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LETTER TO THE EDITOR

Increased risk of stroke after the diagnosis of heart failure: is it a paradox of initiating heart failure treatment?

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Dear Editor,

We have read with great interest the article published by Alberts et al. [1] recently. In their cohort of Rotterdam study, they have evaluated the risk of stroke in heart failure patients. Briefly they have found that the risk of stroke is increased in the early phase after the diagnosis of heart failure and attenuates over the time.

In accordance with this report, Witt et al. [2] reported 17-fold increased risk of stroke within first month of heart failure diagnosis. On the other hand the underlying mechanism of increased risk of stroke after the diagnosis of heart failure has remained unknown. We believe that current literature underestimate or miss the effects of heart failure treatment. Any patient with the diagnosis of heart failure will receive the heart failure treatment. So the "1 month after diagnosis" term includes the treatment modalities as well. Those treatment alternatives are mainly preload reducing diuretics, after-load reducing drugs, namely angiotensin converting enzyme inhibitors or angiotensin receptor II blockers and positive inotropic drug digoxin.

A poorly contracting ventricle allows blood stasis that can lead to coagulation activation [3], thrombus formation and subsequent embolization. Even without gross thrombus formation, these regional blood flow disturbances could lead to an increase in platelet adhesion due to prolonged residence time of platelets in the cardiac chambers [4]. At least 11% of patients with left ventricular (LV) dysfunction have one or more embolic events during the course of their illness [5]. The incidence of LV thrombus in patients with left ventricular dysfunction or dilated cardiomyopathy has been reported in the literature [6–8] as 11–44%. The clinical importance of LV thrombus lies in its potential to embolize. Beyond the visible left ventricular thrombus formation, echocardiographically invisible thrombus formation may also occur in left ventricular cavity. Roberts et al. [7] has demonstrated that even in the absence of LV thrombi by echocardiography, it is possible that mural thrombi or endocardial plaques, most likely resulting from organized thrombi in 25% of patients with dilated cardiomyopathy, may exist in left ventricle.

Recently our group has reported two consecutive cases [9] of newly diagnosed LV systolic dysfunction or dilated cardiomyopathy complicated by stroke within first week of treatment including diuretics, angiotensin converting enzyme inhibitors and digoksin. There was not left ventricular thrombus formation or atrial fibrillation. We have commented that beside the coincidental embolization risk of echocardiographically invisible LV thrombus, relative improvement in LV contraction, preload and after load reduction, and relative decrease of end-diastolic LV pressure might have facilitated the release of freshly formed small thrombi from the LV or less likely from the left atrial cavity.

The effects of initiating heart failure treatment on the risk of stroke has never been thought and assessed. Presuming that all patients, having the diagnosis of systolic heart failure would have received the heart failure treatment concurrently, increased stroke risk early after the diagnosis of heart failure period can be explained by the paradoxical treatment effect of positive inotropic drugs or drugs reducing left ventricular filling pressure.

In conclusion, we can suggest that the period of several months after the diagnosis of systolic heart failure, anticoagulant drugs can be considered in the routine heart failure treatment modalities. This issue should be taken into consideration in future prospective clinical studies as well.

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The Authors' Reply

Dear Editor,

We would like to thank Dr Yetkin and colleagues for proposing a valuable hypothesis to explain the findings from our recent article. Dr Yetkin and colleagues hypothesize that the initiation of treatment of a failing heart with preload and afterload reducing drugs and inotropic drugs, which improves left ventricular contraction, facilitates the embolization of thrombi from the left ventricle. We read this hypothesis with great interest and agree with our colleagues that the proposed mechanism is a plausible explanation for our findings and fits well with our data..

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