Letter by Takeuchi and Nawashiro Regarding Article, “Role of Iron in Brain Injury After Intraventricular Hemorrhage”

To the Editor:

We read with interest the recent study by Chen et al,1 which showed that intraventricular hemorrhage caused iron accumulation in the periventricular area (especially in glia-like cells) and bilateral enlargement of the lateral ventricles. We present a speculative hypothesis regarding the mechanism responsible for ventricular enlargement after intraventricular hemorrhage, based on the authors’ results.

Aquaporin (AQP)-4 is one of the main aquaporins in the central nervous system and acts as a water channel.2 It is located primarily in astrocytic end-feet, glial-limiting membranes, and the basolateral membrane of the ventricular ependymal cells.2 Qing et al3 reported a strong correlation between iron accumulation and AQP-4 expression after intracerebral hemorrhage in rats and also found that AQP-4 upregulation was inhibited by the iron chelator, deferoxamine.

Furthermore, several studies have examined the roles of AQP-4 in ventricular enlargement. Li et al4 reported that the majority of AQP-4-null mice demonstrated smaller ventricular sizes and reduced cerebrospinal fluid production, whereas Tourdias et al5 reported that the degree of hydrocephalus was correlated with upregulation of AQP-4.

Based on the results of these studies, we hypothesize that iron accumulation in the periventricular area after intraventricular hemorrhage may cause upregulation of AQP-4, resulting in ventricular enlargement. Further investigations into the relationships between intraventricular hemorrhage and AQP-4 are required to elucidate the mechanisms responsible for the development of hydrocephalus after intraventricular hemorrhage.

Disclosures

None.

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