POST MENINGITIC HYDROCEPHALUS – FACTORS PREDICTING OUTCOME

DISSERTATION

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CERTIFICATE

This is to certify that the dissertation entitled "**POST MENINGITIC HYDROCEPHALUS – FACTORS PREDICTING OUTCOME**" was done under our supervision and is the bonafide work of **Dr.M.Abirami Sundari**. It is submitted in partial fulfillment of the requirement for the M.Ch (Neurosurgery) examination.

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CONTENTS

S.No.		Page No.
1.	Introduction	1
2.	Aim of The Study	3
3.	Review of Literature	4
4.	Materials and Methods	24
5.	Analysis of Results	30
6.	Discussion	45
7.	Conclusion	52
8.	Bibliography	
9.	Proforma	
10.	Master Chart	

INTRODUCTION

Post Meningitic hydrocephalus is a fairly common cause of morbidity and mortality in the neurosurgical wards and is an important cause for emergency admission. Although the incidence is decreasing and survival is better than before, there is uncertainty when it comes to identifying the factors which predict the outcome of the disease: post meningitic hydrocephalus.

Meningitis can be of varied aetiology namely bacterial, viral or fungal. In our population tuberculous meningitis is a common cause. Whatever the causative organism may be inspite of advanced antimicrobials and other supportive drugs available for treatment today, significant number of these patients invariably develop hydrocephalus.

The most definitive treatment for hydrocephalus is surgery even when the meningitic process is very severe at the time of presentation[1]. The commonest and widely used surgical procedure for hydrocephalus is ventriculo-peritoneal shunt in most hospitals in our country.

It is said that the second most common reason for being sued for negligence in neurosurgery is related to hydrocephalus management[2] Therefore it is essential that one should be clear in understanding the factors, besides surgery, that determine the final outcome of the disease process “Post meningitic hydrocephalus”. This study has been done to understand this question and help in concluding the predictors that affect the final outcome in post-meningitic hydrocephalus.

AIM OF THE STUDY

(i) To identify factors that can predict the outcome of post meningitic hydrocephalus after starting definitive treatment ie., ventriculo peritoneal shunt.

(ii) To evaluate which of the predictors

Clinical features

CSF examination

Computed Tomography of the brain are statistically significant to affect the outcome.

(iii) To correlate and clarify if other associated systemic diseases contribute to the outcome.

(iv) To determine if the time interval from onset of the symptoms to surgical intervention has any contribution in improving the outcome.

(v) If age is a factor influencing the outcome.

(vi) To decide which is the single most reliable predictor of the outcome.

REVIEW OF LITERATURE

Post meningitic hydrocephalus may be of tuberculous origin or non tuberculous origin.

Studies have shown tuberculosis to be commonest cause of post meningitic hydrocephalus. Tuberculosis of the central nervous system accounts for 5% of extra pulmonary tuberculosis. It is most often seen in children but also develops in adults, especially in HIV infected patients. Tuberculous meningitis results from hematogenous spread of primary or post primary pulmonary disease or rupture of a sub ependymal tubercle in subarachnoid space.

The incidence of tuberculosis is on the increase worldwide[3] between 1 and 2% of children with untreated extracranial tuberculosis develop TBM. In India TBM is still a major cause of disability and death. The highest incidence is in the first five years of life, but with the increasing incidence of HIV infection, incidence among adults has also increased. In children, TBM is usually a complication of primary infection. In adults it may occur as an isolated form of tuberculosis or in association with either pulmonary or military tuberculosis. Infection is fatal if not treated within 1-8 weeks and risk of sequelae is high if treatment is delayed[4].

The majority of the TBM are due to Mycobacterium var.hominis. The bovine tuberculous bacillus is responsible for a smaller percentage, while atypical mycobacteria are unusual causative organisms.

Until the decisive study of Rich et al. in 1933[25], it was believed that TBM was a direct and immediate result of haematogenous infection of the meninges. The study of Rich et al. postulated that TBM arises in two stages. Initially, tuberculous lesions form in the brain or the meninges from haematogenous dissemination of bacilli during primary infection and then meningitis develops via discharge of bacilli and tuberculous antigen from a subjacent focus directly into the sub arachnoid space. Host factors such as malnutrition, alcoholism, diabetes though associated with a higher incidence of tuberculosis their association with meningitis cannot be proved[5].

While the major impact of the disease process falls on the basal meninges, parenchymatous lesion of the brain due to direct extension of the inflammatory process or secondary to vascular changes are consistently encountered in most cases[6]. A thick exudate usually fills the basal cisterns and surrounds major blood

vessels at the base of brain leading to morbidity and mortality. The extent of brain involvement is variable. The important parenchymatous reactions are

(a) Border zone reaction

(b) Infarcts

(c) Hydrocephalus

The following factors are responsible for the clinical picture of TBM[7]:

(i) Thick basal exudates (cranial nerve palsy and hydrocephalus)

(ii) Vasculitis and vascular occlusion(focal neurological deficit)

(iii) Allergic reaction to tuberculoprotein (CSF changes)

(iv) Cerebral oedema (impaired consciousness, seizures)

(v) Tuberculomas may present with focal neurological deficit, seizure or obstructive hydrocephalus.

Acute onset of clinical features has been seen in 50% of the children but only 14% of the adults[8]

Based on these, a patient may therefore present with

A. Seizures

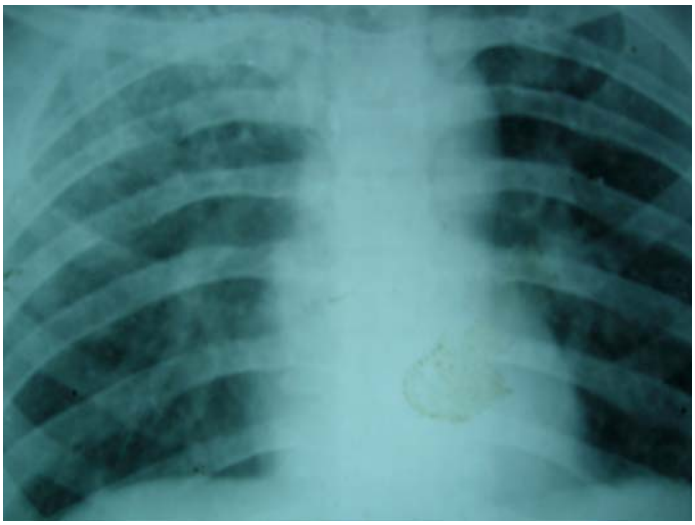
B. Focal neurological deficits

C. Signs of toxicity such as altered sensorium

D. Extra cranial associations such as extracranial tuberculosis, diabetes, hepatitis and HIV.

E. Hydrocephalus with features of increased intracranial tension.

CSF is usually macroscopically clear and colourless and may show a cobweb like appearance on standing. Microscopically moderate pleocytosis with predominantly lymphocytes is seen.



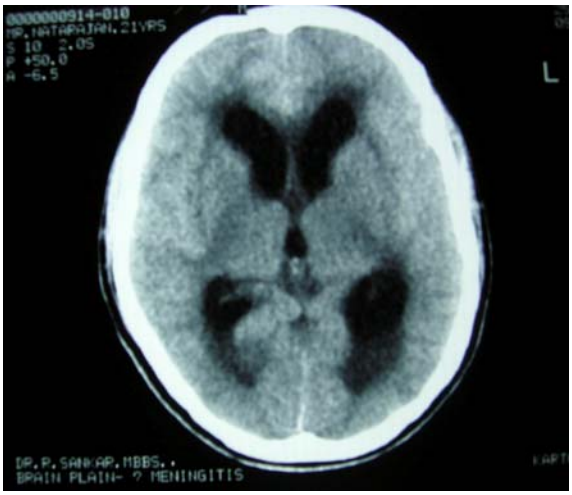


Fig 1, 2. A patient with pulmonary tuberculosis presenting with hydrocephalus

CSF protein is usually elevated even upto 500mg / dl with a moderately reduced CSF sugar. AFB seen in CSF is the most diagnostic of tuberculous meningitis. Test such as C – reactive proteins, CSF-PCR specific for tuberculosis can also be there.

Computed Tomography of the brain usually demonstrates ventricular enlargement in majority of the patients with TBM[9]. CT brain especially with contrast shows associated features like basal exudates, infarcts and rarely tuberculomas.

Pyogenic meningitis

Pyogenic meningitis implies inflammation of the leptomeninges as a result of bacterial infection. It is an important cause of morbidity and mortality among hospitalized patients. The pathogenesis, microbiology, diagnosis vary in different age groups. The susceptibility is greatest in the first year of life. Though overall incidence has been decreasing, meningitis due to Haemophilus type B and Streptococcus is increasing.

The causative organisms vary with age of the patient and also the geographical distribution. In adults, the three main agents are S. Pneumoniae, N. Meningitidis and H. Influenza[10]. In neonates, the major organisms are group B streptococcus, E.Coli and staphalococcus. In young children, H. Influenza is the most common aetiological agent. Other infrequent agents are Klebsiella, pseudomonas, proteus, Listeria, salmonella, flavobatcer, bacteriods, Acinetobacter etc.,

Pathogenic bacteria can reach the CNS by one of the three routes: by direst invasion, if there is communication between the CSF space and integumental surfaces: by bacterial dissemination from infected sources or by haematogenous spread. Factors which predispose to development of meningitis include CSF

rhinorrhoea, shunts, otitis media, paranasal sinusitis or mastoiditis, dermal sinus.

Clinical features may vary with age, severity, duration of illness, bacterial pathogen and with presence of complications. The classic clinical presentation of adults with bacterial meningitis includes headache, fever, and meningismus, often with signs of cerebral dysfunction. These signs are present in only 50% of the adults and their absence does not rule out meningitis. Cerebral dysfunction is manifested primarily by confusion, delirium or a declining level of consciousness ranging from lethargy to coma. Cranial nerve palsies involving cranial nerves iv, vi, vii, ix and x are found in 10-20% of the patients. Seizures occur in 40% of the cases. Recurrent seizures and focal neurological deficits are more common in H.Influenza than meningococcal meningitis.

CSF may be clear or turbid with intense pleocytosis. In majority of cases, CSF glucose is profoundly reduced. There is moderate increase in protein but less when compared to tuberculous aetiology. Counter current electrophorsis method for elimination of CSF antigens for microorganism is fast test for

early diagnosis. Blood culture may aid in diagnosis of source. Culture and gram staining of CSF if positive is most specific for ultimate diagnosis of phylogenic meningitis.

In bacterial meningitis typically the opening pressure of CSF is very high. CSF may be turbid due to either increased cell count or sometimes due to the presence of micro organisms. CSF culture identifies the pathogen in 70-85% of the patient.

Early bacterial meningitis is characterized by sub arachnoid exudates over the cerebrum, brainstem and spinal cord. The exudates are streaky and distributed along the sulci[11]. In late stages the exudates are predominantly over the cortex than the basal meninges. Cortical atrophy and porencephaly may follow resulting in long term sequelae. Ischemic changes may occur but major vessels are usually normal.

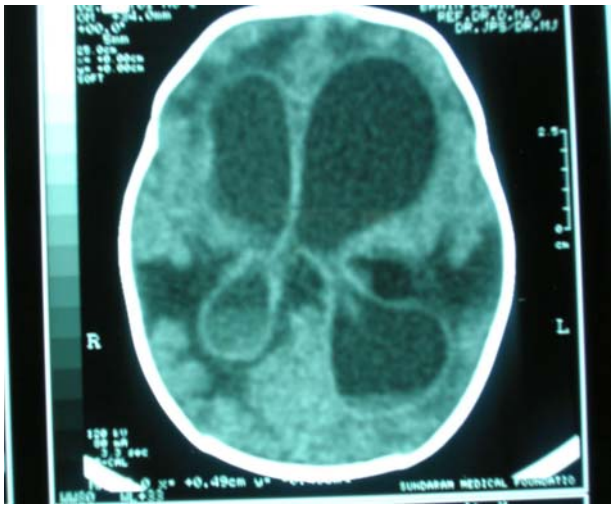


Fig.3, A Child presenting with gross hydrocephalus and fluid level in ventricles - Abscess indicating active meningitic process

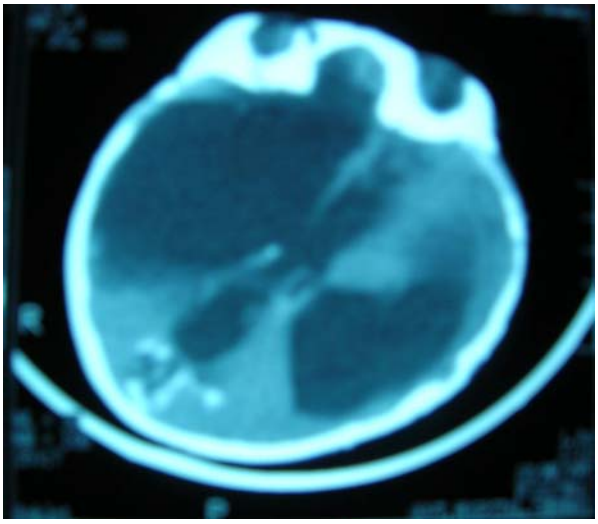


Fig.4, An infant with severe hydrocephalus and intracranial calcifications following intrauterine TORCH infection

Meningitis continues to be one of the major causes of morbidity and mortality throughout the world. Therefore a better

understanding of the factors that cause morbidity and mortality becomes mandatory. Have the factors contributing to the outcome changed over decades or are they the same? If so, what are these factors?

Hydrocephalus is one of the most common result of meningitis. Hydrocephalus has been recognized for centuries. The first description of hydrocephalus was made by Rhazes (850-923 AD)[13]. Vesalius (514-1564) gave a clear account of ventricular dilation and mentioned that infants with grossly enlarged heads could survive into adulthood. Morgagni confirmed in 1761 the accumulation of fluid within the dilated ventricles and pointed out that the brain may be reduced to almost the thinness of a membrane.

The knowledge about absorption of CSF was slower in accumulating and even today we have controversies. Dandy and Blackfen[14] showed that cerebrospinal fluid absorption took place diffusely through the venous sinuses and sub arachnoid spaces and in 1914, Weed provided evidence that the arachnoid villi also absorbed fluid.

The mean rate of CSF formation is 0.37ml/min. This represents a daily formation of 500 ml of CSF or renewal of the total amount of CSF every 6-8 hours.

The arachnoid villi are the principal sites of absorption of CSF under normal conditions. Compensatory CSF absorption through the ependyma or the choroids plexus occurs when then the absorption through the arachnoid villi is impaired. Absorption has also been shown to occur via dilated central canal into the spinal cord.

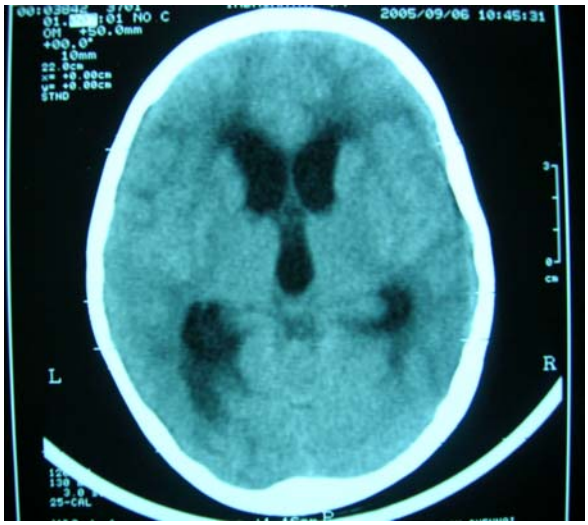


Fig.5, CT Scan of an adult with hydrocephalus and periventricular lucency indicating raised intracranial tension

An increase in the amount of fluid within the ventricles may result from one of the three following mechanisms or a combination of three.

Over production of the fluid

Impaired absorption (arachnoid villus and venous sinus)

Obstruction along CSF pathways.

The dilatation seen in post meningitic patients may be due to impaired CSF absorption or obstruction along CSF pathways.

Obstruction to entry of CSF into the arachnoid villi or the venous sinuses results in accumulation of fluid within distended subarachnoid spaces. In 1931, Symonds popularized the term “otitic hydrocephalus” describing a syndrome of raised intracranial pressure and papilloedema caused by sinus thrombosis of otitic origin. Arachnoid granulations may fail to develop resulting in defective absorption as also seen in inflammatory disease like tuberculous meningitis[15]. Following inflammation the ependyma, the opposing surfaces may come together and get united by glial tissue. Such post meningitic narrowing of the aqueduct can occur in the adults.

Aqueductal dysfunction can be due to obstruction by a micro tuberculoma or due to disturbance in ciliary movement in the aqueduct[16]. When the foramen of Magendie and Luschka are obstructed the fourth ventricle dilates. The commonest inflammatory cause of fourth ventricular obstruction in India is Tuberculosis[17].Various parasites have been known to lodge within the ventricular cavity and occlude it. They produce ventricular obstrcution by dense arachnoiditis. Pyogenic meningitis ranks low in the list of obstructive disorders. Fungi have been noted to gum up the fourth ventricle. Adhesions between the pia and arachnoid can effectively prevent the onward flow of CSF. In some cases intra uterine meningitis may cause adhesive sub arachnoid block. Protein transudates from brain and spinal cord inflammations can also cause sub arachnoid blocks.

Based on their results of neutral phenylsulphanaphthalein tests, Dandy and Blackfann sub divided hydrocephalus into two groups[18]. If chemical injected into the lateral ventricle was recovered within 20 minutes from the spinal sub arachnoid space, the hydrocephalus was termed as ‘communicating’ implying a patent communication between the ventricles and the

sub arachnoid space. If no recovery was possible it was termed non communicating hydrocephalus. With present day radiological techniques it is possible, to localize with accuracy the exact site of blockage of the CSF flow. Hence a more helpful classification is as follows: The hydrocephalus may be due to

(1) Over production of CSF (rare)

(2) Obstruction to flow of CSF in Lateral ventricles

Foramen of Munroe

Third ventricle

Aqueduct of Sylvius

Fourth ventricle

Sub arachnoid spaces

(3) Absorption defects

Enlargement of the ventricles stretches and disrupts the ependyma. This usually starts at the angles of the lateral ventricle[19]. Enlargement and elongation of the cytoplasmic processes of the ependymal cell are accompanied by the loss of cilia and microvilli. Gopinath et al., in their study of kaolin induced hydrocephalus in rabbits, found the ependymal cells in the lateral ventricle to be stretched with clear spaces in between

them. Widening of the gap junctions was noted with resultant communication between the CSF and the sub ependymal region. Scanning electron microscopy in hydrocephalic rabbits revealed separation of clusters of villi emanating from the ependyma. There was marked reduction in ciliary density and pits were seen in the dorsal region of the third ventricle. There was almost total replacement of globular excrescences by pleomorphic microvilli it the infundibular region. Collins proposed that the degree of ependymal damage in hydrocephalus depends on the rate of ventricular dilatation. The character and distribution of the pathological changes are dependent on the age at which the hydrocephalus develops, the rate and extent of ventricular enlargement and the duration of hydrocephalus[20]. Once the ependyma is disrupted sub ependymal astroglial processes offer little resistance to the stretching forces and the CSF escapes into periventricular white matter. In advanced cases the whole of the ventricular ependyma may be denuded or grossly altered. This egress of CSF from the ventricle into the periventricular white matter results in hydrocephalic oedema seen in CT brain as peri ventricular lucency. Caner et al,[21] suggested from experimental evidence that as a result of the ischemia caused by the

hydrocephalus, there is a high level of lipid peroxidation which may lead to the observed vascular changes. Reactive astrocytosis, progressive demyelination and a reduction in the length and number of dentritic branches are other sequelae. All these lead to gradual reduction in the bulk of cerebral tissue, resulting in a large ventricle and a grossly thinned out cortex in advanced cases. The total blood flow is reduced as hydrocephalus advances. This process can be stopped by the insertion of a CSF shunt.

CT brain confirmation of hydrocephalus is mandatory before shunt is undertaken. The normal bifrontal width of the lateral ventricles is on an average 31 percent of the brain width at this level. When the bifrontal ventricular width expressed as a percentage of the brain width exceeds 35% hydrocephalus is diagnosed.

Evan’s index ratio between greatest with of the anterior horns of lateral ventricle and internal transverse diameter of the skull. Ratio of 0.3 or greater indicates abnormal dilatation of the anterior horns the anterior horns of lateral ventricles should normally be semilunar. The size of the bodies of the lateral

ventricle is calculated by Schiersmann index this index is calculated at the parietal area of bodies of lateral ventricle with biparietal distance of outer table of calvarium divided by distance between the bodies of the lateral ventricle. The index is abnormal when it is less than 4. Ventricular size index : Bifrontal diameter / frontal horn diameter, Heinz etal 1980.

Normal	=	30%
Mild hydrocephalus	=	30 – 39%
Moderate hydrocephalus	=	40 – 46%
Severe hydrocephalus	=	> 40%

Once the diagnosis of progressive hydrocephalus is confirmed, the earlier the operation is done, the better. Early surgery helps to restore optimum function and development of neural tissues. Shunts have been done to drain the CSF in to practically every body cavity, organ system and tissue space. These are of historical interest. Ventriculo salphingostomy, lumbar sub arachnoid salphingostomy, lumbar sub arachnoid ileostomy, ventricle to thoracic duct, ventricle to Stensons duct and ventricle to Whartons duct are some of the examples. However ventriculo peritoneal shunt continues to be the main procedure of choice at present. Shunting of CSF into the

peritoneal cavity was introduced by Ferguson S. in 1898[22]. This continues to be the procedure of choice in many centres till date.

MATERIALS AND METHODS

This study is based on a multivariant analysis of fifty three randomized cases of post meningitic hydrocephalus patients admitted and operated in the Institute of Neurology, Madras Medical College, Chennai over a period of twenty four months from July 2003 to June 2005.

Inclusion Criteria

All the patients enrolled in the study had fever with features of meningitis and headache. CSF was not taken into preoperative criteria as lumbar puncture was contraindicated in these patients. Radiologically the CT scan of these patients showed dilated ventricles.

Exclusion Criteria

Any patient requiring revision of shunt were excluded from this study. Patients who did not come for follow up for a minimum period of three months were also excluded. Apart from this patients who did not want surgery were not included in this study. Any patient who died before surgery were also excluded. The outcome was evaluated twelve weeks after shunt surgery.

The factors that were taken for analysis were divided in to five main divisions:

I. Clinical signs and symptoms
II. CSF study
III. CT brain
IV. Age
V. Time interval between the onset of symptoms and surgery.

1. Clinical symptoms and signs

Clinical symptoms and signs with which the patients presented were again sub divided and analyzed individually. Of the various presentations all patients had history of fever and headache. These formed some of the basic criteria for inclusion in the study. The variants analyzed were:

A. Seizures

B. Altered sensorium

C. Focal neurological deficit

D. Extracranial associations.

A. Seizures could be focal seizures or generalized tonic clonic seizures. Seizures at the time of presentation or after admission but before surgery were included.

B. Altered sensorium – in this any patient with a Glascow coma scale of less than 13 were taken into account.

C. Focal neurological deficit: The neurological deficits usually seen were hemiplegia or hemiparesis, cranial nerve palsies mainly of the facial nerve and the abducent nerve. Hypertonia of the limbs with spasm and ophisthotonus were also included in neurological deficit.

D. Extracranial association could be any other systemic disease or pathology. This not only included tuberculosis of extra cranial regions but also diseases like HIV, Diabetes Mellitus, malnourishment and hepatitis.

II. CSF Study:

The two main variants were CSF protein and CSF sugar. Other things like cell count, chloride etc were not considered as they have been already proved to be insignificant. Macroscopic

appearance of CSF was not considered as turbid CSF is a contraindication for shunt in our Institute. In the protein levels and sugar levels they were sub divided into high, normal or low values.

Proteins: High = >51 mgs / dl

Normal = 40-50 mgs / dl

Low = <39 mgs / dl

Sugar: High = >61 mg / dl

Normal = 50-60 mg / dl

Low = <49 mg / dl

Whenever there was increased protein with decreased sugar in CSF it was considered that meningitis was active at the time of surgery as all the CSF studies were from CSF collected at the time of surgery.

III. Computed Tomography of the Brain:

Here the dilatation of ventricles – communicating or obstructive was a basic requirement for the study. Apart from this the presence of

A. Basal exudates

B. Parenchymal changes tuberculomas or infarcts

C. Periventricular lucency were considered as prognostic factors for outcome following shunt.

Under these we studied not only their presence or absence but also individually their significance in each outcome. i.e., for example not only the significance of increased proteins or reduced sugar in CSF was taken into account but also how much of raised proteins was associated with improvement, sequelae or death.

Again a comparison was made between patients with just plain hydrocephalus and those with hydrocephalus associated with basal exudates, periventricular lucencies and other parenchymal changes.

IV. Time Factor:

Under this the time interval between the onset of symptoms and shunt was taken as a possible predictor of outcome. They

also reflected the awareness of our people to importance of early admission and treatment of diseases.

Outcome grading:

Based on these predictors, outcome was classified and studied as

A. Improved

B. Sequelae

C. Dead

Improved patients were those who were evaluated at the end of twelve weeks after shunt and found to have no neurological deficit in terms of function. Factors like intelligence, memory were not evaluated.

Patients with sequelae were those with some neurological deficit (severity of deficit not considered) like weakness of limbs or cranial nerve deficits.

ANALYSIS OF RESULTS

The analysis of factors predicting outcome in patients with post meningitic hydrocephalus was done using various statistical tools like chi-square, anova and multiple comparisons. The

outcome was analysed in terms of improvement, sequelae and death.

CLINICAL CORRELATES

Table 1:

Seizures Vs Outcome

Seizures were included irrespective of whether they were focal or generalized tonic clonic seizures. Patients included were those who had seizures before surgery. Those patients who developed seizures after surgery were not included.

SEIZURES	IMPROVED		SEQUELAE		DEATH	TOTAL	
	No.	%	No.	%	No. %	No.	%
Present	10	35.7	4	30.8	6 50	20 37.7	
Absent	18	64.3	9	69.2	6 50	33 62.3	

Chi square = 1.08, p = 0.581

Table 2 :

Altered Sensorium Vs Outcome

Altered sensorium meant patients with conscious level anything below GCS 13/15.

ALTERED SENSORIUM	IMPROVED No. %	SEQUELAE No. %	DEATH No. %
Present	3 10.7	2 15.4	6 50
Absent	25 89.3	11 84.6	6 50

Chi square : 8.18 , p = 0.0167

Table 3 :

Neurological deficit Vs Outcome

Neurological deficits commonly seen and included in this study were abducent nerve, facial nerve paresis or palsy, hemiparesis or hemiplegia or increased tonicity of limbs.

Neurological deficit	IMPROVED No. %	SEQUELAE No. %	DEATH No. %
Present	5 17.9	11 84.6	6 50
Absent	23 82.1	2 15.4	6 50

Chi Square:16.75 , p = 0.001

Table 4 :

Extracranial associations Vs Outcome

Extracranial associations were extracranial tuberculosis itself or generalized diseases like HIV, hepatitis, diabetes, malnourishment.

Extracranial associations	IMPROVED		SEQUELAE		DEATH	
	No.	%	No.	%	No.	%
Present	6	21.4	3	23.1	2	16.7
Absent	22	78.6	10	76.9	10	83.3

Chi Square = 0.172, p = 0.917

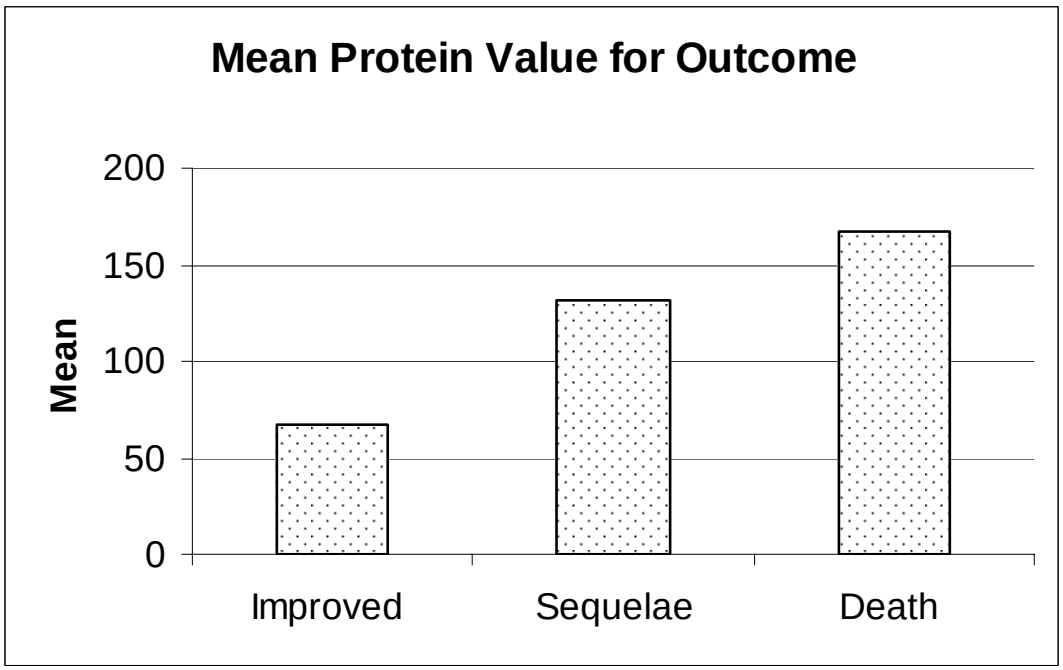
CSF STUDY

Under CSF analysis the level of proteins and sugar were considered. Cell count was not considered as most studies have already disproved their association with outcome.

Table 5:

Protein Vs Outcome

PROTEIN	IMPROVED	SEQUELAE	DEATH
NORMAL	7	1	2
HIGH	16	9	9
LOW	5	3	1



The value protein level in CSF analysis was divided into normal, low or high as quoted in standard textbook[24].

Table 6 :

Sugar Vs Outcome

As in proteins, sugar values in CSF were also divided into normal, high or low and their association with outcome was studied.

SUGAR	IMPROVED	SEQUELAE	DEATH
NORMAL	5	3	1
HIGH	6	2	4
LOW	16	9	7

CSF STUDY:

Variant	n	mean	Std. deviation	Minimum	Maximum
S.Improved	28	46.25	46.25	25	65
Death	12	43.33	43.33	20	75
Sequelae	13	38.23	38.23	20	65
Total	53	43.62	43.62	20	75
P.Improved	28	67.00	40.32	30	190
Death	12	167.25	120.25	35	450
Sequelae	13	132.00	107.81	30	295
Total	53	105.64	91.86	30	450

ANOVA

Variant	Significance
Sugar	0.291
Protein	0.002

Analysis of Variance (ANOVA)

The analysis of variance frequently referred to by the contraction ANOVA is the statistical technique specially designed to test whether the means of more than two quantitative population are equal.

Basically, it consists of classifying and cross – classifying statistical results and testing whether the means of a specified classification differ significantly. In this way it is determined whether the given classification is important in affecting the results.

Assumption :

The assumptions in analysis of variance are as follows :

1) Normality

2) Homogeneity

3) Independence of error.

It may be noted that theoretically speaking, whenever any of the assumption is not met, the analysis of variance technique cannot be employed to yield valid inferences.

Multiple Comparisons

Dependent Variable	(I) outcome	(J) outcome	Significance
Protein	Improved	Death	0.003
		Sequelae	0.059
	Death	Improved	0.003
		Sequelae	0.541
	Sequelae	Improved	0.059
		Death	0.541

COMPUTED TOMOGRAPHY

Table 7 :

CT Findings Vs Outcome

In all patients post operative CT brain was done and was noted that there was a definite reduction in ventricular size.

CT FINDING	IMPROVED		SEQUELAE		DEATH	
	No.	%	No.	%	No.	%
HYDROCEPHALUS	6	21.4	0	0	2	16.7
HYD. WITH ASSO	22	78.6	13	100	10	83.3

Chi Square = 2.72, p = 0.598

Here associated findings considered are periventricular lucency, exudates and parenchymal changes either infarcts or space occupying tuberculomas. Analysis was done by comparison of patients with hydrocephalus as the only finding in

CT and those with above associated findings. The predictive value of each finding i.e., periventricular lucency or exudates or parenchymal changes were individually analysed in relation to outcome.

Table 8 :

Hydrocephalus with exudates Vs Outcome

EXUDATES	IMPROVED	SEQUELAE	DEATH
Present	4	5	4
Absent	22	8	8
TOTAL	28	13	12

Table 9 :

Hydrocephalus with Periventricular lucency Vs Outcome

PERIVENTRICULAR LUCENCY	IMPROVED	SEQUELAE	DEATH
Present	15	12	9
Absent	13	1	3
TOTAL	28	13	12

Among death who had hydrocephalus + exudates = 4/12 = 33.3%

Among Death who had periventricular lucency = 9/12 = 69.2%

Proportion test was done and $p = <0.05$.

TIME INTERVAL BETWEEN ONSET AND SURGERY

Table 10 :

Time Factor Vs Outcome

TIME	IMPROVED	SEQUELAE	DEATH
<1 WEEK	4	2	2
1WEEK-1 MONTH	21	6	8
>1 MONTH	3	5	2
TOTAL	28	13	12

Chi square = 4.814, p = 0.306 (not significant)

AGE

Table 11 :

Age Vs Outcome

AGE	IMPROVED	SEQUELAE	DEATH
0-1 YEAR	6	3	2
1-15 YEARS	12	8	5
15-50 YEARS	7	2	4
>50 YEARS	3	0	1

DISCUSSION

Post meningitic hydrocephalus is one of the most common complications of meningitis which has varied aetiological agents ranging from acute non-specific organisms to tuberculous bacilli. Several factors like age, duration between onset and treatment, degree of neurological damage that has occurred have been

studied extensively in the past. However most of the data were in the pre computed tomographic (CT scan) era. With the improvement in diagnostic tools and better treatment protocols the morbidity and mortality have significantly reduced. Recent studies have mostly evaluated the efficacy of different types of surgical options in the management of postmeningitic hydrocephalus. There is not much data available to compare the commonly available parameters like clinical assessment, CSF analysis and CT scan brain influencing the outcome of the disease. So, this study has evaluated these very factors in order to predict the outcome in patients with post meningitic hydrocephalus who have undergone ventriculo peritoneal shunt. The outcome was analysed after a period of twelve weeks.

On analyzing the 53 patients over two years the following were what the study could decipher about factors predicting the outcome of patients with post meningitic hydrocephalus.

I. **Clinical features** :

a. Seizures

From analyzing the data it was seen that seizures as such, either at the time of presentation or during admission were no

way connected to the final outcome of post meningitic hydrocephalus. Fact that 50% of the patients died in those with seizures and 50% in those without seizures shows that death is unrelated to the presence of seizures.

b. Altered Sensorium

Altered sensorium indicates an ongoing toxic process and active infection. Progressive active disease will naturally lead to more morbidity and mortality. In this study there is a definite correlation between presentation of a patient with altered sensorium and death which confirms our logical thinking. This is one of the most significant factor which predicted the outcome. The presence of sequelae is also high in these patients who did not die. So, in a patient who presents with altered sensorium, it can be confidently predicted that the chances of both morbidity and mortality are high.

c. Neurological Deficit

Presence of neurological deficits such as weakness of limbs, cranial nerve palsies (mainly seen were facial and abducent nerve palsy) and increased tone were commonly noticed deficits at the time of presentation.

It is clear that a patient with these deficits on admission ended up either dead or with permanent neurological deficit. This may be due to increased proteins associated in these patients, which causes blockage and infact leading to permanent deficit or death.

d. Extra Cranial Associations

Any extra cranial association, be it tuberculous (pulmonary infection or lymphadenopathy) or non tuberculous (hepatitis or HIV or diabetes) did not correlate with the outcome of the disease. This is in par with study conducted by Meyers in 1982.

From above discussion it can be predicted comfortably that presence of neurological deficits and altered sensorium are important and significant factors in predicting the outcome of patients with post meningitic hydrocephalus. Again, presence of neurological deficit results in the patients having residual deficit than death whereas in a patient with altered sensorium the risk of mortality is high. The study also reinforces the fact that presence of extracranial associations does not alter the final outcome of the disease[5]. This is more apparent than real as this may be due to advancements in medical field in treating systemic diseases.

To know that seizures do not adversely affect the outcome is a relief as nearly 40% of the patients had seizures. This again coincides with the study conducted by Sunil Karande in 2003[12]. Therefore among clinical features relating to outcome the most predictive factor for sequelae is neurological deficit and for death is altered sensorium.

II. **CSF Study :**

From anova and multiple comparisons it can be concluded that increased proteins and reduced sugar, which means active infection, are associated with more deaths and sequelae.

Increased proteins alone is a significant indicator of increased morbidity. In these studies, sugar does not seem to contribute to prognostication.

Very high protein levels also significantly contribute to mortality levels. This shows that increased proteins in CSF indicate a more fulminant and ongoing meningitic process and therefore a more grave prognosis.

III. **CT Findings** :

In all patients post operative CT brain was done and was noted that there was a definite reduction in ventricular size. It can be contended that though there is no statistical significance in final outcome for patients with just hydrocephalus and those with associated basal exudates or periventricular lucency both of these independently do signify increased morbidity and mortality. This implies that other contributory factors in toto predict the outcome and based on CT brain alone it is improper to conclude outcome.

IV. **Age** :

Though it is generally believed that extremes of age group have higher morbidity and mortality, this study does not show a statistically significant association. To attribute this to improving neonatal and geriatric care does seem proper.

V. **Time between onset of meningitis and surgery** :

As per this study there is no definite correlation between the time of onset of the disease and surgery. It may be assumed that mortality and morbidity in a patient who has undergone shunt is dependent on a combination of factors than the presence of a single factor.

However when hard pressed for requirement of prognostic factors predicting the outcome of a patient with post meningitic hydrocephalus it can be safely said that the most predictive factor is presence of altered sensorium on admission combined with increased levels of proteins in CSF. Here again, the higher the protein level, higher is the morbidity followed by mortality.

On comparison of this study with that of a study conducted on 70 patients by Oh Sh, Choi Ju of Korea in 1983[23]. The following is observed.

The commonest clinical feature in their study was vomiting while in this study was headache. Vomiting was seen in only 40% of patients in this study while it contributed to 67% in their study.

The commonest CT feature apart from ventricular dilatation was basal exudates in their study while it was periventricular lucency in this study. In this study 36 of 53 had periventricular lucency and 13 had basal exudates.

In both the studies mortality was around 20%.

CONCLUSION

1. The severity of the inflammatory process associated with meningitic hydrocephalus largely influenced the outcome after ventriculo-peritoneal shunt rather than hydrocephalus per se.

2. Among the clinical signs and symptoms that were considered to predict outcome, the most predictive factor was altered sensorium in a patient at the time of admission.

3. Factors such as seizures or other associated diseases were not significant in predicting the outcome of post meningitic hydrocephalus.

4. On analyzing the values of protein and sugar in CSF, it can be concluded that protein values are better predictors of outcome than sugar. More over it is also seen that, morbidity and mortality increases with increasing CSF protein.

5. The presence of periventricular lucency and basal exudates predict worse prognosis.

6. In this study neither the extremes of age nor the increased time interval between onset of disease and surgery prove to be statistically significant in predicting the outcome.

7. Among the factors that predict the outcome, altered sensorium and increased protein values in CSF proved to be the most significant. They portend a worse prognosis for the patients.

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PROFORMA

POST MENINGITIE HYDROCEPHALUS – FACTORS PREDICTING OUTCOME

NAME: AGE: IP NO:

ADDRESS: DOA:

DOD:

DOS:

COMPLAINTS :

HISTORY:

EXAMINATION:

CT SCAN BRAIN:

CSF STUDY:

SURGERY:

POST OP PERIOD:

Post OP CT Brain:

COD:

FOLLOW UP AFTER 12 WEEKS:

MASTER CHART

S. No.	Age	Clinical signs and symptoms					CSF Study		Computer Tomographic Findings				Shunt	Post OP CT*	Time Interval	Outcome		
		Fever	Fits	FND	Altered Sensorium / Vomiting	Extra cranial Assoc	Protein	Sugar	Hydrocephalus	Basal exudates	Parenchymal changes	PVL				Improved	Sequelae	Death
1.	2 m	+	-	-	V	A	45	65	P	A	-	P	+	+	10d	+	-	-
2.	3 ½y	+	+	-	-	A	135	40	P	A	-	P	+	+	20d	-	-	+
3.	3y	+	+	N	-	A	255	60	P	P	-	P	+	+	15d	-	-	+
4.	16 y	+	-	-	Al.S	A	35	65	P	A	-	A	+	+	3d	+	-	-
5.	3 y	+	-	-	-	A	190	45	P	P	-	P	+	+	13d	+	-	-
6.	3 m	+	-	-	-	A	55	40	P	A	-	P	+	+	1m	-	+	-
7.	33 y	+	-	-	V	A	45	65	P	A	-	A	+	+	15d	-	-	+
8.	21 y	+	+	-	V	T	95	35	P	A	-	A	+	+	3w	+	-	-
9.	25 y	+	-	N	-	T	260	30	P	P	-	P	+	+	2m	-	+	-
10.	35 y	+	+	-	Al.S	NT	450	30	P	A	-	A	+	+	5m	-	-	+
11.	4 y	+	-	N	-	A	86	32	P	A	-	P	+	+	3w	-	+	-
12.	2 y	+	+	-	V	A	65	35	P	A	-	P	+	+	15d	+	-	-
13.	55 y	+	-	-	-	A	95	40	P	A	+	A	+	+	18d	+	-	-
14.	16 y	+	-	-	Al.S	A	175	25	P	A	+	A	+	+	3m	+	-	-
15.	15 y	+	-	N	V	A	96	35	P	P	-	P	+	+	15d	-	-	+
16.	3 m	+	+	-	-	NT	40	65	P	A	-	A	+	+	2m	+	-	-
17.	35 y	+	+	-	V	A	35	55	P	P	-	P	+	+	1m	+	-	-
18.	1½ y	+	+	-	Al.S	A	220	25	P	A	-	A	+	+	15d	-	-	+
19.	30 y	+	-	N	-	T	88	20	P	A	-	P	+	+	2w	-	-	+
20.	13 y	+	-	-	Al.S	A	55	40	P	A	-	P	+	+	4m	+	-	-
21.	9 y	+	-	N	Al.S	A	195	35	P	P	-	P	+	+	10d	-	-	+
22.	66 y	+	-	-	-	A	35	75	P	A	-	P	+	+	1w	-	-	+
23.	3 y	+	+	-	Al.S	A	40	55	P	A	-	A	+	+	1w	+	-	-
24.	2 y	+	-	N	AlS	A	210	40	P	A	-	P	+	+	10d	-	-	+
25.	2½ y	+	-	N	Al.S	A	255	25	P	A	-	P	+	+	15d	-	+	-
26.	6 y	+	-	N	-	A	285	30	P	A	-	P	+	+	3m	-	+	-
27.	10y	+	-	N	V	A	35	55	P	A	-	A	+	+	1w	+	-	-

S.No.	Age	Clinical signs and symptoms					CSF Study		Computer Tomographic Findings				Shunt	Post OP CT*	Time Interval	Outcome		
		Fever	Fits	FND	Altered Sensorium	Extra cranial Assoc	Protein	Sugar	Hydrocephalus	Basal exudates	Parenchymal changes	PVL				Improved	Sequelae	Death
28.	38y	+	-	N	V	A	55	30	P	A	-	A	+	+	10d	+	-	-
29.	7y	+	+	N	-	A	40	65	P	A	-	P	+	+	1m	-	+	-
30.	25d	+	-	-	-	NT	30	55	P	A	-	P	+	+	3m	-	+	-
31.	1y	+	-	-	V	A	55	35	P	A	-	P	+	+	1m	+	-	-
32.	35y	+	-	-	-	A	35	65	P	A	-	P	+	+	10d	+	-	-
33.	26y	+	-	N	-	T	98	30	P	P	-	P	+	+	15d	+	-	-
34.	5m	+	+	-	-	A	40	55	P	A	-	P	+	+	1m	+	-	-
35.	17y	+	-	N	-	A	30	55	P	P	-	A	+	+	5d	-	+	-
36.	63y	+	-	-	V	T	30	55	P	A	-	A	+	+	10d	+	-	-
37.	9m	+	+	N	-	A	30	50	P	A	-	P	+	+	1m	-	+	-
38.	5m	+	-	-	V	A	40	65	P	A	-	P	+	+	10d	+	-	-
39.	4y	+	+	-	V	A	85	30	P	A	-	P	+	+	11/2m	+	-	-
40.	11y	+	-	N	V	A	55	40	P	P	-	P	+	+	2m	-	+	-
41.	24y	+	-	N	-	T	95	35	P	P	-	A	+	+	5d	+	-	-
42.	16y	+	-	N	-	T&NT	295	20	P	A	+	A	+	+	3m	-	+	-
43.	5y	+	+	N	-	A	95	30	P	A	-	P	+	+	2m	-	+	-
44.	45y	+	+	-	-	T	85	25	P	A	+	A	+	+	2m	+	-	-
45.	2y	+	+	N	V	A	195	25	P	P	-	P	+	+	1m	-	+	-
46.	13y	+	-	N	Al.S	A	93	40	P	A	-	P	+	+	15d	+	-	-
47.	11m	+	+	-	Al.S	A	40	65	P	A	-	A	+	+	15d	+	-	-
48.	7y	+	+	N	V	A	238	35	P	P	+	P	+	+	3m	-	-	+
49.	25d	+	+	-	-	A	40	60	P	A	-	P	+	+	5d	-	-	+
50.	9y	+	-	-	-	A	35	55	P	A	-	P	+	+	20d	+	-	-
51.	4y	+	+	N	Al.S	A	95	30	P	P	-	P	+	+	10d	-	+	-
52.	62y	+	-	-	Al.S	A	40	65	P	A	-	P	+	+	10d	+	-	-
53.	15y	+	-	-	-	A	55	35	P	A	-	P	+	+	20d	+	-	-

Key

+/P = Present ,-/A = Absent, N = Presence of neurological deficit, V = Vomiting, Al.S = Altered sensoriam, T= Extracranial tuberculosis, NT = Non tuberculous aetiology. * - Size of ventricles in post operative CT was measured + denoted a decrease in size.