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LETTER TO THE EDITOR

WILEY

Hypertension is a possible risk factor for cervical artery dissection

About one-fifth of ischemic stroke in people below the age of 50 is caused by cervical artery dissection (CeAD) (carotid and/or vertebral).[1] The exact aetiology of this condition is unknown. Trauma is responsible of 30% of CeADs but the rest occur spontaneously,[2] suggesting a different mechanism. Many environmental and genetic risk factors have been proposed such as α1-antitrypsin deficiency, hyperhomocysteinemia, recent infection, connective tissue disorders, styloid process length, and arterial tortuosity.[3] Few studies specifically addressed the link of CeAD with cardiovascular risk factors. In the Cervical Artery Dissections and Ischemic Patients (CADISP) group, which is a large international multicentre registry, the prevalence of smoking, diabetes and hypertension were statistically lower in CeAD than in non-dissection ischemic stroke.[4] Hypertension, on the other hand, was more statistically associated with CeAD compared to healthy controls, with no difference between the two groups in relation to the other cardiovascular risk factors.

This year we published two meta-analyses of case-control studies to specifically address the association of cardiovascular risk factors with CeAD. In the first meta-analysis we compared 2185 patients with CeAD and 3185 healthy controls.[3] There was a significant association of CeAD with hypertension and no association with smoking or diabetes. There was a negative association with hyperlipidaemia but sensitivity analysis with inclusion of studies that only compared total cholesterol between the CeAD and control groups showed no statistical association. In the second meta-analysis, we compared cardiovascular risk factors between stroke related to CeAD and ischemic stroke of other causes.[5] Smoking, hyperlipidaemia, and diabetes were more significantly associated with non-dissection than with dissection-related ischemic stroke. This association was lacking for hypertension which suggests it could be a risk factor for both types of stroke, further consolidating the results of the first meta-analysis. We also conducted a systematic review of postpartum and pregnancy-related CeAD and found a strong association of this condition with hypertension and hypertensive disorders of pregnancy such as eclampsia/preeclampsia.[6]

All these studies suggest that hypertension is the only cardiovascular risk factor that has an association with CeAD. The lack of this association with other cardiovascular risk factors and the younger age of patients with CeAD compared to patients with non-dissection ischemic stroke and patients with aortic dissection suggests that hypertension exerts its effects on arterial wall through non-atherosclerotic mechanisms. The proposed mechanisms include shear stress across vascular lumen induced by blood pressure fluctuations[7] in the presence of vascular wall weakness such as cystic medial necrosis, which is also more prevalent in patients with hypertension.[8] However, both hypertension and CeAD could represent different manifestations of fibromuscular dysplasia (FMD) that could cause high blood pressure via inducing renal artery dissection and stenosis. FMD is reported in 8% of CeAD in IPSYS CeAD study (Italian Project on Stroke in Young Adults Cervical Artery Dissection)[9] and in 40% in the ARCADIA-POL study (Assessment of Renal and Cervical Artery Dysplasia–Poland),[10] so it is only one cause of CeAD, and CeAD is only one manifestation of FMD. However, the prevalence of hypertension in FMD is about 72%, and 65% of patients with renal artery FMD have coexistent extracranial carotid and vertebral artery disease,[11] suggesting an intercausal relationship between the three.

We think our research should direct future research toward exploring mechanisms via which hypertension exerts arterial wall damage, specifically primary research involving experimental animal models.

AUTHOR CONTRIBUTION

Loay H. Abdelnour has solely contributed to the conceptualization, design, writing, and revision of this manuscript.

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