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The role of viruses in ARVD and atrial dysplasia

Guy Hugues Fontaine
Pierre-and-Marie-Curie University, France

The histologic structure of the RV free wall shows a typical pattern from epicardium to endocardium. In addition to apoptosis and adipogenesis, now reproduced in-the-dish fibrosis bordering or embedding cardiomyocytes is indispensable for a positive diagnosis of arrhythmogenic right ventricular dysplasia (ARVD). The quiescent form of this disease is found in 3.7% of the general population. However, in some patients it is possible to observe patchy distribution of areas of thick fibrosis occupied in some cases by lymphocytes (chronic-active form). This is the marker of myocarditis mostly due to viral infection. Presence of viruses has been found in patients with ARVD. At the end stage of the disease, the cause of death is irreversible heart failure. However, the patients with a LVEF<45% showed major presence of fibrosis and lymphocytes invading both ventricles. This suggested the unexpected association of myocarditis and viruses as a superimposed phenomenon in ARVD patients and the cause of heart failure. Moreover, it has been recently demonstrated (Lopez-Ayala HR 2016) that ARVD patients are more susceptible than others to myocarditis explained by the same genetic factor at the origin of the trouble in development. This concept discovered in ARVD can be extended to all the other forms of cardiomyopathies. This concept is important since it is now possible to treat viral myocarditis.

guy.fontaine2@numericable.fr