Although there are a large number of publications available in the literature recommending the benefits of a shunt procedure in tuberculous meningitis, the subject deserves a more comprehensive analysis. Hydrocephalus frequently accompanies tuberculous meningitis. Mild to severe hydrocephalus is almost always identified on computer based imaging in cases with tuberculous meningitis and occurs early in the pathological events. Despite this fact, all cases of tuberculous meningitis associated with hydrocephalus are not suitable for a shunt operation.

The exact pathogenesis of hydrocephalus in tuberculous meningitis is unclear. The hydrocephalus is probably a result of basal adhesive meningeal reaction wherein the flow of the CSF is obliterated in its course from the point of exit from the fourth ventricle to the site of its absorption in the arachnoid villi. Obliteration of the arachnoid villi themselves and various other speculations have been proposed as to the cause of the hydrocephalus in tuberculous meningitis. Like in tuberculosis elsewhere in the body, in tuberculous infection of the brain, basal arachnoiditis and arteritis suggests presence of an aggressive disease and the presence of tuberculomas suggests an aggressive body response and a relatively favorable outcome.

Basal arachnoiditis can be mild to very extensive in tuberculous meningitis. The tuberculous exudates result in gumming up of the subarachnoid spaces. Visual deficit is in general a result of affection of the optic nerve and its membranes. Optic atrophy and visual deficits, occur relatively early in the clinical course and suggests the severity of the disease. Hemispheric convexity arachnoiditis result in cortical irritation and can cause focal convulsions. Apart from adhesive basal arachnoiditis the other important pathological event that occurs is basal arteritis. Small or large vessel arteritis is the principle crippling factor in the neurological sequel of tuberculous meningitis. Worsening sensorium from drowsiness to unconsciousness, focal neurological deficits, decerebrate spasms and death are a result of arteritis and infarcts in the brainstem, hypothalamus, basal ganglia and the hemisphere.

The primary symptoms in tuberculous meningitis are fever, headache, vomiting, blindness, focal convulsions, worsening consciousness, decerebrate spasms and respiratory embarrassment. All these symptoms are related to the convexity and basal meningeal infection and cannot be attributed directly to raised intracranial pressure or hydrocephalus. Markedly bulging fontanelle and enlarging head size is seldom observed in tuberculous meningitis. The intraventricular pressure in cases of hydrocephalus with tuberculous meningitis is generally low or only marginally raised. The treatment strategy for tuberculous meningitis and hydrocephalus should be aggressive anti-tuberculous drugs and wherever necessary institution of steroids. Ventricular CSF diversionary surgery may not be necessary in most cases and can be avoided. Shunt surgery on the basis of radiological imaging diagnosis and for the sake of helping the situation of hydrocephalus in presence of other florid evidence of tuberculous meningitis, more often than not, is of no benefit. Monitoring of intraventricular pressure can be helpful in situations where there is doubt about the need of a shunt operation. Due to an immunocompromised state of patients with tuberculous meningitis, chances of shunt tube related infection are more predominant.

In adolescents and adults with tuberculous meningitis and hydrocephalus, the signs of increased intracranial pressure and drowsiness are more predominant. Such patients are usually benefited by a shunt. In some cases, when the disease process is dying down, hydrocephalus can develop and result in symptoms of headache, drowsiness and impairment of level of sensorium. Such situations also can occasionally be helped by a shunt surgery. Before a shunt is done it maybe a good idea to assess the intraventricular pressure.