Generalised Anhidrosis Secondary to Intracranial Haemorrhage

Dear Editor,

A man in his 20s presented with a 3-year history of anhidrosis. There were no autonomic symptoms and he was not on any long-term medications. Five months prior to onset of symptoms, he suffered a ruptured intracranial arteriovenous malformation. On examination, there was spastic right hemiparesis. Sensory and postural blood pressure examinations were normal. Dermatological examination, including hair, nails and teeth, were normal.

Thermoregulatory sweat testing was performed in an enclosed room at 32˚C and 68% humidity. An admixture of starch and iodine powders was sprayed over his whole body and almost-complete generalised anhidrosis, including the palms, was observed (Fig. 1). Serum thyroid hormones were normal.

In vivo high-definition optical coherence tomography (HD-OCT)(Skintell®) was performed on multiple sites. Sweat ducts were present and no obstruction of the acrosyringium was visualised (Fig. 2). A cholinomimetic, carbachol (0.1 mL Miostat® 0.01%), was injected intradermally, which stimulated sweat production locally (Fig. 3). Review of brain magnetic resonance imaging (MRI) images revealed haemorrhage in the basal ganglia extending into the third ventricle (Fig. 4). The patient was managed conservatively with advice to avoid strenuous activities and medications that can exacerbate hypohidrosis.

Exogenous, drug and dermatological causes of hypohidrosis were excluded through clinical assessment. HD-OCT demonstrated intact sweat ducts and absence of acrosyringium obstruction, thereby excluding ectodermal dysplasia and miliaria respectively. Normal local sweat production upon intradermal injection of a cholinomimetic indicated that the pathology of anhidrosis was neurological rather than dermatological (such as in acquired idiopathic generalised anhidrosis,[1] a relatively common cause of generalised anhidrosis).

Neurological causes of anhidrosis can result from lesions at the hypothalamus, brainstem, spinal cord or sympathetic chain.[2] In our patient, the pathology was most likely at the hypothalamus as lesions at the other anatomical locations need to be bilateral (with resultant extensive neurological deficits) in order to cause generalised anhidrosis.

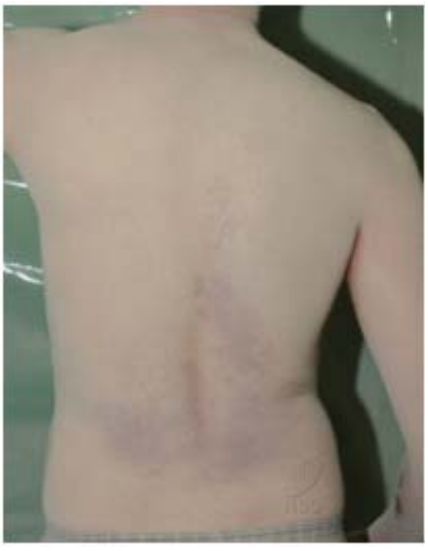

Fig. 1. Patient's back coated with an admixture of starch and iodine powders. Only light purplish patches were observed, indicating marked hypohidrosis. In controls, the powder mix turned dark purple over the whole trunk.

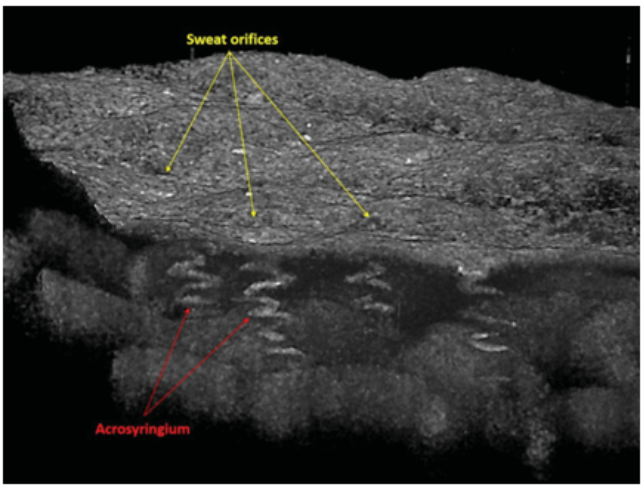

Fig. 2. Three-dimensional reconstruction of high-definition optical coherence tomography (HD-OCT) images of the palm demonstrating normal spiralling acrosyringium (intra-epidermal portion of sweat ducts) in the stratum corneum.

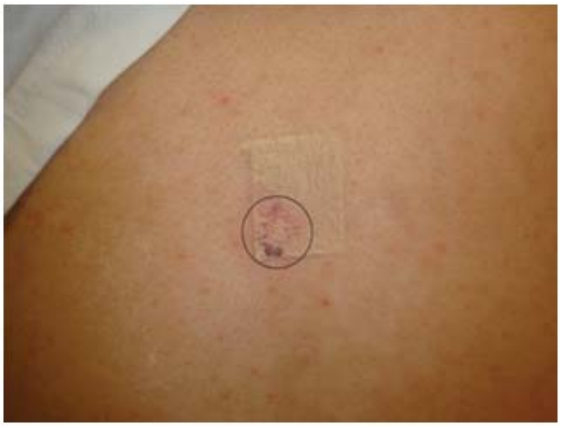

Fig. 3. Intradermal injection of carbachol stimulated sweat production, turning the admixture of starch and iodine powders purple at the sweat orifices (circled).

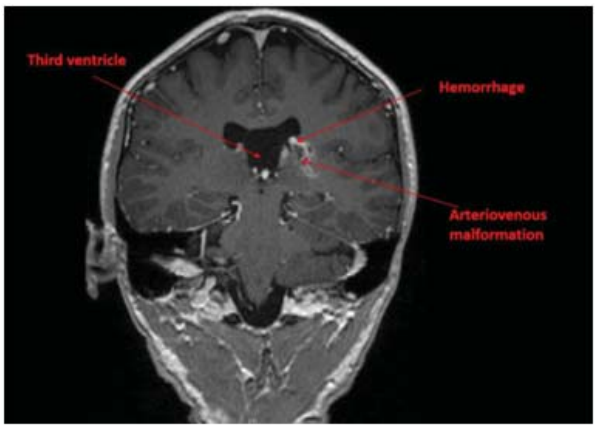

Fig. 4. Magnetic resonance image of the brain showing intracranial haemorrhage in the left basal ganglia with intraventricular extension. Small arteriovenous malformation arising in the thalamus is also visualised.

Only a few cases of anhidrosis involving hypothalamic lesions have been reported, occurring in association with neuromyelitis optica (NMO),[3] lymphocytic infundibuloneurohypophysitis[4] and multiple sclerosis.[5] Similar to the case of NMO, MRI of our patient revealed that the third ventricle, which adjoins the hypothalamus, was affected. Although our patient developed anhidrosis only 5 months after the stroke, it is known that 20% to 40% of patients with subarachnoid haemorrhage can present with delayed ischaemic neurological deficits,[6] an observation thought to be related to cerebral vasospasm from the presence of subarachnoid blood. Another possible reason for the delayed presentation is that the patient's initial poor mobility limited his exposure to heat stress, with symptoms manifesting only months later after rehabilitation and improvement in his neurological function.

We report the first case of intracranial haemorrhage causing generalised anhidrosis. Recognising and differentiating this central neurological cause from exogenous, dermatological and peripheral neurological causes is important in the management of the condition.

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