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LOWN-GANONG-LEVINE SYNDROME IN PROFESSIONAL ATHLETES - IS IT DANGEROUS?

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(Original scientific paper)

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Abstract

Background: Lown-Ganong-Levine (LGL) syndrome is characterized by a short PR interval and normal QRS duration. LGL syndrome typically presents with paroxysms of tachycardia and is generally associated with a favorable prognosis.. Aim: Risk stratification in professional athletes with LGL syndrome. Case Report: We present a case of a 17-year-old professional footballer who experienced intermittent symptoms of tachycardia, fatigue, and chest pain. Over the past 10 days, during training sessions, he noticed a faster heart rate than usual and a sudden onset of pronounced fatigue, leading to an inability to complete his training as he normally would. An electrocardiogram (ECG) revealed a short PR interval, while an echocardiography showed no abnormalities. Additionally, an exercise stress test yielded normal results. Finally, a Holter rhythm recording captured episodes of sinus tachycardia reaching up to 155 beats per minute. Based on these findings, a diagnosis of Lown-Ganong-Levine (LGL) syndrome was made. Since no additional risk factors were identified, the patient was cleared to continue with professional sports. Conclusion: Due to the tendency for tachycardia in individuals with Lown-Ganong-Levine (LGL) syndrome, it is advisable to evaluate the condition to ensure that the athlete can safely continue with sports activities. Fortunately, athletes diagnosed with LGL syndrome generally have a positive prognosis and can typically continue participating in professional sports without significant risks.

Keywords: *LGL, short PR interval, professional sports*

Introduction

Even though athletes are generally fitter, when it comes to sudden death, hey have a higher risk compared to individuals who don't participate in sports.. That shows us that there are many underlying cardiac conditions, which can be identified by pre-participation screening involving physical examination and an electrocardiogram (ECG) [1].

One of those conditions is Lown-Ganong-Levine (LGL) syndrome, which was first described in 1938 and later defined by Lown, Ganong, and Levine in 1952. The syndrome involves an accessory pathway that connects the atria with either the atrioventricular node or the His bundle. As a result, a short PR interval is observed, the QRS complex remains normal, and episodes of paroxysmal tachycardia may occur. [2].

PR interval shows atrial depolarisation and its conduction through the AV node, being measured from the start of the P wave to the QRS complex beginning. Its normal length is considered 0.12 to 0.20 seconds. When it`s shorter, an accelerated atrioventricular conduction happens [3].

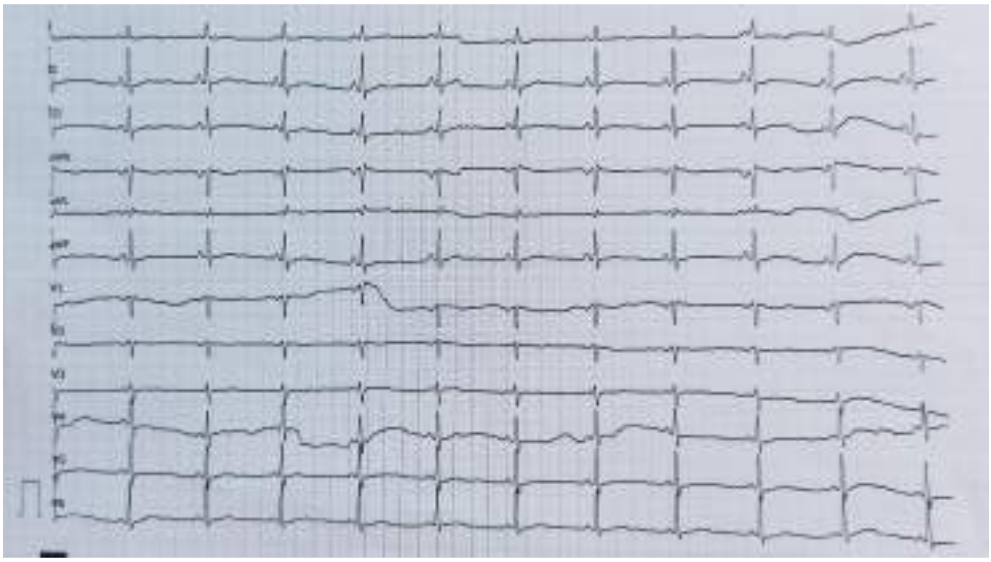
LGL syndrome is usually suspected using an electrocardiogram (ECG), which shows a short PR interval and normal QRS complex, but to confirm this diagnosis usually electrophysiologic evaluation and a holter ECG monitoring or loop recorder is needed. Sometimes routine laboratory tests with hormone levels and electrolytes are evaluated, but their correction hasn`t shown a correlation with the LGL pattern [4].

It is estimated that less than 1% of the population has this condition, and not everyone experiences symptoms. Prognosis seems good, they are typically asymptomatic, but because of their susceptibility to developing atrial fibrillation, atrial flutter, or another tachyarrhythmia, sometimes they report palpitations or other correlated symptoms. In those with pathological arrhythmias, treatment is currently based on antiarrhythmic drugs. The condition itself is rarely associated with hemodynamic compromise, so the treatment is focused on preventing the tachyarrhythmias and that includes digitalis, beta-blockers, calcium

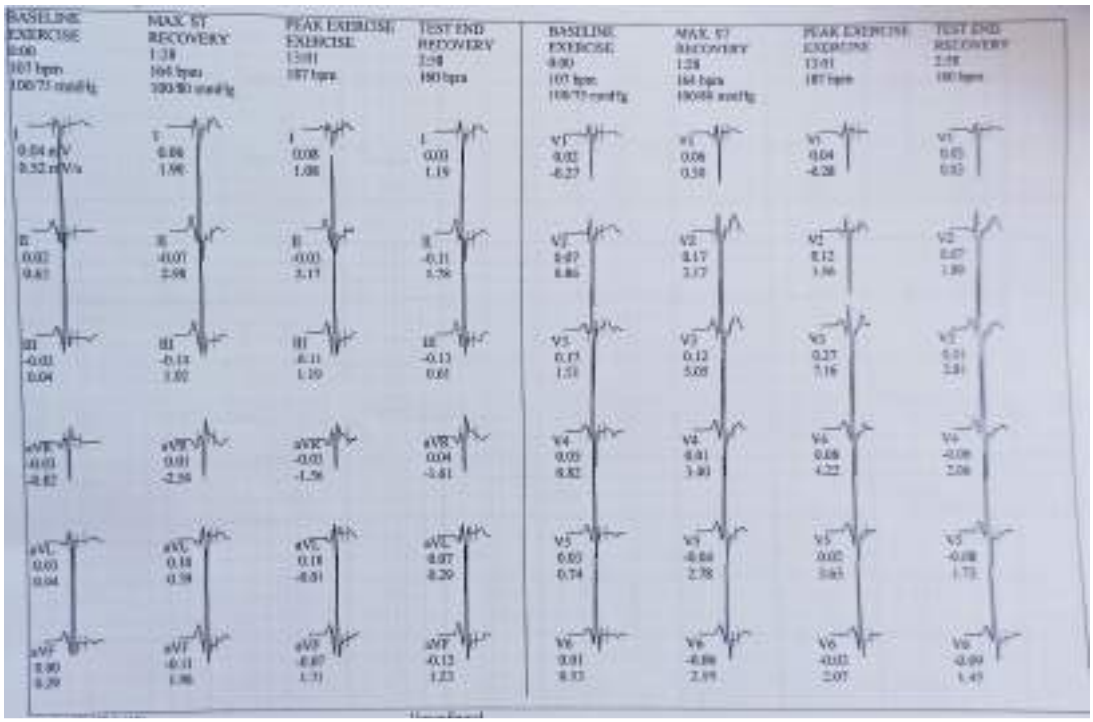
channel blockers, and class I or III antiarrhythmic drugs. When medical therapy doesn't work out, radiofrequency catheter ablation is usually performed [4].

Case report

We are presenting the case of a 17-year-old footballer with a history of occasional symptoms including tachycardia, fatigue, and chest pain. As a professional footballer, he has been experiencing a faster heart rate than usual and a sudden onset of pronounced fatigue during training over the past 10 days. Consequently, he has been unable to complete his training as usual. He mentioned that this is the first time he has experienced these symptoms..



Picture 1. ECG on admission-sinus rhythm with a heart rate of 70 per min., flat T waves in precordial leads, PR interval of 0.08 seconds



Picture 2. QRS and ST segment during the exercise stress test

On admission, an electrocardiogram (ECG) was performed, revealing sinus rhythm and a short PR interval of 0.08 seconds. Heart sounds were found to be rhythmic and clear upon auscultation. Transthoracic

echocardiography was conducted, showing normal dimensions of the left ventricle, normal volume, and normal systolic function (ejection fraction of 64%). Myocardial systolic deformation was normal. Diastolic function was super-normal, with normal pressure of left ventricle filling. DTI E` 0,17m/s, E`L 0,18m/s, E/E`Avg 3,75, GLS- 18%. Right ventricle with slightly manifested middle dimensions and normal global systolic function, FAC 34%. Left and right atriums with normal dimensions. The interatrial septum is aneurismally thinned with systolic excursion, without the presence of a significant shunt. There was insignificant mitral regurgitation. In other words normal echocardiographic findings

The patient runs an exercise stress test according to BRUCE for 13:01 minutes, achieving a maximal work level. His resting heart rate of 126 bpm rose to a maximal heart rate of 190 bpm. This value represents 93% of the maximal, age-predicted heart rate. The resting blood pressure of 100/75 mmHg rose to a maximal pressure of 120/80 mmHg. The cardiac stress test was marked as negative by maximal intensity, the blood pressure was in range, the heart rate was normal, rhythmic action, and ST changes were not registered. The test finished with mild chest pain.

A 24-hour Holter rhythm was recorded which shows sinus rhythm throughout the entire monitoring with an average frequency of 77/min, minimum 50/min, and maximum 155/min, without significant pauses and changes in the QRS and T wave.

After a month, the 48-hour rhythm Holter was repeated, which was identical to the previous one. As the investigations did not show any sign of risk, the patient was allowed to continue with professional sports.

Discussion

A short PR interval in EKG is defined as <120ms. Even though it is recommended that asymptomatic athletes with short PR intervals do not need further investigation, often they go for further evaluation. In the research by G Parry-Williams and the authors, a study of 15,572 athletes aged 14-35 went through cardiac screening using ECG. A short PR was present in 765 (4,9%) athletes, being more common in females vs. males (6.2% vs. 4,2%). Its prevalence was less common in elders, being present in 9% of 14-year-olds, but only 3.2% of 17-35 year-olds. So this shows us a preference for younger and female athletes, which can be explained by having anatomically smaller hearts, higher sympathetic tone, or amplified resting AV node conduction. A higher prevalence of short PR in younger athletes implies that without having symptoms or accessory pathways they don`t need to go for further investigation. Still, this must be confirmed by long-term studies [5].

The study of Bernard Lown et al. picked 200 people with short PR (184 of them with normal QRS complex) and 200 control people with normal PR intervals. The material was obtained from 13,500 successive electrocardiograms taken at the Peter Bent Brigham Hospital from 1947 to 1950. People with conditions that can affect PR interval or provoke rapid heart action were excluded. They investigated the correlation between paroxysmal rapid heart action in people with short and normal PR intervals. Among the first group, there were 23 patients with proven paroxysmal tachycardia, out of which 19 were with short PR intervals and normal QRS complex, as it is in our case. In the group of 184, there were also 15 patients suggestive of rapid heart action. On the other hand in the control group, there was only one case of rapid heart action.

Consequently, 15 patientswere chosen with paroxysmal supraventricular tachycardia from the Peter Bent Brigham Hospital records based on the criteria mentioned previously and were monitored together with the previous 19. The average age when tachycardia first manifested was 33.5 years, but in most cases, it was later in life, that is 14 out of 34 people first experienced tachycardia after the age of 40. Palpitation was the main complaint in 20 of the patients. After its first appearance, tachycardia continued to appear intermittently, with strikes that vary from a few hours to many years, the longest one being 60 years between attacks noted in two patients. The most common arrhythmia was paroxysmal auricular tachycardia. Most of the patients tolerated tachycardia well. Presumably, that was due to not having underlying heart disease. However, two patients died suddenly, both had episodes of auricular fibrillation [6].

According to a study by Mark Abela et al. approved by the University of Malta Research Ethics Committee, students across all schools in Malta in Gozo (average age 15 years) were invited to enter a cardiac screening program for adolescents. Individuals who regularly took part in sports events or had ≥ 4 hours of physical activity were defined as athletes. They were investigated by 12 lead ECGs. There were 2658 students (15±0.33), 39% of which were athletes, 61% being male athletes. Athletes had lower heart rates (77±15 vs 84 ±16), longer PR intervals (140±20 ms vs 137±19 ms), and QRS complex duration was increased (94±30 ms vs 91 ±13 ms) compared to non-athletes. Isolated short PR was more frequent in

females and non-athletes, which can be possibly explained by having anatomically smaller hearts in females and a higher vagal tone in athletes. In the athletic category, short PR interval was the most common finding in power athletes, and by having higher sympathetic tone they are more susceptible to developing tachycardia episodes during exercise [7].

Ventricular preexcitation happens as a result of an accessory pathway from the atria to the ventricles. It is believed that atrial fibrillation can lead to rapid ventricular response and even cause sudden death. The University of Padua assessed the incidence and clinicopathological features of ventricular preexcitation in Northeastern Italy by investigating 273 sudden deaths in children and young adults (≤35 years). Ten patients had ventricular preexcitation, 2 of which were diagnosed by EKG as LGL syndrome having PR interval <120ms and normal QRS complex. The pathological data about the conduction system in LGL syndrome says that the structure of the sinus node was normal in both cases. In One patient there was congenitally AV nodal hypoplasia and because of that, there was less mass specialized tissue to hold up the transmission impulse from the atria to the ventricles. On the second one, there was an atrio-hisian bundle that bypasses the AV node and transmits the signal directly into the His bundle. In both cases, the onset of atrial fibrillation can lead to ventricular fibrillation [8].

Conclusion

LGL syndrome is a condition with a higher prevalence in females and younger athletes. In most cases, it is asymptomatic, while in symptomatic patients it is mostly well tolerated. However, because of their susceptibility to tachycardia, it`s recommended that physicians assess their condition to clarify that the athlete can continue with sports activities and there is no potential danger. Usually, Athletes with LGL syndrome have a good prognosis and can continue to play professional sports without any risk.

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