Pathophysiology and Prevention of Meconium Aspiration Syndrome

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Aspiration of meconium by the fetus at or near delivery may be associated with high infant morbidity and mortality. The meconium aspiration syndrome (MAS) is often preventable, yet cases of MAS continue to occur.

This paper describes the pathophysiology of MAS. The development of MAS involves passage of meconium by a compromised fetus and the subsequent aspiration of that meconium. Respiratory tract obstruction, hypoxia, hypercapnia, and acidosis may all result. Treatment of MAS is primarily supportive, and high mortality rates have been reported with the more severe cases.

There is good evidence that careful suctioning of the infant's upper respiratory tract can in most cases prevent MAS. The suctioning, performed while the infant's head is still on the mother's perineum and prior to the first inspirations, is both a safe and effective preventive procedure.

Delivery of a newborn infant through meconium stained amniotic fluid occurs commonly. The pediatric and obstetrical literature indicates that from 8 to 30 percent of all term deliveries involve some degree of meconium staining.[1-4] The resulting meconium aspiration syndrome (MAS) continues to be a common clinical problem, occurring approximately one third as often as the idiopathic respiratory distress syndrome.[5]

A review of records from the McKay-Dee Hospital Center (a teaching hospital affiliated with the University of Utah) documented the continued existence of MAS. Data were obtained from the delivery room, the nursery, and professional activity study records. For the last six months of 1978, 1,835 babies were delivered, and there were 11 infants who developed MAS. For six months (April through September) of 1979, 1,810 babies were born. During this time period, 12 infants developed MAS and required care in the newborn intensive care unit. (There were no deaths in either time period.)

The infant who develops MAS may present initially with good Apgar scores and be well for several hours, only later to experience respiratory difficulties which may progress to respiratory failure secondary to meconium aspiration pneumonitis. This transient period of absent or minimal symptoms may lull the unwary physician into a false sense of security in dealing with the meconium stained infant.

The family physician is in a unique role in caring for both mother and baby. The family physician is

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in an ideal position to both appreciate the potential seriousness of meconium aspiration to the neonate and to intervene directly during the course of the delivery to help prevent the development of the syndrome.

Pathophysiology

Meconium is present in the fetal intestinal track by 20 weeks of gestation,[6] but is rarely passed by the fetus until after the 37th week.[4]

An important and common factor associated with meconium passage is fetal ischemia. The normal fetal hemoglobin oxygen saturation is approximately 60 percent. When saturation falls to less than half normal (SaO_2 less than 30 percent), the fetal circulation shunts blood differentially to the heart and central nervous system and away from the intestine.[7] This shunting exacerbates intestinal ischemia and results in hyperperistalsis and sphincteric relaxation, both of which may contribute to the passage of meconium into the normally translucent amniotic fluid.[7] (Some authorities place less emphasis on fetal stress and attribute the accentuated intestinal motility and anal sphincteric dilation to increased vagal tone secondary to umbilical cord compression.[8])

Fetal asphyxia also causes pulmonary vasoconstriction, decreased pulmonary blood flow,[9] and possibly, gasping and opening of the fetal glottis.[3] Meconium may then enter the upper trachea and later be aspirated into the lower respiratory tract.

The primary insult to the newborn from aspirated meconium is airway obstruction. Thick, particulate meconium can directly obstruct the bronchioles resulting in local perfusion-ventilation defects, atelectasis, and pulmonary venous desaturation. Obstruction from particulate material may be intermittent with resultant ball-valve effects and the development of local emphysematous areas, air leaks, pneumomediastinum, and pneumothorases. (Air leaks are reported in 9 to 30 percent of MAS cases.[10,11]) Inflammatory infiltrates and edema accompany the aspiration and contribute to luminal narrowing and obstruction. (The inflammatory response is due in part to the relatively low pH of meconium (pH 5.5) and in part to the direct tissue irritant character of meconium.[12]) The overall results of the pulmonary changes are decreased lung compliance and increased right to left shunting which lead to neonatal hypoxia.[6]

Clinical Presentation

Clinical Course

The clinical presentation of the meconium aspiration syndrome varies from mild transient tachypnea to frank respiratory failure. The severity of the symptoms generally parallels the amount of meconium that is aspirated.[1,13] With very minimal aspiration, tachypnea and cyanosis begin shortly after birth, are usually mild, and resolve in 24 to 72 hours. With more significant aspiration, tachypnea, grunting, and cyanosis are more severe and persistent. These signs are accompanied by hypoxia, hypercapnia, and acidosis; and the infant may progress to a state of respiratory failure.

The more severely affected neonates often need immediate resuscitation to establish good initial oxygenation, but nearly all respond well to routine resuscitative efforts.[14] Of the infants who develop respiratory distress—tachypnea, grunting, cyanosis—two thirds do so during the first four hours of life.[14] However, many have a period of several hours during which they appear clinically well only to gradually develop respiratory distress (and even respiratory failure necessitating ventilator support).[1,15]

Differential Diagnoses

The main differential diagnoses include: transient tachypnea of the newborn, pneumonia, and respiratory distress syndrome.

Respiratory Distress Syndrome

The average gestation in MAS infants has been reported at 290 days, 10 days after the expected date of confinement,[1] and this often is helpful in ruling out hyaline membrane disease. In the McKay-Dee Hospital Center series, over 75 percent of the MAS infants were term or post term.

Pneumonia

Differentiation of MAS from group B streptococcal neonatal pneumonia poses a much

greater problem. Authorities generally recommend antibiotic coverage in the management of MAS, because the risk of infant death from unrecognized and untreated newborn group B streptococcal pneumonia is high.[5,15,16]

Prognosis

The prognosis of MAS, like the symptoms, depends on the amounts and consistency of meconium aspirated.[13] However, general mortality figures for babies with MAS average 20 times those of unstained babies.[17]

Vidyasagar et al described their experience with the prognosis for MAS.[14] They examined retrospectively the clinical course of 32 infants with MAS. The average gestational age was 40 weeks and the initial Apgar scores were 5 at one minute and 7 at five minutes. All the depressed infants responded well to initial resuscitation. Within the next 4 hours, 21 of the infants developed significant respiratory distress; and within 12 hours of birth, 17 had advanced to a state of respiratory failure and needed to be placed on ventilators. In spite of artificial ventilation, 11 of the 17 failed to improve their abnormal A-a gradients, low pH values, and low pO_2 values, and died. This high overall mortality (11 deaths/32 cases of MAS, 34 percent) points out the need for identification of infants at risk and for preventive measures.

Risk of Developing MAS

In a prospective study, Gregory and Gooding attempted to identify babies at risk for developing MAS by investigating[1]:

1. The overall frequency of meconium staining of the amniotic fluid
2. The proportion of meconium stained infants who actually aspirated meconium into the tracheobronchial tree
3. The incidence of developing MAS if meconium has been aspirated

They studied 1,000 consecutive births. If "significant" meconium staining of the amniotic fluid was present, an endotracheal tube was passed into the trachea, suction was applied, and the amounts and consistency of any meconium obtained from below the cords was recorded. (They did not intubate infants with essentially normal, but slightly green-tinged fluid who were vigorous at birth.)

They found an incidence of 8 percent of all deliveries to be complicated by meconium staining. They intubated 80 of the neonates, and found meconium below the cords in 46. Thus, 56 percent of those with stained fluid had aspirated meconium. The volume of meconium ranged from 0.5 cc to 7.5 cc. Of the 46 infants with meconium below the cords, 23 had normal and 23 had abnormal results of chest x-ray films. Sixteen infants who had both abnormalities on their chest roentgenograms and meconium below the cords developed MAS. Thus, approximately 20 percent of those with significant staining developed MAS.

Prevention

Thorough suctioning of the infant's upper respiratory tract before the infant breathes has become the primary prevention for MAS. Review of the pediatric literature revealed only one reference, a letter to the editor,[18] which questioned the need for removal of meconium from the airways of meconium stained infants. All of the other authors cited recommended suction to clear the meconium from the respiratory tract.

Even though gasping reflexes have been observed in stressed fetal monkeys before delivery,[19] evidence from other animal studies suggests that only those who aspirate meconium during or after delivery are at risk to develop MAS.[13] Ting and Brady presented evidence for the efficacy of human airway suctioning in 1975.[17] They studied retrospectively over 1,000 newborn intensive care unit admissions, eight percent of which had meconium staining. Some of the stained infants had received tracheal suctioning at birth; of those suctioned, 70 percent developed no respiratory symptoms. There was one death in the suctioned group (a child with Down syndrome and congenital heart disease). Of the infants who did not receive tracheal suctioning, 60 percent developed symptoms, and eight died from respiratory failure. Noting the beneficial effects of suctioning, Gregory and Gooding[1] instituted a policy of endotracheal intubations and suctioning of all meconium

stained infants. Of the subsequent 5,700 births at their institution, eight percent were meconium stained, but all survived and none required artificial ventilation.

Current Management

Anticipation of a potential MAS is the key to prevention. The physician should recognize situations predisposing to meconium aspiration.

Obstetrical situations predisposing to meconium staining of the amniotic fluid include:

1. Conditions related to fetal ischemia[20]:

Maternal hypertensive states
Type II or late deceleration fetal heart tone changes
Chronic pulmonary disease
Post maturity
Anemia
Placenta previa
Abruptions
Cord prolapse

2. Conditions related to cord compression (eg, nuchal cords)

These are often not clinically detectable other than from changes of variable deceleration in fetal heart rate patterns.

Type II dips, or late deceleration changes, accompanied by meconium staining of the fluid is an ominous sign that often signals severe fetal distress. The status of the fetus in this situation needs to be carefully assessed with scalp pH monitoring, and immediate cesarean section may become necessary.[20]

The physician managing the labor and delivery needs resuscitation equipment immediately at hand and an established resuscitation plan.[21]

Equipment

Stethoscope
de-Lee suction
Warmer
"Whistle-tip" suction catheter
Oxygen
3-4 mm internal diameter endotracheal tubes
Laryngoscope, neonatal
Wall suction
Newborn bag, mask, and endotracheal tube connection

Procedure

Immediately on delivery of the baby's head, and before the infant breathes, the whistle-tip catheter or the de-Lee trap is used to thoroughly suck out the nasopharynx and the oropharynx. The suction may be passed into the baby's stomach as well. (Some clinicians have developed a preference for the de-Lee trap, as it allows them to better assess just how thick the meconium is by noting how strong a suction is needed to remove the material.) The baby may remain with only the head delivered for the interval between several contractions. This time is often long enough for nasopharyngeal suctioning to remove nearly all of the meconium from the upper airway before the baby makes strong breathing efforts. The consistency of the meconium will dictate subsequent management (Figure 1).

1. Thin Fluid

If the amniotic fluid is thin, watery, and only mildly green-stained, oro- and nasopharyngeal suctioning should suffice to clear the majority from the infant and any small amounts that may be aspirated should not be clinically significant.[13,22] If the infant with mild staining is vigorous and crying at delivery, endotracheal intubation is unnecessary, as the intrapartum oropharyngeal suctioning is very effective in clearing the meconium.[22]

2. Thick Meconium Staining

If the meconium is "pea soup," or "particulate," vigorous attempts need to be made to clear it from the airway. The intrapartum suctioning with the de-Lee suction may not alone be effective in clearing this thick meconium from the upper respiratory tract.

In this situation, after de-Lee suctioning and as soon as the infant is delivered and placed in the warmer, the vocal cords should be visualized using an infant laryngoscope. If no meconium is visualized at the cords, endotracheal intubation is not necessary.[22] If meconium is seen at or below the cords, an endotracheal tube is placed into the trachea and mouth suction (through the surgical mask) is applied as the tube is slowly withdrawn. (Several of the family practice residents have tried suctioning with a whistle-tip catheter inserted *through* the endotracheal tube, but they have found that thick meconium is difficult to remove in

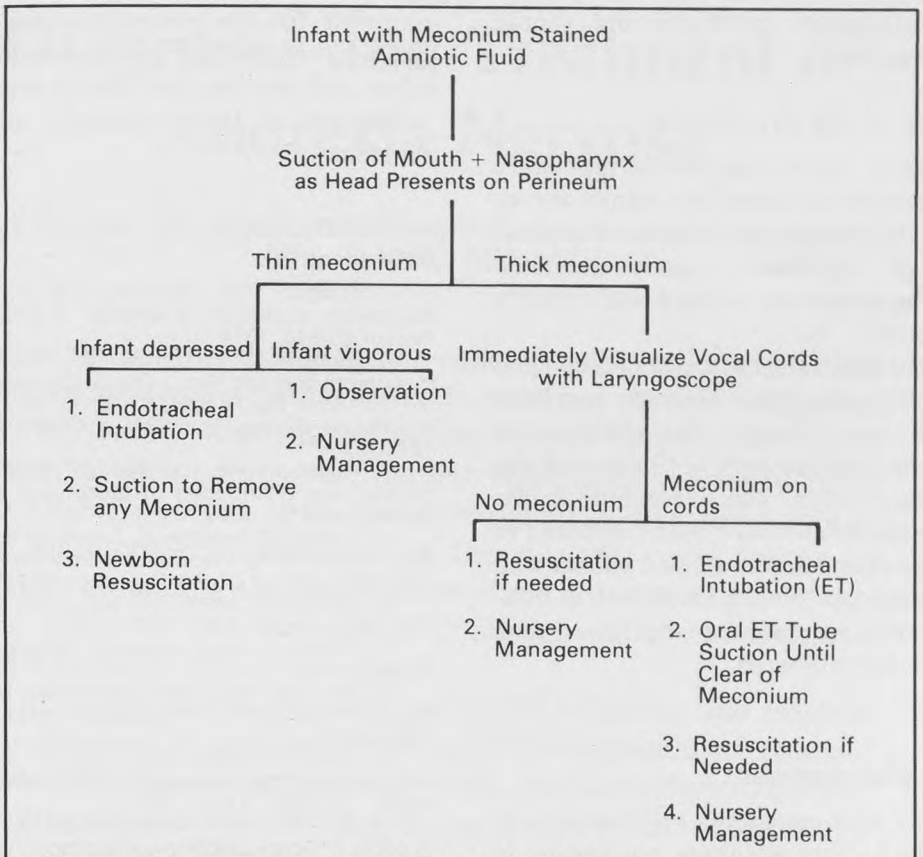

Figure 1. Management of the delivery of infants with meconium stained amniotic fluid

this way.) Occasionally, several intubations have been necessary to remove large meconium masses plugging the trachea.

There are reports of infants with no meconium visualized on the cords and yet with meconium obtained from the trachea on suctioning[1]; however, in Carson's prospective study of 273 infants with meconium staining, intrapartum suctioning of the infant while still on the perineum plus visualization of the cords resulted in only one infant developing MAS and his disease was mild.[22] (Only 2 of Carson's 273 infants had any meconium at all visualized on their vocal cords after the intrapartum suctioning. This is in marked contrast to the 56 percent of infants who had meconium found in the trachea when the infant was suctioned *after* delivery was completed.[1])

Some physicians have attempted lavage of the trachea through the endotracheal tube in an effort to better clear any meconium.[23] Using such a protocol, lavage was tried by the residents and teaching staff. Two or 3 ml normal saline were instilled but usually only a few tenths of a ml were recovered on return suction. These infants had more rales after the attempts at lavage than they did before. Some perinatologists report x-ray evidence of iatrogenic wet lung syndrome from attempts at a similar lavage procedure.[22] The lavage

may add to respiratory morbidity and should probably be abandoned.

Complications of the Procedure

The intrapartum airway suctioning procedure has not been reported to cause any significant infant morbidity.[1,22] There are no reports of induced laryngospasm or significant vagally mediated bradycardia. The procedure is rapid and requires minimal equipment.

In experienced hands the cord visualization and endotracheal intubation have likewise not been associated with dysrhythmias.[1] The visualization and intubation procedures have not produced significant infant depression. The five-minute Apgar scores of intubated infants have been compared to the Apgar scores of meconium stained infants who were not intubated, and the Apgar scores of intubated infants were not found to be lower than those who were not intubated.

Postdelivery Management

For newborns with meconium staining who do not develop MAS, Trendelenberg positioning in the nursery, gentle chest percussion, and intermittent oral-pharyngeal suctioning are all recommended.[15] These modalities should help to clear any residual meconium from the airways. However, it has been shown that labeled meconium travels fairly rapidly toward the periphery of the lung[13]; and although physical therapy, positioning, and nursery oral suctioning would logically seem beneficial in clearing any residual meconium, they have not been subjected to systematic evaluation.

Management of the more severe cases of meconium aspiration syndrome necessitates frequent arterial blood gas determinations, positive-end-expiratory airway pressure,[24] ventilatory support, antibiotic coverage, and intensive newborn supportive care.

Even with modern newborn intensive care modalities, the mortality from MAS can be high and the risk of developing MAS may be overlooked by the unwary physician. It is especially easy to be lulled into a false sense of security if the infant responds well to initial resuscitation, but these infants may later develop the meconium aspiration symptom complex if the meconium is not removed during the delivery. The family physician, responsible for the obstetrical and newborn care, can, in the delivery room, carefully suction the infant and play the key role in preventing the development of this potentially severe newborn problem.

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