

Reducing sudden cardiac death risk in athletes through cardiovascular screening: Are we winners or losers?

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Abstract:

Objective: Sudden cardiac death during sports is unexpected death during exercise or shortly after physical exertion in athletes who otherwise appear perfectly healthy. The aim of this study was to highlight the importance of cardiovascular screening in athletes to reduce sudden cardiac death. **Design:** We realized a prospective cohort study. **Setting:** Participants were recruited and followed up between 2016 and 2021 for cardiovascular screening using electrocardiography with automated add-on module, spiroergometry, and echocardiography, in our cardiology outpatient clinic and sports center. **Participants:** The population comprised 280 athletes divided into 5 groups according to sports (ice hockey, basketball, football, running, and other). **Interventions:** Not applicable. **Main Outcome Measures:** We found significant pathology in 5.3% of study subjects (n = 15). **Results:** Arrhythmias were diagnosed in 13 athletes. Athletes with higher degrees of AV block (n = 1) and ventricular extrasystoles (n = 2) subsequently underwent interventional procedures. Only endurance athletes (n = 5) suffered from atrial fibrillation. Structural heart disease was confirmed in 3 subjects. No correlation could be confirmed between the thickness of the interventricular septum and a higher incidence of arrhythmias. Significant difference of interventricular septum width between the sports groups (p = 0.17) was also not confirmed. There was a significant correlation (p < 0.01) between the IVS and heart rate, PR interval, and VO2max. **Conclusions:** Most of the diseases associated with an increased risk of sudden cardiac death could be identified by changes in resting electrocardiogram. Sudden cardiac death among athletes can possibly be prevented by improving and expanding cardiovascular screening. To sum up, we believe we have saved at least 3 young lives thanks to adequate cardiovascular screening.

Keywords: Athlete's Heart, Cardiomyopathy, Arrhythmia, Electrocardiogram

Introduction

Sports-related sudden cardiac death (SCD) is defined as sudden and unexpected death occurring during, or shortly after, exercise (within 24 hours), if witnessed by a bystander and/or happening in an individual who was otherwise known to be healthy (Mont, 2017). Many of these deaths remain unexplained, however a substantial number of victims harbour an underlying and potentially detectable cardiovascular disease (Harmon, 2015). The SCD rate is estimated to range from 1.40 per 100 000 person-years [95% confidence interval (CI) 0.95, 1.98] in women to 6.68 per 100 000 person-years (95% CI 6.24, 7.14) in men (Eckart, 2011). The majority of SCD in young athletes (≤35 years old) are secondary to inherited cardiac diseases, while ischaemic heart disease predominates in older athletes.

Most cardiac pathologies in athletes are silent and SCD can often be the first and, unfortunately, last symptom (Mont, 2017). The vast majority of these events are due to malignant tachyarrhythmias, as ventricular fibrillation (VF) or ventricular tachycardia (VT) degenerating into ventricular fibrillation (VF). Those occur in individuals with arrhythmogenic disorders (e.g. arrhythmogenic cardiomyopathy, hypertrophic cardiomyopathy, etc). During intensive exercise training after-load and cardiac output increase, as well as electrolyte changes occur, which may trigger ventricular tachyarrhythmias in predisposed individuals (Priori, 2015).

Pelliccia et al. (2014) in a cohort of 2352 known to be healthy Italian Olympic athletes found 3.9% of CV abnormalities, of which (0.4%) included conditions at risk of SCD such as cardiomyopathies, coronary artery disease, perimyocarditis, long QT syndrome and ventricular pre-excitation. Consequently, there is a great interest in early identification of at-risk individuals for whom appropriate treatment, and / or physical activity adjustment, may be applied to minimize the risk of SCD. The role of athletes' screening examination lies also in distinguishing cardiovascular abnormalities from benign athletes heart remodelling (Pelliccia, 2019, Musat, 2009).

However, the role of CV screening in athletes as a feasible and efficient strategy to identify individuals at risk has remained controversial. Over the last decade, recommendations have not changed from the American Heart Association/American College of Cardiology (ACC) being anchored in a standard protocol of history and physical examination (Maron, 2014, Williams, 2019) while the European Society of Cardiology continues to support routine utilisation of ECG (Mont, 2017).

In our study we decided to realize thorough examination of athletes on the top of standard protocol and find out if an additional examination e.g. with echocardiography, could better identify unknown cardiovascular pathologies.

Methods

Study Population

We realized a prospective cohort monocentric study to screen athletes for unrecognized cardiovascular disease. Between 2016 and 2021 we screened 280 athletes (Figure 1) in the cardiology outpatient clinic and our sports centre. We divided the athletes into five groups according to the type of sports (Figure 2), all of which were dynamic. The only inclusion criterium was physical activity of at least 200 minutes per week to meet definition of athlete (Kiško, 2013). Examined athletes belonged to the category 1-3 according to McKay (McKay, 2022). Exclusion criteria were based on a history of pre-existing cardiovascular disease, medication use, doping, and/or a positive family history of SCD before the age of 50 years.

Written informed consent was obtained from all study participants prior to measurements and examinations. The study was approved by the local ethics committee prior to initiation, in accordance with the standards set by the Declaration of Helsinki.

Data Collection

Medical history was taken by anamnesis as well as by questionnaire method focused on risk factors and the presence of CVD symptoms. We initially did physical examination and resting 12-lead ECG with automated module (BTL CardioPoint®-SDS Module), resting blood pressure (BP), body-mass index (BMI). During echocardiography (ECHO) workup (Mylab class c, probe PA 240, Esaote) we monitored cardiac compartment dimensions including interventricular septal (IVS) thickness, valvular function, left ventricular (LV) systolic and diastolic function. If no contraindication was found we realized spiroergometric examination (CPET) (Cortex metalyzer 3B R3) on bicycle (Ergoline ergoselect 200), where we monitored respiratory gases, mainly maximal oxygen consumption (VO₂ max) as indicator of trainability (Dias, 2019), ECG and blood pressure (BP) during exercise. In all patients with IVS thickness >12mm 24- to 72-hour ECG Holter (BTL-08 Holter) was realized as well as genetic testing for Fabry disease. Cardiac magnetic resonance imaging (CMRI), electrophysiological study (EPS) and selective coronarography (SCG) were done if required by the differential diagnostic procedure.

Statistical Methods

Continuous variables are presented as sample means and standard deviations. Normality of data was tested using a Shapiro–Wilk test. Between group differences for categorical variables were estimated using the χ^2 test of independence. Student's *t*-test was used to analyze the between group differences based on equality of variances. When the variables had a markedly abnormal distribution of its nonparametric period the Mann Whitney U test was used. Anova Analysis was employed to verify the hypothesis of equality of several mean values, or its non-parametric equivalent Kruskal Wallis test was used. We studied the dependencies between the variables using regression and correlation analysis. P-values < 0.05 were considered statistically significant.

Results

A total of 280 athletes were enrolled, including 243 (86.78%) males and 37 (13.22%) females, aged between 18 and 62 years (29.4 ± 9.14). Runners represented the oldest athletes with an average age of 35.09 years and footballers formed the group of the youngest athletes with an average age of 24.11 years. We have found significant pathology in 5.3% of study subjects.

A thorough medical history by questionnaire revealed the presence of cardiac symptoms and/or a positive family history of cardiovascular disease in twenty athletes. Warning symptoms were presyncope (*n* = 7), palpitations (*n* = 7), dyspnoea (*n* = 2), tachycardia (*n* = 4).

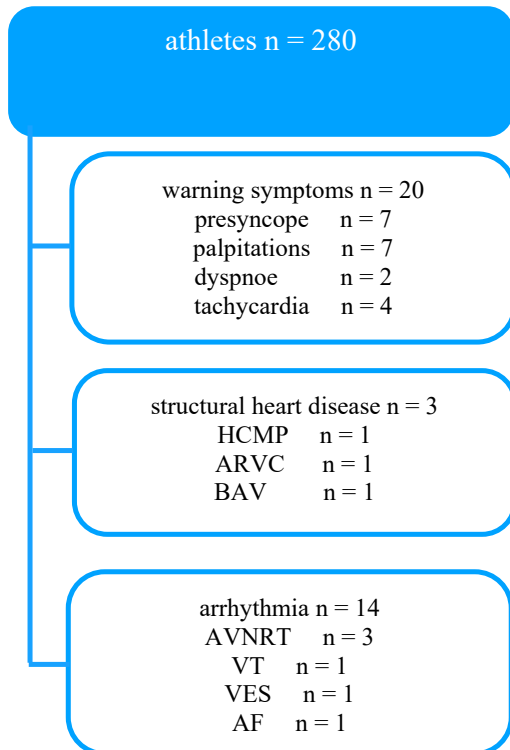


Figure 1. Study population

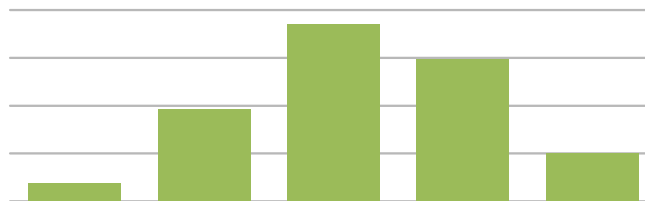
2. Distribution of athletes by the type of sport

On ECHO examination one third of athletes had IVS thickness 12-13 mm followed by group of IVS thickness 13-14 mm (Figure 3). We also performed genetic testing for Fabry disease in all of them with negative results. However, we could not demonstrate the presence of more frequent ectopic activity and complex arrhythmias in patients with IVS >12 mm. There was no statistically significant difference in the thickness of the IVS among the sports 151 monitored ($p = 0.17$).

Figure 3. IVS thickness

In three athletes we found severe heart disease in which IVS thickness averaged 14.5 mm. The disease prohibited them from returning to competitive sport. Other athletes with mild cardiovascular defects (mild aortic stenosis, mild tricuspid regurgitation) are in our dispensary with more frequent check-ups while their sporting

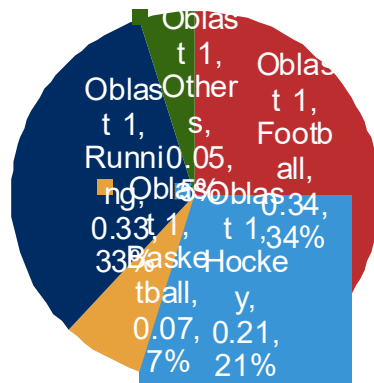
■ < 11mm ■ 11 - 12mm ■ 12 - 13mm ■ 13 - 14mm ■ >14 mm



career is not interrupted. Hypertrophic cardiomyopathy (HCM) was detected in one athlete (Figure 4), a handball player (Vaňová, 2019). The ECHO examination and CMRI showed eccentric thickening of the LV myocardium with no signs of fibrosis. The athlete had been asymptomatic. Genetic testing was realized showing that the patient was heterozygous for the genes for hypertrophic cardiomyopathy - variants c4472C:G in the MYH7 gene and c12-50 12-47dupACAG in the TNNI3 gene. Latter were detected in the heterozygous state inherited from the mother, variant c975C:T in the MYH7 gene and variant c649A:G in the MYBPC3 gene inherited from the father. On the basis of these findings, we ordered the termination of his career as a professional athlete. In the repeated 24- hour ECG HM only sporadic supraventricular and VES were detected, less than 1% of the monitored time, without the presence of complex arrhythmias. The patient is currently not

receiving any pharmacotherapy and remains in follow-up. According to current recommendations implantable cardioverter-defibrillator (ICD) implantation has not yet been indicated (Maron, 2015).

■ Football ■ Hockey ■ Basketball
■ Running ■ Others



One patient was diagnosed with bicuspid aortic valve with severe aortic regurgitation. He underwent aortic valve replacement surgery and cannot continue in competitive sport.

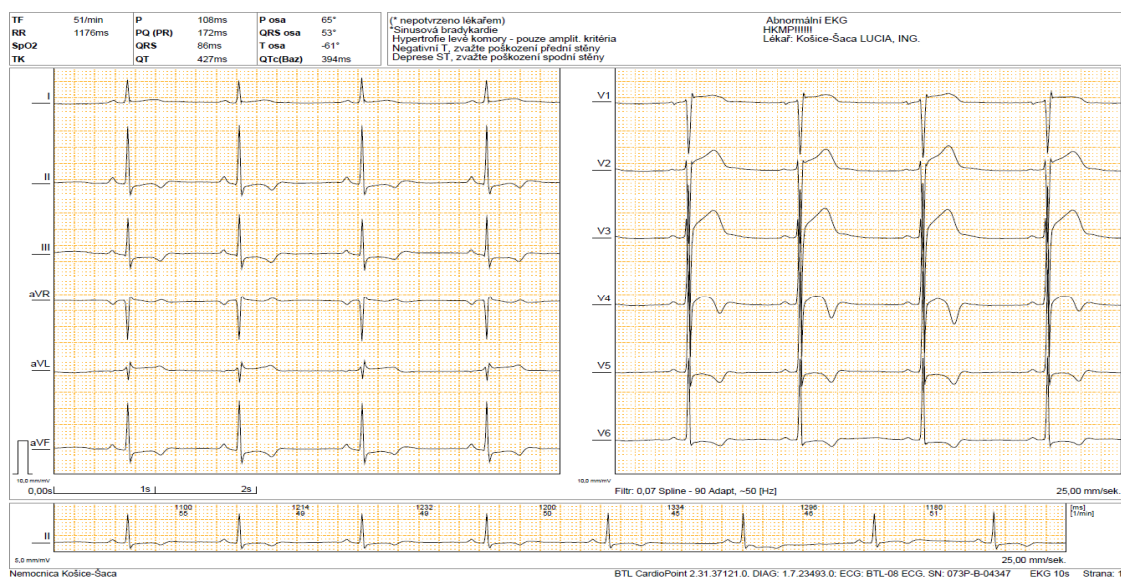


Figure 4. ECG of an athlete with HCMP

During the follow-up period, we detected significant arrhythmias in 14 athletes. Atrioventricular nodal reentry tachycardia (AVNRT) was diagnosed in three athletes, namely one professional cyclist and two professional ice hockey players. All of them underwent successful radiofrequency catheter ablation (RFCA) of the arrhythmogenic pathway at local cardiovascular disease institute. Three months after the procedure 72-hour ECG HM and CPET examination were done with negative results, thus athletes returned to active sporting.

Ventricular tachycardia (VT) was diagnosed in two cases including a triathlete and a professional basketball player. The first case was a continuous, spontaneously terminating VT (Figure 5), with arrhythmogenic right ventricular cardiomyopathy (ARVC) revealed by CMRI and confirmed by endomyocardial biopsy. The athlete had to cease active sport according to current international recommendations (Maron, 2005). He was subsequently implanted with an ICD due to recurrent VT in the secondary prevention of SCD. The second case was a VT finding during a 72-hour ECG HM in a basketball player who experienced repeated palpitations and had pre- collapse. The athlete underwent successful catheter ablation and returned to an active career.



Figure 5. ECG stress test in an athlete with ARVC and VES, ns VT

Two athletes (one basketball and one hockey player) had ventricular extrasystoles (VES) with structurally normal hearts verified by CMRI. They did not require further intervention or therapy for the time being.

We observed a 2nd grade AV block Mobitz type II in two athletes (ice hockey). In both of them the arrhythmias occurred during ECG HM monitoring in sleep and were asymptomatic. ECHO and CPET were without pathological findings. These athletes remain being followed up, as for now without need for interruption or modification in their sports activities. Atrial fibrillation (AF) occurred in five athletes (runners) with a history of palpitations, an average age was 52 years. In all cases it was paroxysmal type, self-terminating, detected during 72-hour ECG HM. According to the low thromboembolic risk - CHA2DSVASc 2 score was 0 in all cases, long-term anticoagulation therapy was not indicated.

Twelve out of fifteen athletes with significant pathological findings returned to the original sport within 3 months after medical intervention.

Basketball players subgroup analysis

In the group of basketball players we confirmed an indirect linear relationship between heart rate (HR) and IVS ($p < 0.01$, $R = -0.61$), HR and VO₂ max ($p < 0.01$, $R = -0.58$), BMI and VO₂ max ($p = 0.02$, $R = -0.503$). With increasing IVS width, being the marker of increased training, the heart rate decreases, because of the activity of the parasympathetic vagus nerve. With increasing PR interval the heart rate decreases as well ($p = 0.015$, $R = -0.54$). The sportier athlete gets, the lower his heart rate is and the longer the PR interval becomes. A direct linear relationship was confirmed between the QT interval and VO₂ max ($p < 0.01$, $R = 0.64$).

Discussion

This study highlights the importance of cardiovascular screening in athletes as part of their regular medical check-ups with the emphasis on reducing SCD in sport. SCD in athletes is typically the result of structural and arrhythmogenic heart disease, which is underdiagnosed because athletes are often asymptomatic. Therefore, the European Society of Cardiology (ESC) has issued recommendations for the inclusion of ECG in preventive sports examinations for all athletes (Mont, 2017). However, there is a steady increase in the number of recreational athletes and practitioners do not have much experience in assessing 12-lead ECG in athletes, consequently they often use the same criteria for its evaluation as in the general population of non-sporting people (Sharma, 2018). That's why automated ECG modules may be preferred to distinguish warning signs of cardiac disease.

In endurance sports, as long distance running, it is typical that an increased cardiac output is maintained for a long time with reduced peripheral vascular resistance, resulting in a continuous volume overload for all cardiac compartments. The morphological reflection of these changes is the echocardiographic image of eccentric hypertrophy, which is characterized by increased wall thickness and LV dilatation. In contrast, high-impact and strength sports, as weightlifting, are characterized by normal or only slightly increased cardiac output and increased peripheral vascular resistance, resulting in increased blood pressure and increased LV afterload. The typical morphological consequence in this type of strain is a thickening of the LV walls with its normal size or only mild dilatation. Then there are sports where these characteristics overlap (football, basketball, hockey, etc.), and the echocardiographic image is also not so clearly defined.

HCMP is anatomically characterized by generalized or localized LV hypertrophy. Available literature data suggest that the risk of SCD in genetically determined hypertrophic cardiomyopathy is 0.6% and the overall

mortality rate is 1.3%. Increased LV wall thickness in athletes results from increased ventricular pressure, volume overload, or both mechanisms (Bent, 2015). A 2-5 mm decrease in LV wall thickness after a deconditioning phase lasting 3-6 weeks almost certainly rules out HCM and supports the diagnosis of athlete's heart (Sarto, 2004). The extreme LV remodelling that occurs in ultra-elite athletes raises concern of potentially adverse clinical consequences, as 10% – 40% of such athletes have a LV end-diastolic cavity diameter above 60 mm. Such enlargement is actually observed in patients with dilated cardiomyopathy. In fact, severe remodelling in a certain percentage of athletes may not recede after the cessation of active activity. LV enlargement persists in about 20% of elite athletes even 5 years after the end of sporting activity (Pelliccia, 2002).

ARVC is characterized by enlargement, dysfunction and fibro-fatty infiltration of the right ventricle. Ventricular tachycardia morphology of LBBB is often triggered by physical activity, as it was in the case of our athlete. Typical symptoms in these patients are pre-syncope, syncope or SCD during sports. T-wave inversion in leads V1-V3 is observed in more than 85% of patients. Epsilon waves as a distinct abnormality on ECG after QRS complex, is observed in less than 1/3 of patients with ARVC. In the athlete's heart, we can also observe inversion of T waves in leads V1-V3, but these normalize during stress testing, whereas in ARVC patients they typically remain inverted. Right ventricular dilatation has also been described in the athlete's heart, but in most cases its function is normal. MRI of the heart and endomyocardial biopsy contributes to the diagnosis of ARVC with a very high sensitivity and specificity (Walker, 2010).

Endurance athletes, both professional and recreational, are more likely to have chronic myocardial remodelling and complex arrhythmias with higher risk of developing AF (Walker, 2010). Supraventricular arrhythmias in athletes are most often asymptomatic and benign. Yet, occasionally, they can be symptomatic and do require therapy, such as RFCA. Our findings confirmed studies, which have shown a three times higher prevalence of paroxysmal AF in highly trained endurance athletes (Pelliccia 2005). It is not uncommon for endurance athletes to exceed physical activity by more than ten times the recommendations for cardiovascular protection. There is a direct correlation between the intensity of endurance exercise and left atrial dilation, which is related to the rise in venous pressure and venous return. Prolonged intense endurance sport leads to mild dilation of both atria and right ventricle and can disrupt the architecture, leading to micro-damage, sinoatrial node changes occur. The most common trigger for paroxysmal AF is ectopic deposits around the pulmonary veins in the left atrium. Left atrial size above 40 mm is relatively common in athletes (20%) with an upper limit of up to 45 mm in women and 50 mm in men (Abdulla, 2009). ECHO may reveal structural heart disease, and anticoagulation therapy for AF is indicated according to current international recommendations, but potential haemorrhagic complications in contact sports remain a concern (Estes, 2017). Asymptomatic paroxysms of AF, which are common not only in athletes but also in the general population and account for approximately 1/3 of AF cases, remain an issue as well. Therefore, longer-term (most often 7-day) ECG HM is required for their diagnosis. Hence, the detection of AF in athletes with a short 12-lead ECG is very low with a high percentage of falsely negative results (Wernhart, 2015).

We cannot forget to point out importance of legislation to secure regular medical check-ups for competitive athletes. Endurance athletes have increased parasympathetic tone with prolongation of the PR interval and a decrease in resting heart rate as part of the heart's adaptation to regular exercise. Isolated voltage criteria for LV hypertrophy are often present, which we have confirmed in our cohort of runners (Bent, 2015). Automated ECG module has high sensitivity and specificity to detect increased risk of SCD and aids in the correct interpretation of ECG changes in athletes (Bent, 2015, Kenttä, 2016).

Pelliccia et al. (2006) evaluated 4450 Italian athletes, and concluded that ECG is an efficient weapon in the identification of HCM. When it comes to the ECG changes, the question is whether we are dealing with athlete's heart or some clinically significant finding. According to ESC recommendations, in cases of ECG abnormalities with high voltages, T-wave inversion or deep precordial Q wave, a more exhaustive examination should be conducted including a family study, ECHO, as well as 24 hour Holter (Sharma, 2018, Corrado, 2005). If doubt persists, cardiovascular resonance imaging should be conducted and athletes should be recommended to temporarily stop participating in competitive sports in order to determine whether findings are reversible or not (La Gerche, 2012). In our cohort we detected arrhythmias in 14 athletes, 5 of them subsequently required interventional procedures and 4 of them returned to active sport. We continue to monitor 6 other athletes for higher degrees of AV block, ventricular extrasystoles and mild valvular heart disease.

More than 2/3 of subjects had IVS >12mm. All of them underwent ECG HM. Genetic testing for Fabry disease among them was negative. As in previously published studies, we haven't found correlation between the IVS thickness and a higher incidence of ectopic activity or complex arrhythmias (Walker, 2010). On the other hand, an IVS thickness of more than 13.0 mm in athletes always raises suspicion of cardiomyopathy and ought to be extensively examined.

Study Limitations

Our study has several limitations. First, the geographical area covered eastern region of country with only caucasian population, thus prevalence of findings may be different in other regions or race. Even though there was relative high percentage of cardiac pathologies in our cohort, there was high heterogeneity of sports activity to identify significant relationships within data set.

Conclusion

The primary scope of the CV screening of athletes' is to identify cardiac diseases clinically silent that may be associated with cardiac arrest/ sudden cardiac death in relation with exercise training and competitive sport participation. Another goal is identification and appropriate management of CV conditions that may worsen because of intensive athletic training.

Based on the Italian experience CV screening is potentially lifesaving (Corrado, 2006). Mortality data derived from a time-trend analysis over a 26 year period demonstrated that the incidence of SCDs in athletes decreased by 89%. Death due to cardiomyopathies were greatly reduced.

In conclusion, this study shows an unexpected prevalence of CV abnormalities in elite athletes, including a small, but important proportion of conditions at risk of SCD. During the follow-up period we detected 15 cardiac pathologies, 3 of them resulting in professional sports career cessation. It is likely that including echocardiography in our protocol amplified the proportion of CVD identified. We confirmed an indirect linear relationship between heart rate and IVS width, being the marker of increased training. No correlation could be confirmed between the thickness of the interventricular septum and a higher incidence of arrhythmias.

As a matter of fact no medical evaluation was universally required for athletes, prior to their participation in the Rio de Janeiro 2016 Summer Olympic Games. We trust that our observations support the concept that medical evaluation of elite athletes is appropriate and necessary. Our observation suggests that athletes are not immune from serious CV disorders and legitimates the concept of a tailored screening programme. Even the athletes with cardiomyopathies denied symptoms or impairment in physical performance, and identification of their disease was made possible only by screening. To sum up, we believe we have saved at least 3 young lives thanks to adequate cardiovascular screening.

Informed Consent Statement: Informed consent was obtained from all subject involved in the study.

Conflicts of Interest: The authors declare no conflict of interest.

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