MECONIUM ASPIRATION SYNDROME: RECENT CONCEPTS

Meconium staining of amniotic fluid has for long been considered to be a bad predictor of fetal outcome because of its direct correlation to fetal distress and increased likelihood of inhalation of meconium with resultant deleterious effects on neonatal lungs. Many of these long standing assumptions, directly linking aspiration of meconium and subsequent respiratory distress, have recently been questioned with a resultant change in the concepts of pathophysiology and management of meconium aspiration syndrome (MAS).

Meconium staining of amnietic fluid occurs in 10.3 to 22% of live births(1,2) with rising frequency along with increase in gestational age of the fetus. It is rare before 38 weeks, increasing to more than 30% in pregnancies of more than 42 weeks gestation(3,4). This corresponds to the hormonal and neural control of meconium passage, which is also maturationally dependant(5-7). Levels of Motilis, an intestinal hormone responsible for bowel peristalsis and defecation, are lower in premature infants and higher in infants who have passed meconium. With the maturation of the nervous system (which is also related to gestational age), parasympathetic stimuli generated by vagal stimulation such as umbilical cord or head compression, may be propagated to initiate meconium passage.

Several recent studies indicate that meconium staining is not an independent marker of fetal distress. Neonatal outcome was found to be better related to fetal heart rate patterns rather than the presence or absence of meconium(8,9). Though meconium staining could be the result of an event which may predispose to or be a consequence of fetal compromise but every such event may not be severe or prolonged enough to cause fetal asphyxia. Therefore presence of meconium in amniotic fluid *per se* without signs of fetal asphyxia, is no longer considered a sign of fetal distress(10).

Meconium aspiration syndrome defined as the presence of meconium below the vocal cords was earlier thought to occur with the infants first breath. This concept led to advocation of oral Delee catheter suctioning and tracheal intubation and suctioning of meconium before infant's first breath, and was accepted as standard delivery room care of all meconium stained infants. This standard care has recently been questioned by several reports which suggest that meconium aspiration is predominantly intrauterine and antepartum events(11,12).

The pathophysiologic concepts of MAS have also undergone a drastic change. It was earlier thought that the injurious effects of meconium on neonatal lungs were primarily responsible for MAS. Several autopsy findings did not confirm to this concept (13,14). Also, there was a lack of correlation between *X*-ray findings and clinical disease(15). The current belief is that respiratory distress and hypoxia of MAS are initiated by pulmonary vascular

disease(13,16,17). Hypoxia and asphyxia *in utero* produce a pulmonary vasohyperreactivity which is directly related to the extent of fetal hypoxic insult. The resultant pulmonary hypoperfusion, in the asphyxiated neonate impairs the normal mechanisms that would otherwise clear the aspirated meconium amniotic fluid from the lung. This has been well documented by experimental studies(14). Presence of meconium may compound the insult by causing localised areas of pulmonary hypoxia, as well as chemical pneumonitis. Superadded infection may further contribute to the injury. Thus inhaled meconium in an unasphyxiated infant may produce only a mild, benign, self limited, mostly asymptomatic respiratory problem; whereas in an injured and severely asphyxiated lung it may severely exacerbate the clinical symptoms.

The management of MAS is fraught with controversies. Till nearly two decades ago, urgent obstetrical intervention was considered whenever meconium was noted in amniotic fluid as the presence of meconium was thought to be a marker of fetal distress. The change in pathophysiologic concepts of MAS led to a change in recommendation which is currently limited to internal fetal heart monitoring. Over the last two decades a combined approach of obstetric and pediatric management of meconium stained infants has been advocated and gained wide acceptance. This included pharyngeal suctioning by Delee or bulb Catheter soon after delivery of the baby's head followed by tracheal suctioning, often along with a bronchotracheal saline lavage. The value of this combined approach has recently been questioned by many investigators, who have found no significant difference in the incidence of MAS between suctioned and non-sunctioned neonates(15,18). Reports of deleterious effects of suctioning like cardiac arrythmias, apnea(19) and introduction of infection(20) further question the propriety of this approach. Re-evaluation of this management protocol is suggested particularly in view of the evidence linking MAS to intrauterine fetal asphyxia rather than aspiration with first breath(21).

Thick meconium, postdated pregnancies and oligohydramnios are known risk factors for MAS. Recently, prophylactic intrapartum amnio infusion has been tried and found to be successful in MAS(22,23). This beneficial effect, however, needs to be further confirmed by larger clinical trials before acceptance as standard care.

Till such a time that better strategies for management of meconium stained neonates are outlined by further studies, the best course would be the careful intrapartum FHR monitoring and other measures to prevent fetal asphyxia. Immediate airway management, need for suction and intubation should be guided by the asphyxial state of newborn rather than presence of meconium.

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