

INTRODUCTION

Coronary artery stenting is the most important advance in the interventional cardiology since the introduction of balloon angioplasty more than 20 years ago ⁽¹⁾. In many centers, up to 80% of of percutaneous coronary interventions are accomplished by means of stent placement ⁽²⁾.

In our cardiovascular department (Nasr City Health Insurance Hospital), the first cases of coronary artery stent implantation were performed in the mid 1990s. With increased operator experience, better catheterization facilities and introduction of different types and sizes of stents, the use of stents dramatically increased and has become a routine procedure performed at our catheterization laboratory.

Stents provide favorable and predictable acute angiographic outcomes and provide favorable angiographic and clinical results in most complex lesion morphologies that are poorly treated using balloon angioplasty alone. In addition, stents improve the safety of angioplasty by successfully treating acute and threatened closures. Also, the use of stents is easy and decreases the total procedural time ⁽¹⁾.

Consequently, with increasing incidence of stent deployment, the problem of in-stent restenosis (ISR) “the Achilles heel of stenting” is becoming increasingly prevalent. The angiographic rates of ISR vary widely depending upon

patient, lesion, procedure and indication variables. The 22%-32% ISR rate previously reported by BENESTENT ⁽³⁾ and STRESS ⁽⁴⁾ trials represents ISR rate in a highly selected lesion type and can not be extrapolated to the “real-world” practice.

The pathophysiology of ISR involves early elements of direct injury to smooth muscle cells, deendothelialization, and thrombus deposition. Over time, this leads to smooth muscle cell proliferation and migration and extracellular matrix deposition. There is increasing evidence to suggest that inflammation plays a pivotal role linking early vascular injury to the eventual consequence of neointimal growth and lumen compromise ⁽⁵⁾.

Different percutaneous approaches are currently used to treat ISR: PTCA alone, lesion debulking with or without adjunctive balloon dilatation, additional stent implantation, cutting balloon angioplasty and more recently endovascular brachytherapy as an adjunctive therapy.

As regard PTCA as a treatment for ISR, some studies support the use of repeat balloon angioplasty as a primary treatment for focal ISR ⁽⁶⁾.

Also it was reported that 62% of lumen enlargement during PTCA of ISR was due to plaque compression, and 38% due to further stent expansion ^(7,8).

Cutting balloon enlarges coronary lumen mainly by in-stent tissue reduction and is associated with a moderate degree of additional stent expansion ⁽⁹⁾.