

بسم الله الرحمن الرحيم





شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم



جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

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بعض الوثائق الأصلية تالفة





بالرسالة صفحات
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**Comparison of angiographic and clinical outcomes after stenting
versus cutting balloon angioplasty in small coronary vessels**

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Introduction

Introduction

Vessel size is inversely correlated with the risk of restenosis and adverse outcome after percutaneous coronary interventions.¹⁻³ This is because a smaller vessel is more limited in the ability to accommodate lumen restenosis, which invariably occurs to some degree in most vessels after balloon dilatation.^{4,5} Interventions in small coronary vessels ($\leq 3.0\text{mm}$) constitute a considerable proportion (30% to 50%)^{1-3, 6-8} of more than one million coronary catheter based procedures performed worldwide each year. PTCA and stenting are the two most frequently used interventions in patients with coronary artery disease.⁹ Large coronary vessels represent an established indication for stenting because of its superiority compared with PTCA, as shown in several randomized clinical trials.¹⁰⁻¹² Stenting is associated with increased procedural costs⁹; however, the improved outcome, with a reduction in the need for reinterventions, has rendered this technique more cost-effective in the long term than PTCA.^{12, 13} A retrospective analysis has shown that stenting might be also superior to conventional balloon angioplasty in small coronary vessels.¹⁴ However still carries some risk of late in-stent restenosis or subacute thrombosis, and these are encountered more in patients with small coronary vessels. The cutting balloon is a new device for coronary angioplasty, which by the combination of incision and dilatation of the plaque, is believed to minimize arterial wall trauma, the neoproliferative response, and subsequent restenosis.¹⁴ Because of the absence of appropriately designed randomized studies, there are no well defined recommendations¹⁵ regarding the intervention of choice in the setting of small coronary vessels. This is currently considered as a limitation in interventional cardiology and represents a great challenge to all coronary artery interventionists.¹⁶

Aim of the work

The aim of this study was to compare between coronary stenting and cutting balloon angioplasty as regard the early and late clinical and angiographic outcomes in small coronary vessels (< 3 mm in diameter) in patients with symptomatic coronary artery disease.



Review of the literature

ATHEROSCLEROSIS AND CORONARY ARTERY DISEASE

Hypercholesterolemia contributes substantially to the development and clinical expression of coronary and other forms of atherosclerosis.¹⁷ Considerable evidence suggests that cholesterol lowering stabilizes atherosclerotic plaques and reduces cardiovascular events, including all-cause mortality.¹⁸ Of all patients, those with established coronary heart disease benefit the most from cholesterol lowering.¹⁹ Eighteen of nineteen angiographic or ultrasound trials of hypolipidemic therapy have shown a reduction in disease progression, and a 20th study has shown an anatomic advantage for aggressive over moderate treatment.²⁰⁻⁴¹ Three large primary and secondary prevention event trials have demonstrated significant reductions in cardiovascular events in patients with a wide range of cholesterol levels.⁴²⁻⁴⁵

These findings suggest that aggressive lipid management can accomplish the same treatment goals as traditional antianginal and interventional therapy, namely, reductions of anginal frequency, exercise intolerance, cardiovascular events, and mortality.⁴⁶

*** Pathophysiology of atherosclerosis:**

In broad terms, coronary atherosclerosis is an initially slow process of endothelial dysfunction, plus intimal lipid, monocyte, and T-lymphocyte

accumulation leading to the migration and proliferation of smooth muscle cells and the elaboration of collagen and matrix in the subintimal layer.⁴⁷

Endothelial dysfunction could be caused by coronary risk factors, mechanical trauma, infections (possibly Chlamydia, cytomegalo virus, and herpes viruses), autoimmune processes, and the progressive modification of low-density lipoproteins (LDLs), predominantly by oxidation.⁴⁸ Once atherosclerotic plaques develop, the combined factors of local plaque inflammation, dissolution of internal plaque collagen, and vasomotion lead to plaque disruption, with ensuing partial or complete vessel thrombosis.⁴⁹⁻⁵¹

Cholesterol lowering has been shown to both slow the progression of coronary atherosclerosis and reduce plaque rupture.⁵²⁻⁵⁵ It may also decrease platelet adhesion to the denuded or ruptured vessel wall.⁵⁶⁻⁵⁷

Atherosclerosis pathways:

► Endothelial Pathway:

Two parallel processes play important roles in the initiation of atherosclerosis: endothelial dysfunction and lipid accumulation and modification⁵⁸⁻⁶¹ (Fig.1).

The endothelium is responsible for vasoregulation (vasodilation and vasoconstriction), vessel growth, aggregation of platelets, adhesion of monocytes, and fibrinolysis.⁶²

Normal endothelium prevents the development of atherosclerosis by promoting vasodilation (nitric oxide) and thrombolysis. All major coronary risk factors (both modifiable and immutable) are associated with endothelial dysfunction.⁶³

The mechanism of hypercholesterolemia and cigarette smoking appears to be through increased production of oxygen free radicals (predominantly superoxide anion) that combine with and deactivate nitric oxide.⁶⁴⁻⁶⁵ Nitric oxide deactivation results in increased platelet aggregation, monocyte adhesion, vasoconstriction and decreased fibrinolysis, all of which are important factors in the development of atherosclerosis, plaque rupture, and vessel thrombosis.^{66,67} Improvements in endothelium-dependent vasodilation have been demonstrated with antioxidant vitamins, B-complex vitamins, and estrogen administration.^{65,68} Antioxidant vitamin administration has also been shown to reduce the endothelial expression of monocyte adhesion molecules in smokers.⁶⁷

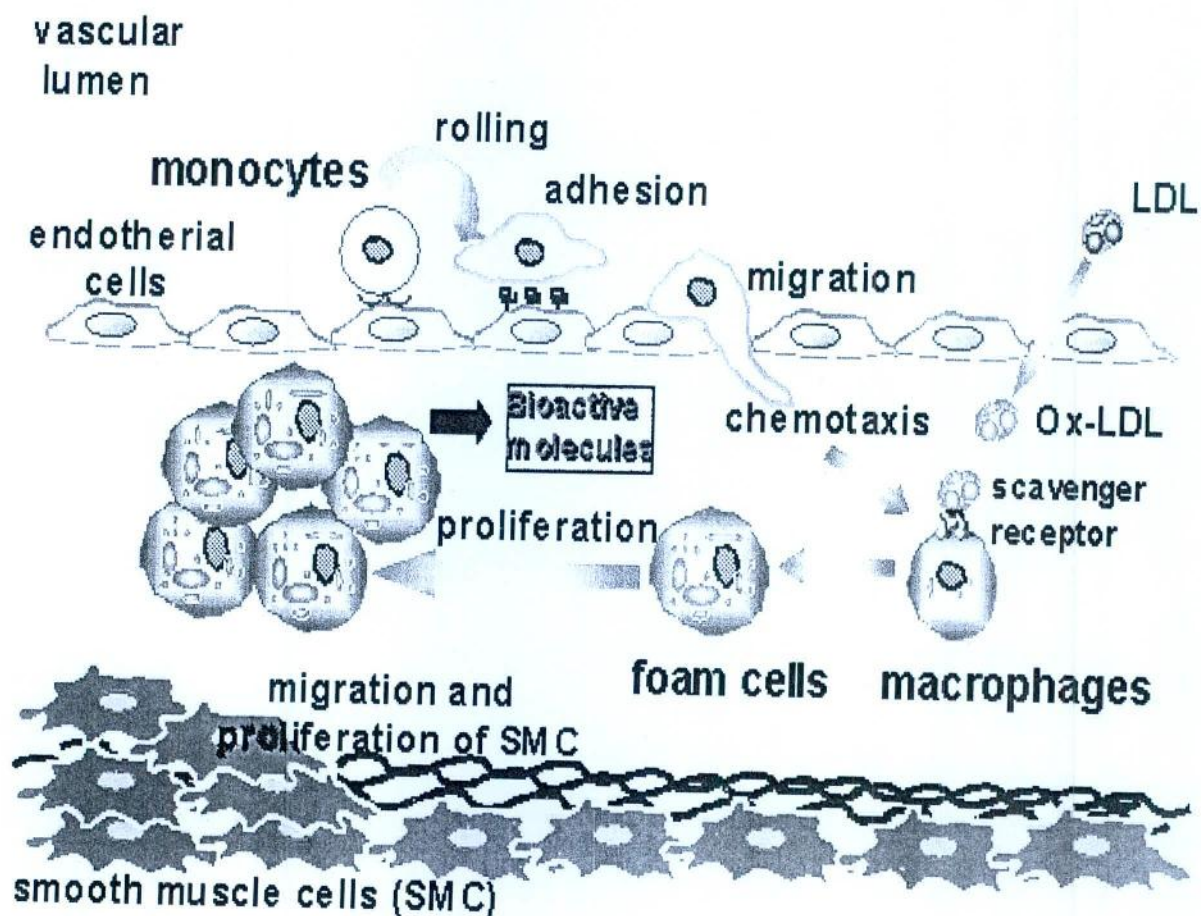


Figure 1

Pathogenesis of atherosclerosis demonstrating two important pathways: endothelial dysfunction and LDL accumulation and modification.

► Cholesterol Pathway:

Cholesterol deposition (predominantly from LDLs) and modification is the second major process initiating atherosclerosis.^{48,59,60} [Fig 1]. Initially, LDLs pass through the endothelial barrier by a process termed transcytosis. This process is accelerated by increases in serum LDLs and decreases in high-density lipoproteins (HDLs). The later in addition of being a source for reverse cholesterol transport back to the liver, it also exert an antioxidant effect.^{69,70}

With time, intimal LDLs undergo progressive modification through oxidation, glycosylation, and acetylation. Macrophages, endothelial cells, and smooth muscle cells are the likely sources of LDL oxidation. Modified LDLs are potent inhibitor of endothelial function. Meanwhile modified LDLs are recognized as foreign material by macrophages and taken up by their scavenger receptors, creating metabolically active foam cells. These elaborate a number of growth factors, which induce smooth muscle cell migration, proliferation, and matrix generation, leading to the formation of a complex atheroma. Dilation of the vessel wall occurs during the development of early atherosclerosis, reducing luminal compromise. Later, expansion of the atheroma exceeds this locally variable compensatory mechanism, leading to luminal narrowing.^{71,72}