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Not all cortical cerebral microbleeds are due to cerebral amyloid angiopathy

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Cerebral microbleeds (CMBs) are commonly detected on routine MRIs of the brain in centres that incorporate a susceptibility sequence as standard. The susceptibility weighted imaging (SWI) sequence is increasingly available and is more sensitive than gradient echo in detection of small susceptibility abnormalities caused by iron from haemoglobin, occurring either as CMBs or cortical superficial siderosis, with the underlying diagnosis usually inferred to be cerebral amyloid angiopathy (CAA) for the more peripheral hemisphere location of CMBs. Cortical superficial siderosis is likely a more specific marker of CAA[1] and in patients with cortico-subcortical CMBs alone, other causes should be considered.

Investigators from Adelaide report in this volume of BMJ Neurology Open a pattern of CMBs consistent with CAA according to the modified Boston criteria but occurring following cardiac surgery, either coronary artery graft surgery or cardiac valve replacement in patients referred to their two TIA clinics.[2] Clinical and radiological criteria for probable CAA were met in 39% of their post cardiac surgery cases. They found that subcortical white matter (SWM) CMBs rather than purely cortical CMBs were more common in the cardiac surgery patients than in the probable CAA patient subset without a history of cardiac surgery. They derived a ratio of purely SWM CMBs to SWM+strictly cortical CMBs which on receiver operating characteristic analysis delivered 93% specificity at a cut-off of 0.45 for their non-CAA cardiac surgery group. In their patients with CAA, the SWM/SWM+strictly cortical CMB ratio was 0.05.

CMBs concluded to be due to CAA have implications for prognosis and choice of anticoagulants and antiplatelet drugs, but for CMBs derived from a single event complicating cardiac surgery, modifying antithrombotic medication choice would be unnecessary. For clinicians receiving reports of MRIs that raise suspicion of CAA, it is worth considering the context, including cardiac surgical history, and perhaps the detail of location as shown in De Sciscio *et al*'s paper. As the authors point out, validation of their WM/WM+cortical CMB ratio should be explored in other series. It is worth noting that community patients have a lower rate of CAA with cortical CMBs than intracerebral haemorrhage patients[3] and likely TIA clinic patients with cortical CMBs will have a lower rate of CAA than stroke unit inpatients with CMBs.

Where there is a doubt about the diagnosis of CAA with CMBs on SWI, stability over time may be a helpful indicator of a more benign prognosis. How long one should wait to be sure that the CMBs are solely the result of a past single event? In a recent study from Boston, not all patients with CAA progressed with new CMBs during a median of 1.34 years between MRIs.[1] Apart from convincing stability of CMB numbers with follow-up over a few years, other more convenient methods will be needed to more quickly confirm or exclude a diagnosis of CAA. New imaging parameters may help and perhaps blood or CSF markers.

The Adelaide investigators raise the possibility of a further revision of the Boston citeria to incorporate past cardiac surgery and its pattern of CMBs. Such a development might inspire more caution in clinicians, including radiologists, before confirming CAA on the basis of a single MRI showing cortical CMBs regardless of context.

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