



INTRODUCTION

The strong evidence that exacerbation of inflammation underlies the instability of atherosclerotic plaques suggests that anti-inflammatory therapy would be of additional benefit to patients already on optimal treatment (*Hansson, 2005*).

Although the concept of directly targeting inflammation in patients with atherosclerosis is not new, choice of a drug for suppressing inflammation in the vessel wall and atherosclerotic plaque requires careful consideration of likely benefit and risk.

Although statin therapy appears to have anti-inflammatory effect, it may be impossible to truly distinguish a direct anti-inflammatory effect from that achieved by low-density lipoprotein lowering alone (*Mora Ridker 2006*).

Statins also have many side effects which may lead some patients to discontinue the therapy such as GIT upset, fatigue, myalgias, hepatic impairment and serious myopathy (*Gundy, et al., 2001*).

The long-term safety of low-dose colchicines stands in stark contrast to long-term use of cyclooxygenase type 2 inhibitors ,which are associated with increased risk of coronary events in patients with coronary disease despite being able to decrease hs-CRP in the setting of acute coronary syndromes (*Lekakis et al., 2006*).

After colchicine is absorbed, it becomes highly concentrated in leukocytes and acts a potent anti-inflammatory agent with wide-ranging effects on the inflammatory process, even at a low dose. The evidence that colchicines may have actions at the level of atherosclerotic plaque,



because it is able to inhibit surface expression of adhesion molecules on T cells and endothelial cells, inhibit monocyte migration, decrease matrix metalloproteinase-9 secretion and inhibit tumor necrosis factor and interleukin-6 synthesis (*Bauriedle et al., 1994*)

Although colchicines use in patients with atherosclerosis has been considered, it has never been tested systematically in the clinical setting. This is surprising because the drug is known to have a number of anti-inflammatory actions that may promote stability of atherosclerotic plaque. It also widely available, inexpensive, well tolerated and effective in other inflammatory conditions at low doses over many years (*Ben-Chetrit, Levy, 1994*).

Colchicines is known to have an array of anti-inflammatory actions that may act to stabilize atherosclerotic plaque and its long-term safety and efficacy over many years has already been shown in patients with familial Mediterranean fever (FMF) (*Cerquaglia et al., 2005*).

C-reactive protein in acute phase synthesized in the liver and its concentration in response to tissue injury, inflammation is rapidly rising, the rate of the synthesis and the secretion of CRP increases within hours of acute injury or with onset of inflammation this is probably under the effect of several mediators such as IL 1&6. In patients with acute coronary syndrome, elevated level of high-sensitivity C-reactive protein >2.0 mg /L, is predictors of future vascular events (*Mark Nidorf, 2005*).