution of signal abnormalities in the splenium on all sequences.
Fig. 6: A 3-month-old girl with episodes of seizures (case 7). Initial MRI (A-C) shows bilateral linear bright SI lesions (arrows) on DWI (A) and low SI (arrows) on an ADC map (B) in the splenium of the corpus callosum, crossing the midline. Ill-defined high SI lesions on DWI, with subtle cortical sulcal effacement, are seen in both occipital lobes (black arrows) without low SI on ADC map, suggesting vasogenic edema. FLAIR imaging (C) shows high SI in the splenium (arrows) and crescentic subdural hygroma in both frontotemporoparietal regions (asterisk). After 1 month, the girl revisited our hospital for general weakness. Follow-up MRI (D-F) shows that the bilateral linear lesions in the splenium are persistent (arrows) on DWI (D) and an ADC map (E). FLAIR imaging (F) shows the amount of subdural hygroma in both frontotemporo-parietal regions has increased, with complicated hemorrhage (asterisk). A burr-hole craniotomy was performed for drainage of the subdural hygroma.
Table 1: Summary of Clinical Data for Patients with Corpus Callosum Lesions

| Patient no. | Age/Gender | Initial symptoms            | Diseases and conditions         | Treatment      | Clinical outcome |
|------------|------------|-----------------------------|---------------------------------|----------------|-----------------|
| 1          | 4 yr/M     | Drowsy mentality            | Trauma by TA                    | Conservative   | Good            |
| 2          | 9 yr/M     | Drowsy mentality            | Trauma by TA                    | Conservative   | Good            |
| 3          | 14 yr/F    | Stuporous mentality         | Trauma by TA                    | Conservative   | Good            |
| 4          | 3 d/M      | Seizure                     | Neonatal seizure                | Phenobarbital  | Good            |
| 5          | 14 yr/M    | Headache, fever             | Clinically mild encephalopathy  | Vancomycin     | Good            |
| 6          | 19 yr/M    | Nausea, vomiting            | Multiple sclerosis              | Steroid pulse therapy with interferon | Relapsing and remitting |
| 7          | 3 mo/F     | Repeated seizures           | Seizure with subdural hygroma   | Phenobarbital and phenytoin | Burr-hole drainage due to aggravation of subdural hygroma |

Note. - d = day, F = female, M = male, mo = months, No = number, TA = traffic accident, yr = years
### Table 2: MRI Findings for Patients with Corpus Callosum Lesions

| Patient no. | Location         | Shape   | DWI  | ADC | T2WI or FLAIR | T1WI | Other lesions                                      | Follow-up MRI |
|------------|------------------|---------|------|-----|---------------|------|---------------------------------------------------|---------------|
| 1          | Splenium and genu | Tiny ovoid | H    | L   | H             | L    | Subdural hygroma and hemorrhagic foci             | 6 mo          |
| 2          | Splenium and genu | Linear  | H    | L   | H             | L    | Hemorrhagic foci in both frontal lobes            | 2 mo          |
| 3          | Splenium and body | Round   | H    | L   | H             | L    | Hemorrhagic contusion in left frontal lobe        | 17 mo         |
| 4          | Splenium         | Linear  | H    | L   | I             | I    | Microhemorrhage in both parietal lobes            | 17 d          |
| 5          | Splenium         | Round   | H    | I   | H             | L    | (-)                                               | 1 mo          |
| 6          | Splenium         | Round   | H    | L   | H             | L    | Multiple sclerosis involving BG, PVWM of left parietal lobe | 17 mo |
| 7          | Splenium         | Linear  | H    | L   | H             | L    | Subdural hygroma                                 | 1 mo          |

Note. no = number; DWI = diffusion-weighted imaging; ADC = apparent diffusion coefficient; H = high signal; L = low signal; I = iso-signal; d = days; mo = months; BG = basal ganglia; PVWM = periventricular white matter; - = negative finding
Conclusion

In conclusion, restricted diffusion in the CC can develop in various diseases, with various pathogeneses. Rapid resolution of the clinical and radiologic findings suggests a good prognosis, and follow-up MRI could confirm the reversibility or irreversibility of callosal lesions. Knowledge of the MRI findings and associated conditions might be helpful in predicting patients' conditions and clinical outcomes.

References

1. Matsukawa H, Shinoda M, Fujii M, Takahashi O, Yamamoto D, Murakata A, et al. Genu of corpus callosum in diffuse axonal injury induces a worse 1-year outcome in patients with traumatic brain injury. *Acta Neurochir (Wien)* 2011; April 9. doi: 10.1007/s00701-011-1002-9

2. Kim SS, Chang KH, Kim ST, Suh DC, Cheon JE, Jeong SW, et al. Focal lesion in the splenium of the corpus callosum in epileptic patients: antiepileptic drug toxicity? *AJNR Am J Neuroradiol* 1999;20:125-129

3. Prilipko O, Delavelle J, Lazeyras F, Seeck M. Reversible cytotoxic edema in the splenium of the corpus callosum related to antiepileptic treatment: report of two cases and literature review. *Epilepsia* 2005;46:1633-1636

4. Mirsattari SM, Lee DH, Jones MW, Blume WT. Transient lesion in the splenium of the corpus callosum in an epileptic patient. *Neurology* 2003;60:1838-1841

5. Matsubara K, Kodera M, Nigami H, Yura K, Fukaya T. Reversible splenial lesion in influenza virus encephalopathy. *Pediatr Neurol* 2007;37:431-434

6. Fukuda S, Kishi K, Yasuda K, Sejima H, Yamaguchi S. Rotavirus-associated encephalopathy with a reversible splenial lesion. *Pediatr Neurol* 2009;40:131-133

7. Ogura H, Takaoka M, Kishi M, Kimoto M, Shimazu T, Yoshioka T, et al. Reversible MR findings of hemolytic uremic syndrome with mild encephalopathy. *AJNR Am J Neuroradiol* 1998;19:1144-1145

8. Kim JH, Choi JY, Koh SB, Lee Y. Reversible splenial abnormality in hypoglycemic encephalopathy. *Neuroradiology* 2007;49:217-222
9. Bourekas EC, Varakis K, Bruns D, Christoforidis GA, Baujan M, Slone HW, et al. Lesions of the corpus callosum: MR imaging and differential considerations in adults and children. *AJR Am J Roentgenol* 2002;179:251-257

10. Gruhbaum B, Salzer H, Nasel C, Lernbass I. Reversible cytotoxic oedema in the splenium of the corpus callosum related to tetracycline therapy. *Pediatr Radiol* 2010;40:1693-1695

11. Tada H, Takanashi J, Barkovich AJ, Oba H, Maeda M, Tsukahara H, et al. Clinically mild encephalitis/encephalopathy with a reversible splenial lesion. *Neurology* 2004;63:1854-1858

12. Karaarslan E, Arslan A. Diffusion weighted MR imaging in non-infarct lesions of the brain. *Eur J Radiol* 2008;65:402-416

13. Takanashi J, Maeda M, Hayashi M. Neonate showing reversible splenial lesion. *Arch Neurol* 2005;62:1481-1482; author reply 1482

14. Kubota T, Kidokoro H, Ito M, Oe H, Hattori T, Kato Y, et al. Diffusion-weighted imaging abnormalities in the corpus callosum after neonatal seizure: a case report. *Brain Dev* 2008;30:215-217

15. Takanashi J, Hirasawa K, Tada H. Reversible restricted diffusion of entire corpus callosum. *J Neurol Sci* 2006;247:101-104

16. Sigal T, Shmuel M, Mark D, Gil H, Anat A. Diffusion tensor imaging of corpus callosum integrity in multiple sclerosis: correlation with disease variables. *J Neuroimaging* 2012;22:33-37

17. Simon JH, Holtas SL, Schiffer RB, Rudick RA, Herndon RM, Kido DK, et al. Corpus callosum and subcallosal-periventricular lesions in multiple sclerosis: detection with MR. *Radiology* 1986;160:363-367

18. Maeda M, Tsukahara H, Terada H, Nakaji S, Nakamura H, Oba H, et al. Reversible splenial lesion with restricted diffusion in a wide spectrum of diseases and conditions. *J Neuroradiol* 2006;33:229-236

**Personal Information**