Prevalence of Endometriosis During Abdominal or Laparoscopic Hysterectomy for Chronic Pelvic Pain

In their large series of women undergoing hysterectomy for benign indications, Mowers et al conclude that “...fewer than 25% of women undergoing laparoscopic or abdominal hysterectomy for chronic pelvic pain had endometriosis at the time of surgery, as determined by a systematic review of the operative findings and pathology report.”

Deposits of ectopic endometrium were not the source of chronic pelvic pain in at least 75% of women in their series. If it is not endometriosis, then what is causing this substantial morbidity among women in Michigan?

There is now persuasive evidence in every organ in the female pelvis that traumatic disruption of pelvic nerves results from difficult intrapartum episodes, physical efforts during delivery, and some medical and surgical procedures for evacuation of the uterus. These three primary sources of injury to pelvic autonomic nerves result in painful clinical presentations associated with aberrant reinnervation 5–10 years later. Injured pelvic nerves express new P2X3 and TRPV-1 receptors that respond to stretch and touch, respectively. Increasing blood flow in the second half of the menstrual cycle may activate these receptors to cause chronic pelvic pain that we label as some forms of endometriosis, chronic pelvic pain, interstitial cystitis, irritable bowel syndrome, dysmenorrhea, vulvodynia, and others.

Rather than distinguishing between women with and without endometriosis, might there be merit in distinguishing between women with intact and injured uterosacral ligaments, because important collections of uterine nerves may result in painful clinical presentations associated with aberrant reinnervation? Injured pelvic nerves may result in painful clinical presentations associated with aberrant reinnervation.

In Reply: Dr. Xia et al note that traumatic injury to pelvic nerves followed by aberrant reinnervation may be a cause of chronic pelvic pain. As such, they suggest that investigation of whether women have intact or injured uterosacral nerves may result in a more meaningful classification of pelvic pain than the presence or absence of endometriosis. We thank Dr. Xia et al for their interest in our article and their insightful comments. We agree that there are many potential causes of chronic pelvic pain, including neurologic, gynecologic, urologic, gastrointestinal, and musculoskeletal sources. Although our knowledge regarding chronic pelvic pain is far from complete, there is increasing evidence to suggest that both the central and peripheral nervous systems play a role in the initiation and maintenance of chronic pelvic pain. The presence of nociceptive Aδ and C fiber nerves infiltrating endometriotic lesions was reported in women and rat models more than 10 years ago. More recently, the ectopic endometrium of women afflicted with endometriosis and other causes of chronic pelvic pain, including adenomyosis and leiomyomas, was found to be imbued with significantly denser nerve networks than pain-free control patients. This phenomenon is therefore reflective of a chronic pain state and not limited to just those with biopsy-proven endometriosis. Studies investigating markers of central pain amplification among women with chronic pelvic pain also show independence from the presence or absence of peripheral pathology. For example,

References


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