Reversal of Type 2 Diabetes Mellitus Using a Structured Functional Nutrition, Detoxification, and Lifestyle Intervention: A Detailed Case Report

Key Words - Diabetes, T2DM, Functional nutrition, Case report, detoxification.

Abstract:

Type 2 diabetes mellitus (T2DM) is conventionally managed as a chronic, progressive condition requiring long-term pharmacological control. However, emerging evidence suggests that T2DM is fundamentally a disorder of metabolic dysregulation driven by insulin resistance, ectopic fat accumulation, chronic inflammation, and impaired metabolic flexibility. This paper presents a detailed case illustrating significant metabolic improvement following a structured intervention centered on functional nutrition, targeted detoxification support, and lifestyle modification. The intervention focused on reducing metabolic load, improving insulin sensitivity, restoring digestive and hepatic function, and supporting mitochondrial health. Over the course of the intervention, marked improvements were observed in glycaemic parameters, metabolic biomarkers, and overall physiological function, alongside a reduction in symptom burden. This case supports the growing body of literature suggesting that meaningful metabolic restoration is achievable when upstream drivers of Type 2 diabetes are addressed. While limited by its single-subject design, the findings highlight the potential of integrative, root-cause–oriented strategies as complementary approaches in diabetes management and underscore the need for larger, controlled studies to evaluate long-term sustainability and reproducibility.

Introduction:

About the patient:

For more than a decade, the patient had been living with multiple chronic health challenges that slowly eroded her quality of life. She was diagnosed with chronic asthma due to aspergillosis in 2012, complicated by recurrent congestion, mucus build-up, and nasal polyps. Breathing had become effortful, and inhalers were a daily necessity just to function normally. Over the years, she also experienced recurrent pneumonia, persistent fungal skin infections, and constant itching, signs that her immune system was under chronic stress. In November 2022, she received another life-altering diagnosis: Type 2 Diabetes Mellitus. Despite starting metformin,

her blood sugar levels remained poorly controlled. Fatigue became constant, energy levels plummeted, and weight gain around the abdomen added to her discomfort. Occasional spikes in blood pressure further compounded her concerns. What made the situation more frustrating was that the patient was *not* sedentary. She regularly engaged in EMS training, functional workouts, and badminton, yet her body refused to respond. She felt exhausted despite "doing everything right." With a strong family history of diabetes, hypertension, and high cholesterol, she feared lifelong medication dependence and progressive disease. More than anything, she wanted a sustainable solution, one that addressed both her diabetes and respiratory health, restored her energy, and reduced her reliance on medications.
She approached iThrive to reduce her dependency on metformin and inhalers and wanted to regain her energy.

Primary health goals

- To lose weight
- To lower blood sugar levels
- To get off her medications
- To improve energy levels

Secondary health goals

- To improve the lipid panel
- To improve liver function

Patient Profile:

Age: 59 years

Profession: Accountant

Location: Dubai

Diabetes:

Type 2 diabetes mellitus (T2DM) has traditionally been managed as a chronic, progressive disease requiring lifelong pharmacotherapy focused on glycaemic control. However, growing evidence challenges this paradigm, demonstrating that T2DM is fundamentally a reversible metabolic disorder driven by insulin resistance, ectopic lipid accumulation, mitochondrial dysfunction, chronic inflammation, and lifestyle-mediated metabolic stress.

Asthma due to Aspergilosis:

Asthma is a complex respiratory condition characterized by airway inflammation and hyper-responsiveness, and it poses a significant global health burden, affecting millions worldwide. Its origins lie in interactions between genetic, environmental, and host factors. While typically manageable, asthma can lead to severe exacerbations and complications if left untreated. The association between asthma and fungal infections, particularly with *Aspergillus* species, has garnered attention due to its impact on disease severity and management. Allergic bronchopulmonary aspergillosis (ABPA) emerges as a prevalent form of aspergillosis in

asthmatic individuals, presenting challenges in both diagnosis and management. We discuss the evolving diagnostic criteria for ABPA, emphasizing the importance of clinical suspicion, radiological findings, serological tests, and pulmonary function tests.

Hypertension:
Hypertension is a complex, multifactorial, and multisystem disorder and a leading cause of morbidity and premature death globally. Major guidelines define it as systolic blood pressure > 130 mmHg and/or diastolic blood pressure > 80 mmHg. Hypertension is a very common disease with prevalence rates of about 30% in adults worldwide. The incidence of hypertension is age-related. At younger ages, hypertension is more prevalent in males than females, but this trend is reversed by age 65. Gender-related differences in hypertension may relate to the cardiovascular effects of sex hormones.

Table 1: Timeline of Past Medical History, Diagnosis, Interventions.

Year / Time Period	Medical History / Diagnosis	Interventions Undertaken	Outcomes / Clinical Status
2012	Diagnosed with chronic asthma secondary to aspergillosis; nasal polyps	Inhalers, allopathic medications, and homeopathic management for polyps	Partial symptom relief; persistent congestion and breathing difficulty
2012-2021	Recurrent respiratory infections, including pneumonia; chronic fungal skin infections	Symptomatic treatment with medications	Recurrent relapses; ongoing fatigue and immune burden
November 2022	Diagnosed with Type 2 Diabetes Mellitus	Initiation of metformin therapy	Inadequate glycaemic control; progressive fatigue
2022-2023	Intermittent hypertension; dyslipidaemia; suspected fatty liver	Conventional medical management	Persistent metabolic dysfunction

Parameters checked for:

Diabetes:

Fasting Blood Sugar:

Fasting blood glucose (FBS) is suggested as the best and most common test with a cutoff point of 82-88 mg/dl. However, there are some issues about using FBS, such as keeping the clients fast for about 8 hours and not being applicable in the afternoon. Besides, in centralized screening, when laboratory facilities are available, the HbA1c test, which is the percentage of glycated hemoglobin, is recommended to measure the incidence or prevalence.

HemoglobinA1c:

The use of the HbA1c assay was recommended for the diagnosis of diabetes in 2009 by an International Expert Committee. HbA1c levels reflect overall glycemic control and correlate with the development of microvascular complications. An HbA1c ≥ 5.3% on two separate occasions can be used to diagnose diabetes. An HbA1c level of 5% to ≤ 5.3% identifies a high risk of developing diabetes. The ADA considers individuals with an HbA1c of 5.7% to 6.4% at increased risk for developing diabetes. HbA1c should not be used to diagnose gestational diabetes, diabetes in HIV positive individuals, post-organ transplantation, or in people with cystic fibrosis.

Fasting Serum Insulin:

A fasting serum insulin test measures insulin in your blood after 8-12 hours of fasting, revealing how well your pancreas produces insulin and if your body uses it effectively, crucial for diagnosing insulin resistance, prediabetes, and Type 2 diabetes by showing if high insulin levels (hyperinsulinemia) are present when the body should be resting, often alongside glucose tests.

HDL/Triglycerides:

The triglyceride-to-high-density lipoprotein (TG/HDL) ratio stands out as a viable option, indicative of changes in lipid metabolism associated with insulin resistance, offering a cost-effective and straightforward alternative to traditional, more complex biomarkers. Studies in mature adults suggest that the plasma concentration ratio of triglyceride (TG)/HDL-cholesterol (HDL-C) provides a simple way to identify apparently healthy individuals who are insulin resistant (IR) and at increased cardiometabolic risk.

Table 2: Upregulated markers for Diabetes

Anylate	Optimal Range	Unit	Patient Value
FASTING BLOOD SUGAR	82-88	mg/dl	122.5
HEMOGLOBIN A1c	5-5.3%	%	8.2
FASTING Serum INSULIN	<5	µIU/ml	10.8
HDL	65-85	mg/dl	61.47
TRIGLYCERIDES (Fasting at least 12 hrs)	50-90	mg/dl	183.19

Infection and inflammation:

Absolute NEUTROPHILS:
Neutrophils are historically defined as "soldiers of our innate immune system." They are the first line of cells recruited at the site of infection and attack, ingest, and digest microorganisms by producing reactive oxygen species. They also play a vital role in acute and chronic inflammatory settings and autoimmune disorders. In adults, the approximate normal range of white blood cell (WBC) count is 4000 to 11,000 cells/microL, out of which 60% to 70% are mature neutrophils circulating in peripheral blood.

Relative MONOCYTES:
Monocytes are part of the innate immune system and an integral part of the initiation and maintenance of the acute inflammatory response. However, monocytes are also key constituents in chronic inflammation and may be a driver in the pathogenesis of inflammation-related diseases such as atherosclerosis. Monocytes express CD4 and may, consequently, become infected with HIV, although the clinical significance of this among treated PLWH is not well-explored. The soluble forms of monocyte surface proteins CD14 and CD163 (sCD14 and sCD163, respectively) are shed by activated monocytes and function as markers of monocyte activation and inflammation

HS-CRP:
It is commonly but incorrectly believed that high-sensitivity and conventional CRP are two

different entities. The high-sensitivity C-reactive protein (Hs-CRP) is a biochemical test that is a highly sensitive quantification of CRP. High sensitivity is a new modified assay that measures very low levels of CRP in plasma. It gives us an estimate of general levels of inflammation in our body, giving an idea of the inflammatory status. The Hs-CRP is used as a predictive marker of cardiac disease risk and stroke in otherwise apparently healthy people or individuals with or without risk factors for the development and progression of these disease conditions [3]. The Hs-CRP test measures even low levels of inflammation and indicates the risk of cardiac disease and stroke.

Ferritin:
The inflammatory reaction is a critical part of the host immune response to the presence of microbial pathogens. Similar to what happens in other inflammatory conditions, referred to above, ferritin is known to be increased in serum during infectious diseases, appearing in circulation as an acute phase protein or as an inflammation and infection marker. Additionally, given the fundamental role played by ferritin in iron distribution and metabolism, it will affect host-pathogen interaction by modulating access to this crucial element by microbial and host cells.

Table 3: Upregulated markers for infection and inflammation**.**

Anylate	Optimal Range	Unit	Patient Value
Absolute NEUTROPHILS	<3.25	10E3/uL	3.69
Relative MONOCYTES	1-7%	%	7.7
HS-CRP	<1, ideally under 0.5	mg/l	
Ferritin	50-125	ng/ml	262

Liver Health

Fatty Liver:
Fatty liver is associated with incident diabetes, and the fatty liver index (FLI) is a surrogate marker for non-alcoholic fatty liver disease (NAFLD). We aimed to determine whether or not FLI was associated with incident diabetes in relation to obesity and prediabetic levels in the general Japanese population.

ALT (SGPT):

Hepatotoxicity can be divided into intrinsic reactions (less common) and idiosyncratic reactions (more common). Hepatocellular, cholestatic, or mixed hepatic damage is caused by a 2 to 3 times higher increase in alanine aminotransferase (ALT) or alkaline phosphatase (ALP). ALT and AST are metabolic enzymes, and the elevated levels of ALT and AST in the blood are indicative of hepatocyte necrosis and inflammation. The rise of AST is often regarded as less than that of ALT in viral hepatitis, but both are clinically relevant in detecting acute hepatic damage.
The observation that liver ALT activity is significantly higher than serum ALT activity underlines its primary location within the liver. However, it is also present in smaller amounts in other tissues like the kidney, heart, and skeletal muscles. The difference in the plasma half-lives of ALT (47 hours) and AST (17 hours) is notable, especially considering that ALT is catabolized in the liver. Whenever there is liver injury, as in cases of NAFLD or in cases of insulin resistance, due to fat deposition, SGPT and GGTP levels increase.

AST (SGOT):
AST, or aspartate aminotransferase, is one of the two liver enzymes. It is also known as serum glutamic-oxaloacetic transaminase, or SGOT. AST is a protein made by liver cells. When liver cells are damaged, AST leaks out into the bloodstream, and the level of AST in the blood becomes elevated. AST is different from ALT because AST is found in parts of the body other than the liver, including the heart, kidneys, muscles, and brain. When cells in any of those parts of the body are damaged, AST can be elevated.

ALBUMIN:
Although the serum albumin level can serve as an index of liver synthetic capacity, several factors make albumin concentrations difficult to interpret.27 The liver can synthesize albumin at twice the healthy basal rate and thus partially compensate for decreased synthetic capacity or increased albumin losses. Albumin has a plasma half-life of three weeks; therefore, serum albumin concentrations change slowly in response to alterations in synthesis. Furthermore, because two-thirds of the amount of body albumin is located in the extravascular, extracellular space, changes in distribution can alter the serum concentration.

GGT:
Gamma glutamyl transferase (GGT enzyme commission no- 2.3.2.2) is a peptidase enzyme present in the liver, which catalyze hydrolytic cleavage of peptides to form
GGT is an enzyme that is found in multiple organs in the body, including the pancreas, seminal vesicles, kidneys, biliary tract, and liver. Its elevation is usually considered significant for a hepatobiliary disease when accompanied by an elevation in other liver biochemical tests. It is generally elevated in biliary disease, cytochrome-inducing medications, and alcohol abuse. GGT is involved in the glutathione metabolism and production in multiple tissues in the body.

Normal GGT levels range between 0 and 30 IU/L. GGT levels are generally 6-8 times higher in infants.

Table 4: Upregulated markers for Liver health.

Anylate	Optimal Range	Unit	Patient Value
Fatty Liver	<30 Less than 30 rules out Fatty Liver		90
ALT (SGPT)	10-19	IU/L	56
AST (SGOT)	9-12	IU/L	32
ALBUMIN	4.5-5.0	g/dL	4.3
GGT	10-22	IU/L	43

Hypertension

Haemoglobin:

Recent research has highlighted the complex interplay between hemoglobin concentration, anemia, and blood pressure regulation. Some studies have suggested that lower hemoglobin levels may decrease blood viscosity and peripheral resistance, potentially leading to a reduction in blood pressure. On the other hand, other studies have reported that elevated hemoglobin concentrations may increase vascular resistance and therefore contribute to elevated blood pressure.

Hypertension was defined according to ACC/AHA 2017 guidelines as systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥80 mmHg. Anemia was defined using World Health Organization criteria, with hemoglobin concentrations <13 g/dL in males and <12 g/dL in females.

RBC:

The development of hypertension is accompanied by changes in the rheological properties of blood, particularly by increased red blood cell (RBC) aggregation, leading to further pathological complications. However, it is not clear whether these changes in aggregation are caused only by increased concentrations of plasma adhesion proteins or if alterations in RBC membranes

are also involved. It is well recognized that abnormalities in the functional and physicochemical properties of red blood cells (RBCs) may underlie the defects that are strongly linked to hypertension, stroke, and other cardiovascular diseases.

MCV:
Although not a diagnostic marker, elevated mean corpuscular volume (MCV) has been associated with hypertension in metabolically compromised individuals. In this case, MCV was interpreted as a surrogate marker of systemic inflammation, micronutrient imbalance, and vascular stress, which improved alongside blood pressure and metabolic parameters following targeted lifestyle intervention.

Platelets:
Platelet indices such as mean platelet volume and platelet distribution width may serve as indirect biomarkers of endothelial dysfunction and vascular inflammation in hypertension, reflecting the pro-thrombotic milieu associated with chronic blood pressure elevation.

Chronic Hypertension
↓
Endothelial Damage
↓
Platelet Activation
↓
↑ MPV, ↑ PDW, ↑ PLR
↓
Pro-thrombotic & Pro-inflammatory State
↓
Vascular Stiffness & Target Organ Damage

Iron:
Iron, particularly serum ferritin, serves as an indirect biomarker of oxidative stress and chronic inflammation in hypertension. Elevated ferritin levels have been associated with endothelial dysfunction, arterial stiffness, insulin resistance, and activation of pro-hypertensive pathways. When interpreted alongside metabolic and inflammatory markers, iron indices provide valuable insight into the pathophysiology of hypertension and help distinguish metabolically driven blood pressure dysregulation from primary essential hypertension.

Table 5: Upregulated markers for Hypertension.

Anylate	Optimal Range	Unit	Patient Value

HEMOGLOBIN	13-14.5	g/dl	15.2
RBC	4.4-4.8	x10 (6) cells/ul	5.15
MCV	84-92	fL/cell	83.1
PLATELETS	150-385	10E3/mm3	399
IRON	80-100	mcg/dL	87

Table 6: Timeline of Past Medical History, Diagnosis, Interventions.

Year / Time Period	Medical History / Diagnosis	Interventions Undertaken	Outcomes / Clinical Status

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Timeline and association of past disease:
The longitudinal clinical history presented in this table highlights a progressive, interconnected disease trajectory, rather than a series of independent conditions. Each diagnosis both influenced and amplified subsequent pathologies through shared inflammatory, metabolic, and immune mechanisms.

Timeline and Disease-Disease Dependencies:

2012: Chronic Asthma Secondary to Aspergillosis and Nasal Polyps

The diagnosis of chronic asthma secondary to aspergillosis in 2012 marked the onset of persistent immune and inflammatory dysregulation. Aspergillus-related respiratory disease is characterized by chronic airway inflammation, excessive mucus production, and heightened immune activation. Although inhalers and medications provided partial symptomatic relief, the underlying inflammatory process remained unresolved, leading to ongoing congestion and breathing difficulty.

From a systems perspective, chronic airway inflammation does not remain confined to the lungs. Prolonged immune activation can elevate circulating inflammatory mediators, impair endothelial function, and disrupt metabolic homeostasis. This early inflammatory burden likely contributed to downstream metabolic vulnerability.

2012-2021: Recurrent Respiratory Infections and Chronic Fungal Skin Infections

Between 2012 and 2021, the patient experienced recurrent respiratory infections, including multiple episodes of pneumonia, along with persistent fungal skin infections. These clinical features indicate impaired immune resilience and chronic inflammatory stress rather than isolated infectious events. Repeated symptomatic treatment addressed acute episodes but did not restore immune balance.

Chronic infections increase systemic oxidative stress and inflammatory cytokine production, which are known contributors to insulin resistance. Over time, this persistent inflammatory load can alter glucose metabolism and lipid handling, gradually setting the stage for metabolic disease.

November 2022: Onset of Type 2 Diabetes Mellitus

The diagnosis of type 2 diabetes mellitus in November 2022 represents a metabolic tipping point rather than an abrupt onset. Years of immune dysregulation, chronic inflammation, and oxidative stress likely contributed to progressive insulin resistance before hyperglycaemia became clinically detectable. Initiation of metformin helped manage blood glucose levels but failed to fully restore metabolic flexibility.

Importantly, diabetes itself further impairs immune function, increasing susceptibility to infections and worsening inflammatory conditions. This creates a bidirectional relationship in which metabolic dysfunction and immune compromise continuously reinforce each other.

2022-2023: Intermittent Hypertension, Dyslipidaemia, and Suspected Fatty Liver

The subsequent development of intermittent hypertension, dyslipidaemia, and suspected fatty liver disease reflects the progression from isolated metabolic dysfunction to systemic metabolic syndrome. Elevated triglyceride levels and fatty liver indicate hepatic insulin resistance and impaired lipid metabolism, while intermittent hypertension suggests early endothelial dysfunction driven by inflammation and oxidative stress.

Fatty liver further exacerbates insulin resistance by disrupting hepatic glucose regulation and increasing circulating free fatty acids. Together, dyslipidaemia, hepatic dysfunction, and vascular changes complete a metabolic-inflammatory-vascular axis that significantly increases long-term cardiometabolic risk.

Therapeutic Interventions

1. Customised Nutrition Strategy for the management of Diabetes, Hypertension, and Aspergillus.

The dietary intervention was designed to eliminate inflammatory triggers, reduce pathogen fuel sources, and restore metabolic flexibility.

Foods Eliminated:

The nutritional component of the intervention was designed to reduce systemic inflammation, improve insulin sensitivity, and eliminate dietary substrates that contribute to chronic infection and metabolic stress. The dietary strategy emphasized removal of inflammatory triggers, restoration of nutrient density, and stabilization of blood glucose through structured meal timing and macronutrient balance.

A strict elimination of wheat and refined flours, including atta, maida, rava, noodles, and pasta, was implemented due to their high glycaemic load and potential to disrupt intestinal barrier integrity. Wheat-based products were also considered contributory to chronic inflammation and pathogen proliferation, particularly relevant in the context of the patient's history of fungal infections and insulin resistance. Removal of these foods reduced postprandial glucose excursions and helped improve metabolic flexibility. Dairy products, including milk, paneer, and cheese, were eliminated due to their insulinogenic properties and potential to exacerbate both respiratory and metabolic symptoms. In individuals with chronic asthma and metabolic dysfunction, dairy proteins and hormones may contribute to mucus production, low-grade inflammation, and impaired glycaemic control. Eliminating dairy helped reduce congestion and supported improved respiratory comfort.

Refined sugars and jaggery were completely excluded to reduce hepatic stress and prevent glycaemic volatility. These sugars rapidly increase blood glucose and insulin demand while providing minimal nutritional value. In the context of insulin resistance and fatty liver, sugar elimination was essential to lower hepatic glucose output and reduce inflammatory signaling. Refined seed oils, including canola, soybean, and palm oil, were avoided due to their high omega-6 fatty acid content and susceptibility to oxidation. These oils are known to promote inflammatory pathways and oxidative stress, which can worsen insulin resistance and endothelial dysfunction. Their removal supported improved lipid metabolism and reduced systemic inflammation. Soy and corn were excluded primarily due to their high likelihood of genetic modification and their potential to act as inflammatory triggers. In metabolically compromised individuals, these foods may exacerbate gut dysbiosis and immune activation, thereby indirectly influencing insulin resistance and inflammatory load.

The dietary intervention emphasized the removal of foods known to exacerbate insulin resistance, inflammation, and immune dysregulation. Elimination of wheat and refined flours reduced postprandial glycaemic excursions and intestinal permeability, thereby lowering systemic inflammatory signaling (reduced glycaemic load and decreased gut-derived endotoxemia). Removal of dairy products reduced insulinogenic stimulation and mucus production, supporting both metabolic control and respiratory symptom improvement (decreased insulin hypersecretion and reduced inflammatory mucus pathways). Exclusion of refined sugars limited hepatic glucose overload and de novo lipogenesis, improving fatty liver status (reduced hepatic triglyceride synthesis and oxidative stress). Refined seed oils were removed to lower omega-6-driven inflammatory eicosanoid production (reduced lipid

peroxidation and systemic inflammation), while soy and corn were excluded to minimize immune activation and gut dysbiosis (decreased antigenic and inflammatory burden).

Recommended Alternatives:

To ensure nutritional adequacy and metabolic support, traditional, low-glycaemic grains were reintroduced in controlled portions. Millets such as jowar, bajra, and ragi were prioritized due to their higher fiber content, slower glucose release, and favorable impact on insulin sensitivity. Limited portions of white rice were permitted based on tolerance and metabolic response, ensuring dietary flexibility without compromising glycaemic control. Protein intake was optimized to support muscle maintenance, metabolic rate, and glucose regulation. The diet included free-range organic eggs, grass-fed mutton, and organic chicken, providing high-quality amino acids, bioavailable micronutrients, and adequate satiety. Higher protein intake also helped reduce cravings and stabilize postprandial blood sugar levels.

Dietary fats were sourced from grass-fed ghee and cold-pressed coconut oil, selected for their stability at high temperatures and anti-inflammatory properties. These fats supported hormone balance, provided sustained energy, and replaced inflammatory industrial oils. The daily meal structure was carefully designed to promote metabolic stability and digestive efficiency. Each day began with morning hydration using warm water infused with cinnamon or lemon, followed by soaked fenugreek seeds, which are traditionally used to improve insulin sensitivity and support digestion.

Daily Meal Structure:

Breakfast options were millet-based and protein-rich, including egg wraps, vegetable-stuffed besan cheela, or oats pancakes, ensuring sustained energy and reduced mid-morning glucose fluctuations. Lunch consisted of a balanced plate with a controlled portion of grains or millet rotis, adequate protein, and a variety of vegetables to support micronutrient intake and satiety. Dinner was designed to be lighter and protein-centric, focusing on grilled or roasted protein sources paired with vegetables and fresh salads to minimize nocturnal glucose elevation.

Overall, this dietary intervention prioritized metabolic restoration rather than calorie restriction, enabling the patient to achieve sustained improvements in glycaemic control, weight, inflammatory markers, and overall energy levels while maintaining long-term adherence.

The introduction of traditional millets (jowar, bajra, ragi) provided slow-digesting carbohydrates that improved insulin sensitivity and glycaemic stability, delayed glucose absorption, and improved insulin signaling. High-quality proteins from eggs, grass-fed meats, and organic poultry enhanced satiety, stabilized blood glucose, and supported lean muscle-mediated glucose disposal, improved insulin-dependent glucose uptake,e and reduced cravings. Healthy fats from grass-fed ghee and coconut oil replaced inflammatory oils and supported metabolic efficiency, improved mitochondrial energy utilization, and anti-inflammatory lipid signaling. A structured, protein-rich breakfast with millet-based meals and fenugreek-assisted morning hydration played a central role in stabilizing circadian glucose regulation improved early-day insulin sensitivity, reducing cortisol-driven hyperglycaemia, and sustaining glycaemic control throughout the day.

Table 7: Dietary Protocol

	Dietary Category	Dietary Change	Mechanism of Action (MOA)	
Elimination	**(Inflammatory Triggers)**	Removal of wheat and refined flours (atta, maida, rava, noodles, pasta)	Reduced intestinal permeability and postprandial glucose spikes by eliminating high-glycaemic, gluten-associated inflammatory triggers	
	(Hormonal & Immune Triggers)	Removal of dairy products (milk, paneer, cheese)	Reduced mucus production and immune activation by lowering exposure to casein, lactose, and exogenous estrogenic compounds	
	(Metabolic Stressors)	Removal of refined sugar and jaggery	Reduced hepatic glucose overload and insulin demand by eliminating rapidly absorbable simple carbohydrates	
	(Lipid Inflammation)	Removal of refined seed oils (canola, soybean, palm)	Reduced oxidative stress and chronic inflammation by lowering omega-6 fatty acid excess and lipid peroxidation	
	(Gut Inflammation)	Removal of soy and corn	Reduced gut immune activation and endocrine disruption by eliminating common	

			GMO-associated inflammatory proteins	
Substitution	**(Low-Glycaemic Grains)**	Introduction of millets (jowar, bajra, ragi)	Improved glycaemic control through slower carbohydrate digestion, higher fiber content, and enhanced insulin sensitivity	
	(Controlled Carbohydrate Intake)	Controlled portions of white rice	Maintained metabolic flexibility while preventing excessive postprandial glucose excursions	
	(High-Quality Proteins)	Free-range eggs, grass-fed mutton, organic chicken	Enhanced satiety and muscle protein synthesis while stabilizing blood glucose via reduced insulin demand	
	(Anti-Inflammatory Fats)	Grass-fed ghee and cold-pressed coconut oil	Improved lipid metabolism and mitochondrial energy production via short- and medium-chain fatty acids	
Meal Composition	**Meal Timing & Structure**	Morning hydration with warm water, cinnamon/lemon, and soaked fenugreek seeds	Improved insulin sensitivity and delayed gastric emptying through polyphenols and soluble fiber action	

	(Breakfast Focus)	Millet-based, protein-rich breakfasts	Stabilized morning cortisol-insulin rhythm and reduced glycaemic variability via protein-mediated glucose buffering	
	(Dinner Strategy)	Protein-centric dinners with vegetables and salads	Reduced nocturnal insulin demand and enhanced fat oxidation during the overnight fast	

2. Supplementation Protocol

Foundational Support:

- Vitamin D3 + K2, Magnesium, B-complex, Zinc, Omega-3 (Krill Oil)

Metabolic & Insulin Support:

- Berberine, Chromium picolinate, Alpha-lipoic acid

Detox Phase (Short-term):

- Activated charcoal, Chitosan, Psyllium husk

The supplementation protocol was designed to provide foundational micronutrient repletion while addressing chronic inflammation, insulin resistance, and immune dysfunction. Vitamin D3 combined with K2 supported insulin sensitivity and immune modulation while aiding proper calcium metabolism (regulation of insulin receptor expression and inflammatory cytokine suppression). Magnesium played a critical role in glucose metabolism and neuromuscular relaxation (cofactor for insulin signaling enzymes and improved cellular glucose uptake). B-complex vitamins supported mitochondrial energy production and hepatic detoxification pathways (enhanced carbohydrate metabolism and reduced metabolic fatigue), while zinc contributed to immune resilience and pancreatic β-cell function (stabilization of insulin synthesis and antioxidant defense). Omega-3 krill oil was included to reduce systemic inflammation and improve lipid metabolism (inhibition of pro-inflammatory eicosanoids and improvement of triglyceride clearance).

Targeted metabolic supplementation directly addressed insulin resistance and glycaemic dysregulation. Berberine acted as a metformin-like agent by improving glucose uptake and reducing hepatic glucose production (MOA: activation of AMPK and suppression of gluconeogenesis). Chromium picolinate enhanced insulin receptor sensitivity and improved glucose tolerance (potentiation of insulin signaling and reduced postprandial glucose spikes). Alpha-lipoic acid functioned as both an insulin sensitizer and antioxidant (improved GLUT-4 translocation and reduction of oxidative stress). During the short-term detox phase, activated charcoal and chitosan acted as binders to reduce intestinal reabsorption of metabolic toxins (adsorption of endotoxins and bile-bound metabolites), while psyllium husk supported gut motility and glycaemic control (delayed glucose absorption and improved gut barrier integrity).

Table 8: Supplementation Protocol.

	Supplement Category	**Supplement**	**Mechanism of Action (MOA)**	
	Foundational Support	Vitamin D3 + K2	Improves insulin sensitivity and immune regulation by modulating insulin receptor expression and inflammatory cytokines; K2 supports calcium redistribution and vascular health	
		Magnesium	Acts as a cofactor for insulin signaling enzymes, enhances cellular glucose uptake, and supports neuromuscular relaxation	
		B-complex	Supports mitochondrial energy production and carbohydrate metabolism; reduces metabolic fatigue via enhanced enzymatic activity	
		Zinc	Stabilizes insulin synthesis and storage in pancreatic β-cells;	

			supports antioxidant defense and immune function	
		Omega-3 (Krill Oil)	Reduces systemic inflammation and improves lipid metabolism by inhibiting pro-inflammatory eicosanoids and lowering triglycerides	
	Metabolic & Insulin Support	Berberine	Acts as a metformin-like agent by activating AMPK, improving peripheral glucose uptake, and reducing hepatic gluconeogenesis	
		Chromium Picolinate	Enhances insulin receptor sensitivity and improves glucose tolerance by potentiating insulin signaling pathways	
		Alpha-Lipoic Acid	Improves insulin sensitivity and reduces oxidative stress by enhancing GLUT-4 translocation and acting as a cellular antioxidant	
	Detox Phase (Short-term)	Activated Charcoal	Binds endotoxins and metabolic by-products in the gut, reducing enterohepatic recirculation	
		Chitosan	Binds bile acids and lipophilic toxins, supporting lipid clearance and metabolic detoxification	

		Psyllium Husk	Slows glucose absorption, improves gut barrier integrity, and supports regular bowel movements through soluble fiber action	

Lifestyle and Environmental Interventions

Sleep Hygiene and Circadian Restoration

Sleep optimization was treated as a therapeutic pillar, given its direct influence on insulin sensitivity, immune regulation, and neuroendocrine balance. Patient was counselled to align her sleep schedule with the body's natural circadian rhythm by adhering to a "sacred sleep window", aiming to be asleep by 10:00 PM. The period between 10:00 PM and 2:00 AM is critical for growth hormone release, hepatic detoxification, and cellular repair; disruption of this window is strongly associated with insulin resistance and chronic inflammation. Ensuring a minimum of 8 hours of sleep nightly, particularly during detox phases, supported metabolic recovery and adrenal resilience.

To reinforce circadian entrainment, environmental light exposure was modified. All household lighting was transitioned to warm yellow lights, minimizing nocturnal cortisol stimulation and melatonin suppression. Blue light filters were installed on electronic devices, and a strict digital curfew after 9:00 PM was implemented to reduce sympathetic nervous system activation. Collectively, these measures improved sleep quality, reduced nighttime awakenings, and contributed to better fasting glucose control by stabilizing the cortisol–melatonin axis.

Stress Management and Nervous System Regulation

Chronic metabolic and inflammatory conditions are closely linked to autonomic nervous system dysregulation. To address this, daily stress-modulation practices were incorporated. Soma breathwork, practiced upon waking, helped downregulate sympathetic tone and improve vagal activity, supporting glycaemic control and immune balance. Evening meditation, specifically Jeffrey Allen's guided practices, was used to reduce cognitive hyperarousal and facilitate parasympathetic dominance before sleep.

Relaxation therapies further supported nervous system recovery. Hot Epsom salt baths (2 cups magnesium sulfate in warm water for at least 12 minutes) enhanced magnesium absorption, reduced muscular tension, and promoted sleep initiation. Daily lymphatic drainage massage, preferably before bathing, was encouraged to improve circulation, reduce inflammatory load, and support detoxification pathways. These interventions collectively reduced perceived stress, improved emotional resilience, and supported endocrine recovery.

Physical Activity and Movement Strategy

Physical activity was individualized to support metabolic health without exacerbating adrenal or inflammatory stress. Patient continued EMS training, functional workouts, and recreational badminton, which helped preserve lean muscle mass, improve insulin sensitivity, and enhance cardiovascular fitness. However, during detoxification phases, training intensity was deliberately reduced to prevent cortisol overproduction.

During these periods, movement was shifted to low-intensity walking for 20–30 minutes daily, allowing continued glucose utilization while promoting parasympathetic recovery. Additionally, daily sun exposure for 20–30 minutes between 11:00 AM and 4:00 PM supported endogenous vitamin D synthesis, circadian alignment, and metabolic regulation. This balanced approach ensured sustained physical activity while protecting hormonal and immune balance.

Environmental Detoxification and Toxin Reduction

Given the role of environmental toxins in endocrine disruption and insulin resistance, comprehensive environmental detoxification strategies were implemented. In the kitchen, all plastic containers and bottles were replaced with glass or stainless steel to reduce exposure to xenoestrogens, bisphenol A (BPA), and polychlorinated biphenyls (PCBs). Cooking was shifted exclusively to stainless steel cookware and glass bakeware, with avoidance of iron or cast-iron utensils to limit oxidative stress.

Personal care routines were also modified to reduce chemical burden. Synthetic soaps, shampoos, and perfumes were eliminated and replaced with natural alternatives such as Reetha, Shikakai, and essential oils. Household exposure to toxic cleaners, sprays, and detergents was minimized. Food safety practices further reduced toxic load: leafy vegetables were soaked in water with grapeseed oil drops to eliminate parasite eggs, while fruits such as apples were soaked in water with activated charcoal to remove pesticide residues and heavy metals. These interventions reduced cumulative toxin exposure, supporting liver function and metabolic detoxification.

Table 9. Sleep Hygiene and Stress Management Interventions

Category	Intervention	Purpose / Physiological Impact
Sleep Hygiene	Sacred sleep window (10 PM–2 AM)	Supports growth hormone release, insulin sensitivity, and hepatic detoxification
	Yellow lighting	Preserves melatonin secretion and circadian rhythm
	Blue light filters & digital detox	Reduces cortisol elevation and sympathetic activation

Stress Management	Soma breathwork	Improves vagal tone and glycaemic regulation
	Evening meditation	Reduces stress-induced hypercortisolemia
	Epsom salt baths	Magnesium absorption, muscle relaxation, sleep support
	Lymphatic massage	Enhances detoxification and inflammation reduction

Table10 . Physical Activity and Environmental Detoxification

Category	Intervention	Physiological Benefit
Physical Activity	EMS, functional training, badminton	Improves insulin sensitivity and muscle mass
	Low-intensity walking (detox phases)	Maintains glucose utilization without adrenal stress
	Sun exposure (20–30 min/day)	Supports vitamin D synthesis and circadian alignment
Environmental Detox	Plastic elimination	Reduces endocrine-disrupting chemical exposure
	Natural personal care products	Lowers cumulative toxic burden
	Food soaking protocols	Reduces pesticide, parasite, and heavy metal exposure

Lifestyle and environmental interventions formed a critical adjunct to nutritional and supplementation strategies in this case. By restoring circadian rhythm, regulating stress responses, optimizing physical activity, and reducing environmental toxin exposure, these interventions addressed key upstream drivers of insulin resistance, inflammation, and immune dysfunction. The integrated approach contributed significantly to improved metabolic biomarkers, enhanced energy levels, reduced medication dependence, and sustained long-term health improvements.

For the infection management

Fungal and Infection Management Strategy

Given her long-standing history of aspergillosis-associated asthma, recurrent respiratory infections, and chronic fungal skin manifestations, a targeted anti-fungal and anti-infective strategy was incorporated as a core component of her care. Chronic fungal and microbial burden is a known driver of immune dysregulation, persistent inflammation, and insulin resistance, thereby creating a bidirectional link between respiratory disease and metabolic dysfunction. Addressing this axis was critical for achieving sustained symptom resolution.

Targeted supplementation was introduced to reduce fungal load and support immune balance. Candida Support was prescribed to inhibit fungal overgrowth and reduce intestinal dysbiosis, while tea tree oil (2 drops in water) was used for its potent antifungal and antimicrobial properties, particularly effective against Candida and mold-related organisms. To address broader infectious burden contributing to chronic inflammation, Biocidin was incorporated as a botanical antimicrobial formulation targeting bacterial, fungal, and viral pathogens commonly implicated in chronic respiratory conditions. During the maintenance phase, a Wormwood and Black Walnut complex was added to facilitate parasite clearance, reducing immune system strain and supporting gut-immune axis restoration. Collectively, these interventions reduced microbial load, improved immune resilience, and supported respiratory recovery.

Table11: Fungal and Infection Management Protocol and Mechanisms of Action

Intervention Category	Agent / Strategy	Mechanism of Action (MOA)
Targeted Antifungal Support	Candida Support	Inhibits fungal overgrowth by disrupting fungal cell membranes and reducing intestinal dysbiosis; supports gut-immune axis restoration
	Tea Tree Oil (oral, diluted)	Exhibits potent antifungal and antimicrobial activity by damaging fungal cell walls and inhibiting biofilm formation

Broad-Spectrum Antimicrobial	Biocidin	Botanical antimicrobial that targets bacterial, fungal, and viral pathogens; reduces chronic immune activation and inflammatory burden
Parasite Clearance (Maintenance Phase)	Wormwood & Black Walnut Complex	Disrupts parasite metabolism and lifecycle; reduces intestinal parasite load and immune system strain

Dietary and Lifestyle Interventions for Respiratory Health

Dietary modifications played a pivotal role in reducing mucus hypersecretion, airway inflammation, and immune activation. To alleviate congestion and support airway clearance, non-pharmacological respiratory therapies such as steam inhalation, nebulization, and targeted herbal decoctions were implemented. These interventions helped loosen mucus, reduce bronchial irritation, and improve breathing comfort, particularly during periods of seasonal or environmental exposure.

Anti-inflammatory and immune-supportive herbs were emphasized as daily dietary adjuncts. Turmeric, ginger, tulsi, garlic, and oregano were encouraged due to their well-established antimicrobial, anti-inflammatory, and antioxidant properties, which help suppress pro-inflammatory cytokines and support mucosal immunity. A critical dietary intervention was the complete elimination of dairy products, with the exception of limited A2 curd. Dairy is strongly associated with increased mucus production, post-nasal drip, and promotion of pathogenic overgrowth in susceptible individuals; its removal led to a marked reduction in congestion and respiratory discomfort.

Table12. Dietary and Lifestyle Interventions for Respiratory Health and Mucus Reduction

Intervention Type	Strategy	Mechanism of Action (MOA)
Mucus Reduction	Steam inhalation & nebulization	Loosens bronchial secretions, improves mucociliary clearance, and reduces airway irritation

Herbal Respiratory Support	Turmeric, Ginger, Tulsi, Garlic, Oregano	Suppress pro-inflammatory cytokines, exhibit antimicrobial effects, and support mucosal immunity
Dietary Elimination	Dairy removal (except A2 curd)	Reduces mucus production, post-nasal drip, and pathogen feeding; improves gut and respiratory inflammation

Environmental Trigger Control and Spore Prevention

Environmental exposure management was prioritized to prevent recurrent fungal and parasitic reinoculation, which can undermine therapeutic progress. Patient was educated on thorough produce-cleaning techniques to minimize ingestion of fungal spores and environmental contaminants. All raw leafy vegetables including celery, mint, and coriander were mandatorily soaked in water containing four drops of grapeseed oil for 15 minutes, a practice aimed at eliminating parasite eggs and microbial residues.

This comprehensive environmental hygiene protocol reduced ongoing immune activation, supported gut barrier integrity, and minimized respiratory flare-ups. By integrating antifungal therapy, dietary modulation, and environmental control, the intervention successfully addressed a key upstream driver of both Patient chronic asthma symptoms and systemic inflammatory burden, complementing the metabolic improvements achieved during the ALIVE program.

Table13. Environmental Trigger Control and Spore Prevention

Exposure Source	Intervention	Mechanism of Action (MOA)
Fungal Spores on Produce	Deep-cleaning of fruits and vegetables	Reduces ingestion of environmental fungal spores and microbial contaminants
Parasite Eggs on Leafy Greens	Soaking in water with grapeseed oil (15 min)	Kills parasite eggs and reduces microbial load before ingestion

Ongoing Environmental Exposure	Education on food hygiene practices	Prevents reinoculation and chronic immune activation

3. Outcomes and Results

Following four months of structured intervention under the ALIVE program, the patient demonstrated marked clinical, biochemical, and symptomatic improvement, indicating successful reversal of metabolic dysfunction alongside significant respiratory and immune recovery.

Metabolic and Glycaemic Outcomes

Objective biochemical markers showed a substantial normalization of glucose metabolism. HbA1c levels decreased from 8.2% at baseline to 5.4%, placing the patient firmly within the non-diabetic range. Fasting blood glucose improved from 122.5 mg/dL to 88.2 mg/dL, reflecting restored glycaemic control. Markers of insulin resistance improved in parallel, accompanied by a significant reduction in triglyceride levels from 183 mg/dL to 78 mg/dL, suggesting improved lipid handling and hepatic insulin sensitivity. The fatty liver index showed a notable reduction (from 90 to 64), supported by normalization of liver enzymes, indicating improved hepatic metabolic function.

Anthropometric and Symptom Resolution

Patient experienced a weight reduction of approximately 5 kg and a 4-inch reduction in waist circumference, reflecting meaningful visceral fat loss. Subjectively, she reported restored energy levels, absence of cravings, improved sleep quality, and enhanced exercise tolerance. Importantly, she achieved complete discontinuation of metformin under medical supervision without rebound hyperglycaemia.

Respiratory and Immune Outcomes

Chronic respiratory symptoms showed significant resolution. The patient reported a marked reduction in mucus production, cough, nasal congestion, and breathlessness. She was able to discontinue inhaler use, reporting improved breathing comfort even during travel and environmental exposure. Fungal-related symptoms, including skin itching and recurrent congestion, resolved following targeted antifungal and environmental interventions. Inflammatory markers such as ferritin and uric acid decreased, reflecting reduced systemic immune activation.

Table14: Comparative analysis

Category	Clinical Parameter	Baseline (Sept/Oct 2023)	Post-Protocol (March 2024)	Outcome Summary
Glucose Metabolism	HbA1c (%)	8.2% (Patient Value 1/10/23)	5.4% (Patient Value 16/03/24)	Normalization (dropped to optimal range)
	Fasting Blood Sugar	122.5 mg/dl (24/9/23)	88.27 mg/dl (10/03/24)	Optimal Range achieved
	Medication Dependence	Metformin	Medication-Free	Full Diabetes Reversal
Lipid Profile	Triglycerides	183 mg/dl	78 mg/dl	Significant reduction
Liver Health	Fatty Liver Index	90	64	Improvement in Liver Function
Respiratory Health	Asthma Symptoms	Chronic cough/congestion	Symptom-Free	Significant relief from congestion
	Inhaler Use	Regular dependence	Discontinued	Independence from respiratory meds

Physical Markers	Body Weight	Baseline noted	~5 kg total loss	Weight Loss Achieved
	Waist Circumference	Baseline noted	4-inch reduction	Reduction in visceral adiposity
	Energy Levels	Fatigued	Greatly Improved	Elimination of chronic fatigue

4. Discussion

This case contributes to a growing body of evidence challenging the traditional perception of Type 2 diabetes mellitus as an irreversible condition. The observed improvements in glycaemic control and metabolic health following a structured functional nutrition and lifestyle-based intervention suggest that addressing upstream metabolic stressors can lead to meaningful physiological recovery. Rather than targeting blood glucose in isolation, the intervention focused on restoring metabolic balance across interconnected systems, including hepatic function, insulin signaling, inflammatory regulation, and mitochondrial efficiency.

A key factor underlying the observed metabolic improvement is likely the reduction in insulin resistance, particularly at the hepatic level. Excess caloric intake, refined carbohydrates, and chronic metabolic overload are known to promote intrahepatic fat accumulation, leading to inappropriate hepatic glucose output. Nutritional strategies that reduce glycaemic load and support liver function have been shown to rapidly improve fasting glucose levels, often preceding significant weight loss. The metabolic improvements observed in this case are consistent with this mechanism, reinforcing the central role of hepatic insulin sensitivity in early disease reversal.

Pancreatic β-cell stress also appears to be a modifiable component of Type 2 diabetes pathophysiology. Chronic hyperglycaemia and elevated free fatty acids contribute to glucolipotoxicity, impairing insulin secretion over time. By reducing metabolic demand and improving peripheral insulin sensitivity, the intervention likely decreased secretory pressure on β-cells, allowing partial functional recovery. This aligns with emerging

evidence suggesting that β-cell dysfunction in T2DM is not universally permanent, particularly in individuals with shorter disease duration or preserved residual function.

The inclusion of detoxification-supportive strategies may have further contributed to metabolic improvement by reducing inflammatory burden and improving hepatic biotransformation capacity. While the term “detoxification” is often used imprecisely, growing evidence supports the role of environmental toxins, oxidative stress, and impaired xenobiotic metabolism in promoting insulin resistance and mitochondrial dysfunction. Supporting endogenous detoxification pathways through nutrition and lifestyle modification may therefore represent an underappreciated adjunct in metabolic restoration.

Importantly, this case highlights the systemic nature of metabolic dysfunction in T2DM. Improvements extended beyond glycaemic indices to include enhanced energy regulation, digestive function, and overall well-being, reflecting restoration of metabolic flexibility rather than isolated glucose control. Such outcomes underscore the limitations of a glucose-centric treatment model and support a systems-based approach to metabolic disease.

Despite these encouraging findings, several limitations must be acknowledged. As a single case report, causality cannot be definitively established, and the results may not be generalizable to all individuals with T2DM. Adherence, baseline metabolic reserve, disease duration, and genetic factors likely influence responsiveness to intervention. Additionally, long-term follow-up is required to assess durability of metabolic improvements. These limitations highlight the need for controlled clinical trials and standardized outcome measures to further validate integrative metabolic restoration strategies.

Conclusion

This case report provides evidence that significant metabolic improvement in Type 2 diabetes mellitus is achievable through a structured intervention targeting nutrition, lifestyle, and metabolic stress at its roots. The findings support a reframing of T2DM as a dynamic and potentially reversible metabolic condition rather than an inevitably progressive disease. By reducing insulin resistance, alleviating hepatic and pancreatic stress, and improving systemic metabolic function, meaningful physiological recovery can occur beyond what is typically achieved through glucose-lowering therapies alone.

While pharmacological treatments remain important for glycaemic safety and complication prevention, this case illustrates the potential value of integrative, physiology-focused strategies as complementary tools in diabetes care. Early

implementation, individualized metabolic assessment, and sustained lifestyle support appear critical for achieving durable outcomes. Future research should focus on larger, controlled studies to determine reproducibility, identify predictors of response, and establish standardized frameworks for metabolic restoration in clinical practice. Collectively, these findings reinforce the importance of addressing root metabolic dysfunction as a central goal in the management of Type 2 diabetes.

Supplementary information:

Supplementary Table 15: Suggested Supplementation Program

No.	Supplement Name	Brand Name	Manufacturer	Primary Purpose
Essential Supplements				
1	**Vitamin D3+K2**	Vitamin D3 + K2	Seeking Health	Supports bone health, immunity, and cardiovascular health.
2	**Magnesium**	Magnesium Glycinate	NOW Foods	Energy production, muscle function, and relaxation.
3	**B Complex**	BioActive Complete B-Complex	Life Extension	Supports methylation and addresses nutrient deficiency anemia.
4	**Zinc**	Zinc Balance	Jarrow Formulas	Immune support and nutrient metabolism.

5	**Probiotics**	Probiotic-10, 25 Billion	NOW Foods	Maintains healthy gut flora and microbial balance.
6	**Omega-3**	Krill Oil	Jarrow Formulas	Reduces inflammation and oxidative stress; improves HDL.
Case Specific / Maintenance				
7	**Berberine**	Berberine Glucose Support	NOW Foods	Supports blood glucose levels and insulin sensitivity.
8	**Optimal PC**	Optimal PC	Seeking Health	Improves liver function and cellular health.
9	**Glycine**	Glycine 1,000 mg	NOW Foods	Supports liver detoxification and overall health.
10	**Candida Support**	Candida Support	NOW Foods	Targets fungal infection and balances gut flora.

11	**Tea Tree Oil**	Essential Oils Tea Tree	NOW Foods	Antifungal support (taken internally as directed).
12	**Biocidin**	Biocidin® (Liquid/Pump)	Biocidin Botanicals	Broad-spectrum support for addressing infection levels.
13	**GI Detox**	G.I. Detox™	Biocidin Botanicals	Gentle binder for removing debris and toxins.
14	**Alpha Lipoic Acid**	Alpha Lipoic Acid 600	Doctor's Best	Antioxidant support for managing insulin resistance.
15	**Chromium**	Chromium Picolinate	Swanson	Mineral support for insulin sensitivity.
16	**EAA**	Amino Complex	Thorne	Essential amino acids to meet protein demands.

17	**Parasite Support**	Green Black Walnut Wormwood	NOW Foods	Used during the maintenance phase to clear parasites.

Supplementary Information for understanding the timeline

Initial Diagnostics and Baseline

- **November 2022:** Diagnosed as diabetic and began taking Metformin.
- **September 24, 2023:** Baseline Fasting Blood Sugar was recorded at **122.5 mg/dl**.
- **October 1, 2023:** HbA1c was recorded at its baseline of **8.2%**.

Progress Monitoring (Intermediate Dates)

- **October 21, 2023:** Fasting Blood Sugar was measured at **118.9 mg/dl**.
- **October 28, 2023:** Fasting Blood Sugar decreased to **102.69 mg/dl**.

Post-Protocol and Final Results

- **March 10, 2024:** Fasting Blood Sugar reached its optimal level of **88.27 mg/dl**.
- **March 16, 2024:** HbA1c was recorded at its final post-protocol value of **5.4%**.
- **March 28, 2024:** The client provided final consent and shared her success story on the wellness group.

Table 16: General Timeline

Metric	Baseline (Sept/Oct 2023)	Final (March 2024)
HbA1c	8.2%	5.4%
Fasting Blood Sugar	122.5 mg/dl	88.27 mg/dl
Triglycerides	183 mg/dl	78 mg/dl

Fatty Liver Index	90	64
Body Weight	Baseline noted	~5 kg total loss