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Case Presentation

# Longitudinal Analysis of Myocardial System Dysfunction using Multifunction Cardiogram Technology in a 55-year-old Male: A Clinical Case Report

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## Abstract

**Background:** Hypertension-related myocardial dysfunction can be difficult to detect with traditional diagnostic tools, particularly when structural abnormalities are absent.

**Case summary:** A 55-year-old male with hypertension, hypothyroidism, metabolic syndrome, and long-term testosterone therapy underwent four serial Multifunction Cardiogram (MCG) evaluations across 26 months. He reported intermittent right-sided chest pain following COVID-19 infection and mRNA-based vaccination. Initial MCG findings demonstrated severe metabolic dysfunction and small-vessel or functional ischemia. Serial evaluations revealed fluctuating patterns of local and global ischemia influenced by hormonal, inflammatory, and metabolic factors. Improvements occurred after optimizing antihypertensive therapy, reducing exogenous hormone doses, and adopting dietary modifications.

**Conclusion:** Longitudinal MCG monitoring provided unique physiologic insights into the progression and partial reversibility of myocardial dysfunction in a complex cardiometabolic case. These findings suggest that serial MCG assessments may complement standard diagnostics and support individualized management strategies.

## Introduction

Hypertension contributes substantially to global morbidity and mortality, and early identification of physiologic myocardial dysfunction remains a clinical challenge.

Conventional diagnostic tools such as ECG and echocardiography may miss subtle metabolic or microvascular abnormalities that precede structural disease [1]. The Multifunction Cardiogram (MCG) is a noninvasive computational systems engineering-based electro-physiological platform designed to detect physiologic patterns associated with ischemic, metabolic, and mitochondrial dysfunction.

Each year, cardiovascular disease (CVD) causes more than 18 million deaths worldwide, accounting for approximately one-third of global fatalities, a figure projected to increase. High blood pressure is the leading cause of CVD [2], resulting in nearly 10 million deaths this year—half of all CVD fatalities—

and surpassing the total deaths from infectious diseases combined [3]. Healthcare providers managing hypertension lack accurate and timely bedside tools to detect, diagnose, monitor, and assess the systemic physiological impacts of their treatment decisions. This technological gap limits clinicians' ability to identify warning signs of organ damage and reliably, effectively and objectively evaluate the success or failure of diagnostic and treatment strategies. As a result, morbidity and mortality rates among patients with poorly managed hypertension—due to inadequate diagnostic tools and suboptimal treatments—remain high.

Advanced deep learning systems in the physiological domain employ intelligent technology to identify distinct computationally derived patterns in Premier Heart's Multifunction Cardiogram™, the MCG [4]. The MCG is a super domain intelligent computational systems physiology agent designed to assist clinicians in diagnosing heart disease at the bedside or anywhere with Internet access. Over the past decades [5-17], empirically identified, reproducible, thoroughly validated, and clinically verified digital mathematical patterns from the MCG have revealed systemic physiological imbalances caused by poorly managed hypertension and other underlying conditions of chronic diseases, such as ischemic heart disease from obstructive coronary artery disease, metabolic disorders, myocarditis, cardiomyopathy, and congenital structural abnormalities. We believe that using this patient-centered, accessible 21st-century digital technology at the bedside can enhance diagnosis and treatment decisions through early detection and preventive measures, improved control of hypertension, objective and quantitative disease monitoring, and the prevention, slowing, or reversal of progression toward acute heart failure and early death.

This case report describes a 55-year-old male who underwent four serial MCG assessments over 26 months. The case illustrates dynamic physiologic changes influenced by metabolic, endocrine, inflammatory, and post-COVID conditions.

**Novelty of this case:** This report represents one of the first longitudinal evaluations using serial MCG testing to track myocardial recovery and physiologic fluctuation in a patient with overlapping endocrine, metabolic, and post-viral cardiometabolic risk factors.

## Case presentation

### Patient information

A 55-year-old male pharmacist and primary care provider presented with hypertension, hypothyroidism (more than 20 years on Synthroid 200 µg daily), adrenal fatigue (2023), metabolic syndrome with fatty liver, and sleep apnea (diagnosed December 2023; uses CPAP). The patient had a family history of coronary artery disease. He reported COVID-19 infection in August 2023 and received two Moderna mRNA-based vaccines (January and February 2021). Medications included Losartan, Metoprolol, Rosuvastatin, Zetia, and testosterone replacement therapy (TRT) since 2017. He was a former competitive powerlifter, consumed alcohol occasionally, and adopted a Mediterranean diet in 2024.

### Clinical history

Before the onset of cardiovascular symptoms, thyroid function was stable on levothyroxine. The patient started TRT in 2017 after reviewing evidence suggesting cardiovascular benefits [18]. However, he later became concerned about TRT-related pulmonary effects [19]. By 2019, antihypertensive and lipid-lowering therapies were initiated for uncontrolled hypertension. Despite pharmacologic management, blood pressure stayed at 150–160/90 mmHg, with a resting heart rate around 105 bpm. In 2023, after a COVID-19 infection, intermittent right-sided chest pain and hypertensive crises (>200 mmHg systolic) developed.

## Investigations

### MCG Test #1 — August 25, 2023

Severity Score: 4.0 (Category D). Severe right-sided small-vessel functional ischemic heart disease; evidence of both acute and chronic myocardial damage (5/5); cardiomyopathy (5/5); mild mitochondrial dysfunction (1/10). Interpretation: localized ischemic heart disease severe metabolic dysfunction with suspected hormonal contributors; retrospective interpretation suggested possible vaccine-associated myocarditis. Follow-up: MTHFR genotyping revealed heterozygosity for the A1298C variant. Thyroid function normalized.

### MCG Test #2 — March 24, 2024

**Severity Score:** 5.4 (Category E). Progression from local ischemia to global ischemia, per MCG criteria, without evidence of anatomically obstructive coronary artery disease; left coronary territory involvement leading to global ischemia; severe mitochondrial dysfunction (8/10); elevated Arrhythmia risk. Interventions: Advised to avoid heavy exertion; started methylfolate 15–20 mg/day; plasma histamine levels were elevated; adopted a Mediterranean diet.

### MCG Test #3 — May 2, 2024

**Severity score:** 2.8 (Category B). Significant improvement in myocardial function; no evidence of anatomic obstructive coronary artery disease; ongoing alternating local and global ischemia (right > left); myocardial damage rated 5/5; myocarditis 2/5; mitochondrial dysfunction 5/10; single PVC noted. Follow-up: Cardio IQ and S1 monocyte panels negative for spike protein; coagulation and OmegaCheck values normal. Interpretation: Unique to the MCG, improvements

in metabolic and mitochondrial function have been observed following lifestyle and medication adjustments.

### MCG Test #4 — October 4, 2025

**Severity score:** 3.7 (Category D). Shows moderate dysfunction with early ventricular fibrillation risk; ongoing small-vessel or functional ischemia of metabolic origin; no myocarditis or large-vessel disease. Medication Changes: Adjusted antihypertensive treatment; lowered TRT and thyroid hormone dosages. Patient Observation: The patient believed that improved blood pressure control could potentially lower the MCG category to B.

### Outcome and follow-up

Over 26 months, serial MCG evaluations revealed fluctuating myocardial supply-and-demand imbalance in performance influenced by hormonal, inflammatory, and metabolic factors. Improvements were observed following medication adjustments, dietary changes, and reductions in exogenous hormone use. No macrovascular coronary disease or evidence of active myocarditis was identified during the final assessment.

This investigation is part of ongoing global efforts, including independent user-initiated Phase IV and V clinical validations, intended to support further validation of the MCG technology. Additional efforts are underway to develop decentralized database registries that enable objective, widespread adoption of the MCG to promote early detection, disease prevention, and physiological monitoring of the entire cardiovascular system, support chronic disease monitoring and promote healthier, longer lifespans.

## Discussion

This longitudinal case demonstrates the potential clinical utility of the Multifunction Cardiogram (MCG) in detecting physiologic cardiac changes that evolve over time in complex cardiometabolic patients. Traditional diagnostic tools such as ECG and echocardiography often lack the sensitivity to identify subtle metabolic or microvascular dysfunction, particularly when structural disease is minimal or absent. Recent literature emphasizes the role of physiologic signal-analysis platforms, including AI-enhanced cardiologic diagnostics, in identifying ischemic and metabolic abnormalities before they manifest structurally [20,21]. In the present case, persistent hypertension combined with metabolic syndrome, hypothyroidism, testosterone therapy, and post-COVID inflammatory changes created a multifactorial burden on myocardial physiology. Post-COVID myocardial dysregulation— including microvascular dysfunction and inflammatory-mediated mitochondrial impairment—has now been described in several studies [22,23], aligning with the patient's early MCG abnormalities. Serial MCG measurements revealed dynamic shifts between local and global ischemia, a long with fluctuating mitochondrial dysfunction. Such physiologic variability is consistent with emerging evidence that microvascular disease and mitochondrial dysregulation may improve with metabolic optimization, antihypertensive adjustments, and targeted lifestyle modification [24,25]. The patient's history of competitive powerlifting may also have contributed to left ventricular remodeling, as resistance-training–associated concentric hypertrophy is well documented and can interact with pre-existing hypertension to alter myocardial workload [26]. The MCG-derived patterns which reference the AMPK [27] and mTOR [28] pathways, reflect recognized mechanisms underlying metabolic cardiomyopathy, mitochondrial stress, and impaired autophagy [29,30]. These mechanistic insights help contextualize the observed improvements following hormone-dose reduction and dietary change. The partial reversibility of metabolic cardiomyopathy observed here is consistent with recent data showing that microvascular and metabolic cardiac dysfunction can improve when underlying endocrine and metabolic drivers are corrected [31]. Overall, this case highlights the potential complementary role of longitudinal MCG monitoring alongside standard clinical evaluation. While MCG should not replace established diagnostic tools, its ability to provide real-time physiologic insight into the entire cardiovascular system's dynamic mitochondrial network functions may enhance personalized management strategies for patients with multisystem cardiometabolic disease.

## Conclusion

Serial MCG testing provided important physiologic insights into the progression and partial reversibility of myocardial dysfunction in a patient with complex endocrine, metabolic, and inflammatory comorbidities. The dynamic patterns observed across four MCG evaluations underscored the influence of hormonal balance, metabolic optimization, and blood pressure control on myocardial performance. These findings align with recent evidence demonstrating that metabolic and microvascular cardiac abnormalities may improve when underlying contributors are appropriately addressed [31,32]. Integrating longitudinal physiologic monitoring such as MCG (e.g., Premier Heart's MCG™), with standard diagnostic methods may support individualized treatment strategies and improve clinical decision-making in patients with multifactorial cardiometabolic conditions.

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