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Commentary

Role of Thyroid Stimulating Hormone in Acute Decompensated Heart Failure

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DESCRIPTION

Cardiologists will think that it makes sense that thyroid dysfunction would be related to a number of cardiovascular conditions. Particularly, it been documented that low free Triiodothyronine (fT3) levels have negative effects on cardiovascular diseases. Earlier studies have shown that there is no consistent relationship between thyroid function and chronic heart failure among cardiovascular diseases. The prognostic value Of Thyroid-Stimulating Hormone (TSH) in chronic heart failure was variable. Furthermore, no previous research has proved the critical role that thyroid function plays in the management of Acute Decompensated Heart Failure (ADHF). Considering this deficiency, it is first required to determine whether or not we are able to differentiate the unfavorable outcome of ADHF patients as per thyroid function upon admission.

Analysis of the Receiver Operating Characteristics (ROC) curve revealed that fT3 performed best. An interesting result from a multivariate study suggests that a low fT3 level was independently associated with a higher in-hospital mortality rate. Others with low fT3 had a significantly higher chance of dying entirely within one year than patients with normal fT3. These results are illuminating and also have significant medical implications. The present study's design, goal, and statistics are reasonable and well-organized because the authors have specialist skills in clinical research and the treatment of heart disease.

A low fT3 level was associated with poor outcomes in patients hospitalized for ADHF, according to the authors' conclusions, and fT3 measurements should be done for ADHF patients to help with risk stratification. Provided that thyroid hormone acts on the myocardium through the specific T3 receptors and that low fT3 levels may worsen contractility, increase susceptibility to arrhythmias, and contribute to mortality in patients with heart failure despite normal T4 and TSH levels, it is also feasible that fT3 has a good prognostic value for ADHF patients than TSH. We should discuss whether a target or severity marker in ADHF is a low fT3 level.

Because thyroid hormones have had an impact on a variety of cells and organs and are related to the maintenance of normal cardiac function, low fT3 levels must result in heart failure. In actuality, decreased ejection fraction and pulmonary capillary wedge pressures were linked to low fT3. Thermogenesis of tissue, cardiac contractility, heart rate, and cardiac output are all enhanced by fT3, which also reduces systemic vascular resistance, including that of the coronary artery, according to previous studies. Taking into consideration these results, low fT3 would be a therapeutic target for ADHF, and a prior clinical trial has shown that short-term T3 replacement is helpful for advanced heart failure.

The ratio of hypothyroidism in pathophysiology is not uniform and has dynamic features; therefore we should establish the causal importance of hypothyroidism in ADHF patients. However, we are concerned that long-term T3 replacement could have unexpected inotropic effects. Hypothyroidism is further brought on by numerous systemic organ damage in heart failure. Cachexia and/or poor nutritional status have lately attracted attention as problems in heart failure, and these topics are strongly mediated by hypothyroidism, especially low fT3 levels, as the authors discuss Cardiologists must take low fT3 levels, at the very least, into consideration as a valuable clinical measure of severity and risk stratification in ADHF, even if the current research was unable to determine whether thyroid function is the aim or a severity marker of ADHF patients.

The measurement of thyroid hormone was not carried out in all ADHF patients, as the authors stated and that the prevalence and prognostic significance of persistent thyroid function abnormality in ADHF patients. The time course of low fT3 levels in ADHF patients and a more detailed assessment of ADHF (echocardiographic, hemodynamic, and exercise tolerance test) are anticipated in the future following studies, that will support the authors' theory. However, the clinical message from of the current authors is intriguing, and we should progressively focus on low fT3, instead of TSH, in ADHF patients.

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