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MYOCARDIAL INFARCTION IN ATHLETES: EPIDEMIOLOGY, PATHOPHYSIOLOGICAL MECHANISMS, DIAGNOSTIC CHALLENGES, AND PREVENTIVE STRATEGIES

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ABSTRACT

Myocardial infarction (MI) among athletes is an uncommon but clinically significant event that has attracted growing attention because of its potential to cause sudden cardiac death (SCD) and long-term morbidity in an otherwise healthy population. This review synthesizes current literature on the epidemiology, pathophysiology, diagnostic evaluation, and preventive strategies for MI in athletes across age groups. In athletes younger than approximately 35 years, MI is rare and is more often attributable to non-atherosclerotic mechanisms—congenital coronary artery anomalies, spontaneous coronary artery dissection, coronary artery spasm, myocardial bridging, an oxygen supply–demand mismatch related to hypertrophic cardiomyopathy, or performance-enhancing substance use—than to classic atherosclerotic coronary artery disease. In masters athletes, generally older than 35 years, atherosclerotic plaque rupture or erosion, frequently triggered by the acute hemodynamic stress of intense exertion, becomes the predominant mechanism. Preparticipation cardiovascular screening, incorporating structured history-taking, physical examination, and standardized electrocardiographic interpretation criteria, together with the widespread availability of automated external defibrillators and bystander cardiopulmonary resuscitation training, constitutes the principal preventive strategy, although its optimal design and cost-effectiveness remain debated. Following an acute event, individualized, shared-decision-making approaches to return-to-play balance the cardiovascular and psychological benefits of continued exercise against residual arrhythmic and ischemic risk. Continued research into advanced imaging biomarkers, genetic testing, and risk-stratification tools is needed to refine clinical care for this distinctive population.

KEYWORDS

Myocardial Infarction, Athletes, Sudden Cardiac Death, Sports Cardiology, Coronary Artery Anomalies, Preparticipation Screening

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1. Introduction

Regular physical activity is one of the most effective and widely recommended interventions for the primary and secondary prevention of cardiovascular disease, improving lipid profiles, blood pressure, insulin sensitivity, and overall longevity. Paradoxically, vigorous exercise can also act as a trigger for acute cardiovascular events, including myocardial infarction (MI) and sudden cardiac death (SCD), in a small subset of individuals who harbor an underlying, often clinically silent, cardiac abnormality. This phenomenon—commonly referred to as the "exercise paradox"—has become a focus of sports cardiology, particularly as participation in organized and recreational endurance sports continues to expand across all age groups.

Myocardial infarction is defined pathologically as myocardial cell death caused by prolonged ischemia and is diagnosed on the basis of clinical presentation, biochemical markers of myocyte injury, electrocardiographic changes, and imaging findings, or, in fatal cases, at autopsy. In the general population, MI is overwhelmingly a consequence of atherosclerotic coronary artery disease complicated by plaque rupture or erosion and superimposed thrombosis. Among athletes, however, particularly those younger than 35 years, the causative spectrum is broader and includes several non-atherosclerotic entities that are rare outside the context of intense physical exertion. Understanding these distinctive mechanisms is essential for clinicians involved in the cardiovascular care of athletes, from primary care and sports medicine physicians conducting preparticipation evaluations to cardiologists managing survivors of an acute event and advising on return-to-play decisions.

This narrative review aims to (1) summarize the epidemiology of MI and exercise-related SCD in athletes across age groups; (2) describe the principal pathophysiological mechanisms implicated in athletic MI, distinguishing type 1 (plaque-related) from type 2 (supply–demand mismatch) and other non-atherosclerotic mechanisms; (3) outline the diagnostic approach to suspected MI in athletes; (4) discuss preparticipation cardiovascular screening strategies and their role in prevention; and (5) review current recommendations for management, secondary prevention, and return-to-play following an acute event.

2. Methodology

A narrative literature review was conducted using the PubMed/MEDLINE and Google Scholar databases, supplemented by manual screening of reference lists of retrieved articles. Search terms included combinations of "myocardial infarction," "sudden cardiac death," "athletes," "sports cardiology," "coronary artery anomalies," "spontaneous coronary artery dissection," "myocarditis," "preparticipation screening," and "return to play." Original research articles, narrative and systematic reviews, case series, consensus statements, and clinical practice guidelines published in English were considered eligible for inclusion, with priority given to publications from the past two decades and to landmark studies frequently cited in the sports cardiology literature. Case reports were included selectively where they illustrated mechanisms or clinical patterns not otherwise well represented in the literature. Articles unrelated to exercise or athletic populations, or addressing MI exclusively in sedentary or elderly populations without reference to athletes, were excluded. Given the heterogeneity of study designs and populations in this field, a narrative rather than systematic or meta-analytic synthesis was adopted.

3. Results

3.1 Epidemiology of Myocardial Infarction and Sudden Cardiac Death in Athletes

Sudden cardiac death in athletes is a rare event, with incidence estimates in the range of approximately one case per 200,000 young athletes per year, although reported rates vary considerably depending on the population studied, the definition of "athlete," and the surveillance methodology used [1, 2]. The relative contribution of MI to overall SCD burden is strongly age-dependent. In athletes younger than 35 years, the vast majority of sudden deaths are attributable to inherited or congenital cardiac abnormalities—hypertrophic cardiomyopathy, congenital coronary artery anomalies, arrhythmogenic right ventricular cardiomyopathy, dilated cardiomyopathy, myocarditis, and primary electrical disorders such as long QT syndrome, Brugada syndrome, and Wolff–Parkinson–White syndrome—rather than to atherosclerotic coronary artery disease [1, 3, 4]. Coronary artery anomalies have been reported as the second most common cause of SCD in young athletes in United States registries, accounting for roughly 17% of cases, whereas United Kingdom autopsy series have reported a lower prevalence, closer to 5% [2, 5]. By contrast, in athletes older than 35 years, atherosclerotic coronary artery disease predominates, and ischemic events—myocardial ischemia and infarction—are estimated to account for up to 80% of SCD cases in this older cohort [1, 3].

The epidemiological picture is further nuanced by sex and sport type: SCD and exercise-related MI occur disproportionately in male athletes, and certain endurance disciplines, such as marathon and long-distance running, have been specifically studied because of their popularity among masters participants with occult coronary disease [6, 7]. Historical analyses of marathon-related deaths have consistently implicated coronary atherosclerosis as the leading substrate in runners older than 40 years, underscoring the importance of age-stratified risk assessment rather than a uniform approach across the athletic population [6].

3.2 Pathophysiological Mechanisms

3.2.1 Type 1 Myocardial Infarction: Atherosclerotic Plaque Disruption

Type 1 MI results from spontaneous atherosclerotic plaque rupture or erosion with superimposed thrombus formation and is the dominant mechanism in masters athletes, mirroring the pathophysiology observed in the general middle-aged and older population [8]. Intense physical exertion can acutely destabilize a coronary plaque through abrupt increases in blood pressure, coronary shear stress, and catecholamine-driven platelet activation, converting a previously asymptomatic, non-flow-limiting lesion into an unstable, thrombogenic one [9]. Notably, several reported cases of exercise-triggered MI in middle-aged athletes have involved plaques that were only mildly stenotic before rupture, highlighting that traditional stress-test-based risk stratification, which primarily detects flow-limiting stenoses, may fail to identify individuals at risk of plaque-related events during exertion [9].

3.2.2 Type 2 Myocardial Infarction: Oxygen Supply–Demand Mismatch

Type 2 MI, caused by an imbalance between myocardial oxygen supply and demand in the absence of acute plaque rupture, is disproportionately represented among younger athletes [8]. Structural cardiac abnormalities such as hypertrophic cardiomyopathy increase myocardial oxygen demand and can impair coronary microvascular reserve, so that during maximal exertion the hypertrophied myocardium becomes ischemic even without epicardial coronary obstruction [9]. Other contributors to type 2 MI in athletes include severe anemia, hypoxemia, tachyarrhythmias, and profound hypotension, each of which can precipitate a supply–demand mismatch superimposed on a structurally or functionally abnormal coronary circulation [8].

3.2.3 Congenital Coronary Artery Anomalies

Anomalous origin of a coronary artery, particularly an anomalous origin from the wrong (opposite) sinus of Valsalva with an interarterial or intramural course between the aorta and pulmonary artery, is among the most clinically significant causes of exercise-related ischemia and SCD in young athletes [10]. The interarterial course can be compressed during exercise-induced expansion of the great vessels, and an intramural (intramyocardial) segment within the aortic wall may collapse under the pressure gradient generated during strenuous activity, producing transient but potentially fatal myocardial ischemia [10]. The relative risk of SCD has been estimated to be substantially higher during strenuous exercise than at rest in individuals with a malignant anomalous coronary origin, which is why the diagnosis, even when identified incidentally, materially affects decisions about eligibility for competitive sport [2, 10]. Diagnosis relies on a combination of transthoracic echocardiography, coronary computed tomography angiography, cardiac magnetic resonance (CMR) imaging, and, where indicated, provocative stress testing, since the condition is frequently entirely asymptomatic until a catastrophic event occurs and is missed at initial pathological examination in a substantial proportion of fatal cases [5, 10].

3.2.4 Spontaneous Coronary Artery Dissection

Spontaneous coronary artery dissection (SCAD), an intimal tear or intramural hematoma that compromises the arterial lumen without an underlying atherosclerotic plaque, has been described as a cause of MI in young and middle-aged athletes, particularly women [9]. Extreme physical or emotional stress, hormonal factors, and underlying arteriopathies such as fibromuscular dysplasia have all been proposed as contributing factors [9]. Because SCAD can mimic classic atherosclerotic ACS on invasive angiography, awareness of this entity is important to avoid inappropriate percutaneous intervention, which can extend the dissection, and to guide conservative management strategies more suited to this distinct pathology [9].

3.2.5 Myocardial Bridging

Myocardial bridging, in which a segment of an epicardial coronary artery—most often the left anterior descending artery—runs intramurally through the myocardium rather than along the epicardial surface, can cause systolic (and occasionally diastolic) compression of the vessel. During intense exercise, when heart rate and contractility increase and diastolic filling time shortens, bridging can produce clinically relevant ischemia, and case reports have linked deep or long bridges to exertional MI in athletes, although the majority of bridges identified incidentally remain benign throughout life [9].

3.2.6 Coronary Artery Spasm and Vasospastic Mechanisms

Coronary artery spasm, whether occurring in an angiographically normal vessel or superimposed on non-obstructive atherosclerosis, can produce transient but severe reductions in coronary flow sufficient to cause infarction. Exercise-associated catecholamine surges, endothelial dysfunction, and, in some cases, the use of vasoconstrictive substances (including certain stimulants and nicotine products) have been implicated as precipitants of vasospasm in athletes presenting with MI in the absence of significant fixed coronary stenosis [8, 9].

3.2.7 Myocardial Inflammation and Myocarditis as a Substrate

Myocardial inflammation, encompassing myocarditis and inflammatory cardiomyopathy, is an important and increasingly recognized cause of cardiovascular morbidity and SCD in athletes, and although it is not synonymous with MI, it can produce myocardial necrosis, elevated cardiac biomarkers, and regional wall-motion abnormalities that clinically and biochemically overlap with an infarction [11]. Cardiac magnetic resonance imaging has substantially improved the ability to identify myocardial edema and late gadolinium enhancement patterns consistent with inflammation, informing both diagnosis and return-to-play decisions after presumed viral or post-infectious myocarditis [11, 12]. Current European Society of Cardiology (ESC) sports cardiology guidance recommends a period of exercise abstention during the acute inflammatory phase followed by comprehensive reassessment, including repeat CMR, before clearance to resume competitive sport; however, contemporary data indicate that only a minority of athletes recovering from acute myocarditis fully meet the imaging-based criteria for return to play within the initially anticipated timeframe, underscoring the need for individualized, rather than fixed-interval, follow-up [12].

3.2.8 Performance-Enhancing Substances and Drug-Induced Myocardial Infarction

Use of anabolic androgenic steroids, exogenous testosterone, and various stimulants remains an underrecognized but clinically important cause of MI in athletes, including relatively young and otherwise fit individuals [9]. Proposed mechanisms include accelerated atherogenesis through adverse shifts in the lipid profile (reduced high-density lipoprotein and increased low-density lipoprotein cholesterol), direct coronary vasospasm, and a prothrombotic state driven by increased platelet aggregability and altered coagulation factors [8, 9]. Beyond anabolic steroids, a broader range of pharmacological agents—including nonsteroidal anti-inflammatory drugs, certain hormonal therapies, and stimulant compounds—has been associated with myocardial injury through endothelial dysfunction, coronary spasm, or thrombosis, mechanisms that are particularly relevant to athletes who may use over-the-counter analgesics, energy supplements, or performance-enhancing agents without medical supervision [8]. Clinicians evaluating an athlete with MI and no traditional risk factors should routinely inquire about supplement and performance-enhancing substance use as part of the diagnostic evaluation [8, 9].

3.2.9 Commotio Cordis and Traumatic Mechanisms

Although distinct from MI in its mechanism, commotio cordis—SCD resulting from a blunt, non-penetrating chest wall impact during a vulnerable phase of the cardiac cycle, most often seen in baseball, hockey, and lacrosse—is frequently discussed alongside exercise-related cardiac events because it, too, produces sudden collapse in an apparently healthy athlete without underlying structural heart disease [3]. It is included here for completeness because its clinical presentation can initially be difficult to distinguish from an acute ischemic event, particularly in the field setting before diagnostic evaluation is available.

3.3 Age-Related Patterns: Young versus Masters Athletes

The etiological spectrum of athletic MI shifts markedly with age, a distinction with direct implications for screening and diagnostic strategy. In young athletes (typically defined as younger than 35–40 years), MI is rare, frequently occurs in the absence of traditional cardiovascular risk factors or a family history of premature coronary disease, and is disproportionately caused by the non-atherosclerotic mechanisms described above—congenital coronary anomalies, SCAD, myocardial bridging, coronary spasm, hypertrophic cardiomyopathy-related supply–demand mismatch, and substance-induced injury [9]. In masters athletes, generally defined as those older than 35 years participating in organized or recreational sport at a competitive level, atherosclerotic coronary artery disease becomes the leading substrate, and the pathophysiology converges with that of MI in the general aging population, with the added trigger of acute hemodynamic stress from vigorous exertion [1, 6]. This age-related shift underlies international recommendations that preparticipation evaluation strategies—and the specific tests employed—should differ between young and masters cohorts, with greater emphasis on exercise stress testing and cardiovascular risk-factor assessment in the latter group [7].

3.4 Clinical Presentation and Diagnostic Evaluation

Myocardial infarction in athletes can present with classic exertional chest pain, dyspnea, and diaphoresis, but a substantial proportion of cases—particularly those due to congenital coronary anomalies or hypertrophic cardiomyopathy—are preceded by no symptoms whatsoever, and sudden collapse or cardiac arrest may be the first manifestation of underlying disease. When symptoms do occur, they are frequently atypical or minimized by athletes who attribute exertional discomfort to fatigue or muscular strain, contributing to diagnostic delay. Diagnostic evaluation follows the same fundamental principles used in the general population: a compatible clinical history, serial electrocardiography looking for ST-segment and T-wave abnormalities (which must be interpreted cautiously given the frequent presence of training-related ECG changes in athletes), and serial high-sensitivity cardiac troponin measurements, bearing in mind that intense endurance exercise itself can produce transient, mild troponin elevations that must be distinguished from a true infarction pattern based on the trajectory and magnitude of the rise and fall [9]. Echocardiography assesses regional wall-motion abnormalities and overall ventricular function, while coronary computed tomography angiography and invasive coronary angiography remain central to identifying the underlying coronary anatomy and mechanism—atherosclerotic, anomalous, dissected, or bridged [10]. Cardiac magnetic resonance imaging has an increasingly prominent diagnostic role, differentiating ischemic scar from myocarditis-related fibrosis and helping to characterize the pattern and extent of myocardial injury, both features that are directly relevant to return-to-play decision-making [12, 13]. In fatal cases, careful forensic cardiac pathology examination, including systematic assessment of coronary artery origin and course, is essential, since anomalies are missed at initial autopsy in a substantial proportion of cases unless a structured protocol is followed [5].

3.5 Preparticipation Cardiovascular Screening and Prevention

Preparticipation cardiovascular screening aims to identify, before competition, athletes harboring occult cardiac disease that predisposes to exercise-related MI, arrhythmia, or SCD [3, 7]. Screening protocols generally combine a structured personal and family history, physical examination, and, in many programs, a resting 12-lead electrocardiogram [7]. The interpretation of the athlete's ECG has evolved considerably over the past two decades, moving from the original 2010 ESC recommendations through the 2013 "Seattle criteria" and subsequent refinements, each iteration aiming to reduce the false-positive rate—and the associated psychological burden and cost of unnecessary secondary testing—without sacrificing sensitivity for detecting conditions associated with SCD [14]. Comparative studies in elite athlete cohorts have shown that the Seattle criteria reduced the proportion of ECGs classified as abnormal from roughly 17% to about 4%, while still identifying every athlete with an underlying cardiac abnormality in the cohort studied [14].

In masters athletes, the diagnostic value of a standard resting ECG for detecting occult coronary artery disease is limited, and international advisory statements instead emphasize a comprehensive evaluation incorporating cardiovascular risk-factor assessment, symptom-directed history, and, in appropriately selected individuals with risk factors or planned high-intensity or endurance events, exercise stress testing, while acknowledging that universal, systematic screening of all masters participants (for example, at mass-participation marathon events) is often logistically and economically impractical [7]. Beyond individual screening, population-level prevention strategies—widespread placement of automated external defibrillators at sporting venues and training of coaches, athletic staff, and bystanders in cardiopulmonary resuscitation—have been consistently associated with improved survival following sports-related cardiac arrest and are considered complementary, rather than alternative, to preparticipation screening [2, 3].

3.6 Management, Return-to-Play, and Prognosis

Acute management of MI in athletes follows standard evidence-based pathways for acute coronary syndromes, including timely reperfusion therapy where indicated, antiplatelet and anticoagulant therapy, and guideline-directed medical therapy for secondary prevention; however, the underlying mechanism identified on angiography or imaging materially affects subsequent decisions [8]. For example, percutaneous coronary intervention, appropriate for atherosclerotic plaque rupture, may be contraindicated or technically hazardous in SCAD, where conservative management is often preferred, and anomalous coronary origins with a high-risk course frequently require surgical unroofing or reimplantation rather than percutaneous treatment [9, 10]. Cardiac rehabilitation has been identified as a critical determinant of long-term outcomes after MI regardless of patient age, and structured rehabilitation programs should be incorporated into the recovery plan for athletes just as for non-athletic patients [9].

Return-to-play decisions after MI or myocarditis in athletes require an individualized, shared-decision-making approach that weighs the demonstrated cardiovascular and psychosocial benefits of continued sports participation against the residual risk of recurrent ischemia, ventricular arrhythmia, or heart failure [2, 12]. Contemporary guidance has moved away from blanket disqualification toward risk-stratified engagement, informed by the extent of residual myocardial scar (a substrate capable of anchoring reentrant ventricular arrhythmias), left ventricular function, inducible ischemia or arrhythmia on stress testing, and the specific underlying etiology [2]. Notably, the compact fibrotic scar that follows myocardial infarction, while composed of electrically inert, non-excitabile tissue, can nonetheless support reentrant circuits at its border zone, giving rise to sustained ventricular tachycardia—a risk that persists well beyond the acute phase and must be factored into long-term surveillance and return-to-play planning, in addition to the risk of primary ventricular fibrillation occurring within the first hours of ischemia [9]. Encouragingly, available data suggest that young patients who survive MI generally have a favorable overall prognosis, reinforcing the importance of accurate diagnosis, mechanism-specific treatment, and structured follow-up rather than reflexive exclusion from athletic activity [9].

4. Discussion

The literature reviewed here highlights that MI in athletes cannot be understood through a single etiological lens; rather, it represents a heterogeneous group of conditions unified by their occurrence in the specific physiological context of intense exertion. This heterogeneity has direct clinical implications. First, diagnostic evaluation of an athlete presenting with suspected MI—or found to have unexplained myocardial scar on imaging—should systematically consider non-atherosclerotic mechanisms, particularly in younger individuals, rather than defaulting to an atherosclerotic paradigm developed primarily in older, sedentary populations. Second, preparticipation screening strategies must remain age-stratified: ECG-based screening, while valuable in young athletes for detecting inherited arrhythmogenic and structural conditions, has limited utility for the atherosclerotic disease that predominates in masters athletes, where risk-factor-based and, in selected cases, stress-testing-based approaches are more appropriate. Third, the ongoing evolution of ECG interpretation criteria illustrates a broader tension in preventive cardiology between sensitivity and specificity: overly permissive criteria generate a burden of false positives, unnecessary investigation, and potentially unwarranted disqualification from sport, with attendant psychological and career consequences, whereas overly restrictive criteria risk missing individuals truly at risk. This tension is not fully resolved and represents an area of active investigation and, at times, genuine disagreement among experts regarding the optimal balance, the cost-effectiveness of universal ECG screening programs, and their applicability across different countries and athlete populations, including possible differences in ECG patterns among athletes of different ethnic backgrounds [7, 14].

A further theme emerging from this review is the shift, across nearly all domains of sports cardiology, from categorical disqualification toward individualized, shared-decision-making frameworks. This is evident in contemporary approaches to return-to-play after both ischemic events and myocarditis, where imaging-based risk stratification—particularly cardiac magnetic resonance assessment of scar burden and residual inflammation—has begun to replace fixed observation periods [12, 13]. This trend reflects a growing recognition that a diagnosis of cardiac disease, including a history of MI, does not automatically preclude safe return to competitive or recreational sport, provided that risk is carefully characterized and monitored over time [2]. Nonetheless, the evidence base guiding these individualized decisions remains limited by the relative rarity of the underlying conditions, the paucity of large, prospective, athlete-specific cohorts, and the ethical and logistical difficulty of conducting randomized studies in this context. Future research priorities include the development of validated, athlete-specific risk-stratification tools; larger multinational registries capturing long-term outcomes after MI in athletes; further clarification of the role of genetic testing in unexplained cases; and continued refinement of imaging-based biomarkers that might allow more precise, individualized return-to-play recommendations.

5. Conclusions

Myocardial infarction in athletes, though uncommon, spans a distinctive and age-dependent spectrum of mechanisms that differs substantially from MI in the general population. In young athletes, non-atherosclerotic causes—congenital coronary anomalies, spontaneous coronary artery dissection, myocardial bridging, coronary spasm, hypertrophic cardiomyopathy-related ischemia, and substance-induced injury—predominate, whereas atherosclerotic coronary artery disease triggered by acute exertional stress becomes the leading cause in masters athletes. Accurate recognition of the underlying mechanism is essential to guide appropriate acute management, secondary prevention, and return-to-play decisions. Preparticipation cardiovascular screening, evolving ECG interpretation criteria, and widespread availability of automated external defibrillators together form the current backbone of preventive strategy, though considerable uncertainty remains regarding the optimal, most cost-effective design of screening programs. A shift toward individualized, imaging-informed, shared-decision-making approaches to sports eligibility—rather than uniform disqualification—reflects the broader trajectory of sports cardiology and offers a favorable outlook for athletes who experience these events, provided that diagnosis, treatment, and follow-up are tailored to the specific mechanism involved.

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