

or more of therapy. The pathogenetic mechanisms involved are uncertain, although sometimes there is evidence of cell-mediated immunity to the penicillins in vitro or in vivo.^{1, 2}

The patients described here had maculopapular rashes within 1 day of receiving the semisynthetic penicillins, ampicillin and piperacillin, respectively. When skin testing with benzylpenicillin and semisynthetic penicillin skin test reagents was done, there was a delayed reaction at the intradermal skin test sites to the implicated semisynthetic penicillin reagents or the closely related derivative, that is, to ampicillin and amoxicillin or to piperacillin but no reaction to the major and minor determinants of benzylpenicillin. The reactions began at 9 to 12 hours and were maximal at 24 hours, fading by 72 hours, which mimicked the clinical course of the reactions.

Immunofluorescence showed no evidence for an antibody-mediated reaction. There was a marked lymphocyte reaction, and the distribution of the infiltrate and concomitant epidermal changes were characteristic of a marked delayed-type hypersensitivity response, with the predominant infiltrate being T cells expressing the CD4 antigen. There was a minor CD-8 positive component, and there were no B cells. The vascular endothelium itself strongly expressed HLA-DR antigen.

The reaction reported here is quite different from the cutaneous late-phase response, which typically follows an immediate hypersensitivity re-

action. That response is maximal at 4 to 8 hours and may persist for 24 to 48 hours. There is a mixed cellular infiltrate that contains both eosinophils and basophils, as well as some CD4-positive T cells. Neutrophils may be present early and late in the reaction.⁴ In contrast, the typical cell-mediated immune response in the skin consists of a mononuclear cell infiltrate with CD4-positive and CD8-positive T cells, which peaks at 24 to 48 hours and lacks a significant contribution from polymorphonuclear leukocytes.

There is therefore clinical and skin test evidence of a rapid cell-mediated immune response apparently specific for side-chain determinants in these two cases of maculopapular rash induced by semisynthetic penicillins. It is unclear how T cells recognize the small antigenic determinants expressed on the side chains of such molecules as ampicillin, amoxicillin, or piperacillin and distinguish them from benzylpenicillin.

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REFERENCES

1. Ahlstedt S, Kristofferson A. Immune mechanisms for induction of penicillin allergy. *Prog Allergy* 1982;30:67-134.
2. Vega JM, Blanca M, Carmona MJ, et al. Delayed allergic reactions to beta lactams. *Allergy* 1991;46:154-7.
3. Levine BB, Redmond AP, Fellner MJ, et al. Penicillin allergy and the heterogenous immune responses of man to benzylpenicillin. *J Clin Invest* 1966;45:1895-1906.
4. Lemanske RF Jr. The late phase response: clinical implications. *Adv Intern Med* 1991;36:171-93.

Bird-egg syndrome in childhood

Belén Añibarro, MD, M. Carmen García-Ara, MD, and
J. Antonio Ojeda, MD Madrid, Spain

In 1985 Maat-Bleeker et al.¹ described the first case of allergy to egg yolk possibly induced by respiratory sensitization to bird serum antigens. This peculiar sensitization has been designated as "bird-egg syndrome."² To date, no case has been described in children. We report a child with exposure to birds who experienced asthma caused by bird feathers when he was 5 years old and food allergy to egg 1 year later.

CASE REPORT

An 8-year-old boy with a family history of atopy had perennial asthma for the last 3 years. He had goldfinches and canaries at home, and the symptoms improved partially when the birds were removed. He reported immediate asthma when he was in contact with hens.

He had never experienced any symptoms of food allergy until 2 years ago when he refused to eat eggs because he said it resulted in cough, abdominal pain, vomiting, and throat pruritus. However, he showed no reaction to chicken meat.

Results of physical examination were unremarkable. The complete blood count and chemistry were normal except for eosinophilia (1000 cells/ml). Chest roentgenogram and results of spirometry were normal. A methacholine test revealed bronchial hyperreactivity.

From the Allergology Service, La Paz Hospital, Madrid, Spain.
Reprint requests: B. Añibarro, MD, Fermín Caballero 90, 8B,
Madrid 28035, Spain.

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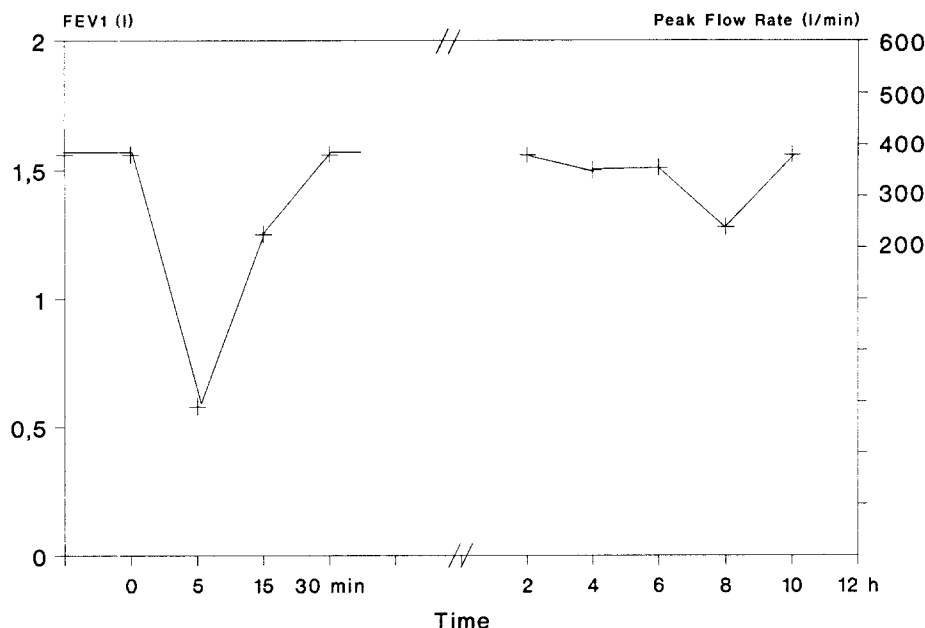


FIG. 1. Results of a bronchial provocation test with lyophilized egg yolk (1 mg/ml).

Skin prick tests with canary feathers 1 mg/ml (Leti, Madrid, Spain), chicken feathers 5% wt/vol (Abelló, Madrid, Spain), chicken meat and egg yolk 1/20 wt/vol (Dome/Hollister-Stier, Madrid, Spain) induced strongly positive reactions; ovalbumin (10 mg/ml) and egg white 1/20 wt/vol (Dome/Hollister-Stier) produced marginal results, and ovomucoid (10 mg/ml) remained negative.

Total IgE was 1105 IU/ml. Levels of specific IgE antibodies as determined by Phadebas RAST (Pharmacia, Uppsala, Sweden) were class 4 for chicken feathers, chicken serum, and egg yolk; class 3 for chicken meat and ovalbumin; and class 2 for egg white and ovomucoid. Results of the investigation of specific IgG antibodies (ELISA) for bird proteins were negative.

Challenge tests were performed during asymptomatic periods, without medication and with the parents' approval. Results of open oral challenge tests with increasing doses and spirometric control were negative with chicken meat but positive with egg white. (One hour after ingestion of approximately 6 gm of uncooked egg white, the patient experienced discomfort, abdominal pain, and vomiting; 30 minutes later urticaria and a 29% decrease in baseline forced expiratory volume in 1 second developed.) No oral challenge test with egg yolk was attempted.

A bronchial provocation test (Fig. 1) with lyophilized egg yolk extract (1 mg/ml) was performed as previously described.³ Five minutes after inhalation of the 10 μ g/ml dilution, the patient experienced shortness of breath and wheezing, and a drop in forced expiratory volume in 1 second of 63% was measured. Peak expiratory flow rate monitoring at 1-hour intervals during the next 10 hours showed a 33% asymptomatic deterioration in lung function at 8 hours, which reversed spontaneously 1 hour later.

A control patient with asthma showed no reaction to intradermal test and bronchial challenge at similar dilutions.

DISCUSSION

The bird-egg syndrome has been described in adults and is elicited when a bird is introduced to the home.^{1, 2, 4} The bird causes respiratory symptoms and oral allergy appears secondarily after egg ingestion. There is sensitivity to bird antigens (feathers, serum, droppings, and meat), as well as to egg proteins (mainly egg yolk), by cross-sensitization. The major cross-reacting antigen determinants are present in the livetin fraction of egg yolk.^{1, 2} α -Livetin, which has been identified as serum albumin, is the cross-reacting protein responsible for this syndrome.⁵

As far as we know, this is the first case of bird-egg syndrome described in a child. The patient had contact with birds since early childhood. The clinical history indicates that the inhalational sensitization to bird feather allergens at the age of 5 might have been responsible for the appearance of egg intolerance 1 year later.

It is important to note that this patient has egg allergy that differs from the typical forms of egg allergy in children: late onset at age 6, prevalence of digestive and respiratory symptoms over cutaneous symptoms, and greater sensitization to egg yolk than to egg white. We believe that these peculiar manifestations of egg allergy may be consistent with the bird-egg syndrome, and in such cases sensitization to bird proteins should be ruled out.

The principal route for sensitization to egg proteins seems to be the digestive one; however, sensitization by the inhalational route has been reported.³ Our patient has a heterogeneous allergic response with both respiratory and oral allergy to egg proteins, indicated by results of both challenge tests.

It has been shown that albumin involved in bird-egg syndrome also induced sensitization to chicken meat.^{2, 4, 5} Our patient also has a positive skin prick test and RAST results to chicken meat, even though he has a good clinical tolerance.

Finally, we think that in bird-egg syndrome avoidance of both inhaled (feathers) and oral (egg) forms of allergens is required.

REFERENCES

1. Maat-Bleeker F, Van Dijk AG, Berrens L. Allergy to egg yolk possibly induced by sensitization to bird serum antigens. *Ann Allergy* 1985;54:245-8.
2. Mandallaz MM, de Weck AL, Dahinden CA. Bird-egg syndrome. Cross-reactivity between bird antigens and egg-yolk livetins in IgE-mediated hypersensitivity. *Int Arch Allergy Appl Immunol* 1988;87:143-50.
3. Blanco JG, Juste S, Garcés M, Rodríguez P. Occupational asthma in the confectionary industry caused by sensitivity to egg. *Allergy* 1992;47:190-1.
4. Añibarro B, Martín Esteban M, Martínez Alzamora F, Pascual C, Ojeda JA. Egg protein sensitization in patients with bird feather allergy. *Allergy* 1991;46:614-8.
5. Pauli G, de Blay F, Bessot JC, Dietemann A. The association between respiratory allergies and food hypersensitivities. *ACI News* 1992;4/2:43-7.

Socioeconomic status and allergy in children with asthma

Linda J. Willies-Jacobo, MD,^a Joyce M. Denson-Lino, MD,^a
Angela Rosas, MD,^b Richard D. O'Connor, MD,^c and Nevin W. Wilson, MD^b
San Diego, Calif., and Morgantown, W. Va.

Minority children with asthma have increased morbidity and mortality rates. This appears to be due to socioeconomic status rather than culture or race. The purpose of this study was to determine whether socioeconomic status (SES) affects the incidence of allergies in children with asthma.

METHODS

Patient population

Allergy skin test results from 315 urban children with chronic asthma from San Diego, California were studied. All were followed up at the University of California, San Diego Pediatric Allergy Clinic, the Logan Heights Family Health Center, or the Division of Asthma and Allergy at the Sharp Rees-Stealy Medical

Abbreviation used

SES: Socioeconomic status

Group. This study was approved by the Institutional Review Boards of the University of California, San Diego and the Logan Heights Family Health Center.

The lower SES group was defined as children from families with no insurance or with Medi-Cal as their only form of health insurance. The higher SES group was defined as children from families with private insurance.

Skin test method

Each subject underwent allergy skin tests within the last 3 years in which the prick-puncture method was used. Saline and histamine controls were used in all patients. A positive skin test result was defined as any wheal at least one half the size of that produced by the histamine control. Wheals were read 15 minutes after placement, and responses were measured in millimeters. Personnel who read the skin tests were unaware of insurance status. Standard mite, grass, weed, tree, and mold mix extracts (Miles Inc., Hollister-Stier Div., New Haven, Conn.) were chosen on the basis of the prevalent allergens in the region.

From ^aUniversity of California San Diego, School of Medicine, Department of Pediatrics; ^bWest Virginia University, School of Medicine, Department of Pediatrics; and ^cSharp Rees-Stealy Medical Group, San Diego, Calif.

Reprint requests: Nevin W. Wilson, MD, West Virginia University School of Medicine, Department of Pediatrics, Section of Allergy and Immunology, G190, Health Sciences Center, Morgantown, WV 26506.

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