RESEARCH LETTERS

Exfoliative erythrodermia induced by pantoprazole

To the Editor,

Although proton pump inhibitors are widely used, allergic reactions to this group of drugs are rarely reported. In this paper we present the case of a patient who developed a delayed reaction to pantoprazole. A 35-year-old female patient complained of generalised erythema with intense itching and desquamation for three weeks before consulting (Fig. 1). The rash was first noticed five days after the initiation of treatment with topical diltiazem, polyethylene glycol 3359 (Milax®), Alevian Duo® (simethicone/pinaverium bromide) for anal fissuring, and pantoprazole for gastritis. Past medical history disclosed appendectomy, chickenpox, and chronic rhinosinusitis.

Physical examination showed xeroderma, widely distributed erythema and generalised desquamation, more pronounced on the hands, and lower limbs (Fig. 2). The diagnosis of exfoliative erythrodermia likely related to drug allergy was made and symptoms resolved completely after treatment with oral prednisone, topical corticosteroids (mometasone furoate and 0.1% methylprednisolone aceponate), and oral antihistamines (desloratadine, cetirizine). No other underlying clinical conditions, including autoimmune, allergic or infectious diseases were present, as deducted from patient questioning and physical examination. Routine laboratory investigations, including haematology, blood chemistry, urine and stool examination were within normal limits. Histopathological examination of the skin was not performed.

Six weeks after the first visit patch tests with drugs, with reading after 72 and 96 h, were performed. Milax®, Alevian Duo® and pantoprazole 10% were applied in white petrolatum. Tests were negative for 2% diltiazem ointment, Alevian Duo® and Milax®. The test was positive for pantoprazole, showing local erythema and oedema at 72 and 96 h (Fig. 3). Patch tests with all drugs mentioned above were negative in 12 voluntary individuals who did not have a history of drug allergy. Avoidance of pantoprazole and substitution with lanozoprazole were recommended.

IgE-mediated allergic reactions to proton pump inhibitors, especially anaphylaxis and urticaria, have been occasionally reported by various investigators.1-4 In regard to delayed reactions, Mockenhaupt et al. observed nine

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Figure 1  Erythrodermic and exfoliative dermatitis in a 35-year-old female patient with delayed allergy to pantoprazole.

Figure 2  Exfoliative erythrodermia induced by pantoprazole. Observe the extensive skin desquamation on hands and lower limbs.
cases of pantoprazole-induced severe cutaneous reactions of the type Stevens–Johnson syndrome and toxic epidermal necrolysis.\textsuperscript{3} Isolated reports of lichenoid eruption, acute interstitial nephritis, neutropenia, and vasculitis attributed to proton pump inhibitors, including pantoprazole, have also been published.\textsuperscript{6–10}

To the best of our knowledge, this is the first report of a case of exfoliative erythrodermia induced by pantoprazole that was confirmed by drug patch testing. Although a positive patch test does not constitute an absolute diagnostic criterion for establishing drug responsibility in this particular case, it is interesting to mention that the test was negative in non-allergic control subjects, and was also negative for all other drugs tested in the present patient. Therefore, an irritant reaction to the patch test seemed less likely to be occurring. Even though this diagnostic tool has not been adequately standardised, it is useful for a more precise diagnostic evaluation especially in patients who are receiving multiple medications, as was the case in the present report.

With regard to the management, since in patients with immediate reactions to proton pump inhibitors cross-reactions between omeprazole and pantoprazole but not with lansoprazole are observed, we recommended the use of lansoprazole, which has been tolerated by this patient during the short three-month follow up period after its initiation.

References


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Resolution of IgE-mediated fish allergy

To the Editor,

Fish is a common allergen source, being one of the most frequent causes of IgE-mediated food allergy in children, in many countries. Severe clinical reactions to multiple fish are usual.\textsuperscript{1} This is mainly justified by their major allergens, parvalbumins, which are thermo-acid resistant proteins (maintaining its structure when cooked or submitted to a pH as low as 2.75 in the digestive process) and ubiquitous in various fish (although expressed in different levels).\textsuperscript{1,2} Fish allergy usually manifests during early childhood and is considered mainly as persistent, likely to be lifelong.\textsuperscript{3} Despite this, there has been one report of fish allergy outgrown in adulthood.\textsuperscript{4} A pattern of fish allergy in which evolution tends towards tolerance of all fish over the years has also been described.\textsuperscript{5} These fish allergic patients are typically young children, who tolerate some fish (Tunidae and Xiphiiidae families), despite reacting to other species.\textsuperscript{6} However, when several severe clinical reactions to various fish from different taxonomic families have occurred for a long period of time or if the patient is highly sensitised to multiple fish, a complete fish exclusion diet is usually advised for life. In this paper, we present and discuss a case of long-term follow-up successful management of a patient with lasting and severe fish allergy that has resolved.