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Case report

MECONIUM ASPIRATION SYNDROME AND HYDROCEPHALUS: A CASE REPORT

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Abstract

Meconium aspiration syndrome (MAS) is an uncommon but severe neonatal condition in which the presence of meconium within the airways leads to mechanical obstruction, chemical inflammation and surfactant dysfunction. Initial stabilization in the delivery room plays a fundamental role in determining outcomes. Although MAS is well described in the literature, intracranial hemorrhage and the subsequent development of hydrocephalus represent exceptionally rare complications, most often associated with severe hypoxia, hemodynamic instability, and the fragility of the neonatal cerebral vasculature.

We present the case of a term male newborn delivered through meconium-stained amniotic fluid who developed respiratory distress. Upon admission to the neonatal intensive care unit, blood gas analysis revealed respiratory acidosis, while chest radiography demonstrated a “snowstorm” appearance with confluent nodular opacities and areas of atelectasis and hyperinflation. The infant required endotracheal intubation and conventional mechanical ventilation, followed by surfactant lavages, antibiotic and corticosteroid therapy, and initiation of a phosphodiesterase-5 inhibitor due to emerging pulmonary hypertension. After extubation, non-invasive ventilation with B-CPAP was applied, followed by oxygen supplementation. On day 40 of hospitalization, neurological abnormalities and the “sunset eye” sign were noted. Subsequent ultrasound and neuroimaging revealed marked ventricular dilation and intracranial hemorrhage. Following neurosurgical consultation, a ventriculoperitoneal shunt was placed, after which the infant was transferred for continued management and ultimately discharged home.

Meconium aspiration syndrome remains an important cause of neonatal respiratory distress and warrants timely recognition of associated comorbidities such as hydrocephalus. Early detection and a multidisciplinary approach are essential for optimizing neurological and respiratory outcomes in newborns.

Keywords: meconium aspiration syndrome, respiratory distress, hydrocephalus, newborn

Introduction

Meconium aspiration syndrome (MAS) is a clinical condition most commonly seen in term and post-term newborns as a result of intrauterine or intrapartum aspiration of meconium-contaminated amniotic fluid[1]. The presence of meconium in the amniotic fluid

indicates intrauterine fetal distress, whereby hypoxia stimulates increased intestinal peristalsis and relaxation of the anal sphincter, leading to premature passage of meconium into the amniotic cavity[2]. Aspiration may occur *in utero*, but most commonly occurs during labor, particularly in the setting of impaired fetal oxygenation. Only 3-12% of infants born through meconium-stained amniotic fluid develop clinically significant MAS. Among them, approximately 20% are non-vigorous at birth, about one-third require intubation and mechanical ventilation, and 5-12% result in mortality[6,7].

The pathophysiology of MAS is multifactorial, involving several key mechanisms: mechanical obstruction of the airways, inactivation or dysfunction of pulmonary surfactant, development of chemical pneumonitis and secondary inflammatory response, as well as pulmonary hypertension. These mechanisms lead to ventilation–perfusion mismatch, culminating in severe hypoxemia and hypercapnia[1,5].

The clinical manifestations of MAS span a wide spectrum, ranging from mild respiratory distress to severe respiratory failure with associated systemic complications. The severity of MAS may be classified as follows: mild disease (FiO_2<40% for less than 48 hours), moderate disease (FiO_2>40% for more than 48 hours without signs of air leak), or severe disease (requiring mechanical ventilation for more than 48 hours and/or development of pulmonary hypertension)[3].

Although MAS is well described in neonatal literature, neurological complications - particularly intracranial hemorrhage - remain exceedingly rare and relatively underreported. Prolonged systemic hypoxia, hemodynamic fluctuations, the need for aggressive mechanical ventilation, and the presence of metabolic dysregulation are all factors that may contribute to injury of the fragile cerebral vasculature in newborns. When intracranial hemorrhage occurs, it may lead to significant consequences, the most important being disruption of cerebrospinal fluid circulation and the development of posthemorrhagic hydrocephalus. According to available data, hydrocephalus develops in 25-50% of infants with intracranial hemorrhage, often necessitating neurosurgical intervention, typically through placement of a ventriculoperitoneal shunt[1].

Given the rarity of this complication in the context of MAS, presenting such a case holds substantial clinical and scientific relevance. A detailed analysis of the clinical course, diagnostic workup, and therapeutic approaches may contribute to better understanding of the underlying pathophysiological mechanisms, facilitate timely recognition of these conditions, and enhance multidisciplinary management in newborns with severe forms of MAS.

Case report

We report the case of a term male newborn, delivered at 42 weeks of gestation via caesarean section,born through meconium-stained amniotic fluid, with the birth weight of 4120 g, the birth length of 52 cm, and the Apgar scores of 7 at both 1 and 5 minutes. Immediately after birth, in the delivery room at the local medical center, an endotracheal suctioning was performed under direct laryngoscopy, yielding thick meconium, after which the infant was placed on supplemental oxygen and was transferred to the Neonatal Intensive Care Unit (NICU) at University Children's Hospital in Skopje. On admission, the infant appeared critically ill, with clinical signs of respiratory distress - tachycardic, tachydispnoic, with marked intercostal retractions and generalized cyanosis. Auscultation of the lungs revealed diminished vesicular breath sounds with bilateral coarse crackles. Immediately after admission, the newborn was sedated, intubated, and placed on invasive conventional mechanical ventilation, requiring high peak inspiratory pressures (PIP) due to increased airway resistance and reduced pulmonary compliance. Initial laboratory testing demonstrated respiratory acidosis. On admission, inflammatory markers were within normal limits (CRP 0.6 mg/L; procalcitonin 0.66 ng/mL), but by day 4 showed a significant rise (CRP 19.5 mg/L;

procalcitonin 7.99 ng/mL), with further upward trending. Additional biochemical analyses revealed mild hypoalbuminemia (albumin 30.5 g/L) and elevated lactate dehydrogenase (LDH 855 U/L). Chest radiography demonstrated bilaterally reduced pulmonary transparency with reticular opacities and areas of confluent shadowing consistent with consolidation.(Fig.1.)

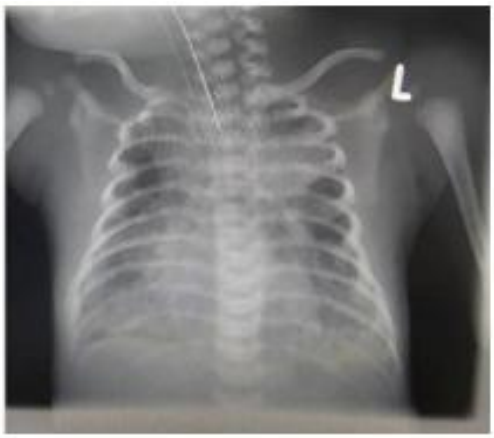

Fig. 1. Chest RTG - bilaterally reduced pulmonary transparency with reticular opacities and areas of confluent shadowing consistent with consolidation

Echocardiography showed hypertrophy of the interventricular septum and right ventricle, along with the presence of patent fetal circulatory pathways. Neurosonography revealed normal ventricular dimensions with evidence of grade I intraventricular hemorrhage. Empiric dual antibiotic therapy with a carbapenem and an aminoglycoside was initiated, along with corticosteroids. Due to hemodynamic instability, inotropic support was introduced. Surfactant was administered on three separate occasions. Despite intensive management, the infant remained clinically unstable in the days that followed, with recurrent desaturation episodes and a persistent need for frequent airway suctioning.

Around day 15 of hospitalization, the clinical condition deteriorated further, characterized by severe tachypnea, generalized edema and oliguria, prompting the initiation of a second inotrope and a diuretic. Sildenafil, a phosphodiesterase-5 inhibitor, was added due to emerging pulmonary hypertension. The infant also received albumin and fresh frozen plasma. In response to the progressive decrease in hemoglobin levels, the infant received a transfusion of filtered red blood cells. Gradual clinical stabilization followed, allowing progressive reduction of ventilatory support. On day 31, the infant was successfully extubated and transitioned to non-invasive ventilation with B-CPAP, and subsequently to oxygen supplementation via high-flow nasal cannula (HFNC) as his condition improved.

Around day 40, the infant developed feeding intolerance with vomiting, altered consciousness, neurological deterioration, and the characteristic “sunset sign,” raising strong suspicion for intracranial pathology. Urgent neurosonography revealed markedly dilated ventricles consistent with hydrocephalus.(Fig.2,A) Brain computed tomography confirmed a significant enlargement of the ventricular system with paraventricular hemorrhage and a focal left occipital subependymal hemorrhage, accompanied by an adjacent hypodense infarct zone.(Fig.2,B).

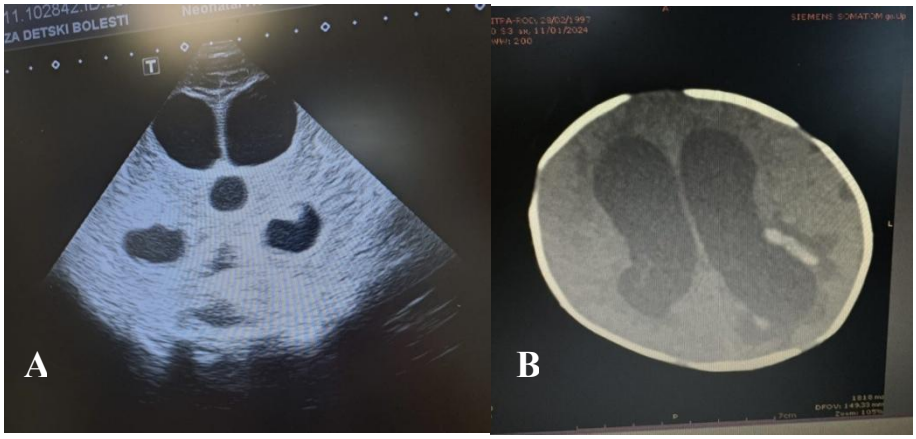

Fig. 2. Neurosonography and brain CT prior to neurosurgical intervention
A – Dilatation of the ventricular system.
B – Dilatation of the ventricular system with hydrocephalus, accompanied by left-sided paraventricular and focal occipital subependymal hemorrhage, as well as an adjacent hypodense infarct zone.

Neurosurgical evaluation established a clear indication for ventriculoperitoneal shunt placement, which was performed without complications.(Fig.3) The infant was readmitted to the NICU for continued intensive management.

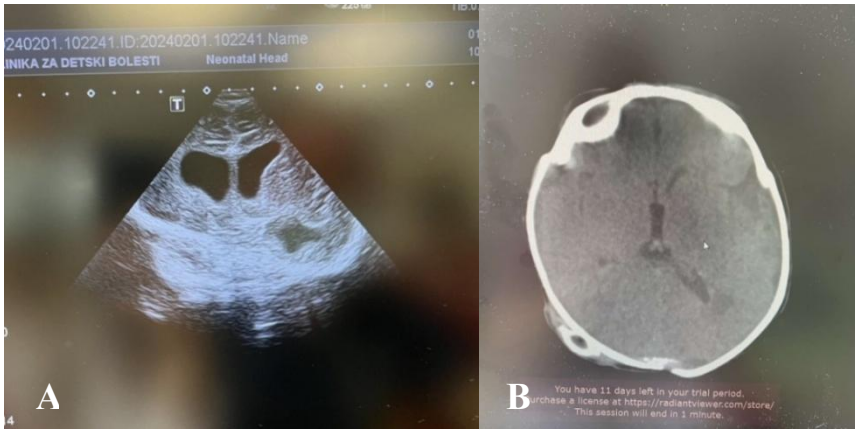

Fig.3 Neurosonography and Brain CT after VP shunt placement
A – Decreased ventricular volume compared to the previous imaging study — 2 weeks after the intervention.
B – Markedly reduced ventricular volume compared to the prior brain CT — 3 weeks after the intervention.

Due to persistent tachypnea and the ongoing need for oxygen supplementation, an additional computed tomography (CT) scan of the lungs was performed.(Fig.4) The imaging revealed residual adhesions, as well as areas of air trapping and atelectasis.

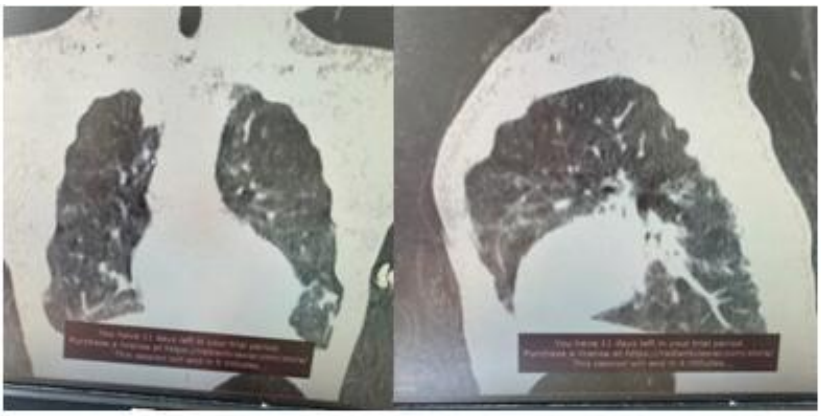

Fig.4 Chest CT - residual adhesions and areas of air trapping and atelectasis, as a consequence of the underlying condition

These findings indicated irreversible damage to the pulmonary parenchyma, characteristic of severe forms of meconium aspiration syndrome, most likely developing as a consequence of the underlying condition.

Discussion

Meconium aspiration syndrome (MAS) remains a significant clinical challenge in neonatal practice despite substantial advancements in perinatal and intensive care. Outcomes in infants with MAS are largely determined by the quality of initial assessment and stabilization in the delivery room, as well as the timely initiation of appropriate treatment in accordance with current international guidelines. Recommendations from the American Academy of Pediatrics (AAP) and the Neonatal Resuscitation Program (NRP) emphasize the importance of a systematic, evidence-based approach to newborns exposed to meconium-stained amniotic fluid (MSAF).

Current neonatal resuscitation protocols clearly state that routine endotracheal suctioning is not indicated for vigorous newborns delivered through MSAF. Conversely, in non-vigorous infants, rapid evaluation of muscle tone, respiratory effort, and heart rate is essential to determine the need for endotracheal suctioning and ventilatory support. Accurate identification of infants who may benefit from deep suctioning is crucial, as inadequate removal of thick meconium can result in significant airway obstruction, atelectasis, hypoventilation, and the development of persistent pulmonary hypertension of the newborn (PPHN). Conversely, unnecessary or overly aggressive suctioning may cause airway trauma, bradycardia, or even secondary hypoxia due to delayed ventilation initiation[3].

In the presented case, although deep suctioning was appropriately performed in the delivery room with successful evacuation of thick meconium, the infant nonetheless developed severe MAS. This highlights the complex pathophysiology of the condition: heavy meconium load, surfactant inactivation, chemical pneumonitis, and excessive pulmonary vasoconstriction act synergistically to precipitate severe respiratory failure - even when initial management is optimal.

Infants who develop the severe respiratory form of MAS frequently require aggressive respiratory support, including high-parameter mechanical ventilation, repeated surfactant administration, vasodilator therapy for PPHN, and inotropic support during hemodynamic instability. Ventilatory support in neonates with MAS is difficult to optimize, given the coexistence of collapsed and hyperinflated lung segments, substantial ventilation–perfusion mismatch, and varying degrees of airway compromise[8,10]. Mechanical ventilation carries certain risks, such as barotrauma, volutrauma, refractory atelectasis and chronic lung

injury. The primary goal of ventilation is to improve oxygenation while minimizing barotrauma, thereby reducing the risk of air-leak syndromes. Studies have demonstrated that infants requiring prolonged mechanical ventilation are at higher risk for long-term respiratory morbidity, including recurrent infections, asthma-like symptoms, impaired lung growth, and reduced pulmonary function later in childhood[8,4].

Neurological complications, although much less common, can be profound. Severe MAS may be associated with hypoxic-ischemic injury, impaired cerebral perfusion secondary to PPHN, hemodynamic fluctuations, and coagulation disturbances-all of which may predispose to intraventricular or parenchymal hemorrhage.There are no large studies directly reporting the incidence of intracerebral hemorrhage specifically in MAS; available data often originate from populations of critically ill neonates or those requiring ECMO support. For example, one ECMO cohort (with MAS as the most common indication) found intracranial hemorrhage in approximately 13% of imaged infants[6]. The true incidence of intracranial hemorrhage in MAS is likely underestimated due to the lack of routine neuroimaging in all affected infants and the potential underdiagnosis of subclinical intraventricular or micro-hemorrhages.

In our case, paraventricular and subependymal hemorrhage with secondary post-hemorrhagic hydrocephalus was identified after 40 days of hospitalization - a rare, severe, and largely unpredictable complication in the context of MAS. Although neurological complications in MAS are considered uncommon, their occurrence cannot always be anticipated, even in infants receiving timely and appropriate therapy. The interplay of severe hypoxia, hemodynamic instability, fluctuations in cerebral perfusion, and inflammatory processes during prolonged critical illness creates conditions in which the fragile neonatal cerebral vasculature becomes particularly susceptible to rupture.

Post-hemorrhagic hydrocephalus, as occurred in this patient, represents a serious consequence of intracranial bleeding. Impairment of cerebrospinal fluid circulation leads to progressive ventricular dilation and increased intracranial pressure. This condition requires urgent neurosurgical intervention - placement of a ventriculoperitoneal shunt - to restore CSF dynamics and prevent further neurological damage. Although often lifesaving, shunt placement marks the beginning of long-term monitoring due to risks of shunt malfunction, infection, and the need for future revisions.

Hemorrhagic and CSF-related complications of this nature have significant implications for long-term neurodevelopment, potentially affecting cognitive, motor, and sensory functions. Therefore, these infants require continuous multidisciplinary follow-up, including regular neurological evaluations, serial neurosonography or neuroimaging, and structured developmental surveillance. Early identification of psychomotor delay enables timely initiation of targeted rehabilitation programs, thereby improving outcomes and minimizing permanent deficits.

This case illustrates that despite adherence to all current protocols, some infants with severe MAS may develop unexpected and difficult-to-predict complications requiring prolonged medical follow-up. Long-term sequelae in infants surviving severe MAS may include pulmonary complications-chronic lung disease, persistent atelectasis, bronchiolitis obliterans, reduced exercise tolerance; and neurological sequelae-delayed acquisition of motor developmental milestones, cognitive impairment, motor dysfunction, tone abnormalities, and consequences of hemorrhagic or ischemic brain injury. For these reasons, international guidelines recommend structured multidisciplinary follow-up, including regular pulmonary, neurological, and developmental assessments; rehabilitation and physical therapy; monitoring of growth and nutritional status; and comprehensive psychomotor and cognitive evaluations during the first two years of life[4,5].

Physical and occupational therapy play a crucial role in infants with prolonged NICU stay with critical illness or neurological injury. Early rehabilitation optimizes motor development, improves tone control, prevents contractures, and enhances sensory integration. Standardized developmental screenings, such as the Bayley Scales, are recommended at 6, 12, and 24 months of age for early detection of potential developmental disorders[9].

Conclusion

The presented case highlights the complexity of meconium aspiration syndrome (MAS), particularly when accompanied by rare but severe neurological complications such as intracranial hemorrhage and post-hemorrhagic hydrocephalus. Timely and accurate initial assessment in the delivery room, together with strict adherence to contemporary neonatal resuscitation protocols, remains essential for reducing the severity of MAS and preventing secondary injury. The clinical course in these infants is often unpredictable and requires dynamic therapeutic adjustments, including optimization of ventilatory support, repeated surfactant administration, management of pulmonary hypertension, and early recognition of systemic consequences arising from prolonged hypoxia.

The neurological complications demonstrated in this case underscore the importance of rigorous monitoring, serial neurosonography, and prompt neurosurgical intervention when indicated. Concurrently, the persistent abnormalities observed within the pulmonary parenchyma point to the potential for long-term respiratory sequelae, thereby justifying the involvement of a pediatric pulmonologist as part of the multidisciplinary follow-up.

In the post-hospitalization period, these infants require structured and prolonged follow-up, including developmental assessments, physical and occupational therapy, and regular neurological and pulmonary evaluations. Early intervention and early detection of developmental impairments can significantly improve functional outcomes and minimize long-term deficits.

This case emphasizes that successful management of severe MAS requires an integrated, multidisciplinary and timely approach, one that begins with optimal care in the delivery room and continues with vigilant follow-up and rehabilitation throughout the child's subsequent developmental stages.

Conflict of interest statement. None declared.

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