

Review Article

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Plant-Based Diet Mitigates Restenosis Post-DCB Angioplasty: Mechanisms and Clinical Evidence

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Abstract

Despite groundbreaking advancements in coronary interventions through Drug-Coated Balloon (DCB) angioplasty, restenosis challenges long-term success in cardiovascular treatment, driven significantly by systemic inflammation, oxidative stress, endothelial dysfunction, and metabolic dysregulation. This provocative review uncovers how traditional reliance on pharmacological strategies inadequately addresses these systemic factors. In stark contrast, adopting a Plant-Based Diet (PBD) emerges as a transformative approach, directly confronting the root biological mechanisms fueling restenosis. A PBD profoundly reduces inflammatory markers, enhances endothelial nitric oxide (NO) bioavailability, optimizes gut microbiome composition, and strengthens mitochondrial function, collectively resulting in superior clinical outcomes. Compelling evidence from Bethsaida Hospital, Indonesia, led by the visionary cardiologist Prof. Dasaad Mulijono, demonstrates unprecedented reductions in restenosis rates (<2%) among nearly 2,000 patients. This review challenges cardiologists worldwide to reconsider therapeutic strategies and incorporate dietary interventions into routine clinical practice, thereby fundamentally redefining success in coronary interventions and cardiovascular health management.

Keywords: Plant-based diet; restenosis; drug-coated balloon; coronary intervention; vascular healing; systemic inflammation; nitric oxide; gut microbiome; oxidative stress; endothelial function; bethsaida hospital; prof. dasaad mulijono

Introduction

DCB angioplasty has emerged as a significant advancement in interventional cardiology, effectively managing coronary artery disease (CAD) without necessitating permanent implants, such as stents [1-10]. Despite this progress, restenosis - characterized by the recurrent narrowing of arteries after intervention - continues to present a clinical challenge, predominantly driven by vascular injury-induced inflammation, oxidative stress, neointimal hyperplasia, and underlying systemic conditions such as dyslipidemia, insulin resistance, and chronic inflammation [11-20]. Traditional therapeutic strategies have primarily focused on pharmacological approaches aimed at local control of these processes. However,

emerging evidence suggests that dietary factors have a significant impact on vascular healing and systemic inflammatory responses. Animal-based diets, notably rich in saturated fats, cholesterol, and pro-inflammatory components, have been consistently linked to an increased risk of restenosis. Conversely, adopting a PBD offers complementary biological benefits, including reduced systemic inflammation, improved endothelial function, optimized gut microbiota, and enhanced NO bioavailability. This review critically evaluates the mechanistic insights and clinical evidence supporting the efficacy of a PBD in mitigating restenosis following DCB angioplasty, supported by substantial real-world outcomes from

Bethsaida Hospital in Indonesia, under the innovative leadership of Prof. Dasaad Mulijono.

Mechanisms by Which Animal-Based Diets Promote Restenosis

Animal-derived dietary components contribute significantly to the pathophysiology underlying atherosclerosis and restenosis:

- a) Saturated Fats and Cholesterol: High intake elevates LDL cholesterol, promoting endothelial dysfunction, inflammation, and plaque formation [21,22].
- b) Oxidative Stress: Animal products lack antioxidants, which facilitates the activity of reactive oxygen species (ROS), endothelial injury, and the progression of atherosclerosis [23-25].
- c) NO Deficiency: Reduced dietary nitrate and increased oxidative stress impair NO synthesis and bioavailability, exacerbating endothelial dysfunction [26-28].
- d) Gut Dysbiosis: Animal-based diets deficient in fibre disrupt gut microbiota, elevating systemic inflammation and enhancing vascular injury [29-32].
- e) Trimethylamine-N-oxide (TMAO): Produced from dietary choline and carnitine, TMAO promotes platelet hyperreactivity and endothelial dysfunction [33-37].
- f) Advanced Glycation End-products (AGEs): Cooking animal products generates AGEs, which activate inflammatory pathways and enhance the risk of restenosis [38-44].
- g) Systemic Inflammation and Insulin Resistance: Animal foods rich in saturated fats and inflammatory amino acids promote systemic inflammation, insulin resistance, and endothelial damage [45-59].
- h) Mitochondrial Dysfunction: Animal-based diets impair mitochondrial function, reducing cellular repair capacity and increasing oxidative damage [60-62].
- i) Telomere Shortening: Low antioxidant intake from animal-based diets accelerates telomere attrition, promoting vascular aging [63-67].
- j) Systemic Acidosis: High-protein animal diets induce chronic acidosis, contributing to endothelial dysfunction and calcification [68-70].
- k) Animals contain IGF-1, heme iron, free radicals, suPAR, Neu5GC, POPs, and preservatives: These have been shown to cause inflammation and vascular injury, which contribute to the development of restenosis post-DCB intervention [57-59,71-98].
- l) Animal-derived foods have been shown to induce obesity, hypertension, hyperglycaemia, and metabolic syndrome, which is known to cause the development of restenosis [99-102].

Mechanisms by Which a PBD Mitigates Restenosis

By transitioning from an animal-based diet to a PBD, individuals automatically cease consuming foods that contribute to the pathological mechanisms outlined above, thus directly reducing the risk of restenosis. In addition, a PBD effectively addresses and reverses these mechanisms through multiple complementary pathways [103-109]:

- a) Reduction of Systemic Inflammation: A PBD lowers inflammatory biomarkers (CRP, interleukins), primarily by eliminating pro-inflammatory saturated fats, cholesterol, and TMAO precursors.
- b) Enhanced NO Bioavailability: Rich in dietary nitrates from leafy greens and vegetables, a PBD boosts NO production, critical for endothelial function, preventing platelet aggregation, and smooth muscle cell proliferation.
- c) Antioxidant Protection: Plant foods supply ample antioxidants (vitamin C, flavonoids, carotenoids) that neutralize ROS, protecting endothelial cells from angioplasty-induced damage.
- d) Gut Microbiome Optimization: High dietary fibre and polyphenols promote the growth of SCFA-producing beneficial microbiota, thereby enhancing gut barrier integrity, reducing systemic inflammation, and limiting TMAO production.
- e) Mitochondrial Support: The polyphenols and nutrient precursors in plant foods, such as coenzyme Q10, sustain mitochondrial function, reduce oxidative stress, and enhance cellular repair.
- f) Telomere Preservation and Epigenetic Stability: Nutrient-rich plant foods containing antioxidants and anti-inflammatory compounds preserve telomere length, modulate gene expression favorably, and slow vascular aging.
- g) Hypersensitivity and Allergic Reaction Prevention: Flavonoids in PBDs minimize hypersensitivity reactions potentially triggered by stent materials (in the case of in-stent restenosis) or associated medications, thus mitigating localized inflammation.

Clinical Evidence from Bethsaida Hospital, Indonesia

Bethsaida Hospital offers robust real-world clinical data that underscores the effectiveness of PBD in post-angioplasty care. Among nearly 2,000 patients undergoing DCB angioplasty, adherence to a plant-based dietary regimen corresponded to remarkably reduced restenosis rates (<2%) compared to standard restenosis rates of 10-20% reported globally. Additionally, there were no reported cases of early stent thrombosis among patients who were compliant with a PBD, emphasizing the enhanced systemic vascular healing.

Conclusion

Effectively mitigating restenosis following DCB angioplasty requires a comprehensive therapeutic strategy that addresses underlying systemic pathologies, rather than focusing solely on local vessel interventions. A PBD uniquely and powerfully targets primary drivers of restenosis, including systemic inflammation, oxidative stress, endothelial dysfunction, mitochondrial impairment, and gut dysbiosis. Clinical evidence, notably from Bethsaida Hospital, demonstrates significantly reduced restenosis rates (<2%) in nearly 2,000 patients adhering to a PBD, compared to global benchmarks (10-20%). These compelling outcomes underscore the critical role of dietary interventions in cardiovascular healing and emphasize the need for broader clinical integration of lifestyle medicine within cardiovascular care protocols. By embracing dietary strategies that synergistically promote systemic and vascular health, interventional cardiology can achieve enhanced patient outcomes, reduced complications, and redefine long-term success in the management of CAD.

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Conflict of Interest

The authors declare no conflict of interest.

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