

Episodic Spontaneous Hyperhidrosis Hypothermia in Human Immunodeficiency Virus–Positive Patients

Episodic spontaneous hyperhidrosis hypothermia (ESHH) was described in 1969 in association with agenesis of the corpus callosum (Shapiro's syndrome) [1, 2]. It has been observed in patients with CNS abnormalities affecting structures involved with thermoregulation [3, 4] and in those without evidence of CNS pathology [5]. Different medications (e.g., clonidine [6] and cyproheptadine [7]) have been used, with varying success, to decrease symptoms. We describe two patients with HIV infection who presented with symptoms and signs suggestive of ESHH.

A 48-year-old male with HIV infection, secondary syphilis, and a CD4 cell count of 624/mL presented with complaints of recurrent episodes of profuse sweating that started several weeks earlier. These episodes were occurring about once every other day. He denied any history of head trauma, impotence, or postural syncope. During physical examination, he was sweating profusely, and his temperature was 94.5°F. He had a normal blood pressure and pulse without orthostasis. Despite hypothermia, he felt hot and was not shivering. His medication included doxycycline, itraconazole, zidovudine, and prednisone.

His complete blood cell count was normal. Blood, urine, and pharyngeal cultures (routine and for mycobacteria) were negative. A chest radiograph and brain MRI showed no abnormalities.

Therapy with oral clonidine (up to 0.2 mg t.i.d.) was started. This treatment resulted in reduction in the severity and frequency of attacks within 4 weeks. He became noncompliant with treatment; soon after, his symptoms intensified, requiring resumption of the medication with subsequent reduction in attacks.

A 48-year-old male with HIV infection, HIV dementia, candidal esophagitis, and CD4 cell counts of <10/mL was seen in the clinic for recurrent episodes of profuse sweating. During physical examination, his vital signs were as follows: temperature, 95.4°F; pulse rate, 87; and blood pressure, 133/88 mm Hg (without orthostasis). He was in no distress and was not shivering but was perspiring profusely. A chest radiograph was unchanged from prior films.

His medications included zidovudine, lamivudine, indinavir, acyclovir, trimethoprim-sulfamethoxazole, omeprazole, and prednisone. No electrolyte abnormalities were found, and his complete blood cell count was normal. A recent CT scan of the head showed only lacunar infarcts within the anterior rim of the right internal capsule. Therapy with oral clonidine (up to 0.6 mg daily) was started. Although he was not completely asymptomatic, he reported less frequent and less severe bouts of sweating and chills.

These cases appear to be consistent with previously reported cases of ESHH. As in other reports [7], our patients had an absence of shivering despite documented hypothermia. Neither patient had symptoms or signs consistent with dysautonomia, which may be seen with HIV infection. With clonidine treatment, both patients responded with reduction in the frequency of attacks.

There has been no consistent finding of definable CNS pathology in ESHH. In Shapiro's syndrome, the absence of the corpus callosum is not believed to be the etiology of ESHH, as patients who have undergone corpuscallostomy have not been described with ESHH [7]. Some investigators have described ESHH as being a manifestation of "diencephalic epilepsy" and reported responses to antiepileptic medications [8]. The absence of seizure foci (despite frequent electroencephalographic abnormalities) during attacks, however, questions this theory.

ESHH in our patients may represent HIV-related neurodegenerative processes involving the thermoregulatory areas. We cannot determine if HIV infection or one of its complicating illnesses was responsible for the development of thermal dysregulation.

ESHH may be a manifestation of unidentified structural abnormalities, either congenital or acquired (e.g., from HIV-related neurodegeneration), resulting in episodic dysfunction of central thermoregulatory areas. Our patients described their symptoms as "fever" and sweats, prompting a search for infectious complications of HIV in both of them. Negative studies and the observation that the sweats occurred in the face of hypothermia led to the current diagnostic and therapeutic strategy.

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