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EXERCISE-INDUCED ASTHMA: CHALLENGES OF EXERCISE AND MANAGEMENT

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ABSTRACT

Exercise-Induced Asthma (EIA), also known as exercise-induced bronchospasm (EIB), is a condition in which the airways become blocked after exercise. Although exercise can cause asthma attacks, a lack of exercise can make asthma worse. Apart from taking medication, exercise can help control asthma attacks, and help improve the quality of life of the asthmatic population. Exercise can be done by people with mild to moderate degrees of airway obstruction, just like healthy people. Swimming, walking, cycling, playing ball and aerobics are good sports for asthma patients. Exercise-Induced Asthma (EIA) can also prevent asthma patients from exercising and affect their quality of life. The diagnosis of EIA is based on symptoms and spirometry or bronchial provocation tests. Nonpharmacological approaches, including avoidance of precipitating factors, are essential. SABA before exercise is also widely used and recommended as first-line therapy. Inhaled corticosteroids are recommended when asthma control is less than optimal. Leukotriene receptor antagonists (LTRA) and mast cell stabilizing agents (MCSA) are potential options.

KEYWORDS *Exercise-Induced Asthma (EIA), Exercise-Induced Bronchospasm, Asthma*

INTRODUCTION

Asthma is a common chronic respiratory disease but remains a major health challenge worldwide for individuals of all ages. Asthma is characterized by inflammation of the respiratory tract and can cause severe respiratory distress. Symptoms of asthma vary including shortness of breath, wheezing, coughing, and chest tightness. Lack of diagnosis and treatment of asthma is frequent, especially in low- and middle-income countries (WHO, 2024). Although its prevalence varies between age groups and ethnicities, it affects an estimated 339 million people worldwide, with rates ranging from 4% to 10% in Western countries (Lin et al., 2019; Ora et al., 2024; Seman et al., 2024).

The dynamic interaction between environmental and genetic factors, suggests that environmental triggers are crucial in initiating and worsening asthma

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symptoms. Risk factors include genetics, atopy, and hyperresponsiveness. Broncular inflammation is a characteristic of asthma, caused by many inflammatory cells, including mast cells, eosinophils, T lymphocytes, macrophages, and neutrophils. Exposure to allergens can cause inflammation and obstruction as the smooth muscles of the bronchi undergo constriction, edema, alterations, and increased mucus production (Brennan Jr et al., 2018).

Asthma sufferers have an inadequate quality of life due to ineffective asthma treatment. They are unable to lead a normal life, especially when it comes to sports or hobbies that demand physical activity because asthma symptoms can worsen. In order to better control symptoms and minimize the risk of asthma exacerbations, the application of non-pharmacological strategies can also be highlighted. Implementing practices such as quitting smoking, prioritizing vaccinations, and actively managing weight and exercising not only improves overall well-being but also plays an important role in comprehensive asthma management (Odeyemi et al., 2020).

Exercise-Induced Asthma (EIA) also known as Exercise-Induced Bronchotictionco (EIB) is one of the symptoms of activity-induced respiratory symptoms, which often leads to a sudden narrowing of the airways that occurs during or after exercise. Although the exact cause of EIB is unknown, asthma attacks can occur in people who have no previous history of asthma. Reducing physical activity is normal for people with asthma. This is caused by airway obstruction and increased sensitivity to various stimuli, including stimuli generated by physical activity. Physical activity is beneficial and important for those with asthma because it can improve the ability to exercise, reduce shortness of breath, and improve breathing ability in connection with activity. Exercise can be done by people with the same degree of mild to moderate airway obstruction as healthy people. Endurance and flexibility are essential components of physical exercise. Swimming, walking, cycling, playing ball, and aerobics are good sports (Hashim et al., 2023; Klain et al., 2024).

Definition

Exercise-Induced Asthma (EIA) is a clinical condition of bronchial inflammation due to acute (temporary and reversible) airway narrowing that occurs during or after exercise and can be observed in patients with chronic asthma and patients without asthma.7 EIA is reported to usually occur within 2 to 5 minutes after exercise, peak after 10 minutes, and disappear in about 60 minutes (Venkatesan, 2023).

Epidemiology

The prevalence of EIA varies from 5 to 20% in the general population. In children ≤ 16 years of age, the prevalence of EIA is also higher than that of the general population, ranging from 3 to 35%, with considerable variation in the prevalence of EIA in children worldwide, for example due to ethnic, urban-rural, and socio-economic differences. Exercise-Induced Bronchotictionco (EIB) causes 22.2% of children aged 4–12 years to develop asthma and 31.8% of children aged 13–17 years avoid sports activities. Caucasian children in urban areas, The

prevalence is 4.5%. Exercise-Induced Bronchotictionco (EIB) with asthma is more common in patients with severe, uncontrolled asthma. Exercise-Induced Bronchotictionco (EIB) occurs in 40-90% of children with asthma, especially in children with uncontrolled severe asthma. Exercise-Induced Bronchotictionco (EIB) without a history of asthma is common in athletes, children, subjects with rhinitis, and respiratory tract infections (Malewska-Kaczmarek et al., 2023).

Etiology

The exact cause of EIA is still unknown, but it is most likely related to mechanical stress and airway drying, leading to increased osmolarity of airway lining fluid and mediator release from immune cells (e.g., histamine, cysteinyl leukotrienes, and prostaglandins), leading to smooth muscle contraction of the airways and bronchoconstriction (Hostrup et al., 2024). Exercise is a common trigger for asthma, and high-intensity exercise, Exposure to allergens, and inhalation of irritants in certain environments play a key role in the increased prevalence of asthma. Epithelial injuries caused by inhalation of air pollutants and poor air conditions during exercise can lead to changes in bronchial blood flow resulting in bronchoconstriction. A family or personal history of atopy to environmental factors has been identified as a known risk for EIB (Aggarwal et al., 2018; Atchley & Smith, 2020).

Pathophysiology and pathogenesis

Obstructions appear during exercise or usually 5 to 15 minutes later and last for 30 to 60 minutes. Sometimes, symptoms go away on their own. The degree of airway obstruction caused by exercise varies depending on the intensity of the exercise, the type of activity performed, and the environment in which the exercise is performed. There are two theories about asthma related to physical exercise, namely the hyperosmolar theory and the osmotic and thermal theory. According to the hyperosmolar theory, during exercise there is an increase in ventilation which results in dry bronchial mucosal membranes (Greiwe et al., 2020; Støle Melsom et al., 2022). This happens because the air passing through the airways must be moistened resulting in the loss of moisture in the airways. As a result, surrounding cells, such as mast cells, are activated and cause bronchoconstriction (Ofiaeli et al., 2023; Price & Simpson, 2023).

Osmotic and thermal theories occur due to increased ventilation during intensive exercise.5 When breathing deeper and faster, they breathe in greater volumes of cool, dry air, leading to airway cooling and dehydration of airway surfaces (Koya et al., 2020; Satia et al., 2021). Osmotic theories state that increased ventilation in the airways during exercise periods leads to the loss of water from the airway surface through evaporation leading to an increase in airway while the concentration of ions (e.g. Nath, Kl-, Ca2th) in the airway surface fluid triggers dehydration of the airways (Figure 1). Events that cause contraction of the smooth muscles of the bronchi. During exercise-related hyperventilation, transient osmotic changes in the airway surface occur due to a decrease in the volume of epithelial fluid, which in turn triggers degranulation of mast cells. As a result, mast cell-mediated release of prostaglandins (prostaglandin D2), leukotrien, histamine, and

triptase occurs. These signaling molecules are known to mediate the contraction of the smooth muscles of the airways and increase mucus production and microvascular permeability as well as sensory nerve activation, and their release is considered a major stimulus for bronchoconstriction and airway edema.

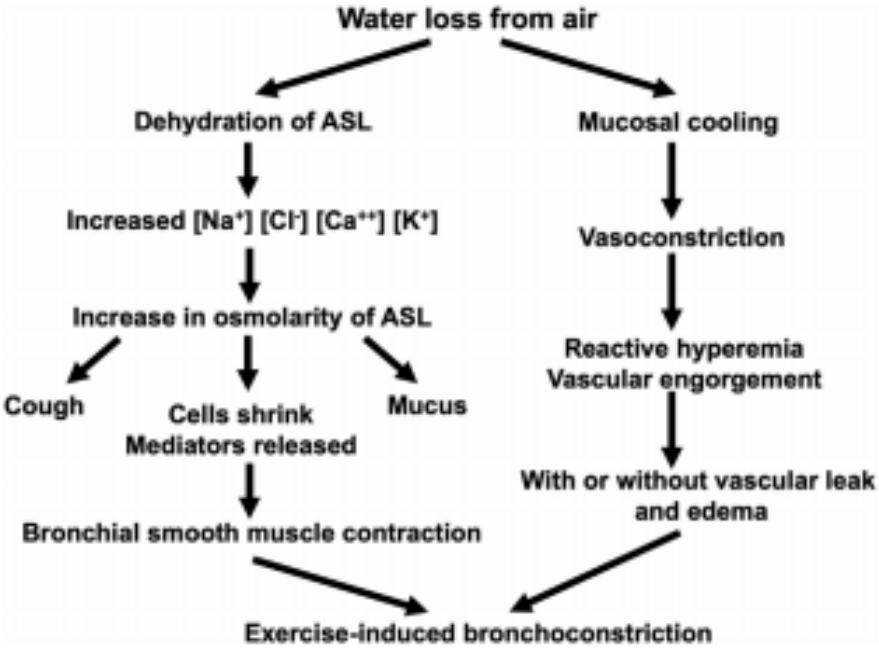

Figure 1. The mechanism of osmotic theory leads to EIB.19

The main cause of EIB is likely to be a combination of several underlying environmental and physiological factors such as stress on the lower airways, sensory and parasympathetic nerve stimulation, and airway epithelial injury (Figure 2).14

a. Dry Air Stressors

One of the functions of the airways is to humidify the dry air inhaled to ensure good cilia function, prevent hyperviscosity of secreted mucus, and protect against damage to the epithelial layer. During periods of high-intensity exercise, pulmonary minute ventilation can increase from a resting value of about 6 L/min to more than 280 L/min. Due to the increased necessary humidification that accompanies this increase in ventilation, there is an increase in the rate of water loss from the lower airways. The shift of water from the mucosa to the airway leads to a relative increase in intracellular osmolarity and a decrease in cell volume. Cell shrinkage leads to the release of soluble phospholipase A2 to produce arachidonic acid, which is taken up by lipid bodies in nearby mast and eosinophil cells. Activation of these cells in the hyperosmolar environment increases the formation and release of pre-formed mediators, such as histamine, prostaglandin D2, and cysteinyl leukotriene, which ultimately contribute to airway smooth muscle contractions, vascular leakage, edema, and EIB symptoms.

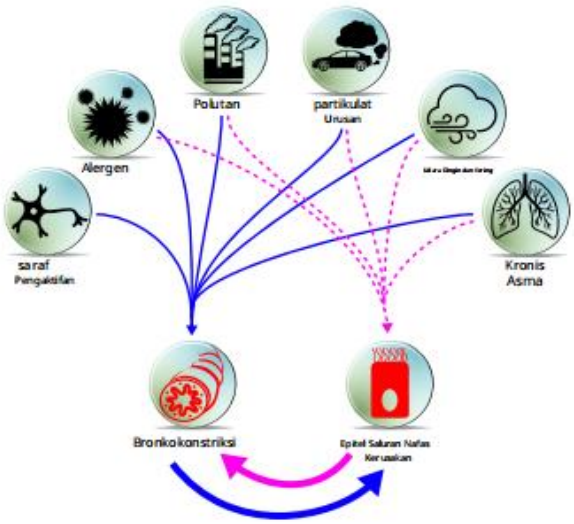

Figure 2 Stressors on the airways during intense and prolonged exercise cause damage to the airway epithelium and bronchoconstriction.14

b. Stressor Suhu

In addition to humidification, heating the inhaled air to ambient body temperature is another component of the air conditioning required before entering the alveoli. One of the proposed EIB mechanisms is that heat transfer from the mucosa into large volumes of relatively cold inspired air during exercise causes microvascular vasoconstriction within the airways, which then leads to hyperemia again after exercise as the airways become warm return. This leads to blood vessel leakage and edema that leads to narrowing of the airways, and is worse with intense, prolonged exercise.

Cold air inspiration was also shown to improve bronchial responses to histamine in subjects with or without asthma, as well as increase the number of granulocytes and macrophages in the lower airways. Cold air has also been shown to increase the stimulation of parasympathetic sensations that contribute to bronchoconstriction, both by the inspiration of cold air and through the mechanism of skin reflexes. In addition to stressors from cold ambient air, high temperatures are known to trigger symptoms in people with asthma.

c. Allergens, Particulates, and Pollutant Stressors

During exercise, The minute ventilation time exceeds about 30 L/minute, there is a switch from nasal breathing to combined nose and mouth breathing. With a greater ratio of inspired air passing through the nasal mucosa, a greater amount of particulates, including pollen, pollutants, and other inflammatory particulates, reaches the lower airways. This ratio can be further improved in patients who suffer from History (whether caused by allergies, non-allergies, or exercise), as inflammation of the nasal mucosa can result in increased resistance to nasal airflow. Even though known to trigger asthma symptoms, few studies have linked pollen sensitivity and exposure to EIB. One of the earliest findings was the finding that

children with asthma experienced increased EIB levels during the season with an increase in the amount of pollen or after provocation direct.

Particulate matter is most likely involved in the production of reactive oxygen species and a decrease in antioxidants, both of which were seen after exercise in athletes with EIB and caused airway inflammation. PM exposure has been associated with an increased number of emergency room visits, increased use of rescue inhalers, and post-exercise bronchoconstriction in patients with and without asthma. Particulate matter can be categorized by its size into fine, fine, or very fine, with increasing The inflammatory nature of the airways is inversely proportional to the size of the inhaled PM. Ultrafine PM, defined as less than 0.1 M in diameter m, has been shown to be stored in much higher amounts in healthy subjects during periods of exercise. Examples of PM encountered among elite athletes include exhaust gases from highway traffic, fossil fuels, electric-powered ice coating machines, air heaters, and passive smoking. Gaseous pollutants, such as ozone, nitrogen dioxide, trichloramine (found in chlorine-containing ponds), and sulfur dioxide are also likely to contribute to the inflammation resulting in EIB. Increased ozone concentrations in particular can lead to decreased tidal volume and FEV11, with increased specific airway resistance and respiratory symptoms during exercise, even among those who do not have of respiratory diseases.

d. Neuronal Activation

Recently, the nervous system has been proposed as another contributor to the EIB. The sensory nerves that mediate bronchoconstriction are activated directly by osmotic stress, and animal models have shown that dry-air hyperpnea results in the release of eucosanoid leukocytes, which also activate sensory nerves. Prostaglandin D2, which is mostly derived from mast cells, initiates sensory nerve activation via the DP1 receptor, which regulates airway tone. Evidence also suggests that increased neurokinin A and cysteineyl leukotrienes in the airways correlate with bronchoconstriction and production of MUC5AC, a gelling mucin, which contributes to airflow obstruction after exercise in EIB patients with asthma.

These findings suggest that bronchoconstriction and mucus release after exercise are at least partially mediated by sensory nerve activation and result in mediated release of mast cells. Differences in basic autonomic regulation of the nervous system among elite endurance athletes compared to non-athletes may also play a role in EIB. High-performing athletes in various sports experienced increased parasympathetic and decreased sympathetic nervous activity compared to non-athletes.

Similarly, a higher maximal oxygen uptake is a marker of a person's fitness level, and this is associated with increased vagal modulation. The endurance training regimen required by elite athletes can result in an increase in baseline vagal tone in response to the sympathetic stimulation often encountered during intensive training.

This increase in vagal activity promotes the stimulation of the parasympathetic nerve in the smooth muscles of the bronchi, thereby lowering the contraction threshold. In a study among elite swimmers, severe airway hyperresponsiveness correlated with increased parasympathetic activity

parameters. A significant correlation has also been observed among patients with EIB and reduced sweat secretion, saliva flow, and eye tears compared to patients without EIB.

e. Cedera Epitel

The airway epithelium serves as an important barrier against inhaled allergens, PM, pollutants, and pathogens, and the loss of the integrity of this barrier constitutes a significant bronchoconstriction mechanism in patients with EIB without asthma. As discussed earlier, with increased minute ventilation during exercise, there is an increased load of allergens, PM, and pollutants entering the airways, as well as increased susceptibility to dry air and thermal pressure. This causes damage to the epithelial lining of the airways through the loss of tight connections, resulting in an inflammatory response in susceptible individuals.

Various studies measuring inflammatory markers of airway inflammation after strenuous exercise support this concept, which shows an increase in the urine Clara Cell 16 protein (a marker of airway epithelial damage) and an increase in the concentration of interleukin 8, leukotriens, neutrophils, and bronchial epithelial cells in the sputum after exercise. In subjects with and without a prior diagnosis of asthma or EIB. In addition to the attraction of inflammatory cells, loss of airway epithelial integrity can interfere with mucociliary clearance and result in exudation of microvascular fluid and edema. Repeated exposure to plasma exudation products can decrease the sensitivity of airway smooth muscle contractions in response to exercise stress.

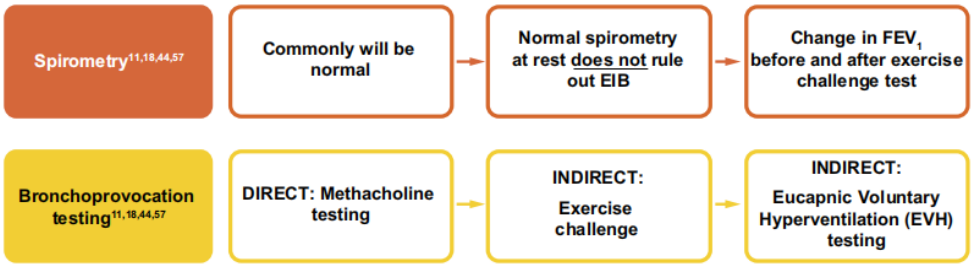

Figure 3. Diagnosis of EIA/EIB using spirometry and bronchoprovoking tests.13

RESEARCH METHOD

Diagnosis

Exercise-induced bronchospasm occurs during exercise and at six to eight minutes of intense aerobic exercise (estimated maximum oxygen consumption > 80% VO2). If the exercise period is shorter, symptoms may arise three to 15 minutes after the exercise is completed. Symptoms of EIB are similar to asthma and include sudden shortness of breath, coughing, mucus production, pain or tightness in the chest, and wheezing that lasts between 30 to 90 minutes and usually disappears spontaneously or may be atypical and difficult to recognize. The diagnosis of EIB is established based on specific changes in lung function triggered by exercise,

rather than on the basis of symptoms. Such testing may involve the use of spirometry and bronchoprovoking techniques (Figure 3).

Spirometry

The American Thoracic Society (ATS) Clinical Practice Guidelines outline a decrease in FEV11 from≥10% of the initial value after exercise or challenge hyperpnea as confirmation of a positive EIB diagnosis. A minimum of two reproducible FEV1 measurements were made in series after exercise, with the highest acceptable values recorded at each interval (usually 5, 10, 15 and 30 minutes after exercise). The lowest percentage of FEV1 reduction1 within 30 minutes post-exercise from pre-exercise levels can then be used to determine EIB severity (mild, 10–<25%; moderate, 25–<50%; severe≥50%).

Bronchoprovoked tests

Many protocols recommend breathing in dry air (10 mg H2O/L) with a nose clip attached when completing the exercise challenge. Some exercise test substitutes in the form of bronchoprovoking tests are available, depending on available resources, may be more suitable than dry air exercise challenges. The widely used methacholine test is the direct bronchoprovoking test; two versions of the methacholine challenge were used, the standard protocol recommended in the ATS guidelines, and a second faster protocol. Alternatively, there are a number of indirect bronchoprovoking tests. The Eucapnic Voluntary Hyperventilation (EVH) test was developed specifically to identify EIB. Dry air (containing 5% carbon dioxide) was hyperventilated at room temperature for 6 minutes with a ventilation target of 30 times FEV1, with a reduction of as much as ≥10% of the diagnostic pre-test value of EIB. EVH testing is considered to be a well-reproducible and standardized test, as well as quick and easy to perform however, it is laboratory-dependent so it is not widely available. Other indirect bronchoprovoking tests include hypertonic saline tests and mannitol tests. The latter was developed to improve the availability and standardization of osmotic challenge testing, but the sensitivity and specificity of mannitol testing are still poorly known.

Although none of these bronchoprovoking tests are sensitive or specific to EIB, they all complement the clinical history to identify airway hyperresponsiveness consistent with the diagnosis of EIB. In addition, although this test can be used for the diagnosis of EIB in patients with or without underlying asthma, it has been suggested that indirect bronchoprocating tests better reproduce the effects of exercise and therefore may be more accurate in diagnosing EIB in patients without asthma. The diagnosis of EIBwa is ultimately established based on a decline in lung function (i.e. bronchial hyperresponsiveness / BHR) triggered by a specific bronchoprovoking challenge.

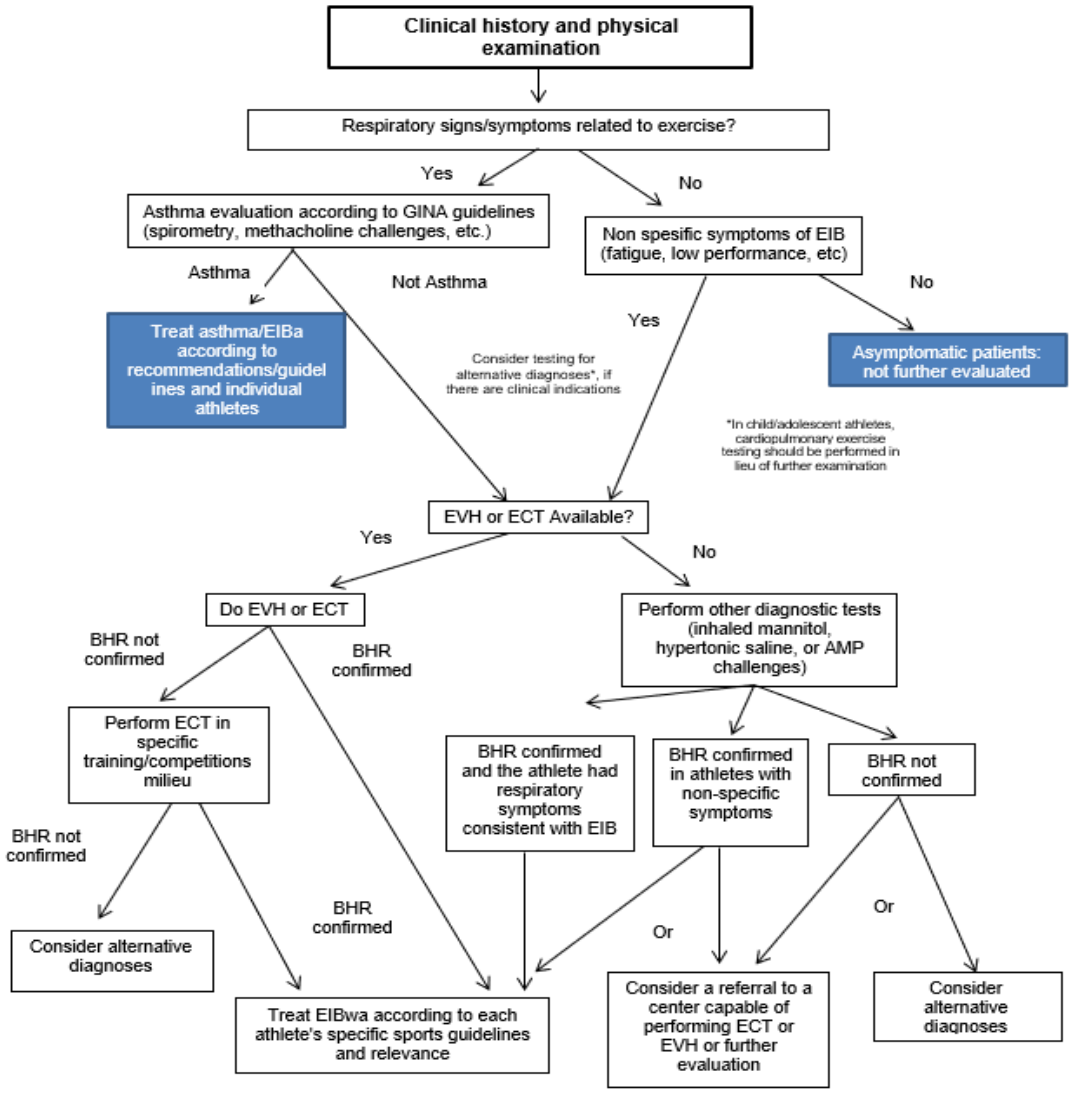

Figure 4. A proposed diagnostic algorithm for the evaluation of exercise-induced bronchoconstriction in athletes.14

Cardiopulmonary exercise test

Cardiopulmonary exercise testing monitors oxygen use, CO2 production, final tidal pCO2 (partial pressure of carbon dioxide), and electrocardiogram. Cardiopulmonary exercise tests (CPET) provide the means to investigate this, and are recommended prior to further screening specific to EIB in children and adolescents whose asthma diagnosis has been previously investigated or who have not responded to asthma treatment.

Tes Exercise Challenge

When using the Exercise Challenge test (ECT) to diagnose EIB, the type of exercise, intensity, and duration may play a role in detecting EIB, with the 2 most important determinants being sustainability, high level ventilation, and water

content in the inspiration air. Laboratory ECT involves training on a treadmill or cycling ergometer. An ideal exercise protocol should involve increasing the intensity of the exercise rapidly. When performed, an increase in the intensity of exercise-specific ECT compared to laboratory ECT in elite athletes should be considered, as the average decrease in FEV11challenge after exercise more than doubled after reaching 95% of the predicted maximum heart rate compared to only reaching 85%.

Before ECT is performed, basic spirometry must be obtained. Whether in the laboratory or field-based ECT, after the cessation of exercise at least 2 reproducible FEV1 measurements should be taken at intervals of 5, 10, 15, and 30 minutes. The highest acceptable value was recorded at each interval, with the largest decrease in FEV11 expressed as a percentage of the pre-workout score. Like mentioned earlier, a decrease in FEV of 110% or more from the baseline value is diagnostic for EIB. However, some experts recommend an even lower threshold (6.5%) for diagnosis in elite athletes, which has been shown to show a lower mean decrease in FEV1 compared to the general population.

Eucapnic Voluntary Hyperventilation

Eucapnic Voluntary Hyperventilation (EVH) is an indirect bronchoprovoking challenge developed for the diagnosis of EIB. Dry air containing 5% carbon dioxide is inhaled for 6 minutes with a target of 30 ventilation. It is possible to change the test parameters to simulate the need for breathing in an environment where symptoms can be triggered, or to better correlate with the environment in which the athlete performs. Examples include lowering the temperature of the inhaled air, increasing the target ventilation rate, and increasing the duration of the test. After the target test duration was reached, FEV1 was measured at 5, 10, 15, and 20 minutes post-challenge. These targets are compared to baseline FEV1 and, similar to ECT, a decrease in FEV11 of at least 10% is diagnostic of EIB. EVH is considered a well-reproducible and standardized test that is relatively quick and easy to perform. It also appears to be more sensitive than laboratory ECT in asthma patients and elite athletes, due to its ability to achieve and maintain higher minute ventilation than ECT. It may also have higher sensitivity and specificity than sport-specific ECT, although this is debatable due to the standardization and variability of the challenges mentioned above.

Although EVH has many advantages, this test is not without its drawbacks. Perhaps the most limiting factor of EVH is its lack of availability. Eukarnik voluntary hyperpnea requires specialized equipment and trained technicians, and it is relatively expensive when compared to ECT. The EVH challenge should be used with caution in recreational athletes, as the high ventilation levels obtained during testing may be much higher than those typically achieved in this population, resulting in false-positive results or clinically irrelevant testing. As with other diagnostic tests for EIB, there is a very varied documented sensitivity and specificity regarding the challenges of EVH for detecting EIB.

Other Diagnostic Testing

Other diagnostic tests used to evaluate EIB include hypertonic salt or mannitol powder inhalation challenges, inhaled AMP challenges, and methacholine challenge testing. These tests have the advantage of greater availability, lack of need for specialized equipment, and ease of deployment. In addition, this test is acceptable to the International Olympic Committee D of the Medical Commission to document evidence of BHR, which authorizes the use of bronchodilator drugs for Olympic participants, except for the AMP challenge. However, these challenges do not mimic the physiological stress of exercise.

Table 1: EIB diagnostic tests in elite athletes

Test	Krieria FEV1
Bronchodilation test	^ FEV1> 12% dan 200 ml
Eukarbic voluntary hyperventilation	↓ FEV1> 10%
Exercise tes	↓ FEV1> 10%
Tes methacholine	↓ FEV1> 20%
Hyperosmolar teas (saline, mannitol)	↓ FEV1> 15%

Eib: Exercise-induced bronchospasm,
Fev1: Volume of forced expiration in one second.22

Table 2: EIB.22 Fracture Degrees

Grade	Decrease in FEV1
Mild	>10% but <25%
Moderate	>25% but <50%
Severe	>50%

Such as ECT or EVH. Although this may indicate BHR, it depends on a higher likelihood of pretest EIB, and caution is advised when making a diagnosis based on a single positive test in patients without typical respiratory symptoms of EIB. For example, in elite athletes or endurance athletes with typical respiratory symptoms of EIB that do not include asthma and ECT or EVH are not available, an inhaled mannitol challenge may be performed (Pigakis et al., 2022).

If BHR is confirmed, the diagnosis of EIBwa must be established. In another example, an athlete who only reports fatigue and poor performance and where EVH or ECT cannot be performed, a mannitol challenge can also be performed. However, since the probability of pretest EIB is likely to be lower due to lack of respiratory symptoms, if BHR is indicated then it makes sense to refer to a center capable of performing ECT or EVH for further evaluation. However, it makes sense to start treatment of EIB with asthma in these patients and monitor their symptoms over time. If the BHR is not confirmed in this example, a referral can still be considered or an alternative diagnosis can be investigated.

Alternative Diagnosis

1 ALTERNATIVE DIAGNOSIS
1. Exercise-induced laryngeal dysfunction a) Vocal cord dysfunction, b) Laryngeal prolapse, c) Laryngomalacia 2. Obstructive/restrictive/vascular pulmonary disease a) PPOK, b) Asma, c) Bronkiektasis, d) ILD, e) PH 3. Neuromuscular diseases 4. Cardiovascular diseases 5.GERD 6. Overtraining syndrome 7. Lifestyle changes (alcohol, smoking, lack of sleep) 8. DEPRESSION – ANXIETY

Figure 3.Alternative diagnoses to consider in the evaluation of exercise-induced bronchoconstriction.22

Differential diagnosis of EIA requires a good medical history and proper testing. Exercise-induced laryngeal dysfunction (EILD) such as vocal cord dysfunction or paradoxical vocal cord movements, exercise-induced laryngeal prolapse, and exercise-induced laryngomalacia often mimic the symptoms of EIB. Exercise-induced laryngeal dysfunction can occur alone or simultaneously with EIB. Gastroesopharyngeal reflux disease (GERD) and laryngopharyngeal reflux can worsen with physical activity and are often associated with the duration and intensity of exercise. CPET can be used to differentiate dyspnea during exercise from EIB in obese patients. CPET may show increased oxygen consumption compared to bronchospasm. Cardiovascular disorders may be related to heart palpitations, dizziness, or syncope. Psychological factors can also be considered, especially if objective testing does not indicate a diagnosis (Anderson & Kippelen, 2023).

RESULT AND DISCUSSION

Governance

Prevention is the cornerstone of EIA therapy and can be achieved through pharmacological and non-pharmacological means. Pharmaceutical treatment of asthma is based on the severity of airway symptoms and is divided into five steps based on GINA guidelines. The nonpharmacological aspects of Exercise-Induced Bronchotictionco Asthma (EIBa) and EIB management emphasize patient education, reducing exposure to allergic environments and irritants as well as treating comorbid conditions, such as rhinitis and GERD. The pharmacological aspects of managing this condition focus on the use of appropriate medications for prophylaxis, symptom control, and rescue (Syabbalo, 2019).

Patients should be educated on the correct inhaler usage techniques, environmental control measures, and the importance of medication adherence. Regular follow-up is important to determine the effectiveness of treatment and ensure compliance. Treatment plans should be reviewed not only with the patient but also with coaches and coaches. Ultimately, EIB should not limit participation or success in strenuous activities and all athletes should be able to participate in any activity they choose without experiencing asthma symptoms. Although there are no restrictions on exercise selection for EIBa patients, choosing exercise based on low asthma potential may reduce symptoms.

Table 3. Treatment goals and effectiveness of EIB therapy.20

Treatment goals and effectiveness of EIB therapy
The main objectives of EIB treatment : - Prevent or minimize symptoms caused by exercise - Able to fully participate in any activity - Achieve and maintain control with as few side effects as possible - Prevent and control risk factors for acute events (e.g. exacerbations) - Maximizes lung function to enable optimal performance - In high-level athletes, ensure compliance with the regulations of the sports authorities **The effectiveness of EIB therapy may vary over time because:** - Changes in airway response over time - Environmental state - Workout intensity - Differences in initial airway response - Susceptibility to tachyphylaxis - Patient compliance - Genetic factors

Non Pharmacological Therapy

Non-pharmacological treatments for exercise-related asthma include avoiding exercise in cold, foggy and dry environments, and wearing a heat exchanger mask when exercising in cold or polluted environments. Similarly, prolonged exercise in hot weather should be avoided. Individuals with EIB should choose exercise with low ventilation levels to avoid hyperpnea during exercise that can trigger bronchoconstriction. Patients who are allergic to pollen aeroallergens should not do strenuous outdoor exercise during high pollen season. The American Thoracic Society recommends a planned warm-up interval or combination of exercises for all patients with exercise-induced bronchoconstriction. Conditioning exercises include warm-up exercises that are performed 45 minutes to 60 minutes before a workout or competition. Warm-up should be done for 10 minutes to 15 minutes with the goal of reaching 50% to 60% of the maximum heart rate.

This level of exercise, ventilation is expected to reach a maximum of 40% to 60%. This is useful due to the refractory nature of sports after warm-up. Exercise that starts suddenly and stops exercising abruptly puts people at greater risk of

developing EIA. Cooling down that lasts for 5 minutes to 10 minutes at the end of the workout is beneficial because it minimizes a sudden decline in cardiopulmonary function. It can also prevent rapid rewarming which can lead to bronchial swelling, airway edema, and bronchoconstriction. Dietary supplements with fish oil (omega-3 fatty acids) and ascorbic acid, and low sodium intake have been suggested to prevent EIB, but do not appear to be conclusive in reducing EIB. Physical conditioning improves the ability to work at lower levels of ventilation. Several cardiopulmonary programs have been recommended for people with asthma.

Pharmacological Therapy

Pharmacological therapy for EIB in individuals with asthma follows the same principles as general therapy for asthma, with an approach based on the severity and frequency of symptoms. Its main long-term goal is to reduce airway inflammation and prevent bronchoconstriction. This is usually achieved by using inhaled corticosteroids (ICS) in individuals with persistent or frequent EIB symptoms. ICS can be used as monotherapy or in combination.

Table 4. The main drug class used for EIB.2,25

Classification	Drug Name	Pharmacological Effects	Indication
Beta 2 Agonis Short Acting	Salbutamol, Terbutalin	Rapid therapy to overcome bronchoconstriction	Suitable for rapid pain relief but not intended for chronic use unless the person is using it simultaneously with ICS or ICS/LABA maintenance therapy
Beta 2 Agonis Long Acting	Formoterol, Vilanterol, Olodaterol	Maintenance care for bronchoconstriction	Not intended for use chronic, except when used in combination with ICS
Inhalation Corticosteroids (ICS)	Beclometasone, Budesonide, Flutikason Furoat, Flutikason propionate	Reduced airway peradangan	Used as monotherapy or combinations and not intended for it Quick help
Agen Muskarinik Short Acting	Ipratropium, Oxitropium	Broncodilation	The use of these drugs before exercise to prevent EIB is the subject of controversy and remains a Experimental approach
Agen Muskarinik Long Acting	Tiotropium, Umeclidinium, Glikopirronium	Maintenance care for bronchoconstriction	There is no evidence regarding Use of this class Drugs in athletes as monotherapy

ICS/LABA	Combination therapy that includes Inhaled Corticosteroids (ICS) and Long-Acting Beta 2 Agonists (LABs)	Asthma management in athletes	Used as needed and for maintenance therapy and is usually considered as a first-line treatment for mild to moderate asthma
Agen Biologis	Omalizumab, Mepolizumab, Benralizumab, Dupilumab	Treatment for the severe asthma in athletes and allergic reactions	Used in severe asthma, and rightfully not contraindicated in athletes with asthma
Leukotriene Modifier	Montelukast, Zafirlukast, Pranlukast	Asthma management in athletes	Reduces bronchoconstriction as a result of sports and providing Protection against Broncoconstriction triggered by exposure to pollutants
Cromon	Sodium chromolin, Natrium nedokromil	Prophylactic treatment for asthma, especially in athlete	Allowed to be used by athletes but may not be accessible in many markets

With long-acting beta agonists (LABs). In athletes, ICS is the cornerstone of asthma therapy, although its use is still less than that of inhaled β2 agonists. With regular use, ICS helps control asthma, improve lung function, and reduce airway responses to various triggers, including physical exercise. ICS should be considered at low daily doses if an athlete needs to take β2 agonists as needed more than twice a week, including doses needed to prevent exercise-induced bronchoconstriction, or if asthma limits exercise tolerance (i.e., the ability to exercise without bothersome symptoms). For some patients, ICS may be considered earlier (for example, if asthma symptoms occur, or treatment is needed more than twice a month, especially if there are risk factors for exacerbations). Adding other drugs, preferably long-acting inhalation β2 agonists, should be considered if ICS alone does not achieve asthma control.

Short-acting beta agonists (SABAs) relieve symptoms of EIB rapidly by relaxing the smooth muscles of the airways. Although athletes may use SABA as a quick-relief inhaler before exercise to prevent bronchoconstriction, recent research and recommendations do not suggest the use of SABA as a single treatment. In contrast, adults with mild asthma are advised to use ICS/formoterol as needed rather than routine ICS maintenance treatment along with SABA as needed. Similarly, adolescents with mild asthma are recommended to use ICS/formoterol as needed or ICS maintenance treatment in combination with SABA as needed or choose ICS/formoterol as needed rather than SABA as needed.

Other chronic therapy options may include leukotriene receptor antagonists (LTRAs) that inhibit leukotriene action, reduce exercise-induced bronchoconstriction, and provide a protective effect against bronchoconstriction

caused by exposure to pollutants or mast cell stabilizing agents (MCSAs). However, it should be noted that some MCSAs are no longer available in the market. The use of short-acting anticholinergics to prevent exercise-induced bronchospasm is still controversial while, there is no evidence of long-term use of anticholinergics. Although the focus of this review is not on doping regulations, it is important to clarify that certain drugs may be banned by the World Anti-Doping Agency (WADA) if they are considered to be performance-enhancing, and harmful to health. For athletes who manage exercise-induced bronchoconstriction (EIB), the use of asthma medications is not intended to improve their abilities but rather to prevent performance degradation due to EIB.9 This difference is key in understanding why WADA allows competing athletes to use inhaled corticosteroids (ICS) and beta2 agonists in specific doses, provided they show documentation proving that the prescription is for therapeutic use.

CONCLUSION

Exercise-Induced Asthma (EIA), also known as Exercise-Induced Bronchotictionco (EIB), is objective evidence of bronchial hyperresponsiveness in response to a variety of pulmonary stressors encountered during exercise, which may or may not present with typical respiratory symptoms and may occur in patients with or without underlying asthma. These airway stressors include the osmotic effects of dry air inhaled, environmental temperature variations, autonomic nervous system dysregulation, sensory nerve reactivity, and airway epithelial injury. The buildup of allergens, PM, and gaseous pollutants into the airways is another common cause of stress in the lungs that contributes to EIB. Athletes are exposed to these stressors much more frequently and for a greater duration than the general population, which is characterized by an increased prevalence of EIB in this population. The quality of life of these athletes often depends on the marginal performance benefits of competing at the professional or international level. A definitive diagnosis of EIB and the accompanying pharmacological and non-pharmacological treatments can improve and prevent symptoms that can impair athletic performance, and more importantly lower morbidity and mortality if asthma (i.e. EIBa) is identified and treated. Therefore, a strong suspicion of EIB should be raised in any athlete who reports respiratory symptoms or more subtle manifestations of EIB. Symptoms should not be relied solely on for diagnosis, but should be considered in the context of the presence of asthma in EIBa, CPET in children and adolescents, or bronchoprovocative challenges confirming BHR when considering EIBwa. Ideally, these challenges should reflect the competitive environment in which the symptoms appear to the fullest. However, more research is needed to validate the individual predictive values of these tests.

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