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Original Research Article

Outcome analysis of ventriculoperitoneal shunt procedures in hydrocephalus due to tubercular meningitis and non-infective cases

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ABSTRACT

Background: Hydrocephalus is one of the most common clinical conditions affecting the central nervous system. There have been several studies which show that the outcome of VP shunt in tubercular meningitis hydrocephalus is worse. We have conducted the present study to find whether VP shunt procedure complication rates are different in these two sub class patients or if the prognosis in TBM patients is worse due to the primary disease process.
Methods: Ours is a retrospective analysis conducted in a tertiary care centre of 50 patients each of hydrocephalus due to tubercular meningitis (TBM) and non-meningitis patients. The data was collected over a period of 3 years. The diagnosis of tubercular meningitis was established by clinical history with examination, cerebrospinal Fluid (CSF) analysis and computed tomography brain (CT - Plain and contrast). All patients underwent Ventriculo Peritoneal shunt (VPS) in a standard protocol. All patients of TBM were put on 4 drugs anti Kochs treatment (AKT) for 3 months and 2 drugs AKT for 1 year. The follow up ranged between 3 years to 6 years.
Results: Data in the form of age, sex, clinical signs/symptoms and shunt related complications were noted in a prescribed format. The complications in the two groups were statistically compared for significance using p value.
Conclusions: There is no statistically significant increase in complication rate of VP shunt in TBM patients versus non infective cases. The worse prognosis of TBM patients may probably be due to the primary disease process rather than functioning of shunt.

Keywords: Communicating hydrocephalus, Obstructive hydrocephalus, Tubercular Meningitis, Ventriculoperitoneal shunt

INTRODUCTION

Hydrocephalus is one of the most common clinical conditions affecting the central nervous system.[1] Historically, the outcome of hydrocephalus used to be appalling.[2] Laurence reported in 1962, a 20% survival rate into adulthood and the surviving patients having severe sequelae.[3] Ventriculoperitoneal (VP) shunts are the gold standard in treatment of symptomatic hydrocephalus. With the advent of VP shunt surgeries, these children and adolescents can be assured of a near normal neurological development.[2] Hydrocephalus can occur following meningitis or following mechanical obstruction. There have been several studies which show that the outcome of VP shunt in tubercular meningitis hydrocephalus is worse.[4] We have conducted the present study to find whether VP shunt procedure complication rates are different in these two sub class patients or if the prognosis in TBM patients are worse due to the primary disease process.

METHODS

Ours is a retrospective analysis conducted in a tertiary care centre of 50 patients each of hydrocephalus due to tubercular meningitis (TBM) and non-meningitis patients. The data was collected over a period of 3 years from August 2010 to July 2013. The diagnosis of tubercular meningitis was established by clinical history with examination, cerebrospinal Fluid (CSF) analysis and computed tomography brain (CT - Plain and contrast). All patients of hydrocephalus underwent CSF testing to establish meningitis or otherwise. AFB staining was done to identify tubercular bacilli. If a clear diagnosis could not be established by the above tests, following tests were done to establish diagnosis-PCR, gene Xpert and CSF Adenosine deaminase (ADA).

Surgery

All patients underwent Ventriculo Peritoneal shunt (VPS) in a standard protocol. Chhabra medium pressure slit and spring valve shunt system was used. In supine position with head turn to the left, a bolster was given below the right shoulder. After due aseptic precautions, a burr is made in the right parietal region 3 cm above and behind the pinna. Ventriculostomy and insertion of the cranial end of the cathether system in the frontal horn of right lateral ventricle is performed. Shunt is assembled after testing the patency. The subcutaneous tunneling is made and the catheter is passed. The abdomen is opened and the distal end of the catheter is inserted in the peritoneal cavity. Wound is closed in layers.

Inclusion criteria

a. GCS >14/15
b. No active tuberculous foci in the body except TBM
c. Age between 1 year to 18 years
d. CSF protein below 200 mg/dL
e. Medically fit

Exclusion criteria

a. Neonate and Infants
b. Tuberculosis involving other systems eg miliary Kochs
c. GCS score below 14/15
d. Severe morbidity and medically unfit patients
e. MDR and XDR Kochs

All patients of TBM were put on 4 drug Anti Kochs treatment (AKT) for 3 months and 2 drugs AKT for 1 year. The follow up ranged between 3 years to 6 years. Follow up included clinical examination and CT brain (P & C). AKT was discontinued only after CSF tests and CT imaging. All patients were called for monthly follow up in the first year and thereafter at 3 monthly intervals. Data in the form of age, sex, clinical signs/symptoms and shunt related complications were noted in a prescribed format. The complications in the two groups were statistically compared for significance using p value.

RESULTS

Data for TBM and non TBM patients were collected and analyzed for demographic distribution (Table 1).

Table 1: Demographic distribution of TBM and non TBM cases.

Age group (years)	Males (TBM)	Females (TBM)	Males (non TBM)	Females (non TBM)
1-4	9	12	10	10
5-8	8	3	5	8
9-12	5	0	3	3
13-16	3	6	3	3
17-18	3	1	3	2
Total	**28**	**22**	**24**	**26**

Table 2: Etiology of non tubercular meningitis hydrocephalus.

Causative factor of hydrocephalus	Number of patients (n=50)	Percentage
Aqueductal stenosis	09	18%
Meningomyelocele	5	10%
Occipital encephalocoele	1	2%
Dandy Walker syndrome	5	10%
Colloid cyst and intraventricular tumors	2	4%
Post fossa tumors	19	38%
Normal pressure hydrocephalus	5	10%
Post Intraventricular hemorrhage	1	2%
Post traumatic	3	6%
Total	**50**	**100%**

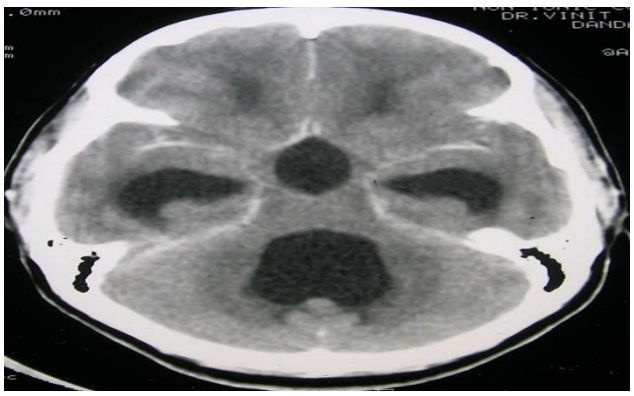

Figure 1: A post contrast axial image of a case of Tubercular meningitis showing dilated temporal horn, third ventricle and fourth ventricle suggestive of communicating hydrocephalus. There is also evidence of basal exudates which are enhancing on contrast administration.

Our study comprised of 50 patients each of tubercular meningitis and 50 patients of non-infective cases. The distribution of non-infective cases are as given in Table 2.

Table 3: Clinical presentation of patients (symptoms).

Symptoms	Number of Patients in tubercular meningitis (n = 50)	Number of Patients in non-tubercular meningitis (n = 50)
Asymptomatic	5 (10%)	3 (6%)
Vomiting	17 (34%)	9 (18%)
Altered sensorium	13 (26%)	2 (4%)
Headache	21 (42%)	34 (68%)
Loss of appetite	31 (62%)	12 (24%)
Photophobia	11 (22%)	6 (12%)
Low grade fever	19 (38%)	2 (4%)
Convulsions	7 (14%)	9 (18%)
Diminution of vision	7 (14%)	5 (10%)

Patients with TBM and non-infective hydrocephalus presented with varied symptoms and signs. The symptomatic distribution of patients is as given in Table 3 and Table 4.

Table 4: Distribution of clinical signs.

Signs	Number of Patients in tubercular meningitis (n = 50)	Number of Patients in non-tubercular meningitis (n = 50)
Increased head circumference	4 (8%)	26 (52%)
Sutural diastasis	2 (4%)	24 (48%)
Sunset sign	3 (6%)	11 (22%)
Altered sensorium	32 (64%)	2 (4%)
Sixth nerve paresis	3 (6%)	4(8%)
Ataxia	3 (6%)	13 (26%)
Hemiparesis	7 (14%)	1 (2%)
Paraparesis	3 (6%)	0 (0%)
Meningomyelocele	0 (0%)	5 (10%)
Occipital encephalocoele	0 (0%)	1 (2%)
Tense anterior fontanelle	5 (10%)	7 (14%)
Fundus examination		
Papilloedema	7 (14%)	25 (50%)
Optic atrophy	2 (4%)	2 (4%)
Diminution of vision	2(4%)	2 (4%)

Table 5: Complications related to VP shunt surgery.

Complications	Number of patients in tubercular meningitis (n = 50)	Number of patients in non-tubercular meningitis (n = 50)
Shunt infection	3 (6%)	2 (4%)
Shunt migration	4 (8%)	2 (4%)
Shunt malfunction	7 (14%)	3 (6%)
Abdominal complications	2 (4%)	1 (2%)
Shunt Revision	15 (30%)	11 (22%)
Shunt extraction	2 (4%)	1 (2%)
Total	**33 (66%)**	**20 (40%)**

Shunt surgery is known for its myriad complications. We have collected and analyzed the complications in the two sub groups separately (Table 5).

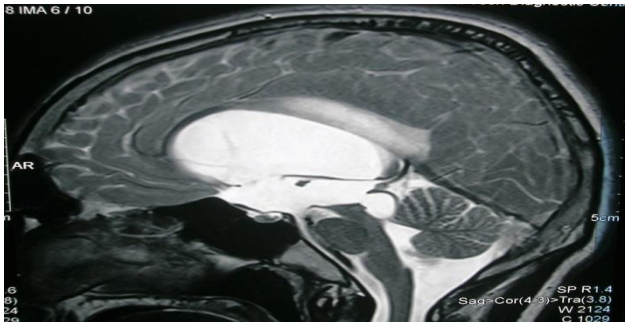

Figure 2: T2 weighted sagittal image of hydrocephalus due to aqueductal stenosis. The fourth ventricle is almost normal in size. There is evidence of periventricular ooze indicative of raised pressure.

Figure 3: Plain CT brain axial image showing ventricular end of VP shunt in situ. The ventricle has collapsed following shunt insertion.

DISCUSSION

Hydrocephalus is a common disorder of central nervous system in children.[1,2] Hydrocephalus has been traditionally classified into obstructive and communicating variety. Communicating variety is largely

due to TBM and the reasons of obstructive hydrocephalus are acqueductal stenosis, tumors etc. The commonest meningitis in our country is post tubercular. While increase in head size is only sign of hydrocephalus among most of the children below two years, older children and adolescents may present with the classical triad of headache, vomiting and papilloedema.[1] The development of shunt surgeries has remarkably changed the outcome in these patients with better prospects of leading a normal life. However, complications like infection and obstruction warranting shunt revisions continue to adversely affect the outcome.

It has been reported that complications of shunt surgery are higher in patients with TBM than in patients with other conditions. The reasons for this are the poor general condition of these patients and also the presence of higher protein and cellular content in the CSF leading to more frequent shunt obstruction.[6] Agarwal et al. reported shunt related complications in 11 (30%) children and three of 37 children had to undergo multiple shunt revisions.[7] Palur et al reported that 26 of 114 (22.8%) patients had to undergo one or more shunt revisions, one patient requiring more than three revisions.[8] 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 TBM with hydrocephalus. Multiple revisions were done in 18.7% of patients.[6] There has been no objective evaluation on whether the high complication rate of VP shunt and associated poor prognosis of TBM patients are due to primary disease process or related to VP shunt surgery. Hence, we planned this study and tried to standardize the two sub groups by excluding morbid patients, extracranial tuberculosis and those with fulminant infection(high protein content).

Tubercular meningitis hydrocephalus

Hydrocephalus is one of the most common complications of TBM.[9] It is more frequent and severe in children than in adults and also occurs at an earlier stage in the disease process. In general, however, TBM is more common in children than in adults, especially in the first 5 years of life. In fact, children aged 0-5 years are affected more commonly with TBM than any other age group. TBM is uncommon, however, in children younger than 6 months and almost unheard of in infants younger than 3 months because the causative pathological sequence takes at least 3 months to develop.[10] A considerable proportion of patients with TBM especially those in the poorer grades have been shown to have infarcts of the basal ganglia, thalamus and brain stem.[5] In our study, we have included only good grade TBM patients so that we can compare the procedural complications.

Not all patients who undergo shunt surgery for TBM with hydrocephalus show a significant improvement in their sensorium or symptoms. It is, therefore, evident that the cause of the altered sensorium and other symptoms in these patients are not always or completely attributable to the hydrocephalus.[5] The encephalitis that is caused by the inflammatory processes enveloping the cerebral hemispheres, the ischemia caused by the arteritis especially of the small perforators at the base of the brain probably contribute in equal if not more than equal measure to the altered sensorium.[5,11]

The outcome of TBM with hydrocephalus is finally dependent on the response of the disease to ATT. It is understandable that in patients with drug resistant TB either multi-drug resistant TB (MDR-TB) or extensively drug resistant TB (XDR-TB), the outcome is likely to be poor.[12] There are however, no studies which have reported on the outcome following surgery for drug resistant TBM.[5,6] In our study we have excluded drug resistant tuberculosis to standardize the two sub group of populations.

A shunt should be promptly offered in case of failure of medical management. Prolonging medical therapy in patients in good grades could be harmful and may lead to irreversible brain damage.[5] In patients with evidence of obstructive hydrocephalus and neurological deterioration who are undergoing treatment for TBM, placement of a ventricular drain or ventriculoperitoneal or ventriculoatrial shunt should not be delayed. Prompt shunting improves outcome, particularly in patients presenting with minimal neurological deficit.[5]

Among persons younger than 20 years, TB infection rates are similar for both sexes; the lowest rates are observed in children aged 5-14 years. During adulthood, TB infection rates are consistently higher for men than for women; the male-to-female ratio is approximately 2:1.[6,13] In our series too there was a slight male predominance in TBM subgroup.

CSF examination remains the mainstay of diagnosis of TBM. Usually, there is a lymphocytic reaction (60-400 per mm^3), with raised protein levels (0.8-4 g/l). In the initial stages of the infection, the white cells seen in the CSF may be polymorphonuclear leukocytes, but soon these are replaced predominantly by lymphocytes. There is gradual decrease in the glucose level in the CSF, which is generally less than 50% of the serum levels, although it is never as low as in pyogenic meningitis.[11] Definitive diagnosis of TBM depends on detection of tuberculous bacilli in CSF either by smear examination or bacterial culture. It has been claimed that if large volumes of CSF can be obtained, repeated centrifugation detects bacteria in a high proportion of cases.[14] Measuring the levels of antibodies to different antigens of M. tuberculosis has also been suggested as a diagnostic tool.[15]

Non infective hydrocephalus

Aqueductal stenosis

The growth of the tectum and tegmentum makes the lumen of the neural tube narrow in the region of mesencephalon, leading to narrowing of aqueduct of

Sylvius. Aqueductal stenosis occurs in approximately 10% of children. Several theories exist regarding primary versus secondary forms of aqueductal stenosis. External pressure on mesencephalon has been proposed to obliterate aqueduct secondarily. Scarring and gliosis following infection or hemorrhage can cause acquired aqueductal stenosis. Tumors from the surrounding structures have potential chance to block the aqueduct. Imaging is confirmatory in such situations.[16]

Meningomyelocele

Hydrocephalus complicates open spina bifida in 85-90% of patients. It may manifest after closure of the meningomyelocele as the sac acts as a CSF sump. This is usually associated with Chiari malformation; it is preferable to treat hydrocephalus simultaneously to facilitate wound healing after the repair of myelomeningocele.[17]

Dandy Walker syndrome

This anomaly comprises agenesis of the cerebellar vermis with cystic dilation of the 4th ventricle, enlargement of the posterior fossa and hydrocephalus. The hydrocephalus manifests in the postnatal period. Additional brain malformations leading to neural developmental delay are reported in 70% of cases. Treatment with placement of proximal catheter in the lateral ventricle, 4th ventricle and both the ventricles with a wide connecter has been described. Ideal situation is to treat 4th ventricular hydrocephalus and subsequently supratentorial hydrocephalus by shunt or endoscopic method.[18]

Subarachnoid hemorrhage

Hydrocephalus can follow 10-15% of patients suffering from subarachnoid hemorrhage. The incidence increases with the presence of intraventricular hemorrhage. The mechanism is impaired absorption due to blockade at multiple sites.[19]

Normal pressure hydrocephalus

Usually seen in adulthood, this is classically characterized by gait deterioration, dementia and urinary incontinence. Imaging usually shows enlarged ventricles. In some cases, there may be an attributable cause like infection or hemorrhage. The ICP related symptoms may not be evident. A number of investigations have been advocated in selection of patient for shunt therapy, like isotope cisternography, infusion tests to detect increased CSF resistance in flow, ICP monitoring and therapeutic lumbar drainage. CSF diversion in carefully selected patients gives favorable clinical outcomes.[19]

Posterior fossa tumors

Treatment of hydrocephalus in posterior fossa tumors in children is still a matter of controversy and different centers have their own routines. Majority of posterior fossa tumors in our series had hydrocephalus (100%) or very high incidence. The percentage of preoperative shunting in hydrocephalic posterior fossa tumors has been quite variable in different studies and depends on the policy of the center where the study is done.[20] Some studies have suggested that preoperative shunting can be encouraged.[21] Obviously, this resulted in 100% of the patients becoming either shunt-dependent or at least having a permanently inserted shunt after surgery for posterior fossa tumors. In 1985, the American Society for Pediatric Neurosurgery commissioned a study to try to determine which position was recommendable. Many hospitals in North America participated in this study, and the reports were collected by Robert McLaurin. Based on this study, the conclusion arrived at was that there was no clear evidence that there was any advantage to routine precraniotomy shunting. Since this survey was concluded, most large pediatric neurosurgical centers have abandoned the practice of preoperative shunting, except in very select cases.[22] In our series we have restricted shunting to only patients with gross hydrocephalus with symptoms of raised Intracranial tension.

Shunt malfunction

According to study by Reddy et al, the overall shunt failure rate requiring shunt revision(s) was 46.3%, and the majority of shunt revisions occurred during the first 6 months after shunt placement. The shunt revision rate was significantly greater in pediatric (<17 years) than in adult (>17 years) patients (78.2% vs. 32.5%, $P < 0.001$). They found that age at the time of shunt surgery, previous treatments to shunt surgery, etiology of hydrocephalus, and hydrocephalus type were independently associated with the incidence of shunt revision. Age at shunt placement and sex were significantly associated with multiple shunt revisions.[23] We encountered shunt malfunction in 14% of cases of TBM and 6% of cases in non TBM. We found no statistically significant difference between the two sub groups.

Shunt infection

Shunt infection is a common complication accounting for significant morbidity and mortality. Reported series have shown the incidence ranging from 5 to 15%. Some centers have reported an incidence as low as 1%. Although many factors appear to contribute to shunt infection, it is likely that the contamination of the shunt system at the time of surgery is the primary cause. Approximately 70% of shunt infections present within 2 months and the remaining by 6 months of the surgical procedure.[24] A high index of suspicion should be maintained for symptoms and signs such as pyrexia, meningismus, irritability and even a general lack of well-being. CSF examination is needed to confirm the diagnosis and may be obtained by the aspiration of reservoir or from the ventricle. Appropriate antimicrobial therapy and management of the shunt system is necessary for a good outcome. The commonest organism is coagulase-negative staphylococci, but *Staphylococcus*

epidermidis and *Staphylococcus aureus* are also often recognized.[25] Our shunt infection rate has been 6% and 4% for TBM and non-infective cases. We found no statistically significant difference between the two sub groups.

CONCLUSION

There is no statistically significant increase in complication rate of VP shunt in TBM patients versus non infective cases. The worse prognosis of TBM patients may probably be due to the primary disease process rather than functioning of shunt. Hence a shunt procedure should be promptly offered in case of TBM hydrocephalus with failure of medical management before irreversible brain damage. Prolonging medical therapy in patients in good grades could be harmful.

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