Drouet and coll (5) described a pork-cat syndrome or crossed allergy between pork meat and cat epithelia in 1994. The pattern shown by our patient differed slightly from previous reports. First, she had no asthma attacks due to cats. Secondly, she developed urticaria, rhinitis and asthma when exposed at work to aerosols from evaporated pig-gut soaking water containing albumin, γ -globulin and a 26 kDa protein. Albumin has been reported to be allergens for food allergies to pig gut and γ -globulin for allergy to beef, but none have ever been reported to be aeroallergens. In conclusion we demonstrated that our patient had occupational asthma due to inhalation of pig albumin, γ -globulin and a 26-kDa protein contained in evaporated pig-gut soaking water.

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Multiple drug intolerance including etoricoxib

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Key words: adverse reactions; drug allergy; etoricoxib; niflumic acid; NSAIDs.

Acetylsalicylic acid (ASA) and other nonsteroidal anti-inflammatory drugs (NSAIDs) are among the drugs that most commonly cause adverse reactions, and in asthmatics these reactions can occur from 4.3 to 10.9% (1, 2). In these patients, cyclooxygenase-2 (COX-2) inhibitors, when indicated, have been proposed as a therapeutic alternative although, even with very recently marketed drugs, such as rofecoxib, positive challenge was observed in about one-third of the patients with previous NSAID hypersensitivity reactions (3); nevertheless, better results were reported by other authors (4). Severe selective allergic reactions to COX-2 inhibitors have also been described (5).

Etoricoxib is a recently introduced member of the COX-2 very selective inhibitors class of NSAIDs. It is available in tablets of 60, 90 and 120 mg and recommended for osteoarthritis and chronic low back pain, rheumatoid arthritis and for acute gouty arthritis and primary dysmenorrhoea (6–7).

There are no previous reports in the literature, to our knowledge, of adverse reactions to etoricoxib.

The authors report a case of a 38-year-old female, with a previous history of

adverse reaction to different NSAIDs, who required an elective arthroscopy to her left knee and had no alternative drug for post-surgery pain relieve. The patient also has asthma and rhinitis and a family history of anaphylaxis to a general anaesthetic.

Our patient had a previous diagnostic of ASA sensitivity evidenced by several episodes of upper airway obstruction in the sequence of ASA ingestion. These episodes required treatment with intravenous corticosteroids for their resolution.

At the age of 23, attempts have been made to introduce nimesulid and acetaminophen with poor results. The patient developed dry cough, nausea and indisposition, which were controlled with H₁-antihistamines.

Three years ago the patient had an episode of upper airway obstruction, face and hand oedema and generalized pruritus following administration of intramuscular diclofenac.

Since then she has been avoiding all NSAIDs.

About 7 months ago she developed generalized urticaria and face angio-oedema in the sequence of the ingestion of 12.5 mg of rofecoxib, prescribed for pain relief after her first arthroscopy to the left knee. This reaction was also controlled with H₁-antihistamines. At present she needs a second arthroscopy and has no available medication for pain relief.

The patient was referred to our department where we decided to perform a single-blinded placebo-controlled challenge with 60 mg of etoricoxib. We started with the administration of placebo followed by 20 and 40 mg of etoricoxib, at 30-min intervals.

About 1½ h after the beginning of the challenge, the patient started to feel nauseated, trembling and referred abdominal pain and headache. The symptoms remitted after treatment with 20 mg of ebastine by mouth.

Another single-blinded placebo challenge was tried with 50 mg of tramadol 1 week later and the patient developed nausea and strong eructation and subsequently vomiting, symptoms that remitted shortly after the administration of 20 mg of ebastine. As the patient had an arthroscopy scheduled for the following day, we

A case of a placebo-controlled positive challenge to etoricoxib with tolerance to niflumic acid

tried the administration of topic niflumic acid on the volar surface of the forearm, without any adverse reactions. Then another placebo-controlled oral trial was performed, with a cumulative dose of 125 mg of niflumic acid, which was negative, allowing the use of the drug.

This case illustrates that not even the most recent NSAIDs are free from adverse reactions and that sometimes other old alternatives must be considered.

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Vesicular and bullous eczema in response to intravenous immunoglobulins (IVIG)

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Key words: IV immunoglobulin therapy; pompholyx.

Standard (0.4 g/kg/day) and high dose (2 g/kg/day) intravenous immunoglobulins (IVIG) are used for therapy of several dermatological and neurological autoimmune diseases (e.g. dermatomyositis, pemphigus vulgaris, multifocal motor neuropathy) (1–3). Cutaneous adverse effects to IVIG have only rarely been reported and encompass case reports on eczema, alopecia, erythema multiforme, and anaphylactic reactions; the latter, however, is usually explanatory by a deficiency in IgA and can be prevented by measuring IgA levels prior to treatment.

We want to draw attention to a peculiar cutaneous side effect which may be more frequent than reported as it resembles common vesicular eczema (pompholyx, dyshidrotic eczema), and which might lead to undue interruption of therapy for fear of a more severe bullous drug reaction.

A 33-year-old patient with multifocal motor neuropathy received 0.4 g/kg/day IVIG for 5 days. One day after completion he developed a vesicular eczema at the lateral aspect of his fingers, which within 3 days extended to volar aspect of fingers and to both palms while vesicles progressed into bullae. He also developed a slightly scaling erythema on the face and capillitium. When after 10 days treatment with topical corticosteroids was initiated bullae started to empty clear fluid and the lesions showed extensive desquamation and healed within 1 week.

For a second course of IVIG the patient received systemic antihistamines and remained under close observation. One day after completion he again

developed vesicular eczema, which however, ran a markedly milder course with fewer lesions and without bullae. Local corticosteroids were not necessary. IVIG were then continued and during the subsequent seven courses the patient developed only very mild vesicular reactions. When he stopped taking antihistamines after the fourth course, there was slight aggravation of the vesicular reaction, but it was transient and did not recur after the following infusions even though antihistamines were not resumed.

For allergologic work-up after the severe bullous reaction we performed (epicutaneous) patch testing as well as prick testing with the corresponding IVIG preparation and preservatives such as chlorocresol and sulfite. All reactions but controls remained negative (patch test up to day 7).

There are only few reports on vesicular eczema or pompholyx after treatment with IVIG: 3 of 23 patients (4) and 1 of 10 patients with IVIG for neurological diseases (5). Extension of vesicular eczema to arms, breast and head area was described for only few of these cases.

The pathogenesis of this adverse effect is not well understood (4, 5). It may be a unique side effect of IVIG therapy, as it has not been reported for other medications (4). It has been suggested to occur only in those patients, which show very high plasma IgG concentrations directly after administration of IVIG (> 3000 mg/dl) (5), but this was not the case in our patient who showed only slightly elevated IgG (11.6–19.7 g/l). However, he presented with remarkably high IgE levels (1806 U/ml) already before IVIG treatment, which were not associated with lymphoma, other myeloid disorders or atopic diathesis. Since imbalances in plasma levels of immunoglobulins are known to be associated with conditions resembling atopic eczema (6) we speculate that IVIG therapy may result in development of dyshidrotic eczema in patients with a certain, transient imbalance in serum immunoglobulins.

If IVIG therapy is to be continued in these cases, companies recommend prophylactic administration of corticosteroids and antihistamines. However, according to our experience and to the few published reports we suggest that vesicular eczemas following IVIG