ORIGINAL ARTICLE

The effect of loratadine in exercise-induced asthma

A Baki, F Orhan

METHODS

Fourteen children (eight boys, six girls; mean age 11.45 (SE 0.87) years, range 7–17) with bronchial asthma, diagnosed within the past month, were studied. All patients were judged atopic on the basis of positive skin tests to common allergens other than pollens, and increased serum concentrations of total and specific IgE. All patients had a history of exercise induced asthma, and had been previously shown to develop airway obstruction after exercise on a treadmill.

We investigated the effect of a 10 mg oral dose of loratadine, once daily for three days, on EIA in children, as we are unaware of any studies of its effects on EIA in the paediatric population.

Aims: To assess the effect of loratadine in exercise induced asthma

Methods: Randomised, double blind, placebo controlled study of 10 mg oral loratadine, once daily for three days in 11 children. At the end of the treatment period FEV1 was measured, and patients were exercised on a treadmill. FEV1 measurements were repeated at intervals after exercise.

Results: Loratadine significantly reduced the decrease in FEV1 after exercise at two, five, 10, 15, and 30 minutes, compared with placebo (p < 0.05). However, the mean decrease in FEV1 at five minutes was more than 15% of baseline in the loratadine group.

Conclusions: Loratadine reduces, but does not prevent, exercise induced asthma in children.

RESULTS

Of the 14 patients enrolled, 11 completed the study with full data available for analysis. Two patients did not fulfill the lung function entry criteria on the first exercise test day and were excluded. One patient dropped out as an acute attack necessitated the use of a systemic steroid.

Mean FEV1 of the remaining 11 patients was 81% (SE 3.7%) of predicted at entry to the study. Mean pre-exercise (baseline) FEV1 was 2.16 (0.19) litres on the loratadine day, and 2.08 (0.16) litres on the placebo day. There was no significant difference between the mean percentage fall in FEV1 after exercise in placebo treated children (p > 0.05). The mean percentage fall in FEV1 after exercise was reduced significantly by loratadine at two, five, 10, 15, and 30 minutes when compared with placebo (p < 0.05; table 1). However, the mean decrease in FEV1 after exercise at five minutes was more than 15% of baseline in the loratadine group.

The difference between mean blood pressure values before and after exercise was not significant. There was no significant differences in temperature and humidity on study days.
DISCUSSION

The use of antihistamines in EIA has generally been disappointing. This may be related to failure to achieve a sufficient concentration of antihistamines at lung H1 receptor sites, as older antihistamines could not be given in high doses because of their sedative and anticholinergic side effects. However, results of studies with the more potent, newer H1 receptor antagonists in EIA have been varied. Inhaled cetirizine, oral azelastine, and higher doses of terfenadine have been reported to protect against EIA, whereas ketotifen failed to show any effect.

In the present study, the fall in FEV1 after exercise was statistically reduced by loratadine. Based on the results, however, loratadine was not effective in the prevention of exercise induced asthma, as in the loratadine group, the mean decrease of FEV1 after exercise was more than 15%. We therefore conclude that loratadine, once daily for three days, reduces exercise induced bronchoconstriction but does not prevent it.

The airway response to histamine, which is known to be increased in asthmatic patients, is widely used to measure airway responsiveness. However, the inhibitory effect of a compound on histamine induced bronchoconstriction may not be predictive of its therapeutic efficacy. Obviously exercise, in contrast to inhaled histamine, is a natural bronchoconstrictor, so the prevention of EIA is of greater clinical value. Although loratadine produced a reduction in EIA, we only studied a limited number of patients, and did not compare various compounds, such as β2 adrenergic agonists or cromolyn sodium, known to be effective in EIA. We suggest that further clinical studies are required to determine the therapeutic role of loratadine in EIA.

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REFERENCES


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