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Differences in virus prevalence and load in the hearts of patients with chronic dilated cardiomyopathy with and without immune-mediated inflammatory disease

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Introduction

Autoimmune responses against the heart and infections with cardiotropic viruses have been suggested to play a major role in the pathogeneses of idiopathic dilated cardiomyopathy (DCM). The interaction and cross-talk between these complex mechanisms is not completely understood, making etiologic distinction difficult.

Aim

We compared the prevalence and quantity of cardiotropic viruses in heart tissue of DCM patients with and without a previously diagnosed immune mediated inflammatory disorder (IMID).

Patients and methods

Myocardial tissue samples and serum was obtained from 159 consecutive patients with DCM and 20 controls. Patients were subdivided into groups based on the absence (n=125) or presence (n=34) of an IMID, and controls (n=20).

Results

The IMID patients showed elevated serum soluble interleukin-2 receptor and neopterin compared to the non-autoimmunity patients and controls, compatible with the fact that these patients had an increased cellular immune activation related to their IMID.

The non-IMID group revealed a higher PVB19 prevalence (100/125) compared to the autoimmunity patients (16/34, p=0.04) and controls (11/20, p=0.02) and PVB19 copy numbers (561 ± 97 vs. 191 ± 92 copies/μg DNA, and 103 ± 47 copies/μg DNA, respectively, both p<0.001).

Both the non-IMID and IMID DCM patients demonstrated increased myocardial inflammation compared to controls (12.5 ± 1.8 and 14.0 ± 3.2 vs. 5.1 ± 0.7 CD45-positive inflammatory cells, both p<0.05).

Conclusion

Our data shows a similar PVB19 prevalence and load in hearts of autoimmunity DCM patients and controls, but increased prevalence and levels in non-autoimmunity DCM patients. These findings suggest that ICM patients in the presence of an IMID have a different pathophysiologic mechanism compared to the virus-induced form of ICM.

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