Kawasaki disease (KD) remains to be the most common acquired paediatric cardiovascular disease in developed countries. It is five decades since its first description and reports of ischaemic heart disease in young adults with a history of KD are accumulating. While the sequelae of arterial inflammation in the acute phase of the illness are well documented, the long-term effects of KD on coronary and systemic arterial function are just emerging. Structural damage during the acute illness with development of persistent giant coronary arterial aneurysms no doubt constitutes the most significant morbidity and mortality after KD. Functional alterations of the coronary and systemic arteries beyond the sites of aneurysmal dilation as characterized by endothelial dysfunction, arterial stiffening, active arterial remodeling, and persistent low grade inflammation years after the acute illness have been demonstrated. Coupled with the late structural change of thickened arterial intima-media thickness, these findings have generated concerns regarding predisposition to premature atherosclerosis. There is further evidence to suggest genetic influence on the development of long-term arterial sequelae. Detailed assessment of arterial sequelae in the long-term has significant implications on risk stratification, approach to longitudinal monitoring of cardiovascular complications, medical management including transitional care, and patient education and counselling.