

Brief communications

Reflex sympathetic dystrophy: An adverse reaction to immunotherapy

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Reflex sympathetic dystrophy (RSD) has recently been redefined as a complex group of disorders that may develop as a consequence of trauma affecting the limbs with or without an obvious nerve lesion. It consists of pain and related sensory abnormalities, abnormal blood flow and sweating, abnormalities in the motor system, and changes in structure of both superficial and deep tissues ("trophic changes").¹ RSD usually occurs after trauma to the affected limb but may occur after a myriad of medical illnesses.² There are two case reports of RSD after vaccination with equine tetanus antitoxin.^{3,4} We believe this to be the first reported case after subcutaneous allergen immunotherapy.

CASE REPORT

A 33-year-old woman had excruciating pain in the right upper arm almost immediately after receiving 0.2 ml of a subcutaneous allergy injection. This was the twentieth increasing dose of weekly immunotherapy containing 25% *Dermatophagoides farinae* (10,000 allergenic units (AU)/ml), 7.5% *D. pteronyssinus* (10,000 AU/ml), 2.5% *Alternaria tenuis* (1:10), 2.5% *Aspergillus fumigatus* (1:10), 2.5% *Fusarium vasinfectum* (1:10), 20% cat (1:10), 10% dog (1:10), and 30% diluent (Hollister-Stier, Spokane, Wash.). Application of epinephrine on cotton ball and use of an ice pack were minimally effective. She was first seen in an emergency department after several hours and was given intramuscular Toradol and subcutaneous epinephrine with minimal pain relief. The pain continued and was only partially relieved by ibuprofen. She had no history of problems with her immunotherapy or recent trauma.

Two days after injection she began to notice tingling sensations, mottled appearance of the skin, intermittent

Abbreviations used

AU: Allergenic units
RSD: Reflex sympathetic dystrophy

coolness, and decreased "perception" without weakness in her right extremity.

Examination 6 days after injection showed the right extremity to be cooler than the left, and the skin showed violaceous mottling. Pulses were normal. No obvious edema and no changes in sweating were noted, and blood pressure was 120/90 mm Hg bilaterally. An area of violaceous darkening could be seen around the injection site, which was slightly tender and blanched with pressure. Results of neurologic examination were normal except for possible slight decrease in intensity of sensory stimuli over the right hand. Roentgenogram of the right arm was normal. Thermography report showed persistent abnormal thermal asymmetry in the upper extremities, with the right side cooler in a diffuse pattern from fingers to elbow; temperature differences reached significance at 1 to 3°C. This pattern correlated with the patient's pain diagram showing right upper extremity symptoms, and was most suggestive of sympathetic dysfunction and compatible with RSD.

Symptoms gradually improved without medication but with normal use of the arm; however, 7 months after injection, tingling and coolness in the right arm and hand lasting several minutes have occurred every other week. The patient elected to discontinue immunotherapy after the reaction started.

DISCUSSION

Local reactions (<20 mm) characterized by pruritus, erythema, and induration lasting up to several hours are commonly associated with immunotherapy. Larger reactions are less common, may cause considerable local discomfort, and may persist for several days. Local reactions may be accompanied by a dull ache but rarely by sharp pain. The features of this case were quite different and fit the diagnostic criteria for RSD because of the severe and persistent pain at the injection site, the phys-

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ical findings of marked autonomic dysregulation, and the thermogram, which confirmed the latter.

Theories of RSD pathogenesis include central and peripheral nerve mechanisms: tissue injury produces nociceptive input to the spinal cord, which results in hyperexcitability of spinal cord neurons. Efferent sympathetic nerves are activated, and through an α -adrenergic mechanism, stimulate peripheral nociceptors, again completing a reflex loop.¹ Treatment is aimed at blocking the sympathetic system to break the cycle and mobilizing the limb to restore normal function. In early or milder cases, limb mobilization may be sufficient to reverse the pathologic process.

RSD after allergen immunotherapy has not been previously reported. We believe that trauma from the needle was the triggering stimulus for RSD, rather than an inflammatory response to the allergen. Future research may help answer ques-

tions about how α -adrenergic activation of nociceptors takes place.

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EMLA cream-induced allergic contact dermatitis: a role for prilocaine as an immunogen

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EMLA cream is an eutectic mixture of lidocaine 2.5% and prilocaine 2.5%, widely used in Europe for many years and recently introduced in the United States. Its topical anesthetic and analgesic properties have led to many therapeutic uses. These include single application before procedures such as venipuncture, vascular cannulation, lumbar puncture, and minor dermatologic procedures¹; and repetitive long-term use for amelioration of pain during cannulation in patients undergoing long-term hemodialysis.² EMLA cream is also

Abbreviation used

PHN: Postherpetic neuralgia

beneficial in mitigating intractable postherpetic neuralgia (PHN).

Despite widespread and frequent use, there are no reports of allergic contact dermatitis caused by EMLA cream. We report such a case in a patient with intractable PHN. Results of patch tests confirmed EMLA cream to be the offending agent and documented prilocaine as the ingredient responsible for the allergic contact dermatitis. Furthermore, this case is unique in being the first report of isolated primary hypersensitivity to the local anesthetic amide, prilocaine.

CASE HISTORY

Topical EMLA cream (15 to 30 gm/day), under an occlusive dressing with cellophane (Saran) wrap pro-

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