

Oral presentation

High pressure hydrocephalus in neonates is associated with increased CSF concentrations of interleukin-18 and interferon gamma

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Background

High pressure hydrocephalus (HC) is associated with micro-glial activation and subsequent white matter damage. In addition to high pressure and ischemia, chronic inflammation may be pathophysiologically involved. In a rat model for HC (HTx rat, based on aqueduct stenosis), anti-inflammatory treatment reduces micro-glial scarring (Miller, 2006 CSFR). In human HC, immuno-regulatory processes involved in white matter damage are still largely undefined. Under various pathological conditions, increased CSF interleukin-18 (IL-18; expressed in micro-glial cells) and interferon gamma (IFNg; expressed in natural killer cells affecting oligodendrocytes) concentrations relate with white matter damage. We hypothesize that CSF IL-18 and IFNg concentrations are increased in neonatal high pressure HC, irrespective of underlying etiology.

Materials and methods

In 45 neonates with congenital high pressure HC (n = 30) CSF IL-18 and IFNg concentrations were determined (ELISA). HC neonates were grouped according to aetiology. Group 1: HC in spina bifida aperta (n = 20), group 2: triventricular non-hemorrhagic HC (n = 4), group 3: post

hemorrhagic HC after fetal intracerebral hemorrhage (n = 6). Low risk neonates who underwent lumbar puncture for exclusion of meningitis (and appeared negative) served as controls (n = 15).

Results

In the three groups of HC neonates, IL-18 concentrations were significantly higher than in controls [medians and range; controls: 12.5 (12.5–158) pg/ml; group 1: 80 (23–232) pg/ml; group 2: 66 (55–226) pg/ml; group 3: 223 (103–406) pg/ml (each group vs. controls, p < 0.01; group 3 vs. group 1, p < 0.01)]. Similarly, IFNg concentrations were significantly higher in CSF of the 3 HC groups [controls: 8 (8–22) pg/mL; group 1: 35 (12–139) pg/ml; group 2: 22 (15–28) pg/mL; group 3: 22 (17–56) pg/mL (each group vs. controls, p < 0.01; between the groups, NS.

Conclusion

Irrespective of underlying aetiology, neonatal high pressure HC is associated with increased CSF IL-18 and IFNg concentrations. The increased CSF concentrations reflect their pathophysiological involvement in inflammatory

white matter damage. We hypothesize that early anti-inflammatory treatment could ameliorate cerebral white matter damage in human neonatal HC.

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