Long-term fate of coronary arteries in Kawasaki disease.

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Introduction.
Late or inadequate acute Kawasaki disease treatment may lead to coronary artery (CA) dilatation. Giant CA aneurysms are recognized to be irreversible and may cause life-threatening complications. Aim of the study was to analyse long-term morphological and functional outcome of patients with CA affection after Kawasaki disease.

Patients and methods.
Performed was a retrospective analysis of 26 patients (20M/6F) followed at our institution between 1996-2016. Median age at diagnosis was 2.8 years (3 months – 11.6 years); median follow-up 8 years (5 months – 20 years).
Defined were baseline and late morphological CA characteristics (by echocardiography and CT-/coronarography), presence of myocardial ischaemia and/or ventricular dysfunction. According to baseline findings were patients divided in groups: 1. with mild CA dilatation (<5mm, without aneurysms), 2. small CA aneurysms (5-8mm); 3. giant CA aneurysms (>8mm).

Results.
Acute phase CA affection was as follows: both CA dilated in 13 (50%), only left CA dilatation in 12 (46.2%) and only right CA dilatation in 1 patient (7.8%). Group 1: 16 patients (61.6%); in all patients mild CA dilatation normalized within 3 months. Group 2: 5 patients (19.2%); in all patients small CA aneurysms were persistent during follow-up. In 2 (20%) late calcifications at aneurysm-site were found, though without significant stenosis; further 3 patients were without progression. No myocardial ischaemia or ventricular dysfunction were present. Group 3: 5 patients (19.2%); giant aneurysms of both CA were present, though right CA was more severely affected (multiple aneurysms/stenosis). Despite anticoagulation therapy late complications (at median 6 years after acute phase) were present in 4 patients (80%): in 1 progressive CA dilatation; in 3 complete right CA occlusion with collateral circulation, while in 1 of them later additional left anterior descending CA thrombosis occurred. In the latter 3 patients also myocardial ischaemia and/or ventricular dysfunction developed. One late death (20%) occurred due to severe ventricular dysfunction.

Conclusions.
After Kawasaki disease giant CA aneurysms have extremely high-risk potential for occlusion at any time during follow-up; this can lead to myocardial ischaemia, ventricular dysfunction or even death. Close clinical monitoring and repeated CA morphological and functional evaluation is therefor mandatory.