Pathology of Post Meningitic Hydrocephalus

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ABSTRACT
Meningitis of Bacterial (Including Tubercular) or non bacterial origin is common and lethal infection of central nervous system in children. Although with the use of modern medical facilities including antibiotics the mortality rate of meningitis have decreased yet number of patients surviving with complications such as hydrocephalus have greatly increased. In this article the etiopathogenesis of post meningitis hydrocephalus had been reviewed. effective use of appropriate antibiotics and shunt procedures have improved the outcome of post meningitis hydrocephalus of bacterial origin but same is not true with that of fungal origin which still carries high mortality and morbidity.

Keywords: hydrocephalus, meningitis.

ETHIOPATHOGENESIS
Hypothetically hydrocephalus may rise in three ways
1. From over section of CSF
2. From obstruction of CSF pathways
3. From impaired venous obstruction
Meningitis of bacterial or non bacterial origin is most common cause of acquired hydrocephalus which produces obstruction in CSF flow more commonly at subarachnoid level.Intrauterine viral infection of central nervous system (CNS) have been proposed as a cause of congenital obstructive hydrocephalus of non commuting variety.

Purulent infection remains the most common cause of communicating hydrocephalus.

Leptomeninges provide only a limited resistance to infection virulent organisms may cause death within few hours following invasion of sub arachnoids space. In the pre antibiotic era meningitis was frequently caused by intracranial extension or bacterial infections from middle ear and paranasal sinuses. However with the use of antibiotics in the early treatment of general body infection meningitis secondary to local infection is rare where as the blood borne infections from resistant organisms have increased the age of patient influences the nature of the infecting organism.

The most common bacterial infections include
1. Birth t 3 months
2. 3 months to 3 years
3. 3 years to older

In acute and sub acute stages of meningitis the subarachnoid space may be engorged with creamy pus .the leptomeninges are hyperemic and exudates may fill the basilar cisterns the cerebral fissures and cortical sulci. The exudates of streptococcal and pneumococcal infections tend to
accumulate mainly over the cerebral convexities those meningococcal tend to aggregate at the base. In meningitis secondary to haemophilus influenza the exudates is copious and diffuse.

In the chronic stage of meningitis, fibroblasts proliferate in the leptomeninges and there may be significant deposition of collagen. the process can be diffuse or patchy and in severe cases subarachnoid space may be obliterated by an adherent scar, the hydrocephalus may be noncommunicating.

Tuberculous meningitis has a predilection for the convexities of the cerebral hemispheres and for the lateral and third ventricles. Exudates are more likely to accumulate at the base. In tuberculous meningitis, exudates may obstruct the foramen of Monro and the aqueduct of Sylvius. Hydrocephalus is a frequent complication of tuberculous meningitis (TBM) and is usually of communicating type. In the chronic stage of meningitis, fibroblasts proliferate in the leptomeninges and there may be significant deposition of collagen. The process can be diffuse or patchy and in severe cases subarachnoid space may be obliterated by an adherent scar, the hydrocephalus may be noncommunicating.

Tuberculosis affects all age groups. Neutrotuberculosis constitutes almost half the cases of childhood tuberculosis and tuberculous meningitis (TBS) is the commonest type of CNS tuberculosis. In tubercular meningitis, exudation, obstruction, vascular involvement and microangiopathy are the major pathological changes which are responsible for the different clinical manifestation and complications. With improvement in treatment, the recovery rate has improved with an increase in serious sequelae as the diagnosis and treatment of primary infection are often delayed. Hydrocephalus is a late complication of TBM some degree of hydrocephalus is nearly always present in cases who have survived for more than 4-6 weeks. In a majority it is due to blockage of the basal cisterns, especially the cisternae pontis and interpedunculares, by the tuberculous exudates in the acute stage and adhesive leptomeningitis in the chronic stage. This results in a communicating hydrocephalus. In a small number of cases, the hydrocephalus is of the non communicating variety due to narrowing or occlusion of the aqueduct may be caused by circumferential compression of the brainstem by the meningeal exudate. Uncommonly intraluminal subependymal tuberculoma or a plug of ependymal exudate, may block the aqueduct. There may be asymmetrical dilatation of the lateral ventricles, due either to associated infarct or to loculation secondary to ependymal adhesion and intra ventricular septa thus in a given case more than one factor may be responsible for the hydrocephalus.

Fungal infection of the CNS is not uncommon now a day, especially with prolonged use of broad spectrum antibiotics in neonates. Hydrocephalus has been reported in up to 21% of cases of candida meningitis. Fungal infection of the CNS tends to localize in the basilar area of brain. The inflammatory response may result in hydrocephalus with involvement of the aqueduct of sylvius and obstruction of the subarachnoid space and spinal fluid channels. Hydrocephalus usually occurs as a delayed sequelae and early diagnosis and prompt aggressive medical therapy may prevent or improve the process. If obstruction of the CSF circulation is detected early and clinical signs do not indicate rapid neurological deterioration medical therapy and close observation without shunting may be tried because shunt may reinfect the lung myocardium and viscera. However, despite the appropriate timing and placement of shunts not all patients respond. Communicating hydrocephalus is an ominous complication of fungal meningitis and carries 50-70% mortality. Protozoal infection like taxoplasma is also attributed to cause of hydrocephalus. It was first identified in the retina of child with hydrocephalus later suspected as a cause of hydrocephaly and finally proves as the agent of a specific Retinopathy with meningoencephalitis. Hydrocephaly is main sign of the diseases and is often associated with the severe damage of the brain. However the electivity of taxoplasmic lesions for the aqueductal area accounts for the good intellectual prognosis in some cases, provided lesions were localized and surgery performed in due time. Hydrocephalus is one of the most common mechanisms of brain damage by causing myelin depletion axonal degeneration and neural fallout initially in cerebral white matter and later in the cortex. The general pathological findings in hydrocephalus tend to reflect both the primary effects of increased intra ventricular pressure and the secondary effects of ventricular enlargement. In any given case there are specific
considerations. The age of the patient the expansibility of the skull the level of intraventricular pressure the duration of the hydrocephalus type of preceded infection and onset and effectiveness of appropriate medical and surgical interventions are all factors that can greatly modifies the pathological finding. With the advent of effective antibiotics and shunt procedures, the prognosis now is much better than, although intellectual ability in young children may suffer considerably, notably after ventriculities. The shunt operation is safe even in preference of mild pleocytosis and increased protein in ventricular fluid provided the patient is otherwise responding to anti tubercular chemotherapy. The operation not only relieves the signs and symptoms of raised pressure but considerable regression of neurological deficit occurs in most of the cases.

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