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Longitudinal Clinical Follow-up of a Patient with Stage IV Non-Small Cell Lung Cancer Receiving Structured Fasting-Based Metabolic Therapy during Standard Oncologic Treatment: A CARE Case Report

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Abstract

Background: Cancer cachexia is a multifactorial syndrome characterized by progressive loss of skeletal muscle mass, involuntary weight loss, anorexia, metabolic dysfunction, and systemic inflammation. It affects approximately 50–80% of patients with advanced malignancies and is particularly common in advanced non-small cell lung cancer (NSCLC), where it negatively influences treatment tolerance, quality of life, functional performance, and overall survival. Supportive metabolic interventions capable of improving nutritional status and physiological resilience remain inadequately studied.

Structured fasting has recently emerged as a potential adjunctive metabolic strategy aimed at improving metabolic flexibility, reducing treatment-associated metabolic stress, enhancing insulin sensitivity, and preserving physiological function during cancer therapy. Long-term clinical observations documenting structured fasting introduced proactively, ahead of chemotherapy, and sustained throughout treatment remain scarce. We report the extended longitudinal follow-up of a patient with Stage IV NSCLC who began a structured one-meal-a-day (OMAD) fasting protocol as a deliberate component of his care approximately one month before starting chemotherapy, continued this protocol throughout chemotherapy and beyond, and who nonetheless experienced a period of severe chemotherapy-associated metabolic deterioration and cachexia before achieving sustained recovery.

Case Presentation: A 51-year-old gentleman with Stage IV non-small cell lung adenocarcinoma (EGFR exon 19 deletion, concurrent TP53 mutation, PD-L1 positive) and glucose-6-phosphate dehydrogenase (G6PD) deficiency was started on a structured one-meal-a-day (OMAD) fasting protocol shortly after diagnosis, as a deliberate, proactive component of his care plan — approximately one month before chemotherapy began. Once chemotherapy started, repeated episodes of severe nausea, vomiting, anorexia, and prolonged involuntary fasting compounded this existing protocol, and his body weight declined from 68.45 kg before treatment to approximately 51 kg, representing severe treatment-associated nutritional deterioration.

Rather than being introduced in response to chemotherapy toxicity, this Structured Fasting-Based Metabolic Therapy (SFMT) protocol was already established before chemotherapy began, and was maintained — with reinforced monitoring and refeeding support — throughout chemotherapy and beyond,

under close clinical supervision. The protocol was centred around the individualized OMAD regimen, progressive nutritional rehabilitation, trigonelline supplementation, and close clinical monitoring, while standard oncologic treatment continued unchanged. The patient remains on this OMAD-based protocol to the present day.

Over subsequent months the patient demonstrated progressive recovery in nutritional status, body weight, physical strength, appetite, and functional capacity. Body weight increased from a nadir of approximately 51 kg to 61.4 kg by November 2025, and to 67 kg at most recent follow-up — approaching his pre-illness baseline, with markedly improved body composition. He has since resumed regular strength training approximately 3–4 times per week, a level of physical function well beyond what would be expected during active treatment for Stage IV disease.

Serial computed tomography throughout follow-up showed a persistently stable primary left upper lobe lesion under EGFR-targeted therapy (osimertinib) — with no new metastatic disease. This sustained disease control, together with the patient's functional and nutritional recovery, represents a major favourable outcome in Stage IV disease. A separate right upper lobe density, first noted shortly after chemotherapy and suspected as infective/inflammatory from an early stage, fluctuated in size and eventually enlarged into a cavitating lesion. Bronchoscopic biopsy of this lesion demonstrated extensive caseating granulomatous inflammation with abundant acid-fast bacilli and no evidence of malignancy, confirming a mycobacterial infection rather than recurrent carcinoma. The patient commenced antimycobacterial treatment shortly afterward and is now in the final phase of a curative course, with completion expected shortly — meaning that, at the time of writing, his lung cancer is under sustained disease control and the separate infection identified during surveillance is on track for full resolution.

Conclusion: This report documents the long-term clinical evolution of a patient with Stage IV NSCLC who began Structured Fasting-Based Metabolic Therapy proactively, about a month before chemotherapy, and has remained on this OMAD-based protocol continuously through chemotherapy and beyond, as an adjunct to standard oncologic management. The patient experienced marked recovery from a period of severe chemotherapy-associated weight loss, with progressive restoration of nutritional status and functional capacity, now approaching his pre-illness weight. The case also illustrates the importance of accurately describing disease control on imaging, and of histopathological confirmation of evolving pulmonary lesions during cancer surveillance.

Keywords: Stage IV NSCLC, Cancer Cachexia, Structured Fasting, OMAD, EGFR-Mutant Lung Cancer, Trigonelline, Metabolic Therapy, Case Report.

Introduction

Non-small cell lung cancer accounts for nearly 85% of all primary lung malignancies and remains one of the leading causes of cancer-related mortality worldwide. Although targeted therapies have significantly improved survival in molecularly selected patients, Stage IV disease continues to present considerable therapeutic challenges owing to tumour burden, treatment-related toxicity, metabolic deterioration, and progressive decline in nutritional status.

Cancer cachexia represents one of the most devastating systemic manifestations of advanced malignancy. Unlike simple starvation, cachexia is characterized by a complex interaction between tumour biology, inflammatory cytokines, endocrine dysfunction, altered substrate metabolism, mitochondrial impairment, and accelerated skeletal muscle proteolysis. These metabolic disturbances frequently persist despite

conventional nutritional supplementation, explaining why increasing caloric intake alone often fails to reverse the syndrome. Among patients receiving chemotherapy, treatment-induced anorexia, nausea, vomiting, mucositis, dysgeusia, and gastrointestinal toxicity further aggravate nutritional decline, contributing directly to reduced performance status, diminished treatment tolerance, increased treatment interruptions, impaired immune function, and poorer survival.

In recent years, fasting and time-restricted feeding have attracted attention because of their potential influence on cellular metabolism, insulin signalling, mitochondrial efficiency, oxidative stress, autophagy, circadian biology, and metabolic flexibility. Experimental studies suggest that fasting-induced metabolic adaptation may modify host responses to physiological stress and improve tolerance to systemic therapies, although clinical evidence remains limited, particularly with respect to long-term observational data in patients with advanced malignancy. Trigonelline, a naturally occurring alkaloid present in fenugreek and coffee, has similarly attracted interest because of reported metabolic, antioxidant, and mitochondrial effects in preclinical studies, though human clinical evidence remains limited.

The case explored in this report differs from the more familiar picture of fasting adopted only after chemotherapy toxicity has already forced a patient into involuntary starvation. Here, structured fasting was the starting point: the patient began an individualized OMAD protocol proactively, shortly after diagnosis and about a month before chemotherapy commenced, and has remained on it continuously ever since. We present the extended follow-up of a patient in whom this pre-established, structured fasting protocol was carried through a period of severe chemotherapy-associated cachexia and a subsequent sustained recovery, alongside an important diagnostic lesson from a separate pulmonary lesion that mimicked, but was not, disease progression.

Diagnostic Background

The patient, with a background of G6PD deficiency, presented with a two-week history of chest pain and exertional breathlessness. Whole-body FDG-PET showed a hypermetabolic left upper lobe mass (approximately 27 × 17 mm, SUV max 9.3) with scattered left lung nodules and a moderate left pleural effusion with pleural activity, without definite distant hypermetabolic disease. Pleural core biopsy confirmed a poorly differentiated lung adenocarcinoma involving the parietal pleura, negative for ALK and ROS1 by immunohistochemistry and positive for PD-L1 (50% of tumour cells). Targeted next-generation sequencing identified an EGFR exon 19 deletion (Tier I, predicting sensitivity to EGFR tyrosine kinase inhibitors) with a concurrent TP53 mutation, a combination associated in the literature with somewhat reduced efficacy of EGFR-TKI monotherapy alone.

Shortly after this diagnostic workup, and approximately one month before starting chemotherapy, the patient began a structured one-meal-a-day (OMAD) fasting protocol as a deliberate, proactive component of his care plan. He commenced chemotherapy afterward while targeted therapy was arranged, then transitioned to an EGFR-directed tyrosine kinase inhibitor (osimertinib) as his definitive systemic treatment, which continued throughout the period of nutritional recovery described below. The OMAD-based protocol continued, essentially without interruption, through chemotherapy, through the switch to osimertinib, and up to the present day.

Clinical Evolution During Long-Term Follow-up

Initial Presentation: Fasting Established Before Chemotherapy

At diagnosis, the patient maintained an active lifestyle with a baseline body weight of 68.45 kg and had

no significant functional limitation. Following diagnosis, he began a structured OMAD fasting protocol proactively, as a deliberate part of his care plan, approximately one month before starting chemotherapy on 20 February 2025. He then underwent chemotherapy on 20 February, 13 March, and 3 April 2025, ahead of starting osimertinib, continuing his OMAD protocol throughout. During the chemotherapy cycles he developed progressively severe nausea, repeated vomiting, anorexia, dysgeusia, and marked food aversion — symptoms that compounded his existing fasting protocol and, at their worst, extended his time without food well beyond the intended structure, with some episodes lasting nearly six consecutive days. The inability to maintain adequate oral intake during the feeding window produced rapid depletion of both adipose tissue and skeletal muscle, resulting in clinically significant cancer cachexia.

This is an important distinction: the periods of prolonged, uncontrolled fasting during chemotherapy were not the origin of the fasting protocol, but a complication superimposed on a fasting protocol that was already in place and had already been running for roughly a month before chemotherapy began. Progressive weight loss was accompanied by generalized weakness, reduced physical endurance, loss of muscle bulk, and increasing fatigue, ultimately affecting the patient's functional independence and quality of life. Serial weight measurements documented a decline from 68.45 kg before treatment to approximately 52.45 kg by the end of May 2025, with the lowest recorded weight approaching 51 kg during the most severe phase — a loss of more than 25% of baseline body weight, fulfilling the clinical features of severe treatment-associated cachexia.

Structured Fasting-Based Metabolic Therapy (SFMT)

Because the OMAD-based Structured Fasting-Based Metabolic Therapy (SFMT) protocol was already established — begun roughly a month before chemotherapy and not introduced in reaction to it — the treating clinicians' task during the cachexia phase was to reinforce and protect this existing structure against the disruption caused by chemotherapy toxicity, rather than to invent a new fasting approach out of chaos. Where chemotherapy-related nausea and vomiting pushed the patient beyond the intended fasting window into longer, more erratic stretches without food, the emphasis was on restoring the predictability and refeeding quality of the original protocol as tolerance allowed. SFMT therefore continued, essentially without interruption, from before chemotherapy through subsequent cycles, the transition to osimertinib, and beyond — the intervention was designed from the outset around the principle that fasting should be structured, individualized, and metabolically supportive, rather than prolonged or uncontrolled, and it was sustained through active treatment rather than paused for a treatment-free recovery window.

The protocol consisted of an individualized One Meal A Day (OMAD) regimen. During the feeding window, emphasis was placed on consumption of nutrient-dense whole foods with sufficient protein, healthy fats, micronutrients, and hydration to support tissue repair and recovery, with meal composition individualized according to treatment tolerance, gastrointestinal symptoms, and the patient's changing nutritional requirements. Trigonelline supplementation was incorporated as an adjunctive component, selected because of emerging experimental data on glucose metabolism, oxidative stress, mitochondrial function, and cellular metabolic homeostasis, and was regarded throughout as supportive rather than disease-modifying. SFMT was never intended to replace conventional oncologic therapy; it was implemented concurrently with ongoing medical management throughout.

Clinical monitoring included serial assessment of body weight, appetite, gastrointestinal tolerance, functional performance, laboratory parameters, radiological response, and overall treatment tolerance.

Progressive improvement became apparent within weeks of initiating the structured protocol: appetite gradually improved, episodes of nausea became less frequent, oral intake increased, and physical activity slowly resumed. Unlike the rapid weight loss observed during chemotherapy, subsequent weight gain occurred gradually and was accompanied by visible improvement in muscle mass, strength, and daily functional capacity. The patient reported improved stamina, greater independence in activities of daily living, and enhanced tolerance to ongoing cancer treatment.

The key clinical observation is that a fasting protocol established before chemotherapy — and deliberately maintained, with reinforced structure and refeeding quality, through the added burden of chemotherapy toxicity — was compatible with full recovery from a severe episode of cachexia, rather than needing to be abandoned when treatment-related illness intensified. At most recent follow-up, more than a year after starting OMAD, the patient's body composition was markedly improved, and he had resumed regular strength training approximately 3–4 times per week — a level of physical function and muscular recovery well beyond what would typically be expected during ongoing treatment for Stage IV disease, and he remains on the same OMAD-based protocol today.

Weight Progression

Time point	Weight (kg)	Context
Baseline	68.45	Active, no functional limitation
End of May 2025	52.45	Nadir ~51 kg; severe chemotherapy-associated cachexia
June 2025	56.25	Early recovery under structured fasting
November 2025	61.40	Sustained recovery
Most recent follow-up	67.00	Approaching pre-illness baseline; improved body composition; strength training 3–4x/week

Radiological Assessment: Disease Control, Not Remission

Serial radiological assessment guided clinical decision-making throughout treatment, interpreted alongside clinical status, nutritional recovery, and laboratory investigations. The primary left upper lobe mass has been serially measured on CT: 32 × 15 mm (23 April 2025, post-chemotherapy baseline) → 32 × 15 mm (26 June 2025) → 31 × 15 mm (16 October 2025) → 31 × 13 mm (16 January 2026), with no new pulmonary nodules, no distant metastatic disease, and only a small, stable pleural effusion. This represents sustained disease control under EGFR-targeted therapy — persistent, non-progressive disease over an extended follow-up period, itself a favourable outcome in Stage IV NSCLC.

A separate, non-primary finding requires its own timeline. A new nonspecific right upper lobe focal density was first seen on the 23 April 2025 CT (13 × 9 mm), with no equivalent lesion on the pre-chemotherapy PET. By 26 June 2025 this had increased to 26 × 14 mm with new small nodular infiltrates extending inferiorly; the reporting radiologist's impression at that time already favoured an infective or inflammatory process over malignancy. On 16 October 2025 it was slightly decreased. By 16 January 2026 it had enlarged into a cavitating, spiculated lesion measuring 23 × 20 mm, with its superior extension increasing to 22 × 13 mm and adjacent nodules increasing in number, plus a new left perianal collection (39 × 15 mm) containing a gas locule not present on prior imaging. Taken together, this lesion's course —

new, then increasing, then slightly decreasing, then increasing again, with an infective impression from the outset — is more consistent with an evolving infective process than with tumour progression, a pattern subsequently confirmed on biopsy.

Because the primary lesion remained stable throughout and the right-sided change was localized and radiologically atypical for widespread progression, multidisciplinary review recommended tissue diagnosis before altering cancer therapy, rather than assuming progression on imaging alone. This proved to be a pivotal point in the patient's clinical course: rather than assuming radiological change reflected tumour progression and modifying anticancer treatment empirically, histopathological confirmation was pursued, which fundamentally altered the diagnostic interpretation of the lesion and prevented its misclassification as recurrent malignancy.

Lesion Timeline Summary

Date	Left upper lobe (primary)	Right upper lobe density
6 Jan 2025 (PET)	~27 × 17 mm, FDG-avid	Not seen
23 Apr 2025 (CT)	32 × 15 mm	New, 13 × 9 mm
26 Jun 2025 (CT)	32 × 15 mm, stable	26 × 14 mm; favoured infective/inflammatory
16 Oct 2025 (CT)	31 × 15 mm, stable	Slightly decreased
16 Jan 2026 (CT)	31 × 13 mm, stable	23 × 20 mm, cavitating; new perianal collection
23 Feb 2026 (biopsy)	—	Confirmed mycobacterial infection; antimycobacterial treatment commenced, now in final phase

Histopathological Evaluation

Bronchoscopic forceps and cryobiopsy specimens of the right-upper-lobe lesion were collected on 23 February 2026 and reported on 25 February 2026. Microscopic evaluation demonstrated extensive areas of necrotic caseous debris with well-formed granulomatous inflammation. Ziehl–Neelsen and Wade-Fite staining demonstrated numerous acid-fast bacilli throughout the specimen, and no viable malignant cells were identified in any sample.

The final pathological diagnosis was: caseating granulomatous inflammation with abundant acid-fast bacilli, consistent with a mycobacterial infection; no evidence of malignancy was identified in the sampled lesion. Microbiological correlation was recommended to distinguish Mycobacterium tuberculosis from non-tuberculous mycobacterial infection, as histopathology alone cannot reliably differentiate between these organisms.

These findings substantially altered the clinical interpretation of the enlarging pulmonary lesion. Prior to biopsy, the increase in lesion size had raised legitimate concern for progression. Histopathology instead demonstrated an infectious granulomatous process, and the oncology and infectious disease teams thereafter managed the two processes separately, with cancer therapy continuing unchanged on the basis of the still-stable primary lesion. This sequence reinforces an important principle in thoracic oncology: new or enlarging pulmonary lesions in patients with advanced lung cancer should not automatically be interpreted as tumour progression, since opportunistic infections may produce radiological appearances that closely resemble recurrent malignancy.

Following confirmation, the patient commenced a standard multi-drug antimycobacterial regimen, with one agent specifically selected in place of the more commonly used alternative to avoid reducing plasma levels of osimertinib, preserving control of his lung cancer while the infection was treated. He is currently in the final phase of this regimen, with completion expected shortly. Unlike his lung cancer, which remains a chronic condition under ongoing disease control, this mycobacterial infection is expected to be fully cured once treatment concludes — a second, distinct piece of good news alongside the continued stability of his primary disease.

Laboratory and Metabolic Follow-up

Serial laboratory investigations assessed treatment tolerance, nutritional status, hepatic and renal function, electrolyte balance, and hematological recovery throughout SFMT. Renal function was preserved (eGFR consistently >90 mL/min/1.73 m²), liver function tests (ALT, AST, GGT, alkaline phosphatase, bilirubin) remained within acceptable limits, and serum albumin was maintained around 40–45 g/L, suggesting preserved hepatic synthetic function and progressive nutritional improvement. Electrolytes remained largely stable throughout follow-up.

Hematological evaluation demonstrated persistent mild anemia during parts of the recovery period (hemoglobin approximately 100–102 g/L), compatible with anemia of chronic disease and systemic cancer therapy; platelet and leukocyte counts remained adequate without clinically significant cytopenias. Baseline inflammatory markers prior to treatment (CRP 6.5 mg/L) were only mildly elevated, and homocysteine was normal (10.9 µmol/L). Overall, there was no biochemical evidence of dehydration, electrolyte disturbance, or renal or hepatic dysfunction attributable to the fasting intervention — the laboratory profile is objective support that the structured fasting protocol was well tolerated throughout.

Discussion

Cancer cachexia remains one of the most challenging complications of advanced malignancy. Unlike uncomplicated starvation, it is a complex metabolic syndrome combining persistent skeletal muscle loss, systemic inflammation, endocrine alterations, insulin resistance, increased resting energy expenditure, and impaired protein synthesis, arising through a multifactorial interaction between tumour-derived mediators, host inflammatory responses, reduced nutritional intake, and treatment-related toxicity. Reversal of cachexia therefore requires considerably more than simple caloric replacement — a complexity this case illustrates clearly. Notably, the patient had already been established on a structured OMAD fasting protocol for approximately a month before chemotherapy began, yet still went on to develop severe cachexia once chemotherapy-induced nausea, vomiting, anorexia, and prolonged involuntary fasting were superimposed on this existing protocol, producing weight loss exceeding 25% of baseline. This indicates that a pre-existing structured fasting protocol did not, by itself, prevent cachexia once severe treatment toxicity intervened — it was the reinforcement of that protocol's structure and refeeding quality during recovery, not its initial adoption, that appears to track most closely with the reversal of cachexia.

The central observation in this case is not simply that the patient regained weight, but how: an already-established, structured fasting protocol was temporarily overwhelmed by chemotherapy-induced gastrointestinal toxicity, and recovery followed once that structure and its refeeding quality were reinforced, rather than either abandoning fasting altogether or leaving it uncontrolled. The same absence of food can therefore carry very different physiological meaning depending on whether it is predictable and paired with adequate refeeding, or erratic and driven by illness — even when, as in this case, the

fasting protocol long predates the chemotherapy that later disrupted it. That the protocol was already in place before chemotherapy, and was sustained through it rather than introduced because of it, is clinically important: it suggests that a pre-existing structured fasting regimen can be protected and maintained through active treatment, with active reinforcement during periods of toxicity, rather than requiring a treatment-free window to either begin or recover within.

Several biological mechanisms have been proposed through which structured fasting may influence host metabolism, including improved insulin sensitivity, enhanced metabolic flexibility, activation of cellular stress-response pathways, improved mitochondrial efficiency, modulation of inflammatory signalling, and increased autophagic activity. These mechanisms remain unconfirmed in patients with advanced cancer, but the stable laboratory parameters and steady, sustained weight gain observed here — now reaching close to the patient's pre-illness baseline — are consistent with a well-tolerated metabolic intervention rather than added physiological stress.

Imaging stability of the primary tumour during this period reflects disease control on EGFR-targeted therapy rather than a fasting-driven anticancer effect. Sustained stable disease over an extended period in Stage IV NSCLC is itself a clinically meaningful and favourable outcome, and in this patient it has coincided with a functional recovery that goes well beyond simple weight restoration — his body composition is now markedly improved, and he has resumed strength training 3–4 times weekly, a level of physical resilience uncommon in patients still undergoing treatment for advanced disease. The separate right-upper-lobe density illustrates a distinct and equally important point — its fluctuating course and early infective radiological impression, later confirmed on biopsy, show that a new or changing pulmonary finding in a heavily-treated patient is not automatically tumour progression, even when it enlarges over time. Opportunistic infections, particularly mycobacterial disease, may closely mimic recurrent lung cancer both clinically and radiologically, and tissue diagnosis remains the gold standard whenever imaging alone is equivocal or would otherwise drive a change in treatment. Taken together, the two findings from this period of surveillance are both favourable: the primary lung cancer remains under sustained disease control, and the separate finding that had raised concern was not a second cancer problem at all but a treatable infection, now approaching cure on a standard antimycobacterial regimen chosen to avoid interfering with his EGFR-targeted therapy.

This report's principal strength is its longitudinal scope: it documents the patient's course from a pre-chemotherapy start on structured fasting, through a period of severe treatment-associated cachexia despite that existing protocol, sustained metabolic recovery while the same protocol continued, serial radiological evaluation, and histopathological confirmation of a separate pulmonary lesion. Limitations are those inherent to a single-patient report: no isolation of SFMT's effect from concurrent oncologic therapy or trigonelline supplementation, and no control for spontaneous improvement, so conclusions regarding efficacy cannot be drawn. Still, the observation that recovery coincided with reinforcement of a fasting protocol that had already been running for over a year by most recent follow-up is a clear, testable hypothesis rather than a settled finding, and it justifies further investigation through prospective observational cohorts and randomized trials incorporating objective body composition analysis, inflammatory biomarkers, treatment tolerance, and quality of life.

Conclusion

This longitudinal CARE case report describes the extended clinical course of a patient with Stage IV, EGFR-mutant NSCLC who began a Structured Fasting-Based Metabolic Therapy protocol — an

individualized OMAD regimen with nutrient-dense refeeding and trigonelline supplementation — proactively, about one month before starting chemotherapy. He subsequently developed profound chemotherapy-associated metabolic deterioration and cancer cachexia, losing over 25% of his baseline body weight, as chemotherapy toxicity was superimposed on this existing fasting protocol. Rather than abandoning the protocol at this point, it was reinforced and sustained through his chemotherapy cycles, the transition to osimertinib, and beyond, and the patient remains on it to the present day.

This coincided with steady, sustained recovery of body weight, nutritional status, functional capacity, and physiological resilience, without biochemical compromise, while standard oncologic treatment continued unchanged throughout. At most recent follow-up his weight had reached 67 kg, approaching his pre-illness baseline of 68.45 kg, with markedly improved body composition; he has since resumed strength training 3–4 times per week. Throughout this period his primary lung lesion remained under sustained disease control on EGFR-targeted therapy — itself a major favourable outcome in Stage IV NSCLC — and, combined with this degree of nutritional and functional recovery, represents a strongly positive overall trajectory for this patient. A separate, initially indeterminate right upper lobe lesion, suspected as infective from an early stage, proved on biopsy to be a mycobacterial infection rather than recurrent malignancy. He commenced a standard antimycobacterial regimen shortly afterward and is now in the final phase of treatment, with cure expected on completion. Taken together, the overall picture at most recent follow-up is a strongly favourable one: his lung cancer remains under sustained disease control, and the separate infection that briefly raised concern for progression is on track to be fully resolved.

The favorable metabolic recovery observed in this patient occurred while standard oncologic management continued uninterrupted; this report does not conclude that SFMT replaced or superseded established cancer therapies. Rather, it suggests that a structured fasting protocol, established proactively ahead of chemotherapy and reinforced rather than abandoned during a subsequent episode of severe treatment-related toxicity, may be compatible with recovery from cachexia in carefully selected patients with advanced malignancy — and that stable disease on serial imaging should not be conflated with a complete radiological response. Prospective clinical studies incorporating standardized fasting protocols, objective body composition analysis, metabolic biomarkers, and long-term follow-up are now needed to determine whether these encouraging findings can be reproduced in larger populations.

Clinical Learning Points

- Cancer cachexia is a multifactorial metabolic syndrome that frequently complicates advanced NSCLC and contributes significantly to treatment-related morbidity.
- A structured fasting protocol can be established proactively, ahead of chemotherapy, and maintained through subsequent treatment toxicity rather than being introduced reactively once involuntary fasting has already begun.
- A supervised OMAD-based fasting protocol with nutrient-dense refeeding and trigonelline supplementation may support recovery from severe treatment-associated cachexia without biochemical harm, when implemented alongside standard oncologic care.
- Recovery of body weight should be interpreted alongside improvements in functional capacity, nutritional status, and overall physiological resilience, rather than weight alone.
- Sustained disease control on serial imaging under targeted therapy, paired with strong functional recovery, is itself a major favourable outcome in Stage IV disease and should be described accurately in clinical documentation and reporting.

- New or evolving pulmonary lesions during cancer surveillance should not automatically be interpreted as tumour progression; histopathological confirmation remains essential whenever imaging findings are equivocal, as infectious granulomatous disease may closely mimic recurrent malignancy.
- Longitudinal follow-up provides important insight into the dynamic interaction between oncologic treatment, metabolic recovery, nutritional interventions, and supportive care.
- At most recent follow-up, the overall clinical picture is favourable on both fronts: the primary lung cancer remains under sustained disease control, and the separate mycobacterial infection identified during surveillance is in the final phase of a curative antimycobacterial regimen.

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