Noncompaction in systemic lupus erythematosus

Finsterer J[1], Zarrouk-Mahjoub S[2]

ACTA REUMATOL PORT. 2014;39:279-280

Dear Editor,

With interest we read the article by Rabelo *et al.* about a 30yo black female with systemic lupus erythematosus (SLE) and acquired and transient left ventricular(LV) hypertrabeculation/noncompaction (LVHT)[1]. We have the following comments and concerns.

Obviously, LVHT in the presented patient was acquired since it has not been detected on transthoracic echocardiography in 2009[1]. Absence of LVHT in 2009 could be explained by various speculations. Either, LVHT was truly absent and developed within the following two years or LVHT was already present in 2009 but hidden by severe dilatation of the LV or by severe LV thickening, as has been previously reported[2]. It is also possible that LVHT was present in 2009 but misinterpreted or overlooked due to unawareness of the phenomenon. Was there severe dilatation or severe thickening of the LV myocardium in 2009? Was the video of the 2009 investigation revised by two independent echocardiographers for LVHT? In case LVHT developed after 2009 it would be interesting to know which trigger stimulated the generation of LVHT. Was it a drug newly given during this period, such as leflunomide, or was it the cardiac function which induced a compensatory cardiac mechanism? How do the authors explain that echocardiography was unremarkable in 2009 but showed eccentric LV hypertrophy in 2011[1]?

LVHT was no longer seen in 2012 and systolic function and pulmonary artery pressure had become normal[1]. Did also LV hypertrophy regress? Which was the reason why LVHT disappeared within 1 year? Disappearance of LVHT has been reported in association with thickening of the LV myocardium[3]. Did LV hypertrophy described in 2011 further increase? It is also conceivable that LVHT was still present in 2012 but invisible due to improved LV contraction status[4]. It is also possible that trabeculations regressed because they were no longer needed for cardiac function. Since LVHT is occasionally mixed up with ventricular thrombi it would be interesting to know if the female ever experienced any cardioembolic events[5]. The patient was put on warfarin since 2011[1]. Could it be possible that oral anticoagulation "resolved" LVHT since it was in fact thrombotic material and not LVHT?[4] Disappearance of LVHT has been also reported after myocardial ischemia and scar formation[6]. Did the presented patient experience myocardial ischemia between 2011 and 2012? Did she ever report anginal chest pain, were ECG or blood chemical investigations ever indicative of myocardial infarction or did coronary angiography indicate coronary heart disease?

To explain the occurrence of LVHT in association with SLE it can be speculated that the myocardium became involved in the inflammatory process and stimulated a compensatory mechanism. Did the patient ever undergo myocardial biopsy? Was there any indication for myocarditis or endocarditis? Did cardiac function improve under immunosuppressive treatment simultaneously with improvement of organs affected by the SLE? It can be also speculated that she suffered from a double trouble. Since acquired LVHT has been most frequently reported in patients with neuromuscular disorders (NMDs)[7], it is essential to exclude a NMD. Did she ever experience symptoms typical of a NMD? Did the family history indicate NMD? Was the patient ever investigated by a neurologist?

Since LVHT may be found also in other family members of an affected patient[8] it would be interesting to know if any of the relatives were echocardiographically investigated. Other family members may show LVHT disregarding if LVHT was associated with a genetic disorder in the index case or not.

Overall, this interesting case raises a number of questions and concerns which need to be addressed before finally assessing if LVHT was truly LVHT, truly acquired, if it truly disappeared, and if there is a need for modification of the current management.

1. Krankenanstalt Rudolfstiftung, Vienna, Austria;
2. Laboratory of Biochemistry, UR "Human Nutrition and Metabolic Disorders" Faculty of Medicine, Monastir; Tunisie

CORRESPONDENCE TO
Josef Finsterer
Postfach 20, 1180 Vienna, Austria
E-mail: fifigs1@yahoo.de

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