Prevalence and Management of Systemic Hypertension in Athletes

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The aim of the present study was to evaluate the prevalence, determinants, and clinical management of systemic hypertension in a large cohort of competitive athletes: 2,040 consecutive athletes (aged 25 ± 6 years, 64% men) underwent clinical evaluation including blood test, electrocardiogram, exercise test, echocardiography, and ophthalmic evaluation. Sixty-five athletes (3%) were identified with hypertension (men = 57; 87%) including 5 with a secondary cause (thyroid dysfunction in 3, renal artery stenosis in 1, and drug induced in 1). The hypertensive athletes had greater left ventricular hypertrophy and showed more often a concentric pattern than normotensive ones. Moreover, they showed a mildly reduced physical performance and were characterized by a higher cardiovascular risk profile compared with normotensive athletes. Multivariate logistic regression analysis showed that family hypertension history (odds ratio 2.05; 95% confidence interval 1.21 to 3.49; p = 0.008) and body mass index (odds ratio 1.32; 95% confidence interval 1.23 to 1.40; p <0.001) were the strongest predictors of hypertension. Therapeutic intervention included successful lifestyle modification in 57 and required additional pharmacologic treatment in 3 with essential hypertension. Secondary hypertension was treated according to the underlying disorder. After a mean follow-up of 18 ± 6 months, all hypertensive athletes had achieved and maintained optimal control of the blood pressure, without restriction to sport participation. In conclusion, the prevalence of hypertension in athletes is low (3%) and largely related to family history and overweight. In the vast majority of hypertensives, lifestyle modifications were sufficient to achieve an optimal control of blood pressure values. **(Am J Cardiol 2017;119:1616−1622)**

Because of their commitment to regular exercise training and healthy lifestyle, athletes are expected to present lower prevalence of hypertension than the general population. Therefore, an apparent paradox exists between the expected and the reported prevalence of systemic hypertension in athletic population.[1−7] The present study was designed to assess the actual prevalence, determinants, and clinical management of systemic hypertension in a large cohort of young-adult, elite competitive athletes, evaluated in the context of our pre-participation medical program.

Methods

The study population consisted of 2,040 athletes, consecutively evaluated from January 2010 to December 2015 at the Institute of Sports Medicine and Science in Rome. All athletes were engaged in National and/or International competitions and were training regularly at the time of the evaluation. According to our medical program, they underwent an extensive cardiologic and clinical evaluation.[8,9] Written informed consent was waived for all athletes. The study design was approved by the Review Board of the Institute.

Based on the predominant characteristics of training, athletes were classified in 4 subgroups: (1) skill (i.e., primarily technical activities, n = 502), including golf, table tennis, equestrian, shooting, karate, archery, and sailing; (2) power (i.e., primarily isometric activities, n = 352) including weightlifting, shot-putting, hammer throwing, gymnastic, wrestling, and short-distance running (100 to 200 m); (3) mixed (i.e., disciplines with both isometric and isotonic components; n = 595), including soccer, basketball, volleyball, handball, water polo, tennis, and fencing; and (4) endurance (i.e. primarily isotonic activities; n = 591) including rowing, canoeing, swimming, long-distance running and marathon, cycling, triathlon, and pentathlon.[10]

Clinical evaluation was performed in all athletes in a resting day. Cardiovascular evaluation included recall of personal and family histories and physical examination.[8] None was taking cardiovascular or vasoactive medications at the time of the study entry. The use of energy drinks, herbal and dietary supplements, or other medications was recorded. Positive family history for hypertension was defined as a BP ≥140/90 mm Hg in a first-degree family member or the use of BP-lowering medications. Office BP was measured in a quiet room by an experienced cardiologist, with the athlete in a sitting position after few minutes of rest and just before the exercise test. The cuff was positioned

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See page 1621 for disclosure information.

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Table 1
Clinical and demographic characteristics of study population according to gender

Variable	Female Athletes (n=730)	Male Athletes (n=1310)	p value
Age (years)	24±5	25±6	<0.001
Height (cm)	170±9	182±10	<0.001
Weight (Kg)	63±11	78±13	<0.001
Body Surface Area (m^2)	1.72±0.18	1.99±0.21	<0.001
Body Mass Index (Kg/ m^2)	21.7±2.8	23.5±2.7	<0.001
Fat Mass (%)	23±5	14±5	<0.001
Smoker	18 (2.5%)	31 (2.8%)	0.888
Systolic Blood pressure (mmHg)	110±11	117±10	<0.001
Diastolic Blood pressure (mmHg)	71±7	75±7	<0.001
Basal Heart Rate (bpm)	59±12	57±11	<0.001
Glucose (mg/dl)	91±8	95±8	<0.001
Total Cholesterol (mg/dl)	186±35	176±34	0.004
HDL Cholesterol (mg/dl)	71±17	59±14	<0.001
LDL Cholesterol (mg/dl)	102±28	104±30	0.356
Triglycerids (mg/dl)	71±28	79±40	<0.002
Creatinine (mg/dl)	0.88±0.15	1.01±0.16	<0.001
Echocardiography			
Left Ventricular Wall Thickness (mm)	8.7±0.9	10.1±1.1	<0.001
Left Ventricular End-diastolic Diameter (mm)	49±3	54±4	<0.001
Relative Wall Thickness	0.35±0.02	0.37±0.03	<0.001
Left Ventricular Ejection Fraction (%)	65±6	64±6	<0.001
Left Ventricular Mass (g)	146±37	210±56	<0.001
Left Ventricular Mass Index (g/m^2)	84±16	106±22	<0.001
Left Atrial antero-posterior diameter (mm)	32±4	36±4	<0.001
E/A ratio	1.95±0.48	1.94±0.51	0.452
e′ wave velocity (cm/s)	14.2±2.0	13.7±2.1	<0.001
E/e′ ratio	6.6±1.2	6.4±1.2	<0.001

at the upper arm (heart level) with cuff size and bladder dimension adjusted to the arm circumference; auscultatory technique was used, and phase I and V of the Korotkoff sounds were used to evaluate the systolic and diastolic BP, respectively. At least 2 measurements were performed, spaced 1 to 2 minutes apart, and the averaged was recorded.[11] Blood pressure levels were classified according to the recommendations of the European Society of Cardiology (ESC), that is, optimal (systolic <120 and diastolic <80 mm Hg), normal (systolic 120 to 129 and/or diastolic 80 to 84 mm Hg), high normal (systolic 130 to 139 and/or diastolic 85 to 89 mm Hg), grade 1 hypertension (systolic 140 to 159 and/or diastolic 90 to 99 mm Hg), and grade 2 hypertension (systolic 160 to 179 and/or diastolic 100 to 109 mm Hg).[11]

Body height and weight were obtained; body mass index (BMI) and body surface area were subsequently calculated. Body composition and fat percentage were measured using Bioelectric Impedance Analysis (BIA 101 Quantum; Akern, Pontassieve, Italy).[12] As a part of the medical program, all athletes underwent ophthalmologic examination including assessment of fundus oculi. Blood samples were collected at the time of clinical evaluation to measure fasting glucose levels, lipid profiles, renal, and thyroid function. The exercise testing was performed on bicycle ergometer (Cubestress XR400; Cardioline SpA, Milan, Italy). The starting load was

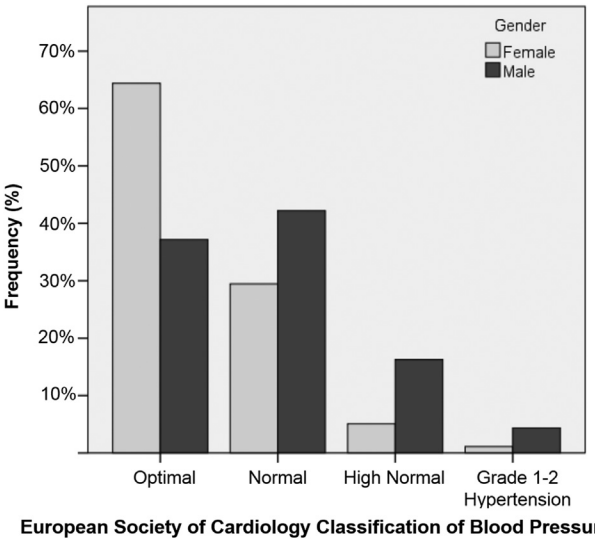

Figure 1. Distribution of the athletic population according to the European Society of Cardiology classification of office blood pressure.

0.5 Watts/kg, with subsequent increase of 0.5 Watts/kg every 2 minutes until exhaustion. The BP during exercise was measured as previously reported.[13] Exercise performance was measured as the maximum exercise workload divided by body weight (Watt/kg).

All athletes identified with high BP on first clinical evaluation, that is, ≥140/90 mm Hg, entered a periodical follow-up evaluation, usually with 6-month interval, and up to 3 years (average 18 ± 6 months).

Two-dimensional and Doppler echocardiography was performed using iE33 or Epiq (Philips Medical System, Andover, Massachusetts). Two-dimensional measurements of left ventricular (LV) cavity, wall thickness, left atrium, and aortic root diameters were performed according to current recommendations.[14] LV ejection fraction was measured by the biplane Simpson's rule. LV mass was measured by Devereux's formula and normalized to body surface area.[11] LV hypertrophy was defined as LV mass index >95 g/m^2 in women or >115 g/m^2 in men.[11,14] The pattern of LV hypertrophy was defined concentric when associated with a relative wall thickness (RWT) >0.42 and eccentric when RWT was ≤0.42; concentric remodeling was defined as normal LV mass with RWT >0.42 and normal geometry as normal LV mass and RWT ≤0.42.[11,14] LV diastolic function parameters included early (E) and late (A) diastolic transmitral velocities measured by pulse-wave Doppler and early (e′) diastolic myocardial velocity measured by tissue Doppler imaging; E/A and E/e′ ratios were subsequently calculated.[10,15]

Continuous data were expressed as mean ± SD, and categorical data as number of observations and frequencies. Statistical significance was set for a p value <0.05. Differences between groups were evaluated with unpaired *t* test and Levene's test for the equality of variance; difference between proportions was calculated by the chi-square test. One-way ANOVA with post hoc Bonferroni correction was instead used to assess differences in terms blood pressure subgroups according to the ESC classification. Reduction of blood pressure during follow-up was assessed with

Table 2
Clinical, metabolic and echocardiographic characteristics of the study population according to European Society of Cardiology classification of office Blood Pressure

Variable	Optimal (n=957)	Normal (n=768)	High Normal (n=250)	Grade 1-2 (n=65)	p value
Age (years)	23±6	25±6	26±6	26±6 *	<0.001
Gender (male)	490 (50%)	550 (71%)	215 (85%)	55 (87%)	<0.001
Height (cm)	174±10	180±11	182±10	184±10 *	<0.001
Weight (Kg)	67±12	75±13	80±15	88±18 *,†,‡	<0.001
Body Surface Area (m^2)	1.81±0.21	1.95±0.21	2.01±0.23	2.10±0.24 *,†,‡	<0.001
Body Mass Index (Kg/ m^2)	22.0±2.4	23.1±2.6	24.2±3.3	26.2±4.8 *,†,‡	<0.001
Fat Mass (%)	18±7	17±7	17±6	22±9 *,†,‡	0.021
Positive Family History	220 (23%)	197 (26%)	69 (27%)	24 (38%) *,†,‡	0.030
Smoker	29 (3%)	16 (2%)	4 (2%)	0	0.246
Systolic Blood Pressure (mmHg)	105±8	118±5	128±5	136±11 *,†,‡	<0.001
Diastolic Blood Pressure (mmHg)	67±6	77±5	79±6	86±7 *,†,‡	<0.001
Basal Heart Rate (bpm)	57±11	58±11	58±11	61±10	0.083
Maximal Workload (Watts)	228±58	253±56	264±58	264±53 *	<0.001
Performance (Watt/Kg)	3.41±0.71	3.38±0.76	3.35±0.73	3.05±0.70 *,†,‡	0.001
Maximal Systolic Blood Pressure (mmHg)	174±20	188±19	197±20	209±22 *,†,‡	<0.001
Maximal Diastolic Blood Pressure (mmHg)	71±8	76±8	77±8	83±10 *,†,‡	<0.001
Glucose (mg/dl)	93±9	93±8	94±8	93±9	0.296
Total Cholesterol (mg/dl)	180±35	181±35	179±35	183±27	0.862
HDL Cholesterol (mg/dl)	65±15	63±18	58±15	58±16	<0.001
LDL Cholesterol (mg/dl)	100±30	106±28	107±30	110±24 *	0.011
Triglycerids (mg/dl)	72±31	78±39	83±44	79±39	0.009
Creatinine (mg/dl)	0.94±0.17	0.97±0.17	1.02±0.16	1.03±0.17	<0.001
Echocardiography					
Left Ventricular Wall Thickness (mm)	9.2±1.1	9.8±1.1	10.2±1.1	10.5±1.1 *,†	<0.001
Left Ventricular End-diastolic Diameter (mm)	50.9±4.3	53.2±4.4	54.5±4.3	55.3±3.9 *,†	<0.001
Relative Wall Thickness	0.36±0.03	0.36±0.03	0.37±0.03	0.37±0.03 *	<0.001
Left Ventricular Ejection Fraction (%)	65±6	64±6	64±6	64±5	0.003
Left Ventricular mass (g)	169±48	198±52	215±54	230±52 *,†,‡	<0.001
Left Ventricular mass index (g/m^2)	92±21	101±22	107±23	108±21 *,†,‡	<0.001
Left Atrial antero-posterior diameter (mm)	33.5±4.1	35.1±4.2	36.4±4.1	37.4±4.2 *,†	<0.001
Aortic root (mm)	29±4	31±4	32±4	32±4 *,†	<0.001
E/A ratio	1.98±0.46	1.92±0.52	1.90±0.54	1.91±0.48	0.052
e′ wave velocity (cm/s)	14.1±1.9	13.8±2.2	13.6±2.2	13.0±2.0 *,†	<0.001
E/e′ ratio	6.5±1.2	6.4±1.2	6.4±1.3	6.8±1.2	0.149

Differences between groups were evaluated with One-way Analysis of Variance. Post-hoc Bonferroni correction was used to identify differences between hypertensive athletes compared to other groups.
*p <0.05 versus optimal; †p <0.05 versus normal; ‡p <0.005 versus high-normal.

paired-samples *t* test. Binary logistic regression analysis was used to identify the variables associated with ESC grade 1 to 2 hypertension. Factors with a univariate value of p <0.05 were included in a stepwise multivariate logistic regression analysis. Categorical variables comprised gender, family history, smoking habit, and type of sport. BMI was inserted as a continuous variable. Specifically, female gender, negative family history, and no-smoking were chosen as the reference status; the impact of type of sport was assessed by a binary categorical variable using N-1 dummy variables, with skill disciplines chosen as the reference value. Statistical analysis was performed with SPSS software (version 22; SPSS Inc., Chicago, Illinois).

Results

The clinical characteristics of the athletes' population according to gender are reported in Table 1. In the overall population, average systolic and diastolic BP were 114 ± 11 and 73 ± 8 mm Hg, respectively. Both values were mildly higher in male compared with female athletes. A positive history of family hypertension was reported in 25% of the study population, and only a small minority of subjects (n = 49; 2.4%) were current smokers.

According to the ESC classification, optimal BP was found in 960 (47%), normal BP in 765 (38%), and high-normal in 252 (12%). Diagnosis of hypertension was made in 65 athletes (3.2%); of these, 64 had grade 1 and only 1 had grade 2 hypertension (Figure 1).

Athletes with hypertension were in the large majority men (87%) and were evenly distributed among skill (n = 12; 2.4%), power (n = 17; 4.8%), mixed (n = 18; 3.0%), and endurance (n = 18; 3.0%) sports disciplines (p = 0.241). Compared with the nonhypertensive subjects, they had a higher prevalence of family hypertension history and larger body size (Table 2). In detail, 51% were overweight (BMI ≥25 kg/m^2), including 22% obese (BMI ≥30 kg/m^2) with a BMI up to 41 kg/m^2 (in a male weightlifter with a fat mass of 31% and a female shot-putter with a fat mass of 39%). Percentage of body fat was consistently increased in

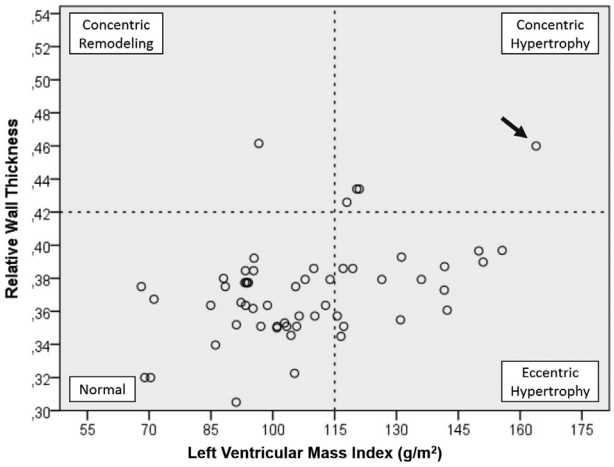

Figure 2. Patterns of LV geometry in athletes. *Dot line* represents the cut-off values of 0.42 for relative wall thickness and 115 g/m^2 for LV mass. The highest degree of concentric hypertrophy was seen in an athlete with renovascular hypertension who had a LV mass index of 164 g/m^2 and a relative wall thickness of 0.46 *(arrow)*.

subjects in the obesity (29 ± 8%) range compared with other BMI categories (17 ± 6%; p <0.001).

With regard to metabolic parameters, no differences were detected in terms of fasting glucose and total cholesterol; conversely, hypertensive athletes had mildly higher LDL and lower HDL compared with the other groups; finally, creatinine level was mildly higher in the hypertensive ones (Table 2). None of the hypertensive athletes had signs of retinopathy on ophthalmic evaluation. On exercise testing, hypertensive athletes reached the highest values of peak systolic and diastolic BP, but their physical performance was lower compared with normotensive athletes.

In terms of cardiac morphology, athletes with hypertension showed larger LV remodeling, as expressed by higher LV wall thickness, cavity size, and mass. Normal LV geometry was found in 73% normotensive and 61% hypertensive athletes (p = 0.039); eccentric LV remodeling was found in 25% normotensive and 31% hypertensive athletes (p = 0.321); concentric remodeling was seldom observed in both groups (0.7% and 1.5%, respectively; p = 0.441); conversely concentric hypertrophy was more frequently seen in hypertensive compared with normotensive athletes (6% vs 0.8%; p <0.001; Figure 2). Aortic root was mildly larger in athletes with hypertension compared with those with optimal and normal BP. LV ejection fraction and diastolic function parameters were within normal limits in all athletes.[10,11]

Multivariate logistic regression analysis showed that family history of hypertension (OR 2.05; 95% CI 1.21 to 3.49; p = 0.008) and BMI (OR 1.32; 95% CI 1.23 to 1.40; p <0.001) were the only predictive indexes of hypertension in our study population (Supplementary Table 1).

Clinical management of athletes with hypertension is depicted in Figure 3. A secondary cause of hypertension was identified in 5 (8% of the hypertensives) and included thyroid function disorders (n = 3), renovascular (n = 1), and drug-induced hypertension (n = 1). Athletes with thyroid dysfunction included Hashimoto's thyroiditis (n = 1) and hypothyroidism (n = 2) that were easily identified on routine blood tests; they were referred to the endocrinologist and advised to start hormone replacement therapy.

The only athlete with grade 2 hypertension was eventually diagnosed as renovascular hypertension. This 19-year-old water-polo player, with negative family history, presented high BP at rest (160/100 mm Hg) that markedly increased during exercise (at peak 280/125 mm Hg). Lifestyle and diet modification failed to normalize the BP, and an extensive investigation eventually revealed right inferior renal artery stenosis. After successful percutaneous transluminal angioplasty, the BP at rest decreased substantially but still remained at borderline level; therefore, pharmacologic treatment with angiotensin receptor blocker was started. At the most recent evaluation, after 6 months of treatment, BP returned within normal limits (<140/90 mm Hg).

The athlete with drug-induced hypertension was a boxer with low back pain treated with 8 mg/day of betamethasone for several weeks. After termination of therapy, the BP normalized.

The other 60 athletes were diagnosed as essential hypertension, in the absence of other abnormalities on cardiovascular examination and blood tests. All were advised for lifestyle and diet modifications according to the ESC guidelines: salt restriction, low alcohol consumption, avoidance of energy drinks or supplements, and large consumption of vegetables and fruits and low-fat diet.[11] After 6 months, these interventions demonstrated to be efficient to obtain normalization of baseline BP in 57 of 60 athletes. Therefore, an additional pharmacologic treatment was initiated in the remaining 3 with angiotensin-converting enzyme inhibitor or angiotensin receptor blockers, which on subsequent evaluations demonstrated to obtain good control of BP values (<140/90 mm Hg).

After an average follow-up of 18 ± 6 months, all the athletes with hypertension on baseline evaluation had achieved and maintained an optimal control of blood pressure values, with an average reduction of −13 mm Hg (95% CI from −13 to −16 mm Hg; p <0.001) for systolic and −8 mm Hg (95% CI from −6 to −10 mm Hg; p <0.001) for diastolic blood pressure.

Discussion

Our results depict a reassuring clinical scenario, by showing that prevalence of hypertension in a large population of competitive athletes (aged 18 to 40 years) is low (i.e., 3%). This prevalence seems to be lower compared with general population of similar age; in Italy, a national cross-sectional study in 493 subjects aged 18 to 35 years reported elevated BP values in 11% of young adults, which seems far higher than our athletic cohort.[16] In economically developed countries, the burden of hypertension in young adults has been reported as 14% and 21% in men aged 20 to 29 and 30 to 39 years, respectively, and as 6% and 10% in women in the same age groups.[17] Therefore, although the present study was not

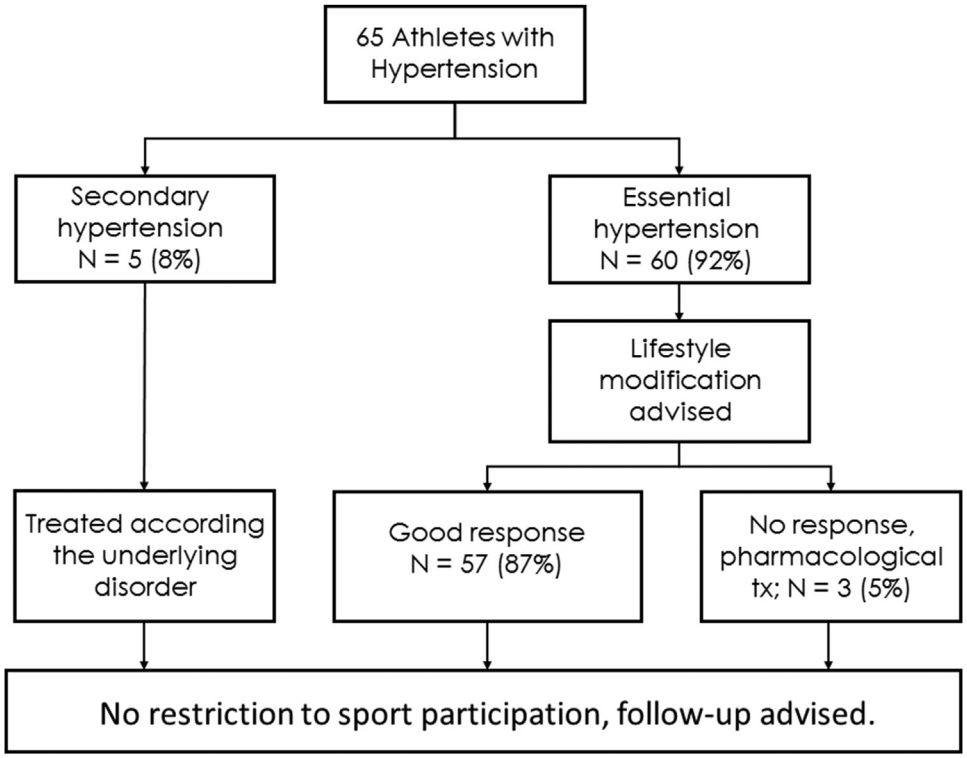

Figure 3. Clinical management of the 65 athletes with hypertension.

specifically addressed to compare athletes to the general population, our observation supports the thesis that athletes show a lower prevalence of hypertension, likely also as result of the beneficial effects of regular exercise training and healthy lifestyle.

Not surprisingly, high BMI and positive family history for hypertension were identified as the strongest predictors of hypertension on multivariate analysis.[16] Conversely, the type of sport participated was not a significant determinant of BP both in univariate and multivariate analyses. This seems to be in contrast with previous literature reporting a higher incidence of hypertension in certain sports disciplines, such as weight lifting, and also American football and baseball.[4,7,18,19] We are inclined to consider that regardless the characteristics of training (mainly isometric), other features should be advocated to explain the high prevalence of hypertension in these disciplines, including the large weight (American Football lineman may reach >300 pounds or 136 kg) and high BMI.[7,20] Consistently with these findings, majority of the hypertensive athletes identified in our study were overweight including some in the range of obesity. Because BMI may not be an accurate measure of obesity in athletes (it is increased also in subjects with increased free-fat muscular mass),[21] we confirmed that those with higher BMI categories were also characterized by larger percentage of body fat; eventually, athletes with hypertension showed higher fat percentage compared with normotensives (Table 2).

In our experience, athletes with hypertension were usually those with less healthy lifestyle, and (as indirect confirmation) of the 60 athletes with essential hypertension who were educated to modify their lifestyle, those who were adherent to diet and weight control also obtained an optimal control of BP values.[11]

Therefore, we recommend lifestyle modification as the first step in the clinical management of hypertensive athletes. However, it is worthy to mention that, in addition to lifestyle assessment, other factors associated with increased BP in young subjects should be assessed, including the use of anti-inflammatory drugs, supplements, high salt intake, psychosocial pressure, or abuse of anabolic steroids agents.[22–25]

In our large athlete's population, a small subset of 5 had a secondary cause of hypertension (8% of subjects with hypertension), with prevalence similar (5% to 10%) to other studies in young subjects.[26] In all the athletes with secondary hypertension, the responsible cause was corrected or adequately treated and all athletes eventually continued in training and competition. Full resumption of training schedule and competition was also favored in our hypertensive athletes by the lack of signs of extracardiac organ damage, including normal renal function and no abnormalities of retinal vasculature.

In terms of cardiac findings, an interesting observation of our study was that the vast majority of hypertensive athletes had a normal geometry or an eccentric hypertrophy of the LV; however, a small but significant minority of 6% had a concentric hypertrophy (compared with <1% in nonhypertensives) including 1 athlete with renovascular hypertension. Therefore, a concentric hypertrophy in a trained athlete should be viewed cautiously because it could be the expression of pathologic conditions, such as hypertensive heart disease, rather than the epiphenomenon of physiologic remodeling.[27–29]

Another remarkable finding was that athletes with hypertension achieved a lower performance on bicycle exercise test and their BP at peak exercise was higher compared with other groups. This result is in line with a previous study that reported reduced exercise capacity in hypertensive athletes, with an average 15% reduction in maximal oxygen consumption compared with nonhypertensive subjects.[30] An early and subclinical vascular dysfunction associated with increased arterial stiffness has been suggested to explain elevated BP at effort, potentially leading to the reduced performance.[5] Indeed, almost a half of hypertensive subjects (n = 30; 46%) attained high BP values at peak exercise (systolic BP> 220 mm Hg in men and >200 mm Hg in women).[13]

Finally, some limitation should be reported with regard to our study: first, our results apply uniquely to the Caucasian ethnicity (which comprises 98% of our athletes) and relatively young subjects; it would be interesting to assess the prevalence of hypertension also in other ethnicities and in older athletes. Then, in many cases, our clinical evaluation was not the first examination in the athlete's life; this may have underestimated the real prevalence of hypertension in the present study. Finally, American football athletes, that is, those described with the highest values of BP, were not included in our study because this sport is not part of the Olympic disciplines.

Disclosures

The authors have no conflicts of interest to disclose.

Supplementary Data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.amjcard.2017.02.011.

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