心衰竭和甲狀腺疾病

Heart failure and thyroid diseases

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Heart failure represents the initial clinical presentation in about 6% of patients with hyperthyroidism, with half having left ventricular (LV) dysfunction. Hyperthyroidism has been shown to result in hemodynamic changes with a reduced ejection fraction (EF) and cardiac output due to a decrease in myocardial contractile reserve. Previous study was able to demonstrate the long-term effect of hyperthyroidism on LV remodeling, cardiac function, myocyte function, and tissue remodeling in animal models. Patients with hypothyroidism may develop increased diastolic blood pressure, via decreased endothelium mediated relaxation and vascular compliance. Conversely in hyperthyroidism, there is a decrease in peripheral vascular resistance, increased blood volume, and increased venous return. These changes may lead to development of heart failure secondary to a high level of output. A cohort study analyzing 2,225 patients throughout 5 years revealed that abnormal thyroid function in patients with symptomatic heart failure and EF < 35% was associated with an increased risk of death. It was demonstrated that abnormal thyroid status was associated with increased risk of mortality; hypothyroid and hyperthyroid states were associated with 58% and 85% increases in relative risk of death compared to euthyroid state. Guidelines for heart failure produced by the American College of Cardiology and the American Heart Association support conducting thyroid function tests in patients with heart failure to determine if thyroid dysfunction may be a primary contributor to heart failure. Anti-thyroid medications are often indicated for the management of thyroid function; however, it may require several weeks to induce a euthyroid state. Definitive treatment with radioactive iodine ablation or thyroidectomy may also be considered to rapidly recover cardiac function. Further research is warranted to elucidate the long-term implications of a hyperthyroid state on the cardiovascular system, as well as to clarify the mechanism for the demonstrated effects, whether indirectly via the vascular system, directly by atrial fibrillation or tachyarrhythmia, or via myocyte remodeling and dysfunction.