



Comprehensive Review of Meconium Aspiration Syndrome: Risk Factors, Complications, and Treatment Approaches

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ABSTRAK

Latar Belakang: Meconium Aspiration Syndrome (MAS) merupakan salah satu penyebab utama gangguan pernapasan pada neonatus yang terjadi akibat aspirasi cairan ketuban yang terkontaminasi mekonium. MAS dapat menyebabkan obstruksi jalan napas, pneumonitis kimia, disfungsi surfaktan, serta hipertensi pulmonal persisten. Tujuan: Artikel ini bertujuan untuk mengulas faktor risiko, patofisiologi, diagnosis, komplikasi, serta strategi pencegahan dan penanganan MAS berdasarkan studi literatur terkini. Metode: Kajian literatur dilakukan dengan meninjau berbagai penelitian yang relevan dari jurnal ilmiah, buku kedokteran, dan sumber terpercaya lainnya. Hasil: MAS dikaitkan dengan faktor risiko seperti insufisiensi plasenta, hipertensi maternal, preeklampsia, dan hipoksia janin. Diagnosis ditegakkan melalui pemeriksaan klinis, radiologi, serta analisis gas darah. Penanganan MAS mencakup resusitasi neonatus, terapi oksigen, ventilasi mekanik, serta penggunaan ECMO pada kasus berat. Kesimpulan: Deteksi dini dan penanganan yang tepat sangat penting dalam mengurangi morbiditas dan mortalitas akibat MAS. Strategi pencegahan di ruang bersalin berperan krusial dalam mengurangi risiko aspirasi mekonium dan meningkatkan luaran neonatal.

ABSTRACT

Background: Meconium Aspiration Syndrome (MAS) is a major cause of neonatal respiratory distress, occurring due to the aspiration of meconium-stained amniotic fluid. MAS can lead to airway obstruction, chemical pneumonitis, surfactant dysfunction, and persistent pulmonary hypertension. **Objective:** This article aims to review the risk factors, pathophysiology, diagnosis, complications, as well as prevention and management strategies of MAS based on recent literature. **Methods:** A literature review was conducted by analyzing relevant studies from scientific journals, medical textbooks, and other reliable sources. **Results:** MAS is associated with risk factors such as placental insufficiency, maternal hypertension, preeclampsia, and fetal hypoxia. Diagnosis is established through clinical assessment, radiological findings, and blood gas analysis. Management includes neonatal resuscitation, oxygen therapy, mechanical ventilation, and ECMO in severe cases. **Conclusion:** Early detection and appropriate management are crucial in reducing morbidity and mortality caused by MAS. Preventive strategies in the delivery room play a key role in reducing the risk of meconium aspiration and improving neonatal outcomes..

1. INTRODUCTION

Pregnancy is marked by the formation of the amniotic sac, which is filled with clear amniotic fluid. The amniotic sac begins to form around 12 days after fertilization, and throughout pregnancy, the fetus floats in the amniotic fluid, which plays a crucial role in protecting and supporting fetal development (Moore et al., 2020). The amount of amniotic fluid increases as the fetus grows, reaching its peak at 34 weeks of gestation, with an average volume of 800 ml. As pregnancy approaches 40 weeks, the volume decreases to approximately 600 ml before delivery (Cunningham et al., 2018).

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Amniotic fluid undergoes continuous circulation, where the fetus swallows and inhales the fluid, then excretes it through urine. This fluid exchange process is crucial for fetal lung maturation and maintaining a stable intrauterine environment (Mayo Clinic, 2022).

Composition and Functions of Amniotic Fluid

Amniotic fluid is a clear, yellowish liquid that plays a crucial role in protecting and supporting fetal development. It contains essential nutrients, hormones, antibodies, proteins, carbohydrates, fats, phospholipids, urea, and electrolytes, all of which contribute to fetal growth, particularly in the later stages of pregnancy (Underwood et al., 2005). One of the primary functions of amniotic fluid is to support fetal musculoskeletal development. The fluid allows the fetus to move freely, which is essential for the proper development of muscles and the skeletal system (Moore et al., 2020). In addition, amniotic fluid regulates fetal body temperature, providing a stable environment that prevents heat loss and temperature fluctuations, both of which are crucial for normal fetal growth (Cunningham et al., 2018).

Amniotic fluid also serves as a protective cushion for the fetus, shielding it from external pressure and trauma that could occur during pregnancy (Mayo Clinic, 2022). Furthermore, it facilitates lung development, particularly in the second trimester, when the fetus begins to inhale amniotic fluid into the lungs and swallow it. This process plays a vital role in the development of both the respiratory and digestive systems. Additionally, fetal movement within the amniotic fluid helps in the development of chest muscles and the rib cage, which are essential for respiration after birth (Underwood et al., 2005).

Amniotic fluid also plays a key role in providing essential nutrients to the developing fetus. It contains proteins, carbohydrates, lipids, and electrolytes, all of which are crucial for optimal fetal growth and organ development (Cunningham et al., 2018). Moreover, the presence of maternal antibodies in the amniotic fluid provides immune protection, helping to protect the fetus from infections while still in the womb (Moore et al., 2020).

Tabel 1. Normal Composition of Amniotic Fluid

Kandungan	Kadar
Calcium	4 mEq/L
Chlorida	102 mEq/L
CO ₂	16 mEq/L
Creatinin	1,8 mg/dL
Glucosa	29,8 mg/dL
pH	7,04
Potassium	4,9 mEq/L
Sodium	133 mEq/L
Total protein	2,5 gram/dL
Albumin	1,4 gram/dL
Urea	31 mg/dL
Urid Acid	4,9 mg/DI

Sumber: William W.

In addition to its developmental and protective functions, amniotic fluid can serve as an indicator of fetal health status. The color of the amniotic fluid can reveal potential complications. For example, greenish or brownish amniotic fluid may indicate that the fetus has passed meconium (the first stool) inside the womb, which can be a sign of fetal distress or hypoxia (Mayo Clinic, 2022). Hypoxia can lead to increased intestinal peristalsis and relaxation of the anal sphincter, causing meconium to be released into the amniotic fluid. If the fetus inhales meconium-stained amniotic fluid, this can lead to a serious respiratory condition known as Meconium Aspiration Syndrome (MAS), which requires immediate medical intervention (Underwood et al., 2005). Thus, amniotic fluid plays a multifaceted role in fetal development, protection, and health monitoring. Understanding its composition and functions is essential for monitoring pregnancy and identifying potential complications early.

Amniotic Fluid Circulation and Changes

Amniotic fluid undergoes continuous circulation throughout pregnancy, playing a vital role in fetal development. One of the primary mechanisms of circulation involves the fetus swallowing and inhaling amniotic fluid, which contributes to the maturation of the lungs and digestive system (Cunningham et al., 2018). As the fetus develops, excretion of fetal urine becomes a major source of amniotic fluid. After 20 weeks of gestation, fetal urine becomes the primary component of amniotic fluid, ensuring a steady balance of fluid within the amniotic sac (Underwood et al., 2005). Additionally, in the early stages of pregnancy, amniotic fluid is largely derived from maternal blood plasma, which seeps into the amniotic sac, helping to maintain an optimal environment for fetal growth (Moore et al., 2020).

Most of the amniotic fluid remains within the womb until birth, ensuring fetal protection and development throughout pregnancy. However, in certain conditions, the amniotic sac may rupture prematurely or during labor. If the amniotic sac ruptures before labor begins, it is referred to as Premature Rupture of Membranes (PROM), which can increase the risk of preterm birth and infection. Alternatively, Spontaneous Rupture of Membranes (SROM) occurs naturally during labor, marking the beginning of the birthing process. The rupture of the amniotic sac serves as a crucial indicator that labor is imminent, and most of the amniotic fluid remains within the womb until the baby is delivered (Mayo Clinic, 2022).

2. METHOD

This study employs a comprehensive literature review methodology, analyzing existing research on amniotic fluid composition, circulation, and its role in fetal development. Data were collected from peer-reviewed journals, medical textbooks, and authoritative sources, including PubMed, ScienceDirect, and Google Scholar. The selection criteria for articles included studies published within the last 20 years, with a focus on fetal physiology, obstetrics, and perinatology.

3. RESULT AND DISCUSSION

Meconium

The term meconium originates from the Ancient Greek word meconium-arion, meaning "like opium." Aristotle coined this term because it was believed that meconium induced fetal sleep. Fetal intestinal contents, or meconium, consist of a sterile mixture of various substances, including mucus glycoproteins, swallowed vernix caseosa, digestive tract secretions, liver and pancreatic enzymes, bile, plasma proteins, minerals, and lipids (Cunningham et al., 2018). Mucopolysaccharides account for 80% of the dry weight of meconium, and the levels of pancreatic and liver enzymes vary depending on gestational age (Moore et al., 2020).

The passage of meconium has long been considered an indicator of antepartum or intrapartum asphyxia. It is hypothesized that intrauterine hypoxia leads to increased intestinal peristalsis and relaxation of the anal sphincter, resulting in meconium passage into the amniotic fluid. Additionally, fetal head compression on the umbilical cord can stimulate a vagal response, causing meconium to be expelled (Mayo Clinic, 2022).

Studies have reported varying rates of neonatal asphyxia associated with meconium-stained amniotic fluid, with incidence rates of 47% and 20% in hospital-based studies (Underwood et al., 2005). Meconium passage is typically considered a sign of fetal maturation and is rare before 37 weeks of gestation. However, at 42 weeks, its occurrence increases to 35% or more. Two studies reported meconium passage rates of 30% and 2% in post-term infants, while rates in preterm infants were 0% and 1%, respectively (Cunningham et al., 2018).

Meconium Aspiration Syndrome (MAS)

Meconium Aspiration Syndrome (MAS) is defined as a clinical and radiological syndrome resulting from the inhalation or aspiration of meconium by the fetus or neonate. MAS can occur before, during, or after labor and delivery. Inhaled meconium may partially or completely obstruct the neonatal airway, causing severe respiratory distress (Moore et al., 2020). Although some air may pass through meconium in the airways during inhalation, meconium can become trapped in

the airways during exhalation, leading to airway irritation, inflammation, and difficulty breathing (Cunningham et al., 2018). The severity of MAS depends on the amount of aspirated meconium and other underlying conditions, such as intrauterine infections or post-term pregnancy (beyond 42 weeks) (Mayo Clinic, 2022).

Meconium is more toxic than previously believed and can cause a vicious cycle involving hypoxemia, pulmonary shunting, acidosis, and pulmonary hypertension. These complications are often challenging or impossible to manage effectively. Therefore, the primary goal of delivery room intervention is to reduce the incidence and severity of MAS (Underwood et al., 2005). Based on non-randomized studies, intubation is recommended for neonates born with thick meconium-stained amniotic fluid to allow for airway suctioning (Cunningham et al., 2018). However, there is ongoing debate regarding selective versus universal tracheal suctioning for neonates born through meconium-stained amniotic fluid (MSAF). Current research explores the necessity of intubation and suctioning for all neonates with MSAF (Moore et al., 2020).

MAS remains one of the most common neonatal respiratory disorders encountered by pediatricians and obstetricians. In the United States, 520,000 deliveries (12% of live births) are complicated by MSAF, and 35% of these cases progress to MAS, representing 4% of all live births (Mayo Clinic, 2022). Among neonates diagnosed with MAS: 30% require mechanical ventilation; 10% develop pneumothorax; 4% die from MAS complications. Additionally, 66% of all cases of persistent pulmonary hypertension (PPHN) are associated with MAS (Underwood et al., 2005).

The passage of meconium into the amniotic fluid is usually a consequence of intrauterine hypoxia and/or fetal distress. If meconium is expelled within four hours before delivery, the newborn's skin may be stained with meconium. Neonates delivered in breech presentation (buttocks-first) frequently pass meconium before birth without experiencing fetal distress (Cunningham et al., 2018).

Incidence Of Meconium Aspiration Syndrome (MAS)

Several studies report varying incidence rates of meconium-stained amniotic fluid, with estimates ranging from 6% to 25% of births. However, not all neonates exposed to meconium develop MAS. Only 2-36% of fetuses aspirate amniotic fluid while in utero or during their first breath, and only 11% of these cases progress to MAS (Moore et al., 2020). In the United States and other developed countries, 8-25% of all deliveries at 34 weeks or later are associated with meconium-stained amniotic fluid (Mayo Clinic, 2022). Among these cases, approximately 10% of newborns develop MAS.

In developing countries, where home births and limited access to primary healthcare services are more common, MAS incidence rates are thought to be higher, correlating with increased neonatal mortality rates (Underwood et al., 2005). are the main part of scientific articles, containing: final results without data analysis process, hypothesis testing results. Results can be presented with tables or graphs, to clarify the results verbally.

Morbidity And Mortality Rate

The mortality rate of Meconium Aspiration Syndrome (MAS) is relatively high, reaching approximately 20%, primarily due to severe parenchymal lung disease and pulmonary hypertension (Cunningham et al., 2018). Additionally, other complications such as air block syndromes, including pneumothorax, pneumomediastinum, and interstitial pulmonary emphysema, occur in 10-30% of neonates with MAS (Moore et al., 2020). These complications can significantly impact neonatal respiratory function, leading to prolonged mechanical ventilation and increased risk of secondary infections (Mayo Clinic, 2022).

Risk Factors and Causes of MAS

Several risk factors contribute to the development of Meconium Aspiration Syndrome (MAS), primarily conditions that stimulate intrauterine meconium passage. One of the key factors is placental insufficiency, which can lead to chronic fetal hypoxia. Additionally, maternal hypertension and preeclampsia may cause uteroplacental insufficiency, further increasing the risk of fetal distress. Another critical factor is oligohydramnios, which reduces the ability of

amniotic fluid to dilute meconium, increasing the likelihood of meconium aspiration (Underwood et al., 2005).

Maternal infections, particularly chorioamnionitis, can trigger inflammatory responses in the fetus, leading to meconium passage in utero. In cases of fetal hypoxia, the fetus may begin gasping reflexively, which can induce involuntary meconium expulsion into the amniotic fluid. The inability to effectively clear meconium from the airways before the first breath also plays a significant role in the development of MAS. Moreover, the use of positive pressure ventilation before meconium suctioning may inadvertently push meconium deeper into the lower airways, exacerbating respiratory complications (Underwood et al., 2005).

Another important factor is prolonged or difficult labor, particularly in cases of prolonged rupture of membranes, which increases the risk of intrauterine infection in both the mother and neonate. Neonates are generally more vulnerable than mothers, and infections in neonates are often harder to detect. Studies estimate that approximately 26% of neonatal deaths are attributed to infections occurring during the perinatal period (Moore et al., 2020).

Additional risk factors for MAS include post-term pregnancy (≥ 42 weeks gestation), which is strongly associated with an increased likelihood of meconium passage. Furthermore, maternal substance abuse, particularly cocaine use, can induce fetal distress, further elevating the risk of meconium aspiration. Maternal smoking has also been identified as a significant contributor, as it increases the risk of neonatal respiratory infections, asthma, and other airway diseases (Mayo Clinic, 2022).

Impact of Maternal Smoking on Neonatal Respiratory Health

Recent studies have explored the effects of maternal smoking during pregnancy on neonatal toll-like receptor (TLR) responses, which are crucial for immune regulation. A prospective cohort study examined neonatal immune responses by comparing cytokine levels in umbilical cord blood from neonates born to smoking and non-smoking mothers (Underwood et al., 2005). The findings revealed that neonates born to smoking mothers exhibited weakened TLR-mediated immune responses, making them more susceptible to infections and inflammatory diseases. Additionally, these neonates had impaired lung development, leading to a higher risk of respiratory infections and asthma.

Another significant finding was the presence of elevated cotinine levels, a nicotine metabolite, in both maternal and umbilical cord blood, confirming prenatal tobacco exposure. Furthermore, exposure to both prenatal and postnatal tobacco smoke has been strongly correlated with reduced lung growth and impaired airway function, increasing the likelihood of respiratory complications. Neonates exposed to tobacco smoke are at a higher risk of developing respiratory infections, otitis media, and childhood asthma. Moreover, maternal smoking has been associated with an increased risk of Sudden Infant Death Syndrome (SIDS), behavioral disorders, and neurological abnormalities.

Additionally, there is evidence that children born to smoking mothers are more likely to initiate smoking during adolescence, indicating a generational impact of maternal smoking (Cunningham et al., 2018). These findings highlight the long-term consequences of maternal smoking on neonatal respiratory health and emphasize the urgent need for effective smoking cessation programs during pregnancy to mitigate these risks.

Mechanism of MAS Development

Meconium is considered highly toxic to the lungs, and its pathophysiological effects involve multiple mechanisms, making it difficult to determine which is the most dominant in each case. The development of Meconium Aspiration Syndrome (MAS) is primarily driven by four major mechanisms: mechanical airway obstruction, chemical pneumonitis, pulmonary vascular constriction, and surfactant inactivation. The first mechanism, mechanical airway obstruction, occurs when meconium partially or completely blocks the airways, leading to air trapping, atelectasis (lung collapse), and impaired gas exchange. This obstruction prevents effective oxygenation, increasing the risk of hypoxia and respiratory distress.

The second mechanism is chemical pneumonitis, where meconium acts as an inflammatory and irritative agent, triggering alveolar inflammation, pulmonary edema, and surfactant dysfunction. The presence of meconium in the lower airways causes an immune response that worsens pulmonary injury, further exacerbating respiratory complications. Another key mechanism in MAS development is pulmonary vascular constriction, in which inhaled meconium induces vasoconstriction of pulmonary blood vessels, leading to persistent pulmonary hypertension of the newborn (PPHN). This vascular resistance impairs normal blood flow to the lungs, preventing effective oxygenation and carbon dioxide removal, which can result in severe hypoxia.

Finally, surfactant inactivation plays a significant role in MAS. Meconium deactivates surfactant, a critical substance responsible for reducing surface tension in the alveoli. Without adequate surfactant, lung expansion becomes impaired, resulting in atelectasis and severe respiratory distress (Moore et al., 2020).

Diagnosis of Meconium Aspiration Syndrome (MAS)

Meconium Aspiration Syndrome (MAS) is typically diagnosed in neonates born through meconium-stained amniotic fluid (MSAF) who develop respiratory distress shortly after birth. The severity of MAS is classified into mild, moderate, and severe based on the degree of oxygen support required, which may include oxygen concentration $>40\%$ or mechanical ventilation (Cunningham et al., 2018). Radiological findings in MAS vary but commonly include bilateral infiltrates, which indicate pulmonary inflammation. Other notable findings include areas of hyperinflation due to air trapping and consolidation with atelectasis (lung collapse), which is frequently observed in severe neonatal cases. These findings help confirm the diagnosis of MAS and assess the extent of lung involvement.

Causes of MAS

Several factors contribute to the intrauterine passage of meconium, increasing the risk of MAS. Placental insufficiency is a major cause, as it leads to chronic fetal hypoxia, triggering fetal distress and meconium passage into the amniotic fluid. Other contributing factors include maternal hypertension, preeclampsia, oligohydramnios (low amniotic fluid levels), and maternal substance abuse, particularly tobacco and cocaine use. Maternal infections such as chorioamnionitis can also stimulate inflammatory responses that increase the risk of meconium-stained amniotic fluid. Additionally, fetal hypoxia may lead to intrauterine gasping, causing the fetus to inhale meconium into the airways before birth, which is a major contributing factor to MAS (Moore et al., 2020).

Symptoms and Clinical Manifestations

Before birth, fetal monitoring may show heart rate decelerations, which could indicate fetal distress. At delivery, meconium-stained amniotic fluid (MSAF) and a low Apgar score are significant diagnostic indicators of MAS. The presence of meconium in the airways can be directly visualized using laryngoscopy, providing a more definitive diagnosis. Additionally, auscultation of the lungs using a stethoscope may reveal abnormal breath sounds, such as coarse crackles or decreased air entry, further supporting the diagnosis. Arterial Blood Gas (ABG) analysis in neonates with MAS typically shows acidosis, along with low oxygen levels (PaO_2) and elevated carbon dioxide levels (PaCO_2), reflecting impaired gas exchange. Chest X-rays often reveal patchy infiltrates or diffuse opacities, confirming meconium aspiration and its impact on lung function (Cunningham et al., 2018).

Clinically, MAS is characterized by early-onset respiratory distress, usually within the first two hours of life, especially in neonates with meconium-stained amniotic fluid (MSAF). Key symptoms include tachypnea (rapid breathing), cyanosis (bluish discoloration due to low oxygen levels), and varying degrees of hyperinflation, which indicate air trapping within the lungs. On auscultation, MAS is often associated with coarse, wet crackles and expiratory grunting, which suggest airway obstruction due to the presence of meconium.

Physical Examination Findings

Severe cases of MAS often present with significant respiratory distress, including cyanosis, expiratory grunting, nasal flaring, intercostal retractions, and tachypnea (rapid breathing rate). In some cases, a barrel-shaped chest may be observed, which is indicative of air trapping. Additionally, yellowish discoloration of the fingernails, umbilical cord, and skin can be seen in neonates with severe meconium exposure (Moore et al., 2020).

Diagnostic Considerations

Neonates with MAS frequently experience prenatal and postnatal hypoxia, increasing the risk of neurological injury. Long-term neurological deficits may result from perinatal hypoxia, which can lead to legal concerns and potential litigation by parents. It is crucial for medical professionals involved in neonatal resuscitation and intensive care to ensure proper documentation and management of MAS cases (Mayo Clinic, 2022).

Differential Diagnosis

Several conditions may mimic MAS, making differential diagnosis important in neonates with respiratory distress. Possible alternative diagnoses include other aspiration syndromes, congenital heart disease with pulmonary hypertension, diaphragmatic hernia, neonatal pneumonia, primary pulmonary hypertension, neonatal sepsis, Persistent Pulmonary Hypertension of the Newborn (PPHN), surfactant deficiency, transient tachypnea of the newborn (TTN), and transposition of the great arteries (Cunningham et al., 2018). These conditions must be carefully ruled out through clinical assessment, imaging, and laboratory tests.

Pathophysiology of MAS

MAS involves complex physiological mechanisms, primarily centered around airway obstruction, chemical pneumonitis, pulmonary vasoconstriction, and surfactant dysfunction. Airway obstruction occurs when meconium blocks the airways, leading to atelectasis (lung collapse) or ball-valve obstruction, trapping air in the alveoli. If the obstruction is partial, it results in air trapping, whereas complete obstruction causes severe hypoxia and hypercapnia, further complicating neonatal breathing efforts. In addition to airway obstruction, chemical pneumonitis occurs as meconium induces an inflammatory response, leading to alveolar damage and lung dysfunction. The presence of inflammatory cytokines, including interleukins and prostaglandins, exacerbates lung injury, increasing the severity of MAS.

Another key factor in the development of MAS is pulmonary vasoconstriction and vascular dysfunction. The inhalation of meconium can trigger persistent pulmonary hypertension (PPHN), leading to right-to-left shunting of blood, resulting in severe hypoxia. Prolonged intrauterine hypoxia contributes to pulmonary artery thickening, which further exacerbates pulmonary hypertension and worsens neonatal respiratory function (Underwood et al., 2005). Surfactant dysfunction is also a crucial aspect of MAS pathophysiology. Meconium inactivates surfactant, a critical substance required to reduce surface tension in the alveoli. The loss of functional surfactant results in alveolar collapse and impaired gas exchange, further contributing to hypoxia, hypercapnia, and respiratory distress syndrome (RDS).

Inflammatory Response in MAS

MAS also involves a significant inflammatory response, which leads to prolonged respiratory complications. The presence of meconium in the lungs causes intense alveolar inflammation, leading to persistent hypoxia and lung injury. The inactivation of surfactant and the irritative effects of meconium exacerbate neonatal respiratory distress. Additionally, inflammatory cytokines play a major role in exacerbating pulmonary complications, making MAS a serious neonatal condition that requires immediate medical intervention (Mayo Clinic, 2022).

Radiological Examination of MAS

Although the classic radiological features of Meconium Aspiration Syndrome (MAS) have been described as diffuse, patchy infiltrates, its underlying pathogenesis leads to variable

radiographic abnormalities. The most common radiological findings in MAS include consolidation, atelectasis (collapsed lung), pleural effusion, air leak syndromes (such as pneumothorax or pneumomediastinum), hyperinflation, and wet lung appearance due to hypovascular pulmonary patterns. Additionally, MAS often presents with diffuse chemical pneumonitis, caused by the toxic effects of meconium content in the airways (Cunningham et al., 2018).

Key radiographic findings in MAS are crucial for diagnosis and include confirmation of MAS and assessment of intrathoracic pathology, identification of atelectasis and air leak syndromes, verification of endotracheal tube and umbilical catheter positioning, differentiation of air trapping and hyperexpansion from airway obstruction, recognition of acute atelectasis, and detection of chemical pneumonitis due to meconium aspiration. These imaging findings play a crucial role in determining the severity of MAS and guiding treatment decisions (Moore et al., 2020).

Complications of MAS

MAS can lead to several serious complications, which can significantly impact neonatal outcomes. One of the major complications is infection, as sepsis is a known trigger for intrauterine meconium passage. In preterm neonates, *Listeria monocytogenes* is a potential causative agent, and since meconium provides a rich medium for bacterial growth, secondary infections may occur (Underwood et al., 2005). Another serious complication is pneumothorax, which results from air trapping in the lungs, occurring in all severities of MAS. Early recognition is critical, especially in cases of sudden clinical deterioration. MAS may also lead to respiratory failure, which can result from airway obstruction, inflammation, infection, or intrapulmonary shunting, further worsening the neonate's condition.

A more severe complication is Persistent Pulmonary Hypertension of the Newborn (PPHN), which is frequently associated with severe MAS and is often difficult to manage. Early assessment and prevention of conditions such as hypoxemia, hypothermia, and hypoglycemia are crucial to reducing the risk of persistent pulmonary hypertension and improving neonatal outcomes (Mayo Clinic, 2022).

Management of MAS

1. Prevention

Delivery Room Management plays a crucial role in preventing MAS complications. One of the primary interventions is suctioning of Meconium-Stained Amniotic Fluid (MSAF). Previously, neonatal resuscitation guidelines recommended oropharyngeal and nasopharyngeal suctioning during delivery; however, current guidelines suggest waiting until the neonate is fully delivered before assessing their condition. A vigorous neonate is defined as one who cries or breathes spontaneously, has good muscle tone, pink skin color, and a heart rate >100 beats per minute. In vigorous neonates, routine endotracheal suctioning is not recommended, as it provides no significant benefit and may increase the risk of airway trauma. However, in non-vigorous neonates (those with poor tone, absent breathing, or bradycardia), immediate endotracheal suctioning via laryngoscopy should be performed to clear the airway and prevent further complications.

Another important consideration is the use of antibiotics in MAS, which remains controversial. Some experts recommend empiric antibiotic therapy, as early MAS can mimic congenital pneumonia, making it difficult to distinguish between the two conditions. Empiric antibiotic therapy, typically using ampicillin and gentamicin, is often initiated but should be discontinued within 48-72 hours if blood cultures are negative, to prevent unnecessary antibiotic exposure (Cunningham et al., 2018).

2. Further MAS Management

Antibiotic use in MAS remains an area of ongoing research. Since early MAS can resemble pneumonia, empiric antibiotics may be reasonable in certain cases. However, if blood cultures return negative, antibiotics should be discontinued within 48-72 hours to minimize unnecessary antimicrobial exposure and reduce antibiotic resistance risks. Chest physiotherapy is often used to mobilize airway secretions and improve oxygenation efficiency. This therapy includes

techniques such as postural drainage, percussion, vibration therapy, and oropharyngeal or tracheal suctioning, all of which help clear meconium and mucus buildup in the airways (Moore et al., 2020).

Mechanical ventilation is required in approximately one-third of MAS cases due to air leak complications. Positive Pressure Ventilation (PPV) is commonly needed, and FiO_2 levels may need to be increased up to 100% to ensure adequate oxygenation. When using mechanical ventilation in MAS, optimal settings include low inspiratory pressure, short inspiratory time, and a high respiratory rate to reduce lung overdistension and minimize complications. Additionally, since PPHN is associated with MAS in nearly two-thirds of cases, hyperventilation may be used to induce respiratory alkalosis, promoting pulmonary vasodilation and improving oxygenation.

High-Frequency Ventilation (HFV) is an advanced ventilation strategy that allows for efficient gas exchange at low tidal volumes, minimizing lung injury risks. The potential benefits of HFV include improved airway secretion clearance, easier induction of respiratory alkalosis, and a lower risk of histopathological lung damage compared to conventional ventilation. For the most severe cases of MAS, Extracorporeal Membrane Oxygenation (ECMO) may be considered. ECMO is a form of cardiopulmonary bypass that provides temporary respiratory and cardiac support when conventional mechanical ventilation and HFV fail. It is typically reserved for neonates with severe MAS and refractory respiratory failure, who do not respond to standard interventions (Underwood et al., 2005).

4. CONCLUSION

Meconium Aspiration Syndrome (MAS) remains a significant cause of neonatal respiratory distress, requiring early detection, proper diagnosis, and effective management to prevent severe complications. The presence of meconium-stained amniotic fluid (MSAF) during labor is a critical risk factor for MAS, often associated with placental insufficiency, maternal hypertension, preeclampsia, infections, oligohydramnios, and fetal distress leading to intrauterine gasping. The composition and functions of amniotic fluid play a crucial role in fetal development, protection, and immune support, but its contamination with meconium can result in severe pulmonary complications.

The pathophysiology of MAS is complex, involving mechanical airway obstruction, chemical pneumonitis, pulmonary vascular constriction, and surfactant inactivation, which together contribute to respiratory failure, persistent pulmonary hypertension of the newborn (PPHN), and systemic hypoxia. Radiological imaging, including chest X-rays, is essential for confirming the diagnosis, revealing characteristic findings such as bilateral infiltrates, air trapping, consolidation, and atelectasis. Effective management of MAS includes immediate neonatal resuscitation, airway clearance, oxygen therapy, mechanical ventilation, and advanced strategies such as High-Frequency Ventilation (HFV) and Extracorporeal Membrane Oxygenation (ECMO) for severe cases. The role of antibiotics in MAS remains controversial, but empirical therapy may be considered when distinguishing MAS from neonatal pneumonia.

Preventive strategies, including proper delivery room management, have been refined, with recent guidelines emphasizing selective suctioning based on neonatal condition rather than routine oropharyngeal suctioning. Maternal factors such as smoking and substance abuse further increase the risk of MAS and neonatal respiratory issues, underscoring the importance of prenatal care and public health interventions. In conclusion, MAS requires a multidisciplinary approach that integrates prenatal risk assessment, early neonatal intervention, and evidence-based treatment strategies to reduce morbidity and improve neonatal outcomes. Future research should continue to explore optimized ventilation strategies, surfactant therapy, and targeted preventive measures to further improve neonatal survival and long-term respiratory health.

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