

Adverse reaction to lidocaine

ADVERSE reactions to local anesthetics (LA) have been widely described, and various mechanisms have been proposed to explain such reactions (1). Most of them are thought to be toxic, eliciting cardiovascular and neurologic symptoms (2-6). Lidocaine is one of the local anesthetics most frequently used in medicine. In this paper, we report on a bizarre adverse reaction to lidocaine.

■ A 16-year-old woman without obvious disease was referred for allergologic investigation, in order to select at least one LA which could be safely used. She had had an allergic reaction 1 year before consulting. The reaction consisted of perioral paresthesia and vomiting, and she became dizzy and confused during the next 24 h. The reaction took place shortly after the administration of an unknown LA agent, which was applied for the removal of a molar.

Skin prick tests were performed with three different undiluted LA: Lidocaine Palex (5% lidocaine), Scandinibsa (2% mepivacaine), and Procaine (1% procaine) with negative result. Intradermal tests were carried out with a 1/100 dilution of each LA tested. As this test yielded a negative reaction, each LA was subsequently administered as a subcutaneous challenge test (single-blind), carried out with increasing doses injected in the lateral side of the patient's arm, with her informed consent and according to the method of Schatz (7).

Twenty minutes after the 0.05-mg dose (0.1 ml of 1:100 dilution), lidocaine elicited perioral paresthesia, nausea, vomiting, vertigo, dizziness, mild agitation, mild drowsiness, euphoria, and logorrhea. Blood pressure, pulse rate, and other data of physical examination were normal.

Challenge tests with mepivacaine and procaine were carried out without adverse reactions.

To confirm that lidocaine caused the reaction, we carried out a placebo-controlled, double-blind, subcutaneous rechallenge test with lidocaine, according to the protocol previously reported. This time, the 0.1-ml dose (1:100) also elicited the same symptoms (perioral paresthesia, nausea, vomiting, dizziness, mild agitation, mild drowsiness, euphoria, and logorrhea), at the same intervals (20 min) as

we described above. The result of the placebo challenge (saline solution) was negative.

■ Lidocaine hydrochloride, an aminoacylamide, is widely used as a local anesthetic and antiarrhythmic agent. All local anesthetics have the potential to cause central nervous system irritability resulting from blockade of cerebral cortical inhibitory pathways. The major toxic effects of lidocaine involve the central nervous system. Severe and even lethal intoxication has been described after administration of high doses of lidocaine (2-6). Toxic manifestations first appear at a serum lidocaine concentration of 5 mg/l and worsen progressively at higher levels. Early symptoms are dizziness, drowsiness, tinnitus,

and perioral paresthesia. More severe symptoms are disorientation, delirium, convulsions, coma, and cardiorespiratory arrest. Cardiotoxic effects include depression of automatic activity and conduction distur-

*A nonallergic,
neuropsychiatric
reaction confirmed by
single-blind
provocation.*

bances causing heart block, ventricular tachyarrhythmia, and asystole.

Lidocaine is mainly eliminated by the liver. The major metabolites, 4-hydroxy-2,6-dimethylalanine, monoethylglycinexylidide (MEGX), and glycinexylidide (GX), are probably responsible for the central nervous effects of lidocaine. Animals with experimentally produced hepatic damage are much more susceptible to the toxic action of lidocaine.

In our case, the challenge tests confirmed that the neuropsychiatric symptoms were due to lidocaine. In view of the small dose of lidocaine that elicited the reported reaction, serum lidocaine concentrations were not determined. We think that an idiosyncratic but not toxic mechanism was the most likely cause.

Another important fact in this case is that this patient reacted to no other LA agents belonging to the amide group (mepivacaine) nor to procaine (with similar chemical structure), a fact which suggests that no cross-reactivity existed among these agents.

Key words: adverse reaction; lidocaine; local anesthetics; neuropsychiatric symptoms.

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Contact dermatitis to chlorprothixene

WE report the case of a 24-year-old nurse who complained of acute, intermittent skin reactions appearing periorbitally and on the cheek and hands. She was first seen in 1992, 1 year after starting work on a psychiatric ward and 6 months after the first symptoms. The symptoms improved during weekends and holidays. The previous year, the ward had been redecorated and so she was initially thought to have contact allergy to the paints used and their ingredients. She had no history of atopic disease. When the patient was first seen, she had already undergone treatment with local corticosteroids. We could see only folliculitis of the face, but the history was typical of airborne contact dermatitis.

A skin prick test panel which included the major airborne allergens and the ingredients of the paints used to redecorate the ward, and also patch testing with the European standard, the drug, and the disinfectant and preservative battery (all Fa.Hermal, Reinbeck, Germany) showed no positive skin reaction except to paraphenyldiamine, a reaction which was not clinically relevant. A high total IgE level with 1250 U/ml was shown without any history of airborne type I allergy or evidence of specific IgE.

Scratch test with neuroleptics belonging to the phenothiazines, the thioxanthenes, and the chemically unrelated butyrophenones, which had all been handled by the patient, showed no acute reactions,

Occupational allergy to a neuroleptic drug in a nurse working on a psychiatric ward.

but a strong reaction to chlorprothixene occurred after 24 h with additional reactions in the face and the neck, lasting 3 days. Patch testing was conducted with all the drugs the nurse handled (phenothiazines, thioxanthenes, butyrophenones, quinine sulfate, clonidine, and calcium dobesilate). No positive skin reaction was observed. Chlorprothixene was tested as syrup and coated tablets. Both showed strong reactions after 24 h, lasting 3 days. Control subjects did not show dermatitis after testing with chlorprothixene.

The nurse, after moving to another ward on which chlorprothixene was not used, was shown to be free of symptoms. After handling chlorprothixene again by mistake, she revealed skin reactions after a few minutes.

On the basis of clinical history and patch testing, the diagnosis of contact allergy due to occupational contact with chlorprothixene syrup was made.

Chlorprothixene belongs to the thioxanthenes, which are tricyclic antipsychotic drugs chemically related to the phenothiazines. This kind of neuroleptic has sedative, anxiolytic, antidepressant, antiemetic, antihistaminic, and anticholinergic effects. After widespread use of these antipsychotic agents in the mid-1950s, there were reports of frequent side-effects in patients, not only of the anticholinergic type but also of skin reactions (1, 2). Some authors estimate that skin reactions to psychotropic drugs are twice as frequent as those caused by most other drugs (3).

In particular, Chlorpromazine caused allergic dermatitis and even phototoxic and photoallergic skin reactions (1, 2, 4). In addition, eczematous reactions of the contact type were described, especially in health-care workers (1).

We report on a nurse who revealed contact dermatitis after 6 months of handling chlorprothixene, especially chlorprothixene syrup. She showed not only skin reaction at the contact site but also airborne contact allergy on the face and neck. To our knowledge, this has only once been previously reported with chlorprothixene (5). Cross-reactions with phenothiazines or group reactions with other thioxanthenes could not be shown in our patient, and there was also no reaction to the butyrophenones used. There was no indication of photosensitive dermatitis. After the cause of the patient's

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