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Oral presentation

Does an osmolarity gradient cause hydrocephalus?

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Background

Contemporary theories suggest that hydrocephalus is a result of a disordered cerebrospinal fluid (CSF) circulation. However, a few papers suggest that osmotic gradients play a role in the pathogenesis. This study was undertaken to examine the role of osmolarity in hydrocephalus.

Materials and methods

Intraventricular cannulae connected to subcutaneous Alzet minipumps were inserted into the lateral ventricle in four groups of adult Sprague-Dawley rats: Group I – artificial CSF, negative control; Group II – FGF-2 (fibroblast growth factor-2), positive control; Group III – 10 KD Dextran, experimental; and Group IV – 40 KD Dextran, experimental. MRI scans were performed prior to injections and on the 12th day of infusion to measure the ventricular volumes.

Results

Group I had no hydrocephalus (n = 6). Group II (n = 4), Group III (n = 8) and Group IV (n = 8) exhibited significant ventriculomegaly compared to Group I ($p < 0.05$). There was no statistically significant difference in the size of the ventricles between groups II, III and IV. Mean osmolarity of the CSF was 307, 337 and 328 mOsm/L in Groups I, III and IV, respectively, and correlated with ventricular size ($r^2 = 0.9315$). Hypertonic saline (0.5 Molar with 910 mOsm/L) also induced significant hydrocephalus compared to Group I ($p < 0.05$; n = 3). Dextran particles were found in the nasal mucosa, cerebral cortex (especially in the walls of capillaries), corpus callosum/septum margin, and spinal cord after ventricular infusion. None of the animals with ventriculomegaly exhibited obstruction of the cerebral aqueduct, indicating that communicating hydrocephalus had been produced.

Conclusion

The presence and degree of dilatation of the lateral ventricles in these experiments were related to the osmolarity of the fluid infused. Although these results are preliminary, the findings prompt a re-thinking the role of osmotic gradients in the genesis of hydrocephalus.