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Exercise-Induced Cardiac Troponin Elevation in Endurance Athletes: Distinguishing Physiological Release from Myocardial Injury – A Narrative Review

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Abstract

Introduction and purpose. Cardiac troponins are central to the diagnosis of myocardial injury, yet transient concentrations above the assay-specific 99th percentile are common after prolonged exercise. This creates a practical problem when an endurance athlete presents after competition with chest symptoms or an unexpectedly elevated high-sensitivity cardiac troponin (hs-cTn). The purpose of this narrative review is to examine the prevalence, kinetics, mechanisms and clinical significance of exercise-associated cTn release and to propose a clinically oriented framework for distinguishing an expected post-exercise response from potentially important myocardial injury.

Materials and methods. A narrative review of PubMed/MEDLINE-indexed literature was undertaken, with targeted searches for cardiac troponin, high-sensitivity troponin, exercise, endurance sport, marathon, triathlon, kinetics, myocardial injury and athlete. Priority was given to systematic reviews, meta-analyses, consensus definitions, serial biomarker studies, prognostic cohorts and representative endurance-event studies. Literature available through August 2026 was considered. This was a narrative review; study selection was not performed according to a systematic-review protocol and no formal meta-analysis or risk-of-bias assessment was undertaken.

Results. Exercise-associated cTn elevation is common and highly assay- and context-dependent. A 2026 meta-analysis of 129 studies and 7,289 athletes found post-exercise values above the upper reference limit in 36% overall, with higher prevalence using high-sensitivity assays, cTnT rather than cTnI, and in endurance disciplines. Exercise-associated cTn release typically shows an early post-exercise peak followed by a decline during recovery; the exact time course varies with the assay, sampling schedule and exercise exposure. However, post-exercise cTn elevation cannot be assumed to be benign in every athlete. Symptoms, ECG findings, serial kinetics, cardiovascular risk, imaging and the possibility of myocarditis, acute coronary syndrome, pulmonary embolism or other causes of myocardial injury remain decisive.

Conclusion. An elevated hs-cTn after endurance exercise should be interpreted as a clinical finding rather than as an isolated laboratory abnormality. An early post-exercise rise followed by a clear decline in an asymptomatic athlete with a reassuring clinical assessment is compatible with exercise-associated release. Persistent or rising concentrations, ischemic symptoms, arrhythmia, hemodynamic compromise or abnormal imaging should prompt evaluation for pathological myocardial injury. Serial assessment and clinical context are more informative than a single post-race value.

Keywords: cardiac troponin, endurance exercise, athletes, myocardial injury, high-sensitivity troponin, marathon, sports cardiology

1. Introduction

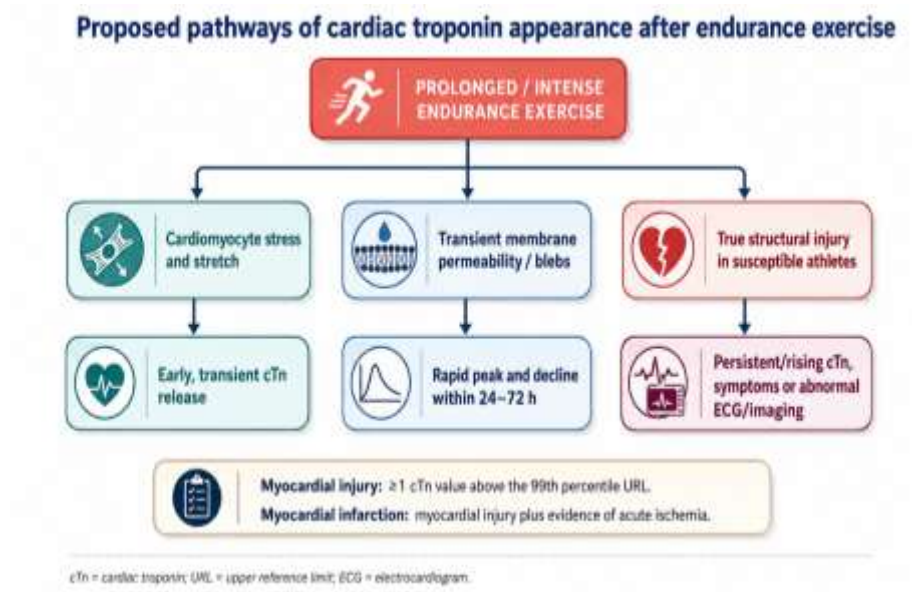
Cardiac troponin I (cTnI) and cardiac troponin T (cTnT) are the preferred biomarkers for detecting myocardial injury. The Fourth Universal Definition of Myocardial Infarction defines myocardial injury by at least one cTn concentration above the assay-specific 99th percentile upper reference limit (URL), while myocardial infarction additionally requires evidence of acute myocardial ischemia [1]. This distinction is particularly important in sports medicine because strenuous exercise can produce a measurable rise in cTn in otherwise healthy athletes. Increased cTn concentrations after strenuous exercise are well documented. Earlier endurance studies reported detectable or elevated cTn after marathons, triathlons and cycling events, and the introduction of high-sensitivity assays made post-exercise elevations even more apparent [2-4]. The largest contemporary synthesis, published in 2026, included 129 studies and 7,289 athletes and found cTn values above the URL in 36% overall. The prevalence was 58% with high-sensitivity assays, 45% for cTnT, and was particularly high in endurance sports lasting approximately three to six hours [5].

What is less straightforward is the interpretation of an elevated result in an individual athlete. On one hand, most post-exercise cTn elevations in healthy athletes are transient and occur without evidence of irreversible myocardial injury. On the other, exercise does not protect an athlete from acute coronary syndrome, myocarditis, pulmonary embolism, arrhythmia or other causes of myocardial injury. Moreover, recent prognostic observations have challenged the assumption that every exercise-associated troponin rise is clinically irrelevant [6].

For the clinician, the relevant question is how much weight should be given to the exercise exposure when an increased cTn concentration is found. This review focuses on endurance exercise and addresses prevalence, biological mechanisms, post-exercise kinetics, modifying factors, prognostic evidence and a practical clinical approach to distinguishing a likely physiological response from clinically relevant myocardial injury.

Accordingly, this review takes a clinical rather than purely biomarker-based approach. Particular attention is paid to the circumstances that alter diagnostic reasoning: symptoms after an endurance event, unexpected serial kinetics, ECG abnormalities, cardiovascular risk and indications for targeted imaging.

Figure 1. Proposed mechanisms of cardiac troponin appearance in the circulation after endurance exercise. The model distinguishes transient exercise-associated release from structural myocardial injury; the pathways are not mutually exclusive.



Source: Authors' own elaboration based on the reviewed literature.

2. Cardiac troponin: what an elevated value actually means

Troponin is a regulatory protein complex involved in excitation–contraction coupling. The cardiac-specific isoforms cTnI and cTnT are highly useful markers of cardiomyocyte injury, but an elevated concentration is not synonymous with myocardial infarction. Acute and chronic non-ischemic conditions can raise cTn, including myocarditis, tachyarrhythmia, heart failure, pulmonary embolism, renal disease and critical illness [1].

The clinical interpretation of hs-cTn therefore requires more than comparison with a single numerical threshold. Assay-specific URLs differ, and reference limits can vary with sex, age and the population used to derive the 99th percentile [7]. In an athlete tested soon after competition, the timing of sampling relative to exercise becomes an additional variable that is rarely relevant in conventional resting populations.

High-sensitivity assays detect very low circulating concentrations and identify small changes that older assays would have classified as negative. This improves sensitivity for acute myocardial injury but reduces the specificity of an isolated post-exercise measurement. A value above the URL after a marathon may represent an expected exercise-associated response, a pre-existing elevation, or a genuinely pathological acute process. Serial change and clinical evidence are therefore essential.

3. How common is exercise-induced troponin elevation?

The reported prevalence varies widely because studies differ in exercise mode, duration, participant characteristics, sampling time and assay generation. An early meta-analysis of cTnT found detectable post-exercise concentrations in approximately 47% of endurance athletes, with higher detection after running than cycling [3]. Later studies using high-sensitivity assays reported values above clinical cut-offs in substantial proportions of marathon runners [8].

The 2026 systematic review and meta-analysis by Stekelenburg and colleagues provides the most comprehensive estimate to date. Across 129 studies, 36% of athletes exceeded the assay-specific URL after exercise, but individual study estimates ranged from 2% to 98%. High-sensitivity assays identified elevations more frequently than conventional assays (58% vs 26%), and cTnT assays more frequently than cTnI assays (45% vs 30%). Triathlon, cycling and running had among the highest prevalence estimates, while walking and contact sports had much lower values [5].

The relationship with duration was non-linear. The highest pooled prevalence occurred after approximately three to six hours of exercise, rather than increasing indefinitely with event duration [5]. This observation matters clinically because the magnitude of post-exercise cTn release should not be interpreted simply as a dose-response marker of 'cardiac damage'. Shorter but relatively intense events can also produce substantial elevations.

Table 1. Factors associated with post-exercise cardiac troponin elevation

Factor	Observed association	Clinical implication
Assay sensitivity	High-sensitivity assays detect post-exercise elevations more often [6].	Always interpret against the assay-specific URL and analytical characteristics.
Troponin isoform	cTnT elevations are reported more frequently than cTnI in pooled data [6].	Results from different assays are not directly interchangeable.
Exercise modality	Endurance disciplines show the highest prevalence [3,6].	Running, cycling and triathlon provide a plausible exercise context for transient elevation.
Exercise duration	Highest pooled prevalence around 3–6 h [6].	Longer duration does not imply progressively greater injury.

Relative intensity	Higher relative exercise intensity has been associated with greater post-race hs-cTnT [11].	Athlete-specific workload may matter more than absolute pace.
Age/training history	Associations are inconsistent across studies [6,12,13].	Do not use age or training status alone to label an elevation benign.

4. Kinetics: one of the most useful discriminators

The time course of exercise-associated release is central to interpretation. In many athletes, cTn begins to rise during or shortly after exercise, typically peaks within the first several hours and then declines; normalization is often seen within 24–72 hours, although timing varies with assay and exercise exposure [4, 9]. A meta-analysis of hs-cTnT kinetics found clear elevations from approximately 0.7 to 25 hours after exercise, with the modeled peak occurring early after completion [4].

This pattern differs conceptually from the sustained release that can accompany larger areas of cardiomyocyte necrosis. The distinction is not absolute—acute myocardial infarction can also show an early peak and decline, and timing of presentation matters—but persistence or continued rise beyond the expected post-exercise window should lower the threshold for further investigation.

Sampling immediately at the finish line can underestimate the eventual peak. Conversely, a single value obtained several hours after an event may appear alarming if the clinician is unaware that exercise itself can produce concentrations above the conventional URL. Serial sampling is therefore more informative than a single measurement in a clinically stable athlete.

5. Mechanisms of post-exercise troponin release

The biological source of circulating cTn after exercise remains debated. The traditional interpretation of troponin release as direct evidence of irreversible cardiomyocyte necrosis is difficult to reconcile with the rapid normalization and absence of structural abnormalities in many athletes. Proposed explanations include transient cardiomyocyte membrane permeability, release from a small cytosolic pool, membrane blebbing and exercise-related mechanical or metabolic stress [9].

A reversible mechanism is attractive because it explains an early rise and rapid decline without persistent ventricular dysfunction. Nevertheless, it is unlikely that a single mechanism explains

all athletes. Very prolonged exercise can produce substantial hemodynamic stress, transient right-ventricular dysfunction and changes in loading conditions, and susceptible individuals may have underlying coronary atherosclerosis, myocardial fibrosis or occult cardiomyopathy. Renal clearance, plasma-volume changes and hydration have also been proposed as modifiers. However, marathon studies have not consistently shown hydration status to be an independent determinant of cTn release [8]. Overall, the available evidence favors a multifactorial model in which exercise dose, individual susceptibility, assay characteristics and transient cardiomyocyte stress interact.

6. cTnI versus cTnT and assay-related problems

The distinction between cTnI and cTnT is clinically relevant because different assays have different analytical properties and URLs. Pooled data indicate that post-exercise elevations above the URL are more frequently detected with cTnT than cTnI [5]. This does not necessarily mean that one biomarker reflects more myocardial injury; it may reflect assay sensitivity, biological release and clearance, or differences in reference thresholds.

The URL itself is not a universal constant. Sex-specific thresholds are used for some assays, and age influences baseline hs-cTn distributions [7]. New data in healthy collegiate athletes also reinforce that baseline values in athletic populations require careful interpretation rather than automatic application of assumptions derived from older clinical cohorts [10].

For research, reporting the exact assay, manufacturer, URL, sampling times and proportion exceeding the URL is essential. For clinical practice, the same principle applies: a laboratory value should never be interpreted without knowing which hs-cTn assay generated it.

7. Marathon and road-running evidence

Marathon running has generated the largest body of evidence. In a cohort of 92 marathon participants, cTnI rose from 14 ± 12 ng/L before the race to 94 ± 102 ng/L afterward, and 69% exceeded the study's clinical cut-off. Younger age and longer exercise duration were associated with greater release, whereas hydration status and exercise intensity were not independent predictors in that analysis [8].

Other work has linked immediate and 24-hour hs-cTnT concentrations with relative exercise intensity [11]. Training history may also influence the response, although findings across studies are not fully consistent [12]. A systematic review and meta-regression of amateur running studies confirmed significant post-race increases in both cTnI and cTnT and emphasized heterogeneity related to participant and race characteristics [13].

After a marathon, cTn concentrations can overlap values that would trigger investigation in a resting emergency-department patient. The exercise context changes pre-test probability, but it does not remove the need to evaluate symptoms and ECG findings.

8. Triathlon, cycling and other sports

Troponin release is not unique to running. The 2026 pooled analysis found high prevalence after triathlon and cycling, and earlier literature documented elevations after ultradistance events [2, 5]. Cycling may produce lower rates than running in some datasets, possibly because of differences in eccentric loading, relative intensity, event characteristics or participant selection [3].

Intermittent sports can also produce measurable cTn release. A serial study of elite, amateur and junior basketball players found a post-match rise in cTnI, with 26% exceeding the URL [14]. This observation is important because it shows that the phenomenon is not restricted to multi-hour endurance races. Nevertheless, endurance events remain the setting in which large post-exercise elevations are most frequently encountered clinically.

Table 2. Features favoring exercise-associated release versus pathological myocardial injury

Feature	More compatible with exercise-associated release	Raises concern for pathology
Symptoms	No symptoms or expected transient post-race fatigue	Ischemic chest pain, disproportionate dyspnea, syncope, persistent palpitations
ECG	Normal or athlete-appropriate pattern	New ischemic ST-T changes, pathologic Q waves, significant ventricular arrhythmia
Troponin kinetics	Early rise followed by a clear decline, interpreted together with symptoms, ECG and examination	Continued rise, delayed/unexpected trajectory, or persistent elevation with concerning clinical findings
Hemodynamics	Stable	Hypotension, pulmonary edema, persistent tachycardia unexplained by recovery

Imaging	Normal ventricular function and no focal injury	Regional wall-motion abnormality, edema/LGE on CMR, new ventricular dysfunction
Clinical context	Recent strenuous exercise with plausible timing	Viral prodrome, thromboembolic risk, known CAD, strong family history, exertional collapse

9. When elevated troponin is not 'just exercise'

A recent endurance event may explain an elevated cTn concentration, but it does not remove the need to investigate a symptomatic athlete. The universal definition requires clinical evidence of ischemia before diagnosing myocardial infarction, but cTn elevation still establishes myocardial injury and demands an explanation [1]. An endurance athlete can develop plaque rupture, supply–demand mismatch, spontaneous coronary artery dissection, myocarditis or pulmonary embolism.

Myocarditis deserves particular attention because young athletes may present with chest pain, arrhythmia or reduced exercise tolerance after a viral illness. Troponin can be elevated, and cardiovascular magnetic resonance may demonstrate edema and non-ischemic late gadolinium enhancement. In this setting, recent exercise is a confounder rather than an alternative diagnosis.

Masters athletes create a different problem. They may have decades of training exposure but also age-related coronary atherosclerosis. Reviews of long-term intensive endurance exercise describe a subgroup with higher coronary calcium, atrial arrhythmias or myocardial fibrosis despite the overall mortality benefit of physical activity [15]. Thus, a post-race cTn elevation in a 55-year-old athlete with chest pressure and coronary risk factors should be approached differently from the same numerical value in an asymptomatic 20-year-old after competition.

10. Prognostic significance: still unresolved

For many years, transient post-exercise cTn release was treated as a benign laboratory phenomenon. Long-term data are more nuanced. In recreational marathon runners, coronary atherosclerosis burden rather than transient post-marathon hs-cTnI elevation predicted long-term events in one cohort [16]. This finding supports the view that the troponin rise itself may not represent irreversible injury in most healthy runners.

However, another prospective study of older long-distance walkers found that post-exercise cTnI above the URL was associated with a higher risk of mortality and cardiovascular events during follow-up [6]. These populations and exercise exposures differ, so the results should not be treated as contradictory. Instead, they suggest that post-exercise cTn may have different meaning depending on age, baseline cardiovascular risk and underlying disease.

Whether an exaggerated exercise-induced cTn response identifies a susceptible cardiovascular phenotype, or simply accompanies other risk factors, remains uncertain. Current evidence does not support routine post-exercise troponin screening of asymptomatic athletes outside a research or otherwise clinically justified setting.

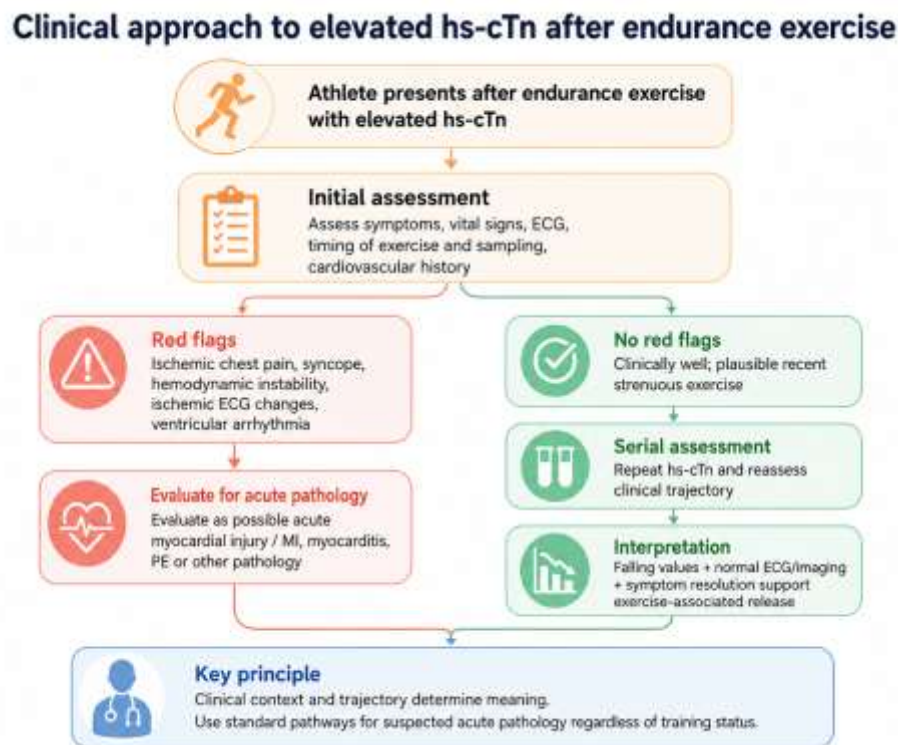
11. Clinical assessment of the athlete

The reason for ordering troponin is an important starting point for interpretation. Routine testing of asymptomatic finishers is generally unhelpful because a substantial proportion will have an abnormal result without actionable disease. Troponin becomes clinically meaningful when it is ordered because of chest pain, dyspnea, syncope, arrhythmia, abnormal vital signs or another concern.

The first assessment should document the exercise exposure, exact time of completion, time of blood sampling, symptoms during and after exercise, recent infection, medications or supplements, cardiovascular history and family history of premature sudden death or cardiomyopathy. A 12-lead ECG and repeated clinical examination are central. Echocardiography is appropriate when symptoms, ECG or hemodynamics suggest structural or functional abnormality.

Serial hs-cTn should be interpreted alongside the clinical trajectory. A falling value in a symptom-free athlete with a normal ECG and reassuring examination supports exercise-associated release. Persistent or increasing cTn, recurrent chest pain, new ECG abnormalities or ventricular dysfunction should prompt investigation according to the suspected pathology rather than according to the athlete's training status. In an asymptomatic athlete with persistently discordant troponin elevation despite otherwise reassuring evaluation, analytical interference or a troponin macrocomplex is an additional, uncommon diagnostic consideration [17].

Figure 2. Proposed clinical approach to an endurance athlete with elevated high-sensitivity cardiac troponin after exercise



Source: Authors' own elaboration based on the reviewed literature.

12. Role of echocardiography, CMR and coronary imaging

Echocardiography is accessible and can identify regional wall-motion abnormalities, ventricular dysfunction, pericardial effusion or alternative structural disease. A normal echocardiogram does not exclude myocarditis or coronary disease, but it substantially changes the immediate risk assessment in a stable athlete.

Cardiovascular magnetic resonance is particularly valuable when myocarditis or non-ischemic myocardial injury is suspected. Tissue characterization can identify myocardial edema and fibrosis patterns that are not visible on routine echocardiography. Coronary CT angiography or invasive angiography may be required when the presentation suggests coronary disease, especially in masters athletes or those with ischemic symptoms.

Imaging should not be triggered by the troponin number alone. The decision should integrate symptoms, serial biomarker behavior, ECG, pre-test probability and exercise context. This avoids both over-investigation of expected post-race elevations and under-investigation of genuine disease.

13. Return to exercise

There is no need to restrict an asymptomatic athlete solely because a research or incidental post-event sample showed a transient cTn rise that normalized and was accompanied by a reassuring clinical assessment. Conversely, athletes with suspected myocarditis, acute coronary syndrome, significant arrhythmia or structural heart disease require diagnosis-specific return-to-sport pathways.

When the presentation falls into a grey zone, a conservative approach is appropriate: confirm symptom resolution, document normalization or a convincing downward trajectory of cTn, reassess ECG findings and complete indicated imaging before return to high-intensity training. The athlete's age, sport, competition level and cardiovascular risk should influence the threshold for further testing.

14. Current controversies and knowledge gaps

Despite extensive study, the mechanism responsible for exercise-associated cTn release has not been fully resolved. Rapid kinetics support reversible release in many athletes, but direct evidence separating membrane leakage from microscopic structural injury remains incomplete. A single mechanism is unlikely to account for the wide inter-individual variability.

An additional difficulty is the absence of a universally accepted post-exercise reference range for athletes. Creating such a range is challenging because values depend on assay, sex, age, exercise modality, intensity, duration and sampling time. A threshold that labels a value 'normal for exercise' could also be unsafe if it discouraged evaluation of a symptomatic athlete.

The evidence base also remains uneven across demographic groups. Sex can influence hs-cTn reference limits, while many exercise studies include relatively small and selected athletic cohorts; this limits generalizability across athlete populations [7, 18].

15. Complementary evidence across exercise models

Several older and contemporary studies broaden the evidence beyond the main marathon cohorts. Early work documented exercise-associated cTn changes alongside prothrombotic markers, while a major state-of-the-art review summarized the mechanistic and clinical uncertainty surrounding exercise-induced cTn release [19]. Meta-analytic data also show modest, transient changes in left-ventricular function after prolonged exercise, whereas detailed endurance-race studies suggest that the right ventricle may be particularly sensitive to acute volume and pressure stress [20].

Boston Marathon cohorts demonstrated that biomarker release can coincide with transient echocardiographic abnormalities and is more pronounced in less-trained runners [21-22]. Ultra-endurance evidence from the Hawaii Ironman similarly showed that cTn release may occur together with transient changes in ventricular function [23]. These observations should not be equated with permanent injury, but they explain why biomarker findings are best interpreted together with symptoms and imaging.

Age and exercise dose further complicate interpretation. Controlled studies in adolescent runners show that both intensity and duration can influence cTnT release [24]. Conversely, cTnI can rise after relatively short, high-intensity exercise [25], and intermittent exercise bouts can also alter cardiac biomarkers. Thus, prolonged endurance exercise is not the only setting in which a transient troponin rise occurs.

Recent work continues to refine this picture. Serial cTn patterns have been characterized in athletes completing an extreme triathlon [26], and a 2026 clinical review emphasizes the importance of kinetics, symptoms and multimodal assessment rather than isolated thresholds [27]. The broader athlete-heart literature likewise frames acute biomarker release within the continuum of physiological adaptation and exercise-related cardiac stress [28-29]. Chronic training produces well-established structural and mechanical adaptation, while small CMR studies in veteran endurance athletes have reported focal fibrosis in a subset of highly exposed individuals [30]. These chronic observations are relevant to risk stratification but do not demonstrate that transient post-exercise cTn release itself causes fibrosis.

Less is known about the significance of repeated cTn release across many years of endurance training. Contemporary reviews of lifelong intensive exercise acknowledge potential adverse remodeling in susceptible individuals while emphasizing that high physical activity remains associated with lower cardiovascular and all-cause mortality at the population level [15]. The biomarker should therefore not be used to portray endurance exercise itself as inherently cardiotoxic.

16. Strengths and limitations of the evidence

The evidence base is now large enough to establish that post-exercise cTn elevation is common, reproducible and strongly influenced by assay and exercise context. Recent meta-analyses provide more stable prevalence and kinetic estimates than early small cohorts [4-5, 13].

Important limitations remain. Many studies are observational, use heterogeneous exercise protocols and collect blood at different time points. Clinical cut-offs have changed with successive assay generations. Most studies were designed to describe biomarker behavior

rather than determine hard cardiovascular outcomes. Prognostic cohorts are relatively small and involve different age groups and exercise exposures.

This review is narrative rather than systematic. It prioritizes clinically relevant systematic reviews, meta-analyses, consensus definitions and representative primary studies, but it does not provide a formal risk-of-bias assessment or quantitative synthesis.

17. Summary and conclusions

Cardiac troponin elevation after endurance exercise is common and frequently exceeds the same 99th-percentile thresholds used to define myocardial injury in clinical practice. The 2026 evidence synthesis confirms that this phenomenon is especially frequent with high-sensitivity assays and in endurance disciplines. In many healthy athletes, cTn rises early during recovery and subsequently declines; normalization commonly occurs during the following one to three days, although the time course is assay- and protocol-dependent [4].

The presence of recent strenuous exercise, however, is not a diagnostic exemption. An elevated hs-cTn in a symptomatic athlete should be evaluated in the same clinical framework used for other patients, with particular attention to symptoms, ECG findings, hemodynamic status, serial biomarker kinetics and targeted imaging. Persistent or rising cTn, ischemic symptoms, significant arrhythmia or abnormal imaging should not be attributed to exercise without an appropriate diagnostic evaluation.

Taken together, the evidence supports distinguishing a transient post-exercise pattern from a presentation in which the clinical findings suggest pathological myocardial injury. Serial assessment and pre-test probability remain more informative than any isolated post-race troponin concentration.

Declaration of the use of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the author used ChatGPT (OpenAI) to assist with language refinement, improvement of clarity and readability, and manuscript structuring and formatting. After using this tool, the author critically reviewed and edited the content as needed and takes full responsibility for the final content of the manuscript.

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