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Abstract (poster session)

Prevalence and quantitative detection of common cardiotropic viruses in endomyocardial tissue samples of sudden cardiac death victims in northern east of France, 2008-2012

D. Giusti, F. Tissier, N. Leveque, F. Renois, C. Boulagnon, P. Fornes, L. Andreoletti* (Reims, FR)

Objectives: Several common cardiotropic viruses are suspected to be etiological causes of sudden cardiac death (SCD) in young adults. Here, we identified cardiac viral infections and we assessed the viral load levels in the cardiac tissues of SCD victims . Patients and methods: Common cardiotropic viruses (Human enteroviruses (HEVs), HCMV, EBV, , HSV1, HSV2, VZV, HHV6, HHV7,HHV8) were detected by PCR assays coupled to microarray hybridization analyses (Clart Entherpex V8.0; Genomica, Madrid, Spain) and by specific real–time PCR assays for adenovirus and human parvovirus B19 (PVB19). A normalized quantitative viral genomic detection was performed by classic real-time PCR assays in virus positive SCD cases. One hundred and ninety-seven frozen heart tissue samples obtained from 24 SCD adult victims (mean age = 34 years +/-13; M/F= 14/10) and 28 frozen heart samples collected from 14 healthy heart controls (mean age 57 years +/-15) who died from committed suicides or traumatic accidents were retrospectively analyzed. All of these study patients had been autopsied between 2008 and 2012 in the Reims University Hospital Centre (Northern east of France). Results : Single (n=13) or multiple (n=6) viral cardiac infections were detected in 19 (79%) of 24 SCD adult victims: PVB19=9 (47.4%), PVB19-HHV6=4 (21%), HHV6=3 (15.8%), EBV=1 (5.2%), EBV-PVB19=1 (5.2%), EBV-PVB19-HHV6=1 (5.2%). PVB19 median load value was 197 UI/µg of extracted total nucleic acids, whereas HHV6 median load value was 28 DNA copies/µg of extracted total nucleic acids. Among the 19 viral positive SCD victims, 15 (79%) demonstrated an absence of any known acute or chronic cardiac pathologies associated with an absence of heart histological abnormalities, whereas only 4 (21%) of them displayed the presence heart histological abnormalities compatible with a chronic myocarditis (n=1) or genetic or unexplained dilated cardiomyopathies (n=3) (21 vs. 79%; P<0.001). Only one (7%) of the 14 healthy heart control subjects was positive for cardiac viral genome detection (PVB19) (79 vs. 7%, P<10-3). Conclusions : We identified single or mixed PVB19 and HHV6 cardiac infections as leading potential causes of SCD in adult subjects living in the Northern east of France. The low viral load levels were compatible with chronic persistent or latent cardiac infections. Further additional investigations would be necessary to assess the physiopathological role of these viruses in SCD events occurring in young adult subjects.