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Chapter

Out-of-Hospital Cardiac Arrest in General Population and Sudden Cardiac Death in Athletes

Bettina Nagy, Boldizsár Kiss, Gábor Áron Fülöp and Endre Zima

Abstract

Sudden cardiac death (SCD) is still one of the leading causes of cardiovascular death in the developed countries. The incidence of out-of-hospital cardiac arrest in Europe varies from 67 to 170 per 100,000 population. The chain of survival will be described in detailed steps. We are going to summarize the treatment options for sudden cardiac arrest from recognition of SCD to resuscitation and post cardiac arrest care. The role of awereness and Automated External Defibrillator and Public Access Defibrillation (AED-PAD) programs will be discussed in brief. SCD is one of the most common causes of death among athletes. Sport can trigger SCD in individuals who already have unknown form of heart disease. Our aim was to detail the underlying causes of SCD in athletes and to identify the possible screening techniques. Existing disease (e.g., myocardial hypertrophy, fibrosis) can be seen as a substrate, and sport as a trigger can cause arrhythmias, increased catecholamine release, acidosis, and dehydration. We will highlight the importance of sports medicine and periodic examination in screening for these conditions. Depending on the etiology, this may include exercise ECG, Holter monitor, CT, MR, echocardiography, and coronagraphy. We are going to conclude the new recommendations for COVID-19 post-infection care for athletes.

Keywords: sudden cardiac death, out-of-hospital cardiac arrest, in-hospital cardiac arrest, resuscitation, post cardiac arrest care, COVID-19 infection, athletes

1. Introduction

Despite the advances in diagnosis and treatment during the past decades, sudden cardiac death (SCD) is one of the most common causes of cardiovascular mortality. There are several definitions of SCD in the literature [1]. According to the most widely used one, sudden cardiac death is a set of symptoms in which natural, unexpected death occurs within 1 hour of the onset of symptoms. However, this definition applies only if the death itself has an eyewitness. Failing this, SCD is considered to be the cause of death if the person was still being well, 24 hours before the body was found [2–4].

Based on the location of SCD, one can divide out-of-hospital (OHCA) and in-hospital sudden cardiac arrest (IHCA). Data on adult mortality from sudden cardiac

death due to cardiovascular disease could not be adequately characterized for a long time. This is mainly due to a lack of well-designed clinical research, inaccurate data collection, and an unclear definition.

Sudden cardiac death is rare among athletes. It is a devastating phenomenon, as athletes are associated with the image of a strong, healthy, resilient body. These cases usually get in focus of media publicity, and even if, for a short time, sudden cardiac death gets the spotlight. This is also of great importance to the public because it draws attention to the importance of expertise in resuscitation.

We are going to point out the importance and process of resuscitation and prevention strategies in this chapter. The role of screening of athletes will be discussed.

2. Incidence of out-of-hospital sudden cardiac death (OHCA)

The epidemiological, clinical, and pathological characteristics of out-of-hospital SCD are inadequately defined for several reasons.

First, in a significant proportion of clinical research studies, OHCA is not detected because it occurs unexpectedly. Death certificates, medical histories, consultations with relatives, and questionnaires completed by them are reliable sources of

Figure 1.
The most common causes of OHCA (own flowchart).

information but often show uncertainty about the cause of death. Second, very few studies consider autopsy-based data that determine the cause of sudden cardiac arrest. In all cases, a complete autopsy, including a toxicological and histopathological examination, should be performed, to investigate the possibility of sudden cardiac death [5]. On the other hand, causes of death based on death certificates are inaccurate and, as a consequence, overestimate the incidence of sudden cardiac arrest [6, 7]. Third, emergency documentation often does not include cases of SCD outside the hospital without an eyewitness, and sometimes these medical records are not available. Last, different SCD definitions are used in the studies, making them difficult to compare.

The incidence of sudden cardiac deaths is usually estimated from studies in developed countries. Fortunately, we still have some reliable data regarding the incidence. OHCA is recorded in about 70% of European countries, but unfortunately, the data record is not uniform. The European Registry of Cardiac Arrest (EuReCa) involved 29 countries with an annual incidence of OHCA in Europe of between 67 and 170 per 100,000 population [1]. The causes show significant variations by gender and age. Incidence is 3–4 times higher in men than in women and increases with age [8].

2.1 Etiology of OHCA

Ischemic heart disease is the most common cause of out-of-hospital sudden cardiac death. Causes of death also include cardiomyopathy, cerebrovascular disease, and arrhythmia (see **Figure 1**). In contrast, there are cases where the death due to a sudden cardiac arrest occurs outside the hospital, and this is the first appearance of the disease [3, 9, 10].

3. Awareness

Prevention is separated by definition to primary and secondary prevention. In the case of primary prevention, the goal is to screen patients who are at high risk for SCD but have not had SCD in their lifetime and have not had a malignant arrhythmia. Prevention of sudden cardiac death primarily involves the elimination and treatment of cardiovascular risk factors with smoking cessation, increased physical activity, special diet, treatment of high blood pressure, diabetes, high blood fat, and weight loss in case of obesity. Preventive strategies also aim to define groups or individuals at increased risk of SCD in specific populations. Typically, patients with decreased left ventricular ejection fraction are at high risk for SCD, but a cardiovascular risk assessment of competitive athletes would also be essential.

During secondary prevention, the goal is to further treat patients who have successfully resuscitated after SCD or who have had a malignant arrhythmia and to prevent another arrhythmia.

4. Treatment of circulatory arrest: resuscitation

The formal professional opinion on resuscitation is published every 5 years by the European Resuscitation Council (ERC) in the form of a recommendation, following the scientific preparation of the International Liaison Committee on Resuscitation (ILCOR). The ERC Directive 2021, published this year, is currently in force [11].

According to the terminology of the ILCOR Consensus on Science with Treatment Recommendations (CoSTR), resuscitation should be initiated in any person who is "unresponsive and absent or abnormal in breathing [12]. This terminology is also included in the most recent basic life support (BLS) directive 2021 [11].

4.1 Basic life support (BLS)

In Hungary, the average arrival of an ambulance is 5–8 minutes, and the first shock is delivered 8–11 minutes after the announcement [13]. During this time, the survival of the patient is in the hands of the layman. The use of BLS and the use of an automated external defibrillator (AED) can significantly improve long-term survival.

Patient survival is determined by elements of the chain of survival from circulatory arrest. It includes early recognition of cardiac arrest and calling for help, circulatory maintenance with high-quality chest compression and rescue breathing, early defibrillation (if necessary), and post-cardiac arrest intensive care after achieving return of spontaneous circulation (ROSC) (**Figure 2**).

The survival chain shown in the figure symbolizes the steps required to perform the circulatory arrest. The first three steps on site can be done by anyone. Abbreviations: AED: Automated External Defibrillator.

4.1.1 How to recognize a sudden cardiac arrest?

- Any person who does not respond and breathing is absent or is abnormal, one should consider that the patient is in cardiac arrest. CPR should be started immediately.

- Slow, agonal breathing should be considered a sign of sudden cardiac arrest, as in almost 40% of cases, this symptom occurs immediately after circulatory arrest.

- Short-term, seizure activity may occur at the onset of cardiac arrest, the person should be examined after the termination of the seizure: if he does not respond and is short of breath or abnormal, CPR should be started.

4.1.2 What to do in case of sudden cardiac arrest?

How to call for help?

- The National Ambulance Service must be notified immediately for resuscitation.

- If the eyewitness is alone and has a mobile phone after the emergency call, one should turn on the speaker of the phone and start CPR immediately.

- If the eyewitness is alone and does not have a cell phone, the patient should first be left there until he or she notifies the ambulance service, and then CPR should begin as soon as possible.
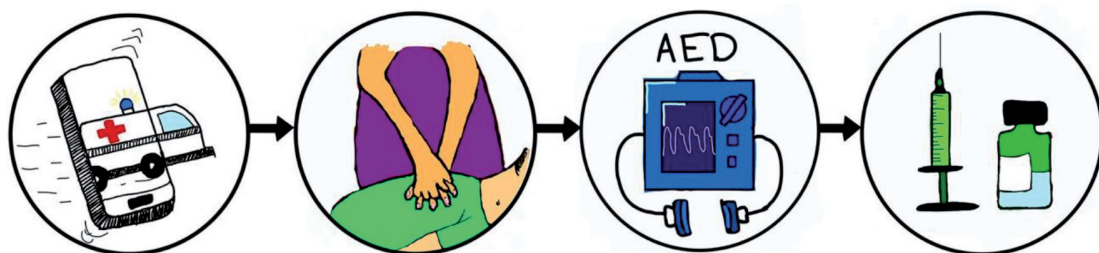
Figure 2.
The survival chain (own flowchart).

How to perform quality chest compression?

• Compression should be performed on the lower half of the patient's sternum.

• Depth can be approximately 5 cm, but not more than 6 cm.

• Its frequency is 100–120 compression/minute.

• Full chest release is required, do not lean on the chest.

• If possible, it should be performed on a patient lying on a hard surface.

How to perform rescue breath?

• After 30 chest compressions, two rescue breaths are to be given.

• If rescue breathing is not possible, chest compression must be continued without interruption.

• In case of risk of infection (for example: during the COVID-19 epidemic), continuous chest compression is required until professional help is obtained, without rescue breathing.

4.1.3 AED-PAD program

Local-regional AED-PAD (automated external defibrillator–public access defibrillation) programs are essential to reduce time till return of spontaneous circulation, the central nervous system ischemic injury, and to improve survival. In the case of a shockable rhythm, defibrillation within 3–5 minutes of collapse can result in survival of up to 50–70% [13–15]. Delaying defibrillation reduces survival by approximately 10–12% per minute. AEDs are easy to use, but proper training is required to master them properly. The PAD program aims to reduce the number of deaths due to sudden cardiac arrest in public places. The AED is to be located in high-traffic areas that are accessible to all, e.g., airports, stadiums, schools, shopping malls. It helps laypeople with simple voice instructions during resuscitation. Based on a defined algorithm, it performs a rhythm analysis every 2 minutes and, if it detects a rhythm to be shocked, charges the defibrillation capacitor, and then the resuscitator can deliver the shock with the push of a highlighted button.

4.2 Advanced life support (ALS)

The ERC guidelines are based on the 2020 ILCOR CoSTR. There have been no significant changes to the 2021 adult ALS guidelines [11]. Priority remains on high-quality chest compression with minimal interruption and early defibrillation. Chest compression should be paused only when necessary, for as short a time as possible, in the event of a shock, care should be taken not exceeding 5 seconds. The use of airway management devices must comply with the principle of gradation, i.e., from the simplest to the more complex. There has also been no change in resuscitation drugs. In the case of a non-shockable rhythm, 1 mg of adrenaline should be used as soon as possible, and in the case of a shockable rhythm, an additional 1 mg of adrenaline should be given every 3–5 minutes after the third shock and until the return of spontaneous circulation. If the shockable rhythm cannot be terminated, an iv. bolus of 300 mg amiodarone after the third shock, an additional iv. bolus of

150 mg amiodarone should be given after the fifth shock. During the advanced life support, the aim is to clarify reversible causes according to 4H/4 T: hypoxia, hypo-hyperkalemia, hypovolemia, hypothermia, thromboembolism (coronary, pulmonary), toxin, tamponade, tension pneumothorax. The new guideline recognizes the growing role of bedside point-of-care ultrasound (POCUS) in clarifying the etiology, but stresses the need for a competent handler and to minimize interruptions during chest compression associated with the use of POCUS. The guidelines also reflect the consideration of increasingly evidence-based extracorporeal techniques (eCPR) as rescue therapy to facilitate ALS failure and to facilitate certain interventions (coronary angiography, percutaneous coronary intervention, pulmonary thrombectomy).

5. Post-cardiac arrest syndrome (PCAS)

In 2015, the ERC and the European Society of Intensive Care Medicine developed the first combined resuscitation treatment guidelines, which were jointly published in Resuscitation and Intensive Care Medicine [16]. The latest version, published in 2021, including the directive with new results since 2015, supplemented by further recommendations [17].

In post-resuscitation care, ensuring adequate oxygenation, ventilation, and circulating cardiac output remain one of the cornerstones of care, and cerebral protection is paramount. The Recommendation describes the four key components of the PCAS to be focused on during early treatment that are still accepted today. These include post-circulatory central nervous system damage, post-circulatory myocardial dysfunction, systemic ischemic and reperfusion responses, and persistent underlying disease-causing circulatory arrest.

If the patient's spontaneous circulation has returned, treatment of PCAS is initiated on-site, with the pillars of stabilization of the hemodynamic state, prevention of arrhythmia recurrence, prevention of cellular damage, and normalization of organ perfusion. Following on-site stabilization, patient care will be continued in specialized centers capable of providing full diagnostic and therapeutic care, which will include a high-level intensive care, angiography, and electrophysiology laboratory, CT/MRI, neurology, mechanical circulatory support, and cardiac surgery.

One of the most important elements of intensive PCAS treatment is controlled targeted temperature management, which is essential to prevent hypoxic and further secondary damage to the brain. Neurological damage is exacerbated during hyperpyrexia, epileptiform seizure activities, and hypoglycemia during PCAS [6]. The quality of treatment applied in the early post-resuscitation period significantly determines the outcome, especially concerning neurological recovery [18].
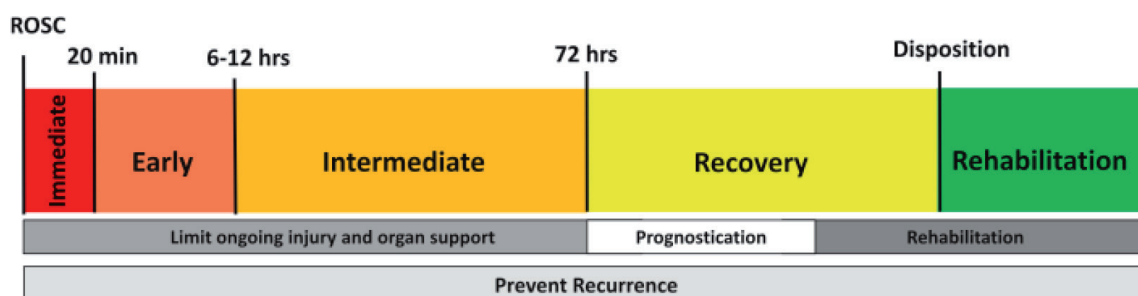

Figure 3.
Phases of return of spontaneous circulation (ROSC) [19].

The consecutive phases of PCAS and the intervals of care are illustrated in **Figure 3**. Time intervals may vary from patient to patient after the first 20 minutes after a spontaneous return of circulation (ROSC), depending on the severity of the PCAS, the rate of recovery, or any progression that may occur. At each stage, care is aimed at limiting ongoing injuries and preventing recurrence of cardiac arrest.

1. Immediate phase: The first 20 minutes after ROSC, part of primary care and delivery to a designated health care facility, stabilization.

2. Early phase: The period between 20 minutes and 6–12 hours after ROSC. Early interventions (causal treatment and initiation of therapeutic hypothermia) may be most effective here.

3. Transitional phase: 12–72 hours, the injury mechanisms are still active, usually aggressive treatment is required.

4. Recovery phase: from about 72 hours to the 7th day. The onset may vary from patient to patient, the timing being influenced by cardiovascular status or the use of therapeutic hypothermia. The prognosis estimation is already reliable at this stage.

5. The rehabilitation phase.

6. Sudden cardiac death in athletes

Sudden cardiac death is rare among athletes, but it is still a devastating phenomenon, as athletes are associated with the image of a strong, healthy, resilient body. These cases usually get into the focus of media attention, and even if, for a short time, SCD gets in the spotlight. This is also of great importance to the public because it draws attention to having expertise in resuscitation.

The likelihood of OHCA in athletes is also influenced by age, gender, the type of sport, and the existing diseases. The incidence is between 1/50,000 and 1/100,000 in young athletes on an annual basis [20]. An athlete is considered to be young under the age 35. Among them, SCD is usually a consequence of some congenital disease. The incidence rises between 1/15,000 and 1/18,000 in elderly athletes [21]. In these cases, sudden cardiac death is usually associated with coronary disease [20]. Similar to the general population, male athletes have a higher risk of SCD compared with their female counterparts (5:1) [21].

The type of sport and the intensity of the activity performed are also influencing factors. Observations suggest that basketball, football, and athletics have the highest risk of SCD [21]. Hobby athletes who have congenital disorders should choose a sport that can be pursued at a constant energy level, avoiding a sudden increase in the heart rate. In addition, it is also worth avoiding extreme environment during sports, such as high temperature and high humidity. These can adversely affect blood pressure and electrolyte balance [20].

In some cases, the underlying cardiovascular disease in athletes remains asymptomatic, and the first symptom is sudden cardiac arrest. Although in the minority of the cases, syncope, chest pain, and ventricular arrhythmias appear as warning symptoms.

Cardiac and non-cardiac diseases leading to sudden cardiac death in athletes, grouped by age, are listed in **Table 1**.

Causes of SCD (under 35 years)	Causes of SCD (over 35 years)
Hypertrophic cardiomyopathy	Coronary disease
Arrhythmogenic right ventricular cardiomyopathy	Aortic dissection
Coronary heart disease	Pulmonary embolism
Aortic aneurysm	Ischemic heart disease
Myocarditis	
Ion channel mutation	
Ruptured aortic aneurysm	
Heart valve disease	
Congenital heart disease	

Table 1.
Causes of SCD based on age.

6.1 Etiology

Hypertrophic cardiomyopathy is a genetic disorder associated with left ventricular hypertrophy that can lead to SCD via ventricular tachycardia/fibrillation. Basso et al. have found that in athletes, arrhythmogenic right ventricular cardiomyopathy (ARVD) causes sudden cardiac arrest even more often than hypertrophic cardiomyopathy. This genetic disease causes a fatty-fibrous remodeling of the right ventricular wall muscle, and sometimes it affects the left ventricle or the interventricular system as well, which can lead to the aneurysm-like dilation of the ventricular free wall [22].

Congenital coronary abnormalities may also be in the background of sudden cardiac death. According to one study, these are responsible for 17% of athletes' deaths from cardiovascular disease. One of the most severe coronary malformations is the origin of the coronary artery from behind the pulmonary trunk, which causes severe symptoms from an early age. However, other lesions with less pronounced symptoms can also cause SCD, especially during increased exercise. In these cases, the coronary can be derived from the sinus on the opposite side, which will make its course abnormal. During exercise, an abnormally grown coronary artery cannot increase blood flow sufficiently, leading to the development of ischemic episodes. As a result, the myocardium will be injured, and the resulting scar tissue will serve as a basis for ventricular arrhythmias and ventricular fibrillation [23].

Pathologists do not find any myocardial lesion that causes SCD in 30% of autopsies. In such cases, cardiac arrest may have been caused by ion channel mutations (long QT syndrome, Brugada syndrome), which may eventually result in ventricular fibrillation. Mutation of ion channels may be alerted by ECG abnormality. The disease is inherited by autosomal dominant, and the mutation is found in genes encoding sodium and potassium channels. Catecholaminergic polymorphic ventricular tachycardia can develop due to a mutation in calcium receptors. In this case, we do not see any difference in the resting ECG. However, with increased sympathetic activity during sports activities, arrhythmias may occur above a heart rate of 120–130/min [22].

Among the valve defects, mitral valve prolapse and aortic stenosis, which are arrhythmogenic, should be highlighted. The most common cause of aortic stenosis is calcareous disease of the aortic valve, with manifestation at a much younger age in addition to bicuspid aortic valves [22].

Acquired cardiac abnormalities may also be among the underlying causes, such as myocarditis due to a viral infection, vascular disease, and aortic dissection [23].

6.2 Why is screening important?

Sport activity can trigger sudden cardiac death in individuals who already have some kind of heart disease. Existing disease (e.g., hypertrophy, fibrosis) can be considered as a substrate and sport may be a trigger, which can cause arrhythmias, increased catecholamine release, acidosis, and dehydration [23].

The American Heart Association and the Sports Cardiology Study Group of the European Society of Cardiology recommend physical examination of high school and college students before a sporting activity in addition to their own and family history recordings. The European guidelines complement this with an ECG registration. The effectiveness of this screening method is supported by observation published by Corrado et al.; they found that the incidence of sudden cardiac death between 1979 and 1980 decreased from 3.6 athletes/100,000 people/year to 0.4 athletes/100,000 people/year for the year 2004 due to the introduction of screening.

7. COVID-19 and sports

In our experience to date, COVID-19 can damage heart itself and its function in at least two basic ways. Directly, when the virus enters the body by binding to human ACE-2 receptors, which are expressed not only in the lungs but also in the myocardium and several other areas of the cardiovascular system. In the early stages of COVID-associated myocarditis, the virus replicates within myocytes, followed by a subacute immunological response including both the T-cell and B-cell immune responses. At this time, the host's immune system may even worsen myocardial damage through cytokine activation and the production of antibodies to viral proteins. In the chronic phase of myocarditis, fibrosis and dilatation of the ventricles may occur. This may manifest as deterioration in pump function and may lead to heart failure [24]. According to a study presented by Linder et al. [25], the presence of Sars-Cov-2 in the myocardium (24/39 autopsy pattern, 61.5%) and active viral replication suggested that direct viral invasion may be more common than previously it was previously assumed. In these studies, the samples were taken from patients treated in hospitals; therefore, their applicability to athletes in the younger population has not been established. The other way in which an intense "cytokine storm" during a severe disease leads to a decrease in heart function, similarly to other forms of sepsis, with mechanisms that are overlapping the development of "stress" or catecholamine-induced cardiomyopathy [25].

Although hospitalization for acute infection in young, healthy individuals is uncommon, there is concern that subclinical myocardial damage may be present in several cases and may manifest as prolonged malaise, or even more may develop malignant arrhythmias and may act as substrate for cardiac death. Exercise during the acute phase of myocarditis caused by a viral infection can accelerate or prolong the disease and trigger malignant and nonmalignant arrhythmias (**Figure 4**) [20].

Therefore, it is important to emphasize that training started before the recommended recovery period of time may contribute to involve cardiovascular system even in asymptomatic individuals. Athletes must undergo a full cardiological examination before returning to daily training. Transthoracic echocardiography is currently considered the first line in post-COVID-19 monitoring in athletes. In addition, exercise ECG and 24-hour Holter monitoring are also a useful help in the "return-to-play" algorithm to detect possible supraventricular and ventricular arrhythmias but have low sensitivity to identify myocarditis [26]. Finally, the levels of serum biomarkers (troponin, CK-MB, BNP) should be monitored thoroughly.

Athletes having positive COVID 19 test or experienced symptoms should abstain from sports and strenuous physical activity for 2 weeks. In the case of myocarditis
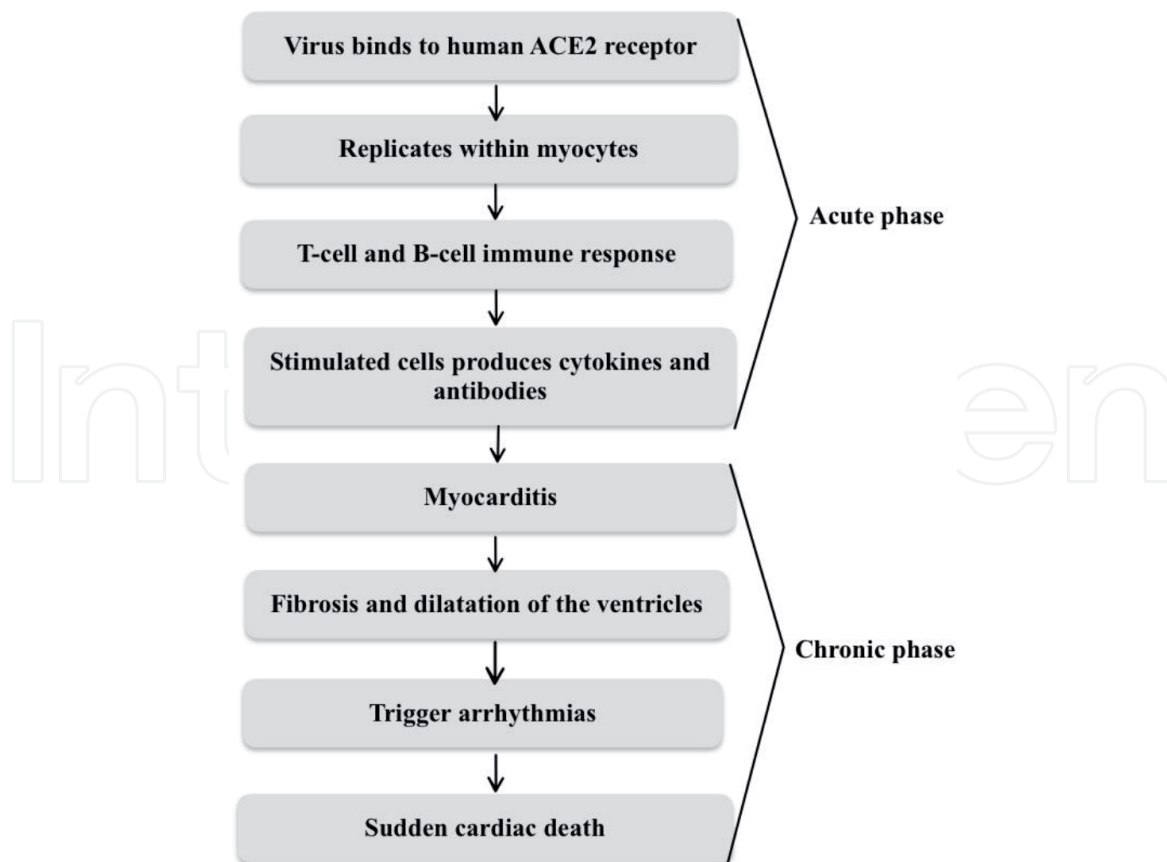
Figure 4.
COVID-19 disease progression in the heart.

or myopericarditis, athletes need to be withheld in sport activity for up to 3–6 months depending on the course and severity of the disease [27]. Thereafter, if the left ventricular systolic function has returned to normal, serum biomarkers for myocardial damage started to decrease, and no clinically significant supraventricular or ventricular arrhythmia has been observed in the 24-hour Holter monitoring or during exercise test, the athlete can return to sport activity [28].

8. Summary

More than half of cardiovascular deaths in the 35–49-year-old population are out-of-hospital non-traumatic sudden cardiac arrest. The rate, for men, is 3–4 times higher than for women; and it gradually increases with age. The classical cardiovascular risk factors are obesity, hypertension, diabetes mellitus, hyperlipidemia, and smoking, in addition to cardiac morphology as known coronary disease or cardiac myopathies can cause sudden cardiac death.

Regular screening significantly reduced the incidence of sudden cardiac death among athletes as well.

A growing number of studies suggest that many COVID-19 survivors experience some type of heart damage, including arrhythmias, heart failure, and myocarditis, even if they were asymptomatic. Congenital structural diseases and myocarditis are a leading cause of sudden cardiac death in competitive athletes so they must be properly treated by their cardiologists before the return to sports.

Exponentially increasing number of scientific data on OHCA and IHCA are available to help with prevention as well. Future studies processing histopathological and toxicological data from autopsy may provide adequate medical data to develop a strategy to prevent cardiovascular death.

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