Clinical Notes

Generalized anhidrosis

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Anhidrosis is defined as a condition where perspiration does not occur despite adequate and sufficient impulse.1 This condition may be encountered as localized or diffuse through the whole body. The amount of perspiration caused by temperature, pharmacological agents, and other factors may differ inter-personally. Generalized anhidrosis may be as a result of blockage of the canals of the perspiratory glands, lack of perspiratory glands, diabetes mellitus, neuropathies, CNS lesions, Sjögren’s syndrome, or Fabry’s disease.1,2 Idiopathic acquired anhidrosis cases are observed very rarely, and the mechanism of these is not yet known.

A 42-year-old male patient presented with lack of perspiration for 16 years and excessive discomfort to heat. His skin was dry, and his skin color was normal when attending the clinic. He had a body temperature of 36°C, pulse rate of 85 bpm, and blood pressure of 110/80 mm Hg. Systemic physical examination and neurologic examination were normal. Increased blood glucose was found on biochemical assessments. His ECG and chest x-ray, brain and hypophysis MR imaging, electromyography, and sympathetic skin responses were all evaluated as normal. Perspiratory gland biopsy was carried out to assess the perspiratory glands, and they were evaluated as normal morphology. There was no history of drug usage or family members with the same complaints.

Anhidrosis occurs due to the loss of the perspiration ability of some regions or the whole body. The perspiratory glands are surrounded with post-ganglionic unmyelinated C type nerve fibers. The neurotransmitter is acetylcholine for the nervous transduction of perspiratory glands.2 Impulses emerge by stimulation of thermosensitive neurons at the front hypothalamus and preoptic area, and are transferred to the medulla spinalis via the autonomous nervous system and to the perspiratory glands of the skin.1

Perspiration disorders are seen in 3 different pictures as a decrease of perspiration, total lack, or increased perspiration. The amount of perspiration caused by temperature, pharmacological agents, and other factors may differ inter-personally. This results in a subjective diagnosis of perspiration disorders.1 If anhidrosis is limited to a small part of the body, it usually does not cause any complaints. However, the complaint of a patient with anhidrosis over a great proportion of the body is not anhidrosis, but hyperhidrosis at the intact body parts in order to regulate body temperature during hot weather.3 If anhidrosis does not allow the regulation of body temperature then the classical symptoms will emerge. These patients develop fatigue, nausea, headache, dizziness, and tachycardia in a high temperate environment or after exercise, and they should be investigated for occlusion of perspiratory gland canals, anhidrotic ectodermal dysplasia, perspiratory gland dysfunction, a CNS lesion, Sjögren’s syndrome, diabetes mellitus, hypothyroidism, inflammatory and hyperkeratotic skin diseases that may block sweat flow, Fabry’s disease, and Ross syndrome.1,2

Neurologic examination was normal in our patient, and the fact that there were no abnormal findings, and CNS pathology and cranial MRI were also normal guided us away from the CNS pathologies. The determination of normal morphologic perspiratory glands also guided us away from the causes of anhidrosis, such as the absence or agenesis of perspiratory glands. Heat shock and exposure to radiating heat are reported causes of anhidrosis in the literature, however, no such history was available for our patient. The absence of anhidrosis in the family history also guided us away from familial causes. Normal thyroid function tests lead us to eliminate hypothyroidism, which may decrease the body temperature and thus cause perspiration disorders. No clinical findings suggesting any dermatological disorder that may explain anhidrosis were found during physical examination. Also, there were no additional systemic findings that may suggest Fabry’s disease. Blood glucose was increased on blood biochemistry tests, and we observed that diabetes mellitus, which is one of the causes of anhidrosis, accompanied the anhidrosis in our patient.

Perspiration disorder in diabetes mellitus is a well-known condition. From the literature, anhidrosis was observed particularly in the lower body and lower extremity in diabetic patients.3 Diabetic anhidrosis is caused by the impact of diabetic neuropathy on peripheral sympathetic fibers, and the diagnosis is established by electrophysiological tests. Two types of symmetrical sensorial polynuropathies are observed in patients, namely, wide and small fiber polynuropathy. In small fiber polynuropathy, the electromyography may be normal and sensorimotor skin responses may be absent. In wide fiber polynuropathy, the electromyography shows slowing of motor conduction and needle electromyography shows chronic partial denervation.4 The electromyography performed in our patient did not show peripheral neuropathy, and the sympathetic skin response of both palms and soles was evaluated as normal.
The pathogenesis of idiopathic acquired anhidrosis is unknown. Typical histopathologic findings include CD3- positive lymphocyte infiltrations of the sweat glands. Corticosteroids treatment may improve anhidrosis. In our patient, steroid treatment was given for 5 days in a 1g/day dosage. Over the following 2 months, 1g/day pulse steroid treatment was given for 5 days. After this treatment, perspiration was observed at the forehead by heat and effort (Figure 1).

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In conclusion, there are numerous diseases and causal factors that may cause anhidrosis. The establishment of a specific cause may necessitate broad investigations. Corticosteroid treatment can be tried in the treatment of idiopathic generalized anhidrosis.

References


ILLUSTRATIONS, FIGURES, PHOTOGRAPHS

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