DISSERTATION ON

IMMEDIATE CLINICAL OUTCOME OF NEWBORNS WITH MECONIUM STAINED AMNIOTIC FLUID IN AN URBAN REFERRAL CENTRE

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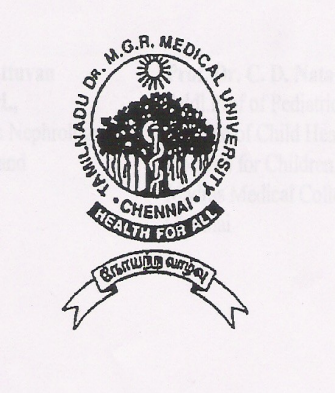

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Certificate

CERTIFICATE

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*I declare that this dissertation entitled "**IMMEDIATE CLINICAL OUTCOME OF NEWBORNS WITH MECONIUM STAINED AMNIOTIC FLUID IN AN URBAN REFERRAL CENTRE"** has been conducted by me at the Institute of Child Health and Hospital for children, under the guidance and supervision of my unit chief, Director & superintendent **Prof.Dr.* **R.KULANDAI KASTHURI, *M.D., DCH**. It is submitted in part of fulfillment of the award of the degree of M.D [Pediatrics)] for the March 2007 examination to be held under The Tamil Nadu Dr. M.G.R. Medical University, Chennai. This has not been submitted previously by me for the award of any degree or diploma from any other university.*

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INTRODUCTION

The detection of meconium-stained amniotic fluid during labour often causes anxiety in the delivery room because of its association with increased perinatal mortality and morbidity. However, experts continue to debate whether the risk of harm is associated with the meconium itself, or whether the overall risk is increased because of the underlying condition leading to the passage of meconium.
The obstetric literature is fraught with controversy and unanswered questions regarding the significance of meconium in the aminotic fluid and the appropriate management protocols that should be followed when it is discovered. It is believed by some medical experts that the passage of meconium is triggered by fetal stress, such as hypoxia or asphyxia, and that the presence of meconium in the fluid may be considered an indicator of fetal distress. Others point out that the presence of meconium in the amniotic fluid also may be a result of gastrointestinal maturity. Thus, the presence of meconium in the amniotic fluid is only one factor to be considered along with many variable and clinical markers that may help to explain the etiology and timing of irreversible brain damage. There are many variables and unanswered questions concerning the time it takes for meconium to initiate pathologic placental changes or to cause damage to umbilical cord vessels for meconium to be a useful marker to time brain injury.

MECONIUM

Meconium is a thick, odorless, blackish green material, first demonstrable in the fetal intestine during the third month of gestation.
It is a sterile mixture of water (75-95%), mucopolysaccharides (80% dry weight), gastrointestinal secretions (bile salts and pancreatic, liver enzymes), solids, (vernix caseosa, lanugo, and squamous cells), blood minerals and lipids (free fatty acids).It's pH ranges from 5.5 to 7. Meconium first appears in the fetal ileum between 10 to 16 weeks of gestation[1].
The term meconium is derived from ancient Greek word meconium-'arion', or opium like, from the Greek word 'mekoni' meaning poppy juice. Aristotle coined the term, because he believed that the substance induced fetal sleep[1].

INCIDENCE

Meconium staining of amniotic fluid (MSAF) occurs in approximately 10% to 26% of all deliveries, with the highest rates reported from North America[1-5]. Although meconium appears in the intestine very early in gestation, MSAF rarely occurs before 38 weeks of gestation. Incidence of MSAF increases thereafter and approximately 30% of newborns have MSAF if born after 42 weeks of gestation[1]. Meconium stained liquor is rare in premature infants (<5 percent of preterm pregnancies); if it does occur, there may be an association with infection and chorioamnionitis. The incidence of meconium aspiration syndrome varies between 1 and 5 percent of all deliveries where there has been meconium stained liquor, with higher rates reported from North America compared to Europe[1-3,5].
There are a number of factors associated with an increased risk of developing meconium aspiration syndrome. These include lack of antenatal care, black race, male fetus, abnormal fetal heart rate monitoring, thick meconium, oligohydromnios, operative delivery, poor activity, pulse, grimace, appearance and respiration (APGAR) scores, no oropharyngeal suctioning and the presence of meconium in the trachea[8].

ETIOLOGY

Many theories have been proposed to explain the passage of meconium in utero; however, the precise mechanisms remain unclear. The increased incidence of MSAF with advancing gestational age probably reflects the maturation of peristalsis in the fetal intestine[1]. Motilin, an intestinal peptide that stimulates contraction of the intestinal muscle, is in lower concentration in the intestine of premature Vs postterm infants. Umbilical cord motilin concentration is higher in infants who passed meconium than in infants with clear amniotic fluid. Intestinal parasympathetic innervation amd myelination also increase throughout gestation and may play a role in the increased incidence of passage of meconium in late gestation. Inutero passage of meconium is also associated with fetal asphyxia and decreased umbilical venous blood PO_2[1].

Experimentally, intestinal ischemia produces a transient period of hyperperistalsis and relaxation of anal sphincter tone, leading to the passage of meconium. The fetal diving reflex, which shunts blood preferentially to the brain and heart and away from the visceral organs during hypoxia may enhance intestinal ischaemia[6].

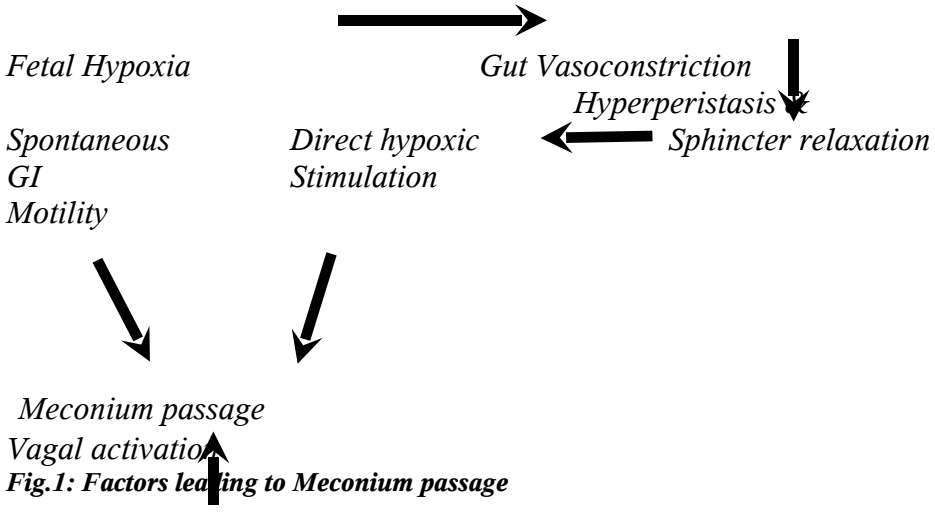


Fig.1: Factors leading to Meconium passage

MSAF AND FETAL DISTRESS

Is meconium an independent marker of fetal distress ?

Previously meconium passage was a sign of fetal death or impending death. Several modern studies have examined data collected around the time of birth to correlate meconium passage and fetal distress. Such data must be viewed cautiously because the association of events of labour and particularly of delivery does not necessarily represent the intrauterine environment at the time of meconium passage. For eg.Apgar score analysis to correlate fetal distress with meconium stained fluid is misleading because it is affected by tracheal intubation. For meconium to be an independent marker of fetal distress, infants with meconium staining would have adverse perinatal outcomes without other evidence of fetal compromise. This has not been found to be the case in all[12].

In general, the neonatal outcome is in no way different in meconium stained amniotic fluid and clear amniotic fluid in labours with similar FHR abnormalities like normal heart rate pattern, fetal tachycardia, decreased FHR variability and absence of acceleration.

Thus meconium is not an independent marker of fetal distress, but rather of an event like cord compression and often an environment such as decreased amniotic fluid that may predispose to or be a consequence of fetal compromise. Some fetuses with meconium stained fluid will be compromised but most will not. As Miller et al have stated the presence of meconium in the amniotic fluid without signs of fetal asphyxia is not a sign of fetal distress[13].

MSAF found at the onset of labour reflects a previous event which may or may not have been hypoxia and so the significance of such a finding is not clear. Routine amnioscopy, amniocentesis or artificial rupture of membranes to observe the colour of liquor have been suggested and put into practice with varying degrees of enthusiasm. However, such screening methods have not been subjected to evaluation by prospective randomized controlled trials[7].

Exactly why some babies suffer varying degrees of the meconium aspiration syndrome and others do not is unclear but two important observations, strongly associated with bad outcome, require mention.

(a). Thick, Viscid MSAF
(b). MSAF in the presence of an abnormal fetal heart rate pattern.

(a) Thick, Viscid MSAF

Thick MSAF implies that there has been coexisting oligo-hydromnios, which in itself is associated with fetal compromise. When MSAF occurs in the post mature fetus and oligohydromnios supervenes, an often fatal combination seems to coexist[18]. The airways become inspissated with this tenuous material which cannot be easily cleared by the neonate and so any preexisting hypoxia appears exaggerated. MSAF and oligohydromnios is a particularly difficult clinical problem because the sparsity of fluid can mean that it may remain undetected until the baby is delivered. Extreme vigilance is therefore required in labouring patients thought to have no amniotic fluid.

b) MSAF in the presence of an abnormal fetal heart rate pattern

The presence of MSAF without an abnormal fetal heart rate pattern does not appear to jeopardize fetal outcome, but when late decelerations are present the risk of meconium aspiration seems to be enhanced and the neonatal outcome may be prejudiced[18].

INTRAPARTUM FETAL MONITORING

FHR MONITORING

Since the introduction of fetal heart auscultation in the early 19-th century by Evory Kennedy, the estimation of FHR has evolved considerably and became the main stay of intrapartum fetal monitoring. Continuous electronic FHR monitoring was introduced in the late 1960's and since then has attained wide acceptance in clinical obstetrics. During the interpretation of FHR recording, a systematic approach should be used to assess the following aspects.

1. Baseline rate
2. Baseline variability
3. Presence of accelerations
4. Presence of decelerations and the type and severity.

A normal FHR trace is one with a baseline between 110 and 150 beats per minute showing good variability (more than 5bpm) with accelerations and no decelerations. In the presence of a normal FHR tracing, there is only a 2% chance for the fetus to be acidotic (pH<7.20) and a 1% chance for it to have a 5 minute apgar score <7. A definitely ominous or pathological FHR trace is one with a baseline tachycardia (>150 beats/minute) with absent variability and repetitive late or variable decelerations. Other sinester patterns are prolonged severe bradycardia (<80 beats/minute) for >10 minutes or a sinusoidal pattern without accelerations[5].

PREDICTIVE VALUE OF ABNORMAL CTG IN LABOUR

Using a low 5 minute Apgar score as a end point (Apgar<7), abnormal intrapartum FHR has a high negative predictive value of over 90%, but a low positive predictive value of about 30% . This means that a normal trace indicates a fetus that is not hypoxic, while an abnormal trace is associated with a large number of false positives[5].

MECONIUM ASPIRATION

Meconium found below the vocal cords defines meconium aspiration syndrome. It occurs in approximately 35% of live births with MSAF or in approximately 4% of all livebirths[1]. Meconium aspiration syndrome describes a wide spectrum of respiratory disease, ranging from respiratory disease to severe disease and death despite mechanical ventilation. It typically presents as respiratory distress, tachypnea, prolonged expiratory phase and hypoxemia soon after birth in an infant heavily stained on the nails, hair and umbilical cord with meconium or born through thick meconium. Less severe meconium aspiration, typically of nonparticulate meconium, may present with the appearance of a

pneumonitis with mild increased work of breathing or peaceful tachypnea reaching a peak at one to three days and resolving slowly over the first weeks of life[9].

Aspiration of meconium into the distal airways can occur either antenatally or postnatally, but in the majority of the affected infants, the exact timing is not clear. It leads to inflammation of pulmonary tissues and hypoxia. There is also evidence that the free fatty acids in meconium strip away alveolar surfactant[9]. In severe cases, the pathological progresses to persistent pulmonary hypertension, other morbidity and death. Even with prompt and appropriate therapy, seriously affected infants frequently die or suffer long-term neurological sequelae.

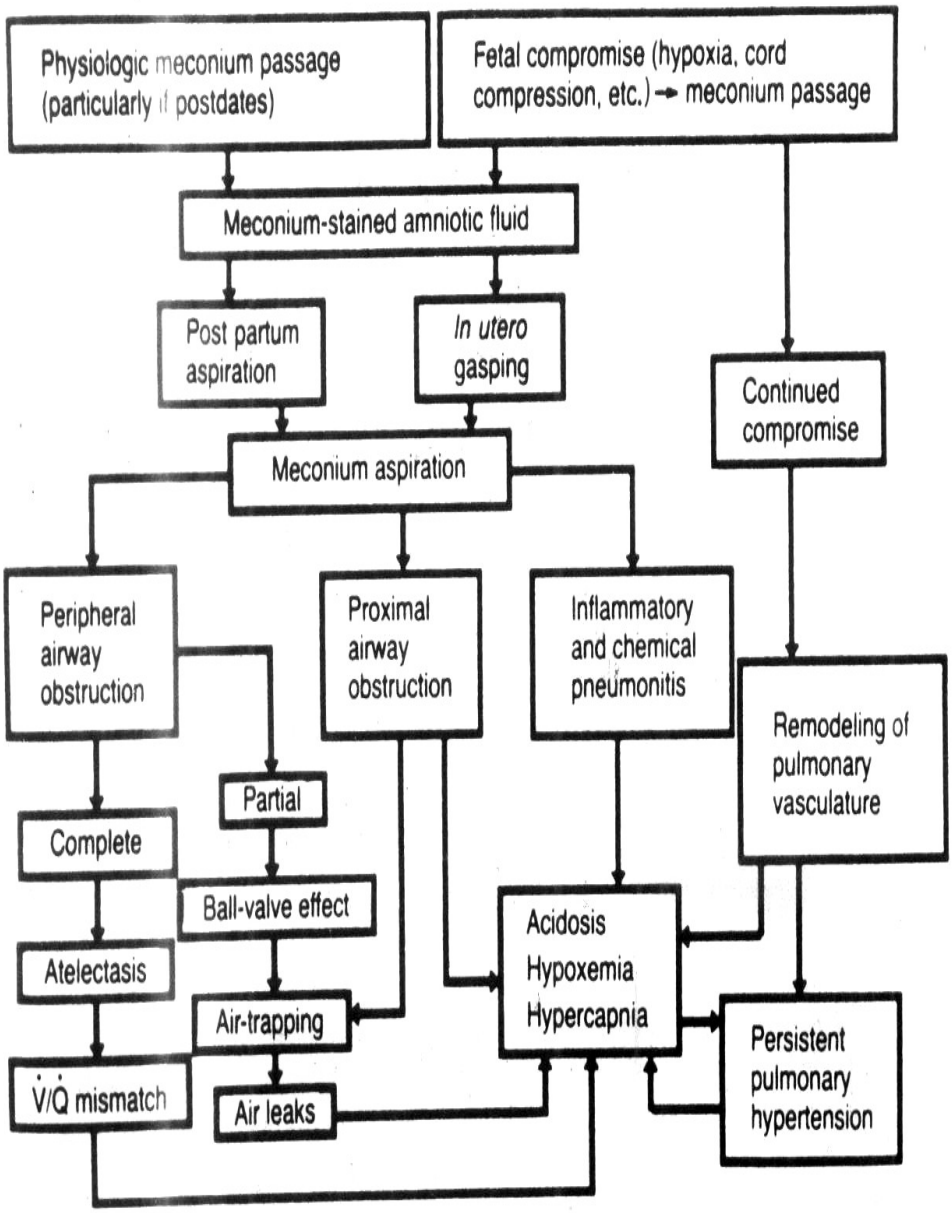
PATHO PHYSIOLOGY

The quantity of meconium will affect the appearance and viscosity of the amniotic fluid, which ranges from a green tinged, thin meconium stained fluid to one with a thick, pea soup consistency[1]. Meconium in the amniotic fluid may stain the umbilical cord, placenta and fetus. The time from passage of meconium to delivery of the infant can possibly be estimated from the depth of penetration of meconium pigment into the placental membranes with subsequent uptake by placental macrophages. Under normal circumstances fetal respiration is associated with movement of fluid from the airways out into the amniotic fluid. When fetal distress is present gasping may be initiated in utero. Under these circumstances amniotic fluid and particulate matter contained therein may be inhaled into the large airways. It may be present in the trachea or larger bronchi and may result in airways obstruction at delivery[8]. Meconium and squames may be found as far as alveoli in stillborn infants, and it is now widely recognized that meconium aspiration may occur antenatally. After airbreathing has commenced, especially if accompanied by gasping respirations, there is a rapid distal migration of meconium within the lung.

Pathophysiologically, the pulmonary problems are due to airway obstruction and a ball value phenomenon created by the presence of meconium within the airways. Areas of atelectasis develop within the lung, resulting from total small airway obstruction, adjacent to areas overexpansion from gas trapping in regions with partial obstruction[2]. Airleaks, including pneumomediastinum and pneumothorax, are more likely to occur after aspiration of meconium and cellular debris. Chemical inflammation secondary to aspirated meconium presumably leads to pneumonitis. There is also a concentration – dependent inhibition of surfactant by meconium, all of which could aggravate atelectasis, hypoventilation and intrapulmonary shunting. PPHN can be a major clinical problem in infants with meconium aspiration. The etiology of increase in pulmonary vascular resistance is some what controversial. Hypoxic pulmonary arterial vasoconstriction is most likely a major attributing factors.

Postmortem examination of lungs from infants with severe MAS reveals meconium, vernix, fetal squamous cells and cellular debris in the air spaces from the airways to the alveoli. An inflammatory response with polymorphonuclear leukocytes, macrophages, and alveolar edema maybe observed, but large quantities of meconium may be present without histologic signs of inflammation. Hyaline membrane formation, pulmonary hemorrhage, and necrosis of pulmonary microvasculature and parenchyma can occur. Platelet rich microthrombi in small arteries and increased muscularization of distal arterioles have been described in some infants during of MAS.

DIAGRAM OF PATHOPHYSIOLOGY[2]:



CLINICAL FEATURES

GENERAL APPEARANCE

The baby is usually mature or post mature, with long fingernails and a dry skin which soon starts to flake. The skin, nails and umbilical cord are often stained greenish yellow. The baby is not usually febrile, unless secondarily infected.

RESPIRATORY:

The infant has tachypnea, which may exceed 120/mt. Because he is mature with firm rib cage, marked sternal retraction as seen in preterm neonates with RDS is rare, but intercostals and subcostal recession, the use of accessory muscles and flaring of the nostrils are usually present. An expiratory grunt may be heard. The meconium in the airways causes widespread sticky crepitations and occasional rhonchi, airtrapping and an over distended chest with an increased antero posterior diameter. The baby may

remain symptomatic for only 24hrs, or he may be very dyspnoic for 7 – 10 days before recovery[18].

CARDIOVASCULAR

In the absence of asphyxial damage to the myocardium there are no specific cardiovascular features of MAS. Hypotension suggests myocardial damage, as do signs of congestive heart failure. In uncomplicated MAS the heart tends to be around 110-125/min and the blood pressure is maintained within normal range. If PPHN develops S_2 may remain single and there may be the murmur of tricuspid incompetence[18].

CENTRAL NERVOUS SYSTEM

Depending on the severity of the coexisiting neurological insults the baby may behave normally or show any of the neurological features of birth asphyxia, ranging from the common over alert, stage I HIE to the rare, flaccid unresponsive ventilated baby in stage III. Convulsions may occur and jitteriness persists for days.

INVESTIGATIONS

Chest X-ray

The chest radiographs shows coarse, irregular pulmonary densities with areas of diminished aeration or consolidation[2]. Pneumothorax and pneumomediastinum are common in infants with severe meconium aspiration syndrome. Hyperinflation of the chest and flattening of the diaphragm are sometimes noted on radiograph. Cardiomegaly also might be detected, possibly as a manifestation of the underlying perinatal hypoxia.

ECG, ECHO

In uncomplicated MAS, both will be normal. If associated with severe intrapartum asphyxia there may be ECG changes suggesting subendocardial ischemia and the echocardiogram will show reduced cardiac contractility in those going to develop PPHN, changes in the systolic time interval will be seen on M-Mode echocardiography.

Blood Gases

Arterial blood gases characteristically reveal hyopoxemia with evidence of right to left shunting. Hyperventilation can result in respiratory alkalosis, although infants with severe disease usually have combined respiratory and metabolic acidosis secondary to hypoxia and respiratory failure.

PREVENTION OF PASSAGE OF MECONIUM IN UTERO

Mothers at risk for uteroplacental insufficiency include those with preeclampsia or increased blood pressure, chronic respiratory or cardiovascular disease, poor uterine growth, post term pregnancy. These women should be carefully monitored during pregnancy and the fetal heart rate should be monitored during labour, with fetal scalp blood samples obtained for pH determination when indicated[6].

PREVENTION OF MECONIUM ASPIRATION PNEUMONIA

American Academy of Pediatrics/American College of Obstetrics and Gynecology guidelines recommend that when meconium of any consistency is present intrapartum, the obstetrician should clear the infant's nose and oropharynx before delivery of the shoulders or chest. This can be done with either a bulb syringe or a DeLee suction catheter, which are equally effective[10].

During the initial assessment at a delivery complicated by meconium, the pediatrician should determine whether the infant is vigorous, demonstrated by heart rate >100 beats per minute, spontaneous respirations, and good tone (spontaneous movement or some degree of flexion). If the infant appears vigorous, routine care should be provided, regardless of the consistency of the meconium[10]. If respiratory distress develops or the infant becomes depressed, the trachea should be intubated under the

direct laryngoscopy and intratracheal suctioning performed. In questionable cases, it is safer to intubate and suction, as MAS can occur in infants delivered through thinly stained amniotic fluid[17].
Tracheal suctioning should be performed using an appropriately sized endotrocheal tube and a large bore meconium aspirator. In the presence of severe asphyxia, it may not be possible to clean the trachea of all meconium, and clinical judgment must be used to determine the amount of suctioning. However, meconium aspiration syndrome continues to occur in infants who are adequately suctioned in the delivery room. Aspiration of meconium or amniotic fluid inutero probably occurs in some with MAS, particularly in those with perinatal asphyxia. Studies have found that 7% to 10% of meconium – stained infants had meconium in their tracheas even when there was not any meconium visible at the vocal cords.

ANTENATAL THERAPIES

Amnioinfusion

The use of transcervical amnioinfusion with normal saline solution in women whose labor is complicated by meconium stained amniotic fluid and repetitive fetal heart rate decelerations may reduce the incidence of meconium aspiration[7]. The rationale behind amnioinfusion is that by increasing the liquor volume, meconium will be diluted. In addition, in cases of oligohydramnios, the increased volume will prevent cord compression, subsequent hypoxia, fetal gasping and passage of meconium. However, the use of amnioinfusion requires further evaluation, as the therapy is associated with a number of complications, including a higher incidence of instrumental delivery and endometritis.

Timing and mode of delivery

In pregnancies that continue past the due date, induction as early as 41 weeks may help prevent MAS by avoiding passage of meconium. Delivery mode does not appear to significantly impact the risk of aspiration. Although most studies suggest that infants with MAS are more likely to be delivered by caesarean section than by vaginal delivery, this is largely due to suspicion or confirmation of fetal compromise. There is currently no evidence to suggest that MAS would be prevented by elective delivery by caesarean section of infants with meconium stained liquor.

MANAGEMENT OF MAS

The aim of treatment is to support the baby until his alveolar macrophages clear the debris and lung function returns to normal. Treatment consists of.

1. Conventional management &
2. Non conventional management.

Conventional management consists of oxygen, mechanical ventilation, and fluid/nutritional support[14].
Non conventional management consists of high frequency ventilation (HFV), exogenous surfactant, inhaled nitric oxide, liquid ventilation, and extracorporeal membrane oxygenation (ECMO)[14].

OXYGEN

The administration of oxygen is critically important in infants with MAS and in many infants is the only respiratory therapy needed. The pulmonary vasculature in the term infants is exquisitely sensitive to oxygen tension, and failure to overcome hypoxaemia almost inevitably will lead to progressive pulmonary hypertention. Oxygen should be administrated early and liberally in any baby suspected of having inhaled meconium. The suggested target range of oxygen saturation is 94-98%; target PaO_2 60-90 mmHg.

NASAL CPAP

CPAP at pressure of 4-7 cmH_2O may increase the PaO_2 in babies with MAS. However, it is likely to increase the risk of pneumothorax in a disease characterized by air trapping, and the use of a nasal

prong in a term neonates usually makes them irritable and restless, with a fall in PaO_2.

INTUBATION & POSITIVE PRESSURE VENTILATION

INDICATIONS

Persistent hypoxaemia (SaO_2<90%, PaO_2<50%) in 100% oxygen, respiratory acidosis with pH<7.20[11].

METHOD

Infants with MAS are frequently very difficult to manage once intubated and often require high peak inspiratory pressure (30-35cmH_2O) to achieve gas exchange. Most evidence favours a high positive end expiratory pressure (6-8cm H_2O) and a long expiratory time. The latter can be achieved using ventilatory rates of 40-60 breaths per minute, with an inspiratory time of 0.5-0.6 seconds[11]. When there is concomitant pulmonary hypertension, deep sedation should be maintained after intubation and muscle relaxation should be continued if the disease is severe.

GENERAL SUPPORTIVE CARE

It includes cardiovascular support such as volume and inotrope therapy, fluid restriction, IV therapy and nil orally until the respiratory distress is settled.

ANTIBIOTICS

Differentiating between bacterial pnenumonia and meconium aspiration by clinical course and chest x-ray findings may be difficult. Although few infants with MAS have documented infections, the use of broad spectrum antibiotics (eg. Ampicillin and gentamycin) is usually indicated in infants when an infiltrate is seen on chest radiograph[14].

HFV

The most commonly used methods of HFV are high frequency oscillatory ventilation and high frequency jet ventilation. No clinical trials have demonstrated benefits of either type of HFV compared to conventional therapy.

SURFACTANT

Because of meconium associated surfactant dysfunction, it would seem logical that use of exogenous surfactant could benefit babies with MAS. To date, only the small trial of Findlay et al has assessed such therapy in babies with MAS in randomized controlled trial. They found exogenous surfactant to be benefit. However, they used one and a half times the standard surfactant dose which was not administrated in a bolus, but via a low infusion over 20 minutes[15].

NITRIC OXIDE

Over the last 2 years, several large investigations have assessed the use of inhaled nitric oxide in the management of respiratory failure in newborn infants . Approximately half of the infants in these trials had MAS as their principal respiratory disorder. But NO has not shown to be superior to conventional therapy in preventing death or the need for ECMO[16].

ECMO

It remains the therapy of last resort for MAS. Infants with MAS make up the most common diagnostic category of those treated with ECMO (36%) and have the highest survival rate of all ECMO treated babies. But there is relatively high associated morbidity with this therapy, which most commonly involves venoarterial cardiopulmonary byepass, ultimately necessitating ligation of right carotid artery[19].

Thus this therapy should not be taken lightly and should still be reserved only for those infants who have a high likelihood of death without ECMO.

LONG TERM SEQUELE

1. MSAF by itself does not predict cerebral palsy. The overall incidence is 2 in 1000 live births .Finding of meconium alone does not prove that the infant experienced a degree of asphyxia sufficient for later neurologic abnormality. However MSAF with low APGAR (0-3) subsequently developed cerebral palsy in 9.4%[13].
2. Long term pulmonary function testing at, 6 to 11 years after birth revealed large components of obstructive airway disease like spontaneous wheezing, exercise induced bronchospasm suggesting small airway injury and diseases in up to 40%. Chronic lung disease is relatively uncommon, occurring in only 5% of ventilated infants.
3. Infants who have undergone ECMO that is who have typically needed the greatest amount of ventilatory support to survive, it is the barotrauma and high O_2 requirement likely contribute to the development of lung disease.

LITERATURE REVIEW

Wong SF, et al., in a retrospective study at Kowloon Hong Kong in July 1996 - June 1999 studied the relative risk of 'fetal distress' in pregnancy associated with meconium stained liquor at different gestations. Total of 9542 singleton pregnancies, delivered in a tertiary obstetric unit in Hong Kong were included in the study. They attempted to quantify the relative risk of non-reassuring CTG or fetal distress in pregnant women complicated by MSAF for preterm, term and post term deliveries. 20.4% identified as having meconium stained liquor, ranging from thin to thick staining. There was a strong association between incidence of meconium stained liquor ($P < 0.0005$) and moderate / thick meconium stained liquir with advanced gestational age. The incidence of non-reassuring CTG in women presenting with meconium stained liquor was significantly higher (9.8% Vs. 6.4%). The relative risk of non-reassuring CTG in women with meconium stained liquor increased with more advanced gestation. Premature labour was associated with higher incidence of fetal distress but the presence of meconium did not pose an additional risk[20].

Zhu L, et al in a Cohort Study at Beijing, China in Feb. 2003 to study the epidemiology of meconium stained amniotic fluid on hospital basis. 6206 deliveries were divided into meconium stained amniotic fluid and normal amniotic fluid groups. Incidence of MSAF was 16.4%. There was no significant difference on maternal medical complications between two groups. The average APGAR score in meconium stained group was lower than that in normal group ($P=0.001$). Relative factors on meconium stained fluid were maternal parity, gestational weeks $\geq$ 42 weeks and large gestional age babies. Morbidity of newborn with MSAF was higher[21].

Alchalabi H, et al in a prospective study between May 1997 to September 1997 at Princess Badea Teaching Hospital, Irbid to explore details of clinical relationship between MSAF in labour, abnormal fetal heart pattern and meconium aspiration. Incidence of MSAF was 8.5%. LSCS incidence was 10.5%, indication being fetal distress which was diagnosed by abnormal fetal heart rate pattern. Many infants in MSAF group had a low Apgar score and required ventilation at birth.

5% developed meconium aspiration with a mortality of 15.6%. There was an association between MSAF, abnormal FHR pattern in labour and a low Apgar score which should be considered a high risk situation. They concluded that meconium aspiration a problem that occurs with particulate meconium was significantly related to abnormal fetal heart rate pattern and longer length of labour[22].

Berkers MD, et al., in a cohort study at University of Texas Health Science Centre at San Antonio in July 1994, to determine the risk of adverse neonatal outcome associated with MSAF independent to antepartum and intrapartum abnormalities. 2200 consecutive deliveries were examined and FHR tracings analyzed independently. Thick MSAF increased the risk of adverse outcome in more than three fold (RR 3.2, 95% confidence interval 2.0 - 5.2). This was independent of FHR tracing abnormalities or maternal hypertension, kidney or heart disease. They concluded that thick meconium alone should alert the physician to a high risk fetal condition[23].

Gupta V, et al., in a prospective study at Banaras Hindu University, Varanasi in April 1996 to find out antenatal, intra partum and neonatal attributes in MSAF. 1426 live births occurring in 1500 consecutive deliveries over 1 year period were studied. Incidence of MSAF was 14.3%. Thick meconium 69.11%. Hepatitis in mother, fetal distress during labour and IUGR were significant factors associated with MSAF. Consistency of meconium had direct bearing on neonatal outcome. Severe birth asphyxia occurred in 27% with thick meconium and 6.3% with thin meconium. MAS was observed in 6.38%. All deaths occurred in thick meconium group and were associated with SGA[3].

Sheiner E, et al., in a prospective study at Faculty of Health Sciences, Scroka University Medical Centre, Israel in Jan 2002 to find out the effect of meconium on perinatal outcome. They compared perinatal outcome of parturients with thick and thin MSAF to those with clear AF. The rate of MSAF

was 18.1%. Thin meconium - 13.3%. Thick meconium - 4.8%. The rate of oligohydromnios was significantly higher among pregnancies complicated with thick meconium. (OR 7.2, 95% CI), 2.1-24.1; P=0.002). A statistically significantly higher risk for neonatal intensive care unit admission was observed among patients with thick meconium as compared to those with clear amniotic fluid (p=0.006), even after adjustment for oligohydromonios and abnormal fetal heart rate patterns. Therefore, concluded that thick MSAF should be considered a marker for possible fetal compromise, and lead to careful evaluation of fetal well-being[24].

Greenwood, et al., in a prospective study at Dublin, Ireland in July 2003 to examine the incidence of meconium that which have only been passed intrapartum and to determine its neonatal associations and whether its absence is a useful sign. 8394 low risk laboring women at term with clear amniotic fluid at early amniotomy were studied. Meconium was passed in 5.2% of labour but was not detected until delivery of the fetal head in 51.5%. It was associated with moderate - severe acidosis, low Apgar score at 5 minutes and neonatal seizures. However, the sensitivity for these outcomes of the intrapartum passage of meconium and particularly its detection before delivery was very poor. Although correlated with adverse neonatal outcome, most affected infants had clear amniotic fluid throughout labour. The presence of clear amniotic fluid is an unreliable sign of fetal well being[25].

David AN, et al., conducted a prospective study at Nigerian University Teaching Hospital during March-June 2003 to find out incidence and factors associated with meconium staining of the amniotic fluid. The incidence of MSAF was 20.4%. The rate increased with gestational age. No case was found below 37 weeks (p = 0.001). Primi parity, PROM, obstructed labour were more often associated with MSAF[26].

Gonzalez De Dios J did a retrospective study at Department of Pediatrics, Hospital Universitario San Juan, Alicante in Jan 1998 to find out the neonatal morbidity associated with meconial amniotic fluid. The incidence of MSAF in all of the deliveries during 4 year period were studied. Associated morbidity in newborns with MSAF admitted in NICU and perinatal differences between newborns with thin Vs. thick MSAF were compared. Incidence of MSAF was 18%. Thin meconium - 10.8%. Thick meconium - 2.8%. Neonatal morbidities associated with MSAF were perinatal asphyxia in 56.1%, pulmonary pathology in 34% (MAS in 32 cases and other respiratory abnormalities in 43) and gastrointestinal pathology in 30.5% (transitory feeding intolerance in all cases).

They concluded that although the relationship between MSAF and perinatal asphyxia was controversial, their association increases neonatal morbidity. Thick meconium was implicated as a risk factor influencing the well being during the intrapartrum and post partum periods[27].

Ziadesh SM, et al., in a prospective study at Jordan University of Science and Technology, Amman during April-November 1999 evaluated MSAF in term of fetal distress, MAS, perinatal morbidity and mortality. Women with a singleton cephalic pregnancy of completed 37-42 weeks and with no pre-defined risk factor were recruited into the study. 390 patients with MSAF and 400 patients as controls without meconium stained were studied. Perinatal mortality increased from 2 per 1000 births with clear AF to 10 per 1000 with meconium ($p<0.001$). Delivery by caesarean section also increased with meconium from

7-14% ($p<0.001$). They observed that women with thin meconium in the presence of normal fetal heart rate can be safely managed at the clinical level. Moderate thick meconium alone should alert the obstetrician to a high risk fetal conditions. Continuous FHR monitoring during labour and reassurance of fetal well being by acid-base assessment were most significant factors in the reduction of MAS[28].

STUDY JUSTIFICATION

A major goal of obstetric care is to prevent fetal morbidity and to bring down mortality as low as possible. This involves the early identification of the markers of fetal distress. The classical signs or markers of fetal hypoxia are variations in fetal heart rate pattern, presence of meconium in amniotic fluid, decrease in scalp pH, presence of intrapartum moulding, sudden increase or loss of fetal movements. Among the above mentioned traditionally, the fetal heart variation and meconium staining of amniotic fluid have been accepted as the most classical indicators of fetal distress. The appearance of meconium during labour has long been considered as a clinical sign of fetal distress. According to the classical conception, this is an indirect sign representing the response of the fetal gastro intestinal tract to hypoxic conditions.

Lately, the appearance of meconium as a sign of fetal distress has been questioned. Yet meconium aspiration is a severe life threatening illness in the neonate and need a good understanding of the circumstances under which this events occurs.

In light of the many controversial aspects, this study was undertaken to determine whether MSAF had influence on neonatal outcome.

AIM OF THE STUDY

a. To find out the immediate clinical outcome of new borns with meconium stained amniotic fluid.
b. Factors responsible for meconium stained amniotic fluid.

SUBJECTS AND METHODS

METHODOLOGY:

Study design: Case control study

Study place: Labour ward, Postnatal ward, New Born Unit, Maternity Hospital, Egmore, Chennai

Study period: March 2005 - October 2006

Study Population: Cases include newborns delivered with meconium staining of amniotic fluid.

Foe each case, two controls will be taken. These are new borns who did not have MSAF or otherwise having clear amniotic fluid.

Sample size: 217

Maneuver

- Newborns who had meconium staining of the amniotic fluid were taken in to the study.
- Gestational weeks of the baby noted in terms of term, preterm or post term pregnancy.
- In case of preterm baby, modified Ballord's score was determined.
- In case of term/SGA babies, ponderal index was calculated, depending upon which classified into symmetrical or asymmetrical IUGR.
- Mode of delivery of baby noted, whether is a labour naturalis, emergency or elective LSCS or via forceps delivery.
- If delivered through LSCS, indication for LSCS was noted.
- Antenatal USG if done noted, with particular mention to amniotic fluid index whether had oliogohydromnios or not.
- Placenta is examined for calcifications.
- Intrapartum fetal monitoring (CTG) if done was noted for any fetal heart rate variability.
- Grading of meconium done either as thin or thick meconium.
- Type of baby noted as either vigorous or non vigorous baby.
- Type of resuscitation done for these babies are noted in terms of routine initial steps of resuscitation or required tracheal toileting, bag mask ventilation or intubation.
- One minute and five minute Apgar determined.
- Those babies who were not admitted were followed up in the postnatal wards for 3 days. During each visit, respiratory rate, heart rate, feeding difficulties if any noted.
- Those who are admitted in new born unit were followed up till discharge.
- During hospital stay, Downe's score given till the respiratory distress settled, and hours of settlement of respiratory distress noted.
- Type of management of these babies entered whether required oxygen, oxygen with IVF, oxygen, IVF with antibiotics, and anticonvulsants or required mechanical ventilation.
- CXR taken in all babies who were admitted. Repeat CXR are also taken if indicated.
- Outcome like seizures, airleak, MAS etc., were looked for.
- For each MSAF baby, two newborn without MSAF were recruited and looked for maternal risk factors and fetal risk factors. These include maternal risks such as PIH, APH, anaemia, heart disease, gestational diabetes, maternal malnutrition, jaundice complicating pregnancies, fetal factors include IUGR, post dated babies etc.,

STATISTICAL ANALYSIS

To study the factors associated with MSAF, OR (95% CI) was arrived by univariate analysis. To adjust for the confounders multivariate analysis was done to arrive at adjusted OR (95% CI).

OBSERVATION

Total No of cases : 217

Table 1:

Incidence of Type of meconium

Type of meconium	*n*	*%*
Thin	141	64.97%
Thick	76	35.02%

Incidence of MSAF is 16.9%. Among 217 cases of MSAF, thin meconium constituted 141 cases (64.97%) and thick meconium 76 cases (35.02%). Incidence of thin meconium is more when compared to thick meconium.

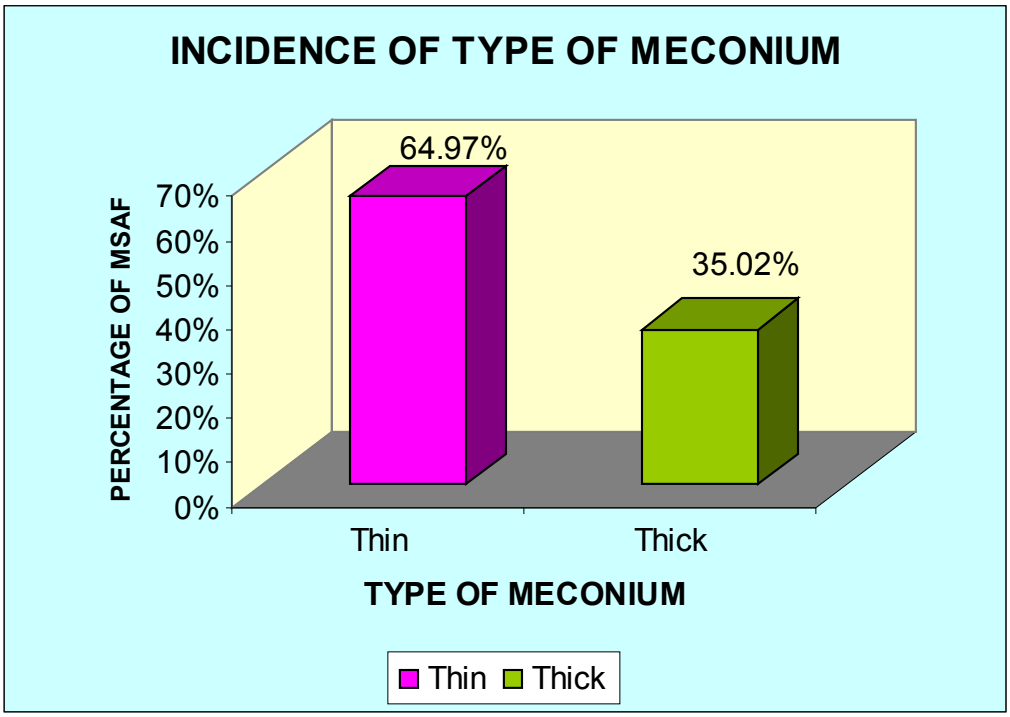


Table - 2

Type of Babies in MSAF

Type of Baby	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	
Vigorous	170	78.7	127	74.7	43	25.3	0.00
Non vigorous	47	21.65	14	29.78	33	70.21	

Of the 217 cases of meconium stained liquor, 170 (78.7%) cases are vigorous babies and 47 (21.6%) are nonvigorus. Out of 47 babies who were non vigorous, most of them (70.2%) had thick meconium

stained liquor when compared to 25.3% among vigorous babies. So thick meconium stained amniotic fluid was more common among non vigorous babies. This was found to be statistically significant (Table 2; p=0.00)

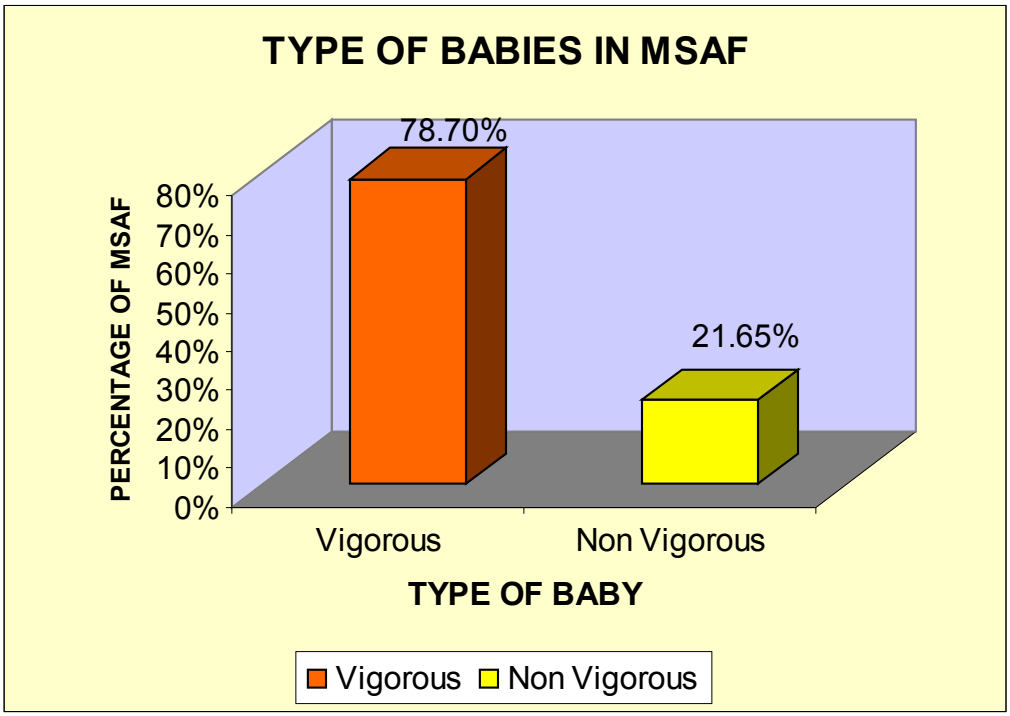


Table - 3
Incidence of MSAF with respect to gender

Sex of Baby	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	
Male	108	49.76	75	69.4	33	30.6	0.20
Female	109	50.23	66	60.55	43	39.44	

In the meconium stained liquor, 108 (49.76%) are male and 109(50.23%) are female, almost in the ratio of 1:1. Among 108 male babies, 75 babies (69.4%) are associated with thin meconium staining and
33 (30.6%) with thick meconium staining. Among 109 female babies, 66(60.55%) are with thin meconium when compared to 43 (39.44%) babies who are with thick meconium staining. P value is 0.20 which is statistically not significant (Table - 3; p = 0.20)

Table - 4
Age of the mother & meconium stained amniotic fluid

Age of mother	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	
<20 years	48	22.2	34	70.8	14	29.2	0.42
20-35 years	154	70.8	96	62.1	58	37.9	
>35 years	15	6.9	11	73.3	4	26.7	

Out of 217 cases of meconium staining, 48 cases (22.2%) occurred in age group of mothers less than 20 years. Out of which 34 (70.8%) babies presented with thin meconium staining and 14 babies (29.2%) with thick meconium staining. 154 cases occurred in age group of mothers between 20-35 years, out of which 96 (62.1%) are thin meconium and 58 cases (37.9%) are thick meconium stained. There are 15 cases of meconium, occurred in age group more than 35 years. Out of which 11 cases (73.3%) are thin meconium and 4 (26.7%) are thick meconium. In all the age groups thin meconium staining predominant reflecting the general trend. P value is 0.42, which is statistically not significant (Table 4; p = 0.42).

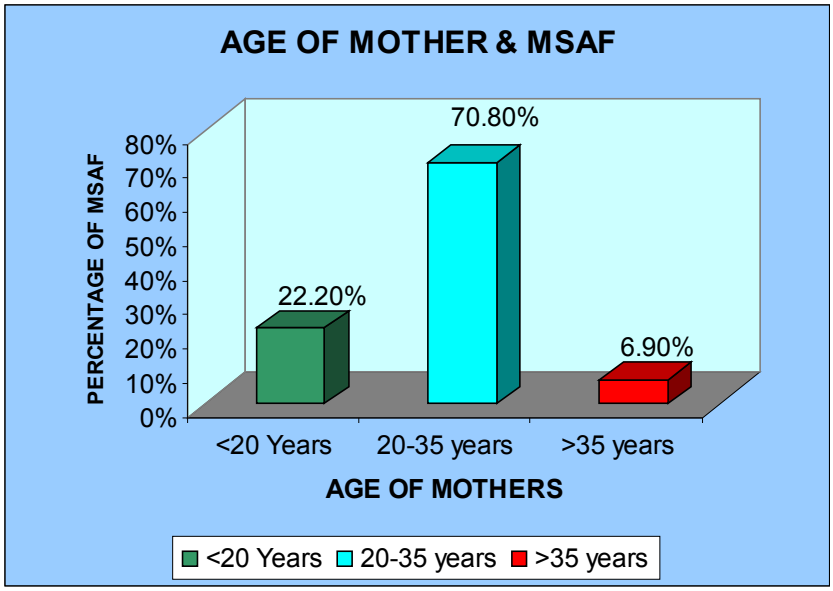


Table - 5
Antenatal risks in MSAF

Antenatal risks	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	

PIH	41	19.0	20	48.8	21	51.2	0.05
Anaemia	13	6.0	8	61.5	5	38.5	
GDM	3	1.4	3	100.0	0	0.0	
CVS	1	0.5	0	0.0	1	100.0	
No risks	159	73.1	110	69.0	49	31.0	

In MSAF, 41 cases (19.0%), had PIH as a complication in their mother. Of which 20 (48.8%) had thin meconium staining and 21 (51.2%) had thick meconium staining. 13 cases had anaemia as an antenatal risks, of which 8 (61.5%) had thin meconium and 5 (38.5%) had thick meconium. 3 cases had GDM as a complication and all the 3 (100%) had thin meconium staining.

Majority of MSAF, i.e., 159 cases (73.1%) had no antenatal risks, otherwise born to a normal mother, of which 110 cases (69.0%) are with thin meconium staining and 49 cases (31.0%) are with thick meconium staining.

Table - 6

Requirement of admission in MSAF babies

Admission	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	
Yes	106	48.84	54	38.29	52	68.42	0.00
No	111	51.15	88	62.41	23	30.26	

Of the 217 cases of MSAF, 106 cases (48.84%) required admission and 111 cases (51.15%) did not require admission. In thin MSAF, 88 (62.4%) did not require admission, whereas 54 (38.29%) needed admission. In thick MSAF 52 (68.42%) required admission and 23 (30.26%) did not require admission. Thus the admission rate is higher in thick meconium group. P value is 0.00, which is statistically significant (Table 6; $p = 0.00$)

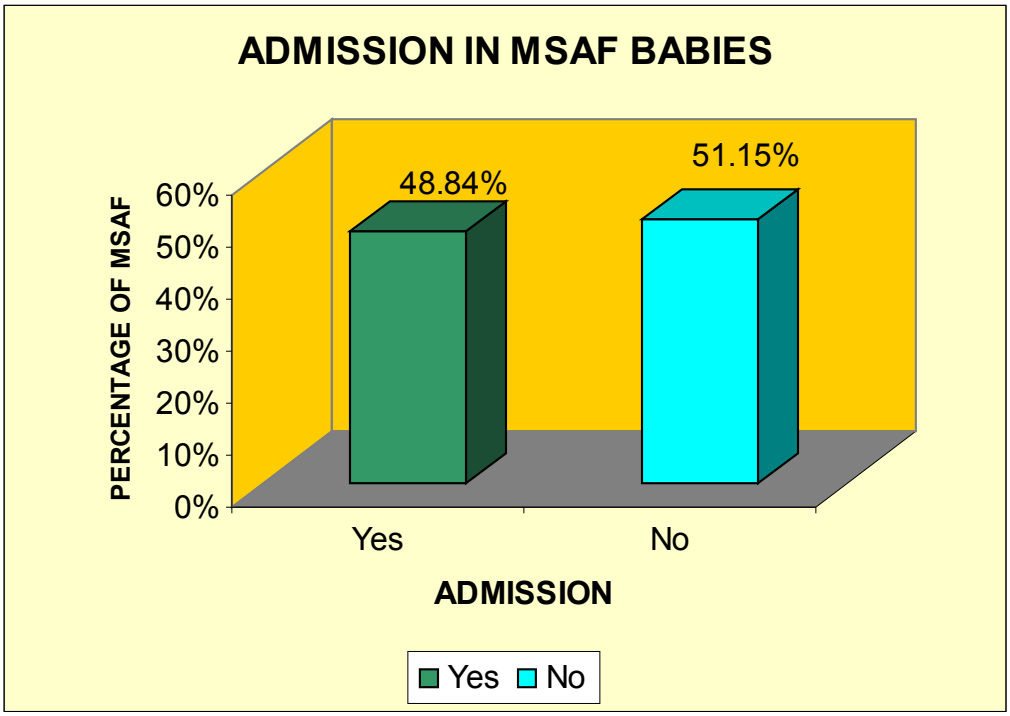


Table - 7
Type of resuscitation required in MSAF babies

Type of resuscitation	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	
Routine	172	79.2	128	74.3	44	25.7	0.00
Tracheal toileting	21	9.7	8	38.1	13	61.9	
Tracheal toileting with BMV	23	10.6	5	21.7	18	78.3	
BTV	1	0.5	0	0.0	1	100.0	

Of the 217 cases of MSAF, majority i.e., 172 cases (79.2%) required routine initial steps of resuscitation. 23 required tracheal toileting with BMV, 21 required tracheal toileting alone. One case (0.5%) required bag and tube ventilation. Routine, initial steps of resuscitation alone required in 128 cases (74.3%) of thin meconium staining and 44 cases (25.7%) of thick meconium staining. Tracheal toileting needed in 13 (61.9%) cases of thick meconium stained liquor. BTV needed in 1 case (100%) of thick meconium staining. Tracheal toileting with BMV required in 18 cases (78.3%) of thick meconium liquor when compared to 5 cases (21.7%) of thin MSAF. P value is 0.00, which is statistically significant (Table 7; p = 0.00)

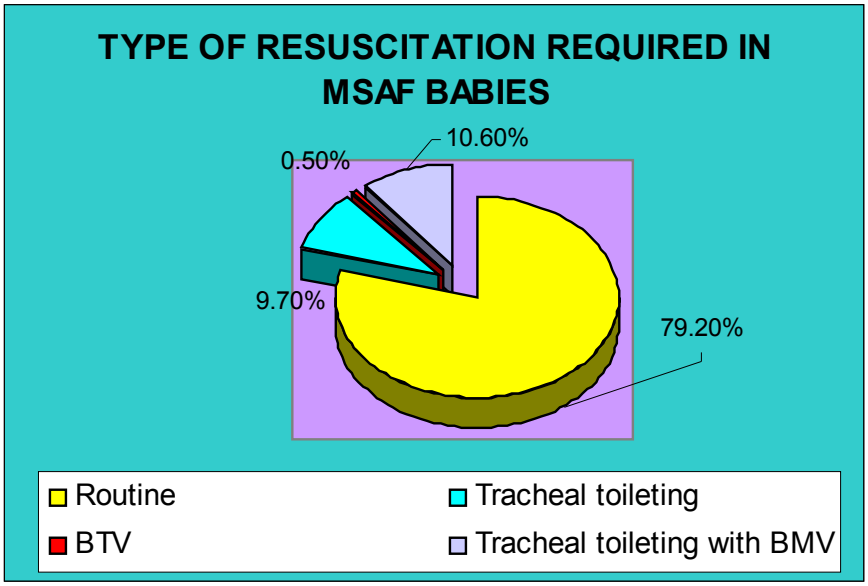


Table - 8
X-ray findings in MSAF babies

Chest X-ray	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	
Normal	81	76.5	47	58.02	34	41.97	0.01
MAS	20	18.9	4	20.0	16	80.0	
Pneumothorax	6	5.7	3	50.0	3	50.0	

81 cases (76.5%) had a normal x-ray. 20 cases (18.9%) had features of meconium aspiration. 6 cases (5.7%) had pneumothorax. Majority of normal x-rays i.e. 47 cases (58.02%) belong to thin MSAF. Meconium aspiration is found in 16 cases (80.0%) of thick meconium, when compared to 4 cases (20.0%) of thin meconium. Pneumothorax occurred equally in both the groups. P value is 0.01, which is statistically significant (Table - 8; p = 0.01).

Table - 9
Settlement of RD in MSAF babies

Hours of settlement of Resp. distress	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	
<6 hours	66	61.3	42	63.1	24	36.9	0.02
6 - 24 hours	20	18.9	8	40.0	12	60.0	
24 - 48 hours	6	5.7	1	16.7	5	83.3	
48-72 hours	1	0.9	0	0	1	100.0	
> 3 days	4	3.8	1	25.0	3	75.0	
Resp. failure	10	9.4	2	20.0	8	80.0	

In majority of the cases, 66 (61.3%) respiratory distress settled within 6 hours of admission. In 20 cases (18.9%) distress settled between 6 hours - 24 hours. 10 cases (9.4%) present with respiratory failure. Respiratory distress settled within 6 hours in 42 cases (63.1%) of thin meconium stained liquor when compared to 24 cases (36.9%) of thick meconium stained liquor. Those who presented with respiratory failure, majority i.e., 8 cases (80.0%) had thick MSAF and only 2 cases (20%) had thin MSAF. Respiratory distress took more time to come down among who had thick meconium stained liquor when compared to those who had thin meconium stained. This was found to be significant statistically (Table 9; $p = 0.02$)

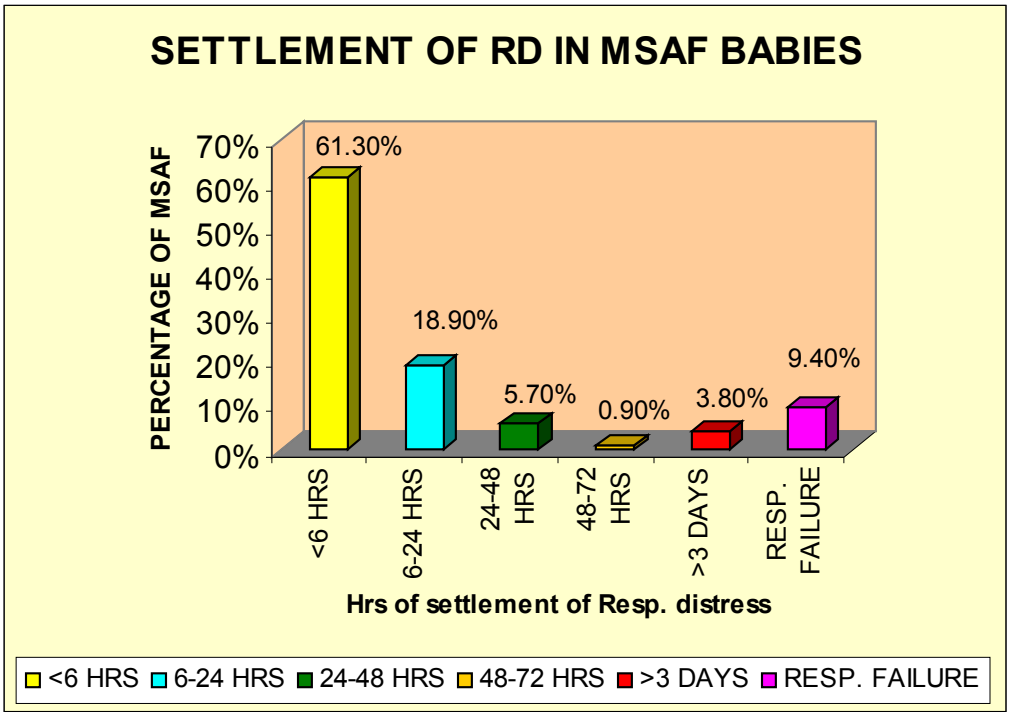

SETTLEMENT OF RD IN MSAF BABIES
PERCENTAGE OF MSAF
70%
60%
50%
40%
30%
20%
10%
0%
61.30%
18.90%
5.70%
0.90%
3.80%
9.40%
<6 HRS
6-24 HRS
24-48 HRS
48-72 HRS
>3 DAYS
RESP. FAILURE
Hrs of settlement of Resp. distress
<6 HRS
6-24 HRS
24-48 HRS
48-72 HRS
>3 DAYS
RESP. FAILURE

Table - 10
Type of management required in MSAF babies

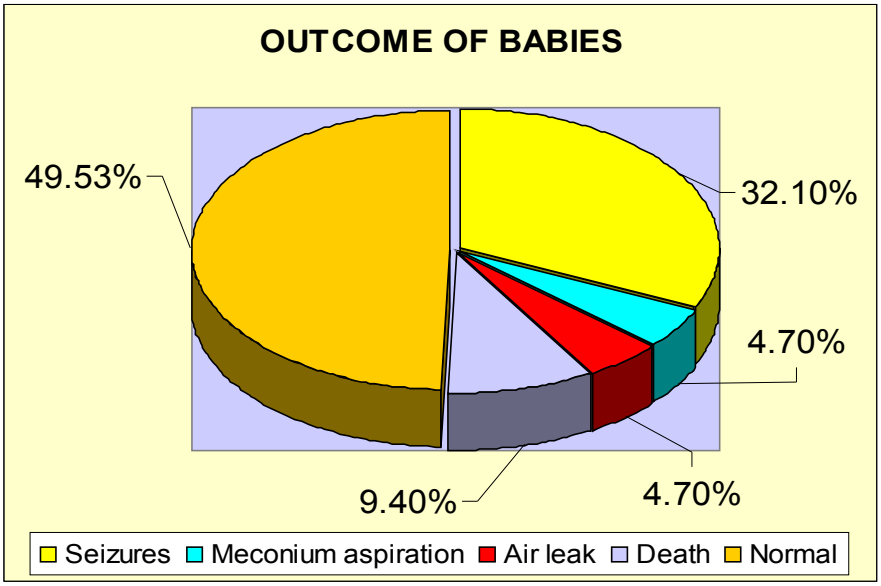
Type of management	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	
O_2 with IVF	42	39.25	30	71.42	12	29.3	0.003
O_2 with IVF, AB	22	20.8	7	31.8	15	68.2	
O_2 with IVF, AB, ACT	33	31.1	15	45.5	18	54.5	
Ventilator	10	9.4	2	20.0	8	80.0	

42 cases (39.25%) required O_2 with IVF. 33 cases (31.1%) required O_2, IVF, antibiotics with anti-convulsants, of which 18 cases (54.5%) are thick MSAF and 15 cases (45.5%) are thin MSAF. 10 cases (9.4%) required mechanical ventilation, of which 8 cases (80%) are thick MSAF and 2 cases (20%) are thin MSAF. Thus, mechanical ventilation required more in thick MSAF when compared to thin MSAF. O_2, IVF, antibiotics are required in 15 cases (68.2%) of thick MSAF and 7 cases (31.8%) of thin MSAF. P value is 0.003, which is statistically significant (Table 10; $p = 0.003$).

Table - 11
Outcome of babies delivered through MSAF

Outcome	*N*	*%*	*Type of meconium*				*P value*
			Thin		*Thick*		
			n	%	n	%	
Seizures	34	32.1	16	47.1	18	52.9	0.08
Meconium aspiration	5	4.7	1	20.0	4	80.0	
Air leak	5	4.7	2	40.0	3	60.0	
Death	10	9.4	2	20.0	8	80.0	
Normal	53	49.53	33	62.26	20	37.73	

Out come of MSAF was normal in 53 cases (49.1%), seizures seen in 34 cases (32.1%), meconium aspiration in 5 cases (4.7%), Airleak in 5 cases (4.7%) and death in 10 cases (9.4%). Among seizure group there were higher proportion of babies born with thick meconium stained liquor. Similarly in other outcomes like meconium aspiration, airleak and death, there were higher proportions of babies with thick meconium staining when compared to thin meconium staining. So, thick MSAF indicates a high risk fetal condition. P value is 0.08 which is statistically insignificant (Table 11; p = 0.08)



COMPARISON OF MATERNAL & FETAL FACTORS AMONG NEWBORNS WITH AND WITHOUT MSAF-UNIVARIATE ANALYSIS

Table - 12
Parity and its association with MSAF

Parity	*N*	*%*	*MSAF*		*Non MSAF*		*OR*	*95% CI*	*P Value*
			n	%	n	%			

Primi	367	55.7	146	67.28	221	50	3.1	2.1, 4.7	0.00
Multi gravidae	74	11.2	33	15.20	41	9.2	3.8	2.1, 6.8	
II gravidae	218	33.1	38	17.51	180	40.72	1.0	Ref.	

In the meconium stained group 146 cases (67.28%) are primigravidae, 33 cases (15.20%) are multigravidae. In non-MSAF group 221 cases (50%) are primi gravidae and 180 cases (40.72%) are II gravidae. Odds of newborns with MSAF being born to multigravidae is 3.8 when compared to those who did not have MSAF. [OR (95% CI) = 3.8 (2.1,6.8)]. Odds of newborns with MSAF being born to primi gravidae is 3.1 when compared to those who did not have MSAF [OR (95% CI) = 3.1 (2.1,4.7)].

Table - 13
Antenatal risk in 2 groups

Antenatal risk	*N*	*%*	*MSAF*		*Non MSAF*		*OR*	*95% CI*	*P Value*
			n	%	n	%			
PIH	60	9.1	42	70.0	18	20.0	6.1	3.4,11	0.00
Anaemia	18	2.7	13	72.2	5	27.8	6.9	2.4, 19.5	
Nil	574	87.2	158	27.5	416	72.5	1.0	Ref.	

There are 60 cases of PIH, of which 42 (70.0%) are associated with meconium stained liquor and 18 (30%) are associated with clear liquor. There are 18 cases of anaemia, 13 of which (72.2%) belong to MSAF group and 5 (27.8%) to non-MSAF group. PIH as antenatal risk occurred in 42 cases (70.0%) of MSAF and 18 cases (20.0%) of non MSAF group. Anaemia occurred in 13 cases (72.2%) of MSAF group when compared to 5 cases (27.8%) of non MSAF group.
Odds of newborns with MSAF having anaemia as a complication antenatally in mothers is 6.9 when compared to non-MSAF [OR (95% CI) = 6.9 (2.4,19.5)]. Odds of newborns with MSAF having PIH as a complication antenatally in mother is 6.1 when compared to non-MSAF [OR (95% CI) = 6.1 (3.4,11)].

Table - 14
Mode of deliveries in 2 groups

Mode of delivery	*N*	*%*	*MSAF*		*Non MSAF*		*OR*	*95% CI*	*P Value*
			n	%	n	%			
Emergency LSCS	388	58.9	166	42.8	222	57.2	3.2	2.2, 4.6	0.00
Labour naturalis	271	41.1	51	18.8	220	81.2	1.0	Ref	

Emergency LSCS as a mode of delivery is seen in 166 (42.8%) cases of meconium stained liquor and in 222 (57.2%) cases of non- meconium stained liquor. Majority of MSAF 166 cases (76.49%) were delivered through emergency LSCS and only 51 (23.50%) were born through labour naturalis. Whereas in non MSAF group the mode of delivery being emergency LSCS or labour naturalis are equal.
Odds of newborns with MSAF being delivered through emergency LSCS is 3.2 when compared to those who did not have MSAF [OR (95% CI) = 3.2 (2,2, 4.6)]

Table - 15
Indication of LSCS in 2 groups

Indication of LSCS	*N*	*%*	*MSAF*		*Non MSAF*		*OR*	*95% CI*	*P Value*
			n	%	n	%			
Previous LSCS with FD	33	8.4	24	15.48	9	4.22	9.9	4.2, 23.7	0.00
Fetal distress	117	29.8	75	48.38	42	19.71	6.7	3.8, 11.6	
CPD	81	20.7	27	17.41	54	25.35	1.9	1.0, 3.5	
Previous LSCS	137	34.9	29	18.70	108	50.70	1.0	Ref	

Majority of indication for LSCS in MSAF is FD reported in 75 cases (48.38%). In 27 cases (17.41%), the indication for LSCS is CPD and in 24 cases (15.48%) previous LSCS with fetal distress.
Odds of newborns in MSAF delivered two emergencies LSCS indication being previous LSCS with fetal distress is 9.9 when compared to newborns without MSAF [OR (95% CI) = 9.9 (4.2, 23.7)].
Odds of newborns in MSAF delivered two emergencies LSCS indication being fetal distress is 6.7 when compared to newborns without MSAF [OR (95% CI) = 6.7 (3.8, 11.6)].

Table - 16
Placental Calcification in 2 groups

Placental calcification	*N*	*%*	*MSAF*		*Non MSAF*		*OR*	*95% CI*	*P Value*
			n	%	n	%			
Yes	26	4.0	24	92.3	2	7.7	27.4	6.5,11.6	0.00
No	630	96.0	190	30.2	440	69.8	1.0	Ref.	

Placental calcification seen in 26 cases (4.0%) of which 24 cases (92.3%) are with MSAF and only 2 (7.7%) are without MSAF. Placental calcification which denotes placental insufficiency are more in MSAF group.
Odds of newborn with MSAF having placental calcification is 27.4 when compared to babies born to non MSAF [OR (95% CI) = 27.4 (6.5, 11.6)].

Table - 17
Association of IUGR in MSAF

Growth Retardation	*N*	*%*	*MSAF*		*Non MSAF*		*OR*	*95% CI*	*P Value*
			N	%	n	%			
SGA (Asymmetrical IUGR)	33	5.0	27	81.8	6	18.2	10.5	4.3,25.8	0.00
AGA	623	94.5	187	30.0	436	70.0	1.0	Ref.	

Out of the 33 SGA babies (asymmetrical IUGR) 27 cases (81.8%) occurred in MSAF group and 6 cases (18.2%) in non-MSAF group. Hence incidence of IUGR is more in MSAF.
Odds of newborn with MSAF, being small for gestational age (asymmetric IUGR) is 10.5 when compared to those who did not have meconium stained amniotic fluid. [OR (95% CI) = 10.5 (4.3, 25.8)].

Among the factors which are analysed,

Primigravidae [OR (95% CI) = 2.1 (1.4, 3.4)]

Multigravidae [OR (95% CI) = 2.1 (1.1, 4.1)]

PIH [OR (95% CI) = 4.4 (2.2, 8.5)]

Anaemia [OR (95% CI) = 5.1 (1.6, 16.6)] and
CTG with fetal heart variability [OR (95% CI) = 4.5 (2.8, 7.3)]
are the factors which are found to be independently associated with meconium staining of aminiotic fluid.

DISCUSSION

- In our study, with respect to parity of mother the meconium stained amniotic fluid had 146 (67.28%) cases of primigravidae when compared to multigravidae (15.20%) and II gravidae. Thus primi parity is associated to MSAF, which is similar to the study conducted by David et al[26]. Incidence of primi is higher in our study when compared to the study of Narang et, where the incidence was 57.14% in primi and 42.86% in multigravidae[5].
- Majority of MSAF are delivered through emergency LSCS in our study with 76.49% incidence. This indicates that majority of MSAF are being delivered through caesarean section which is similar to the study of Wong et al[20].
- In our present study the major indication for LSCS in MSAF group is fetal distress, which being reported in 75 cases (48.38%). This is higher when compared to the study of Alchalabi et al, where the indication of LSCS for fetal distress was only 10.5%[22].
- There is no antenatal risk in 158 cases (74.17%) in meconium stained group. 42 cases (19.71%) had PIH as an antenatal risks and anaemia in 13 cases (6.10%). This is similar to the study of Kanula et al., where PIH was the main antepartum complication leading to meconium stained liquor. Other factors like hepatitis in mother, asthma, APH which were significant in their study was not so in our study. This is contrast to the study of Zhu et al., whom concluded that there is no correlation between MSAF and maternal medical complication[21].
- Significant fetal heart rate variability in meconium stained group is observed which is similar to the study of Sheiner et al., where they found a significant linear association between meconium stained amniotic fluid and abnormal fetal heart rate[24].
- Majority showed placental calcification in meconium stained group. This is found to be significant in our study and no previous study mentioned this as a factor in MSAF.
- Though it was mentioned in the study of Berkus et al., that males had a preponderance of getting meconium stained liquor[23], in our present study both male and female had equal change of getting MSAF., But when the consistency is considered, 75 cases (69.4%) of thin meconium are male and 43 cases (56.5%) of thick meconium are female.
- In our present study, majority of meconium stained liquor are term babies with 215 cases (99.07%) and 2 (0.01%) are post-term and no preterm babies. This is in contrast with Zhu et al study, where they concluded that the relative factors on MSAF were gestational weeks >42 weeks and large for gestational age babies[21].
- In our study, out of 33 cases of SGA, 27 (81.8%) had MSAF. All of them are asymmetrical IUGR babies. Thus the IUGR babies had a higher incidence of MSAF when compared to AGA babies. This is comparable to previous study of Gupta et al, where IUGR was also associated with MSAF[3].
- In our study, thin meconium seen in 141 cases (64.8%) and thick meconium in 76 cases (35.2%). This is comparable to previous studies done by Sheiner E et al[24], in which thin meconium out numbered the thick meconium.
- The morbidity associated with thick meconium is higher in our study. Out of 20 cases of meconium aspiration (detected by x-ray). 16 cases (80%) are with thick meconium and 4 cases (20%) in thin meconium.
- Among 10 cases who presented with respiratory failure, 8 cases (80%) had thick meconium staining and only 2 (20%) with thin meconium. 18 cases (54.5%) of thick meconium required anticovulsants along with O_2, IVF and antibiotics. The need for mechanical ventilation also high in thick meconium group. Out of 10 cases which needed mechanical ventilation 8 (80%) are associated with thick meconium and only 2 (20%) with thin meconium. Seizures, MAS, airleak are more observed in thick meconium group. Out of 10 death, 8 (80%) cases are with thick meconium. This morbidities with thick meconium are similar to the study of Sheiner et al., Gonzalez et al., where they concluded that consistency of meconium had a direct bearing on neonatal outcome and thick meconium alone should alert the physician to a high risk fetal condition[27].
- Outcome of MSAF in our study is seizure in 32.1% MAS in 4.7%, Airleak in 4.7%, and death in 10% of cases. This is similar to the study of Manju Latha Sharma et al with seizures in 25.64%, airleak in 12.8% and death in 10.25% cases[29].

CONCLUSION

Meconium staining of amniotic fluid by itself has no significance. In our study, not all the babies who were born through meconium stained amniotic fluid required admission. Only a part of them required admission. Even in the admitted babies, respiratory distress settled within few hours of supportive measures of treatment. But, meconium stained amniotic fluid becomes a significant factor when associated with factors like maternal PIH, anaemia, oligohydromnios, IUGR babies, CTG showing FHR variability and placental calcifications. In these cases MSAF is associated with morbidity and mortality; majority of which are thick meconium suggesting that consistency of meconium had direct bearing in neonatal outcome. Although mode of delivery in MSAF does not prevent meconium aspiration, majority of them are delivered through caesarean section which is questionable.

PROFORMA

Name
Sex
Time/Date
Mother IP No
Address
Age
Height
Weight
Hb%
Parity
Mode of Delivery
Indication for LSCS
USG Mother
Fetal Heart Rate Monitoring (CTG)
Antenatal Factors

Intrapartum Factors

Details of Baby

Term Preterm wks Post term wks

Birth Weight

IUGR Ponderal Index
Symmetrical
Asymmetrical

Type of Baby

Vigorous Non Vigorous

Type of resuscitation

Apgar at 1 mint

Apgar at 5 mints

Admission Yes No

Follow up in Postnatal wards

	Cry	Colour	Activity	HR	RR	Feeding Difficulties	Others
Day 1							
Day 2							
Day 3							

Discharged on - Day of Life

Investigations (for admitted babies)

Chest X-ray RPT-CXRS

CBG

USG - Cranium

Diagnosis

Follow up in NICU:

Days	*IVF*	*O_2*	*Antibiotics*	*Seizures*	*Anticonv*	*RDS (Downe's score)*

Outcome

BIBLIOGRAPHY

1. Cleary GM, Wiswell TE. Meconium-stained amniotic fluid and the meconium aspiration syndrome: An update. Pediatr Clin North Am 1998; 45: 511-529.
2. Wiswell TE, Bent RC. Meconium staining and the meconium aspiration syndrome: Unresolved issues. Pediatr Clin North Am 1993; 40: 955-981.
3. Gupta V, Bhatia BD,. Mishra OP. Meconium stained amniotic fluid: Antenatal, intrapartum and neonatal attributes. Indian Pediatr 1996; 33: 293-297.
4. Suresh GK, Sarkar S. Delivery room management of infants born through thin meconium stained liquor. Indian Pediatr 1995; 32: 1177-1181.
5. Narang A, Nair PM, Bhakoo ON, Vashishi: K. Management of meconium stained amniotic fluid: A team approach. India Pediatr 1993; 30: 9-13.
6. Carson BS, Losey RW, Bowes WA, Simmons MA. Combined obstetric and pediatric approach to prevent meconium aspiration syndrome. Am J Obstet Gynecol., l 976; 126: 712-71 5.
7. Dye T, Aubry R, Gross S. Artal R. Amnioinfusion and the intrauterine prevention of meconium aspiration. Am J Obstet Gynecol, l994; 171: 1601-1605.
8. Winsewell TE. Handling the meconium - stained infant. Semin Neonatal 2001; 6: 225-31.
9. Gregory GA, Gooding CA, Phibbs RH, Tooley WHO Meconium aspiration in infants. A prospective study. J Pediatr 1974; 85: 848-853.
10. Ting P, Brady JP. Tracheal suction in meconium aspiration. et Gynecol l975; 122: 767-771.
11. Linder N, Aranda JV, Tsur M, Matoth I, Yotsiv I, Mandelbe Need for endotracheal intubation and suction in meconium-stained, neonates. J Pediatr 1988;112:613-615.
12. Am J Obstet Gynecol Jan 1992 Vol. 166, Part 1: 171-183.
13. Miller FC. Sacks Da, Yen Sy et al. Significance of Meconium during labour: AMJ. O and G 1975: 122: 573-80.
14. Krishan L, Nastruddin B, Prabhakar P, Bhaskaranand N. Routine antibiotic cover for newborns intubated for aspirating meconium. Is it necessary? Indian Pediatr 1995; 32: 529-531.
15. Findlay RD, Taeusch HW, Walther FJ. Surfactant replacement therapy for meconium aspiration syndrome. Pediatrics 1996; 97: 48-52.
16. The Neonatal Inhaled Nitric Oxide Study Group. Inhaled nitric oxide in full-term and nearly full-term infants with hypoxic respiratory failure. New Engl J Med 1997;336: 597-604.
17. Katturinkel J, ed. Text book of neonatal resuscitation. Elk Gr Vill e, IL: American Academy of Pediatrics; 2000.
18. Reed/Claireaux and cockbur; Diseases of the fetus and newb e 2) : 1352-53.
19. Foust R Tran NN, Cox C, Metter TFJ, Greenspon JS, Wolfson MR. An alternative ventilatory strategy for acute meconium aspiration injury. Pediatric Pulmonol 1996; 21: 316-321.
20. Wong SF, Chow KM, The relative risk of 'fetal distress' in pregnancy associated with meconium-stained liquor at different gestation. Obstet Gynaecol. 2002 Nov; 22 (6): 594-9.

21. Zhu L, Wong F, Bai J. The epidemiology of meconium stained amniotic fluid on hospital basis. Zhongguo Yi Xue Ke Xue Yuan Xue Bao. 2003 Feb; 25 (1): 63-5.
22. Alchalabi H, Abu-Heija AT, El-Sunna E, Zayed F, Badria LF, Obeidat A. Meconium-stained amniotic fluid term pregnancies - a clinical view. J Obstet Gynaecol. 1999 May; 19 (3): 262-4.
23. Berkus MD, Langer O, Samueloff A, Xenalkis EM, Field NT, Ridgway LE. Meconium-stained amniotic fluid: increased risk for adverse neonatal outcome. Obstet Gynecol. 1994 Jul; 84 (1): 115-20.
24. Sheiner E, Hadar A, Shoham-Vardi I, Hallak M, Katz M, Mazor M. The effect of meconium on perinatal outcome: a prospective analysis. J Matern Fetal Neonatal Med. 2002 Jan; 11 (1): 54-9.
25. Greenwood C, Lalchandani S, Macquillan K, Sheil O, Murphy J, Impey L. Meconium passed in labor: how reassuring is clear amniotic fluid? Obstet Gynecol. 2003 Jul; 102 (1): 89-93.
26. David AN, Njokanma OF, Iroha E. Incidence of and factors a with meconium staining of the aminotic fluid in a Nigerian Unive ing Hospital.
27. Gonzalez de Dios J, Moya Benavent M,Barbal Rodoreda A, Dura Trave T, Juste Ruiz M, Castano Iglesias C, Gonzalez Montero R. Neonatal morbidity associated with meconial amniotic fluid. An Eps Pediatr. 1998 Jan; 48(1): 54-9.
28. Ziadeh SM, Sunna E. Obstetric and perinatal outcome of pregnancies with term labour and meconiumstained amniotic fluid. Arch Gynecol Obstet. 2000 Sep; 264(2): 84-7.
29. Manju Lata Sharma. A study of newborn infant with meconium stained amniotic fluid congres' de Valencia. 2000 Sep; 1-2.