Impact of Thyroid Status on Prognosis of Acute Coronary Syndrome – an editorial

H N Sarker[a]

Acute coronary syndrome (ACS) remains a major cause of global mortality despite advances in early diagnosis, reperfusion therapy, and secondary prevention. Increasing evidence suggests that non-cardiac comorbidities substantially influence outcomes after ACS, yet endocrine factors, particularly thyroid dysfunction are often overlooked in routine cardiovascular risk assessment. Accumulating data indicate that thyroid status at the time of ACS has important prognostic implications and may offer incremental risk stratification beyond conventional predictors.

Thyroid hormones play a central role in cardiovascular homeostasis, regulating myocardial contractility, heart rate, vascular tone, lipid metabolism, and endothelial function. Both excess and deficiency of thyroid hormones can adversely affect coronary perfusion and myocardial oxygen balance, thereby modulating the clinical course of ACS. Even subtle alterations within the so-called "subclinical" range may carry prognostic significance in patients with established coronary artery disease [1].

Hypothyroidism is associated with accelerated atherosclerosis through dyslipidaemia, increased systemic vascular resistance, endothelial dysfunction, and low-grade inflammation. In patients presenting with ACS, overt hypothyroidism has been linked to impaired left ventricular systolic function, higher rates of heart failure, and increased long-term mortality [2]. Subclinical hypothyroidism, which is far more prevalent, has also been consistently associated with adverse cardiovascular outcomes, particularly when thyroid-stimulating hormone (TSH) concentrations exceed 10 mIU/L [3]. These findings suggest that even mild thyroid hormone deficiency may hinder myocardial recovery and vascular repair following plaque rupture.

In contrast, hyperthyroidism increases myocardial oxygen demand, promotes coronary vasoconstriction, and predisposes to malignant arrhythmias. Suppressed TSH levels reflecting overt or subclinical hyperthyroidism have been associated with higher short-term mortality and arrhythmic complications following ACS, although data are less robust and sometimes inconsistent compared with hypothyroid states [4]. The pro-arrhythmic milieu of thyrotoxicosis, particularly the high incidence of atrial fibrillation, may further complicate haemodynamic stability and worsen outcomes in the acute phase.

A particularly important and frequently observed abnormality in ACS is non-thyroidal illness syndrome (NTIS), also known as low T3 syndrome. This condition characterised by reduced circulating triiodothyronine (T3) with normal or low TSH, reflects altered peripheral thyroid hormone metabolism in response to severe illness. NTIS is highly prevalent in patients with ACS and has been strongly associated with infarct size, reduced left ventricular ejection fraction, cardiogenic shock, and both in-hospital and long-term mortality [5]. Importantly, low T3 levels appear to predict adverse outcomes independently of traditional risk factors and established prognostic scores, highlighting their potential role as a biomarker of disease severity [6].

Despite compelling observational evidence linking thyroid dysfunction to poor prognosis in ACS, major uncertainties remain. It is unclear whether thyroid abnormalities directly contribute to worse outcomes or merely reflect the severity of the underlying illness. Randomised controlled trials evaluating thyroid hormone replacement in subclinical hypothyroidism or NTIS in the context of ACS are notably lacking. Consequently, current guidelines do not recommend routine treatment of mild thyroid dysfunction detected during acute cardiac illness, although selective screening is increasingly advocated for prognostic assessment and longer-term management [7].

In summary, thyroid status exerts a significant influence on the prognosis of patients with ACS. Overt and subclinical thyroid dysfunctions, as well as low T3 syndrome, are consistently associated with increased mortality and cardiovascular complications. Routine assessment of thyroid function may improve risk stratification and deepen understanding of the complex interplay between endocrine and cardiovascular disease. Well-designed interventional trials are urgently needed to determine whether targeted correction of thyroid abnormalities can translate into improved outcomes for patients with ACS.

[a] *Professor (Ex), Medicine, Sher-E-Bangla Medical College, Barishal, and Sheikh Sayera Khatun (ORRCID: 0000-0001-6523-9395)*

Received: 14 Jan 2026
Published Online: 16 Feb 2026
DOI: 10.5281/zenodo.18654489

Volume: 8, Number: 2, Page: 1-1
e-ISSN: 2789-5912
ISSN: 2617-0817

References

1. Klein I, Danzi S. Thyroid disease and the heart. Lancet. 2007;370:192–203.
2. Jabbar A, Pingitore A, Pearce SHS, et al. Thyroid hormones and cardiovascular disease. Nat Rev Cardiol. 2017;14:39–55.
3. Rodondi N, den Elzen WPJ, Bauer DC, et al. Subclinical hypothyroidism and the risk of coronary heart disease and mortality. JAMA. 2010;304:1365–1374.
4. Collet TH, Gussekloo J, Bauer DC, et al. Subclinical hyperthyroidism and the risk of coronary heart disease and mortality. Arch Intern Med. 2012;172:799–809.
5. Iervasi G, Pingitore A, Landi P, et al. Low-T3 syndrome: a strong prognostic predictor of death in patients with heart disease. Circulation. 2003;107:708–713.
6. Friberg L, Drvota V, Bjelak AH, et al. Association between increased levels of reverse triiodothyronine and mortality after acute myocardial infarction. Am J Med. 2001;111:699–703.
7. Biondi B, Cooper DS. The clinical significance of subclinical thyroid dysfunction. Endocr Rev. 2008;29:76–131.