Cerebrospinal Fluid Research

BioMed Central

Oral presentation

Open Access

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

Axel Heep[1], Ursula Felderhoff-Mueser[2], Thomas Schmitz[2], Arie Bos[3], Eelco Hoving[4], Carlo Schaller[6] and Deborah Sival*[3,5]

Address: [1]Dept of Neonatology, University of Bonn, Sigmund Freud Strasse 25, D-53105 Bonn, Germany, [2]Dept of Neonatology, Charité, Campus Virchow, Klinikum Universitätsmedizin Berlin, Augustenburger Platz 1, D-13353 Berlin, Germany, [3]Dept of Pediatrics, University Medical Center, University of Groningen, P.O. Box 30.001, 9700 RB Groningen, the Netherlands, [4]Dept of Neurosurgery, University Medical Center, University of Groningen, P.O. Box 30.001, 9700 RB Groningen, the Netherlands, [5]Dept of Pediatric Neurology, University Medical Center, University of Groningen, P.O. Box 30.001, 9700 RB Groningen, the Netherlands and [6]Dept of Neurosurgery, University of Bonn, Sigmund Freud Strasse 25, D-53105 Bonn, Germany

Email: Deborah Sival* - d.a.sival@bkk.umcg.nl

* Corresponding author

from 51st Annual Meeting of the Society for Research into Hydrocephalus and Spina Bifida
Heidelberg, Germany. 27–30 June 2007

Published: 20 December 2007

Cerebrospinal Fluid Research 2007, **4**(Suppl 1):S48 doi:10.1186/1743-8454-4-S1-S48

This abstract is available from: http://www.cerebrospinalfluidresearch.com/content/4/S1/S48

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.