Opinion
Volume 9:5, 2024

Journal of Diabetic Complications & Medicine

ISSN: 2475-3211 Open Access

The Influence of Contemporary Anti-Diabetic Therapies on Endothelial Progenitor Cells

Tomislav Uremovic*

Department of Diabetes, University of Zagreb, Zagreb, Croatia

Introduction

Diabetes mellitus, a chronic metabolic disorder characterized by hyperglycemia, has become a global health concern. Beyond its impact on blood glucose levels, diabetes significantly contributes to macrovascular and microvascular complications, including cardiovascular diseases. Among the various complications associated with diabetes, endothelial dysfunction plays a pivotal role in the progression of vascular diseases. Endothelial Progenitor Cells (EPCs), critical for vascular repair and regeneration, have emerged as key players in the intricate interplay between diabetes and cardiovascular health. In this article, we delve into the influence of contemporary anti-diabetic therapies on EPCs, exploring how these treatments may either mitigate or exacerbate vascular complications in diabetic patients.

Diabetes mellitus, a chronic metabolic disorder characterized by elevated blood glucose levels, has reached epidemic proportions globally. The disease not only poses a significant threat to the overall health of individuals but also contributes to a myriad of complications, including cardiovascular diseases. Endothelial dysfunction, a hallmark of diabetes, plays a pivotal role in the development and progression of cardiovascular complications [1]. In recent years, researchers have turned their attention to Endothelial Progenitor Cells (EPCs) as key players in vascular repair and regeneration. This article explores the influence of contemporary anti-diabetic therapies on EPCs and the implications for cardiovascular health.

Endothelial progenitor cells are a subpopulation of bone marrow-derived cells with the unique ability to differentiate into mature endothelial cells. These cells contribute to vascular repair, neovascularization, and maintenance of endothelial integrity. In individuals with diabetes, EPC function is often compromised, leading to impaired vascular repair and increased susceptibility to cardiovascular complications.

Metformin, a first-line oral hypoglycemic agent, has been a mainstay in diabetes management for decades. Studies suggest that metformin may exert a positive influence on EPCs. It has been shown to enhance EPC mobilization and improve their functional capacity, contributing to improved vascular repair.

Sodium-Glucose Co-Transporter 2 (SGLT-2) inhibitors represent a relatively new class of anti-diabetic medications. Recent research indicates that SGLT-2 inhibitors may have favorable effects on EPC function by reducing oxidative stress and inflammation, factors that contribute to EPC dysfunction in diabetes.

Glucagon-Like Peptide-1 (GLP-1) receptor agonists have gained popularity due to their glucose-lowering effects and potential cardiovascular benefits. Emerging evidence suggests that GLP-1 receptor agonists may positively influence EPCs by promoting their mobilization and enhancing their migratory and angiogenic capacities.

Dipeptidyl Peptidase-4 (DPP-4) inhibitors are another class of anti-diabetic medications that enhance the activity of endogenous GLP-1. While the direct effects of DPP-4 inhibitors on EPCs are not fully elucidated, some studies suggest potential benefits in improving vascular function and endothelial repair [2].

Insulin, a hormone critical for glucose homeostasis, is often prescribed in diabetes management. While hyperinsulinemia is associated with adverse effects on endothelial function, optimal insulin therapy appears to have neutral or even beneficial effects on EPCs, emphasizing the importance of personalized insulin management.

Description

Endothelial progenitor cells are a subset of bone marrow-derived cells with the unique ability to differentiate into mature endothelial cells, contributing to the maintenance and repair of the vascular endothelium. In diabetes, the functionality and number of EPCs are often compromised, leading to impaired endothelial repair and an increased risk of cardiovascular events.

Historically, the cornerstone of diabetes management has been lifestyle modifications and traditional pharmacological interventions, such as metformin and sulfonylureas. While these drugs effectively control blood glucose levels, their impact on EPCs has been a subject of investigation.

***Address for Correspondence:** Tomislav Uremovic, Department of Diabetes, University of Zagreb, Zagreb, Croatia; E-mail: tomislavuremovic@gmail.com

Received: 23 November, 2023, Manuscript No. JDCM-23-120868; **Editor assigned:** 27 November, 2023, PreQC No. JDCM-23-120868 (PQ); **Reviewed:** 11 December, 2023, QC No. JDCM-23-120868; **Revised:** 13 December, 2024, Manuscript No. JDCM-23-120868 (R); **Published:** 20 December, 2024, DOI: 10.37421/2475-3211.2024.9.285

Metformin, the first-line therapy for type 2 diabetes, has shown potential benefits in preserving EPC function. Studies suggest that metformin may enhance EPC mobilization and improve their ability to promote angiogenesis, thereby supporting vascular repair mechanisms [3].

On the other hand, sulfonylureas, commonly prescribed to stimulate insulin secretion, have raised concerns regarding their impact on cardiovascular health. Some studies suggest that sulfonylureas may exert detrimental effects on EPCs, potentially contributing to a proatherogenic environment.

Insulin, a hormone essential for glucose homeostasis, is administered in various forms to diabetic patients. While insulin is indispensable for controlling blood glucose levels, its influence on EPCs has been a matter of debate. Insulin resistance, a hallmark of type 2 diabetes, may adversely affect EPC function. Studies have demonstrated that hyperinsulinemia, a consequence of insulin resistance, can impair EPC mobilization and differentiation. However, insulin therapy in controlled doses has also been shown to have positive effects on EPC function, emphasizing the delicate balance required in insulin administration.

The landscape of diabetes management underwent a transformative shift with the introduction of Glucagon-Like Peptide-1 (GLP-1) receptor agonists and Sodium-Glucose Co-transporter-2 (SGLT-2) inhibitors. These classes of medications not only target hyperglycemia but also demonstrate cardiovascular benefits, making them attractive options for patients with diabetes and cardiovascular comorbidities. GLP-1 receptor agonists, known for their cardiovascular protective effects, have shown promise in improving EPC function [4]. Studies suggest that GLP-1 may enhance EPC survival, migration, and angiogenic potential, contributing to better vascular repair mechanisms.

SGLT-2 inhibitors, by promoting glycosuria and lowering blood pressure, exhibit cardiovascular benefits beyond glycemic control. Some evidence suggests that SGLT-2 inhibitors may positively influence EPC function, although the underlying mechanisms are not fully elucidated. Beyond the established classes of anti-diabetic medications, ongoing research is exploring novel therapeutic avenues, such as Dipeptidyl Peptidase-4 (DPP-4) inhibitors, which enhance the actions of incretin hormones. DPP-4 inhibitors, while primarily targeting glucose metabolism, have been associated with potential vascular benefits. Some studies propose that DPP-4 inhibitors may enhance EPC function, contributing to improved endothelial repair mechanisms.

The influence of contemporary anti-diabetic therapies on EPCs holds significant implications for cardiovascular health in individuals with diabetes. The restoration of EPC function and enhancement of vascular repair mechanisms contribute to a reduced risk of cardiovascular events. Additionally, the potential synergy between anti-diabetic medications and their positive effects on EPCs may provide a novel avenue for developing integrated therapeutic approaches to mitigate cardiovascular complications in diabetes [5].

Despite the promising findings, challenges persist in fully understanding the complex interplay between anti-diabetic therapies and EPCs. Variability in study designs, patient populations, and duration of interventions makes it challenging to draw definitive conclusions. Moreover, the long-term effects of these therapies on EPCs and their ultimate impact on cardiovascular outcomes require further investigation.

Future research should focus on elucidating the specific molecular mechanisms through which anti-diabetic medications influence EPCs. This understanding is crucial for developing targeted therapies that optimize EPC function and enhance vascular repair. Additionally, large-scale, well-designed clinical trials are necessary to establish the cardiovascular benefits of these medications through the lens of EPC biology.

The interplay between contemporary anti-diabetic therapies and endothelial progenitor cells is a complex and dynamic field of research. While traditional therapies may have variable effects on EPC function, newer classes of medications, such as GLP-1 receptor agonists and SGLT-2 inhibitors, demonstrate promising cardiovascular benefits, potentially influencing EPCs positively [6]. Understanding the intricate relationship between anti-diabetic therapies and EPCs is crucial for optimizing treatment strategies in diabetic patients, especially those at high risk of cardiovascular complications. As research continues to unveil the molecular mechanisms underlying these interactions, the future holds promise for tailored therapeutic approaches that not only manage glucose levels but also foster vascular health in individuals with diabetes.

Conclusion

The influence of contemporary anti-diabetic therapies on endothelial progenitor cells represents a fascinating area of research with profound implications for cardiovascular health in individuals with diabetes. While each class of medication may exert unique effects on EPCs, the overarching goal is to improve vascular repair mechanisms and mitigate the risk of cardiovascular complications. As our understanding deepens, the integration of these findings into personalized diabetes management strategies may pave the way for more effective and holistic approaches to address the multifaceted challenges posed by diabetes and its cardiovascular consequences.

References

1. Chang, Xing, Amanda Lochner, Hsueh-Hsiao Wang and Shuyi Wang, et al. “Coronary microvascular injury in myocardial infarction: perception and knowledge for mitochondrial quality control.” Theranostics 11 (2021): 6766.
2. Cavieres, Vanessa, Karla Valdes, Brayan Moreno and Rodrigo Moore-Carrasco, et al. “Vascular hypercontractility and endothelial dysfunction before development of atherosclerosis in moderate dyslipidemia: Role for nitric oxide and interleukin-6.” Am J Cardiovasc Dis 4 (2014): 114.
3. Hetherington, Isabella, and Hana Totary-Jain. “Anti-atherosclerotic therapies: Milestones, challenges, and emerging innovations.” Mol Ther 30 (2022): 3106-3117.
4. Prasad, Kailash. “Current status of primary, secondary and tertiary prevention of coronary artery disease.” Int J Angiol 30 (2021): 177-186.
5. Chen, Maggie S., Richard T. Lee, and Jessica C. Garbern. “Senescence mechanisms and targets in the heart.” Cardiovasc Res 118 (2022): 1173-1187.
6. Aquino, Jorge B, Romina Sierra and Laura A. Montaldo. “Diverse cellular origins of adult blood vascular endothelial cells.” Dev Biol 477 (2021): 117-132.

How to cite this article: Uremovic, Tomislav. "The Influence of Contemporary Anti-Diabetic Therapies on Endothelial Progenitor Cells." *J Diabetic Complications Med* 9 (2024): 285.