ETIOLOGY

**Use of inhaled corticosteroids was associated with the development of cataracts**


**Objective**
To determine the effect of inhaled corticosteroid use on the development of cataracts in older adults.

**Design**

**Setting**
District general hospital west of Sydney, Australia.

**Patients**
4433 patients who were born before 1943 were identified by door-to-door census and invited to attend the study clinic for an eye examination. 3654 attended, of whom 3313 (mean age 66 years, 57% women) provided information on corticosteroid use.

**Assessment of risk factors**
Patients completed questionnaires specifying inhaled corticosteroid use (currently, in the past, or never). Investigators categorized beclomethasone use for usual number of puff/s/wk and calculated the estimated lifetime dose. Other risk factors assessed were history of smoking, diabetes, and hypertension; socioeconomic status; and sun-related skin damage.

**Main outcome measures**
Nuclear cataracts were identified, and their severity was graded by comparing photographs of the patients' eyes with 4 standard photographs; cortical and posterior subcapsular cataracts were graded by estimating the percentage of the lens covered by opacity. Relative-prevalence (RP) ratios were calculated for each type of cataract.

**Main results**
The rates of posterior subcapsular, cortical, and nuclear cataracts were 6%, 24%, and 19%, respectively. 11% of participants had used inhaled corticosteroids. The use of inhaled corticosteroids at any time was associated with increased prevalence of nuclear cataracts (RP 1.5, 95% CI 1.2 to 1.9) and posterior subcapsular cataracts (RP 1.9, CI 1.3 to 2.8). Higher cumulative lifetime doses of beclomethasone were associated with increased prevalence of posterior subcapsular cataracts. For a lifetime dose of beclomethasone ≥ 2000 mg among current users, the RP for posterior subcapsular cataracts was 5.5 (CI 2.3 to 13.0, P for trend < 0.001). The prevalence of nuclear cataracts was also greater at that dose of beclomethasone (RP 4.0, CI 1.3 to 9.3). An increased prevalence of cortical cataracts was seen only with current use of inhaled corticosteroids (RP 1.4, CI 1.1 to 1.7). Adjustment for risk factors, including systemic corticosteroid use, did not affect the strength of the association between inhaled corticosteroid use and the presence of posterior subcapsular cataracts (odds ratio for lifetime dose of beclomethasone ≥ 2000 mg 10.0, CI 3.3 to 33.2, P for trend < 0.001).

**Conclusion**
Use of inhaled corticosteroids was associated with increased risk for the development of nuclear and posterior subcapsular cataracts.

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Modern asthma protocols recommend that clinicians treat patients aggressively to achieve and maintain best lung function. Inhaled corticosteroids revolutionized the management of chronic asthma (1). They are effective in reducing or even abolishing symptoms, correcting abnormal physiology, and reducing the risk for death from asthma. Additional benefits include simplicity of administration by inhalation and the first-pass effect.

The unwanted effects of inhaled steroids are the same as those of their systemic homologues. However, the interpretation of the association between inhaled steroids and adverse effects is complicated by previous or concurrent use of systemic steroids and by the reduction in the amount of systemic steroids needed when inhaled steroids are introduced (1). The study by Cumming and colleagues confirms the increased risk for cataracts in patients using inhaled steroids. Although causal inferences from case-control studies are often questionable, it seems unlikely that bias can explain the odds ratio that Cumming and colleagues found for the association between a lifetime dose of beclomethasone ≥ 2000 mg and the presence of cataracts. The data indicate that clinicians should treat inhaled steroids with the same respect with which they treat systemic steroids. It is essential that they are used in patients for whom there is evidence about efficacy—that is, patients with asthma, not patients with nonreversible chronic obstructive pulmonary disease. Objective evidence of efficacy is very important. It seems logical to reduce doses to the minimum level and to stop and restart therapy rather than to continue therapy indefinitely. We need more information about dose–response and dose–adverse effect relations. We need better methods of documenting the effect of therapy with inhaled steroids. In addition to such indicators as symptoms, peak flow, and bronchial reactivity, tests that might provide serial estimates of airway inflammation should be evaluated (2, 3).

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References
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