

of positive epicutaneous tests to corticosteroids are estimated between 0.2% and 5.0% in patients with adverse reactions to them (2). Urticaria, angioedema and/or anaphylaxis are seen less frequently in such cases, almost always appearing after systemic intake of the corticosteroid.

A 64-year-old Caucasian woman was diagnosed with allergic alveolitis caused by parakeet feathers, which improved clinically with intravenous corticosteroid (methylprednisolone). She was discharged with a domiciliary tapering treatment of deflazacort at an initial dosage of 60 mg/day, which was to be reduced progressively. Thirty days after the beginning of treatment she showed generalized itchy blotches together with lip edema. At that point the patient was mistakenly taking a deflazacort dose of approximately 120 mg/day. Two antihistamines (hydroxysine 25 mg t.i.d. and ebastine 10 mg q.d.) were added to the treatment without any improvement. Consequently the deflazacort was substituted by prednisone and the symptoms disappeared immediately.

Cutaneous tests with deflazacort, 6-methylprednisolone, hydrocortisone, and dexamethasone were performed, following the usual procedural recommendations and using non-irritating doses (3,4). All of the prick tests were negative, and the intradermal reactions were only positive with deflazacort. The epicutaneous tests were positive with deflazacort (at 1% in ethanol with erythema-edema and vesicles at 48 h) and with hydrocortisone (1% in ethanol with erythema-edema at 48 h).

The oral provocation test with 30 mg of deflazacort was positive, with the immediate appearance of symptoms the same as the initial episode. The patient tolerated well all the other corticosteroids.

Deflazacort is a prednisolone-derived glucocorticoid for oral use, introduced recently to the market. It is distinguished by a 16 α -17 α -2-methyloxazolinic ring. It is a prodrug having rapid gastrointestinal absorption, being hydrolyzed to its active metabolite: 21-OH deflazacort. Hypersensitivity reactions to this drug are extremely rare, with only one case reported of delayed hypersensitivity (4), and we

have not found any cases with urticaria, angioedema or anaphylaxis.

We describe the first case of urticaria-angioedema by deflazacort, confirmed by deflazacort-positive epicutaneous, intradermal reaction and provocation tests. In this case, we have not found clinical cross-reactivity to any other corticosteroid tested.

*Carmen Mogio Gómez
C/Jacinto Benavente, 8
21800 Moguer
Huelva
Spain

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Oral desensitization to 5-ASA

S. Varela*, M.S. Díez, C. Gonzalez,
C. Gonzalez de la Cuesta, L. Arenas,
R. Feijóo, M. Menéndez

Key words: 5-aminosalicylic acid; anaphylaxis; desensitization; hypersensitivity; mesalazine.

● The 5-aminosalicylic acid (5-ASA) is the active component of sulfasalazine (5-ASA, sufapyridine), structurally related to the salicylates. It is a well established treatment for inflammatory bowel disease. Pure 5-ASA preparations were developed to deliver the active ingredient of sulfasalazine while minimizing adverse effects (1).

Hypersensitivity to 5-ASA is infrequent and a few cases of successful oral desensitizations have been reported (2–4). We report a case of successful 3 days oral desensitization in a patient with anaphylaxis.

The patient was a 49-year-old man with a 3-year history of Crohn's disease. He had been taking 1.5–3 g of daily oral 5-ASA (Claversal®), for 3 years. One night, he developed a generalized

A case of anaphylaxis successfully treated

urticaria, facial angioedema, shortness of breath, vomiting and abdominal pain. He was attended to in an emergency room. Ten hours before, he had taken 50 mg of diclofenac for back pain, and 3 h before 1 g of 5-ASA. After this, the 5-ASA was discontinued. He didn't have a past history of ASA or AINE hypersensitivity. Skin-prick test with a panel of inhalative and food allergens (ALK Abello, Spain), and 5-ASA 10 mg/ml (Quintasa® enema) were negative. After obtaining written informed consent, a single-blind

Table 1. 5-ASA desensitization protocol

Dose	Time	Milligrams
Day 1		
		10 mg/ml *
1	9:00	1 mg=0.1 ml
2	9:20	2 mg=0.2 ml
3	9:40	4 mg=0.4 ml
4	10:00	8 mg=0.8 ml
5	10:20	15 mg=1.5 ml
6	10:40	30 mg=3 ml
7	11:00	60 mg=6 ml
		100 mg/ml *
8	11:20	100 mg=0.1 ml
9	11:40	150 mg=1.5 ml
10	12:00	200 mg=2 ml
Day 2		
1	9:00	200 mg=2 ml
2	9:30	300 mg=3 ml
3	10:00	500 mg=1 tablet**
Day 3		
1	9:00	1 g=2 tablets**
2	12:00	1 g=2 tablets**
3	22:00	1 g=2 tablets**

*. Solutions of pure 5-ASA were prepared by the pharmacy in our hospital

**. Claversal® 500 mg

placebo-controlled oral challenge test with paracetamol and 5-ASA were performed on different days. For testing, 250 mg, 500 mg and 1 g of paracetamol tablets was administered at 1-h intervals, with good tolerance. Sixty minutes after the first dose of 5-ASA (125 mg, 1/4 tablet of Claversal), he developed pruritus, facial eritema, and hives on his arms. He was immediately treated with 0.5 cc of subcutaneous epinephrine, oral metilprednidsolone, and cetirizine, improving within 1 h. Then Claversal was prohibited. After stopping the 5-ASA, the patient's Crohn's disease worsened. He presented several episodes, one of them required hospitalization. On the whole, we decided to carry out a 3-day desensitization protocol (Table 1). The patient was admitted to the hospital during this time. Finally, he tolerated 3 g of 5-ASA per day, without adverse reaction during desensitization procedure.

Adverse effects to 5-ASA agents are infrequent but may include nausea, dyspepsia, and headache. Uncommon hypersensitivity reactions, including worsening of colitis, pancreatitis,

pericarditis, pneumonitis, hepatotoxicity, fever, exantema, have been reported (1,4). Turunen et al. (1), found that 5-ASA tolerance in patients with previous hypersensitivity to sulfasalazine is about 80%, but patients with severe reactions from sulfasalazine should be treated with caution with 5-ASA (2). This patient developed an anaphylactic reaction caused by 5-ASA, as proved with the oral challenge. We couldn't demonstrate an IgE mechanism. We didn't carry out oral challenge with ASA or NSAIDs because of the risk of anaphylaxis and considered not to be essential to the patient. There are a few reports of desensitization to 5-ASA, by oral routes in 20 and 32 days, and rectal routes (2-4). Because our patient developed an immediate reaction we decided to carry out a 3-day desensitization protocol, obtaining a very good result.

*Complejo Hospitalario de Ourense
Unidad de Alergología
Servicio de Digestivo
Rúa Ramón Puga 52-56
32005 Ourense

Spain

E-mail: susavar@arrakis.es

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