B-type natriuretic peptide and cirrhosis: Is there any relation?

B tipi natriüretik peptid ve siroz, aralarında ilişki var mıdır?

To the Editor,

We read with great interest the recently published article performed by Yılmaz et al.(1) regarding the relationship between B-type natriuretic peptide (BNP) and progression of cirrhosis. The use of BNP in the evaluation of cirrhosis may be a more useful and easy-to-use predictor of the disease in comparison to previously described models such as the Child-Turcotte-Pugh classification and the Model for End-Stage Liver Disease (MELD) scoring system. Although a significant association has been shown between increased BNP levels and the severity of the disease, the results of the study reported by Yilmaz et al. are questionable and open to criticism as a consequence of three arguments.

First, in the study performed by Yılmaz et al., the authors used the statement "brain" type natriuretic peptide instead of "B-type" natriuretic peptide in the manuscript. Brain natriuretic peptide was identified from the porcine brain tissue initially and is primarily synthesized from the ventricle. The name was subsequently changed to B-type natriuretic peptide (BNP) (2,3).

Second, in this study, patients were divided into three groups according to the Child-Turcot-Pugh classification. We found that upper ranges of BNP in these three groups were over 1000 most likely in pg/ml. The absence unit of BNP is a very interesting point in this study. It has been reported that a level of BNP over 500 pg/ml is diagnostic for the congestive heart failure presented and diagnosed by lower ejection fraction on echocardiographic examination (4). Therefore, unless the cardiac ejection fractions of these patients were provided, the relationship of increased serum BNP levels with the severity of cirrhosis remains quite questionable.

Third, in the conclusion of the abstract section, Yılmaz et al. attributed the increased level of serum BNP, which was released predominantly from the cardiac ventricles, to portal hypertension. This assumption needs to be clarified by the cardiac catheterization and echocardiographic findings, especially by E and A wave ratio (E/A ratio). The E/A ratio is a useful indicator of the pulmonary capillary wedge pressure and left ventricular end-diastolic pressure (5). Invasive and non-invasive cardiac data are needed to suggest this unclear hypothetic mechanism as a conclusion. Therefore, attribution of the increased serum BNP levels to the portal hypertension is a quite-disputable opinion.

In conclusion, even though the positive significant results of the study reported by Yilmaz et al. seem promising, evident design bias and the absence of enough data that clarify the attribution of the increased BNP to portal hypertension and to the severity of the disease limit its validity and make its conclusions controversial.

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