INTRODUCTION
Takayasu's arteritis (TA) is a chronic granulomatous pan-arteritis characterized by stenosis, occlusion or sometimes aneurysm of large elastic arteries mainly the aorta and its major branches including pulmonary and coronary arteries (Kerr, 1995; Fraga et al, 2002 and Parra et al, 2003).

Epidemiologically, it is more prevalent in Asian and Latin American countries and constitutes the most common vasculitis in India and the third most common vasculitis in pediatric age group worldwide. In western countries TA is also known as "pulseless disease" because of the absence of pulses in the patients due to the obstruction of subclavian and/or brachial arteries. The disease occurs more commonly in young females with peak incidence between 15-20 years of age and it is an important cause of vascular hypertension, whose effect on heart, kidney and brain may result in congestive heart failure, chronic renal failure and cerebral hemorrhage, respectively leading to significant morbidity and mortality (Jain et al, 1996; Brogan et al, 2000 and Johnston et al, 2002).

Inflammatory damage of vascular tissues constitutes the primary pathogenic event in TA. It is followed by intimal hyperplasia and adventitial and medial fibrosis in the vascular wall that eventually culminates in stenosis or luminal occlusion leading to different clinical manifestations of the disease such as claudication and hypertension, which actually represent ischemia of organs supplied by the diseased blood vessels (Sarkisov et al, 1989 and Filer et al, 2001). The etiopathogenesis of TA is still an enigma. However, most of the clinical, pathological and experimental data available to date suggest that it is an autoimmune disease and both cellular and humoral immune mechanisms are involved in the pathogenesis of the disease (Noris, 2001).

Histological picture of TA shows a large number of perforin secreting T-cells, monocytes/macrophages along with other leukocytes and
increased expression of heat shock protein (HSP)-65 in the lesions (Seko et al, 1994 and Inder et al, 2000) as well as deposition of immunoglobulins in the vessel wall (Gupta, 1981). The other main pathological findings of the disease include presence of circulating activated T-cells bearing restricted T-cell receptor (TCR) repertoire (Nityanand et al, 1997a) and circulating antibodies mainly the AECA in majority of the patients with TA (Eichhorn et al, 1996). These studies together, point towards a fundamental role of T-cells, monocyte/macrophages and anti-endothelial cell antibodies (AECA) in the development of the disease. Thus the exact mechanism(s) via which autoreactive T-cells, monocyte/macrophages and AECA mediate tissue damage or other pathological processes and immunodominant antigens that stimulate generation of these immune components, are worth investigating in order to get newer insights into the immnopathogenesis of TA.

Therefore, we undertook this study as our research work on TA to elucidate the pathogenic mechanisms of T-cells, monocyte/macrophages and AECA in TA and to identify the putative antigen(s) that might induce the generation of these immune components in the disease.