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Case Report

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Extensive anterior acute myocardial infarction in young high-performance athlete without coronary risk factors

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ARTICLE INFORMATION

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Abbreviations
STEMI: ST-segment elevation myocardial infarction

ABSTRACT

Physical exercise avoids cardiovascular diseases but when it is intense, it must have correct planning and dosage and, if possible, a previous ergometric test and a guidance specialist could be very useful; because it can also cause cardiovascular events in high performance athletes, among other things, for not complying with a well-defined schedule with a scientific base. The most feared complication is sudden cardiac death, usually due to malignant arrhythmias and ischemic heart disease. Here is presented the case of a 34-year-old high-performance athlete, in the detraining phase and without cardiovascular risk factors or other history of interest, who suffered an extensive acute myocardial infarction, with post-infarction angina, due to a stricture suboclusive of the proximal left anterior descending artery. A rescue angioplasty was performed, with implantation of a drug-eluting stent and the patient evolved favorably.

Keywords: Myocardial infarction, Risk factors, High-performance athlete, Percutaneous transluminal coronary angioplasty

Infarto agudo de miocardio con elevación del segmento ST anterior extenso en joven deportista de alto rendimiento sin factores de riesgo coronario

RESUMEN

El ejercicio físico evita las enfermedades cardiovasculares pero cuando es intenso debe tener planificación y dosificación correctas y, de ser posible, es muy útil contar con una prueba ergométrica previa y un especialista orientador; porque también puede producir accidentes cardiovasculares en deportistas de alto rendimiento, entre otras cosas, por no cumplir con una programación bien definida con un basamento científico. La complicación más temida es la muerte súbita cardíaca, habitualmente por arritmias malignas y enfermedad isquémica. Se presenta el caso de un deportista de alto rendimiento, de 34 años de edad, en fase desentrenamiento y sin factores de riesgo cardiovascular u otros antecedentes de interés, que sufrió un infarto agudo de miocardio anterior extenso, con angina postinfarto, debido a una estenosis suboclusiva de la descendente anterior proximal. Se realizó angioplastia de rescate, con implantación de un stent farmacoactivo y el paciente evolucionó favorablemente.

Palabras clave: Infarto de miocardio, Factores de riesgo, Deportista de alto rendimiento, angioplastia coronaria transluminal percutánea

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INTRODUCTION

Physical activity and sport help reduce overall and cardiovascular mortality[1]. From ancient Greece to the present day, its regular, systematic practice is strongly recommended by different medical organizations, since there is a good deal of scientific evidence associating the practice of physical activity and sports with the aforementioned reduction in mortality and improvement in quality of life[2-6]; However, it is also known that physical exercise, especially if intense, temporarily increases the risk of cardiovascular events and, in particular sudden cardiac death, which may be the first manifestation of undiagnosed cardiovascular disease in previously asymptomatic athletes[7]. Its causes vary according to age. In patients under 35 it may occur mainly due to congenital or hereditary diseases, and in patients over this age, it occurs essentially due to coronary disease[1,2]. In general, in addition to atherosclerotic ischemic heart disease, hypertrophic and dilated cardiomyopathies, coronary congenital malformations, myocardial bridges, myocarditis, Marfan syndrome and, especially, cardiac arrhythmias (arrhythmogenic right ventricular dysplasia and channelopathies) are important causes of sudden death[1,6-8].

Cardiovascular risk factors in athletes are not very different from those in the general population, and therefore −in addition to non-modifiable risks− toxic habits, obesity, sedentary lifestyle, diabetes mellitus, high blood pressure and dyslipidemia are the main factors contributing to atherogenesis[7-8].

CASE REPORT

We present the case of a 34-year-old white man with no family or personal pathological history of heart disease in any of its manifestations, a high-performance athlete (Greco-Roman wrestling) in the process of detraining, who regularly and intentionally practiced physical exercise, without apparent stress, phlegmatic, neither a smoker nor a drinker, with a body mass index of 24 kg/m^2), and without known risk factors, who suddenly presents with ST-segment elevation acute myocardial infarction (STEMI).

During the early morning of a typical day he was suddenly awakened by a strong precordial, retrosternal, oppressive pain radiating to the neck and upper left limb, which progressively increased in intensity and was accompanied by sweating, coldness, anxiety and a feeling of imminent death, so he presented to a nearby polyclinic, where the symptoms were interpreted as extracardiac pain, given the previous knowledge that the patient was an athlete. Intramuscular analgesics were administered, but since the pain persisted, an electrocardiogram (ECG) was performed which showed significant ST-segment elevation in V_2-V_5 (**Figure 1A**). Now completely certain of the diagnosis of STEMI, he was urgently transferred to the referral hospital; but no thrombolytic treatment was administered since more than 6 hours had elapsed since the onset of symptoms. All other routine therapeutic measures were applied, including nitroglycerin infusion which was increased to a dose of 0.3 mcg/kg/min given the persistence of pain.

Some results of his hemochemical profile are shown in the **table**, including CPK-MB which amounted to 654 IU/L. The transthoracic (bedside) echocardiogram showed marked septoapical and lateral hypokinesia, with mild-moderate left ventricular dysfunction (ejection fraction 42%), normal wall and valve thickness, and absence of intracavitary thrombi.

Unfortunately, we do not yet have an Interventional Cardiology Service in our province. Therefore; due to post-infarction angina, in the context of a STEMI, without fibrinolytic treatment in a high performance athlete undergoing detraining and facing

Table. Main results of the patient's hemochemical tests.

Paremeter	Value
HDLc	1.9 mmol/l
LDLc	0.3 mmol/l
Total cholesterol	2.2 mmol/l
Triglycerides	1.4 mmol/l
Blood glucose	5.2 mmol/l
Hematocrit	0.50 Vol. %
Urea	4.1 mmol/l
Creatinine	124 µmol/l
Uric acid	175 mmol/l
Full coagulation test	Normal
GGT (Gamma-Glutatile-Transferase)	36 UI/L
LDH (lactic acid dehydrogenase)	64 UI/L
CPK-MB	654 UI/L

CPK-MB, creatine phosphokinase isoenzyme MB; HDLc, high density lipoproteins; LDLc: low density lipoproteins.

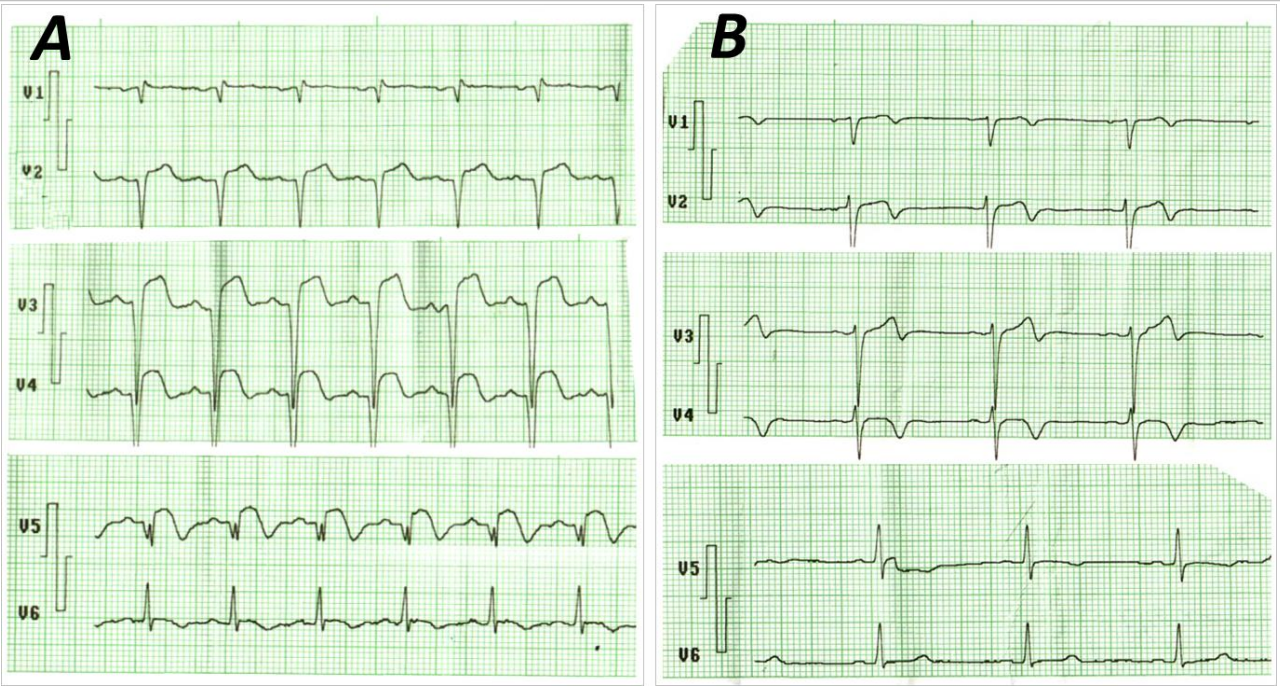

Figure 1. Precordial electrocardiographic leads, (A) before and (B) after angioplasty, clearly showing ST-segment elevation regression owing to extensive anterior myocardial infarction.

the evident risk of death, his transfer to the *Instituto de Cardiología y Cirugía Cardiovascular* was scheduled. Coronary angiography showed critical stenosis (98%) of the proximal segment of the anterior descending artery (**Figure 2**). A 3.5 × 14 mm drug-eluting stent was implanted, and good angiographic results were obtained.

The patient had a favorable outcome; his pain was completely relieved and normal ST- segment was eventually restored (**Figure 1B**). Despite the time elapsed, the echocardiogram on discharge (48 hours after the surgical procedure) showed left ventricular ejection fraction of 49%, which reached 57% at the 30-day follow-up appointment.

In the second month he underwent an assessment stress test which yielded excellent results with no residual ischemia; hence, he went on with his cardiac rehabilitation program and his detraining process under strict monitoring by the Sports Medicine and Cardiology Departments. While this case is presented, he was only receiving dual antiplatelet therapy (aspirin and clopidogrel) and statins.

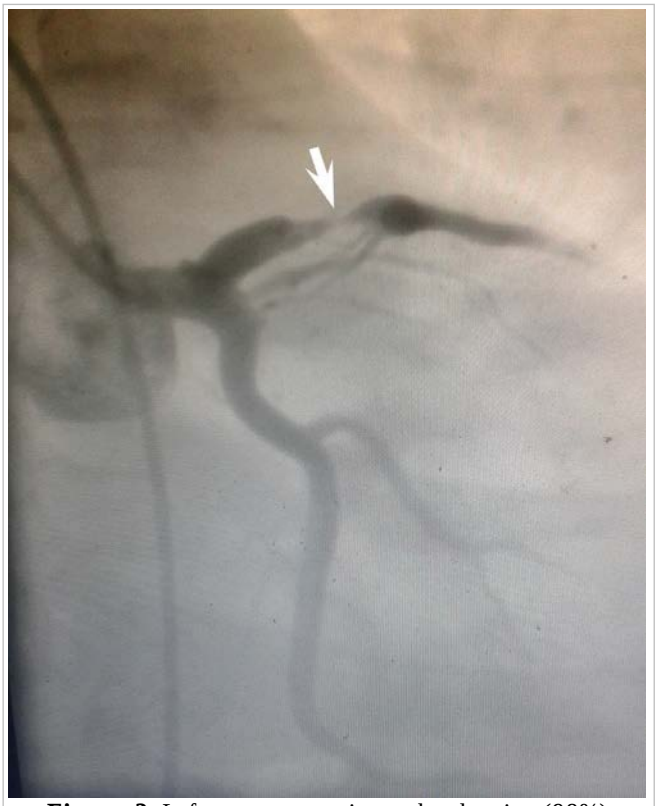

Figure 2. Left coronary angiography showing (98%) proximal left anterior descending subocclusive stenosis (arrow).

COMMENTS

Since the 19th century it has been suggested that

both long-term, intense physical exercise and systematic sports training can produce dramatic changes or chronic cardiovascular adaptations. Therefore, trainers, physiologists and practitioners have shown a special interest in this particular issue, either seeking to understand its influence on health or on the performance of athletes[7-9]. Yañez[9], William and Arnols studied some participants in a marathon race in 1899 and reported that they were suffering from signs of heart fatigue as a result of the test, confirmed by an acute increase in heart size at chest percussion and the presence of mitral insufficiency on auscultation in 84% of the runners. On the other hand, the author himself[9] points out that, previously, in 1898, Henschen described, by means of chest percussion, the growth of the heart in cross-country skiers, which he considered to be a physiological trait due to athletic training, which was favorable for obtaining good sports results. This can be considered as the first description in the literature of what is nowadays defined as "athlete's heart".

The patient in our report did not have a STEMI during physical activity but at rest. However, it is important to note that in the detraining phase every physician should review the anatomical and functional impact of intense and systematic physical exercise on the circulatory system, possible cardiovascular responses and adaptations produced by high-intensity exercise, training the duration and criteria and methods that allow differentiation between phenomena considered physiological and typical of sports practice and pathological processes; a matter which, in some scenarios, could be the cause of sudden death[7-11]. The physician must also know and apply the standard assessments currently recommended by medical and sports organizations to try to avoid such a cardiac event, especially the value of the electrocardiogram in athletic patients[4-13].

However, some cases like ours are exceptional. The patient had complied with all medical assessment protocols prior to sports practice, and was already in the detraining phase; yet he suffered a STEMI due to proximal left anterior descending subocclusive stenosis, which –on the one hand– differs from what has been published in the medical literature on the subject[1-3], in addition to the absence of risk factors[14-16]; but, on the other hand, is consistent with all research identifying ischemic heart disease as a major cause of cardiovascular events and sudden death in high performance athletes[1,6-8,10-12].

Several investigations[8,10,11,14,17] have highlighted that some factors causing or contributing to cardiovascular events in athletes may be: increased homocysteine, increased free radicals, change in the metabolism of arachidonic acid, prostacyclines and thromboxane A_2, in addition to endothelial dysfunction, inflammation and sudden electrolyte changes.

Specific diagnostic tests such as CT scans, MRIs, radioisotope studies and genetic tests have specific indications and should be individualized. However, the electrocardiogram, stress test and occasionally Holter monitoring are essential for all high performance athletes[1,4-7,13], because of their ability to predict cardiovascular disease and potential complications.

A STEMI is a medical emergency and should be treated as such[17], which is why the patient should go to hospital immediately after the onset of symptoms[18] and a high degree of suspicion should be maintained during the first medical apointment[19], which did not occur in this case, resulting in the non-application of fibrinolytic treatment. As rightly stated by Reimer and Jennings[20] more than 40 years ago, "time is muscle" and delays in this type of patient claim thousands of lives each year[17,19].

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