Letters to the Editor

Allergy to Chicken in Patients Sensitized to Anisakis Species

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To the Editor: I have read the study by Armentia and colleagues [1] and have found certain mistakes in it and I would also like to discuss some findings described. The authors found that subjects highly sensitized to Anisakis simplex can develop allergic reactions due to the presence of Anisakis species allergens in chicken meat when chickens are fed with fishmeal.

The discussion of the immunoblotting findings is insufficient. How do the authors explain that in the immunoblotting results for cereal-fed chicken (without Anisakis species antigen), a pool of sera from patients infested by A. simplex and a pool of sera from 8 patients of the study recognized a band of around 67 kDa? In immunoblotting using fishmeal-fed chicken, some sera from patients bound immunoglobulin (Ig) E in a band of around 16 kDa. However, this band did not appear in the results with A. simplex full-body extract or in A. simplex secretor extract. What is the explanation for this?

In their tables the authors stated that symptoms after avoiding a chicken and fish diet improved in all patients. I think this deserves a better explanation. Which patient had a positive oral challenge? It would be interesting to know specific IgE levels and immunoblotting results in this patient. Based on my experience in challenging patients with Anisakis species extract and larvae, it is difficult to believe that this patient had angioedema due to ingestion of chicken meat fed with fishmeal.

In the discussion the authors claim that Añíbarro and Seoane [2] reported that “asymptomatic patients tolerate the ingestion of dead larvae” and that “after double-blind, placebo-controlled food challenge with A. simplex lyophilized larva 10 of the 11 patients challenged showed positive conjunctival challenge test results.” In the cited article Añíbarro and Seoane only described a case of a fishmonger with a positive conjunctival challenge. Probably, they wanted to mention the study by Lluch-Bernal et al [3] (listed as reference 19 in the article by Armentia and colleagues [1] but never cited in the text). However, Lluch-Bernal et al only performed conjunctival challenge but never oral challenge. The oral challenge was published in another article by our group [4] that is not cited by Armentia et al. Also missing from among the studies that should be cited is one by Daschner et al [5], and one by Alonso-Gómez et al [6]. Finally, a study by Gómez et al [7], listed as reference 16 by Armentia and colleagues, only describes the etiopathogenic implications of Anisakis species in eosinophilic gastroenteritis.

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References


Author’s Reply

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To the Editor: I would like to thank Dr Sastre for his interest in our recent article [1].

The fact that a band around 65 kDa was recognized in immunoblotting tests using sera from cereal-fed chicken (without Anisakis species antigen) both by sera from a pool of samples from patients infested by Anisakis simplex and sera from 8 patients of the study could be explained by protein similarities to nematodes that frequently infest chickens (Ascaridia galli, Heterakis gallinae, etc). The possible explanation for the second issue Dr Sastre raises is that when the antigen in blotting is the secretor A. simplex antigen, the sera from patients sensitized to A. simplex larvae recognize more bands if they have been infested by ingesting this parasite. The reason why sera from patients 2, 4, 5, 6, 7, and 8 did not recognize more bands was probably that these patients were not primarily sensitized by the oral route and did not respond to secretor antigen.

Patient 2 worked in an animal food factory and patients 4 and 7 worked as chicken breeders. These patients could have become sensitized to A. simplex after inhaling airborne allergens. Patients 5 and 6 were cooks and also could have become sensitized when handling food.

In response to the third question, we performed oral challenge with cereal-fed chicken in all patients in order to rule out primary sensitization to chicken meat. Only patient 8, a medical student, consented to an oral challenge with meat from chickens that presented A. simplex antibodies in its serum. Specific immunoglobulin E levels to A. simplex in this patient, as appears in the table was 57.3 kU/L. Immunoblotting results can be seen in lane 7 of all of the figures. The patient presented angioedema after 10 minutes of a challenge with 160 mg of raw meat (accumulated dose of 300 mg).

There is a mistake in our reference 19, as Dr Sastre points out. The correct reference 19 would have been a short communication by Sastre and colleagues [2] in which they suggest that “the allergen or allergens responsible for clinical manifestations are produced by live larvae as secretory antigens, and thus may not be...
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present in sufficient quantities in dead specimens.” Añibarro and Seoane [3] published the first report, in 1998, that examined the possibility that *A. simplex* may also be an airborne allergen. The study by Spanish authors Gómez et al [4], which we included as reference 16, is a good description of different clinical pictures and diseases caused by *A. simplex* that should be taken into account (contact dermatitis, rheumatic symptoms, Crohn disease, etc). Obviously there are many good studies on *A. simplex* allergy and I apologize that it was impossible to cite all of them.

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References