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Improvement in Glycaemic Control Following External Counter-pulsation (ECP) Therapy in Diet-Controlled Type 2 Diabetes: A Case Report

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ABSTRACT

External Counter-pulsation (ECP) is an FDA-approved, non-invasive therapy primarily indicated for patients with refractory angina and selected cases of heart failure. ECP works by sequentially inflating and deflating pneumatic cuffs on the lower limbs, synchronised with the cardiac cycle, to augment diastolic coronary perfusion and reduce cardiac workload. Beyond its cardiovascular applications, emerging evidence suggests that ECP may exert favourable effects on metabolic parameters, including glycaemic control, through mechanisms such as improved peripheral circulation, enhanced endothelial function, and increased insulin sensitivity. However, data on its efficacy in patients with diet-controlled type 2 diabetes mellitus (T2DM) remain limited. We present the case of a 69-year-old Chinese female with a history of diet-controlled T2DM, who underwent 40 sessions of ECP therapy over a period of several weeks. During the intervention, no changes were made to her diet, physical activity level, or medication regimen. Pre- and post-intervention biochemical assessments demonstrated a reduction in fasting plasma glucose from 7.9 mmol/L (142.2 mg/dL) to 6.2 mmol/L (111.6 mg/dL). Glycated haemoglobin (HbA1C) decreased from 6.9% to 6.1%, indicating improved long-term glycaemic control. Furthermore, her Triglyceride-Glucose (TyG) index, a surrogate marker of insulin resistance, decreased from 9.22 to 9.09 despite a mild increase in serum triglycerides from 1.6 mmol/L (141.7 mg/dL) to 1.8 mmol/L (159.4 mg/dL). In addition to the objective biochemical improvements, the patient reported notable subjective benefits, including enhanced energy levels and improved overall well-being, without experiencing any adverse effects attributable to ECP. This case highlights the potential of ECP as an adjunctive intervention for metabolic optimisation in patients with T2DM, particularly those who are managed with lifestyle modification alone. While the observed improvements in glycaemic markers are encouraging, the single-patient nature of this report limits the applicability of the findings.Further controlled studies with larger sample sizes are warranted to elucidate the underlying mechanisms, determine the magnitude and durability of metabolic benefits, and assess the role of ECP in comprehensive diabetes management strategies. If substantiated, ECP may

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represent a novel, non-pharmacological option for enhancing glycaemic control in select patients with T2DM.

1. Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by a progressive decline in pancreatic β-cell function and increasing insulin resistance in skeletal muscle and adipose tissue [1]. Globally, an estimated 589 million adults are living with diabetes, and this number is projected to rise to 853 million by 2050 [2]. In Malaysia, the 2019 National Health and Morbidity Survey (NHMS) reported a 9.4% prevalence of known diabetes in 2024, affecting approximately 2 million adults [3]. Despite extensive public health initiatives to raise awareness and the availability of oral hypoglycaemic agents, non-compliance with dietary regimens, lifestyle modifications and pharmacological therapy remains a persistent challenge, particularly among older adults and patients with milder disease. This has led to growing interest in adjunctive, non-pharmacological therapies that may help improve glycaemic control in patients not receiving pharmacotherapy.

One such approach worth exploring is External Counter-pulsation (ECP), a non-invasive FDA-approved therapy, that applies sequential pressure to the pelvis and lower limbs via pneumatic cuffs, timed precisely during the diastolic phase of the cardiac cycle as monitored by an electrocardiogram (ECG). This mechanism increases venous return and improves endothelial function, microvascular circulation, and exercise tolerance [4–6]. Braith *et al.* demonstrated significant improvement in flow-mediated dilation in patients with chronic angina following ECP, while Masuda *et al.* proposed that reduced peripheral vascular resistance could result from increased vascular shear stress during ECP, which may upregulate endothelial nitric oxide synthase expression, thereby improving peripheral circulation in patients with chronic stable angina pectoris [5,6]. These improvements in endothelial function and peripheral perfusion may enhance insulin-mediated glucose uptake in skeletal muscle, suggesting that the vascular benefits observed in these studies could also contribute to better glycaemic control.

In addition to vascular effects, emerging evidence suggests that ECP may exert systemic metabolic benefits. Studies have shown improvements in insulin sensitivity and metabolic markers following ECP, particularly in individuals with metabolic syndrome or high cardiovascular risk. Sardina *et al.* reported reductions in HbA1C of 11.5% at 48 hours, 19.6% at two weeks, and 14.3% at three months post-treatment, achieving at least an 11 mmol/mol (IFCC units) reduction, these changes associated with an estimated 40% decrease in diabetes-related complications [7]. However, further research is needed to elucidate the effects of ECP on glycaemic control using cost-effective and accessible surrogate markers such as the triglyceride–glucose (TyG) index.

The TyG index is a validated surrogate marker strongly associated with metabolic risk and is a reliable metric for assessing insulin resistance [8–10]. Unlike more complex and costly measures such as the hyperinsulinaemic–euglycaemic clamp (the gold standard) or the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), which requires fasting insulin measurement, the TyG index is easily calculated from fasting triglyceride and glucose levels, making it practical for both clinical and research settings. Several studies have shown that the TyG index may have superior predictive ability for insulin resistance and future diabetes risk compared with HOMA-IR, particularly in large cohorts and resource-limited settings where insulin assays are not readily available [9–12]. However, evidence on the impact of ECP on the TyG index particularly in patients with diet-controlled T2DM still remains limited.

2. Case Report

This case highlights the metabolic impact of ECP in a patient with type 2 diabetes mellitus managed without pharmacological intervention, demonstrating measurable improvements in fasting blood glucose, HbA1c, and the TyG index after a full treatment course.

A 69-year-old Chinese female with a five-year history of type 2 diabetes mellitus (T2DM), managed without pharmacologic treatment at the time of initial encounter, presented to a wellness centre for ECP therapy as part of an alternative cardiovascular risk reduction program. She had no history of ischemic heart disease, myocardial infarction, or heart failure, and was not taking any anti-diabetic, lipid-lowering, or antihypertensive medications nor any traditional medications. Her vital signs including blood pressure of 128/84 mmHg with mean arterial pressure of 99 mmHg and pulse rate of 78 beats per minute along with other physical examination were all normal. Her random capillary blood glucose during first presentation was 8.3mmol/L. Her anthropometry was within normal limits with reported Body Mass Index (BMI) of 22kg/m^2. Her other blood parameters including liver and renal function test along with uric acid level were within normal range.

This case explores the potential metabolic benefits of ECP therapy in a patient with type 2 diabetes mellitus (T2DM) managed without any intended pharmacological agents. Following the completion of 40 ECP sessions, the patient's fasting blood glucose (FBG) and haemoglobin A1c (HbA1c) levels decreased, accompanied by a modest reduction in the TyG index, a validated and novel surrogate marker for insulin resistance. These improvements occurred despite a slight increase in triglyceride levels, suggesting that the observed glycaemic benefits may be attributable to enhanced peripheral glucose uptake and improved insulin sensitivity resulting from vascular and microcirculatory adaptations induced by ECP.

3. Results

The patient's diabetes control had been monitored through periodic laboratory testing and dietary adjustments. She underwent a complete course of 40 ECP sessions delivered according to standard treatment protocols. Importantly, no changes were made to her dietary habits, physical activity level, or medication use during the treatment period. The therapy was well tolerated, with no adverse events reported. In addition to the laboratory findings, the patient described subjective improvements in energy levels, reduced fatigue, and greater ease in performing daily activities.

Baseline and post-treatment laboratory values are summarized in **Table 1**. At baseline, fasting blood glucose (FBG) was elevated at 7.9 mmol/L (142.2 mg/dL), HbA1c was 6.9% (52 mmol/mol), and triglycerides measured 1.6 mmol/L (141.7 mg/dL), yielding a TyG index of 9.22. Following completion of ECP, FBG decreased to 6.2 mmol/L (111.6 mg/dL), representing a 21.5% reduction. HbA1c declined to 6.1% (43 mmol/mol), corresponding to an 11.6% relative improvement. The TyG index improved modestly to 9.09 despite a small increase in triglycerides to 1.8 mmol/L (159.4 mg/dL).

Baseline and post-treatment laboratory results are shown in **Table 1**. Measurements included fasting blood glucose (FBG), hemoglobin A1c (HbA1c), triglycerides, and the TyG index. The TyG index was calculated using the formula:

$$TyG\ index = ln\left(\frac{Triglycerides\ (mg/dL) \times Fasting\ Glucose\ (mg/dL)}{2}\right) \quad (1)$$

Table 1
Laboratory results before and after completion of 40 sessions of ECP therapy

Parameter	Baseline	Post-ECP
Fasting Blood Glucose (mmol/L)	7.9	6.2
Fasting Blood Glucose (mg/dL)	142.2	111.6
HbA1c (%) [DCCT/NGSP]	6.9	6.1
HbA1c (mmol/mol) [IFCC]	52	43
Triglycerides (mmol/L)	1.6	1.8
Triglycerides (mg/dL)	141.7	159.4
Triglyceride-Glucose Index (TyG)	9.22	9.09

(Abbreviations; DCCT: Diabetes Control and Complications Trial, NGSP: National Glycohemoglobin Standardization Programme, IFCC: International Federation of Clinical Chemistry)

(1)

The overall pattern suggested improved glycaemic control and reduced insulin resistance following ECP. These changes occurred in the absence of pharmacological intervention, lending further support to ECP's potential role in modulating metabolic outcomes. Although the possibility of unmeasured confounders cannot be fully excluded, the consistency of biochemical improvements across multiple indices strengthens the plausibility of a causal link between ECP therapy and enhanced glycaemic control.

4. Discussion

Recent evidence suggests that ECP therapy may have beneficial effects beyond angina relief and perfusion enhancement, particularly in metabolic regulation. This case report highlights the potential of ECP therapy to improve glycaemic control and reduce insulin resistance in patients with type 2 diabetes mellitus (T2DM) who are not receiving pharmacological treatment. The observed reductions in fasting blood glucose, HbA1c, and TyG index indicate that ECP may exert systemic metabolic benefits beyond its established role in cardiovascular disease management which may be attributed to several interconnected vascular and cellular mechanism by involving improved endothelial function, enhanced microvascular perfusion, and increased skeletal muscle glucose uptake secondary to haemodynamic and vascular adaptations [7]. Such physiological changes may collectively contribute to better insulin sensitivity, aligning with prior evidence from studies on vascular compliance and metabolic parameters following ECP therapy.

While the direct cellular mechanisms by which ECP modulates glucose metabolism remain not fully understood, accumulating evidence supports a vascular-metabolic pathway in which improvements in endothelial function, microcirculation and inflammation collectively improves insulin sensitivity. ECP generates a supraphysiologic hemodynamic state caused by increased diastolic augmentation and shear stress on the endothelium. This mechanical stimulus enhances endothelial nitric oxide (NO) release, which not only improves vasodilation but also increases glucose uptake in skeletal muscle and adipose tissue by facilitating insulin signalling pathways. These changes may facilitate greater glucose delivery and uptake at the cellular level, thereby improving insulin sensitivity [5-6, 13,14]. By reducing vascular stiffness and improving arterial compliance, ECP restores microvascular perfusion, thereby enhancing tissue-level insulin delivery and glucose utilization. This effect is complemented by the therapy's role in ameliorating endothelial dysfunction, a major contributor to insulin resistance.

Beyond these hemodynamic effects, ECP has also been associated with the mobilization of circulating progenitor cells such as hematopoietic progenitor cells (HPCs) and endothelial progenitor cells (EPCs). Circulating HPCs and EPCs , identified by $CD34^+$ and $CD133^+$ markers, increase following ECP [15]. Interestingly, Kiernan *et al.* found that ECP preferentially mobilizes HPC rather than EPC, whereas Tartaglia *et al*. reported significant increases in $CD34^+/CD133^+$ EPCs in refractory angina patients [15,16]. These variability suggests that ECP may preferentially mobilize different progenitor cells depending on patient's phenotype, but both HPC and EPC mobilization point towards a common mechanism in which ECP contributes to vascular repair, angiogenesis and microcirculatory improvement, thereby augmenting metabolic homeostasis. The vascular adaptations reported in earlier ECP studies help contextualise the metabolic improvements seen in our patient, suggesting a plausible physiological connection between ECP therapy and improvements in insulin sensitivity.

In addition to these hemodynamic and progenitor cell responses, ECP appears to influence angiogenic signalling pathways that sustain vascular repair and metabolic health. Shear stress generated during treatment promotes the release of vascular endothelial growth factor-A (VEGF-A) from endothelial and skeletal muscle cells, with downstream activation of VEGF receptor-2 (VEGFR-2) on circulating progenitor cells [17]. This interaction is critical for progenitor cell survival, migration, and differentiation into mature endothelial lineages. The expansion of the microvascular network through angiogenesis increases capillary surface area for nutrient and hormone exchange, thereby improving the delivery of insulin and glucose to peripheral tissues. Furthermore, ECP has been shown to reduce inflammatory and endothelial activation marker such as hsCRP, TNF-α, MCP-1 and sVCAM thereby improving the chronic low-grade inflammation known to impair insulin receptor signalling [5, 18]. These combined effects of vascular regeneration, angiogenesis, and reduced inflammation provide a coherent explanation for how ECP may indirectly improve insulin sensitivity, even in patients not receiving pharmacological therapy.

Our findings are consistent with those of Sardina *et al.*, who reported significant HbA1c improvement sustained for three months following ECP in patients with non–insulin-dependent T2DM [7]. Their results are particularly relevant to our case because, like our patient, participants maintained glycaemic improvements without concurrent pharmacological adjustment. This reinforces the potential for ECP to independently enhance insulin sensitivity, while acknowledging that adjuncts such as dietary modification and physical activity may further augment its efficacy. Likewise, Casey *et al.* and Nicholas *et al.* have documented improved arterial compliance and reduced vascular resistance after ECP which is related to reduced insulin resistance [19, 20]. Such haemodynamic changes may facilitate more efficient nutrient and hormone delivery to peripheral tissues, thereby increasing glucose uptake and utilisation, which could explain the improvement in glycaemic indices observed in our patient.

Although existing research has explored the cardiovascular and metabolic benefits of ECP, including its potential applications in patients with diabetes, no direct studies have examined its effect on surrogate markers of insulin resistance such as the TyG index. The use of the TyG index in this case provides valuable insight into ECP's metabolic effects, particularly given the paucity of published reports evaluating this marker in relation to ECP therapy. As a recognised surrogate marker for insulin sensitivity and a predictor of cardiometabolic risk, improvement in the TyG index alongside reductions in FBG and HbA1c suggests potential benefit of ECP on insulin resistance. Several studies have noted that the TyG index performs as well as, or better than, HOMA-IR in detecting insulin resistance. Notably, Vasques *et al.*, using the hyperglycaemic clamp as the gold standard, demonstrated the TyG index's superior predictive performance in a Brazilian population [21]. To our knowledge, reports directly examining ECP's impact on the TyG index remain scarce, making this case a meaningful contribution to the literature.

Given that the TyG index is increasingly recognized as a reliable and practical tool for assessing metabolic risk, this represents a novel and innovative area for research expansion [9, 10]. A prospective interventional study assessing changes in the TyG index before and after ECP could provide critical evidence for its role as a metabolic-modifying therapy in addition to its established cardiovascular benefits, potentially positioning ECP as a novel adjunctive therapy for patients with type 2 diabetes or metabolic syndrome. Figure 1 depicts the evidence-based role of ECP in ameliorating insulin resistance.

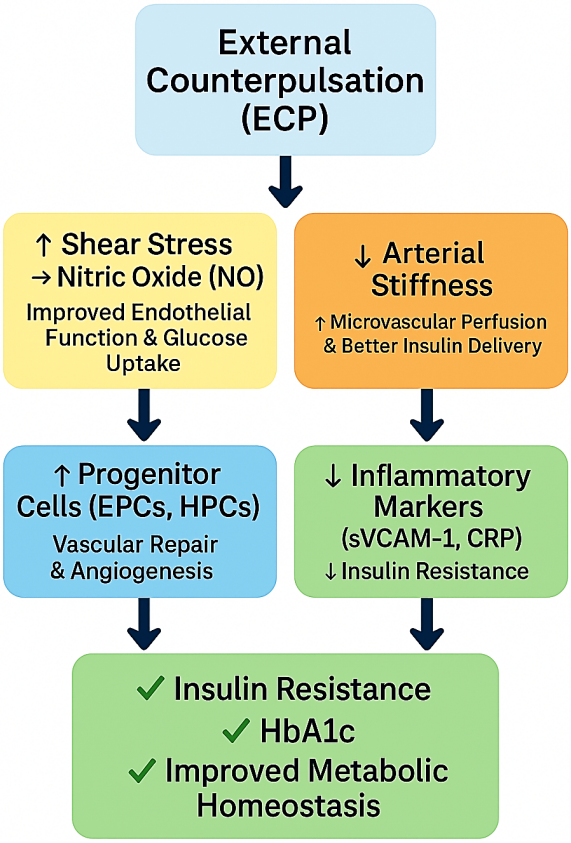

Fig. 1. ECP mechanism improving Insulin Resistance

(Abbreviations; EPC: endothelial progenitor cells, HPC: hematopoietic progenitor cells, sVCAM-1: soluble vascular cell adhesion molecule-1, CRP: C-reactive protein)

5. Conclusion

While the present findings are encouraging, they are derived from a single-patient observation, limiting their applicability to broader population. Larger, well-designed prospective pilot studies are warranted to confirm the reproducibility of these effects, delineate the mechanistic pathways, and determine the sustainability of glycaemic improvements over time. This case has several limitations, including its single-patient design, the absence of direct serum insulin measurements, and the extended period over which ECP sessions were completed.

Future research should also evaluate the role of ECP in broader metabolic contexts which is currently underexplored. Given the intricate interplay between metabolic dysfunction and cardiovascular risks, evaluating ECP in prediabetes, metabolic syndrome, insulin resistance–related cardiovascular risk, as well as in metabolic dysfunction-associated fatty liver disease (MAFLD) including metabolic associated steatohepatitis (MASH) could uncover novel therapeutic and

adjunctive treatment modalities. To establish ECP as credible adjunct in metabolic disease management, comparative studies are essential. These should assess its efficacy with established interventions such as pharmacotherapy (e.g. Glucagon-like peptide 1 (GLP-1) receptor agonists, sodium-glucose cotransporter 2 (SGLT2) inhibitors), structured exercise regimens, and dietary with necessary lifestyle modification tailored to individual metabolic profiles. Such studies would help define whether ECP can serve as a viable adjunctive therapy for metabolic disease management particularly in long-term outcomes, patient adherence, cost-effectiveness and quality of life metrics alongside its already established cardiovascular benefits. Ultimately, integrating ECP into a multidisciplinary treatment framework could offer a non-invasive, low-risk option for patients who are contraindicated for certain medications or struggle with lifestyle and pharmacological adherence.

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