showed a tendency to decrease (P>0.05). C-peptid levels decreased by 34.62% (P<0.05) vs the control. IR1 and C-peptid initial levels in group III increased by about 4 times compared to group I and the control (P<0.05). These factors decreased 25.81% (P<0.05) and 52.68% (P<0.05) compared to the initial values and the control. The concentration of VEGF in group I was twice as high as in the control at 29.80±4.73 ng/ml vs 19.33±2.09 ng/ml in the control (P<0.05). E-1 levels for group I did not change at 2.63±0.95 pg/ml vs 1.50±0.15 pg/ml in the control (P>0.05). VEGF level decreased to that of the control (P<0.05) and E-1 level remained at that value. VEGF levels in groups II and III appeared to be 1.5-2 times higher than the control. VEGF after treatment decreased but was still higher than the control by 22.65% (P<0.05). The E-1 blood levels in groups II and III were higher by a factor of 5 and 7 times (P<0.05) vs the control. E-1 levels decreased by a factor of 2-3, but did not reach the control. In conclusion, moxonidine decreased the level of hyperinsulinemia, normalized the insulin secretion and endothelial vasoregulation in patients with AH and IR.

Key Words: Endothelial Dysfunction, Insulin Secretion, Moxonidine

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PRESERVATION OF THE ARTERIAL PRESSURE RESPONSE TO LEPTIN IN DIET-INDUCED OBESE MICE: A POTENTIAL MECHANISM FOR OBESITY HYPERTENSION
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We have previously demonstrated that diet-induced obese mice have selective resistance to leptin with preservation of the renal sympathetic nerve activity response to leptin despite loss of the feeding- and weight-reducing actions of leptin. The preserved renal sympathetic response to leptin associated with the hyperleptinemia observed in mice on high fat diet might be expected to increase arterial pressure in these obese mice.

To test the ability of leptin to increase arterial pressure in the obese mice we compared the action of intraperitoneal administration of leptin in mice on normal chow and high fat diet for 10 weeks. Radiometric mean arterial pressure (MAP) was recorded in the conscious unrestrained state for 7 days. Lean and obese mice then received 12 days treatment with vehicle or leptin (60 mg, twice daily, IP) with continued MAP recordings. After 10 weeks, mice on high fat diet weighed about 10% more than those fed the normal chow (30.8±0.2 vs 27.9±0.2 g, P<0.001). Mice on high fat diet showed higher baseline pressure than the mice on normal chow (110±1 vs. 100±1 mmHg, P<0.001). In mice on normal chow, 12-day leptin treatment caused a significant increase in MAP as expected (+9±6 and +2±2 mmHg for leptin- and vehicle-treated mice respectively, P<0.001). Leptin caused also a significant increase in MAP in the high fat-fed mice (+6±5 vs. +5±1 mmHg, P<0.01). Leptin treatment caused a significant decrease in body weight, food intake and fat mass in mice on normal chow, but not in the high fat-fed mice. This study demonstrates that there is preservation of the arterial pressure response to leptin in a dietary model of obesity despite resistance to the appetite and weight reducing actions of leptin. This represents a potential mechanism for increases in arterial pressure and adverse cardiovascular actions of leptin in obesity.

Key Words: Hypertension, Leptin, Obesity

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METABOLIC SYNDROME INCREASES THE RISK OF HYPERTENSION-INDUCED ORGAN DAMAGE IN HYPERTENSIVES ATTENDING PRIMARY CARE CENTERS. ERIC-HTN STUDY
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The objective was to assess the influence of metabolic syndrome on hypertension-related organ damage (left ventricular hypertrophy and renal function) in a large survey of hypertensives who attend primary care clinics.

Subjects and Methods: Hypertensive subjects of both genders, aged >55 years were randomly selected among those attending primary care centers all over Spain. In all subjects, the existence of cardiovascular disease and cardiovascular risk factor status (body mass index, baseline glucose, lipid profile, and diabetes) were assessed. The EKG-left ventricular hypertrophy was assessed by using the Cornell criteria, and glomerular filtration rates were calculated using the Cockcroft-Gault formula. Metabolic Syndrome was defined according the NECP, in which BMI >28.8 kg/m² for males and >26.2 kg/m² for females instead of waist circumference was considered as a criteria. Risk for LV hypertrophy and/or renal insufficiency were sought by using a logistic regression analysis in which age, sex, and body mass index were included.

Results: A total of 12857 subjects (mean age 67.6 years, 55.4% women) were included. After adjusting for age, sex and BMI, the relative risk for LV hypertrophy was significantly higher in diabetics 2.0 [1.8-2.2] and in non-diabetics with Metabolic Syndrome 1.4 [1.2-1.6] as compared with non-diabetic in absence of Metabolic Syndrome (p<0.001). Likewise, risk for renal failure was also significantly higher for diabetes 1.6 [1.5-1.8] and for non-diabetics with Metabolic Syndrome 1.3 [1.2-1.5], (p<0.001).

Conclusions: In a population of hypertensives going to primary care facilities, metabolic syndrome was accompanied by a higher prevalence of hypertension-induced organ damage, LV hypertrophy and renal insufficiency. Diabetes further increases the risk.

Key Words: Left Ventricular Hypertrophy, Metabolic Syndrome, Renal Function

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A ACTIVATED IMMUNE SYSTEM AS A CAUSE OR AN EFFECT OF THE SYNDROME X. THE IMPORTANCE OF THE C3
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Introduction: Pickup has pointed out the role played by the immune system in the pathogenesis of the metabolic syndrome, specially in type 2 diabetic (DM2) patients. However, its participation in the early stages of this disease remains unknown. On the other hand, all its clinical and biochemical components are known to lead to atherosclerotic disease via complex mechanisms. The activation of the complement system stands out in this process, particularly through the C3 component. Nevertheless, its importance as a marker of insulin resistance (IR) deserves further investigation.

Aims: 1. To characterize a cardiovascular risk population according to its C3 serum levels. 2. To correlate such levels with anthropometric and metabolic parameters.