Acta Paediatrica Hungarica, 26 (4), pp. 281—287 (1985)

Neonatal Ventricular Haemorrhage: Treatment by Puncture

G Simon

Department of Paediatrics, County Hospital, Székesfehérvár, Hungary

Transfontanellar ventricular puncture and intermittent lumbar punctures were carried out in 10 patients affected by intraventricular haemorrhage during the period between 1980 and 1983. In only three of them was later a shunt necessary for treatment of hydrocephalus. Six children developed normally, in two severe mental retardation ensued, and two died later, one of heart failure after cardiac surgery and one of ventriculitis following shunt implantation. Puncture therapy can be recommended for intraventricular haemorrhage of the neonate.

The most frequent and most severe form of intracranial haemorrhage in the neonatal period is periventricular and/or intraventricular haemorrhage (IVH). The disorder plays an important role in perinatal morbidity and mortality [17]. In recent years several reviews on the pathology and aetiology of IVH have been published [2, 6, 7, 8, 21, 23] and also an excellent summary of the clinicopathological findings [17]. The affected babies are mostly premature but the disorder may also occur in mature newborns [5, 9, 16]. Follow-up of the survivors [13] has shown that a favourable outcome can be expected in cases affected by IVH of stages I or II according to the classification of Papile et al. [18]. The advent of sonography and computer tomography allowed to diagnose the condition in vivo and a more exact follow-up of the haemorrhage itself [1, 3, 14, 18].

Few reports deal with the therapy [15, 19, 24]. There is almost general agreement that there is no effective treatment, the overwhelming majority of patients dies, and the survivors are severely damaged.

Continuous drainage of IVH was based on the idea that blood in the CSF plugs the resorptive surface, thus resulting in intracranial hypertension after the disappearance of blood from the CSF. This in turn leads to further ventricular dilatation and brain damage. Removal of blood by drainage prevents obstruction of the resorptive surfaces and its consequences [19]. This has been supported by animal experiments [20]. Mantovani et al. [15] found that in cases of IVH daily lumbar puncture is not a sufficient method for prevention of hydrocephalus.

Material and Method

Between January 1, 1980, and December 31, 1983, transfontanellar ventricle puncture was performed in 18 instances in ten

TABLE I

Serial number of patient	Gestational age, weeks, Gender	Birth weight, g	Course of pregnancy and delivery	Apgar score	Postnatal events	Cerebrospinal fluid
1	41 M	3600	25 yrs, parity: 2, oedema, precipitated delivery	2/3/5	Resuscitation, spontaneous respiration after 10 minutes, 9 hours later generalized fits, sustained inconsciousness	Lumbar and ventricular puncture at age of 12 hours: massive blood. Two ventricular punctures, 3 lumbar punctures
2	32 M	1550	28 yrs, parity: 2, breech delivery (Bracht extraction)	3/4	Resuscitation, spontaneous respiration after 5 minutes. Repetitive apnoeic spells, generalised muscle hypotension, fontanellar bulging	Lumbar and ventricular puncture at 36 hours. Blood containing fluid. Total protein: 2.2 g/l. One ventricular puncture, 3 lumbar punctures
3	41 F	2380	17 years, primipara, during pregnancy Henoch-Schönlein purpura, meconium containing amnial fluid	8/9	Clonic fits on extremities at 36 hrs. At 48 hours: bulging fontanelle, right palpebral ptosis, repetitive apnoeic spells	Ventricular punctures at 48 h, dark-yellow fluid, total protein: 1.7 g/l. One ventricular puncture, 3 lumbar punctures
4	33 F	1660	37 yrs, parity: 3, EPH gestosis, fragmented, defective placenta	6/7	36 h: spactic muscles, opisthotonus, ocular symptoms	Lumbar and ventricular puncture at 48 hrs, blood containing CSF, total protein: 1.94 g/l. Two ventricular and 2 lumbar punctures
5	41 M	3950	25 yrs, primipara, 2/3 earlier imminent abortion	2/3	Admitted from another ward at 4 h. Spastic muscles, opisthotonus, ocular symptoms, small fontanelle	Lumbar and ventricular puncture at 12 hours, blood in CSF. Total protein: 1.21 g/l. 3 ventricular and 3 lumbar punctures
6	41 M	4130	33 yrs, parity: 2. Protracted expulsion phase	4/7/8	24 h: irritability, spastic muscles, generalized fits, ocular symptoms, small fontanelle	Lumbar and ventricular puncture at 48 hrs, blood containing CSF. Total protein; 5.1 g/l. Tvo ventricular, seven lumbar punctures

7	40 M	3620	33 yrs, parity: 3, cephalic presentation, uneventful delivery	9/10	After 48 h repetitive apnoeic spells, spastic muscles	Lumbar and/ventricular puncture at 72 h, blood containing CSF. Total protein: 2.94 g/l. Two ventricular and 8 lumbar punctures
8	36 M	2740	22 yrs, primipara, hypertension, EPH gestosis, protracted extrusion phase	6/9	22 h: apnoeic spells, muscular hypotension	Lumbar and ventricular puncture at 36. Total protein 7.42 g/l. Two ventricular and 5 lumbar punctures
9	36 M	2750	26 yrs, parity: 3, cephalic presentation, uneventful delivery	9/10	24 h: irritability, apnoeic spells, partial exchange transfusion for hyperviscosity, 72 h: bulging fontanelle	Lumbar and ventricular puncture at 72 h, blood in CSF, total protein: 2,94 g/l, two ventricular and 5 lumbar punctures
10	41 M	3190	27 yrs, primipara, protracted expulsion phase, meconium stained amnial fluid	6/9	8 h: pallour, bradycardia, 24 h: ocular symptoms, spastic musculature, generalized fits	48 h: lumbar and ventricular puncture. Total protein: 2.02 g/l. One ventricular and 4 lumbar punctures

patients affected by IVH. Table I sums up the most important data of the perinatal period. The diagnosis was based on clinical symptoms which were not very characteristic and had much in common with those of hypoxaemic and ischaemic cerebral damage, on sonography and CSF cytology, the latter to exclude artificial bleeding. In addition, severity of symptoms (increasing intracranial pressure, convulsions, unconsciousness) was considered in indicating ventricular puncture.

The puncture was performed after diazepam pretreatment under sterile conditions, using a lumbar cannula. First the subdural space was punctured, then the needle was directed towards the frontal horn of the lateral ventricle. In none of the ten cases reported here was there a subdural haemorrhage, in nine cases the ventricular fluid contained macroscopic blood, in one patient it had a deep yellow colour. In order to avoid sudden volume reduction in the ventricles, the ventricular space was rediluted by injecting body-warm physiological saline; this also enhanced removal of cellular elements from the CSF. In patients in whom clearance of the fluid could not be achieved, puncture was performed on the opposite side 24 hours later. This treatment was supplemented by lumbar punctures carried out every day or every second day. The following drugs were used: synthetic vitamin K_1, vitamin C, rutin, calcium gluconate, oestriol disodium succinate, furosemide and phenobarbital and, for preventing infections, antiobiotics were applied. The patients received parenteral nutrition until consciousness had been regained. In nine patients leaving the department, neurohabilitation after Katona's method [10, 11, 12] was introduced.

Results

There are several methods for determining the time of onset of haemorrhage [3, 4, 22]. In our cases the diagnosis was established in two patients within 24 hours of age, in six between 24—48 hours, and in two in 72 hours. Protracted clearing up of the CSF was seen in four patients (Nos 6, 7, 8 and 9); in three of these (Nos 6, 7 and 8) implantation of a ventricular shunt was necessary. In the six remaining patients the CSF became clear gradually, there was no sign of rebleeding. The outcome of our cases in May, 1984, is demonstrated in Table II.

Discussion

A large proportion of patients affected by neonatal intracranial, periventricular or intraventricular haemorrhage dies and many of the survivors exhibit brain damage. Early diagnosis can be achieved by sonography. Severe clinical symptoms, signs of increased intracranial pressure and blood in the lumbar CSF indicate a transfontanellar puncture of the lateral ventricle. Removal of the blood, prevention of obstruction of the resorptive surface and of sustained intracranial hypertension reduces the mortality and improves the quality of life of the survivors.

Since transfer of the newborn affected by acute haemorrhage is not recommended, the treatment must be carried out in the local pathological newborn ward. A few days after onset of intraventricular haemorrhage, when transfer is already possible, initiation of treatment is too late and the results are disappointing.

TABLE II

Serial number of patient	Age at checking, months	Hydro-cephalus	Shunt	Motor handicap	Epilepsy	Other handicaps	Mental development	Note
1	42	—	—	spastic tetraplegia	BNS 4 mo	—	Severely retarded	Uneducable
2	36	—	—	—	—	hypermotility, disordered sleep	Coherent speech	
3	36	—	—	—	—	convergent squint	adequate for age	Wears glasses
4	36	—	—	—	—	—	adequate for age	
5	10	—	—	—	—			Dropped out, emigrated
6	12	+	+	retarded motor development	—	—	retarded	Checked in National Institute of Neurosurgery
7	12	+	+	retarded motor development		secondary generalised	slight retardation	12 months: CT, shunt, 3 mo later death of ventriculitis
8	12	+	+	—	—	—	adequate for age	
9	6	—	—	motor and generalised muscular hypotension	—	transposition of great arteries	slightly retarded	6 mo: death after cardiac surgery
10	10	—	—	—	—	convergent squint	adequate for age	

We did not encounter convulsions, apnoea, increase of bleeding tendency or infection during puncture treatment.

Because of the small number of cases and the lack of a control group, valid conclusions cannot be drawn from our experience, but it appears encouraging against the pessimistic approach to therapy of intraventricular haemorrhage: 6 out of 10 patients are intact survivors.

Acknowledgments

I am indebted to Dr. K. Lőrinczi for permission to perform this study in his department, to Dr. A. Guseo (County Hospital, Székesfehérvár) for cytological studies; to Dr. F. Katona (Institute of Developmental Neurology, Budapest), and to Dr. E. Paraicz (Institute of Neurosurgery, Budapest), for valuable help and suggestions.

References

1. Allan WC, Roveto CA, Sawyer LR, Courtney SE: Sector scan ultrasound imaging through the anterior fontanelle. Am J Dis Child 134: 1028, 1980
2. Cole VA, Durbin GM, Olaffson A, Reynolds EOR, Rivers RPA, Smith JF: Pathogenesis of intraventricular haemorrhage in newborn infants. Arch Dis Child 49: 722, 1974
3. Dolfin T, Shidmore MB, Fong KW, Hoskins EM, Sheunan AT: Incidence, severity and timing of subependymal and intraventricular haemorrhages in preterm infants born in a perinatal unit as detected by serial real-time ultrasound. Pediatrics 71: 541, 1983
4. Emerson P, Fujimura M, Howat P, Howes D, Keeling J, Robinson RO, Salisbury D, Tizard JPM: Timing of intraventricular haemorrhage. Arch Dis Child 52: 183, 1977.
5. Fenichel GM, Webster DL, Wong WKT: Intracranial hemorrhage in the term newborn. Arch Neurol 41: 31, 1984
6. Fujimura M, Salisbury DM, Robinson RO, Howat P, Emerson PM, Keeling JW, Tizard JPM: Clinical events relating to intraventricular haemorrhage in the newborn. Arch Dis Child 54: 409, 1979.
7. Godard J, Lewis RM, Armstrong DL, Zeller RS: Moderate, rapidly induced hypertension as a cause of intraventricular hemorrhage in the newborn beagle model. J Pediat 99: 1057, 1980
8. Goldberg RN, Chung D, Goldman SL, Bancalari E: The association of rapid volume expansion and intraventricular hemorrhage in the preterm infant. J Pediatr 99: 1060, 1980
9. Guekos-Thöni U, Boltshauer E, Willi UV: Intraventricular haemorrhage in fullterm neonates. Develop Med Child Neurol 24: 704, 1982
10. Katona F: Complex investigation of impaired brain function during the first postnatal month. Acta Paediatr Acad Sci Hung 22: 147, 1981
11. Katona F: Neue Methoden in der Säuglingsneurologie und Rehabilitation. Acta Paediatr Acad Sci Hung 15: 67, 1974
12. Katona F: Manual de prevención diagnostico y habilitación precoz de los daños cerebrales. Iamer, Madrid 1982
13. Krishnamoorthy KS, Shannon DC, DeLong GR, Todres ID, Davis KR: Neurologic sequelae in the survivors of neonatal intraventricular hemorrhage. J Pediatr 64: 233, 1979
14. Lemburg P, Brettschneider A, Storm W: Ultraschall zur Diagnostik morphologischer Hirnveränderungen bei Neugeborenen. Mschr Kinderheilk 129: 190, 1981
15. Mantovani JF, Pasternak JF, Mathew OP, Allan WC, Mills MT, Casper J, Volpe JJ: Failure of daily lumbar punctures to prevent the development of hydrocephalus following intraventricular hemorrhage. J Pediatr 97: 279, 1980
16. Palma PA, Miner ME, Morris FH: Intraventricular hemorrhage in the neonate born at term. Am J Dis Child 133: 941, 1979
17. Pape KE, Wigglesworth JS: Haemorrhage, Ischaemia and the Perinatal Brain. William Heinemann, London 1979
18. Papile LA, Burstein J, Burstein R, Koffler H: Incidence and evolution of subependymal and intraventricular hemorrhage. J Pediatr 92: 529, 1978

19. Paraicz E, Dobronyi I: Therapeutical possibilities of intraventricular hemorrhages at infancy. Proc Symposium on Brain Damage Budapest 1977
20. Pásztor A, Paraicz E, Pásztor E: Experimental approach and therapeutical possibilities of intraventricular hemorrhage in infancy. J Neurosurg Sci 22: 185, 1978
21. Simmons MA, Adcock EW, Bard H, Battaglia FC: Hypernatremia and intracranial hemorrhage in neonates. New Engl J Med 291: 6 1974,
22. Tsiantos A, Victorin LH, Relier JP, Dyer N, Sundell H, Brill AB, Stahlman MJ: Intracranial hemorrhage in the prematurely born infant: Timing of clots and evaluation of clinical signs and symptoms. J Pediatr 8: 854, 1974
23. Volpe JJ: Neonatal intraventricular hemorrhage. New Engl J Med 304: 886, 1981
24. Wise BE, Ballard R: Hydrocephalus secondary to intracranial hemorrhage in premature infant. Child's Brain 2: 234, 1976.

Received 5 February 1984

G Simon MD
County Hospital
H-8001 Székesfehérvár, Hungary