

Anaphylaxis after *Argas reflexus* bite

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Key words: anaphylaxis; *Argas reflexus*; European pigeon tick; specific IgE; tick bite.

• DUE to the growing populations of wild urban pigeons, the natural hosts of parasitic ticks such as *Argas reflexus* (*Arg r*), there is an increased risk of allergic reactions to tick bites, presumably caused by the proteins secreted by the tick's salivary glands. Allergic reactions with symptoms

ranging from local manifestations at the bite site (itching, edema, and erythema) to

Two severe allergic reactions after nocturnal bites by the pigeon tick *Argas reflexus*.

severe systemic reactions (urticaria-angioedema and shock) have been reported in Europe (1–5). Here we report two patients who experienced severe systemic reactions after a bite by *Arg r*.

A 37-year-old man, living in a small town in central Italy near an old building, experienced two severe systemic reactions: the first one with urticaria-angioedema and the second one with cardiovascular failure and loss of consciousness. Both episodes occurred after nocturnal bites by a pigeon tick, identified as *Arg r*. Clinical history, skin prick test, and specific IgE measurements (CAP System, Pharmacia & Upjohn, Sweden) did not indicate any sensitization to common inhalants or food allergens. Total IgE (UniCAP) was 32 kU/l. Interestingly, the patient had experienced local reactions to venom from *Apis mellifera* (honeybee). Specific IgE was detected to *A. mellifera* (2.34 kU/l, CAP class 2) and to *Arg r* (5.73 kU/l, CAP class 3). In contrast, no specific IgE could be detected to allergens of

Dolichovespula maculata, *Vespa crabro*, *Vespula* spp., *Polistes* spp., *Solenopsis invicta*, *Aedes communis*, *Chironomus thummi*, *Tabanus* spp., *Blatella germanica*, and *Sitophilus granarius*.

The second patient was a 26-year-old man, also living in an ancient city of central Italy. He experienced several local reactions to bites of the pigeon tick, subsequently identified as *Arg r*, before developing two episodes of systemic reactions, with anaphylactic shock, to the tick bite. History, skin prick test, and specific IgE to common inhalant and food allergens were negative. Total IgE was 77.1 kU/l; specific IgE to *Arg r* was 1.27 kU/l (CAP class 2).

Severe, potentially life-threatening reactions in our patients confirm former cases of anaphylactic manifestations after *Arg r* tick bite (2, 3, 5). The patients described were living close to old buildings. Attics or roofs might have served as pigeon breeding sites, and ticks such as *Arg r* could have migrated to apartments in the neighborhood. Repeated tick bites can induce IgE-mediated sensitization to allergens of the soft tick even in subjects without atopic predisposition. Severe immediate-type systemic reactions, as suggested by the clinical histories, were directly confirmed by the presence of specific IgE to *Arg r* and indirectly by the absence of specific IgE (except for *A. mellifera* in one case) to other arthropods, including stinging insects, taxonomically different from *Arg r*. IgE-mediated cross-reactivity between proteins of the tick and other arthropods seems unlikely, in view of the specificity of the test employed to assess specific IgE to *Arg r*. Future efforts should be made to identify and use recombinant single allergenic proteins of *Arg r* to demonstrate more accurately an IgE-mediated reaction to pigeon tick bite.

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Anaphylaxis to E466

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Key words: allergy; anaphylaxis; carboxymethylcellulose; spontaneous case reports.

• CARBOXYMETHYLCELLULOSE (CMC), a hydrophilic derivative of cellulose, is widely used in both pharmaceutical and food industries as a suspending, emulsifying, and thickening agent. Despite its widespread use, severe adverse events such as anaphylactic shock are extremely rare. A search in the English and German

literature revealed only a few documented case reports of anaphylactic reaction after parenteral or oral application (1–4, 6).

The following cases were reported to the Swiss Drug Monitoring Centre (SANZ) by the allergy unit of the Department of Dermatology at the University Hospital, Zurich, Switzerland, where all allergy tests were performed.

Case no. 1. Because of serious achillodynia, the heel of a 54-year-old nonatopic woman was infiltrated with triamcinolone acetonide (Kenacort® 40 mg/ml) and lidocaine hydrochloride 2%.

Two cases of serious systemic reactions to carboxymethylcellulose (E466).

Within 5 min, she experienced generalized pruritus, urticaria, and angioedema, and she then collapsed. Under antiallergic treatment, all symptoms resolved within a short time.

The patient was referred to the allergy unit for allergologic investigation. After exclusion of allergy to local anesthetics and additives, Kenacort was tested. The patient showed a positive prick test to Kenacort A40 7.5 mg/ml 1:1 (+, >3 mm) and a strongly positive, intracutaneous reaction to 0.05 ml aqueous Kenacort 1:1000 (+++, >15 mm) and 1:100 (++++, >20 mm and pseudopodia), whereas six control patients showed no reaction to the same tests. With a positive scratch test to CMC sodium 7.5 mg/ml 1:1 (++, >5 mm), the substance causing the reaction could be isolated. No other components of Kenacort provoked a positive reaction (1 ml Kenacort: triamcinolone acetonide 10.5 mg, CMC sodium 7.5 mg, benzylalcohol 9.9 mg, and polysorbate 80 0.4 mg).

Case no. 2. A 41-year-old man received an intra-articular injection of 1 ml triamcinolone acetonide (Kenacort A 10 mg/ml) and 4 ml lidocaine 1% for inflammation of the left sacroiliac

joint. Fifteen minutes after the injection, he developed generalized pruritus and skin rash followed by a shock reaction with hypotension, generalized urticaria, and loss of consciousness. Resuscitation led to complete recovery.

Again, allergy to anesthetics was excluded. Prick tests to Kenacort A40 and to CMC sodium, both up to 1:2 dilution, were negative. But intracutaneous testing of the single components of Kenacort revealed sensitization to CMC (type I reaction). The test showed an intermediate reaction (+/–, <5 mm) to 0.05 ml CMC (0.75 mg/ml=1:10) and a significantly positive result (+, >5 mm) to 0.05 ml CMC (3.75 mg/ml=1:2). Intracutaneous testing of all other components of Kenacort in our patient and of CMC in control patients was negative.

These reports demonstrate the pattern of a rare but serious immediate allergic reaction (type I) to CMC. The first report of CMC as an allergen in patients dates from 1973 (1). More than 15 years later, well-documented cases were published describing reactions 15–30 min after parenteral application of steroids containing CMC (2–5). The reactions began with symptoms such as pruritus and urticaria, leading to hypotension and anaphylaxis. In 1997, an anaphylactic reaction due to orally applied barium sulfate containing CMC was published (6).

The beneficial features of CMC have led to its widespread use in the food industry. However, medical indications require caution in its application. Special attention should be given to allergic reactions associated with ingested CMC. Moreover, given the fact that CMC does not decompose biologically and accumulates in sewage sludge, substitutes for CMC are desirable where possible. Perhaps the reports of allergic reactions should be taken as an incentive to develop such substitutes.

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