



## Pathology of Post Meningitic Hydrocephalus

Authors

**Dr Anil K Dubey<sup>1</sup>, Dr Mudgal<sup>2</sup>**

Department of Surgery,  
Raipur Institute of Medical Sciences, Raipur

### 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 etipathogenesis 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 leptmeninges are hyperemic and exudates may fill the basilar cisterns the celebral 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 adhesive scar, the hydrocephalus may be noncommunicating.

Tuberculosis affects all age groups. Neurotuberculosis constitutes almost half the cases of childhood tuberculosis and tuberculous meningitis (TBM) 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 subarchanoid 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 toxoplasma 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 toxoplasmic 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 intra ventricular 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 ventriculitis. The shunt operation is safe even in presence 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.

## REFERENCES

1. Portnoy HD, Croissant PD . A practical method for measuring hydrodynamics of cerebrospinal fluid surg neurol 1976 5:273-277
2. Dirocco C, Ditrapani G Pettorassi VE, et al. On the pathology of experimental hydrocephalus induced by artificial increase in endoventricular CSF pulse pressure. Cluids Brain 1979; 5:81-95
3. casely Smith R Flodi M. Brocsok E. The perymphatic pathways of the brain as revealed by cervical lymphatic absorption and the passage of particles. Br|Exp pathol
4. kazman R.Hussey F . A simple constant infusion manometric test for measurement of CSF absorption. Rationale and method. Neutology 1970: 170: 330-332
5. Milhorat Ath, Hydrocephalus and the cerebrospinal fluid, Baltimore. The Williams & Wilkins company 1972;104
6. Dandy WE. Experimental hydrocephalus Ann Surg 1919;70(2):129-142
7. Milhorat TH, Hydrocephalus and the cerebrospinal fluid,Baltimore. The Williams & Wikins company 1972;104
8. De SN,Streptomycin in development of hydrocephalus in tuberculous meningitis Br.Med J 1949;2:214.