Correlation of Myocardial Bridging to Regional Hypo-, A- or Dyskinesis of Left Ventricular Function in Takotsubo Cardiomyopathy

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ARTICLE INFO

Received Date: Janaury 26, 2020
Accepted Date: February 10, 2020
Published Date: February 11, 2020

KEYWORDS

Myocardial Bridging
Takotsubo Cardiomyopathy
LAD

Citation for this article: Stefan Peters. Correlation of Myocardial Bridging to Regional Hypo-, A- or Dyskinesis of Left Ventricular Function in Takotsubo Cardiomyopathy. SL Clinical And Experimental Cardiology. 2020; 3(1):121

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OPINION

The causes of regional hypo-, a- or dyskinesis of left ventricular function in takotsubo cardiomyopathy remains unclear. These changes are transient in many cases, but a chronic takotsubo cardiomyopathy exist in a few cases [1].

A couple of explanations for regional contraction abnormalities exist: spasm, myocardial bridging and recurrent segment of the left anterior descending coronary artery (LAD). The ECG changes, left ventricular function and patients complaints last only a few weeks and normalize without scar formation. If cardiac MRI after normalization is done, minimal regional changes of left ventricular function without ejection fraction reduction and fibrosis is often present. Longitudional strain is a risk parameter in long-term follow-up [2].

In a moderate number of patients of my collective with myocardial bridging had a significant correlation to hypo-, a- and dyskinetic contraction impairments of the left ventricle. The determinant of typical (apical ballooning) or atypical (midventricular or basal ballooning) was recurrent segment of the left anterior descending coronary artery. If the course of the LAD was short nor rearching ventricular apex, midventricular or basal ballooning was present.

If the essential factor of takotsubo cardiomyopathy is myocardial edema of unknown origin, myocardial edema probably pushes down the course of the LAD into the myocardium leading to apical or midventricular/basal ballooning. After a few weeks there is a reduction of myocardial edema thus normalizing left ventricular function at echocardiography or – in a small amount of cases – left ventricular angiography.

Myocardial edema occurs in myocardial infarction, myocarditis, and takotsubo cardiomyopathy. In takotsubo cardiomyopathy the amount of myocardial edema is very high [3].

Thoracic computer tomography after normalization of left ventricular function should be called for in numerous cases in order to make a statement whether myocardial bridging still exists and MRI is necessary to stratify what the amount of myocardial edema is.

Only with these steps the origin of takotsubo cardiomyopathy (or syndrome) can be elucidated. The differential aspect of cardiomyopathy (still present) or syndrome (only sporadic without any MRI changes) can be solved.

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