Brief communications

Fixed drug eruption induced by mepivacaine

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Less than 1% of adverse reactions to local anesthetics are allergic in nature.[1] In particular, two types of immunologically mediated reactions to local anesthetics are commonly described: (1) IgE-mediated reactions (Gell and Coombs type I) and (2) allergic contact dermatitis (Gell and Coombs type IV).

Although extremely rare, IgE-mediated reactions do occur in response to both amide- and ester-group agents.[2, 3] They may be characterized by hives, angioedema, bronchospasm, rhinorrhea, or, in severe cases, by shock. Topical anesthetics are not only widely used but are also potent contact sensitizers. They produce more than 80% of the total allergic reactions to local anesthetics.[4] They are characterized by contact dermatitis.

On the basis of limited patch testing data, it has been postulated that ester-group anesthetic agents cross-react with each other, but not with the amide-group agents. This postulate appears to hold true with regard to contact hypersensitivity but has never been adequately studied with regard to IgE-mediated hypersensitivity.[5]

Fixed drug eruption is characterized by an isolated erythematous macular pruritic lesion. An acute phase is followed by desquamation and hyperpigmentation. The lesion usually appears in the oral or genital mucous membranes but occasionally arises on the face. With re-stimulation, lesions always appear in the same place. The agents usually involved are: phenolphthalein, tetracycline, sulfonamides, barbiturates, and nonsteroidal antiinflammatory drugs.

CASE REPORT

We present a case of fixed drug eruption caused by mepivacaine. A 22-year-old woman had a personal history of cholinergic rhinitis, and her mother had a history of atopic asthma. She had, on four occasions after oral surgery, itching and violet erythema on the upper right buccal commissure, lower lips, left buccal commissure, and upper right eyelid. The lesions appeared 20 minutes after administration of Scandinibsa (mepivacaine NaCl) and had a maximum expression at 24 hours after injection of the drug. The lesions disappeared 12 to 14 days later with desquamation, leaving a slight brownish hyperpigmentation. The dose of drug administered was not known by the patient.

Six months later, an allergy evaluation was undertaken. We developed an intradermal test with commercial Scandinibsa 2% S/A (Braun Medical SA). (Aqueous solution of mepivacaine 2% in NaCl is a preparation that does not contain parabens.) We used 1:1 and 1:10 dilutions in saline solution, and 0.02 ml was injected (2 mg and 0.2 mg, respectively). Negative results were obtained.

We then carried out a double-blind, placebo-controlled challenge test. We injected 0.2 ml (20 mg) of Scandinibsa subcutaneously. Twenty minutes later, the itching and erythema appeared in the same places and with the same characteristics as the patient had experienced on the previous four occasions. We administered dexchlorpheniramine 5 mg intramuscularly, 6-methylprednisolone 125 mg intramuscularly and deflazacort 30 mg orally on 2 consecutive days; and the lesions disappeared by the end of the second day. This treatment aborted the progression of symptoms.

One week later, we carried out an intradermal test with commercial lidocaine 2% S/A, (Braun Medical SA). (Aqueous solution of lidocaine 2% in NaCl does not contain parabens.) We used 1:1 and 1:10 dilutions in saline solution with negative results.

One month after the administration of Scandinibsa, we carried out a double-blind, placebo-controlled challenge test with another anesthetic. We injected commercial lidocaine in therapeutic doses subcutaneously, and the patient had no reaction. She has since tolerated lidocaine.

DISCUSSION

Preservatives are an integral component of most local anesthetic preparations. Parabens, including methylparaben and propyl paraben, are the preservatives most widely used. Because of concern that parabens may account for some allergic reactions, paraben-free preparations have become commer-

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cially available. In our case, the drug that induced the reaction and the compound that was well tolerated were paraben-free.

The exact mechanism that produces fixed drug eruptions is not well known. The short interval of initiation of the symptoms is not common; usually in fixed drug eruptions, symptoms appear after an interval of 6 hours or later, although the maximum expression of skin lesions takes a period of 24 to 48 hours or longer after administration of the drug. However, unreported evidence from our group indicates that with other drugs, such as sulfonamides and erythromycin, initiation of itching and erythema may occur as early as 20 minutes after drug intake. Quantitation of histamine in blood and urine has not shown evidence of the release of this mediator.

We believe this to be the first reported case of a fixed drug eruption induced by mepivacaine in a patient with good tolerance to lidocaine.

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Effective long-term α-interferon treatment for hypereosinophilic syndrome

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Hypereosinophilic syndrome (HES) is a disorder characterized by unexplained severe (>1.5 × 10^9/L) eosinophilia, lasting more than 6 months, often with multiple organ damage.[1] The traditional treatment for HES involves corticosteroids and cytotoxic agents (e.g., hydroxyurea, vincristine, etoposide); but some authors have recently described, in cases of HES not responsive to those treatments, the successful use of α-interferon (α-IFN).[2-4] We used α-IFN as initial treatment for a case of HES and achieved good long-term clinical efficacy with no side effects.

Abbreviations used
FEV_1: Forced expiratory volume in 1 second
HES: Hypereosinophilic syndrome
IFN: Interferon

CASE REPORT

A 24-year-old man first came to us for treatment in July 1991. The clinical history was not significant. None of the drugs that could be responsible for pulmonary eosinophilia had been administered. The disease suddenly manifested in February 1991 as Löffler's syndrome with severe asthma and eosinophilia. Bronchoscopy performed at that time showed an inflamed mucosa, and eosinophils represented more than 40% of the cells in the bronchoalveolar lavage fluid. Steroid treatment (prednisone 50 mg/day) improved the clinical condition, but any subsequent attempt to taper the dose of the drug led to an increase in eosinophil count and severe asthma symptoms. The patient received 25 mg of prednisone daily until admission to our department.

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