Impact of chronic kidney disease on carotid plaque vulnerability.

Little is known about the effect of chronic kidney disease (CKD) on plaque morphology in cerebral vessels. We therefore analyzed plaque composition and metabolic and chemical parameters with regard to clinical outcome in patients with advanced carotid artery stenosis (>70%) and normal or impaired renal function. Carotid endarterectomy plaques were collected from 114 patients, 51 with CKD and 63 without CKD (mean estimated glomerular filtration rate, 49 ± 9 vs 88 ± 14 mL/min), and analyzed by histology and immunohistochemistry. Serum levels of matrix metalloproteinases (MMP-1, -2, -3, -7, -8, and -9), calcium, phosphate, parathyroid hormone, fetuin-A, osteoprotegerin, and inflammatory factors, including fibrinogen, and high-sensitive C-reactive protein (hsCRP) were measured by appropriate enzyme-linked immunosorbent assay. Compared with patients without CKD, patients with CKD had significantly more early-stage (11.2% vs 2.8%, P = .002) and end-stage (7.4% vs 0.2%, P = .036) calcification, unstable (50.8% vs 20.4%, P = .001) and ruptured (53.1% vs 32.8%, P = .035) lesions, and a significantly lower amount of collagenous fibers (39.2% vs 54.6%, P = .001). Serum samples of CKD patients had significantly enhanced levels of fibrinogen (393 ± 88 vs 331 ± 60 mg/dL, P = .018), hsCRP (1.7 ± 2.9 vs 0.8 ± 0.9 mg/dL; P = .042), parathyroid hormone (47.3 ± 24.1 vs 32.8 ± 12.2 ng/L, P = .010),
fetuin-A (0.21 ± 0.05 vs 0.18 ± 0.04 mg/mL, P = .039), and MMP-7 (13.0 ± 5.3 vs 8.3 ± 3.0 ng/mL; P6 months before carotid surgery was significantly increased in CKD patients (84.0% vs 26.2% P< .001). In patients with CKD and advanced carotid artery stenosis, morphologic changes in plaque composition may contribute to plaque vulnerability and consequently to the risk of cerebrovascular events. Furthermore, relevant serum markers of inflammation, vascular calcification, and vessel wall degradation might be an indication of stroke risk in CKD patients.

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