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Primary Dysmenorrhea: Diagnosis and Therapy

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As the co-directors of a clinical translational research program studying female visceral pain, we commend the Ferries-Rowe et al for the extensive update on the etiology and management of dysmenorrhea in the November 2020 issue.[1] However, we also challenge both the authors and the general readership to be aware of some additional key developments in our understanding of dysmenorrhea's mechanisms that were not captured in this review. Oddly, the pathophysiology of menstrual pain historically has not been viewed using the overarching neural framework of chronic pain. An inadequate understanding of abnormal sensory function in dysmenorrhea may explain why so many menstrual pain sufferers do not respond to nonsteroidal anti-inflammatories or hormonal suppression.

While we and others have found evidence that dysmenorrhea is linked to a higher risk of developing a chronic pelvic pain syndrome, it is somewhat illogical to refer to dysmenorrhea as a central sensitization syndrome, as that would mean 40–90% of women have an abnormal physiological state.[2,3] Indeed, our research shows that in general, women with moderate-to-severe dysmenorrhea, without chronic pain symptoms, still report mild levels of daily nonmenstrual pelvic pain (average 25/100). However, they do not have evidence of impaired conditioned pain modulation, an indicator of central sensitization commonly observed in chronic pain conditions.[4]

On deeper inspection, a fifth of such moderate-to-severe dysmenorrhea sufferers, despite being chronic-pain–free, harbor silent evidence of multisensory hypersensitivity when provoked with a simple noninvasive bladder distension task, and impaired conditioned pain modulation with quantitative sensory testing. In the absence of chronic pain symptoms, such multisensory hypersensitivity may reflect early, aberrant changes in either the threat processing networks of the cerebral cortex, or the spinal dorsal horn, ultimately found in several chronic pelvic pain conditions.[5–7] The authors astutely emphasize that identifying dysmenorrhea and providing effective treatment is essential to limit chronic pelvic pain emergence, including endometriosis-associated pelvic pain and bladder pain syndromes. To be most effective, that prevention strategy will need us to further define dysmenorrhea with objective, reproducible neurobiological features that convey excessive risk for chronic pelvic painP emergence, perhaps paralleling the framework widely practiced to identify preinvasive cervical disease.

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