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Magnetic Resonance Imaging of White Matter Changes in Brains of Patients with Dementia

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Abstract

White matter hyperintensities (WMHs) are frequently observed on magnetic resonance imaging (MRI) in brains of patients with dementia. Initially they were considered as the reflection of ischemic changes in the white matter. However, more recently this statement has been questioned. The most relevant articles concerning the presence of WMHs on MRI in patients with different types of dementia are presently reviewed. In vascular dementia and in its more specific form, called Binswanger's disease, the WMHs are to be considered as representing ischemic changes. This is based on the frequent association with small-vessel disease and lacunar infarcts. Also hereditary cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (Cadasil) is a significant ischemic cause of WMHs on MRI. The WMHs are linked to the most relevant cortical neurodegenerative changes in "pure" Alzheimer's disease, frontotemporal lobar degeneration, Lewy body disease, and progressive supranuclear palsy and corticobasal degeneration. They have to be considered as the expression of Wallerian degeneration with secondary myelin loss, rather than ischemic of origin. In most mixed dementias vascular ischemic lesions are combined with neurodegenerative changes in the cerebral white matter.

Keywords: Magnetic resonance imaging; White matter hyperintensities; Vascular dementia; Alzheimer's disease; Frontotemporal lobar degeneration; Lewy body disease; Progressive supranuclear palsy; Corticobasall degeneration

Introduction

White matter changes are frequently observed in several diseases of infancy and adult life, including in demyelinising diseases, such as multiple sclerosis [1-5]. The development of brain white matter microstructure starts during the first 3 years of life with further continued white matter maturation during later childhood and adolescence [6]. The diffusion characteristics on magnetic resonance imaging (MRI) differ between the regions of white matter changes and the surrounding normal looking white matter, suggestive of a Wallerian-type degenerative pattern [7]. There exist different visual MRI rating scales for the white matter hyper-intensities (WMHs) [8-10]. Overall, WMHs are considered to mainly reflect white matter ischemia [11-15]. However some studies suggest that they also can be due to Wallerian degeneration and demyelisation as result of the severe overlying cortical neurodegenerative changes [16-18].

Even in cognitive normal elderly brains an increase of WMHs is observed compared to those of young and middle-aged persons [19]. The mixed forms of dementia occur in 30% of all dementia cases. They are more frequent in the oldest patients and have more severe WMHs than in those with a single type of dementia [20-22]. So the question remains whether the WMHs in all dementia diseases are due to cerebrovascular disturbances or secondary to neurodegenerative changes of the overlying cerebral cortex.

Arterial vascularisation of the cerebral white matter

The majority of the cerebral white matter is supplied by medullary branches issued from the leptomeningeal cerebral arteries. In contrast to the cortical branches, they have no significant side branches and cross perpendicularly the cerebral cortex to reach the white matter. The short medullary branches end in the subcortical arcuate fibres. The long ones end in the

periventricular white matter at some distance from the ventricular wall [23]. The deep periventricular white matter is supplied in the frontal regions by ventriculofugal end-branches of the lateral striatal arteries and in the parieto-occipital regions by ventriculufugal branches issued from the choroid arteries. They form together with the medullary branches the periventricular arterial border-zones [24]. They are the remnants of the continuous loops, which connect the surface vessels in the premature brains to the periventricular located germinal layer, from which maturating neurons migrate to the surface to form the cerebral cortex. The regression of these continuous loops lead to the development of the periventricular arterial border-zones [25].

Vascular dementia

Periventricular infarction due to carotid artery stenosis can be the cause of a single stroke. This rare type of stroke is considered as a pure hemodynamic induced event [26]. Vascular dementia is causing around 15% of all dementia cases [27]. It is the second most frequent cause of dementia [28]. The most classical vascular dementia due to selective white matter ischemia with lacunar infarcts and without cortical involvement is the chronic Binswanger's subcortical encephalopathy [29]. A severity scale has been proposed for vascular dementia [30]. Another rare cause of dementia with selective white matter ischemia is the hereditary cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL) due to the Notch homolog 3 [31]. However many cases of vascular dementia are due to a mixture of WMHs with cortical macro- and micro-infarcts, macro- and micro-bleeds, and lacunar infarcts. The WMNs are more severe and widespread than in most neurodegenerative diseases [32,33]. Cerebral amyloid angiopathy (CAA) can contribute to vascular dementia [34]. The number of cortical micro-infarcts and cortical micro-bleeds is increased in CAA but the severity of the WMCs is not amplified [35-37].

Alzheimer's disease

Alzheimer disease (AD) is by far the most frequent form of dementia with an incidence of 15% in the overall population older than 65 years [38]. On MRI WMHs are found in approximately 62% of patients with AD. CAA is present in 58% of the cases [39]. Overall patients with AD-CAA have more WMHs than in those without CAA [40]. However, only those with severe CAA display the most severe cerebrovascular lesions, including an increase in WMHs [41]. The frontal, central and parieto-occipital white matters are approximately involved to the same degree in the severe forms of AD [42]. The patients with CAA are overall older than those without [43].

Frontotemporal lobar degeneration

Frontotemporal lobar degeneration (FTLD) is a heterogeneous disorder with various genetic and histological subtypes. This entity is characterised by severe atrophy of the frontal cortex and the superior temporal gyrus. The underlying white matter is severely affected in the frontal and temporal lobes [44]. On MRI, in addition to the severe WMHs, the incidence of cortical micro-bleeds is increased in the most affected regions [45,46]. A low incidence of cerebrovascular risk factors has been observed in FTLD [47]. Also CAA is very rare in this disease [48]. Amyotrophic lateral sclerosis (ALS) has been linked to FTLD [49]. Positron emission tomography of the brain shows functional imaging abnormalities predominantly in the frontal and temporal regions in a mixed ALS-FTLD case [50]. The WMHs in ALS are also predominant in the frontal and temporal regions. These findings are mainly observed in patients with memory disturbances [51,52]. Similar to FTLD a favourable vascular risk profile is observed in ALS [53].

Lewy body dementia

Lewy body dementia (LBD) is related to Parkinson's disease dementia. They are considered as the extremes of a continuous spectrum [54]. WMHs are a relative early feature in LBD [55]. They can be more severe than in Parkinson's disease with dementia. Also the cortical gray matter is more severely affected [56]. AD-related pathology is frequently associated to LBD and considered as the main cause of the WMHs [57, 58]. CAA is present in 30% of the cases and lipohyalinosis in 10% [57]. CAA does not increase the degree of the WMHs [59]. Associated cerebrovascular lesions are frequently associated and for frequent in Parkinson's disease compared to their incidence in other neurodegenerative diseases, including AD [60].

Progressive supranuclear palsy

Progressive supranuclear palsy (PSP) is a sporadic disease with tau pathology, mainly involving the thalamus, the pallidum and the brainstem [61]. However, neocortical areas can also be involved [62]. The global severity of the WMHs in the cerebral hemispheres of PSP is more or less similar to that in normal age-matched controls [63]. The association of CAA does not influence the degree of WMHs [64]. In PSP the midbrain, pons and the regions close to the basal ganglia are the regions where the main WMHs occur [65]. The pathways of degeneration mainly involve the connections between frontal areas and deep gray matter structures [66]. The WMHs are less severe in PSP than in Parkinson's patients [61]. The degree of the WMHs correlates with the clinical scores of disease severity and cognitive impairment [67]. The classic Rirchardson's syndrome of PSP has more spatial abnormalities in the frontal white matter than in the parkinsonian type [68]. However, overall the WMHs progress more significantly over time in PSP-parkinsonism [69].

Corticobasal degeneration

Corticobasal degeneration (CBD) is a rare disease characterised by a progressive asymmetrical severe cortical atrophy, mainly of the frontal and temporal lobes [70]. There are sporadic case reports mentioning white matter degeneration in the adjacent regions [71,72]. In our recent study of 8 CBD cases the WMHs are significantly more severe in the affected hemisphere, compared to their occurrence in age-matched non-demented controls [73]. The WMHs are, however, less severe in CBD than in Parkinson's patients with and without dementia [60].

Discussion

MRI is the best way to detect the severity of grey and white matter changes, using visual rating scales [74]. In the white matter of the brain myelin and iron are closely linked due to the presence of iron in myelin generating oligodendrocytes [75]. Myelin loss is followed by increase of intra- and extra-cellular water content. These changes are more pronounced among elderly people [76].

In vascular dementia the WMHs are to be considered as reflecting ischemic changes [77,78]. Small vessel disease causes the WMHs on MRI [79]. In young adults without evidence of cerebrovascular disease control of modifiable cardiovascular risk factors induces less occurrence of WMHs on MRI [80]. In particular control of blood pressure in hypertensive patients reduces significantly the occurrence of WMHs [81]. The impact of WMHs has been demonstrated to be a major risk factor for progressive cognitive decline [82]. However, the progression of WMHs does not predict the conversion from mild cognitive impairment to dementia [83]. The progression of the WMHs in AD is similar to that in the correspondent cortical regions with the most severe neurodegeneration and to be considered as due to Wallerian degeneration with secondary myelin loss [84]. The degree of WMHs is related to the amyloid load of the brain but not to the tau burden [85].

The co-occurrence of vascular brain damage is frequent and underscored in AD brains. Most AD cases are frequently to be considered as a mixed type of dementia [86]. In FTLD the severity of the WMHs is clearly linked to the underlying most affected cortical regions and probably reflects Wallerian degeneration with secondary myelin loss [87-88]. FTLD has a very low vascular risk profile so that an additional ischemic contribution is highly improbable [46-47]. Global cortical amyloid burden is high in LBD [89]. However, the WMHs reflect mainly AD-related pathology rather than cerebrovascular changes [58]. Apolipoprotein (AOE) E 4 may influence the association between WMHs and cognitive performance [90]. Severe WMHs appear to be predominantly associated with frontal/executive dysfunction, irrespective of APOE 4 allele presence [91]. So the Increase of WMHs in LBD has to be considered as mainly reflecting neurodegenerative changes.

WMHs contribute to the motor, cognitive and behavioural deficits in PSP [92]. Mainly axial and diffusivity changes are prominent [93]. The cognitive impairment is mainly related to decreased gray matter of deep nuclei and cerebellum [94]. The clinical phenotypes of CBD vary considerably and can change according to the disease progression [71]. CBD has a low vascular risk factor [47]. The MRI of the brain shows mainly focal atrophy of the bilateral frontal cortex and aymmetrical regional WMHs of the sub-adjacent white matter. The latter changes primarily reflect the progression of neuronal degeneration, especially the demyelination secondary to axon loss or change [95]. The long fronto-parietal connecting tracts, the intraparietal associative fibres, and the corpus callosum are predominantly affected [96-98]. It can be concluded that WMHs only reflect ischemic changes in VaD. In all single forms of neurodegenerative dementia diseases WMHs on MRI reflect Wallerian degeneration with myelin lost, secondary to the main cortical lesions. AD brains are frequently mixed forms of dementia with associated cerebrovascular pathology.

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