Endoscopic third ventriculostomy

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This paper is a further report on the remarkable and unique group of patients with hydrocephalus treated by Dr. Warf and his colleagues at the CURE Children’s Hospital of Uganda from 2000 to 2007. In this analysis the authors focus on 900 patients of the 2329-patient cohort who had a VP shunt, in terms of the possible role of prior endoscopic third ventriculostomy (ETV) and/or choroid plexus coagulation (CPC) in subsequent shunt failure. This is an important question in the minds of all pediatric neurosurgeons: Are there other procedures, or surgical maneuvers that can be done in patients with hydrocephalus, that would ultimately improve shunt survival, and at what cost?

The findings are extremely interesting. There was an improvement in shunt survival in those patients who had an initial successful ETV that subsequently failed, but the improvement was restricted to those who had postinfectious hydrocephalus (PIH). There was no apparent benefit of prior CPC. There was no apparent increased risk conferred by attempting these procedures as opposed to just inserting a VP shunt de novo.

There are a number of limitations to the study, which the authors are careful to discuss, including the retrospective nature of the study; the historical sequence of the surgical strategies that are being compared; possible confounding effects between patients in whom an ETV immediately failed—due to turbid CSF, or scarring beneath the floor of the third ventricle, as opposed to those in whom an ETV failed after initial success; and the diagnostic and follow-up difficulties experienced in a resource-constrained health care system.

While the authors perform the analysis starting at the point of shunt insertion, from the surgical perspective, the patients are quite heterogeneous in how they arrive at that time point. Some simply had a shunt insertion, some had an attempted ETV (with or without CPC) that was deemed to have immediately failed based on surgical video imaging features and had a shunt inserted at the same procedure, and others had an ETV (with or without CPC) that subsequently failed clinically and had a shunt placed weeks to months later. And importantly, not included in this analysis are patients who had a durable successful ETV with or without CPC. Separating the effects of the various surgical maneuvers in patients with potentially confounding presentations or clinical courses is difficult, to say the least. The authors go to great lengths to investigate possible confounding effects, but lingering uncertainty pertaining to the analysis of the various surgical interventions remains. Within these limitations, somewhat disappointingly, there is no major benefit to either ETV or CPC in reducing subsequent shunt failure, and fortunately no apparent harm.

The relevance of the results to patients in different countries and health care systems is an important issue. Perhaps the greatest difference may be in the presenting etiology of the patients here, which was predominantly postinfectious. The role of either ETV or CPC in reducing shunt failure in other health care settings where posthemorrhagic hydrocephalus or neoplasms are more common remains unknown, but the results here suggest that the effect, if any, might be small.

Nothing should detract from the remarkable achievements of the authors in addressing a compelling and common health care problem in an underdeveloped country and amassing the largest clinical series to date for this particular surgical intervention so that the rest of the world, developed and otherwise, can benefit from their careful analysis.

Disclosure

The author reports no conflict of interest.

References

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tious origin and primary management with endoscopic third ventriculostomy. J Neurosurg (1 Suppl) 102:1–15, 2005

Response

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We are grateful for Dr. Drake’s generous comments. The results of this study are reassuring in that there seems to be no adverse effect of a simultaneous or previous endoscopic procedure on subsequent shunt survival, including infection. However, we must confess to some surprise and disappointment that the study failed to demonstrate any benefit of previous ETV or CPC in regard to decreasing the risk of subsequent shunt failure. There did, indeed, appear to be a robust effect among patients with PIH, but, as discussed in the paper, we suspect this was due to selection bias, and there was no benefit among patients with other etiologies of hydrocephalus.

We agree with Dr. Drake that, even though the patient population in Uganda is different from those in more developed countries, our findings suggest that any benefit in other populations would likely be small. The main distinction of this study cohort is the large number of infants with PIH, which is a disease of poverty, and the absence of patients with posthemorrhagic hydrocephalus of prematurity, which is a disease of prosperity. We have previously pointed out the similarities in pathophysiology between these 2 etiologies of hydrocephalus.1,2 Moreover, there is nothing unique about the remaining 322 patients in the nonpostinfectious hydrocephalus cohort, which includes patients with common congenital causes of hydrocephalus (for example, myelomeningocele, aqueductal stenosis, encephalocele, Dandy-Walker complex) and those with obstructing lesions such as tumors and cysts.

Despite the acknowledged limitations of the study, we believe the results will likely prove relevant across geographic and economic boundaries. Although the primary treatment of infant hydrocephalus by ETV-CPC does not appear to confer protection from future shunt failure, it also does not increase the risk of subsequent infection or failure for those who ultimately require shunt placement and it avoids shunt dependence and its consequences altogether for the many who do not.

References


Please include this information when citing this paper: published online October 5, 2012; DOI: 10.3171/2012.6.PEDS12230.