European Review for Medical and Pharmacological Sciences 2012; 16: 1513-1518

Anaphylaxis caused by helminths: review of the literature

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Abstract. – BACKGROUND: Anaphylaxis is a severe, life-threatening, generalized or systemic hypersensitivity reaction. In many individuals with anaphylaxis a pivotal role is played by IgE and the high-affinity IgE receptor on mast cells or basophils. Less commonly, it is triggered through other immunologic mechanisms, or through nonimmunologic mechanisms. The human immune response to helminth infections is associated with elevated levels of IgE, tissue eosinophilia and mastocytosis, and the presence of CD4+ T cells that preferentially produce IL-4, IL-5, and IL-13. Individuals exposed to helminth infections may have allergic inflammatory responses to parasites and parasite antigens.

AIM: To summarize the evidences about the role of helmiths in triggering anaphylaxis

MATERIALS AND METHODS: PubMed search was performed by combining the terms (anaphylaxis, anaphylactic, anaphylactoid) with each one of the etiological agents of human helminthiasis for the period January 1950 to September 2012.

RESULTS: The PubMed search identified 609 papers. Only four genera of helminths were associated with anaphylaxis. (*Echinococcus* spp, 302 papers; *Anisakis* spp, 73 papers; *Taenia solium* cysticercosis, 7 papers; and *Ascaris* spp., 243 papers).

CONCLUSIONS: The risk of anaphylaxis in patients with helminthiasis can vary according to the pathogens, occurring more frequently during echinococcosis of after anisakis infestation and being extremely rare after other helminth infestations. However, physicians, allergist and parasitologist in particular, should be aware of a potential anaphylaxis caused by helminths.

Key Words:
Anaphylaxis, *Anisakis*, Ascaris, *Echinococcus*, Helminth, *Taenia*.

Introduction

Anaphylaxis is a severe, life-threatening, generalized or systemic hypersensitivity reaction. The reaction usually develops gradually, most often starting with itching of the gums/throat, the palms, or the soles, and local urticaria; developing to a multiple organ reaction often dominated by severe asthma; and culminating in hypotension and shock[1].

In many individuals with anaphylaxis a pivotal role is played by IgE and the high-affinity IgE receptor on mast cells or basophils. Less commonly, it is triggered through other immunologic mechanisms, or through nonimmunologic mechanisms[2].

Anaphylaxis episodes range in severity from those that are mild and resolve spontaneously to those that are fatal within minutes[2].

Helminths are eukaryotic worm-like organisms that live and feed off living hosts, receiving nourishment and protection while disrupting their hosts' nutrient absorption, causing weakness and disease. From a taxonomic point of view they are classified in cestodes (tapeworms), trematodes (flukes) and nematodes (roundworms). They can live inside humans as well as other animals. Helminths can invade the intestinal tract as well as urinary tract, and blood. In effect, any organ can be affected by adult worms or their larvae[3,4].

The human immune response to helminth infections is associated with elevated levels of IgE, tissue eosinophilia and mastocytosis, and the presence of CD4+ T cells that preferentially produce IL-4, IL-5, and IL-13[5]. Individuals exposed to helminth infections may have allergic inflam-

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matory responses to parasites and parasite antigens. Parasitic helminths in endemic areas tend to cause chronic infections – individual adult parasites may survive for many years in their human host – that are associated with few allergic-type reactions and a more tightly controlled Th2 response. Regulation of the Th2 response may be important for parasite survival and may allow the host to escape potentially damaging inflammation in the tissues[6].

Today, it is largely assumed an inverse association between helminth infection and allergy, although helminths induce a polarized Th2 response[7,8]. However, very little data are available to explain how helminth infection might protect against allergy. Some Authors demonstrated that this protection involves immunoregulatory mechanisms that block production of allergen-specific IgE[9], with protection being mediated at least in part by the production of IL-10. Studies on animal models showed that infection by helminths as *Schistosoma mansoni* protects mice from an experimental model of systemic fatal anaphylaxis[10]. However, another study demonstrated that *Trichinella spiralis*-infected mice had exacerbated anaphylaxis[11]. The differences between the studies could be due to the differences in the infectivity and immunity of the parasites and in the allergy models used.

It is also known that helminths may be rare causes of acute and chronic urticaria, through an IgE mediated release of histamine[12,13]. Moreover, anaphylactic reactions are extremely rare.

Literature Review

PubMed search of human cases of anaphylaxis occurring during helminthic diseases was performed combining the terms (anaphylaxis, anaphylactic, anaphylactoid) with each one of the etiological agents indicated in Table I for the period January 1950 to September 2012; references were also checked for relevant articles, including review papers. A study was considered eligible for inclusion in the systematic review if it reported data on patients with helminthiasis who had signs or symptoms of anaphylaxis.

Results

The PubMed search identified 609 papers. Only four genera of helminths were associated with anaphylaxis (*Echinococcus* spp, 302 papers; *Anisakis* spp, 73 papers; *Taenia solium* cysticercosis, 7 papers; and Ascaris spp., 243 papers).

Echinococcosis in humans occurs as a result of infection by the larval stages of taeniid cestodes of the genus *Echinococcus (E.)*. Six species have been recognized, but four are of public health concern: *Echinococcus granulosus* (which causes cystic echinococcosis), *E. multilocularis* (which causes alveolar echinococcosis), and *E. vogeli* and *E. oligarthrus* (which cause polycystic echinococcosis)[14].

E. granulosus is a cestode whose life cycle involves dogs and other canids as definitive hosts for the intestinal tapeworm, as well as domestic and wild ungulates as intermediate hosts for the tissue-invading metacestode (larval) stage. The metacestode (echinococcal cyst) is a fluid-filled, spherical, unilocular cyst[15]. Each cyst is surrounded by a host-produced layer of granulomatous adventitial reaction. Small vesicles called brood capsules bud internally from the germinal layer and produce multiple protoscolices by asexual division. In humans, the slowly growing hydatid cysts can attain a volume of several liters and contain many thousands of protoscolices.

With time, internal septations and daughter cysts can form, disrupting the unilocular pattern typical of the young echinococcal cysts[14].

It is well known that anaphylaxis, including urticaria, edema, and respiratory symptoms, may occur in persons infected with *E. granulosus*. The first reports in literature about anaphylactic shock due to echinococcus infestation dates back to the 70's[16-18].

Table I. Parasitic helminths.

Ancylostoma spp, *Angiostrongylus* spp, *Anisakis* spp, *Ascaris* spp, *Baylisascaris procyonis; Brugia* spp; *Clonorchis* spp; *Dicrocoelium dendriticum; Dioctophyme renale; Diphyllobothrium* spp, *Dirofilaria* spp, *Dracunculus medinensis, Echinococcus* spp, *Echinostoma echinatum, Enterobius vermicularis, Fasciola* spp, *Fasciolopsis buski, Gnathostoma* spp, *Hymenolepis nana, Hymenolepis diminuta, Loa loa, Mansonella streptocerca; Metagonimus* sp, *Necator americanus, Onchocerca volvulus, Opisthorchis* spp, *Paragonimus* spp, *Schistosoma* spp, *Spirometra erinaceieuropaei, Strongyloides stercoralis, Taenia* spp, *Thelazia* spp, *Toxocara* spp, *Trichinella* spp, *Trichobilharzia regenti, Trichuris trichiura, Wuchereria bancrofti*

Anaphylaxis may occur if fluid from the cyst is released into a host who has developed IgE from previous leakage of fluid; a severe reaction can result from release of fluid from the cyst, either spontaneously or after trauma or surgery[19]. Anaphylaxis complicates 10% of all intraperitoneal ruptures of cysts[20]. The prevalence of anaphylactic shock during surgery treatment for cystic echinococcosis consistently varies in the studies. In a Chinese report of 2011 it is about 2%, being anaphylaxis more frequent in people with younger age and cysts occurring in the lungs[21]. A meta-analysis that took account of 14 published articles with 952 subjects who underwent surgical intervention for cystic echinococcosis from 1990 to 2001 showed a rate of 25.1% of major reactions including anaphylactic shock. However, the real rate of anaphylaxis in not stated, including the rate other reactions, as cyst infections, abscesses, sepsis etc[22]. The same work analysed the prevalence of anaphylaxis during percutaneous drainage (consisting of Puncture, Aspiration, Injection and Reaspiration [PAIR]) in 769 subjects with hepatic cystic echinococcosis: the rate was of about 1%[22].

However, anaphylaxis can occur without any macroscopic rupture of the cyst[23-26], maybe caused by microscopic spillage. Moreover, patients can present a history of recurrent anaphylactic shock due to small, incomplete ruptures of hydatid cyst[24,26,27]. Among sudden deaths occurring in echinococcosis, anaphylaxis represents the cause in 20% of cases[28].

Anisakis (A.) simplex is a nematode found in fish and cephalopods. It infects humans accidentally when raw or undercooked fish contaminated with larvae is consumed. These larvae are in their third developmental stage (L3) and are developmentally arrested until ingested by sea mammals such as seals and dolphins, where upon they progress through two more developmental stages until adulthood is achieved. Usually within a few hours after the ingestion of a living worm, *A. simplex* causes an acute and transient infection that may lead to abdominal pain, nausea, vomiting, and/or diarrhea. Some patients develop syndromes simultaneously exhibiting clinical manifestations of allergy and infections after eating living parasites[29].

A. simplex is today widely known as cause of hypersensitivity reactions in subjects eating raw or uncooked fish[29] and in subjects working in contact to fish, as fishermen, fishmongers and fish-processing workers[30-32]. The relationship between allergic symptoms and *A. simplex* has been studied only in the late 1990s[33], from then reports are becoming more and more widespread. *A. simplex*. is one of the most frequent causes of anaphylaxis in Countries were the use of raw fish is common[30,34]. In the literature there are several case reports about anaphylaxis due to *Anisakis simplex*[34-42]. Among the anaphylaxis occurred in the Emergency Unit in Spain, *A. simplex* was responsible in 10.8% of cases and was more frequent in middle-aged subjects[43]. Another retrospective study reported *A. simplex* as the main cause of anaphylaxis due to hidden food allergens: 58% of cases[44]. In a group of 17 patients with specific IgE and positive skin prick test to *Anisakis simplex*, anaphylaxis has been reported in about one-third of patients[45]. Another work involving 23 patients with allergic reactions after seafood ingestion and with positive specific IgE and/or skin tests to *A. simplex* shower a rate of anaphylaxis of about 22% (5/23 patients)[46]. In Spain again, 25% of all the acute urticarial reactions due to *A. simplex*, progress to anaphylactic shock[47]. A Korean study on 10 subjects with anisakis allergy, 30% had anaphylaxis[48]. In an Italian research involving 19 allergy centres scattered throughout the Country, *A. simplex* was found cause of anaphylaxis in only one patient among 58 food-allergic patients which experienced at least 1 episode of anaphylaxis[49]. In the light of these findings, it appears that *A. simplex* is a common cause of IgE-madiated anaphylaxis even after exposure to very small doses: there are two reports on anaphylaxis occurred after skin prick-test with an *A. simplex* extract[34,50].

Taenia (T.) solium has a complex two-host life cycle. Human beings are the only definitive host and harbour the adult tapeworm (taeniasis), whereas both people and pigs can act as intermediate hosts and harbour the larvae or cysticerci. Taeniasis occurs only in the human host, after ingestion of undercooked pork infected with cysticerci. Cysticercosis is an infection with the larval form of the pork tapeworm, which resides in the small intestine of humans. After ingestion oncospheres are carried by the blood stream to various organs and lodge in the small blood vessels where they may or may not develop into viable cysts. Viable cysts form after 2-3 months[51].

The literature reports two cases of relapsing angio-oedema, hypereosinophilia and increased IgE levels maybe linked to cysticercosis, diagnosed by a strong positivity of ELISA for *T. solium* metacestodes on sera samples[52], a case of anaphylaxis following intraperitoneal rupture of

a cysticercosal cyst mimicking pelvic peritonitis[53], and a fatal case in a patient with *T. solium* taeniasis possibly due to the rupture of a unknown cysticercosal cyst[54].

Ascaris (A.) lumbricoides, and *Ascaris suum* are parasitic nematode (Family Ascarididae) infections of humans and pigs respectively. The human roundworm *A. lumbricoides* is one of the most common human parasites in the world. The spectrum of disease associated with *A. lumbricoides* infection is known as ascariasis[55].

It is known that *A. lumbricoides* may induce strong allergic reactions in infected individuals living in regions where transmission of infection is seasonal[6]. The presence of specific IgE to *A. lumbricoides* correlates with higher total IgE levels and higher prevalence of allergic rhinitis and asthma[56,57] (ascaris asma, Dold ascaris abstract). Today is known the cross-reactivity between Ascaris and mites, determined by several allergens including tropomyosin and glutathione-S-transferase[58] and cockroach[59]. However, the potential role of this cross reactivity on several aspects of allergy is still controversial. In literature it is reported a case of a woman who experienced two episodes of anaphylactic shock after eating oysters and white fish, respectively. The patient showed specific IgE to Ascaris. However, the patient showed specific IgE also to *Anisakis* spp, *Echinococcus* spp, oyster and prawns, so it was not possible to establish the specific etiological agent[60].

Conclusions

The risk of anaphylaxis in patients with helminthiasis can vary according to the pathogens, occurring more frequently during echinococcosis of after anisakis infestation and being extremely rare after other helminth infestations. However, physicians, allergist and parasitologist in particular, should be aware of a potential anaphylaxis caused by helminths.

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