COMPUTED TOMOGRAPHY OF BRAIN IN SYMPTOMATIC BIRTH ASPHYXIA

G. Khanna
R.K. Kapoor
P.K. Misra
K.L. Srivastava
M.C. Pant
P.K. Srivastava

ABSTRACT

Twenty five asphyxiated newborns (seventeen term and eight preterm) with mean gestational age of 37 weeks (range 28-48 weeks) and mean birth weight of 2.4 kg (range 0.75 kg to 3.5 kg), respectively, constituted the cases in present study. Normal CT scan was found in five term (29.4%) and two preterm babies (25%). CT abnormalities noted in term babies included hemorrhage (subarachnoid 5.8%, intracerebral 11.6%), hypodensity (mild 23.2%, moderate 11.6% severe 5.8%); hypodensity with hemorrhage 5.8% and cerebral atrophy 5.8%. In the preterm babies abnormalities included intraventricular hemorrhage in 25%, isolated hypodensity in 37.5% and hypodensity with hemorrhage in 12.5% cases. Where as mild hypodensity on CT scan in the absence of hemorrhage or other gross abnormality was indicative of a favourable outcome, moderate to severe hypodensity was indicative of ischemic brain injury and an unfavourable outcome. The presence of intraventricular hemorrhage irrespective of the size of bleed was associated with mortality in all in the present study.

Key words: *Newborn, Asphyxia, Hemorrhage, Computed tomography.*

Asphyxia is the single most important perinatal cause of brain damage in term infant(1). In an attempt to determine the extent of cerebral injury and to predict the immediate and long term outcome, a number of clinical and laboratory efforts have been made. The much utilized Apgar score is an unreliable screening test for predicting cerebral damage, as it is a highly subjective parameter, is not always recorded accurately and may be influenced by maternal sedation, trauma or rarely neuromuscular disease(1). Computed tomography is now accepted as routine diagnostic procedure in conjunction with neurological evaluation to detect brain damage in neonates(2).

The present study was undertaken to evaluate computerized tomographic scan changes in asphyxiated newborns to correlate CT scan changes with clinical severity of asphyxia and the neonatal outcome.

Material and Methods

The present study was conducted in 25 newborns babies either delivered at Queen Mary's Hospital, Lucknow or admitted to neonatal Unit, Children's Hospital of Gandhi Memorial and Associated Hospitals, Lucknow. Gestational age of the neonates based on Dubowitz varied from 28-40 weeks. The neonates included in the study group fulfilied any one of the following criteria: (*i*) Those needing resuscitation at birth, (*ii*) Meconium (except in breech delivery), (*iii*) Those presenting with signs

From the Departments of Pediatrics and Radiology, K.G. Medical College, Lucknow.

Reprint requests: Dr. (Mrs.) P.K. Misra, Professor and Head, Department of Pediatrics, K.G. Medical College, Lucknow 226 003.

Received for publication February 19, 1990; Accepted May 6, 1991

and symptoms of hypoxic ischemic encephalopathy (HIE) following perinatal asphyxia within 72 h of birth based on Sarnat's criteria(3). Patients with congenital infections or congenital neurological abnormalities were excluded from the present study.

Neonates were assessed and graded clinically into three stages of hypoxic-ischemic encephalopathy on the basis of modified Sarnat staging system(3).

The patients with symptomatic asphyxia were scanned in SCT-2000 TII Simadzue of third generation within 2 weeks of birth. The neonates were sedated with oral triclofos sodium (20-30 mg/kg) or injection phenobarbitone (10 mg/kg) IM ½-1 hour before the scan. Precautions were taken to prevent hypothermia by covering the babies in warm clothes and blanket.

Each infant was scanned in infant mode in the axial cuts and in limited cases, when needed, coronal cuts were also taken. Plain scans were done in most of the neonates except in few, in whom contrast material (Sodium lothamate 300 mg/kg) was given so as to visualize the areas of hemorrhage in a better way.

Areas of increased density (>60 HU) (Hounsefield unit) were considered to represent hemorrhage and were further classified according to their location into subdural, subarachnoid, intraventricular or intracerebral hemorrhage. On the other hand, hypodense area (<25 HU) assumed to represent cerebral edema were graded as mild, moderate or severe edema(4). Ventricular size was measured and classified into 3 grades according to lateral ventricular ratio(4).

The immediate postnatal outcome of the neonates was classified into

(*a*) Favourable *i.e.,* neonates who survived from acute hypoxic insult and were discharged irrespective of the neurological status.

(*b*) Unfavourable which included neonates dying in the immediate post natal period.

Results

Twenty five asphyxiated newborns constituted the present study of which seventeen were term and eight were preterm. The mean gestational age and birth weight of the neonates in the study group was 37 weeks (range 28-40 weeks) and 2.4 kg (range 0.750-3.5 kg), respectively. Twenty neonates were delivered vaginally (16 vertex and 4 breech presentation). One of these vaginally delivered baby required forceps application. The remaining five cases were delivered by LSCS (all emergency).

Eleven of the twenty five asphyxiated babies expired, of these seven were term and four preterm. Two newborns died of pygonic meningitis while one premature weighing 0.750 kg died of extreme prematurity and septicemia.

All 25 babies were scanned within 15 days of birth (except 2 in whom CT scan was done on 20th and 38th day of life). The mean age of neonates at the time of scan was 7 days (range 1-38 day). CT changes in term and preterm babies are shown in *Table I*.

It is observed that seven (28%) of the neonates had normal CT scan, while various types of hemorrhage alone were found in five babies and in two the hemorrhages were combined with hypodensity. The intraventricular hemorrhage (IVH) in two patients was small and in only one of the patients larger IVH was present, associated with secondary subarachnoid leak. Of the patients showing severe hypodensity, one had associated compressed ventricles,

TABLE I—*CT Changes in Term and Preterm Babies*

CT Findings	Term babies (n=17)	Preterm babies (n=8)
1. Normal	5 (29.4)	2 (25.0)
2. Hemorrhage		
Subarachnoid (Primary)	1 (5.8)	–
Intraventricular	–	1 (12.5)
Intraventricular + Secondary subarachnoid	–	1 (12.5)
Intracerebral	2 (11.6)	–
3. Hypodensity		
Mild	4 (23.3)	2 (25.0)
Moderate	2 (11.6)	1 (12.5)
Severe	1 (5.8)	–
4. Moderate hypodensity + hemorrhage	1 (5.8)	–
Severe hypodensity + Hemorrhage	–	1 (12.5)
5. Cerebral atrophy	1 (5.8)	–

Figures in parentheses denote percentages.

the other patients had severe hypodensity along with dilatation of ventricles which was secondary to the small intraventricular bleed seen in the lateral and third ventricles that had probably caused obstruction to CSF flow (the scan in this child was done on the 20th day of life). One infant with severe asphyxia who was scanned on day 38 showed focal cerebral atrophy.

On comparing the CT findings in term neonates with those in preterm babies, it is observed that intraventricular hemorrhage was seen only in preterm babies whereas intraparenchymal hemorrhage was commonly observed in term babies.

The comparative CT findings in patients with or without convulsions are shown in *Table II*.

It was observed that 8 of the 11 patients who were free of seizures also had near normal CT scan (mild hypodensity/normal CT) while 9 of 14 patients with convulsions had major CT abnormalities either in the form of hemorrhage of moderate/severe hypodensity/cortical atrophy.

Table III depicts the comparative study of clinical staging, CT findings and the neonatal outcome in the asphyxiated babies. It was observed that 5 of the 6 neonates in stage I HIE had near normal CT scan and all 6 were discharged while all 3 patients in Stage III HIE had abnormal CT findings and all expired. While in Stage II despite abnormal CT findings in 3 neonates only 1 expired. One child who was clinically graded into stage I HIE had evidence of hemorrhage on CT scan and yet survived. This child had shown primary subarachnoid hemorrhage which had produced neither any clinical manifestation nor did it affect the neonatal outcome adversely. The neonates belonging to Stage II HIE who showed evidence of hemorrhage were having supratentorial intracerebral

TABLE II—*Comparative CT Findings in Patients with or without Convulsions*

Convulsions	n	CT findings						
		Normal	Hemorrhage	Hypodensity			Hemorrhage Hypodensity	Cortical atrophy
				Mild	Moderate	Severe		
Present	14	4	4	1	1	1	2	1
Absent	11	3	1	5	2	—	—	—

TABLE III—*Comparison Between Sarnat Staging CT Changes and Outcome of Neonates*

HIE stage (n = 17)	n	CT findings							Outcome	
		Normal hypodensity	Mild hypodensity	Moderate hypodensity	Severe hypodensity	Hemorrhage	Edema + hemorrhage	Cortical atrophy	Dead	Alive
I	6	3	2	—	—	1	—	—	—	6
II	8	2	2	2	—	1	1	—	1	7
III	3	—	—	—	1	1	—	1	3	—

hemorrhage not associated with intraventricular hemorrhage and both the babies survived.

Discussion

The present study was undertaken to evaluate CT scan changes in patients with symptomatic birth asphyxia and to correlate these changes with clinical severity and immediate neonatal outcome. While a number of workers have studied the problem of CT scan changes in birth asphyxia(3-8,10), our study differed from them in the criterion of selection of patients and in the nature of outcome under evaluation.

Normal CT scans were found in 28% of our neonates, of which all but one survived. This child who expired was a premature neonate, weighing 0.75 kg and had died of extreme prematurity and septicemia. None of the patients dying of asphyxia had a normal CT scan in series of Flodmark *et al.*(2) However, 21.4(9) 6.5% term patients(8) and 7.6% term subjects(4) in earlier series had a normal CT scan.

Hypodense brain tissue was seen in twelve patients (48%) of which six (24%) had mild hypodensity and remaining six (24%) had moderate to severe hypodensity. Of the patients with mild hypodensity, 3 (1 term and 2 preterm babies) had only mild periventricular hypodensity and in 3 (all term) areas of hypodensity were few and in peripheral cortical tissue away from the periventicular white matter. Similar findings have been reported by Schrump *et al.*(9), in whose series also periventricular low density was noted more often in prematures, whereas cortical low density was found more often in term babies.

Intracerebral hemorrhage was seen in seven neonates, in two hemorrhage was suspected clinically because of presence of convulsions, full fontanel, fall in hematocrit and prematurity and later it was confirmed by CT that they had IVH. In the four remaining neonates except for focal or multifocal convulsions no other clinical feature suggested possibility of intracerebral hemorrhage, which was only detected on CT scan.

Both patients with IVH in present study expired, in contrast only one of the three patients with isolated intraparenchymal hemorrhage had unfavourble outcome. This is in contrast with the observations of Merchant *et al.*(4) in whose series all the three neonates with intraventricular hemorrhage or intraparenchymal hemorrhage had an adverse outcome. Patients having subarachnoid hemorrhage had a favorable outcome in both the present, and an earlier(4) study. We can, therefore, infer that the initial pessimism related to intracranial hemorrhage may not be justified in every case.

A good correlation between CT Scan changes with the Sarnat staging and neonatal outcome was observed. An abnormal CT Scan in a Stage III HIE predicted a grave outcome, while neonates in HIE Stage I with normal or near normal scan survived the acute hypoxic insult. However, in a follow up study (9-18 months of age) Khare and Merchant(11) concluded that repeat CT scan may not be useful in ultimate prognostification as 5 out of 11 cases with clinical abnormalities had normal CT scan. The predictive value of CT scan in determining the neonatal outcome in the present study can be evaluated as shown in *Table IV*. Ten of the 18 patients with grossly normal scan had survived while 8 of 12 patients with abnormal CT findings expired. Thus the CT scan has good predictive value in determining immediate

TABLE IV—*Outcome of Asphyxiated Newborns in Relation to CT Findings*

Outcome	CT scan findings (n=25)		
	Normal	Abnormal	Total
Favourable (discharged)	10	4	14
Unfavourable (expired/ otherwise)	3	8	11
	13	12	25

(χ^2 =4.81, p<0.05)

neonatal outcome in newborns with abnormal CT scans (χ^2 = 4.81, p<0.05).

On comparison of CT results with neonatal outcome in term and preterm babies it is seen that predicative value of CT in determining neonatal outcome is significant in term babies (χ^2 = 5.88, p<0.05) while in preterm babies it is not (χ^2 = 1.14, p<0.1).

In conclusion CT scan can be considered a good diagnostic and prognosticating aid in HIE for neonatal outcome along with its clinical presentation.

REFERENCES

1. Drage JS, Kennedy C, Borende SH. The Apgar score as an index of infant morbidity. Dev Med Child Neurol 1966, 8: 141-143.
2. Flodmark O, Becker LE, Harwood-Nash DC, Fitzhardinge PM, Fitz CR, Chuang SH. Correlation between computed tomorgraphy and autopsy in premature and full term neonates that have suffered perinatal asphyxia. Radiology 1980, 137: 93-97.
3. Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress. Arch Neurol 1976, 33: 696-705.
4. Merchant R, Shah A, Abhyankar S, Joshi NC. CT-scan evaluation of term neonates with symptomatic birth asphyxia. Indian Pediatr 1987, 24: 57-58.
5. Fitzhardinge PM, Flodmark O, Fitz CR. The prognostic value of computed tomography as an adjunct to assessment of the full-term neonate with post-asphyxial encephalopathy. J Pediatr 1981, 99: 777-781.
6. Fitzhardinge PM, Flodmark O, Ashby S. The prognostic value of computed tomography of the brain in asphyxiated premature infants. J Pediatr 1982, 100: 476-480.
7. Flodmark O, Fitz CR, Harwood Nash DC. The CT diagnosis and short term prognosis of intracranial hemorrhage and hypoxic/ischemic brain damage in neonates. J Comput Assist Tomogr 1980, 4: 775-777.
8. Merchant RH, Chablani V, Parekh B, Goudinho N, Vas NP. Computerized tomography and electroencephalography in the immediate evaluation of term infants with symptomatic birth asphyxia. Indian Pediatr 1984, 51: 327-332.
9. Schrump JD, Schrim S, Killpack J, Prady JR, Hirata T, Mednick JP. Correlation of early neurologic outcome and CT findings in neonatal brain hypoxia and injury. J Comput Assist Tomogr 1980, 4: 445-450.
10. Volpe JJ. Perinatal hypoxic ischemic brain injury. Pediatr Clin N Am 1976, 3: 303-387.
11. Khar MD, Merchant RH. Diagnostic and prognostic value of CT brain scan in term neonates with moderate and severe birth asphyxia. Indian Pediatr 1990, 27: 267-271.