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Issue 2

CAPture

A Commentary on Recent Allergy Papers

SYNOPSIS

- Children with asthma (n=76), atopic eczema (n=91) and seasonal rhinitis (n=69) were selected.
- Health care costs were estimated for the first 8 years of life.
- Costs were evaluated for each year and for each disease separately.
- Allergy diagnostics use (any) accounts for only ~ 1% of total costs in asthma and atopic eczema.
- Due to the very low total cost of seasonal rhinitis the diagnostics accounts for a relatively high percentage (19.3 %).
- Average cost of asthma treatment was US$ 627 per year compared to US$ 1 840 per year for severe asthma mainly due to hospital inpatient.
- Strong tendency towards lower costs with disease progression while maintaining steroid treatment in asthma.

Citation: Weinmann S et al. The costs of atopy and asthma in children: Assessment of direct costs and their determinants in a birth cohort. Pediatr Allergy Immunol 2003; 14:18-26

Early risk assessment, using allergy diagnostics, is suggested as a cost-saving strategy in early childhood asthma

The aim of this study was to assess the health care cost in asthma and other atopic diseases. Asthma treatment incurred an average cost of US$ 627 per year compared to US$ 219 and US$ 57 for eczema and rhinitis respectively. Twenty per cent of the children with asthma caused 76% of the health care costs. A remarkable finding was that 25% of asthmatic children with severe symptoms were not treated accordingly to established guidelines. Most steroid treatment was not initiated until the first hospital stay. Hospital stay caused 44% of the asthma treatment costs, whereas only 1% was due to allergy diagnostics. The authors imply that considerable cost-saving potential could be obtained by earlier preventive treatment of asthmatics and earlier risk assessment by using allergy diagnostics and family history. Another important remark from the authors in this study was that they considered seasonal rhinitis to be under-diagnosed.

SYNOPSIS

- A three-year follow-up study using daily diary cards.
- Asymptomatic adults with positive SPT (n=15) to birch, birch-pollen allergic patients (n=6) and healthy controls (n=25).
- Tested for specific IgE antibodies (Pharmacia CAP System™), skin prick test, conjunctival/nasal challenges, intradermal late-phase reaction.
- 60% (n=9) of the asymptomatic patients developed clinical symptoms to birch during follow-up.
- Specific IgE antibodies ≥0.7 kU_A/L was 87.5% predictive for symptoms compared to 69% for skin prick test (SPT).
- All patients with more than one sensitization and/or late phase reactions to birch developed clinical symptoms to birch.

Citation: Bodtger U et al. Asymptomatic skin sensitization to birch predicts later development of birch pollen allergy in adults: A 3-year follow-up study. J Allergy Clin Immunol 2003 Jan;111(1):149-54.

Birch-specific serum IgE ≥0.7kU_A/L predicts clinical symptoms within 3 years in adults with asymptomatic birch sensitization

In this small cohort, 15 asymptomatic birch pollen sensitized adults were followed-up after three years. Sixty percent of the patients developed clinical allergy. The positive predictive value (PPV) of birch pollen specific IgE ≥ 0.7 kU_A/L (class 2) to develop clinical symptoms was 87.5% with an odds ratio of 17.5 and a negative predictive value (NPV) of 71%. Skin prick test was less predictive (PPV=69%). All patients with more than one allergen sensitization at inclusion, developed a clinical birch pollen related allergy. There was no relation to age or sex for development of clinical symptoms. The authors conclude that further allergy testing could avoid unnecessary preventive attempts. In prediction of clinical consequences the results point at the importance to use a true quantitative assay to measure sensitization, but also to test if other sensitizations are present.

SYNOPSIS

- Sera and blood cells (PBMC) were obtained from 27 patients with WDEIA and controls without wheat allergy.
- IgE, IgA and IgG antibodies to crude gliadin and ω5-gliadin were measured by ELISA.
- Cell-mediated responses were assayed by histamine-release, lymphocyte proliferation and cytokine mRNA.
- All patients had IgE antibodies to crude gliadin and ω5-gliadin.
- Levels of specific IgA antibodies to ω5-gliadin ($p<0.001$) and crude gliadin ($p<0.01$) were increased in patients compared to controls.
- Histamine release ($p<0.001$) and lymphocyte proliferation ($p<0.01$) were increased in patients compared to controls.
- Increase in IL-10 mRNA ($p<0.01$) was less in patients than in controls.

Citation: Lehto M et al. Humoral and cellular responses to gliadin in wheat-dependent, exercise-induced anaphylaxis. Clin Exp Allergy 2003 Jan;33(1):90-5.

Increased levels of anti-gliadin IgA in patients with wheat-dependent, exercise-induced anaphylaxis (WDEIA)

One of the most frequent causes of food-dependent exercise-induced anaphylaxis is wheat. The researchers have earlier shown that ω5-gliadin in wheat is a major allergen. The aim of this study was to examine ω5-gliadin-specific IgA and IgG antibodies. Sera were obtained from 27 adult patients that had experienced anaphylactic reactions when exercising after ingesting wheat products.

The patients showed significantly higher levels of IgA antibodies to ω5-gliadin and crude gliadin than the controls. No significant increase could be seen in IgG antibodies. All patients had IgE antibodies to crude gliadin and purified ω5-gliadin. The authors speculate that ω5-gliadin-specific IgA could have a role in the removal of the gliadin from the circulation. The results indicate that gliadin IgA antibodies may be a useful marker of this serious reaction in wheat IgE-sensitized individuals, however studies are needed to confirm this.

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