Aortic Wall Stiffness Studies in Children with Kawasaki Disease without Coronary Artery Abnormalities – A Follow-up Study from North India

**Abstract**

The main focus of concern in Kawasaki disease (KD) hitherto has been on children who develop coronary artery abnormalities (CAA). There is emerging evidence to suggest that several long term cardiovascular sequelae (e.g. endothelial dysfunction), can occur in children with KD even without CAA. Abdominal aortic wall stiffness (AWS) is a surrogate marker of endothelial dysfunction. We evaluated AWS in a cohort of 30 North Indian children with KD without CAA, at least 5 years after the acute phase. There is paucity of literature on follow-up studies of AWS in KD in this setting.

**Patients and methods** 30 children with KD without CAA, diagnosed at least 5 years back, and who had a normal echocardiography examination at time of enrolment were included in the study. The diagnosis of KD was made on basis of American Heart Association criteria. Age, sex and socio-economic status matched healthy children were included as controls. All children underwent abdominal aorta studies by an experienced cardiologist (RM). Clinical measurements were obtained on Siemens Acuson Sequoia CS12 Echocardiography Machine. The study protocol was approved by Institute Ethics Committee.

**Results** Mean age of cases was 11.24±3.48 years with a mean interval of 7.72±2.37 years from initial diagnosis of KD. Mean aortic strain was 0.345±0.145 in cases as against 0.369±0.123 in controls (p > 0.05). Mean pressure strain elastic modulus (SEM) was 124.454±57.052 N/m² in cases as against 112.526±48.752 in controls (p > 0.05). Mean normalised pressure SEM was 1.898±1.023 in cases as against 1.700±0.700 in controls (p > 0.05). Mean peak flow velocity was 0.931±0.277 m/sec in cases and 0.915±0.215 in controls. No statistically significant differences could be detected between cases and controls in terms of aortic root diameters.

**Conclusion** Though a distinct trend towards higher AWS and lower distensibility was discernible in children with KD (without CAA), the difference did not achieve statistical significance. This could be because of the small sample size in this study. More long term studies on a greater number of subjects are needed to ascertain the extent of endothelial dysfunction in children with KD without CAA. This may have implications on long-term prognostication of children with KD without CAA.