INTRODUCTION

Rheumatoid arthritis is a generalized disorder which, predominantly affect synovial joints and may have many extraarticular manifestation. The synovial membrane displays a chronic non-suppurative inflammation which can be associated with erosive destruction of the joints, cartilage, ligaments, tendons and subchondral bone ***(Manciourt et al., 1993).***

Recent studies indicate cell transfer between fetus and mother during pregnancy and can persist in both for decades after. The presence within one individual of a small population of cells from another genetically distinct individual is referred to as microchimerism ***(Adams and Nelson, 2004).***

Persistence of these cells has been demonstrated for many years after birth in the mother and child. Many autoimmune disorders like systemic lupus erythromatosis, primary biliary cirrhosis, autoimmune thyroid disease have all demonstrated the presence of fetal microchimerism ***(Sakar and Miller, 2004).***

The clinical similarities between some of autoimmune disease and the chronic graft versus host disease, the increased incidence of autoimmune disease observed in women after child bearing age, and the long – term persistence of microchimerism have raised the hypothesis that microchimerism could be

involved in the pathogenesis of autoimmune disease like rheumatoid arthritis ***(Turco and Bambara, 2004).***

Persistent fetal cells (CD34 + ve and CD38 +ve) have recently been found to persist in maternal blood for many years. CD34 +ve and CD38 +ve cells are progenitor cells that can differentiate into immune competent cells. Which ay lead to many immunological disorder like scleroderma, sjogren syndrome and Graft – versus host rejection ***(Evans et al., 2004).***

AIM OF THE WORK

The aim of the work is to study the role of microchimerism in patients with rheumatoid arthritis by detecting CD34 + ve and CD38 +ve cells.