P0479
Poster Session I
Other viral infections

COMMON VIRAL GENOMES AND IMMUNOHISTOLOGICAL INFLAMMATORY MARKERS DETECTION IN ENDOMYOCARDIAL BIOPSIES OF PATIENTS WITH UNEXPLAINED DILATED CARDIOMYOPATHY

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Objectives: Cardiotropic viruses are suspected to be etiological causes or cofactors in the development of idiopathic dilated cardiomyopathy (IDCM). In the present study, we assessed the presence of common viral genomes and inflammatory markers in endomyocardial biopsies (EMB) of IDCM, explained DCM and control patient groups.

Patients and methods: Between 2008 and 2013, EMBs were prospectively performed in Reims University Hospital according to ESC and AHA guidelines in 13 patients (sex ratio M/F=2.5, mean age 47.5±11 years) suffering from IDCM and 7 suffering from explained dilated cardiomyopathies (DCM) (sex ratio M/F=2.5, median age 49±15.5 years). In each patient, 3-6 fragments were sampled and 2 were immediately frozen for molecular investigations. Human enteroviruses (HEV), HCMV, EBV, HSV1, HSV2, VZV, HHV6, HHV7, and HHV8 were detected by PCR assays coupled to microarray hybridization analyses (Clart Entherpex V8.0; Genomica, Madrid, Spain) and adenovirus and human parvovirus B19 (PVB19) were detected by specific real–time PCR assays. Classical histology (HPS staining) and CD3, CD68, HLA-DR immunohistochemical assays were performed for each patient. Molecular and histological results were compared to those obtained from large tissue samples of 14 healthy heart controls (sex ratio M/F=6, mean age 37+/-12 years) who died from suicides or traumatic accidents and who were autopsied in Reims University Hospital (control group). Qualitative variables were compared using Fischer exact test. *P* value <0.10 was considered as significant.

Results: Cardiac infections by HEV, PB19 HHV6 or EBV were identified by PCR microarray in respectively 84.6% (11/13), 71.4% (5/7) and 100.0% (7/7) of IDCM, DCM and negative control patients. HEV RNA was detected in 23.1% (3/13) of IDCM patients but not in DCM and negative control patients (*P*=0.09). PVB19 DNA was detected in 76.9% (10/13), 71.4% (5/7) and 85.7% (6/7) of IDCM, DCM and negative control patients (*P*=0.99). HHV6 and EBV DNA were detected in 38.4% (5/13), 0% and 71.4% (5/7) of IDCM, DCM and control patients. CD3, CD68 and HLA-DR immunohistochemical assays were positive in 53.8% (7/13), 57.1% (4/7) and 0% of IDCM, DCM and in negative control patients whereas HPS staining was negative in all study patients. Cardiac Inflammatory markers were detected in 100% (3/3) of EV positive IDCM and only in 60% (6/10) of PVB19 positive IDCM patients (*P*=0.49).

Conclusions: HEV-RNA detection was positive and was always associated with cardiac inflammatory markers in only 23% of IDCM patients, whereas PVB19-DNA detection was detected in more than 70% of IDCM, DCM and control patients and was not always associated with the presence of cardiac inflammation markers. These findings suggested that only HEV but not PVB19 could be an etiological cause or a cofactor in the development of a subset of DCM cases.