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Original article

Prevalence of Atherogenic Dyslipidemia in Spanish Hypertensive Patients and Its Relationship With Blood Pressure Control and Silent Organ Damage

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ABSTRACT

Introduction and objectives: To assess the prevalence of atherogenic dyslipidemia in hypertensive patients and its relationship with risk profile and blood pressure control.
Methods: The study included 24 351 hypertensive patients from the Spanish Ambulatory Blood Pressure Monitoring Registry. Atherogenic dyslipidemia was defined as the presence of hypertriglyceridemia (> 150 mg/dL) and low levels of high-density lipoprotein cholesterol (< 40 mg/dL in men and < 46 mg/dL in women). Blood pressure control was assessed by office and ambulatory monitoring.
Results: Atherogenic dyslipidemia was present in 2705 patients (11.1%). Of these, 30% had hypertriglyceridemia and 21.7% had low levels of high-density lipoprotein cholesterol. Compared with patients without these risk factors, the former group were more often male (60% vs 52%), younger (57 years vs 59 years), had other risk factors and organ damage (microalbuminuria, reduced estimated glomerular filtration rate, and left ventricular hypertrophy), worse office, diurnal, and nocturnal blood pressure values (odds ratio 1.09, 1.06, and 1.10, respectively), and the lowest nocturnal blood pressure reduction (odds ratio = 1.07), despite the greater use of antihypertensive drugs.
Conclusions: Atherogenic dyslipidemia is present in more than 10% of hypertensive patients and is associated with other risk factors, organ damage, and poorer blood pressure control. Greater therapeutic effort is needed to reduce overall risk in these patients.

Prevalencia de dislipemia aterogénica en hipertensos españoles y su relación con el control de la presión arterial y el daño orgánico silente

Palabras clave:
Hipertensión arterial
Dislipemia aterogénica
Monitorización ambulatoria de la presión arterial
Lesión del órgano diana
Control de la presión arterial
Hipertrigliceridemia

RESUMEN

Introducción y objetivos: El objetivo es evaluar la prevalencia de dislipemia aterogénica en pacientes hipertensos y su relación con el perfil de riesgo y el control de la presión arterial.
Métodos: Se estudió a 24.351 hipertensos del Registro Español de Monitorización Ambulatoria de la Presión Arterial. La dislipemia aterogénica se definió por la presencia de hipertrigliceridemia (> 150 mg/dl) y colesterol unido a lipoproteínas de alta densidad bajo (< 40 mg/dl en varones, < 46 mg/dl en mujeres). El control tensional se evaluó por clínica y monitorización ambulatoria.
Resultados: La dislipemia aterogénica estaba presente en 2.705 pacientes (11,1%). Un 30% presentaba hipertrigliceridemia y un 21,7%, colesterol unido a lipoproteínas de alta densidad bajo. Comparados con los pacientes sin dichas alteraciones, los primeros eran con más frecuencia varones (el 60 frente al 52%) y de menos edad (57 frente a 59 años) y se agregaban otros factores de riesgo y lesión orgánica (microalbuminuria, reducción del filtrado glomerular estimado e hipertrofia ventricular izquierda). El control de la presión arterial clínica, diurna y nocturna era peor (*odds ratio* de 1,09, 1,06 y 1,10 respectivamente) y el descenso tensional nocturno menor (*odds ratio* = 1,07) que en los pacientes sin alteraciones, pese a la mayor utilización de fármacos antihipertensivos.
Conclusiones: La dislipemia aterogénica está presente en más del 10% de los hipertensos y se asocia a otros factores de riesgo, lesión orgánica y peor control de la presión arterial. Es necesario un mayor esfuerzo terapéutico para la reducción general del riesgo de estos pacientes.

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Abbreviations

AD: atherogenic dyslipidemia
BP: blood pressure
HDL-C: high-density lipoprotein cholesterol
LDL-C: low-density lipoprotein cholesterol

INTRODUCTION

Hypertension is considered to be the main risk factor for death worldwide because of its impact on cardiovascular disease, which remains the leading cause of death.[1] However, the risk attributable to elevated blood pressure (BP) levels is modified by the presence of other risk factors. Lipid disorders are among the most important additional risk factors. The risk of hypertension and of dyslipidemia is exacerbated by the high prevalence of hypertension and dyslipidemia in combination.[2,3] These disorders are probably found together because they share common pathogenic factors, especially those stemming from the environment.[4,5] It is known that patients with hypertension have a higher rate of lipid disorders than the general population.[6,7]

The 2 main lipid disorders that appear in hypertensive patients are high levels of low-density lipoprotein cholesterol (LDL-C) and atherogenic dyslipidemia (AD), which is characterized by elevated triglyceride levels and low levels of high-density lipoprotein cholesterol (HDL-C) with or without high levels of LDL-C or non-HDL cholesterol. Non-HDL cholesterol is transported by all atherogenic lipoproteins and is used as a proxy for LDL-C levels when these cannot be calculated due to the presence of hypertriglyceridemia. Atherogenic dyslipidemia may occur in isolation or more often with other body fat distribution and carbohydrate metabolism abnormalities known as metabolic syndrome.[8] Little is known about the impact of AD on BP control or about the effects of therapeutic efforts in patients with AD.

Thus, the aim of the study was to assess the prevalence of AD and its components (low HDL-C levels and increased triglyceride levels) and its relationship with office and ambulatory BP control and silent organ damage in a large cohort of hypertensive patients.

METHODS

Patient Selection

The study included 24 351 patients from the Spanish Ambulatory Blood Pressure Monitoring registry (ABPM). This registry is based on the distribution of BP monitors to more than 1000 physicians in the 17 Spanish autonomous communities. The physicians were mainly from primary care centers, but were also from specialized cardiology, nephrology, and internal medicine units. The ABPM records and clinical patient data were sent to an internet-based platform. The physicians then received a report in realtime describing the main results. The details of the process have been described in previous articles published by our group.[9–13]

The registry began in June 2004 and by December 31, 2010 the database included 104 904 patients. The present study included patients with satisfactory ABPM record quality and basic clinical data, and laboratory data concomitant with the APBM readings (3 months) that included total cholesterol, HDL-C and triglyceride levels, and the number and type of antihypertensive and lipid-lowering drugs. Of the 24 351 patients included, 54.6% were men and the mean age was 59 (SD, 14) years. In total, 16 254 were receiving some type of antihypertensive treatment and the remaining 8097 patients were receiving no treatment.

Blood Pressure Measurement and Definition of Variables

Office systolic and diastolic BP were measured twice with a mercury sphygmomanometer or validated semiautomatic device after a 5-minute rest. The mean of the 2 measurements was used in the analysis.

Ambulatory blood pressure monitoring was preferentially conducted on a day of normal activity using a cuff matched to the size of the patient's arm. A record was considered valid when the percentage of successful readings was $\geq$ 70% of the total number of readings and the time between readings was never more than 1 hour. The means and standard deviations of 24-hour, diurnal (activity), and nocturnal (resting) systolic and diastolic BP and heart rate values were calculated based on patient logs.

Normotension/hypertension or control/no control (with or without antihypertensive treatment) were defined according to Spanish[14] and international[15] recommendations at the cutoff values of $<$ 140/90 mmHg for office BP, 130/80 mmHg for 24-hour BP, 135/85 mmHg for diurnal BP, and 120/70 mmHg for nocturnal BP.

The circadian pattern was estimated by calculating the night/day ratio for each parameter (systolic BP, diastolic BP, and heart rate). Risk profiles were defined according to the decrease in nocturnal systolic BP compared with diurnal systolic BP. The following categories were established: dipper (10%-20% decrease), extreme dipper (more than 20% decrease), nondipper (less than 10% decrease), and riser (nocturnal systolic BP higher than diurnal systolic BP).[11]

Definition of Clinical and Laboratory Variables

Body mass index (BMI) was calculated by dividing weight (in kilos) by the square of height (in meters). Patients were considered to be smokers if they had consumed any kind of tobacco in the last year. Diabetes mellitus was defined as glycemia $>$ 125 mg/dL or treatment with antidiabetic agents. Left ventricular hypertrophy was defined according to electrocardiographic criteria (Sokolow-Lyon index $>$ 38 mm or Cornell voltage-duration product$>$ 2440 mm/ms). Microalbuminuria was defined as urinary albumin excretion $>$ 30 mg/g. Chronic kidney disease (CKD) was defined as an estimated glomerular filtration rate $<$ 60 mL/min/1.73 m^2 using the Chronic Kidney Disease Epidemiology Collaboration equation.[16] Prior cardiovascular events were defined as documented myocardial infarction, stroke, hospitalization for unstable angina, or coronary or peripheral vascularization procedure.

Lipid levels (total cholesterol, triglycerides, HDL-C and LDL-C) were measured in the laboratories of each participating center using standard analytical procedures. Atherogenic dyslipidemia was defined as triglyceride levels $>$ 150 mg/dL and HDL-C levels $<$ 40 mg/dL in men and $<$ 46 mg/dL in women, regardless of lipid-lowering drug treatment and according to the normal values proposed for hypertensive patients.[14,15]

Statistical Analysis

Data are expressed as mean (standard deviation) for continuous variables with a normal distribution, median [interquartile range] for continuous variables with a non-Gaussian distribution (Kolmogorov-Smirnoff), or as percentages for categorical variables. Four groups were established: Patients with normal triglyceride and HDL-C levels, patients with normal triglyceride and low HDL-C