# *Letters to the Editor*

**Non-indicated Oral Anticoagulation in Left Ventricular Hypertrabeculation/Noncompaction May Jeopardize These Patients**

With interest we read the article by Bohrer et al., on a patient with left ventricular hypertrabeculation/noncompaction (LVHT) under oral anticoagulation (OAC) in whom a substitution in the CYP2C9 gene was detected.[1] The article raises some objections and concerns.

It is a common error to attribute LVHT solely to an arrest in the embryonic compaction of the myocardium. Noncompaction of the myocardium is only one of several pathogenetic scenarios that explain the development of LVHT. The noncompaction theory does not apply to repeatedly reported cases in which LVHT developed during adulthood.[2]

Since there are no reliable data on the prevalence of LVHT in children and adults and the figures reported are frequently estimations, the comparison between the prevalence of LVHT in children and adults is not reputable.

No consensus has been reached so far as to whether LVHT is associated with an increased risk of stroke or embolism or not. In a study of 62 of our own patients with LVHT we did not find an increased risk of these patients developing stroke or embolism as compared with age-, sex-, and left ventricular fractional shortening-matched controls.[3] Though stroke or embolism have been reported in single patients with LVHT, we regard it as not justified to generally propose OAC for LVHT patients, unless there is concomitant atrial fibrillation, severely reduced left ventricular function, or any other established indication for OAC. There is also no evidence for a general clotting defect in LVHT patients, which would require OAC.

What was the cause of recurrent syncopes in the presented patient? Which type of rhythm abnormality was detected to indicate the implantation of a pacemaker and the cardioverter defibrillator? What were the results of the ambulatory ECG? What was the cause of hypertrophic cardiomyopathy?

Was the patient investigated for neuromuscular disorders, which are associated with LVHT in up to 80% of the cases?[4] Neuromuscular disorders associated with LVHT are dystrophinopathies, dystrobrevinopathies, myotonic dystrophy, zaspopathies, myoadenylate-deaminase deficiency, Charcot-Marie-Tooth disease, mitochondrial disorder, Barth syndrome, Friedreich ataxia, or Pompe's disease.[5]

Was the C430T substitution in the CYP2C9 gene a mutation or a polymorphism? Were the criteria for a mutation fulfilled?

Were causes other than the CYP2C9 substitution excluded as causes of the increased INR value, in particular co-medication and nutritional factors? Was the metabolisation of substances other than phenprocoumon, also metabolised via the CYP2C9 pathway, also disturbed?

Though mutations in the G4.5 gene are frequently associated with LVHT, various other mutated genes have been described as being associated with LVHT, such as the lamin A/C, cypher/ZASP, GAA, DMPK, AMPD1, mitochondrial, frataxin, or PMP22 genes.[5] Was there any indication that the CYP2C9 substitution was directly responsible for the development of LVHT?

Since there is no general indication for OAC in LVHT patients, we suggest withdrawing the coumadin therapy in the presented patient. Since many issues concerning the phenomenon of LVHT are still unsolved, we also suggest reliance strictly on evidence.

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