Published in final edited form as:

Nutr Metab Cardiovasc Dis. 2009 October ; 19(8): 532–541. doi:10.1016/j.numecd.2008.11.004.

Age- and gender-specific awareness, treatment, and control of cardiovascular risk factors and subclinical vascular lesions in a founder population: The SardiNIA Study

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Abstract

Aim—We investigated the gender-specific control of cardiovascular (CV) risk factors and subclinical vascular lesions in a founder population in Italy.

Methods and Results—6148 subjects were enrolled (aged 14–102 years) from four towns. Hypertension (HT), diabetes mellitus (DM) and dyslipidemia (LIP) were defined in accordance with guidelines. A self-reported diagnosis defined awareness of these conditions, and the current use of specific medications as treatment.

Prevalence was HT 29.2%, DM 4.8%, LIP 44.1% and was higher in men than in women. Disease prevalence increased with age for every CV risk factor. Men were less likely than women to take anti-HT drugs and to reach BP control (9.9% vs. 16%). Only 17.6% of HT >65 years had a BP 140/90 mmHg, though 48.5% were treated. The use of statins was very low (<1/3 of eligible subjects >65 years, those with the highest treatment rate). The ratio of control-to-treated HT was lower in subjects with, than in those without, thicker carotid arteries (31.5% vs. 38.8%, $p < 0.05$) or stiffer aortas (26.0% vs. 40.0%, $p < 0.05$) or carotid plaques (26.3% vs. 41.1%, $p < 0.05$).

Conclusion—A large number of subjects at high CV risk are not treated and the management of subclinical vascular lesions is far from optimal.

Keywords

Hypertension; Diabetes; Hypercholesterolemia; Subclinical vascular disease; Treatment; Control; Population

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Introduction

Although mortality from cardiovascular (CV) disease in Westernized countries has declined over the past several decades, cardiovascular mortality and morbidity remain alarmingly high in the developed world, and are rapidly increasing in the developing world [1–3]. This may reflect, in part at least, inadequate control of population-wide traditional CV risk factors. [4–6]. Additionally, the emergence of disease in individuals deemed at low risk by current classification schemes has focused attention on early structural and functional large artery alterations (stiffening, thickening, and dilatation) that are shown to have predictive power after controlling for conventional risk stratification [7].

The cohort of the SardiNIA Study, originally conceived as a study of a Sardinian founder population investigating the genetics of complex traits/phenotypes, including CV risk factors and arterial properties [8,9, 10] enables the investigation of the effect of gender and age on the population burden of CV risk, explored by measuring traditional CV risk factors as well as large artery structure and function.

Methods

Study population

The SardiNIA Study was conceived as a study of a Sardinian founder population investigating the genetics of complex traits/phenotypes, including CV risk factors and arterial properties [8, 9,10]. Over a three- year period, 6148 subjects were enrolled, comprising over 60% of those aged 14–102 years in a cluster of four towns. Each subject came to the clinic before consuming breakfast, signed consent forms and gave a fasting blood sample so that all tests would be uninfluenced by meals. Each subject underwent a detailed medical history and full medical examination, blood pressure and anthropometric measurements, a 12-lead resting EKG, measurements of arterial structure and function and personality testing.

For the purpose of the present study, 25 subjects were excluded because of incomplete records. Age groups were classified as: <35, 35–49, 50–64 and 65 years.

Variables measured

Blood pressure—Blood pressure determinations were performed in the morning after a light breakfast with subjects in the seated position, and following a 5-min quiet resting period. Blood pressure was measured in both arms with a mercury sphygmomanometer using an appropriately sized cuff. The blood pressure values used in this study are the average of the second and third measurements of both the right and left arm. Values for systolic blood pressure (SBP) and diastolic blood pressure (DBP) were defined by Korotkoff phase I and V, respectively. Pulse pressure was computed as $PP = (SBP - DBP)$; mean BP was computed as $MBP = DBP + (PP/3)$.

Anthropometry—Height, weight and waist circumference were determined for all participants. Body mass index (BMI) was calculated as body weight (kg)/height (m)².

Fasting plasma lipids and glucose—Blood samples were drawn from the antecubital vein between 07:00 and 08:00 h after an overnight fast. Subjects were not allowed to smoke, engage in significant physical activity or take medications prior to the collection of the samples. The concentrations of plasma triglycerides and total cholesterol were determined by an enzymatic method (Abbott Laboratories ABA-200 ATC Biochromatic Analyzer, Irving, TX, USA). The concentration of HDL cholesterol was determined by a dextran sulfate–magnesium precipitation procedure. LDL cholesterol concentrations were estimated by the Friedewald formula. Fasting plasma glucose concentration was measured by the glucose oxidase method (Beckman Instruments Inc., Fullerton, CA, USA).

Arterial structure and function—Carotid-femoral PWV was measured as previously described [8]. A minimum of 10 arterial flow waves from the right common carotid artery and the right femoral artery were recorded simultaneously using nondirectional transcutaneous Doppler probes (Model 810A, 9- to 10-MHz probes, Parks Medical Electronics Inc, Aloha, OR, USA), and averaged using the QRS for synchronization. The foot of the flow, i.e., the point of systolic flow onset, was identified off-line by a custom-designed computer algorithm, and verified or manually adjusted by the reader after visual review. The time differential between the feet of simultaneously recorded carotid and femoral flow waves was then measured. The distance traveled by the flow wave was measured with an external tape measure over the body surface, as the distance from the right carotid sampling site to the manubrium, subtracted from the distance from the manubrium to the right femoral sampling site. PWV was calculated as the distance traveled by the flow wave divided by the time differential.

High-resolution B-mode carotid ultrasonography was performed by use of a linear-array 5- to 7.5-MHz transducer (HDI 3500; ATL Ultramark Inc.) as previously described [8]. The subject lay in a supine position in a dark, quiet room. The stabilized BP after 15 min from the onset of testing was used for subsequent analyses. The right CCA was examined with the head tilted slightly upward in the midline position. The transducer was manipulated so that the near and far walls of the CCA were parallel to the transducer footprint and the lumen diameter was maximized in the longitudinal plane. A region 1.5 cm proximal to the carotid bifurcation was identified, and the IMT of the far wall was evaluated as the distance between the luminal–intimal interface and the medial–adventitial interface. IMT was measured on the frozen frame of a suitable longitudinal image with the image magnified to achieve a higher resolution of detail. The IMT measurement was obtained from five contiguous sites at 1-mm intervals, and the average of the five measurements was used for analyses. CCA diastolic diameter was individuated through an ECG gating and measured similarly to IMT. CCA plaque was defined as focal encroachment of the arterial wall PWV, CCA IMT and diameter measurements were performed off-line by a single observer (A. S.) who was blinded to the identity of the participants.

In accordance with recent Guidelines [11], a CCA IMT >0.9 mm or a PWV >12 m/s were considered markers of large artery target organ damage.

Definition of hypertension

Hypertension was defined as an average BP 140/90 mmHg or if the participant was taking antihypertensive medications. The same BP criteria applied to diabetic participants. Awareness of hypertension was defined as an individuals' positive response to the question of whether they had ever been told by a doctor or other health professional that they had high blood pressure. Participants were considered to be treated if they were taking antihypertensive drugs. Hypertension was considered to be controlled in those participants receiving treatment if their average BP was <140/90 mm Hg.

Definition of diabetes

Awareness of diabetes was defined as an individuals' positive response to the question of whether they had ever been told by a doctor or other health professional that they had diabetes. We used the American Diabetes Association (ADA) diagnostic criteria for undiagnosed diabetes (fasting blood glucose level 126 mg/dl after 9 and <24 h fasting). Treatment of diabetes mellitus was defined as self-reported current use of insulin or antidiabetic pills. Glycemic control was defined as a glycated hemoglobin reading of <7.0%.

Definition of dyslipidemia

Presence of dyslipidemia was defined by LDL 130 mg/dL. Participant awareness of high LDL cholesterol was defined as a self-report of any prior diagnosis of hypercholesterolemia by a health-care professional. Treatment of high LDL cholesterol was defined as self-reported current use of cholesterol-lowering medication.

Definition of the metabolic syndrome

The Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (ATP III) [12] defined the MetS as an alteration in three or more of the following five components: abdominal obesity, triglycerides, HDL cholesterol, blood pressure (systolic or diastolic), and fasting glucose. According to this definition, the following cut-off values to define alterations were used: waist circumference >102 cm for men or >88 cm for women, triglycerides 150 mg/dL, HDL cholesterol <40 mg/dL for men or <50 mg/dL for women, blood pressure 130/ 85 mmHg, and fasting glucose 110 mg/dL.

In 2005 the International Diabetes Federation proposed an alternative definition of the metabolic syndrome [13]. According to the IDF definition, someone has the metabolic syndrome if he or she has central adiposity (waist circumference for Europid >94 cm for men and >80 cm for women) plus two or more of the following four factors: elevated concentration of triglycerides: 150 mg/dL or specific treatment for this lipid abnormality; reduced concentration of HDL cholesterol: <40 mg/dL in men and <50 mg/dL in women or specific treatment for this lipid abnormality; elevated blood pressure: systolic blood pressure 130 mmHg or diastolic blood pressure 85 mmHg or treatment of previously diagnosed hypertension; and elevated fasting plasma glucose concentration 100 mg/dL or previously diagnosed type 2 diabetes.

Statistical analysis

All analyses were performed using the SAS package for Windows (version 9.1; SAS, Cary, NC, USA). Data are presented as mean $\pm$ SD unless otherwise specified. Differences in mean values for each of the measured variables among groups were compared by ANOVA, followed by Bonferroni's test for multiple comparisons. ANCOVA analysis was used to test for interaction between age and gender.

Results

Effects of age and sex on average levels of traditional CV risk factors

A total of 6123 subjects, 2605 men and 3518 women, between the ages of 14 and 102 years completed the first SardiNIA examination.

Measurements of blood pressure, lipid, glucose, and metabolic parameter levels are provided in Table 1. All measurements differed significantly by age, with the exception of HR, and by sex, the exception being the insulin levels and the HOMA-IR index. An interaction term between sex and age resulted in significant differences for most variables (although with different statistical significance). This indicates that the effect of age on the traditional CV risk factor levels differed in men and women.

To better illustrate these trends, average levels of traditional CV risk factors were plotted by age decade in men and women. As shown in Fig. 1, BMI and fasting glucose both increased with age in men and women up to the sixth decade when it reached a plateau (and when BMI became higher in women than in men). Total and LDL cholesterol levels showed an inverted U- shaped trend with a peak in the fourth to sixth decade in men and in the fifth to seventh decade in women; of note, after the fifth decade levels in women were higher than in men. SBP showed a linear increase with age in both men and women, with an average of 5 mmHg higher values in men than in women. DBP levels peaked in the fifth decade in both men and women.

Prevalence of the metabolic syndrome and obesity: Effects of age and sex

The overweight or obese were more likely to be men and to be older. Prevalence of abdominal obesity, whether defined in accordance with the ATP III criteria or to specific values of European populations suggested by the IDF, was higher in women than in men (80% by ATP criteria; 20% by IDF criteria) (Table 2).

Prevalence of the metabolic syndrome was higher when defined in accordance with the IDF guidelines than when defined according to the ATP III criteria, which may reflect lower waist circumference threshold values adopted for European populations by the IDF (Table 2). Regardless of the definition adopted, men showed a higher prevalence of the metabolic syndrome than women and its prevalence increased sharply with age. The number of altered components of the metabolic syndrome increased with age and with regard to individual altered components, both low HDL and high triglycerides showed a similar occurrence after the age of 35. Conversely, elevated BP impaired glucose and abdominal obesity showed a linear increase with advancing age. The distribution of altered components of the metabolic

syndrome differed by gender. Men were more likely to have elevated BP or triglycerides and impaired glucose, whereas women more frequently had abdominal obesity and low HDL cholesterol.

Awareness, treatment and control of hypertension, diabetes mellitus and dyslipidemia: Effects of sex

Gender-specific results with regards to prevalence, treatment and control of hypertension, diabetes and dyslipidemia are provided in Table 3 (left columns).

Of the participants with hypertension, men were less likely than women to report the condition, to take antihypertensive medication and to reach adequate blood pressure control.

Of the participants with diabetes or with dyslipidemia, no gender difference was observed in the awareness and treatment of these conditions. Rate of control of diabetes was similar in men and women.

Awareness, treatment and control of hypertension, diabetes mellitus and dyslipidemia: Effects of age

Age-specific results with regards to prevalence, treatment, and control of hypertension, diabetes, and dyslipidemia are provided in Table 3 (right columns). The general pattern is consistent with a greater prevalence of these risk factors in the older age groups. With increasing age, the prevalence of systolic hypertension increased and that of diastolic was biphasic hypertension, with a stable prevalence of systo-diastolic hypertension (approximately 22% of the population after age of 50 (Fig. 2)).

Awareness and treatment of hypertension increased with age, but the control rate was very low: only 17.6% of hypertensive older subjects had their blood pressure controlled (BP 140/90 mmHg).

The awareness of diabetes mellitus was higher in older groups: more than half of the subjects aged 35–49 years had newly diagnosed diabetes according to their fasting glucose levels as compared to 28% of those subjects 65 years and older. The rate of control of diabetes (HbA1c <7%) was similar across age groups, comprising approximately two thirds of the affected population.

The awareness of dyslipidemia was higher in older groups. A more accurate definition of the condition would have required adopting a different LDL threshold according to the associated conditions (diabetes, prevalent cardiovascular disease, etc.). However, the use of lipid-lowering therapy was still very low; interestingly, less than one third of the subjects in the older group expected a benefit from the treatment—the group with the highest treatment rate.

Subclinical vascular lesion and management of traditional CV risk factors

Emerging evidence over the last decade indicates that age-associated alterations in large artery structure (thickening) and function (stiffening) are markers of subclinical vascular lesions and significant independent predictors of vascular events [14–18]. More recently, guidelines indicated specific threshold values for CCA IMT and carotid-femoral PWV as the equivalent of target organ damage [11], thus requiring more aggressive treatment of the affected subjects. In our population the prevalence of abnormal IMT was 1.6% (2.5 in men and 1.0 in women), with an age-related increase in the prevalence (from 0.2% in subjects 35–49 years old to 8.7% in subjects 65 years and older). The same trend was observed for elevated PWV: 3.2% in the whole population (3.5% in men and 3.0% in women) with an age gradient from 0.7% in subjects 35–49 years old to 15.7% in subjects 65 years and older) and for CCA plaque: 5.1% in the whole population (7.6% in men and 3.3% in women) with an age gradient from 0.9% in subjects 35–49 years old to 22.1% in subjects 65 years and older).

As expected, the prevalence of hypertension, diabetes mellitus, and dyslipidemia was higher in these subjects at higher risk of CV events (see Table 4). The management of subclinical vascular lesions or vascular target organ damage is far from optimal. In fact, in spite of higher treatment rates compared to “unaffected” subjects, the proportion of hypertensive subjects with BP <140/90 mmHg was lower in subjects with vascular damage, indicating a lower efficacy of treatment. For instance, the ratio of control-to-treated hypertensive subjects was much lower in those with than in those without thicker CCA (31.5% vs. 38.8% respectively, $p < 0.05$) or stiffer aorta (26.0% vs. 40.0% respectively, $p < 0.05$) or CCA plaque (26.3% vs. 41.1% respectively, $p < 0.05$).

Discussion

Major advances in our understanding of cardiovascular disease has occurred during the last several decades. The conceptualization of ‘risk factors’ has become the basis of current preventive cardiology [19–21]. The importance of high blood pressure, serum lipids, aging, gender and smoking in determining an individual's risk of developing a vascular event is widely accepted and adopted in clinical practice. It has been further supported by clinical trials showing that reduction in traditional CV risk factor levels resulted in a significant reduction of CV events. In fact, a small reduction in blood pressure, for instance, could reduce the risk of heart failure, stroke, and myocardial infarction markedly [22]. This evidence is reflected in guidelines from Scientific Medical Societies [11,12,23,24].

Although the SardiNIA Study was originally designed as a study of genetic determinants of aging-associated conditions, including traditional CV risk factors and arterial structure and function, it is valuable for the study of the health status of an Italian population because of its large sample size, complex sampling design, good quality control, and comprehensive content. Our observation in the SardiNIA cohort indicated that a considerable number of subjects, regardless of gender and age, were not aware of being affected by conditions such as hypertension, diabetes, and dyslipidemia thereby exposing them to increased risk of CV disease.

It is difficult to ascertain whether the considerable proportion of the population that was unaware of being affected by hypertension, diabetes, or dyslipidemia reflects limits in the screening strategy. Other studies reported an elevated percentage of undiagnosed hypertension [25–27] or diabetes [28–32]. These conditions are risky. For instance, population-based studies showed that the presence of unrecognized diabetes is associated with an increased risk of death [30,31,33]. It is important to note that patients with newly diagnosed diabetes have been shown to have a significantly increased risk for coronary artery disease [32,34]. Additionally, vascular wall abnormalities have been demonstrated in patients before the onset of diabetes mellitus [35,36].

The present study also extended a previous observation that a large number of subjects are not treated or are undertreated in a large Italian population of broad age range. This phenomenon has been reported for hypertension [5,6,37,38], diabetes [39,40] as well as for dyslipidemia [4,41,42]. In particular, the rate of treated/controlled hypertensive subjects was lower in older subjects. This may reflect a higher occurrence of systolic hypertension in older groups, a condition known to be challenging for adequate treatment [43,44]. Additionally, it may reflect clinical inertia and persistence of the (incorrect) paradigm that at older age traditional CV risk factors are no longer significant determinants of CV events and that prevention is not effective at older ages. Some studies reinforced this common prejudice. In fact, it has been suggested that the excess mortality associated with diabetes in younger populations is attenuated in the elderly [45–47]. However, large population studies have confirmed that diabetes is associated with excess mortality in the elderly even in those 85 years and older and that ischemic heart disease and stroke increased with age in diabetic subjects [48,49].

Additional novel information provided by the present study is the prevalence of vascular target organ damage, measured as carotid IMT >0.9 mm or PWV >12 m/s in accordance with recent Guidelines [11], in a general population. Of note, subjects with vascular target organ damage, and, thus, at a higher risk of developing CV events presented lower levels of control of established CV risk factors. This unpleasant observation has been reported in several other European countries [50,51] in subjects at high risk of CV events, but was not based upon detection of vascular organ damage or subclinical vascular lesions.

In conclusion, it is clear that the public health challenge posed by control of CV risk is substantial. Of course, in the years to come a characterization of the genetic factors that modulate [9,52] CV risk factors and how genetic factors affect the natural history of the progression from risk factor exposure to CV event or affect the response to specific therapy of subjects at risk of CV disease, will improve our tools for an effective and safe management of subjects at risk of CV events.

Acknowledgments

This work was supported by the Intramural Research Program of the National Institute on Aging (NIA), National Institutes of Health (NIH). The SardiNIA (“ProgeNIA”) team was supported by Contract NO1-AG-1-2109 from the NIA.

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Figure 1.
Average levels of traditional CV risk factors in men and women by age decade.

Figure 2. Distribution of hypertension type in men and women, in younger and older subjects. IDH, diastolic hypertension; SDH, systo-diastolic hypertension; ISH, systolic hypertension.

Table 1

Average levels of established CV risk factors by age and gender in the study population

	Men				Women				Age effect	Sex effect	Age × Sex Inter.
	<35 yrs	35–49 yrs	50–64 yrs	65+ yrs	<35 yrs	35–49 yrs	50–64 yrs	65+ yrs			
N	908	727	582	388	1253	1033	743	489			
Age (years)	24.8 ± 6.2	42.1 ± 4.2	57.1 ± 4.4	72.5 ± 5.9	25.3 ± 6.0	42.2 ± 4.4	57.0 ± 4.2	72.5 ± 6.3			
Current smoking (%)	38.7	30.5	17.5	9.3	21.5	19.0	7.8	0.8	***	***	**
Waist (cm)	82.0 ± 9.1	91.4 ± 9.0	96.4 ± 9.5	97.8 ± 9.7	73.0 ± 8.5	79.3 ± 10.6	88.2 ± 12.4	92.5 ± 13.0	***	***	***
BMI (kg/m ²)	23.6 ± 3.4	26.7 ± 3.5	28.1 ± 3.7	28.2 ± 4.0	21.7 ± 3.4	24.5 ± 4.4	27.8 ± 4.9	28.5 ± 4.8	***	***	***
Glucose (mg/dL)	85.4 ± 19.9	92.3 ± 19.2	102.7 ± 30.2	104.3 ± 33.9	80.0 ± 13.3	84.7 ± 13.9	94.2 ± 27.4	100.5 ± 29.4	***	***	*
Insulin	7.6 ± 5.8	8.5 ± 7.1	9.7 ± 8.1	8.9 ± 9.1	8.0 ± 6.2	8.0 ± 8.6	8.9 ± 9.0	10.3 ± 12.6	***	n.s.	**
HbA1c (%)	5.2 ± 0.6	5.4 ± 0.7	5.9 ± 1.2	6.0 ± 1.4	5.1 ± 0.4	5.3 ± 0.4	5.7 ± 0.9	5.9 ± 1.0	***	***	n.s.
HOMA-IR ^a	1.6 ± 1.4	2.0 ± 2.0	2.6 ± 2.9	2.4 ± 3.8	1.6 ± 1.3	1.8 ± 2.9	2.1 ± 2.5	2.7 ± 4.6	***	n.s.	**
Total Cholesterol (mg/dL)	182 ± 40	220 ± 39	227 ± 40	216 ± 40	190 ± 35	209 ± 37	231 ± 40	226 ± 40	***	*	***
LDL cholesterol (mg/dL)	110 ± 32	139 ± 33	143 ± 34	133 ± 35	110 ± 28	126 ± 31	143 ± 34	138 ± 36	***	*	***
HDL cholesterol (mg/dL)	56 ± 12	58 ± 12	61 ± 13	63 ± 15	67 ± 15	69 ± 14	69 ± 15	69 ± 15	***	***	***
Triglycerides (mg/dL)	77 ± 52	108 ± 66	114 ± 65	102 ± 58	64 ± 32	72 ± 40	91 ± 48	94 ± 47	***	***	***
Serum creatinine	0.9 ± 0.2	0.9 ± 0.2	0.9 ± 0.2	1.0 ± 0.4	0.7 ± 0.1	0.7 ± 0.2	0.7 ± 0.2	0.8 ± 0.2	***	***	*
Uric acid	4.8 ± 1.1	5.2 ± 1.3	5.5 ± 1.4	5.9 ± 1.6	3.2 ± 0.9	3.4 ± 1.0	4.0 ± 1.1	4.5 ± 1.5	***	***	***
Sodium (mEq/L)	142 ± 4	143 ± 3	143 ± 4	143 ± 4	142 ± 3	142 ± 3	143 ± 3	143 ± 4	***	***	***
Potassium (mEq/L)	4.3 ± 0.4	4.4 ± 0.4	4.5 ± 4	4.6 ± 0.4	4.3 ± 0.4	4.4 ± 0.4	4.5 ± 0.4	4.5 ± 0.4	***	***	n.s.
SBP (mmHg)	121 ± 12	128 ± 14	138 ± 18	145 ± 18	112 ± 10	119 ± 15	133 ± 18	141 ± 20	***	***	***
DBP (mmHg)	73 ± 8	82 ± 10	85 ± 11	83 ± 10	70 ± 7	76 ± 9	81 ± 10	82 ± 10	***	***	***
MBP (mmHg)	89 ± 8	97 ± 10	103 ± 12	104 ± 11	84 ± 7	90 ± 10	99 ± 12	102 ± 12	***	***	***
PP (mmHg)	48 ± 10	46 ± 10	53 ± 13	62 ± 15	42 ± 8	43 ± 10	52 ± 13	60 ± 16	***	***	***

	Men				Women				Age effect	Sex effect	Age × Sex Inter.
	<35 yrs	35–49 yrs	50–64 yrs	65+ yrs	<35 yrs	35–49 yrs	50–64 yrs	65+ yrs			
HR (bpm)	64 ± 11	64 ± 11	66 ± 12	64 ± 11	70 ± 11	69 ± 10	68 ± 10	69 ± 10	n.s.	***	***

* $p < 0.05$

**

$p < 0.01$

*** $p < 0.001$; n.s., not significant.

^a Excluding those subjects on insulin therapy.

Table 2

Prevalence of obesity, metabolic syndrome and its altered components by age and sex

	All	Men	Women	<35 yrs	35–49 yrs	50–64 yrs	65+ yrs
Overweight (%)	17.1	23.4	12.6	6.4	16.6	28.8	27.1
Obese (%)	15.7	16.4	15.2	3.7	12.7	27.1	34.3
Metabolic syndrome ATPIII (%)	6.5	7.1	6.1	0.9	3.5	12.2	18.0
Metabolic syndrome IDF(%)	9.0	11.8	7.1	1.3	5.9	17.4	21.9
Impaired glucose (%)	7.3	9.9	5.3	0.9	3.6	13.4	21.0
Low HDL cholesterol (%)	7.3	4.6	9.3	10.1	6.1	5.3	5.9
High triglycerides (%)	9.6	15.1	5.6	4.0	10.8	14.9	13.3
Elevated BP (%)	36.0	47.0	27.8	10.9	30.9	59.8	72.6
Abdominal obesity ATP (%)	22.1	15.1	27.3	4.8	15.9	38.5	52.9
Abdominal obesity IDF (%)	42.8	38.7	45.8	14.9	40.0	70.0	76.3
ATP III metabolic syndrome altered components							
0	48.0	41.0	53.0	74.1	52.8	22.3	11.7
1	29.8	35.0	26.0	22.2	31.3	38.5	33.0
2	15.6	16.9	14.9	2.8	12.4	27.0	37.3
3	5.2	5.5	4.9	0.7	2.8	9.5	14.1
4	1.2	1.5	1.0	0.1	0.5	2.4	3.5
5	0.2	0.1	0.2	0	0.2	0.2	0.3

Table 3

Awareness, treatment, and control of hypertension, diabetes mellitus, and dyslipidemia by age and sex

	All	Men	Women	<35yrs	35–49 yrs	50–64 yrs	65+ yrs
Hypertension (%)	29.2	36.6	23.8	4.4	21.1	50.9	73.7
Aware ^a (%)	29.2	34.8	49.8	5.2	22.6	43.9	56.0
Treated ^a (%)	33.3	27.6	39.8	3.1	14.0	33.8	48.5
Controlled ^a (%)	12.8	9.9	16.0	3.1	6.2	13.1	17.6
Diabetes (%)	4.8	6.0	4.0	0.6	1.9	8.6	15.5
Aware ^a (%)	66.7	63.3	70.0	100	42.1	62.8	71.6
Treated ^a (%)	43.4	41.0	46.1	64.3	24.2	42.1	47.1
HbA1c <7% ^a (%)	64.3	62.8	66.0	28.6	66.7	68.4	64.0
Type 1 Diabetes (%)	0.4	0.5	0.3	0.5	0.3	0.2	0.3
Dyslipidemia							
LDL 160 mg/dL (%)	16.6	19.2	14.7	6.0	17.5	29.1	22.4
LDL 130 mg/dL (%)	44.1	46.9	42.1	22.5	48.8	65.1	56.7
Aware (%)	12.5	12.3	12.7	2.9	10.2	21.6	27.4
Lipid lowering therapy (%)	5.7	6.2	5.3	0.0	1.8	6.4	17.1

^aRefers to the affected population (hypertensive or diabetic or dyslipidemic subjects).

Table 4

Prevalence and management of hypertension, diabetes, and dyslipidemia according to the presence of vascular target organ damage

	<u>CCA IMT >0.9 mm</u>		<u>PWV >12 m/s</u>		<u>CCA plaque</u>	
	No	Yes	No	Yes	No	Yes
Hypertension (%)	28.4	73.0	27.5	77.9	26.8	73.2
Treated ^a (%)	32.5	52.1	32.5	41.9	30.9	49.8
Controlled ^a (%)	12.6	16.4	12.9	11.0	12.7	13.1
Diabetes (%)	4.6	20.0	4.3	21.6	4.0	20.1
Treated ^a (%)	43.3	45.0	44.0	39.5	41.9	49.2
Controlled ^a (%)	64.3	65.0	63.8	67.4	62.8	69.8
Dyslipidemia						
LDL 160 mg/dL (%)	16.5	27.0	16.4	24.1	16.3	23.0
LDL 130 mg/dL (%)	43.9	59.0	43.6	59.8	43.2	61.0
Lipid lowering therapy (%)	5.6	11.5	5.4	12.6	4.8	18.3

^aRefers to the affected population (hypertensive or diabetic or dyslipidemic subjects).