# LETTERS TO THE EDITOR

## Chronic Aquagenic Urticaria

*Sir,*

Aquagenic urticaria is a rare form of chronic physical urticaria (1), consisting of the occurrence of wheals in the skin in contact with water. Reaction occurs regardless of the temperature of water. We report the case of one patient with chronic physical aquagenic urticaria and discuss the pathogenetic mechanisms involved.

A non-atopic 55-year-old man, having suffered from gout for 5 years, referred to us a 1-year history of itching episodes, erythema and wheal formation every time he was exposed to water regardless of its temperature. Wheezing, difficulty swallowing and subjective respiratory distress also occurred with drinking water. Forced expiratory volume (VEMS) was not measured. Urticarial reaction appeared immediately in the zone of skin in contact with water and tended to generalize involving one limb entirely. Urticarial reaction persisted *for minutes even 1 hour* after interruption of contact with water. Water used for toilette, sea water, swimming-pool water and even rain triggered wheal formation. His own saliva on the skin provoked the occurrence of wheals. The patient presented moderately positive dermographism. Laboratory tests revealed serum uric acid, 8.80 mg/dl; serum IgA, 670 mg/dl (normal range, 72–232); and serum IgE 137 UI/ml (normal range, 14–120). Skin biopsy taken immediately after wheal induction showed massive degranulation of dermal mast cells.

With the preliminary diagnosis of chronic physical contact urticaria induced by water, a series of examinations were conducted in order to confirm the diagnosis and ruling out other types of physical urticaria. Challenge test consisted of immersion of forearm in water and application of packs soaked in distilled water at room temperature on the forearm. Both tests were strongly positive.

Intradermic injection of methacholine was negative. Application of a glass tube full of water at 2°C induced mild erythema when the tube began to sweat. Application of a glass tube full of water at 40°C induced mild erythema by heat. With the diagnosis of chronic physical contact urticaria induced by water we observed the ability of different organic solvents to reproduce urticarial reaction and the ability of different substances to prevent water contact urticaria (2).

Effective organic solvents, acetone and ethanol, induced wheal formation after 5 min in contact with dry skin areas. Wheals developed earlier if water was added to both solvents. With the purpose of antagonizing the presumed cholinergic component (1–3), scopolamine at 9% was applied during 10 min, on dry skin; although "test for sweating" was not done. Scopolamine did not prevent wheal formation either in petrolatum or in aqueous excipient. The topical application of petrolatum, olive oil, two antihistamines (Atarax® and Hismanal Syrup®, a chemical protective glove (Guante Blanco®) composed by stearic acid, glycerin, propyleneglycol, sorbitol and silicone, a water substitute (Cethaphyl®) composed by stearyl alcohol, cetyl alcohol, propylparaben, butylparaben, sodium lauryl sulphate, methylparaben and finally, clobetasol propionate did not impede (or prevent) urticarial reaction after contact with water. Only intradermal injection of triamcinolone acetonide (40 mg; 1 $cm^3$) prevented the development of wheals after contact with water.

The initial treatment was the combination of prednisone and hydroxycine hydrochloride. The patient is presently receiving 25 mg daily of hydroxyzine. He is comfortable with routine activities such as washing hands, drinking water or walking in the rain.

Sibbald et al. (3) studied two patients with urticaria localized in those zones of skin contacting with water. In those patients, local release of acetylcholine was shown, suggesting an essential step in the pharmacogenesis of wheals in aquagenic urticaria. Sibbald et al. based their statement in the observation of the suppressor effect of local application of scopolamine in water-induced wheals. We failed to confirm this fact in our patient. Methacholine test was also negative. Application of scopolamine at 9% in petrolatum did not prevent the development of wheals after contact with water.

How the water, on the surface of the skin, initiates wheal formation and itch is unknown (1). The follicular localization of wheals would suggest that some toxic substances, formed by the action of water on sebum or sebaceous glands, through the pilosebaceous unit, would exert an effect of local mast cell degranulation. No urticaria occurred in zones with few and small sebaceous glands.

Active organic solvents (acetone) *per se* did not trigger wheals. Nor did they prevent wheal formation by water exposure (2). The patient we studied developed wheals after the skin contact with different solvents like acetone or ethanol and lesions appeared earlier when water was added to the solvent. According to the hypothesis of Czarnetzki et al. (3), prolonged application of water on the skin would cause solubilization of an antigen. Acetone is a better solvent than water and could enhance urticaria whereas alcohol, benzene and gasoline would be inefficacious. An unknown substance would penetrate into the epidermis and contact with skin mast cells, which could carry the unknown antigenic substance with specific IgE on its surface. The fact that our patient developed wheals when contacting with acetone and ethanol supports this hypothesis.

## REFERENCES

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