Cerebral microbleeds (CMBs) are found in healthy geriatric population as well as in association with stroke and cognitive impairment. CMBs are small, round, hypointense lesions found incidentally on T2*-weighted gradient echo and susceptibility-weighted sequences of magnetic resonance imaging (MRI) brain. Studies have shown that in ischemic stroke patients, CMBs not only increase the subsequent intracranial hemorrhage (ICH) risk but also increase the risk of ischemic stroke. Lobar CMBs are very specific for cerebral amyloid angiopathy in Western (Caucasian) population with ICH but not in Oriental (Asian) population as well as in the absence of ICH. While CMBs in the subcortical areas as well as infratentorial locations are thought to be secondary to long-standing hypertension and arteriosclerotic changes, it has been seen that ethnicity does not confound the association between CMBs burden and ischemic stroke or ICH risks.

Potigumjon et al. in their study (Prevalence of cerebral microbleeds in Thai patients with ischemic stroke) published in the current issue of this journal showed that the prevalence of CMBs was 20% in the Thai population which was never reported earlier. This was lower than the Japanese cohort but was at par with the published Chinese population. In their study, hypertension and small vessel disease on MRI especially Fazekas stage 3 related to hypertension was associated with increased risk for CMBs. Stroke etiology also seems to be important clue to the presence of CMBs. CMBs are seen more commonly in association with lacunar stroke and large artery atherosclerosis rather than cardioembolic etiology, especially in Asian population. Turc et al. found that increased CMBs burden was associated with worse 3-month modified Rankin Scale functional outcome in acute ischemic stroke patients treated with thrombolytics but was not significant after adjustment of confounding factors. There are meta-analyses which suggest that hemorrhagic transformation as well as symptomatic ICH is significant in the presence of CMBs, especially for those strictly located in the lobar areas, as compared to those without CMBs when treated with thrombolytics in acute ischemic stroke. However, the authors in their paper discussing CMBs prevalence in Thai population did not find a significant difference in hemorrhagic transformation rates in patients with CMBs as compared to those without CMBs, treated with thrombolytics for acute ischemic stroke. As such presence of CMBs on the neuroimaging should not preclude the use of thrombolytics in the treatment for acute ischemic stroke, more data are available.

Wilson et al. showed that in the presence of increased CMBs burden of ≥5, treatment with antithrombotics substantially increased the absolute risk of ICH as compared to absolute risk of ischemic stroke. Clinicians still do face the dilemma of using antithrombotics in patients with minor cerebrovascular events who have increased CMBs burden on neuroimaging. Interestingly, Vernoij et al. in their cross-sectional study found increased prevalence of CMBs among antiplatelet users than anticoagulant users. It was found in a study that CMBs >4 was associated with cognitive decline and increased risk of dementia. The prevalence of CMBs increases with age and hypertension, and to some extent based on ethnicity, CMBs pose a clinical dilemma with regard to acute and preventive treatment in acute ischemic stroke population. Further randomized controlled trials are needed to answer these questions.