INTRODUCTION

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Rheumatoid arthritis (RA) is the most common inflammatory arthritis, affecting about 1% of the general population worldwide. Because of its prevalence and the ready accessibility of joint samples for laboratory investigation, RA has in many ways served as a model of inflammatory and immune mediated diseases ***(Firestein, 2001)***.

RA is a systemic inflammatory disease. The hallmark of the disease is a chronic for at least 6 weeks, symmetric polyarthritis that affects the hands and feet, although any joint lined by a synovial membrane may be involved ***(Howard and Sergent, 1997)***.

Joint destruction in RA is caused by invasion of articular cartilage by the synovial pannus, by localized osteoporosis, bone erosions and also by joint swelling and destruction of periarticular structures. The biologic processes that produce these morphologic changes are inflammation, proliferation and sepsis ***(Carson and Salvatore, 1977)***.

RA often begins with prodromal symptoms such as fatigue, anorexia, weakness, generalized aching and stiffness that is not localized to articular cartilage ***(Duthie et al., 1994)***.

Thrombin is a multifunctional enzyme that plays a crucial role in the haemeostasis and coagulation. It has proinflammatory

properties with a number of effects on the vascular endothelium as a stimulation of neutrophil adhesion to the vessel wall and prostacyclin release through up-regulation of arachidonic acid release. A side of these factors, thrombin acts as a mitogen for synovial cells. the most important physiological inhibitor of thrombin is Antithrombin III. This single chain glycoprotein has a molecular weight of 58000 Dalton is synthesized primarily in the liver and belongs to the serpin superfamily ***(Jones et al., 1998)***.

Activation of the coagulation cascade and over expression of thrombin within joints may contribute to the sustained joint inflammation of rheumatoid arthritis suggesting that the coagulation pathway, in particular thrombin mediated coagulation and signaling events, may be a target for therapy in joint inflammation ***(Nathaline Busso et al., 2001)***.

It has been found that major plasma proteinase inhibitors as α_1 antitrypsin, α_2 macroglobulin and AT III, was explored in RA, psoriatic arthritis and Reiter's syndrome. Experimental evidence is assesed to the potential role of serine proteinase and their inhibitors in the pathophysiology of inflammatory arthritis ***(Rothschild et al., 1985)***.

AT III is a member of blood proteins and belongs to the serine plasma inhibitors and which account for about 10% of total proteins in plasma. They provide one mechanism for the control of protein activity thus regulating many important biological reactions such as blood coagulation and complement activity control ***(Bodmer and Schnebl, 1984)***.