Cerebrospinal Fluid Research

Oral presentation

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Lymphatic cerebrospinal fluid absorption is impaired in a kaolin-induced hydrocephalus model in the rat

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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):S43 doi:10.1186/1743-8454-4-S1-S43

This abstract is available from: http://www.cerebrospinalfluidresearch.com/content/4/S1/S43

Background

It has been assumed that the pathogenesis of hydrocephalus includes a cerebrospinal fluid (CSF) absorption deficit. Since a significant portion of CSF absorption occurs into extracranial lymphatic vessels located in the olfactory turbinates, the purpose of this study was to determine if CSF absorption was compromised at this location in a kaolin-induced hydrocephalus model in rats.

Materials and methods

Kaolin (n = 10) or saline as control (n = 9) was introduced into the basal cisterns of Sprague Dawley rats and the development of hydrocephalus was assessed using MRI. The degree of ventriculomegaly was calculated for each animal as the Evans ratio. Human serum albumin (^{125}I-HSA) was injected into the lateral ventricles. The enrichment of ^{125}I-HSA in the olfactory turbinates at 30 minutes post injection provided an estimate of CSF transport through the cribriform plate into the nasal lymphatic vessels.

Results

Injection of kaolin produced hydrocephalus in the majority of animals. The average Evans ratio in the kaolin group (0.48 ± 0.02) was significantly greater than that in the saline injected animals (0.35 ± 0.01; p = 0.0042). The CSF tracer enrichment in the olfactory turbinates (expressed as percent injected/gm tissue) in the kaolin rats averaged 0.99 ± 0.39 and was significantly lower than that measured in the saline controls (5.86 ± 0.32; p = 0.0019). A plot of the Evans ratio versus lymphatic transport revealed a significant negative correlation (p = 0.0002) with the largest degree of ventriculomegaly associated with the lowest levels of lymphatic CSF uptake.

Conclusion

We conclude that lymphatic CSF absorption pathways are compromised in a kaolin hydrocephalus model suggesting an important role for extracranial lymphatic vessels in this CSF disorder.