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Case Study

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SEVERE SYMPTOMTIC SINUS BRADYCARDIA ASSOCIATED WITH THE USE OF ANABOLIC ANDROGENIC STEROIDS IN BODYBUILDING

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ABSTRACT

The use of anabolic androgenic steroids is a widespread practice in bodybuilding for its well-known effect on enhancing skeletal muscle gain despite their adverse effects on cardiovascular system. We report a first case of a 27 years old male amateur bodybuilder who presented a severe symptomatic sinus bradycardia after using an anabolic androgenic steroids to gain muscle mass. *Learning objective*: Cardiovascular screening should be performed in every amateur or professional athlete who has used anabolic androgenic steroids.

KEYWORDS: Sinus bradycardia, anabolic androgenic steroids, Dianabol, methandrostenolone, bodybuilding.

INTORDUCTION

Long term use of anabolic androgenic steroids (AAS) has been associated with many structural and functional cardiac adverse effects including cardiac autonomic dysfunction in the form of enhancement of sympathetic tone and attenuation of vagal tone.[1-2] To our knowledge, no cases of AAS use induced sinus bradycardia have been reported in the medical literature.

CASE REPORT

A 27 years old male was referred to our Autonomous nervous system (ANS) exploration unit for symptomatic sinus bradycardia. Two months before his referral he started reporting dizziness, fatigue and weakness. He has no noticeable personal or family medical history and he is not taking any medications for any medical condition. He is a social smoker and drinker, he denies using drugs. He is a recreational bodybuilder and has been working out regularly for one year. He trains once to three times a week for 90 to 120 minutes. Otherwise, the patient claims that because he had not gain enough muscle, he started taking Dianabol (methandrostenolone). At first, he took 3 pills of 10mg a day for eleven days then he started taking 6 pills at once the day of workout for three more weeks on the advice of a more experienced friend at the gym. In addition, he started taking at the same time half a spoon of creatine powder with water immediately after each workout, there was no change in workout intensity nor diet. The onset of the symptoms was slow and progressive and could be tracked between the second and third week of the start of his plan.

Cardiac examination found no particular signs except a sinus bradycardia at 36 bpm for a blood pressure of 109/60 mmHg. Respiratory and neurological examination was normal. ECG showed a sinus bradycardia at 36 bpm and a rest ECG performed two years earlier in a pre-recruitment medical check-up showed a normal sinus rhythm at 69 bpm. Laboratory test including CBC, electrolytes, renal function, liver function and thyroid function were normal. Trans-thoracic cardiac ultrasonography was normal, Holter ECG revealed a sinus basal rhythm during the 24 hours with a lowest heart rate of 28 beats per minute and highest rate of 130 as well as non-significant supraventricular excitability abnormalities.

ANS testing results were as follow *(Table 1)*: Baseline heart rate (HR) at 36 bpm and Blood pressure (BP) was 111/60 mmHg in the right arm and BP: 102/58 mmHg in the left arm. After 30 minutes of rest BP was 108/62 mmHg and HR: 36 bpm. Deep breathing test showed an increase of 114% in HR jumping up from 34 to 74 bpm (fig. 1) and a decrease in BP from 107/57 mmHg to 102/56 mmHg. Hyperventilation test showed an increase in HR from 38 to 56 bpm and a decrease in BP from 111/66 mmHg to 100/61 mmHg. Hand grip test increased BP from 101/64 mmHg to 113/76 mmHg and HR from 40 to 53 bpm. Mental stress test increased BP from 107/61 to 121/69 mmHg and HR from 38 to 47 bpm. Tilt Test was performed by a passive movement from a supine position to an upright position that was held for 10 minutes then followed by 10 minutes rest in a supine position. In the first minute HR increased from 42

bpm and then continued rising till 54 bpm. BP increased from 101/56 to 114/76 in the first minute then started decreasing to 98/65mmHg. Return to the supine position was associated with a drop in HR to 38 bpm and an increase in BP. Those tests showed very severe vagal hyperactivity associated with a normal alpha sympathetic activity and an increase in beta sympathetic activity.

Table 1: Results of cardiac autonomic system tests showing the variability of heart rate and blood pressure during the test compared to the baseline value before the test.

		Heart Rate			Blood Pressure		
		Baseline (bpm)	Highest /lowest (bpm)	Variability Δ HR (%)	Baseline (mmHg)	Highest /lowest (mmHg)	Variability Δ BP (%)
Baseline			-	-		-	-
Deep Breathing		34	74	+117	107/57	102/56	-4,7
Hand Grip		40	53	+32	101/64	113/76	+11
Hyperventilation		38	56	+47	111/66	100/61	-9
Mental Stress		38	47	+23	107/61	121/69	+13
Tilt Test	**1st minute**	42	54	+28	101/56	114/76	+10
	10 next minutes	54	61	+12	114/76	98/65	-14%
	Supine	38	-	-	102/64	-	-

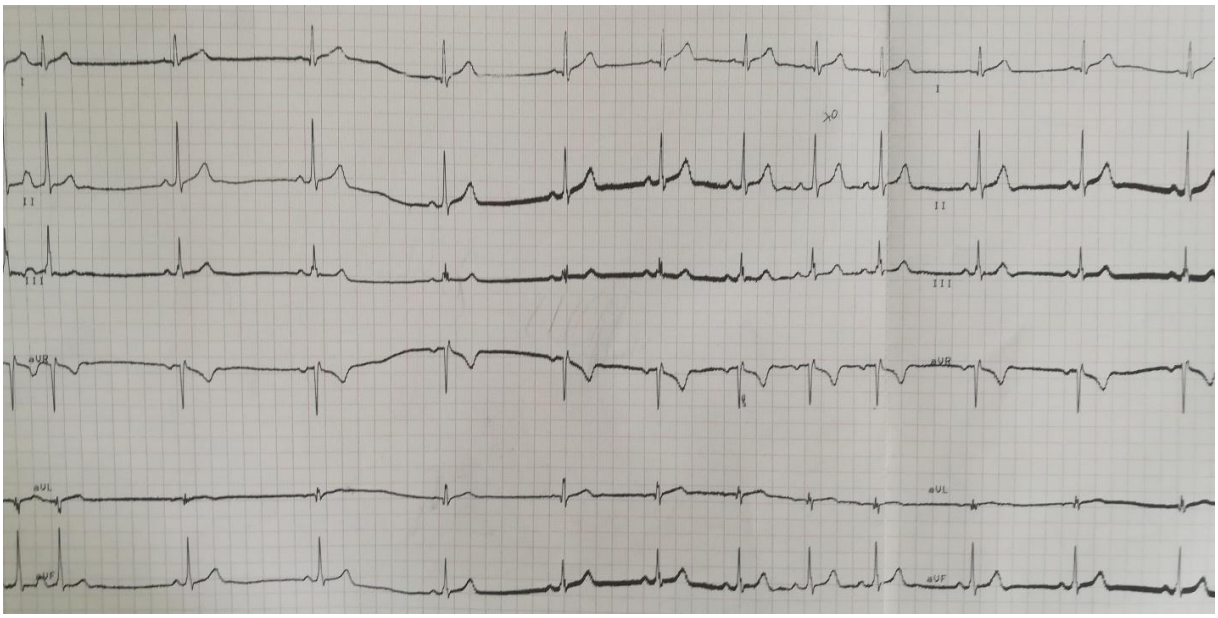

Fig. 1: ECG during Deep breathing test showing a severe sinus arrhythmia with an increase in heart rate in expiration and a decrease in inspiration which reflects a hypervagal tone.

Dianabol and creatine powder were discontinued, the patient continued his training as before, and he was advised non pharmacological measures (2 to 3l water intake and isometric counter pressure maneuvers). HR was self-monitored by a finger oximeter three times per day at rest and the results were recorded. At a three months follow up he claimed to be asymptomatic, ECG showed a sinus rhythm at 52 bpm. Self-monitored HR showed a progressive raise in HR with a lowest HR at 46 BPM the week before the follow up.

DISCUSSION

In our case, the ANS study has shown that the sinus bradycardia was the consequence of a significant increase in vagal activity demonstrated by the deep breathing test and the orthostatic test. This vagal hyperactivity could not be explained by the exercise because of its short duration, moderate intensity and the occurrence of symptoms which is not a common in athletes. We related the increased vagal tone to the use of Dianabol because of its absence in a previous ECG, the concomitant occurrence of clinical symptoms and the absence of any other cause that can explain it.

Unlike our results, Neto and al found that long term AAS use in bodybuilders is associated with a marked sympathetic predominance and vagal attenuation.[3] The bodybuilders included in Neto's study were different from our patient in regard to the duration of use and the engagement to exercise. Our patient is a recreational bodybuilder who used AAS for one month while Neto's bodybuilders were competitive and used AAS for two years or more. Furthermore, the substances used were not mentioned. We consider this point of high

importance as other steroids have shown to have different effects on the ANS. First, Testosterone is the steroid hormone whose effect on the cardiovascular system has been studied the most. Animal studies have shown that short or long-term testosterone administration to rats leads to rest bradycardia.[4] In addition, testosterone has a modulatory effect on baroreflex bradycardia by enhancing vagal activity.[5] Androgen depletion by castration or androgen receptor blockade attenuates baroreflex bradycardia.[6] In patients with heart failure and testosterone deficiency, testosterone supplementation improves the vagally mediated baroreflex sensitivity[7] and reduces peripheral vascular resistance which implies an attenuation of alpha adrenergic activity.[8] Secondly, acute occurrence of sinus bradycardia following oral or venous administration of corticosteroids has been widely reported in the medical literature.[9,10]

The occurrence of hypervagal tone and sinus bradycardia in this case raises many questions: are steroids effects on cardiac autonomous system molecule, dose and duration dependent? To which extent does it interact with exercise, diet and other doping agents? Conducting strong studies to answer those questions raises in itself ethical issues of doping in sports and is prohibited in many countries.

CONCLUSION

The adverse effects of AAS as doping agents are undeniable and could be fatal. Their risk of occurrence is difficult to assess because of the diversity of molecules, durations and protocols of use, besides the association with many other doping agents at different exercise levels. We suggest that every amateur or professional athlete who has used AAS should undergo a cardiovascular screening.

CONFLICT OF INTEREST AND SOURCE OF FUNDING

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