DOI: http://dx.doi.org/10.3315/jdcr.2014.1187

Journal of Dermatological Case Reports

PHOTOLETTER TO THE EDITOR

Acquired idiopathic generalized anhidrosis

Uwe Wollina

Department Dermatology and Allergology, Academic Teaching Hospital Dresden-Friedrichstadt, Dresden, Friedrichstrasse 41, 01067 Dresden, Germany.

Corresponding author:
Uwe Wollina, MD, Professor of Dermatology and Venerology, Allergology & Environmental Medicine, Chair of the Department of Dermatology & Allergology, Friedrichstrasse 41, 01067 Dresden, Germany. E-mail: *wollina-uw@khdf.de*

Abstract

Anhidrosis is a failure in sweat production in response to physiological thermal or chemical stimuli. Acquired idiopathic generalized anhidrosis is a rare disorder without sweat gland pathology and without neurologic symptoms. Most cases have been reported from Far East. We report a case of a 58-year-old Caucasian male who suffered from heat intolerance, heat-induced cutaneous burning and failed to sweat even in sauna for five years. A skin biopsy disclosed no pathologies. He had no neurologic disorders. The diagnosis of acquired idiopathic generalized anhidrosis was confirmed and treatment with methylprednisolone initiated. This led to improvement of heat tolerance, remission of burning and partial remission of sweating. (*J Dermatol Case Rep*. 2014; 8(4): 120-121)

Key words:

acquired anhidrosis, minor starch test, eccrine sweat glands, corticosteroids

Anhidrosis is a failure in sweat production in response to physiological thermal or chemical stimuli. Acquired idiopathic generalized anhidrosis is a rare disorder. A 58-year-old Caucasian male presented with heat intolerance, burning skin when exposed to heat and failure to sweat even during sauna. Five years ago this condition developed without any precursor symptoms. Before that time the patient easily sweated and reported on diminished quality of life due to excessive sweating. His medical history was positive for arterial hypertension. There was no family history of sweating disorders nor neurologic diseases.

On examination we found an obese patient (body mass index 35.7) without neurological or cutaneous symptoms except the failure to sweat. Minor's iodine starch test was performed but remained negative (Fig. 1).[1] A skin biopsy was taken, which revealed normal eccrine gland morphology, no inflammatory infiltrate (Prof. W. Sterry, Charité Berlin). Serum IgE was in the normal range.

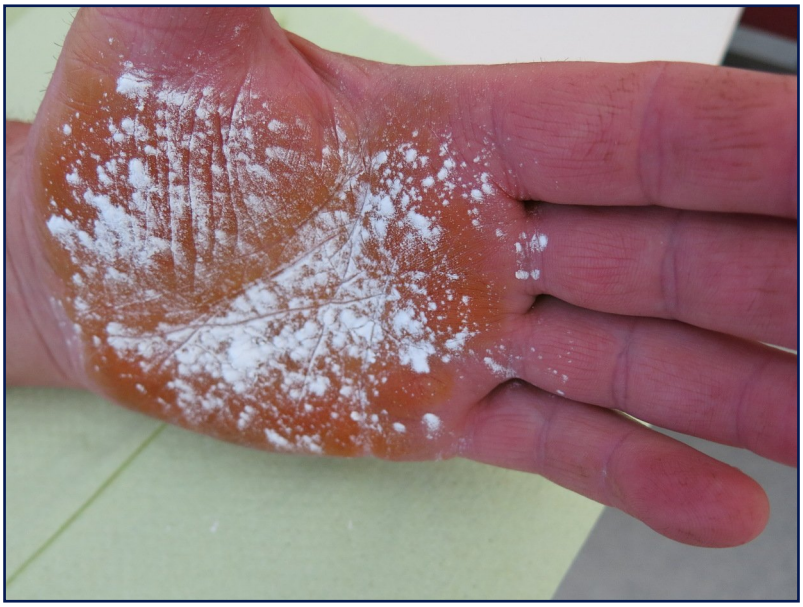

Figure 1

Minor's starch test in acquired idiopathic generalized anhidrosis. No response at all.

The diagnosis acquired idiopathic generalized anhidrosis was confirmed. He was treated with 80 mg methylprednisolone per day for 8 weeks. The treatment was well tolerated. A second iodine starch test demonstrated a partial response of eccrine sweat glands (Fig. 2). The patient reported improvement of heat tolerance. Burning sensations were no longer existent even during sauna. The corticosteroid was tapered down to zero.

Anhidrosis is a failure in sweat production in response to physiological thermal or chemical stimuli. Eccrine secretion of sweat is important in body temperature control, innate immune system, skin barrier function, mineral and hormonal metabolism.[2] Sweat glands are also an important source of somatic stem cells in skin contributing to epidermal wound healing.[3]

Congenital absence of eccrine sweat glands, hereditable disorders, neuropathies, or drugs may lead to anhidrosis.[4] In contrast to these diseases, acquired idiopathic generalized anhidrosis is a rare disorder without sweat gland pathology and without neurologic symptoms.[5]

Unlike our patient most reported cases were young men form China, Japan or Singapore.[5,6] It has been speculated that a postsynaptic defect in the cholinergic receptors or secretory cells of eccrine glands might be responsible. Three subtypes of the condition may be differentiated (Table 1). Our patient fulfilled the criteria of idiopathic pure sudomotor failure. Treatment options are limited but higher doses of systemic corticosteroids have been used successfully in several patients.[5,6]

In conclusion, acquired idiopathic generalized anhidrosis is a rare disease — in particular in Caucasians. It severely impairs quality of life due to diminished heat tolerance. Systemic corticosteroids can improve sweating and quality of life. However, dosage and duration of treatment are not standardized.

Figure 2

Minor's starch test with a partial response after 8 weeks treatment with 80 mg methylprednisolone/d.

Table 1. Subtypes of acquired idiopathic anhidrosis.

Type	Histopathology	IgE	Heat induced pain or urticarial
Sweat gland dysfunction	inflammation around eccrine glands	normal	Absent
Idiopathic pure sudomotor failure	normal	↑	pain and/or urticaria
Sudomotor neuropathy	↓VIP-positive nerve endings around sweat glands	normal	Absent

References

1. Minor V. Ein neues Verfahren zu der klinischen Untersuchung der Schweißabsonderung. *Dtsch Z Nervenheilkd*. 1928: 101: 302-307.
2. Peng Y, Cui X, Liu Y, Li Y, Liu J, Cheng B. Systematic review focusing on the excretion and protection roles of sweat in the skin. *Dermatology*. 2014; 228: 115-120. PMID: 24577280.
3. Lu C, Fuchs E. Sweat gland progenitors in development, homeostasis, and wound repair. *Cold Spring Harb Perspect Med*. 2014; 4(2). pii: a015222. PMID: 24492848.
4. Indo Y. Nerve growth factor, pain, itch and inflammation: lessons from congenital insensitivity to pain with anhidrosis. *Expert Rev Neurother*. 2010; 10: 1707-1724. PMID: 20977328.
5. Tay LK, Chong WS. Acquired idiopathic anhidrosis: a diagnosis often missed. *J Am Acad Dermatol*. 2014; 71: 499-506. PMID: 24856478.
6. Chia KY, Tey HL. Approach to hypohidrosis. *J Eur Acad Dermatol Venereol*. 2013; 27: 799-804. PMID: 23094789.