Oxybutynin for Hyperhidrosis in an Acquired Brain Injury Patient: A Case Report

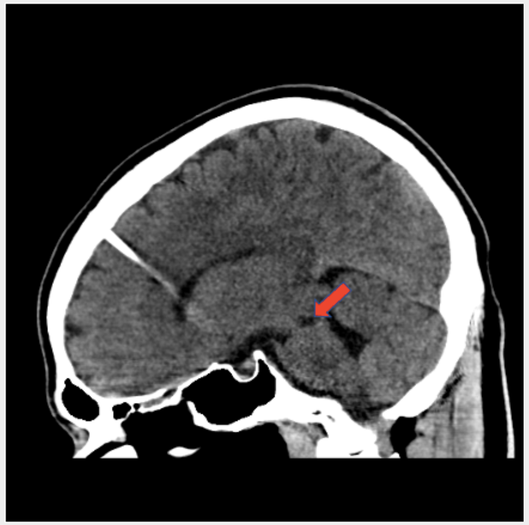
Matthew Weinstein, DO, Faiza Humayun, MD
University of South Florida, Physical Medicine and Rehabilitation Residency Program

INTRODUCTION

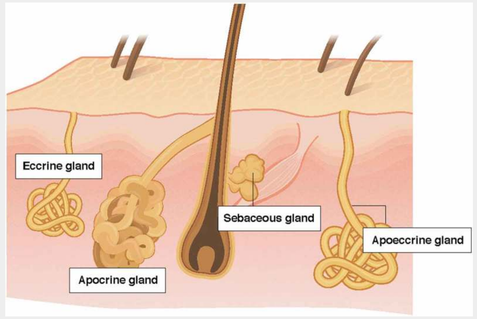
Hyperhidrosis is often an under-recognized complication of brain injury that is difficult to treat. This case examines a 27-year-old male Veteran status post left paramedian midbrain hemorrhage with asymmetric hyperhidrosis that resolved with Oxybutynin.

CASE DESCRIPTION

This patient presented to inpatient rehabilitation two months after left paramedian midbrain hemorrhage secondary to a cavernous malformation. Upon rehabilitation admission, impairments included right hemiparesis, spasticity, dysphagia, dysarthria, diplopia, cerebellar cognitive affective syndrome, discoordination, and neurogenic bowel/bladder. Approximately seven months post stroke, the patient experienced excessive sweating predominantly affecting the right face, neck, chest, and upper extremity. Possible offending medications and environmental factors were removed without resolution. About four weeks after onset of excessive perspiration, Oxybutynin was started for co-existing urinary urgency. Hyperhidrosis resolved within days of initiating Oxybutynin with no further episodes.



CT head obtained six months post-injury. Arrow is pointing to the hypodensity within the posterior midbrain, representing encephalomalacia.



Different types of sweat glands.

DISCUSSION

There are numerous published cases describing hyperhidrosis associated with brainstem infarction, hemispheric strokes, hypothalamic lesions, and spinal cord injury. The pathway regulating perspiration is thought to originate in the cortex, course through the hypothalamus and medial brainstem, and terminate in the contralateral thoracic spinal cord. All published cases in our review documented onset of excessive sweating within days of central nervous system (CNS) insult. This patient's presentation was unusual with later onset of symptoms. To our knowledge, anticholinergic medication has not been used for any case of hyperhidrosis in this patient population. The pathophysiology of hyperhidrosis is linked to activation of sympathetic cholinergic receptors. This would lend support to anticholinergic medication being a viable option for treatment. More research is needed in this patient population to establish guidelines for use of an anticholinergic medication for hyperhidrosis.

CONCLUSIONS

There is ample evidence in the literature of excessive sweating following CNS injuries. While some cases resolve spontaneously, many patients have long-lasting hyperhidrosis related to autonomic dysfunction. This case demonstrates the role of anticholinergic medication, specifically Oxybutynin, in the management of post-stroke hyperhidrosis.

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