WHAT IS THE BEST TREATMENT FOR ASYMPTOMATIC CAROTID STENOSIS (ACS) CEA, CAS OR MEDICAL TREATMENT? NEW MARKERS AND NEW APPROACH: CRITICAL OVERVIEW

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Introduction: Controversies between CEA and CAS are lasting more than 15 years. Recently accumulating evidence suggests that current best medical treatment alone is sufficient for ACS. What to do in such situation and what should we tell the patient?
Aim: Critical overview by using new markers and new approach.
Material & methods: Data from Cochrane, Embase, ISI (1995-2011),own material:566 subjects 1.C-healthy controls(n=102),2.AH-arterial hypertension(n=117)3.CAD-Coronary artery disease(n=72), 4.iCI-ischemic cerebral infarction(n=92), 5.postCEApts(n=183),Lp-PLA2 (Dia Dexus IncUSA), stiffness, intima media tickness (IMT-Sphygmocor, At Cor Sydney), plaque morphology (USG), baroreceptor sensitivity (Finometer, Amsterdam), LDL-subfractions –Lipoprint Inc.USA). Prospective multicenter, multidisciplinary study
Results: CEA/CAS in SCS and ACS moderately reduced stroke risk. Results led to increasingly perform CAS. At that time aspirin was the sole antiplatelet therapy, before time of statins, ACI, new antihypertensive agents. Better results using CEA still exist. Documented advent of modem medical therapy, its increasing use; there are new markers, consequently new approaches. Our study showed significant increase in PWV (m/s), augmentation index (%), IMT(mm), plaque morphology,HDL3-7,LP-PLA2 in AH, iCI and CAD groups. Theoretically when lipid and LP-PLA2 are low, this situation is associated with stable plaque, when the lipid and LP-PLA2 are high, this is associated with unstable plaque and its rupture. It brings unfavorable conditions for CEA/CAS.LP-PLA2 is a key inflammatory marker, specifically linked to plaque inflammation/rupture. It represent novel therapeutic target for slowing/stopping atherosclerotic plaque formation and rupture, and new results in CEA/CAS. Comprehensive medical prevention (platelets, statins, antidiabetics, BP regulation) has shown decreasing annual ipsilateral stroke rate to 0.34%.
Conclusions: This has been remarkable time in stroke prevention and therapy. It brought new challenges for better perspectives of stroke patients. However, the job is not finished. Trials are appropriate only when substantial uncertainties about important questions exist. New studies are needed

Supported by EU/intern.grantITMS26220220099