AIMS AND OBJECTIVES: We aimed to study associations of HLA-E variants (HLA-E*01:01 and HLA-E*01:03) with disease susceptibility, phenotype and disease free survival in Indian patients with Takayasu arteritis (TA).

MATERIAL AND METHODS: Consecutive TA patients from Rheumatology and Cardiology clinics, Christian Medical College, Vellore, between August 2012 and December 2014 were followed up prospectively with demographic, clinical, ITAS-2010, TADS, laboratory and angiography details. Blood bank donors comprised the healthy unrelated age matched controls. Genotyping for HLA-E variants was performed using Amplification Refractory Mutation System PCR (ARMS-PCR). SPSS 16 was used for nonparametric and Chi square tests. Kaplan-Meier curve to depict outcome, multivariate logistic regression to assess independent associations and cluster analysis to define pattern of arterial involvement were performed.

RESULTS: HLA-E *01:01 and *01:03 allele frequencies in 150 TA (51.3% and 48.7%) and 264 controls (48.9% and 51.1%) were similar. HLA-E*01:01 homozygosity protected against $P+$ disease (OR 0.12, 95% CI- 0.14- 0.98, p= 0.047) and DCMY (OR 0.2, 95% CI- 0.05-1.03, p= 0.055). Sustained response, relapsing-remitting and persistent activity was observed in 60.2%, 20.4% and 19.4% respectively with $\delta$TADS=0 in majority. Low CRP and infra-
diaphragmatic disease predicted nonprogression; HLA-E*01:01/*01:01 genotype and normal ESR predicted angiographic stability.

Cluster analysis revealed symmetry, contiguousness and distinct supra-diaphragmatic versus coronary-infra-diaphragmatic clusters.

CONCLUSIONS: Our study on Indian TA patients did not reveal association of HLA-E variants *01:01 and *01:03 with disease susceptibility. HLA-E*01:01 homozygous genotype was protective against pulmonary artery involvement and DCMY as well as it predicted angiographically stable disease. CRP level below 11 mg/L at baseline is a predictor of good outcome in TA.

Key words: Takayasu arteritis, HLA-E, Vellore