Branch atheromatous disease: an important cause of small subcortical infarction in Asia

Jong Sung Kim

Branch atheromatous disease (BAD) refers to atheromatous occlusion of a small, perforating vessel. BAD produces small subcortical infarction (SSI) and presents clinically with lacunar syndrome. However, this is pathophysiologically distinguished from traditional small vessel disease (SVD) caused by lipohyalinosis primarily affecting the distal part of perforators. According to vascular imaging findings, BAD may be divided into infarcts associated with parental artery disease (PAD) and those without. Although perforating vessels cannot be visualized by current imaging technologies, SSI abutting on the parenteral vessel can be considered a BAD caused by proximal atherosclerotic occlusion of a perforating vessel. Thus, in clinical practice, SSI may be divided into SSI with PAD (SSI + PAD), proximal SSI without PAD (pSSI-PAD) and distal SSI without PAD (dSSI-PAD). We examined the prevalence of SVD markers (leukoaraiosis and microbleeds) and atherosclerosis markers (cerebral atherosclerosis and coronary heart disease) among these groups, and found that SSSI + PAD had the highest prevalence of atherosclerosis markers and the lowest prevalence of SVD markers, whereas dSSI-PAD had the lowest prevalence of atherosclerosis markers and the highest SVD markers. pSSI-PAD showed intermediate features. The prevalence of these markers also differed according the vascular territories; atherosclerosis markers significantly increased while SVD markers decreased as the vascular territory became lower (from middle cerebral artery, basilar artery to vertebral artery). These results illustrate that SSI has a heterogeneous pathogenesis. Since intracranial atherosclerosis is a major cause of stroke in Asia, the importance of BAD should be appropriately recognized.