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Report about: Exercise Induce Bronchoconstriction(EIB)

Abstract (Summary)

Exercise-induced asthma, or more appropriately, exercise-induced bronchoconstriction (EIB), occurs in 80 to 90% of individuals with asthma and in approximately 11% of the general population without asthma. EIB is characterised by post-exercise airways obstruction resulting in reductions in forced expiratory volume in 1 second of greater than 10% compared with pre-exercise values. The mechanism of EIB remains elusive, although both cooling and drying of airways play prominent roles. Cold, dry inhaled air during exercise or voluntary hyperventilation is the most potent stimulus for EIB. Inflammatory mediators play central roles in causing the post-exercise airways obstruction.

Introduction

Diagnosis of EIB requires the use of an exercise test. The exercise can be a field or laboratory based test, but should be of relatively high intensity (80 to 90% of maximal heart rate) and duration (at least 5 to 8 minutes). Pre- and post-exercise pulmonary function should be compared, and post exercise pulmonary function determined over 20 to 30 minutes for characterisation of EIB. A pre- to post-exercise drop in of greater than 10% is abnormal. Approaches to treatment of EIB include both nonpharmacological and pharmacological strategies. A light exercise warm up prior to moderate to heavy exercise reduces the severity of EIB. More recently, studies have supported a role for dietary salt as a modifier of the severity of EIB, suggesting that salt restrictive diets should reduce symptoms of EIB. Short acting, inhaled β_2 -agonists constitute the most used prophylactic treatment for EIB. However, antileukotriene agents are emerging as effective, well tolerated, long-term treatments for EIB.

Discussion

Study One: Standardized exercise challenge tests, symptom scores and whole-blood eosinophil and basophil counts were made before and during the pollen season in 32 children suffering from hay fever or hay fever and asthma. All participants developed rhinitis symptoms during the season. The hay fever group showed in addition a significant seasonal increase in cough score (but in no other asthma symptom) and in circulating eosinophils, mean exercise-induced bronchoconstriction (EIB) did not change despite a slight increase in a few subjects. The asthma group showed seasonal increases in EIB, asthma symptom score, and total eosinophil count. The increase in the latter was significantly higher than that in the hay fever group. The relative basophil count remained unchanged in both groups. In conclusion, the hay fever group and the asthma group could be clearly distinguished with respect to EIB during natural pollen exposure. The significantly higher increases in EIB and circulating eosinophils observed in the asthma group might possibly be due to greater pollen antigen sensitivity in the asthmatics.

Result

EIB occurs in response to the loss of water from the lower airways that results from heating and humidifying large volumes of air in a short period. The resulting hyperosmolar environment activates various cellular mechanisms to release mediators from mast cells, eosinophils, epithelial cells, and sensory nerves. These mediators, in turn, lead to airway smooth muscle contraction and bronchoconstriction. Airway hyperresponsiveness in elite athletes may develop from a process of airway injury and changes in the contractile properties of airway smooth muscle.

Conclusion

EIB commonly affects individuals with and without clinically recognized asthma, especially those who participate in competitive athletics. Through years of research, the pathophysiology of EIB is now better understood and involves a complex interaction between several different cell types and mediators. Continued research to improve the knowledge regarding the mechanisms of EIB should aid the identification, diagnosis, and treatment of this common condition.

References:

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