**CASE REPORT**

Inguinal hyperhidrosis in a patient with a mildly elevated autonomic symptom score being misdiagnosed as urinary incontinence

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**INTRODUCTION**

Primary focal hyperhidrosis is a common disorder resulting in sweat in excess of physiologic needs affecting approximately 15 million individuals or up to 4.8% of the general U.S. population. Hyperhidrosis not only occurs in the axillae, palms, soles, and craniofacial areas but also affects the inguinal region or groin. Inguinal sweating can be debilitating and result in profound anxiety, social life impact, and mental exhaustion. We describe a case of inguinal hyperhidrosis, where the severity and rapidity of onset of the hyperhidrosis led to multiple misdiagnoses, unnecessary treatments, and patient anxiety. An innovative diagnostic method, conceived by the patient herself, achieved the correct diagnosis.

**CASE REPORT**

A 49-year-old nurse, presented with chief complaints of groin and axillary hyperhidrosis. Her symptoms began 5 years prior with the onset of large volumes of inguinal fluid bilaterally that constantly soaked and stained her clothes (Fig 1, A). The fluid was clear, odorless, and equally distributed to the medial aspect of the inner thighs bilaterally corresponding to the femoral triangle. No fluid seemingly emanated from the vagina, urethra, or the perianal area. She described the discharge as a “leaking faucet” that occurred daily at a sustained intensity with no obvious temporal associations. The inguinal fluid caused significant anxiety, especially in social situations, and it severely disrupted her normal daily routine. The patient noticed that her inguinal symptoms were exacerbated by physical exertion, warm weather, and stress. A review of systems was remarkable for autonomic complaints, including palpitations, fatigue, brain fog, chest pain, bowel complaints, and poor sleep. Her medical history included hypothyroidism and polycystic ovarian disease; however, as her inguinal discharge continued without resolution, she had developed mixed anxiety disorder, major depression, and a binge eating disorder. Her surgical history included an appendectomy and a cholecystectomy. At presentation, her medications included sertraline, bupropion, levothyroxine, metformin, propranolol, polyethylene glycol, and glycopyronium. She reported that her symptoms did not change with new medications or dosage alterations. She denied any allergies. Her physical examination was noncontributory. Her primary physician referred her to urogynecologic specialists with a suspected, but unconfirmed, diagnosis of urinary incontinence from a neurogenic bladder.

Despite inconclusive results of an extensive workup for urinary incontinence, it remained the presumed diagnosis. After conservative treatment failed, including lifestyle modifications such as Kegel exercises and antimuscarinic medications, management escalated to percutaneous tibial nerve stimulation and bladder Botox injections. Her symptoms continued unabated over the ensuing year. Invasive therapies such as implantation of a bladder nerve stimulator were performed without any symptomatic improvement. The second bladder nerve stimulator reimplantation was also done because the first one was thought to be incorrectly positioned. When this also failed, bladder irritation by the patient's
intrauterine device was blamed, and the intrauterine device was removed. The patient remained without symptomatic relief.

Then, the patient noticed that her inguinal discharge seemed temporally related to increased axillary sweating and wondered if her inguinal discharge was sweat. As a nurse, she knew that phenazopyridine turns urine dark orange to red and purchased over-the-counter phenazopyridine 200 mg and ingested it 3 times a day after meals. She observed that her groin fluid discharge did not turn orange, whereas her urine did. This strongly suggested that the inguinal discharge was sweat rather than urine. To corroborate this, she underwent an iodine–starch test with positive results (Fig 1, B). Her final diagnosis was primary focal axillary and inguinal hyperhidrosis with associated dysautonomia. The patient was then referred for specialized treatment options.

**DISCUSSION**

In another reported case, hyperhidrosis was misdiagnosed as a clear vaginal discharge for 3 years in a 17-year-old girl. After appropriate recognition, that patient’s complaints were resolved by a simple topical application of aluminum chloride. Our patient self-diagnosed her condition using phenazopyridine that is known to turn urine dark orange. Woolery-Lloyd et al also described a patient who had inguinal hyperhidrosis that was misdiagnosed as urinary incontinence, and the patient was successfully treated with Botox injections.

The visible pattern of staining in the inguinal region may potentially cause significant embarrassment because unlike in the axillae or other body parts, sweating is not the only possible etiology. Although the precise etiology of both axillary and inguinal hyperhidrosis is unknown, a prevailing theory is that axillary hyperhidrosis is due to the hyperexcitation of enlarged apocrine glands.

Interestingly, our patient suspected that urinary incontinence was a misdiagnosis when her increased axillary sweating seemed temporally related to her bilateral inguinal discharge. Although Hexsel et al acknowledged that hyperhidrosis is a primary dysautonomia, they commented that their patients with inguinal hyperhidrosis did not seem to exhibit other signs of dysautonomia. In direct contrast, our patient described several other symptoms of primary dysautonomia, including palpitations, poor sleep, and chronic, moderate constipation, which necessitated daily polyethylene glycol. To quantify her dysautonomic symptoms objectively, a validated questionnaire evaluating 6 domains of autonomic function—composite autonomic symptom score 31 (COMPASS 31)—was administered. This was mildly elevated at 5.7 with her highest score being in the gastrointestinal domain.

In the case of our patient, repeated misdiagnosis and ineffective treatment seemed to have led to affective disorders requiring pharmacologic management. On the other hand, patients with hyperhidrosis may be predisposed to affective disorders due to their sympathetic overactivity. Owens et al, building on William James’ theory of the “physical basis of emotion,” contend for example that any psychological morbidity may, in fact, be due to an underlying primary autonomic pathology such as sympathetic overactivity. They suggest that the viscerosensory afferent nerves conveying the autonomic state (part of an interoceptive feedback) can produce exaggerated autonomic responses that are manifested
phenotypically as affective symptoms in patients with hyperhidrosis. We also have noted that primary hyperhidrosis can have a significant psychologic impact on patients, and when effectively ameliorated, feelings of somatic anxiety subside.8,9

In summary, we present an interesting case of inguinal hyperhidrosis misdiagnosed as urinary incontinence but diagnostically remedied using a simple method. Hyperhidrosis should be considered an etiology in any patient with autonomic symptoms and concomitant inguinal discharge.

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Conflicts of interest
None disclosed.

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