Ambrogio Capria MD

Curriculum Vitae

Ambrogio Capria MD, born in Rome on 04/23/1951, graduated in Medicine and Surgery (1975), specialist in Internal Medicine (1980) and in Cardiology (1984), is in service since 1985 as a Researcher – Aggregate Professor - at the Department of Internal Medicine, University of Rome "Tor Vergata", from 1985, MED09 sector. He is actually full-time structured at the AFA of Medicine, U.O. of Internal Medicine, Policlinico Tor Vergata, Rome.
Professor at the Integrated Course of Internal Medicine, Faculty of Medicine, University of Rome "Tor Vergata", participates with lectures, exercises to students and clinical mentoring. He is also a lecturer at the Degree Course in Obstetrics and teaches in the Schools of Specialization in Internal Medicine, Cardiology, Respiratory Diseases, Endocrinology and Allergy and Immunology Clinic of the University of Rome "Tor Vergata". He is thesis supervisor Degree in Medicine and Surgery and Postgraduate thesis supervisor in Internal Medicine and Cardiology. He has commissioned to Professional Internship in Internal Medicine.
He is referee for the American Journal of Cardiology, the British Medical Journal and other medical Journals.
The scientific activity is documented by more than 150 scientific papers and reports presented at national and international conferences, with contributions that relate to pharmacology and cardiovascular pathophysiology in clinical conditions such as ischemic heart disease, cardiomyopathy, hypertension, the neurally mediated syncope, the primary or secondary dysautonomia involving the Parkinson Disease, the connective tissue disease, as soon as the abnormal autonomic control of the gastrointestinal system in various pathological conditions, presented at national and international conferences as well as published in full or in press.
The head research topics are regarding the study of endothelial function in connective tissue, which have as their object the functional study and cardiovascular cardiovascular imaging in scleroderma, implemented methods electrocardiographic, echocardiographic, and imaging (CT, MRI) documenting anatomical and functional alterations that underlie the high cardiovascular morbidity and mortality of their scleroderma; They have been identified some peculiar pattern of scleroderma disease, relevant to the correct pathophysiological classification and treatment, with functional impairment and significant prognostic variable, the risk of mortality, sometimes sudden. The endothelial functional study of scleroderma has also documented a severe endothelial damage, characterized by a marked reduction in vasoreactivity endothelium-dependent and endothelium-independent, with clinical expression in Raynaud's phenomenon and acral critical ischemia, with erratic and unpredictable response to treatment with prostanoids and other systemic vasoactive drugs.
They also looked into the peculiarities of the autonomic nervous system in Sjogren disease, where it is clear a marked parasympathetic overactivity, indirect expression of immune-mediated damage, which in the presence of a normal endothelium-dependent vasoreactivity, characterizes the symptoms frequently observed in such autoimmune exocrine disease with marked lability of the blood pressure and high frequency of postural hypotension with inappropriate sinus tachycardia to posture changes.
Other studies have as their object the endothelial functional involvement in rheumatoid arthritis, lupus erythematosus and psoriatic arthritis. In particular, they were conducted serial clinical evaluations, biochemical and instrumental in patients with rheumatoid arthritis unresponsive DMARDs, analyzing in detail the endothelial functional behavior in the clinical setting; This has allowed to document that endothelial dysfunction is a sensitive marker of active systemic disease, being detectable from the early stages of the diseases of ia, significantly reversible in response to treatment with various biological agents anti-TNFa, both at the medium and at long term, and the predictive value of a loss of endothelial response to treatment for subsequent systemic adverse

events, in combination with the evolution of clinical and biochemical indicators of inflammation. So, these studies support a marked and significant observed consistency between the changes of inflammatory markers, the regression of the articular inflammation and the restored endothelium-dependent vasoreactivity in response to treatment, that clearly supports the prevailing role of TNF in rheumatoid disease, compared to other pro-inflammatory cytokines. Other recent studies are evaluating the clinical role of the peripheral vascular reactivity, driven by the autonomic system activity and the endothelial function levels, in subjects affected by neuromediated syncope. Finally, other in-progress studies are currently evaluating the clinical and pathophysiological role of the observed changes in the endothelial function in patients with advanced heart failure, non responders to the usual pharmacologic therapy, that are currently treated and responding to CRT (Cardiac Resynchronization Therapy) devices, used for selected patients with advanced heart failure.
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