Post-typhoid anhidrosis: a clinical curiosity

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Summary

A 19-year-old girl developed generalised anhidrosis following typhoid fever. Elaborate investigations disclosed nothing abnormal. A skin biopsy revealed the presence of atrophic as well as normal eccrine glands. This appears to be the third case of its kind in the English literature. It is postulated that typhoid fever might have damaged the efferent pathway of sweating.

Keywords: anhidrosis, hypohidrosis, sweat gland, typhoid fever

Anhidrosis is defined as the inability of the body to produce and/or deliver sweat to the skin surface in the presence of an appropriate stimulus and environment¹ and has many forms (box 1). The localised form, despite giving a vital diagnostic clue to the underlying disease (eg, leprosy, Horner's syndrome) does not cause bodily discomfort to the patient. The generalised form upsets the thermoregulatory mechanisms and causes thermal intolerance; it may result in heat stroke and even death in tropical climates.² This report describes a curious case of generalised anhidrosis following typhoid fever. The extreme rarity of this complication of typhoid prompted this report.

Case report

A 19-year-old woman was investigated in May 1992 for generalised absence of sweating. She was the only female child of her consanguineous parents and was fourth of seven siblings. None of her family members had any evidence of ectodermal dysplasias or neuroendocrine diseases. She had a history of high (39–40.5°C) intermittent febrile illness for three weeks when she was 12 years old. It was diagnosed and treated as typhoid fever by her

family physician. Shortly after convalescence she felt vague discomfort and later recognised that she was not sweating as before. In the past seven years she never noticed sweating in any part of her body. During the summer and after physical exercise she was disabled by an episodic rise of body temperature (41.4°C was recorded once). Such episodes were associated with general malaise, headache, palpitations, dyspnoea, chest pain, sore throat, dry mouth, muscular cramps, dizziness, syncope, inability to concentrate, and leucorrhoea. She attained menarche at the age of 12 and her menstrual cycles were normal. Hypothalamic functions such as hunger, thirst, emotions, libido, and sleep were normal. Two years before admission she had been investigated at another centre. A skin biopsy performed there reported normal eccrine sweat glands.

An elaborate physical examination of general physique, heart, lungs, thyroid, abdomen, genitalia, eyes, mouth, salivary and lacrimal glands, lymph nodes, ear, nose and throat disclosed nothing abnormal. There were no evidences of sensory, motor or autonomic disorders of the nervous system. The skin was dry and scaly. Other ectodermal derivatives such as teeth, hair, nail and breast were normal. A 10 × hand lens revealed normal sweat pores in the palmar creases. A continuous body temperature recording showed normal early morning temperature and a tendency for this to rise towards evenings.

Haematological and biochemical investigations as well as radiographs of skull were normal. Elaborate screening tests for occult infections, tumours, metabolic and autoimmune diseases were negative. Starch-iodine test,⁸ thermal sweating test and exercise test demonstrated a total absence of sweating. During the thermal sweating test she developed diffuse flushing of the skin, tachycardia, dyspnoea and fine rales over both lung fields. A pilocarpine test was suggested but not performed. A skin biopsy from the axilla revealed normal epidermis, dermis, hair follicles, sebaceous and apocrine glands. Most of the eccrine glands were of normal morphology while a few were atrophic. There was no immune cell infiltration around the atrophic glands (figure).

She was diagnosed as suffering from acquired idiopathic generalised anhidrosis and she was reassured. Her symptoms have remained unaltered during the subsequent two-year follow-up. She accepts a sedentary life-style and has learnt to use cold water to reduce body temperature.

Anhidrosis
<ul style="list-style-type: none">• congenital or acquired• primary or secondary• localised or generalised• partial (hypohidrosis) or complete (anhidrosis)• permanent or temporary (eg, sweat gland fatigue, miliaria)

Box 1

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Figure Skin biopsy showing an atrophic eccrine gland. Haematoxylin-eosin stain. Magnification 100 ×

Discussion

Generalised anhidrosis without any organic lesion is extremely rare (see boxes 2–4). It has been variously named acquired generalised idiopathic anhidrosis, chronic idiopathic anhidrosis and pure progressive sudomotor failure.^{3–5} It is generally believed to be due to an interruption in the neural pathway of sweating, although the exact cause and mechanism of this neural interruption is largely unknown. A history of typhoid fever preceding the onset of anhidrosis in the above reported case may provide a clue to this mystery.

On a survey of the literature, only two similar cases could be found in which anhidrosis was reported to follow typhoid. In 1936 Fog reported generalised anhidrosis in a 25-year-old man following an attack of paratyphoid fever.⁶ Fog assumed the lesion to be at the hypothalamic sweating centre and implicated paratyphoid fever as the cause. Engelhardt and Melvin reported the second case, a 49-year-old woman, in whom generalised anhidrosis followed an attack of typhoid fever.⁷ This case appears to be the third of its kind in English literature.

Sweating is controlled by the anterior nucleus of the hypothalamus. The efferent sudomotor pathway starting from the hypothalamus is relayed through the brainstem, intermediolateral horn of the spinal cord, sympathetic ganglia and non-myelinated C fibres.⁸ There are enough data to state that neural cells of the sweating pathway undergo destructive changes at high body temperature. Shelley *et al* cited 13 cases of heat stroke in whom a reduction in the cell count of the hypothalamus was demonstrated.¹ Delgado *et al* reported lesions in the intermediolateral horn of the spinal cord in a case of fatal heat stroke.⁹

Causes of physiological anhidrosis

- newborn period
- dehydration due to vomiting, diarrhoea or diabetes
- senile skin

Box 2

Causes of generalised anhidrosis

- heat stroke
- hysteria
- anorexia nervosa
- poisons and drugs (eg, atropine, arsenic, morphine)
- post exfoliative dermatitis
- miliaria
- sweating fatigue (tropical anhidrosis)
- sun burn
- Sjogren's syndrome
- diabetes mellitus
- orthostatic hypotension
- occult malignancy especially lymphomas
- autoimmune anhidrosis
- scleroderma
- idiopathic

Box 3

Causes of localised anhidrosis

- myelitis
- alcoholic or diabetic polyneuritis
- oedema and urticaria
- dermatitis (seborrheic, atopic, contact, exfoliative, etc)
- pressure effect
- neurodermatitis
- leprosy
- iatrogenic (eg, sympathectomy, skin grafts, etc)
- radiodermatitis
- antiperspirant chemicals and cosmetics

Box 4

Experimental data suggest that nerve cells can tolerate a maximum of 42°C for 40 to 60 minutes.¹⁰ Neural complications were reported after whole body hyperthermia at a maximum of between 40 and 43°C for six hours.¹⁰ Hence it is probable that the prolonged high fever in the reported case might have damaged the neural cells of the sweating pathway. The generalised nature of the illness indicates that the lesion is unlikely to be at the level of the spinal cord or at the peripheral neurons. Selective neuronal damage at the sweating centre of the hypothalamus appears to be the correct explanation.

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