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Neurodevelopmental Outcome at 12 months in Preterm Infants with Post-Haemorrhagic Ventriculomegaly, Hydrocephalus and Periventricular Echodensities.

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ABSTRACT

Objective: To determine the neurodevelopmental outcome at 12 months of age in four different groups of infants with itraventricular hemorrhage.
Materials and Methods: Prospective study of 118 inborn preterm babies divided into four groups. Group I: SEH/ IVH without ventriculomegaly; group II: IVH with ventriculomegaly; groupIII: IVH hydrocephalus; group IV: with parenchymal echodensities. The four groups were matched for gestational age, weight, antecedent events of haemorrhage. Follow-up assessment was done at a 12 months corrected age and on a monthly basis for assessment of growth and development.
Results: All infants were below 32.5 weeks gestation. The ventriculomegaly group (N=30) was divided into; mild (16.6%), moderate (53.3%), and severe (30%). The hydrocephalus group (N=29) was divided into; progressive (17.8%), stationary (50.3%), and decreased (32.1%). Twelve out of 28 children with parenchymal echodensities showed severe head circumference reduction at one year of age. Chi-square was used as statistical analysis and reveals a significant increase in neurological abnormality and developmental delay at 12 months in group IV (p value<0.0001).

Conclusion: Neuromotor outcome is more a function of parenchymal damage than of ventriculomegaly per se and hydrocephalus following hemorrhage should be managed aggressively to prevent severe neurological damage

INTRODUCTION

Intraventricular Haemorrhage in preterm infants leads to serious complications of progressive ventricular dilatation, hydrocephalus and parenchymal damage. The post-haemorrhagic ventricular dilatation results from obstruction of the cerebrospinal fluid pathways and arachnoid villi, by multiple small clots. Hydrocephalus may also result later by basal cistena arachnoiditis producing permanent obstruction (1). Real time ultrasound measurements of ventricular width can be easily monitored and centile values are available relating this to gestational age (2). Ventriculomegaly produces stretching of the axons in the periventricular area and causes neurological damage. Progressive ventricular dilatation and head enlargement is usually accompanied by some increase in cerebrospinal fluid pressure, although the upper limit of normal (0.8 kp or 6mm Hg) is only slightly exceeded in some affected infants (3). Increased cerebrospinal fluid pressure may cause periventricular oedema, distortion of developing pathways and decreased cerebral perfusion; many of the affected infants may however, have already sustained cerebral hypoxic ischaemic damage in the perinatal period, which is likely to result in later impairments.

Unilateral periventricular echogenicities, corresponding to the side in which the intraventricular haemorrhage is greater, are considered to be extensions of intraventricular haemorrhage and can result in cerebral parenchymal damage. The principal outcome measures are the neurodevelopmental outcome with a specific type of complication. We report here the neurodevelopmental outcomes at 12 months of age in four different groups of infants with intraventricular haemorrhage and its various complications.

MATERIALS AND METHODS

A total of 118 inborn preterm babies, admitted into the neonatal I.C.U. and followed up in the preterm clinic of our department, were registered for the study. The neonates were divided into four groups. Group 1 with SHE/IVH without ventriculomegaly; Group II IVH with ventriculomegaly, Group III with IVH hydrocephalus and Group IV with parenchymal echodensities. The four groups were matched for gestational age, weight, antecedent events of haemorrhage, namely RDS requiring assisted ventilation, and the occurrence of patent ductus arteriosus. Serial cranial ultrasound examinations were performed in the unit and when indicated as an out-patient procedure. The ventriculomegaly was classified as mild, moderate and severe and the specific area of dilatation was recorded. Hydrocephalus was diagnosed if there was a rapid increase in head circumference of more than 2cm/week or if signs of increased intracranial pressure were present. It was classified as acute, subacute and late onset. Parenchymal echodensities were classified as anterior, middle and posterior and further classified as small and large.

All the babies were studied longitudinally in the special preterm follow up clinic where neuromotor assessment was done according to prepared protocols. Development was assessed by modified Griffiths scales and a score of <70 was considered as delayed. Follow-up assessment was done at a 12 month corrected age for final results and on a monthly basis for assessment of growth and development.

The growth parameters, including head circumference were measured using growth records for infants in relation to gestational age, foetal and infant names for combined sexes. Vision and hearing were assessed clinically to ensure that the child could participate in the developmental assessment. The outcome was summarized as head circumference, defective hand control, axial balance defective, hypotonia or hypertonia of one or more limbs.

The final comparison between the four groups was recorded as neuromotor abnormality (one of the above signs) alone, neuromotor and visual abnormality and neuromotor and developmental delay. Global abnormality was recorded as neuromotor + auditory + visual + DR < 70.

RESULTS

A total of 118 preterm infants were assessed at a corrected age of 12 months. Table 1 shows the clinical characteristics of all the infants enrolled for the study. The lowest mean weight was 1250 gms and highest was 1620 gm. All the babies were below 32.5 weeks gestation. Associated factors of IRDS were found in 65 % of the infants of Group I and 92 % of those of Group II.

The characteristics of study groups with their descriptions are shown in Table 2 and also the difference between ventriculomegaly and hydrocephalus.

Table 3 gives the details regarding the ventriculomegaly group with respect to severity and the specific site and time of occurrence of the ventriculomegaly. In 53.3% (16 patients) the ventriculomegaly was noticed within the first week and in 6.7% after 4 weeks. Concerning the head circumference in this group, 25/30 were in the range of mean + ISD, 4 had mean + 2SD and 1 was less than 1SD. Four had neuromotor abnormalities. But in Group II 25 had an abnormal head circumference at 1 year of age and the visual and auditory abnormalities were increased in 10/30. Group III, consisting of 28 children, showed 42.8% and developed hydrocephalus between 1 week and 4 weeks of age and 32.74% at more than 4 weeks of age (Table 4). The hydrocephalus was stationary in 50.3%. Eleven of the hydrocephalus group had management in the form of repeated lumbar puncture and 9 had pharmacologic management with furesemide and diamox. Table 5 illustrates the site of the parenchymal echodensities and also the evolution of the echodensities. Table 6 gives the neuromotor and developmental outcome of various groups.

Twelve out of 28 children with parenchymal echodensities showed significant reduction in head circumference at one year of age, which corresponded to the delayed brain growth or brain atrophy found in these infants. Seventeen of the twenty-eight had severe neuromotor abnormality and 25/28 had neuromotor and a delayed developmental quotient; 25/30 had global delay.

Table 7 shows the comparison of Groups III & IV using Chi-square analysis which reveals a significant increase in neurological abnormality and developmental delay at 12 months in Group IV (p value < 0.00001).

DISCUSSION

The pathogenesis of brain injury secondary to ventricular dilatation, hydrocephalus and periventricular haemorrhage is multifold (10).

In grade I haemorrhage it is associated with destruction of glial precursors in the germinal matrix. Significant neuromotor abnormality was found only in 1/32 infants with haemorrhage alone. This was in the form of hypertonia, and developmental assessment was normal at 12 months.

Ventriculomegaly causes axonal stretching and is known to result in destruction of periventricular white matter. The destruction of white matter may result in neuromotor and visual or auditory impairments.

In our study, 13% with ventriculomegaly alone had neuromotor abnormality. Hydrocephalus can cause decreased cerebral perfusion pressure, compression of periventricular white matter and cerebral arteries. Hence the neuromotor and developmental deficits are more marked. In our series the incidence of visual abnormalities was very marked in this group - 10/28 (36%) - and global abnormality was again 36%. More than half of these infants had early drainage of CSF with repeated lumbar punctures. As expected, the presence of a parenchymal lesion was associated with a poorer long term outcome and nearly all these children had neuromotorimpairments (Table 7). These lesions due to secondary venous infarction, are fairly localized and lead not only to predominantly neuromotor sequalae but also to auditory and visual defects and global developmental delay.

By analysis of the four groups we conclude that the neuromotor outcome is more a function of parenchymal damage than of ventriculomegaly per se and that hydrocephalus following haemorrhage should be managed aggressively to prevent extensive neurological damage.

Table 1: Advances in the etiology and prevention of preeclampsia

Mean Weight	**Group I 620 gms**	**GroupII 1250 gms**	**Group III 1310 gms**	**Group IV 1350 gms**
Mean Gestational Age	32.1 wks	31.6 wks	31.1 wks	30.16 wks
IRDS Ductus	65%	68%	80%	92%
Mean Weight	3%	10%	12%	25%

Table 2: Characteristics of study groups

SHE/IVH without Ventriculomegaly	Group I
IVH with Ventriculomegaly	Group II
IVH with Ventriculomegaly & Hydrocephalus	Group III
Parenchymal Echodensities	Group IV
Ventriculomegaly: Ventricular dilatation alone. Hydrocephalus: Progressive Ventricular Dilatation Head Circumference > 2cms/wk. Evidence of Increased Intracranial Pressure	

Table 3: Ventriculomegaly (N=30)

Mild	16.6%	
Moderate	53.3%	
Severe	30%	
	Right	Left
a. Frontal Horn	40%	40%
b. Body	72.8%	70.4%
c. Occipital Horn	64.2%	59.3%
d. Temporal Horn	11.0%	9.2%
Third Ventricle	23.4%	

Fourth Ventricle	04.2%	
Cisterns	13.3%	
Timing of Ventriculomegaly		
< 1 Week	53.3%	
1 Week - 4 Wks	40%	
> 4 Weeks	6.7%	

Table 4: Hydrocephalus (N = 29)

Timing of Hydrocephalus	
< 1 Week of Age	21.42%
1 Week and 4 Wks. of Age	42.84%
> 4 Weeks of Age	35.74%
Natural History of hydrocephalus	
Progressive	17.8% (5)
Stationary	50.3% (14)
Decreased	32.1% (9)
Management	
Repeated L.P.	11
V.P. Shunt	2
Ventriculostomy	1
Pharmacologic	9

Table 5: Evolution of Parenchymal Echodensity

Intraventricular HMGE	Birth to 72 Hours
Periventricular Echodensities	Birth to 72 Hours
Periventricular Leucomalacia	CA of 7 - 8 Wks.
Location of Echodensities	
Anterior	16/28 = 57.14 %
Middle	11/28 = 39.3 %
Posterior	1/28 = 3.6 %

Table 6: Neuromotor and Developmental Outcome At 12 Months

Clinical Signs & Assignment	**Group I n = 32**	**Group II n = 30**	**Group III n = 28**	**Group IV n = 28**
1. Head Circumference at				
- 1 year of age > 1 SD	-	4	25	7
- Mean + 1 SD	32	25	3	8
- < SD	-	1	-	12
2. Suck & Swallow disability	-	-	2	5
3. Defective Visual & Fixing and following	-	1	2	12
4. Squint	-	2	8	15
5. Nystagmus	-	-	1	1

6. Hearing Defect	-	1	2	8
7. Defective Head Control	-	2	4	8
8. Axial Balance	-	0	4	13
9. Hypotonia of 1 or more limbs	-	4	2	2
10. Hypertonia of 1 or more limbs	1	4	7	15
11. Neuromotor Abnormality	1	1	9	17
12. Neuromotor + Visual	-	5	10	16
13. Neuromotor + DR < 70	-	-	8	25
14. Neuromotor + Auditory	-	-	2	8
15. Neuromotor + Auditory + Visual + Dr < 50	-		15	25

Table 7: comparison of group II & IV

	Group II	**Group IV**
Neuromotor Abnormality	9/28 (32.1 %)	20/28 (71.4 %)
NM + V + ADR < 70	10/28 (35.6 %)	26/28 (92.9 %)
X2 (Chi Square for analysis of proportions Sig P Value <.0001)		

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