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Targeting hypertension in patients with the cardio-renal metabolic syndrome

Manuel Velasco², Edward Rojas¹, Zafar Israili³, Valmore Bermudez¹, Peter Bolli⁴ and Luigi Cubeddu⁵

¹University of Zulia. Endocrine and Metabolic Diseases Research Centre "Dr. Felix Gomez". Maracaibo – Venezuela ²Central University of Venezuela. Clinical Pharmacology Unit of José María Vargas School of Medicine, Caracas – Venezuela ³Emory University School of Medicine, Atlanta, USA

⁴Ambulatory Internal Medicine Teaching Clinic, St Catharines, USA ⁵Nova Southeastern University, Health Professions Division, Fort Lauderdale, USA

piabetes Mellitus (DM) and Hypertension are two of the most prevalent diseases in developed countries among adult population. By 2010 the estimated DM prevalence rose to 285 million representing 6.4% of the world's adult population¹ and its coexistence with hypertension is greater than chance alone would predict.² Several mechanisms have been described explaining synergistic associations between DM and hypertension. Hypertensive patients have shown to have altered composition of skeletal muscle tissue (increased fat interspersed between skeletal muscle fibres), decreased blood flow to skeletal muscle tissue due to vascular hypertrophy and vasoconstriction (decreased delivery of insulin and glucose) and post receptor signalling alterations in the IRS insulin pathway, all inducing insulin resistance states³ which partially explains why blood pressure goals in DM patients are lower (<130/80mmHg) than normoglycemic.^{4,5} There is evidence β -Blockers have shown to increase the risk of hypertensive patients to develop DM due to weight gain and decreased blood flow to skeletal muscle tissues^{6,7} and thiazides are shown to induce hyperglycemia. Although optimal first-step antihypertensive drug therapy in type 2 DM or impaired fasting glucose levels (IFG) is uncertain and each patient should be individualized, angiotensin-renin-aldosterone system antagonists have demonstrated in some but not all⁸ studies to decrease the rate of development of proteinuria and diabetic renal disease. According to the ACCF/AHA 2011 Expert Consensus Document on Hypertension, elderly persons with diabetes, hypertension, and nephropathy should be treated initially with Angiotensin Converting Enzyme Inhibitors (ACEI's) or Angiotensin Receptor Blockers

(ARB's)⁹ By the other hand, the ACCOMPLISH Trial showed that over the background of ACEI patients with diabetes mellitus who were treated with amlodipine had a 21% relative risk reduction and 2.2% absolute risk reduction in CV events compared with hydroclorotyazide plus the ACEI.¹⁰ Based on available data, reduction of macrovascular and microvascular complications in diabetics or metabolic syndrome patients with hypertension depends more on effectively reducing the blood pressure than on the type of drugs used and drug choice may also depend on associated comorbidities.

Biography

Manuel Velasco, M.D., is a Professor of Pharmacology at the Central University of Venezuela, School of Medicine, and Director of Clinical Pharmacology Unit (CPU) at the Vargas School of Medicine in Caracas, Venezuela. He has contributed significantly in the understanding of Endogenous Amines in blood pressure control, and his observations on anti-hypertensive drugs have helped spark the renewed interest in clinical pharmacology of this disease. He has also served as a member of the Editorial Board or Reviewer ad hoc of the following international journals: Hypertension, the Journal of Cardiovascular Pharmacology, Clinical Pharmacology and Therapeutics, the Journal of Hypertension, the Journal of Clinical Pharmacology. He also serves as a Member of the Editorial Board of the American Journal of Therapeutics and reviewer ad hoc of the Journal of Hypertension and the Journal of Clinical Pharmacology. Dr. Velasco has edited several international congress series of Elsevier (The Netherlands). In the area of Cardiovascular Physiology, Hypertension, Clinical Pharmacology and Therapeutics and Internal Medicine. He also serves as Editor-in-Chief of two of the most accredited Scientific Journals in Latin America: "Archivos Venezolanos de Farmacología y Terapeutica" y la "Revista Latinoamericana de Hipertensión". Dr. Velasco is one of the top scientists involved in Hypertension and Clinical Pharmacology combining years of extensive research and clinical experience. Dr Velasco is an author and co-author of many scientific papers in the field of cardiovascular diseases and metabolic syndrome diseases (Hypertension, prediabetes, diabetes, obesity, insulin resistance, dyslipidemias, etc).