and subendocardial fibrosis along with patchy myocardial necrosis. Several workers have documented an ultrastructural defects. However, some degree of chronic impairment from preoperative ultrastructural abnormalities may persist. Singh and colleagues [4] demonstrated chronotropic impairment, blunted blood pressure response to exercise, and depressed ST segments in asymptomatic long-term survivors of ALCAPA repair. This suggests that the reverse LV remodeling plateaus in its beneficial effects in the late postoperative period. Impaired myocardial flow reserve in the left coronary territories and the presence of patchy fibrosis in the interstitial tissue surrounding viable myocytes may account for the incomplete reverse LV remodeling [4].

Successful revascularization sets in motion the process of reverse LV remodeling which improves LV function and for the majority of patients, leads to resolution of MR. The controversy regarding the requirement for a mitral valve procedure at the time of ALCAPA repair has been commented on elsewhere [5]. Although most patients show good functional recovery at rest, exercise testing still may reveal subtle persistent functional disability. This fact brings to light the importance of long-term follow-up and consideration of the modalities most appropriate for the evaluation of long-term survivors of ALCAPA repair. The optimal means of evaluation for asymptomatic long-term survivors of ALCAPA repair remains to be determined.

References


The degree of myocardial fibrosis appears to be the major determinant of postoperative functional recovery [2]; this underscores the importance of early revascularization in ALCAPA.

Preoperatively, most patients demonstrate markedly impaired left ventricular (LV) function and mitral regurgitation (MR). Ischemic papillary muscle dysfunction, LV free wall dyskinesia and LV dilatation are responsible for the MR. Both LV dysfunction and MR invariably improve after successful revascularization as a result of reverse LV remodeling. However, several degrees of chronic impairment from preoperative ultrastructural abnormalities may persist.

Ojala’s group has demonstrated the excellent functional results in children after anomalous origin of coronary artery from pulmonary artery (ALCAPA) repair [1]. The chronic ischemia occasioned by ALCAPA is not without lasting myocardial ultrastructural defects. Several workers have documented an important degree of fibrosis with altered but viable myocytes, endocardial and subendocardial fibrosis along with patchy myocardial necrosis [2, 3].

eComment: Incomplete left ventricular reverse remodeling after revascularization of anomalous left coronary artery from the pulmonary artery

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