The Role of Oedema in Lacune Formation

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I read with interest the recent paper by Ma and Olsson [1] concerning the proposed role of oedema in the formation of lacunes in Binswanger's encephalopathy. From my own neuropathological observations I agree that the lesion they refer to as 'oedema-associated gliosis' is a common finding in the ageing brain, which is often associated with classical type 1 [2] lacunes and with diffuse white matter degeneration. The similarity of such lesions to those seen in rodent models of brain oedema may indeed suggest that focal blood-brain barrier breakdown at least contributes to their formation. However, their conclusion that these represent 'developing' lacunes is potentially misleading; the pathological features they describe suggest that they are of no more recent onset than the classical cavitated type 1 lacune with which they are compared. The relative sparsity of macrophages compared to classical lacunes is a reflection of the relatively minor tissue damage, and is, I suggest, a manifestation of so-called 'incomplete infarction' rather than evolving or developing infarction. Incomplete infarction has long been recognised as a consequence of global cerebral circulatory insufficiency, but is now recognised also to result from arterial occlusion of short duration and/or moderate severity [3, 4]. The importance of this concept is that it implies mechanisms other than permanent occlusive lesions of penetrating endarteries which, by definition, will produce foci of coagulative necrosis of all cellular elements (or classical type 1 lacunes). Possible underlying causes might therefore include transient embolic occlusion and transiently or moderately reduced local cerebral blood flow, perhaps as a consequence of global cerebral circulatory insufficiency with local subtotal small-vessel stenosis. With regard to the former possibility, we have been impressed by a relatively high frequency of possible sources of cerebral emboli in our cases, as well as by the pathological similarity of the lesions to those seen in a rat model of cerebral platelet emboli [5]. As regards the latter, the consensus view as to the aetiopathogenesis of Binswanger's subcortical arteriosclerotic encephalopathy is that it is ischaemic in origin, whether this is mediated by cerebral small-vessel narrowing, blood pressure or cerebral blood flow autoregulatory dysregulation or other factors [reviewed in 6].

Whilst not denying the possible importance of oedema-mediated tissue damage in any brain infarct, it is perhaps more useful to regard 'oedema-associated gliosis' as 'incomplete' small, deep (lacunar) infarcts. This interpretation may better focus the search for the underlying cause.

References


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