agnostic modalities are limited. Cardiac sarcoidosis (CS) has been reported to be in up to 30% of systemic sarcoidosis and an important prognostic factor due to heart failure and fatal arrhythmias. Thus, isolated type of cardiac sarcoidosis (isolated CS) is thought to be rare, but its prevalence, clinical characteristics and prognosis are still unknown. Therefore, we examined the clinical characteristics and the results of the medical therapy in isolated CS, compared with systemic sarcoidosis with cardiac involvement (systemic CS).

Method and results: Total one-hundred ninety Japanese patients with cardiac sarcoidosis were collected from 43 hospitals (clinical diagnosis in 82, and biopsy proven in 108 cases, M/F=58/132, mean age at diagnosis = 62.4±12.1 years). Among them, 16 patients (8.4%) were diagnosed as isolated CS by endomyocardial biopsy (M/F=5/10, mean age at diagnosis = 52.3±12.3 years), however there were 15 more patients (7.9%) with a strong clinical suspicion but no histological evidence of CS. The clinical findings of isolated CS were not different with those in systemic CS (isolated vs. systemic CS: mean left ventricular ejection fraction at diagnosis was 47.0±19.1% vs. 42.0±17.4%, septal thinning by echo cardiography; 50% vs. 47%, high grade AV block; 56% vs. 49% and sustained ventricular tachycardia/fibrillation; 31% vs. 25%). Mean angiotensin-converting enzyme (ACE) were lower in isolated CS (15.6 vs. 19.2 IU/l, $P<0.05$) but lysozyme was not different. Corticosteroid was given in all patients and defibrillation device (ICD/CRTD) rescued 17% of patients during a follow-up period of 5.9 years.

Conclusion: Isolated CS without clinical evidence of sarcoid involvement in other organs, is not rare. Age at diagnosis was relatively young but its clinical characteristics are similar to systemic CS. However, half of them are still undiagnosed by endomyocardial biopsy.

P4247 | BEDSIDE
Determinants of prolonged impairment of global longitudinal strain post Takotsubo cardiomyopathy

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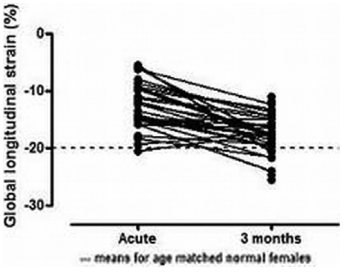
Purpose: In patients with Takotsubo cardiomyopathy (TTC), left ventricular wall motion (LVWM) usually recovers rapidly, therefore residual symptoms may be described to depression. However, we have previously shown that inflammatory activation and accentuated BNP release persist for at least 3 months post TTC, and this is associated with impaired global longitudinal strain (GLS). We have now sought to determine (1) the bases for heterogeneity of residual GLS and (2) whether this results either from severe acute inflammation or slow resolution of inflammatory changes.

Methods: Data from TTC patients with adequate 2D speckle-tracking echocardiographs were evaluated. Correlations between GLS and (i) severity of acute episode [peak CK, NT-proBNP, metanephrine, high sensitive C-reactive protein (hsCRP), asymmetric dimethylarginine (ADMA) levels, platelet responsiveness to NO, LVWM score index, and myocardial T2 signal intensity on MRI], (ii) residual markers of inflammation were evaluated by univariate and multiple linear regression analyses.

Results: In 51 TTC patients aged 66±12 (SD), acute GLS was significantly impaired compared to 3 months (-12±4% vs -18±3%, p=0.0001). GLS at 3 months was significantly correlated with acute elevation of NT-proBNP and metanephrine levels (r=0.34, p=0.02; r=0.4, p=0.009, respectively). Moreover, metanephrine levels represented an independent predictor of poor recovery (β=0.4; p=0.03). Furthermore, residual GLS was significantly correlated with simultaneous NT-proBNP (r=0.47, p=0.003); this association persisted with multiple linear regression (β=0.51; p=0.002).



Conclusions: Prolonged impairment of GLS post TTC is correlated with extent of acute catecholamine elevation, and can be predicted in individual patients on the basis of persistent NT-proBNP elevation.

P4248 | BEDSIDE
Blood myeloid dendritic cell reduction and differences between myocarditis and dilated cardiomyopathy

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Aims: The role of dendritic cells (DCs) in cardiomyopathies is unknown yet. Dilated cardiomyopathy (DCM) is often preceded by myocarditis, hence the two conditions often clinically overlap. In order to investigate the role of DCs in diagnosing myocarditis/DCM, we prospectively investigated the circulating DC precursors (DCPs) as well as mature DCs in endomyocardial biopsies (EMBs) from patients with acute myocarditis and newly diagnosed DCM.

Methods: Blood myeloid (mDCPs) and plasmacytoid (pDCPs) DC precursors were flow cytometrically analyzed in patients with newly onset heart failure (HF) and biopsy proven DCM (n=43, mean EF 29±10%) and patients with MRI confirmed myocarditis (n=23). Individuals with excluded heart disease were used as controls (n=52). Left-ventricular EMBs were performed when clinically indicated (n=45) and were immunohistologically analyzed for the presence of mature myeloid DCs (CD83+) and tissue fibrosis (Sirius red). Analyses of circulating DCPs were performed initially and at repeated follow-up intervals of 6 months. Correlation analysis was performed between circulating DCPs and mature DCs in EMBs, as well as clinical, echocardiographic and invasive left-ventricular parameters.

Results: Relative and absolute numbers of circulating mDCPs were reduced in patients with myocarditis compared to controls (0.065% vs 0.2%,$p<0.001$). Following clinical recovery of myocarditis, circulating mDCPs tended to normalise (0.015% vs 0.2%,p=0.008). Circulating mDCPs of DCM patients were at initial presentation unchanged compared to controls (0.18% vs 0.2%,p=0.258), but were significantly reduced at 6- and 12-month-follow-up (0.16% vs 0.2%,p=0.047 and 0.14% vs 0.2%,p=0.021). No statistical significance was observed for pDCPs. Correlation analysis revealed inverse correlation of blood mDCPs with myocardial mature DCs in EMBs (r=-0.522,p=0.026). We also found an inverse correlation of blood mDCPs with cTNI (r=-0.826,$p<0.001$), CRP (r =-0.388,p=0.014), invasively measured EF (r=-0.383,p=0.026), but not BNP (p=0.921). Blood mDCPs positively correlated with tissue fibrosis (r=0.395,p=0.05), mitral and tricuspid regurgitation (r=0.395,p=0.012 and r=0.47,p=0.002).

Conclusions: Circulatory mDCs migrated into the myocardium of these patients. While their peripheral reduction was acute in myocarditis, it was delayed and chronic/progressive in DCM. Thus, inflammatory processes in DCM were not causative, but rather secondary to structural myocardial changes. DCs could prove to be discriminatory between myocarditis and DCM in early diagnosis.

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Left ventricular outflow tract velocity time integral correlates with low cardiac output syndrome in patients with acute decompensated heart failure

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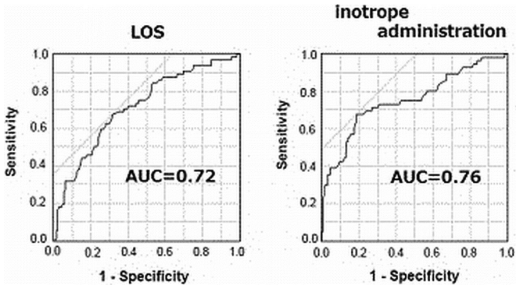
Purpose: Left ventricular outflow tract velocity time integral (LVOT VTI) is a simple method by Doppler echocardiography to calculate left ventricular stroke volume. In ESC guidelines, LVOT VTI<15cm is defined as an abnormality suggesting reduced left ventricular stroke volume, however it is unknown whether it correlates with low cardiac output syndrome (LOS) or not. The aim of this study is to elucidate the usefulness of LVOT VTI when diagnosing LOS and to decide the optimal cut-off value for inotrope administration in acute decompensated heart failure (ADHF).

Methods: A total of 212 patients admitted for ADHF were divided into two groups according to LVOT VTI. Clinical characteristics were compared between High-VTI (VTI≥15cm, n=99) and Low-VTI (VTI<15cm, n=113).

Results: Low-VTI showed younger age (p=0.0001), lower systolic blood pressure (p=0.005), faster HR (p=0.0004), higher plasma BNP levels (p=0.0134), lower %FS (16.1 vs. 23.6%, $p<0.0001$), compared with High-VTI. Low-VTI showed higher prevalence of inotrope administration (37.2 vs. 14.1%, p=0.0002) and LOS signs, such as pulsus alternans (17.7 vs. 8.1%, p=0.0006), cool extremities (26.6 vs. 9.1%, p=0.0012) and proportional pulse pressure<25% (17.7 vs. 8.1%, p=0.0436). There was no difference in in-hospital and 3-year mortality between two groups.

In ROC curve analysis, the optimal cut-off value of LVOT VTI for prediction of LOS signs and inotrope administration were 13.9cm (sensitivity 0.68; specificity 0.69; AUC 0.72) and 12.3cm (sensitivity 0.68; specificity 0.81; AUC 0.76), respectively.



Conclusions: Low-VTI was associated with the presence of LOS signs and requirement of inotrope administration in patients with ADHF, suggesting importance of LVOT VTI measurements to assess LOS in ADHF.