Posttubercular Hydrocephalus

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Abstract

Posttubercular hydrocephalus is one of the common sequelae of tubercular meningitis. It is more severe in children and seen more frequently than in adults. It is usually of the communicating type but occasionally can be obstructive in nature. Early diagnosis is essential for a successful outcome. Delay in diagnosis is a significant cause for mortality in underdeveloped countries. The initial treatment consists of antitubercular drugs, steroids, and decongestive therapy. CSF diversion procedures are necessary when there is progressive neurological deterioration and/or radiological progression of hydrocephalus. Ventriculoperitoneal shunt remains the procedure of choice. Regular neurological assessment is helpful to diagnose shunt malfunction and institute timely treatment. Endoscopic

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third ventriculostomy is a viable option in complex and chronic posttubercular hydrocephalus with a reasonably good outcome in hands of an experienced team. The management of posttubercular hydrocephalus in multidrug-resistant tuberculosis is challenging. The role of endoscopic third ventriculostomy in acute and subacute hydrocephalus is controversial. The long-term outcome of aqueductoplasty in posttubercular hydrocephalus is still unclear. Apart from hydrocephalus, further research is necessary to determine the treatment for vasculitis complicating tuberculous meningitis and its impact on long-term prognosis.

Keywords
Endoscopic third ventriculostomy · Hydrocephalus · Meningitis · Pediatric · Tuberculous · Ventriculoperitoneal shunt

Introduction

Tuberculosis (TB) is a chronic granulomatous disease caused by acid-fast bacilli, the *Mycobacterium tuberculosis* complex. Tuberculous meningitis is a dreadful disease causing inflammation of the meninges, basal exudates, vasculitis, and hydrocephalus (Figs. 1 and 2). It usually manifests with clinical symptomatology of headaches, vomiting, fever, and altered sensorium. The development of hydrocephalus is due to inflammatory exudates occupying the basal subarachnoid spaces resulting in decreased CSF absorption or occasionally leading to blockage of the ventricular pathways. It can therefore be two types, *communicating* being more common than the *obstructive* type.

Tuberculous meningitis is an ancient disease but became treatable only recently (Hugh Cairns 1951). Ventricular estimation and taps and intrathecal streptomycin were introduced in 1951. In his treatise, Christiaan Barnard studied 259 patients of tuberculous meningitis of which 23% had an early diagnosis. He remarked that internal hydrocephalus was rare. Successful treatment hinged on early diagnosis. The best results were obtained with intrathecal and intramuscular streptomycin, oral PAS, INH, and oral cortisone. In addition to meningitis, treatment for arteritis also needs to be sufficiently emphasized. Close observation for relapse is necessary, and cases admitted in later stages were best left untreated (CN Barnard, *The Treatment of Tuberculous Meningitis, Thesis University of Cape Town, 1953*).

Tubercular meningitis with hydrocephalus (TBMH), the disease, was recognized as early as 1768 when Whytt et al. mistook the sequelae, i.e., the collection of fluid in the ventricles, for the disease itself and started a trend which, for more than half a century, kept the eyes of investigators fixed on "hydrocephalus" rather than the lesion in the meninges. It still remains whether neurosurgeons are falling into a similar trap (Tandon PN, Handbook of clinical neurology).The treatment of meningitis and hydrocephalus needs to be addressed in its entirety rather than in isolation. It is very important that the TBMH be diagnosed and treated early for a successful outcome. The initial treatment options include pharmacotherapy of TB, steroids, and decongestants for raised intracranial pressure (ICP). It should be monitored closely, and in the event of progressive neurological deterioration and radiological progression, timely CSF diversion procedure is helpful in the resolution of symptoms and determines long-term outcome. Posttubercular hydrocephalus can be complex, and sometimes treatment can be frustrating.

Pathogenesis

Central nervous system (CNS) TB is a devastating disease, especially TB meningitis (TBM) that accounts for almost 10% of cases. It has a propensity to affect multiple organs and is potentially life-threatening. The occurrence of vasculitis, arachnoiditis, direct parenchymal affection (encephalitis), and raised intracranial pressure is responsible for the poor outcome.

The most common organism causing central nervous system tuberculosis is *Mycobacterium tuberculosis*, although in immunocompromised patients other species may be involved. Following

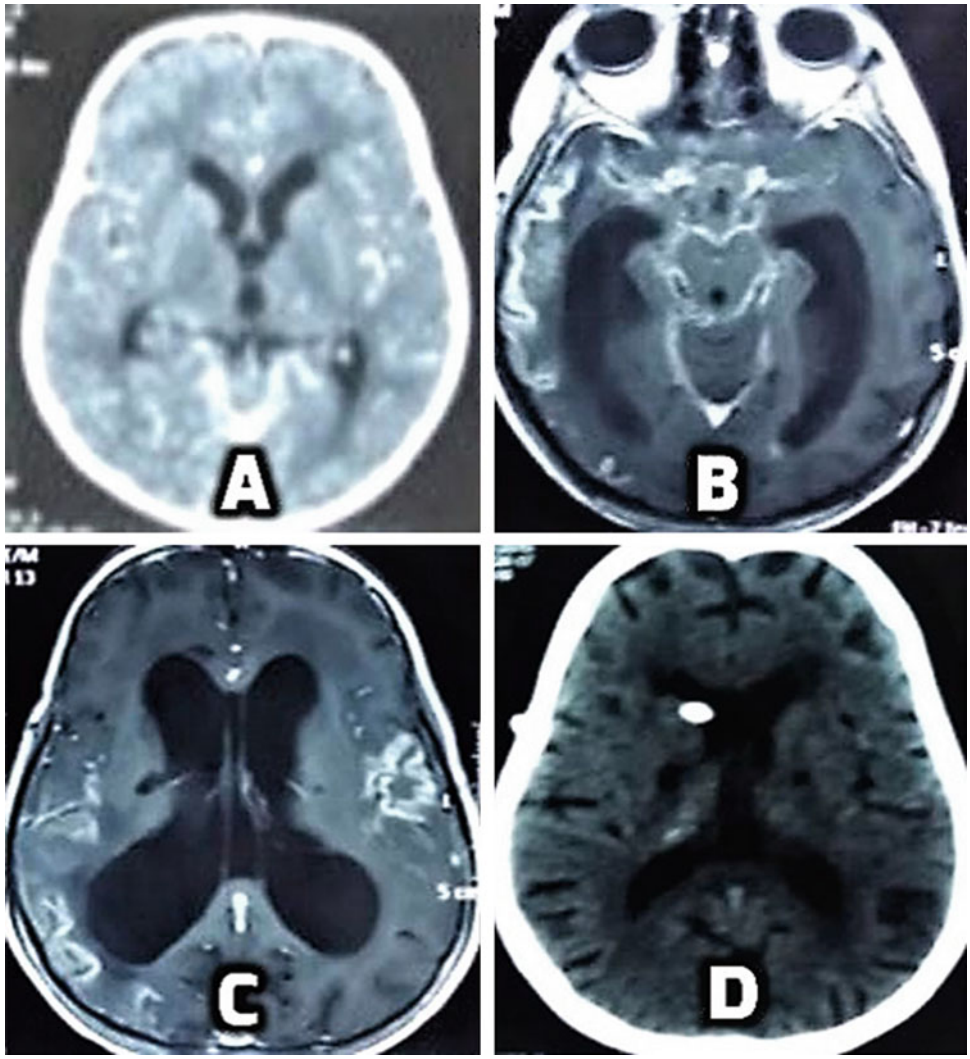

Fig. 1 Hydrocephalus developing in a patient treated successfully with a ventriculoperitoneal shunt (**a**) Axial post-contrast CT showing basal exudates without hydrocephalus (**b** and **c**) Axial post-contrast MR image showing basal exudates along with hydrocephalus (**d**) Axial plain CT image showing decompressed ventricular system following ventriculoperitoneal shunt insertion

initial pulmonary infection, known as the Rich focus, the tuberculous bacteria may enter the systemic circulation and subsequently reach the central nervous system (Rich and McCordock 1933). It establishes itself in the meninges, subpial or subependymal regions of the brain, or the spinal cord (Figs. 3 and 4). Subsequent rupture of the Rich focus into the subarachnoid space or ventricular system leads to meningitis. Alternatively, the meninges can be involved due to rupture of a tuberculoma into a vessel in the subarachnoid space. They can very rarely be involved following contiguous spread of infection from the adjacent bone. Cell-mediated immune response is usually seen. The classic feature of tuberculous CNS disease is the formation of dense, gelatinous, inflammatory exudates along the basal surface of the brain. In advanced cases, involvement of the leptomeninges over the cerebral convexities and extension into the ventricular system causing ependymitis and choroid plexitis can occur. Fulminant ventriculitis is uncommon; however,

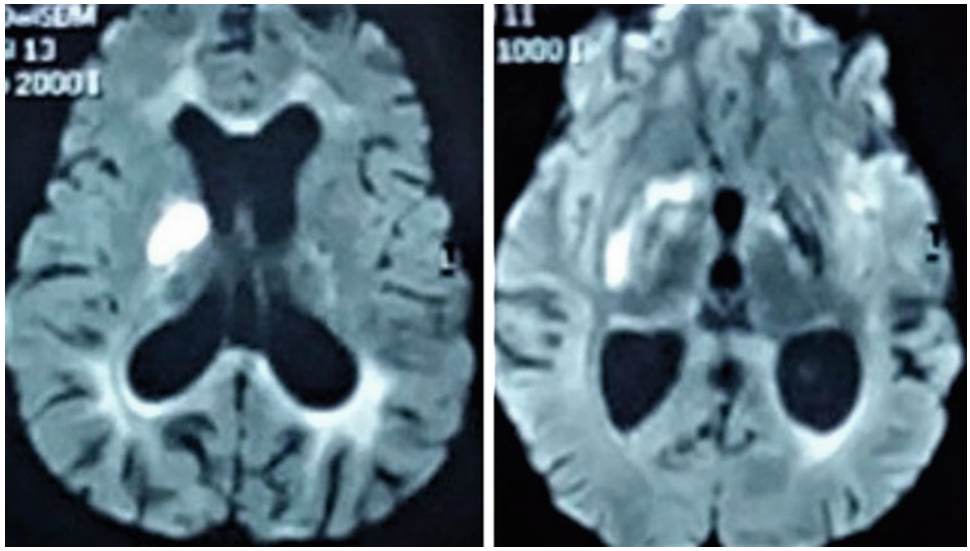

Fig. 2 Vasculitis in a patient complicating TBMH. Axial flair MR images showing restricted diffusion in the right basal ganglionic region suggestive of complicated tuberculous meningitis with infarction

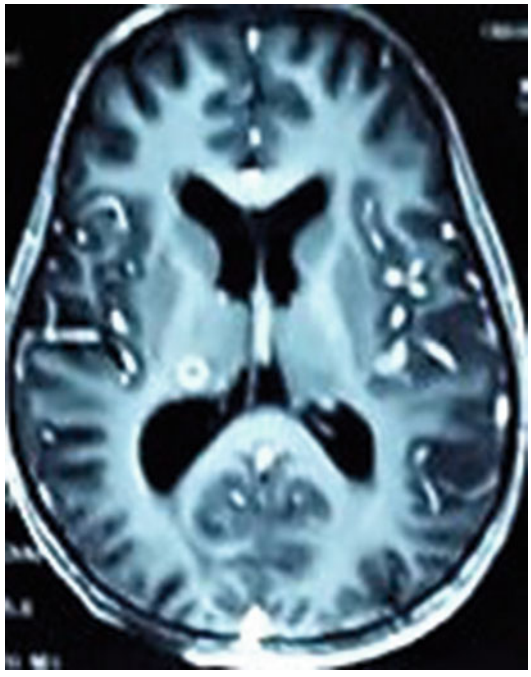

Fig. 3 Tuberculomas without significant hydrocephalus in a patient with military TB. Axial post-contrast MR images showing tuberculomas with normal-sized ventricles

adhesions within the ventricles may lead to isolated temporal horn or a trapped fourth ventricle (Figs. 5 and 6).The tuberculous affliction of the central nervous system manifests itself in a variety of clinical syndromes. Tuberculous hydrocephalus develops when the inflammatory exudates block the CSF circulation. In the earlier stages, when there is free flow of CSF from the ventricles into the subarachnoid space, it is termed "communicating" type of hydrocephalus, which is the more common variety. Any obstruction in the flow proximally results in "obstructive" or "non-communicating hydrocephalus."

The pathogenesis of TBMH can be summarized as (Dastur et al. 1995):

1. Dense, thick basal exudates blocking the arachnoid cisterns leading to communicating hydrocephalus.
2. Exudate blocking the outlet of the fourth ventricle and the aqueduct leading to obstructive hydrocephalus.
3. Inflammatory exudate extending to the ventricle leading to choroid plexitis and increased CSF production.
4. Collar of exudates surrounding the brainstem leading to brainstem edema and displacement of the brainstem eventually leading to aqueductal obstruction or secondary aqueductal stenosis.
5. Strategic tuberculoma leading to aqueductal obstruction or secondary aqueductal stenosis.
6. Tuberculoma leading to fourth ventricular obstruction.

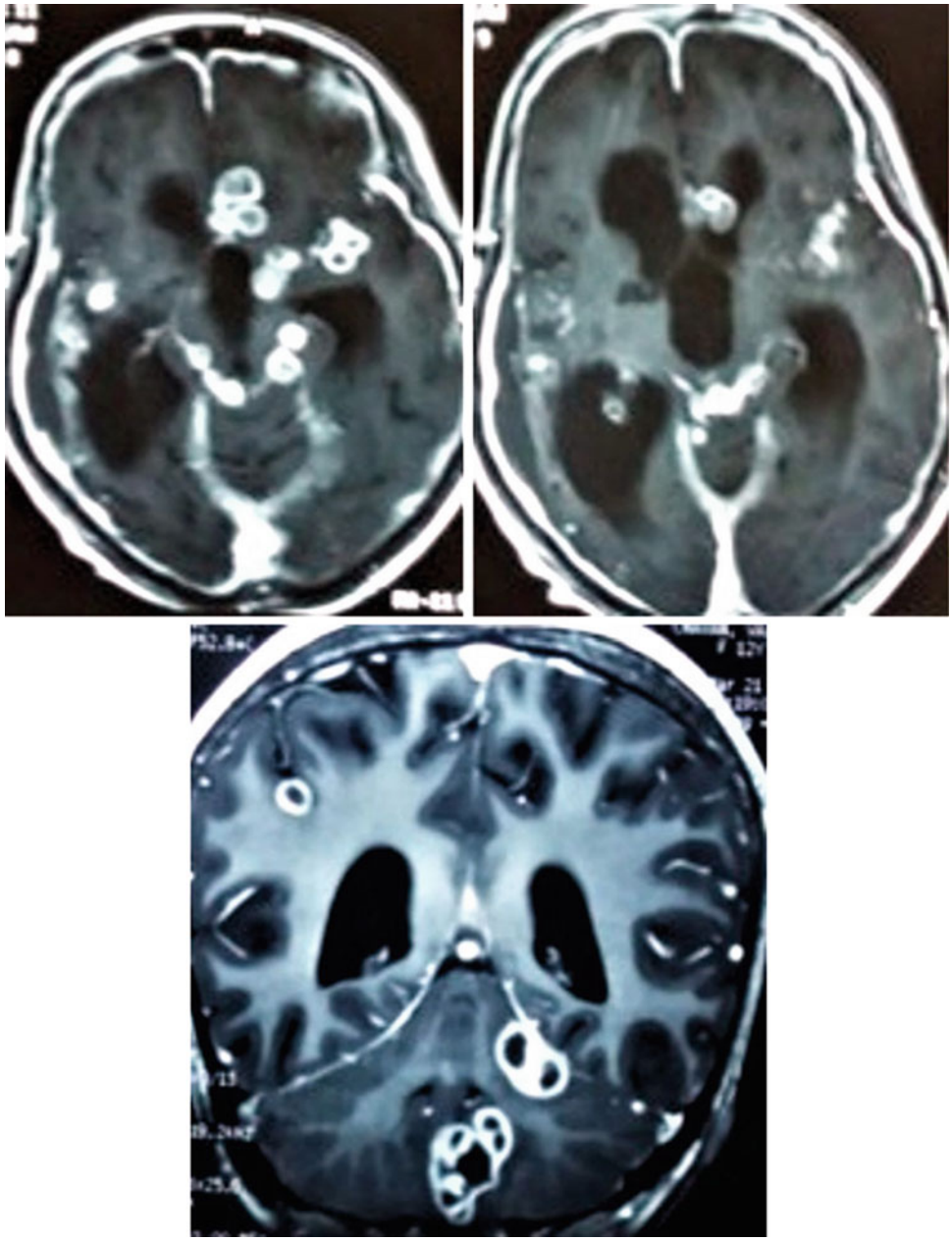

Fig. 4 Multiple tuberculomas with significant hydrocephalus. Axial and coronal post-contrast MR images showing multiple intracranial tuberculomas presenting with hydrocephalus

Incidence of Hydrocephalus

Tuberculous basal meningitis with its resultant complications forms a major medical stigma in developing countries even in the twenty-first century. In Africa, TBM complicates almost every 1 out of 100 cases of pediatric tuberculosis (Marquez et al. 2016; Murthy 2010). The incidence of hydrocephalus in tuberculosis varies between 45% and 80% in various studies. As against an incidence of 12% in adults, hydrocephalus complicates TBM in almost 71–85% of children. The occurrence of hydrocephalus on CT scan of patients with tuberculous

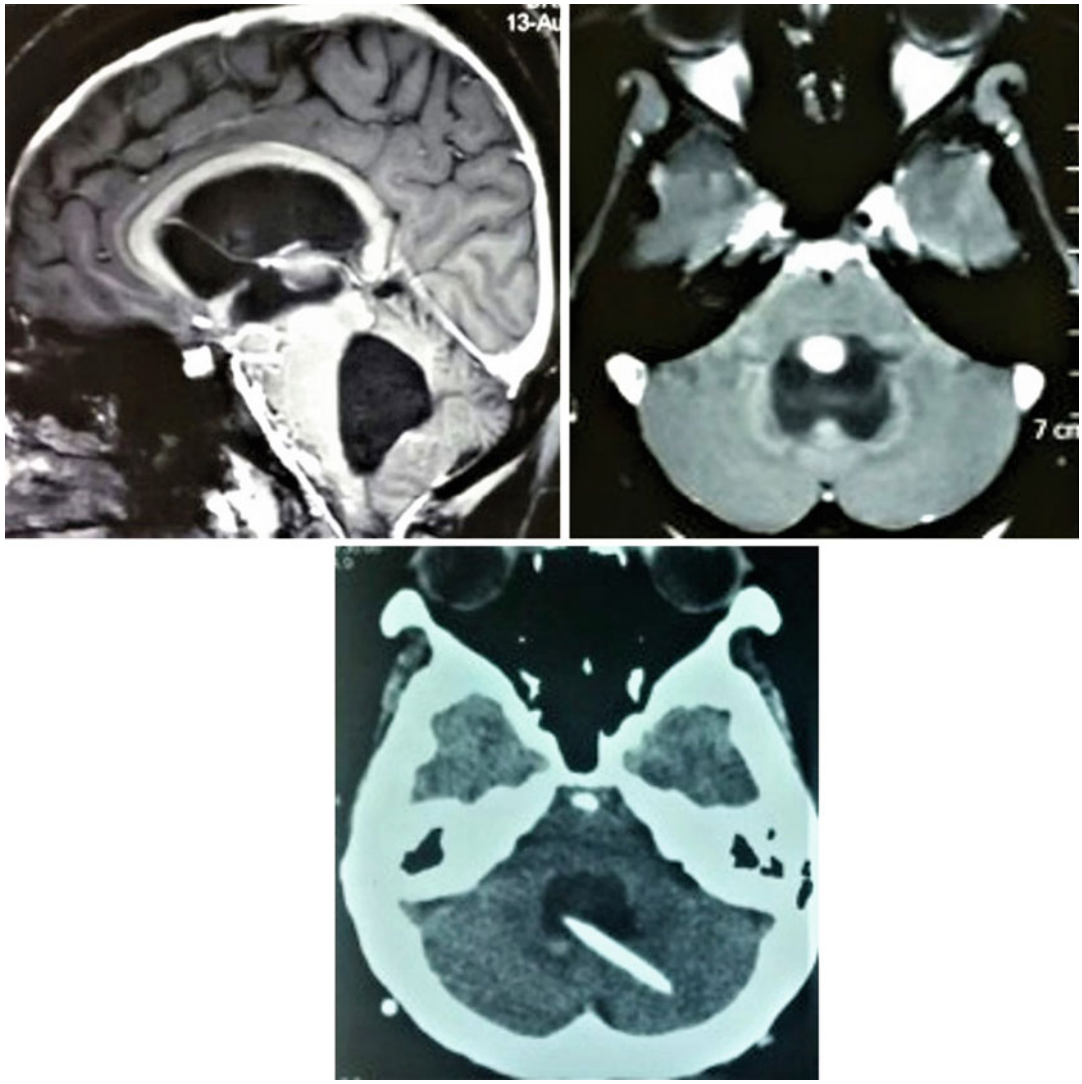

Fig. 5 Trapped **fourth ventricle presenting with headache after relief of hydrocephalus with a VP shunt 18 months earlier**. Sagittal and axial post-contrast MR images showing an isolated fourth ventricle. A fourth ventricular shunt was performed with relief of headache. Shunt tube seen in situ in the fourth ventricle on the axial plain CT image

meningitis is reported to be approximately 80.4–83%. The hydrocephalus is usually of communicating type. According to Singh et al., the incidence of communicating hydrocephalus is 45.7%, while non-communicating is 54.3%, while Sil et al. reported 78.1% communicating hydrocephalus and 21.9% non-communicating type in their study (Singh and Kumar 1996; Sil and Chatterjee 2008). Bhagwati et al., in a study of 260 patients over a 10-year period, reported 80.6% incidence of communicating hydrocephalus (Bhagwati et al. 2010).

Clinical Presentation

The clinical symptomatology of posttubercular hydrocephalus can be nonspecific. Hydrocephalus is an important consideration in a suspected

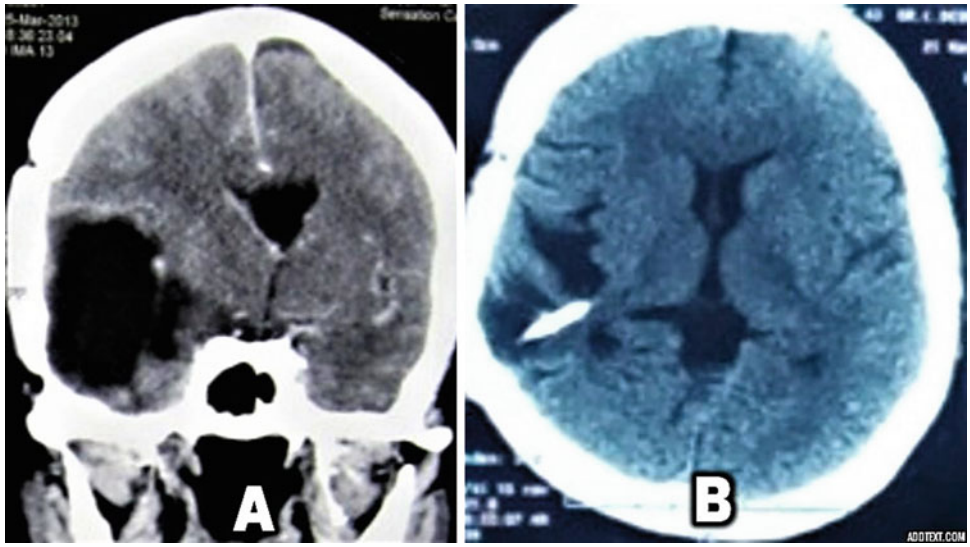

Fig. 6 Trapped temporal horn – image-guided drainage converted to shunt. (**a**) Coronal CT image showing trapped right temporal horn. The shunt tube seen in situ in the axial plain CT image (**b**) Revised shunt draining the temporal horn

Table 1 Medical research council (MRC) staging for tuberculous meningitis (Sil and Chatterjee 2008)

Stage	Description
1	Fully conscious, no paresis
2	Decreased level of consciousness, Localizing pain
3	Deeply comatose ± gross paresis

Table 2 Vellore grading of tuberculous meningitis with hydrocephalus (Palur et al. 1991)

Grade	Description
I	Headache, vomiting, fever ± neck stiffness No neurological deficit Normal sensorium
II	Normal sensorium Neurological deficit present
III	Altered sensorium but easily arousable Dense neurological deficit may or may not be present
IV	Deeply comatose Decerebrate or decorticate posturing

Table 3 Modified Vellore grading of tuberculous meningitis and hydrocephalus incorporating GCS (Mathew et al. 1998)

Grade	Description
I	GCS 15 Headache, Vomiting, fever ± neck stiffness No neurological deficit
II	GCS 15 Neurological deficit present
III	GCS 9–14 Neurological deficit may or may not be present
IV	GCS 3–8 Neurological deficit may or may not be present

GCS Glasgow Coma Scale

patient of TBM with altered sensorium. It can also be present in an alert patient with signs of raised intracranial pressure, viz., headache, blurred vision, and vomiting. Papilledema may or may not be present. The CSF picture in TBMH shows lymphocytosis, increased proteins, and decreased glucose levels. The diagnostic yield of CSF for culture of acid-fast tubercle bacilli is rather poor. Other adjunct tests available are CSF-ADA estimation and nucleic acid amplification tests. CSF pressure studies are reported, but they are not reliable indicators for future treatment (Ahuja et al. 1994). In order to better stratify these patients, various grading systems have been proposed as follows (Tables 1, 2, and 3):

Investigations

Imaging Studies

1. **CT brain with contrast**
   This is the most preliminary and yet the best investigation in many patients, especially in the pediatric population. It is easily available, quick, avoiding the need for sedation or anesthetist, and inexpensive and can be performed even in unstable patients or patients with altered sensorium. It reveals information about the presence of hydrocephalus, ischemia or infarction, periventricular ooze, parenchymal enhancement, degree of ventricular dilation and basal exudates, as well as associated tuberculomas (Figs. 1, 2, 3, and 4). The hydrocephalus is graded as mild if the third ventricle or temporal horns are dilated (>2 mm), as moderate if rounding of the frontal horns has occurred, and as severe if the periventricular lucency is present. However, CT scan cannot always differentiate between communicating and non-communicating hydrocephalus. Subacute to chronic brainstem and other infarcts may sometimes be seen on CT (Brewer et al. 2004).
2. **MRI brain with MR angiogram**
   Contrast-enhanced MR imaging shows abnormal meningeal enhancement in the basal cisterns and Sylvian fissures. The cerebral convexities show enhancement in severe and late-stage TBM. It reveals information about hydrocephalus, tuberculomas, brainstem and basal ganglia vasculitis, edema, and infarcts (Fig. 2). It can also differentiate, to some extent, between communicating and obstructive hydrocephalus.
3. **Dynamic invasive studies like air encephalography or CT Ventriculogram**
   It is the most reliable way to determine the level of obstruction. It can differentiate between communicating and non-communicating hydrocephalus. After measuring the initial pressure parameters as described below, about 10 ml of air is injected in lumbar CSF space. Air, if visible on skull X-ray in the ventricular system, confirms communicating hydrocephalus. In patients with non-communicating hydrocephalus, air is typically demonstrated only at the base of the brain. This procedure can also be clubbed with CT instead of X-ray skull. Complications involve pneumocephalus and potential risk of seizures (Figaji et al. 2005). Since it is an invasive procedure and with wider availability of MR imaging, this technique is not widely used.
4. **Cine MRI**
   It is the noninvasive analogue of pneumoencephalogram. It can also document direction of the flow of CSF. Cine MRI is used as an adjunct with endoscopic procedures for TBH (Chugh et al. 2009).

ICP Monitoring

One hour recording of lumbar CSF pressure can be performed for all patients on admission and every week thereafter for the first month of treatment (Schoeman et al. 1985). A raised intracranial pressure is diagnosed if one or more of the following criteria is satisfied:

(a) Mean baseline CSF pressure above 15 mm hg
(b) Pulse wave more than 3 mm hg
(c) Presence of pressure waves – Lundberg B-waves or plateau waves.

However, the clinical signs of raised ICP and degree of hydrocephalus estimated by imaging studies had a poor correlation with recorded CSF pressure (Schoeman et al. 1985).

Treatment

Medical Management

1. **Pharmacotherapy of TB**
   In patients with communicating hydrocephalus, medical management is the first line of treatment (Lamprecht et al. 2001). All patients receive four drugs for antituberculous therapy (IAP 2010). Most countries follow the WHO recommendation wherein the duration of treatment is 9–12 months. The drugs administered are isoniazid (15–20 mg/kg), rifampicin

(15–20 mg/kg), pyrazinamide (40 mg/kg), and ethambutol (20 mg/kg). The optimum regime and duration of therapy are controversial. It may range from 1 to 2 years (Prasad and Sahu 2010). In some cases, if the disease is in a chronic active state associated with vasculitis or tuberculomas or in cases with multidrug-resistant tuberculosis, it may be extended for 1 more year. Second-line antituberculous drugs are administered in cases of drug-resistant tuberculosis.

2. **Steroids**
   Tapering doses of dexamethasone (0.2 mg/kg/day) in three to four divided doses via parenteral route or oral prednisolone (1 mg/kg/day) can be given for a period of 4–6 weeks. It has shown to reduce the incidence of hydrocephalus and neurologic sequelae if administered early in the course of the disease. Decrease in brain edema by stabilizing the blood brain barrier improves the survival rate significantly. It also prevents crippling sequelae of vasculitis and arachnoiditis. The mortality rate has been reduced to 43% from 59%. In a few cases, the patients are likely to become steroid dependent, and hence, lower-dose steroids need to be administered for a prolonged period of time (Girgis et al. 1991; Schoeman et al. 1991).
3. **Mannitol**
   Mannitol, 0.25–1 gm/kg in divided doses, every 4–6 h is recommended only in cases with life-threatening situation. Due to reverse sink effect, its use beyond 72 h can lead to rebound intracranial hypertension, and hence its long-term use is restricted.
4. **Diuretics**
   Frusemide (1 mg/kg at 6 h interval) by oral, IM, or IV route and acetazolamide (100 mg/kg/day) are an effective diuretic. It can be given for longer periods up to a month with monitoring of electrolyte disturbances. It leads to a decrease in the CSF production but not in the ventricular size. Diuretics work effectively toward lowering the ICP when combined with ATT. The response to therapy depends on multiple factors. MRC stage III TBM and high degree of basal enhancement seen on CT scan reflecting granulomatous arachnoiditis are poor prognostic factors. All patients need close neurological observation and monitoring to detect early neurological deterioration and should be considered for surgical intervention
5. **Intrathecal hyaluronidase injection**
   Hyaluronidase is potentially capable of resolving the tuberculous basal arachnoiditis and eliminating some of the causes for TBMH. Intrathecal administration of hyaluronidase has been tried in children with TBM with some success. However, long-term promising results are not available (Bhagwati and George 1986).

Indications for Surgery

(i) Grade I and II communicating hydrocephalus after adequate trial of medical therapy
(ii) Rapid deterioration in patient's consciousness
(iii) Radiological evidence of progressive enlargement of ventricles
(iv) CSF manometry suggesting progressive increase in intracranial pressure
(v) Obstructive pattern of hydrocephalus.

Pitfalls of Medical Therapy

Continuous monitoring of patient in hospital is required for a prolonged period of time, which increases costs, and rapid deterioration may occur with poor outcome, especially if shunt is delayed.

Surgical Management

1. **Ventricular tap**
   As early as 1951, Cairns performed frontal burr hole to tap the lateral ventricle in a case of TBMH (Cairns 1951). Twist drill craniostomy at the Kocher's point is used as an emergency measure for lateral ventricular tap in patients with altered sensorium to reduce the CSF pressure and stabilize the neurological condition. It is performed in conditions wherein there is lack of operative facilities, concurrent pyogenic infection, or poor general condition of the patient. It can also yield ventricular CSF for laboratory examination. If surgical facility is

immediately not available, serial ventricular tap every 6–8 h can be done till definitive CSF diversion procedure is contemplated or an external ventricular drain or Ommaya reservoir can be placed.

2. **External ventricular drain (EVD)**
The use of EVD is controversial, and many surgeons refrain from using it in view of high risk of infection. EVD has its appropriate place in management protocol as advocated by Palur et al. and subsequently modified by Mathew et al. (Palur et al. 1991; Mathew et al. 1998). It can be used as a temporizing tool in patients with altered sensorium in stage IV as per the Vellore classification, and those who improve on EVD are assumed to potentially benefit from shunt surgery.
3. **Shunt surgery**
Bhagwati et al. were among the first few to propagate the use of ventriculoatrial (VA) shunt for TBMH (Bhagwati 1971). However, VA shunts had a higher risk of dissemination of the disease and possibility of venous thrombosis. Hence ventriculoperitoneal (VP) shunts became popular since the early 1980s. The Indian Chhabra shunt (Surgiwear, India) is a low-price device extensively used in Asia and Africa (Agrawal et al. 2005). The shunt chamber is manually compressible, and hence in suspected blockade, it can be pressed to increase the drainage and flush the proteinaceous debris. A shunt malfunction rate of 16% has been reported by Agrawal et al. which is lower than contemporary studies. Singh et al. studied 58 patients and strongly encouraged active intervention to lower raised ICP in patients who failed medical therapy (Singh and Kumar 1996).

The indications for shunt surgery are symptomatic hydrocephalus with persistent raised intracranial pressure, radiology showing ventriculomegaly with periventricular ooze, and clinical improvement following ventricular tapping.

Ventriculoperitoneal (VP) shunting for postmeningitic hydrocephalus has been aboon as well a nightmare for the neurosurgeons for its potential complications, which can be troublesome. Shunt blockade and shunt infection are two major causes of shunt malfunction. Shunt blockade can be due to increased proteins or cells in the CSF, peritoneal pseudocyst formation, and catheter perforation of the abdominal viscera. Shunt infection is usually due to breach in the aseptic barrier during surgery. A poor general condition and malnutrition due to the TBM are predisposing factors in these patients for infection. The risk of tuberculous dissemination is no longer feared. The incidence of shunt obstruction and shunt infection varies from 16% to 43% and 14% to 15.6%, respectively, in various reported case series. 18.7% to 22.8% patients require more than one shunt revisions (Singh and Kumar 1996; Agrawal et al. 2005). Antibiotic-impregnated shunts are advocated by some groups but not supported by available literature so far. Shunt malfunction should also be suspected in tuberculous spinal arachnoiditis leading to acquired Chiari I malformation and syrinx (Sil and Chatterjee 2008).

Schoeman et al. based their management on ICP monitoring and air encephalogram (Schoeman et al. 1985). Patients found to have non-communicating hydrocephalus were directly subjected to normalization of ICP in 86% of children who were managed with shunt surgery as against 63% who were managed medically. However, shunt-related complications marred this success. Interestingly, Schoeman et al. found that the ventricular size reduction was significant in shunted patients after the first month of treatment but identical to those treated with medical line of treatment at 6 months. There's also the question of how well the ventricular size reduction corresponds to resolution of raised ICP and ultimately improvement in clinical outcome (Schoeman et al. 1991).

In HIV-positive patients, a significant relationship is noted between the CD4 counts and outcomes. In a study of a group of 30 patients of TBM with hydrocephalus by Nadvi et al., 15 were HIV positive. They compared outcome between the two groups and reported poorer outcome and higher mortality in HIV-infected group after 1 month of shunt surgery and ATT (Nadvi et al. 2000). They

suggested that HIV-positive patients with TBM should undergo a trial of ventricular or lumbar CSF drainage. And only those who show an improvement following EVD should undergo shunt surgery.

4. **Management of tuberculoma and associated hydrocephalus** (Figs. 3 and 4)
   Most of the tuberculomas disappear or show substantial resolution on antituberculous treatment. Surgery is rare because of effective antituberculous chemotherapy along with antiedema measures and dexamethasone. The current indications for surgery are only in situations where their diagnosis from a brain tumor is uncertain even with the latest advances in neuroimaging, and there are features of acute rise in intracranial tension in spite of medical treatment. Bhagwati et al. reported on 31 children, of whom 5 needed surgical intervention, in which 4 of them because they were thought to harbor brain tumors and the other 1 because of significant mass effect in spite of treatment (Bhagwati and Parulekar 1986). Subtotal removal may be done with the aim to establish the diagnosis and reduction of ICP rather than attempting any radical removal.
5. **Endoscopic interventions**
   Endoscopic third ventriculostomy (ETV) is now a well-established treatment modality for obstructive hydrocephalus with a success rate of 60–85% in most series (Figaji et al. 2003; Jonathan and Rajshekhar 2005). The first use of ETV in TBMH has been reported by Figaji et al., Jonathan et al., and Husain et al. (Figaji et al. 2006; Jonathan and Rajshekhar 2005; Husain et al. 2005).

   ETV diverts the CSF to areas which were previously inaccessible and clears exudates from the areas which had impaired absorption (Jonathan and Rajshekhar 2005, Husain et al. 2005). It also decreases the transventricular pressure gradient and the demyelination of periventricular brain parenchyma, which could contribute to some symptoms of hydrocephalus (Ghosh and Chandy 1992; Rajshekhar and Chandy 1997; Yen et al. 2003). The improved CSF dynamics allow better penetration of antituberculous drugs. Alteration in CSF hemodynamics may also allow better drug delivery (Husain et al. 2005; Rajshekhar and Chandy 1997).

Figaji et al. described a simple methodology of success of ETV where entry of air in the ventricular system by lumbar puncture route has been considered a negative predictor due to communicating nature of hydrocephalus (Figaji et al. 2005). Chugh et al. concluded in 2009 that endoscopic third ventriculostomy should be considered as the first surgical option for CSF diversion in patients with TBMH. They strongly advocated the use of Cine MRI as noninvasive tool of assessment and for comparison postoperatively. They found that patients with chronic disease and longer duration of ATT administration responded well (Chugh et al. 2009).

On the contrary, patients with higher stage of illness and cisternal exudates as observed intraoperatively had a poorer outcome. Singh et al. reported a 77% success rate of ETV – 60% early and 17% delayed. They concluded the presence of thin and transparent third ventricular floor to be a favorable prognostic indicator (Singh et al. 2005).

Thus, it seems that success of ETV mainly depends on thickness of third ventricular floor and favorable cisternal anatomy around the third ventricle (Chugh et al. 2009) in this disease. However, TBMH is notorious for thick and almost fibrous exudates in the interpeduncular and the perimesencephalic cisterns. The floor of the third ventricle is frequently thick, and the underlying subarachnoid space is often obliterated by exudates (Fig. 7). This also adds to the difficulty in identifying the anatomical landmarks and increases the risk of complications. ETV certainly eliminates the need for a shunt but is technically demanding. It should be done by an experienced neurosurgeon who has good training and expertise in ETV (Figaji and Fieggen 2013). The most commonly reported complications of ETV are failure to perform the ETV due to anatomical distortions and CSF leaks (Jonathan and Rajshekhar 2005).

In summary, ETV has a better success rate in chronic hydrocephalus with obstructive

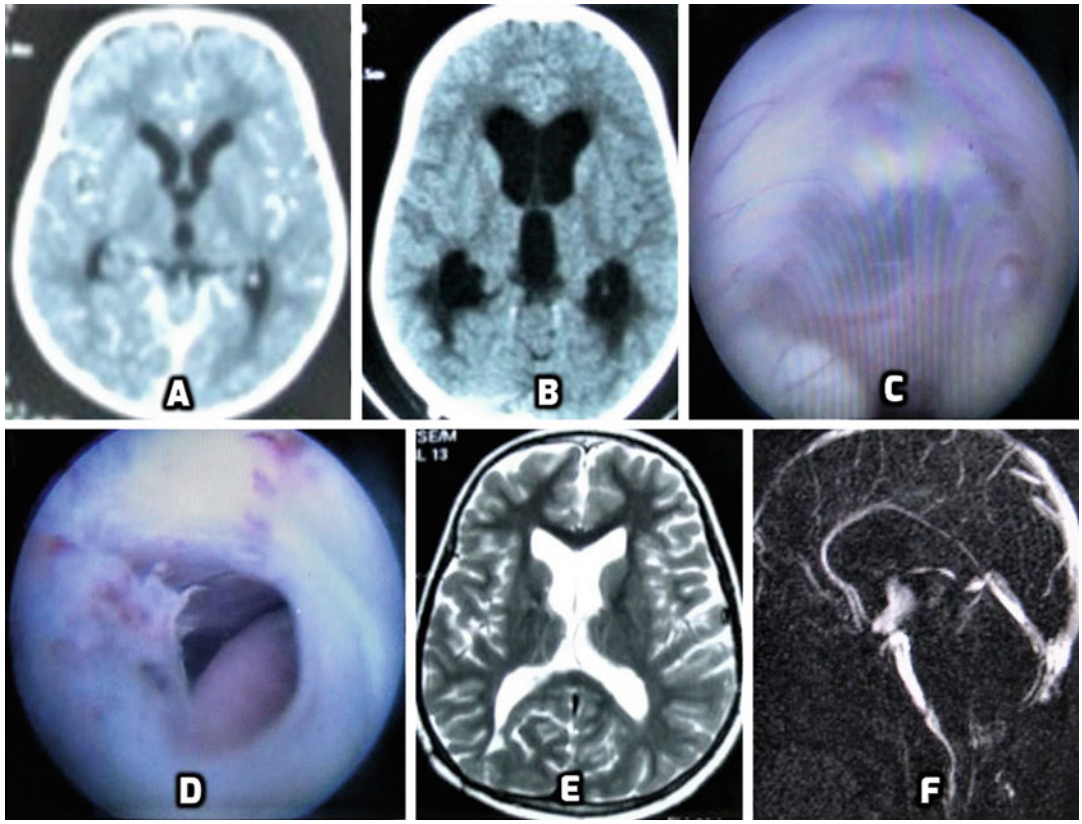

Fig. 7 **ETV in an opaque third ventricle floor** (**a**) Axial post-contrast CT image showing no hydrocephalus. (**b**) Axial plain CT image showing hydrocephalus. (**c**) Endoscopic view of the thin translucent third ventricular floor. (**d**) Endoscopic view following third ventriculostomy. The basilar artery is seen in the prepontine cistern. (**e**) Axial plain T_2-weighted MR image showing reduction in ventricular size. (**f**) MR cisternography showing functioning stoma at the third ventricular floor

ventricular pattern in our experience in spite of technical difficulties. The role of aqueductoplasty is still unclear.

Some management protocols have been drawn over time to guide management of TBH (Rajshekhar 2009) (Fig. 8).

Schoeman et al. adopted medical management as the first tier of treatment in patients with communicating hydrocephalus (Schoeman et al. 1991). They were treated with ATT combined with acetazolamide and furosemide. At 1 month of follow-up, should the patient be diagnosed as "medical failure," he or she was subjected to shunt surgery. The lumbar CSF pressure does not correlate with ICP in patients with obstructive hydrocephalus, and they are also at the risk of tonsillar herniation. These patients were directly taken up for shunt surgery.

ICP was assumed to have normalized if the following criteria were met:

1. Normal lumbar CSF pressure, occasional B-waves permitted
2. Ventricular size remaining the same or less and periventricular edema absent or markedly reduced (just visible) after first month.

Complications of Treatment

Shunt surgery probably has the highest rate of complications among all neurosurgical

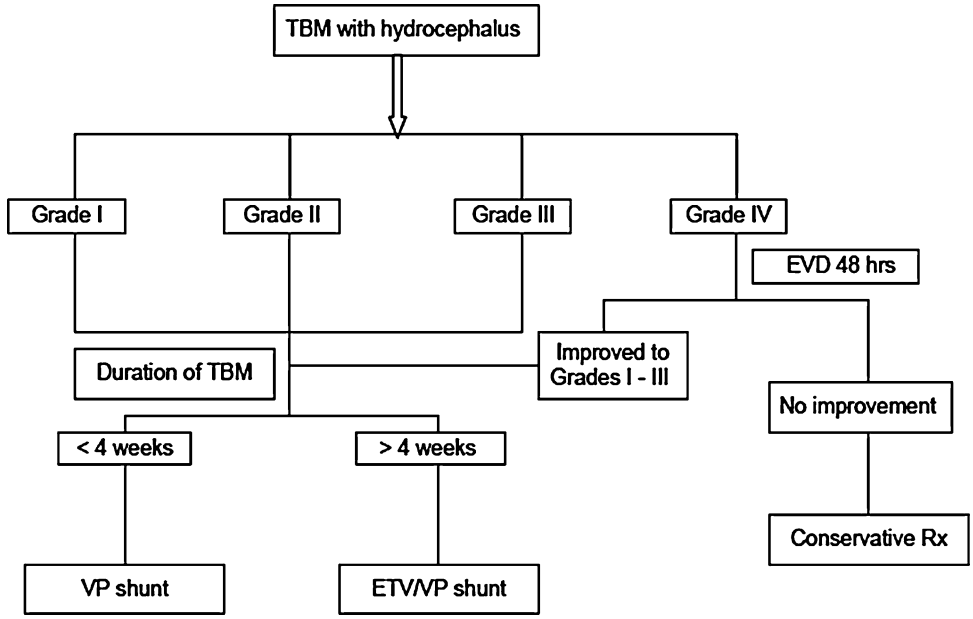

Fig. 8 Management protocol of TBM with hydrocephalus as proposed by V. Rajshekhar et al. 2009

procedures. Approximately 40% shunts fail in the first year. Infection rate, even in best hands, is up to 7% (Yadav et al. 2011, 2016). Once a shunt, always a shunt is not true anymore as ETV success is being increasingly reported in shunt malfunction, even in postinfective hydrocephalus (Cinalli et al. 1998; O'Brien et al. 2005, Deopujari et al. – personal communication) (Fig. 9). Endoscopic septostomy may also be performed for persistent unilateral hydrocephalus following shunt (Fig. 10).

The complications of shunts can be grouped as:

A. Proximal end: Block at the ventricular end is the most common cause of shunt obstruction. It is most likely due to stuck choroid plexus or collapsed ventricular wall ependyma.
B. Shunt migration, disconnection or displacement is also possible.
C. Valve malfunction can lead to underdrainage or overdrainage with formation of a subdural hematoma, slit ventricle syndrome, trapped ventricle syndrome, and double compartment syndrome.
D. Distal end: Shunt obstruction, shunt kinking, skin erosion, infection, intestinal perforation, intestinal obstruction, ascites, and abdominal pseudocysts can occur.

Poor general condition of the patient with TBMH, high CSF protein, and cellular content in ventricular CSF are responsible for frequent shunt blockages. Agrawal et al. reported shunt-related complications in 11 (30%) children, and 3 of 37 children had to undergo multiple shunt revisions (Agrawal et al. 2005). Palur et al. reported that 26 of 114 (22.8%) patients had to undergo one or more shunt revisions, 1 patient requiring more than 3 revisions (Palur et al. 1991). Sil and Chatterjee reported a shunt infection rate of 15.6% and revision rate of 43.8% in their series of 37 children who underwent shunt surgery for TBMH with hydrocephalus (Sil and Chatterjee 2008). Multiple revisions were needed in 18.7% of their patients.

Outcome Following Shunt Surgery and Endoscopic Third Ventriculostomy

Tubercular hydrocephalus has a high mortality and morbidity. Until 1991, the reported mortality

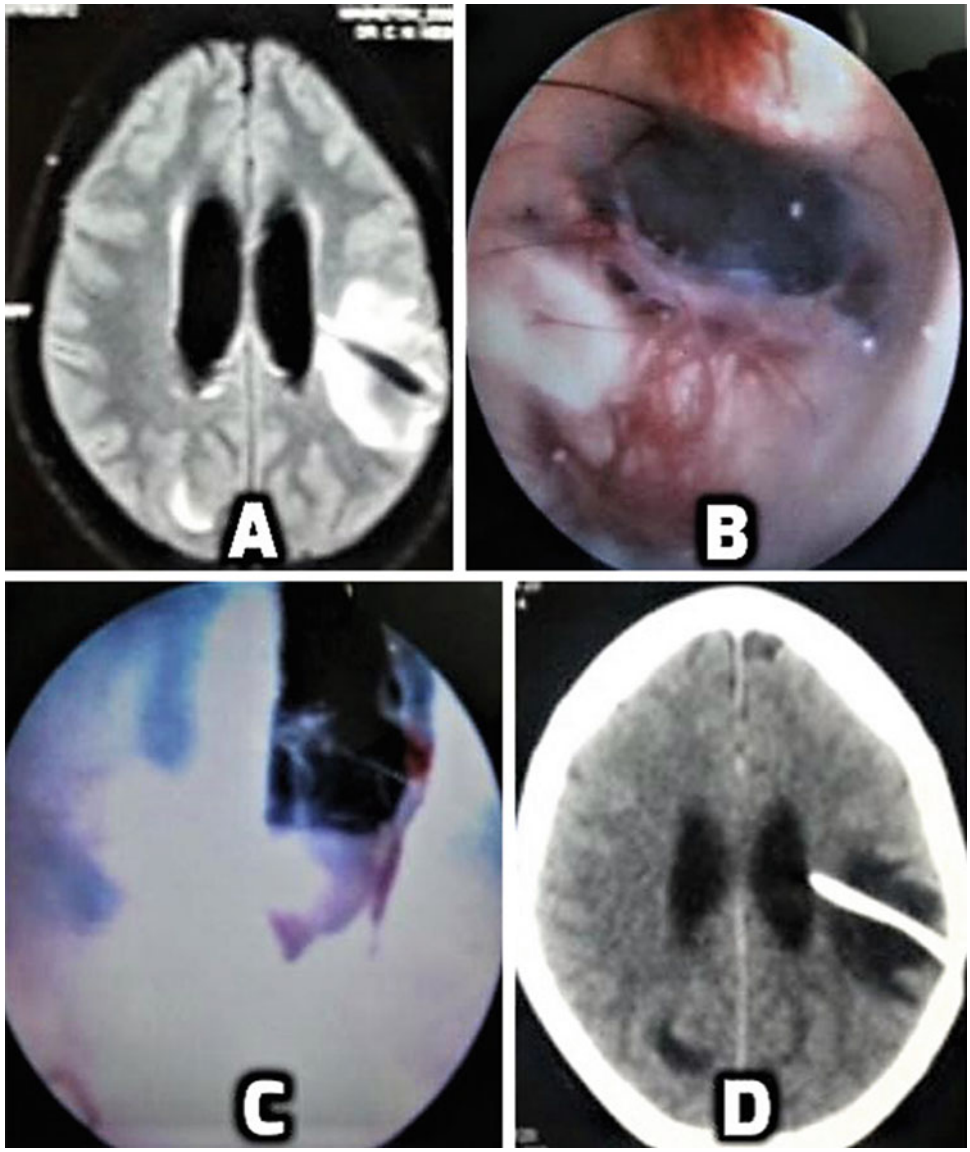

Fig. 9 **Secondary ETV in a malfunctioning shunt** (**a**) Axial flair image showing hydrocephalus with shunt tube in situ and peri-catheteral ooze. (**b**) Endoscopic view of the widened third ventricular floor. (**c**) Endoscopic view following third ventriculostomy. The basilar artery is seen in the prepontine cistern. (**d**) Axial plain CT image showing reduction of ventricular size with the shunt tube in situ

rates for patients with altered sensorium ranged from 10.5 to 57.1% and for those with normal sensorium from 0% to 12.5% (Rajshekhar 2009).

Bhagwati et al. reported mortality in three out of seven patients treated with shunt surgery (Bhagwati 1971). Sil and Chatterjee reported that all patients who had a poor outcome following shunt surgery in their series of 37 children had evidence of infarcts on their CT scans (Sil and Chatterjee 2008).

Agrawal et al. reported good outcome in 16 (43%) out of 37 patients included in their study. They had an average follow-up period of 9 months (range 6–24 months). Thirteen (35%) had moderate disability and 6 (16%) had severe disability at 3 months of follow-up. Children in

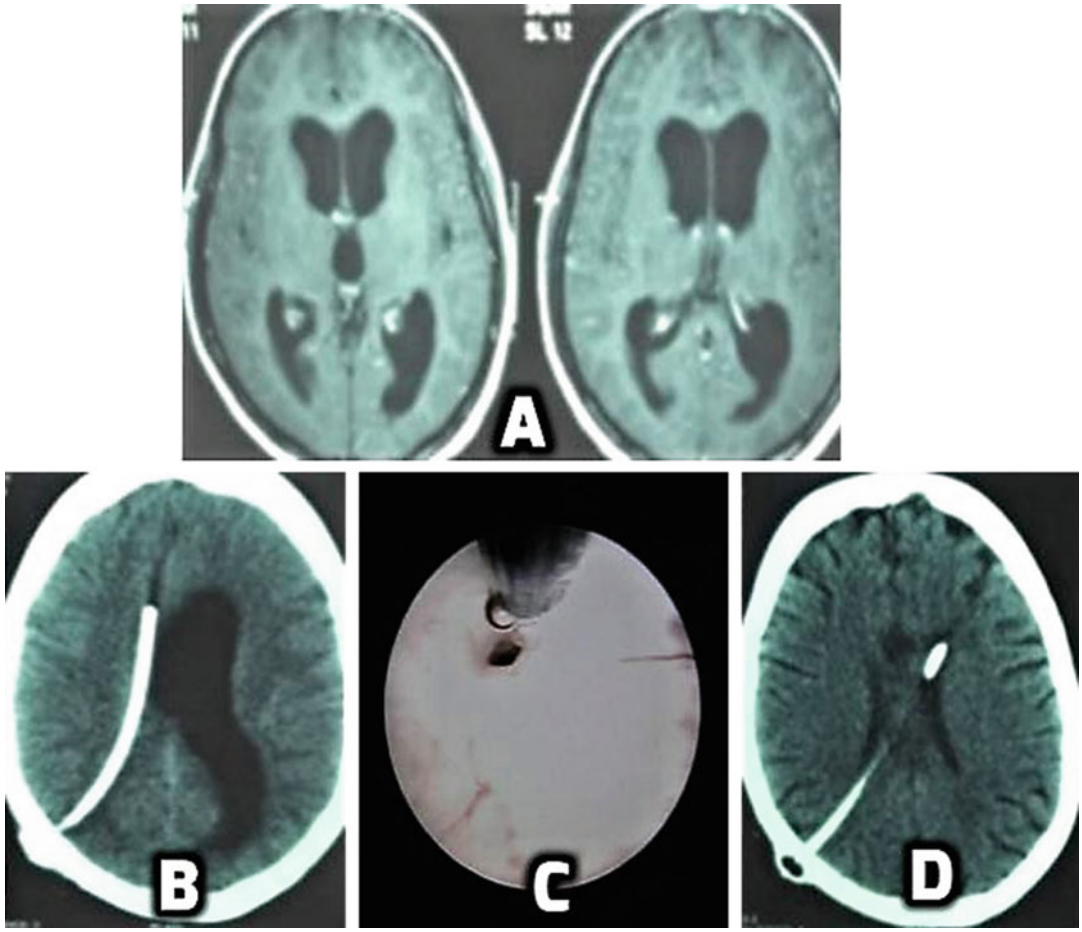

Fig. 10 Endoscopy for septostomy in non-communicating hydrocephalus developing in a shunted patient (**a**) Axial plain MR image showing hydrocephalus (**b**) Axial plain CT image showing isolated left lateral ventricle due to foraminal block (**c**) Endoscopic view showing septostomy (**d**) Axial plain CT image showing reduction in the left lateral ventricular size following septostomy. Ventricular catheter seen in situ

grade II had the best results (62%) when compared with grade III patients (good outcome in 40%). All six children with grade IV disease had a poor outcome. Two children, both having multiple infarcts, died, and the remaining four were left with severe disability (Agrawal et al. 2005).

Palur et al. reported 42.1% mortality with 55% of patients with good outcome or moderate disability. They reported that only admission grade was a statistically significant factor that affected the outcome (Palur et al. 1991). The same group has shown that patients with basal ganglia and internal capsular infarcts are also known to have poorer outcome (Rajshekhar 2009). Modified Vellore grading system is a reliable system to predict outcome following shunt surgery in TBM with hydrocephalus (Mathew et al. 1998).

Schoeman et al. reported that the mortality rate and degree of disability did not differ between patients whose ICP was normalized immediately (surgical group) and those with gradual normalization (medical group) (Schoeman et al. 1985). The final neurological outcome depended on the extent of brain damage and neurological status at presentation.

The overall success rate of ETV in TBM hydrocephalus was 73.1% (19 patients) in a series of 26 patients (Chugh et al. 2009). The correlation

with the stage of illness and presence of intracisternal exudates were statistically significant. They had reported better outcomes in patients who had received ATT for 4 weeks prior to ETV than in those operated earlier (Rajshekhar and Chandy 1997; Chugh et al. 2009). Singh et al. reported a success rate of ETV in 77% of 35 patients of TBM with hydrocephalus (Singh et al. 2005). The success rates were not related to the communicating or non-communicating nature of hydrocephalus. However, the presence of a thin and transparent floor of the third ventricle seemed to be associated with a higher success rate of 87%. Figaji et al. concluded that although ETV was technically possible in patients with TBM, only experienced surgeon should perform the surgery, as the procedure is more demanding than in other situations (Figaji et al. 2003, 2006, 2007).

Conclusion

Early diagnosis of tubercular meningitis is vital for prevention of complications and sequelae. Progressive hydrocephalus should be monitored for timing of surgical intervention. Ventriculoperitoneal (VP) shunt remains the mainstay of treatment in TBMH. ETV is indicated in properly selected cases. Surgical intervention for hydrocephalus not only reduces intracranial pressure but also has independently shown to be useful for better penetration of drugs and reduction in vascular sequelae. Multidrug-resistant tuberculosis should be looked for and aggressively treated, though role of CSF diversion in these patients is not clear. Poor clinical grade has uniformly shown to be a negative predictor, but judicious evaluation of patients for surgery may improve outcome in some cases.

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