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Studies on Bacterial Meningitis Caused Due to Hydrocephalus

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Case Report

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ABSTRACT

Meningitis is estimated to kill 164000 children globally each year. According to population based studies, the incidence of acute bacterial meningitis in China ranges from 6.95 to 22.3 cases/100000 children<5 years of age, and acute bacterial meningitis is more common in resource poor settings. The negative consequences of bacterial meningitis in developed countries have been significantly reduced by vaccination strategies, antibiotic treatment, and good quality care facilities. Many developing countries are still facing cases of bacterial meningitis in children, which is attributable to non-implementation of vaccination programs against meningeal pathogens. Although the mortality rate associated with bacterial meningitis is lower than other major causes of childhood disease, it continues to be high, and approximately 50% of children who survive bacterial meningitis develop neurologic complications such as subdural effusions or emphysemas, cerebral abscesses, focal neurologic deficits, hydrocephalus, cerebrovascular abnormalities, altered mental status, and seizures. Accurate monitoring of pathogen specific estimates of bacterial meningitis is challenging in many countries because of the limited availability of laboratory based surveillance and the misuse of antibiotics. Late and insufficient results for cerebrospinal fluid cultures and gram stain make treatment more difficult, particularly in patients with neurologic complications. With the widespread use of medical imaging technology, hydrocephalus caused by bacterial meningitis has recently attracted increasing concern. A metaanalysis of the global and regional risks for disabling squeal from bacterial meningitis found that since 2010 the overall increase in hydrocephalus in children older than 1 month with bacterial meningitis is

7.1%, 4 and hydrocephalus is the main factor of mortality. We conducted a single center study to investigate the clinical features of and risk factors related to hydrocephalus in children with bacterial meningitis, in order to facilitate early identification and accurate evaluation.

Congenital hydrocephalus is a serious condition that can arise from multiple comprises a diverse group of conditions which result in impaired circulation and absorption of cerebrospinal fluid. The incidence is 0.4–0.8 per 1000 live borns and stillbirths. This condition is caused by mutations in the gene at Xq28 encoding for L1, a neural cell adhesion molecule. In general, recurrence risk for congenital hydrocephalus excluding X-linked hydrocephalus is low; empiric risk figures found in various studies range from <1% to 4%. We stress the importance of additional clinical investigations.

Keywords: Meningitis; Hydrocephalus; Cerebrospinal fluid; Monitoring; Neurologic complications

INTRODUCTION

This were observed in a majority of cases of hereditary hydrocephalus in humans, Escherichia coli is one of the most common causative pathogens of neonatal meningitis but the presence of E-coli in an immune competent adult, causing meningitis is rare, with an annual incidence of less than 1 case per year. Diabetes mellitus, alcoholism, cirrhosis, HIV infection, malignancies are some of the risk factors to develop E-coli meningitis [1]. A distant source is usually identified, either from the urinary or digestive tract [2]. In most of the cases, E-coli meningitis responds to ceftriaxone, gentamicin, ampicillin, penicillin G and amoxicillin, and meropenem. The mortality rate is higher in E-coli meningitis patients as compared to other pathogens [3].

Hydrocephalus is characterized by the excessive accumulation of Cerebrospinal Fluid (CSF) within brain ventricles, which results in ventricular dilatation and damage to the surrounding brain parenchyma. CSF is primarily produced at the choroid plexus, and it flows from the lateral ventricles to the third ventricle via narrow passageways and then to the fourth ventricle via the aqueduct of sylvius [4]. From the fourth ventricle, CSF passes through foramina into the subarachnoid space where it is believed to be drained via nasal lymphatic's or absorbed through the arachnoids villi The circulation of CSF within the Central Nervous System (CNS) is aided by the pulsations of the choroid plexus and by movement of motile cilia on ependymal cells [5]. Hydrocephalus can be caused by blockage of aqueducts connecting the brain ventricles, by impaired flow of CSF, by reduced CSF absorption, or even by excessive CSF production [6]. Hydrocephalus resulting from an obstruction in CSF flow along one or more of the narrow passages connecting the ventricles is classified as non-communicating hydrocephalus, whereas cases resulting from impaired absorption of CSF

in the subarachnoid space are termed communicating hydrocephalus. Abnormalities of the cerebral aqueduct or subarachnoid space [7].

Specific phobias

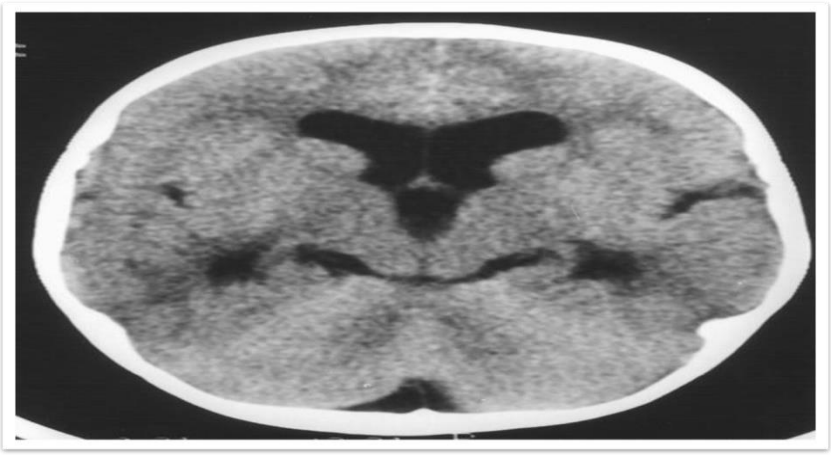
The most common type of anxiety disorder is known as specific phobia, and it encompasses any situation or stimulus that causes fear or anxiety. The National Institute of Mental Health defines a phobia as an intense fear of or aversion to specific objects or situations. People who have a phobia typically anticipate terrifying consequences from encountering the object of their fear, which can be anything from an animal to a location to a bodily fluid to a specific situation. Between 5% and 12% of the population worldwide suffers from specific phobias. Normal fears are flying, blood, water, expressway driving, and passages. People with specific phobias frequently go to great lengths to avoid encountering their phobia, which can result in trembling, shortness of breath, or rapid heartbeat. Individuals with explicit fears comprehend that their apprehension isn't corresponding to the real possible risk, yet they can in any case become overpowered by it.

CASE PRESENTATION

A 19 year old male of Asian descent with no recent history of traveling but a significant history of congenital hydrocephalus since his childhood, with sudden onset of complaints of vomiting, severe headache and altered level of consciousness [8]. The patient had symptoms of high grade fever, associated with neck stiffness and generalized seizures [9]. He denied nausea, blurring of vision, severe muscle pain and red blotchy spots over the skin, chest pain, shortness of breath, cold hand and feet, irritability with bright light exposure (Figure 1) [10].

- The boy's blood pressure was 117/66 mm Hg and his occipitofrontal circumference was 50.6 cm (>90th centile). Initial investigations showed hemoglobin concentration 81 g/l, white cell count 23.9 × 109/l (neutrophils 18.9 × 109/l), platelets 226 × 109/l, serum urea and electrolytes normal, and C reactive protein 137 mg/l., he was ventilated. Intravenous ceftazidime was given. The boy had been previously well, was fully immunized, and had been making appropriate neuro developmental progress [11]. His large head had been noted previously but was attributed to a familial tendency and was not investigated [12].

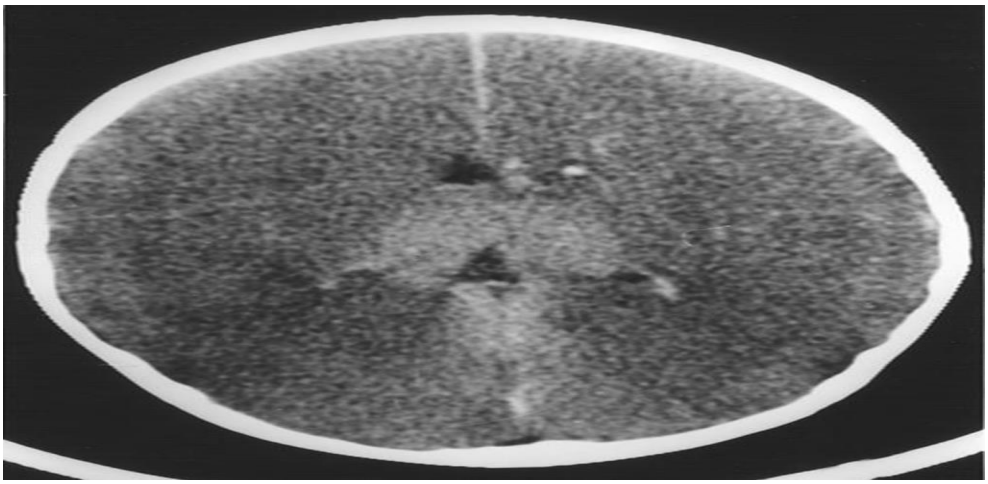
Figure 1: Computed tomogram of patient in case showing dilatation of the lateral and third ventricles with reduction of the extra axial spaces; grey white differentiation is well preserved.



RESULTS AND DISCUSSION

The boy's intracranial pressure continued to fluctuate at or above mean arterial pressure, and next morning repeat computed tomograms showed widespread cerebral necrosis (Figure 2) [13]. Intensive care support was withdrawn. A lumbar puncture was performed. The results were white cell count 45/mm3, red cell count 3/mm3, protein 4.05 g/l, and glucose 2.2 mmol/l, in keeping with obstruction to flow of cerebrospinal fluid. Latex agglutination tests on the blood culture taken on admission to hospital and on lumbar cerebrospinal fluid were positive for E. Coli postmortem examination confirmed that he had acute purulent meningitis with exudate around the base of the brain; culture of cerebrospinal fluid grew. There was no congenital cerebral abnormality [14].

Figure 2. Computed tomogram of patient in case showing dilatation of the lateral and third ventricles with reduction of the extra axial spaces; grey-white differentiation is well preserved.



Cerebrospinal fluid is formed by the choroid plexus in the lateral ventricles, from where it flows via the third and fourth ventricles to the subarachnoid space. It is reabsorbed subsequently by the arachnoids villi in the intracranial venous sinuses. The flow of cerebrospinal fluid may be blocked at the third or fourth ventricles (obstructive hydrocephalus) or at the arachnoid villi (communicating hydrocephalus). In bacterial meningitis, neutrophil migration into the subarachnoid space follows bacterial invasion. The resultant purulent exudates tends to collect in the Rolandic and Sylvanian sulci over the cerebral hemispheres and in the basal cisterns, where the subarachnoid space is deepest and where, presumably, cerebrospinal fluid flow is most sluggish. The exudates interferes with absorption of cerebrospinal fluid by the arachnoids villi and may also cause obstructive hydrocephalus by obstructing the foramina of Luschka and Magendie. Typically, the obstruction occurs towards the end of the second week of the illness, when neutrophils begin to degenerate and fibroblasts proliferate in the exudates.

Bacterial invasion into the cerebrospinal fluid from the nasopharynx in pneumococcal meningitis occurs via the choroid plexus and cerebral microvasculature and in the primate model the highest concentration of organisms early in the course of E. coli meningitis is in the lateral ventricles. Presumably the route of infection was not via the choroid plexus.

The acute inflammatory exudates in pneumococcal infection are particularly tenacious. Only one major textbook of pediatrics acknowledges that ventricular cerebrospinal fluid can be sterile at the same time as lumbar cerebrospinal fluid is purulent [15].

Ventriculostomy is a safe and relatively simple procedure. It enables cerebral perfusion pressure (mean arterial pressure minus intracranial pressure) to be calculated and intracranial pressure reduced by removal of cerebrospinal fluid. Unlike cerebral oedema, which is common in meningitis, hydrocephalus can be treated by appropriate drainage of the cerebrospinal fluid, and therefore needs to be identified.

CONCLUSION

Acute obstructive hydrocephalus is thought to be an uncommon presenting feature of bacterial meningitis, usually occuring in younger children who have had previous treatment with antibiotics. We could find no record of the incidence of this complication in published reports, nor any other case report, but since preparing this manuscript we have seen two other children with obstructive hydrocephalus as a presenting feature of bacterial meningitis. All patients with suspected meningitis and decreased level of consciousness should have urgent brain imaging to exclude obstructive hydrocephalus before lumbar puncture.

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