

Correspondence

Anisakiasis Mistaken for Dientamoebiasis?

TO THE EDITOR—*Dientamoeba fragilis* is one of the most common intestinal parasites of humans, infecting 43% of patients submitting stool samples for parasitological examination in Denmark [1]. Asymptomatic carriage is common, and the parasite can be found in at least 30% of healthy individuals (unpublished observations). Moreover, familial clustering of *D. fragilis* infections is common; this is potentially a consequence of vector-borne transmission by pinworm [2].

Gray et al [3] presented a case of a family cluster of *Dientamoeba fragilis* infections associated with peripheral eosinophilia (PE). Generally, reports on PE in association with intestinal protozoan infections are extremely rare, and it is likely that intestinal protozoa generally do not elicit this type of immune response. Although eosinophilia has previously been described in *D. fragilis* carriers, we know of no studies of sufficient size and controlled design documenting such findings as not merely coincidental.

We learn in the case report that the primary case—a 45-year-old man—presented with marked eosinophilia ($10.9 \times 10^9/L$ [total white blood cell count, $21.5 \times 10^9/L$] compared to $0.09 \times 10^9/L$ 2 years before) along with a 2-week history of mild abdominal distention. It turns out that 4 additional family members also presented with PE. Importantly, all of these individuals had frequently shared meals of raw fish (sashimi).

Ingestion of raw fish containing anisakids (mostly *Anisakis simplex* and

Pseudoterranova decipiens) may lead to anisakiasis in humans. The final hosts of *Anisakis* and *Pseudoterranova* include marine mammals (dolphins, whales, seals, walruses, etc); humans may be accidental hosts. L3 larvae in ingested raw fish meat may penetrate and embed themselves in the gastric or intestinal mucosa; in humans, the larvae usually die off instead of maturing to adult nematodes. Local, eosinophilic infiltrates may be a typical consequence, but PE may be a pathological finding as well [4, 5].

The diagnosis of anisakiasis is difficult as direct detection relies primarily on endoscopy, whereas serologic testing for anti-*Anisakis* simplex IgE can assist in the diagnosis of intestinal, ectopic, and allergic disease [5]. Therefore, the diagnosis of anisakiasis often relies on symptoms and relevant exposure. More than 90% of reported cases are from Japan [5], which may be why anisakiasis is not always included in differential diagnostic considerations in other parts of the world.

Based on the considerations above, we believe that the familial clustering of PE described in the article by Gray et al [3] is likely attributable to infection by anisakids and not to *D. fragilis*. In any event, anisakiasis should be included in differential diagnostic considerations in such situations. Furthermore, we find the recommendation to screen household members, irrespective of symptoms, for *D. fragilis*, to be unjustified in light of present-day knowledge, in particular the high prevalence of *D. fragilis* among healthy individuals.

Note

Potential conflicts of interest. Both authors: No reported conflicts.

Both authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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