Exercise induced atrio-ventricular (AV) block during nuclear perfusion stress testing: a case report

Blocco atrio-ventricolare indotto dall’esercizio durante durante scintigrafia perfusionale miocardica: descrizione di un caso

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Background. Exercise causes enhanced sympathetic discharge and results in physiologic tachycardia. However, in some patients with a diseased conduction system resulting from acute ischemia, exercise can precipitate heart block.

Methods and results. In this report we describe a 51 years old male patient with transient advanced degree atrio-ventricular (AV) block developed during recovery from exercise stress testing, resolved after the administration of atropine. Nuclear perfusion imaging demonstrated stress-induced ischemia of the inferior-apical segments, and recovery of perfusion in the images obtained at rest. Coronarography showed critical stenosis of the right coronary artery, which was treated by percutaneous coronary intervention (PCI) and drug eluting stent (DES) deployment.

Conclusion. Nuclear myocardial perfusion imaging provides noninvasive evidence that transient ischemia of the infero-apical segment can result in advanced degree AV block in patient with critical severe right coronary disease.

Keywords: atrio-ventricular block, nuclear myocardial perfusion imaging, exercise stress testing.

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Figure 1. - Pre-test standard 12-lead electrocardiogram (ECG).

Figure 2. - ECG at the second recovery minute: ischemic changes in D1, aVL and V4-V6 leads appeared and a complete symptomatic (dizziness) AV block occurred, with idioventricular rhythm at 30 bpm.
One year after PCI + DES exercise stress test was repeated with the same Bruce protocol. Block did not recur and the patient remained symptom-free during the follow-up.

**Discussion**

Experimental studies in animals have demonstrated that excitation of vagal sensory nerve endings from myocardial ischemia involving the inferoposterior wall of the left ventricle activates potent cardioinhibitory reflex resulting in bradycardia and hypotension [1, 2]. In humans, similar observations have been made under particular conditions of severe transmural inferior ischemia and its reperfusion, such as those occurring with myocardial infarction, vasospastic angina, or angioplasty of the right coronary artery [3-7]. Despite these well-recognized clinical observations, little attention has been paid to the question of exercise-induced atrioventricular (AV) block during nuclear perfusion stress testing: a case report.
as to whether this reflex could be evoked by exercise-induced ischemia that is usually subendocardial with the manifestation of ST depression and that might be recurrently experienced during daily activities [8-11]. The present case indicated that exercise-induced subendocardial ischemia may augment vagal activity and may evoke the cardioinhibitory reflex, which would in turn influence postexercise heart rate decay. This is in agreement with Tahara et al. [12] that reported on fifty-two patients who developed sinus deceleration during exercise testing, all of whom had angiographically documented RCA lesion. The authors speculated the role of Bezold-Jarisch reflex in this mechanism and stated that the prevalence of deceleration during exercise appears to be very low. Sinus deceleration during exercise may be an extreme example caused by an ischemia-mediated reflex [13, 14].

Thus this reflex phenomenon is presumably operative during exercise-induced ischemia as well as during post exercise reperfusion; however, we focused on post exercise heart rate dynamics for the following reasons. Since vagal activity is physiologically attenuated in proportion to the increase in exercise intensity, this reflex might be masked during exercise. In contrast, potent reactivation of vagal nerve activity after exercise may accelerate the appearance of this reflex under a higher vagal condition after exercise. The physiological implication of this reflex, namely, what role this reflex may play, is unknown. The possibility that the reflex cardioprotectively works thorough the reduction in myocardial oxygen demand or that the resultant high vagal tone prevents the development of serious ventricular arrhythmias is of interest [15, 16]; however, there are few available data to support this so far.

In conclusion, we can consider that the vagal over-activation after exercise may be useful in predicting the presence of inferior ischemia when significant exercise-induced ST depression are observed. It may also be useful in patients after angioplasty of RCA to predict restenosis or to confirm the therapeutic effects.

**Riassunto**

**Introduzione.** Durante l’esercizio fisico si verifica una complessa serie di eventi che permette al cuore di aumentare la sua funzione di pompa. Il più importante di questi meccanismi è l’incremento della frequenza cardiaca, attraverso l’attivazione del sistema simpatico. Tuttavia, in alcuni pazienti con una malattia del sistema di conduzione secondaria ad ischemia miocardica acuta, l’esercizio fisico può determinare l’insorgenza di un disturbo di conduzione tipo blocco atrio-ventricolare all’ECG di superficie.

**Materiale e metodi.** Riportiamo il caso clinico di un maschio di 51 anni di età, che durante la fase di recupero di un test da sforzo condotto al tappeto rotante secondo il protocollo di Bruce, ha presentato...
l’insorgenza di un blocco atrio-ventricolare avanzato, risolto con la somministrazione di atropina e l’infusione endovenosa di liquidi.

Lo studio perfusionale miocardico condotto con metodica scintigrafica GATED-SPECT, mostrava un difetto reversibile a carico dei segmenti infero-apicali del ventricolo sinistro. La coronarografia successiva metteva in evidenza una stenosi critica dellaarteria coronarica destra, trattata con angio-plastica percutanea ed applicazione di stent medicato.

Conclusioni. Nel caso presentato, la scintigrafia miocardica di perfusione ha permesso di mettere in evidenza una sofferenza ischemica miocardica transitoria associata ad un blocco atrio-ventricolare avanzato all’ECG di superficie, in un paziente con severa patologia aterosclerotica della coronaria destra.

References


