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IMPACT OF THYROID DISORDER ON CARDIOVASCULAR DISEASE

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Background: The thyroid gland is a primary regulator of human hemostasis and exerts a profound modulating effect on the cardiovascular system. Thyroid hormones, specifically triiodothyronine (T3), possess positive inotropic and chronotropic properties, regulating pacemaker activity and decreasing peripheral vascular resistance. Dysregulation of these pathways is closely associated with increased cardiovascular risk, including ischemic heart disease, heart failure, and arrhythmias. While hypothyroidism is traditionally linked to atherosclerosis, hyperthyroidism presents a more immediate risk of atrial fibrillation and high-output heart failure.

Pathophysiology of Hyperthyroid Cardiomyopathy: In the early stages of hyperthyroidism, increased cardiac output and left ventricular hypertrophy are commonly observed. However, long-standing thyrotoxicosis paradoxically exhausts myocardial contractile reserve, leading to biventricular dilatation and congestive heart failure. High levels of thyroid hormones disrupt cellular metabolism and over-activate the renin-angiotensin-aldosterone system, resulting in sodium and water retention, pulmonary edema, and hepatic congestion. Approximately 1% of patients develop thyrotoxic cardiomyopathy, a potentially lethal manifestation of the disease.

Clinical Manifestations and Comorbidities: Key clinical signs include dyspnea on exertion, tachycardia, and atrial fibrillation, the latter affecting 5% to 20% of patients. Recent evidence also indicates that pulmonary artery hypertension occurs in 35%–47% of hyperthyroid individuals. Data from a cross-sectional study of patients aged 51–60 years demonstrate significant comorbidities, including diabetes mellitus (38%), myocardial infarction (37%), and hypertension (36%), highlighting the systemic burden of the disease.

Evidence from Meta-Analysis: Systematic reviews indicate that overt hyperthyroidism is associated with a 20% increase in cardiovascular mortality. Data from the Framingham Heart Study and the Cardiovascular Health Study reveal that a Thyroid-Stimulating Hormone (TSH) level of 0.1 mIU/L or lower correlates with a 3.3-fold higher risk of developing atrial fibrillation. Even in patients aged 65 and older with normal free thyroxine (T4) levels, a low TSH (<0.45 mIU/L) doubles the risk of arrhythmic events.

Conclusion**:** Hyperthyroidism induces a progressive transition from high cardiac output to biventricular dilatation and heart failure. The high prevalence of atrial fibrillation and pulmonary artery hypertension contributes to increased morbidity. However, cardiovascular complications are often reversible through the timely correction of the underlying thyroid dysfunction. Early screening and effective management of hyperthyroidism are essential to prevent the progression to irreversible thyrotoxic cardiomyopathy.