

Brief 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)

Mats Borjesson, Mikael Dellborg, Josef Niebauer, Andre Lagerche, Christian Schmied, Erik E Solberg, Martin Halle, Paolo Emilio Adami, Alessandro Biffi, François Carré, et al.

▶ To cite this version:

Mats Borjesson, Mikael Dellborg, Josef Niebauer, Andre Lagerche, Christian Schmied, et al.. Brief 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). European Journal of Preventive Cardiology, 2020, 27 (7), pp.770-776. 10.1177/2047487319876186 . hal-02304380

HAL Id: hal-02304380 https://univ-rennes.hal.science/hal-02304380v1

Submitted on 2 Sep 2020

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Executive summary:

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)

Mats Borjesson 1,2, MD, FESC, Mikael Dellborg 3, MD, Josef Niebauer 4, MD, Andre

LaGerche 5, MD, Christian Schmied 6, MD, Erik E Solberg 7, MD, FESC, Martin Halle 8,

MD, Paolo Emilio Adami 9, MD, FESC, Alessandro Biffi 10, MD FESC, Francois Carré 11,

MD, Stefano Caselli 12, MD, FESC, Michael Papadakis 13, MD, FESC, Axel Pressler 14a+b,

MD, Hanne Rasmusen 15, MD, Luis Serratosa 16, MD, Sanjay Sharma 17, MD, FESC, Frank

van Buuren 18, MD, and Antonio Pelliccia 19, MD, FESC.

- 2. Dept of Neuroscience and Physiology, Gothenburg University and Sahlgrenska University Hospital/Östra, Gothenburg, Sweden.
- 3. Dept of Medicine, Sahlgrenska University Hospital/Östra, Gothenburg, Sweden
- 4. University Institute of Sports Medicine, Prevention and Rehabilitation, Paracelsus Medical University, Salzburg, Austria
- 5. Baker Heart and Diabetes Institute, Melbourne, Australia.
- 6. Kardiologisches Ambulatorium, Sportmedizin/Sportkardiologie, Universitäres Herzzentrum Zürich, Switzerland.
- 7. Department of Medicine, Diakonhjemmet Hospital, Oslo, Norway
- 8. Dept. Prevention, Rehabilitation and Sports Medicine, Medical Faculty, University Hospital, Technical University Munich, Germany
- 9. Institute for Sports Medicine and Science, Italian Olympic Committe, Rome, Italy
- 10. Institute for Sports Medicine and Science, Italian Olympic Committe, Rome, Italy
- 11. Sport Medicine Department-Rennes University Hospital, LTSI INSERM UMR 1099, Rennes France,
- 12. Institute for Sports Medicine and Science, Italian Olympic Committe, Rome, Italy and Ospedale San Pietro Fatebenefratelli, Rome, Italy
- 13. Cardiology Clinical Academic Group, St George's, University of London, UK

14. Center for general, sports and preventive cardiology, Munich, Germany; 14b Department of prevention, rehabilitation and sports medicine, Technical University of Munich, Munich, Germany;

- 15. Department of Cardiology, Bisbebjerg University Hospital, Copenhagen, Denmark
- 16. Hospital Universitario Quironsalud Madrid, Spain. Ripoll y De Prado Sport Clinic, FIFA Medical Centre of Excellence, Spain
- 17. Cardiology Clinical Academic Group, St George's, University of London
- 18. Catholic Hospital Southwestfalia, St. Martinus-Hospital Olpe
- 19. Institute for Sports Medicine and Science, Italian Olympic Committe, Rome, Italy

Corresponding author:

Mats Borjesson, prof, MD, FESC

Center for Health and Performance and Dept of Neuroscience and Physiology, Göteborg University and

Sahlgrenska University Hospital, Göteborg, Sweden

Mail: mats.brjesson@telia.com

¹ Dept of Food, Nutrition and Sports Science, Gothenburg Univcersity

Introduction

In subjects >35years of age, including athletes, CAD is the main cause of myocardial ischemia (2). Major risk factors, in addition to age and sex, include family history of CAD, hypercholesterolemia and smoking, particularly if combined (3). 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 (4).

On the other hand, observational data indicate that intensive exercise training (beyond 7 times per week or 18 hours of strenuous exercise per week), increases the mortality risk in patients with CAD (5). Cardiac events during sports are believed to be triggered by neuro-hormonal activation, precipitating plaque rupture (6), hypercoagulability, endothelial erosion (7) and/or by very high intensity exercise, exceeding the threshold of ischemia in patient-athletes with chronic and stable CAD (8) (9).

During recent years, novel cardiac imaging techniques including coronary artery calcium score (CACS) and CT coronary angiography (CTCA) have made the diagnosis of subclinical coronary artery disease increasingly possible (10). 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. Exercise testing has the advantage of being widely available, providing also functional information, such as peak- and submaximal exercise capacity, blood pressure response and with the capability to detect exercise-induced arrhythmias (11). 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 (12).

2

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. The specific type of sport (13) should additionally be considered, since some are more likely to induce myocardial ischemia than others, in relation to intensity and duration of the event/game (Figure 1). 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 (3).

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 (14).

1. Coronary artery disease- CAD

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.

1.1 Asymptomatic athletes with absence of clinically evident CAD

CAD 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. Therefore, clinical evaluation should include (Figure 2):

-evaluation of functional ischemia,

-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.

- In general, if the maximal exercise-test is **normal**, and CV 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 0,15mV, not typically ascending ST segment, etc.) as well as in the case of an uninterpretable ECG (pre-existing LBBB or ventricular pacing), we recommend performing an additional stress-test, such as stress-Echo/-MRI/PET/SPECT. 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, 15), the patient-athlete should undergo coronary angiography.

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; 1.2 below).

Given the net benefits of exercise, we recommend that patient-athletes with asymptomatic coronary disease defined as CAD with no evidence of inducible ischemia on functional tests, may be advised for participation in all types of exercise programs, 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: IIa, level of evidence C.

1.2 Clinically proven CAD

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). Recommendations on eligibility for competitive sports should primarily be based on:

- Presence of exercise-induced myocardial ischemia
- 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 (16)

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 ischemia on maximal exercise testing
 Absence of major ventricular tachyarrhythmias (i.e., non-sustained ventricular tachycardia (NSVT)(17), 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 ischemia, >0.1 mV ST depression (horizontal or down-sloping (at 80 ms after the J point) in 2 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 (18)
- Dyspnea at low exercise intensity (angina equivalent)
- Relevant ventricular tachyarrhythmias (i.e., non-sustained ventricular tachycardia (NSVT) (17), polymorphic or very frequent ventricular extra beats (VEBs), at any time,
- Dizziness or syncope on exertion
- High degree of myocardial scarring on CMR imaging

If ischemia is present during functional testing despite adequate treatment, revascularization may be primarily considered. If despite adequate treatment ischemia 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 ischemia may more likely be avoided ((19); figure 1).

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 hemodynamic 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-years old (20) -Level of recommendation: Class IIa, level of evidence C
- Athletes-patients 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 ischemia during exercise, anti-ischemic therapy needs to be optimized. In case of continued ischemia, revascularization ought to be performed (20, 21). -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 (3). Typically, in the athlete-patient with ACS or 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 (22). In those with NSTEMI or stable CAD with complete revascularization and without remaining ischemia, exercise training can be progressively increased with a faster pace to the previous levels. 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-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. Periodical cardiac evaluation, at least on a yearly basis, is advised.

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: IIa; level of evidence C

2. Non-CAD related myocardial ischemia

2.1. Congenital coronary artery anomalies, CAA

CAA are associated with SCA/SD, often in young, asymptomatic individuals (23). The incidence in the normal population is not fully known, but approximated to 0.5-1% (24). Resting ECG, echocardiography and 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 (25). Multi-slice contrast-enhanced computed tomography (CT) or CT coronary angiography are primary diagnostic tools, while in clinical practice (preparticipation cardiac evaluation), echocardiography may reveal or raise the suspicion of an abnormal origin of the coronary artery.

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

- 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/SD 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 ischemia 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 ischemia, 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 ischemia and, in this case, no restriction exist regarding competitive sport participation. -Level of recommendation: Class IIa level of evidence C.

2.2 Coronary artery dissection

There is little epidemiological evidence on the incidence of sudden coronary artery dissection (SCAD) 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 (26, 27). There are some estimates of incidence and some reports have suggested significant rates of recurrence in exercise-triggered SCAD (28).

- 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). Class of recommendation III, level of evidence C.

2.3 Myocardial bridging

Myocardial bridging (MB) 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 ischemia. MB without other underlying diseases (e.g. hypertrophic cardiomyopathy) and with no evidence of inducible myocardial ischemia/CAD, seems to have a good prognosis.

- In the absence of inducible effort-related ischemia 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 (21). Level of recommendation: Class IIa, level of evidence C.
- Conversely, in those with evidence of ischemia or symptoms, beta-blockers are the first line therapy. If this therapy fails, then surgical repair may be considered, whereas stenting is discouraged (29, 30). 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 statement

?

References

1. Montalescot G, Sechtem U, Achenbach S, Andreotti F, Arden C, Budaj A, et al. 2013 ESC guidelines on the management of stable coronary artery disease. Eur Heart J. 2013;34:2949-3003.

2. Adabag A, Peterson G, Apple F, Titus J, King R, Luepker R. Etiology of sudden death in the community: results of anatomical, metabolic and genetic evaluation. Am Heart J. 2010;159:33-9.

3. Piepoli M, Hoes A, Agewall S, Albus C, Brotons C, Catapano A, et al. 2016 European guidelines on cardiovascular disese prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology amnd Other Societies on Cardiovascular Disease Prevention in Clinical Practice. . Eur Heart J. 2016;37:2315-81.

4. Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ, Muller JE. Triggering of acute myocardial infarction by heavy physical exertion. Protection against triggering by regular exercise. . N Engl J Med. 1993;329:1677-83.

5. Mons U, Hahmann H, Brenner H. A reverse J-shaped association of leisure-time physical activity prognosis in patients with stable coronary heart disease: evidence from a large cohort with repeated measurements. Heart. 2014;100:1043-9.

6. Thompson PD, Franklin BA, Balady GJ, Blair SN, Corrado D, EstesIII NAM, et al. Exercise and acute cardiovascular events. Placing the risks into perspective. A scientific statement from the American heart Association Council on nutrition, physical activity, and metabolism and the Council on Clinical Cardiology. Circulation. 2007;115:2358-68.

7. Quillard T, Franck G, Mawson T, Folco E, Libby P. Mechanisms of erosion of atherosclerotic plaques. Current opinion in lipidology. 2017;28:434-41.

8. Kim JH, Malhotra R, Chiampas G, d'Hemecourt P, Troyanos C, Cianca J, et al. Cardiac arrest during long-distance running races. Race Associated Cardiac Arrest Event Registry (RACER) Study Group. N Engl J Med. 2012;366:130-40.

9. Shaw LJ, Weintraub WS, Maron DJ, Hartigan PM, Hachamovitch R, Min JK, et al. Baseline stress myocardial perfusion imaging results and outcomes in patients with stable ischemic heart disease randomized to optimal medical therapy with or without percutaneous coronary intervention. Am Heart J. 2012;164:243-50.

10. Erbel R, Möhlenkamp S, Moebus S, Schmermund A, Lehmann N, Stang A, et al. Coronary trisk stratification, discrimination, and reclassification improvement based on quatification of subclinical coronary atherosclerosis. The Heinz Nixdorf Recall Study. J Am Coll Cardiol. 2010;56:1397-406.

11. Börjesson M, Dellborg M. Exercise-testing post-MI: still worthwhile in the interventional era? Eur Heart J. 2005;26:105-06.

12. Shuval K, Finley CE, Barlow CE, Gabriel KP, Leonard D, Kohl3rd HW. Sedentary beavior, cardiorespiratory fitness, physical activity, and cardiometabolic risk in men: the Cooper Center Longitudinal Study. Mayo Clin. 2014;89:1052-62.

13. Pelliccia A, Adami P, Quattrini F, Squeo M, Caselli S, Verdile L, et al. Are Olympic athletes free from cardiovascular diseases? Systematic investigation in 2352 participants from Athens 2004 to Sochi 2014. Br J Sports Med. 2017;51:238-43.

14. Vanhees L, Rauch B, Piepoli M, van Buuren F, Takken T, Borjesson M, et al. Importance of characteristics and modalities of physical activity and exercise in the management of cardiovascular health in individuals with cardiovascular disease (Part III). Eur J Prev Cardiol. 2012;19(6):1333-56.

15. Vrints C, Senior R, Crea F, Sechtem U. Assessing suspected angina: requiem for coronary computed tomography angiography or exercise electrocardiogram? Eur Heart J. 2017;38:1792-800.

16. Börjesson M, Urhausen A, Kouidi E, Dugmore D, Sharma S, Halle M, et al. Cardiovascular evaluation of middle-aged/senior individuals engaged in leisure-time sport activities: position stand from the section of exercise physiology and sports cardiology of the European Association of Cardiovascular Prevention and Rehabilitation. Eur J Cardiovasc Prev Rehabil. 2011;18:446-58.

17. Priori S, Blomstrom-Lundqvist C, Mazzanti A, Blom A, Borggrefe M, Camm J, et al. 2015 ESC Guidelines for the mangement of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Eur Heart J. 2015;17:1601-87.

18. Fletcher GF, Ades PA, Kligfield P, Arena R, Balady GJ, Bittner VA, et al. Exercise standards for testing and training: a scientific statement from the American Heart Association. Circulation. 2013;128:873-934.

19. Niebauer J, Hambrecht R, Velich T, Hauer K, Marburger C, Kälberer B, et al. Attenuated progression of coronary artery disease after 6 years of multifactorial risk intervention: role of physical activity. Circulation. 1997;96:2534-41.

20. Harris K, Crewswell L, Haas T, Thomas T, Tung M, Isaacson E, et al. Death and cardiac arrest in U.S. triathlon participants, 1985 to 2016: a case series. Ann Intern Med. 2017;167:529-35.

21. Iqbal J, Zhang Y-J, Holmes D, Morice M-C, Mack M, Kappetein A, et al. Optimal medical therapy improves clinical outcomes in patients undergoing revascularization with percutaneous coronary intervention or coronary artery bypass grafting: insights from the Synergy Between Percutaneous Coronary Intervention With TAXUS and Cardiac Surgery (SYNTAX) trial at the 5-year follow-up. Circulation. 2015;131:1269-77.

22. Thompson P, Myerburg R, Levine B, Udelson J, Kovacs R. Eligibility and disqualification recommendations for competivite athletes with cardiovascular abnormalities: Task Force 8: Coronary artery disease. A scientific staement from the American Heart Association and the American College of Cardiology. Circulation. 2015;132:e310-4.

23. Hill SF, Sheppard MN. A silent cause of sudden cardiac death especially in sport: congenital coronary artery anomalies. Br J Sports Med. 2014;48:1151-6.

24. Yamanaka O, Hobbs R. Coronary artery anomalies in 126,595 patients undergoing coronary arteriography. Cathet Cardiovasc Diagn. 1990;21:28-40.

25. Basso C, Maron BJ, Corrado D, Thiene G. Clinical profile of congenital coronary artery anomalies with origin from the wrong sinus leading to sudden death in young competitive athletes. J Am Coll Cardiol. 2000;35:1493-501.

26. Nishiguchi T, Tanaka A, Ozaki Y, Taruya A, Fukuda S, Taguchi H, et al. Prevalence of spontaneous coronary artery disseaction in patients with acute coronary syndrome. Eur H J Acute Cardiovasc Care. 2016;5:263-70.

27. Rashid H, Wong D, Wijesekera H, Gutman S, Shanmugam V, Gulati R, et al. Incidence and characterisation of spontaneous coronary artery dissection as a cause of acute coronary syndrome- a single-centre Australian experience. Int J Cardiol. 2016;202:336-8.

 Tweet M, Hayes S, Pitta S, Simari R, Lerman A, Lennon R, et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. Circulation. 2012;126:579-88.
 Cerrato E, Barbero U, D'Ascenzo F, Taha S, Biondi-Zoccai G, Omede P, et al. What is the optimal treatment for symptomatic patients with isolated coronary myocardial bridge? A systematic review and pooled analysis. J Cardiovasc Med. 2017;18:758-70.

30. Tarantini G, Migliore F, Cademartini F, Fraccaro C, Iiiceto S. Left anterior descending artery myocardial bridging: a clinical approach. J Am Coll Cardiol. 2016;68:2887-99.