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Allergy to gelatin

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Key words: allergy; gelatin; paresis.

● On 27 March 1998, a 28-year-old man was admitted to the neurosurgery clinic of the University of Messina (Italy) for bilateral back pain with a left prevalence, which had begun about 2 months before. Therefore, before admission, he underwent conventional anti-inflammatory and analgesic treatment without result, and acupuncture and physiotherapy with moderate results. A month before admission, in view of the accentuation of symptoms, the patient underwent magnetic resonance imaging and computerized axial tomography. These methods detected a median posterior hernia at the L3–L4 level. The consultant neurosurgeon advised surgery.

An operation by the L3–L4 left interlaminar approach was carried out on 7 April 1998. At 24 h after surgery, the patient

first complained of tingling of the lower limbs and, subsequently, paresis and complete bilateral deficit of foot dorsiflexion. Therefore, he underwent repeated magnetic resonance imaging of the lumbosacral spine, and this showed dural sac L3–L4 compression because of the edematous reaction of the soft tissues.

A subsequent allergy consultation led to the suspicion of an allergic reaction to a hemostatic gelatin sponge (Spongostan, Ferrosan, Denmark) placed at the end of surgery at the site of the wound. Therefore, the patient underwent disruption of the wound, cleansing of the phlogistic focus, removal of the Spongostan, and biopsy.

The biopsy examination, carried out on a specimen obtained from the dural sac compression zone, showed the presence of phlogistic infiltration, mainly by eosinophils, neutrophils, and histiocytes, together with chips of the gelatin sponge.

After removal of the Spongostan, there was a gradual and rapid improvement of the neurologic objectiveness, supported by courses of daily rehabilitative physiotherapy. The patient was discharged on 2 May 1998.

After 2 months, the patient returned for a checkup, during which patch tests were carried out with the International Contact Dermatitis Research Group (ICDRG) series (True Test, Pharmacia, Milan, Italy) and an extemporaneous preparation consisting of a dilution in vaseline of the gelatin sponge.

The patch tests were scored at 48 and 72 h by the 1+ to 3+ system recommended by the ICDRG. They proved to be highly positive for gelatin (+++) at 48 h, and negative for the other substances.

Absorbable gelatin sponges have been used for many years either as a wound-dressing material (1) or for transcatheter arterial embolization in the treatment of various disorders (2). In recent years, cases of immediate-type allergic reaction caused by gelatin present in the vaccines (3) or in

the recombinant human erythropoietin (4) have been reported in which specific IgE to gelatin was found in the sera of the patients.

It has also been shown that there is a possibility of delayed-type hypersensitivity to gelatin, consisting of granulomatous arteritis with massive eosinophil leukocyte infiltration subsequent to transarterial embolization therapy with a gelatin sponge (5).

We describe here an adverse reaction subsequent to the application of a gelatin sponge, at the end of surgery for a disk hernia, at the site of the wound. The clinical course, the biopsy findings, and the high positivity to the specific patch test suggest either a causative role of the gelatin or a pathogenic mechanism of delayed-type hypersensitivity. Our case represents the first report of lower limb paresis by dural sac compression related to an allergic reaction to gelatin.

Lastly, since the number of operations for disk hernia is very high and gelatin sponges are used frequently as hemostat, such an allergic reaction should always be taken into consideration.

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Selective immediate hypersensitivity to ceftriaxone

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Key words: ceftriaxone; immediate hypersensitivity; skin tests.

• CEFTRIAZONE is a third-generation cephalosporin used for serious infections. There have been very few reports of type I allergies (1), even though the frequency of hypersensitivity reactions to ceftriaxone is 3% (2).

We studied a 68-year-old man who had developed urticaria, dyspnea, shortness of breath, dizziness, tachycardia, and severe hypotension within 15 min after intramuscular administration of the first gram of ceftriaxone as prophylaxis for a cataract operation. After treatment with subcutaneous epinephrine, 6-methyl-

prednisolone, and chlorphenamine intravenously, symptoms resolved completely in 12 h.

Ten months later, we skin-tested the patient with penicilloylpolylysine (Allergopen, Reinbek, Germany), minor determinant mixture (Allergopen), benzylpenicillin, ampicillin, and amoxicillin, as well as ceftriaxone, cefuroxime, cephalothin, ceftazidime, cefotaxime, and cefamandole, as previously described (1). All cephalosporins were used at a concentration of 2 mg/ml in 0.9% NaCl. The patient responded positively to prick testing with ceftriaxone (wheal 7×8 mm). Prick and intradermal histamine tests produced wheals with maximum diameters of 8 and 12 mm, respectively. Intradermal tests with the above cephalosporins were negative in 40 healthy control subjects, 10 of whom had previously tolerated ceftriaxone.

Assays (UniCAP[®] specific IgE; Pharmacia & Upjohn, Uppsala, Sweden) for specific IgE to penicilloyl G, penicilloyl V, ampicilloyl, amoxicilloyl, ceftriaxone, cefuroxime, cefaclor, cephalixin, cefotaxime, and cefatrizine were negative (<0.35 kU/l). All the cephalosporin assays except cefaclor were experimental prototypes.

Single-blind, placebo-controlled oral challenges with penicillin V (1 000 000 IU), ampicillin (1 g), cephalixin (500 mg), cefaclor (500 mg), and cefixime (400 mg) were also performed (each on a different day). Increasing amounts (0.01, 0.1, and 1.0)

of the therapeutic doses, indicated in parentheses, were administered at 1-h intervals. No reactions were observed to the challenges.

Our clinical findings and the positive prick test with ceftriaxone suggest that the patient developed type-I hypersensitivity.

We could not show specific IgE by RAST. It should be considered that we used experimental prototypes, and that cephalosporin allergenic determinants have not been fully identified (3). Furthermore, in our case, the assays were performed 10 months after anaphylactic shock and it is possible that the IgE titer had meanwhile fallen below the cutoff value, as observed with penicilloyl IgE antibodies (4).

We had previously diagnosed a case of immediate hypersensitivity to ceftriaxone (1). This subject showed skin-test and RAST positivity to both ceftriaxone and cefotaxime, which have identical (2-amino-4-thiazolyl) (methoxyimino) acetyl side-chains; thus, his reaction was probably caused by selective sensitization to side-chain determinants, as confirmed by skin tests and *in vitro* assays, as well as negative challenge results with other β -lactams.

The present subject displayed skin-test and RAST negativity to cefotaxime as well as to the other drugs and tolerated challenges with other β -lactams. Thus, the patient's reactivity could have been to the entire ceftriaxone molecule (Fig. 1), as previously demonstrated with cefaclor (3).

To our knowledge, this is the first case in

Anaphylactic shock presumed to be allergic.

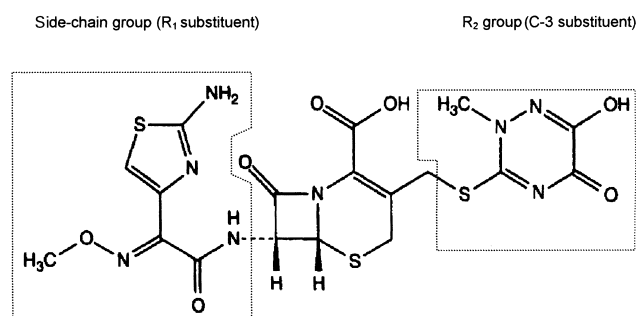


Figure 1. Chemical structure of ceftriaxone.