Introduction

Diabetic nephropathy is a microvascular complication of diabetes, specifically, it represents a major cause of morbidity and mortality in type 1 and type 2 diabetic subjects ***(Marshall, 2004***).It appears to develop as a result of interactions between environmental insults and genetic susceptibility. Indeed, hyperglycemia is a clinical prerequisite for this complication, but it should be noted that only a subset of diabetic subjects will ultimately develop nephropathy**.** ***(Rossing, 2005)***

Over recent decades, cellular and molecular mechanisms underlying diabetic nephropathy have been increasingly delineated. In particular, diabetic kidney disease appears to occur as a result of deleterious effects of both metabolic and haemodynamic insults, which at the cellular level lead to the activation of intracellular signaling pathways and transcription factors, thus triggering the production\ release of cytokines, chemokines and growth factors, which mediate and\or amplify renal damage. This ultimately leads to the structural and functional features characteristic of diabetic kidney disease (***Giunti et al, 2006)***

TNF –α a is a pleiotropic cytokine with a wide range of biological activities, its main function is to stimulate inflammation ***(Varfolomeev and Ashkenazi ,2004)*** .It contributes to the pathogenesis of both acute and chronic inflammatory diseases and has been a target for the development of new anti-inflammatory drugs***(Staniforth et al., 2004)***

Recently, most of the attention has been focused on the implications of TNF-α in the setting of diabetic nephropathy. it has an essential role in mediating inflammatory processes, furthermore TNF-α is cytotoxic to glomerular, mesangial, and epithelial cells and may induce significant renal damage ***(Navarro et al.,2005 (a)***