



Case Report

Mitochondrial Insufficiency and Microvascular Dysfunction in a Post-Vaccination Long COVID Patient

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Abstract

Following the initial resolution of a post-vaccination mitochondrial insufficiency challenge, persistent multi-system limitations led to the hypothesis that an overlapping microvascular process was impeding complete clinical recovery. Recent biomedical literature suggests that SARS-CoV-2 pathogenesis may involve anomalous amyloid fibrin microclots capable of obstructing the microvasculature. This case report presents a longitudinal, self-experimental evaluation of an advanced five-component protocol, designated P3C, which builds upon a previously published mitochondrial-insufficiency framework by integrating the oral fibrinolytic enzyme Nattokinase. Executed via a structured challenge-and-rechallenge methodology by an independent retired investigator, the therapeutic transition from metabolic support alone to a combined metabolic-microvascular targeting regimen resulted in a rapid, profound escalation of quality-of-life parameters from a plateaued 6–7/10 to a sustained 9.5–10/10. Objective functional markers corroborated this clinical response, demonstrating a 22.5% to 27% improvement in forced expiratory volume in 1 second (FEV1) and an 80% increase in ambulatory distance without adverse effects. These outcomes provide hypothesis-generating evidence indicating that a dual-barrier model—simultaneously addressing mitochondrial energy depletion and systemic microvascular congestion—warrants controlled clinical evaluation as an accessible therapeutic strategy for post-acute sequelae of COVID-19.

Keywords: Long COVID; Microvascular disease; Endothelial inflammation; Fibrinoid microclots; Nattokinase; Mitochondrial dysfunction; PASC; Fibrinolysis

Introduction

Post-acute sequelae of COVID-19 (PASC), or Long COVID, represents a massive and expanding global public health crisis with few evidence-based therapeutic interventions available. Emerging clinical literature indicates that the underlying pathogenesis is highly complex, involving a multi-system convergence of cellular metabolic failure and vascular endothelial disruption [1,2,11,12]. Specifically, viral interaction with the vascular endothelium is reported to initiate an inflammatory cascade, precipitating endothelial injury and anomalous thromboembolic presentations

[5]. Biomedical investigations have identified persistent, anomalous fibrinoid microclots in the plasma of PASC patients that exhibit an inherent resistance to conventional, endogenous fibrinolytic pathways [1,2].

A significant clinical deterioration occurred within 24 hours of receiving a fifth COVID-19 vaccine booster dose in a patient who had largely recovered from a primary viral infection. Initial therapeutic attempts utilizing isolated metabolic support successfully mitigated symptoms of cellular energy failure but resulted in a persistent recovery plateau. To resolve this stagnation, a novel protocol (P3C) was engineered to introduce systemic microvascular targeting alongside metabolic stabilization. This case study details the clinical progression, longitudinal outcomes, and systemic implications of this dual-barrier therapeutic model,

serving as an extension of a previously peer-reviewed and published report on post-vaccination mitochondrial insufficiency [11].

Case Presentation

A highly disabled patient presenting with severe multi-system post-vaccination sequelae was monitored closely across multiple functional dimensions using daily objective and subjective metrics. The clinical timeline and persistent multi-system symptomatology demonstrated significant phenotypic overlap with post-viral Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS), yet were uniquely characterized by an acute, fulminant deterioration following a booster immunization. Prominent clinical manifestations included profound systemic fatigue, balance disturbances, transient palpitations, cognitive cloudiness, and severe memory impairment.

Magnetic resonance imaging (MRI) of the brain previously demonstrated cerebral microvascular ischemia and associated white-matter alterations, while positron emission tomography (PET) indicated focal amyloid accumulation. These structural and metabolic findings were highly consistent with chronic impairment of cerebral blood flow and served as the secondary diagnostic foundation for developing the advanced protocol. In contrast, routine hematological coagulation profiles and standard clinical diagnostic evaluations remained entirely within normal parameters, underlining the necessity for specialized diagnostic modeling in complex PASC presentations.

Diagnostic Assessment

Based on the patient's multi-system symptom topology and structural imaging findings, a cohesive pathophysiological mechanism was modeled to guide intervention. In this dual-barrier framework, viral-induced endothelial injury initiates a prothrombotic cascade, giving rise to anomalous fibrinoid microclots that are structurally resistant to normal fibrinolysis [1,2,5,9]. The subsequent accumulation of these persistent microclots within systemic capillary beds causes widespread microvascular obstruction and localized tissue hypoxia [17,18]. This tissue-level oxygen deprivation directly impairs cellular mitochondrial respiration and downstream energy production, compounding a pre-existing metabolic barrier where viral-induced enzymatic activation (PARP and CD38) has already depleted intracellular NAD⁺ reserves [6,11]. This localized oxygen delivery limitation combined with severe nutrient/NAD⁺ depletion provides a mechanistically coherent explanation for the simultaneous presentation of severe musculoskeletal, neurological, and pulmonary symptoms despite largely unremarkable standard laboratory blood panels.

Therapeutic Intervention

Following approximately three months of stable but plateaued symptom management utilizing metabolic support alone, the patient initiated an advanced clinical experiment to address suspected microvascular obstruction. On April 10, 2026, the underlying metabolic regimen—consisting of highly specific daily doses of Ubiquinol, Huperzine-A, Vitamin B⁺ Complex, and Nicotinamide Riboside (NR-500)—was maintained uniformly. To target the suspected vascular barrier, an oral, highly purified fibrinolytic enzyme, Nattokinase (NSK-SD), was introduced to the protocol at a dosage of 2,000 fibrinolytic units (FU) administered twice daily (morning and afternoon).

Crucially, systematic challenge-and-rechallenge testing demonstrated that the absolute delivery vehicle for this combined protocol was fresh-squeezed orange juice. The substitution of standard water, pasteurized commercial orange juice, or any alternate fluid substrate repeatedly disabled the therapeutic efficacy of the P3C protocol, resulting in a rapid return of baseline symptoms. Concurrently, a stable baseline of required prescription medications, including Nortriptyline, Lisinopril, Nifedipine, and scheduled Vitamin B12 injections, was maintained completely unchanged across all protocol transitions.

Follow-up and Outcomes

Prior to the integration of Nattokinase, the patient's functional recovery had completely plateaued at a self-assessed score of 6–7 on a 10-point visual analogue scale, with objective home spirometry demonstrating a severely restricted forced expiratory volume in 1 second (FEV1) of approximately 1,500 to 1,550 mL. Following the initiation of Protocol P3C on April 10, 2026, a profound multi-system clinical response occurred within 24 to 72 hours. By April 11 (4 days post-intervention), the patient's functional score rose sharply to 9/10, marked by an immediate clearance of cognitive cloudiness, restored balance, and the complete resolution of a chronic spatial 'drift' phenomenon.

Objective metric verification followed rapidly. Within seven days of adding Nattokinase, home spirometry recorded an FEV1 of 1,900 mL, representing an immediate 22.5% improvement over the pre-Nattokinase baseline. Long-term follow-up through late August 2026 demonstrated sustained, stable clinical recovery. The patient's functional score consolidated at 9.5–10/10, and objective spirometry systematically stabilized above 2,000 mL, achieving peaks of 2,300 mL (a 27% peak improvement). Maximum ambulatory distances expanded by 80%, up to 2,500 feet, without the induction of post-exertional weariness. Orthostatic vital signs remained highly stable (blood pressure: 125/65 mmHg; pulse: 96 bpm), matching historical parameters. Furthermore, compliance

with the daily regimen was excellent, and the patient experienced zero adverse clinical events, spontaneous bleeding, bruising, or gastrointestinal distress despite the concurrent use of prescription antihypertensive therapies under ongoing medical supervision.

Discussion

This n=1 longitudinal self-case report cannot definitively establish a causal relationship between the P3C protocol and the documented clinical recovery; however, the striking temporal correlation and the objective improvement across disparate organ domains provide strong, hypothesis-generating evidence. The abrupt therapeutic transition from a stable symptom plateau on metabolic support alone to near-total functional restoration upon adding Nattokinase strongly supports a dual-barrier pathophysiological model. Addressing cellular metabolic failure independently appears insufficient if the capillary beds remain obstructed by persistent fibrinoid microclots that restrict essential oxygen and substrate delivery to tissue beds.

The rapid, simultaneous resolution of pulmonary restriction, cerebral ischemia symptoms, and musculoskeletal drainage within days of initiating Nattokinase points toward a global, systemic therapeutic process rather than an organ-specific effect. While alternative influences such as natural fluctuation, pacing modifications, unmeasured sleep adaptations, or a robust placebo response cannot be definitively excluded, the substantial and sustained shift in objective spirometry (FEV1) suggests a concrete physiological modification.

Pharmacological caution is strictly warranted. Nattokinase is an active fibrinolytic agent capable of altering systemic coagulation pathways. Its administration alongside established antihypertensive or antithromboembolic medications (such as Lisinopril and Nifedipine) introduces a risk of therapeutic synergy or hemorrhagic complications. Consequently, the clinical deployment of the P3C protocol or similar natural enzyme regimens must always be restricted to implementation under direct, continuous professional medical supervision.

Conclusion

The systematic three-protocol progression detailed in this case report (P3A metabolic rescue, P3B optimization, and P3C microvascular targeting) provides a highly coherent, reproducible framework for future controlled clinical investigations. For individuals currently living with the profound disabilities of PASC, these findings offer a valid rationale for therapeutic optimism, demonstrating that severe multi-system post-vaccination restrictions may be partially reversible through targeted metabolic and microvascular interventions undertaken with professional medical oversight.

Declarations

Conflicts of Interest: The author declares no conflicts of interest. No commercial or institutional funding was received for this work. All evaluation components consist of generic, over-the-counter substances, and the author holds no financial stake in any pharmaceutical or supplement manufacturer.

Ethical Oversight and Patient Consent: This report describes a self-directed observation and self-experimentation protocol where the investigator serves as the sole subject. No external human subjects research was conducted. In accordance with standard academic and institutional policies, formal institutional review board (IRB) review is waived for an independent, unaffiliated researcher conducting self-observation. The author provides full, unconstrained informed consent for the publication of all clinical details, tracking data, and case timelines.

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