Methods 74 patients (95 stents) with OCT post-stent implantation were included in the study. Cross-sectional OCT images were analysed at 1-mm intervals (every 15 frames), and 302 cross-sectional images with lipid or calcific lesions under the stent struts were selected. The struts in these sections were divided into four groups according to the plaque conditions under the struts: group 1—struts on the normal vessel, group 2—on the fibrotic lesion, group 3—on the calcific lesion, group 4—on the lipid rich lesion. The neointimal hyperplasia thickness on the stent struts were measured by OCT.

Results The plaque conditions under 806 struts could be detected clearly by OCT. Among them, 157 struts were on the normal vascular wall (the intimal thickness were less than 250 μm by OCT), 344 struts were on the fibrotic lesions, 145 struts on calcific lesions and 160 struts on lipid lesions. The neointimal thickness were 0.152±0.081 μm, 0.145±0.091 μm, 0.150±0.105 μm and 0.166±0.083 μm respectively in group 1–4. The p value was 0.011.

Conclusions The plaque type has impact on the in-stent neointimal hyperplasia. The underlying lipid or calcific components in plaque may promote neointimal hyperplasia after stent implantation.

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**e0321 THE CORRELATION OF CARDIAC TROPTONIN I AND DECOMPENSATED HEART FAILURE**

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Objective Correlation of Cardiac Troponin I and Decompensated heart failure. Small studies report that cTnI is elevated in severe heart failure (HF) and may predict adverse outcomes.

Methods Serial blood samples were instantly collected from 336 patients who presented Decompensated heart failure (NYHA class III-IV) at Admission. Patients with acute myocardial infarction or myocarditis were excluded from analysis. Measurement of cTnI and Brain natriuretic peptide (BNP), echocardiography was done after Disease condition Improved. cTnI was detectable (cTnI >0.40 ng/ml) in serum of 161 patients (47.9%). Patients with detectable cTnI levels had significantly higher B-type natriuretic peptide (BNP) levels (p<0.001). A significant correlation was found between detectable cTnI and progressive decline in ejection fraction over time. Furthermore, detectable cTnI was associated with increased mortality risk (RR, 2.57; 95% CI, 1.43 to 3.78). After adjustment of other factors associated with adverse prognosis including age, sex, ejection fraction, and coronary artery disease, cTnI remained a significant predictor of death. cTnI used in conjunction with BNP further improved prognostic value.

Conclusions cTnI is associated with elevated BNP levels and progressive left ventricular dysfunction in patients with severe HF. cTnI may be a novel, useful tool in identifying patients with HF who are at increased risk for progressive ventricular dysfunction and death.

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**e0322 TO INVESTIGATE INAPPROPRIATE DISCHARGE OF**

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Objective In order to avoid and decrease inappropriate discharge caused by implantable cardioverter defibrillators (ICD), we analysed the cause of inappropriate discharge.

Methods Clinical follow-up data of inappropriate discharges by ICD in 6 patients were studied. All the 6 patients were male, 2 were dual chamber ICD, while 7 were single chamber ICD. The patients were followed-up from 0.5 to 9 (averaged 4.5) years.

Results During follow-up, all the patients survived. Arrhythmias were detected 358 times, ICD treatment was performed 71 times, inappropriate discharges occurred 14 times. 2 patients who implanted ICD earlier experienced all the 14 inappropriate discharges. The causes of inappropriate discharges include over-sensing sinus tachycardia (3 times), electromagnetic interference (2 times), lead insulation breaks (9 times). They accounted for 21.4%, 14.2%, 64.2% of total inappropriate discharges, respectively.

Conclusion Inappropriate discharge of ICD should be taken seriously consideration.