# Meconium aspiration syndrome

This article has been translated from WikiSkripta; ready for the **editor's review**.

**Meconium aspiration syndrome** (MAS) is defined as early-onset respiratory distress in a baby born with meconium-clouded amniotic fluid. MAS is accompanied by poor pulmonary compliance, hypoxemia, and a typical radiographic picture.[1]

Intrauterine stress can cause the departure of pitch (meconium) into the amniotic fluid. The meconium-cloudy amniotic fluid may subsequently be inhaled by the fetus during gasping (gasping breathing) or deep breathing during hypoxia or hypercapnia. The presence of meconium in the trachea can cause airway obstruction. Lung tissue responds to the presence of pitch by inflammation, degradation of surfactant and persistent pulmonary arterial hypertension. respiratory failure accompanied by hypoxaemia develops. MAS often requires artificial pulmonary ventilation, surfactant administration, nitric oxide administration, and sometimes the indication criteria for extracorporeal membrane oxygenation (ECMO) may be met.[1][2]

MAS affects only about 5% of babies with meconium-clouded amniotic fluid. It primarily affects preterm and term newborns. Smolka departure in asphyctic fetuses before 34 weeks is uncommon (biliary reflux in intestinal obstruction is a more common cause).[1]

## Pathophysiology

After the passage of the pitch into the amniotic fluid, aspiration of pitch-cloudy amniotic fluid may occur before birth or at birth. Before birth, the fetal lungs are filled with viscous fluid, which prevents the development of meconium aspiration syndrome. After birth, the fluid from the lungs is absorbed and the meconium in the airways progresses more distally.

Meconium can cause **airway obstruction** and atelectasis of the relevant part of the lung. If only a partial airway obstruction occurs, the ball-valve phenomenon occurs and air trapping ('*air trapping*) and alveolar hyperexpansion occur in the relevant part of the lung. There is an increased risk of *air leak*.

When meconium comes into contact with lung tissue, *chemical pneumonia* develops. Pulmonary oedema and narrowing of the small airways occur. Mekonium inactivates surfactant in the alveoli. Partial airway obstruction and atelectasis result in uneven ventilation, carbon dioxide retention and hypoxaemia.

One third of children with MAS develop *persistent pulmonary hypertension* (PPHN) caused by the release of proinflammatory cytokines and vasoactive substances during meconium aspiration. Pulmonary vascular resistance is further increased by alveolar hypoxia, acidosis and pulmonary hyperinflation. Increased pulmonary resistance can lead to right atrial shunts and pennate duct (PDA), exacerbating hypoxemia.[1]

Aspiration of meconium causes **surfactant dysfunction**.[3]

The presence of meconium in amniotic fluid reduces antibacterial properties and thus increases the risk of bacterial infection. In addition, meconium irritates the skin and increases the incidence of *erythema toxicum*.[3]

In newborns with MAS, green discoloration of urine may occur in the first 24 hours after birth due to absorption of meconium pigments by the lungs and their subsequent excretion in the urine.[3]

### Risk factors

Risk factors for meconium excretion into the amniotic fluid and subsequent aspiration include:

- postterm pregnancy;
- pre-eclampsia;
- maternal hypertension;
- maternal diabetes mellitus;
- abnormal fetal heart rate;
- IUGR;
- abnormal biophysical profile;
- oligohydramnios;
- heavy smoking; * chronic respiratory or cardiovascular disease of the mother;
- low Apgar score at minute 5;
- fetal distress;
- home birth.[1]

## Clinical picture

Clinical manifestations depend on the severity of the hypoxic insult and the amount and viscosity of the aspirated meconium. Typically, they are transfused hypotrophic neonates with long nails and peeling yellow or green colored skin, umbilicus, nails, and calluses. The umbilical cord becomes discoloured after 15-60 minutes of contact with

meconium, the nails become discoloured after 4-6 hours and the cerebellum (*vernix caseosa*) after approximately 12 hours.

Respiratory distress develops immediately after birth or during postnatal adaptation. With severe asphyxia, they are hypotonic and have respiratory depression after birth. Aspiration of large amounts of meconium can cause obstruction of the large airways - the newborn is cyanotic, not breathing or gasping for breath. Aspiration of meconium into the small airways is manifested by respiratory distress - tachypnea, alary coaction, intercostal retraction and cyanosis.[1]

# Differential diagnosis

- Aspiration;
- Congenital diaphragmatic hernia;
- Pneumonia;
- Idiopathic pulmonary hypertension;
- Persistent pulmonary hypertension of the newborn;
- Sepsis;
- Transient tachypnea of the newborn;
- Transposition of the great arteries.[3]

# Examination

- blood gas examination, ABR - hypoxemia, respiratory acidosis, in perinatal asphyxia mixed respiratory and metabolic acidosis;
- monitoring of serum electrolytes (Na, K, Ca) - SIADH and acute renal failure may develop as a result of perinatal asphyxia;
- blood count and differential (to exclude anemia, polycythemia, perinatal bacterial infection);
- X-ray of the lungs - lung hyperinflation, diaphragm obliteration, irregular spotty pulmonary infiltrates, pneumothorax, pneumomediastinum;
- echo - assessment of the severity of pulmonary hypertension, exclusion of congenital heart disease.[1][3]

# Treatment

- Thermoneutral environment (to minimize oxygen consumption).
- Minimal manipulation (to prevent restlessness, which leads to increased right shunts and exacerbation of hypoxia and acidosis).
- As needed sedation.
- Adequate ventilation support to maintain oxygenation (conventional mechanical ventilation, high-frequency oscillation, jet ventilation, ECMO), monitoring oxygenation with pulse oximetry.
- Administration of surfactant to replace inactivated surfactant and to remove meconium (surfactant acts as a detergent).
- Nitric oxide inhalation to vasodilate the pulmonary vasculature in PPHN. Possibly phosphodiesterase inhibitors (milrinone, sildenafil).
- Umbilical artery cannulation to monitor blood pressure and arterial blood gases.
- Maintaining adequate systemic blood pressure (maintaining higher systemic than pulmonary pressure to reduce right shunt flows).
- Parenteral nutrition to maintain normoglycemia, moderate fluid restriction.
- Broad-spectrum antibiotics when pneumonia is suspected by X-ray.[3][1]

# Links

## Related articles

- Aspiration pneumopathy

## References


1. GOMELLA, TL, et al. *Neonatology : Management, Procedures, On-Call Problems, Diseases, and Drugs.* 6. vydání. Lange, 2009. s. 574-579. ISBN 978-0-07-154431-3.
2. DORT, Jiří, et al. *Neonatologie : vybrané kapitoly pro studenty LF.* 1. vydání. Praha : Karolinum, 2005. ISBN 80-246-0790-5.
3. CLARK, MB, et al. *Meconium Aspiration Syndrome* [online]. Medscape, ©2012. [cit. 2013-04-09]. <https://emedicine.medscape.com/article/974110-overview>.