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生发基质出血（germinal matrix hemorrhage, GMH）是早产儿最常见的颅内出血类型，常继发广泛的脑白质损伤，导致患儿远期神经发育障碍。针对发育期大脑的特殊性，探索GMH后白质损伤的动态演变过程，对揭示其病理机制具有重要意义。然而，GMH后白质损伤的病理进程复杂，常规影像学手段难以灵敏捕捉白质微观结构的进行性改变。

扩散张量成像（diffusion tensor imaging, DTI）是一种先进的磁共振成像（magnetic resonance imaging, MRI）技术，可通过定量分析各向异性分数（fractional anisotropy, FA）、平均扩散系数（mean diffusivity, MD）、轴向扩散系数（axial diffusivity, AD）和径向扩散系数（radial diffusivity, RD）来评估白质结构的完整性，在新生儿脑损伤研究中展现出独特的应用价值。

本研究以新生5日龄SD大鼠构建GMH模型，采用纵向DTI技术，对纹状体、海马、胼胝体等关键脑区的FA、MD等参数的定量分析，揭示了GMH后脑白质损伤的动态演变规律。结果显示，GMH后早期血肿邻近脑区（如纹状体、海马等）即发生白质结构完整性受损，表现为FA值降低伴MD、RD值升高，并持续至慢性期，提示GMH可诱发广泛而持续的白质结构损伤。至GMH后30天，多个脑区（如纹状体、海马、胼胝体等）均呈现明确的偏侧性白质损伤特征，表现为损伤侧FA值较对侧降低。机制研究表明，GMH后早期神经炎症反应可能驱动了白质损伤进展。本研究提示DTI可作为评估GMH远期神经预后的有效工具，并为临床早期诊断与干预策略的制订提供了试验依据。详见内文第68页。

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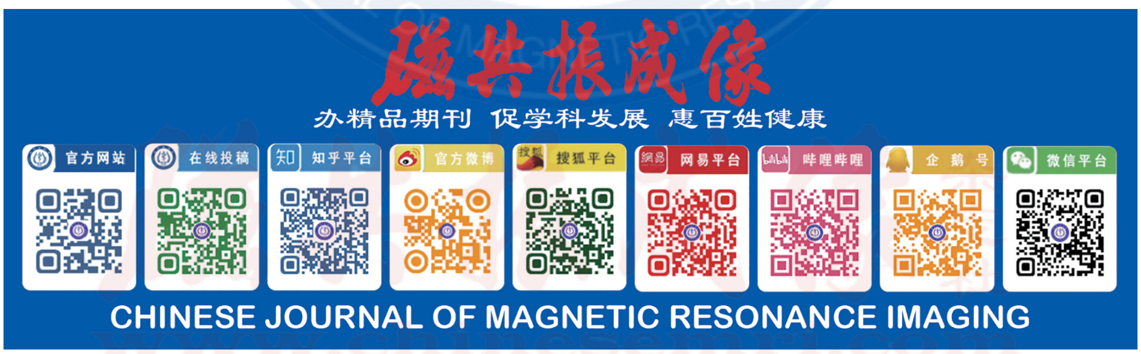
ORIGINAL ARTICLES

About the cover

Germinal matrix hemorrhage (GMH) is the most common type of intracranial hemorrhage in preterm infants and frequently leads to secondary white matter injury, resulting in long-term neurodevelopmental impairments. Given the unique characteristics of the developing brain, elucidating the dynamic evolution of white matter injury following GMH is essential for understanding its underlying pathophysiology. However, the pathological processes involved are complex, and conventional imaging techniques lack sufficient sensitivity to detect progressive microstructural changes in the white matter.

Diffusion tensor imaging (DTI) is an advanced magnetic resonance imaging (MRI) technique that enables noninvasive assessment of white matter microstructural integrity by quantifying fractional anisotropy (FA), mean diffusivity (MD), axial diffusivity (AD), and radial diffusivity (RD). DTI has demonstrated unique value in the study of neonatal brain injury.

In the present study, a GMH model was established using postnatal day 5 Sprague-Dawley rats. Longitudinal DTI was employed to quantitatively analyze FA and MD values in key brain regions, including the striatum, hippocampus, and corpus callosum, thereby characterizing the spatiotemporal progression of white matter injury after GMH. The results showed that white matter integrity in regions adjacent to the hematoma, such as the striatum and hippocampus, was compromised during the acute phase, as indicated by decreased FA values accompanied by increased MD and RD values. These alterations persisted into the chronic phase, suggesting that GMH induces widespread and sustained microstructural damage to white matter. By day 30 post-GMH, multiple brain regions, including the striatum, hippocampus, and corpus callosum, exhibited pronounced lateralization of white matter injury, with FA values significantly lower on the ipsilateral side than on the contralateral side. Mechanistic investigations indicated that early neuroinflammatory responses following GMH may drive the progression of white matter injury. These findings suggest that DTI may serve as a valuable tool for assessing long-term neurological outcomes after GMH and provide experimental evidence to support early diagnosis and the development of intervention strategies in clinical settings. Please see text page 68.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