

Dermatology flips me out



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Key words: flipped curriculum; hemangioma; MiTES; poikilodermatous plaque-like hemangioma; trigeminal trophic syndrome.

Medical knowledge grows exponentially—in 2020 it is expected to double every 73 days—mandating that clinicians be innovative in their approach to staying current.¹ In this issue of the *Journal of the American Academy of Dermatology* alone, there are 3 articles that flipped my thinking.

Generational disparities in accruing knowledge became apparent to me this year when I delivered the introductory dermatology lecture to our first-year medical students in a virtually empty lecture hall. Despite my initial response of chagrin, with further reflection, I realized that perhaps the students are taking the right approach (to a degree), learning by lecture capture at their own pace.² Liu et al³ acknowledge that medical education is transitioning from traditional lectures to a “flipped” classroom where students review educational content outside the classroom, so that interactive, learner-centered activities occur in the classroom. Video education and simulation significantly increased residents’ skin excision skills and operative self-confidence in this prospective study of 31 first- and second-year dermatology residents at 3 residency programs.³

Trigeminal trophic syndrome (TTS) is usually observed in elderly women. How strange it is to see similar lesions in young children, with similar deep facial ulcerations and scars reminiscent of TTS. Inamadar et al⁴ explain that such cases are due to the newly described midface toddler excoriation syndrome (MiTES), an autosomal recessive disease associated with biallelic mutations in the gene PR-domain containing protein 12 (*PRDM12*). The authors presented 5 new cases from 3 families. *PRDM12* influences the

Abbreviations used:

HSAN8:	hereditary sensory and autonomic neuropathy type 8
MiTES:	midface toddler excoriation syndrome
PPH:	poikilodermatous plaque-like hemangioma
PRDM12:	PR-domain containing protein 12
TTS:	trigeminal trophic syndrome

development of sensory neurons into nociceptors. Biallelic mutations also cause hereditary sensory and autonomic neuropathy type 8 (HSAN8), manifesting as mutilated extremities due to widespread pain insensitivity. MiTES is a highly localized form of HSAN8, with similarities to TTS, presenting as midfacial excoriations from a combination of neuropathic itch and scratching due to lack of pain sensation.⁴

Aside from cherry angiomas, how often do you think of hemangiomas in the geriatric population? Semkova et al⁵ detailed 16 cases of a novel entity they named “poikilodermatous plaque-like hemangioma” (PPH). Lesions were usually solitary, erythematous to violaceous poikilodermatous plaques distributed on the lower extremities and pelvic girdle. The mean age was 72 years with a male predominance. Histopathology demonstrated a distinctive band-like proliferation of vascular channels suggestive of postcapillary venules within the superficial dermis with background of fibrosis, edema, and loss of elastic fibers. Despite the poikilodermatous appearance, acanthosis was frequently observed. The authors addressed differentiating PPH from mycosis fungoides, fixed drug eruptions, acrodermatitis atrophicans, pigmented

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purpuric disorders, acquired elastotic hemangioma, microvenular hemangioma, hobnail hemangioma, and Kaposi sarcoma. The etiology of this persistent lesion is obscure. Fortunately, the clinical course appears to be indolent and benign.⁵

I can empathize with students and clinicians feeling overwhelmed and flipping out over the plethora of information we are expected to absorb. I suggest doing a backflip instead—be grateful that we enter a new decade participating in a medical-scientific revolution with the capability of providing hope and cures that were previously unimaginable.

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