



New oral anticoagulants for thromboembolic risk from noncompaction?

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Letter to the Editor

With interest we read the article by Cevik et al. about a 62 years-old female who was diagnosed with left ventricular hypertrabeculation / noncompaction (LVHT) and multiple intraventricular thrombi, for which she received oral anticoagulation but died from multiorgan failure due to unknown cause [1]. We have the following concerns: Which was the cause of death in this patient? Were ventricular arrhythmias recorded before decease? Did systolic function deteriorate shortly before decease? Was there clinical evidence for cardioembolism? Did she develop an infectious disease before death? LVHT is not congenital in each case. A number of patients with acquired LVHT have been reported [2]. Were previous echocardiographies reviewed to confirm that LVHT was present already before

diagnosis? Did the patient undergo cardiac MRI to confirm the diagnosis of LVHT?

The patient is described as disorientated [1]. Which was the cause of her disorientation? Was a cerebral MRI carried out? Was there any indication for stroke, intracerebral bleeding, migraine, stroke-like episode, transient global amnesia, or seizures? Did she have a hepatic problem or hypothyroidism? We should know if sedating drugs were responsible for impaired consciousness and orientation. How do the authors know that heart failure was the consequence of LVHT? Why not the other way round? Is it conceivable that LVHT represents a compensatory mechanism in patients with cardiomyopathy? Cardiac MRI is not as valuable as illustrated, since it may overlook LVHT in single patients. The patient complained about general weakness [1]. Which was the cause of general weakness? Was

general weakness attributable to heart failure, muscle weakness, acidosis, infection, or respiratory failure? Was the patient a smoker or non-smoker and investigated for neuromuscular disorders (NMDs) or chromosomal abnormalities, frequently associated with LVHT [3]. Were family members other than the index case investigated for NMDs? Were other family members investigated to look for familial occurrence of LVHT, as has been previously reported [4]. The authors propose the use of new oral anticoagulants (NOAKs) in patients with LVHT and additional risk factors for primary prevention of cardioembolism. However, NOAKs have a number of disadvantages. NOAKs cannot be monitored, in case of bleeding only an antidote for dabigatran is available, the bleeding-risk may be increased in older patients because of renal insufficiency, patients with intracerebral bleeding and ischemic stroke within the last three months were excluded from the rating approval studies, in patients with a confusional state it is not guaranteed that they swallow the drug without chewing, and it may not be guaranteed that the drug is adequately stored [5].

Overall, we recommend not using NOAKs for primary or secondary prophylaxis as long as these drugs are not tested for the indication LVHT. We recommend to anticoagulate patients with LVHT only if they present with risk factors for thromboembolism other than LVHT, such as atrial fibrillation, systolic dysfunction, or previous embolism. Vitamin-K-antagonists should be preferred as long as NOAKs have been shown to be more effective in this indication.

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