Perspective Article

Hypothyroidism and the Heart

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The association of thyroid hormone deficiency and atherosclerosis was recognized by Theodore Kocher in patients who had undergone total thyroidectomy.¹ The prevalence of thyroid deficiency is about 15% in adult females and little less in adult males.² During the evaluation of patients with suspected ischemic heart disease, seldom is thyroid function test included. Apart from its direct modulation of the cardiovascular system, hypothyroidism synergistically acts with some of the classic risk factors like dyslipidemia, hypertension, smoking and homocysteinemia, accelerating the progress of ischemic heart disease.²

The effects of thyroid hormone on the heart and vasculature are primarily mediated by triiodothyronine (T₃) through numerous positive and negatively regulated processes. The hormone acts directly on the heart through genomic and non-genomic reactions, involving several cardiac proteins, ionic channels and enzymes. The final outcome is a reduction in systemic vascular resistance, an increase in the heart rate, increased contractility of the heart and a secondary increase in the blood volume.³

In hypothyroidism, the direct cardiovascular effects are bradycardia, diastolic hypertension, increased systemic vascular resistance, reduced contractility, and increased atherosclerosis and coronary artery disease. It has been estimated that the risk posed by hypothyroidism maybe equal to that of the traditional risk factors.²

Overt hypothyroidism results in increased serum lipid levels, increased cholesterol, low density lipoproteins and apolipoprotein B. The suggested mechanism is the reduced clearance of LDL due to reduced receptors and receptor activity. T₃ mediated catabolism of cholesterol by cholesterol 7 alpha hydroxylase is also muted.^{1,4} Several studies have shown that the increased risk of atherosclerosis in patients with overt hypothyroidism was independent of the other traditional risk factors like serum lipid levels and body mass index (BMI).⁴ Conflicting results have been observed in patients with subclinical hypothyroidism.¹

Apart from its direct action on the lipid metabolism and accelerated atherosclerosis, hypothyroidism

may have an additive effect on traditional risk factors such as hypertension, smoking and insulin resistance. Several newer risk factors for cardiovascular disease have been identified such as homocysteinemia, elevated C-reactive protein (CRP) levels, coagulation abnormalities and insulin resistance. In overt hypothyroidism there are elevated homocysteine and CRP levels and treatment of hypothyroidism resulted in normalization of their levels. In severe hypothyroidism, patients are predisposed to thrombosis due to high D-dimer levels, low alpha antiplasmin, low tissue plasminogen and lower tissue plasminogen activator and inhibitor antigens.¹

Treating a patient for hypothyroidism becomes a catch-22 situation. Theoretically, Thyroxine in a patient with ischemic heart disease can precipitate arrhythmias, worsen angina and exacerbate heart failure. Not treating it will worsen the hyperlipidemia and cause accelerated atherosclerosis. There is profound vasoconstriction in the overt hypothyroid state, which improves with replacement therapy. In a hypothyroid state the myocardial fibers are elongated with resultant enlarged dimensions and wall stress, which can also be mitigated with replacement. It is thought that even in the face of ischemic heart disease overall improvement in cardiac function will result from thyroid replacement. It would be reasonable to first proceed with revascularisation procedures before replacement in severely symptomatic patients with critical coronary disease. In patients with stable coronary disease, thyroid hormone replacement can be started at a lower dose and gradually titrated to achieve a normal TSH value.¹ Some of the complications following cardiac surgery in a hypothyroid patient are congestive heart failure, slow recovery from anesthesia, lower incidence of febrile response and gastrointestinal symptoms.

Another subgroup is the patients with subclinical hypothyroidism who are generally asymptomatic. Though a TSH value of >10 mIU/mL warrants levothyroxine supplementation, patients with milder form of subclinical disease may also benefit from supplementation if they have associated cardiovascular disease or deranged lipid profile.^{4,5}

Hypothyroidism is a common disorder that affects women more commonly than men. Accelerated atherosclerosis occurs in these patients and acts synergistically with other traditional risk factors for coronary artery disease. Management of these patients will present a challenge to the medical professionals involved in their care.

References

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