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VEGF-A is elevated in CSF of pediatric patients undergoing surgery for hydrocephalus

Joseph R Madsen*[1], Joon W Shim[1], Gani Abazi[1], Laurel Fleming[1], Brian Fernholz[1,3], Susan Connors[2] and Judah Folkman[2]

Address: [1]Department of Neurosurgery, Harvard Medical School, 300 Longwood Ave., Boston, MA 02115, USA, [2]Vascular Biology Program, Children's Hospital Boston and Harvard Medical School, 300 Longwood Ave., Boston, MA 02115, USA and [3]Robert Wood Johnson Medical School, UMDNJ, Piscataway, NJ 08854, USA

Email: Joseph R Madsen* - joseph.madsen@childrens.harvard.edu

* Corresponding author

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Background

Vascular endothelial growth factor A (VEGF-A) is a member of the larger family of VEGF-related cytokines that mediates multiple functions of endothelial cells including proliferation, migration, and permeability. Current thinking on the pathogenesis of communicating hydrocephalus focuses on pulsatile vascular mechanisms, but the biochemical and biological underpinnings remain obscure. Insertion of a shunt, the most common treatment of this disorder, often fails. We wished to begin exploration of a link between hyperpulsatility and increased VEGF, which is a potent vascular permeability factor. The success of anti-VEGF treatments in very low dose for edema associated with macular degeneration and diabetic retinopathy suggest that anti-VEGF treatment in hydrocephalus could in some cases be a therapeutic alternative to shunt insertion.

Materials and methods

Forty-three cerebrospinal fluid (CSF) samples of pediatric patients undergoing intradural surgery including hydrocephalus were assayed by enzyme-linked immunosorbant assay (ELISA) to measure VEGF-A level (the lowest assay limit: 15.6 pg/ml). For pair-wise comparison, the Mann-Whitney test was used between the control (n = 23) and hydrocephalic group (n = 20).

Results

ELISA demonstrated that the CSF VEGF-A in hydrocephalic patients was elevated over controls (median, 63 and 15.6 pg/ml, respectively). Specifically, Mann-Whitney and Wilcoxon tests indicated that the two patient groups of control and hydrocephalus differ in CSF VEGF-A (pg/ml) at 95% confidence level ($p = 0.00033$). Indeed, patients with other conditions of altered CSF flow pathways, which would also be expected to interfere with the intracranial or intraspinal pulsation absorption mechanisms (as in Chiari malformations, some arachnoid cysts, and the like) also had elevations in CSF VEGF-A.

Conclusion

Forty-three pediatric cases demonstrated that CSF VEGF-A was significantly higher in the group with hydrocephalus. This suggests that VEGF-A may play an important role in the pathogenesis of hydrocephalus and inhibition of VEGF-A may be a potential therapeutic approach.