folia medica

Folia Medica 65(1):177-182
DOI: 10.3897/folmed.65.e72810

Case Report

Rhythm and Conduction Complications after COVID-19 Infection in Physiological Hypertrophy of Myocardium (Athlete's Heart)

Ludmila Vladimirova-Kitova[1,2], Slavi Delchev[3], Spas Ivanov Kitov[1,2]

[1] *First Department of Internal Diseases, Section of Cardiology, Faculty of Medicine, Medical University of Plovdiv, Plovdiv, Bulgaria*

[2] *Clinic of Cardiology, St. George University Hospital, Plovdiv, Bulgaria*

[3] *Department of Human Anatomy, Histology and Embryology, Faculty of Medicine, Medical University of Plovdiv, Plovdiv, Bulgaria*

Corresponding author: Ludmila Vladimirova-Kitova, First Department of Internal Diseases, Section of Cardiology, Faculty of Medicine, Medical University of Plovdiv, 15A Vassil Aprilov Blvd., 4002 Plovdiv, Bulgaria; Email: kitov@vip.bg; Tel.: +359 888 428 255

Received: 10 Aug 2021 ♦ **Accepted:** 23 Nov 2021 ♦ **Published:** 28 Feb 2023

Citation: Vladimirova-Kitova L, Delchev S, Kitov, SI. Rhythm and conduction complications after COVID-19 infection in physiological hypertrophy of myocardium (athlete's heart). Folia Med (Plovdiv) 2023;65(1):177-182. doi: 10.3897/folmed.65.e72810.

Abstract

The term 'athletic heart syndrome' (AHS) is used to describe specific circulatory and morphological changes in individuals who participate in sports competitions. The syndrome is characterized by normal cardiac function and reversible myocardial remodelling.

The incidence and severity of the post-COVID-19 cardiac pathology in active athletes are so far unclear. One of the complications involving the heart is myocarditis. We present a case of a 23-year-old rower after having a moderate COVID-19 infection. Electrocardiograms showed evidence of a shift in conduction and rhythm disturbances ranging from Group 1 (normal ECG findings) to Group 2 (abnormal ECG findings) on the background of an AHS. Echocardiography (with new methods of evaluating deformity – Global Longitudinal Strain) revealed an area with mildly reduced left ventricular deformity around the apex. To assess the subtle alterations in the myocardium, magnetic resonance imaging was used and focal myocarditis was detected. In our patient, considering the degree of severity of his COVID-19 infection – a moderate one, a decision was taken to perform a clinical and instrumental reassessment of his cardiovascular complications 6 months after the infection.

This clinical case presents two substantial issues. First, is the AHS more susceptible to rhythm and conduction disturbances after a COVID-19 infection than that of a person who does not actively participate in sports? Second, what the reversibility or the definitive nature of these disturbances is, and how this impacts the prognosis associated with an active sporting activity.

Keywords

athletic heart syndrome, COVID-19 infection, incomplete left bundle branch block, myocarditis, ventricular extrasystoles

INTRODUCTION

Endurance sports (running, cycling, swimming, rowing) are types of aerobic training that trigger changes in the cardiovascular physiology by increasing VO_{2max} consumption, cardiac output, stroke volume, and systolic blood pressure during exercise with an associated fall in heart rate at rest. The result is an increase in cardiac preload but a reduction in the afterload, which leads to volume-loaded left ventricle.[1] The term 'athletic heart syndrome' (AHS) is used to describe specific circulatory and morphological changes in individuals who participate in sports competitions. AHS is characterized by a slow pulse, heart hypertrophy, and ventricular dilation. The cardiac function is normal and the morphological changes are reversible.[2]

Standard echocardiography (EchoCG) has an essential role in differentiating between physiological and pathological ventricular hypertrophy.[3,4] Left ventricular (LV) wall thickness can contribute to distinguishing between athlete's LV hypertrophy and hypertrophic cardiomyopathy (HCM).

The alterations in the structure and function characterizing AHS influence the electrocardiogram (ECG) at rest. Physiological ECG changes have been described in athletes aged 18 to 35, engaged in systematic training.[5,6] Modern interpretation of ECG distributes the changes observed in active athletes into three main groups.[7,8] Group 1 (normal ECG findings) includes common changes associated with the training, such as sinus bradycardia, first degree atrioventricular block, incomplete right bundle branch block (RBBB), early repolarization, etc. Group 2 (abnormal ECG findings) includes uncommon (<5%) and training-unrelated ECG abnormalities: ST-segment depression, pathological Q waves, complete left bundle branch block (LBBB), ventricular arrhythmias, etc. The third 'borderline' group comprises left or right axis deviation, left or right atrial enlargement, and complete RBBB.

The incidence and severity of cardiac pathology in active athletes following a COVID-19 infection are so far unclear.[9] There is a growing concern regarding the complications following a COVID-19 infection. One of the complications involving the heart is myocarditis. According to evidence from literature, it is the main cause of death in people not engaged in sports, either independently (7%) or in combination with involvement of other organs (33%).[10,11] On the other hand, myocarditis is one of the causes of sudden cardiac death (SCD) during training in athletes who have not had COVID-19.[12]

Presence of acute myocarditis with subsequent pathological remodeling is difficult to differentiate from the physiological hypertrophy of myocardium in athletes by using routine methods of study.[13] In focal myocarditis, ECG is not sufficiently definitive. EchoCG and the new methods of evaluating deformity (global longitudinal strain) are the methods of choice in diagnosing cases of post-COVID-19 complications in athletes.[13,14] The gold standard in evaluating the subtle alterations in the myocardium is magnetic resonance imaging (MRI). This method is recommended in athletes who have had mild COVID-19, so that a more precise diagnosis can be established and the risk of fatal complications minimized.[15]

CASE REPORT

A 23-year-old patient with anamnestic data of systematic rowing training for 10 years had no subjective complaints. He had been doing rowing sprint workouts 3 times a week, at a non-professional level, no supplement intake. During a control examination (3 years ago) in year 6 after engaging in rowing sprint workouts the ECG findings were normal: sinus bradycardia (45 beats/min) and incomplete RBBB.

The findings on EchoCG displayed remodeling typical of physiological LV hypertrophy: LV mass – 322.25 g, LV mass index – 152.73 g/m^2, LV end-diastolic volume – 192 mL, interventricular septum thickness at end-diastole (IVSd) – 12.15 mm), LV EF 66%, LV SV 45 mL, left ventricular posterior wall thickness at end-diastole LVPWd – 11.45 mm). Reported dimensions of the right ventricle (RV) were: RV free wall 38 mm, RV basal diameter 2.7 cm, RV base to apex diameter – 7 cm), transmitral E/A ratio – 2.276.

At the beginning of March 2021 the patient had a mild COVID-19 infection (was PCR positive) with a toxic infective syndrome, fever up to 38°C and bronchial pulmonary syndrome. The laboratory results revealed moderate to severe inflammatory process – increased number of white blood cells, elevated C-reactive protein, ferritin, lactate dehydrogenase and hs-Troponin I. The alterations in the pulmonary parenchyma visualized on the computed tomography of lung and mediastinum were pathognomonic for a COVID-19 infection (areas of ground glass opacity, as well as ones characterized by a crazy paving pattern), involving predominantly the left lung.

A follow-up EchoCG was performed, which confirmed the findings of the previous one – signs of physiological myocardial hypertrophy: LV mass – 298.12 g (cut-off for men 115 g/m^2), LV mass index – 130.44 g/m^2, LV end-diastolic diameter – 4.26 cm, LV end-systolic diameter 2.73 cm, LV ejection fraction (LVEF) – 65.8%, interventricular septum thickness at end-diastole (IVSd) – 1.14 cm, left ventricular posterior wall thickness at end-diastole (LVPWd) – 1.14 cm ***(Fig. 1A)****.*

The diastolic function of LV (LV filling during diastole) is determined by the ratio of E (early filling of LV) to A (loading by left atrium systole). The E/A index is referred to as the compliance of the LV and must be greater than 2. The evaluation of the mitral valve diastolic blood flow demonstrated a supranormal transmitral E/A ratio – 2.276 (>2) ***(Fig. 1B)****.*

The tissue Doppler imaging revealed increased e' velocity in the septal (e' sept = 0.172 m/s), and to a lesser extent, the lateral segments of left ventricle (e' lat = 0.122 m/s), ***(Figs 1C, 1D)****. Low values were found on calculating E/e' – 5.30.*

The ultrasound indices of the right cavities revealed normal size as well as parameters for normal venous pressure on the right. The parameters of the right ventricle were as follows: RV free wall 0.49 cm, RV basal diameter 2.7 cm, RV base to apex diameter – 7 cm. The inferior vena cava (IVC) parameters at the right atrium infusion site were IVC size 2.5 mm, IVC respiratory reactivity 55%.

The follow-up ECG recordings one month after the COVID-19 infection disclosed occurrence of a second hemiblock– an incomplete LBBB and high-grade ventricular extrasystoles in addition to the incomplete RBBB. A 24-hour Holter ECG was recorded one month after recovery from COVID-19, which did not reveal heart rate below 40 beats/min. Occurrence of a new incomplete LBBB was confirmed, side by side with the presence of the old incomplete RBBB. Occurrence of new ventricular rhythm disturbances was recorded as well – 3 421 ventricular extrasystoles, monotopic up to IV b grade by the Lown system – 25 episodes of ventricular volleys ***(Fig. 2)****, two episodes of short-term low-fre-*

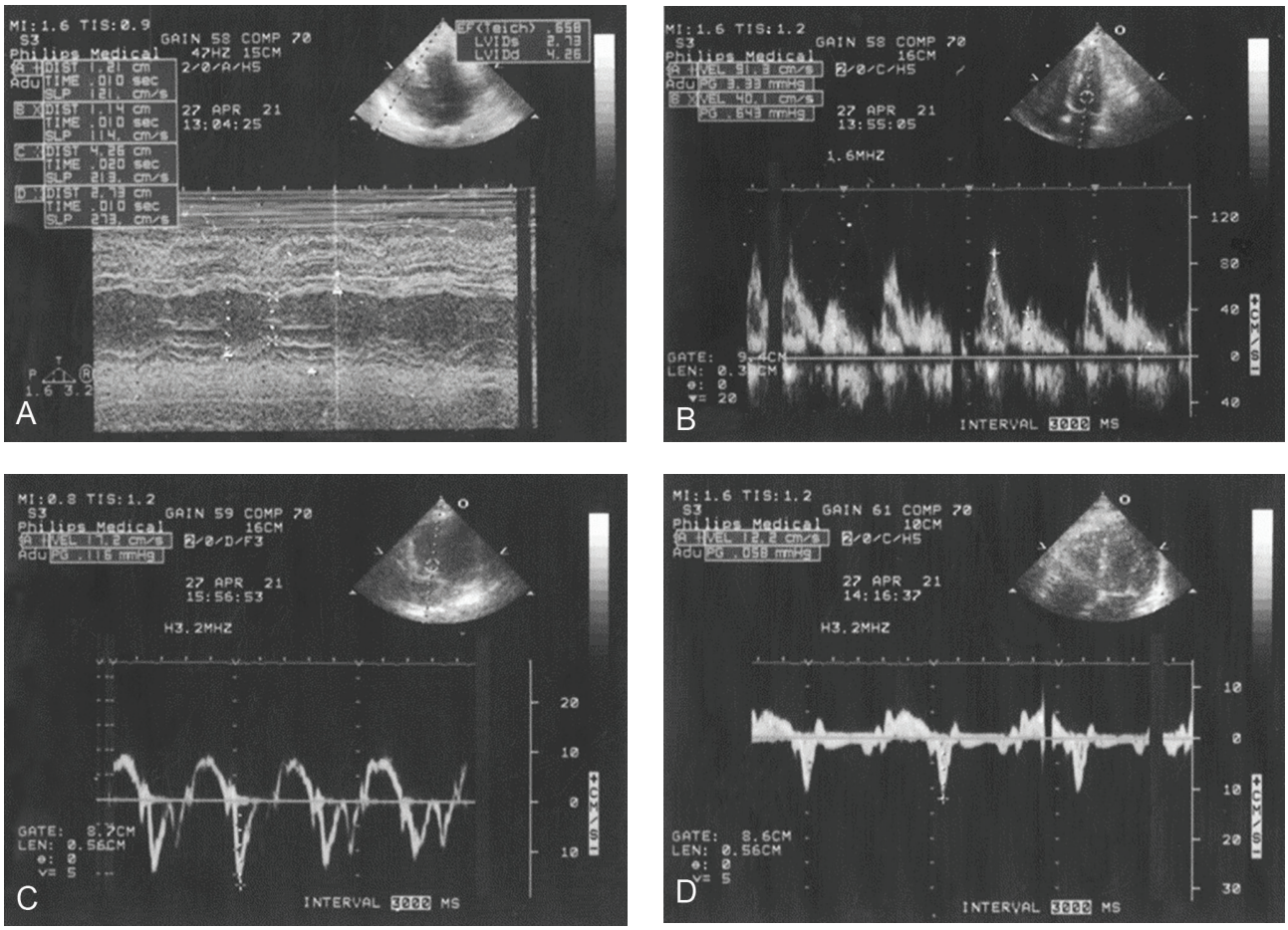

Figure 1. (**A**) EchoCG of a 23-year-old patient with evidence of physiological LV hypertrophy; (**B**) 183 diastolic blood flow determined by Pulse-Doppler of the mitral valve; (**C**) tissue Doppler in the septal 184; (**D**) lateral segments of the left ventricle.

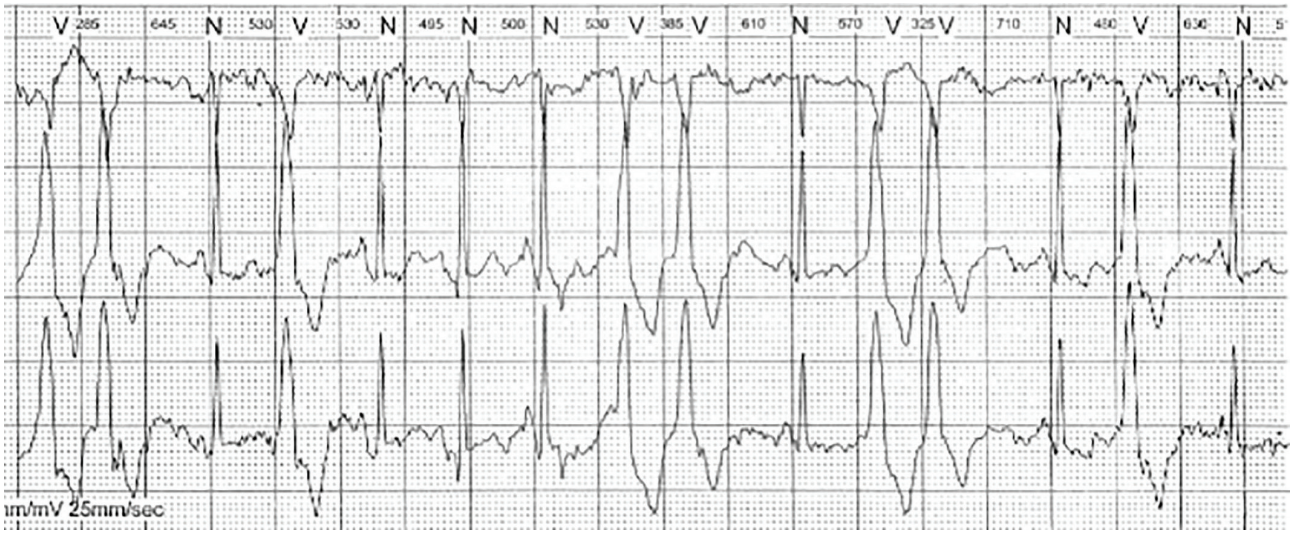

Figure 2. A 24-hour Holter ECG recording of the patient following the COVID-19 infection - ventricular volleys.

quency ventricular tachycardia (heart rate 100 beats/min). The ventricular extrasystoles were managed by amiodarone.

The follow-up EchoCG revealed no dynamics in the ejection fraction of heart (LVEF%). Only the Global Longitudinal Strain-mean marked an area with a mildly reduced LV deformity around the apex. The Global Longitudinal Strain-mean was slightly reduced below the normal values – 19% (reference level ≥20%) ***(Fig. 3)****.*

The EchoCG findings gave us grounds to perform magnetic resonance imaging (MRI), which is a gold standard in

Folia Medica

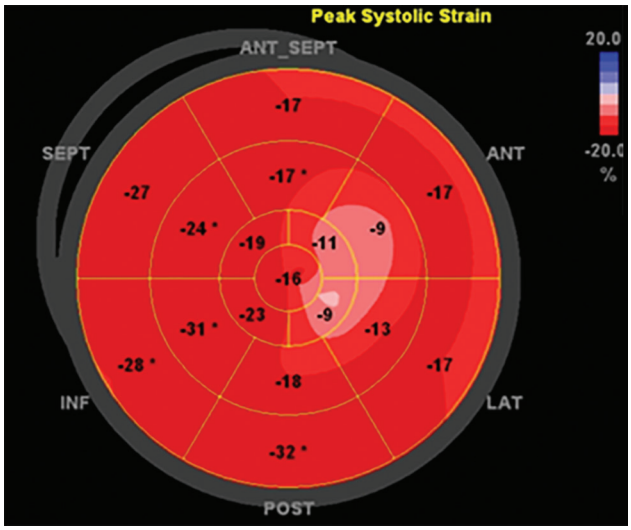

Figure 3. Global Longitudinal Strain of the left ventricle.

assessing morphological alterations in the myocardium. In the first post-infection month, the findings revealed that the patient had had a focal myocarditis in the apical segment of left ventricle ***(Fig. 4)****.*

In the second month following the COVID-19 infection, the rhythm disturbances were controlled by amiodarone; however, the new conduction disturbance – an incomplete LBBB persisted, as well as the old incomplete RBBB.

DISCUSSION

The case presented here demonstrates typical remodeling of the athletic heart syndrome in a rower with 10 years of sporting activities and alterations in the type of conduction and rhythm disturbances, which occurred after COVID-19 infection. The AHS was confirmed by the EchoCG parameters presented. The myocardial hypertrophy observed involved equally all segments of the LV, in contrast to hypertrophic cardiomyopathy, in which the basal segments are predominantly involved.[16] The Global Longitudinal Strain-mean before the COVID-19 infection demonstrated normal values (21.3%), which indicates preserved ventricular deformity.[14]

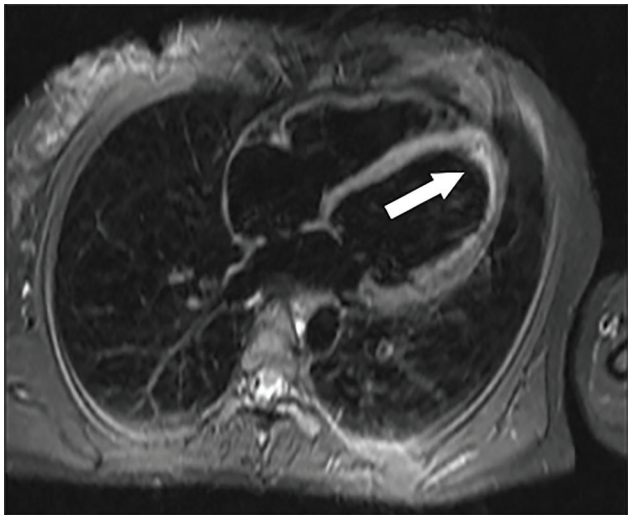

Figure 4. Cardiac MRI one month after COVID-19. Arrow - focal myocarditis in the apical segment of left ventricle.

The compliance of the ventricle is determined by the structural properties of the cardiac muscle (e.g., muscle fibers and their orientation, and connective tissue), as well as by the state of ventricular contraction and relaxation. Cardiac disease conditions, such as different forms of restrictive cardiomyopathy, lead to a decrease in ventricular compliance. In cases of physiological hypertrophy of myocardium, as the one presented here (AHS), E/A ratio is over 2, which is a sign of normal LV loading.

The post-infection occurrence of rhythm disturbances in the patient (ventricular extrasystoles up to IV b grade by the Lown system), accompanied by a conduction disturbance, are associated with the COVID-19 infection. This fact is confirmed by the MRI findings, which revealed a chronic inflammatory process at the apex of left ventricle resulting from focal myocarditis. The presence of an incomplete LBBB is definitive in nature in the second month following infection. Other possible diagnoses discussed in this particular patient were: coronary artery disease (no clinical evidence, no change in ECG findings or enzyme level), bradycardia-associated extrasystoles, dyselectronemia, and hypertrophic cardiomyopathy.

As reported in literature, the screening investigation of athletes after a COVID-19 infection has shown that the incidence of inflammatory complications of myocardium is less than 1%, and in such cases sporting activities should be discontinued for a minimum of three months.[17] Afterwards, reassessment of the morphological and functional alterations have to be made.[18] If the results of the investigations are favorable, the patient can return to moderate load training.[19] In our patient, considering the severity of his COVID-19 infection – a moderate one, a decision was taken to perform a clinical and instrumental reassessment of his cardiovascular complications in the 6th month following the infection. The reassessment will have to include an ECG, an EchoCG, and a 24-hour Holter; afterwards, based on the data obtained, a cardiopulmonary exercise test (CPET) can follow if pertinent, as well as a stress EchoCG and MRI. In case no pathological findings are obtained, the athlete can return to training. If a clinically significant arrhythmia is found, the sporting activities have to be discontinued for a period of 3 – 6 months, after which investigations have to be performed again. The likelihood of the patient having to discontinue his active sporting activities is not excluded, for the purposes of preventing SCD.

The case presented here places the accent on a very important and still not clarified issue associated with having a COVID-19 infection on the background of an AHS. The rhythm disturbances that occur after recovery (ventricular extrasystoles IV b grade by the Lown system and a conduction disturbance – incomplete LBBB) on the background of

an AHS and most likely resulting from the focal myocarditis, change the risk category of the athlete from Group 1 (normal ECG findings) to Group 2 (abnormal ECG findings). An autonomic dysfunction possibly resulting from the COVID-19 infection could also be the cause of the newly occurring hemoblock.[20] Although it is accepted that the AHS is a physiological alteration, some scientists are of the opinion that intensive training can lead to development of malignant ventricular arrhythmias and may be associated with SCD.[6,12] On that background and after a COVID-19 infection, the risk of rhythm and conduction disturbances is likely to be raised, which places at risk further participation in sporting contests. It is appropriate that the patient be monitored before returning to active training and the intensity of the exercise stress should be reduced.

This clinical case presents two substantial issues. First, is the athlete's heart more susceptible to rhythm and conduction disturbances after COVID-19 infection from that of a person not actively engaged in sports? Second, is the nature of these disturbances reversible or definitive, and in what way does it influence the prognosis associated with an active sporting activity? Extensive clinical studies should be conducted in the future to find the answer to these questions.

CONCLUSIONS

Instrumental studies (ECG, EchoCG, MRI) have shown evidence of shift in the conduction and rhythm complications from Group 1 (normal ECG findings) to Group 2 (abnormal ECG findings) on the background of physiological hypertrophy of heart in a 23-year-old rower following a moderate in severity COVID-19 infection. Following recovery, reassessment of the cardiovascular status must be made, both at rest and during exercise. This would prevent the occurrence of fatal events such as SCD when the athlete returns to active sporting activities. In what way a COVID-19 infection associated with myocardial involvement alters the prognosis of athlete's heart syndrome remains a controversial issue.

Author contributions

L.V-K. was responsible for writing the manuscript and took care of the patient. S.D. drafted the initial manuscript, performed the literature review, provided critical feedback, helped format the manuscript, and edited the final version. The published version of the manuscript has been read and approved by all authors. These authors contributed equally to this work and share first authorship.

REFERENCES

1. Maron B, Peliccia A. The heart of trained athletes; cardiac remodeling and the risk of sports, including sudden death. Circulation 2006; 114(15):1633–44.
2. Pelliccia A, Maron BJ, De Luca, et al. Remodeling of left ventricular hypertrophy in elite athletes after long-term deconditioning. Circulation 2002; 105(8):944–9.
3. Galderisi M, Cardim N, D'Andrea A, et al. The multi-modality cardiac imaging approach to the athlete's heart: an expert consensus of the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging 2015; 16:353.
4. Augustine DX, Howard L. Left ventricular hypertrophy in athletes: differentiating physiology from pathology. Curr Treat Options Cardiovasc Med 2018; 20:96.
5. Corrado D, Pelliccia A, Heidbuchel H, et al. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. Eur Heart J 2010; 31(2):243–59.
6. Sharma S, Drezner JA, Baggish A, et al. International recommendations for electrocardiographic interpretation in athletes. J Am Coll Cardiol 2017; 69(8):1057–75.
7. Corrado D, Calore C, Zorzi A, et al. Improving the interpretation of the athlete's electrocardiogram. Eur Heart J 2013; 34(47):3606–9.
8. Drezner JA. 18 highlights from the International Criteria for ECG interpretation in athletes. Br J Sports Med 2020; 54(4):197–9.
9. Kim JH, Levine BD, Phelan D, et al. Coronavirus disease 2019 and the athletic heart: emerging perspectives on pathology, risks, and return to play. JAMA Cardiol 2021; 6(2):219–27.
10. Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel Coronavirus-infected pneumonia in Wuhan, China. JAMA 2020; 323(11):1061–9.
11. Driggin E, Madhavan MV, Bikdeli B, et al. Cardiovascular considerations for patients, health care workers, and health systems during the COVID-19 pandemic. J Am Coll Cardiol 2020; 75(18):2352–71.
12. D'Silva A, Sharma S. Exercise, the athlete's heart, and sudden cardiac death. Phys Sportsmed 2014; 42(2):100–13.
13. Phelan D, Kim JH, Elliott MD, et al. Screening of potential cardiac involvement in competitive athletes recovering from COVID-19: An expert consensus statement. JACC Cardiovasc Imaging 2020; 13(12):2635–52.
14. Caselli S, Montesanti D, Autore C, et al. Patterns of left ventricular longitudinal strain and strain rate in Olympic athletes. J Am Soc Echocardiogr 2015; 28(2):245–53.
15. Hawryluk M. What Covid means for the athlete's heart. Kaiser Health News 2021; Available from: https://khn.org/news/article/what-covid-means-for-athlete-heart-how-post-covid-19-risks-change-sports/
16. Kreso A, Barakovic F, Medjedovic S, et al. Echocardiography differences between athlete's heart hearth and hypertrophic cardiomyopathy. Acta Inform Med 2015; 23(5):276–9.
17. Martinez MW, Tucker AM, Bloom OJ, et al. Prevalence of inflammatory heart disease among professional athletes with prior Covid-19 infection who received systematic return-to-play cardiac screening. JAMA Cardiol 2021; 6(7):745–52.
18. Dores H, Cardim N. Return to play after COVID-19: a sport cardiologist's view. Br J Sports Med 2020; 54:1125–35.
19. Mohr M, Nassis GP, Brito J, et al. Return to elite football after the COVID-19 lockdown. Manag Sport Leis 2022; 27(1-2):172–80. DOI: 10.1080/23750472.2020.1768635.
20. Barizien N, Le Guen M, Russel S, et al. Clinical characterization of dysautonomia in long COVID-19 patients. Sci Rep 2021; 11(1):14042

Осложнения ритма и проводимости после инфицирования COVID-19 при физиологической гипертрофии миокарда (синдром спортивного сердца)

Людмила Владимирова-Китова[1,2], Слави Делчев[3], Спас Иванов Китов[1,2]

[1] *Первая кафедра внутренних болезней, Секция кардиологии, Факультет медицины, Медицинский университет – Пловдив, Пловдив, Болгария*

[2] *Клиника кардиологии, УМБАЛ „Свети Георги", Пловдив, Болгария*

[3] *Кафедра анатомии человека, гистологии и эмбриологии, Факультет медицины, Медицинский университет – Пловдив, Пловдив, Болгария*

Адрес для корреспонденции: Людмила Владимирова-Китова, Первая кафедра внутренних болезней, Секция кардиологии, Факультет медицины, Медицинский университет – Пловдив, бул. „Васил Априлов" №15А, 4002 Пловдив, Болгария; Email: kitov@vip.bg; тел.: +359 888 428 255

Дата получения: 10 августа 2021 ♦ **Дата приемки:** 23 ноября 2021 ♦ **Дата публикации:** 28 февраля 2023

Образец цитирования: Vladimirova-Kitova L, Delchev S, Kitov, SI. Rhythm and conduction complications after COVID-19 infection in physiological hypertrophy of myocardium (athlete's heart). Folia Med (Plovdiv) 2023;65(1):177-182. doi: 10.3897/folmed.65.e72810.

Резюме

Термин „синдром спортивного сердца" (ССС) используется для описания специфических циркуляторных и морфологических изменений у лиц, участвующих в спортивных соревнованиях. Синдром характеризуется нормальной сердечной функцией и обратимым ремоделированием миокарда.

Частота и тяжесть сердечной патологии после COVID-19 у активных спортсменов до сих пор неясны. Одним из осложнений, связанных с сердцем, является миокардит. Мы представляем случай 23-летнего гребца, перенёсшего умеренную инфекцию COVID-19. На электрокардиограмме отмечен сдвиг нарушений проводимости и ритма от 1-й группы (нормальные показатели ЭКГ) до 2-й группы (отклонения от нормы ЭКГ) на фоне ССС. Эхокардиография (с новым методом оценки деформации – Global Longitudinal Strain) выявила область с умеренно сниженной деформацией левого желудочка вокруг верхушки. Для оценки малозаметных изменений в миокарде использовали магнитно-резонансную томографию и выявили очаговый миокардит. У нашего пациента, учитывая степень тяжести инфекции COVID-19 – среднюю, было принято решение о проведении клинико-инструментальной переоценки сердечно-сосудистых осложнений через 6 месяцев после заражения.

Этот клинический случай представляет две существенные проблемы. Во-первых, является ли ССС более восприимчивым к нарушениям ритма и проводимости после заражения COVID-19, чем у человека, который активно не занимается спортом? Во-вторых, какова обратимость или окончательный характер этих нарушений и как это влияет на прогноз, связанный с активной спортивной деятельностью.

Ключевые слова

синдром спортивного сердца, инфекция COVID-19, неполная блокада левой ножки пучка Гиса, миокардит, желудочковая экстрасистолия