International Journal of Contemporary Pediatrics
Afsar S et al. Int J Contemp Pediatr. 2016 Nov;3(4):1185-1188
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291
DOI: http://dx.doi.org/10.18203/2349-3291.ijcp20163137

Original Research Article

Evaluation of neuro-developmental outcome among babies with meconium aspiration syndrome

Suhaim Afsar, Naresh P. Motwani*, Sudhakar C., Uma Chaturvedi

Department of Pediatrics, Chandulal Chandrakar Memorial Hospital, Bhilai, Chhattisgarh, India

Received: 10 August 2016
Accepted: 22 August 2016

***Correspondence:**
Dr. Naresh P. Motwani,
E-mail: nareshmot@yahoo.com

ABSTRACT

Background: Meconium aspiration syndrome (MAS) is respiratory distress in a neonates caused by the presence of meconium in the tracheobronchial airways. Despite adequate management, there is a high risk of morbidity in the form of seizures, cerebral palsy, mental retardation, respiratory problems of childhood and mortality. Hence, this study was performed in view of developmental issues concerning young infants and pre-school children as maximum brain growth happens in the first three years of life.
Methods: This was a prospective study conducted in Chandulal Chandrakar memorial hospital, Bhilai from 1st September 2013 to 31st February 2015, with history of meconium stained amniotic fluid (MSAF) in both out-born and in-born neonates. Neonates after meeting inclusion criteria were included in the study. The data were recorded in pre-designed proforma. The data were analyzed using appropriate Chi square test. Level of significance was set at $p < 0.05$.
Results: Incidence of MAS was significantly more in children of >2.5 kgs (80%) and common in primiparous mothers (60%) with lower segmental caesarian sections. MAS commonly seen in post -term babies (53.33%) than those of term (36.66%) or pre-term (10%) gestation. Fetal distress was the common complication observed in most of the cases (91.1%) and one death related to this was noted. At the end of 1 year there were predominantly more children (40%) who developed respiratory morbidities. Delayed development was seen among 13.3% children, transient tone abnormalities were noted in about 2% of infants.
Conclusions: The findings of the present study suggest that neonates diagnosed with MAS displayed neuro-developmental delay in13% cases. This study gave an overview of all meconium aspiration cases and the neuro-developmental outcome in these babies. However further research should be done with large sample size to confirm these findings.

Keywords: Foetal distress, MAS, Neuro-developmental outcome

INTRODUCTION

Meconium aspiration syndrome (MAS) is a serious, life-threatening respiratory disorder of the newborn that occurs in approximately 2% to 5% of infants born through meconium-stained amniotic fluid.[1] Despite adequate management, there is a high risk of morbidity in the form of seizures, cerebral palsy, mental retardation, respiratory problems of childhood and mortality.

The rate of severe mental retardation, cerebral palsy and neonatal seizures is significantly higher among infants born through meconium stained amniotic fluid (MSAF) who had developed MAS.[2,3] Infants with MAS manifest later with neuro-developmental delays even if they have responded to earlier conventional treatment and it was found that 7% had cerebral palsy and 14% had developmental delays by 12 months of age.[4] Hence this study was performed in assessing developmental issues

related with MAS in young infants and pre-school children as maximum brain growth happens in the first three years of life.

METHODS

This was a prospective study conducted in Chandulal Chandrakar memorial hospital, Bhilai from 1st September 2013 to 31st Febrauary 2015, with history of meconium stained amniotic fluid (MSAF) in both out born and inborn neonates.

All in-born babies with MSAF related respiratory distress and out born babies with meconium staining of skin and umbilical cord with respiratory distress and babies all babies showing features of meconium in the upper respiratory tract or on chest examination with respiratory distress were included in the study. Exclusion criteria were babies with congenital anomalies and babies born of breech or other abnormal presentations.

The presence of MSAF and consistency of meconium, presence of fetal distress, and mode of delivery were noted. The condition of infant in delivery room and any interventions observed were reviewed. The following data like APGAR score, respiratory distress, non-homogenous infiltrates with or without hyperinflation in chest X-rays were retrieved. The gestational age was assessed as per new Ballard score. Babies who had significant respiratory distress were started on non-invasive ventilation, continuous positive airway pressure (CPAP) or mechanical ventilation when indicated. The data was collected for variables and determinants related to MSAF and development of MAS.

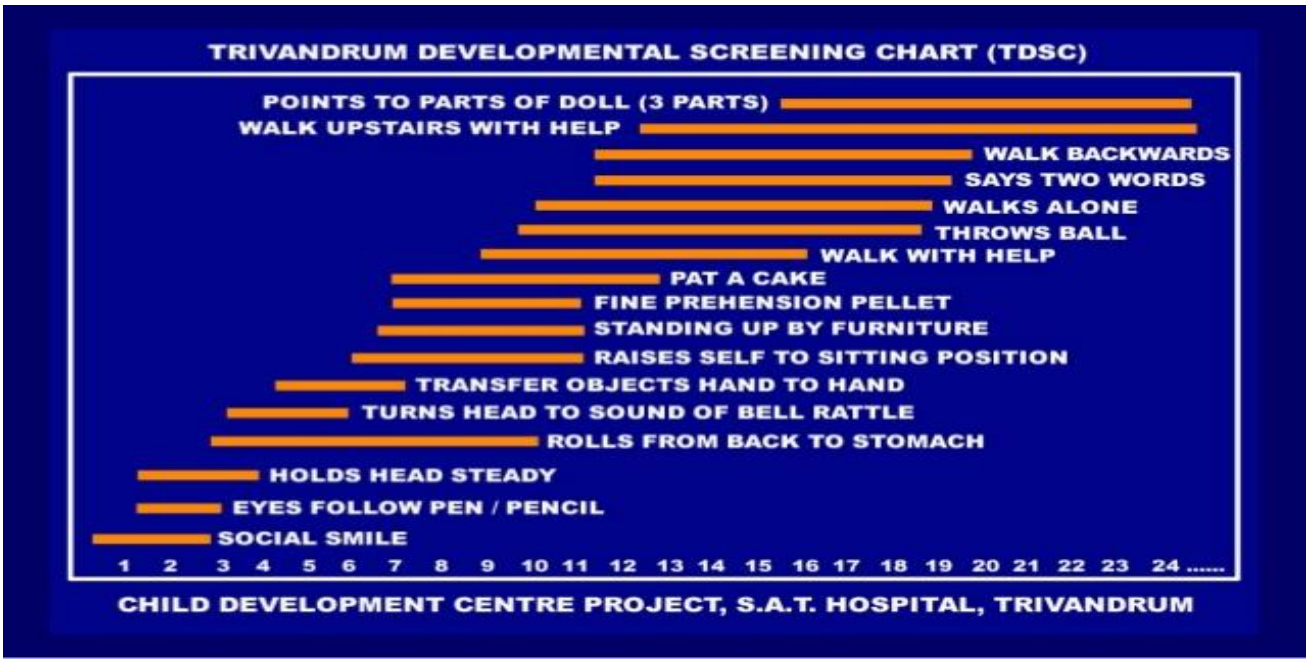

Figure 1: Trivandrum development screening chart-0-2 years.

The babies were followed up for 1 year after discharge and were observed for neurological development outcome using Trivandrum development screening chart-0-2 years as given in Figure 1.

Sample size calculation

According to previous hospital records the percentage of babies with MAS among babies with MSAF was 4% and sample size was calculated by Cochran formula.[5]

Incidence of MAS in MSAF (as per past records) = 4%
P = proportion of MAS in MSAF = 4% = 0.04
Q =1- p = 96% = 0.96

Cochran Formula, N = $\frac{1.96^2 * p * q}{e^2}$ = 59.007 = 60 (After rounding off)
1.96 = Z score for 95% confidence level; e= 0.05 precision.

Statistical analysis

Differences between categorical data was analysed with Chi square test. To describe the data, minimum, maximum, mean, range and standard deviation or medians were reported for continuous variables. For categorized variable, percentage was used. P value <0.05 was considered to be significant and P value <0.0001 was considered to be highly significant.

RESULTS

Table 1: Demographic data.

Parameters	Neonates with MAS
Birth weight	
(>2.5 kgs)	12 (20%)
>2.5 kg	48 (80%)
Mode of delivery	
Vaginal	17 (28.33%)
Lower segment caesarean section	43 (71.66%)
Parity	
Primipara	36 (60%)
Multipara	24 (40%)
Gestational age	
Pre-term	6 (10%)
Term	22 (36.66%)
Post term	32 (53.33%)
Fetal distress	
Present	41 (91.11%)
Immediate outcome	
Deaths	1 (1.66%)
Discharges	59 (98.33%)

In this study, MAS with MSAF was observed in 60 neonates. The demographic profile of the study was presented in Table 1. Incidence of MAS was observed in neonates born with weight more than 2.5 kgs (80%) through lower segmental caesarean section (71.6%) and observed more common in primiparous mothers (60%) with post term gestational age (53.3%). Fetal distress was the most common feature among babies of MAS and observed in 41 (91.1%) babies. Among 60 neonates with MAS, one (1.66%) baby was dead.

Table 2 shows the outcome after one year follow - up. Forty pcnt of children developed respiratory morbidities. Delayed development was seen among 13.3% children, transient tone abnormalities were noted in about 2% of infants, who outgrew subsequently with no developmental delay or any further changes in tone or posture. However children 26 (43.33%), did not develop any morbidities on follow up.

Table 2: Outcome on follow –up after one year.

Follow up		No of Babies	%
Respiratory system	Bronchopneumonia	18	30
	Bronchiolitis	6	10
CNS	Delayed developmental milestones	8	13.33
Sepsis		2	3.33
Normal on follow up		26	43.33
Total		**60**	**100**

Table 3 relates the delayed developmental outcomes with APGAR score, consistency of meconium, respiratory distress and sepsis. Children with moderate to mild asphyxia had shown developmental delay as compared to children with good APGAR scores. It was more common among babies who had thick meconium (17.39%) during perinatal period than those with thin meconium (0%). Respiratory distress was assessed by Downes score after birth. Patients with Downes score 4-6 had shown developmental delay in 6 (21%) and score with >7 showed delay in 2 (7.4%) patients. Sepsis associated developmental delay was observed in 21 children out of 59.

Table 3: Relation of development delay with other variables.

Variables		Number	Development delay	
			Present	**Absent**
APGAR score	>7 (8-10)	2 (4.4%)	0	2 (100%)
	5-7 mild asphyxia	30 (66.6%)	5 (16.66%)	25 (83.33%)
	<5 moderate asphyxia	13 (28.8%)	1 (7.69%)	12 (92.3%)
Chi square test-0.95, d.f.-2, p value >0.05.				
Consistency	Thick	46 (76.6%)	8 (17.39%)	38 (82.60%)
	Thin	14 (23.3%)	0	14 (100%)
Chi square test-2.80, d.f.-1, p value >0.05.				
Respiratory distress score (downes score)	0-3	5 (8.33%)	0	5 (100%)
	4-6	28 (46.6%)	6 (21%)	22 (78.5%)
	>7	27 (45%)	2 (7.4%)	25 (92.5%)
Chi square test-3.178, d.f.-2, p value >0.05.				
Sepsis	Present	21 (35%)	6 (28.5%)	15 (71.42%)
	Absent	39 (65%)	2 (5.12%)	37 (94.8%)
Chi square test-6.49, d.f.-1 , p value <0.01 (Significant).				

DISCUSSION

MAS are one of the distinct clinical syndromes in neonates. Severe MAS occurs in one in 500 deliveries i.e., 0.2% of all births.[6,7] Fetal distress and birth asphyxia in uterus before birth were the stimulating factors enhancing the passage of meconium in uterus.[8] Aspiration of thick meconium leads to respiratory distress, pulmonary damage and pulmonary hypertension. Severe hypoxia leads to brain injury and hypoxic ischemic encephalopathy (HIE).[9] Thick meconium considered as a marker for foetal distress, as it produces a significant effect on the APGAR score of neonates. The present observations was comparable with the Gregory et al study.[10] As per Gupta et al incidence of birth asphyxia was significantly higher in thick meconium compared to thin meconium reported cases. In this study 76.6% cases were reported with thick meconium and 23.3% cases with thin meconium.[11]

In the present study the incidence of birth asphyxia was observed in 13% of babies indicating the prime importance of delivery room management of MAS babies to decrease the incidence of hypoxic ischemic encephalopathy (HIE). Similar observations was made by Finer et al as the incidence of HIE stage 2 in MAS babies was 15%.[3] In a study done by González de Dios et al it was found that the MAS babies had a 21% incidence of delayed developmental milestones where as in another study by Sasikala et al the incidence was found to be 16%.[12,13] Developmental delay with cerebral palsy and speech delay made the cases of poor neurological outcome in the study by Beligere et al which comprised of a total of 21%.[14] In our study it was found that 13.3% had delayed developmental milestones signifying that MAS is a very strong indicator of poor neurological outcome. In the present study it was found that 40% of cases had respiratory problems as similar to the observations of González de Dios et al in which 34% cases had reported various respiratory problems.[11] Also in the present study, sepsis associated MAS was reported in 3.33% of babies and this was similar in the study of Anwar et al (27.5%).[15]

CONCLUSION

According to this study, foetal distress, birth asphyxia, consistency of meconium and sepsis are leading causes of MAS. The findings of the present study suggest that neonates diagnosed with MAS displayed neuro-developmental delays in 13% cases. This study gave an over-view of meconium aspiration cases with neuro-developmental delay. However further research should be done with large sample size attribute these to MAS alone.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Wiswell TE, Tuggle JM, Turner BS. Meconium aspiration syndrome: have we made a difference? Pediatrics. 1990;85(5):715-21.
2. Yeh TF, Harris V. Roentegraphic findings in infants with meconium aspiration syndrome. JAMA. 1979;242:60.
3. Finer NN, Richards RT, Peters RN. Hypoxic ischemic encephalopathy in term neonates: perinatal factors and outcome. The journal of pediatrics. 1981;98:112.
4. Todres ID, Rogers MC. Neonatal resuscitation. JAMA. 1992;268:16.
5. Cochran WG. Sampling techniques. 3rd ed. New York. John Wiley and Sons; 1977:81.
6. Cleary GM, Wiswell TE. Meconium stained amniotic fliuid and MAS. An update. Ped Clin North AM. 1998;45(3):511-25.
7. Berkus MD, Langen O, Samueloff A, Xenakis EM, Field NT, Ridgway LE. Meconiumstained amniotic. uid: increased risk for adverse neonatal outcome. Obstet Gynecol. 1994;84:115.
8. Wiswell TE, Bent RC. Meconium staining and meconium aspiration syndrome. Ped Clin North Am. 1993;40:957.
9. Bascik RD. Meconium aspiration syndrome. Pediatr Clin North Am. 1977;24:467.
10. Gregory GA, Charles A, Roderic H. Meconium aspiration in infants: a prospective study. The journal of paediatrics. 1974;85:848.
11. Gupta V, Bhatia BD. Meconium stained amniotic fluid: antenatal and neonatal attributes. Indian Paediatrics. 1996;33:293.
12. González de Dios J, Moya Benavent M, Barbal Rodoreda A, Dura Trave T, Juste Ruiz M, Castaño Iglesias C, et al. Neonatal morbidity associated with meconium stained amniotic fluid. An Esp pediatr. 1998;48:54.
13. Sasikala A, Raghavan S. Perinatal outcome in relation to mode of delivery in meconium stained amniotic fluid. Indian journal of paediatrics. 1995;62:63.
14. Beligere N, Rao R. Neurodevelopmental outcome of infants with meconium aspiration syndrome: report of a study and literature review. J Perinatol. 2008;28(3):93-101.
15. Anwar Z, Butt TK, Anjum F, YaqubKazi Md. Mortality in meconium aspiration syndrome in hospitalized babies. Journal of the College of Physicians and Surgeons Pakistan. 2011;21(11):695-9.

Cite this article as: Afsar S, Motwani NP, Sudhakar C, Chaturvedi U. Evaluation of neuro-developmental outcome among babies with meconium aspiration syndrome. Int J Contemp Pediatr 2016;3:1185-8.