

Original Research Article

Biochemical abnormalities in neonatal seizures in term and preterm neonates

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ABSTRACT

Background: Neonatal seizures may arise as a result of diverse etiologies and can have varied presentations. Biochemical abnormalities are commonly observed in neonates which can be either primary or secondary. Early recognition and treatment of biochemical disturbances is essential for optimal management and satisfactory long-term outcome.

Methods: A total of 100 neonates presenting with seizures admitted to NICU of JJM Medical College, Davanagere, from November 2015 to April 2017 were enrolled in the study. Detailed antenatal, natal, postnatal history along with detailed examination was done along with baseline characteristics of convulsing were recorded at admission along with relevant biochemical investigations before instituting any specific treatment.

Results: In the present study, out of 100 neonates studied, 64 were full term of which 49(76.5%) were AGA and 15(23.5%) were SGA, whereas 36 cases were preterm. Most neonatal seizures occurred in first 3 days of life, i.e. 59% of which majority occurred on first day of life (34%). Birth asphyxia and septicemia are common cause of neonatal seizures in present study (38 cases each), followed by pure metabolic disturbances 19%. In pure metabolic seizures, hypoglycemia (47.8%) is most common more in preterm babies (55%) followed by hypocalcemia.

Conclusions: Biochemical abnormalities are common in neonatal seizures and often go unrecognized and may significantly contribute to seizure activity. Hence, a biochemical work up is necessary for all cases of neonatal seizures.

Keywords: Birth Asphyxia, Hypocalcemia, Hypoglycemia, Neonatal seizures

INTRODUCTION

Seizure is defined as paroxysmal involuntary disturbance of brain function. It may manifest as impairment or loss of consciousness, abnormal motor activity, behavioural abnormality, sensory disturbance or autonomic dysfunction.¹ Any abnormal, repetitive and stereotypic behaviour in neonates should be evaluated as possible seizure. Neonatal seizures by definition occur within the first 4 weeks of life in a full-term infant and up to 44 weeks from conception for premature infant and are most

frequent during the first 10 days of life.^{2,3} Neonatal seizures is a common neurological problem with a frequency range from 0.95 to 3.5/1000 live births.⁴ Seizure incidence is higher during this period than in any other period of life. Incidence is as high as 57.5 per 1000 in infants with birth weights lower than 1500 g, and only 2.8 per 1000 in infants with birth weights of 2500 to 3999 g.⁵⁻⁷ The presence of seizure does not constitute a diagnosis, but it is a symptom of an underlying central nervous system disorder due to systemic or biochemical disturbances. Among various etiologies, birth asphyxia;

neonatal meningitis and biochemical abnormalities are the commonest etiologies of neonatal seizures. Biochemical abnormalities occur either as an underlying cause or as an associated abnormality. Early recognition and treatment of these biochemical disturbances is essential for the optimal management and satisfactory outcome. So biochemical abnormalities should be excluded in every case of neonatal seizure inspite of presence of other causes such as meningitis or asphyxia and structural abnormalities. Hence, authors intend to study the biochemical abnormalities in neonatal seizures, which would help in early treatment and better prognosis.

The objectives are to study clinical presentation and time of onset of seizures in term and preterm neonates and to study the biochemical abnormalities in seizures in term and preterm neonates.

METHODS

A hospital based prospective observational study was conducted in NICUs of all the three hospitals attached to JJM Medical College, Davangere. A total of 100 neonates with seizures were included in the study from November 2015 to April 2017 after taking written and informed consent.

Detailed antenatal, natal and postnatal history was taken as per the proforma enclosed. Baseline characteristics of convulsing neonate including sex, gestational age, birth weight, head circumference and length were recorded at admission. Clinical details of each seizure episode reported by the mother and subsequently observed by the resident doctors on duty were recorded i.e. age at onset of seizures, duration of seizure, number and type of seizure.

Relevant investigations were done depending upon clinical presentation which included complete blood count, sepsis screening (TLC, ANC, immature to total neutrophil ratio, CRP), blood glucose, serum electrolytes like sodium, potassium, calcium, phosphorus, magnesium and other metabolic screening includes serum ammonia, serum lactate, urine ketones and urine for reducing substance if metabolic disorders were suspected. Lumbar puncture was considered in all cases with suspected meningitis or septicemia.

Neurosonogram was done in all neonates with seizures to rule out intraventricular/ parenchymal hemorrhage, congenital anomalies of brain. EEG was done in all neonates requiring anti-convulsant therapy. CT scan or MRI Brain was done as and when necessary.

Statistical analysis

Data was entered into Microsoft Excel (Windows 7; Version 2007) and analyses were done using the Statistical Package for Social Sciences (SPSS) for Windows software (version 20.0; SPSS Inc, Chicago).

Microsoft word and excel have been used to generate graphs, tables etc.

RESULTS

In present study, out of 100 babies, 56 (56%) were males and 44 (44%) were female babies with a male to female ratio of 1.28:1.

In the present study, 64 were full term of which 49 cases were AGA 28 cases (57%) were male and 21 cases (43%) were female and 15 cases were SGA. 7 cases (47%) were male and 8 cases (53%) were females, whereas 36 were preterm 21 cases (58%) were male and 15 cases (42%) were females.

Table 1: Association between gestational age and gender.

Gender	Group			Total
	Preterm (n=36) n (%)	Term AGA (n=49) n (%)	Term SGA (n=15) n (%)	
Female	15 (41.7)	21 (42.9)	8 (53.3)	44
Male	21 (58.3)	28 (57.1)	7 (46.7)	56
Chi-Square Test, P value=0.728, Not Significant				

In the present study, 19 out of 100 babies were <1.5kg, 17 were between 1.5-1.99 kg, 16 were between 2.0-2.49 kg, 41 were between 2.5-2.99 kg and 7 were between 3.0-3.49 kg.

Table 2: Association between gestational age and birth weight.

Birth weight (kg)	Group			Total
	Preterm (N=36) n (%)	Term AGA (N=49) n (%)	Term SGA (N=15) n (%)	
<1.5	19 (52.8)	0 (0.0)	0 (0.0)	19
1.5-1.99	16 (44.4)	0 (0.0)	1 (6.7)	17
2.0-2.49	1 (2.8)	1 (2.0)	14 (93.3)	16
2.5-2.99	0 (0.0)	41 (83.7)	0 (0.0)	41
3.00-3.49	0 (0.0)	7 (14.3)	0 (0.0)	7

Chi-Square Test, P value <0.001, Significant

In the present study, on first day 34 cases developed seizures, 18 cases developed on second day, 7 cases developed on third day, 29 cases developed seizures from 4th-7th days.

During 2nd week 8 cases developed seizures, 3 cases developed in 3rd week and 1 case developed seizures in 4th week. 59% of cases developed seizures in first three days, 70 % (29 cases) of the remaining 41% had seizures in 4th-7th day, late onset seizures i.e. after 8days of life constitute 30% (12 of 41 cases).

Table 3: Association between gestational age and day of onset of seizures.

Day of onset of seizures	Group			Total
	Preterm (n=36) n (%)	Term AGA (n=49) n (%)	Term SGA (n=15) n (%)	
1	7 (19.4)	23 (46.9)	4 (26.7)	34
2	6 (16.7)	8 (16.3)	4 (26.7)	18
3	4 (11.1)	2 (4.1)	1 (6.7)	7
4	7 (19.4)	4 (8.2)	1 (6.7)	12
5	4 (11.1)	2 (4.1)	1 (6.7)	7
6	3 (8.3)	2 (4.1)	0 (0.0)	5
7	1 (2.8)	2 (4.1)	2 (13.3)	5
8	1 (2.8)	1 (2.0)	0 (0.0)	2
9	1 (2.8)	1 (2.0)	1 (6.7)	3
10	0 (0.0)	1 (2.0)	0 (0.0)	1
12	1 (2.8)	0 (0.0)	0 (0.0)	1
13	0 (0.0)	1 (2.0)	0 (0.0)	1
15	0 (0.0)	1 (2.0)	0 (0.0)	1
18	0 (0.0)	1 (2.0)	1 (6.7)	2
25	1 (2.8)	0 (0.0)	0 (0.0)	1

Chi-Square Test, P value = 0.595, Not Significant

In present study, most common type of seizures was subtle (42 cases), followed by focal clonic (19 cases), multifocal clonic (18 cases), generalized tonic (14 cases), subtle with GTC (4 cases) and subtle with clonic (3 cases). Most common type of seizures in term is subtle (24 of 64 cases) followed by multifocal clonic seizures (12 of 64 cases). Whereas in preterm most common type is subtle (18 of 36 cases) followed by focal clonic seizures (7 of 36 cases).

Birth asphyxia and septicemia were common causes of neonatal seizures in present study (38 cases each), followed by pure metabolic disturbances were seen in 19 cases, intracranial bleed in 3 cases and unknown etiology in 2 cases.

Most common causes of neonatal seizures in term was birth asphyxia (34 of 64 cases) followed by septicemia (20 of 64 cases). Whereas in preterm most common cause was septicemia (18 of 36 cases) followed by pure metabolic disturbance (12 of 36 cases).

Table 4: Association between gestational age and etiology.

Etiology	Group			Total
	Preterm (n=36) n (%)	Term AGA (n=49) n (%)	Term SGA (n=15) n (%)	
Birth asphyxia	4 (11.1)	29 (59.1)	5 (33.3)	38
Septicemia	18 (50.0)	14 (28.5)	6 (40.0)	38
Metabolic	12 (33.3)	3 (6.1)	4 (26.7)	19
IC Bleed	2 (5.5)	1 (2.0)	0 (0.0)	3
Unknown	0 (0.0)	2 (4.0)	0 (0.0)	2

In present study, on first day 34 cases developed seizures of which 28 were due to birth asphyxia and 4 were due to hypoglycemia, on the 2nd day 18 cases developed seizures of which 8 were due to birth asphyxia and 5 were due to hypoglycemia, 7 cases developed seizures on third day of which hypocalcemia (3 cases) was common.

Table 5: Association between etiology and day of onset of seizures.

Day of onset of seizures	Etiology						Total
	Birth asphyxia	Hypocalcaemia	Hypoglycaemia	IC bleed	Septicemia	Others	
1	28 (73.7)	1 (20.0)	4 (36.4)	0 (0.0)	0 (0.0)	1 (50.0)	34
2	8 (21.1)	1 (20.0)	5 (45.5)	1 (33.3)	0 (0.0)	0 (0.0)	15
3	2 (5.3)	3 (60.0)	1 (9.1)	1 (33.3)	0 (0.0)	0 (0.0)	7
4	0 (0.0)	0 (0.0)	1 (9.1)	0 (0.0)	10 (26.3)	1 (50.0)	12
5	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	7 (18.4)	0 (0.0)	7
6	0 (0.0)	0 (0.0)	0 (0.0)	1 (33.3)	4 (10.5)	0 (0.0)	5
7	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	5 (13.2)	0 (0.0)	5
2 nd week	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	8 (21.1)	0 (0.0)	9
3 rd week	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (7.9)	0 (0.0)	3
4 th week	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (2.6)	0 (0.0)	1

In the present study, biochemical abnormalities were seen in 75 cases, of which 52 (69.3%) were non-metabolic and 23 (30.6%) were pure metabolic seizures. The most common biochemical abnormality in neonatal seizures is hypoglycemia 45.3% (34 of 75 cases) followed by

hypocalcemia 22.7% (17 of 75 cases). Among pure metabolic abnormalities, hypoglycemia was documented in 47.8% (11 of 23 cases) followed by hypocalcemia (21.7%), hypophosphatemia (17.4%) and hypomagnesemia (13%).

Among non-metabolic abnormalities, hypoglycemia was noted in 44.2% (34 of 75 cases) followed by hypocalcemia (23.1%), hypophosphatemia (15.4%),

hypernatremia (11.5%), hyponatremia (3.8%) and hypomagnesemia (1.9%).

Table 6: Comparison of biochemical abnormalities between metabolic and non-metabolic seizures.

Etiology	Biochemical abnormalities						Total
	Hypo-natraemia	Hyper-natraemia	Hypo-glycaemia	Hypo-phosphatemia	Hypo-magnesaemia	Hypo-calcaemia	
Metabolic	0 (0.0)	0 (0.0)	11 (47.8)	4 (17.4)	3 (13.0)	5 (21.7)	23 (100)
Non-metabolic	2 (3.8)	6 (11.5)	23 (44.2)	8 (15.4)	1 (1.9)	12 (23.1)	52 (100)
Total	2 (2.7)	6 (8.0)	34 (45.3)	12 (16)	4 (5.3)	17 (22.7)	75 (100)
P value	0.340	0.089	0.773	0.827	0.048	0.898	

Table 7: Distribution of overall biochemical abnormalities in neonatal seizures.

GA	Biochemical abnormalities						Total
	Hypo-natraemia	Hyper-natraemia	Hypo-glycaemia	Hypo-phosphatemia	Hypo-magnesaemia	Hypo-calcaemia	
Preterm (n=36)	0 (0.0)	2 (5.6)	13 (36.1)	6 (16.7)	3 (8.3)	8 (22.2)	32
Term AGA (n=49)	2 (4.1)	3 (6.1)	16 (32.7)	3 (6.1)	1 (2.0)	4 (8.2)	29
Term SGA (n=15)	0 (0.0)	1 (7.1)	5 (35.7)	3 (21.4)	0 (0.0)	5 (35.7)	14

In present study, most common biochemical abnormality among term neonates with seizures was hypoglycemia (33.33%) followed by hypocalcemia (14.3%). Whereas in preterm 36.1% had hypoglycemia and 22.2% had hypocalcemia.

Of 75 cases of neonatal seizures associated with biochemical abnormalities, 40 babies (53.3%) were male and 35 babies (46.7%) were female. Of the 52 cases of non-metabolic seizures, which are associated with biochemical abnormalities, hypoglycemia was most common abnormality 23 cases (44.2%), 12 cases (52.1%) were associated with birth asphyxia and 11 cases (47.9%) were associated with septicaemia.

In the present study, 18 of 38 cases (16 were term and 2 were preterm) of birth asphyxia were associated with biochemical abnormalities of which 11 cases were associated with hypoglycemia, followed by 5 cases associated with hypocalcemia and 2 cases associated with hyponatremia.

Of 38 cases of sepsis, 22 cases (12 were term and 10 were preterm) were associated with biochemical abnormalities of which 11 cases were associated with hypoglycemia, 6 cases are associated hyponatremia and 5 cases were associated with hypocalcemia.

Of the 22 cases of metabolic abnormalities, 11 cases had seizures due to hypoglycemia (6 were preterm and 5 were term) followed by 5 cases due to hypocalcemia (3 were preterm and 2 were term), 2 cases due to hypocalcemia with hypomagnesemia (2 preterm) and 1 case is due to

hypomagnesemia (preterm). Of 3 cases of IC bleed, 1 case is associated with hypocalcemia (33.3%).

DISCUSSION

In the present study, out of 100 neonates with seizures, 64% (64 cases) were full term of which 49 cases (76.5%) were appropriate of gestational age and 15 cases (23.5%) were small for gestational age, whereas 36% (36 cases) were preterm.

Majority of neonatal with seizures in present study were seen in full term babies, birth asphyxia was the commonest cause of seizures in full term babies and was associated with perinatal complications like MSAF in 22 cases, and prolonged second stage of labour in 10 cases. Yadav R K et al has shown similar observations to present study where full-term babies presented with seizures were 60%, preterm babies were 34% and post term babies were 6%.⁸

In the present study, 19 out of 100 were <1.5kg, 17 were between 1.5-1.99, 16 were between 2.0-2.49, 41 were between 2.5-2.99 and 7 were between 3.0-3.49. In a study by Marzoki JM et al.⁹ 93.1% were weighting > 2500g, 2.3% were very low birth weight <1500 g and the remaining 2.3% were low birth weight (1500-2500).

59% of cases developed seizures in first three days, 70 % (29 cases) of the remaining 41% had seizures in 4th-7th day, late on seizures i.e. after 8days of life constitute 30% (12 of 41 cases). In a study by Asif Aziz et al 83 neonates (83%) presented with seizures within the first 72 hours of

life.¹⁰ Rose et al also found early onset seizures in 75 (50.33%) babies whereas Coen RW et al found that 81% of babies had early onset seizures which is similar to present study.^{11,12}

Subtle seizures (42 cases) were most common type of seizures in present study, followed by focal clonic (19 cases), multifocal clonic (18 cases), generalized tonic (14 cases), subtle with GTC (4 cases) and subtle with clonic (3 cases). Most common type of seizures in term was subtle (24 of 64 cases) followed by multifocal clonic seizures (12 of 64 cases). Whereas in preterm most common type was subtle (18 of 36 cases) followed by focal clonic seizures (7 of 36 cases). In a study of neonatal seizures by A.L Bairwa et al most common type of seizures were subtle 42.6% followed by multifocal clonic 26.08%, focal clonic 17.39% and tonic seizures 8.6%.¹³ However, a study by Ajay Kumar et al multifocal clonic seizures (42.24%) were the most common seizure type followed by generalized tonic (21.55%), subtle (8.19%), focal clonic (6.47%) and myoclonic (0.86%).¹⁴

Most common causes of neonatal seizures in term was birth asphyxia (34 of 64 cases) followed by septicemia (20 of 64 cases). Whereas in preterm most common cause was septicemia (18 of 36 cases) followed by pure metabolic disturbance (12 of 36 cases). In a study by Moaryedi AR et al most common etiology of neonatal seizures was HIE (36.4%) followed by infections (19.1%), metabolic disorders and inborn errors of metabolism (7.3%), Intra Cranial Hemorrhage (ICH) (2.7%), structural disorders (1.8%) and in thirty six cases (32.7%) the etiology was not defined.¹⁵

In present study, on first day 34 cases developed seizures of which 28 cases (82.35%) were due to birth asphyxia and 4 (11.76%) were due to hypoglycemia, on the 2nd day 18 cases developed seizures of which 8 were due to birth asphyxia and 5 were due to hypoglycemia, 7 cases developed seizures on third day of which hypocalcemia (3 cases) was common.

In the present study, biochemical abnormalities were seen in 75 cases, of which 52 (69.3%) were non-metabolic and 23 (30.6%) were pure metabolic seizures. The most common biochemical abnormality in neonatal seizures was hypoglycemia 45.3% seen in 23 cases of non-metabolic seizures and 11 cases of pure metabolic seizures, followed by hypocalcemia 22.7% seen in 12 cases of non-metabolic seizures and 5 cases of pure metabolic seizures.

Among pure metabolic abnormalities most common abnormality was hypoglycemia 47.8% (11 of 23 cases) followed by hypocalcemia (21.7%), hypophosphatemia (17.4%) and hypomagnesemia (13%). Among non-metabolic seizures, which are associated with biochemical abnormalities, hypoglycemia was most common abnormality 23 cases (44.2%). Of which 12 cases (52.1%) were associated with birth asphyxia and 11

cases (47.9%) were associated with septicemia. Sood A et al in his study showed that hypoglycemia is the most common biochemical abnormality associated with birth asphyxia followed by hypocalcemia, hypomagnesemia and hyponatremia.¹⁶ Ericksson M et al also reported that most cases of birth asphyxia are as CNS infections in present study was associated with hypoglycemia in 11 cases followed by hypocalcemia in seven cases, hypernatremia and hyperphosphatemia in 6 cases each.¹⁷ Basu P et al showed that the severity of encephalopathy and cellular damage varies with the severity of hypoglycemia. associated with hypoglycemia similar to present study.¹⁸

Pure metabolic seizures were seen in 23 cases. Most common cause of metabolic seizures was due to hypoglycemia 11 cases (47.8 %) followed by hypocalcemia in 5 cases (21.7%), hypophosphatemia in 4 cases (17.4%) and hypomagnesemia in 3 cases (13%). Of the 11 cases of hypoglycemia, 6 cases (54.5%) were preterm, 2 cases (18.2%) were term AGA and 3 cases (27.3%) were term SGA. In a study of hypoglycemia by Lawrence LD et al 41% of hypoglycemic neonates were SGA.¹⁹ Singhal PK et al in his study showed that preterm babies have 3 times increased risk (12.6%) as compared to term babies (3.6%) for hypoglycemia and that manifest as seizures (30.2%).²⁰

CONCLUSION

The recognition of the etiology for the neonatal seizures is often helpful with respect to prognosis and treatment. Seizures are a symptom of underlying CNS disorder, which can be due to systemic or biochemical disturbances. Biochemical disturbances are commonly transient and rapidly correctable or less commonly inherited as persistent causes. They occur frequently in neonatal period either as an underlying cause or as an associated abnormality. In their presence, it is difficult to control seizures and there is a risk of further brain damage. Early recognition and treatment of biochemical disturbances are essential for optimal management and satisfactory long-term outcome.

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REFERENCES

1. Berg A, Jallon P, Preux P. The epidemiology of seizure disorders in infancy and childhood: definitions and classifications. In: Dulac O, Lasseonde M, Sarnat HB, eds. Handbook of clinical neurology. Pediatric Neurology. 3rd edition. Elsevier, Amsterdam, Netherlands; 2013;1:381-398.
2. Singh M. Neurological disorders. In textbook of care of newborn. 5th ed. New Delhi: Sagar Publication; 1999:340-344.

3. Vigevano F. Benign familial infantile seizures. *Brain Dev.* 2005;27:172.
4. Ann M. Bergin. Neonatal Seizures. John P Cloherty. *Manual of Neonatal Care*, 7th ed. Lippincot manual. 2012;56:729-742.
5. Lanska MJ, Lanska DJ. Neonatal seizures in the United States: results of the national hospital discharge survey, 1980-1991. *Neuroepidemiol.* 1996;15(3):117-25.
6. Lanska MJ, Lanska DJ, Baumann RJ, Kryscio RJ. A population-based study of neonatal seizures in Fayette County, Kentucky. *Neurol.* 1995;45(4):724-32.
7. Ronen GM, Penney S, Andrews W. The epidemiology of clinical neonatal seizures in Newfoundland: a population-based study. *J Pediatr.* 1999;134(1):71-5.
8. Yadav RK, Sharma IK, Kumar D. Clinicoetiological and biochemical profile of neonatal convulsions. *Int J Med Res Rev.* 2015;3(9):1057-63.
9. Jasim MA. Marzoki. Clinico-biochemical profile of neonatal seizures. *QMJ.* 2010;6(10):163-4.
10. Aziz A, Gattoo I, Aziz M, Rasool G. Clinical and etiological profile of neonatal seizures: a tertiary care hospital based study. *Int J Res Med Sci.* 2017;3(9):2198-203.
11. Rose AL, Lombroso. CT. A study of clinical, pathological and electroencephalographic features in 137 full term babies with a long term follow up. *Paediatr.* 1970;45:404-25.
12. Coen RW, Mc Cutchen CB, Wermer D, Snyder J, Gluck FE. Continuous monitoring of EEG following perinatal asphyxia. *J Pediatr.* 1982;100:628-30.
13. Kumar MB, Bairwa AL. Study of the clinical profile of neonatal seizures. *Indian J Res.* 2014;3(2):201-9.
14. Kumar A, Gupta A, Talukdar B. Clinico-etiological and EEG profile of neonatal seizures. *Indian J Pediatr.* 2007;74(1):33-7.
15. Moayedi AR, Zakeri S, Moayedi F. Neonatal seizure: etiology and type. *Iranian J Child Neurol.* 2008;2(2):23-6.
16. Sood A, Grover N, Sharma R. Biochemical abnormalities in neonatal seizures. *Indian J Paed.* 2003;70(3):221-4.
17. Eriksson M, Strom ZR. Neonatal convulsions. *Acta Paediatr Scand.* 1979;68:807-11.
18. Basu P, Som S, Choudhuri N, Das H. Contribution of the blood glucose level in perinatal asphyxia. *Euro J Pediatr.* 2009;168(7):833-8.
19. Lilien LD, Grajwer LA, Pildes RS. Treatment of neonatal hypoglycemia with continuous intravenous glucose infusion. *J Pediatr.* 1977;91(5):779-82.
20. Singhal PK, Singh M, Paul VK. Prevention of hypoglycemia: a controlled evaluation of sugar fortified milk feeding in small- fordate infants. *Indian Pediatr.* 1992;29:1365-9.

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