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Secondary hypermenorhea due to uterine leiomyomatosis as a factor in headache chronification in a patient with endometriosis: a case report

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Introduction
Endometriosis predisposes to headaches through shared mechanisms of neuroinflammation and central sensitization. This neurological vulnerability is often exacerbated by severe hormonal fluctuations and metabolic consequences, such as iron deficiency from abnormal uterine bleeding. This report describes how secondary hypermenorrhea due to uterine leiomyomatosis acted as a trigger and chronification factor for headache in a patient with endometriosis.

Case presentation
A 24-year-old female medical student with a three-year history of endometriosis presented without hormonal treatment. She had severe dysmenorrhea, chronic pelvic pain, and disabling catamenial headaches (VAS=6–7). Over the last 12 months, her pelvic pain worsened, and menstrual flow increased significantly, with prolonged bleeding, blood clots, and refractory cramps (VAS=9). Concurrently, she developed fatigue, orthostatic dizziness, concentration deficits, and a decline in academic performance. She was diagnosed with uterine leiomyomatosis via transvaginal ultrasound, which identified a submucosal nodule. The headaches became daily following the hypermenorrhea exacerbation. Four months ago, she progressed to daily persistent headache (ICHD-3), holocranial, pressing, with a mean intensity of VAS=8 and menses exacerbations reaching VAS=10. There was a clear temporal association between menstrual flow, pelvic pain, and headache worsening. She overused dipyrone, ibuprofen, and sumatriptan, achieving only partial relief. The HIT-6 score was 68 points (severe impact). Lab tests showed hemoglobin at 10.8 g/dL and ferritin at 12 ng/mL, confirming iron deficiency anemia. Brain MRI was normal. Continuous desogestrel 75 mcg/day, elemental iron replacement, and specialized gynecological follow-up were initiated. After four months, menstrual flow was suppressed and pelvic pain decreased (VAS=3). Headache frequency dropped from daily to four days per month (VAS=3). The HIT-6 score decreased to 48 points, with significant functional improvement.

Discussion
The headache initially associated with endometriosis became chronic after the onset of the submucosal fibroid and subsequent hypermenorrhea. Chronic inflammation from endometriosis promotes central sensitization, while the submucosal component exacerbates blood loss. Abrupt hormonal fluctuations and iron depletion lower the pain threshold and alter neurotransmitter synthesis, perpetuating pain. The concurrent improvement of gynecological and neurological symptoms following continuous treatment reinforces this pathophysiological link.

Conclusion
Hypermenorrhea due to submucosal leiomyomatosis can contribute to headache chronification in endometriosis. Managing interrelated hormonal, inflammatory, and metabolic factors led to outstanding clinical recovery, highlighting the importance of gynecological investigation in young women with persistent headaches.

Keywords:
Endometriosis
Submucosal fibroid
Persistent headache
Iron deficiency

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