WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 14, Issue 13, 1721-1725. Review Article ISSN 2277–7105

REVIEW ON, THE DRUG UTILIZATION PRACTICES OF PATIENTS WITH CORONARY ARTERY DISEASE UNDERGOING STENTING'

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Article Received on
18 May 2025,

Revised on 08 June 2025,
Accepted on 29 June 2025

DOI: 10.20959/wjpr202513-37469

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ABSTRACT

Percutaneous coronary intervention (PCI) is the primary method for coronary artery revascularization, historically advancing from balloon angioplasty to the introduction of coronary stents, which initially addressed issues such as vascular dissections and arterial recoil. Despite these improvements, the challenge of neointimal accumulation persisted, leading to in-stent restenosis (ISR) in a significant proportion of cases. The evolution of drug-eluting stents (DESs) aimed to mitigate ISR by delivering anti-proliferative agents directly to the arterial injury site. While DESs have enhanced outcomes post-PCI, concerns remain regarding delayed re-endothelialization and stent thrombosis. This review explores the pathophysiology of ISR and stent thrombosis, summarizing clinical evidence on first- and second-generation DESs. Recent advancements in understanding ISR pathogenesis are discussed, alongside emerging therapeutic strategies aimed at improving clinical efficacy and patient.

KEYWORDS: Percutaneous Coronary Intervention (PCI), Drug-Eluting Stents (DES), Stent Thrombosis, Coronary Artery Disease (CAD), Neointimal Hyperplasia, Bare-Metal Stents (BMS), Re-endothelialization, Dual Antiplatelet Therapy (DAPT), Vascular Remodeling, Smooth Muscle Cell Proliferation, Target Lesion Revascularization (TLR), Very Late Stent Thrombosis (VLST).

INTRODUCTION

Coronary stents are expandable tubular metallic devices used to treat coronary arteries that are narrowed due to atherosclerotic disease. This revascularization procedure is referred to as

percutaneous coronary intervention (PCI), or coronary angioplasty with stent placement (Baydoun et al., 2019). Before the introduction of stents, balloon angioplasty was the standard method for coronary revascularization. In this technique, a balloon-tipped catheter was introduced percutaneously through an arterial entry in the extremity and advanced into the coronary arteries. Once positioned, the balloon was inflated to compress the atherosclerotic plaque against the arterial wall, restoring myocardial perfusion. However, this method had significant limitations, including acute vessel closure due to arterial recoil, coronary artery dissection, acute thrombosis, and restenosis caused by neointimal hyperplasia.[7]

With the advent of coronary stents, particularly bare-metal stents (BMS), many of these complications were addressed. The metallic meshwork of the stent eliminated arterial recoil and dissection by providing structural support to the vessel.[6]

TYPES OF STENTS

1. **BES (Biolimus-Eluting Stent):** Coated with biolimus, this stent type prevents restenosis by inhibiting cell proliferation within the arterial wall. It is part of the newer generation of drug-eluting stents (DES), showing better outcomes in selected populations, including women.[6]
2. **BMS (Bare Metal Stent):** Made from stainless steel or cobalt-chromium, BMS are uncoated stents that provide mechanical support. While they reduced acute arterial closures compared to balloon angioplasty, they were associated with higher rates of in-stent restenosis (ISR) due to neointimal proliferation.[7]
3. **BVS (Bioresorbable Vascular Scaffold):** These stents gradually dissolve over time, potentially restoring natural vasomotion and reducing long-term complications like very late stent thrombosis (VLST).[8]
4. **DAPT (Dual Antiplatelet Therapy):** Typically comprising aspirin and a P2Y12 inhibitor, DAPT is critical in preventing thrombus formation after stent implantation and reduces the risk of stent thrombosis.[5]
5. **DES (Drug-Eluting Stent):** These stents release antiproliferative drugs such as sirolimus, everolimus, zotarolimus, or biolimus, significantly reducing restenosis rates (Kar, 2019).

6. **EES (Everolimus-Eluting Stent):** A type of DES, EES effectively inhibits cell growth within the arterial lining, making it especially beneficial in acute myocardial infarction cases in women.[6]
7. **ISR (In-Stent Restenosis):** A condition where the artery narrows again due to tissue proliferation at the stent site. ISR is a major limitation of BMS and early-generation DES.[2]

The major mechanism of isr after stenting

Neointimal hyperplasia after PCI is best described by the "response-to-injury" model proposed by Ross and Glomset. Mechanical disruption of the endothelium initiates a cascade of inflammatory responses. In the first 3 days (thrombotic phase), platelet activation leads to microthrombus formation. This is followed by recruitment of inflammatory cells such as macrophages and lymphocytes from the vascular lumen and vasa vasorum between days 3–8 (recruitment phase).[2]

These inflammatory cells release cytokines and growth factors that stimulate smooth muscle cells (SMCs) in the intima-media to proliferate and migrate into the stent, secreting extracellular matrix proteins like proteoglycans (proliferative phase). The hygroscopic and stiff nature of this matrix contributes to restenosis and limits balloon expansion effectiveness in ISR treatment.[5]

Re-endothelialization after stent placement plays a variable role in neointimal growth. Animal studies show that injury from balloon-expandable stents is more severe and persistent than balloon angioplasty alone, resulting in more tissue proliferation. Nevertheless, the gain in lumen diameter with stents outweighs these effects, leading to better outcomes.[4]

Targeted, local drug delivery via stents that release anti-proliferative agents is now considered the most effective strategy for reducing ISR without causing systemic toxicity.[6]

8. **MI (Myocardial Infarction):** Commonly known as a heart attack, MI results from prolonged ischemia due to arterial blockage. MI is more frequently observed in younger patients and women, where pathophysiological mechanisms might differ (Mehilli & Presbitero, 2020.[4]
9. **PCI (Percutaneous Coronary Intervention):** A non-surgical approach to open stenotic coronary arteries, PCI often includes balloon dilation followed by stent deployment.[8]

10. PES (Paclitaxel-Eluting Stent): A DES that releases paclitaxel, which inhibits excessive tissue proliferation. It was among the first-generation DES used in clinical practice.[7]

11. SES (Sirolimus-Eluting Stent): Another first-generation DES that effectively reduced neointimal hyperplasia by inhibiting cell cycle progression in vascular SMCs.[7]

12. ST (Stent Thrombosis): A serious complication wherein a thrombus forms at the stent site, causing acute vessel occlusion. DAPT significantly lowers this risk.[5]

13. TLF (Target Lesion Failure): Defined by adverse events like MI or need for repeat intervention at the treated site, TLF is a major clinical endpoint in trials evaluating stent efficacy.[6]

14. TLR (Target Lesion Revascularization): Refers to re-intervention at the previously treated lesion due to restenosis or thrombosis, seen more frequently in BMS and early DES.[7]

15. TVF (Target Vessel Failure): Involves failure of the entire treated vessel and may necessitate repeat PCI or coronary artery bypass grafting (CABG**)**.[8]

16. VLST (Very Late Stent Thrombosis): Occurring months or years after stent implantation, VLST is a life-threatening complication, especially associated with first-generation DES or poor DAPT compliance.[5]

17. ZES (Zotarolimus-Eluting Stent): A second-generation DES that releases zotarolimus, designed to provide a balance between effective restenosis prevention and lower thrombosis risk.[6]

Understanding these mechanisms and stent types is essential for clinicians to tailor treatment for coronary artery disease and improve patient outcomes. Stent selection, procedural technique, and adherence to medical therapy—including DAPT—remain critical to the long-term success of PCI.

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