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Hyperthyroidism and noncompaction

Editor,

With interest, we read the article by Habib *et al.* about a 26-year-old Afro-American female with left ventricular hypertrabeculation/noncompaction (LVHT) in whom hyperthyroidism (Grave's disease) was assumed to have triggered heart failure.[1] We have the following comments and concerns.

We do not agree with the notion that exclusively hyperthyroidism triggered the development of heart failure.[1] Although hyperthyroidism may induce heart failure,[2] it has to be excluded that heart failure was due to LVHT alone, in which heart failure is a common complication.[3]

Autoimmune disease in a single organ can be associated with autoimmune disease in other organs, such as the liver, heart, intestines, or muscle.[4] Were there indications for extrathyroid autoimmune disease or manifestations of hyperthyroidism in the skin? Was the family history positive for autoimmune disease?

LVHT has familial occurrence.[5] Were first-degree relatives screened for LVHT? In how many of these family members, was LVHT detected in addition to the presented patient? Did first-degree relatives manifest with cardiomyopathies other than LVHT?

The authors regard LVHT as a genetic cardiomyopathy.[1] Although LVHT has been reported in association with mutations in >40 different genes, with polymorphisms, and numerous chromosomal defects,[6] a causal relation between these defects and the occurrence of LVHT awaits to be established. Arguments which make a causal relation unlikely are that LVHT can be acquired, that in case of autosomal dominant transmission, LVHT may not occur in each generation, that cardiac disease associated with a particular genetic defect can be highly variable within a family, that in families with an inherited genetic defect and LVHT, LVHT can be found only in some of the mutation carriers (LVHT does not segregate with a certain mutation), and that the number of mutated genes claimed to cause LVHT is large.[7]

LVHT is associated with neuromuscular disorders in up to 80% of the cases, particularly if patients with a neuromuscular disorder are systematically screened for cardiac involvement.[8] In addition, hyperthyroidism may go along with secondary myopathy. Was the patient seen by a neurologist? Were there indications for a neuromuscular disorder or weakness, wasting, easy fatigability, myalgias, muscle cramps, hyperCKemia, or a myopathic electromyography?

Overall, the report about this interesting case could profit from provision of a family screening for LVHT or other cardiomyopathies, exclusion of autoimmune disease in organs other than the thyroid gland, and from neurological investigation of the index case.

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Conflicts of interest

There are no conflicts of interest.

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