Edição 1

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Edição I

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1st Brazilian and International Medical Schools Association Conference (I BIMSAC)

O evento ocorreu dia 11 e 12 de dezembro de 2021, em português e inglês, com tradução simultânea para ambas as línguas.

APRESENTAÇÃO DO EVENTO

O ensino de pneumologia pediátrica é de extrema importância no contexto da formação acadêmica. Nesse sentido, o I BIMSAC visou aproximar os estudantes de medicina dos principais temas dessa área, proporcionando um evento científico para ensino e compartilhamento de experiências.

Assim, dada a necessidade e preocupação constante com a qualificação dos profissionais em formação, esperamos que os futuros médicos presentes no evento, através do contato com diversos especialistas e referências na área, possam estar mais qualificados para atuação profissional. A fim de trazer experiências e conhecimentos provindos de fora do Brasil, trouxemos profissionais que atuavam na Austrália, a fim de prover outras experiências e conhecimentos para os estudantes.

Para isso, contamos com o apoio e colaboração médicos e alunos da Austrália, do grupo ANIMA, da Faculdade de Minas (FAMINAS BH), e do comitê local da FAMINAS BH da "International Federation of Medical Students Association" (IFMSA Brazil).

Ao todo, 8 resumos foram aprovados. Desses, os 3 (três) melhores trabalhos foram, respectivamente: "Adhesion to Asthma Treatment in Pregnant Women During the Third Trimester"; "Challenges in Diagnosis of Community-acquired Pneumonia (CAP) in pediatric patients: a literature review"; e "Case Report: Low Vision Secondary to Severe Complicated Community-acquired pneumonia".

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ANALYSIS OF THE EFFECTS OF OBESITY AND DIABETES MELLITUS ON THE CONTAGION AND DISTURBANCE OF PATIENTS WITH COVID-19

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Introduction: Since its outbreak in Wuhan (China), the new coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome (Sars-CoV-2), has affected millions of people in more than 200 nations worldwide. After the widespread dissemination of the disease, discussions have arisen about the mechanism involved in the infection, as well as the characteristics of the most affected individuals. It has been proven that obesity is a risk factor, both for infection and for possible complications later in the disease. This occurs mainly due to the phenotype of the host, which makes it susceptible to a severe clinical picture with high mortality risk. Accordingly, diabetes mellitus (DM) is associated with severe disease, acute respiratory distress syndrome (ARDS), and increased mortality from COVID-19. **Objectives:** To describe how obesity and diabetes mellitus influence the increased risk of infection from COVID-19 and to detail the mechanisms responsible for causing worsening in these patients. **Methodology:** A literature review was carried out based on a selection of current and relevant data found in scientific articles from the last year (2020), published in the SciELO and PubMed platforms, in English. The search was based on the descriptors COVID-19, obesity and diabetes. Some publications were excluded during the active search process, for not addressing the studied proposal, or for being available as abstracts, theses or dissertations. **Discussion:** It is known that the accumulation of body fat can cause harm to the host. Obese patients have an increased risk of exacerbation in viral respiratory infections, besides having a decreased overall compliance of the respiratory system, related to increased pulmonary blood volume and accumulation of fat around the ribs, diaphragm and abdomen. In addition, the expression of Angiotensin-converting enzyme 2 (ACE-2) (a protein that facilitates entry of SARS-CoV-2 into the body) is higher in visceral adipose tissue than in subcutaneous tissue, causing it to act as a reservoir for the virus that, once stored, puts other parts of the body at risk. With this, obese individuals with extensive visceral adipose tissue develop an exacerbated systemic response of the Angiotensin II and AT1 receptor axis, and may store an expansive viral load and lead to the development of a more severe form of the disease. Furthermore, obese patients have chronically reduced concentrations of adiponectin (anti-inflammatory adipokine) and higher levels of leptin (pro-inflammatory adipokine), resulting in excessive inflammation. The abundance of these pro-inflammatory mediators in adipose tissue leads to dysfunction of innate immunity, making the individual vulnerable to future reinfection. On the other hand, DM is associated with reduced expression of ACE-2. Under physiological conditions, this degrades angiotensin-II, and to some extent angiotensin-I, into smaller peptides, angiotensin (1-7) - responsible for the anti-inflammatory and antioxidant role - and angiotensin (1-9), respectively. Therefore, the imbalance in the ACE2 activation pathways triggers an increase in the inflammatory state, as a consequence of the increase in angiotensin II levels and a decrease in angiotensin 1-7. **Conclusion:** Obesity and diabetes mellitus are pathologies extremely relevant to the pathophysiology of COVID-19, considering that patients with such comorbidities have an increased

risk of infection and poor prognosis. Therefore, it is necessary to understand the increased severity of COVID-19 in these affected patients, and its pathophysiological basis, in order to reaffirm the prevention measures in place in the ongoing pandemic.

Keywords: Obesity; Diabetes Mellitus; COVID-19; Risk Factor; Comorbidity.

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ABSTRACT: ADHESION TO ASTHMA TREATMENT IN PREGNANT WOMENDURING THE THIRD PREGNANCY TRIMESTER

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INTRODUCTION: Asthma is a chronic condition that occurs in 4[1]-13[2]% of pregnant women worldwide, and prevalence in this population has continuously increased[1,3].Inadequate asthma control is frequent in during pregnancy and has several consequences[1-3], such as: intrauterine growth retardation; preeclampsia; premature birth; low weight at birth; and even perinatal death[1]. Thus, proper management isextremely important in this population, and in the third trimester of pregnancy, due to theincreased intrauterine pressure, women are more prone to decompensation[1]. The most recommended treatment consists of fast-acting beta agonists for relief and inhaledcorticosteroids (ICS) for asthma control[2]. However, studies have shown that pregnant women tend to discontinue asthma medication during pregnancy[2], increasing the

number of complications and the occurrence of asthma symptoms during childbirth, with a variable rate of 10-20% in pregnant women with asthma[1].

OBJECTIVES: To investigate the best management of asthma in pregnant women during the third trimester of pregnancy based on findings from the last 3 years.

METHODOLOGY: Search in the pubmed platform with the descriptors “third trimester pregnancy” and “asthma” associated by the Boolean operator “AND”. As inclusion criteria: articles published in the last 3 years. Exclusion criteria: articles that addressed COVID-19, focusing on complications in the newborn. 45 articles were found in the database. After applying the exclusion criteria, 9 articles were selected by title, 5 by abstract and 4 by full reading of the article.

DISCUSSION: In a study with 80 pregnant women with asthma, 56% reported worsening of asthma during pregnancy, with incorrect inhalation technique being observed in 64.4% of them, which data is similar to a North American study with more than 4,000 women. Furthermore, although 78.5% of them thought that asthma could affect the fetus, 44% did not know the effects that it could have on the fetus and 39% thought that ICS would bring more risk to the fetus than asthma[1]. In addition, a meta- analysis showed that the use of ICS decreases in early pregnancy[2] concluding that the prevalence of the medication use in pregnant women with asthma is 41% worldwide[2]. The same study emphasizes that prescriptions of asthma medication did not decrease, only usage. In other words, pregnant women interrupt treatment on their own[2]. It is noteworthy that international guidelines emphasize the importance of preventive therapywith medications for asthma during pregnancy, considering that the risk of severe or chronically undertreated asthma outweighs the risks of medications[1]. In the same study, 42% pregnant women had asthma exacerbation in the peripartum and delivery[1]. Furthermore, 30%, 18.6% and 21% of patients with uncontrolled asthma during pregnancy reported not knowing the correct asthma medications, being afraid of ICSside effects and lack of regular follow-up, respectively, which were the main reasons for poor asthma control[1]. A study from United States of American involving 4,315 pregnant women with asthma revealed that 12.7% of women received rescue corticosteroids during pregnancy, 11.1% had asthma-related emergency room visits, and 6.3% were

hospitalized. Of the 4,315 women in the study, 42% of women had inadequate knowledge about prescribed medications[1]. Regarding the pathophysiology of asthma during pregnancy, it was observed that the dynamics of the markers is attenuated duringpregnancy, as well as the effects of atopic conditions, favouring a healthy environment for the child and fewer symptoms in pregnant women with asthma[4]. There is evidence that B cells favour maternal-fetal tolerance, and the protective state of equilibriumobserved during pregnancy. These cells are important for allergic responses and showed quantitative and qualitative changes from the third trimester of pregnancy to the postpartum period[4].

CONCLUSION: The pathophysiological changes can favour the non-occurrence of symptoms in pregnant women, considering that the body is in a state of compensation, due to the presence of the fetus. Thus, we can speculate that pregnant women'sadherence drops due to the absence of exacerbated symptoms. Lack of adherence was noted in the studies found, being related to lack of information. Therefore, strategies should be adopted to ensure that asthma control has been achieved, and that causes of poor asthma control are addressed in future researchs.

Key words: third trimester pregnancy; asthma; treatment.Referências:

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CHALLENGES IN DIAGNOSIS OF COMMUNITY-ACQUIRED PNEUMONIA (CAP)IN PEDIATRIC PATIENTS: A LITERATURE REVIEW

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INTRODUCTION: Community-acquired pneumonia (CAP) is the leading cause of death in children under 5 years old worldwide. Furthermore, it is also one of the most frequent infectious diseases in pediatric patients. There are many etiologic agents of this disease and, in most cases of CAP in children, the specific pathogen is not identified. The identification of the etiological agent in CAP is important for the implementation of adequate treatment strategies, since the inappropriate use of drugs increases the possibility of microbial resistance, aggravating the clinical condition of the patient. Therefore, due to this difficulty in identifying the specific pathogen responsible for CAP in pediatric patients, the optimal management of the disease in children is not well defined. **OBJECTIVE:** This review focuses on pediatricCAP, specifically highlighting the diagnostic challenges, including the disease severityassessment, often encountered by medical providers. **METHODS:** To ensure a complete literature review, a literature search was conducted using two electronic databases: Medline (PubMed interface) and Biblioteca Virtual de Saúde (BVS). Two keywords were employed, “pneumonia” and “children". A total of 8980 articles were found. The exclusion criteria included the lack of compatibility to the theme chosen, articles published over the last 5 years and articles related to Covid -19. Based on the exclusion criteria and after all the irrelevant records were excluded via checking the title and abstract, articles were reduced to 5. **DISCUSSION:** Pediatric pneumonia guidelines include criteria that were modified from adult criteria and define

pneumonia severity to assist with resource allocation and site-of-care decision-making. At present, mostly clinical aspects are used to predict CAP in children, which includes hypoxemia, altered mental state, age <3-6 months, dyspnea,multilobar infiltrates, and moderate/large pleural effusions. Along with clinical assessment, radiographic and laboratory findings can help to identify the etiology. Current methods for diagnosing and attributing the causes of pneumonia are suboptimal. Current guidelines for radiological diagnosis, recommends chestradiography only in children admitted to hospital with severe symptoms or suspected complications as it has several diagnostic limitations. Emerging evidence suggests that ultrasound is highly sensitive and specific for diagnosing pneumonia in children. In addition, it shows considerable advantages, as it is less affected by movement or crying, less costly than chest radiography and can be performed by a non-radiologist.Despite that, it is not clear if it changes patient outcome or management. Therefore, further research is needed. Currently available host biomarkers lack accuracy for distinguishing bacterial or mixed bacterial-viral infections from viral infections. Because of this, BTS guidelines for CAP recommended microbiological testing onlyin children who need to be taken to the ICU or those with complications. Although it requires validations, analysis of the gene expression pattern, using microarray or RNAseq technology, can offer diagnostic signatures, help to assess disease severity,and, potentially, prognostic indicators, as it identifies the trigger specific pattern-recognition receptors on immune cells**.** **CONCLUSION:** Current methods of diagnosing CAP in pediatric patients are suboptimal, but new emerging approaches like ultrasound and RNAseq technology may improve assessment disease severity and patient outcomes. Optimizing the management of these children can potentially decrease use of antibiotics, thus prevent antibiotic resistance, lower health care costsand reduce mortality.

Keywords: Children; Pneumonia; Diagnostic;

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CASE REPORT: LOW VISION SECONDARY TO SEVERE COMPLICATED COMMUNITY-ACQUIRED PNEUMONIA

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INTRODUCTION: Community-acquired pneumonia (CAP) is pneumonia obtained in the social environment, outside of the hospital setting, or that manifests itself within 48 hours after hospitalization. Its main etiologic agent is Streptococcus pneumoniae. Despite its knowledge, studies on the etiology of CAP do not always identify the pathogen early, hindering the rational use of antibiotics, increasing complications and mortality. **CASE DESCRIPTION:** V.A.F., 5 years old, male, resident in Belo Horizonte, was hospitalized on December 04, 2019, due to severe respiratory condition. High frequency oscillatory ventilation was started, with good response, but maintaining extensive non-hypertensive pneumothorax. Chest CT showed moderate pleural effusion on the right with gas bubbles, mostly adjacent to the posterior upper lobe segments and lateral basal and upper lower lobe segments on the right. Voluminous free pneumothorax with formation of a hydro-aerial level. Extensive areas of lung consolidation associated with air bronchograms, sparse throughout both lung fields, especially in the middle and lower thirds. Fistulous tracts with the pleural cavity in this topography and basal lateral segment of the lower lobe of this lung were also identified. One day after admission, two continuous suction drains were implanted on the right with no resolution of the pneumothorax. The patient underwent thoracoscopy for lung decortication and bronchopleural fistula closure. During induction of anesthesia there was massive aspiration with evolution to severe septic shock, and volume expansion and administration of vasoactive amines were performed, with improvement of the condition. Pleural fluid culture showed P. aeruginosa, being treated with Ciprofloxacin for 28 days. After decortication, the drains were replaced by the Pneumostat drainage system. The patient still presented acute renal failure and deep venous thrombosis in the right lower limb requiring intermittent renal replacement therapy and anticoagulation, respectively. Mechanical ventilation was discontinued on January 14, 2020, and the use of oxygen via tracheostomy mask was initiated. As the respiratory condition improved, sedation was started to be reduced. Upon awakening, reduced visual acuity was noted, in addition to generalized muscle weakness. Brain MRI showed neurological alterations compatible with hypoxic-ischemic encephalopathy. The patient evolved with moderate visual impairment of cerebral origin, also known as visual impairment of central origin (VAD), reduced visual acuity (20/160 in both eyes), upper and lower temporal campimetric defect (confrontation fields), hyporeactive pupils in both eyes, small-angle divergent strabismus, and temporal pallor of the optic nerves. He started motor rehabilitation with physical therapy and visual rehabilitation with occupational therapy specializing in low vision. On February 10, 2020 the patient was discharged from the hospital with directions to maintain follow-up with pediatrics, neurology, ophthalmology, thoracic surgery, vascular surgery, physical therapy, occupational therapy, and speech therapy. **DISCUSSION:** The reported case highlights the complexity of a community-acquired pneumonia and one of its complications, low vision, with significant morbidity. Among the brain injuries that cause visual impairment, the most frequent are: hypoglycemia, hemodialysis, hydrocephalus, trauma, seizures, neurodegenerative diseases, encephalitis/meningitis, and other infectious diseases.

CONCLUSION: Despite the significant reduction in mortality rates from lower respiratory tract infections in recent decades, CAP still ranks third as a cause of death in Brazil, a fact that associated with the high cost of treatment makes it a challenge for public health in the country. The understanding of the neuropathology of childhood brain injuries associated with advances in prevention and forms of treatment allow improvements in prognosis, reduced morbidity and mortality, and impacts on the quality of life of these patients. Furthermore, the importance of early intervention with professionals specialized in visual and comprehensive rehabilitation of the visually impaired person is highlighted, aiming to ensure maximum motor, sensory and cognitive development.

Key Word: Community-acquired pneumonia; low vision; morbidity; children

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EXPANDED SUMMARY: OBSTRUCTIVE SLEEP APNEA SYNDROME AS AN ETIOLOGY OF ATRIAL FIBRILLATION: A CASE REPORT

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INTRODUCTION: Obstructive sleep apnea syndrome (OSAS) is a breathing disorder characterized by episodes of upper airway collapse during sleep. Atrial fibrillation (AF) is an irregular and rapid heart rhythm in which the atria do not contract properly. Their conduction system receives too much electrical stimulation, resulting in an abnormal level of impulse transmission, causing irregular ventricular rate. Over the past few decades there has been growing evidence that OSAS can cause AF, even though this is still an underdiagnosed consequence in clinical practice. **DESCRIPTION**: The patient F.D.M., male, 65 years old, hypertensive, former smoker, alcoholic, BMI 32.6, comes to the emergency room with complaints of palpitations. Physical examination revealed heart rate of 104bpm, irregular pulses and blood pressure of 123 x 78 mmHg. A 24-hour Holter was requested, which showed paroxysmal atrial fibrillation mainly at night. Transthoracic echocardiogram did not show significant alterations. In addition, blood tests did not identify any abnormalities. The patient was instructed to abstain from alcohol consumption and was administrated on Amiodarone, Bisoprolol, and Warfarin. Despite a partial improvement, after 3 months of follow-up, the patient continued to have paroxysmal episodes of atrial fibrillation, evidenced by maintenance of the palpitation symptom and by a new electrocardiographic study. A polysomnographic study was requested, which confirmed OSAS. The use of CPAP was started during nighttime sleep, as well as a weight loss program along with lifestyle habits changes. After 1 month of follow-up the patient had no further episodes of AF. Thus, the previously prescribed medications were discontinued. After 12 months of clinical and electrocardiographic follow-up the patient had no recurrence of AF. Besides, he presented an improvement of OSAS and a decrease in BMI to 28. **DISCUSSION**: Obstructive sleep apnea syndrome (OSAS) is a condition, where one has difficulties in breathing, resulting in hypoxemia and hypercapnia, awaking the individual intermittently. In the attempt to overcome it, muscular contractions open the airways in a period of hyperventilation, what leads to their collapse, forming thus a cycle. The forced inspiration against the occluded pharynx is accompanied by a negative pressure in the pleural space, increasing the pulmonary arterial pressure and overloading the right chambers of the heart. As a consequence, the right atrium dilates and pushes the interatrial septum, diminishing the left atrium and increasing the pressure inside it. Hence, there is myocardial dilation with structural changes, causing atrial fibrillation (AF). In the case described, the etiology of AF was not correctly diagnosed, and drug treatment was initiated. Prior to the suspicion of OSAS, a beta blocker was given by the medical team, since beta 1 receptors are abundant in the heart and are responsible for increasing heart rate, which can cause hypoxic conditions. Amiodarone, an antiarrhythmic drug used for treatment, was also prescribed to treat AF. In addition, a score 2 in the CHADS-VASc, which is an estimated clinical predictor of stroke risk for patients with non-rheumatic AF, supports the use of anticoagulants. Due to the refractoriness of the treatment initiated, a

possible association of AF with OSAS was considered. This association was confirmed by polysomnography and was followed by adequate CPAP treatment, resulting in disappearance of AF episodes. The use of CPAP is the most effective method for neutralizing the negative pressure caused by OSAS, since it forces the upper airway to open with positive pressure, normalizing breathing. Finally, it was fundamental to reduce the patient's BMI, since fat deposits in tongue muscles and tracheal wall favor airway obstruction, causing OSAS. **CONCLUSION**: The reported case depicts the association of obstructive sleep apnea syndrome (OSAS) and atrial fibrillation (AF). This association is usually not suspected at first contact with the patient. When patients with risk factors for OSAS present AF, or when there is refractoriness in the initial treatment of AF, the association of these two entities should be kept in mind for correct diagnosis and adequate treatment.

HEALTH SCIENCES DESCRIPTORS: Obstructive Sleep Apnea Syndrome; Continuous Positive Airway Pressure; Upper Airway Resistance; Atrial Fibrillation.

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EXPANDED SUMMARY: HISTOLOGICAL AND PHYSIOLOGICAL CHANGES IN THE AIRWAY DUE TO ASTHMA: A LITERATURE REVIEW

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INTRODUCTION: Asthma is a lower airway pathology characterized by bronchial inflammation, resulting from inflammatory cells, mediators, external stimuli and respiratory structural cells. In this context, asthma is a common disease that affects 300 million people worldwide and represents 250,000 cases of death per year. Thus, given such significance, a better pathophysiological understanding of asthma is sought, with the aim of understanding the mechanism of asthma and its histophysiological consequences. **METHODOLOGY:** A literature review was executed based on 16 articles, found through the PubMed and Scielo platforms, using the descriptors "asthma elastin", "asthma smooth muscle", "bronchial remodeling", "asthma basement membrane" and "asthma epithelium". A compilation of the main findings of these articles was created. **OBJECTIVES:** Show the main changes resulting from asthma in the various histological strata of the airways and their components. **DISCUSSION:** Asthma is a bronchial inflammation resulting from external and internal stimuli. A series of physical changes that lead to this inflammatory process were evidenced from the articles studied, such as changes in the respiratory epithelium, hyperplasia and hypertrophy of submucosal glands, vascular changes, degradation and fragmentation of elastic fibers and smooth muscle hypertrophy. The reviewed articles converge in considering that the respiratory epithelium undergoes a reduction in ciliated cells, desquamating this tissue, making it fragile and allowing the entry of antigens in the occlusion zonules. Moreover, during bronchoconstriction and bronchospasms, epithelial cells are subjected to a compressive force, resulting in mechanical stimulation, activating them to produce TGF-β, stimulating cells of the immune system to act there. Another altered component is the submucosal glands, which asthma can lead to hyperplasia and hypertrophy, contributing to excessive production of mucus, especially in severe asthma. Furthermore, ectasia of glandular ducts can occur, in which the presence of mucus in the lumen can increase resistance during bronchial smooth muscle contraction, so that the muscle has to do more force to contract, causing smooth muscle thickening. In regard to vascular changes, they are an essential feature of airway remodeling in severe asthma. Mainly, in fatal asthma, there is great bronchial vasodilation at the capillary level due to inflammatory mediators and growth factors. Hence, edema is favored as a consequence of bronchial microcirculation extravasation. In addition, the increase in mucus thickness leads to a decrease in caliber internal airway, in which vascular remodeling is a characteristic of asthma inversely proportional to the capacity of exhalation, being an important factor in airway flow obstruction. With regard to elastic fibers, there can be two possible mechanisms of injury in asthma: the action of proteases and elastases released by inflammatory cells and mechanical rigor. Enzymes have the ability to degrade elastin, which is the main fiber-forming protein. Mechanical rigor contributes to the rupture of these fibers. When elastin is degraded, elastin recoil decreases, which is the energy released from the deformation suffered by elastin fibers. Thus, the

lungs are able to exert less exhalation force, making it difficult for air to escape, which in turn causes dyspnea. Besides, the elastin fragments elucidate cellular effects that cause further inflammation and proliferation. In respect of smooth muscle, its hypertrophy in the airways as well as its hyperplasia can be caused by inflammatory agents, contractile antagonists, cytokines and growth factors released with asthma. In this way, the contraction of the bronchial smooth muscle is stimulated, resulting in bronchial narrowing. Airway smooth muscle cells in asthmatic patients can multiply twice as much as expected, characterizing muscle hypertrophy. According to the reviewed literature, the increase in number of myocytes results in overabundant narrowing of bronchi lumen. A genetic factor, the ADAM33 gene, which is strongly expressed in airway tissues fibroblasts and smooth muscle cells, can influence bronchial remodeling. This gene can lead to muscle hypertrophy, and consequent bronchial narrowing. **CONCLUSION:** The reviewed studies converge to the alteration and remodeling of several histological strata of the airways and present a complex histophysiological dynamic that determines the clinical scenery of asthma.

HEALTH SCIENCES DESCRIPTORS: Airway remodeling asthmatic; Bronchial; Pathology; Bronchial hyperreactivity.

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