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Selective severe anaphylactic reaction due to ketorolac tromethamine without nonsteroidal anti-inflammatory drug intolerance

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To the Editor:

Ketorolac tromethamine (Toradol) is a nonsteroidal anti-inflammatory drug (NSAID) with analgesic properties that is widely used as an alternative to opioid in the control of moderate pain. It is particularly useful in the emergency department. Allergy-like reactions to Ketorolac have been rarely reported despite its increased use as the first injectable NSAID approved for short-term pain management.^{[1](#) [2](#)}

We report the case of a 60-year-old woman who reported urticaria accompanied by laryngeal edema, cough, and breathing difficulties, followed by hypotension and a loss of consciousness 5 minutes after oral intake of ketorolac 10 mg. She had no personal or family history of atopic disease, chronic sinusitis, nasal polyps, or other drug sensitivity. She was not taking any concomitant therapy at the time of her adverse reaction.

Several months after the acute episode, the patient was referred to our allergy clinic for etiologic diagnosis. After obtaining the patient's written consent, we performed intradermal testing with ketorolac. A strong positive dose response reaction starting at a dilution of 0.01 mg/mL was observed (Table I).

TABLE I. Results of skin testing

Serial ketorolac dilution (mg/mL)	Wheal/flare diameter (mm)	
	Skin prick test	Intradermal test
0.001		No reaction
0.01		No reaction
0.1		No reaction
1		Erythema
		26 with pseudopods

Saline solution produced a negative result, and a histamine 1 mg/mL epicutaneous prick test resulted in an 8-mm wheal with flare. Three healthy donors had negative skin test responses to ketorolac. Increasing doses of aspirin, diclofenac, acetaminophen, meloxicam, and nimesulide, reaching therapeutic doses, were orally administered to the patient without any adverse reaction.

No cases of ketorolac sensitivity in the presence of proven tolerance to other NSAIDs have been reported. By contrast, several reports of ketorolac-induced asthma in patients affected by aspirin or other NSAID intolerances have been described.^{3 5} However, NSAID intolerance reactions do not have an immunologic basis, but the underlying mechanism seems to be the inhibition of cyclo-oxygenase, with consequent block of prostaglandin synthesis. By contrast, the dose-related positivity observed during intracutaneous injection of ketorolac suggests that a type I hypersensitivity to ketorolac was the mechanism involved. Therefore our data demonstrate the existence of selective sensitivity to ketorolac in a patient without aspirin or other NSAID intolerances.

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