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Anhidrosis following exfoliative dermatitis

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Mr J. J., a 32-year-old Iraqi, presented for investigation of anhidrosis. In 1967 he first developed a pruritic erythroderma with exfoliation, alopecia, nail loss and mucosal ulceration. There was no history of drug ingestion. Recovery was gradual. Since 1968 the patient has been unable to sweat. As a result he is heat intolerant, with 10 min in a hot environment causing palpitation, headache and fever.

Examination

The patient appeared fit. The skin, hair, nail and oral mucosa were normal, except for a patchy eczematous rash localized in the presternal area. There was no evidence of a congenital ectodermal defect or of miliaria. Blood pressure was normal, lying and standing.

Investigations

(1) Routine investigation and electrophoretogram were normal.

(2) Exercise for 10 min in warm surroundings caused a rise in oral temperature to 40.6°C. As a result, the patient had dry flushed skin, felt unwell and had a pulse rate of 160/min. Extensive testing with Quinizarin powder revealed complete anhidrosis. Starch paper painted with 3% iodine in 100% alcohol is a more sensitive indication of sweating when pressed firmly against the skin with cottonwool for 3-5 s. This test confirmed anhidrosis, except for slight sweating of the finger tips. 5% Phthalaldehyde in xylene (Shelley, 1967) paint was also negative for sweating and sweat pores.

(3) The anhidrosis was not due to central or sympathetic dysfunction because raising the oral temperature from 36.4°C to 38°C, by semi-immersion in a hot bath, caused a reflex rise in skin blood flow measured by skin thermometry (see Fig. 1). A cold bath caused vasoconstriction and pilo-erection. Further evidence that the postganglionic sympathetic pathway was
intact was obtained by injection of \(2 \times 10^{-7}\) mol/l nicotinic acid tartrate which caused axon reflex erythema and pilo-erection but no sweating.

(4) A defect residing in the sweat gland itself was confirmed by the failure of 10 μm and 0.1 mg intradermal injections of Mecholyl to cause local sweating of any site. The Mecholyl injections were repeated with sellotape stripping and again were negative. The application of lanolin and other hydrophilic waxes also failed to promote the delivery of sweat (Sulzberger & Hermann, 1954).

(5) Biopsies were taken from palm, forehead, back and axilla and the sweat glands examined. Serial sections showed apparently normal ducts and sweat glands. In the axillary specimen, there was fibrosis around many of the ducts. The biopsy from the rash on the chest showed epidermal thickening and lymphocyte cuffing of the blood vessels. Direct immunofluorescence studies were negative.

Discussion

Anhidrosis may be due to failure of sweat production or interference with the delivery of sweat to the surface. Abnormalities of the sympathetic nerve pathway will interfere with sweat production. The investigations done on this patient suggest that the nerve supply to blood vessels and hair follicles was normal. There is no selective test for sympathetic nerve function to the sweat glands, so an isolated neuro-glandular or receptor abnormality cannot be excluded.

A structural abnormality of the ducts should be seen on biopsy. Shelley, Horvath & Pillsbury (1950) state that following exfoliative dermatitis, the sweat ducts may be atrophic or absent with no visible change in the sweat coil itself. Our patient’s biopsies showed normal appearing ducts in most areas. A high level blockade was excluded by stratum corneum stripping. The patient therefore appears to have a widespread idiopathic anhidrosis. In Iraq, summer temperatures reach 48–58°C, making life difficult for those with heat intolerance.
Totel (1974) studied physiological responses to heat using resting men in an environmental chamber at 38°C temperature and 20% relative humidity. Healthy individuals tolerated 150 min with a weight loss of 259 g/h and no physiological strain. A burned patient, with 56% loss of sweat glands, also completed 150 min with a weight loss of 192 g/h and marginal physiological strain. However, patients with hereditary anhidrotic ectodermal dysplasia had to be withdrawn after 75 min, in acute distress with hyperthermia, tachycardia and panting. They had a weight loss of only 68 g/h of which 54% was pulmonary water loss from over-breathing. The increased respiratory muscle efforts caused a marked rise in oxygen consumption and this panting response seemed to contribute to the heat stress.

Our patient reacts to heat in a similar way to this hereditary dysplastic group. Fortunately, air-conditioning enables him to continue living in Iraq.

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References

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