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Impaired Recovery Duri ng Congested Competition in an Elite Football Player: A Longitudinal Integrative Case Report

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Abstract

Background

Interindividual variability in recovery responses represents a central challenge in elite sports, particularly during congested competition periods. Traditional biomarkers provide limited predictive value when used in isolation, highlighting the need for integrative multimodal monitoring approaches.

Methods

This longitudinal case report describes a 28-year-old elite professional football player monitored over a 6-week congested competition period. Data collection encompassed heart rate variability (HRV; RMSSD), resting heart rate (RHR), sleep parameters, neuromuscular performance (CMJ), serial biochemical markers, and genetic profiling via high-density SNP array. All data were analyzed within a within-subject longitudinal framework.

Results

Progressive autonomic suppression was observed (RMSSD: 62 → 38 ms, − 39%; RHR: 56 → 65 bpm). Sleep duration fell below 6 hours pre-competition and CMJ declined by ~ 20%. Biochemical findings indicated substantial physiological stress: CK reached 710 U/L, CRP increased from 0.3 to 2.0 mg/L, testosterone fell from 663 to 322 ng/dL (− 51.4%), cortisol rose from 13 to 25 µg/dL (+ 92.3%), and the testosterone-to-cortisol ratio decreased by ~ 74.7%. Following a 3–4 week individualized intervention, partial recovery was observed across all domains.

Conclusion

This case illustrates a multifactorial model of impaired recovery in elite sport, in which autonomic imbalance, sleep disruption, accumulated load, and genetic susceptibility interact dynamically. Integrated monitoring combining physiological, behavioral, and genomic data may enhance individualized load management in high-performance environments.

1. Introduction

Interindividual variability in response to training load constitutes a central challenge in elite sport. Despite standardized training protocols, athletes exhibit markedly heterogeneous physiological adaptations, particularly regarding recovery capacity, fatigue accumulation, and injury susceptibility. Understanding the mechanisms underlying this variability is essential for implementing effective individualized load management strategies.

Traditional biomarkers—including creatine kinase (CK), cortisol, and testosterone—provide limited predictive value when interpreted in isolation. Daily HRV assessment via RMSSD has gained traction as a field-applicable index of cardiac autonomic modulation, with its value enhanced when interpreted alongside resting heart rate (RHR)—the two variables together capturing the reciprocal vagal-sympathetic dynamic more robustly than either alone [2, 3].

Despite these advances, the integration of genetic background with real-time physiological monitoring and training load data remains poorly explored in applied sports science. Polymorphisms in genes such as CLOCK and PER3 have been associated with circadian regulation and sleep phenotype; AMPD1 variants may influence energy metabolism during high-intensity efforts; and collagen-encoding genes (COL5A1, COL1A1) and inflammatory modulators (IL6, TNF, MMP3) have been linked to musculoskeletal injury susceptibility [14–17].

Genetic variants should not be interpreted as deterministic predictors. Rather, they function as a background susceptibility layer—modulating physiological responses to environmental stress without determining outcomes. Their clinical relevance emerges when integrated with longitudinal physiological data, not in isolation [15, 22].

This report presents a professional elite football player who exhibited progressive impaired recovery during a congested competition period, characterized through an integrative framework combining HRV–RHR monitoring, sleep analysis, CMJ performance, serial biochemical profiling, and genetic susceptibility profiling.

2. Methods

Data were collected using wearable monitoring technology, standardized neuromuscular testing, and serial biochemical assessment within a longitudinal within-subject framework over a six-week period.

2.1. Heart Rate Variability and Resting Heart Rate

Cardiac autonomic status was assessed each morning via a validated short-duration recording protocol using a chest-strap sensor (Polar H10; Polar Electro Oy, Finland) in the supine position, initiated after a brief stabilization interval. Recordings were processed in Kubios HRV software with low-threshold artifact filtering and manual quality review. RMSSD served as the primary HRV index. RHR was recorded simultaneously; the combined daily RMSSD–RHR trajectory was used as the primary index of autonomic status throughout the monitoring period.

2.2. Neuromuscular Performance (CMJ)

Daily neuromuscular readiness was evaluated using a bilateral countermovement jump (CMJ) protocol. Each session comprised three maximal efforts with hands placed on the iliac crests, preceded by a standardized activation sequence, with 45-second passive recovery intervals between jumps. The

highest recorded value per session was retained for longitudinal analysis. Mandatory assessments were scheduled on MD-2 and MD-1 relative to each competitive fixture.

2.3. Sleep Monitoring

Nocturnal sleep data were obtained continuously using a photoplethysmography-based ring device (Oura Ring; Oura Health Oy, Finland). Nightly outputs included total sleep time, sleep onset latency, wake-after-sleep-onset, and nocturnal heart rate. Data were reviewed in the context of travel schedules and competition timing.

2.4. Biochemical Assessment

Fasted morning venous blood draws were performed one to two times per week. The panel included serum CK, high-sensitivity CRP, total testosterone, morning cortisol, and additional metabolic indicators. The testosterone-to-cortisol (T/C) ratio was computed from absolute values as a composite index of anabolic-catabolic balance. All values were tracked longitudinally against the athlete's own pre-season baseline.

2.5. Genetic Analysis

Genetic profiling was performed using a high-density SNP array (VersaGene Full Array; DASA Laboratório, Brazil). Variants were selected a priori based on published associations in the sports genomics literature:

- CLOCK rs1801260 (C/T) — circadian regulation
- PER3 VNTR (4/5) — sleep phenotype and circadian preference
- AMPD1 rs17602729 (C/T) — purine nucleotide metabolism
- COL5A1 rs12722 (T/T) — type V collagen structure
- COL1A1 rs1800012 (G/T) — type I collagen structure
- IL6 rs1800795 (− 174 G/C) — interleukin-6 signaling
- TNF rs1800629 (− 308 G/A) — TNF-α inflammatory signaling
- MMP3 rs3025058 (5A/6A) — matrix metalloproteinase-3 and ECM remodeling

Genetic data were incorporated as a contextual susceptibility layer and were not used in isolation for clinical or training decisions.

2.6. Data Integration

All data were integrated within a within-subject longitudinal framework. Individual baseline values served as the primary comparator. Temporal variation, competition schedule, and multi-domain response to intervention were considered conjointly. Genetic findings were contextualized against the physiological phenotype rather than evaluated as independent predictors.

3. Case Presentation

A 28-year-old male professional football player (midfielder, Brazilian Campeonato Brasileiro Série A) with 15 years of structured competitive training history was monitored over a six-week period coinciding with a congested competition schedule (two matches per week), including frequent interurban and interstate travel.

The athlete reported persistent fatigue unrelieved by standard rest, subjective reduction in high-intensity performance capacity, delayed post-match recovery, and sleep disturbances particularly in the 24–48 hours preceding competition. His clinical history was notable for recurrent posterior chain injuries: proximal hamstring strains (2023 and 2026), adductor injury (2024), and soleus involvement. No acute illness, infection, or medication changes were reported during the monitoring period.

4. Results

Table 1 presents the longitudinal changes across all monitored domains.

Table 1
Longitudinal multi-domain monitoring data before and after intervention.

Variable	Baseline	Nadir (pre-intervention)	Post-intervention	Δ (%)
HRV (RMSSD, ms)	62	38	44	+ 15.8%
Resting HR (bpm)	56	65	62	−4.6%
Sleep duration (h)	~ 7–8	< 6	~ 7–7.5	+ 15–25%
Sleep latency	Normal	Increased	Reduced	—
CMJ height (%)	100	~ 80	~ 88–92	+ 8–12%
Creatine kinase (U/L)	—	710	Decreased	—
Cortisol (μg/dL)	13	25	Partial normalization	+ 92.3%
Testosterone (ng/dL)	663	322	Partial normalization	−51.4%
T/C ratio	51.0	12.9	—	−74.7%
CRP (mg/L)	0.3	2.0	0.7	—

HRV = heart rate variability; RMSSD = root mean square of successive differences; RHR = resting heart rate; CMJ = countermovement jump; CK = creatine kinase; CRP = C-reactive protein; T/C = testosterone-to-cortisol ratio.

4.1. Autonomic Function

RMSSD decreased from 62 ms at baseline to 38 ms (− 39%), with partial recovery to 44 ms post-intervention (+ 15.8%). Concomitantly, RHR increased from 56 to 65 bpm at nadir, with partial reduction to 62 bpm following intervention. The reciprocal RMSSD–RHR trajectory is consistent with accumulated physiological stress and incomplete autonomic recovery.

4.2. Sleep

Total sleep time fell below 6 hours on multiple pre-match nights, with prolonged sleep latency and elevated nocturnal heart rate. Following sleep optimization, total sleep time recovered to approximately 7–7.5 hours with improved efficiency and reduced latency.

4.3. Neuromuscular Performance

CMJ performance declined progressively to approximately 80% of baseline (~ 20% decrement). Post-intervention values recovered to 88–92% of baseline (+ 8–12% relative to nadir), indicating partial but incomplete neuromuscular restoration.

4.4. Biochemical and Endocrine Profile

CK reached 710 U/L and CRP increased from 0.3 to 2.0 mg/L, followed by reduction to 0.7 mg/L post-intervention. Testosterone decreased from 663 to 322 ng/dL (− 51.4%) while cortisol increased from 13 to 25 µg/dL (+ 92.3%), yielding a T/C ratio decline from 51.0 to 12.9 (− 74.7%), consistent with severe catabolic imbalance.

4.5. Genetic Profile

Genotyping identified variants across multiple biological axes: CLOCK rs1801260 (C/T) and PER3 VNTR (4/5) in the circadian axis; AMPD1 rs17602729 (C/T) in the metabolic axis; COL5A1 rs12722 (T/T), COL1A1 rs1800012 (G/T), and MMP3 rs3025058 (5A/6A) in the connective tissue axis; and IL6 rs1800795 and TNF rs1800629 in the inflammatory axis.

5. Intervention

A 3–4 week individualized, multidisciplinary intervention was implemented.

5.1. Load Management

Global external training load was reduced by approximately 30–40%, with specific reductions in high-speed running (> 21 km/h), sprint distances (> 25 km/h), and high-intensity accelerations/decelerations, guided by GPS metrics and daily HRV–RHR trends.

5.2. Training Modification

High metabolic load sessions were replaced with lower-density technical-tactical activities. Periodization was adjusted to ensure adequate inter-stimulus recovery within the congested match schedule.

5.3. Eccentric Load Management

High-intensity eccentric loading was suspended for 7–10 days, followed by graduated reintroduction with individualized progression criteria based on HRV, RHR, and CMJ response.

5.4. Sleep Optimization

A structured sleep hygiene protocol was implemented: caffeine restriction after 14:00 h; consistent sleep and wake timing; blue-light blocking in the evening; and travel-day napping protocols. Melatonin (1 mg) was administered 60–90 minutes before sleep as a chronobiotic adjunct to support circadian alignment.

6. Discussion

This case provides a longitudinal, integrative characterization of impaired recovery in an elite football player under congested competition. The central contribution is methodological: individualized multimodal integrated monitoring—rather than any single biomarker—provides the most comprehensive assessment of athlete physiological status and guides effective intervention.

6.1. Autonomic Imbalance: HRV and RHR as Complementary Markers

The reciprocal RMSSD–RHR trajectory constitutes a robust autonomic signal. The combined HRV–RHR longitudinal trend is more informative than either variable in isolation and is increasingly recognized as the preferred autonomic index in applied sports physiology monitoring [3, 21]. The persistence of deviation post-intervention indicates that full autonomic recovery was not achieved, underscoring the severity of accumulated physiological load.

6.2. Sleep Disruption as a Central Mediator

Pre-competitive sleep disruption—recurring across multiple match nights—likely functioned as both a consequence and an amplifier of autonomic dysregulation. Elevated nocturnal heart rate, prolonged sleep latency, and reduced total sleep time suggest an anticipatory sympathetic response potentially amplified by travel-induced circadian misalignment. This pattern likely contributed to the failure of overnight recovery to offset accumulated daily physiological strain [7, 8].

6.3. Neuromuscular Decline

The ~ 20% CMJ decline is clinically meaningful, particularly given the athlete's recurrent posterior chain injury history. The partial post-intervention recovery (88–92% of baseline) suggests that neuromuscular readiness had not been fully restored—a relevant consideration for return-to-full-training decisions [9, 10].

6.4. Endocrine and Inflammatory Profile

A testosterone reduction of 51.4%, cortisol elevation of 92.3%, and T/C ratio decline of 74.7% constitute objective evidence of a severe catabolic shift [13]. The simultaneous CRP elevation (0.3 → 2.0 mg/L) represents a transient low-grade inflammatory response attributable to accumulated stress rather than immune pathology. Partial normalization of all markers post-intervention is consistent with the HRV, RHR, and CMJ trajectories, reinforcing the coherence of the integrated physiological model.

6.5. Genetic Susceptibility: Contextual Mechanistic Role

The genetic data provide biological plausibility for the observed phenotype, not causal explanation. The center of this work is individualized integrated physiological monitoring; genetics contextualizes, helps explain susceptibility, and provides mechanistic plausibility. This is the epistemologically mature role of genetics in applied sports science today [15, 22].

CLOCK and PER3 variants represent the strongest genetic axis given the athlete's documented sleep phenotype. AMPD1 variants provide a plausible metabolic substrate for elevated fatigue and CK under repeated high-intensity efforts. COL5A1, COL1A1, and MMP3 variants are contextually consistent with the recurrent posterior chain injury history. IL6 and TNF variants provide plausibility for the sustained CRP elevation and prolonged recovery window.

6.6. Limitations

The single-case design precludes causal inference and limits generalizability. Confounders including nutritional status, psychological stress, and travel burden were not systematically quantified. Wearable devices carry inherent measurement variability. Post-intervention absolute CK values were not fully quantified. Genetic findings remain exploratory and non-deterministic.

7. Conclusion

This case demonstrates that impaired recovery in elite sport emerges from the dynamic interaction of training load, sleep disruption, autonomic imbalance, endocrine stress, and genetic susceptibility. No single biomarker provides adequate sensitivity in isolation; integrated multimodal monitoring within a longitudinal within-subject framework is required.

The quantified multi-domain data—RMSSD declining 39%, RHR increasing 16%, T/C ratio decreasing 74.7%, CRP rising 6.7-fold, CMJ dropping 20%—converge to describe a coherent and clinically actionable physiological state. Genetic profiling contributed contextual mechanistic plausibility consistent with the observed phenotype, without assuming deterministic or causal roles.

This approach represents a translational model for precision load management and injury risk reduction in high-performance environments.

Declarations

Ethics approval and consent to participate: The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from the athlete.

Consent for publication: The athlete provided written consent for anonymized publication of all data.

Availability of data and materials: Available from the corresponding author upon reasonable request.

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Authors' contributions: RSK: conceptualization, data collection, analysis, interpretation, and manuscript drafting. RenSK: biochemical data collection and analysis, manuscript revision. RESK: genetic analysis, data interpretation, and manuscript revision. All authors approved the final manuscript.

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