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Global Ischemic In A 3-Months-Old Child With Hydrocephalus: A Case Report of Good Outcome

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ABSTRACT

Introduction: Ventriculomegaly causes compression within the cranial vault, which increases intracranial pressure and leads to severe brain damage. Usually progressive. if untreated, may be fatal.

Case Description: 3-month-old male child with increasement of head circumference for 2 months. Fever, cough, cold, vomiting, seizure was not found. MRI show extensive dilatation of ventricular system wide-open foramen of Luschka and Magendie with thinning of cerebral cortex and also global ischemic hypoxia.

Discussion: The CBF is regulated by the Monroe-Kellie doctrine which state that space of the cranial cavity is fixed in volume. Congenital hydrocephalus occurs in one in 500-1000 babies born in the United States. Lipid peroxides, formed by oxygen free radical damage to membranes, have been detected in hydrocephalic brains by detection of thiobarbituric acid reactive substances. The mechanism of periventricular axon damage includes calcium mediated activation of proteolytic calpains that damage cytoskeletal proteins, similar to the processes that follow acute traumatic or ischemic injury. The gold standard permanent treatment for hydrocephalus is CSF diversion by placement of a shunt. A shunt has three basic component parts: a ventricular catheter placed in the lateral ventricle, a valve regulating the flow of CSF out of the brain, and a distal catheter that terminates in a cavity. The most common shunt, a ventriculoperitoneal shunt (VPS), has been well accepted since its inaugural use. Surgical treatment is associated with a 50% reduced risk of death overall

Conclusion:

Keyword: Hydrocephalus; Global-Ischemic; Pediatry

1. Introduction

Hydrocephalus is a clinical and neuroradiographic diagnosis characterized by an abnormal accumulation of cerebrospinal fluid which can occur in conjunction with, or in absence of, changes to intracranial pressure. [1] Hydrocephalus can develop at any age and is estimated to affect 1 in 1000 infants during their initial birth hospitalization. [2]

Coexistence of brain vascular disease and hydrocephalus treated with ventriculoperitoneal shunt may predispose to acute ischemic stroke in case of shunt dysfunction and subsequent increased

intracranial pressure (ICP). Increased ICP, further, decreases cerebral perfusion pressure (CPP) that is already impaired in patients with vasculopathy.[3]

Since hydrocephalus is associated with tissue compression, reduced cerebral blood flow, and periventricular white matter ischemia, studies have also shown that periventricular white matter undergoes hypoxic, oxidative, and nitrosylative changes. Ventriculomegaly causes compression of brain tissue within the cranial vault, which increases intracranial pressure and leads to severe brain damage.[4] Hydrocephalus is usually progressive and, if untreated, may be fatal. [2]

2. Case Series

A case of hydrocephalus was taken, a 3-months-old male child was admitted to Haji Adam Malik with a chief complaint of the child head circumference was increasing at an abnormal rate since 2 month ago. He had been no history of fever, cough, cold, vomiting, seizures, etc. After getting all primary treatment, patient was suggested to undergo examination including CT-Scan, EEG, X-ray, and blood investigation. MRI of brain reveals extensive dilatation of ventricular system wide-open foramen of Luschka and Magendie with thinning of cerebral cortex. MRI show suggestive of communicating hydrocephalys and global ischemic hypoxia . EEG record showed no abnormalities.

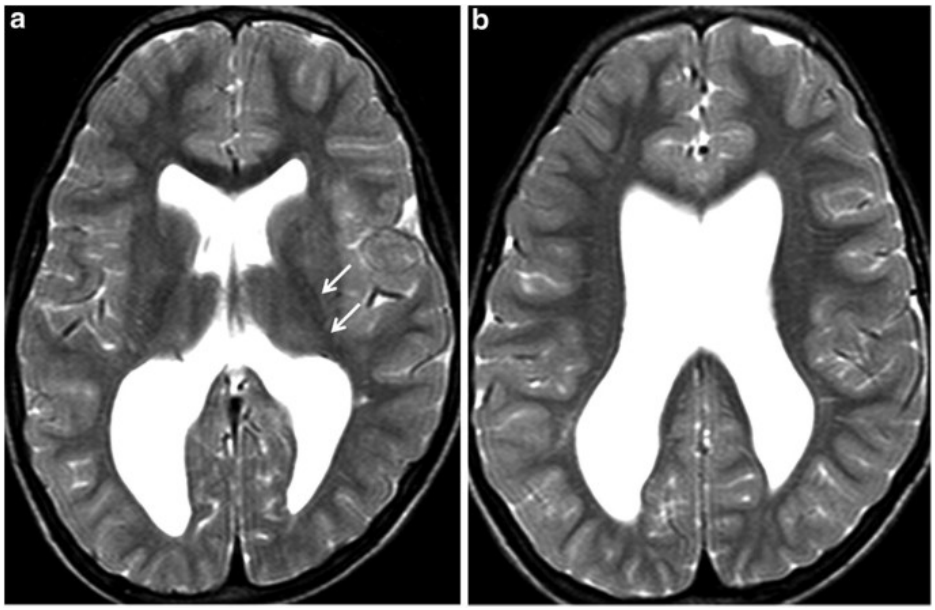

Figure 1. Hypoxia-Ischemic MRI

3. Discussion

Hydrocephalus is a brain disorder in which circulation of cerebrospinal fluid (CSF) is altered in a manner that causes expansion of fluid-filled intracranial compartments. Most CSF is produced in the ventricles by the choroid plexus although a significant amount comes from the brain extracellular fluid. CSF flows through the ventricles to the outside of the brain, where it is absorbed into the venous sinuses through arachnoid villi or into lymphatics along cranial and spinal nerves. [5]

The CBF is regulated by the Monroe-Kellie doctrine which state that space of the cranial cavity is fixed in volume, so an increase of one intracranial component (brain, CBF, or CSF) must, therefore, result in the loss of another component in equal amounts: there is an inverse correlation between CBF and CSF. In patients with ventricular shunt for hydrocephalus, a malfunctioning of the

device can lead to a rapid increase of ICP undermining the CBF and resulting in possible tissue ischemia. [3]

Hydrocephalus can be considered the single mechanical consequence of multiple processes which may affect secretion, absorption, transport, and movements of the CSF, because of either a loss of compliance, or of a secretion-absorption mismatch, or both. Mechanically it affects the brain by compressing the vascular bed, which results in parenchymal ischemic changes. The CSF-parenchyma molecular exchanges also may be affected by the CSF flow alterations and by the deleterious effect of hydrocephalus on the ependyma and the subependymal progenitor zone. [6]

Enlargement of the subarachnoid space is called external hydrocephalus; there are few obvious direct consequences of this abnormality on the brain. Enlargement of the ventricles is called internal hydrocephalus. [5] Communicating hydrocephalus occurs when bulk flow is unobstructed and results from a failure to absorb CSF via the normal drainage pathways or an accumulation of CSF due to an over-production. As the name suggests, non-communicating hydrocephalus occurs when there is an obstruction to the normal CSF flow throughout the CNS, and this can be due to a number of precipitating causes. Primary hydrocephalus may result from a multitude of genetic factors during fetal development. Genomic discovery-based assays have contributed to our understanding of the causes of primary hydrocephalus as up to 50% genetic. Secondary hydrocephalus may result from a multitude of causes including—but not limited to—infection, hemorrhage, and traumatic insult. In developed countries, post-hemorrhagic hydrocephalus (PHH) represents the most common causes of secondary hydrocephalus in pediatric patients. [1]

Congenital hydrocephalus occurs in one in 500-1000 babies born in the United States and acquired hydrocephalus may occur as the consequence of stroke, intraventricular and subarachnoid hemorrhage, traumatic brain injuries, brain tumors, craniectomy or may be idiopathic, as in the case of normal pressure hydrocephalus.[7] If one considers that the clinical hallmarks of normal-pressure hydrocephalus include the clinical triad (cognitive impairment, motor functional decline, and loss of bladder control) with symptomatic progression in the presence of chronic ventriculomegaly, or progressive cognitive and motor decline with or without urinary incontinence but in the absence of progressive ventriculomegaly. [8]

Doppler blood flow studies in hydrocephalic infants as well as PET, SPECT, MRI, and CT studies have all been used to show reductions in blood flow, particularly in white matter. Many animal experiments confirm that hydrocephalus can causes alterations in oxidative metabolism. Lipid peroxides, formed by oxygen free radical damage to membranes, have been detected in hydrocephalic brains by detection of thiobarbituric acid reactive substances. The mechanism of periventricular axon damage includes calcium mediated activation of proteolytic calpains that damage cytoskeletal proteins, similar to the processes that follow acute traumatic or ischemic injury. [5]

As the cerebral ventricles enlarge, tissue compression and axonal stretching and tearing occur, but hydrocephalus also adversely affects cerebral metabolism, cerebral blood vessels, and cerebral blood flow especially in the white matter. In particular, white matter ischemia or hypoperfusion happens simultaneously to the tissue damage, and there is reduced cerebral blood flow, which is correlated with the size of the ventricles in infants. [4]

Clinical applications of transcranial Doppler sonography in pediatric hydrocephalus are as indication and timing of drainage procedure; monitoring of effectivity of drainage procedure—internal drainage systems (shunts), ETV, external ventricular drainage, and cerebrospinal fluid derivation from subcutaneous reservoir (determination of frequency and amount of aspired cerebrospinal fluid); monitoring of function and detection of malfunction of external and internal drainage systems and ETV; assessment of the dependence of the child on drainage system (shunt-

dependence)—important for indication of external ventricular drainage or subcutaneous reservoir conversion on internal drainage system (shunt) or ETV, need for a revision of dysfunctional drainage system. [9]

The carbonic anhydrase inhibitor acetazolamide (Diamox) can reduce CSF production by ~60%.Furosemide, a diuretic that inhibits sodium reabsorption in the renal tubules, can indirectly influence fluid balances in the brain. Ouabain, a potent inhibitor of the Na+-K+- ATPase pump on cell membranes, has not been used for treatment of hydrocephalus because of its systemic toxicity. Nimodipine and magnesium sulfate provide some degree of behavioral and structural protection, likely through an effect on smooth muscle cell calcium channels and improved cerebral blood flow. [5]

Obstructions to CSF movement can be removed or bypassed. Bypass can be accomplished by creating a new pathway for CSF through brain tissue (ie, third ventriculostomy) or by redirecting CSF to other body sites using a shunt procedure (especially the peritoneal cavity and heart). The use of silicone elastomer has allowed the manufacture of flexible shunt systems that are relatively biocompatible. Survival and outcome of children with hydrocephalus improved dramatically as a consequence of shunt use. [5]

The gold standard permanent treatment for hydrocephalus is CSF diversion by placement of a shunt. A shunt has three basic component parts: a ventricular catheter placed in the lateral ventricle, a valve regulating the flow of CSF out of the brain, and a distal catheter that terminates in a cavity. The most common shunt, a ventriculoperitoneal shunt (VPS), has been well accepted since its inaugural use.[1] Surgical treatment is associated with a 50% reduced risk of death overall.[2]

Neuroendoscopy which was endoscopic third ventriculostomy (ETV) is an alternative to shunting therapy in specifically indicated patients, such as those with obstructive hydrocephalus, and involves placement of an endoscope into the ventricular system for the purposes of addressing primary pathology or changing the bulk flow of CSF. [1] The advent of endoscopes ushered in the endoscopic third ventriculostomy (ETV) technique and later endoscopic choroid plexus cauterization (CPC) as alternatives to shunting for hydrocephalus. [10]

Assessment of Doppler parameters of cerebral circulation before drainage procedure in children with hydrocephalus revealed alteration of cerebral circulation which is potentially reversible. Doppler sonography showed the improvement of cerebral circulation after the drainage procedure in pediatric hydrocephalus. [9]

4. Conclusion

Hydrocephalus in infancy can lead to significant cerebral compression, impaired cerebral perfusion, and global hypoxic–ischemic injury if not promptly recognized and managed. This case highlights that even in the presence of extensive ventriculomegaly with radiological features of global ischemia, early diagnosis and appropriate intervention may still result in a favorable neurological outcome. Neuroimaging plays a crucial role in establishing the diagnosis and guiding management. Timely cerebrospinal fluid diversion remains the cornerstone of treatment and is essential to prevent irreversible brain damage, reduce mortality, and improve long-term neurodevelopmental prognosis in pediatric hydrocephalus.

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