

References

1. HAMILTON RG, BIAGNI RE, KRIEG EF, et al. Diagnosis of natural rubber latex allergy. Diagnostic performance of Food and Drug Administration-cleared serologic assays for natural rubber latex-specific IgE antibody. *J Allergy Clin Immunol* 1999;**103**:925–930.
2. TURJANMAA K, REUNALA T, RASANEN L. Comparison of diagnostic methods in latex surgical glove contact urticaria. *Contact Dermatitis* 1998;**19**:241–247.
3. VALLIER P, BALLAND S, HARF R, VALENTA R, DEVILLER P. Identification of profilin as an IgE-binding component in latex from *Hevea brasiliensis*: clinical implications. *Clin Exp Allergy* 1995;**25**:332–339.
4. DIEZ-GÓMEZ ML, QUIRCE S, CUEVAS M, et al. Fruit-pollen-latex cross-reactivity: implication of profilin (Bet v 2). *Allergy* 1999;**55**:951–961.

Is tropomyosin an allergen in *Anisakis*?

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Key words: cross-reactivity; invertebrates; nematodes; prevalence; recombinant tropomyosin.

• The fish parasite *Anisakis simplex* induces IgE-mediated reactions. Detection of specific IgE and prick test with a crude parasite extract are the current methods for diagnosis of sensitive patients, but high levels of specific IgE in asymptomatic individuals are detected (1). A recent study in Spain revealed that only 8/22 subjects having specific IgE to *Anisakis* were diagnosed as *Anisakis* allergics (2). Tropomyosin (TPM) has been proposed as a panallergen of invertebrates because it appears to be allergenic in many invertebrate sources, as both a food and inhalant allergen (3–5).

Tropomyosin is responsible for high titers of specific IgE in asymptomatic subjects.

Ten patients with clear clinical manifestations after ingestion of *Anisakis*-contaminated sea fish (group A), 62 patients with suspected *Anisakis* allergy (group B), and 16 patients with inhalant allergies to household insects were selected to evaluate, by *in vitro* techniques, the presence of TPM in *A. simplex* extract and its prevalence. The presence of TPM in extracts from *Anisakis* and *Ascaris* and their cross-reactivity to cockroach TPM were detected by shrimp-TPM antiserum and sera pool from patients allergic to household insects. None of 10 sera from *Anisakis*-sensitive patients reacted, by immunoblotting, with natural and recombinant *Anisakis* TPM, obtained as previously described (6). In contrast, when the inclusion criterion of sera was only the presence of IgE detected by CAP, the prevalence increased to 13% (8/62). These results suggested that TPM is not an important allergen in *Anisakis* sensitization. In food allergy to TPM, high-dose exposure of the allergen under

the harsh conditions of the gastrointestinal tract is necessary for sensitization, but, in the case of *Anisakis*, both the ingesta of parasites and the presence of TPM on the parasite cuticle are extremely low. In contrast, the implication of *Onchocerca volvulus* TPM in host protective responses to microfilariae in onchocerciasis has been recently reported (7). Interestingly, at least one of our patients in group B reported a previous infection with *O. volvulus*, and all but one of the residues of the described B-cell epitope of *Onchocerca* TPM (7) are conserved in *Anisakis* TPM.

Immunoblotting-inhibition using serum from an *Anisakis* TPM-positive patient showed inhibition of IgE-binding to *Anisakis* TPM when the serum was incubated with TPM from other invertebrates such as mite, cockroach, and shrimp (Fig. 1). The structural and immunochemical similarities of *Anisakis* TPM to TPM from other invertebrates, as demonstrated in this work, make the diagnosis of *Anisakis* allergy difficult. This

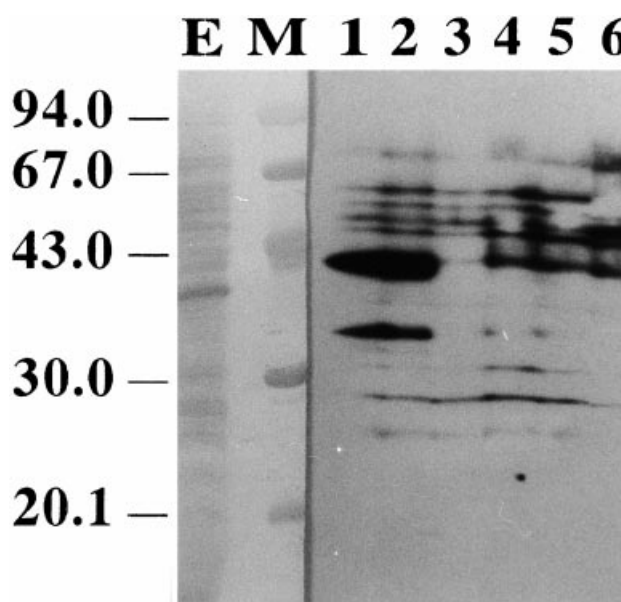


Figure 1. Immunoblot inhibition results. *Anisakis* crude extract (0.1 mg) was incubated with *Anisakis*-allergic patient serum preincubated with PBS alone (lane 1), 40 µg/ml of BSA (lane 2), natural TPM from *A. simplex* (lane 3), *P. americana* (lane 4), *P. atlanticus* (lane 6), and recombinant TPM from *D. pteronyssinus* (lane 5). *Anisakis* crude extract (E) and molecular mass markers (M) were stained with amido black.

similarity is one reason that CAP and prick skin tests produced many positive results in patients without clinical manifestations of *Anisakis* sensitivity.

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References

1. MONEO I, AUDICANA MT, ALDAY E, CUIEL G, DEL POZO MD, GARCÍA M. Periodate treatment of *Anisakis simplex* allergens. *Allergy* 1997;**52**:565–569.
2. DEL POZO MD, AUDICANA M, DIEZ JM, et al. *Anisakis simplex*, a relevant etiologic factor in acute urticaria. *Allergy* 1997;**52**:576–579.
3. REESE G, AYUSO R, LEHRER SB. Tropomyosin: an invertebrate pan-allergen. *Int Arch Allergy Immunol* 1999;**119**:247–258.
4. ASTURIAS JA, GÓMEZ BAYÓN N, ARILLA MC, et al. Molecular characterization of American cockroach tropomyosin (*Periplaneta americana* allergen 7), a cross-reactive allergen. *J Immunol* 1999;**162**:4342–4348.
5. ASTURIAS JA, ARILLA MC, GÓMEZ BAYÓN N, MARTÍNEZ A, MARTÍNEZ J, PALACIOS R. Sequencing and high level expression in *E. coli* of the tropomyosin allergen (Der p 10) from *Dermatophagoides pteronyssinus*. *Biochim Biophys Acta* 1998;**1397**:27–30.
6. ASTURIAS JA, ERASO E, MARTÍNEZ A. Cloning and high level expression in *Escherichia coli* of an *Anisakis simplex* tropomyosin isoform. *Mol Biochem Parasitol* 2000;**108**:263–267.
7. JENKINS RE, TAYLOR MJ, GILVARY NJ, BIANCO AE. Tropomyosin implicated in host protective responses to microfilariae in onchocerciasis. *Proc Natl Acad Sci U S A* 1998;**95**:7550–7555.

Hypereosinophilic syndrome presenting as acute myocardial infarction

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Key words: acute myocardial infarction; hypereosinophilic syndrome.

● **HYPEREOSINOPHILIC** syndrome is a leukoproliferative disorder characterized by a sustained overproduction of eosinophils. The distinctiveness of the syndrome, in addition to eosinophilia, is its marked predilection to damage specific organs, including the heart.

A 63-year-old woman was admitted to our hospital because of new onset anginal pain. She had a history of asthma treated with inhaled steroids, which had replaced oral steroids for the last 2 months.

Physical examination on admission was unremarkable, while laboratory tests revealed marked eosinophilia ($14.7 \times 10^9/l$) and elevated creatine kinase isoenzyme (CPK), which was later followed by elevated lactate dehydrogenase (LDH). Electrocardiography showed ST depressions in the lateral wall. Low-molecular-weight heparin, β -adrenergic receptor blockers, and ACE inhibitors were administered, while the presumptive diagnosis was acute myocardial infarction. Initial investigation including blood smear, stool examination, serum vitamin B₁₂ level, and chest radiography were all within normal limits, while serum IgE level was markedly elevated (1247 IU/l). On the second and third hospital days, the patient was asymptomatic with marked hemodynamic stability. Two I.V. boluses of high-dose steroids were given. On the fourth

hospital day, sudden pulmonary edema, hypotension, and new left-bundle branch block were noticed. While supported by mechanical ventilation, the patient was transferred to the intensive care unit. Echocardiogram showed left ventricle hypertrophy, severe systolic dysfunction due to posterior and basal hypokinesia, and a small pericardial effusion. Cardiac catheterization demonstrated normal coronary arteries.

The patient deteriorated with further hemodynamic compromise and died a few hours later. Postmortem bone-marrow aspiration and biopsy demonstrated 30% mature eosinophils characterized by hypersegmentation and pale granules. Myocardial biopsy showed heavy eosinophilic infiltration between myocardial fibers, with patchy myocardial necrosis.

The cardiac involvement in our patient was attributed to fulminant and atypical presentation of HES. Cardiac manifestations are common and usually are manifested late in the course of the disease, but, as a presenting symptom or sign, they are rare (1). The presenting manifestation of HES usually tends to be more insidious and present for months or longer. The most common symptoms include tiredness, cough, breathlessness, muscle pain, angioedema, rash, fever, and visual disturbances, all of which were absent in our patient (2). Our patient's initial evaluation suggested myocardial ischemia, which was not confirmed by cardiac angiography. HES was diagnosed on the basis of the characteristic histopathology findings.

The damage to the heart in HES ranges from early necrosis to subsequent thrombosis and fibrosis. Eosinophilic-mediated heart damage can evolve through three stages. The first is an acute necrosis stage in which the duration of the illness is short. This stage is not recognized clinically and is usually diagnosed at autopsy. The

The clinician should be alert when hypereosinophilia is found.