

Mechanical dispersion and ventricular dysfunction by myocardial strain is related to malignant arrhythmias in patients with arrhythmogenic right ventricular cardiomyopathy

Kristina Hermann Haugaa^{1,2},
 Sebastian Imre Sarvari^{1,2}, Ole Gunnar Anfinnsen¹,
 Otto A. Smiseth^{1,2}, Erik Kongsgård¹,
 Jan P. Amlie¹, Thor Edvardsen^{1,2}

- ¹ Department of Cardiology,
 Oslo University Hospital, Rikshospitalet,
 Oslo, Norway
- ² Institute for surgical research,
 Oslo University Hospital, Rikshospitalet,
 Oslo, Norway

Background: Life-threatening ventricular arrhythmias are frequent in arrhythmogenic right ventricular cardiomyopathy (ARVC). Mechanical dispersion (heterogeneous contraction) can be assessed by strain echocardiography and may reflect electrical dispersion, which is a strong arrhythmogenic factor found in ARVC patients. We hypothesized that RV dysfunction by myocardial strain is related to ventricular arrhythmia in patients with ARVC.

Methods: We included 37 patients with ARVC based on clinical criteria and 22 asymptomatic ARVC mutation carriers recruited by family genetic screening. Healthy individuals (n=30) were included for comparison. Strain was assessed by speckle tracking echocardiography. Myocardial function by strain was assessed in right (RV) and left ventricle (LV). The contraction duration in the 6 right ventricular (RV) segments were measured as the time from onset R on ECG to maximum shortening by strain in each segment. The standard deviation (SD) of the 6 contraction durations was calculated as a parameter of mechanical dispersion, reflecting heterogeneous contraction.

Results: Ventricular arrhythmias (VT or VF) were documented in all 37 patients with overt ARVC. RV and LV function by strain was decreased in those with arrhythmias (-19±7% and -16±5%) compared to those without (-24±5% and -20±2%, both p<0.01). Patients with arrhythmias showed increased RV mechanical dispersion compared to those without (53±25ms vs 33±20ms, p<0.01). Figure shows increased mechanical dispersion in an ARVC patient with arrhythmias compared to an ARVC patient without. White vertical arrows indicate time at maximum myocardial shortening. Mechanical dispersion was pronounced in so far asymptomatic ARVC mutation carriers compared to healthy individuals (33±20ms vs. 15±8ms, p<0.05), indicating subclinical cardiac involvement.

Conclusion: RV mechanical dispersion assessed by strain was increased in ARVC patients with arrhythmias. RV and LV strains were decreased in ARVC patients, reflecting biventricular involvement. Subclinical RV dysfunction was present in so far asymptomatic ARVC mutation carriers. These novel markers may become important tools in risk stratification of ARVC patients.

