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ePoster Viewing

Virology non-HIV/non-hepatitis

Detection of viral genomes and inflammatory markers in endomyocardial tissues of patients with idiopathic and explained dilated cardiomyopathy and healthy heart and surgery control groups

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Objectives: Cardiotropic viruses are suspected to be etiological causes or cofactors of dilated cardiomyopathy (DCM). Here, we analysed Endomyocardial Biopsy samples (EMBs) of DCM patients for the presence of cardiac viral infection and inflammatory markers.

Patients and methods: Between 2008 and 2014, EMBs were performed in Reims University Hospital according to ESC guidelines in 13 patients (sex ratio MF=2.5, mean age 47.5±11 years) suffering from idiopathic DCM (iDCM) and 10 suffering from explained DCM (eDCM) (sex ratio MF=2.33, median age 47.3±15.9 years). For each of these patients, 3-6 fragments were sampled and 2 were immediately frozen for virological analyses. Human enteroviruses (EV), human parvovirus B19 (PVB19) and all Human Herpes virus (HHV) were detected by specific real-time PCR assays (Argene Biomérieux®). CD3, CD68 and HLA DR immuno-staining were performed. All results were compared to those obtained from large cardiac samples of 11 healthy heart controls (sex ratio MF=4.5, mean age 36.4+/-11.1 years) who died from suicide, intoxication or traumatic accident and were autopsied at Reims University Hospital. EV and PVB19 genomic detection results were compared to those obtained from 47 right atrium tissues sampled in patients during extracorporeal circulation (sex ratio MF=4.22, mean age 68.2±10.2 years) (surgery controls). Qualitative variables were compared using Fischer exact test. Quantitative variables were compared using Mann Whitney U test. A p value <0.05 was considered as significant.

Results: EV, PVB19, HHV6, HHV4, HHV7 or HHV1 genomes were identified in 84.6% (11/13), 80% (8/10), 100% (11/11) and 78.2% (37/47) of iDCM, eDCM, healthy heart and surgery control groups, respectively. EV RNA was detected in 23.1% (3/13) of iDCM patients but not in eDCM, healthy heart and surgery controls ($P=0.003$). Mean EV viral load was 803 copies/μg in iDCM patients. PVB19 DNA was detected in 76.9% (10/13), 80% (8/10), 63.6% (7/11) and 78.2% (37/47) of iDCM, eDCM, healthy heart and surgery control groups ($P=0.99$). Mean PVB19 viral loads were: 413, 346, 1428 and 71 copies/μg in iDCM, eDCM healthy heart and surgery control groups ($p=0.31$). CD3, CD68 and HLA-DR immunohistochemical assays were positive in 53.8% (7/13), 57.1% (4/7) and 0% of iDCM, eDCM and healthy heart control groups. Cardiac Inflammatory markers were detected in 100% (3/3) of EV positive iDCM and only in 60% (6/10) of PVB19 positive iDCM patients.

Conclusions: EV-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 approximately 70% of iDCM, eDCM healthy heart and surgery control groups and was not always associated with the presence of cardiac inflammation markers. These findings suggested that only a persistent EV infection could be an etiological cause or a cofactor in the development of a subset of iDCM cases.