Clinical and Biochemical Asphyxia in Meconium Stained Deliveries

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Presence of thick meconium in the amniotic fluid is a predisposing factor towards adverse perinatal outcome mainly in the form of its aspiration and further sequelae. Intrauterine passage of meconium may occur as a physiological event or secondary to fetal hypoxia and acidosis (1-3). The diagnosis of perinatal asphyxia is generally made on the basis of Apgar score taken one minute after birth. It is affected by several factors including the gestational age, oropharyngeal management and drugs administered to the mother and is subject to personal bias. The presence of acidosis in the umbilical arterial blood, used as a biochemical marker for perinatal asphyxia may provide a useful alternative to evaluate the significance of intrauterine passage of meconium vis-a-vis asphyxia. Mitchel et al. (2) reported that meconium below the cords and meconium aspiration syndrome are a direct manifestation of fetal acidosis. However, Yeoman et al. (4) observed that though increasing umbilical cord acidemia resulted in increased frequency of meconium aspiration, yet it did not predispose to-

wards developing meconium aspiration syndrome. Only a few Western studies are available and Indian literature is silent on this particular aspect. The present study was conducted to correlate clinical and biochemical asphyxia with each other and separately, with the occurrence of meconium below the vocal cords and meconium aspiration syndrome (MAS) in babies born to mothers with thick meconium stained amniotic fluid (MSAF).

Subjects and Methods

The present prospective study was conducted over a period of 6 months in a tertiary care hospital where the annual delivery rate is around 5000 per year with 3-4% incidence of thick meconium in the amniotic fluid. Subjects included 45 consecutive, term neonates weighing more than 2500 grams, delivered vaginally by vertex presentation to mothers having thick meconium stained amniotic fluid (MSAF).

Arterial blood samples were drawn immediately after birth from a double clamped segment of the placental end of the umbilical cord. Resuscitation was carried out as per standard guidelines (5), all babies were intubated and direct tracheal suctioning was done soon after birth. Apgar score was assigned at 1 and 5 minutes. The children were transferred to the neonatal unit for observation and management. Two chest radiographs were done in all babies, the first within 6 hours and the second 24 hours following birth. Prophylactic antibiotics or steroids are not administered to neonates born through MSAF as a unit policy.

Acidosis in the cord blood was defined as a umbilical artery pH (UapH) of < 7.2 and further classified into metabolic, respiratory and mixed type (6). Meconium aspiration was defined as presence of meconium below the vocal cords.
RATION SYNDROME was defined as respiratory distress in an infant, who was meconium stained at birth, had compatible radiological findings and whose symptoms could not be otherwise explained(7) Other coexisting causes were ruled out by maternal history, neonatal examination, examination of gastric aspirate for polymorphs, cultures from sterile sites, septic screen, acid base status, oxygen saturation and metabolic workup Severe, moderate and mild birth asphyxia were labelled by one minute Apgar scores of < 2, 3 and 4, and 5 to 7 respectively(8) Standard statistical tests were used for comparison

Results

As per the Apgar scores assigned at 1 minute, 2 (4.4%), 4 (8.9%) and 13 (28.9%) infants suffered severe, moderate and mild asphyxia, respectively At five minutes, only 9 (20%) children were having an Apgar score of 7 or less Two of these nine children were in the moderate group and none had Apgar score of less than 3 UapH was < 7.0 in 2 (4.4%) cases, between 7.0 and 7.2 in 17 (37.8%) cases and > 7.2 in the remaining (57.8%) newborns Out of 19 children thus labelled as acidic, 12 infants had metabolic, three had respiratory and four had mixed type of acidosis UapH was significantly correlated with Apgar score at one minute \( r = 0.4403, p < 0.01 \) and five minutes \( r = 0.4223, p < 0.01 \)

Out of 45 children, 17 (37.8%) and 22 (48.9%) cases had evidence of meconium expiration and meconium aspiration syndrome, respectively Distribution of cases and relationship of Apgar score and UapH with meconium aspiration and meconium aspiration syndrome is depicted in Table I.

Discussion

Traditionally, presence of thick meconium stained amniotic fluid is taken as a sign of fetal distress This has been debated and questioned in various studies(3,7,9,10) In our study, only 42.2% neonates with thick MSAF suffered from asphyxia (Apgar score < 8) at one minute and acidosis was not found to be a uniform feature of all babies born through thick meconium This suggested that MSAF by itself alone may not be an excellent marker of fetal distress Recent data also indicates that some fetuses with MSL will be compromised but most will not be unless associated with other abnormalities such as type II deceleration and fetal heart rate arrhythmias(10)

Meconium inhaled below vocal cords is considered a respiratory challenge It may be related to the concentration of meconium as well as to the duration of exposure to MSL We tried to correlates the degree of asphyxia and acidosis to this event Almost all patients (5 out of 6) who had severe acidosis (UapH < 7.0) or an Apgar score of less than five at 5 minutes had meconium below their vocal cords In a recent study, umbilical artery pH < 7.0 is found to be associated with neonatal multiorgan morbidity including respiratory complications(1) Severe degree of fetal acidosis and hypoxia lead to neuromuscular depression and fetus is more likely to aspirate meconium into the lungs under these circumstances

All babies with severe or moderate asphyxia developed meconium aspiration syndrome as compared to rest, amongst whom only 41% (16/39) had evidence of MAS Five out of 6 newborns (83.3%) with Apgar score less than 5 had meconium below their vocal cords as compared to 12 out of 39 (30.7%) infants with mild or no asphyxia (Fisher's exact test, \( p < 0.05 \)) In normal fetal breathing, the net flow of fluid is out of the lungs Intrauterine hypoxia causes fetal gasping and alters the direction of fluid flow This allows meconium to be aspirated(12) Model studies on guinea pigs support this view(13)
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MA Meconium aspiration  
MAS Meconium aspiration syndrome
Brief Reports

pigs suggest that extent of meconium aspiration is not related to amount of meconium in the amniotic fluid but to the length and degree of asphyxia(13). In the present work, both babies with severe acidosis, developed meconium aspiration syndrome. Interestingly, moderate degree of acidosis did not predispose towards developing MAS. Using logistic regression analysis, a recent study has been able to identify at least 6 potential predictors of MAS (sensitivity, 92%, negative predictive value, 99%) in pregnancies complicated by moderate or thick MSAF and at least three of these predictors are related to perinatal asphyxia, i.e., fetal heart tracing, Apgar score and need for intubation(14).

It is to be emphasised that in spite of our active airway intervention in all infants, meconium aspiration syndrome could not be prevented entirely. Meconium aspiration was present in 17 infants while MAS was diagnosed in 22 newborns. Infants born with thick meconium stained amniotic fluid might aspirate meconium into the lungs in utero which can not be removed by aggressive airway management. Such newborns may develop MAS without any tangible evidence of meconium below the cords(15). Thus it is imperative that all infants born through thick MSAF, especially the asphyxiated ones must be closely monitored for developing MAS and its complications.

References


Assessment of Iodine Deficiency in Pondicherry

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As per information available, more than 1.5 billion population of the world are at the risk of Iodine Deficiency Disorders (IDD) out of which, it is estimated that about 200 million people are in our country. The survey conducted by the Central and State Health Directorates. Indian Council of Medical Research and Medical Institutes have clearly demonstrated that not even a single State/UT is free from the problem of IDD. Out of 275 districts surveyed so far, IDD is a major public health problem in 235 districts(1,2). No data is however available from the Union Territory (UT) of Pondicherry on the prevalence of iodine deficiency(1). Hence the present pilot study was conducted in the year 1997 to establish the prevalence of iodine deficiency in the UT where there is no ban on sale of non iodized salt in the UT(1).

Subjects and Methods

The study was conducted in the UT of Pondicherry. The UT has a total population of 8,07,785(2). The expected total population of children in 6-11 years was 12% (96,924). The Indian Council of Medical Research has suggested that approximately 1% of the population can provide valid estimates in a large homogenous population(3). Hence, in the present study a sample of more than 2% of the total (2065) children in the age group of 6-11 years constituted the study population. All children were examined by the first author for different goitre grades. The children in 6-11 years of age were selected as this age group is representative of the community for assessment of iodine deficiency because of their combined high vulnerability and representativeness(2).

Pondicherry state was divided into 5 geographical zones and from each zone 1 school was randomly selected for the detailed survey. In each school, all children in the age group of 6-11 years who attended the school on the day of the survey were studied. In each class, children were assembled and briefed about the study objectives. All the children were clinically examined for goitre by the palpation method. Goiter