



**19th International Conference on New Findings
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**Title: Discovering Dynamic Pathways of Semen Quality Decline: A
Longitudinal Cohort Study and Temporal Association Rule Mining
in Bandar Abbas, Iran**

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Abstract

Background: Male factor infertility is a major global reproductive health concern, accounting for approximately 50% of all cases. Traditional clinical diagnostics rely on static semen analysis guidelines (e.g., World Health Organization reference limits), which fail to capture dynamic, longitudinal fluctuations of semen parameters or map temporal cause-and-effect networks (the 'domino effect') of sperm decay over time. In region-specific clinics such as Bandar Abbas, environmental stressors (such as extreme heat and humidity) and localized systemic inflammation create unique regional subphenotypes that universal cutoffs do not adequately capture. **Objective:** To develop and evaluate a purely data-driven machine learning framework using Temporal Association Rule Mining (TARM) and local quartile discretization to discover longitudinal pathways and directed causal links in the decline of semen parameters in a regional cohort. **Methods:** A retrospective longitudinal cohort study was conducted on 18,217 clinical records from 3,012 unique male patients (each tracked over 4 consecutive years with at least 4 tests) attending a specialized fertility clinic in Bandar Abbas, Iran. Continuous parameters analyzed included volume, viscosity, progressive motility (PR), non-progressive motility (NP), morphology anomalies (head, neck, tail), semen WBC, blood ESR, and Sperm DNA Fragmentation Index (DFI). To bypass standard clinical cut-off biases, data-driven regional quartiles (25th and 75th percentiles) were calculated for local state discretization. An optimized TARM pipeline was deployed to extract sequential transition rules of the form {Antecedent}_t --> {Consequent}_{t+1}. Longitudinal causal directions were validated using multivariate Granger Causality models ($p < 0.05$). **Results:** The regional cohort exhibited significant baseline variations, with local quartile thresholds reflecting specific environmental adaptation: PR progressive motility (10.00% to 45.00%), head anomalies (95.00% to 98.00%), and DFI (4.00% to 5.00%). Granger Causality confirmed directed, longitudinal pathways from early systemic inflammation (elevated ESR at time t) to progressive motility decline and structural morphological defects at $t+1$ (F-statistic, $p < 0.05$). Sequential rule mining successfully identified the primary domino cascade of sperm deterioration: severe systemic inflammation acted as a primary temporal trigger ({ESR = High}_t), transitioning to severe structural head anomalies ({Head Anomalies = High}_{t+1}) with a Support of 6.46%, Confidence of 26.69%, and Lift of 1.468. Furthermore, neck anomalies ({Neck Anomalies = High}_t) strongly predicted head anomalies at $t+1$ (Support = 5.31%, Confidence = 28.22%, Lift = 1.552), which subsequently culminated in high genetic fragmentation ({DFI = High}_{t+1}) triggered by low progressive motility ({PR = Low}_t --> {DFI = High}_{t+1}: Support = 6.01%, Confidence = 23.50%, Lift = 1.204). **Conclusion:** This study presents a shift from static cross-sectional semen diagnostic guidelines to predictive patient trajectory forecasting. By utilizing pure data-driven regional thresholds and temporal association rules, clinicians can identify early warning triggers of sperm decay and determine optimal treatment windows, providing a powerful, personalized, and region-sensitive clinical decision-support tool in male reproductive medicine.



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Keywords: Male Infertility, Temporal Association Rule Mining, Longitudinal Cohort, Sperm DNA Fragmentation, Bandar Abbas

Introduction

Male factor infertility represents a substantial global public health challenge, contributing to approximately 50% of all infertility cases worldwide. Standard clinical diagnostics rely heavily on the World Health Organization (WHO) reference limits, which assess seminal parameters such as count, motility, and morphology in a static, cross-sectional manner. However, human spermatogenesis is a highly dynamic and continuous biological process. Standard semen analysis represents only a temporary snapshot, completely failing to capture the longitudinal fluctuations, biological degradation, or the complex cause-and-effect networks known as the 'domino effect' of cellular decay over time.

Furthermore, universal diagnostic thresholds suffer from geographical and environmental biases. In localized subtropical regions such as Bandar Abbas, Iran, extreme ambient temperatures, humidity, and regional environmental stressors can significantly trigger localized and systemic inflammatory responses. These factors contribute to a distinct regional reproductive sub-phenotype characterized by subclinical inflammation (elevated Erythrocyte Sedimentation Rate [ESR] and seminal White Blood Cell [WBC] counts) and high genetic vulnerability, which is completely misclassified under traditional global guidelines.

To address these critical limitations, this study presents a novel, data-driven machine learning framework that shifts the clinical paradigm from static diagnostics to temporal patient trajectory forecasting. By utilizing Temporal Association Rule Mining (TARM) and local quartile discretization, we systematically map the prospective pathways of sperm parameter decline. We utilize a highly robust, large-scale longitudinal cohort comprising 18,217 sequential records from 3,012 unique patients in Bandar Abbas, Iran, tracked consistently over four years. Longitudinal causal links are further validated using multivariate Granger Causality autoregressive models, establishing a powerful and personalized clinical decision-support tool.

Materials and Methods

Patient Cohort and Study Design

A retrospective longitudinal cohort study was conducted on patients attending a specialized andrology and fertility center in Bandar Abbas, Iran, between 2018 and 2024. Following the strict application of observational guidelines (STROBE), a highly robust cohort consisting of 18,217 clinical records from 3,012 unique patients was compiled. Each patient was tracked over four consecutive years, with a minimum of four standardized tests. Inclusion criteria comprised: male patients aged 18 to 60 years, with complete longitudinal records for semen parameters, inflammatory markers, and Sperm DNA Fragmentation Index (DFI). Standard specimen collection was maintained with an abstinence duration of 2 to 7 days, followed by complete liquefaction at 37°C for 30 to 60 minutes before standardized processing.

Laboratory and Clinical Parameter Assessment

Standardized clinical assessments were performed in a certified regional reference laboratory. The clinical parameters were classified into four primary domains: physical and macroscopic metrics (volume, viscosity), concentration and count metrics, motility dynamics (progressive motility [PR] and non-

progressive motility [NP]), morphological anomalies (head, neck, and tail anomalies), and inflammatory and genetic biomarkers (seminal WBC, blood ESR, and Sperm DFI representing single- and double-strand DNA breaks).

Temporal Feature Engineering and Local Discretization

To capture the biological delay (time lag) of spermatogenic cycles (~74 days), patient records were grouped by a unique anonymized identifier and sorted chronologically. We engineered mathematical Lag features representing the prior year's state ($X_{lag} = X(t-1)$) and Lead features representing the subsequent year's state ($X_{lead} = X(t+1)$). To bypass universal standard biases, continuous parameters were discretized into discrete states (Low, Medium, High) using purely data-driven, local quartile thresholds (25th and 75th percentiles) calculated directly from the 18,217 regional records of the Bandar Abbas cohort.

Temporal Association Rule Mining and Causal Validation

An optimized TARM pipeline was implemented in Python using a local launcher to mine sequential rules of the form $\{Antecedent\}_t \rightarrow \{Consequent\}_{t+1}$. Rules were evaluated and filtered using Support (Support $\geq 5\%$), Confidence, and Lift (Lift > 1.0). To mathematically validate directed causality from early systemic and seminal inflammation (ESR, WBC) to subsequent semen decay, multivariate Granger Causality tests were executed on patients with three or more temporal data points (F-test, $p < 0.05$).

Results

The baseline characteristics and regional thresholds calculated for the 18,217 records of the Bandar Abbas cohort are presented in Table 1 and Table 2, respectively. The data-driven local thresholds revealed marked differences from standard WHO cut-offs, reflecting the unique environmental and inflammatory sub-phenotypes of the local population. Notably, the local high-risk threshold (75th percentile) for Sperm DNA Fragmentation (DFI) was established at 5.00%, which is significantly lower than the standard global WHO limit of 15.00% or 30.00%, indicating a highly sensitive regional baseline.

Equations

To preserve the integrity and reproducibility of our machine learning pipeline, the core mathematical formulations used for causal validation (Granger Causality) and temporal rule evaluation (Confidence and Lift) are presented below.

1. $X_t = \sum_{i=1}^p \alpha_i X_{t-i} + \sum_{i=1}^p \beta_i Y_{t-i} + \varepsilon_t$
2. Confidence ($A_t \rightarrow B_{t+1}$) = $P(B_{t+1} | A_t)$
3. Lift ($A_t \rightarrow B_{t+1}$) = $P(B_{t+1} | A_t) / P(B_{t+1})$



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18 September 2026 -TBILISI GEORGIA

Tables and Figures

Table 1. Cohort baseline characteristics (n = 18,217 records from 3,012 patients)

Parameter (Biomarker)	Mean	Standard Deviation (SD)	Clinical Range
Blood ESR (mm/hr)	10.37	11.20	0.00 - 80.00
Seminal WBC ($\times 10^6/\text{mL}$)	1.73	2.83	1.00 - 100.00
Progressive Motility (PR, %)	28.94	20.31	0.00 - 94.00
Non-Progressive Motility (NP, %)	34.04	16.64	0.00 - 93.00
Head Morphological Anomalies (%)	95.32	6.42	0.00 - 100.00
Neck Morphological Anomalies (%)	62.22	19.69	0.00 - 99.50
Tail Morphological Anomalies (%)	21.64	13.19	0.00 - 99.50
Sperm DNA Fragmentation Index (DFI, %)	5.68	4.43	2.00 - 80.00

Table 2. Calculated local quartile thresholds for regional state discretization

Semen / Clinical Parameter	Low Threshold (25th Percentile)	High Threshold (75th Percentile)
Blood ESR (mm/hr)	4.00	11.00
Seminal WBC ($\times 10^6/\text{mL}$)	1.00	2.00
Progressive Motility (PR, %)	10.00	45.00
Non-Progressive Motility (NP, %)	23.00	45.00
Head Morphological Anomalies (%)	95.00	98.00
Neck Morphological Anomalies (%)	45.00	80.00



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18 September 2026 -TBILISI GEORGIA

Tail Morphological Anomalies (%)	14.00	26.00
Sperm DNA Fragmentation Index (DFI, %)	4.00	5.00

Granger Causality testing confirmed directed, longitudinal pathways from early systemic inflammation (elevated blood ESR at time t) to progressive motility decline and structural head morphology defects at t+1 (F-statistic, $p < 0.05$). Transitioning these temporal causal pathways into sequential association rule mining yielded highly structured rules (Support $\geq 5\%$, Lift > 1.0) shown in Table 3.

Table 3. Mined sequential association rules representing sperm quality decline pathways

Antecedent State (t)	Consequent State (t+1)	Support (%)	Confidence (%)	Lift
{Neck Anomalies = High}_t	{Head Anomalies = High}_{t+1}	5.31%	28.22%	1.552
{Head Anomalies = High}_t	{Neck Anomalies = High}_{t+1}	5.13%	28.34%	1.515
{Neck Anomalies = High}_t	{Blood ESR = High}_{t+1}	6.77%	35.98%	1.509
{Blood ESR = High}_t	{Head Anomalies = High}_{t+1}	6.46%	26.69%	1.468
{Head Anomalies = High}_t	{Tail Anomalies = High}_{t+1}	6.41%	35.36%	1.474
{Progressive Motility = Low}_t	{Sperm DFI = High}_{t+1}	6.01%	23.50%	1.204
{Sperm DFI = High}_t	{Progressive Motility = Low}_{t+1}	5.69%	29.06%	1.130

Discussion

The empirical findings derived from the 18,217 records of our longitudinal cohort provide profound insights into the multi-year progression of male reproductive pathology. By analyzing the temporal



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relationships, we successfully uncovered the clinical 'domino effect' where early triggers act as prospective drivers of subsequent parameter decay. Most notably, the rule linking elevated blood ESR at time t to high head anomalies at $t+1$ (Support = 6.46%, Confidence = 26.69%, Lift = 1.468) provides robust statistical evidence of a time-delayed spermatogenic insult driven by systemic inflammation. Chronic inflammation induces systemic oxidative stress, which subsequently impairs the highly sensitive spermatogenesis process within the testicular microenvironment, resulting in morphological defects in the following spermatogenic cycles.

Furthermore, our results demonstrate a strong bidirectional structural deterioration loop between head and neck anomalies over consecutive test intervals (Lift of 1.552 and 1.515, respectively). This sequence indicates that morphological damage is progressive and cumulative; structural instability in the neck region at year t significantly compromises morphological integrity, resulting in severe head anomalies in year $t+1$. Crucially, the sequential cascade culminating in severe genetic fragmentation ($\{ \text{Progressive Motility} = \text{Low} \}_t \rightarrow \{ \text{Sperm DFI} = \text{High} \}_{t+1}$; Lift = 1.204) highlights motility decline as an early-stage functional warning trigger. Reduced motility is indicative of cellular fatigue and mitochondrial dysfunction, which precedes double- and single-strand DNA breaks (DFI) in the sperm genome.

The calculated local thresholds (Table 2) underscore the necessity of establishing region-specific baselines. The regional high-risk threshold for Sperm DFI was established at 5.00%. In comparison with traditional clinical thresholds (e.g., standard WHO cut-offs which define DNA pathology at >15.00% or >30.00%), our localized data-driven threshold suggests that the local population in Bandar Abbas exhibits a highly sensitive, shifted baseline. This shift is likely driven by extreme regional heat and humidity, which exacerbate systemic oxidative stress and lower the threshold at which genetic damage manifests, requiring earlier clinical interventions.

Conclusion

This study presents a fundamental paradigm shift in male reproductive diagnostics, moving from traditional static, cross-sectional semen guidelines to predictive patient trajectory forecasting. By utilizing purely data-driven local thresholds and Temporal Association Rule Mining on 18,217 longitudinal records, we successfully mapped the dynamic pathways and sequential domino effects of sperm degradation. The discovered rules establish seminal and systemic inflammation as primary temporal triggers of subsequent structural and genomic decay. This approach provides clinicians with a powerful, personalized, and region-sensitive clinical decision-support tool to identify early-stage risk factors and optimize the timing of clinical interventions.

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