Analysis between clinical and MRI findings of childhood and teenagers with epilepsy after hypoxic-ischemic encephalopathy in neonates periods

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Aims and objectives

- Perinatal Hypoxic-ischemic encephalopathy (HIE) may result in chronic long-term neurologic sequelae or even death. Among them neonatal seizures accounted for 60% [1]. Currently, most research was around neonates with HIE. Research reports on epilepsy occurring in children and teenagers of HIE leading to long-term chronic cerebral injury were limited.
- The objective of the study is to assess and analyze the relationship between clinical data and MRI findings of epilepsy occurring in children and teenagers after the neonatal HIE in order to improve awareness of neurologic and neuroradiologic physicians.

Methods and materials

- 114 patients with epilepsy had a history of hypoxic-ischemic in their perinatal period, including 83 males and 31 females, age range from 1 to 24 years (13.1 ± 6.3 years). The inclusion criteria was excluding patients with metabolic diseases, congenital malformations, infections, genetic abnormalities, family history of epilepsy and undergoing therapeutic hypothermia, who with epilepsy had HIE in neonatal periods.
- All patients performed electroencephalography (EEG) and brain scan with Philips Achieva 1.5T MR Systems (Axis position: T1WI, T2WI, liquid inversion recovery sequence (Flair); sagittal: Flair; coronal: Flair, T2WI). Images were collected by using an 8-channel head coil. MRI findings of all patients in the group were analyzed by two senior neuroradiologic physicians.
- Because the study was confined to sequelae lesions of children and teenagers with HIE, it should not be applied to grading criteria the neonatal period. According to lesion involving numbers of cerebral lobes and whether associated with malacia lesions, the latter of which had low signal intensity of T1WI and Flair images in cerebral damage area by MRI, the patients were divided into three groups: lesions on MRI involving periventricular white matter or one to six lobes without malacia as mild injuries, with malacia as moderate injuries, and lesions involving equal or more than seven lobes with malacia or gray matter nuclei as severe injuries.
- Statistical analysis: the significant differences between all onset age groups and groups of cerebral injury, and between damage severity of preterm and full-term infants were compared by using $x^2$ test, respectively. The significant differences between duration of epilepsy course and severity of cerebral damage were compared by $x^2$ test, too. The relationship between MRI findings of cerebral injury and epileptiform discharges area on EEG during
intermittent and onset periods was analyzed by correlation test. P<0.05 was considered to have significance by using SPSS version 13.0.

Results

• Epilepsy onset age range of the patients was from birth to 15 yrs. According to ages they were divided into four groups, including 15 cases of <1 yrs onset age, 64 of 1-5 yrs, 23 of 6-10 yrs and 12 of >10 yrs, with 13 preterm neonates and 101 full-term neonates.

• Epilepsy onset course range was from 1 to 240 months. According to duration of course, the patients were divided into four groups, including 40 cases of #5 yrs onset course#35.1%##39 of 6-10yrs(34.2%#12 of 11-15 yrs#10.5%##23 of >15 yrs#20.2%#.

• MRI showed that hypoxic ischemic brain damage lesions involving periventricular white matter accounted for 110(96.5%), including only periventricular white matter involved in 13(11.4%)[Picture 1], cortical and subcortical area of occipital-parietal lobes involved in 95(83.3%)[Picture 2-3], frontal lobes involved in 55 (48.2%)[Picture 4], temporal lobe involved in 23(20.2%)[Picture 5], and thalamus-basal ganglia involved in 2 (1.7%) [Picture 6], respectively. Mild, moderate and severe cerebral injury groups were 36(31.6%), 60(52.6%) and 18(15.8%), respectively. Low signal intensity of T1WI and Flair as malacia indicators appeared in cerebral damage areas. Correlation test showed positive correlation between involving numbers of cerebral lobes and malacia lesions(r=0.298, p=0.001).

• Cerebral injury severity of preterm neonates was more serious than that of full-term neonates (χ²=6.112#p=0.047)[Table 1]. There was statistically significant difference among the onset age groups in mild, moderate and severe cerebral injury (χ²=13.9#p=0.031#[Table 2], while in the four groups of the epilepsy onset course no significant difference was found as to cerebral injury of mild, moderate and severe degrees (χ²=1.642,#p=0.950).

• Lesions of HIE symmetrically involved bilateral cerebral lobes in 85 (74.6%). Left or right cerebral lobes involved were dominant in 18 (15.8%) and 11 (9.6%), respectively (Picture 7-8). Correlation test showed that there was statistically positive correlation between injury degrees of left or right hemisphere and hemisphere sides of epileptiform discharges detected by EEG(r=0.340#p=0.0001).

Table 1:Comparison of cerebral injury groups between preterm and full-term neonates

<table>
<thead>
<tr>
<th>Neonate</th>
<th>Mild %</th>
<th>Moderate %</th>
<th>Severe %</th>
<th>X²</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preterm</td>
<td>2 15.4</td>
<td>6 46.2</td>
<td>5 38.5</td>
<td>6.112</td>
<td>0.047</td>
</tr>
<tr>
<td>Full term</td>
<td>34 33.7</td>
<td>54 53.2</td>
<td>13 12.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 2: Comparison of cerebral injury groups in each onset age group

<table>
<thead>
<tr>
<th>Age yrs</th>
<th>Mild %</th>
<th>Moderate%</th>
<th>Severe%</th>
<th>total</th>
<th>( X^2 )</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>2 13.3</td>
<td>9 60.0</td>
<td>4 26.7</td>
<td>15</td>
<td>13.9</td>
<td>0.031</td>
</tr>
<tr>
<td>1-5</td>
<td>17 26.6</td>
<td>38 59.4</td>
<td>9 14.1</td>
<td>64</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-10</td>
<td>11 47.8</td>
<td>7 30.4</td>
<td>5 21.7</td>
<td>23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;10</td>
<td>6 50.0</td>
<td>6 50.0</td>
<td>0 0</td>
<td>12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>36 31.6</td>
<td>60 52.6</td>
<td>18 15.8</td>
<td>114</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Images for this section:
Fig. 1: Axial Flair: hypoxic-ischemic cerebral injury only involving periventricular posterior horn white matter belonged to mild group. Flair showed high signal intensity of periventricular posterior horn white matter.
Fig. 2: Axial Flair: watershed area of bilateral occipital-parietal lobes involved without malacia belonged to mild group. Flair showed high signal intensity of bilateral occipital-parietal lobes.
Fig. 3: Axial Flair: watershed area of bilateral occipital-parietal lobes involved with malacia belonged to moderate group. Flair showed high-low mixed signal intensity of bilateral occipital-parietal lobes.
Fig. 4: Axial Flair: watershed area of bilateral frontal lobes and occipital-parietal lobes involved with malacia belonged to severe group. Flair showed high-low mixed signal intensity of bilateral frontal lobes and occipital-parietal lobes.
**Fig. 5:** Axial Flair: watershed area of bilateral temporal lobes and occipital-parietal lobes involved with malacia belonged to severe group. Flair showed high-low mixed signal intensity of bilateral temporal lobes.
**Fig. 6:** Axial flair: cortical and subcortical area involved of bilateral frontal lobes and occipital-parietal lobes with a malacia associated with thalamus-basal ganglia involved belonged to severe group. Flair showed high-low mixed signal intensity of bilateral frontal lobes and occipital-parietal lobes associated with thalamus-basal ganglia.
Fig. 7: Axial flair: watershed area of dominant right occipital-parietal lobes involved without malacia belonged to mild group. Flair showed high signal intensity of dominant right occipital lobes.
Fig. 8: Axial flair: watershed area of dominant left occipital-parietal lobes involved without malacia belonged to mild group. Flair showed high signal intensity of dominant left parietal lobes.
Conclusion

• Cerebral damage regions in sequelae of HIE were most common in periventricular posterior horn white matter regions, cortical and subcortical areas of bilateral occipital-parietal lobes, followed by the same regions of bilateral frontal lobes and temporal lobes involvement. Typical hypoxic-ischemic injury regions were watershed zone of middle-posterior cerebral artery as well as anterior-middle cerebral artery.
• Seizures onset age of children and teenagers after neonates HIE was related to cerebral injury severity on MRI. The younger the age onset group with seizures was, the more common serious cerebral injuries were. Cerebral injury of preterm neonates was more serious than that of full-term neonates in HIE sequelae. These may explain that the severity of brain injury in neonatal HIE depends on brain maturation [2]. Immature brain suffering from HIE had more serious cerebral damage.
• There was significant correlation between an epileptiform discharge hemisphere side detected by EEG and in-cerebral injury side showed on MRI. This result illustrated that the serious side of cerebral injury could be the side of epileptogenic lesion.
• For epileptogenic lesion after hypoxic-ischemic encephalopathy MRI could display location, extension and severity of cerebral injury, and may contribute to determine epileptogenic hemisphere side together with the EEG, it could affect preoperative evaluation and the choice of treatment plan.

Personal information

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References