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EXERCISE-INDUCED BRONCHOCONSTRICTION IN ATHLETES - PATHOMECHANISM, CLINICAL SYMPTOMS, AND CURRENT METHODS OF TREATMENT AND PREVENTION

Anna Maruszak *(Corresponding Author, Email: anna.maruszak@onet.pl)*
American Heart of Poland S.A., Ustroń, Poland
ORCID ID: 0009-0009-2871-8800

Urszula Majda
Jędrzej Śniadecki Specialist Hospital in Nowy Sącz, Nowy Sącz, Poland
ORCID ID: 0009-0001-5144-2883

Magdalena Michalik
Hospital of the Brothers Hospitallers of Saint John of God, Cracow, Poland
ORCID ID: 0009-0003-6463-0802

Magdalena Mida
Sosnowiec Municipal Hospital, Sosnowiec, Poland
ORCID ID: 0009-0006-2815-6893

Julia Kwiecień
Municipal Hospital in Ruda Slaska, Ruda Śląska, Poland
ORCID ID: 0009-0009-2458-3224

Julia Stołtny
Zagłębie Clinical Hospital in Czeladź, Czeladź, Poland
ORCID ID: 0009-0008-0162-1809

Justyna Goryczka
District Hospital in Chrzanów, Chrzanów, Poland
ORCID ID: 0009-0004-4660-8318

Magdalena Lisik
Szpital Grochowski im. dr med. Rafała Masztaka SPZOZ, Warszawa, Poland
ORCID ID: 0009-0000-6196-3386

Witold Kimla
District Hospital in Chrzanów, Chrzanów, Poland
ORCID ID: 0009-0001-0695-9541

Natalia Hajok
Individual Medical Practice, Poland
ORCID ID: 0009-0003-5305-6075

ABSTRACT

Introduction and objective: This article discusses the issue of exercise-induced bronchoconstriction (EIB), also known as exercise-induced asthma (EIA). The aim of this paper is to discuss current methods of diagnosis and treatment of EIB in athletes in light of current guidelines.
Methodology: The ATS (American Thoracic Society) guidelines and available laboratory tests assessing bronchial hyperresponsiveness to indirect stimuli, such as exercise tests on a treadmill or cycle ergometer, eucapnic hyperventilation (EVH) and provocation tests using mannitol or hypertonic saline, and measurement of nitric oxide concentration in exhaled air (FENO).
Results: It was established that the diagnosis of EIB requires a decrease in forced expiratory volume in 1 second (FEV) of at least 10% after physical exercise. The prevalence of the condition is estimated at 12–15% in the general population, between 10–20% in athletes competing in summer Olympic sports, and increasing to 50–70% in athletes competing in winter sports. The key mechanisms of post-exercise bronchoconstriction are the thermal and osmotic theories.
Conclusions: Effective prevention of EIB includes both non-pharmacological methods and pharmacotherapy (primarily short- and long-acting beta2-mimetics, anti-leukotriene drugs). In patients with asthma, proper control of airway inflammation significantly reduces the severity of exercise-induced symptoms.

KEYWORDS

Exercise-Induced Asthma, EIB, Diagnosis, FEV1, Athletes, Prevention

CITATION

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Definition

Both exercise-induced bronchoconstriction (EIB) and exercise-induced asthma (EIA) are definitions that describe transient bronchoconstriction occurring in a group of people during or after intense physical exertion [1]. To diagnose exercise-induced bronchoconstriction, a decrease in forced expiratory volume in 1 second (FEV1) of at least 10% after physical exercise compared to the baseline value before exercise must be demonstrated [2].

Some researchers recognize a diagnostic criterion of a 15% decrease in FEV1 from the baseline value, particularly in relation to screening tests performed for epidemiological or field purposes [3].

It has been proven that the highest degree of bronchoconstriction is observed between 3 and 10 minutes after the end of physical exertion [4]. The bronchoconstriction response to physical exertion may also be delayed, occurring 3–8 hours after the end of exertion [5]. Some patients develop bronchospasm during exercise, but in some cases, symptoms may not appear during prolonged exercise due to bronchial dilation caused by the release of catecholamines during physical exertion [1]. During the refractory period, which lasts between 60 minutes and up to 4 hours after the end of activity, repeated physical exertion causes a less severe bronchospastic reaction or no reaction at all [1].

Objective: The aim of this study is to present current diagnostic criteria, pathophysiological theories, and methods of prevention and treatment of EIB in athletes.

Methods: This article is a narrative review of the literature, the aim of which was to provide a synthetic and critical discussion of current data on the diagnosis and treatment of exercise-induced bronchoconstriction (EIB) in athletes. Due to the heterogeneity of the available studies, the diversity of diagnostic protocols used, and the differences between the study populations, no quantitative analysis or meta-analysis was performed.

The literature review was based on publications available in international databases, including PubMed, Google Scholar, and Scopus, as well as current guidelines and consensus documents from scientific societies (ATS, ERS, EAACI, GINA) and the positions of sports organizations. Both original studies and review articles, consensus reports, and selected experimental reports that made a significant contribution to the understanding of the pathophysiology, diagnosis, or treatment of EIB were included. Publications published between 2000 and 2024 were included, with a particular focus on works from the last 10 years. The search included the following keywords and their combinations: exercise-induced bronchoconstriction, exercise-induced asthma, athletes, diagnosis, and treatment.

The selection of publications was deliberate, and the criteria for their inclusion were: clinical relevance for the diagnosis or treatment of EIB, timeliness of data, methodological quality, and frequency of citations in the literature. Particular emphasis was placed on studies involving competitive athletes and studies using indirect provocation tests, such as exercise testing, eucapnic hyperventilation with dry air (EVH), tests with mannitol or hypertonic NaCl solution, and measurement of fractional exhaled nitric oxide (FeNO).

Non-English publications were included when they were relevant to the topic under discussion or referred to local clinical conditions. Due to the narrative nature of the review, no formal assessment of systematic error risk or reporting schemes specific to systematic reviews (e.g., PRISMA) were used.

Epidemiology and characteristics of exercise-induced bronchoconstriction

The estimated prevalence of exercise-induced bronchoconstriction ranges between 12–15% in the general population [2]. Among athletes, it ranges from 10–20% among those practicing summer Olympic sports, and increases to 50–70% among athletes practicing winter sports [6,7].

Asthma, EIB, and allergic and non-allergic bronchitis are particularly common in athletes, which is influenced by factors such as the type of sport practiced, the training environment, and genetic factors [7].

Physical exercise is a trigger for asthma, and intense training, exposure to allergens, and inhalation of irritants are key factors in the increased incidence of asthma among athletes [8].

In addition, it has been observed that the airways of athletes are more sensitive, which makes them more vulnerable to environmental factors such as allergens, air pollution, and temperature changes, which exacerbate asthma symptoms and, as a result, can complicate treatment [7].

Among athletes, bronchial hyperresponsiveness is milder than in the general population with asthma [1].

The impact of EIB on the performance and quality of life of athletes

Regular physical activity can lead to a reduction in the severity of exercise-induced bronchoconstriction, improved lung function parameters, and reduced airway inflammation in people with EIB. This effect is associated with an improvement in overall physical fitness and adaptive changes in the respiratory system that develop in response to prolonged, systematic physical exercise. Early diagnosis of EIB, confirmed by objective changes in lung function occurring during or immediately after exercise, as well as the implementation of appropriate therapeutic measures, can significantly improve patients' quality of life and enable them to maintain regular physical activity [10].

Improperly treated or untreated asthma and EIB can negatively affect training and athletic performance, as well as contribute to chronic airway inflammation. Long-term inflammation can lead to damage to the bronchial epithelium, airway remodeling, and the development of fibrosis. At the same time, the presence of asthma or EIB is not currently an absolute limitation to achieving high athletic performance, as advances in therapeutic strategies allow people affected by these conditions to compete at a competitive level [10].

In the context of competitive sports, it is important that the treatment methods used comply with current clinical guidelines and applicable anti-doping regulations. Both athletes and the medical staff caring for them should monitor the dosage of the drugs used in order to minimize the risk of unintentional anti-doping violations [10].

Pathogenesis and mechanisms of EIB

There are two main theories explaining the pathomechanism of exercise-induced bronchoconstriction: the osmotic theory and the thermal theory [1].

The osmotic theory explains that during physical exercise, hyperventilation occurs, resulting in “drying” of the airways. This leads to an increase in the concentration of Na^+, Cl^-, Ca^{++}, and K^+ ions, which increases the osmolarity of the fluid covering the surface of the airway mucosa [1]. The resulting increase in osmolarity causes water molecules to shift from mast cells and eosinophils and the release of inflammatory mediators, which are histamine, leukotriene LTE4, and prostaglandin PGD2 [1]. The action of these mediators causes bronchial smooth muscle contraction and increased blood vessel endothelial permeability.

In addition, sensory nerves become irritated, causing mucosal edema and bronchoconstriction [1]. The thermal theory explains that the cooling of the bronchial mucosa during exercise and its subsequent warming after physical exertion causes an increase in mucus secretion, congestion, and then swelling of the mucous membrane [1]. It is currently believed that the thermal process further enhances the osmotic processes that occur during exercise [11].

The results of numerous studies have also shown that repeated hyperventilation can cause multiple damage and repeated multiple repair of the bronchial epithelium, which consequently results in an increased influx of both neutrophils and eosinophils and causes an increase in the concentration of leukotrienes in bronchoalveolar lavage fluid (BALF). -bronchial fluid (BALF, broncho-alveolar lavage fluid) [6,11,12,13].

During training in demanding conditions of low temperatures or dry air, inflammation and irritation of the respiratory tract epithelium occurs. This triggers the release of inflammatory mediators from cells, causing bronchoconstriction [9].

A biopsy performed on competitive skiers who had not previously suffered from asthma showed an elevated neutrophil count compared to patients with previously diagnosed asthma, accompanied by a lower eosinophil count [14].

Active athletes also show elevated levels of respiratory tract inflammatory mediators, i.e., increased numbers of eosinophils, neutrophils, and columnar epithelial cells [15,16,17,18].

Environmental conditions have a significant impact on EIB in athletes, with cold and dry air, pollution, inhaled allergens, and temperature fluctuations having a particular effect, as they can trigger exacerbation of symptoms [9].

Differential diagnosis

In the differential diagnosis of EIB, particular attention should be paid to chronic respiratory and cardiovascular diseases, gastroesophageal reflux, and hyperventilation syndrome. Both overtraining and low physical fitness can cause EIB [19,20].

In competitive athletes, vocal cord dysfunction (VCD) should also be considered in the differential diagnosis, while in swimmers, swimming-induced pulmonary edema (SIPE) and exercise-induced arterial hypoxemia (EIAH) should also be taken into account [6,11].

There is a group of patients in whom physical exertion can trigger a post-exercise anaphylactic reaction. Symptoms include hypotension, generalized urticaria, and angioedema [1].

Symptoms

Typical symptoms observed in people suffering from exercise-induced bronchoconstriction mainly include shortness of breath accompanied by coughing, wheezing, chest tightness, and shallow breathing [1].

Less common symptoms of EIB include epigastric pain, chest pain, dizziness, and muscle cramps [7].

Diagnosis

Relying solely on clinical history data to diagnose or rule out asthma and exercise-induced asthma/bronchoconstriction (EIA/EIB) may lead to both overdiagnosis and underdiagnosis of these conditions [21].

Previous studies indicate that symptom severity questionnaires have limited diagnostic value as a screening tool for suspected exercise-induced bronchoconstriction (EIB) [22].

The diagnosis of exercise-induced bronchoconstriction is based on objective confirmation of airway obstruction in response to a controlled exercise stimulus [3,6,7,23].

According to current guidelines, the response to a bronchodilator challenge is assessed based on the percentage change in forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) relative to the patient's predicted values. An increase in these parameters exceeding 10% of the predicted value is considered a positive result [24].

Contraindications to provocation tests according to the American Thoracic Society can be divided into relative and absolute [3].

Relative contraindications include: difficulties in performing spirometry, pregnancy, FEV1<60% of the predicted value or <1.5 l

Absolute contraindications include: heart attack or unstable ischemic heart disease in the preceding 3 months, poorly controlled hypertension, aortic aneurysm, breastfeeding*, treatment with cholinesterase inhibitors (patients with myasthenia gravis)*[3].

*Applies to tests performed with methacholine.

Exercise provocation test

Exercise tests can be performed on both a treadmill and a cycle ergometer, but due to the higher minute ventilation achieved during running or walking, treadmill tests are more commonly preferred. During the test, it is recommended to monitor heart function using at least a three-lead electrocardiogram. In people over 60 years of age or with additional risk factors for ischemic heart disease, continuous 12-lead ECG monitoring is necessary throughout the test [1].

In addition, continuous assessment of arterial blood oxygen saturation using pulse oximetry is recommended from the start of exercise until the end of the test. The laboratory performing exercise tests should be adequately prepared in terms of patient safety, including equipment for cardiopulmonary resuscitation, bronchodilators, and access to medical oxygen [1].

According to the guidelines of the American Thoracic Society (ATS), during exercise testing, the patient should breathe air with specific physicochemical properties — at a temperature of 20–25°C and humidity not exceeding 50%, with the optimal water content in the inhaled air being less than 10 mg/l. During the test, it is recommended to monitor ventilation, basic gas exchange parameters, as well as the humidity and temperature of the air inhaled by the patient [1].

Since the exercise challenge test is used to assess bronchial hyperresponsiveness, the use of drugs that may modify the test result should be discontinued before the test, taking into account the duration of action of individual medications [1].

Treadmill exercise test protocol

The total duration of the exercise test is 8 minutes, but for children under 12 years of age it is reduced to 6 minutes. The first 2-3 minutes are spent selecting the appropriate load so that the remaining test time — 5-6 minutes for adults and 4 minutes for children — is performed at a constant level of effort. During this phase, the heart rate should remain at 80-90% of the maximum heart rate, determined by the formula: 220 minus the age of the subject. Ventilation should correspond to 40–60% of maximum voluntary ventilation, calculated using the formula: $FEV_1 \times 35$. It should be emphasized that the main criterion for selecting the load should be minute ventilation, not heart rate alone [1].

Exercise test protocol on a cycle ergometer

The target load during the cycle ergometer test, expressed in watts, is calculated using the formula: $[(53.76 \times FEV_1 \text{ value}) - 11.07]$ [1]. In the first minute of the test, 60% of the calculated value is used, in the second minute 75%, in the third 90%, and in the fourth 100% of the target load. At a fixed level of effort, the subject continues the exercise for another 6 minutes [1].

The exercise test is considered one of the most effective methods of inducing exercise-induced bronchoconstriction (EIB), with a decrease in FEV_1 of at least 10% from baseline values being the diagnostic criterion [25]. However, some experts advocate a more stringent threshold of over 6.5% in the diagnosis of elite athletes. This is because the average maximum decrease in FEV_1 in this group is usually lower than in the general population, and the use of a lower threshold better reflects the presence of EIB among professional athletes [26].

Diagnostic criteria

The flow-volume curve during forced exhalation is recorded before the start of the exercise test and then at 5, 10, 15, 20, and 30 minutes after its completion. The last measurement may be omitted if the FEV_1 value at 20 minutes is close to the baseline value. A decrease in FEV_1 of more than 10% from the baseline value is considered abnormal, although some authors consider a change of ≥15% to be more diagnostic [3, 23].

In patients not taking inhaled or systemic glucocorticosteroids, a decrease in FEV_1 of 10–25% is classified as mild, 25–50% as moderate, and values above 50% as severe [2, 23]. Similar to standard bronchial hyperresponsiveness tests, in the event of a significant decrease in FEV_1 after the test, a short-acting β_2-mimetic should be administered and a control spirometry performed. If a single exercise test proves negative, EIB cannot be completely ruled out [27].

Alternative tests

Physical exercise causes bronchoconstriction indirectly, through the activation of inflammatory cells and stimulation of nerve endings, which release inflammatory mediators and cytokines, leading secondarily to airway obstruction. A similar mechanism underlies the action of other agents used in the assessment of bronchial hyperresponsiveness, such as non-isotonic aerosols (e.g., mannitol, hypertonic NaCl solution) and isocapnic hyperventilation with dry air [28].

Isocapnic hyperventilation with dry air

Isocapnic hyperventilation with dry air (EVH), often referred to as the "gold standard" of diagnosis, is particularly important in the diagnosis of exercise-induced bronchoconstriction (EIB) [29, 30]. Some studies indicate that EVH may induce bronchoconstriction more easily than classic physical exercise [30, 31].

Mannitol test

The mechanism of action of mannitol is similar to the effect observed after the use of a hypertonic sodium chloride solution. An increase in the osmolarity of the extracellular space leads to the release of inflammatory mediators from mast cells and nerve endings, which in turn induces bronchoconstriction [32].

Specially prepared powdered mannitol is placed in gelatin capsules in doses of 5, 10, 20, and 40 mg (± 0.2 mg). After performing a placebo inhalation, consisting of the use of an empty capsule, mannitol is administered using a dedicated inhaler in double doses, starting from 5 mg up to 160 mg. Administration of the preparation is continued until a 15% decrease in FEV_1 is achieved or until a maximum cumulative dose of 635 mg is reached. FEV_1 is measured 60 seconds after each inhalation [1].

The specificity of the mannitol challenge test in the diagnosis of exercise-induced bronchoconstriction (EIB), depending on the diagnostic threshold adopted (10%, 15%, or 20%), ranged from 58.6% to 78.6%, while the specificity ranged from 60.8% to 65.2% [33].

Similar to the mannitol test, the bronchial hyperresponsiveness test using a 4.5% sodium chloride solution administered via an ultrasonic nebulizer can be used in the diagnosis of exercise-induced bronchoconstriction (EIB) [34].

An alternative method to aid in the diagnosis of asthma is the measurement of fractional exhaled nitric oxide (FeNO). A recent multicenter retrospective analysis of 488 athletes showed that a FeNO value ≥ 40 ppb has good specificity, indicating its usefulness in confirming the diagnosis of exercise-induced bronchoconstriction (EIB). However, it should be emphasized that due to its limited sensitivity and low negative predictive value, FeNO measurement should not be considered a substitute for indirect bronchial provocation tests in the athlete population [35].

Discussion

Exercise-induced bronchoconstriction is a significant clinical problem both in the general population and among competitive athletes, in whom its incidence is significantly higher. The data presented in this study confirm that EIB remains an underdiagnosed condition, especially when diagnosis is based solely on medical history and reported symptoms. Numerous studies have shown a weak correlation between subjective complaints and objective indicators of bronchial obstruction, leading to both overdiagnosis and underdiagnosis of EIB.

The key element of diagnosis remains the demonstration of reversible bronchoconstriction in response to a provocative stimulus, with physical exercise and indirect tests, such as eucapnic hyperventilation with dry air or the mannitol challenge, having greater diagnostic value than direct tests. In particular, EVH is considered the gold standard for diagnosing EIB in athletes, although its use may be limited in untrained individuals due to difficulties in achieving the required minute ventilation. On the other hand, tests with mannitol and hypertonic saline, despite their moderate sensitivity, remain a useful diagnostic tool, especially in outpatient settings.

The lack of uniform interpretation criteria for elite athletes remains an important issue. Although a ≥10% decrease in FEV_1 after exercise is widely accepted as the diagnostic threshold, the validity of using lower cut-off values in this population is increasingly being questioned due to the different physiology of the respiratory system and the smaller range of FEV_1 decreases observed compared to the general population. This issue requires further research and standardization.

Osmotic and thermal mechanisms play a special role in the pathogenesis of EIB, leading to the activation of inflammatory cells and the release of pro-inflammatory mediators. In competitive athletes, a specific phenotype of airway inflammation is also observed, often with a predominance of the neutrophil component and features of epithelial remodeling, even in the absence of previously diagnosed asthma. This may explain the different response to anti-inflammatory treatment and the partially limited effectiveness of standard therapeutic regimens.

The therapeutic approach to EIB is not aimed at improving physical performance, but at preventing its deterioration as a result of bronchoconstriction. Although short-acting β_2-mimetics remain the most effective acute treatment, their frequent use, especially as monotherapy, raises significant concerns. Current guidelines recommend preferring anti-inflammatory treatment, including inhaled glucocorticosteroids, especially in athletes who require frequent use of rescue medications. The phenomenon of reliance on SABA despite GINA recommendations indicates a significant gap between recommendations and clinical practice.

Non-pharmacological strategies, including properly planned warm-up, modification of environmental conditions, and education of athletes, also remain an indispensable part of management. Reports on the phenomenon of refraction confirm that a properly conducted warm-up can temporarily reduce the severity of EIB, providing a valuable supplement to pharmacotherapy. Despite growing evidence, significant gaps remain, including the lack of standardized diagnostic thresholds for elite athletes and limited prospective studies comparing indirect provocation tests.

In summary, EIB is a condition with complex pathophysiology, requiring a multifaceted diagnostic and therapeutic approach. Despite advances in understanding the mechanisms of the disease and the availability of effective treatments, significant challenges remain in diagnosis, optimization of therapy, and tailoring recommendations to the specific needs of competitive athletes.

Treatment

The principles of therapeutic management of exercise-induced bronchoconstriction (EIB) are the same for both competitive athletes and individuals who do not participate in sports at a professional level [36, 37].

If asthma is confirmed, treatment should be based on current guidelines. Prevention of exercise-induced bronchoconstriction (EIB) includes both non-pharmacological and pharmacological interventions.

Non-pharmacological treatment

Non-pharmacological treatment of exercise-induced bronchoconstriction (EIB) involves several strategies.

Stickland et al. compared the effectiveness of different warm-up routines: high-intensity interval training, continuous low- or high-intensity training, and variable-intensity training, which included both light and very intense exercises [38].

The analysis showed that the greatest reduction in EIB symptoms was achieved with high-intensity interval warm-ups and warm-ups with varying intensity levels. These results suggest that incorporating high-intensity exercise elements into the warm-up routine may be an effective, short-term method of non-pharmacological reduction of exercise-induced bronchoconstriction symptoms [9].

It is recommended to perform a short but intense warm-up approximately 50–60 minutes before the planned exercise, aimed at inducing the phenomenon of refraction; the warm-up should last 10–15 minutes, and its intensity should be adjusted so that the heart rate increases to 50–60% of its maximum value. Secondly, when exercising in cold air, it is beneficial to cover your mouth and breathe through your nose, which reduces irritation of the respiratory tract. Another recommendation is to gradually reduce the intensity of exercise and avoid sudden warming of the airways, as studies indicate that controlled breathing after intense exercise in a cold environment may reduce the risk of EIB [7, 12]. In addition, it is advisable to follow a low-salt diet and increase the intake of omega-3 fatty acids from fish, which have anti-inflammatory properties [39, 40].

Pharmacological treatment

Appropriate treatment of allergic diseases, including allergic rhinitis, with intranasal steroids, antihistamines, or specific immunotherapy, which has a beneficial effect on improving the control of the underlying disease symptoms, may consequently result in a reduction in the severity of EIB [7].

An important clinical issue remains the fact that the effectiveness of EIB treatment may be limited and does not always lead to complete control of symptoms. Despite the need for periodic verification of the diagnosis, it should be emphasized that currently available pharmacotherapy methods do not allow for the complete elimination of exercise-induced bronchoconstriction [41]. In addition, athletes exhibit specific characteristics of the inflammatory response of the airways, which in some cases may be weakened or less severe, which may affect the effectiveness of treatment and the interpretation of symptoms.

In athletes, inhaled glucocorticosteroids (ICS) are a fundamental component of asthma treatment, even though in clinical practice they are used less frequently than inhaled β_2 receptor agonists [42]. Systematic use of ICS enables more effective control of the disease, improvement of lung function parameters, and reduction of airway hyperresponsiveness to various stimuli, including physical exertion. The inclusion of low-dose daily ICS should be considered in athletes who require short-acting β_2 agonists more than twice a week, also for the prevention of exercise-induced bronchoconstriction, as well as in situations where asthma symptoms limit exercise tolerance.

In certain situations, earlier initiation of ICS treatment may be justified, for example in the presence of asthma symptoms or the need to use rescue medication more than twice a month, especially in the presence of risk factors for exacerbations. If ICS monotherapy does not provide adequate control of the disease, combination therapy should be considered by adding another controller medication, preferably a long-acting inhaled β_2 agonist [42].

Short-acting β_2 agonists (SABAs) may be used in athletes on an ad hoc basis prior to physical exertion in order to reduce the risk of bronchospasm, but current scientific data and clinical recommendations do not recommend their use as monotherapy. In the treatment of adults with mild asthma, a strategy based on the ad hoc use of a combination product containing an inhaled glucocorticosteroid and formoterol is currently preferred as an alternative to a regimen involving regular use of ICS and SABA as needed [42].

Despite the high efficacy of inhaled short-acting β_2 receptor agonists in rapidly relieving the symptoms of exercise-induced bronchoconstriction and their prophylactic effect when administered 10–15 minutes before physical activity, many athletes continue to use these preparations routinely during training and competitions. This practice persists despite the current recommendations of the Global Initiative for Asthma (GINA), which, in cases of stable respiratory disease, recommend therapeutic strategies based on anti-inflammatory treatment with combined ICS/LABA preparations instead of the ad hoc use of SABA. This phenomenon indicates a significant discrepancy between current guidelines and actual clinical practice in the athlete population [37].

Conclusions

Exercise-induced bronchoconstriction (EIB) is a common and clinically significant problem in athletes, which can lead to reduced exercise tolerance and limited athletic performance. The diagnosis of EIB should be based on objective provocation tests, as clinical history alone has limited diagnostic value and may lead to both overdiagnosis and underdiagnosis of the disease. Effective therapeutic management requires a comprehensive approach, including anti-inflammatory treatment in accordance with current guidelines, rational use of bronchodilators, and appropriately selected non-pharmacological prevention strategies. The goal of treating EIB in athletes is to prevent deterioration in exercise capacity and training safety, not to improve physical abilities beyond physiological levels. Patient education, coaches, and medical teams, as well as close interdisciplinary cooperation, enabling effective treatment while complying with applicable anti-doping regulations. Further research, especially of a prospective nature, is necessary to optimize diagnostic and therapeutic strategies and to better adapt recommendations to the specifics of different sports disciplines.

Author’s Contributions:

Conceptualization: Magdalena Michalik, Justyna Goryczka
Methodology: Magdalena Mida, Urszula Majda
Software: Magdalena Mida, Julia Kwiecień
Check: Anna Maruszak, Julia Stołtny
Formal Analysis: Magdalena Lisik, Magdalena Michalik
Investigation: Julia Kwiecień, Natalia Hajok
Resources: Julia Stołtny, Anna Maruszak
Data Curation: Justyna Goryczka, Witold Kimla
Writing-Rough: Anna Maruszak, Urszula Majda
Writing-Reviewand and Editing: Anna Maruszak, Urszula Majda
Visualization: Witold Kimla, Magdalena Lisik
Supervision: Natalia Hajok, Julia Kwiecień

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