



The Effect of Pyospermia on Male Infertility: A Cross-Sectional Study in Sri Lanka

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ABSTRACT

Background: Pyospermia, characterized by an elevated number of leukocytes in semen, is associated with oxidative stress and impaired sperm function. This study investigates the relationship between pyospermia and seminal parameters among infertile men in Sri Lanka.

Methods: A cross-sectional study was conducted among 105 male partners of infertile couples attending a fertility clinic in Sri Lanka. Semen samples were analyzed for volume, sperm count, motility, morphology, and pus cell count. Subjects were categorized into two groups: Group 1 (5-10 pus cells/HPF) and Group 2 (>10 pus cells/HPF). An age-matched control group of non-pyospermic men was included. Data were analyzed using the Wilcoxon signed-rank test and linear regression.

Results: No significant differences were found between the control and moderate pyospermia groups ($P > 0.05$). However, severe pyospermia (>10 pus cells/HPF) significantly reduced sperm motility ($P = 0.004$) and morphology ($P = 0.001$). Regression analysis revealed strong negative correlations between pus cell count and both motility ($R = 0.845, P = 0.001$) and morphology ($R = 0.900, P = 0.001$).

Conclusion: Pyospermia exceeding 10 pus cells/HPF adversely affects sperm motility and morphology, likely due to oxidative damage caused by leukocyte-derived reactive oxygen species (ROS). Early identification and management may improve fertility outcomes.

Keywords: Male infertility, Pyospermia, Sperm motility, Morphology, Oxidative stress

1. Introduction

Male infertility contributes to 30–40% of global infertility cases^[1]. Sperm quality determined by seminal volume, count, motility, and morphology is the cornerstone of male reproductive potential. Deviations from these parameters can lead to infertility. Pyospermia (or leukocytospermia) is defined as more than 1×10^6 leukocytes per ml of semen or > 5 pus cells per HPF^[2,3]. While low leukocyte levels provide immune protection, excessive levels promote inflammation and ROS generation, damaging sperm DNA, membranes, and mitochondria^[4]. ROS-induced stress leads to lipid peroxidation and loss of sperm motility^[5]. Globally, pyospermia prevalence ranges from 5% to 15% among infertile men^[6]. Despite global studies, Sri Lankan data are scarce. Hence, this study examines pyospermia's impact on seminal parameters in local infertile males.

2. Research Problem

Does pyospermia adversely affect seminal parameters such as volume, count, motility, and morphology?

3. Hypothesis

Pyospermia has a significant negative effect on seminal volume, sperm count, motility, and morphology.

4. Methods

4.1 Study Design and Ethics

This cross-sectional study adhered to the Declaration of Helsinki (1964). Institutional ethical approval and informed consent were obtained.

4.2 Study Population

A total of 105 male partners of infertile couples attending a fertility clinic in Sri Lanka (Gampaha district) were enrolled. Inclusion: men ≥ 18 years. Exclusion: systemic illness, substance abuse (e.g., marijuana), and hormonal or steroid therapy.

4.3 Sample Size Calculation

Sample size was calculated using the formula for prevalence studies ^[7]:

$$N = 4 Z_{\alpha}^2 P (1 - P) / D^2$$

Where $Z_{\alpha} = 1.96$ (for 95% confidence), P = estimated prevalence, and D = total width of confidence interval (0.125). Using $P = 0.08$ (male infertility prevalence), $N \approx 72$; for $P = 0.05$ (pyospermia prevalence), $N \approx 46$. The actual $N = 105$ exceeded both values, ensuring sufficient power.

4.4 Control Group

Age-matched non-pyospermic men from the same clinic, with female-factor infertility partners, served as controls.

4.5 Semen Collection and Analysis

Semen was collected after 3 days of abstinence and analyzed following WHO 2010 guidelines ^[8]:

- Volume: > 2 ml (normal)
- Sperm count: > 25 million/ml (normal)
- Motility: $\geq 50\%$ (normal)
- Morphology: $\geq 30\%$ (normal)
- Pus cells: ≤ 5 /HPF (normal)

5. Results

5.1 Moderate Pyospermia (5–10 pus cells/HPF)

No significant differences vs. control in volume (2.30 ± 0.40 vs 2.04 ± 0.43 ml, $P > 0.05$), count (65.3 ± 11.0 vs $69.6 \pm 13.1 \times 10^6$ /ml, $P > 0.05$), motility (51.2 ± 12.2 vs $61.7 \pm 12.7\%$, $P > 0.05$), or morphology (35.0 ± 8.6 vs $44.3 \pm 8.8\%$, $P > 0.05$).

5.2 Severe Pyospermia (>10 pus cells/HPF)

Severe cases showed significantly lower sperm count (34.3 ± 9.1 vs $69.6 \pm 13.1 \times 10^6$ /ml, $P = 0.006$), motility (40.2 ± 10.4 vs $61.7 \pm 12.7\%$, $P = 0.004$), and morphology (29.0 ± 6.6 vs $44.3 \pm 8.8\%$, $P = 0.001$).

Regression analysis revealed strong negative correlations between pus cell count and motility ($R = 0.845$, $R^2 = 0.714$, $P = 0.001$) and morphology ($R = 0.900$, $R^2 = 0.896$, $P = 0.001$).

6. Discussion

This study confirms that while moderate pyospermia exerts minimal influence, severe pyospermia markedly impairs motility and morphology. Similar patterns were observed globally ^[9–11]. ROS and proteolytic enzymes (e.g., elastase, myeloperoxidase) from leukocytes degrade sperm membranes and mitochondrial function, reducing ATP and motility ^[12, 13]. ROS-induced peroxidation alters head and tail morphology, disrupting fertilizing ability ^[14].

Elgozali et al. (2015) reported a direct relationship between pus cells > 10/HPF and reduced motility [15]. Sultan et al. (2012) found higher pus cell counts in teratozoospermic men. Khodamoradi et al. (2020) emphasized oxidative stress as the principal mediator [16]. The threshold phenomenon (>10 pus cells/HPF) supports the concept that inflammation must surpass a critical limit to induce measurable sperm damage [17]. Moderate inflammation may not disrupt spermatogenesis or seminal plasma antioxidants sufficiently to alter parameters [18]. Clinically, managing pyospermia through antibiotics, antioxidants, or anti-inflammatory therapy may improve semen quality [19]. Early detection can prevent progression and secondary oxidative damage [20].

7. Conclusion

Severe pyospermia (> 10 pus cells/HPF) significantly reduces sperm motility and morphology, likely via oxidative and enzymatic injury to sperm. Moderate pyospermia (5–10 pus cells/HPF) shows negligible impact. Integration of pyospermia assessment and management into infertility workups is recommended to enhance reproductive outcomes.

8. Limitations and Future Directions

Limitations include a modest sample size and the lack of microbial culture for etiology. Future work should employ molecular pathogen identification, oxidative stress biomarkers (e.g., MDA, SOD activity), and evaluate therapy responses to antibiotics or antioxidants..

9. References

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