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Hydrocephalus-induced Neuropathological Changes

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Editorial

Hydrocephalus is a physiologic disturbance of the CSF. The choroid plexus secretes CSF in a metabolically active process involving ion pumps and enzyme systems similar to those found in the kidney's distal tubule. Because water and electrolytes pass freely in and out of the brain across the ependymal surfaces of the ventricular system, CSF is thought to be produced entirely by the brain.

Regardless of intracranial pressure (ICP), CSF secretion continues at an essentially constant rate of about 20 mL/hour in adult humans as long as the choroid plexus and the brain itself are perfused. Hydrocephalus pathogenesis is influenced by genetic factors.

For the purposes of this review, we divide hydrocephalus into congenital (present at birth and frequently associated with developmental defects) and acquired (occurs after brain and ventricle development). The clinical trial of gait disturbance, mental deterioration, and urinary incontinence, along with enlargement of the cerebral ventricles on computed tomography (CT) or magnetic resonance imaging (MRI), is suggestive of normal-pressure hydrocephalus syndrome [1-5].

Causes of hydrocephalus

Hydrocephalus can affect people of all ages; however, treatment and prognosis vary greatly depending on the cause and age of onset. The patterns of hydrocephalus encountered in a single practise or institution can vary greatly depending on programmatic or referral factors. The management of the newborn with myelomeningocele, as well as the management of antenatally diagnosed congenital hydrocephalus, is covered elsewhere in this volume. Walking difficulties and postural imbalance are the most common symptoms of NPH and are the most likely to improve after a shunt. There is no "classical" NPH gait pattern: in a subset of mild NPH patients, the gait may be ataxic and wide-based, whereas in more severe cases, the gait becomes short-stepped and shuffling, with difficulty initiating walking ("magnetic phenomenon"), postural instability, and frequent falls. There is a scarcity of epidemiological data on NPH. NPH is a (very) rare cause of dementia, with estimates ranging from 0% to 5%. This variable rate may be explained by inconsistencies in NPH definitions, such as series in which NPH was diagnosed solely on clinical and neuroimaging criteria without confirmation by improvement after a shunt [1-3].

Genetics in hydrocephalus

Recurrence risk for congenital hydrocephalus, excluding X-linked hydrocephalus, is generally low. The prevalence of autosomal recessive congenital hydrocephalus ranges from 1% to 4%, indicating its rarity. Several human families have been reported to have congenital hydrocephalus. Human autosomal recessive congenital hydrocephalus loci or genes have not yet been identified, but there is at least one locus for this trait. In contrast to X-linked or recessive congenital hydrocephalus with stenosis of the aqueduct of Sylvius (HSAS), where these abnormalities are common, there was no mental retardation or pyramidal tract dysfunction in this study. Another study discovered a relative with an 8q12.2-q21 microdeletion. Molecular genetic studies have revealed that the gene encoding L1CAM at Xq28 is responsible for X-linked human congenital hydrocephalus. The mutations are spread out across the functional protein domains. The precise mechanisms by which these mutations cause a loss of L1 protein function are still being researched [4,5].

Diagnostic procedures

Neuropsychological evaluation: This test, however, underestimates subcortical and frontal mental impairment and is insufficient for detecting mild cognitive decline in NPH patients. When gait disturbance is the only visible symptom of NPH, adequate psychometric "frontal" tests such as the Trail-Making Tests B and C, the Symbol Digit Memory Test, and the Stroop Test can detect intellectual decline. The memory deficit in NPH is primarily comprised of recall issues, as opposed to mildly impaired or normal recognition [1,2].

CT (computed tomography): The frontal horn ratio, which is the maximal frontal horn ventricular width divided by the transverse inner diameter of the skull at the same level, can be used to calculate the size of hydrocephalus. A ratio of 0.32 or higher is required to consider NPH, but most NPH patients have ratios of 0.40 or higher. Transependymal CSF absorption has been linked to frontal and occipital periventricular lucencies (PVLs) in NPH. In fact, many shunt-responsive patients have no PVLs or have PVLs caused by spongiosis, decreased nerve cell density, or ischemic lesions. The proton relaxation times on MRI can be used to differentiate the causes of PVLs.

CSF extraction: Continuous external lumbar CSF drainage (ELD) of approximately 100-200 ml daily via catheter for 3- 5 days has been reported as a good predictor of outcome after shunting. Although the CSF is drained through a closed system, meningitis has been reported; subdural hematomas and radicular inflammation have also been reported.

Cisternography: Isotope cisternography appeared to be a promising technique for assessing CSF circulation at first. A disturbed CSF flow in NPH consists of a so-called "reversed pattern" with CSF stasis in the ventricles for 48 hours or longer. CT cisternography, which was introduced with the advent of CT, was also used to demonstrate disturbed CSF circulation with a similar reversed CSF flow. With a few exceptions, most clinicians now regard cisternography as an unreliable predictive test [2].

Prevention

The ideal solution to posthemorrhagic hydrocephalus is to avoid the haemorrhage in the first place. Unfortunately, neonatologists' remarkable successes in the management of pulmonary immaturity have not been replicated with IVH. In a series of randomised, controlled trials, antenatal phenobarbital and vitamin K administration did not show any benefit. In a meta-analysis of previously published trials, antenatal corticosteroids were found to reduce the incidence of IVH in premature children. When antenatal corticosteroids were compared to placebo, the combined odds ratio for the development of IVH was 0.38. Postnatal interventions have included the use of phenobarbital, indomethacin, vitamin E, ethamsylate, and pharmacologic paralysis to protect the developing infant. Indomethacin administered antenatally resulted in modest reductions in all degrees of IVH as well as severe IVH. Cognitive enhancements have yet to be demonstrated convincingly. Similarly, vitamin E and ethamsylate have been shown to reduce the incidence of IVH, but their effectiveness in reducing severe IVH or long-term disability is unknown [4,5].

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Conflict of Interest

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