Glutathione

Reported Symptom Changes During Intramuscular Glutathione Use

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EVIDENCE TYPE

Retrospective, uncontrolled N-of-1 human case report. This is not a non-clinical experiment or controlled clinical trial.

MANUSCRIPT STATUS

Prepared for clinical and scientific review. Publication consent is not documented. This manuscript has not been peer reviewed.

EDITORIAL NOTE

The eight-page manuscript follows unchanged. This repository edition adds a cover and page numbering to the watermarked RTP Research report. Observational findings do not establish efficacy or safety.

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Reported Symptom Changes During Intramuscular Glutathione Use

A retrospective N-of-1 observational case report
January to October 2026 | Prepared 20 October 2026

Abstract

Background. Glutathione participates in cellular antioxidant defense. This report describes self-reported symptoms during intramuscular reduced glutathione exposure and examines historical laboratory context without treating temporal association as evidence of causation.

Case. A male participant, aged 35 at initiation and 36 at follow-up, reported starting 100 mg/week in January 2026 and titrating to 400 mg twice weekly during an approximately January-April initial phase. After approximately week 12, he reported maintenance at 300 mg once weekly. Facial flushing and hand/joint swelling reportedly resolved within about two weeks, eye redness and itching began to subside in the same interval, and white nail appearance improved over about six weeks. Energy and focus were first perceived to improve the day after the first injection and were later characterized as a sustained secondary benefit. No recurrence of the reported target symptoms or noticeable adverse effects was reported through the October 2026 interview cutoff.

Interpretation. Alcohol intake fell from more than 20 drinks weekly to approximately 6-10 beers weekly during the first two weeks, creating a major concurrent exposure change. Tirzepatide and other incompletely dated therapies further limit attribution. There were no original prespecified objective endpoints, no verified product certificate of analysis, and no contemporaneous symptom measurements. Routine laboratory results do not establish liver recovery or enhanced acetaldehyde clearance. These observations are hypothesis-generating, not a controlled trial or evidence of treatment efficacy.

Scope and source method

This manuscript integrates all five available glutathione interview rounds, the participant's current exposure summary, original April 2024, December 2025 and August 2026 laboratory reports, and the existing lab progress report. The KPV case report provides a style reference and relevant overlapping history. Calendar dates for dose changes and symptom resolution remain approximate. No values were imputed, no statistical hypothesis tests were performed, and no future laboratory values were modeled. Personal identifiers are withheld.

Case presentation and exposure

Before glutathione initiation, the participant described alcohol-associated facial flushing, swollen hands or joints, eye redness and itching, and white fingernails. The whitening reportedly involved the entire nail without horizontal lines. No examination, photographs, formal nail diagnosis, documented liver disease diagnosis, or diagnostic explanation for the symptom cluster was supplied.

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Period	Reported exposure and context
January 2026	Reduced glutathione initiated intramuscularly at 100 mg/week. Exact start day is unknown.
Initial phase through approximately April	Titrated to 400 mg twice weekly, totaling 800 mg/week. Intermediate amounts and dates are unavailable.
After approximately week 12	Maintenance 300 mg once weekly; day of administration varies. Continued through October follow-up.
First two weeks	Alcohol reduced from >20 drinks/week to 6-10 beers/week; liquor eliminated. Beer volume and alcohol concentration were not recorded.
Concurrent treatment	Tirzepatide confirmed in Round 5; its precise initiation and dose history were not supplied in the glutathione interviews.

The January-April description and the transition after week 12 are approximate recollections, not an exact four-month dosing log. The interview-specific 300 mg weekly maintenance record takes precedence here over the standing project protocol, which lists a different glutathione frequency. Total cumulative exposure cannot be calculated reliably.

Product characterization

The participant identified a Fuan Pharmacy product labeled 1500 mg and described it as reduced glutathione and “pharma grade.” These are participant-reported product claims. No original certificate of analysis (COA), batch verification, independent identity or potency testing, sterility/endotoxin testing, or corroborating product documentation was supplied. The 1500 mg label is not evidence of the amount actually administered. Preparation, storage, injection volume and administration records were unavailable.

Other exposures and record reconciliation

The prior KPV report describes semaglutide from October 2025 to March 2026, tirzepatide from March 2026, GHK-Cu around May, and KPV beginning around June. These dates are secondary self-report and were not independently confirmed in this interview. The broader project lists multiple hormonal and peptide exposures, but the current regimen cannot be assumed to have applied throughout January-October. A complete dated medication history remains unavailable.

Reported outcomes and maintenance durability

Observation	Reported timing	Evidence and qualification
Facial flushing	About two weeks	Resolved; no return of redness reported with subsequent drinking.
Hand and joint swelling	About two weeks	Reported resolution; no examination or circumference measurement.
Eye redness and itching	First two weeks	Began to subside; exact date of complete resolution not established.

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Observation	Reported timing	Evidence and qualification
White nail appearance	About six weeks	Pink color reportedly returned gradually from base toward tip; no serial photographs.
Energy and focus	Day after first injection	Later described as secondary, stable across the week, with less grogginess.
Clearer skin	A couple of weeks	Gradual subjective change rather than an immediate response.
Maintenance	Through October 2026	No reported recurrence of target symptoms; dosing day varied.
Tolerability	Reported exposure period	No adverse effects noticed or reported; no structured safety surveillance.

During Rounds 3-5, the participant continued to report no maintenance-phase symptoms and no flushing despite ongoing alcohol consumption. He perceived the consistent weekly total as more important than the weekday of administration. This describes his experience; it does not establish pharmacokinetic equivalence of schedules, a minimum effective dose, or persistence after discontinuation. There was no withdrawal phase, blinded assessment, or standardized alcohol challenge.

Participant perspective

In Round 1, the participant attributed symptom improvement to glutathione because earlier products had provided little or no perceived relief. Later rounds clarified the gradual skin and nail changes, early perceived energy change, and sustained secondary energy/focus benefit. This attribution is preserved as the participant's perspective rather than adopted as the report's causal conclusion. Questions about other exposures and research documentation were not fully answered; unanswered items are treated as missing data.

Overlapping histories remain unresolved

The KPV report attributes relief of substantial joint swelling to KPV in June 2026 and places an alcohol reduction in June, while the glutathione interviews describe swelling resolution and alcohol reduction within two weeks of January initiation. These may concern different episodes or symptom components, but the supplied records do not establish that distinction. The discrepancy is retained explicitly. The two reports must not be counted as independent confirmation of either intervention's effect.

Historical laboratory comparison

Collection dates, rather than filenames or report dates, are used below. April 2024 and December 2025 precede glutathione initiation. August 2026 was collected during reported maintenance. These are contextual routine tests, not prospectively defined glutathione endpoints. [S1-S4]

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Marker and unit	18 Apr 2024	15 Dec 2025	11 Aug 2026
AST (U/L)	30	31	23
ALT (U/L)	23	22	24
Alkaline phosphatase (U/L)	42 low	40 low	38
Total bilirubin (mg/dL)	0.4	0.5	0.7
Albumin (g/dL)	4.5	4.5	4.2
Total protein (g/dL)	6.9	7.3	6.9
Creatinine (mg/dL)	1.17	1.06	1.11
eGFR (mL/min/1.73 m^2)	84	94	88
BUN (mg/dL)	15	10	12
Glucose (mg/dL)	101	88	74
HbA1c (%)	NM	NM	4.8
Total cholesterol (mg/dL)	191	186	NM
Triglycerides (mg/dL)	171	64	NM
HDL cholesterol (mg/dL)	62	77	NM
LDL cholesterol (mg/dL)	100	97	NM
ApoB (mg/dL)	NM	NM	68
WBC (10^3/µL)	5.7	4.6	6.8
Platelets (10^3/µL)	256	293	294

NM means not measured or reported in the available panel, not zero. IU/L and U/L are harmonized as U/L. Low ALP flags reflect Labcorp intervals of 44-121 in 2024 and 47-123 in 2025; August Quest's interval was 36-130. The 2024 glucose was flagged high, but the original report does not specify fasting; the participant previously described draws as fasting. December and August reports explicitly document fasting.

This is a selected laboratory comparison rather than a complete safety panel. Primary reports govern transcription and reference flags. The August source filename contains an earlier date, but the report itself records collection on 11 August 2026. No September values are substituted into the requested August comparison.

Interpretation and scientific context

What the laboratory record supports

AST changed from 31 to 23 U/L between December and August, while ALT changed from 22 to 24 U/L; both were within their source intervals at all three dates. Bilirubin and albumin also remained within source intervals. There is no pretreatment transaminase elevation that

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subsequently normalized, and the mixed changes do not demonstrate a treatment effect. Normal aminotransferases do not exclude steatosis or clinically significant fibrosis. No imaging, elastography, biopsy, or original liver-specific outcome assessment was supplied. [S1-S4, 4]

Triglycerides declined from 171 to 64 mg/dL and HDL rose from 62 to 77 mg/dL before January 2026, so those improvements cannot be attributed to glutathione. August lacks a comparable lipid panel. Creatinine/eGFR differences are descriptive and do not establish renal benefit or injury from this exposure. WBC is nonspecific; no serial CRP, ESR, glutathione redox ratio, or oxidative-stress assay was available. Glucose changes cannot be separated from incretin therapy and other changes.

Glutathione and oxidative stress

Glutathione is an endogenous antioxidant involved in peroxide handling and electrophile defense. Alcohol metabolism produces acetaldehyde and can generate oxidative stress, providing biological plausibility for studying antioxidant pathways. Plausibility does not show that an injected product reaches the relevant cellular compartment or improves the symptoms in this case. Alcohol dehydrogenase converts ethanol to acetaldehyde; aldehyde dehydrogenase converts acetaldehyde to acetate. These distinct steps should not be equated with antioxidant activity. In ethanol-fed rats, impaired mitochondrial glutathione uptake and increased susceptibility to oxidant stress have been demonstrated; this preclinical finding is not evidence of clinical benefit from intramuscular supplementation. [1, 7]

In 54 nonsmoking adults, a six-month randomized trial of oral glutathione at 250 or 1000 mg/day reported increased glutathione stores in measured compartments. It did not test this intramuscular schedule, alcohol-associated flushing, or nail changes. An open-label NAFLD pilot enrolled 34 participants, used lifestyle intervention followed by oral glutathione 300 mg/day for four months, and reported an ALT reduction among completers. Its uncontrolled design, disease population and route limit applicability here. [2, 3]

Acetaldehyde hypothesis

A 2024 randomized crossover study in 40 participants tested oral yeast extract containing 50 mg glutathione with an alcohol challenge. It reported lower serum acetaldehyde concentrations, but no significant between-group difference in hangover questionnaires. The formulation and acute setting differ from this case. Lower measured concentrations do not by themselves distinguish reduced formation from increased clearance or establish a general effect of purified injected glutathione. [5]

Accordingly, glutathione is not proven to accelerate acetaldehyde clearance in this participant or with the reported intramuscular regimen. No ethanol or acetaldehyde kinetics, ALDH activity or genotype was measured. Absence of flushing does not demonstrate normalized alcohol metabolism, protection against alcohol-related injury, or safe alcohol tolerance. [1, 5, 6]

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Limitations and conclusions

The main limitations are retrospective recall, one participant, no control or blinded comparison, no original objective endpoints, incomplete dosing and co-exposure records, no independent symptom confirmation, unverified product composition, and no COA. Alcohol reduction occurred during the same early interval as reported improvement and is a major alternative explanation. Changes in diet, sleep, weight, activity, seasonal exposure and expectation were not quantified. Conflicting symptom and alcohol timelines across the KPV and glutathione accounts further weaken attribution.

Nail color and peripheral swelling are nonspecific observations. They cannot be used to diagnose previous liver failure or establish liver regeneration. The reported six-week nail change was not measured, and no clinical classification of the whitening is possible from text alone. Sustained perceived energy/focus improvement is meaningful to the participant but is not evidence of objective cognitive enhancement; no validated cognitive, fatigue or sleep instrument was administered.

No adverse effects were reported, but the observation period and ascertainment method cannot establish safety or exclude subclinical, delayed or uncommon harm. The report does not recommend the described product or regimen. A future observational record could improve interpretability through a dated exposure diary, standardized symptom ratings, serial photographs, verified product documentation and clinician-directed laboratory or liver assessment. Such measurements would establish new observations, not reconstruct missing baseline endpoints. Deliberate alcohol rechallenge is not needed to document the current account.

Conclusion

The participant reported improvement in flushing, swelling, eye symptoms, nail appearance, skin clarity and secondary energy/focus during intramuscular glutathione use, with no recurrence of target symptoms reported through October 2026 on 300 mg weekly maintenance. The contemporaneous alcohol reduction, concurrent therapies, incomplete objective documentation and overlapping histories prevent causal inference. Historical laboratory findings do not establish reversal of liver disease, and this report does not demonstrate accelerated acetaldehyde clearance. The case supports a research question rather than a therapeutic conclusion.

Ethics and disclosures

Prepared at the participant's request from supplied interviews and records. This document is an observational case narrative, not a registered trial, investigator-directed intervention or peer-reviewed publication. Written publication consent, author affiliations, funding declarations, conflicts of interest and any applicable institutional determination have not been documented. Preparation at the participant's request is not represented as formal publication consent. Branding does not imply institutional approval. Identifiers, addresses and specimen identifiers are excluded from the manuscript.

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Data availability

The participant retains the underlying private laboratory records and interview history. No public dataset is asserted. The laboratory table is selectively scoped at the participant's request; full source reports remain necessary for comprehensive clinical review. Literature verification was performed on 20 October 2026 using PubMed records and official source material; this was a focused review, not a systematic review.

References and source notes

[1] National Institute on Alcohol Abuse and Alcoholism. Alcohol Metabolism. Official explanatory source. Accessed 20 October 2026. https://www.niaaa.nih.gov/publications/alcohol-metabolism

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Private source ledger

[S1] 2024 Labs.html. Labcorp collection 18 April 2024. Chemistry page 1; lipids and blood counts pages 2-3. Fasting not specified.

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[S2] Dec2025 Labs.html. Labcorp collection 15 December 2025, fasting yes. Chemistry page 1; lipids and blood counts pages 2-3.

[S3] 2026-08-09 23_13_23.345496+00-results.pdf. Quest collection 11 August 2026, fasting yes. Chemistry, ApoB and A1c page 1; blood counts page 2.

[S4] lab-progress-report.pdf. Prepared 15 October 2026; used for reconciliation against original reports, not as a replacement for primary records.

[S5] Write KPV Research Paper conversation. Glutathione interview Rounds 1-5, retrieved in full; participant's continuation request supplies route, initial titration range and January-April period.

[S6] RTP_Research_KPV_Case_Report_Watermarked.pdf. Dated 17 October 2026. Style reference and secondary co-exposure history; conflicting timelines are explicitly retained in this report.

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