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CASE REPORT

Rapid Reduction of Severe Hypertriglyceridemia and Hyperglycemia in Metabolic Syndrome through Ayurvedic Intervention: A Case Report

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ABSTRACT

Type 2 diabetes mellitus with severe hypertriglyceridemia represents a critical metabolic emergency carrying a high risk of acute pancreatitis and cardiovascular events. Conventional pharmacological approaches may require weeks to achieve glycemic and lipid targets. Ayurveda offers multitarget interventions that have the potential for rapid metabolic restoration. Here is a case report of a 50-year-old male with known hypertension (4 years) and dyslipidemia presenting with complaints of nocturia, polydipsia, and craving for sweet and cold foods. Investigations revealed hyperglycemia and hypertriglyceridemia, but the patient refused to take conventional medicines. A 15-day Ayurvedic treatment protocol comprising *Punarnavashtak Kwath*, *Madhumehari Churna*, *Paniya Kalpana* of *Daruharidra*, *Guduci*, *Musta*, and *Arogyavardhini Vati*, combined with prescribed dietary modifications, resulted in rapid clinical improvement. At the 15-day follow-up, all parameters, including fasting blood sugar and triglyceride levels, were within normal limits. This case demonstrates the potential of a structured Ayurvedic protocol to achieve rapid, clinically significant metabolic correction in severe hyperglycemic–dyslipidemia crisis.

1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder of global pandemic proportions. The International Diabetes Federation estimated 537 million adults living with diabetes worldwide in 2021, with India bearing the second-highest burden at approximately 74.2 million cases. The coexistence of severe hypertriglyceridemia (triglycerides ≥1,000 mg/dL) with uncontrolled T2DM is associated with a high risk of acute pancreatitis.

Ayurveda describes a disease cluster termed *Prameha* (~obstinate urinary diseases) that closely resembles the modern description of diabetes, metabolic syndrome, and insulin resistance. Classical Ayurvedic texts, including the *Charaka Samhita* and the *Sushruta Samhita,* mention 20 varieties of *Prameha,* among which *Madhumeha* represents a clinical entity equivalent to T2DM.[1]

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The Ayurvedic pathophysiology of *Prameha* involves vitiation of *Kapha dosha* and *Medas dhatu*, leading to obstruction of *Basti* (~ urinary bladder) and *Srotas* (~ structural/functional channels). This pathogenesis bears remarkable conceptual parallels to insulin resistance, adiposity, and dyslipidemia in modern medicine. Accordingly, Ayurvedic treatment focuses on pacifying *Kapha-Meda, Sroto-shodhana* (~channel purification), and restoring *Agni* metabolism.

This case report, prepared in accordance with the 2013 CARE guidelines, describes the remarkable response of a patient with newly diagnosed diabetes and dyslipidemia to a 15-day Ayurvedic intervention.

2. PATIENT INFORMATION

A 50-year-old Indian male presented to the *Kayachikitsa* (internal medicine) outdoor patient department on July 30, 2025. He reported a 20-day history of the following symptoms: (1) Increased frequency of urination, predominantly nocturnal (nocturia); (2) increased thirst with intense craving for sweet and cold food items; and (3) increased weight.

The patient has been a known case of hypertension (HTN) and dyslipidemia for approximately 4 years, for which he was receiving olmesartan medoxomil (40 mg) + amlodipine (5 mg) + hydrochlorothiazide (12.5 mg) once a day, but still had uncontrolled blood pressure ranging from 140 to 150/90 to 100 mm Hg. No known drug allergies were reported. The patient's father is also a known case of HTN and diabetes. The patient was a non-smoker and reported no addictions.

3. CLINICAL FINDINGS

The patient's appetite and bowel habits were normal, and the patient had no difficulty sleeping. On general examination, pulse was 84 bpm, blood pressure was 110/70 mmHg, waist circumference was 100 cm (~39.5 inches), body weight was 95 kg, and height was 185.4 cm, yielding a body mass index of approximately 27.7 kg/m^2 (obese category, as per Asia-Pacific guidelines).

For Ayurvedic assessment, *Ashtasthana pariksha* (~eight-fold examination of the patient) was done (Table 1).

4. TIMELINE

A detailed timeline of events is illustrated in Table 2.

5. DIAGNOSTIC ASSESSMENT

The patient's diagnosis was made based on the patient's history, clinical presentation, and test findings. Initial laboratory investigations (July 26, 2025) are presented in Table 3.

Ultrasonography of the whole abdomen (performed July 29, 2025) revealed: (1) Grade I fatty liver with hepatomegaly (liver span 182 mm); (2) increased parenchymal echogenicity with bright appearance, and (3) left renal simple cortical cyst measuring 61 × 60 mm. The remaining abdominal organs were reported within normal limits. This finding is consistent with studies indicating that T2DM-induced hyperglycemia enhances the pathogenicity of non-alcoholic fatty liver disease (NAFLD) by increasing triglyceride, LDL-C, and glucose levels.[2]

The Ayurvedic diagnosis integrates the classical framework of *Nidana* (etiology), *Samprapti* (pathogenesis), and *Lakshana* (symptomatology). The patient's presenting features – excessive urination (*Prabhuta mutrata*), craving for sweet/cold (*Madhura-Sheetepsa*), and weight gain (*Sthoulya*) – are classical cardinal signs of *Prameha* as described in *Charaka Samhita.*

The *Samprapti* (disease mechanism) was identified as *Kapha-Mamsa-Medas* dominance with secondary *Pitta* and *Vata* involvement, obstructing the *Mootravaha srotas* (urinary channels) and *Medovaha srotas* (lipid channels), consistent with the combined presentation of hyperglycemia, hyperlipidemia, and hepatic steatosis.

6. THERAPEUTIC INTERVENTION

The Ayurvedic treatment protocol was prescribed across three consecutive prescriptions (July 30, August 16, and October 18, 2025). The core formulations used are described in Table 4 with their classical rationale.

Treatment of the patient was carried out in accordance with the management guidelines for *Santarpana Nimitta Vyadhi* and *Prameha*.[3] For *Apatarpana chikitsa*, specific *lekhana dravyas* were chosen to break the pathological axis of *Kapha-Mamsa-Medas dushti* and to clear obstructions in the *Mutravaha* and *Medovaha srotas*.

6.1. Dietary and Lifestyle Modifications

The patient's detailed dietary and lifestyle history was taken, and, as per the management guidelines for *Santarpan-nimittaja vikara*, the patient received a structured, handwritten *Pathya-Apathya* chart, which is provided in Chart 1 to 4.

7. FOLLOW-UP AND OUTCOMES

7.1. Primary Clinical Outcomes at 15 Days

Remarkable improvement was documented at the first 15-day follow-up visit. Key outcome parameters are presented in Table 5.

7.2. Follow-up

At subsequent follow-up visits, the patient reported continued improvement in the chief complaints. After 11 weeks of treatment initiation, HbA1c decreased from 12.5% to 7.2%, indicating sustained improvement in glycemic control. Fasting blood sugar (FBS) remained within the normal range at 82 mg/dL. Triglycerides were 139 mg/dL within the borderline-normal range. The lipid profile otherwise showed total cholesterol 122 mg/dL, LDL 45 mg/dL, and HDL 49 mg/dL, all within normal limits.

8. DISCUSSION

The various pathogenic pathways of metabolic syndrome include insulin resistance, chronic inflammation, and neurohormonal activation.[4]

Defects in insulin action and hyperglycemia could lead to changes in plasma lipoproteins in patients with diabetes. The lipoprotein abnormalities commonly present in Type 2 diabetes, previously termed non-insulin-dependent diabetes mellitus, include hypertriglyceridemia and reduced plasma HDL cholesterol. In contrast to Type 1 diabetes, this phenotype is not usually fully corrected with glycemic control. Therefore, abnormalities in insulin action, rather than hyperglycemia *per se*, are associated with this lipid abnormality.

This case presents a remarkable magnitude and rapidity of metabolic response: An 88.4% reduction in FBS (727–84 mg/dL) and 89% reduction in triglycerides (1,147–126 mg/dL) within 15 days is clinically significant. In conventional medicine, standard pharmacological management of severe hypertriglyceridemia typically achieves a 30–50% reduction in TG over 4–12 weeks.[5] Concurrent glycemic normalization, usually requiring weeks of pharmacotherapy titration, reinforces the hypothesis of a multitarget mechanistic effect.

The Ayurvedic diagnosis of *Prameha* provides a conceptual framework that addresses the underlying metabolic syndrome – rather than treating isolated glycemic or lipid parameters, which are consistent with modern evidence linking insulin resistance as the unifying pathophysiology behind T2DM, dyslipidemia, NAFLD, and HTN.

Several formulations in this protocol have pharmacological mechanisms that could account for the observed outcomes:

Punarnavashtak kwatha – As it has *Tikta, Kashaya, Ushna, Laghu*, and *Ruksha Lekhana Dravya,* which help in *Ama-Pachana, Sroto-Shodhana*, and the removal of excess *Kleda* and kapha from *Mutravaha Srotas*. *Madhumehari Churna* acts through *Agnideepana*

(enhancing digestive fire), *Amapachana* (digesting metabolic toxins), *Srotoshodhana* (cleansing channels), and *Kapha-Medohara* (reducing excess Kapha and fat). Its contents have been documented to have hypoglycemic, antioxidant, and insulin-sensitizing properties in experimental models. *Boerhavia diffusa* (Punarnava) has documented diuretic, anti-inflammatory, and anti-fibrotic effects relevant to the renal and hepatic findings in this case.

Daruhaldi contains berberine, a well-documented AMPK activator with hypoglycemic, lipid-lowering, and hepatoprotective effects comparable to metformin in pre-clinical studies.[6] *Giloy* (*Tinospora cordifolia*) and *Nagarmotha* (*Cyperus rotundus*) have immunomodulatory and antihyperglycemic action.[7,8] Multiple meta-analyses confirm berberine's efficacy in reducing FBS, HbA1c, and triglycerides, with mechanisms including AMPK activation, inhibition of dipeptidyl peptidase-4, and gut microbiome modulation.[9] *Tinospora cordifolia* (*Giloy*) demonstrated alpha-glucosidase inhibition, enhanced insulin secretion, and antioxidant activity in experimental models.[10]

Arogyavardhini Vati is a classical mineral-herbal formulation used for liver disorders, obesity, and metabolic syndrome. Contains purified *Shilajit, Triphala, Chitrak, Kutki, Tamra Bhasma*, and *Loha Bhasma*. Documented hypolipidemic, hepatoprotective, and anti-obesity actions; particularly relevant for NAFLD and dyslipidemia. It is reported to significantly reduce serum triglycerides, total cholesterol, and liver enzymes in clinical studies of NAFLD and metabolic syndrome, potentially through the hepatoprotective and iron-regulatory effects of *Kutki* (*Picrorhiza kurroa*) and *Loha Bhasma*.[11]

However, critical methodological limitations must be acknowledged. This is a single-patient observation without a control arm. The simultaneous dietary and lifestyle modification – often underestimated – is a powerful confounding factor. Severe caloric restriction combined with a high-fiber, low-glycemic diet alone can produce dramatic FBS and TG reductions within days in newly diagnosed T2DM.[12] Disentangling pharmacological contributions from dietary effects is not possible in this single-case design.

9. CONCLUSION

This case report documents a clinically significant and rapid metabolic response to a structured Ayurvedic intervention in a patient with newly diagnosed T2DM presenting as a metabolic crisis with an FBS of 727 mg/dL, HbA1c of 12.5%, and severely elevated triglycerides of 1,147 mg/dL. Within 15 days, FBS normalized to 84 mg/dL (88.4% reduction), triglycerides fell to 126 mg/dL (89% reduction), body weight decreased by 6 kg, and symptomatic improvement was reported.

The Ayurvedic framework of *Prameha* management – targeting *Kapha-Medo dushti* through multitarget formulations offers a biologically plausible mechanism consistent with modern pharmacology. The concurrent dietary modification (*Pathya*) likely played a synergistic, possibly dominant role.

This case demonstrates the potential of Ayurvedic pharmacotherapy to facilitate rapid metabolic normalization during a crisis. Prospective randomized controlled trials are needed to validate these observations, establish optimal dosing regimens, and distinguish pharmacological efficacy from contributions of dietary effects in Ayurvedic management of T2DM with metabolic syndrome.

10. PATIENT PERSPECTIVE

The patient reported significant subjective improvement in quality of life within the first 15 days of treatment. Nocturia, the most distressing presenting complaint, was markedly reduced, enabling better sleep quality. The patient expressed relief at avoiding insulin initiation for severe hyperglycemia, which had been a concern. Appetite and energy levels normalized. The patient remained adherent to the prescribed dietary regimen and herbal medications throughout the documented follow-up period.

Written informed consent was obtained for the use of anonymized clinical data for academic publication.

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Nil.

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All authors give equal contributions to the making of this manuscript.

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14. CONFLICT OF INTERESTS

The authors declare no conflicts of interest regarding the publication of this paper.

15. ETHICAL STATEMENT

Ethical approval was not required for this study as it is a case report.

16. DATA AVAILABILITY STATEMENT

The data analyzed in this review were obtained from publicly available sources, including peer-reviewed articles, observational studies, and surveys accessible through databases.

17. DISCLAIMER/VIEW AND OPINIONS

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18. AI-USE DECLARATION

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Table 1: *Ashtavidha Pariksha*

Nadi (~pulse)	Gambheera*, manda* (~slow);
Mala (~excreta)	*Sama*
Mutra (~urine)	*Kincita pita varna* (~slightly yellow in color), bahumutrata, madhur gandha
Jihwa (~tongue)	*Sama, shweta, picchila*
Shabda (~voice)	*Gambhira*
Sparsha (~tactile examination)	*Ruksha, Sakandu*
Drik (~eye)	*Shweta, Snigdha*
Akruti (~body stature)	*Sthoola*

Table 2: A detailed timeline of events

Date	Signs and symptoms	Investigations	Management
July 2025	Onset of nocturia, increased thirst, craving for sweet and cold items, weight gain, lethargic feeling		
July 26, 2025	The intensity of these symptoms increased	Fasting blood sugar (727 mg/dL), HbA1c (12.5%), triglycerides (1,147 mg/dL), Total cholesterol (203 mg/dL), HDL cholesterol (35 mg/dL)	
July 29, 2025		USG whole abdomen • Grade-1 Fatty Liver • Hepatomegaly 162 mm with parenchymal bright echogenicity • Left renal simple cortical cyst (61×60mm)	
July 30, 2025	The patient visited *Kayachikitsa* OPD as he refused to take oral hypoglycemic agents		Oral medications, along with *Pathya-Apathya,* were prescribed
August 16, 2025	Moderate improvement in nocturia and lethargic feeling Lost 6 kg of weight	Fasting blood sugar (84 mg/dL), triglycerides (126 mg/dL), total cholesterol (81 mg/dL), HDL cholesterol (44 mg/dL)	Continued with the same oral medicines and *pathya-apathya*
September 18, 2025	Complete improvement in nocturia.		Continued with the same oral medicines and *pathya-apathya*
October 9, 2025		HbA1c (7.2%)	Oral medicines were continued with *pathya-apathya*
November 9, 2025	Relief in all symptoms		Advised to continue *pathya-apathya*

HbA1c: Hemoglobin A1C

Table 3: Laboratory parameters

Parameters	Results
Fasting blood sugar (mg/dL)	727
HbA1c (%)	12.5
Triglycerides (mg/dL)	1,147
Total cholesterol (mg/dL)	203
HDL-cholesterol (mg/dL)	35
LDL-cholesterol (mg/dL)	44
VLDL-cholesterol	124
Non-HDL cholesterol	168
LDL: HDL cholesterol	1.26
High sensitivity CRP	7.16 mg/L
RFT	Urea (53.50 mg/dL), creatinine (1.49 mg/dL)
LFT	SGPT (52MG/DL) and ALP (174 U/L)

Table 4: Ayurvedic formulations prescribed, dosage, and classical rationale

Formulation	Dose and frequency	Rationale
Punarnavashtak Kwath (Decoction)	40 mL, twice, on an empty stomach	For detoxification and to expel excess *kleda* through diuresis
Madhumehari Churna (Powder)	6 g, twice, before meals with lukewarm water	To bring down blood-glucose levels and reduce excess *Kapha dosha* and fat.
Daru Haldi (*Berberis aristata*), *Guduci,* and *Nagarmotha Paniya Kalpana*	*Daruharidra* 3 g+ *Guduci* 3 g+ M*usta* 3 g medicated water	To improve fat and glucose metabolism
Arogyavardhini Vati (Tablet)	500 mg, twice after meals with lukewarm water	To scrape out excess fatty deposition and improve gut metabolism

Table 5: Treatment Outcomes

Parameter	Baseline (July 26 and 29, 2025)	15th-day (August 16, 2025)	2nd follow-up (October 8, 2025)	Change (%)
Fasting blood sugar (mg/dL)	727	84	82	88.7
Hemoglobin A1C	12.5%		7.2%	42.4
Triglycerides (mg/dL)	1147	126	139	87.9
Body weight (kg)	95	89	85	10.5
Nocturia (episodes/night)	4–5	1–2	1	75–80
Waist circumference	39.5 inches	37 inches	35.5 inches	10.1
Blood pressure (mmHg)	150/80	110/70	110/70	Change from Stage 2 hypertension to normal range

Chart 1: Pathya Ahara (recommended foods)

Category	Permitted food items	Clinical remarks
Cereals and grains	Barley, Bajra, Maize/Corn, Chickpea	Preferred — coarse grains; low glycaemic index; satiety-promoting
Pulses and legumes	Moong, Masoor, Horse gram	Lighter dals preferred; moderate quantity
Rice	Old rice only	In limited quantity; fresh rice to be avoided
Green vegetables	Bottle Gourd, Bitter Gourd, Pointed Gourd, Ridge Gourd, Sponge Gourd, Apple Gourd, Eggplant, Fenugreek, Fennel, Spinach, Amaranth Leaves	Freely permitted — low-carb, high-fiber vegetables
Other vegetables	Cucumber, Long Melon	Permitted; cooling and low calorie
Fruits	Papaya, Sapota (Chikoo), Apple, Nutgrass, Jamun, Amla, Raisins — small qty	Antioxidant-rich; blood sugar modulating
Dairy	Buttermilk	Milk — limited; buttermilk in restricted qty; NO milk at night
Oils	Mustard oil	Preferred cooking medium
Beverages	Lukewarm water	Drink warm/room temperature; only when thirsty
Prepared dishes	Khichdi, Daliya, Soup	Light, easily digestible preparations
Miscellaneous	Roasted grains, Barley-chickpea sattu	Digestive support; sustained energy release
Condiments and herbs	Garlic, Dry ginger/Sonth, Boiled radish)	Metabolism-boosting; insulin sensitizing

Chart 2: Apathya: Foods to avoid/restrict

Category	Items to avoid/restrict	Clinical rationale
Heavy Grains	Excess rice, Fresh wheat preparations	High glycemic index; aggravate Kapha dosha
Root Vegetables	Potato, Cauliflower, Kidney Beans, Elephant Foot Yam, Jackfruit, Taro Root	High starch; rapid blood glucose elevation
Banana	Banana	High glycaemic fruit; increases weight
High-Fat Dairy	Curd, Cottage Cheese, Dried Milk Solids (Mawa), Cream (Malai), Thickened Sweet Milk (Rabri), Rice Pudding (Kheer), Sweetened Strained Yogurt (Shrikhand), Buffalo Milk	High fat; promotes obesity; raises blood sugar
Sugar and Sweeteners	Excess salt and sugar	Directly raises blood glucose; promotes fluid retention
Egg	Meat, Egg	As per protocol; inflammatory potential
Alcohol	Alcohol	Hepatotoxic; impairs glucose metabolism
Fast food and drinks	Fast food, Cold drinks, Refined flour	Empty calories; dysglycaemic; obesity-promoting
Cold items	Cold water, Cold foods, Ice cream	Impairs digestion (Agni); aggravates Kapha
Milk at Night	Milk at bedtime	Increases Kapha; promotes weight gain overnight
Fried Items	Excess fried food; limit peanuts	High saturated fat; promotes dyslipidemia
Unsuitable Fruits	Milk-fruit combination	Incompatible food combination (Viruddhahara)
Tea	Tea — especially with milk/sugar	Stimulant; raises insulin; disturbs sleep
Stale Food	Stale food	Toxin formation; impairs digestion

Chart 3: Meal timing guidelines

Time/occasion	Dietary instruction
Morning	Wake up early, begin with lukewarm water on an empty stomach
Between meals	Avoid snacking between meals
Dinner timing	Dinner to be completed before 7:00 PM
Post-Meal	Minimum 3-h gap between dinner and sleep
Meal regularity	Eat at fixed times every day
Water intake	Drink water only when thirsty; avoid drinking in large quantities at once
Intermittent fasting	1–2 fasts/week recommended
Bedtime	Sleep on time; avoid daytime sleeping

Chart 4: Lifestyle recommendations

Type	Lifestyle recommendation	Clinical rationale
Do ✓	Wake up early daily	Regulates circadian rhythm; improves insulin sensitivity
Do ✓	Sleep on time every night	7 h minimum sleep; metabolic restoration
Do ✓	Daily brisk walk/ light exercise	Promotes glycogen utilization; reduces visceral fat
Do ✓	Physical labor/ active work	Non-exercise activity thermogenesis
Do ✓	Light massage/ gentle stretching	Improves circulation; reduces stiffness
Do ✓	Warm fomentation when restless	Relieves local discomfort without oil massage
Avoid ✗	Cold air/cold wind exposure	Impairs peripheral circulation; aggravates Kapha
Avoid ✗	Cold water bathing/drinking	Suppresses Agni (digestive fire)
Avoid ✗	Prolonged sitting in one place	Increases insulin resistance; promotes obesity
Avoid ✗	Oil massage when feeling uneasy	Contraindicated in acute symptoms
Avoid ✗	Skipping meals or irregular eating	Dysregulates blood glucose; promotes overeating
Avoid ✗	Daytime sleep	Increases Kapha; promotes weight gain