Brief Reports

Role of Shunt Surgery in Pediatric Tubercular Meningitis with Hydrocephalus

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This study was designed to evaluate the indications for ventriculoperitoneal shunting in cases of children with tubercular meningitis, presenting with hydrocephalus. Thirty seven children (less than 18 years of age) of tubercular meningitis with hydrocephalus (TBMH) who underwent ventriculoperitoneal shunting over a three year period (1999 to 2001) were included in the study. Sixteen (43%) children were Palur stage II, 15 (40%) stage III, and 6 (16%) stage IV. Fifteen (40%) children had received antitubercular therapy for less than 4 months and 17 (46%) received therapy for more than 4 months prior to presentation. Five (14%) children had not previously received antitubercular therapy. Shunt related complications occurred in 11 (30%) children and 3 children had undergone revision of the shunt multiple times. Good outcome was seen in 16 (43%) children. Thirteen (35%) had moderate disability and 6 (16%) had severe disability at 3 months of follow up. 62% (n = 10) children in grade II had a good outcome compared to 40% (n = 6) in grade III. All six children in grade IV had a poor outcome. 2 children, both having multiple infarcts, died and the remaining 4 were left with severe disability. We recommend shunt placement in all children of grade II and III TBMH as this policy has yielded the best results. For grade IV children external ventricular drainage, followed by shunting if improvement occurs remains the most cost-effective procedure.

Key words: Hydrocephalus, Tubercular meningitis, Ventriculo-peritoneal shunt.

THE indications and timing of surgical intervention for tubercular meningitis associated hydrocephalus (TBMH) remain controversial, and only few studies have tried to address this issue previously(1-9). Although previous studies have shown age to be an independent prognostic factor in TBMH, there are very few studies dedicated to the pediatric population(1,2). The general tendency is to avoid shunting in this age group, due to its attendant complications, besides the risk of making the child shunt dependent. Shunt malfunction is also very common in children and is related partly to the CSF protein content, which may be very high in the majority of the cases. In this study we retrospectively analyzed the outcome in 37 children with Palur grade II to grade IV TBMH managed with ventriculoperitoneal shunting.

Subjects and Methods

A retrospective analysis was performed on children (less then 18 years of age) with

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TBMH who underwent placement of a ventriculoperitoneal shunt in the Department of Neurosurgery at All India Institute of Medical Sciences, between January 1999 and December 2001. The diagnosis of TBM was made on the basis of typical clinical, radiological and CSF features, as well as positive sputum or CSF culture wherever possible. Children were graded according to the grading system devised by Palur, *et al.*(9) (*Table 1*), and only grade II to grade IV children were considered for this study.

All children received standard four drug antitubercular therapy (consisting of rifampicin, ethambutol, isoniazid and pyrazinamide) according to body weight. Steroids were given only if CT showed thick basal exudates and / or there was evidence of infarct(s).

Children presenting in Palur Grade II and III (having a neurological deficit or altered sensorium) underwent ventriculoperitoneal shunt placement, as soon as the diagnosis of TBMH was established. Postoperatively, active chamber pressing was initiated, which was continued till patient's clinical condition stabilized. An external ventricular drain

TABLE I- Grading system (Palur, et al.) (9)

Grade	Description
I. 1.	Headache, vomiting, fever, and/or neck stiffness
2.	No neurological deficit.
3.	Normal sensorium.
II. 1.	Normal sensorium.
2.	Neurological deficit present.
III.1.	In altered sensorium but easily arousable.
2.	Dense neurological deficit may or may not be present.
IV.1.	Deeply comatose.
2.	Decerebrate or decorticate posturing

(EVD) was placed in grade IV children for 48 hours and only those children who showed neurological improvement during this period were taken up for shunt surgery. Children were followed up on OPD basis regularly and evaluated regarding their scholastic performance and ability to perform daily activities. Based on these parameters the children's outcome was graded into five outcome groups using the Glasgow outcome scale (GOS)(10).

Results

Thirty-seven children underwent ventriculoperitoneal shunt placement for TBMH during the study period. Their age ranged from one year to 18 years with a mean of 11.42 years. There were 25 males and 12 females. Sixteen (43%) cases were Palur stage II, 15 (40%) stage III, and 6 (16%) stage IV. Five (14%) children were diagnosed as TBM only after they presented with hydrocephalus and antitubercular therapy was started subsequently, as compared to 15 (40%)children who had received antitubercular therapy for less than four months prior to presentation, and 17 (46%) children who received therapy for more than four months. Fundus findings had been recorded in 19 (51%) children. Amongst them papilloedema was present in 10 (27%), primary optic atrophy in 1(3%) and secondary optic atrophy in 3 (8%). The fundus was found to be normal in 5 (14%) children. CSF protein content was less then 50 mg% in 12 (32%) children, between 50 mg% and 200 mg% in 13 (36%) children and was greater than 200 mg% in 12 (32%) children.

All children had a contrast enhanced CT (CECT) scan of the head which showed marked ventriculomegaly with periventricular ooze in 14 children, mild to moderate hydrocephalus with periventricular ooze in 16

children and without periventricular ooze in 7 children. Thick basal exudates were present in 11 children and exudates were absent or minimally present in the remaining 26 children. Interestingly five out of the six grade IV children had thick exudates on CT compared to only six out of the 21 children in grade II or III. Though none of the grade II or III children had infarcts, two children, both in grade IV had multiple infarcts on CT.

Shunt related complications occurred in 11 (30%) children and consisted of shunt malfunction (mechanical failure) in 6 (16%) and shunt infection in 5 (14%) children. Three children underwent revision of the shunt more than once (one child had two revisions and two children had three revisions each).

The average follow up was nine months with a range of six months to 24 months. Good outcome was seen in 16(43%). Thirteen (35%) had moderate disability and 6 (16%) had severe disability at 3 months of follow up. There was no child in vegetative state. As expected, children in grade II had the best results, with 10 out of 16 children (62%) having a good outcome compared to 40% (n = 6) in grade III. All six children in grade IV had a poor outcome. Two children, both having multiple infarcts, died and the remaining 4 were left with severe disability.

Discussion

Cairns for the first time made bilateral burr holes for estimation of ventricular size as well as CSF sampling in TBMH in 1951(11). Bhagwati in 1971 described ventriculoatrial shunting in TBMH in 7 patients(6). However, due to fear of miliary spread of the disease, ventriculoatrial shunting has since been replaced by ventriculoperitoneal shunting universally. Lamprecht, *et al.*(1) recommended shunting in patients only if they failed maximal medical therapy, deteriorated neurologically, or developed obstructive hydrocephalus to reduce shunt related complications. We use Indian shunts (like the Chhabra shunt[®]) costing Rs. 1200-1500 (US\$ 30) for all shunt procedures and they have proven to be very cost effective in the setting of TBMH. The chamber of the these shunt are also suited for manual pressing invivo, which is important in TBMH as the CSF protein content may be very high in these children, resulting in shunt malfunction. Czosnyka, et al.(12) simulated chamber pressing in a Chhabra medium pressure shunts by the addition of a pulsating waveform of variable amplitude to the proximal pressure (frequency varied from 90 revolutions/min to 5/min) and showed that the lower end of the pressure-flow performance curve consistently shifted to the left (towards lower pressures). Thus chamber pressing results in increased drainage of CSF and may also help in 'flushing' the system off proteinaceous debris, decreasing shunt malfunction rates. Our shunt malfunction rate of 16% is much less than that of other recent studies on TBMH(1,5). This view is however, not shared by all and some feel that the negative pressure applied to the chambers sucks in ventricular fimbria and blocks the shunt(13).

The shunt infection rate in our study was 14%, which is similar to other studies(1,5) in TBMH. All the shunts were done on an emergency basis, and this factor could have had a bearing on the increased shunt infection rates. Recently, antibiotic impregnated shunts and ventricular drainage systems have been introduced, which reportedly have a much lower rate of colonization(14,15), but are relatively expensive. We, however, do not have any experience in using such devices. Another likely cause of the high incidence of shunt infection could be the depressed immunity, in such children. Other infective shunt complications such as peritonitis and

Key Messages

- Shunt placement is advisable in all children in Palur grade II and III TBMH.
- For Palur grade IV children external ventricular drainage for 48 hours, followed by shunting if improvement occurs, remains the most cost-effective procedure.

pseudocyst formation, mentioned in the literature(13), were not seen in our series.

Using multivariate analysis Misra, et al. (16) have shown that hydrocephalus, besides stage of TBM, age and cranial nerve palsies have significant prognostic value in TBM. Other studies have also shown the presence of hydrocephalus to be associated with persistent disability and poor prognosis(5,7). Although communicating hydrocephalus is seen in up to 82% of the cases of TBMH(3), it is the obstructive variety that carries a poorer prognosis. This differentiation is however superfluous if there is a policy (like ours) of shunt placement in all cases of hydrocephalus, irrespective of the fact whether it is obstructive or communicating in nature. Demarcation of hydrocephalus into obstructive and communicative varieties may, in fact, prove counterproductive, as it instills a sense of complacency in treating the communicating variety, as seen in the study by Lamprecht(1). The role of CT scan in evaluating the intracranial pressure is also controversial and periventricular lucencies may be indicative of inflammatory process rather then an increase in intraventricular pressure(5). The reverse is also true and children can have a high intracranial pressure without priventricular lucencies. We have also seen the CSF to be under high pressure on inserting the ventricular end of the shunt, in a number of children who did not have periventricular lucencies on CT scans.

down guidelines for shunt surgery in TBMH. They recommended that grade 1 and grade 2 patients with TBMH are ideal candidates for shunt surgery and poor grade patients (Grade III and IV) should initially undergo external ventricular drainage via a ventriculostomy, and only if clinical improvement occurs in the neurological status within 48 hours, they could be taken up for shunt placement. Although being a seminal study on TBMH, the recommendation of shunt placement in Grade I patients is controversial in view of the fact that only 5 out of their 114 patients with TBMH were in grade 1. We excluded grade I children from our study as their number was small (n = 3) and all had shown improvement on medical therapy (antitubercular therapy with the addition of acetazolamide and furosemide). Also the fact that only three children in palur grade I presented with hydrocephalus is a pointer towards the extremely good prognosis this group. Bullock, et al.(5), did not shunt all patients in grade I and II TBMH, and reserved the procedure for those cases which showed progressive increase in ventricular size on CT in spite of full medical treatment. We, however, shunt all children of grade II and III TBMH, and in grade IV initially an external ventricular drain was put for 48 hours, proceeding to shunting only if there is improvement in neurological status. We keep the EVD for 48 hours as a previous study at our center has shown that the rate of CSF colonization and infection increases dramatically after this period(17). This policy has yielded good results with 62%

Palur, et al.(9), for the first time laid

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children in grade II and 40% in grade III having good outcome in our series.

Role of steroids remains controversial in the treatment of TBM. One Meta analysis involving 595 patients has shown beneficial effects of steroids in TBM(18). Steroids were associated with fewer deaths and a reduced incidence of death and severe residual disability. We therefore add steroids if CT shows thick basal exudates and / or there is evidence of infarct(s). As these findings were seen mainly in grade IV patients, steroids were prescribed in this group. We however could not document any beneficial effect of addition of steroids, as all grade IV children had a poor prognosis.

In conclusion we recommend shunt placement in all children of grade II and III TBMH as this policy has yielded the best results. For grade IV children external ventricular drainage, followed by shunting if improvement occurs remains the most costeffective procedure in this group of children.

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Correlation of Serum Parathormone Level with Biochemical Parameters in Chronic Renal Failure

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A prospective study was carried out to assay the level of serum intact parathormone and its correlation with biochemical parameters in patients with chronic renal failure (CRF). The study included 64 children (44 with CRF, and 20 age and sex matched controls). Serum intact parathormone (iPTH), serum creatinine, urea, calcium, inorganic phosphate and alkaline phosphatase were estimated. Creatinine clearance (Ccr) was estimated by Schwartz formula. Patients with CRF were divided into four groups based on their Ccr (mild CRF with mean Ccr $59.17 \pm 1.18.53 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 34.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr } 4.98 \pm 7.75 \text{ mL/min}/1.73 \text{ m2} (n = 6) \text{ m2} (n = 6) \text{ moderate CRF with mean Ccr }$ m2 (n = 7); severe CRF with mean Ccr 17.71 \pm 5.40 mL/min/1.73 m2 (n = 15); and end-stage renal disease with mean Ccr 6.46 ± 1.71 mL/min/1.73 m2 (n = 16). Mean serum iPTH levels were 93.00 \pm 46.62 pg/mL in CRF and 16.52 \pm 9.35 pg/mL in controls. Groupwise mean serum (iPTH) levels were 48.50 ± 4.76 , 67.29 ± 7.91 , 82.42 ± 9.67 and 130.66 ± 58.74 pg/mL in mild, moderate, severe CRF and end-stage renal failure respectively. Mean serum iPTH level of CRF (93.00 \pm 46.42 pg/ mL) negatively correlated with mean Ccr $(22.02 \pm 18.53 \text{ mL/min/l.73 m2})$ (P < 0.001) and mean serum calcium (7.30 \pm 1.02 mg/dL) (P<0.001) and positively correlated with mean inorganic phosphate (5.76 \pm 1.1 mg/dL) (P<0.05) and mean alkaline phosphatase (355.14 \pm 185.53 UL) (P < 0.001). We conclue that increased iPTH level occur even early in the course of CRF and progressive hypocalcemia and hyperphosphatemia are the initiating factors for the development of hyperparathyroidism.

Keywords: Chronic renal failure, Hyperparathyroidism, Parathormone

CHRONIC renal failure (CRF) produces a number of abnormalities of calcium and phosphorus metabolism. Secondary hyperparathyroidism develops early in the course of chronic renal insufficiency(1), even at the glomerular filtration rate (GFR) of 50-80 mL/min/ $1.73 \text{ m}^2(2)$. It is generally thought to result from hypocalcemia as a result of

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