# OVERT AND SUBCLINICAL REACTIONS TO STREPTOKINASE IN ACUTE MYOCARDIAL INFARCTION AND IMPLICATION ON THE RENAL FUNCTION

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## INTRODUCTION

A cute myocardial infarction describes the development of ischemia and necrosis of a portion of the myocardium. It is a common manifestation of ischemic heart disease and a leading cause of serious cardiac complications ( Goldberger and Wheat, 1983).

Almost all myocardial infarctions result from atherosclerosis of the coronary arteries ,generally with superimposed coronary thrombosis . The genesis of the coronary atherosclerotic lesion is a complex and controversial issue, and a number of risk factors have been associated with the development atherosclerosis . However , regardless of the etiology and pathogenesis of the atherosclerotic process, the end result is plaques that cause luminal narrowing of the coronary arterial tree and in many instances ,a thrombus that causes further narrowing and often total occlusion (Baroldi , 1979).

Below a certain critical level of blood flow, myocardial cells develop ischemic injury. The pathophysiological studies revealed that irreversible myocardial damage occurs four to six hours following coronary occlusion, and trials of reperfussion should be performed within that period (*Kloner*, 1983).

In 1980, before the introduction of thrombolytic therapy, the mortality rates during hospitalization and the year following infarction were approximately ten percent ( *Braunwald*, 1987). But now thrombolytic therapy has been effective in reducing the morbidity



and mortality of acute myocardial infarction ( Yusuf et al., 1985).

Three thrombolytic agents ,Streptokinase [SK], recombiant tissue type plasminogen activator, [rtPA], and anisolyated plasminogen streptokinase activator complex [APSAC] are currently approved by the food and drug administration for intravenous use in patients with acute myocardial infarction (Heymann and Culling, 1994)

By far, the greatest international experience has been accumulated with streptokinase in part owing to its low cost and demonstrated efficacy in reducing mortality rates in very large trials (Yusuf et al.,1985). It dissolves clots by conversion of plasminogen to plasmin which is a fibrinolytic enzyme (Goldberger, 1983).

Recent to streptococci exposure or streptokinase , which is a foreign protein produced by B hemolytic streptococci . produces some degree of -mediated resistance to streptokinase (James, 1973) . In the international tPA/SK mortality trial allergic reactions were seen in 1.7% of patients given streptokinase. The reported reactions were rigors , rashes , less often generally mild as bronchospasm and dypsnea. A number of severe reactions has been documented mainly serum acute anaphylaxis . Also sickness and less often has documented proteinuria been (Alexopoulos et al., 1990). Hypotension is expected in four to ten %. Bleeding complications are of course the most important and most serious. Seventy



percent of bleeding of bleeding episodes occur at the site of vascular punctures (Gore et al., 1991).

Where as all these reactions are obvious clinically, less evident reactions may be over looked in routine clinical practice (Argent and Adams, 1991)

The aim of this study is to assess the possibility of predicting allergic reactions to streptokinase by measuring antistreptokinase antibodies before and after streptokinase therapy and by intradermal skin testing, and to determine if streptokinase infusion is associated with subclinical changes in the renal functions.