

## A case of asymptomatic ST segment changes in cyclist with two myocardial bridges

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### Abstract

A 65-year-old male regularly involved in competitive cycling came to our sports medicine laboratory for the annual mandatory pre-participation screening. Cycling screening protocol includes a cardiological examination, basal ECG and cardiac stress test. The clinical examination was unremarkable, and the patient's blood pressure was 120/75 mmHg. The rest-ECG was normal. The cardiac exercise stress test showed a 2 mm ST-segment inversion in the anterior leads (V3-V4-V5-V6) at peak exercise. No presence of arrhythmias or symptoms were reported. Due to an ST segment depression recorded during the cardiac exercise stress test, we performed a coronary computed tomography angiography (CCTA) that showed two myocardial bridges of the left coronary artery. However, we observed no atherosclerotic plaque of the coronary lumen. A dobutamine stress echocardiogram and a bicycle stress echocardiogram were normal. We concluded for ventricular repolarisation abnormalities during maximal exercise testing due to the electrocardiographic findings in an asymptomatic athlete without any coronary artery disease. In literature, myocardial bridging is regarded as a common anatomic variant rather than a congenital anomaly. Nevertheless, some reports show that myocardial ischemia and myocardial infarction or sudden death could be caused by myocardial bridging. In addition, intramyocardial bridging is a recognised cause of sudden death in athletes. Therefore, according to the Italian cardiological guidelines for competitive sports (COCIS 2009), we recommended the patient avoid physical overload and sport practice.

**KEY WORDS:** Athletes; coronary artery anomalies; coronary computed tomography angiogram; exercise stress test; myocardial bridge.

## Riassunto

Un uomo di 65 anni regolarmente impegnato in gare di ciclismo agonistico è giunto al nostro ambulatorio di medicina dello sport per la visita medica obbligatoria annuale di idoneità. Il protocollo di screening per il ciclismo include una visita specialistica cardiologica, un esame elettrocardiografico di base ed uno da sforzo. La visita medica non ha evidenziato alterazioni significative e la pressione arteriosa del paziente era pari a 120/75 mmHg. L'ECG a riposo era normale. Il test cardiaco da sforzo evidenziava un'inversione del tratto ST di 2 mm nelle derivazioni anteriori durante lo sforzo massimale. Nessuna aritmia né sintomi ischemici venivano riferiti. A causa della depressione del segmento ST registrato durante il test da sforzo, abbiamo effettuato una Tomografia computerizzata angiografica delle arterie coronariche che ha messo in evidenza la presenza di due ponti miocardici dell'arteria coronarica sinistra. Tuttavia, non abbiamo osservato alcuna placca aterosclerotica del lume coronarico. Un esame ecocardiografico da stress alla dobutamina ed un esame ecocardiografico da stress al cicloergometro sono risultati nella norma. Abbiamo concluso per la presenza di alterazioni elettrocardiografiche della ripolarizzazione ventricolare durante il test da sforzo massimale in atleta asintomatico senza segni di malattia coronarica. In letteratura, il ponte miocardico è considerato una variante coronarica anatomica piuttosto che un'anomalia congenita. Nonostante ciò, alcuni studi dimostrano che l'ischemia e l'infarto miocardico o la morte improvvisa potrebbero essere causati dal ponte miocardico. Inoltre, il ponte miocardico è una causa riconosciuta di morte improvvisa negli atleti. Pertanto, secondo le linee guida cardiologiche italiane per gli sport competitivi (COCIS 2009), abbiamo raccomandato al paziente di evitare il sovraccarico fisico e la pratica sportiva.

### TAKE-HOME MESSAGE

*Myocardial bridging has been associated with myocardial ischemia, myocardial infarction or sudden death in athletes. Cardiac stress test and, eventually, coronary computed tomography angiography (CCTA) should be indicated in competitive athletes for the annual mandatory pre-participation screening.*

**Competing interests** - none declared.

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## INTRODUCTION

Myocardial bridging is a congenital variant of a coronary artery in which a portion of an epicardial coronary artery takes an intramuscular course [1]. It is generally considered to be a benign and frequently asymptomatic condition, but in many cases it has been proposed as a cause of angina-like chest pain, acute coronary syndromes [2], myocardial ischemia [3] and other serious cardiac events such as sudden cardiac deaths [4–6]. The link between myocardial bridging and such serious and uncommon events is still controversial and unclear [7]. Patients with hypertrophic cardiomyopathy have been found to have a high prevalence of myocardial bridging, with reported rates of up to 80% on angiographies, but myocardial bridging is also present anatomically in approximately 25% of patients based on autopsy and coronary computed tomography angiography (CCTA) results [7–9]. Clearly, it is clinically silent in the majority of cases [7]. After hypertrophic cardiomyopathy, coronary artery anomalies (CAAs) are considered to be the second major cause of death in young athletes [10, 11]. In this paper, we focused on the relationship between myocardial bridging and eligibility for competitive sports, showing a case of an asymptomatic competitive amateur cyclist with ECG signs of repolarisation abnormalities associated with myocardial bridging in stress testing.

## CASE REPORT

A 65-year-old male regularly involved in competitive cycling came to our sports medicine laboratory for the annual mandatory pre-participation screening. Cycling screening protocol includes a cardiological examination, basal ECG and cardiac stress test. Family and personal history were negative for cardiac diseases. He was not a smoker and denied use of any medicaments. His family history was negative for ischemic heart disease or premature sudden death. The cyclist was in excellent athletic condition, with a weight of 66 kg, height of 1.74 m and Body Mass Index of 21.8 kg/m<sup>2</sup>.

The clinical examination was unremarkable

and the patient's blood pressure was 120/75 mmHg. The rest-ECG was normal. The cardiac exercise stress test showed a 2 mm ST-segment inversion in the anterior leads (V3-V4-V5-V6) at peak exercise, when the patient reached 180 beats per minute at peak exercise heart rate and 300 Watt of peak mechanical power (Figure 1). His blood pressure was normal. No presence of arrhythmias or symptoms were reported. As a second-level examination, we performed a Holter ECG monitoring that showed an absence of ventricular arrhythmias. Afterwards, we performed a CCTA to analyse the coronary anatomy and patency, which showed the presence of two myocardial bridges, in the medium tract of the anterior interventricular branch and in the medium tract of a large intermediate branch of the left coronary artery, both approximately 20 mm long and 2 mm depth (Figure 2). No proximal atherosclerotic plaque of the coronary lumen was found. A dobutamine stress echocardiography and a bicycle stress echocardiography performed after two months were normal. No cardiac symptoms during the test and ECG changes or significant arrhythmias were reported. We concluded for ventricular repolarisation abnormalities during maximal exercise testing due to electrocardiographic findings in an asymptomatic athlete without any coronary artery disease. According to the Italian cardiological guidelines for competitive sports (COCIS 2009), we recommended the patient avoid physical overload and sport practice [12].

## DISCUSSION

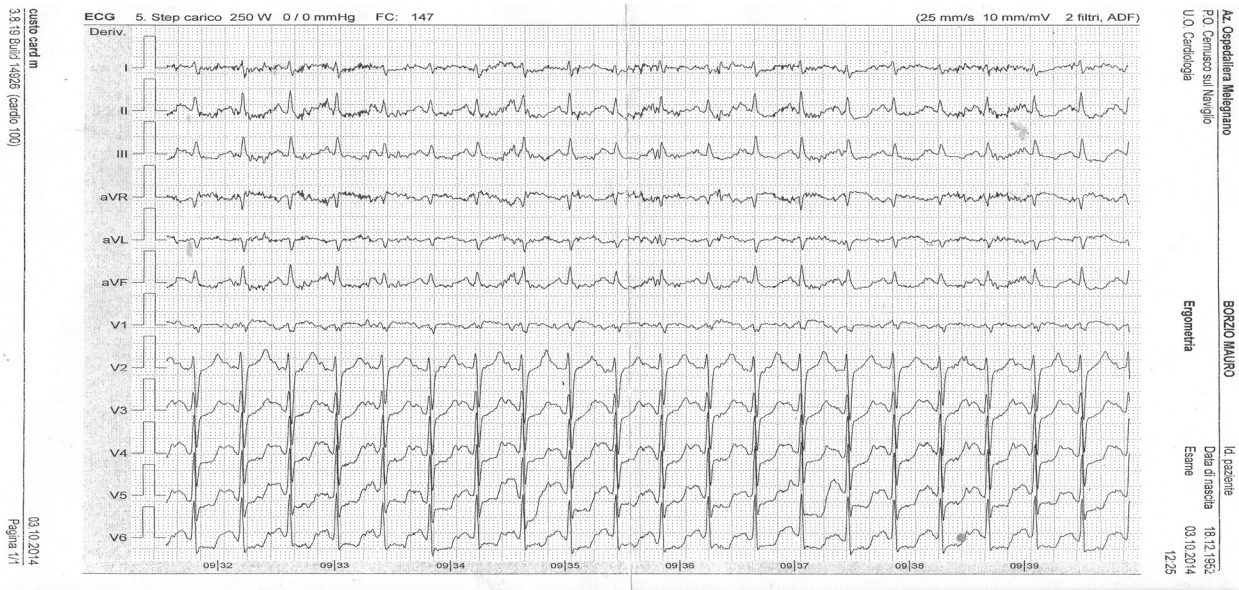
In our case, we performed a cardiovascular evaluation, including an exercise test to exhaustion. The Italian Society of Sports Cardiology Guidelines (COCIS 2009) suggests this approach for all subjects over 40 before granting them eligibility to participate in competitive sports [12]. Due to an ST segment depression recorded during the cardiac exercise stress test, we also performed a CCTA that showed two myocardial bridges of the left coronary artery. Moreover, we observed no atherosclerotic plaque of the coro-

nary lumen. A dobutamine stress echocardiography and a bicycle stress echocardiography were also normal. However, as the COCIS 2009 Guidelines suggest, we recommended our patient avoid physical overload and sport practice.

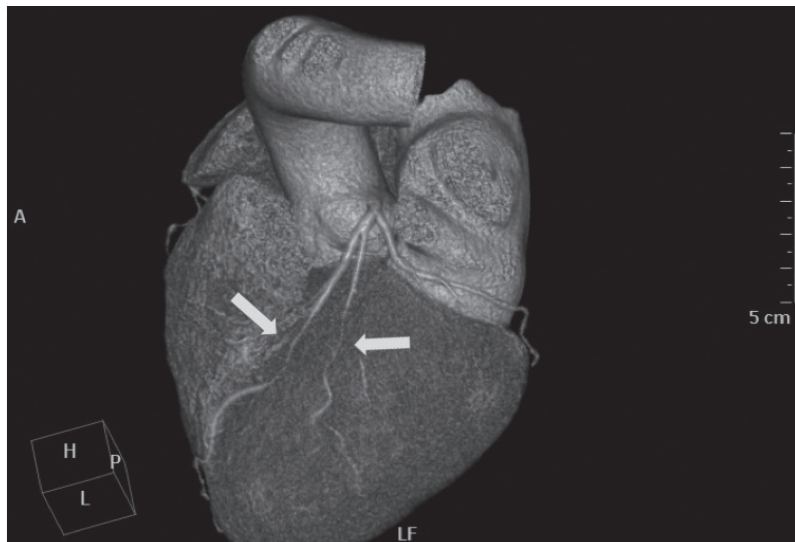
Our assessment for the fitness of competitive sport was difficult; on one side in literature, myocardial bridging is regarded as a common anatomic variant rather than a congenital anomaly [13]. On the other side, there are some reports showing that myocardial ischemia and myocardial infarction or sudden death could be caused by myocardial bridging [14]. Intramyocardial bridging is a recognised cause of sudden death in athletes [15]. Kersemans et al. report a case of a 24-year-old professional cyclist with myocardial bridging who presented with a prolonged episode of exertional chest pain, ST segment inversion in the anterior ECG leads and anterior hypokinesia on an echocardiography. However, the authors conclude that the myocardial bridge had no significant hemodynamic effect in baseline conditions and the evaluation of the hemodynamic significance of frequently encountered mild-to-moderate cases of myocardial bridging can present diagnostic difficulties for the clinician [9]. Myocardial bridging is a frequent finding during coronary angiography, because 'milking' is limited to systole and does not impair myocardial perfusion that occurs in diastole [9]. Quaranta et al. present a case of an asymptomatic competitive athlete who was diagnosed with myocarditis and, as an incidental finding, a myocardial bridge. They illustrate the relevance of anamnesis and combined techniques such as ECG, echocardiography and CCTA for this type of diagnosis [16]. Bolognesi et al. describe a case of a middle-aged competitive cyclist who presented a positive exercise test and was withdrawn from competition for the coexistence of a deep and long myocardial bridge at high risk of sudden death. According to Bolognesi et al., however, anomalies such as myocardial bridging should be regarded as a particular group of congenital disorders whose manifestations and pathophysiological me-

chanisms are highly variable. Interestingly, the authors hypothesised an association between the length and depth of the intramural coronary artery segment and the risk of sudden cardiac death [17]. Therefore, according to the authors, when ECG findings of inducible silent myocardial ischemia at low workload are present in healthy athletes, it is mandatory to perform a CCTA in order to identify a high-risk profile [17]. In Italy, the COCIS 2009 Guidelines include myocardial bridging among the congenital abnormalities of the coronary circulation and state that this disease precludes suitability for competitive sports, as such athletes are at risk of sudden death, even in the absence of signs of inducible stress test ischemia. According to Bolognesi et al., however, this cannot be considered an absolute dogma [17]. Indeed, ECG abnormalities may be detected that represent expression of an underlying heart disease that puts the athlete at risk of arrhythmic cardiac arrest during sports, but physiological patterns that should cause no alarm are also frequent [18]. For instance, Josephson et al. demonstrated positive ST segment responses have a modestly greater (although still relatively low) risk for future ischemic events than negative responses [19]. Moreover, the presence of a myocardial bridge is common in athletes, but only in few cases can this anomaly be deemed a risk of sudden death [17].

In conclusion, it is important to recognise the clinical significance of inducible ischemia detected during exercise testing in asymptomatic athletes and to perform all diagnostic tests. It is also possible to issue a temporary sports eligibility when a diagnostic doubt persists after such tests. However, further research is needed to understand the relationship between the length and depth of the intramural coronary artery segment and the risk of sudden cardiac death in order to better define a protocol for the assessment of fitness for competitive sports.



**Figure 1.** Cardiac exercise stress test showing a 2 mm ST-segment inversion in the anterior leads.



**Figure 2.** Coronary computed tomography angiography (CCTA) showing two myocardial bridges of the left coronary artery.

## References

1. Angelini P, Velasco JA, Flamm S. Coronary anomalies: incidence, pathophysiology, and clinical relevance. *Circulation*. 2002;105:2449–2454.
2. Ebel R, Ge J, Mohlenkamp S. Myocardial bridging: a congenital variant as an anatomic risk factor for myocardial infarction? *Circulation*. 2009;120:357–359.
3. Tauth J, Sullebarger JT. Myocardial infarction associated with myocardial bridging: case history and review of the literature. *Cath Cardiovasc Diagn*. 1997;40:364–367.
4. Marchionni N, Chechi T, Falai M, Margheri M, Fumagalli S. Myocardial stunning associated with a myocardial bridge. *Int J Cardiol*. 2002;82:65–67.
5. Feld H, Guadanino V, Hollander G, Greengart A, Lichstein E, Shani J. Exercise-induced ventricular tachycardia in association with a myocardial bridge. *Chest*. 1991;99:1295–1296.
6. Cutler D, Wallace JM. Myocardial bridging in a young patient with sudden death. *Clin Cardiol*. 1997;20:581–583.
7. Lee MS, Chen CH. Myocardial bridging: An up-to-date review. *J Invasive Cardiol*. 2015;27(1):521–528.
8. Yetman AT, McCrindle BW, MacDonald C, Freedom RM, Gow R. Myocardial bridging in children with hypertrophic cardiomyopathy- a risk factor for sudden death. *N Engl J Med*. 1998;339:1201–1209.
9. Kersemans M, Van Heuverswyn F, De Pauw M, Gheeraert P, Taeymans Y, Drieghe B. Hemodynamic effect of myocardial bridging. *Circ Cardiovasc Intervent*. 2009;2:361–362.
10. Van Camp SP, Bloor CM, Mueller FO, Cantu RC, Olson HG. Nontraumatic sports death in high school and college athletes. *Med Sci Sports Exerc*. 1995;27:641–647.
11. Eckart RE, Scoville SL, Campbell CL, Shry EA, Stajduhar KC, Potter RN, et al. Sudden death in young adults: a 25-year review of autopsies in military recruits. *Ann Intern Med*. 2004;141:829–834.
12. COCIS. *Protocolli cardiologici per il giudizio di idoneità allo sport agonistico*. Rome: Casa Editrice Scientifica Internazionale; 2009, pp. 1–188.
13. Konen E, Goitein O, Di Segni E. Myocardial bridging, a common anatomical variant rather than a congenital anomaly. *Semin Ultrasound CT MR*. 2008;29:195–203.
14. Vales L, Kanei Y, Fox J. Coronary artery occlusion and myocardial infarction caused by vasospasm within a myocardial bridge. *J Invasive Cardiol*. 2010;22:E67–E69.
15. Basso C, Thiene G, Mackey-Bojack S, Frigo AC, Corrado D, Maron BJ. Myocardial bridging, a frequent component of the hypertrophic cardiomyopathy phenotype, lacks systematic association with sudden cardiac death. *Eur Heart J*. 2009;30:1627–1634.
16. Quaranta F, Guerra E, Sperandii F, De Santis F, Pigozzi F, Calò L, et al. Myocarditis in athlete and myocardial bridge: An innocent bystander? *World J Cardiol*. 2015;7(5):293–298.
17. Bolognesi M, Bolognesi D. Asymptomatic st-segment changes in athletes with myocardial bridge: unremarkable or dangerous? *Clin Case Rep Rev*. 2015;1(2):27–30. doi: 10.15761/CCRR.1000111.
18. Corrado D, Pelliccia A, Bjørnstad HH, Vanhees L, Biffi A, Borjesson M, et al. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*. 2005;26:516–524.
19. Josephson RA, Shefrin E, Lakatta EG, Brant LJ, Fleg JL. Can serial exercise testing improve the prediction of coronary events in asymptomatic individuals? *Circulation*. 1990;81:20–24.