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Editorial

The Impact of Cerebral Amyloid Angiopathy in Neurodegenerative and Cerebrovascular Diseases

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Dementia in most aged patients is due to a combination of different neurodegenerative processes and frequently associated to cerebrovascular lesions [1].

The clinical diagnosis of cerebral amyloid angiopathy (CAA) is made accordingly the Boston criteria proposed by Greenberg. It has to be mainly suspected in elderly patients presenting with a lobar haematoma and without any known cerebral vascular risk factors such as arterial hypertension, hypercholesterolemia and cardiovascular disease [2]. CAA does not occur during the normal aging process [3].

CAA can occur independently from other cerebral diseases. However, it is mainly associated to Alzheimer's disease (AD), but can also occur together with other neurodegenerative diseases. Brains with mild and severe AD and with CAA contain more haematomas, cortical micro-infarcts and micro-bleeds, and more severe white matter changes than those without CAA. In the CAA brains with severe AD features, also more cortical territorial infarcts are observed, compared to those with mild AD features [4]. Cerebrovascular lesions are, however, more severe and frequent in CAA brains without AD than in those with AD [5].

In contrast to the frequent association of AD and CAA, frontotemporal lobe degeneration, which is a heterogeneous disorder with sometimes overlapping features, CAA and cerebrovascular lesions are rare. CAA is also not observed in brains with amyotrophic lateral sclerosis [6].

Lewy body disease (LBD) is the second most frequent neurodegenerative disease associated to CAA. However, the impact of CAA is restricted to an increase of cortical micro-infarcts. Lobar haematomas are absent. The severity of the white matter changes is similar to LBD brains without CAA [7].

The only impact of CAA in brains with progressive supranuclear palsy (PSP) brains is the frequent occurrence of cortical superficial siderosis [8].

Vascular dementia (VaD) is mainly due to lacunar infarcts and white matter changes. However, CAA is also an important contributor to VaD. It is responsible for an increased occurrence of lobar haematomas, territorial infarcts and cortical micro-bleeds and micro-infarcts. The severity of white matter changes is unchanged compared to VaD brains without CAA [9].

In conclusion CAA is an important associated pathology in many neurodegenerative and cerebrovascular diseases, causing dementia.

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