

Abstract

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Increased systemic oxidative stress after elective endarterectomy: relation to vascular healing and remodeling.

Mezzetti A, Guglielmi MD, Pierdomenico SD, Costantini F, Cipollone F, De Cesare D, Bucciarelli T, Uchino S, Chiarelli F, Cuccurullo F, Romano F.

Centro per lo Studio dell'Iipertensione Arteriosa delle Dislipidemie e dell'Aterosclerosi, University "Gabriele D'Annunzio", Chieti, Italy.

BACKGROUND: It has been reported that systemic and local redox state may have an important role in the functional and organic changes characterizing the process of vascular response to injury. Carotid endarterectomy to remove atherosclerotic plaque is followed by a long lasting healing and remodeling process that can be carefully followed over time with noninvasive ultrasonography.

OBJECTIVE AND METHODS: Plasma vitamin C concentration and native LDL (n-LDL) content in lipid peroxides, vitamin E, beta-carotene, and lycopene as well as LDL susceptibility to peroxidation were assessed in 45 patients undergoing elective endarterectomy for internal carotid stenosis, at baseline, 24 hours, 3 and 15 days, and 1 month after surgery. Serial duplex scans were performed in all patients postoperatively and 3, 6, and 12 months. The changes in far wall thickness (FW) and % renarrowing from postoperatively to 12 months were used as remodeling indices.

RESULTS: Plasma antioxidant vitamins and lag-phase showed a sharp and significant decrease during the first 24-hours after surgery remaining unchanged until the third day, whereas, an opposite trend was evidenced for n-LDL content in lipid peroxides and serum ceruloplasmin. After the third day all the parameters returned progressively to baseline within one month from endarterectomy. Interestingly, the n-LDL lipid peroxide content, the serum ceruloplasmin and the plasma vitamin C concentration, measured at 24 and 3 days from surgery, were significantly associated to the change in % renarrowing from postoperatively to 12 months. The higher the LDL content in lipid peroxides, the higher the serum level of ceruloplasmin, the lower the plasma content in vitamin C and the higher the % of vessel renarrowing.

CONCLUSION: In conclusion, carotid endarterectomy with atherosclerotic plaque removal is associated with an acute and prolonged increase in systemic oxidative stress that influences vascular healing and late luminal loss.

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