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THE SIGNIFICANCE OF DIAGNOSTIC COMPONENTS OF MECONIUM ASPIRATION SYNDROME IN THE MANAGEMENT OF NEWBORNS

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Summary.

Effective and timely diagnosis represents a crucial component in the prevention and treatment of all neonatal diseases. It serves as the coordinating mechanism in the comprehensive management of newborns with meconium aspiration syndrome (MAS), a severe condition that may originate prenatally, progress rapidly, and result to potentially dangerous consequences manifesting both immediately postpartum and during later developmental stages.

The MAS diagnostic process requires that obstetricians and neonatologists possess extensive knowledge and skills across multiple medical disciplines, encompassing an understanding of biochemical and cellular pathological processes, integrated comprehension of organ system function, and whole-body physiology, while accounting for gestational age at birth. Development of precise management strategies for neonates delivered through meconium-stained amniotic fluid relies on pregnancy monitoring data, ultrasound evaluation of fetal status, placental characteristics, amniotic fluid analysis, and cardiotocography results.

Meconium aspiration syndrome arises from the aspiration of meconium-stained amniotic fluid occurring before, during, or immediately after delivery. Meconium functions as a noxious substance that rapidly injures immature and hypersensitive pulmonary tissue, initiating systemic pathological cascades. The release of cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1β, IL-6, IL-8, and IL-13, triggers diffuse pneumonitis through airway and parenchymal irritation. Pneumonitis may develop within hours due to meconium-derived enzymes, bile salts, and free fatty acids. Quantification of TNF-α, IL-1β, IL-6, IL-8, and IL-13 provides diagnostic value for pneumonitis in MAS. Continuous monitoring of acid-base status and blood gas composition is essential for assessing MAS severity, as perinatal stress-induced metabolic acidosis combines with respiratory acidosis, while parenchymal injury is associated with persistent pulmonary hypertension of the newborn (PPHN).

MAS disrupts homeostatic balance through hypoxia and metabolic stress, potentially resulting in electrolyte disturbances. Sodium, potassium, and calcium ions play essential roles in cardiac function, vascular tone regulation, and neuromuscular conduction. Hyponatremia may serve as an early indicator of the syndrome of inappropriate antidiuretic hormone secretion (SIADH), which is frequently observed in severe MAS cases due to hypoxic stress or pulmonary injury. In SIADH, hyponatremia arises from water retention, thereby increasing the risk of cerebral edema and seizures. Acute kidney injury may induce potassium imbalances, commonly manifesting as hyperkalemia, a potentially life-threatening condition, alongside elevated nitrogenous waste products and acid-base disturbances. Both hypokalemia and hyperkalemia can precipitate severe cardiac arrhythmias, while tissue hypoxia or renal impairment may exacerbate potassium release into the circulation. Neonatal hypocalcemia may further aggravate cardiovascular instability and respiratory dysfunction associated with MAS.

In addition to the local effects of meconium on respiratory mucosa and pulmonary parenchyma, hypoxia and infection contribute significantly to the pathogenesis of MAS.

Primary fetal oxygen deprivation triggers a stress response characterized by hypoxia-induced anal sphincter relaxation and enhanced intestinal peristalsis, resulting in meconium passage into the amniotic fluid.

Concurrent hypoxia may provoke fetal gasping, a suffocation-like reflex, and respiratory spasms, facilitating aspiration of meconium into the airways prior to delivery. Early aspiration induces three principal pathophysiological mechanisms: mechanical airway obstruction, chemical pneumonitis with activation of inflammatory cascades, and surfactant dysfunction, collectively exacerbating neonatal hypoxia. Intrauterine infection, particularly chorioamnionitis, represents an independent risk factor for MAS development, substantially expanding the spectrum of required diagnostic parameters.

Complete blood count analysis (CBC) remains an essential routine diagnostic modality in neonates. CBC parameters serve as early indicators of pathological alterations, enabling clinicians to assess the severity of MAS. Specifically, erythrocyte count, platelet levels, hematocrit, and neutrophil/granulocyte concentrations reflect oxygen-carrying capacity and the risk of neonatal hemorrhage, infection, and chronic hypoxia.

Neurological examination allows detection of hypoxic brain injury, although optimal diagnostic yield requires prior patient stabilization. When clinically indicated, neuroimaging modalities, including magnetic resonance imaging (MRI), computed tomography (CT), or cranial ultrasonography, may provide additional diagnostic information.

Chest radiography performs three essential diagnostic functions in meconium aspiration syndrome (MAS): confirmation of the diagnosis and evaluation of disease severity, identification of atelectasis and air leak syndromes, and verification of correct positioning of endotracheal tubes and umbilical catheters. In contemporary neonatology, lung ultrasound (LUS) has demonstrated diagnostic efficacy comparable to conventional radiography. Echocardiography is indispensable for the assessment of cardiac anatomy and function, particularly for evaluating the severity of pulmonary hypertension and the presence of right-to-left shunting. In neonates with MAS-associated respiratory failure, echocardiographic assessment during the «golden hour» is strongly recommended.

This article emphasizes the imperative of systematic diagnostic approaches to MAS, which are fundamental for guiding acute management, preventing complications, and mitigating long-term sequelae.

Keywords: *Meconium Aspiration Syndrome; Meconium-Stained Amniotic Fluid; Diagnosis of MAS.*

Meconium-stained amniotic fluid (MSAF) occurs in 1 out of 7 pregnancies, accounting for 400,000 to 600,000 deliveries annually in the United States. The incidence increases with advancing gestational age. Meconium-stained amniotic fluid is observed in 27% of post-term pregnancies. MSAF is associated with adverse neonatal and maternal outcomes. Approximately 20-30% of neonates born through meconium-stained amniotic fluid demonstrate respiratory and neurological depression at birth. Approximately 10% of cases of neonatal respiratory distress are attributable to MAS. The incidence of MAS increases exponentially from 38 to 42 weeks of gestation. Globally, MAS frequency has decreased in developed countries due to improvements in obstetric practices and perinatal care, while remaining a significant problem in developing countries [62].

One of the most severe complications of meconium-stained amniotic fluid is meconium aspiration syndrome (MAS), occurring in in 5% of affected neonates. MAS is defined as respiratory distress in an infant born through meconium-stained amniotic fluid, with symptoms that cannot be otherwise explained [29]. The average incidence of MAS in Ukraine ranges from 2% and 3% of deliveries, whereas meconium-stained amniotic fluid is observed in 9-15% of cases [63].

In 5-12% of children with MAS, chronic bronchopulmonary diseases and neurological disorders develop [62]. The mortality rate associated with MAS is approximately 12% [29].

A systematic diagnostic approach ensuring early detection, accurate severity assessment, and coordinated multidisciplinary interventions is essential for preventing MAS development and its associated complications. This systematic approach comprises three interconnected levels: prenatal, intrapartum, and postnatal. At the prenatal stage, identification of high-risk groups (advanced gestational age, intrauterine infection, signs of fetal stress) enables preventive strategies, including planned labor induction or intraamniotic amnioinfusion, which significantly reduce MAS likelihood [29].

Intrapartum diagnostics involve continuous cardiotocographic monitoring, assessment of meconium-stained fluid, and immediate neonatal evaluation with preliminary diagnostic conclusions within the first seconds of life. This includes rational and maximally effective utilization of the 'golden minute,' objective Apgar score assessment, and timely initiation of personalized medical care algorithms for infants born through meconium-stained amniotic fluid.

Postnatal diagnostics are based on clinical examination, radiographic and ultrasound lung evaluation, and prompt laboratory monitoring (blood gas analysis, inflammatory markers, hypoxia indicators). Integrating these data within an interprofessional team, comprising neonatologist, pediatrician, radiologist, and laboratory specialist, enhances the accuracy of MAS severity assessment and facilitates optimal treatment algorithm selection [7,20,29,30,31,33,36,38,46,51]. The key diagnostic components of MAS in neonates primarily include pregnancy and delivery history data, fetal monitoring data through cardiotocography (CTG), stethoscope auscultation, and fetal heart rate assessment using handheld Doppler, together with the presence of meconium-stained amniotic fluid, which increases the risk of aspiration syndrome. Inflammatory processes in the amniotic sac stimulate cytokine and inflammatory mediator production, promoting meconium release and aspiration. Furthermore, intra-amniotic infection increases bacterial colonization of meconium, exacerbating chemical and infectious pneumonitis in neonatal lungs and impairing gas exchange. Infection-induced fetal systemic inflammatory syndrome enhances vasospasm and pulmonary hypertension, further compromising tissue oxygenation [48].

The diagnosis of MAS is established when meconium-stained amniotic fluid is present, traces or meconium are detected during tracheal suction, signs of neonatal respiratory distress are observed in the newborn, and characteristic radiographic abnormalities are identified. Clinical examination and physical assessment of the neonate with severe respiratory disturbances carry significant diagnostic weight in MAS cases, including findings such as cyanosis, tachypnea, grunting at end-expiration, nasal flaring, intercostal retractions, and barrel-shaped chest (increased anteroposterior diameter) due to air trapping, with auscultation revealing rales and grunting in some cases. Pulmonary effects may result in severe ventilation-perfusion mismatch (V/Q), oxygen dependence, and the requirement for mechanical ventilation, which contribute to determination of the specific severity grade of MAS.

Three severity grades of MAS are recognized:

- Mild: respiratory disturbances requiring oxygen therapy with FiO_2 <40% for less than 48 hours.
- Moderate: respiratory disturbances requiring oxygen therapy with FiO_2 ≥40% for more than 48 hours.
- Severe: respiratory disturbances requiring mechanical ventilation for more than 48 hours with development of persistent pulmonary hypertension syndrome [8].

Enzymes, bile salts, and free fatty acids in meconium irritate the airways and parenchyma, triggering cytokine release, including tumor necrosis factor (TNF)-α, interleukin (IL)-1β, IL-6, IL-8, IL-13, and initiating diffuse pneumonitis that may develop within hours after aspiration [43,48]. TNF-α, IL-1β, IL-6, IL-8, and IL-13 may serve as diagnostic markers for pneumonitis in MAS.

Laboratory investigations in MAS should include assessment of acid-base status, which is crucial for maintaining respiratory and acid-base homeostasis [41]. Metabolic acidosis caused by perinatal stress is compounded by respiratory acidosis, whereas parenchymal disease is associated with persistent pulmonary hypertension of the newborn (PPHN). Proper MAS management requires arterial blood gas measurements (pH, pCO_2, pO_2) and continuous oxygenation monitoring via pulse oximetry. The severity of lung injury can be assessed by the oxygenation index, interpreted as follows: an oxygenation index (PaO_2/FiO_2) <300 indicates acute lung injury, and an oxygenation index (PaO_2/FiO_2) <200 indicates severe acute respiratory distress syndrome. Serum electrolyte determination should also be incorporated in the diagnostic workup, with sodium,

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potassium, and calcium levels measured within the first 24 hours of life in MAS-affected neonates, considering that common complications of perinatal stress in these infants include syndrome of inappropriate antidiuretic hormone secretion and acute kidney injury.

For neonatal clinicians, performing a complete blood count (CBC) is a routine yet diagnostically valuable procedure. First, erythrocyte levels and hematocrit values reflect blood oxygen-carrying capacity: elevated hematocrit may indicate chronic intrauterine hypoxia, whereas decreased values suggest anemia and risk of insufficient tissue oxygenation in neonates. Such alterations are frequently observed in infants born through meconium-stained amniotic fluid and are associated with hypoxic stimulation of erythropoiesis or, conversely, reduced hematopoiesis during infectious processes. Second, platelet count possesses important prognostic significance: thrombocytopenia during the first hours of life increases the risk of neonatal hemorrhage, including pulmonary hemorrhage, which significantly aggravates MAS progression and elevates mortality in neonatal intensive care units. Early detection of thrombocytopenia permits timely correction (platelet transfusion) and reduces complications. Third, differential leukocyte count, particularly neutrophils and other granulocytes, facilitates identification of intrauterine or perinatal infection, representing an independent risk factor for MAS. Neutrophilia with left shift or neutropenia indicate bacterial infection that exacerbates chemical pneumonitis following meconium aspiration. Furthermore, an elevated immature-to-total neutrophil ratio serves as an early marker of systemic inflammation and assists in determining the necessity for antibiotic therapy within the first hours of life [64]. Thrombocytopenia increases the risk of neonatal hemorrhage, and intrauterine or perinatal blood loss, as well as infection, contributes to postnatal stress. Therefore, in MAS cases, the following CBC patterns may be observed: neutropenia or neutrophilia with left shift may indicate perinatal bacterial infection, whereas polycythemia may develop secondary to chronic fetal hypoxia. Polycythemia is associated with reduced pulmonary blood flow and may exacerbate hypoxia related to MAS and PPHN. Studies demonstrate that infants with MAS exhibit higher absolute nucleated red blood cell counts compared to neonates born through meconium-stained amniotic fluid without MAS manifestations [35, 53].

Imaging studies include chest radiography, which is essential for confirming MAS diagnosis and determining its severity, identifying areas of atelectasis and air leak syndromes, and verifying proper placement of endotracheal tubes and umbilical catheters. Lung ultrasound (LUS) may serve as a convenient, non-invasive, and accurate imaging modality for MAS diagnosis. Key MAS features identified by LUS include pulmonary consolidation with air bronchograms (all patients), pleural line abnormalities and absent A-lines (all patients), alveolar-interstitial syndrome or B-lines in non-consolidated areas (all patients), atelectasis in severe MAS (16.2% of severe cases), and pleural effusion (13.7% of patients) [39]. Patients with MAS require thorough cardiac evaluation and echocardiography to detect congenital heart defects and PPHN of the newborn. Determining the degree of PPHN prior to initiating treatment is crucial, making echocardiography ideally performed during the «golden hour» of the affected neonate's life.

Brain imaging also holds significant diagnostic value since hypoxia, which damages both fetal and neonatal nervous systems, represents a key factor in MAS development. Neurological examination should follow stabilization of the infant's condition. If abnormalities are detected, brain imaging (e.g., magnetic resonance imaging [MRI], computed tomography [CT] scanning, cranial ultrasonography) is indicated.

During evaluation and management, it is essential to exclude the following differential diagnoses: aspiration syndromes, congenital heart defects with pulmonary hypertension, congenital diaphragmatic hernia, neonatal idiopathic pulmonary arterial hypertension, congenital pneumonia, neonatal sepsis, persistent pulmonary hypertension of the newborn, respiratory distress syndrome, transient tachypnea of the newborn, and transposition of great arteries.

Conclusions

Meconium aspiration syndrome (MAS) is a severe, rapidly progressive neonatal lung disease with high complication rates and risk of fatal outcomes. The application of modern informational, clinical, and laboratory-instrumental diagnostic methods from the period of initial risk development in utero is crucial for early personalized MAS diagnosis.

The MAS diagnostic process requires detailed interpretation of results obtained through contemporary diagnostic tools detecting changes in affected neonates, which in turn demands neonatologists continuously enhance their knowledge and skills in both individual and team-based practice.

A multidisciplinary approach to early diagnosis will help prevent long-term consequences that directly impact the developmental prognosis and health of infants with MAS.

Thus, the diagnostic components of MAS–personalization, timeliness, completeness, and systematic application of modern diagnostic tools throughout all perinatal stages–form the foundation for effective management of this complex neonatal condition.

Future research directions. Early, dynamic, and continuous monitoring of infants born through meconium-stained amniotic fluid from the first seconds of life, with determination of diffuse pneumonitis markers (TNF-α, interleukin (IL)-1β, IL-6, IL-8, IL-13) during the «golden hour», is essential to predict MAS development, its severity grade, and inflammatory process intensity. Correlating these findings with respiratory disturbance severity will augment current diagnostic criteria in routine neonatal practice and facilitate personalized management decisions for this condition specific to the perinatal period.

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ЗНАЧЕННЯ ДІАГНОСТИЧНИХ СКЛАДОВИХ СИНДРОМУ АСПІРАЦІЇ МЕКОНІЮ У МЕНЕДЖМЕНТІ НОВОНАРОДЖЕНИХ

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Резюме.

Ефективна і своєчасна діагностика – обов'язковий елемент профілактики та лікування усіх захворювань періоду новонародженості. Вона ж і відіграє роль диригента у злагодженому процесі ведення новонародженого із синдромом аспірації меконію (САМ). Це серйозне захворювання, що може починатися ще до народження дитини, швидко прогресувати та спричиняти ризики розвитку небезпечних наслідків, які можуть проявлятися як одразу після народження, так і у старшому віці.

Діагностичний процес САМ вимагає від лікаря-акушера, неонатолога глибоких знань та навичок в усіх галузях медичної науки, від розуміння патологічних процесів на біохімічному, клітинному рівнях до цілісного розуміння функціонування органів, систем та організму в цілому, з урахуванням гестаційного віку дитини на час народження. Чітку стратегію і тактику надання допомоги щойно народженій дитині крізь меконіальні води допомагає прийняти використання результатів супроводу вагітності та ультразвукового дослідження стану внутрішньоутробної дитини, плаценти, характеристик навколоплідних вод та результатів кардіотокографії зокрема.

Синдром аспірації меконію – це стан, який виникає внаслідок аспірації меконіальних вод до, під час або одразу після народження. Меконій – агресивне середовище, яке швидко пошкоджує незрілу, надчутливу легеневу тканину та запускає каскад змін у всьому організмі дитини. Вивільнення цитокінів, включаючи фактор некрозу пухлин-альфа (ФНП-α), інтерлейкін (IL)-1β, IL-6, IL-8, IL-13), що ініціюють дифузний пневмоніт, подразнюючи дихальні шляхи та паренхіму. Розвиток пневмоніту може статися протягом кількох годин після аспірації внаслідок впливу ферментів, жовчних солей та вільних жирних кислот меконію. Визначення ФНП-α, інтерлейкінів (IL)-1β, IL-6, IL-8, IL-13 діагностично цінне для пневмоніту при САМ. Безальтернативне значення у діагностиці ступеня тяжкості САМ має динамічна оцінка кислотно-лужного стану та газового складу крові, оскільки метаболічний ацидоз, спричинений перинатальним стресом, поєднується з респіраторним ацидозом, а паренхіматозне захворювання – зі стійкою легеневою гіпертензією новонароджених (ПІПЛГ).

Порушення гомеостатичного балансу при САМ – гіпоксія і метаболічний стрес може призводити до змін рівня електролітів. Натрій, калій і кальцій є критичними для нормальної роботи серця, регуляції тонусу судин, нервово-м'язової провідності. Понижений рівень натрію (гіпонатріємія) може бути першим маркером синдрому невідповідної секреції антидіуретичного гормону (НСАДГ), який часто виникає при тяжкому САМ через стресову гіпоксію або пошкодження легень. При НСАДГ спостерігається гіпонатріємія, через затримку води в організмі, ризик набряку мозку і судом. При гострій нирковій недостатності змінюються рівні калію (часто гіперкаліємія – дуже небезпечна), можливе наростання азотистих шлаків, порушення кислотно-лужного балансу. Порушення рівня калію (гіпокаліємія або гіперкаліємія) може спричинити небезпечні аритмії. Пошкодження нирок або тканинна гіпоксія при САМ може призводити до вивільнення калію у кров. Гіпокальціємія у новонароджених може підсилювати серцево-судинну нестабільність та дихальні розлади, спричинені САМ. Поряд із локальним впливом меконію на слизові дихальних шляхів і паренхіму легень важливу роль у розвитку синдрому аспірації меконію відіграють гіпоксія та інфекція. По-перше, гострий дефіцит кисню в плода призводить до стресової відповіді: під дією гіпоксії розслаблюється анальний сфінктер, стимулюється перистальтика кишківника й відбувається викид меконію у навколоплідні води. Одночасно гіпоксія може викликати гаспи (схожі на задуху) дихальні спазми плода, що сприяють потраплянню меконію в дихальні шляхи ще до народження. У результаті ранньої аспірації формуються три головні механізми пошкодження легень: обструкція дихальних шляхів, хімічний пневмоніт із запаленням і порушення сурфактантної функції, що лише посилює гіпоксичний стан новонародженого. Окремий фактор ризику розвитку САМ створює інфекція, особливо внутрішньоматкова (хоріоамніоніт), наявність якої значно збільшує перелік складових діагностичного процесу.

Рутинним діагностичним інструментом, що не втрачає своєї діагностичної актуальності у новонароджених, залишається розгорнутий загальний аналіз крові. Показники загального аналізу крові, як ранні маркери патологічних змін, що відбуваються в організмі новонародженого, допоможуть відповідальному лікарю у розумінні глибини змін, що відбуваються у дитини з синдромом аспірації меконію. Детальної лікарської інтерпретації, відображеної у первинній медичній документації, заслуговують показники загального аналізу крові, а саме: кількість еритроцитів, тромбоцитів, величина гематокриту, кількість нейтрофілів та інших гранулоцитів, оскільки перераховані показники свідчать про рівень забезпечення здатності крові переносити кисень, наявність ризику виникнення неонатальної кровотечі, інфекції та перенесеної хронічної гіпоксії.

Ознаки впливу гіпоксії на головний мозок може виявити і неврологічне обстеження, що буде максимально інформативним після стабілізації стану, візуальні дослідження мозку (магнітно-резонансна томографія [МРТ], комп'ютерна томографія [КТ], ультрасонографія черепа) за наявності показань.

Ключове місце в діагностиці САМ займає рентгенографія грудної клітки, що необхідна для підтвердження діагнозу САМ та визначення ступеня тяжкості, виявлення ділянок ателектазу та синдромів витоку повітря; перевірки правильного розташування ендотрахеальної трубки, пупкових катетерів. Ультразвукова діагностика (УЗД) легень набула в неонатології, не меншої важли-

вості, ніж рентгенографія органів грудної клітки. Ехокардіографія потрібна для оцінки структури серця та серцевої функції, а також для визначення тяжкості легеневої гіпертензії та шунтування справа наліво, бажано це обстеження дитині з дихальними розладами, пов'язаними з САМ виконати в першу «золоту» годину життя.

У статті акцентована увага на роль системного підходу до діагностики синдрому аспірації меконію у профілактиці, лікуванні та попередженні віддалених наслідків САМ.

Ключові слова: синдром аспірації меконію; меконіальні навколоплідні води; діагностика САМ.

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