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CURRENT OPINION

Recommendations for participation in leisure time or competitive sports in athletes-patients with coronary artery disease: a position statement from the Sports Cardiology Section of the European Association of Preventive Cardiology (EAPC)

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Introduction

This article presents an update of earlier recommendations from the Sports Cardiology section of the European Association of Preventive Cardiology (EAPC)[1] on sports-participation in patients with coronary artery disease (CAD), coronary artery anomalies (CAAs), or spontaneous dissection of the coronary arteries (SCAD), all entities being associated with myocardial ischaemia. Myocardial bridging (MB) is also a potential cause of myocardial ischaemia and will be discussed.

Since the focus of these recommendations does not lie on the routine work-up of CAD patients in general,[2] but on patients who wish to engage in leisure-time or competitive sports, these patients will be referred to as *athletes-patients*. While we acknowledge that competition offers an additional level of stress, adherence to training in different sports may convey a very-high intensity exercise schedule, and represents a major challenge. Cardiovascular pre-participation screening in athletes to detect CAD lies outside the scope of this article and is discussed elsewhere.[3,4]

In subjects >35 years of age including athletes, CAD is the main cause of myocardial ischaemia.[5] Major risk factors, in addition to age and sex include family history of CAD, hypercholesterolaemia, and smoking, particularly if combined.[6] Coronary

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artery disease in subjects below 35 years is rare and most often caused by familiar hyperkolesterolaemia. Physical inactivity is an additional risk factor for CAD, and conversely, regular physical training reduces the risk of developing CAD, as well as the risk of sudden cardiac death or arrest (SCD/SCA) during vigorous exertion.[7]

On the other hand, observational data indicate that intensive exercise training (beyond 7 times per week or 18 h of strenuous exercise per week), increases the mortality risk in patients with CAD.[8] Nevertheless, data from multiple endurance and non-endurance sporting events with participation demographics suggest that actual incidence of acute events is very low.[9–11] The risk of sudden death is two to three times higher in triathlon, possibly due to the added risk of immersion pulmonary oedema.[12] Thus, according to these observational evidences the benefits of regular physical activity and sport participation outweigh by far the increased risk for coronary events triggered by acute, intensive physical activity.

Myocardial ischaemia during exercise is caused by a demand-supply mismatch and may be provoked by an increase in heart rate, blood pressure, and workload such as typically occurs during exercise. Cardiac events during sports are believed to be triggered by neuro-hormonal activation, precipitating plaque rupture,[13] hypercoagulability, endothelial erosion,[14] and/or to very-high intensity exercise, exceeding the threshold of ischaemia in patient-athletes with chronic and stable CAD.[9,15]

Common symptoms include chest pain (typical or atypical angina), dyspnoea, palpitations, light-headedness, or syncope, which all are typically effort-related. With regard to the clinical presentation, it should be noted however, that athletes might present with atypical symptoms such as overall reduction of exercise-capacity, and unusually elevated heart rate during exercise, although they might occasionally be symptom-free, possibly due to improved collateral coronary circulation.[16] Absence of symptoms during effort, regardless the extent of underlying coronary pathology, is likely associated with a lower risk of ischaemia during acute, intense exercise. Indeed, in stable angina the extent of ischaemia does not seem to predict clinical long-term outcome.[15,17]

During recent years, novel cardiac imaging techniques including coronary artery calcium score, computed tomography (CT) coronary angiography and, to a lesser extent, cardiac magnetic resonance imaging (CMR) have made the diagnosis of subclinical coronary artery disease increasingly possible.[18] However, imaging techniques do not provide information relative to the coronary flow and reserve, which represents the key point to assess the risk of SCD/SCA associated with exercise. In this regard, the different methods of stress testing (e.g. cycle ergometry or treadmill testing), stress echocardiography, adenosine or dobutamine stress CMR, or positron emission tomography (PET)/single-photon emission computed tomography (SPECT), play a major role. Exercise testing has the advantage of being widely available, providing also functional information such as peak- and submaximal exercise capacity, blood pressure response, and the capability to detect exercise-induced arrhythmias.[19] However, exercise testing is known to have a low sensitivity, especially in asymptomatic individuals and in less advanced cases of CAD. Maximal exercise effort is often not adequately pursued in the routine clinical practice, whereby potential pathological findings may be missed. Therefore, in the setting of evaluation of competitive athletes with suspected CAD, maximal exercise capacity should be assessed, which is an important prognostic marker by itself.[20]

When patient-athletes are assessed for eligibility to competitive sports, it is understood that the patient-athlete should be eligible to participate in sport at very-high intensity level, without any limitation, since during competition maximal exertion may well occur.

A different attitude occurs when advising participation in leisure time and amateur sport, in which a measure of control of the intensity and duration of the exercise load is feasible. Moreover, the specific type of sport[21] should additionally be considered, since some are more likely to induce myocardial ischaemia than others, in relation to intensity and duration of the event/game (*Figure 1*). In advising patient-athletes with CAD to engage in competitive sports, we should carefully balance the documented benefits of exercise programmes with the potential risk for adverse events. Given the wealth of evidence supporting the benefits of exercise for primary and secondary prevention of CAD, we believe that individuals should be restricted from competitive sport only when a substantial risk of adverse event or disease progression is present.

Indeed, leisure time activity is advised and should be recommended individually (i.e. exercise prescription), to all individuals with risk factors for, as well as with manifest CAD.[6]

Thus, these recommendations aim to encourage regular physical activity including participation in sports and, with reasonable precaution, ensure a high level of safety for all individuals with CAD.[22] The present document is based on available current evidence, but in most instances because of lack of scientific evidence, also on clinical experience and expert opinion. The available class and level of evidence is given for each recommendation.

Coronary artery disease

Clinically, CAD in a previously healthy individual is typically suspected upon the presence of anginal symptoms, in addition to the traditional risk factors being present.[23] Symptomatic athletes with clinical suspicion of CAD should be assessed according to established guidelines[1,24] (*Figure 2*).

Importantly, most athletes with risk factors for and/or underlying CAD may be asymptomatic, as they may be detected during pre-race or pre-competition medical evaluations, or during cardiac screening and/or functional physical capacity testing.

Asymptomatic athletes with absence of clinically evident coronary artery disease

Coronary artery disease evolves gradually with subclinical disease becoming progressively more likely to be detectable over time. Although contentious, there is an increasing tendency to include coronary imaging in screening algorithms. As a result, clinical decision-making regarding asymptomatic coronary disease is encountered with increasing frequency. Therefore, clinical evaluation should include (*Figure 2*): (i) evaluation of functional ischaemia and (ii) assessment of coronary risk factors (with adequate treatment).

Exercise stress testing remains the pivotal test to evaluate the patient-athlete who wishes to enter competitive sports.

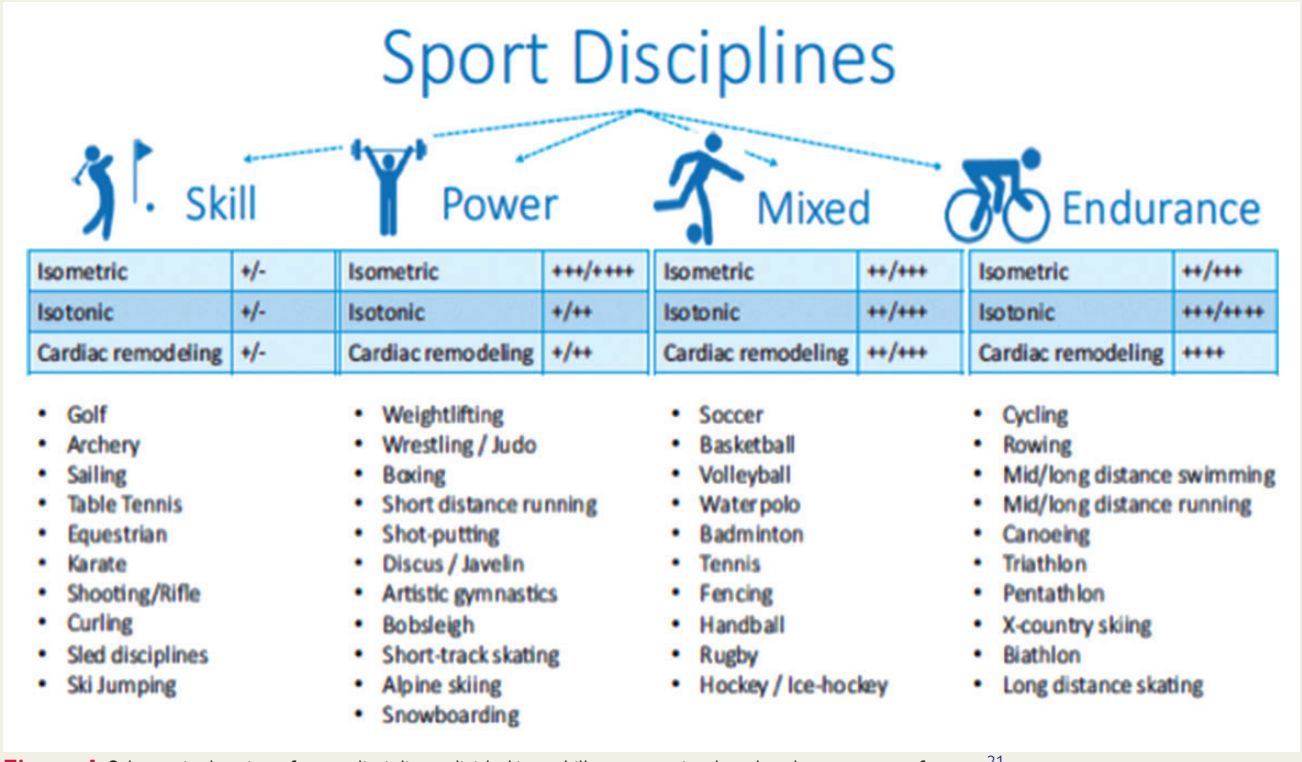

Figure 1 Schematic drawing of sport disciplines, divided into skill, power, mixed, and endurance type of sports.[21]

- In general, if the maximal exercise-test is *normal*, and cardiovascular risk factor profile is low, the presence of relevant CAD is assumed to be unlikely. In this instance, no additional tests are mandatory and no restriction for competitive sports is advised. Risk factor management should be adequate and annual follow-up is recommended.
- In case of a *borderline* or equivocal exercise test result (e.g. ST depression of 0, 15 mV, not typically ascending ST segment, etc.) as well as in the case of an uninterpretable electrocardiogram (ECG) (pre-existing left-bundle branch block (LBBB) or ventricular pacing), we recommend performing an additional stress test such as stress-echo/-CMR/PET/SPECT. This panel advices maximal exercise SPECT as first diagnostic step in athletes. However, we also acknowledge the option of exercise echocardiography or nuclear perfusion techniques (exercise or pharmacological). The choice of these tests is guided by their diagnostic accuracy, being dependent on local expertise and by their availability.
- If the exercise test is *positive*, preferentially CT or coronary angiogram should be performed to confirm presence and extent of CAD. In case CT shows the presence of significant lesions, according to routine clinical criteria,[1,24] the patient-athlete should undergo coronary angiography. It should be noticed that master endurance athlete show a higher degree, and a more diffuse distribution of coronary calcium in the coronary tree compared with non-athletes at similar low risk-factor level.[25] At present, the long-term clinical implications of these findings are debated.

Subsequently, the patient management as well as decision-making regarding sports participation should be made according to the diagnosis of CAD (see athletes-patients with clinically proven CAD; section 'Clinically proven CAD' below).

Risk factor assessment and treatment should be pursued, as advised in detail in guidelines; there is no evidence that athletes should be treated any differently than non-athletes.[1] In fact, treatments for dyslipidaemia and arterial hypertension have comparable efficacy and similar adverse effects, although there is some evidence that statin-associated myalgia may be slightly more common amongst athletes.[26]

Given the net benefits of exercise, we recommend that patient-athletes with asymptomatic coronary disease defined as CAD with no evidence of inducible ischaemia on functional tests, may be advised for participation in all types of exercise programmes including competitive sports, based on an individual careful evaluation.
Effective risk factor management according to guidelines is mandatory.[1]
The athlete-patient should periodically be reassessed regarding risk profile and progression/regression of CAD—Level of recommendation: Class IIa, level of evidence C.

Clinically proven coronary artery disease

For athletes-patients with proven CAD, as documented by an earlier clinical event, CT scan or coronary angiography, advice relative to sport participation should be based on individual assessment

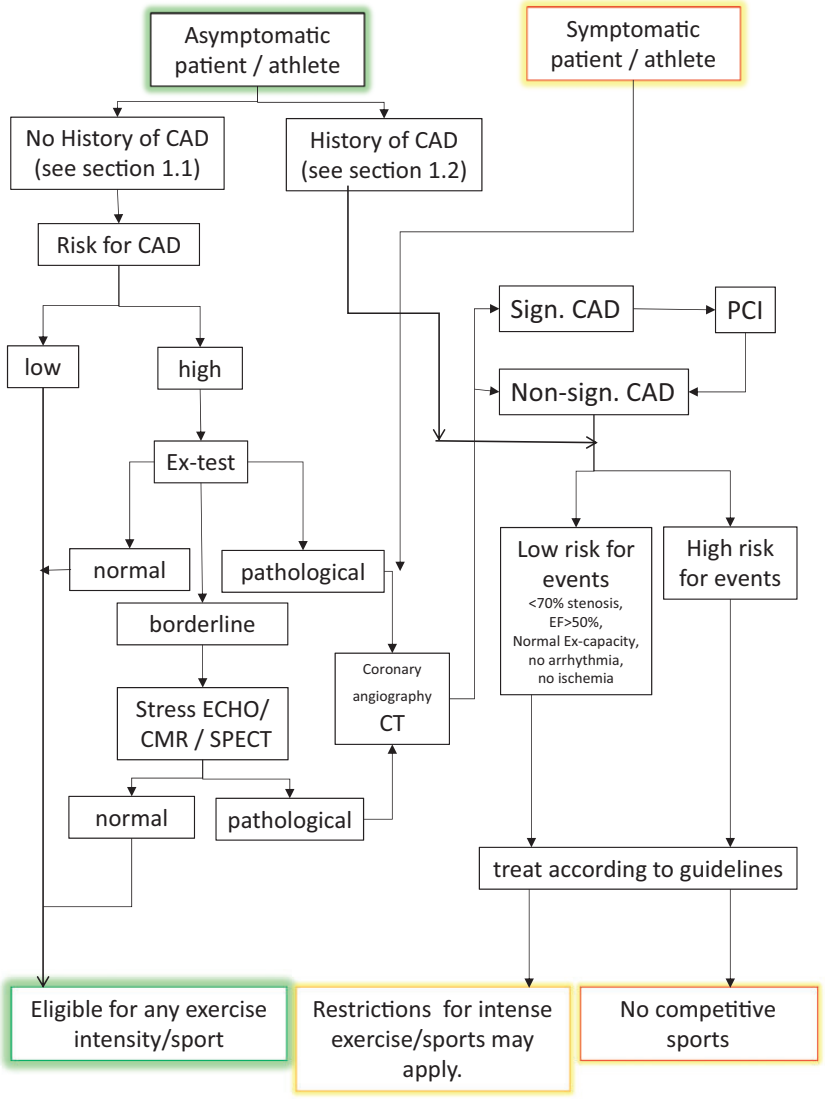

Figure 2 Clinical evaluation and recommendations of eligibility in athletes with coronary artery disease or risk of coronary artery disease.

(*Figure* 2). Recommendations on eligibility for competitive sports should primarily be based on:

- Presence of exercise-induced myocardial ischaemia
- Exercise induced arrhythmia
- Evidence of myocardial dysfunction
- Type and level of sport competition
- Fitness level of the individual patient-athlete
- Profile of cardiovascular risk factors[3]

According to the results of diagnostic testing, we recommend to stratify athletes-patients with proven CAD as follows:

Low probability for exercise-induced adverse cardiac events, if *all* of the following apply:

- Absence of critical coronary stenoses (i.e. <70%) of major coronary arteries or <50% of left main stem on coronary angiography
- Ejection fraction ≥50% on echocardiography, CMR or angiography (and no wall motion abnormalities)
- Normal, age-adjusted exercise capacity
- Absence of inducible ischaemia on maximal exercise testing
- Absence of major ventricular tachyarrhythmias (i.e. non-sustained ventricular tachycardia (NSVT),[27] polymorphic or very frequent ventricular extra beats (VEBs), at rest and during maximal stress testing)

High probability for exercise-induced adverse cardiac events, if *at least one* of the following applies:

- Presence of at least one critical coronary stenosis of a major coronary artery (>70%) or left main stem (>50%) on coronary angiography
- Ejection fraction <50% on echocardiography (or other tests)
- Exercise-induced ischaemia, >0.1 mV ST depression (horizontal or
- down-sloping at 80 ms after the J point) in two chest leads or ST elevation >0.1 mV (in a non-Q-wave lead and excluding aortic valve replacement) or new left bundle branch block at low exercise intensity or immediately post-exercise[28]

- Dyspnoea at low exercise intensity (angina equivalent)
- Relevant ventricular tachyarrhythmias (i.e. NSVT,[27] polymorphic or very frequent VEBs, at any time)
- Dizziness or syncope on exertion
- High degree of myocardial scarring on CMR imaging

As general consideration, this panel believes that:

If ischaemia is present during functional testing despite adequate treatment, revascularization may be primarily considered. Specifically, in case the athlete-patient wants to participate in competitive sports, revascularization should be preferred, since during maximal exercise the high myocardial oxygen consumption attained and the neurohormonal activation increases the likelihood of myocardial ischaemia and cardiac events (N.B.: expert consensus). Furthermore, antianginal medications such as beta-blockers may be less well tolerated in athletes.

If despite adequate treatment ischaemia cannot be completely resolved, then the athlete-patient should be restricted from competitive sport and advised to enter leisure-time sports activities, which are associated with less physical demands and lower intensity, so that ischaemia may more likely be avoided (Ref.[29]; *Figure 1*).

If the athlete-patient is going to engage in leisure-time physical activity, revascularization may be not be strictly required,[29–31] as the evidence supporting revascularization over medical management for stable CAD remains contentious.[31–34]

Importantly, the present recommendations are in agreement with the European Society of Cardiology (ESC) guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden death, including that any ventricular tachycardia and/or other relevant arrhythmia, should be handled according to established standards.[27]

Recommendations

- Athletes-patients with clinically proven CAD and considered to be at *low-risk for cardiac events* may be selectively advised to participate in competitive sports (*Figure 2*). However, as a measure of caution due to the high haemodynamic load and possible electrolyte imbalance, restrictions may apply on an individual basis for certain sports with the highest CV demand (such as extreme power and endurance disciplines, see *Figure 1*). Moreover, older athletes-patients with CAD and even low risk profiles deserve special attention, and a more cautious advice, as recent studies have shown that the risk of SCD during endurance events may be considerably higher in men >60-year-old[35]—Level of recommendation: Class IIa, level of evidence C
- Patient-athletes with clinically proven CAD, defined *as high risk*, should be temporarily restricted from competitive sport and receive appropriate management (*Figure 2*). As in all patients, also in patient-athletes with CAD and significant ischaemia during exercise, anti-ischaemic therapy needs to be optimized. In case of continued ischaemia, revascularization ought to be performed.[35,36] Level of recommendation: Level of recommendation: Class IIa, level of evidence C

The individual athlete-patient who has been revascularized following angina, an acute coronary syndrome or AMI should be encouraged to start exercise programs without delay as per the cardiac rehabilitation guidelines of the ESC.[6] In the early phase, exercise should be prescribed in a graduated fashion, starting with low-intensity exercise of limited duration and progressively increased to a point at which the athlete/patient is able to perform exercise without limitation, with careful attention to the development of new symptoms.[37] Typically, in the athlete-patient with acute coronary syndrome (ACS) or acute myocardial infarction (AMI) the duration of this process is dependent upon the extent of myocardial injury and remodelling and should be serially assessed, at least after 3 months, in accordance with current American guidelines.[37] In those with non-ST elevation myocardial infarction (NSTEMI) or stable CAD with complete revascularization and without remaining ischaemia, exercise training can be progressively increased with a faster pace to the previous levels. The graduated exercise schedule can be performed in the setting of a structured cardiac rehabilitation program, but not all rehabilitation programmes are ideally suited to younger athlete-patients motivated to improve fitness. A prompt return to exercise should be encouraged in either a structured programme or with individualized advice with serial controls for adherence and efficacy.

Eventually, a more intense training and participation in competition should only be considered after a graduated and progressive increase in rehabilitation training load.

In summary, provided the patient-athlete is considered as having a "low-probability" for cardiac events, we recommend a minimum of three months after-percutaneous coronary intervention (PCI), before participation in competitive sports can be resumed. Contact sports should be avoided while the athlete-patient is under dual antiplatelet therapy, because of the risk of bleeding, but may be considered afterwards.

Eligibility assessment should always be combined with advising the athlete-patient on the correct approach to training (e.g., warm-up and cool-down; adequate hydration, awareness of the weather conditions. . .). Periodical cardiac evaluation, at least on a yearly basis, is advised. The risk factors should be properly managed with appropriate pharmacologic and lifestyle modifications, as they may affect the speed of progression of the atherosclerotic disease.

In summary, athletes-patients with clinically proven CAD, considered as having a low probability for events (anatomically as well as functionally), are eligible for most sports, also at competitive level based on individual evaluation.
However, exceptions apply for high-intensity sports (intensive power and endurance sport) and athletes-patients of older age (>60 years).
Level of recommendation: Class IIa; level of evidence C.

Non-coronary artery disease related myocardial ischaemia

Congenital coronary artery anomalies

In this section, we refer to the CAAs that include origins of the coronary vessel from the wrong sinus and anomalous origin from the pulmonary artery. These CAA are associated with SCA/SD, often in

young, asymptomatic individuals.[38] The incidence in the normal population is not fully known, but approximated to 0.5–1%.[39]

Resting ECG, echocardiography and even exercise testing frequently are not able to show any abnormal findings. Chest pain or syncope on exertion, or even SCD, may be the first symptoms of CAA.[40] Mechanisms leading to SCD likely include (repeated bursts of) ischaemia with consequent increase of fibrous tissue and a proclivity to develop ventricular arrhythmia during exercise. Ischaemia may be the consequence of compression of the anomalous vessel coursing between the aorta and the pulmonary artery and/or due to the acute angled take-off from the aorta. In case of anomalous origin of the left main CA from the pulmonary trunk, there is a chronic ischaemic condition, being exacerbated by exercise.

Multi-slice contrast-enhanced CT or CT coronary angiography are primary diagnostic tools, while in clinical practice (pre-participation cardiac evaluation), echocardiography may reveal or raise the suspicion of an abnormal origin of the coronary artery. In view of limiting radiation exposure, in particular to adolescent patients, cardiac CMR may also be an option.

Eligibility for competitive sport is based on the anatomical type of CAA, as well as on the presence of ischaemia.

- Specifically, in CAA originating from the wrong sinus, with acute angled take-off from the aorta and anomalous coursing between the aorta and the pulmonary artery, the risk for SCA/SCD is believed to be the highest. Strong consideration should be given to surgical correction of such an anomaly in symptomatic patients. Prior to successful correction, participation in high-intensity sport is discouraged. Level of recommendation: Class II, level of evidence C.
- Traditionally, CAAs without inter-arterial course have been considered having a low risk of SCA/SCD. In the absence of ischaemia and arrhythmias on stress testing or symptoms (dizziness, fainting or syncope), there is no indication for surgical repair or treatment. At present, because of a lack of adequate data, an individualized approach for competitive sports participation is recommended, based on comprehensive evaluation (N.B.: expert consensus). Level of recommendation: Class III, level of evidence C.
- In case of previous surgical correction and lack of persistent, inducible ischaemia, all competitive sports are allowed. Level of recommendation: Class III, level of evidence C.
- In other types of CAA, such as anomalous origin of the circumflex artery from the right sinus, it is relevant to confirm the absence of inducible ischaemia and, in this case, no restriction exist regarding competitive sport participation. Level of recommendation: Class IIa, level of evidence C.

Coronary artery dissection

There is little epidemiological evidence on the incidence of spontaneous coronary artery dissection as a cause of acute coronary events or sudden death in athletes, but some have suggested that there may be substantial under-appreciation of this entity.[41,42] There are some estimates of incidence and some reports have suggested significant rates of recurrence in exercise-triggered SCAD.[43]

Treatment for SCAD is similarly uncertain with a range of options including percutaneous coronary intervention, bypass surgery and/or medical therapy with dual anti-platelet therapy or anticoagulation. There is no evidence comparing the efficacy and risks of these strategies. Similarly, the duration of medical therapies is empirical. However, frequently SCAD is seen as a result of coronary plaque formation, which emphasizes the need for optimal risk factor management.[44]

- There is currently insufficient evidence on the risk of SCAD in athletes and and we would favour using this as a call for further research so that future guideline iterations can be more informed.
- In the meantime, it is prudent to treat this condition cautiously and discourage intense competitive sport on the grounds of an increased rate of recurrence in predisposed individuals and the potential for severe cardiac injury or death as a consequence. While this panel believes that individuals with SCAD, at present, should be discouraged from competitive sport participation, leisure time activity is advised, and should be recommended individually (i.e. exercise prescription). Level of recommendation: Class III, level of evidence C.

Myocardial bridging

Myocardial bridging may be occasionally discovered at imaging testing required to solve the ambiguity of an abnormal exercise ECG. Similar to CAA, MB should be suspected in athletes who present with exertional angina or syncope.

Evaluation of the individuals with MB aims primarily at assessing the presence of inducible ischaemia. Recently, it has been shown that the percentage of arterial compression in MB may be directly related to the atherosclerotic burden, proximal to the MB.[45] Observational studies have shown that in patients without obstructive CAD on coronary CT, the presence of an intramural course of a coronary artery was not associated with a clinical worsening in 5-year follow-up.[46] Thus, MB without other underlying diseases (e.g. hypertrophic cardiomyopathy) and with no evidence of inducible myocardial ischaemia/CAD, seems to have a good prognosis.

- In the absence of inducible effort-related ischaemia or complex ventricular tachyarrhythmias (i.e. NSVT, polymorphic or very frequent VEBs, induced by exercise), there is little evidence for exercise-induced harm. Therefore, asymptomatic athletes-patients with myocardial bridging can participate in all competitive and leisure-time sports.[36] Level of recommendation: Class IIa, level of evidence C.
- Conversely, in those with evidence of ischaemia or symptoms, beta-blockers are the first line therapy. If this therapy fails, then surgical repair may be considered, whereas stenting is discouraged.[47,48] These individuals should be restricted from participation in competitive sports, and should be properly advised regarding leisure-time activities. Level of recommendation: Class IIa, level of evidence C.

Conflict of interest: none declared.

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