INTRODUCTION

The most common pathogenesis of coronary artery disease is atherosclerotic plaque formation in the coronary arteries leading to narrowing of the blood vessel and impairment of blood flow. Therapeutic measures are aimed at revascularization and increased blood flow. While coronary angioplasty is widely used today, the occurrence of restenosis in which the lesion regenerates in approximately 30 % of patients within three months ***(Burgos et al., 2005).***

Drug eluting stents with anti-inflammatory and anti-proliferative properties have been successfully proved to reduce neointimal hyperplasia and in-stent restenosis rates ***(Stone et al., 2004).***

Implantation of stents during percutenous coronary intervention are associated with vascular injuries which subsequently induces the activation of platelets, coagulation cascades and the secretion of inflammatory mediators. Further reactions such as the mobilization of inflammatory cells, proliferation of smooth muscle cells and the production of matrix ensue in the pathogenesis of neointimal hyperplasia. Therefore the inflammatory mechanisms that are activated after vessel injury are considered to play a key role in the development of in-stent restenosis ***(Bhatia et al., 2004).***

Of the inflammatory mediator substances the interleukins are the most well characterized interleukin 6 (IL-6) is the predominant determinant for production of acute phase protein such as C reactive protein and shows proinflammatory properties in addition to cellular

effects which are associated with restenosis IL-6 concentration which are raised in unstable angina are also raised after angioplasty, suggesting that IL-6 may be a sensitive marker reflective of the post procedure initial inflammatory response and also possible predictor of restenosis ***(Bennett., 2003)***.

C – reactive protein (CRP) is an acute phase protein produced in the liver under the influence of cytokines such as interleukin-6 which are secreted by activated macrophages. Serum CRP level are reported to rise and reach peak values about 48 hours after coronary stenting and persistently elevated CRP level beyond 48 hours are reported to be associated with an increased risk of in stent restenosis ***(Gottsauner et al., 2000)***.