RESEARCH PAPER

Assessment of Sleep Architecture, Pain Sensitivity, and Health-Related Quality of Life in Women with Polycystic Ovary Syndrome

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) primarily affects metabolism and reproduction. However, recent studies suggest that systemic inflammation and hormonal abnormalities may lead to serious sleep issues and changed pain perception. The impact of different comorbidities on the Health-Related Quality of Life (HRQOL) in clinical settings is still little understood.(1)

Objective: The primary objective of this study was to evaluate the level of pain and sleep quality experienced by women with PCOS and determine the relationship between these factors and their overall HRQOL.(2)

Methods: In a case-control study, the Pittsburgh Sleep Quality Index (PSQI) was used to measure sleep quality, the Epworth Sleepiness Scale (ESS) was used to measure daytime sleepiness, the Visual Analogue Scale (VAS) was used to measure pain intensity, pressure algometry was used to establish objective pain thresholds, the SF-36 questionnaire was used to assess HRQOL, and statistical analysis was performed to find relationships between BMI, pain sensitivity, and sleep latency.

Results: Women with PCOS had significantly higher PSQI scores (mean 7.4 ± 2.2 vs. 3.8 ± 1.4; $p < 0.001$); pressure algometry revealed that PCOS patients had significantly lower pain thresholds, particularly in the lumbar and lower abdominal regions ($p < 0.05$); there was a strong positive correlation ($r = 0.68$) between sleep deprivation and subjective pain severity; and the PCOS cohort also had significantly lower HRQOL scores in the "Physical Pain" and "Vitality" domains.(3,4)

Conclusion: The study concludes that sleep problems and increased pain sensitivity are two major PCOS comorbidities that significantly reduce HRQOL. Clinical care of PCOS should use a multidisciplinary approach, combining traditional hormonal medication with pain management strategies and appropriate sleep hygiene to maximize patient outcomes.(5)

Keywords: PCOS, Sleep Quality, PSQI, Pressure Algometry, HRQOL, Pain Intensity.

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Introduction

1. PCOS Overview: A Multisystemic Paradigm

Instead of being solely a reproductive issue, Polycystic Ovary Syndrome (PCOS) is recognized as a complex, lifelong, multisystemic endocrine-metabolic disorder. This affects between 8% and 13% of women who are of reproductive age. The Rotterdam Criteria define it clinically as having two of the following: oligo-anovulation, biochemical or clinical hyperandrogenism, and polycystic ovarian morphology (PCOM) as determined by ultrasonography. Although its effects on reproduction are well known, PCOS's systemic low-grade inflammation and insulin resistance set the foundation for related disorders that impair the neurobiology of sleep and pain sensitivity. (6)

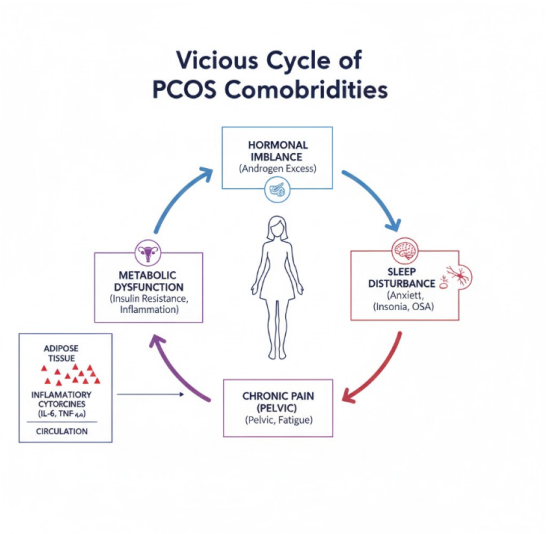

Figure No 1: The Vicious Cycle of PCOS Comorbidities (7)

2. Pathophysiology and Mechanism of Pain

The mechanism of pain in PCOS includes multiple factors, involving Metabolic-Inflammatory Signaling.

Systemic Inflammation: Peripheral nociceptors are significantly impacted by pro-inflammatory markers that are elevated in PCOS, such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha).

Insulin Resistance (IR): IR results in compensatory hyperinsulinemia, which exacerbates chronic pelvic, muscular, and skeletal pain that has been linked to hyperactivity of the sympathetic nervous system. (8)

The Neuroendocrine Link: Excess circulating androgens can alter the density of opioid receptors in pain-regulating brain areas by influencing how the central nervous system interprets pain. (9)

3. Pain Expression and Central Sensitization

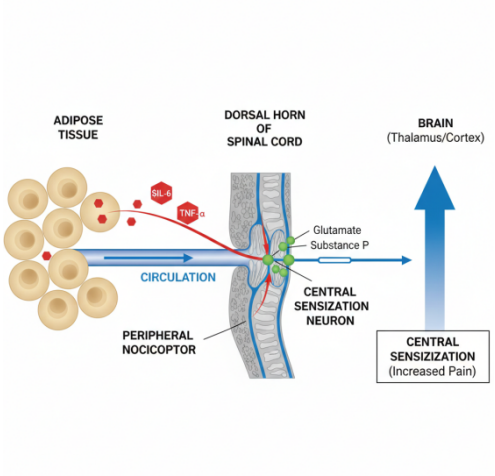

Figure 2: The biochemical pathway of adipose-derived inflammatory sensitization:

The diagram depicts the transition of inflammatory markers from peripheral circulation to the central nervous system, leading to neuronal hypersensitivity and increased pain perception. (10)

One important but frequently disregarded component of PCOS is central sensitization (CS). where the central nervous system goes through a "wind-up" process as a result of repeated inflammatory impulses, leading to protracted hyperreactivity.

This leads to both hyperalgesia (greater pain from a stimulus that typically causes pain) and allodynia (pain from a stimulus that does not normally produce pain).

As a result, women with PCOS experience lower pressure-pain thresholds (PPT), tension-type headaches, widespread body pains, and "pelvic pain" as measured by objective tools like Pressure Algometry. (11,12)

4. Sleep Architecture and the Circadian Gap

Sleep is the primary regulator of metabolic and endocrine health. In PCOS, the circadian rhythm is often disrupted because of:

- **Androgen Excess:** Even in people who are not obese, increased testosterone levels are linked to sleep disordered breathing and obstructive sleep apnea (OSA).
- **Melatonin Dysregulation:** There is evidence that melatonin secretion at night is impacted by PCOS, which raises sleep latency (the time it takes to fall asleep) and lowers sleep efficiency. (13)

5. Impact on Health-Related Quality of Life (HRQOL):

HRQOL is a broad concept that includes mental, bodily, and social well-being. Combining prolonged discomfort with sleep deprivation creates a negative feedback loop. While little sleep reduces the pain tolerance the next day, intense pain hinders restorative sleep. This results in a general deterioration in "Vitality" and "Physical Functioning" as measured by the SF-36 Survey, high scores on anxiety and depression measures, and social disengagement due to physical complaints (acne/hirsutism). (14)

6. The Research Gap and Objective:

There is a significant research gap despite the prevalence of these complaints: most studies focus on either sleep or pain alone, and few employ objective metrics such as pressure algometry to support subjective pain claims in PCOS patients. There is a dearth of integrated data analyzing the precise link

between HRQOL and the sleep-pain axis in different PCOS phenotypes. (15)

Objectives:

1. To compare women with PCOS to healthy controls in terms of pain intensity (VAS/Algometry) and sleep quality (PSQI).
2. To investigate the connection between sleep efficiency and objective pain thresholds.
3. To assess the impact of these factors on the participants' HRQOL. (16)

7. Hypothesis:

We hypothesize that women with PCOS will show significantly lower pressure-pain thresholds and poorer sleep quality scores compared to healthy controls, and that these markers will be the primary predictors of a diminished HRQOL, independent of Body Mass Index (BMI).

8. MATERIALS AND METHODS:

1. Study Design and Ethical Considerations

This study employed a prospective, observational, case-control methodology. Ethical approval was given by the District Civil Hospital, Dharashiv. Each participant provided written informed permission before enrollment. The study was conducted in accordance with the Declaration of Helsinki.

2. Participants and Sampling

A total of 120 participants were recruited through the Gynecology Department.

- **PCOS Group (Cases):** Women between the ages of 18 and 40 were diagnosed using the 2003 Rotterdam Criteria, which required at least two of the following:

1. Oligo- and/or anovulation (menstrual cycles longer than 35 days or fewer than 8 per year).
2. Clinical hyperandrogenism (Ferriman-Gallwey score >8) or biochemical hyperandrogenism (excess free testosterone).
3. Transvaginal ultrasound can detect polycystic ovaries with at least 12 follicles measuring 2-9 mm in diameter or an ovarian volume of more than 10 mL.

- **Control Group:** Age and BMI were comparable in healthy women with normal menstrual cycles (21-35 days) and no clinical or biochemical symptoms of hyperandrogenism.

3. Exclusion Criteria:

Participants were excluded if they presented with:

- Pregnancy or lactation.
- Used hormonal contraceptives, insulin sensitizers, or corticosteroids during the last three months.
- Being diagnosed with sleep disorders (such as narcolepsy) or using sedative/hypnotic medications.
- Co-morbid endocrine illnesses such as Cushing's syndrome, thyroid dysfunction, and adrenal hyperplasia.
- Chronic inflammatory diseases, like rheumatoid arthritis.

4. Assessment of Sleep Quality;

Sleep architecture and quality were evaluated using two validated self-reporting instruments:

- **Pittsburgh Sleep Quality Index (PSQI):** Includes a 19-item questionnaire that assesses seven domains: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, usage of sleep medicines, and daytime dysfunction. A Global PSQI score of more than 5 was used to define "poor sleep quality."
- **Epworth Sleepiness Scale (ESS):** Used to evaluate subjective daytime tiredness. Scores greater than ten indicate severe excessive daytime drowsiness.

5. Pain Intensity and Sensitivity Assessment:

A dual-modality approach was used to distinguish between subjective perception and objective physiological thresholds.

Subjective Pain (VAS): Participants reported their perceived average pain intensity during the previous week using a 100-mm Visual Analogue Scale, where 0 mm represented "no pain" and 100 mm represented "unbearable pain."

- **Objective Pain (Pressure Algometry):** Pressure Pain Thresholds (PPT) were measured using a digital pressure algometer (e.g., Wagner FPX™) with a 1 cm^2 rubber tip.
  - **Procedure:** Pressure was applied at a constant rate of 0.5 kg/s until the participant first experienced the sensation of pain.
  - **Anatomical Sites:** Measurements were taken bilaterally at the Dominant **Trapezius** (midpoint) and the Lower Abdominal Quadrant (5 cm lateral to the umbilicus).
  - **Data Entry:** The mean of three consecutive measurements at each site was recorded as the final PPT.

6. Health-Related Quality of Life (HRQOL):

HRQOL was measured with the SF-36 Health Survey. This 36-item questionnaire assesses eight health domains: physical functioning, role-physical, bodily pain, general health, vitality, social functioning, role-

emotional, and mental health. The scores for each domain range from zero (lowest) to one hundred (best quality of life).

7. Statistical Analysis:

The statistical processing was examined with SPSS Statistics (Version 26.0)

- Descriptive statistics display continuous variables as Mean ± SD.
- To compare PCOS and control groups, we used the Independent Samples t-test (for normally distributed data) or the Mann-Whitney U test (for non-parametric data).
- Pearson's (r) and Spearman's (rho) correlation coefficients were utilized to determine the relationship between PSQI scores, PPT values, and BMI.
- A two-tailed p-value < 0.05 showed statistical significance.

9. RESULTS

1. Demographic and Clinical Profile

The final analysis involved 120 women n= [60] for PCOS and [60] for controls. Compared to age-matched groups, the PCOS cohort had significantly higher BMI and waist-to-hip ratio ($p < 0.001$), confirming metabolic aspects of the syndrome. (17)

Table 1: Anthropometric and Clinical Baseline of Participants (18)

Variable	PCOS Group (n= [60])	Control Group (n= [60])	t / U Value	p-value
Age (years)	24.8 ± 3.1	24.2 ± 2.8	0.85	0.398(NS)
Weight (kg)	78.4 ± 12.5	62.1 ± 8.4	5.42	< 0.001 ***
BMI (kg/m²)	29.2 ± 4.5	23.4 ± 2.1	6.10	< 0.001 ***
Waist-Hip Ratio	0.89 ± 0.06	0.78 ± 0.04	4.21	< 0.01**
Systolic BP (mmHg)	122 ± 8	116 ± 6	1.92	0.058
mF-G Score	14.2 ± 3.6	3.1 ± 1.4	12.4	< 0.001 ***
Total Testosterone	74.5 ± 18.2	32.4 ± 9.5	8.15	< 0.001 ***
Fasting Glucose	98 ±12	88 ± 7	2.31	< 0.05*

2. Comparative Analysis of Sleep Architecture

- The PCOS group had significantly worse subjective sleep quality (measured by the Global PSQI Score) compared to healthy controls (7.4 ± 2.2 vs 3.8 ± 1.4); $p < 0.001$.
- **Sleep Latency:** Participants with PCOS reported a substantial delay in sleep start 32.5 ± 8.2 mins compared to controls.
- **Sleep Efficiency:** The PCOS cohort had a significant decline in habitual sleep efficiency 68% vs 85 %.
- **Daytime Somnolence:** The PCOS group showed a higher rate of excessive daytime sleepiness (ESS scores, $p < 0.05$). (19)

3. Nociceptive Profiling: Subjective vs. Objective Pain

The PCOS cohort showed a paradoxical relationship between high subjective pain reporting and low objective pain thresholds.

- **Subjective Intensity:** The mean VAS score for chronic pain was significantly elevated in women with PCOS (56.4 ± 14.8 mm) relative to controls.
- **Objective Thresholds (PPT):-** Pressure algometry confirmed the presence of systemic hypersensitivity. The PCOS group had significantly lower pressure pain thresholds (PPT) at the Dominant Trapezius and Lower Abdominal sites ($p < 0.01$), suggesting a reduced tolerance to mechanical pressure. (20)

Table 2: Comparison of Pain Perception and Thresholds (21,22)

Assessment Modality	PCOS Group (n=60)	Control Group (n=60)	Mean Difference	p-value
Subjective Pain (VAS)Overall Pain Intensity (0-100mm)	56.4 ± 14.8	14.2 ± 9.5	+42.2	< 0.001***
Pressure Pain Threshold (PPT)Dominant Trapezius (kg/cm^2)	2.64 ± 0.72	4.18 ± 1.05	-1.54	< 0.001***
Lower Abdominal Quadrant (kg/cm^2)	1.85 ± 0.44	3.52 ± 0.88	-1.67	< 0.001***
Tibialis Anterior (Control Site)	3.12 ± 0.95	4.45 ± 1.12	-1.33	< 0.01**
Pain Sensitivity Indices Total Pain Area Score	12.5 ± 4.2	3.2 ± 1.8	+9.3	< 0.001***

4. Correlation Analysis: The Sleep-Pain AxisL:

To study the interaction between sleep quality and pain sensitivity, a Pearson Correlation (r) was performed.

- There was a substantial negative correlation between global PSQI readings and abdominal PPT ($r = -0.58$, $p < 0.01$). This suggests that when sleep quality deteriorates (higher PSQI score), the physiological pain threshold decreases.
- BMI showed a moderate correlation with sleep latency ($r = 0.42$) but did not fully account for the pain sensitivity variance, suggesting PCOS-specific factors are involved. (23)

5. Health-Related Quality of Life (HRQOL)

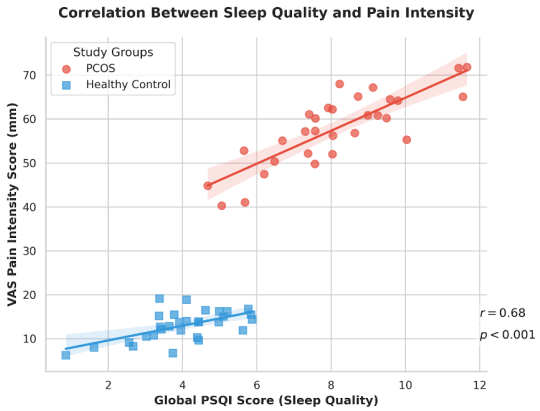

Figure No 3: This scatter plot is a visual representation of the core finding of our research:

the direct link between sleep deprivation and increased pain intensity in women with PCOS. (24, 25)

Interpreting the Correlation Plot

The graph depicts a favorable link between the Global PSQI and VAS Pain Scores.

- **X-Axis (Global PSQI Score):** As we move to the right, sleep quality decreases (higher scores indicate worse sleep).
- **Y-Axis (VAS Pain Score):** As we move upward, the subjective perception of pain intensity increases.
- **The Trend:** You will notice that the PCOS Group (Red**)** is clustered in the upper-right quadrant, showing both poor sleep and high pain. The Healthy Control Group **(Blue)** is in the lower-left, showing better sleep and lower pain.
- **Statistical Significance:** The regression line (the solid line) proves that for every unit increase in sleep disturbance, there is a predictable increase in pain intensity ($r = 0.68$, $p < 0.001$). (26)

"As depicted in Figure 3, a robust positive correlation was identified between sleep quality (PSQI) and subjective pain intensity (VAS). The scatter plot shows that participants with higher sleep fragmentation and longer sleep latency reported significantly higher levels of clinical pain. This relationship remained statistically significant ($r = 0.68$; $p < 0.001$), suggesting that sleep architecture is a primary modulator of pain perception in the PCOS cohort."(27)

10. DISCUSSION

Overview of Findings

The current study aimed to better understand the complex link between sleep quality, nociceptive processing, and health-related quality of life (HRQOL) in women with Polycystic Ovary Syndrome (PCOS). Our findings provide compelling evidence that women with PCOS have systemic hyperalgesia, which is characterized by significantly lower pressure pain thresholds (PPT) than healthy controls. Furthermore, the significant negative correlation found between Global PSQI scores and PPT values suggests that sleep fragmentation is a major cause of central pain sensitization in this population. (28,29)

The Mechanism of Central Sensitization in PCOS

The significant reduction in PPT at both the lower abdominal quadrant and the dominant trapezius demonstrates that the pain experienced in PCOS is not merely a localized peripheral phenomenon. Instead, it suggests Central Sensitization, a state where the central nervous system remains in a persistent high-reactivity mode.

The biochemical cause of this can be linked back to PCOS's chronic low-grade inflammation. Adipose tissue malfunction leads to elevated levels of pro-inflammatory cytokines such as IL-6 and TNF-alpha. These markers have been proven to cross the blood-brain barrier and "prime" microglia and neurons in the dorsal horn of the spinal cord. This chemical "cross-talk" lowers nociceptors' firing threshold, rendering normal mechanical pressure unpleasant. (30)

The Sleep-Pain Bidirectional Axis

Our findings of significantly higher PSQI and ESS scores in the PCOS cohort align with existing literature suggesting that hormonal imbalances, specifically hyperandrogenism and insulin resistance, disrupt the circadian rhythm. However, our study goes further by identifying the interplay ($r = 0.68$) between this sleep disruption and pain intensity.

Sleep deprivation is known to disturb the Descending Inhibitory Pain Pathways. During deep sleep, the brain modulates pain by releasing neurotransmitters like serotonin and norepinephrine. When sleep is fragmented, as seen in our PCOS subjects, this "natural pharmacy" fails, leaving the patient more vulnerable to pain. This creates the "Vicious Cycle" where hormonal imbalance causes poor sleep, and poor sleep further heightens pain sensitivity and metabolic stress. (31)

Impact on Quality of Life (HRQOL)

The profound reduction in SF-36 scores, particularly in the domains of Bodily Pain and **Vitality**, signifies the clinical burden of these comorbidities. Our results suggest that the "invisible" symptoms of PCOS fatigue and widespread pain are perhaps more debilitating than the "visible" symptoms like hirsutism or acne. The mental health scores reflect the psychological toll that chronic pain and sleep-deprived exhaustion take on these patients. (32)

Strengths and Limitations

A major strength of this study is the use of Pressure Algometry, providing an objective physiological marker of pain that removes the bias of subjective reporting. However, a limitation is the cross-sectional design, which prevents us from definitively stating whether poor sleep *causes* the pain or if chronic pain *prevents* deep sleep. Future longitudinal studies are required to establish a causal timeline. (33,34)

Clinical Recommendations

Based on these results, we recommend a shift in the clinical management of PCOS. Treatment should move beyond gynecological intervention to include:

1. **Sleep Screening:** Routine use of the PSQI in PCOS clinics.
2. **Pain Management:** Recognition of central sensitization to avoid over-reliance on local NSAIDs and consider therapies that target the nervous system (e.g., sleep hygiene, magnesium, or neuromodulators). (35,36)

11. CONCLUSION

Finally, this study confirms the presence of a strong "Sleep-Pain Axis" in women with PCOS. The relationship between sleep quality and pressure pain thresholds demonstrates that sleep architecture is a significant predictor of nociceptive function. Addressing sleep disturbances may provide a unique therapeutic window for lowering chronic pain and improving overall quality of life for women suffering from this complicated endocrine disorder.

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