Menopause Screening in Chronic Pain: A Quality Improvement Audit and the Role of Estrogen in Pain Mechanisms

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SAFETY / QA / QI PROJECT

Background: Estrogen, Menopause and Pain

Estrogen fluctuation alters central sensitization, neuroinflammation and sleep architecture, each a driver of pain burden[1].

Estrogen modulates pain pathways

Central sensitization

- Modulates NMDA receptor activity
- ⬇️ excitatory signaling (glutamate, substance P)
- Supports descending inhibition (serotonin, norepinephrine, endogenous opioids)

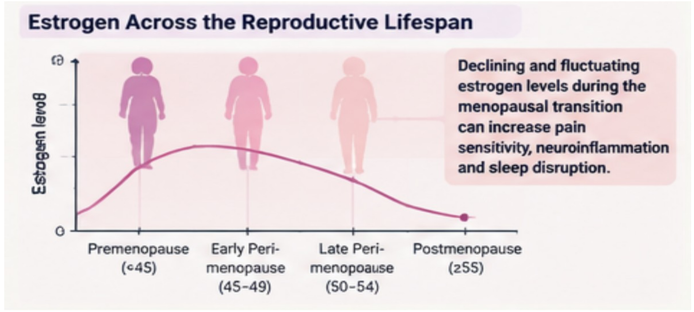
Neuroinflammation

- ⬇️ pro-inflammatory cytokines (IL-1β, TNF-α, IL-6)
- ⬇️ microglial activation

Sleep architecture

- Supports sleep continuity
- Decline linked to fragmentation and night-time awakenings

Estrogen decline across menopausal transition may amplify pain through all 3 pathways[1].



Aim: Quantify menopause documentation, symptom screening and OB/GYN referral among women with chronic pain at a quaternary pain center and describe outcomes in referred patients.

Methods: Retrospective QI audit of 120 consecutive women (40–68 y; mean 53.1 ± 8.0) seen across two visits by five providers. Extracted menopause status; vasomotor, sleep and mood symptom querying; HRT; OB/GYN referral; NRS at both visits. Referred charts reviewed for HRT initiation and outcomes. Fisher's exact test; t-test.

Quality Improvement Audit Results

Menopause screening depended on one provider, not on a clinic-wide process.

80% menopause status unknown (96/120)

83% never asked about menopausal symptoms (100/120)

5% referred to OB/GYN (6/120)

Figure 1. Documentation and screening rates (N = 120)

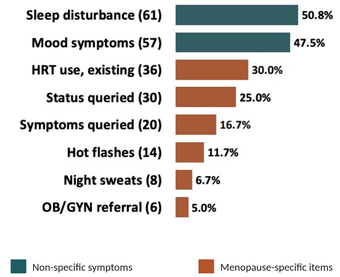
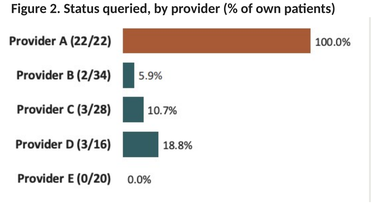


Figure 2. Status queried, by provider (% of own patients)



Provider A queried status in all 22 of their patients and made all 6 OB/GYN referrals; the other four combined queried 8 of 98 and made none.

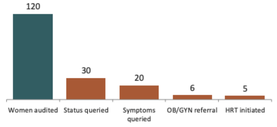
Table 1. Menopause status noted, by pain category

Pain category	Status noted	%
Pelvic	6/22	27.3
Spine	6/25	24.0
Neuropathic	5/23	21.7
Joint	4/23	17.4
Headache	2/15	13.3
Widespread	1/12	8.3

No significant difference across categories (Fisher's exact, p = .38). Status noted = peri/post-menopausal status assigned (n = 24); excludes 6 queried with status unknown.

All patients: mean NRS fell from 7.50 (SD 1.68) to 6.63 (SD 2.17) at follow-up (median 79 days); change did not differ by HRT use (t = 1.09, df = 118, p = .277), consistent with meta-analytic evidence[3].

Figure 3. Menopause care touchpoints (N = 120)



Outcomes in referred patients (n = 6, exploratory)

5/6 started HRT

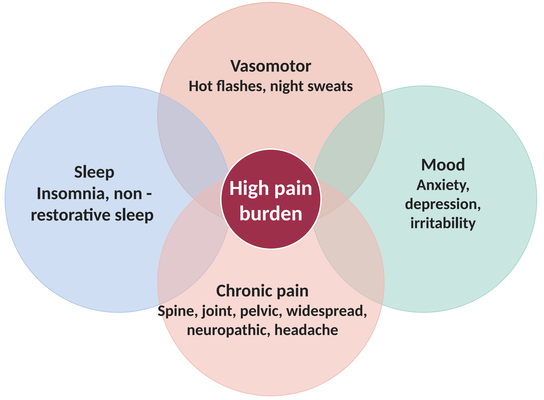
4/6 improved sleep and mood (66.7%)

7.5 → 5.7 mean NRS on HRT (n = 5; 82 days)

Clinical Implications and Care Pathways

Vasomotor, sleep and mood symptoms overlap with pain-clinic presentations, while specialty referral pathways are underused.

Overlapping symptoms in the pain clinic



Proposed Phase 2 pain clinic pathway

1. Screen
Menopausal status, vasomotor, sleep and mood symptoms, current/past HRT

2. Stratify
Flag overlapping symptoms adding to pain burden

3. Refer
OB/GYN; sleep medicine or mental health as indicated

4. Reassess
Pain, sleep, mood and function at follow-up; re-audit

Limitations: single-center retrospective review; documentation may under-capture discussion; small referred subgroup with no comparison group.

Menopause is largely unassessed in this chronic pain population.
Brief intake screening may close the gap; Phase 2 will pilot standardized screening and an OB/GYN referral pathway, followed by re-audit.

References 1. Barcelon E, et al. Cells. 2023;12(16):2028. 2. Lu CB, et al. Neural Plast. 2020;2020:8842110. 3. Overton R, et al. Post Reprod Health. 2026;32(1):52–68.

De-identified retrospective QI review, exempt from formal IRB review.