

an early >70% reduction of initial ST shift 90 min after reperfusion. SA and total protein levels were assessed on blood samples at admission.

Results: The mean age of the 45 patients enrolled was 63 ± 14 years, 38 were male, 20 had anterior MI. Median SA level was 3.71 ± 0.47 g/dl, median total protein level was 6.39 ± 1.07 g/dl. The median CTFC value was 13.1 ± 8.1 .

Patients with a lower MBG (<2) had a slower epicardial coronary flow expressed as higher CTFC (21.1 vs 11.1 , $p=0.001$). Patients with MBG <2 had significant lower SA (3.2 g/dl vs 4.1 g/dl, $p=0.01$) and total proteins levels (6.2 g/dl vs 7 g/dl, $p=0.02$). SA level was also lower in patients ST-resolution <70% (3.2 g/dl vs 3.9 g/dl, $p=0.03$). We found a significant negative linear correlation between CTFC and both SA (figure 1) and serum total proteins levels (respectively: $R = -0.35$, $p=0.028$; $R = -0.39$, $p=0.021$).

Conclusions: Among patients with STEMI treated with pPCI, baseline lower SA levels are associated with worse myocardial reperfusion. This study suggests that hypoalbuminemia may contribute to the development of microvascular obstruction.

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Microvascular function detected by timi frame count (TFC) and myocardial blush grade (MBG) in patients with takotsubo syndrome

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Introduction: Takotsubo Syndrome (TS) is a pathological entity characterized by an acute and generally reversible left ventricular dysfunction. Clinical presentation mimics acute coronary syndromes (ACS), and is typically associated with kinesis abnormalities and angiographically undamaged coronary arteries. To date, causes and pathophysiological mechanisms underlying the disease remain uncertain; the most reliable hypothesis is that microvascular dysfunction could be responsible of myocardial stunning that is realized in the areas involved by kinesis abnormalities.

Purpose: Aim of this study was to analyze microvascular function in patients with TS and anterior myocardial infarction with ST-segment elevation (STEMI) by using two quantitative angiographic parameters: Timi Frame Count (TFC) and Myocardial Blush Grade (MBG).

Methods: 67 patients with TS (mean age 68 ± 10 years) were retrospectively analyzed. A control group of 67 patients with anterior STEMI and same age, left ventricular ejection fraction (LV-EF) and cardiovascular risk factors was selected. Patients with STEMI were further divided into two subgroups based on successful myocardial reperfusion ($n=35$) or persistence of microvascular obstruction (MVO; $n=32$). Epicardial flow was assessed by TFC according to the Gibson method. Myocardial perfusion was studied using the QuBE software (Quantitative Blush Evaluator).

Results: In patients with TS the anterior interventricular (AIV) artery TFC was higher than patients with reperfused STEMI ($p=0.0018$), while it was similar to the group of subjects with STEMI with persistent microvascular obstruction ($p=0.58$). QuBE score in patients with TS was lower than reperfused control group ($p=0.008$) and higher than patients with STEMI and MVO ($p=0.0005$). Furthermore, microvascular dysfunction in patients with TS was widespread and not limited to AIV artery territory alone.

Conclusions: Our results support the hypothesis that microvascular dysfunction is one of the pathophysiological mechanisms underlying TS. Microvascular dysfunction was less severe compared to STEMI with persistent MVO and milder compared to STEMI with successful reperfusion. Moreover it was wide spread and did not pertain to the territory of a single epicardial coronary artery.

PATHOPHYSIOLOGY OF CORONARY ARTERY DISEASES

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Natural history of coronary lesions in the distal segment of total occlusions after successful percutaneous recanalization

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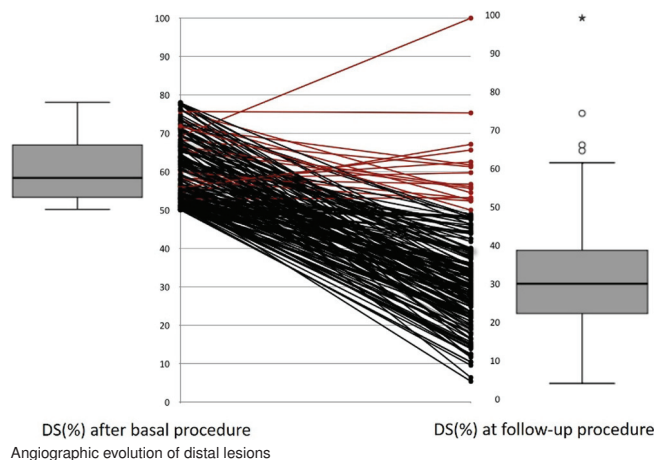
Background: After successful coronary total occlusion revascularization, the segment distal to the occlusion itself undergoes physiological changes with documented increases in vessel diameters when control angiography is performed at follow-up.

Objectives: Aim of this study is to investigate this phenomenon in a large population of patients undergoing total occlusion recanalization and assess its clinical implications.

Methods: Two big randomized-control trials, which enrolled patients with successfully recanalized coronary occlusion treated with DES implantation, were used as data source for this study. Quantitative coronary analysis (QCA) was performed immediately after baseline procedure and at 8–9 months follow-up, to specifically address the characteristics of the segment distal to the CTO lesion. Measures included mean lumen diameter (MeanLD), minimal lumen diam-

eter (MLD), reference vessel diameter (RVD), and mean and minimal lumen gain (MeanLG and MLG). Moreover, the presence of binary stenosis was assessed in this segment at baseline and follow-up procedures.

Results: Of the 634 patients forming the original study populations of the two trials, 425 were eligible for this analysis. Mean length of the distal segment analysed was around 40mm. Baseline QCA disclosed a MeanLD of 1.97 ± 0.49 mm and a MLD of 1.33 ± 0.53 mm, with a reference diameter of 2.28 ± 0.46 mm. Binary stenosis was detected in 167 (39.3%) patients. Follow-up QCA showed increase in all measures (MeanLG 0.42 ± 0.35 mm and MLG of 0.54 ± 0.45 mm). A lumen gain >0.1 mm was observed in 351 (82.6%) patients. Moreover, 151 out of 167 (90.4%) patients with binary stenosis at baseline showed regression of the lesion at follow-up. Percutaneous intervention in the distal segment was necessary in only 7 cases.



DS(%) after basal procedure

Angiographic evolution of distal lesions

DS(%) at follow-up procedure

Conclusions: Our results, deriving from the largest population so far analyzed with this purpose, suggest that lesions in the segment downstream the coronary occlusion can be safely deferred from PCI and eventually re-assessed with follow-up angiography if judged untrustworthy.

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Association of brain amygdalar activity with plaque burden and vulnerability in coronary artery

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Background: Emotional stimulation was reported to be related to coronary events, but, there is a lack of direct mechanistic evidence between metabolic activity of amygdala, a neural stress center, and coronary artery disease (CAD) including acute coronary syndrome. In this study, we aimed to evaluate whether brain amygdala activity validated by ¹⁸F-FDG PET/CT could be associated with the occurrence of CAD and plaque vulnerability of the patients who underwent coronary artery evaluation; invasive coronary angiography (ICA) or coronary CT angiography (CCTA).

Methods and results: We analyzed patients who underwent ¹⁸F-FDG PET/CT and examination of coronary artery (ICA or CCTA) at Korea University Guro Hospital (Seoul, Korea). Among 1802 patients underwent ¹⁸F-FDG PET/CT from October 1 2009 to 31 December 31 2015, 230 patients received ICA or CCTA. After excluding 36 patients who had active cancer or brain disease, 194 were stratified according to degree of coronary severity based on Syntax score and diseased vessel number [none ($n=96$); mild to moderate ($n=61$); severe ($n=37$)]. Brain amygdalar activity on PET was quantified as target-to-background ratio (TBR) of standardized uptake value (average of both amygdalar SUVmean/cerebellar SUVmax). The amygdalar TBR was significantly higher in the patients of severe coronary disease rather than those who had no coronary stenosis or mild to moderate group in terms of syntax score ($P<0.01$, respectively). Intriguingly, among 37 patients had severe CAD, the amygdalar TBR increased more prominently in the 10 patients clinically presented as myocardial infarction (MI) compared to non-MI, severe stenotic CAD patients ($P<0.01$).

Conclusions: The amygdalar activity assessed by ¹⁸F-FDG PET/CT was significantly higher in patients with severe CAD. This increased amygdalar activity was more evident in patients who was presented as MI. Our results suggest that brain amygdalar activity could contribute to plaque development and instability in coronary artery.