Letter by Shao and Zhang Regarding Article, “Matrix Metalloprotease 3 Exacerbates Hemorrhagic Transformation and Worsens Functional Outcomes in Hyperglycemic Stroke”

To the Editor:

We read with interest the recent study by Hafez et al1 titled “Matrix Metalloprotease 3 Exacerbates Hemorrhagic Transformation and Worsens Functional Outcomes in Hyperglycemic Stroke.” The authors performed a study to investigate a potential role and mechanism of matrix metalloproteinase 3 (MMP3) during the acute phase in hyperglycemic stroke, which could provide treatment option for reducing cerebral bleeding and improving clinical outcomes in hyperglycemic stroke. Based on the authors’ results, we wish to communicate to the authors.

Blood–brain barrier in the brain mainly consists of vascular endothelial cells and tight junctions between them.2 MMP3 is a member of the class of zinc-dependent proteases known to degrade the extracellular matrix. Importantly, the level of MMP3 is correlated with reduction of tight junctions proteins, such as occludin, zonula occludens-1, and claudin-5, in several neurological diseases.3,4 It is known that MMP3 plays a key regulatory role in vascular permeability by degrade the tight junctions proteins. Thus, we suggest that reduction of tight junctions proteins are involved in MMP3-induced blood–brain barrier breakdown after hyperglycemic stroke. Further studies are still needed.

Furthermore, double labeling was performed to detect the expression of MMP3 in neurons, endothelial cells, pericytes, astrocytes, but the authors did not examine whether MMP3 expressed in microglials after hyperglycemic stroke. As microglials were important in the inflammatory response poststroke, and inflammation could trigger the induction and release of MMP3, therefore, we suggest further study to confirm the expression of MMP3 in microglials after hyperglycemic stroke.

Disclosures

None.

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