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# ACUTE ASTHMA ATTACK IN CHILDREN

# AKUTNI ASTMATIČNI NAPAD KOD DECE

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***Summary:*** INTRODUCTION. Asthma is a chronic inflammatory disease of the respiratory tract. In young children and infants the symptoms are caused by mechanical obstruction of the bronchial lumen with secretions and/or mucosal edema. Infant asthma can be defined as the presence of typical symptoms (wheezing, difficulty breathing, tachypnea) in the absence of other conditions that can lead to obstruction. Asthma in children is more related to heredity and the presence of allergies. An acute asthma attack is an episode of progressive worsening of choking, coughing, and/or wheezing.
AIM of this paper is to gain insight into the basic principles of asthma attack therapy in children. The target group is all the profiles of medical workers who come into direct contact with children with asthma.
METHODOLOGY. Guidelines from the GINA (Global Initiative for Asthma), the Guide to Good Clinical Practice, as well as guidelines from the available literature.
RESULTS show that it is best to use a step-by-step approach to a sick child by assessing the risk at the reception, considering the symptoms of the disease, clinical signs, as well as objective parameters, and then approaching urgent treatment. The following symptoms should be considered in the clinical examination: prolonged expiration, impaired breathing, early inspiration cracks, wheezing, respiratory frequency, heart rate. In the differential diagnosis: epiglottitis, acute bronchitis, airway obstruction by a foreign body, pulmonary edema, pneumonia. In therapy, leading role have beta 2 agonists, oxygen therapy and steroids. Not helpful in an acute asthma attack: antibiotics, expectorants, adrenaline parenterally or by inhalation, sedatives, antitussives, antihistamines.
CONCLUSION. In the approach of a child with an acute attack, we rely on a precise anamnesis, clinical status and objective measurement of lung function (SatO2 and PEF) to assess the severity of the attack. The most useful parameters in monitoring are the clinical appearance of the child and gas analysis. The goal of therapy is to stop the attack and finally educate the child and parents about the adequate response in case of further deterioration.
***Key words:*** acute asthma attack, children, neonates

## INTRODUCTION

Asthma is a chronic inflammatory disease of the respiratory tract. In atopics, this inflammation is the cause of repeated episodes of wheezing, choking and coughing, during the day and at night and after physical fatigue. All these symptoms are a consequence of the increased response of the airways to various stimuli. As a result, diffuse airway obstruction occurs, which is of varying degrees and is lost either spontaneously or under the influence of bronchodilators and/or steroids. [1,2]

This definition is acceptable in adults and older children but is not appropriate for infants and young children. Among them, the inflammatory process is sometimes minimal or completely absent, and the symptoms are caused by mechanical obstruction of the bronchial lumen with secretions and/or mucosal edema. The response to bronchodilators (reversibility of obstruction) may be only partial or completely absent. Therefore, infant asthma (so-called infantile asthma) can be defined as the presence of typical symptoms (wheezing, difficulty breathing, tachypnea) in the absence of other conditions that can lead to obstruction, especially in a child with signs of atopic status, the presence of atopic dermatitis, family anamnesis, high total IgE antibodies in umbilical cord blood, positive skin tests for inhalation and/or nutritional allergens, etc). [1,3]

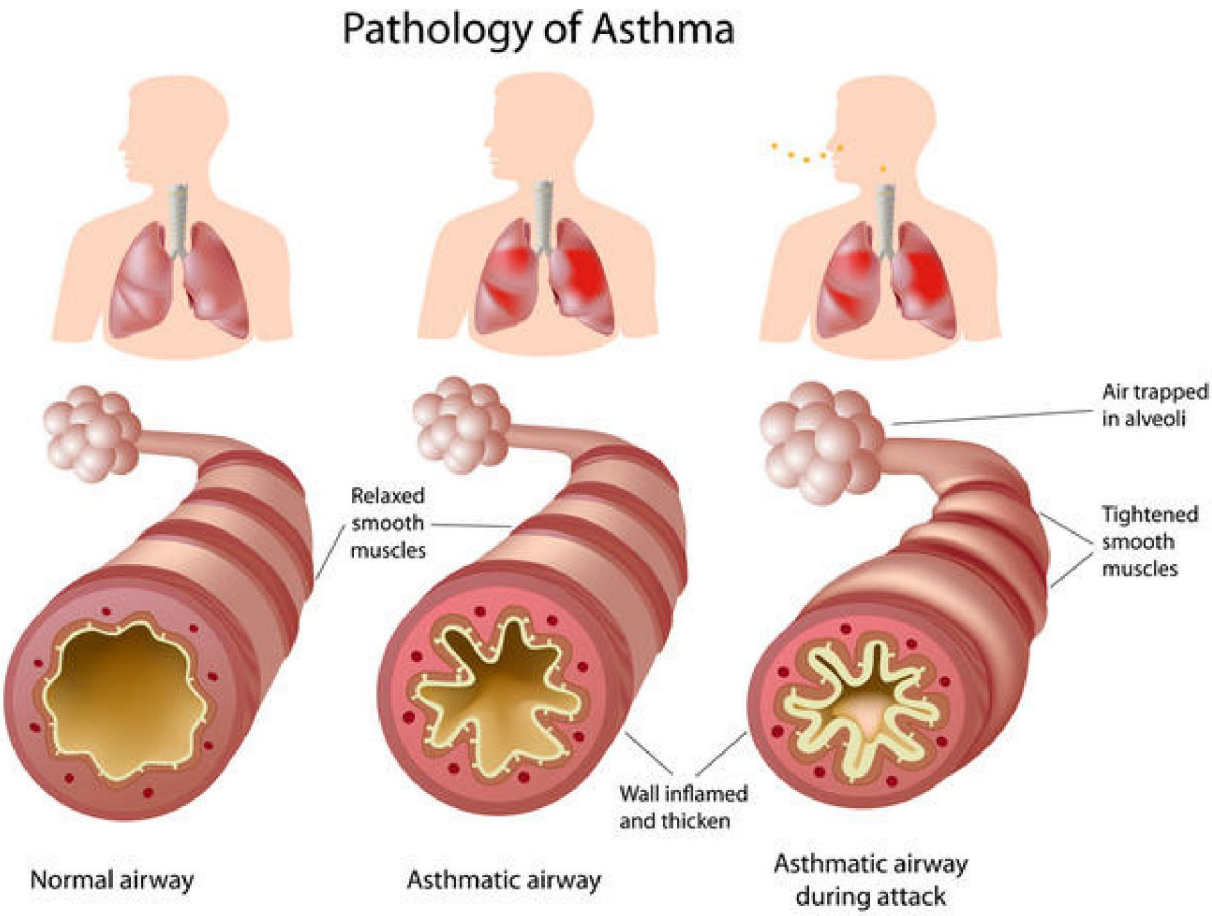


Figure 1. Pathology of asthma (Adapted and taken from Asthma NCLEX review. Photo credit: Alila Medical media).

Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 1

A child's lungs and their immune system are developing and this is the main reason that separates adult asthma from children's asthma. Asthma in children is more related to heredity and the presence of allergies. The higher the allergic predisposition, the earlier the disease will manifest itself and the more severe the course. Thus, 80% of children with asthma have the onset of symptoms in the first five years of life. Asthma in children comes in several forms. The most difficult is the so-called persistent asthma, which occurs in the first three years of life and is associated with high atopic (allergic) status. In them, obstructions are repeated very often, and the disease does not show a tendency towards remissions. The main pathological change is allergic inflammation, which is dominated by two types of cells, activated Th2 helper lymphocytes and eosinophils. [2] In allergic asthma, T lymphocytes are activated by constant contact with environmental allergens (the most important are mites), and occasionally the reaction is intensified by a certain type of viral infection (rhinoviruses, coronaviruses, parainfluenza, etc.). Helping factors in the development of the reaction are smoking and air pollution, a short period of breastfeeding, and perhaps vaccinations. Factors that suppress the development of allergic inflammation in asthma are: recurrence of infection with certain respiratory viruses, intestinal infections and infestations. Therefore, poor socioeconomic status protects a child from developing asthma and allergies. Regardless of how the activation of T helper lymphocytes occurred, it secretes increased amounts of its products, the key interleukin-5 (IL5) which brings and activates eosinophils to the reaction site. They contain a large number of cytotoxic proteins, which destroy the bronchial epithelium and cause an inflammatory reaction. Pathologically, inflammation in asthma is described as chronic eosinophilic desquamative bronchitis. After several years of the disease, inflammation becomes independent of environmental factors, because the cells that participate in it acquire the ability to activate themselves, as well as to activate the cells around them. Chronic inflammation in the bronchial wall has two pathological consequences. The bronchi thicken (up to 2.5 times), and these changes lead to a change in their reaction. They become extremely sensitive, narrowing easily and excessively (this tendency is described as bronchial hyperreactivity) under the action of stimuli that have no effect in healthy individuals. These stimuli are very heterogeneous (physical exertion, smoke, viral infections, psychological stress, etc.). This excessive tendency to narrow also contributes to the markedly increased contractility of the smooth muscles of the bronchi (which is hypertrophic in asthma). [2-5]

The second group of asthmatics in childhood are those in whom the disease occurs after the third year of life, who have allergies (but not as pronounced as the first group) and whose disease has a variable course. Most of them belong to relatively mild forms, which gradually subside, and in about half, the disease completely disappears at puberty. This group has significantly less pronounced inflammation in the bronchial wall, and the degree of activation of Th2 cells is significantly lower. [1,2]

The third group are children who have repeated episodes of wheezing, without allergic sensitization. This type of asthma is often called infectious asthma, due to the importance of viral infections in the development of obstructions. Since virus infections are occasional, and susceptibility to them decreases with age, these children markedly improve towards puberty (especially obstructions become thinner after the age of six). Most people completely lose their asthma symptoms after puberty. [1-3]

The fourth group are children with true non-allergic asthma, similar to that in adults. This form is very difficult, but fortunately also very rare. Such children usually develop bronchiectasis by puberty, and asthma is very difficult to control with medication.

## PATHOPHYSIOLOGY OF ACUTE ATTACK

An acute asthma attack is an episode of progressive worsening of choking, coughing,

and/or wheezing. Depending on the mechanism of occurrence and severity of asthma (mucus secretion, mucosal edema at one end of the spectrum - contraction of smooth muscles at the other), it can occur quickly or gradually, pass spontaneously or progress to respiratory failure. Although extremely frightening for the child and parents, complications are rare, and the fatal outcome is almost always the result of a mistake in treatment (late recognition of symptoms, inadequate application of therapy, especially delay in the use of steroids). Since many seizures can be recognized in the prodromal stage (in 85% of cases there are signs of rhinovirus infection, most often runny nose, sneezing, itchy nose, insomnia, nervousness, etc.), every patient must try to use beta-2 agonist at this stage of the disease. The results of the study showed that 70% of attacks can be prevented if a beta-2 agonist is used in the prodromal phase. They should be given for six hours in the usual dose (2 breaths of MDR) for two days. If the obstruction does not occur during those two days, they should be stopped. The use of steroids in the prodromal phase (neither systemic nor inhaled) is irrelevant. [1-5]

Several factors contribute to the obstruction of breathing that is characteristic of asthma. A mild asthma attack is characterized by mild bronchoconstriction and a less inflammatory reaction. With increasing severity of the attack, mucosal edema occurs, followed by submucosal edema with enlarged capillaries and hypertrophic glands. The inflammatory process is accompanied by the presence of eosinophils and later neutrophils in the walls of the airways with hypersecretion of mucus. This process leads to obstruction of the distal parts of the airways, primarily the bronchioles. Such lungs are in hyperinflation with air and cannot collapse. With increasing obstruction, bronchoconstriction also increases and leads to danger to the patient's life due to complete obstruction of breathing. [1,2] Physiologically, as a consequence of significant obstruction, ventilation increases, as well as a significant difference between ventilation and perfusion, which leads to hypoxemia followed eventually by both respiratory and metabolic acidosis. Hypoxemia seen in asthmatics is a consequence of obstruction of the small airways. As this process progresses, the patient becomes tired, and pCO2, which was initially reduced due to hyperventilation, now begins to grow. Hypocapnia and mild respiratory alkalosis can even be expected at the beginning. Normal levels of pCO2 in asthmatics are a sign of initial respiratory insufficiency and a clear indication for hospitalization and more aggressive therapy. [1-5]

## AIM

The aim of this paper is to gain insight into the basic principles of asthma attack therapy in children, as well as to acquaint the general medical public with it. The target group is all the profiles of medical workers who come into direct contact with children with asthma, who should be educated for the right help for such children.

## METHODOLOGY

Guidelines from the GINA (Global Initiative for Asthma), the Guide to Good Clinical Practice, as well as guidelines from the available literature dealing with the problem of asthma attacks were used.

## RESULTS

The results show that it is best to use a step-by-step approach to a sick child by assessing the risk at the reception, considering the symptoms of the disease, clinical signs, as well as objective parameters, and then approaching urgent treatment. [2-4]

## CLINICAL PRESENTATION

History (occurrence of prodromal symptoms, existence of signs of viral infection of the upper respiratory tract before the attack, applied therapy and response to that therapy, when was the last deterioration, severity of previous attacks and their treatment, previous

Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 1

hospitalizations, current preventive therapy and its dosage). [1,2,6,7]
Clinical examination (dyspnoea, speech, behavior, signs, respiratory rate, use of auxiliary respiratory muscles, whistling, pulse rate). [1,2,8]
The following symptoms should be considered in particular in the clinical examination:
- Prolonged expiration
- Impaired breathing
- Early Inspiration cracks
- Wheezing
- Respiratory frequency (Table 1.)
- Heart rate (Table 2.)

| Respiratory rate - awake | |
|---|---|
| **Age** | **Normal rate** |
| **< 2 months** | < 60/min |
| **2-12 month** | < 50/min |
| **1-5 years** | < 40/min |
| **6-8 years** | < 30/min |
| **Older** | <25/min |

Table 1. Respiratory rate in chilndren

| Heart rate | |
|---|---|
| **Age** | **Normal rate** |
| **Infants (2-12month)** | <160/min |
| **1-2 years** | <120/min |
| **3-8 years** | <110/min |
| **Older** | 50-100(60-90)/min |

Table 2. Heart rate in children

Tests (oxygen saturation with hemoglobin, monitoring of PEFR and / or spirometry, acid-base status). [6-9]
The following conditions should be considered in the differential diagnosis:
- Epiglottitis
- Acute bronchitis
- Airway obstruction by a foreign body
- Pulmonary edema
- Pneumonia

Taking into account all the data, the severity of the asthma attack is estimated according to certain parameters listed in the table 3. The patient being admitted due to an acute asthma attack or status is most often dyspnoeic, dysphasic, cyanotic, in acid-base status with normal pCO2 (initially pCO2 is reduced due to hyperventilation as a compensatory mechanism), with acidosis and requires urgent therapy. [1-9]
The most useful parameters in monitoring patients with acute severe seizures are the clinical appearance of the child and monitoring of gas analyzes. Unnecessary analyzes are spirometry or PEFR, chest X-ray (Figure 2) (worsening obstruction, exhausting the patient), blood count (beta-2 agonists increase the number of leukocytes, steroids increase the number of polymorphonuclears), lung scan (only if the child's condition does not improve). Atelectasis or pneumothorax or pneumomediastinum may be noted. [7-9]

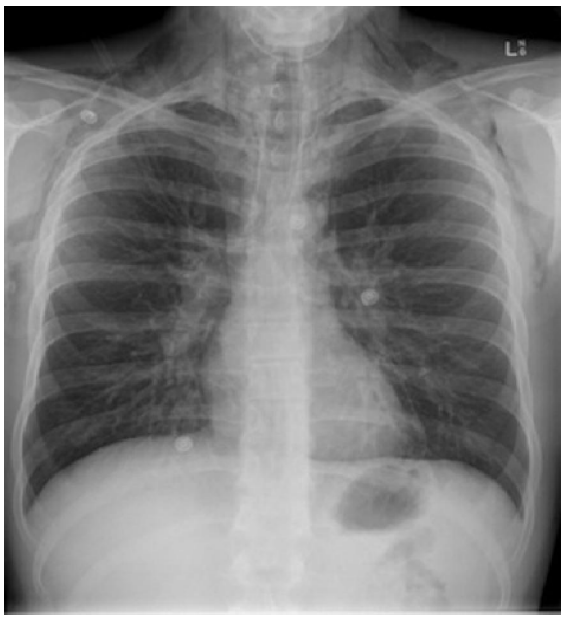

Figure 2. Chest X-ray in children with asthma attack.

## THERAPY

The goal of treating an acute attack is as follows:
- Suppression of obstruction
- Suppression of inflammation
- Correction of hypoxemia
- Correction of lung function
- Relapse prevention

If there is a possibility of starting therapy at home, it should be started with short-acting β2 agonists which, if it is a mild attack, show effect even after 4 hours of application, and in

that case the therapy continues with the same. If symptoms such as agitation, moaning, rapid breathing, palpitations, raised shoulders, paleness or bluishness, work of the auxiliary respiratory muscles appear, it is a worsening of the acute attack. If the attack is judged to be severe and the child's condition is poor, the child should be referred immediately to hospital (first given steroids, β2 agonists and aminophylline, and oxygen and fluid infusion during transport. [1,2,8-10]

If the child is not for hospitalization, it is necessary to take the initial administration of salbutamol - for children under 5 years of age 2 doses of MDI across the chamber or 0.05 mg / kg of salbutamol to a maximum of 1.25 mg (0.25 ml) by nebulization after 20 minutes), ie for children over 5 years 2 doses of MDI or 2.5 mg (0.5 ml) of nebulized salbutamol. [1,2,5,8-10] If the response is good (no wheezing and difficulty breathing, the response to beta-2 agonists is present even after 4 hours, and PEF is above 80% predicted) it is a mild attack, so therapy is continued with beta-2 agonists for 4 hours in the first 12 hours, and then it can be diluted to 6 hours (do not give in three daily doses, because the effect of salbutamol lasts about 6 hours). If children are not on prophylactic therapy with inhaled steroids, double or triple the steroid dose during the period of salbutamol administration. If the response is incomplete, it is a moderately severe attack (maintenance is maintained, difficulty breathing is still present after 4 hours of β2 agonist administration, and PEF 50-80% of predicted), a single dose of β2 agonist (2 breaths of MDI or 1.25 mg nebulized for children under 5 years or 2.5 mg for older children) is given every 2 hours to 3 inhalations in total. [7-10] At the same time start with prednisolone per os, 1-2 mg / kg for 5-10 days without gradual discontinuation. If after 3 inhalations there is no improvement, the child should be sent to the hospital and treated as an acute severe attack. If the condition improves, continue β2 agonists for 4 hours in the same doses for a total of 24-36 h, then dilute the dose to 6 h. In hospital conditions, initial therapy consists of administering oxygen through a mask or nasal cannula (approximately 2-4 l / min), inhalation with salbutamol (2.5 mg) with the addition of ipratropium bromide (0.5 ml solution) via a nebulizer and steroids 0.5 - 1 mg / kg in one daily dose per os, and if the child vomits methyl-prednisolone 0.5 - 1 mg / kg i.v. every 6 hours and i.v. fluid replacement. In case of a good reaction (improvement of obstruction, increase in PEFR, SatO2> 92%) continue with inhalations of salbutamol and ipratropium (or fenoterol with ipratropium) by nebulization for 2-4 hours, methyl-prednisolone for 6 hours i.v. the first 24-36h, then per os 1-2 mg / kg in the morning dose more 5 days. In case of poor response, administer aminophylline i.v. 3 mg / kg for 20 min for a child under 6 months of age, 5 mg / kg for an older child and 7 mg / kg for adolescents. If there is no response consider differential diagnoses (lung scan to exclude pneumonia, pneumothorax, atelectasis, etc.), admit the child into ICU in case of persistent hypoxia or hypercapnia, respiratory muscle fatigue and impaired consciousness and prepare him for intubation and mechanical ventilation. If mechanical ventilation is indicated, do not insist on complete normalization of blood gases, but allow a certain degree of hypercapnia (to avoid barotrauma). Insist that mechanical ventilation lasts as short as possible. Ventilation with the highest possible concentration of oxygen should be started immediately, over a mask, with an always prepared set for endotracheal intubation. [6,8,9] You should also have an open I.V. line and monitor the patient. If intubation is performed, the child is ventilated with 100% oxygen. Be sure to auscultate above the epigastrium and above the lungs to confirm the presence of tubes in the bronchi. In a very severe attack, a small amount of inhaled drug reaches the alveoli, so that an aerosol solution can be given directly through the endotracheal tube (0.25 ml salbutamol dissolved in 3 ml NaCl). Salbutamol can also be given i.v. 0.2 μg / kg and increase by 0.1 μg / kg depending on the response. An alternative to this therapy is adrenaline given by S.C. 0.3-0.5 ml 1: 1000 and which can be repeated every 15 minutes. If cardiac arrest occurs, give 5-10 ml 1: 10000 I.V. Such patients are in acidosis

Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 1

and bicarbonates should be included in the therapy if the pH is <7.2. These bicarbonates have antagonistic effects on β2 agonists. Occasionally, the child may not respond to all previous measures of therapy, so in that case, resort to inhalation anesthesia, usually halothane. However, halothane can lead to myocardial depression and arrhythmias, as well as worsen hypoxemia. But although halothane has a negative inotropic effect and arrhythmogenic potential, this is generally not observed in patients. [9,10]

| Estimated attack severity **Parameter** | **Mild attack** | **Intermediate** | **Heavy attack** | **Life-threatening attack** |
|---|---|---|---|---|
| **Dyspnoea** | Present only when walking | When speaking or laughing | Present at peace | Expressive, cyanosis |
| **Body position** | Can lie on his/her back | Prefers sitting | Ortopnoeic (a sitting position with your hands resting on solid objects) | Due to weakness, lying on a pillow |
| **Sleep** | Sleeping, waking up, coughing | Often wakes up, has bad dreams | Not sleeping, sitting in bed | Blurred sensory |
| **Use of auxiliary respiratory muscles** | Absent | Present, there is indentation of intercostal spaces | Pronounced, there are contractions of the sternocleido-mastoideus | Almost absent (muscle fatigue) Paradoxical movements of the thoraco-abdominal muscles |
| **Wheezing** | Present at the end of the expiration | Throughout the expiration | Inspirational-expiratory | Slightly audible, breathing also impaired |
| **Respiratory rate** | Moderately elevated | Tachypnea | Distinct tachypnea | Bradypnea |
| **Speech** | Unchanged | Tired of speaking (3-5 words in a row) | Speaks only short words | Do not talk |
| **PEFR** | Above 80% of predicted values | Between 50 and 80% for age | Below 50% for age | Below 30% for age |
| **Paradoxical pulse** | Below 10 mmHg | 10-20 mmHg | Over 20 mmHg | |
| **Tachycardia** | Moderate | Expressed | Very high | |
| **Saturation O2** | Over 95% | 91-95% | Below 91% | |

Table 3. Estimated attack severity according to parameters (Taken and adapted from: Nacionalni vodič kliničke prakse za dijagnostikovanje, lečenje i praćenje astme u dečjem uzrastu, Septembar 2002).

| **Admission risk assessment** | | |
|---|---|---|
| **Anamnesis** | - Data on current deterioration<br>- Frequency and severity of day-night symptoms and activity limitations<br>- Frequency of bronchodilator use<br>- Current drugs and their doses<br>- Possible triggers<br>- Previous use of systemic steroids, ambulance visits, hospitalizations, intubations, life-threatening conditions | |
| **Clinical assessment** | - Vital signs, dyspnea, use of auxiliary respiratory muscles, anxiety, changes in mental status<br>- Pulse oximetry<br>- Pulmonary function | |
| **Risk factors** | | |
| **Therapy** | | |
| **Medications** | **Mechanism** | **Side effects, caution** |
| Oxygen<br>(mask, nasal catheter) | Treats hypoxia | Monitor with pulse oximeter<br>Saturation O2> 92%<br>Cardiorespiratory monitoring |
| Inhaled short-acting β2 agonists (Albuterol) | Bronchodilator | Possible vasodilation of pulmonary blood vessels, ventilation / perfusion disorder, hypoxemia<br>Palpitations, tachycardia, arrhythmia |
| Systemic corticosteroids (Prednisolone) | Anti-inflammatory | In exposed smallpox give IG, risk of complications in Herpes simplex and TB<br>Dosing at 8 in the morning reduces adrenal suppression<br>Long-term use = Peptic ulcer, Cushing's Sy, suppression of response to infection or injury, osteoporosis, adrenal atrophy, metabolic and mental disorders, cataracts |
| Anticholinergics<br>(Ipratropium) | Mucolytic / Bronchodilator | Do not give as 1st line of therapy already<br>In combination with β2 agonists |
| Adrenaline | Bronchodilator | Respiratory failure (even after high doses of β2 agonists) |
| Terbutaline<br>(selective β2 agonist) | | Follow complete monitoring at i.v. administration |
| **Discharge risk assessment** | | |
| **Drug stability** | If there is an improvement in symptoms and taking bronchodilators is at 3 hours or more, normal physical findings, PEF> 70% of predicted, Sat O2> 92% in room air | |
| **Home treatment** | Ability to self-medicate and observe as well as adequate response in disease exacerbation | |
| **Asthma education** | | |

Table 4. Admission risk assessment.

Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 1

They are not helpful in an acute asthma attack and should not be given: [2,7-10]

- Antibiotics (unless there is verified pneumonia)
- Expectorants
- Adrenaline parenterally or by inhalation (high risk of heart attack or heart rhythm disorders)
- Sedatives (strictly prohibited)
- Antitussives
- Antihistamines (although not strictly contraindicated, not particularly useful)

| Medication | Single dose | Notice |
|---|---|---|
| Salbutamol | 2 doses of MDI or 1.25 mg in nebulization for children under 5 years - 2.5 mg for older children | Always give oxygen with it (there is a risk of hypoxia) |
| Ipratropium bromide | 0.5 ml for infants, 1 ml for older children | Never give as monotherapy, always with beta-2 agonists |
| Aminophylline | 3 mg / kg for children up to 6 months, 5 mg / kg for the rest | Giving slowly i.v. (pressure drop) |
| Methil-prednisolone | 1 mg / kg in the first dose, then 0.5-1 mg / kg in the next | Maximum dose 20 mg for children up to 5 years, 30 mg for older children |
| Oxygen | 2-4 l/minute | |
| Infusion | Daily needs increased by 30% (in the first hour physiological / glucose in a ratio of 1:1, later 4:1) | Higher concentrations only irritate the mucous membranes Be careful not to overdo the amount of fluid (ADH inadequate secretion syndrome) |

Table 5. Medication doses used in treating acute asthma attack in children (Taken and adapted from: Nacionalni vodič kliničke prakse za dijagnostikovanje, lečenje i praćenje astme u dečjem uzrastu, Septembar 2002).

## CONCLUSION

In the approach of a child with an acute attack, we rely on a precise anamnesis, clinical status and objective measurement of lung function (SatO2 and PEF) to assess the severity of the attack. The most useful parameters in monitoring are the clinical appearance of the child and gas analysis. In hospital conditions, in case of failure of drug therapy, endotracheal intubation, assisted ventilation, and even introduction to general anesthesia are used. The goal of therapy is to stop the attack and finally educate the child and parents about the adequate response in case of further deterioration.

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Southeast European Journal
of Emergency and Disaster Medicine
Vol. VIII, year 2022, No. 1

# AKUTNI ASTMATIČNI NAPAD KOD DECE

**Milan Đorđević**

Služba hitne medicinske pomoći Jagodina

***Sažetak:*** UVOD. Astma je hronična inflamatorna bolest respiratornog trakta. Kod male dece i odojčadi simptomi su uzrokovani mehaničkom opstrukcijom lumena bronhija sa sekretom i/ili edemom sluzokože. Dečja astma se može definisati kao prisustvo tipičnih simptoma (zviždanje, otežano disanje, tahipneja) u odsustvu drugih stanja koja mogu dovesti do opstrukcije, I više povezana sa nasleđem i prisustvom alergija. Akutni napad je epizoda progresivnog pogoršanja gušenja, kašljanja i/ili vizinga.
CILJ ovog rada je da se stekne uvid u osnovne principe terapije napada astme kod dece. Ciljna grupa su svi profili medicinskih radnika koji dolaze u direktan kontakt sa decom obolelom od astme.
METODOLOGIJA. Korišćene su smernice GINA (Globalne inicijative za astmu), Vodič za dobru kliničku praksu, kao i smernice iz dostupne literature.
REZULTATI pokazuju da je najbolje pristupiti bolesnom detetu korak po korak procenom rizika na prijemu, s obzirom na simptome bolesti, kliničke znakove, kao i objektivne parametre, a zatim pristupiti hitnom lečenju. U kliničkom pregledu treba uzeti u obzir: produženo izdisanje, otežano disanje, rane inspirijumske pukote, vizing, učestalost disanja, rad srca. U diferencijalnoj dijagnozi: epiglotitis, akutni bronhitis, opstrukcija disajnih puteva stranim telom, plućni edem, pneumonija. U terapiji vodeću ulogu imaju beta 2 agonisti, terapija kiseonikom i steroidi. Nisu od pomoći kod akutnog napada astme: antibiotici, ekspektoransi, adrenalin parenteralno ili inhalaciono, sedativi, antitusivi, antihistaminici.
ZAKLJUČAK. U pristupu deteta sa akutnim napadom oslanjamo se na preciznu anamnezu, klinički status i objektivno merenje plućne funkcije (SatO2 i PEF) da bismo procenili težinu napada. Najkorisniji parametri u praćenju su klinički izgled deteta i gasne analize. Cilj terapije je zaustaviti napad i konačno edukovati dete i roditelje o adekvatnom odgovoru u slučaju daljeg pogoršanja..
***Ključne reči***: akutni astmatični napad, deca i odojčad

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