Introduction

Chest pain is one of the most common complaints in emergency departments, and internal medicine and cardiology clinics. Patients’ comorbidities and the clinical characterization of chest pain are cornerstones to guide symptom differentiation. Information about pain intensity, duration, localization, changes with posture/movement, and its reaction to nitroglycerin helps diagnose cardiac chest pain. Typical angina pectoris is the presence of substernal chest pain or discomfort provoked by exertion or emotional stress, and is relieved by rest and nitroglycerin. Angina pectoris is attributed to myocardial ischemia due to the unbalance between myocardial oxygen supply and myocardial oxygen demand elicited during exercise or emotional stress.

The diagnosis and treatment workup in patients with moderate to high cardiovascular risk presenting angina pectoris is quite straight-forward. Nevertheless, exertional chest pain in a young man can be challenging and yield a common but overlooked diagnosis, as in the present case report.

Case report

An 18-year-old professional, right-handed tennis player sought out a sports cardiology clinic due to several exertional chest pain episodes. In the last four months, the patient has been experiencing frequent bouts of oppressive, substernal chest pain, provoked after 20-30 minutes of running or during long tennis rallies. The pain was severe enough to make him stop exercising but usually lasted less than five minutes. These episodes were limiting the patient’s ability to train and compete. There were no complaints about syncope, pre-syncope, dyspnea, or palpitations. Past medical history was unremarkable, except for allergic rhinitis. The patient denied using any drugs, tobacco, or even nutritional supplements. There was no family history of coronary artery disease or sudden cardiac death.

Physical exam was normal, with unremarkable heart and lung auscultation. At rest, blood pressure was 120 x 70 mmHg, and the heart rate was 71 bpm. The resting electrocardiogram presented tall T waves in precordial leads that were compatible with a vagotonic pattern.

The patient was submitted to a maximal cardiopulmonary exercise test following an incremental ramp protocol on a treadmill (ATL, Inbrasport, Brazil). Oxygen uptake (VO₂), carbon dioxide (VCO₂), and ventilation (VE) were registered every ten seconds using a metabolic cart (Handymet, MDI, Brazil). Forced expiratory volume in one second (FEV₁) was measured immediately before the exercise test and in several moments after peak exercise (immediately, 5 minutes, 10 minutes, and 15 minutes) (Smart One, MIR, USA). A 12-lead electrocardiogram was continuously recorded (XCribe, Mortara, USA), and non-invasive blood pressure was measured each two-minutes.

The ventilatory threshold was identified by the combination of the following methods: the point of the first upward inflection of the ventilation vs. time curve, at the beginning of a consistent increase in the ventilatory equivalent for O₂ (minute ventilation/oxygen consumption) without a concomitant increase in the ventilatory equivalent for carbon dioxide (minute ventilation/carbon dioxide production), and at the beginning of an increase in expired oxygen fraction.

Keywords

Chest Pain; Exercise; Angina Pectoris; Asthma Exercise-Induced; Bronchial Spasm.

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The ventilatory threshold was considered as the point identified by at least two of these three criteria.

Respiratory compensation point would have been identified at the point of the second upward inflection of the ventilation vs. time curve, which was concomitant with the beginning of a consistent increase in the ventilatory equivalent for carbon dioxide (minute ventilation/carbon dioxide production). However, the respiratory compensation point did not occur in this test. The maximum value of each variable during the final 30s of the exercise was used as peak variables.

The patient complained of mild chest pain after 8 minutes of exercising, and the test was interrupted at 10:40 min due to lightheadedness, moderately intense chest pain, and dyspnea. Chest auscultation at peak exercise revealed mild wheezing in both lungs. Symptoms disappeared in the first 5 minutes of recovery. Exercise electrocardiogram, VO$_2$, and oxygen pulse curves were normal, excluding exercise-induced myocardial ischemia.

Peak VO$_2$ was within normal values (95.5% of predicted, Table 1), but these were far below what is expected for athletes with a high aerobic component of training (>125% of predicted). Rest FEV1 was 3.76 L. There was a decrease in FEV1 during recovery, reaching a nadir of 2.67 L at 10 minutes after peak exercise. This decrease in 29% of FEV1 measured at the tenth minute of recovery is compatible with moderate exercise-induced bronchoconstriction (Figure 1).

After the diagnosis, the patient started treatment with the daily administration of an inhaled corticosteroid (Fluticasone) and a leukotriene receptor antagonist (montelukast), with no symptoms to date (follow-up of six months). The patient is training and competing at a local level with no discomfort.

**Discussion**

Exercise-induced bronchoconstriction represents the narrowing of the acute airway during exercise.$^4$ Although this diagnosis can be linked to asthma, it may occur in up to 70% of athletes without asthma.$^5$ Symptoms of exercise-induced bronchoconstriction are nonspecific, and the presence of respiratory symptoms (such as dyspnea) are not always present. As this syndrome is usually present in athletes, the inability to perform high-intensity endurance exercise is one of the most common complaints.$^4,6,7$

Chest-tightness is a frequent complaint in these cases and usually leads to a full workout to rule out the risk of sudden cardiac death. One of the fundamental core competencies of the cardiologist who is taking care of an athlete is to reduce the risk of adverse cardiovascular outcomes during intense physical activity.$^5$ Nevertheless, once lethal causes of chest pain have been ruled out, we must remember that most chest pain in athletes is musculoskeletal or respiratory. In this context, if the diagnosis is kept unrevealed after anamnesis and physical examination, the cardiopulmonary exercise test is the gold standard to investigate exercise-induced chest discomfort in athletes.$^3,7$

Nevertheless, one must take into account that the usual cardiopulmonary testing protocol does not include FEV1 measurements after peak exercise.$^3$ Moreover, wheezing may not be present in cases of

| Table 1 – Cardiopulmonary exercise test parameters at the ventilatory threshold and peak exercise |
|-------------------------------------|-----------------|-----------------|
| Parameter                           | Ventilatory threshold | Peak exercise   |
| Heart rate (bpm)                    | 142              | 181             |
| Heart rate (% predicted)            | ----             | 89,6            |
| Treadmill speed (km/h)              | 10,9             | 17,2            |
| Treadmill grade (%)                 | 2                | 2               |
| VO$_2$(mL/Kg/min)                   | 30,7             | 46,7            |
| VO$_2$ (% predicted)                | ---              | 95,5%           |
| Ve/VCO$_2$ slope                    | ---              | 27,4            |
| Ve (L/min)                          | 55,03            | 122,7           |
| VCO$_2$/VO$_2$                      | 0,92             | 1,31            |
exercise-induced bronchoconstriction. Thus, in cases where exercise-induced bronchoconstriction is being investigated, a specific testing protocol should be considered. The protocol should include FEV1 measurements before and at several moments after exercise (minutes 0, 5, 10, 15, and 30 of recovery). The severity of exercise-induced bronchoconstriction can be graded according to the maximum percent of fall in FEV1 from the pre-exercise level, as follows: mild ≥ 10% but < 25%; moderate ≥25% but < 50%; and severe ≥50%.

Conclusion

Exercise-induced bronchoconstriction should be considered a cause of chest pain elicited during exercise in athletes after ruling out other potentially lethal causes.

Author contributions

Conception and design of the research: Castro R. Acquisition of data: Castro R, Giffoni J. Analysis and interpretation of the data: Castro R, Roberta Castro, Moreno A. Writing of the manuscript: Castro R, Orsini M. Critical revision of the manuscript for intellectual content: Castro R, Moreno A, Orsini M. Supervision / as the major investigator: Castro R.

Potential Conflict of Interest

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Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.
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